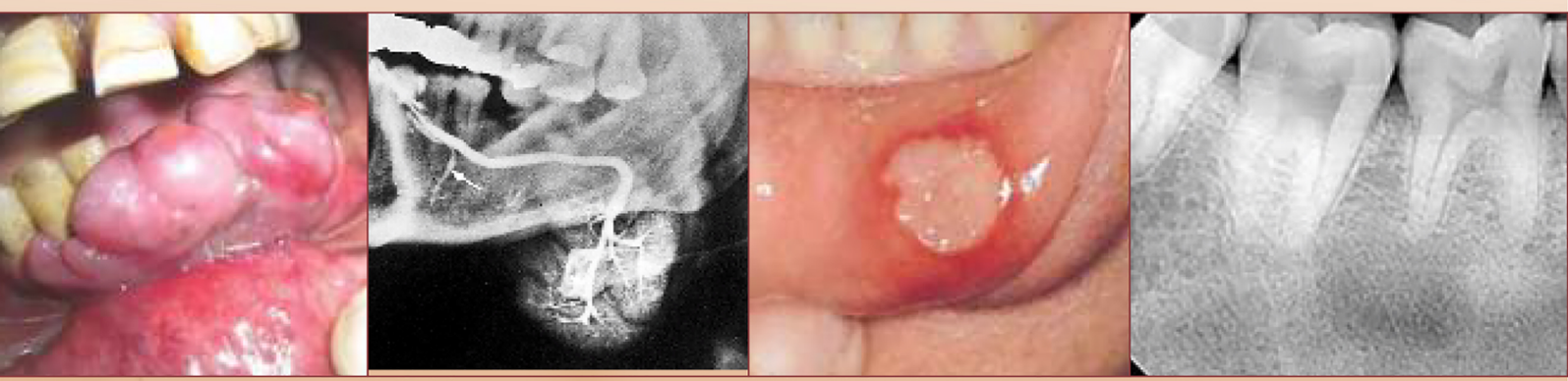
Oral Medicine and Radiology

Question–Answer format for
Review and
Exam Preparation



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Oral Medicine and Radiology

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the book was a dream
come true because of teamwork

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Preface

It gives me immense pleasure to write and publish this book. I do not claim the originality and full completeness of the matter. In fact the book is based on various concepts laid down in standard textbooks. This book was possible only because I had the advantage of authentic literature provided by these authors.

Presently, there are many textbooks on oral medicine and radiology by various authors, having appropriate coverage of the subject. The purpose of this book is to prepare the student for examination, especially for competitive examination and viva voce. The book attempts to highlight small aspects of the subject which have more value especially during seminars and various postgraduate activities. I have tried to collect various subject materials important for undergraduate and postgraduate curriculums from various books, so that different topics will be briefed under one heading.

The question and answer format in fact is made so that the students can have a very simpler approach towards the subject. Several attempts are made in this book to orient the subject and make the subject easy to remember, recollect and reproduce. The book carries some clinical tips too which will guide during clinical postings.

I am very much indebted to Dr Birangane RS, Principal, Professor and Head, Department of Oral Medicine and Radiology, PDU Dental College, Solapur, for his constant support in writing this book. His stand and dynamic leadership before and during the write up was a great source of inspiration.

I am thankful to Dr Sanjeev Onkar, Professor, Oral Medicine and Radiology, PDU Dental College, Solapur, for his contribution in collecting, analyzing and editing the matter. The enthusiasm shown by him during this project was really encouraging.

I am also thankful to Dr Swapnali Chowdhary, *Ex*-Faculty (Reader), Oral Medicine and Radiology, PDU Dental College, for her open-handed contribution in writing this book. The prompt response and inclination towards this project shown by her was simply incredible.

I am pleased for the support provided by Dr Rohan Chowdhary, Senior Lecturer, Oral Medicine and Radiology, PDU Dental College, for his every single assistance and valuable hints for completeness of this project.

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I am really indebted to Dr Sumeet P Shah for his zeal, interest and meaningful contribution to this project.

Dr Abdulla Kazi, PG student of Oral Medicine and Radiology, PDU Dental College, has a great role in this project. The disciplined approach, the regularity, punctuality and inclination towards the subject was amazing. His overall role as writer and editor was vital.

I am grateful to Ramesh Krishnammachari and all team members of CBS Publishers & Distributors for the support shown by them for this project.

I will be ever indebted and grateful to my wife Dr Priya, my children Atharva and Ayushi and all my family members for their constant support during the project. This book would never have been possible without their support.

I am thankful to my teachers for their valuable guidance.

Last but not least is the Almighty to whom I will be highly abide to whatever he has created in this world and directions he is giving us to run on.

My special thanks to Mr YN Arjuna (Senior Vice President Publishing, Editorial and Publicity), Mrs Ritu Chawla (AGM Production), Mr Prasenjit Paul, Mr Parmod Kumar and Mr Rohan Prasad, for their skilful service and immense help in editing and preparing illustrations of this book.

Abhay Suresh Kulkani

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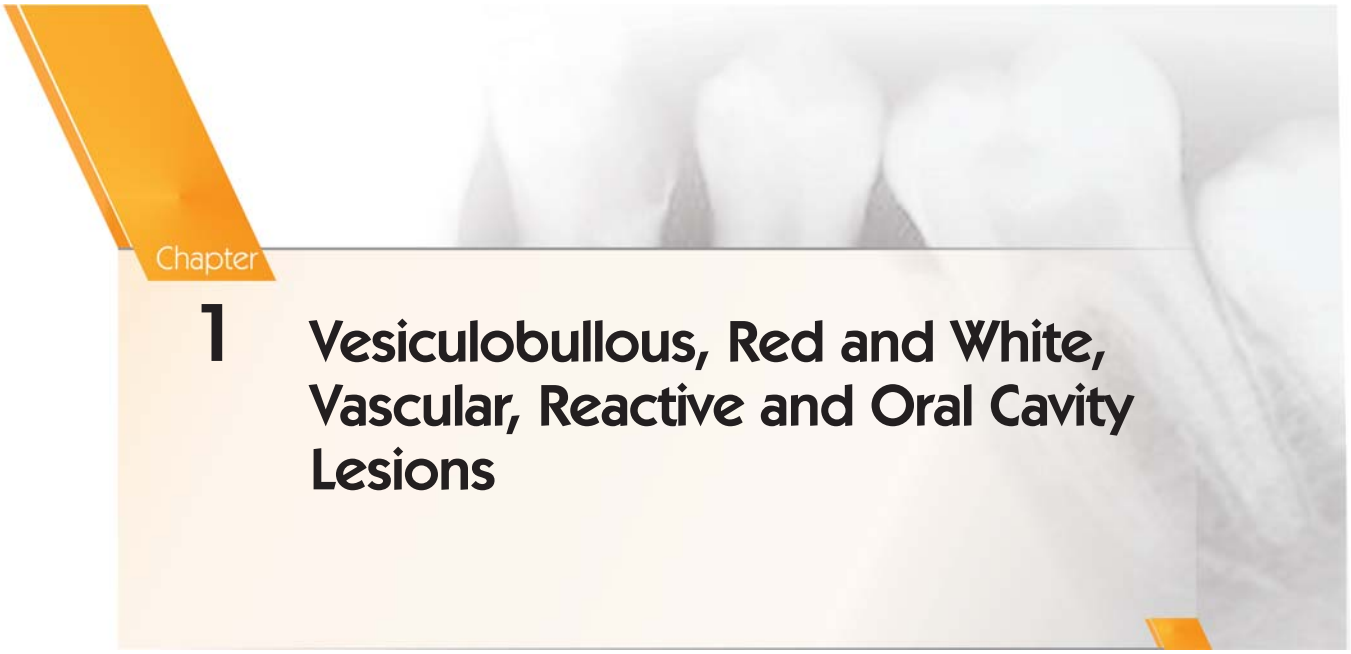


Chapter

1 Vesiculobullous, Red and White, Vascular, Reactive and Oral Cavity Lesions

1. What is lesion? What are primary and secondary lesions?

Lesion: An abnormal change in structure of an organ or part due to injury or disease, especially one that is circumscribed and well defined.

- *Primary lesions:* These are physical changes in the skin considered to be caused directly by the disease.
- *Secondary lesions:* These are those lesions that are characteristically brought about by modification of the primary lesion either by the individual with the lesion or through the natural evolution of the lesion in the environment.

2. What is the decision tree of oral mucosal lesions?

The decision tree can be simplified as:

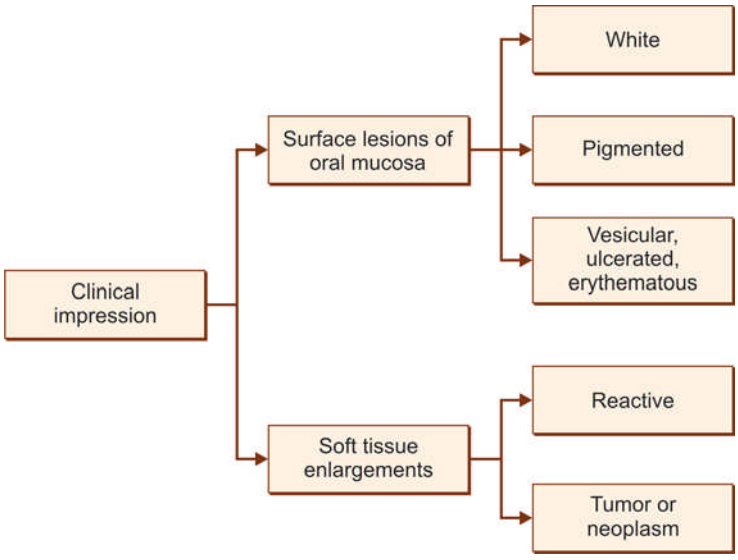
A. **Soft tissue enlargements** are characterized by being persistent and progressive; they do not resolve without treatment. They are usually not painful early in their development, and the growth rate varies from weeks to years. These include tumor/cysts/neoplasm and reactive.

Reactive soft tissue enlargements may increase and decrease (fluctuate) in size and usually eventually regress. Reactive enlargements are often, but not always, tender or painful and usually have a more rapid growth rate (measured in hours to weeks) than tumors. Some reactive enlargements begin as a diffuse lesion and become more localized with time. Sometimes reactive lesions are associated with tender lymph nodes and systemic manifestations, such as fever and malaise. Once it is decided that a soft tissue enlargement is reactive, the next step is to determine what the lesion is reacting to, such as bacterial, viral, or fungal infections or chemical or physical injury.

Soft tissue tumors are characterized by being persistent and progressive; they do not resolve without treatment. They are usually not painful early in their development, and the growth rate varies from weeks to years.

Tumors—benign and malignant

- *Benign tumors* are typically better defined or circumscribed and have a slower growth rate, measured in months and years, than malignant neoplasms.
- *Malignant neoplasms* are more likely to be painful and cause ulceration of the overlying epithelium than benign lesions.



Since malignant neoplasms invade or infiltrate surrounding muscle, nerve, blood vessels, and connective tissue, they are fixed or adherent to surrounding structures during palpation. Some benign tumors are also fixed to surrounding structures, while other benign tumors are surrounded by a fibrous connective tissue capsule, which may allow the lesion to be moved within the tissue independent of surrounding structures.

B. **Surface lesions of oral mucosa** consist of lesions that involve the epithelium and superficial connective tissue of mucosa and skin. They do not exceed 2–3 mm in thickness. Clinically, the surface lesions are slightly thickened or flat rather than swellings and enlargements. Surface lesions are divided into three categories based on their clinical appearance: White, pigmented, and vesicular, ulcerated, erythematous. These include white pigmented, vesicular, ulcerated and erythematous lesions.

3. Define vesicle, bulla. Can vesicle turned into bulla? What are coalesced vesicles?

- **Vesicle** is elevated blister containing clear fluid which is less than 1 cm in diameter.
- **Bulla** is elevated blister containing clear fluid more than 1 cm in diameter.

Vesicle and bulla both are primary lesions by definition. One primary lesion cannot turn into other, if it happens it is secondary lesion. Vesicle cannot change into bulla. However, in some diseases the smaller vesicles may coalesce to form larger vesicles which may clinically appear as bullae. These larger and fused vesicles may be called coalesced vesicles or larger vesicles.

4. Enumerate the vesiculobullous lesions which present predominantly as bullous lesions.

Pemphigus vulgaris, bullous pemphigoid, benign mucuous membrane pemphigoid, bullous lichen planus, erythema multiforme, Stevens-Johnson syndrome, epidermolysis bullosa, linear IgA disease.

5. Enumerate the vesiculobullous lesions predominantly presents as vesicular lesion.

Herpes simplex virus infection (primary herpetic gingiostomatitis), varicella (chickenpox), herpes zoster (shingles), hand-foot-and-mouth disease, herpangina, dermatitis herpetiformis.

6. How the size of pemphigus bulla varies compared to other diseases?

As a general rule, the pemphigus bulla is smaller than the bulla in benign mucous membrane pemphigoid and considerably larger than those seen in the viral diseases such as herpes and hand-foot-and-mouth disease.

In short viral disease bullae are smaller than pemphigus and pemphigus bullae are smaller than benign mucous membrane pemphigoid.

7. What is Nikolsky's sign? State all conditions in which it is positive.

Gentle pressure on clinically unaffected mucosa and skin produces new lesion (in form of vesicle/bullae/stripping of mucosa) in that area is Nikolsky's sign.

It is positive in pemphigus, paraneoplastic pemphigus, benign mucuous membrane pemphigoid, toxic epidermal necrolysis, burns, epidermolysis bullosa, bullous lichen planus, bullous impetigo, staphylococcal scalded skin syndrome, mycosis fungoids and the sign also allegedly occurred in a patient with systemic sclerosis who developed D-penicillamine-induced pemphigus vulgaris.

8. What are phenotypes of pemphigus? What are characteristics of pemphigus lesions?

Mucosal dominant and mucocutaneous dominant.

As the antigen is located in epithelium flaccid bulla may develop which will soon rupture and form ulceration.

Oral lesions may be initial sign (80–90%) and last to disappear.

Females are most commonly affected.

9. What is paraneoplastic syndrome? What are diagnostic criteria of paraneoplastic pemphigus?

Paraneoplastic syndromes are a group of clinical disorders associated with malignant diseases that are not directly related to the physical effects of primary or metastatic tumor. Or *paraneoplastic syndromes* are cancer-associated clinical syndromes caused by biologic or humoral factors, including hormones, cytokines, and immunoglobulins. It may parallel the underlying malignancy and successful treatment of tumor may lead to disappearance of syndrome. Sometimes may be the first sign of malignancy and its recognition may be critical for early detection of cancer.

1. Painful, progressive stomatitis, with preferential involvement of tongue. The tongue involvement is very much consistent.
2. Histologically acnthatholysis/lichenoid/interface dermatitits. Sometimes repeated biopsies are necessary to detect acantholysis (even though it is readily detectable in oral lesions) due to masking by necrosis and secondary inflammation. Lesions on skin sometimes are not detectable and sometimes the lesions are lichenoid and erythema multiforme like. Direct immunofluorescence (DIF) is negative frequently.
3. Antiplakin antibodies are seen.
4. Presence of underlying lymphoproliferative neoplasm and two-thirds of cases are associated with malignant disease (Hodgkin's lymphoma, CLL) and others are associated with Castleman's disease, abdominal lymphoma, thymoma, retroperitoneal sarcomas. CT sacn may be used to detect.

10. Enumerate the conditions causing palatal petechiae.

- Trauma from fellatio (direct trauma and negative pressure)
- Trauma from severe vomiting
- Prodromal sign of hemostatic disease
- Sometimes in children from herpangina
- In bulimia nervosa trauma from finger
- Trauma from severe coughing
- Prodromal sign of infectious mononucleosis
- Streptococcal pharyngitis
- Sometimes in scarlet fever (early sign)
- Leukemia (early sign)

11. State all categories under which lichen planus can be classified and justify. Why?

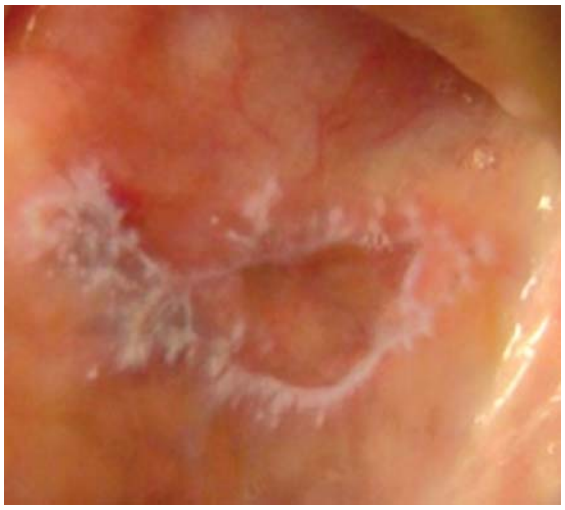
Lichen planus can be classified as:

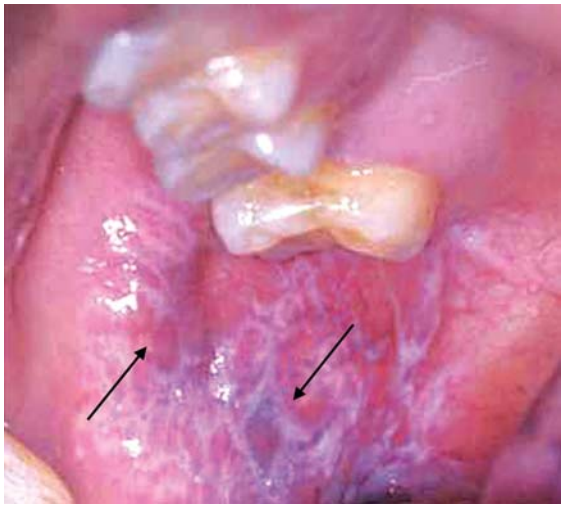
a. **Autoimmune disease:** As clinically:
   1. Female predilection.
   2. Late occurrence of disease
   3. Most of time bilateral occurrence
b. **Potentially malignant disorder of oral cavity:** As the erosive variety has got malignant potential
c. **White lesion:** It may exhibit as asymptomatic white lesion, e.g. reticular lichen planus
d. **Red and white lesions:** As it exhibits both red and white components, e.g. atrophic and erosive variety
e. **Psychosomatic disorders affecting oral cavity:** Many authors stated that psychological intervention may be warranted given the fact that level of anxiety and salivary cortisol in oral lichen planus (OLP) patients are high, supporting the relationship of OLP with stress. The most frequent conditions which may lead to lichen planus are depression, anxiety and stress. Exacerbation of orallichen planus has been linked to period of psychological stress and anxiety.
f. **Vesiculobullous lesions:** As it has bullous variety.
g. **Mucocutaneous disorders:** As it may involve skin and oral mucosa simultaneously.

12. What are Wickham's striae?

The term Wickham's striae (WS) was coined by Louis Frédéric Wickham in the year 1895 and corresponds to fine whitish points or grey lacy lines or dots seen on the top of the popular rash and oral mucosal lesions of lichen planus (LP). These resembles the lichen type moss that is often seen on rocks. The Wickham's striae are accentuated by immersion of oil on skin surface.

The pathogenesis of Wickham's striae is believed to be thickening of granular layer. The same type of keratotic striae is seen in lupus erythematosus but these are more delicate and subtle than seen in lichen planus and show characteristic radiation from a central focus.





13. What is fountain sign?

Hypertrophic lichen planus is most pruritic form of lichen planus and fountain sign is seen during intralesional injections. Lesions of LPH are characterized by hypertrophic verrucous plaques predominantly distributed over the shins. While injecting these plaques with corticosteroids by a 26 G needle, it has been often found that the medicine comes out through the follicular openings in a jet mimicking a

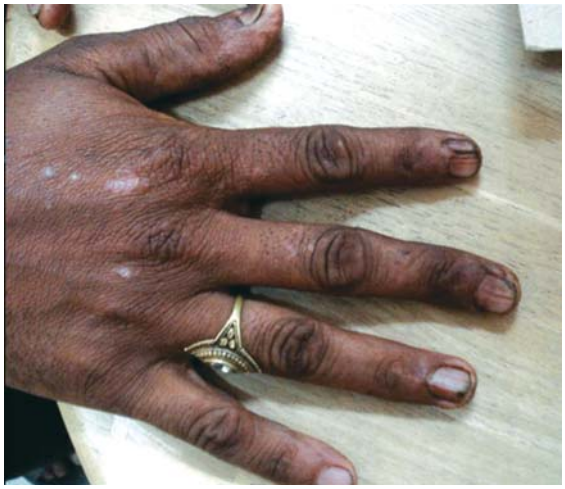
"fountain". This phenomenon is mostly seen in LPH lesions of less than 2 years duration.

14. What are syndromes associated with lichen planus?

- **Vulvovagina gingival syndrome:** The involvement of vulva, vagina and gingiva with lichen planus is called vulvovagina gingival syndrome. The erosive type is most frequent type.
- **Grinspan syndrome:** This is the triad of oral lichen planus, diabetes mellitus, and hypertension. Because drug therapy for diabetes mellitus and hypertension is capable of producing lichenoid reactions of the oral mucosa, the question arises as to whether Grinspan's syndrome is an iatrogenically induced syndrome.
- **Graham Little syndrome (Graham Little-Piccardi-Lassueur syndrome):** This consists of LPP of scalp, non-cicatrical pubic/axillary hair loss, follicular keratotic papules resembling keratotis pilaris on limb, trunk and retroauricular areas and typical cutaneous or mucosal lichen planus. This is a rare syndrome.

15. What is pup-tent sign?

Discrete red or violaceous papules in the nail bed may lift and split the overlying nail plate



longitudinally, and split lateral edges angle forward to give a pup-tent appearance. This has been referred to as pup-tent sign. The "tenting" or "pup-tent" sign is observed as a result of nail bed involvement that elevates the nail plate and may cause longitudinal splitting.

16. Why do you get white lesions of oral cavity?

Mucosal lesions clinically appear white because of many reasons:

1. Increased thickness of epithelium as a result of increased number of constituent cells (hyperplasia/acanthosis), e.g. frictional keratosis
2. Increased and abnormal production of keratin (hyperkeratosis), e.g. frictional keratosis, linea alba (physiological keratosis), actinic keratosis, white sponge nevus
3. Imbibition of fluid by surface keratin/or epithelial cells. This results in hydration and oedema results in cloudy white lesions
4. Deposition of exogenous material like material alba
5. Surface debris lesions associated with necrosis of overlying epithelium. Formation of pseudomembrane which is white that results from necrosis which results from coagulation of surface tissue. Removal of pseudomembrane may leave raw mucosal

surface with tiny bleeding spots. These are painful lesions, e.g. thermal burn, chemical burn, psuedomembranous candidiasis, fibrin clot

6. White lesions due to subepithelial change—these have normal overlying epithelium, but changes in the connective tissue partially mask blood vessels and cause the area to appear white, yellow or tan. These lesions have a smooth translucent surface, do not rub off, and are not painful. Lack of vascularity in hyperplastic connective tissue. This is responsible for pale or opaque appearance as seen in scar tissue. Oral submucous fibrosis, scarring (subepithelial fibrosis)
7. Submucosal deposits of sebaceous glands give rise to yellowish granular appearance (Fordyce's granules).

17. Classify white lesions of oral cavity.

1. **Hereditary/developmental:**
   a. Leukoedema
   b. White spongy nevus
   c. Hereditary benign intraepithelial dyskeratosis
   d. Pachyonychia congenita
   e. Dyskeratosis congenita
2. **Reactive:**
   a. Frictional keratosis
   b. Morsicatio buccarum
   c. Nicotine stomatitis
   d. Tobacco pouch keratosis
   e. Chemical burn
3. **Immunologic:**
   a. Lichen planus
   b. Lichenoid mucositis
   c. Discoid lupus erythematosus
   d. Graft-versus-host disease
4. **Bacterial/viral/fungal:**
   a. Candidiasis
   b. Mucous patches in secondary syphilis
   c. Oral hairy leukoplakia
5. **Systemic disease:**
   a. Uremic stomatitis
6. **Potentially malignant disorders:**
   a. Leukoplakia
   b. Actinic cheilitis
7. **Neoplastic:**
   a. Squamous cell carcinoma

18. Enumerate hereditary white lesions.

White sponge nevus, HBID (Witkop's disease), follicular keratosis (Darrier's disease).

19. Enumerate the conditions causing bilateral white lesions on buccal mucosa.

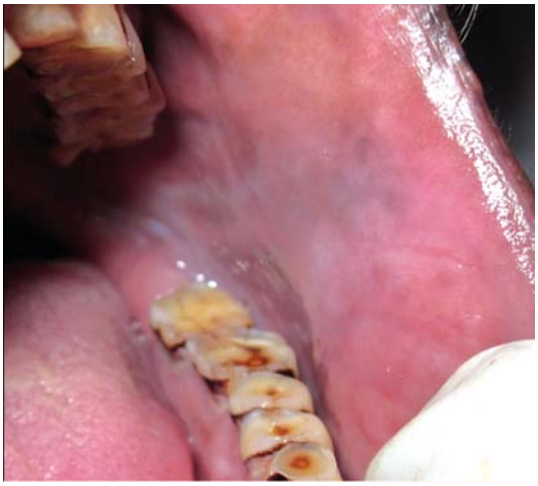
Linea alba, oral submucuos fibrosis, white sponge nevus, HBID, lichen planus, lichenoid drug reaction, cheek chewing, lupus erythematosus, candidiasis.

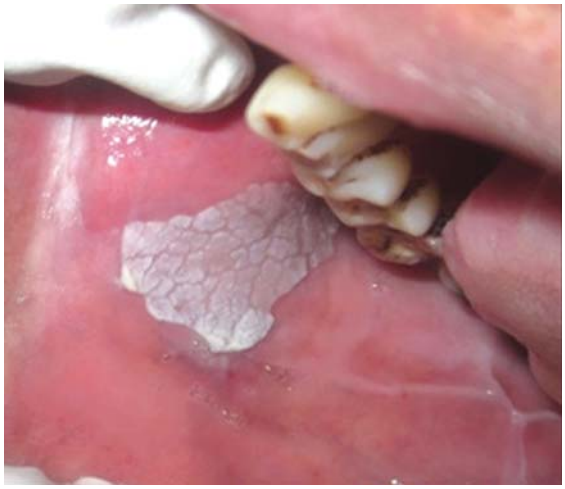
20. What is Witkop's disease?

It is rare, hereditary condition (autosomal dominant), it is also called hereditary benign intraepithelial dyskeratosis. Early onset of bulbar conjunctivitis and oral white lesions (usually first year of life). Oral lesions are soft, asymptomatic, white folds and plaques of spongy mucosa.

21. What is preleukoplakia?

A preleukoplakia definite entity with specific diagnostic criteria and behaviour, it is characterized by low-grade or mild reaction of mucosa, conceived as a precursor stage of leukoplakia. It is grey or greyish-white area but never completely white lesion with indistinct borders. It may have lobular pattern and distinct borders. It describes a diffuse white lesion of the oral mucosa, less dense and less marked than leukoplakia. It is strongly associated with tobacco smoking. This preleukoplakia terminology is been replaced by the terminology as thin, smooth leukoplakia. The prevalence in India varies from 0.5 to 4.1%. Approximately 15% of preleukoplakia progress to leukoplakia, 0.4% may progress to oral cancers. Malignant transformation may occur from preleukoplakia and may convert to leukoplakia and then to malignancy.



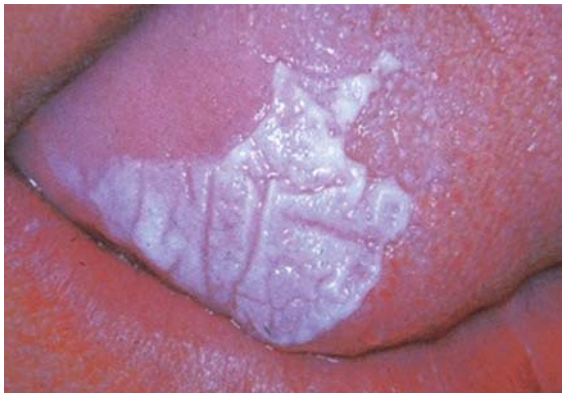


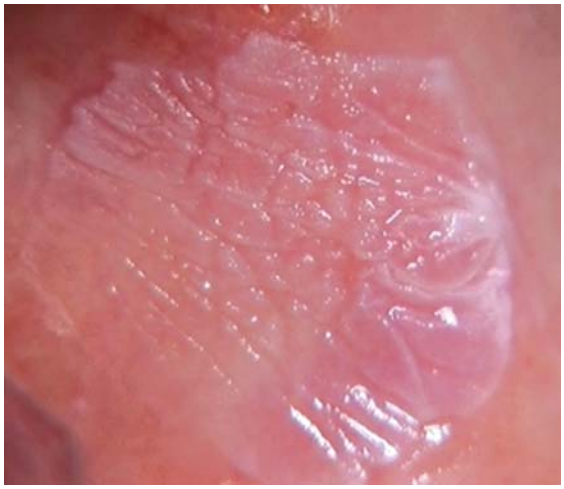
22. Define leukoplakia. What are the clinical types of leukoplakia?

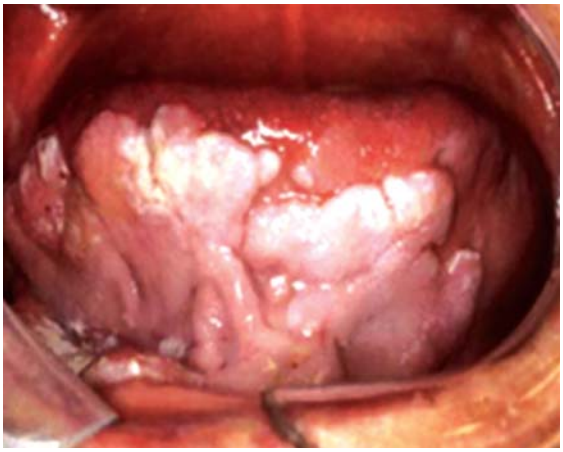
Leukoplakia is defined as a white patch or plaque that cannot be characterized clinically or histologically as any other lesion.

Types of leukoplakia

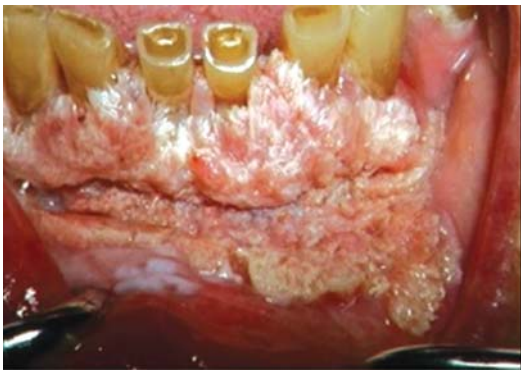
- **Homogeneous:** Uniformly white patch with slightly raised mucosa (cracked mud appearance)
- **Proliferative verrucous leukoplakia (PVL)** is an aggressive form of oral leukoplakia that is persistant and refractory to treatment with a high-risk of malignant transformation, begins as benign hyperkeratotic lesion and often becomes multifocal.

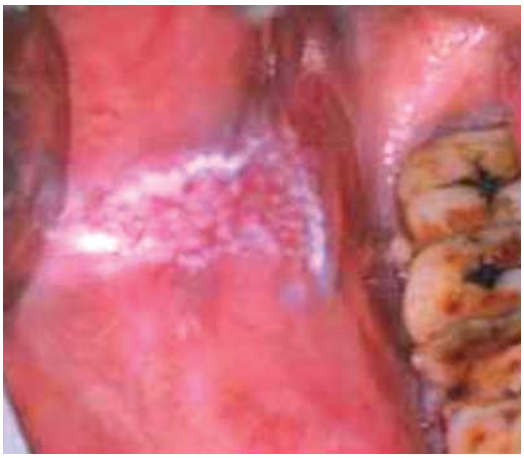


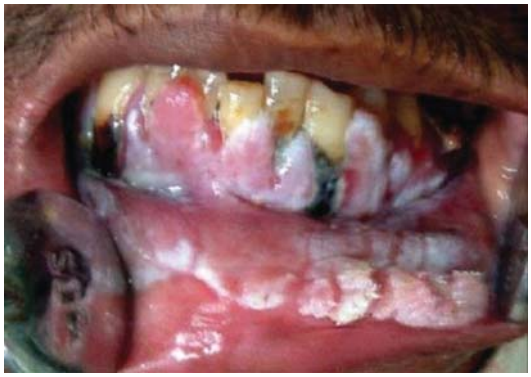


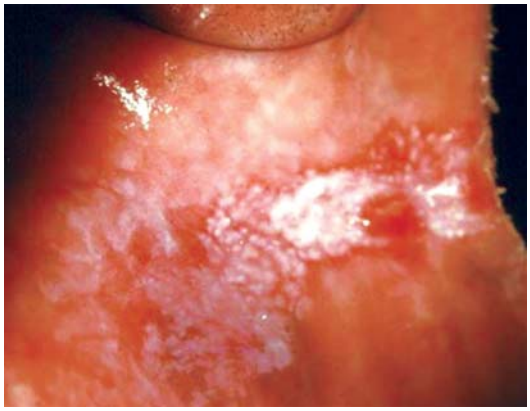


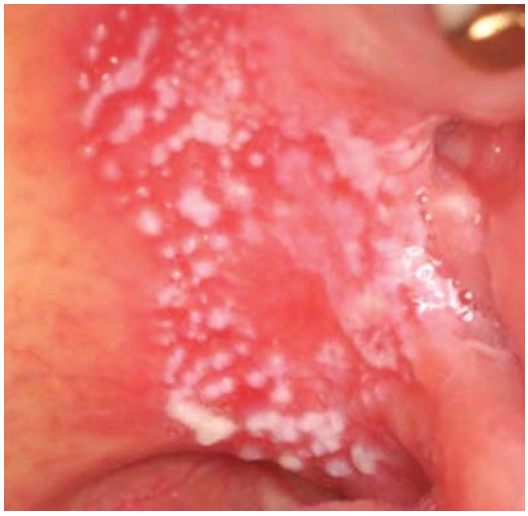
- **Nodular lesion:** White lesion with granular surface associated with candida infections.
- **Speckled leukoplakia:** Combined red and white lesions with irregular surface.

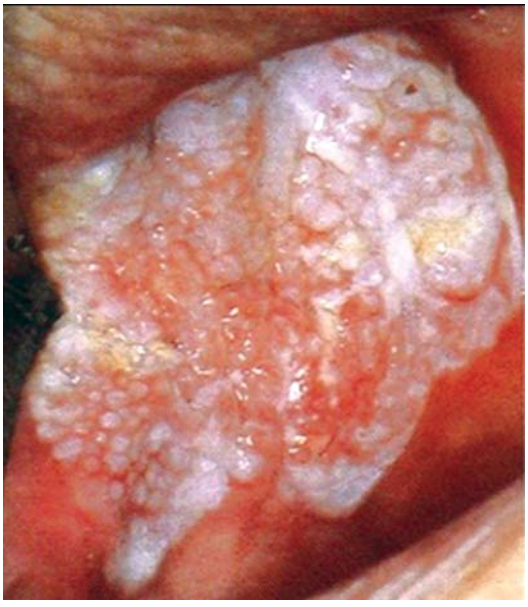












- **Reversible and irreversible leukoplakias:** Leukoplakia may also be divided into two types according to whether it spontaneously disappears after the chronic irritant has been eliminated. Lesions that disappear

are referred to as **reversible leukoplakias**, whereas the persistent lesions are termed **irreversible leukoplakias.**

23. Give classification of leukoplakia according to malignant change and prognosis.

In 2002 WHO has given this classification

- Phase I: Thin, smooth leukoplakia—better prognosis
- Phase II: Thick, fissured leukoplakia
- Phase III: Proliferative verrucous leukoplakia (PVL)—higher malignant transformation rate
- Phase IV: Erythroleukoplakia—poor prognosis.

24. Name the systemic condition associated with leukoplakia.

Dyskeratosis congenita, syphilis

25. What is stippled leukoplakia?

Leukoplakic lesions as a white patch with red dots of thin mucosa within it are called stippled leukoplakia. These are different from erythroleukoplakia as mixture of white and red patches.

26. What are thin, thick and granular leukoplakias?

Thin (early) leukoplakia is subtle white patch may show epithelial dysplasia on biopsy.

Thick leukoplakia is thick white lesion may show epithelial dysplasia.

Granular leukoplakia, a small leukoplakic lesion with a rough, granular surface. The biopsy may show dysplasia. Such a lesion would be easily missed during examination.

27. What are the variants of homogenous and non-homogenous leukoplakias?

Homogenous: Lesion that was uniformly white and unscrapable. Flat, corrugated, wrinkled, pumice like.

Non-homogenous: Lesion predominantly white and speckled with red verrucous, ulcerated, nodular, erythroleukoplakia.

28. Why leukoplakia of floor of mouth and ventrolateral surface of tongue are attributed to malignization?

The floor of mouth and ventrolateral tongue with extension back into the lateral soft palate and tonsillar area forms high-risk zone. There are two major factors that explain this as high-risk zone.

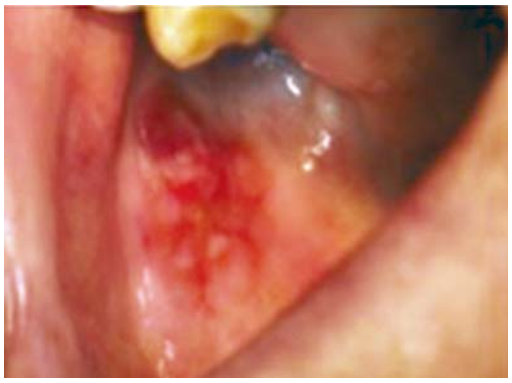
1. These sites are easily bathed by carcinogens, as any carcinogens will mix with saliva and get pooled in this region.
2. These lesions of mouth are covered by thinner, non-keratinized mucosa which offers less protection.

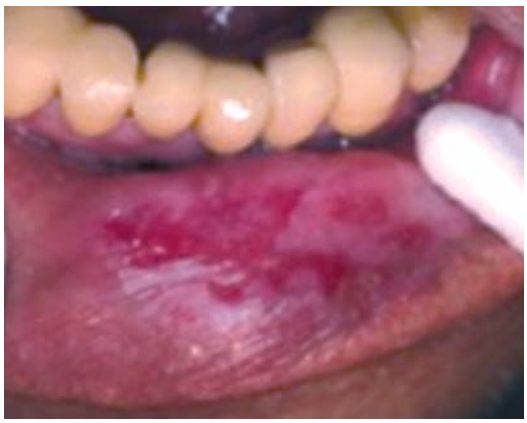
29. What are the high-risk leukoplakia?

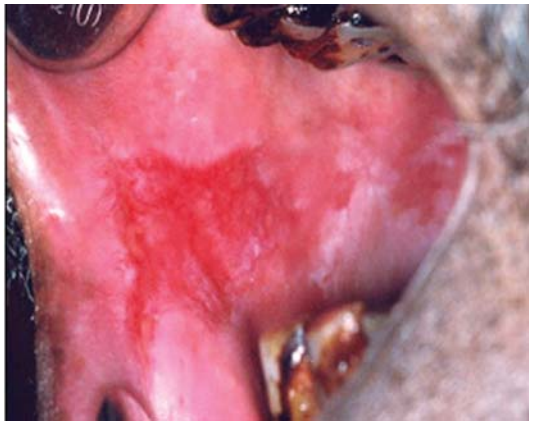
- Red component
- Raised component
- Presence in high-risk oval
- Tobacco and alcohol use
- Nonsmoker and unknown etiology of lesion
- Non-reversible type
- Microscopic atypia
- The malignant potential is low in homogenous leukoplakia, higher in verrucous leukoplakia and highest in speckled leukoplakia.

30. What is erythroplakia? What are the types of erythroplakia?

Erythroplakia can be defined as a persistent velvety red patch that cannot be identified as any other specific red lesion such as inflammatory erythemas or those produced by blood







vessel anomalies or infection. Three different clinical appearances were described by Shear

1. The *homogeneous form,* which is completely red in appearance,
2. *Patches* of EP and leukoplakia occurring together, and
3. *Speckled* EP, in which small leukoplakic specks are scattered over an area of EP.

31. Enumerate the tobacco associated lesions less likely to become cancers.

These lesions have not shown excess risk for malignant transformation.

1. Preleukoplakia
2. Leukoedema
3. Smokers palate
4. Palatal erythema
5. Palatal erythema with papillary hyperplasia,
6. Tobacco pouch keratosis (tobacco-lime users lesion)
7. Pan (betel quid) stain
8. Pan encrustration
9. Lichenoid reactions.

32. What is differentiating point between erythroplakia and pseudomembranous candidiasis?

Erythroplakia has a sharper border while lesions of acute and chronic pseudomembranous candidiasis has got diffuse border.

33. Define oral submucous fibrosis (OSF). Why is it classified as potentially malignant disorder?

It is defined as slowly progressive chronic fibrotic disese of oral cavity and oropharynx characterized by fibroelastic change and inflammation of the mucosa, leading to a progressive inability to open the mouth, swallow or speak.

Pinderberg (1972) given criteria that considered this as potentially malignant disorder:

1. Higher occurrence of OSF in oral cancer patients
2. Higher incidence of squamous cell carcinoma in patients with OSF
3. Histological diagnosis of cancer without any clinical suspicion in OSF
4. High frequency of epithelial dysplasia
5. Higher prevalence of leukoplakia among OSF.

34. What are the initial clinical symptoms and signs suggestive of oral submucous fibrosis?

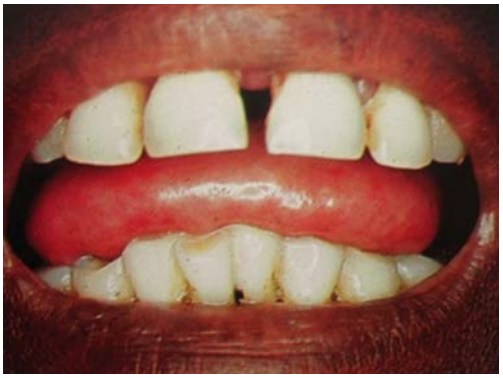
Symptoms: The most common feature of submucous fibrosis is burning sensation of mouth aggravated by spicy food (42%) followed by either hypersalivation or dryness of mouth (25%).

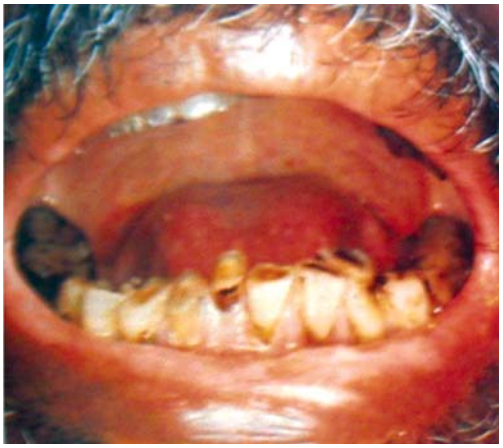
Signs: The common sign is blanching, i.e. marble-like appearance of oral mucosa. It may be localized, diffuse or reticular. The patient with localized blanching who chew only areca nut the incubation period is short, while betel quid chewers it is long. Reticular blanching (lace-like) consist of blanched areas with intervening, clinically normal mucosa giving

it lace-like appearance. Over the time one type of blanching may change to other type.

35. What is elliptical rima oris?

In oral submucous fibrosis when lips are involved, the connective tissue and muscle bands in the lips run around the rima oris like a thin band. In severe labial involvement, the opening of mouth is altered to an elliptical shape.

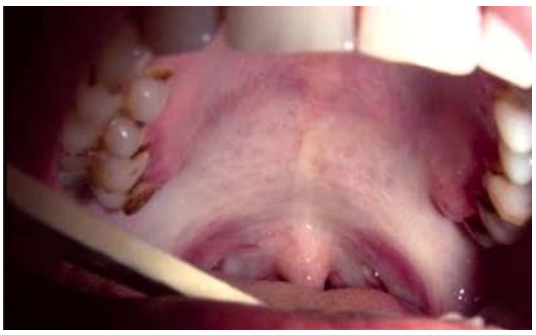




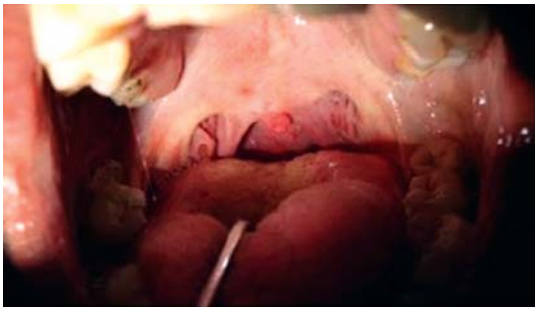
36. What is heavy curtain like appearance and hockey stick like appearance and bud-shaped uvula?

Involvement of soft palate in submucous fibrosis is marked by fibrotic change and a clear delineation of soft palate from the hard palate as if a "heavy curtain' is hanging from the hard palate.

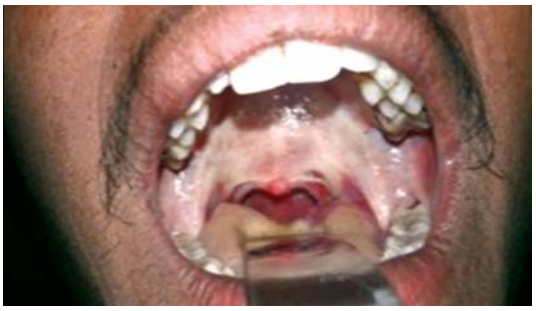
Hockey stick uvula: The uvula in submucous fibrosis is sunked and hooked up like a hockey

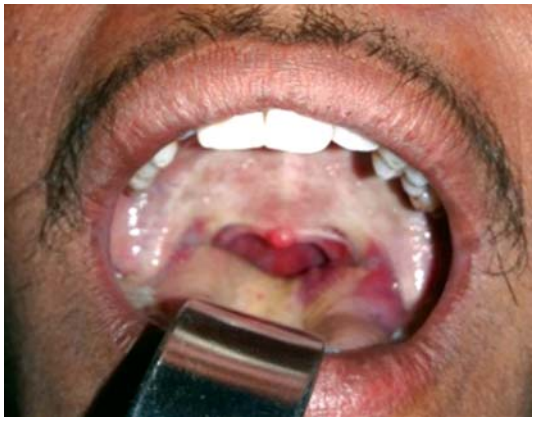


Heavy curtain like appearance



Heavy curtain and shrunken uvula appearance





Heavy curtain like appearance and bud-shaped uvula

stick due to fibrosis and is called hockey stick uvula (turned to one side).

Bud-shaped uvula: The uvula in submucous fibrosis is shrunken and small is called bud shaped (it is not tuned to one side).

37. Mention the conditions considered for differential diagnosis of oral submucous fibrosis.

In early stages anemia because of pale mucosa may be mistaken for blanching. In severe anemic condition, the oral mucosa is pale and hyperpigmented, the tongue is depapillated and buccal mucosa is coarse, the criteria of palpable bands is diagnostic.

Scleroderma (oral manifestation) is another entity to be considered. It is generalized. The occurrence of scleroderma is rare and rarer is still oral involvement. Sometimes the blanching is well-circumscribed, i.e. localised can be mistaken for leukoplakia in the absence of other features of oral submucous fibrosis.

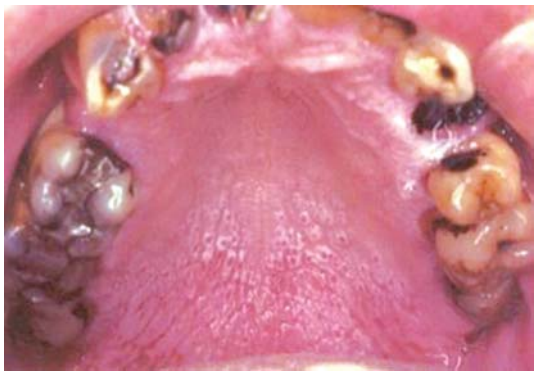
38. Name the various disorders of oral mucosa associated with smokeless tobacco.

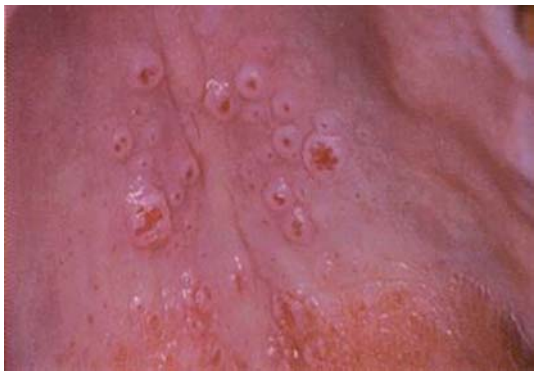
Excessive use of tobacco has been associated with several lesions in the oral cavity, which include tooth stain (brown to black mainly on lingual aspects of molars), tobacco-related blanching of mucosa seen in chewers (generally palate), palatal erosions, tobacco-induced pigmentation (greyish white), lichenoid type of reactions, sometimes areas of depigmentation intermingled with pigmentation. Gingivitis, gingival recession, periodontal conditions, acute necrotizing ulcerative gingivitis. Abrasions, tobacco excrescence, burns, hyperkeratotic lesions mainly on lateral border of tongue, hairy tongue, tobacco pouch keratosis and leukoplakia. Verrucous carcinoma if present shows adjacent tobacco pouch keratosis (ST mucosal lesions), squamous cell carcinoma and oral field cancerization.

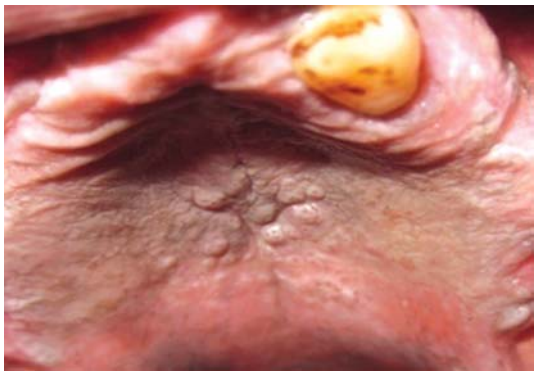
39. Name the lesions associated with tobacco smoking.

The lesions are as follows:

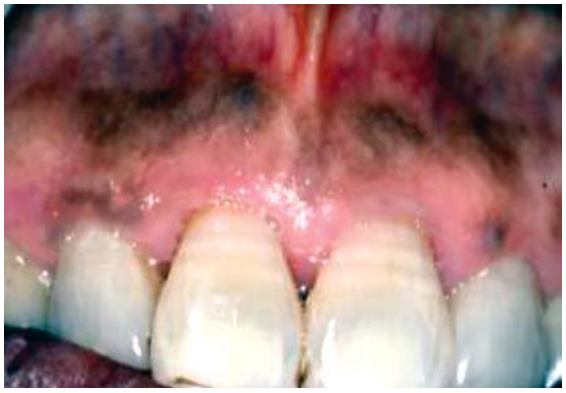
1. **Nicotinic stomatitits (smoker's palate):** Hard palate shows characteristic changes in the form of greyish-white to dramatic wrinkled, fissured surface texture, numerous erythematous spots may distributed or the diffuse blanching and the multinodular appearance of the hard palate. The orifices of the palatal minor salivary glands can be appreciated as minute, red spots.
2. **Palatal erythema associated with the palatal excrescences** in individuals in heavy smokers. The elevated red areas are the orifices of the palatal minor salivary glands.



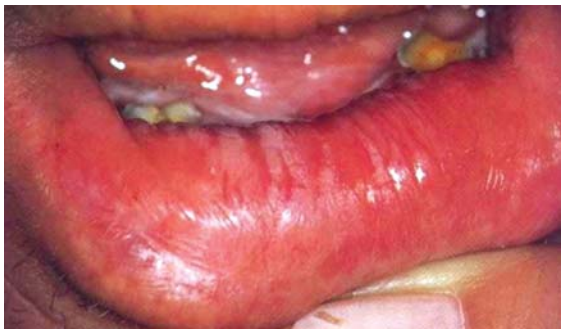




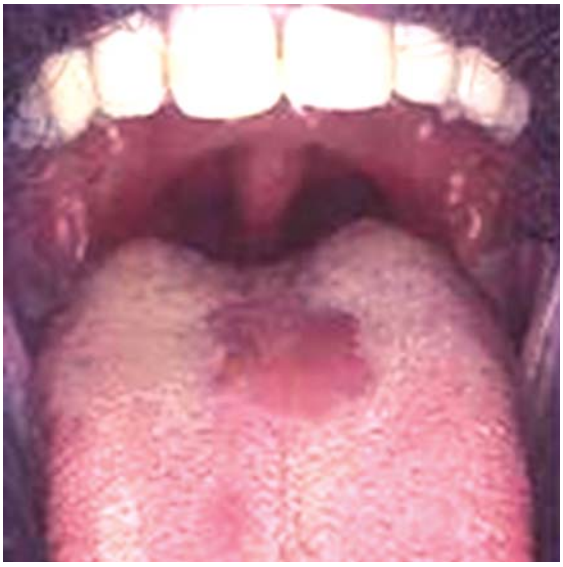
3. **Palatal white patches in reverse smoking:** The combustion product of tobacco and extreme heat responsible for palatal changes which range from white to erythematous patches.
4. **Smokers melanosis:** Common condition in dark-skinned ethinic group. It presents as diffuse, brown, pigmented patch. Anterior gingivae, buccal mucosa common site while tongue may be involved. Smoker's melanosis is caused by stimulation of melanin production by melanocytes due to chemical substances in cigarette smoke.



5. **Cigarette smoker's lip:** It is a localized, usually well-defined, flat or slightly elevated lesion of the lips that corresponds to the area where the patient holds cigarettes. The lesion usually begins as a reddened area but becomes whiter with time.



6. **Central papillary atrophy of the tongue:** This is noted in the bidi smokers. This is reduced spontaneously once the habit cessation occurs.



7. **Smoker's patch:** This condition describes altered condition of epithelium due to smoking, most of lesions are in the midline or one side. Patch or roughly oval-shaped area. It is small in size roughly oval in shape. The surface of patch may be smooth and not ulcerate or exociarated and afterwards it may become covered by yellowish material, sometimes red and crusted area, not tender.
8. **Preleukoplakia:** Greyish-white patch

40. What are the changes in palate due to smoking?

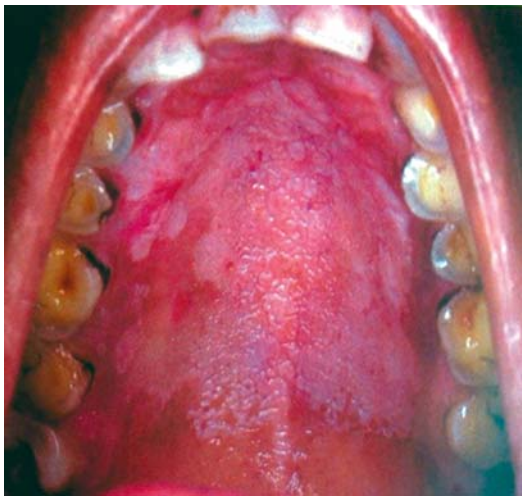
These changes are as follows:

1. **Keratosis:** Diffuse whitening of entire palatal mucosa.
2. **Excrescences:** 1–3 mm elevated nodules often with red spots which represent initial palatal reaction and are transient.
3. **Patches:** Well-defined elevated white plaques.
4. **Red areas:** Well-defined reddening of palatal mucosa.
5. **Ulcerated areas:** Crater-like areas covered by fibrin.
6. **Non-pigmented areas:** Palatal mucosa devoid of pigmentation.

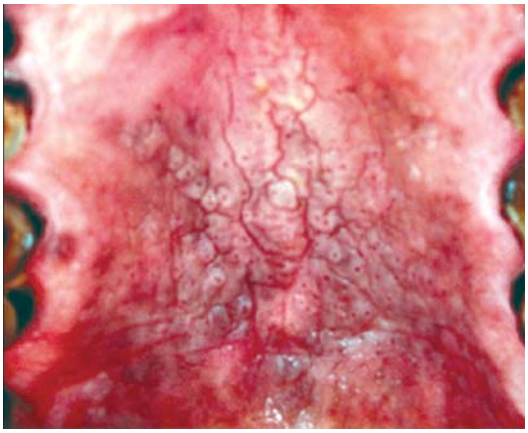
41. What are the palatal changes associated with reverse smoking?

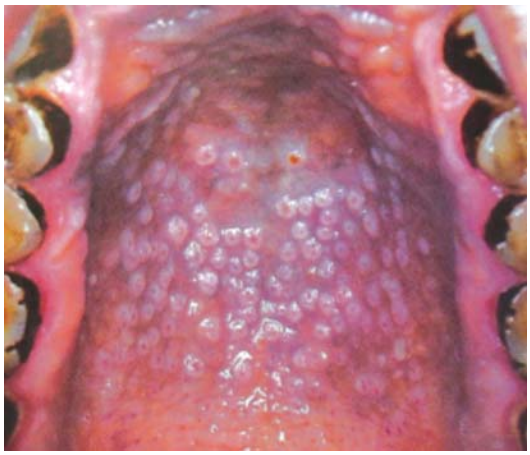
Palatal mucosal changes in reverse smokers were of varying degrees ranging from adaptive changes to potentially malignant lesions and ulcerations. The adaptive changes are hyperpigmentation and excrescence. Depigmented areas are the transition regions between the adaptive and potentially malignant lesions. Potentially malignant lesions were leukoplakia and erythroplakia.

Keratosis: May be independently or co-exist with other components. Account for 55% of component.

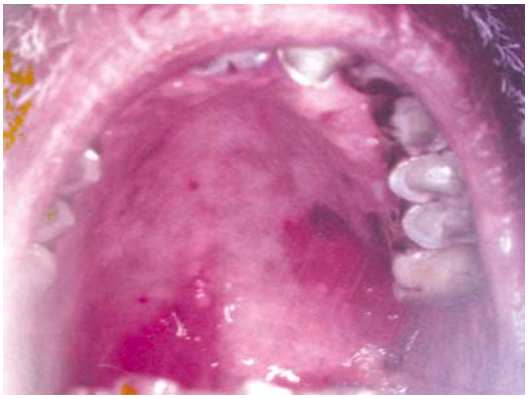


Excrescences: It is present in severe form in reverse smokers. Account 46% of palatal components.

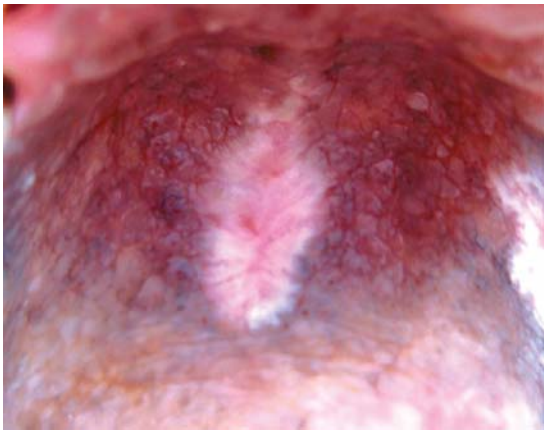


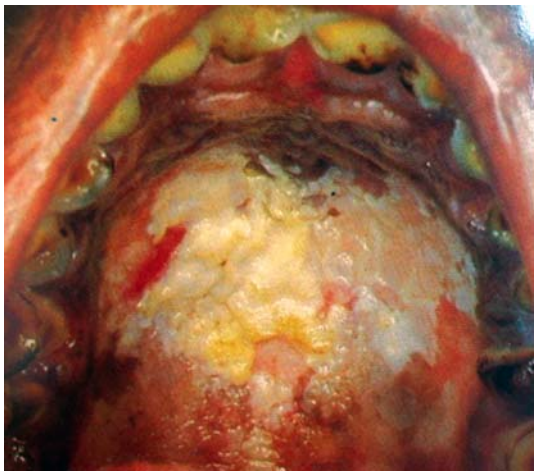


Reverse dhumti smoker's lesion: Mostly in Goa, lesion is less severe than by reverse chutta smoking.

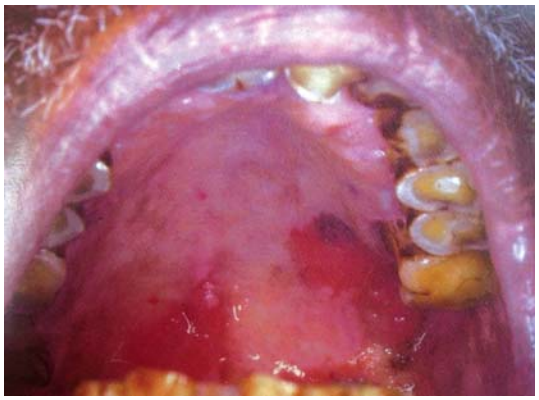


Patches: Same as leukoplakia, but differ histologically, account for 12% of component. Leukoplakia seen in areas of depigmentation.

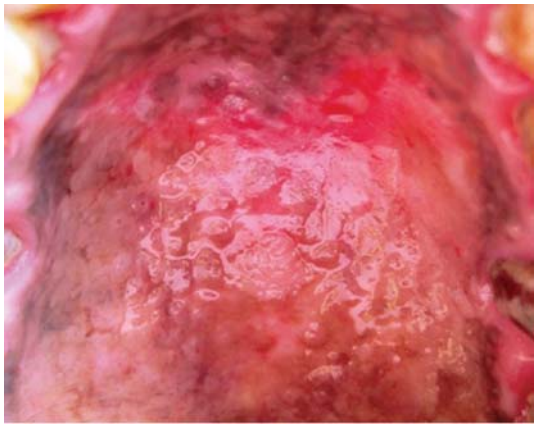


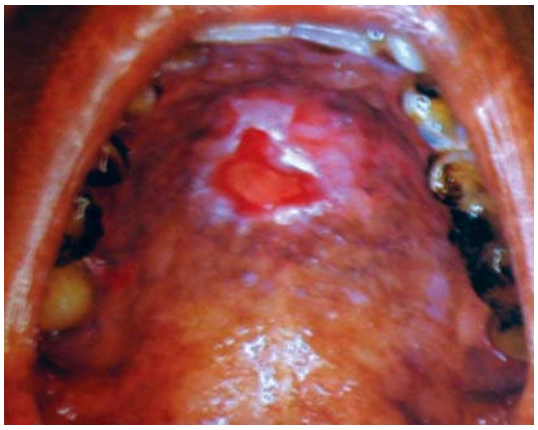


Red areas: Indistinguishable from erythroplakia and account for 2% of palatal components.

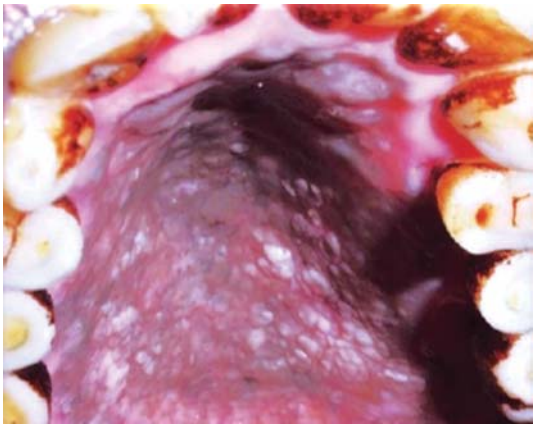


Ulcerated areas: They represent burn type of reaction, ulcers and account for 2% of palatal components.

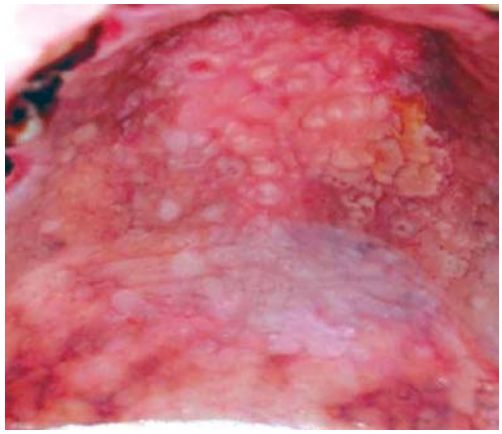




Hyperpigmentation: It presents melanin pigmentation in spotted, linear, patchy, diffuse and reticular types.

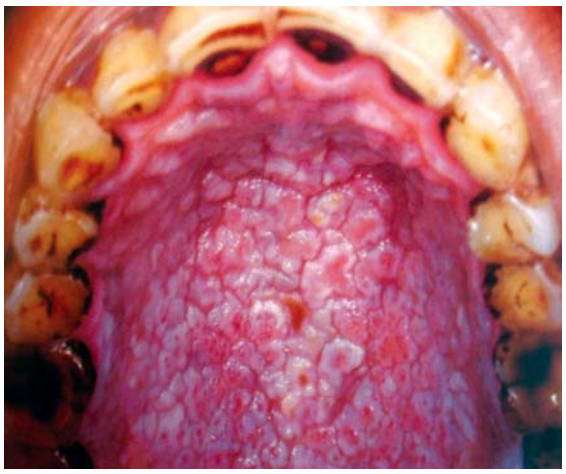


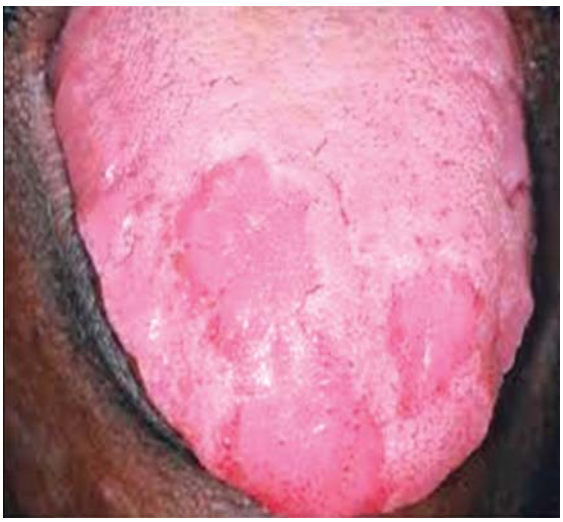
Non-pigmentated areas: Clinically devoid of melanin pigmentation and are surrounded by hyperpigmentation.

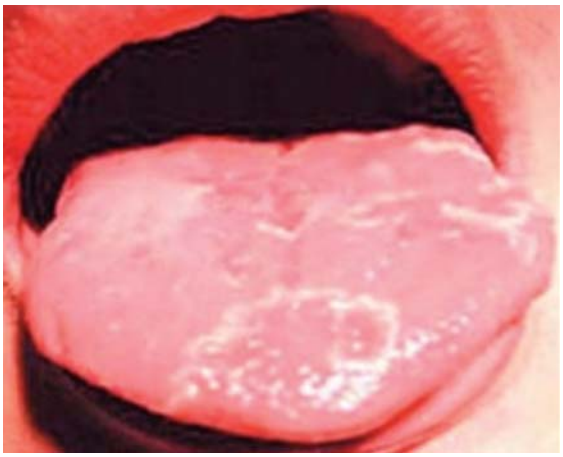


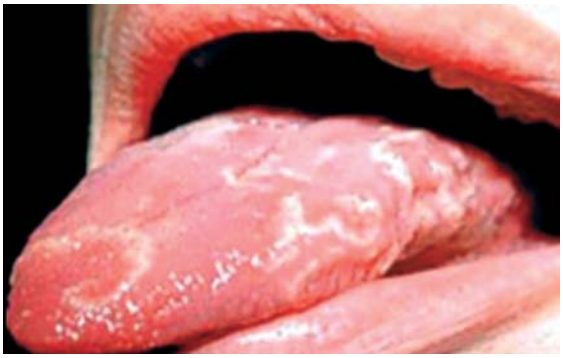
42. What are multimorphic lesions in palate due to smoking?

Keratosis and excrescences co-exist more frequently, then excrescences and patches, red areas and patches. The co-existent changes with non-palatal changes include leukoplakia on dorsum of tongue which is an otherwise uncommon location for it and may progress to malignancy (0.3%). This indicates that deleterious habit of reverse smoking may extend to other locations that are in close proximity to the lightened end of chutta.



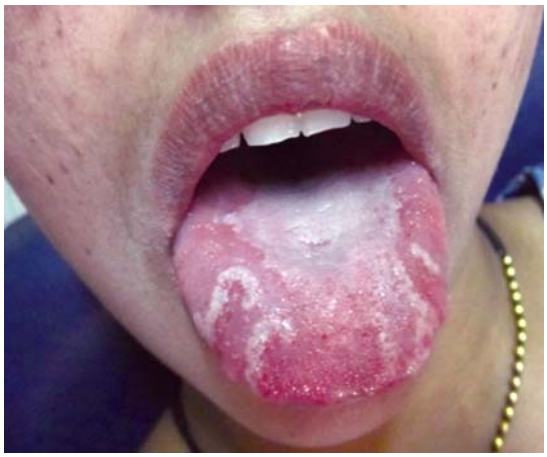






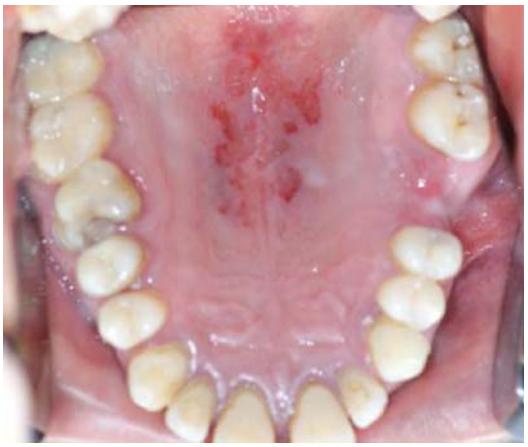
43. What is geographic tongue? What is etiology and types of it?

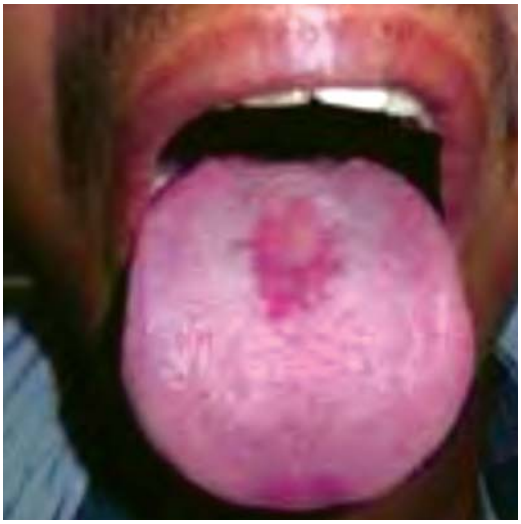
Benign migratory glossitis also known as geographic tongue is a recurrent condition of unknown etiology characterized by loss of epithelium particularly of the filiform papillae on the dorsum of the tongue. Clinically the appearance is of multifocal, circinate, irregular erythematous patches bounded by slightly elevated, white colored keratotic bands.

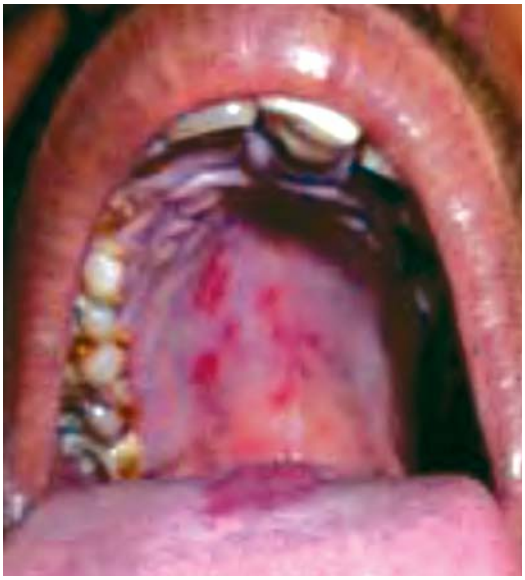


The etiology and pathogenesis remains obscure. Many risk factors have been proposed including hormonal disturbances, oral contraceptive use, juvenile diabetes mellitus, pustular psoriasis, allergic conditions such as atopy, hay fever and rhinitis, fissured tongue, Robinow's syndrome, Reiter's syndrome, Down's syndrome, psychological factors, nutritional deficiencies, lithium therapy, familial predisposition, fetal hydantoin syndrome and Aarskog's syndrome.

- **Type I:** Lesions confined to tongue in both active and remission phases. No other lesions in oral cavity.
- **Type II:** Same as type I with similar lesions in other areas of oral cavity.
- **Type III:** Lesions on the tongue that are not typical and accompanied by lesions in other areas of oral cavity. It has got two forms fixed and abortive.
  - ***Fixed:*** A few areas of tongue affected, no movements of lesion seen. They may disappear and reappear on the same location.
  - ***Aborative:*** The initial lesion is yellowish-white patches. These disappear before acquiring the typical appearance of geographic tongue.
- **Type IV:** No tongue lesions but the migratory lesions are present elsewhere in the oral cavity (erythema circinata).

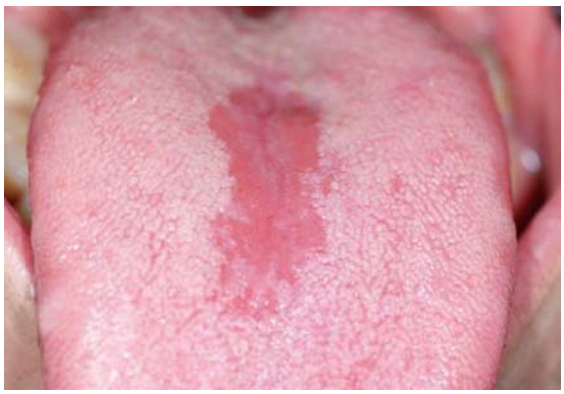






44. What is kissing lesion?

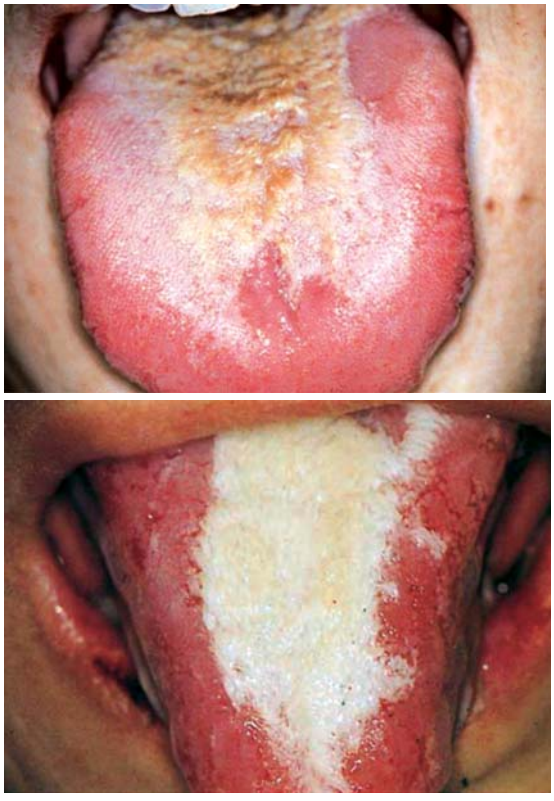
Median rhomboid glossitis (MRG) is concomitant with a palatal inflammation corresponding to contact with the involved area on



the tongue, it is called 'kissing lesion' and this finding may suggest the prolonged contact between the Candida-infected dorsum of the tongue and the hard palate. In these patients immunosuppression should be suspected and investigated and it has been considered as a marker of HIV infection.

45. What is furred tongue?

It is also called hairy tongue. It is defined as thickened surface of tongue with color change from black to white. It results from hypertrophy of filiform papillae and the desquamated epithelium is trapped into it resulting in large plaque. It is seen in febrile illness and smokers. The patients with soft diet show furred tongue and the patients who are not having diet high in roughage and fibre. The associated findings are xerostomia and palatitis nicotina in mouth breathers and smokers respectively. Proper brushing and diet modification are necessary.

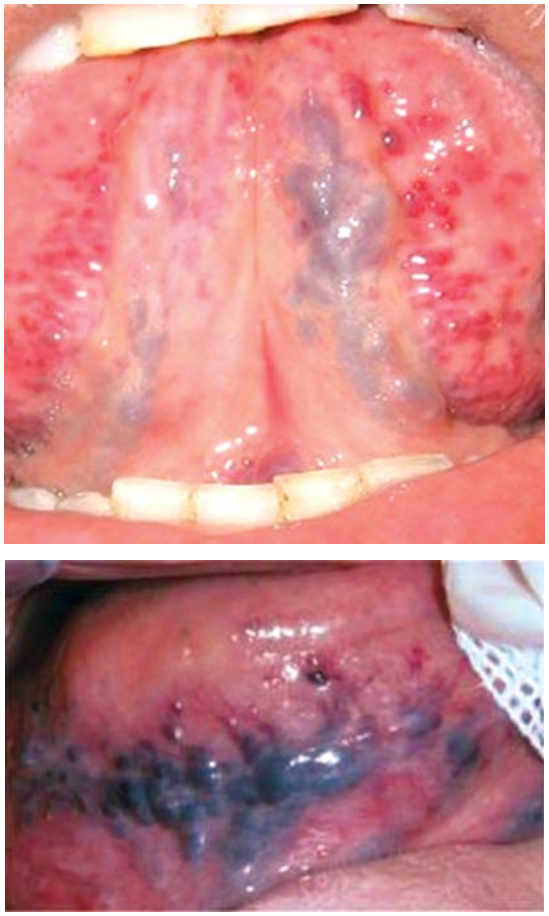


Hairy tongue and furred tongue

Some authors quote that furred tongue is an uncommon disease which is occurring during febrile diseases appear as thick or thickish white yellow coating on dorsal surface of tongue. This lesion is due to lengthening of filiform papillae by 3–4 mm and accumulation of food debris and bacteria. It disappears shortly.

46. What are the varices of tongue?

It is also called sublingual varices. It is defined as tortuous veins undersurface of tongue bilaterally. The other presentation is multiple blue purple or elevated pappular blebs on ventral/lateral surface of tongue. The reason is vasodilation and venous ectasia with aging, which is due to loss of connective tissue tone supporting vessels. Occasionally, a single varix may be noted with soft purple papule



which intends with firm palpation. Lips and buccal mucosa are the other areas where it is present. The lesions are asymptomatic except where secondary thrombosis occurs.

47. What is cheilocandidiasis?

A diffuse form of chronic candidiasis characterized by pain, swelling, erythema with focal ulcerations, and crusting is called cheilocandidiasis. It represents a secondary candidal infection superimposed on areas of trauma from mechanical or solar factors.

48. What is superficial and deep fungal infections?

Superficial fungal infections have an affinity for keratin and epidermis and adnexal structures. These usually have a characteristic clinical finding (none to a painful burning sensation causing dysphagia) and diagnosis is done on clinical findings, e.g. candidiasis. These are managed with topical antifungal agents. The superficial infections have incubation period relatively short, the onset of disease is sudden and the symptoms are initially severe but decrease in severity with time, so that spontaneous healing may occur.

Deep fungal infections are less common and affect deeper structures, internal organs. These affect sites other than oral cavity and may be an indicator of systemic diseases. These are commonly noted in immunosuppressed individuals (HIV, AIDS and malignancies). These are more dangerous than superficial. Diagnosis is confirmed by biopsy, e.g. aspergillosis, histoplasmosis deep infections, on the other hand, have a protracted incubation period, the symptoms are insidious in their onset, and the course of disease becomes increasingly severe.

49. What is opportunistic fungal infections?

The opportunistic fungal infections are those which affect mostly immunocompromised patients and debilited patients (AIDS, leukemia/lymphoma, particularly during chemotherapy and in patients who are receiving immunosuppressive agents and broad spectrum antibiotics), e.g. mucormycosis and aspergillosis. Other risk factors are organ transplants patients, variety of systemic/immunological disorders (systemic lupus erythematosus), alcohol/IV drug abuse. Recipients of previous treatment with corticosteroids, cytotoxic agents, prolonged antibiotic therapy.

50. What are types of oral candidiasis?

These are primary and secondary.

Primary infections are limited to oral and perioral sites, e.g. acute erythematous, acute pseudomembranous, chronic erythematous, chronic psuedomembranous and chronic hyperplastic, chronic plaque like, and nodular candidiasis. Candida associated lesions are denture stomatitis, angular cheilitis, and median rhomboid glossitis.

Secondary infections are accompanied by systemic mucocutaneous manifestations, e.g. familial chronic mucocutaneous candidiasis, diffuse chronic mucocutaneous candidiasis, candidiasis endocrinopathy syndrome, familial mucocutaneous candidiasis, severe combined immunodeficiency, DiGeorge syndrome, chronic granulomatous disease and AIDS.

51. Which is the painful candidiasis type?

Acute atrophic or erythematosus variety.

52. Which type of candidiasis may show malignant transformation?

Hyperplastic type which is refractory to treatment.

53. What are the host factors associated with candidiasis?

These factors are divided into local and systemic in nature.

Local causes are inhaled corticosteroids cause erythematous candidiasis. They suppress localized cellular immunity and phagocytosis promotes establishment of candida. Impaired salivary flow, as salivary flow serves to dilute and remove potential pathogenic microorganisms.

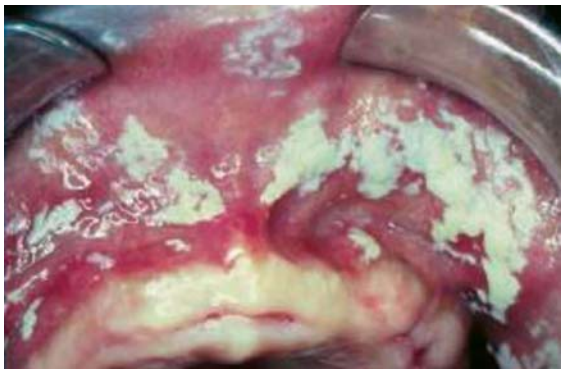
Systemic causes are medications causing xerostomia antihistaminics, tricyclic antidepressants, some antihypertensives, hypnotics and sedatives. Broad spectrum antibiotics increase susceptibility to Candida infection by altering local flora that naturally inhibit candidal growth. Other medications are antineoplastic agents, immunosuppressive agents (azathioprine and glucocorticoids), anticholinergic medications. Smoking, DM, endocrinopathies, immunosuppressive conditions, malignancies and nutritional disorders.

54. Enumerate various drugs used for treatment of fungal infections.

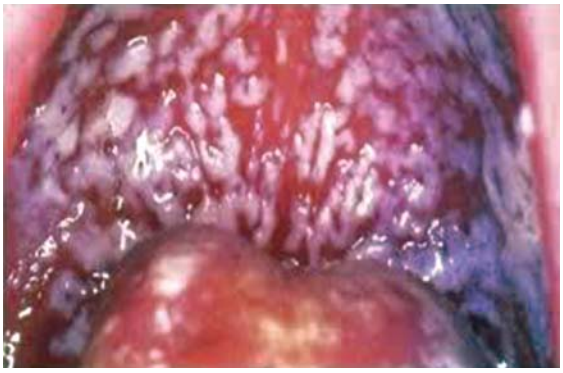
1. Azoles-imidazoles (clotrimazole, miconazole, ketoconazole), triazoles (fluconazole, itraconazole)
2. Polyenes (amphotericin and nystatin)
3. Echinocandins (caspofungin)
4. Pyrimidines (flucytosine)

55. What is curdled milk appearance and reddened bald appearance?

Oral lesions of pseudomembranous candidiasis (thrush). It is characterized by the presence of adherent white plaques. This is also called cottage cheese appearance. These can be easily wiped off leaving either erythematous area or normal mucosa which can easily bleed. It is often seen in baby's mouth or lips. It occurs in newborns and infants. It may be passed from mother to baby if the delivering mother has yeast infection.

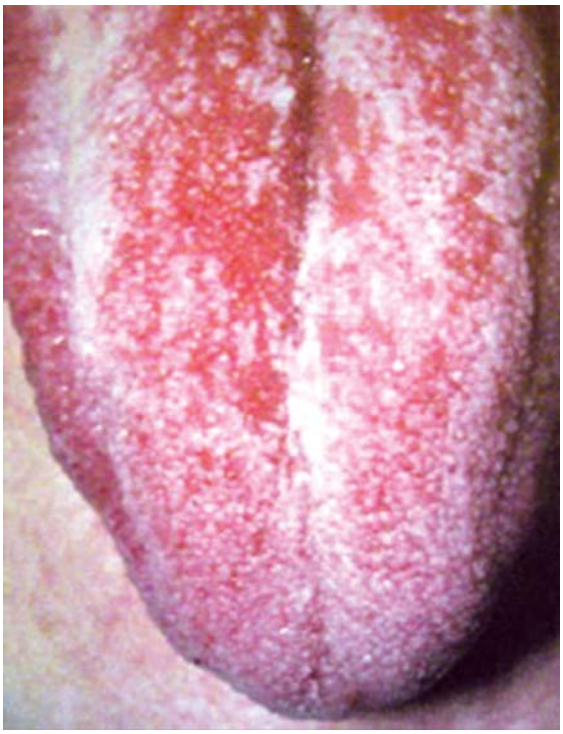


This is also called cottage cheese appearance (curdled milk appearance)



Cottage cheese appearance

Reddened bald appearance: This is seen in erythematous candidiasis. It shows painful reddened mucosa with a little or no white component. In acute form the dorsal surface of tongue will usually show diffuse loss of filiform papillae resulting in reddened bald appearance. This is also accompanied by burning sensation. It typically follows a course of broad spectrum antibiotics. Patient often complains that mouth feels as if hot beverage has scalded it.



56. What is id reaction?

Occasionally, a papular/pustular widespread cutaneous eruption appears (occasionally vesicles) after the commencement of systemic antifungal treatment—this is so called id reaction. This is immunological response (autosensitization) and not an adverse drug reaction. It is a distant skin manifestation of an established fungal infection. The most common location is on trunk and extremities, even on palms and fingers the lesions are seen. No specific therapy is required.

57. Enumerate the differentiating points between hemangioma and vascular malformation.

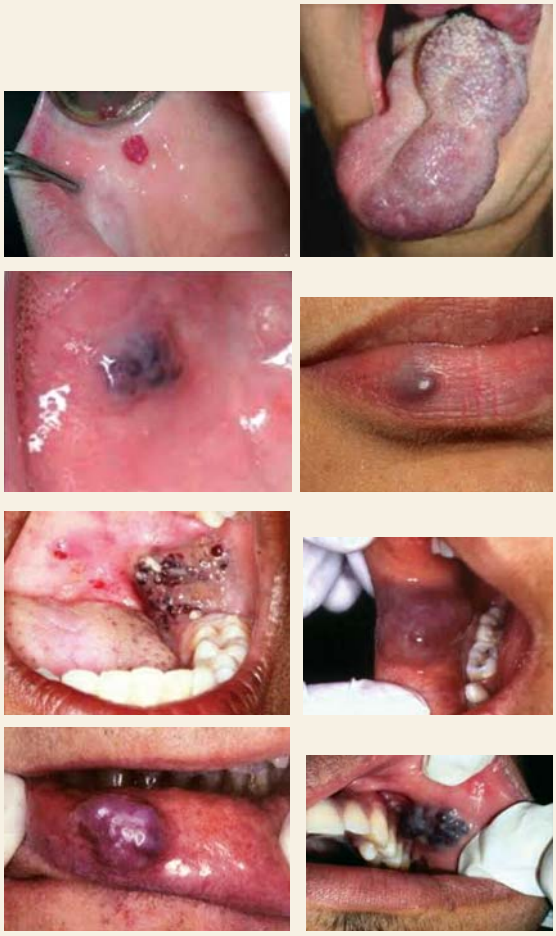
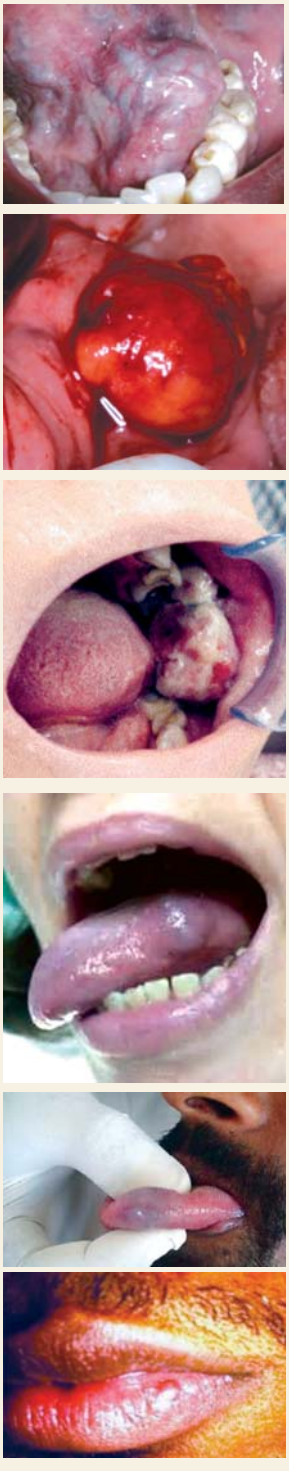
Differentiating points between hemangioma and vascular malformation is shown in Table 1.1.

Table 1.1: Characteristic differences between hemangioma and vascular malformation

	Hemangioma	*Vascular malformations*
Definition	**Hemangiomas** are noncancerous growths that form due to an abnormal collection of blood vessels.	"Vascular malformation" is a generalized term used to describe a group of lesions, present at birth, formed by an anomaly of angiovascular or lymphovascular structures
Description	Proliferation of abnormal endothelial cell	Development of abnormal blood vessels occurs
Elements	Capillaries are increased in numbers	A mix of arteries, veins and capillaries (A-V shunt)
Growth pattern	Congenitally rapid growth	It grows with patient
F:M ratio	3–5:1 and severe cases 7:1	1:1
Boundaries	Often circumscribed	Poorly circumscribed
Bone involvement	Rarely affects bone (distortion but no invasion)	May affect bone (distortion, thinning, underdevelopment, hypertrophy, invasion, destruction and rarely lytic lesions)
The vibrations and abnormal sounds	Not associated with thrill or bruit	May be associated with thrill or bruit
Involution	Spontaneous involution is possible	Does not involute
Radiology	Doppler sonography: Fast flow lesion MRI—tumoral mass with low voids Arteriogram—lobular tumor	Doppler ultrasonography: Slow-fast flow MRI—hyperintense signal (slow) Flow-voids (fast) Arteriogram—A-V shunting
Immunohistochemistry	High expression of PCNA, VEGF	Lack of expression
Hematology	No coagulopathy	Risk of bleeding (disseminated intravascular coagulation)
Resection	Persistent lesions are resectable	Difficult to resect; surgical hemorrhage
Recurrence	Recurrence uncommon	Recurrence common

Contd...

Table 1.1: Characteristic differences between hemangioma and vascular malformation *(Contd.)*

	Hemangioma	***Vascular malformations***
Clinical presentation		

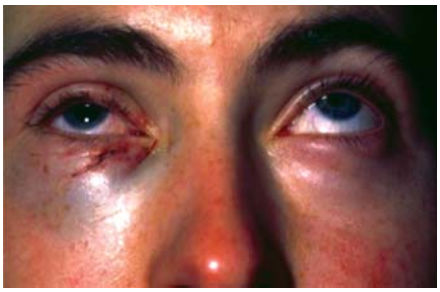
58. What are the complications of AVH?

The two most common AVH-related emergencies that the oral and maxillofacial specialist may need to manage are a life-threatening bleed and a rapid proliferation of the AVH that may obstruct the airway.

59. Enumerate the extravascular blood lesions.

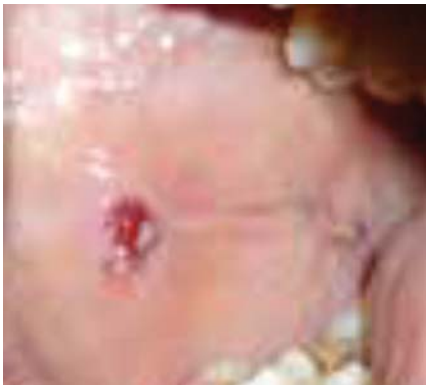
Extravascular blood lesions are due to the presence of blood outside of blood vessels. They do not blanch and typically resolve within a month. The patient often has a history of trauma or bleeding problem.

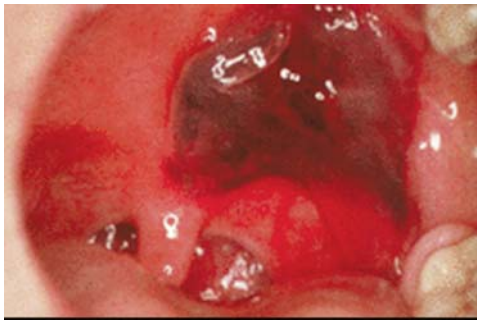
Ecchymosis is a bruise. It occurs due to hemorrhage and accumulation of blood in the connective tissue. It is usually the result of trauma, but may also be secondary to deficiency of platelets and/or clotting factors and viral infections. An ecchymosis is typically flat and red, purple, or blue in color. If the ecchymosis is due to trauma, then it will resolve spontaneously and no treatment is necessary. If it is secondary to a systemic disease, then further work-up is indicated.

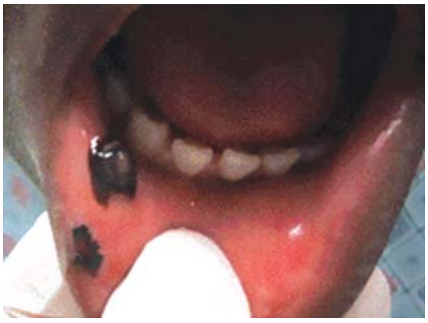


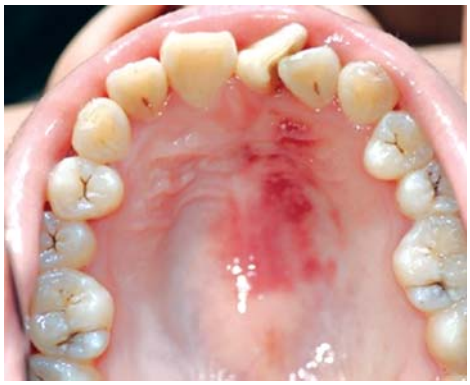
Ecchymosis on soft palate and on hard palate, subconjunctival ecchymosis

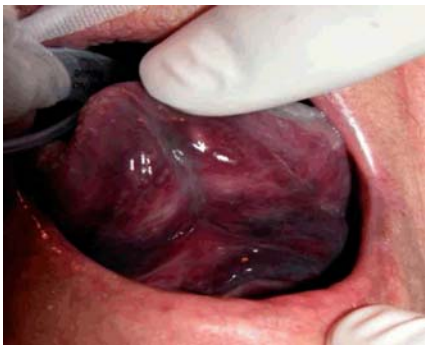
A **hematoma** is the result of hemorrhage with pooling of blood in the connective tissue. A







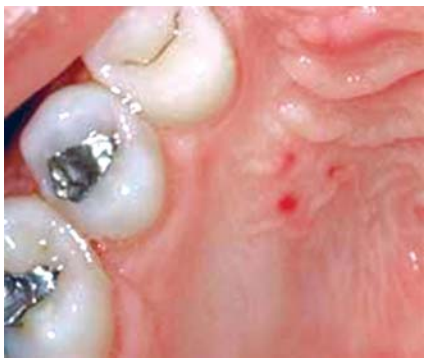


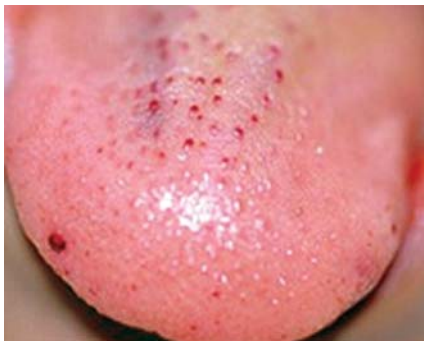


Hematoma on right buccal mucosa

hematoma causes thickening or enlargement of the mucosa. It is purple to black in color. No treatment is necessary once a diagnosis is made. A hematoma will resolve spontaneously in several weeks to over a month.

Petechiae are round, red, pinpoint areas of hemorrhage. Petechiae are usually caused by trauma, viral infection, or a bleeding problem. They resolve over a few weeks. Petechiae do not require treatment. Investigation of the cause of petechiae may be indicated.





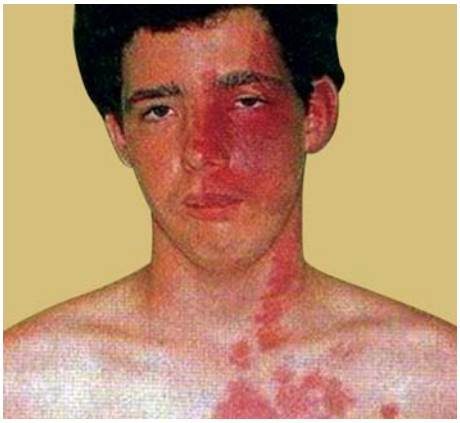
60. Name syndromes associated with cavernous hemangioma.

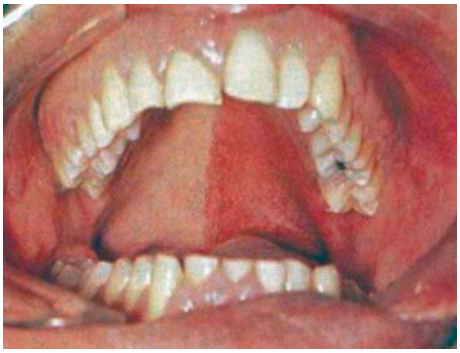
1. ***Kasabach-Merritt syndrome:*** In this syndrome, a large cavernous or arteriovenous hemangioma is complicated by thrombocytopenic purpura.
2. ***Maffucci's syndrome:*** Multiple hemangiomas and enchondromas.
3. ***Blue-rubber bleb nevus syndromes:*** This rare syndrome, an autosomal-dominant trait, produces a peculiar blue hemangioma that has the appearance and texture of a rubber nipple or rubber skin. Identical lesions appear in various locations in the gastrointestinal tract, primarily the small intestines.

61. What are angiomatous syndromes? Enumerate it.

These are associated with vascular malformations. The list includes:

1. ***Hereditary hemorrhagic telangiectasia (Osler-Weber-Rendu syndrome):*** It is an uncommon autosomal dominant disease. This multisystem disorder can affect the nose, skin, gastrointestinal tract, lungs, liver and brain. Epitaxis is the most common presenting problem. Patients with HHT have abnormal blood vessel development that manifests as telangiectasias and arteriovenous malformations (AVMs).
2. ***Blue rubber bleb nevus syndrome (BRBNS):*** It is a rare vascular anomaly syndrome consisting of multifocal venous malformations (VM). The malformations are most prominent in the skin, soft tissues, and gastrointestinal (GI) tract, but may occur in any tissue. These patients develop anemia and requires lifelong supplementation of iron and blood transfusions.
3. ***Bannayan-Zonana syndrome:*** It is a rare hamartomatous disorder, characterized by macrocephaly, multiple lipomas, and hemangiomas. Inheritance is by autosomal dominant transmission with male predominance.
4. ***Sturge-Weber syndrome (encephalotrigeminal angiomatosis):*** It has a vast spectrum of cutaneous, neurologic and ophthalmic manifestations which may or may not be associated with one another. The oral manifestations include ipsilateral port-wine stains of oral mucosa along with the hypervascular changes and angiomatous lesion of gingiva. Gingival hyperplasia can also be attributed to anticonvulsant medication and secondary to poor oral hygiene in mentally retarded patients. Macroglossia and maxillary bone hypertrophy have also been reported in a few cases.





5. ***Klippel-Trénaunay syndrome (Klippel-Trénaunay-Weber syndrome; Angio-osteo-hypertrophy):*** It is characterized by the triad of vascular malformation (capillary hemangioma or port wine stain), venous varicosity and soft tissue and/or bony hypertrophy.
6. ***Servelle-Martorell syndrome:*** It is a congenital vascular malformation associated with soft tissue hypertrophy and bony hypoplasia.
7. ***von Hippel-Lindau syndrome (vHL syndrome):*** It is caused by a gene mutation which frequently induces both non-malignant tumors and malignant tumors (or cancers) that can spread to other organs (become metastatic). Many of the tumors may involve abnormal growth of blood vessels.
8. ***Maffucci's syndrome:*** This syndrome is non-hereditary and is characterized by multiple enchondromas and hemangiomas.

62. What is acquired hemangioma?

A majority of hemangiomas are congenital, but some are acquired later in life. Some of the acquired capillary hemangiomas of the oral cavity may develop from infantile hemangiomas (IH) mostly on the gingivae. The conditions may be right for certain IH lesions with many patent capillaries to develop significant blood flow during the IH stage.

Such capillary systems remain after the irritant has been eliminated and the inflammation subsides. The resultant lesion is usually nodular and bluish-red, usually bleeds easily, and may blanch on pressure. Indicated treatment is sclerosis, excision, or perhaps a combination of these modalities after determination of the blood supply to the lesion.

63. Why central hemangiomas are called great imitators?

These lesions of bone have been referred to as *the great imitators* because they can produce so many different radiographic images. It is not pathognomonic and can stimulate many other lesions. Worth et al. have prepared an excellent and thorough review of the various radiographic appearances.

In about 50% of cases a multilocular appearance can be detected, small (honeycomb) and large (soap bubble) loculations.

Another form these lesions can take reveals coarse, linear trabeculae that appear to radiate from an approximate centre of the lesion. Small, angular, linear trabeculae of varying shapes are seen; however, the general outline is round (cartwheel appearance) and sometimes the trabaculae are right angles to surface (sunburst appearance).

A third appearance that may be observed is a cyst-like radiolucency with an empty cavity and sometimes a hyperostotic border.

The radiographic margins of these images may be well or poorly defined. Resorption of roots of the involved teeth occurs with some frequency, and calcifications (phleboliths) appearing as radiopaque rings are occasionally seen.

Central hemangioma is a great mimicker, as it resembles:

1. Osteosarcoma;
2. Fibrous dysplasia;
3. Central giant cell granuloma;
4. Ameloblastoma;
5. Multiple myeloma;
6. Dentigerous cyst; and
7. Odontogenic cyst radiographically. It also may clinically mimic: (i) A central arteriovenous fistula; (ii) Aneurysms; or (iii) A shunt.

64. What is erosion and ulcer?

An erosion has been defined as a shallow crater in the epithelial surface that appears on clinical examination as a very shallow erythematous area and implies only superficial damage.

Ulcer has been defined as a deeper crater that extends through the entire thickness of surface epithelium and involves the underlying connective tissue and associated with underlying molecular necrosis.

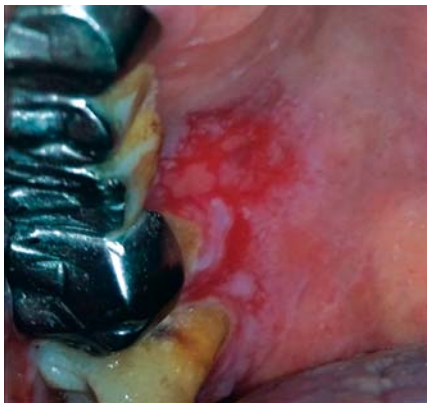
65. What are short-term and persistent ulcers?

Short-term ulcers (those that persist no longer than 3 weeks and regress spontaneously or as a result of non-surgical treatment). The majority of traumatic ulcers, recurrent aphthous ulcers (RAUs) (except major aphthae), recurrent intraoral herpetic ulcers, and chancres fall into the category of short-term ulcers, and persistent ulcers (those that last for weeks and months).

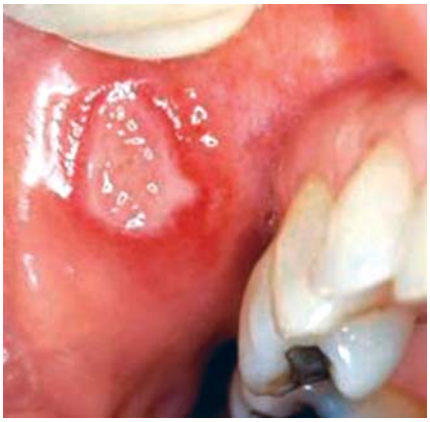
Occasionally, traumatic ulcers, major aphthae, and ulcers from odontogenic infection, malignant ulcers, gummas, and ulcers secondary to debilitating systemic disease are classified as persistent ulcers and may remain for months and even years. Persistent ulcers should be considered malignant until proved otherwise.

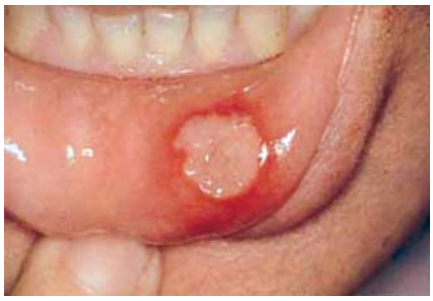
66. Name the conditions causing recurrent ulcers.

Recurrent aphthous stomatitis (RAS), recurrent-intraoral herpes simplex (RIHS), major aphthous ulcer (major AU), and herpetiform aphtha (HA), recurrent erythema multiforme, Behçet's syndrome.



Erosion on palatal gingival





Ulcer on left buccal mucosa and lower lip

67. Enumerate the syndromes associated with aphthous stomatitis.

Behçet's syndrome: It is characterized by recurring oral ulcers, recurring genital ulcers and eye lesions.

Magic syndrome: Mouth and genital ulcers and polychondritis.

Marshall's syndrome (PFAPA): Periodic fever, aphthous stomatitis, pharyngitis and cervical adenitis.

HIV/AIDS: Recurrent aphthous stomatitis (RAS) is one of the common oral manifestations of HIV/AIDS. In this condition, the ulcers are similar to those of non-infected group but are long lasting and are less responsive to routine medications. In many patients, major aphthae are found and are associated with advanced HIV infection (CD4+ counts less than $50/mm^3$) which suggests that immune compromise is a factor predisposing to aphthous stomatitis.

Sweet syndrome (SS): It is characterized by a constellation of clinical symptoms, physical features, and pathologic findings which include fever, neutrophilia, tender erythematous skin lesions (papules, nodules, and plaques), and a diffuse infiltrate consisting predominantly of mature neutrophils that are typically located in the upper dermis. Sweet syndrome is classified in three main types: Classical, paraneoplastic and drug induced. Classical type is more common in women between the ages of 30 and 50 years, is often preceded by upper respiratory tract infection and may be associated with inflammatory bowel disease and pregnancy. The paraneoplastic type is associated commonly with hematogenous malignancy mainly acute myelogenous leukemia. The commonly associated solid tumors are those of genitourinary organs, breast and of gastrointestinal tract. Drug-induced Sweet syndrome most commonly occurs in patients who have been treated with granulocyte colony stimulating factor, however, other medications may also be associated.

68. What is Adamantiades syndrome?

It is also called Behçet's syndrome or Behçet's disease. Benedict Adamantiades described in 1931 as chronic ocular inflammation and orogenital ulcerations. In 1937, Hulusi Behçet defined the disease with classic triad of aphthous ulcerations, genital ulcerations and uveitis. Other less common lesions are cutaneous involvement, arthritis, thrombophlebitis, gastrointestinal manifestations and CNS involvement.

69. What is pathergy test?

It is a clinical test done to diagnose Behçet's disease (BD). Skin lesions resembling erythema nodosum or large pustular lesions occur in over 50% of patients with BD. These lesions may be precipitated by trauma, and it is common for patients with BD to have a cutaneous hyper-reactivity to intracutaneous injection or a needle stick (pathergy).

One of the most important skin manifestations is the presence of positive pathergy. One or two days after the injection of an inert substance (e.g. sterile saline) using a 20 gauge needle, a tuberculin-like skin reaction or sterile pustule develops. This hyper-reactivity appears to be unique to BD and is seen in 40–88 per cent of patients with this disease.

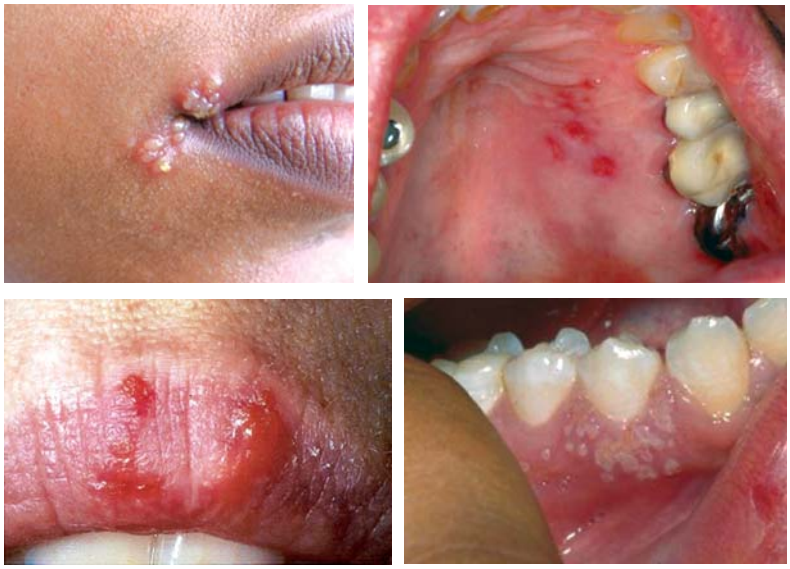
70. What are the features of atypical RIHS?

- RIHS of gingival papilla
- Persistent infection of gingivae
- Persistent enlarged ulcers
- RIHS in immunoincompetence

71. What are the sites of recurrent herpes infections?

Recurrent herpes lesions may begin with a prodrome of burning, tingling, or pain without a visible lesion. Next, a vesicle may appear that will soon rupture into a moist ulcer, or an ulcer will emerge directly. Such secondary lesions have a predisposition for the vermilion and vermilion-skin edge of the lips (recurrent herpes labialis).

The lesions otherwise have a predisposition for the keratinized surfaces of the palate and gingiva.



Recurrent intraoral herpes on palate and gingiva

72. Enumerate the differentiating points of erythema multiforme and herpes infection.

	Erythema multiforme	*Herpes infection*
Age	Young adults	Children
Size	Large ulcers	Small ulcers
Vesicles		Thin walled, generally at mucocutaneous junction
Location	Oral/lip ulcers	Oral/perioral ulcers
Skin lesions	These are target lesions	Skin ulcers
Severity	Mild to severe	Moderate to severe
Intraoral sites	Buccal mucosa, tongue, lips, palate	Gingiva and lips
Extraoral sites	Extremities	Perioral skin
Cause	Hypersensitivity	HSV
Treatment	Symptomatic	Antiviral drugs

73. Enumerate the differentiating points between aphthous ulcers and recurrent herpes simplex infection.

	Aphthous ulcers	*Herpes infection*
Cause	Immune dysfunction	HSV 1
Triggers	Stress, trauma, diet, hormones, depressed immunity, smoking	Stress, trauma, depressed immunity, UV light
Prodrome	Little prodrome	Prodromol symptoms
Clinical appearance	No vesicles	Vesicles precede ulcers
	Single oval ulcers	Multiple confluent ulcers
Microscopic appearance	Non-specific	Viral cytopathic changes
Sites	Non-keratinized	Keratinized
Treatment	Corticosteroids, tetracycline	Antiviral treatment

74. Name the conditions exhibiting oral fissures.

1. Angular cheilosis, exfolative cheilitis
2. Denture irritation hyperplasia, epulis fissuratum
3. Congenital cleft, median rhomboid glossitis-fissured variety
4. Fissured tongue, Melkersson-Rosenthal syndrome
5. Squamous cell carcinoma—fissured variety
6. Syphilitic rhagades, cheilitis granulomatosa
7. Plasma cell gingivostomatitis
8. Riboflavin deficiency, Plummer-Vinson syndrome
9. Crohn's diseases, pyostomatitis vegetans, Down's syndrome

75. What is symblepharon? What is its further stage called? What are the other ocular lesions of pemphigoid?

The ocular lesions of pemphigoid are symblepharon, triachiasis and entropion.

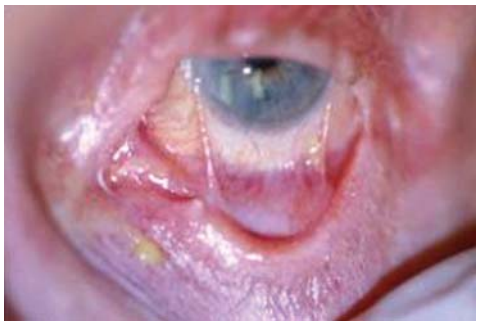
The condition is seen in cicatricial pemphigoid. The conjunctiva will be reddened but will produce minimal symptoms. Scarring, particularly of the conjunctiva, is the mechanism of debilitation associated with cicatricial pemphigoid (hence its name).

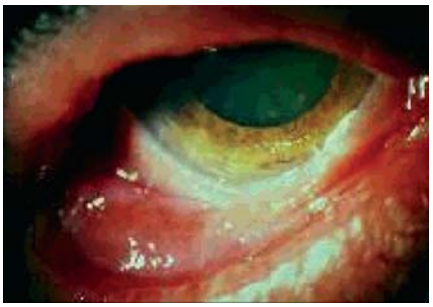
As the disease continues, scar bands form between the bulbar conjunctiva (the conjunctiva over the globe) and the palpebral conjunctiva (the conjunctiva over the inner eyelids), called symblepharon. This scar band in particular contracts to invert usually the lower lid eyelashes toward the cornea in a condition called trichiasis. This, in turn, abrades the cornea, causing ulceration, which leads to opacifications and even blindness in 15% of cases. In severe cases, scar bands will connect the upper and lower palpebral conjunctiva to partially close the eye, a condition called ankyloblepharon.

The other lesions are triachiasis and entropion.

Entropion of the eyelid occurs when the lid margin inverts or turns against the eyeball.

Trichiasis is the eyelashes grow inward towards eye touching cornea or conjunctiva.





Entropion after epilation

76. What are the differentiating points of pemphigus and benign mucous membrane pemphigoid?

	Pemphigus	*Benign mucous membrane pemphigoid*
Bulla location	Intraepithelial	Subepithelial
Antibodies are directed against	Desmogleins	Collagen proteins
Lesions distributed on	Oral mucosa and skin	Oral mucosa and eyes
Bulla	Thin	Thick
Rupture of bullae	Easily	Not easily
Prevalence	Common	Infrequent
Scarring	Absent	Present
Acantholysis	Absent	Present

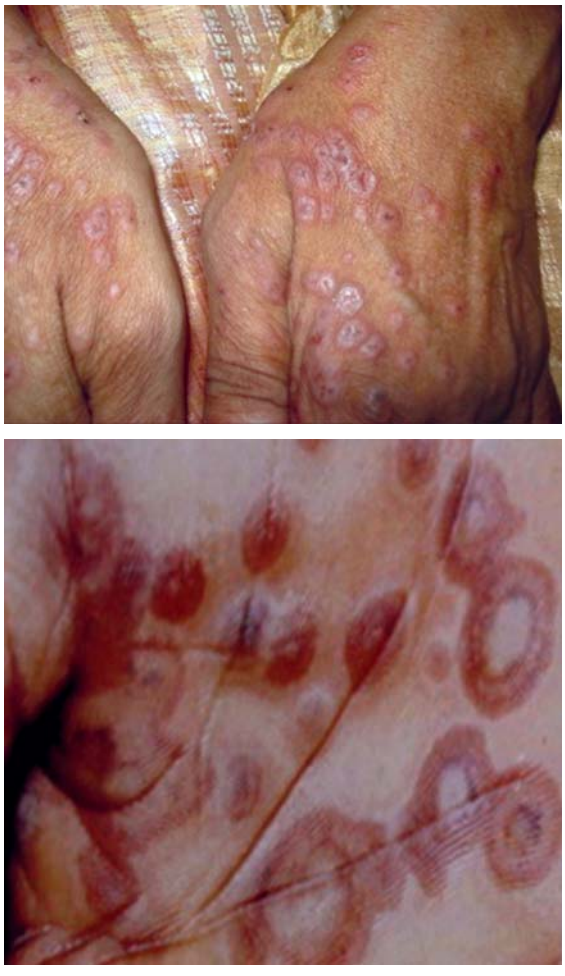
77. How erythema multiforme differs from SJS and TEN?

Erythema multiforme (EM)	*SJS and TEN*
Erythema multiforme (EM) is typically mild, self-limiting and recurring mucocutaneous reaction	SJS and TEN are less common and more severe condition
Immune mediated inflammatory reaction	Hypersensitivity reaction
Common under 40 years of age, rarely seen under the age of 3 and over 50 years	Adults
Little gender difference	Women are affected twice as men.
Seasonal clustering in spring	Nothing
No hereditary	There may be hereditary component
Not associated with HIV	Increased incidence in HIV-infected population
Infectious in origin HSV-1, HSV-2, progesterone therapy	80–95% of TEN and 50% SJS are precipitated by drugs (sulfonamides, NSAID, penicillin and anticonvulsants, other causes are graft *vs* host disease, mycoplasma pneumonia).
Epidermal necrosis of keratinocytes termed satellite necrosis	Much more widespread necrosis of epidermis and little vascular inflammation of dermis
Periascular infiltrate of CD4 and CD8 lymphocytes	Remarkable absence of lymphocytes
Skin lesions show multiple target/iris lesions	Target lesions are not well-defined and larger
All lesions typically present within 3 days of onset	Lesion may appear 45 days of drug treatment and a few days of repeat exposures
Nikolsky's sign not positive	Nikolsky's sign positive
Lip involvement is almost universal	Lip, buccal mucosae and palate may be involved.
Erythema multiforme shows variety of lesions but only 10% of body surface is involved	These lesions spread over much surface of body within short time. Large lesions more than 30% define TEN
Prodormal symptoms are rare and if present are typically mild and non-specific (cough, rhinitis, low-grade fever)	Prodrome occurs 7 to 14 days in advance of lesion (fever, malaise, headache, cough, rhitis, sore-throat, myalgia, arthralgia)
EM is recurrent phenomenon with highly variable frequency and severity of episodes	Repeat attacks do not occur if the offending drug is strictly avoided
Medical complication related to EM is rare	The sloughing may involve oesophagus and respiratory tree, conjunctival lesions may proceed to blindness, and genital ulceration may proceed to urinary retention and phimosis. Sepsis from skin infections, cardiac complications, and renal failure may cause death.
Laboratory investigations—no significant abnormality	Blood sedimentation rate is increased, moderate leukocytosis, hyponatremia, anemia
Investigative focus is identification of infectious agent particularly HSV	The effort to identify the drug
Treatment is prevention HSV infections	Indefinite avoidance of drug

78. What are typical and atypical target lesions?

Typical target lesions—skin lesions of erythema multiforme are pathognomonic.

They may accompany the oral condition, or there may be cutaneous manifestation without oral involvement. Skin lesions have a characteristic "bullseye" or "target" appearance. Although the palms of the hands are a classic location, these lesions may occur anywhere on the skin. A target lesion consists of concentric zones of color change with evidence of damage to the epidermis in central zone such as bulla formation or crust. Early target lesions will have a central dusky zone and a red outer zone but may evolve to three zones of color change.



Typical target lesions

Atypical target lesions are dark red macules which have a central blister, may or may not be raised and have a poorly delineated border or they may have two concentric zones and have area of palpable, rounded edematous lesions with poorly defined borders. They are more common in SJS and TEN.

The typical target lesions are acral while atypical target lesions are usually truncal.

79. What is centripetal and centrifugal spread? Give examples of oral mucosal lesions with same spread?

The lesions of the disease which spread towards centre or axis are called centripetal. The lesions of the disease which spread away from the centre or axis are called centrifugal. The target lesions of erythema multiforme form on extensor surfaces of acral extremities and spread centripetally, rocky mountain spotted fever has centripetal rash, staphylococcal TSS has erythematous rash which spreads centripetally.

Chickenpox rash begins on trunk and spreads on face—centripetal spread while it also has centrifugal spread to extremities. Varicella rash has centripetal distribution, in contrast, variola rash has centrifugal distribution and common on trunk, face.

Varicella spreads centripetally from skin to neurons centripetally to dorsal root ganglion where it remains latent and upon reactivation results in centrifugal spread which results in shingles.

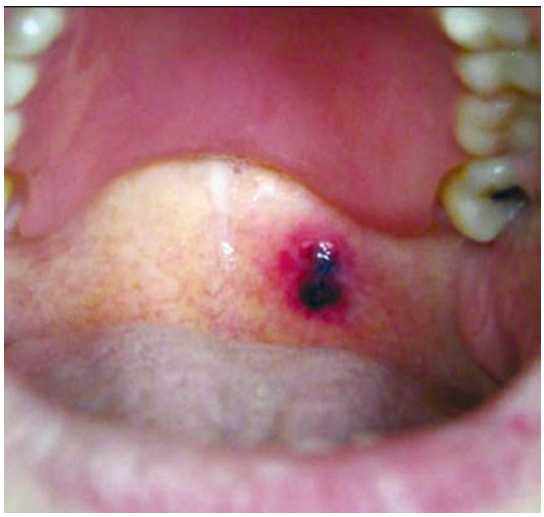
Lesions of ACLE also involve palms and soles and may spread centripetally.

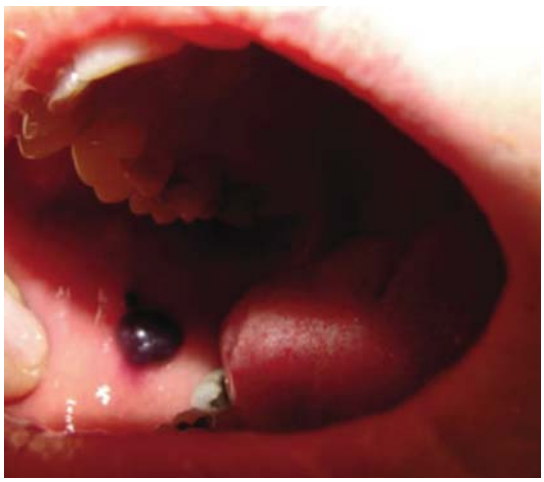
Cutaneous invasive squamous cell carcinoma may spread centripetally rarely along the trigeminal nerve axis into the cavernous sinus and gasserian ganglion.

Rabies virus may spread centrifugally from CNS to salivary acinar cells.

80. What is angina bullosa hemorrhagica?

Angina bullosa hemorrhagica is a rare and benign disorder, usually localized in the subepithelial layer of the oral, pharyngeal and esophageal mucosa. The lesions are characterized by their sudden onset. They appear as a painless, tense, dark-red and blood-filled blister in the mouth that rapidly expand and rupture spontaneously in 24–48 hours. The bullae are present in the absence of any systemic disorder and hematological disease.

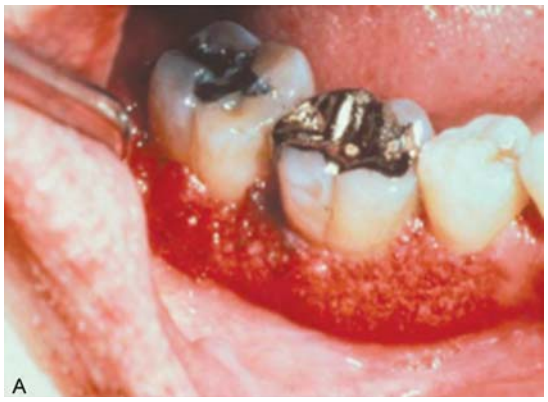


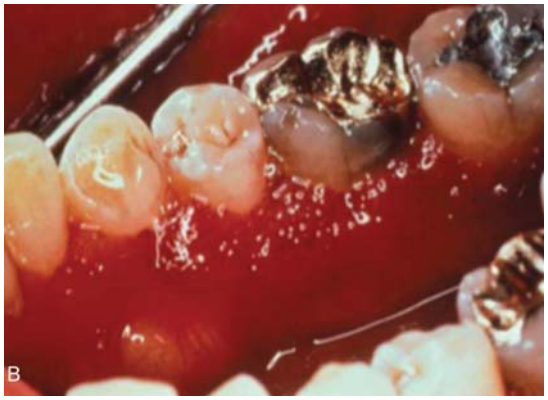


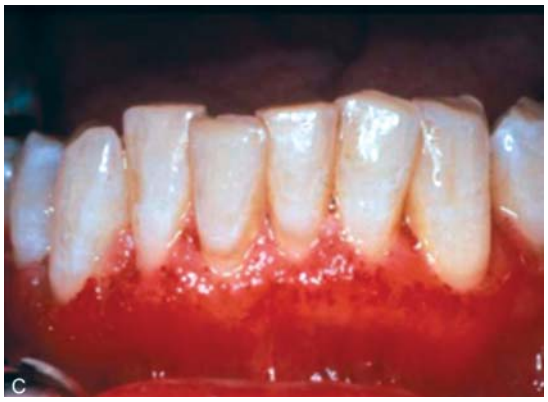
81. What are strawberry like lesions in and around oral cavity?

These are strawberry gingivitis, strawberry tongue and strawberry nevus.

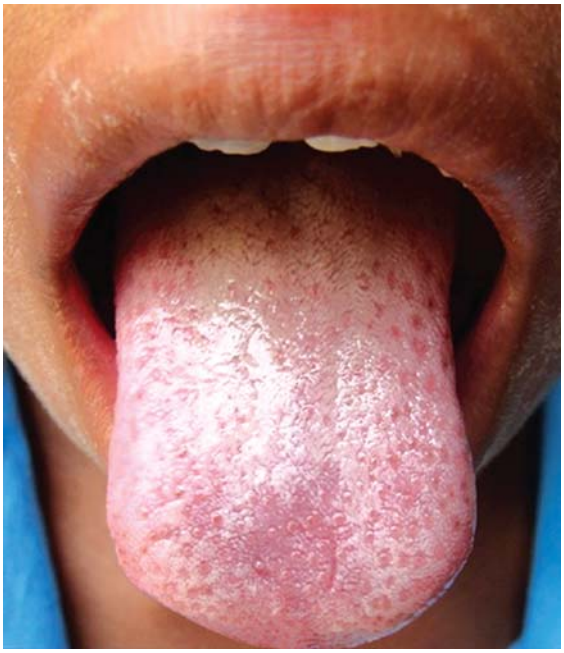
Strawberry gingivitis: Particularly strawberry-like gingivitis or hyperplastic gingival lesions are suggested to be a characteristic sign of granulomatosis with polyangitis. This sign consists of reddish-purple exophytic gingival swellings with petechial hemorrhages, thus similar to strawberries. This lesion can remain localized in the oral cavity for unusually long periods of time before multi-organ involvement occurs.



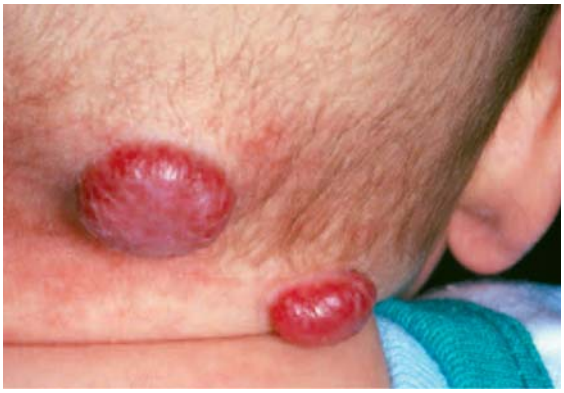




Strawberry tongue: It is seen in scarlet fever. The tongue is initially coated with a white, furry tongue with red projecting papillae (white strawberry tongue), by third or fifth day the white coat slough off leaving a red swollen tongue (red-strawberry tongue).



Strawberry nevus (strawberry hemangioma): It is also called congenital hemangioma appears around time of birth and disappears at childhood. These are raised, verrucous, cutaneous vascular lesions with a reddish-purple color.



82. What are the types of denture stomatitis?

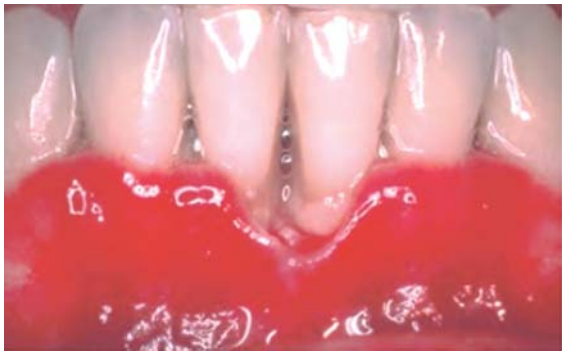
- **Type 1:** Localized inflammation or pinpoint hyperemia.
- **Type 2:** More diffuse erythema (redness) involving part or all of the mucosa which is covered by the denture.
- **Type 3:** Inflammatory nodular/papillary hyperplasia usually on the central hard palate and the alveolar ridge.

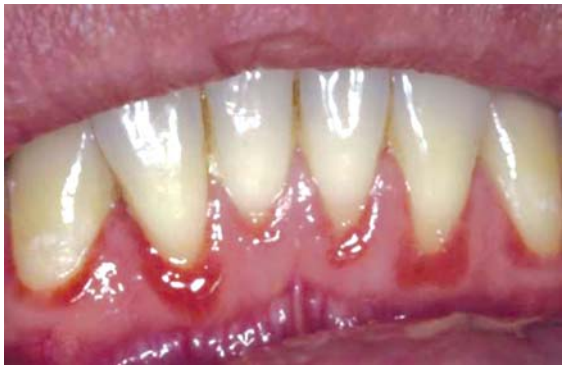
83. Enumerate the conditions in which oral mucosal and conjunctival lesions are seen.

Benign mucous membrane, cicatricial pemphigoid, SJS, TEN, hereditary benign intraepithelial dyskeratosis even sometimes lesions of discoid lupus erythematosus. Even lichen planus is also reported in the literature.

84. What is linear gingival erythema? Enumerate the conditions causing linear gingival erythema.

This is unusual form of gingivitis appears with a distinctive fiery linear band of erythema





that involves free gingival margin and extends 2–3 mm apically. Sometimes it is seen as petechiae and diffuses gingival erythema and involves alveolar mucosa too. Formerly it was called HIV gingivitis. It is seen in HIV associated periodontal disease and abnormal immune response. It is seen in association with candidosis. The lesions may be generalized or involve one or two teeth. No ulceration, pocketing or loss of attachment is observed. Bleeding on probing is not present. The characteristic feature is maintaining good oral hygiene and gingival debridement does not resolve the problems.

Similar clinical feature may be seen in neutropenia, the clinician should rule out by doing routine blood investigations.

Treatment should include debridement, providone iodine irrigation, chlorhexidine mouth rinses and antifungal medication.

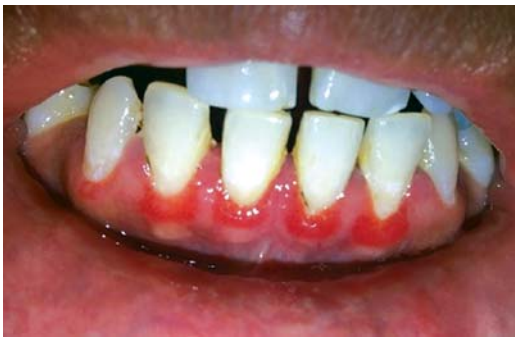
85. What is plasma cell gingivitis?

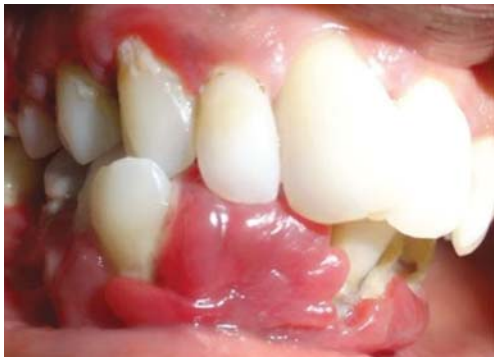
Plasma Cell Gingivitis

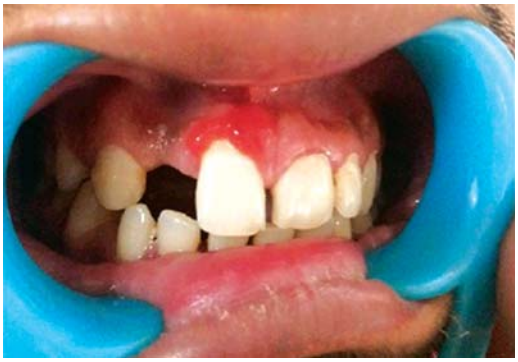
Plasma cell gingivitis (atypical gingivitis, plasma cell gingivostomatitis). This distinct form of gingivitis first reported in the United States, usually arises as a hypersensitive reaction to a component of chewing gum, dentifrices, or some of the dietary components. It commonly presents as a mild marginal gingival enlargement, sometimes extending to involve the attached gingiva.

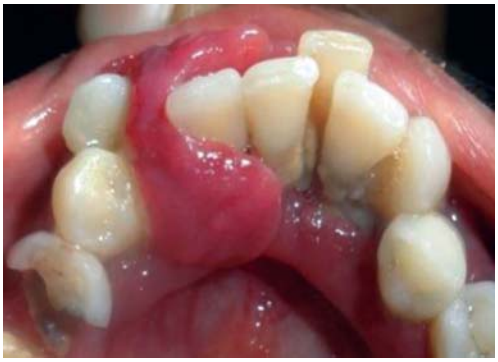
Clinical Features

This disease is more prevalent in young women. The initial symptom is soreness of the mouth, which is intensified by hot or spicy food. It starts as mild marginal gingival enlargement and extends to attach gingiva, and in severe cases, extends to buccal and vestibular mucosa. Gingiva appears swollen, erythematous, and friable with loss of stippling. It may be granular sometimes. The involvement of other oral tissues like the tongue and lips is common. They appear atrophic, dry, and exhibit cracks or fissures. The differentiating point from plaque induced gingivitis is that it involves the oral aspects of attached gingival tissue.









Treatment and Prognosis

Possible allergens should be identified by careful study of the patient's history and eliminated. Topical and systemic steroids give good results.

86. Enumerate the conditions in which desquamative gingivitis is seen.

Desquamative gingivitis (DG) is a descriptive term first introduced by Prinz in 1932, i.e. synonymous with the presence of erythema, desquamation, erosion and blistering of attached and marginal gingiva. It is a clinical term used to describe red painful, glazed, friable gingiva. Nikolsky's sign often shows positive reactions with DG. It is more common in middle aged to elderly female, affects labial/buccal gingival frequently spares the marginal gingiva. It can involve whole thickness of attached gingiva. It does not respond to conventional periodontal therapy or traditional oral hygiene measures. Nisengard and Levine suggested following criteria standard for clinical appearance of desquamative gingivitis:

1. Gingival erythema not resulting from plaque
2. Gingival desquamation
3. Other intraoral and sometimes extraoral lesions
4. Complaint of sore mouth particularly after eating spicy foods.

A. *Dermatological disorders*
- Cicatricial pemphigoid
- Lichen planus
- Pemphigus
- Psoriasis
- Bullous pemphigoid
- Epidermolysis bullosa
- Contact stomatitits
- Linear IgA disease

B. *Endocrine disturbances*
- Estrogen deficiency (following oophorectomy and in postmenopausal stages)
- Testosterone imbalance
- Hypothyrodism

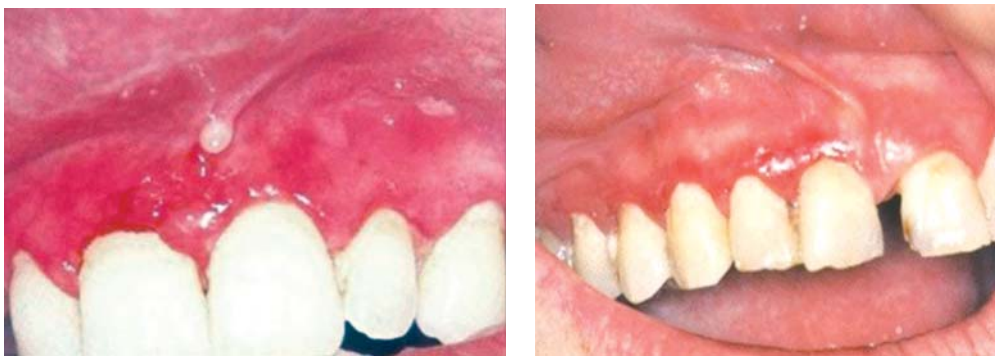
C. *Aging*

D. *Immunological disorder*
- Crohn's disease.
- Chronic ulcerative stomatitis

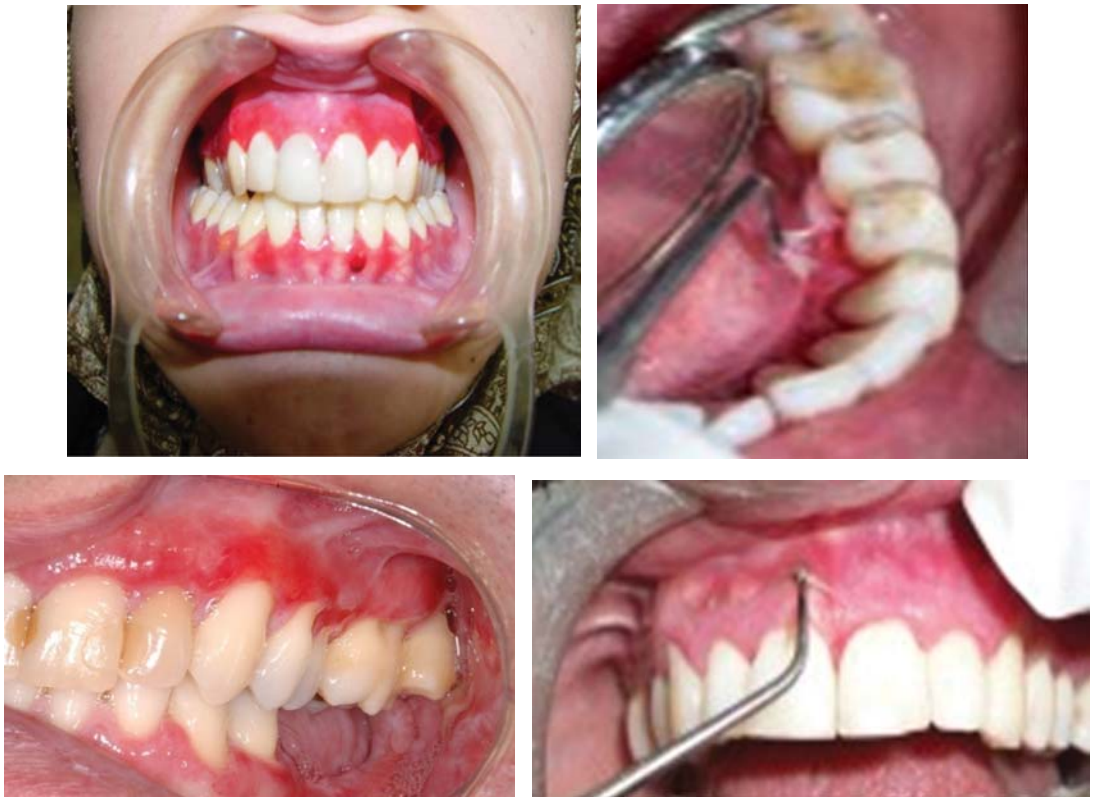
E. *Idiopathic*

F. *Chronic infections*
- Tuberculosis
- Chronic candidiasis
- Histoplasmosis

Overall mucous membrane pemphigoid, oral lichen planus are most common causes accounting for 80% of cases while pemphigus is second most cause. Desquamative gingivitis is the main oral feature of mucous membrane pemphigoid and may be sole presenting feature.
- Pemphigus
- Benign mucous membrane pemphigoid
- Erosive lichen planus
- Desquamative gingivitis is not a diagnosis.



Presentation in pemphigus and cicatricial pemphigoid



Characteristics peeling of tissues showing desquamation

87. What are reactive lesions appearing in the oral cavity? Enumerate various reactive lesions.

Reactive soft tissue enlargements are caused by injury, such as infections, physical trauma, chemical trauma, or allergic reactions. Reactive soft tissue enlargements usually have a rapid onset (short duration) and may increase and decrease (fluctuate) in size and usually eventually regress. Reactive enlargements are often, but not always, tender or painful and usually have a more rapid growth rate (measured in hours to weeks) than tumors. Sometimes patients with reactive enlargements will be able to report the source of injury. Sometimes reactive lesions are associated with tender lymph nodes and systemic manifestations, such as fever and malaise. Once it is decided that a soft tissue enlargement is reactive, the next step is to determine what the lesion is reacting to, such as bacterial, viral, or fungal infections or chemical or physical injury.

Some examples of reactive soft tissue enlargements:

- Mucocele (salivary extravasation phenomenon)
- Necrotizing sialometaplasia
- Periodontal abscess
- Radicular (periapical) abscess
- Fibrous hyperplasia
- Inflammatory papillary hyperplasia

Varix and acquired malformations, pyogenic granuloma, peripheral giant cell granuloma, scarlet fever.

Reactive lesions of salivary gland origin—mucous extravasation phenomenon, mucous retention cyst (obstructive sialoadenitis), maxillary sinus retention cyst, necrotizing sialometaplasia, adenomatoid hyperplasia.

Reactive osseous and chondromatous metaplasia. Occasionally, cartilage or bone may be discovered within soft tissue specimens removed from the oral cavity.

88. What are inflammatory hyperplasias? What are the characteristics of inflamatory and fibrous hyperplasias?

This is an increase in the size of an organ or tissue due to an increase in the number of its constituent cells, as a local response of tissue to injury. It can be considered as an over-exuberant reparative response.

- No malignant potential
- Periphery oral mucosa—trauma
- Recurrences—failure to eliminate source of chronic irritation
- Bone erosion—rare aggressive, malignancy.
- Local excision—microscopic examination (fibrous, granulation).

89. Enumerate the various inflammatory reactive hyperplasias and name there etiological factors.

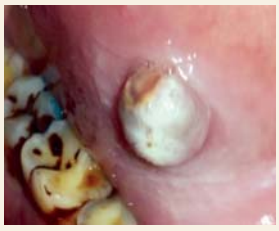
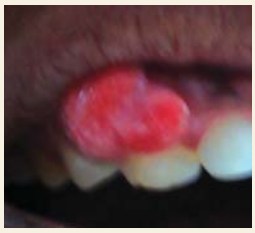
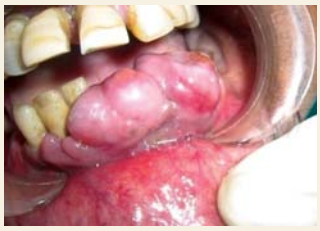
- Irritational fibroma
- Chronic inflammatory gingival enlargements
- Pyogenic granuloma
- Hormonal tumor—puberty gingivitis, pregnancy tumor
- Fibroepithelial polyp/mucoepithelial polyp
- Epulis fissuratum
- Parulis
- Peripheral giant cell granuloma
- Pulp polyp
- Gingival polyp
- Epulis granulomatosum

Causative factors—trauma (chronic)—calculus, ill-fitting dentures, overhanging dental restorations, biting injury, fractured teeth.

Circulating hormones.

90. Discuss the differential diagnosis of most commonly occurring inflammatory reactive hyperplasia.

The commonly occurring inflammatory reactive hyperplasia are fibroma, pyogenic granuloma and peripheral giant cell granuloma.

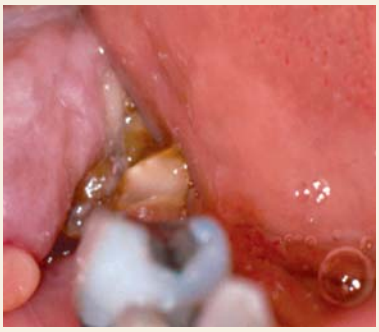
	Fibroma	*Pyogenic granuloma*	*Peripheral giant cell granuloma*
Age of occurrence	4th to 6th decade	Children, young adults	31–41 years
Intraoral common site	Buccal mucosa	Gingiva/maxilla	Gingiva/mandible
Bleeding	Does not bleed	Bleeds	May/may not
Overlying surface	Smooth, pale surface, pink, slow growth	Ulcerates, red, rapid growth	May ulcerate, red to pale Not rapid growth
Clinical photographs of lesion			

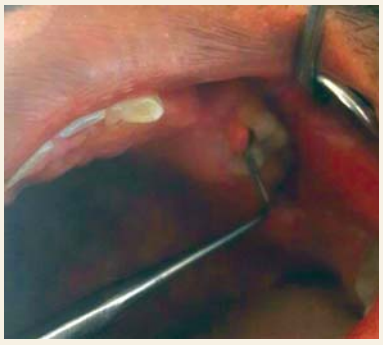
91. Why pyogenic granuloma is called pyogenic in spite of no frank pus formation?

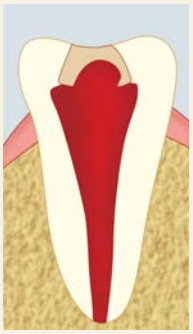
Clinically the asymptomatic reddish papule, nodule or polyp usually shows at least part of its surface to be rough. Ulcerated and necrotic. The fact that this necrotic white material clinically resembles pus prompted early clinicians to refer to the lesion pyogenic granuloma. However, there is no pus in the lesion.

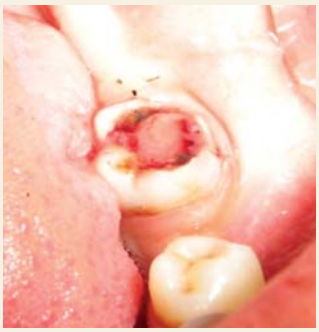
92. Describe differences between gingival polyp and pulp polyp.

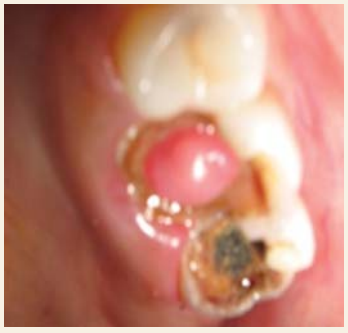
Gingival polyp	*Pulp polyp*
Originates from gingiva	Originates from pulp
Runs towards centre	Runs outwards from the centre
Broad base	Narrow stalk
Proximal surface	Occlusal surface
Can be separated from the tooth	Attached to the tooth
Bleeds easily on probing	May not bleed easily
Excision/local debridement	Endodontic/extraction

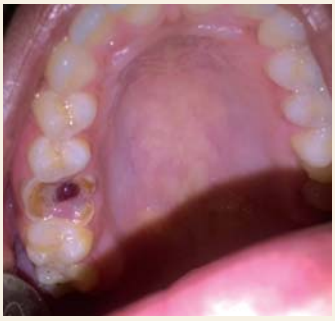






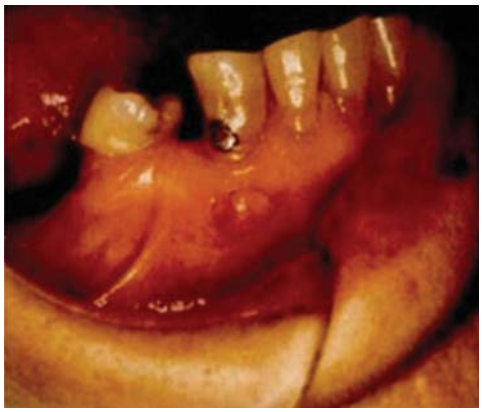


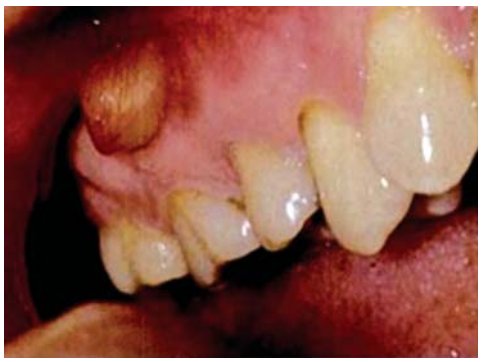




93. Why peripheral giant cell granulomas (PGCG) occur anterior to molar regions?

The etiology and nature of PGCG still remains undecided. In the past, several hypotheses had been proposed to explain the nature of multinucleated giant cells including the explanation that they were osteoclasts left from physiological resorption of teeth or reaction to injury to periosteum. There is strong evidence that these cells are osteoclasts as they have been shown to possess receptors for calcitonin and were able to excavate bone *in vitro*. Giant cells are present mainly in the anterior to molar regions which are required for resorption of deciduous tooth, hence peripheral giant cell granulomas occur anterior to molar region.



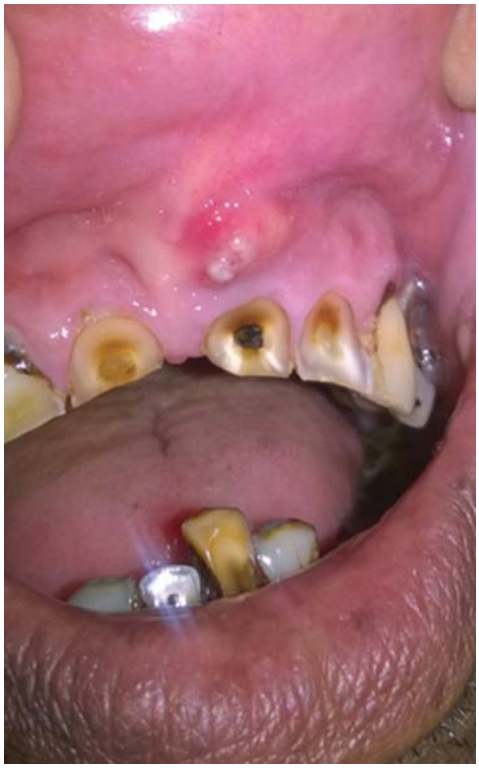


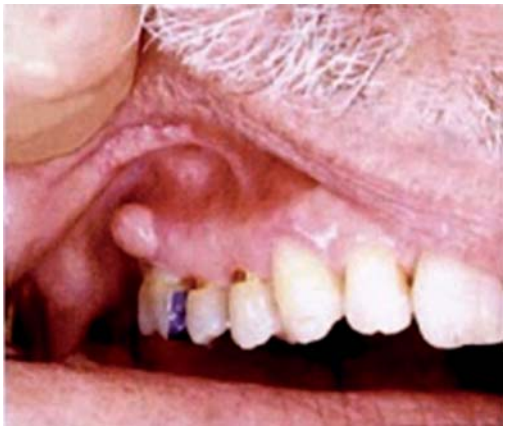
94. In which conditions central giant cell lesions are seen?

Central giant cell lesions can be seen in hyperparathyroidism ("brown tumors"), cherubism, Noonan syndrome and neurofibromatosis type 1. In the last two, this association may be coincidental.

95. What is parulis?

It is also called gum boil yellowish-white gingival swelling caused by submucosal pus. The cause is periodontitits or tooth abscess, causing sinus tract. Usually, on buccal gingival of children and young adults. The mandibulo-alveolar mucosa and palate may also be





involved. Slight digital pressure on the periphery of a parulis may force a drop of pus from the sinus opening, and this is almost pathognomonic. The lesion usually regresses spontaneously after the chronic odontogenic infection has been eliminated. If size is considerable and there is a substantial amount of fibrosis, however, the lesion regresses somewhat and then persists as FH (fibrous hyperplasias).

96. What is bleb? Name the conditions exhibiting gingival bleb.

Bleb is pooling of fluid below tissues. Gingival blebs are seen in benign mucous membrane pemphigoid, cicatrical pemphigoid. Blue to purple colored blebs are seen in lingual varieces.

97. Enumerate the necrotizing conditions of oral cavity.

Acute necrotizing ulcerative gingivitis, noma, necrotizing sialometaplasia, toxic epidermal necrolysis (TEN). Diffuse gangrenous stomatitis (severe debilitating conditions like diabetes, uremia, leukemia, etc). However, erythema multiforme, pemphigus in severe extent and lesions caused by electrical burns can be necrotizing in nature. Necrotizing ulcerative periodontitis (NUP) and necrotizing ulcerative stomatitis (NUS) in HIV patients. Necrotic ulcers may occur in debilitating systemic diseases such as leukemia, sickle cell anemia, and uremia.

98. What are the types of contact drug reactions?

These are of three types.

First type is a direct drug toxicity: Whereby the pH or an active chemical site produces a physical injury to the tissue. "Aspirin burn" caused by the acidic pH of aspirin and the chemical burn of capsicum in sharp peppers are examples of direct drug toxicity. Such reactions are direct physical injuries, not actual immune-based reactions. The lesions are usually a localized, white painful area with a fibrinous base or slough surrounded by a small zone of erythema.

Second and most predominant type is a T-cell mediated immune reaction: Initially, the offending drug is topically absorbed through intact skin or mucosa. During absorption, the drug contacts the Langerhans' cells, which exist in the middle zone of the prickle cell layer in both skin and mucosa. The Langerhans' cell, which seems to be a type of histiocyte, processes the drug as an antigen. It will present the drug on its cell membrane to T-lymphocytes, creating antigen-sensitized T-lymphocytes. When the drug is topically absorbed a second time, the sensitized T-lymphocytes will react by secreting an array of lymphokines that produce inflammation and tissue injury characteristic of contact reaction "allergies." This may affect skin (contact dermatitis) or oral mucosa (contact stomatitis). Such reactions will mostly be areas of boggy erythema corresponding to the drug contact and the pattern dispersal by tongue, lip, and swallowing movements. If the reaction is more severe, actual vesicles or ulcers may form. The prime offending agents have been cosmetics, including lipsticks and lip balm, and dental preparations, such as toothpaste and some of its ingredients. In particular, mint and cinnamon flavourings have been implicated in oral contact stomatitis. Cosmetic and skin care preparations have been such prominent offenders that complete lines of hypoallergenic products have been developed. Included in this group is hypoallergenic surgeon gloves, which were developed because the powdered starch in many gloves induced a typical contact dermatitis. One supposed cause of contact stomatitis that has been grossly overstated for years is that related to denture acrylic. There is little if any direct evidence to support a true "denture contact stomatitis." Most so-called denture reactions represent other diseases, such as candidiasis, pemphigoid, lichen planus, or mere chronic injury from an ill-fitting denture.

Third type of contact drug reaction is a B-cell mediated immune reaction whereby antibodies are produced: In a fashion similar to the mechanism noted for T-cell-mediated immune reaction, Langerhans' cells may present processed antigen to B lymphocytes, which in turn manufacture specific antibody to the absorbed drug. On a subsequent absorption, antibodies attack the antigen at the epithelial or subepithelial level to produce inflammation and tissue injury. In plasma cell gingivitis, e.g. the plasma cells are prominent because they produce specific antibody in the area of antigen (the contact drug) absorption. Plasma cell gingivitis was common in the late 1960s and early 1970s but is rare today. It is believed that the peak of incidence was related to formula changes and ingredients in several dental preparations, which have since been eliminated by the manufacturers.

99. Where is oral exfoliative cytology indicated?

Oral exfoliative cytology is recommended as an adjunct to open biopsy, for prebiopsy assessment, for the examination of broad surface lesions, and for the evaluation of patients after definitive treatment.

100. In which conditions exfoliative cytology is not useful?

Exfoliative cytology is unsuitable for the following lesions: Homogeneous leukoplakias. Smooth surfaced exophytic lesions, submucosal lesions, unulcerated pigmented lesions, verruca vulgaris, papilloma, condyloma acuminata, etc.

101. What is diascopy? State in which conditions it is positive.

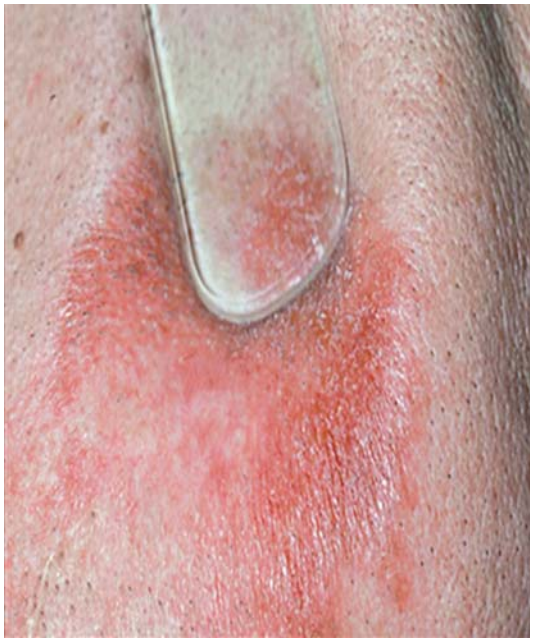
Diascopy (vitropression) is a procedure in which a diascope (more commonly a microscope slide (rather than thin glass plate) or magnifying glass, even a wafer of clear acrylic) is pressed against a lesion with gentle pressure down on to tissue or gently rocked from one side to other to see whether it blanches.

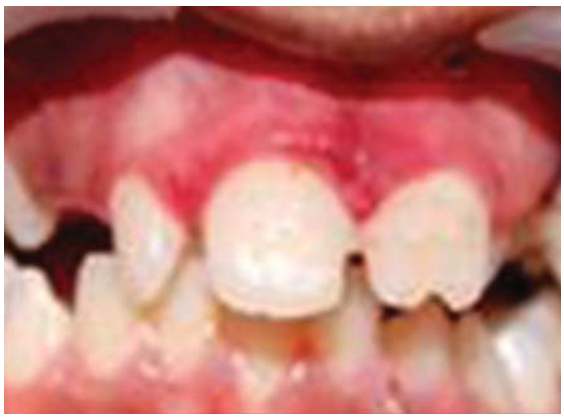
Orit is a procedure of applying pressure to a suspected vascular lesion to visualize evacuation of coloration and may facilitate the differentiation of small vascular lesion from a pigmented lesion. It will evacuate the blood from small vessels allowing evaluation of other colors. It can be done intraorally also.

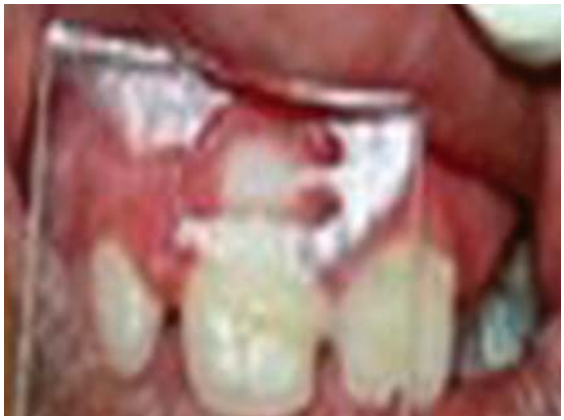
Erythema due to vasodilatation blanches when the vessels are compressed, while purpura, on the other hand, does not blanch because the blood is present in tissue spaces not in vessel. Telangiectasis will also blanch.

Diascopy is used to determine whether a lesion is vascular, non-vascular (nevus), or hemorrhagic (petechia or purpura). Hemorrhagic lesions and non-vascular lesions do not blanch; inflammatory and vascular lesions do. Diascopy can also help identify sarcoid skin lesions, which, when tested, turn an apple jelly color. This is tested in lupus vulgaris.

It is detected to differentiate from localised area of vasoconstriction from a hypopigmented and depigmented skin patch, i.e. vitiligo. In the former the diascopy will blanch the lesion and in later the patch is still detectable.







In treating spider angiomas and cherry angiomas by laser it arrests the flow in central arteriole by releasing the pressure it refills superficial branches making amenable for laser treatment.

102. Enumerate the diascopy results of various varicosities and hemorrhagic lesions.

Lesion	*Blanching with diascopy*
Petechiae	Negative
Purpura	Negative
Ecchymosis	Negative
Hematoma	Negative
Lingual varicosities	Positive
Venous varix (Venous lake)	Positive
Hemangioma	Positive
Lymphangioma	Positive
Telangiectasis	Positive

103. Why the prednisone should be taken in the morning and when the slow taper is indicated?

The adrenals normally secret most of their daily equivalent of 5 to 7 mg of prednisone in the morning, all the prednisone should be taken early in the morning to stimulate the physiologic process, thus minimizing the pituitary-adrenal axis and side-effects.

Slow steroid taper is not necessary if treatment lasts for less than 2 weeks because adrenal suppression is minimal.

104. Mechanism of action of corticosteroids in suppressing inflammation.

The anti-inflammatory actions of corticosteroids include:

1. A reduction in the exudation of the leukocytes and plasma constituents
2. Maintenance of cellular membrane integrity with prevention of cellular swelling
3. Inhibition of lysozyme release from granulocytes and inhibition of phagocytosis
4. Stabilization of the membranes of the intracellular lysosomes containing hydrolytic enzymes
5. Decreased scar formation by inhibiting proliferation of fibroblasts and
6. Possible effect on antibody formation when administered in large doses.

Corticosteroids have also been shown to suppress T-cell formation (Wood & Goaz, 5th ed.).

105. Which is the steroid of choice for mucosal lesions? What are different steroid regimens?

Prednisone, an anti-inflammatory glucocorticoid, is the first drug of choice. This drug affects mostly the cellular phase of inflammation and in particularly lymphocytes. Dexamethasone is more potent and affects the exudative phase of inflammation, ultimately it remains an excellent drug for reducing surgical inflammation (mostly edema fluid).

Systemic corticosteroids are the most predictable medications used to control autoimmune diseases and certain immune-based inflammatory diseases. Most of these diseases are incurable; the goal of therapy is a long-term and if possible permanent drug-induced remission.

Systemic Corticosteroid Regimen I

It is indicated for most oral lesions associated with pemphigus vulgaris, erosive lichen planus, and severe non-ocular pemphigoid

1. Prednisone, 100 to 120 mg per day by mouth (1.5 mg/kg per day) for 2 weeks.
2. A tapering schedule is instituted till 20 mg per day is reached. It may be achieved by reducing prednisone by 20 mg per day each week.
3. This dose of 20 mg per day continued for 1 month.
4. Then 10 mg per day for 3 months.
5. The dose is then reduced to 10 mg every other day for another 3 months.
6. Followed by 5 mg every other day for 6 months.
7. After 6 months of a 5 mg dose of prednisone every other day, the drug may be discontinued with a high possibility of an extended remission in a drug-free state.

The rationale for this approach is to gain a rapid suppression of disease activity with a high loading dose and to taper this dose rapidly enough to avoid most of the more serious side-effects of high-dose prednisone. The 20 mg/day dose is significant because at that dose or below, side-effects are significantly reduced. The tapered dose is extended in length with each decrease in dose to prevent an exacerbation at the time of dose reduction or after drug discontinuation. The every other day dose is designed to permit the hypophyseal-adrenal cortical axis to regain its function.

The 5 mg, every other day, dose is called a "maintenance dose" because 5 mg of prednisone equals the daily 20 mg of cortisol the adrenal cortex produces in an unstressed individual, and the every other day use continues to allow the adrenal cortex to regain activity.

This regimen is the preferred regimen because it facilitates a long-term remission and has reduced side-effects. It is very effective but requires close attention by the clinician and absolute compliance by the patient.

Systemic Corticosteroid Regimen II

It is usually applied to resistant pemphigus cases and selected cases of systemic lupus erythematosus or sarcoidosis. This regimen is also indicated in pemphigus vulgaris, erosive lichen planus, and severe non-ocular pemphigoid. It is the preferred regimen in the Stevens-Johnson form of erythema multiforme.

1. This regimen begins with prednisone, 100 to 120 mg per day by mouth (1.5 mg/kg per day), for a period of 2 weeks, at which time the drug is abruptly discontinued.
2. The rationale for this approach is to gain rapid suppression of disease activity and then discontinue the drug before side-effects develop or significant adrenal suppression occurs. This approach is effective and much more straightforward than systemic corticosteroid regimen I. Its drawback is that exacerbations are more frequent, and the disease process is less controlled.

Systemic Corticosteroid Regimen IIIA

This approach is suited to those cases with disease intensity and organ involvement that lower doses of prednisone cannot control.

1. This regimen begins with prednisone, 100 to 120 mg per day by mouth for 2 weeks.
2. A tapering schedule reduces prednisone by 20 mg per day each week until the lowest possible prednisone level is reached without exacerbating the disease. Many individuals remain on 20 mg per day or even higher doses for long periods because lesser dosages are associated with disease exacerbations.

These patients require lifelong dosage adjustments and follow-up. They also develop many of the late complications of ongoing corticosteroid therapy.

Systemic Corticosteroid Regimen IIIB

The approach is reserved for refractory cases and for patients in whom corticosteroid complications pose a greater risk (diabetes, a history of tuberculosis, peptic ulcer disease, osteoporosis (in women), and cataracts). It is recommended as a regimen for ocular pemphigoid.

1. This regimen begins with prednisone, 100 to 120 mg per day by mouth for 2 weeks.
2. A tapering schedule reduces prednisone by 20 mg/day each week until a prednisone level is reached at which the disease is exacerbated. This level and slightly higher levels of prednisone may still be associated with disease activity.
3. Cyclophosphamide, 50 to 100 mg twice daily by mouth; azathioprine, 50 to 100 mg twice daily by mouth; and methotrexate, 25 to 50 mg per week, individually or in combinations, is then added to the prednisone therapy.

The rationale for this approach is to affect the disease with double-drug therapy so that the dosage and, therefore, the side-effects of each can be reduced.

106. What are steroid sparing agents (SSAs)? Enumerate various SSAs.

These are drugs mainly immunosuppressive which are used along with long-term steroid therapy to reduce the side-effects of steroids. Hydroxychloroquine, azathioprine and mycophenolate mofetil are systemic steroid sparing agents that are more commonly used, while levamisole, cyclosporin A, methotrexate can also be used.

- *Hydroxychloroquine* (200–400 mg daily)
- *Azathioprine* (5 to 150 mg daily)
- *Mycophenolate mofetil* (1 gm twice daily for 6 months)

107. What are indications of antihistaminics in oral lesions?

Chronic ulcerative stomatitis, sometimes in geographic tongue. Allergic lesions of oral mucosa and as an adjunct in treatment of orofacial angioneuratic edema.

Erythema multiforme systemic administration and oral rinse of antihistamines mixed with kaopectate in 50:50 are recommended. Radiation mucositis oral rinse is recommended. These oral rinses are used to control pruritis in lichen planus. Chelitis glandularis systemic antihistamins may be used.

The recomanded preparation is dexamethasone elixir 0.5 mg/5 ml (100 ml) rinse with one teaspoonful for 3 min four times daily, and expectorate until the lesions resolve and not to swallow.

Systemic anaphylaxis occurring in dental office.

108. What is recommended antibiotics trial for aphthous stomatitis?

For aphthous ulcers that are numerous and frequent enough to debilitate patients, a trial with antibiotics is useful before resorting to systemic corticosteroids. The three most effective antibiotic regimens are:

1. Erythromycin, 250 mg, by mouth four times daily
2. Tetracycline (achromycin, lederle), 250 mg, by mouth four times daily, and
3. A mixture often called "tetranydril elixir," which consists of 250 mg tetracycline and 12.5 mg diphenhydramine hydrochloride (Benadryl, Warner Lambert) per 5 ml of kaopectate (Pharmacia and Upjohn).

The patient is instructed to use 1 tsp at a time and swish, hold the solution in their mouth as long as possible, and swallow, three times daily.

These regimens have been variably useful in controlling the number, frequency, and duration of lesions. Patients will have "breakthrough" lesions, but they are a few and much more tolerable. Any of these regimens can be continued for extended periods (2 to 6 months) before withdrawal to attempt continued control in a drug-free stage.

If these antibiotic regimens fail, systemic corticosteroids are the treatment of choice.

109. What are the side-effects of topical corticosteroids? What is sufficient therapeutic effect?

Candidiasis, epithelial atrophy, telangiectasias. On skin it may produce skin-striae, hypopigmentation, acne folliculitis. These effects may occur after prolonged or intense dermatologic use.

The therapeutic effect for oral ulcers (especially aphthous ulcers) 15 g of topical steroid over 2–4 weeks and with minimal complications.

110. What are advantages of topical agents?

There is minimal risk for topical use and nearly every drug can be formulated to use this. The drug preparations are safe (low serum levels for systemic side-effects). Ease of use, convenience (as medication can be applied at home), ability to treat large lesions and to reach those of critical sites. Better cosmetics result than surgery. Rapid onset of action.

Limited drug-to-drug interaction. After the application of small amounts of drug locally very high local concentration of drug can be achieved, these may prove toxic if administered systematically.

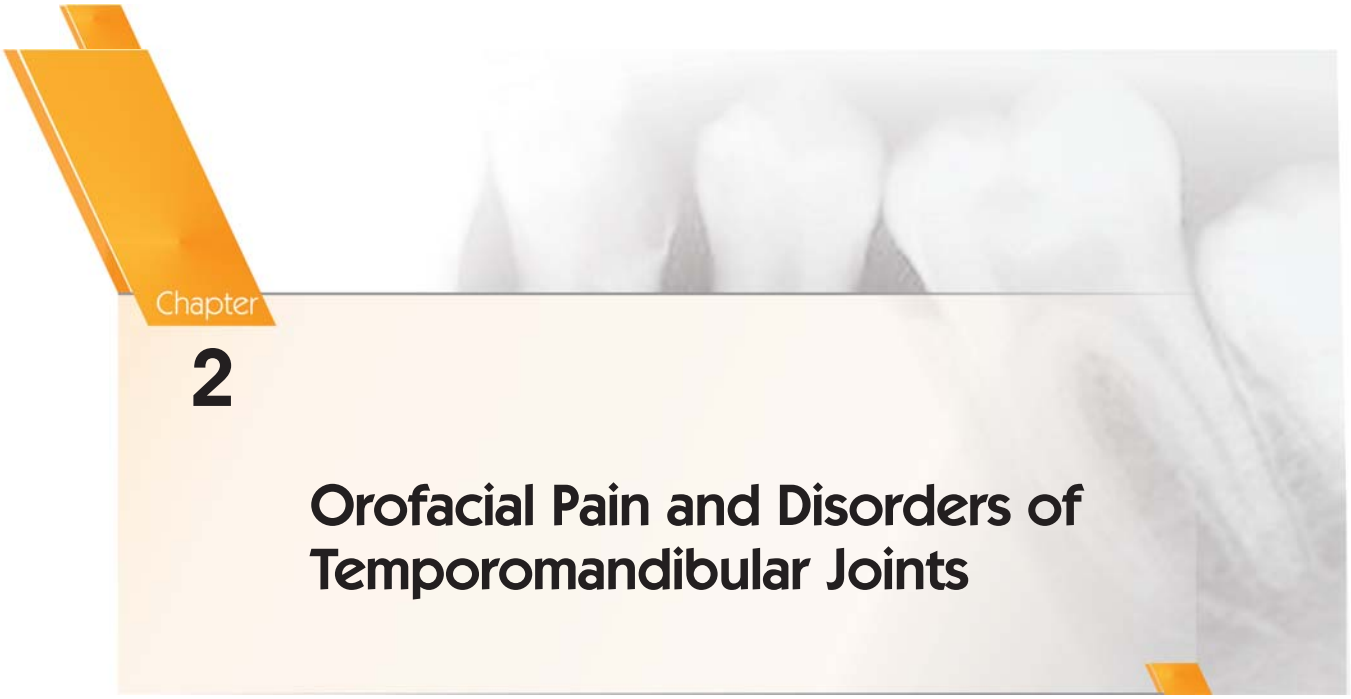
Alternative method for patients who cannot swallow pills or fear of needles.

After administration in skin they enter skin and then variable quantity is distributed through the body, finally eliminated. The concentration of topical agents decreases from skin to the subsurface, whereas opposite is true for systemic administration so if lesion is in epidermis the topical applications are favoured (as highest doses are delivered).

111. What is plasmapheresis? Enumerate the conditions in which it is indicated.

Plasmapheresis is a procedure in which blood is separated into cells and plasma (liquid). The plasma is removed and replaced with fresh frozen plasma, a blood product called albumin and/or a plasma substitute. The procedure is often referred to as plasma exchange. The terms plasmapheresis (PP) and plasma exchange (PE) are often used interchangeably, but when properly used, denote different procedures. Plasmapheresis refers to a procedure in which the plasma is separated from the blood either by centrifugation or membrane filtration. Once separated the plasma can be manipulated in a variety of ways. Plasma exchange refers to discarding the plasma totally and substituting a replacement fluid.

1. Pemphigus
2. Thrombotic thrombocytopenic purpura
3. Myasthenia gravis
4. Severe systemic lupus erythematosus.



Chapter

2

Orofacial Pain and Disorders of Temporomandibular Joints

1. Define pain. What is orofacial pain? What is the unit of pain measurement?

IASP defines pain as an unpleasant sensory and emotional experience associated with actual or potential tissue damage, or described in terms of such damage.

OR

It is the subject's conscious perception of modulated nociceptive impulses that generate an unpleasant sensory and emotional experience associated with actual or potential tissue damage or described in terms of such damage (Bell's).

Orofacial pain can be attributed to conditions of pain in and around the eyes, ears, nose, including sinuses, teeth including paradontics, mouth including lips, jaw bones, salivary glands, throat, cheeks, temporomandibular joints and periauricular area (Huggar et al. 2006).

OR

Orofacial pain (OFP) symptom of broad spectrum of diseases that involve diseases of orofacial structures, musculoskeletal diseases, rheumatic diseases, diseases of peripheral or central nervous system, psychological abnormality, referred from intracranial pathology or cervical muscles.

Del is the unit to measure pain, dolorimetry is a lab method to measure painfulness and dolorimeter is the device used to measure the pain. Human body can bear up to 45 Del of pain.

2. What is chronic pain?

It is defined as pain that lasts longer than six months and often presents with different circumstances, the more functional definition is pain that lasts longer than normal healing time. Chronic pain is often not a symptom but a disease in its own right.

3. What is field of orofacial pain?

This is the discipline of dentistry which includes the assessment, diagnosis and treatment of patients with complex chronic orofacial pain and dysfunction disorders, oromotor and jaw behaviour disorders and chronic head and neck pain. As well as pursuit of knowledge of underlying pathophysiology and mechanism of these disorders.

4. What is chronic orofacial pain?

It is a term used to describe painful regional syndromes with a chronic unremitting pattern.

Clinically

It is subdivided into three classes: Musculoskeletal (temporomandibular disorders), neurovascular (migraine, facial migraine, cluster headache, hemicrania, paroxysmal hemicrania and SUNCT), and nauropathic (trigeminal neuralgia, painful post-traumatic neuropathies, burning mouth syndrome, glossopharyngeal neuralgia).

5. Classify facial pain.

Facial pain can be classified broadly into three groups:

1. Pain arising from diseases of orofacial structures.
2. Pain arising from disorders of nerves and central nervous system.
3. Pain arising in distant organs and referred to facial region.

6. What are different behaviours of pain?

Intermittent pain is one in which suffering distinctly comes and goes, leaving pain free intervals of noticeable duration, e.g. focal reversible pulpitis, TMJ dysfunction syndrome, trigeminal neuralgia.

Continuous pain is one in which pain-free intervals do not occur, e.g. non-arthritic inflammatory pain in TMJ, post-traumatic neuropathy, neuritis.

Recurrent pain is one in which episodes of pain, whether continuous or intermittent, are separated by an extended period of freedom from discomfort only to be followed by another similar episode of pain, e.g. trigeminal neuralgia, headache or facial pain attributed to TMJ disorders.

Momentary pain is one in which duration can be expressed in seconds, e.g. ankylosis of TMJ, developmental disorders of TMJ pain is momentary associated with stretching of ligament, cracked tooth syndrome, dentinal hypersensitivity caused by abrasions, fractures.

Longer lasting pain is one in which duration is expressed in minutes, hours and day, e.g. migraine, TMJ dysfunction, atypical facial pain, irreversible pulpitis.

Protracted pain is one in which pain continues from one day to the next, e.g. post-herpetic neuralgia (it develops protracted pain syndrome), masticatory pain dysfunction syndrome.

Localized pain is one in which patient is able to define pain in exact anatomic location, e.g. periodontal pain is localized owing to mechanoreceptors, lost/cracked tooth, synovitis/capsulitis produce localized pain, atypical odontalgia.

Diffuse pain is one in which the location is less well-defined and somewhat vague and anatomically variable, e.g. myospasm, i.e. myogenic pain in TMJ diffuse distribution on facial area, fibromyalgia, pain originating in mucosa may be associated with widespread mucosa, systemic disease, and food impaction pain may be diffuse.

Radiating pain is one in which rapidly changing pain is there or when the pain is experienced from one part of body towards another. It is the pain that is felt from the source and all along the route of nerve, e.g. suppurative and tender gingival lesions can cause pain radiating to ear, throat and floor of mouth, glossopharyngeal neuralgia pain radiates to ear, entrapment of inferior alveolar nerve can cause pain which radiates from TMJ to mandible, chest pain may radiate to lower jaw, causes pulpitis type of pain in one or several mandibular teeth and extends as far as mental nerve outflow.

Spreading pain is one in which more gradually pain is seen, e.g. acute orofacial pain due to gingival/periodontal conditions may spread to remote areas of head/face. Neuropathic trigeminal orofacial pain usually has a narrow well-defined field (single tooth) and then spreads wider to involve more teeth. TMJ pain sometimes may occur in temples and may spread downneck/shoulders.

Lancinating pain is one in which a momentary cutting exacerbation is seen, e.g. trigeminal neuralgia, glossopharyngeal neuralgia, secondary expansion caused by contaminated amalgam can produce lancinating pain.

Enlarging pain is one in which pain progressively involves adjacent anatomic areas.

Migrating pain is one if the pain changes from one location to another, e.g. atypical facial pain, vascular type of pain.

Referred pain is a spontaneous heterotrophic pain that is felt in an area innervated by a different nerve from the one that mediates the primary pain. It never crosses midline unless it originates in the midline. Referred pain is generally referred upwards, e.g. acute orofacial pain can refer itself, i.e. acute pulpitis in decayed upper molar can refer pain to a non-decayed lower tooth, pain from lower molar is referred to ear, referred dental pain is frequently felt as headache, it may be felt in orbital or frontal area, in maxillary sinus area, in auricular/preauricular parts or throughout face. Pain due to angina is referred to as mandible. The superior part of masseter can refer the pain to TMJ and maxillary teeth while deep layer refer to mandible sternocledomastoid muscle can refer to TMJ. In oral submucous fibrosis pain in ear due to stenosis of eustachian tube.

7. What is sharp stabbing pain?

Short duration which is seen in fractured tooth or restoration, hypersensitivity of dentine, exposed dental pulp (acute pulpitis due to caries), neuralgias (glossopharyngeal, trigeminal), salivary gland duct obstruction.

8. What is projected pain? Name the condition causing projected pain in orofacial region.

It is the pain which is felt in the peripheral distribution of the same nerve that mediates the primary nociceptive input, e.g. the radicular pain of posterior root compression. Paroxysmal neuralgia, peripheral neuritis, herpes zoster and post-herpetic neuralgia.

9. What is splintting pain?

This is a protective function. It involves muscle tightening and occurs when any part of body is injured and requires rest. In orofacial region splintting of masticatory muscle may be in response to increased tension, stress, clenching, bruxism, recent dental restoration, occlusal interferences, local anesthesia, and TNJ pain. Management includes removal of cause, moist heat, rest, short course of muscle relaxants.

10. What is neuropathic pain?

Neuropathic pains are those which are generated within the nervous system itself and are due to abnormalities in components of nervous system and not the peripheral or deep structures in which the pain is felt. Neuropathic pains are associated with burning, hyperalgesia, dysasthesia and sometimes anesthesia, e.g. neuralgic pain, neuritis, stomatodynia, phantom tooth pain, etc.

11. What is formication? Enumerate the conditions causing formication in orofacial region. What is the significance of it?

Formication (from Latin word ***Formica,*** which *means* to *ant*), a neurosis that produces the sensation of snakes or insects crawling on or under the skin. It is also called parasitosis.

Orofacial complications of methamphetamine abuse may show formication in orofacial area. The other entities sometimes may show post-herpetic, post-traumatic and post-surgical neuralgias. Anesthesia dolorosa may show formication. It may be seen in regenerative phase of tissue injury. It can be seen with use of (more likely withdrawal from) substances such as cocaine, amphetamines and even alcohol.

Tinel's sign of formication is seen in variety of somatic conditions such as delirium, high fever, diabetic neuropathy, herpes zoster, peripheral nerve regeneration and neuropathy.

The significance is the sensation causes the patient to attempt to remove the perceived

parasites, usually by picking at the skin with fingernails, resulting in widespread traumatic injury. The factitial damage can alter dramatically the facial appearance in short period of time, these lesions are called speed bumps, methsores or crank bugs.

12. Enumerate the conditions of facial pain that may be a symptom of neurological disorder.

Orofacial pain associated with paresthesia, anesthesia or weakness indicate that it is associated with neurological disorders. Many of the neurological disorders which cause headache may also cause facial pain. (These are cluster headache, temporal arteritis and occasionally migraine.)

13. Give indications of opiate analgesics in orofacial region.

Opiate analgesics alone or in combination are routinely used for chronic, intractable pain. After implant surgery these are indicated with ibuprofen/acetaminophen. In oral cancer patients for control of pain, opoid analgesics should be avoided in atypical odontalgia.

14. Which are the adjuvant analgesics in orofacial pain?

These are anticonvulsants (carbamezapine, gabapentin, and pregablin), antidepressants (clonazepam, doxepin), anxiolytics (amitryptaline, nortryptaline) and muscle relaxants (tizanidine).

15. What is anesthesia dolorosa?

The denervated area continues to hurt even though it is anesthetic/report by a patient that a painful area which is numb/pain in the area which is anesthetic. It is a chronic intractable pain syndrome that may persist indefinitely. This arises after damage to the nerve or ganglion. Neurosurgery in the area may also be the cause. Or even neurolytic alcohol and glycerol blocks treatment by tricyclic antidepressants.

16. Enumerate the conditions causing paresthesia and anesthesia.

1. Injury to regional nerves:
   i. Anesthesia needles
   ii. Jaw bone fractures
   iii. Surgical procedures
2. Malignancies
3. Medications—hypnotics, tranquilizers, sedatives
4. Neuritis
5. Diabetes
6. Pernicious anemia (megaloblastic)
7. Multiple sclerosis
8. Acute infections of jaw bones
9. Psychoses
10. Trigeminal neuralgia

17. Enumerate the causes of glossopyrosis.

The preferred site for the pain is the anterior portion of the tongue. Sometimes the anterior portion of the hard palate and the labial mucosa of the lip may be involved.

- Low concentrations of vitamin B complex (notably B_{12}), folic acid and iron that can be identified by blood tests.
- Oral candidiasis (oral thrush) where small white plaques (specks) are present on the mucosa.
- Some patients report the onset of BMS after a significant 'life event' (e.g. death/separation of spouse or other major emotional shock).
- The majority of BMS patients, however, are idiopathic (unknown cause).

18. What is burning mouth syndrome? Enumerate different types of it.

Burning mouth syndrome (BMS) is characterized by burning and painful sensations in oral cavity with clinically normal mucosa with no known organic cause. The sensation is more common on tongue (tip or one side), inner surface of lips. Gingiva is more resistant. The diagnosis is by application of topical anesthetic to painful site. Treatment by analgesics for

palliative treatment, remove the cause, anti-inflammatory agents and medical consultation, if necessary.

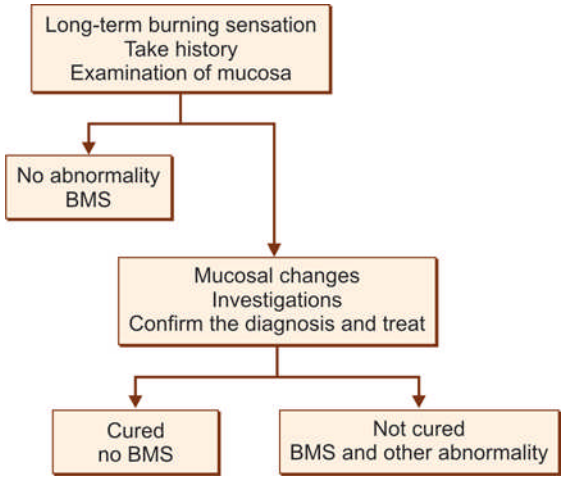
Lamey and Lewis classified into three types:

Type 1: Involves symptom-free walking, with sensations developing in the morning and progressively increasing to severe by evening.

Type 2: Involves continuous symptoms throughout the day.

Type 3: Includes intermittent symptom-free periods throughout the day.

19. What are the points considered for diagnosis of burning mouth syndrome?



20. What are different taste disturbances? Enumerate the causes of taste disturbances.

Ageusia (absence of taste), hypogeusia (blunted taste), cacogeusia (unpleasant taste), dysgeusia (altered taste sensations).

The diseases known to cause Bell's palsy, zinc deficiency, diabetes mellitus with perioral neuropathy, cancer/oral head and neck irradiation, oral candidiasis, gingivitis, periodontitis, hypothyroidism, multiple sclerosis, parkinsonism, pernicious anemia (vitamin B_{12} related), renal failure, Sjögren's syndrome, Zn-deficiency, upper respiratory disturbances, influenza.

21. What is styloid process? What are the different points considered during examination of styloid process?

The name styloid process was derived from the Greek word **"Stylos"** meaning a *pillar.* It is a bony cylindrical needle-shaped projection which originates from the postero-inferior side of petrous bone, immediately in front of the stylomastoid foramen and goes obliquely down and forward. The points during examination are length, angulation, and morphology.

1. Length of the styloid process

- Short, i.e. less than 2 cm, long 2–4 cm and elongated more than 4 cm.

2. Angulation of the styloid process

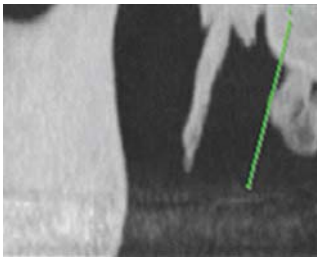
- Narrow is less than 65 degree, normal 65–75 degree and wide is more than 75 degree. It ranges from 55° to 90.5° in the transversal plane and from 76° to 110° in the sagittal plane.

3. Morphology of styloid process

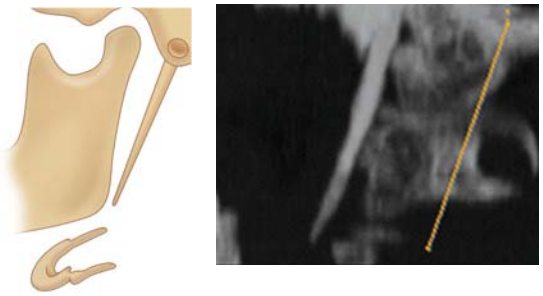
- Absence, normal appearance and other morphological findings.

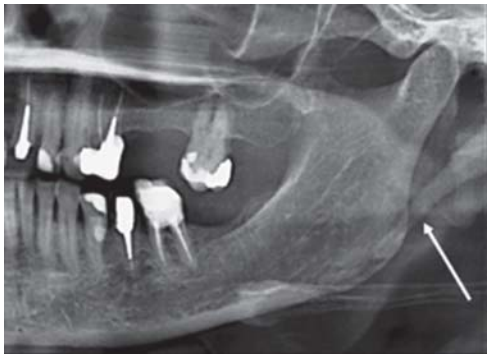
22. What are different types of styloid process?

Normal styloid process: Cylindrical bone arising from temporal bone in front of the stylomastoid foramina, which is not elongated and less than 30 mm in length.

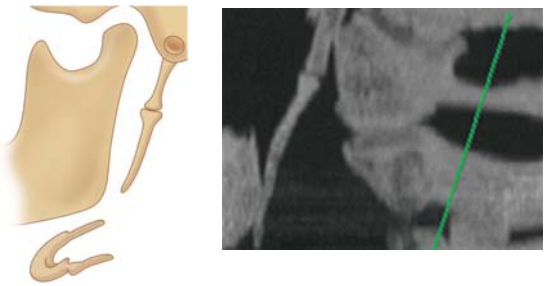


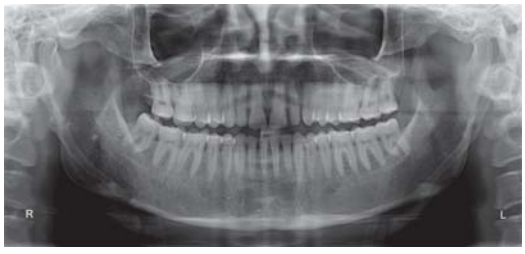
Type 1: Uninterrupted styloid process, styloid process longer than 30 mm without any interruptions.



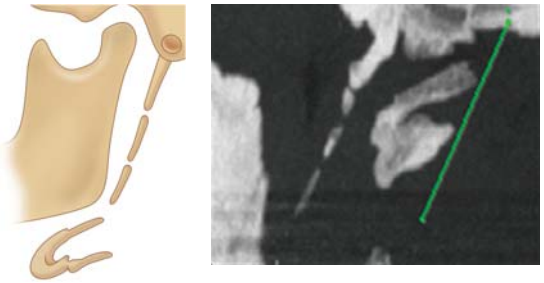


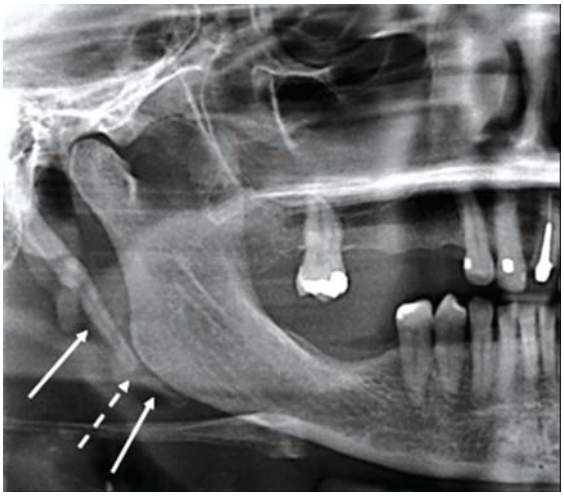
Type 2: Pseudoarticulation, it is characterized by styloid process apparently being joined by the stylohyoid ligament by a single pseudo-articulation giving the appearance of an articulated elongated styloid process.





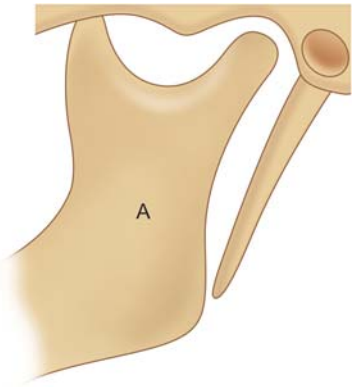
Type 3: Segmented styloid process, it consists of interrupted segments of the mineralized ligament sometimes creating the appearance of multiple pseudoarticulations.



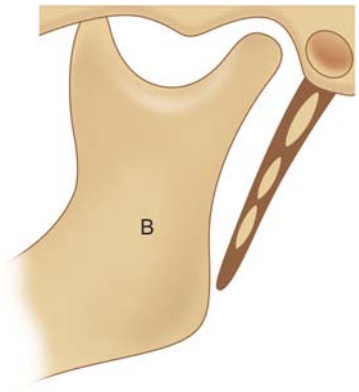


According to the Calcifications

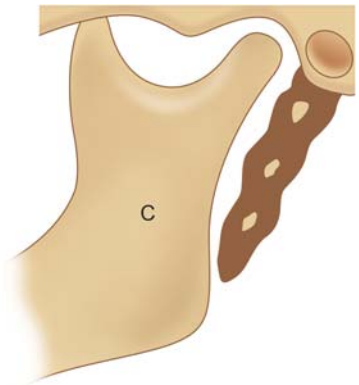
a. Calcified outline, it is also called external calcification or boundary calcification. Image of the styloid process is continuous with no signs of calcifications.



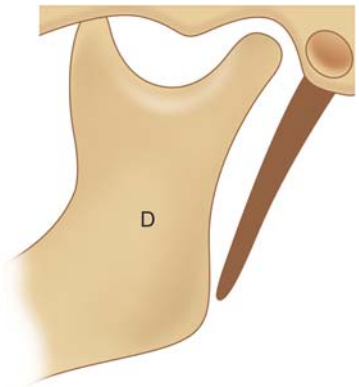
b. Partially calcified, image of the styloid process with the presence of radiopaque calcified segments.



c. Nodular, styloid process image with nodular are radiopaque calcifications.



d. Completely calcified, image of the styloid process is radiopaque, calcified and totally homogeneous.



23. What is stylagia? What are the symptoms associated with the elongation of styloid process?

Autonomous entity related to abnormal length of the styloid process or to the mineralization of the stylohyoid ligament complex. It is an important cause of paroxysmal dull pain in the throat and ear.

Symptoms are sore throat dysphagia otalgia the sensation of foreign body in the throat. Facial pain radiating to ear or along the mandible and head and neck.

24. What is Eagle's syndrome?

Eagle's syndrome is the condition in which the elongated styloid process can elicit painful symptoms when it is forced into adjacent soft tissues of the neck during normal head movements. The updated term is diffuse intraosseous skeletal hypertrophy (DISH) syndrome. This complex symptom is now recognized as only one part of a total body syndrome known as diffuse intraosseous skeletal hypertrophy, or DISH syndrome. DISH syndrome is suspected to be a reactive immune-based disease similar to myositis ossificans but targeting only ligaments.

25. Which are the other syndromes considered in differential diagnosis of Eagle's syndrome?

Trotter's syndrome (Morgagni's sinus syndrome), Costen's syndrome, myofacial pain syndrome, stylohyoid complex syndrome.

Trotter's syndrome consists of three symptoms in patients suffering from nasopharyngeal carcinoma. These are neuralgiform pain in lower jaw, deafness and blockage in ear associated with palatine asymmetry and trismus (due to pterygoid muscles).

Costen's syndrome has auricular, articular and cranial symptoms. Pain in joint, hearing is poor, dizziness, and headache.

Myofacial pain syndrome—it manifests muscular spasm, restricted mobility.

26. What are the types of Eagle's syndrome?

Eagle described two types: The classic and the styloid carotid artery syndrome.

The classic type is characterized by the symptoms like dysphagia, foreign body sensation in the neck and oropharyngeal, cervical and craniofacial pain. The pain is often

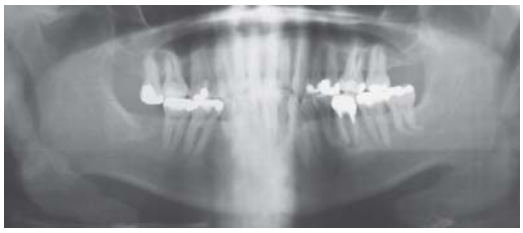
exacerbated by rotation of the head to the contralateral side, swallowing, extending the tongue, and yawning and may radiate to distant areas. The pain is referred to the ear, neck, tongue, teeth, temporomandibular joint (TMJ) area and even the chest and upper limbs. Risk factors for the development of this syndrome are an elongated or more anteriorly angulated styloid process as well as ossified stylohyoid or stylomandibular ligaments.

The styloid-carotid artery syndrome is characterized by ipsilateral headache, orbital pain and transient neurological symptoms in form of transitory ischemic attacks caused by a transient compression of the internal carotid artery and sympathetic chain. It was also suggested to distinguish three forms:

- The classical form, caused by trauma
- The styloid syndrome is the most common form, defines the condition in which the patient's symptoms appear earlier in life owing to a non-traumatic developmental anomaly in the ossification of the stylohyoid ligament or to an elongated stylohyoid process, and
- The pseudostyloid syndrome, on the other hand, defines a tendinosis at the junction of the stylohyoid ligament and the lesser cornu of the hyoid in older individuals with no history of trauma and no evidence of styloid process elongation or stylohyoid ligament ossification on radiologic examination.

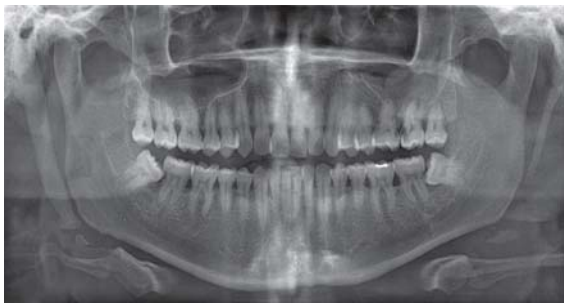
27. What is stylohyoid complex? What is stylohyoid complex syndrome?

The styloid process, the stylohyoid ligament and the lesser horn of the hyoid bone form the stylohyoid chain or stylohyoid complex. This complex traverses the area from the lateral skull base to the anterolateral neck, forming a band that crosses the upper lateral neck. The term 'stylohyoid complex syndrome' has been proposed to classify all lateral neck and/or facial pain conditions resulting from an elongated styloid process, ossified stylohyoid ligament, or elongated hyoid bone. Three different pathologies, elongated styloid process, calcified stylohoid ligament, and an elongated hyoid bone, can cause the same symptom complex. Symptoms can include lateral neck pain especially with chewing, swallowing, and head movement, and pain in the area of the submandibular space and deep to the angle of the mandible. We believe that the symptoms are related to the tension of the stylohoid complex that irritates surrounding structures and causes pain with movement of the complex. Surgery to interrupt this complex at any point is likely to improve the symptoms.



Orthopantomograph demonstrating a large radiolucent area running below, laterally and parallel to the mandible on the right side

Three-dimensional computed tomography confirming a complete and very thick hypertrophic ossified stylohyoid chain on the right side



Stylohyoid complex calcification on right side significantly

28. What are conservative and surgical treatment modalities of Eagle syndrome?

The conservative treatment involves injections of long-lasting anesthetics and/or local corticosteroid into the tonsillar fossa as well as administration of non-steroidal anti-inflammatory drugs. Other means are administration of anti-epileptics and anti-histaminics, vasodilators, neuroleptics, anti-depressants and tranquilizers as supportive therapy. Injections of corticosteroids diluted in local anesthetics close to the styloid process is another alternate. Such injections have been described for diagnostic as well as palliative purposes, via mostly an intraoral approach transpharyngeally, into the region of anterior pillar and deeply into the anterior tonsillar fossa. Even injection of long-acting local anesthetics are also used.

Manual fracture of the ossified styloid process under local anesthesia is another approach that has, however, unsatisfactory results. Also, the fracture should be regarded critically because of its vicinity to the internal carotid artery. Surgical removal of styloid process is a method of choice.

29. What are psychosomatic diseases? Enumerate the various psychosomatic diseases affecting oral cavity.

The term psychosomatic means something pertaining to mind–body relationship or having bodily symptoms of psychic, emotional or mental origin. Psychosomatic diseases are actual physical diseases which may be produced or aggravated by emotional disturbances. In general, psychosomatic disorders are harmful effects resulting from psychic influences on the organic control of tissues.

Thorakkal Shamim recently produced classification on primary symptom. This includes pain related disorders, disorders related to altered oral sensation, disorders induced by neurotic habits, autoimmune disorders, and miscellaneous disorders. Altered perception of dentofacial form and function were added to provide the revised classification.

1. Pain related disorder

a. Myofacial pain dysfunction syndrome (MPDS)
b. Atypical facial pain (AFP) and atypical odantalgia (AO)
c. Phantom tooth pain (PTP)
d. Munchausen's syndrome (MS)
e. Self-mutilation and oral artefactual disease (OAD)

2. Disorders related to altered taste sensations

a. Burning mouth syndrome (BMS)
b. Idiopathic xerostomia
c. Disturbances of taste

3. Miscellaneous

a. Recurrent aphthous stomatitis (RAS)
b. Lichen planus
c. Psoriasis
d. Geographic tongue
e. Chronic periodontal disease
f. Acute necrotizing ulcerative gingivitis (ANUG)
g. Erythema multiforme and herpes labialis
h. Bruxism
i. Body dysmorphic disorder (BDD), also known as dysmorphophobia, is an under recognized yet relatively common and severe mental disorder. These people are concerned about facial profile, teeth, chin, smiling, talking, and laughing.
j. Anorexia nervosa (AN) and bulimia nervosa (BN)

k. Delusional halitosis (DH). It is a psychosomatic condition in which some individuals believe that they have an offensive mouth odour which neither the dentist nor any other clinician can perceive. There is no local or systemic disease.

30. What is pre-trigeminal neuralgia?

This is early form of trigeminal neuralgia, characterized by a dull continuous pain (days to years) in one of jaws. It has been noted in 18% of trigeminal neuralgia patients. It may be mistaken for dental pathology (toothache/sinusitis pain) by throbbing pain caused by thermal stimuli. The pain may be aching type with several hours duration. The pain is spontaneous but with no trigger zones. The duration of PTN pain may be in minutes or hours or in some cases constant in comparison to the pain in seconds/minutes as in classic TN. It is responsive to carbamezapine.

31. What is atypical neuralgia?

Post-traumatic trigeminal neuralgia combines characteristics of both painful neuritis and paroxysmal neuralgias. The disorder has persistent, unremitting, variable bright, burning pain that suggests painful neuritis and it may be accompanied by other sensory, motor and/or autonomic effects. The basic background neuritic pain may be interrupted by paroxysms of neuralgic pains. This is called atypical trigeminal neuralgia.

32. What is para-trigeminal neuralgia?

It is also called reader's syndrome. It is characterized by frontotemporal pain and occulosympathetic paresis. Tic like pain sensation in the first and second division of trigeminal neuralgia. The pain may be described as intense or throbbing, there may also be drooping of eyelid and contraction of pupil (miosis). Some experience dysgeusia possibly due to involvement of chorda tympani nerve.

33. What are the doses of carbamazepine? How are they started? What care has to be taken?

Carbamazepine is the primary drug used in the medical management of pain of trigeminal neuralgia. A starting dose of 200 mg everyday is recommended; this can be increased by up to 200 mg everyday as tolerated against side effects, to a maximum of 800 mg per day. The dosage must be regulated for best therapeutic effect, and a baseline complete blood count is recommended because of its dose-related effect of bone marrow suppression. To keep the tegretol dose low and reduce side-effects if they occur, gabapentin, 300 mg three times daily, or divalproex sodium 50 mg everyday, and/or amitriptyline, 50 mg by mouth at bedtime, may be added to the tegretol.

34. Why routine blood investigations are advised during carbamazepine therapy?

The carbamazepine is given 100 to 200 mg twice daily. Usual maintenance day is 600–1200 mg/day, in divided doses until the symptoms subside. The common side-effects are drowsiness, dizziness, nausea, anorexia. Rarely complications like aplastic anemia, leucopenia and agranulocytosis may occur. Periodic complete cell blood count, liver and kidney function tests are necessary in these patients. Complete blood count every two weeks during first two months and quarterly thereafter is recommended. The complete blood count has to be done because of its dose-related bone marrow suppression.

35. What is oromandibular dystonia?

It produces involuntary, excessive contractions of tongue, lip and jaw muscles. The proposed pathogenesis is related to defective inhibitory control of basal ganglion of the forebrain, thalamus and brainstem. Treatment by injection of botulinum toxin and neurosurgical interventions.

36. What is orofacial dyskinesia?

These are abnormal, involuntary movements of tongue, lips and jaws. This may be a

contributing factor for TMJ degenerative changes, mucosal lesions, damage to teeth and prosthesis. Complete loss of teeth is considered to be one cause of oral dyskinesia. Lack of replacement and ill-fitting dentures may initiate it. Clinically, characterized by observation of involuntary mouth movements and their effect on jaw muscles, TMJ, oral mucosa and teeth. Emphasis on prevention as no treatment is safe.

Tardive dyskinesia is associated with antipsychotic medications. Tardive dyskinesia has been reported to cause facial pain. It may be persistent even after drug therapy is stopped. The management is by changing medications with discussion with physician and clonazepam, baclofen.

37. What are trigger points?

Myofascial trigger point

A hyper-irritable spot, usually within a taut band of skeletal muscle or in the muscle fascia, that is painful on compression and that can give rise to characteristic referred pain, tenderness (secondary hyperalgesia), motor dysfunction and autonomic phenomena.

From the trigger points masticatory impulses pass into CNS and return in the form of painful sensations on some other structures. Such places where the patients feel pain sensation are called zones of impact (zone of referral, referral zone).

38. What are trigger zones?

Pain in TN is precipitated by light touch on a "trigger zone" present on the skin or mucosa within the distribution of the involved nerve branch. Common sites for trigger zones include the nasolabial fold and the corner of the lip. Shaving, showering, eating, speaking, or even exposure to wind can trigger a painful episode, and patients often protect the trigger zone with their hand or an article of clothing. Intraoral trigger zones can confuse the diagnosis by suggesting a dental disorder, and TN patients often first consult a dentist for evaluation. The stabbing pain can mimic the pain of a cracked tooth, but the two disorders can be distinguished by determining whether placing food in the mouth without chewing or whether gently touching the soft tissue around the trigger zone will precipitate pain. TN pain will be triggered by touching the soft tissue, whereas pressure on the tooth is required to cause pain from a cracked tooth (Burkitt's 12th ed.).

39. What are the characteristics of hypertension headaches and tension headache?

Headache in the occipital area may be due to severe hypertension. The nature is pounding like. This is often accompanied by blurred vision, ringing in ears, tingling in hands/feet, shortness of breath and fatigue.

The pain of tension type of headache is bilateral, dull, aching and sometimes band like. Increases with age and is more common in females. The patient may report an increased tenderness of pericranial muscles and its attaching fascia. It gets relieved by rest and anxiolytic medicines and rest.

40. Why TMJ is called synovial joint?

The internal surfaces of superior and inferior joint cavities are surrounded by specialized endothelial cells that form a synovial lining. This lining along with a specialized synovial flinge located at the anterior border of the retrodiscal tissues produces synovial fluid (serous in nature) which fills both joint cavities and the TMJ is referred to as synovial joint. The synovial fluid serves by providing metabolic requirements to the articular disc which is avascular and as a lubricant during function. This also has bacteriocidal property.

41. What are functions of inferior and superior lateral pterygoids?

The superior and inferior lateral pterygoid muscles are present anterior to condyle disc complex. The inferior pterygoid muscle inserts on neck of condyle, whereas superior lateral pterygoid muscle inserts on neck of condyle and the articular disc. Superior lateral pterygoid is considerably smaller.

The inferior lateral pterygoid muscle is active with depressing muscles, i.e. mouth opening.

Bilateral inferior pterygoid muscle contraction simultaneously results in mandibular protrusion. Unilateral contraction creates a mediotrusive movement of that condyle and causes a lateral movement of mandible to opposite side.

Superior lateral pterygoid muscle is shown to active with elevator muscles, i.e. mouth closing.

The superior lateral pterygoid muscle is a stabilizing muscle for condyle disc complex specially during unilateral chewing. It helps slight anterior and medial force on disc. It helps in power stroke (the movement that involves closure of mandible against resistance such as in chewing or clenching the teeth together) and when the teeth are held together.

42. What are temporomandibular disorders?

Temporomandibular disorders are the functional disturbances of masticatory system. This is a collective term used for a number of clinical problems that involve the masticatory muscle, temporomandibular joint and/or associated structures.

43. What is synovitis?

Inflammation of synovial lining or the synovial tissues that line the recess areas of the joint become inflamed. The pain is localized and enhanced with joint movement. It may cause swelling due to effusion within joint cavity and alteration of synovial fluid. It is caused by irritation within joint, localized trauma, abusive use, toxemias, specific infections or an allergic response. It may occur as a manifestation of arthritis.

44. What is capsulitis?

Inflammation of capsular lining or capsular ligament becomes inflamed. Tenderness on extra-auricular palpation. Occasionally fluctuant swelling as well. The pain is localized and even in static joint position. The joint movements increase the pain. The pain is not increased by clenching the teeth nor by bitting against separator. Pain occurs due to inflamed capsule is stretched by translator movement of the condyle and therefore is accentuated by protrusion, contralateral excursion and on wide opening. Macrotrauma and injury in open mouth position. It may also develop to adjacent tissue breakdown and inflammation. It may occur due to habits that entail excessive mandibular movements and from abusive joint hypermobility.

45. What is retrodiscitis?

Retrodiscitis is inflammation of retrodiscal tissue. It is characterized by dull aching pain often increased by clenching (as the retrodiscal tissues are highly vascularised and innervative and are not in a position to tolerate the loading forces) and such pain is decreased by biting against a separator on the same side. The pain is increased by forced ipsilateral excursive movements of mandible. Trauma is major etiologic factor, macrotrauma in open mouth, blow to chin causing the forceful movement of condyle on retrodiscal tissues.

Acute malocclusion may be caused as swelling may occur and force the condyle slightly forward down the posterior slope of the articular eminence. Intraorally seen as disengagement of ipsilateral posterior teeth and heavy contacts of contralateral canines.

The inferior lateral pterygoid muscle pain may produce same symptoms but can be differentiated by absence of pain in resisted protrusion of mandible in retrodiscitis.

46. What are different joint sounds?

These are also called TMJ joint noises. Articular disc displacements are more common cause of joint noises. Clicking, popping, snapping and crepitus are different types of joint noises. Clicking single, brief event, e.g. articular disc displacements. Pop is usually loud click that is audible click and may be very annoying to the patients. It is also described as loud noise or thud. It usually occurs during maximal mouth opening, it is seen in joint hypermobility. Pop is also noted during maximum

opening in adherence when patient tries to relief stiff which has occurred. Popping is usually reversible and indicates an internal derangement, it may suggest osteophyte or tumor. Snapping sound is noted in disc displacement with reduction. Crepitus (crepitations) is long-grating sound, e.g. degenerative diseases. This is multiple, rough, gravel like sound and is complicated.

Subluxation of TMJ mouth opens to fullest extent, a momentary pause occurs, followed by sudden jump or leap to maximally open position. This jump is accompanied by a thud, sound which needs to be differentiated from click.

47. What is end feel test?

End feel test is done when mouth opening is restricted. The end feel describes the characteristics of the restriction that limits the full range of joint movement. A normal range of mouth opening is considered 53 to 58 mm. A restricted mouth opening is considered to be any distance less than 40 mm.

The end feel can be evaluated by placing the fingers between the patient's upper and lower teeth and applying gentle and steady force in an attempt to passively increase the interincisal distance.

Soft end feel is said if the opening can be achieved and has to be done slowly. The pain which is present is intensified. This suggests muscle-induced restriction.

Hard end feel is said to be if no opening is achieved. This suggests intracapsular sources (disc dislocations), acute non-reducing disc displacements obstructing translation, unilateral ankylosis.

Hard end feel may be suggestive of surgical intervention while soft end feel suggest no indication of surgical intervention.

48. What is restricted mouth opening?

The normal range of mouth opening when measured interincisally is between 53 and 58 mm. The patients having extreme deep bites vertical overlap of the anterior teeth must be considered when determining normal range of movement. A restricted mandibular opening is considered to be any distance less than 40 mm.

49. Enumerate the causes of restricted movements of mandible.

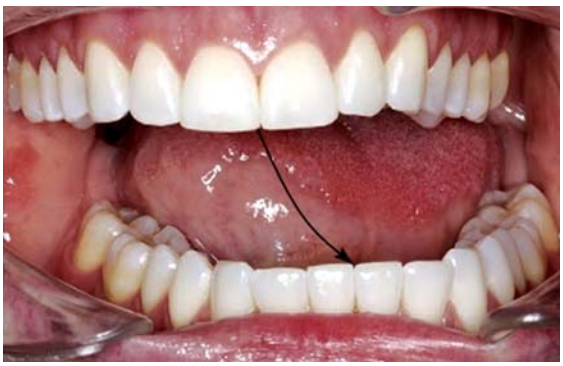
These are extracapsular or intracapsular sources. Extracapsular causes are related to muscle disorder. The intracapsular are due to disc–condyle function and surrounding ligaments and are related to disc derangement disorder.

The extracapsular muscle restrictions are due to elevator muscle (masseter/medial pterygoid) spasm and pain. These muscles restrict the translation and thus limit opening. There is no effect on lateral and protrusive movements. Soft end feel is present and they present deflection.

The intracapsular restrictions are due to disc derangement disorder. The movement is restricted due to structural resistance. The muscle also restricts the translation. They present deflection.

50. What is deflection?

The opening pathway is shifted to one side and becomes greater with opening. At maximum opening the midline is deflected to its greatest distance or it can be defined as any shift of midline to one side that becomes greater with opening and does not disappear at maximum opening. It is due to restricted movement in one joint.



Deflection on ipsilateral side is seen on capsular fibrosis, degenerative joint disease, periarticular and capsular inflammation and fibrous ankylosis.

Deflection on contralateral side is seen due to immobilization of TMJ due to invasive malignant tumors.

51. What is deviation?

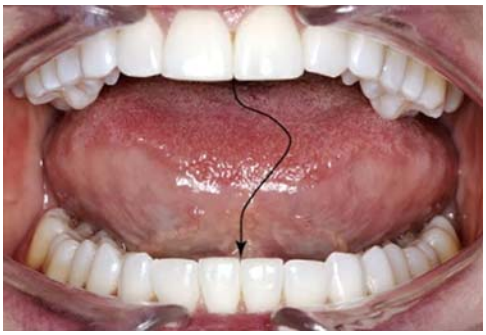
The opening pathway is altered but returns to normal midline relationship at maximal opening or it can be defined as any shift of jaw midline during opening that disappears with continued opening (a return to midline). It is the result of condylar movement necessary to get past the disc during translation. Once the condyle has overcome this interference the straight midline path is resumed. This is seen in disc displacement with reduction, structural incompatibility and muscle disorders (muscle engrams), subluxations.

The deviation seen in disc displacement with reduction (abnormalities in discal movement), the speed of opening alters the location of deviation while the one seen in structural incompatibility speed of opening and closing does not alter the interincisal distance and location of deviation.

Deviation caused by muscle disorders are commonly large, inconsistent, sweeping movements not associated with joint sounds. Ipsilateral deviation is seen in unilateral hypoplasia of mandibular condyle and unilateral bony ankylosis. Contalateral deviation is seen in unilateral hyperplasia of condyle.

52. How the examination for joint sounds is done?

1. **Palpation:** Place the fingers over lateral surfaces of joints and ask the patient to open and close. These can be perceived by fingertips. Examination of joint sounds by placing in the ear is not advised, as additional sounds may noted by the tissue

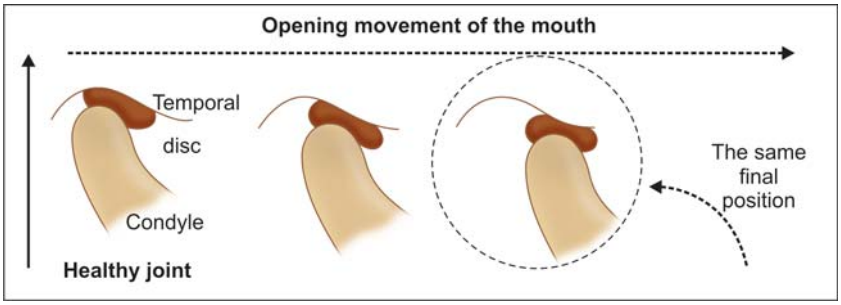


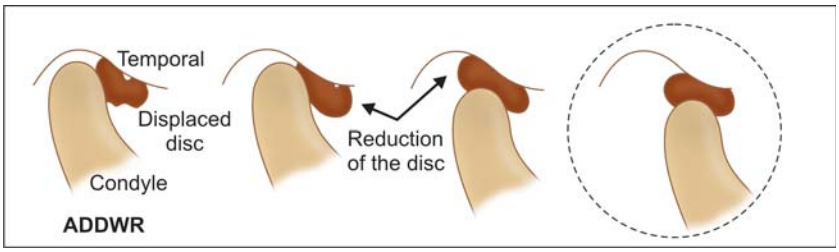
of ear canal cartilage is forced against the posterior border of condyle and this force displaces condyle and produce additional sounds.

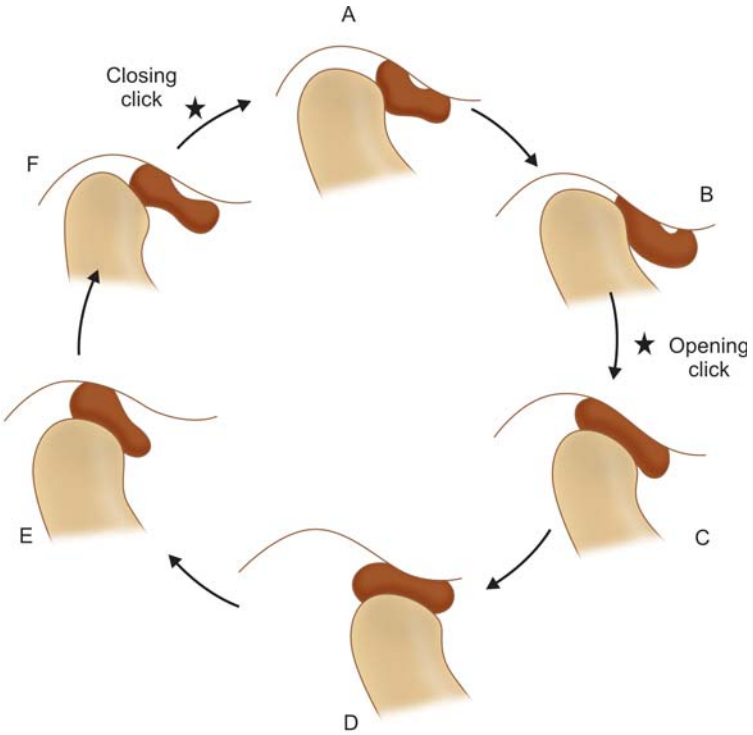
2. **Auscultation:** This is done by stethoscope. This may detect more sounds.
   Note character of sound, degree of interincisal opening with sound, whether it occurs during opening and closing.

53. What is disc displacement with reduction?

The disk is displaced from its position between the condyle and eminence to an anterior and medial or lateral position (more common is anterior or anteromedial) but is reduced in full mouth opening usually resulting in a noise.



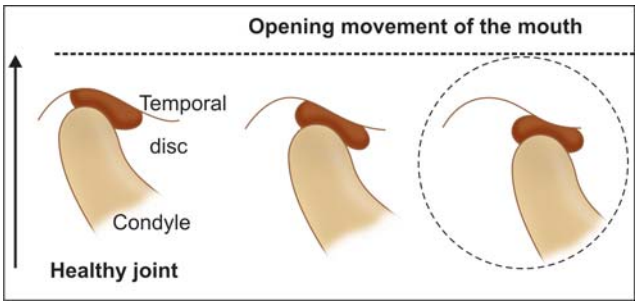


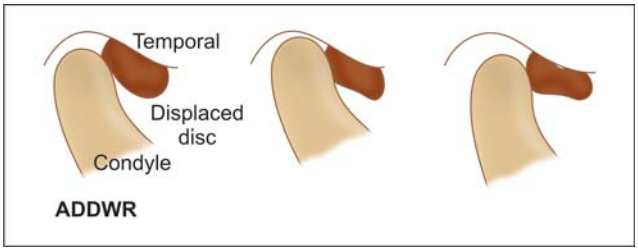


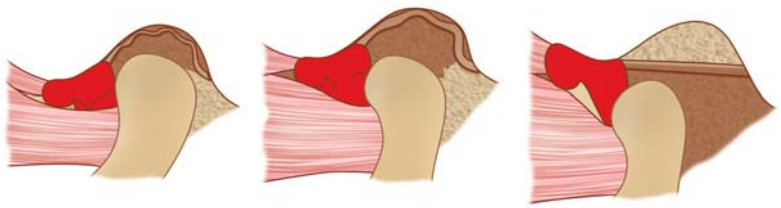
Click occurs on both vertical opening and closing. The opening click is at least 5 mm greater than on closing and this click is eliminated on protrusive opening. It may cause a popping sound also. Pain may be present when chewing hard foods. This condition can be seen in chronic clicking condition in patients who tend to clench and grind the teeth at night and in patients with missing posterior teeth with subsequent overclosure of bite. It does not require treatment if the patient can open reasonably wide without discomfort, if pain occurs mild analgesics can be used.

54. What is disc displacement without reduction?

Disc displacement (medially, laterally and anteriorly) does not assume the normal position. It is also called closed lock. In this case the disc has been permanently displaced and its shape has been deformed so that it prevents the condyle of the mandible from translating to a full open position. It can be seen with and without limited opening. It is diagnosed mainly by absence of joint sounds and deflection. If the disc displacement is associated with limited opening, then maximum unassisted opening will be less than 35 mm and







may increase passively by less than 4 mm and contralateral movement less than 7 mm. Pain and change in patients perception of the bite generally results.

If it is associated without limited opening maximum unassisted opening is more than 35 mm and passively increases by more than 5 mm and contralateral movement is more than 7 mm. The treatment involves analgesics and splints may help.

55. What is click? What are different types of clicks? State in which conditions click is seen.

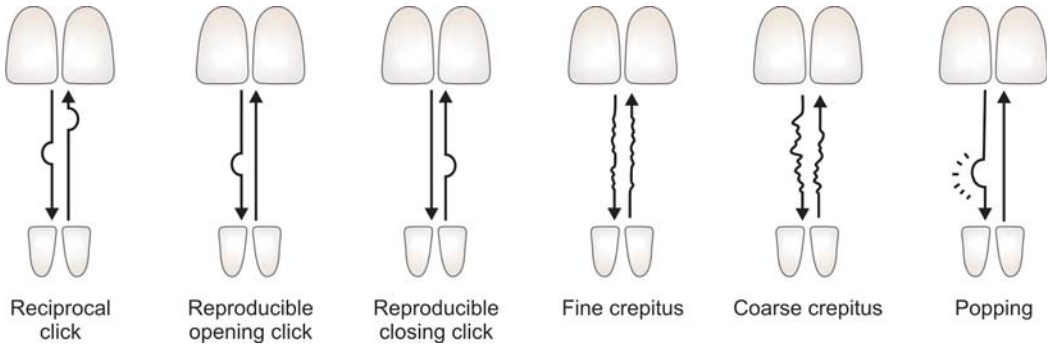
The joint sound if it is a single, brief event of short duration is called click. Articular disc displacements (ADD) are thought to be most common cause of clicking. Clicking can be seen in individuals who have normal disc position on MRI, condylar hypermobility, enlargement of the lateral pole of the condyle, structural irregularities of the articular eminence, loose intra-articular bodies than discs and dysfunctional movements, patterns or incordination. Single click during opening represents the early stages of internal derangements. Click can also be seen during early stage of osteoarthritis. Retrodiscitis produced by intrinsic trauma may produce clicking. Clicks can also occur during adherence (i.e. temporary sticking of articular surfaces), shows single click while breaking the adherences and can be differentiated from disc displacements by the fact that they occur only once following a period of static loading.

Reciprocal click is one in which click is noted during opening and closing of jaws, this is seen in disc displacement with reduction. In this a sound is heard during mandibular opening when condyle is moving across the posterior

border of the disc to its normal intermediate zone and closing click occurs near the closed or intercuspal position. Structural incompatibility of articular surface of joints adherence can occur and reciprocal clicks can be seen, here the clicks while opening can occur where the mandibular opening is disrupted and while closing, it occurs at the same interincisal opening while disc displacements show clicks at different interincisal opening, this is differentiating point.

Reproducible clicks are the sounds which are present consistently on clinical examination and not only as patient complaints. Reproducible opening click (if they appear on two or three openings from maximum intercuspation) while reproducible closing clicks if they appear on two or three closing.

56. Give the schematic representation of TMJ sounds.



57. What is arthritis? What are the different conditions affecting the temporomandibular joints?

Inflammation of the articular surfaces of the temporomandibular joints is referred as arthritis. The different arthritic conditions are osteoarthritis, osteoarthrosis, polyarthritides. Osteoarthritis is more common.

58. What are differentiating points between rheumatoid arthritis and osteoarthritis?

Soft tissue swelling rather than bony enlargements is a characteristic difference between OA and RA.

Table 2.1: Difference between osteoarthritis and rheumatoid arthritis

	Osteoarthritis	*Rheumatoid arthritis*
Age of onset	It is <40 years	Any age after 20 years
Prevalence	Much more common	Less common (1/10)
Cartilage breakdown	It is deterioration of cartilage and becomes thinner and overgrowth of bone spurs, due to age (wear and tear)	It is related to body's own immune system, causes inflammation of connective tissue in joints (synovial membranes) that leads to destruction of articular cartilage.
Pain	It gets worse at the end of day or after exercising	
Symmetry	Symmetry not necessarily present in osteoarthritis	If one joint is involved, the other side is also affected.
Duration of morning stiffness	<15 minutes (it becomes worse after sitting down)	>40 minutes
Joint involvement	– One or two joints – Irregular involvement at distal interphalangeal joint	– Diffuse involvement – Symmetric involvement

Contd.

Table 2.1: Difference between osteoarthritis and rheumatoid arthritis *(Contd.)*

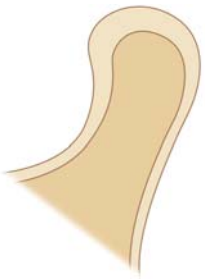
	Osteoarthritis	*Rheumatoid arthritis*
Types of joint involvement	Weight bearing joints (knees, hips)	Small joints (hands, wrists, elbows)
Rate of development	Can take many years to develop	It can come in months
Inflammation	Mild/absent	Severe
Anemia	Rarely	More common
Tenderness	None/mild	Moderate/severe
ESR	Not raised	Raised
ANA	–ve	+ve
Rheumatic factor	Absent	Present
Radiographic appearance	Bony lumps and other deformities	Bone looks thin and may show erosions
Treatment modalities	NSAIDs (acetaminophen, aspirin, ibuprofen, Cox-2 inhibitors) dietary supplements to prevent glucosamine/chondratin	glucocorticoids (prednisone) and immunosuppression agents in addition to NSAIDs dietary supplements fruits, vegetables and fish

59. What is crepitus? State in which all conditions crepitus is seen.

Crepitus is long grating sound (grinding/gritty feeling). It is irreversible. It is a multiple rough gravel like sound. It is seen in arthritides (diseases which involve destructive bony changes), it indicates bone to bone contact. The more common is in degenerative diseases (osteoarthritis) and suggest lack of synovial fluid to adequately lubricate joints. It is seen in later stage of internal derangement.

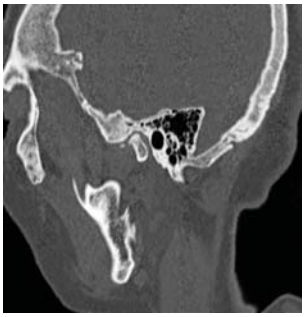
60. Enumerate the various degenerative changes involving condyle.

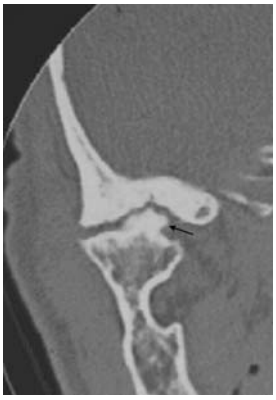
Normal condyle



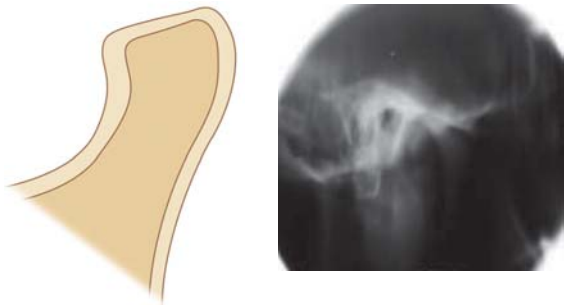
The degenerative changes are joint deterioration and bone proliferation.

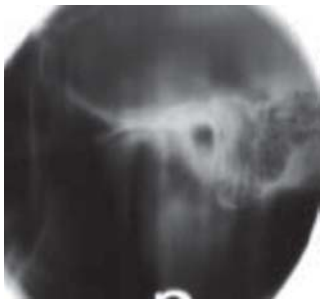
1. **Osteophyte formation:** New bone formation at periphery of articular surface (proliferative component). They are seen on anterior superior surface of condyle and lateral surface of condyle. These may break off and lie within the joint space and called as joint mice (deterioration component).



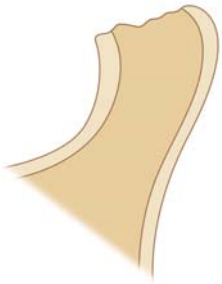


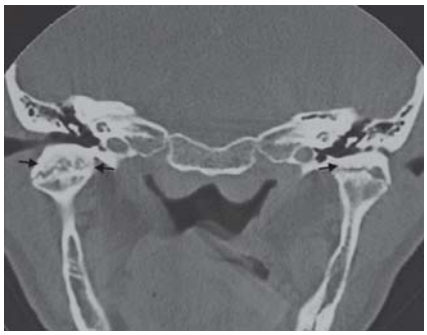
2. **Flattening of condyle (deterioration component and even sometimes flattening of articular eminence):** These are suggestive of remodelling in absence of other features.



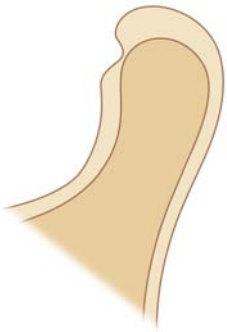


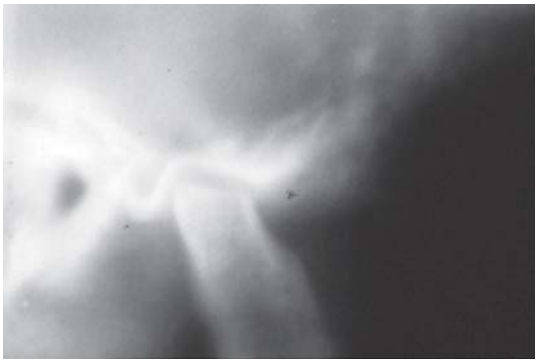
3. **Irregularity on condylar surface:** (Deterioration component—irregularity and erosion)



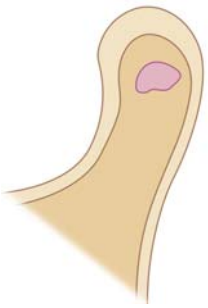


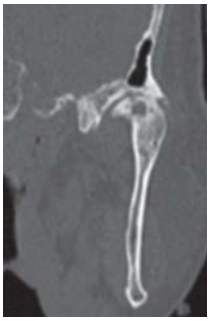
4. **Erosion of condylar head:** Small to large bites or scoops out of the articulating surfaces of the joint resulting in loss of continuity of cortices and eventually bone volume (deterioration component—severe arthritis erosion).

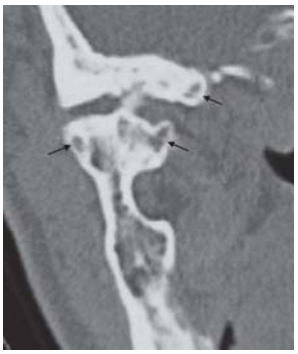




5. **Ely's cyst formation:** Ely's cysts are not true cyst. These are areas of degeneration that contain fibrous tissue, granulation tissue

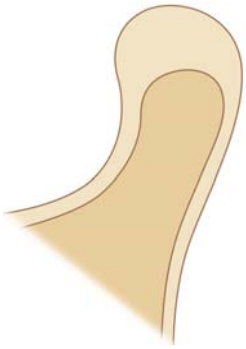


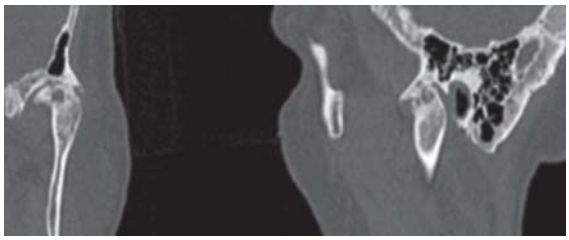




and osteoid. Radiographically small, round, radiolucent areas with irregular margins surrounded by increased areas of bone density deep to articular surfaces (proliferative component).

6. **Sclerosis of head:** New bone formation in subchondral region (proliferative component).





61. What are stages of osteoarthritis? What are primary and secondary osteoarthritis?

It is a degenerative disease of joint. Long-term studies have shown the disc displacements and osteoarthritis can pass through three stages:

1. The first stage of clicking/catching
2. Mandibular movement restrictions and pain
3. Decrease in pain and joint sounds are present. Osteoarthritis is supposed to be caused by mechanical overloading of joint. If the cause of osteoarthritis is unknown, it is called primary osteoarthritis. If the prescise cause can be identified it is called secondary osteoarthritis. Disc dislocation without reduction can cause osteoarthritis.

62. What is condylysis?

It is condylar resorption. It can be defined as progressive loss of condylar shape with decrease in mass. It primarily affects the adolescent girls. It involves sudden lysis of condyle that creates a rapid change in the bite of individual resulting in open bite even that is noticed by an individual. The occurrence of progressive overjet is suggestive of condylysis. As a result, most patients exhibit a decrease in posterior face height, retrognathism and progressive anterior open bite with clockwise rotation of the mandible.

63. Define internal derangement of TMJ and describe its various stages.

Internal derangement of TMJ is defined as an abnormal positional relationship of the disc relative to the mandibular condyle and the articular eminence.

OR

Internal derangement of the temporomandibular joint (TMJ) is defined as a disruption within the internal aspects of the TMJ in which there is a displacement of the disc from its normal functional relationship with the mandibular condyle and the articular

Stage	*Clinical*	*Imaging*
I. Early	Painless clicking; no limitation of opening. The disc is reduced to maximal opening which refers to sliding back and corresponds to audible click.	Mild disc displacement (on pening) with early reduction; normal disk osseous contours are normal
II. Early/Intermediate	Occasional painful clicking, intermittent locking.	Mild to moderate disk displacement with late reduction, mild disc deformity. The osseous contour remains normal.

Contd.

Stage	*Clinical*	*Imaging*
III. Intermediate	Joint tenderness, limited mouth opening, frequent pain, painful chewing. Disc is subjected to deformity, as condyle pushes disc downward and forward.	Displaced, non-reducing disk moderate thickening of disc. Early stage it may reduce and in later stage may not. The osseous contour remains normal.
IV. Intermediate/late	Chronic pain, limited opening	Markedly thickened disc, severe displacement without reduction, degenerative osseous change and abnormal contours in condyle and articular eminence.
V. Late	Variable joint pain, joint crepitus, pain with mandibular opening	Grossly deformed disc, non-reduction of disk with perforation of disk attachment or disk degenerative osseous changes. Deterioration and abrasion of articular cartilage and disc surfaces, occurrence of thickening and remodelling of underlying bone.

Osteoarthrosis may be a final common pathway for several joint conditions.

portion of the temporal bone. These are the disturbances in the arrangements of components within joint itself primarily disc. It is most common disorder of TMJ. These derangements are clicking joint type or locking joint type.

Wilkes (1989) established 5 stages based on clinical and imaging criteria.

64. What are the different treatment modalities for internal derangements?

The internal derangements are treated with non-surgical methods and when these methods are unsuccessful they are managed by surgical methods.

1. **Non-invasive methods:** Non-surgical methods such as modification of the diet, occlusal splints, physical therapy, pharmacotherapy, transcutaneous electrical nerve stimulation (TENS) and stress reduction technique.
2. **Physical therapy:** It is used to keep the synovial joint lubricated, and to maintain the jaw motion. One of the exercises for the jaw is to open the mouth to a comfortable fully-open position and then apply a slight additional pressure to open the mouth fully. Another exercise includes stretching the jaw muscles by doing various facial expressions. Avoiding extreme jaw movements, taking medications, applying moist heat or cold packs, eating soft foods are other ways that may keep the disorder from worsen.
3. **Splints:** They are plastic mouthpieces that fit over the upper and lower teeth. They prevent the upper and lower teeth from coming together, lessen the effects of clenching or grinding the teeth. Splints are effective in reducing the intensity of pain for patients with pain in jaw and masticatory muscles by compensating for or correcting perceived bite defects of the sufferer.
4. **Pharmacotherapy:** The pharmacologic agents which are commonly prescribed non-steroidal anti-inflammatory drugs (NSAIDs) to reduce inflammation. Muscle relaxants also prescribed for treatment of muscle pain and spasm. To increase their benefit, muscle relaxants combination with NSAIDs are used.
5. **Arthroscopy:** Lysis of adhesions and joint lavage are the most commonly performed TMJ arthroscopic surgical procedures to relieve painful hypomobility. The objective is to eliminate

restrictions on the disc and lateral capsule, to wash out micro-debris resulting from the breakdown of the articular surfaces, to irrigate the joint by enzymes and prostaglandins and to stimulate the normal lubricating action of the synovial membrane. In addition, the presence of fibrous adhesions in the superior joint space limits normal translatory function of the disc condyle complex.

6. **Arthrocentesis:** It is the simplest and minimal invasive form of surgery in the TMJ, aim to release the articular disc and to remove adhesion between the disc surface and the mandibular fossa by means of hydraulic pressure from irrigation of the upper chamber of the TMJ. This is very effective procedure in patients with persistent or chronic closed lock and anchorage in the upper articular space. Lavage of superior joint space with saline exerts its effects through its ability to eliminate joint effusion to reduce the symptoms. It is considered as an intervening treatment modality between non-surgical treatment and arthroscopic surgery. Being the least invasive and simplest form of surgical interventions into the TMJ, this procedure carries a very low-risk and relatively easy to proceed in dental chair office under local anesthesia alone or in combination with conscious sedation.
7. **Dissectomy and disc replacement:** Dissectomy used to regain the mandibular motion and to reduce orofacial pain, and may be followed by disc replacement.
8. **Joint reconstruction:** Several techniques have been proposed for re-construction of portions of the joint or the entire joint itself. A hemiarthroplasty may be used to replace the superior articulating joint surface. During re-construction, joint adhesions are lysed and a vitallium alloy fossa—eminence prosthesis, manufactured by TMJ implants, is implanted to replace the temporal component of the joint.
9. **Intra-articular:** Injection of corticosteroid intra-articular injection of corticosteroids alone or after arthrocentesis provides long-term palliative effects on subjective symptoms and clinical signs of TMJ pain. Recently, sodium hyaluronate (SH) has been proposed as an alternative therapeutic agent which is high viscous, high molecular substance plays an important role in joint lubrication and protection of the cartilage.
10. **Low level:** Laser therapy clinical studies of LLLT used on patients with disc derangement disorders using either AlGaAs 830 nm diode laser in continuous wave mode or He-Ne laser 632 nm combined with a diode laser 904 nm in pulsed mode have shown clinical benefits in terms of reduction in pain and clicking. The application of laser beams diminishes pain while simultaneously reducing muscle contraction. The main effects of laser light used in LLLT on tissue include: Analgesic, biostimulating, anti-inflammatory, etc. Advantages include aseptic, non-invasive, painless, non-pharmaceutical and reversible therapy, if used properly has no side-effects. It has no post-operative discomfort. Disadvantage has been the high-cost compared to the conventional therapies and the fast development in the field
11. **Tissue engineering of the TMJ:** The disc shows biomechanical properties that may be matched more easily in tissue-engineered constructs in contrast to other musculoskeletal soft tissues.

    Currently low level laser therapy and tissue engineering show long-term promise for meeting this demand.

65. What is anchored disc phenomenon?

It is characterized by sudden, severe and persistent limited mouth opening that is characterized by more than disc displacement without reduction (10–30 mm). Since the disc

is not anatomically displaced the highly innervated retrodiscal tissue is not compressed and pain is experienced during forced mouth opening. It is acute disc displacement without reduction.

66. What is coronoid impedance?

It is a hypomobility disorder. During the mandibular movement the coronoid process will pass between lateral surface of maxilla and zygomatic arch anterioinferiorly if its pathway is impeded it will not slide smoothly and restriction of movements will occur. This is called coronoid impedence. Clinically limitations especially during protrusion generally painless. If it is unilateral the mandible will deflect to affected side. The causes are:

a. Elongation of coronoid process (result of chronic temporalis activity)
b. Tissure fibrosis (may be because of trauma during surgery or prior infection)
c. Fractured zygomatic arch displaced medially.

Treatment is remove the cause and gentle passive stretching with ultrasound.

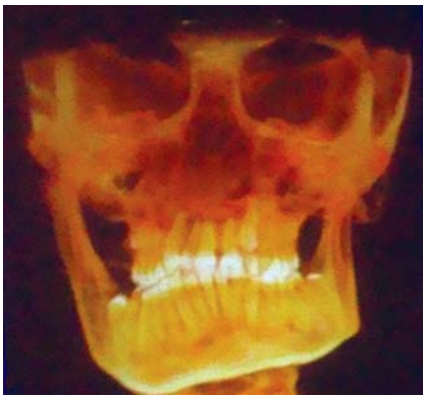
67. How the coronoid hyperplasia diagnosed? What is the radiographic sign?

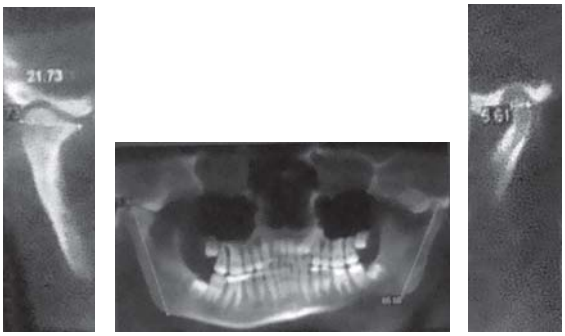
Radiographically the coronoid process extends 1 cm above the zygomatic arch and restricting the mouth opening. The appearance is called as drumstick appearance on OPG or PA waters view.

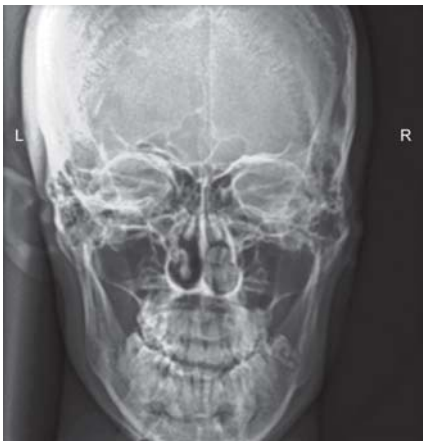
68. What are the features of condylar hyperplasia?

Developmental anomaly, growth self-limiting and stops with completion of skeletal growth.

- Mandibular asymmetry and chin deviates to unaffected side.
- The movements may be restricted.
- A depression may be noted at inferior border of mandible where affected side meets unaffected side.
- Ramus vertical height is increased.
- Radiographically normal cortical thickness and trabecular pattern may be seen (this distinguishes from condylar tumor)







PA view showing right condyle elevated

69. What is anterior positioning appliance?

An interocclusal device that encourages the mandible to assume a position more anterior than intercuspal position (ICP). It is indicated to treat disc displacements, disc dislocations with reduction. Patients with single or reciprocal click. Intermittent or chronic locking of joints. Retrodiscitis, an inflammatory disorder,

may be managed as anterior positioning may be comfortable. The goal of treatment is to change the position temporarily so as to enhance the adaptation of retrodiscal tissues.

70. What is palpation? Describe various types of palpation methods used for orofacial structures.

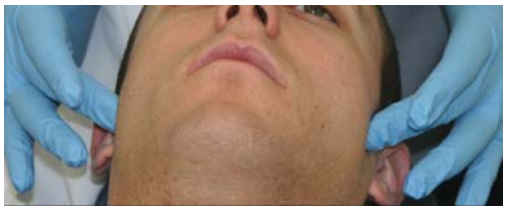
Palpation is a procedure where examiner feels or presses the structures examined. It gives more details about texture, dimension, consistency, temperature and functional events.

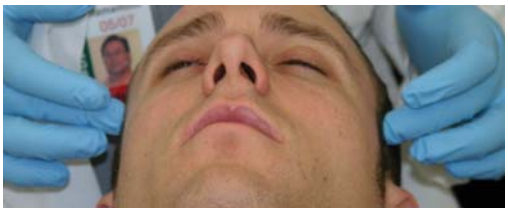
Extraoral palpation: The structure is palpated extraorally, e.g. parotid gland palpation, skin over surface of swelling.

Intraoral palpation: Structure is palpated intraorally, e.g. lateral pterygoid examination.

Unilateral palpation: Structure of one side is palpated at a time, e.g. submandibular lymph node, palpation of lateral pterygoid intraorally.

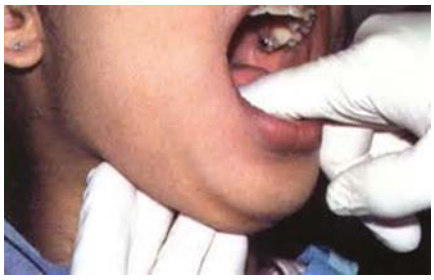
Bilateral palpation: Same structures on both sides are palpated. Temporalis muscle, TMJ palpation (extra-auricular/intra-auricular), carotid artery not to be bilaterally palpated to avoid carotid sinus stimulation and brady-cardial reflex.



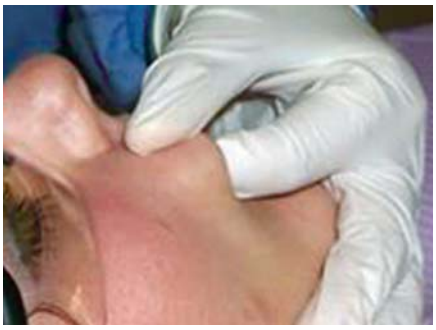


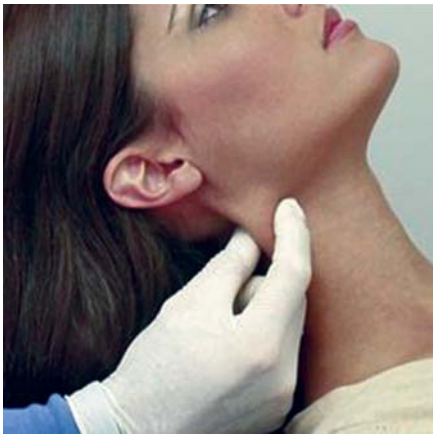
Bilateral palpation of TMJ and temporalis

Bimanual palpation: One structure is palpated by fingers of both hands, e.g. duct of submandibular gland and styloid process, body of masseter muscle with one finger intraorally and other extraorally corresponding the same location.



Bi-digital palpation: With the help of two fingers of one hand (thumb and index finger),





Bi-digital palpation of upper labial mucosa, sternocleidomastoid, buccal mucosa

e.g. sternocleidomastoid muscle, tongue musculature, nodules of lips and buccal mucosa and helix of ear. The edentulous alveolar ridge, unilateral or bilateral when a clinician encounters pathosis, he or she should investigate the contralateral region of the body to determine whether the condition is bilateral. As a rule, if similar masses are present bilaterally and in the same locations, they are most likely normal anatomic structures.

The carotid bulb in the bifurcation of the artery. The mastoid process, the lateral processes of the cervical vertebrae, and the wings of the hyoid bone are such bilaterally occurring anatomic structures that are frequently mistaken for pathologic masses. Bilateral palpation coupled with a knowledge of anatomy is obligatory if these normal structures are to be differentiated from pathologic masses.

71. What are the different ways of palpating muscles in orofacial region?

Flat muscle palpation: When muscle can be palpated over bone, e.g. masseter, temporalis.

Pincer palpation: When the belly of muscles can be held between two fingers, e.g. sternocleidomastoid and even for examination of trigger zones.

Functional Method

Inferior lateral pterygoid: Patient asked to open the mouth against resistance if it pains then it is the source, as the contraction of this muscle produces protrusion and/or opening of mouth. This muscle is stretched when the teeth are clenched so even while clenching pain will also suggest pain in inferior lateral pterygoid.

Superior lateral pterygoid: Contraction of this during clenching, produces pain if tongue blade is placed during maximal intercuspation still the pain increases, this is differentiating point from inferior lateral pterygyoid where pain decreases on keeping tongue blade. It also stretches during clenching, if the pain occurs during this, it is due to superior head of lateral pterygoid, at the same time if opening elicits no pain, then only superior pterygoids are affected and not elevator muscles and sideways turning of mandible against resistance.

Palpation of sternocleidomastoid muscle trapezius against resistance.

72. What are the indications of hot and cold in orofacial region?

Application of moist heat or cold can be helpful in chronic centrally mediated myalgia moist heat pack for 15–20 minutes while cold compresses for 5–7 minutes whichever will work, will be repeated.

Moist heat/ice can be used if the patient finds useful in disc dislocations with reductions. Myospasm of muscles of mastication can be treated by vapocoolant spray, ice.

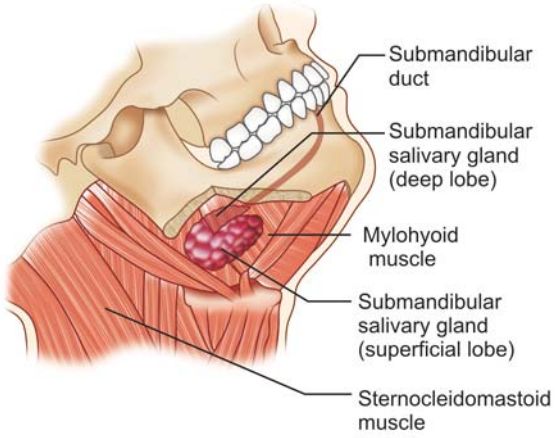
Moist heat can be used to reduce pain and inflammation after TMJ disclocation.

Coolant therapy is effective and simple in reducing pain and the cold does relaxation of muscles that are in spasm, and thus relieves the pain. Ice should be applied directly to the face on affected area and moved in a circular motion without pressure to tissues until patient feels numb and even moist heat (moist towel or hot bottle) should be applied to the muscle that increases circulation to the area by vasodilation.

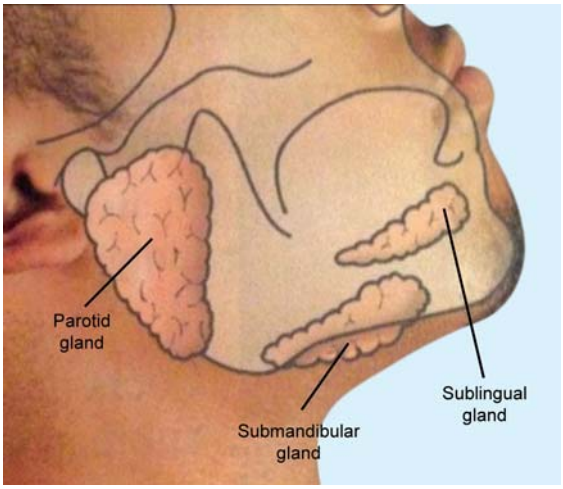
Moist heat during pain episodes in TMD's heat is contraindicated in infectious swelling as it may aggravate the swelling and probably will cause rupture.

73. What are superior and superficial locations?

Superior location is the structure which is superior in location related to any structure, while superficial location is the one when a structure is closer to skin surface. For example, the lingual nerve, submandibular duct and the hypoglossal nerve are superficial to the hyoglossus. The submandibular gland has large superficial lobe, the Stensen's duct extends superficial to parotid gland. The deep lobe of submandibular gland is superior to superficial lobe.



Deep lobe of submandibular node is superficial (close to skin) to mylohyoid and superior lobe



Sublingual gland is superior to submandibular gland and submandibular gland is superficial to sublingual gland

74. What is closed lock?

An intracapsular biomechanical disorder involving the condyle disc complex. In closed mouth position the disc is in an anterior position to the condylar head and the disc does not reduce with opening the mouth. Medial and lateral displacements of the disc may also be present. This disorder is associated with persistent limited mandibular opening that does not resolve with the clinician or the patient performing a specific manipulative maneuver. This is also referred to as "closed lock".

75. What are the types of intracapsular disorders of TMJ?

There are two types of intracapsular disorders of TMJ.

1. Derangements of condyle disc complex, (e.g. disc displacements with and without reduction, disc displacement with reduction, disc dislocation without reduction).
2. Structural incompatibility of articular surfaces.

76. What are TMJ subluxations and TMJ dislocations?

Subluxation of joint is the articular surfaces maintain a partial contact and condyle is able to return back to the glenoid fossa voluntarily.

Dislocation of TMJ occurs when condylar head travels anteriorly to articular eminence on maximal incisal opening and becomes locked in front of eminence such that the patient is unable to close the mouth.

These can be chronic/recurrent dislocations. These are the one which occurs on periodic basis. Prolonged/permanent dislocation. The dislocation which is remaining for prolonged time and patient ultimately get habituated to it.

77. State Laskin's criteria for MPDS.

There are four cardinal signs for MPDS. They are as follows:

Positive Findings

1. Unilateral pain in the pre-auricular region
2. Muscle tenderness in the pre-auricular region
3. Clicking or popping noises in the TMJ
4. Restriction and deviation of the jaw to the affected side during normal opening motion (Garg et al., MPDS an overview)

Negative Criteria

1. No radiographic evidence
2. No tenderness in TMJ via external auditory meatus.

78. What is jump sign?

In patient with myofascial pain dysfunction syndrome, when a myofascial trigger point is

detected and palpated the patient gives a typical behavioural reaction, acknowledging the tenderness felt in the area of pain reference, known as the 'jump sign' or it is involuntary withdrawl of stimulated muscle, when taut bands are palpated on trigger points. It is characteristic of fibromyalgia of cervical spine.

79. What is functional dislocation of disc?

The posterior border of disc becomes thin and the attachment of superior lateral pterygoid muscle pulls the disc forward through discal space. The disc is trapped in forward position. The translation of condyle is inhibited because anterior and medial position of disc. The individual feels joint being locked in a limited closed position, because the articular surfaces have actually been separated this condition is called functional dislocation of disc. There will be no joint sounds the point to help differentiate from functional dislocation.

80. What is loading of TMJ surfaces?

TMJ loading (compression) is done for the diagnosis of intracapsular disorder and myofascial pain. The operator is standing behind the patient with placing fore fingers under both sides of the mandible (it exerts bilateral upward pressure), at the same time thumbs are placed in the mentolabial area of chin with slight downward pressure. This maneuver forces condyle into superior and anterior position.

The pain in the TMJ region suggests intracapsular disorder (retrodiscitis/capsulitis)

While pain which radiates to other than TMJ structures in orofacial area suggests myofascial pain.

81. What is TMJ arthrocentesis? What are its indications?

It consists of TMJ lavage, placement of medications into joint, and examination under anesthesia. Intravenous sedation may or may not be used. It is indicated in painful limited mouth opening, the conditions that involve inflammation in the joint. The technique involves injection of two 18-gauge needle into superior joint space under local anesthesia. One needle 100–300 ml of Ringer's lactate solution is injected into superior joint space, the second needle acts as an outflow portal, which allows lavage of joint cavity. Steroids or sodium hyaluronate may be injected at the end of lavage to alleviate intracapsular inflammation. Soft diet and analgesics may be prescribed.

82. Describe various uses of botox in oral facial disease.

Uses of botox in oral medicine:

1. **In diagnosis:** Diagnostic applications of botox—it can be used to verify the correct diagnosis, e.g. to differentiate between pulpal and muscular pain. The muscular pain will be relieved.
2. **In TMJ disease:**
   - MPDS—botulinum toxin has been advocated for the treatment of MPDS.
   - Recurrent subluxations and dislocation—treatment of recurrent subluxations and dislocations of temporomandibular joint.
   - Temporomandibular disorders—TMJ disorders in the muscles which inflict soreness and discomfort.
3. **In parafunctional habits:**
   - Treatment of bruxism by bilateral intra masseteric injection.
   - Snoring and sleep apnea—injection of botox into masticatory muscles will enable the patients jaw more slightly forward during sleep, this opens the airway sufficiently to reduce snoring.
4. **Cosmetics purpose:**
   - Used in anti-ageing treatment to reduce wrinkling of facial skin.
   - It weakens masseter muscle giving less angle at the angle of mandible.
   - Prominent gums—display of excessive gingival tissue in maxilla upon smiling (gummy smile). Oral hygiene and esthetic issue.
   - Asymmetrical smiles—these result due to facial assymetries (acquired/familial) injection of botox into overactive muscles

produce gentle relaxation of muscles resulting in a symmetrical smile.

5. **Muscle relaxation and dysfunction:**
   - Oromandibular dystonia—injection of botox in masseter, medial/lateral pterygoid has been reported to resolve muscle dysfunction.
   - Hemifacial spasm and mandibular spasm—involuntary, irregular or chronic contractions of muscles innervated by facial nerve on ipsilateral side of face.
   - Blepharospasm—botox is injected superficially to the orbicularis oculi in four locations in periphery of each eye.
   - Palatal and stapedius myoclonus—tinnitus is caused, relieved by botox injection in soft palate and local application respectively.
6. **In neurological disorders and pain:**
   - Treatment of oral incontinence in facial paralysis
   - Facial nerve paresis
   - In trigeminal neuralgia and injected in trigger zones
   - Post-herpetic neuralgia
   - In refractory cases of migraine and headache
   - Neck pain and torticollis
7. **In salivary gland diseases:**
   - Salivary fistula—injection of botox in proximity of parotid gland results in the blockage of parotid secretions which is followed by glandular atrophy allowing salivary fistula to eventually heal.
   - Ranula—resolution of ranula by injection of botox.
   - Sialorrhea and drooling—intraglandular botulinum toxin injections can be used to improve sialorrhea in patients with Parkinson's disease, parkinsonian syndromes, motor neuron disease, and cerebral palsy.
   - Frey syndrome
8. **Miscellaneous**
   - Dental implants and surgery
   - Rhinitis

83. What are the limitations, complications and contraindications of botox?

There are a number of complications associated with botox therapy.

Limitations: The therapeutic levels inhibit masticatory functions temporarily and masticatory force will eventually return to previous levels once effect of drug has subsided.

Complications: Mild pain with injection, local edema, erythema, hematoma, transient numbness, weakness of muscle at sit of injection, post-injection bruising, local spread causing unwanted paralysis of nearby muscles.

- Mild nausea, transient headache, production of neutralising (IgG) antibodies against botox, facial nerve palsy, dysphagia, flu-like symptoms, development of tolerance and non-targeted muscle weakness.
- The complications are transient and generally resolves within some days. Overdose can be treated by antitoxin.

Contraindications: It is contraindicated in myasthenia gravis, Eaton-Lambert syndrome, motor neuron disease, concurrent uses of aminoglycosides and sensitivity to toxin and pregnant women and nursing women.

84. What are complementary and alternative medicine in persistent orofacial pain?

Complementary and alternative medicine (CAM) are group of unconventional medical systems, practices and products not presently considered part of the conventional biomedical care provided by medical doctors and other conventionally trained health professionals. These measures are:

1. **Mind–body interventions:** These increase the mind's capacity to enhance bodily functions and reduce symptoms, e.g. biofeedback, relaxation, meditation, hypnosis, yoga and spiritual approaches (prayers).
2. **Manipulative and body-based therapies:** These are physical modalities such as massage therapy, chiropractic adjustments and osteopathic manipulations.

3. **Biologically based therapies:** Foods, vitamins, minerals, herbal products, etc.
4. **Energy therapies:** (1) The practitioners intend to manipulate biofields theorized to exist within and around the patient. (2) Use of unconventional use of electromagnetic fields for therapeutic purposes.
5. **Alternative medical systems:** Homeopathy and naturopathy.

85. What is functional diagnosis? Give example of it.

A functional diagnosis is one that is more comprehensive and centred around biomechanics and disease, relating the two into a more conceptual definition. To diagnose the organ must be in function, e.g. diagnosis of disc displacements, TMJ subluxation.

86. Enumerate the muscles of mastication.

The muscles of mastication are the paired masseter, paired medial and paired lateral pterygoid, and paired temporalis muscles. The paired buccinators are considered as accessory muscles of mastication.

87. Enumerate the uses of steroids in non-mucosal agents.

1. More recently for central giant cell lesion the intralesional corticosteroid use has shown some value, inducing complete involution in many cases and partial involution in others. The suggested treatment is triamcinolone, 10 mg/ml, of which 1 ml is injected for each 1 cm of jaw involvement throughout the lesion, once a week for 6 weeks. Each injection sequence is performed with local anesthesia (bupivacaine) added to the injection solution.
2. Intra-articular injections:
   a. In the superior compartment of temporomandibular joint involving the disorders like degenerative disease and rheumatoid arthritis. Dexamethasone commonly used and more frequently in acute phases.
   b. These can be used in relieving pain and restricted movements.
   c. These can be used to prevent fibrosis after reduction of TMJ dislocations.
   d. Chemical arthroplasty in arthrosis of TMJ (hydrocortisone/triamcilone).
3. Systemic use of corticosteroids is not indicated for TMDs the exception is acute generalized muscle and joint inflammation with polyarthrides.
4. Severe myositis and TMJ dysfunction.
5. Dental surgeons are often advised to use corticosteroids during and after third molar removal and other dentoalveolar surgery to reduce post-surgical edema. The most commonly used forms of corticosteroids in dentoalveolar surgery include dexamethasone (oral), dexamethasone sodium phosphate and dexamethasone acetate, and methylprednisolone acetate and methyl prednisolone sodium succinate. Methylprednisolone is usually administered via the intramuscular or intravenous route though the possibility of topical (intra-alveolar) application has been described, with a reduction in morbidity and possible side-effects.
6. Topical and intralesional steroid injections in tonsillar fossa in Eagle's syndrome
7. Intracanal corticosteroid in root canal therapy.
8. Anaphylaxis.
9. Temporal arteritis.
10. Post-herpetic neuralgia
11. Ramsay Hunt syndrome
12. Bell's palsy.

Chapter

3

Benign Lesions of Oral Cavity

1. What is tumor? What is benign tumor? What are the differentiating points of benign and malignant tumors?

It is an abnormal mass of tissues the growth of which exceeds and is uncoordinated with that of normal tissues, and persists in the same excessive manner even after the cessation of stimuli which evoked the change.

The term benign implies mild and non-progressive. It is one which lacks the malignant properties. Thus, benign tumor by definition does not grow in an unlimited aggressive manner, does not invade the surrounding tissues and does not metastasize.

2. What are hamartoma, choriostoma and teratoma? Give an example of each.

1. **Hamartomas** are non-neoplastic over-growth of mature/differentiated tissues indigenous to the specific part of the body in which they develop.

Features	*Benign*	*Malignant*
Periphery	Smooth, well-defined, encapsulated	Ill-defined, absence of encapsulation
Cortication	Often present	Lack of cortication
Surrounding tissue	Compressed	Invaded
Size	Usually small	Often large
Growth rate and mode	Slow and with expansion	Rapid and infiltrative
Progression	Slow progressive, remain stationary and may regress and rarely fatal if untreated.	Progressive and always fatal if untreated.
Spread	Direct extension	Metastasis
Location	Specific anatomic site	Anywhere
Internal structure	RO, RL, mixed	RL
Effect on surrounding structure	Resorb teeth root, displace the teeth in bodily fashion without causing mobility	Destroy supporting alveolar bone so that the teeth may be floating.

A hamartoma is also defined as a disorganized proliferation of mature tissues, composed of elements that are normally found in the specific location in which it develops, often with one predominating element.

Hamartoma represents a dysmorphic proliferation of tissue that is native to the area and does not have the capacity for continuous growth but merely parallel that of the host. The distinction between a hamartoma and a benign neoplasm is often arbitrary; in fact, most benign tumors of infancy and childhood are actually developmental hamartomas. The salient features of hamartomas are that they cease growing at some points in their course and they do not infiltrate into surrounding tissues. Therefore, in bone they may be treated for cure by enucleation procedures and in soft tissue by local pericapsular excisions. Hamartomas are limited proliferations of tissue with no known stimulus and no truly invasive. For example, hemangioma, lymphangioma, odontome, pigmented nevi, etc.

2. **Choristoma** that is defined as a histologically normal tissue proliferation or nodule of tissue, of a type not normally found in the anatomic site.

   It is also known as a tumor-like mass consisting of normal cells in an abnormal location.

   A choristoma is defined as a histologically normal tissue proliferation or nodule of soft or hard tissue type not normally found in the anatomic site of proliferation.

   Choristoma tends to resemble tumors, as they present a relatively well-organized proliferation. Choristomas are similar to hamartomas except that they are dysmorphic proliferations of tissue that are not native to the site. These are one of rarities and has limited proliferation.

   For example, the rare heterotopic gastrointestinal cyst, which may be found in the tongue or floor of the mouth of infants and contains gastrointestinal glandular structures.

   The rare finding of bone or cartilage in the tongue, and the occasional development of thyroid tissue in the posterior tongue.

   The more commonly seen ectopic sebaceous glands known as Fordyce granules and salivary gland tissue within lymph nodes may also be considered choristomas. Each has a limited proliferation.

   Salivary gland choristoma central and gingival, both have ectopic salivary gland tissue appearing as a raised tumor-like mass, must not have any connection with normal minor or major salivary glands. Cartilaginous and/or osseous choristomas of oral soft tissue are rare lesions occurring most frequently on the tongue.

   Cartilaginous choristoma: It is composed of mature hyaline cartilage in fibrous tissue that resembles perichondrium; usually multilobulated; chondrocytes vary from small to large, but lack atypia osseous choristoma: composed of dense mature bone; osteocytes are compact and unremarkable; no prominent osteoblastic rimming; occasionally bone and cartilage are present in the same lesion, lingual thyroid choristoma, lingual sebaceous choristoma, glial choristoma gastric/respiratory mucosal choristoma (solid, cystic).

3. **Teratoma** is a congenital growth which contains tissue or organ components from all 3 embryologic germ layers or Weaver et al defines teratoma as a tumor consisting of multiple tissues that are not indigenous to their site of origin.

   In greek teratoma is monsterous tumor. It is a tumor composed of multiple tissues foreign to the normal organ from which it arises. Teratomas are true neoplasms originating from pluripotent cells and are composed of tissues from all three germinal layers, usually benign in nature. It is rare to find in head and neck regions.

   Epignathus is commonly used to describe a congenital teratoma in the oropharyngeal

region. Teratomas of the oral cavity are divided anatomically depending on their location. They can be sublingual, geniohyoid and lateral. Teratoma of tongue may exhibit skin, hair, bone, cartilage or mucous membrane on the surface. The dermoid cyst is sometimes considered a variant of teratoma containing ectodermal and mesenchymal derivative.

Teratomas, which are often thought of as hamartomas or choristomas, are actually true neoplasms with the capacity of continual growth. They are neoplasias that arise from multiple germ layers and thus produce tissues that are foreign to the part in which they develop. They are distinct from other neoplasias that may also show tissue diversity, such as pleomorphic adenomas of salivary glands, which are derived from native tissue. The majority of teratomas occur within the ovary (usually benign), or in the testes (predominantly malignant). Teratomas are also seen in the neck, the jaws, and the floor of the mouth on rare occasions.

One tumor of particular interest is the benign cystic teratoma of the ovary. Because of its origin from three germ layers, it will form various components of skin (including hair and sebaceous glands), teeth, and bone. The degree of differentiation and normal arrangement can be quite remarkable including bone with erupted teeth and even a fibrous periodontal ligament.

3. Enumerate the tumors that can cause large and disfiguring size.

Ossifying fibroma is one of a few allowed by patients to reach the largest and most disfiguring size in all the benign tumors of the head and neck areas. The others are ameloblastomas, odontogenic myxomas, neurofibromas, and pleomorphic adenomas. The reason is probably because of its persistently steady but slow rate of growth and its painless character.

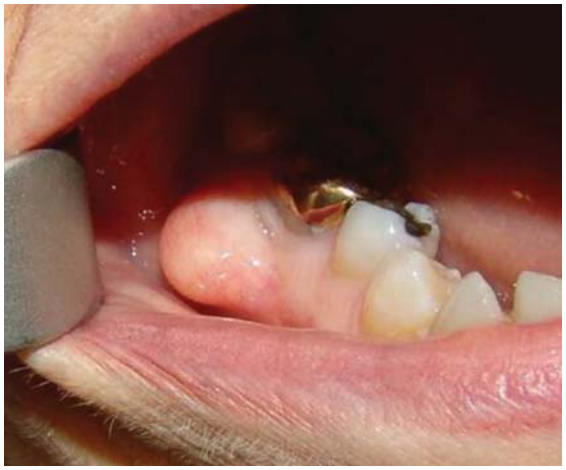
4. Give differentiating points of fibrous dysplasia and ossifying fibroma.

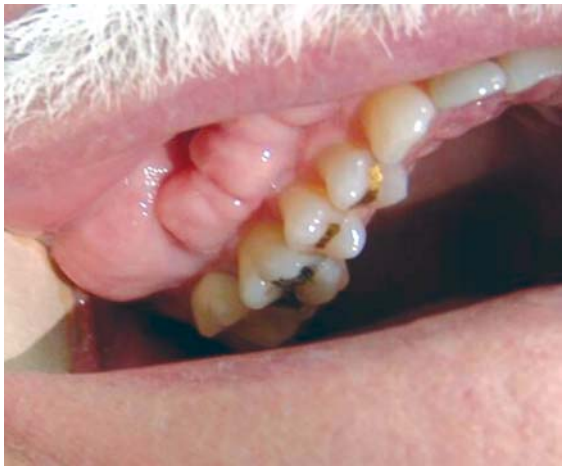
	Fibrous dysplasia	*Ossifying fibroma*
Age	Ist and IInd decades	IIIrd and IVth decades
Sex predilection	Monostotic equal predilection Polyostotic female predilection	More in women
Common jaw site	Maxilla	Mandible
Types	Monostotic/polyostotic	Juvenile/psammomatoid form
Radiographically	Opacity diffuse border blending	Opacity circumscribed borders well-defined
Growth pattern	Self-limited stabilizes at puberty	Continuous not hormone related
No. of bones involved	One (monostotic) or more (polyostotic)	Only one
Syndrome associated	McCune-Albright syndrome	None
Malignant potential	Less than 1% usually with polyostotic	Nil
Histologically	Vascular matrix woven bone trabeculae	Cellular fibrous matrix bony islands and trabeculae
Treatment	Recontour for cosmetics	Excise

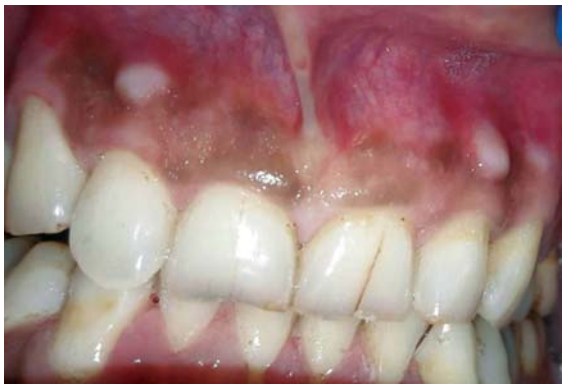
5. What are exostoses?

These are single or multiple localised bony excrescences that arise from the cortical plates, asymptomatic along the buccal aspects of alveolar bone most often in posterior region. These are benign growths may be related to stress placed on bone from function of teeth. They are less common than tori. They are

discovered most often in adults. Exostosis are seen less commonly in the lower jaw. Buccal exostoses occur as a bilateral bony hard nodules along the facial aspect of maxillary and mandibular alveolar ridge.







6. What are tori? What are the varieties of palatal torus and torus mandibularis?

Localised nodular enlargements (exostoses) of the cortical bone at midline of palate (torus palatinus) and lingual aspects of mandible (torus mandibularis).

Classification of Tori

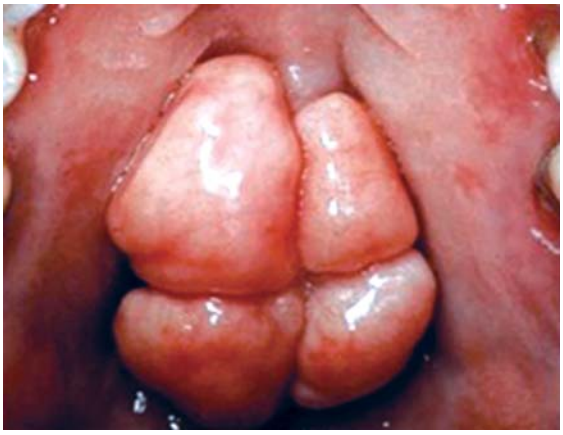
Class 1: Tori are absent or minimal in size and do not interfere with existing denture.

Class 2: Clinically several tori of moderate size, often mild difficulties in denture construction and use of surgery is not required.

Class 3: Tori are of large size, they compromise function of dentures and require surgical excision.

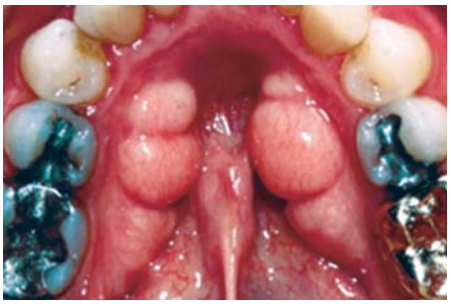
Palatal torus can be classified according to their morphological appearance into:

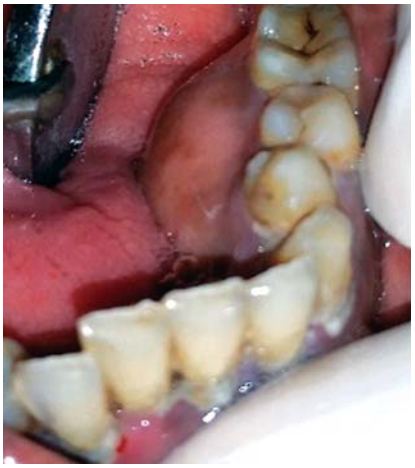
a. **Flat torus:** Broad base, slightly convex smooth surface. The extension is symmetrically on both sides of midpalatine raphe.
b. **Spindle torus:** A midline ridge is present along the palatal raphe and sometimes median groove is present.
c. **Nodular torus:** It is seen as multiple protuberances each with an individual base. The protuberances may coalesce forming grooves between them.
d. **Lobular torus:** It is a multilobulated mass and arises from single base. They may be sessile or pedunculated.

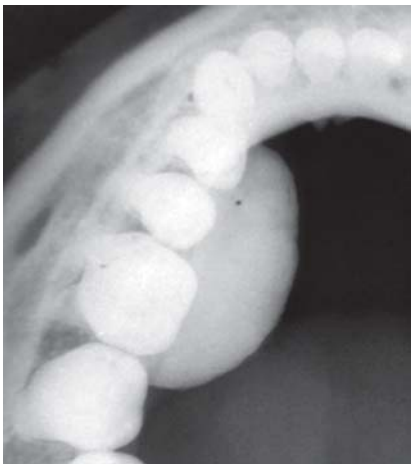


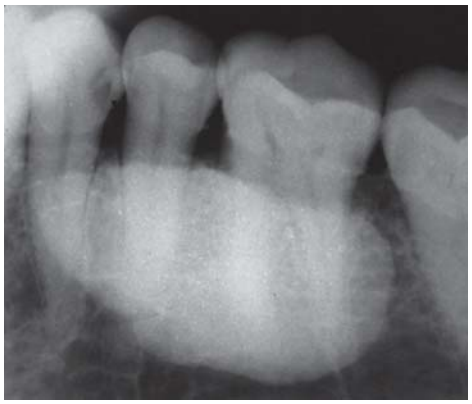
Mandibular torus presents as bony protuberance along the lingual aspect of mandible above the mylohyoid line most commonly in the region of premolars. They may present as

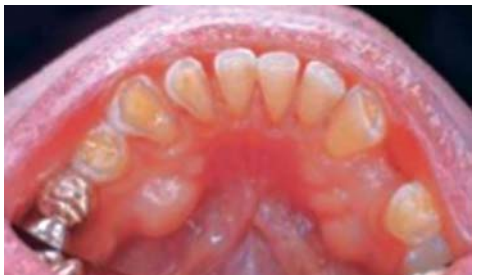
a. Single nodules
b. Multiple lobules paralleling the teeth
c. Rarely do they become large enough to meet in midline.

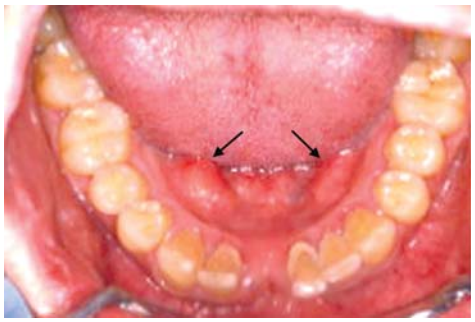










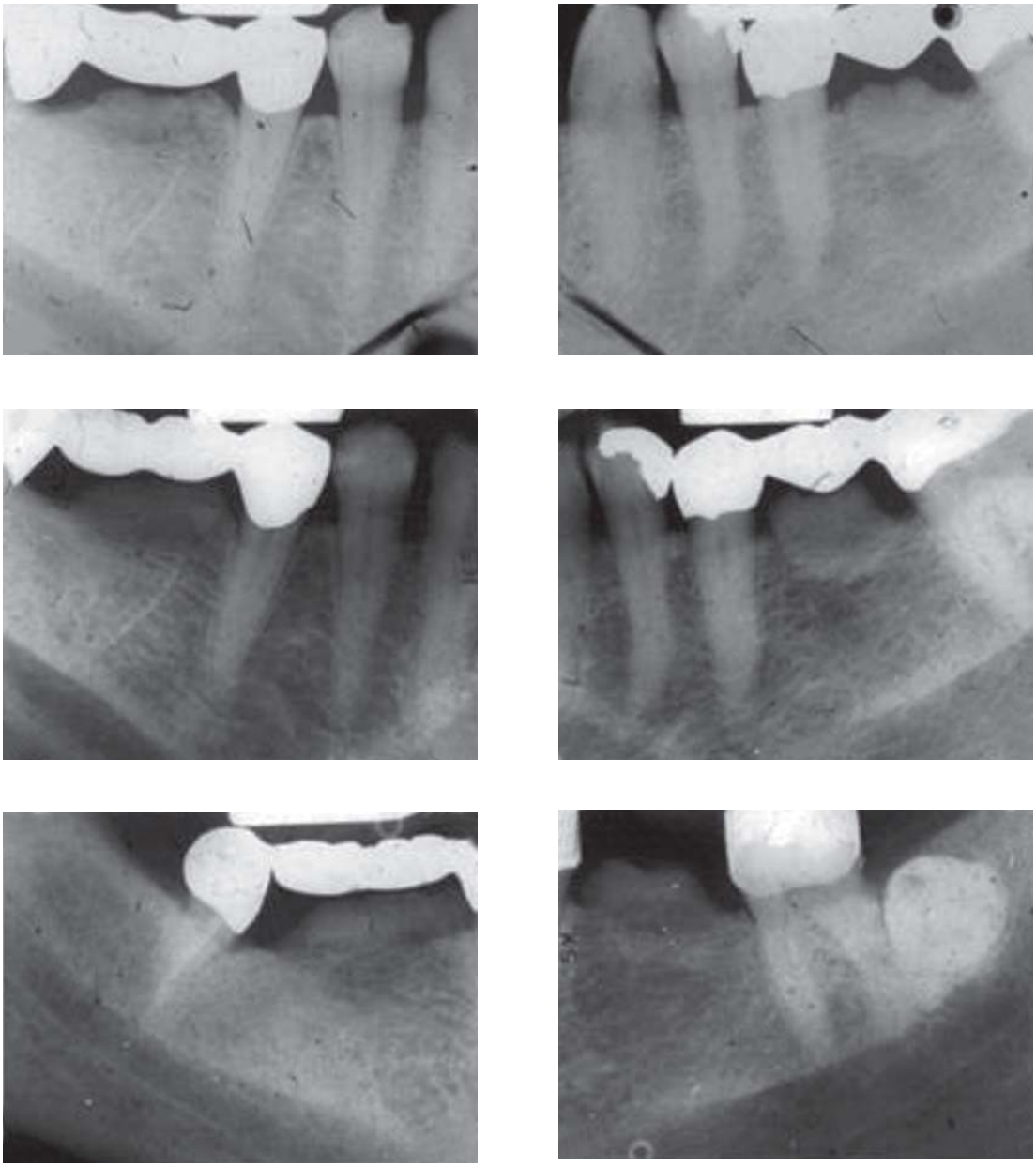


7. What are the differences among buccal exostosis, torus palatinus, and torus mandibularis?

These entities are all very site specific. The palatal torus is seen only in the midline of the hard palate, whereas the torus mandibularis occurs only on the lingual surface of the mandible. Buccal exostosis is found only on the facial surface of the alveolar bone, most commonly the maxillary alveolar process.

8. What is reactive subpontine exostosis?

It is also called subpontic osseous proliferation or subpontic osseous hyperplasia. It is an uncommon but interesting variant. This



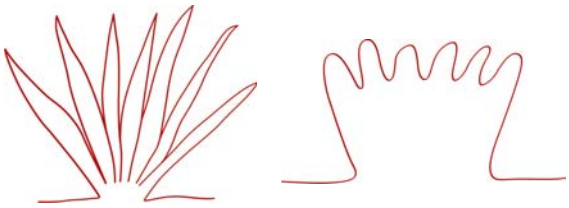
develops from alveolar crestal bone beneath the pontic of posterior bridge. It may need to excise for oral hygiene purpose.

9. What is exophytic growth? What are the mechanisms by which exophytic lesions are produced?

Any pathologic growth that projects above the normal contours of the oral surface is called exophytic growth, hypertrophy, hyperplasia, neoplasia, and the pooling of fluid are four mechanisms by which exophytic lesions may be produced.

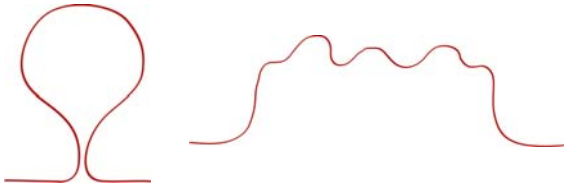
10. What are different shapes of exophytic lesions?

Papillomatous, verrucous, nodular, dome shaped, polypoid, and bosselated.



1. Papillomatous, e.g. squamous papilloma,
2. Verrucous, e.g verrucous ca., verruca vulgaris proliferative verrucous leukoplakia

3. Nodular, e.g. exostosis, tori, fibroma
4. Dome shaped, e.g. ranula, buccal space infections



5. Polypoid, e.g. pulp polyp, gingival polyp
6. Bosselated, e.g. torus palatinus.

Papillomatous or verrucous originate in surface epithelium, e.g. verruca vulgaris, papillomas, squamous cell carcinoma, verrucous carcinoma and keratoacanthoma. Those with smoothly contoured shaped originate in the deeper tissues and are beneath and separate from stratified squamous epithelium. For example, tori, fibroma, lipoma and malignant mesenchymal tumor.

11. What are oral papillary lesions?

Papillary lesions represent swellings with finger like projections imparting cauliflower appearance; these projections are rounded and blunt like fungiform papillae of tongue. Oral papillary lesions (OPL) are growths of oral mucosa which are verrucous (white with rough surface) and papillary (multiple finger like projections) in nature. The entities which are included in this are squamous papilloma, verruca vulgaris and condyloma accuminatum and heck disease. Clinically, OPLs can appear as a sessile/pedunculated, white (heavily keratotic) or corol pink growths. Generally, solitary (except condyloma accuminatum seen in clusters) and asymptomatic. They are small (less than 1 cm) can occur intraorally and on vermillion border. OPLs are mildly contagious. Treatment is surgical excision. Interferon-alpha intralesionally may be useful specially in HIV patients. They have favourable prognosis except for laryngeal papillomatosis.

Papilloma and giant cell fibroma are more common papillary lesions. Diffuse papillary lesions represent oral markers of systemic disorders.

Squamous papilloma: Most common benign oral epithelial neoplasm. The common sites are ventral tongue, frenal area, palate and mucosal surface of lips. These may occur spontaneously.

Verruca vulgaris: It is common skin wart appear on vermillion border and less commonly intraorally. The intraoral locations are palate and keratinized gingival tissues. It is associated with HPV (2, 4, 40).

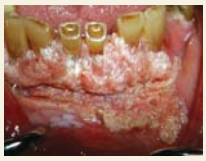
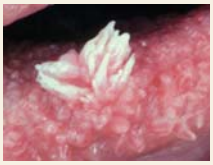
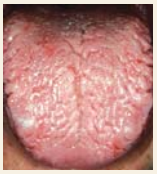
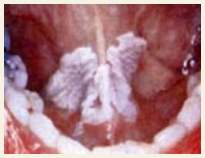
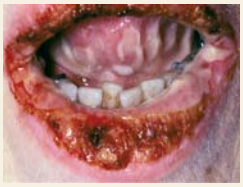
Condyloma accuminatum: It characteristically situated in anogenital area and presence in mouth indicated sexual transmission. Intraoral common sites are lips—commisures and gingival mucosa. It is associated with HPV (6 and 11) and frequently seen in HIV positive patients.

12. What is the difference between intraoral growth and swelling?

The growth projects above the surface of normal contour, while swelling is present in between two surfaces. The growth is excised while swelling is aspirated before it goes for surgical excision, e.g. irritational fibroma may produce growth on buccal mucosa while minor salivary gland tumor or mucocele may produce swelling on the same location.

13. Describe various surface textures of oral soft tissue lesions.

Various surface textures of oral soft tissues are:

Types	*Example*	*Diagram*	*Clinical photograph*
Verrucous	Verrucous leukoplakia		
Papillomatous	Squamous papilloma		
Fissured	Fissured tongue		
Corrugated	Hyperkeratosis		
Crusted	Paraneoplastic syndrome		

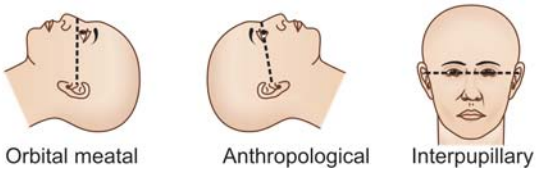
14. Enumerate oral psoriasiform lesions.

Psoriasiform mucositis (microscopically engorgement of capillaries in the connective tissue and mixed inflammatory infiltrate). Reports of oral psoriasis that are well-documented show no consistent lesion pattern. Patterns range from raised, white, scaling lesions predominantly on the palate or buccal mucosa to well-demarcated, flattened, erythematous lesions with a slightly raised, white, annular or serpiginous border. Benign migratory glossitis, reactive arthritis (previously called Reiter's syndrome), erythema migraines.

15. Which are the anatomical baselines used in cephalometric and extraoral radiography?

The anatomical baselines which are used in cephalometrics and extraoral radiography are:

1. Anthropological
2. Orbital meatal
3. Interpupillary



16. Enlist what all types of fibromas can be seen in oral cavity? Name the syndromes associated with fibroma.

The true fibroma, irritational fibroma, giant cell fibroma, desmoplastic fibroma, central cementifying/ossifying fibroma and central odontogenic fibroma. The true fibroma (fibroma) is a connective tissue tumor which is rare to find. Irritational fibroma (traumatic fibroma) is common benign exophytic oral lesion that develops secondary to tissue injury, trauma, and local irritation. Giant cell fibroma is a benign tumor of oral mucosa with distinctive clinicopathological features. The name is given as it shows large multinucleated fibroblasts that tend to occur in close proximity to overlying epithelium and shows nodular appearance clinically.

Desmoplastic fibroma is rare. It shows fibroblast and collagen fibres seen in mandible commonly and more than 30 years of age. It shows painless enlargement of jaws aggressive lesion.

Central ossifying/cementifying fibroma has cellular fibrous stroma and calcification.

It is seen in adults in 3rd/4th decades. Commonly in female and in mandible. Radiographically shows lucent/opaque appearance.

Central odontogenic fibroma is uncommon, slow growing variety. It is non-aggressive, well-defined lesion. Radiographically showing unilocular, root resorption.

Syndromes Associated with it

1. **Cowden's syndrome (multiple hamartoma and neoplasia syndrome):** Multiple fibromas, multiple papules on lips and gingivae producing cobblestone appearance.
2. **Tuberous sclerosis:** Seizures, mental retardation with hamartomatous glial proliferation, wart-like lesion on cheek in butterfly-like distributon.

17. Enumerate the lesions which are associated with sun exposure.

Actinic chelitis, basal cell carcinoma, solar keratosis (squamous epithelium may undergo hyperplasia or atrophy), actinic keratosis (erosion and white brown crusting on vermillion border of lip), freckle. Actinic cheilosis is characterized by atrophy of the vermilion border, which may develop dry, scaly changes. As the condition progresses, ulcerated sites may appear which partially heal, only to recur at a later date. (The patient often mistakes these recurring ulcerated lesions for "fever blisters.") The evolving cancer slowly becomes a crusted, non-tender, indurated. Keratoacanthoma seen on upper lip on sun exposed lesions. Ephelides darken on sun exposure.

18. What are causes of gingival hyperplasias?

1. **Local:** Plaque, calculus, bacteria
2. **Systemic:**
   a. *Hormonal imbalance:* Estrogen, testosterone
   b. *Drugs:* Phenytoin (dilantin), cyclosporin, nifedipine and other calcium channel blockers
   c. Leukemia (leukemic infiltrates and/or local factors)
   d. Genetic factors
   e. Syndromes

19. Enumerate the syndromes associated with gingival fibromatosis.

1. **Murray-Puretic-Drescher syndrome:** Gingival fibromatosis with multiple hyaline fibromas of the skin and scalp
2. **Laband syndrome:** Gingival fibromatosis with formative defects of the ear, nose, nails, bone and hepatosplenomegaly.
3. **Rutherford syndrome:** Gingival fibromatosis and corneal opacities.
4. **Cross syndrome:** Gingival fibromatosis with microphthalmia, skin hypopigmentation, mental retardation, and corneal opacities.

In each of the above syndromes, the gingival fibromatosis is usually less severe than it is in hereditary gingival fibromatosis.

20. Enumerate the causes of swellings in floor of mouth.

1. **Inflammatory/trauma:** Mucous retention cyst (ranula)—elevated, fluctuant bluish white mass in lateral floor mouth. Most are due to sialoliths.
   Ploughing ranula: When the contents herniates through mylohyoid muscle and presents as swelling in submandibular or cervical areas.
2. **Developmental:** Lymphoepithelial cyst asymptomatic nodule less than 1 cm in diameter.

Epidermoid

Dermoid cyst: Asymptomatic mass usually in midline.

3. **Neoplasms:** Salivary gland tumor—solitary, asymptomatic, firm mass. Malignant tumors may cause pain, parasthesia.
   Mesenchymal neoplasms: Firm, asymptomatic rarely malignancies less likely to recur lipoma.
4. **Infectious:** Ludwig's angina.

21. Enumerate the causes of swellings of palate.

Differential diagnoses of palatal swelling

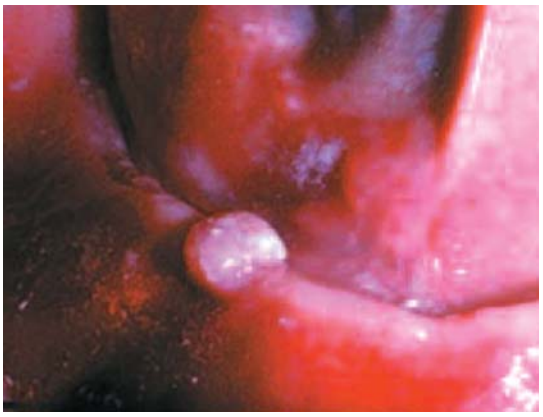
Onset and course	
Acute	Palatal abscess (painful and non-vital tooth)
Chronic	Torus palatinus, median palatal cyst, pleomorphic adenoma, hyperplastic candidiasis
Shape	
Globular	Palatal abscess, torus palatinus, median palatal cyst, pleomorphic adenoma
Peppled	Hyperplastic candidiasis
Consistency	
Fluctuant/tense	Palatal abscess, median palatal cyst
Rubbery/firm	Torus palatinus, pleomorphic adenoma, hyperplastic candidiasis, lymphoma
Associated pain	
Yes	Palatal abscess, hyperplastic candidiasis, infective or traumatic complications of: Torus palatinus, median palatal cyst, pleomorphic adenoma (malignant variety may cause parasthesia)
No	Uncomplicated: Torus palatinus, median palatal cyst, mucous extravasation phenomenon, (bluish) pleomorphic adenoma
Associated fever	
Yes	Palatal abscess (but may not be present)
No	Hyperplastic candidiasis, uncomplicated: Torus palatinus, median palatal cyst, pleomorphic adenoma
Patient attributes	
Poor oral hygiene/caries	Palatal abscess
Diabetes/HIV	Palatal abscess
Denture wearer	Hyperplastic candidias
Other regions/systems	Neoplasm of maxilla or maxillary sinus: Palatal swelling with or without ulceration tumor arising from CNS.

22. What is cutright lesion?

It is also called reactive osseous and chondromatous metaplasia. Occasionally

1. Cartilage or bone may be discovered within soft tissue specimens removed from the oral cavity.
2. Cartilaginous rests are known to exist in the area of the nasopalatine duct.
3. Cartilaginous metaplasia secondary to chronic denture trauma leads to formation of cartilage within flabby soft tissue removed from maxillary edentulous alveolar ridges of long-term denture wearers.

Clinically it shows an extremely tender and localized area of the alveolar ridge associated with local enlargement. These changes almost always arise in patients with extensive atrophy of the mandibular alveolar ridge, leading to a knife edge-like crest and sometimes in maxilla. Although most examples involve the posterior mandible, similar areas may rarely be seen overlying the maxillary alveolar ridge or associated with anterior portions of the mandible. It is also called cutright tumor. This represents hyperplasia of existing embroyonic cartilaginous rests due to inflammatory factors induced by ill-fitting dentures.



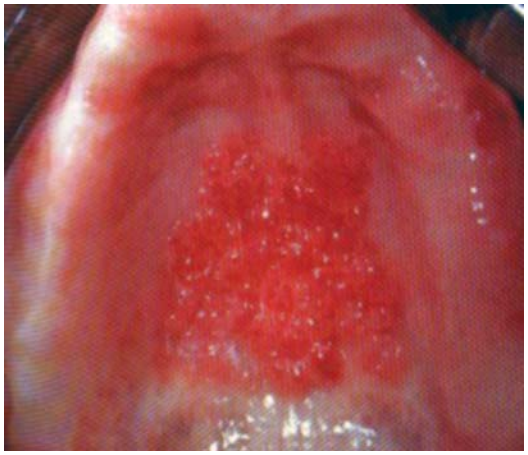
Tender, elevated nodule along the thin crest of mandibular alveolar ridge

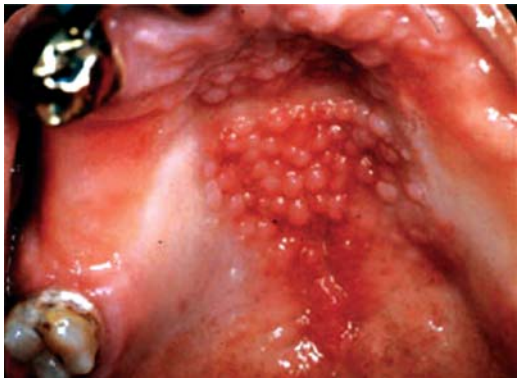
They differ from soft tissue choristoma in that they arise directly from the bone beneath the lightly bound mandibular alveolar ridge.

These are treated by recontouring the thin mandibular ridges and supplemented with graft material. Implants may also reduce the traumatic injury to the ridge and lessen the chance of recurrence. If the ridge modification is not made, the continued injury to the site occasionally results in recurrence of the lesion.

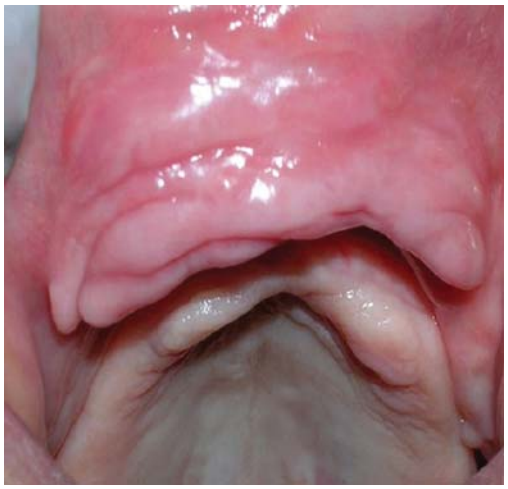
23. Name the oral mucosal lesions that are caused by acrylic dentures.

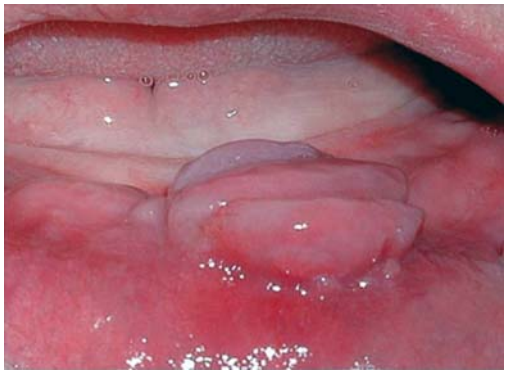
1. **The inflammatory hyperplasia (IH) lesion known as papillary hyperplasia:** The palatal hyperplasia (PHP and palatine papillomatosis) occurs almost exclusively on the palate beneath a complete or partial removable denture. It is more commonly associated with a flipper-type partial denture or a full denture. Approximately 10% of the people who wear maxillary dentures have this condition, and most wear their dentures continuously.



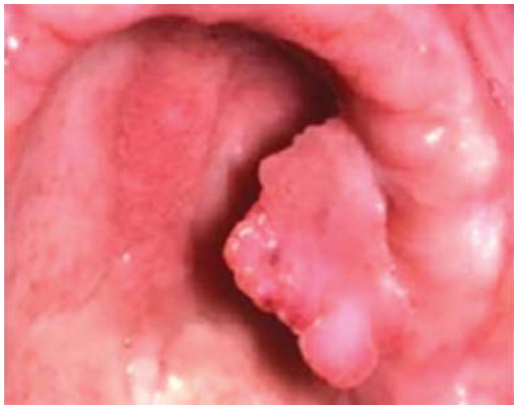


2. **Epulis fissuratum (denture induced fibrous hyperplasia, inflammatory fibrous hyperplasias, denture injury tumor, denture epulis)** is an IH lesion observed at the borders of ill-fitting dentures. In most instances the dental flanges overextend secondary to alveolar bone resorption and settling of the denture. The exophytic, often elongated lesion usually has at least one cleft into which the denture flange fits, with a proliferation of tissue on each side. Commonly seen in vestibular mucosa and less likely in lingual sulcus.

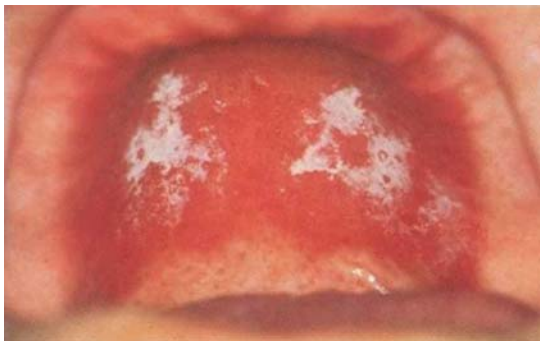




3. **Leaf like denture fibroma (fibroepithelial polyp):** This characteristic lesion is a flattened pink mass attached to palate by a narrow stalk, occurs on hard palate beneath maxillary denture. This is a flattened mass closely applied to palate and sits in a slightly cupped-out depression. The pedunculated nature can be easily demonstrated by lifting with probe. The edge of lesion is often serrated and resemble like a leaf.

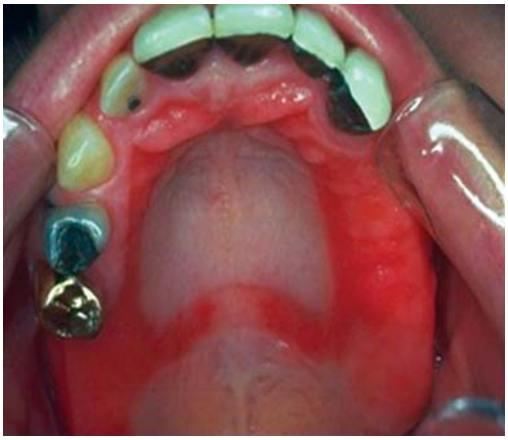


4. **Denture stomatitis:** The diffuse redness of the palate seen under dentures has posed a diagnostic problem for years. It is often classified as a form of erythematous candidiasis. Causes are apparently multifactoral; denture trauma, improper designing of denture causing unusal pressure, allergy to denture base, inadequate curing, denture plaque and candidal infection are the most important. Denture stomatitis occurs under either complete or partial dentures and is found more frequently in women. The lesions are usually confined to the palate and seldom if ever involve the mandibular ridge. In approximately 50% of the patients, there is an associated angular cheilitis with or without an inflammatory papillary hyperplasia of the palate.

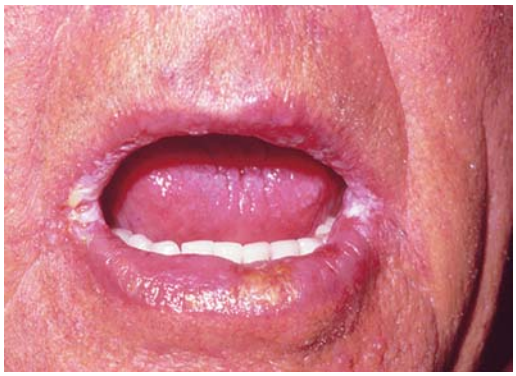


5. **Denture papillomatosis (inflammatory papillary hyperplasia):** Some investigators classify as a part of denture stomatitis. The causes are ill-fitting dentures, poor dental hygiene, wearing the dentures continuously and possibly candida. Clinically, occurs under hard palate beneath the denture rarely on mandibular mucosa. The mucosa is erythematous and has pebbly surface, most cases are associated with denture stomatitis.

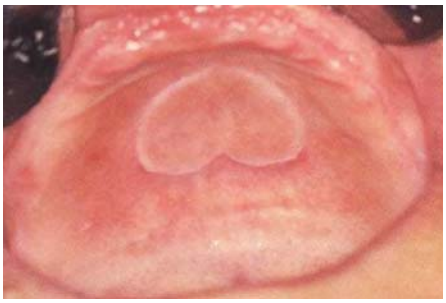
6. **Atrophic candidiasis:** The most common clinical presentation of atrophic candidiasis is the red, velvet-like, tender palatal mucosa under a denture. Here the high-frequency, low-intensity trauma of a denture surface compressing the palatal mucosa during swallowing alters the barrier defense, allowing the normally innocuous candida organisms to become pathogenic.

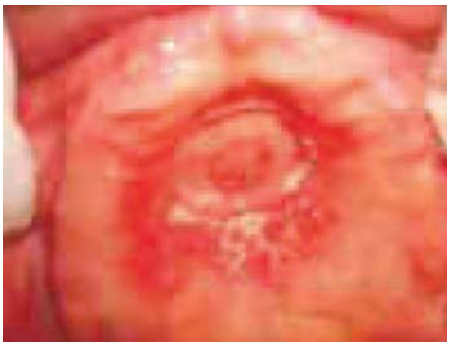


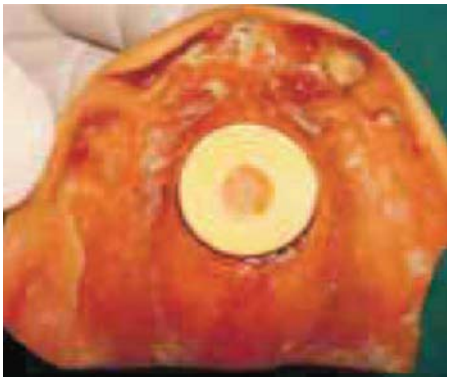
7. **Angular cheilitis:** Dentures with reduced vertical dimensions. It is also called perlèche, which often begins when a loss of occlusal vertical dimension creates a constant moisture and cracking at the commissure and predisposes the tissue to candida proliferation and invasion. In this form, the commissures are tender, fissured, and often crusted, and there may also be some skin erythema.

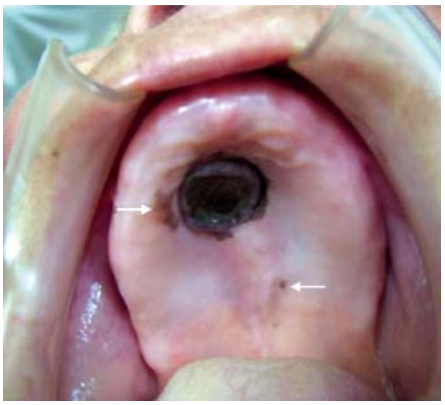


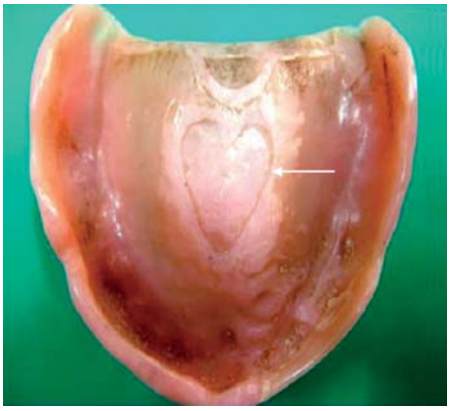
8. **Focal hyperkeratosis (frictional hyperkeratosis):** Most commonly seen in alveolar ridge associated with ill-fitting lower denture.
9. **Ulcers and erythema:** These are associated with denture flange.
10. **The lesions associated with suction disc:** On palate with upper denture and hyperplasia due to negative pressure a heart shaped or round area of mucosal heperplasia may appear on hard palate. This type of hyperplasia occurs if a relief chamber exists at centre of basal plate of denture.



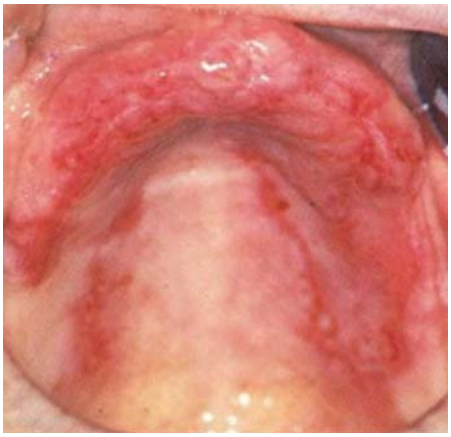


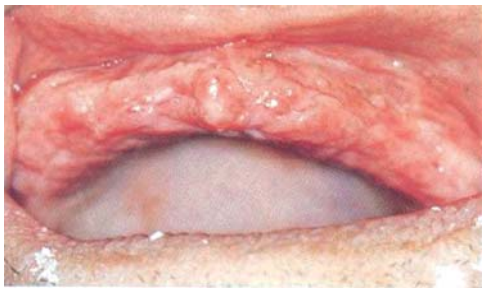






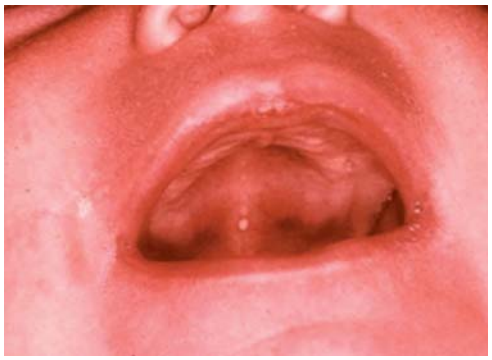
11. **Cutright tumor (reactive osseous and chondromatous metaplasia):** It is a cartilaginous choristoma with very mature and localized submucosal cartilage.
12. **Acrylic resin:** Burn autopolymerizing acrylic resins which are used for the construction of temporary prosthesis may cause local burns either due to heat evolving during polymerization or to monomer excess. The mucosa is red with or without erosions.
13. **Atrophy of maxillary alveolar ridge:** This may occur due to excessive occlusal trauma because of poor fitting denture common in women in anterior maxilla. The alveolus becomes flabby and red and may co-exist with epulis fissuratum.

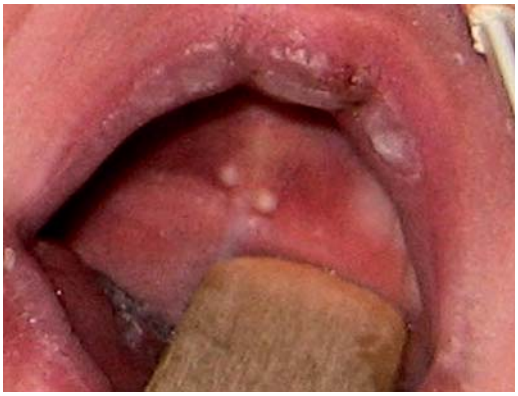


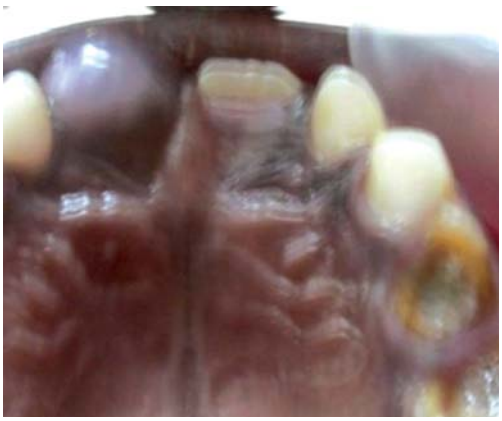


24. What are Epstein's pearls?

A similar palatal cyst of the newborn is commonly found in the posterior midline of the hard palate, where it arises from epithelial remnants remaining in the stroma after fusion of the palatal processes which meet medially to form the palate. As originally described, the cysts along the median raphe of the palate were called Epstein's pearls and the term Bohn's nodules was used for cysts which originated from palatal gland structures and were scattered more widely over the hard and soft palates. Today these two terms are used interchangeably for both palatal and gingival cysts of newborns.

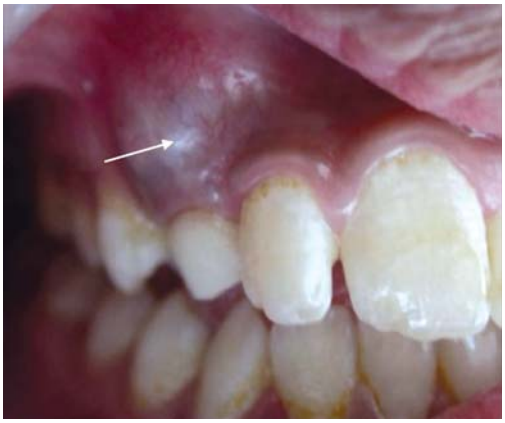


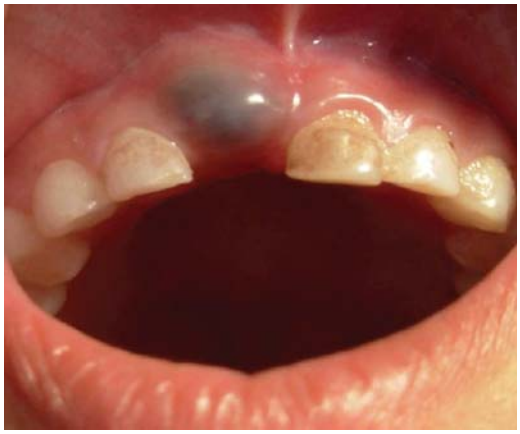


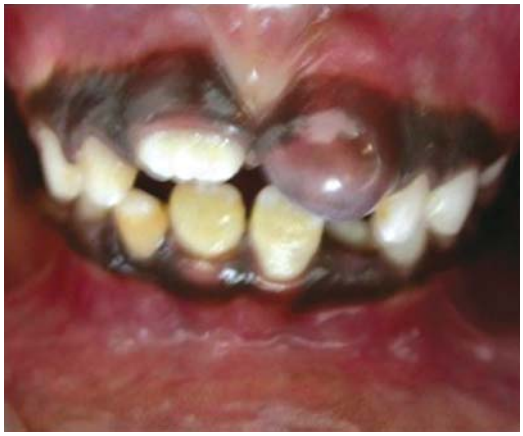


25. What is eruption hematoma?

It develops as a result of separation of dental follicle around the crown of erupting tooth, i.e. within the soft tissues overlying the alveolar bone. It is seen in crown of deciduous and permanent teeth (eruption cyst). The presentation is soft, transclucent swelling



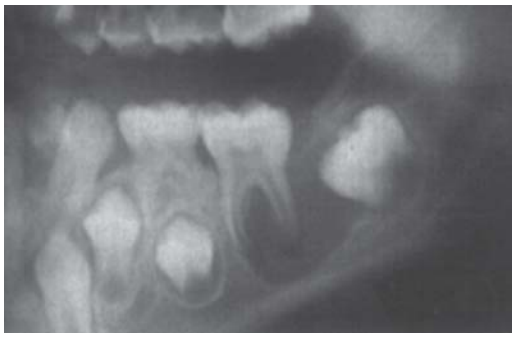


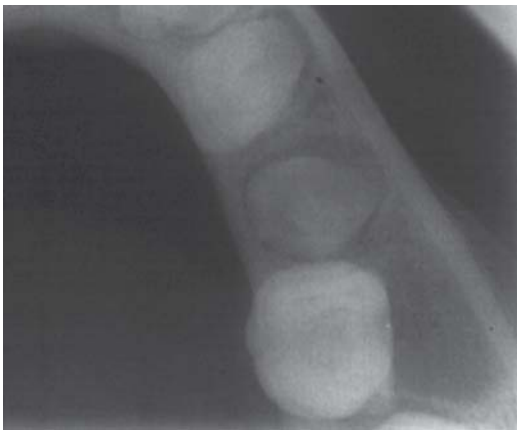


overlying gingival mucosa. Commonly seen in children younger than 10 years of age, with deciduous mandibular central incisors most commonly affected, then first permanent molars and then deciduous maxillary incisors. The surface trauma may result in considerable amount of blood which imparts a blue to purple brown color. These lesions are called eruption hematoma.

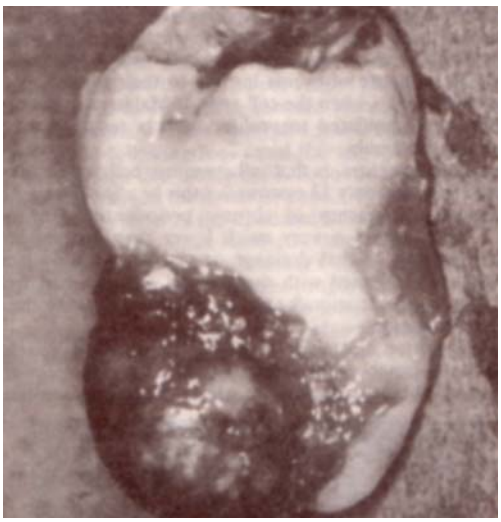
26. What are buccal bifurcation cyst and paradental cysts?

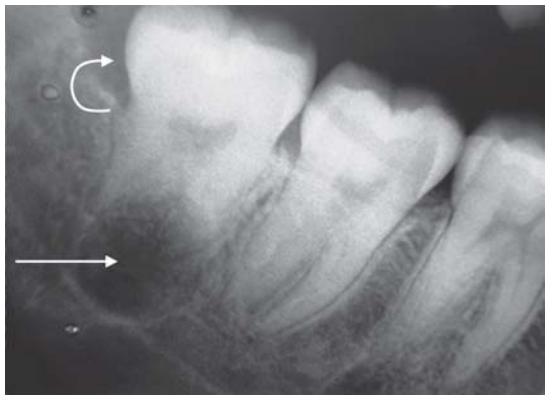
Buccal bifurcation cyst is an uncommon, inflammatory, odontogenic cyst that characteristically develops on buccal aspect of mandibular first permanent molar (commonly) and second permanent molars. Common in pediatric population, may be seen bilaterally. The affected tooth is vital. It has been associated with teeth that demonstrate buccal enamel extensions into bifurcation area.





The term paradental cyst sometimes has been used synonymously for buccal bifurcation cyst. These lesions occur distal or buccal to the partially erupted mandibular third molars with pericoronitis. It should be distinguished from inflamed dentigerous cyst.





Paradental cyst attached with mandibular third molar while radiograph showing radiolucency at bifurcation area by *straight arrow* and preservation of follicular space *curved arrow*

27. What are two-thirds tumor?

The adenomatoid odontogenic cyst (AOC) has been sometimes referred as "two-thirds tumor" because about two-thirds occur in the maxilla, two-thirds occur in young women (pre-teen and teenage years), two-thirds are associated with an unerupted tooth, and two-thirds of those teeth are canine teeth. The two-thirds statistics vary slightly, but the rough distribution is accurate.

It is a cystic hamartoma arising from odontogenic epithelium. It will characteristically have a lumen lined by epithelium from which proliferations fill much and sometimes all of the lumen space, then mimicking a solid tumor. This cyst will present as an expansile lesion usually in the anterior region of either jaw. The cyst's clinical emergency may be subtle and discovered only as an incidental clinical or radiographic finding, or it may be discovered by rapid clinical expansion causing alarm and pain. Some will reach in very large sizes (10 cm) and distort facial contours.

28. What are the radiographic appearances of calcifying odontogenic cysts?

These are discovered as an incidental radiographic finding. Early in their development, they will appear completely radiolucent. As they mature, they develop calcifications

that produce a well-circumscribed, mixed radiolucent-radiopaque appearance.

Three General Patterns of Radiopacity are Seen.

- One is a salt-and-pepper pattern of flecks,
- The second is a fluffy cloud-like pattern throughout, and the third is a crescent-shaped pattern on one side of the radiolucency in a "new moon" like configuration.
- The third is a crescentic-shaped pattern.

29. In which conditions residual cyst occur?

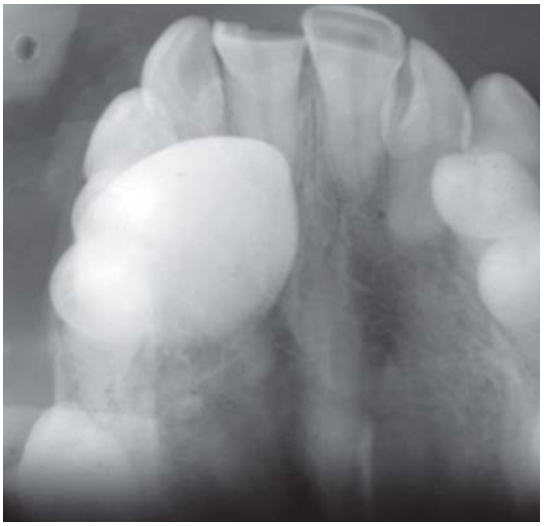
Residual cyst may occur after endodontic therapy which has failed either to eliminate inflammatory focus or had undiagnosed accessory canal. Less frequently it may occur with successful seal where residual infection in periapical area is remaining.

30. Enumerate the cysts occurring in the midline region of the oral cavity.

Glandular odontogenic cyst, globullomaxillary cyst, nasolabial cyst, median mandibular cyst, nasopalatine cyst.

31. What is cystography?

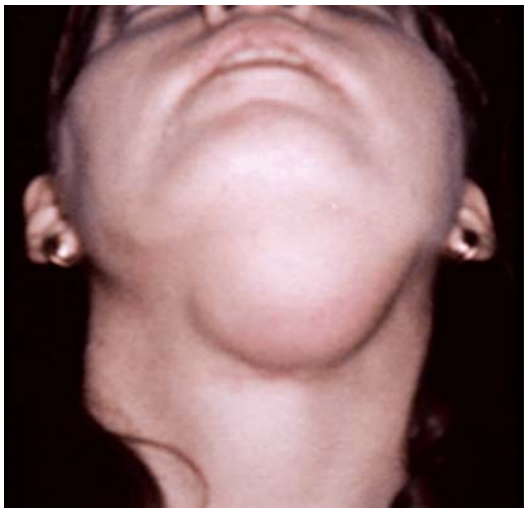
Cystography is a procedure of injecting a radiopaque dye into the cystic cavity to visualize the boundaries of lesion. The injection of radiopaque contrast medium ('conray 420').

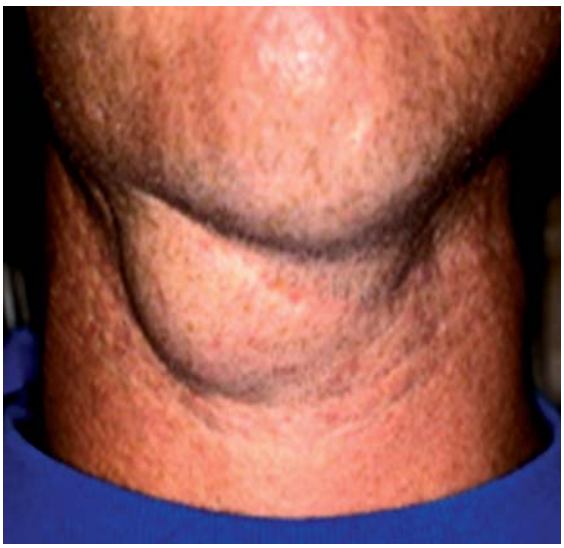


Maxillary cross-sectional occlusal radiograph with contrast reveals well-defined ovoid radiopacity 2 cm in greatest diameter superimposed on the crowns of 1514 and roots of 13–11.

32. What is cervical ranula?

A plunging ranula, also known as a cervical or diving ranula, is a rare clinical entity. The name ranula derives from its similar appearance to the air sacs in a frog's neck where the Latin name **rana** means *frog*. A ranula is an extravasated mucocele derived from the sublingual gland which lacks a proper capsule. Rupture of the main ducts or acini of the sublingual gland occurs due to obstruction which can lead to the formation of a ranula. The extravasated mucus triggers a localized inflammatory response and becomes enveloped in fibrous granulation tissue. In general, ranulas continue to enlarge because the sublingual gland is a constitutive secretor of mucus. Ranulas can be limited to the intraoral region or they can expand and herniate through or around the mylohyoid muscle which serves as an anatomical barrier between the sublingual and submandibular regions. When a ranula extends through or around the mylohyoid muscle, it is termed a plunging ranula and often presents with swelling in the submandibular or cervical areas.





33. What are pseudocysts? Enumerate the pseudocysts occurring in oral and paraoral structures.

Pseudocysts appear radiographically as cyst like lesions but microscopically exhibit no epithelial lining.

Aneurysmal bone cyst (more common in mandible): Traumatic (simple) bone cyst intrabony cavity related to trauma. Static bone cyst (Stafne's bone defect) developmental entity mainly due to entrapment of salivary gland tissue.

Focal osteoporotic bone marrow defect (hematopoietic bone marrow defect): An angle of mandible and tuberosity area, where hemopoiesis is seen.

Maxillary sinus retention cyst: Lining of maxillary sinus.

Mucocele, ranula and plunging ranula.

34. What are the types of aneurysmal bone cyst (ABC)?

ABCs exist in two clinicopathological forms—as a primary or as a secondary lesion arising from another osseous condition. It includes fibrous dysplasia, cementifying fibroma, giant cell granuloma and certain unspecified lesions.

Histologically Three Variants

Conventional or vascular type (95%): Osteolytic lesion with blood-filled cavities and sinusoidal spaces, separated by fibrous connective tissue septa with osteoid trabeculae. Variable amount of hemosiderin and giant cells can be found.

Solid type (5%): This form is non-cystic variant with osteoclast-like giant cells. Osteoblastic differentiation areas with osteoid and calcifying fibromyxoid tissue complete the picture.

Mixed type: A third form or mixed variant demonstrates features of both the vascular and solid types. It may be a transitory phase of the lesion because sudden activation or rapid enlargement of stable lesions has been reported.

35. What are different cystic lesions associated with maxillary sinus?

a. **Antral pseudocyst (benign mucosal cyst of maxillary sinus):** Detected on routine radiological examination (localised dull pain in antral region radiographically spherical, ovoid, dome-shaped radiopacity with smooth uniform outline). Most cases showed a possible source from an adjacent infection.

b. **Sinus mucoceles:** These are due to accumulations of mucin that are encased by epithelium. It occurs after trauma (surgical ciliated cyst, traumatic ciliated cyst) and surgery (postoperative maxillary cyst). It may occur from an obstruction of the sinus ostium blocking normal drainage.

c. **Retention cysts:** No evidence clinically and are located around the ostium or within antral polyps and discovered during histological examination of antral polyp. These are called intrinsic cyst of maxillary sinus.

Extrinsic Cysts

Develop outside sinus but encroach upon it.

- A thin sclerotic border usually delineates the cyst from the rest of the sinus.

- May occupy the entire sinus and may be difficult to diagnose.
- The differentiation is on that the normal antrum has an undulating outline whereas a cyst will be more rounded.
- Odontogenic-radicular, kerato, dentigerous and neurogenic meningocele—neurogenic cystic condition, i.e. surgical ciliated cyst which follows surgical procedure and sometimes after many years remains as an implantation cyst.

Intrinsic Cysts

These are not true cysts as they are not epithelial lined.

a. *Mucous retention cyst:* Due to obstruction of duct of minor seromucinous gland
b. *Serous retention cyst:* Inflammatory in origin. Consists of loculated fluid in the mucoperiosteum and thus has no epithelial lining
c. Mucocele due to gradually expanding collection of fluid within the sinus initiated by the obstruction of the ostium. Rare in maxillary sinus. Mucous retention cyst and serous retention cyst generally also referred to as mucocele.

36. Classify diseases of maxillary sinus.

1. **Developmental diseases:** Agenesis, aplasia, hypoplasia, supernumerary maxillary sinus
2. **Inflammatory diseases:** Mucositis, maxillary sinusitis, empyema, antral polyps
3. **Cysts of maxillary sinus:** Odontogenic, non-odontogenic, intrinsic and extrinsic
4. **Traumatic diseases:** Oroantral fistula, root/foreign body in antrum, pneumocele
5. **Tumors involving maxillary sinus:** Benign and malignant
6. **Other diseases:** Fibro-osseous diseases, granulomatous diseases, antroliths.

37. What is unicystic ameloblastoma?

The term unicystic ameloblastoma is an important example of this problem. It has been used to describe an ameloblastoma developing within the lining, lumen, or wall of a cyst as well as an invasive ameloblastoma that has a single cystic space rather than multicystic spaces.

38. What is ameloblastoma *in situ* and mural ameloblastoma?

Ameloblastoma *in situ* is an ameloblastoma developing in the lining or into the lumen of a dentigerous cyst, because dentigerous cyst epithelium retains some primordial odontogenic cells capable of ameloblastoma expression as discussed in the preceding section. The ameloblastoma *in situ* may initially encompass a focal area of the cyst lining (mural ameloblastoma).

39. What is peripheral ameloblastoma?

This ameloblastoma is not a true neoplasm and lacks the biologic potential of the central ameloblastoma. It is a hamartomatous proliferation of odontogenic epithelium arising from rests of Serres or perhaps from the basal cells of the oral mucosa. Usually not attaining a size larger than 3 cm, it will present as a firm single or polyploid mass arising exophytically from the gingiva. It does not invade bone, and, as a true peripheral ameloblastoma, it is not associated with a radiolucent area. Some central ameloblastomas have been misdiagnosed as peripheral types when they eroded through the alveolar bone and presented with a soft tissue mass. Peripheral ameloblastomas are managed by local soft tissue excision with 2 to 3 mm margins. Recurrence is not seen.

40. What is desmoplastic ameloblastoma?

The desmoplastic ameloblastoma is a rare but histopathologically and radiographically unique ameloblastoma. It is otherwise identical to the more common central ameloblastoma and is treated in the same fashion. Its unique radiographic appearance is that of a mixed radiolucent-radiopaque lesion unlike the strictly radiolucent quality of other ameloblastomas. Therefore, it is usually a surprise diagnosis from a differential list composed mostly of fibro-osseous diseases and odontogenic cysts and tumors that are characteristically radiolucent-radiopaque. These include ossifying fibromas, fibrous dysplasia, osteoblastomas, osteosarcomas, calcifying epithelial odontogenic tumors, and calcifying odontogenic cysts.

41. What is extreme ameloblastoma?

Occasionally, the oral and maxillofacial surgeon may be confronted with an extreme ameloblastoma of excessive size. These patients are usually from third world rural areas and brought to medical centres for treatment. Although these ameloblastomas are benign, they are nonetheless life threatening. Many have eventuated in death due to airway obstruction, starvation from restriction of feeding, and complications of hypoproteinemia produced partially by the restriction of feeding and partially by protein loss into the cystic spaces of the tumor, which then leaks it out through the mouth.

42. What is odontoameloblastoma?

The odontoameloblastoma is an extremely rare odontogenic neoplasm of which only a handful have ever been reported. It essentially represents an invasive ameloblastoma occurring simultaneously with an odontoma in what may be thought of as a "collision tumor". Neither seems to arise from the other. The unfortunate term ameloblastic odontoma is often used to describe this lesion as well, but its name believes the invasiveness characterized by an ameloblastoma. It instead suggests the biologic behaviour of an odontoma, which is entirely misleading; therefore, it should not be used.

The odontoameloblastoma, like most ameloblastomas, presents as a painless jaw expansion. The radiograph usually shows a multilocular, mixed radiolucent-radiopaque lesion, although it can also be unilocular. The odontoma component will most likely be of a complex type because most occur in the molar ramus region, but its mineral density will be greater than that of bone. It will approach the mineral density of dentin. This tumor is rare because of the remote likelihood of two coinciding odontogenic developmental disturbances: One an aberrant attempt at tooth formation, the other a neoplastic genetic alteration of odontogenic epithelium. There is no evidence that these two components are part of the same process.

43. What is malignant ameloblastoma?

Malignant ameloblastoma is defined as an ameloblastoma with typical benign histologic features that is deemed malignant because of its biologic behaviour, namely metastasis. The histologic features may not correlate with the clinical behaviour. The clinical behaviour and not the histology that justifies a diagnosis of malignant ameloblastoma (metastasizing ameloblastoma). Metastatic ameloblastoma are mostly seen in lungs.

44. What is ameloblastic carcinoma?

Ameloblastic carcinoma is a rare ameloblastoma and primary odontogenic malignancy exhibiting the histologic criteria of a malignant neoplasm, such as increased and abnormal mitosis and hyperchromatic, large, pleomorphic nuclei. It is a lesion which histologically shows the features of both ameloblastoma and squamous cell carcinoma. This may arise *de novo* or in pre-existing benign odontogenic tumor or cyst.

45. What is carcinoma ex-ameloblastoma?

It is a carcinoma arising from an ameloblastoma. It is also called carcinoma in ameloblastoma and ameloblastic carcinoma secondary type. This entity arises when an ameloblastoma undergoes dedifferentiation, that is, when a less differentiated proliferative clone arises within an ameloblastoma. This aggressive clone overgrows the ameloblastoma and becomes the dominant component.

46. What is *de novo* ameloblastic carcinoma?

If the carcinoma lacks component of conventional ameloblastoma its direct categorization as an ameloblastic carcinoma is less warranted. These lesions diagnosed on subjective interpretation are called *de novo* ameloblastic carcinoma.

47. What is atypical ameloblastoma?

Atypical ameloblastoma is used to denote lesions with fatal outcome for various reasons either metastasis, histological atypia or relentless local spread. It shows basilar hyperplasia

and increased mitotic index. Some authors called it proliferative ameloblastoma.

48. What is the differentiating point of AOT from dentigerous cyst in pericoronal location?

The AOT also shows complete radiolucency in pericoronal location in early stages and is difficult to distinguish from dentigerous cyst while evidence of more or less calcifications is suggestive of AOT.

49. What are the reasons for calling odontogenic keratocyst into keratocystic odontogenic tumor?

Keratocystic odontogenic tumor (KOT) is defined as "a benign uni or multicystic, intraosseous tumor of odontogenic origin, with a characteristic lining of parakeratinized stratified squamous epithelium and potential for aggressive, infiltrative behaviour.

1. Clinically shows aggressive growth.
2. A tendency to recur after surgical excision.
3. The mitotic activity is increased in cystic epithelium and tumor like characteristics of lining epithelium.
4. Potential for budding in the basal layer, unlike cysts which generally grow by osmotic pressure the epithelium has got innate potential consistent with benign tumor.
5. Presence of daughter cysts in cystic wall.
6. Association of nevoid basal cell carcinoma syndrome.
7. Chromosomal abnormalities and genetic alterations such as mutation of PTCH gene.

   Although the gross and radiological appearance is like cystic in nature.

50. Enumerate the syndromes associated with keratocystic odontogenic tumor.

Basal cell nevus syndrome (nevoid basal cell carcinoma syndrome or Gorlin-Goltz syndrome)—multiple nevoid basal cell carcinomas on skin, skeletal abnormalities, CNS abnormalities, eye lesions and multiple KOTs.

51. What are the various characteristics of keratocystic odontogenic tumor? Why the anteroposterior expansion is seen?

It may be asymptomatic, may be small, unilocular/large multilocular an odontogenic keratocyst may present in a variety of sizes and situations. Some are small and unilocular, others large and unilocular. The larger cysts will cause expansion and may cause tooth mobility. Some will rupture and leak keratin into the surrounding tissue, provoking an intense inflammatory response that causes pain and swelling. The cysts will not affect nerve sensation, although they will frequently displace the inferior alveolar neurovascular bundle to the inferior border.

Their resorption of bone will include cortex and inferior border, but at a slower rate than the intermedullary trabecular bone, which is less dense. Therefore, they extend further anteroposteriorly than buccolingually.

This principle of further extension through bone that is less dense also explains the finding of greater buccal expansion than palatal expansion in the maxilla. The cysts also frequently resorb the roots of adjacent teeth in a smooth and regular pattern.

52. Which tumor is associated with pain?

Osteoid osteoma (it is bone-forming tumor characterized by small size, limited growth potential and disproportionate pain) and osteoblastoma. The osteoblastoma is larger than 2 cm while the osteoid osteoma is smaller than 2 cm. The pain in osteoid osteoma responds to aspirin and other NSAIDs while in osteoblastoma does not.

53. Enumerate the bony lesions can cross midline.

Keratocystic odontogenic tumor, central giant cell granuloma, large ameloblastomas, calcifying odontogenic cyst, osteitis deformans cause midline. Osteosarcoma, glandular odontogenic cyst (sialo-odontogenic cyst), odontogenic myxomas cross midline especially in mandible, lymphomas, gigantiform

cementoma starting as multilobular masses, primary intraosseous carcinoma.

Fibrous dysplasia does not cross midline.

54. What are the intraoral sites of neurofibromatosis?

The tongue, alveolar mucosa and gingival.

55. What is axillary freckle sign?

One of the most characteristic locations for an NF-1-related café-au-lait macule is the axilla, which is specifically examined in suspicious cases (called the "axillary freckle sign" or "crowe sign"). Café-au-lait macules almost always develop years before neurofibromas. They can be distinguished from a freckle (ephelis) by their larger size and their occurrence on areas not exposed to the sun. These develop in 90% of NF-1 related patients, especially in middle of childhood.

56. In which condition bag of worm feeling is seen?

A neurofibroma may present as an asymptomatic mass within the subcutaneous or submucosal tissues. The mass will be diffused. The edges will gradually blend into normal tissue and there will be no clear distinction. The mass will infiltrate into and incorporate normal tissues such as muscle, glands, and lymph nodes. Its palpable quality will be that of a lobulated surface is called bag of worms feeling.

57. What are fine needle aspiration biopsy (FNAB) and fine needle aspiration cytology (FNAC)? Name the different aspirates.

FNAC are diagnostic procedures used to investigate superficial lumps or masses. In these techniques a thin hollow needle is inserted into the mass for sampling of the cells that after being stained are examined under microscope. There could be cytological examination of aspirate (FNAC) or histological examination (FNAB). Aspiration is withdrawal of fluid from body cavities. Aspirate is the material withdrawn. If nothing is been aspirated then several attempts should be made by moving to adjacent areas to obtain the aspirates. Aspiration of every swelling in oral cavity should be done. Aspiration should be done with 16 to 20 gauge needle. Some viscous fluids may not be aspirated.

Aspiration of pus indicates inflammatory lesions.

Radiolucent lesions in bone may represent idiopathic bone cavities, infections, cysts, and cystic or solid tumors. Aspiration of most radiolucent lesions is done before biopsy to assess for potential bleeding that may occur during the biopsy. Aspiration should be accomplished with a 20-gauge or larger needle. The lesion should also be aspirated in three areas by directing the needle tip to three locations within the lesion from a single entrance point. This maneuver is recommended because many lesions are compartmentalized into solid and fluid spaces. A 10 ml syringe containing 1 ml of saline is used. The clinician should observe the first component of the aspirate. If bubbles of air or a serosanguineous fluid precede blood, an idiopathic bone cavity is likely. Aspirated blood does not confirm an arteriovenous hemangioma. Lesions of lower vascular pressure such as cavernous hemangiomas, central giant cell tumors (including what was formerly called "aneurysmal bone cysts"), and idiopathic bone cavities will return blood upon aspiration. If blood is returned, it is useful to disconnect the syringe barrel from the needle. Low-pressure vascular lesions, which will not represent a significant clinical bleeding concern, will yield blood flow from the needle hub but stop within 1 minute. High-pressure vascular malformations will result in spurting blood from the needle hub.

- **Space abscess:** Usually aspiration produces yellowish pus.
- **Subcutaneous emphysemas and laryngoceles** are masses that are filled with air and can be completely deflated by aspiration.
- **Cysts:** When aspirated from cyst, cystic content must be withdrawn and then again aspirated to withdraw content from the

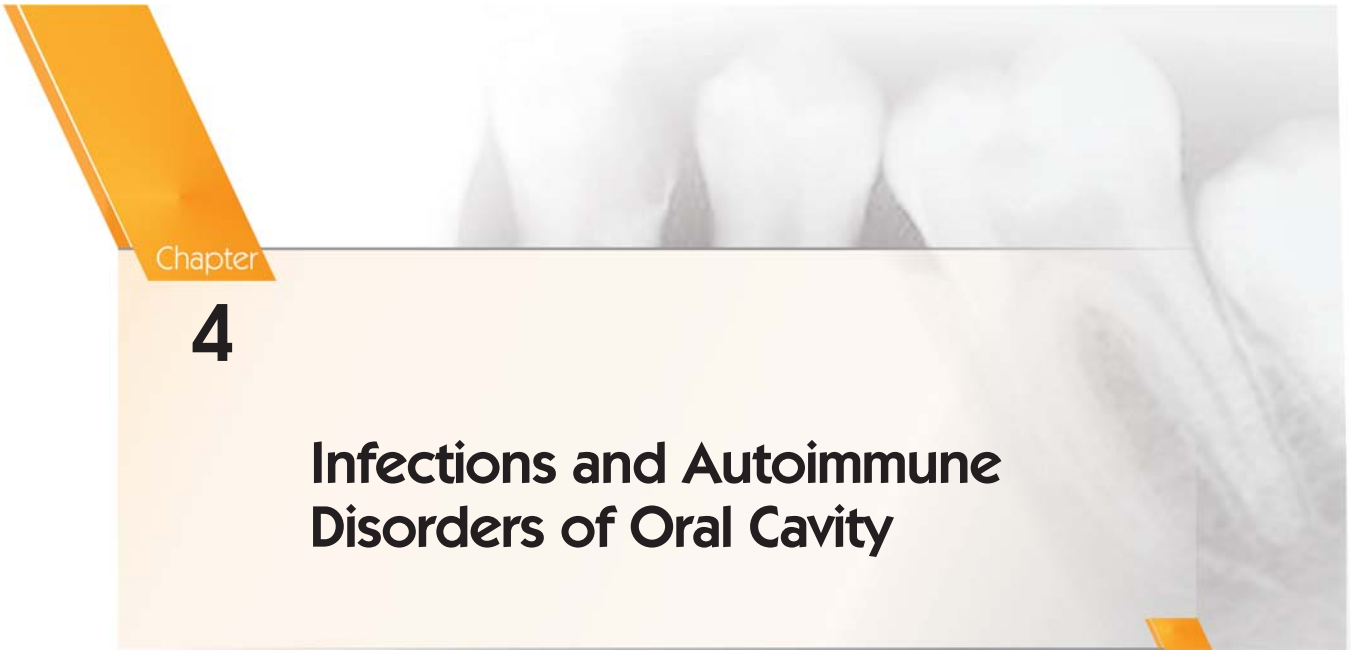
capsule of the straw-coloured fluid or blood-tinted fluid may indicate cyst. Odontogenic cysts both developmental and inflammatory share an identical cytomorphology. Aspirates contain abundant anucleated squamous cells similar in appearance to superficial and intermediate cells of stratified squamous epithelium, and occasionally keratocystic odontogenic tumor will show evidence of mineralization within squamous cells.

- **Non-infected cyst:** Shows a thin, straw-colored fluid.
- **Noninfected radicular cyst:** Produces alight, straw-colored fluid, usually containing an abundance of shiny granules (cholesterol crystals).
- **Mucocele, cysts of glands of blandin and nuhn, ranula and sometimes from the tumors of minor salivary glands:** Show a clear, viscous, sticky fluid. Occasionally, a low-grade mucoepidermoid tumor produces enough mucus to clinically resemble a mucocele and yields mucus on aspiration.
- **Benign retention cyst:** An aspiration is usually clear and has watery to viscous consistency.
- **Nodular oncocytic hyperplasia:** More than one nodule in salivary gland. Aspiration contains cohesive clusters of cells.
- **Acute inflammatory salivary gland lesion:** Fibrin, cellular debris, neutrophills, lymphocytes and histocytes.

  Ultrasound guided fine needle aspiration biopsy is useful in case of deep, mobile or non-palpable lesions. It is indicated for distinct salivary gland masses and for evaluation of pathology of submandibular space.
- **Sialocele:** Clear/non-transperant , stringy, sometimes blood tinged often brownish fluid with a very low cellular content.
- **Traumatic bone cyst:** Aspiration usually is fruitless, but in some cases serosanguineous fluid, a small quantity of blood or a serum-like fluid may be obtained.
- **Dentigerous cyst:** Aspiration often yields a straw-colored, thin liquid. Cholesterol crystals may be seen in the aspirate when the syringe is slowly rotated in front of a strong light.
- **Nasopalatine duct cyst:** It will usually return straw-colored fluid, confirming its cystic nature.
- **Keratocystic odontogenic tumor:** It may reveal a thick, yellow, cheesy material (keratin).
- **The papillary cystic adenoma and papillary cystadenoma lymphomatosum:** Are often fluctuant and contain thin, straw-colored liquid that can be aspirated.
- **Lipoma:** Aspirates of lipomas usually yield an oily fluid that when smeared has an glistening appearance.
- **An idiopathic bone cavity:** Aspirates will often return blood. It does so not because it is a blood-filled lesion, but because a tight needle fit through the cortex will cause an excessive negative pressure in the marrow space, which disrupts capillaries and causes a return of blood. To distinguish this phenomenon from the return of blood associated with an arteriovenous malformation or a cavernous hemangioma, 1 ml of saline should be placed in the syringe before aspiration and the first part of the return observed carefully. An idiopathic bone cavity will return a few air bubbles or a small amount of straw-colored fluid into the syringe before blood appears. The syringe should be disconnected from the needle after a syringe of blood is obtained, keeping the needle in place. If an idiopathic bone cavity is present, the oozing of blood from the needle will cease, whereas more prominent vascular lesions will show a continued brisk oozing or even a spurting. After aspiration, a cortical window is removed and the bony walls are curetted.
- **Warthin's tumor:** Will yield thin, watery mucoid appearance and consists of a mixed population of lymphocytes, occasionally plasma cells and oncocytes.

- **Ex-pleomorphic adenoma:** Usually cellular and contain dual population of malignant cells/residual benign PA cells. The aspirates are usually dominated by malignant cells because of low cohesiveness of malignant cells.
- **Lymphangioma:** Aspiration yields lymph fluid that is high in lipid content.
- **Hemangioma:** Aspiration shows bluish blood with a fine-gauge needle, scant of cellularity. The aspirates are characterized by a highly bloody background in which are dispersed single, plump, spindle-shaped cells and clusters of spindle-shaped to ovoid cells. Most of the aspirates are scant of cellularity. The aspirated tissue fragments often show a three-dimensional arcade architecture with preserved lumen.
- **Cavernous hemangioma:** Aspiration of an intrabony hemangioma readily yields a copious amount of blood and caution is necessary. A recommended approach is to introduce the needle through the mucosa some distance from the point where the bone is to be perforated rather than to introduce the needle directly over the hemangioma. The bleeding that results from the former method usually is more easily arrested. Since the mucosa over the point where the bone was penetrated is still intact and the channel through the mucosa can be more effectively compressed.
- **A-V shunt:** If on insertion of the needle, the plugger is almost forced out of the syringe by the flow of blood, an arteriovenous shunt is suspected, e.g. blood under pressure is A-V shunt. The hemostasis is difficult to attain.
- **Thalassemia:** Patients aspiration biopsy of the bone marrow reveals a specimen with very active, immature hematopoietic tissue.

FNAC is highly accurate procedure for differentiating benign and malignant lesions. Diagnosis of aspirates from cystic lesions may be less specific than the solid lesions due to paucity of specific lesional cells in the former and also due to superimposed infection.



Chapter

4

Infections and Autoimmune Disorders of Oral Cavity

1. What are epidemic, pandemic and endemic diseases? Give an example of each.

The element **dem** in *epidemic*, *endemic*, and *pandemic* comes from the ancient Greek word *demos*, which meant *people* or *district*.

An **epidemic** (*epi* (*among*) + *demos* = **epidemic**) is a widespread occurrence of an infectious disease in a community at a particular time. Annual influenza epidemics follow a winter seasonal pattern in the United States with typical activity peaking during late December to early February. An intense flu epidemic spreading across the nation has already taken a tragic toll in Michigan.

Endemic (*en* (*in*) + *demos* = **endemic)** is an adjective that refers to a disease or condition regularly found among particular people or in a certain area. In many malaria-endemic countries, malaria transmission does not occur in all parts of the country. Polio remains endemic in three countries—Afghanistan, Nigeria, and Pakistan. Endemic dental fluorosis in the North State of Minas Gerais, Brazil, pertussis is endemic worldwide, even in areas with high vaccination rates.

A disease becomes **pandemic** (*pan* (*all*) + *demos* = **pandemic**) when it spreads beyond a region to infect large numbers of people worldwide. The black death was one of the worst pandemics in human history, killing at least 75 million people on three continents, the Franco-Prussian war triggered a smallpox pandemic of 1870–1875 that claimed 500,000 lives. The 1918 spanish influenza pandemic is estimated as being responsible for the deaths of approximately 50 million people or more. Dental caries and periodontal diseases are pandemic. AIDS is also becoming endemic to pandemic.

2. What is focus of infection?

It is referred to as circumscribed area of tissue, which is infected with exogenous pathogenic micro-organisms and usually located near the mucous membrane or cutaneous surface.

3. What is focal infection?

It is a localized or generalized infection caused by dissemination of microorganisms or toxic products from focus of infection.

4. What are oral foci of infection? What is its significance?

Following varieties may present as oral foci of infection:

1. Infected periapical lesions such as cysts, granuloma and abscess
2. Teeth with infected root canals
3. Periodontal diseases with special references to teeth extractions and manipulations.

Bacteremia has been found to be closely related to severity or degree of periodontal disease present after manipulation of gingival or more commonly tooth extraction. Bacteremia can be produced by simple gingival massage. Transient bacteremia in patients who have undergone tooth extraction with periodontal disease. The holding of tooth in socket with forcep before extraction produces bacteremia.

Pumping action occurring during dental extraction may force microorganisms from gingival crevice into capillaries. Extraction of teeth and even minor trauma may produce bacteremia.

These oral foci may either cause or aggravate many systemic diseases. These are:

1. Arthritis—mainly rheumatoid type and rheumatic fever.
2. Valvular heart disease particularly SBE
3. GIT diseases
4. Ocular diseases
5. Skin diseases
6. Renal diseases.

5. What is granuloma? What are types of granuloma?

Granulomas are aggregates of macrophages that accumulate for the purpose of phagocytosis.

It is a distinct compact microscopic structure composed of epitheloid-shaped macrophages typically surrounded by rim of lymphocytes. Sometimes it may show central area of caseous necrosis.

There are three types of granulomas:

1. *Foreign body granulomas:* Foreign body granulomas, which consist of immature macrophages that accumulate because of the presence of concentrated inert material such as silica or talc.
2. *Epithelioid granulomas:* It consists of activated or mature macrophages and typically develop through the action of T-cells in a delayed hypersensitivity reaction. Often this is the consequence of infection by certain microorganisms, such as *Mycobacterium tuberculosis.*
3. *Immune granuloma.*

6. What are the reasons of granulomatous inflammatory conditions involving oral and paraoral tissues?

Foreign body reaction is the most common source. These may be endogenous/exogenous. The other infections such as tuberculosis, leprosy, cat-scratch disease, tertiary syphilis, histoplasmosis, cryptococcosis, blastomycosis, paracoccidioidomycosis, sarcoidosis, Crohn's disease, orofacial granulomatosis.

7. What is Miescher's granulomatosis?

It is the presentation of orofacial granulomatosis. The clinical presentation is often variable. The orofacial granulomatosis when involving lips alone is called Miescher's granulomatosis.

8. What are the causes of sinuses around lower jaw?

Osteomyelitis, tuberculosis, and actinomycosis.

9. What is woody tongue and bull neck?

It is seen in Ludwig's angina when there is a massive swelling of neck that often extends close to clavicles. Involvement of sublingual space results in elevation, posterior enlargement and protrusion of tongue which can compromise airway, this is called woody tongue.

Submandibular space spread causes enlargement and tenderness of the neck above the level of the hyoid bone that is called bull neck.

10. What are lepromas and what are oral manifestations of leprosy?

It is oral manifestation of leprosy. Lepromas are discrete focus of granulomatous inflammation and nodular lesion caused by leprosy on skin and mucous membranes. They usually appear in lepromatous leprosy and occur in 20–60% of cases. They may take the form of multiple nodules (lepromas) that progress to necrosis and ulceration. The ulcers are

slow to heal, and produce atrophic scarring or even tissue destruction. The usual location is on the hard and soft palate, in the uvula, on the underside of the tongue, and on the lips and gums. There may also be destruction of the anterior maxilla and loss of teeth. The diagnosis, based on clinical suspicion and the lepromin test.

In tuberculoid leprosy involvement of all nerves supplying the oral and related structures occurs, sensory and motor involvement of trigeminal and facial nerves also occurs. In lepromatous leprosy facial and oral involvement with ulceration, scarring and typical lepromas occur resulting in numerous esthetic and functional deformities. Nerve involvement may become manifest if the disease progresses. There may be transition and overlap of signs and symptoms between these two types.

11. What is patch test? How is it done intraorally?

Patch-testing is a diagnostic procedure that is most commonly used for identifying the possible causes of contact dermatitis (type IV hypersensitivity reaction). This can be done type I hypersensitivity reactions and in DRESS (drug reaction with eosinophilia and systemic symptoms) syndrome (type IVb hypersensitivity reaction).

The procedure involves mixing the allergen to be tested with white petrolatum (i.e. the vehicle), and applied in close proximity with the skin. The first reading is taken after 30 minutes to look for type 1 hypersensitivity reaction, while a second reading is taken after 48 hours to investigate for type 4 delayed hypersensitivity reaction. Between the 2 readings, the patient should be instructed not to wet, rub or scratch the testing area, avoid exercise and sweating. The readings are evaluated using the International Contact Dermatitis Research Group (ICDRG) grading system: '–' stands for negative, '+' stands for a weak (non-vesicular) positive reaction, '++' stands for a strong (vesicular) positive reaction and '+++' stands for extreme (bullous) positive reaction.

A pectin, gelatin, sodium carboxymethylcellulose, and plasticized hydrocarbon gel base (orabase) may be used as a vehicle to test for contact allergy within the oral cavity. The suspected chemical is incorporated in the vehicle and applied to the oral mucosa.

Fenretinide mucoadhesive patches were attached (q.d. 30 min for 10 consecutive days) to the right buccal mucosa (blank patches on left buccal mucosa) immediately posterior to the intraoral commissure of the upper and lower lips.

Intraorally patch testing can be done by keeping the antigen in the maxillary denture base and holding it in the mouth.

12. In which diseases amyloidosis is likely to occur?

It is likely to occur in chronic disease like rheumatoid arthritis, chronic osteomyelitis, chronic renal failure and 10% myeloma patients.

13. What is strawberry tongue and raspberry tongue?

These manifestations are seen in scarlet fever. The tongue develops a white coat within which reddened and swollen fungiform papillae stand out ("strawberry tongue"). As the disease progresses, the skin erythema fades and the white coating on the tongue is lost, leaving a swollen, irregular, beefy tongue ("raspberry tongue").

14. What is valley fever?

Coccidioidomycosis is endemic in United States that is known as valley fever.

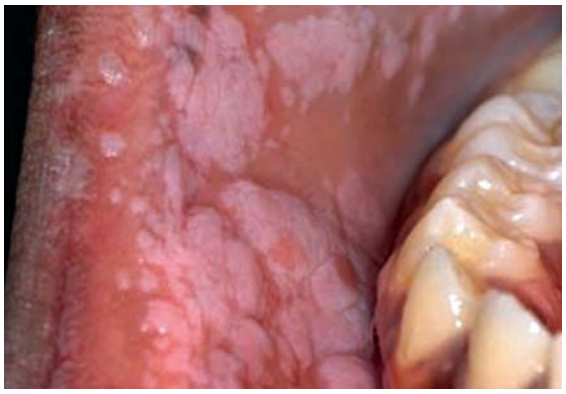
15. Name the lesion associated with HPV.

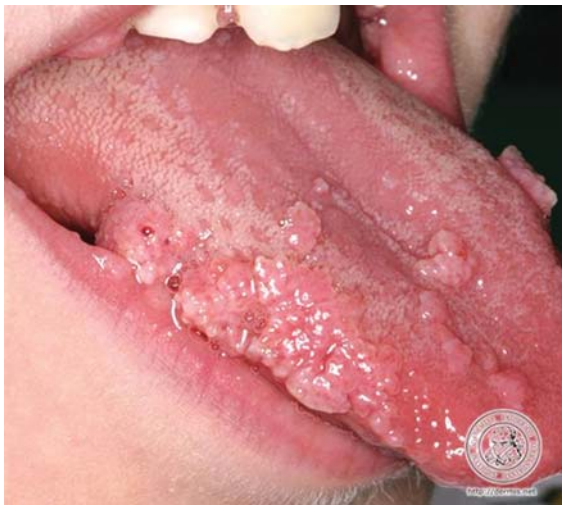
Oral papilloma (types 6 and 11) verruca vulgaris (types 2 and 57) and condyloma acuminatum (CA) (6 and 11) are benign, rough-surfaced exophytic hyperplasias of epithelial tissue (focal epithelial hyperplasia), dysplastic wart, verrucous carcinoma.

16. What is Heck's disease?

It is also called focal epithelial hyperplasia. These are most common in children with

normal mucosal color, caused by HPV. They are characterized by multiple slightly raised papules that can become papillary.





17. What are various lesions caused by herpes simplex virus (HSV)?

Primary herpes labialis, acute herpetic gingivostomatitis, recurrent herpes labialis, recurrent intraoral herpes simplex infection, herpetic genitalis, meningoencephalitis, conjunctivitis, disseminated herpes simplex infection of newborn, herpetic whitlow (herpetic paronychia).

Herpes gladiatorum (scrumpox): Infection in the wrestlers and other atheletes who have close physical contact with others.

Herpes barbae: Bearded region of face HSV infection into minor injuries created by daily shaving.

Eczema herpeticum (Kaposi's varicelliform eruption): Diffuse chronic skin disease and pemphigus may have life-threatening HSV infection.

18. Enumerate the diseases caused by coxsackie virus.

Hand-foot-mouth disease, herpangina, lymphonodular pharyngitis. Acute febrile respiratory disease, Bornholm *disease* (epidemic pleurodynia) *coxsackie* B (B is for body) *virus* can cause life-threatening pericarditis and myocarditis in infants.

19. Enumerate the diseases caused by varicella-zoster virus.

Primary disease (varicella, chickenpox), secondary disease (herpes zoster, shingles), post-herpetic neuralgia (morbid sequelae of HZI).

20. Enumerate the lesions associated with Epstein-Barr virus.

Infectious mononucleosis, Burkitt's lymphoma, oral hairy leukoplakia, Kaposi's sarcoma, rarely viral sialadenitis.

21. Enumerate the sites of latency of various viruses.

Coxsackie virus in connective tissue probably in the endothelium of connective tissue, white blood cells and monocytes.

Herpes virus after the primary infection travels along the sensory nerve axons and remains latent in the sensory ganglions such as (trigeminal ganglion). Extraneuronal latency is epithelium and plays role in lip lesions.

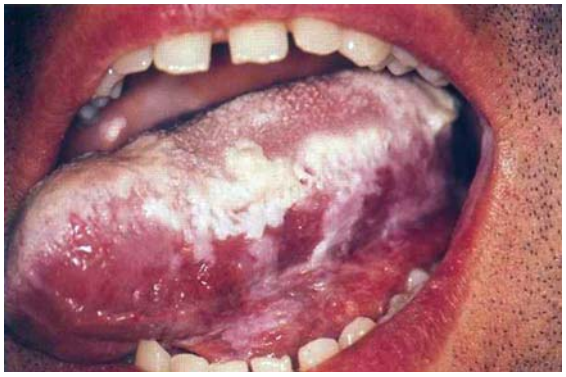
Varicella-zoster virus remains latent in dorsal root ganglia or ganglia of cranial nerves.

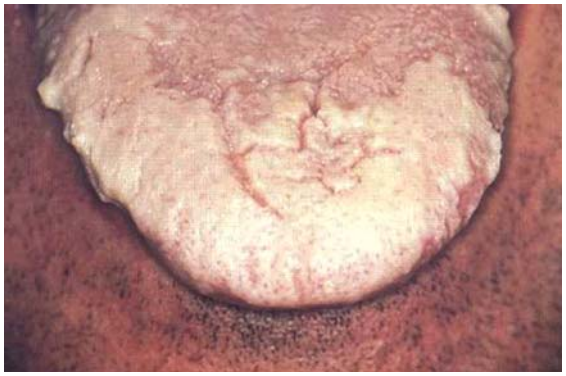
Cytomegalovirus establishes latency in certain undifferentiated cells like bone marrow derived cells of monocytes (CD 14+), CD34+, CD33+ progenitors are predominant sites of latency.

22. What is greenspan lesion?

Sir Waal Van Der (1996) suggested this term. Greenspan lesion, as an alternative term, is suggested for hairy leukoplakia. The term

'hairy leukoplakia' is a misnomer due to several reasons. First of all, hairy leukoplakia is a definable lesion. Furthermore, the lesion is not premalignant in nature. Therefore, the use of the term should be abandoned. As an alternative, the term 'greenspan lesion' has been suggested.





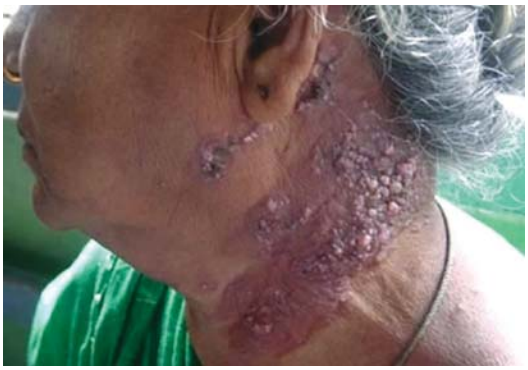
23. What are zosteriform lesions?

Lesions distributed along a dermatome or lesions take linear arrangement along nerve route. These are also called dermatomal patterns. Common examples are herpes zoster and sometimes lichen planus and psoriasis, other examples are zosteriform lentiginous nevus, zosteriform metastasis.

(Zosteriform metastatic skin-coloured solid papules, nodules, and papulovesicles scattered and confluent with a few crusted plaques seen on the left side of the neck in a typical zosteriform distribution involving the left C3 dermatome.)

24. What is Forchheimer's sign?

Forchheimer's sign refers to an enanthem of red macules or petechiae confined to the soft palate in patients with rubella. It may extend on hard palate. Generally arises with the rash, becoming evident in about 6 hrs after the first symptom and does not last longer than 12–14 hours. However, in 20% of the patients it appears during prodormal period or on the first day of the exanthema. Similar situation seen in infectious mononucleosis and has been termed Forchheimer's spots.



25. What is AIDS-related complex (ARC)?

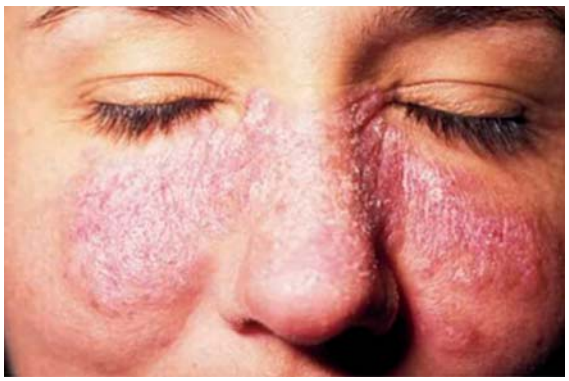
This is a syndrome caused by the AIDS virus and characterized primarily by chronically swollen lymph nodes and persistent fever—sometimes a precursor of AIDS. Fatigue, night sweats, diarrhea are other symptoms.

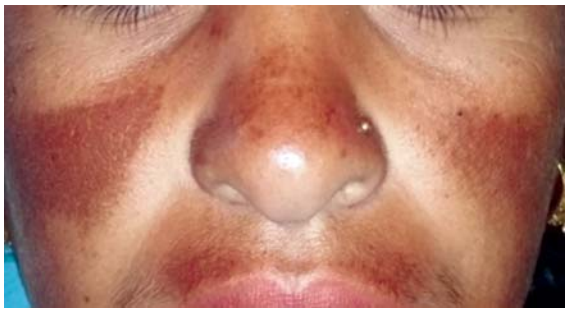
26. What is malar rash? Enumerate the conditions causing malar rash.

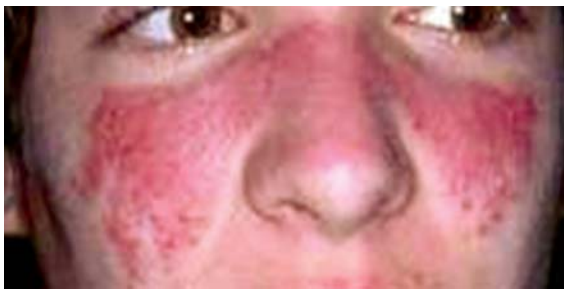
Malar rash (butterfly rash) is a mask-shaped erythematous eruption involving the malar areas and the bridge of the nose. It is characterized by a symmetrical, fixed, erythematous, maculopapular lesions with slight scales. It is transient (days to months). It is typical for systemic lupus erythematosus (SLE) (96%), other cases moderately sensitive (57%) may show findings.

Other causes are:

a. Other autoimmune diseases SLE, discoid lupus erythematosus (DLE) and dermatomyositis
b. Infectious diseases (Lyme disease, erysipelas)







c. Vitamin diseases (pellagra)
d. Some chromosomal disorders (Bloom syndrome)
e. Parvovirus.

It is seen in SLE, DLE and dermatomyositis. The malar rash of SLE does not involve nasolabial fold. It involves nasolabial fold in dermatomyositis, as it is caused by inflammation of muscles and skin.

Infectious causes include Lyme disease and erysipelas caused by streptococci. Lyme disease is diagnosed by patient's history, biopsy, ELISA and confirmed by western blot. Erysipelas diagnosed by pain, special stains and aerobic cultures.

Pellagra is determined by laboratory levels of vitamins (especially vitamin B_3)

Bloom syndrome (an autosomal recessive mutation in BLM gene) rash occurs in childhood and adolescence. It has to be confirmed by genetic studies.

Other cutaneous diseases include rosacea, drug-induced photosensitivities, sunburn, and pemphigus erythematosus. The dermatitides including atopic, allergic, contact and seborrheic, parvovirus, photoallergic reaction and polymorphous light eruption.

27. What is Gottron's sign?

It is a characteristic finding in dermatomyositis typified by scaly erythematous eruption seen on the dorsa of hands, metacarpophalangeal joints and proximal and interphalangeal joints.

28. What are the different lesions of systemic lupus erythematosus (SLE)?

The presenting symptoms of SLE are moreover nonspecific constitutional signs such as fever, fatigue and weight loss. Lupus is known as great mimic. Then involvement of variety of organs like:

- **Mucocutaneous disease (Malar rash, discoid lesions, photosensitivity and oral ulcerations):** Skin affected in 85% and cutaneous lupus can occur without multisystem involvement. Raynaud's phenomenon.
- **Renal disease:** It ranges from asymptomatic proteinuria to rapidly progressive glomerulonephritis (lupus nephritis indicator of poor overall outcome).
- **Musculoskeletal:** Arthralgia, nonerosive symmetric arthritis, myalgia and myositis.
- **Neuropsychiatric manifestations:** It is due to cerebral vasculitis (memory impairment, delirium or coma, seizures, psychosis).
- **Cardiovascular disease:** Vasculitis and pericarditis classically. Atherosclerosis is also noted (valvular heart disease).
- **Hematological manifestations (anemia, leukopenia, etc):** The disease course is characterized by disease flares which may require immunosuppression.

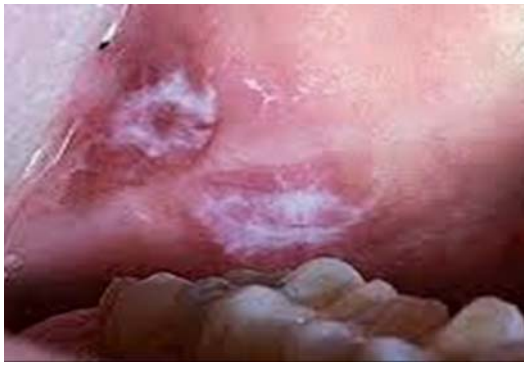
29. Describe oral manifestations of systemic lupus erythematosus.

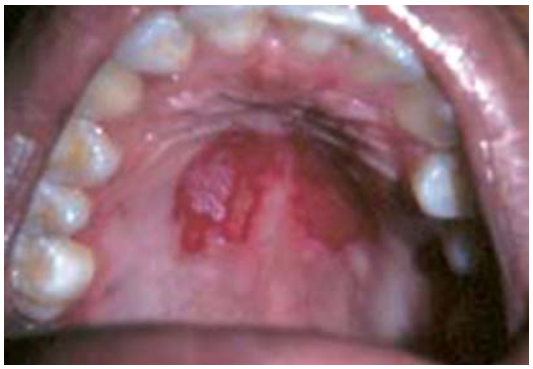
The predominant oral lesions are ulcerations, erythematous lesions, and discoid lesions.

The ulcerations are characteristically painless, occur frequently on palate and oropharynx.

Isolated erythematosus areas are also common especially on palate. Atrophy and telangiectasias are also present.

Discoid oral lesions are similar to those occurring on skin and appear as whitish striae frequently radiating from central erythematous area giving the so-called brush border appearance. Buccal mucosa, labial mucosa and gingival are common sites.





It is unclear if the presence of oral lesions is predictive of disease activity in SLE.

30. Which conditions show grass fire lesions?

These are the skin lesions of discoid lupus erythematosus. The development of dusky red localized skin plaques, 5 to 20 mm in diameter, on the face. These plaques will have a predilection for the hairline and sun exposed areas. A butterfly malar rash develops in DLE, just as it does in SLE. The skin lesions as they mature, they develop a broad atrophic white area with a perimeter of red resembling the pattern of a grass fire. These are called grass fire lesions. The scar within this white area will be devoid of hair follicles, leaving patchy bald areas in men's beards or a patchy alopecia on the scalp. In darker-skinned individuals, these lesions leave multiple depigmented regions.

31. What is patriotic sign?

It is seen in progressive systemic sclerosis (PSS) and crest syndrome. Raynaud's phenomenon is a vasospastic response brought on by cold. It is the initial clinical manifestation of all forms of PSS in 50% of cases. It can be brought about by immersing the individual's hands in cold water. Raynaud's phenomenon occurs in three stages: The first is pallor (white), which is due to vasospasms that are painful and paresthetic; the second is cyanosis (blue), which heralds relaxation of the vasospasm and is caused by pooling of venous blood; and the third is hyperemia (red), in which the relaxation of the vasospasm creates a reactive hyperemia that actually represents a mild reperfusion injury. Because the red, white, and blue are often seen together, Raynaud's phenomenon is known as the "patriotic sign".

32. What is Auspitz sign?

Sir Heinrich Auspitz described this terminology. It is the appearance of punctate bleeding spots when psoriasis scales are scraped off. This happens because there is thinning of the epidermal layer overlying the tips of the dermal papillae and blood vessels within the papillae are dilated and tortuous, which bleed readily when the scale is removed. It is also called Auspitz's symptom.

Demonstrated in three steps

- When the psoriatic plaque is scraped with glass slide the scales are accentuated and separate from plaque, as silvery flakes.

- A thin glazed membrane appears, which is removable into further scraping.
- Within a few seconds of mechanical removal of the membrane, the pinpoint droplets of blood appear on the erythematous surface.

33. What is Koebner's phenomenon?

In 1876 Sir Heinrich Koebner described this entity. It is also called 'isomorphic phenomenon' in patients suffering from psoriasis, the development of psoriatic lesions following trauma of normal appearing skin is known as 'Koebner's reaction or phenomenon'. Also seen in lichen planus and vitiligo.

34. What is angioedema?

It is a particular form of urticaria in which massive, abrupt and short-lived swelling occurs in the area especially, of low pressure, i.e. mid face, around eyes, neck and mucous membranes. Herditary and acquired (trauma, infection and temperature changes, medicine induced, allergic, idiopathic).

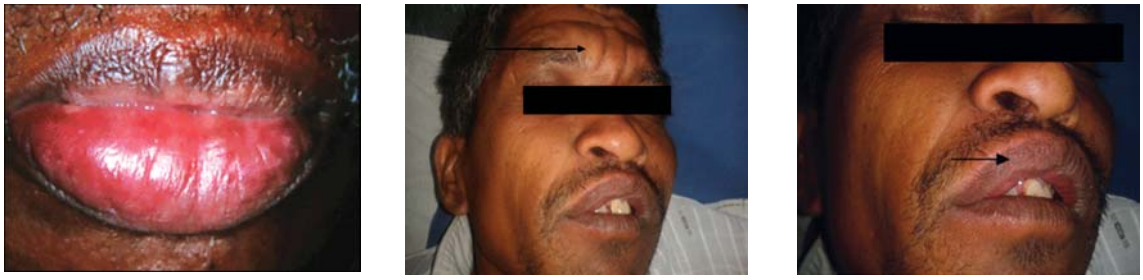
Angioedema is known side effect of drugs commonly used in day-to-day practice. Angioedema is generally self-limited most of the times but sometimes may result in respiratory tract obstruction, which can prove fatal . The angioedema associated with allergy or drugs typically present without urticaria.

The pathophysiology of angioedema consists of the increase in local vascular permeability causing plasma extravasation and consequently swelling of subcutaneous tissue. The major cells involved in the causation of angioedema are mast cells and in a lesser extent basophils, which on activation release histamine from preformed granules—a principle mediator involved in angioedema.

Oral lesions typically self-limiting and heal in 2–3 days.

35. What is acrosclerosis?

Scleroderma limited to hands is called acrosclerosis. It shows thinning of fingers with Raynaud's phenomenon.



Angioedema of lower lip and skin/upper lip following drug administration

Chapter

5

Potentially Malignant Disorders of Oral Cavity and Oral Cancers

1. What is potentially malignant disorder? Why this terminology has been implicated?

WHO (2005) defined potentially malignant disorder as the risk of malignancy being present in a lesion or condition either at time of initial diagnosis or at future date. Or, these can be defined as clinical presentations that may have a potential to become a cancer conveying the process of multistep process of cancer development, but unlikely on priori grounds, that there is uniformity in the way individual patients or tissue behave. These disorders may arise from preceeding lesions or may arise from normal appearing mucosa. Precancerous lesion and precancerous conditions were the terminologies used previously. Confusion prevailed between these two terminologies and many opinioned that the prefix 'pre' quotes that all the precancerous lesions will eventually become cancer, whereas studies found this to be untrue. Hence, it was recommended in the WHO workshop of 2005 to abandon the distinction between precancerous lesions and conditions and to use instead the term potentially malignant disorders incorporating both the terminologies.

The potentially malignant disorder conveys:

i. Not all the lesions and conditions described under this term turns into cancer.
ii. There is a group of conditions showing morphological alterations among which, some may have increased potential for malignant transformation.
iii. These disorders of oral mucosa are also indicators of risk of likely future malignancies elsewhere (normal appearing mucosa) in oral mucosa and not the only site-specific predictators.

2. Enumerate the potentially malignant disorders affecting oral cavity.

Recognized potentially malignant disorders are leukoplakia, erythroplakia, palatal lesions in reverse smokers, submucous fibrosis, acinic cheilitis, keratosis and elastosis and lichen planus; carcinoma *in situ* rare potentially malignant disorders include Fanconi's anemia, discoid lupus erythematosus, dyskeratosis congenita and xeroderma pigmentosum, epidermolysis bullosa, syphilis and sideropenic dysphagia.

Leukoplakia is most common potentially malignant disorders of oral mucosa.

3. Classify potentially malignant disorders of oral cavity.

1. **High risk:** Erythroplakia, leukoplakia, oral submucous fibrosis, erosive lichen planus.
2. **Lifestyle related:** Smokeless tobacco keratosis, reverse smoker's palate, actinic cheilitis.
3. **Infection:** Hyperplastic candidiasis, viral (HPV, HIV, EBV, HBV, HSV), tertiary syphilis.
4. **Immunodeficiency:** Solid organ transplantation, graft vs host disease, chronic cutaneous lupus erythematosus.
5. **Inherited disorders:** Xeroderma pigmentosum, dyskeratosis congenita, epidermolysis bullosa, Bloom syndrome, Fanconi's anemia.

4. Enumerate the malignant transformation potential of potentially malignant disorders.

Oral submucous fibrosis—the malignant transformation potential is 7% to 13%. Incidence over 10-year period at approximately 8%. Epithelial dysplasia in oral submucous fibrosis tissues appeared to be 7–26%.

Oral lichen planus—risk is low and the incidence of malignant transformation is 0–2% per year. The prevalence is between 0.3% and 3%. The forms that undergo malignant transformation are erosive and atrophic.

Leukoplakia has an overall malignant transformation rate of 15.6–39.2%. Homogeneous leukoplakia has a low annual malignant transformation rate between 1% and 7%. Verrucous leukoplakia, annual malignant transformation rate between 4% and 16%. Speckled leukoplakia, annual malignant transformation rate between 18% and 47%. Proliferative verrucous leukoplakia's (PVL) annual malignant transformation rate is between 63% and 100%. In Western countries somewhat high figures have been mentioned. Following factors may be considered while studying malignant transformation rate of oral leukoplakia—length of observation period, type of study, and therapeutic approach.

For erythroplakia the malignant transformation potential is very high at 14% to 50%. The prevalence is at 0.02–0.83%.

Actinic cheilitis malignant transformation potential ranges between 1.4% and 36%.

Plalatal keratosis associated with reverse smoking has a malignant transformation rate of 12.5%.

5. What are the various diagnostic aids in detection of potentially malignant disorders of oral cavity?

1. **Clinical methods**
   a. Conventional oral examination (COE)
   b. Vital staining
2. **Optical methods**
   a. Vizilite
   b. Micro Lux DL
   c. VEL scope
   d. Fluorescence spectroscopy.
3. **Imaging methods**
   a. Computed tomography (CT)
   b. Magnetic resonance imaging (MRI)
   c. Positron emission tomography (PET)
   d. Thalium-201 (201Tl) scintigraphy
   e. Photoactive imaging
   f. Optical coherence tomography (OCT)
   g. Narrow band imaging (NBI)
   h. Nano diagnostic methods
4. **Histopathological methods**
   a. Scalpel biopsy
   b. Oral CDx brush test
   c. Cytology
   d. Laser capture micro-dissection
5. **Molecular methods**
   a. Immunohistochemistry
   b. Flow cytometry
   c. Polymerase chain reaction (PCR)
   d. Blotting techniques
   e. Spectral karyotyping
   f. AgNOR
   g. Fluorescent *in situ* hybridization (FISH)
   h. DNA microarray
   i. Comparative genomic hybridization
6. **Salivary diagnostic methods**
   a. Protein electrophoresis
   b. Sialo chemistry

6. What are risk factors and risk indicators?

Risk factor: Something that increases the likelihood of a disease occurring.

Risk indicator: A marker for the presence of another disease or condition. Risk indicator term may be given to tumor markers like alpha-5 and beta-6, the proteins found in leukoplakia which indicates high risk of occurring cancer and surviving found in oral submucous fibrosis patient also indicates risk of carcinogenesis.

In other words, a risk factor is causative (i.e. chewing tobacco is a risk factor for oral cancer), while a risk indicator suggests that another condition will be present but is not causative (i.e. low educational and socioeconomic status and heart disease). People with less education and lower socioeconomic status may engage in unhealthier behaviour resulting in more heart disease, but their educational or socioeconomic status is not considered cause of heart disease.

7. What does CD stands for?

Cluster designation (CD) is a system for classifying cell surface markers that are expressed by leukocytes. These markers are used to distinguish cell lineages, stages of development, and functional subsets. The system is based on computer analysis of monoclonal antibodies against human leukocyte antigens with antibodies having similar specificity characteristics grouped together and assigned a number (e.g. CD1, CD4, etc).

8. How cancer word is derived? What is mortality rate of cancer?

Cancer is latinized from Greek word *karkinos*, meaning *crab*, denoting how carcinoma extends its claws like a crab into the adjacent tissues. Cancer is the second most leading cause of mortality in economically developed countries (following heart diseases) and the third most leading cause of death in developing countries (following heart diseases and diarrhoeal diseases). In the Indian subcontinent the prevalence of oral cancer is the highest among all cancers in men even though it is only the sixth most common cancer in men and 12th most common in women worldwide. Cancer of oral cavity accounts for 3% of all malignancies annually worldwide.

9. What are common signs and symptoms of malignancies in jaw bones?

Old age more common, male predominance, pain, paresthesia, loose teeth, premature tooth loss, and rapid growth of lesion (swelling/ulcer).

Radiographic appearances: Spike like resorption of teeth, ill-defined lesion, uniformly widened periodontal membrane space.

10. Compare the features of carcinoma appearing on upper/lower lip.

Carcinomas of the upper lip and commissure grow more rapidly, ulcerate sooner, and metastasize earlier than lower lip cancer. Prognosis of upper lip is worse while prognosis of lower lip is favourable. Approximately 90% lip cancers occur on lower lip, 7% on upper lip and remainder on commissures. The most frequent cause for lower lip cancers is ultraviolet radiation and for upper lip is basal cell carcinoma. They are treated as skin cancers rather than oral cavity cancers. Salivary gland tumors may be more common in upper lip than in lower lip.

Lesions larger than 2 cm or involving the upper lip or extending to the lateral commissure have a poorer prognosis. Mandibular involvement with the tumor also results in a poor prognosis and a higher incidence of regional metastases.

11. What are the characteristics of verrucous carcinoma?

Verrucous carcinoma is a separate and distinct form of oral carcinoma. It suggests a superficially spreading but non-metastasizing, non-invasive type of papillary and exophytic cancer, but this is true only of a true verrucous carcinoma. The true verrucous carcinoma will present as a rough textured, exophytic, white to red papillary lesion.

12. What are the other names and the common sites of occurrence of verrucose carcinoma?

Ackerman's tumor, Buschke-Löwenstein tumor, florid oral papillomatosis, epithelioma cuniculatum, carcinoma cuniculatum. The common sites are buccal mucosa, mandibular alveolar crest, gingival and tongue with glottis larynx is most frequent nonoral site.

13. Enumerate the fibroblastic malignancies.

Fibromatoses, fibrosarcoma, malignant fibrous histiocytoma.

14. What is odontogenic carcinoma?

Residual odontogenic epithelium from several sources (rests of Serres, rests of Malassez, and reduced enamel epithelium) is composed of cells that are sufficiently undifferentiated so that a series of genetic alterations can produce specific oncogenes or cause the loss of tumor suppressor genes, resulting in a malignancy. Today, we recognize four types of odontogenic carcinomas: Primary intraosseous carcinoma ex-odontogenic cyst (the primary intraosseous carcinoma ex-odontogenic cyst may present as an uncomplicated dentigerous cyst; the diagnosis can be established only when the histopathologic studies are completed).

Primary intraosseous carcinoma de novo: The primary intraosseous carcinoma *de novo* presents as a more clinically aggressive and radiographically destructive radiolucent lesion in which the border with adjacent bone is irregular. It may also cause an irregular and jagged type of root resorption, in contrast to the smooth regular root resorption seen with odontogenic cysts and benign tumors.

Malignant ameloblastoma: The malignant ameloblastoma will most commonly present as a multilocular radiolucent jaw expansion typical of a benign ameloblastoma, but with a concomitant metastatic focus, usually in the lungs.

Ameloblastic carcinoma: Ameloblastic carcinoma differs from the malignant ameloblastoma in the following features. First, the pre-ameloblasts are cytologically atypical, whereas in the malignant ameloblastoma they appear cytologically benign. Their behaviour is similar to that of an aggressive squamous cell carcinoma, for which they are often mistaken until the histopathology identifies the pre-ameloblast cellular atypia.

15. What are clinical presentations of oral squamous cell carcinoma?

The clinical presentations include:

a. **Exophytic variety:** It is mass forming generally irregular, fungating, papillary and verruciform variety. It is often indurated. The color may vary from red to white depending upon vascularity and keratin production respectively.
b. **Endophytic variety:** This is invasive, burrowing and ulcerated variety. The most common presentation is depressed, irregular shaped, ulcerated, central area with surrounding rolled border with normal, red, white mucosa. Rolled edges may occur due to invasion of tumor downward and laterally under the adjacent epithelium.
c. **Leukoplakic:** It is white patch.
d. **Erythroplakic:** It is red patch.
e. **Erythroleukoplakic:** Combined with red and white patch.

16. What are different presentations of carcinoma of tongue?

It is mostly asymptomatic (two-thirds). The common presentations are an indurated, painless masses or nonhealing ulcer, as a red lesion, as a white lesion and as a red and white lesion. The most common location is posterior lateral border then anterior lateral border, ventral surface and rarely dorsal surface. It is the site of only congenital carcinoma reported.

17. What are characteristics of tongue cancer?

Infiltrative and exophytic are the two most common morphologic types of cancer. Exophytic types have less tendency to infiltrate deeply. Early in the disease they appear as an area of focal thickening or clinical leukoplakia

or as a painless superficial ulceration. The infiltrative type may show minimal or no surface ulceration until late in its development.

Cervical metastases occur more frequently from cancer of the tongue than any other site within the oral cavity. Forty percent of patients have nodal metastases on admission.

Bilateral or contralateral metastases are uncommon, in contrast to tumors of the tongue base, but may be present when the tumor involves the midline of the tongue. Metastases usually occur first in upper deep jugular (subdigastric) lymph nodes and then spread downward along the jugular chain. Tumors arising from the anterior third of the tongue tend to metastasize slightly less frequently than those arising from the middle third. Unlike in the lip, the size of the primary lesion in tongue cancer is not necessarily correlated with the presence or likelihood of cervical metastases.

18. What are most common intraoral malignancies? What are their common intraoral sites?

Squamous cell carcinoma of tongue is most common intraoral malignancy, the most common location is posterolateral surface of tongue (25–40%), floor of mouth is second most site (15–35%). The floor of mouth is almost as frequently as tongue in men, but is involved much less frequently in women. In descending order of frequency is soft palate, gingival, buccal mucosa, labial mucosa and hard palate.

19. Enumerate the hematologic malignancies.

There are three main groups of hematologic malignancies: Leukemia, lymphoma, and plasma cell tumors.

20. What are common oral cancers and oral squamous cell carcinomas?

Acinic cell carcinomas are second most common intraoral malignancies. Mucoepidermoid carcinomas are most common malignancies of salivary gland (26%), i.e. on palate (50–65%) followed by polymorphous low grade adenocarcinoma non-Hodgkin's disease. Lymphoma is second most non-epithelial malignancy involving salivary glands. Lymphoma is second most malignancy in AIDS patients after Kaposi's sarcoma.

21. Which is rare location for I/O squamous cell carcinoma?

In the hard palate the squamous cell carcinomas are rare but adenocarcinoms are common in this area.

22. What are the morphological types of squamous cell carcinoma in oral cavity?

Three gross morphologic growth patterns of squamous cell carcinoma occur in the oral cavity: Exophytic, ulcerative, and infiltrative. Malignancies often display more than one of these manifestations.

The exophytic form is least common, except on the lip. The term exophytic is used to describe outwardly growing tumor. It tends to grow more superficially and metastasizes later, less frequently than the other types. This form begins as an area of thickened epithelium, which heaps up and can protrude 1 cm or more above the surrounding mucosa. Ulceration occurs early in its development. Exophytic carcinomas gradually become deeply infiltrative in more advanced cases. On the lip this form of tumor may reach a size of 6 or 7 cm, with little local destruction of tissue.

The ulcerative type is the most common form of squamous cell carcinoma in the oral cavity. These cancers burrow deep into mucosa with breach in surface. It begins as a round or oval ulcer with a grey, shaggy base that bleeds readily. Ulcerative types manifest a greater tendency for rapid infiltration and usually have a higher histologic grade than the exophytic type. The ulcer eventually may heap up and become exophytic or remain lower than surrounding mucosa. The metastasis is more frequent than exophytic cancers.

Infiltrative malignancies are common in the tongue and initially appear as a firm mass or plaque covered by mucosa. This type of tumor extends deeply into underlying tissues,

with minimal elevation above surrounding mucosa. As the neoplasm progresses, ulceration and exophytic manifestations may be observed.

Verrucous carcinoma, which is a clearly defined but uncommon variant of squamous cell carcinoma. The term verrucous indicates its fine finger like projections. It typically occurs in elderly patients with poor oral hygiene or ill-fitting dentures and most commonly affects the buccal mucosa of males and females with a history of tobacco chewing or snuff dipping. The tumor has a warty, bulky, elevated, and fungating appearance. It may grow considerably through lateral spread and occasionally may be multifocal; it does not invade deeply into underlying tissue. Verrucous carcinomas have an indolent biologic behavior and do not metastasize.

Tumors must be sectioned serially so that the entire specimen is examined for a more invasive squamous cell carcinoma. It has got excellent prognosis.

23. What is secondary osteosarcoma?

Osteosarcoma occurring at the site of another disease process is called secondary osteosarcoma. Commonly seen above 50 years of age. The disease is seen along with Paget's disease, previous radiation treatment, fibrous dysplasia, endochondromatosis, giant cell tumor, bone infarcts, chronic osteomyelitis, osteochondromas, and osteogenesis imperfecta.

24. What are the reasons of death in cancer patients?

The following are the mechanisms of death in order of their frequency.

Tumor load, infection (pneumonia), complications of treatment, progression of comorbidities, paraneoplastic syndromes.

Tumor Load

It is the most common, the slowest, and the gentlest pathway to death from oral cancers. Most patients lose weight, become weak, lapse into a coma, and die from a cardiorespiratory arrest. The weight loss and anemia of chronic disease develop despite gastric feeding tubes, iron and vitamin supplements, and even metabolic steroids. It has been observed that individuals will lose weight despite the intake of over 5,000 calories per day and otherwise normal bowel absorption. This is possible because diabolic nature of cancer, i.e. its replication rate, enzymes, and growth factors it secretes. The normal cells are prevented from utilizing and metabolizing nutrients efficiently because as the tumor load increases, the tumor secretes a greater amount of blocking factors and several vascular down-regulating enzymes, known as antiangiogenic factors. These antiangiogenic factors, which were first discovered in cancer cells, prevent normal tissues from recruiting new capillaries and instead promote the formation of new capillaries into the cancer.

It is important to note that this "tumor load" effect often is thought of as a failing immune system. Such a concept is incorrect. The white blood cell and immunoglobulin counts are usually normal and in proper function. Even when individuals progress to pneumonia, it is not caused by a tumor-related depression in their immune cells, but rather a loss of their protective reflexes and their physical barriers to infection or from medication-induced immune dysfunction.

Infection (Pneumonia)

As the tumor load increases and causes systemic weakness, infections, specifically pneumonia, become more likely. Individuals are often anemic, hypoproteinemic, and possibly malnourished by the secretory products of the tumor load (discussed above) and/or by the tumor-related dysfunction of the oral cavity and the effort required to eat or to be fed by someone else. This weakness and subsequent sedentary existence promotes atelectasis, which progresses to pneumonia. Many individuals are also prone to pneumonias via aspiration related either to the tumor itself, to surgery, or to radiotherapy. In

addition, narcotic analgesics required for pain control both reduce the cough reflex and depress respiration, thus enhancing the likelihood for a pneumonia.

Complications of Treatment

A smaller percentage of patients die from direct complications of treatment or from side-effects of therapy. Chemotherapy occasionally produces a severe marrow suppression, reducing white blood cell counts to less than 500/mm^3 and resulting in a fatal systemic infection. Surgery has produced events such as the "carotid blow-out" or internal jugular vein exsanguination. It also poses the threat of upper airway obstruction, where patients die after the attachments of the tongue are removed or bulky flaps are placed in the airway. Even when tracheotomies are accomplished, obstruction can occur from dried secretions in the tracheotomy cannula or from displacement of the tracheotomy as a result of patient movement. More rarely, exsanguinations result from the tracheotomy cannula eroding through the trachea and then through the brachiocephalic vein. This is usually associated with long-term tracheotomies requiring ventilator support. Radiotherapy rarely produces life-threatening complications during its treatment. However, the delayed effects of radiotherapy can be a significant factor in the patient's death. Progressive later dysphagia from fibrosis in the pharyngeal musculature reduces nutritional intake and promotes aspiration. Osteoradionecrosis, limited jaw opening, xerostomia, caries, and secondary candidiasis commonly affect anyone who has had therapeutic radiotherapy over 6,000 cGy.

Progression of Comorbidities

Most of these patients are often older and has a significant alcohol and smoking history. Many who present with an oral cancer also have hypertension, chronic bronchitis, emphysema, peripheral vascular disease, and ischemic cardiovascular disease. The physiologic impact of surgeries, chemotherapy, and radiotherapy often exacerbate these diseases, making them more difficult to control and increasing their rate of progression. It is not uncommon to have gained some control or even a potential cure of the oral cancer at 3 years only to have the patient die of a myocardial infarction or a cerebral vascular accident.

Paraneoplastic Syndromes

As discussed in the tumor load section above, cancers secrete a wide variety of enzymes, growth factors, and blocking factors. When a tumor secretes a growth factor or a hormone with a single physiologic response, it is often termed a paraneoplastic syndrome. One example of this is commonly seen arising from small cell (oat cell) lung carcinomas, but can also be seen rarely arising from some oral cancers. This is the tumor secretion of a parathyroid hormone-related peptide (PTHrp). This PTHrp is sufficiently close in its amino acid sequence and its active site conformation to native PTH that it has an identical effect. Such patients become significantly hypercalcemic, often with accompanying overt symptoms of confusion, constipation, and bone pain. Untreated, this leads to death. Similarly, some oral cancers will trigger a syndrome of inappropriate antidiuretic hormone (SIADH) by secretion of a small peptide chain nearly identical to antidiuretic hormone (ADH). These patients will have an independent cancer-related increase in ADH above the regulated native ADH levels, causing them to retain free water, develop hyponatremia, and undergo a characteristic decreased serum osmolality (> 280 mOsm/kg) and an increased urine osmolality (< 150 mOsm/kg). Another example that may be seen in advanced cancers is a paraneoplastic pemphigus, in which the cancer secretes desmoplakin to which the immune system develops antibodies. These antibodies attack the intercellular bridge area, as often occurs in autoimmune pemphigus vulgaris, because of the

similarity of the desmosomal antigen in pemphigus (desmoglein) to the cancer-related antigen desmoplakin. The result is the development of progressive, painful vesiculobullous lesions.

25. How the cancer embolus travels from oral cavity to lungs?

The mechanism of oral cavity primary site metastasis to the lungs is via the venous system. The route begins with:

1. The intravasation of cancer cells into a small vein;
2. This cancer embolus may then drain into the pterygoid plexus or another local vein;
3. It then drains into the larger veins, such as the facial vein or retromandibular vein, and into the internal jugular vein;
4. it then flows through the brachiocephalic vein, which forms at the junction of each internal jugular vein and the respective subclavian vein;
5. The cancer embolus next passes through the superior vena cava into the right atrium of the heart;
6. As the heart contracts, the cancer embolus is pumped past the tricuspid valve into the right ventricle;
7. From there, further cardiac contractions, pump it through the pulmonary valve into the pulmonary artery.

The cancer embolus then passes further into the branches of the pulmonary artery system, which progressively narrows, until it physically wedges into a small arteriole or capillary.

26. Which malignancies of body are likely to get metastasize to jaw bones?

Statistics vary somewhat among reports, but the most common originating site is the female breast (35%) followed by the lungs (28%) and kidneys (15%). After these, several other areas deposit metastatic foci in the jaws at nearly the same relative incidence: Prostate (6%), thyroid (6%), stomach, and lower gastrointestinal tract (6%).

27. Which the body sites are likely to get affected by malignancies of body metastasis?

The most frequent organs involved, ranked in decreasing order are: Lung, bone, liver, gastrointestinal tract, brain, skin, and kidney. Carcinomas metastasizing to the bone are most frequently adenocarcinomas.

28. What is occult neck disease?

It is defined as cancer present in the lymph nodes in the neck that cannot be palpated clinically. Prophylactic neck dissections are important in the management of early stage of oral squamous cell carcinoma. This is necessary for tongue squamous cell carcinoma, while floor of mouth, bucccal mucosa, maxillary gingiva, mandibular gingival and lips carry lower but significant risk of the same.

29. What is oral field cancerization?

Oral field cancerization (OFC) implies that oral cancer does not arise as an isolated cellular phenomenon but rather as an anaplastic tendency involving many cells at once and results in the multifocal development of cancer at various rates within the entire field in response to a carcinogen especially tobacco. It explains the concept by which the secondary primary tumors (SPTs) develop. The mucosa of head and neck had undergone a change due to exposure of carcinogen, and therefore more susceptible to development of many foci of malignant cells. Oral verrucous carcinoma associated with leukoplakia or submucous fibrosis may be indication of "field cancerization".

1. Large areas of arodigestive tissue are affected by long-term exposures to carcinogens. In this preconditioned epithelium multifocal carcinomas can develop as a result of independent mutations.
2. A single cell is transformed and gives rise to one large extended premalignant field by clonal expansion and gradual replacement of normal mucosa.

30. What is fixation and induration?

Fixation occurs due to abnormally dividing cells invading the deeper areas of muscles and bone. Induration is mucosal firmness or hardness caused by an increased in number of epithelial cells secondary to an inflammatory infiltrate. Both are signs of carcinomas. Induration can be seen in granulomatous and inflammatory diseases.

Fixation and induration may be result of neoplastic infiltration of deeper tissues if the ulcer is malignant. In these instances fixation is not accompanied by tenderness.

31. What is WHO scale for oral mucosites?

Grade 0: No oral mucositis

Grade 1: Erythema and soreness

Grade 2: Ulcers, able to eat solid foods

Grade 3: Ulcers, require liquid diet because of mucositis

Grade 4: Ulcers, alimentation not possible because of mucositis.

32. What is sentineal node?

Gould et al. (1960) described the term sentineal node. The first tumor in drainage pathway of malignant tumor or it is first lymph node to receive drainage from tumor.

Metastasis of oral squamous cell carcinomas is mostly to the regional lymph nodes of the neck. Cancers that have metastasized from the anterior floor of the mouth, anterior alveolar ridge between the mental foramen, and lower lip will first appear in the submental triangle lymph nodes along one of the anterior digastric muscles (level I, green).

Cancers located more posterior in the floor of the mouth, in the oral tongue, the buccal mucosa, or the alveolar ridge between the mental foramen and the retromolar trigone will usually first appear in the submandibular triangle (level II, yellow).

The submandibular triangle lymph node, which is located on the surface of the submandibular gland and just deep to the marginal mandibular branch of the facial nerve, is often called the node of Stahr after the German anatomist Hermann Stahr, who in 1909 noted that cancer of the tongue would settle in this node.

Cancers that appear in the retromolar trigone, tonsillar fossa, and pharyngeal tongue will often first appear in the jugulodigastric lymph node located where the posterior digastric crosses the internal jugular vein (level III, orange). Squamous cell carcinomas arising from the nasopharynx area metastasize to the posterior digastric lymph nodes around the spinal accessory nerve (level V, pink). Each of these nodes is referred to as the sentinel node for its respective tumor location.

33. Enumerate the various chainside investigations.

Pulp vitality test, toluidine blue staining, Lugol's iodine brush biopsy, Velscope, intraoral radiograph, aspiration biopsy, incisional biopsy.

34. Enumerate various NRT products.

Inhaler, *trans* dermal patch, nicogel, nicotine pastiles, magneto therapy, chantix, buccal spray, electrical cigars, Zyban, gum, nasal spray.

Chapter

6

Diseases of Salivary Glands, Pigmented Lesions of Oral Cavity

1. Enumerate the diseases associated with salivary gland ducts.

1. **Accessory salivary gland ducts:** Extra ducts may be present which either join the main duct or may open separately in the oral cavity. The most common location is superior and anterior to Stensen's duct.
2. **Diverticuli:** A diverticulum is pouch or sac protruding from the wall of duct, in major salivary gland duct may produce pooling of saliva and recurrent sialadenitis.
3. **Atresia of salivary ducts:** Rare congenital disorder absence of ducts or even occlusion of ducts that may produce retention cyst.
4. **Sialodochitis:** Inflammation of salivary gland duct.
5. **Salivary duct:** Cysts are acquired. Cysts that are believed to develop from marked cystic dilatation of a salivary gland/duct, and ductal obstruction is thought to be a principal etiologic factor. Salivary duct cyst may occur in major (parotid) or minor glands.
6. **Mucocele:** Retention or extravasation type retention of saliva due to obstruction and rupture of duct and spillage into mucosa respectively, ranula may be due to extravasation/retention in floor of mouth, the extension in neck is ploughing ranula.
7. **Salivary fistula:** Injury to the duct may lead to extravasation of saliva in the gland tissue, if the main duct is involved it is called 'major fistula' and if collecting ducts are involved it is called minor fistula.
8. **Stricture is narrowing of salivary gland duct:** It may produce obstruction. It may result from faulty chewing habit or external trauma. It may produce inflammation and swelling of gland. Four types of strictures are seen: (1) Sialectasis, (2) Pseudosialectasis, (3) Punctate sialectasis, (4) Sialodochitis. Sialectasis (sialodochitis) is "dilation of a salivary duct" (ptyalectasis) from [sial – + Greek *ektasis*, a stretching]: A condition resulting from duct obstruction of the parotid or submandibular glands associated with pain and swelling. The term pseudosialectasis has been introduced for the radiographic appearances of these lesions as in autoimmune disease, they represent duct disruption than ectasia. Punctate sialectasis refers to saccular dilation of small ducts within gland, while sialodochitis refers to dilation of large ducts.

9. **Darier's disease:** It also shows abnormalities like duct dilatation with periodic strictures in the main system.
10. **Salivary duct stone:** Sialolithiasis most commonly seen in submandibular duct.
11. **Ductal papillomas:** Sialoadenoma papilloferum, inverted ductal papilloma, intraductal papilloma.
12. **Salivary duct:** Carcinoma, intraductal carcinoma.
13. **Iatrogenic perforation:** Iatrogenic perforation of salivary gland duct specially parotid gland due to its curvature during sialography.

2. What are the functional disorders of salivary glands?

Saliva is stimulated by parasympathetic and sympathetic nervous system. The nature is watery (serous) and thick (mucinous) respectively.

Sialorrhea (Ptyalism)

It is increased salivation. The causes are:

1. **Physiologic causes:** Infancy, starvation, during eruption of teeth, foreign body (may be dentures)
2. **Pathologic causes:**
   a. *Local causes:* ANUG, erythema multiforme, metal poisoning, ulceration
   b. *Systemic diseases:* Mental retardation, epilepsy, racial paralysis, alcoholic neuritis, parkinsonism, morphine addiction
   c. *Drugs:* Potassium iodide, pilocarpine, mercurial salts.

Xerostomia (asialorrhea, pasties, cottonmouth)

It is decreased production of saliva. The causes are:

1. **Physiological:** Menopause
2. **Pathological causes:**
   a. *Local causes:* Salivary gland diseases (aplasia, hypoplasia, irridation of salivary glands, etc.)
   b. *Systemic causes:* Fever, dehydration, lung infections, typhoid, uncontrolled diabetes, hyperthyroidism, vitamins A and B complex deficiency, occupational exposure to dust and Zn poisoning, Sjögren's syndrome, dehydration or renal failure, and shock
   c. *Drugs:* Antihistaminics, anticholinergic (atropine), tricyclic antidepressants, antiparkinsonism, antihypertensive drugs, monoamine oxidase inhibitor.

3. Enumerate obstructive diseases of salivary glands.

Obstructive diseases are more common in submandibular gland than parotid gland.

These are mucous retention cyst (obstructive sialoadenitis), sialolithiasis, strictures and stenosis.

Stenosis may be of four types:

1. Membranous stricture
2. Large stricture
3. Diffuse stenosis of main duct
4. Diffuse generalized stenosis

4a. Without localised strictures
4b. With multiple strictures.

4. Mention unilateral causes of salivary gland swellings.

1. Unilateral swelling with acute pain, swelling, fever (acute bacterial sialoadenitis)
2. Unilateral swelling with recurrent swellings pain (chronic sialoadenitis, lymphoma, Sjögren's syndrome, sialolithiasis)
3. Unilateral painless slow growing mass (neoplasm benign or malignant)

5. Mention bilateral causes of salivary gland swellings.

1. Bilateral swelling with acute pain, fever (viral sialoadenitis)
2. Bilateral swelling with systemic symptoms and slow course (Sjögren's syndrome, lymphoma and mycobacterial disease).

6. What are the non-neoplastic enlargements of parotid gland region?

Non-neoplastic enlargements of parotid gland are as follows:

Inflammation or Infection

Bacterial
Viral (mumps)
HIV sialopathy

Acute suppurative sialoadenitis, sclerosing polycystic adenosis
Chronic sialoadenitis, uveoparotid fever

Autoimmune Diseases

Chronic recurrent parotitis
Sjögren's syndrome
Granulomatous diseases
Sarcoidosis, tuberculosis, actinomycosis
Wegener's granulomatosis
Kimura's disease

Miscellaneous

Sialosis
Sialadenosis (hormonal, thyroid, drug-induced)
Polycystic disease
Pneumoparotid
Benign lymphoepithelia lesions (Mikulicz's disease)
Parotid cyst
Radiation sialadenitis

Obstructive Enlargement

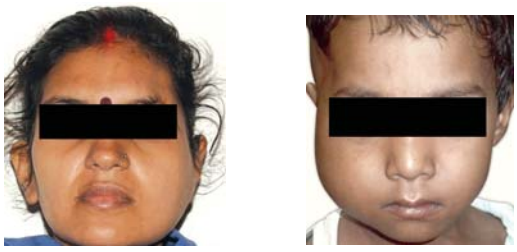
Sialoliths

Enlargements which are non-parotid origin

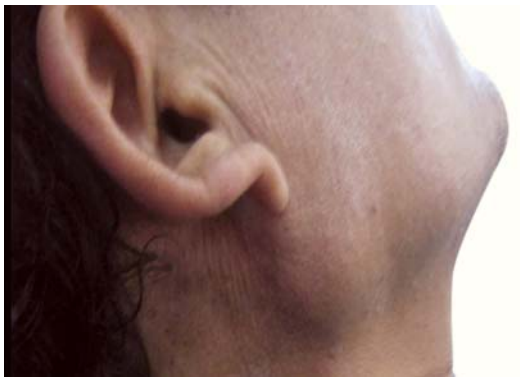
Preauricular lymph node swelling
- Idiopathic masseter muscle hypertrophy
- Sebaceous cyst
- Preauricular cyst or abscess.

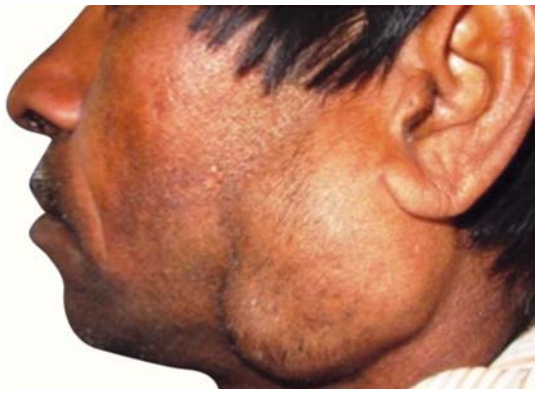
7. In which diseases the position of ear lobule affected?

The swellings with tale of parotid elevates the ear lobe and submasseteric space infections. The swelling obscures the visualization of ear lobe.



Submasseteric space infection hiding the visualization of ear lobe on left side and right side in child patient





Pleomorphic adenoma raising the ear lobule on right and left side respectively

8. What are differentiating points of masseteric hypertrophy and parotid swellings?

The bulk of parotid gland is anatomically located adjacent to upper two-thirds of ear, while masseter muscle bulk is positioned at lower level on lower half of mandibular ramus. The enlarged parotid will accentuate the ovalness of the face, while masseteric hypertrophy highlights facial rectangularity. The rectangularity due to masseteric hypertrophy is marked when it is present unilaterally, it alters the facial lines, generating discomfort and negative cosmetic impacts by many patients.

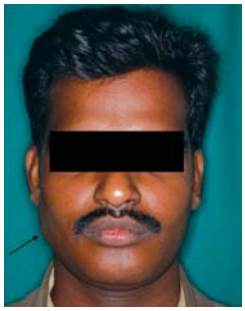
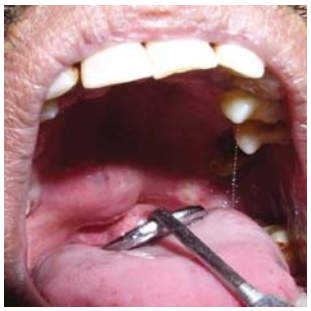
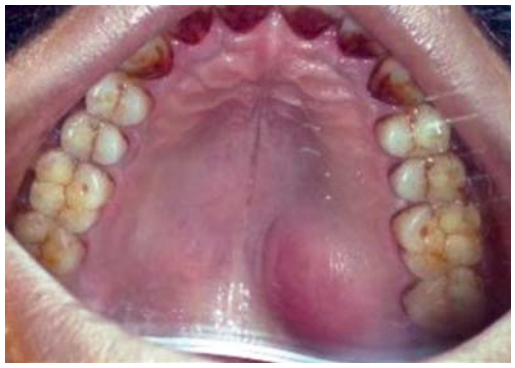
On palpation the masseteric swelling will increase in size when patient clenches the teeth.

In some cases, the area of insertion of the masseter on the mandibular ramus will respond by forming a bone projection that may

be palpable or seen on an anteroposterior radiograph. Presumably, this is due to the tension created by the masseter muscle during these contractions, which produce a periosteal reaction and activity.

9. What are common presentations of pleomorphic adenoma?

The two most common clinical presentations are a painless firm mass in the superficial lobe of the parotid gland and a painless firm mass in the posterior palatal mucosa.

Pleomorphic adenoma involving right side of superficial lobe of parotid gland and intraorally soft and hard palate respectively

10. What are the presentations of pleomorphic adenoma in parotid gland?

Eighty percent of all pleomorphic adenomas in the parotid gland develop in the superficial lobe. It presents as a freely movable, firm mass. Peculiarly and rarely, these can fluctuate in size or be painful. When a pleomorphic adenoma arises from the deep lobe of the parotid gland, it usually goes unrecognized for a number of years until its size creates symptoms of dysphagia or gagging. These present orally as a bulge arising from the tonsillar fossa area.

11. Which are most common tumor of minor and major salivary gland?

The pleomorphic adenoma is the most common tumor both in major and in minor salivary glands. Warthin tumors, basal cell adenomas, oncocytomas, acinic cell carcinomas, and sebaceous tumors have a strong predilection for the major glands, but the polymorphous low-grade adenocarcinoma has a marked predilection for the minor glands. While most intraoral salivary gland tumors favour the palate, the canalicular adenoma favours the upper lip. The three most common sites of minor gland tumors are palate, lip and buccal mucosa.

12. Which sites in oral mucosa where salivary gland tumors are less likely to occur?

Midline and anterior hard palate, gingiva, and attached alveolar mucosa.

13. Name the tumor of non-salivary gland origin and nonductal origin which occurs in the substance of salivary gland.

Tumors occurring within salivary glands are predominantly of epithelial origin. However, nonepithelial neoplasms also may arise within the gland that are not actually of salivary gland or ductal origin. Most of these are found in the parotid gland. Among the more common benign tumors is the hemangioma, which is the most frequently occurring tumor in the parotid gland in children. Lipomas and neurogenic tumors also may be seen. Hodgkin's and non-Hodgkin's lymphomas and, most infrequently, soft tissue sarcomas may develop. Metastatic tumors such as renal cell carcinoma and melanoma may also occur.

14. Enumerate lesions occurring at the junctions of soft and hard palate.

Mass lesions at the junction of the hard and soft palate mucosa are usually minor salivary gland tumors (adenoid cystic carcinoma, mucoepidermoid carcinoma, adenocarcinoma,

and polymorphous low-grade adenocarcinoma). Although other conditions and tumors such as neural tumors, non-Hodgkin's lymphoma, and palatal abscesses are possible, they may be diagnosed with the same biopsy principles.

Epstein pearls occur along median palatine raphe.

Petechiae in infectious mononucleosis are almost pathognomonic but can be seen in HIV and rubella. Oral petechiae and ecchymosis are seen in thrombocytopenia. Fellatio can result in palatal petechiae at the junction.

Necrotising sialometaplasia early in stage as dusky red swelling while afterwards as ulcer.

15. Enumerate the diseases occurring on the posterior part of palate?

Herpangina, intraoral mixed tumor, malignant salivary gland tumor, necrotising sialometaplasia.

16. What is superficial mucocele and deep mucocele?

A separate type of minor salivary gland mucocele, known as a superficial mucocele, is formed after a duct rupture in the subepithelial location. Preferred sites are the palate and retromolar areas. The mucoceles will appear as small (1 to 5 mm) yellow to grey vesicles. Superficial lesions take on bluish to transparent hue.

Deeper mucoceles, which often occur on the ventral surface of the tongue or the floor of the mouth unassociated with the sublingual gland, are frequently grey or yellowish in colour. These are common in children and young adults. Deep lesions are of normal colour.

17. What entities are considered in the differential diagnosis of mucocele?

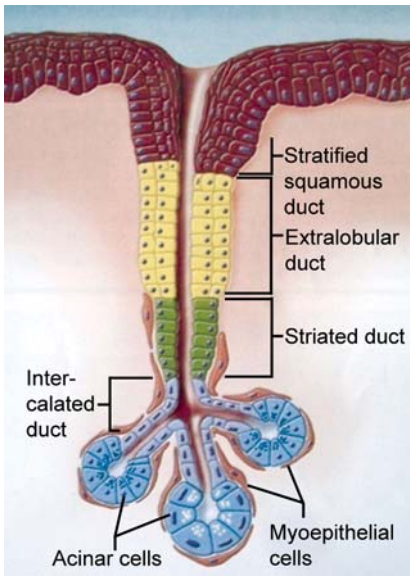
Mucoceles of minor salivary gland origin are bluish and the history of trauma may be vague, other lesions that may appear blue must be considered.

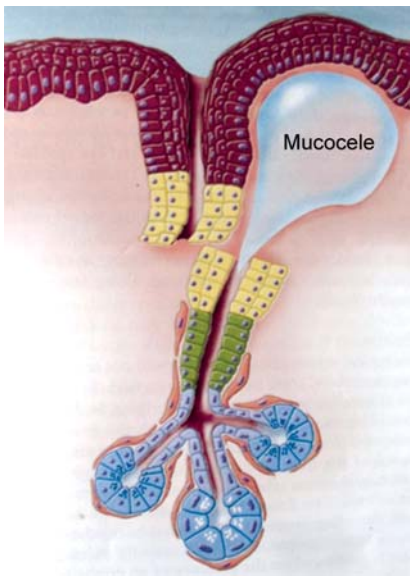
Low-grade mucoepidermoid carcinomas are the most significant. They will also produce mucin in vacuolated spaces within the tumor, imparting the blue colour.

Cavernous hemangiomas, small lymphangiomas, and a venous varix will give a similar appearance. Although rare, a vascular leiomyoma appears blue and has a predilection for the lip.

18. What is MEP and MRC?

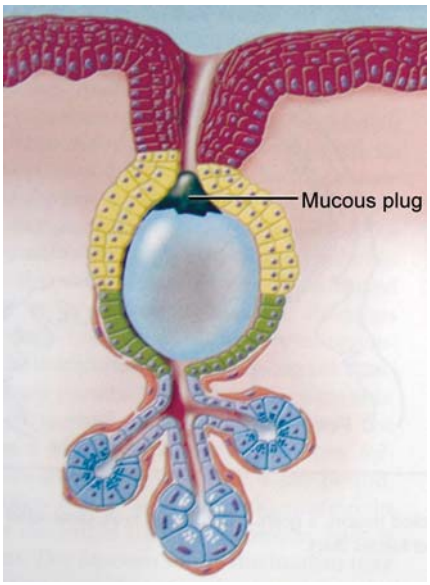
The word mucocele is a clinical term that applies to mucous extravasation phenomenon, that is, MEP. This occurs when saliva spills into the connective tissue and forms a mass, due to trauma to minor salivary gland ducts. These

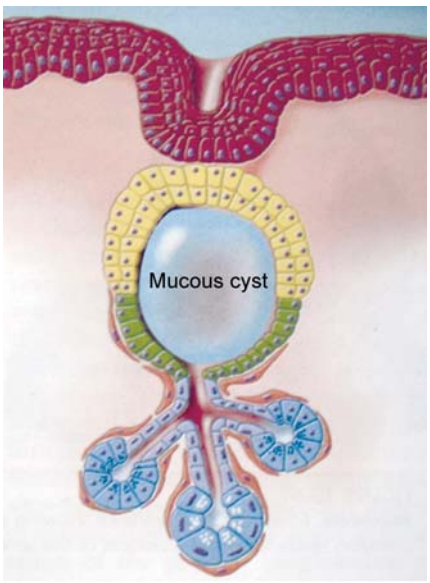




are common among children and adolescents. The common sites are lower lip, buccal mucosa, ventral tongue, floor of mouth and retromolar region. MEP appear as soft, bluish lesions with smooth surface.

A clinically similar lesion is the mucous retention cyst that is called MRC. This is also called mucous cyst. These are true salivary duct cysts and some may occur from retention of saliva in ductal tissues (by plunging from sialolith/mucous plugs) which cause dilation of ductal structure. These are more common in adults. The common sites are upper lip, buccal mucosa and floor of mouth. MRC are soft mobile masses similar to adjacent mucosal colour.





MRC is less common than MEP. Both are generally asymptomatic. Treatment for both is generally surgical excision.

19. What is sialocele?

Sialoceles are mucoceles of major salivary glands. These are tissue reaction to extravasation of saliva from a gland/duct complex and has a nonsecretary, nonepithelial lining consisting primarily of fibroblasts and capillaries.

Sialoceles of the submandibular gland and parotid gland are uncommon. When they do occur, they are more often related to penetrating injuries rather than to surgery. In either situation, the ductal injury is found in the main duct or in one of the large interlobar ducts.

Sialoceles of the parotid gland are more common than those of the submandibular gland. In the parotid gland, the extravasation of saliva forms a cutaneous collection, which thins the overlying skin, frequently to the point of rupture, or communicates to an area of penetrating injury to form a salivary cutaneous fistula. This can occur after parotidectomy.

Seroma is clinically similar to sialocele but differs in that it has lower amylase content on fluid analysis.

20. Enumerate conditions associated with drooling.

Drooling is an abnormal unintentional spillage of saliva from mouth onto the face on clothing. Primarily affecting patients with neuromuscular impairment and mental retardation. It is also called salivary incompetence. Drooling is not only unesthetic but can also affect speech, eating, lead to functional, social, psychological and clinical consequences for patients, family, and caregivers. It can cause skin irritation about the face and has been

reported to contribute to dehydration. The presence of significant drooling warrants a systematic approach to intervention. Conditions associated with drooling are as follows:

1. Physiological in healthy infants stops by about 18 months, abnormal if it continues beyond 4 years.
2. Infants (teething period, i.e. 4 days before a tooth emerges, day of emergence and 3 days after it)
3. Alzheimer's disease
4. Stroke
5. Pseudobulbar palsy or bulbar palsy
6. Cerebral palsy
7. Parkinsonism
8. Down's syndrome
9. Bell's palsy
10. Ataxia telangiectasia
11. Amyotropic lateral sclerosis
12. Drug induced (clozapine, nitrazepam)
13. Oral or dental deformities
14. Gastroesophageal reflux disease
15. Nasal obstruction
16. Painful swallowing (dysphagia)
17. Esophageal obstructions
18. Epilepsy

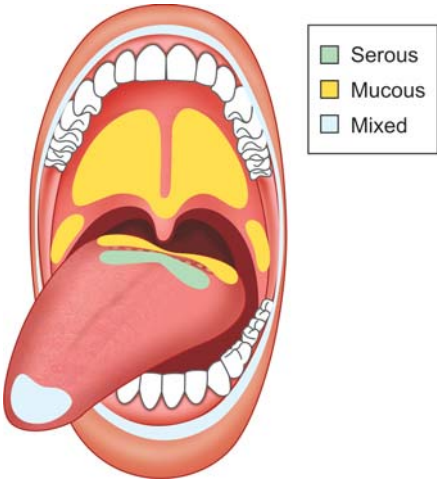
21. Enumerate the drugs causing salivary gland pain.

Antihypertensives, antithyroid agents, chlorhexidine, cytotoxics, ganglion blocking agents, iodides, phenothiazines and sulfonamides may cause pain in salivary glands.

22. How many numbers of minor salivary glands are present? What are their locations?

Minor salivary glands are 600–1000 in number and are of 1–5 mm in dimension. They have got single duct which directly secretes into oral cavity and produce half of saliva in oral cavity at rest.

1. **Labial and buccal glands:**
   - Mixed but predominantly mucous
   - Under epithelium of lips and cheeks
2. **Palatine glands:**
   - Purely mucous
   - Glandular region of hard and soft palate and uvula
   - Opening of ducts on palatal mucosa
3. **Glands of tongue:**
   - Anterior lingual glands: Mixed
   - Posterior lingual glands: Purely mucous
   - von Ebner's glands: Purely serous



23. What is the difference between hypersalivation and ptyalism?

Hypersalivation refers to excessive production of saliva while ptyalism includes both hypersalivation and drooling.

24. What are methods of saliva collection?

Draining, suction, spitting, and absorbent (sponge) method. The saliva can be collected under rest and in stimulated stage. Under stimulation collection includes intraoral duct cannulation and Lashley's cups and their modification as well as Schneyer's device.

25. Enumerate the conditions causing discoloration of saliva.

Discoloration of saliva and other body fluids into red or orange color may be seen in patients treated with clofazimine, levodopa, rifampicin, and rifabutin therapy.

26. What is salivaomics?

The term "salivaomics" was coined in 2008 to reflect the rapid development of knowledge about the various "omics" constituents of saliva ("salivary proteome", "transcriptome", "microRNA" (miRNA), "metabolome" and "microbiome").

27. What are the signs and symptoms of oral dryness?

The symptoms are related to decrease fluid in the oral cavity and have an effect on mucosal hydration and oral functions. Dryness of all mucosal surfaces including lips, throat. Difficulty in chewing, swallowing and speaking. Other associated complaints oral pain, oral burning sensation, chronic sore throat, pain in swallowing. Mucosa may be sensitive to spicy or coarse food. Dry lip is most common sign of oral dryness.

28. Which tests are done to indicate oral dryness?

The tests are lipstick test and tongue blade sign. Saliva would normally wet mucosa and aid in cleansing teeth. In lipstick sign the presence of lipstick or shed epithelial cells on labial surfaces of maxillary anterior teeth is an indication of reduced saliva while positive tongue blade sign indicates that the tongue blade when gently pressed against mucosa will adhere the tissue. This tongue blade sign is also called "mouth mirror sign".

29. Why the parotid calculi are radiolucent?

The so-called stones that form in the parotid duct system are rarely calcified and are actually mucous plugs that do not appear on radiographs. Stones that form in the submandibular duct system are almost always radiopaque because they are composed of calcium carbonate and calcium phosphate. Stones are believed to be more common in the submandibular duct system because of the more viscous glycoprotein consistency of the secretions and the upward course of the duct, which makes it more prone to stasis and retrograde bacterial invasion. The parotid gland secretion is much less viscous because of its higher protein levels and because the course of Stensen's duct is downward and low mineral content. There is no association in either gland with altered serum calcium levels or renal stones 70% of parotid calculi are radiolucent and are observed and filling defect on sialogram or can be directly visualized on sialoendoscope.

30. What is sialodochoplasty?

It is a procedure by which the stones that are accessible in the floor of the mouth are removed via a direct approach. The damaged duct is sutured to the mucosa of the floor of the mouth. With this approach there is always concern about the course of the lingual nerve in the floor of the mouth.

31. Define sialography.

A radiographic procedure used to study the salivary ducts and glands with the aids of contrast media or radiographic study of salivary glands and ducts after injection of contrast material.

32. What are the indications of sialography?

Calculi, strictures, sialectasia (dilatation), fistulae and other pathology.

33. What are the contraindications of sialography?

Allergy to compounds containing iodine:

- Periods of acute infection/inflammation, when there is discharge of pus from the duct opening.
- When clinical examination or routine radiographs have shown a calculus close to the duct opening, as injection of the contrast medium may push the calculus back down the main duct where it may be inaccessible.

34. What are the complications of sialography?

Injection of contrast medium can put the sialolith further back, extravasation of medium in substance of gland, edematous ductal orifices and damage to orifice (specially by conventional sialography), granuloma formation, rupture of salivary gland duct or

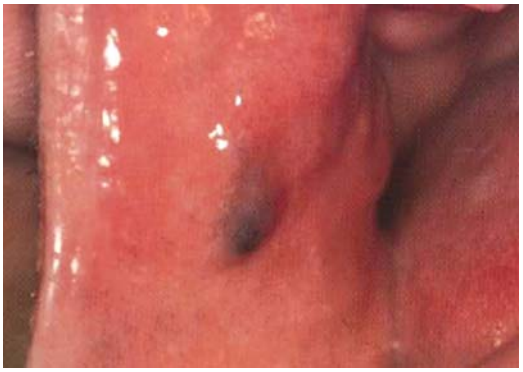
perforation of duct which is more common in parotid gland due to its curvature, overfilling of duct, exacerbation of infection, adverse reaction to contrast material and overfilling may result in misdiagnosis of sialectasis.

35. What are the types of intraoral nevus?

Nevi are developmental malformations originating from defective melanoblasts of neural crests. Usually, seen in skin and rarely in oral mucosa. They may be congenital/acquired. The following are the categories of acquired nevi.

Intramucosal Nevus

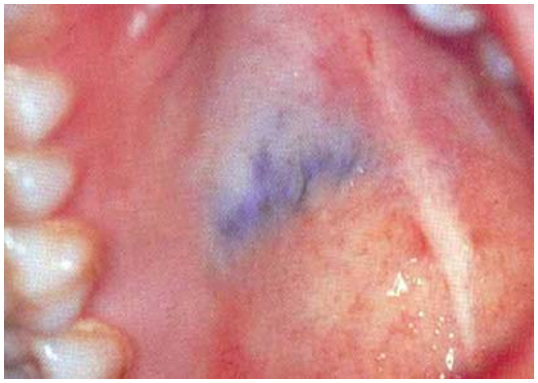
It is common representing 55%. The cells are in connective tissue and are separated from epithelium by a band of collagen. More in females and in any age. Asymptomatic, flat, plaque like with brown or brownish black color. Common location is palate and buccal mucosa rarely seen on gingival and lips. Little potential for malignancy.



Intramucosal nevus on buccal mucosa

Blue Nevus

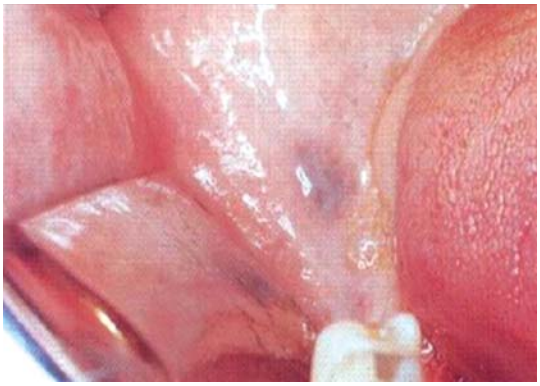
It is the second most frequent nevus of oral mucosa. This accounts for 30–36% of oral nevi. The melanocytes are elongated and slender containing melanin pigment and arranged in pattern parallel to epithelium in middle and lower parts of lamina propria. No junctional activity. There are two types: One is involving oral mucosa and skin while other is specific for skin. Seen at any age, no sex predilection. Clinically, asymptomatic slightly elevated or flat spot or plaque with oval or irregular shape, brown or blue in colour. Common on hard palate. No malignant transformation potential.



Blue nevus on palate

Junctional Nevus

It is less common of oral nevi (3–5%). Histologically shows nests of nevus cells along the basal layer of epithelium and some drop into connective tissue showing junctional activity. Clinically these are seen as black or brown, flat or slightly raised spots. Common location on palate, buccal mucosa and alveolar mucosa. They never seem to convert to a compound nevus. Transformation into a melanoma may occur.

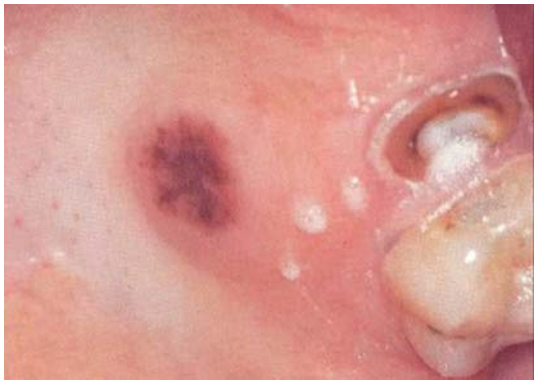


Junctional nevus on retromolar area

Compound Nevus

The nevus cells are present in epithelium and underlying connective tissue. They present characteristic of intramucosal and junctional nevus. It represents about 6–9% of all oral nevi.

Clinically, asymptomatic, slightly elevated flat spot with red brown or black brown color. The size may be up to 1 cm. Common intraoral sites are buccal mucosa, palate and gingival. It may transform into malignant melanoma.



Compound nevus on palate

36. Enumerate the various pigments and the color given by them in the oral cavity.

Hemoglobin, hemosiderin and melanin are the pigments associated with color in oral cavity.

Hemoglobin imparts red/blue color and indicates lesions with vascular disease.

Hemosiderin appears brown, as a result of extravasation of blood and defect in hemostatic mechanisms.

Melanin imparts black pigmentation and indicates increased sun exposure, certain drugs and genetic factors.

37. Enumerate the oral mucosal lesions that may show blue counterpart.

1. Petechiae and purpura—they are red but turn bluish macules.
2. Congenital hemangioma, cavernous hemangioma, congenital A-V malformations, varicosities and lymphangioma. Lymphangioma is less blue than hemangioma.
3. Peripheral giant cell granuloma, eruption cyst, eruption hematoma, superficial ranula.
4. Oral nevus and mucocele.
5. Chronic poisoning with heavy metals (such as mercury, lead, bismuth, and silver), i.e. heavy metal lines may result in a bluish–black discolouration in the oral cavity. Frequently the heavy metals are deposited as a line or band on inflamed marginal gingiva.
6. Prodromal signs of infectious mononucleosis may occur a few days before the patient becomes ill as 6 to 20 petechiae in the soft palate. The hematoma is a pool of effused blood confined within the tissues. When it is superficial, it appears as an elevated, bluish swelling in the mucosa.
7. Addison's disease, hemochromatosis, argyria, caviar tongue, oral nevus, cyanosis, chloasma gravidarum.
8. In rarities aniline intoxication, arsenic poisoning, carotenemia, chloroquine therapy.

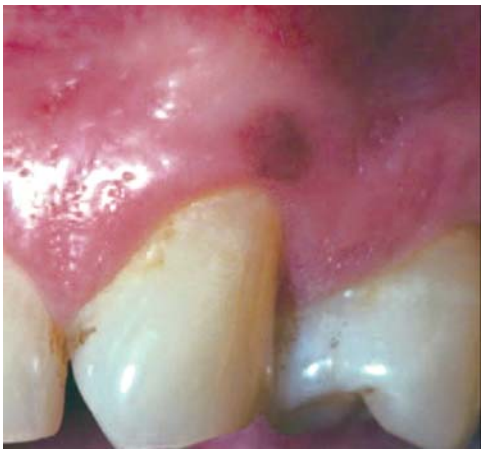
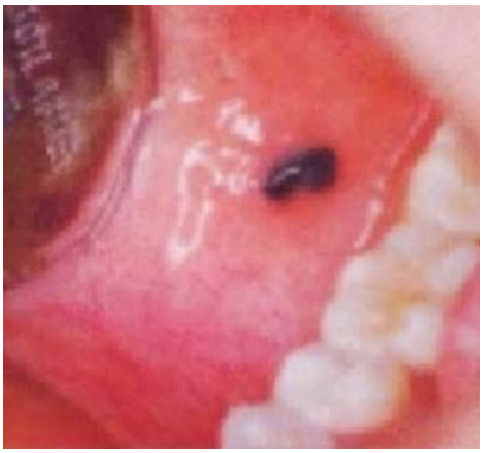
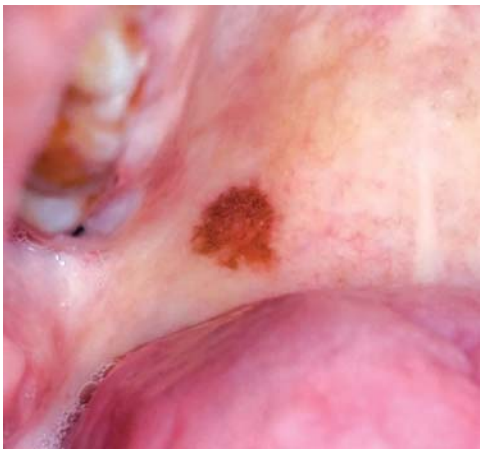
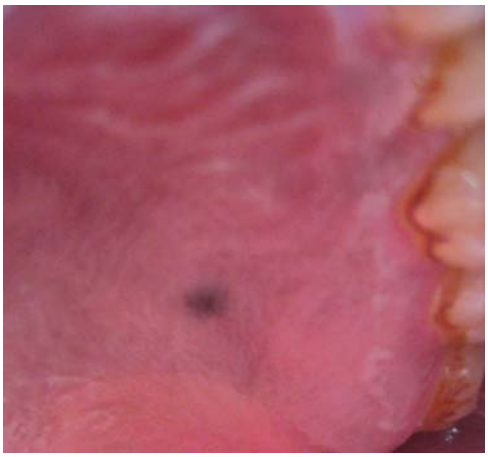
38. Enumerate melanocytic lesions.

Melanocytic lesions appear brown or black due to the deposition of melanin.

Ephelis: It is a freckle. It is flat, brown or black in colour, and occurs on sunexposed surfaces. It is due to increased production of melanin by melanocytes. An ephelis requires no treatment.

Oral melanotic macule: It is a localized pigmented lesion associated with increased melanin pigmentation of the stratified squamous epithelium. It is asymptomatic, flat and not thickened, and appears similar to an ephelis (freckle) of skin. It is a harmless lesion, but its significance lies in distinguishing it from nevus or early melanoma. A biopsy should be performed if any doubt exists about the diagnosis. If an oral pigmented lesion is not thickened, but is larger in diameter, has any variation in colour, cannot be diagnosed as tattoo based on radiographic findings, or has irregular borders it should be excised.

Melanocytic nevus: It is a benign proliferation of nevus cells (melanocytes). Nevi of skin first appear in childhood and progress through a series of clinical and microscopic stages. Most people have between 10 and 40 nevi on their skin. Nevi of skin that have uniform colour and borders and are not changing in size or surface texture, are not considered premalignant lesions and do not need to be removed unless

they are chronically irritated or having a cosmetic problem. Nevi of oral mucosa are relatively rare. They occur most commonly on the gingiva and hard palate. Nevi of oral mucosa should be completely excised because they cannot be differentiated from melanoma based on their clinical features.

Melanoma: It is a malignant neoplasm of melanocytes. Melanoma of skin has increased significantly in incidence, while melanoma of oral mucosa is relatively rare. The most important clinical features of cutaneous melanoma are asymmetry of the lesion, variation in colour (brown, black, red, white, blue), and diameter greater than 6 mm. Oral melanoma begins as an irregular, brown to black macule. Later the lesion will develop thickening and sometimes ulceration. The most common locations are the hard palate, gingiva, and alveolar ridge. It is not possible to distinguish an oral melanocytic nevus from early melanoma. If oral nevus and/or melanoma are included in the clinical differential diagnosis, then a biopsy is indicated. Biopsy is also indicated for flat, non-thickened pigmentations that are changing or have atypical colour, borders, or size. Treatment for melanoma is complete surgical excision. The thickness of the lesion and depth of invasion are the most important prognostic factors.

39. What is melanoplakia?

These are racial pigmentation common oral mucosal discoloration usually and exclusively in non-caucasian population. This is incidental finding, commonly present as symmetrical, band like zone of brown–black discoloration

at the junction of attached gingival and alveolar mucosa. Buccal mucosa, tongue, hard palate, and lips are also involved. Recognition and monitoring is recommended. Melanoplakia may be present at birth and persists for life.

40. What is ABCDE of melanoma?

The clinical evaluation system is developed.

a. Asymmetry because it has uncontrolled growth pattern.
b. Border irregularity with notching.
c. Colour variation, it shows shades of brown to black, white, red, and blue. These shades vary depending on amount and depth of melanin pigmentation.
d. Diameter greater than 6 mm (i.e. diameter of a pencil eraser).
e. Evolving as lesions show changes with respect to size, shape, color, surface or symptoms over time.

41. What are presentations of oral melanoma intraorally?

The oral melanoma may present as one of four lesions: A pigmented macule (various shapes, possibly linear); a pigmented nodule; a large, pigmented exophytic lesion perhaps associated with macular pigmentation; or an amelanotic (nonpigmented) variety of any of these three forms.

42. What are coast of California and coast of Maine?

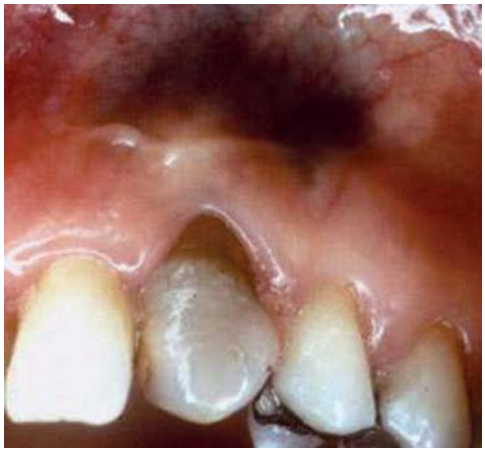
These are patterns of café au lait spots.

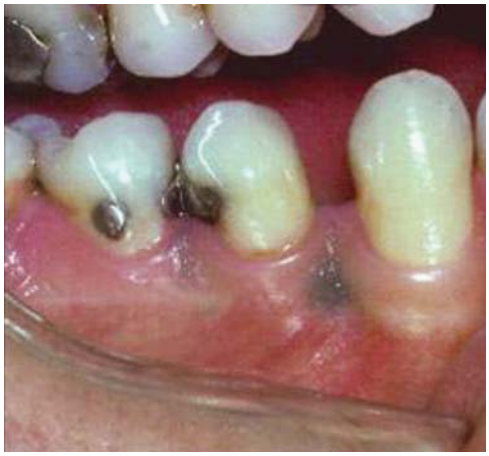
Coast of California: Café au lait macules of NF-I have smooth edges. These macules are small.

Coast of Maine: Café au lait macules of fibrous dysplasia syndromes (McCune Albright's syndrome) are craggy and irregular. These are larger.

43. What is focal argyrosis?

It is an iatrogenic lesion that follows traumatic soft tissue implantation of amalgam particles or a passive transfer by chronic friction of mucosa against amalgam restoration. The lesions are macular and grey seen in gingival and buccal mucosa. These are called amalgam tattoo.





44. Enumerate endocrine abnormalities associated with oral pigmentation.

The abnormalities are Albright's syndrome, Addison's disease, acromegaly, Cushing's disease and hyperthyroidism.

Albright's syndrome: Polyostotic fibrous dysplasia, abnormal pigmentation, precocious puberty.

Addison's disease: Diffuse brownish or black discoloration in any part of oral mucosa commonly buccal mucosa, lips, tongue, gingival. It is due to adrenocortical deficiency.

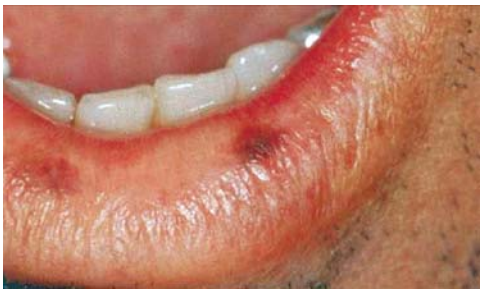
Acromegaly: Hyperpigmentation is similar to Addison's disease and seen in 40% patients. It is usually due to pituitary tumor.

Cushing's disease: It is from excessive ACTH production from pituitary tumor.

Hyperthyroidism: It is due to increased MSH production. Pigmentation is same as Addison's disease.

45. What are freckles (ephelis)?

These are common in Caucasians and Chinese population. An ephelis is a common small hyperpigmented macule of the skin that represents a region of increased melanin production. Ephelides are seen most often on the face, arms, and back of fair-skinned, blue-eyed persons, they may be associated with a strong genetic predilection (autosomal-dominant). The skin discoloration is produced by a relative excess of melanin deposition in the epidermis, not by a local increase in the number of melanocytes.



Freckle on lower vermillion border

No treatment is necessary for ephelides. The use of sunscreen scan prevent the appearance of new freckles and help prevent the darkening of existing lesions.

Chapter

7

Developmental Disturbances, Physical and Chemical Injuries to the Oral Cavity

1. Enumerate the exophytic anatomic structure.

- Accessory tonsillar tissue
- Buccal fat pads
- Circumvallate papillae
- Foliate papillae
- Genial tubercles
- Lingual tonsillar tissue
- Palatine rugae
- Palatine tonsils
- Palatine papillae
- Retrocuspid papillae
- Retromolar papillae
- Stensen's papillae
- Sublingual caruncles
- Tongue
- Uvula

2. Enumerate yellow conditions of oral mucosa.

Fordyce's granules	Fibrin clot
Superficial abscess	Superficial nodules of tonsillar tissue
Yellow hairy tongue	Acute lymphonodular pharyngitis
Lipoma	Lymphoepithelial cyst
Epidermoid and dermoid cysts	Pyostomatitis vegetans
Jaundice or icterus carotenemia	Lipoid proteinosis

Rarities

Amyloidosis
Lesions of sebaceous glands pseudoxanthoma clasticum
Psoriasis

Many potentially yellow lesions may not appear yellow because the covering mucosa masks their color. Normal fat covered by a thin layer of mucosa appears yellow. Salivary gland tissue infiltrated with fat appears yellow. This condition occurs most often in the soft palate. Bone and salivary stones covered by a thin mucosa may impart a yellowish tinge to the mucosa.

3. What is caviar tongue?

A varicosity, a distended vein, is a common occurrence in the oral cavity, especially in older individuals. It may also result from partial blockage of the vein proximal to the distension. The varicosities most frequently observed by the clinicians are superficial, painless, and

bluish; they appear somewhat congested and accentuate the shape and distribution of the vessel. The most frequent site is the ventral surface of the tongue.

When many of the sublingual veins are involved, this condition is called caviar tongue (phlebectasia linguae).

4. What is ankyloglossia? What is partial ankyloglossia? What is classification?

Ankyloglossia (more commonly called "tongue-tie") is a congenital anomaly characterized by an abnormally short lingual frenum, which may restrict tongue tip mobility. It is defined as a limitation of the possibilities of protrusion and elevation of the tip of the tongue due to either the shortness of the frenulum or the genioglossus muscle or both. The problems associated with it are trouble in breastfeeding, dental caries malocclusion, gingival recession, and restricted alveolar bone growth in children. Mostly tongue tie presents as partial ankyloglossia from tongue tip to base of tongue.

Clinically acceptable, normal range of free tongue → 16 mm

Class I: Mild ankyloglossia = 12–16 mm

Class II: Moderate ankyloglossia = 8–11 mm

Class III: Severe ankyloglossia = 3–7 mm

Class IV: Complete ankyloglossia = <3 mm

Class I and II cases are self-correcting, thus wait and watch along with speech therapy. Class III and IV surgical therapies are desirable.

5. What is commissural oral pit and what is its significance?

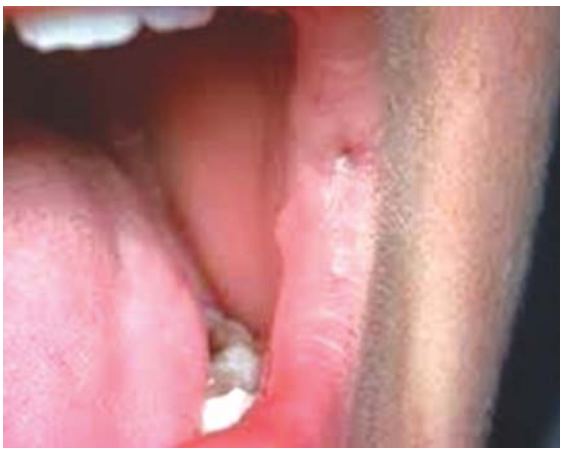
The commissural lip pit is a relatively common developmental disorder, although there is disagreement concerning its incidence. The commissural lip pit may be bilateral or unilateral. Unilateral pits occur as often on the right as on the left side of the mouth. The pits are located at the angles of the mouth, with the tracts diverging dorsolaterally into the cheek. They range in size from a shallow dimple to a tract measuring 4 mm in length, and the tissue is slightly raised around the opening. On microscopic examination the tract is lined with stratified squamous epithelium that continues into the vermilion tissue of the lip. Mucous gland ducts may empty into the sinus; as a result, mucous frequently can be milked from the tract.

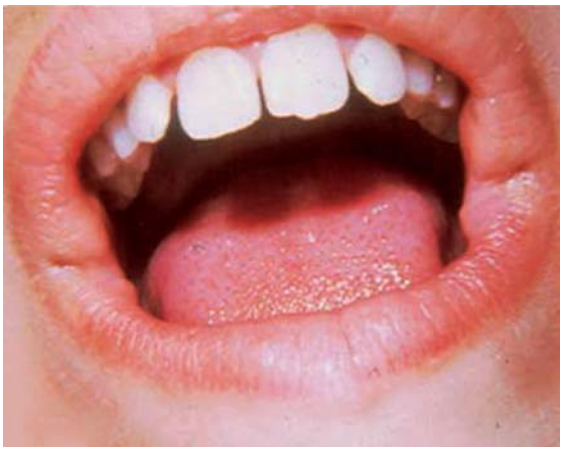
Differential Diagnosis

The commissural lip pit especially must be differentiated from the congenital lip pit, which is seen on the vermilion border of the lower lip but not at the commissures. The congenital lip pit, however, is extremely rare, occurring in approximately 1 of 2 billion births.

Management

The commissural lip pit is asymptomatic and requires no treatment.





6. What is caliber-persistent artery?

A caliber-persistent artery is a common vascular anomaly in which a main arterial branch extends up to the superficial submucosal tissues without a reduction in its diameter. Similar to oral varices, caliber-persistent arteries are seen more frequently in older adults. This suggests that their development may be an age-related degenerative phenomenon in which there is a loss of tone in the surrounding supporting connective tissue.

The caliber-persistent artery occurs almost exclusively on the lip mucosa. Either lip may be affected, or some patients have bilateral lesions or lesions on both lips. The average patient age is 58 years and the gender ratio is nearly equal. The lesion presents as a linear, arcuate or papular elevation that ranges from pale to normal to bluish in colour. Stretching the lip usually causes the artery to become inconspicuous. The unique feature is pulsation—not only vertically but also in a lateral direction. However, usually it is not possible to feel a pulse in a caliber-persistent artery with gloved fingers. The lesion is usually asymptomatic being discovered as an incidental finding during an oral examination, rarely a patient may notice a pulsatile lip nodule. A few cases have been associated with ulceration of the overlying mucosa. In addition, a couple of examples have been found adjacent to labial squamous cell carcinomas, although this is probably coincidental.

The ulcer is attributed to continual pulsation from the large artery running parallel to the surface. Ligation of artery appears successful.

7. What are ectopic lymphoid tissue?

Lymphoid tissue appears yellow or yellow-white with dome-shaped elevations, asymptomatic. Ectopic locations are posterolateral aspect of tongue (lingual tonsil), soft palate, floor of mouth and tonsillar pillars.

8. What is Ascher's syndrome?

Ascher syndrome is characterized by a triad at features:

- Double lip
- Blepharochalasis
- Nontoxic thyroid enlargement in a person with blepharochalasis, recurring edema of the upper eyelid leads to sagging of the lid at the outer canthus of the eye.

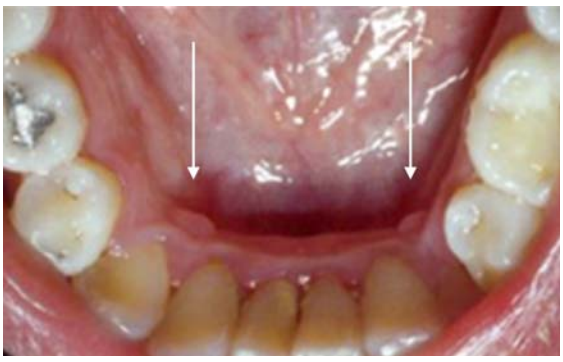
This drooping may be severe enough to interfere with vision. Both the double lip and blepharochalasis usually occur abruptly and simultaneously, but in some cases they develop more gradually. The nontoxic thyroid enlargement occurs in as many as 50% of patients with Ascher's syndrome and may be mild in degree. The cause of Ascher's syndrome is not certain autosomal dominant inheritance has been suggested in some cases.

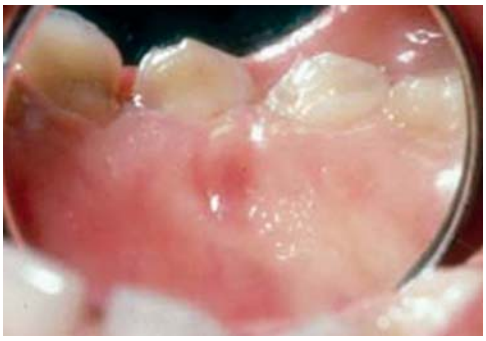
9. In which conditions double chin appearance seen?

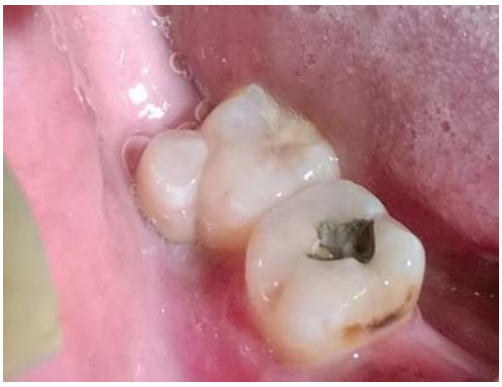
Genetic predisposition for same, submental space infection often produces a swelling below chin giving double chin appearance. Inframyloid variety of dermoid cyst, achondrogenesis, adipose tissue distribution with familial partial lipodystrophy, in the treatment of Cl III in surgical orthodontics setting back of chin often leads to drooping of soft tissues and with hyperactivity of mentalis muscle leads to double chin appearance.

10. What is retrocuspid papilla?

It is a fibroepithelial pink papulae (less than 5 mm in diameter) on attached gingival usually bilaterally lingual to mandibular canines. The prevalence in 25% to 99% in children and young adults and drops to 6% to 19% in older adults suggesting that it is a normal anatomic variation disappears with age.



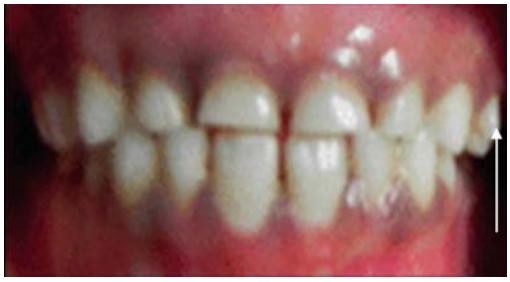




11. What are clinical presentations of fissured tongue?

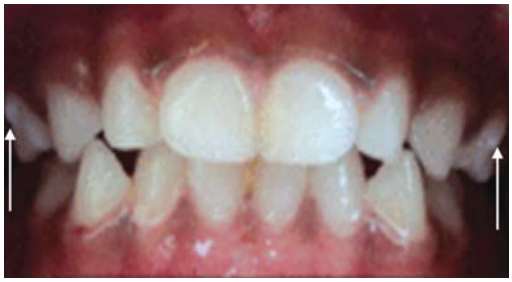
Three presentations are commonly seen:

1. A prominent median groove as a sole manifestation
2. A prominent median groove with accessory grooves radiating laterally at right angles to the median groove
3. Multiple grooves arranged in irregular circinate pattern



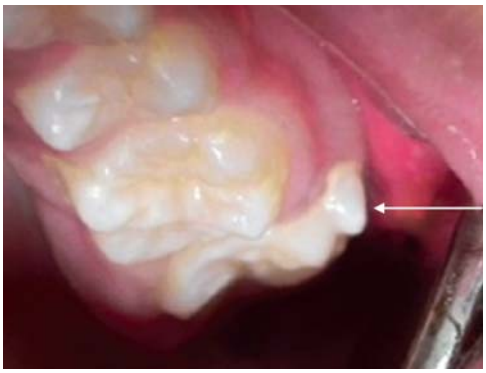
12. What is Cupid's bow lip?

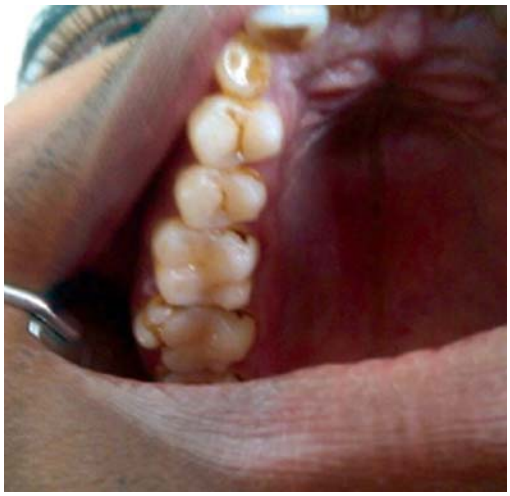
Developmental disorder of lip exhibiting double lip appearance. The lip is divided into two by horizontal fissure. However, it is not prominent when lip is in relaxed position. Involved upper lip is tensed, the double lip resembles "Cupid's bow''.



13. What is protostylid?

A protostylid is a supernumerary cusp located on the mesial half of the buccal surface on the upper and lower molars. These may be seen on primary molars and permanent molars. They are more common on primary molars.





14. What are nonmetric dental traits?

Cusp of Carabelli, protostylid, central accessory cusp, variations in the cusp number and number of roots are all termed "nonmetric dental traits". The nonmetric features are recorded visually in terms of presence, absence, degree of development or expression of these features.

15. What are different variations in cuspal morphology of teeth?

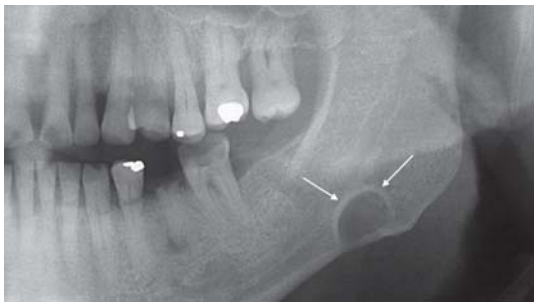
1. **Cusp of Carabelli:** It is an accessory cusp located on the palatal surface of the mesiopalatal cusp of maxillary molar. More prominent on first molar and then less prominent on second and third molars. It is seen in permanent and deciduous dentition. The form may vary from a definite cusp to small intended pit and fissure. Protostylid is analogous accessory cusp on mandibular deciduous or permanent molars.
2. **Talon's cusp:** It is a well-delineated additional cusp that is located on the surface of an anterior tooth and extends at least half the distance from cementoenamel junction to incisal edge. It is common on lingual surfaces of lateral incisors and central incisors. Rarely facial talons are also seen. These may seen in association with Rubinstein-Taybi syndrome, Mohr syndrome, Ellis-van Creveld syndrome, and even in Sturge-Weber's syndrome.
3. **Dens evaginatus:** It is a cusp like elevation of enamel located on the central groove or lingual ridge of the buccal cusp of premolar or molar teeth. It is also called central tubercle, occlusal pearl, Leong's premolar (tuberculated premolar) frequently, it is seen in association with shovel-shaped incisors, affected incisors demonstrate prominent lateral margins, creating a hollowed lingual surface that resembles scoop of shovel. Typically the thickened marginal ridge converge at cingulum. Maxillary lateral and central incisors most commonly affected.

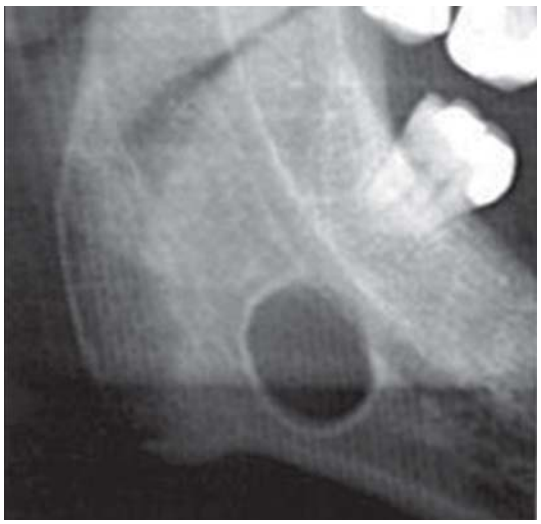
16. What is Stafne's bone cyst? What are the other names of it?

Stafne's bone defects are usually presented as asymptomatic radiolucency below mandibular canal between molar teeth and angle of mandible. These defects are static in nature containing ectopic salivary tissue. These are incidentally found on radiographs. These may be unilateral occasionally bilateral, sometimes in anterior mandible and even in ramus containing portion of parotid. The two proposed mechanisms are:

1. Failure of normal bone formerly occupied by cartilage
2. Localised pressure atrophy of adjacent surface of salivary gland.

The other names are Stafne's bone defects, Stafne's bone cavities, static bone cyst, pseudocyst, lingual mandibular defect, and lingual salivary gland depression.





17. What are different types of amelogenesis imperfecta?

It is the term applied to number of rare genetically disorders of enamel formation which affect both primary and permanent dentition, without any systemic disturbances. There are three major types: Hypoplastic, hypocalcified, hypomaturation. However, Witkop described fourth type as amelogenesis imperfecta with taurodontism.

Hypoplastic

This is due to defects in the secretary process of ameloblasts resulting in thin or pitted enamel. Thin enamel of normal calcification. It varies from localized pitting of enamel to a general diminution of enamel formation with lack of interproximal contacts.

Hypocalcified Type

This is due to inability of crystallites to nucleate properly causing abnormal crystallite growth and decreased mineral enamel content. Enamel is of normal thickness but with low radiodensity, low mineral content, and is therefore soft and quickly lost exposing the dentine. It shows great variability and enamel of cervical portion of teeth may be highly mineralized.

Hypomaturation Type

Normal amounts of enamel matrix that is deficiently mineralized. Enamel is as radiodense as dentine and has a characteristic mottled brown, yellow-white appearance, and easily chips away from dentine. Snow capped teeth are variations which show varying proportions of incisal and occlusal aspects of crowns, have an opaque white appearance.

18. What is Dean's fluorosis index?

Dean's fluorosis index modified criteria (1942): Classification criteria

Procedure

To obtain Dean's fluorosis index, the examiner's recording is based on the two most affected teeth. However, if the two teeth are not equally affected, the score for the less affected tooth is recorded. When teeth are scored; the examiner should start at the higher end of the index, and eliminate each score or category until he or she arrives at the present condition. If there is any doubt, the lower score should be recorded.

Normal (0): The enamel represents the usual translucent semivitriform type of structure. The surface is smooth, glossy, and usually of a pale, creamy white colour.

Questionable (0.5): The enamel discloses slight aberrations from the translucency of normal enamel, ranging from a few white specks to occasional white spots. This classification is used in those instances where a definite diagnosis of the mildest form of fluorosis is not warranted and a classification of 'normal' not justified.

Very mild (1.0): Small, opaque, paper white areas scattered irregularly over the tooth, but not involving as much as approximately 25% of tooth surface. Frequently included in this classification are teeth, showing not more than or about 1–2 mm of white opacity at the tip of the summit of the cusps of bicuspid or second molars.

Mild (2.0): The white opaque areas in the enamel of teeth are more extensive, but do not involve as much as 50% of tooth.

Moderate (3.0): All enamel surfaces of the teeth are affected and subject to attrition show wear. Brown stain is frequently a disfiguring feature.

Severe (4.0): All enamel surfaces of the teeth are affected and hypoplasia is so marked that the general form of the tooth may be affected. The major diagnostic sign of this classification is discrete or confluent pitting. Brown stains are widespread and teeth often present a corroded-like appearance.

19. What are hypodontia and oligodontia?

Hypodontia—a commonly used term to describe the absence of one to six teeth and is

most common dental developmental anomalies in human. The absence of more than six teeth excluding third molars, is called 'oligodontia'. It is also called 'severe hypodontia'. Oligodontia is usually a part of syndrome, and seldom seen as an isolated entity. These are classified as isolated (non-syndromic) or syndromic hypodontia or oligodontia.

20. What are the other symptoms associated with hypodontia and oligodontia?

Dental malposition, periodontal damage, lack of alveolar bone height, esthetic and functional impairment are well-known symptoms.

21. What are the syndromes associated with hypodontia and oligodontia?

Ectodermal dysplasia, Down's syndrome, Ellis-van Crevald syndrome, cleft lip and palate, hemifacial microsomia, Van der Woude syndrome.

22. When tooth is called congenitally missing tooth?

Congenitally missing teeth are defined as those teeth that fail to erupt in oral cavity and remain invisible in the radiograph, there is no previous history of extraction or trauma which implies that this is caused by disturbances during the early stages of tooth development.

23. What are ghost teeth?

This is an unusual, uncommon, nonhereditary, developmental dental anomaly in which one or several teeth in a localized area are affected in an unusual manner (i.e. extensive adverse effects on formation of enamel, dentin and pulp). Apparently the maxillary teeth are involved more frequently than the mandibular, the most frequently affected teeth being the maxillary permanent central incisor, lateral incisor, and cuspid. In the mandible, the same three anterior teeth are most often affected. Ipsilateral involvement of both arches and bilateral changes in same jaw have been reported.

Clinical Features

The ghost teeth (regional odontodysplasia) exhibit either a delay or a total failure in eruption, malformed teeth, early exfoliation and noninflammatory gingival enlargement. This is seen in both dentition with little female predominance. Their shape is markedly altered, being generally very irregular in appearance, often with evidence of defective mineralization. Pulpal necrosis is common due to long pulpal horns and clefts in dentin.

Radiographic Features

The radiographs are uniquely characteristic, showing a marked reduction in radiodensity so that the teeth assume a 'ghost' appearance. Both the enamel and dentin appear very thin and the pulp chamber is exceedingly large. The enamel and dentin layer often is not evident. Short roots and open apices are seen.

24. What are shell teeth?

Dentinogenesis imperfecta type II is called shell teeth. Shell teeth demonstrate normal thickness enamel in association with extremely thin dentin and dramatically enlarged pulps. The thin dentin may involve the entire tooth or be isolated to the root. This rare abnormality has been seen most frequently in deciduous teeth in the presence of dentinogenesis imperfecta. The alteration may be unassociated with dentinogenesis imperfecta as an isolated finding in both dentitions and demonstrate normal tooth shape, coloration, a negative family history, and diffuse involvement. In the isolated variant slow but progressive root resorption occurs.

25. What are the other names of dentinogenesis imperfecta?

Hereditary opalescent dentin, Capdepont's teeth.

26. Enumerate the conditions in which enlarged pulp chambers are seen.

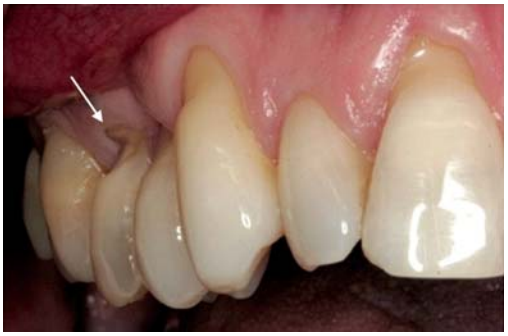
Regional odontodysplasia, dentinogenesis imperfecta, taurodontism and trichodento-osseous syndrome.

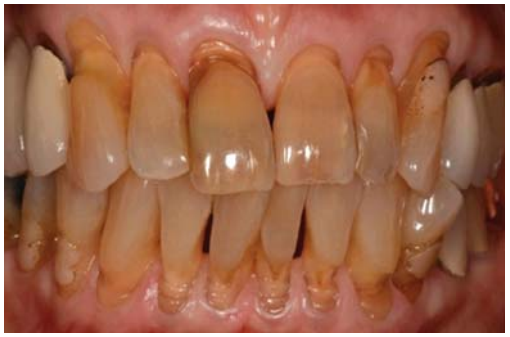
27. What is abfraction?

The term abfraction derived from the Latin verb *frangere. Fregi, fractum* (*to break*) is used

to describe a special form of wedge-shaped defect at the cementoenamel junction (CEJ) of a tooth (2). Such lesions observed on a single tooth or on non-adjacent teeth are hypothesized to be the result of eccentrically applied occlusal forces, leading to tooth flexure rather than to be the result of abrasion alone abfraction, appears as wedge-shaped defects limited to the cervical area of the teeth, and may closely resemble cervical abrasion or erosion. Clues to the diagnosis include defects that are deep, narrow, and V-shaped (which do not allow the toothbrush to contact the base of the defect), and often affect a single tooth with adjacent unaffected teeth.

In addition, occasional lesions are subgingival, a site typically protected from abrasion and erosion. The lesions are seen almost exclusively on the facial surface and exhibit a much greater prevalence in those with bruxism. A higher frequency is noted in the mandibular dentition, presumably because the lingual orientation makes them more susceptible to the concentration of tensile stresses at the cervical regions.



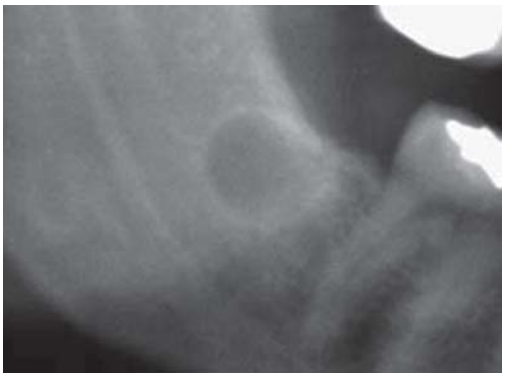


28. What is meth mouth?

Methamphetamine is central nervous system stimulant. A very common and visible sign of long-term meth use is extreme tooth decay also known as meth mouth. Users with **meth mouths** have often blackened, attrition, cracks, erosion, stained and rotting teeth that often cannot be saved. Meth damages blood vessels and decreases blood flow to all parts of body. Meth users have xerostomia or meth mouth.

29. What is myospherulosis?

It is also called **paraffinoma.** It is a unique foreign body reaction which results from placement of topical antibiotic in a petroleum base in surgical site. It is an inflammatory granulomatous lesion resulting from the action of lips substances on extravasted arythrocytes. It may occur in soft tissue or bone and paranasal sinuses. The area is asymptomatic and may show swelling and radiographically circumscribed radiolucency in extraction site. It is treated by surgical removal of foreign material and affected tissue.



30. Enumerate the conditions causing palatal perforations.

Palatal perforation can be defined as a communication between the nasal cavities and the oral cavity.

1. **Developmental conditions:** During the sixth week of prenatal period, palatal shelve coalescence to form the hard palate. Failure to this integration results in cleft palate. Some syndromes, maternal alcohol

consumption and cigarette smoking, folic acid deficiency, corticosteroid use and anticonvulsant therapy are some causative agents for this abnormality.

2. **Infectious:** There are some infections resulting in palatal perforation. In leprosy, tertiary syphilis, tuberculosis, rhinoscleroderma, naso-oral blastomycosis, leishmaniasis, actinomycosis, histoplasmosis, coccidiomycosis and diphtheria. The palatal roof may be perforated.
3. **Autoimmune:** There are some autoimmune diseases which results in palatal perforation. Examples are lupus erythematous, sarcoidosis, Crohn's disease and Wegener's granulomatosis.
4. **Neoplastic:** Many tumors can extend from maxillary sinus or nasal cavity and perforate the palate. Although these neoplasms usually form a mass, but in advance cases perforation of palate may occur in course of disease or even following treatment. Minor salivary gland malignancy adenoid cystic carcinoma is reported to cause palatal perforation. Other malignancies like nasal T-cell lymphomas, carcinoma, melanoma, etc.
5. **Drug related:** Palatal perforation due to cocaine abuse is a well-known situation. Other drugs (heroine, narcotics) can be responsible for palatal perforation.
6. **Iatrogenic:** Sometimes following a tooth extraction an oroantral fistula remains. Other procedures such as tumor surgery (maxillectomy), corrective surgeries (e.g. septoplasty) or intubation can cause palatal perforation.
7. **Traumatic origin:** Case reported with animal injury, ingestion of hot foods (thermal injury). Tantrum oris.
8. **Miscellaneous:** Midline nonhealing granuloma. Rhinolith can result in palatal perforation. Patients with psychologic problems may present with a fictitious palatal perforation. Sometimes an elderly edentolous denture wearer complains of a perforation in his/her palate. Due to bone resorption a previously impacted canine may emerge in the form of a palatal perforation. An orthopantomograph can easily reveal the impacted tooth.

31. What are the lesions associated with electrogalvanism?

Oral galvanism observed in persons having metal fillings of diverse electrical potentials in teeth that are either next to each other or opposite to each other. Saliva serves as conductor through which mild conductor passes and creating an appropriate environment for ion exchange, this causes electrogalvanism and electric current. This may result in pain that can be reduced by applying varnish. It is called galvanic keratosis, it is due to weak electric current from dissimilar restorative metal, nonscrapable lesion disappears on removal of restorations.

Electrogalvanic lesions consist of chronic gray patches or ulcerations at the site of two dissimilar metallic dentures.

Electrogalvanic or mercury contact allergy perhaps should be considered an entity separate from leukoplakia. The two types of clinical lesions that a microgalvanic current from dissimilar metal restorations can produce on adjacent gingivae, tongue, or buccal mucosa are: (1) Keratotic plaque lesions like leukoplakia, and (2) a variation that mimics LP. Electrogalvanism has been found to be associated with carcinoma cases developed from oral leukoplakia, glossodynia.

32. What are the injuries likely to happen with the misuse of cotton in oral cavity?

1. **Cotton roll stomatitis (cotton roll burn, cotton roll ulcer, cotton roll injury):** Cotton rolls are used during restorative and endodontic procedures. Oral mucosa can adhere to dry cotton rolls, and rapid removal of the rolls from the mouth often can cause stripping of the epithelium in the area. Clinically, the lesions appear as painful erosions covered with a whitish pseudomembrane

and may heal within 4–6 days. Clinically, it is similar to acetylsalicylic acid burns.

2. **Mucosal injury:** Caustic materials (e.g. sodium hypochlorite) can leak into the cotton roll and be held in place against the mucosa for an extended period, with mucosal injury resulting from the chemical absorbed by the cotton. The affected mucosa may be red and painful with superficial erosions and heals spontaneously within 4–6 days. The use of the rubber dam can dramatically reduce iatrogenic mucosal burns.

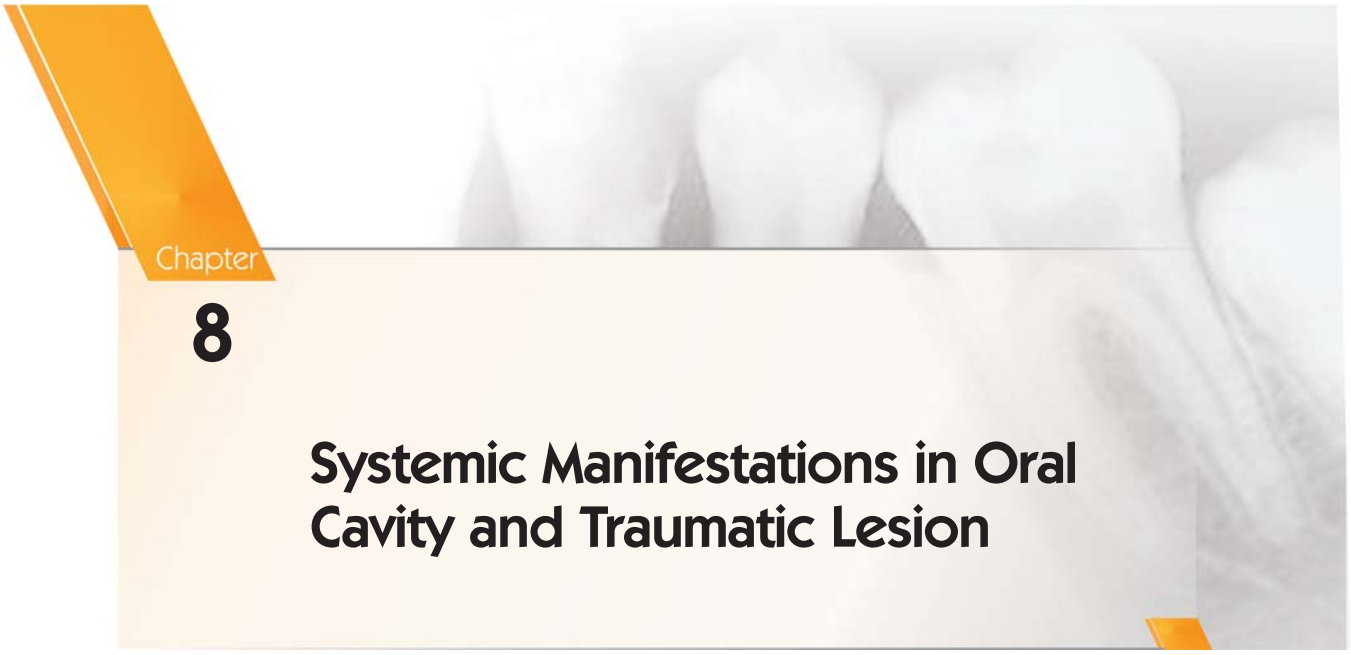
33. What are oral lesions associated with orogenital sexual practices?

The orogenital sexual practices are common, although these are considered illegal in many jurisdictions. These acts are almost universal amongst homosexual males and females. Even though the frequency of orogenital sexual practices is as high as 90% in married heterosexual couples especially younger age, the frequency of associated traumatic oral lesions is surprisingly low.

Clinical Features

1. **Lesions on palate:** It is the most common lesion. This lesion is described as submucosal palatal hemorrhage secondary to fellatio. The lesions appear as erythema, petechiae, purpura or ecchymosis of the soft palate. The areas are often asymptomatic and resolve without treatment in 7 to 10 days. Recurrences are possible with repetition of the inciting (exciting) event. The erythrocytic extravasation is thought to result from the musculature of the soft palate elevating and tensing against an environment of negative pressure.
2. **Lesion on tongue:** Oral lesions also can occur from performing cunnilingus, resulting in horizontal ulcerations of the lingua frenum. As the tongue is thrust forward, the taut frenum rubs or rakes across the incisal edges of the mandibular central incisors. The ulceration created coincides with sharp tooth edges when the tongue is in its most forward position. The lesions resolve in 7 to 10 days but may recur with repeated performances. Linear fibrous hyperplasia has been discovered in the same pattern in individuals who chronically perform the act.
3. **HSV:** HSV1 is common in pharynx and intraoral sites, while HSV2 is common in genital area and skin below the waist. The changing sexual practices have made both lesions common in oral cavity and below waist.
4. **Gonococcal pharyngitis:** More common in male homosexuals and lesions show mixture of signs of inflammation, edema, vesiculation, ulceration, and pseudomembranes.
5. **HPV associated oral and oropharyngeal cancers:** These may be seen in frequent sexual and orogenital sexual contact especially in younger patients who are nonsmokers.
6. **Molluscum contagiosum:** To oral partner in orogenital sex.

No treatment is required, and the prognosis is good. Palatal petechiae can be prevented through the use of less negative pressure and avoidance of forceful thrusting and abstinence of act. Smoothing and polishing the rough incisal edges of the adjacent mandibular teeth can minimize the chance of lingual frenum ulceration.



Chapter

8

Systemic Manifestations in Oral Cavity and Traumatic Lesion

1. Define oral medicine.

It may be defined as a branch of dentistry that deals with the relationship between oral and systemic diseases and also with the diseases that are not manifested orally

or

It is the specialty of dentistry that is concerned with the oral health care of medically compromised patient and with the diagnosis and nonsurgical management of medically related disorders or conditions affecting the oral and maxillofacial region

or

Oral medicine is that specialty of dentistry concerned with the basic diagnostic procedures and techniques useful in recognizing the disease of the oral tissues of local and constitutional origin, and their medical management.

2. What are vital signs? Enumerate various vital signs. Which is fifth vital sign?

Vital signs represent the real significance of life. It can be defined as body's physiological status and provide information critical to evaluating homeostatic balance. Temperature, pulse, respiratory rate along with blood pressure reading are known as vital signs. Assessment of pain is referred to as 'fifth vital sign'. However, some authors do recommend ECG, oxygen saturation and tobacco use as fifth vital sign. These are taken daily or only once in a week. However, in critically ill-patients the machines are attached.

3. What is evidence-based dentistry?

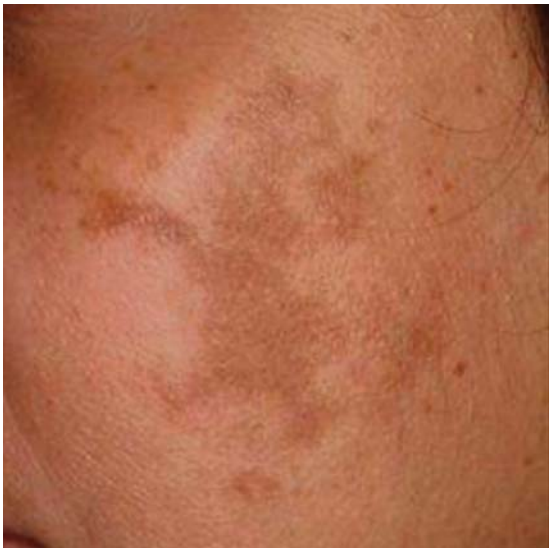
American Dentist Association (ADA) defines it as an approach to oral health care that requires the judicious integration of systematic assessment of clinically relevant scientific evidences, relating the patient's oral and medical conditions and, history with the dentist's clinical expertise, and the patient's treatment needs and preferences.

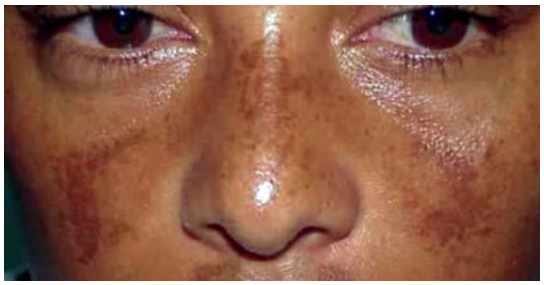
4. Enumerate pregnancy associated oral and paraoral changes.

The degree to which the associated changes occur is partly related to the previous health and the effectiveness of preventive procedures performed by pregnant woman.

1. **Caries, gingivitis, and periodontal disease:** Tooth mobility is associated with increased hormone levels when it is not associated with periodontitis, the condition is reversible.
2. **Xerostomia.**

3. **Enamel erosion:** May be seen when gastric acids present in the vomitus may erode the lingual surfaces of anterior teeth. Most common in first trimester.
4. **Pregnancy-induced gingivitis:** Increase in metabolism of estrogens by gingival may increase sensitivity to local irritants like plaque and food debris causing inflammation. Increased synthesis of prostaglandins results sensitivity to inflammation.
5. **Pregnancy tumor:** Granuloma gravidum usually develops in second trimester resolves after delivery. Histologically, pyogenic granuloma closely resembles. It occurs on gingiva, lips, tongue, and buccal mucosa. It is seen commonly on facial aspects of upper anterior teeth, excision not needed unless it impedes mastication or hinders routine dental care.
6. **Chloasma gravidarum** is the tanned mask seen on the cheeks, nose, and infraorbital areas of pregnant, light-skinned women during the latter half of pregnancy. It is occasionally accompanied by a diffuse browning of the oral mucosa. The pigmentation slowly disappears after delivery. The increased level of adrenocorticotropic hormone (ACTH) during pregnancy is thought to account for the increased melanocyte activity.





7. **Emerging evidence** has shown that periodontal disease may be associated with preterm, low birth weight (LBW) babies.
8. **Recurrent aphthae** may stop or even become severe during pregnancy.

5. What are the causes of premature loss of teeth?

Trauma (avulsed teeth), aggressive periodontitis (premature alveolar bone loss), acrodyania, immunocompromised states (AIDS, leukemia, chemotherapy), diabetes, especially juvenile onset (increased susceptibility of infection and severity of periodontitis), osteomyelitis (bone destruction, loosening of teeth), cyclic or chronic neutropenia (increased susceptibility to infection, premature bone loss), Langerhans cell histiocytosis (premature alveolar bone loss), dentin dysplasia type 1 (rootless teeth), regional odontodysplasia (ghost teeth), Papillon-Lefèvre syndrome (premature periodontitis), Down's syndrome (premature periodontitits), hypophosphatasia (lack of ementum production), scurvy (vitamin C deficiency), Ehlers-Danlos syndrome, agranulocytosis.

In teenagers it is indication of underlying destructive process of bone, leukemia, histocytosis X or benign neoplasm.

6. What is halitosis? Enumerate the different causes of halitosis.

Halitosis is a term used to describe a noticeably unpleasant odour exhaled in breathing. The other names are fetor oris, breath malodour, foul breath. The cause may be oral/non-oral. Oral malodour is the term used to describe odour from the oral cavity. It is classified as genuine halitosis, pseudohalitosis and halitophobia. Pseudohalitosis is the condition

Not associated with disease	*Associated with disease*
Morning breath Medications, mouth breathing Aging, poor denture hygiene Fasting/starvation Tobacco, foods like onion, garlic, meats, alcohol	Oral/paraoral structure: Periodontal disease, xerostomia (associated with underlying disease), gingivitis, stomatitis, glossitis, cancer, candidiasis, parotitis GI disorders Gastro-esophageal reflux, hiatus hernia, cancer Nasal Rhinitis, sinusitis, tumors, foreign bodies Pulmonary bronchitis, pneumonia, TB, cancer Psychiatric Delusions Other causes Diabetes, uraemia, hepatic disease, blood dyscrasias, rheumatologic diseases, dehydration, and fever, etc.

where malodour is not perceived by others but patient always complains of its existence. Halitophobia is psychosomatic halitosis or imaginary bad breath associated with psychiatric disorder.

From oral cavity: Poor oral hygiene, poor hygiene of prosthesis, oral infection (candidiasis, pericoronitis, dry socket), oral ulcerative/erosive lesions

Nasal cavity: Nasal infections, polyps, sinusitis. Dietary considerations—volatile sulphur containing foods (garlics, onions), hydrogen sulphide

Salivary gland: Xerostomia

Obstruction of airflow: Tonsils, infections

Systemic diseases: Bronchial/pulmonary infection, renal failure.

7. What is oral allergy syndrome?

Swelling of lips, tongue and palate, throat along with oral pruritis and irritation, sometimes associated with other allergic clinical features including rhino conjunctivitis, urticaria and even anaphylaxis has termed oral allergy syndrome. It seems to be precipitated by fresh foods including apples in people who have been sensitized to cross-reacting allergens in pollens particularly birch.

8. What is lingual papillitis? What are the different patterns of it?

Common disease in which affected individuals experience clinical alterations which involve variable number of fungiform papillae of tongue. It is also called lie bumps, tongue torches. The suggested causes are local irritation, stress, GIT diseases, hormonal fluctuation, URTI, viral infection, topical hypersensitivity to foods, and drinks or oral hygienic products.

Three patterns have been documented.

1. It is localized and involves one to several fungiform papillae that become enlarged and present as elevated papules that are red, may demonstartae yellow ulcerated cap. These are common on anterior portion of tongue, with mild to moderate pain resolves spontaneously within hours to week.
2. Involvement is more generalized and affects more papillae, tip, and lateral portion may be involved. Fever and cervical lymphadenopathy may be seen. Resolution in week, rarely recurrence.
3. More diffuse involvement. Papillae are asymptomatic, elevated white to yellow papules called papulo keratotic variant.

9. What are the different types of glossitis? What are Moeller's glossitis and Hunter's glossitis?

Glossitis is a condition refer to an inflammation of the tongue; it may be caused by injury, infection, allergy reaction. All these causes can

be inducing glossitis with varying severity. Bacterial or viral infections (including oral herpes simplex).

Mechanical irritation or injury from burns, rough edges to teeth or due to appliances, or other trauma.

Exposure to irritants such as tobacco, alcohol, hot food or spices.

Allergic reaction to toothpaste, mouthwash.

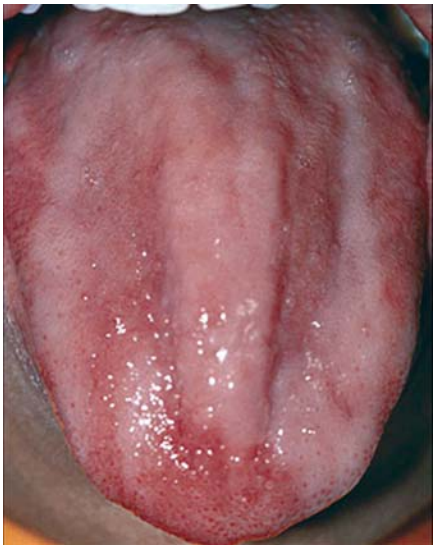
Disorders such as iron deficiency anemia, pernicious anemia, and other vitamin B deficiencies, oral syphilis and others.

Yeast infection, dry mouth associated with connective tissue disorder, such as Sjögren studies. Occasionally glossitis can be inherited.

Moeller's and Hunter's glossitis is used to describe the condition associated with vitamin B_{12} and pernicious anemia.

10. Describe various changes occurring in tongue in various anemias.

Iron deficiency anemia	The tongue is often described as red and bald with glazed and dry appearance. The tongue may have waxy appearance and glossitis.
Pernicious anemia	Soreness, severe pain, itching or burning of tongue. Atrophy of lingual papillae with some complaining of painful glossitis. Beefy red tongue is characteristics. If the colour is less beefy red and surface is smooth, then indications that pernicious anemia is disappearing or its mild form. Glossitis and atrophy of papillae occur more common with pernicious anaemia and less often with iron deficiency anemia.
Megaloblastic anemia	Initially the tongue is fissured/lobulated eventually becomes smooth flabby, atrophic. Depapillated beefy red tongue (Hunter's glossitis/Moeller's glossitis), altered sensation of tongue is characteristic.



Bald tongue

11. What is chloroma?

It is granulocytic sarcoma and called chloroma. Granulocytic sarcomas are composed of a localized collection of immature myeloid cells and are considered to be specific lesions of AML or the onset of a blast crisis in chronic myelogenous leukemia (CML). Localization in the oral cavity is rare. Granulocytic sarcoma was described for the first time by burns in 1811. In 1873, the King dubbed ''chloroma" because of its green colour to the cut.

12. What is Quincke's disease?

Angioedema or angioneurotic edema is also called Quincke's disease. It is a diffuse edematous swelling of the soft tissues that most commonly involves the subcutaneous and submucosal connective tissues but may affect gastrointestinal and respiratory tract to result fatally. It is characterized by the relatively rapid onset of soft, nontender tissue swelling, which may be solitary or multiple.

13. What is Chvostek sign (Chvostek-Weiss sign)?

It is given by Frantisek Chvostek in 1876. The Chvostek sign is one of the signs of tetany seen in hypocalcemia. It refers to an abnormal reaction to the stimulation of the facial nerve.

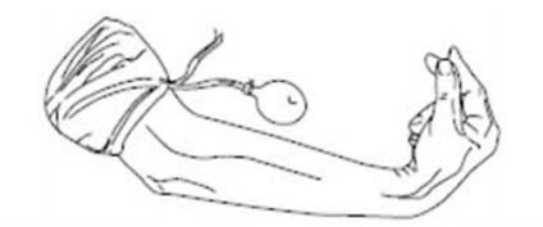
Chvostek I phenomenon: Contracture of facial muscles produced by tapping on the facial nerve at a specific point which is located 0.5 to 1 cm below the zygomatic process of the temporal bone, 2 cm anterior to the ear lobe, and on a line with the angle of mandible.

Chvostek II phenomenon: It can be produced by tapping on different location of the face. The point is located on the line joining the zygomatic prominence and corner of mouth, one-third of distance from the zygoma.

In both instances the response is twitching of the muscles supplied by facial nerve on that side. This is because of hypocalcemia (i.e. from hypoparathyroidism, pseudohypoparathyroidism, hypovitaminosis D) with resultant hyperexcitability of nerves. Though classically described in hypocalcemia, this sign may also be encountered in respiratory alkalosis, such as that seen in hyperventilation, rickets, diphtheria, measles, scarlet fever, whooping cough, and myxedema.

14. What is Trousseau's sign?

Named after Sir Armand Trousseau, a French physician (1801–1867), it is a test for latent tetany in which carpal spasm is induced by inflating a sphygmomanometer cuff on the upper arm to a pressure exceeding systolic blood pressure for 3 minutes. A positive test may be seen in hypocalcemia and hypomagnesemia. Occlusion of the brachial artery causes flexion of the wrist and metacarpophalangeal joints, hyperextension of the fingers and flexion of the thumb on the palm producing the characteristic posture called main d'accoucheur.



The patients with positive Trousseau's sign may also experience paresthesia of fingers, muscular fasciculations or twitches of the fingers and a sensation of muscular cramping or stiffness.

15. What is pathognomonic sign? What are the different pathognomonic signs?

Pathognomonic (*patho* means disease, *gnoma* means *signature*, pathognomonic is signature of the disease), especially distinctive or characteristics sign of the disease or pathological condition on which a diagnosis is made) or a pathognomonic sign is a sign whose presence means that a particular disease is present beyond doubt.

- Ptregyoid sign which is pathognomonic for TMJ pain origin. The examiner faces the patient and to examine the right side faces the tip of little finger to the above and slightly behind maxillary tuberosity. If the patient winces the sign is positive and same is done for left side. It has been described by Frost (1972).
- Pterygoid and greenstick fractures in adults are almost pathognomonic for osteomalacia.
- Hemarthrosis for hemophilia.
- Exophthalmos for hyperthyroidism.
- Chvostek sign and Trousseau signs for hypocalcemia.
- Subperiosteal erosions, especially of the middle phalanges are almost pathognomonic for hyperparathyroidism, which are the recalcitrant benign giant cell lesion of the jaws.
- In fact, the maxillary enlargements resulting in prominent cheek bones and anterior displacement of the incisors produce a characteristic "rodent" facies, which when

coupled with the sinus hypoplasia is pathognomonic of thalassemia.

- A round, radiopaque enlargement of the condyle with an anterior radiopaque projection along the lateral pterygoid tendon. This radiographic picture is pathognomonic for an osteochondroma.
- Specifically the clinician should form a strong impression of central hemangioma (CH) when encountering a pumping tooth (a tooth that can be pushed apically and then rebounds to its original position) or localized gingival bleeding around a loose tooth coupled with radiographic evidence of bony change in the region. This impression may be further strengthened when large quantities of blood are easily aspirated from the area.
- Soft, dark tooth structure that can be penetrated by dental explorer or sharp instrument clinically is pathognomonic sign for caries or tooth decay.
- Hairy leukoplakia is almost pathognomonic for HIV.
- Pseudomembranes on tonsils, pharynx and diphtheria are pathognomonic for diphtheria.
- Strawberry gums is red-puplish granular gingivitis—a rare but pathognomonic sign of Wegener's granulomatosis.
- Painful local rash for herpes zoster.
- Measles show Koplik's spot.
- In the early vesicular stage, pathognomonic features include the ballooning of the epithelial cells is pathognomonic for intraoral herpes lesions.
- The presence of numerous neurofibromas or a plexiform type of neurofibroma is characteristically pathognomonic for NF1 (neurofibromatosis 1 or von Recklinghausen's disease.
- The pathognomonic features, such as small purple or red flat papules on extensor surfaces, particularly the elbows and joints of the hand (Gottron's papules). These are pathognomonic for dermatomyositis.
- Pathognomonic sign for necrotizing fasciitis of head and neck dusky, purplish discoloration of skin, small purplish patches with ill-defined borders, necrosis of fascia, and gangrene of overlying skin.
- Slight digital pressure on the periphery of a parulis may force a drop of pus from the sinus opening and this is almost pathognomonic.
- Oral lesions in varicella zoster are characteristic and pathognomonic prodrome followed by unilateral vesicular eruptions soon ulcerates.
- Peutz-Jeghers syndrome shows perioral pigmentation.
- Wash-leather slough on floor is pathognomonic of gummatous ulcer.
- Slip sign in subcutaneous lipoma, the edges are well-defined and when pressed away, it sleeps away.
- The presence of oral and perioral gonorrhea/syphillis is pathognomonic for sexual abuse.
- The gingiva between teeth is missing or punched out is characteristic for ANUG.

16. What is prodromal symptom? Enumerate various prodromal symptoms.

A prodrome is an early symptom (or set of symptoms) that might indicate the start of a disease before specific symptoms occur or it is a symptom that may be the first indication of disease. It is derived from Greek word *prodromos* which indicates forerunner of the event.

Neuralgia in the prodrome stage of zoster is followed by vesicles and ulcers similar in appearance to those caused by herpes simplex. Because the lesions follow a nerve distribution, they extend to the midline and stop.

Recurrent aphthous ulcers: These may begin as prodromal burning, itching or stringing 24 to 48 hours before the ulcer begins (little symptoms).

Mumps: They may have headache, chills, moderate fever, pain below ear, which lasts for 1 week. The child usually has a nonproductive

cough, photophobia due to conjunctivitis, and often a discharge or crusting around the eyes. This set of signs and symptoms is often called the prodrome because it precedes any skin rash. Oral lesions called 'Koplik spots' also appear during this prodrome and are pathognomonic of the disease. Koplik spots appear as flat, erythematous maculas with tiny white "salt crystal" centres.

Primary herpes simplex infections: May be preceded by fever, headache, nauseas, malaise and lymphadenopathy. These symptoms may be useful to differentiate from vesiculobullous lesions of oral cavity.

Secondary herpes (herpes labialis): May show tingling, burning or pain at the site.

Erythema multiforme lesions: May have fever, headache, general malaise.

Dermatitis herpetiformis: May have burning sensation before the lesion occurred.

Acute radiation syndrome: May have anorexia, nausea, vomiting, diarrhoea, weakness, fatigue.

Rubella: May have Forchheimer's sign which refers to an enanthem of red macules or petechiae confined to the soft palate in one-fifth of patients.

Syncope: May not have prodormal symptoms.

17. What is adverse drug reaction? What are the various manifestations of adverse drug reactions in the oral cavity?

An adverse drug reaction is defined by Edwards and Aronson in 2000 as "an appreciably harmful or unpleasant reaction, resulting from an intervention related to the use of a medicinal product, which predicts hazard from future administration and warrants prevention or specific treatment or alteration of the dosage regimen or withdrawal of the product."

or

An adverse drug reaction is defined by WHO as "a response to a drug which is noxious and unintended, and which occurs at doses normally used in man for the prophylaxis, diagnosis, therapy of diseases or for the modification of physiological function".

The author makes a distinction between an adverse effect (adverse outcome attributed to an action of the drug), and an adverse event (adverse outcome that occurs when a patient is on the drug but that may not be caused by the drug). An adverse drug event includes the following:

1. Harm caused by a drug (commonly known as adverse drug reaction)
2. Harm caused by appropriate drug use (usually referred to as a side-effect)
3. Medication errors.

These can be classified as:

1. **Salivary gland disorders:** Xerostomia, ptyalism, salivary gland pain, discoloration of saliva.
2. **Oral ulceration:** Nonspecific ulcerations, aphthous ulcerations, fixed drug eruptions, mucositis, pemphigoid like reactions, pemphigus, erythema multiforme, lupoid reactions.
3. **Oral malodor**
4. **White lesions:** Lichenoid reactions, oral candidiasis, black hairy tongue
5. **Taste ulcerations**
6. **Mucosal pigmentation**
7. **Teeth discoloration**
8. **Swellings:** Gingival and mucosal swellings.

18. Enumerate various drug associated oral lesions.

Common reactions produced in the oral cavity are stomatitis, ulceration, necrosis, hemorrhage, gingival hyperplasia, pigmentation, altered salivary function and altered taste sensation.

The most common allergic reaction is erythema multiforme producing ulcers in mouth with pain and discomfort. Followed by anaphylactic stomatitis, intraoral fixed drug reactions, lichenoid drug reactions, and lupus erythematous like eruptions, pemphigus like eruptions and nonspecific like vesiculoulcerative lesions. Stevens-Johnson's

syndrome, toxic epidermal necrolysis, medication-induced mucositis.

A number of different medications cause oral mucosal lesions that do not appear to be allergic reactions but rather represent a toxic side-effect of the medication. These mucosal lesions can present as nonspecific ulcers, erosions or may resemble erosive lichen planus. They do not necessarily appear immediately after the patient begins taking the medication.

Some drugs become bound to cell membranes in one or many organs as part of their mechanism of action or their elimination. If, through sensitization, the drug has stimulated antibody production by the coupling of antibody to the antigen fixed to the cell membrane, the drug in question may cause cell lysis. This type of reaction, which may be very severe and cause extensive tissue destruction, is thought to be the mechanism responsible for erythema multiforme and, although less severe, so called lichenoid drug reaction. It has been known to produce a hemolytic anemia when the drug is fixed to red blood cell membranes.

While any drug can cause a systemic drug reaction, some drugs historically have produced a higher incidence than others. The following substances have the highest incidence of these reactions:

1. Levothyroxine sodium
2. Gold salts
3. Allopurinol
4. Colchicine
5. Methyldopa
6. Nifedipine (procardia)
7. Penicillin (particularly ampicillin)
8. Codeine
9. Propranolol.

Patients with drug-induced avascular necrosis of bone will present with exposed white- or yellow-colored bone, either in the maxilla or the mandible that is obviously nonvital.

Inedentulous individuals, the mylohyoid ridge, and the crestal bone are the more commonly affected sites, apparently because of denture pressure. In dentate areas, any of the teeth may be involved in what will look like advanced periodontal disease.

Clinically, contact drug reactions produce a soft, spongy, red attached gingiva. They may also affect to a lesser degree the labial mucosa, tongue, and commissures. The gingiva and tongue are reported to have a dull, burning sensation.

19. What are morbilliform drug reactions?

These are also called maculopapular or exanthematous drug reactions. Symmetric erythematous macules/papules which may become confluent. Oral mucosa may show erythema or lichenoid damage. These are seen from less than week after starting treatment until several weeks after discontinuation of drug. Topical steroids and antihistaminics may be used. The most common drugs are antibiotics, antiepilectics, allopurinol, NSAIDs, antianxiolytics, antihypertensives, diuretics. Generalised morbilliform reactions are common with ampicillin and amoxicillin. Viral infections may increase the incidence. It is tenfold common in HIV patients. This is often the initial presentation of more serious reactions including toxic epidermal necrolysis, hypersensitivity syndrome, and serum sickness.

20. What are fixed drug eruptions?

This is peculiar phenomenon whereby one or more inflammatory patches appear at the same cutaneous or mucosal site on each occasion that the patient ingests a culprit drug. The time may vary from 2 to 24 hours. Common sites are genitalia, torso, hands, feet, face, and intertriginous areas, i.e. axilla and groin. Common drugs are NSAIDs, tetracyclines, chlorpromazine, and calcium channel blockers, sulfonamides, barbiturates, analgin, and chlorhexidine.

The oral lesions appear as localized areas of erythema and edema commonly seen on labial mucosa and can later develop into

vesiculoulcerative lesion even mouth ulcerations and post-inflammatory pigmentation.

It can be seen as manifestations of oral hypersensitivity reaction. Primary treatment is withdrawal of the drug and avoidance in future. Potent topical corticosteroids may be given to avoid further complications and post-inflammatory pigmentation.

The third generation antihistamine levocetirizine was reported in a case of FDE involving the oral (lower lip and tongue), and genital tissues (glans penis). Use of acetaminophen was reported to result in erythematous and papular FDEs on the hard palate and skin; naproxen and oxicams have caused lesions on the lips and fluconazole has caused lesions of the palatal mucosa and oral bullae.

21. Enumerate drug-induced oral reactions.

These reactions are:

Hyposalivation/xerostomia, lichenoid reaction/lichen planus, aphthous-like ulcers, bullous disorders, pigmentation, fibrovascular hyperplasia, keratosis/epithelial hyperplasia, dysesthesia, osteonecrosis of the jaws, infection, angioedema, and malignancy.

22. Name the different medications associated with tooth discoloration.

Clinically evident tooth discolorations can be seen due to different medications that become incorporated into the developing tooth. The severity of the alterations is dependent on three factors, i.e. the time of administration, the dose and the duration of the drug's use. The discolorations may be in the form of white spots, yellow to dark brown and gray generalised discoloration. These stains may be extrinsic and intrinsic. The other effects of drugs on teeth include physical alteration of tooth structure and changes in tooth sensations.

Several patterns of staining are noted in the dentition. Fully erupted teeth typically reveal a blue-gray discoloration of the incisal, three-fourths with the middle one-third being maximally involved. The exposed roots of erupted teeth demonstrate a dark green discoloration, although the roots of developing teeth are stained dark black. The dental discoloration remains (because of the extensive involvement of dentin) even though the cutaneous staining fades, after the discontinuation of medication,

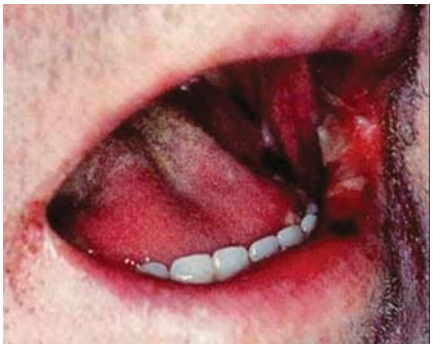
1. Atropine, antidepressants, antihistaminics, opioid analgesics can cause reduced salivary flow and may be causative factor for carious destruction, the most common tooth discoloration.
2. Sucrose containing oral medication such as cough syrups or nutritional supplements can cause caries.
3. Tetracycline in the affected teeth can cause bright yellow to dark brown discolorations and, in UV light showing a bright yellow fluorescence.
4. Chlortetracycline causes gray-brown discoloration and oxytetracycline causes yellow.
5. Minocycline hydrochloride appears to bind preferentially to certain types of collagenous tissues (e.g. dental pulp, dentin, bone, etc.). Once in these tissues, oxidation occurs and may produce the distinctive discoloration. Additionally blue-gray appearance on palate or anterior alveolar mucosa that represents the black bone showing through the thin, translucent oral mucosa.
6. Chlorhexidine gluconate, liquid preparations of iron salts and some antibiotics in liquid form and essential oils can cause extrinsic staining.
7. Fluoride causes intrinsic staining.

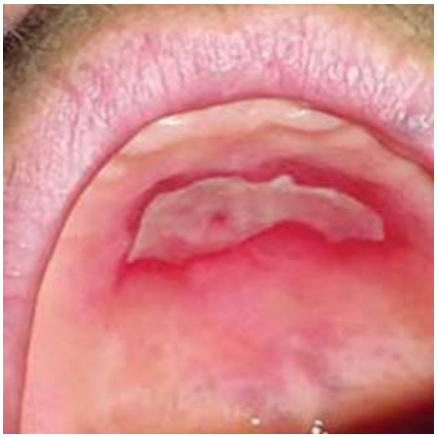
Suggested aesthetic solutions include restorations, full crowns, external bleaching of vital teeth, internal bleaching of nonvital teeth, bonded restorations, composite build-ups, and laminate veneer crowns.

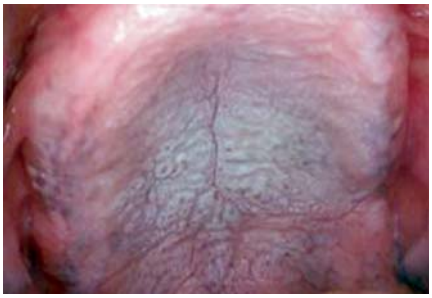
23. What are oral manifestations of renal disease?

Ammonia like taste and smell, stomatitis, gingivitis, decrease in salivary flow, xerostomia, parotitis. Early symptom and particularly in morning. Uremic fetor and ammonical odor is due to high concentration of urea in

the saliva and is subsequent breakdown to ammonia. Salivary urea levels may correlate well to BUN, but no fixed relationship. Uremic stomatitis. It occurs BUN level >150 mg/dl. Resolves with medical treatment. It is regarded as chemical burn or general loss of tissue resistance and inability to withstand normal/traumatic influences. Erythemopultaceous form—red mucosa covered by thick exudate and pseudomembrane. Ulcerative form—frank ulcerations with redness and a pultaceous coat.







Uremic Frost

White plaques occasionally on skin and rarely on oral mucosa. Residual area crystals left on skin after perspiration evaporates/decreased salivary flow.

24. What are care oral health considerations in renal disesaes?

Elective dental care should be deferred until recovery. No contraindications if peritoneal dialysis. During surgery meticulous technique should be followed. Wound by primary closure and use of local hemostatic aids (cellulose).

Elective procedures should be done on nondialysis days, avoid on dialysis day. Timing early in the dialysis cycle. Use of arm on arteriovenous site I/M, I/V injections should be avoided and BP checking should be avoided. Avoid sitting, permit walking during long procedures.

25. Which are the drugs avoided in renal diseases?

Steroids, tetracyclines and nonsteroidal anti-inflammatory agents, and aminoglycoside.

26. What care should be taken while managing the patients with renal failure, kidney dialysis, and kidney transplants in dental practice?

Consult nephrologist

Check and correct bleeding time

Need of antibiotic cover prior and need of supplemental steroids

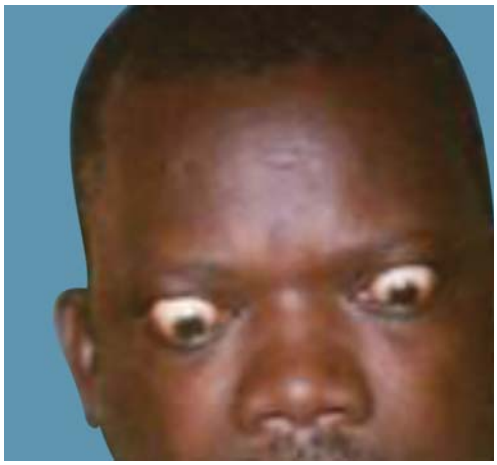
Drugs avoided—NSAIDs, aspirin, and tetracyclines.

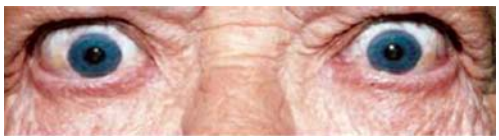
Procedures should be avoided on the day of dialysis.

27. What are exophthalmos and proptosis?

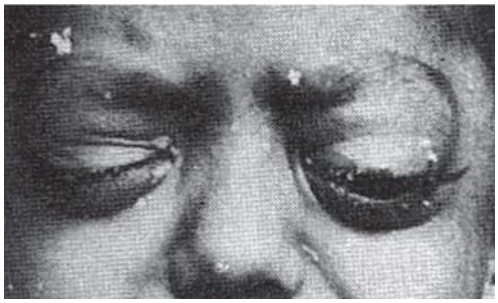
Both these conditions describe abnormal protrusion of eyeball. Proptosis is forward displacement of organ while exophthalmos is only related to eyes. Henderson reserves the use of the word exophthalmos for those cases of protrusion, secondary to endocrinological dysfunction and indicates bilateral protrusion. For example, thyroid dysfunction and Graves'

disease. Proptosis is axial protrusion of eyeball, may be unilateral or bilateral. The aetiological basis of proptosis can include inflammatory, vascular, infectious, and cystic, traumatic. The protrusion is more in proptosis than exophthalmos.

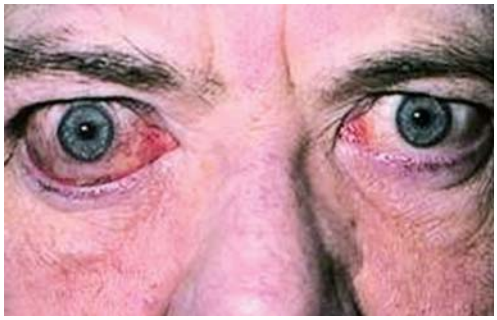




Exophthalmos in hyperthyroidism



Proptosis of left eye in leukemic patient



Right eye proptosis

28. What care should be taken for patients with gastrointestinal disease in dental practice?

Routine dental treatment should be provided for patients with inflammatory bowel disease and ulcerative colitis by taking prior history. Aspirin avoided and acetaminophen recommended.

Antibiotics and dietary supplements should be taken 2 hours before after antacids.

Tetracycline should be avoided if patients are on antacids like gelucil that contains aluminium hydroxide as the absorption may be prevented.

Oral fungal infections are likely to get precipitated in long-term antibiotics.

29. What care should be taken in diabetic patients in dental practice?

1. Morning appointments and preferably short appointments as these patients are more stable in morning.
2. Stable diabetics should take medicines as prescribed while unstable diabetics require physician's guidance.
3. Stress must be reduced. Use of analgesics and/or other premedication should be considered. Use of bathrooms during procedures and availability of small snacks will reduce stress, as the stress releases endogenous epinephrine which leads to mobilization of glycogen from liver ultimately leading hyperglycemia.
4. Unstable diabetic patients should be covered by antibiotics (amoxycillin/clindamycin are the choices of drug) for invasive procedures.
5. Need for dietician after procedure.

30. What is INR?

INR has been introduced in 1983. It is the ratio of prothrombin time (PT) that adjusts for the sensitivity of thromboplastin reagents, such that normal coagulation profile is reported as an INR of 1.0. This test evaluates the extrinsic coagulation system and measures the presence or absence of clotting Fs I, II, V, VII and X. Its most common use is to measure the effects of coumarin anticoagulants and reduction of vitamin K dependant Fs II, VII, IX and X.

31. What is capillary fragility test? What are the other names of it? State in which conditions it is positive.

It is a nonspecific evaluation to measure capillary wall weakness and deficiencies in platelet number and function. An inflated blood pressure cuff at specific pressure for a fixed period of time produces increased pressure and hypoxia in the capillaries distal to cuff. Decreased capillary resistance causes capillary to rupture and leads to bleeding and petechiae formation.

The blood pressure cuff in the arm is inflated in midway between clients systolic and diastolic blood pressure (but not more than 100 mm of Hg). Leave the cuff inflated for at least for 5 minutes and observe at least 1 inch distal to The cuff for formation of petechiae. (The pre-existing petechiae are counted additional.) Negative—no petechiae males 5 or less petechiae, females 10 or less petechiae.

It is also called capillary fragility test, negative pressure test, Rumpel-Leede capillary fragility test, dengue test, Hess test, vitamin C test, etc.

The test is positive in purpura, vessel wall defect, scurvy, liver disease, dengue fever, factor VII deficiency, aplastic anemia acute leukemia, chronic nephritis, decreased estrogen level in postmenopausal woman, disseminated intravascular coagulation, fibrinogen deficiency, hereditary telangiectasia, drugs like long-term steroid use.

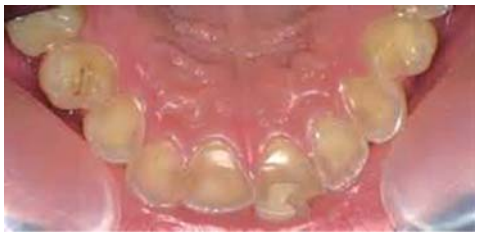
32. What is the difference between Cushing's disease and Cushing's syndrome?

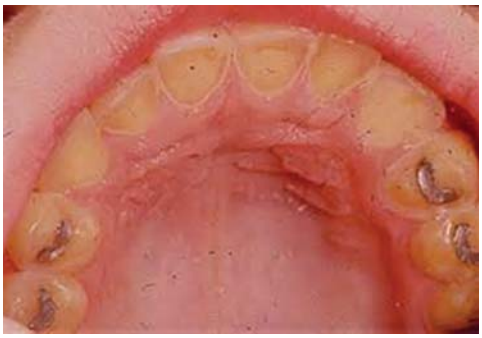
Cushing's syndrome is caused by excess amount of glucocorticoid production and Cushing's disease is excessive cortisol is produced by excessive ACTH production.

Cushing's syndrome more commonly noted. Clinical findings may be similar.

33. What is perimolysis?

Perimolysis is a form of tooth erosion, characterized by dental tissue demineralization without bacterial involvement; that is, wear or mineral loss from the tooth surface caused by chemical agents, it can be of intrinsic or extrinsic origin. Extrinsic origin is considered when it is caused by excessive consumption of acid drinks and foods such as soft drinks, alcoholic beverages, acid fruit and prescription drugs for daily use. When caused by gastrointestinal disorders, such as those resulting from bulimia, anorexia nervosa and gastroesophageal reflux, it is considered of intrinsic origin. The acid action can cause erosion on palatal/lingual incisor surfaces, and occlusal and palatal/lingual posterior tooth surfaces causing pain.





34. What is erosion? What are the different types of erosion?

It is defined as the loss of dental hard tissue as a result of chemical process not involving bacteria. Erosion derived from the Latin verb *erodere, erosi* (to *gnaw*, to corrode), *Wuszm* describes the process of gradual destruction of the surface of something usually by electrolytic or chemical processes.

Types of Erosion

1. Regurgitation erosion
2. Dietary erosion
3. Industrial erosion

or

i. Extrinsic erosion
ii. Intrinsic erosion

35. Enumerate the noncarious causes of chronic destructive process leading to tooth wear.

Number	*Terminology*	*Causes of loss of hard tissue*
1	Attrition	Mechanical process involving tooth-to-tooth contact
2	Abrasion	Mechanical process involving foreign objects or substances
3	Erosion	Chemical etching and dissolution
4	Demastication	Mechanical interaction between food and teeth
5	Abfraction	Mechanical process involving tooth flexture by eccentric occlusal forces
6	Resorption	Biological degradation

36. What are factitial injuries?

Self-induced lesions because of abnormal habit, some psychological problem or even accidental. They may overlap number of physical and chemical injuries or these are the oral lesions created by the patient that are not attributable to oral disease or accidental trauma. These are difficult to diagnose as they are to treat.

The common type is gingival abrasion by fingernail, obsessive tooth brushing, use of inappropriate aids to clean the teeth and burns caused by aspirin. Even self-extraction/auto-glossectomy are reported in schizophrenic. Patient education and symptomatic treatment is the choice of treatment.

37. What is factitious cheilitis?

The cases of cheilitis arise from chronic injury are termed factitious cheilitis, which is an unusual chronic condition of the lips characterized by crusting and peeling of superficial epithelium often associated with discomfort or burning. A marked female predominance is seen in cases of factitious origin, with younger than 30 years. Mild cases feature chronic dryness, scaling, or cracking of the vermilion border of the lip and with progression, the vermilion can become covered with a thickened, yellowish hyperkeratotic crust that can be hemorrhagic or that may exhibit extensive fissuring.

This often starts from the centre of lower lip and spreads to involve whole of the lower lip or both lips. No association with dermatologic and systemic cause but a few cases seen with HIV.

Some authors also called it exfoliative cheilitis.

In those cases with no underlying physical, infectious, or allergic cause, psychotherapy may achieve resolution. Patient's education regarding discontinuation of potentially causative habits or behaviour is important. Although for refractory cases (1) highly variable, protective moisturizing preparations (topical petrolatum jelly) and tacrolimus ointment (0.1%) once/twice daily may be effective.

38. Name the various factitious disorders.

The term factitious means willfully produced. Factitious disorders are characterized by intentionally produced or feigned psychological or physical signs and/or symptoms in order to assume a sick role in the absence of any known external incentives for the behaviour.

or

Factitious disorders can be defined as mental disorder leading the patient to feign multiple signs and symptoms for the purpose of assuming sick role.

Classification of Factitious Oral Lesions (FOL) by Stewart and Kemohan

Type A: Those superimposed on pre-existing lesion.

Type B: Involving injuries secondary to an established habit.

Type C: Unknown or complex aetiology.

1. **Munchausen syndrome** by proxy refers to the condition where one person induces illness in another for the purpose of indirectly satisfying his/her own unconscious needs. Common mother causes injury to child.
2. **Gilles de la Tourette's syndrome** is a neuropsychiatric syndrome characterized

by motor and vocal tic sudden, purposeless, repetitive nonrhythmic stereotyped movements, e.g. eye twitching, throat clearing, grunting, barking, behavioural problems, and self-mutilation.

3. **Trichotillomania** is a type of compulsive behaviour. Pulling of hair out of root from scalp, eyebrows, eyelashes or pubic area and some other habits such as nail biting and skin picking.
4. **Autism** shows speech difficulties, unusual behaviour. Various degrees of cognitive impairment. Self-injury, e.g. strike his own face, strike body surfaces against hard surfaces like floor walls, self-biting, self-scratching causing bruises, scratches which can become infected.
5. **Lesch-Nyhan syndrome** is genetic disorder affecting males. Self-injury or self-mutilation is a distinctive feature. Kidney problems affecting the level of uric acid in the body.
6. **Riley-Day syndrome** shows reduced pain, temperature sensations. Lack of tear production, ulcer on lips, toe, finger, and unstable gait.

39. What is numb chin syndrome?

"Numb chin syndrome" (NCS) called "mental neuropathy", is a sensory neuropathy characterised by hypoesthesia, paraesthesia or less commonly pain over the chin in the region supplied by the mental nerve and its branches. It was first described by Charles Bell. It is often caused by the presence of a metastatic tumor in the mandible or the base of skull and represents advanced malignancy. Patients may bite their lip inadvertently causing painless ulcers. Symptoms are usually unilateral but up to a third may have bilateral symptoms. Since then, studies have reported a positive correlation linking neuropathies of the mental nerve to metastatic cancers. The most notable are recurrent cancers in the breast, lung, and prostate, as well as leukemia and lymphoma; however, the strongest relationship with numb chin syndrome has been with breast cancer and lymphoma. Multiple sclerosis diabetes other benign tumors radiotherapy osteomyelitis, abscess, dental related iatrogenic trauma, extractions, mandibular surgery, implants, ill-fitting dentures.

40. What is Frey's syndrome?

Frey's syndrome, also known as auriculotemporal syndrome, Baillarger's syndrome, Dupuy's syndrome, is an unusual phenomenon, which arises as a result of damage to the auriculotemporal nerve (due to parotid gland surgery, infection or trauma), and subsequent reinnervation of sweat glands by parasympathetic salivary fibres which leads to gustatory sweating.

The minor iodine starch test can be used to delineate the extent of the lesion. The test is administered by applying an alcohol-iodine oil solution to the affected side of face. The solution is allowed to dry and lightly covered with starch powder. The patient is given a lemon candy for gustatory stimulus for 10 minutes blue discoloration of the starch iodine mixture is interpreted as a positive finding.

Initial management consist of topical Anticholinergics (scopolamine, glycopyrolate, diphenamanil methyl sulphate or aluminium chloride).

41. What are different oral piercings?

Intra and Perioral Piercing

Body piercing is the act of puncturing or cutting a part of the human body, creating an opening in which jewellery may be worn. This may be the reflection of cultural, religious, spiritual practice, and even superstitious beliefs. Eyebrow, ear, ala of the nose, lip, tongue, nipple, navel, and genitals are the areas of piercing and are decorated with an ornament for fashion.

Oral piercing is a form of body art that involves perforating various oral sites for beautification by mostly metallic ornaments. The tongue and cheek may be pierced for religious purpose.

Various Specifications of Oral Piercing

- Mostly barbell types, often pierced in the midline in the tongue towards lingual sulcus. Other most common sites are dorsolateral site of the tongue labial frenum uvula and anterior tongue.
- Other commonly used is ball shaped (94%), cone shaped (4%).
- The materials used for fabrication are titanium acrylic steel and niobium.

Complications

Oral cavity contains millions of bacteria and has a warm and moist environment. These bacteria without proper sterilization care may migrate from the piercing wound to blood stream and cause multiple complications, some of them may be life-threatening like hematoma, septicemia, infective endocarditis, and toxic shock syndrome.

a. Complications during initial piercing procedure (loss of consciousness, bruising, swelling, bleeding, poor anatomical positioning of piercing).
b. Primary post-operative complication (swelling, bleeding, tenderness, nerve damage, gingival inflammation, metallic taste).
c. Secondary post-operative complications (scar formation, kelioid formation, gingival recession, periodontitis, gingival overgrowth, periodontal and periapical abscesses, tooth mobility, tooth abrasions, and sensitivity of the pulp because of galvanism, calculus build-up).
d. Complications that can occur any time (transmission and development of hepatitis C, B, tetanus, HIV candidiasis, infection, cellulitis at piercing site, endocarditis, allergic reactions, choking aspiration or swallowing of loose jewellery, interference with diagnostic test like MRI, improper fit of jewellery, gaging, and drooling.)
e. Perkins et al. have reported a case of Ludwig's angina, secondary to tongue piercing.
f. Forked tongue also has been reported as a complication of tongue piercing.

Forked Tongue

Split tongue and bifid tongue are a rather recent addition to the art of body modification, with a few associated publications. In this practice the anterior one-third of the tongue is split. This has been performed slowly by pulling fishing line through a pierced hole and tightening the loop over a period of 3 weeks or using a surgical instrument or laser to quickly separate the two halves. Some form of cautery is necessary to prevent the halves from reuniting.

Another practice with unique or official manifestation is implantation of a form of talisman (magical charm) called susuk (charm needles, charm pins). This practice is common in Southeast Asia, especially Malaysia, Thailand, Singapore, Indonesia, and Brunei. The susuk is placed by a native magician or medicine man termed bomoh and is thought to enhance or preserve beauty, relieve pain, bring success in business, or provide protection against harm. The majority of the individuals with susuk are Muslims, although Islam strictly prohibits black magic. Therefore, many affected individuals will deny placement of susuk, even when confronted directly with hard evidence.

In most of the occasions piercing is done by non-medical individuals who do not have adequate knowledge about sterilization and disinfection patients should be educated regarding the sequela and the potential dangers of oral piercing. The dental examination should be done by removing oral piercing.

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Section 2

Radiology

9. Radiology—Part 1
10. Radiology—Part 2

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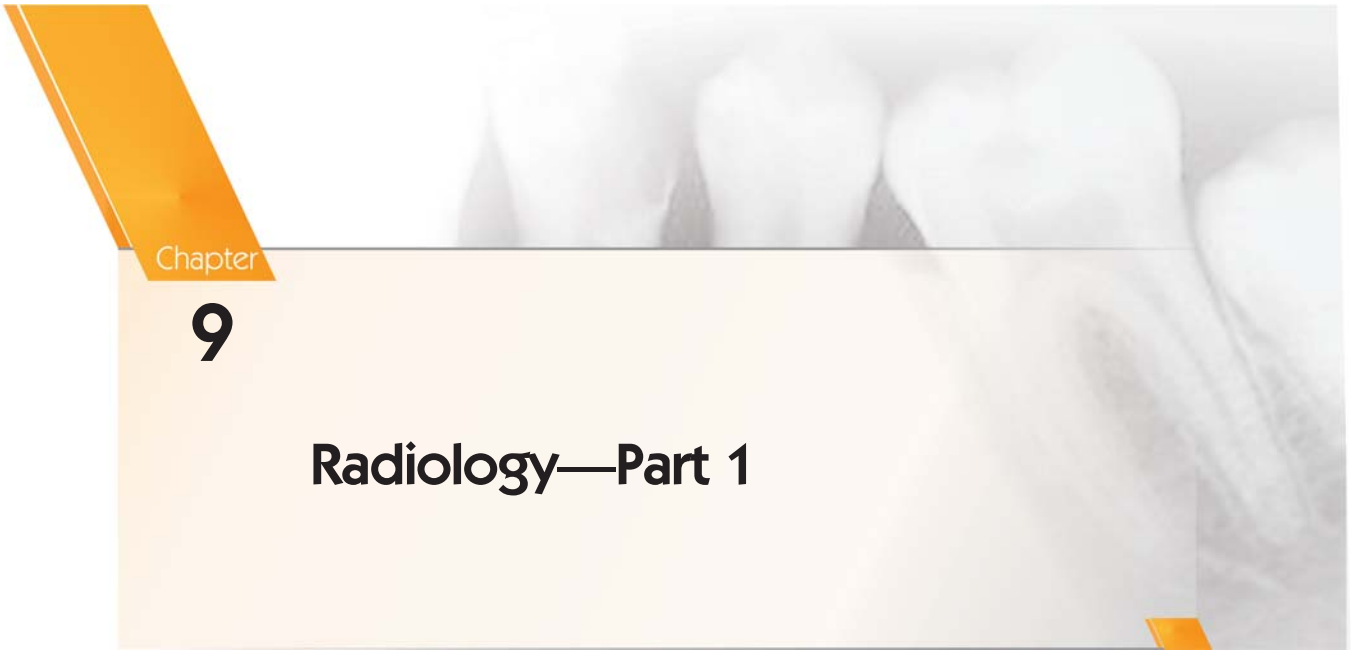


Chapter

9

Radiology—Part 1

1. Define radiology.

It is the medical speciality which embraces all the fields in which electromagnetic radiations are used or produced.

or

It is defined as study and use of radiant energy including roentgen rays, radium, and radioactive isotopes as applied to medicine and dentistry.

2. Define X-rays.

A form of ionizing radiation; which is weightless, neutral bundles of energy (photons) that travel in a wave at the specific frequency with the speed of light; a beam of energy that has the power to penetrate the substances and record image shadows on photographic films also known as roentgen rays.

or

Mechanically generated electromagnetic radiation having an extremely short wavelength of 1 to 10^{-3} Å and have the properties of penetrating various thicknesses of solids and producing secondary radiations by impinging on material bodies, and of acting on photographic films and plates as light does (Merriam-Webster).

or

X-rays may be defined as electromagnetic radiation of wavelength 10^{-5} to 100 Å produced by deceleration of high energy electrons and/or by electron transitions in the inner orbits of atom.

3. Define radiation.

It may be defined as energy in the form of waves or particles, emanating from its source through space in divergent straight lines. However, radiation in the form of charged particles may be deflected from its linear path by electric and/or magnetic fields.

4. Define roentgen.

It is unit of radiation exposure, i.e. amount of ionizing radiations required to produce one electrostatic unit of electricity in one mm^3 at 0°C at standard atmospheric pressure.

5. Define rad.

Amount of radiation energy transferred to mass.

6. Define rem.

It is the dose of any radiation to the biological effect of that dose.

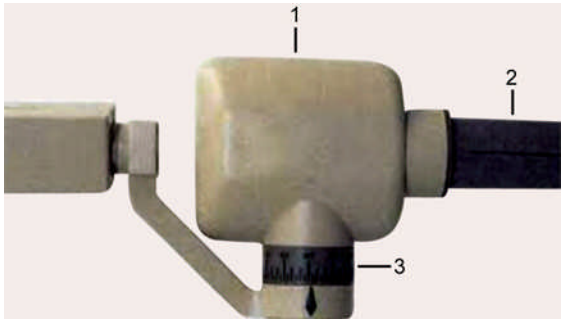
7. What is radiograph? What is ideal radiograph?

Radiograph is processed film or a radiograph is actually a photographic recording produced by the passage of radiation through a subject onto a film, producing what is called a latent image of the subject.

An ideal radiograph has been defined by HM Worth as the one which has the desired density or overall darkness and which shows the part completely, without distortion, with maximum detail and with the right amount of contrast to make the detail fully apparent.

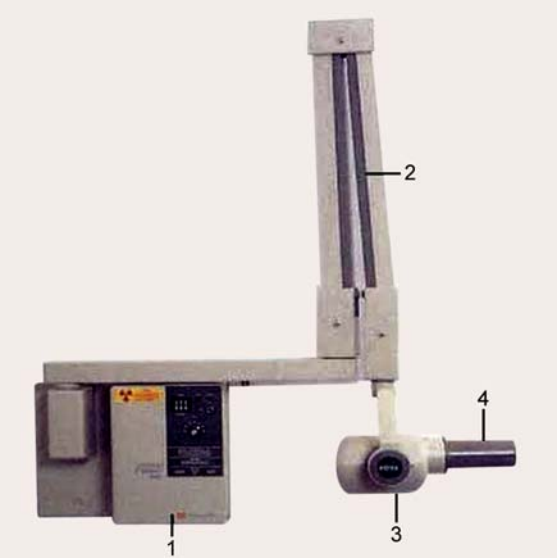
8. What are the components of dental X-ray units?

1. **Control panel:** It is the part where the circuit boards and controls that allow the operator to adjust the correct setting for each patient are located. It is the part where the on and off switch is located, along with selection of mA and kVp and electronic timer. It is the portion of the X-ray machine that houses the major controls.
2. **Arm assembly:** It is attached firmly to wall in the X-ray room. The flexible extension allows the operator to position the tube head for various procedures.
3. The tube head is where X-ray vacuum tube and step-up and step-down transformers are located. The tube head is made-up of leaded glass and has unleaded window (aperture) from where the X-ray exits. It is six inches long and one-half inch in diameter often called a 'coolidge tube'.



(1) Tube head, (2) position indicating device (PID), (3) vertical indicator scale

4. **Position indicating device (PID):** It is also called 'spacer cone'. It is a device for setting the distance from focal spot and skin. It is made-up of plastic and lead lined. It is conical, rectangular and round type.



(1) Control panel, (2) extension arm, (3) tube head, (4) position indicating device (PID)

9. What is the other name of exposure button?

It is also called "Dead man exposure switch". It is called so as it acts as safety feature because it is designated to permit radiation exposure while the switch is depressed. Release of the switch must terminate the exposure. This prevents the exposure for operator when enters the radiation area.

10. What are the diagnostic properties of X-rays?

1. Travel in straight line.
2. Penetrate solid, liquid, and gases.
3. Can produce image on photographic plate.
4. Cause biological changes in living cells.
5. They show the property of fluorescence, ionization, and absorption.

11. What are different uses of X-rays?

- **Radiography:** The main use of X-rays is in medicine. A common application is in the form of X-ray machines, which take photos of a patient's body and teeth. X-rays easily

penetrate into low density substances but do not pass through high density objects like bone. This property helps to find the diseases like TB of lungs and fracture of bone.

- **Radiotherapy:** X-rays can also be used to kill cancer cells, but also kill healthy cells, so must be used with much care. In cancer like diseases it is allowed to fall on affected part of the body.
- **In detective department:** X-rays are used to detect contraband goods like gold, arm, etc. Hidden in sealed boxes or in body. Other uses are in industry, at airports to check customers and baggage.
- **In engineering:** X-rays are used to detect cracks, flaws and air bubbles in the iron girders fitted in buildings and bridges. Accidents may be avoided by replacing these defective girders.
- **In trade:** X-rays are used to distinguish artificial diamonds from real diamonds, presence of pearls in oysters and to test the homogeneity of rubber, and wool, etc.
- **Fluoroscopy:** This allows doctors to see moving images of the internal structures of patients. The patient stands between an X-ray source and a fluorescent screen and a large number of images are taken to form a short film. These are often used to investigate problems with the digestive system.
- **In laboratories, spectroscopy and crystallography:** X-ray diffraction is also very important in spectroscopy and as a basis for X-ray crystallography. The diffraction of X-rays by a crystal where the wavelength of X-rays is comparable in size to the distances between atoms in most crystals is used to disperse X-rays in a spectrometer and to determine the structure of crystals or molecule.
- **Sterilization:** For tinned foods.

12. What are Grenz rays?

These are also called 'Bucky rays'. These were discovered by Sir Gustav Bucky in 1923. Grenz is German word for boundary and refers to its position in the spectrum between the UV rays and ordinary X-rays in electromagnetic spectrum. These are low energy X-rays and are produced by X-ray machines operating 10–20 kV. These can be produced by special Grenz machines. The HVL is less than 0.035 mm Al. They are essentially absorbed between first 2 mm of skin. They are used for variety of inflammatory skin disorder.

13. Which factors are concerned in X-ray measurement?

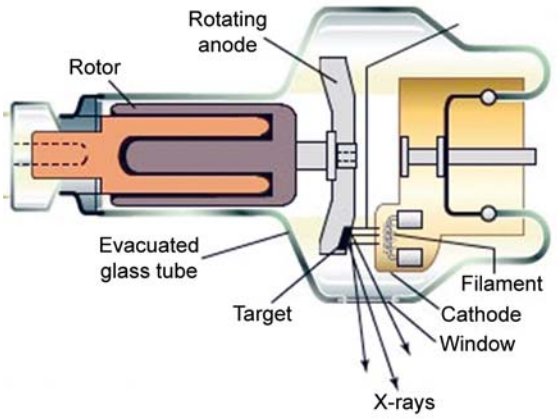
The X-ray spectrums concerned with the following four physical quantities in measurement of X-rays:

1. Frequency
2. Wavelength
3. Energy
4. Intensity

14. What is anode? What are different types of anodes? What are functions of anode?

The positive terminal of an X-ray tube is called the 'anode'. Anodes are of two types:

1. **Stationary anode:** Square/rectangular plate of tungsten 2/3 mm thick embedded in large copper.
2. **Rotatory anode:** The purpose of rotating anode is to spread the heat produced during an exposure over the large area of anode as it increases the total target area. It serves three important functions: (i) It provides a complete circuit for purposes of accelerating the electrons, (ii) it houses the target material, and (iii) it helps to cool the tube.



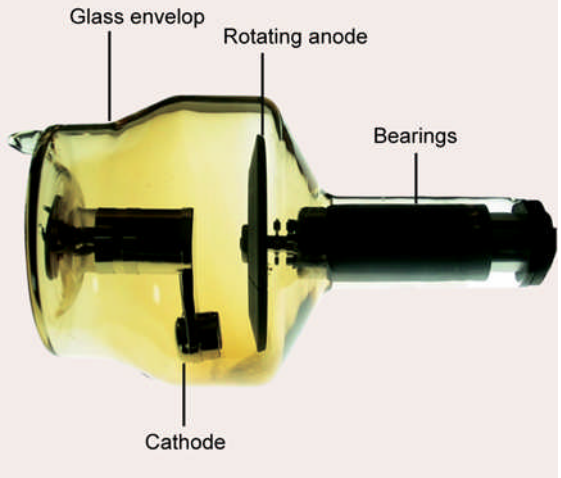
15. What is cathode? What are the elements of cathode and functions of each?

Cathode is negative terminal of X-ray tube. There are two elements of cathode:

1. Filament
2. Connecting wires. The cathode may be referred to as filament.

Filament serves as source of electron. The filament is made up of tungsten wire 0.2 mm diameter that is coiled to form a vertical spiral of 0.2 cm diameter and 1 cm or less in length.

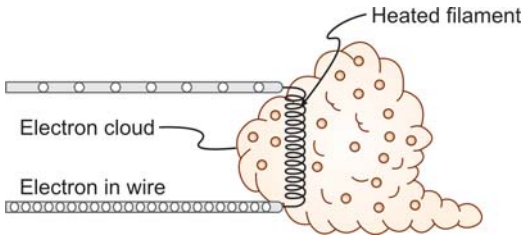
Connecting wires supply voltage (10 V) and amperage.



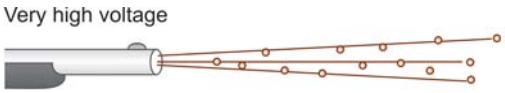
16. Enumerate the factors that are needed for production of X-rays.

The things needed for production of X-rays are:

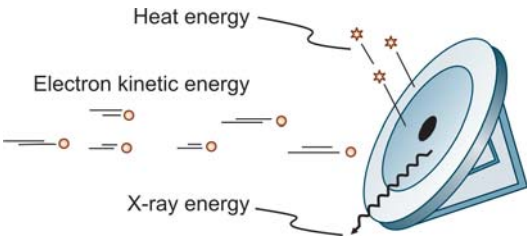
1. Source of electron
2. Motion—high voltage potential to move electrons,
3. Sudden stop.



Source of electrons



Motion



Sudden stop

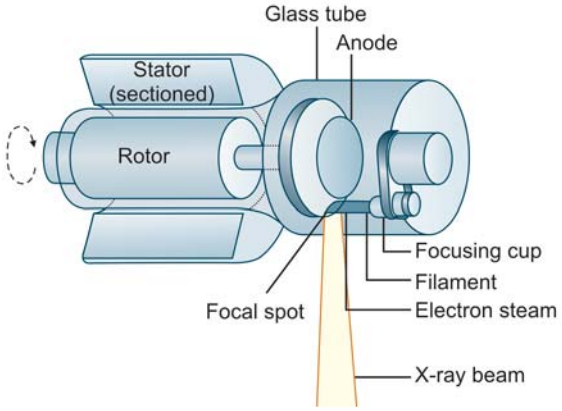
Electrons striking an anode target of high atomic number causes sudden deceleration or stoppage of rapidly moving electrons, which produces heat and X-ray energy.

17. What is Edison effect?

The tungsten wire is heated some electrons in the metal acquire enough energy that moves them from the surface of metal. This escape is thermionic emission. The electron cloud surrounding the filament produced by thermionic emission is called Edison effect.

18. What is stator and rotor?

The stator: The stator is the induction-motor electromagnet that turns the anode and is the only part of the cathode or anode assemblies that is located outside the vacuum of the glass envelope. If the stator fails, the rotor will cease to turn the anode, resulting in immediate



melting of a spot on the target that will damage the anode.

The rotor: The rotor is located inside the stator and inside the glass envelope. It is composed of a hollow copper cylinder attached to the anode disk and affected by the electromagnetic field of the stator, causing the anode to turn.

19. What is line focus principle?

The Benson line focus principle (angle of truncation) explains the relationship between the anode surface and the effective focal spot size. It is developed in 1918.

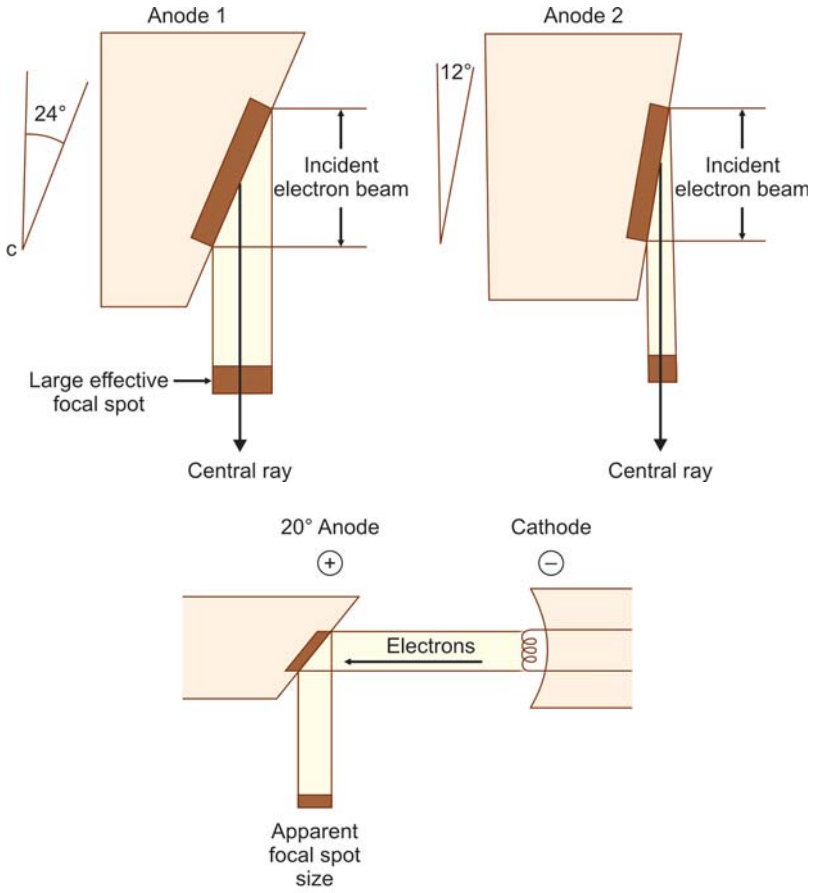
Tilting the anode surface so that there is an angle between the surface and the X-ray beam spreads the heat over a larger area while maintain the smaller focal spot for the sharper images is called 'line focus principle'.

The focal spot is the area of the target upon which the electron beam impinges and actual size is 1 × 3 mm^2. The anode is angled at 20° to the central ray of electron beam (it may vary from 6° to 20° according to various tube manufactures), making the effective focal spot smaller, i.e. 1 × 1 mm^2. If the anode angle is made smaller, the effective focal spot is smaller. The effective focal spot is the beam projected onto the patient. As the anode angle decreases, the effective focal spot decreases.

Thus, the line focus principle helps to resolve the following issues:

1. Heat dissipation, and
2. Sharp image by having smaller effective focal spot with heat capacity of large focal spot.

The limitations of line focus principle are the area covered by the beam reduces with the target angle and anode heel effect.

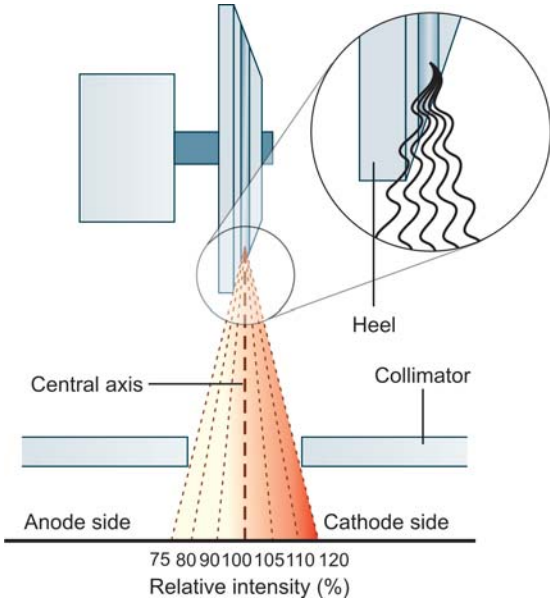


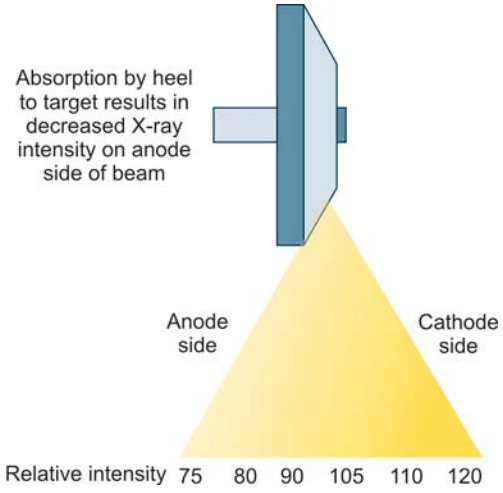
20. What is heel effect?

X-rays produced within anode travel in all directions (isotropic) and intensity of emergent beam is not uniform because some part is absorbed in focal spot itself. X-rays produced within anode must pass through a portion of target and are therefore attenuated on their way out of target. The target is usually tungsten (Z = 74), which has attenuation properties similar to lead (Z = 82), the attenuation is greater in anode direction than cathode direction because of the difference in the path length within target. This is called 'heel effect' (results in higher X-ray intensity at cathode end and lower intensity at anode end).

The magnitude of heel effect depends on anode angle, source to image detector (SID) distance and field size. To reduce heel effect the anode angel is increased, SID is increased and field size is decreased. The effect is increased when the target angle is reduced.

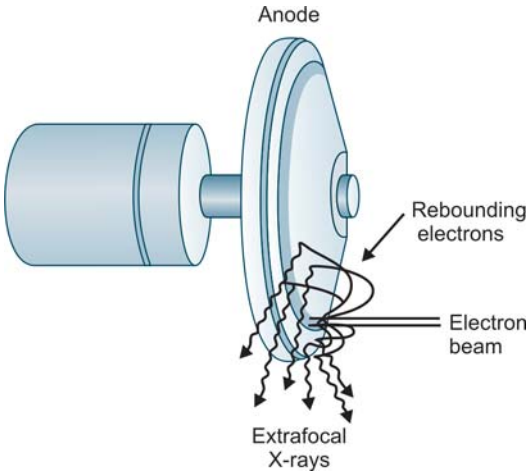
The heel effect can be taken advantage by keeping the denser part of body towards cathode side and thinner side towards anode side.





21. What is off focal radiation?

The electrons can rebound and interact with other areas of anode other than focal areas. These areas can produce X-rays too. This is called off focal radiation. A diaphragm is placed between the tube and collimator reduces off focal radiation.

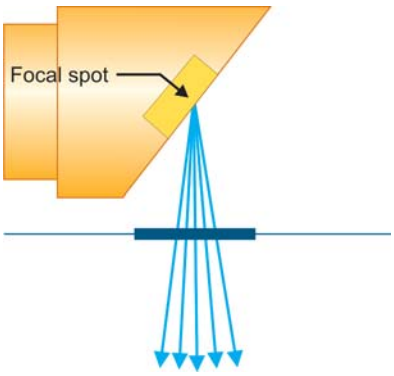


22. What is focal spot?

The focal spot is the area on the target to which the focusing cup directs the electrons from the filament.

or

The focal spot is the area on the target to which the focusing cup directs the electrons and from which X-rays are produced. Orit is the apparent source of X-rays from the tube.



23. What factors determine the size of focal spot?

Size and also shape of focal spot is determined by the size and shape of the electron beam which is ultimately determined by filament shape, dimensions of filament tungsten coil, position of filament in focusing cup, construction of focusing cup and electric field created between anode and cathode are the factors for size of focal spot. If the focal spot is too big, sharpness of image will be lost while if the focal spot is too small, then the anode will get too hot.

24. What are the characteristics of X-ray beam?

Quantity of beam and quality of beam are the two terms describes characteristics of beam. Quantity describes the number of photons while quality describes the energy of photons.

25. What is intensity of X-ray beams?

It is defined as number of photons in the beam multiplied by energy of each photon in beam. The intensity is measured in roentgen per minute (R/min, or C/kg in SI system). It depends upon the kVp, tube current, target material, and filtration.

26. Why tungsten is used as a target in X-ray tube?

The target is made of tungsten, a material that has several characteristics of an ideal target material.

1. It has a high atomic number (74) which makes it more efficient for production of X-rays.
2. High melting point (3370°C) which helps to withstand the high temperature produced.
3. High thermal conductivity which helps in dissipating heat into copper stem.
4. Low vapour pressure at high temperatures helps in maintaining the vacuum in the tube at high operating temperatures.
5. All metals expand when heated but at different rates. The bonding between tungsten and copper provides technical problems as the coefficient of expansion for both metals are different. Ultimately, it is a good material for absorption of heat and for rapid dissipation of heat from the target area.

27. What are the other materials used in anode?

In addition to tungsten, molybdenum or graphite are layered under the tungsten target. These materials are less dense materials than tungsten which makes easier to rotate anode. Molybdenum also has high melting point while graphite backed anodes can double the heat loading capacities without increasing wear to bearings.

28. What is space charge and space charge effect?

Space charge: The electron emitted from the tungsten filament in the immediate vicinity of filament form a small cloud, this collection of negatively charged electrons forms space charge.

Space charge effect: The cloud of negatively charged electrons tends to prevent the other electrons from being emitted from the filament until they have acquired sufficient thermal energy to overcome the force caused by the space charge. This tendency of space charge to limit the emission of more electrons from the filament is called 'space charge effect'. The space charge effect limits X-ray tubes to maximum mA range of 1,000 to 1,200.

29. What is transformer? What is step up, step down, and autotransformers?

An electromagnetic device for changing the alternating current coming into the machine. Required to decrease or increase the ordinary 100 V current that enters the X-ray machine.

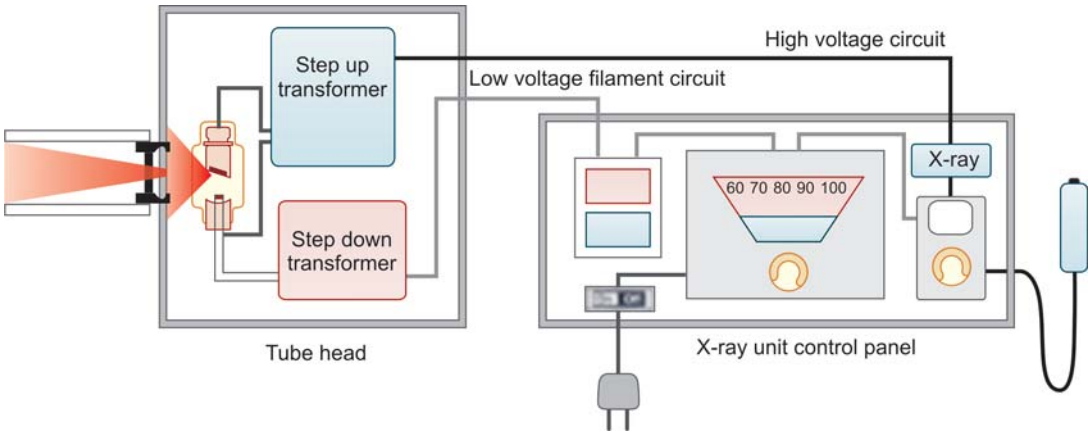
Step down transformer: This decreases the voltage from the wall outlet to approximately just enough to heat the element and form an electron cloud.

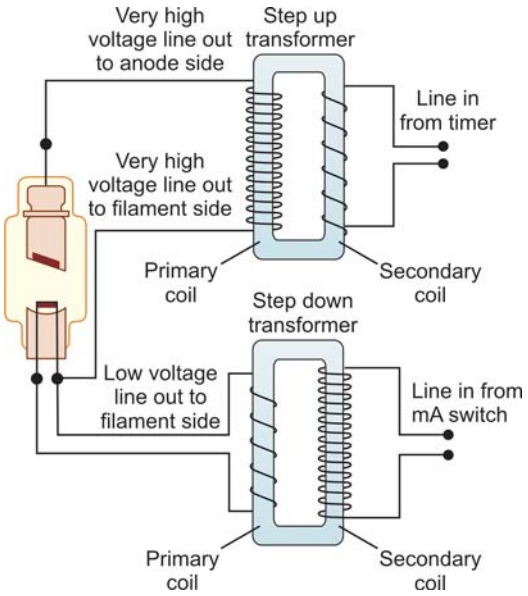
Step up transformer: Increases voltage as required. Begins to flow through the cathode anode circuit when the exposure button is pressed. Located in tube head.

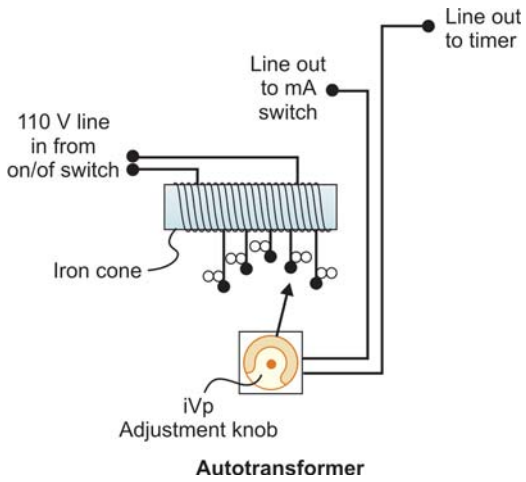
Autotransformer: A voltage compensator corrects minor fluctuations in current flowing through the wires.

30. What is attenuation of X-ray?

Attenuation is the reduction in the intensity of X-ray beam as it traverses matter by either absorption or deflection of photons from the beam. It depends on quality and quantity of photons in X-ray beam. The intensity of X-ray beam is the product of quantity and energy of photons so it depends on both quality and quantity of beam.







31. What is beam hardening effect?

It refers to the effect of filter on a polychromatic X-ray beam containing a range of X-ray photon energies. Beam energy is the preferential loss of low energy photons. It does not occur with monochromatic X-ray beams.

32. What are beam restrictors?

Beam restrictors are the device attached to the X-ray tube housing, to regulate the size and shape of X-ray beam. They are:

1. Aperture diaphragms (sheet of lead with a hole)
2. Cones and cylinder
3. Collimators.

33. What are the factors controlling X-ray beam?

X-ray beam may be modified by altering the beam exposure duration (timer), exposure rate (mA), energy (kVp and filtration), shape (collimation) and intensity (target–patient distance), these can be divided into qualitative and quantitative factors.

1. **Tube voltage (kVp):** As kVp increases, it results in increase in efficiency of converting electron energy into photons. Increasing kVp will increase: (i) The number of photons generated, (ii) mean energy of photons, (iii) maximal energy of photons. High energy (short wavelength) are preferred as they have greater penetrating power—qualitative and quantitative.
2. **Exposure time(s):** When the time of exposure is doubled, the number of photons generated are doubled. Thus, it increases the number of photons and controls the quantity of exposure. The range of photon energy is not changed—quantitative.
3. **Tube current (mA):** It is directly proportional, so doubling the tube current will double the number of photons produced. The quantity is increased—quantitative.
4. **Filtration:** Aluminium filters used will remove the long wavelength photon with less penetrating power—qualitative and quantitative (reduces the quantity).
5. **Collimation:** When the X-rays are directed at patient only 10% give useful information while 90% are responsible for scattered radiation. Scattered radiation produces fog. Collimation reduces size of X-ray beam and ultimately the volume of irradiated tissue—qualitative and quantitative.
6. **Target–patient distance:** Intensity of X-ray beam (the number of photons per cross-sectional area) at a given point is inversely proportional to square of distance from the source of radiation—quantitative.

The factors found on X-ray machine are:

1. Tube voltage
2. Exposure time
3. Tube current

34. What is inverse square law?

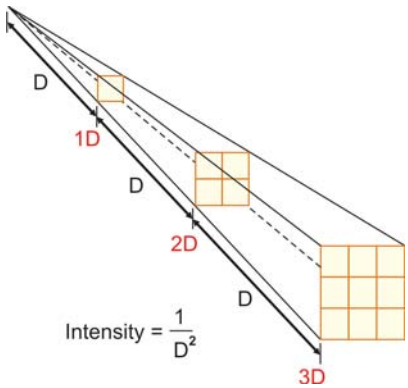
It states that the intensity of radiation is inversely proportional to the square of the distance from the source of radiation. The intensity of an X-ray beam at a given point (number of photons per cross-sectional area per unit exposure time) depends on the distance of the measuring device from the local spot.

The reason for this decrease in intensity is that the X-ray beam spreads out as it moves from the source.

The relationship is as follows:

$$\frac{I_1}{I_2} = \frac{(D_2)^2}{(D_1)^2}$$

where I is intensity and D is distance. Therefore, if a dose of 1 gray (Gy) is measured at a distance of 2 m, a dose of 4 Gy will be found at 1 m, and 0.25 Gy at 4 m.



Other example is position indicating device (PID) is changed from 8 inches to 16 inches, the source to receptor distance is doubled and the resultant beam is one-fourth as intense and if the PID is changed from 16 inches to 8 inches, the resultant beam is four times more intense.

35. What is filtration?

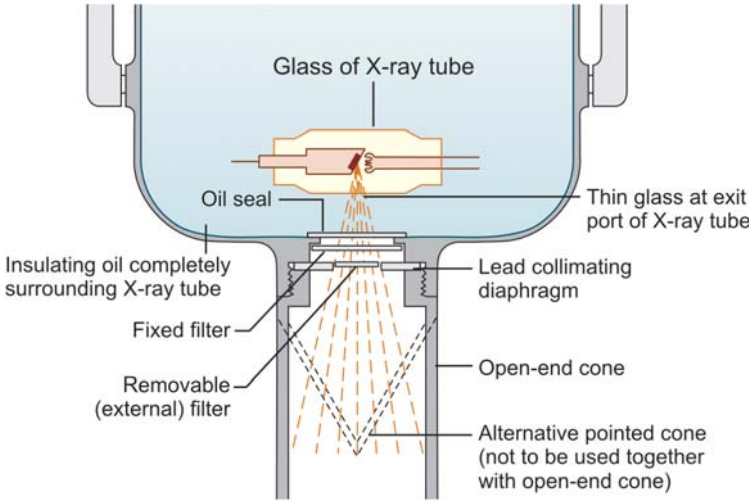
The removal of soft, low energy, long wavelength X-rays from beam is called filtration.

Although X-ray beam consists of a spectrum of X-ray photons of different energies, only photons with sufficient energy to penetrate through anatomic structures reach the image receptor (usually film) are useful for diagnostic radiology. Those that are of low energy (long wavelength) contribute to patient exposure (and risk) but do not have enough energy to reach the film.

Purpose of filtration is to remove soft, low energy, long wavelength X-rays from the beam.

Consequently, to reduce patient dose, the less-penetrating photons should be removed. This can be accomplished, in part, by placing an aluminium filter in the path of the beam. The aluminium preferentially removes many of the lower energy photons with lesser effect on the higher-energy photons that are able to penetrate to the film. In determination of the amount of filtration required for a particular X-ray machine, kVp and inherent filtration of the tube and its housing must be considered.

Inherent filtration consists of the materials that X-ray photons encounter as they travel from the focal spot on the target to form the usable beam outside the tube enclosure. These

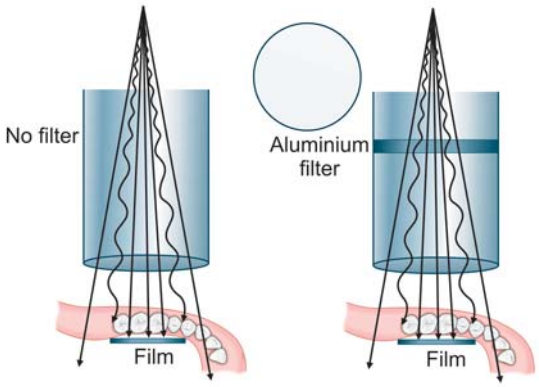


materials include the glass wall of the X-ray tube, the insulating oil that surrounds many dental tubes, and the barrier material that prevents the oil from escaping through the X-ray port. The inherent filtration of most X-ray machines ranges from the equivalent of 0.5 to 2 mm of aluminium.

Total filtration is the sum of the inherent filtration plus any added external filtration supplied in the form of aluminium disks placed over the port in the head of the X-ray machine. Governmental regulations require the total filtration in the path of a dental X-ray beam to be equal to the equivalent of 1.5 mm of aluminium to 70 kVp, and 2.5 mm of aluminium for all higher voltages.

Aluminium and copper are used in compound filter.

A dental X-ray machine operating at 50 to 69 kVp should have at least 1.5 mm equivalent Al filtration, whereas machine operating 70 kVp or more should have 2.5 mm Al equivalent filtration.



36. What is collimation and collimator?

Collimation is the term used to indicate shaping of the X-ray coming from the tube head into a column or beam of X-ray.

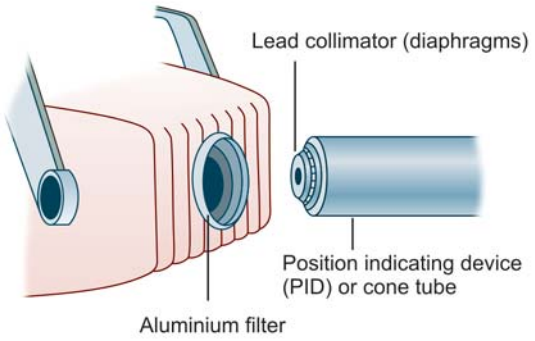
or

It is process of confining and restricting the X-ray beam to given area.

or

Collimation means restriction of the cross sectional area of the beam.

A collimator is beam restrictor. It defines the size and shape of X-ray field that emerges from X-ray tube. A collimator is a metallic barrier with an aperture in the middle used to reduce the size of the X-ray beam and therefore the



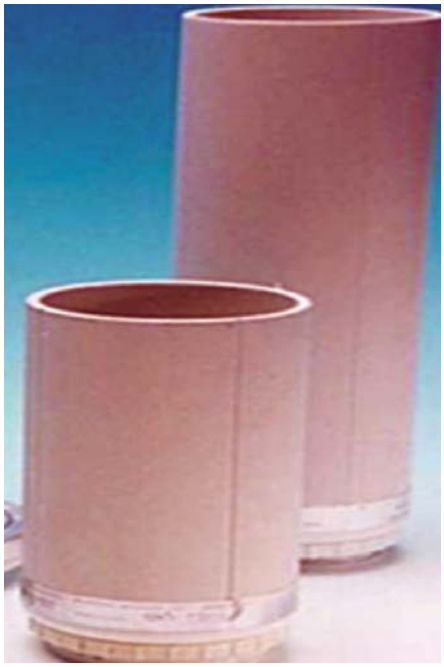
volume of irradiated tissue within the patient. Collimation does not change the energy or number of X-rays in X-ray beam that reach the film. It just limits size and shape of beam.

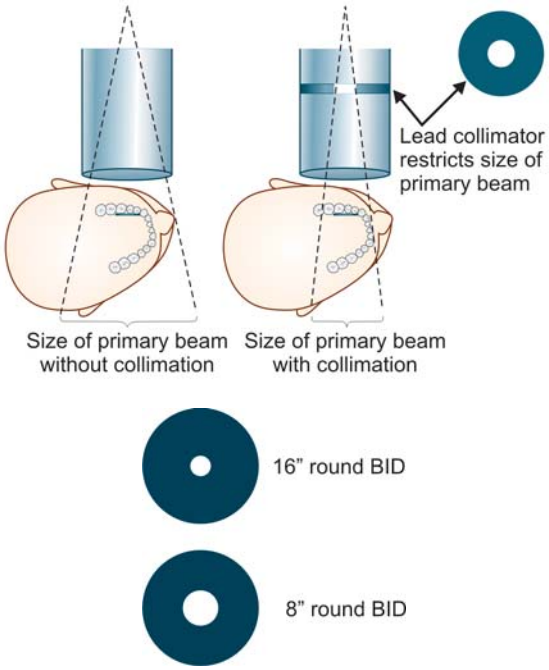
Round and rectangular collimators are most frequently used in dentistry. Dental X-ray beams are usually collimated to a circle 23/4 inches (7 cm) in diameter or it is collimated such that useful beam does not exceed 2.75 inches in diameter. Its functions are as follows:

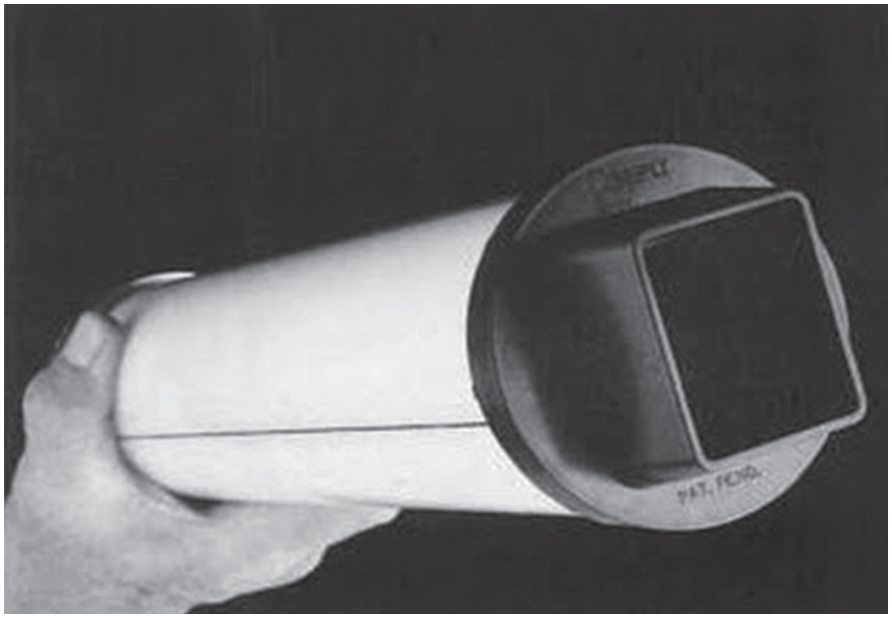
1. The size of film is reduced, ultimately the volume of irradiated tissue and indirectly reducing the scattered radiation.
2. Reduces exposure by reducing the size of beam.
3. Film quality is increased by improving the contrast.

- A round collimator (diaphragm) is a thick plate of radiopaque material (usually lead) with a circular opening centred over the port in the X-ray head through which the X-ray beam emerges. Typically, round collimators are built into open-ended aiming cylinders.

  It produces a beam that is 2.75 inches in diameter, considerable larger than size of 2 films. Main disadvantage is typically it produces large pneumbra at periphery of beam.
- Rectangular collimators further limit the size of the beam to just larger than the size







Dentsply rinn universal collimator attached to long round BID

of 2 X-ray films. It is important to reduce the beam to the size of the film to reduce further unnecessary patient exposure.

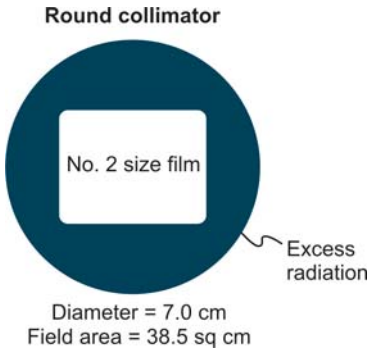
Some types of film-holding instruments also provide rectangular collimation of the X-ray beam. A rectangular collimator exposes 60% less tissue than circular collimator.

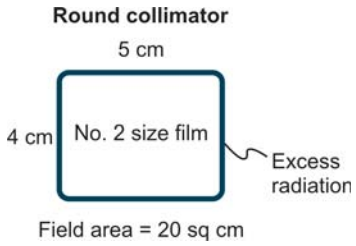
- Tubular collimator is simply a tube lined with or a radiopaque material.
- Slit collimator used in OPG machine.

Margraph long rectangular BID

Collimator side—left long, round BID. Middle short round BID, right rectangular, long BID smallest

Rectangular collimation showing less radiation exposure than round collimator.
Rectangular collimation reduces the dose fivefold to circular one.





37. What are the uses of aluminium in X-ray system?

Aluminium is used as:

1. It is used material to filter X-ray beam.
2. As a reference material to measure the penetrating ability (HVL) of X-rays.

38. What is HVL and TVL?

HVL (half value layer) is that thickness of specified material which will reduce the intensity of X-radiation to half its original value. Increase in penetrating ability of a radiation increases its HVL. It is also called half value layer thickness, half value thickness (HVT).

TVL is the thickness of stated medium that will reduce the intensity of narrow beam of X-rays to exactly one-tenth of its original value.

39. What is timer? What are different types of timers?

Timer is the device into high voltage circuit to control the duration of X-ray exposures (length of exposure). The switch is like dead man type which means it has to be continuously depressed for X-ray production.

There are four types of exposure timers:

1. **Mechanical timers:** Mainly used in single phase generator circuit. The accuracy and reproducibility is poor rarely used today.
2. **Electronic timers:** The length of exposure is determined by the time required to change the capacitor through the resistance. The electronic timer controls the length of time that the high voltage is applied to the tube and therefore the time during which tube current flows and X-rays are produced. It is set by turning the selector knob, depressing the marked push button or touching a keypad.

3. **Photo timers:** The actual amount of radiation transmitted through the patient is measured. It terminates the exposure when the voltage receptor has received a preselected X-ray exposure. Three types of photo timers are: (i) Ionization chamber, (ii) solid state diodes, (iii) scintillations with photomultiplier tube.
4. **Pulse counting timers:** It uses technology of voltage pulse counting to control the time of short exposure technique. In this high frequency oscillations are generated by the oscillation in quartz crystal.
5. **mAs timer:** For any particular X-ray tube and kV the radiation exposure is controlled by tube current and exposure time. The mAs timer does it automatically and it terminates the exposure as soon as the desired mAs has passed through the X-ray tube and into the condenser.

The timer serves to regulate the duration of the interval that the current will pass through the X-ray tube.

Time settings of less than a second may be indicated on fractions and impulses.

New machines are accurate to 1/100 sec intervals. Time determines the duration of exposure.

40. Enumerate various types of intraoral films and the dimension of each film pack.

Periapical View

Periapical views are used to record the crowns, roots, and surrounding bone. Film packs come in five sizes:

1. For small children (22 × 35 mm);
2. Which is relatively narrow and used for views of the anterior teeth, adult anterior (24 × 40 mm);
3. The standard film size used for adults (31 × 41 mm).
4. Relatively long size bitewing (54 × 27 mm)
5. Occlusal film (57 × 76 mm).

Bitewing View

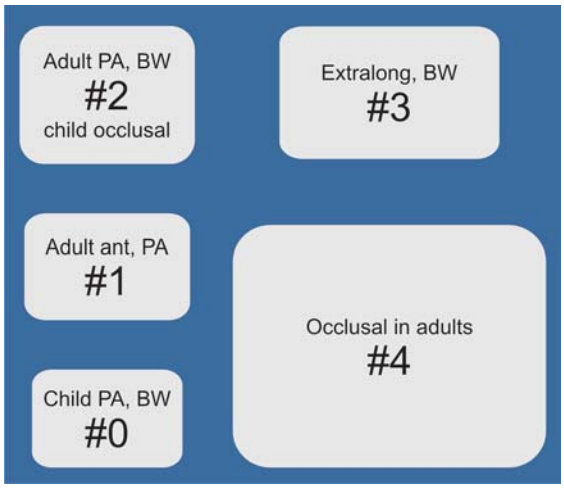
Bitewing (interproximal) views are used to record the coronal portions of the maxillary and mandibular teeth in one image. Size 2 film is normally used in adults; the smaller size 1 is preferred in children. In small children, size 0 may be used. A relatively long size 3 is also available. Bitewing films often have a paper tab projecting from the middle of the film, on which the patient bites to support the film. This tab is rarely visualized and does not interfere with the diagnostic quality of the image. Film-holding instruments for bitewing projections are also available.

Occlusal View

The size 5 film occlusal film is more than three times larger than size 2 film (57 × 76 mm). It is used to show larger areas of the maxilla or mandible than may be seen on a periapical film. These films are also used to obtain right-angle views to the usual periapical view. The name derives from the fact that the film is usually held in position by having the patient bite lightly on it to support it between the occlusal surfaces of the teeth.

Type-size number	*Dimensions*	
	mm	*in*
Periapical		
1.00	21 × 32	4/5 × 11/4
1.0	22 × 35	7/8 × 13/8
1.1	24 × 40	15/16 × 19/16
1.2	31 × 41	11/4 × 15/8
Bitewing (interproximal)		
2.00	21 × 32	4/5 × 11/4
2.0	22 × 35	7/8 × 13/8
2.1	24 × 40	15/16 × 19/16
2.2	31 × 41	11/4 × 15/8
2.3	27 × 54	11/16 × 2
Occlusal		
3.4	57 × 76	21/4 × 3

Note: The digit at the left of the decimal point represents the use of the film (periapical, bitewing, or occlusal). The digits on the right indicate the size of the film (00, 0, 1, 2, 3, or 4)



Size 0: Children's film
Size 1: Narrow anterior periapical film
Size 2: Adult regular for posterior periapical and bitewing
Size 3: Longer in use
Size 4: Occlusal film

41. What are the sizes of bitewing films?

Size 0: (For children) 22 mm × 35 mm

Size 1: For adult (anterior teeth) 24 mm × 40 mm

Size 2: For adult (posterior teeth) 31 mm × 41 mm

Size 3: For adult (for all posterior teeth of one side in one film) 27 × 54 mm.

42. What are the differences between screen and nonscreen films?

Characteristics	Direct exposure film	Indirect exposure film
Exposed by	Only X-rays	Mainly by visible light
Used	Without screens	With screens
Emulsion layer	Thick	Thin
Image formation	In deep	Superficially
Processing time	More	Less
Resolution	More	Less
Characteristic curve	No apparent shoulder region in useful density range	Shoulder region in useful density range
Screen artifact	No	May be possible
Exposure dose	More	Less
Uses	Orbit and extremities radiography and industrial radiography	General radiography

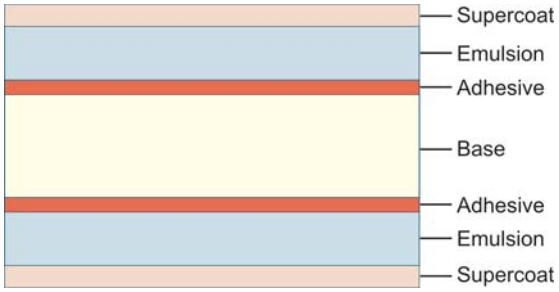
43. What is composition of X-ray film?

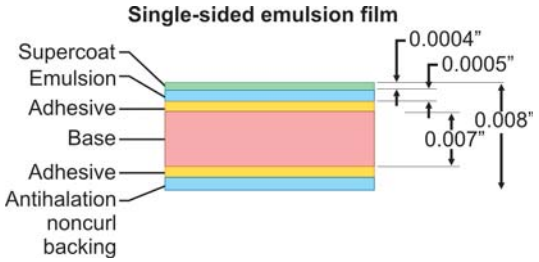
X-ray film has four basic principal components:

1. Film base
2. Adhesive layer
3. Film emulsion
4. Protective layer. The emulsion, which is sensitive to X-rays and visible light, records the radiographic image. The base is a plastic supporting material onto which the emulsion is coated.

Cross-section of Double Emulsion Film

1. **Film base:** It is a flexible piece of polyester plastic (polyethylene terephthalate—the thickness is 0.18 to 0.2 mm or 0.007 inches), the function of the film base is to support the emulsion. The base must have the





proper degree of flexibility to allow easy handling of the film. The semi-clear film base (cellulose acetate) is uniformly translucent and casts no pattern on the resultant radiograph. Some believe that a base with a slight blue tintometrics image quality by emphasizing the contrast. The film base must also withstand exposure to processing solutions without becoming distorted and do promote easy handling and provides strength.

2. **Adhesive layer (subcoating, substratum, subbing layer):** A thin layer added on both sides of film before emulsion is added and serves to adhere the emulsion and to base. This layer is mixture of gelatin and film base.
3. **Film emulsion:** It is homogeneous mixture of silver halide grains (sensitive to X-radiation and visible light), and a vehicle matrix (gelatin and nongelatin material). Each emulsion layer is about one thousand of an inch thickness.
   a. *Halide crystals:* Halides are compounds of halogen such as chlorine, bromine, and iodine. The silver halide grains are composed primarily of crystals of silver bromide in dental X-ray films (80–99%) and to a lesser extent silver iodide (1–10%). During radiation exposure the silver halide crystals store energy from which they have been exposed and react with the chemicals in the processing tank to form a black (radiolucent) region on the film. Those crystals which are not been stuck they wash off during processing. Iodide is added to ultraspeed film because its large diameter (compared with bromine) disrupts the regularity of the silver bromide crystal structure, thereby increasing its sensitivity to X-radiation. The photosensitivity of the silver halide crystals also depends on the presence of trace amounts of a sulphur-containing compound. In addition, trace amounts of gold are sometimes added to silver halide crystals to improve their sensitivity. In the manufacture of film, the silver halide grains are suspended in a surrounding vehicle that is applied to both sides of the supporting base. The vehicle composed of gelatinous and nongelatinous materials, keeps the silver halide grains evenly dispersed. To ensure good adhesion of the emulsion to the film base, a thin layer of adhesive material is added to the base before the emulsion is applied. During film processing, the vehicle absorbs the processing solutions, allowing the chemicals to reach, and react with the silver halide grains.
   b. *Gelatin:* It must be uniform in thickness (0.0002–0.0004 inch 5–10 Mu microns). It is composed of cattle bone and used to suspend and disperse the millions of microscopic halide crystals over base during film processing. Gelatin absorbs the silver halide crystals and allows the chemicals to enter and react with halide crystals.
4. **Protective coat (super coat, antiabrasive layer):** An additional thin layer of vehicle (tough coating of hard protective gelatin) is added to the film emulsion as an overcoat; this barrier helps protect the film from damage by scratching, contamination, or pressure from rollers when an automatic

processor is used. It is also less likely to pick-up dirt. It is treated to achieve anti-static behaviour. It should not be too hard to prevent penetration.
Film base is 0.007 inch while protective coat. Emulsion and adhesive layer on either side is 0.0005 inch.

44. What is coating weight?

Coating weight is the amount of silver halide crystals in emulsion. The screen and non-screen films consist of silver bromide as main constituent. Nonscreen films have higher coating weight than screen film. The consideration is important to obtain silver in recovery process.

45. What is safe film?

The film base is made up of acetate polyester which is not easily inflammable, then the film is called safe film or safety film.

46. What are the differences between single emulsion film and double emulsion film?

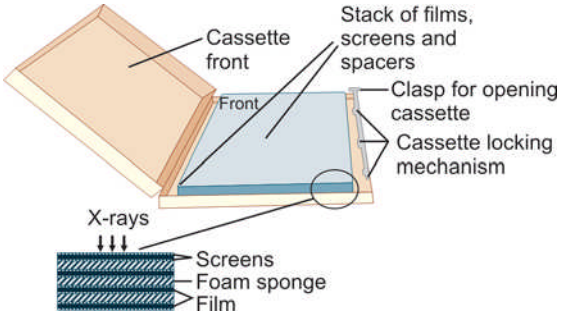
Characteristic	*Single coated*	*Double coated*
Emulsion layer	One side	Both side
Patient radiation dose	More	Less
Noncurl back layer	Present	Absent
Radiographic detail	More	Less
Average gradient (G)	Very less	More
Parallax effect	No	Yes
Contrast	Less	More

47. What is cassette? What are functions of cassettes?

A flat, light-tight container in which X-ray films are placed for exposure to ionizing radiation and usually backed by lead to eliminate the effects of back scatter radiation, containing front and back intensifying screens.

Cassettes are used in association with intensifying screens and screen films. They have related functions:

1. To contain a film
2. To exclude light



3. To maintain the film in close, uniform contact with both screens during the exposure.
4. To protect the intensifying screens from physical damage.

48. What are the properties of cassette? How the care of cassette is taken?

The properties are:

a. **Weight:** It should be light for easy manipulation.

b. **Robust structure:** Cassettes in daily use are subject to considerable stress and wear. Screens may fail to maintain contact with the film or leakage of light at the edges can occur. Cassettes deserve and should have stringent care in handling.

c. i. *Nonflexible:* So as not to allow the film to bend.

ii. *Flexible cassettes:* For panoramic machines. Flexible cassettes are necessary for the specialized equipment associated with panoramic radiography. They are mounted within a simple envelope of plastic material, folded at one end and fastened with press buttons or velcro of conventional design. The cassette is attached to a drum and is rigid for the duration of the exposure.

d. **Size:** Slightly larger than the X-ray beam and area to be radiographed.

e. Ease of operation.

49. How the care of cassettes is taken?

Treated with care X-ray cassettes and intensifying screens are good for years of hard work.

Their general care is aimed at the avoidance of rough handling by all who use them.

It is helpful to mark each cassette with identifying numerals which are inconspicuous, that makes it easy to eliminate, if radiographic faults are observed, ascribable to damage of some kind, e.g. cracks in the intensifying screens or light leaks. Screens come with a sticker indicating the film speed and this sticker is placed on the outside of the cassette.

50. Enumerate the errors in loading and uses of cassette and useful hints to avoid them.

Problem	*Cause*	*How to correct*	*Useful hints*
Overall grayness or blackness along one edge or corner of film (fog)	Damaged cassette (light leak) or film exposed to light	Tape edges of soft cassette, replace damaged hard cassette	Cassettes should be inspected regularly for light tightness
Little or no image is visible on film	Screens reversed	Replace screens properly	Dull surface of screen should face film, not shiny
White streaks on image	Damaged (scratched) screens	Handle screens carefully	Use screen cleaning solutions and soft cloth to clean screens
Black marks, round clusters or lightning bolt of film from cassette	Static electricity	Avoid too rapid removal	Use of antistatic mats or humidifier can reduce static
Multiple images	Double exposure	Remove film from cassette after each exposure	Store unexposed and exposed cassettes separately

51. What are intensifying screens?

Early in the history of radiography, scientists discovered that various inorganic salts or phosphors fluorescence (emit visible light) when exposed to an X-ray beam. The intensity of this fluorescence is proportional to the X-ray energy absorbed. These phosphors have been incorporated into intensifying screens for use with screen film. The sum of the effects of the X-rays and the visible light emitted by the screen phosphors exposes the film in an intensifying cassette.

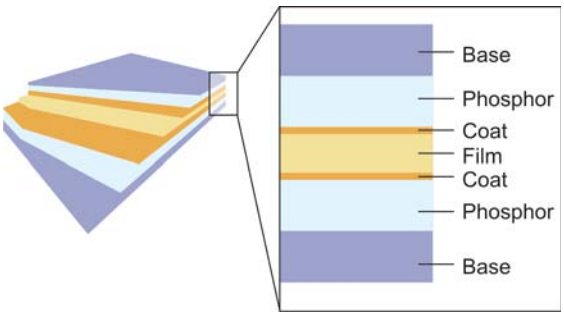
Function

The presence of intensifying screens creates an image receptor system that is 10 to 60 times more sensitive to X-rays than the film alone. Consequently, use of intensifying screens means a substantial reduction in the dose of X-radiation to which the patient is exposed.

Intensifying screens are used with films for virtually all extraoral radiography, including panoramic, cephalometric, and skull projections. In general, the resolving power of screens is related to their speed: The slower the speed of a screen, the greater is its resolving power and vice versa. Intensifying screens are not used intraorally with periapical or occlusal films because their use would reduce the resolution of the resulting image below that necessary for diagnosis of much dental disease.

Composition

Intensifying screens are made of a base supporting material, a phosphor layer, and a protective polymeric coat. In all dental applications, intensifying screens are used in pairs, one on each side of the film, and they are positioned



inside a cassette. The purpose of a cassette is to hold each intensifying screening contact with the X-ray film to maximize the sharpness of the image. Most cassettes are rigid, but they may be flexible.

Base

The base material is mostly of card, polyester and plastic that is about 0.25 mm thick. The base provides mechanical support for the other layers. In some intensifying screens the base is also reflective; thus it reflects light emitted from the phosphor layer back toward the X-ray film. This has the effect of increasing the light emission of the intensifying screen. However, it also results in some image "unsharpness" because of the divergence of light rays reflected back to the film. Some fine detail intensifying screens omit the reflecting layer to improve image sharpness. In other intensifying screens the base is not reflective, and a separate coating of titanium dioxide is applied to the base material to serve as a reflecting layer.

Phosphor Layer

The phosphor layer is composed of phosphorescent crystals suspended in a polymeric binder. When the crystals absorb X-ray photons, they fluorescence. The phosphor crystals often contain rare earth elements, most commonly lanthanum and gadolinium. Their fluorescence can be increased by the addition of small amounts of elements such as thulium, niobium, or terbium. Some rare earth compounds are efficient phosphors. In the energy range typically used in dental radiography, a pair of rare earth intensifying screens absorbs about 60% of the photons that reach the cassette after passing through a patient. These phosphors are about 18% efficient in converting this X-ray energy to visible light. Rare earth screens convert each absorbed X-ray photon into about 4,000 lower-energy, visible light expose the film.

Different phosphors fluorescent in different portions of the spectrum. It is important to match green-emitting screens with green-sensitive films and blue-emitting screens with blue-sensitive films. The speed and resolution of a screen depend on many factors including:

- Phosphor type and phosphor conversion efficiency
- Thickness of phosphor layer and coating weight (amount of phosphor/unit volume)
- Presence of reflective layer
- Presence of light-absorbing dye in phosphor binder or protective coating
- Phosphor grain size.

Fast screens have large phosphor crystals and efficiently convert X-ray photons to visible light but produce images with lower resolution. As the size of the crystals or the thickness of the screen decreases, the speed of the screen also declines but image sharpness increases. Fast screens also have a thicker phosphor layer and a reflective layer, but these properties also decrease sharpness. In deciding on the combination to use, the practitioner must consider the resolution requirements of the task for which the image will be used. Most dental extraoral diagnostic tasks can be accomplished with screen-film combinations that have a speed of 400 or faster.

Protective Coat (super coat)

A protective polymer coat (up to 15 Jlm thick) is placed over the phosphor layer to protect the phosphor and provide a surface that can be cleaned. A strong, smooth, protective layer of cellulose acetate is used. The intensifying screens should be kept clean because any debris, spots, or scratches may cause light spots on the resultant radiograph. This layer is—resistant to abrasion, moisture protecting the fluorescent layer, minimal thickness reduces image sharpness.

52. How the care of intensifying screens is taken?

Screens are easily damaged. Their fluorescent emission will be affected if the active surface is soiled even slightly. Screens must thus be kept clean, otherwise light photons will be prevented from reaching the screen and creating an image and the screen in that area

will appear clear. Dirt will also create "high" spots which will create wear. Screens are best cleaned with antistatic solution (at least once in 6 months). Use a damp cloth and rub gently. Ensure that the screen is dry before closing the cassette, otherwise the gelatin on the surface of the screens will stick together. Never leave the cassette open as it will accumulate dirt and dust on the screen. Avoid touching of screen and keep the film processing area free of dirt. Check for proper screen film contact before exposure. The foam supporting the screens should be intact and is capable of holding screen closely with films.

Procedure

1. Choose a clean location to clean screens.
2. Moisten a lint-free wipe with a small amount of commercially available screen cleaners and antistatic solutions. Mild soap and water solution and 70% of isopropyl alcohol may be used as an alternative, but screen cleaners and antistatic solutions must be used.
3. Clean, dry screen avoid excess pressure.
4. Use a second lint-free wipe inside and clean the frame (single screen).
5. Stand the cassettes on edge to dry.
6. UV light is used to inspect dust, dirt particles.

Regular Inspection

1. The screens in a flat cassette may come loose and should be reattached immediately. Loose screens are an invitation to error in the darkroom. It is easy, when loading a cassette, to slip the film on top of both screen if they are unattached.
2. The felt pad or foam rubber in the back of the cassette may have become insecure or worn. This can result in failure of the intensifying screens to maintain uniform contact with the film and this causes a localized area of unsharpness on the radiograph, due to the spread of fluorescent light between the screens and the emulsion. There is nothing that can be done for a cassette which is failing to maintain contact between the intensifying screens.
3. Screens which are old or cracked can be seen to have fairly mottled appearance and this will be reproduced on radiographs. When this is noticed it is time to discard the screens.

53. What are the speeds of intensifying screens?

1. **Fast screens (lanex):** Rare earth thick layer, and relatively large crystals used, maximum speed is attained but with some sacrifice in definition. They use gadolinium or lanthum phosphors.
2. **Slow screens:** Standard slow screens or high definition screens—a thin layer and relatively small crystals are used; detail is the best, but speed is slow necessitating a higher dose of ionizing radiation. They use calcium tungstate phosphors.
3. **Medium screens-standard:** Medium thick layer of medium-sized crystals in order to provide comprise between speed and definition.

54. What are the factors of regular inspection of intensifying screens?

1. Intensifying screens in a flat cassette may come loose and should be reattached immediately. Loose screens are an invitation to error in the darkroom. It is easy, when loading a cassette, to slip the film on top of both screen if they are unattached.
2. The felt pad or foam rubber in the back of the cassette may have become in secure or worn. This can result in failure of the intensifying screens to maintain uniform contact with the film and this causes a localized area of unsharpness on the radiograph, due to the spread of fluorescent light between the screens and the emulsion. There is nothing that can be done for a cassette which is failing to maintain contact between the intensifying screens.
3. Screens which are old or cracked can be seen to have fairly mottled appearance and this will be reproduced on radiographs.

When this is noticed it is time to discard the screens.

As they are sold in pairs, there is a little to be done except to replace both screens in the cassette. (Like one glove on its own, it may subsequently never have a match.)

55. What are functions of intensifying screen?

1. Reduce the patient's exposure to radiation.
2. It reduces blurring of the image due to subject movement by shortening the exposure time.
3. It reduces geometric blur by making it possible to use a small focus X-ray tube.
4. This makes possible use of portable X-ray tube and extend the life of X-ray tube.
5. Increase the contrast of radiograph.

56. What is luminescence?

Luminescence is the emission of light from a substance bombarded by radiation. These are of two types: Fluorescence and phosphorescence.

Fluorescence means that luminescence is excited only during the period of irradiation and will terminate at completion of the X-ray exposure. The phosphors in intensifying screens produce fluorescence.

Phosphorescence is after glow. The irradiated material continues to emit light for a time after cessation of exposure to radiation and will continue to produce an image which you do not want.

57. What are the characteristics of intensifying screen?

1. An intensifying screen consists of a base of polyester or cellulose triacetate similar to radiographic film.
2. This base must be radiotransparent.
3. It must be chemically inert.
4. It must combine characteristics of toughness and flexibility.
5. It should neither curl nor discolour with age.
6. The base is first coated with a reflective layer of titanium dioxide to bounce light back onto the film.
7. This is covered with a thin transparent super coat consisting of gelatin. The purpose of the latter is protective, and is very thin and care is always required in handling intensifying screens to avoid any kind of abrasion.
8. The flexibility of the material is important to allow the screen to bend without cracking—an intensifying screen of this type is used in the panoramic cassette.

58. What are grids? Mention their functions.

When an X-ray beam strikes a patient, many of the incident photons undergo compton interactions and produce scattered photons. Typically, the number of scattered photons in the remnant beam that reach the film is two to four times the number of primary photons that do not undergo absorption. The amount of scattered radiation increases with increasing subject thickness, field size, and kVp (energy of the X-ray beam). These scattered photons produce fog on the film and reduce the subject contrast. By virtue of function and composition, grids and collimators are same but differ by location.

The grid is placed in front of patient. It allows the parallel rays and absorbs the scattered rays. The problem is it forms its own shadow and the problem can be overcome by moving the grid.

Function

The function of a grid is to reduce the amount of scattered radiation exiting a subject that reaches the film.

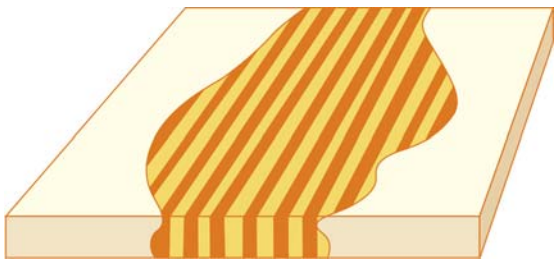
The grid, which is placed between the subject and the film, preferentially removes the scattered radiation and spares primary photons; this reduces non-imaging exposure and increases subject contrast.

Composition

A grid is composed of alternating strips of a radiopaque material (usually lead) and strips of radiolucent material (often plastic). When secondary photons generated in the subject are

scattered toward the film, they are usually absorbed by the radiopaque material in the grid. This occurs because the direction of the scattered photons deviates from that of the primary beam, and consequently they cannot pass through the parallel plates of the grid. Focussed grids are used most often. In a focussed grid the strips of radiopaque material are all directed toward a common point, the focal spot of the X-ray tube, some distance away. Because the lead strips are angled toward the focal spot, their direction coincides with the paths of diverging photons in the primary X-ray beam. The lead strips absorb the scattered photons as their paths diverge from those of the primary photons. A focussed grid can be used only within a range of distances from the focal spot where the alignment of lead strips closely coincides with the path of the diverging X-ray beam. The range of distances is specified on the grid. Grids are manufactured with a varying number of line pairs of absorbers and radiolucent spaces per inch. Grids with 80 or more line pairs per inch do not show objectionable grid lines on the image. The ratio of grid thickness to the width of the radiolucent spacer is known as the grid ratio. The higher the grid ratio, the more effectively scattered radiation is removed from the X-ray beam. Grids with a ratio of 8 or 10 are preferred.

To compensate for the absorbing materials in the grid, the exposure required when a grid is used is approximately double that needed without a grid. Therefore, grids should be used only when the improvement in diagnostic image quality is sufficient to justify the added exposure. For example, with lateral cephalometric examinations made for assessing the growth and development of the facial region use of a grid usually is not indicated because the improved contrast does not aid in identification of anatomic landmarks.



59. Enumerate the various ways to eliminate gagging.

Gagging is also called retching which refers to strong involuntary effort to vomit. It is protective mechanism. It is caused by stimulation of sensitive tissues of palate.

Causes—Psychogenic Stimuli

These are stimuli which originate in mind and causes the gag stimuli even the film is not placed in mouth. The psychic stimuli can be overcome by conditioning the patient appropriately.

Tactile Stimuli

These stimuli originate when the film is touched at soft palate, retromylohyoid space and lateral posterior third molar region. The ninth nerve or glossopharyngeal nerve covers the reflexes. The gag reflex is problem especially taking upper and lower molar region radiographs.

1. Attitude—dental radiographer must be confident. Every effort should be made to avoid gagging. If the patient gags the film should be removed and assured that gag reflex is less in patients who are relaxed and comfortable and explain in short the procedure.
2. Never suggest gagging—one should not use term gagging so that patients have psychogenic stimuli.
3. Keep the exposure factors and tube placement ready before placing the film in mouth.
4. The patient should take deep breath and asking the patient to concentrate on breathing when image receptor is in position or divert patients mind by asking him to count the numbers or making *yoga* (*anulom vilom*), patient is asked to look fixedly at

one point in room or asked to bite hard on bite plate.

5. The patient is asked to swallow immediately before insertion.
6. Ask the patient to gargle with ice cold water and patient sucking a local anaesthetic lozenge before attempting the position of image receptor or xylocaine ointment or spray.
7. Salt is placed on patient's tongue.
8. Place image receptor flat position as in occlusal film and giving steep downward angulation.
9. Use of film holders as when patient bites the floor of mouth is relaxed.
10. Stephen Cohen said, ask the patient not to blink, if the patient does not blink when film and sensor is kept in patient's mouth, the sense of gagging is eliminated and remove the film or sensor as soon as possible the procedure is over.
11. If everything fails, go for extraoral radiograph.

60. What are the other names of paralleling angle technique?

It is also known as extension cone paralleling (XCP) technique, right angle technique, long cone technique.

61. What are the fundamental rules of paralleling angle (right angle) technique?

The rules are:

1. The film is placed in mouth that is parallel to long axis of tooth.
2. The central ray is directed perpendicular to both long axis of tooth and plane of tooth.

62. What are advantages of long cone or paralleling angle technique?

- When performed accurately the image formed has linear and dimensional accuracy to help valid diagnosis, with less dimensional distortion, i.e. geometrically accurate images and true lateral image of tooth.
- The shape of teeth and relation to surrounding structures is more accurate.
- The technique used with aiming ring the alignment of X-ray beam is simplified.
- Head position is not critical as paralleling instrument and aiming ring will take care of alignment of X-ray beam.
- Eliminates the need of predetermined angulation.
- The image will have same shape of tooth but will have a little magnification, i.e. slightly larger than actual tooth size.
- The shadow of the zygomatic buttress appears above the apices of the molar teeth and it does not superimpose on apices of maxillary molar teeth because central ray is perpendicular to long axis of molars and enters below level of zygomatic arch.
- The periodontal bone levels are well-represented.
- The periapical tissues are accurately shown with minimal foreshortening or elongation.
- The crowns of the teeth are well-shown enabling the detection of proximal caries.
- The horizontal and vertical angulations of the X-ray tube head are automatically determined by the positioning devices if placed correctly. These film holding devices are useful in contour chairs or if patients are treated in supine positions, as in these positions it is difficult to place occlusal plane parallel to floor.
- The X-ray beam is aimed accurately at the centre of the film, all areas of the film are irradiated and there is no coning off or cone cutting.
- Reproducible radiographs are possible at different visits and with different operators and ultimately easy to standardize.
- The relative positions of the film packet, teeth and X-ray beam are always maintained, irrespective of the position of the patient's head. This is useful for some patients with disabilities.

- Dose reduction in paralleling technique. The vertical angulation in paralleling technique is either +10 degrees as compared to vertical angulation of +45 degrees compared to short cone bisecting angle technique. The lack of extreme vertical angulation reduces the exposure to the thyroid gland and lens of eye because they are in no longer path of primary beam.
- It is easier to standardize and serial comparison of radiographs have a greater validity. (Important in assessing alveolar bone levels after periodontal surgeries.)

63. What are the disadvantages of paralleling angle technique?

Positioning of the film packet can be very uncomfortable for the patient, causes discomfort, particularly for posterior teeth, often causing gagging. In children it is difficult.

- Positioning the holders within the mouth can be difficult for inexperienced operators.
- The anatomy of the mouth sometimes makes the technique impossible, e.g. a shallow, flat palate.

The apices of the teeth can sometimes appear very near the edge of the film:

- Positioning the holders in the lower third molar regions can be very difficult.
- The technique cannot be performed satisfactorily using a short focal spot to skin distance (i.e. a short spacer cone) because of the resultant magnification.
- The holders need to be autoclavable or disposable.
- The objection is to long cone PID also because they are difficult to operate in short operatory, longer exposure time may make patients movement.
- Difficult to place—when extremely long roots and rubber dam in place.

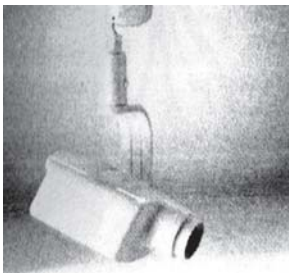
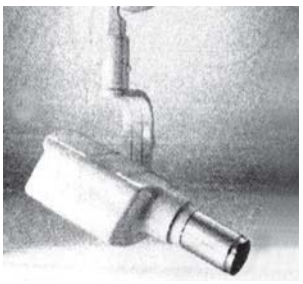
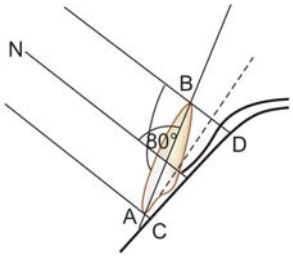
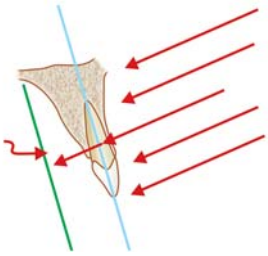
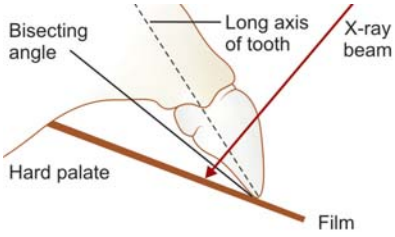
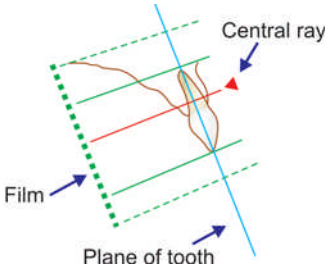
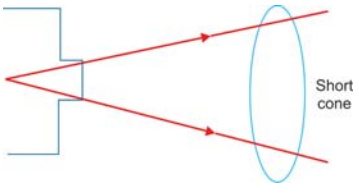
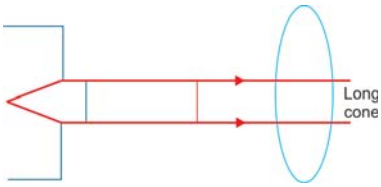
64. What are the advantages of bisecting angle technique?

- The proper technique represents the image of tooth with correct linearly, i.e. same length of tooth is formed on radiograph.
- Positioning of film is comfortable and patient friendly as the film does not impinge on tissues.
- Positioning in all areas of mouth can be reasonable, easily achieved.
- Simple and quick as does not require film holders.
- Radiographs can be taken with low output X-ray machines.
- No anatomical variation restrictions. The film can be angled to accommodate the different anatomical variations.

65. What are the disadvantages of bisecting angle technique?

- It represents the dimensional distortion, because the film and long axis of tooth are not parallel.
- Harder to position X-ray beam—since the film holders are not used, it is difficult to visualize where the X-ray beam directed.
- Film less stable—as it is used with finger chances of moving are more.
- The radiopaque image of zygomatic arch is often superimposed on apices of maxillary molars, making diagnosis difficult. This happens because the point of entry of central beam is along zygomatic arch.
- To implement the Cieszynski's rule of isometry in multirooted teeth, the central ray must be directed differently for different roots.
- The alveolar ridge is positioned coronally than its usual position.
- Cone cut chances are more.
- Incorrect vertical angulation (elongation/foreshortening) and incorrect horizontal angulation (overlapping).
- Less useful in periodontal diseases as levels are poorly represented.
- It is difficult to obtain reproducible views.
- The crowns are often distorted, difficult to detect proximal caries.
- The buccal roots of premolars and molars are often foreshortened.

66. What are differences between long cone paralleling and short cone bisecting angle technique?

	Bisecting	*Paralleling*
TFD	*6–8 inch*	*16–18 inch*
The size of cone		
Object film distance	Touches the tooth	Away from the tooth
		
Position of the film	Filmat angle	Film parallel to long axis of tooth
		
Central ray	Angulated	Parallel
Patient's exposure		
	More	Exposure is reduced

67. What is the modification of paralleling angle technique?

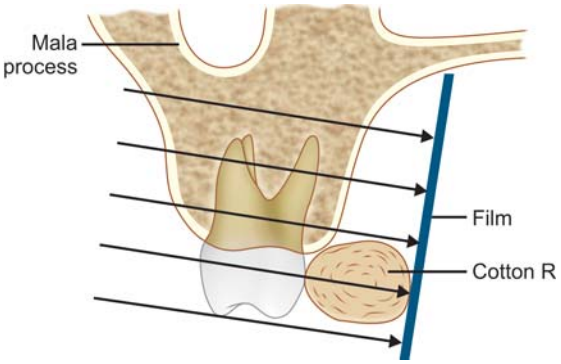
Walton has given modification of paralleling angle technique in which the central ray is directed perpendicular to the film but not the teeth. This helps to overcome the disadvantages of paralleling angle technique. This technique is useful for following situations:

1. Shallow palatal vault
2. Maxillary tori
3. Extremely long roots
4. Uncooperative
5. Gagging patients.

68. What is modification of bisecting angle technique?

The image receptor is placed in mouth in occlusal plane (flat) and the position of X-ray tube head is adjusted accordingly. This technique is used for:

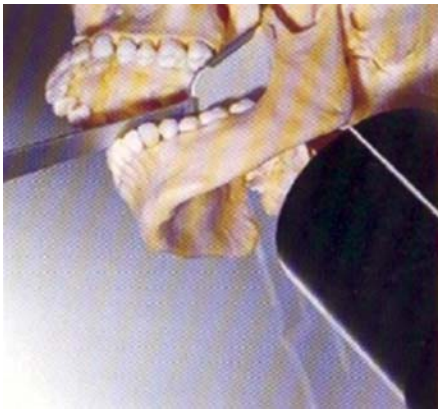
1. To avoid gagging

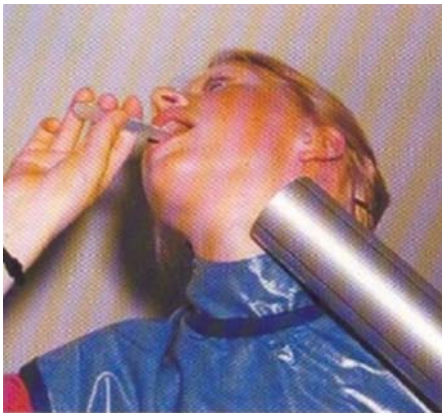


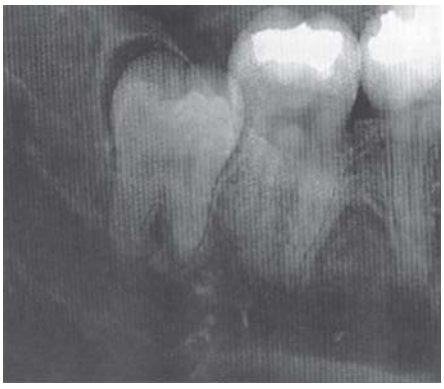
2. In children when difficulty in placing image receptor intraorally.

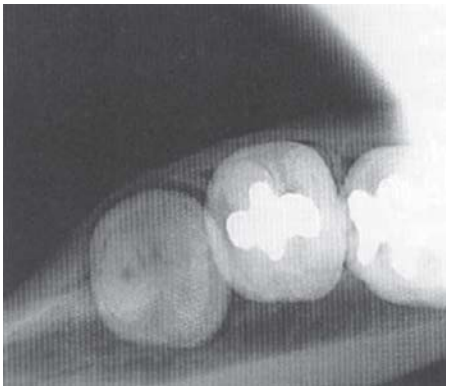
69. What is Le master technique?

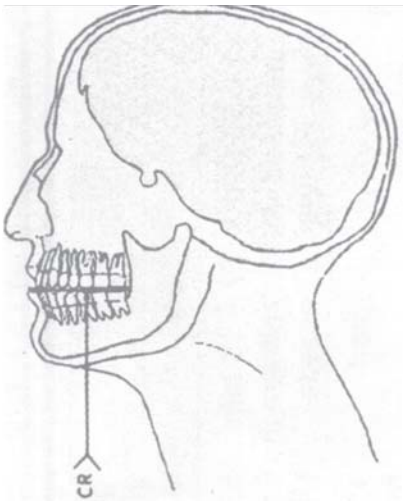
In Le master's technique a cotton roll is placed between the film and the palate so that the film is moved away from the teeth. The vertical angulation is then reduced and the central ray is directed from below the zygomatic process of maxilla to prevent its superimposition on the dentoalveolar structures.











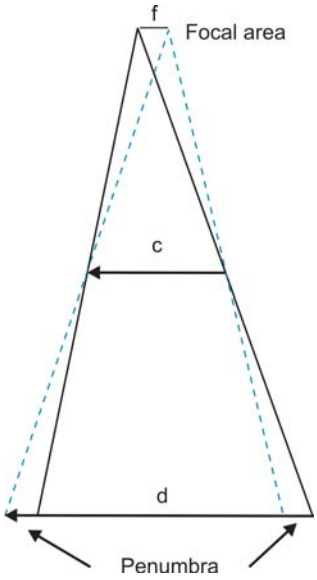
70. What is Donovan's technique?

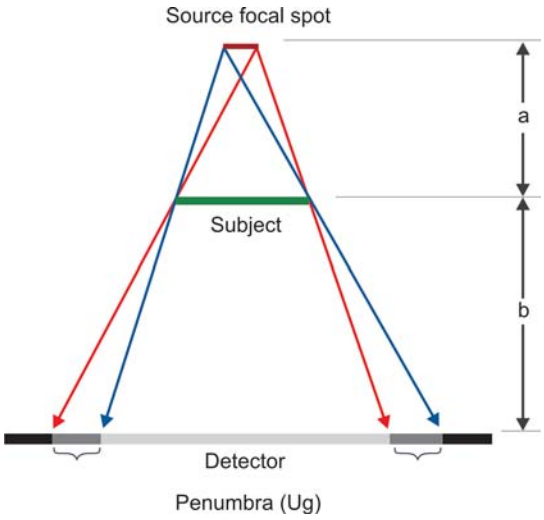
For studying of mandibular third molar impaction this technique is used. This technique is developed in 1952. The steps are as follows:

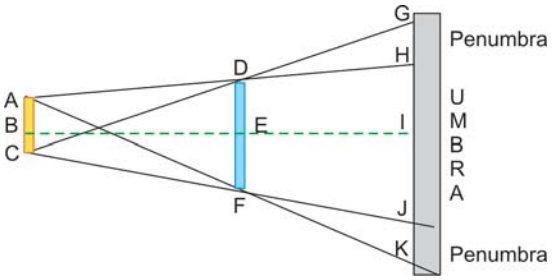
1. The film is places as far back as possible and the patient is asked to bite gently.
2. To make angle prominent the head is tilted downwards and forwards.
3. The central ray is perpendicular to film. Impacted third molars are visualized in buccal and lingual plane. The other name is Rita-Manuer technique.

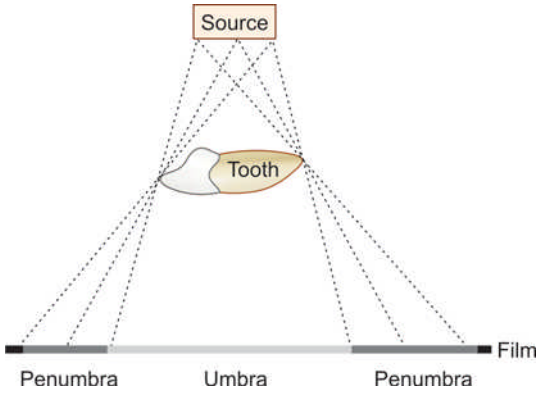
71. What is umbra and penumbra?

The word penumbra is derived from two Latin words *pene* means *almost* and *umbra* means shadow. The region of complete image is called umbra. Penumbra is the region of partial illumination that surrounds the umbra or complete shadow. It is also called geometric unsharpness or edge gradient.



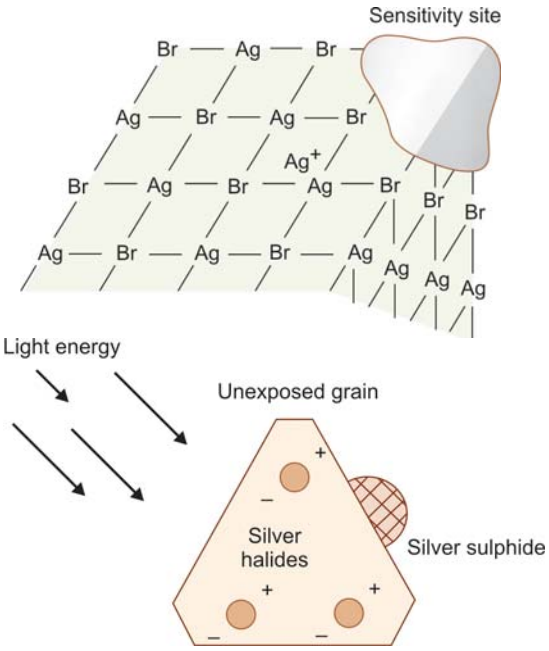






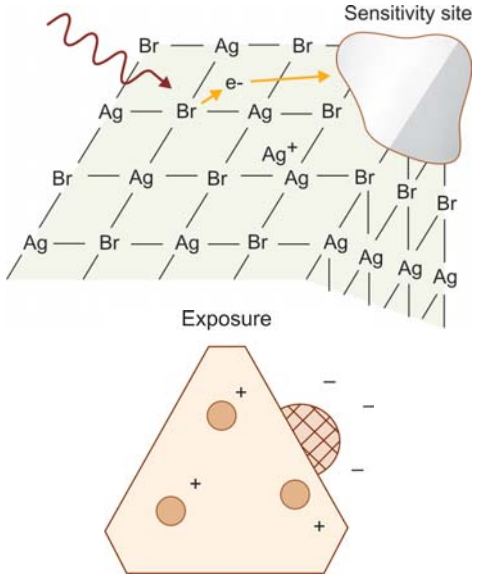
72. What is latent image?

Film emulsion consists of photosensitive crystals containing primarily silver bromide suspended in a vehicle and layered on a thin sheet of transparent plastic base. Some crystals also contain small amounts of silver iodide. These silver halide crystals also contain a few free silver ions (interstitial silver ions) in the spaces between the crystalline lattice atoms. The crystals are chemically sensitized by the addition of trace amounts of sulphur compounds, which bind to the surface of the crystals. The sulphur compounds play a crucial role in image formation. Along with physical irregularities in the crystal produced by iodide ions, sulphur compounds create sensitivity sites, the sites in the crystals that are sensitive to radiation.

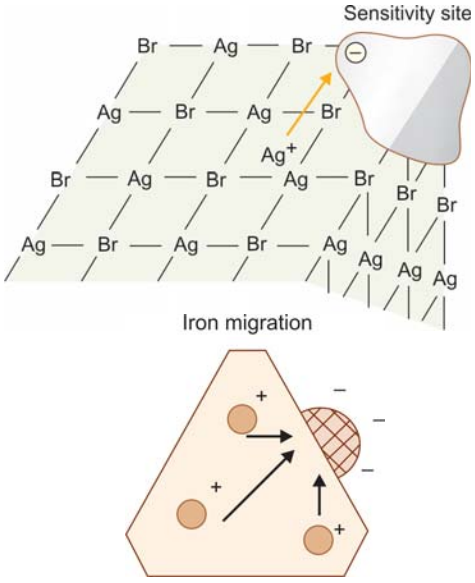


Each crystal has many sensitivity sites, which begin the process of image formation by trapping the electrons generated when the emulsion is irradiated. Exposure to radiation chemically alters the photosensitive silver halide crystals to produce the latent image.

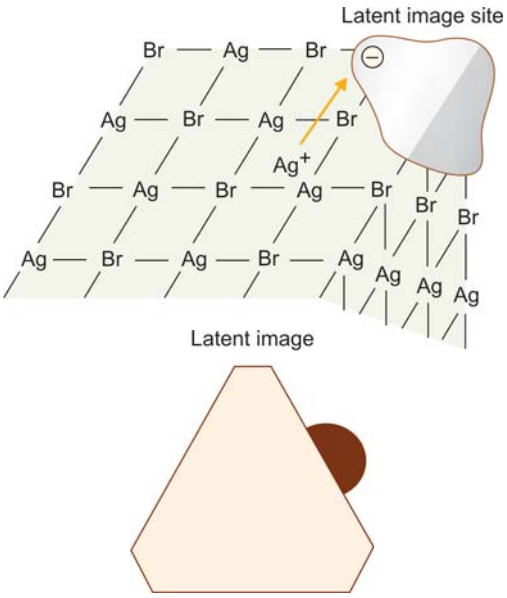
Processing the exposed film in developer and fixer converts the latent image into the visible radiographic image. When the silver halide crystals are irradiated, X-ray photons interact primarily with the bromide ions by compton and photoelectric interactions. These interactions result in the removal of an electron from the bromide ions. By the loss of an electron, a bromide ion is converted into a neutral bromine atom. The free electrons move through the crystal until they reach a sensitivity site, where they become trapped and impart a negative charge to the site.



The negatively charged sensitivity site then attracts positively charged free interstitial silver ions.

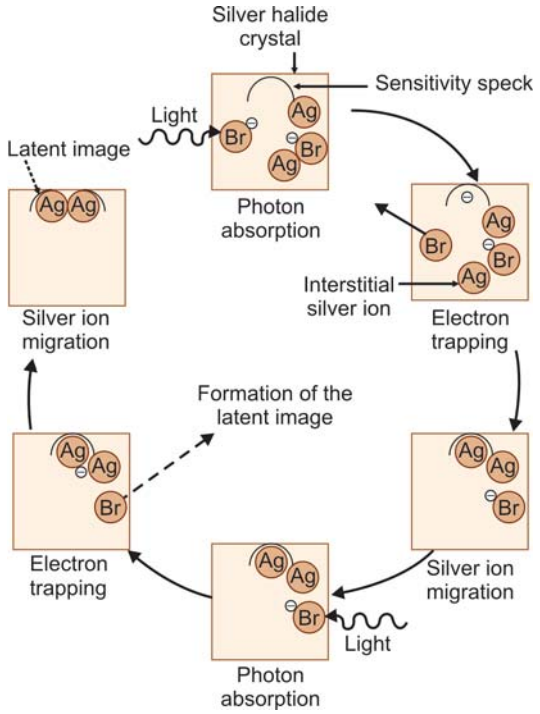


When a silver ion reaches the negatively charged sensitivity site, it is reduced and forms a neutral atom of metallic silver.



The sites containing these neutral silver atoms are now called latent image sites.

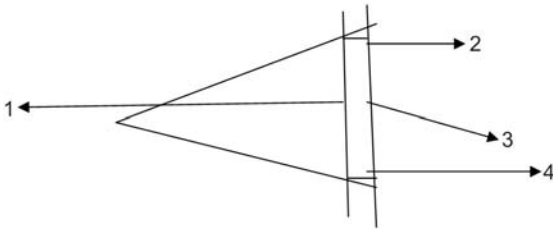
This process occurs numerous times within a crystal. The overall distribution of latent image sites in a film after exposure constitutes the latent image. Film processing converts the latent image into one that can be visualized. The neutral silver atoms at each latent image site render the crystals sensitive to development and image formation. The larger the aggregate of neutral silver atoms, the more sensitive the crystal is to the effects of the developer. Most latent image sites that are capable of being developed in an optimally exposed film have at least four or five silver atoms. Developer converts silver bromide crystals with neutral silver atoms deposited at the latent image sites into black, solid silver metallic grains. These solid silver grains block light from a view box. Fixer removes unexposed, undeveloped silver bromide crystals (those without latent image sites), leaving the film clear in unexposed areas. Thus, the radiographic image is composed of the light (radiopaque) areas, where a few photons reached the film, and dark (radiolucent) areas of the film that were struck by many photons.



The process repeats and is also called Gurney-Mott hypothesis

73. What is parallex?

Parallex results from an apparent change in position or size of a subject when it is viewed from a different perspective. This causes loss of image unsharpness due to double emulsion. The images recorded on each side may vary because of the divergent nature of X-ray beam. The use of intensifying screens also causes parallex distortion because light from one screen may cross the film base and reach the emulsion on the opposite side. The problem

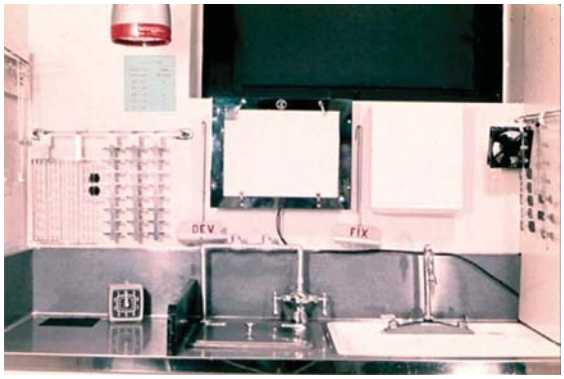


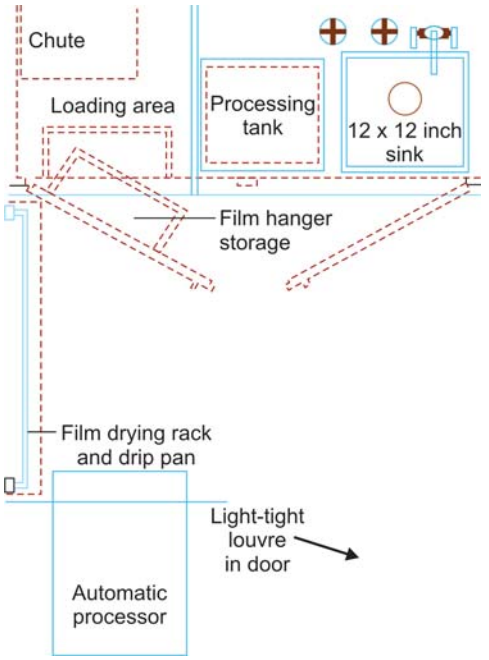
1 and 3—the two sides of emulsion,
2 and 4—the horizontal lines are parallex

can be solved by incorporating dyes that absorb light. Parallex is seen more in wet films.

74. What is darkroom?

The darkroom should be convenient to the X-ray machines and dental operatory and should be at least 4 × 5 feet (1.2 × 1.5 m). One of the most important requirements is that it should be lightproof. To accomplish this, a light-tight door or door less maze (if space permits) is used. The door should have a lock to prevent accidental opening, which might allow an unexpected flood of light that can ruin opened films. The room must be well-ventilated for the comfort of those working in the area and to exhaust the heat from the dryer and moisture from the drying films. Also, a comfortable room temperature helps maintain optimal conditions for developing, fixing, and washing solutions. If supplies (including unexposed X-ray film) are to be stored in the darkroom, ventilation is doubly important because temperatures of 90°F or higher can cause a generalized increase in density (film fog) on the film.





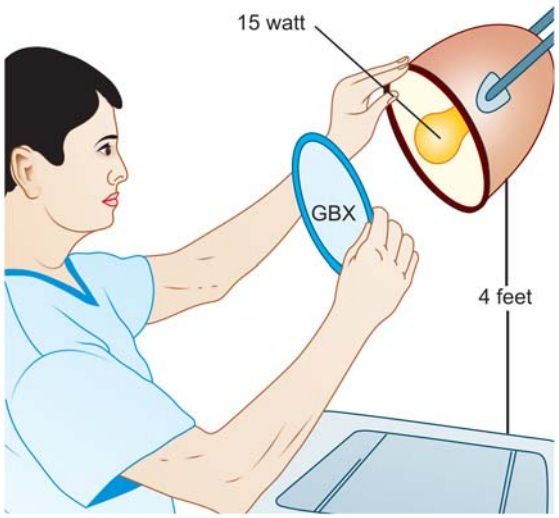
75. What is safe light?

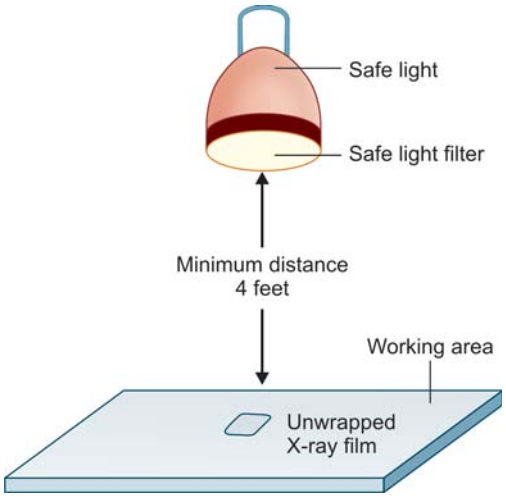
It is defined as a darkroom lamp with one or more color filters to screen out rays that can affect the photosensitive film. Safe lights allow darkroom workers to handle the photosensitive materials under limited illumination. It consists of light source (combusting gas or other fuel, incandescent light bulbs, fluorescent light tubes) and filter to limit the range of wavelengths that are used for illumination. X-ray films are not sensitive to the entire spectrum of visible radiation. They are sensitive to only UV blue, green emission and not sensitive to radiations in certain bandwidth of radiations in blue, green areas. If we use low lightning filters through colour filters, the film is safe.

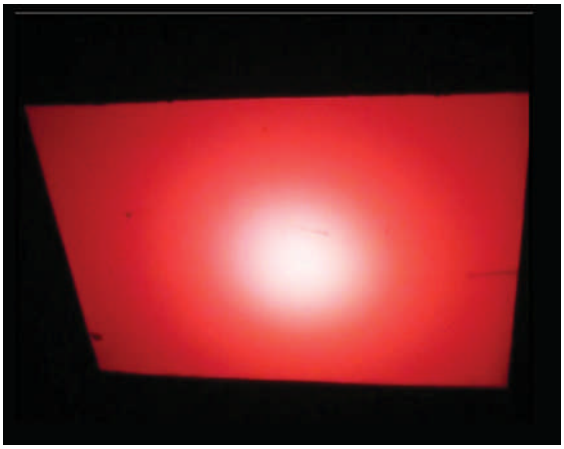
Type of Filter

This should be compatible with the color sensitivity of film used, i.e. blue, green or ultraviolet.

- **Condition of filters:** Scratched filters should be replaced.
- **Wattage of the bulb:** 15 watt bulb is used when direct illumination is used while 25 watt bulb is used when facing the ceiling. The pre-exposed films are 8 times sensitive than unexposed films so these should be kept for less time under safe light.
- **Their distance from the work surface:** Ideally they should be at least 1.2 m (4 ft) away from working place.
- **Overall safety (i.e. their fogging effect on film):** The simple quality control measure for doing this is known as the coin test or penny test.



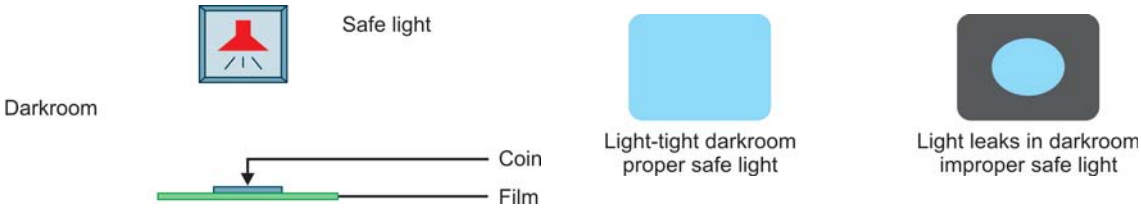


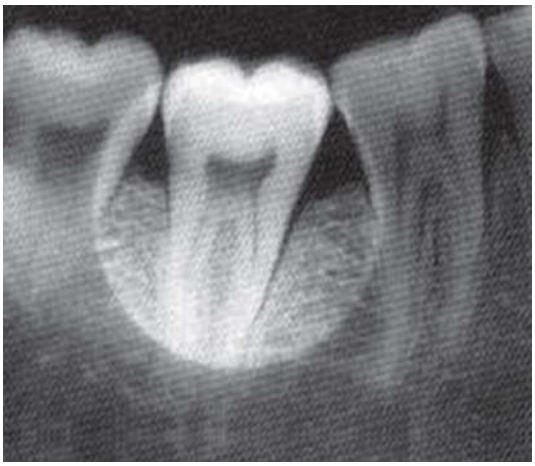


76. What is penny test?

It is also called coin test or safe light test. The following simple penny test can be used monthly to evaluate for fogging caused by inappropriate safe lighting conditions.

1. Shut all lights and start safe light.
2. Open the packet of an exposed film and place the test film in the area where the films are usually unwrapped and clipped on the film hanger.
3. Place a penny on the film and leave it in this position for the approximate time required to unwrap and mount a full-mouth set of films, usually about 5 minutes.
4. Develop the test film as usual. If the image of the penny is visible on the resultant film, the room is not light-safe for the particular film tested. Each type of film used in the office should be tested to measure the integrity of the darkroom.
   Note: Coin test can also be performed to assess the amount of light transmission through the safety glass of automatic processors by performing the test within the processor under the safety glass under the normal daylight conditions.





77. How the coin test can be performed for automatic processors? What is other test performed to test the safety of automatic processor?

The coin test can also be used to assess the amount of light transmission through the safety glass of automatic processors by performing the test within the processor under the safety glass under the normal daylight loading conditions.

The other test is:

1. Unwrap two unexposed films: Expose one to light.
2. Process both the films in automatic processor.
3. Check the results.
4. If the unexposed film appears clear and dry, and if the film exposed to light appears black and dry, the automatic processor is functioning properly.
5. If the unexposed films do not appear clear and dry and film exposed to light does not appear black and dry, the corrections must be made before the films are processed.

78. What are the contents of developing solution?

The developing solution contains four components, all dissolved in water:

1. **Developer:** The primary function of the developing solution is to convert the exposed silver halide crystals into metallic silver grains. This process begins at the latent image sites, where electrons from the developing agents are conducted into the silver halide crystal and reduce the constituent silver ions (approximately 1 billion to 10 billion) to solid grains of metallic silver. Two developing agents are used in dental radiology: A pyrazolidone-type compound, usually phenidone (1-phenyl-3-pyrazolidone), and hydroquinone (para-dihydroxybenzene). Phenidone serves as the first electron donor that converts silver ions to metallic silver at the latent image site. This electron transfer generates the oxidized form of phenidone. Hydroquinone provides an electron to reduce the oxidized phenidone back to its original

active state so that it can continue to reduce silver halide grains to metallic silver. Unexposed crystals, those without latent images are unaffected during the time required for reduction of the exposed crystals.

2. **Activator:** The developers are active only at alkaline pH values, usually around 10. This is achieved with the addition of alkali compounds (activators) such as sodium or potassium hydrozide. Buffers are used to maintain this condition—usually sodium bicarbonate. The activators also cause the gelatin to swell so that the developing agents can diffuse more rapidly into the emulsion and reach the suspended silver bromide crystals.
3. **Preservative:** The developing solution contains an antioxidant or preservative, usually sodium sulphite. The preservative protects the developers from oxidation by atmospheric oxygen and thus extends their useful life. The preservative also combines with the brown oxidized developer to produce a colorless soluble compound. If not removed, oxidation products interfere with the developing reaction and stain the film.
4. **Restrainer:** Bromide, usually as potassium bromide and benzotriazole are added to the developing solution to restrain development of unexposed silver halide crystals. Although bromide and benzotriazole depress the reduction of both exposed and unexposed crystals, they are much more effective in depressing the reduction of unexposed crystals. Consequently, the restrainers act as antifog agents and increase contrast.

79. What are the contents of fixing solution?

Fixing solution also contains four components, all dissolved in water:

1. **Clearing agent:** After development the film, emulsion must be cleared by dissolving and removing the unexposed silver halide. An aqueous solution of ammonium thiosulphate (hypo) dissolves the silver halide grains. It forms stable, water soluble complexes with silver ions, which then diffuse from the emulsion. The clearing agent does not have a rapid effect on the metallic silver grains in the film emulsion, but excessive fixation results in a gradual loss of film density because the grains of silver slowly dissolve in the acetic acid of the fixing solution.
2. **Acidifier:** The fixing solution contains an acetic acid buffer system (pH 4 to 4.5) to keep the fixer pH constant. The acidic pH is required to promote good diffusion of thiosulphate into the emulsion and of silver thiosulphate complex out of the emulsion. The acid fixing solution also inactivates any carryover developing agents in the film emulsion, blocking continued development of any unexposed crystals while the film is in the fixing tank.
3. **Preservative:** Ammonium sulphite is the preservative in the fixing solution, as it is in the developer. It prevents oxidation of the thiosulphate clearing agent, which is unstable in the acid environment of the fixing solution. It also binds with any colored oxidized developer carried over into the fixing solution and effectively removes it from the solution, which prevents oxidized developer from staining the film.
4. **Hardener:** The hardening agent most often used is aluminium sulphate. Aluminium complexes with the gelatin during fixing and prevent damage to the gelatin during subsequent handling. The hardeners also reduce swelling of the emulsion during the final wash. This lessens mechanical damage to the emulsion and limits water absorption, thus shortening drying time.

80. What are the components of automatic processors? What are functions of each?

1. **The processor housing:** Encases all the components of automatic processor.
2. **Film feed slot:** An opening of the outside of processor housing used to insert the unwrapped film in the processor.

3. **Rollers for transporter:** Consists of series of rollers driven by constant speed motor that operates through gears, chains or belts. These functions: (i) Primarily to move the film through developing solutions. (ii) The motion of rollers keeps the solutions agitated. (iii) The solution exchange is achieved in emulsion, as the roller press on emulsion forcing solution out and again it is refilled by next solution. (iv) The top rollers at the crossover point between the developer and fixer tanks remove rapidly the developing solution minimizing the risk of carrying the developer into fixer.
4. **Developer compartment:** Holds the developer solutions.
5. **Fixer compartment:** Holds the fixer solution.
6. **Water compartment:** Holds the circulating water.
7. **Drying chambers:** Used to hold the wet films to make it dry
8. **Replenished pump and solution:** Used to maintain proper solution concentration and levels automatically.
9. **Film recovery slot:** It is an opening at the outside of processing housing where the dry, processed film emerges out of automatic processor.

81. What is cutting reducer?

Farmer's solutions were previously called cutting reducer. It is used to lighten the radiograph. Farmer's reducer, a well-known reducer composed of:

Mix solution A: Plain hypo as follows: Sodium thiosulphate 480 grams (equal to 1–1/2 cups) water at 70°F to make 2 litres mix farmer's reducer.

Solution B: Ferricyanide as follows: Potassium ferricyanide 38 grams (equal to 2 tablespoons and 1 teaspoon) water at 70°F, 500 ml.

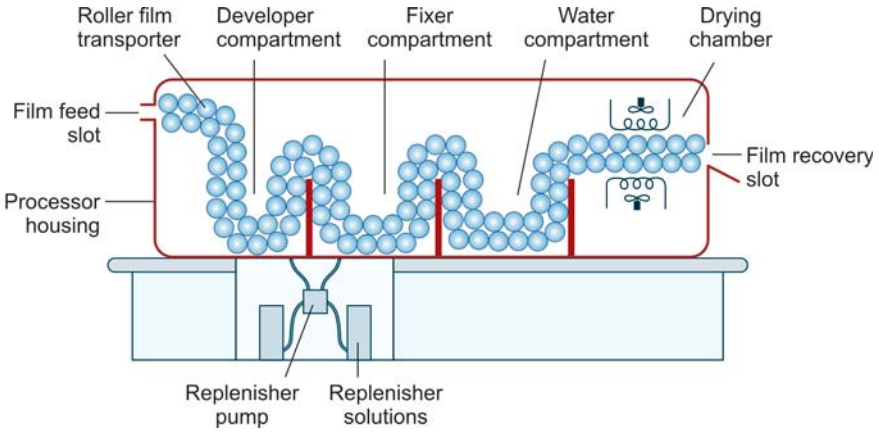
82. What are factors considered in image attributes of radiograph?

Following factors are considered in image attributes of radiograph:

1. Density
2. Contrast
3. Detail/resolution
4. Sharpness
5. Speed
6. Latitude
7. Noise

83. What is radiographic density?

The overall degree of darkening of an exposed film is referred to as radiographic density or overall blackness produced on radiographic film. An image that is neither too dark nor too light when seen on viewing monitor is said to have correct radiographic density. Density is primarily controlled by varying mAs usually by increasing or decreasing exposure time. kVp and SID do affect but are not used to



control the same. This density can be measured as the optical density of an area of an X-ray film where optional density is given as follows:

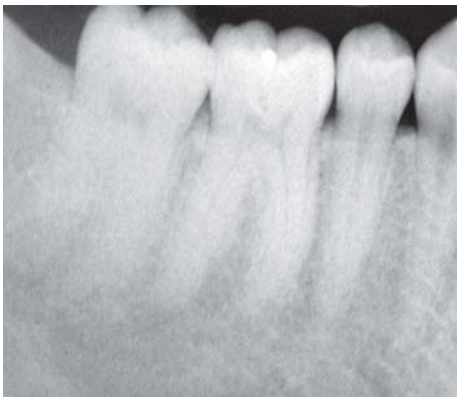
$$\text{Optical density } I_t = \text{Log}_{10} I_0$$

where I_0 is the intensity of incident light (e.g. from a view box) and it is the intensity of the light transmitted through the film. Thus, the measurement of film density is also a measure of the opacity of the film. With an optical density of 0, 100% of the light is transmitted with a density of 1, 10% of the light is transmitted with a density of 2, 1% of the light is transmitted, and so on.

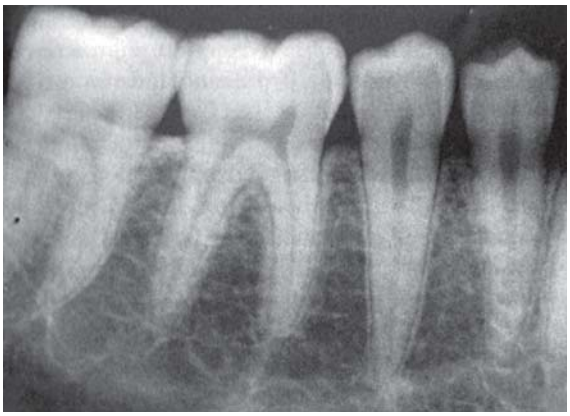
Film is of greatest diagnostic value when density is between 0.6 and 3.0.

84. Define radiographic contrast.

Radiographic contrast is a general term that describes the range of densities on a radiograph. It is defined as the difference in densities between light and dark regions on a radiograph. Thus, an image that shows both light and dark areas has high contrast. This is also referred to as a short grey scale of contrast because a few shades of grey scale are present between the black and white images on the film. A radiographic image composed of only light grey and dark grey zones has low contrast, also referred to as having a long grey scale of contrast. The radiographic contrast of an image is the result of the interplay of subject contrast, film contrast, and scattered radiation.

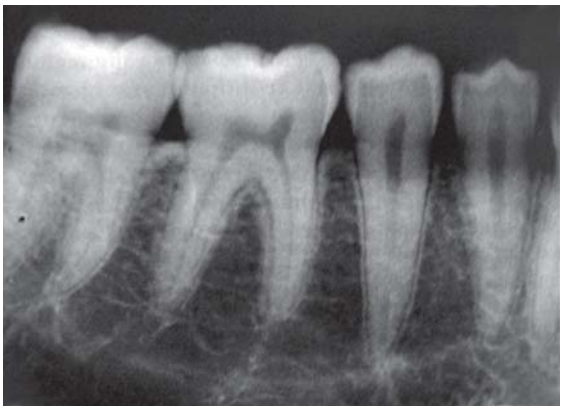


Low density light radiograph



Low contrast

High density, dark radiograph



High contrast

85. What is relationship between radiographic contrast and density?

When the contrast is changed, density is also altered. However, when only the density is changed, the contrast remains unchanged. The reason is:

- mA is the prime controlling factor for density, has no effect on contrast.
- kVp which is the prime controlling factor for contrast, also affects density.

Working rule: For every 10 kVp increase, mA must be halved.

86. What is resolution?

It is the ability of the radiograph to record separate structures that are close together. Measured by radiographing an object made up of series of lead strips with alternating spaces of same thickness. They are arranged in an increasing order of lines and spaces/mm. Resolution is the highest number of line pairs/mm that can be distinguished on the radiograph with low power magnification. Indirect films have 5 line pairs/mm. Direct films have 20 line pairs/mm.

87. What is sharpness?

It is the ability of the radiograph to define an edge precisely. Slower the film speed, smaller the grain size, better the sharpness. Intensifying screens decrease sharpness, avoided by close contact between screen and film. In dental radiographs, structures closer to the film are sharper than structures away from the source. Therefore, the lingual/palatal cusps will appear sharper than the buccal cusps.

88. What is radiographic speed?

Radiographic speed refers to the amount of radiation required to produce an image of a standard density. Expressed as the reciprocal of exposure in RA 'fast' film requires less exposure to achieve the same density as compared to a 'slow' film . Film speed depends on the size of the AgBr crystals and their silver content, bigger the crystals, faster the film. Speed can be increased by processing at higher temperature but this causes graininess and fog.

89. What is film lattitude?

It is a measure of the range of exposures that can be recorded as distinguishable densities on a film. Wide latitude can record a subject with a wide range of contrast. Useful when both hard and soft structures have to be recorded, e.g. lateral cephalograms.

Latitude of CCD and CMOS detectors is greater than film. PSPs have larger latitudes.

90. What is radiographic noise?

It is the appearance of uneven density on a uniformly exposed film. The causes are:

1. Radiographic mottle
2. Radiographic artifact

91. What is static electricity? What are the effects of static electricity on film called?

It refers to development of an electric charge due to friction. This goes on exposing the films, which is common on extraoral radiograph and rare on intraoral radiographs. These are star burst, smudge, grassroot, tree branch or lightning bolt.

Naked tree markings on the radiographs are a result of electrical discharges that do not produce any visible light.

Smudge type static electrical markings are caused because of low potential charges which produce visible light.

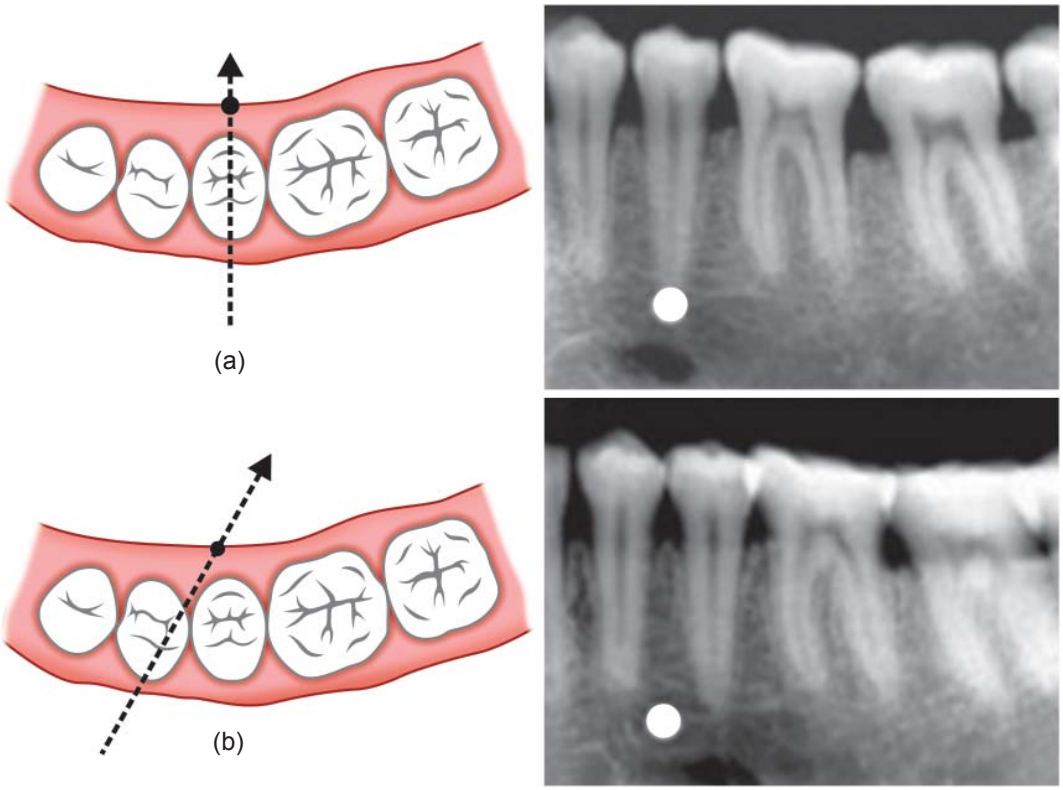
92. What is SLOB rule? What are the other names of SLOB technique?

This technique separates and identifies the facial and lingual structures. It is described by Clark in 1910. The rationale for this procedure derives from the manner in which the relative positions of radiographic images of two separate objects change when the projection angle at which the images were made is changed. If the tube is shifted and directed at the reference object (e.g. the apex of a tooth) from a more mesial angulation and the object in question also moves mesially with respect to the reference object, the object lies lingual to the reference object. Alternatively, if the tube is shifted mesially and the object in question appears to move distally, it lies on the buccal aspect of the reference object:

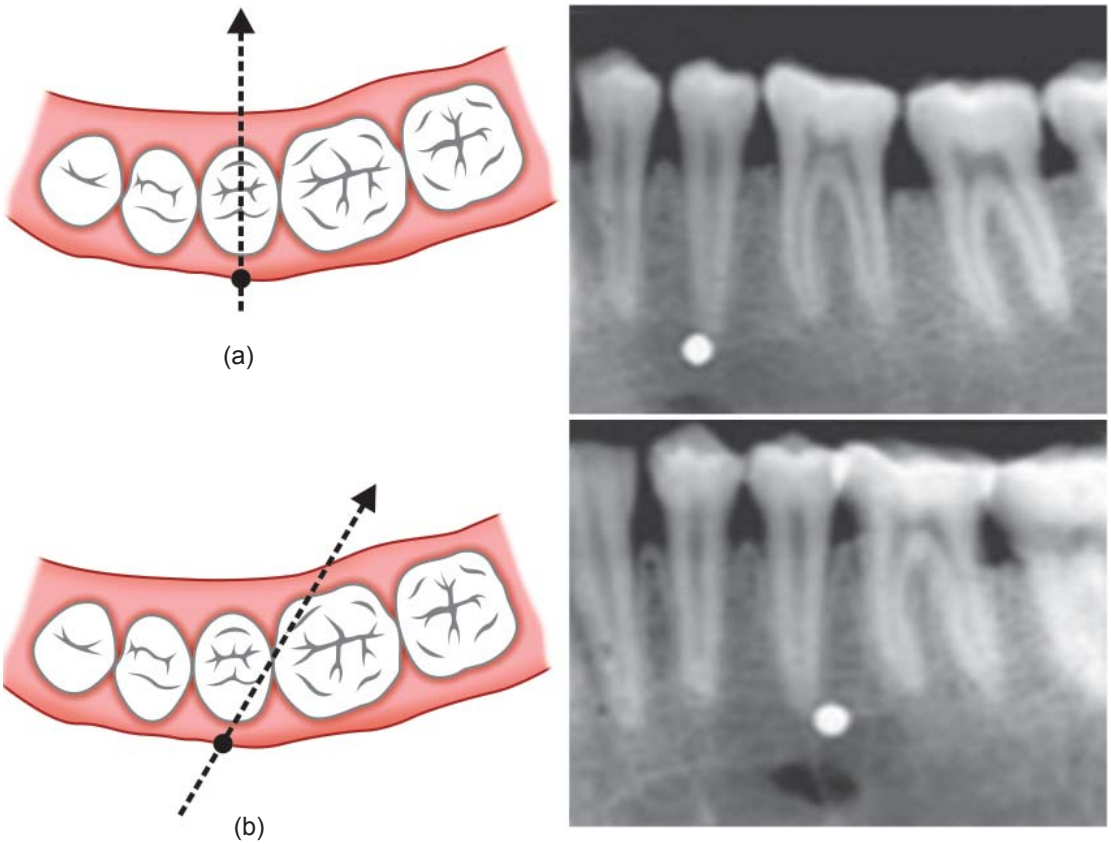
- The objects (which has to be separated from buccal and lingual) and the film are kept on fixed position.
- The tube head is moved for radiograph.
- The resultant radiograph shows the buccal objects moved away from the central ray of projection while lingual object is moving towards the same direction of projection (hence same lingual and opposite buccal).

The other names are BOR (buccal object rule), BOMM (buccal object moves most), Clark's rule, Walton's projection, cone image shift technique, and parallax technique.

Examination of a conventional set of full-mouth films with this rule in mind demonstrates that the incisive foramen is indeed located lingual (palatal) to the roots of the central incisors and that the mental foramen lies buccal to the roots of the premolars.



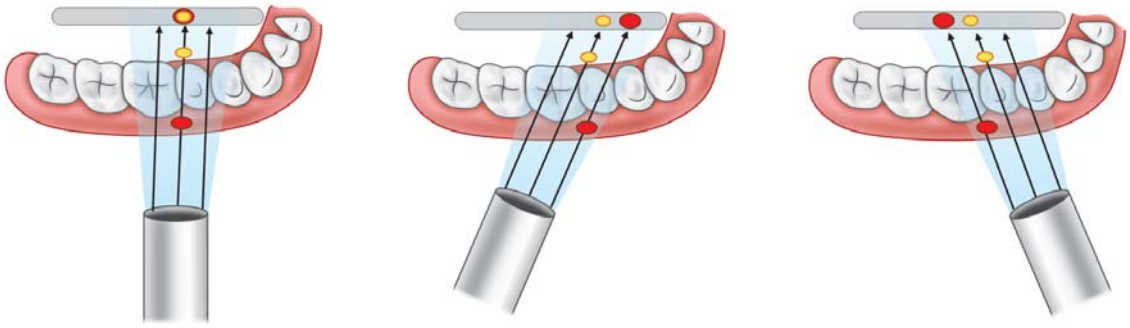
(a) Shows an IOPA of RO object placed lingually to apical area of mandibular second premolar with ideal horizontal angulation, (b) Shows the tube head is moved mesially and central ray directed distally showing the mesial movement of RO object which depicts its lingual position



(a)

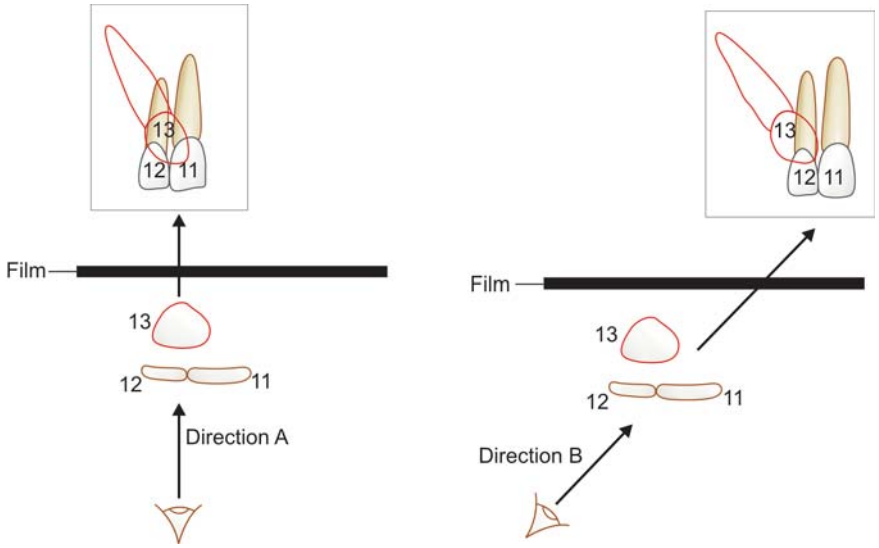
(b)

(a) Shows an IOPA of RO object placed bucally to apical area of mandibular second premolar with ideal horizontal angulation, (b) Shows the tube head is moved mesially and central ray directed distally showing the distal movement of RO object which depicts its buccal position



(a) (b) (c)

(a) Shows red is buccal object, yellow is lingual object with ideal horizontal angulation, (b) Showing distal movement of tube head with central ray mesially moving the buccal object moves (red dot) in opposite direction more than the lingual object (yellow dot) showing little, (c) Shows mesial movement of tube head with central ray directed distally showing the buccal object (red dot) moving more and lingual with a little change



(a) Shows IOPA showing normal incisor positions and impacted canine using ideal horizontal angulation, (b) The central ray moved distally and image of impacted canine has moved along with it distally indicating palatal impaction of canine.

93. What is panorama? What are the other names of panoramic radiography?

Panorama is an unobstructed view, it is also called bird's view, and extensive view. Panoramic radiography is also known as pantomography or rotational radiography, rotational panoramic radiography, orthopantomography [OPG], dental panoramic tomography [DPT], and panora. It is named after the panoramic X-ray machine.

94. What are the ten steps in panoramic radiography?

1. Load cassette.
2. Set exposure factors.
3. Remove patients jewellery, place apron on patient.
4. Ask the patient to bite on bite rod.
5. Adjust chin tilt.
6. Position the side guides.
7. Ask the patient to stand up straight right.
8. Ask the patient to swallow, place the tongue in the roof of mouth and hold still.
9. Expose the film.
10. Process the radiograph.

95. What are the indications of panoramic radiography?

Indications of panoramic radiography are as follows:

- For initial examinations of new patients in all age groups that can provide insight or idea in determining other projections and other screening.
- To evaluate the cause of multiple missing teeth or unerupted teeth.
- To assess the pathological conditions below complete and partial dentures (retained teeth/roots)
- As part of an orthodontic assessment where there is a clinical need to know the state of the dentition and the presence/absence of teeth.
- To assess tooth development especially in mixed dentition period.
- As attaining a larger field size than possible with periapical.
- To assess bony lesions or an unerupted tooth that are too large to be demonstrated on intraoral films and to attain larger field size than is possible.

- Prior to dental surgery under general anaesthesia.
- As part of an assessment of periodontal bone support where there is pocketing greater than 5 mm.
- Assessment of third molars, at a time when consideration needs to be given to whether they should be removed or not.
- Fractures of all parts of the mandible except the anterior region.
- Antral disease—particularly to the floor, posterior and medial walls of the antrum.
- TMJ disease—TMJ dysfunction syndrome, to investigate disease within joint, to investigate pathological conditions affecting condylar heads, fracture of condylar head and/or neck, condylar hypo/hyperplasia.
- Destructive diseases of the articular surfaces of the TMJ.
- Vertical alveolar bone height as part of preimplant planning.
- Asymmetries of jaws and face.
- Painful or asymptomatic swellings of jaws.
- When intraoral radiography is not possible (e.g. severe trismus, gagging).

96. How the dose in panoramic radiography is reduced compared to full mouth X-rays with periapical and bite wing radiographs?

In panoramic radiology the dose of radiation to the patient is 10 times less than full mouth survey using round BID and E+ film and it is 4 times less than 4 bite wings using long, round, and BID with E+ film.

The panoramic dose is equal to that of four bite wings using the long, rectangular BID and E+ film.

97. How does the normal panoramic radiograph look like?

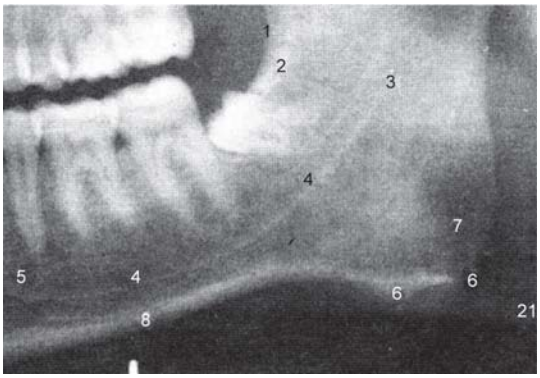
1. The mandible is U shaped.
2. Position of condyles: (a) About an inch inside the edges of film and one-third of way down from the top edge of film.
3. The occlusal plane exhibits a slight curve or smile line upwards.
4. The roots of maxillary and mandibular teeth are readily visible with minimal distortion.
5. Magnification is equal on both sides of midline.

98. What is tangential effect?

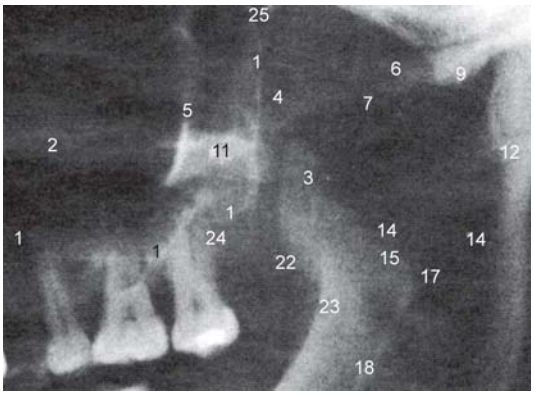
This is a characteristic effect of roentgen rays. By using normal exposure parameters if the structure is at right angle to central ray they will be visible only if the structure is of sufficient density or thickness.

The tangential effect of X-rays renders clearly visible in the irradiated space only those hard tissues with either high density or significant thickness:

1. **Flat surface structures:** The bony lamellae would not be visible unless they are parallel to central ray. The cancellous bone, only a portion bone will be visible.



Only basal portion of mandible is seen because of transverse tangentially by central ray



Zygomatic bone within radiolucency of maxillary sinus (6, 7) encountered tangentially.

2. **Curved surface structures:** Basal portion of mandible only seen as a tangential effect, temporal surfaces of zygomatic bone in maxillary sinus.

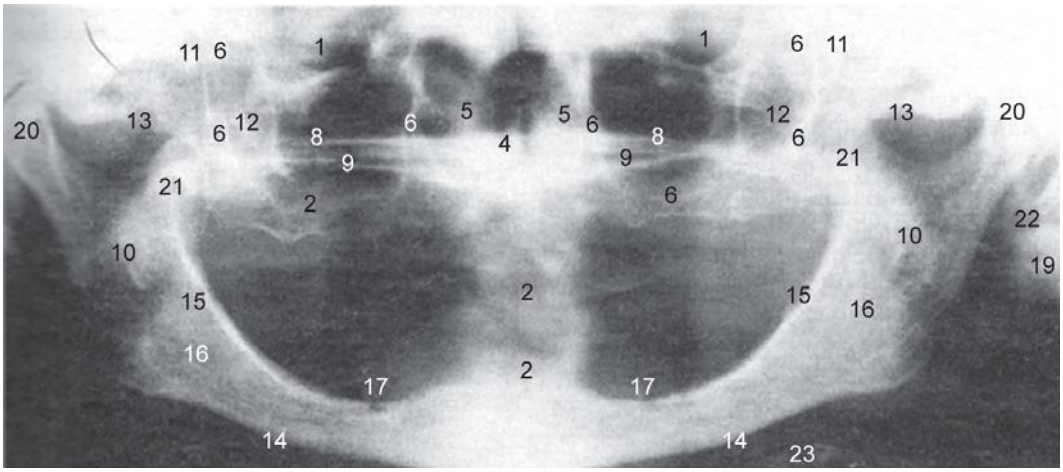
99. What is summation effect?

These are characteristic effects on radiographs by roentgen rays. The central ray along its traverse through tissues penetrates various objects and superimposes such objects upon each other and portrays the third dimension on two-dimensional radiograph. There are two types of summation effect, namely addition and subtraction effects. In both effects the clarity is lost by different mechanisms.

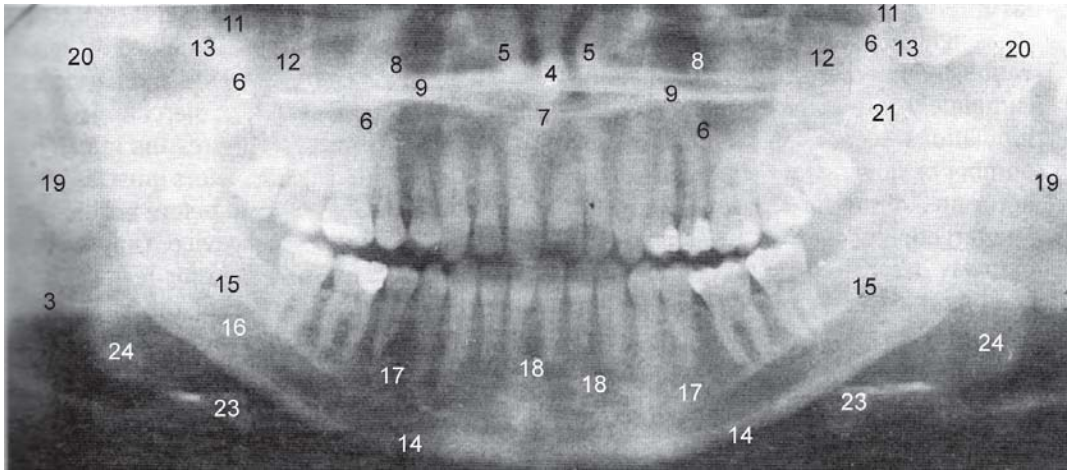
Addition Effect

The structure of interest lose clarity due to summation of radiopaque objects, e.g.

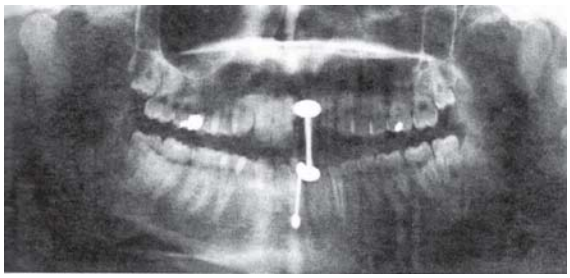
1. Mandibular anterior region by vertebrae. In children and adolescent this imposition is less disturbing as the hydroxyapetite content is low. As the age advances the addition effect is increased and even cannot be manipulated by changing kV setting or slowing speed of electrons.
2. The parts which are closer to image receptor are superimposed by both sides of the mandible. This especially in asymmetrically patients can lead to addition effects.



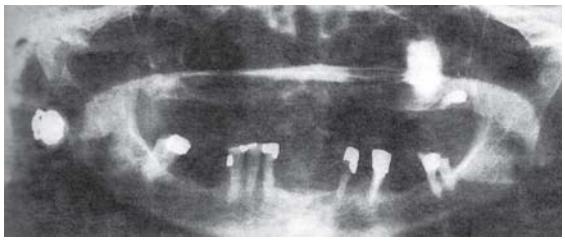
No. 2 is representing addition effect



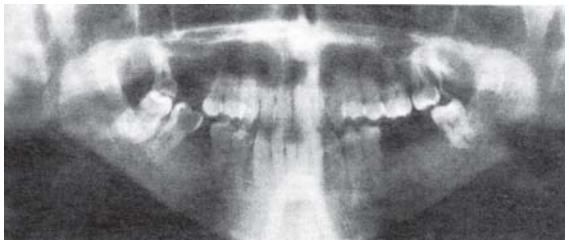
3. The foreign bodies, normal and pathological structures outside the plane of focus. The hairpins, necklace, eyeglasses, earrings, sialoliths, and calcified lymph nodes can obscure the diagnosis.



Radiopacity by piercing tongue



The earring with ghost image



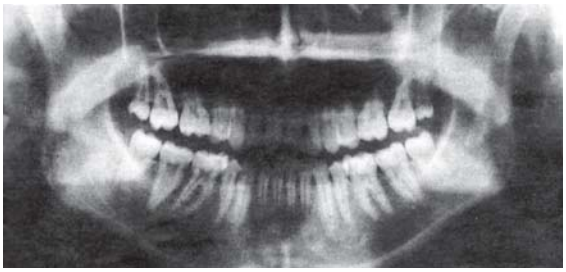
Radiopacity caused by metal zipper's placed on woman dresses, necklace

In this way the rays while travelling encounters soft tissues, osseous structures, and other radiopaque objects the ray is weakened or diminished by such material and ultimately the object shadow is with less radiation intensity, producing circumscribed shadowing.

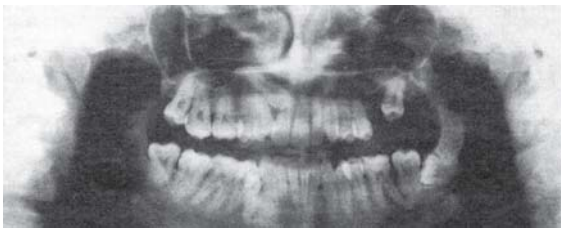
Subtraction Effect

The clarity is lost because of over radiation.

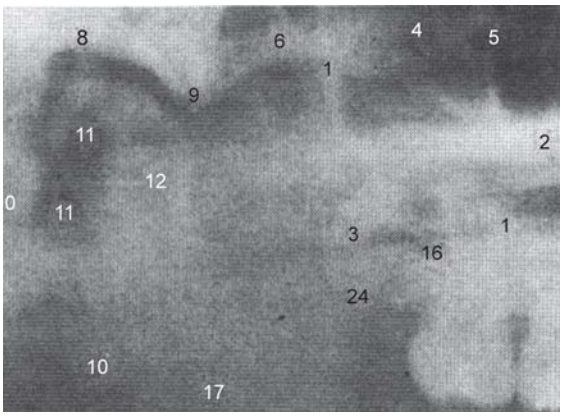
1. If dorsum of tongue is not pressed against the palate air containing space results, it promotes the uninhibited penetration of central ray in maxillary anterior region which obscures the images.



2. The shadow of epipharynx (if patient improperly takes deep breath and holds breath before exposure) as a radiolucency on ascending mandible creates radiolucency.



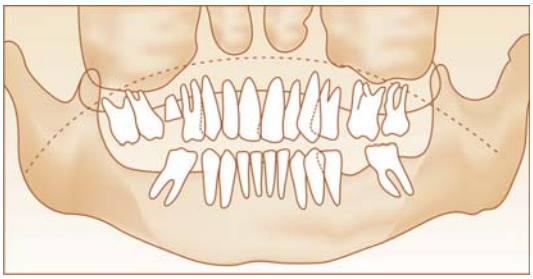
3. Osteolysis can be misdiagnosed by air containing external auditory meatus on mandibular condyle (circumscribed subtraction effect).

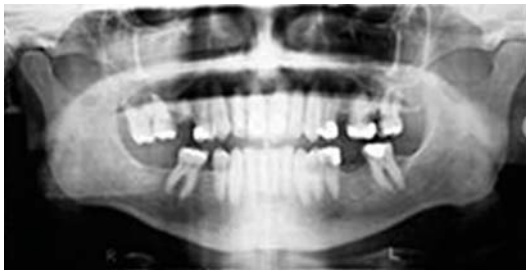


100. What is burn out effect?

This is due to improper positioning of tongue during projection. The roots of maxillary teeth, the structures of the maxilla, the boundaries of

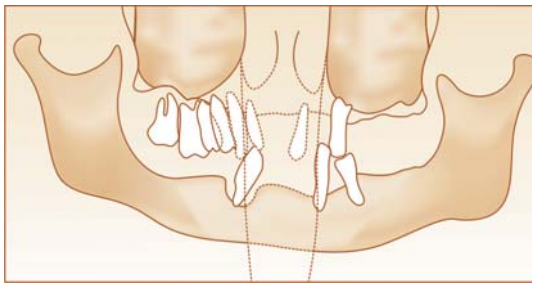
nasal and maxillary sinuses are not properly visible because of air. The air is negative contrast substance and is obliterating it. The radiation over-radiates that area and ultimately the structures are effected from final results. This is called burnout effect.

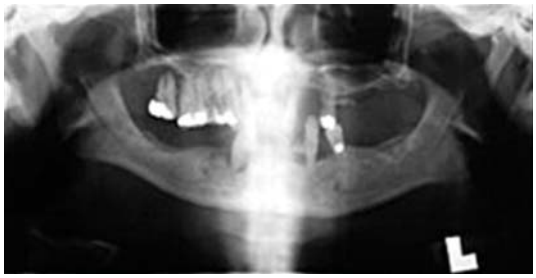




101. Which is the anatomic ghost seen on panoramic radiograph?

The ghost image of cervical spine is seen on radiograph.





102. How the ghost image of cervical spine is reduced on panoramic radiograph?

The ghost image of cervical spine is avoided by asking the patient to sit straight or asked to stand straight and the neck also straight, extended rather than slouched during exposure. If the patient is slouched, the X-ray beam traverses several cervical vertebrae.

103. What points are considered in the overall assessment of panoramic radiograph?

Divide panoramic radiograph into 6 zones:

Zone 1: Dentition

Zone 2: Nose sinus

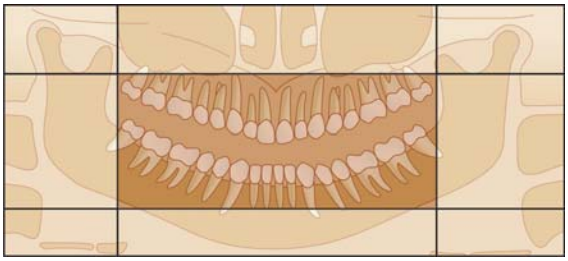
Zone 3: Mandibular body

Zones 4 and 6: Four corners; condyles and hyoid

Zone 5: Ramus spine

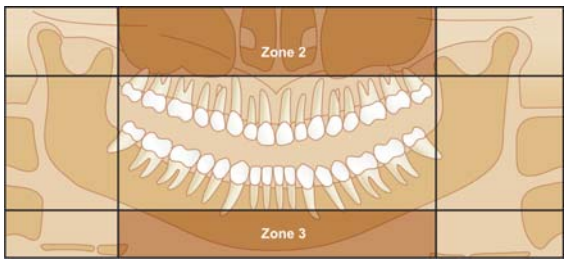
Zone 1: Dentition

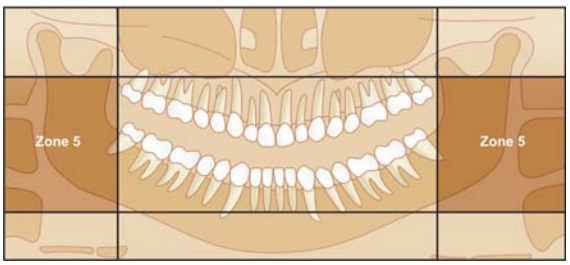
- Teeth should be arranged with a smile-like upward curve posteriorly and separated from each other.
- Anterior teeth should be neither too large nor so narrow.
- Posterior teeth should not be larger or smaller on one side than the other.



Zone 2: Nose sinus

- Images of inferior turbinates and their surrounding air spaces should be contained within nasal cavity.
- Soft tissue of nasal cartilage should not be seen.
- Hard palate shadow and sometimes ghost images of palate must be within maxillary sinus.
- Tongue must be in contact with hard palate.





Zone 3: Mandibular body

- Inferior cortex of mandible should be smooth and continuous.
- Double image or ghost image of body of hyoid should be absent in this area.
- Midline area should not be overly enlarged superiorly-inferiorly.

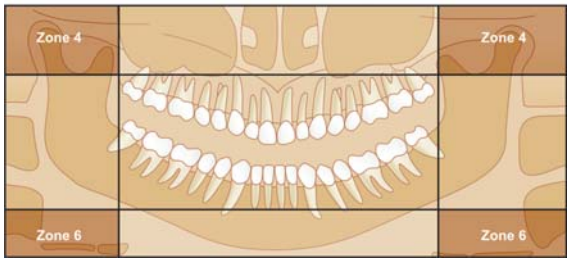
Zones 4 and 6: Four corners; condyles and hyoid

Zone 4:

- Contains condyles bilaterally.
- Should be more or less centred within this zone.
- Should be of equal in size and on same horizontal plane.

Zone 6:

- Contains body of hyoid.
- Should appear as a double image equal in size bilaterally.
- Should not spread across the mandible.

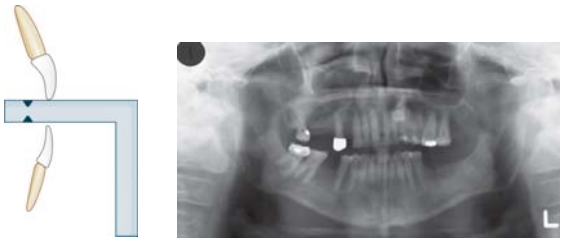


Zone 5: Ramus spine

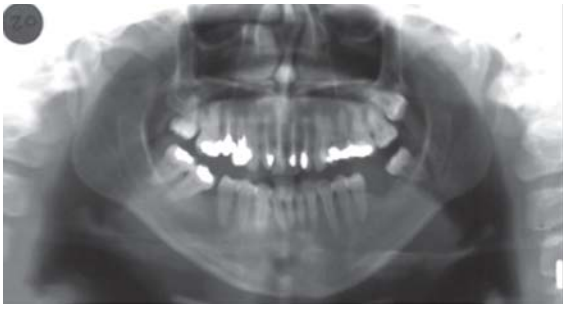
- Ramus of mandible should be of the same width bilaterally.
- Spine should not superimpose on ramus.
- When present distance between spine and ramus should be same bilaterally.

104. Enumerate some of the useful panoramic errors.

1. **Too for forward positioning:** To visualize nasal fossa and sinus. This results in an improved image of nasal cavity and maxillary sinus. Useful in detection of sinus disease, extension of disease into nose, and deviated nasal septum.



2. **Chin too low:** To visualize anterior maxillary teeth.



3. **Chin too high:** To visualize anterior mandibular teeth.

105. Classify panoramic errors.

1. Loading and uses of cassettes
2. Exposure errors
3. Positioning errors
4. Errors during exposure

106. What are the exposure errors during panoramic radiography?

Problem	*Cause*	*How to correct*	*Hints*
Light, pale film with a few dark areas	Too little exposure	Increase mA or kVp or use next higher settings on machine	Rule out, worn out or reversed screens
Dark film with loss of details, amalgams and unexposed areas are still clear	Too much exposure	Decrease machine settings	Do not confuse with film fogging which is overall grayness to film
White opacities on film; little or no image is visible on film	Ghosts of metal Jewellary	Remove prior to exposure	Watch out for necklaces
White opacity in palate	Tongue bar	Remove prior to exposure	Image is projected high onto palate instead of in floor of mouth
White opacity at bottom of film shaped like inverted "V" or "sharkfin"	Lead apron above colour line and X-ray beam	Adjust and properly place the apron	Watch for bunching at back of neck

107. Enumerate the panoramic positioning errors.

1. **Anterior positioning errors**

Problem	*Cause*	*How to correct*	*Hints*
Anterior teeth blurry, too small and narrow, spine visible on sides of film	Patient biting too far forward on bite rod	Make sure anterior teeth are located in grooves on rod	Make sure mandibular incisors are in groove also, and bite rod is not being bent forward
Anterior teeth blurry and wide, ghosting of mandible, spine, and condyles close to edge of film	Patient is biting too far back on rod or not at all	Make sure anterior teeth are located in grooves on rod	If anterior teeth are missing, use edentulous guide

2. **Chin tilt errors**

Problem	*Cause*	*How to correct*	*Hints*
Roots of lower incisors blurry, mandible-shaped like a "V", too much smile line, condyles at top of film, spine forms arch	Patient's chin is tipped too far down	Reposition using proper guidelines for that machine, such as ala-tragus line	Make sure patient does not have unusual occlusal plane orientation
Maxillary incisors blurry, hard palate superimposed on roots, flat occlusal plane, mandible is broad and flat, condyles at edge of film	Patient's chin is tipped too far up	Reposition using proper guidelines for that machine such as ala-tragus line	Make sure bite rod remains seated in its guide

3. Head twist error

Problem	*Cause*	*How to correct*	*Hints*
Teeth are wide on one side, narrow on other side of midline; ramus is wider on one side than the other; uneven pattern of blurring throughout arch; nasal structures not clear	Patient's head is twisted in machine causing midline asymmetry	Reposition using proper guidelines for that machine	Make sure patient does not try and look towards technician, but straight ahead. Always use front-surface mirror on machine to check alignment
Condyles are not equal in height, nasal structures distorted	Patient's head is rotated in machine (tipped)	Reposition using proper guidelines for that machine	Make sure patient's head remains level through ears

4. Slumping errors

Problem	*Cause*	*How to correct*	*Hints*
White tapered opacity in middle of image (Washington monument shape)	Ghost of spinal column due to slumping	Have patient take a step forward and straighten neck	Do not allow patient to reach forward into machine; then make step forward
Dark vertical line extending from top to bottom edge of film	Cassette hit shoulder and temporarily stopped	Straighten neck as above. Check apron for interference	Have patient keep elbows tucked into sides

5. Errors during exposure

Problem	*Cause*	*How to correct*	*Hints*
White vertical line on film running from top to bottom edge of film letting go of exposure	Cause exposure stopped briefly, probably due to button	Hold exposure button down firmly during exposure	Modern machines will return to start position if this happens
Images of springs or rectangular radiolucencies visible on film	Cassette was placed in machine backwards	Label tube side; place lead foil "X" on back side of cassette	Left and right will be reversed on film if this happens

6. Processing errors

Problem	*Cause*	*How to correct*	*Hints*
Thin, washed-out images	Depleted chemistry frequently	Replenish more	Consider X-OMAT processor
Fogged film, overall grey or very dark film	Improper filter in daylight loader	Use red filter or cover viewing area on daylight loader	You can use cardboard to cover filter area while loading panoramic film

108. List common factors that need to consider during panoramic exposure.

Factors to consider	*Exposure setting*
Obese patient with	Use the next highest kVp or mA setting
Large bone structure	Use the next highest kVp or mA setting
Patient with small bone structure	Use the next lower kVp or mA setting
Patient that is edentulous	Use the next lower kVp or mA setting

109. What is innominate line? What are the patterns of innominate lines?

It is a thin vertical radiopaque line on panoramic radiographs in the posterior third of antrum. The line is an artifact corresponding in its lower half to the posterior surface of the zygomatic process of maxilla and in its upper half to the posterior surface of frontal process of maxilla. It should be distinguished from vertical septate, reinforcement webs and posterior wall of maxillary sinus. The posterior region behind this line may be misinterpreted as available bone for implants.

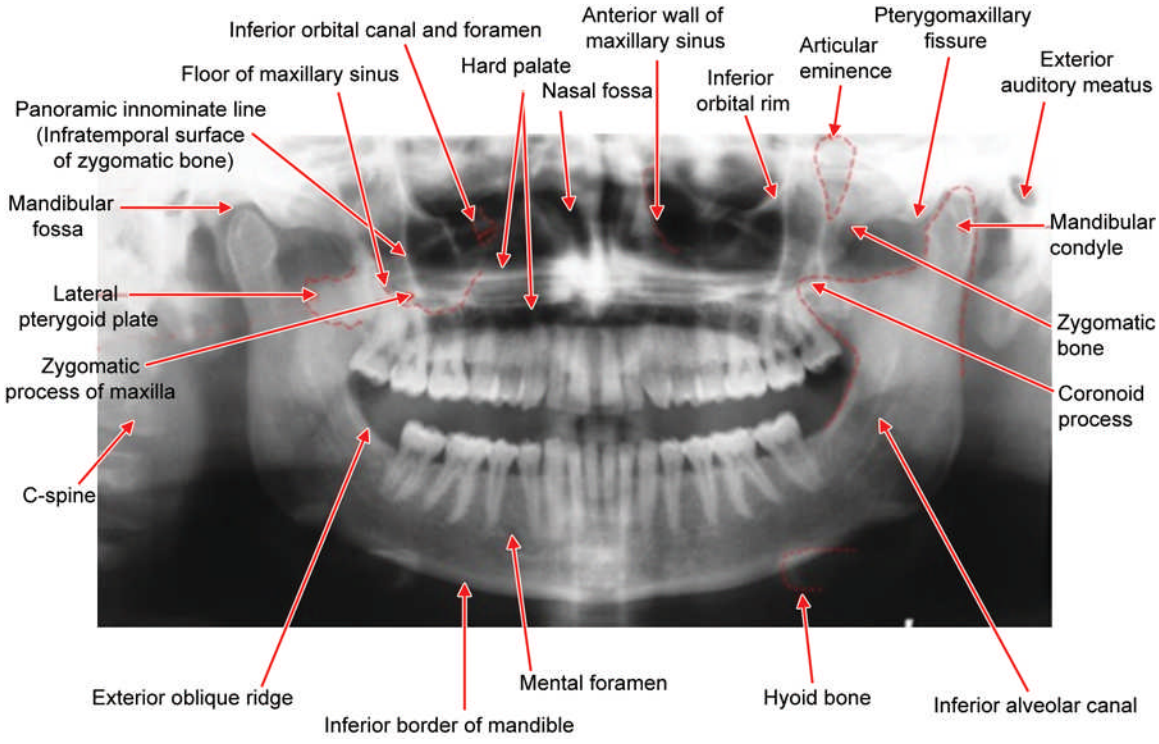
On skull radiograph and PA projections it represents the tangentially viewed greater wing of sphenoid. Panoramic innominate line indicates infratemporal surface of zygomatic bone.

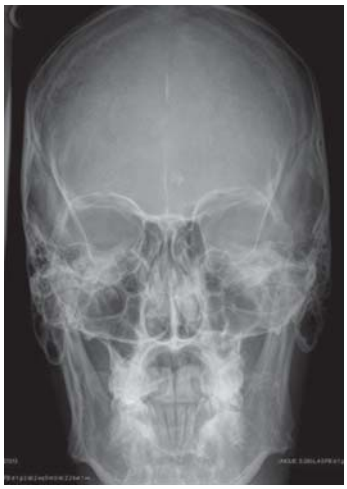
The three pathological processes affecting the innominate line, namely destruction, hyperostosis, and expansion.

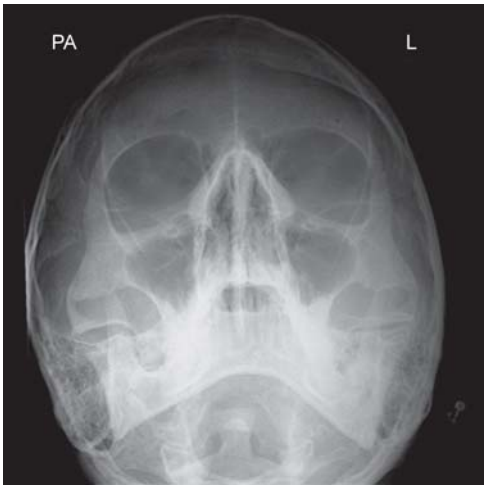
There are four patterns on radiographs:

- No visualization
- Visualization of whole length
- Visualization of upper half
- Visualization of lower half

Lower half innominate line is confused with bone tips of blow out fracture.







110. What is tomography and zonography?

Tomography is a general term for a technique that provides a distinct image of any selected plane through the body, while the images of structures that lie above or below are blurred.

Narrow angle tomography, which uses an angle of less than 10° is called zonography because a relatively thick zone of tissue (up to 25 mm) is sharply imaged, it is particularly useful when subject contrast is low because of little difference in physical density between adjacent structures. Because subject contrast is low in soft tissue, zonography is the preferred tomographic technique when soft tissues are imaged.

111. What is digital imaging?

Digital imaging is the art of making digital images—photographs, printed texts, or artwork through the use of a digital camera or image machine, or by scanning them as a document. Digital imaging uses sensors instead of a films. It requires low exposure time in comparison to conventional radiography and image is displayed on computer almost instantly without any need for chemical processing.

112. What are advantages of digital radiography over conventional radiography?

Advantages over conventional film-based radiography

- Lower dose of radiation required as both types of digital image receptors are much more efficient at recording photon energy than conventional films. It is nearly following 50% compared to F speed films.
- Film images and films are obtained much more quickly and time is saved and consultation can be expedited.
- Simultaneous availability of images at multiple sites without additional exposure.
- No need for conventional processing, thus avoiding all processing film faults and the hazards associated with handling the chemical solutions.
- Ultimately processing expenses are saved.
- No need to store films and darkroom space.
- No film degradation/no lost films.
- Easy storage and archiving of patient information and incorporation into patient records.
- Easy transfer of images electronically (teleradiology).
- Image enhancement and processing. Current software packages allow several image enhancement techniques including:
  - Inversion (reversal)
  - Alteration in contrast
  - Embossing or pseudo 3-D
  - Magnification
  - Automated measurement
  - Pseudo-colorization.

113. What is a sensor?

It is the key component of digital imaging system. It is a technology used to record the electronic images. A sensor consists of light sensitive elements arranged in a regular grid of rows and columns. A rectangular two-dimensional grid of sensor elements form a pixel.

114. What is analog to digital conversion?

At each pixel of the sensor, absorption of X-rays generate small voltage. Analog to digital conversion takes place in two steps:

1. **Sampling:** A small range of voltage values are grouped together as a single value.
2. **Quantization:** Every sampled signal is assigned a value.

These values are stored in computer and represent an image. These values range from 0 to 255 where 0 represents black and 255 represents white. All the values in between 0 and 255 represent different shades of grey.

115. What is the meaning of 'digital' in digital imaging?

'Digital' in digital imaging represents the numeric format of the image content and its discreteness.

116. What are the different digital image detectors?

The different digital image detectors are:

1. Charge coupled device (CCD)
2. Complementary metal oxide semiconductor (CMOS)
3. Photostimulable phosphor (PSP)
4. Bulk charge modulated device (BCMD)
5. Thin film transistor (TFT)
6. Flat panel detector (FPD)

117. What is charge coupled device (CCD)?

Sir George Smith and Willard Boyle on October 17, 1969 first mapped out CCD. CCD is a first direct digital imaging receptor that was used for intraoral imaging. It uses thin wafer of silicone for image recording. As CCD is more sensitive to light than X-rays, scintillating layer is also added. The scintillators used are gadolinium oxybromide and caesium iodide.

118. Where CCDs are used?

CCDs are involved in many aspects of everyday life. They are used in video cameras for home use and those set-up to automatically trap speeders on British highways, hospital X-ray imagers and high speed oscilloscopes, and digital cameras used as quality control monitors.

119. How does CCD work?

When silicone atoms are exposed to radiation, X-ray photons provide energy to electrons in valence band thus releasing them to the conduction band. This results in "electron hole" charge pair—electron in conduction band and hole in valence band. Number of these electron hole charge pairs formed is proportional to the amount of exposure received. Electrons in the conduction band are attracted towards the most positive potential in the device resulting in formation of "charge packets". These charge packets represent latent image site for CCD. Each charge packet represents one pixel. To read the image, each row of pixel charges is transferred from one pixel to next in "bucket brigade" fashion. When charge reaches at the end of each row, it is transferred to a readout amplifier and transmitted as a voltage to analog to digital convertor located within or connected to the computer.

120. What is pixel size range for CCD?

For CCD pixel size ranges from 20 microns to 70 microns. Smaller the pixel size, more expensive is the manufacturing of CCD.

121. What are the advantages of CCD in comparison to conventional film imaging?

1. Image can be seen on the computer screen almost instantly. There is no time lag between exposure and image display.
2. As no film processing is done, darkroom is not required.

3. As no chemicals are used for processing, disposal of those hazardous chemicals is eliminated.
4. As low exposure time is required in comparison to conventional imaging, resultant absorbed dose to the patient is also less.
5. Image manipulation is possible for better interpretation.
6. Teletransmission is easily possible.

122. What are the disadvantages of CCD in comparison to conventional film imaging?

1. In comparison to conventional films, CCD sensors are very expensive.
2. Sensors are bulky in comparison to the films and results in patient discomfort.
3. Active area of sensor is smaller than its total surface area.
4. Life expectancy of CCD sensor is unpredictable and mainly depends on its handling.
5. Blooming artefact—blooming is similar to allowing too much light through a viewer box, blinding the operator and washing out radiographic information in the excessive bright image. In CCD this blooming occurs by excess charge leakage to other pixel.

123. What is complementary metal oxide semiconductor?

The construction and working of CMOS is similar to CCD, only difference is in the way in which pixel charges are read. In CMOS each pixel is isolated from its neighbouring pixel and is directly connected to a transistor.

124. What are the advantages and disadvantages of CMOS over CCD?

CMOS has low manufacturing cost in comparison to CCD. The disadvantages of CMOS are that it has small active area for image acquisition and the sensors may not perform well in low light condition.

125. What are the other names for photostimulable phosphor radiography?

Other names for photostimulable phosphor radiography are digital luminescence radiography, storage phosphor radiography, computed radiography, and radioluminography.

126. What is the principle of PSP?

PSP absorb and store energy from X-rays and then release this energy as light when stimulated by other light of appropriate wavelength.

127. Explain the working of PSP.

PSP is made of europium doped barium fluorohalide. Barium fluorohalide forms a crystal lattice and europium creates imperfections in this lattice.

When exposed to radiation, valence electrons in europium absorb energy and are released to conduction band. In conduction band these electrons are trapped in halogen vacancies known as "F centres". In PSP these F centres represent latent image site. On exposure to light, trapped electrons in F centres release energy in the form of green light and go back to the valence band. This green light is detected by photomultiplier tube and is converted into electrical energy. These voltage signals are sent for analogue to digital convertor and digital image is formed.

128. What is the function of photomultiplier tube in PSP?

Red filter at the photomultiplier tube selectively removes the stimulating light. Green light released from trapped electrons is detected and converted to voltage in photomultiplier tube.

129. What are the safety precautions for use and storage of PSP plates?

1. Before exposure, PSP plates should be erased to eliminate "ghost images" from prior exposures. This can be done by flooding the plate with a bright light source. Most of the times, the processing system is incorporated with automatic plate erasing lights. For intraoral plates, phosphor side of the plate can be placed on dental view box for 1–2 minutes to erase it.
2. Erased plates should be kept in light-tight containers.

3. For intra-oral plates, sealable polyvinyl envelopes impervious to oral fluids and light are used for packaging.
4. For extraoral plates, conventional cassettes without intensifying screens can be used. Some manufacturers provide extraoral plates with cassette.
5. A semi-dark environment is recommended for plate handling.
6. Red safelights found in most darkrooms are not safe for exposed PSP plates as they are more sensitive to red light spectrum.

130. What are the advantages of PSP intra-oral plate over CCD sensor?

PSP intra-oral plate does not have electric cord attached to it like CCD sensor so placement of PSP plate in oral cavity is easy. Also, PSP plates have less thickness than CCD sensor so they are more comfortable to the patient.

131. What are the disadvantages of PSP intra-oral plate over CCD sensor?

In case of CCD, image is displayed on computer screen almost instantly after exposure. In case of PSP, the plates have to be processed through the system so it takes more time in comparison to CCD. Also since PSP plates are less thick in comparison to CCD sensor, they can get damaged easily.

132. What are flat panel detectors?

FPDs are currently used in cone beam computed tomography (CBCT). They provide relatively large matrix areas with pixel sizes less than 100 microns and allow direct digital imaging of larger areas of body.

133. What are the types of flat panel detectors?

FPDs are of two types: Indirect FPD and direct FPD.

Indirect FPDs are sensitive to visible light. Intensifying screens with gadolinium or caesium are used to convert X-rays to visible light. Use of intensifying screens makes indirect FPDs more efficient but leads to image unsharpness.

Direct FPDs use a photoconductor material like selenium due to which there is more efficient absorption of X-rays providing higher resolution.

134. What is spatial resolution?

Spatial resolution is capacity for distinguishing fine details. It is measured in units of line pairs per millimetre. For IOPA film resolution is 20 lp/mm, for OPG it is 5 lp/mm, for most digital systems resolution is more than 7 lp/mm and for CT resolution is 12 lp/mm.

Chapter

10

Radiology—Part 2

1. What is Aunt Minnie phenomenon?

It was coined by famous radiologist Ben Felson. The Aunt Minnie phenomenon in radiology is a phenomenon of correctly identifying a disease process by its unique radiological presentation. Aunt Minnie represents an abnormality which looks like one that the evaluator has seen before or been told about. It will be difficult to recognize new findings using this approach. Cousin Harry represents an abnormality which the evaluator has not seen for a long time, but would like to see while uncle Fred represents an abnormality which is often present.

2. Enumerate the radiographic techniques to visualize posterior border of maxillary sinus.

Posterior wall separates the maxillary sinus from pterygoplatine fossa. Posterior border of maxillary sinus is visualized in IOPA of posterior maxillary areas, lateral occlusal view, panoramic X-rays, submentovertex view, lateral cephalogram view. Although PA Waters view being best projection to visualize sinuses the posterior wall cannot be seen on it.

3. What are Campbell's lines?

These are the lines to be seen on occipitomental radiograph.

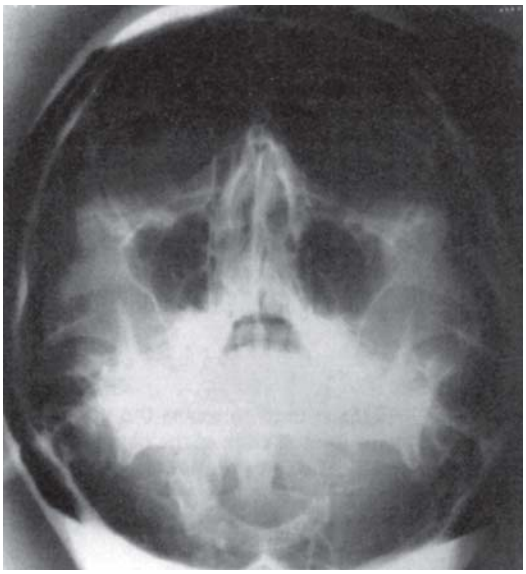
Line 1: Joins the two zygomaticofrontal sutures. It runs along the superior orbital margin on each side and centrally across the region of glabella. This is check for any separation of the zygomaticofrontal suture and look at the integrity of the superior orbital margin.

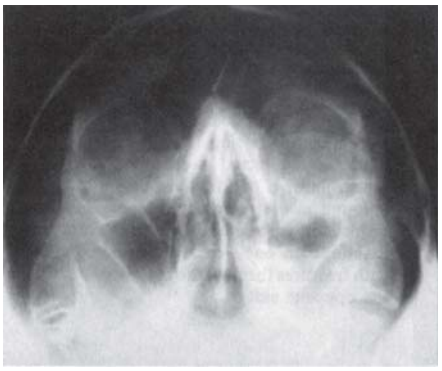
Line 2: It is traced from the zygomatic arch. It follows the zygomatic bone and continues along the inferior orbital margin across the frontal process of maxilla and lateral wall of the nose through the septum. It then follows a similar course on the other side. Check the zygomatic arch fractures, then compare the transverse width of the frontal process of maxilla and vertical dimensions of the zygomatic bones on the left and right sides. A symmentry indicates fracture. Look for break in continuity of the inferior orbital margin, particularly at the junction of inner third and outer two-thirds. A downward blow out fracture of the orbit may be seen (tear drop sign).

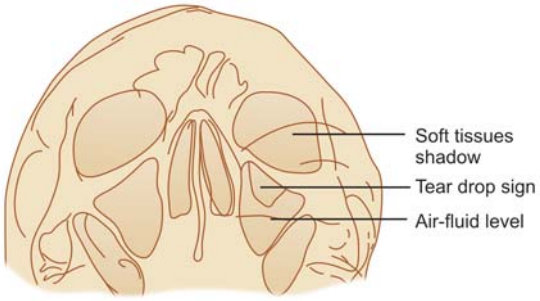
Line 3: Starts at the condyle of the mandible and traces across the mandibular notch and coronoid process to the lateral wall of maxillary antrum. It continues through the medial wall of the antrum or lateral wall of the nose at the level of the nasal floor and follows a similar course on opposite side. Check the continuity of the maxillary antral walls and look for any depression of the orbital floor.

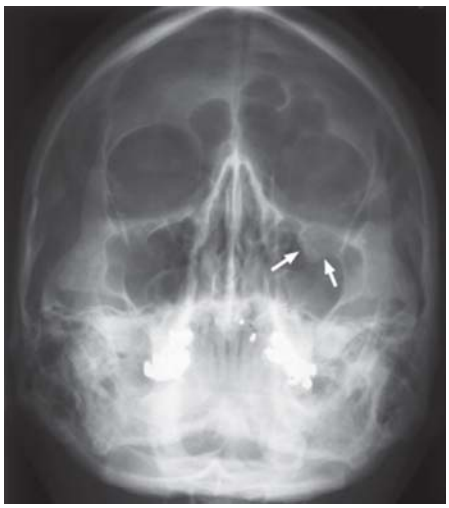
Line 4: Follows the occlusal curve of the upper and lower teeth. Check for evidence of mandibular fractures. Specific views are indicated for diagnosis.

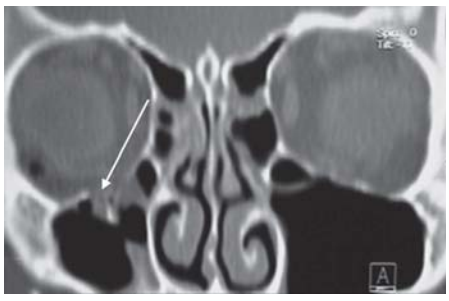
Line 5: It is also called Trapnel's line, which traces the line of lower border of the mandi.











4. What is tear drop sign?

Blow out fracture of inferior orbital margin. This injury occurs when an object (e.g. ball) hits the eye rather than zygoma. The contents of the orbit are pushed down through orbit since it is weakest point.

The inferior rectus muscle is trapped causing diplopia on upward gaze. The classical appearance on X-ray is ball of proptosed tissue described as "tear drop" appearance.

If this sign is observed, the patient is not supposed blow through nose.

5. Which projections are used to visualize coronoid process? Which is the best projection?

Panoramic view, PNS, SMV, PA mandible, transpharyngeal view. PNS is the best projection.

6. Enumerate the uses of ultrasound imaging in oral and maxillofacial region.

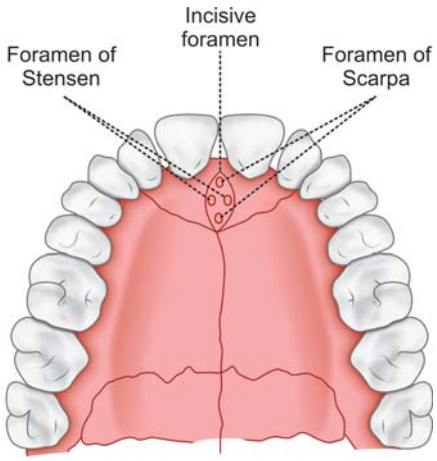
It is an easy method to detect noninvasive and soft tissue related diseases in oral and maxillofacial regions.

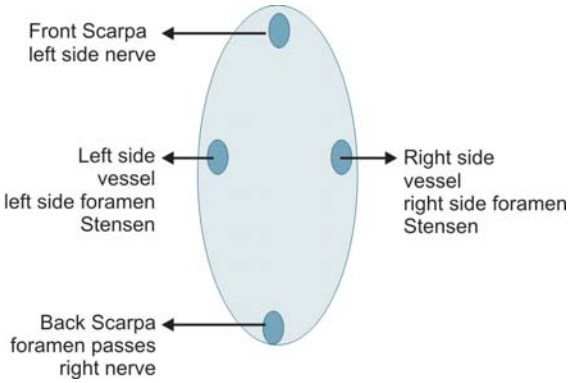
1. **Normal anatomical structures:** It is in particular in the oral and maxillofacial region in analyzing normal and abnormal anatomical structures, as Doppler images associated with the B-mode can provide vascular information associated with morphology of soft tissues.
2. **In salivary gland diseases:** The examination done by ultrasonography can clearly identify the presence or absence of mass-like lesions and hence readily detect and diagnose salivary gland-related diseases.
3. **In lymph node diseases:** The above-mentioned property of mass detection by USG helps in detection of lymph node pathology.
4. **Ultrasound with fine needle aspiration biopsy:** The accuracy of this procedure is been shown to be relatively high in spite of noninvasive procedure.
5. **Interventional radiology using fine needle aspiration by USG:** The resolution of Ranulas and plunging Ranula occurred after administration of sclerotherapeutic agents under ultrasound guidance.
6. **Diagnosis in tongue lesions:** In tongue cancer US imaging is often used to accurately estimate tumour size or thickness and to define adequate resection margins with tumour extension and deep infiltration.
7. **Diagnosis of metastatic lymph nodes:** Metastatic cervical lymph nodes are seen better than CT. It is difficult to palpate the remaining lymph nodes in neck after cancer treatment because the normal tissues are replaced by cicatrix produced by granulation tissues and so they are even difficult to palpate also. Examination of thyroid gland by USG is possible to detect metastasis from oral cancers.

7. What is foramen of Stensen and Scarpa contain?

A little behind the central incisor teeth and in the line of median palatine suture there is a pit called foramen incisivum. At the bottom of this may be seen openings of small canals ranging from one to four. These are arranged in two pairs—one being placed side by side and the other is placed in median plane, one forward and one behind.

Foramen of Stensen is one which is arranged in side by side and transmits the greater palatine arteries which ascend to reach the nasal cavities. The foramen of Scarpa is the pair





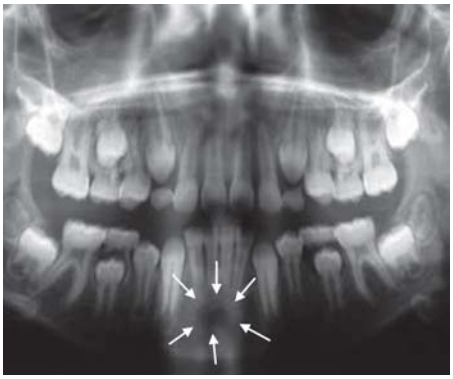
which is present in front and behind open into left and right nasal cavities respectively, pass left/right nasopalatine nerves respectively.

8. What are stalagmites of maxillary sinus?

Stalagmites are found on caves formed by percolating water. Antral floor sometimes show radiopacities that are small osseous excrescences and small bony masses resemble like stalagmites. They are formed on floor of antrum and seldom reach 3 mm height, are white masses. Differantiation from root tip has to be done as root tip shows shadow of pulp canal.

9. What is president's tumor?

In the past president of US was retiring from the office and as a part of administrative process of the retiring the president was scheduled for an extensive physical examination including a dental check-up and had undergone panoramic X-ray. There was a radiolucency observed at the root apices of anterior teeth in lower jaw. The consultants who observed all this radiolucency agreed that the nature of the radiolucency is pathologic. The intraoral radiographs taken in this area appeared completely normal and this panoramic radiolucency seen in individuals



with prominent depression in midline of the labial mental area. This pseudolesion on panoramic radiograph is called presidents tumor.

10. What is cervical burnout?

It is a radiolucent band at the neck of teeth radiographically appearing as radiolucent areas at mesial and distal surfaces at neck, cervical portion of tooth and crest of alveolar ridge. It is an artifact caused by less absorption of X-ray in that area. In the cervical area there is less tissue for X-ray beam to pass through and it leads less accentuation of X-ray beam, hence no opaque shadow is cast. Additionally, contrast of radiopaque enamel, dentin and alveolar bone also give perception.

11. Enumerate the differentiating points of cervical burnout and cervical caries.

	Cervical burnout	*Cervical caries*
The cause of radiolucency	– It is an artifact due to less attenuation of X-ray beam. – Anatomic differences shape of CEJ and various root configurations.	It defects in enamel and cementum.
Clinically	Normal	Capitation
Exposure factors	It is increased when exposure is greater and contrast is more	Not necessarily associated.
Affected teeth	Usually, all teeth on radiograph are affected specially smaller premolars.	It may be associated with affected tooth.
Condition of alveolar bone	Mostly the alveolar bone intacts as it is one of the factors for appearance.	If associated with cemental caries, the recession is present and alveolar bone loss may be seen.
CEJ	Knife edge at CEJ is intact.	Knife edge at CEJ is affected.

Contd.

Contd.

	Cervical burn out	*Cervical caries*
Associated with restoration	It is increased adjacent to metallic restoration.	It can be seen nearby the metallic prosthesis but the amount of radiolucency is not affected by the presence.
Location	Nearer to alveolar crest	Nearer to contact point.
Shape	Band shaped in anterior teeth or wedge (triangular) shaped, in posterior teeth. The triangle becomes less apparent towards the centre of tooth.	Mostly saucerized and irregular shaped.
Borders	Well-demarcated Bounded above by enamel cap or restoration and below by the alveolar bone.	Diffuse Apparently no upper and lower demarcating borders.
Defect in angulation	Seen when horizontal angulation is incorrect.	Seen horizontal angulation is correct.
Changing angulation	With different angulation the defect may not be seen.	It does not show change.

12. What is Mach band effect?

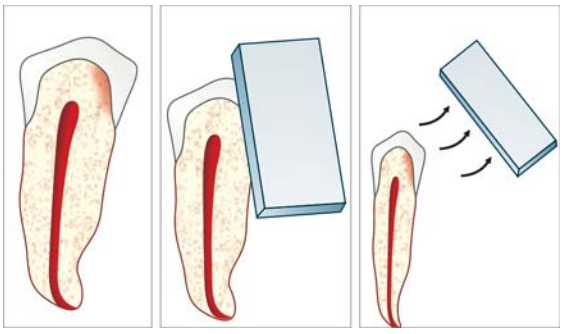
This is an optical illusion, described by Sir Ernst Mach in 1865. It occurs within retina and results from physiological process of lateral inhibition. It occurs at the junction of two regions of differing radiodensity. It is experienced upon viewing the enamel dentinal junction. The enamel margin immediately adjacent to DEJ is white, whereas the dentin tends to black.

The uniformly dark shade meets uniformly light shade (dentin meets enamel in radiograph), the dark shade (dentin) appears more darker and light shade (enamel) appears even more lighter as they appear DEJ (interface). This edge enhancement phenomenon does not result from actual density change in film emulsion but from lateral neural inhibitory interactions within the eye of the beholder. The light shade which is brighter, is called positive Mach band effect while darker appearance of dark shade is called negative Mach band effect. The negative Mach band effect may show fictitious Mach band effect resembling caries (common in incisors, canines, premolars less frequently and to the least extent in molars). The Mach band effect generally extends 0.5 mm below the DEJ.

13. How Mach band effect differentiated from actual carious lesion?

When there is confusion regarding differentiating Mach band from an actual carious lesion. The following experiment is done:

1. The radiograph under consideration is carefully examined for the negative Mach band effect.
2. The opaque card is used to cover the enamel of the radiograph (light shade) if Mach



(a) (b) (c)

Figure showing Mach band effect: (a) Dark shade meets light shade as dentin meets enamel, (b) Unmasking by putting an opaque object will make to disappear the dark shade, (c) It will again appear after unmasking.

band effect, i.e. radiolucency disappears then it is positive Mach band effect, but if radiolucency does not go off, then that is active carious lesion.

14. What is Wolff's law?

It has been stated by Sir Julius Wolff in late 1800s. It states that the number and the distribution of bony trabeculae are dependent on the strains and stresses to which the bone is subjected.

or

Every change in the form and function of bones or of their function alone is followed by certain definite changes in the configuration in accordance with mathematical laws.

For example, when the tooth is removed, the bone from which tooth has been removed appears to have less strain as compared to the bone if tooth is present, ultimately the bone in this area appears to be radiolucent and the trabeculae less organized.

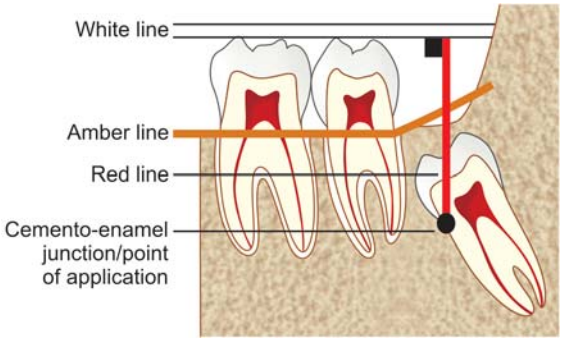
15. What are Winter lines?

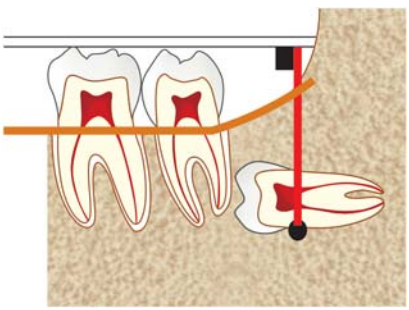
These are called war lines of Winter. The position and depth of an impacted third molar can be assessed by taking intraoral X-ray or even lateral extraoral X-ray and tracing can be done, which was originally advocated by Sir George Winter. Three imaginary lines are drawn which are known as 'Winter's lines'.

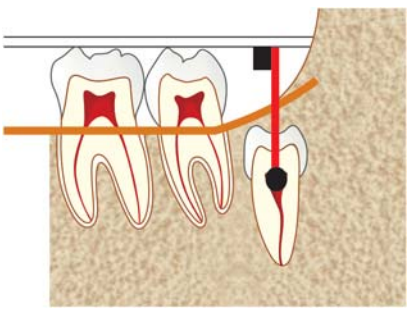
White line corresponds to the occlusal plane. The line is drawn touching the occlusal surfaces of first and second molar and is extended posteriorly over the third molar region. It indicates the difference in occlusal level of second and third molars. It indicates depth of impaction.

Amber line represents the bone level. The line is drawn from the crest of the interdental septum between the molars and extended posteriorly distal to third molar or to the ascending ramus. This line denotes the alveolar bone covering the impacted tooth and the portion of tooth not covered by the bone.

Red line is drawn perpendicular from the amber line to an imaginary point of application of the elevator. It indicates the amount of bone that will have to be removed before elevation, i.e. the depth of the tooth in bone and the difficulty encountered in removing the tooth. Howe said that 1 mm increase in length causes three times difficulty.







If the length of red line is 5 mm or less, then the tooth can be conveniently removed. If the length is more than 5 mm, it has to undergo extraction under GA or with sedation.

16. How the caries is classified on radiograph?

E0—No visible radiographic caries lesion

E1—Lesion in outer one-half of enamel.

E2—Lesion in the inner one-half of enamel

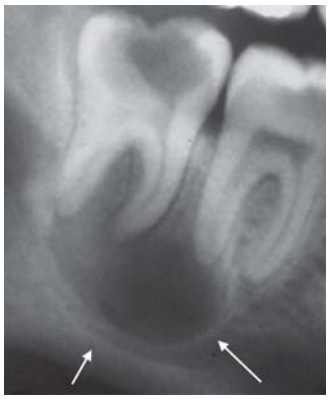
D1—Lesion in outer one-third of dentin

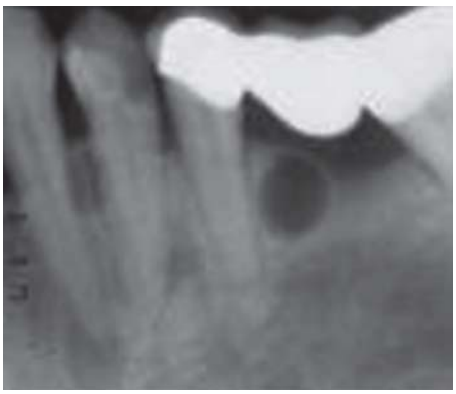
D2—Lesion in middle one-third of dentin

D3—Lesion in inner one-third of dentin.

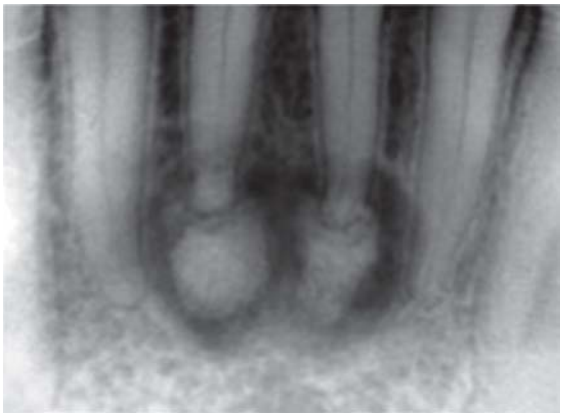
17. What are sclerotic and corticated border? Name conditions in which sclerotic and corticated borders are seen.

A corticated margin is a thin, fairly uniform radiopaque line of reactive bone at the periphery of the lesion. This is commonly seen in cyst. Lobulated and multilocular lesions corticated with scalloping.



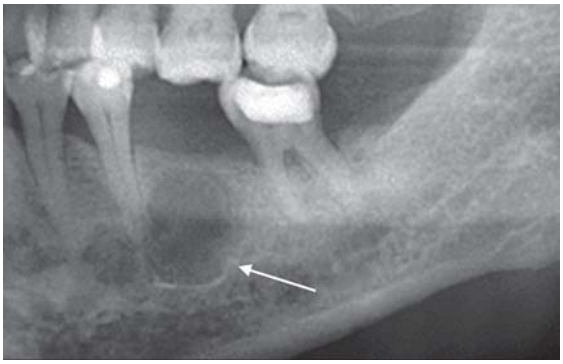


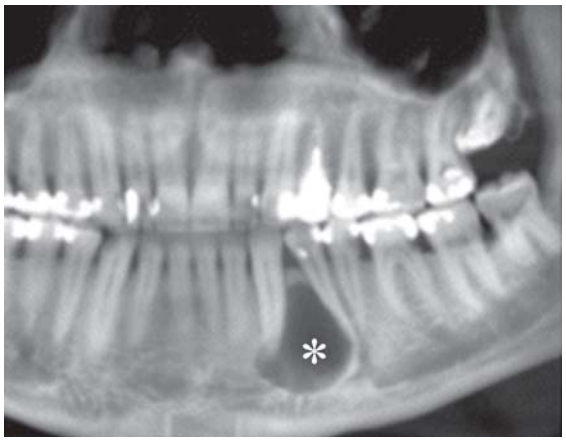
A sclerotic margin is a wide, radiopaque border of reactive bone that usually is not uniform in width. This may be seen in periapical cemental dysplasia and may indicate a very slow growth rate or potential for the lesion to stimulate the production of bone. Unilocular lesions display well-defined sclerotic border. Most chronic inflammatory bony lesions demonstrate sclerotic border which reflects the reaction of surrounding trabecular bone to inflammation. Some malignant lesions may demonstrate sclerotic border.



18. What are diagnostic criteria of odontogenic and nonodontogenic lesions on radiograph?

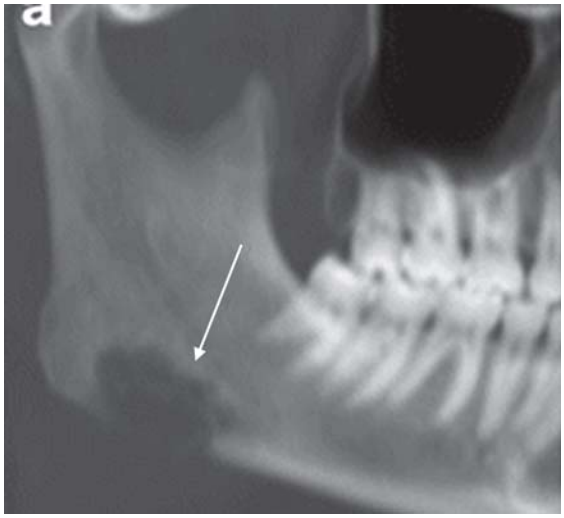
If the epicentre of the lesion is above the inferior alveolar canal, it is suggestive that the lesion is probably composed of odontogenic tissue. For example, residual cyst, radicular cyst.

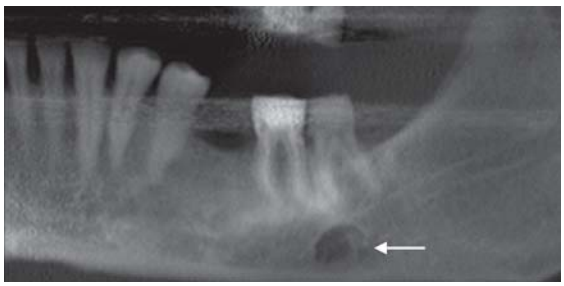


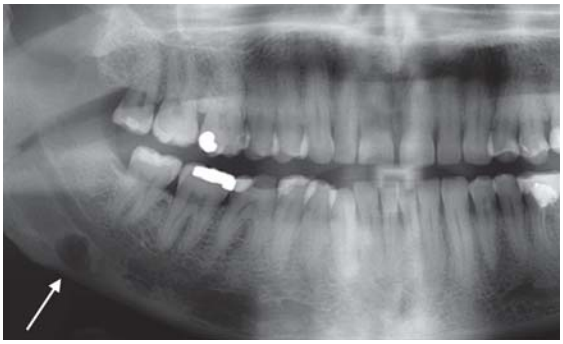


If the epicentre of the lesion is below the inferior alveolar canal, it is less likely to be odontogenic origin and mostly nonodontogenic in nature, e.g. Stafne's bone cyst.

The neural and vascular lesions have the epicentre in the inferior alveolar canal.



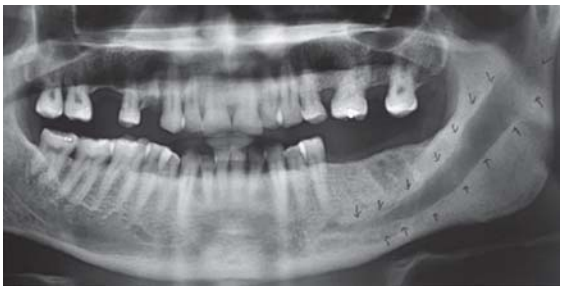


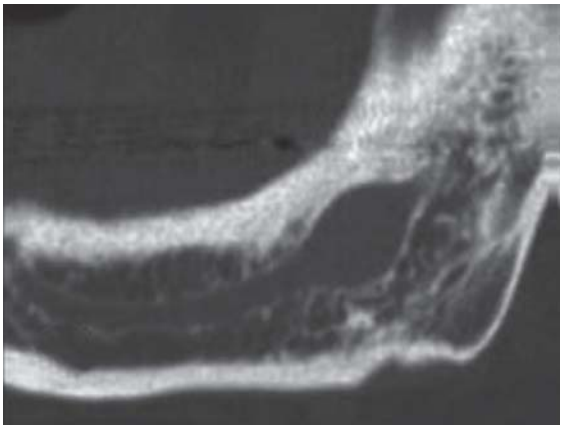


The cartilaginous lesions originate in condyle.

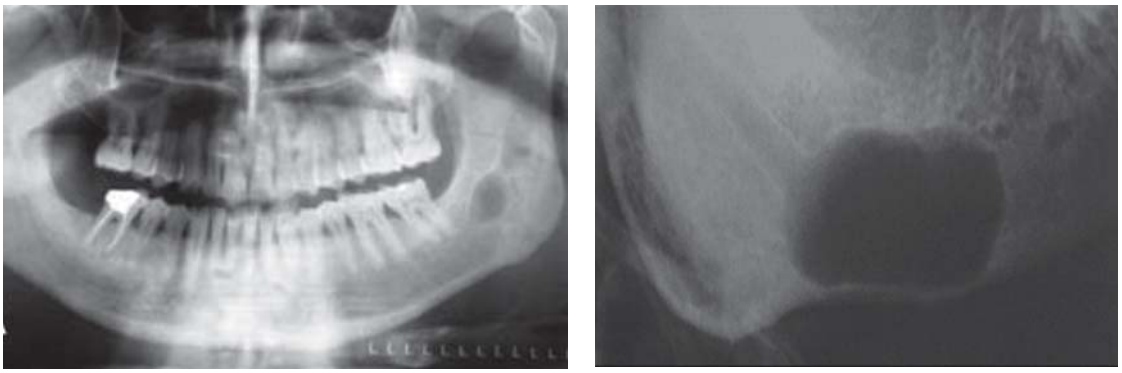
19. What are the characteristics of neural and vascular lesions?

Neural neoplasms cause expansion in more concentric fashion creating fusiform shape while vascular lesions increase the girth of the canal down the entire length and often a shape into serpiginous form.

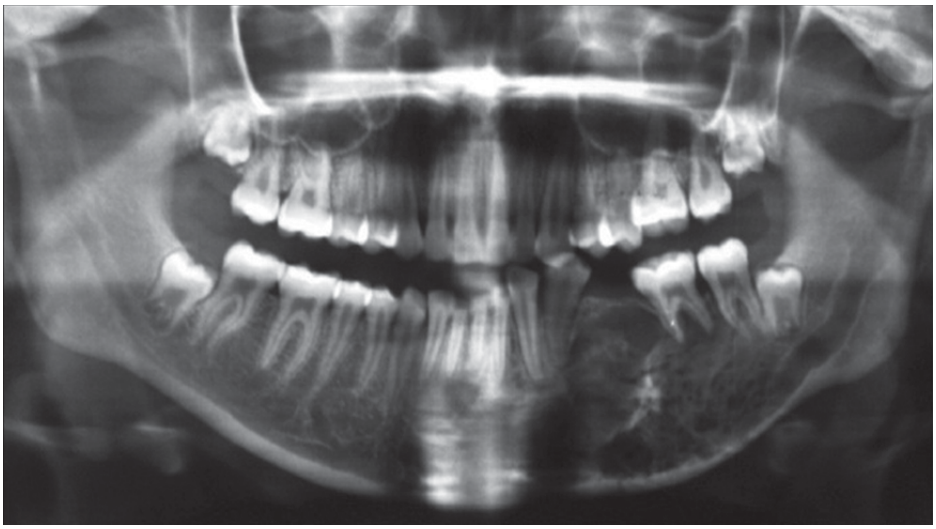




Neural lesion involving entire length and fusiform shaped



Fusiform expansion indicating neural lesion



Vascular lesion showing widening of inferior canal in serpiginous form

20. What is mineralization? Name the lesions with and without mineralization.

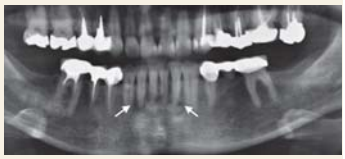
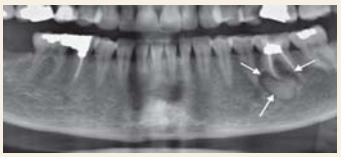
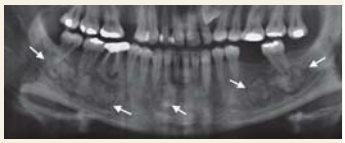
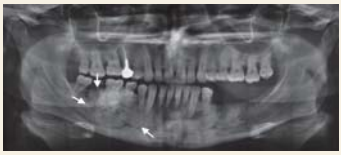
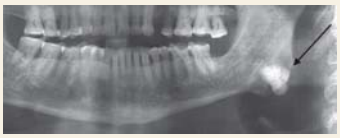
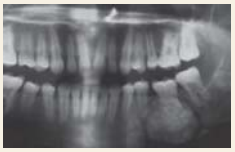
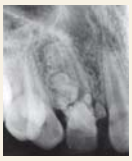
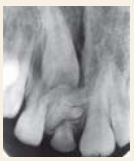
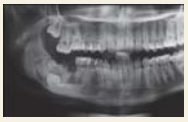
Mineralization refers to the elaboration of mineralized products by the lesion itself; such products include enamel, dentin, and cementum or cementum like calcified tissue. Mineralization produces lesions with varying degrees of opacity and are classically described as radiopaque or lesions of mixed opacity. Nonmineralized odontogenic lesions are classically radiolucent and fail to demonstrate internal mineralization.

Odontogenic lesions with mineralization—odontoma, odontogenic myxoma.

Odontogenic lesions without mineralization—ameloblastoma, keratocystic odontogenic tumor and radicular cyst (this may show mineralization in chronic cases).

21. What is radiolucent rim around lesion? Give the differential diagnosis of lesions having radiolucent rims.

A radiolucent rim or soft tissue capsule is a kind of well-defined border at the periphery of radiopaque jaw lesions that presents as a radiolucent line. It can be observed in association with a corticated border.

Entity	*Age/sex*	*Common location*	*Distinguishing features*	*Diagrammatic representation*
Periapical cemento-osseous dysplasia	>30 yrs F>M	Anterior mandible	Vital teeth, <1 cm often multiple, circular size	
Focal cemento-osseous dysplasia	Mean age 37 yrs F>M	Posterior mandible	Local jaw expansion and mild discomfort	
Florid cemento-osseous dysplasia	Middle to elderly F>M	Premolar molar area both jaws	Bilateral, symmetrical extensive	
Cemento-ossifying fibroma	10–40 yrs F>M	Molar mandible	Round, expansive, painless, slow growth. Displacement, root resorption	
Osteoid osteoma	Mean 19 yrs F>M	Long bones jaws 1%	Nocturnal pain	
Osteoblastoma	Mean 23 yrs F>M	Body of mandible	Slight pain, swelling, expansion	
Compound odontoma	Children and adolescent F>M	Anterior maxilla	Denticles, pericoronal lesion	 
Complex odontoma	F>M	Posterior mandible	Slow now aggressive	
Cementoblastoma	10–30 yrs M>F	First molar mandible	Asymptomatic, nodular, expansive	  

22. Enumerate the multilocular radiolucencies.

Ameloblastoma
Central giant cell granuloma
Giant cell lesion of hyperparathyroidism
Cherubism
Odontogenic myxoma
Odontogenic keratocyst
Aneurysmal bone cyst
Metastatic tumors to the jaws
Vascular malformations and central hemangioma of bone.

Rarities

Ameloblastic variants, arteriovenous malformations, Burkitt's lymphoma, calcifying epithelial odontogenic tumor, cemento-ossifying fibroma, central calcifying odontogenic cyst, central giant cell tumor, central salivary gland tumors, central odontogenic and nonodontogenic fibromas, chondroma, chondrosarcoma, fibromatosis, fibrous dysplasia, fibro-odontogenic dysplasia, hemangiopericytoma, immature odontoma Langerhans' cell disease (eosinophilic granuloma), leiomyoma lingual mandibular bone defect, neurilemoma, neuroectodermal tumor of infancy, osteomyelitis pseudotumor of hemophilia, squamous odontogenic tumor.

23. Enumerate the multilocular lesions associated with mandibular expansion.

Three entities well-known to cause mandibular expansion with a multilocular presence are the ameloblastoma, the odontogenic myxoma, and the central giant cell tumor. Less common, but well-known to be multilocular, is a central arteriovenous hemangioma of the jaws. In addition, if the lesion is a small, multilocular lesion between premolar or canine teeth, a botryoid odontogenic cyst is a strong consideration.

24. What is Garrington's sign?

It is a widening of periodontal ligament space symmetrically on radiograph is an early sign of osteosarcoma. It is also seen in chondrosarcoma.

25. Enumerate the conditions causing ground glass appearance.

Fibrous dysplasia, hyperparathyrodism, Paget's disease of bone.

26. Name the conditions causing generalised loss of lamina dura.

Hyperparathyroidism and Paget's disease, fibrous dysplasia, osteomalacia, rickets, multiple myeloma, osteoporosis, Pyle's disease, hypophosphatasia, renal osteodystrophy and leukemia. While thinning is seen in osteoporosis and Cushing's syndrome.

27. Name the conditions in which thickening of lamina dura is seen.

Local trauma from occlusion. Marked malposition or served as abutments for fixed bridges. Systemic hypoparathyroidism and bisphosphonate related osteonecrosis of jaw.

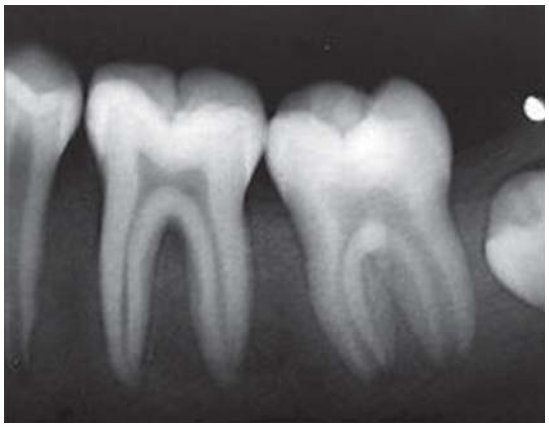
28. Enumerate the radiographic signs of trauma from occlusion.

Widening of periodontal membrane space, thickening of lamina dura, hypertrophy of cementum and root resorption.

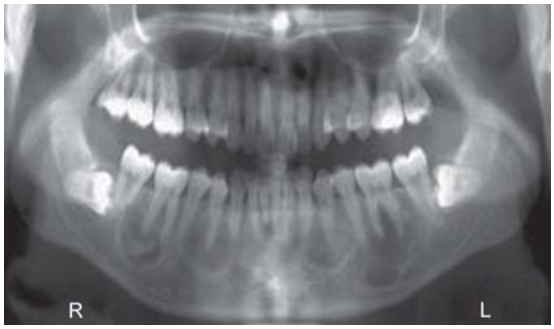
29. What are various radiographic appearances of hyperparathyroidism?

Only one in five patients has got noticeable radiographic changes:

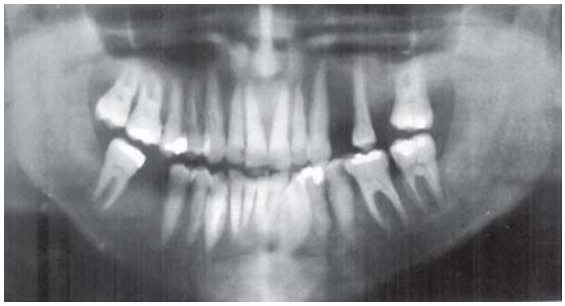
1. Earliest and reliable manifestation is subtle erosions of bone from the subperiosteal surfaces of phalanges of hands.

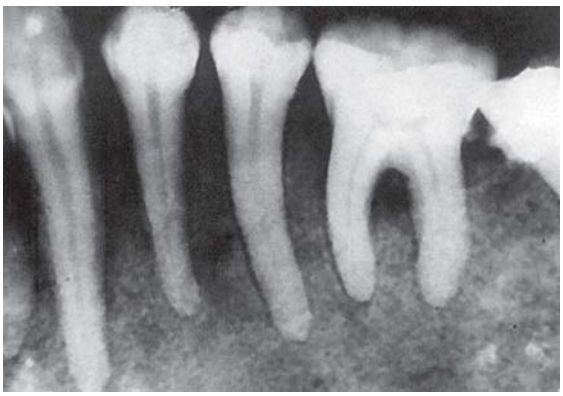


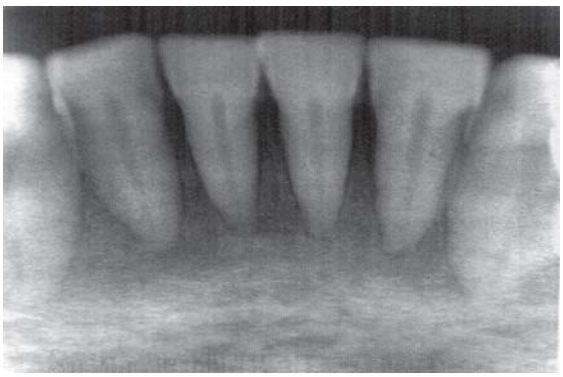
2. Unusual radiolucent appearance from demineralization of bone.



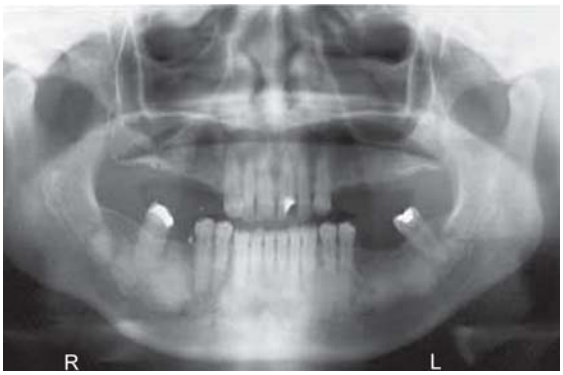
3. Osteitis fibrosa cystic-localized loss of all bone/replaced by fibrous tissue.
4. Punctate/nodular appearances of pathologic calcifications in the joints and kidneys.
5. Demineralization of skull shows radiolucent appearance. Entire calvarium has a granular appearance (in prominent hyperparathyroidism) due to loss of central (diploic) trabeculae and thinning of cortical tables.
6. Radiograph of skull shows 'pepper-pot' appearance as medullary/inner and outer cortices lose their differential pattern.
7. Brown tumor in late of disease (about 10% of cases). These peripheral or central tumors of bone are radiolucent. The gross specimen has a brown or reddish-brown colour.
8. Loss of lamina dura complete or partial, may involve one or several teeth (10% cases) may give the tooth a tapered appearance.





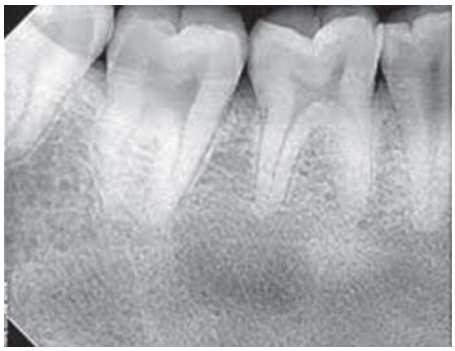


9. Demineralization and thinning of cortical boundaries often occur in the jaws in cortical boundaries such as the inferior border, mandibular canal, and the cortical outlines of the maxillary sinuses.

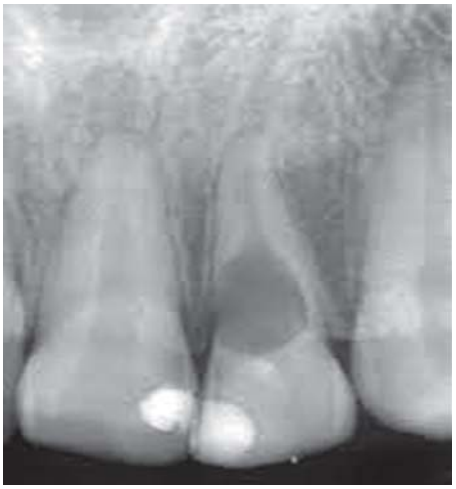


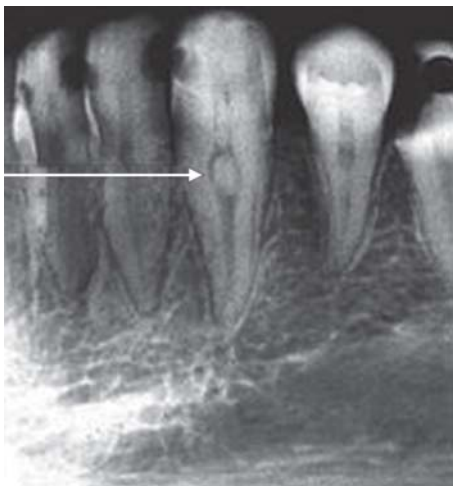
10. The density of the jaws is decreased, resulting in a radiolucent appearance that contrasts with the density of the teeth. The teeth standout in contrast to the radiolucent jaws.

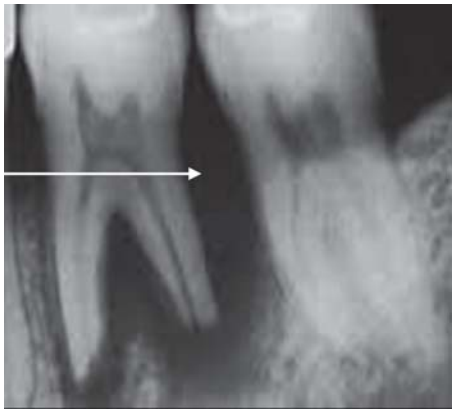
11. A change in the normal trabecular pattern may occur, resulting in a ground glass appearance of numerous, small, randomly oriented trabeculae.



12. Pulp stones and root resorption.







30. What are radiological appearances suggestive of cancers?

- Radiolucency with ragged and vague borders.
- Band-like widening of the periodontal ligament.
- Combined radiolucent-radiopaque lesion with a vague pattern.
- Radiopacity with vague borders.
- Sunburst appearance from the border of the bone. Possibly combined with changes in the first three appearances.
- Hanging tooth or naked tooth.
- Onion skin appearance from the border of the bone.
- Possibly combined with changes in the first three appearances.

31. What are the radiographic appearances of osteogenic sarcoma?

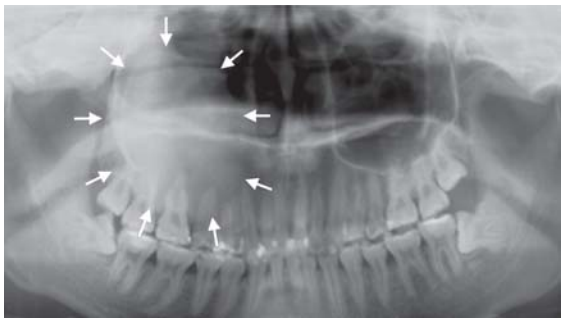
Generally, one of the radiographic features of osteogenic sarcoma is apparent: Sunburst appearance; cumulus cloud appearance; Codman's triangle; asymmetric, band-like widening of the periodontal ligament; or onion skin appearance of redundancy of the cortical plate.

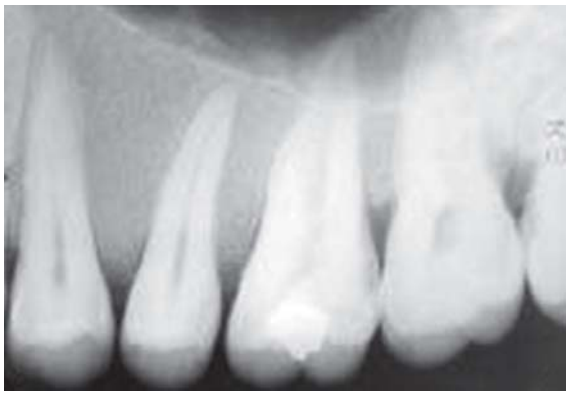
32. Describe variable radiographic appearances of fibrous dysplasia.

Fibrous dysplasia is defined as a benign osseous disease characterised by a process of normal bone resorption, followed by an abnormal proliferation of a disorganised

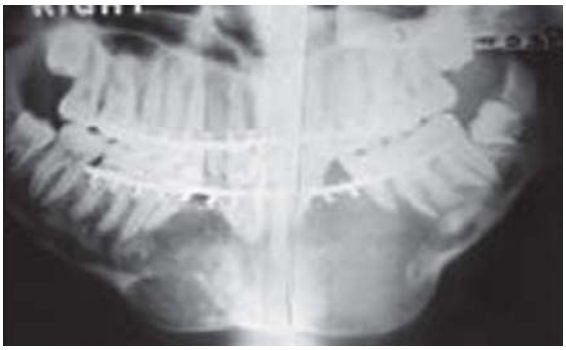
fibro-osseous tissue. It ranges from a radiolucent lesion to radiopaque mass. The borders are poorly defined and blending:

1. The classic lesion is ground glass (peau d'orange effect). This effect is more common on I/O radiographs. This is also called frosted glass appearance, it appears on CT also. There is homogeneous radiopacity by myriad dispersed minute spicules of bone within bone.

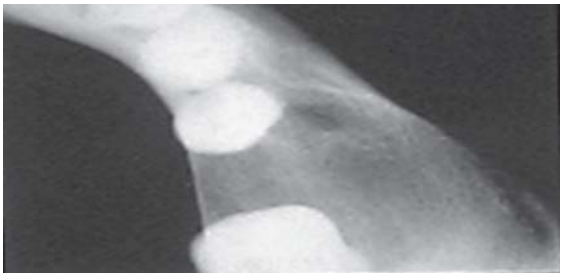




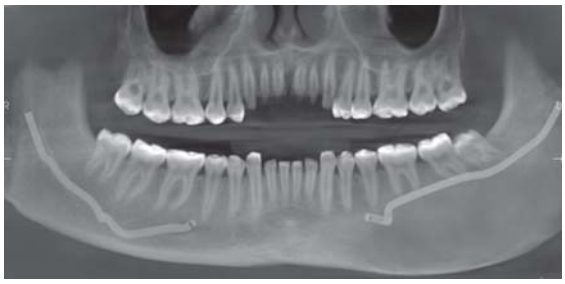
2. The plain radiographic features are classified into: (1) Pagetoid (56%) it appears radiographically similar to Paget's disease having radiolucency with patchy, irregular opacities resulting in mottled radiographic appearance, (2) Sclerotic (23%), (3) Cystic (21%).
3. Unilocular/multilocular appearances in long bones. Long-standing disease may show mottled radiolucent and radiopaque appearance.



4. Fingerprint appearance.



5. Superior displacement of mandibular canal is classic feature.



Radiographic appearance may vary according to age and history of tumor.

In very young patients with rapidly expanding fibrous dysplasia multilocular radiolucent lesion with cortical thinning is seen. The teeth may spread apart and evidence of dental eruption. In older patients with clinically quiescent lesions it may show mixed radiolucent/radiopaque or that of simply radiopaque mass.

The radiographic appearance in FD varies greatly depending upon the stage into 3 types:

Type 1: A small unilocular or multilocular radiolucency with a well-circumscribed border containing a network of fine bone trabeculae.

Type 2: Similar but with increased trabeculation rendering a more opaque and mottled appearance.

Type 3: The lesion is quite opaque with many delicate trabeculae giving a ground glass or peau d'orange appearance to the lesion. This type is not well-circumscribed but blends with the normal bone.

Fibrous dysplasia and maxillary sinus: It may expand into antrum by displacing its cortical boundary and subsequently occupying most of maxillary sinus. Extension occurs from lateral wall and last section is posterorsuperior portion.

The lamina dura disappears and abnormal bone pattern is laid down.

Superior displacement of inferior nerve canal is characteristic of fibrous dysplasia.

33. What are the radiographic appearances of chronic osteomyelitis?

Chronic osteomyelitis may demonstrate four distinct radiographic pictures—completely radiolucent, mixed radiolucent and radiopaque, completely radiopaque, and proliferative periostitis. The last can be recognized as a somewhat opaque layering of the periosteum, with bone proliferating peripherally.

It varies according to underlying inflammatory response and age of patient.

Acute Osteomyelitis

1. Ragged, patchy, moth-eaten area of radiolucency. The outline of the area of destruction is poorly defined.
2. Evidence of small radiopaque sequestrate of dead bone occasionally within the radiolucency.
3. Evidence of subperiosteally bone formation, usually beyond the area of necrosis, particularly along the lower border of mandible.

Chronic Osteomyelitis

1. Localized or moth-eaten areas of bone destruction.
2. Sclerosis of the surrounding bone.
3. Evidence of small radiopaque sequestra of dead bone sometimes within the area of bone destruction.
4. Evidence of an involucrum surrounding the area of destruction following extensive subperiosteal bone formation.

The main finding is progressive radiopacity with effacement of cortico-cancellous junction histologically correlate to bone sclerosis this may be associated with radiolucency corresponding to small resorptive defects which are vascularised.

Radiolucent lines that intersect of radiopaque cortical bone are indicative of sequestration.

Chronic focal sclerosing osteomyelitis (condensing ostetitis) radiodensity localized to apex of tooth.

Diffuse sclerosing osteomyelitis seen in older patients may be showing diffuse radiopacity in edentulous region. The diffuse patchy sclerosis is often described as cotton wool appearace similar to Paget's disease of bone. It may be extensive and sometimes bilateral and may involve maxilla and mandible.

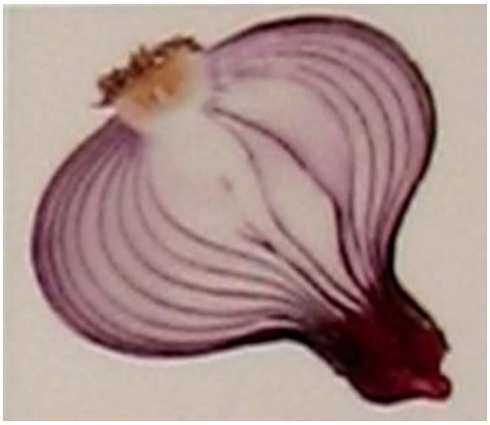
Proliferative periostitis incease mandibular thickening common in children, the thickening relates to multilamellar periosteal deposition.

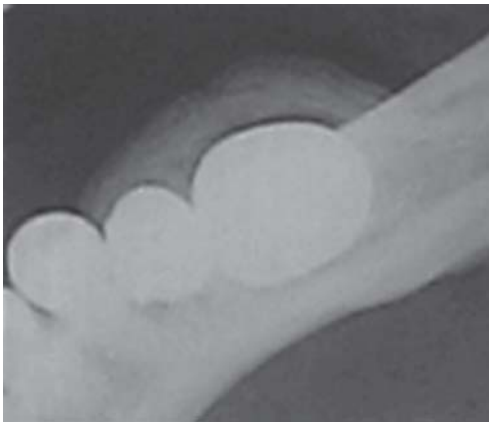
Primary osteomyelitis (course is insidious, lacking an acute stage)	Secondary osteomyelitis (more than 4 wk).
Areas of increased radiopacity with loss of bony trabeculae, effacement of cortico-cancellous bone junction affecting hemimandible.	Areas of increased radiopacity with loss of bone trabeculae
Minor part of radiolucency	Sequestra formation
Rarely periosteal reaction	Calcified periosteal reaction
Temporomandibular joint involvement	Pathological fractures
Stable radiographic changes may undergo mild changes in case of relapse.	Normal bone structures expected after healing.

Histologically sclerotic bone deposition and trabecular spaces filled with cellular connective tissue component.

Chronic recurrent multifocal osteomyelitis (CRMO) shows diffuse bony radiopacity.

Garré's osteomyelitis (chronic osteomyelitis with proliferative periostitis) young patients with onion skin appearance.





34. Enumerate the sign suggestive of close relation of inferior alveolar canal and mandibular roots.

Howe and Payton (1960) suggested the signs.

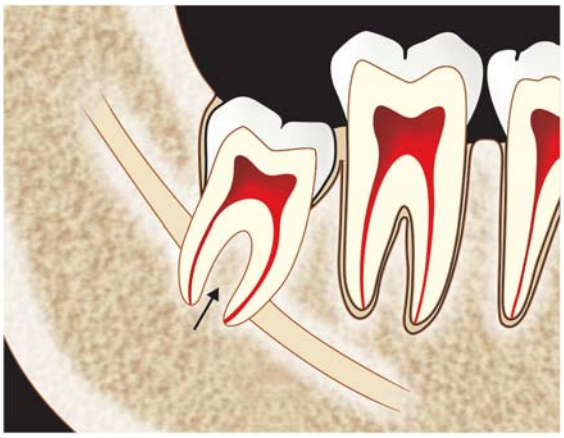
These are seen on:

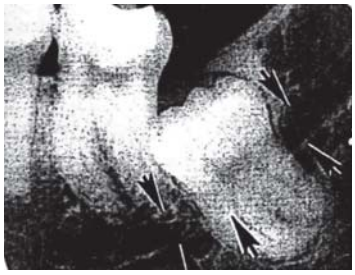
1. **Teeth roots**
   a. *Darkening of the root:* When there was impingment of canal on tooth root there was loss of density of tooth root and the root appears dark (dark band phenomenon).
   b. *Deflected roots:* Deflected roots or root hooks were seen as an abrupt deviation when the root approaches the canal.
   c. *Narrowing of root:* It is seen when inferior alveolar canal crossed the apex and identified by double periodontal membrane space at the apex.
   d. *Bull's eye:* Overlapping of buccal and lingual root tips.
   e. Illustration of discontinuous image of the mandibular canal (canal discontinuous phenomenon).
2. **Inferior alveolar canal**
   a. *Interruption of white lines (danger sign showing true relationship with inferior alveolar canal and tooth):* The corticated border (superior and inferior) of inferior alveolar canal is observed. It is considered to be interrupted if it disappeared immediately before it reaches apex. It indicates:
      1. Deep grooving if it occurs along the root.
      2. Perforation of the root if it is seen with narrowing of root canal.

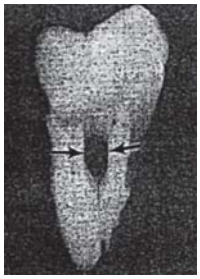
b. *Diversion of the inferior alveolar canal:* It is considered to be diverted, if while crossing the inferior canal there was a change of its direction.
c. *Narrowing of inferior canal:* It is considered to be narrowed when the root of mandibular third molar was crossed by it, showing reduction in diameter.
d. *The hourglass form:* It indicates the displacement of upper/lower border toward each other. It indicates a partial or complete encirclement of canal.

35. What is canal discontinuous phenomenon?

The radiograph shows discontinuation of image of mandibular canal as it crosses the roots of mandibular third molars. This also shows close proximity of roots.

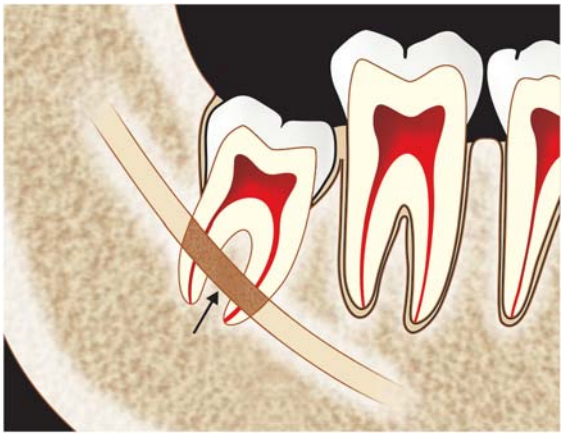


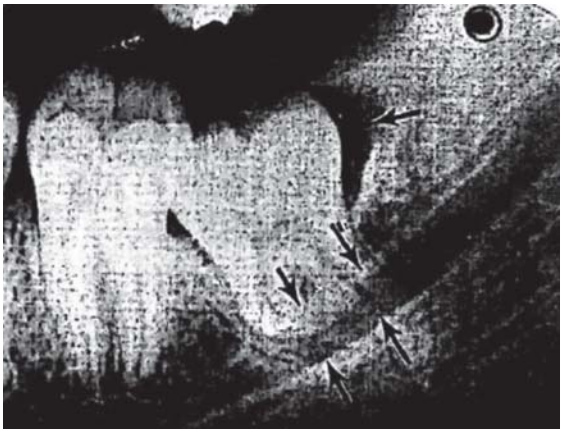




36. What is dark band phenomenon? What are the other factors considered for proximity of root surface?

The proximity of impacted mandibular third molars roots to inferior alveolar canal has been the radiograph of an impacted mandibular third molar, the radiolucent shadow of mandibular canal appears as a dark band crossing the roots of third molar indicates that the canal is probably in close proximity of roots. The constriction of canal while crossing the roots of impacted mandibular third molar indicates that the canal is in close proximity with roots. The darkening of roots is sometimes indicative of notching of roots.





37. Enumerate different types of periosteal reactions and give examples of each.

Periosteal reaction (periostitis/periosteitis) is a nonspecific radiographic finding that occurs with the periosteal irritation. The periosteal reactions result when cortical bone reacts to one of many possible insults (tumor, infection, trauma, certain drugs, and arthritic changes, etc.). The appearance depends upon the intensity.

In children the reactions are aggressive and appear more earlier as the periosteum is more active and less adherent to underlying cortex. In jaws these are rare and probably the imaging is not sufficient to visualize them. It has been thought to arise after the periosteum is stripped from the cortex. The frequency of periosteal group is highest in osteomyelitis group followed by malignant conditions. The intra-articular lesions show no periosteal reaction because there is no periosteum intra-articularly.

Classification

These are classified as benign and aggressive reactions.

Benign Periosteal Reaction

Low-grade chronic irritation allows time for the formation of normal or near-normal cortex. The cortex will be thick and dense and have a wavy or uniform appearance. Benign periosteal reactions can be seen in callus formation in a fracture or with slowly growing tumours.

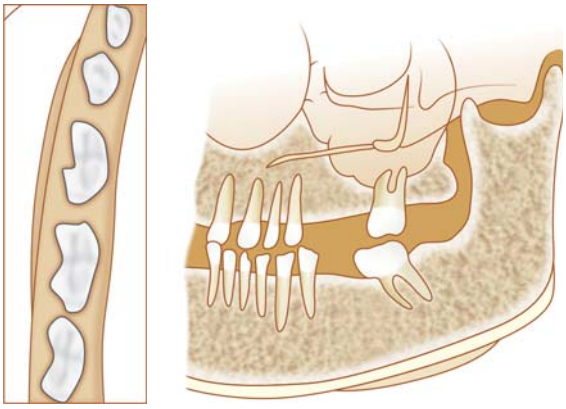
Aggressive Periosteal Reaction

Rapid irritative processes do not allow the periosteum time to lay down and consolidate new bone to form normal cortex. The cortex may appear lamellated, amorphous, or sunburst-like. Aggressive periosteal reactions cannot only be seen with malignant tumors, but also with more benign processes like infection, eosinophilic granuloma (Langerhans' cell histiocytosis), aneurysmal bone cyst, osteoid osteoma, and trauma.

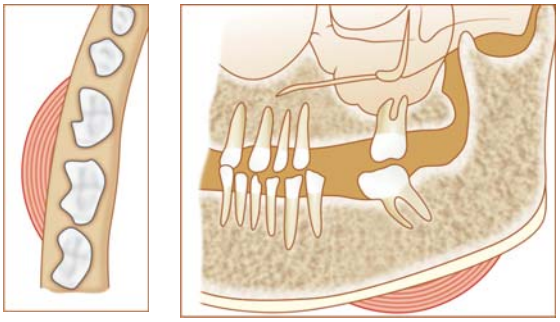
Uniqueness for Specific Reaction Process

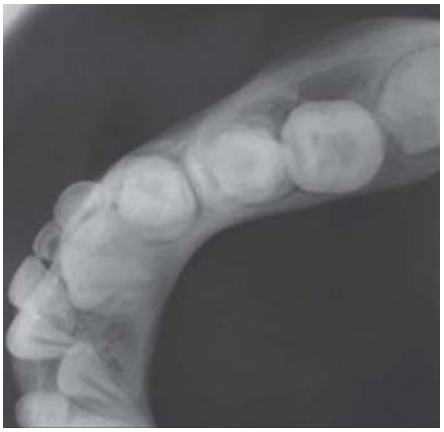
Although no specific reaction is unique for any process:

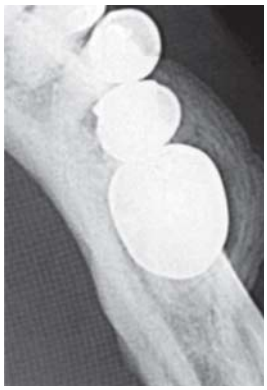
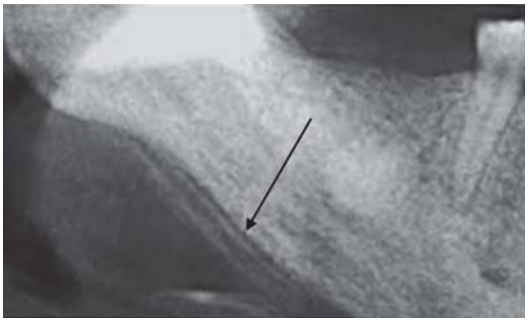
1. **The uninterrupted lamellar:** An uninterrupted indicates a benign process, which is slow growing, indolent and long-standing. It is seen in osteomyelitis and may be only radiologic manifestation. As the process is slow growing the periosteum has ample time to respond and can produce new bone as fast as the lesion is growing and may produce uninterrupted new bone along the margins of the affected bone.



2. **The onion skin pattern (lamellated):** It is a multilayered periosteal reaction and demonstrates multiple concentric parallel layers of new bone adjacent to cortex, reminiscent of layers on an onion. The layers are thought to be result of variable growth. It indicates more aggressive process, as the periosteum cannot produce the bone as fast as the lesion is growing so intermittent bouts are seen and not as solid

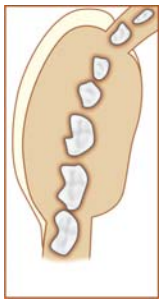
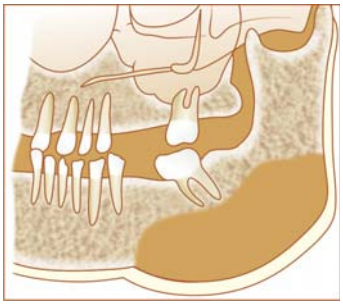




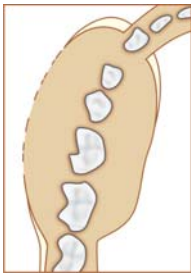
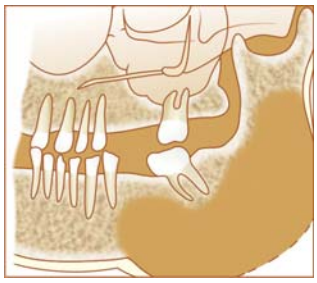




thick compact mass. Garré's osteomyelitis (other conditions are Ewing's sarcoma, chondrosarcoma, osteosarcoma) as many as 12 laminations are seen. The affected areas show increased radiodensity which may be attributed to osteosclerosis. It is seen in panoramic radiographs, reverse Townes view, PA views. The occlusal view is best to see these reactions.

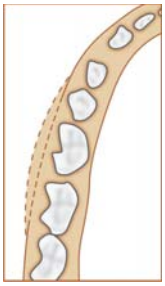
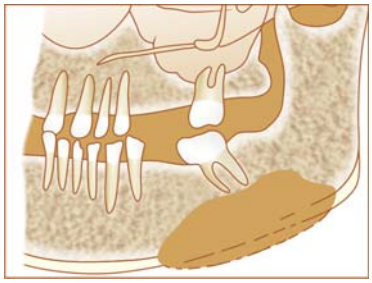
3. The thick compact type is most commonly noted with resolving Garré's osteomyelitis (individual lamenations become indistinct), osteoid osteoma, odontogenic fibroma. The periosteum here has got ample time to produce the bone.

4. **Buttress formation:** It is seen with slow growing process especially tumors. It produces triangular bone and invisible expanded cortical margins. These slow growing tumor produce focal thickening. The cortex beneath the buttress is frequently intact, e.g. ameloblastomas and ameloblastic fibromas. CT may be correct modality to visualize.

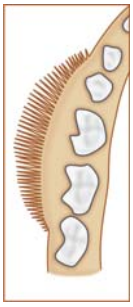



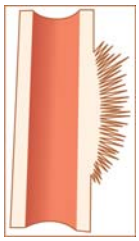
5. **Interrupted lamellar:** It is seen in osteosarcoma and its distinction is important. The subperiosteal layer is disturbed and not continuous.

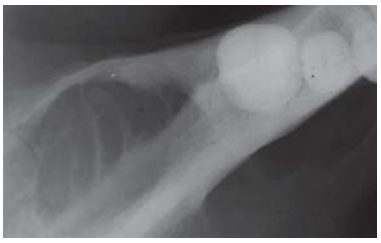



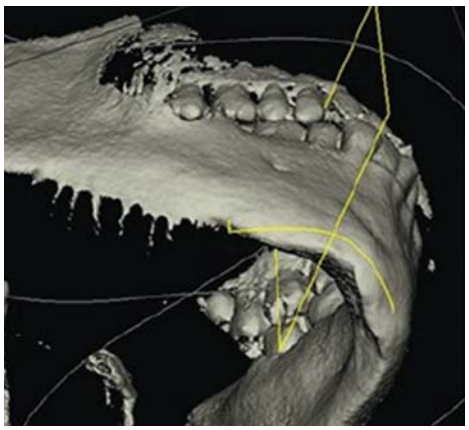
6. **The solid irregular mass:** Diffused masses are of radiopacity and seen in osteosarcoma.

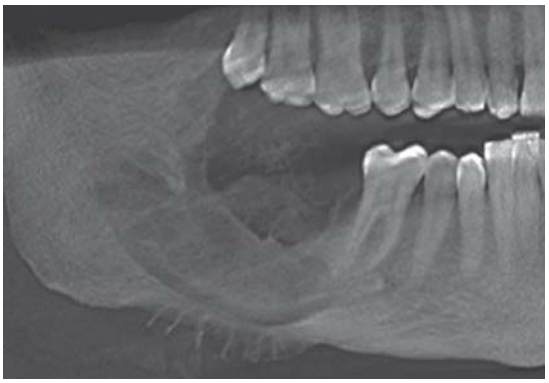
7. **The sunburst appearance is seen in osteosarcoma and chondrosarcoma:** This is an aggressive form of periosteal reaction. Perforation and expansion of the cortical margin by stretching periosteum this appearance is produced. In the sunburst subtype of periosteal reaction, the spicules of new bone radiate in a divergent pattern instead of perpendicular to the cortex. The spicules are not straight but forms irregular, wavy lines. Some authors claim that this is not a true periosteal reaction but outgrowth of tumor matrix, e.g. odontomas, odontogenic myxomas, occasionally in intrabony vascular malformations and intraosseous hemangioma.



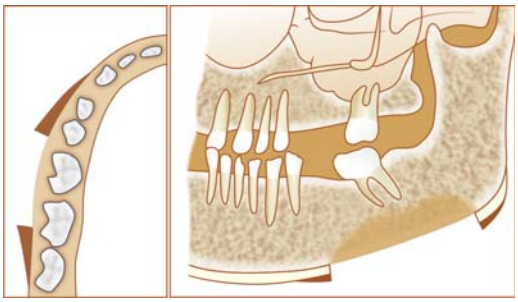


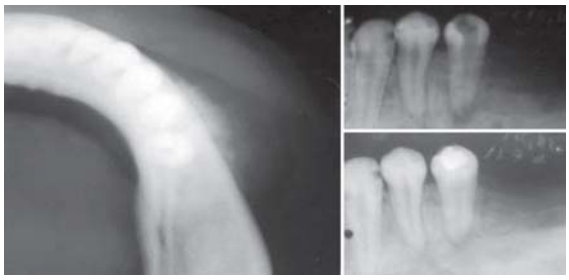


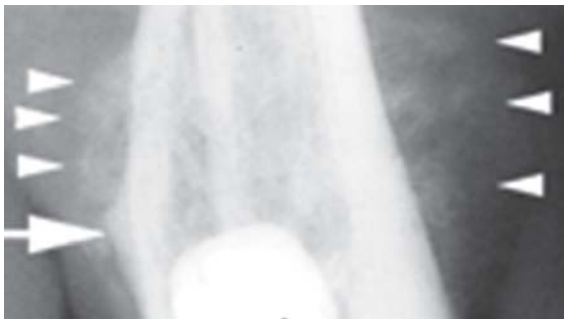




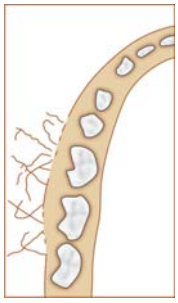
8. **Codman's triangle:** This is present in bone sarcomas and consists of entirely periosteal bone and should be avoided in taking biopsy. This is a bit misnomer as there is no complete triangle. In fast growing process the periosteum is raised and edges will ossify, as the lesion is aggressive and has no time to ossify. Periosteal newborn formation with tent like lifting of the cortex (due to tumor, pus and hemorrhage) leads to the appearance of a Codman's triangle. It forms an angle when a little bit of ossification is seen tangentially. Taking of biopsy from this region should be avoided.

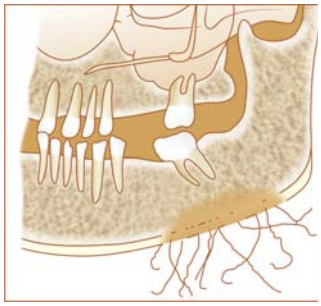




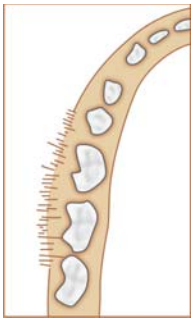


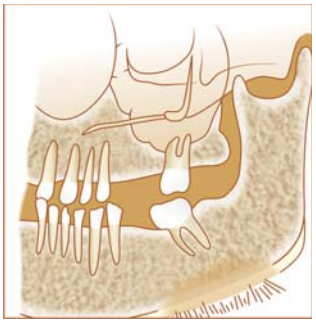
9. Irregular spicules: These form pattern of interlacing and relatively coarse spicules. It is seen in osteosarcoma. The spicules are much more disorganized manner and bizzare pattern.

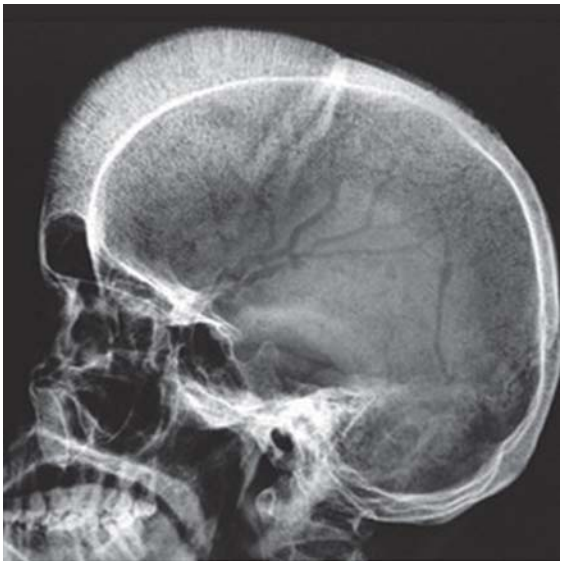


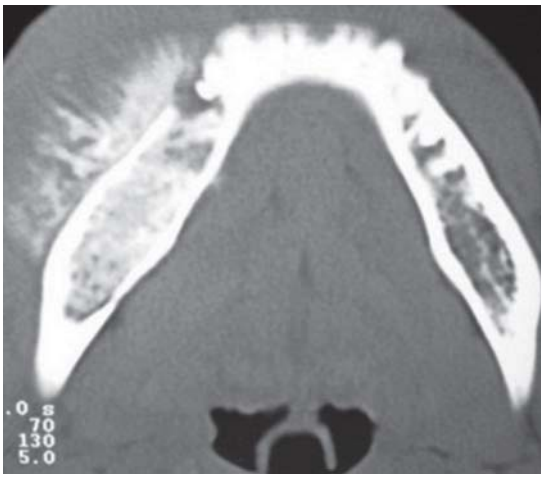


10. **Hair-on:** It is appearance of long, thin vertical striations of calcified spicules perpendicular to bone surface that look like hair on end. The end appearance rare in jaws may be seen in Ewing's sarcoma. The spicules are fine and short sometimes in odontogenic myxoma. On skull it is classically seen in thalassemia and sickle cell anemia. The hair-on-end sign is a finding seen in the diploic space on skull radiographs and has the appearance of long, thin vertical striations of calcified spicules perpendicular to bone surface that look like hair standing on end. It is classically seen in children/adolescents with hemolytic anemias, in particular, thalassemia major. Hair-on-end appearance refers to the skull abnormalities seen predominantly in patients with hemolytic anemias, that is, thalassemia major, sickle cell anemia, pyruvate kinase deficiency—hereditary elliptocytosis and spherocytosis. It is due to periosteal reaction with neo-osteogenesis of the outer cranial table which results in marked calvarial thickening, external displacement and thinning of the inner table. The changes are due to marrow hyperplasia. Hair-on-end appearance is also seen in congenital syphilis-syphilitic periostitis of tibia, metastatic neuroblastoma, iron-deficiency









anemia, cyanotic-right-to-left shunt-congenital heart disease, osteomyelitis, polycythemia vera, thyroid acropachy and hemangiomas. Similar appearance in facial bones is rare and suggests an extreme degree of medullary erythropoiesis.

38. Enumerate the periosteal bone reactions seen in osteomyelitis.

Uninterrupted lamellar, interrupted lamellar, onion skin, thick compact type of cortical thickening.

39. Enumerate what are the patterns of periosteal reaction sarcomas can show.

Onion skin appearance, interrupted lamellar, solid irregular mass, Codman's triangle, sunburst appearance, irregular spicules, hair on end.

40. How is the pattern of destruction of cortex present in the vicinity of periosteal reaction?

The pattern of the destruction of the cortical bone around the site of the periosteal reaction is depicted on CT. This pattern is categorized into the following four types:

1. No destruction

2. Point destruction where there is a small defect in the cortex with or without a defect in the trabecular bone.

3. Gross destruction which is continuous with a large defect in the trabecular bone; and

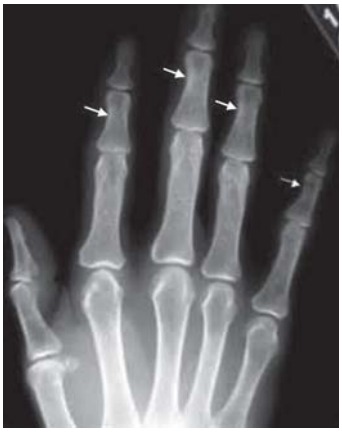
4. Permeative destruction in which numerous small defects are present in the cortex with a large defect or rarefaction of the trabecular bone.

41. Enumerate what are the patterns of periosteal reactions odontogenic myxoma may show.

The sunburst pattern, irregular spicules and hair on end.

42. What is brush-fire sign?

Subperiosteal erosion of the radial surface of middle phalanges of the second and third



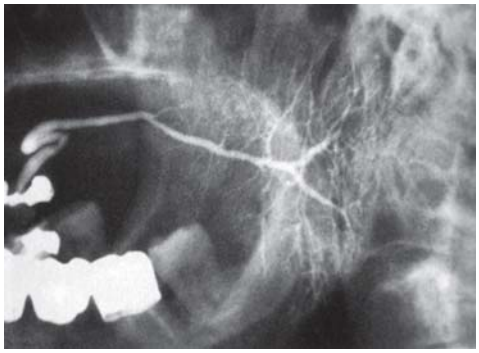
digits may be the sensitive indicator of secondary hyperparathyroidism. This is called as 'brush fire sign'.

43. What is tail sign?

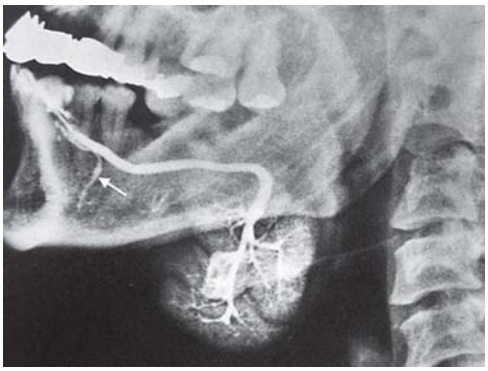
It is an extension of ranula from sublingual gland in the sublingual space, commonly seen on CT and MRI. It is core of CT diagnosis of plunging ranula. It provides an important clue to differentiate plunging ranulas from other cystic lesions arising in or near submandibular space such as cystic hygroma, thyroglossal duct cyst, second brachial cleft cyst, abscess and dermoid/epidermoid cyst. A dividing ranula has a distinct tail sign within sublingual space with bulk of cyst seen in submandibular space.

44. Describe various sialographic appearances.

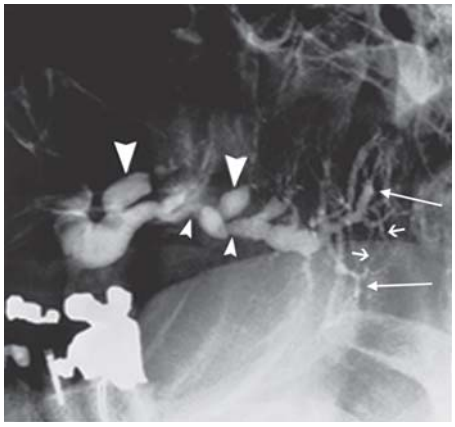
1. **Normal parotid:** "Leafless tree" or "tree in winter".



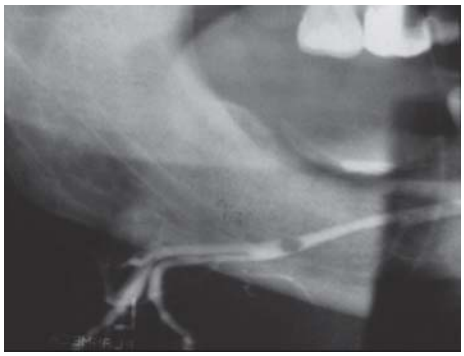
2. **Normal submandibular gland:** "Bush in winter"



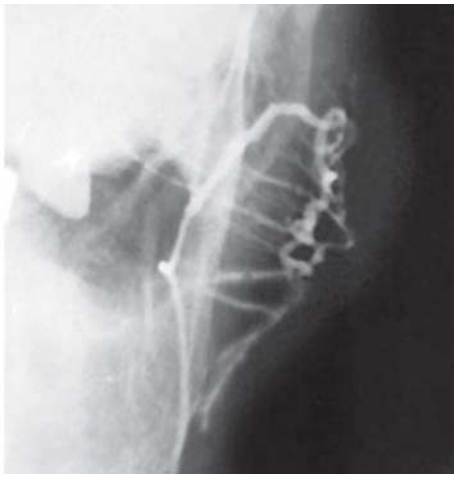
3. **Sialodochitis:** Segmental strictures of duct sausage-link appearance.



4. **Sialolith:** Filling defect with retention of dye.



5. **Benign tumors of parotid gland:** A mass within the gland is inferred by the



appearance of the ducts displaced around the lesion. It is called 'ball in hand appearance'. It is suggestive of space occupying mass.

45. What are shovel-shaped incisors? What is shovel-shaped incisor syndrome?

Shovel-shaped incisors show normal crowns except for prominent marginal ridges surrounding deep lingual fossa in maxillary central and lateral incisors. It is considered as an anatomical variant rather than a morphological defect because of its high prevalence.

Double shovel is shovel-shape associated with accentuated marginal ridges, which is seen occasionally. Shovel-shaped incisors may be seen in Apert's syndrome. In Klinefelter's syndrome correlation between taurodontism and shovel-shaped incisors is present. Shovel-shaped incisors with prominent cingula are associated with dens invaginatus.

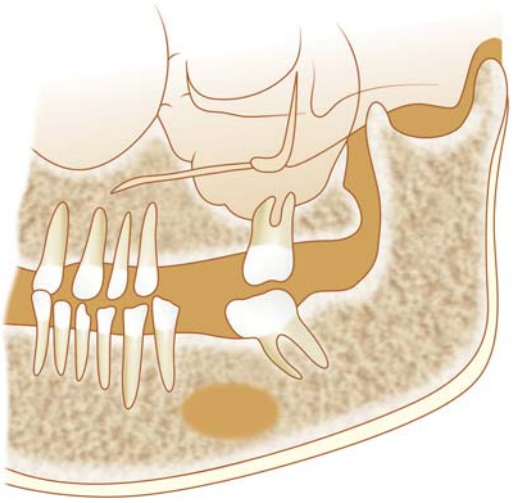
When shovel-shaped incisors are seen clinically following abnormalities should be looked for interproximal caries, lingual pit caries, periapical lesions of pulpal origin, the presence of shortened or tapered roots. The combination is called shovel-shaped incisor syndrome.

46. What are the different types of characteristics of lesional tissues?

1. **Bone destruction**
   a. *Geographic pattern:* Moth-eaten patterns and permeative patten.
   b. *Multilocular lesions:* Honeycomb, soap bubble, tennis racket and scalloping.
2. **Mineralisation of tumor matrix:** Osseous foci, calcific foci, calcific spherules and calcific massules, odantomatous and dentinomatous calcification.
3. **Internal margins:** A narrow zone of transition, a wide zone of transition.

Radiolucent lesions are caused by bone destruction. The medullary destruction may remain inapparent but as the lesion destroys the endosteal surface, it may appear on radiograph especially on panoramic.

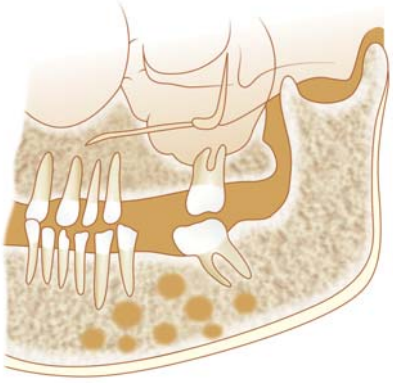
1. **Geographic patterns:** Implies a large area of lysis, absence of expansion and is not associated with specific type of margins. It indicates monolocular or nonseparated benign/malignant lesion. These are characterized by a large solitary hole in bone with sharply demarcated edge. These are suggestive of less aggressive and slow growing lesion.



It is seen in:

- *Unilocular ameloblastoma:* Early aggressive lesion benign in nature.
- *Residul cyst/traumatic cyst:* Slow growing, nonaggressive condition.
- *Ossifying fibroma:* Early stage of lesion that may have calcified tumor matrix.
- *Suspected malignant disorder:* Geographic patterns suggest slower, less aggressive growth, e.g. solitary or multiple myeloma, mucoepidermoid carcinoma, and metastatic disease.

2. **Moth-eaten pattern:** Implies several small areas of lytic bone destruction. It also implies an absence of expansion, but it indicates a more rapid, aggressive process than geographic pattern. It is seen with benign/malignant conditions. (Here radiographically separation from benign/malignant is not possible as margins are same.)



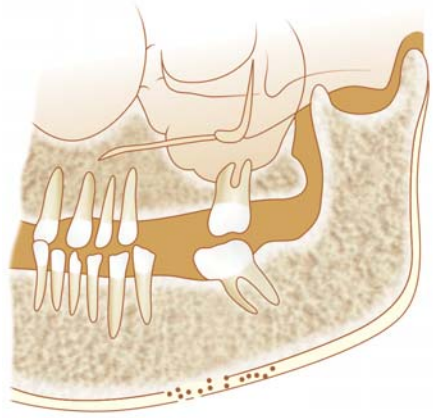
It is seen in:

- *Inflammatory conditions:* Chronic osteomyelitis chiefly and osteoradionecrosis.
- *Malignant conditions:* Metastatic disease.

3. **A permeative pattern:** Implies an absence of expansion but is almost seen in aggressive, rapidly destructive malignant disease. These may be seen in medullary areas or seen in adjacent cortex also. It is characterized by numerous tiny radiolucencies in between the residual bone trabeculae. Due to the minute size of radiolucencies the lesion may be difficult to see and to delineate on the plain film. Generally, the more rapidly growing a lesion, the more difficult it is to see on plain film. It indicates destruction of both medullary and cortical bone.

   It is seen in:

   - Metastatic diseases and malignant disorders.



- Lytic malignant diseases in jaws manifest mainly as moth-eaten and permeative patterns, although geographic changes are seen.

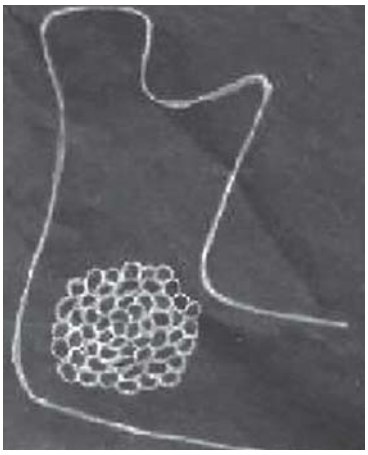
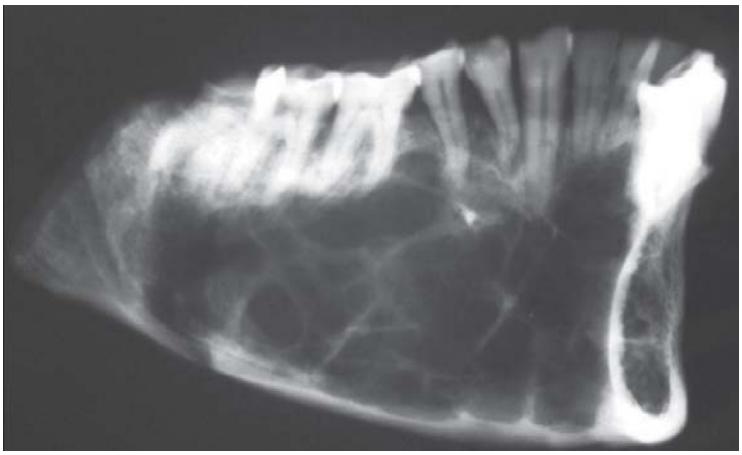
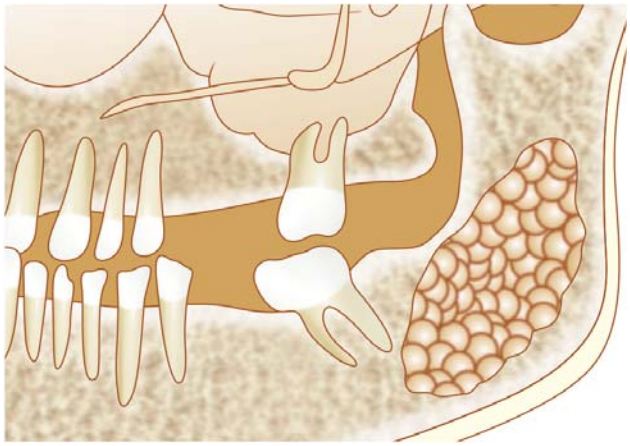
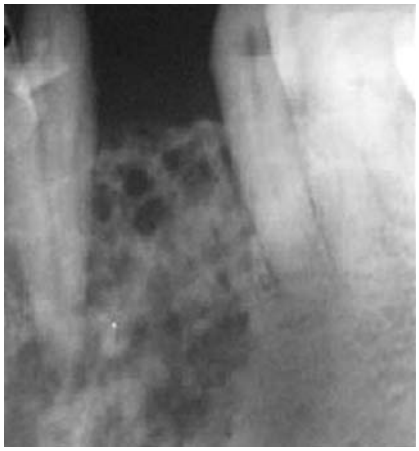
47. What are the patterns of multilocular patterns of bone destruction?

Multilocular lesions indicate internal septation.

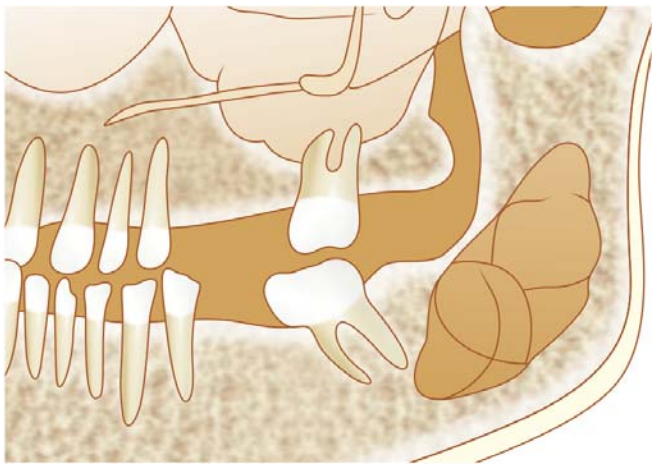
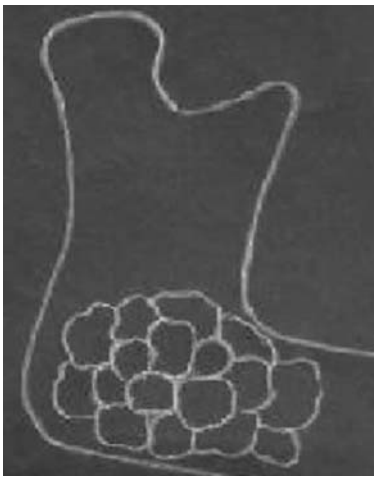
The Septation Rules Out

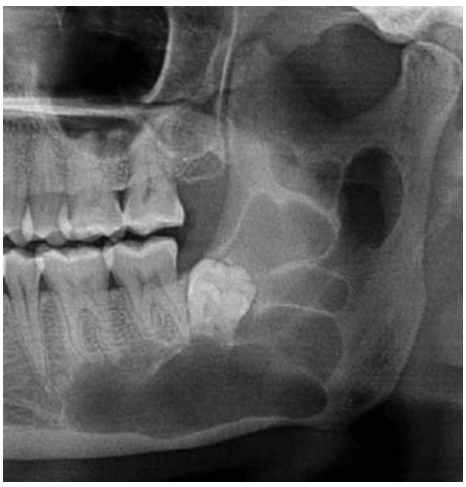
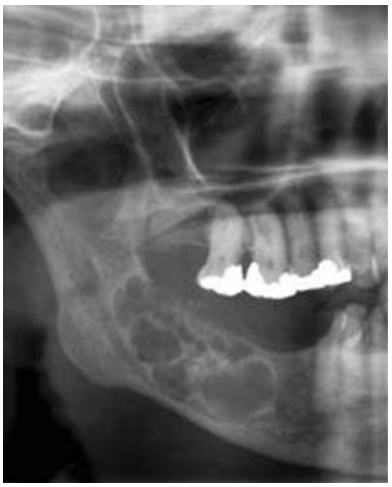
- Some benign lesions and all cysts (except traumatic bone cyst).
- It also rules out different pattern of calcification in tumor matrix.
- With a few exceptions (e.g. central mucoepidermoid carcinoma), these are indicative of benign, aggressive lesions.
- These indicate cortical expansion and cortical expansion indicates aggressiveness in benign lesion. The recurrence rate is higher in multilocular lesions. Honeycomb, soap bubble, tennis racket and scalloped are variants. Honeycomb is an earlier change than soap bubble, probably honeycomb may turn into soap bubble.

1. **Honeycomb pattern:** Circles are little smaller and probably of the same size and meeting each other. Ameloblastoma central giant cell granuloma.

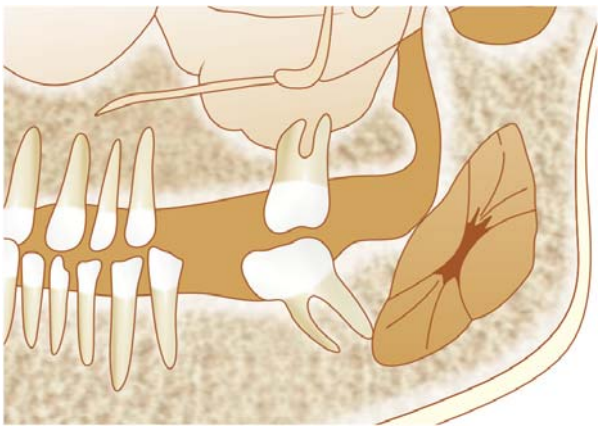





2. **Soap bubble:** Larger circles. Unequal size circles, coalesing sometimes or overlapping somewhat. Examples are ameloblastoma, central giant cell granuloma.

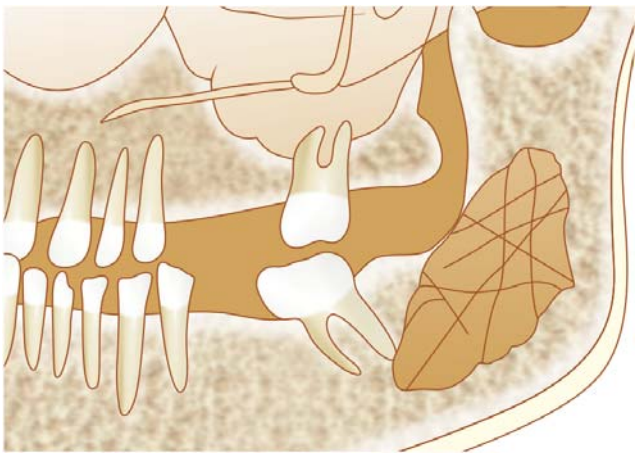
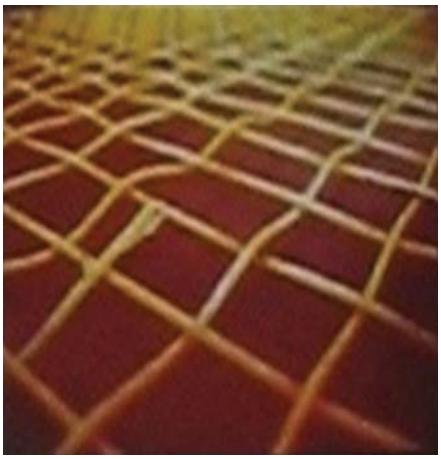



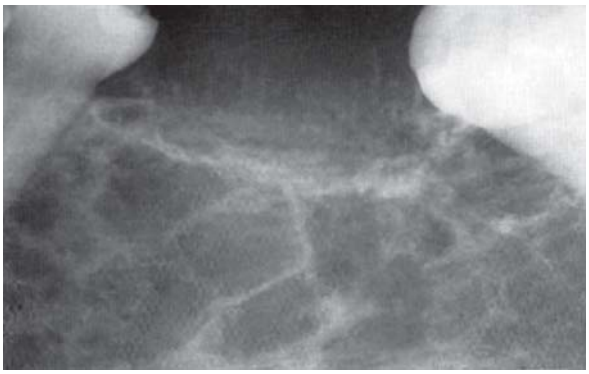



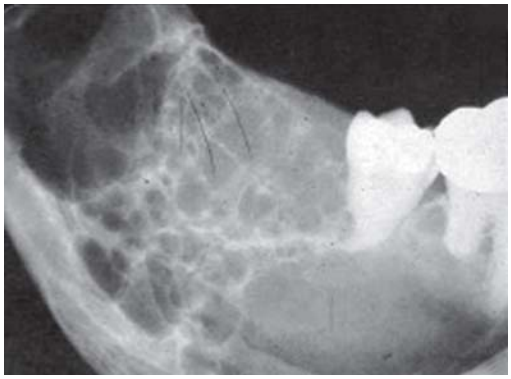
2a. Spider is a variant in which the septae radiate from central body.

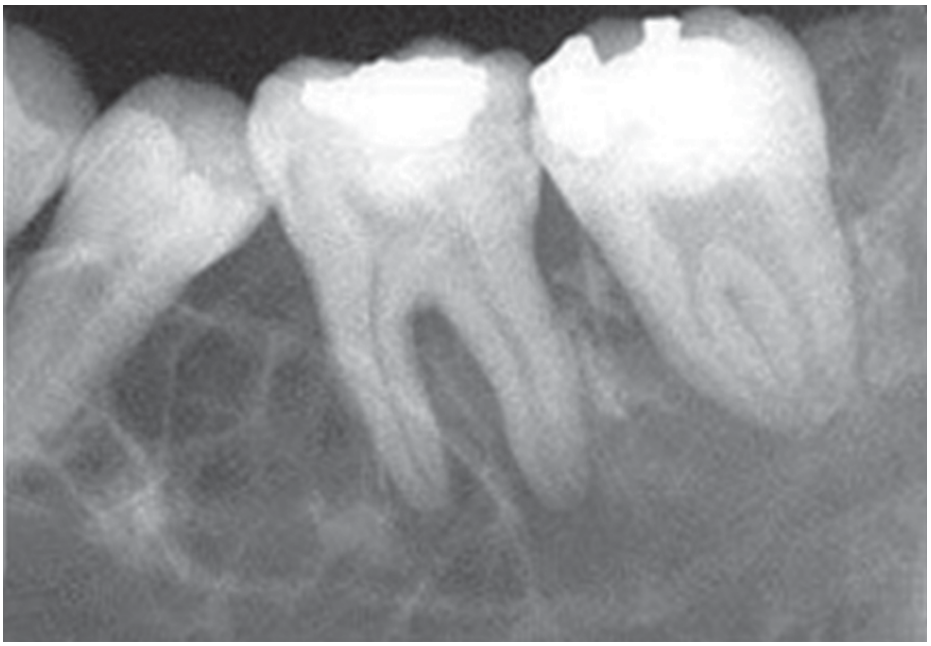



3. **Tennis racket:** Irregularly shaped septae meeting at right angle. Exclusively odontogenic myxoma.

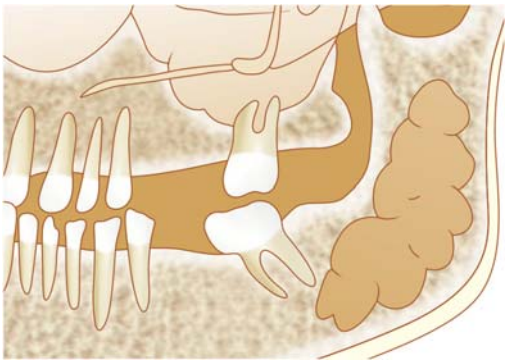



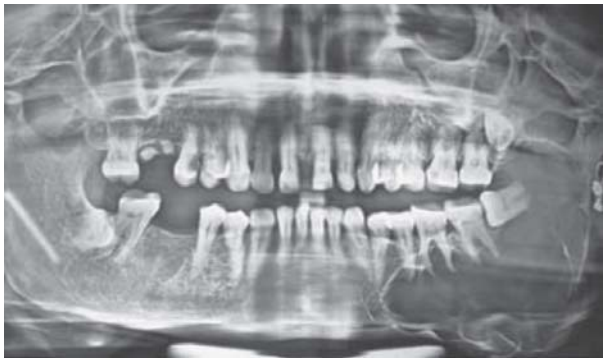


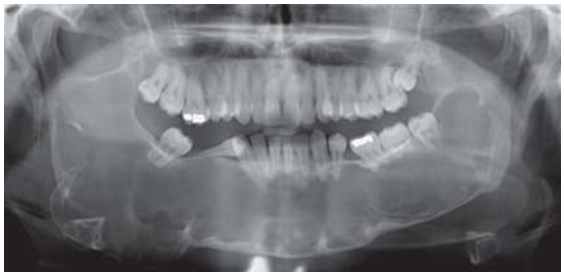




4. **Scalloped variant:** Keratocystic odontogenic tumor. Sometimes in central giant cell granuloma.





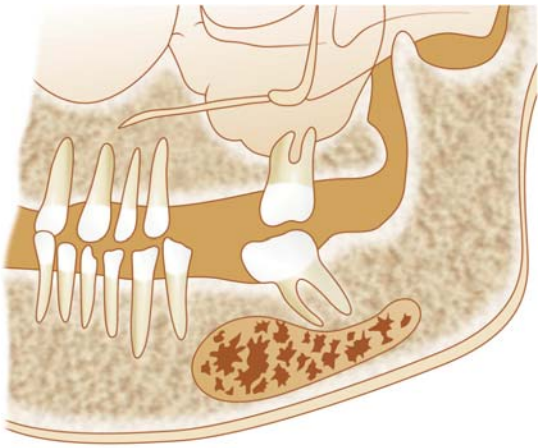


Multilocular patterns appear earlier, while scalloped/crenated margin is later change. A scalloped margin may or may not be associated with expansion.

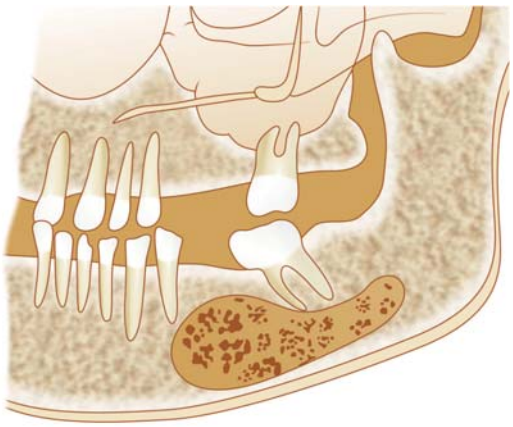
48. What are mineralization of tumor matrix?

The lesions may develop radiolucency and some may show radiodense appearance. The patterns are osseous foci, calcific foci, calcific spherules and massules, odontomatous calcifications, dentinomatous calcifications.

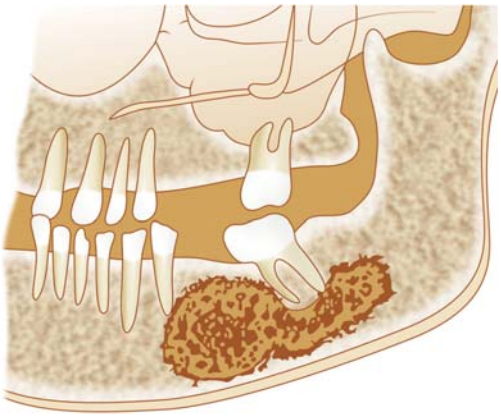
Osseous foci (flecks): These indicate bone or osseous trabeculae histologically. These foci may join together and form clumps. Trabeculae may project from foci.



Calcific foci: These denote mineralization of chondroid matrix. The density is less than surrounding bone. They are round, tiny and sometimes invisible. Use of magnifying lens is necessary. The tendency of foci to clump together is almost diagnostic. Small tumors show small/punctate forms and may form snowflake like patterns. Large tumors show nodules, flocculent or popcorn like rings and arc of calcific density. Still it is not more dense than surrounding bone.

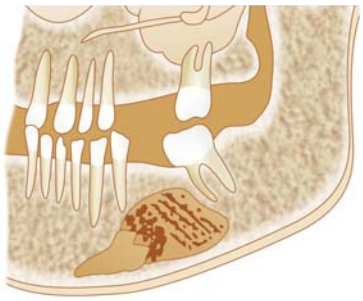


Calcific spherules and calcific massules: These are mineralized flecks seen in cemental lesions. Calcific spherules are tiny circular (0.2–0.5 mm in diameter) structures with radiolucent structures, faintly radiopaque. Outline. These are commonly seen in ossifying fibromas and benign cementoblastomas. These spherules join together and form massules (0.5–1.0 mm in diameter).

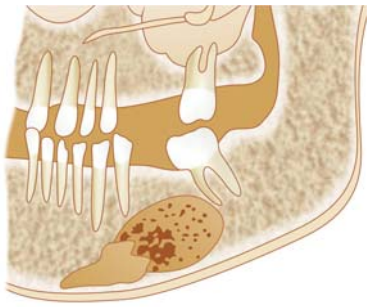


Odontomous calcification: These considered to be pathognomonic. They present three forms:

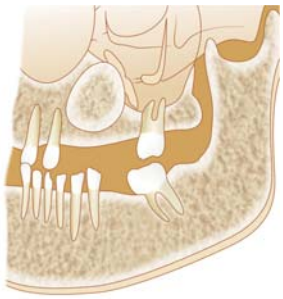
- The density is similar to teeth and tend to be denser than surrounding bone and is most common.



Pindborg flecks



Gorlin flecks



AOT flecks

- Washer like radiodensity with a radiolucent centre.
- Hodgepodge dental structures with little to identify them as odontomous calcification.

These calcifications can be associated with dentigerous cyst, Gorlin cyst, ameloblastic odontoma, and ameloblastic fibro-odontoma.

Dentinomatous calcification—rarely seen. It is present in dentinoma and ameloblastic fibrodentinoma.

49. What is zone of transition?

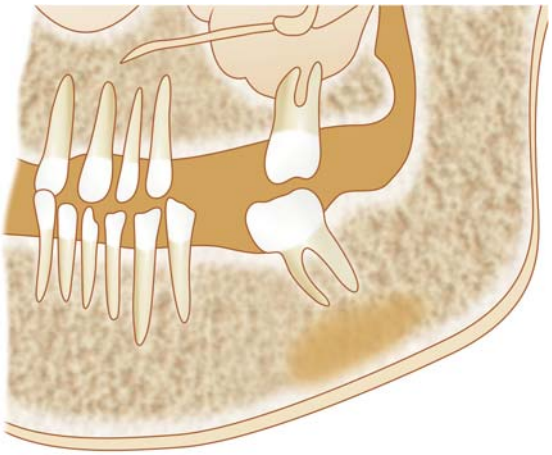
Zone of Transition

In order to classify osteolytic lesions as well-defined or ill-defined, we need to look at the zone of transition between the lesion and the adjacent normal bone.

The zone of transition is the most reliable indicator in determining whether an osteolytic lesion is benign or malignant.

The zone of transition only applies to osteolytic lesions since sclerotic lesions usually have a narrow transition zone.

- **A narrow zone of transition:** Sclerotic bone is deposited by host. Indolent or slow growing lesions are usually marginated by sclerotic bone. Rapid growth shows no sclerotic bone or may be seen in some area. In aggressive bone growth the sclerotic margins may be thinned out, discontinuous and even break out (discussed below).
- **A wide zone of transition:** It indicates that the lesion is aggressive, margin between healthy and abnormal bone is wider. It may be seen in benign and malignant lesions. The benign lesions which are aggressively growing are having rapid growth. Examples—eosinophillic granuloma, central giant cell granuloma and aneurysmal bone cyst. A wide zone of transition in the absence of expansion suggests a malignancy such as metastatic disease.



Wide zone of transition

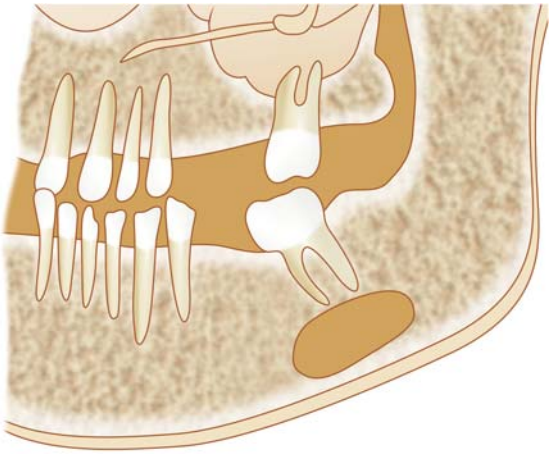
50. What are the different types of internal margins?

Internal margin relates to the interface between the lesion and host bone within which it occurs. It provides the information regarding aggressiveness and growth rate.

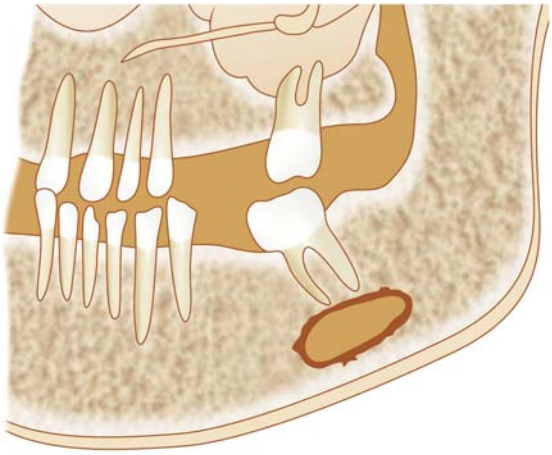
- Thick condensed sclerotic rim, thick diffuse sclerotic rim, thin condensed sclerotic rim, punched out lesions with no sclerosis at the margin.

Benign tumors/cysts have a condensed type of sclerosis while reactive process usually have a diffuse type of marginal sclerosis. Examples—diffuse and thick sclerotic rims at margins of cementomas which indicates slow growing lesions. Odontogenic myxoma have highest rate and probably will be marked by condensed sclerotic bone reactions.

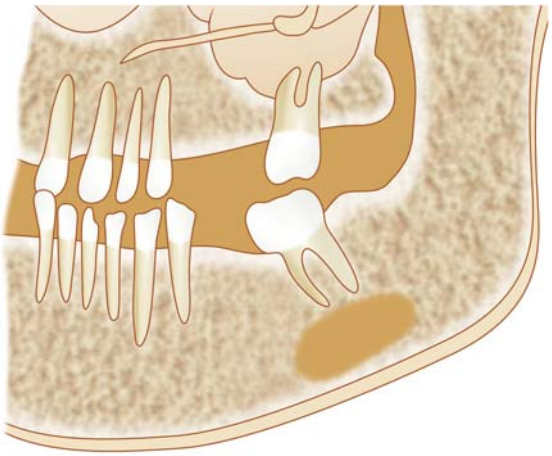
Myeloma may show punched out margins sharply defined but lack sclerotic margins, this may indicate that periosteum may be injured by disease.



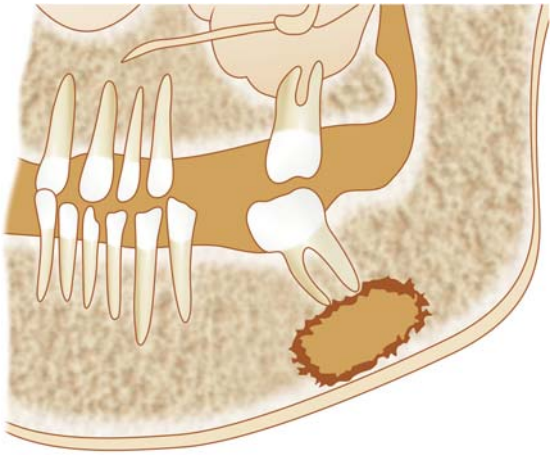
Thin condensed sclerotic rim



Thick condensed sclerotic rim



Punched out lesion



Thick diffuse sclerotic rim

51. How the relationship of the teeth and of resorption at root apex helps in diagnosis?

Teeth Conditions

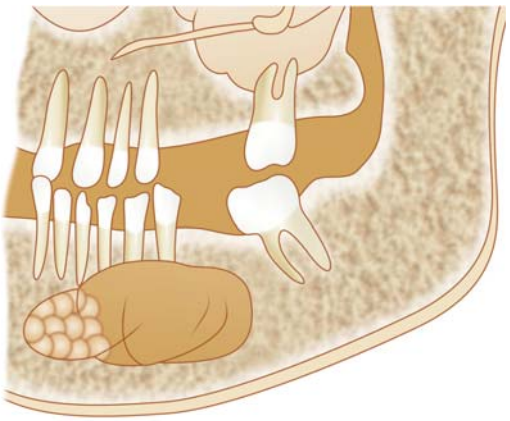
1. The conditions associated with the crown of unerupted teeth are almost odontogenic in nature (cysts/benign tumors). The presence of radiopaque foci further enhance the diagnosis.
2. Some lesions may displace the teeth while others cause unerupted teeth to become impacted. Benign process displace tooth (ossifying fibroma may displace, cementoblastoma does not) displacement of

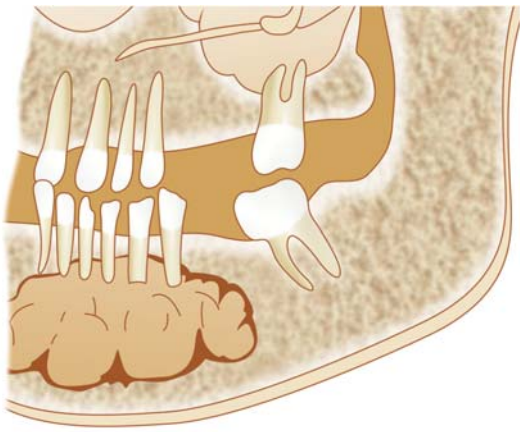
unerupted teeth may be seen in dentigerous cyst, Gorlin's cyst, inflammatory paradental cyst and in keratocystic odontogenic tumor. It is also seen in odontogenic mysoma, ameloblastoma and brown tumor. Ameloblastic fibro-odontoma and ameloblastic odontoma are associated with displaced tooth and are more aggressive.

3. No displacement and causing unerupted teeth to become impacted, then the lesion is less aggressive. Odontoma is associated with nondisplaced impacted tooth.

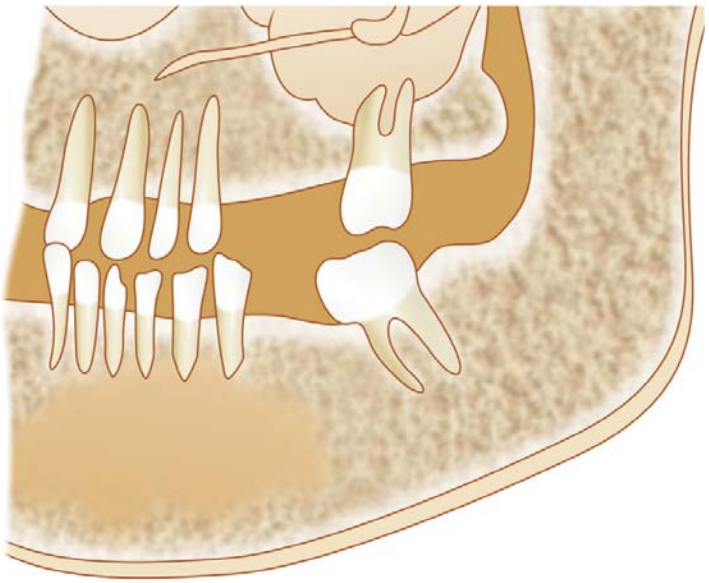
Resorption of Root

1. Resorption of one/several teeth root apex suggest benign process with aggressive nature.
2. Rarely resorption may be associated with malignant disease which may suggest a slower malignant growth pattern.
3. Knife edge resorption may be seen in ameloblastoma.
4. Multiple root planes resorption is common in central giant cell granuloma and sometimes in ameloblastoma.



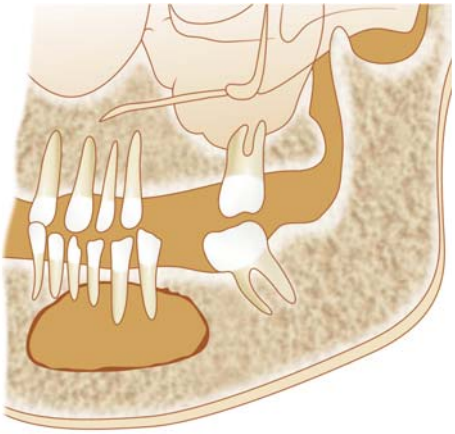


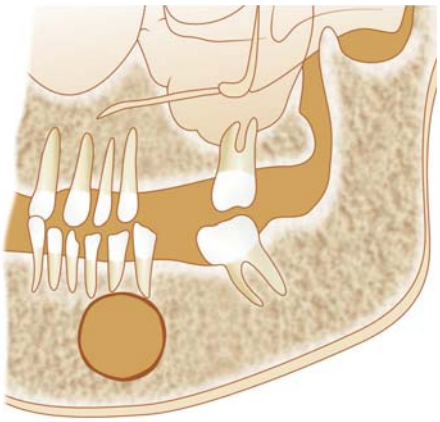
5. Spiking root resorption may suggest malignant disorder. The malignant disorders are too rapidly destructive.



52. What are the other factors in root are considered for radiological diagnosis?

1. The deposition of material on root apex is indicative of benign process, e.g. hypercementosis and cementoblastoma (the dentinal outline is lost in benign cementoblastoma, whereas it can be seen in hypercementosis).
   Paget's disease and Gardener's syndrome are said to be associated with generalized hypercementosis.
2. Relationship of root apex with radiolucent lesion. If apex of one or more root tip protrude in the lesion indicates neoplasia, (as tumors go around the tooth) while in cystic lesion the apices do not protrude and are in close proximity of root (cysts either cut off the teeth or cyst margins stop at apices of teeth). Resorption may or may not be a feature of cyst or tumor.





3. Traumatic bone lesion is only one lesion that characteristically straddles the roots. The superior portion projects up between teeth with or without destruction of lamina dura.

53. How the cortical changes are useful for radiological diagnosis?

The expanded cortex is suggestive of locally aggressive benign lesion:

- The absence of septation within the expanded cortex is suggestive of less aggressive lesion and is hallmark of slow growing benign lesion.
- The expanded cortex on radiograph may be intact and visible, intact and invisible or perforated. The perforated cortex is a sign of most aggressive benign lesions, with propensity to recur and even few low-grade malignant conditions also show this feature. CT may show perforations exactly.

– The characteristics	– Cystic exapansion	– Neoplastic expansion.
– The nature of pressure	– Hydraulic effect is applied on cortex.	– The vector growth is often but not exactly perpendicular to the cortex.
– The direction of pressure	– The pressure is equally on all margins.	– The pressure applied is not uniform.
– Nature of expansion of cortex	– The cortex is expanded evenly and smoothly.	– Not always uniform. It may be paper thin, may have a slightly wavy, irregular surface.
– Disappearance of cortex	– The cortex may not seem to disappear at the greatest bulk.	– It may seem to disappear.
– The meeting angle with normal cortex.	– The expanded cortex meeting the normal cortex at an obtuse angle on both sides.	– Acute angle on one side and different acute or obtuse angle on other side.

- Once the cortex is perforated the soft tissue may be herniated through it. This characteristic may seen on MRI (e.g. odontogenic myxoma, aneurysmal bone cyst).
- Jaws give rise to more cystic lesions than any other bones in skeleton.
- Scalloping of the cortex appears at endosteal surface of the cortex (e.g. keratocystic odontogenic tumor/central giant cell granuloma).
- Saucerization may be seen in outer cortex and may seen with lesions arising from gingival and periosteum (Examples are submandibular, sublingual and parotid gland depressions. Scleroderma, gingival cyst of adults, neural sheath tumors and traumatic neuroma and peripheral giant cell granuloma).

54. Classify side-effects of radiotherapy. What are temporary and permanent side effects of radiotherapy?

These can be classified as:

1. Acute (early, temporary) or chronic (late, permanent).
2. Stochastic/nonstochastic.

The temporary or acute side-effects are short term side-effects. These may occur close to the time of treatment and usually gone completely within a few weeks of finishing the treatment. Hairloss may be temporary, fatigue, skin changes and mucosites are the examples. Chronic, long-term or permanent may take months or years to develop and usually are permanent. Hairloss may be permanent. Hearing loss in children.

55. Describe how different cell type affects radiosensitivity.

Different cells from various organs of the same individual may respond to irradiation quite differently.

This variation was recognized as early as 1906 by the French radiobiologists Bergonie and Tribondeau. They observed that the most radiosensitive cells are those that:

1. Have a high mitotic rate,
2. Undergo many future mitoses, and
3. Are most primitive in differentiation,
4. Cells that do not perform any specialized functions.

High radiosensitive cells include white blood cells, bone marrow, and eyecells.

Radiosensitive organs are blood forming organs (bone marrow, lymph nodes, and thymus and spleen).

Low radiosensitive are red blood cells, muscle cells, bone cells, and cells of nervous system.

Undernourished cells are generally less radiosensitive than normal cells.

These findings are still true except for lymphocytes and oocytes, which are very radiosensitive even though they are highly differentiated and nondividing. Mammalian cells may be divided into five categories of radiosensitivity on the basis of histologic observations of early cell death:

1. Vegetative intermitotic cells are the most radiosensitive. They divide regularly, have long mitotic futures, and do not undergo differentiation between mitoses. These are stem cells that retain their primitive properties and whose function is to replace themselves. Examples include early precursor cells, such as those in the spermatogenic or erythroblastic series, and basal cells of the oral mucous membrane.
2. Differentiating intermitotic cells are somewhat less radiosensitive than vegetative intermitotic cells because they divide less often. They divide regularly, although they undergo some differentiation between divisions. Examples of this class include intermediate dividing and replicating cells of the inner enamel epithelium of developing teeth, cells of the hematopoietic series that are in the intermediate stages of differentiation, spermatocytes, and oocytes.
3. Multipotential connective tissue cells have intermediate radiosensitivity. They divide irregularly, usually in response to a demand for more cells, and are also capable of limited differentiation. Examples are vascular endothelial cells, fibroblasts, and mesenchymal cells.

4. Reverting postmitotic cells are generally radioresistant because they divide infrequently. They also are generally specialized in function. Examples include the acinar and ductal cells of the salivary glands and pancreas as well as parenchymal cells of the liver, kidney, and thyroid.
5. Fixed postmitotic cells are most resistant to the direct action of radiation. They are the most highly differentiated cells and, once mature, are incapable of division. Examples of these cells include neurons, striated muscle cells, squamous epithelial cells that have differentiated and are close to the surface of oral mucous membrane, and erythrocytes.

56. Enumerate the radiosensitive and radioresistant tumors of jaw bones.

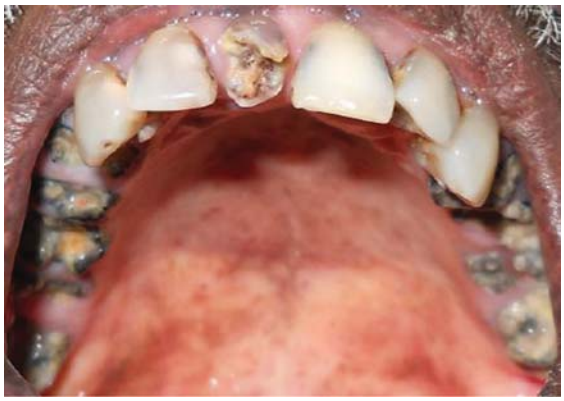
Those tumours that respond to treatment with radium or X-rays are called radioactive or radiosensitive. Example—lymphoma because of open lymphatic channels, squamous cell carcinoma (especially poorly differentiated). Basal cell carcinoma and some adenocarcinoma, Ewing's sarcoma. Those tumors of other type resist treatment by radium and X-rays are called radioresistant. Examples—ameloblastoma, salivary gland neoplasms (parotid tumors are the least radiosensitive, while tumors in ectopic sites are relatively radiosensitive), osteogenic sarcomas, fibrosarcomas and malignant melanoma, eosinophilic granuloma.

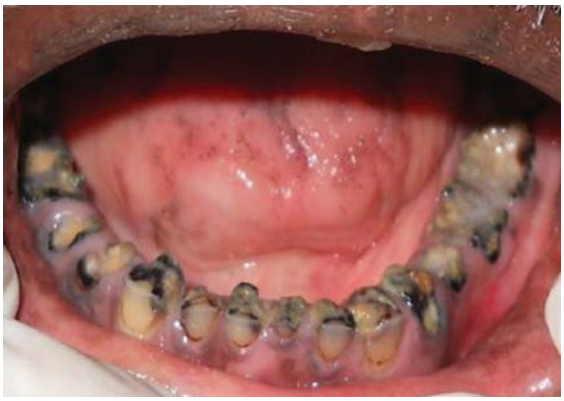
Oral carcinoma once it invades jaw bones surgery is the treatment of choice as it becomes radioresistant once it involves jaw bones. Mandible is four times more radiosensitive than maxilla, radiosensitivity does not necessarily indicate curability, nor should radioresistance be taken to imply incurability.

57. What is radiation caries? What are different types of radiation caries?

It is rampant form of dental caries that may occur in individuals who receive radiotherapy or radiation caries is a term used to describe rapidly advancing caries, which characteristically occur at incisal or cervical aspects of teeth, starting at incisors and canines. The rapid onset and widespread attack are characteristics of radiation caries. The caries often begins at cervical area, encircle the tooth aggressively causing the entire crown to be lost, with only root fragments remaining. It may occur as early as three months and can progress at an alarming rate, rarely pain is associated. The lesion resembles more of demineralization than caries. It sweeps around the tooth and may cause amputation at tooth neck. Teeth are brittle and pieces of enamel may fracture. Clinically there are three types:

1. Widespread superficial lesion attacking buccal, occlusal, incisal, and palatal surfaces. This is most common type.
2. Involving cementum and dentin
3. Dark pigmentation of entire crown.





Blackish discolored multiple root stumps (shows multiple black discolored root, the posterior maxilla and right central incisor with stumps in mandible) class V caries in maxillary anteriors

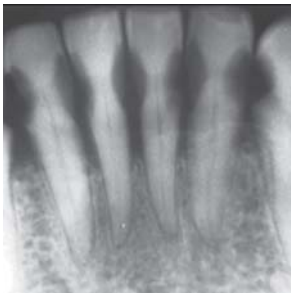
Radiation caries results from changes in salivary glands and saliva. These are:

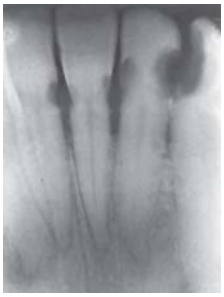
1. Reduced flow
2. Decrease pH
3. Decrease buffering capacity
4. Altered flora
5. Low concentration of Ca^{++}. This results in greater solubility of tooth structure and greater demineralization.

Direct effect of radiation on teeth makes them more prone to flaking particularly in areas of occlusal loading or stress.

Apple core appearance: It appears as punched out radiolucency seen on radiograph, radiographically radiolucent shadows at necks of teeth most obvious on mesial and distal aspects.

Use of topical fluoride as remineralizing solution and meticulous oral hygiene are helpful.





58. What is brachy therapy?

Brachio is short. It uses selected isotopes or specialized instruments to directly administer radiation to tumor or its bed. The radiation sources are placed either adjacent to surface of a tissue mass or bed or inside tumor itself.

The treatment may involve permanent implantation of radioactive sources (e.g. permanent ^{125}I seeds into recurrent nasopharyngeal mass).

It travels only short distance to target lesion and its dose intensity falls of rapidly with distance according to inverse square law. Major advantage spares normal tissue at distant locations while major disadvantage is heterogenous distribution of dose deposition in tissue may lead to tumor recurrences at low dose.

59. What are radioisotopes?

Isotopes are the nuclei which are having same number of protons and different numbers of neutrons, i.e. same atomic number but different mass number. These are produced in a nuclear reactor by exposing the target material to the neutrons in a reactor.

Radioisotopes are a version of chemical element that has an unstable nucleus and emits radiation during its decay to stable form. Radiations given by some of radioisotopes are very effective in curing certain diseases, e.g. ^{60}Co radiocobalt is used in treatment of brain tumor, ^{32}P radiophosphorus in bone diseases, ^{131}I in thyroid cancer.

60. What are radiopharmaceuticals?

These are unique medicinal formulations containing radioisotopes. It may be ^{133}Xe, ^{131}I iodinated proteins and Tc 99m labelled compounds. These are sterile and non-pyogenic.

61. What are newer developments in radiotherapy?

1. **Nonsealed injectable radionuclides:** Isotopes I^{131} I/V administered iodine is absorbed by thyroid gland. Strontium 29 after systemic administration, they become concentrated at osteoblastic activity which is used to palliate painful bone metastasis and treatment of osteogenic sarcoma.
2. **Radioimmune therapy:** One therapeutic approach that has demonstrated potential involves the selective targeting of radionuclides to cancer-associated cell surface antigens using monoclonal antibodies. Such radioimmunotherapy (RIT) permits the delivery of a high dose of therapeutic radiation to cancer cells, while minimizing the exposure of normal cells. Radiation emitting isotopes conjugated with high affinity antibodies to generate radioimmunoglobulins. Yttrium 90 (^{90}Y) and rituximab two injections (Zevalin).
3. Gene therapy and radiotherapy

4. **Total and subtotal skin electron beam therapy (TSEB):** This treatment is directed at a large surface, or the entire surface, of the body. The radiation penetrates the outer layers of the skin without affecting deeper organs or tissue. Temple is the most experienced centre in the region to offer this therapy to patients with rare cancers and conditions such as cutaneous T-cell and cutaneous B-cell lymphoma and Kaposi's sarcoma.
5. **Hyperthermia:** Heating tumor cells in combination with radiation therapy can help shrink tumors and relieve symptoms for patients who have failed prior conventional treatment, including radiation, surgery or chemotherapy.
6. **Three-dimensional:** Conformal radiation therapy (3D-CRT)—the radiation beam is absolutely tailored to the shape and configuration of the tumor to avoid nearby normal critical structures.

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Oral Medicine and Radiology

Question–Answer format for
Review and
Exam Preparation

is based on various concepts laid down in standard textbooks. There are several books on oral medicine and radiology written by various authors having appropriate coverage of the subject. This book aims to prepare the students for their examinations, especially for the competitive examinations and viva voce. It attempts to highlight those aspects of the subject which have comparatively higher value. Various pieces of subject material important for the undergraduate and postgraduate students have been compiled and organised in a reader-friendly format.

The question–answer format followed in the book aims to help the students •understand, •remember, •recollect, and •reproduce the facts easily. The book carries some clinical tips too which will guide the readers during their clinical postings.

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is Reader, Department of Oral Medicine and Radiology, Pandit Deendayal Upadhyay Dental College, Solapur, Maharashtra. He has nearly 30 publications to his credit. He believes that oral medicine and maxillofacial imaging is the basis of dentistry, and having thorough knowledge of this subject is necessary for all academicians and practitioners. He aims at finding the best possible option for mucosal diseases and is eager to develop the oral medicine to full extent. He is also of the opinion that oral medicine and radiology has a hidden preventive aspect which needs to be explored and implemented for further growth of dentistry.

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