

## Chemical plaque control

The action of these chemical could fit into four categories:

1. Anti\_adhesive.
2. Antimicrobicrobial.
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.

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, the second effect could be bactericidal agent ,destroys all of the microorganisms either by attaching or already attached to the tooth surface.

Plaque removal agents: in mouth rinse to reach all tooth surfaces and act in an identical manner to a tooth brush and remove bacteria from the tooth surfaces have attracted the terminology of the chemical tooth brush e.g. Hypochlorite's.

Anti-pathogenic agents: might inhibit the expression of plaque microorganism's pathogenicity without necessarily destroying them and directly approaches to alter plaque ecology to a less pathologic flora, e.g. Antimicrobial agents with bacteriostatic effect.

### Chemical supra-gingival plaque control:

Chemical agents have been incorporated into mouth rinses and tooth pastes with the objective of inhibiting the formation of plaque and calculus .antiplaque agents may also have a significant clinical effect of resolving an established gingivitis.

Chlorohexidine digluconate:

CH is frequently used as a mouth rinse (0.2% or 0.12% w/v).the compound can also be applied as a gel ,spray, varnishes and has been incorporated into tooth paste, chewing gum, slow release vehicles (perio chip),periodontal packs and sub gingival irrigation.

At low concentrations, chlorhexidine is bacteriostatic, at high concentrations, it is bactericidal. The mode of action of chlorhexidine in killing bacteria is dependent upon the

drug having access to cell walls. This is facilitated by electrostatic forces, since chlorhexidine is positively charged, while the phosphate and carboxyl groups of bacterial cell walls carry negative charges. Binding causes disruption of the osmotic barrier and interference with membrane transport.

Rinsing with chlorhexidine reduce the number of bacteria in saliva by between 50% and 90% .a maximum reduction of 95% occurs around 5 days, after which the numbers of bacteria increase gradually to maintain an overall reduction of 70 %-80% at 40 days.

#### **Clinical uses of chlorhexidine:**

1. as an adjunct to oral hygiene and professional prophylaxis.
2. Post oral surgery including periodontal surgery or root planning.
3. for patients with jaw fixation.
4. Medically compromised individual predisposed to oral infections.
5. High risk caries patients.
6. in denture stomatitis.
7. Oral mal odor.
8. Recurrent oral ulceration.
9. Removable and fixed orthodontic appliance wearers.
10. Immediate preoperative chlorhexidine rinsing and irrigation.
11. reduced salivary flow.
12. for oral hygiene and gingival health benefits in the mentally and physically handicapped.

In oral use as a mouth rinse chlorhexidine has been reported to have a number of local side effects, thus it is only used for a few weeks at a time when it is not possible to carry out other oral hygiene procedures.

These side effects are;

1. Brown discoloration of the teeth and some restorative 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. Unilateral or bilateral parotid swelling.
5. Oral mucosal erosion.

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

CH is nontoxic even if digested or topically applied and has a broad antimicrobial action including wide range of gram positive & gram negative m.o. it is also effective against fungi and yeast including candida and some viruses (HBV & HIV).no report of bacterial resistance even after prolonged uses of CHX were recorded.

Studies suggest a slow release of CHX from surfaces to produce a persistent bacteriostatic action lasting for about 12hr. that's why it should be used twice a day.

### **Antimicrobials:**

The use of systemic antimicrobials in the management of periodontal disease should be restricted to the following conditions

1. Severe necrotizing ulcerative gingivitis.
2. Multiple or severe periodontal abscesses with involvement of regional lymph nodes.
3. Some cases of aggressive periodontitis.
4. Refractory periodontitis.

### **Route of administration:**

It is impossible to mechanically remove plaque completely from narrow grooves, narrow furcations and other bacterial reservoirs within the pockets. Thus it is appropriate to combine mechanical plaque control with antimicrobials. Since only a few bacterial species are potentially periodontal pathogenic, it is reasonable to eliminate these groups specifically. These groups contain bacteria that can invade periodontal tissues, making mechanical therapy alone ineffective.

Within the periodontal environment a concentration of the drug that is sufficient either to kill (bactericidal) or arrest growth (bacteriostatic) of pathogenic microorganisms.

Systemically ingested antimicrobials, whereby the drug enters the crevicular fluid and is able to bathe sub-gingival flora.

### **Advantages:**

.Eliminating pathogens, not only from periodontal lesions but also from the oral cavity. (Reach widely distributed microorganisms).

.Such an action may have prophylactic benefits and reduce the risk of reinfection of the periodontal sites.

.broad spectrum of activity.

### **Disadvantages:**

-systemic side effects.

-the possible elimination of non-pathogenic “beneficial” bacteria.

-low concentration within the tissues.

-bacterial resistance.

-requires good patient compliance.

-interaction with other medications.

-Allergic reactions.

-Super infections of opportunistic bacteria.

-High doses of antimicrobials are administered.

### **Advantages of local route of administration:**

-lower doses of antimicrobials are administered.

-High local concentrations of the drugs are achieved locally in periodontal pockets so better effect against biofilms.

-Minimal or no side effects.

-Administration is not dependent upon patient compliance.

-placement is site specific.

-When the matrix (vehicle) biodegrades to release the drug (controlled slow release device), an antimicrobial sustain its localized concentration of effective levels for a sufficient time.

### **Disadvantages:**

-Narrow, limited spectrum of efficacy.

-Possible reinfection of non-treated sites.

-The placement can be time consuming when the treatment of multiple sites is indicated.

-The extent to which the drug penetrates the connective tissues may be less predictable than when systemic dosing is undertaken.

### **Tetracycline:**

Is a group of related bacteriostatic antimicrobials .they provide a broad spectrum of activity against both gram-positive and gram-negative microorganisms.tetracycline is effective against most spirochetes and many anaerobic and facultative bacteria. Additional properties of tetracycline that may be valuable in the management of periodontal disease are

- inhibition of collagenase (inhibit tissue destruction).
- Anti-inflammatory actions.
- Enhancement of fibroblast attachment to root surfaces.
- inhibition of bone resorption and may aid bone regeneration.
- High drug concentration to be delivered into pocket (concentration in gingival sulcus 2-10 times that in serum).

In chronic periodontitis, systemic tetracycline has little advantage when used as an adjunct to other procedures. Systemic tetracycline is valuable in the management of localized aggressive periodontitis and refractory periodontitis. In localized aggressive periodontitis, the prime pathogen is *Aggregatibacter actinomycetemcomitans* (A.a), which is very susceptible to tetracycline.

Sub-antimicrobial dose of doxycycline 20mg 2/d for 3 months for maximum of 9 months approved and indicated as an adjunct to S&RP in the treatment of periodontal disease, e.g. refractory periodontitis, which act by a mechanism called host modulation that refers to the concept of modulating the host response to the presence of bacteria with methods such as inhibiting collagen destructive enzymes hence, this regimen creates no bacterial resistance.

Tetracycline has been incorporated into slow release devices for adjunctive local treatment following S&RP. e.g. Minocycline ointment, also been available for local application.

### **Metronidazole:**

Antibacterial activity against anaerobic –cocci, gram –negative and gram-positive bacilli has led to the use of metronidazole in the treatment of periodontal disease.

In the cell, metronidazole binds and disrupts DNA synthesis leading to cell death. This process results in rapid killing of anaerobic microorganisms (bactericidal). It is effective against *Porphyromonas gingivalis*.

In periodontal treatment, metronidazole has been used systemically; common dosage is 200mg three times a day for 3-5 days. For more severe infections the dose is increased to 400mg twice daily for 3-5 days.

Metronidazole has been found to be very effective when combined with amoxicillin in the treatment of refractory localized aggressive periodontitis that has not responded to conventional periodontal treatment and tetracycline therapy. A 7 days (250mg of each drug).

Efficacy studies suggest that two applications of 25% metronidazole gel (1 week apart) in periodontal pocket are as effective as conventional non-surgical management in reducing probing depths and bleeding on probing.

**Amoxicillin:**

Had extended antimicrobial spectrum that includes gram positive and gram negative bacteria by inhibiting bacterial cell wall production and therefore are bactericidal, hence may be useful in the management of patients with aggressive periodontitis, the dosage is 500mg for 8 days.

Augmentin (Amoxicillin with clavulanate), this combination makes it resistant to penicillinase enzymes produced by some bacteria, hence may be useful in the management of patients with refractory or localized aggressive periodontitis. The Augmentin with Metronidazole combination have an additive effect regarding suppression of A.a in localized aggressive periodontitis.

**Nonsteroidal Anti-inflammatory Drugs (NSAID):**

May be of therapeutic value in treating periodontal disease because of their ability to inhibit the inflammatory process, drug such as flurbiprofen, ibuprofen, mefenamic acid and naproxen.

## Classification of periodontal disease

- Classification of disease is necessary to try to separate conditions into distinct categories in order to aid clinical and laboratory diagnosis and specific treatment
- The idea of separating diseases in this way should ideally be based on aetiology, histopathology and where appropriate, genetics rather than age of onset and rates of disease progression
- Over the last three decades there have been three major attempts to classify periodontal disease

1- First World Workshop in Clinical Periodontics in 1989 ( American Academy of Periodontology 1989)

2- Second, First European Workshop in Periodontics in 1993 (Attstrom & van der Velden 1994)

Those classification had many shortcomings including

- 1) Considerable overlap in disease categories
- 2) Absence of gingival disease component
- 3) The age of onset of disease& rates of progression are not clear
- 4) Unclear classification criteria

**3- The third** attempt was started by the American Academy of Periodontology in 1997, who organised the International Workshop for a Classification of Periodontal Diseases and Conditions in 1999

- At this workshop, a new classification was agreed upon (Armitage 1999)
- This attempted to develop a comprehensive classification of gingival diseases, periodontal diseases, necrotizing ulcerative gingivitis/periodontitis, periodontal abscesses, periodontitis associated with an endodontic lesion, developmental or acquired deformities and conditions mucogingival deformities and conditions in addition to occlusal trauma
- This classification includes both separate conditions and a number of other factors which may affect their severity or clinical presentation and is shown in tables 1 and 2

**The main changes in this classification are:**

- 1-** The addition of a comprehensive section on **gingival diseases**
- 2-** Replacement the term of **adult periodontitis** with **chronic periodontitis**, since epidemiological evidence suggests that chronic periodontitis may also be seen in some adolescents
- 3-** The term **refractory periodontitis** is no longer considered as a separate disease entity and has been excluded from the classification. This term can be applied to any disease that proves non response to treatment



The old term **recurrent periodontitis** denotes the return of the disease and is not a separate disease entity and has been excluded from the classification

- Elimination of these separate categories because of the lack of evidence that they represent separate conditions but rather describe the response to treatment that result from differences in patient susceptibility

4- Replacement of the term **early onset periodontitis** ( includes: 1) prepuberty, 2) juvenile and 3) rapidly progressive periodontitis with **aggressive periodontitis**, largely because of the clinical difficulties in determining the age of onset in many of these cases

5- A new classification group of periodontitis **as a manifestation of systemic disease** has been created and this includes those cases of prepubertal periodontitis directly resulting from known systemic disease

6- There are also new group categories on periodontal abscesses, periodontic-endodontic lesions and developmental or acquired deformities or conditions

In summary the classification of periodontal disease include the following 8 categories:

- I) Gingival diseases
- II) Chronic periodontitis
  - A. Localized (< 30% involved sites)
  - B. Generalised (>30% involved sites)
- III) Aggressive periodontitis
  - A. Localized
  - B. Generalised
- IV) Periodontitis associated with systemic disease
- V) Necrotising periodontal diseases
  - A. Necrotizing ulcerative gingivitis
  - B. Necrotizing ulcerative periodontitis
- VI) Abscess of the periodontium
- VII) Periodontitis associated with endodontic lesions
- VIII) Developmental or acquired deformities & conditions

Table 1 Classification of gingival diseases:

| <u>A Plaque induced gingival disease</u>                   |  | 3. Gingival disease of fungal origin              |
|--|--|---|
| 1. Gingivitis associated with dental plaque only           |  | a) <i>Candida</i> species infections              |
| a) without other locally contributing factors              |  | i) generalized gingival candidiasis               |
| b) with locally contributing factors                       |  | b) linear gingival erythema                       |
| 2. Gingival disease modified by systemic factors           |  | c) Histoplasmosis                                 |
| a) associated with endocrine system                        |  | d) other  |
| i) puberty-associated gingivitis                           |  | 4. Gingival diseases of genetic origin            |
| ii) menstrual cycle-associated gingivitis                  |  | a) hereditary gingival fibromatosis               |
| iii) pregnancy-associated gingivitis or pyogenic granuloma |  | b) other  |
| iv) diabetes mellitus-associated gingivitis                |  | 5. Gingival manifestations of systemic conditions |
| b) associated with blood dyscrasias                        |  | a) mucocutaneous conditions                       |
| i) leukaemia-associated gingivitis                         |  | 1) lichen planus                                  |
| ii) other  |  | 2) pemphigoid                                     |
| 3. Gingival disease modified by drugs                      |  | 3) pemphigus vulgaris                             |
| a) drug-influenced gingival diseases                       |  | 4) erythema multiformi                            |
| 1) drug-influenced gingival enlargement                    |  | 5) lupus erythematosus                            |
| 2) drug-influenced gingivitis                              |  | 6) drug-induced                                   |
| a) oral contraceptive-associated gingivitis                |  | 7) other  |
| b) other   |  | b) allergic reactions                             |
| 4. Gingival disease modified by malnutrition               |  | 1) dental restorative materials                   |
| a) ascorbic acid-deficiency gingivitis                     |  | a) mercury  |
| b) other   |  | b) nickel   |
|  |  | c) acrylic  |
|  |  | d) other  |
|  |  | 2) reactions attributable to:                     |
|  |  | a) toothpastes/dentifrices                        |
|  |  | b) mouthrinses/mouthwashes                        |
|  |  | c) chewing gum additives                          |
|  |  | d) foods and food additives                       |
| <u>B Non plaque-induced gingival lesion</u>                |  | 6. Traumatic lesions                              |
| 1. Gingival disease of specific bacterial origin           |  | (factitious, iatrogenic, accidental)              |
| a) <i>Neisseria gonorrhea</i> -associated lesions          |  | a) physical injury                                |
| b) <i>Treponema pallidum</i> -associated lesions           |  | b) chemical injury                                |
| c) <i>Streptococcal</i> species-associated lesions         |  | c) thermal injury                                 |
| d) other   |  | 7. Foreign body reactions                         |
| 2. Gingival diseases of viral origin                       |  | 8. Not otherwise specified (NOS)                  |
| a) Herpes virus infections                                 |  |   |
| 1) primary herpetic gingivostomatitis                      |  |   |
| 2) recurrent oral herpes                                   |  |   |
| b) oral Epstein-Barr virus lesions                         |  |   |
| c) <i>Varicella-Zoster</i> infections                      |  |   |
| d) others  |  |   |

## Classification of Gingival diseases

### A. Dental plaque induced gingival diseases

There are four main types of plaque-associated gingival diseases

1. Gingivitis associated with **dental plaque only**
2. Gingivitis modified by **systemic factors**
3. Gingivitis modified by **medications**
4. Gingivitis modified by **malnutrition**

There are common characteristics to all gingival diseases associated with plaque, modified by systemic diseases, medications and malnutrition:

- 1- Signs and symptoms that are confined to the gingiva
- 2- The presence of dental plaque to initiate and /or exacerbate the severity of the lesion
- 3- Clinical signs of inflammation (enlarged gingival contours due to oedema or fibrosis, colour transition to a red and or bluish-red, elevated sulcular temperature, bleeding upon stimulation, increased gingival exudate)
- 4- No loss of attachment
- 5- Reversibility of the disease by removing the etiological factors

## 1. Gingivitis associated with dental plaque only

It is called plaque induced gingivitis and it is inflammation of the gingiva resulting from dental plaque only, it is either

- With or without local contributing factor
- The local contributing factors can be defined as a local feature that may influence the presentation of the disease, such as:
  - Overhanging restoration
  - Dental calculus
  - Prosthetic and orthodontic appliances

### Characteristics of plaque-induced gingivitis:

- 1- Plaque presents at a gingival margin
- 2- Disease begins at the gingival margin
- 3- Change in gingival contour
- 4- Change in gingival colour
- 5- Change in sulcular temperature
- 6- Increased gingival exudation
- 7- Bleeding upon probing
- 8- Absence of bone loss
- 9- Absence of attachment loss
- 10- Histological changes
- 11- Reversible with plaque removal

## 2. Gingival diseases modified by systemic factors

### A. Associated with the endocrine system:

- 1- Puberty-associated gingivitis
- 2- Menstrual cycle-associated gingivitis
- 3- Pregnancy-associated **gingivitis** and **pyogenic granuloma**
- 4- Diabetes mellitus-associated gingivitis

**1- Puberty-associated gingivitis** is pronounced inflammatory response of the gingiva to the dental plaque and hormones during the circumpubertal period (11-16) years

**2- Menstrual cycle-associated gingivitis** is a pronounced inflammatory response of the gingiva to the plaque and hormones immediately prior to ovulation

**3- Pregnancy-associated gingivitis** is pronounced inflammatory response of the gingiva to the dental plaque and hormones usually occurring during the second and third trimesters

**4- Pregnancy-associated pyogenic granuloma** is a localised, painless, protuberant, exophytic genial mass that is attached by a sessile or pedunculated base from the gingival margin or more commonly from interproximal pregnancy. It is more common in maxilla and may develop as early as the first trimesters and may regress or completely disappear following parturition (delivery)

**4- Diabetes mellitus-associated gingivitis:** is an inflammatory response of the gingiva to plaque aggravated by poorly controlled blood glucose levels

**B. Associated with blood dyscrasia:** is associated with abnormal function or number of blood cells

- Leukaemia-associated gingivitis is pronounced inflammatory response of the gingiva to the dental plaque, resulting in increased bleeding and enlargement subsequent to leukaemia
- Gingival bleeding is a common sign in patients with leukaemia, which is considered as the initial oral sign and or symptoms in 17.7 % and 4.4% of patients with acute and chronic leukaemia respectively
- Gingival enlargement initially begin at the interdental papilla followed by marginal and attached gingiva



## Dental plaque biofilm

Bacteria are the primary etiologic agents in periodontal disease. It has been found more than 500 distinct microbial phenotypes present in dental plaque. These kinds of bacteria have evolved to survive in the environment of tooth surface, gingival epithelium and oral cavity.

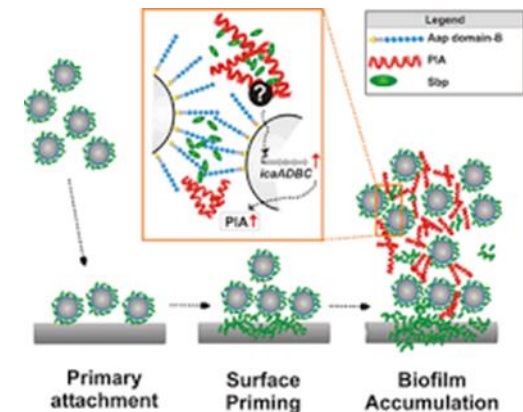
**Dental plaque** is defined as a soft yellow-greyish deposits that form the biofilm adhering to the tooth surface or other hard surfaces in the oral cavity such as removable and fixed restorations.

- Dental plaque consists primarily from microorganisms and intercellular matrix along with scattering epithelial cells, leukocytes and macrophages.
- The presence of tough extracellular matrix makes it impossible to remove by rinsing or using sprays

**Biofilm** is defined as the relatively undefinable microbial community associated with a tooth surface or any other hard non shedding material.



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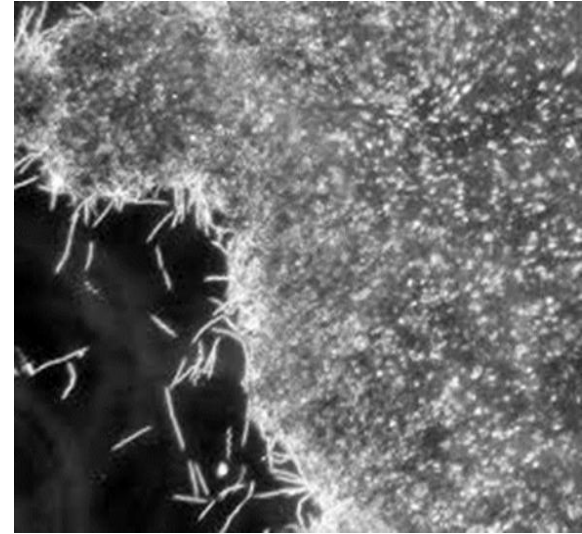
## Dental plaque as a biofilm

Structurally dental plaque is now considered to be as a biofilm of complex and dynamic microbial community areas.

- It contains areas of high and low bacterial biomass interlaced with aqueous channels of different sizes comprise the nutrient channels for bacterial colonization.
- The intercellular matrix forms a hydrated gel in which bacteria can survive and proliferate.
- Biofilm adheres firmly to the tooth surface and resists to mechanical removal as well as antibiotics
- Biofilm is a fascinated structure, which functions look like multicellular organisms ,characterised by shedding of bacterial surface components ( antigens, which can activate a host immune response) and release of various toxins ( endotoxin, which can activate a host inflammatory response) which cause host tissue damage
- The biofilm plays a major role in protecting the colonisation species from host defence mechanisms

## Biofilm structure

- Biofilm is composed of microcolonies of bacterial cells (15-20% by volume), which are distributed in matrix or glycocalyx ( 70- 80% by volume)
- Thick biofilms have demonstrated presence of water channels between the microcolonies.
- These water channels permit the passage of nutrient and other agents through out the biofilm acting as a circulating system
- Some of the functions of the biofilm depend on the ability of bacteria and microcolonies within the biofilm to communicate with each other
- This activity is called ' quorum sensing' in which bacteria secrete a signalling 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 reached at a high-cell density and therefore bacteria sense that the population has reached a critical mass or quorum
- Some evidence showed that the intercellular communication can occur after cell-cell contact and herein may not involve secreted signaling molecules



**Plaque** is different from other deposits that may be found on the tooth surface such as **Materia alba** and **calculus**

**Materia alba** refers to soft accumulations of bacteria, food matter and tissue cells that lack of the organised structure of dental plaque and can easily displaced with a water spray

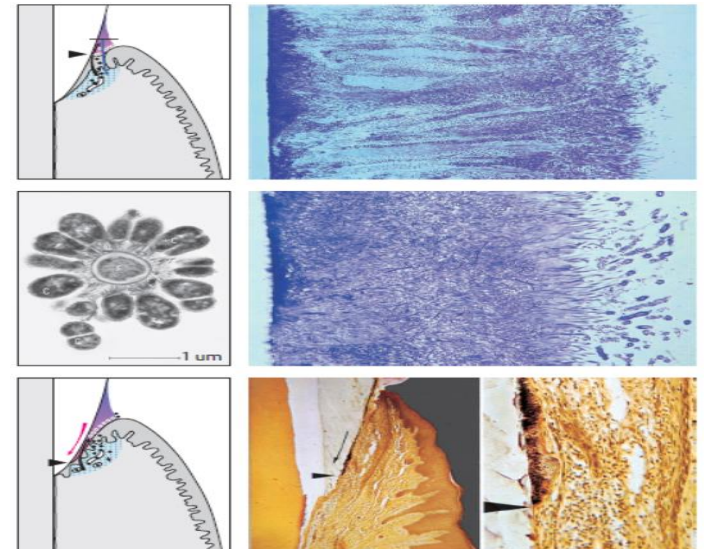
**Calculus** is a hard deposit that forms by mineralisation of dental plaque and is generally covered by a layer of unmineralized plaque

### Classification of dental plaque

1. Supragingival plaque
2. Subgingival plaque

**Supragingival plaque** is found on or above gingival margin, and where it located in a direct contact with the gingival margin is referred to a marginal plaque

**Subgingival plaque** is found below the gingival margin between the tooth and the gingival sulcular epithelium



**Microbiological studies** indicate that there is a difference between tooth-associated and tissue associated regions of subgingival plaque

- Different regions of plaque have significant different processes associated with disease of periodontium
- For example, marginal plaque is of importance in the development of **gingivitis**
- While supragingival and tooth-associated bacteria are critical in **calculus** formation and **root caries**
- Tissue-associated subgingival plaque is important in the soft tissue destruction ( different forms of periodontitis)

**Dental plaque** is composed of microorganisms, where one gram of plaque in a wet condition contains  $2 \times 10^{11}$  bacteria

- The number of bacteria in supragingival plaque on a single tooth surface can range from  $10^3$  on a healthy crevice compared to  $>10^8$  bacteria in a deep pocket

In addition to bacteria dental plaque contains non bacterial microorganisms such as yeasts, protozoa and viruses

- It contains cells such as epithelial cells, macrophages and leukocytes

The inter cellular matrix , estimated to comprise 20-30% of plaque mass

- It consists of **organic** and **inorganic** materials derived from saliva, gingival crevicular fluid
- **Organic constituents** include polysaccharides, proteins glycoproteins and lipid material
- **Inorganic constituents** is mainly **calcium** and **phosphorus** with trace amount of other minerals such as sodium, potassium and fluoride
- The source of inorganic component of supragingival plaque is from saliva
- Whereas in subgingival it is derived from crevicular fluid

## Formation of dental plaque

Dental plaque may be visualised on teeth after 1-2 days with no oral hygiene measures

- Movement of tissues and food materials over teeth results in mechanical removal of plaque
- Such removal of plaque is effective on the coronal two thirds of the tooth surface
- Therefore, plaque is typically observed on the gingival third of the tooth surface

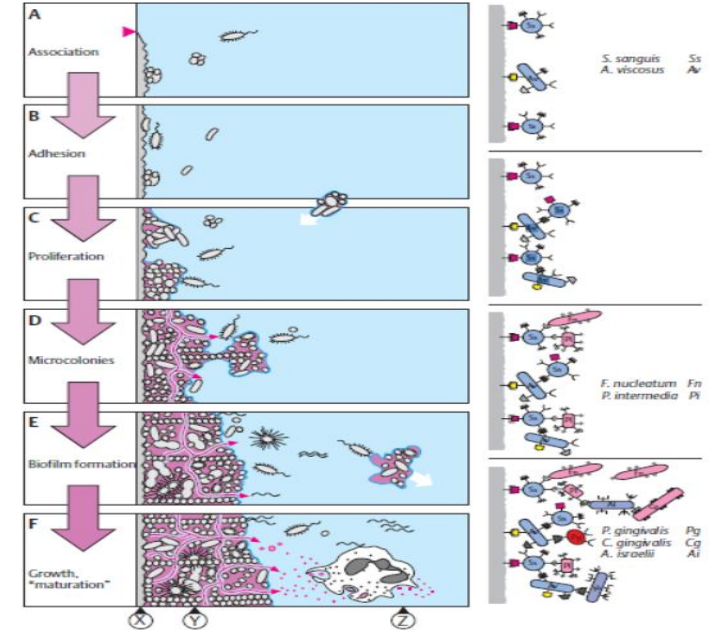
The **process of plaque formation** can be divided into three phases

- 1- Formation of the pellicle coating on the tooth surface
- 2- Initial colonisation by bacteria
- 3- Secondary colonisation and plaque maturation

## Formation of the pellicle

All surfaces in the oral cavity including hard and soft tissues are coated with a layer of organic material known as pellicle

- Its derived from saliva and crevicular fluid components, bacterial and host tissue cells products and debris
- The pellicle on the tooth surface consists of more than 180 peptides, proteins and glycoproteins including keratins, mucins, histidine-rich proteins, proline rich proteins and phosphoproteins
- The mechanisms involved in enamel pellicle formation include electrostatic van der Waals and hydrophobic forces
- Salivary pellicle can be detected on clean surfaces within 1 min
- By 2 hours, the pellicle is essentially in equilibrium between adsorption and detachment, although further pellicle maturation can be observed for several hours



## Pellicle functions

- 1- Protective barrier, providing lubrication for surfaces and preventing tissue desiccations
- 2- It provides a substrate to which bacteria can attach, as bacteria do not contact the enamel directly but interact with the enamel pellicle ( the pellicle is not merely a passive adhesion matrix)
- 3- Many proteins retain enzymatic activity when incorporated into the pellicle and some of these proxidases and amylase may affect the physiology and metabolism of adhering bacterial cells

## Initial colonisation of the tooth surface

Tooth brushing removes most but not all bacteria from the exposed surfaces of the teeth

- However, recolonization begins immediately and bacteria can be detected within 3mins of introducing sterile enamel into the mouth
- There are specific molecules on the bacterial surface called **adhesin**, which interact with receptors present in the dental pellicle
- This can determine whether or not a bacterial cell will remain associated with the surface
- Only a small proportion of oral bacteria possess adhesin that interact with receptors in the host pellicle and these microorganisms are generally the most abundant bacteria in the biofilms on tooth enamel shortly after cleaning
- Over the first 4-8 hours, 60-80% of bacteria present are members of the genus streptococcus

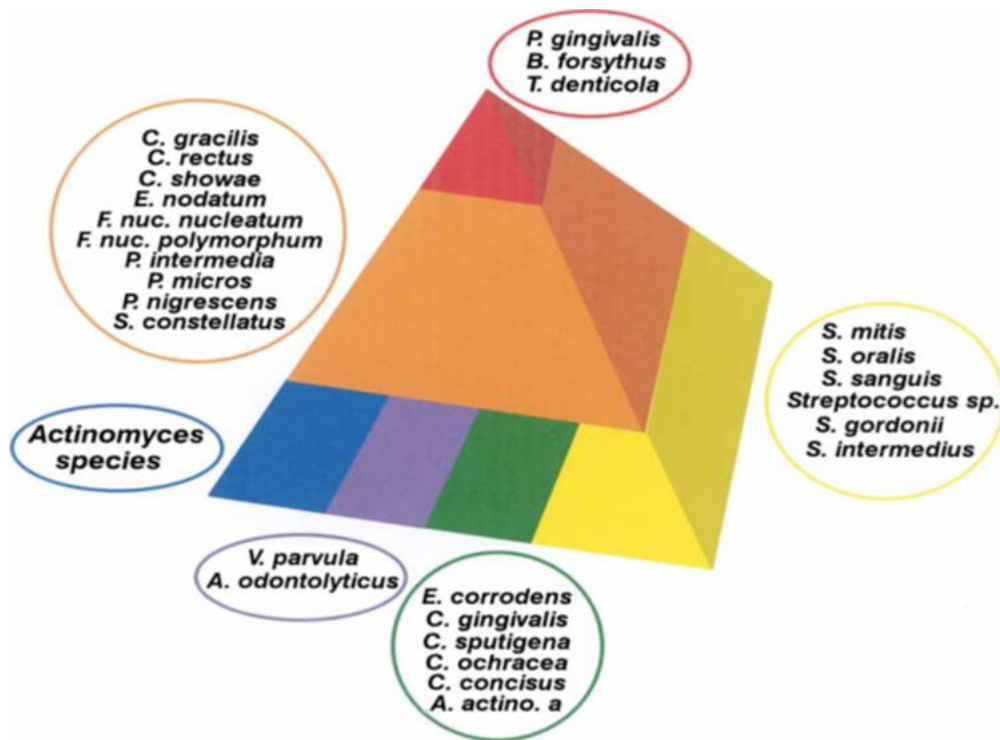
- Other bacteria commonly present at the this time include species that cannot survive without oxygen (obligate aerobes) such as *Haemophilus* spp and *Neisseria* spp, as well as organisms that can grow in the presence or absence of oxygen (facultative anaerobes) including *Actinomyces* spp and *Veillonella* spp
- These species are considered the primary colonisers on the tooth surface, which provide new binding sites for adhesion by other oral bacteria
- The metabolic activity of the primary colonisers modifies the local microenvironment in a way that can influence the ability of other bacteria to survive in the dental plaque biofilm
- For example, by removing oxygen, the primary colonisers provide conditions of low oxygen tension that permit the survival and growth of obligate anaerobes

### Secondary colonisation and plaque maturation

- The primary colonising bacteria adhered to the tooth surface provide new receptors for attachment with other bacteria in a process known as **co-adhesion**
- Co-adhesion leads to the development of microcolonies and eventually to a mature biofilm
- Different species or even different strains of a single species have distinct sets of coaggregation partners
- Secondary colonisers microorganisms include *Prevotella intermedia*, *Capnocytophaga* spp., *Fusobacterium nucleatum* and *Porphyromonas gingivalis*
- *Fuso* bacteria coaggregate with all other human oral bacteria while *Veilloella* spp, *Capnocytophaga* spp and *Prevotella* spp bind to *stpretococci* and or *actinomyces*



- Each newly accreted cell becomes itself a new surface and therefore may act as a coaggregation bridge to the next potentially accreting cell type that passes by well-characterised interactions of secondary colonisers with early colonisers include the coaggregation of *F. nucleatum* with *S. sanguinis*
- The transition from early supragingival dental plaque to mature plaque growing below the gingival margin involves a shift in the microbial population from primarily gram-positive organisms to high numbers of gram-negative bacteria
- Examples of these types are coaggregation of *F. nucleatum* with *P. gingivalis* or *Treponema denticola*



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## Dental biofilms: difficult therapeutic targets

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Periodontal diseases are infections caused by micro-organisms that colonize the tooth surface at or below the gingival margin. While these infections have many properties in common with other infectious diseases, they exhibit unique properties conferred by their site of colonization and the nature of the environment in which they reside. Table 1 presents an overly simplified summary of four crude categories

body and the infection is usually rapidly resolved by a "cure", by removal of some body part or by demise of the patient. Treatment of these infections is usually supportive, although antibiotics are often used in more severe cases. Examples of such infections include local abscesses caused by organisms such as *Staphylococcus aureus*, upper respiratory infections caused by organisms such as *Streptococcus*

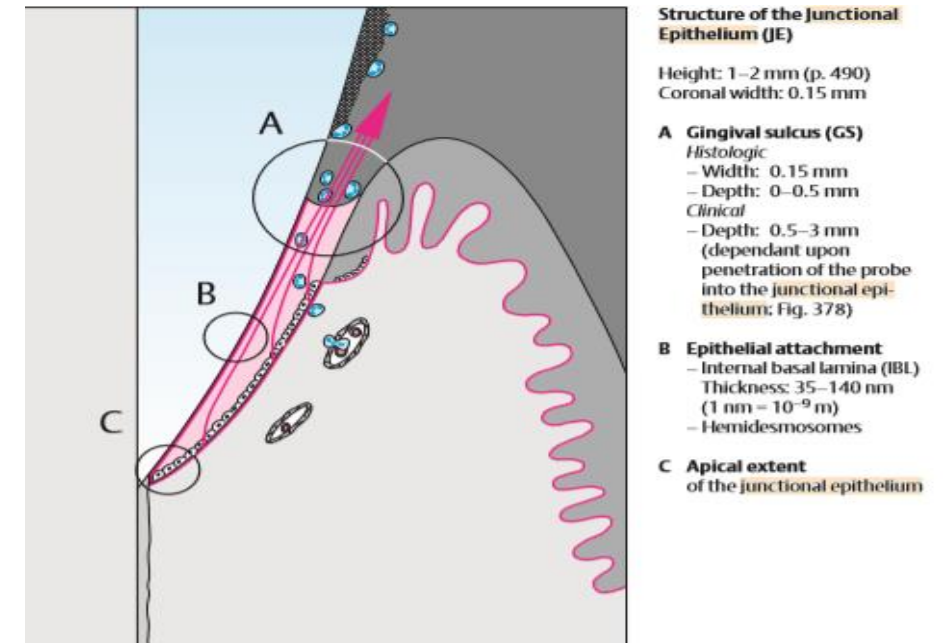
## Junctional epithelium (JE)

The type of epithelium that attached the gingiva to the tooth surface. It consists of stratified squamous non-keratinizing epithelium. It is usually consisted of 3-4 of thick layers in early life, however, the number of layers increases with age to 10-20. It is thicker in the coronal portion but become thinner toward cemento-enamel junction.

Junctional epithelial cells can be grouped in two layers:

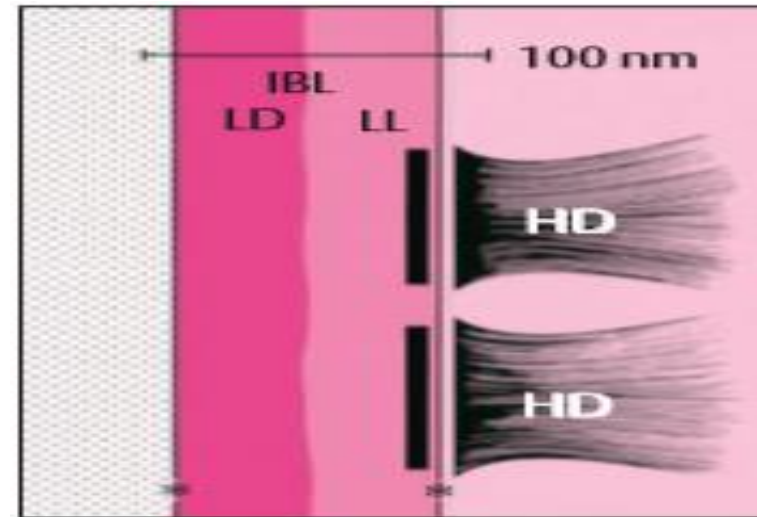
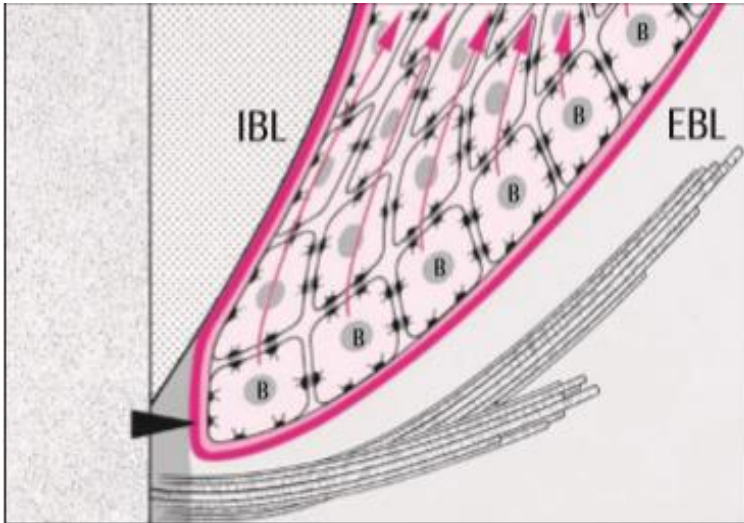
- 1- The basal layer
- 2- the supra-basal layer

It is continuously renewed through cell division in the basal layer and the cells migrate coronally to the base of the gingival sulcus, where the cells can shed (cell turn over).



JE is attached to the tooth surface by means of an internal basal lamina and hemidesmosomes, whereas it is attached to the gingival connective tissue by an external basal lamina and hemidesmosomes

- Healthy JE demonstrates no rete pegs where connects to the connective tissue



- JE has a prime role in the maintenance of periodontal health, it comprises the firm epithelial attachment ,that connects the soft tissue to the tooth surface.
- JE is quite permeable and thus serves as a pathway for diffusion of bacterial plaque products into connective tissue.
- An opposite movement is also found towards the sulcus of the host defence substances, which help to mount an immune response

## The differences between the JE and sulcular and oral epithelium

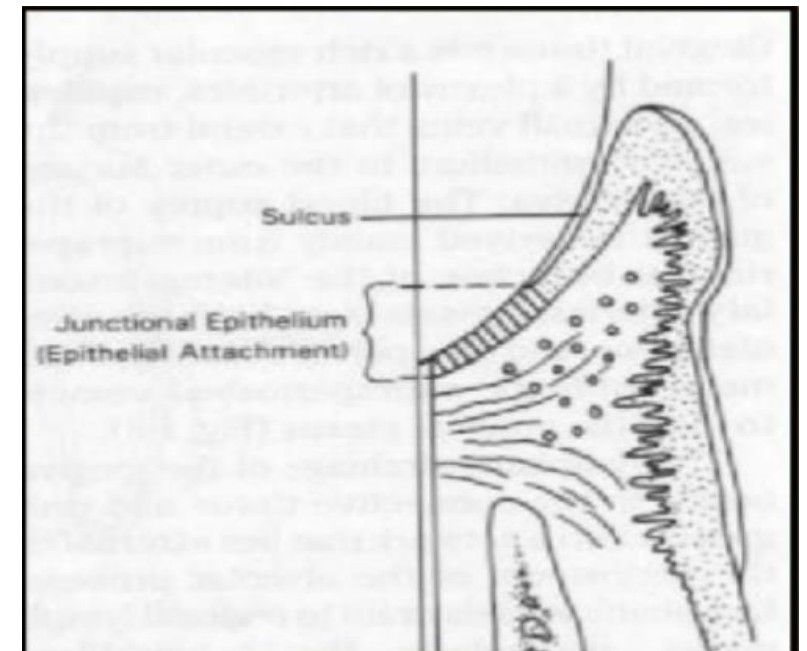
- 1- The size of the cells of JE is relatively larger than oral epithelium
- 2- The intercellular space of JE is wider than in the oral epithelium ( the intercellular space is preferred route for tissue fluids and inflammatory cells to migrate from the connective tissue to the gingival sulcus)
- 3- Desmosome's number is fewer in JE than in the oral epithelium, which may explain JE susceptibility to tear during probing in addition, the permeability to migrate cells and fluids
- 4- The sulcular and JE are not as thick as the oral epithelium, because both are not keratinised and have no rete pegs in health conditions
- 5- The turn over rate of JE is very high (4-6 days) compared to oral epithelium, which has the longest turn over ( 6-12 and up to 40 days)
- 6- JE forms the attachment of the gingiva to the tooth surface, whereas oral sulcular epithelium have no attachment to the tooth surface

## Gingival crevicular fluid (GCF)

- GCF is continuously secreted from gingival connective tissue into the gingival sulcus through the sulcular epithelium
- In purely normal gingival condition, little or no fluid can be collected but increase in GCF flow is a first sign of inflammation
- GCF contains a variety of enzymes, cells, electrolytes, proteins and antibodies

## The functions of GCF

- 1- Mechanical cleaning of the sulcus
- 2- Antimicrobial properties
- 3- Possess immune antibodies that enhance resistance of the gingiva to the inflammation
- 4- Contain plasma proteins which may improve the adhesion of the epithelium to the tooth surfaces



## Gingival connective tissue (CT)

It is known as lamina propria and consists of 2 layers:

1- The papillary layer: consists of papillary projections invaginated in epithelial rete pegs

2- The reticular layer: is continuous with the periosteum of the alveolar bone

The major components of the CT are:

1- Collagen fibers 60%

2- Cells 5%

3- Ground substance, nerves, blood and lymphatic vessels

## Gingival fibers

1- Collagen fibers are the most predominant type of fibers in the gingival CT

2- Oxytalan fibers

3- Elastic fibers

## Arrangement of gingival fibers

The supra alveolar crest fibers are arranged in groups of bundles according to their insertion and orientation in the tissue

**1- Circular fibers:** pass through the CT of the marginal and interdental gingiva and encircle the tooth as a ring like fashion

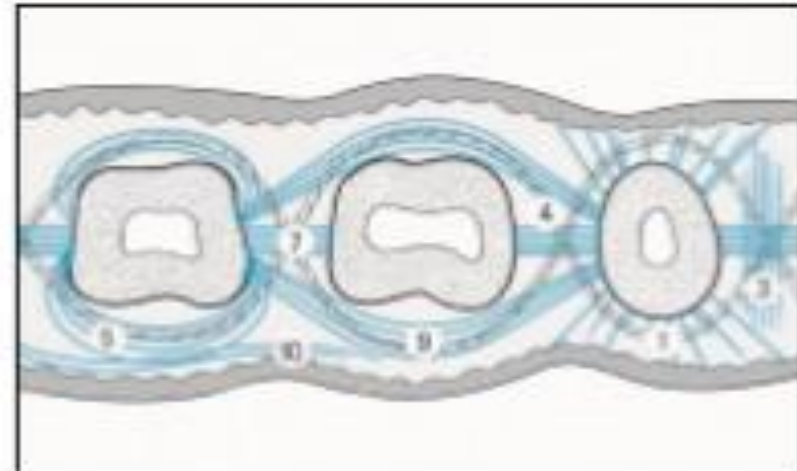
**2- Dentogingival fibers:** project from the cementum in a fan shape fashion towards the free gingiva

**3- Dentoperiosteal fibers:** extend from the cementum in an apical direction to the periosteum of the alveolar bone and terminate in the attached gingiva

**4- Trans-septal fibers:** located interproximal, arranged in horizontal bundles that extend between the cementum of approximating teeth into which they are embedded

Course of the gingival fiber bundles (see also Fig. 21)

- 1 Dentogingival
  - Coronal
  - Horizontal
  - Apical
- 2 Alveologingival
- 3 Interpapillary
- 4 Transgingival
- 5 Circular, semicircular
- 6 Dentoperiosteal
- 7 Transseptal
- 8 Periosteogingival
- 9 Interdicular
- 10 Interstitial





## Functions of the gingival fibers

- 1- To brace of the gingival margin firmly against the tooth surface
- 2- To provide the rigidity to withstand the force of mastication without being deflected away from the tooth surface
- 3- To unite the free gingival margin with the root cementum and the adjacent attached gingiva

## Cellular elements of the gingival CT

```
graph TD; A[Cellular elements of the gingival CT] --> B[Fibroblasts]; A --> C[Mast cells]; A --> D[Macrophages]; A --> E[Inflammatory cells];
```

### Fibroblasts

- The most predominant cells (65%)
- Synthesise collagen, elastic fibers and CT matrix
- Regulate collagen degradation

### Mast cells

- Responsible for production of some of matrix components
- Produce vasoactive substances, which may control the flow of blood through the tissue

### Macrophages

- Demonstrated phagocytic function
- Involved in the defence of the tissue against irritating substances

### Inflammatory cells

- Polymorphonuclear leukocytes (PMNL)
- Lymphocytes
- Plasma cells
- All have different immunological functions



## Matrix of the CT (ground substance)

It fills the space between fibers and cells and has a high water contents. The matrix is first produced by fibroblasts although some constituents are produced by mast cells and some derived from blood.

- It is considered as a medium in which the CT are embedded and it is essential for its normal function
- Thus the transportation of water, electrolytes, nutrients and metabolites to and from the CT cells occurs within this matrix
- The main constituents of it are **proteoglycans** and **glycoproteins**

## Blood supply of the gingiva

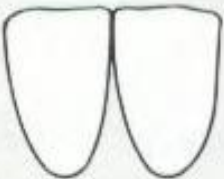



Gingival tissue has rich vascular supply which arise from the terminal branches of the internal maxillary artery

Gingival blood supply consists of

- Supra periosteal vessels
- Vessels come from PDL and alveolar bone
- These blood vessels coalesce ( merge) in the gingival papilla as a gingival plexus

## Innervation of the gingiva

- Is derived from the terminal branches of the maxillary and mandibular branches of the trigeminal nerve

|                   |  |  |
|-------------------|--|--|
| Score<br><b>0</b> | No plaque  |   |
| <b>1</b>          | Thin film of plaque at the gingival margin, visible only when scraped with an explorer                                   |   |
| <b>2</b>          | Moderate amount of plaque along the gingival margin; interdental space free of plaque; plaque visible with the naked eye |   |
| <b>3</b>          | Heavy plaque accumulation at the gingival margin; interdental space filled with plaque                                   |  |

|                   |  |
|-------------------|--|
| Grade<br><b>0</b> | normal gingiva, no inflammation,<br>no discoloration, no bleeding  |
| <b>1</b>          | mild inflammation, slight color change,<br>mild alteration of gingival surface,<br>no bleeding                   |
| <b>2</b>          | moderate inflammation, erythema, swelling,<br>bleeding on probing<br>or when pressure applied                    |
| <b>3</b>          | severe inflammation, severe erythema and<br>swelling, tendency toward spontaneous<br>hemorrhage, some ulceration |

### 59 Gingival Index (GI)

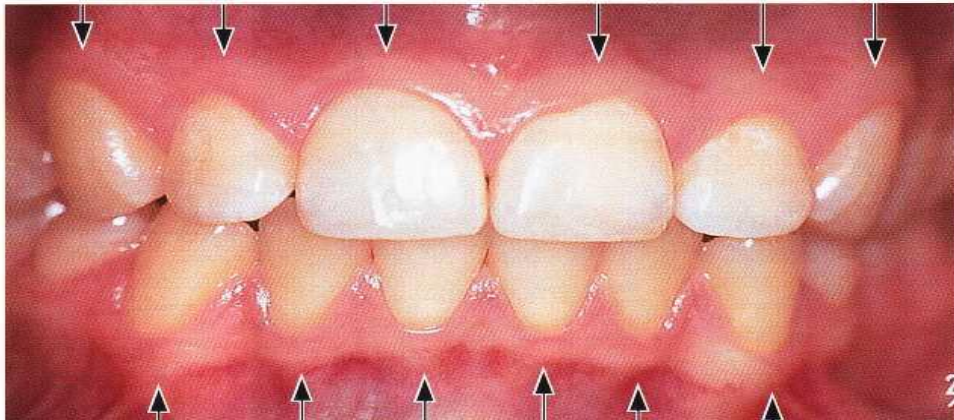
This index is used worldwide in epidemiological studies and scientific investigations. The GI scores gingival inflammation on the facial, lingual and mesial surfaces of all teeth. The symptom of bleeding comprises a score of 2.

The GI is recommended for epidemiological studies. It is less applicable for individual patients because the differences between the scoring levels are too gross.

## Histology of the oral mucosa

**Oral mucosa:** includes the tissue which lining the mouth. It consists of:

**1- Masticatory mucosa:** encompassing the attached gingiva in addition the one that cover the hard palate. Its boundaries are from the free gingival margin to the mucogingival junction (MGJ) on facial and lingual surfaces. This tissue is firmly attached to the underlying bone and covered with keratinized epithelium to withstand the frictional forces of food during mastication.

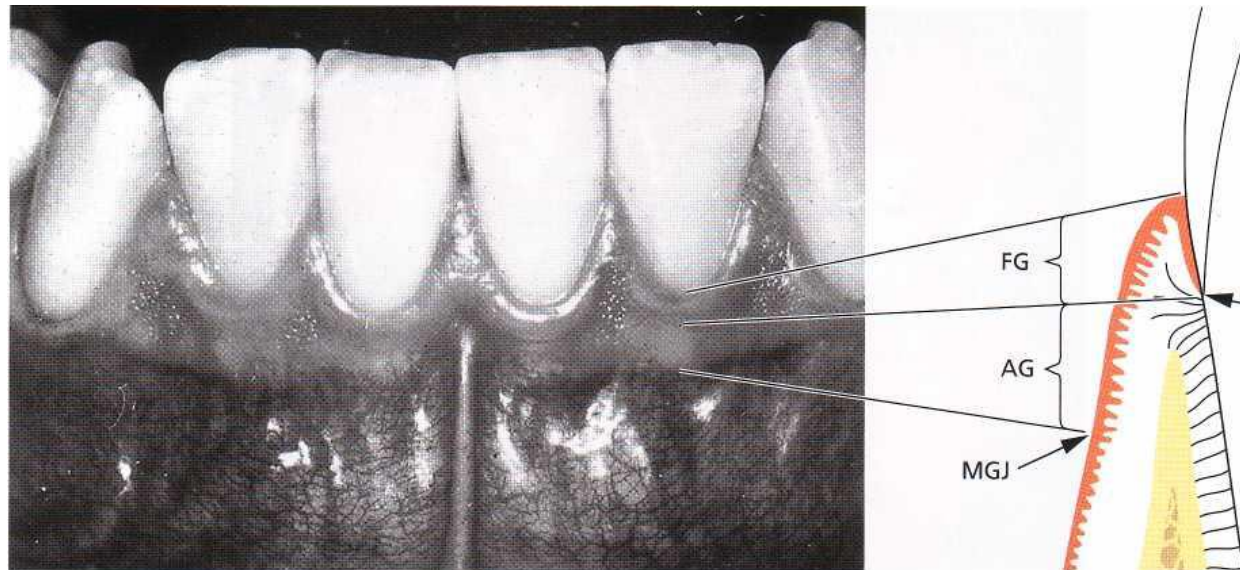




2- **Specialised mucosa**: covers the dorsum side of the tongue.

3- **Lining mucosa**: It is loosely attached to the underlying bone and covered by non- keratinised epithelium. The tissues that cover Lips, cheeks, floor of the mouth, inferior surface of the tongue, soft palate and alveolar mucosa ( located apical to the attached gingiva, extending to the mouth vestibule) are examples of the lining mucosa.

- Alveolar mucosa is darker red and moveable due to containing high number of elastic fibres



## Gingiva

Is the part of the masticatory mucosa, covering the alveolar process and surrounding the cervical portion of the teeth. It is divided into 3 parts:

- 1- Marginal gingiva (free or unattached gingiva)
- 2- Attached gingiva
- 3- Interdental gingiva

### 1- Marginal gingiva

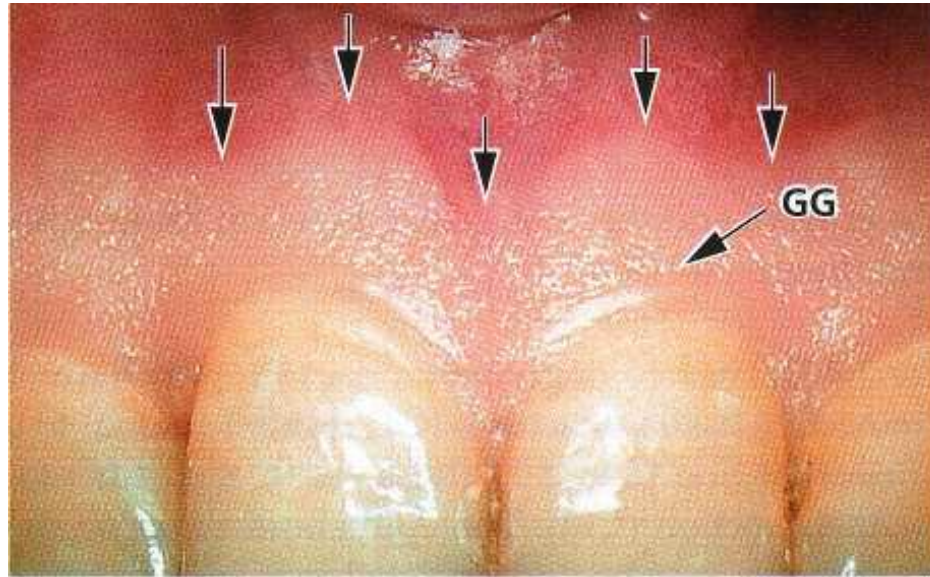
Is the most coronal portion of the gingiva, surrounding the teeth in a collar like fashion but not attached to them. It is demarcated apically from the attached gingiva by the free gingival groove.

- Free gingival groove: a shallow linear depression of about 1mm width and is positioned at a level corresponding to the cemento-enamel junction, it is present only in about 30-40% of adults.
- Free gingiva forms the soft tissue wall of the gingival sulcus.
- Gingival sulcus: is the space bounded by the free gingival margin, the tooth and the most coronal attachment of the junctional epithelium. Its range of healthy measurement is 1-3mm, however more than this measurement is considered as a pathological pocket.



## 2- Attached gingiva

It extends coronally from the free gingiva by the free gingival groove to the mucogingival junction in an apical direction. It is firm, resilient and tightly bound to the underlying teeth and periosteum of the alveolar bone. The stippling surface of gingiva, which is similar to the orange surface found in 40% of adults.



The width of the attached gingiva varies in different area of the mouth.

On the facial surface of the mouth it is:

- Widest on the maxillary lateral incisor
- Narrowest on the mandibular canines and first premolar

However on the lingual surface it is:

- Widest near the first and second molars
- Narrowest adjacent to the incisors and canines



### 3- Interdental gingiva:

It is located in the interproximal space beneath the area of teeth contact. It is triangular in shape regarding the mesio-distal aspect

The shape of the interdental papilla is determined by:

- 1- The contact relationships between teeth
- 2- The width of the approximal tooth surfaces
- 3- The course of the cement-enamel junction

Generally there are 2 shapes of the interdental papilla:

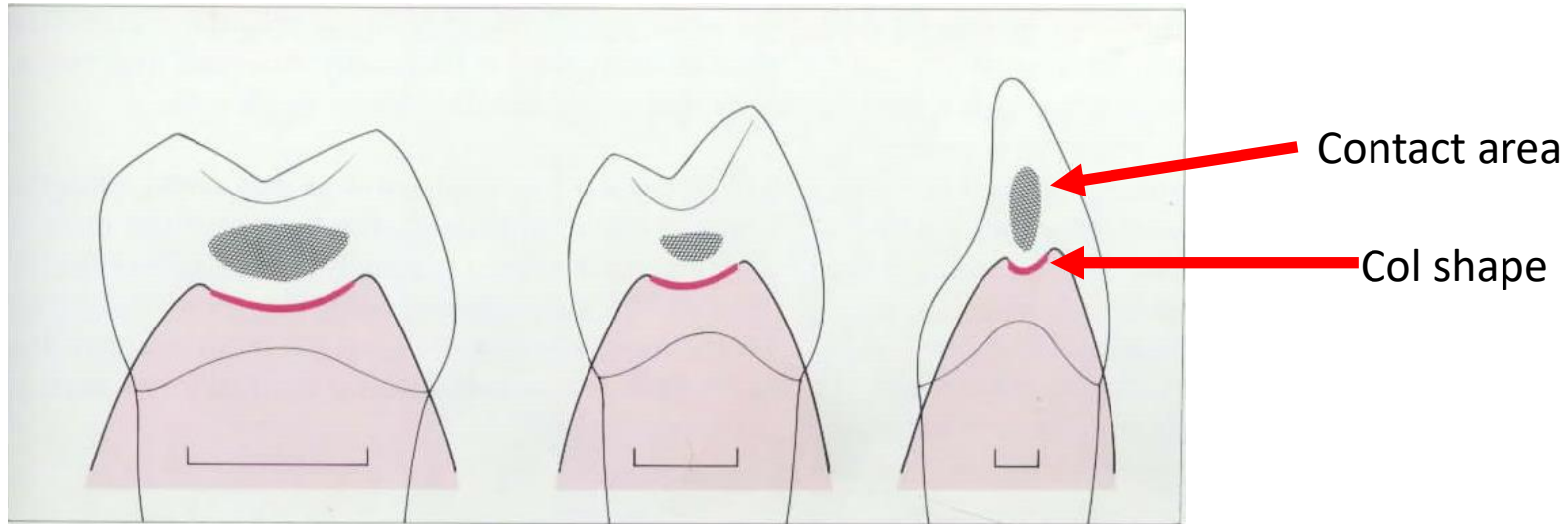
#### 1- Pyramidal shape:

Occur in the anterior region of the dentition where there is approximal contact point between 2 neighbouring teeth and one papilla with its tip immediately beneath the contact point

## 2-Col shape

The interdental papilla between the posterior teeth are more flattened, having a concave depression that connects the buccal (facial) and lingual papilla, taking the shape of the interproximal contact surface.

- In case of gingival recession, no Col shape will be seen
- Col area covered by non-keratinised epithelium, which is most susceptible for periodontal disease process



## Clinical descriptive criteria of health and inflamed gingiva

### 1- Gingival colour:

Coral pink is the normal colour of the gingiva, with some variations depending on the amount of melanin pigment in the tissues (dark skinned people often exhibit dark blue or brown colour), thickness of the epithelium, the degree of keratinisation and the vascularity of the connective tissue.

However, the inflamed gingiva may appear red to bluish red as a result of vasodilation, which may lead to bleeding tendency.

### 2. Gingival contour

The gingiva usually ends coronally in a knife edged margins and scalloped in contour.

Inflamed gingiva shows rounded and enlarged contours due to vascular stagnation and increases formation of collagen fibres.

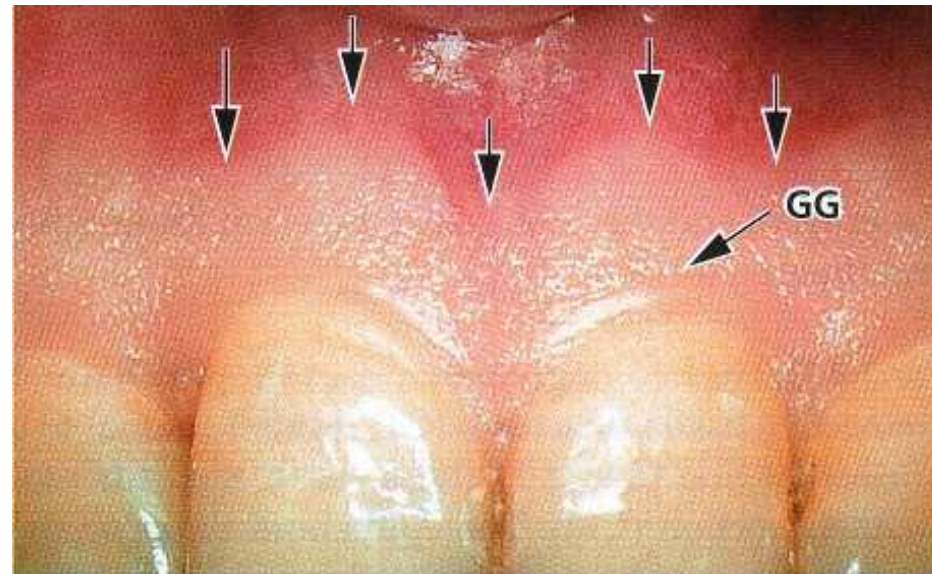
### 3- Gingival consistency

The gingiva is usually resilient, firm and bound to the underlying bone by the dense collagenous nature of the gingival connective tissue.

On the contrary, in inflamed gingiva, the consistency may be soft owing to the vascular stagnation and decrease in the amount of gingival collagen fibers or extremely firm due to excessive formation of collagen (fibrosis) as in chronic inflammation.

### 4- Gingival surface texture

The attached gingiva has usually stippled surface, whereas, the free gingiva is smooth.

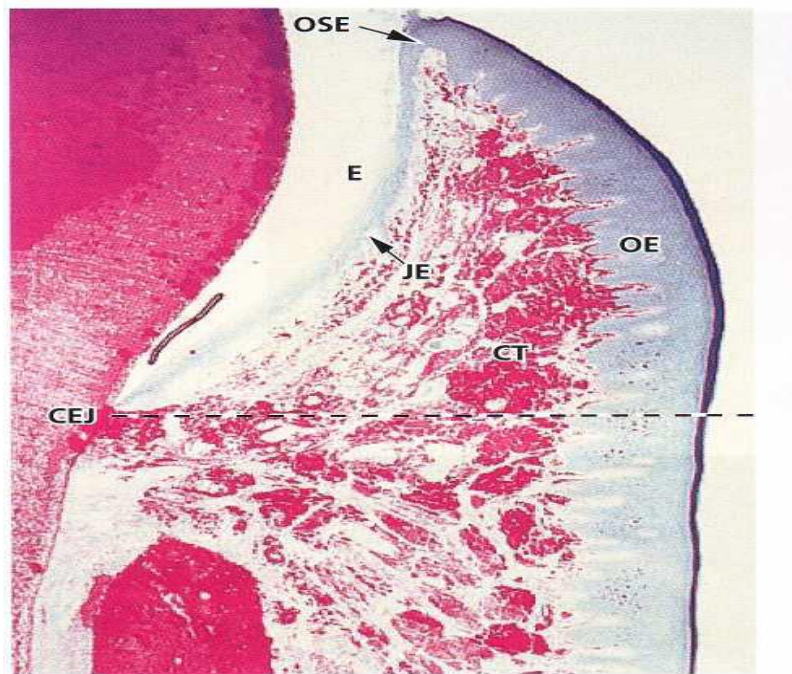


## Normal microscopic features

Gingiva consists of fibrous connective tissue known as **lamina propria**, covered by stratified squamous epithelium.

Gingival epithelium is described as follows:

- 1- Oral epithelium faces the oral cavity
- 2- Sulcular epithelium faces the tooth in the gingival sulcus only
- 3- Junctional epithelium provides the contact between the gingiva and the tooth

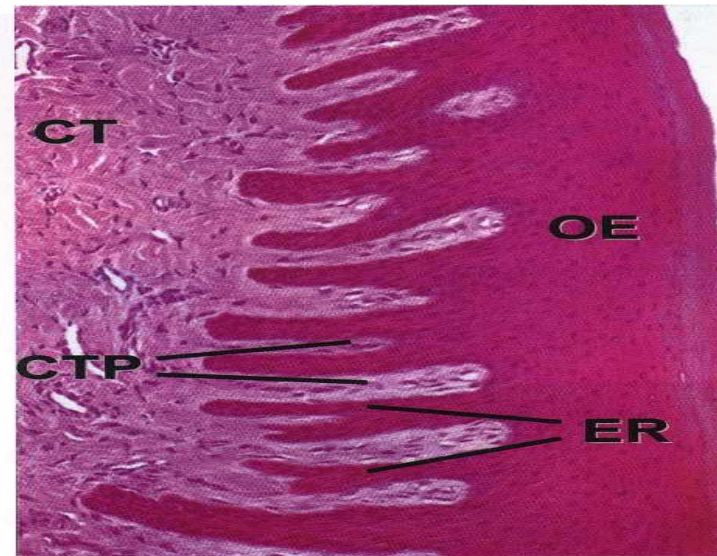
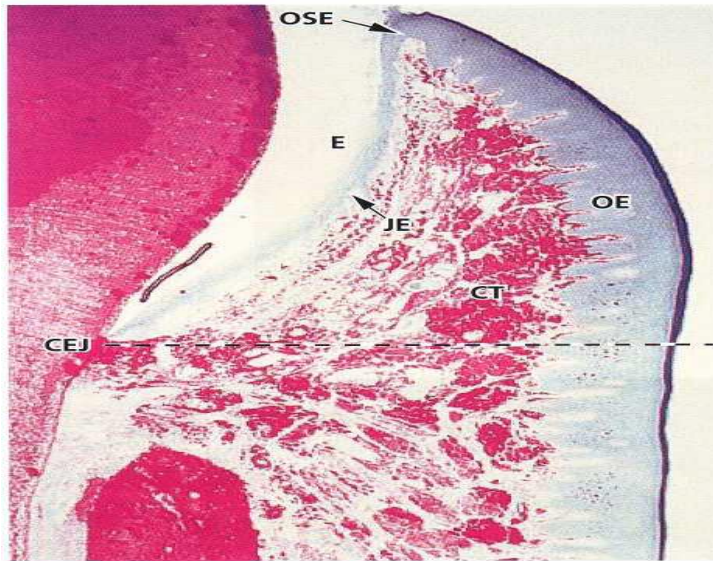




## Oral epithelium

It covers the crest and the outer surface of the marginal and attached gingiva. It is either keratinised ( without nuclei) or parakeratinised (retained nuclei).

- The boundaries between the oral epithelium and the underlying connective tissue has a wavy course, known as (Rete pegs or rete ridges).
- The intervening connective tissue portions, projecting into the epithelium are called connective tissue papilla
- This alternating pattern of depression and protuberances of the connective tissue papillae with epithelial rete pegs is supposed to give the stippled appearance



## Layers of oral epithelium

### Stratum basale (basal layer)

- Basal layer of cuboidal cells along the basement membrane
- Here the epithelial cell replication and differentiation begin
- Melanocytes reside in this layer

### Stratum spinosum (prickle cell layer)

- The thickest layer
- Cells appear to have cytoplasmic spines
- It contains Langerhans cells

### Stratum granulosum (granular cell layer)

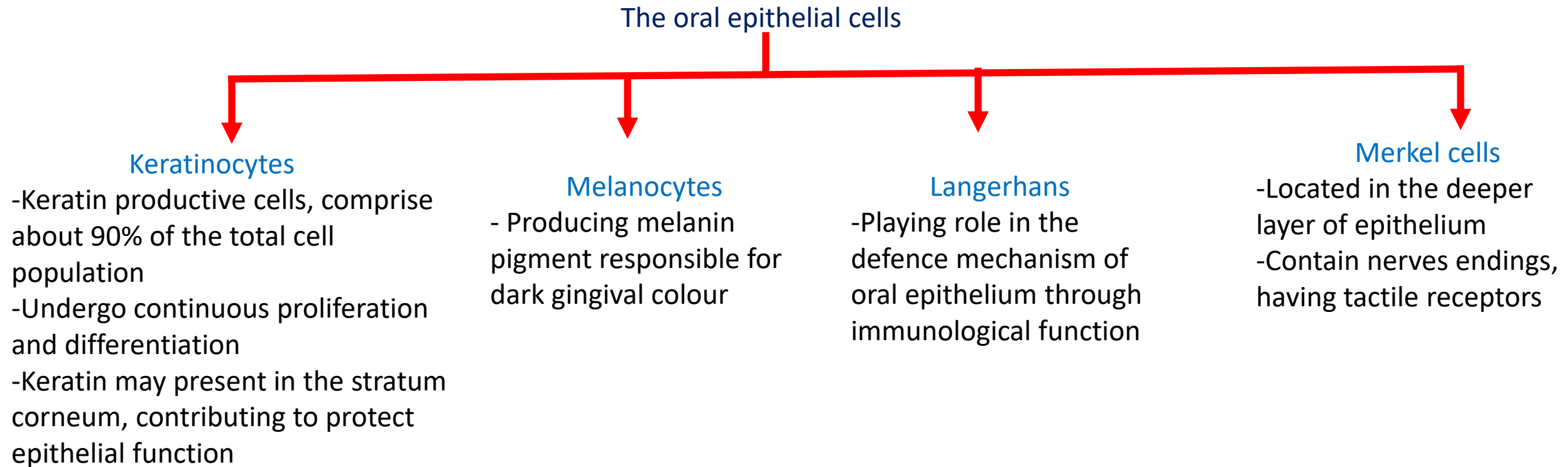
- Cells appear to be flattened
- Keratohyalin granules may be seen here

### Stratum corneum (keratinised cell layer)

- Keratinisation occur in this layer
- more superficial layer



The epithelial cells that forming the basal layer, gradually undergo keratinisation process. This process is achieved by cell proliferation and differentiation ( change in their characterisations) and migrate towards the surface layer



Under normal conditions there is a homeostasis (equilibrium) between cell renewal and desquamation (cell turn over). It takes approximately 3-4 weeks for keratinocytes to migrate from basal cell layer until reach the outer epithelial surface.

- The basal cells are found immediately adjacent to the connective tissue and separated from this tissue by a basement membrane (basal lamina).



The basement membrane is consisted of:

1- Lamina Lucida: is located immediately beneath the basal cell layer.

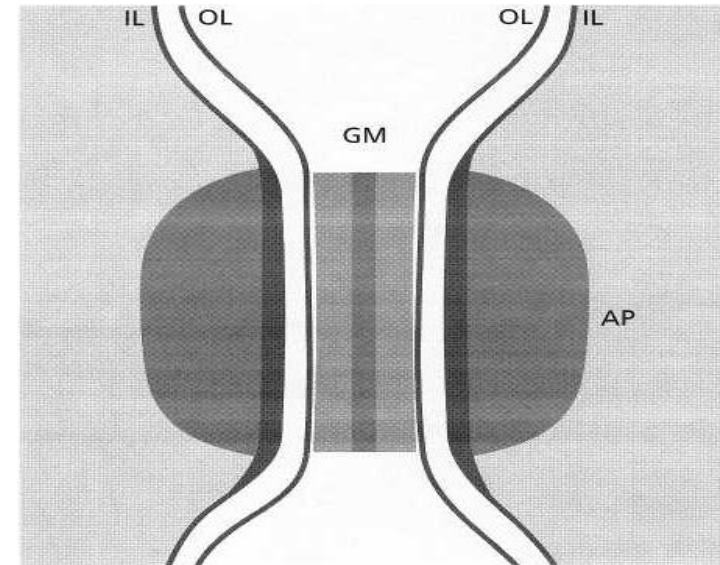
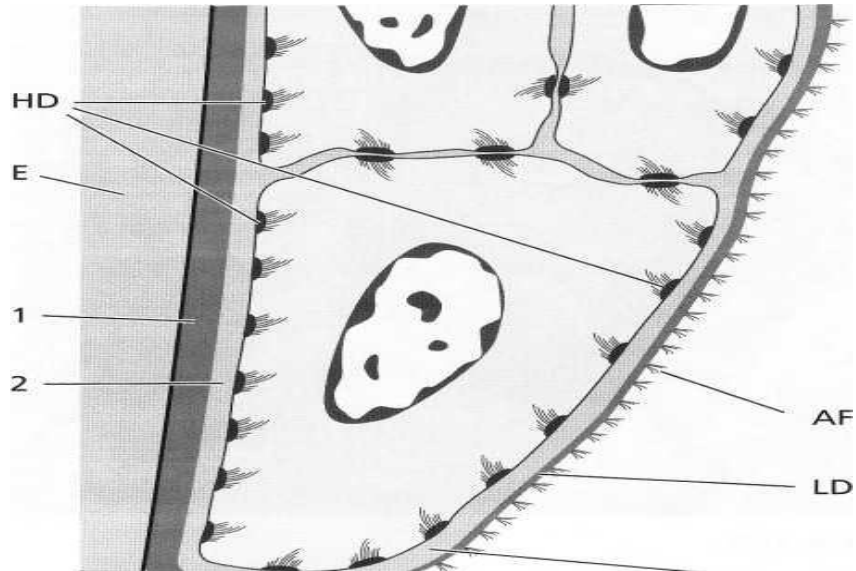
2- Lamina Densa: is located beneath the lamina lucida. The anchoring fibers project from it towards the connective tissue.

-The epithelial cells are joined together by specific structure called desmosomes, which is composed of two **hemidesmosomes** separated from each other by granulated material.



The **hemidesmosome** is composed of :

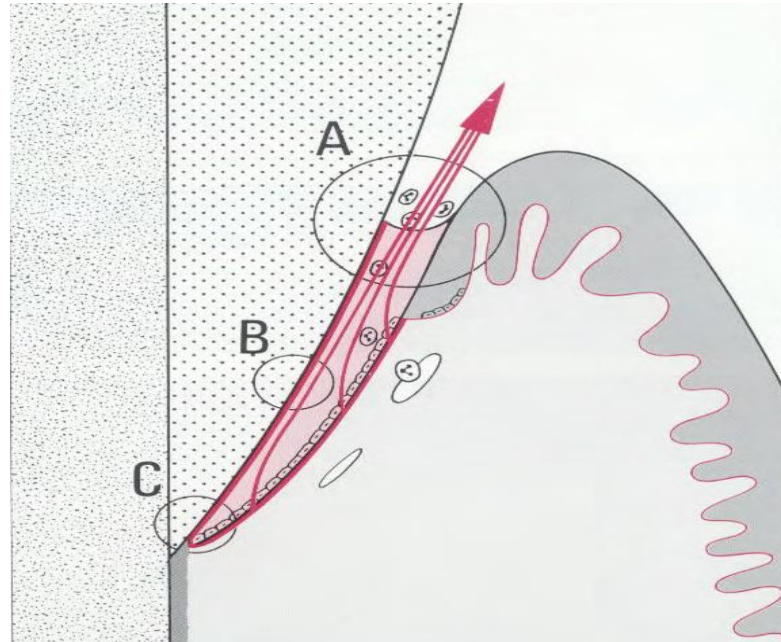
- 1- The outer leaflets (OL) of cell membrane of two adjoining cells.
- 2- The inner leaflet (IL) which is thicker leaflet of cell membrane
- 3- The attachment plaque which represent granular and fibrillar material in the cytoplasm



## Sulcular epithelium

It lines the gingival sulcus, has a thin, non-keratinised stratified squamous epithelium without rete pegs. It extends from coronal limit of the junctional epithelium to the crest of the gingival margin.

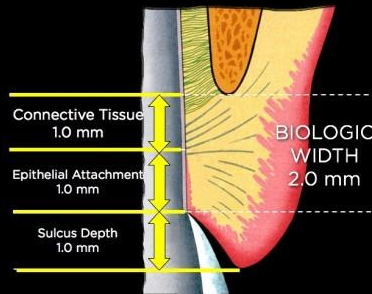
- The importance of sulcular epithelium is coming from its thin consistency and may act as a semipermeable membrane through which the fluid can seep from the gingiva into the sulcus, make it easier for bacterial products of dental plaque to penetrate into the connective tissue, stimulating inflammation and tissue destruction
- So it is considered as a poor barrier against bacterial infection.



## Biological width

- Described as combined heights of the connective tissue and junctional epithelial attachment to the tooth.
- The junctional epithelium and the connective tissue attachment have an average height of 1mm each.
- So the biological width is 2mm
- There are variations from 0.75-4mm
- The clinical significance of biological width is the relative importance to the position of restorative margins and post surgical tissue position.
- If the restorative margin is placed too deep below the tissue, it will invade the biological width and two possible outcomes might occur; gingival inflammation and bone resorption

Classic illustration of the average human attachment apparatus as described by Gargiulo, Wentz, and Orban in 1961



The Connective tissue attachment and epithelial attachment form the "Biologic Width" averaging 2mm in most patients

A patient who presented with porcelain bonded crowns placed 6 months earlier, she is unhappy with their appearance, and the severe gingival inflammation, the margins are all within 1mm of bone



**Instruments used for scaling & root planing are classified as:**

- ❖ Hand instruments.
- ❖ Ultrasonic & sonic instruments.
- ❖ Motor driven devices incorporating diamond coated tips.
- ❖ Rotating instruments.
- ❖ Laser instruments.

**Hand instruments:** A hand instrument is composed of three parts:

- The working part (the blade) which is often made of carbon steel, stainless or tungsten carbide.
- The shank.
- The handle.

**Curettes:** are fine instruments used for sub gingival S&RP thus designed to adapt to the root surfaces and good access to deep pockets without causing trauma to the soft tissue. When curette is used it should be hold in modified pen grasp with finger rest support, the curette is gently inserted into the pocket with its distal end facing the soft tissue, when the instrument reach the base of the pocket, it turned to cutting position and moved coronally with a pulling stroke (scaling stroke), that followed by moderate to light pulling stroke (finishing stroke) to produce a smooth surface. Curette have spoon shaped blade with a rounded tip. There are 2 types of curettes

1. Universal curettes: have 2 cutting edges that afford access to all areas and root surfaces by altering their position and angulation.
2. Area specific curettes: a set of many curettes with only one cutting edge are designed to adapt to specific areas of the dentition and instrument specific root surfaces (e.g. Gracey curettes).

**Sickles:** the sickle is manufactured with either a curved or a straight blade which has triangular cross section and 2 cutting edges that converge into a sharp tip. They should not use subgingivally as they will traumatize the gingiva.

**Hoes:** has only one cutting edge. The blade is turned at a 100 angle to the shank with the cutting edge beveled at a 45 angle. The blade can be positioned at 4 different inclinations in relation to the shank: facial, lingual, distal and mesial. Two of them used for anterior and two for posterior teeth. The hoes are used for subgingival scaling by insertion the blade to the base of the periodontal pocket then a firm pull stroke toward the crown is activated.



**Chisel:** is designed for the proximal surfaces of the spaced anterior teeth. The blades are slightly curved and have a straight cutting edge beveled at 45°. The chisel is inserted from the facial surfaces and activated with a push motion.

**Cumine:** is used for the removal of supragingival calculus with pulling action, and manufactured with two ends; spoon shape end for labial / buccal and lingual / palatal surfaces and sickle shape end for interproximal surfaces.

### **Ultrasonic scalers:**

Ultrasonic scalers convert electrical current to mechanical energy into the form of high frequency vibrations at the instrument tip (the vibration frequencies ranging from 18000\_45000 HZ) that lead to fracture & dislodge the calculus.

**Sonic scalers:** use air pressure to create mechanical vibration of the instrument tip (frequencies of vibration ranging from 2000-6000 HZ); thus it is considerably slower than ultrasonic scalers. Studies demonstrated that scaling with hand & ultrasonic instruments are equally effective.

Ultrasonic scalers are, contraindicated in

1. Individuals with infectious disease due to the risk of airborne infection.
2. Individuals with cardiac pacemaker & hearing aids.
3. Individuals with strong gag reflex.
4. Young children.
5. Individuals who experience pain on use.

### **The advantages of ultrasonic scalers over hand instruments:**

1. requires less effort, less pressure, less trauma & less time; thus, it is quicker, simple & better for the patient's comfort.
2. stain removal easier.
3. water spray cleans the area of loose debris.

### **Disadvantages of ultrasonic instruments:**

1. Production of heat, thus requires coolant water spray.
2. Good suction is required.
3. Loss of visibility due to coolant spray.
4. Water spray droplets will contain microorganisms from the mouth & these droplets remain in the air for some time which leads to the risk of airborne infections.
5. Loss of tactile sensation that may produce uneven root surfaces.
6. Interfere with cardiac pacemaker and hearing aids.

7. Damage of restoration (porcelain).
8. Enamel abrasion.
9. Pain and patient discomfort.

**Reciprocating instruments:** a special designed hand piece will give 20000\_30000 strokes per min. with a 1.2mm reciprocating motion of a specially designed working tips for S & RP, less time consuming than hand instrument, less root surfaces loss and produce equivalent clinical outcome compared to hand, sonic or ultrasonic scalers.

**Rotating instruments:** used to debride root furrows, furcation areas and root surfaces in deep narrow pockets because in these situations cannot be properly debride with hand inst. a fine grained diamond bur is usually used with great care to avoid excessive removal of tooth substances.

**Laser:** recently laser devices been introduced to be used in different aspects of periodontal therapy including S & RP.

**Polishing:** technique used to remove plaque, pellicle, materia alba, exogenous stain and to produce smooth surface thus reduce surface roughness, by using rubber cup, brushes on a slow speed hand piece, dental tape, air powder abrasive system and prophylaxis pastes that contain fluoride should be used; and kept moist to minimize friction heat.

### **Evaluation of the effect of the initial, cause-related therapy:**

Reevaluation of the patient's periodontal conditions & caries activity should be performed no earlier than 6-8 weeks following the last session of the S&RP procedures, in order to provide time for the tissues to heal by the formation of a long junctional epithelium & sufficient practice with oral hygiene skills.

Clinical attachment level (CAL); is the distance from the cemento-enamel junction (CEJ) to the location of inserted periodontal probe tip (bottom of gingival crevice or periodontal pocket).

When we evaluate the results of our treatment, we can see one of the following conditions:

1. Patient with improved oral hygiene, no gingival inflammation, no bleeding on probing with marked reduction in probing pocket depth, in such situation no further periodontal treatment is required and the patient directly advanced to maintenance phase of periodontal therapy.
2. Patient with proper standard of oral hygiene but having some sites of bleeding on probing with no significant reduction in probing depth. Such patient may need to be advanced to corrective phase including the periodontal surgery.

3. Patient with inadequate oral hygiene due to lack of motivation or lack of ability to do proper home care, such patient should be remotivated and reinstructed to improve their oral hygiene because if the oral hygiene not improved the periodontal disease will recurrent even if we conduct periodontal surgery.

**Periodontal instruments are classified according to the purposes they serve as follows:**

1. Periodontal probes are used to locate ,measure and mark pockets as well as determine their course on individual tooth surfaces,usually long, thin,and blunted at the end
2. Explorer are used to locate calculus deposits and caries.

**Periodontal probes:**

- a. marquis colour couded probe.
- b. Unc\_15 probe.
- c. University of Michigan "o" probe.
- d. Michigan "o"probe with marking 3,6 and 8.
- e. W.H.O. probe.
- f. NABERS PROBE.

**Kinds of grasp:**

**1. Standerd pen grasp**

The side of the middle finger rests on the shank.

**2. Palm and thumb grasp:**

Used for stabilizing instruments during sharpening and manipulating air and water syringe,but not recommended for periodontal instrumentation.

**3. Modified pen grasp**

-most effective and stable grasp.

-the pad of the middle finger rests on the shank.

-produces tripod effect which

- a. enhances control.
- b. enhances tactile sensitivity.



## Lec. 12

# Interdental cleaning

د. نهی عکاب

1. removal food & plaque stagnation.
2. earliest area to be affected .
3. the tooth brush does not reach the interproximal spaces efficiently as they are difficult to access.



# Factors we need to consider when selecting the appropriate interdental cleaning method are :

- the contour & consistency of the gingival tissue .
- 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, flossing remove up to 80 % of proximal plaque .Even subgingival plaque can be removed since dental floss can be introduced 2\_3.5mm 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.
- 3.Afloss holder to facilitate flossing might be used.
- 4.tape: a type of broadened dental floss used for cleaning bridge pontics.
- 5.super floss used for patients with crowns ,bridges & orthodontic appliances.

# 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, they 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.



- 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, 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:

- For cleansing areas which cannot be reached without other devices. They are designed to improve access to distal surfaces of posterior molars, tipped & rotated teeth, around & under fixed appliances, pontic, orthodontic appliances and teeth affected by gingival recession & furcation involvement.





# Adjunctive aids:

- 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 inflammatory effect.with specially designed tips the fluid may penetrate deeply into the pocket.



# 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 & 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, 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, cause gingival erosion & gingival recession. Trauma to hard tissues lead 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 brushing force, and improper use of both manual and powered tooth brushing.



# Scaling & root planning:

- Scaling is the process by which plaque & calculus are removed from both supragingival & subgingival tooth surfaces.
- Root planing is the process by which residual embedded calculus and softened cementum are removed from the roots to produce smooth, hard & clean root surfaces.
- The supragingival scaling is the initial phase of debridement of the dentition in patient with periodontal disease in order to facilitate the subsequent subgingival scaling, so supragingival calculus & gross overhang or metal crown should be removed first, then the dentition are polished so that the patient can start self performed plaque control program.



- Subgingival S & Rp, although they are considered as two separated procedures with different objectives but in clinical work they are always carried out together & can be carried out in sessions, the number of teeth included in each session for RP depend on the skills of the operator and severity of case, usually (4-6)teeth.at the beginning the area is probed to identify:
- 1.probing pocket depth. 2.anatomy of the root . 3.location of the deposits.

# Scaling & root planning aims to :

- Restore the gingival health by the removal of bacterial plaque ,calculus & the superficial layer of cementum however ,calculus & plaque grow in surface irregularities of cementum furthermore,bacterial products (such as endotoxine) penetrate into the cementum surface.
- The creation of clean &hard root surfaces that is as smooth as possible (which inhibits further plaque retention) must be achieved to promote tissue healing possibly with the formation of a long junctional epithelium and aids soft tissue reattachment.

- **Sub gingival scaling & root planning** are performed as either closed or open procedure. The closed procedure implies subgingival instrumentation without displacement of the gingiva, thus less trauma, pain, bleeding and minimal recession (which is important for esthetics, especially anteriorly) were achieved. In addition, wound healing occurs more rapidly following closed procedures. Closed therapy is the definitive treatment for mild & moderate periodontitis. Closed therapy limitations include its performance without direct vision & good access for the instruments.

- its success is dependent on knowledge of root morphology. Even the experienced hygienist will not always effectively treat all root surfaces, nor completely remove all plaque & calculus from all surfaces, e.g. S & RP of poorly accessible, irregular root surfaces, in deep, narrow or distal pocket & substantial furcation involvement, even in patients with minimal mouth opening capacity & with expansively progressive disease.
- **Open procedure** calls for exposure of the affected root surface by displacement of the gingival tissue, thus gingiva is incised and reflected to facilitate access for the instrument and visibility for the operator.



Lec.17

د. نهی عکاب

# Periodontal Surgery



## ❖ Objectives of periodontal surgery:

1. Accessibility and direct vision for proper S & RP.
2. Reduction of plaque retentive area especially periodontal pockets that have not responded to initial therapy.
3. Eliminate inflamed periodontal tissues.
4. Enhancing the regeneration of periodontal tissues.
5. Create physiologic morphology of the dentogingival area that will facilitate efficient self performed plaque control.
6. Correct mucogingival defect and improve periodontal aesthetic.
7. Provide access to correct bony defects.

- **Gingivectomy:** This surgical procedure aimed at the excision of the soft tissue wall of pathologic periodontal pocket.

### **Indications:**

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



- **Contraindications:**

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 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.

- **Gingivectomy Procedure:**

- Giving local anesthesia, then marking the pocket depth by: 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 horizontal forceps tip to mark depth of pocket, and by repeating this procedure at each tooth surface, a series of bleeding points is created, which are used as a guide for incision. Primary beveled incision which carried out 1 mm apical to bleeding points by Kirkland knife. Secondary incision is made to separate interproximal soft tissues from interdental periodontium by Orban knife. Careful removal of the incised tissues by Curette or Cumine.

Plaque, calculus and granulation tissues is removed by using Curette to get smooth teeth surfaces. Placing gauze packs to control bleeding. Put dressing to cover the wound with pressure to prevent bleeding.



- **Flap Surgery:**

**Indication:**

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

- **Modified Widman flap Advantages:**

1. Good access to root surface to facilitate S & RP as well as removal of pocket epithelium and inflamed connective tissue.
2. Maintain the width of keratinized gingiva.
3. Replacement of flap at presurgical location leads to less exposure of root surface thus minimizes problem of aesthetic and root hypersensitivity.
4. Cause minimal amount of trauma to periodontal tissues and discomfort to the patient.

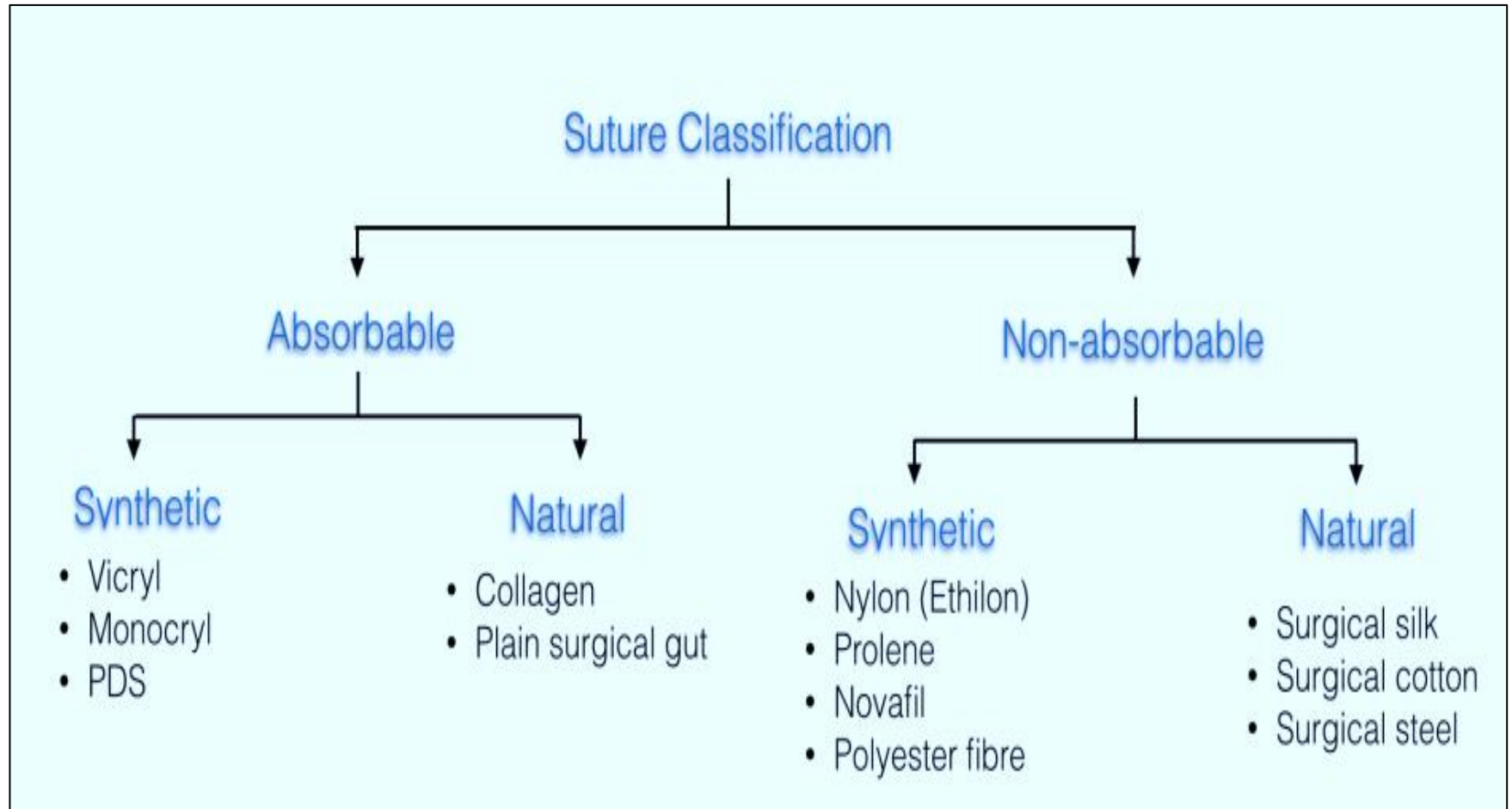
5. Possibility of obtaining close adaptation of soft tissues to root surfaces.
6. Provides better access to re-establish the contour of alveolar bone as well as the potential for bone regeneration in sites with angular bony defects.
7. Furcation areas can be exposed.

- **Procedure of Modified Widman flap:**

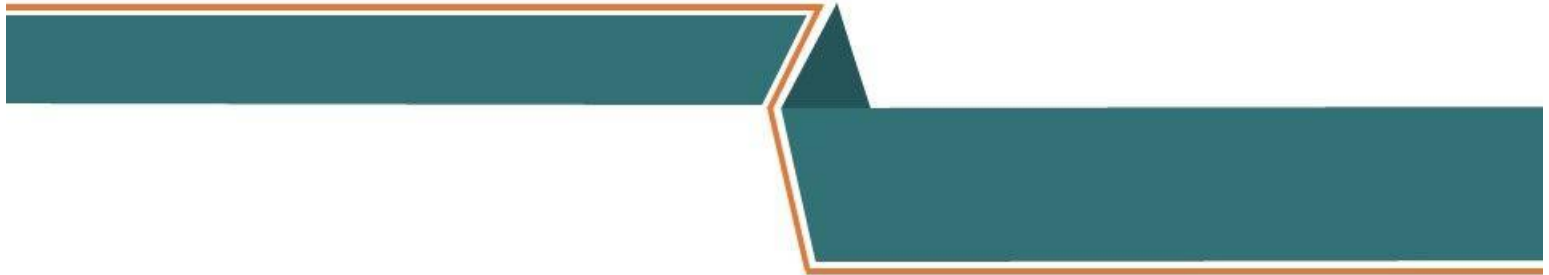
The initial gingival incision should be made with knife that can be directed parallel to the long axis of the tooth. The distance of the incision from the gingival margin vary from 0.5 to 2 mm. A second incision is made around the neck of each tooth from the bottom of pocket to the alveolar crest. Full thickness flap is elevated for only 1-2 mm from the alveolar crest as needed for access to the root surface and the interproximal one. The third incision is made along the alveolar crest thus separating the infiltrated tissue from healthy supporting connective tissue. Then, fine curettes are used to remove remnants of pocket

epithelium and granulation tissue, calculus necrotic cementum to obtain smooth, hard, clean surface. Root planning is performed with repeated rinsing. Afterwards, the flaps are closed using interrupted sutures.

# • Suture Classification:



**“Thank You”**





## Lec. 1

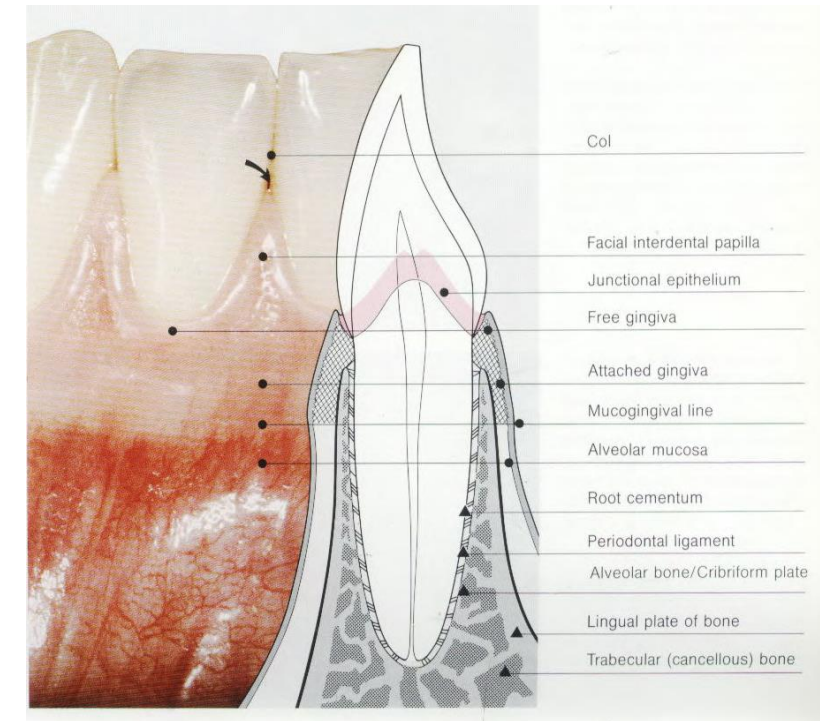
د. نهی عکاب

### What is Periodontology????

The clinical science that deals with teeth and their supporting structures (periodontium) in health and disease conditions.

Periodontium: is the tissue that surround and support teeth. It composed of :

1. Periodontal ligament (PDL).
2. Gingiva.
3. Cementum
4. Alveolar bone



Periodontics: is the branch of dentistry that specified to treat and prevent periodontal disease.

Periodontal disease: the pathological process involved the periodontium leading to gingivitis and periodontitis.

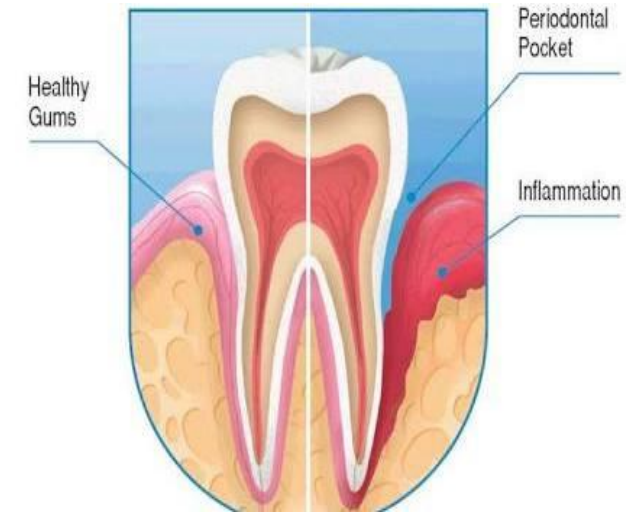
**Gingivitis:** a reversible inflammation of the gingiva, without loss of attachment of PDL, usually associated with erythema (redness), swelling and bleeding on probing.



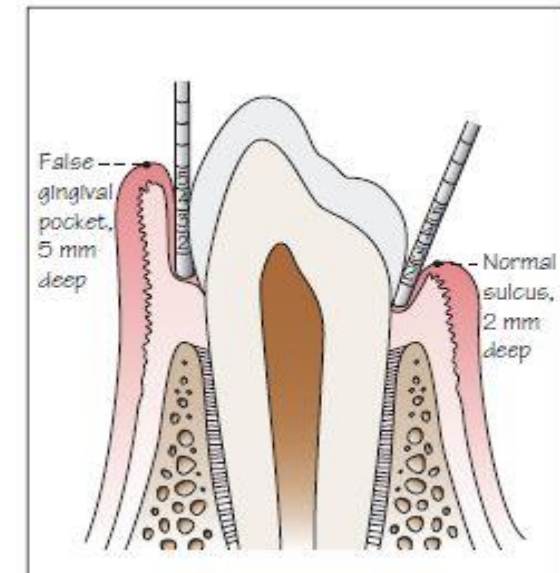
**Periodontitis:** Inflammation of the tooth supporting tissues, resulting in permanent destruction of the periodontium.



**True periodontal pocket:** a pathological migration of junctional epithelium from the cementoenamel junction causing deepened gingival sulcus due to loss of connective tissue attachment as a result of a progressing of periodontal disease



**Pseudo pocket (false pocket):** deepening in gingival sulcus due to gingival enlargement by certain pathological conditions, without migration of junctional epithelium or destruction of periodontal tissues.



**Periodontal ligament (PDL):** connective tissue connect the root to the alveolar bone.

It consists of:

1. Bundles of intermingling collagen fibres.
2. Cellular elements.
3. Ground substance.

It is worth mention that PDL and cementum develop from the **follicular sac**, derived from mesenchyme. PDL development occur during root formation and tooth eruption.

Components of PDL fibres:

**A. Principal fibres:** comprise the majority of PDL, consisting of collagen fibres, arranged in bundles in an S-shaped course.

- The development of principal fibres has been noticed as small, fine and brush like fibrils detected arising from the root cementum, projecting into the PDL side.
- At this stage, alveolar bone surface was covered by osteoblasts, with small number of radiating thin collagen fibrils.
- The number and thickness of fibres entering the bone increase and gradually become longer, whereas the fibres originating from the cementum are still short.
- These fibres are seen increased in length and thickness and fused with the that originated from the alveolar bone.
- Following tooth eruption, the principal fibres become organised in bundles, continuously connecting the root cementum to the alveolar bone. However, the tooth underwent active eruption thought to consist of two separated parts; one is located towards the cementum and the other towards the alveolar bone and connect together at the mid way through intermediate plexus.
- Sharpy's fibres are the part of principal fibres that insert into the cementum from one side and the alveolar bone from the other side.

Principal fibres are arranged in five groups:

**1- Alveolar crest fibres (ACF):** obliquely extend from the root cementum to the crest of alveolar bone in an apical direction. ACF prevent the extrusion of the tooth and resist lateral tooth movements.

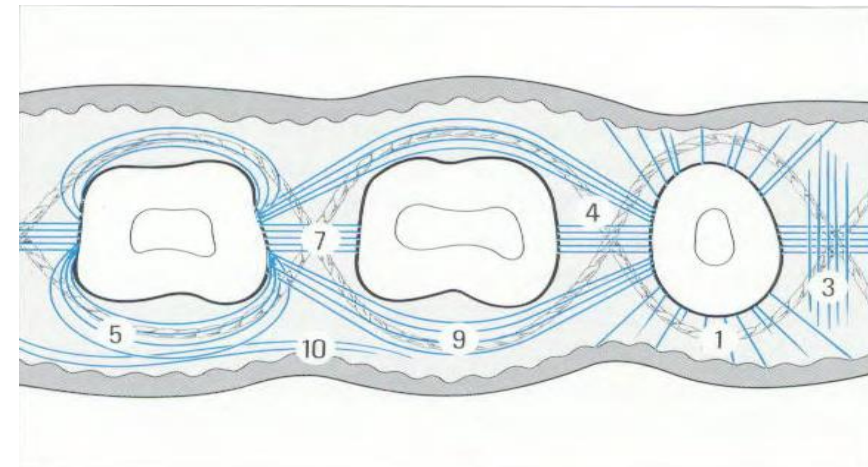
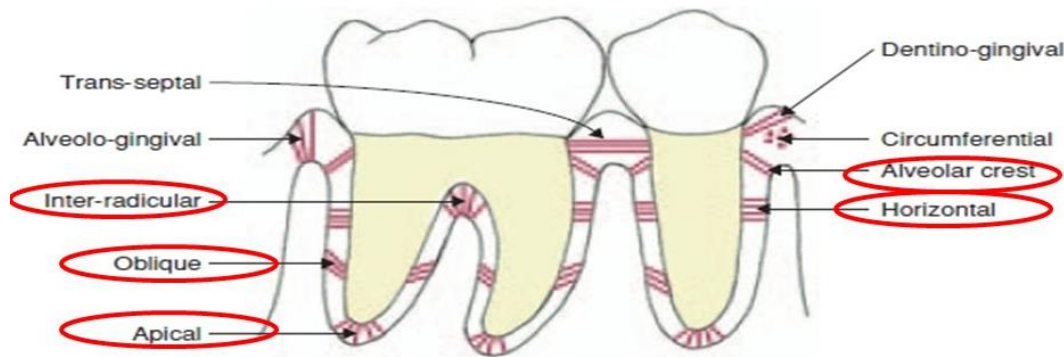


2- **Horizontal fibers (HF)**: extend in a right angle to the long axis of the tooth, running from the cementum to the alveolar bone.

3- **Oblique fibers (OF)**: are the largest group in the PDL, extending obliquely from the cementum in coronal direction to the alveolar bone. Its function is to withstand the masticatory force.

4- **Apical fibers (AF)**: radiate from the cementum to the alveolar bone at the apical region of the tooth.

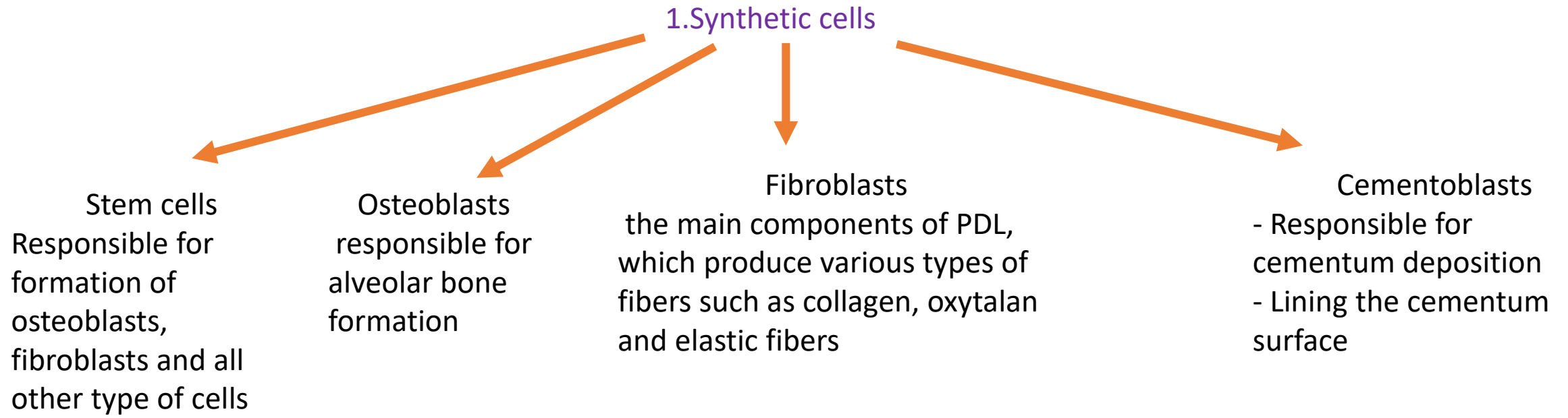
5- **Inter-radicular fibers (IF)**: connect the cementum to the alveolar bone at the furcation area of multirooted tooth.



B. **Elastic fibers**: are associated with blood vessels and are relatively few.

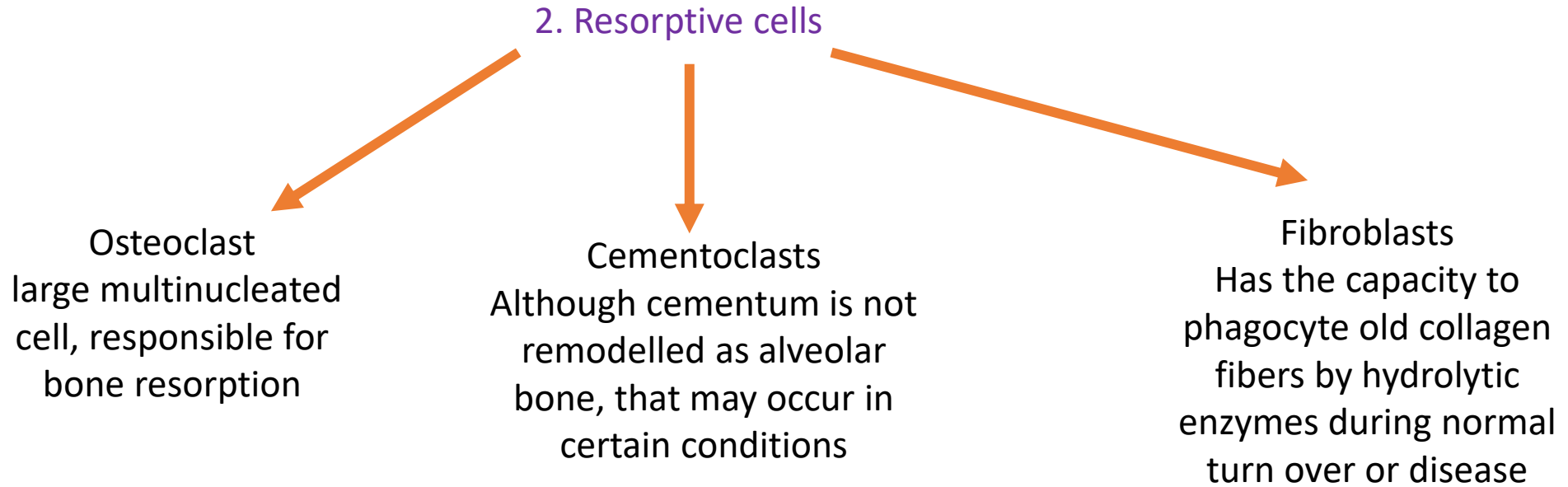
C. **Oxytalan fibers**: are immature forms of fibers which may regulate vascular flow.

## PDL include different cells:





## 2. Resorptive cells



```
graph TD; A[2. Resorptive cells] --> B[Osteoclast  
large multinucleated  
cell, responsible for  
bone resorption]; A --> C[Cementoclasts  
Although cementum is not  
remodelled as alveolar  
bone, that may occur in  
certain conditions]; A --> D[Fibroblasts  
Has the capacity to  
phagocyte old collagen  
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**3. Epithelial rests of malassez:** are found close to the cementum, which are remnants of Hertwig root sheath. They proliferate in response to a stimulus and participate in formation of periapical and lateral root cysts.

**4. Immune system cells:** Neutrophils, Mast cells (contain histamine which play an important role in inflammatory process), macrophages (phagocytosis) and lymphocytes.

**5. Cells associated with neurovascular elements**

## Ground substance of PDL:

Fills the PDL space between cells, fibers, blood vessels and nerves.

Containing 70% of water

The main 2 components of it are:

1. Glycosaminoglycans
2. Glycoproteins

## PDL width:

The width of PDL space varies in respect to age, location of tooth and the degree of stress to which the tooth is subjected.

- It found thinner on the mesial root surface than the distal.
- Hyperfunctional tooth may have wider PDL space.
- Normal PDL width is 0.25mm in normal function.
- Widest PDL space at cervical and apical parts, whereas narrowest at the middle.

## PDL elasticity:

It comes from:

1. The wavy course of the principle fibers, which allows for a slight movement of teeth.
2. Intermediate plexus
3. The presence of oxytalan and elastic fibers, although they are relatively few.

# PDL functions

```
graph TD; A[PDL functions] --> B[Physical]; A --> C[Formative and remodelling]; A --> D[Nutritive]; A --> E[Sensation];
```

## Physical

- Attachment of teeth to the bone
- Transmission of occlusal force to the bone
- Resistance to the occlusal impact forces
- Protect the vessels and nerves from mechanical force injury

## Formative and remodelling

- Formation and resorption of bone and cementum
- Break down and replace the old cells and fibres

## Nutritive

- By blood vessels, which supply bone, gingiva and cementum
- Provide lymphatic drainage

## Sensation

- Provide sensation, tactile, pressure and pain
- Provide mechanoreceptors, transmit sense of localisation unlike the pulp (no sense of localisation)

## Blood supply of PDL

It derived from inferior and superior alveolar arteries of mandible and maxilla respectively, reach the PDL from three sources;

1. Apical vessels supply the apical region of the PDL.
2. The transalveolar vessels through alveolar bone
3. Anastomosing vessels from the gingiva

## Treatment planning for patients with periodontal diseases



## **The treatment may be divided into 4 phases:**

- 1. Systemic phase of therapy.
- 2. Initial phase (cause related therapy).
- 3. Correction phase (additional therapeutic measures).
- 4. Maintenance phase (supportive periodontal therapy).

- **Treatment goals:**
- 1. Reduction or resolution of gingivitis (bleeding on probing, BOP).
- 2. Reduction in probing pocket depth (PPD).
- 3. Elimination of open furcations in multi-rooted teeth.
- 4. Absence of pain.
- 5. Individually satisfactory esthetic & function.



- BOP measurement: a periodontal probe inserted to the bottom of gingival crevice or periodontal pocket and move gently along the root surfaces, if bleeding occurs within 30 seconds the site give score (1).



- PPD measurement: is the distance from the gingival margin to the most apical penetration of the periodontal probe insert into the gingival crevice or periodontal pocket without pressure or force and measure in mm.



## **Systemic phase of therapy includes:**

**1)** Precautions for protecting the general health of the dental team and other patients against infectious & contagious diseases e.g. infectious hepatitis, HIV infection, Herpes simplex virus & TB.

**2)** Protection of patient's health against harmful systemic effects of routine therapy. the complications: Infection, bleeding, cardiovascular incidents, allergic reactions.

- **Infection:** patients with cardiac disease and disorders involving the endocardium are susceptible to infective endocarditis as a result of blood –borne infection. Antibiotic prophylaxis 1hour before dental procedure e.g. Amoxicillin, Clindamycin and Azithromycin with a high standard of oral and dental health is reserved for those Patients.
- **Bleeding:** patients on anticoagulant drugs (e.g. Salicylate), liver cirrhosis or high alcohol consumption, blood dyspraxia or hemophilia are at a risk for bleeding complications, thus it is recommended to render treatment in small segments.
- **Cardiovascular incidents:** they are often treated with anticoagulant drugs (e.g. Aspirin, indomethacin) may develop bleeding problems. Antihypertensive, diuretic, anti-arrhythmic may increase hypotensive episodes. Therefore, keep procedures short and control anxiety & pain with those patients.

- **Allergic reaction and drug interactions:**

The most common allergic reactions are allergies to local anesthesia (Novocain), Penicillin, Sulfa derivatives and Iodine. No new drugs should be prescribed as part of periodontal therapy without understanding their interference with the effectiveness of the drugs that the patient is already taking (e.g. Antidepressants) or alcohol, or create hazardous or synergistic action with such drugs.



**3)** All attempts should be made to decrease the effects of systemic disease such as blood disorders and diabetes mellitus before the periodontal treatment is initiated. Patients taking cortisone over a long period of time may yield a reduced rate of fibroblastic activity and hence a lowered resistance to infection during healing. It has to be realized that periodontal treatment may have a beneficial effect on the systemic health of the patient as well.

**4)** Controlling anxiety & low pain threshold:

**a.** Valium to be taken the night before, in the morning, and half an hour before extensive or surgical procedure.

**b.** Apply local anesthesia to control pain.

**c.** post-operative analgesics such as Voltaren, Ponstan may be prescribed.



**5)** Smoking constitutes the second most important risk factor in the etiology & pathogenesis of periodontal diseases after poor oral hygiene standards.



**6)** Treatment of emergencies:

Such as acute necrotic ulcerative gingivitis or periodontitis, periodontal abscess, acute endodontic periodontal lesion and extraction of hopeless teeth.

- **Objectives of initial phase (cause-related therapy):**

This is accomplished by:

- 1. Motivating the patient to combat dental disease (patient information).
- 2. Giving the patient instructions on proper oral hygiene techniques (self performed plaque control methods).
- 3. Scaling & root planning.
- 4. Antimicrobial therapy (local or systemic).
- 5. Control or elimination of additional retention factors for plaque such as: correction of restorative and prosthetic irritational factors & excavation of caries and restoration.
- 6. Occlusal therapy.
- 7. Orthodontic treatment.



- **Motivation:** detailed information about periodontal disease, its etiological factors, symptoms, consequences, prognosis the relationship between the presence of plaque & calculus in the mouth. Mechanical plaque control demands active participation of the individual subject and the establishment of proper oral homecare habits. patient's positive attitude to treatment may have a positive long term effect on his/hers tooth cleaning efforts.



- **Disclosing agent:** since dental plaque is white, sometimes it cannot easily be identified, particularly if it is not thick enough & /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.



- **Self-performed plaque control:**

Supragingival plaque is exposed to saliva and to the natural self-cleansing mechanisms existing in the oral cavity .the regular use of personal oral hygiene measures 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, inhibit the progression of the disease & inhibit the formation of supra & subgingival calculus which is plaque retentive factor.

- **Brushing:** 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, and the efficacy of brushing with regard to plaque removal is dependent on:
  - The design of brush.
  - The skill of the individual using the brush.
  - Frequency & duration of brushing.



- **Tooth brushes requirements:**

- ✓ It must be Nylon, Soft-medium strength ,rounded ends filaments.
- ✓ 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 area of the oral cavity.
- ✓ toothbrush is to be replaced every 2-3 months, because a worn toothbrush with frayed filaments loses resilience & is less effective in removing plaque than new brush.

**Dentifrices:** facilitating plaque removal and applying agents to the tooth surfaces for therapeutic or preventive reasons. The most important active ingredients in toothpaste are:

- ✓ **-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 & shape which facilitate plaque & stain removal without producing hard & soft tissue damage.
- ✓-**Whitening agents**: whiten stained teeth.
- ✓-**Detergents**: sodium lauryl sulfate has antimicrobial & plaque inhibitory properties.