بسم الله الرحمن الرحيم

Cementum and Alveolar bone

- أعداد:
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Cementum:

A thin specialized calcified tissue covering the roots surfaces of the teeth.

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There are two main types of root cementum : acellular (primary) and cellular (secondary) .

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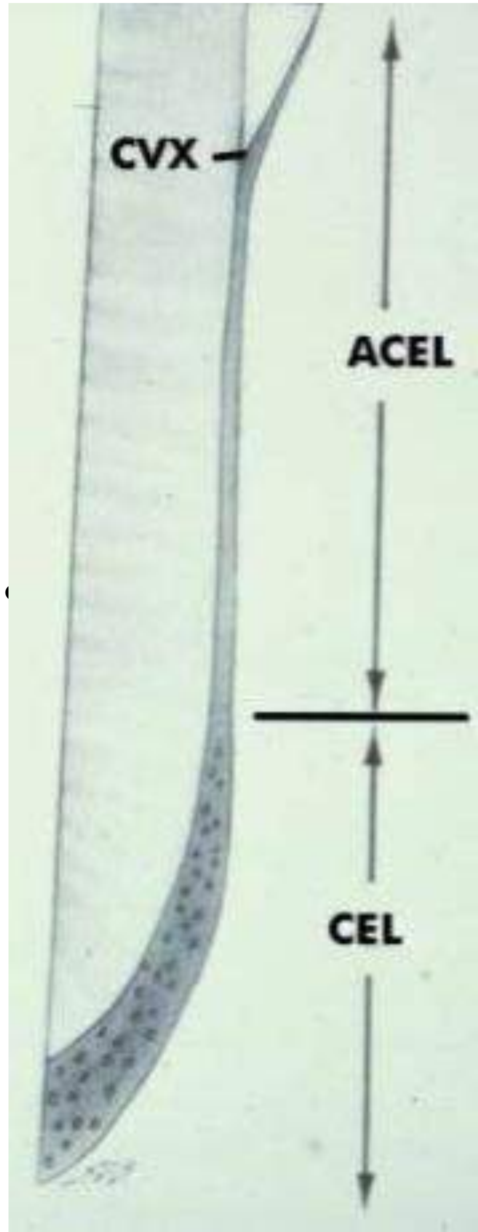


Functions of cementum:

- 1- Anchorage of the tooth in the alveolus.
- 2-To attach the PDL fibers to the teeth.
- 3- To contribute to the process of repair after damage to the root surface and following regenerative periodontal surgical procedures.

Structures of cementum:cementum consist of :

1- Fibrous elements (collagen fibers).

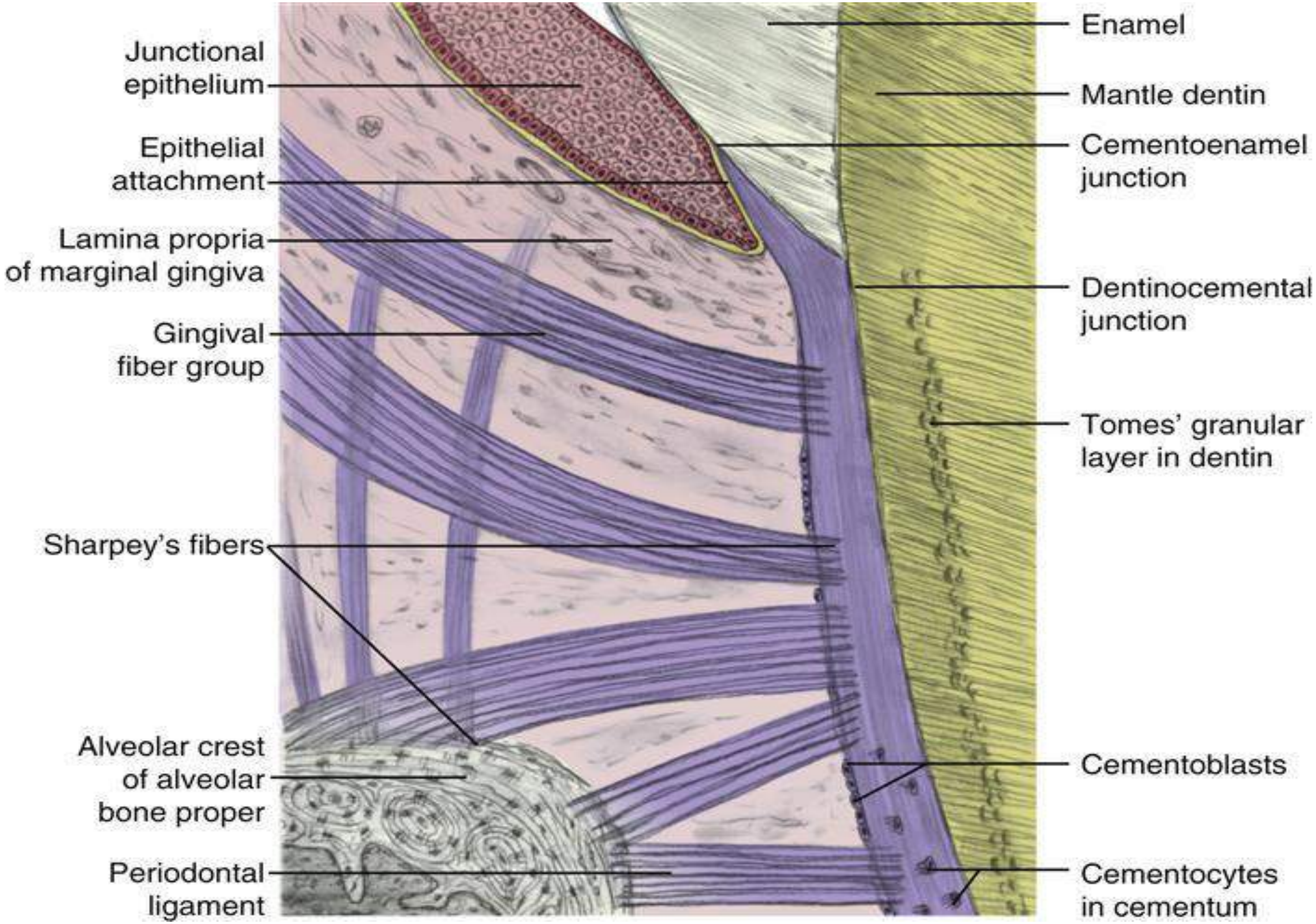
- **Extrinsic fibers(sharpeýs fibers).**
- **Intrinsic fibers.**

2-Cellular elements (cementoblast, cementocyte, fibroblast, cementoclast) .

3-Calcified interfibrillar matrix (proteoglycans, glycoprotein, phosphoprotein).

Extrinsic fibers: (sharpeys fibers): which are the embedded portion of the principles fibers of the PDL and are formed by the fibroblast cells. Sharpeys fibers make up most f the structure of acellular cementum and they are inserted at right angles to the root surface and penetrate deep into the cementum.

Intrinsic fibers: these fibers are poroduced by cementoblast cells are oriented more or less parallel to the long axis of the root and form a cross banding arrangement with sharpeys fibers.


Junctional epithelium
Epithelial attachment
Lamina propria of marginal gingiva
Gingival fiber group
Sharpey's fibers
Alveolar crest of alveolar bone proper
Periodontal ligament
Enamel
Mantle dentin
Cementoenamel junction
Dentinocemental junction
Tomes' granular layer in dentin
Cementoblasts
Cementocytes in cementum

1- cementoblast: responsible for the formation of both cellular and acellular cementum.

2-cementocyte: are found only in cellular cementum , they are located within spaces (lacunae) that communicate with each other through canaliculi for transportation of nutrition through the cementum and contribute to maintenances of the vitality of this tissue.

3-fibroblast: these are belong to the PDL where they are responsible for the synthesis of principle fibers but since these fibers become embedded in cementum, fibroblasts indirectly particitate in the formation of cementum.

4-cementoclast: these cells are responsible for extensive root resorption that lead to primary teeth exofoliation . Permenant teeth do undergo physiological resorption but localized cemental resorption may occur which appear as concavities in the root surface and may be caused by local or systemic causes: local condition include, trauma from occlusion, orthodontic movement , cyst., while in systemic condition caused in calcium deficiency and hypothyroidisms.

Mineralization of cmentum:

- **Occur by the deposition of hydroxyapatite crystals, first within the collagen fibers, later upon the fiber surface and finally in the interfibrillar matrix.**
- **cellular cementum is less calcified than acellular cementum.**
- **cenentum mineralization I less than that of the bone, enamel and dentin.**

Development of cementum:

1- Both cellular and acellular cmentum are produced by cementoblast cell.

2-cementoid is first formed which is a non calcified tissue containing collagen fibrils distributed in matrix.

3-cementum thickness increase with age.

4- cementum thickness in more in apical area and furcation area than cervical area.

5-cementum is thicker in distal surface than in mesial surface because of functional stimulation from mesial drift.


And the opposing tooth begins to extrude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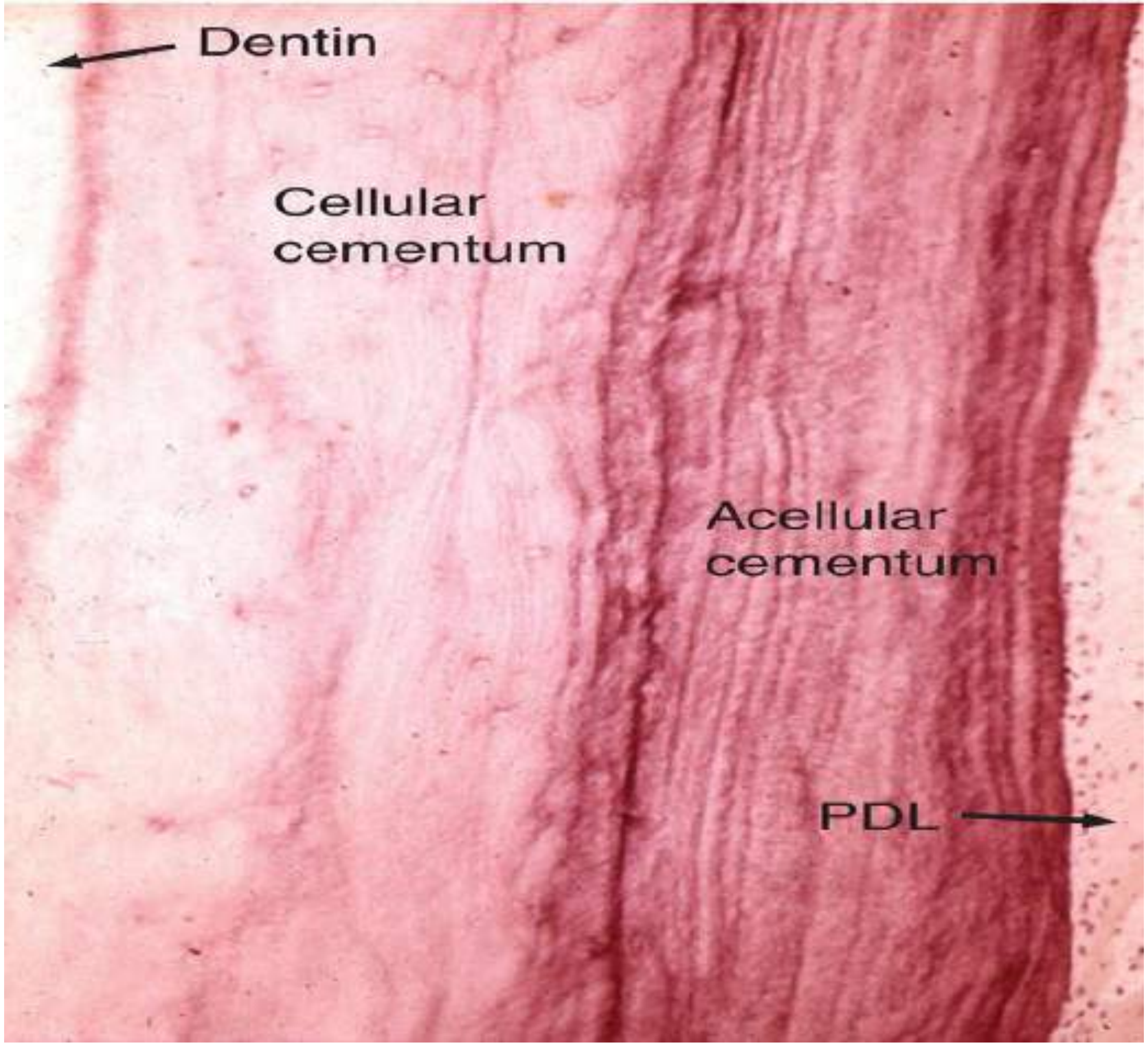
There are two types of cementum(both produced by cementoblast cells):

1- Primary (acellular cementum).

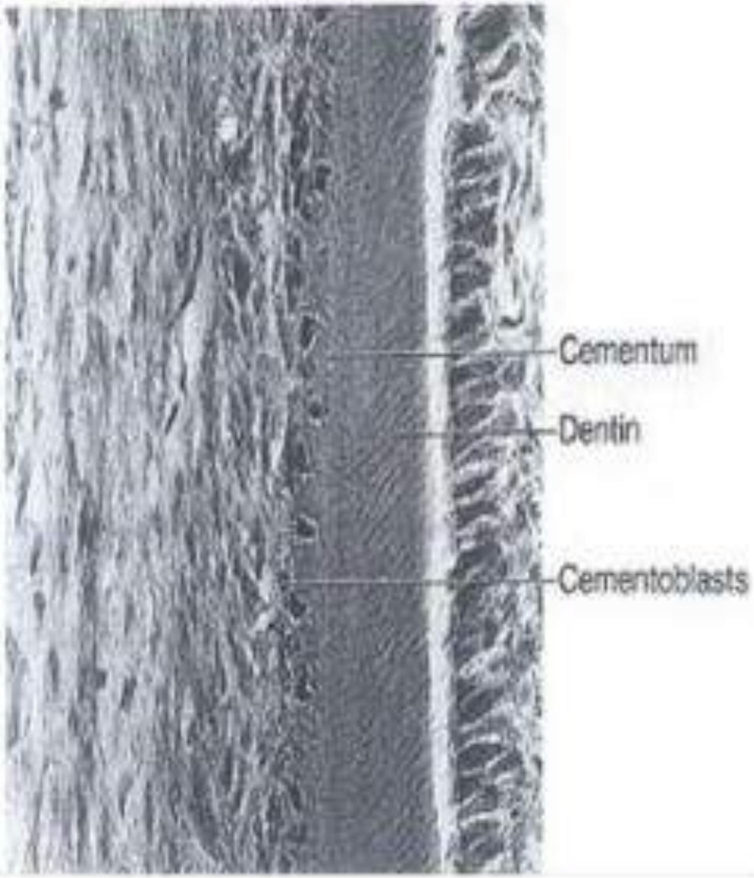
- First to be formed in conjunction with root formation and tooth eruption.
- it is not contain cells and sharpys fibers make up most of its structures.
- It cover the cervical third of the root.

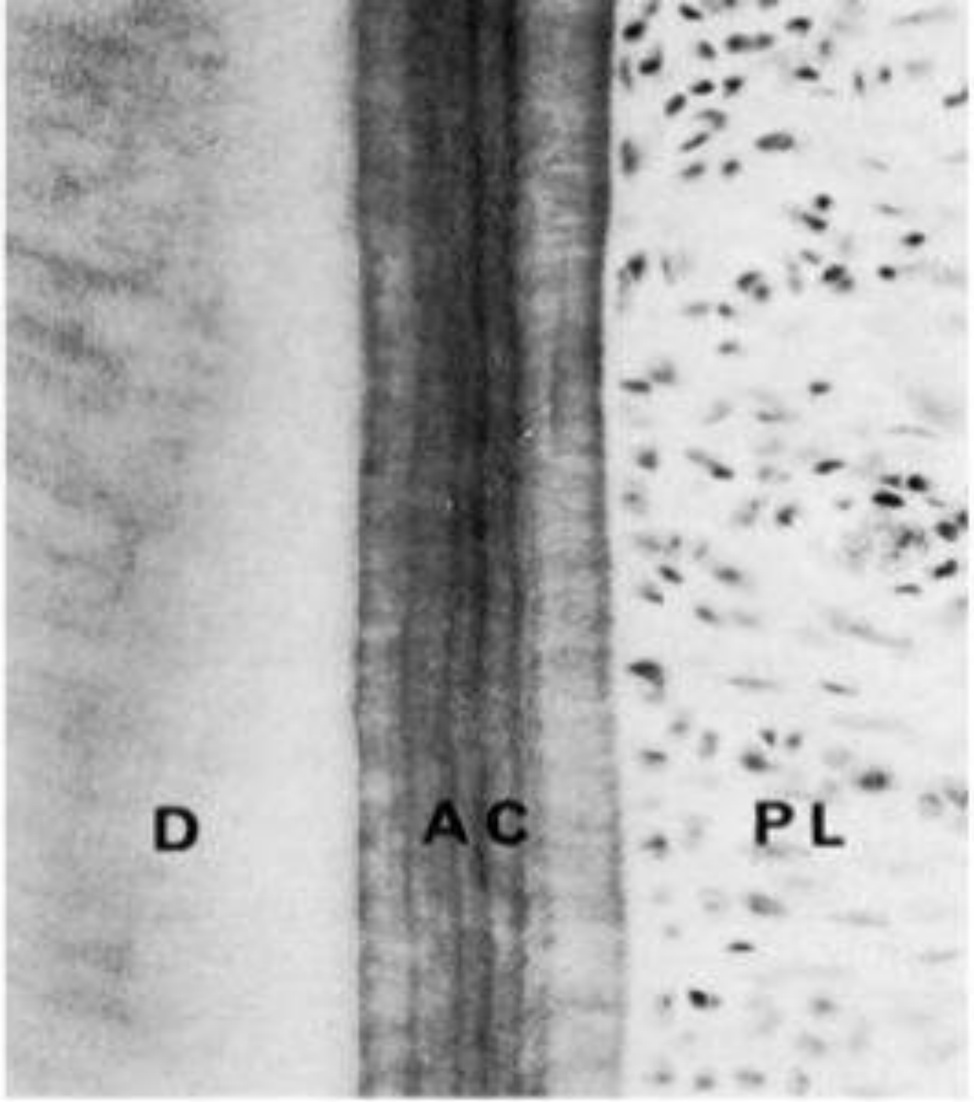
2- Secondary (cellular cementum).

- formed after tooth eruption in response to functional demand.
- Grow faster and over a thin layer of acellular cementum at the apical third of the root and furcations of multirooted teeth.
- Contain cells(cementocyte), Sharpys fibers occupy small portion.
- Is less calcified than a cellular type.


Dentin
Cellular
cementum
Acellular
cementum
PDL

CELLULAR CEMENTUM





Acellular cementum (AC) showing incremental lines running parallel to the long axis of the tooth. These lines represent the appositional growth of cementum. Note the thin, light lines running into the cementum perpendicular to the surface; these represent Sharpey's fibers of the periodontal ligament (PL). D, Dentin.

Cemento-enamel junction(C.E.J):

There are three types of relationhips involving the cementum may exist at the C.E.J:-

- Cementum overlaps the enamel(60-65%).

-Edge-to edge(30%)

-cementum and enamel fail to meet (5-10%).

+ in the last condition, there is a possibility of gingival recession which may result in sensitivity because the dentin is exposed

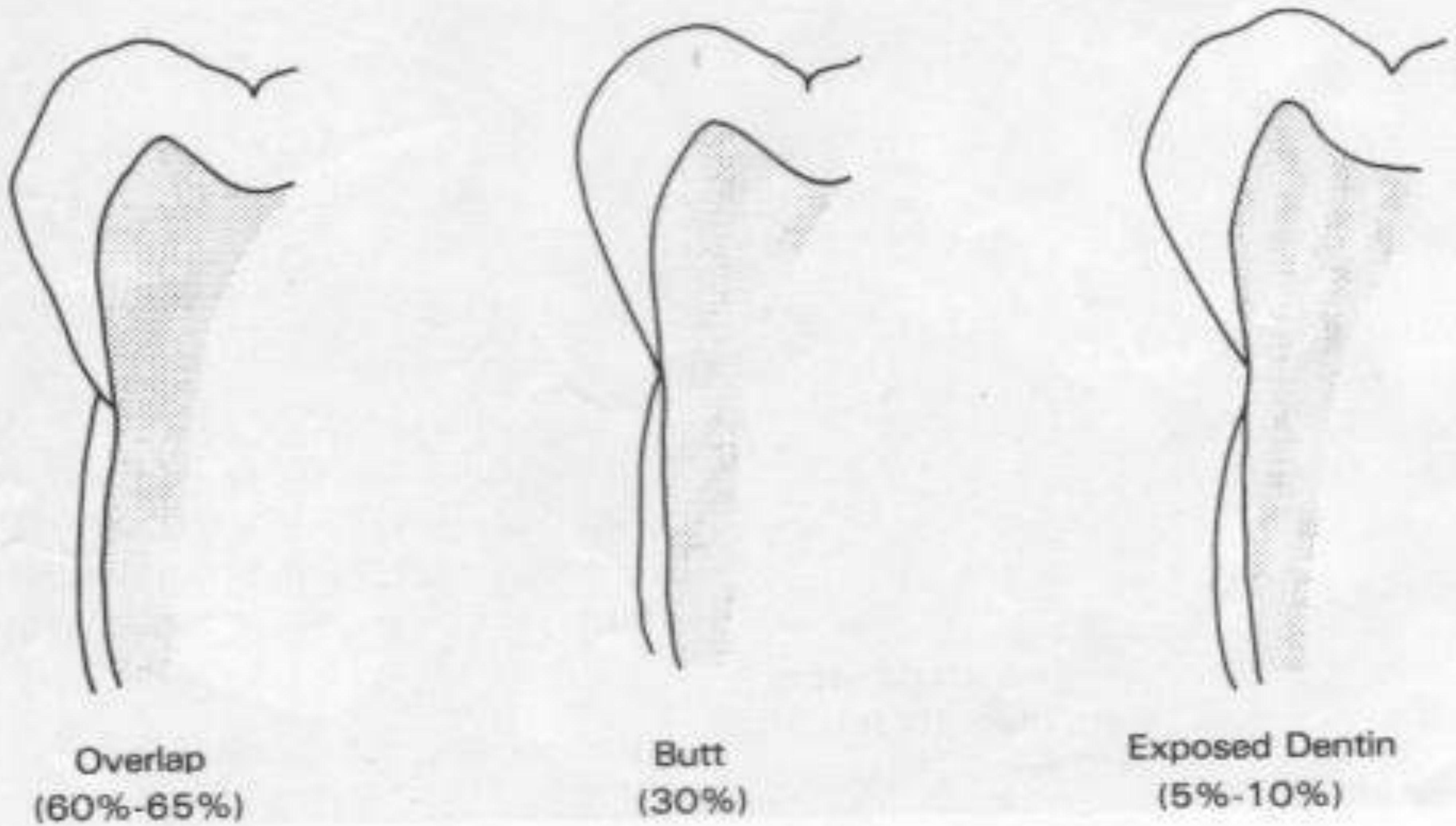
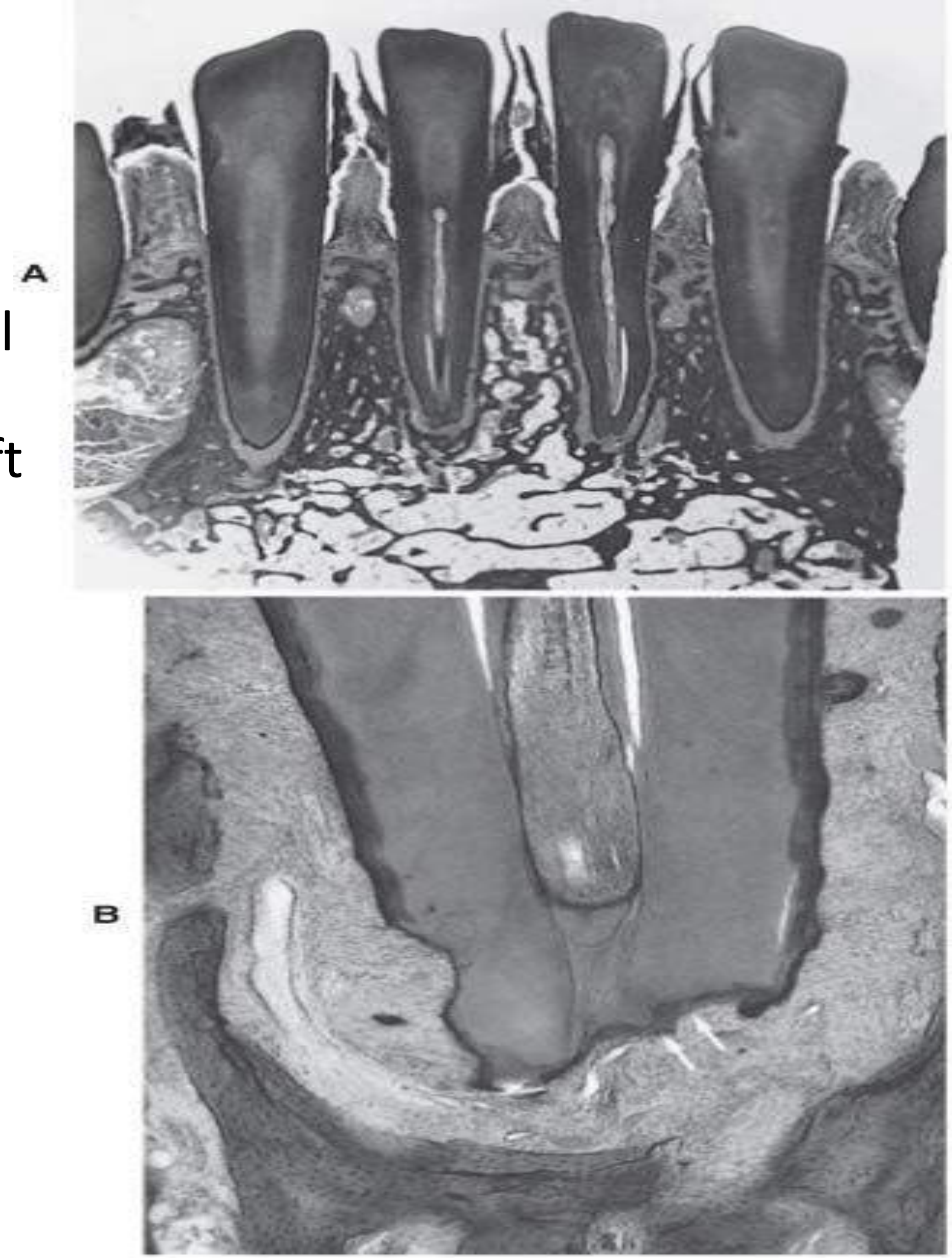


Fig. 1-10

Cementum Resorption and Repair

- Cementum resorption may be due to local or systemic causes (trauma from occlusion; orthodontic movement; cysts, and tumors; replanted and transplanted teeth calcium deficiency, hypothyroidism, Paget's disease).
- Cementum resorption is not continous, may alternate with periods of repair

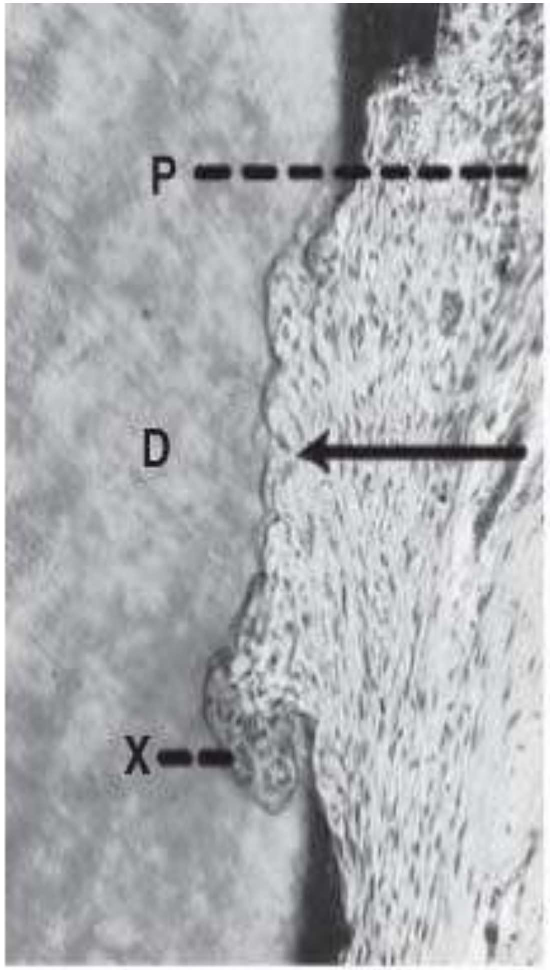
Cemental resorption associated with excessive occlusal forces. A, Low-power histologic section of mandibular anterior teeth. B, High-power micrograph of apex of left central incisor shortened by resorption of cementum and dentin. Note partial repair of the eroded areas (arrows) and cementicle at upper right.



Reversal line:

The newly formed cementum is demarcated from the root by deeply staining irregular line which delineated the border of the previous resoption.

Resorption of cementum and dentin. A multinuclear osteoclast in seen at X. The direction of resorption is indicated by the arrow. Note the scalloped resorption front in the dentin (D). The cementum is the darkly stained band at the upper and lower right. P, Periodontal ligament.



Trauma from occlusion:

- **Forces that exceed the adaptive capacity of the periodontium and produce injury.**

Chronic trauma from occlusion

- is more common than the acute form and is of greater clinical significance.

- It most often develops from gradual changes in occlusion produced by tooth wear, drifting movement, and extrusion of teeth, combined with parafunctional habits such as bruxism and clenching,

(CARRANZA'S clinical periodntology 9th edition - 396)

Hypercementosis:

Refer to a prominent thickening of the cementum, it may be localized to one tooth e.g. tooth without antagonists or with periapical lesion, and sometimes affect the entire dentition that may occur in patient with pagets diseses.

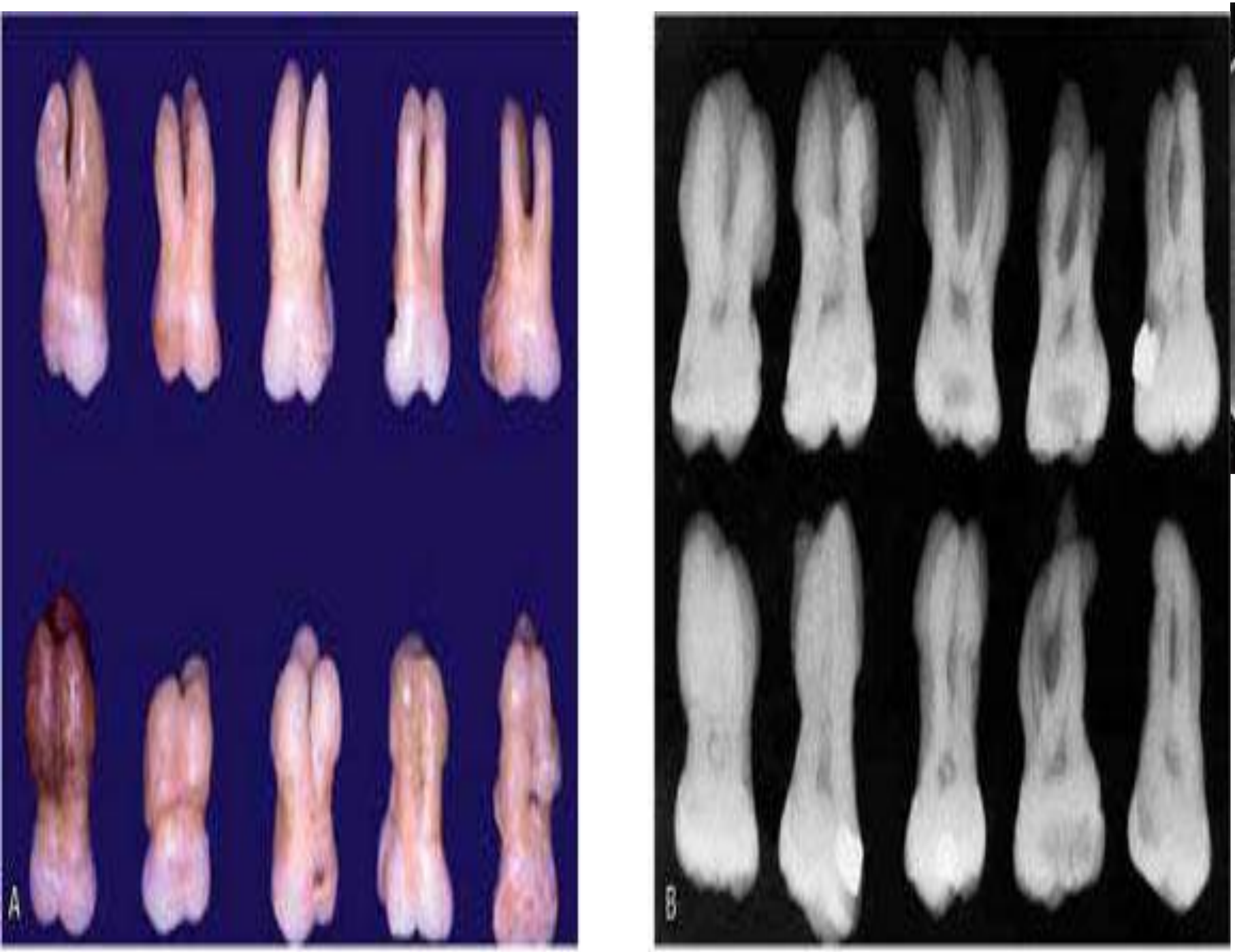


FIGURE 2 - Upper molars are the most affected teeth with hypercementosis, in various intensity levels, as these specimen, obtained from a sample of 21,573 teeth.[6]

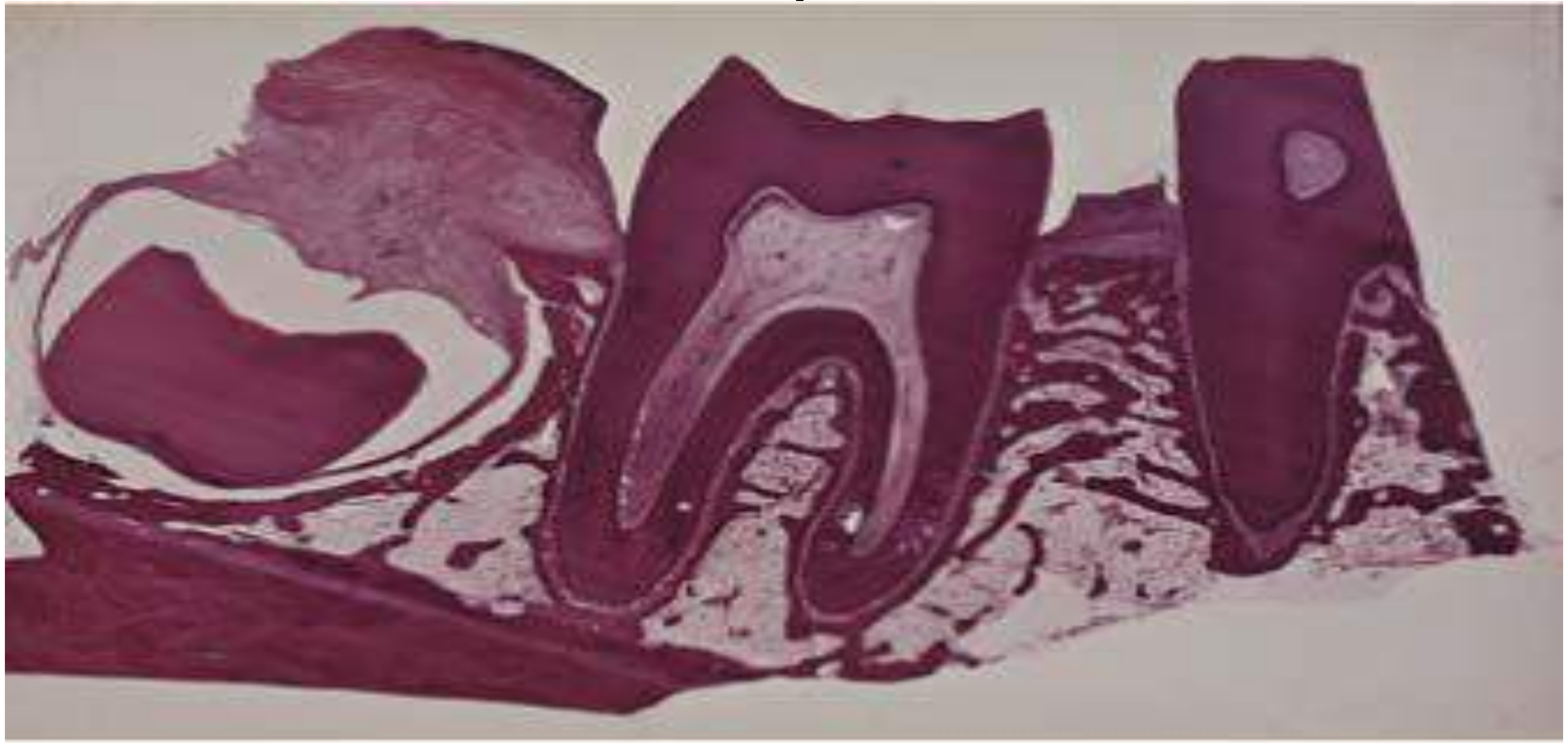
Ankylosis:

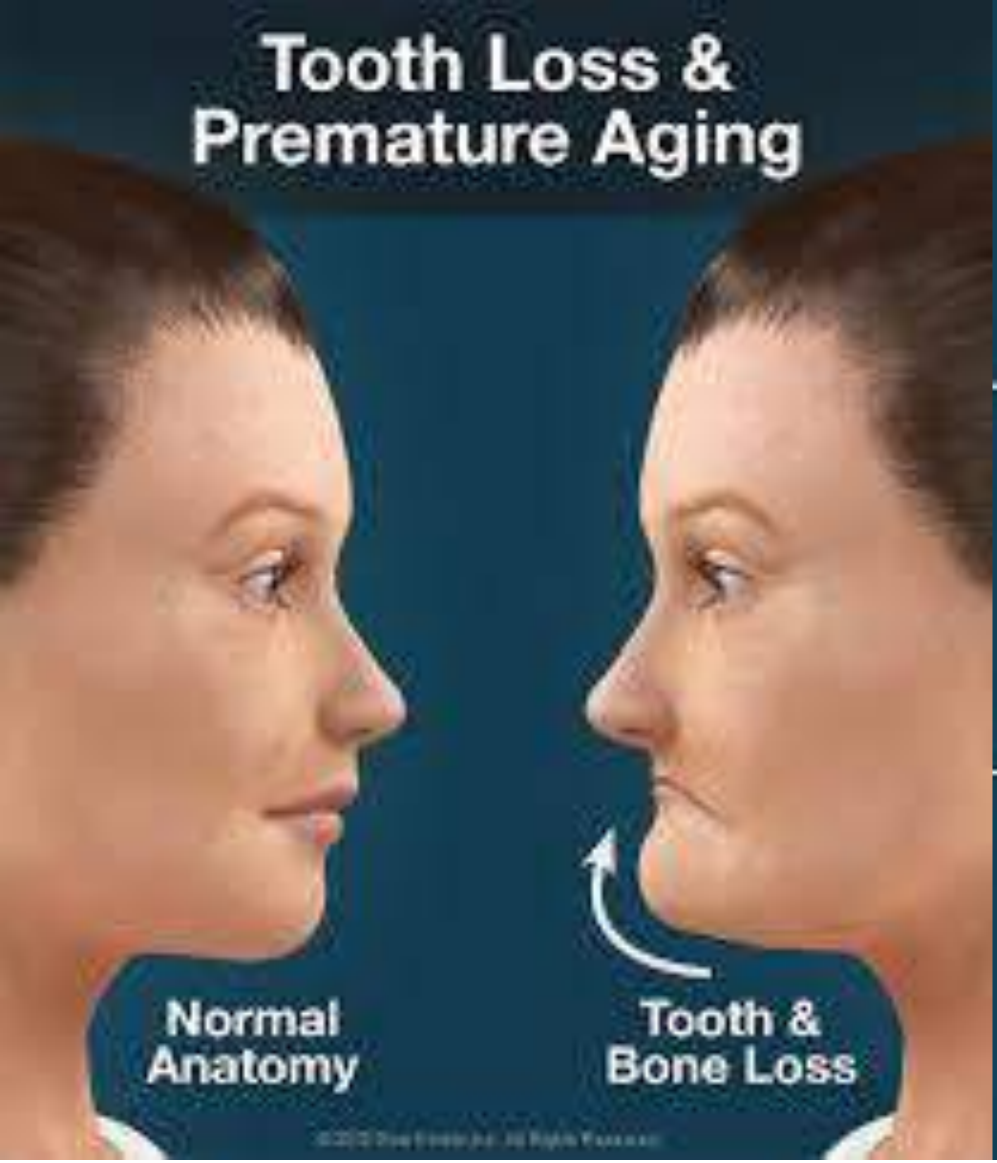
Fusion of the cementum and alveolar bone with obliteration of the PDL . It result in resorption of the cementum and it gradual replacement by bone tissue and it may develop after chronic periapical inflammation and occlusal trauma.

Fig. 2

Alveolar process(AP):

- **Is the portion of the maxilla and mandible that forms and support the tooth socket(alveoli).**
- **It develop in conjunction with the formation of and during the eruption of the teeth and it is gradually resorbed if the teeth are lost, thus it is tooth dependent structure.**




Tooth Loss &
Premature Aging
Normal
Anatomy
Tooth &
Bone Loss


Normal Anatomy
Tooth & Gradual Bone Loss
Tooth & Continued Bone Loss


Normal Jaw Bone
Jaw Bone After Long-Time
Denture Wear

Functions of alveolar process:

1-comprises the attachment apparatus and the supporting tissue of the teeth together with root cementum and PDL fibers.

2-provide the osseous attachment to PDL fibers.

3- Distribution and resorb forces generated by mastication and other tooth contact.

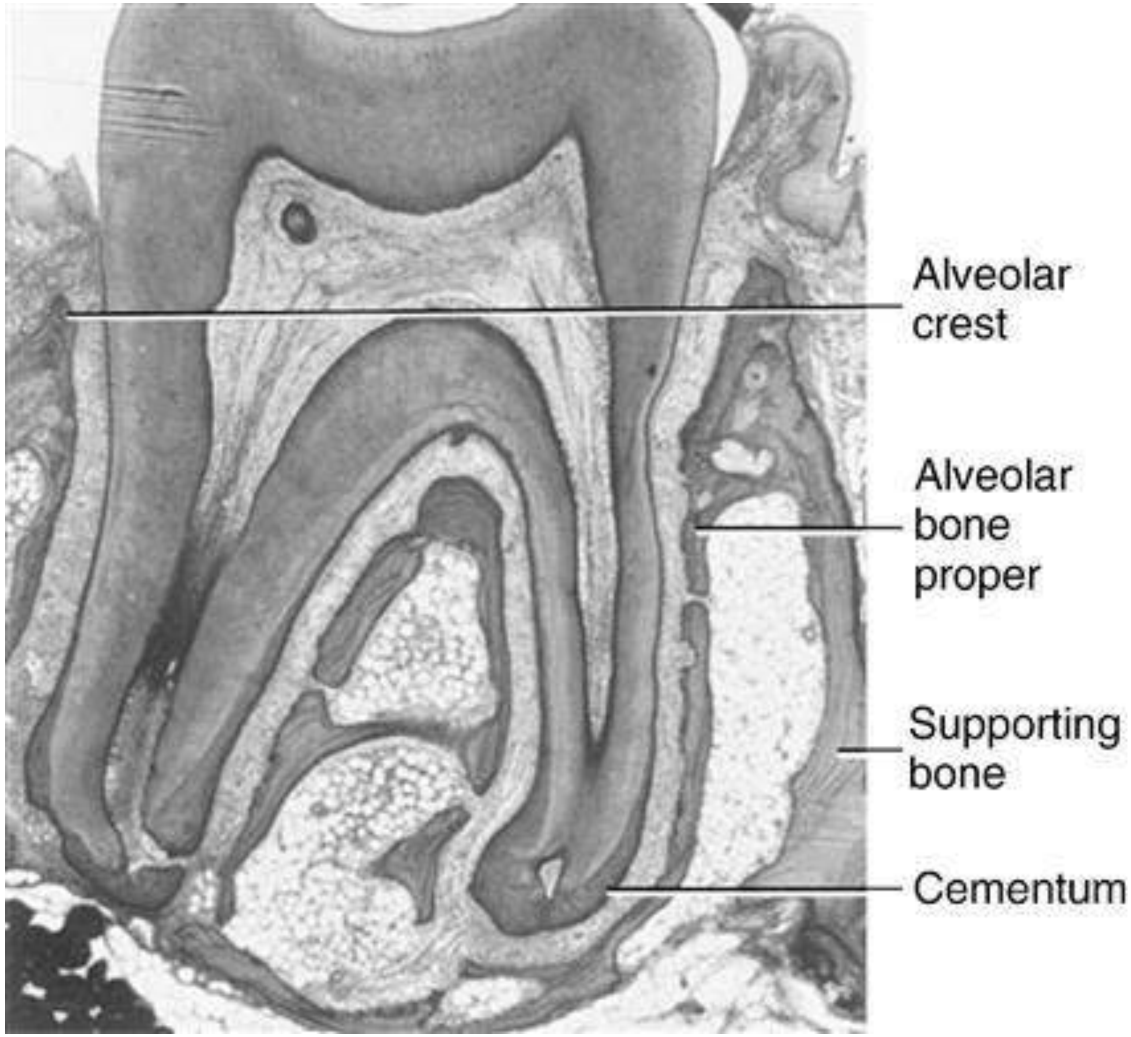
Alveolus: is the space in the alveolar bone that accommodates the roots of the teeth.

Parts of the alveolar process:

1- Alveolar bone proper (bundle bone) •

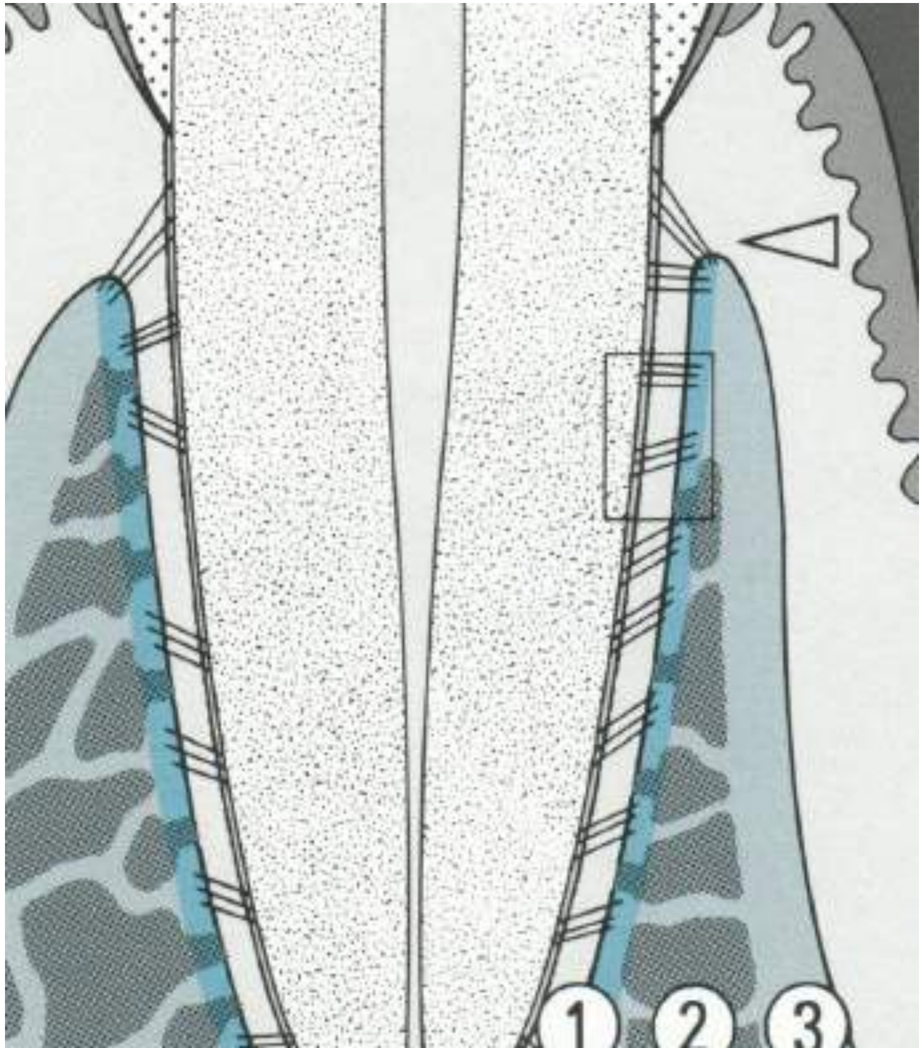
2- An external plate of cortical bone •

3- cacellous trabeculae or spongy bone •



Lamina Dura:

The layer of alveolar bone proper(ABP) appears a white line suurounding the root of the tooth on radiographs.



1 Alveolar bone, or

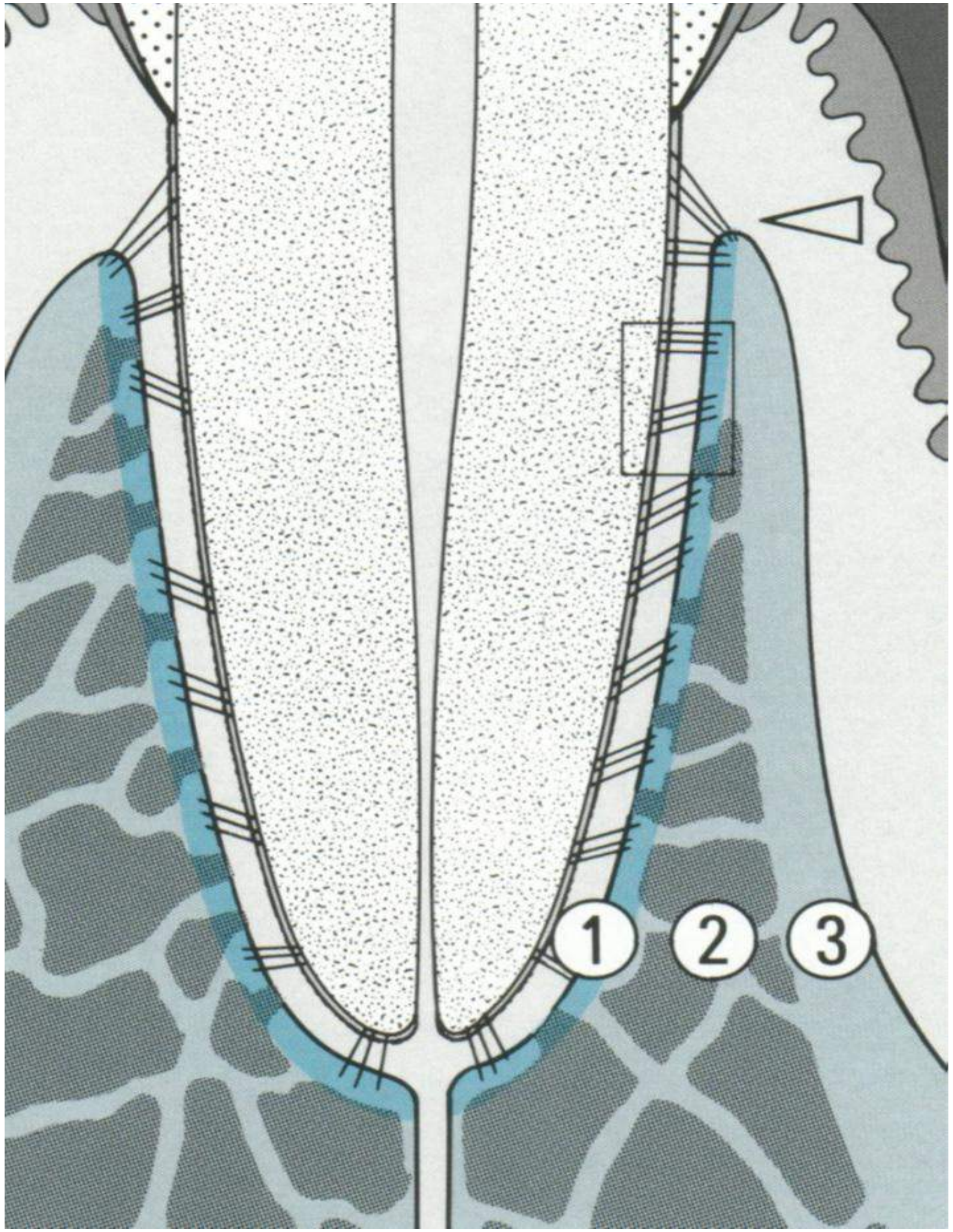
Cribiform plate

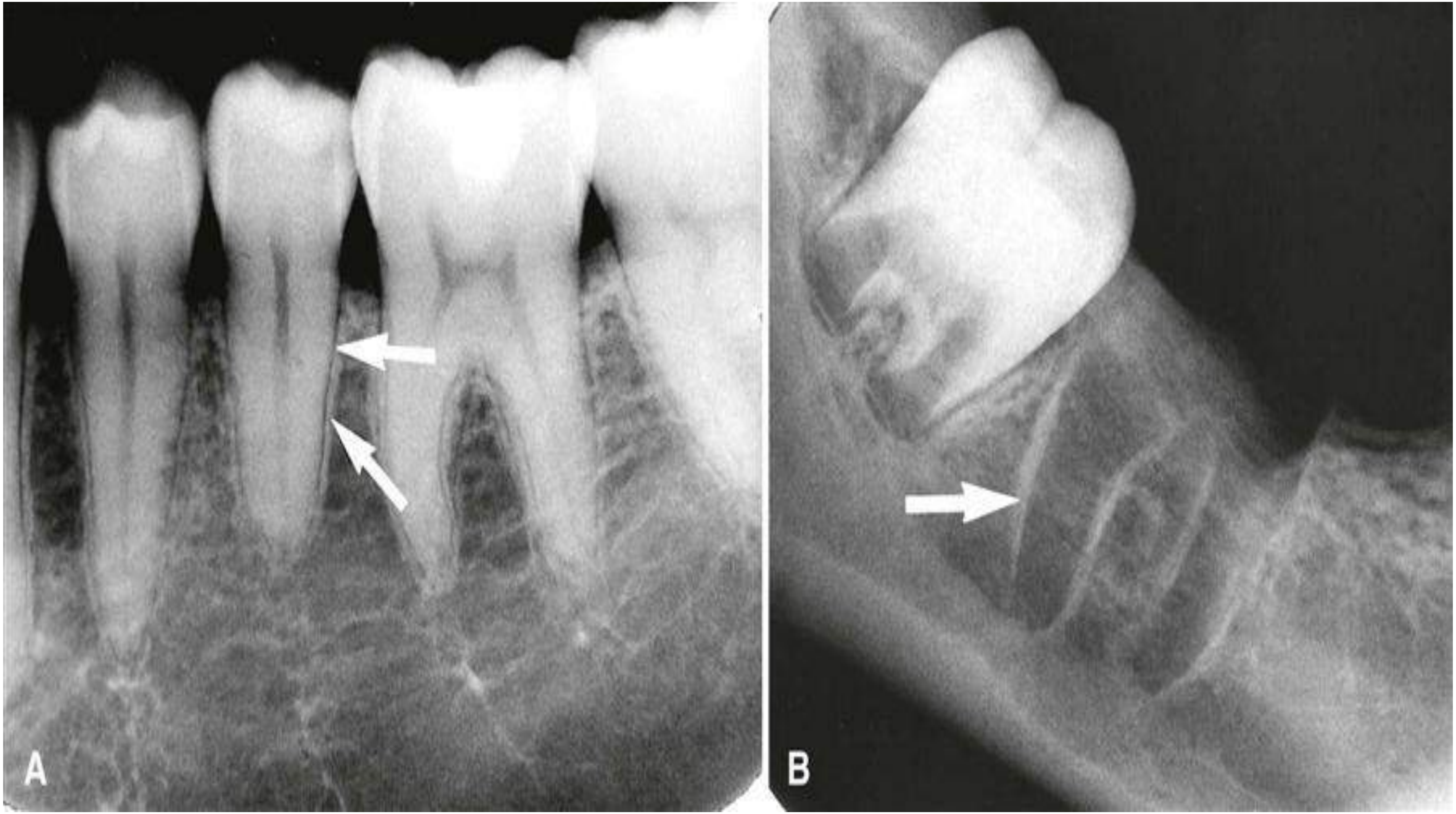
Alveolar wall

Lamina dura

2 Trabecular bone

3 Compact bone




A
B

The alveolar process are subdivided according to their anatomical relationship to the teeth :

1-Interproximal bone (interdental septum)(between two teeth)

2-Inter radicular bone.(between two root in multirooted teeth_

3-Radicular bone.(located in facial, palatal, lingual surface of teeth)

★ **The distance between crest of alveolar bone and C.E.J is about *2.81mm.***

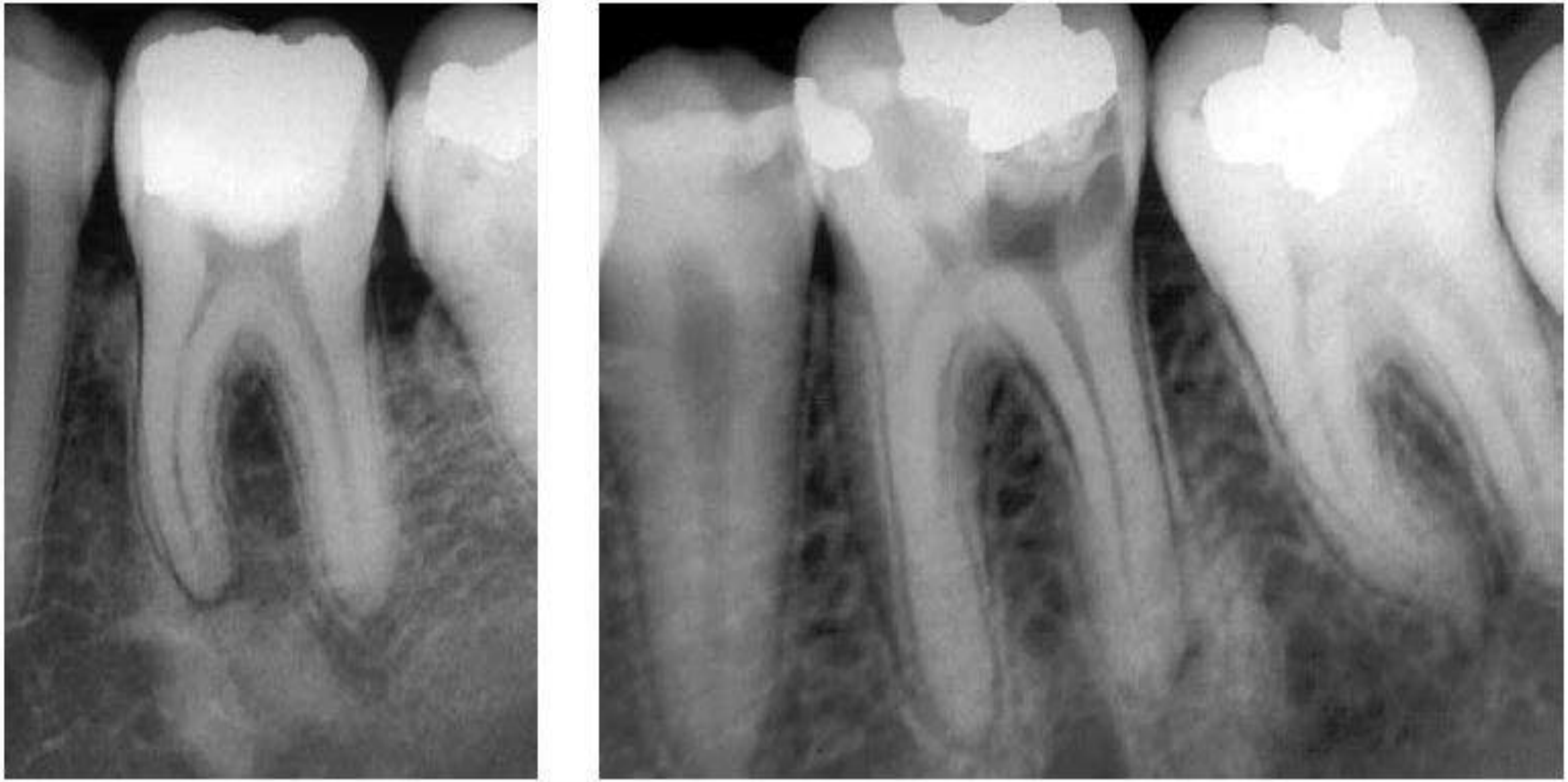
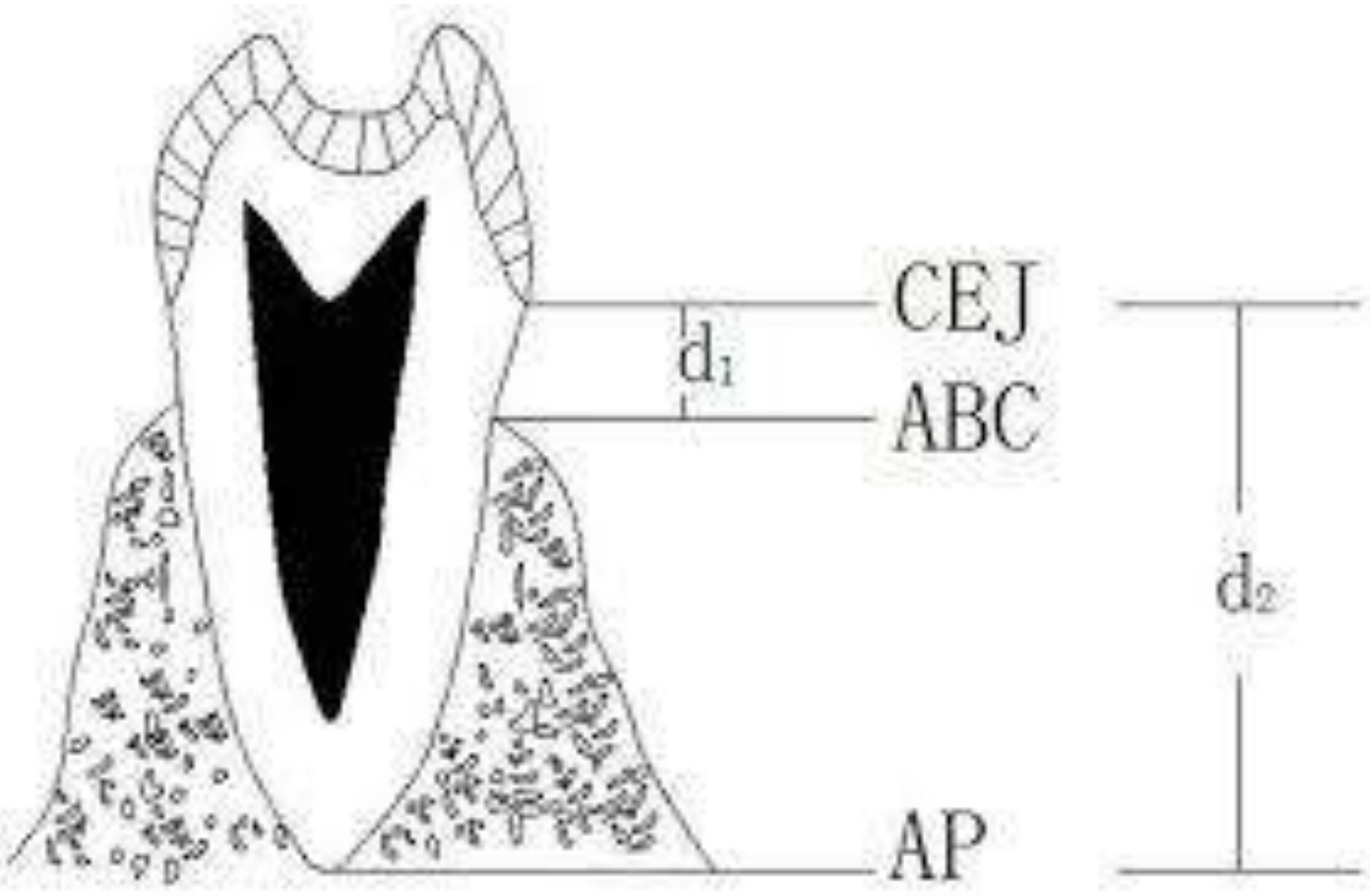
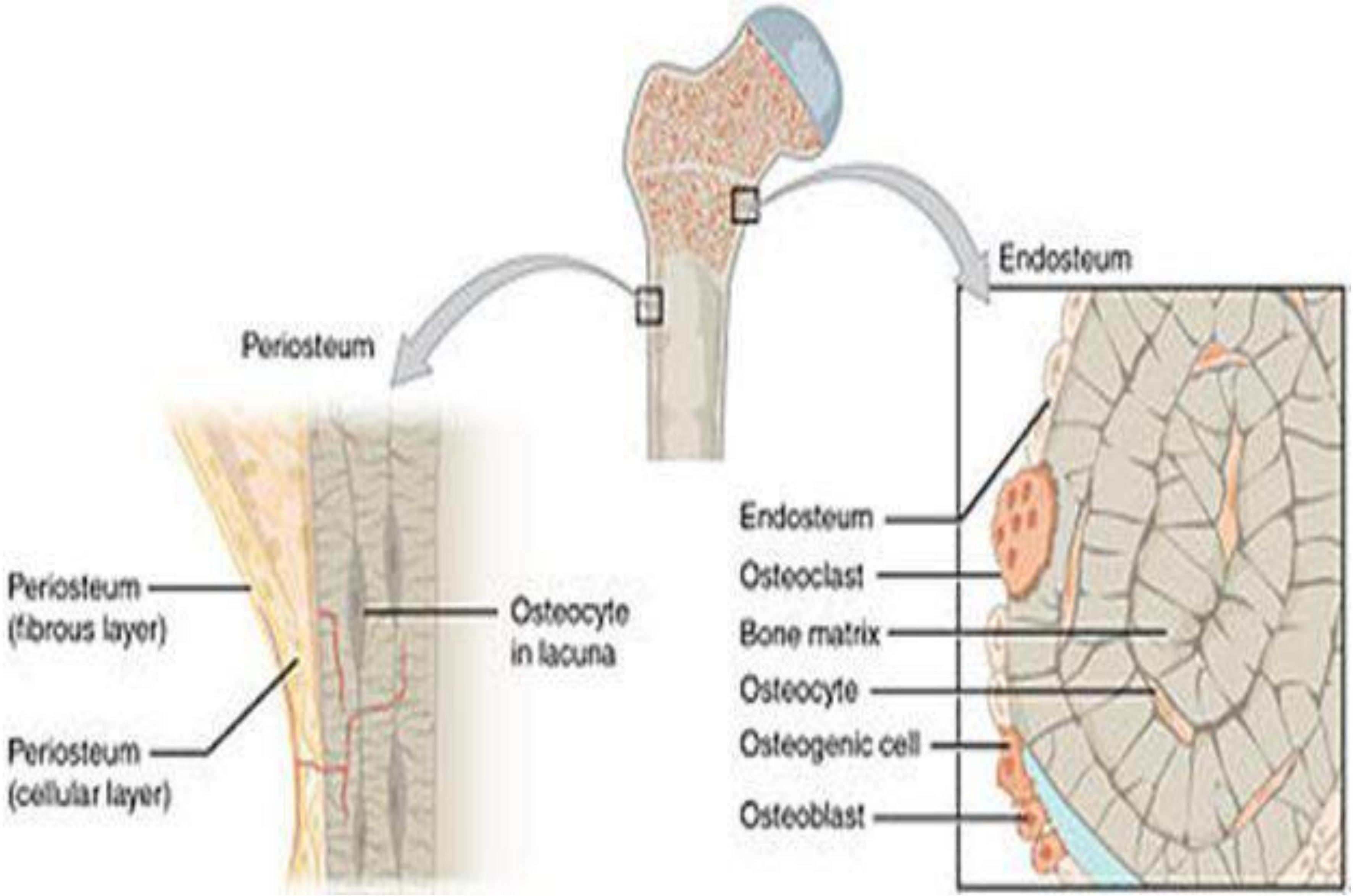


Figure 1 - Focal chronic sclerosing osteitis related to pulp necrosis and chronic periapical lesion, with small and irregular thin bone areas around the root canal opening, with predominant radiopaque images in the surroundings of thin bone areas.


CEJ
d1
ABC
d2
AP

Periosteum: it is a layer of tissue covering the outer surface of bone, it contain collagen fibers and cells(osteoblast) with blood vessels, nerves and fibers.

Endosteum: the marrow spaces inside the bone are lined by • endoteum, this tissue contain cells(osteoblasts).

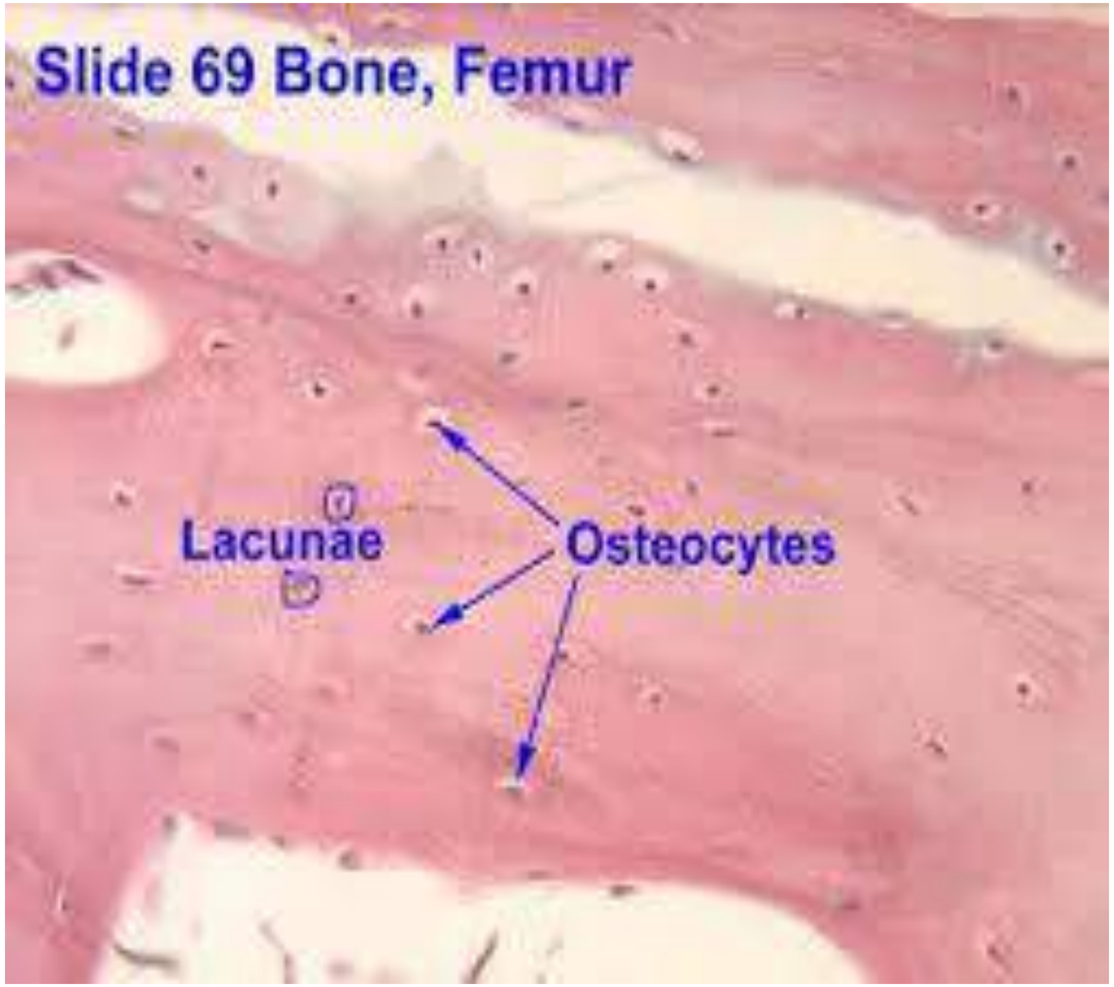

Endosteum
Periosteum
Periosteum
(fibrous layer)
Osteocyte
in lacuna
Periosteum
(cellular layer)
Endosteum
Osteoclast
Bone matrix
Osteocyte
Osteogenic cell
Osteoblast

Bone cells:

1-Osteoblast cells(bone forming cells).

2- osteoclasts cells.

3-osteocyte cells.



Composition of the bone:

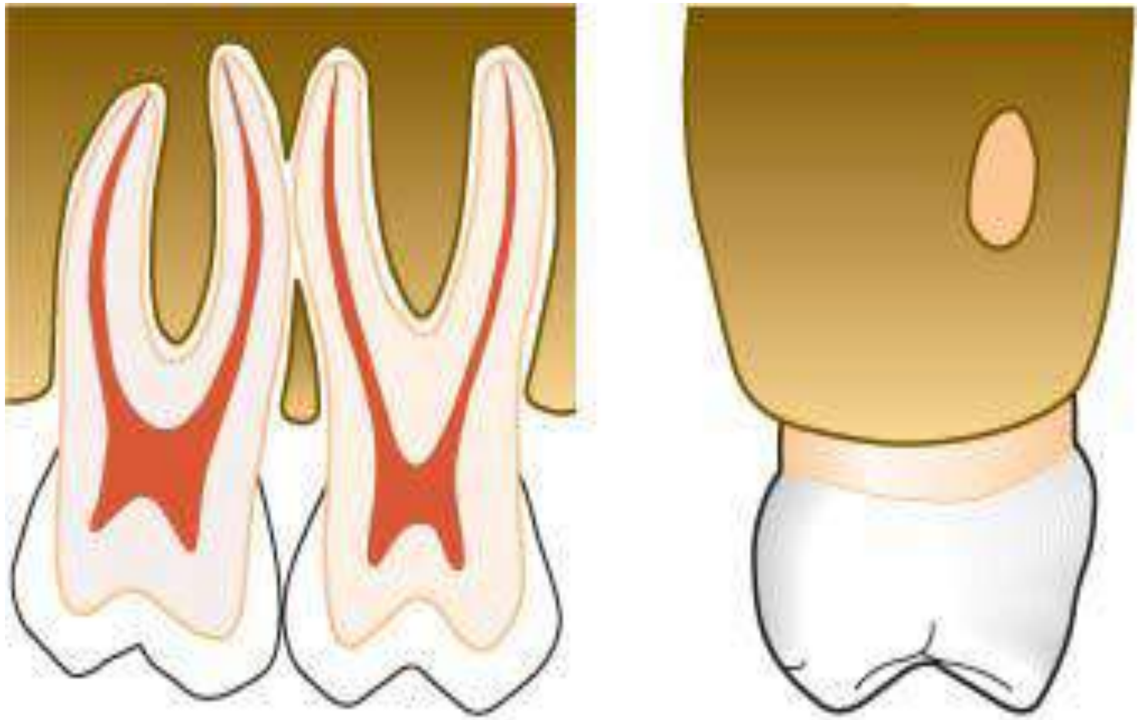
1- consist of 2/3 inorganic matter (minerals< •
calcium and phosphate>)

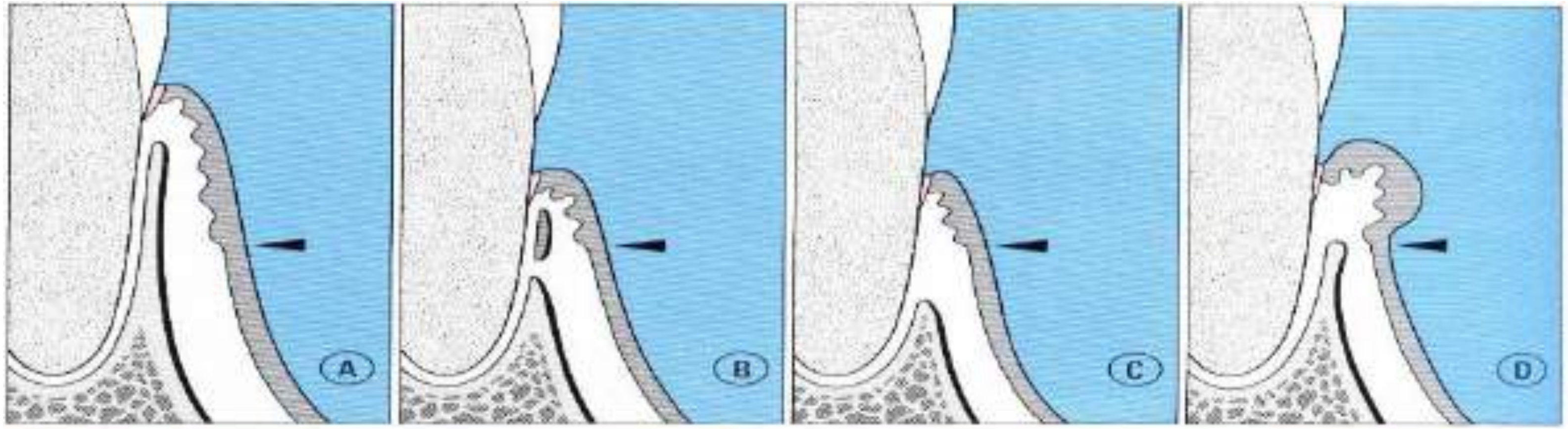
2- 1/3 organic matter consist of 90% of collagen •
fibers

Anatomical defects of bone:

1- Fenestration (Window)

2- Dehiscence




A
B
C
D

Remodeling of Alveolar Bone

- Internal remodeling (resorption and formation), which are regulated by local and systemic influences.
- Local influences include functional requirements on the tooth as well as age-related changes in bone cells.
- Systemic influences are probably hormonal (parathyroid hormone, calcitonin, and others).
- Remodeling of alveolar bone affects its height, contour, and density

Physiologic Migration of the Teeth

- **Tooth movement does not end when active eruption is completed and the tooth is in functional occlusion.**
- **With time and wear, the proximal contact areas of the teeth are flattened and the teeth tend to move mesially. This is referred to as physiologic mesial migration. By age 40, it results in a reduction of about 0.5 cm in the length of the dental arch from the midline to the third molars. Alveolar bone is reconstructed in compliance with the physiologic mesial migration of the teeth.**
- **Bone resorption is increased in areas of pressure along the mesial surfaces of the teeth, and new layers of bundle bone are formed in areas of tension on the distal surface**

Comparison of cementum upon bone:

Cementum like bone in many aspect but different in:

1- Has no innervation.

2- Has no blood or lymph vessels .

3- does not undergo physiological remodeling (resorption and deposition), but it is characterized by continuous deposition throughout life.

4-It is a microscopic organization.

بسم الله الرحمن الرحيم

Chemical plaque control

اعداد:

د. نور صباح ارحيم

Gingivitis and periodontitis are highly prevalent diseases and prevention of occurrence or recurrence is dependent on supra-gingival plaque control. Tooth cleaning is largely influenced by the compliance and dexterity of the individuals thus the concept of using chemical plaque control is just an adjunctive mean to overcome inadequacies of mechanical cleaning.

-**The action of theses chemicals could fit into four categories:** -

1-Anti-adhesive. -

2-Antimicrobial. -

3-plaque removal -

4-Anti-pathogenic. -

Anti-adhesive agents

They would act at the pellicle surface to prevent the initial attachment of the primary plaque forming bacteria and development of biofilms, although the amine alcohol, delmopinol, which appears to interfere with bacteria matrix formation and therefore fits between the concepts of anti-adhesion and plaque removal. Has been shown effective against plaque and gingivitis.

Antimicrobial agents

They could inhibit plaque formation through one of two mechanisms alone or combined. The first would be the inhibition of bacterial proliferation therefore could exert their effects either at the pellicle coated tooth surface before the primary plaque formation bacteria attach or after attachment but before division of these bacteria, this effect would be bacteriostatic in type while, the second effect could be bactericidal, whereby the antimicrobial agents destroys all of the microorganisms either attaching or already attached to the tooth surface.

Plaque removal agents

Such agents contained in a mouth rinse to reach all tooth surfaces and act in an identical manner to a tooth brush and remove bacteria from a tooth surface have attracted the terminology of the chemical tooth brush e.g. hypochlorites.

Anti-pathogenic agents

- These agents might inhibits the expression of plaque microorganisms pathogenicity without necessarily destroying them e,g. antimicrobial agents with bacteriostatic effects.

Chlorhexidine digluconate (CHX)

-CHX is frequently used as a mouth rinse(0.2% or 0.12%). The compound can also be applied as a gel, spray, varnishes, and has been incorporated into tooth paste, chewing gum, periodontal pack and sub gingival irrigation.

-at low concentration, CHX is bacteriostatic, at high concentration , it is bactericidal. The mode of action of CHX in killing bacteria is dependent upon the drug having access to cell walls.

This is facilitated by electrostatic forces, since CHX is positively charged , while the phosphate and carboxyle groups of bacterial cell walls carry negative charged . Binding causes disruption of the osmotic barrier and interfere with membrane transport.

- -rinsing with CHX reduce the number of bacteria in saliva by between 50% and 90%. A maximum reduction of 95% occur around 5 days. After which the number increase gradually to maintain an overall reduction of 70%-80% at 40 days.
- -An important properly of CHX is its substantivity that is, the retention in the mouth and subsequent release from oral structures. After a 1 minute oral rinse of 10ml CHX 0.2% approximately 30% of the drug is retained, within 15 seconds of rinsing, half will bonded to receptor molecules.

Clinical uses of CHX:

1-As an adjunct to oral hygiene and professional prophylaxis.

2-post oral surgery including periodontal surgery or rot planning.

3-for patients with jaw fixation.

4-medically compromised individuals predisposed to oral infections.

5-high risk caries patients.

6-in denture stomatitis.

7-oral mal Oder.

8-recurrent oral ulceration.

9-removable and fixed orthodontic appliance wearer.

10-immediate preoperative CHX rinsing and irrigation.

11-reduce salivary flow.

12-for oral and gingival health benefits in mentally and physically handicapped.

Side effects of CHX:

1-Brown discoloration of the teeth and some restoration materials and the dorsum of the tongue.

2-Taste perturbation where the salt taste appears to be preferentially affected to leave food and drinks with a rather bland taste.

3-Enhanced supra-gingival calculus formation.

4-Oral mucosal erosion.

5-unilateral or bilateral parotid swelling.

6-CHX also has a bitter taste which is difficult to mask completely.

-CHX is non toxic even if digested or topically applied and has a broad antimicrobial action including wide range of gram positive and gram negative m.o., it is slao effective against fungi and yeast including candida some viruses (HIV and HBV).

-It was demonstrated that rinsing for 60 seconds twice per day with 10ml of 0.2% gluconate solution in absent of tooth cleaning inhibited plaque regrowth and development of gingivitis. After that the patients should not eat or drink anything for up to 30min. With tooth brushing by using tooth paste, CHX mouth wash should be used after 30min. Of brushing ;otherwise cross reaction may occur and reduce the plaque inhibition of CHX.

-Studies suggest a slow release of CHX from surface to produce a persistence bacteriostatic action lasting for about 12hr. That's why it should be used twice a day.

THANK YOU

Classification of periodontal diseases and conditions (2017)

م. سها اسود دهش

B.D.S, MSc. Periodontology

ATTEMPTS AT CLASSIFICATION:

- Classification of disease is necessary to try to separate conditions into distinct categories so as to aid clinical and laboratory diagnosis and specific treatment.
- The criteria for separating diseases in this way should ideally be based on etiology, histopathology and, where appropriate, genetics rather than age of onset and rates of disease progression. Over the last three decades there have been **four major attempts** to classify periodontal disease.
- Major changes were made in the 1999 classification of periodontitis, which has been in use for the last 19 years. Periodontitis was reclassified as chronic, aggressive (localized and generalized), necrotizing and as a manifestation of systemic disease.

- The workshop in 2017 agreed on a classification framework for periodontitis further characterized based on a multidimensional staging and grading system that could be adapted over time as new evidence emerges

Classification of periodontal diseases and conditions (2017):

- **1- Periodontal health and gingival diseases and conditions**
  - **Periodontal health and gingival health**
  - **Dental biofilm induced gingivitis**
  - **Non-dental biofilm induced gingival disease**
- **2- Periodontitis**
  - **Periodontitis**
  - **Necrotizing periodontal diseases**
  - **Periodontitis as a manifestation of systemic disease**
- **3- Other conditions affecting the periodontium**
  - **Periodontal abscess and endodontic periodontal lesions**
  - **Mucogingival deformity and conditions**
  - **Traumatic occlussal force**
  - **Tooth and prosthetic related factors**

- **4- Peri-implant disease and conditions**
  - **Peri- implant health**
  - **Peri-implant mucositis**
  - **Peri-implantitis**
  - **Peri-implant soft and hard tissues deficiency**

1. Periodontal health and gingival diseases and conditions

- **Periodontal health and gingival health:**
- absence of clinically detectable inflammation.

Clinical gingival health can be restored following treatment of gingivitis and periodontitis.

bleeding on probing score (BOP%)

- assessed as the proportion of bleeding sites when stimulated by a standardized (dimensions and shape) periodontal probe with a controlled (~0.25 N) force to the apical end of the sulcus.

Clinical gingival health on an intact periodontium

- Absence (or minimum) bleeding on probing (less than 10%).
- Absence of attachment and bone loss.
- Probing depth ≤3mm.

Clinical gingival health on a reduced periodontium that include:

- **Non-periodontitis patient (e.g. recession, crown lengthening)**

➢ absence (or minimum) bleeding on probing (less than 10%).

➢ *presence of reduced clinical attachment and bone levels.*

➢ probing pocket depth ≤3.

- **Stable periodontitis patient:**

➢ absence (or minimum) bleeding on probing (less than 10%).

➢ *presence of reduced clinical attachment and bone levels.*

➢ probing pocket depth ≤4.

➢ no bleeding on probing at site with 4mm pocket depth.

Periodontal Health on a Reduced Periodontium in a Non-periodontitis Patient. This is a 54-year-old male patient with a **history of excessive frequent daily toothbrushing** (he brushed five to six times a day with a hard-bristled toothbrush).

Dental biofilm induced gingivitis

- An inflammatory lesion resulting from interactions between the dental plaque biofilm and the host's immune-inflammatory response, which remains contained within the gingiva and does not extend to the periodontal attachment (cementum, periodontal ligament and alveolar bone).

localized gingivitis(BOP score ≤10% and ≤30%)

Generalized gingivitis(BOP score > 30%)

Dental biofilm induced gingivitis

- **Gingivitis on an intact periodontium.**
- **Gingivitis on a reduced periodontium in a non-periodontitis patient (e.g., recession, crown lengthening).**
- **Gingival inflammation on a reduced periodontium in a successfully treated periodontitis patient (remission periodontitis).**

In the periodontitis patient, the term 'gingival inflammation' is used rather than 'gingivitis'

The classification of dental biofilm induced gingivitis:

- **A- Associated with bacterial dental biofilm only.**
- **B-Mediated by systemic or local risk factors**

Systemic conditions

Sex steroid hormones
Hyperglycemia
Leukemia
Smoking
Malnutrition

Oral factors enhancing plaque accumulation

- Prominent subgingival restoration margins
- Hyposalivation

Image 4

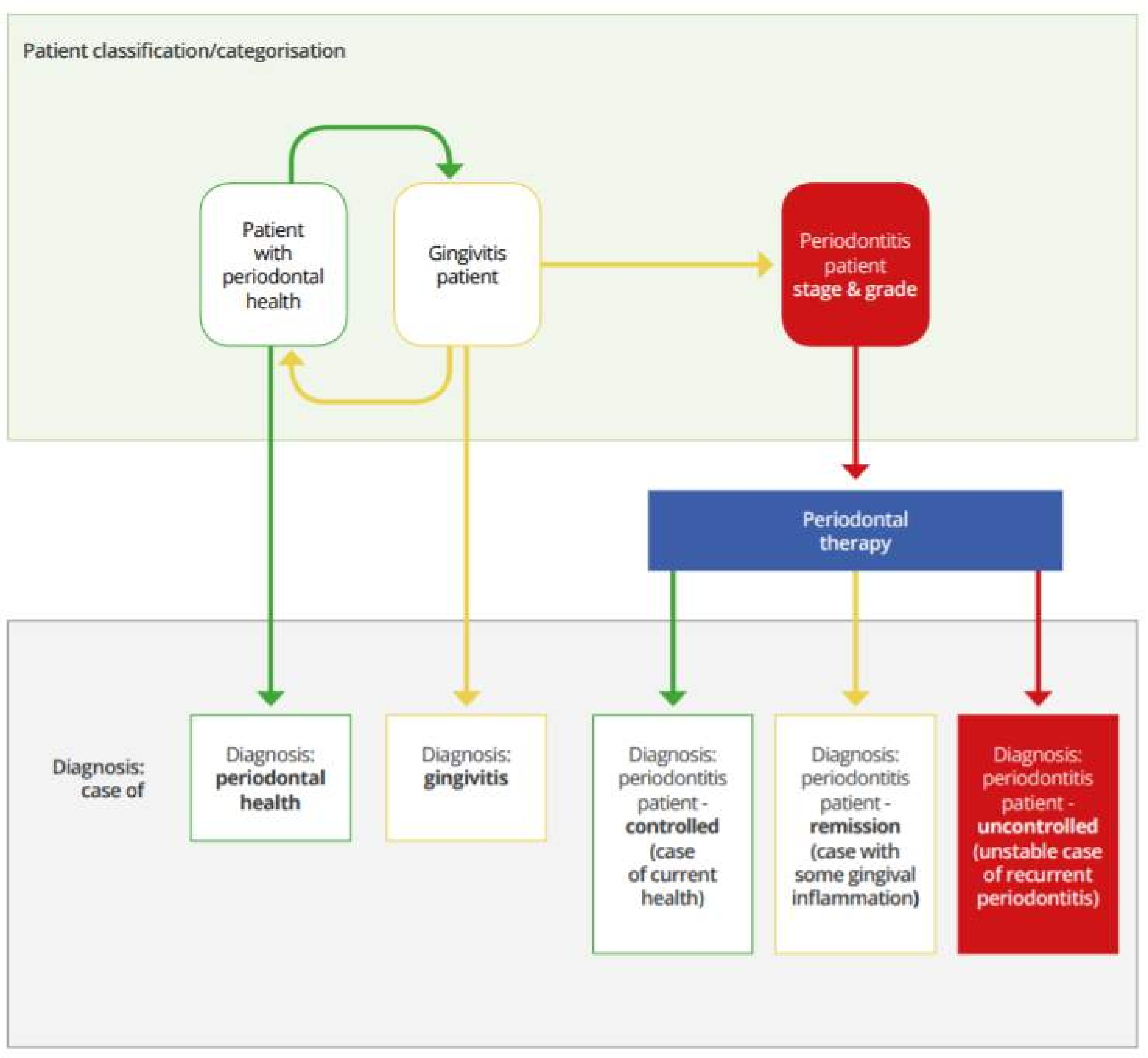
Predisposing factors (local risk factors), e.g. plaque retention factors

The classification of dental biofilm induced gingivitis:

- **C- Drug-influenced gingival enlargements**Drugs that may cause gingival overgrowth include anticonvulsant (e.g. phenytoin), immunosuppressant (e.g. cyclosporine A), and calcium channel blockers (e.g. nifedipine, verapamil).

The common clinical characteristics of drug-influenced gingival enlargement include:-

- Variation in interpatient and intrapatient pattern (genetic predisposition).
- Predilection for anterior gingiva.
- Higher prevalence in children and younger age group.
- Onset within 3 months of use.
- Change in the gingival contour leading to modification of gingival size.
- Enlargement first observed at the interdental papilla.
- Change in gingival color.
- Increased gingival exudate.
- Bleeding upon provocation.
- Pronounced inflammatory response of gingiva in relation to the plaque present.
- Reduction in dental plaque can limit the severity of the lesion.


Patient classification/categorisation
Patient with periodontal health
Gingivitis patient
Periodontitis patient stage & grade
Periodontal therapy
Diagnosis: case of
Diagnosis: periodontal health
Diagnosis: gingivitis
Diagnosis: periodontitis patient - controlled (case of current health)
Diagnosis: periodontitis patient - remission (case with some gingival inflammation)
Diagnosis: periodontitis patient - uncontrolled (unstable case of recurrent periodontitis)

The classification of Non- dental biofilm induced gingival lesions:

1- Genetic/developmental disorders

Hereditary gingival fibromatosis

2- Specific infections

A- Bacterial origin

- Neisseria gonorrhoeae (gonorrhea)
- Treponema pallidum (syphilis)
- Mycobacterium tuberculosis (tuberculosis)
- Streptococcal gingivitis (strains of streptococcus)

B- Viral origin

- Coxsackie virus (hand-foot-and-mouth disease)
- Herpes simplex 1/2 (primary or recurrent)
- Varicella-zoster virus (chicken pox or shingles affecting V nerve)

C- Fungal Candidosis

The classification of Non- dental biofilm induced gingival lesions:

3- Inflammatory and immune conditions and lesions

A- Hypersensitivity reactions

- Contact allergy
- Plasma cell gingivitis
- Erythema multiforme

B- Autoimmune diseases of skin and mucous membranes

- Pemphigus vulgaris
- Pemphigoid
- Lichen planus
- Lupus erythematosus

C. Granulomatous inflammatory conditions (orofacial granulomatosis)

- Crohn's disease
- Sarcoidosis

The classification of Non- dental biofilm induced gingival lesions:

4- Reactive processes

- Epulides

Fibrous epulis

5- Neoplasms

A- Premalignant

- Leukoplakia
- Erythroplakia

B- Malignant

- Squamous cell carcinoma
- Leukemia
- Lymphoma

6- Endocrine, nutritional, and metabolic diseases

•Vitamin deficiencies

The classification of Non- dental biofilm induced gingival lesions:

7- Traumatic lesions

A- Physical/mechanical insults

- Toothbrushing-induced gingival ulceration
- Factitious injury (self-harm)

B- Chemical (toxic) insults

- Etching
- Chlorhexidine
- Acetylsalicylic acid

C-Thermal insults

- Burns of mucosa

8- Gingival pigmentation

- Smoker's melanosis
- Amalgam tattoo

Thank you

Classification of periodontitis

م. سها أسود دهش العزاوي

B.D.S, MSc. Periodontology

In 1999 the periodontitis were classified in to:

- **<u>Chronic periodontitis:</u>**
- localized if ≤30% .
- generalized if >30% .
- Slight =1-2 mm CAL , moderate =3 - 4 mm CAL, and severe = ≤5 mm CAL.
- **<u>Aggressive periodontitis: (A.P.)</u>**
- localized if ≤30% .
- generalized if >30% .
- The term aggressive periodontitis replaced the previous name early-onset periodontitis (prepubertal, juvenile periodontitis & rapidly progressive periodontitis).

clinical features of chronic periodontitis

- Most prevalent in adults, but can occur in children and adolescents;
- Amount of destruction is consistent with the presence of local factors;
- Subgingival calculus is a frequent finding;
- Associated with variable microbial pattern;
- Slow to moderate rate of progression, but may have periods of rapid progression;
- Can be associated with local predisposing factors (e.g. tooth-related or iatrogenic factors);
- May be modified by and/or associated with systemic diseases (e.g. , diabetes mellitus)
- Can be modified by factors other than systemic diseases such as cigarette smoking and emotional stress.

clinical features of aggressive periodontitis

- Except for the presence of periodontitis, patients are otherwise clinically healthy;
- Rapid attachment loss and bone destruction;
- Familial aggregation;
- Amounts of microbial deposits are inconsistent with the severity of periodontal tissue destruction;
- Elevated proportion of *aggregatibacter actinomycetemcomitans* and, in some populations, *porphyromonas gingivalis*, may be elevated;
- Phagocyte abnormalities
- Progression of attachment loss and bone loss may be self-arresting.

- **Recently, based on pathophysiology, three clearly different forms of periodontitis have been identified according to new classification system proposed by the American Academy of Periodontology (AAP) and the European Federation of Periodontology (EFP) in 2017:**

1. **Periodontitis.**

2. **Periodontitis as a direct manifestation of systemic diseases**

3. **Necrotizing periodontitis**

Periodontal diseases and conditions

- Periodontal health, gingival diseases and conditions
  - Periodontal and gingival health
  - Gingivitis: biofilm induced
  - Gingival diseases: non biofilm induced
- periodontitis
  - Necrotizing periodontal diseases
  - Periodontitis as manifestation of systemic disease
  - Periodontitis
- Other conditions affecting the periodontium
  - Systemic diseases or conditions affecting the p.l supporting tissues
  - P.L abscesses &endodontic p.l lesions
  - Mucogingival deformeties&conditions
  - Traumatic occlusal forces
  - Tooth and prosthetic related factors

Peri-implant diseases and conditions

- Peri-implant health
- Peri-implant mucositis
- Periimplantitis
- Peri-implant soft and hard tissue difficiencies

Key elements in the new classification of periodontitis

- **Severity:** degree of periodontal breakdown.
- **Extent:** number and distribution of teeth with detectable breakdown.
- **Complexity of management:**

- Probing depths.
- Type of bone loss.
- Furcation lesions.
- Tooth mobility.
- Missing teeth.

- **Rate of progression.**
- **Risk factors:** smoker, diabetes

Staging and Grading system

I. **Periodontitis:** was classified according to different form of staging and grading. Staging relies on the standard dimensions of severity and extent of periodontitis at presentation.

The extent and distribution for each stage described as as molar/ incisor pattern or localized if the involved sites < 30% or generalized if the involved site ≤30%.

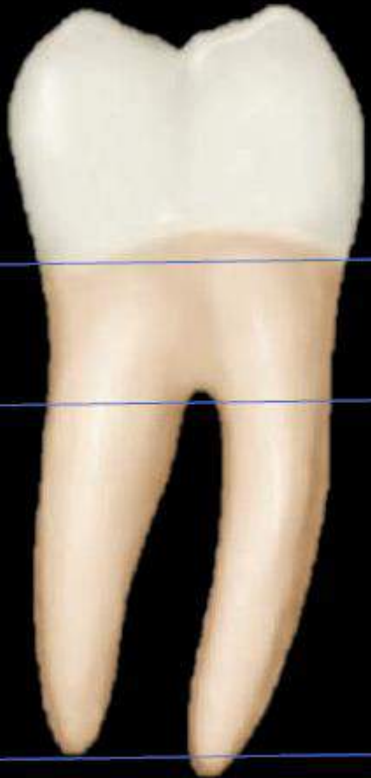
Severity

Guesstimate attachment loss

- *STAGE 1*: 1-2mm *4-5mm pocket*
- *STAGE 2*: 3-4mm *6-7mm*
- *STAGE 3*: >5mm *>8mm*
- *STAGE 4*: >5mm *>8mm*

Severity

Guesstimate radiographic bone loss



1/3 STAGE 1,2

2/3 STAGE 3,4

Staging and Grading Periodontitis

The 2017 World Workshop on the Classification of Periodontal and Peri-Implant Diseases and Conditions resulted in a new classification of periodontitis characterized by a multidimensional staging and grading system. The charts below provide an overview. Please visit **perio.org/2017wwdc** for the complete suite of reviews, case definition papers, and consensus reports.

PERIODONTITIS: STAGING

Staging intends to classify the severity and extent of a patient's disease based on the measurable amount of destroyed and/or damaged tissue as a result of periodontitis and to assess the specific factors that may attribute to the complexity of long-term case management.

Initial stage should be determined using clinical attachment loss (CAL). If CAL is not available, radiographic bone loss (RBL) should be used. Tooth loss due to periodontitis may modify stage definition. One or more complexity factors may shift the stage to a higher level. See **perio.org/2017wwdc** for additional information.

	Periodontitis	Stage I	Stage II	Stage III	Stage IV
Severity	**Interdental CAL** *(at site of greatest loss)*	1 – 2 mm	3 – 4 mm	≥5 mm	≥5 mm
	RBL	Coronal third (<15%)	Coronal third (15% - 33%)	Extending to middle third of root and beyond	Extending to middle third of root and beyond
	Tooth loss *(due to periodontitis)*	No tooth loss		≤4 teeth	≥5 teeth
Complexity	**Local**	• Max. probing depth ≤4 mm • Mostly horizontal bone loss	• Max. probing depth ≤5 mm • Mostly horizontal bone loss	In addition to Stage II complexity: • Probing depths ≥6 mm • Vertical bone loss ≥3 mm • Furcation involvement Class II or III • Moderate ridge defects	In addition to Stage III complexity: • Need for complex rehabilitation due to: – Masticatory dysfunction – Secondary occlusal trauma (tooth mobility degree ≥2) – Severe ridge defects – Bite collapse, drifting, flaring – < 20 remaining teeth (10 opposing pairs)
Extent and distribution	**Add to stage as descriptor**	For each stage, describe extent as: • Localized (<30% of teeth involved); • Generalized; or • Molar/incisor pattern			

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PERIODONTITIS: STAGING

STAGE 3,4

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TOOTH LOSS

Minimal complexity

HIGH COMPLEXITY

What is grading?

RATE
OF PROGRESSION

Grades of periodontitis:

- Irrespective of the stage at diagnosis, periodontitis may progress with different rates in individuals, may respond less predictably to treatment in some patients, and may or may not influence general health or systemic disease.
- Grading or rate of progression can be estimated by measurement of percentage of radiographical bone loss divided by the age of patient.
- **Grade A periodontitis:** is assigned if the maximum amount of radiographic bone loss in percentage terms is less than half the patient's age in years (for example, less than 30% in a 60-year-old or less than 40% in an 80-year-old)
- **Grade C periodontitis:** is assigned if the maximum amount of bone loss in percentage terms exceeds the patient's age in years (for example, more than 30% in a 28-year-old or more than 50% in a 49-year-old)
- **Grade B periodontitis:** all other situations

GRADING

START

- ***GRADE A:*** *Slow rat*
- ***GRADE B:*** *Moderate rate*
- ***GRADE C:*** *Rapid rate*

Steps to reach diagnosis

- **Step1**: Evaluate new patient and assess whether that patient is suspect of be a periodontitis case.
- **Step2**: Determine the extent&severity.
- **Step3**: Staging.
- **Step4**:Grading.

Periodontitis as a manifestation of systemic diseases :

1. **Systemic disorders that have a major impact on the loss of periodontal tissues by influencing periodontal inflammation:**

- ***A-*** Genetic disorders.
- B- Acquired immunodeficiency diseases .
- C- Inflammatory diseases :
- **2. Other systemic disorders that influence the pathogenesis of periodontal diseases**
- **3. Systemic disorders that can result in loss of periodontal tissues independent of periodontitis**

Necrotizing periodontal diseases:

- Necrotizing ulcerative gingivitis
- Necrotizing ulcerative periodontitis
- Necrotizing stomatitis

Other condition affecting the periodontium:

- **A. Periodontal abcesses and endodontic periodontal lesion**
- **Periodontal abscesses** (PA) :
- **1-Periodontal abscess in periodontitis patients.**

A- Acute exacerbation:

- In untreated periodontitis.
- In "refractory" periodontitis.

B- After different treatments:

- Scaling and root planing or professional prophylaxis
- Surgical periodontal therapy
- Systemic antimicrobial intake, without subgingival debridement
- Use of other drugs: e.g., nifedipine.
- **2- Periodontal abscess in non- periodontitis patients.**

previously called gingival abscess

PA can also occur in previously healthy sites because of:

- Impaction of foreign bodies
- Harmful habits
- Orthodontic factors
- Gingival enlargement
- Alterations of the root surface:

➢Severe anatomic alterations, such as invaginated tooth, dens evaginatus (grooves) or odontodysplasia.

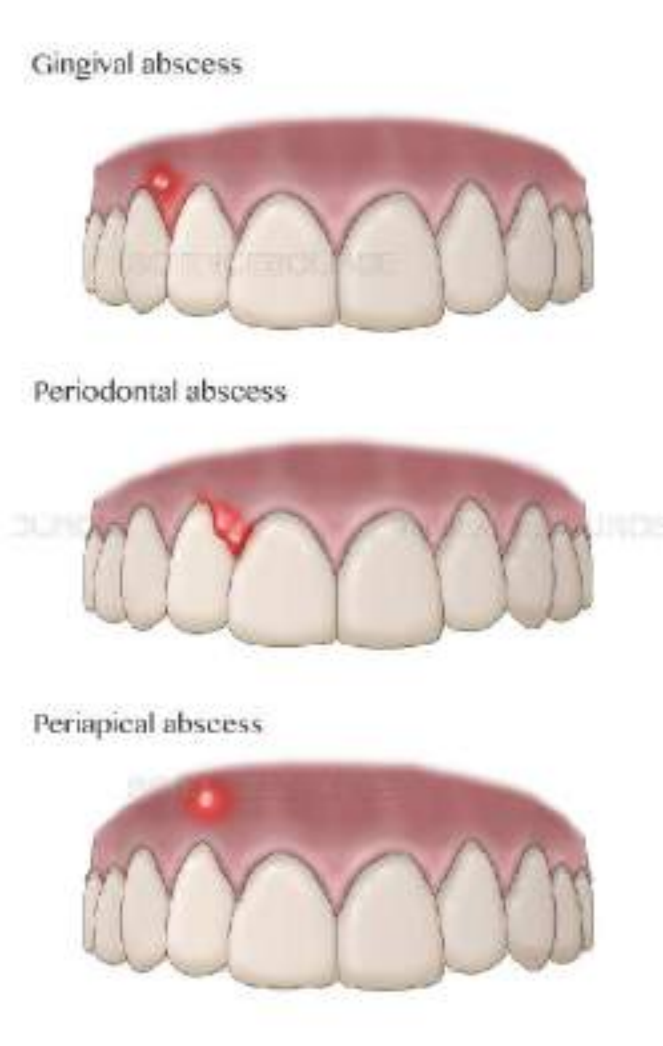
➢ Minor anatomic alterations, such as cemental tears, enamel pearls or developmental grooves.

- Iatrogenic conditions, such as perforations.
- vertical root fracture or cracked tooth syndrome extending through the root.
- External root resorption.

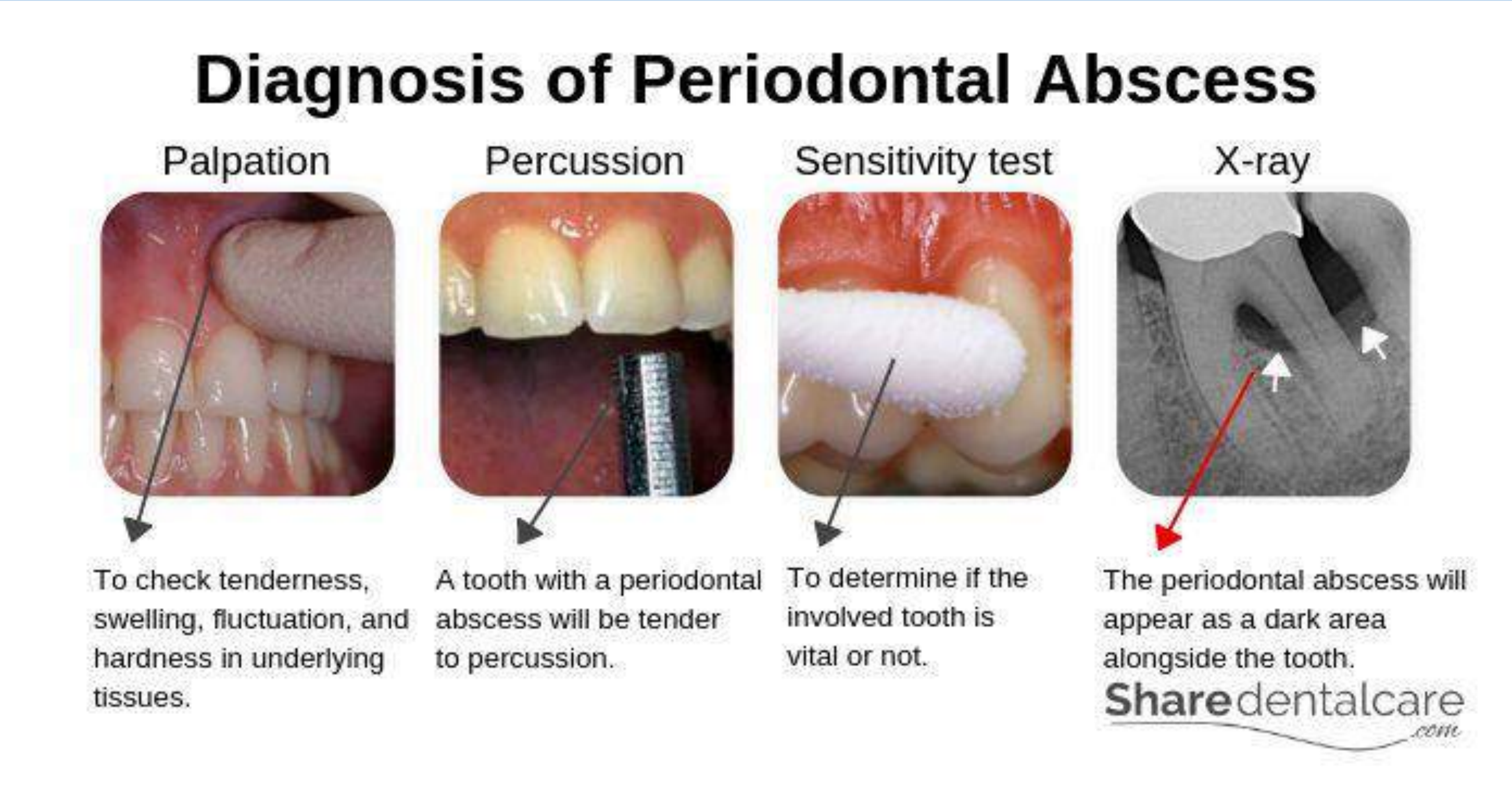
dens evaginatus

- PA may be associated with various combinations of the following clinical features:
- Pain,
- swelling,
- color change,
- tooth mobility,
- extrusion of teeth,
- purulence,
- sinus tract formation,
- fever, lymphadenopathy, and there may be a radiolucency of the affected alveolar bone.
- The acute periodontal abscess characterized by slight discomfort to severe pain and swelling. Chronic periodontal abscess is usually a symptomatic or with dull pain with a history of intermittent exudate.

The periodontal abscess need to be differentiated from the periapical abscess in the followings:



	Periodontal abscess	Periapical abscess
1.	The tooth is vital.	**Tooth is not vital.**
2.	The lesion lateral to the root surface.	**The lesion is most likely periapical.**
3.	X-ray finding shows area of radiolucency along the lateral surface of the root.	**X-ray finding shows apical radiolucency.**
4.	**The tooth is tender to lateral percussion.**	**Tooth tender to vertical percussion.**


Diagnosis of Periodontal Abscess
Palpation
Percussion
Sensitivity test
X-ray
To check tenderness, swelling, fluctuation, and hardness in underlying tissues.
A tooth with a periodontal abscess will be tender to percussion.
To determine if the involved tooth is vital or not.
The periodontal abscess will appear as a dark area alongside the tooth.
Sharedentalcare.com

Endodontic periodontal lesions:

- Clinical conditions involving both the pulp and periodontal tissues and may occur in acute or chronic forms.
- When they are associated with a recent traumatic or iatrogenic event (e.g. root fracture or perforation), the most common manifestation is an abscess accompanied by pain.
- endo- periodontal lesions, in subjects with periodontitis, normally present slow and chronic progression without evident symptoms.
- **The most common signs and symptoms :**
- **1-** Deep periodontal pockets reaching or close to the apex.
- **2-** Negative or altered response to pulp vitality tests.

- **The other signs and symptoms reported, in order of prevalence, are:**

1. bone resorption in the apical or furcation region,
2. spontaneous pain or pain on palpation and percussion,
3. purulent exudate,
4. tooth mobility,
5. sinus tract,
6. crown, and gingival color alterations

Endo-periodontal lesions associated with endodontic and periodontal infections might be triggered:

1. by a carious lesion that affects the pulp and, secondarily, affects the periodontium.
2. by periodontal destruction that secondarily affects the root canal.
3. or by both events concomitantly.

Endo-periodontal lesions associated with trauma and iatrogenic factors

- These conditions usually have a poor prognosis as they affect the tooth structure. The most common lesions in this category were:

1. root/pulp chamber/furcation perforation (e.g. because of root canal instrumentation or to tooth preparation for post retained restorations)
2. root fracture or cracking (e.g., because of trauma or tooth preparation for post-retained restorations)
3. external root resorption (e.g., because of trauma)

B- Mucogingival deformities or conditions around teeth

- gingival biotype
  - Thin scalloped
  - Thick scalloped
  - Thick flat
- Gingival/soft tissue recession
  - Facial or lingual surfaces
  - Interproximal (papillary)
  - Severity of recession
  - Gingival thickness
  - Gingival width
- Lack of keratinized gingiva
- Decreased vestibular depth
- Aberrant frenum/muscle position
- Gingival excess
  - Pseudopocket
  - Inconsistent gingival margin
  - Excessive gingival display
  - Gingival enlargement
- Abnormal color

gingival biotype

Fig 1 *Clinical appearance of thick gingival type.*

Fig 2 *Clinical appearance of thin gingival type.*

Gingival recession

- Is location of the gingival margin apical to the cemento- enamel junction.
- The causes of gingival recession:
  - Plaque accumulation will cause destruction of the junctional epithelia as a result of the inflammatory process.
  - Traumatic gingival recession:
    - Fault tooth brushing
    - Tooth malposition
    - High frenal attachment
    - Overhanging fillings
    - Prosthetic appliances
    - Habits as nail biting.

Miller's classification of gingival recessions

c. Tooth and prosthetic related factors :

- a- Localized tooth-related factors that modify or predispose to plaque-induced gingival diseases/periodontitis
  - Tooth anatomic factors
  - Root fractures
  - Cervical root resorption, cemental tears
  - Root proximity
  - Altered passive eruption

b-Localized dental prosthesis-related factors

- Restoration margins placed within the supracrestal attached tissues
- Clinical procedures related to the fabrication of indirect restorations
- Hypersensitivity/toxicity reactions to dental materials

d.Traumatic occlusal force

- Primary occlusal trauma
- Secondary occlusal trauma
- Orthodontic force

- <u>Occlusal trauma</u>: Injury resulting in tissue changes within the attachment apparatus as a result of occlusal force(s).
- **<u>Primary occlusal trauma</u>:** Injury resulting in tissue changes from traumatic occlusal forces applied to tooth or teeth with normal support. It occurs in the presence of:
- Normal bone levels, 2) Normal attachment levels, and 3) Excessive occlusal force(s).
- **<u>Secondary occlusal trauma</u>:** Injury resulting in tissue changes from normal or traumatic occlusal forces applied to a tooth or teeth with reduced support. It occurs in the presence of:
  - Bone loss, 2) Attachment loss, And 3) "Normal"/excessive occlusal force(s).

e.Peri-implant diseases and conditions

- **peri-implant health**
- In health, the peri-implant site is characterized by absence of erythema, bleeding on probing, swelling and suppuration.
- **peri-implant mucositis:** the diagnosis of peri-implant mucositis requires: Visual inspection demonstrating the presence of periimplant signs of inflammation: red as opposed to pink, swollen tissues as opposed to no swelling. Presence of profuse bleeding and/or suppuration on probing, an increase in probing depths compared to baseline; and absence of bone loss beyond crestal bone level changes resulting from the intial remodeling.
- **peri-implantitis**: the diagnosis of peri-implantitis will involve radiographic bone loss associated with gingival recession or increased probing depth in addition to signs associated with peri-implant mucositis

Thank you

Clinical

Periodontoly

Fourth grade

Presented by:

Dr. NOOR SABAH IRHAYYIM

-The by- products of bacterial plaque contribute to the development of both dental caries and periodontal diseases. At-home mechanical removal of plaque is a necessary part of preventing these diseases. Regular, effective plaque removal is essential for preventing or controlling caries and periodontal diseases.

Patient's motivation:

1- Information: Aims at increasing the compliance of the patient, so the patient recognizes oral health as a valuable goal of therapy, this can be achieved by following the step by step motivation system which includes simple demonstration for:

A-Symptoms of disease vs. healthy sites, bleeding, pocket depth, recession, etc.

Healthy gingiva, explain the normal, healthy features to your patient

Inflamed gingiva, show the patient the Signs of inflammation

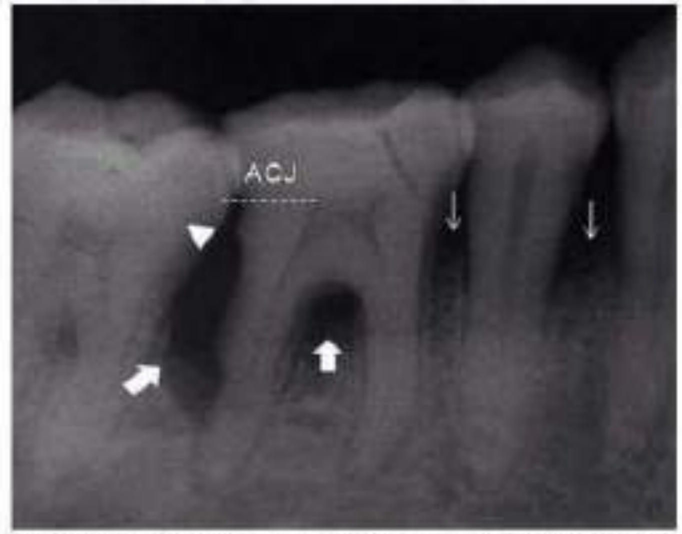
More pictures to be shown to your patient, explain to the patient what would happen if the periodontal disease left untreated (transition from health to disease):

-Healthy gingiva → Gingivitis → periodontitis

-*Gingivitis (reversible), periodontitis (irreversible* but treated to prevent further progression of the disease).

B-Radiographic bone height, normal vs. loss.

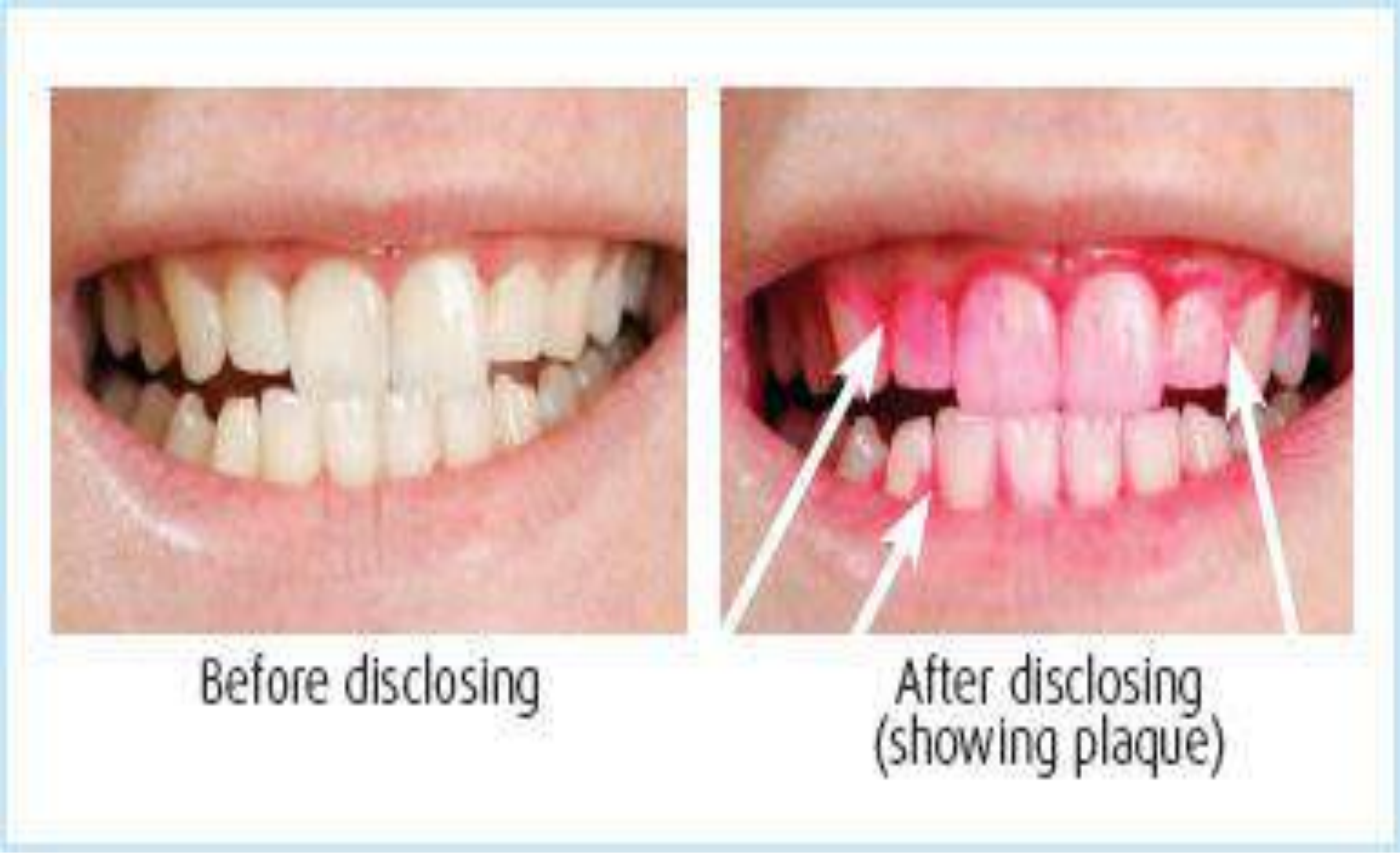
X-ray for normal periodontium



Periodontitis X-ray, show the patient the bone loss (arrows)

c- Explain reasons of disease; bacterial plaque.

d- Demonstrate plaque accumulation by using ***disclosing agent***, ex: erythrocin tablet, which converts the color of dental plaque into red.



-Example of disclosing agents: disclosing solution &erythrocin tablets.

2- Testing the acquirement of knowledge: by simply asking the patient to repeat what you already demonstrate in information step, if the patient seems not understand what you have explained then repeat the information step in simpler language.

3- Acquiring the knowledge will lead to **change in patient's attitude.**

4- Change in patient behavior is the logical result which is expected from motivation.

Oral Hygiene Instructions

Plaque control is the regular removal of microbial plaque and the prevention of its accumulation on the teeth and adjacent gingival surfaces. Microbial plaque is the major etiology of periodontal diseases and is related to dental caries; therefore gaining patient cooperation in daily plaque removal is critical to long-term success of all periodontal and dental treatment.

Mechanical plaque control

The most common method for plaque control since it is cheap, simple and doesn't required long time to perform:

- Brushing
- Interdental tools

Chemical plaque control

It is an adjunctive to mechanical means & not consider as alternative to mechanical cleanings.

This category includes: mouth wash, dentifrices & gel.

Professional plaque control

This method is expensive, time consuming & required man power& it is indicated only in extreme cases like: severe disability, coma, etc.

Brushing method

The objectives of tooth brushing are to remove plaque, debris from the teeth and to stimulate the gingiva. There are many tooth brushing techniques the patient can use to accomplish these goals.

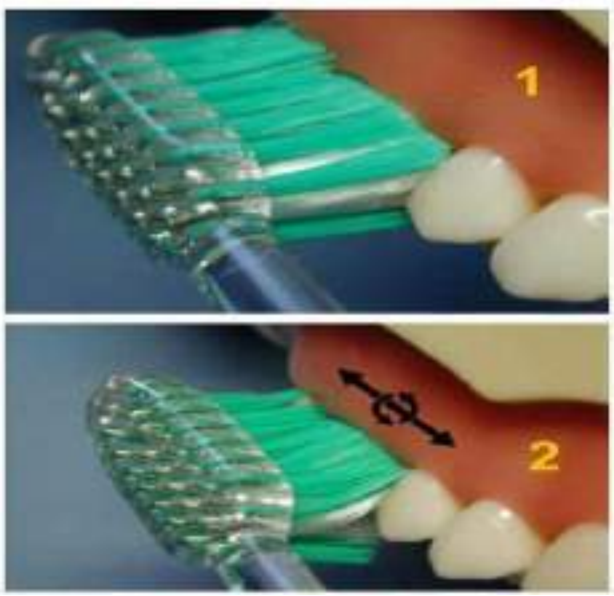
- Several methods suggested: Roll, Vibrating, Circular, Vertical, and Horizontal.

- No method is clearly superior.

-illingness and ability are more important than the technique.

1- Sulcular (Bass) method:

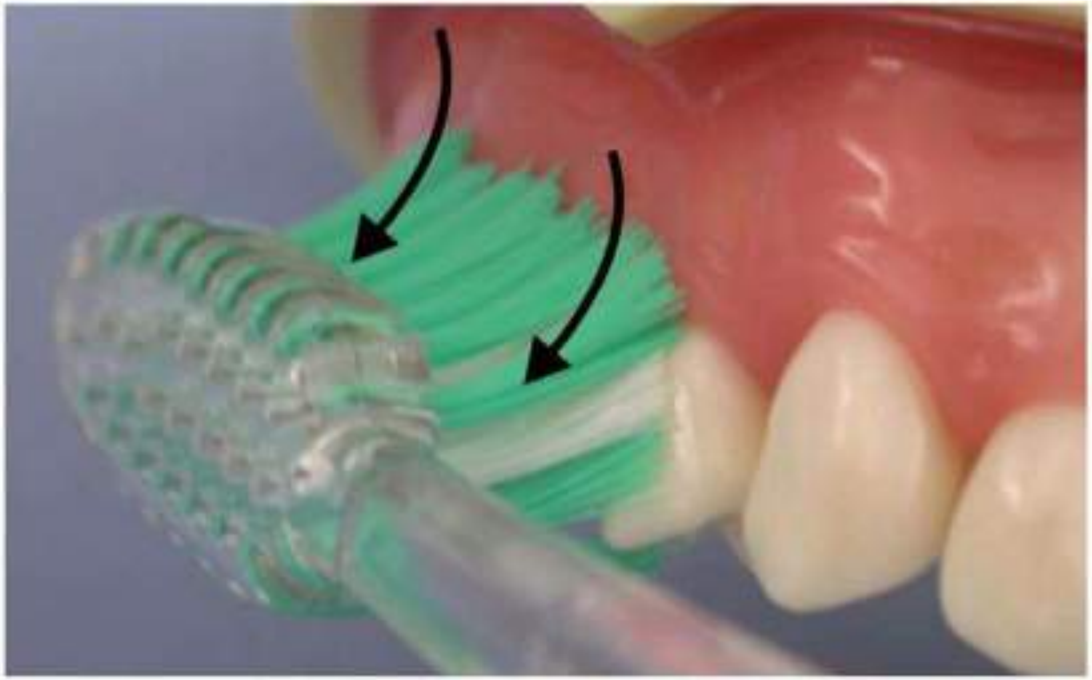
The bristles pointed at a 45- degree angle into the gingival sulcus, vibrate the brush gently forth & back about 20 times. This method is useful for patients with periodontal problems.



1. 45° angle to the gingival margin
2. Vibrate

2-Rolling method:

This method is useful for stimulation of the gingiva. Place the brush above the free gingiva, exerting slight pressure; draw the brush toward the occlusal surface.

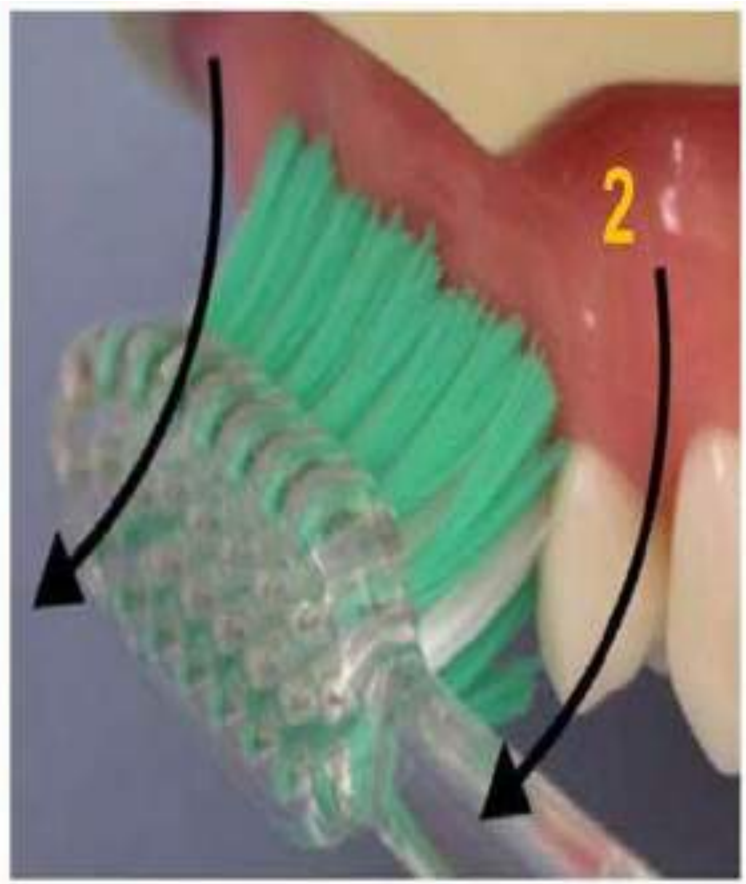


3- Charter´s method:

This method is useful for patients with severe loss of interdental papilla height, fixed prosthesis, previous gingival surgery, or subsided ulcerative gingivitis.

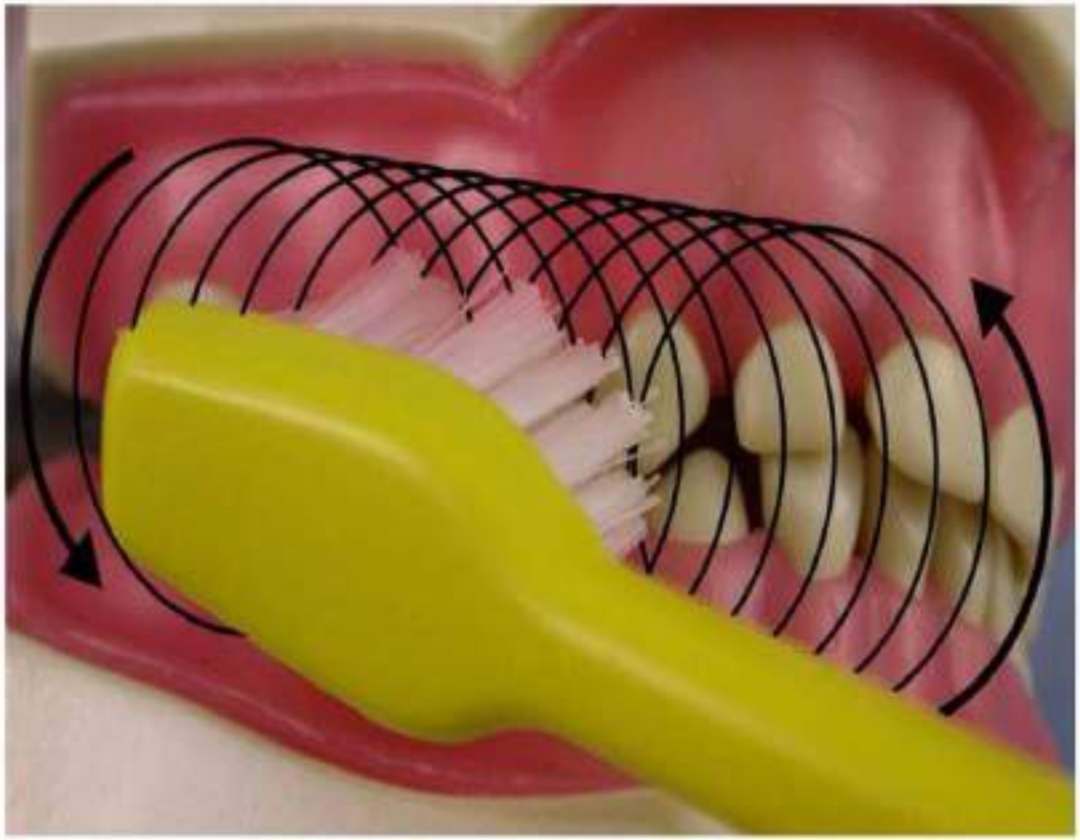
4- Modified stillman´s method:

1. Press and Vibrate



2. Roll

5- fone´s method:



Toothbrush

Toothbrushes vary in size and design as well as in length, hardness, and arrangement of the bristles.

However, all agree that use of hard toothbrushes, vigorous horizontal brushing, and use of extremely abrasive dentifrices may lead to cervical abrasions of teeth and recession of gingiva.

Vigorous brushing is not necessary and can lead to gingival recession, wedge-shaped defects in the cervical area of root surfaces, and painful ulceration of the gingiva.

The American Dental Association (ADA) recommends that individuals brush twice per day and use floss or other interdental cleaners once per day to effectively remove microbial plaque and prevent gingivitis. Toothbrushes must also be replaced periodically every 3 to 4 months.

Vigorous tooth brushing

with an abrasive dentifrice can result

in trauma to the gingiva and wearing

away of the tooth surfaces, especially

root surfaces, and can contribute to gingival recession.

Powered toothbrush

Powered toothbrushes have been shown to improve oral health for (1) children and adolescents, (2) children with physical or mental disabilities, (3) hospitalized patients, including older adults who need to have their teeth cleaned by caregivers, and (4) patients with fixed orthodontic appliances.

The vibrations have also been shown to interfere with bacterial adherence to oral surfaces.

INTERDENTAL CLEANING AIDS

Periodontal lesions are predominantly found in interdental locations, so tooth brushing alone is not sufficient to control gingival and periodontal diseases. It has been demonstrated in healthy subjects that plaque formation begins on the interproximal surfaces where the toothbrush does not reach.

Patients need to understand that the purpose of interdental cleaning is to remove microbial plaque, not just dislodge food wedged between teeth.

Many tools are available for interproximal cleaning and they should be recommended based on the size of interdental spaces, presence of furcations, tooth alignment, and presence of orthodontic appliances or fixed prostheses.

Also, ease of use and patient cooperation are important considerations.

Common aids are dental floss and interdental cleaners such as wooden or plastic tips and interdental brushes.

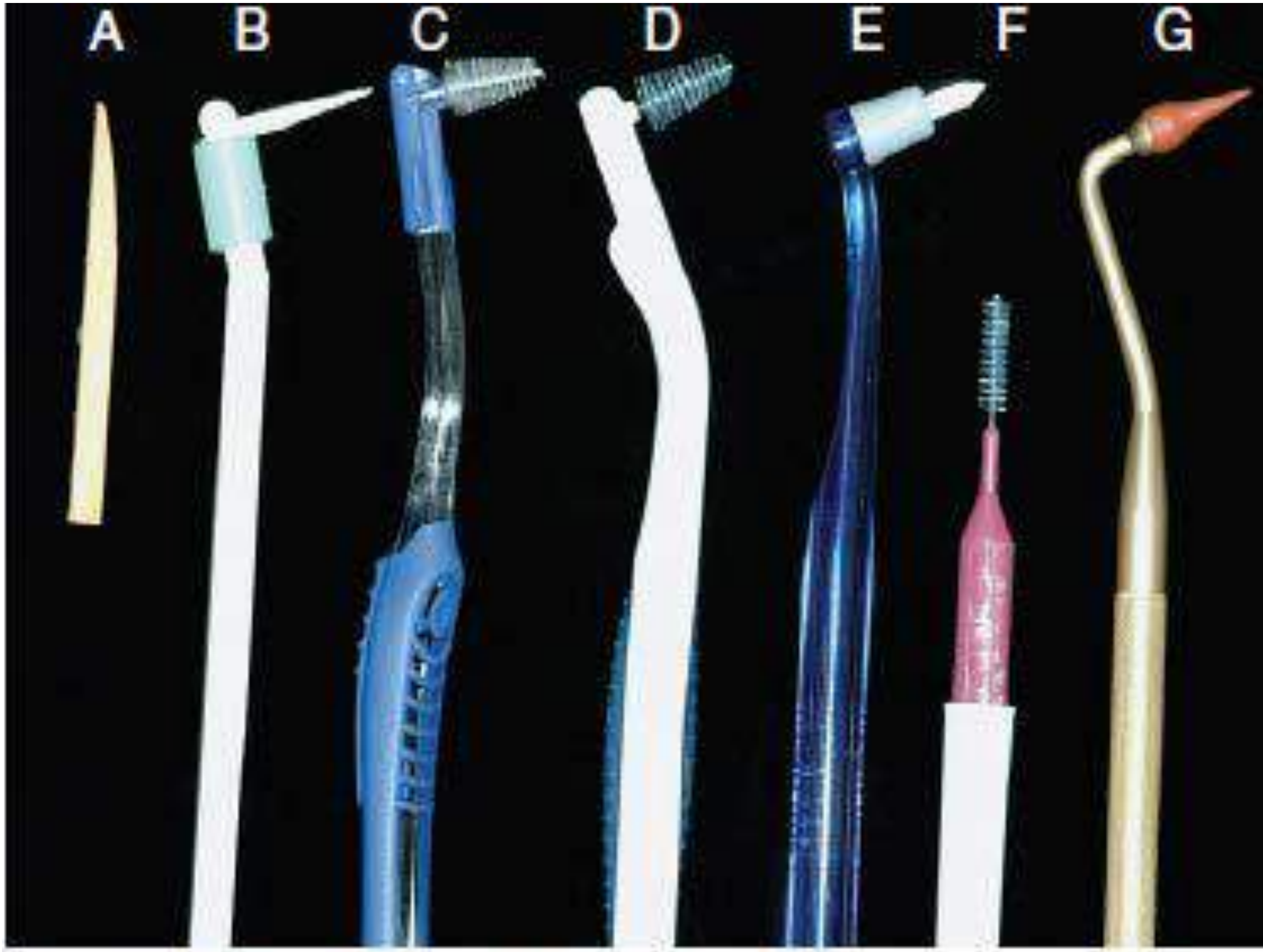


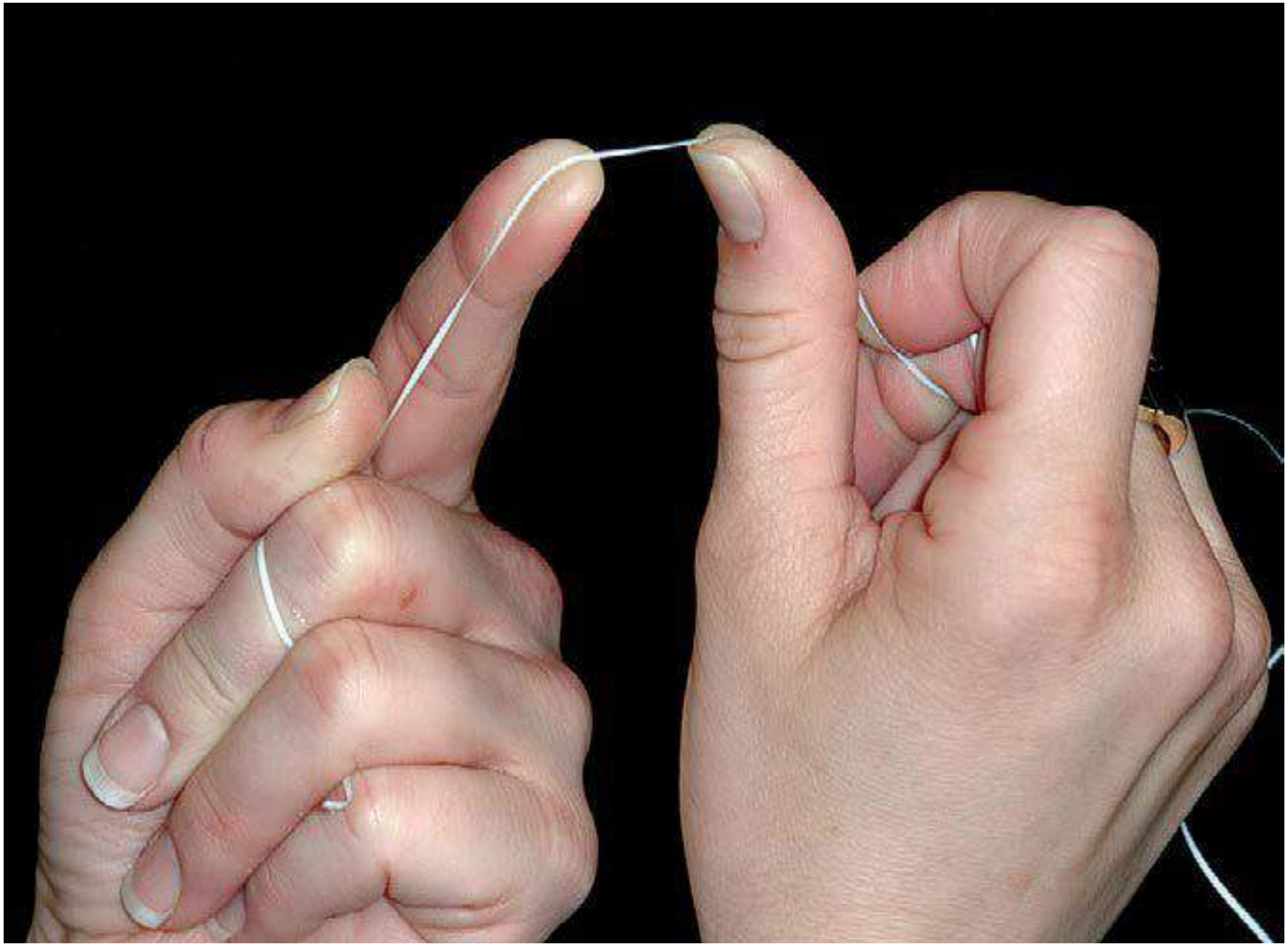
Figure 44-12 Interproximal cleaning devices include wooden tips (**A** and **B**), interproximal brushes (**C** to **F**), and rubber tip stimulators **(G).**

1- Dental Floss

Dental floss is the most widely recommended tool for removing plaque from proximal tooth surfaces. Floss is made from nylon filaments or plastic monofilaments, and can be waxed, unwaxed, thick, thin, and even flavored.

Technique. The floss must contact the proximal surface from line angle to line angle to clean effectively. It must also clean the entire proximal surface, including accessible subginigival areas, not just be slipped apical into the contact area.

Dental floss should be held securely in the fingers or tied in a loop.



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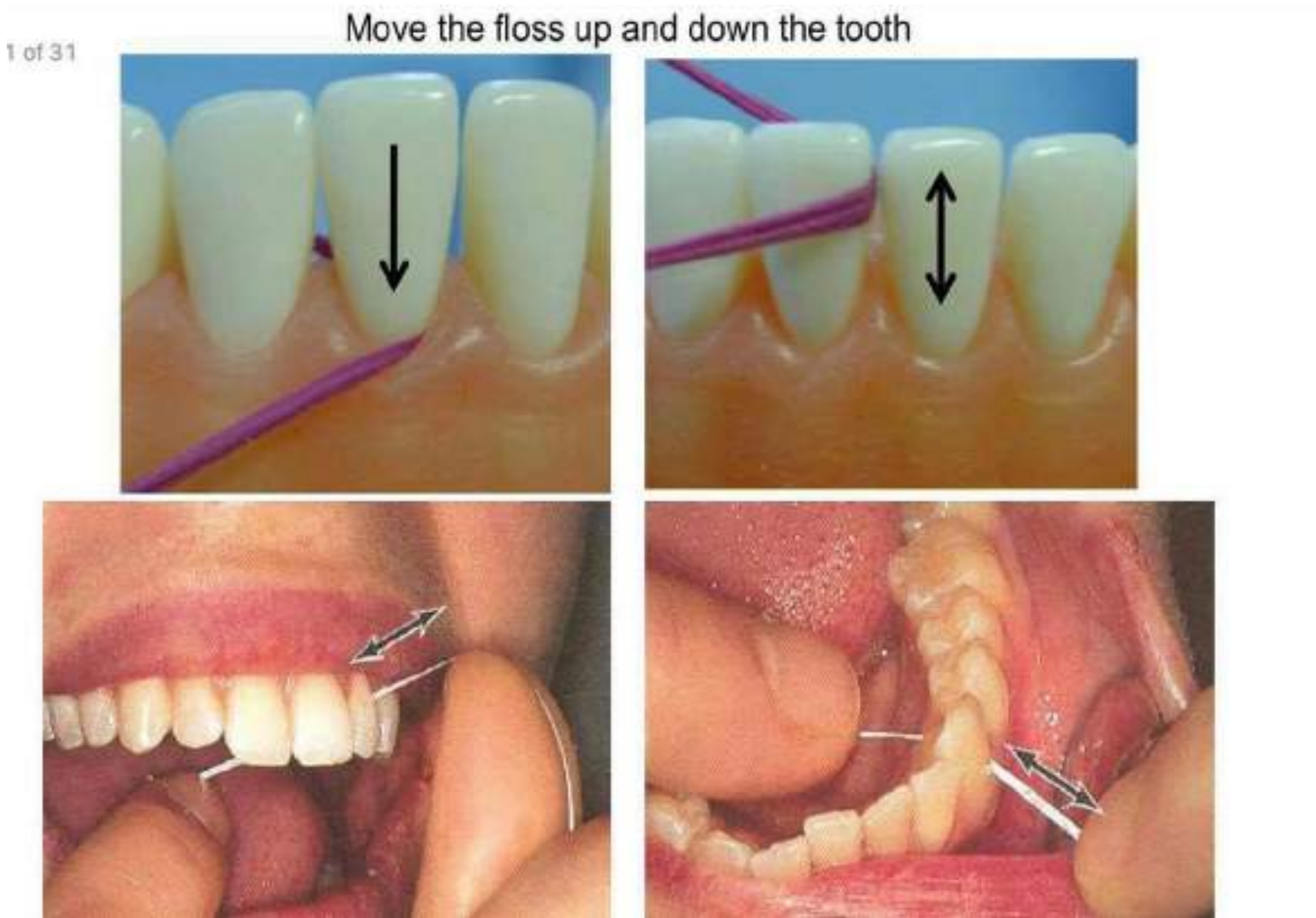
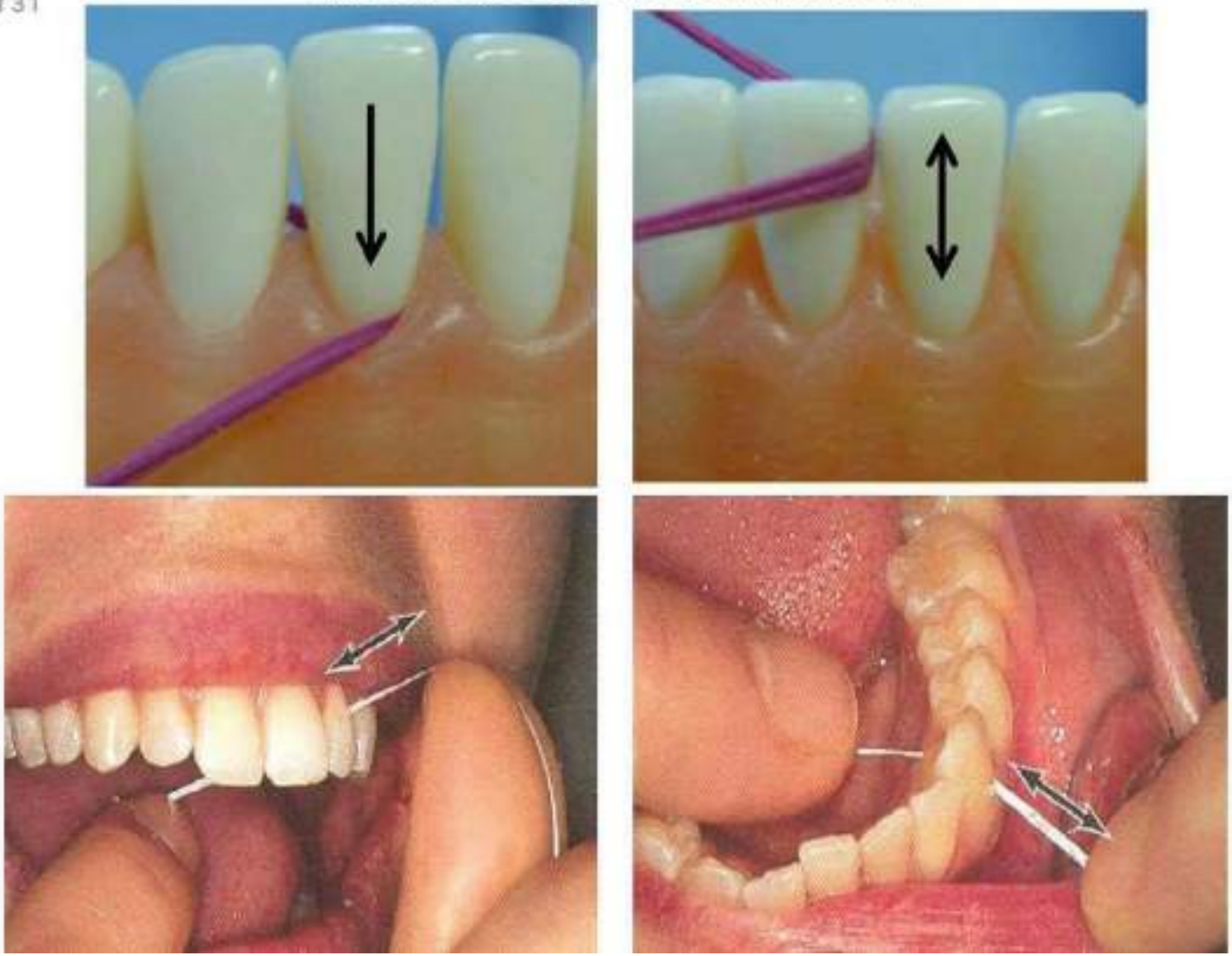


Move the floss up and down the tooth



<u>*Dental tape*</u> is similar to dental floss only wider

Super floss it is marketed for use in patients with crowns, orthodontics appliances and bridge work. It has a stiffened end to thread between the teeth, a spongy portion to brush the teeth and a floss section for a conventional flossing.

2- Interdental brushes

Concave root surfaces and furcations are often present in periodontal patients who have experienced significant attachment loss and recession, and they are not cleaned well with dental floss. Embrasure spaces vary greatly in size and shape. As a general rule, the larger the space, the larger the device used to clean it should be.

Technique.

Interdental brushes of any style are inserted through interproximal spaces and moved back and forth between the teeth with short strokes.

For the most efficient cleaning, the diameter of brush should be slightly larger than the gingival embrasures to be cleaned. This size permits bristles to exert pressure on both proximal tooth surfaces, working their way into concavities on the roots.

Single-tufted brushes provide access to furcation areas, or isolated areas of deep recession, and work well on the lingual surfaces of mandibular molars and premolars. These areas are often missed when using a toothbrush

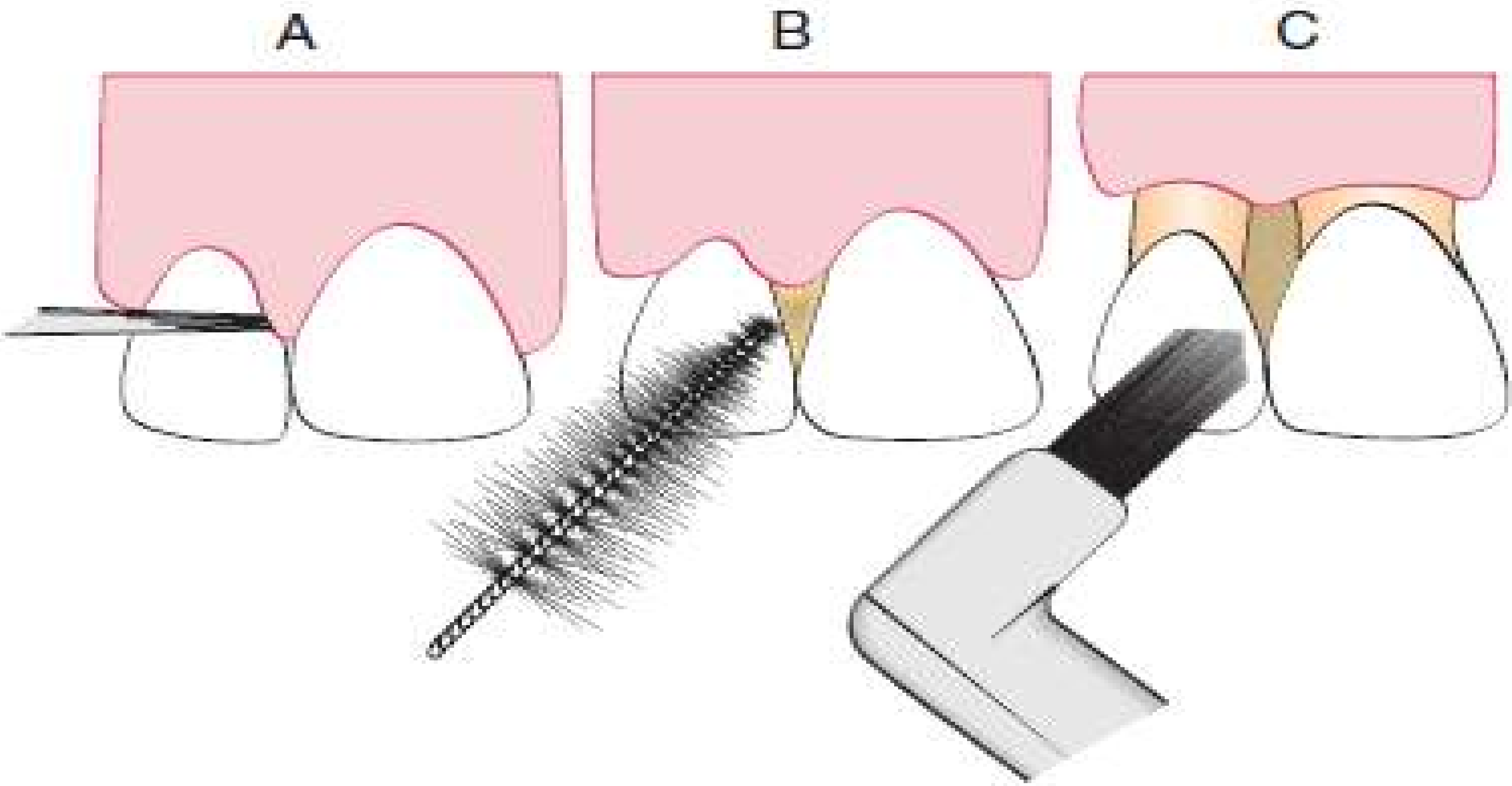


Figure 44-11 Interproximal embrasure spaces vary greatly in patients with periodontal disease. In general, **A,** embrasures with no gingival recession are adequately cleaned using dental floss; **B,** larger spaces with exposed root surfaces require the use of an interproximal brush; and **C,** single-tufted brushes clean efficiently in interproximal spaces with no papillae.

3- Wooden or Rubber Tips

Soft, triangular wooden picks or plastic alternatives are placed in the interdental space with the base of the triangle resting on the gingiva and the sides in contact with the proximal tooth surfaces. The pick is then repeatedly moved in and out of the embrasure to remove plaque. The disadvantage of triangular wooden or plastic tips is that they do not reach well into the posterior areas or on the lingual surfaces and are not shown to remove plaque but can be used to remove food debris.

Wooden toothpick

Triangular wooden tips are also popular with patients. The

tip is inserted between the teeth, with the triangular portion resting on the

gingival papilla. The tip is moved in and out to remove plaque; however, it

is very difficult to use on posterior teeth and from the lingual aspect of all

teeth.

Chemical plaque control (Antiseptices):

Adjunctive to mechanical methods; e.g.: chlorhexidine 0.2% mouth wash.

How should I instruct my patient to use Chlorhexidinemouth wash?

1- Volume used = 10ml.

2- Brush your teeth then use the mouth wash after 30 min.

3- Rinse your mouth with Chlorhexidine for 60 sec.

4- After you spit it, don’t eat or drink for at least 30 min. to let the mouth wash takes its action.

5- Use it twice daily at morning & evening.

6- If you notice any allergic reaction due to its use, stop using it immediately.


KEEPS TEETH
CLEANER & BRIGHTER
NEW Advanced
LISTERINE
with Tartar Protection
ANTISEPTIC
ARCTIC MINT
1.5 LITERS (1 qt 1 pt 2.7 fl oz)
SENSODYNE
سنسوداين
GENTLE
MINT
CORSODYL
MOUTHWASH
GINGIVITIS (GUM DISEASE) TREATMENT
ORIGINAL
CORSODYL
MOUTHWASH
GINGIVITIS (GUM DISEASE) TREATMENT

Periodontal instruments

Diagnostic instrument

-dental mirrior
-probe
-twizeer

Manual scaling instrument

supragingival

-sickle scaler
-cumine
-push scaler

subgingival

Hoe instrument

Root planning instrument

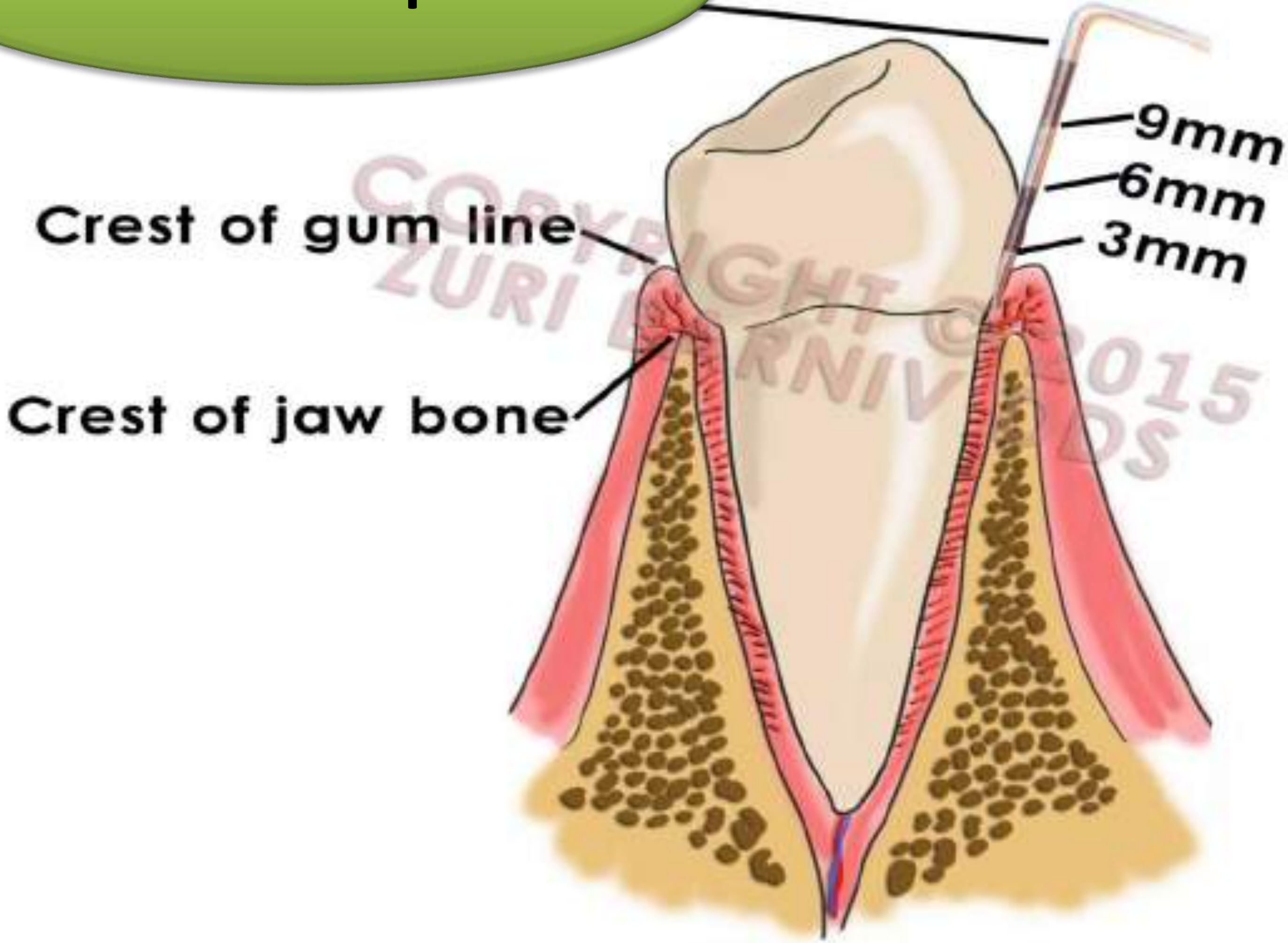
- Area specific curette
- Universal curettes

Polishing instrument

-rubber cups
-bristles brush
-dental tape

NORMAL HEALTHY GUMS AND BONE

Periodontal probe



Periodontal probes

Periodonticsinstruments

Explorers

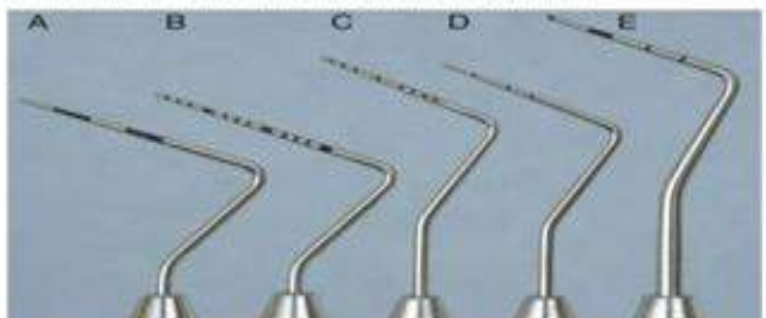
- Use: **1.<u>Locate Sub-gingival Deposits and Caries.</u>**
  2.<u>Check Smoothness of root surface after planning.</u>
  3.<u>Assess Restorative problems.</u>

- Thin, Flexible, Wire-like working end. Taper to sharp point.
- Curved, Right-angled & Area specific.

Periodontal Probes

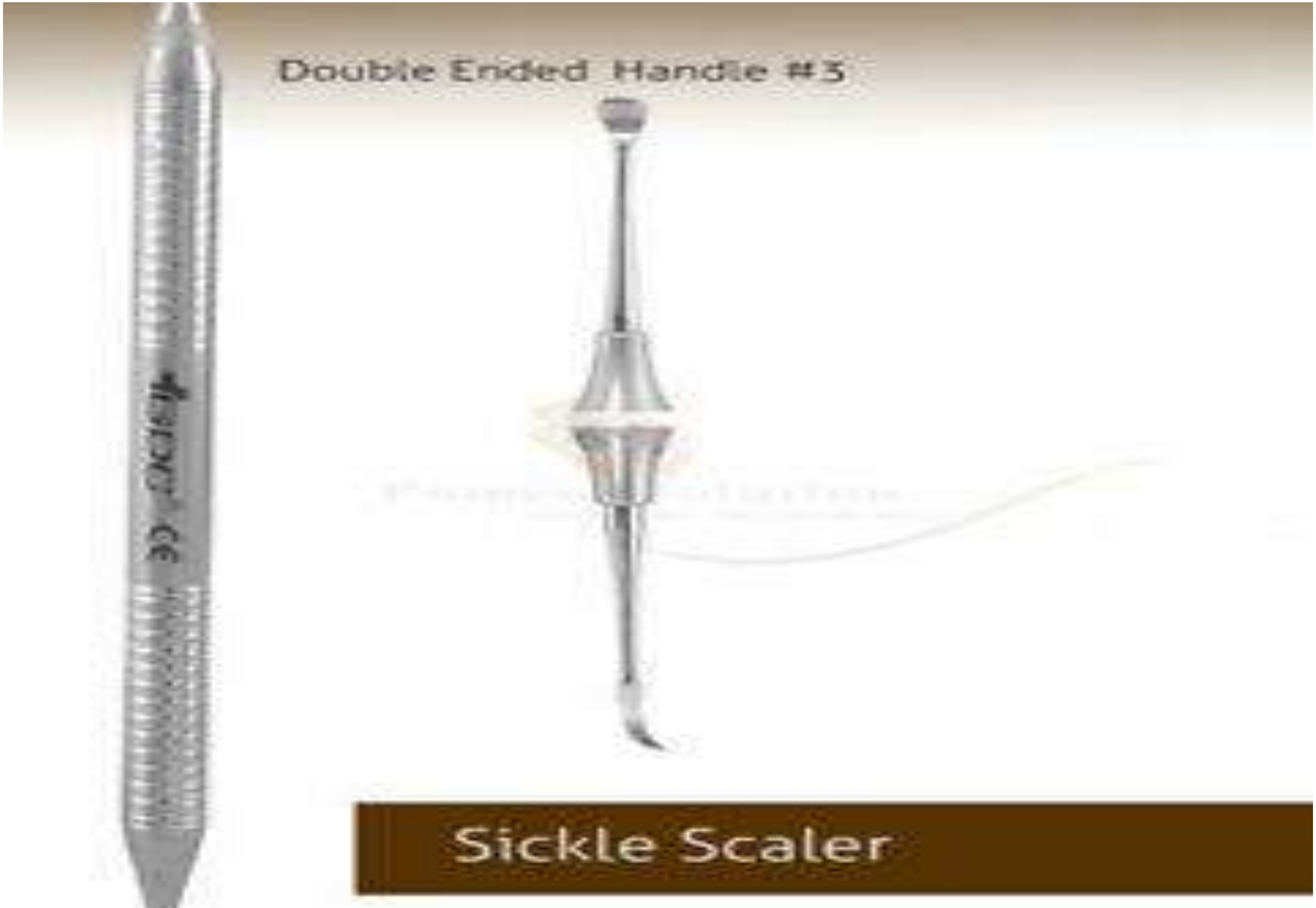
- Use: **1.<u>Locate &Measure depth of pocket and determine it's configuration.</u>**
  2. <u>Assess Loss of Attachment.</u>
  3. <u>Detect Sub-gingival Deposits.</u>

- Tapered with blunt round tip, mm markings for accuracy.
- Ball-end to avoid penetration into junctional epithelium.
- Diameter less than or equal 0.6 mm.
- Probing Force more than 0.25 N traumatize healthy tissue. (25-50g for clinic)
- Ball-end to avoid penetration into junctional epithelium.

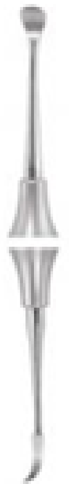


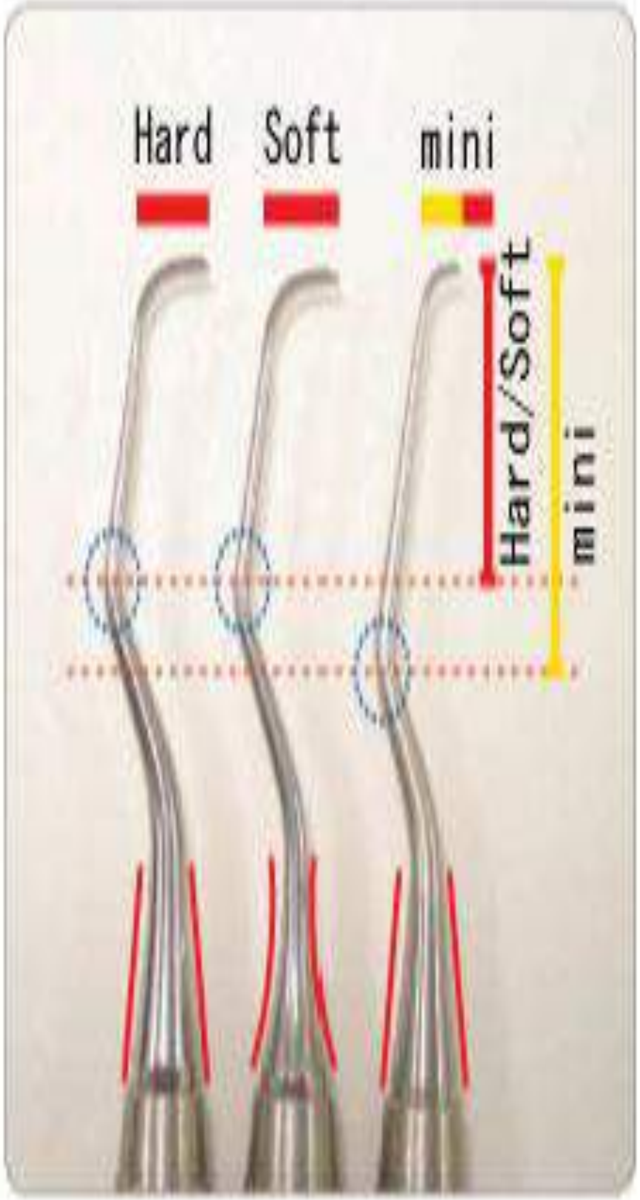
A, Marquis color-coded probe. **B,** UNC-15 probe. **C,** University of Michigan "O" probe, with Williams markings. **D,** Michigan "O" probe with markings at 3, 6, and 8 mm. **E,** World Health Organization (WHO) probe.

Periodontal probe for measure depth of sulcus or pocket


Double Ended Handle #3
Sickle Scaler

Scale scaler for removing of supragingival calculus with pull stroke not used for sugingivally because of caused trauma




Hard
Soft
mini
Hard/Soft
mini


#G11-12
#G15-16

curette

Hoe scaler

HSA12-13

- Anterior Hoe Scaler

For anterior buccal and lingual surfaces.

Rubber cups

Brushes for polishing


SELECT
300
Motor Running
MAX

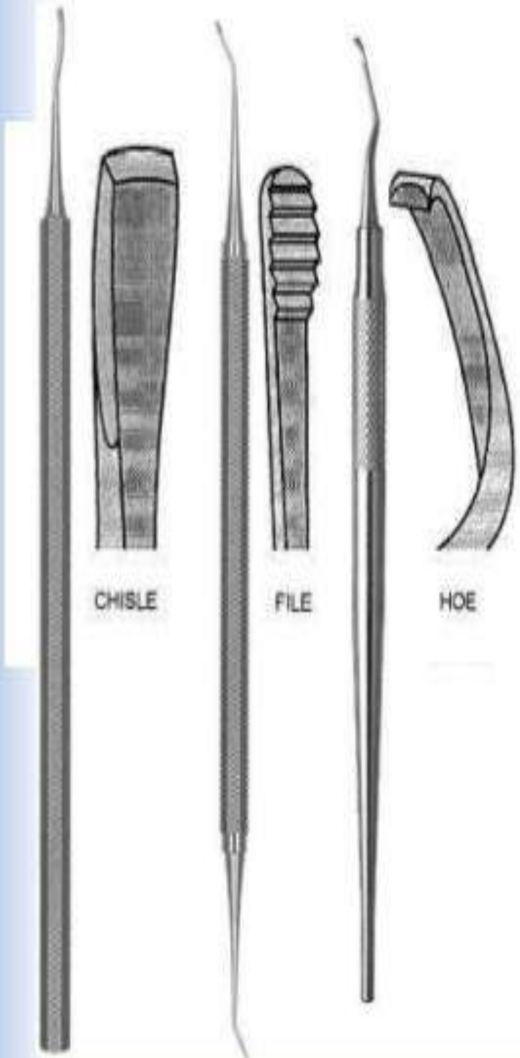

Snap-on
Screw type
Latch type
dentp.en.alibaba.com
Tapered
Flat
Cup-shape

Pumice for polishing

Rubber cups for polishing and smoothening


BEFORE
AFTER

SUBGINGIVAL SCALERS



Chisel scaler

Used in interproximal area
Used in a push motion

File

Used to crush large pieces of calculus deposits.

Hoe

Efficient in removing subgingival calculus
Blade is beveled at 45 degrees
Working end is bent at an angle of 99 degrees to shank.

Ultrasonic scaler

Ultrasonic scaler

THANK YOU

Dental Calculus

م. سها أسود دهش العزاوي

B.D.S, MSc. Periodontology

- The primary cause of gingival inflammation is bacterial plaque, other predisposing factors including calculus, faulty restoration, orthodontic therapy, use of tobacco, and others.

- **Calculus** consists of mineralized bacterial plaque that forms on the surface of natural teeth and dental prosthesis.

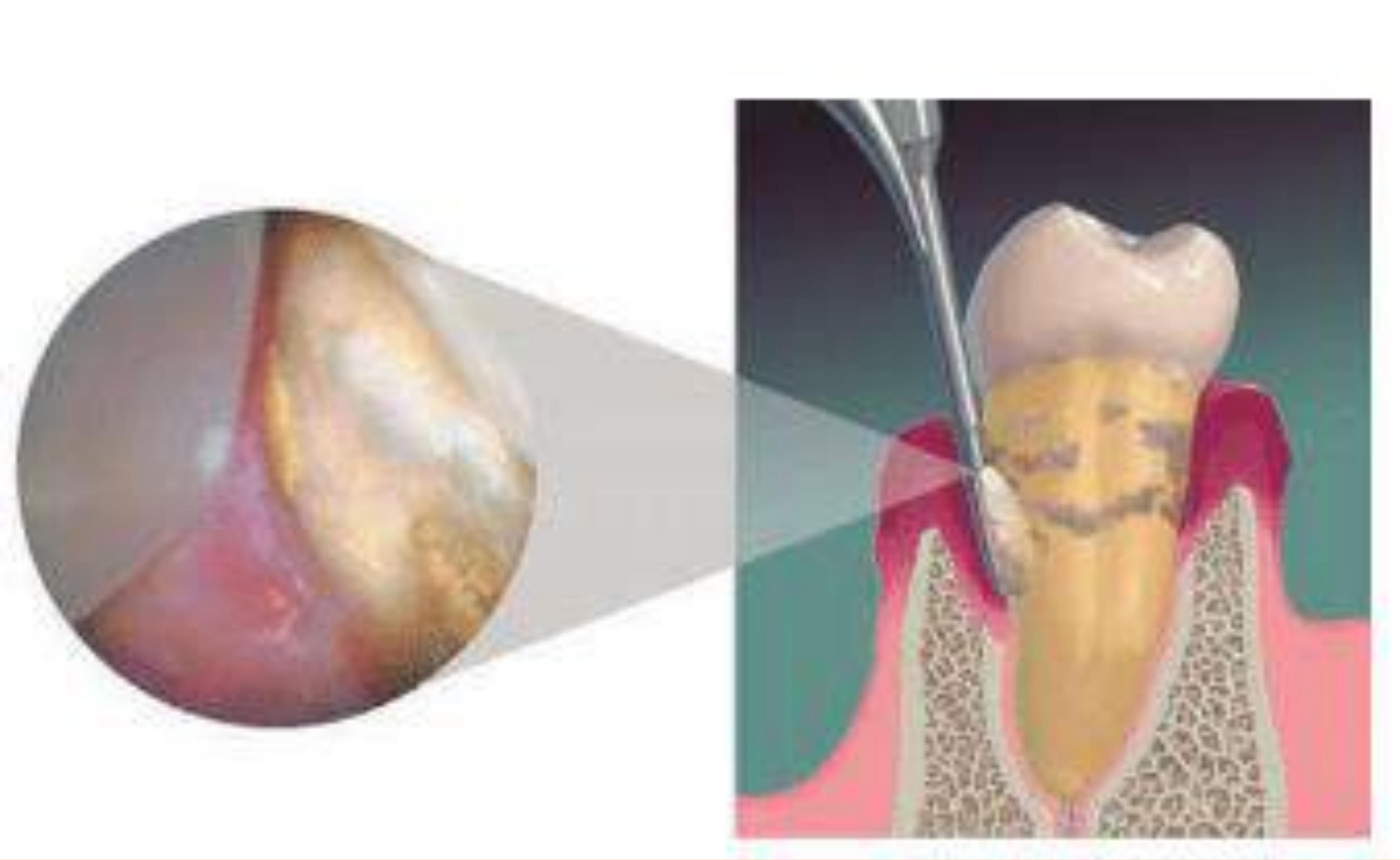
Supra and sub-gingival calculus

- **Supragingival calculus** is located coronal to the gingival margin and therefore visible in the oral cavity,
- usually white to yellowish in colour, hard with claylike consistency and easily detected from the tooth surface, its colour is influenced by contact with such substances as tobacco and food pigments.
- It may localize on a single tooth or group of teeth or it may be

generalized throughout the mouth.

- The two most common locations for supragingival calculus to develop are the buccal surface of maxillary molars and the lingual surfaces of mandibular anterior teeth, saliva from the parotid gland flows over the facial surfaces of the upper molars via the parotid duct, while the submandibular and the sublingual glands empty onto lingual surfaces of lower incisors via the submandibular and lingual ducts respectively.

- **Subgingival calculus** is located below the crest of the marginal gingiva and therefore not visible on routine clinical examination,
- the location and extent of the subgingival calculus maybe evaluated by careful tactile perception with a delicate dental instruments such as a dental explorer,
- subgingival calculus is typically hard and dense, frequently appears dark brown or greenish black in colour and firmly attached to the tooth surface.
- When the gingival tissue recede, subgingival calculus becomes exposed and its therefore classified as supragingival.
- Both supra and sub gingival calculus maybe seen by radiograph.



Subgingival calculus detection

Composition

- **Inorganic content** : supragingival calculus consist of inorganic (70% to 90%) and organic components,
- The major inorganic proportions of calculus have been reported as approximately 76% calcium phosphate $Ca3(PO4)$; 3% calcium carbonate $CaCO3$; traces of magnesium phosphate $Mg3(PO4)$ and other metals .
- At least two thirds of the inorganic component is crystalline in structure,
- The four main crystal forms and their approximate percentage are as follows:
- hydroxyapatite 58%, magnesium white-lockite 21%, octacalcium phosphate

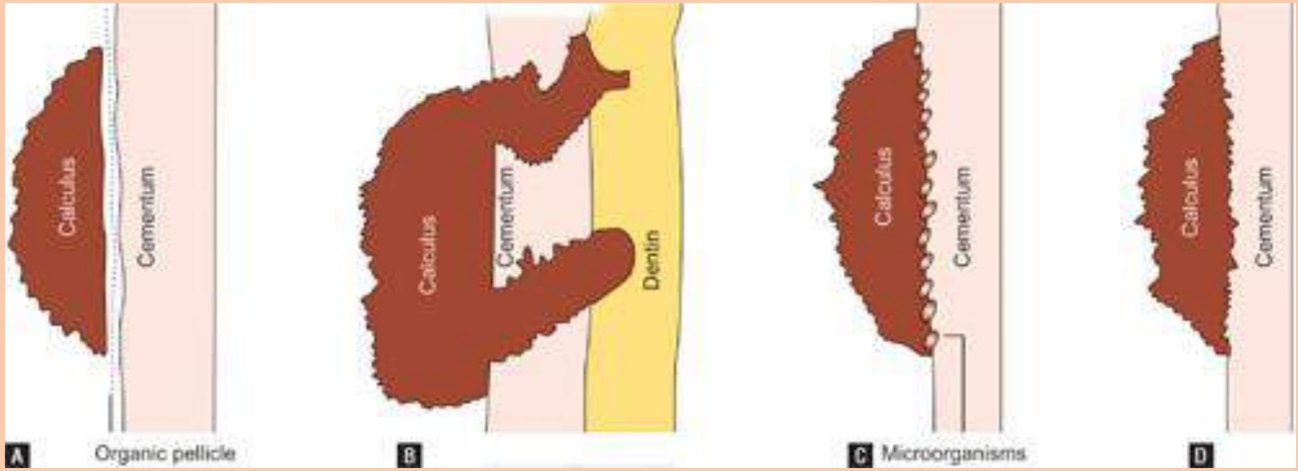
12% and brushite 9%.

- Generally two or more crystal forms are typically found in the sample of calculus, **hydroxyapatite and octacalcium phosphate** are detected most frequently in the supragingival calculus and constitute the bulk of the specimen.
- **Brushite** is more common in the mandibular anterior region and
- **magnesium white-lockite** is in the posterior areas,
- The composition of subgingival calculus is similar to that of supragingival calculus with some differences, it has the same hydroxyapatite content, more magnesium white-lockit, and less brushite and octacalcium phosphate

- Organic content: the organic component of calculus consists of a mixture of protein-polysaccharide complexes, desquamated epithelial cells. Leukocytes and various types of microorganisms.
- Salivary proteins present in supragingival calculus are not found subgingivally.

Attachment of calculus to the tooth

- 1. By means of an organic pellicle.
- 2. Mechanical locking into surface irregularities such as carious lesions and resorption lacuna.
- 3. penetration by bacterial calculus into cementum and may appear morphologically similar to cementum and thus termed calculocementum.
- 4. Close adaptation of the undersurface of calculus to depressions or gently sloping surfaces of the unaltered cementum surface.



Formation

- Calculus is dental plaque that has undergone mineralization; the soft plaque is hardened by precipitation of mineral salts which is usually started between first and fourteenth days of plaque formation.
- Calcification started as soon as 4-8 hours, calcifying plaque may become 50% mineralized in 2 days, 60%-90% mineralized in 12 days. However, the formation of dental calculus with the mature crystalline composition of old calculus may require months to years.
- Saliva is the source of mineralization for supra gingival calculus whereas the serum transudate (Gingival crevicular fluid) is source of mineralization of sub gingival calculus.
- Microorganisms are not always essential in calculus formation because calculus occurs readily in germ free rodents,

- The initiation of calcification and rate of accumulation vary among teeth in same individual, so person may be heavy, moderate or slight calculus former.
- Calculus formation continues until it reaches maximum after which it reduced in amount due to mechanical wear from food and the cheeks, lip and tongue, also the use of anti-calculus (anti tarter) dentifrices reduce both quality and quantity of calculus.

Theories regarding the mineralization of Calculus

- The theoretical mechanisms by which plaque becomes mineralized can be stratified into two categories:

1. Mineral precipitation results from a local rise in the degree of saturation of calcium and phosphate ions, which may be brought about in the following several ways:

- A rise in the pH of the saliva causes precipitation of calcium phosphate salts by lowering the precipitation constant. The pH may be elevated by the loss of carbon dioxide and the formation of ammonia by dental plaque bacteria or by protein degradation.

- Colloidal proteins in saliva bind calcium and phosphate ions and maintain a supersaturated solution with respect to calcium phosphate salts.
- Phosphatase liberated from dental plaque, desquamated epithelial cells, or bacteria precipitates calcium phosphate by hydrolyzing organic phosphates in saliva, thus increasing the concentration of free phosphate ions.

- **2. Seeding agents induce small foci of calcification that enlarge and coalesce to form a calcified mass.** This concept has been referred to as the **epitactic concept** or more appropriately, hetero- geneous nucleation. The seeding agents in calculus formation are not known, but it is suspected that the intercellular matrix of plaque plays an active role. The carbohydrate-protein complexes may initiate calcification by removing calcium from the saliva (chelation) and binding with it to form nuclei that induce subsequent deposition of minerals.

Etiologic Significance

- The non-mineralized plaque on the calculus surface is the principle irritant for initiating gingivitis.
- The underlying calcified portion is a significant contributing factor since it provides a fixed nidus for the continued accumulation of plaque and remains it close to gingiva.
- calculus plays an important role in maintaining periodontal diseases by keeping plaque in close contact with the gingival tissue and creating area where plaque removal is impossible unless we remove calculus.
- it is a secondary etiologic factor for periodontitis and it is the most prominent plaque retentive factor which has to be removed as a basis for adequate periodontal therapy and prophylactic activities..Removal of supra and sub gingival plaque and calculus constitute the cornerstone of periodontal therapy.

OTHER PREDISPOSING FACTORS

- **Iatrogenic Factors:**

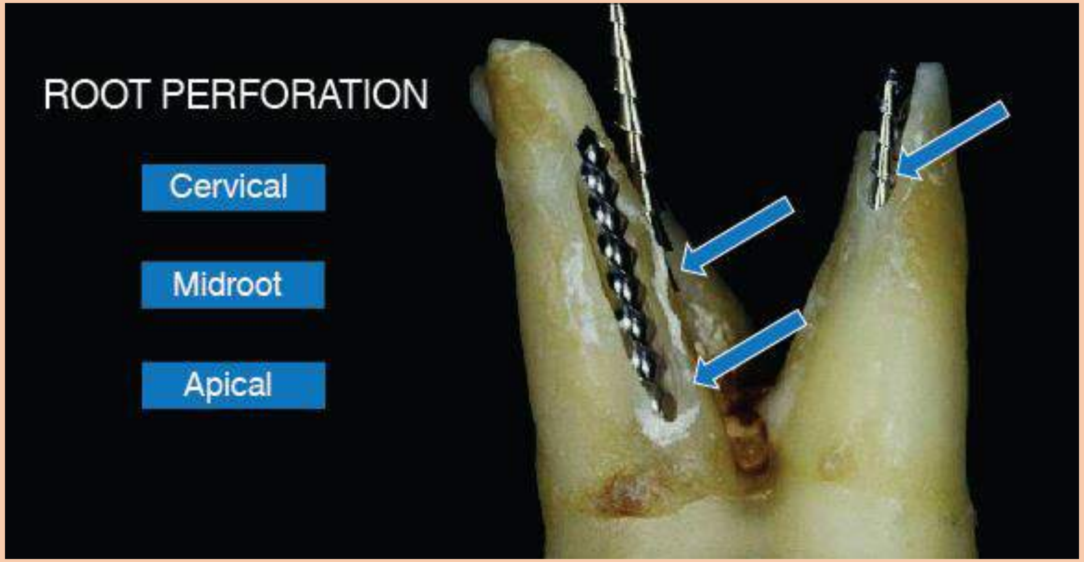
Inadequate dental procedures that contribute to the deterioration of the periodontal tissues are referred to as iatrogenic factors.

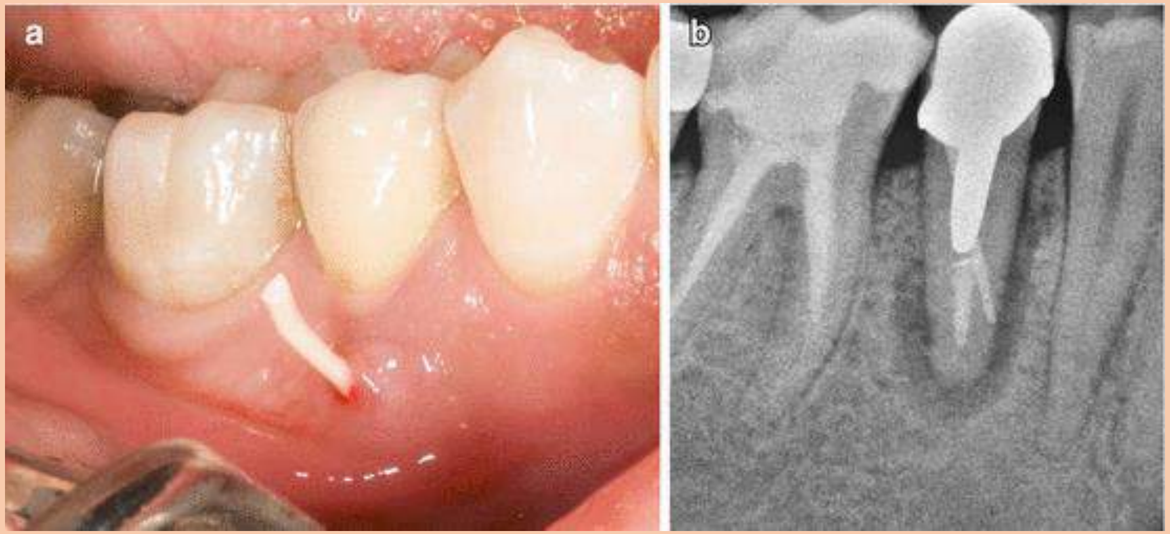
Deficiencies in the quality of dental restorations or prostheses are contributing factors to gingival inflammation and periodontal destruction.

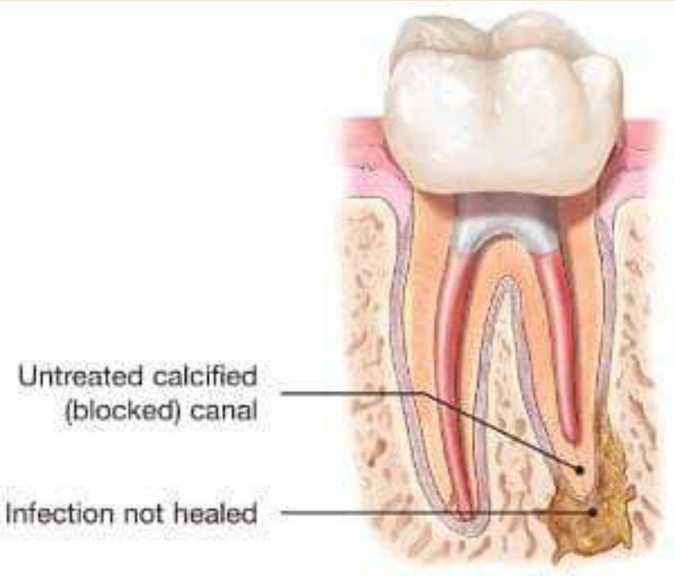
Iatrogenic endodontic complications that can adversely affect the periodontium include root perforations, vertical root fractures, and endodontic failures.

The location of the gingival margin for the restoration, the space between the margin of the restoration and the unprepared tooth, the contour of restorations, the occlusion, materials used in the restoration, the restorative procedure itself, and the design of the removable partial denture. These characteristics are related to the etiology of periodontal disease.

endodontic complications





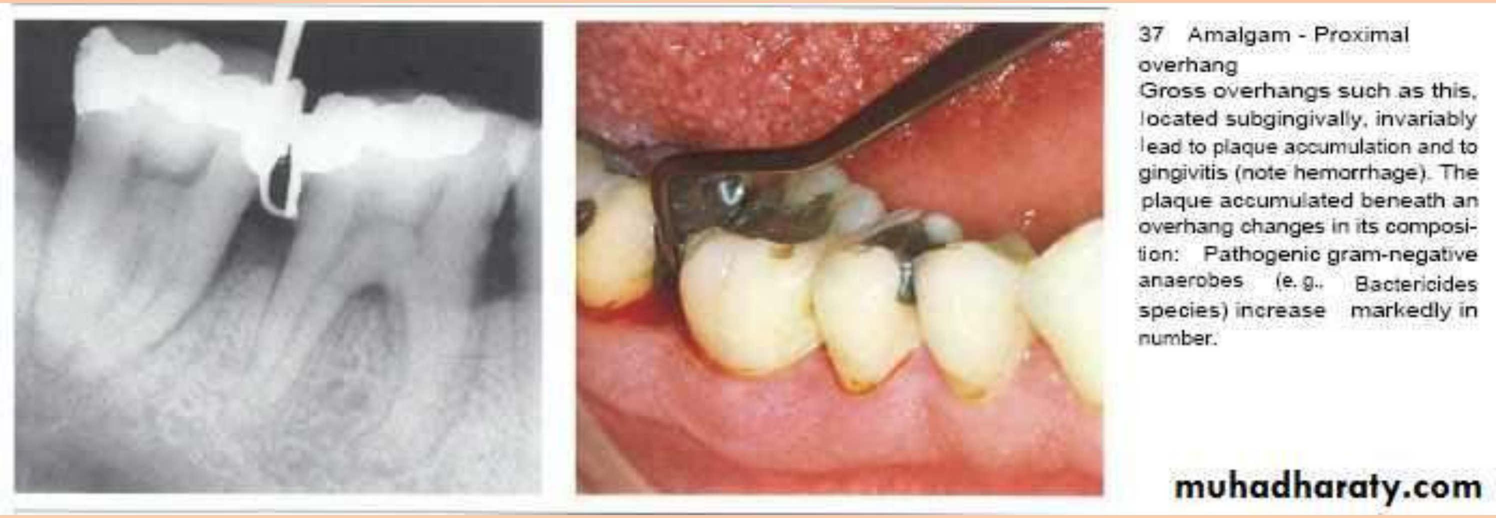


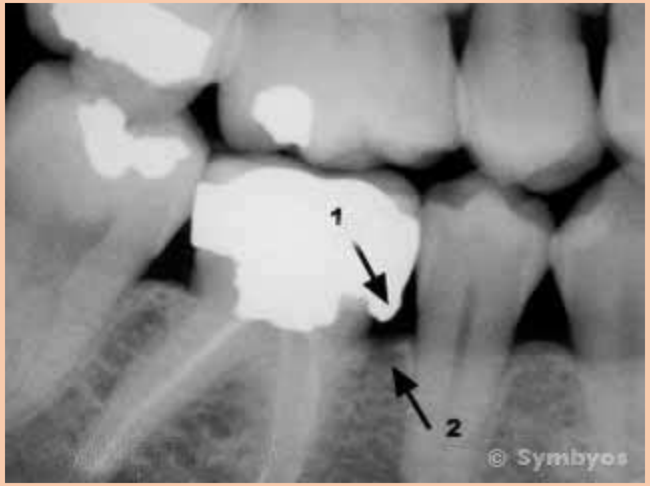
- **Margins of Restorations**

Overhanging margins of dental restorations contribute to the development of periodontal disease by (1) changing the ecologic balance of the gingival sulcus to an area that favors the growth of disease-associated organisms (pre-dominantly gram-negative anaerobic species) at the expense of the health-associated organisms (predominately gram-positive facultative species)and (2) inhibiting the patient’s access to remove accumulated plaque.

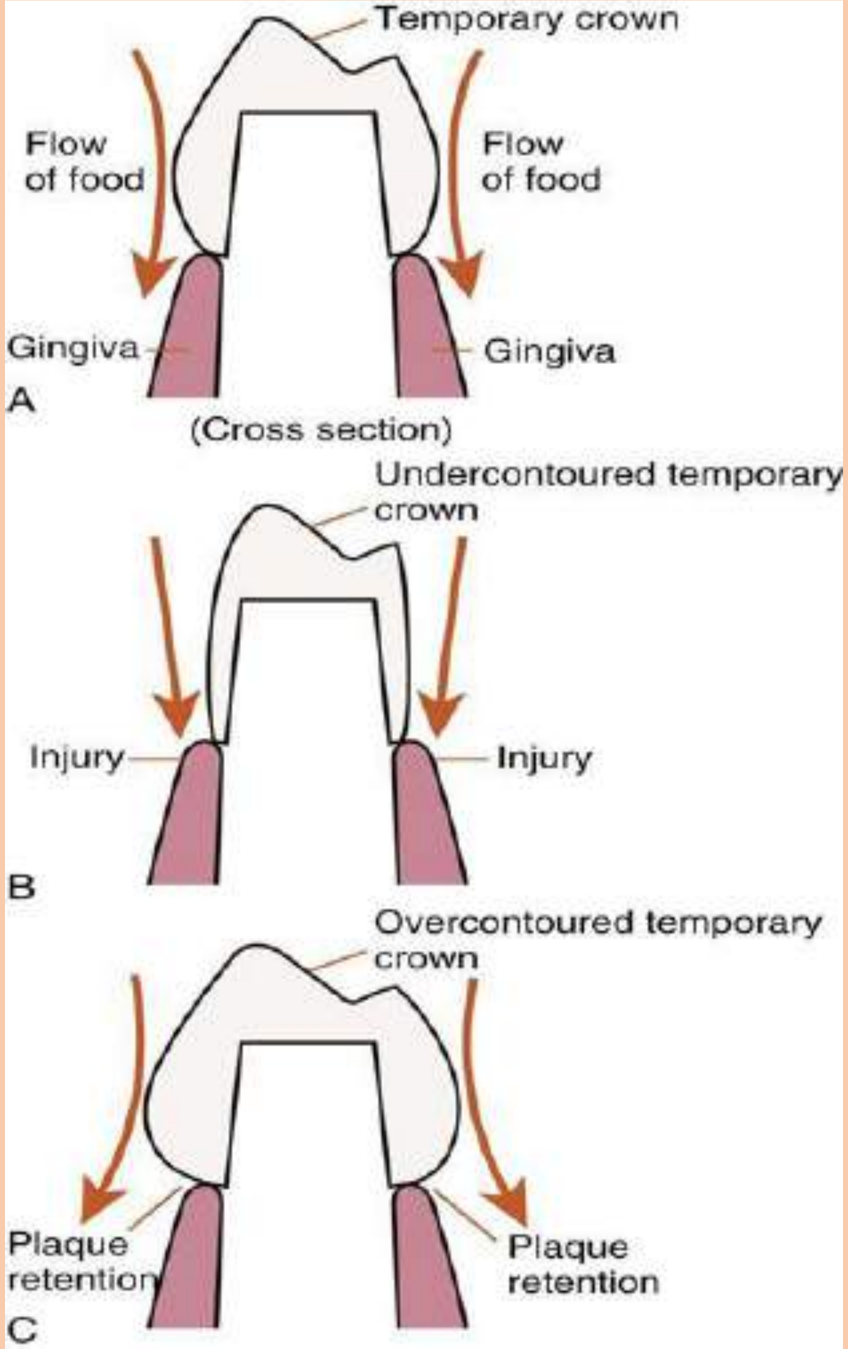
Subgingival margins are associated with large amounts of plaque, more severe gingivitis, and deeper pockets. Margins placed at the level of the gingival crest induce less severe inflammation, whereas supragingival margins are associated with a degree of periodontal health.

Overhanging margins





- **Contours/Open Contacts**
- Over contoured crowns and restorations tend to accumulate plaque and possibly prevent the self- cleaning mechanisms of the adjacent cheek, lips, and tongue. Restorations that fail to reestablish adequate interproximal embrasure spaces are associated with papillary inflammation.



Thank you

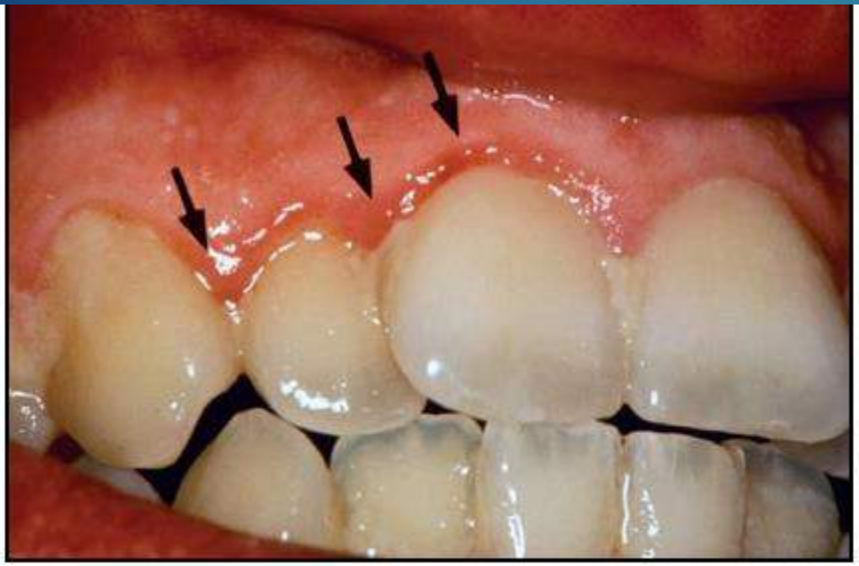
DENTAL PLAQUE BIOFILM

م. سها أسود دهش العزاوي

B.D.S, MSC. PERIODONTOLOGY

- Oral biofilms are functionally and structurally organized polymicrobial communities that are embedded in an extracellular matrix of exopolymers on mucosal and dental surfaces.
- Dental plaque is defined clinically as a structured resilient yellow-grayish biofilm that adheres firmly to the intraoral hard surfaces, including removable and fixed restorations. The tough extracellular matrix makes it impossible to remove plaque by rinsing or the use of sprays. Plaque can thus be differentiated from other deposits that may be found on the tooth surface such as materia alba and calculus.
- Microorganisms represent the main component of dental plaque, with approximately 10^{11} bacteria contained in one gram of plaque (wet weight)

- Using highly sensitive molecular techniques for microbial identification, it has been estimated that more than 500 distinct microbial phenotypes can be present as natural inhabitants of dental plaque. Any individual may harbor 150 or more different species. Non- bacterial microorganisms that are found in plaque include archaea, yeasts, protozoa, and viruses.



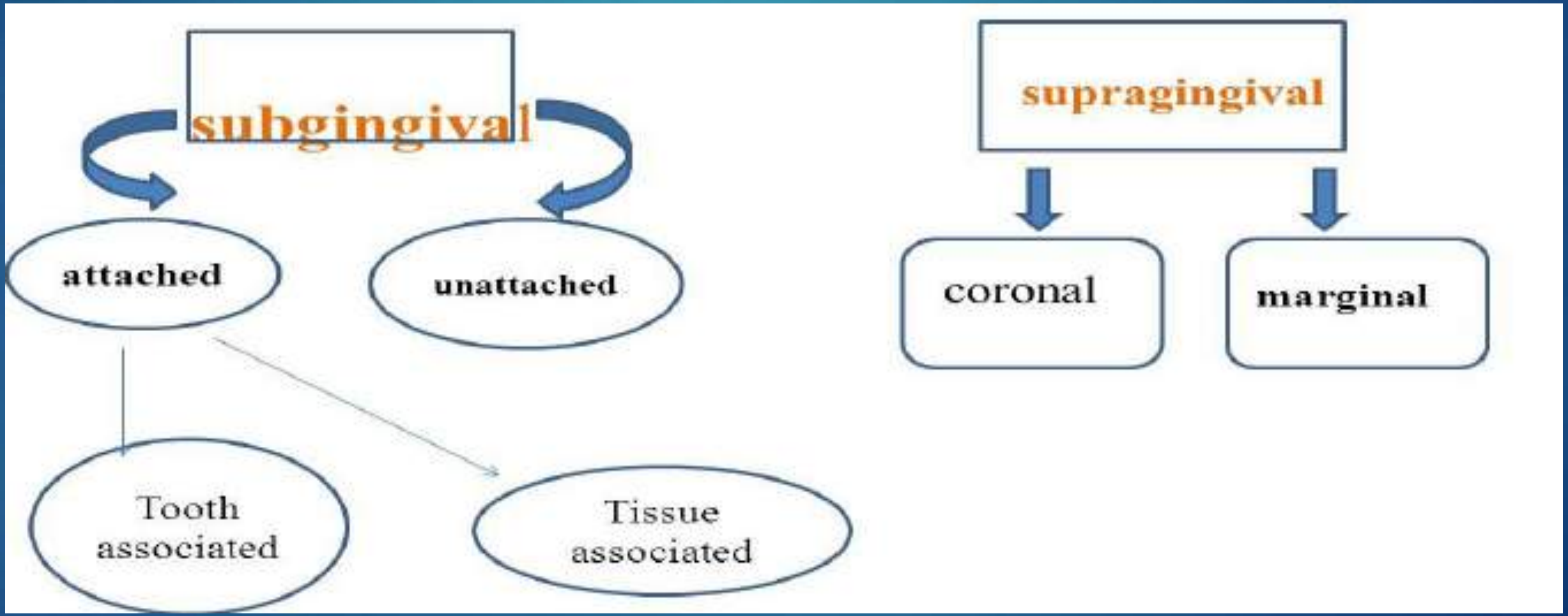
Clinical picture of 10-day-old supragingival plaque. Balck rows indicate early signs of gingival inflammation.

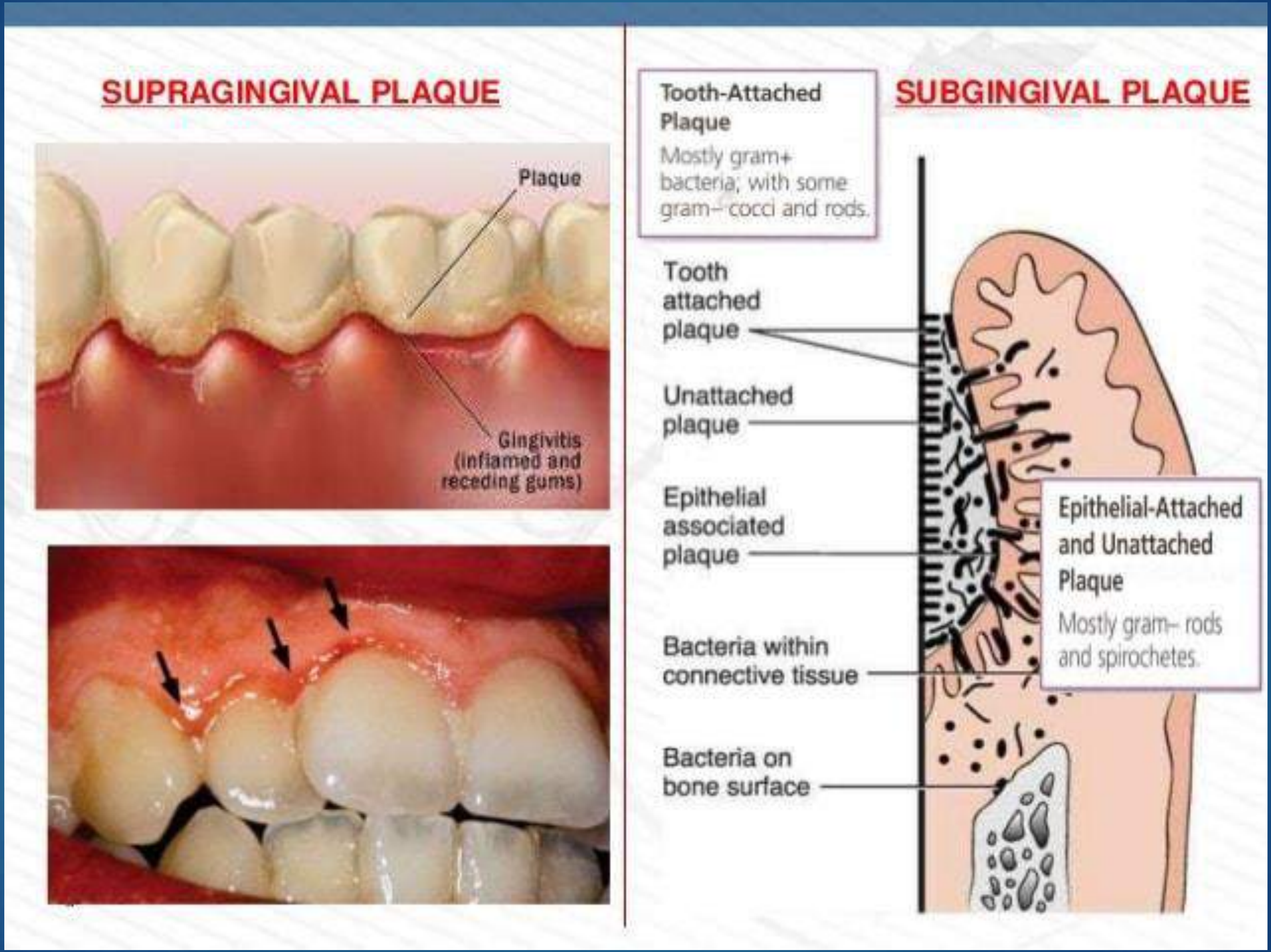
Supragingival calculus is depicted on the buccal surface of maxillary molars adjacent to the orifice for the parotid duct.

	Materia alba	Dental plaque	Claculus
1	White cheese-like accumulations	Resilient clear to yellow grayish	Hard deposits formed by mineralization of dental plaque
2	A soft accumulation of salivary proteins	Primarily composed of bacteria in a matrix of salivary proteins	
3	Lack of organized structure (not complex) as dental plaque)	Considered as a biofilm	Generally covered by a layer of un-mineralized dental plaque
4	Easily displaced by a water spray	Removed only by mechanical rinsing (tooth brushing)	

Classification of Plaque

Based on its position on the tooth surface toward the gingival margin



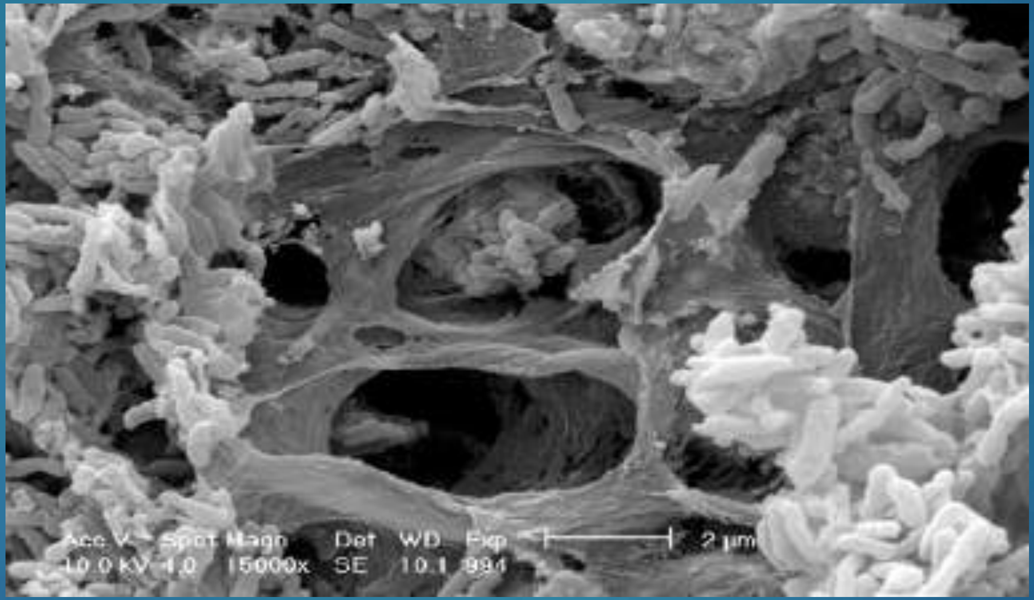

SUPRAGINGIVAL PLAQUE
Plaque
Gingivitis (inflamed and receding gums)
SUBGINGIVAL PLAQUE
Tooth-Attached Plaque
Mostly gram+ bacteria; with some gram– cocci and rods.
Tooth attached plaque
Unattached plaque
Epithelial associated plaque
Bacteria within connective tissue
Bacteria on bone surface
Epithelial-Attached and Unattached Plaque
Mostly gram– rods and spirochetes.

Supragingival plaque typically demonstrates a stratified organization of a multilayered accumulation of bacterial morphotypes. Gram-positive cocci and short rods predominat at the tooth surface, whereas gram-negative rods and filaments, as well as spirochetes, predominate in the outer surface of the mature plaque mass

In general, the subgingival microbiota differs in composition from the supragingival plaque, primarily because of the local availability of blood products and a low reduction-oxidation (redox) potential, which characterizes the anaerobic environment.

- Both morphologic and microbiologic studies of subgingival plaque reveal distinctions between the tooth-associated and soft tissue-associated regions of subgingival plaque.
- The tooth-associated cervical plaque, adhering to the root cementum, does not markedly differ from that observed in gingivitis. At this location, filamentous microorganisms dominate, but cocci and rods also occur. This plaque is dominated by gram positive rods and cocci, including *S. mitis, S. sanguinis, Actinomyces oris*. However, in the deeper parts of the pocket, the filamentous organisms become fewer in numbers, and in the apical portion they seem to be virtually absent. Instead, the microbiota is dominated by smaller organisms
- The apical border of the plaque mass is separated from the junctional epithelium by a layer of host leukocytes

- The layers of microorganisms facing the soft tissue lack a definite intermicrobial matrix and contain primarily gram-negative rods and cocci as well as large numbers of filaments, flagellated rods, and spirochets. Host tissue cells (e.g., white blood cells and epithelial cells) may also be found in this region. Bacteria are also found within the host tissues, such as in the soft tissues and within epithelial cells, as well as in the dentinal tubules.

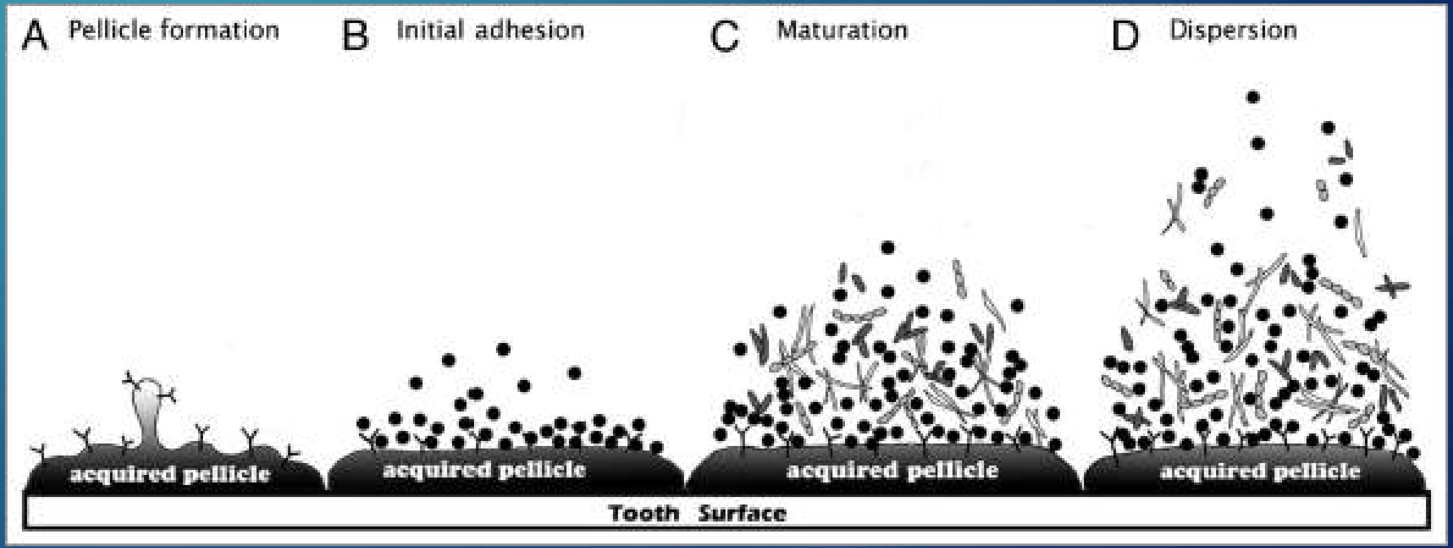


- The composition of the subgingival plaque depends on pocket depth; the apical part is more dominated by spirochetes, cocci and rods, whereas in the coronal part more filaments are observed.

- The site specificity of plaque is significantly associated with diseases of the periodontium.
- Marginal plaque, for example, is of prime importance in the initiation and development of gingivitis.
- Supragingival plaque and tooth-associated subgingival plaque are critical in calculus formation and root caries; whereas
- Tissue associated subgingival plaque is important in the tissue destruction that characterizes different forms of periodontitis.

Accumulation of a Dental Plaque Biofilm

- The process of plaque formation can be divided into several phases:
- 1- The formation of the pellicle on the tooth surface.
- 2- Initial adhesion/attachment of bacteria.
- 3- Colonization/plaque maturation.



Formation of the Pellicle

- It is translucent, homogenous, thin film covering all surfaces in the oral cavity, formed by adsorption of proteins on tooth surface.

- The pellicle on tooth surface consists of more than 180 peptides, proteins, and glycoproteins, and other molecules that can function as adhesion sites (receptors) for bacteria.
- Salivary pellicle can be detected on clean enamel surfaces within 1 minute.
- Bacteria that adhere to tooth surfaces do not contact the enamel directly but interact with the acquired enamel pellicle.

SIGNIFICANCE OF PELLICLE:

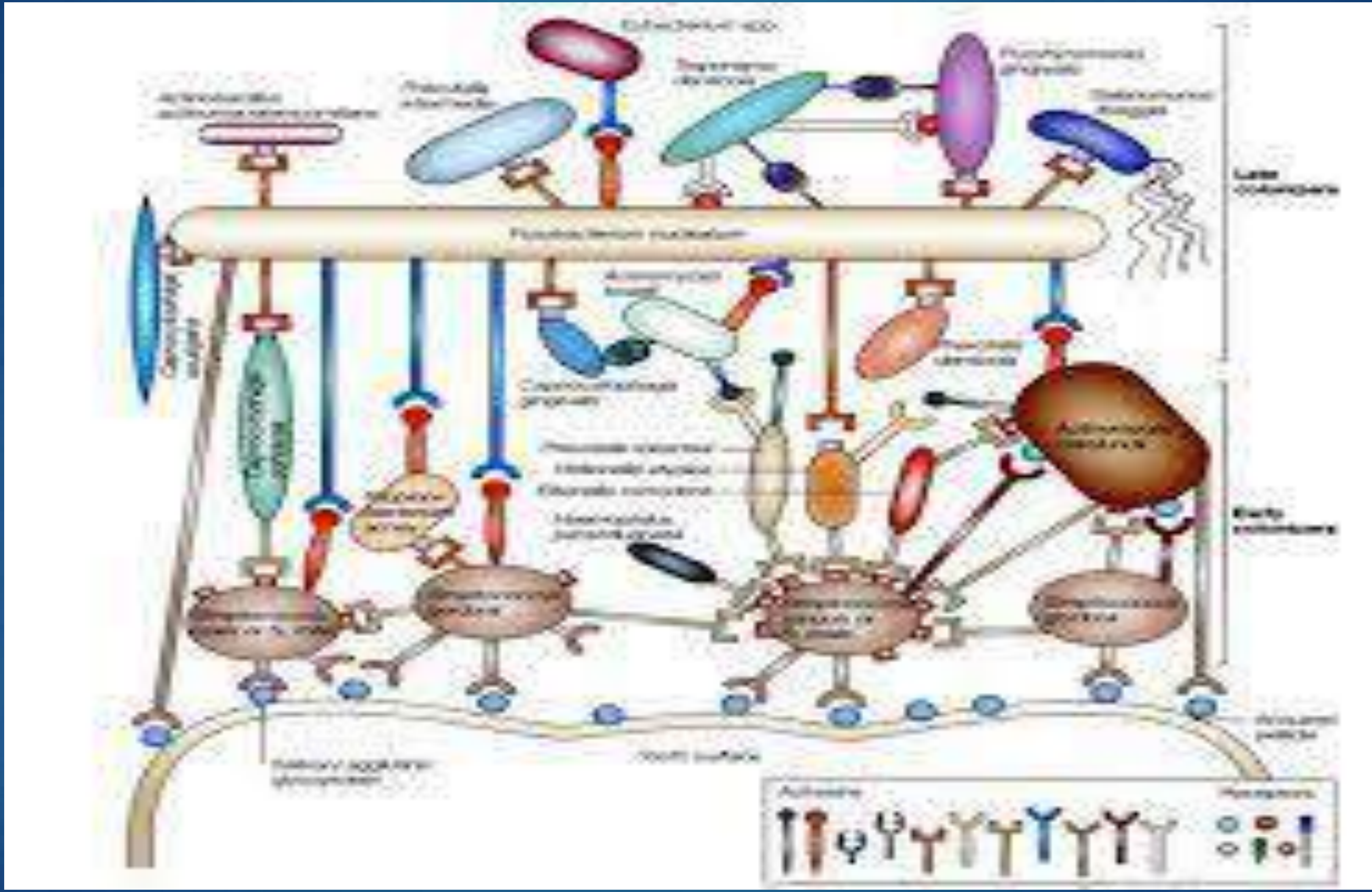
- PROTECTIVE: provide barrier against acids thus may reduce dental caries attack.
- LUBRICATION: keep surface moist prevent drying.
- NIDUS FOR BACTERIA: Plaque formation by adherence of microorganisms.
- ATTACHEMENT OF CALCULUS: A mode of calculus attachment in acquired pellicle.

Initial Adhesion/Attachment of Bacteria.

- The initial steps of transport and interaction with the surface are essentially nonspecific (i.e., they are the same for all bacteria).
- Specific interactions between microbial cell surface "adhesin" molecules and receptors in the salivary pellicle that determines whether a bacterial cell will remain associated with the surface.
- small proportion of oral bacteria possess adhesins that interact with receptors in the host pellicle, and these organisms are generally the most abundant bacteria in biofilms on tooth enamel shortly after cleaning. These species are considered the "primary colonizers" of tooth surfaces.
- The primary colonizers provide new binding sites for adhesion by other oral bacteria.

Colonization and Plaque Maturation.

- The primary colonizing bacteria adhered to the tooth surface provide a new receptors for attachment by other bacteria in a process known as "coadhesion."
- Together with growth of adherent microorganisms, coadhesion leads to the development of micro colonies and eventually to a mature biofilm.
- Different species or even different strains of a single species have distinct sets of coaggregation partners. Fusobacteria coaggregate with all other human oral bacteria while Veillonella spp., Capnocytophaga spp. and Prevotella spp. bind to streptococci and/or actinomyces. Each newly cell becomes itself a new surface and therefore, may act as a coaggregation bridge to the next potentially accreting cell type that passes by.



- Secondary colonizers ,such as *Prevotella intermedia*, *Prevotella loescheii*, *Capnocytophaga spp.*, *F. nucleatum*, and *P. gingivalis* do not initially colonize clean tooth surfaces but adhere to bacteria already in the plaque mass.

COMPOSITION OF PLAQUE:

COMPOSED mainly of:

- Microorganisms ,
- Intermicrobial matrix

(a) Bacterial portion:

- 70 to 80 % of total solid plaque volume.
- 1 g (WET WT)contains approx. 10^{11} bacteria.

(B)Intermicrobial Matrix

- the material present between the bacteria in the dental plaque is called the intermicrobial matrix.
- accounts for approx 25% of plaque volume.

Three sources may contribute to the intermicrobial matrix:

- The plaque microorganisms
- The saliva
- Gingival exudates

Factors Affecting Supragingival Dental dental plaque formation

- During the first 24 hours starting from a clean tooth surface, plaque growth is nearly undetectable clinically.
- after 4 days, on average 30% of the total coronal tooth area will be covered with plaque.
- the microbial composition of the dental plaque will change with a shift from the early aerobic environment characterized by gram-positive facultative species to a highly oxygen-deprived environment in which gram negative anaerobic microorganisms predominate.

Topography of Supragingival Plaque

- initial growth along the gingival margin and from the interdental space. Later, a further extension in the coronal direction.
- Plaque formation can also start from grooves, cracks, or pits.

Irregular plaque growth patterns follow tooth surface irregularities

Surface Micro roughness.

- Rough intraoral surfaces (e.g. crown margins, implant abutments, and denture bases) accumulate and retain more plaque and calculus in terms of thickness, area, and colony-forming units. Ample plaque also reveals an increased maturity/pathogenicity of its bacterial components, characterized by an increased proportion of motile organisms and spirochetes and/or a denser packing of them. Smoothing an intraoral surface decreases the rate of plaque formation.

Individual Variables Influencing Plaque Formation.

- The rate of plaque formation differs significantly between subjects,
- A distinction is often made between “heavy” (fast) and “light” (slow) plaque formers.

Variation within the Dentition.

Early plaque formation occurs faster:

- in the lower jaw (when compared to the upper jaw);
- in molar areas; on the buccal tooth surfaces when compared to palatal sites (especially in the upper jaw); and
- in the interdental regions when compared to the buccal or lingual surfaces.

Impact of Gingival Inflammation and Saliva

- Several studies clearly indicate that plaque formation is more rapid on tooth surfaces facing inflamed gingival margins than on those adjacent to healthy gingival. These studies suggest that the increase in crevicular fluid production enhances plaque formation. Probably, some substance(s) from this exudate (e.g. minerals, proteins, or carbohydrates) favor both the initial adhesion and/or the growth of the early colonizing bacteria.
- Additionally, it is known that during the night, plaque growth rate is reduced by some 50%. This seems surprising, since one would expect that reduced plaque removal and the decreased salivary flow at night would enhance plaque growth. The fact that the supragingival plaque obtains its nutrients mainly from the saliva appears to be of greater significance than the antibacterial activity of saliva.

The Impact of Patient's Age.

- Although older studies were contradictory, more recent papers clearly indicate that a subject's age does not influence de novo plaque formation.

Spontaneous Tooth Cleaning.

- Many clinicians still believe that plaque is removed spontaneously from the teeth such as during eating. However, based on the firm attachment between bacteria and surface, this seems unlikely. Even in the occlusal surfaces of the molars, plaque remains, even after chewing fibrous food.

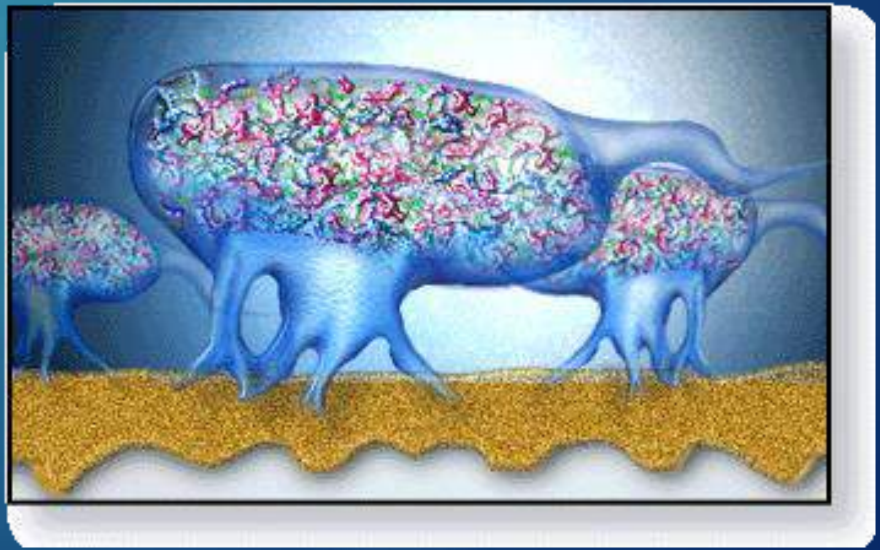
No reduction of 100 hours old dental plaque before dinner **(A)**, and after dinner **(B)** with eating fibrous food

Metabolism of Dental Plaque Bacteria

- The majority of nutrients for dental plaque bacteria originate from saliva or GCF.
- Although the host diet provides an occasional but nevertheless important food supply.
- The growth of *P. gingivalis* is enhanced by metabolic byproducts produced by other microorganisms, such as succinate from *Capnocytophaga ochrecea* and protoheme from *Campylobacter rectus*. Overall, the total plaque population is more efficient than any one constituent organism at releasing energy from the available substrates.
- Metabolic interactions occur also between the host and plaque microorganisms. Increases in steroid hormones are associated with significant increases in the proportions of *P. intermedia* found in subgingival plaque. These nutritional inter-dependencies are probably critical to the growth and survival of microorganisms in dental plaque and may partly explain the evolution of highly specific structural interactions observed among bacteria in plaque.

PLAQUE AS A BIOFILM

- As the bacteria attach to a surface and to each other, they cluster together to form microcolonies that are attached to the surface.
- Each microcolony is a tiny, independent community containing thousands of compatible bacteria.
- Different microcolonies may contain different combinations of bacterial species.



Communication between Biofilm Bacteria

- Bacterial cells do not exist in isolation. In a biofilm, bacteria have the capacity to communicate with each other. One example of this is quorum sensing, in which bacteria secrete a signaling molecule that accumulates in the local environment and triggers a response such as a change in the expression of specific genes once they reach a critical threshold concentration. The threshold concentration is reached only at a high-cell density, and therefore bacteria sense that the population has reached a critical mass, or quorum. Responses are induced only when a threshold concentration of the peptide is attained, and thus the peptides act as cell density, or quorum, sensors.

Biofilms and Antimicrobial Resistance

- organisms in a biofilm are 1000 to 1500 times more resistant to antibiotics than in their planktonic state.
- The mechanisms of this increased resistance differ from species to species, from antibiotic to antibiotic, and for biofilm growing in different habitats.
- An important mechanism of resistance appears to be the slower rate of growth of bacterial species in a biofilm, which makes them less susceptible to many but not all antibiotics. The biofilm matrix, although not a significant physical barrier to the diffusion of antibiotics, does have certain properties that can retard antibiotic penetration
- In addition, extracellular enzymes such as β-lactamases, formaldehyde lyase, and formaldehyde dehydrogenase may become trapped and concentrated in the extracellular matrix, thus inactivating some antibiotics

Reference

- Dental plaque biofilm lecture(4th stage)/University of Baghdad/College of Dentistry/Department of Periodontology.

Thank you

Dental Stain

م. سها أسود دهش العزاوي

B.D.S, MSc. Periodontology

Dental Stain

- Pigmented deposits on the tooth surface.

- There has been a recent increase in interest in the treatment of tooth staining and discolorations as shown by the large number of tooth whitening agents appearing on the market.

- The correct diagnosis for the cause of discoloration is important as, invariably, it has a profound effect on treatment outcomes.

COLOUR AND COLOUR PERCEPTION

- Teeth are typically composed of a number of colours and a gradation of colour occurs in an individual tooth from the gingival margin to the incisal edge of the tooth. The gingival margin often has a darker appearance because of the close approximation of the dentine below the enamel.
- In most people canine teeth are darker than central and lateral incisors and younger people characteristically have lighter teeth, particularly in the primary dentition. Teeth become darker as a physiological age change; this may be partly caused by the laying down of secondary dentine, incorporation of extrinsic stains and gradual wear of enamel allowing a greater influence on colour of the underlying dentine.

CLASSIFICATION OF TOOTH DISCOLOURATION

- **INTRINSIC DISCOLORATION**
- Intrinsic discoloration occurs following a change to the structural composition or thickness of the dental hard tissues.
- A number of metabolic diseases and systemic factors are known to affect the developing dentition and cause discoloration as a consequence. Local factors such as injury are also recognized:
- 1.Alkaptonuria
- 2. Congenital erythropoietic porphyria
- 3. Congenital hyperbilirubinaemia
- 4. Amelogenesis imperfecta
- 5. Dentinogenesis imperfecta

6. Tetracycline staining
7. Fluorosis
8. Enamel hypoplasia
9. Pulpal haemorrhagic products
10. Root resorption
11. Ageing

- **EXTRINSIC DISCOLORATION**
- Extrinsic discoloration is outside the tooth substance and lies on the tooth surface or in the acquired pellicle. The origin of the stain may be:
- 1. Metallic
- 2. Non-metallic

- **INTERNALIZED DISCOLORATION**
- Internalized discoloration is the incorporation of extrinsic stain within the tooth substance following dental development. It occurs in enamel defects and in the porous surface of exposed dentine. The routes by which pigments may become internalized are:

1. Developmental defects
2. Acquired defects

a) Tooth wear and gingival recession

b) Dental caries

c) Restorative materials

THE MECHANISMS OF TOOTH DISCOLORATION

- **INTRINSIC TOOTH DISCOLORATION** occurs during tooth development and results in an alteration of the light transmitting properties of the tooth structure.
- *1. Alkaptonuria:* This inborn error of metabolism results in incomplete metabolism of tyrosine and phenylalanine, which promotes the buildup of homogentisic acid. This affects the permanent dentition by causing a brown discolouration.

- *2. Congenital erythropoietic porphyria:*
- Metabolic disorder in which there is an error in porphyrin metabolism leading to the accumulation of porphyrins in bone marrow, red blood cells, urine, faeces and teeth causes A red-brown discolouration of the teeth.
- *3. Congenital hyperbilirubinaemia:* The breakdown products of haemolysis will cause a yellow-green discolouration.
- *4. Amelogenesis imperfecta:* In this hereditary condition, enamel formation is disturbed with regard to mineralization or matrix formation and is classified accordingly. The appearance depends upon the type of amelogenesis imperfecta, varying from the relatively mild hypomature 'snow-capped' enamel to the more severe hereditary hypoplasia with thin, hard enamel which has a yellow to yellow-brown appearance.

Amelogenesis Imperfecta

- *5. Dentinogenesis imperfecta:* Dentine defects may occur genetically or through environmental influences.
- The teeth are usually bluish or brown in color, and demonstrate opalescence on transillumination. The pulp chambers often become obliterated and the dentine undergoes rapid wear, once the enamel has chipped away, to expose the amelo-dentinal junction.

Figure 1. *Generalized opalescent tooth discoloration.*

- *6. Tetracycline staining:* Systemic administration of tetracyclines during development is associated with deposition of tetracycline within bone and the dental hard tissues. Tetracycline is able to cross the placental barrier and should be avoided from 29 weeks *in uterus* until full term to prevent incorporation into the dental tissues. Since the permanent teeth continue to develop in the infant and young child until 12 years of age, tetracycline administration should be avoided in children below this age and in breast-feeding and expectant mothers. Teeth affected by tetracycline have a yellowish or brown-grey appearance

- *7. Fluorosis:*
- This may arise endemically from naturally occurring water supplies or from fluoride delivered in mouth rinses, tablets or toothpastes as a supplement. The severity is related to age and dose.
- The enamel is often affected and may vary from areas of flecking to diffuse opacious mottling, whilst the color of the enamel ranges from chalky white to a dark brown/black appearance. The brown/black discoloration is posteruptive and probably caused by the internalization of extrinsic stain into the porous enamel. Fluoride only causes fluorosis in concentrations of greater than 1 ppm in drinking water.

- *8. Enamel hypoplasia:* This condition may be localized or generalized. The most common localized cause of enamel hypoplasia is likely to occur following trauma or infection in the primary dentition.Such localized damage to the tooth-germ will often produce a hypoplastic enamel defect, which can be related chronologically to the injury. Disturbance of the developing tooth germ may occur in a large number of fetal or maternal conditions eg maternal vitamin D deficiencies, rubella infection, drug intake during pregnancy and in pediatric hypocalcaemic conditions. Such defects will be chronologically laid down in the teeth depending on the state of development at the time of interference. There may be pitting or grooving which predisposes to extrinsic staining of the enamel.

- *9. Pulpal hemorrhagic products:*
- The discoloration of teeth following severe trauma was considered to be caused by pulpal hemorrhage.
- Haemolysis of the red blood cells would follow and release the hem group to combine with the putrefying pulpal tissue to form black iron sulphide.
- The depth of dentinal penetration determines the degree of discolouration.
- *10. Ageing:* The natural laying down of secondary dentine affects the light-transmitting properties of teeth resulting in a gradual darkening of teeth with age.

EXTRINSIC DISCOLORATION

- The causes of extrinsic staining can be divided into two categories; those compounds which are incorporated into the pellicle and produce a stain as a result of their basic colour, and those which lead to staining caused by chemical interaction at the tooth surface.
- Direct staining has a multi-factorial aetiology with chromogens derived from dietary sources or habitually placed in the mouth. Tobacco smoking and chewing are known to cause staining, as are particular beverages such as tea and coffee.
- Indirect extrinsic tooth staining is associated with cationic antiseptics and metal salts. The agent is without colour or a different colour from the stain produced on the tooth surface.

Extrinsic tooth discolouration has usually been classified according to its origin, whether metallic or nonmetallic.

- *Non-metallic stains:* The non-metallic extrinsic stains are adsorbed onto tooth surface deposits such as plaque or the acquired pellicle. The possible etiological agents include dietary components, beverages, tobacco…etc.
- *Metallic stains:* Extrinsic staining of teeth may be associated with occupational exposure to metallic salts and with a number of medicines containing metal salts, e.g:

-The characteristic black staining of teeth in people using iron supplements and iron foundry workers.

- potassium permangenate producing a violet to black colour when

used in mouth rinses;

- silver nitrate salt used in dentistry causes a grey colour, and
- stannous fluoride causes a golden brown discolouration.

INTERNALIZED DISCOLORATION

- The stains taken up into the body of enamel or dentine are the same as that causing extrinsic tooth discolouration, including in particular dietary chromogens and the by-products of tobacco smoking. Dental defects permitting the entry of chromogenic material can be classified under the headings of 'developmental and acquired'.
- *1. Developmental defects:* The most important defects are considered under the 'intrinsic tooth discoloration' section. developmental defects may expose dentine either directly or later caused by early loss of enamel as in dentinogenesis imperfecta. Chromogens are then able to enter the dentine directly or facilitated almost certainly by the tubule system.

- *2. Acquired defects:*
- *a) Tooth wear and gingival recession :*Tooth wear is usually considered to be a progressive loss of enamel and dentine due to erosion, abrasion and attrition. As enamel thins the teeth become darker as the colour of dentine becomes more apparent. Once dentine is exposed the potential of chromogens to enter the body of the tooth is increased.
- *b) Dental caries:* The various stages of the carious process can be recognized by changes in colour as the disease progresses. For instance, the initial lesion is characterized by an opaque, white spot. The hard, arrested lesion is black having picked up stain from exogenous sources.
- *c) Restorative materials including amalgam:* Some of the materials used in restorative dental treatment may have an effect on the color of teeth.Eugenol and phenolic compounds used during root canal therapy contain pigments which may stain dentine. Some of the poly antibiotic pastes used as root canal medicaments may cause a darkening of the root dentine. Clinicians are familiar with the dark grey to black colour of dentine following the removal of a long-standing amalgam restoration.

HOW CAN WE PREVENT TEETH DISCOLORATION?

- By making a few simple lifestyle changes, you may be able to prevent teeth discoloration. For example, if you are a coffee drinker and/or smoker, consider cutting back or quitting all together. Also, improve your dental hygiene by brushing and flossing regularly and getting your teeth cleaned by a dental hygienist every 6 months.

WHAT TREATMENT OPTIONS ARE AVAILABLE TO WHITEN TEETH?

- Dental treatment of tooth discoloration involves identifying the etiology and implementing therapy.
- A- Diet and habits: Extrinsic staining caused by foods, beverages, or habits (e.g., smoking, chewing tobacco) is treated with a thorough dental rophylaxis and cessation of dietary or other contributory habits to prevent further staining.
- B- Tooth brushing: Effective tooth brushing twice a day with a dentifrice helps to prevent extrinsic staining. Most dentifrices contain an abrasive, a detergent, and an anti tartar agent. In addition, some dentifrices now contain tooth-whitening agents.

- C- Professional tooth cleaning: Some extrinsic stains may be removed with ultrasonic cleaning, rotary polishing with an abrasive prophylactic paste, or air-jet polishing with an abrasive powder. However, these modalities can lead to enamel removal; therefore, their repeated use is undesirable.
- D- Bleaching (tooth whitening): Bleaching includes 2 types of techniques: vital and nonvital.
- Vital bleaching: Currently, the bleaching agents most commonly used are carbamide and hydrogen peroxide.
- **In office "power" bleaching** involves the use of a 15-40% hydrogen peroxide solution and must be performed by a dental professional because careful isolation of the teeth is required to protect the soft tissues from the caustic effects of the bleaching agent.
- **home bleaching systems** may be used alone or in combination with in-office bleaching. The systems must be used under the careful supervision of dentists or dental hygienists. Patients apply a 10-22% carbamide peroxide solution into a custom-made mouth guard. After repeated daily and/or nightly (often while patients sleep) applications for 2-6 weeks, the teeth are gradually bleached.

- Non vital bleaching
- Non vital bleaching is indicated for the treatment of teeth with discoloration secondary to pulpal degeneration. This technique involves placing a mixture of 30% hydrogen peroxide and sodium perborate into the pulp chamber for as long as 1 week.

Reference

- Dental stain lecture(4th stage)/University of Baghdad/College of Dentistry/Department of Periodontology.

Thank you

بسم الله الرحمن الرحيم

ETIOLOGY OF PERIODONTAL DISEASES

أعداد:

د. نور صباح أرحيم

Periodontal diseases:

are the most prevalent and multi-factorial diseases that involved hard and soft dental tissues, bacterial colonization, and immune responses of the host. periodontal diseases affecting a large population worldwide. plaque , being the primary etiological agent, a genetic origin may be play a part. there are two types of periodontal diseases, gingivitis and periodontitis.

GINGIVITIS:

It is inflammation of the gingiva in which the junctional epithelium remain attached to the tooth at its original level. It is characterized by areas of redness and swelling, and there is a tendency for the gingiva to bleed easily. Gingivitis is limited to the epithelium and gingival connective tissues. It is important to note that there is no tissue recession or loss of connective tissue or bone. The occurrence of gingivitis is wide spread in the population. It is reversible condition .

Periodontitis:

it is a common , chronic inflammatory disease (inflammation of the supporting tissues of the teeth leading to permanent destruction of theses tissues) caused by the accumulation of bacterial matrix at the gingival line. it is characterized by clinical attachment loss, periodontal pocketing and alveolar bone loss. it is irreversible disease can cause a breakdown of the periodontium result in the loss of the tissue attachment and destruction of the alveolar bone.

Contained gingivitis:

in some individuals gingivitis may not progress to periodontitis for a long period of time even if is not treated and this condition depend on host response and the pathogenicity of bacteria.

Risk factors:

Can be defined as characteristics or factors that when present increases the risk that an individuals will get the diseases. It is important to make the distinction that risk factors are associated with a disease but do not necessarily cause the disease, can be classified into local and systemic risk factors.

LOCAL RISK FACTORS:

1-anatomical risk factor

-enamel pearls.

-root groove (palatogingival groove).

-furcation.

-gingival recession.

2-tooth position

-misalignment.

-crowding.

-tipping.

-migration.

-occlusal forces.

3-Iatrogenic risk factor

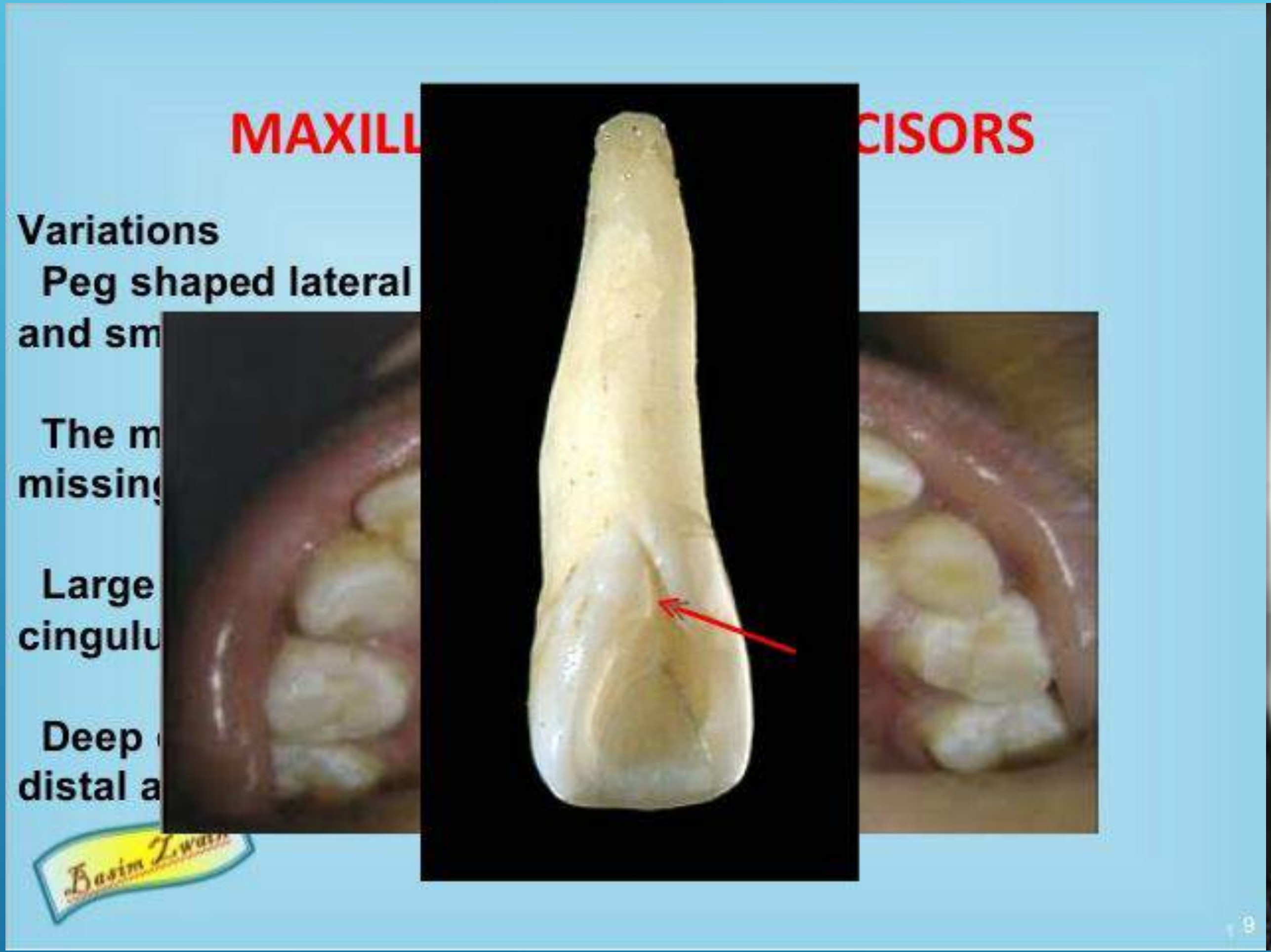
-partial dentures

-overhang restoration.

-orthodontic appliance.

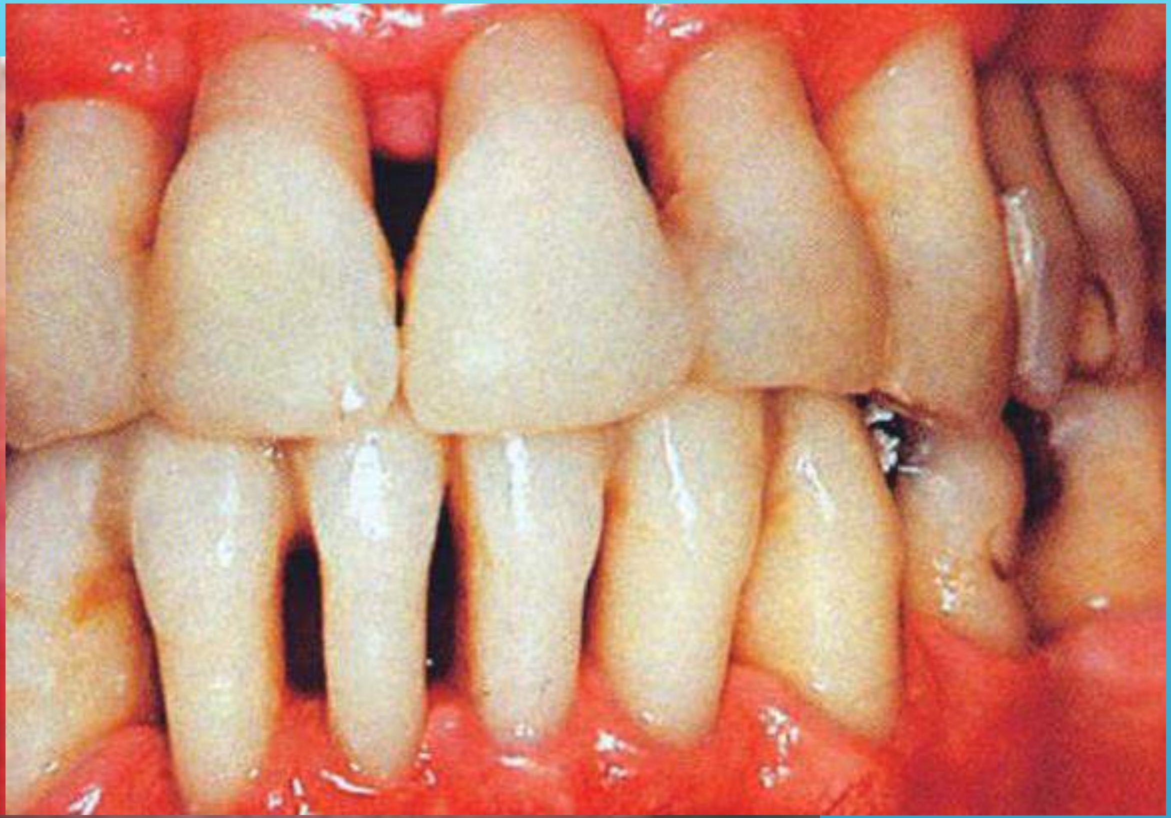
ENAMEL PEARLS

PALATOGINGIVAL GROOVE



FURCATION AREA





Upper Crowding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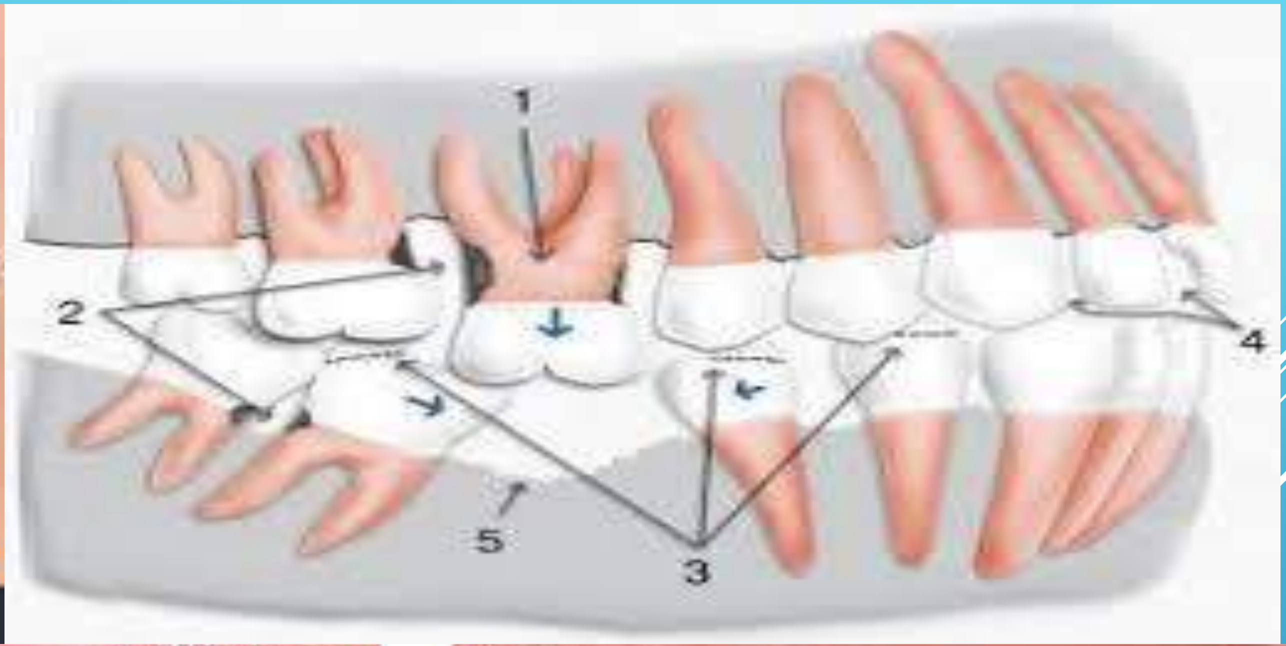
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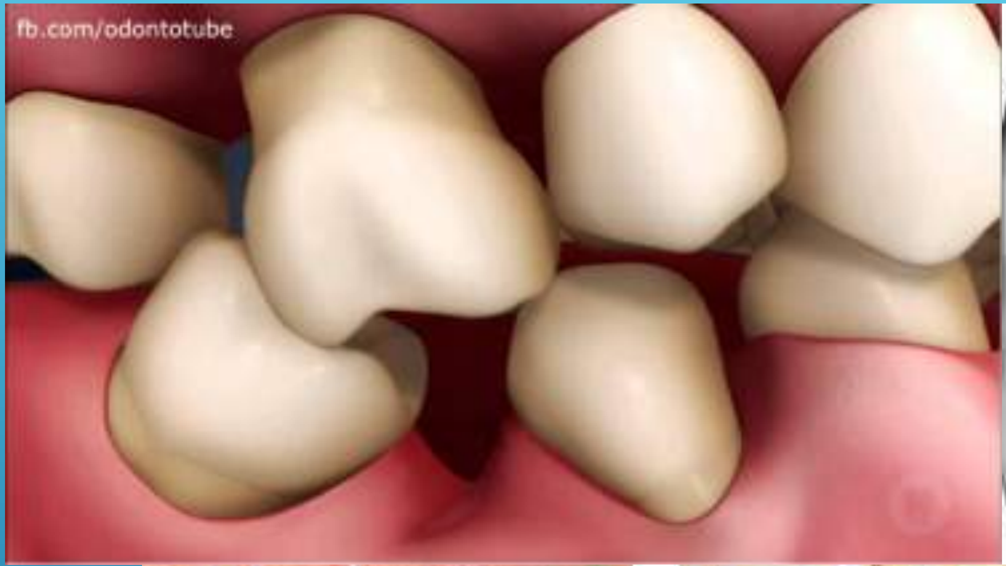
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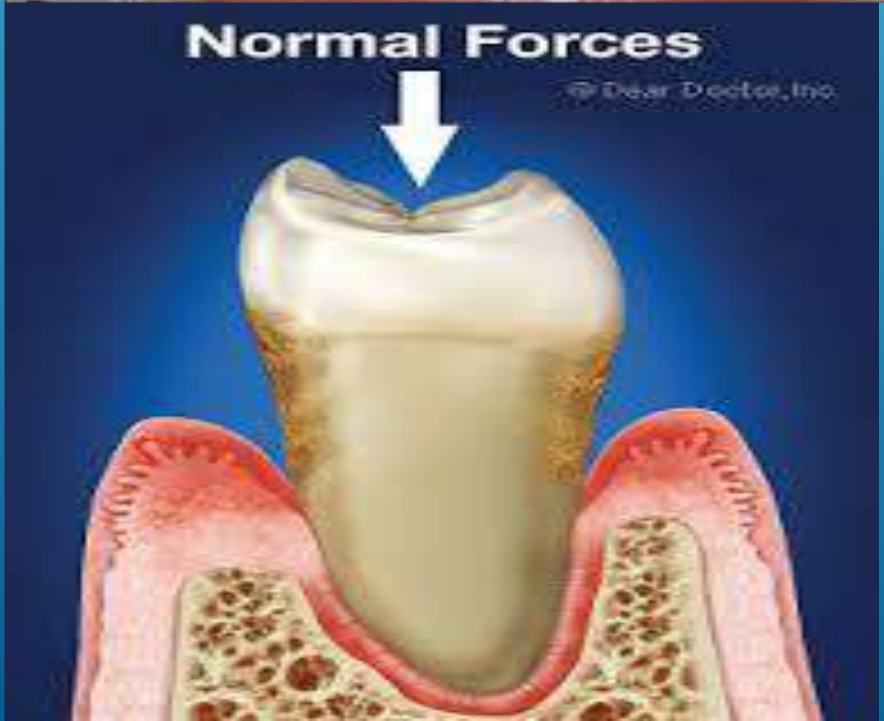
Normal Forces
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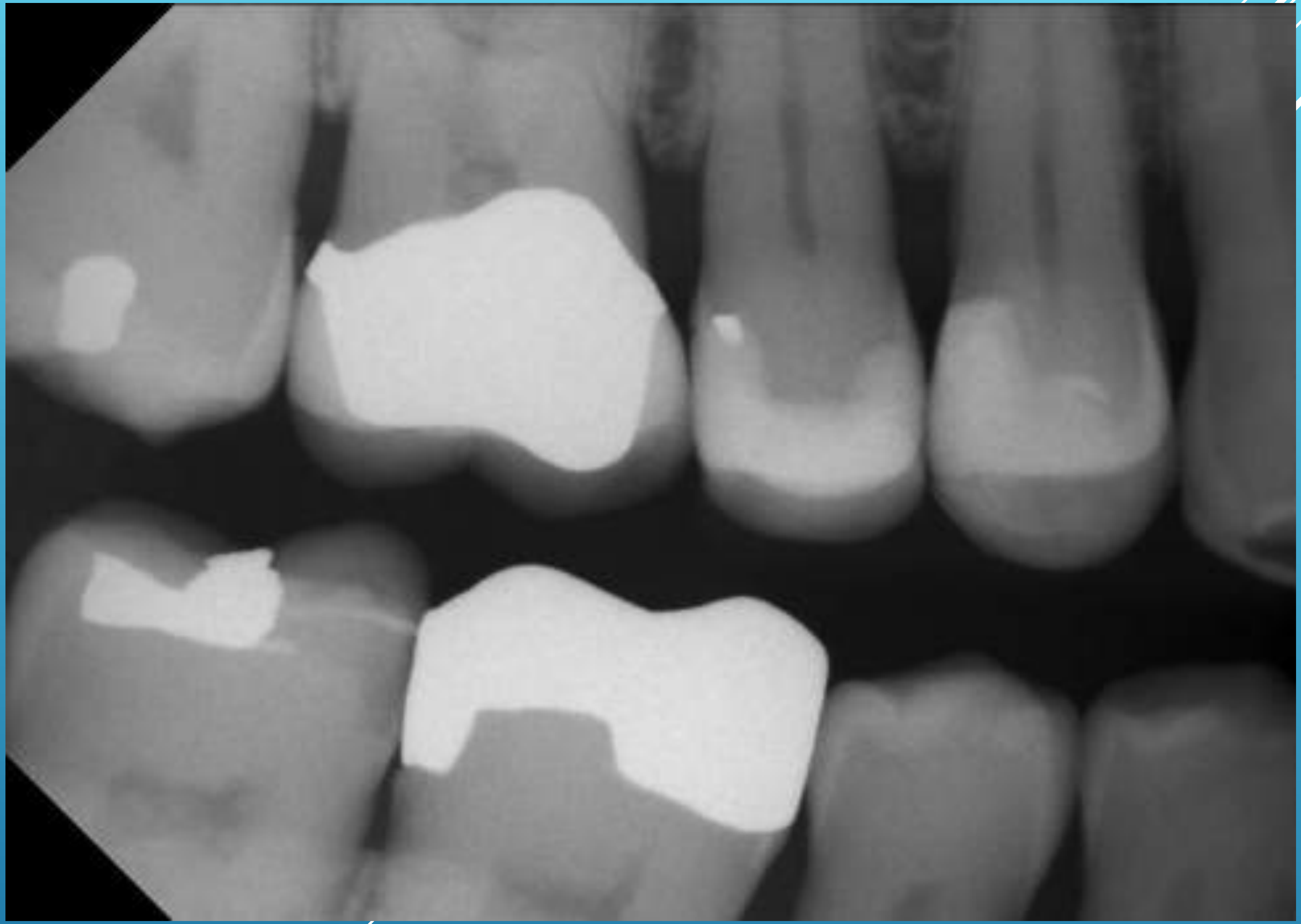
overhang restoration





partial denture

Orthodontic appliance

THANK YOU

GINGIVA 2

PRESENTED BY :

DR. NOOR SABAH IRHAYYIM

Normal microscopic feature of gingiva

-The gingiva consists of fibrous connective tissue known as lamina properia covered by stratified squamous epithelium.

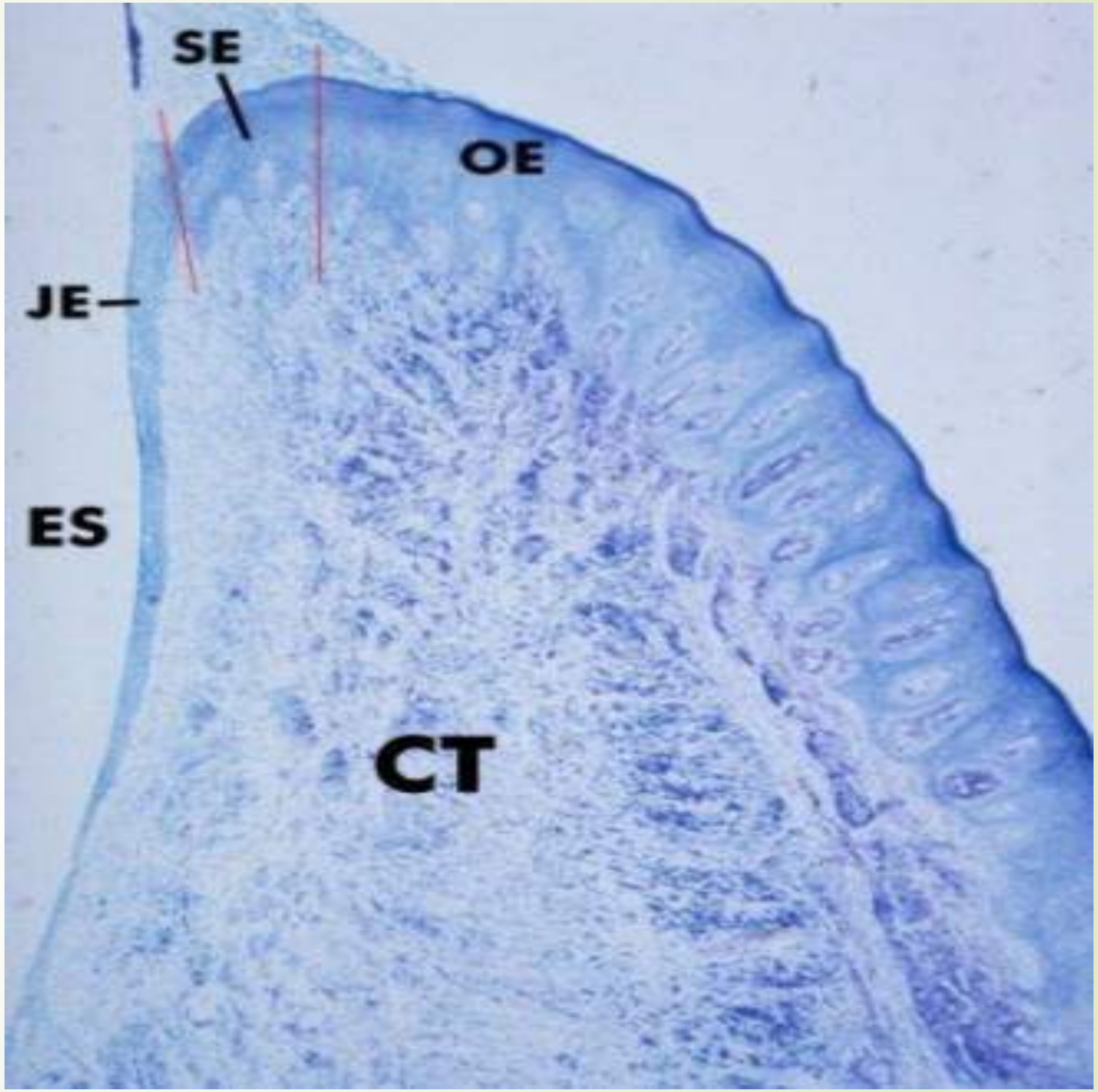
-gingival epithelium may be differentiated as follows:

1-oral epithelium: which faces the oral cavity

2-Sulcular epithelium: which faces the tooth in the gingival sulcus without being in contact with the tooth surface.

3-Junctional epithelium: which provides the contact between the gingiva and the tooth.

CT, gingival connective tissue
ES, ⑥🕘 enamel space
JE, ⑥🕘 junctional epithelium
OE, oral ⑥🕘 epithelium
SE, ⑥🕘 sulcular epithelium

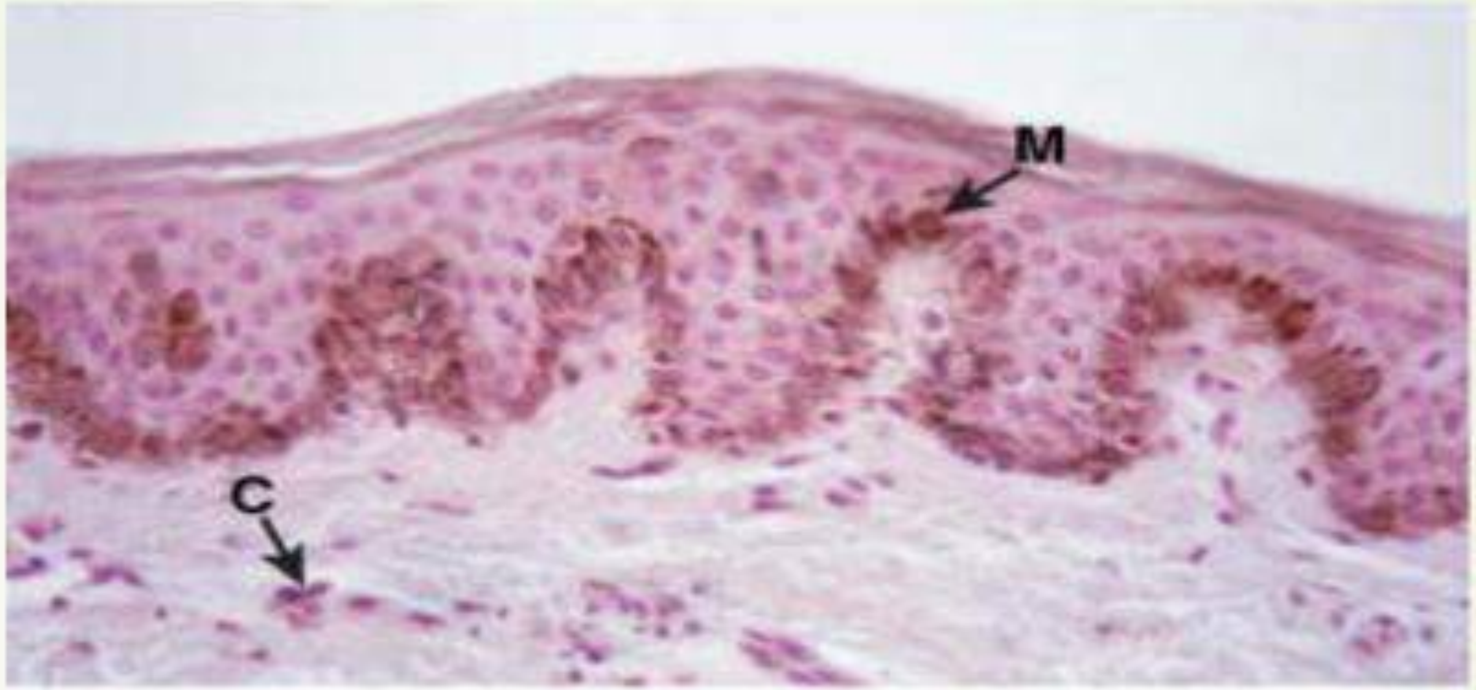


Oral epithelium:

- -It covers the crest and the outer surface of the marginal and attached gingiva. It is either keratinized (no nuclei) or parakeratinized (retained nuclei). The boundary between the oral epithelium and the underlying connective tissues has a wavy course.

- -The projection of the epithelium cells into he connective tissues are known as (Rete Pegs). The intervening connective tissue portion which project into the epithelium are called connective tissue papillae.

- -This alternating pattern of depression and protuberances of the connective tissue papillae and epithelium rete pegs thought to give the attached gingiva (the stippling appearances)

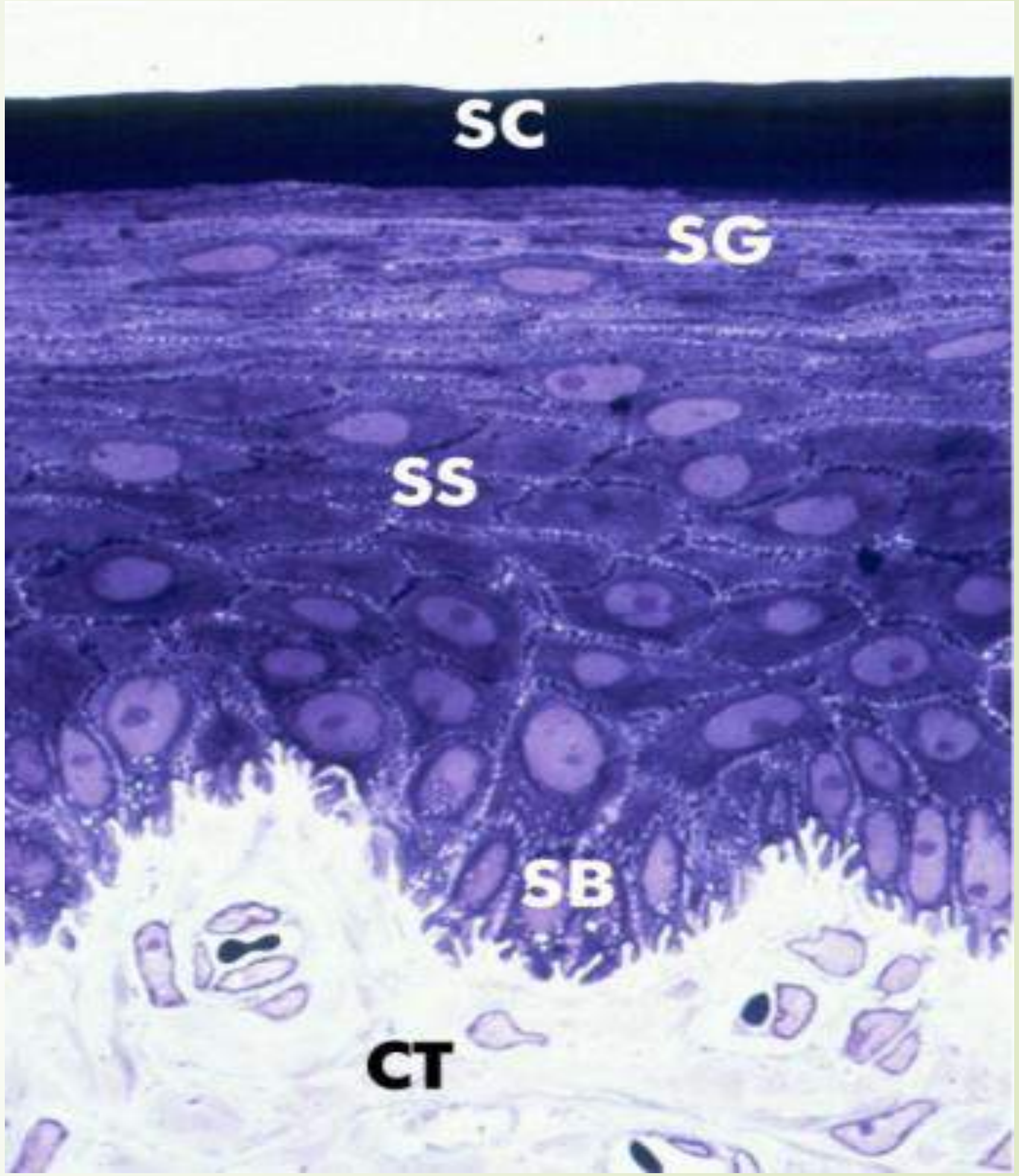


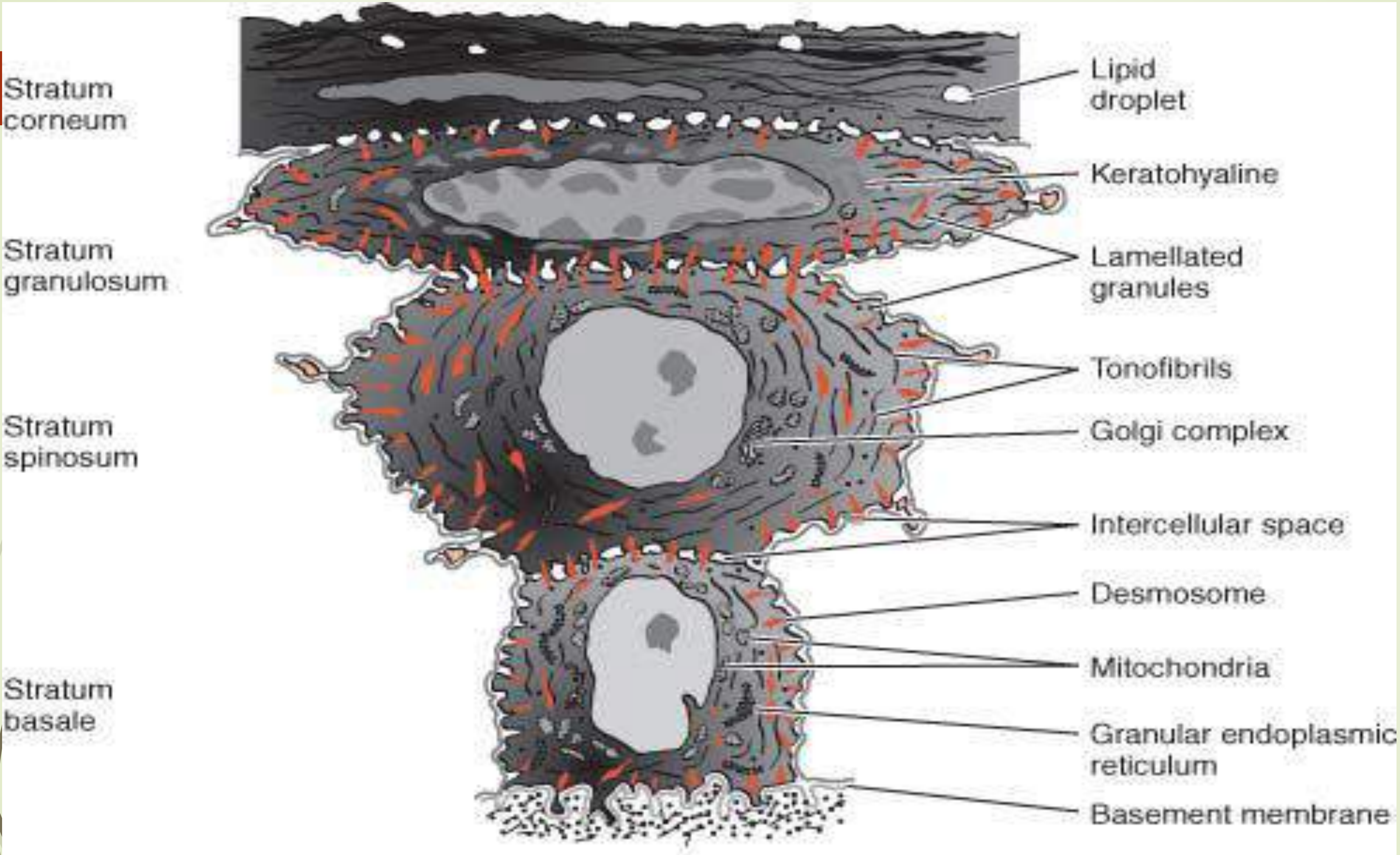
Pigmented gingiva of dog showing melanocytes (M) in the basalepithelial layer and melanophores (C) in the connective tissue (Glucksman technique).

The oral epithelium consists of four layers of cells

- **1-Stratum basale:** Basal layer of cuboidal cells along the basement membrane . This is where epithelial cell replication and cell differentiation begins.(melanocytes are found in this layer)
- **2-Stratum spinosum**: the cells appear to have cytoplasmic spines. This is the thickest cell layer and Langerhans cells are found in this layer.
- **3-Stratum granulosum**: Keratohyaline granules may be seen in this layer. Cells appear to be flattened.
- **4-Stratum corneum:** This is the layer where both para or the orthokeratinized occur. It is the most superficial layer.
- -The epithelium cells are formed as basal cells and gradually they undergo the process of keratinization, this is achieved by proliferation and differentiation of these cells (change to the characteristic of each of the cell layer) as they migrate towards the surface layer.

SC, stratum corneum (cornified layer)
SG,stratum ⑥🕘 granulosum (granular layer)
🕘⑥**SS,** stratum spinosum (spinous layer)
🕘⑥**SB,**stratum basale (basal layer)
CT, connective ⑥🕘 tissue




Stratum
corneum
Stratum
granulosum
Stratum
spinosum
Stratum
basale
Lipid
droplet
Keratohyaline
Lamellated
granules
Tonofibrils
Golgi complex
Intercellular space
Desmosome
Mitochondria
Granular endoplasmic
reticulum
Basement membrane

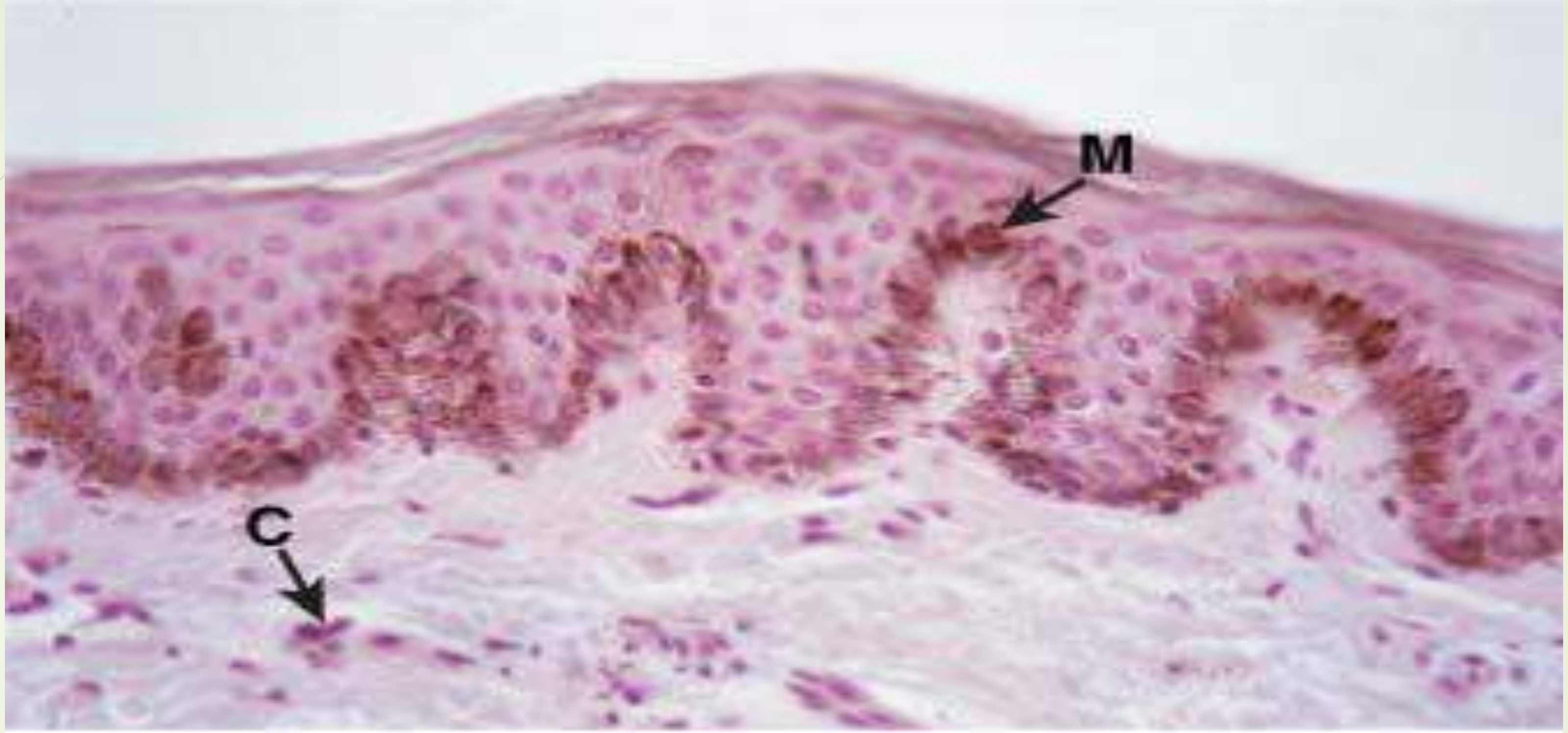
The oral epithelium contains the following types of cells:

1- Keratinocytes cells: These are Keratin producing cells which comprise about 90% of the total cell population. These cells undergo continuous proliferation and differentiation from basal layer to the surface of epithelium. Keratin may be found in the stratum corneum and contribute to the protective function of epithelium.

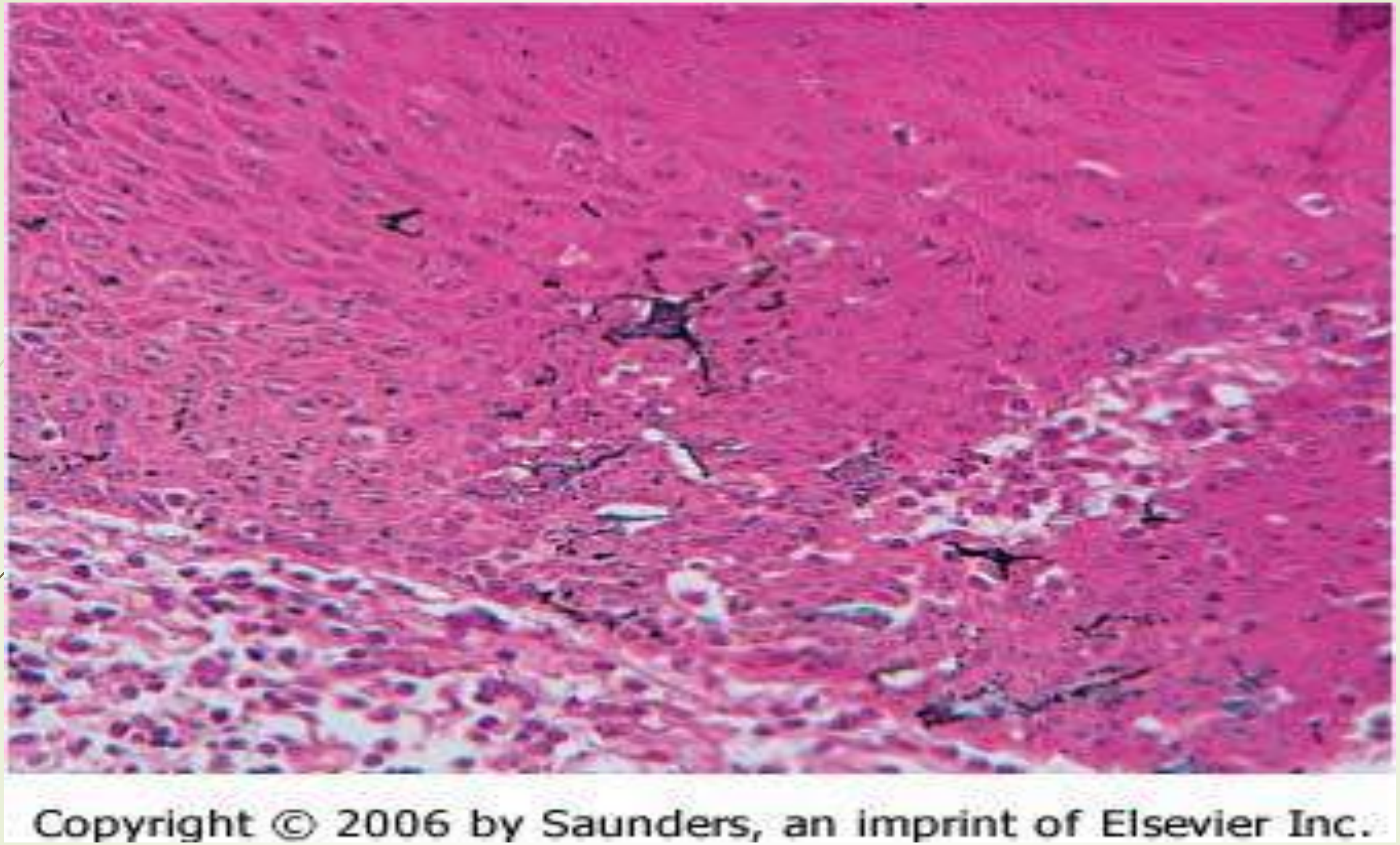
2-Melanocytes: cells of basal layer that produce melanin pigment granules.

3-Langerhans cells: these cells play a role in the defense mechanisms of the oral epithelium. They have an immunological function by recognizing and processing antigens.

4-Markel cells: these are located in the deeper layers of the epithelium , harbor nerve ending. They have been identified as tactile receptors.

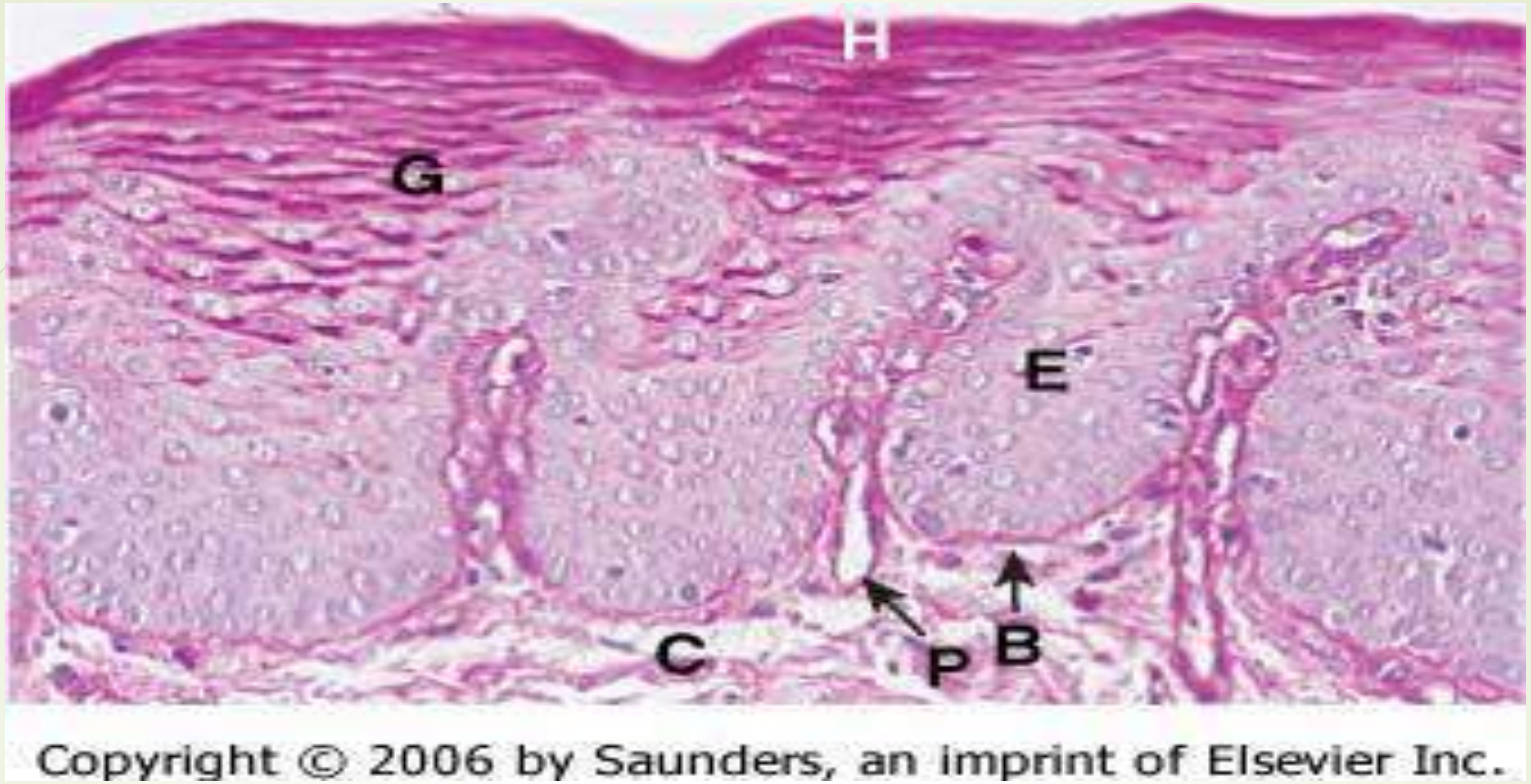


Pigmented gingiva of dog showing melanocytes (M) in the basalepithelial layer and melanophores (C) in the connective tissue (Glucksman technique).



Human gingival epithelium, oral aspect. Immunoperoxidase technique showing Langerhans cells. Function: On infectionof an area of skin, the local Langerhans' cells will take up and process microbialantigensto become fully-functional antigen-presenting cells

- Under a normal condition, there is complete equilibrium between cell renewal and desquamated (cell turn over). It takes approximately 3-4 weeks for keratinocytes to migrate from basal layer until reach the outer epithelial surface, where it become desquamated from stratum corneum.
- Basal cells are found immediately adjacent to the connective tissue and are separated from this tissue by a basement membrane (basal lamina).
- The basement membrane consists of :
- 1-Lamina lucida: which is located immediately beneath the basal cell layer.
- 2- lamina densa: located beneath the lamina lucida, from this structure the acchoring fibers project into the connective tissue.
- -The epithelial cell are joined together by structure known as desmosome, which is composed of two hemi desmosome separated from each other by granulated material.



Normal human gingiva stained with the periodic acid-Schiff (PAS) histochemical method. The basement membrane (B) is seen between the epithelium (E) and the underlying connective tissue (C). In the epithelium, glycoprotein material occurs in cells and cell membranes of the superficial hornified (H) and underlying granular layers (G). The connective tissue presents a diffuse, amorphous ground substance and collagen fibers. The blood vessel walls stand out clearly in the papillary projections of the connectivetissue (P).

A hemi desmosome composed from the following structures:

1- the outer leaflets (OL)

2-the inner leaflet (IL)

3-the attachment plaque (AP)

Sulcular Epithelium

- 1-It lines the gingival sulcus .
- 2- It is a thin, non keratinized stratified squamous epithelium without rete pegs and extend from the coronal limit of the junctional epithelium to the crest of the gingival margin.
- 3-The sulcular epithelium important because it is thin and may act as a semipermeable membrane through which tissue fluid from the gingiva seep into the sulcus and makes easier for bacterial products of dental plaque to penetrate into the connective tissue of the gingiva and stimulate the inflammation and tissue destruction , That why the sulcular epithelium id considered as a poor barrier against bacterial infection.

Junctional epithelium (JE)

-the epithelium that attaches the gingiva to the surface of the tooth

-it consists of stratified squamous non-keratinized epithelium

-it is 3-4 layers thick in early life but the numbers of increases with age to 10-20

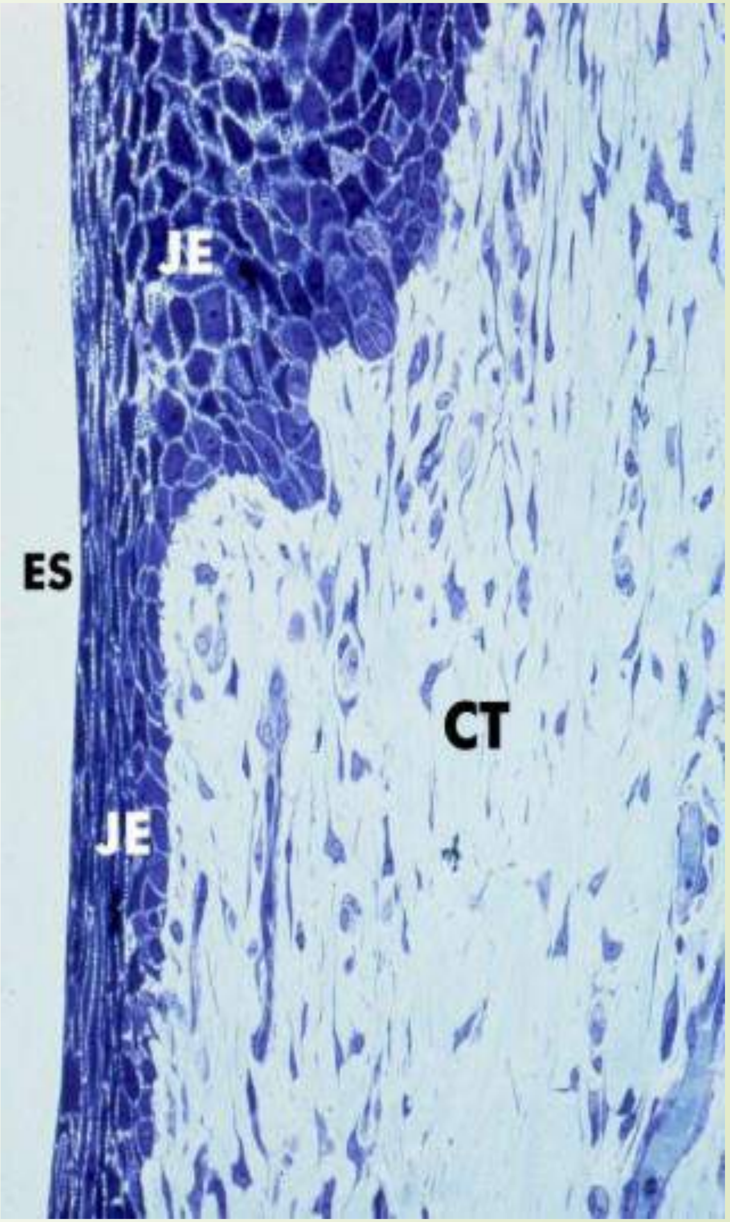
-it is thicker in coronal portion nut become thinner towards cmento-enamel junction only a few cell layers

- the junctional epithelium cells can be grouped in two layers: the basal and suprabasal layer.

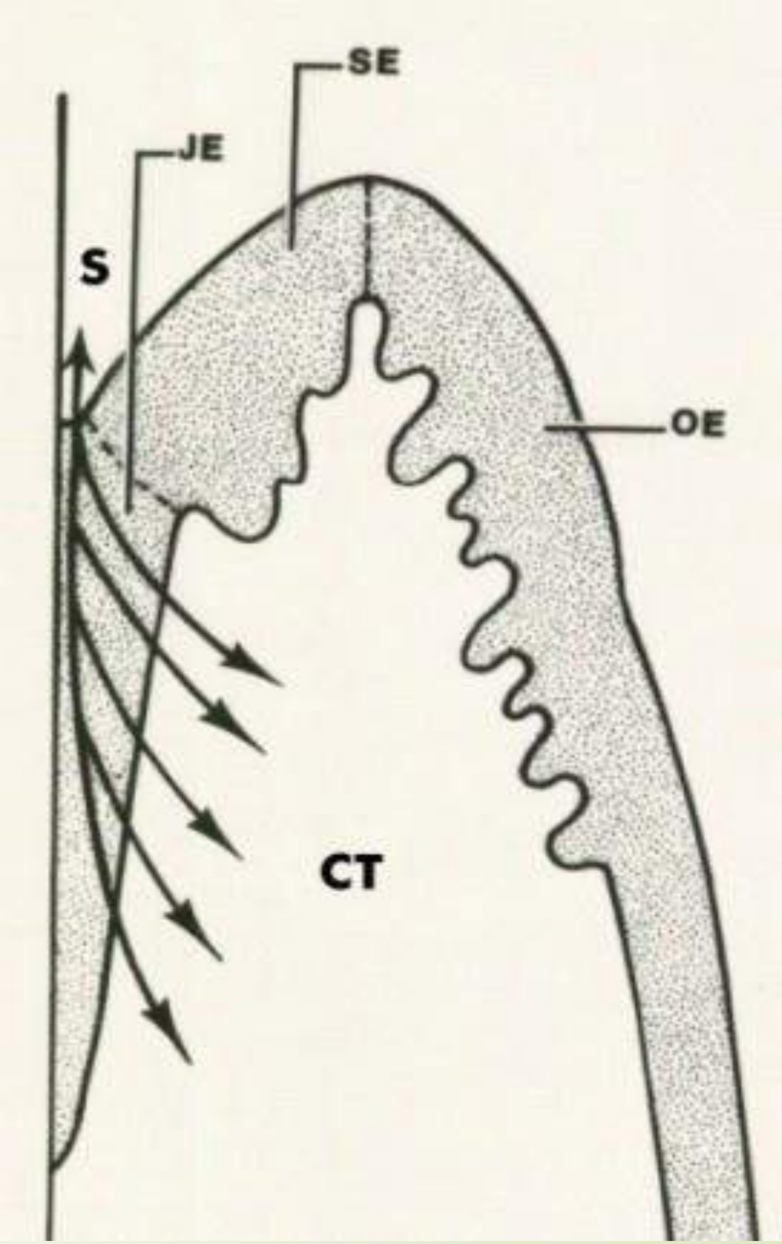
The epithelium is continuously renewed through cell division in the basal layer and the cell migrate coronally to the base of the gingival sulcus from they are shed (cell turn over)

-the JE assume a key role in maintenance of periodontal health, it creates the firm epithelium attachment that connect the soft tissue to the tooth surface, it is quite permeable and thus serve as a pathway for diffusion of products plaque bacteria to connective tissue, there is also diffusion in the opposite direction moving towards the sulcus of host defense substance, this help to maintain the immune response.

CT,connecti
ve tissue
ES, enamel
space
JE,junctiona
l epithelium



CT, connective tissue
JE, junctional epithelium
OE, oral epithelium
S,gingival sulcus
SE, sulcular epithelium



There are distinct differences between the JE, sulcular and the oral epithelium :

- 1-The size of the cells in the JE is relatively larger than the oral epithelium
- 2-the intercellular space in the JE is wider than in the oral epithelium. The intercellular space of the JE is preferred route for tissue fluid and inflammatory cells to migrate from the CT to gingival sulcus.
- 3-The number of desmosome (intercellular junction) is fewer in the junctional epithelium than in the oral epithelium , this may explain the JE susceptibility to tear during probing and it is greater permeability to migrate cells and fluid.
- 4-The sulcular and junctional epithelium are not as thick as the oral epithelium, because they are not keratinized and in health have no retepegs.
- 5-JE turnover rate is very high (4-6) days compared to oral epithelium that has longest turn over rate (6-12 days) or (up to 40 days).
- 6-JE forms the attachment of the gingiva to the tooth surface while oral and sulcular epithelium have no attachment to the tooth surface.

Gingival connective tissue:

- The connective tissue of the gingiva known as the lamina properia and consists of two layer:
- 1-The papillary layer: it consists of papillary projections between the epithelium retepeges.
- 2-The reticular layer: it is contiguous with the periosteum of the alveolar bone
- -The major components of the CT are:
- 1-collagen fibers 60%
- 2-cells 5%
- 3-ground substances, blood vessels, nerve, and lymphatics(35%).

Cells of gingival CT:

1-Fibroblasts
2-Mast cells
3-macropgage
4-inflammatory cells

Ground substances of gingival CT

- 1-the matrix fill the space between fibers, cells and have high content of water, electrolytes, nutrition, metabolites, glycoprotein and proteoglycan.
- 2-it is produced by fibroblast and some from the mast cells and other from the blood.
- 3-it is a medium for the cells of CT to embedded and for maintained the normal function of the CT

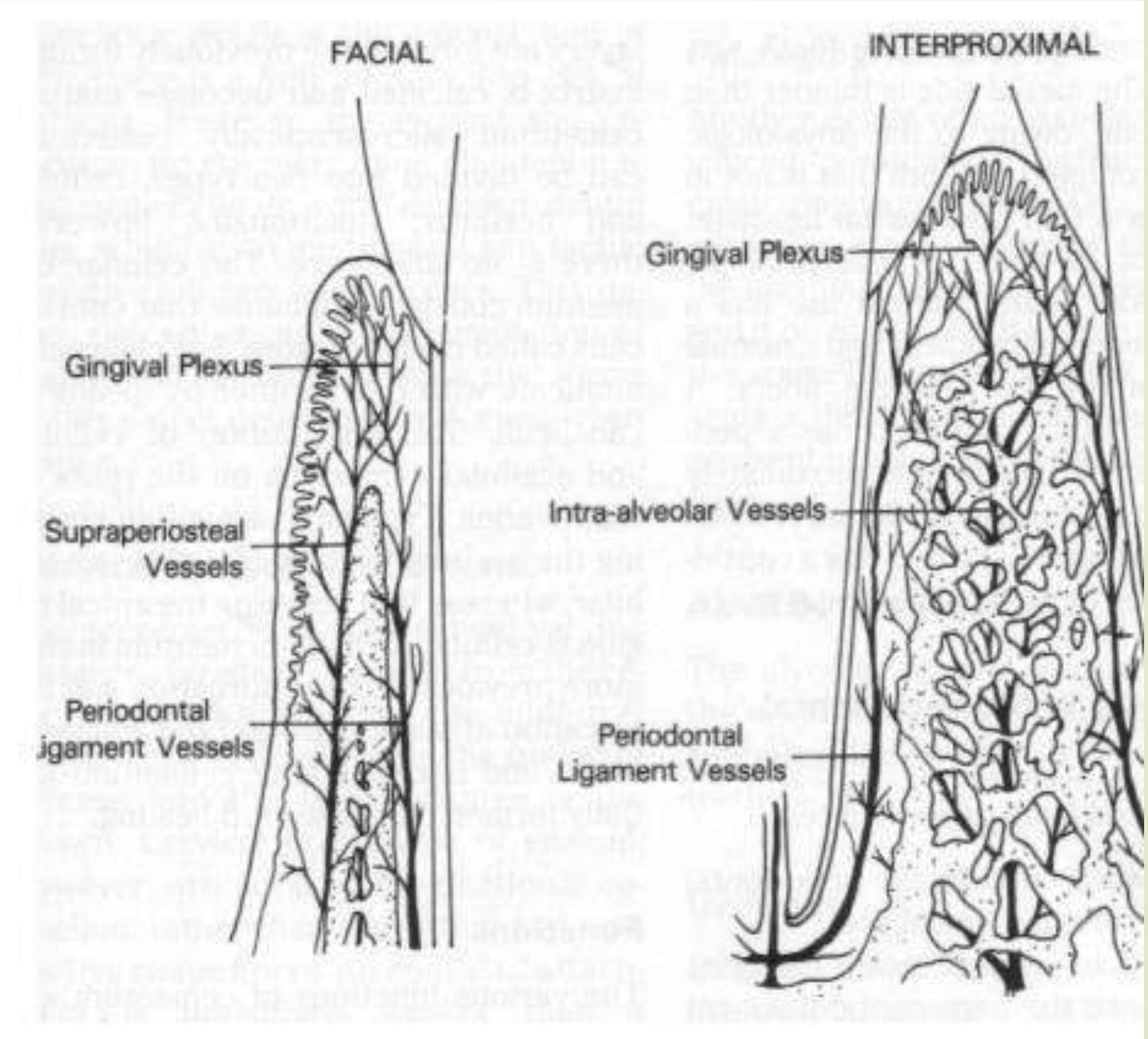
Blood vessels of gingiva

- gingival tissue has rich vascular supply which arise from the terminal branches of internal maxillary artery.

-consist of

1-supra periosteal vessels'

2-vessels from the PDL and bone


FACIAL
INTERPROXIMAL
Gingival Plexus
Supraperiosteal Vessels
Periodontal Ligament Vessels
Gingival Plexus
Intra-alveolar Vessels
Periodontal Ligament Vessels

Innervation of the gingiva

- Nerve supply to gingiva derived from the terminal branches of the maxillary and mandibular branches of the trigeminal nerve.

THANK YOU

Impact of periodontal infection on systemic health

Presented by:

Dr. Noor Sabah Irhayyim

Periodontal disease is an inflammatory disease initiated by bacterial pathogens. Environmental, physical, social, and host stresses may affect and modify disease expression through a multitude of pathways. Certain systemic conditions can affect the initiation and progression of gingivitis and periodontitis. Systemic disorders that affect neutrophil, monocyte, macrophage, and lymphocyte function result in the altered production or activity of host inflammatory mediators. These alterations may manifest clinically as the early onset of periodontal destruction or as a more rapid rate of destruction than would occur in the absence of such disorders.

- There are many **systemic conditions** that can modify the host's susceptibility
- to periodontitis. For example, patients with **immune suppression** may not be able to amount an effective host response to subgingival microorganisms, thereby resulting in more rapid and severe periodontal destruction. Conversely, individuals with a significant increase in the production of proinflammatory mediators may respond to periodontal pathogens with an exuberant inflammatory response that results in the destruction of periodontal tissues.

- Conditions in which the influences of periodontal infection are documented include **coronary heart disease (CHD)** and **CHD-related events** such as **angina**, **infarction**, **atherosclerosis**, and other vascular conditions; **stroke**; **diabetes mellitus**; **preterm labor**, **low-birth-weight delivery**, and respiratory conditions such as **chronic obstructive pulmonary disease**

Subgingival environment as a reservoir for bacteria:

- The subgingival microbiota in patients with periodontitis provides a significant and persistent gram-negative bacterial challenge to the host that is met by a potent immunoinflammatory response. These organisms and their products, such as lipopolysaccharide (LPSs), have ready access to the periodontal tissues and to the circulation via the sulcular epithelium, which is frequently ulcerated and discontinuous. Even with treatment, the complete eradication of these organisms is difficult .

❖ The mechanisms by which periodontal infections may influence systemic health have been described as follows

1. Oral-hematogenous spread of periodontal pathogens and direct effects to target organs.

2. Transtracheal spread of periodontal pathogens and direct effects to target organs.

3. Oral-hematogenous spread of cytokines and antibodies with effects at distant organs

Periodontal Disease and Coronary Heart Disease/Atherosclerosis/stroke:

- To further explore the association between periodontal disease and
- CHD/atherosclerosis, investigators have studied specific systemic disorders and medical outcomes to determine their relationship to periodontal status. CHD-related events are a major cause of death. Myocardial infarction (MI) has been associated with acute systemic bacterial and viral infections. Traditional risk factors such as smoking, dyslipidemia, hypertension, and diabetes mellitus do not explain the presence of coronary atherosclerosis in a large number of patients. Localized infection that results in a chronic inflammatory reaction has been suggested as amechanism underlying CHD in these individuals. The association between poor
- dental health and MI was independent of known risk factors for heart disease, such
- as age, cholesterol levels, hypertension, diabetes, and smoking. Because
- atherosclerosis is a major determinant of CHD-related events, dental health has also
- been related to coronary atheromatosis

- Damage to the vascular endothelium, with a subsequent inflammatory
- reaction, plays a major role in atherosclerosis and ischemic organ damage. increased
- viscosity of blood may promote major ischemic heart disease and cerebrovascular
- accident (stroke) by increasing the risk of thrombus formation because of increased
- fibrinogen levels which increase blood viscosity. Fibrinogen levels and white blood
- cell counts are often increased in patients with periodontal disease, so individuals
- with poor oral health may also have significant elevations in coagulation factor VIII.
- Thus, periodontal infection may also promote increased blood viscosity and
- thrombogenesis, which leads to an increased risk for central and peripheral vascular
- disease. C reactive protein (CRP) induces monocytes and macrophages to produce
- tissue factor, which stimulates the coagulation pathway and increases blood
- coagulability. Periodontal infections may contribute to atherosclerosis and
- thromboembolic events by repeatedly challenging the vascular endothelium and
- arterial wall with bacterial LPSs and proinflammatory cytokines.

Thus, periodontal diseases may have both direct effects on the major blood •

vessels (e.g., atheroma formation) and indirect effects that stimulate changes in the cardiovascular system (e.g., elevation of systemic inflammatory responses). Oral organisms may be involved in coronary thrombogenesis. Platelets selectively bind some strains of *Streptococcus sanguinis*, which is a common component of supragingival plaque, and ***Porphyromonas gingivalis***, which is a pathogen closely associated with periodontitis •

- It has been found that gram negative bacteria & associated lipopolysaccharides (LPSs) plays an important role in atherogenesis because it has the ability to trigger the release of interleukin 1, tumor necrosis factor- alfa (TNF-α) & thromboxane that initiate platelet aggregation, promote the deposition of cholesterol & enhance atheroma formation. The role of (LPSs) as a systemic trigger for the development of atheroma has led the investigators to search for an infection site which would provide a source for (LPSs).

- In individuals with periodontitis, the periodontium can serve as a reservoir for
- (LPSs)& inflammatory cytokines. Many studies have demonstrated a significant association between periodontal disease severity & stroke, myocardial infarction &coronary atherosclerosis. These investigations seem to suggest that because of chronicity of periodontal diseases & sustained release of bacteria & endotoxins into blood stream, periodontitis can contribute to systemic effects as atheroma development.

Ischemic cerebral infarction, or stroke, is often preceded by systemic bacterial or viral infection. Stroke is classified as either hemorrhagic or nonhemorrhagic. Nonhemorrhagic stroke, or ischemic stroke, is usually caused by thromboembolic events and cerebrovascular atherosclerosis, whereas hemorrhagic stroke often results from a vascular bleed such as an aneurysm. Periodontal disease has been associated primarily with an increased risk of nonhemorrhagic stroke. Numerous studies have shown that periodontal disease is associated with an increased risk of stroke.

Dental diseases may be indicators of general health practices. For example, •

periodontal disease and CHD are both related to lifestyle and share numerous • risk factors, including smoking, diabetes, and low socioeconomic status. Bacterial infections have significant effects on endothelial cells, blood coagulation, lipid metabolism, and monocytes and macrophages.

•

Periodontal disease and diabetes mellitus: •

The role of diabetes in periodontal disease is bi-directional, that is diabetes is a known risk factor for periodontitis and periodontitis in turn affects the glycemic control in individuals with diabetes. Periodontal infections result in an elevation of serum pro-inflammatory markers, these may adversely affect metabolic control, may result in insulin resistance which •

in turn over time can lead to hyperglycemia and type II diabetes. Chronic gram negative periodontal infections in individual with diabetes may also worsen •

glycemic control. Patients that harbor periodontal pathogens have significantly •

higher markers of systemic inflammation like C-reactive protein (CRP), IL-6 and •

fibrinogen than patients without these pathogens. This mechanism would explain the •

worsening of glycemic control associated with severe periodontitis. Because type II •

diabetes is strongly associated with insulin resistance, periodontal therapy that •

reduces systemic inflammation may improve insulin sensitivity and result in •

improved glycemic control. Conversely, type I diabetes is not strongly associated •

with insulin resistance, so reduced inflammation after periodontal therapy may not •

have a major effect on insulin sensitivity in patients with type I diabetes, which •

would minimize the impact of periodontal treatment in these patients. •

Periodontal disease and pregnancy outcome: •

Infants born before the completion of 37 weeks of gestation are referred to as •
preterm infants. Preterm infants weigh usually lower at birth (< 2500 gm) and •
prematurity is associated with increased perinatal mortality. •
The most significant factor for preterm delivery is maternal infections attributing to •
about half of the preterm births. Bacteria from the maternal genital tract infections •
elicit a proinflammatory response in the mother, which ultimately results in release •
of prostaglandins and matrix metalloproteinases. This in turn causes smooth muscle •
contraction and membrane weakening respectively and triggers premature cervical •
ripening. This bacterial- host inflammatory response is considered to be the •
association between maternal periodontal disease and adverse pregnancy outcomes. •
In addition to the above pathway, bacteremia associated with periodontal disease •
may reach the uterus thus exposing the maternal-fetal unit to the bacteria and their •
products. This may elicit the above mentioned inflammatory response leading to •
preterm delivery. •

Periodontal infection and respiratory disorders: •

- The oral cavity plays an important role in infections acquired in hospitals and nursing homes, especially infections of the respiratory tract. Several studies have demonstrated that the teeth of patients in the intensive care unit (ICU) became
- colonized by respiratory pathogens such as *Pseudomonas aeruginosa*, *Enteric species* and *Staphylococcus aureus*. Similar studies have shown that the teeth of nursing home residents can also serve as reservoirs for respiratory infection. An
- association between oral conditions such as periodontal infections and respiratory conditions such as pneumonias and chronic obstructive pulmonary disease has beenfound. Evidence has suggested the oropharyngeal region as a likely source of
- bacteria implicated in respiratory infection. Oral periodontopathic bacteria can be aspirated into the lung to cause
- aspiration pneumonia. Respiratory pathogens have been shown to colonize the dental plaque of hospitalized intensive care and nursing home patients. Once established in the mouth, these pathogens can be aspirated into the lung to cause
- infection. Cytokines originating from periodontal tissues may change respiratory epithelium to promote infection by respiratory pathogens. A systematic review of the epidemiologic and clinical evidence found that poor periodontal health increases the risk of developing chronic obstructive pulmonary disease (COPD).

Chronic Obstructive Pulmonary Disease (COPD): •

Chronic obstructive pulmonary disease (COPD) is characterized by airflow • obstruction that results from chronic bronchitis or emphysema. Bronchial mucous • glands enlarge, and an inflammatory process occurs during which neutrophils and • mononuclear inflammatory cells accumulate within the lung tissue. • Periodontal Disease and Acute Respiratory Infections. COPD shares similar • pathogenic mechanisms with periodontal disease. With both diseases, a host • inflammatory response is mounted in response to chronic challenge by bacteria in • periodontal disease and by factors such as cigarette smoking in COPD. The resulting • neutrophil influx leads to the release of oxidative and hydrolytic enzymes that cause • tissue destruction directly. The recruitment of monocytes and macrophages leads to • further release of proinflammatory mediators •

Periodontal Disease and Acute Respiratory Infections: •

The upper respiratory passages are often contaminated with organisms derived from the oral, nasal, and pharyngeal • regions. Pneumonia is an infection of the lungs that is caused by bacteria, viruses, fungi, or mycoplasma and is broadly categorized as either community-acquired or hospital-acquired. A wide variety of bacteria can cause pneumonia, and the spectrum of offending organisms differs greatly between community-acquired and hospital-acquired infections. Communityacquired

bacterial pneumonia is caused primarily by the inhalation of infectious aerosols or the aspiration of oropharyngeal • organisms. *Streptococcus pneumoniae and Haemophilus influenzae* are the most common, although numerous other species

may be found, including anaerobic bacteria. Antibiotic therapy is highly successful for the resolution of most cases of • community-acquired bacterial pneumonia. To date, no associations have been found between oral hygiene or periodontal disease and the risk for acute respiratory conditions such as pneumonia in communitydwelling

individuals. The same cannot be said for individuals in the hospital setting. Hospital-acquired (nosocomial) bacterial • pneumonia has a very high morbidity and mortality rate. The incidence of nosocomial pneumonia is highest among severely ill patients, such as those in intensive care units or on ventilatory support. Although nosocomial pneumonia is most often caused by gram-negative aerobic organisms, many cases are the result of infection by anaerobic bacteria, including those that are typically found in the subgingival environment. Hospital-acquired pneumonia is

usually caused by the aspiration of oropharyngeal contents. •

Periodontal disease and asthma •

Asthma is a chronic disease of the airways characterized by inflammation and •

bronchoconstriction that occurs in people of all ages. Children with chronic • medical

disorders, like asthma, who require long-term medication have an increased •

susceptibility to dental diseases in three ways: frequent use of sugar containing •

syrups, use of sedatives causing a decreased saliva secretion, and use of •

corticosteroids. •

- The possible interactions between medications used for asthma and the induction of periodontal changes have also been positively correlated in the literature data from some studies suggest that inhalers can lead to changes in pH and a decrease in saliva production and therefore increase biofilm accumulation and calculus. In addition, the immunosuppressive effect of corticosteroids may have some influence on the response of the periodontal tissues. These agents act by inhibiting the host
- response, thus hampering the clinical expression of gingivitis. The association between periodontal disease and respiratory/lung diseases has
- been shown previously. Once installed, gingival diseases (Gingivitis / periodontitis)
- may contribute indirectly through recurrence or worsening of respiratory attacks.
- Some mechanisms for the interrelationship between diseases have been proposed,
- such as aspiration of biofilm and hematogenous dissemination or inflammatory
- chemical mediators from the periodontal pockets. Thus, treatment and maintenance
- of gingival health can improve pulmonary function and decrease the frequency of
- respiratory attacks.

Conclusion: •

There is sufficient evidence to suggest that periodontal • disease and systemic health have a two- way relationship, in that, the periodontal disease can cause adverse systemic conditions and that certain systemic diseases cause periodontal disease. It is vital that the physicians and other health care providers educate the

patients about this association and to recommend dental • care facilitate restoration of oral health in these individuals. The evidence suggests that treatment of one disease could lead to better outcomes for the other. This knowledge should be used to attain better patient outcomes in future.

Pathogenesis of periodontal disease

Inflammatory responses in the periodontium

م. سها أسود دهش العزاوي

B.D.S, MSc. Periodontology

Periodontal disease is initiated and sustained by factors (substances) produced by the subgingival microbiota (the biofilm). Some of these substances can directly injure host cells and tissues. Other microbial constituents may activate inflammatory or cellular and humoral immune systems that cause damage to the periodontal tissues. It is the latter pathway which accounts for most injury to the periodontal tissues.

Microbial virulence factors

- *Lipopolysaccharide*
- *Bacterial enzymes*
- *Microbial invasion*
- *Fimbriae*
- *Bacterial DNA*

Host-Derived Inflammatory Mediators

- *Cytokines*
- *Prostaglandins*
- *Matrix metalloproteinases*

Lipopolysaccharide

- large molecules composed of a lipid component (lipid A) and a polysaccharide component. They are found in the outer membrane of gram-negative bacteria (LPS is frequently referred to as endotoxin). They elicit strong immune responses in animals.
- TLR-4 recognizes LPS from gram-negative bacteria.
- LPS triggers a series of intracellular events, the net results of which are the increased production of inflammatory mediators (most notably cytokines) and the differentiation of immune cells (e.g., dendritic cells) for the development of effective immune responses against the pathogens. LPS is of key importance for initiating and sustaining inflammatory responses in the gingival and periodontal tissues.
- Porphyromonas gingivalis has an atypical form of LPS that is recognized by both TLR-2 and TLR-4.

Microbial invasion

The invasion process is considered a common strategy shared between various microbial pathogens and is thought to have evolved in order to access nutrients and shelter from host defences. P. gingivalis has the ability to bind and subsequently invade a range of eukaryotic cells other than oral epithelial cells, including fibroblasts, endothelial cells, and multiple cell lines, although it is not clear whether the mechanisms involved are the same for all cell types. The establishment of microbial species within an intracellular environment represents a challenging issue for extracellular and intracellular host defence mechanisms. The intracellular environment is not exposed to normal host immune defense mechanisms or many therapeutic agents, which represents a feature of a number of virulent organisms. It has been shown that Aggregatebacter actinomycetemcomitans can penetrate human epithelial cells by switching its morphological phenotype during the invasion process.

Enzymes and Noxious Products

Microorganisms produce a variety of soluble enzymes that may digest extracellular host proteins and other molecules and there by produce nutrients for bacterial growth. In addition to enzymes, bacteria also release numerous, harmful metabolic waste products, such as ammonia, indole, hydrogen sulfide, and butyric acid. Amongst the enzymes released by bacteria in the biofilm, proteases (proteinases) are capable of digesting collagen, elastin, fibronectin, fibrin, and various other components of the intercellular matrix of both epithelial and connective tissues

Proteinases (proteases)

Periodontal disease results in tissue degradation, and thus proteases, derived both from the host and from bacteria, are central to the disease processes. Proteinases (collagenase, elastase-like and trypsin-like, as well as serine and cysteine proteinases) cleave proteins by hydrolyzing peptide bonds and may be classified into two major classes, endopeptidases and exopeptidases, depending on the location of activity of the enzyme on its substrate.

Proteinase inhibitors

Release of proteinases in the gingiva and the crevicular area promotes inflammatory reactions and contributes to connective tissue damage. In contrast, proteinase inhibitors would dampen the inflammatory process. Among such inhibitors alpha-2 macroglobulin (A2-M) and alpha1 antitrypsin (A1-AT) must be recognized. In fact, gingival collagenase inhibition by A2-M has been demonstrated to occur in gingival tissues and polymorphonuclear leukocyte (PMN) collagenase is inhibited by A1-AT. Many host and microbial enzymes are likely to be present in the crevice at any one time. Realizing the potentially destructive features of such enzymes, consideration should be given to the source of these enzymes, their relative proportions and the inhibitory mechanisms available within the crevice. The main enzyme activity is host derived and specific and non-specific inhibitors are plentiful within the crevice and thus enzyme activity will be localized and short live

Matrix metalloproteinases (MMP)

Matrix metalloproteinases (MMPs) hydrolyses components of the extracellular matrix. These proteinases play a central role in many biological processes, such as embryogenesis, normal tissue remodeling, wound healing, and angiogenesis, and in diseases such as atheroma, arthritis, cancer, and tissue ulceration. Currently 28 MMP genes have been identified in humans. The periodontium is structurally comprised of fibrous elements, including collagen, elastin, and glycoproteins (laminin, fibronectin, proteoglycans), minerals, lipids, water, and tissue-bound growth factors. In addition there are variety of extracellular matrix components, including tropocollagen, proteoglycans, and other proteins (elastin, osteocalcin, osteopontin, bone sialoprotein, osteonectin, and tenascin). All of these matrix components are constantly in a state of turnover and thus there is much matrix enzyme activity in health, disease, and tissue repair and remodelling. It is evident that the activity of MMPs and their inhibitors is associated with tissue turnover as well as with gingivitis, destructive periodontitis and with the healing of the periodontal tissues following therapy.

The periodontal ligament is one of the most metabolically active tissues in the body, and collagen metabolism represents most of this activity. The biological reason for this activity probably relates to its ability to adapt to occlusal forces generated during function. An important feature of connective tissues in general and the periodontal ligament in particular, is the process of constant renewal of the extracellular matrix components involving MMP. The regulation of extracellular matrix (ECM) turnover is influenced by the action of both TNF-α and TGF-β, in that TNF-α can promote the ECM degradation by enhancing expression of MMPs. This action is balanced by TGF-β which down regulates the secretion of these MMPs and enhances the production of their inhibitors (tissue inhibitor of matrix metalloproteases (TIMPs)). Normal homeostasis is maintained when MMPs are in equilibrium with inhibitors, the TIMPs. However, TIMPs can be degraded by P. gingivalis which could contribute to such dysregulation.

Cytokines

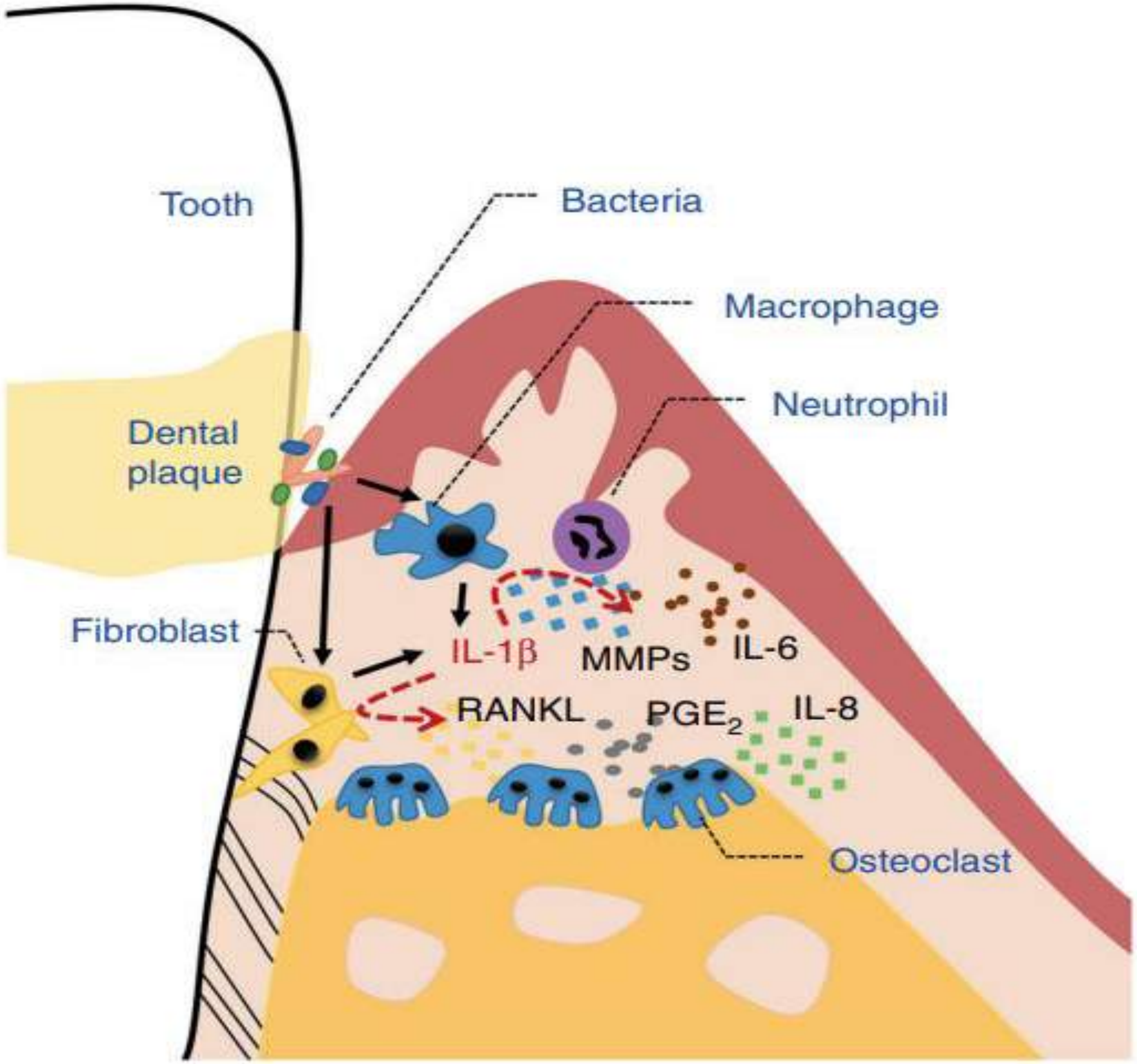
- Cytokines are soluble proteins, secreted by cells, which act as messenger molecules that transmit signals to other cells.
- They have numerous actions which include initiation and maintenance of immune and inflammatory responses and regulation of growth and differentiation of cells. The interleukins are important members of the cytokine group and are primarily involved in communication between leukocytes and other cells, such as epithelia, endothelia and fibroblasts, involved in both immune and inflammatory processes. These molecules are released in small amounts and have a variety of actions on cells which carry the specific receptor for the particular interleukin. Cytokines are numerous, many have overlapping functions and they are interlinked forming an active network which controls the host response.

Pro-inflammatory cytokines:

Cytokines such as interleukin (IL)-1α, IL-lβ and tumour necrosis factor (TNF)-α stimulate bone resorption and inhibit bone formation.

Chemotactic cytokines:

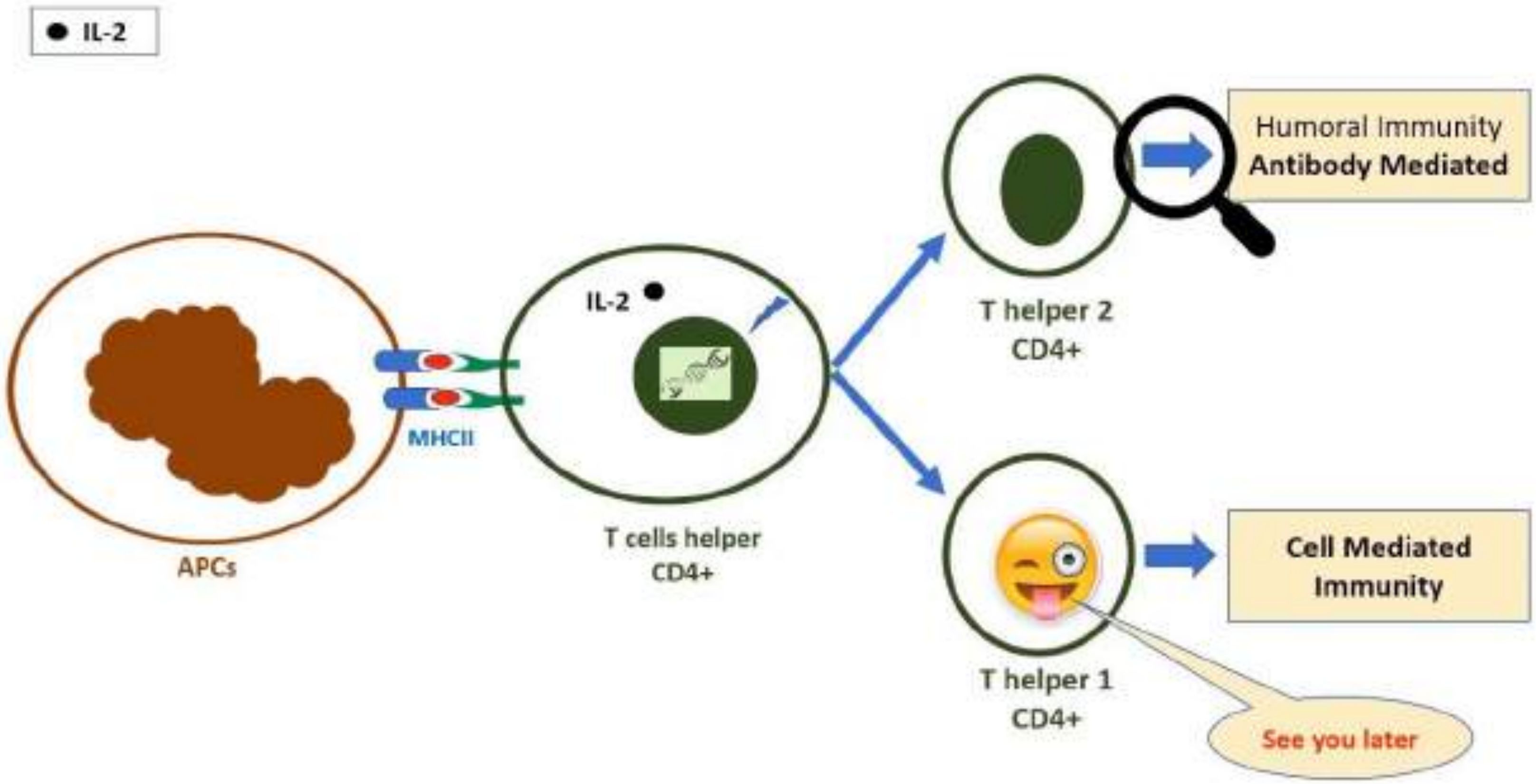
A series of more than 20 molecules have been identified, among which the most famous and best characterized is interleukin 8 (IL-8), which has powerful chemotactic functions for leukocytes particulary for neutrophils but also for lymphocytes and macrophages.



Role of IL-1β in periodontitis. IL-1β promotes the secretion of MMPs, RANKL, PGE2, IL-6, IL-8, etc., which promotes osteoclastogenesis

Lymphocyte signaling cytokines:

T helper cells are lymphocytes within the tissues which regulate both the humoral and cell mediated immune responses via cytokines. The humoral immune response is promoted by a T helper cell type 2 (TH-2) which produces characteristic cytokines namely IL-5, IL-10 and IL-13. The TH-1 lymphocytes release IL-2 and interferon (IFN)-γ which enhance cell mediated responses. These cytokines provide a precise mechanism for the control of the immune response so that a sufficient response is produced to deal with the offending pathogen. Cytokines can influence the immune response through determining the class of immunoglobulin being produced, which may have quite a profound effect on antibody function.


IL-2
IL-2
MHCII
APCs
T cells helper
CD4+
T helper 2
CD4+
Humoral Immunity
Antibody Mediated
T helper 1
CD4+
Cell Mediated
Immunity
See you later

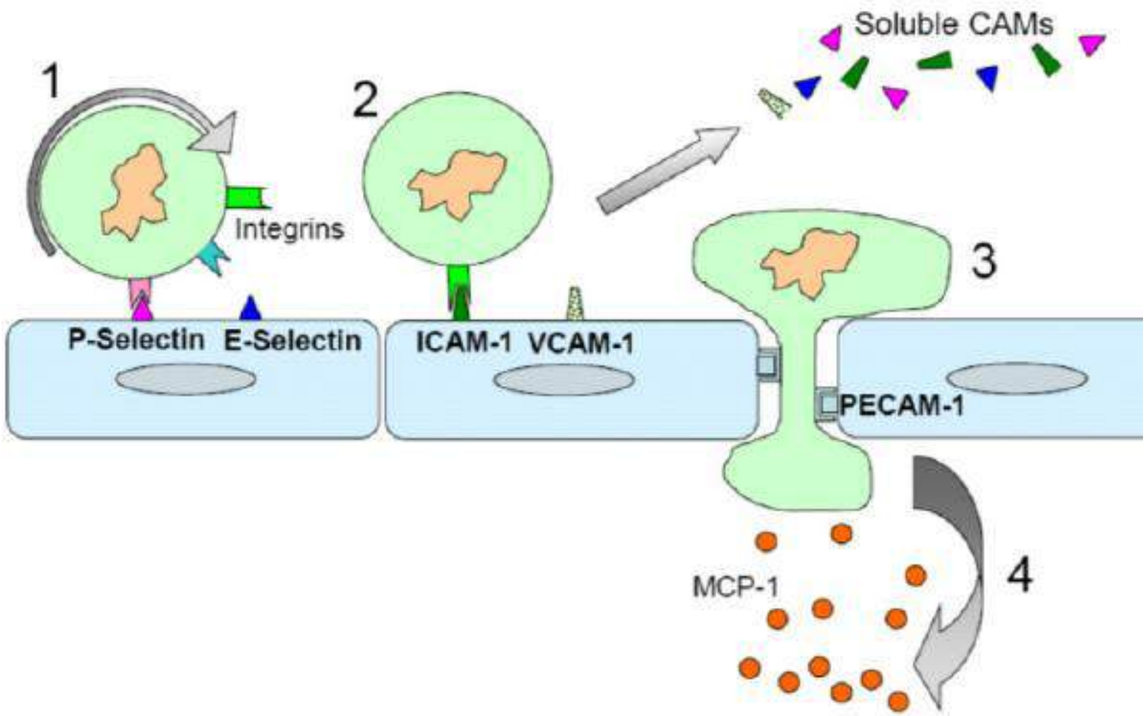
For example IgM molecules are more effective at bacteriolysis and IgG molecules are more effective at opsonization. The IgG antibodies exist as four distinct subclasses (IgG1, IgG2, IgG3 and IgG4) based on differences in the Fc portion of these molecules. The antibody subclass influences antibody function, IgG2 being less strong in binding antigen than IgG1. Several researchers have found IgG2 to be elevated over IgG1 in patients with severe periodontitis and propose that IgG subclass levels are important factors in susceptibility to periodontitis.

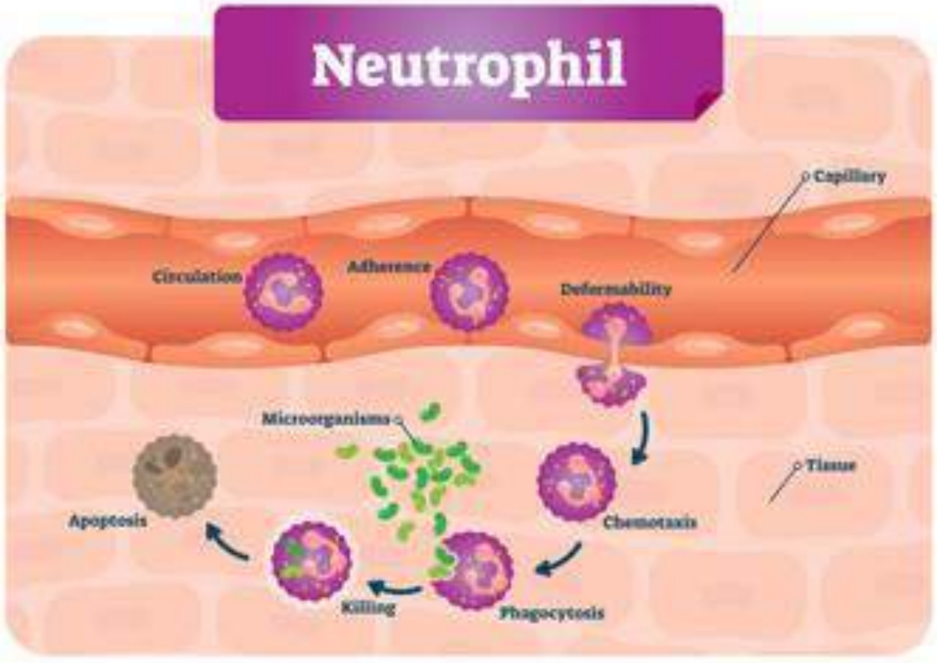
Prostaglandins

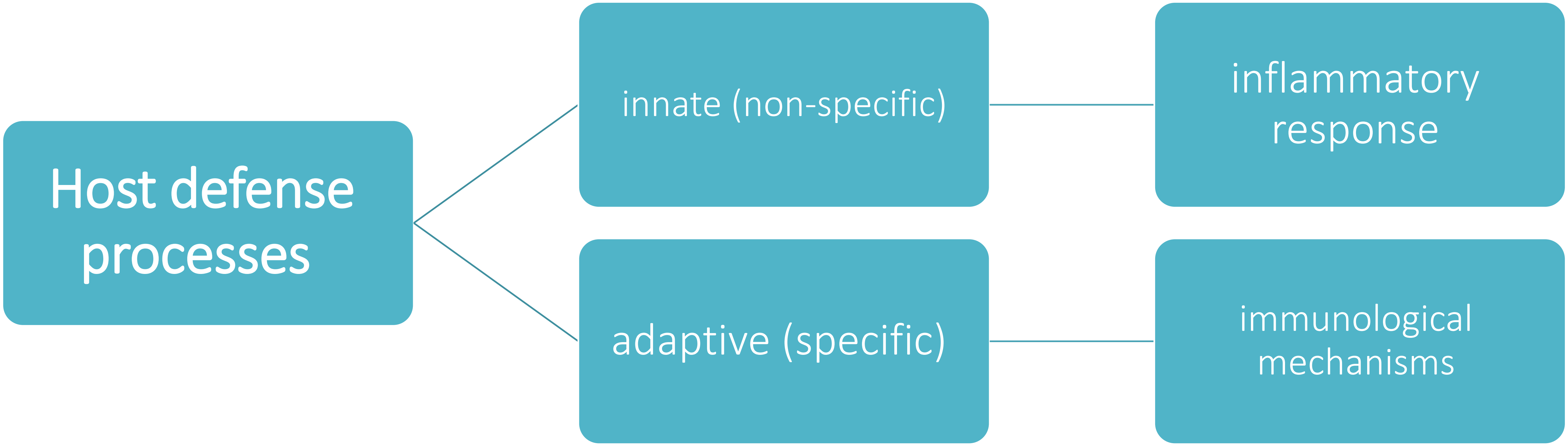
Prostaglandins are arachidonic acid derivatives which are important mediators of inflammation. The pro-inflammatory cytokines are capable of inducing macrophages and other cells to produce copious amounts of prostaglandins, particularly PGE2 which are potent vasodilators and inducers of cytokine production by various cells. PGE2 acts on fibroblasts and osteoclasts, together with cytokines, to induce MMP production, which is relevant to tissue turnover and in the destructive process in periodontitis. Many studies have examined the association of PGE2 with periodontal disease and suggest that its concentration in gingival crevicular fluid increases in gingivitis relative to health and is at very high concentrations during periods of periodontal disease progression.

Polymorph nuclear leukocytes (PMNs)

The PMN is the predominant leukocyte within the gingival crevice/pocket in both health and disease. PMNs from the circulation are attracted to the area via chemotactic stimuli elicited from microorganisms in the biofilm, and histologically PMNs can be seen traversing the gingival connective tissue in inflammation. Migration of leukocytes from the vessels into the gingival connective tissue, and through the junctional epithelium into the gingival crevice, is controlled via adhesion molecules. Cellular migration involves three main structures: the endothelial cells, the cell adhesion molecules and the extravasating cells. Adhesion of leukocytes appears to be essential in controlling cellular traffic into inflamed areas and it has been proposed that cytokines may play an important role in regulation of this traffic


Soluble CAMs
1
2
3
4
Integrins
P-Selectin
E-Selectin
ICAM-1
VCAM-1
PECAM-1
MCP-1


Neutrophil
Capillary
Circulation
Adherence
Deformability
Microorganisms
Tissue
Apoptosis
Chemotaxis
Killing
Phagocytosis


Host defense processes
innate (non-specific)
adaptive (specific)
inflammatory response
immunological mechanisms

THE INNATE DEFENSE SYSTEMS

Innate immune mechanisms operate without any previous contact with the disease causing microorganism. These mechanisms include physical barriers of the oral mucosal epithelial surfaces, vascular and cellular aspects of the inflammatory responses. The epithelial surface is the first region of the periodontium which comes into contact with and responds to bacteria attaching and colonizing the dento- gingival region. Prevention of attachment and colonization is important for the host defenses. The oral mucosa itself is not simply a barrier but has a chemical composition which may be detrimental to bacteria. Furthermore, the cells of the epithelium can respond to the bacteria by (1) producing and/or releasing cytokines and other molecules that kill the microbes and (2) releasing other molecules (such as IL-1) capable of inducing or enhancing the inflammatory reaction. The epithelium can also respond by increasing expression of surface molecules such as cell adhesion molecules which can function with cytokines and chemo attractants to bring leukocytes to the region.

The major functions of the innate immune system include:

- Acting as a physical and chemical barrier to infectious agents.
- Recruiting immune cells to sites of infection, through the production of chemical factors, including specialized chemical mediators, called cytokines
- Activation of the adaptive immune system through a process known as antigen presentation.
- Activation of the complement cascade to identify bacteria, activate cells, and promote clearance of antibody complexes or dead cells.
- Identification and removal of foreign substances present in organs, tissues, the blood and lymph, by specialized white blood cell.

Adaptive immune system

The adaptive immune system, also known as the acquired immune system or, as the specific immune system, is a subsystem of the overall immune system that is composed of highly specialized, systemic cells and processes that eliminate or prevent pathogen growth.

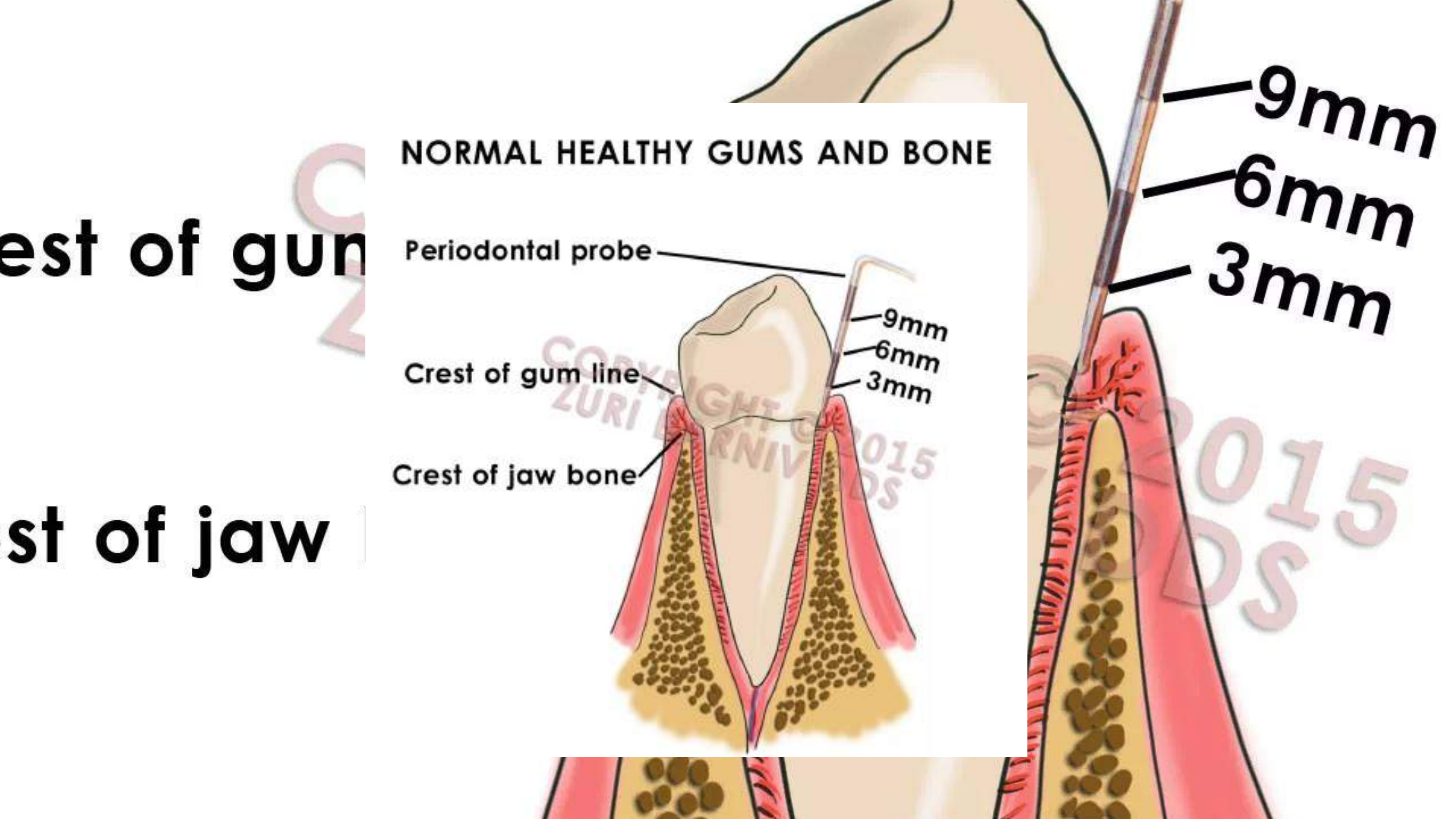
Adaptive immunity creates immunological memory after an initial response to a specific pathogen, and leads to an enhanced response to subsequent encounters with that pathogen.

Unlike the innate immune system, the adaptive immune system is highly specific to a particular pathogen.

Functions

1. Recognition of specific antigens during the process of antigen presentation.
2. Generation of responses that are tailored to maximally eliminate specific pathogens or pathogen-infected cells.
3. Development of immunological memory, in which pathogens are "remembered" through memory B cells and memory T cell

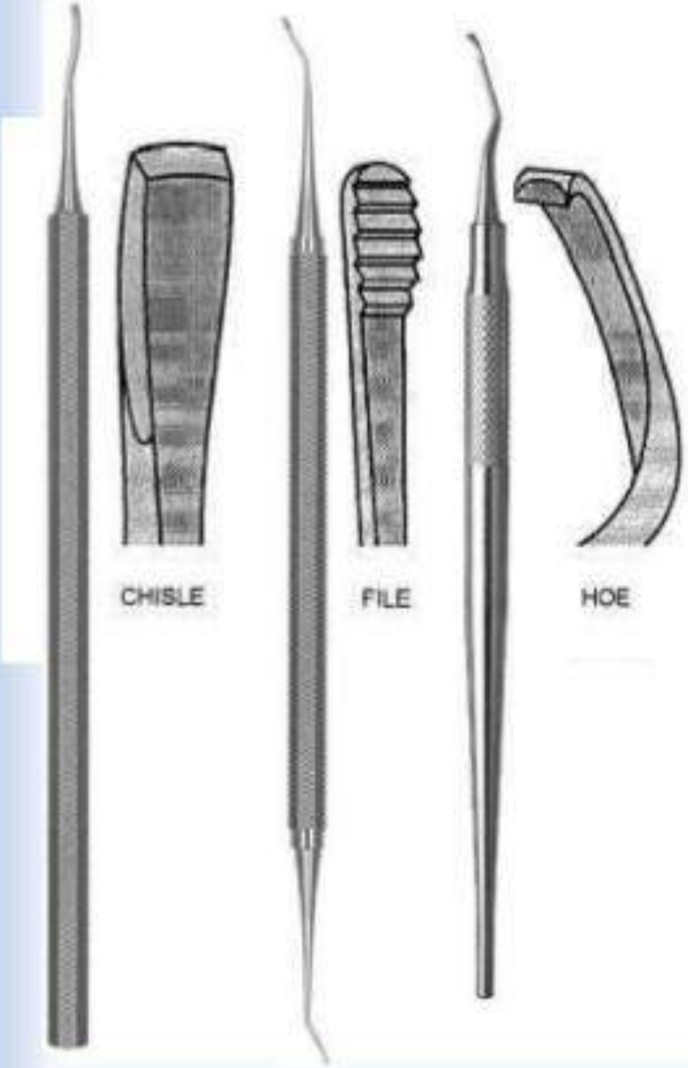
Thank you



NORMAL HEALTHY GUMS AND BONE
Periodontal probe
9mm
6mm
3mm
Crest of gum line
Crest of jaw bone
9mm
6mm
3mm


CORSODYL
Spray
chlorhexidine digluconate
ALCOHOL F
MINT FLAV
CORSO
0.2% MOUTHW
CHLORHEXIDINE DIG
CORSODYL

SUBGINGIVAL SCALERS



Chisel scaler

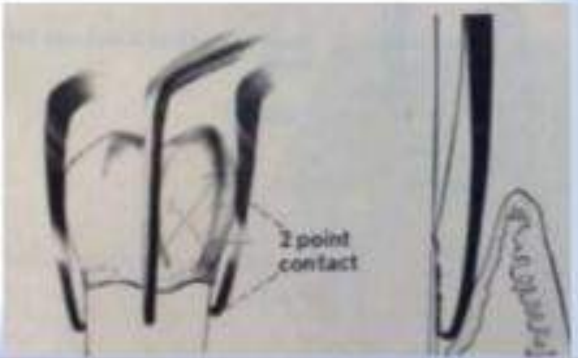
Used in interproximal area
Used in a push motion

File

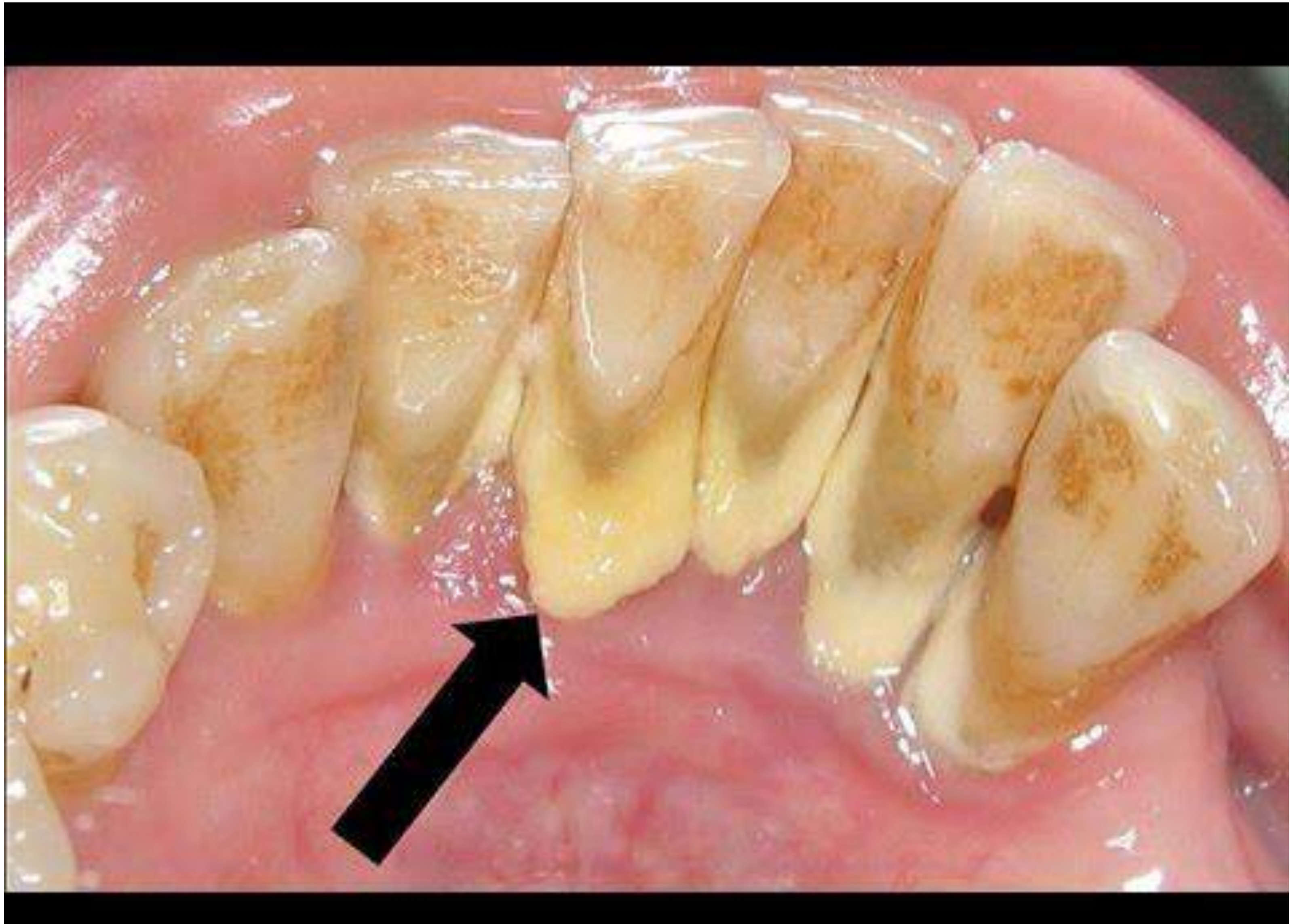
Used to crush large pieces of calculus deposits.

Hoe

Efficient in removing subgingival calculus
Blade is beveled at 45 degrees
Working end is bent at an angle of 99 degrees to shank.




Ultrasonic Scaler
eshopbid666



SCALING, ROOT-PLANING AND CURETTAGE INSTRUMENTS

A. **Curette :** for sub gingival scaling, root planning and removal of the soft tissue lining the pocket

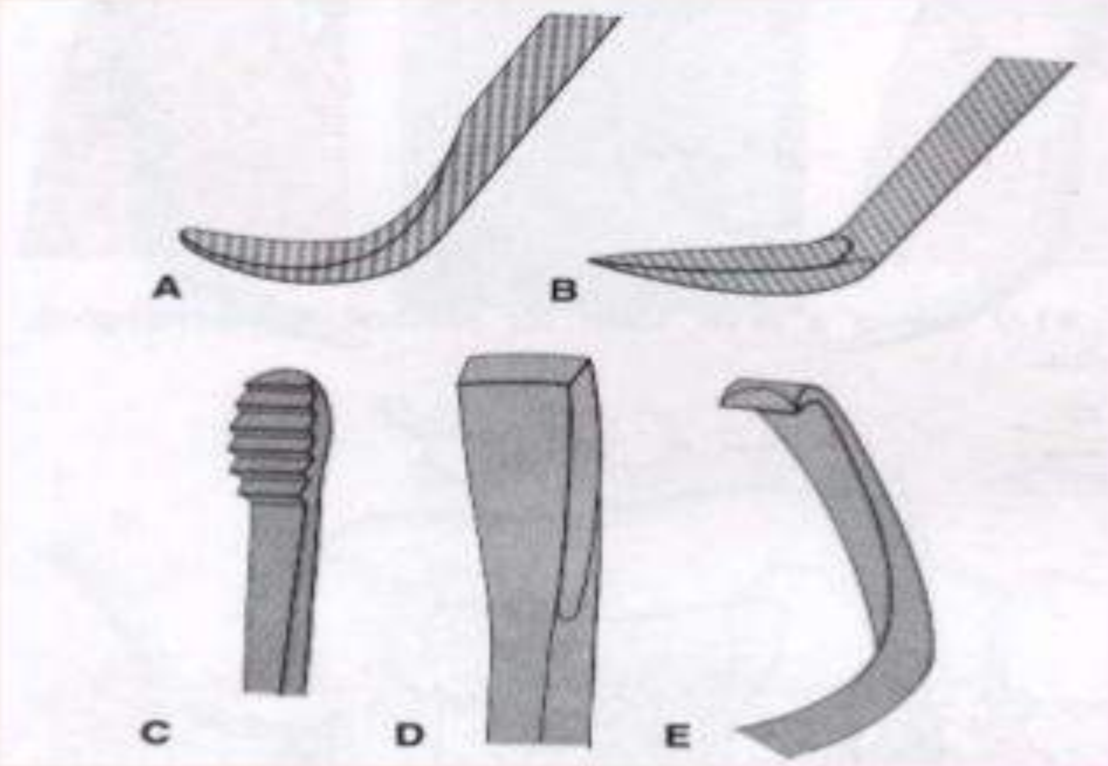
B. **Sickle Scaler :** to remove supra gingival calculus

C. **File**

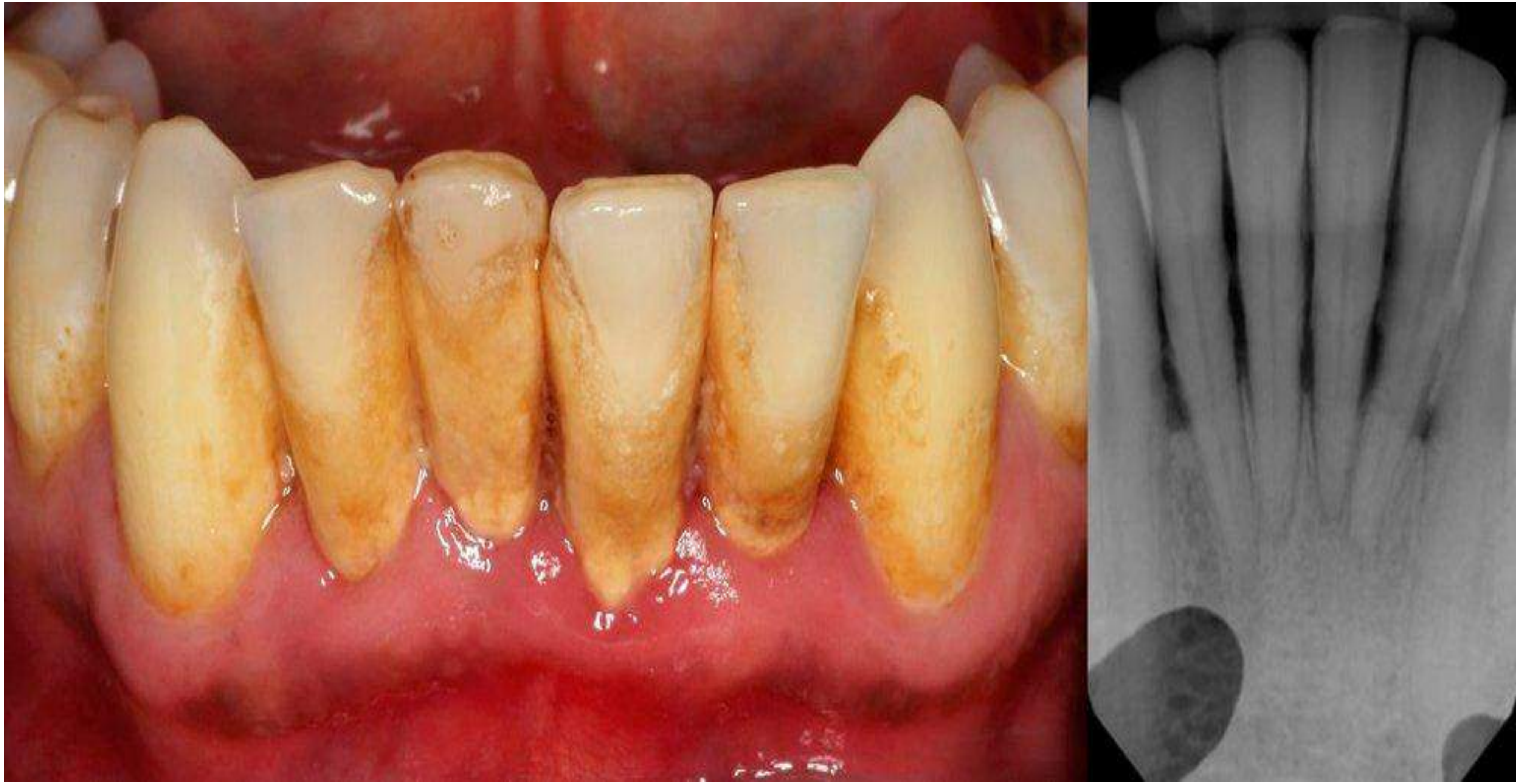
D. **Chisel**

E. **Hoe**

to remove tenacious calculus and altered cementum




BEFORE
AFTER



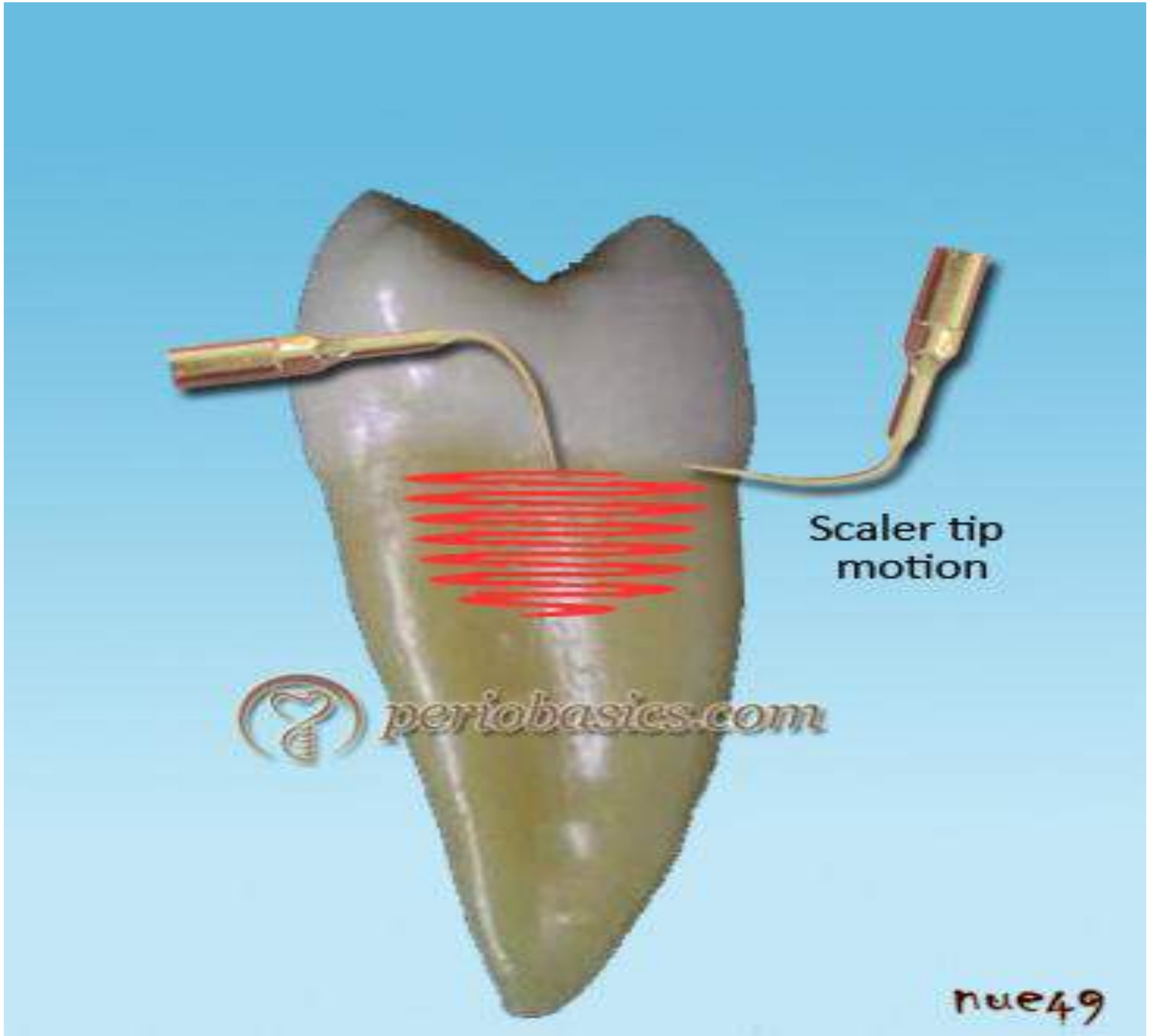

SPEAR
Procedure: SCALING ROOT PLANING

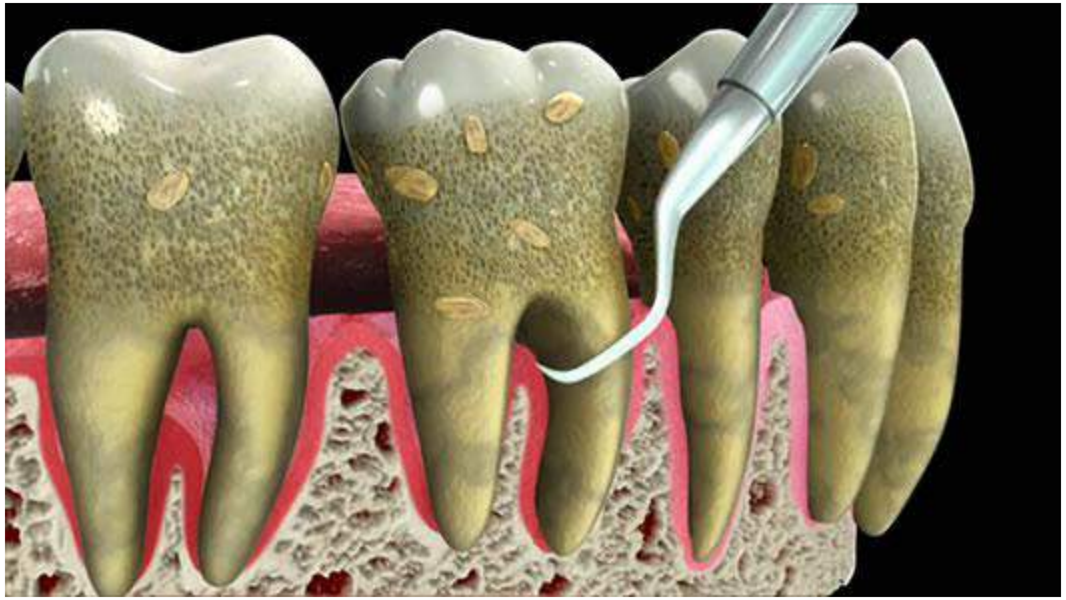

PUMICE POWDER


1:1

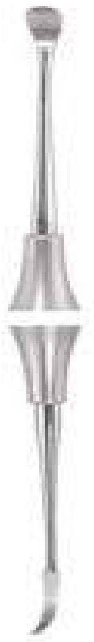
SELECT
300
Motor Running
Micro Motor
MAX


Snap-on
Screw type
Latch type
dentp.en.alibaba.com
Tapered
Flat
Cup-shape


Scaler tip
motion
periobasics.com
nue49




Double Ended Handle #3
Sickle Scaler




AR
AR INSTRUMED

PeriodonticsInstruments

Explorers

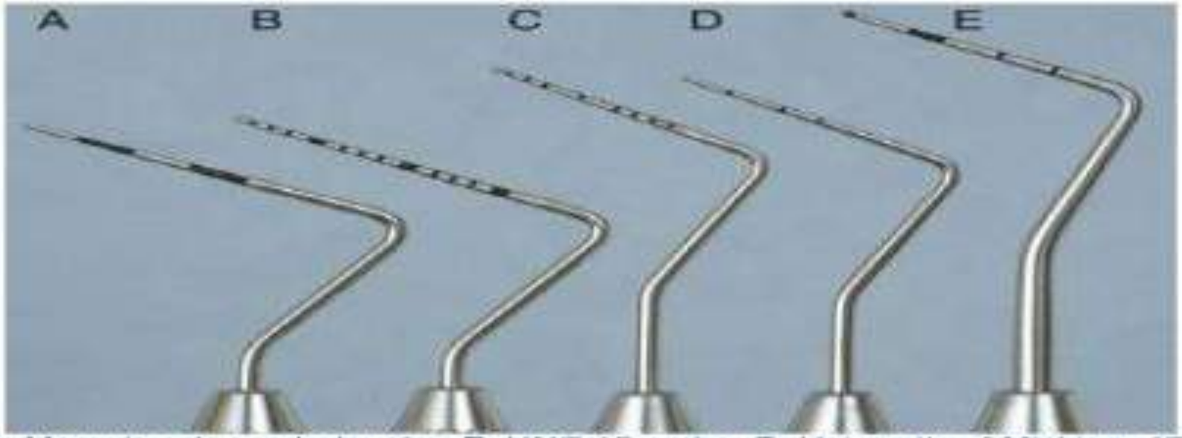
- Use: 1.**Locate Sub-gingival Deposits and Caries.**
  2.**Check Smoothness of root surface after planning.**
  3.**Assess Restorative problems.**

- Thin, Flexible, Wire-like working end. Taper to sharp point.
- Curved, Right-angled & Area specific.

Periodontal Probes

- Use: 1.**Locate &Measure depth of pocket and determine it's configuration.**
  2. **Assess Loss of Attachment.**
  3. **Detect Sub-gingival Deposits.**

- Tapered with blunt round tip, mm markings for accuracy.
- Ball-end to avoid penetration into junctional epithelium.
- Diameter less than or equal 0.6 mm.
- Probing Force more than 0.25 N traumatize healthy tissue. (25-50g for clinic)
- Ball-end to avoid penetration into junctional epithelium.



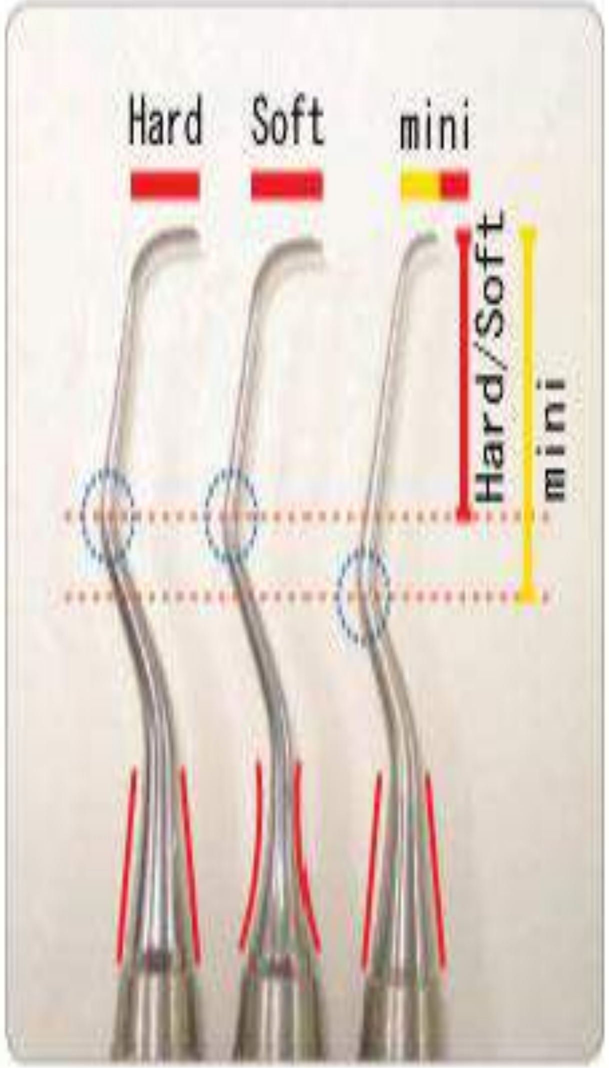
A, Marquis color-coded probe. **B,** UNC-15 probe. **C,** University of Michigan "O" probe, with Williams markings. **D,** Michigan "O" probe with markings at 3, 6, and 8 mm. **E,** World Health Organization (WHO) probe.

Hoe scaler

HSA12-13

· Anterior Hoe Scaler

For anterior buccal and lingual surfaces.



Hard
Soft
mini
Hard/Soft
mini

#G11-12
#G15-16

Microbiologic Specificity of Periodontal Diseases

م. سها أسود دهش العزاوي
B.D.S, MSc. Periodontology

1. Traditional Nonspecific Plaque Hypothesis.
2. Specific Plaque Hypothesis.
3. Updated Nonspecific Plaque Hypothesis.
4. Ecologic Plaque Hypothesis.
5. Keystone Pathogen Hypothesis.

Traditional Nonspecific Plaque Hypothesis

- Periodontal noxious products by the entire plaque flora are responsible in a proportional way to the severity of the gingival inflammation.
- According to this thinking, when only small amounts of plaque are present, the noxious products are neutralized by the host. Similarly, large amounts of plaque would produce large amounts of noxious products, which would essentially overwhelm the host's defenses. The NSPH have focused the quantity of plaque that determined the pathogenicity without discriminating between the levels of virulence of bacteria

- Believing this, the host would have a threshold capacity to detoxify bacterial products (e.g., saliva neutralizing acid) and disease would only develop if this threshold was surpassed and the virulence factors could no longer be neutralized.
- **The conclusion** was that if any plaque has an equal potential to cause disease, the best way of disease prevention would be non-specific mechanical removal of as much plaque as possible by e.g., tooth brushing

- **Several** observations contradicted these conclusions. **First**, some individuals with considerable amounts of plaque and calculus, as well as gingivitis, never developed destructive periodontitis. **Furthermore,** individuals who did present with periodontitis demonstrated **considerable site specificity in the pattern of disease**. Some sites were unaffected, whereas advanced disease was found in adjacent sites. In the presence of a uniform host response, these findings were inconsistent with the concept that all plaque was equally pathogenic. Recognition of the differences in plaque at sites of different clinical status (i.e., disease versus health) led to a renewed search for specific pathogens in periodontal diseases and a conceptual transition from the nonspecific to the specific plaque hypothesis.

- **In addition,** the improvement of techniques to isolate and identify bacteria led to the abandoning of the NSPH. Although the nonspecific plaque hypothesis has been discarded in favor of the specific plaque hypothesis or the ecologic plaque hypothesis, much clinical treatment is still based on the nonspecific plaque hypothesis through mechanical plaque removal that represents the most efficient way of preventing disease.

Specific Plaque Hypothesis

- The specific plaque hypothesis states that **only certain plaque is pathogenic, and its pathogenicity depends on the presence of or increase in specific microorganisms.** This concept predicts that plaque harboring specific bacterial pathogens results in a periodontal disease because these organisms produce substances that mediate the destruction of host tissues. Acceptance of the specific plaque hypothesis was spurred by the recognition of ***A. actinomycetemcomitans*** as a pathogen in localized aggressive periodontitis.

Specific Plaque Hypothesis

- In the 1970s, culture-based techniques and microscopy allowed discrimination of specific bacterial species and opened the hunt for disease-related micro-organisms.
- It was noticed that the antibiotic kanamycin was particularly effective against cariogenic species such as oral streptococci and reduced caries formation. This suggested that removing cariogenic bacteria from the oral cavity using antibiotics could prevent caries. In 1976, Walter J. Loesche announced the "Specific Plaque Hypothesis" (SPH), postulating that dental caries was an infection with specific bacteria in the dental plaque of which the most relevant were "mutans streptococci" (main species: *Streptococcus mutans* and *Streptococcus sobrinus*) and *lactobacilli.*

- This hypothesis proposed that use of antibiotics against specific bacterial species could cure and prevent caries.

- However, results from clinical studies, then and today, are not very promising. For instance, even though the use of kanamycin resulted in an overall reduction of caries, at some surfaces the caries rate increased. This indicates that kanamycin failed to penetrate certain niches allowing cariogenic species to have a selective advantage and accumulate there. Furthermore antibiotics reduced the abundance of cariogenic bacteria but failed to eliminate them thus as soon as the treatment was stopped, abundance increased, while a long period of treatment leads to antibiotic resistance.

- The development of the anaerobic hood in the 1970s for the first time allowed cultivation of the strict anaerobic species. This extended the SPH to periodontal diseases which were proposed to be inflammations caused by specific periopathogens and antibiotic treatment would be effective. However, in line with the use of antibiotics in caries treatment,
- Recent clinical studies evaluating the effectiveness of antibiotics as adjunct in periodontal therapy have not booked significant success either.
- Potential periopathogens included: spirochetes, streptococci, and actinomyces. In addition, Gram-negative, anaerobic rods including black-pigmented *Bacteriodes* such as *Prevotela melaninogenica* and *Campylobacter* and facultative anaerobic, Gram-negative rods of the genera *Capnocytophaga, Eikenella* and *Actinobacillus* were identified as periopathogens. However, these findings were limited due to the large number of uncultivable species (∼50%) and the bias toward easily cultivable species.

- The finding of different species related to periodontal disease led to the idea that oral disease could be initiated by a number of specific pathogens. This idea was further investigated over the next decades and led to the famous Socransky-complexes which include bacterial clusters based on their association with periodontal disease.

Updated Nonspecific Plaque Hypothesis

- Theilade also noticed that the “specific-pathogens” from the SPH were indigenous bacteria and sometimes common bacteria in health, which led to an updated NSPH in 1986 focusing on periodontal disease. At this time most researchers seemed to agree that gingivitis was a non- specific inflammatory reaction to a complex indigenous microbiota. However, the updated NSPH took into consideration that some indigenous subgingival bacteria can be more virulent than others and that plaque composition changes from health to disease.

- Nevertheless, it stated that all bacteria in plaque contribute to the virulence of the microflora by having a role in either colonization, evasion of the defense mechanism, and/or provocation of inflammation and tissue destruction. Theilade's statement that "any microbial colonization of sufficient quantity in the gingival crevice causes at least gingivitis" **was supported by the fact that a non-pathogenic plaque (i.e., not causing gingivitis in the absence of oral hygiene) had never been observed.**
- Additionally, it was considered that some people have gingivitis for a lifetime without tissue and bone destruction, while others encounter rapid progression into periodontitis. Unlike the classic NSPH, the updated NSPH could explain this by taking into account that differences in the plaque microbial composition could lead to differences in pathogenic potential.

Ecologic Plaque Hypothesis

- In 1994 Philip D. Marsh proposed a hypothesis that combined key concepts of the earlier hypotheses. In his “Ecological Plaque Hypothesis” (EPH), disease is the result of an imbalance in the total microflora due to ecological stress, resulting in an enrichment of some “oral pathogens” or disease-related micro-organisms. This idea was not entirely new since Theilade , in the review proposing the U-NSPH concluded that “increased virulence of plaque (leading to disease) is due to a plaque ecology unfavorable to the host and favorable for overgrowth by some of the indigenous bacteria having a pathogenic potential”.
- Importantly, **Marsh expanded this theory** and related the changes in microbial composition to changes in ecological factors such as the presence of nutrients and essential cofactors, pH and redox potential (Marsh, 1994, 2003).

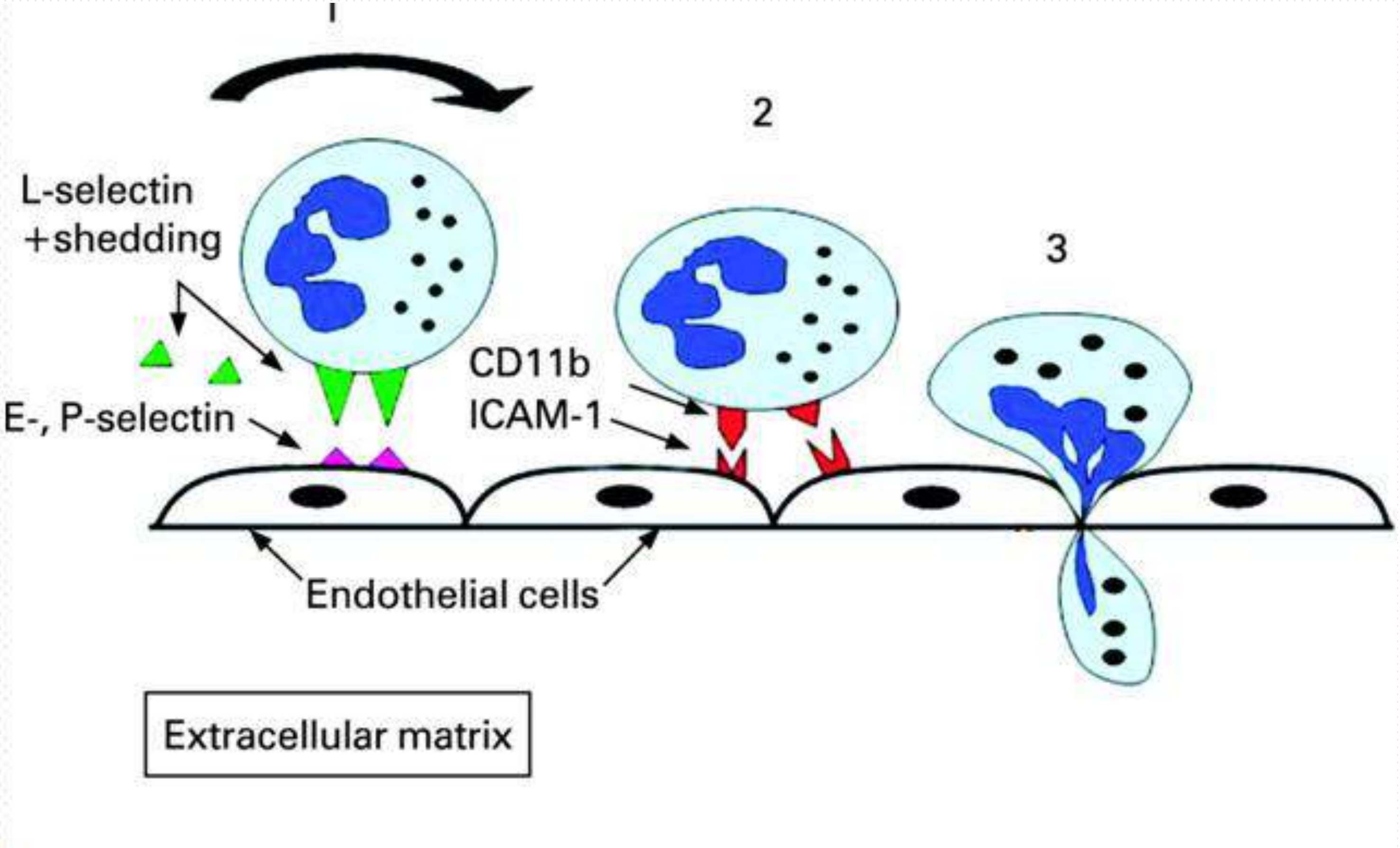
- For example, frequent exposure to a low pH, for instance as the result of sugar fermentation, leads to a relative increase of acid-tolerant species. The thought arose that disease could be prevented by interfering with processes that break down homeostasis and change composition. For example, non-fermentable sweeteners could be used to replace sugar and thus prevent acidification.
- Thus, the classical "everything is everywhere, but, the environment selects" was successfully applied to dentistry.
- Marsh also considered the reverse: the bacteria in dental plaque affect the environment. For instance, early colonizers of supragingival dental surfaces, are usually facultative anaerobic bacteria that use the oxygen, producing carbon dioxide and hydrogen. This lowers the redox potential giving strict anaerobes a chance to settle and multiply in the biofilm.

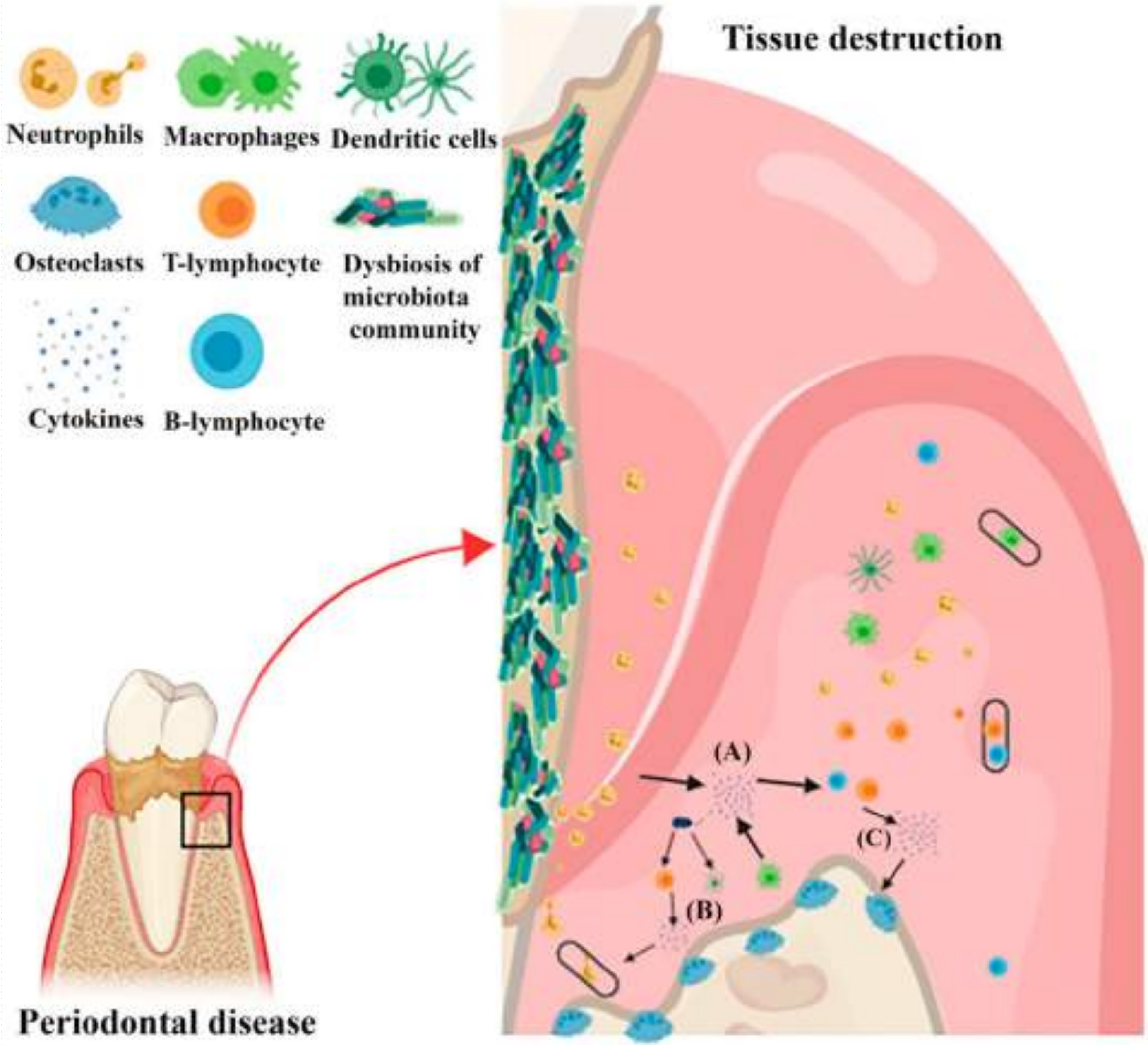
Keystone Pathogen Hypothesis

- Certain species have an effect on their environment that is disproportional relative to their overall abundance.
- The Keystone-Pathogen Hypothesis" (KPH) indicates that certain low-abundance microbial pathogens can cause inflammatory disease by increasing the quantity of the normal microbiota and by changing its composition. For instance, *Porphyromonas gingivalis* is shown to be able to **manipulate the native immune system** of the host. By doing so it was hypothesized that it does not only facilitate its own survival and multiplication, but of the entire microbial community.

- In contrast to dominant species that can influence inflammation by their abundant presence, **keystone pathogens can trigger inflammation when they are present in low numbers**. When disease develops and advanced stages are reached, the keystone pathogen are detected in higher numbers. Importantly, even though their absolute number increases, keystone pathogens can decrease in levels compared to the total bacterial load which increases as plaque accumulates in periodontitis.
- The KPH was developed by observing the properties of the "red complex" bacterium *P. gingivalis*.
- Studies in mouse models showed that very low presence of *P. gingivalis* (<0.01% of the total bacterial count in plaque) could alter the plaque composition, leading to periodontitis. In germ-free mice, *P. gingivalis* was able to colonize by itself, but was not able to trigger disease without the presence of other bacterial species. This indicates that (some of) the commensal microbiota is essential in the disease process.

- The role of the host-immune system is critical in the KPH. At health, periodontal tissue contains a wall of neutrophils, between the plaque and the epithelial surface, residing just outside the epithelial cells. Expression of mediators such as interleukin 8 (IL-8), intercellular adhesion molecule (ICAM) and E-selectin is required to form this neutrophil wall. E-selectin is required for neutrophil migration from the highly vascularized gingival tissue, IL-8 is a key neutrophil chemo- attractant produced by epithelial cells, and ICAM facilitates adhesion of neutrophils to the tissue allowing formation of this wall. Furthermore, the epithelium expresses low levels of a wide range of toll-like receptors (TLR's), including TLR1-TLR9 that mediate the response to a broad range of microorganisms.
- The array of different TLRs in combination with the multitude of bacterial species lead to a large variety of cytokines that are expressed at health.


1
2
3
L-selectin
+shedding
E-, P-selectin
CD11b
ICAM-1
Endothelial cells
Extracellular matrix



Neutrophils
Macrophages
Dendritic cells
Osteoclasts
T-lymphocyte
Dysbiosis of microbiota community
Cytokines
B-lymphocyte
Tissue destruction
(A)
(B)
(C)
Periodontal disease

- Studies in germ-free mice show that there are low levels of innate host mediators, such as IL-1B, present in the periodontal tissue. This indicates that a basic level of cytokine expression is genetically programmed without bacterial challenge. The composition and amount of bacteria in plaque modifies cytokine expression further.

- Evidence was found of three major KPH mechanisms of *P. gingivalis* that could impair the above mentioned host defenses:
- (1) Toll-like receptor (TLR) response manipulation,
- (2) interleukin 8 (IL-8) subversion and
- (3) the corruption of the complement system.

- *In vitro*, the TLR response is manipulated by *P. gingivalis* with the help of **two types of lipopolysaccharides (LPS)** with different lipidA structures Pg LPS (**type I**) and Pg LPS (**type II**). Type I is a TLR4 agonist thus activating the immune system, while Type II is aTLR4 antagonist inhibiting the immune response to *P. gingivalis*. The concentration of iron determines which type of LPS is expressed. In the oral cavity, the main source of iron is hemin, found in the gingival crevicular fluid (GCF). During inflammatory process, GCF increases stimulating *P. gingivalis* type II LPS expression, thus reduces the TLR4 response. It was proposed that this could facilitate survival and multiplication of the entire microbial community.

- *Porphyromonas gingivalis* can block production of IL-8 *in vitro*, which is produced by gingival epithelial cells in response to other bacteria, by secreting a serine phosphatase that inhibits the synthesis of IL-8. This process is called “local chemokine paralysis” and delays the recruitment of neutrophils preventing proper neutrophil wall formation, of which was proposed that it could facilitate initial microbial colonization of the periodontium. Other “red complex” bacteria such as *T. denticola*, are also able to manipulate the IL-8 response of the host.

- The third and best *in vivo* documented key stone pathogen mechanism is the interference with the complement system.
- To be a successful pathogen in humans (and any other mammal) a microorganism needs to be able to avoid complement-mediated detection and killing. Again, the best-studied example in the oral cavity is *P. gingivalis* that produces membrane bound and soluble arginine-specific cysteine proteinases called "gingipains". Gingipains can cleave complement factors C3 and C5 into active fragments C5a (cell activator) and C3b (phagocytosis enhancer). These fragments can be further degraded by gingipains resulting in loss of their function. More relevant is that in the presence of gingipains the levels of the inflammatory mediator C5a increase within seconds. This leads to an increased activation of the C5a receptor (C5aR) on leukocytes.

- C5aR is involved in cross talk with TLR2, which is activated in parallel by *P. gingivalis* (and other bacterial) surface ligands. While this crosstalk leads to increased inflammation, it impairs the killing capacity for leukocytes. In mouse models this mechanism has a major role in accelerating periodontitis development and bone loss. A *P. gingivalis* strain that lacks gingipains failed to change the oral microbiota and induce bone loss. Additionally, periodontitis did not develop in mice lacking one of the two involved receptors C5aR or TLR2. This provides clear evidence that in mice the dysbiosis caused by *P. gingivalis* is mainly due to complement subversion.

- In conclusion, it was proposed that currently known and unknown keystone pathogens use a combination of these and presently unknown mechanisms to manipulate the innate defense system leading to destructive periodontitis.

Reference

- Microbiologic Specificity of Periodontal Diseases lecture(4th stage)/University of Baghdad/College of Dentistry/Department of Periodontology.

Thank you

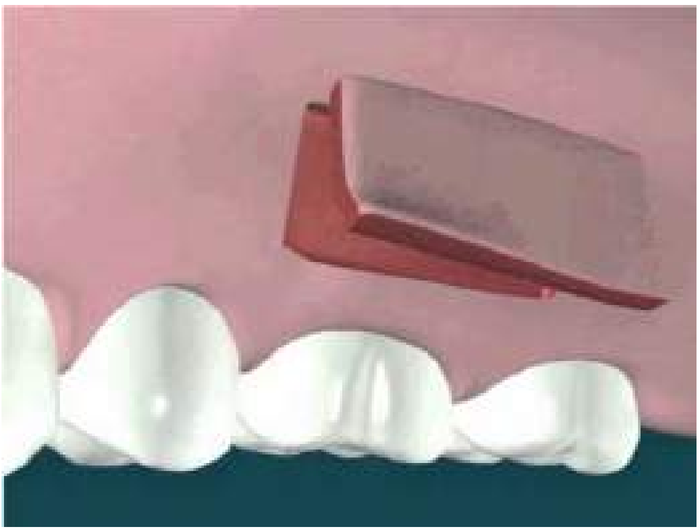
Periodontics

Mucogingival surgery

Periodontal treatment involving procedures for correction of defects in morphology,position and/or the amount of soft tissue (gingiva and alveolar mucosa) and underlying bone support at teeth and implants.
These procedures are varied from simple *gingivectomies or *crown lengthening procedures (e.g. To increase the clinical crown length if there is a gummy smile with
a high lip line), to complex gingival grafting procedures. In patients with bone defects *GTR and *bone grafting (Guided bone regeneration, GBR) may also be employed to increase the bulk of available alveolar bone, grafting procedures generally aim Io cover exposed roots, to increase the bulk of the width of keratinized gingiva and to prevent further gingival recession.

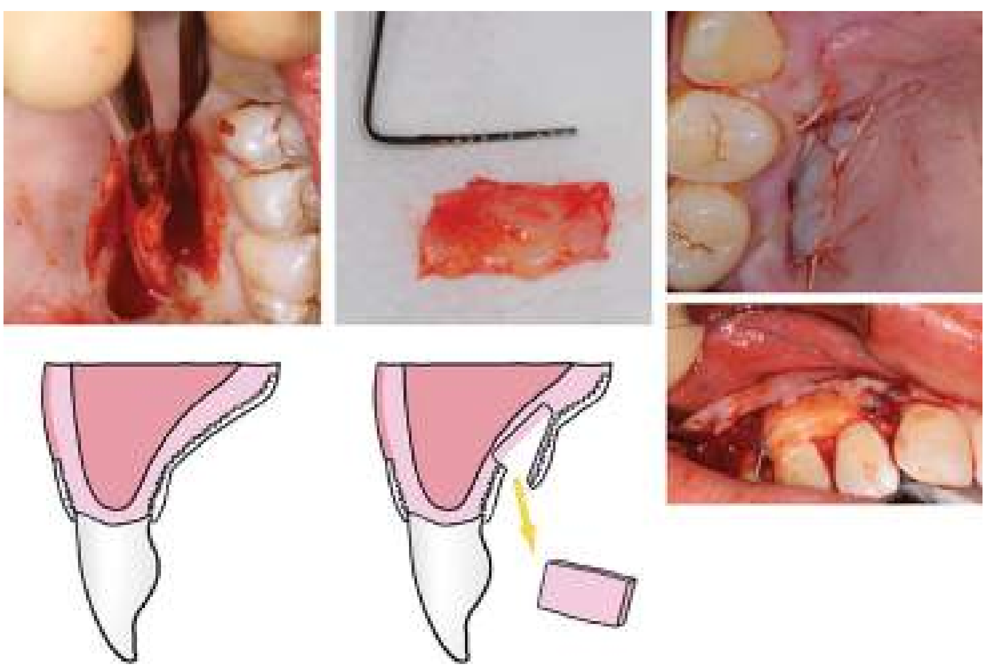
Grafting procedures include

- Free gingival graft (epithelium + connective tissue)
- The pedicle sliding graft (Lateral repositioned graft)
- The sub epithelial connective tissue graft (connective tissue)

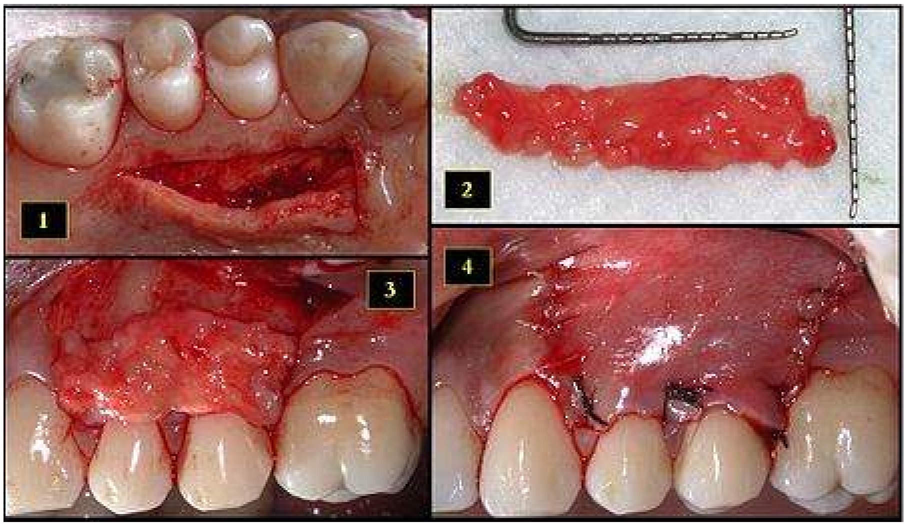


Free gingival graft

Free gingival graft

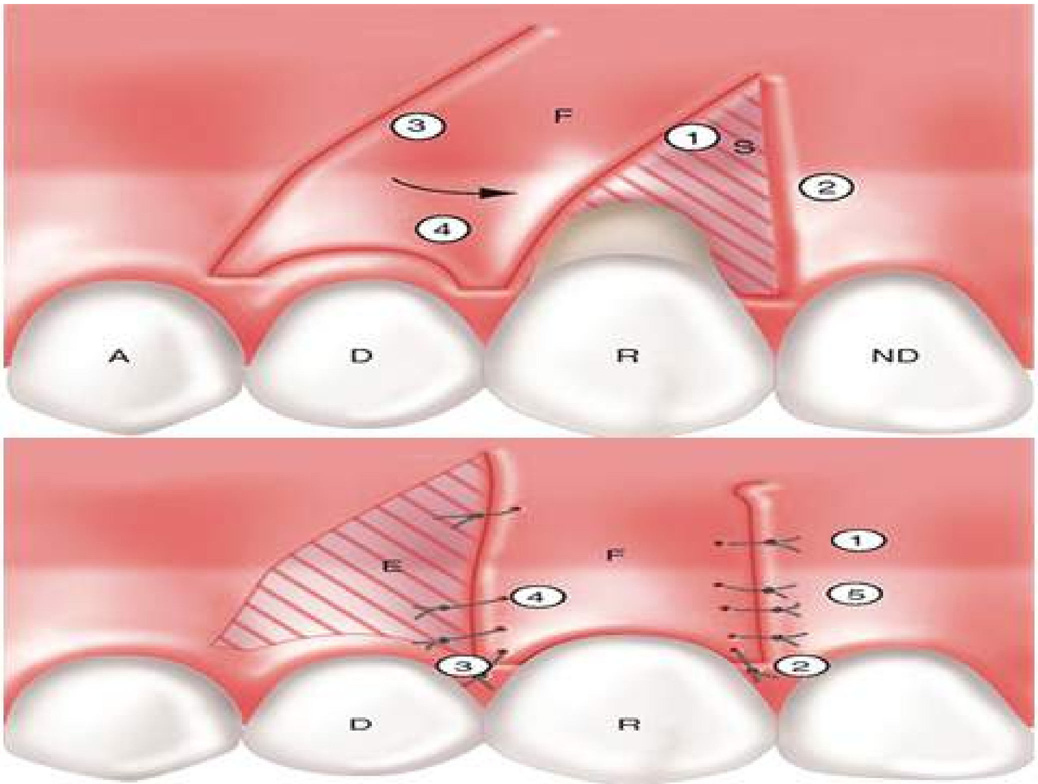


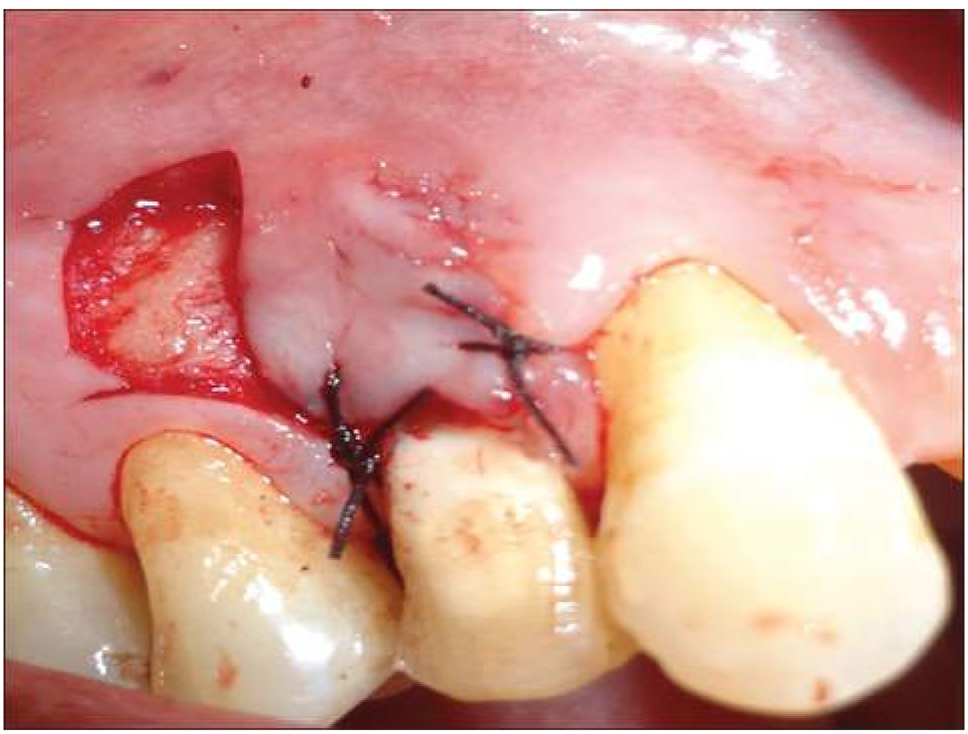
Sub epithelial connective tissue graft



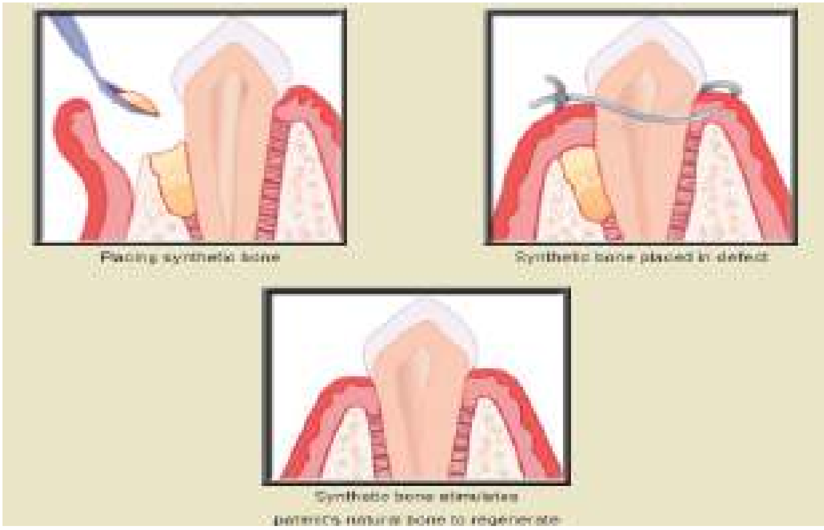
Sub epithelial connective tissue graft

Lateral repositioned graft

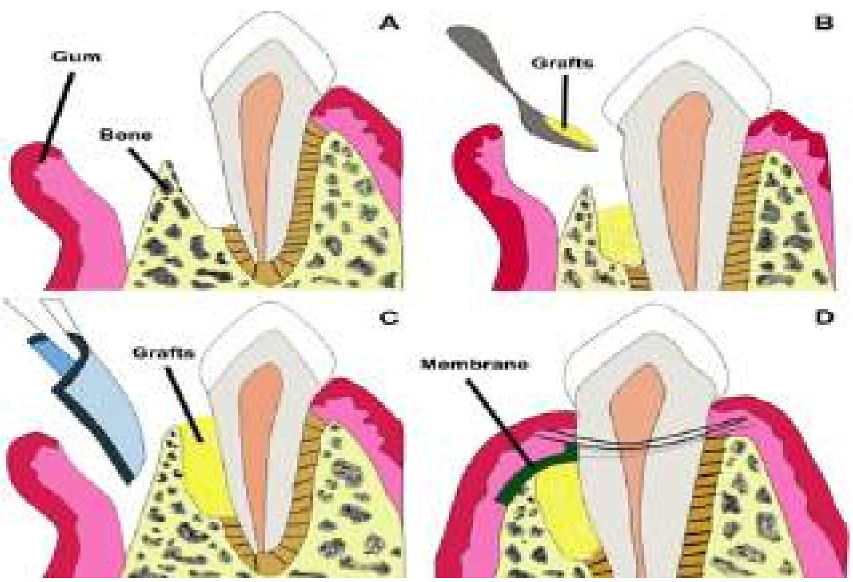




Lateral repositioned graft



Bone graft (GBR)

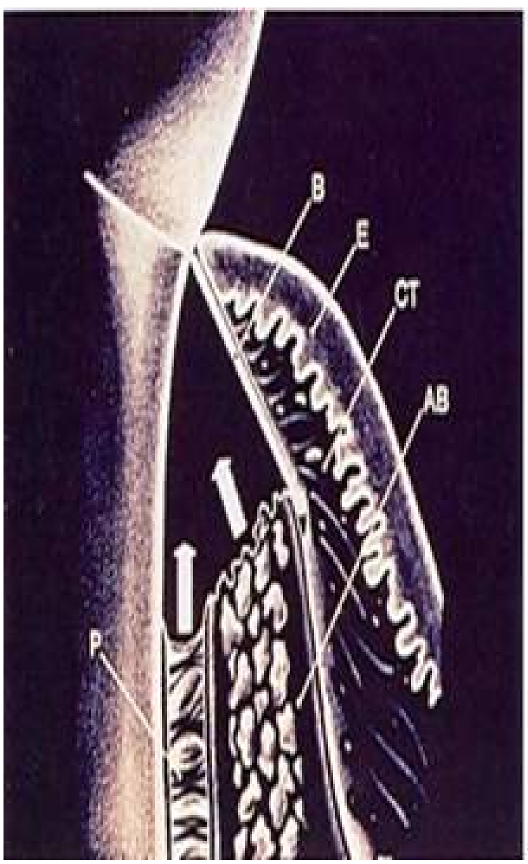


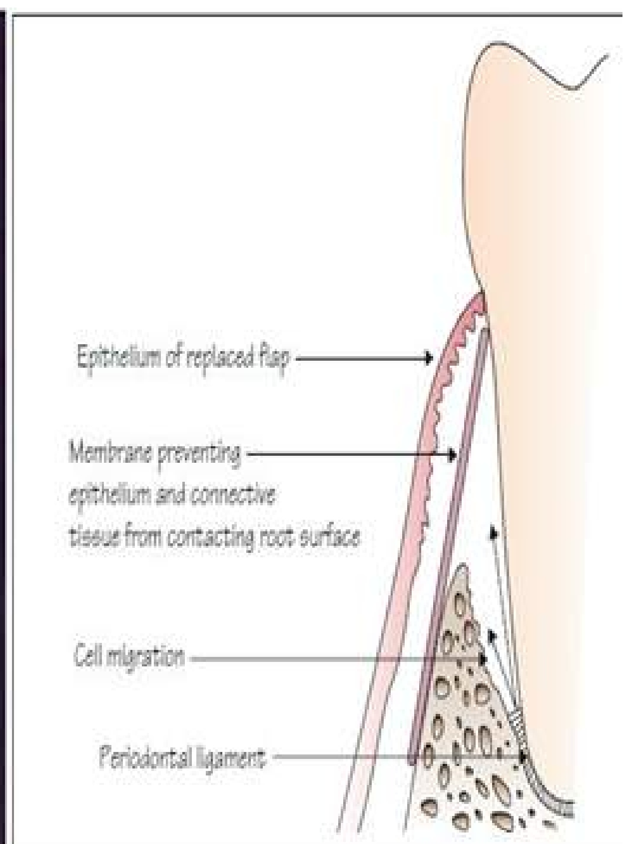
GTR + GBR graft

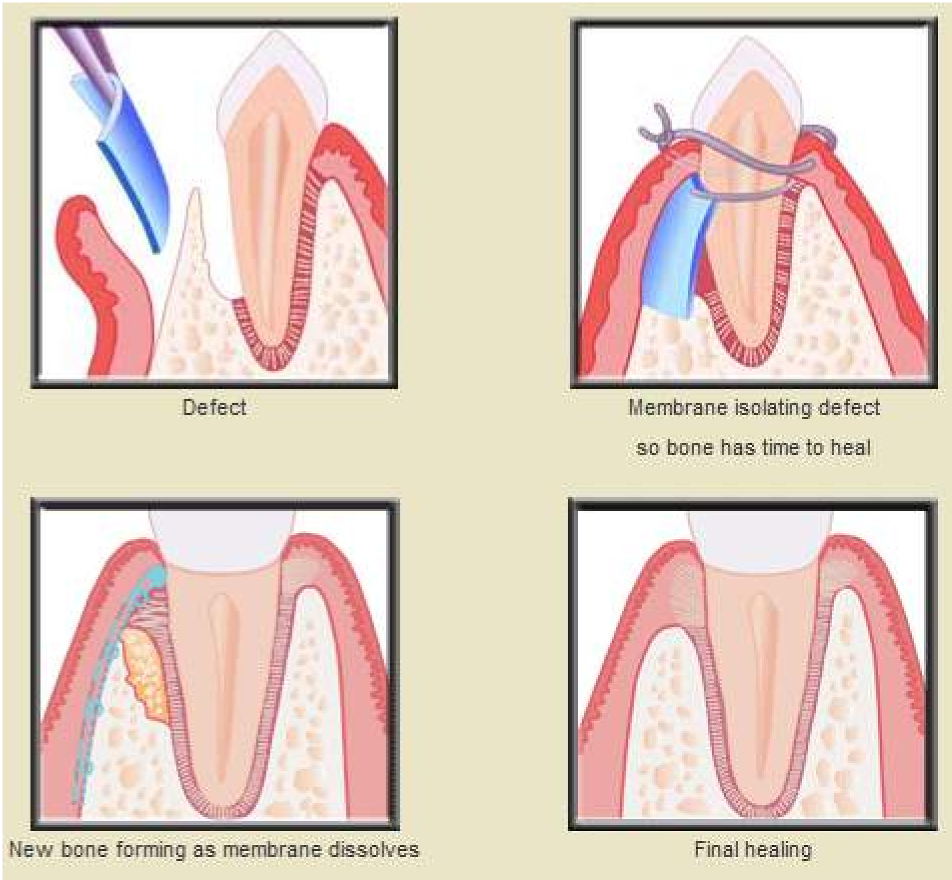
<u>Guided tissue regeneration GTR</u>

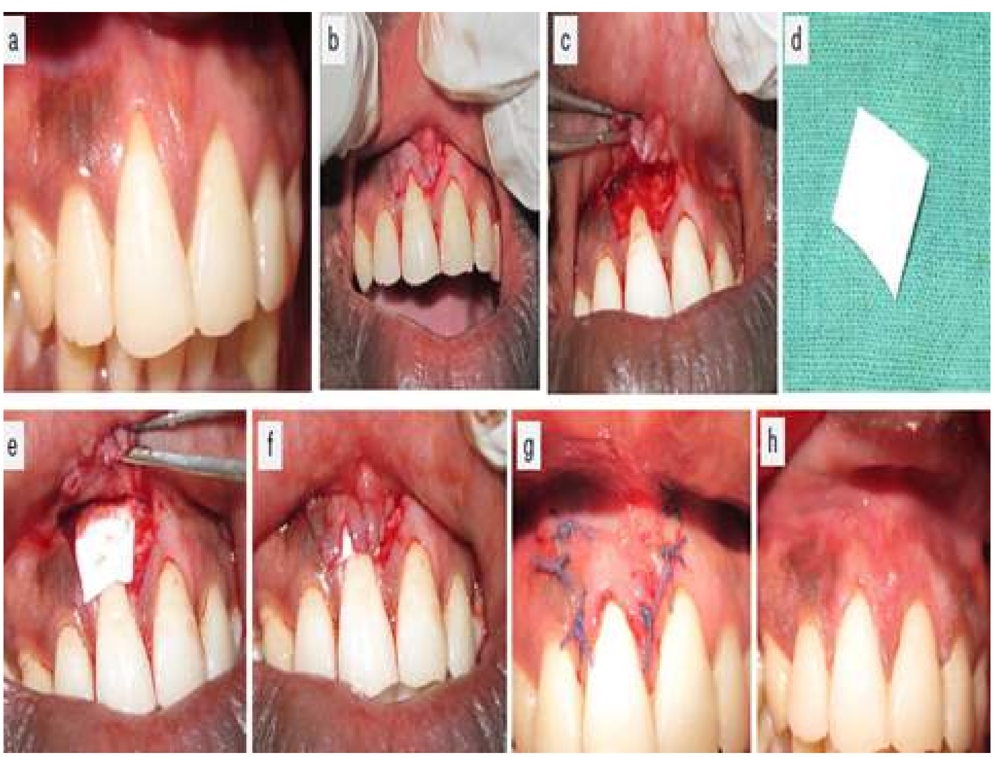
Following periodontal surgery, the instrumented root surface is colonized by gingival epithelial cells to form a long junctional

epithelium which prevent the formation of new connective tissue attachment to the root surfaces, thus GTR is achieved by placing barrier membrane over periodontal defect to exclude gingival epithelium and connective tissues cells, and to create a space into which the proliferating cells from periodontal ligament and bone can migrate into healing area. These cells have the
capability to differentiate into fibroblast, cementoblast and osteoblast and thus can produce new periodontal ligament fibers, cementum and bone to regenerate the lost connective tissue attachment to the root surface. Membranes are either non-
resorbable which require removal 4-6 weeks after placement or resorbable which biodegrade within the tissue over 12 months






Defect
Membrane isolating defect
so bone has time to heal
New bone forming as membrane dissolves
Final healing


a
b
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d
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g
h

Crown lengthening

Indication

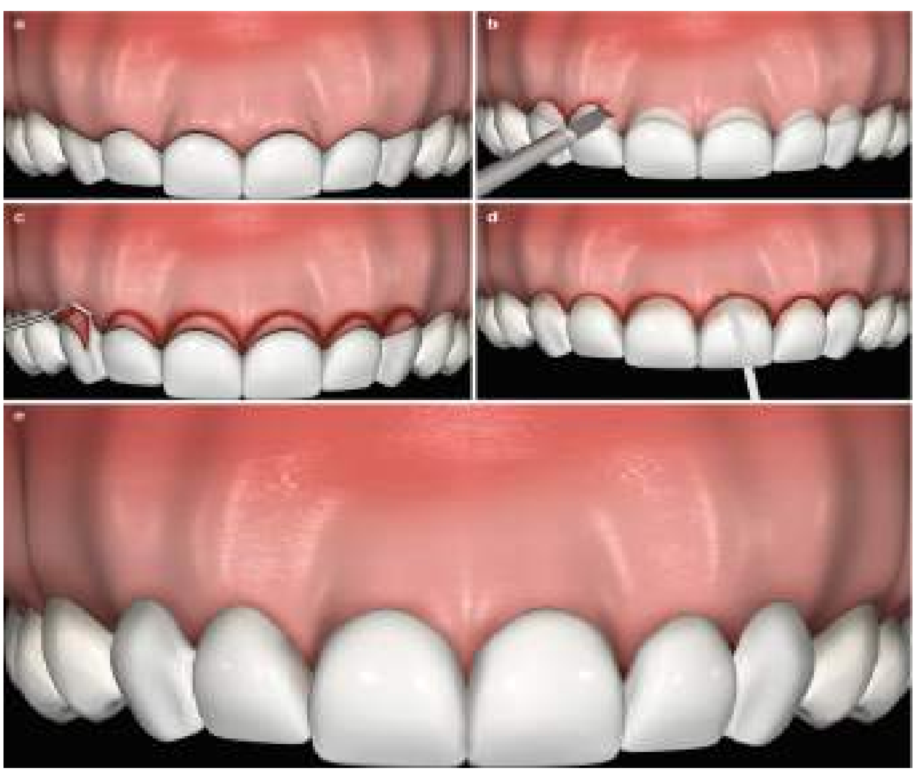
1-Short clinical crown require increased retention for placement of full coronal restoration (including cases of gross tooth wear requiring full mouth rehabilitation)

2-Deep subgingivally located crown preparation margins, resulting in difficulty finishing margins and taking impressions also encroachment on the biologic width

3-Sub gingival caries

4-Root fractures or root resorption in the cervical third of the tooth root

5-Aesthetic improvement of anterior teeth with short clinical crowns and high lip line

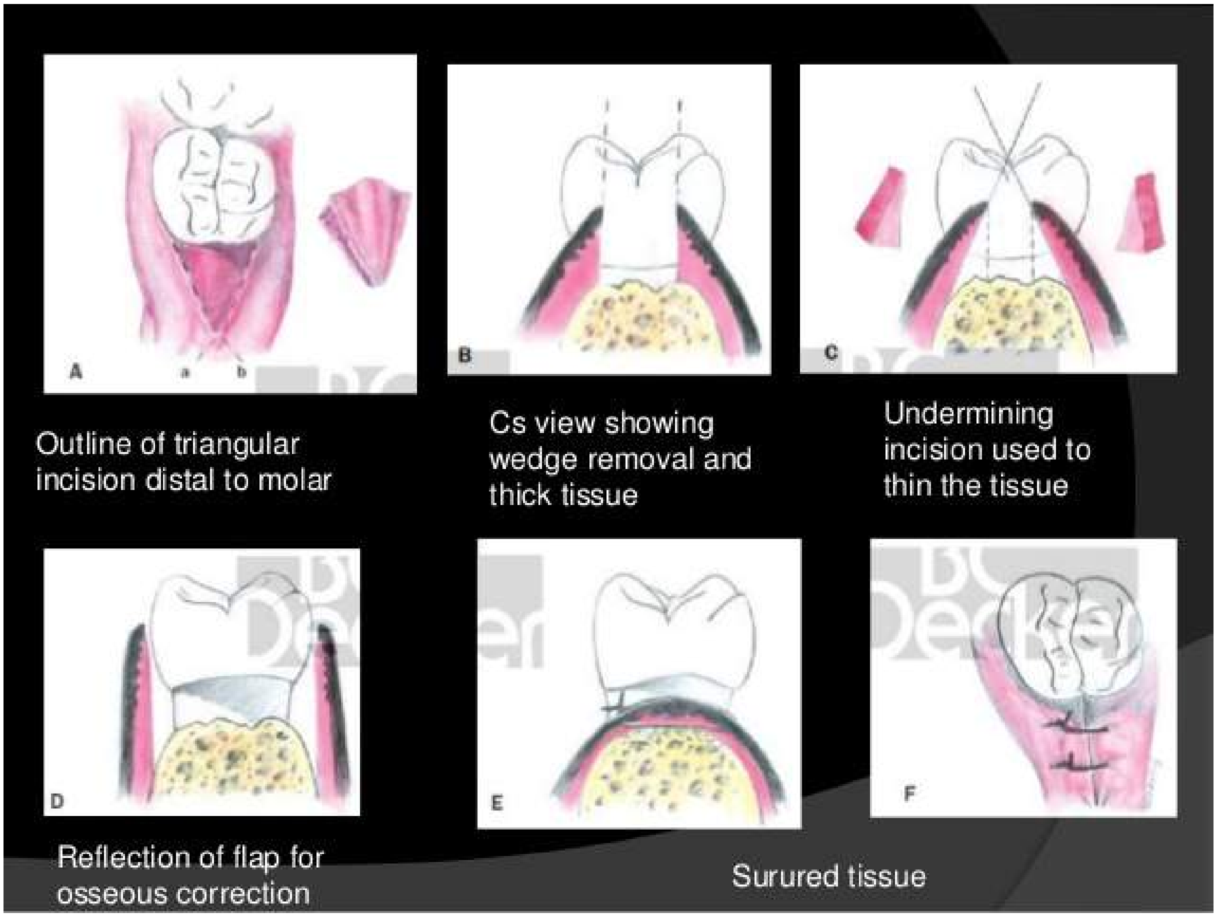


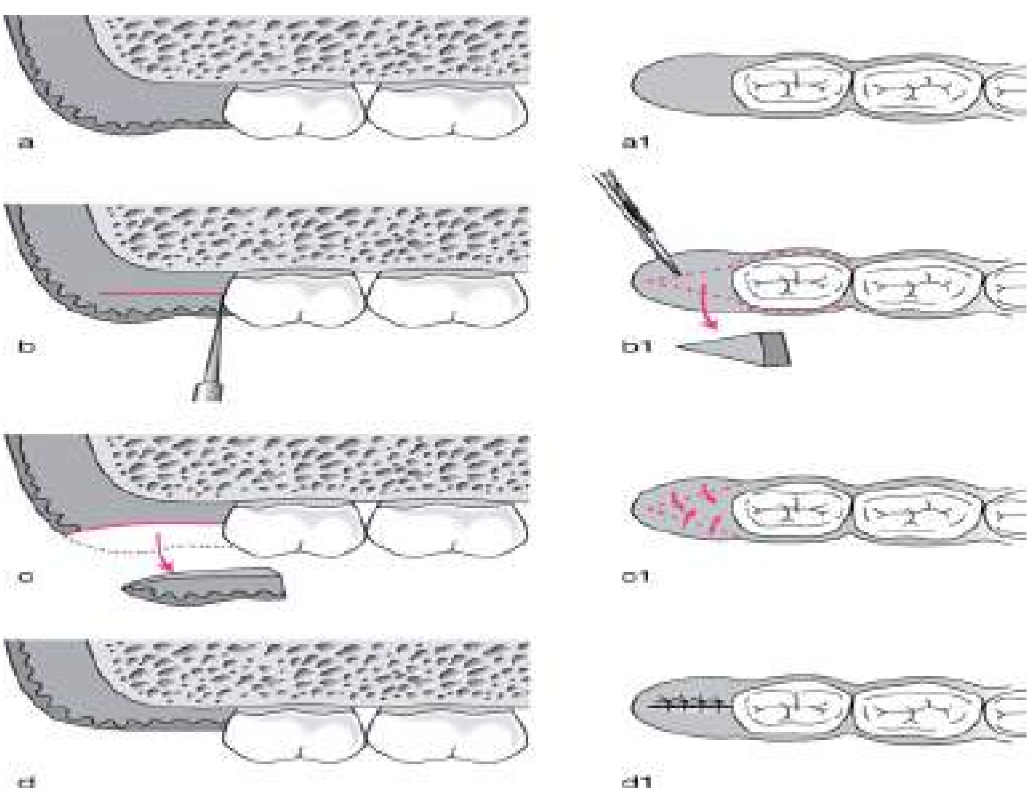

Before
After

Distal wedge procedures

In many cases the treatment of periodontal pockets on the distal surface of distal molars is complicated by the presence of bulbous tissue over the tuberosity or by a prominent retromolar pad. The direct aproach to pocket elimination in the maxillary
jaw is the gingivectomy, however when limited amount of keratinized gingiva are present, or not at all, or a distal angular bony defect has been diagnosed,the bulbous tissue should be reduced in size rather than being removed, this may be accomplished by the distal wedge procedure which facilitate access to the osseous defect,eliminating the deep pocket and preserve sufficient amount of gingiva and mucosa to achieve soft tissue coverage of the remaining periodontium.

Retro molar flap operation: This can be used distally to last molar near to an edentulous area to gain access for RP and pocket reduction or elimination. Initial incision is done buccally and palatally/lingually (distal wedge) .Tissues between the two incisions(triangular –shaped wedge exicision) are removed & the flap is reflected as much as possible for better visualization of the root surface .The second incisions serve to undermine and thin the buccal and palatal/lingual tissue flaps overlying the alveolar bone. Repositioning the flaps with sutures.


A
a
b
B
C
D
E
F
Outline of triangular incision distal to molar
Cs view showing wedge removal and thick tissue
Undermining incision used to thin the tissue
Reflection of flap for osseous correction
Surured tissue


a
a1
b
b1
c
c1
d
d1

Techniques for the removal of the frenum

A frenum is a fold of mucous membrane, usually with enclosed muscle fibers, that attaches the lips and cheeks to the alveolar mucosa and/or gingiva and underlying periosteum.A frenum becomes a problem if the attachment is too close to the marginal gingiva.Tension on the frenum may pull the gingival margin away from the tooth.This condition may be conducive to plaque accumulation and inhibit proper brushing of the teeth with pocket formation.Also may tend to open the sulcus and gingival recession.

Frenectomy or Frenotomy

The term frenectomy is complete removal of the frenum,including its attachment to underlying bone and may be required in the correction of an abnormal diastema between maxillary central incisors.

Frenotomy is the incision of the frenum and relocating the frenal attachment.

Frenal problems occur most often on the facial surface between maxillary and mandibular central incisors and in the canine and premolar areas.They occur less often on the lingual surface of the mandible.

The technique for the removal of the frenum accomplished as follows:

1.After anesthetizing the area, engage the frenum with a hemostat inserted to the depth of the vestibule.

2.Incise along the upper surface of the hemostat,extending beyond the tip.

3.Make a similar incision along the undersurface of the hemostat.

4.Remove the triangular resected portion of the frenum with the hemostat.This exposes the underlying brushlike fibrous attachment to the bone.

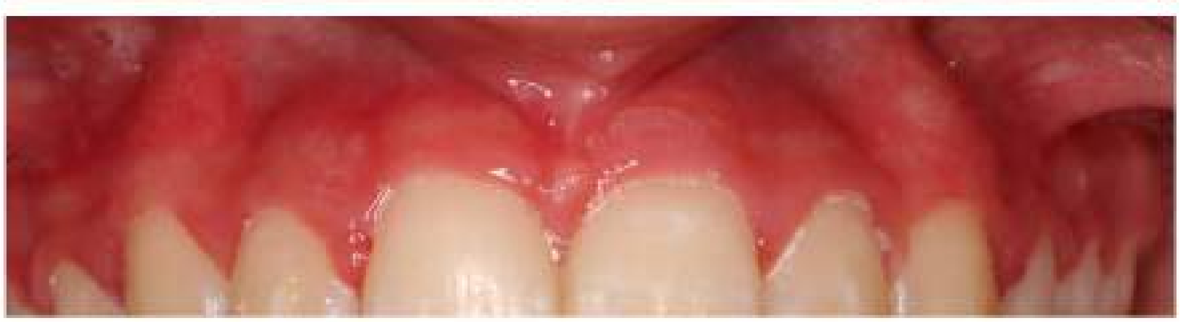
5.Make a horizontal incision, separating the fibers,and bluntly dissect to the bone.

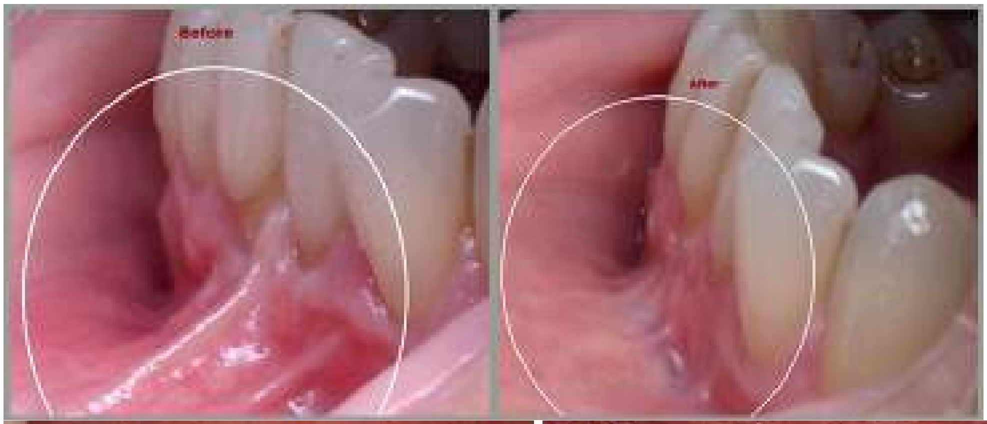
6.Undermining the incision to approximate the border of incisions for suturing.

7.Clean the field of operation and pack with gauze sponges until bleeding stops.

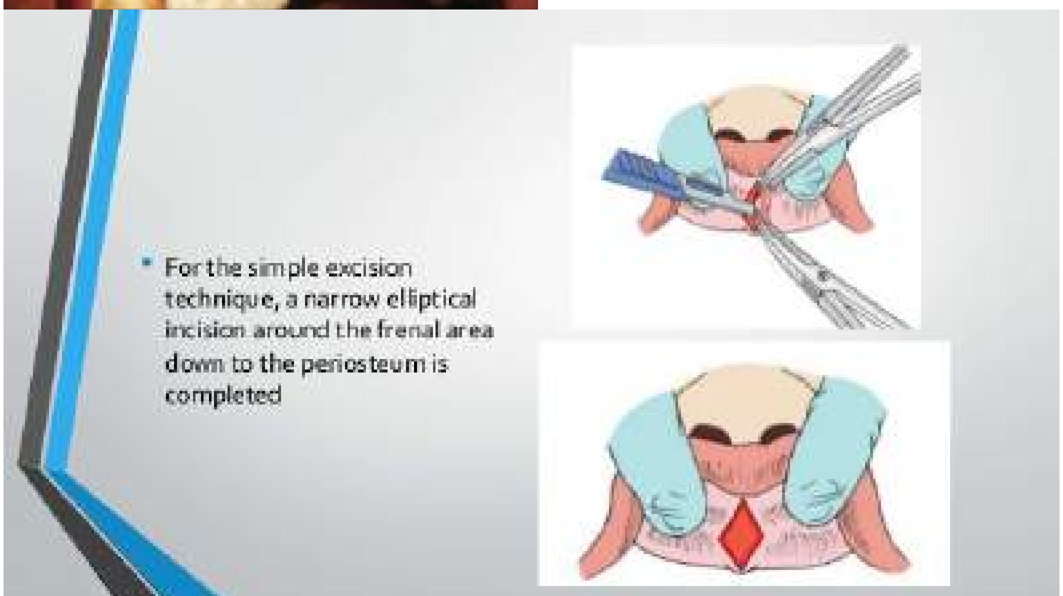
8.Cover the area with periodontal pack.

9.Remove the pack after 1 week.One month is usually required for the formation of an intact mucosa with the frenum attached in its new position.




Before

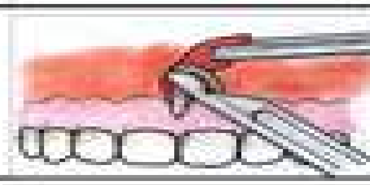
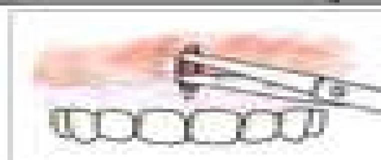
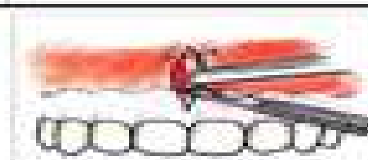

Luiz Fernando Ferreira



• For the simple excision technique, a narrow elliptical incision around the frenal area down to the periosteum is completed

Anterior Labial Frenectomy

Keys to Success

Step 1 – Initial Incision *Make sure the 15 or 15c blade is angled 45 degrees to the underlying bone*	
Step 2 – Excision of Overlying Tissue *Use Adson tissue forceps to hold the flap edges taut and hold the fully mobile mucosal flap*	
Step 3 – Soft Tissue Release *Pull the wound edges taut with the Adson tissue forceps and spread the incision with the Metzenbaum scissors*	
Step 4 – Excision of Frenum *Firmly grasp the frenum with Adson tissue forceps throughout the entire step*	

Periodontal dressing: are mainly used :

I· To protect the wound post surgically

2-To obtain and maintain a close adaptation of the mucosal flaps to the underlying bone (especially when a flap has been repositioned apically)
3-For the comfort of the patient

4-Prevent post operative bleeding during the initial phase of healing

5-Prevent the formation of excessive granulation tissue
Periodontal dressing should have the following properties:

1-Should be soft but still have enough plasticity and flexibility to facilitate its placement in the operated area and to allow proper adaptation.
2-Should harden within a reasonable time

3-After setting should be sufficiently rigid Io prevent fracture and dislocation.
4-Should have a smooth surface after setting to prevent irritation to the check and lips

5-Should preferably have bactericidal properties to prevent excessive plaque formation

6-Not detrimentally interfere with healing

<u>Types of dressing</u>

I-Zinc-oxide eugenol pack: eugenol in this type may induce an allergic reaction
2-Non eugenol pack: e.g. Coe pack; one tube contain zinc oxide and lorothidol (Fungicidal) and the second tube contain non ionizing carboxilic acids and chlorothymol (bacteriostatic agent)

3-Light cured dressing

Maintenance phase(supportive periodontal therapy SPT)

Preservation of the periodontal health of the treated patient requires a supportive program that is just as important as the therapy used to treat the periodontal disease.The maintenance phase of periodontal treatment starts immediately after the completion of phase I therapy.While the patient is in the maintenance phase,the necessary surgical and restorative procedure are performed.This insures that all areas of the mouth retain the degree of health attained after phase I therapy.

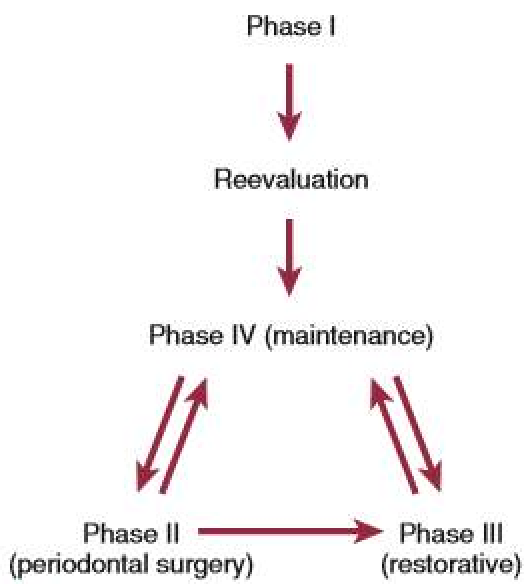


Fig. 72.2 Correct sequence of periodontal treatment phases.

The primary goal of maintenance therapy include

I-Maintenance of oral health (cancer screening)

2-Prevention of new infection

3-Prevention of re-infection and disease recurrence

The time interval between the recall appointments should be based on a periodontal risk assessment(type and severity of periodontitis, systemic and local risk factors, degree of motivation, compliance, manual dexterity and the patient success to maintain a proper personal oral hygiene standard.

It is important to emphasize that the recall program must be designed to meet the individual need of the patient, some patients should be recall every month while other may have to be checked only once a year

Findings From long-term clinical trials have suggested that recall appointments, once every three month is effective in preventing disease recurrence.

There are three parts to an SPT appointment:

1.examination

2.treatment

3.report,clean up and scheduling

Part I: Examination
(Approximate time: 14 minutes)
Patient greeting
Medical history changes
Oral pathologic examination
Oral hygiene status
Gingival changes
Pocket depth changes
Mobility changes
Occlusal changes
Dental caries
Restorative, prosthetic, and implant status

Part II: Treatment
(Approximate time: 36 minutes)
Oral hygiene reinforcement
Scaling
Polishing
Chemical irrigation or site-specific antimicrobial placement

Part III: Report, Cleanup, and Scheduling
(Approximate time: 10 minutes)
Write report in chart.
Discuss report with patient.
Clean and disinfect operatory.
Schedule next recall visit.
Schedule further periodontal treatment.
Schedule or refer for restorative or prosthetic treatment.

The time required for a recall visit for patients with multiple teeth in both arches is approximately 1 hour.

Recurrence of Periodontal Disease

Occasionally, lesions may recur, which is often due to inadequate plaque/biofilm control on the part of the patient or failure to comply with recommended SPT schedules. It should be understood, however, that it is the responsibility of the dentist to educate and motivate patients to improve their oral hygiene techniques. Surgery should not be undertaken unless the patient participates in disease prevention and demonstrates proficiency in plaque/biofilm control.

Other causes for recurrence include the following:

1.Inadequate or insufficient treatment that has failed to remove all of the potential factors favoring biofilm accumulation. Incomplete calculus removal in areas of difficult access is a common source of problems.
2. Inadequate restorations placed after the periodontal treatment was completed.
3. Failure of the patient to return for periodic maintenance care . This may be a result of the patient’s conscious or unconscious decision not to continue treatment or the failure of the dentist and staff to emphasize the need for periodic supportive therapy.
4. Presence of some systemic diseases that may affect host resistance to previously acceptable levels of biofilm.

A failing case can be recognized by the following:

1. Recurring inflammation revealed by gingival changes and bleeding of the sulcus on probing.
2. Increasing depth of sulci, leading to the recurrence of pocket formation.
3. Gradual increases in bone loss, as determined by radiographs.
4. Gradual increases in tooth mobility, as ascertained by clinical examination.

The decision to retreat a periodontal patient should not be made at the preventive maintenance appointment but should be postponed for 1 to

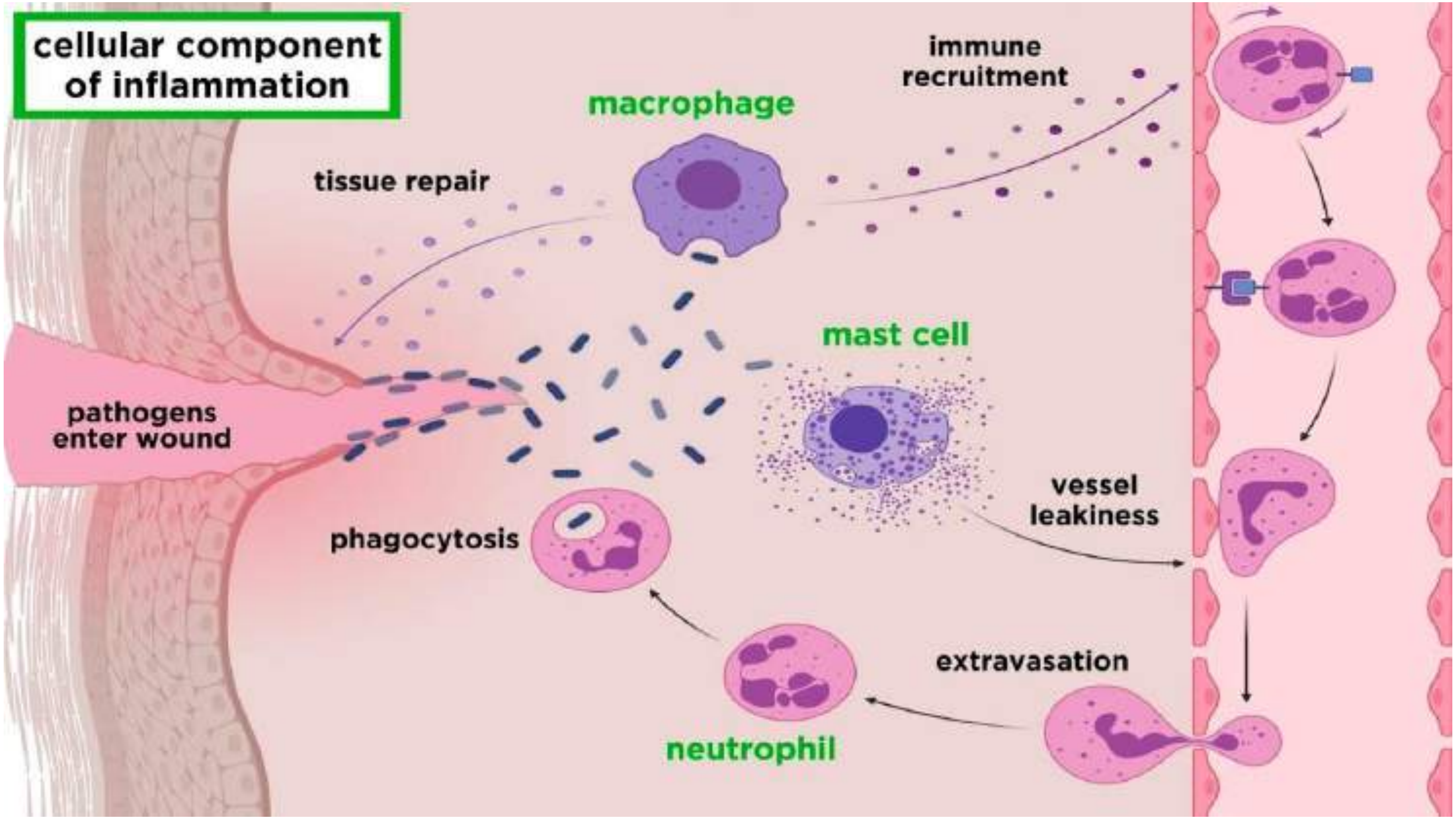
2 weeks. Often, the mouth appears improved at that time because of the resolution of edema and the resulting improved tone of the gingiva. Table summarizes the symptoms of the recurrence of periodontal disease and their probable causes.

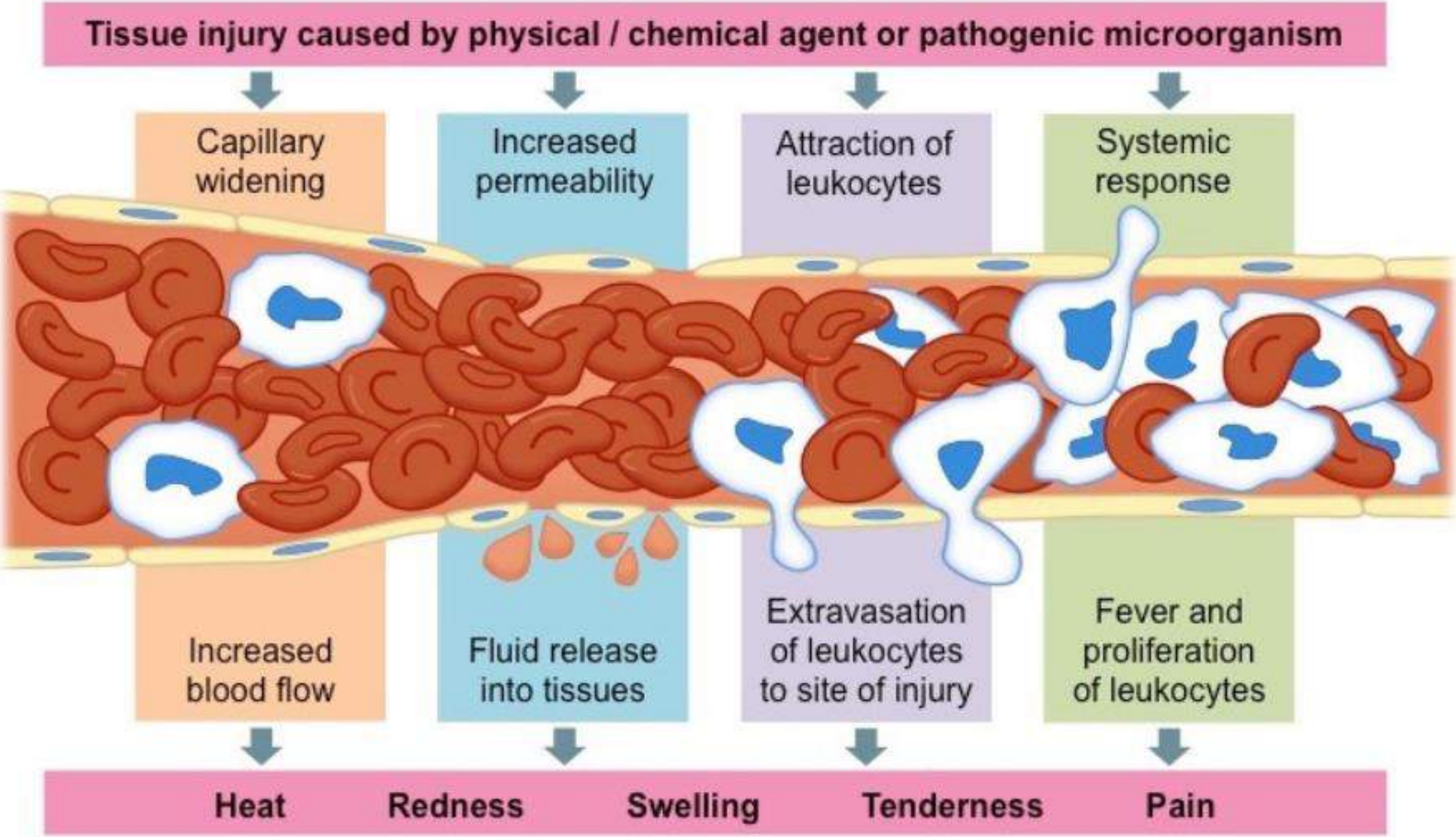
Symptom	Possible Causes
Increased mobility	Increased inflammation Poor oral hygiene Subgingival calculus Inadequate restorations Deteriorating or poorly designed prostheses Systemic disease modifying host response to plaque
Recession	Toothbrush abrasion Inadequate keratinized gingiva Frenum pull Orthodontic therapy
Increased mobility with no change in pocket depth and no radiographic change	Occlusal trauma caused by lateral occlusal interference, bruxism, high restoration Poorly designed or worn-out prosthesis Poor crown-to-root ratio
Increased pocket depth with no radiographic change	Poor oral hygiene Infrequent recall visits Subgingival calculus Poorly fitting partial denture Mesial inclination into edentulous space Failure of new attachment surgery Cracked teeth Grooves in teeth New periodontal disease Gingival overgrowth caused by medication
Increased pocket depth with increased radiographic bone loss	Poor oral hygiene Subgingival calculus Infrequent recall visits Inadequate or deteriorating restorations Poorly designed prostheses Inadequate surgery Systemic disease modifying host response to plaque Cracked teeth Grooves in teeth New periodontal disease

Pathogenesis of periodontal disease

م. سها أسود دهش العزاوي
B.D.S, MSc. Periodontology

- Inflammatory and immune reactions to microbial plaque are the predominant features of gingivitis and periodontitis.
- Inflammatory and immune processes operate in the gingival tissues to protect against local microbial attack and prevent microorganisms or their damaging products from spreading into or invading the tissues. These host defense reactions are, however, also considered potentially harmful to the host in that inflammation can damage surrounding cells and connective tissue structures.
- These "defensive" processes could therefore paradoxically contribute to the tissue injury observed in gingivitis and periodontitis.


cellular component
of inflammation
immune
recruitment
macrophage
tissue repair
mast cell
pathogens
enter wound
vessel
leakiness
phagocytosis
extravasation
neutrophil



Tissue injury caused by physical / chemical agent or pathogenic microorganism
Capillary widening
Increased permeability
Attraction of leukocytes
Systemic response
Increased blood flow
Fluid release into tissues
Extravasation of leukocytes to site of injury
Fever and proliferation of leukocytes
Heat
Redness
Swelling
Tenderness
Pain

The inflammatory reaction is visible both clinically and microscopically in the affected periodontium.

INITIATION OF PERIODONTAL DISEASE

- Pathogenesis → biological and histological events that occur in the tissue during the process of conversion from the healthy state to diseased one.
- Most normal subjects maintaining a high standard of oral hygiene are not likely to develop advanced periodontal disease.
- Microorganisms quickly start to colonize clean tooth surfaces once an individual abstains from mechanical tooth cleaning; within a few days microscopical and clinical signs of gingivitis are then apparent. These inflammatory alterations are resolved or reversed when adequate tooth cleaning measures are resumed. The inflammatory changes may remain confined to the gingival area for several years, but at some sites gingivitis eventually shifts to destructive periodontal disease resulting in loss of connective tissue attachment and alveolar bone.
- Clearly some imbalance of the host-microbial relationship is occurring in the destructive lesions, which may be unique to that site and to periodontally susceptible individuals.

MECHANISMS OF PATHOGENICITY

For a periodontal pathogen to cause disease, it is essential that the pathogen be able to

(1) Colonize the subgingival area and

(2) Produce factors that either directly damage the host tissue or lead to the host tissue damaging itself.

To colonize subgingival sites, a species must be able to:

(1) Attach to one or more of the available surfaces,

(2) Multiply,

(3) Compete successfully against other species, and

(4) Defend itself from host defense mechanisms.

Adhesions

To establish in a periodontal site, a species must be able to attach to one or more surfaces including the tooth, the sulcular or pocket epithelium or other bacterial species attached to these surfaces. Some of the adhesins that have been identified on subgingival species include fimbriae and cell associated proteins. Receptors on tissue surfaces that species adhere to them include galactosyl residues and proline rich proteins.

Coaggregation

While many species attach directly to host surfaces, other species attach to bacteria attached to such surfaces. This phenomenon is called coaggregation.

Multiplication

The gingival crevice and/or periodontal pocket might be considered a lush area for microbial growth, but is in fact a rather stringent environment for a bacterial species to live. The mean temperature of the area averages about 35'C and ranges from 30'-38"C. The pH is rather restricted (pH 7. 0-8.5). Three sources of nutrient are available to subgingival organisms (diet, host and other subgingival species). Certain nutrients essential to some bacterial species must be formed by other species in that area. Gingival crevice fluid is not particularly rich in nutrients, creating a major competition for the small amounts available. In addition, nutrients delivered in relative abundance to the outer layers of plaque may not reach deeper layers.

Interbacterial relationships

Bacterial interactions play important roles in species survival. Some inter-species relationships are favorable, in that one species provide growth factors or facilitate attachment of another. Other relationships are antagonistic due to competition for nutrients and binding sites or to the production of substances which limit or prevent growth of a second species such as production of hydrogen, peroxide by S. sanguis which suppress the growth of A. actinomycetemcomitans. On the other hand, the growth of s. sanguis has been shown inhibited by a bacteriocin produced by A. actinomycetemcomitans‘.

Overcoming host defence mechanisms

Subgingival plaque microorganisms appear to overgrow and lead to severe disease in immune-compromised hosts, particularly those with neutrophil disorders. A bacterial species has a number of host-derived obstacles to overcome when colonizing a subgingival site' These include the flow of saliva and gingival crevice fluid and mechanical displacement by chewing and speaking. Substances in saliva and gingival crevice fluid may aid in the prevention of colonization by blocking the binding of bacterial cells to mammalian surfaces' Such factors include specific antibodies, salivary glycoproteins and mucins, which may act as non-specific blocking agents.

Desquamation of epithelial cells presents a new cleansing mechanism, which is overcome by certain species by their ability to bind to underlying epithelial cells. Other species are able to invade the epithelial cells and may multiply intracellularly and spread to adjacent cells. Specific antibody in the subgingival area could act by preventing bacterial attachment or, in some instances, by making the bacterial cell susceptible to various phagocytic or killing mechanisms.

A number of subgingival species have evolved mechanisms for evading the effect of specific antibody. Species including P. gingivalis, P. intermedia, P' melaninogenica and Capnocytophaga species possess IgG and IgA proteases that can destroy antibody' other species are capable of evading antibody by changing their surface antigens or possibly by mimicking the host,s antigens species. A number of bacterial mechanisms exist that might including the production of leukotoxin by A. actinontycetemcomitans and capsules by P' gingivalis and other species that inhibit phagocytosis. In addition, a number of species have developed strategies to interfere with the killing mechanisms of the polymorph nuclear leukocytes.

Virulence Factors

Two general mechanisms of pathogenesis have been hypothesized:

- The first involves invasion by subgingival species.
- The second suggests a "long-range" attack where cells of the pathogenic species remain in the pocket but fragments of cells as well as other "**virulence factors" enter the underlying periodontal tissues and either directly damages the tissues or cause "immune pathology" (indirectly).**

Virulence factors can be divided into three categories:

1- Substances that damage tissue cells (e.g. H2S),

2- Substances that affect the intercellular matrix (e.g. collagenase). and

3-Substances that cause cells to release biologically active substances (e.g.lipopolysaccharide)

Virulence factors of Aggregatibacter actinomycetemcomitans

- **Leukotoxin**; kills PMNs and monocytes.
- Cytolethal distending toxin.
- Immunosuppression factors that inhibit antibody production and activate T-suppressor cells.
- **Inhibition of PMNs functions**.
- **Resistant to complement-mediated killing**.
- **Lipopolysaccharides**.
- Surface antigens.
- Antimicrobial resistance.

Virulence factors of P. gingivalis

- Gingipain is a protease secreted by Porphyromonas gingivalis. they work to degrade cytokines , thereby down regulating the host response in the form of reduced inflammation.
- Capsular polysaccharide.
- Fimbriae, hemagglutinins.
- Proteinases, hemolysins.
- Collagenase.
- secreting a serine phosphatase that inhibits the synthesis of IL-8.
- Lipopolysaccharides.

Red complex

The red complex is a group of bacteria that are categorized together based on their association with severe forms of periodontal disease. The red complex-among a number of other complexes were classified by Sigmund Socransky in 1998.

The three members of the red complex are:

1- **Porphyromonas gingivalis.**

2- Tannerella forsythia.

3- Treponenta denticola.

NORMAL"CLINICALLY HEALTHY" GINGIVA

Normal gingiva is characterized clinically by its pink colour and firm consistency and the gingival margin exhibits a scalloped outline, The interdental papillae are firm, do not bleed on gentle probing and fill the space below the contact areas. The gingiva often exhibits a stippled appearance and there is a knife edge margin between tooth and soft tissue. Normal gingiva is theoretically free from histological inflammation, but this "ideal" healthy condition has two types: a super healthy or "pristine" state which histologically has little or no inflammatory infiltrate, and the "clinically healthy" gingiva which looks similar clinically but histologically features an inflammatory infiltrate.

In clinically healthy gingiva features an infiltrate of inflammatory cells, **predominantly neutrophil**s associated with the junctional epithelium and lymphocytes in the subjacent connective tissue. The infiltrate at this stage may occupy as much as 5% of the connective tissue volume and is composed of monocytes, macrophages, lymphocytes and neutrophils. These cells are found in the junctional epithelium as well as in the connective tissue of clinically healthy gingiva.

HISTOPATHOLOGICAL FEATURES OF GINGIVAL INFLAMMATION

An experimental gingivitis study in dogs was done by Page and Schroeder had compared the cellular and structural composition of the affected area before and during the development of gingivitis over a period of 28 days. At Day 0 of this experiment the normal gingival unit has virtually no inflammatory cells and is comprised of approximately 40-45% epithelium and 55-60 of connective tissue. The connective tissue zone consists of collagen (60%), fibroblasts (13%),vessels (7%) and other tissue constituents, such as intercellular matrix and nerves 20%). Following plaque accumulation, neutrophils and mononuclear leukocytes readily migrate to this area and the connective tissue begins to form and increase in volume over the 28-day period.

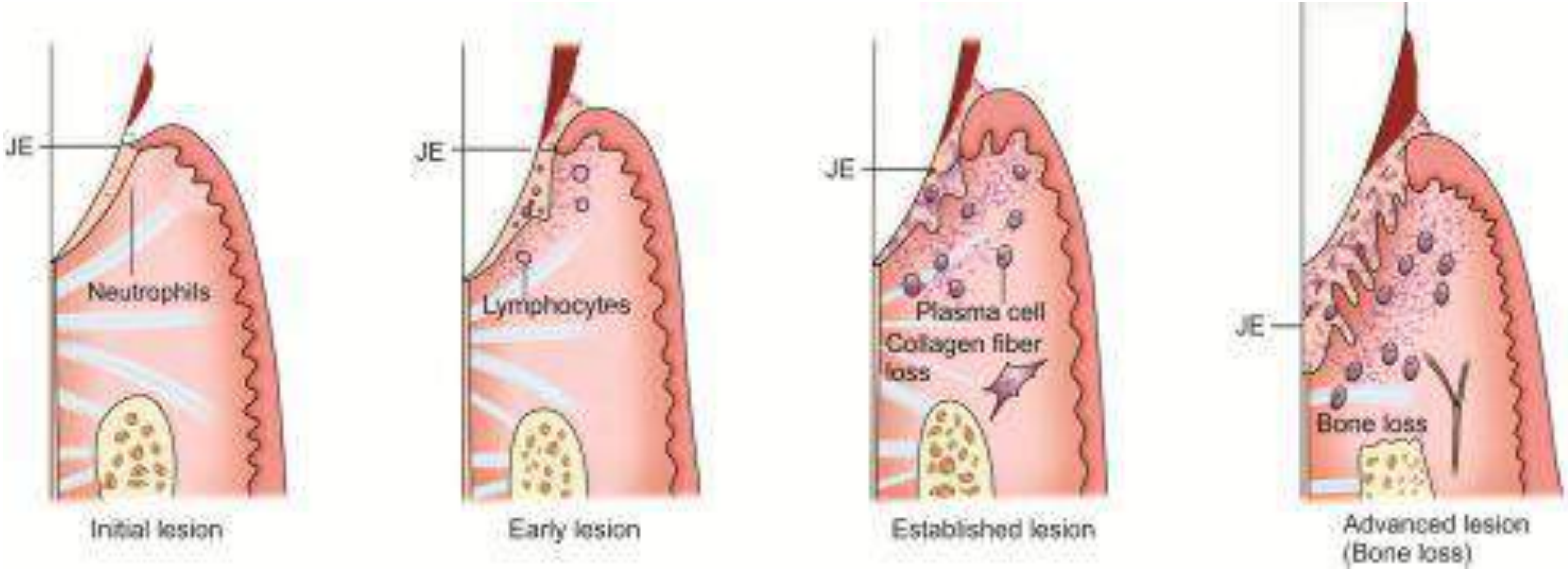
At this 28-day interval the connective tissue is comprised of lymphocytes, plasma cells and macrophages which adhere to the collagen matrix and remain in the tissue, whereas neutrophils continue to migrate into the gingival sulcus. With the extensive influx of leukocytes, a marked reduction in the amount of collagen and fibroblasts occurs and the volume of residual tissue (intercellular matrix, degraded collagen, exudates material, degenerated or dead cells) and small blood vessels increases. Page and Schroeder classified the progression of gingival and periodontal inflammation on the basis of **clinical and histopathological evidence into four phases: initial, early, established and advanced stages or lesions.**

The initial lesion (clinically healthy gingiva)

Inflammation quickly develops as plaque is deposited on the tooth. Within 24 hours

marked changes are evident in the microvascular plexus beneath the junctional epithelium as more blood is brought to the area. Dilation of the arterioles, capillaries and venules of the dentogingival plexus is evident histopathologically. Hydrostatic pressure within the microcirculation increases and intercellular gaps form between adjacent capillary endothelial cells. As the lesion enlarges, and gingival crevicular fluid flow increases, noxious substances from microbes will be diluted both in the tissue and the crevice.

Bacteria and their products may thus be flushed from the sulcus. Plasma proteins escaping from the microcirculation include defensive proteins such as antibodies, complement and protease inhibitors and other macromolecules with numerous functions, probably within 2-4 days of plaque build-up the cellular response is well established and is helped by chemotactic substances originating from the plaque microbiota as well as from host cells and secretions. PMNs move through the connective tissue and the majority seem to accumulate in the junctional epithelium and gingival sulcus region.


JE
Neutrophils
Initial lesion
JE
Lymphocytes
Early lesion
JE
Plasma cell
Collagen fiber loss
Established lesion
JE
Bone loss
Advanced lesion (Bone loss)

The early lesion (early gingivitis)

The early gingival lesion occurs after approximately one week of plaque accumulation. The gingiva is erythematous in appearance as a result of proliferation of capillaries and continued vasodilatation. Increasing vascular permeability leads to increased GCF flow, and transmigrating neutrophils increase significantly in number. The predominant infiltrating cell types are neutrophils and lymphocytes (primarily thymic lymphocytes [T-cells]) and the neutrophils migrate through the tissues to the sulcus, and phagocytose bacteria. Fibroblasts degenerate, primarily via apoptosis (programmed cell death), which increases the space available for inhltrating leukocytes. Collagen destruction occurs, resulting in collagen depletion in the areas apical and lateral to the junctional and sulcular epithelium.

The basal cells of these epithelial structures begin to proliferate to maintain an intact barrier against the bacteria and their products, and as a result the epithelium can be seen proliferating into the collagen depleted areas of the connective tissues. As a result of edema of the gingival tissues, the gingiva may appear slightly swollen, and accordingly, the gingival sulcus becomes slightly deeper. thereby rendering effective plaque control more difficult. **The early gingival lesion may persist indefinitely or may progress further.**

The established lesion (established gingivitis)

Generally there is a further enhancement of the inflammatory state as exposure to plaque continues. There is increased fluid exudation and leukocyte migration into the tissues and the gingival crevice. Clinically this lesion will exhibit more edematous swelling than the "early gingivitis". Plasma cells are seen situated primarily in the coronal connective tissues and the rete pegs extend deeper into the connective tissue in an attempt to maintain epithelial integrity and a barrier to microbial entry.

The pocket epithelium that now has formed has a heavy leukocyte infiltrate, predominantly of PMNs which eventually migrate across the epithelium into the gingival pocket. In comparison to the original junctional epithelium, the pocket epithelium is more permeable to the passage of substances into and out of the underlying connective tissues and may in places be temporarily ulcerated. The pocket epithelium is less able to resist the passage of the periodontal probe, so bleeding on probing is a common feature of chronic gingivitis. It is important to remember that these inflammatory changes are still completely reversible if effective plaque control is reinstituted.

The advanced lesion (periodontitis)

The advanced lesion marks the transition from gingivitis to periodontitis. This transition is determined by many factors, include the bacterial challenge (both the composition and the quantity of the biofilm), the host inflammatory response, and susceptibility, factors, including environmental and genetic risk factors.

So the final stage in this process is known as the advanced lesion. The lesion is no longer localized to the gingival, and **the inflammatory cell infiltrate extends laterally and apically into the connective tissue of the true attachment apparatus**. The advanced lesion has all the characteristics of the established lesion but differs importantly in that alveolar bone loss occurs, fiber damage is extensive, the junctional epithelium migrates apically from the cementoenamel junction, and there are widespread manifestations of inflammatory and immunopatholcgical tissue damage. It is generally accepted that plasma cells are the dominant cell type in the advanced lesion.

Reference

Pathogenesis of periodontal disease lecture(4th stage)/University of Baghdad/College of Dentistry/Department of Periodontology.

Thank you

Periodontal management of medically compromised patients

Many patients seeking dental care have significant medical conditions that may alter both the course of their oral diseases and the therapy provided. The older age of the periodontal patients increases the likelihood of underlying diseases. Therefore therapeutic responsibility of the clinician includes identification of the patients medical problem to formulate proper treatment plans. Thorough medical histories are important and sometimes consultation with or referral of the patients to an appropriate physician may be indicated. This ensures correct patients management and provided medico-legal coverage to the clinician.

From the most common medical problems are the following:

1-cardio-vascular diseases:-

These diseases are the most prevelant category of systemic diseases and more common with increasing age. They include hypertension, angina pectoris, myocardial infraction, previous cerebrovascular accident, congestive heart failure, presence of cardiac pacemaker and infective endocarditis. In most cases the patient physician should be consulted, especially if stressful or prolonged treatment is anticipated. Short appointment and a calm , relaxing environment help to minimize stress.

a)Hypertension:

It is the most common cardiovascular disease and it is defined as a systolic blood pressure of 140 mmHg or greater, or a diastolic blood pressure of 90 mmHg or greater , and it is diagnosed on a single elevated blood pressure recording but it is based on the average value of three or more blood pressure readings taking at three or more appointment. If hypertension persist and increase in severity, it may lead to coronary heart disease, angina, congestive heart failure , cerebrovascular accident or kidney failure. Management of those patients will be based as follows:

1-No periodontal treatment should be given to patients who is hypertensive and not under the medical management.
2-The dentist should inform the physician about the degree of stress, blood loss and length of the periodontal procedure so that avoid excessive bleeding.

3-Local anesthesia without epinephrine may be used for short procedure (less than 30 minute)or use local anesthesia with an epinephrine concentration not more than 1:100:000 to control pain and minimize stress (dental treatment for hypertensive patients is generally safe as long as stress is minimized.
4-Avoid sudden positional changes or syncope.

b) Angina pectoris:

Angina occurs when myocardial oxygen demand exceed supply, resulting in temporary myocardial ischemia. Patients with a history of unstable angina pectoris (angina that occurs irregularly or on multiple occasions without predisposing factors) should be treated for emergencies only and in consultation with the patient physician. Patient with a history of stable (angina that is associated with stress and easily controlled with medication and rest) can be treated with the following precautions:
1-Premedication if needed as valium.
2-Morning and short appointment.
3-Nitroglycerine medication sublingually 5 minute before the procedure.
4-If during the periodontal procedure , the patient become fatigue or uncomfortable , the procedure should be discontinued.

c)Cardiac pacemaker:

Some cardiac arrhythmias are treated with implanted pacemakers which usually implanted in the chest wall and enter the heart transvenously. These electrical devices are used to regulate heart beats and no electro-physiologic problems may occur with such implanted device. Managements of such patients will be as follows:
1- Consult with the physician to get information about the underlying cardiac reason for pacing and to explain the periodontal treatment plan to him.
2-The patients should be positioned so that minimal pressure will be exerted on the implanted site.
3-Limited use of electrical equipment that generated electromagnetic field sush as ultrasonic devices so that to avoid interferences with the artificial pacemaker. Try to keep these device at least 30 cm from the patient. However, most pacemakers are adequately to prevent these changes.

d) Infective endocarditis

It is a diseases in which microorganisms colonize the damaged endocardium or heart valve . it is a serious diseases with poor prognosis. The term infective endocarditis is preferred to the previous term bacterial endocarditis because the disease can also be caused by fungi and viruses. The organisms most commonly encountered in IE are a hemolytic streptococci (streptococcus viridans) . other m.o. found in the periodontal pocket and associated with this disease are Eikenella corrodens, A.a., capnocytophaga and lactobacillus species. The practice of periodontics is intimately concerned with the prevention of IE. Any dental procedures that involve bleeding may induce a transient bactermia, so prophylactic antibiotic should be recommended before the procedures which is associated with significant bleeding as periodontal surgery, scaling and root planning. However , bactermia may occur even in the absence dental procedures, especially in individuals with poor hygiene and significant periodontal information. The preventive measures to reduce the risk of IE should consists of following:

1-Define the susceptible patients: those patients at high risk to develop IE following dental treatment include those with rheumatic heart diseases , congenital heart diseases, cardiac surgery , prosthetic heart valves.
2-Provide oral hygiene instruction: in patients with significant gingival inflammation, oral hygiene should be initially limited to gentle procedures (oral rinses as chlorhexidin mouth rinse and gentle tooth brushing with soft brush). As gingival health improves, more aggressive oral hygiene may be initiated.
3-During periodontal treatment, recommended prophylactic antibiotic regimens should be practiced with all susceptible patients. The regiment used is the following:

Regimen	Antibiotic	Dosage
Standard oral regimen	amoxicillin	2 g 1 hour before procedure

Alternate regimen for patients allergic to amoxicillin or penicillin	Clindamycin or	600mg 1 hour before procedure
	azithromycin	500mg 1 hour before procedure
Patient unable to take oral medication	ampicillin	2g intramuscularly or intravenously within 30 minute before procedure
Patients unable to take oral medication and allergic to penicillin	clindamycin	600mg intravenously within 30 minute before procedure (must be diluted and injected slowly)

Patients with aggressive periodontitis often have high level of Aggrigatibacteractinomycetemcomitans in the sub-gingival plaque. This organism has been associated with IE and is often resistant to penicillin , therefore in patients with aggressive periodontitis who are also at risk for IE, it has been suggested using tetracycline 250mg four times daily for 14 days to eliminate or reduce A.a. , followed by the conventional prophylaxis protocol at the time of dental treatment.

4) Periodontal treatment should be designed according to the degree of severity and involvement of periodontal tissues:- periodontal therapy is prolonged procedure , it is mostly not a one day antibiotic regimen, multiple visits and easily elicit gingival bleeding , so periodontal treatment plans must be developed for patients susceptible to IE and as follows:

a- In order to reduce the wide range systemic effect of periodontal diseases in these patients, teeth with severe periodontitis and poor prognosis have to be extracted rather than retained and treated.

b-All periodontal treatment procedures (including probing) require antibiotic prophylaxis. Pretreatment chlorhexidin mouth rinse are recommended before all procedure because it reduce the presence of bacteria on mucosal surfaces.

c-Reduce the number of visits required so that to minimize the risk of developing resistant bacteria.

d-It is preferably that the appointments allowed between 10-14 days, if it is not possible then select an alternative antibiotic regimen.

e- The need for antibiotic prophylaxis before suture removal is controversial when possible use the resorbable suture in such patients.

f- Regular recall appointment are important with reinforcement on good oral hygiene to maintain periodontal health.

2- Renal disease

Patients with chronic renal failure have a progressive diseases that may require kidney transplantation or dialysis. The patients who are receiving hemodialysis require special precautions. Those patients have a high incidence of viral hepatitis, anemia, and prolonged hemorrhage. The risk of hemorrhage is related anticoagulants during dialysis. Also they have either an internal arteriovenous fisula ar an external arteriovenous shunt. This shunt is often located in the arm and must be protected from trauma.

The management of those patients will be as follows:

1-Consult with patient physician.
2-Screen for hepatitis B surface antigen and antibodies prior to any treatment.

3-Avoid drugs that metabolized by the kidney ex. Tetracycline , streptomycin, aminoglycoside, aspirin……

4-Provide antibiotic prophylaxis to prevent infective endocarditis .

5-Screen for bleeding disorder(bleeding time and platelets time)
(normally bleeding time= 1-6 seconds, platelets =140,000-400000/mm)

6- Monitor blood pressure because those patients usually hypertensive.

Patients with renal transplantation take immunosuppressive drugs that greatly reduce resistance to infection, So management of those patients will be as follows:

1-Prophylactic antibiotic to prevent infection (prescribed by the physician)

2- May need supplemental corticosteroid .

3-Teeth with furcation involvement , periodontal abscesses should be extracted if it is not savable before transplantation to reduce possibility of infection.

4-Surgical excision of the gingiva may be needed because of gingival overgrowth secondary to cyclosporine therapy.

3-Endocrine disorders

a)Diabetes Mellitus

The diabetic patients require special precautions before periodontal therapy. The two major types of diabetic type 1 (formerly known as insulin-dependent diabetics) and type 2 (formerly called non-insulin dependent diabetic). Diabetic patients are managing their blood glucose levels (glycaemia) through diet, oral agent and insulin therapy. The classic signs of diabetic include polydipsia (excessive thirst). Polyuria (excessive urination), and polyphagia (excessive hunger with unexplained weight loss). If the patients has any of these signs and symptoms , physician consultation is indicated for further investigation because periodontal therapy has limited success in the presences of undiagnosed or poorly controlled diabetic. If the patients is suspected of having undiagnosed diabetic , following procedures should be performed:

1-Consult patients physician

2-Analyze laboratory tests

a) Fasting blood glucose ≥126mg/dl. Fasting is defined as no caloric intake for at least 8 hours (normal fasting glucose 70-100mg/dl).

b) Symptoms of diabetic plus non fasting plasma glucose ≥200mg/dl. Non fasting glucose may be drawn at any time of the day without regard to time since the least meal.

c) 2 hours postprandial glucose tolerance test. (the glucose level is measured immediately before and 2 hours after a person drinks a liquid containing 65 g of glucose dissolved in water). Normal 2 hours postprandial glucose is ≤140mg/dl.

3- Provide emergency periodontal therapy only for such patient like acute periodontal abscess until diagnosed is established.

If a patients is known to have diabetic, it is important to determine the level of glycemic control before initiating periodontal treatment. The primary test used to assess glycemic control in a known diabetic individuals is the glycosylated or glycated hemoglobin test (HbA1c) is a fraction of hemoglobin that reflected blood glucose concentration over the preceding 6 to 8 weeks and may provide an indication of the potential response to periodontal therpy. Well-controlled diabetic patient (HbA1c ≤ 8.5%) usually respond to therapy in a manner similar to non diabetic individuals. Poorly controlled patients (HbA1c ≥8.5%) often have a poor response to treatment, with more postoperative complications.

b) Thyroid disorders:

Hyperthyroidism or thyrotoxicosis may increase risk for hypertension, angina, congestive heart failure. So

1-Avoid any periodontal treatment for patients with thyrotoxicosis until good medical control.

2-Avoid epinephrine in completely treated patient.

3-Avoid stress and control periodontal infection to prevent the occurrence of thyrotoxic crisis in untreated patient.

4- Once under good medical management , patient may receive dental treatment.

In Hypothyroidism

1-Avoid stress and infection to prevent the occurrence of hypothyroid coma.

2-Avoid narcotic and tranquilizer in untreated hypothyroid patients because of inability to tolerance drugs.
3-In patients under good medical management, dental treatment may be performed.

4) Pregnancy

The aim of periodontal therapy for pregnant woman is to reduce the exaggerated inflammatory response of the periodontal tissues to local factor which related to hormonal changes associated with pregnancy.

-The second trimester is the safest time for treatment (scaling. Polishing, root planning) while surgical procedures should be postponed after delivery.

-In the third trimester, treatment should is not advisable because of supine hypotension syndrome of pregnancy , loss of consciousness may occur due to pressure of the uterus on inferior vena cava.

-No medication is given that the placenta and affect the fetus.

-No radiograph unless necessary with persuasions.

5) Hemorrhagic disorders:

Patients with a history of bleeding problems caused by disease or drug should be managed to minimize risks of hemorrhage. Identification of these patients via the health history. Clinical examination and clinical laboratory tests is important. Health questioning should cover

1-History of bleeding after previous surgery or trauma.

2-Past and present drug history.

3-History of bleeding problems among relatives.

4-Illnesses associated with potential bleeding problems.

Clinical examination should detect the existence of jaundice, ecchymosis, petechial, spontaneous gingival bleeding.

Laboratory tests include bleeding time, prothrombin time, complete blood count, partial thromoboplastin time and coagulation time. Bleeding disorders may include the following:

1-Hemophillia **A** (result in a deficiency of coagulation factor VIII).

2-Hemophillia **B** (result in a deficiency of factor IX).

3- Von Wille brands disease (result from deficiency of Von willebrand factor which mediates adhesion of platelet to the injured vessels wall).

4- Liver disease: Most coagulation factor are synthesized by the liver or it is the site for production of the clotting factors. Long term alcohol abusers or chronic hepatitis patients often demonstrated inadequate coagulation.

5-Patients taking anti-coagulant drugs: patients with prosthetic heart valves, or histories of myocardial infraction, stroke or thromboembolism are frequently placed on anticoagulation therapy using dicumarol and warfarin. These drugs are vitamin K antagonists.

Another drug is aspirin which interfers with normal platelete aggregation and can result in prolonged bleeding. For patients taking more than 325mg of aspirin per day, the drug should be discontinued at least 7 to 10 days before periodontal therapy in consultation with the physician.

6-Thrombocytopenia purpuras

Thrombocytopenia is defined as a platelet count ≤100.000mm. the purpuras could result from radiation, chemotherapy , leukemia, or infection and it is characterized by spontaneous petechiae (small red patches)or ecchymosis.

In general speaking for patients with bleeding disorders, never do any type of periodontal treatment unless consultation with the physician and it is preferable to do periodontal surgery if needed in the hospital.

.

8) Infectious diseases:

Because medical histories are often in accurate or in complete, all periodontal patients should be treated as they have as infectious diseases. Protection of patients, clinicians and office staff require use of universal (standard) persuasions for each patient. An example of these disease are hepatitis, AIDS, and Tuberculosis.

AIDS:

AIDS is characterized by impairment of the immune system. The human immunodeficiency viruses (HIV) was isolated in 1984 as the causative agent or virus of AIDS. Most of the patients develop long lasting acute illness with flu-like symptoms last for 10-14 days with enlarged lymph nodes, night sweating, weight loss, fever, malaise, and chronic diarrhea. Oral manifestation characterized by oral hairy leukoplakia and oral candidacies, necrotizing ulcerative gingivitis or periodontitis (NUG OR NUP) . Periodontal management of AIDS patients involves.

-Using full barrier technique.
-Care in use of all sharp instruments.
-Proper sterilization.
-Do not use ultrasonic instrumentation.

Tuberculosis

The patients with tuberculosis should receive emergency care only. Physician should be consulted for the result of sputum cultures for mycobacterium tuberculosis. When the results are negative, the patients may be treated normally. When the results are positive – we have to know that adequate treatment of tuberculosis require a minimum of 18 months with a post treatment follow up. So periodontal treatment should include emergency only. In general, in case of infectious diseases it is preferable to wear double gloves and double asks. The sterilization should done in autoclave 120-130 c for about one to two hours.

Hepatitis

Six distinct viruses causing viral hepatitis have been identified A,B,C,D,E, AND G viruses. These forms differ in their virology, epidemiology, and prophylaxis.
1-Hepatitis A and E are both self limiting infection with no associated chronic liver disease and these viruses transmitted via fecal-oral route.
2-Hepatitis B infection may result in chronic liver diseases, it transmitted mainly through hematogenous routes and through contaminated instruments or needle in the dental office. Hepatitis B vaccine is recommended for all care health workers.
3-Hepatitis D viruses require the presence of hepatitis B virus to survive and replicate because the virus genetic material is package within the hepatitis B virus surface antigen coating. So prevention of this virus depends strongly on hepatitis B virus vaccination.
4-Hepatitis C is the most serious infection due to high chronic infection rate. Only 15% of patients infected with this virus recover completely and 85% develop chronic infection which increase the risk for cirrhosis and liver failure. No vaccine is available for this virus.
5-Hepatitis G is a newly discovered virus and it is virology is not clearly understood and it known to be transmitted via blood.

>for patients with past history of hepatitis, consult the physician to determine the type of hepatitis, course, length of the disease and mode of the transmission.

>if the disease is in the active stage, do not provide periodontal treatment.

>for recovered type A or E hepatitis patients, perform routine periodontal care.

>for recovered type B and D hepatitis you must screen for HBsAG. If this test is positive , so the patient is infective.

>patient with positive anti-HBs may be treated routinely.

>patients with active hepatitis and need emergency treatment, we should do the following:

1-using full barrier techniques including masks, gloves, and eye glasses.
2-do not use ultrasonic instrument or air syringe so that not to transfer the infection by the saliva.
3-rinsing with chlorhexidin mouth wash is recommended.
4-when the procedure is complete, all instruments should be sterilized carefully.

Periodontal indices

Plaque index (Silness and Loe 1964)

- **This index measures the thickness of plaque on the gingival one third of the teeth.**
- used to evaluate the level and rate of plaque formation on tooth surfaces, and to test the efficacy of oral care products for removal and prevention of plaque deposits from these surfaces.
- Used on all teeth (28, wisdom teeth are excluded) or selected teeth (6 teeth) .
- Used on all surfaces (4)(M, B, D, L).
- • **Score 0** No plaque
- • **Score 1** A film of plaque adhering to the free gingival margin and adjacent area of the tooth, which can not be seen with the naked eye. But only by using disclosing solution or by using probe.
- • **Score 2** Moderate accumulation of deposits within the gingival pocket, on the gingival margin and/ or adjacent tooth surface, which can be seen with the naked eye.
- • **Score 3** Abundance of soft matter within the gingival pocket and/or on the tooth and gingival margin.

Gingival Index (GI) (Loe and Silness,1967)

- measures the degree of gingival iflammation. Tissues surrounding each tooth divided into 4 gingival scoring units: distal facial papilla, facial margin, mesial facial papilla, lingual gingival margin.
- **Score of gingival index**
- **Score 0** Normal gingiva
- **Score 1** Mild inflammation — slight change in color, slight edema. No bleeding on probing
- **Score 2** Moderate inflammation — redness, edema and glazing. Bleeding on probing
- **Score 3** Severe inflammation — marked redness and edema. Ulceration. Tendency to spontaneous bleeding.

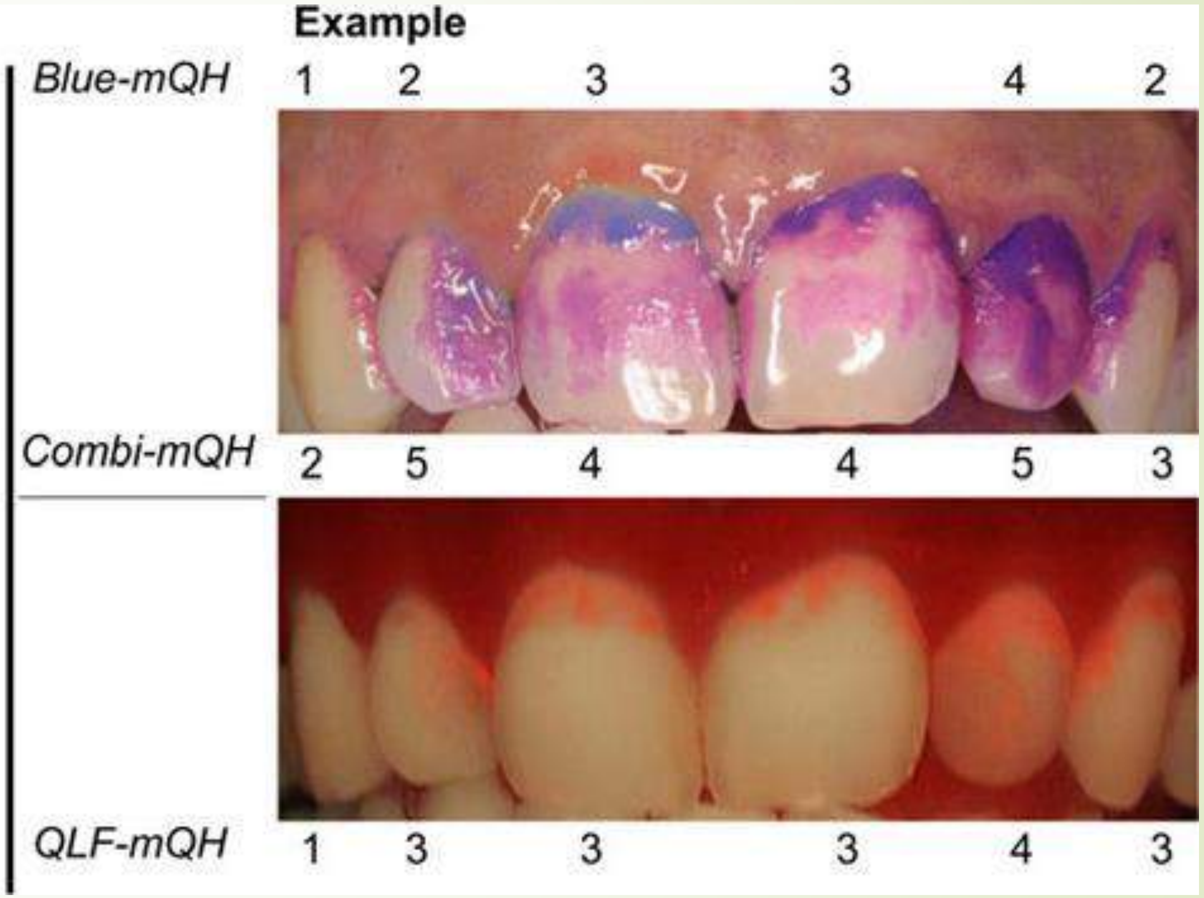
The GI may be used for the assessment of prevalence and severity of gingivitis in populations, groups and individuals.

Plaque index (O'leary)

- a commonly used oral hygiene index for assessing oral health skills. This index provides sufficient information for patient education.
- Suitable disclosing solusion is painted on all exposed tooth surfaces.
- The operator (using an explorer or a tip of a probe) examined each stained surface for soft accumulations at the dentogingival junction. When found, they are recorded by making a dash/red colour in the appropriate spaces on the record form.

Plaque index (Quigley Hein)

Score	
0	No plaque at the cervical margin
1	Separate flecks of plaque at the cervical margin of the tooth
2	A thin continuous band of plaque (≤1 mm) at the cervical margin of the tooth
3	A band of plaque wider than 1 mm but covering less than one-third of the crown of the tooth
4	Plaque covering at least one-third but less than two-thirds of the crown of the tooth
5	Plaque covering two-thirds or more of the crown of the tooth



Bleeding on probing (BOP)

- A periodontal probe is inserted to the —bottom of the gingival/periodontal pocket by applying light force and is moved gently along the tooth (root) surface. If bleeding is provoked upon retrieval of the probe, the site examined is considered —BoP — positive and, hence, is inflamed

Calculus Index (CI)

- Calculus is mineralized material on the tooth surface.
- The calculus index refers to the amount of calculus on a tooth.
- **CI 0** — No observable calculus.
- **CI 1** — Supragingival calculus covering not more than 1/3 of the exposed tooth surface.
- **CI 2** — Supragingival calculus covering more than 1/3 but not more than 2/3 of the exposed tooth surface or presence of flecks of subgingival calculus.
- **CI 3** — Supragingival calculus covering more than two-thirds of the exposed tooth surface or a continuous heavy band of subgingival calculus around the cervical portion of the tooth.

Probing pocket depth

- The probing depth, that is the distance from the gingival margin to the bottom of the gingival sulcus/pocket, is measured to the nearest millimetre by means of a graduated periodontal probe.
- The probe should be inserted parallel to the vertical axis of the tooth and "walked" circumferentially around each surface of each tooth to detect the areas of deepest penetration.This turn means that single-rooted teeth have to be examinated at four sites at least (e. g. mesial, buccal, distal, and oral) and multirooted teeth at six sites at least (e. g. mesiobuccal, buccal, distobuccal, distooral, oral, and mesio-oral)

Clinical attachment loss (CAL)

- is amore accurate indicator of the periodontal support around the tooth than probing depth alone.
- CAL is measured from a fixed point on the tooth that doesn't change, the CEJ.
- To calculate CAL, two measurements are needed:
- **1-In recession :** probing depth + gingival margin to the CEJ (add)
- **2- In tissue overgrowth :** probing depth – gingival margin to the CEJ(subtract)

BASIC PERIODONTAL EXAMINATION (BPE)

- The BPE is a simple and rapid screening tool that is used to indicate the level of examination needed and to provide basic guidance on treatment need. Please note; the **BPE does not provide a diagnosis**.
- 1. The dentition is divided into 6 sextants:

  upper right (17 to 14), upper anterior (13 to 23), upper left (24 to 27)

  lower right (47 to 44), lower anterior (43 to 33), lower left (34 to 37)
- 2. All teeth in each sextant are examined (with the exception of 3rd molars).
- 3. For a sextant to qualify for recording, it must contain at least 2 teeth. (If only 1 tooth is present in a sextant, the score for that tooth is included in the recording for the adjoining sextant).

BASIC PERIODONTAL EXAMINATION (BPE)

- 4. A WHO BPE probe is used (World Health Organisation probe). This has a “ball end” 0.5 mm in diameter, and a black band from 3.5 to 5.5 mm. Light probing force should be used (20-25 grams).
- 5. The probe should be “walked around” the sulcus/pockets in each sextant, and the highest score recorded. As soon as a code 4 is identified in a sextant, the clinician may then move directly on to the next sextant, though it is better to continue to examine all sites in the sextant. This will help to gain a fuller understanding of the periodontal condition, and will make sure that furcation involvements are not missed. If a code 4 is not detected, then all sites should be examined to ensure that the highest score in the sextant is recorded before moving on to the next sextant.

WHO BPE probe

BASIC PERIODONTAL EXAMINATION (BPE)

Scoring codes

0	No pockets >3.5 mm, no calculus/overhangs, no bleeding after probing *(black band completely visible)*
1	No pockets >3.5 mm, no calculus/overhangs, but bleeding after probing *(black band completely visible)*
2	No pockets >3.5 mm, but supra- or subgingival calculus/overhangs *(black band completely visible)*
3	Probing depth 3.5-5.5 mm *(black band partially visible, indicating pocket of 4-5 mm)*
4	Probing depth >5.5 mm *(black band entirely within the pocket, indicating pocket of 6 mm or more)*
*	Furcation involvement

Both the number and the * should be recorded if a furcation is detected - e.g. the score for a sextant could be 3* (e.g. indicating probing depth 3.5-5.5 mm PLUS furcation involvement in the sextant).

An example BPE score grid might look like:

4	3	3*
-	2	4*

When to record the BPE ?

- All new patients should have the BPE recorded
- For patients with codes 0, 1 or 2, the BPE should be recorded at least annually
- For patients with BPE codes of 3 or 4, more detailed periodontal charting is required: - Code 3: record full probing depths (6 sites per tooth) in the sextant(s) where the code 3 was recorded, in addition to recording the BPE in those sextants with scores 0, 1 or 2 . Code 4: if there is a code 4 in any sextant, then record full probing depths (6 sites per tooth) throughout the entire dentition
- BPE cannot be used to assess the response to periodontal therapy because it does not provide information about how sites within a sextant change after treatment. To assess the response to treatment, probing depths should be recorded at 6 sites per tooth pre- and post-treatment
- For patients who have undergone initial therapy for periodontitis (i.e. who had pretreatment BPE scores of 3 or 4), and who are now in the maintenance phase of care, then full probing depths throughout the entire dentition should be recorded at least annually.

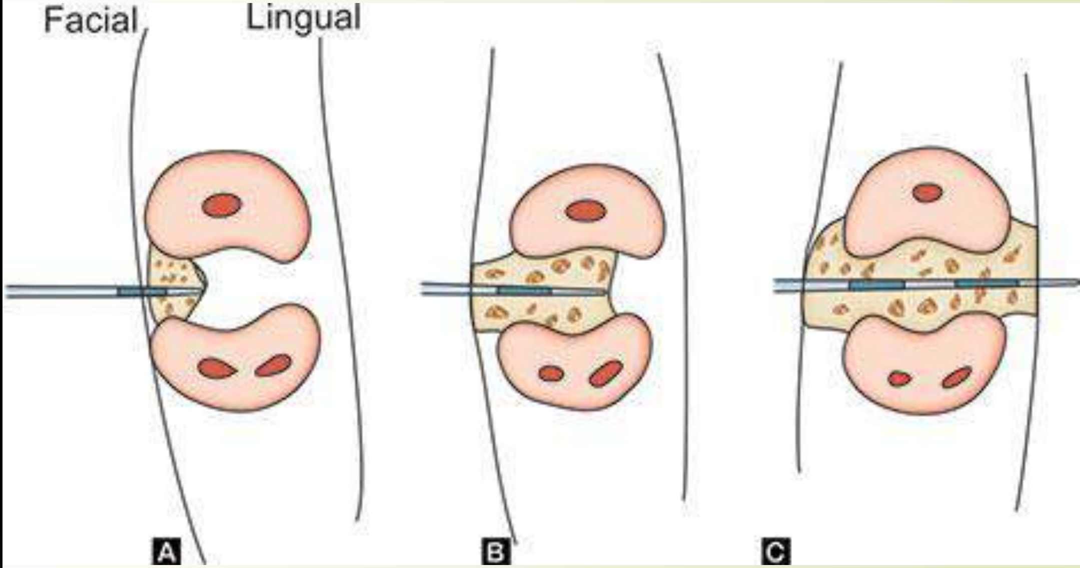
Guidance on interpretation of BPE score

0	No need for periodontal treatment
1	Oral hygiene instruction (OHI)
2	OHI, removal of plaque retentive factors, including all supra- and subgingival calculus
3	OHI, root surface debridement (RSD)
4	OHI, RSD. Assess the need for more complex treatment; referral to a specialist may be indicated.
*****	OHI, RSD. Assess the need for more complex treatment; referral to a specialist may be indicated.

Furcation involvement index

Glickman`s Classification(1953)

- Grade I Incipient Furcation
- Grade II cul-de-sac
- Grade III Communicating or Through and Through Furcation
- Grade IV Recession and clinically visible furcation



Recession index (Miller)

Table 1. Miller's classification of gingival recession defects.

	Symptoms	Treatment	Success
Class I	Recession that does not extend to the mucogingival junction	Complete root coverage is achievable	100%
Class II	Recession that extends to or beyond the mucogingival junction, with no periodontal attachment loss (i.e bone, soft tissue)	Complete root coverage is achievable	100%
Class III	Recession that extends to or beyond the mucogingival junction, with periodontal attachment loss in the interdental area or malpositioning of the teeth	Only partial root coverage possible to the height of the contour of interproximal tissue	50-70%
Class IV	Recession that extends to or beyond the mucogingival junction, with severe bone or soft-tissue loss in the interdental area and/or severe malpositioning of the teeth	Root coverage is unpredictable and requires adjunctive treatment (ie orthodontics)	<10%

Recession index (Miller)

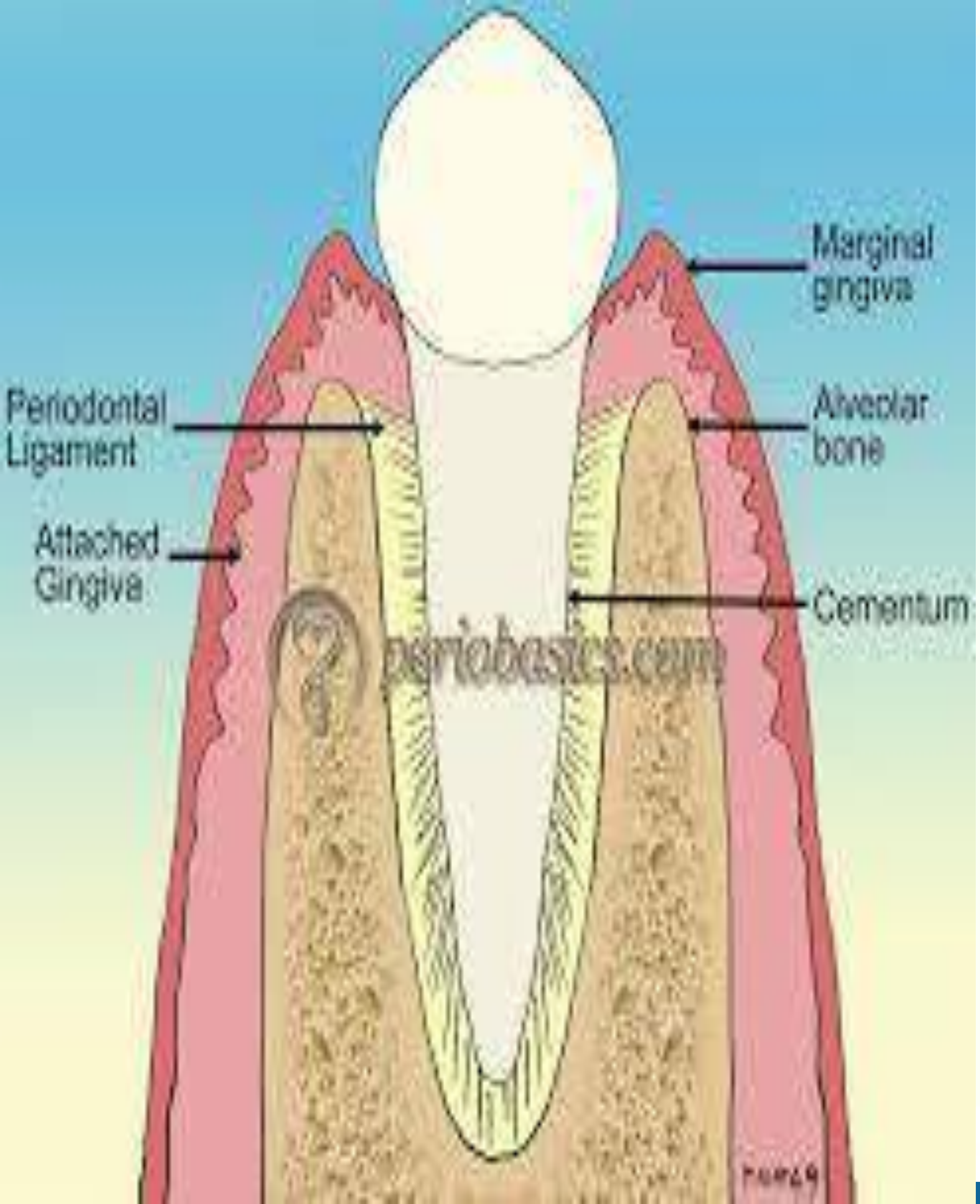
Thank you

بسم الله الرحمن الرحيم

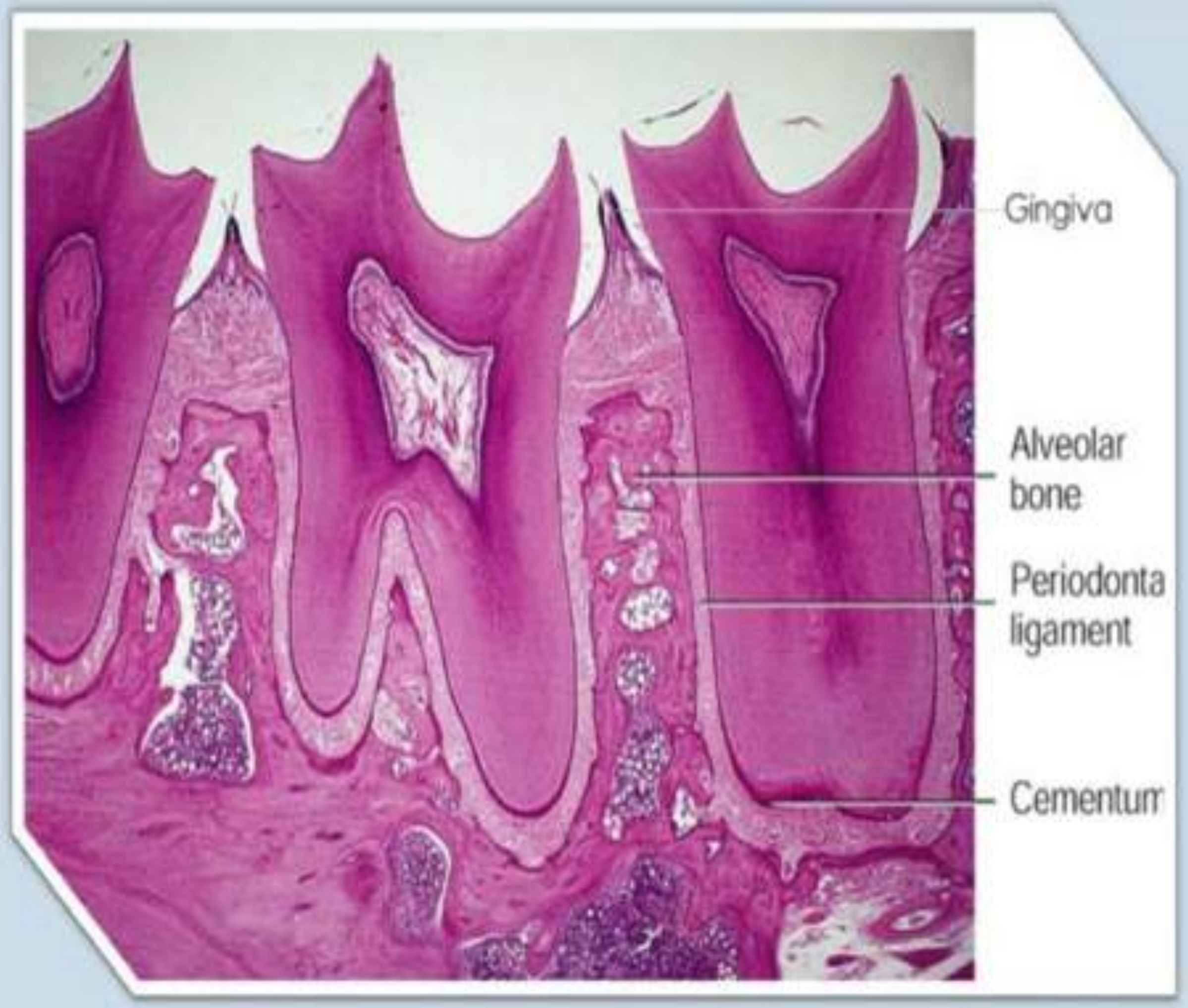
PERIODONTAL LIGAMENT

أعداد:

د. نور صباح أرحيم



PERIODONTIUM



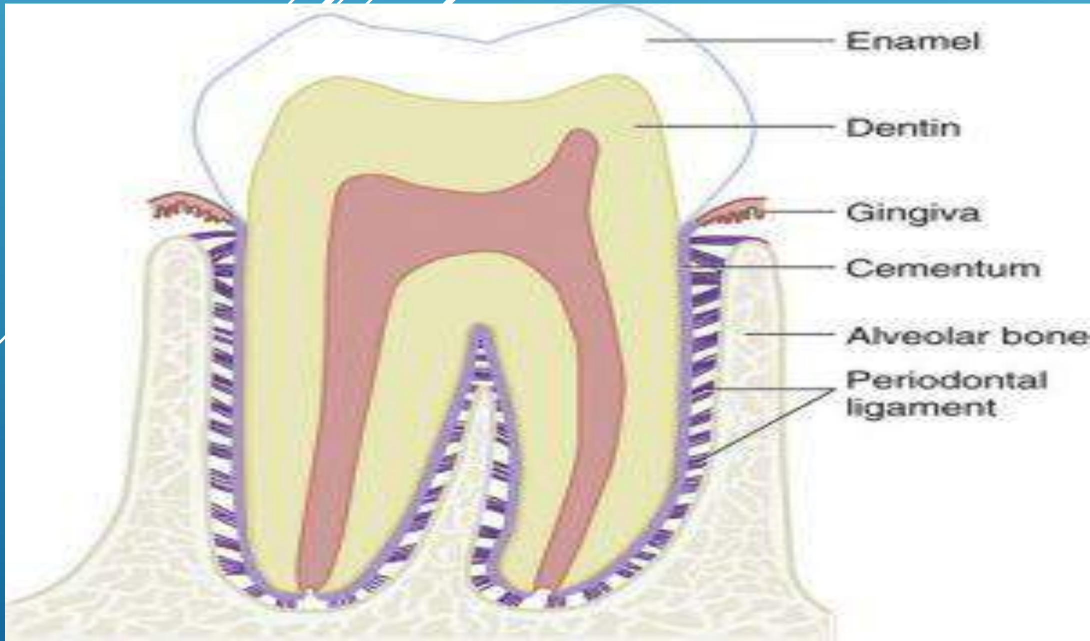
PERIODONTAL LIGAMENT (PDL)

- **It is a connective tissue surrounding the root and connecting it with the bone.**
- **It is consist primarily of:**

1- Bundles of intermingling collagen fibers.

2- Cellular elements.

3- Ground substance.



DEVELOPMENT OF PDL

- **The PDL and the cementum develop from follicular sac which derived from mesenchyme. The development of PDL occur during root formation and tooth eruption.**

FIBERS OF PDL

((A)) The Majority of fibers in PDL are collagens fibers (Principles fibers of PDL).

((B)) Elastic fibers.

((C)) Oxytalan fibers.

PDL

SHARPEY´S FIBERS:

It is the terminal portions of the principles fibers that insert into the cementum and bone.

Intermediate plexus:

It is the parts of the principle fibers, one is located toward the cementum and the other toward the alveolar bone and spliced together in the mid way between the cementum and bone to form intermediate plexus.

Sharpey's fibers

- The ends of the periodontal fibers that are embedded in alveolar bone and cementum are called Sharpey's fibers.

- On the cementum side these Sharpey's fibers are much thinner in diameter and insert at closer intervals as compared with the alveolar bone side

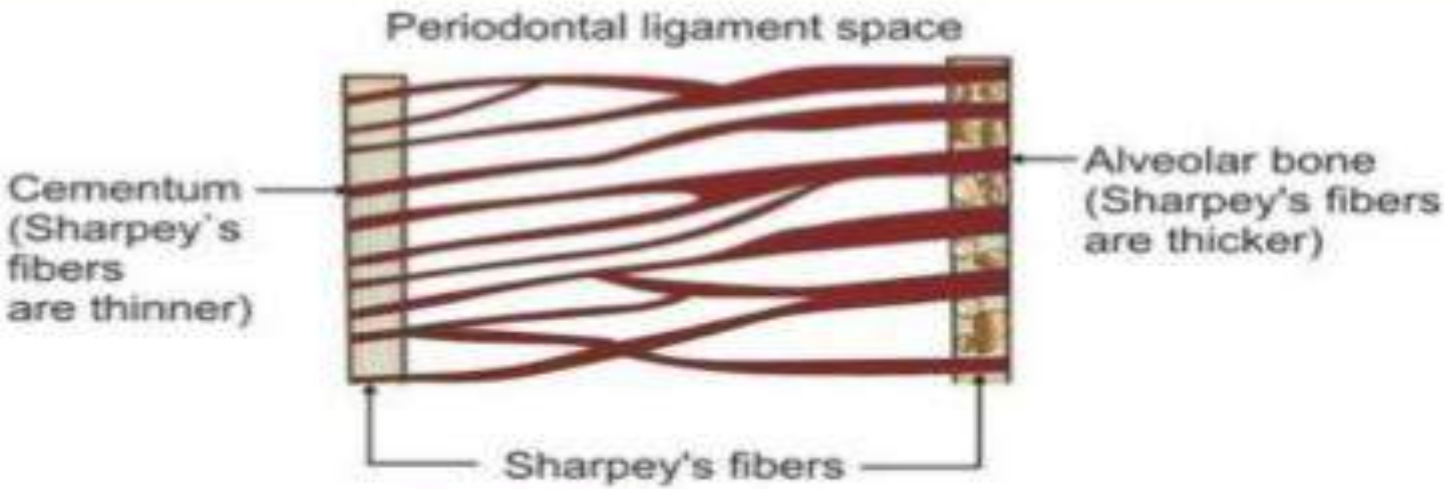
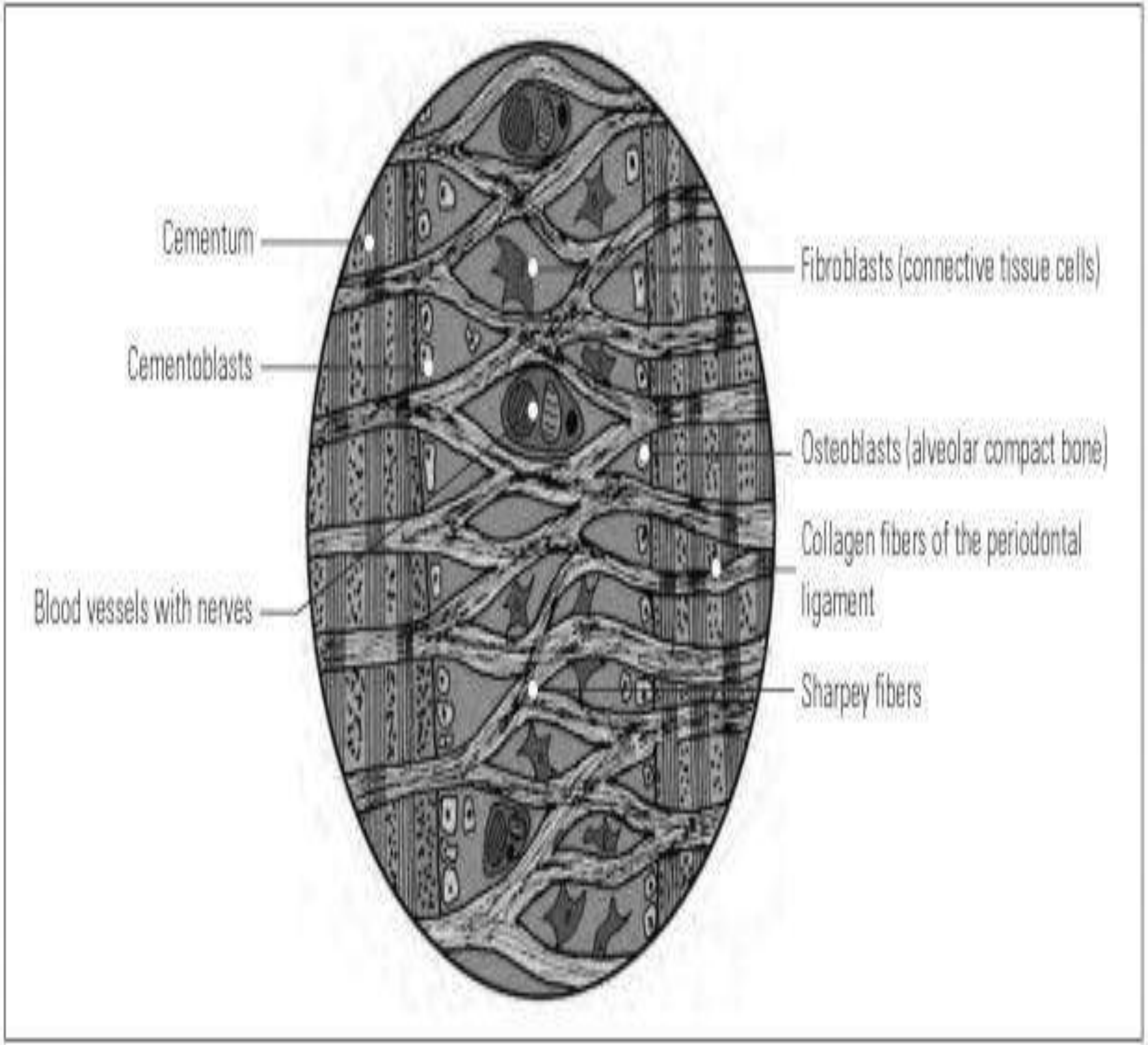
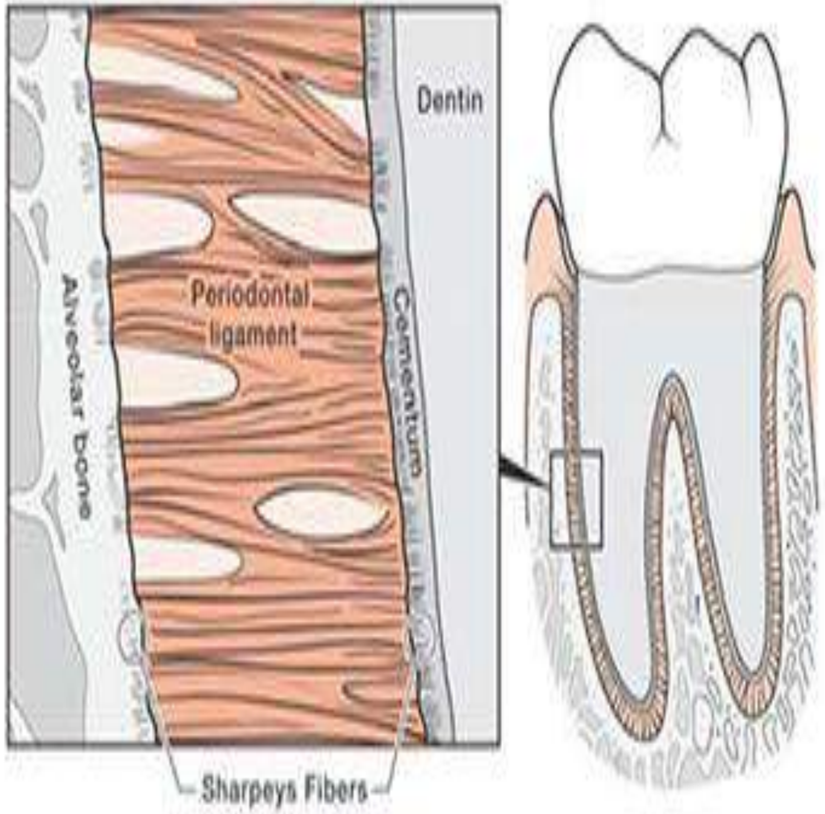


Fig. 2.3: Sharpey's fibers



Sharpey's Fibers



THE PRINCIPLES FIBERS OF THE PDL:

1- Alveolar crest fibers (ACP).

2- Horizontal fibers (HF).

3- Oblique fibers (OF).

4- Apical fibers (AF).

5- Interradicular fibers (IF).

6-Trasseptal Fibers (TF).

1-Alveolar crest fibers (ACF):

They extend obliquely from the cementum to the crest of alveolar bone, they run in an apical direction. They prevent the extrusion of the tooth and resist lateral tooth movement.

2-Horizontal fibers (HF):

They extend at right angles to the long axis of the tooth from the cementum to the alveolar bone.

3-Oblique fibers (OF):

They are the largest group in the PDL, extend from the cementum in a coronal direction obliquely to the bone. They withstand the vertical masticatory forces.

4-Apical groups (AP):

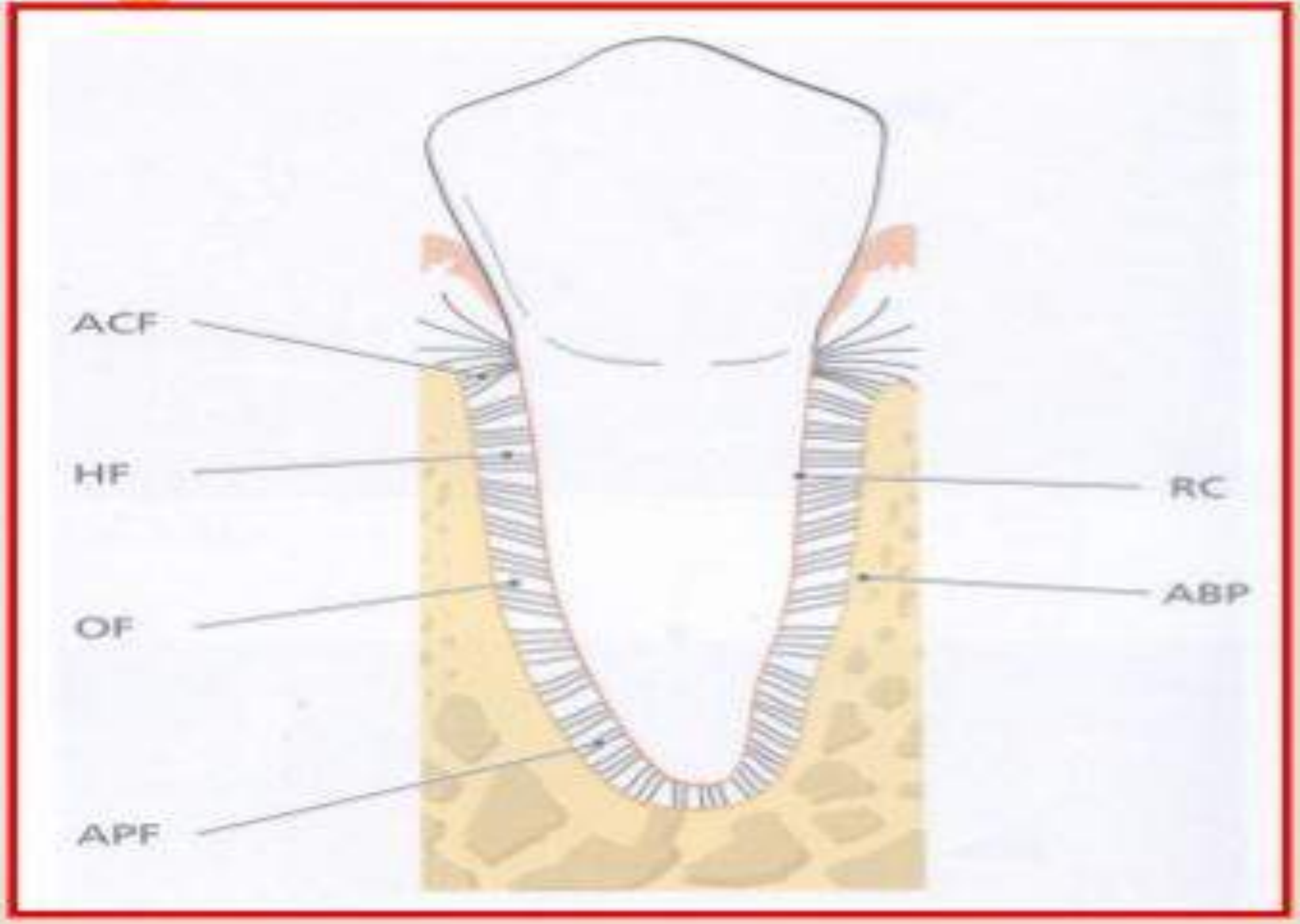
They radiate from the cementum to the bone at the apical region of the socket.

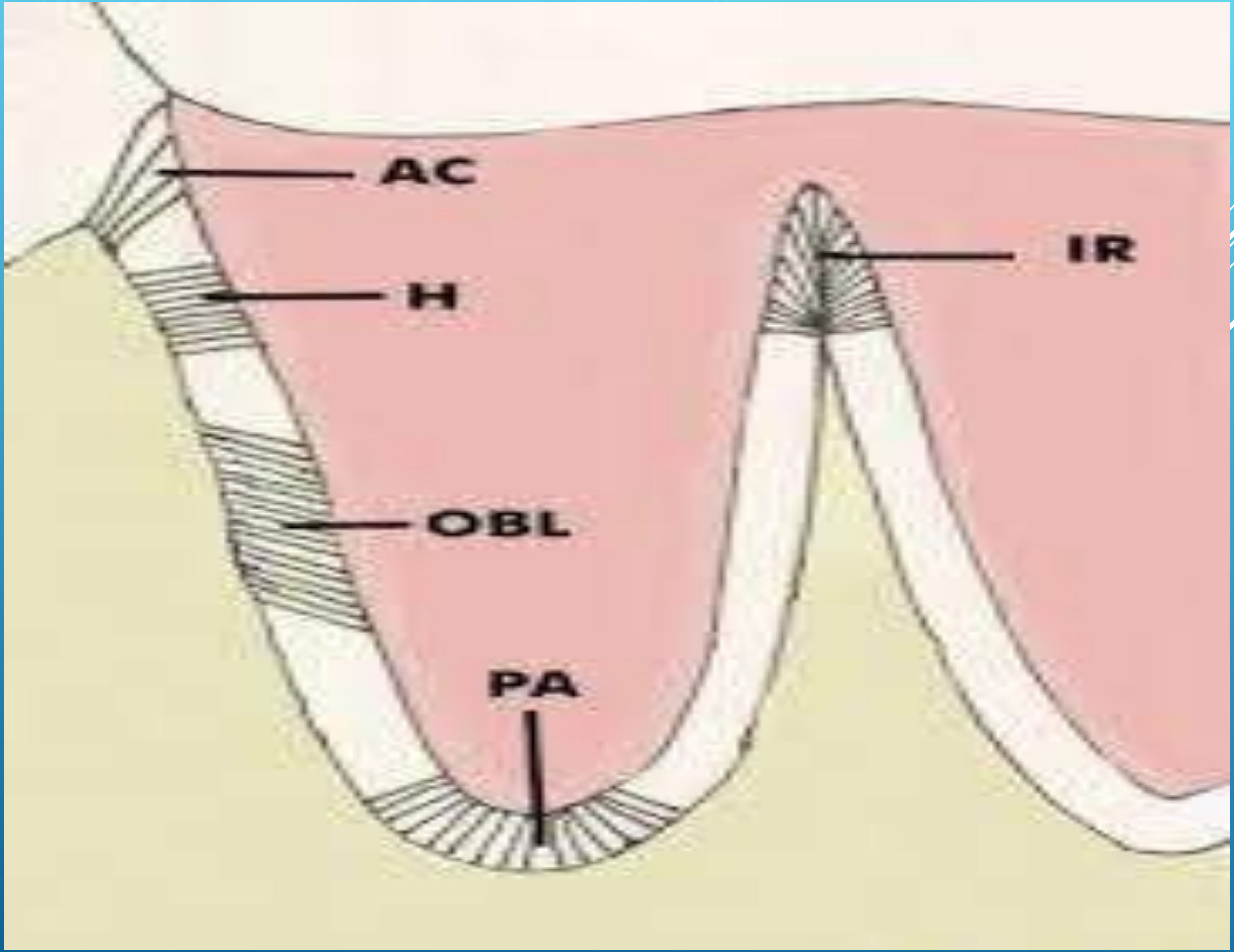
5-Interredicular fibers (IF):

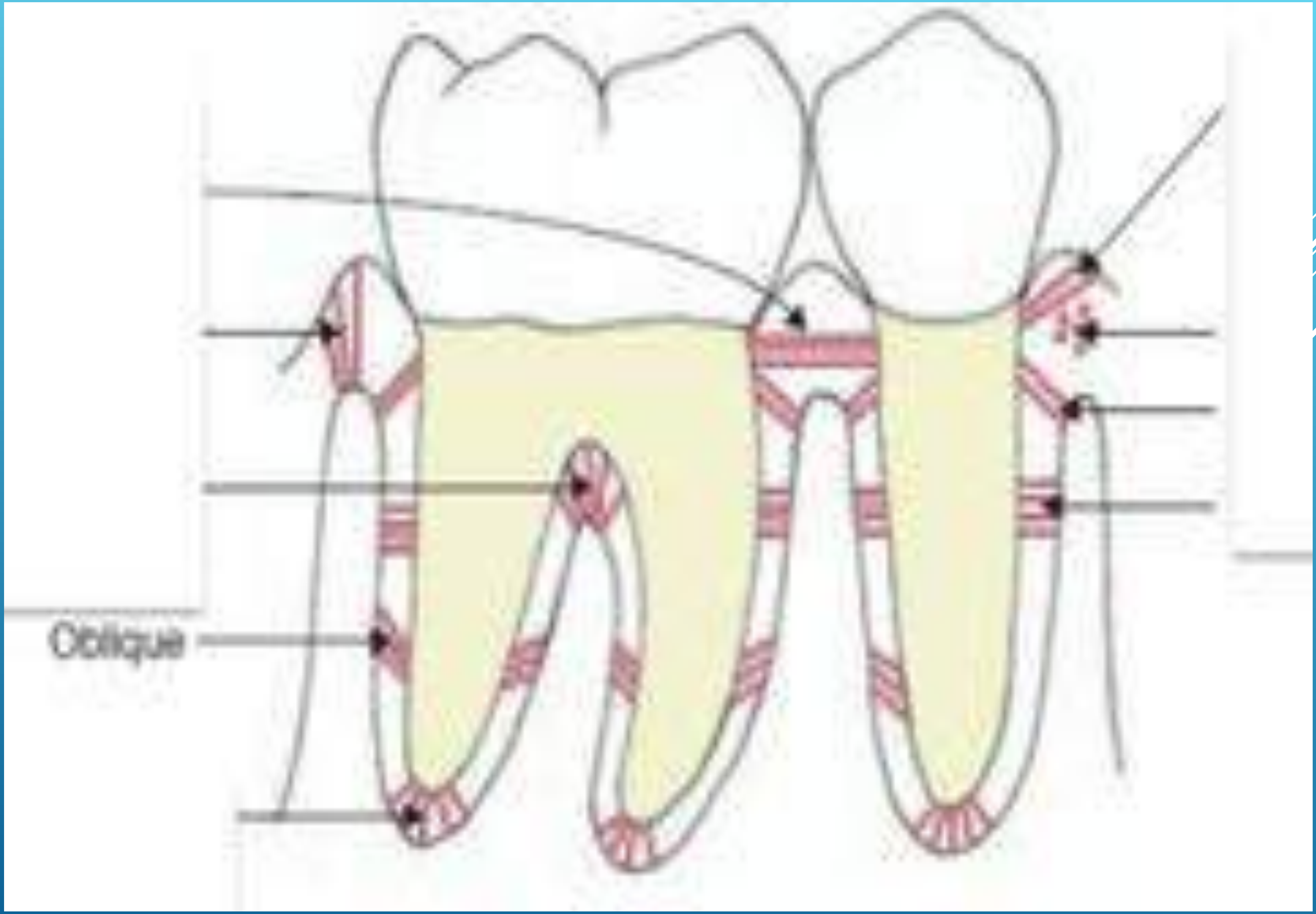
They run from the cementum to the bone in the furcation areas of multirooted teeth.

Principal fibers of PDL

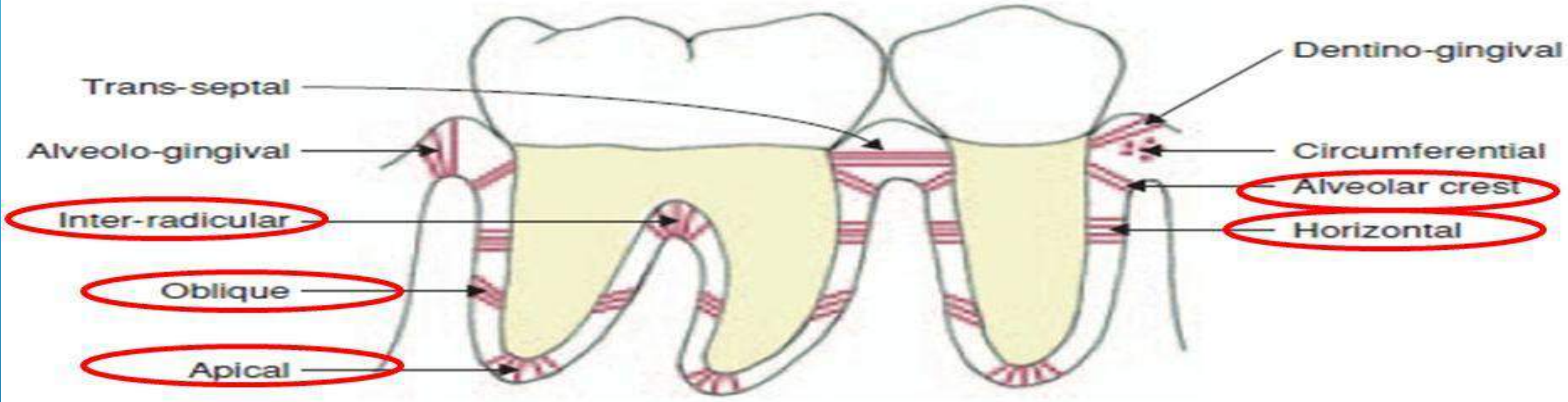
- Trans septal
- Alveolar crest group
- Horizontal
- Oblique
- Apical
- Inter – radicular




AC
H
OBL
IR
PA


Oblique

A- THE PRINCIPAL FIBERS



1- principle fiber bundle of the PDL:

i. The alveolar crest group.
ii. The horizontal group.
iii. The oblique group.
iv. The apical group.
v. The interradicular group.

B-Elastic fibers:

These are relatively few, and associated with the blood vessels.

C- Oxytalan fibers:

These immature forms of the fibers are thought to regulate vascular flow.

CELLULAR ELEMENTS OF PDL:

1- Synthetic cells

(a)Osteoblasts

(b) Fibroblasts

(c) Cementoblasts

2- Resorptive cells

(a)Osteoclast

(b)Fibroblasts

(c)Cementoclasts

3- Epithelial rests of Malassez cells.

4-Immune system cells.

- *Osteoblasts:* these cells cover the osseous surface of the PDL and are responsible for the formation of the alveolar bone.
- *Fibroblast:* these are the most common cells in the PDL. The main function of fibroblast is the production of various fibers such as collagen fiber, oxytalan fibers and elastic fibers.
- *Cementoblasts:* are seen lining the cementum surface of the PDL and are responsible for cementum deposition.
- *Osteoclasts:* these are large multinucleated cells and responsible for the bone resorption.
- *Fibroblaste:* these cells synthesize collagen and also possess the capacity to phagocytic old collagen fibers and degrade them by enzyme hydrolysis. The process of fibers resorption occur either during disease or physiological turnover.
- *Cementoclast:* cementum not remodeling as the alveolar bone and PDL but it undergoes continual deposition during life.

Epithelial rest malassez:

these are found close to the cementum . They are reminants of Hertwigs root sheath. They proliferate when stimulated and particitate in the formation of periapical cysts and lateral root cysts.

Immune system cells:

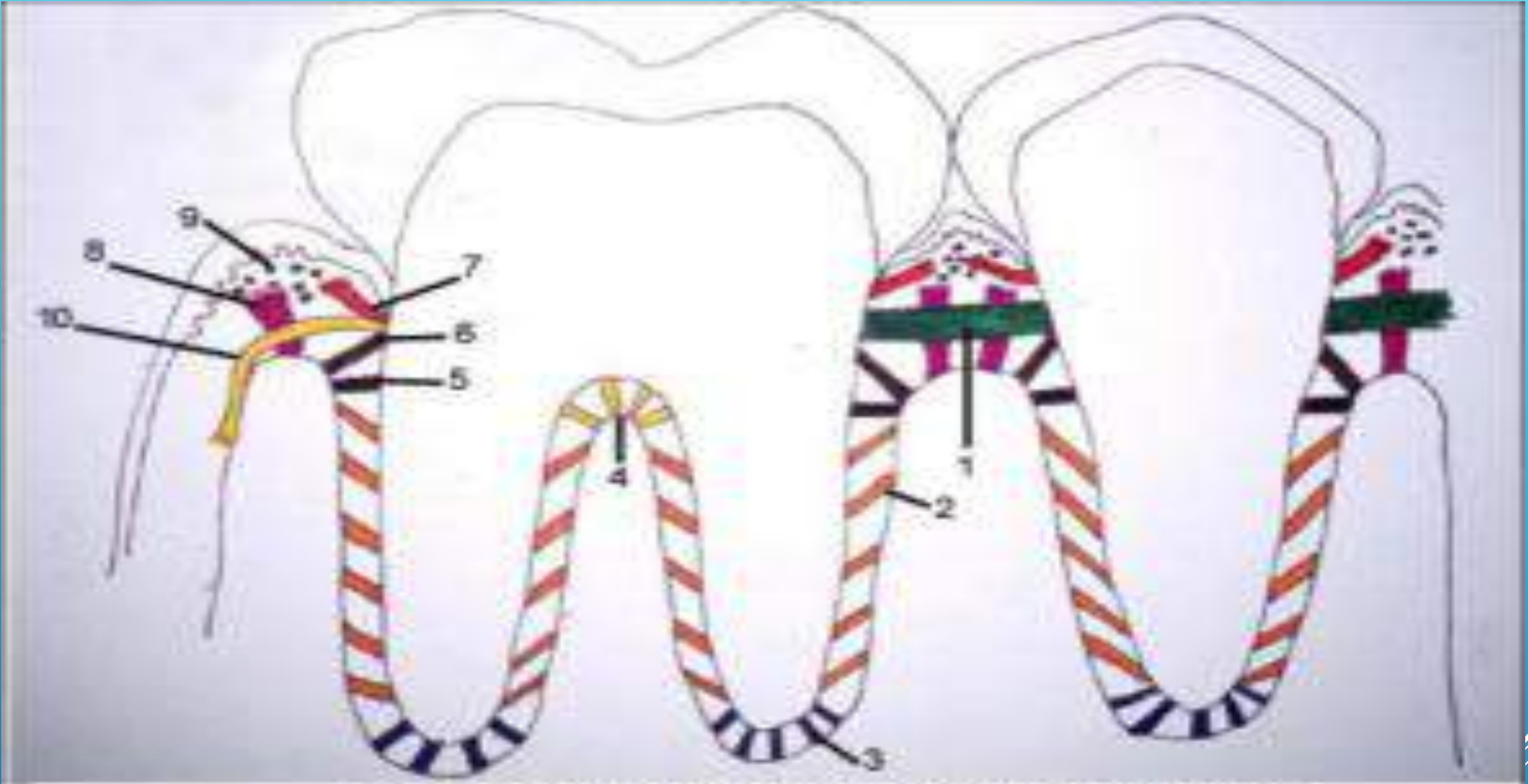
Include mast cells, macroghage , lymphocyte neutrophil .

GROUND SUBSTANCE OF PDL:

A- **Glycosaminglycan.**

B- **Glycoprotein.**

➡ **Water content 70%.**



Principal fibers of the periodontal ligament

1. transseptal fibers
2. oblique
3. apical
4. interradicular
5. horizontal
6. alveolar crest
7. dento-gingival (free gingival)
8. alveolar-gingival
9. circular
10. dento-periosteal

WIDTH OF PDL

- Width of PDL apace is about 0.25mm.
- In hyperfunction , the tooth has wider PDL space.
- In hypofunction , the tooth has narrow PDL space.
- The space widest at the cervical and apical portion of the root and narrowest at the middle.
- in physiological tooth migration , the PDL space in the mesial root is thinner than the PDL space in distal root.
- PDL space decrease with age.
- in orthodontic treatment, the pressure side have smaller PDL pace than tension side.
- in Implant and ankylois, there is no periodontal ligament space

Pdl space

Widened pdl space

Narrow PDL space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ELASTICITY OF PDL

1- Wavy coarse of the principal fibers.

2- Intermediate plexuses.

3-The Presence of oxytalan and Elastic fibers.

FUNCTIONS OF THE PDL

1-Physical function.

2- Formative and Remodeling function.

3- Nutritive function.

4- Sensory function.

Physiological function:

1-Transmission of occlusal forces to the bone.
2-Attachment of the teeth to the bone.
3-Resistance to the impact of occlusal forces(shock absorption).
4-Provision of a soft tissue (casing) to protect the vessels and nerves from injury by mechanical forces.

Formative and remodeling function

Cells of the PDL participate in the formation and resorption of cementum and bone which occur in physiological tooth movement , in accommodation of the periodontium to occlusal forces and in the repair of injury.

Nutritive function

The PDL supplies nutrition to the cementum, bone and gingiva by way of the blood vessels and provide lymphatic drainage.

Sensory function

The PDL is supplied with sensory nerve fibers which transmit tactile-pressure- pain sensation by the trigeminal pathway in addition the PDL is supplied with mechanoreceptor that transmit sense of localization.

BLOOD SUPPLY OF PDL

- The blood supply to the supporting structures of the tooth is derived from the inferior and superior alveolar arteries to the mandible and maxilla respectively and reach the PDL from three sources:

  (A)Apical vessels supply the apical region of the PDL.

  (B)The transalveolar vessels from the alveolar bone.

  (C)Anastomosing vessels from the gingiva.


Thank you

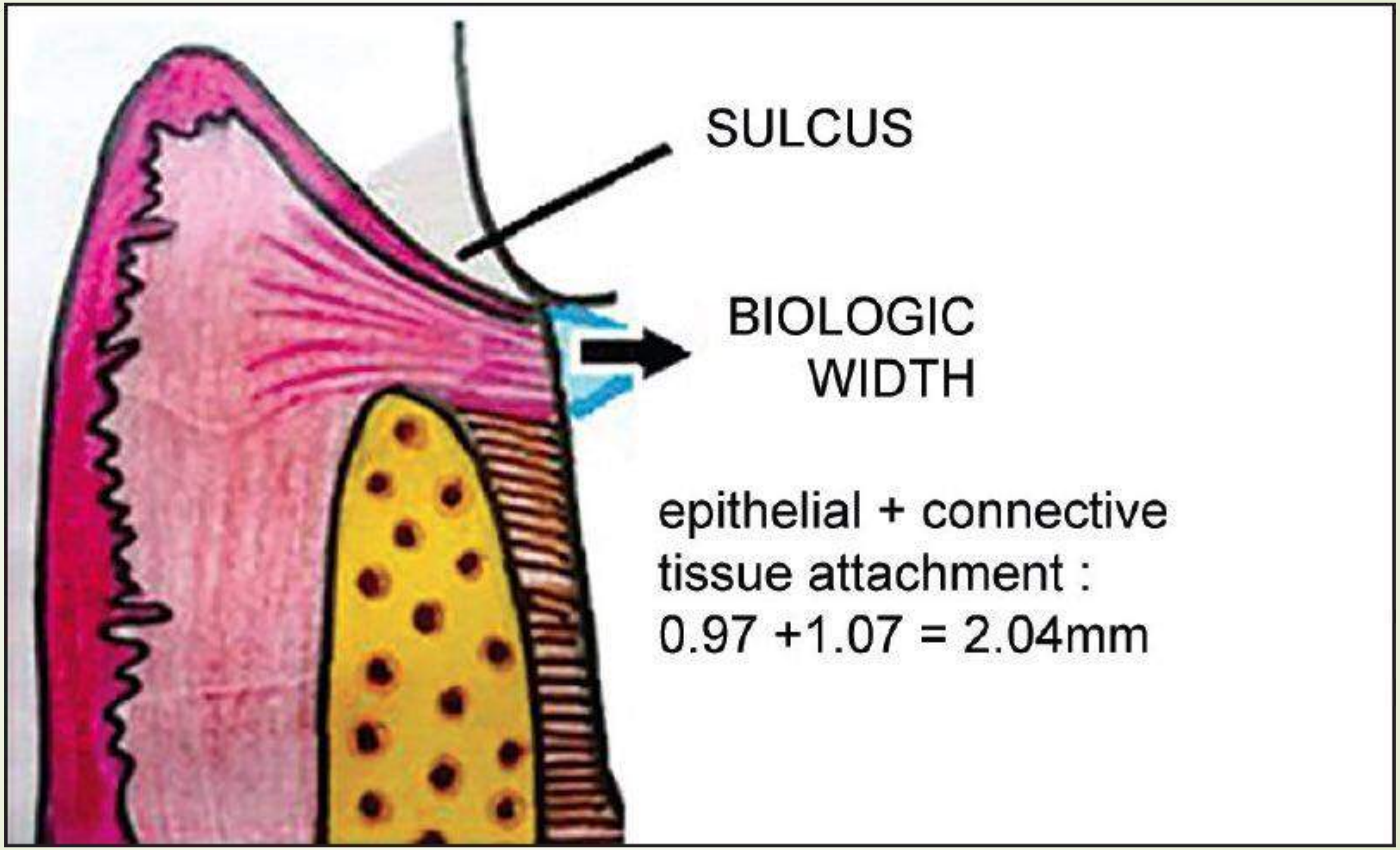
Gingival and periodontal pocket

م. سها أسود دهش العزاوي

B.D.S, MSC. PERIODONTOLOGY

Tooth gingival interface

- The interface between a tooth and the surrounding gingival tissue is a dynamic structure. The gingival tissue forms a crevice surrounding the tooth, resemble fluid-filled moat, The depth of this crevice, known as a sulcus, is in a constant state of flux due to microbial invasion and subsequent immune response. Located at the depth of the sulcus is the epithelial attachment, consisting of approximately 1 mm of junctional epithelium and another 1 mm of gingival fiber attachment, comprising the 2 mm of biologic width naturally found in the oral cavity.


SULCUS
BIOLOGIC
WIDTH
epithelial + connective
tissue attachment :
0.97 +1.07 = 2.04mm

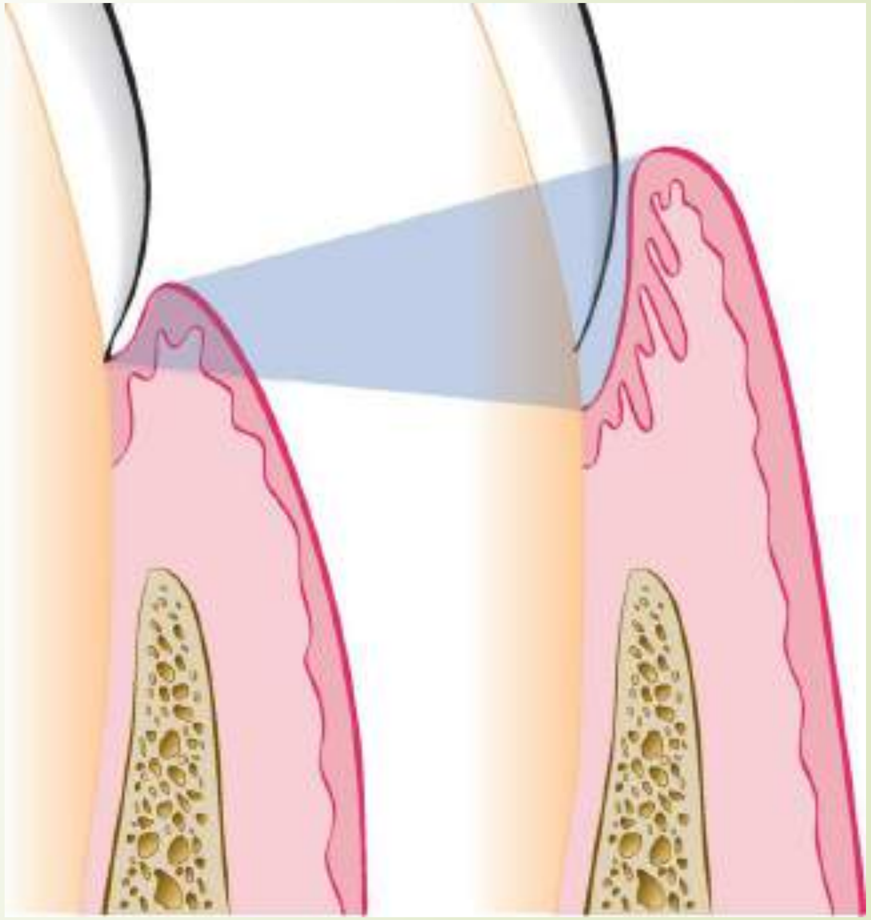
Gingival sulcus

A healthy sulcular depth is 3 millimeters or less. sulcular depths of 3 millimeters or less are readily self-cleansable with a properly used toothbrush or the supplemental use of other oral hygiene aids. When the sulcular depth is chronically in excess of three millimeters, regular home care may be insufficient to properly cleanse the full depth of the sulcus, allowing food debris and microbes to accumulate, forming dental biofilm. If accumulated microbes remain undisturbed in a sulcus for an extended period of time, they will penetrate and ultimately destroy the delicate soft tissue and periodontal attachment fibers. If left untreated, this process may lead to a deepening of the sulcus, recession, destruction of the periodontium, including the bony tooth socket, tooth mobility, and tooth loss.

- **Gingival and periodontal pockets** are dental terms indicating the presence of an abnormal depth of the gingival sulcus

pocket

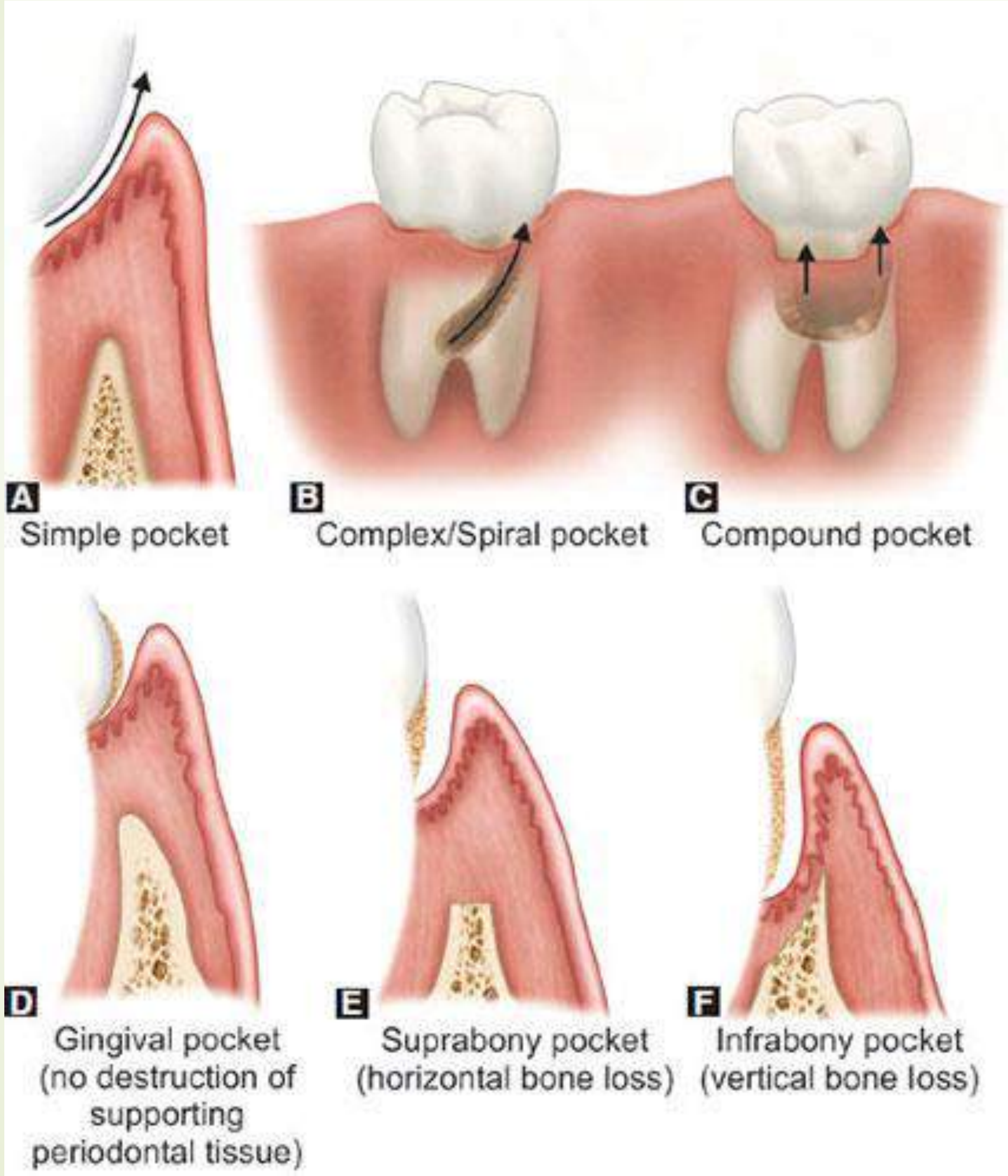
- It’s an inflammatory changes and apical migration of junctional epithelium; it is also defined as a pathological deepening of gingival sulcus, which occurs by coronal movement of gingival margin, apical displacement of gingival attachment, or both.



Classification:

- **1-According to the involved tooth surface:**
  - Simple pocket: involve one surface
  - Compound pocket: involve more than one surface
  - Complex or spiral pocket: originating on one surface and twisting around the tooth to involve one or more additional surface. most common in furcation areas.

Simple pocket Compound pocket Complex pocket


A
Simple pocket
B
Complex/Spiral pocket
C
Compound pocket
D
Gingival pocket
(no destruction of
supporting
periodontal tissue)
E
Suprabony pocket
(horizontal bone loss)
F
Infrabony pocket
(vertical bone loss)

- **2-According to its location:**

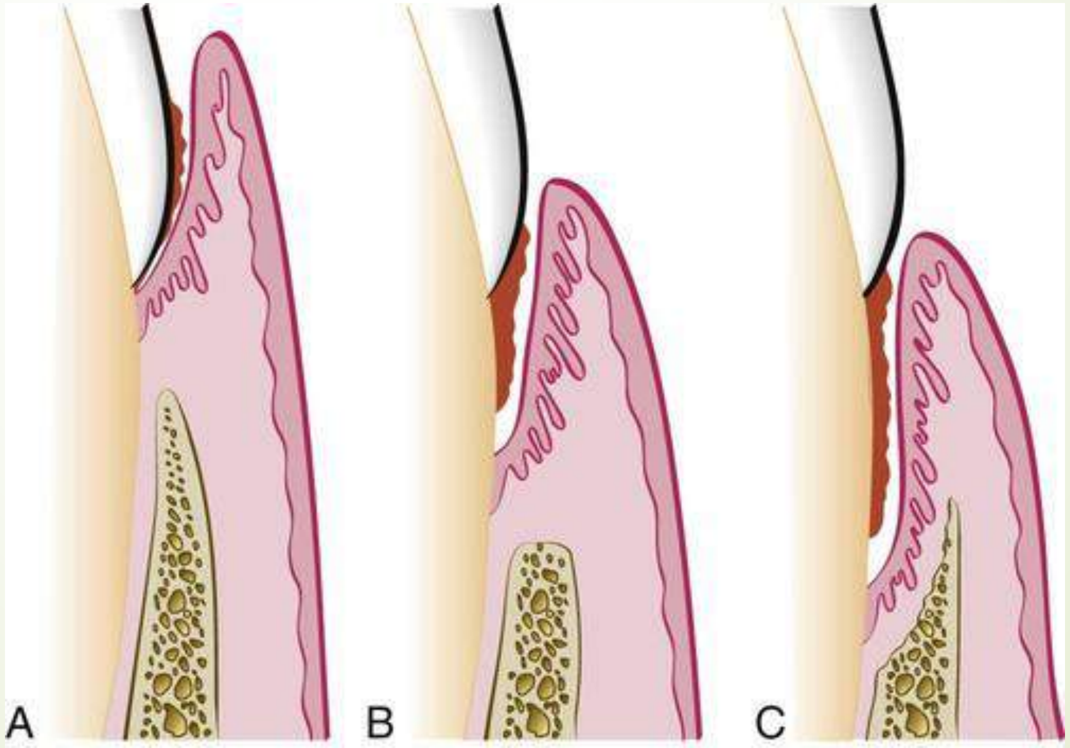
  A- **Gingival pocket:** which is formed by gingival enlargement without destruction of underlying periodontal tissue. The sulcus is deepened because of increased bulk of the gingiva.

- This phenomenon is also referred to as a false pocket or pseudopocket. The epithelial attachment does not migrate, it simply remains at the same attachment level found in pre-pathological health. The only anatomical landmark experiencing migration is the gingival margin in a coronal direction.
- In a gingival pocket, no destruction of the connective tissue fibers (gingival fibers) or alveolar bone occurs. This early sign of disease in the mouth is completely reversible when the etiology of the edematous reaction is eliminated and frequently occurs without dental surgical therapy.
- However, in certain situations, gingivectomy is necessary to reduce the gingival pocket depths to a healthy 1–3 mm.

- **B- periodontal pocket**, which is defined as a pathologically deepened gingival sulcus, is one of the most important clinical features of periodontal disease. All different types of periodontitis, share histopathologic features, such as tissue changes in the periodontal pocket, mechanisms of tissue destruction, and healing mechanisms. However, they differ with regard to their etiology, natural history, progression, and response to therapy.

3. According to its relation to alveolar crest:

- Suprabony pocket: also called supra crestal or supra alveolar. The base of the pocket is coronal to the level of underlying bone. The bone loss is horizontal
- Infrabony pocket: also known as sub crestal or intra alveolar pocket. The base of the pocket is apical to the level of adjacent bone. The bone loss is vertical.



Diagnosis/ detection of pockets

- Careful exploration with periodontal probe. (this method is accurate).
- Radiographic: pockets are not detected by the radiographic examination because pockets are soft tissue changes.
- A calibrated silver points or gutta percha points can be used with radiographic to assist in determining the level of attachment of periodontal pocket.

Pathogenesis

- The initial lesion in the development of periodontitis is the inflammation of the gingiva in response to a bacterial challenge. Changes involved in the transition from the normal gingival sulcus to the pathologic periodontal pocket are associated with different proportions of bacterial cells in dental plaque. Healthy gingiva is associated with few microorganisms, mostly coccoid cells and straight rods. Diseased gingiva is associated with increased numbers of Spirochetes and motile rods. However, the microbiota of diseased sites cannot be used as a predictor of future attachment or bone loss, because their presence alone is not sufficient for disease to start or progress.

Sequences in pathogenesis of periodontal pocket

- Accumulation of microorganisms on the supra gingival tooth surface and its extension into gingival sulcus.
- Inflammatory changes in the connective tissue wall of the gingival sulcus.
- Cellular and fluid inflammatory exudate causes degeneration of the connective tissue including the gingival fibers.
- Collagen fibers gets destroyed apical to the junctional epithelium and the area becomes occupied by the inflammatory cells and edema.
- The coronal portion of the junctional epithelium detaches from the root as the apical portion migrates.
- Polymorphonuclear neutrophils invade the coronal end of the junctional epithelium in increasing number.
- With continued inflammation the gingiva increase in bulk and the crest of the gingival margin extends coronally.
- The junctional epithelium continues to migrate along the root and separate from the root.

Mechanisms of collagen loss:

Two mechanisms involved:

- **First mechanism:**

Collagenases and other enzymes secreted by fibroblast, PMNs and macrophages in healthy and inflamed tissues, become extracellular and destroy collagen.

- **Second mechanism:**

Fibroblast phagocytize collagen fibers by extending cytoplasmic processes to the ligament-cementum interface and degrade the inserted collagen fibrils and the fibrils of cementum matrix.

Content of the pocket:

- 1)Microorganisms .
- 2) Bacterial products (enzymes and endotoxins).
- 3)GCF.
- 4)Remnants of food
- 5)Salivary mucin.
- 6)Desquamated epithelial cells.
- 7)Leukocytes
- 8)Plaque covered calculus usually projects from the tooth surface
- 9)Purulent exudates may be present

Periodontal pocket as healing lesions:

- Periodontal pockets are inflammatory lesions and constantly undergoing repair. Complete healing does not occur because of persistence of bacterial attack which continues to stimulate an inflammatory response causing degeneration of new tissues elements formed during the continuous effort at repair.
- The condition of the soft-tissue wall of the periodontal pocket results from the interplay of the destructive and constructive tissue changes. Their balance determines clinical features such as color, consistency, and surface texture of the pocket wall..

- **Edematous pocket walls**: when the inflammatory component predominates(inflammatory fluid and cellular exudate predominate) the lateral wall appears soft, edematous friable, with smooth shiny surface and bluish red discoloration.
- **Fibrotic pocket wall**: when reparative changes predominates,(If there is a relative predominance of newly formed connective tissue cells and fibers), the pocket wall is more firm and pink the gingiva appears fibrotic and pink.
- In some cases both lesions present in the same pocket as outer surface of a pocket wall fibrotic, the inner surface of soft tissue wall is inflamed and ulcerated.

Clinical features/histopathological feature:

A.1- bluish red discoloration of the gingival wall of pocket, this caused due to circulatory stagnation.

2- flaccidity of tissue: due to destruction of gingival fibers.

3- smooth shiny surface: due to atrophy of the epithelium and edema.,

4- pitting on pressure: due to edema and degeneration.

B.Gingival wall may be pink or firm when fibrotic changes predominates over exudation and regeneration.

C.Bleeding on probing: due to

Increased vascularity.

Thinning and degeneration of epithelium.

Proximity of engorged vessels to inner surface.

D.Probing is generally painful: due to ulceration of the inner aspect of the pocket wall.

E.Pus may be present: due to suppurative inflammation.

F.Other clinical features

Thickened marginal gingiva.

Loss of stippling.

Tooth mobility and diastema formation

Periodontal disease activity

1- Period of quiescence or inactivity this period characterized by reduced inflammatory response and little or no loss of bone and connective tissues.

A buildup of unattached plaque with its gram negative and anaerobic bacteria

2-period of exacerbation or activity bone and connective tissue attachment are lost and the pocket deepens

This period may last for days, weeks, months and eventually followed by period of remission and quiescence in which G+ve bacteria proliferate and more stable condition is established

Clinical feature shows bleeding spontaneous or on probing and greater amount of gingival exudates

Histological features , pocket appear thin and ulcerated ,infiltrate composed of plasma cells and PMNs leukocytes

Pocket probing:

We have two different pocket depths:

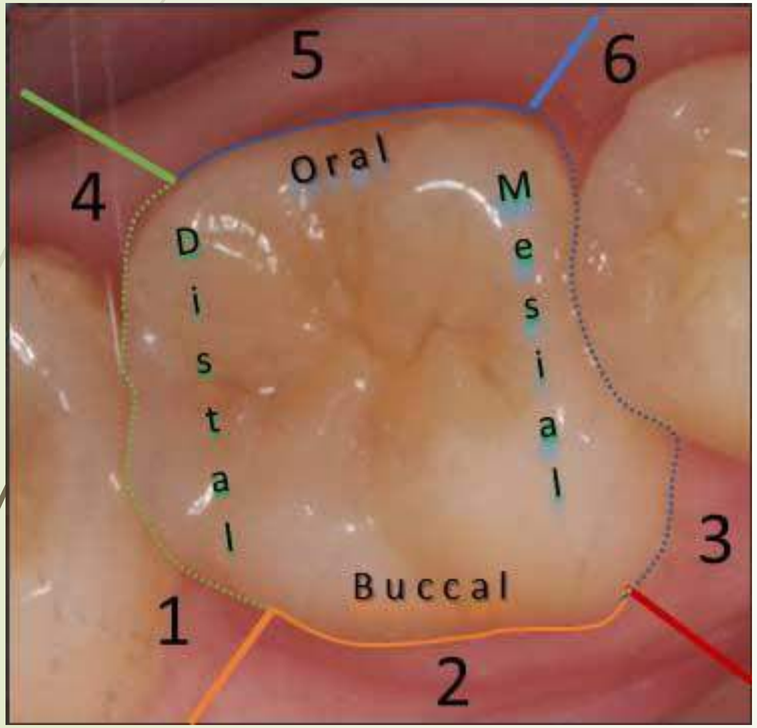
- **Biologic or histologic depth**: distance between gingival margin and base of the pocket. measured histologically(accurate measurement but not used routinely)
- **Clinical or probing depth**: distance to which a probe penetrates into the pocket.
- The standardized force used for penetration of probe is 25 pounds or 23 grams (0.75 N).

- **Probing Pocket depth PPD:** Distance between base of pocket and gingival margin.
- **Extent**
- The "extent" of disease refers to the proportion of the dentition affected by the disease in terms of percentage of sites. Sites are defined as the positions at which probing measurements are taken around each tooth and, generally, six probing sites around each tooth are recorded, as follows:
- Mesiobuccal
- Midbuccal
- distobuccal
- mesiolingual
- mid-lingual
- distolingual

- If up to 30% of sites in the mouth are affected, the manifestation is classified as "localized"; for more than 30%, the term "generalized" is used.

Probing techniques

- Occlusal view: six surfaces measured in periodontal probing
- In multirooted teeth, the possibility of furcation involvement should be carefully explored with specially designed probe (eg. Nabers probe).
- The probe should be inserted parallel to the vertical axis of the tooth and walked circumferentially around each tooth to detect the area of deepest penetration.
- To detect internal crater: the probe should be placed obliquely from both facial and lingual surfaces, so as to explore the deepest point of the pocket located beneath the contact point.


5
6
4
Oral
D i s t a l
M e s i a l
3
1
Buccal
2

<u>Level of attachment loss   CAL</u>

Distance between base of pocket and a fixed point on the tooth such as CEJ.

Severity: The "severity" of disease refers to the amount of periodontal ligament fibers that have been lost, termed "clinical attachment loss"

- *STAGE 1*: 1-2mm *4-5mm pocket*
- *STAGE 2*: 3-4mm *6-7mm*
- *STAGE 3*: >5mm *>8mm*
- *STAGE 4*: >5mm *>8mm*

Treatment:

1-Non surgical treatment:

- Oral hygiene instruction.
- Scaling and root planning
- Use curettes for subgingival scaling, root planing and removal of the soft tissue lining the pocket.
- Root planing strock should be moderate to light pull strock for final smoothing and planing of root surface.
- Continuous series of long, overlapping shaving strock is achieved.
- To avoid over instrumentation, a delicate transition from short, powerful scaling strocks to longer, lighter root planning strokes must be made as soon as calculus and initial roughness have been eliminated.
- Periodontal medication as application of tetracyclines

2-Surgical treatment

Pocket depth reduction through different surgical procedures

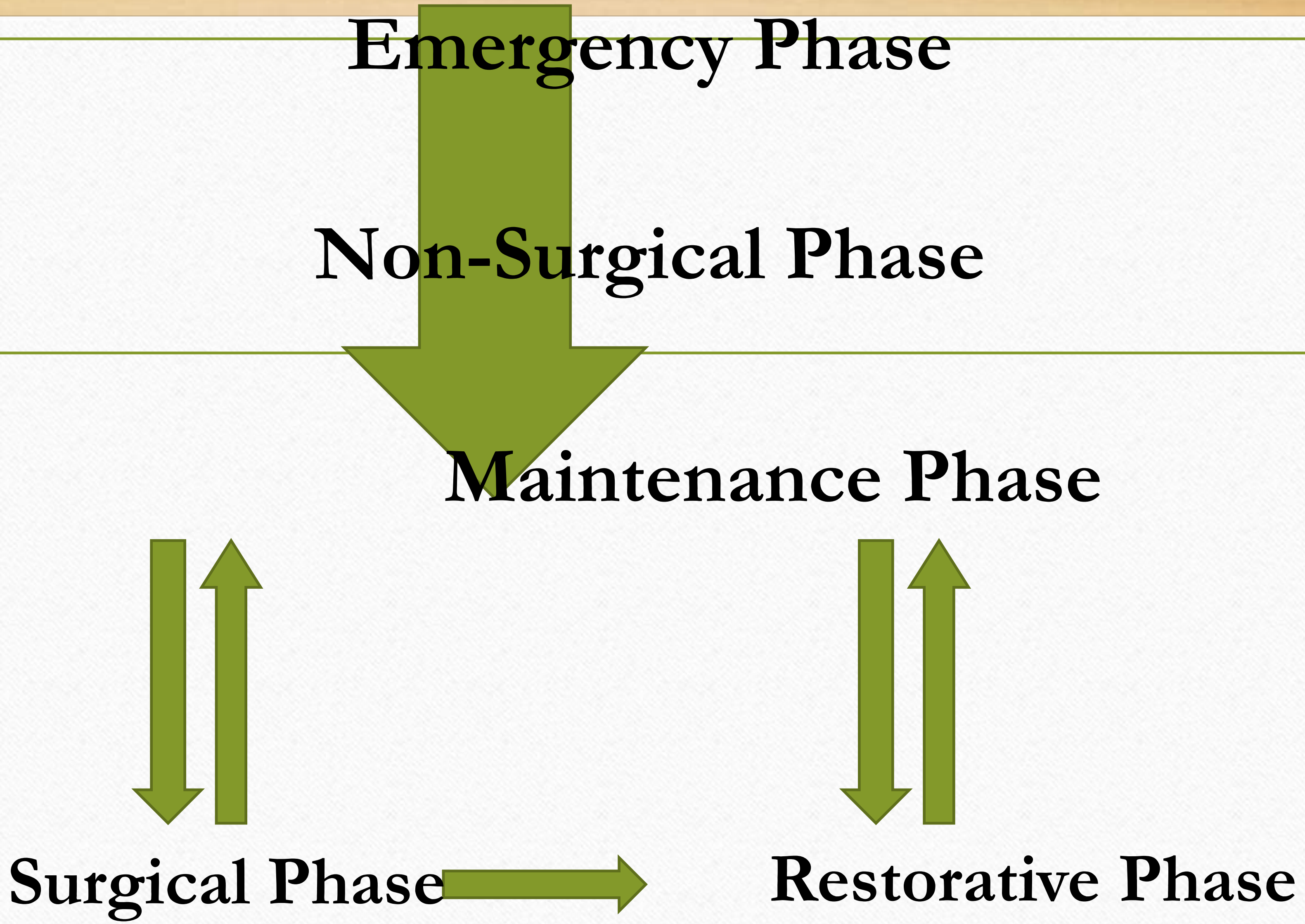
- 1-gingival curettage
- 2-gingivectomy
- 3-periodontal flap procedures
- 4-osseous surgery
- 5-Periodontal regeneration procedures

Thank you

Phase II Surgical Therapy

م. سها أسود دهش العزاوي

B.D.S, MSc. Periodontology


Emergency Phase
Non-Surgical Phase
Maintenance Phase
Surgical Phase
Restorative Phase

Reevaluation after phase 1 therapy

All patients should be treated initially with scaling and root planing and the final decision on the need for periodontal surgery should be made only after evaluation of the effects of phase 1 therapy no less than 1 to 3 months after completion of phase one therapy.

- Patients with adequate oral hygiene and no remaining pockets: put in maintenance phase.
- Patients with inadequate oral hygiene: repeate phase 1 focusing on OHI.
- Patients with adequate oral hygiene but remaining pockets of ≥5mm: periodontal surgery for pocket elimination.

In this phase the surgical techniques used for the following purposes :

- 1-Controlling or eliminating periodontal disease(surgical pocket therapy)
- 2-Correcting anatomic conditions that favor periodontal disease, impair aesthetics or impede placement of prosthetic appliances(plastic surgery, aesthetic surgery, pre prosthetic techniques).
- 3-Placing implants to replace lost teeth and improving environment for their placement and function.

Periodontal surgery

1. **Pocket reduction surgery:**

- Resective (gingivectomy , apically displaced flap and un-displaced flap with or without osseous resection .
- Regenerative (flaps with grafts , membranes, etc.

2. **Correction of anatomic/morphologic defects:**

- Esthetic surgery :e.g:covering deuded roots and augmentation of attached gingiva.
- Preprosthetic surgery (crown lengthening, ridge augmentation and vestibular deepening)

3. **Placement of dental implants :**

Periodontal surgery

- Successful cause-related therapy (by the removal of plaque and calculus) will reduce gingival inflammation (edema, hyperemia and flabby tissue) there by making assessment of true gingival contour and pocket depth possible. In addition the soft tissue will be more fibrous and thus firmer, which facilitate surgical handling of the soft tissues. The propensity for bleeding is reduced, making the inspection of the surgical field easier.

Periodontal surgery

- The effectiveness of the patient's home care which is of decisive importance for the long term prognosis must be properly evaluated; lack of effective self performed plaque control will often mean that the patient should be excluded from surgical treatment.

Objectives of periodontal surgery

- 1-Accessibility and direct vision for proper S+ RP.
- 2-Reduction or elimination of plaque retentive area especially periodontal pockets that have not responded to initial therapy.
- 3-Eliminate inflamed periodontal tissue.
- 4-Create a physiologic morphology of the dentogingival area that will facilitate efficient self performed plaque control.

- 5-Provide access to correct bony defects.
- 6-Enhancing the regeneration of periodontal tissue.
- 7-Correct mucogingival defect and improve periodontal aesthetic.

Surgical treatment include

- 1-Gingivectomy.
- 2-Flap surgery.
- 3-Distal wedge procedure.
- 4-Mucogingival surgery for correction of mucogingival and aesthetic defect.
- 5-Crown lengthening to increase clinical crown length
- 6-Guided tissue regeneration (GTR) to regenerate periodontal supporting structures.

Gingivectomy

- This surgical procedure aimed at the excision of the soft tissue wall of a pathologic periodontal pocket and this pocket elimination was usually combined with recontouring of the diseased gingiva to restore physiologic form(e.g. Drugs induced gingival enlargement and the resulting false pocket can be removed by this method).

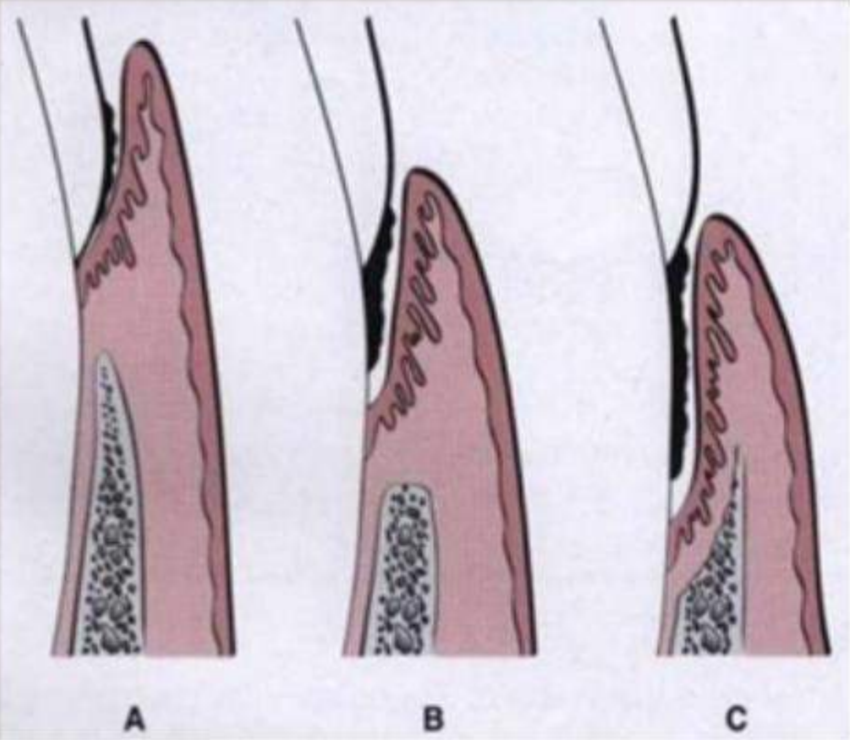
Indication

- 1-Gingival enlargement or over growth.
- 2-Idiopathic gingival fibromatosis.
- 3-Shallow suprabony pocket.
- 4-Minor corrective procedure.

1. According to the relation to the crestal bone

Suprabony
supracrestal/
supraalveolar

Intrabony
subcrestal/
intraalveolar



Contraindication

- 1-Infrabony pocket.
- 2-Thickening of marginal alveolar bone and the need for bone surgery.
- 3-Attached gingiva is narrow or absent.

Advantages

- 1-Technically simple, good visual access.
- 2-Complete pocket elimination.
- 3-Restoration of a physiologic gingival contour.

Disadvantages

- 1-Gross wound, post operative pain.
- 2-Healing by secondary intention.
- 3-Danger of exposing bone.
- 4-Loss of attached gingiva.
- 5-Phonetics and aesthetic problem in the anterior area with sensitivity due to exposure of the cervical area of tooth.

1. SURGICAL GINGIVECTOMY

Instruments Required In Surgical Gingivectomy

- Krane Keplan Pocket Marker
- Kirkland Periodontal knife
- Orban periodontal knife
- Bard – parker handle
- Bard – Parker blades no 11 & 12
- Supra & subgingival scalers
- Curettes

Pocket marking forceps

- Paired (L & R)
- **Use:** indicate the location of the base of

Gingivectomy knives

- GV knife (Kirkland, L & R)
- Papilla knife (Orban, L & R)
- Universal knife

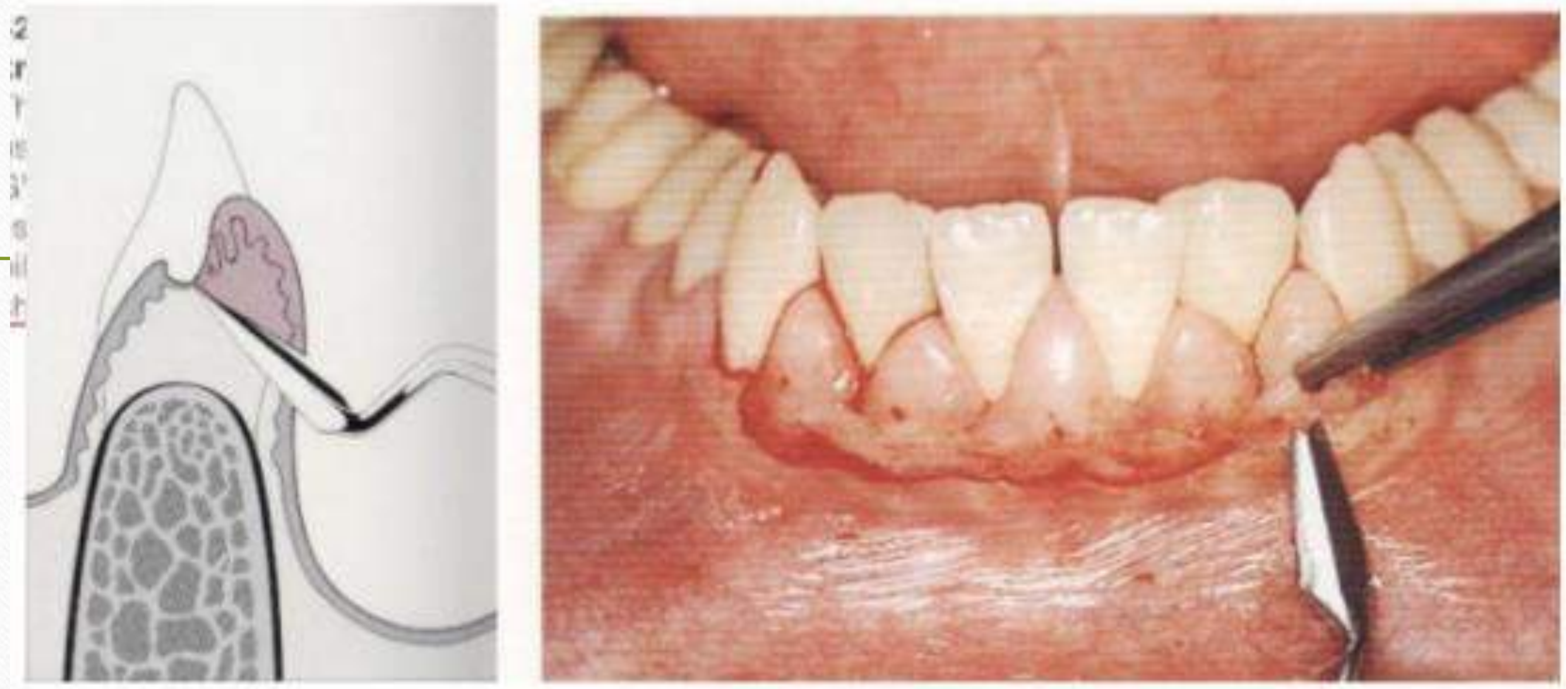
Gingivectomy procedure

- Giving local anesthesia.
- Marking the pocket depth: the straight arm of pocket depth marker forceps is guided into buccal pocket, when the base of pocket is encountered,the forceps is pinched together causing the horizontal forceps tip to mark depth of pocket, by repeating this procedure at each tooth surface ,a series of bleeding points is created, which are used subsequently as a guide for incision.

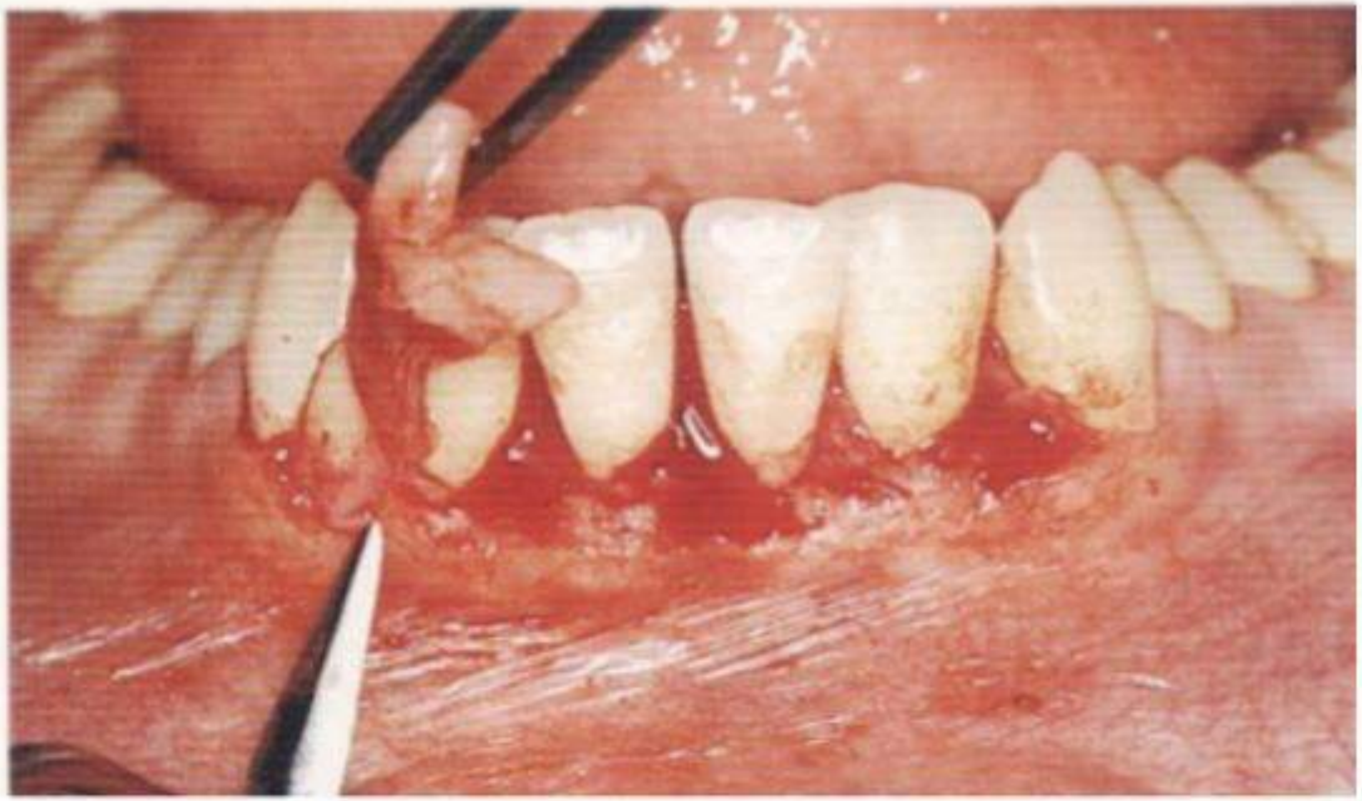
Marking the base of the pocket

Primary beveled incision which carried out slightly apical to bleeding points by Kirkland knife.

Continuous incision or interrupted , straight or scalloped is made.



Secondary incision to separate the interproximal soft tissues from the interdental periodontium by Orban knife.



Careful removal of the incised tissues by a currete or a cumine.

By curette remove plaque, calculus and granulation tissues then smoothing teeth surfaces.

Gingivectomy wound after scaling

Use Kirklaned knife for gingivoplasty(minor alterations in gingival morphology without tissue excision)by shaving wound margin to create thin margin.

Control bleeding by placing gauze packs Put dressing to cover the wound with pressure to prevent the bleeding with consequence formation of granulation tissue under dressing and without interference with occlusion or mobile mucosa

Flap surgery

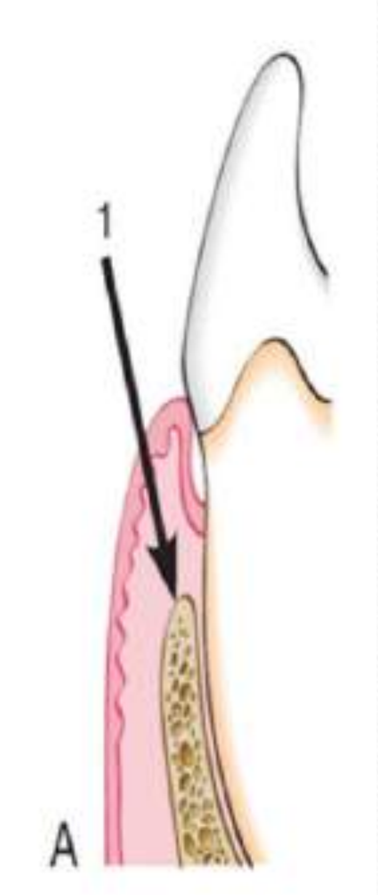
- **Indications**
- 1-In treatment of infrabony pockets.
- 2-When the gingivectomy will lead to an unacceptable aesthetic results.
- 3-Osseous recontouring (elimination of bony defect).

Modified widman flap

- Reported in 1974 by Ramfjod and Nissle,it is a replaced flap. There are three incisions in this flap, it is usually conducted as following:

A- Primary incision: First incision-scalloping

- The scalloped incision is performed on both labial and palatal aspects,using the double-edge 12B scalpel. It is an inverse bevel incision extending to the alveolar crest.This incision thins the gingival tissue and permits complete closure of the interdental osseous defects postoperatively. The distance of the incision from the gingival margin may vary from 0.5 to 2mm.



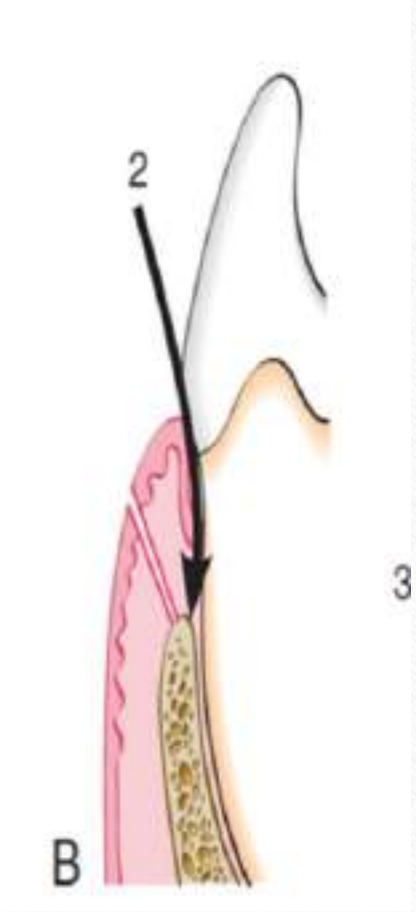

#10
#11
#12
#15

B- Flap retraction:

- An elevator is used to raise a full thickness mucoperiosteal flap as atraumatically as possible.The flap is reflected only to permit direct visualization of the root surface and the alveolar crest.In most cases it is possible to stay within the boundaries of the attached gingiva,without extending beyond the mucogingival line.

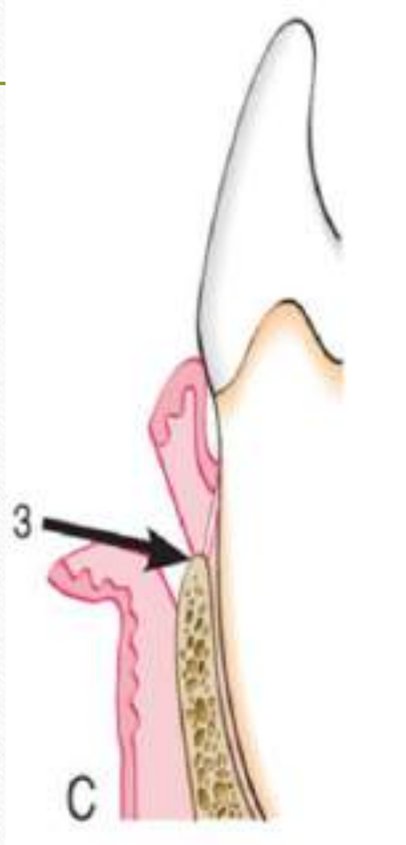
C-Secondary incision: Second incision-crevicular

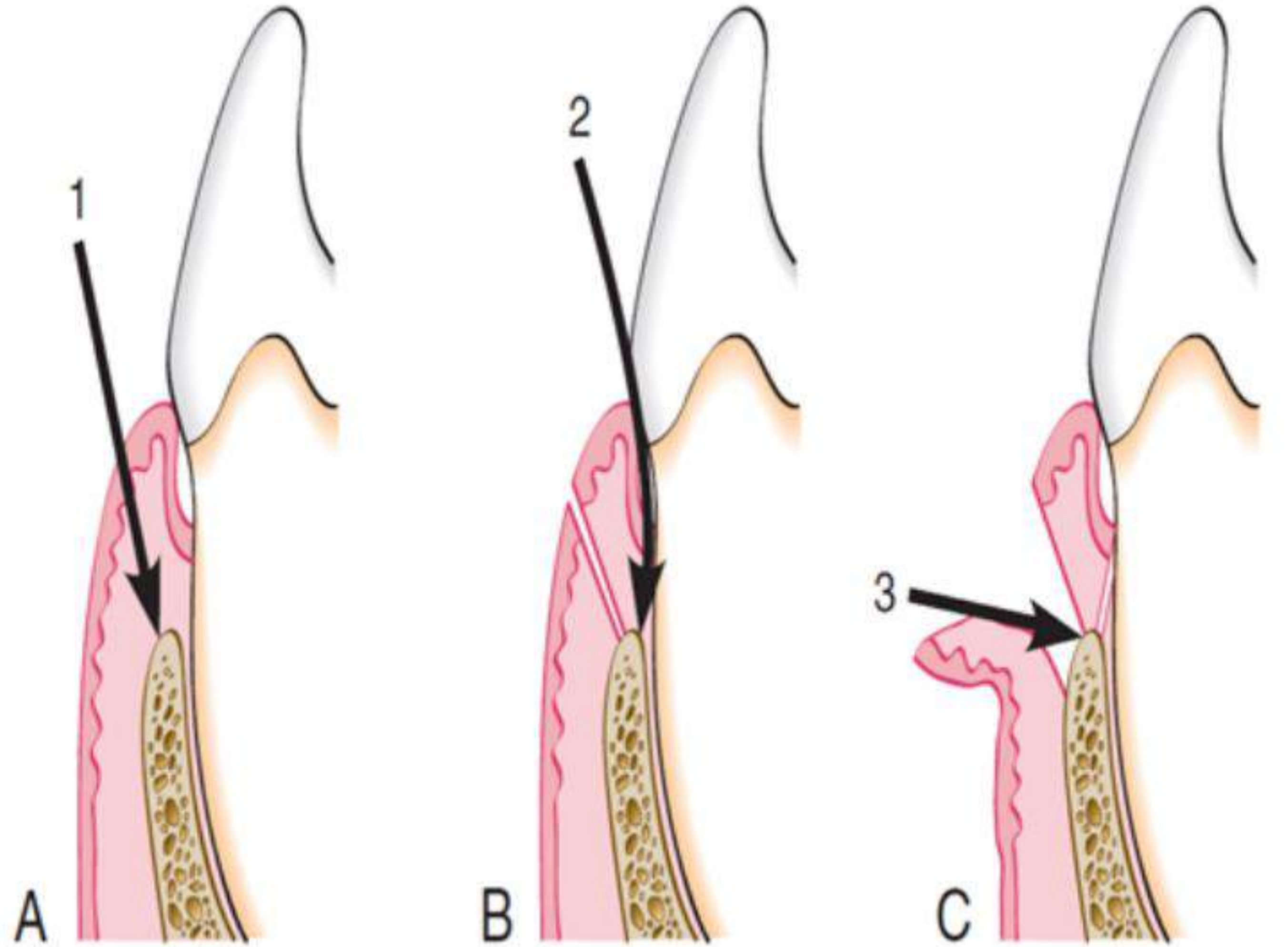
- This incision is carried around each tooth, between the hard tooth structure and the diseased pocket epithelium, to the depth of the junctional epithelium.The 12B scalpel is used.



d:Third incision:Third incision-horizontal

- The horizontal incision is carried along the alveolar crest thus separating the infiltrated tissue from healthy supporting connective tissue,specially in the interdental area.The incision also permits atraumatic removal of the diseased tissue.





Three incisions of Modified widman flap

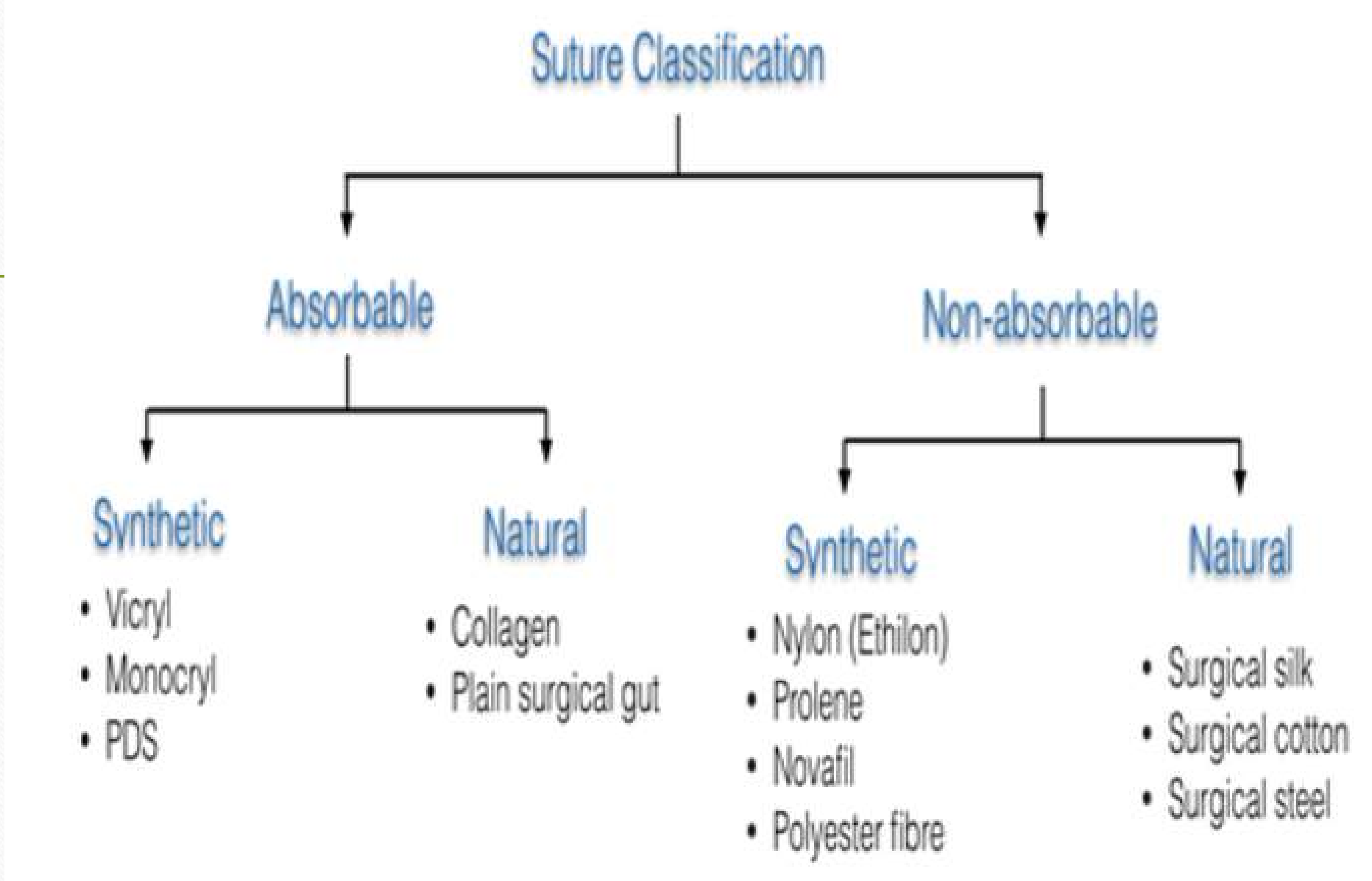
e:Direct root planing:Root planing with direct vision

- Fine curettes are used to remove remnants of pocket epithelium and granulation tissue, calculus necrotic cementum to obtain smooth, hard, clean surface.
- Root planing is performed with repeated rinsing.
- Root planing is the most important part of both the modified Widman procedure and all other periodontal surgical procedures.

f: Suturing:Complete coverage of interdental defects

- The labial and palatal flaps are closed over the interdental areas without tension, using interrupted sutures.The flaps should be adapted to the underlying bone and the necks of the teeth. New papillae where created by the scalloped form of the initial incision.These make it possible to cover interdental defects (e.g.bony defects)even when the interdental space is wide. For this reason, placement of a periodontal dressing is not absolutely necessary.


Preoperative clinical view
Internal bevel incision is placed (Facial view)
The flap is elevated
Wedge of marginal tissue not yet removed
After thorough debridement


Suture Classification
Absorbable
Non-absorbable
Synthetic
• Vicryl
• Monocryl
• PDS
Natural
• Collagen
• Plain surgical gut
Synthetic
• Nylon (Ethilon)
• Prolene
• Novafil
• Polyester fibre
Natural
• Surgical silk
• Surgical cotton
• Surgical steel

The Modified Widman flap Advantages

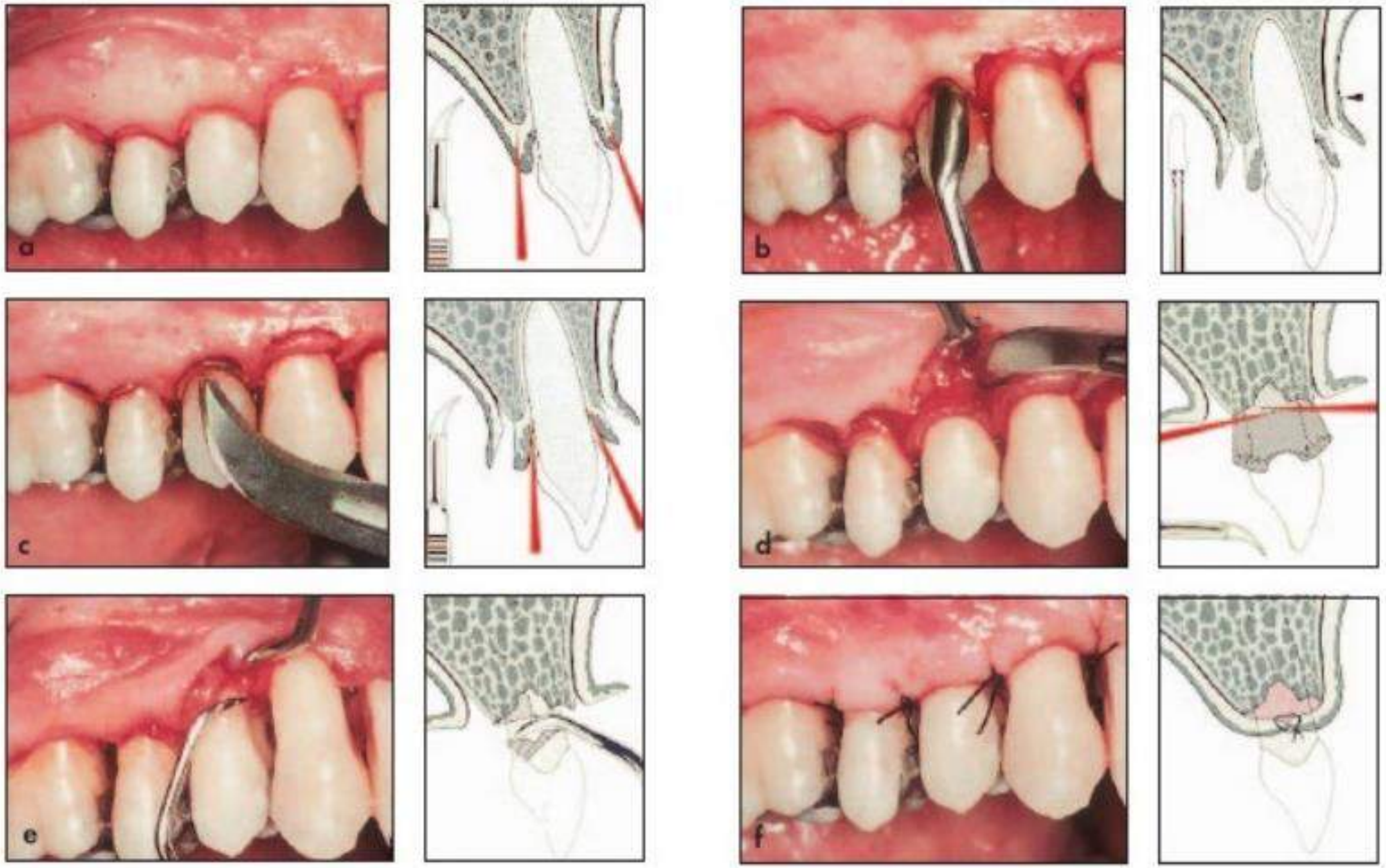
- 1-Good access to root surface to facilitate S+ RP as well as the removal of the pocket epithelium and the inflamed connective tissue.
- 2-Width of keratinized gingiva is maintained.
- 3-Replacement of the flap at presurgical location leads to less exposure of the root surfaces thus minimizes problem of aesthetic (especially anteriorly) and root hypersensitivity.
- 4-Cause minimal amount of trauma to the periodontal tissues and discomfort to the patient.

- 5-the possibility of obtaining a close adaptation of the soft tissues to the root surfaces.
- 6-provides better access to re-establish proper contour of the alveolar bone as well as the potential for bone regeneration in sites with angular bony defect.
- 7-furcation areas can be exposed.

Healing after flap surgery

- Following flap procedures and the removal of plaque, calculus and chronically inflamed granulation tissue, healing occurs by the formation of a Long junctional epithelium, this lead to reduced probing depth but that epithelium is more susceptible to plaque induced breakdown than the original connective tissue attachment and consequently post operative plaque control must be a very high standard, a new connective tissue attachment may form following flap procedures, although this cannot be predicted with certainty.

- Transient root hypersensitivity and recession of the gingival margins frequently accompany the healing process following close and open S+ RP, thus the patient should be awarned that these results may happen.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d
e
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Thank you

بسم الله الرحمن الرحيم

اعداد:

د. نور صباح رحيم

Systemic risk factors

Systemic risk factors can be divided into :-

1-modifiable

-specific microbiota.

-smoking.

-diabetic mellitus.

-stress.

-obesity.

-oral hygiene.

-immunodeficiency.

-drugs (certain medication).

-diet.

-osteoporosis.

-other systemic diseases.

2-non-modifiable

-race.

-age.

-genetic.

-hormonal influence (such as some hormones related to pregnancy).

1-Dental plaque and oral hygiene (microorganisms)

-the primary etiological factor in the development and initiation of periodontal diseases is dental plaque.

-dental plaque is a bacterial aggregation on the teeth or other solid structure in the oral cavity.

-dental calculus which mineralized dental plaque is consider as secondary etiological factor of periodontal disease and it is covered with dental plaque so it serve as reservoir for bacterial plaque (retentive area).

-oral hygiene can favorably influence the ecology of the microbial flora in shallow to moderate pocket, but it does not affect host response. Oral hiygene alone has a little effect on sub-gingival micro flora in deep pocket and personal oral hiygene practices among health professional have been shown to be unrelated to periodontitis in these individuals.

Specific microorganisms:-

-although there is sufficient evidence that accumulation and maturation of plaque biofilm is necessary for the initiation and progression of periodontal diseases, studies show that bacterial species colonizing the gingival pocket play variable roles in the pathogenesis of these diseases and may therefore posses different level of risk of periodontal tissues loss . The subgingival microflora in periodontitis can harbor bacterial species but only small number has been associated with the progression of diseases such as

-Aggregatibacter actiomycetemcomitance (A.a)

-P. gingivalis.

-P.intermedia.

-T.forsythia

Theses m.o. produce endotoxine that will cause sever periodontal destruction.

Tobacco Smoking:

-Tobacco smoking is a major risk factor for increasing the prevalence and severity of periodontal destruction . It was found that the increased risk for periodontitis in smoker was 2.5-7 times greater than non smoker.

-The smoker appear have less gingival inflammation and less bleeding in the gingiva may be explained by deceased gingival vascularity, which includes decreased vascular density, reduce lumen area of gingival vessels (increased vasoconstriction).

-Studies suggested that nicotine increase rate of proliferation of gingival epithelium which can contribute to the reduction of inflammatory clinical signs in the gingival tissues. These are physiological effect of smoking on the etiology of periodontal disease , with decreased gingival crevicular fluid flow.

-Smoking suggest an imbalance between bacterial challenge and host response which may due to changes in the composition of subgingival plaque with increase in the number and or virulence of pathogenic organisms.

The microbiological effect of smoking:

- 1- it is increase the colonization of shallow periodontal pockets by periodontal pathogens and increase levels of periodontal pathogens in deep periodontal pocket.
- 2-smokers may have higher level of Tannerella forsythia, P.gingivalis and T. denticola.
- 3- smoke derived aryl hydrocarbons and bacterial LPS may act additively to inhibit bone formation , may explain why periodontal bone loss is greater and bone healing is less successful in smokers than non smokers with periodontal infections.

The immunological effect of smoking: •

- 1-Nicotine causes decrease immune response and impair PMNs chemotaxis and phagocytosis.
- 2-Increase the production of TNF-alpha , IL6 , these immune mediators are known to lead to more sever destructive inflammation in the periodontal tissue.
- 3-Reduction in the serum concentration of Immunoglobulin as IgGs which is essential in the production against periodontal infection. Also smoking decrease the level of salivary IgA antibodies.
- ----The risk of periodontal disease increase with number of cigarettes smoked per day. smoking of cigars and pipes carries the same risk as smoking cigarettes. Exposure to secondhand smoke may be associated with an increased risk for developing periodontal disease.

Diabetes mellitus:

-Diabetes is a modifiable factor in the sense that though it cannot be cured, it can be controlled.

-studies have been done which suggest that poorly controlled diabetes respond less successfully to periodontal therapy relative to well-controlled and non-diabetic.

-it is a complex metabolic disease characterized by chronic hyperglycemia.

-uncontrolled diabetic is associated with a reduction in the defense mechanisms (neutrophil dysfunction, impairment of chemotaxis and phagocytosis), atherosclerosis and reduce normal gingival flow, increased susceptibility to infection include periodontitis and poor wound healing..

-Diabetic mellitus does not cause gingivitis or periodontitis , but it alters the response of periodontal tissues to local factor.

-Diabetic patients with poor oral hygiene may have very sever gingival inflammation , deep periodontal pockets, rapid bone loss, and frequent periodontal abscess.

Neutropenia:•

-The diagnosis of Neutropenia is based on clinical signs and • symptoms as well as absolute neutrophil count a relatively deficiency in neutrophil number can dramatically increase susceptibility to infectious disease.

-Neutropenia is considered clinically significant when the neutrophil • count falls below 1,000 cell/ml (normal adult range: 1,800-8,000 cell/ml).

-Disorders of neutrophil function also increase the host susceptibility • to infection, by decrease chemotaxis , migration, phagocytosis and killing.

Osteoporosis:

- -many of the studies conducted to data suggest there is a relationship between skeletal osteoporosis and bone loss to the extent that postmenopausal osteoporosis may result in dental osteopenia (reduction in bone mass due to imbalance between bone formation and resorption) involving the jaw , and particularly the mandible. Osteoporosis was significant associated with sever alveolar bone loss and the prevalence of periodontitis causes in post menopause women and in men with advancing age.

Drugs (medications):

-Gingival enlargement is a well –known consequence of the administration of some drugs as anticonvulsants (Phenytoin or Delantin), immunosuppressant (Cyclosprine) and Ca channel blockers (Antihypertensive drugs) such as

-Amelodipine.

-Dilitazam .

-Nicardipine.

-Nifidipine.

Nisoldipine.   -

-Verapamil   -

Obesity:

-Obesity is one of the most significant health risks of modern society . A condition associated with obesity (the metabolic syndrome) , this a clustering of dyslipidemia and insulin resistance may exacerbate periodontitis. Obesity has been postulated to reduce blood flow to the periodontal tissues, promoting the development of periodontal diseases. Furthermore, obesity may enhance immunological or inflammatory disorders , which might be the reason obese subjects tend to exhibit escalating poor periodontal status relative to non-obese individuals.

Psychological factors:

-Studies have been demonstrated that individuals under psychological stress are more likely to develop clinical attachment loss and loss of alveolar bone.

-One possible link in this regard may be increased glucocorticoid secretion that can depress immune function , increase insulin resistance , increase in production of IL-6 in response to increased psychological stress and potentially increased risk of periodontitis. Anther study suggest that host response to *P. gingivalis* infection may be compromised in psychological stress individuals also the relationship is simply due to the fact that individuals under stress are less likely to perform regular good oral hygiene and prophylaxis.

Nutrition:

-The potential mediating role of nutrition in the oral health-systemic diseases relationship has increased interest in the effect of nutrition in oral health and periodontal disease. However, efforts to correlate the nutritional deficiency to periodontal disease have yielded conflicting results. Vitamins are coenzyme required for metabolism and health.

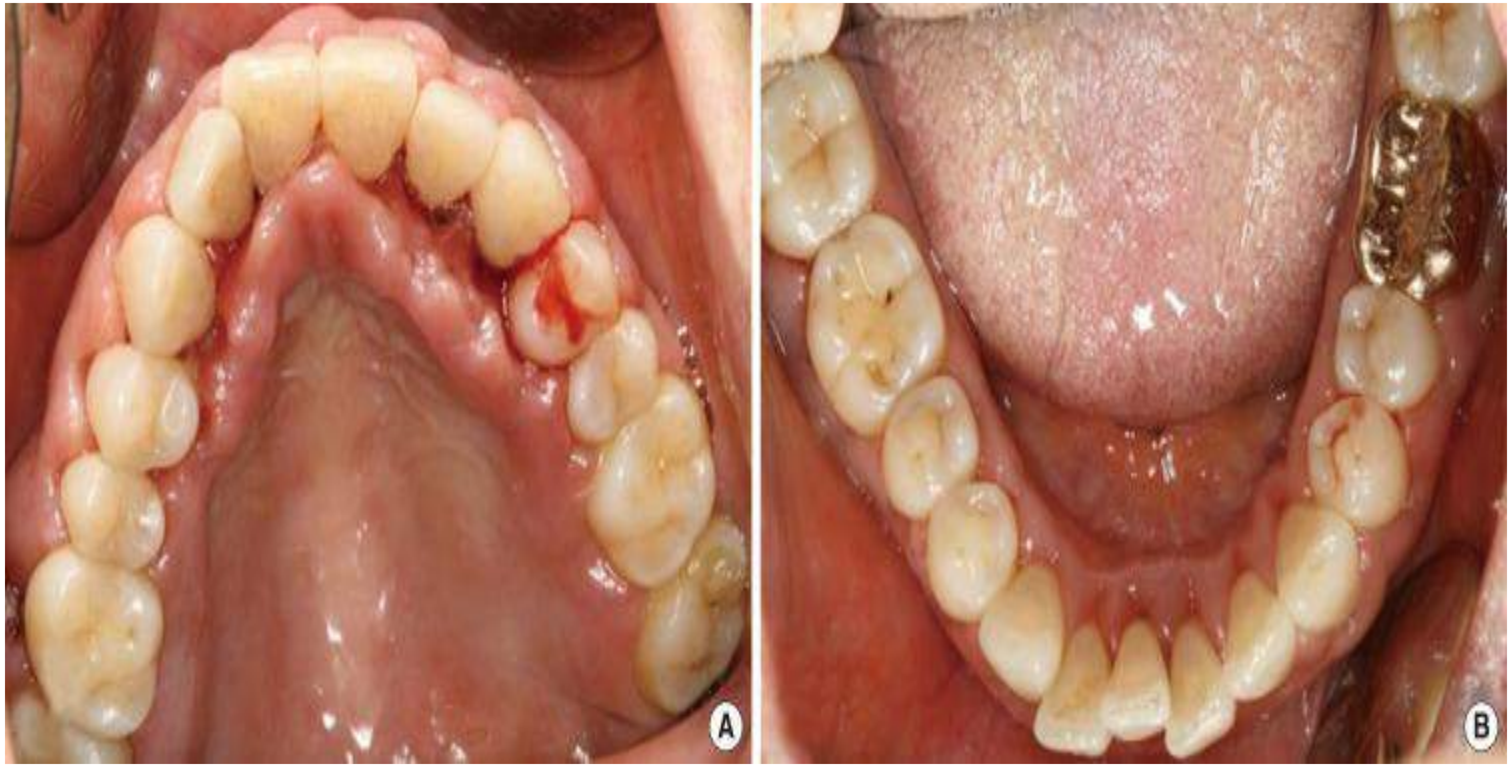
HIV/AIDS

It has been stated that the immune dysfunction (immunosuppression) associated with human immunodeficiency virus (HIV) infection and acquired immunodeficiency syndrome (AIDS) increases susceptibility to periodontal disease. Those patients often had severe periodontal destruction characterized by necrotizing ulcerative periodontitis.

Hematological factors

- Hemorrhagic gingival overgrowth with or without necrosis is a common early manifestation of acute leukemia. Patients with chronic leukemia may experience similar but less severe periodontal changes. Chemotherapy or therapy associated with bone marrow transplantation may also adversely affect the gingival health.


A
B
C


A
B

Non –modifiable systemic risk factors: •

-Genetic •

-Female Hormonal Alteration •

-Race •

-Gender •

Genetic factors

There is evidence that genetic differences between individuals may explain why some patients develop periodontal disease and other do not. Genetic factor may play an important role in determining the nature of the host response and may effect the function of phagocytic immune cells or the structure of the epithelium or connective tissue.

-One of the disease is papillon- Lefevre syndrome which is a rare hereditary disease characterized by hyperkeratotic skin lesion in the palms, soles, knee and elbow and severe destruction to the periodontium with early loss of primary and permanent teeth.

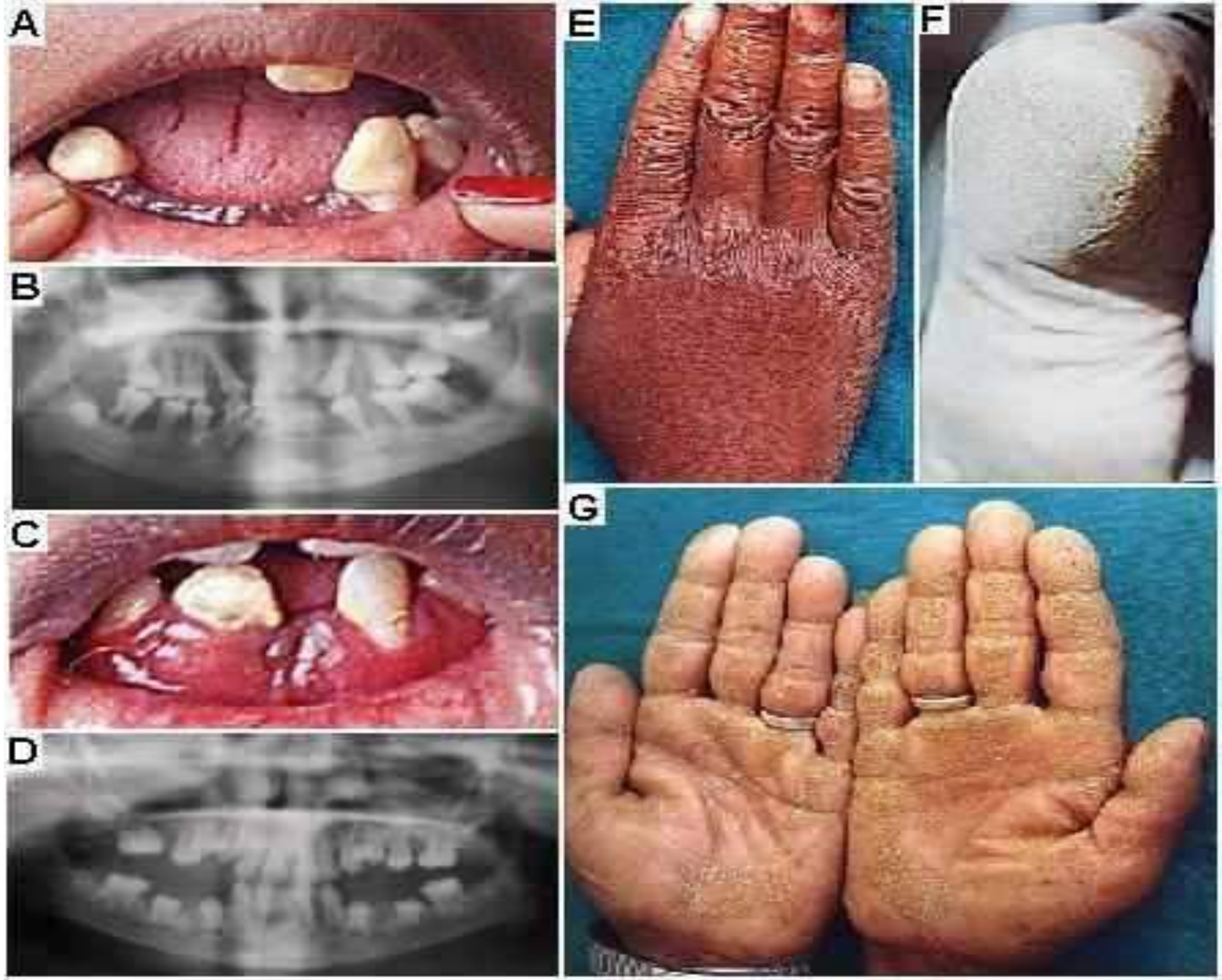
-Other disease is aggressive periodontitis which has familial aggregation (which may be seen in one family) . Some immunological defects are associated with aggressive periodontitis. The periodontal disease has a link with some genetic factor by:

1-A specific interleukin (IL-1) genotype has been associated with severe chronic periodontitis.

2-Neutrophil abnormalities are under genetic control.

3-Genetic play a role in regulating the titer of protective IgG2 antibody response to A.a in patients with aggressive periodontitis.


الطب | سلسلة المتلازمات
12 – متلازمة فرط التقرن الراحي الأخمصي
Papillon Lefevre syndrome


A
B
C
D
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G

Pregnancy, puberty, menopause(hormonal) (Female hormonal alteration):

- -Pregnancy associated gingivitis is inflammation of the gingival tissues associated with pregnancy. This condition is accompanied by increase in steroid hormones in GCF and increase in the levels of (P. intermedia) bacteria which use steroid as a growth factors. The increase in sex hormones may exaggerate the inflammatory response to dental plaque which means small amount of plaque may lead to gingivitis.
- -Puberty is also accompanied by an exaggerated response of the gingiva to local irritation.
- -During menopause, estrogen deficiency will reduce bone mineral density.


GINGIVA IN PREGNANCY


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Gender •

-Numerous studies reported higher periodontal destruction among males • compared to the female population. The reason for these sex differences are not clear, but they are though to be related to ignorance of oral hiygene, which is usually observed among males.

Race: •

-the level of attachment loss is also influenced by race/ethnicity, although the • exact role of this factor is not fully understood . Certain racial/ethnic groups, particularly subjects of African and Latin American background , have a higher risk of developing periodontal tissue loss than other group.


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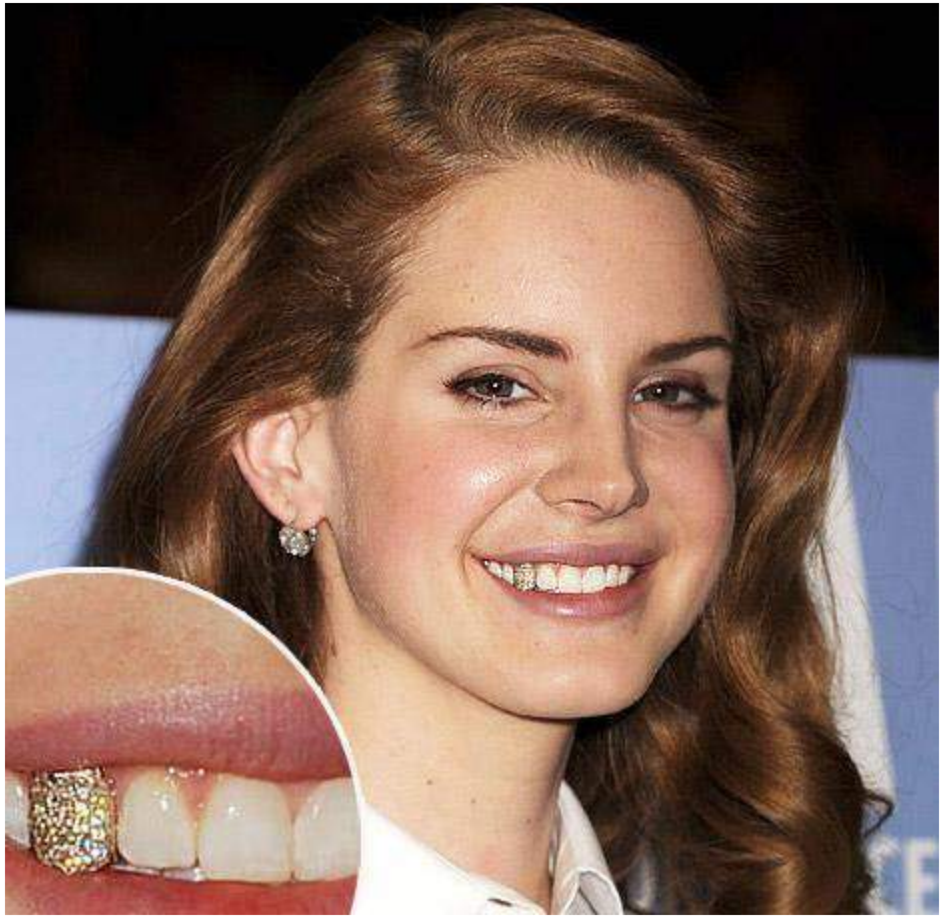


THANK YOU.

TREATMENT PLANNING OF PERIODONTAL DISEASES

PRESENTED BY:
DR: NOOR SABAH IRHAYYIM

Aim of the treatment planning:

-A treatment plan is a therapy plan formulated only after a thorough examination has been completed, diagnosis and prognosis have been determined and the needs and desires of the patient have been taken into consideration. It must be recognized that as diagnosis and prognosis will change with treatment, therapeutic needs may also change. As such, the treatment plan must be changed accordingly.

The treatment plan for patients with periodontal disease includes the following phases

1.Phase I therapy (initial, cause-related therapy, non-surgical therapy •

2.Phase II therapy (surgical therapy) •

3.Phase III therapy (restorative therapy) •

4. •

Phase IV therapy (maintenance therapy)

The aim of the treatment plan is total treatment that is, coordination of all the • immediate, intermediate, and long-term goals to create a well-functioning dentition in a healthy periodontal environment.

The Immediate goals *are the elimination of all infections and inflammatory* • *processes that cause periodontal and other oral problems that may hinder the patient's general health.*

The intermediate goals *are the reconstruction of a healthy dentition that not only* • *fulfils all functional and aesthetic requirements but lasts many years.*

The long-term goal *is the maintenance of health through prevention and* • *professional supportive therapy. The long-term goal is set, and both the patient and the clinician work toward it from the very first visit.*

The treatment plan involves decisions regarding the following:

1-Emergency treatment (pain, acute infections)

2-Removal of nonfunctional and diseased teeth, and possibly strategic extraction of healthy teeth to facilitate the prosthetic reconstruction of the patient

3-Treatment of periodontal diseases (surgical or nonsurgical, regenerative or resective)

4-Endodontic therapy (necessary and intentional)

5-Caries removal and placement of temporary and final restorations

6-Occlusal adjustment and orthodontic therapy

7-Replacement of missing teeth with removable or fixed dental prostheses or dental implants

8-Aesthetic demands

9-Sequence of therapy

- •

Extracting or Preserving a Tooth

Removal, retention, or temporary(interim)retention of one or more teeth is an important part of the overall treatment plan. A tooth should be extracted under the following conditions:

- **It is so mobile that function becomes painful.**
- **It can cause acute abscesses during therapy.**
- **There is no use for it in the overall treatment plan.**

In some cases, a tooth can be retained temporarily, postponing the decision to extract until after treatment is completed. A tooth in this category can be retained under the following conditions:

- **It maintains posterior stops; the tooth can be removed after treatment when it can be replaced by an implant or another type of prosthesis.**
- **It maintains posterior stops and may be functional after implant placement in adjacent areas. When the implant is restored, these teeth can be extracted.**
- **In the anterior aesthetic zone, a tooth can be retained during periodontal therapy and removed when treatment is completed and a permanent restorative procedure can be performed. The retention of this tooth should not jeopardize**

Sequence for the treatment of periodontitis stages I, II and III •

1. The first step in therapy (behavioral and risk factor modification) is • aimed at guiding behavior change by motivating the patient to undertake successful removal of supragingival dental biofilm and risk factor control and may include the following interventions:

• Supragingival dental biofilm controh •

• Interventions to improve the effectiveness of oral hygiene [motivation, • instructions (oral hygiene instructions).

• Professional mechanical plaque removal, which includes professional • interventions aimed at removing supragingival plaque and calculus, as well as possible plaque-retentive factors that impair oral hygiene practices.

• Risk factor control, which includes all the health behavioral change • interventions eliminating/mitigating the recognized risk factors for periodontitis onset and progression (smoking cessation, improved metabolic control of diabetes).

- This first step of therapy should be implemented in all periodontitis patients, irrespective of the stage of their disease, and should be re-evaluated frequently
- The second step of therapy (cause-related therapy) is aimed at controlling (reducing/eliminating) the subgingival biofilm and calculus
- 3. The third step of therapy (surgical phase) is aimed at treating those areas of the dentition non-responding adequately to the second step of therapy (presence of pockets ≥4 mm with bleeding on probing or presence of deep periodontal pockets [≥6 mm])

4.Supportive periodontal care (maintenance phase) is aimed at maintaining periodontal stability in all treated periodontitis patients combining preventive and therapeutic interventions defined in the first and second steps of therapy

Objectives of initial phase (cause-related therapy):

- 1-motivation the patients to combat dental disease (patient information)
- 2-giving the patients instruction on the proper oral hygiene techniques (self-performed plaque control methods)
- 3-scaling and root planning.
- 4-antimicrobial therapy (local or systemic)
- 5-control or elimination of additional retention factors for plaque such as : correction of restoration and prosthetic irrational factors and excavation of caries and restoration.
- 6-occlual therapy
- 7-orthodontic treatment

Motivation:

- Detailed information must be given to the patients regarding his/her periodontal diseases , its etiological factors symptoms , consequence, prognosis and the relationship between the presence of dental plaque and calculus in the mouth and the location of sits showing dental diseases by using plaque disclosing agents.

The information are aimed at motivating the patients to cooperate in the treatment hence without compliance (which has been described by dental professional). A good treatment outcome will not be achieved.

Mechanical plaque control demands active participation of the individuals subject and the establishment of proper oral homecare habits is a process that depends on the behavioral changes, thus the patient positive attitude to treatment may have a positive long-term effect on his/her tooth cleaning efforts. In addition, dental professional should try to emphasize on the role of the patients personal oral hygiene procedures in the prevention of dental diseases and they should encourages the patients to take responsibility for his/her own oral health.

Disclosing agents:

- Is a chemical compound (tablet or solution) that stains dental plaque such as erythrosine, fuschcin or fluorescein ,(dental plaque is white and it cannot easily be identified so when it is thick enough and/or the observer is not well trained so can use disclosing agent

These agents should be used to demonstrate the presence and location of plaque in addition to the evaluation of the efficacy of the patients homecare technique thus they should be applied after tooth brushing and interdental cleaning.

Brushing:

- The most widespread mean of actively removing plaque at home is tooth brushing. The efficacy of brushing with regard to plaque removal is dependent on:
- 1-the design of brush
- 2-the skill of individuals using of bush(manual dexterity)
- 3-frequency and duration of brushing
- 4-the morphology of the dentition(crowding, spacing, gingival phenotype, longer teeth, exposed dentin, …)

Oral hygiene instruction component:

- 1-self assessment
- 2-self examination
- 3-self monitoring
- 4-self instruction
- For this purpose disclosing agent is applied before brushing, and the aid of mirror, the patients can identify the amount of plaque formed after last brushing and give information about his/her cleaning performance.

Methods of tooth brushing

1-horizontal brushing (scrub) •

2-vertical brushing (leonard technique) •

3-circular brushing (fones technique) •

4-vibrotary technique(stillman technique) •

5-roll technique (modified stillman) •

6-charter technique •

7-sulcular technique (Bass technique) •

8-modified bass technique •

1-horizontal brushing (scrub) : most individuals use such method since it is simple. The head of the brush is positioned at a 90 angle to the tooth surface and then a horizontal movement is applied. The occlusal, lingual, palatal surface of the teeth are brushed with open mouth and vestibular surface are cleaned with the mouth closed.

2-vertical brushing (leonard technique)

It is similar to the horizontal brushing technique, but the movement is applied in a vertical direction using up and down motion.

3-circular (fones technique): with the teeth closed, a circular motion is applied that extend from the maxillary gingiva to the mandibular gingiva. Horizontal movement are used on the lingual and palatal tooth surface.

4-vibratory (stillman technique) the head of the brush is positioned in an oblique direction toward the apex, with the bristles placed partly on the gingival margin and partly on the tooth surface. Light pressure with a vibratory movement is then applied to the handle without moving the brush from its original position.

5-roll (modified stillman technique): the brush is positioned in a similar manner to the vibratory technique, but after applying a small vibratory pressure, the head of the brush is rolled in an occlusal direction.

6-charters technique: the head of the brush is positioned in an oblique direction with the bristles directed towards the occlusal surface. A vibratory(rotary) movement is then applied without moving the brush from its position. This method is effective in cases with receded interdental papilla because the bristles can penetrate the interdental space.

7-sulcular (bass technique): the head of brush is positioned in an oblique direction towards the apex and bristles are directed into the sulcus at 45 to the long axis of the tooth. The brush is moved in a back and front direction using short strokes. On the lingual surface in the anterior region the brush head is kept in the vertical direction. This method is effective in removing plaque not only at the gingival margin, but also could reach a depth of about 1mm sub gingivally.

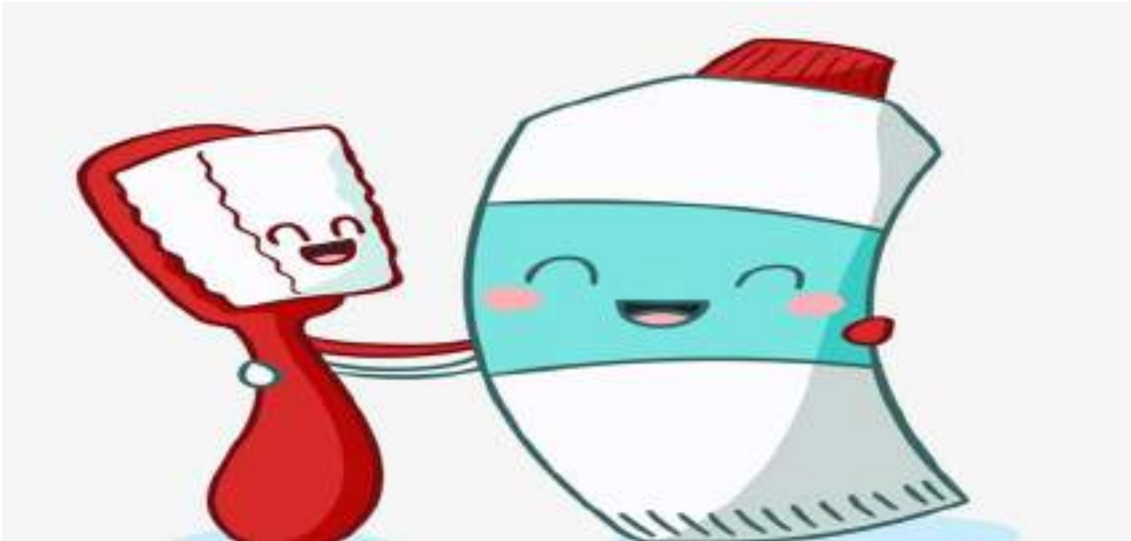
8-modified BASS technique: the brush is positioned similarly to the bass/ stillman technique , but after applying a back and front movement , the head of the brush is rolled in an occlusal direction. It is combination of the bass and modified stillman technique.

Tooth brushing requirement:

- -the features of manual toothbrush in periodontics must be Nylon, Soft-medium, rounded ends filaments, the small head is easier to reach all areas of oral cavity and should be trimmed flat and must be multi-tufted with same length.
- -the 3 brush head clean vestibular, occlusal, oral tooth surfaces this design is superior to other types of brush.

- **Frequency of brushing**: 2 in a day , just before going to bed
- **Duration:** for a minimum 2 minutes, toot brush should replaced every 2-3 months, because a worn toothbrush with frayed filaments loss resilience and less effective in removing of plaque





Electric toothbrushes:

Used mainly: •

1- elderly •

2-those with arthritis in their hands and wrists •

3-children •

4-hospitalized individuals •

5-physically or mentally landicapped •

6- neurological disorders •

7-consider easier and faster than manual. •

Dentifrices:

- -used in combination with tooth brush for plaque removal, should have:
- 1-fluoride: prevent caries
- 2-desensitizing agent.: for exposed dentin
- 3-anti-plaque agents(Triclosan, Stannous fluoride, cho
- Lorhexidine)
- 4-anti-calculus agent
- 5-bicarbonate , reduce the acidity of dental plaque
- 6-cleaning and polishing agent (abrasive agent)
- 7-whitening agents
- 8-detergents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Dental Floss:

-have a variety of thickness and type •

-have with or without wax •

-large interdental space use a thicker type than thinner one •

-40 cm floss used wind around middle finger, 10cm between middle • finger, 3cm between thumbs

-using a sawing movement •

-avoid fast movement to avoid damage of gingiva •

-do not worry if gum is bleed slightly after first one use , this stop • after number time of use,

- -**unwaxed** used in normal tooth contact
- -**waxed** used in tight proximal tooth contact
- -floss holder to facilitated flossing to use
- -**Tap**: type of broad dental floss used to clean bridge pontics
- -**Super floss**: used for crown , bridge and orthodontic appliance
- -flossed used in vertical direction not horizontal to avoid a develop of grooved surface
- -flossing is difficult and time consuming

Woodsticks:

- -made from wood and have a triangular cross section and a vailable in different thickness
- -hold between thumbs and first finger
- -don’t worry if gum bleed from the first cleaning with wood stick , this disappear after a repeat using of it.

Single tufted tooth brush

-used to clean can not reach by other type of brush •
such as locks, wire brace of orthodontic, groove

-back surface of last molar, long standing tooth. •

-used also in fixed appliance (crown and bridge) •


TePe
Pink
Orange
Red
Blue
Yellow
Green
Purple
Grey
Black


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Tongue cleaner:

-called scraper •

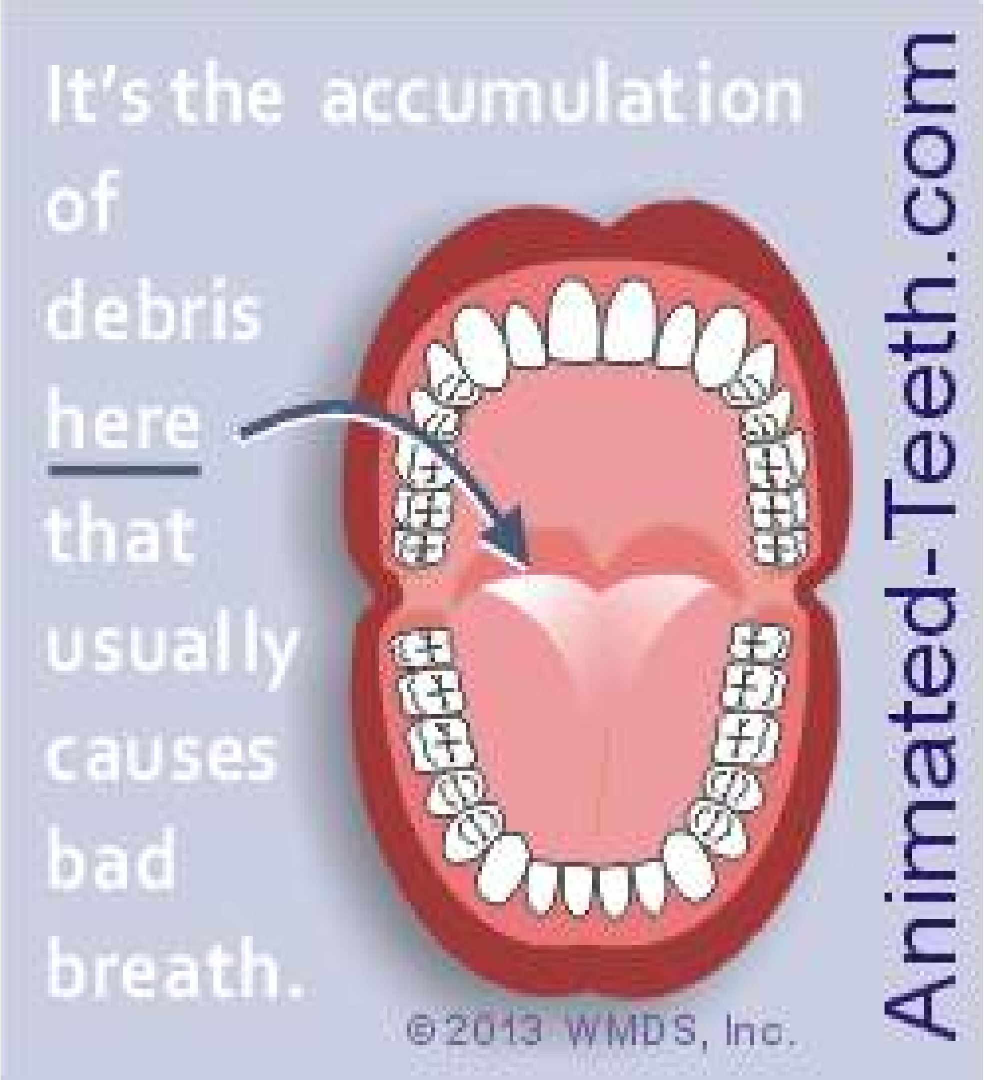
-gag reflex develop after using •

-remove bacteria between tongue groove •

-most effective one is loop shaped •

-clean mostly middle and first part •

-used mainly in patients with halitosis •


It's the accumulation of debris here that usually causes bad breath.
Animated-Teeth.com
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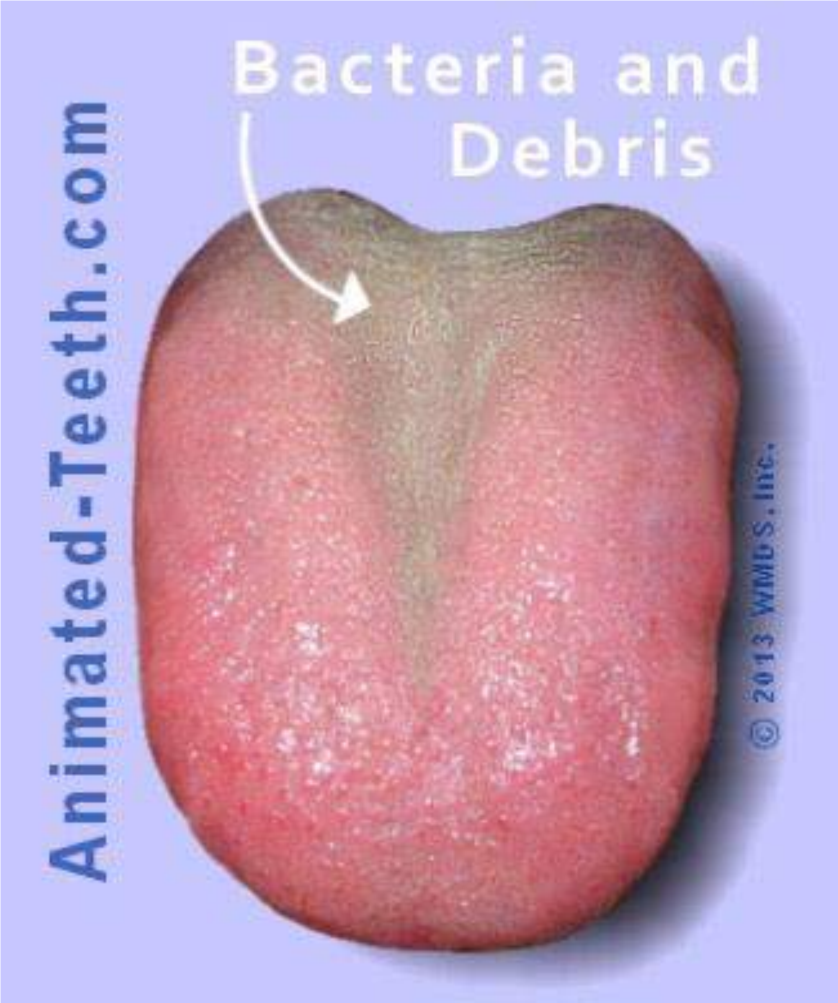
Bacteria and Debris
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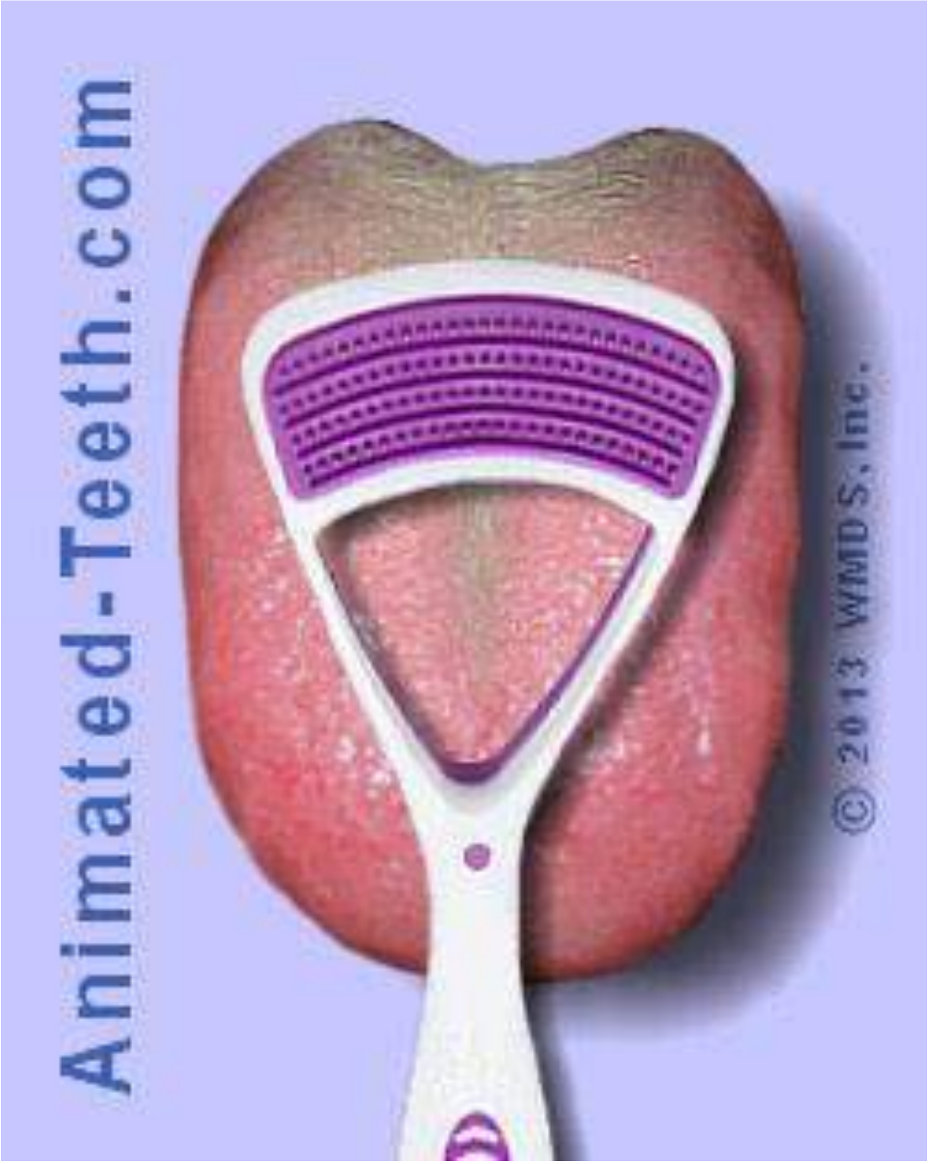
Animated-Teeth.com
© 2013 WMDS, Inc.

Inter dental brushes

Used when we have: •

-widely open interdental spaces •

-when root surface with concavities or groove have been • exposed

-in through –and-through furcation defects in periodontal • patients

-have different size and shape, most common forms are • cylindrical,conical shaped head

-can be used for carry fluoride or chlorhxidine gel •

-if is not properly used can be lead to dentin hypersensitivity •

-used without dentifrices •


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Dental water jet

- The daily used of oral irrigation has been shown to reduce gingivitis and bleeding
- -the pulsating hydrodynamic forces produced by irrigator can rinse away food debris from interdental spaces and plaque retentive area
- -use adjunct to brushing and flossing, and used with water or chorhxidine to improve plaque inhibition and had ati-inflammatory effect


An effective
Interdental
Cleaner

THANKYOU •

Periodontics

Treatment planning for patients With Periodontal Diseases

Dr. noor sabah irhayyim

The ultimate goal for every patient is to bring his or her mouth to a state of health and maintain it long term. This begins with educating the patient on the problems in his or her mouth and the etiologies, treatment, and prevention of these problems. A properly formulated treatment plan is paramount to achieving this goal. A treatment plan is a plan for therapy formulated only after a thorough examination has been completed, diagnosis and prognosis have been determined and the needs and desires of the patient have been taken into consideration. It must be recognized that as diagnosis and prognosis will change with treatment, therapeutic needs may also change. As such, the treatment plan must be changed accordingly.

The treatment plane for patients with periodontal disease include the following phases:

1. Phase I therapy (initial, cause related therapy, non-surgical therapy)

2. Phase II therapy (surgical therapy)

3. Phase III therapy (restorative therapy)

4. Phase IV therapy (maintenance therapy)

The aim of the treatment plan is total treatment that is, coordination of all the immediate, intermediate, and long-term goals for the purpose of creating a well-functioning dentition in a healthy periodontal environment.

The Immediate goals are the elimination of all infections and inflammatory processes that cause periodontal and other oral problems that may hinder the patient’s general health. Basically, the immediate goals are to bring the oral cavity to a state of health. This may require patient education on infectious oral diseases and disease prevention, periodontal procedures, endodontics, caries control, oral surgery, and treatment of oral mucous membrane pathologies. Referral to other dental and medical specialities may be necessary.

The intermediate goals are the reconstruction of a healthy dentition that not only fulfils all functional and aesthetic requirements but lasts many years. Restoration of health, function, aesthetic and longevity involves endodontic, orthodontic, periodontal, and prosthodontic considerations as well as the age, health, and desires of the patient.

The long-term goal is maintenance of health through prevention and professional supportive therapy. The long-term goal is set, and both the patient and the clinician work toward it from the very first visit. Once active disease has been controlled, all infectious and inflammatory processes have been eliminated and health has been attained, health should be maintainable for the rest of the patient's life.

Maintenance of health requires patient education on disease prevention and oral hygiene at the onset of treatment, meticulous daily home care by the patient, and patient adherence to professional recall maintenance at a regular interval.

The treatment plane is **the blueprint** for case management. It includes all procedures required for the establishment and maintenance of oral health and involves decisions regarding the following:

- Emergency treatment(pain,acute infections)
- Removal of nonfunctional and diseased teeth,and possibly strategic extraction of healthy teeth to facilitate the prosthetic reconstruction of the patient
- Treatment of periodontal diseases (surgical or nonsurgical, regenerative or respective)
- Endodontic therapy (necessary and intentional)
- Caries removal and placement of temporary and final restorations
- Occlusal adjustment and orthodontic therapy
- Replacement of missing teeth with removable or fixed dental prostheses or dental implants

· Aesthetic demands

· Sequence of therapy

Extracting or Preserving a Tooth

Removal, retention, or temporary(interim)retention of one or more teeth is an important part of the overall treatment plan. A tooth should be extracted under the following conditions:

· It is so mobile that function becomes painful.

· It can cause acute abscesses during therapy.

· There is no use for it in the overall treatment plan

In some cases, a tooth can be retained temporarily, postponing the decision to extract until after treatment is completed. A tooth in this category can be retained under the following conditions:

· It maintains posterior stops; the tooth can be removed after treatment, when it can be replaced by an implant or another type of prosthesis.

· It maintains posterior stops and may be functional after implant placement in adjacent areas. When the implant is restored, these teeth can be extracted.

· In the anterior aesthetic zone, a tooth can be retained during periodontal therapy and removed when treatment is completed and a permanent restorative procedure can be performed. The retention of this tooth should be not jeopardizing the adjacent teeth. This approach avoids the need for temporary appliances during therapy.

· Extraction of hopeless teeth can also be performed during periodontal surgery of the adjacent teeth. This approach reduces the number of appointments needed for surgery in the same area.

Sequence of Therapy

The periodontal treatment sequence is presented in the following figures. Immediately after completion of phase I therapy, the patient should be placed on the maintenance phase IV to preserve the results obtained and prevent any further deterioration and recurrence of disease. While on the maintenance phase, with its periodic evaluation, the patient enters into the surgical phase II and the restorative phase III of treatment.

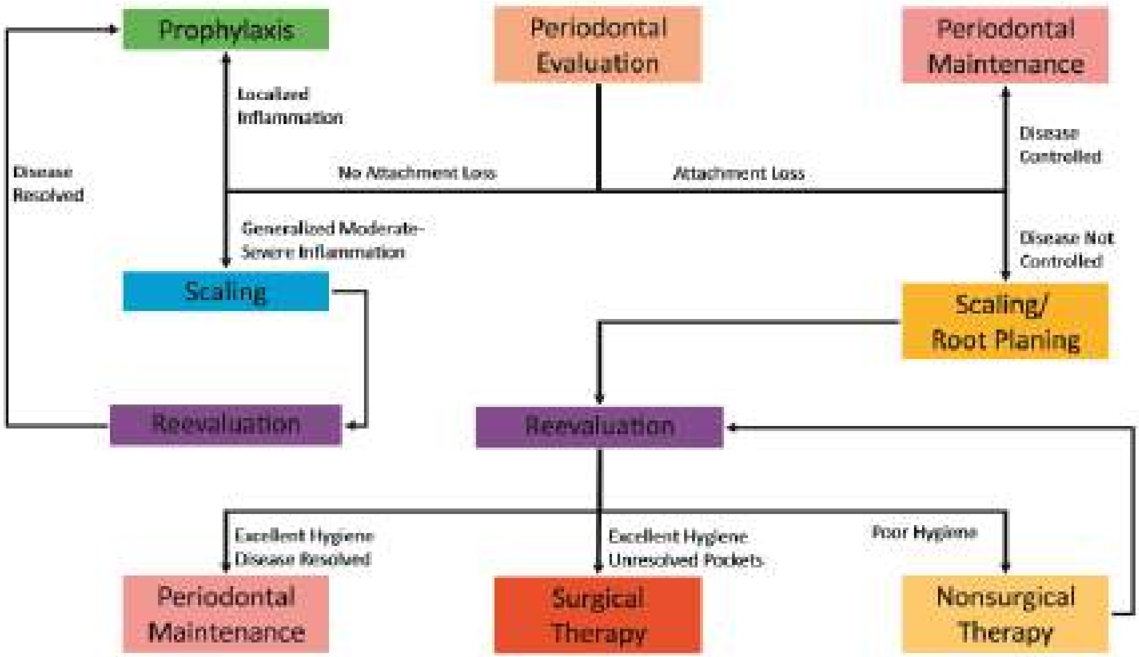

Fig. 36.1 Periodontal treatment decision tree.

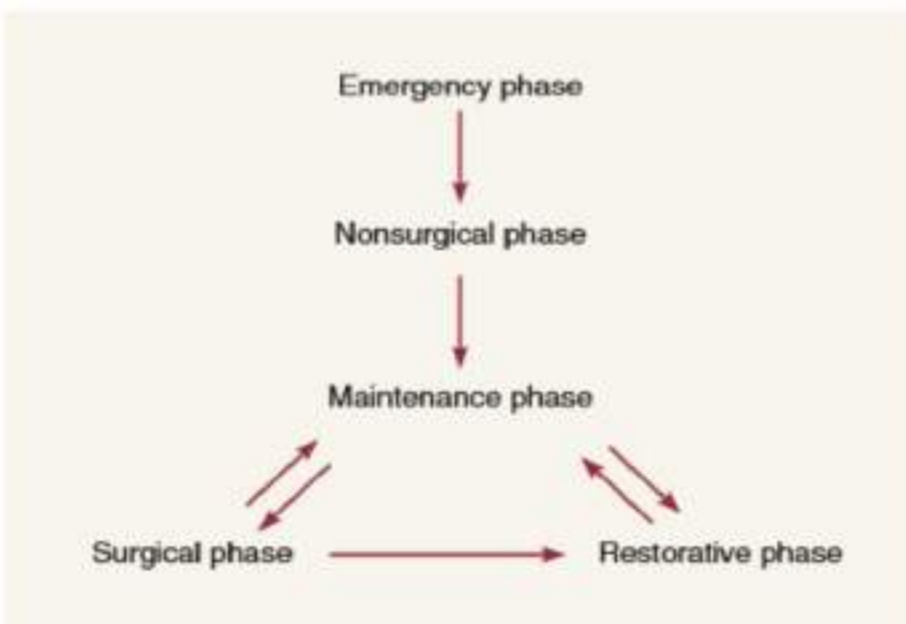

Fig. 36.2 Preferred sequence of therapy.

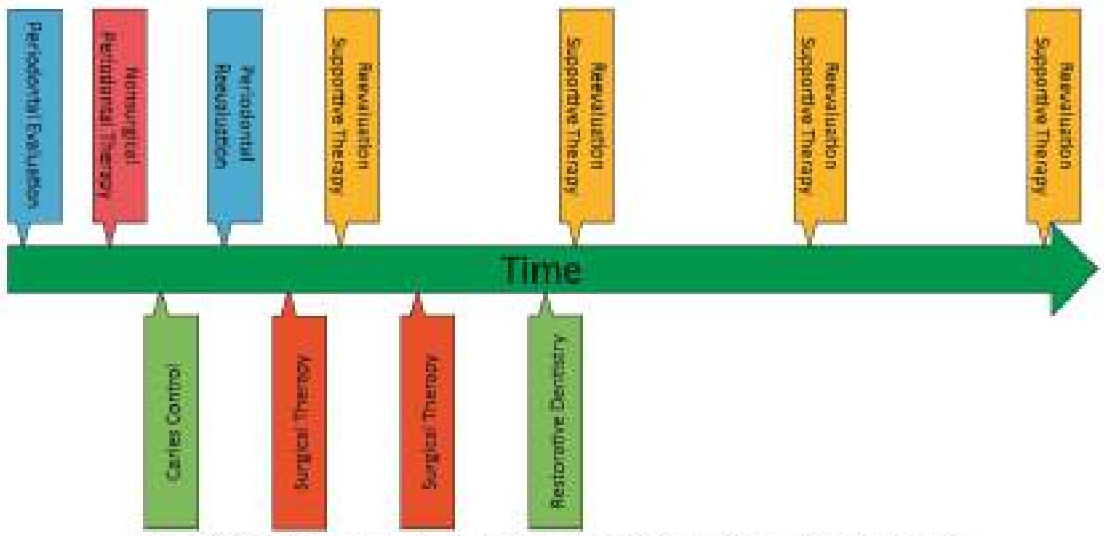

Fig. 36.3 Sample treatment timeline. Following nonsurgical periodontal therapy, the patient is placed on periodontal maintenance at regular intervals. Surgical and restorative treatments are scheduled between periodontal recalls.

Phase I Therapy

Objectives of initial phase I (cause – related therapy):

The objective is to alter or eliminate the microbial etiology and factors that contribute to gingival and periodontal diseases to the greatest extent possible, thereby halting the progression of disease and returning the dentition to a state of health and comfort. The phase I therapy aimed at removal of pathogenic biofilms, toxins and calculus and the reestablishment of a biologically acceptable root surface.

This is accomplished by:

1) patient education and oral hygiene instruction for plaque or biofilm control.

2)Complete removal of supragingival and subgingival plaque or biofilm and calculus (Scaling & root planning).
1) Possible use of antimicrobial agents (local or systemic).
2) Correction or replacement of poorly fitting restorations and other prosthetic devices
3) Restoration or temporization of carious lesions.
4) Treatment of Occlusal trauma.
5) Treatment of food impaction areas
6) Orthodontic tooth movement treatment.
7) Extraction of hopeless teeth

Motivation: Detailed information must be given to the patient regarding his/her periodontal disease, its etiological factors, symptoms, consequences, prognosis and the relationship between the presence of dental plaque and calculus in the mouth and the location of sites showing dental disease by using plaque disclosing agents. This information are aimed at motivating the patient to cooperate in the treatment hence without compliance (which has been described as the degree to which a patient follows a regimen prescribed by a dental professional), a good treatment outcome will not be achieved.

Mechanical plaque control demands active participation of the individual subject and the establishment of proper oral homecare habits is a process that depends on the behavioral changes, thus the patient's positive attitude to treatment may have a positive long-term effect on his/her tooth cleaning efforts. In addition, dental professionals should try to emphasize on the role of the patient personal oral hygiene procedures in the prevention of dental diseases &they should encourage the patient to take responsibility for his/her own oral health. Finally, if the clinician can establish the link between oral health & general health for the patient, this individual may be more willing to establish proper hygiene measures as part of his/her lifestyle.

Disclosing agent: Since dental plaque is white, sometimes it cannot easily be identified, particularly if it is not thick enough and/or the observer is not well trained. A disclosing agent is a chemical compound (tablets or solution) that stains dental plaque such as erythrosine, fuschsine or a fluorescein. These agents should be used to demonstrate the presence and location of plaque in addition to the evaluation of the efficacy of the patient's homecare technique thus they should be applied after tooth brushing and interdental cleaning.

Self-Performed Plaque Control:

Dental plaque is a bacterial biofilm that resides on tooth surfaces or soft tissues and is not easily removed from the surfaces of teeth. Supragingival plaque is exposed to saliva and to the natural self-cleansing mechanisms existing in the oral cavity, but such mechanisms do not adequately remove plaque. Therefore, the regular use of personal oral hygiene measures (refer to the efforts of the patient to remove suragingival plaque) is essential to the dental and periodontal health because plaque is the major etiological factor in periodontal disease thus plaque removal reduce symptoms of inflammation (bleeding, redness, swelling), inhibit the progression of the disease & inhibit the formation of supra & subgingival calculus which is a plaque retentive factor.

Furthermore, meticulous, long-term self-performed plaque removal measures can modify both the quantity & composition of subgingival plaque therefore, prevention of gingivitis, periodontitis and loss of attachment are based on the achievement of sufficient plaque removal. These practices require not only the appropriate motivation and instruction of the patient, but also the adequate tools.

Brushing: Although different cleaning devices have been used in different cultures (toothbrushes, chewing sticks, chewing sponges … etc.), the most widespread means of actively removing plaque at home is tooth brushing, The efficacy of brushing with regard to plaque removal is dependent on:

a) The design of the brush.

b) The skill of the individual using the brush.

c) Frequency & duration of brushing.

Therefore, oral hygiene instructions should include components such as self-assessment, self-examination, self-monitoring and self-instruction. For this purpose, plaque disclosing solution is applied before brushing, and with the aid of a mirror, the patient can identify the amount of plaque formed after the last brushing episode thus receiving information about his/her cleaning performance. Later on, the disclosing agent should be applied after brushing, which allows the patient to identify areas needing additional cleaning efforts.

Methods of tooth brushing:

Tooth brushing instruction should involve a description of specific brushing methods, the grasp of the brush, the sequence & amount of brushing, the area of limited access, and supplementary brushing of the occlusal surfaces and the tongue.
However, the design of brushes or a specific brushing method are of secondary importance to the skills of the individual in using the brush. Thus the simplest, least time consuming procedures that will effectively remove plaque without causing any damage to the tissues and use of the technique on a regular basis should be recommended.

If a patient prefers a specific method the clinician can evaluate & modify the technique to maximize the effectiveness rather than changing it. However, there is no single method that is correct for all patients. The morphology of the dentition (crowding, spacing, gingiva phonotype… etc.), the type and severity of the periodontal tissue destruction, the patient's own manual dexterity (= skill), as well as morphologic situation (longer teeth, Open interdental spaces, exposed dentin) during the course of periodontitis therapy determine what kind of hygiene aids and methods are to be used. Different tooth brushing methods have been recommended. Such methods can be classified based on the position & motion of the brush.

-***Horizontal brushing (scrub):*** Most individuals use such method since it is simple. The head of the brush is positioned at a 90° angle to the tooth surface and then a horizontal movement is applied. The occlusal, lingual & palatal surfaces of the teeth are brushed with open mouth and the vestibular surfaces are cleaned with the mouth closed.

· ***Vertical brushing (Leonard technique):*** It is similar to the horizontal brushing technique, but the movement is applied in a vertical direction using up & down motion.

· **Circular brushing (Fones Technique):** with the teeth closed, a circular motion is applied that extends from the maxillary gingiva to the mandibular gingiva. Horizontal movements are used on the lingual and palatal tooth surfaces.

· **Vibratory technique (Stillman technique)**:The head of the brush is positioned in an oblique direction toward the apex, with the bristles placed partly on the gingival margin and partly on the tooth surface. Light pressure with a vibratory movement is then applied to the handle without moving the brush from its original position.

· ***Roll technique (Modified Stillman technique):*** The brush is positioned in a similar manner to the vibratory technique, but after applying a small vibratory pressure, the head of the brush is rolled in an occlusal direction.

· ***Charters Technique:*** The head of the brush is positioned in an oblique direction with the bristles directed towards the occlusal surface. A vibratory (rotary) movement is then applied without moving the brush from its position. This method is effective in cases with receded interdental papilla because the bristles can penetrate the interdental space.

· ***Sulcular technique(Bass technique*)** The head of the brush is positioned in an oblique direction towards the apex and bristles are directed into the sulcus at 45° to the long axis of the tooth. The brush is moved in a back & forth direction using short strokes. On the lingual surfaces in the anterior regions the brush head is kept in the vertical direction. This method is effective in removing plaque not only at the gingival margin, but also could reach a depth of about 1mm **subgingivally**.

· ***Modified Bass technique:*** The brush is positioned similarly to the Bass/Stillman technique, but after applying a back-and-forth movement, the head of the brush is rolled in an occlusal direction. It is a combination of the Bass & the modified Stillman techniques.

Tooth brushes requirements:

The features of a manual toothbrush in periodontics must be Nylon, Soft-medium strength, rounded ends filaments. A smaller head is easier to reach all areas of the oral cavity and should be trimmed flat and be multi-tufted with all tufts being of the same length. The 3 brush heads clean vestibular, occlusal & oral tooth surfaces thus this design was superior to other brushes.

Frequency: Brushing twice a day is recommended, especially at night, just before going to bed.

Duration: Brush for a minimum of **2 minutes**, covering all areas of the oral cavity.

* It is recommended that the toothbrush is to be replaced every 2-3 months, because a worn toothbrush with frayed filaments loses resilience and is less effective in removing plaque than a new brush.

Electric toothbrushes: Studies have shown that efficiency in plaque removal with the electric toothbrushes is at least as good as correctly used manual toothbrush, but there are added benefits for those with limited manual dexterity, that includes the elderly, those with arthritis in their hands and wrists, children, hospitalized individuals, physically or mentally handicapped & patients with neurological disorders. Electric brushes has also been recommended to non-compliant patients as they are easier and faster than manual

Dentifrices: A dentifrice is usually used in combination with tooth-brushing with the purpose of facilitating plaque removal and applying agents to the tooth surfaces for therapeutic or preventive reasons. The most important active ingredient in toothpaste:

- **Fluoride:** prevent caries,
- Desensitizing agent: alleviate sensitivity of exposed dentin.
- Anti-plaque agents:

a. **Triclosan:** antibacterial agent.
b. **Stannous** fluoride.
c. **Chlorhexidine:** plaque inhibiting agent.

- **Anti- Calculus agent:** reduces the formation of supragingival calculus.
- **Bicarbonate:** reduce the acidity of dental plaque.
- **Cleaning + Polishing agents:** these abrasive agents should have particle size and shape which facilitate plaque & stain removal without producing hard & soft tissue damage
- **Whitening agents:** whiten stained teeth.
- **Detergents:** sodium Iauryl sulfate has antimicrobial & plaque inhibitory properties.

Interdental Cleaning

Since interproximal areas are
1) The worst for food & plaque stagnation
2) Earliest areas to be affected.
3) The tooth brush does not reach the interproximal spaces
efficiently as they are difficult to access.

Thus, gingivitis & periodontitis are usually more pronounced in these areas. Caries also occur more frequently in the interdental region;

therefore, interdental plaque removal, which cannot be achieved with toothbrush, is of critical importance for most patients. A number of interdental cleaning methods have been used for this purpose however, all these devices are effective but not all of them suit all patients or all types of dentitions.

Factors we need to consider when selecting the appropriate interdental cleaning method are:

- The contour & consistency of the gingival tissues.
- The size & shape of the interproximal space.
- The morphology of the proximal tooth surface.
- Tooth position & alignment.
- The manual dexterity & motivation of the patient.
- Fixed dentures & orthodontic appliances.
- Restorations

Dental floss & tape:

Flossing is the most universally applicable method. Clinical studies show that when tooth brushing is used together with flossing more plaque is removed from the proximal surfaces than by brushing alone. Flossing removes up to 80% of proximal plaque. Even subgingival plaque can be removed since dental floss can be introduced 2-3.5 mm below the tip of the papilla. Dental floss is most useful where the interdental papilla completely fill the embrasure space in healthy patients. Several types of floss are available:

1) Unwaxed is used in normal tooth contacts because it slides easily.
2) Waxed is used in tight proximal tooth contacts & after brushing because the wax deposits prevent fluoride from the toothpaste to

precipitate on teeth. However, no difference in the effectiveness between both types was demonstrated.
3) A floss holder to facilitate flossing might be used.
4) Tape: a type of broaded dental floss used for cleaning bridge pontics.
5) Super floss used for patients with crowns, bridges & orthodontic. Appliances. Recently, powered flossing devices have been introduced. Floss is used in a vertical direction. If it is used in a horizontal motion, the teeth can develop a grooved surface. Finally, flossing is a difficult & time-consuming method.

Wood sticks:

They are indicated for plaque removal, if the interdental spaces are slightly open (recession) and even in cases of poor manual dexterity since they are easy to use. Wood sticks are usually made of soft wood & have a triangular shape. Recently, brush sticks have been introduced they are elastic with tiny hair-like bristles and fine plastic files. Concavities can be cleaned very well with these devices.

Interdental brushes:

These are the aid of choice for:

- Widely open interdental spaces.
- When root surfaces with concavities or grooves have been exposed.
- In through-and-through furcation defects in periodontitis patients.

They are manufactured in different sizes & forms. The most common forms are cylindrical or conical shaped head. It is believed that the most efficient cleaning results are achieved if the brush selected slightly larger than the interdental space. They are easy to use & can also be used as a carrier to apply fluoride or chlorhexidine gel into the interdental space. When brushes are not properly used, they may cause dentin hypersensitivity, thus interdental brushes should be used without dentifrices except in special cases and for short term.

Single tufted brushes:

They are ideal for cleansing areas which cannot be reached with other devices. They are designed to improve access to distal surfaces of posterior molars, tipped & rotated teeth, to clean around & under fixed appliances, pontic, orthodontic appliances and teeth affected by gingival recession & furcation involvement.

Adjunctive aids:

1-Dental water jet: The daily use of oral irrigation has been shown to **reduce gingivitis** & **bleeding**. The pulsating hydrodynamic forces produced by irrigators can rinse away food debris from interdental spaces & plaque retentive areas. Irrigation is not however, a monotherapy but used as an adjunct to brushing & flossing. They may be used with water or with chlorhexidine that lead to improved plaque inhibition and had an anti-inflammatory effect. With specially designed tips the fluid may penetrate deeply into the pocket.

2-Tongue cleaners: The dorsum of the tongue harbors a great number of microorganisms. These bacteria may serve as a source of bacterial dissemination to other parts of the oral cavity (e.g. Tooth surfaces) and may contribute to dental plaque formation & halitosis. Therefore, tongue brushing or scraping has been advocated as part of daily home oral hygiene, together with tooth brushing & flossing to remove microorganisms & debris from the tongue. Patients should be informed t0 clean particularly the posterior portion of the dorsum.

Effects & sequelae of the incorrect use of mechanical plaque Removal devices:

Tooth brushing can cause damage both to soft & hard tissues. Trauma to the soft tissues results in gingival erosion & gingival recession. Trauma to hard tissues leads to cervical abrasion of the tooth surface which is mainly caused by the abrasives in the dentifrice. These lesions have been associated with toothbrush stiffness, the method of brushing, brushing frequency/time, excessive brushing force, and improper use of both manual and powered tooth brushing. The use of dental floss, interproximal brushes & wood sticks may also induce soft tissue damage; however, in most cases this damage is limited to acute lesions, such as lacerations and gingival erosions.