Lec. 1

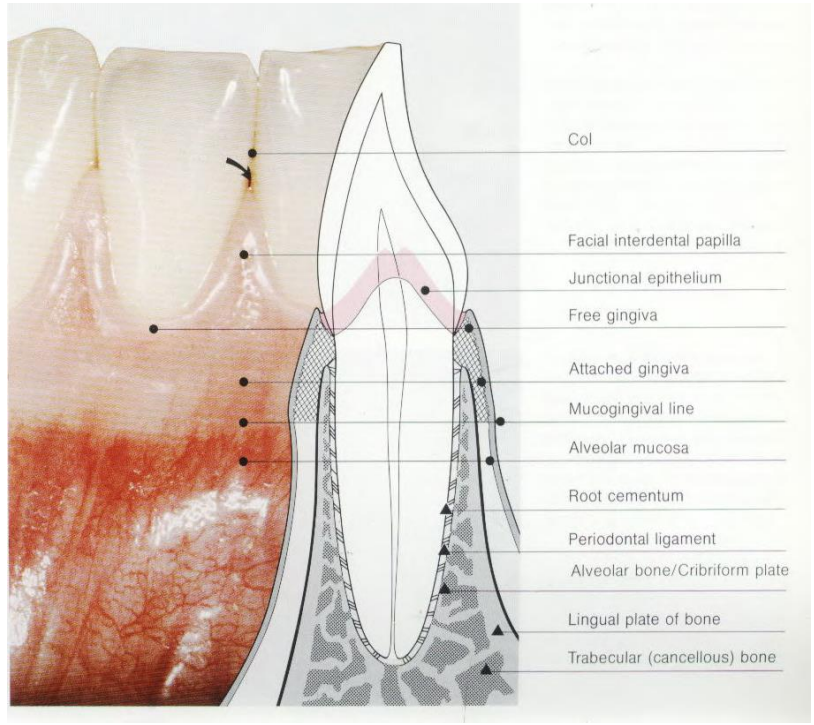
د.نهى عكاب

What is Periodontology?????

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

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

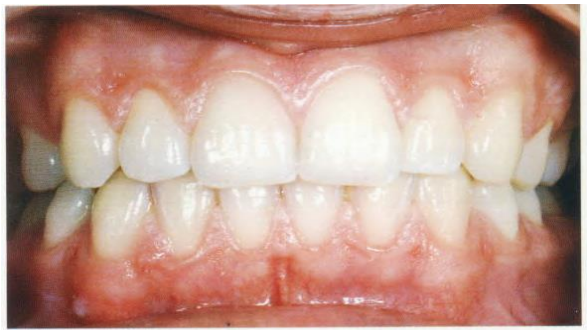
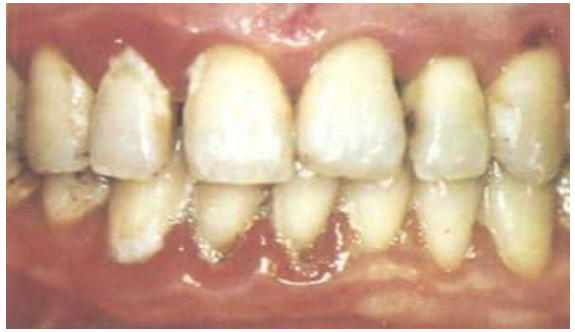
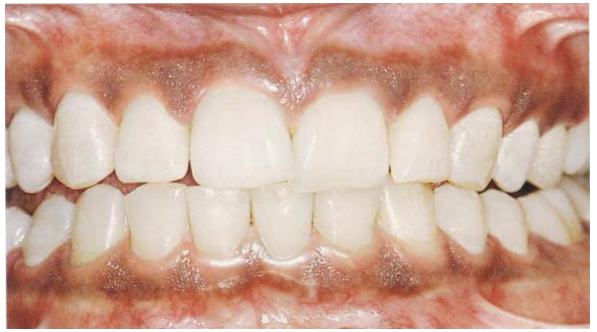
1. Periodontal ligament (PDL).
2. Gingiva.
3. Cementum
4. Alveolar bone



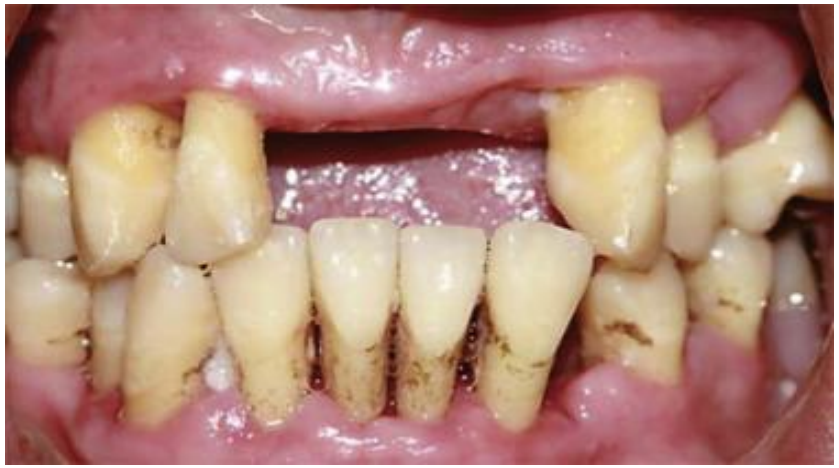
Periodontics: is the branch of dentistry that specified to treat and prevent periodontal disease.

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

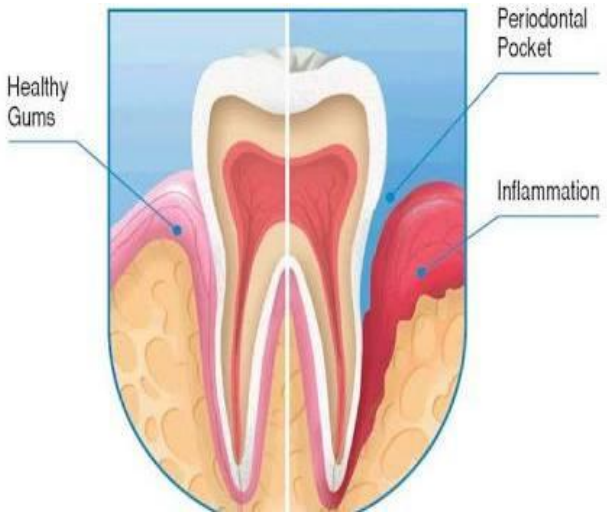
Gingivitis: a reversible inflammation of the gingiva, without loss of attachment of PDL, usually associated with erythema (redness), swelling and bleeding on probing.

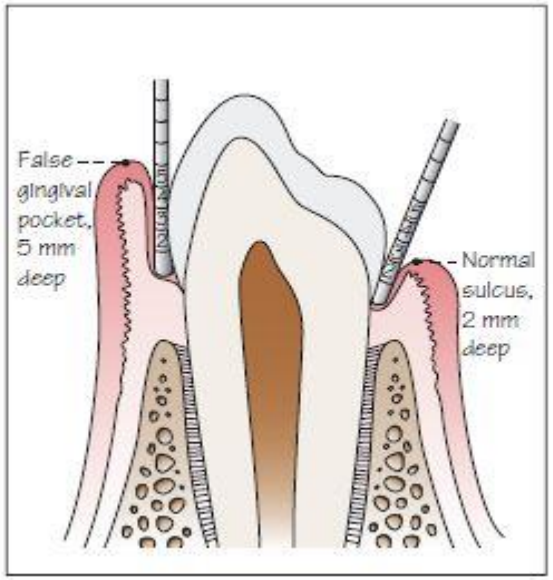
Periodontitis: Inflammation of the tooth supporting tissues, resulting in permanent destruction of the periodontium.

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.

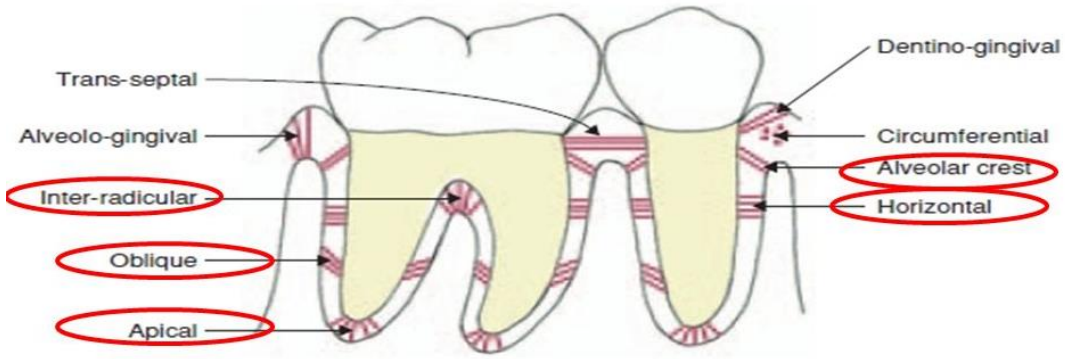
-Following tooth eruption, the principal fibres become organised in bundles, continuously connecting the root cementum to the alveolar bone. However, the tooth underwent active eruption thought to consist of two separated parts; one is located towards the cementum and the other towards the alveolar bone and connect together at the mid way through intermediate plexus.

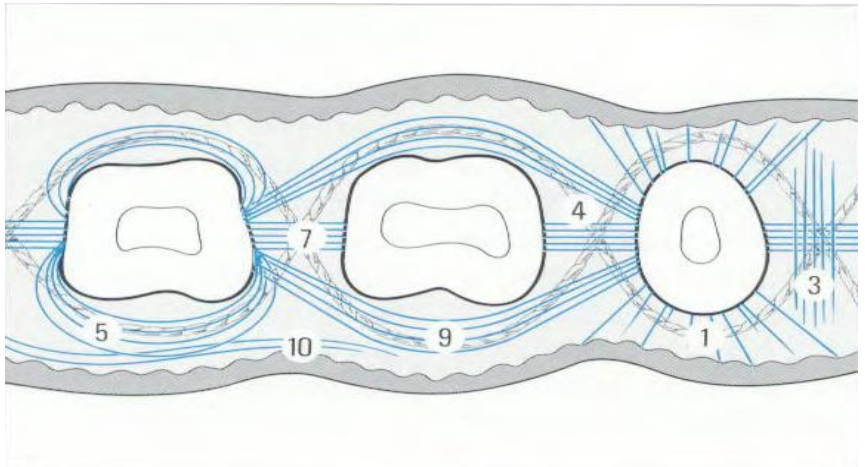
- Sharpy's fibres are the part of principal fibres that insert into the cementum from one side and the alveolar bone from the other side.

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:

1.Synthetic cells

Stem cells
Responsible for formation of osteoblasts, fibroblasts and all other type of cells

Osteoblasts
responsible for alveolar bone formation

Fibroblasts
the main components of PDL, which produce various types of fibers such as collagen, oxytalan and elastic fibers

Cementoblasts
- Responsible for cementum deposition
- Lining the cementum surface

2. Resorptive cells

- **Osteoclast**: large multinucleated cell, responsible for bone resorption
- **Cementoclasts**: Although cementum is not remodelled as alveolar bone, that may occur in certain conditions
- **Fibroblasts**: Has the capacity to phagocyte old collagen fibers by hydrolytic enzymes during normal turn over or disease

3. Epithelial rests of malassez: are found close to the cementum, which are reminants of hertwig root sheath. They proliferate in response to a stimuli 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:

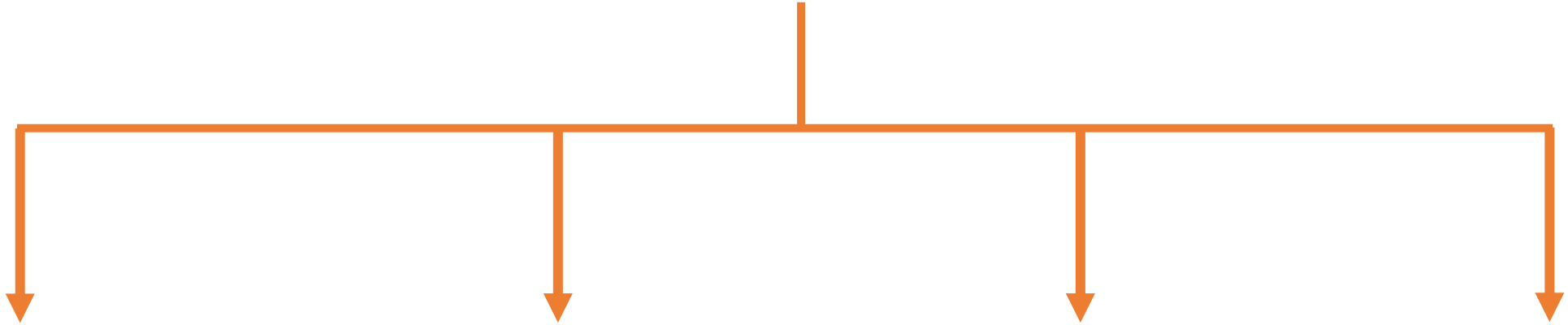
The width of PDL space varies in respect to age, location of tooth and the degree of stress to which the tooth is subjected.
-It found thinner on the mesial root surface than the distal.
- Hyperfunctional tooth my have wider PDL space.
- Normal PDL width is 0.25mm in normal function.
- Widest PDL space 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



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 resorptivion 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

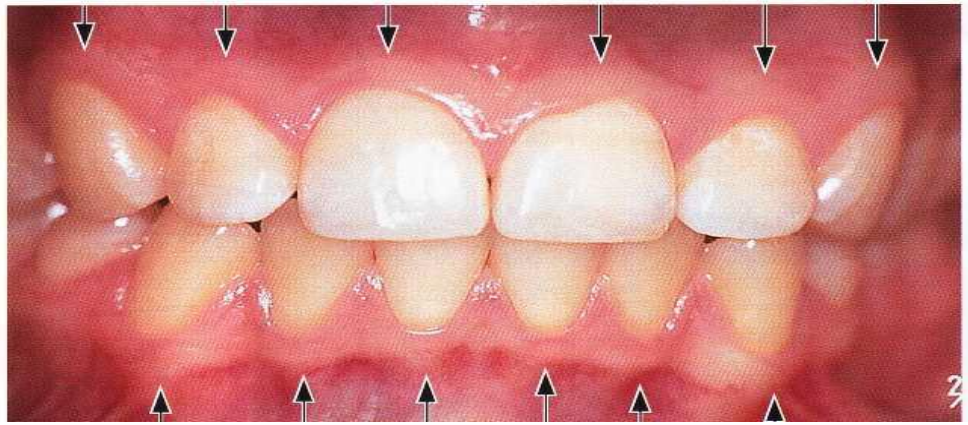
Lec. 2

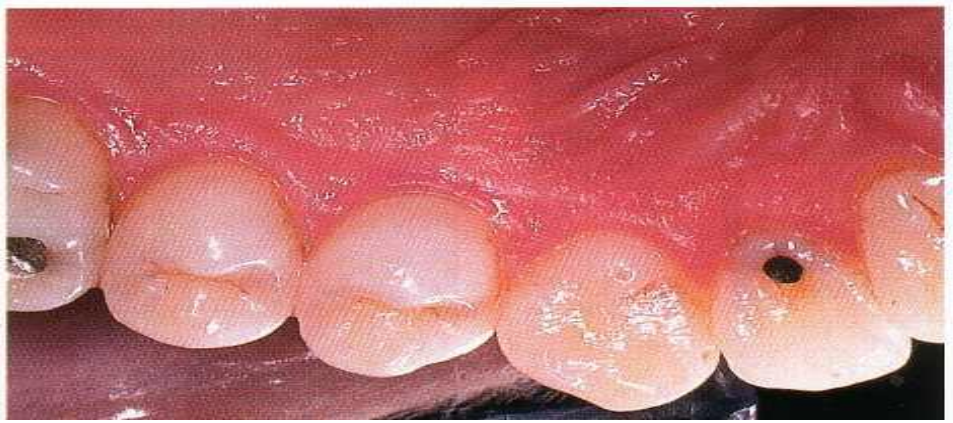
د.نهى عكاب

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.

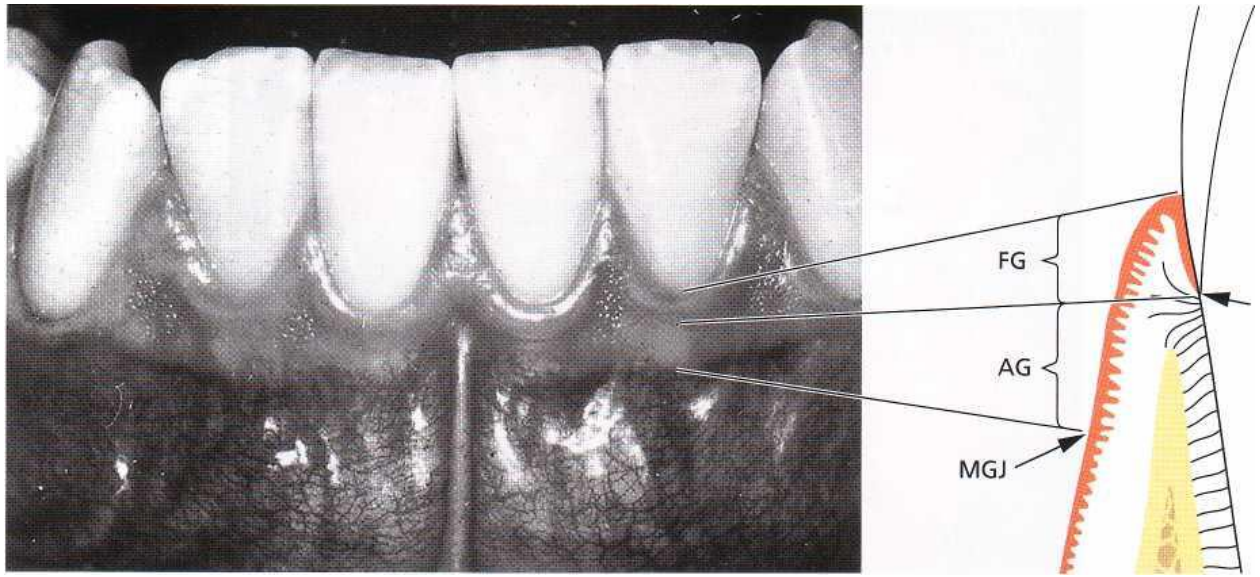




2- Specialised mucosa: covers the dorsum side of the tongue.

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

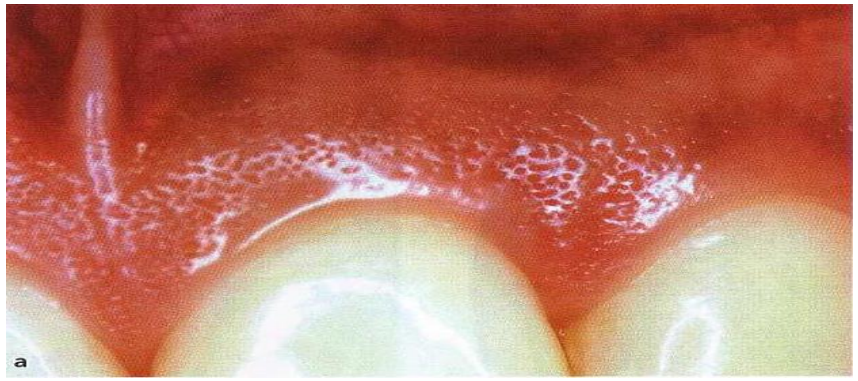
Is the part of the masticatory mucosa, covering the alveolar process and surrounding the cervical portion of the teeth. It is divided into 3 parts:

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

1- Marginal gingiva

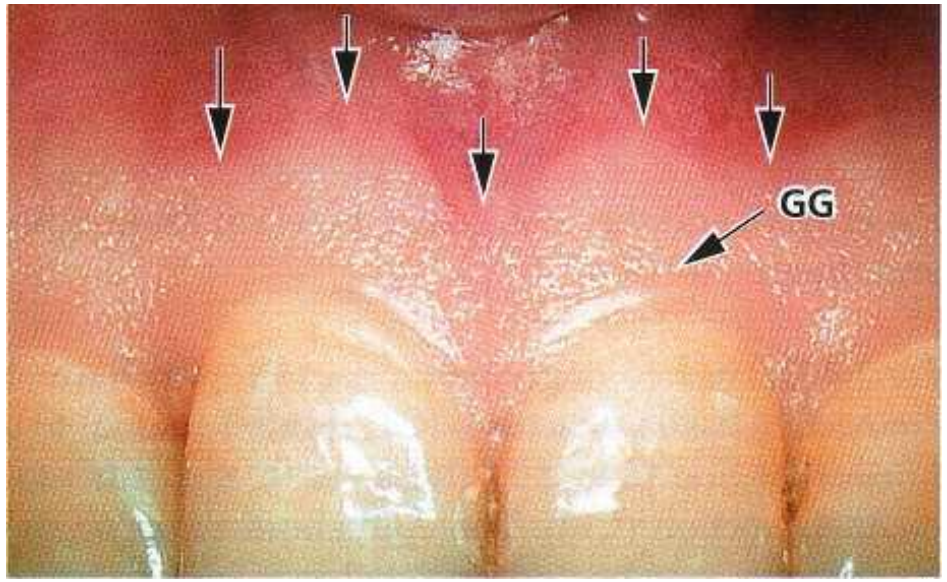
Is the most coronal portion of the gingiva, surrounding the teeth in a collar like fashion but not attached to them. It is demarcated apically from the attached gingiva by the free gingival groove.

- 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:

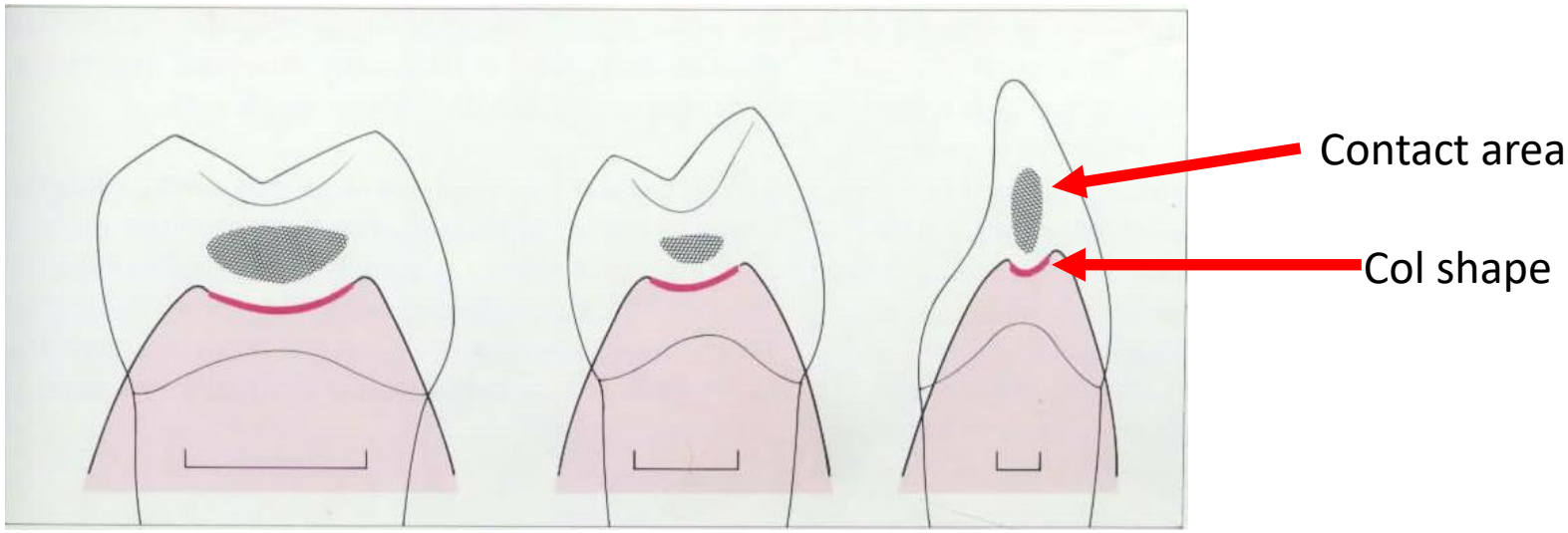
1- Pyramidal shape:

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.

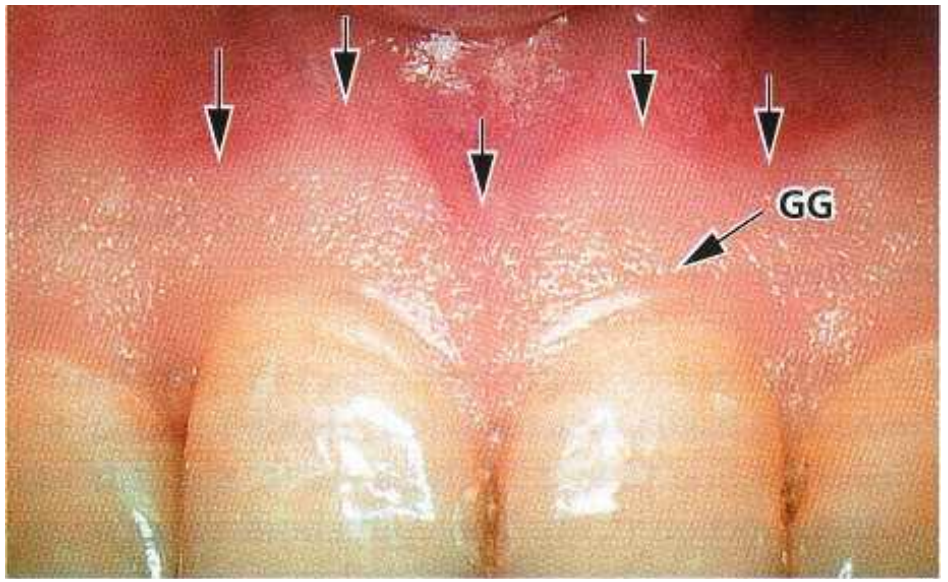
3- Gingival consistency

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

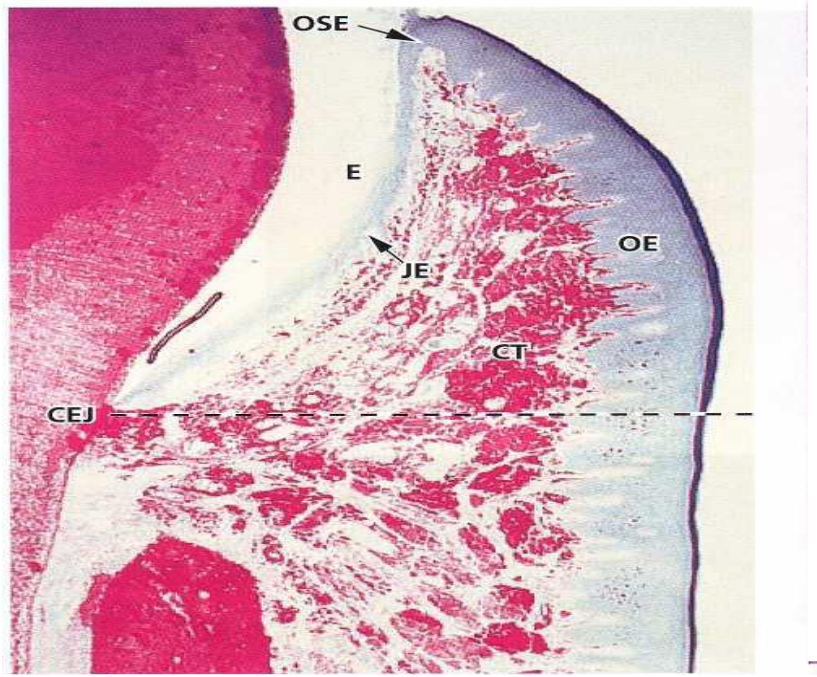
The attached gingiva has usually stippled surface, whereas, the free gingiva is smooth.



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

Gingival epithelium is described as follows:

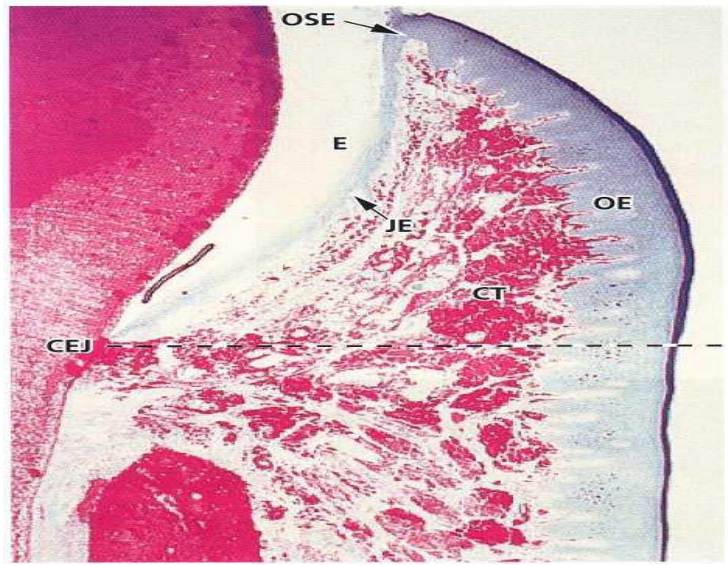
1- Oral epithelium faces the oral cavity
2- Sulcular epithelium faces the tooth in the gingival sulcus only
3- Junctional epithelium provides the contact between the gingiva and the tooth

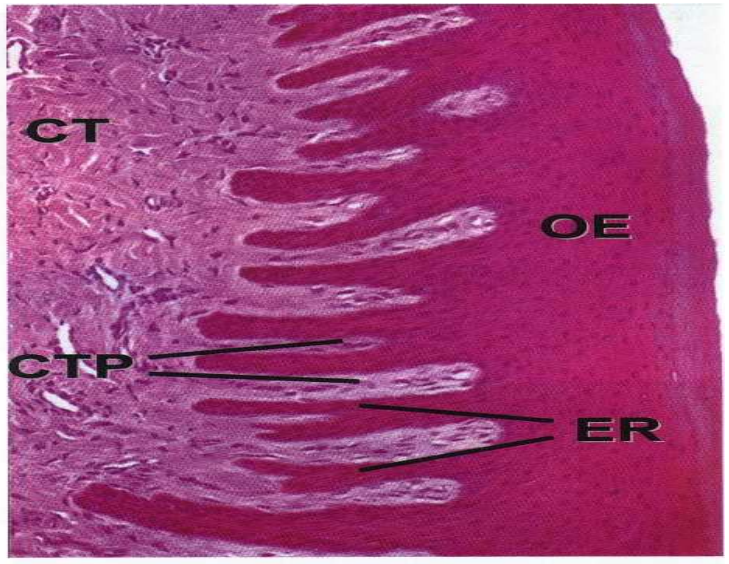


Oral epithelium

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)

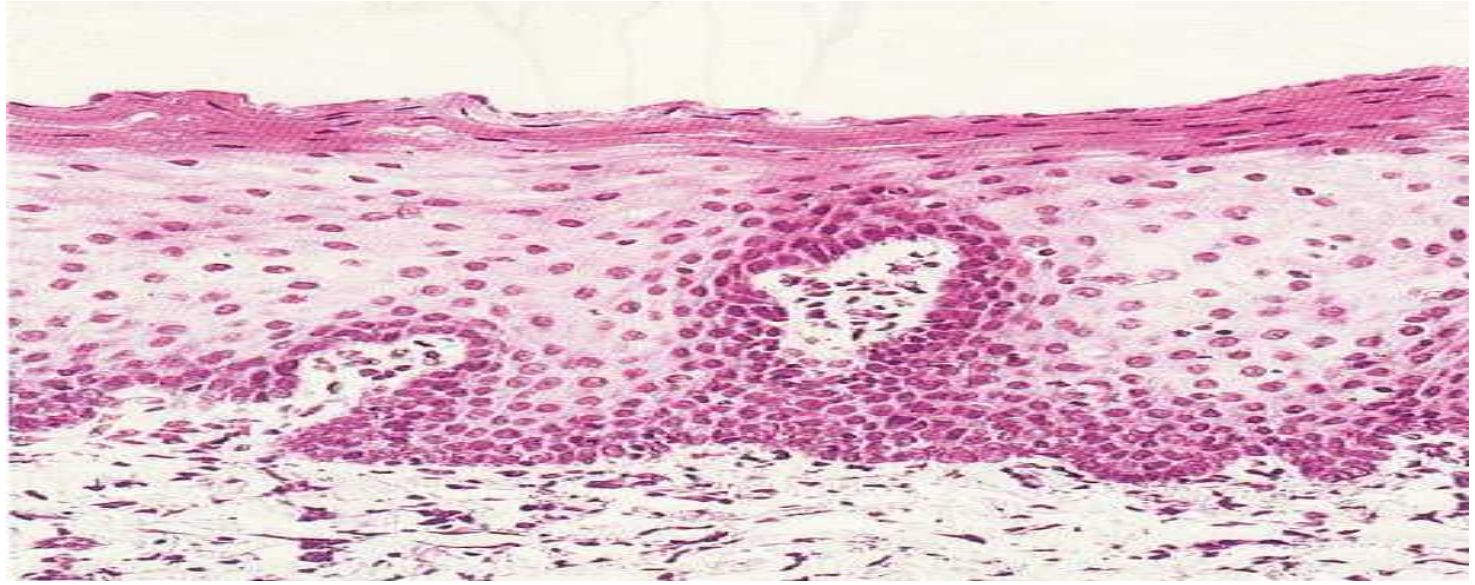
-The thickest layer
-Cells appear to have cytoplasmic spines
- It contains Langerhans cells

Stratum granulosum (granular cell layer)

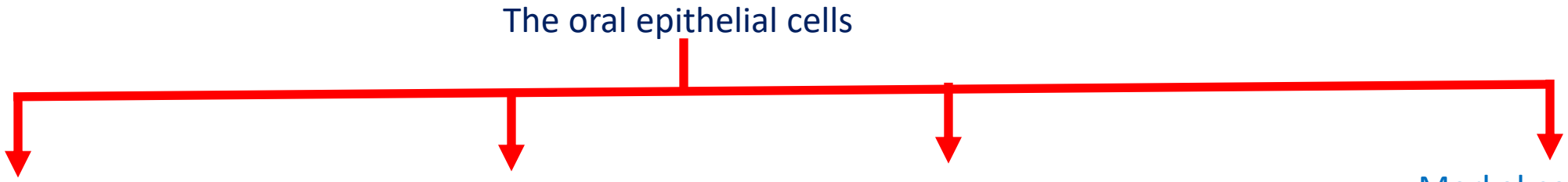
-Cells appear to be flattened
-Keratohyalin granules may be seen here

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



Keratinocytes

-Keratin productive cells, comprise about 90% of the total cell population

-Undergo continuous proliferation and differentiation

-Keratin may present in the stratum corneum, contributing to protect epithelial function

Melanocytes

- Producing melanin pigment responsible for dark gingival colour

Langerhans

-Playing role in the defence mechanism of oral epithelium through immunological function

Merkel cells

-Located in the deeper layer of epithelium

-Contain nerves endings, having tactile receptors

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.

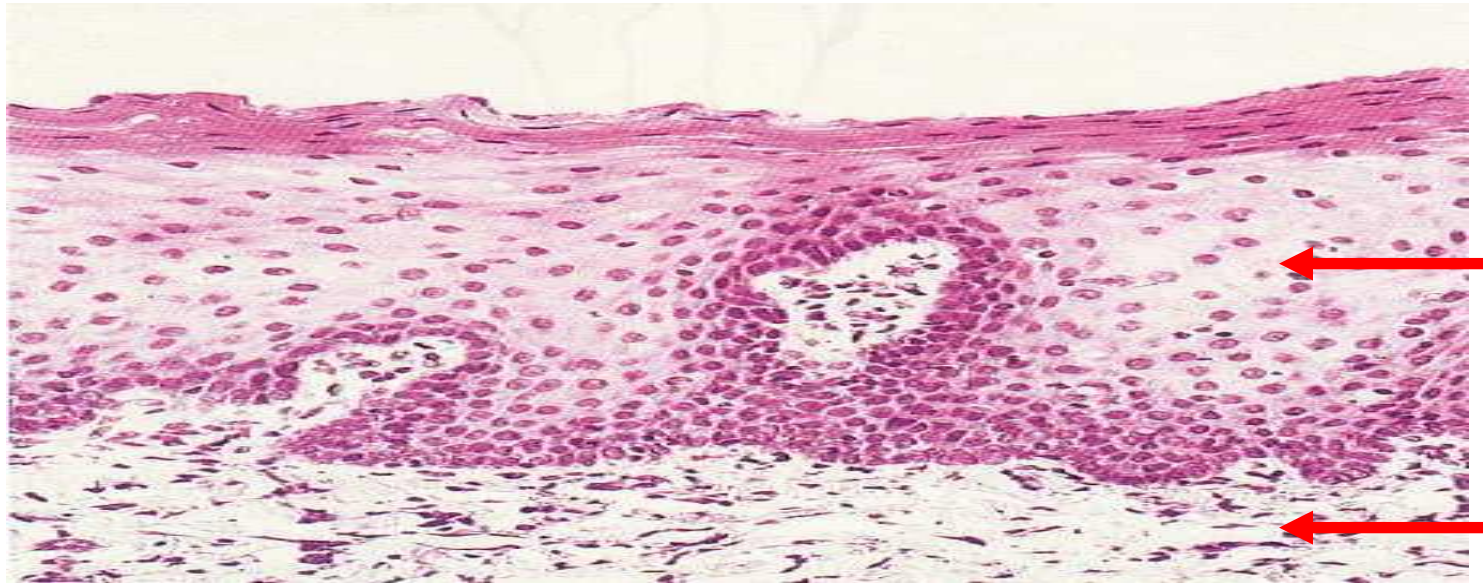
- The basal cells are found immediately adjacent to the connective tissue and separated from this tissue by a basement membrane (basal lamina).

The basement membrane is consisted of:

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

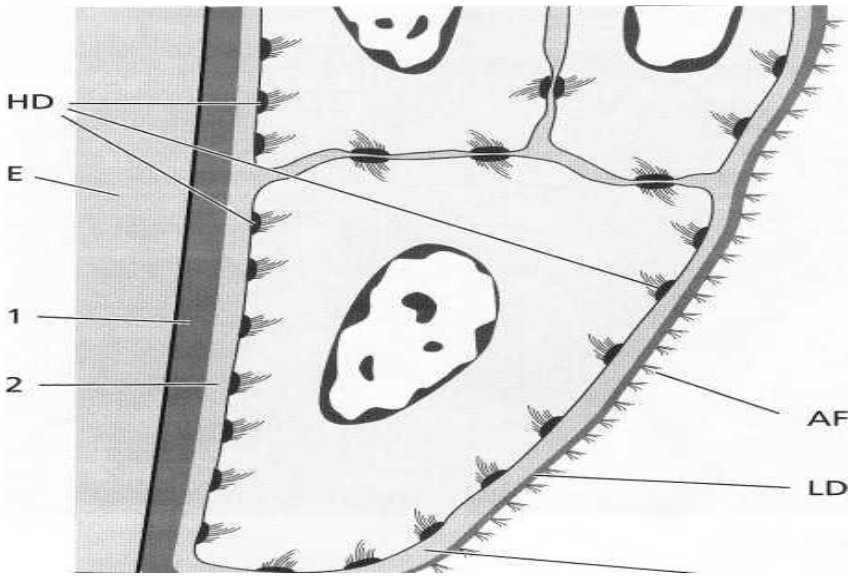
2- Lamina Densa: is located beneath the lamina lucida. The anchoring fibers project from it towards the connective tissue.

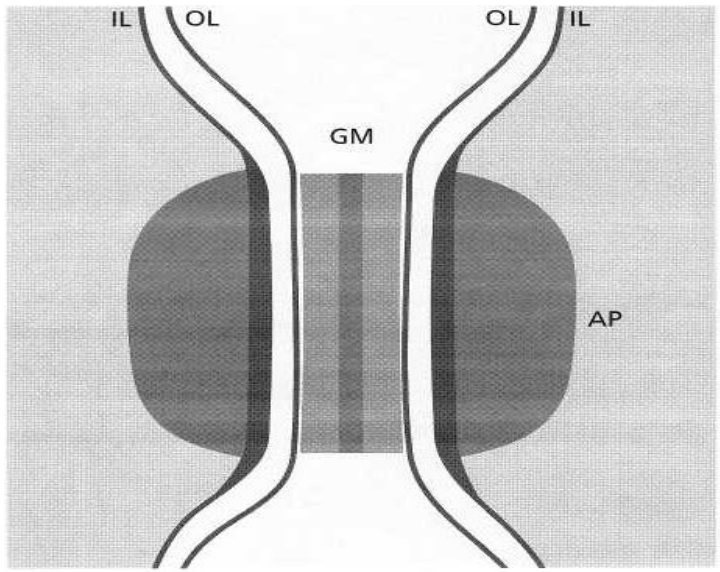
-The epithelial cells are joined together by specific structure called desmosomes, which is composed of two hemidesmosomes separated from each other by granulated material.



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

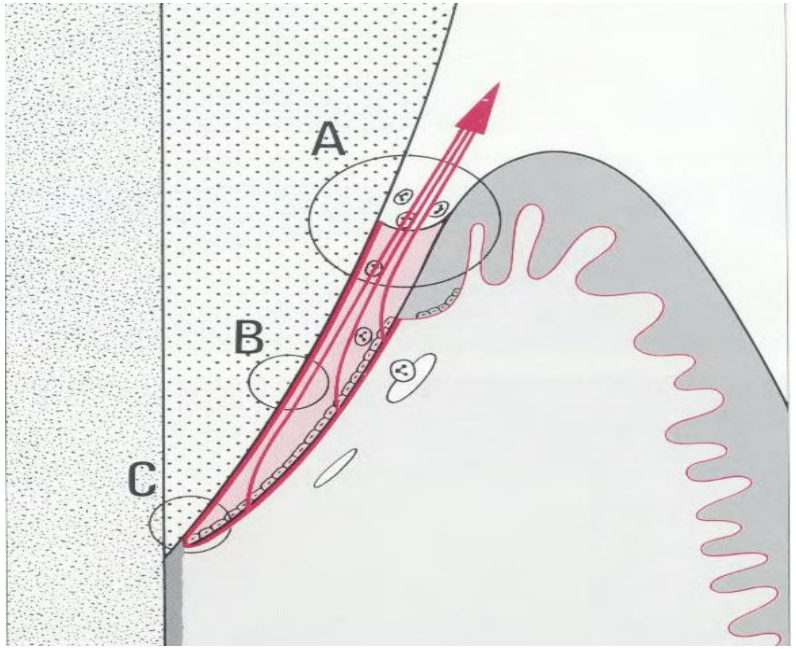




Sulcular epithelium

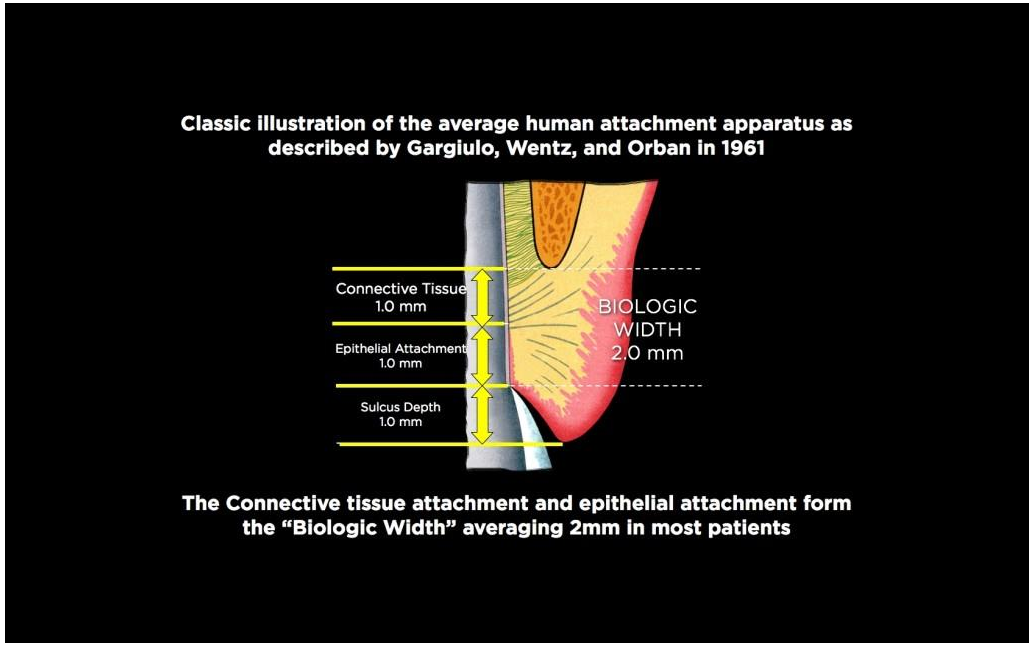
It lines the gingival sulcus, has a thin, non-keratinised stratified squamous epithelium without rete pegs. It extends from coronal limit of the junctional epithelium to the crest of the gingival margin.

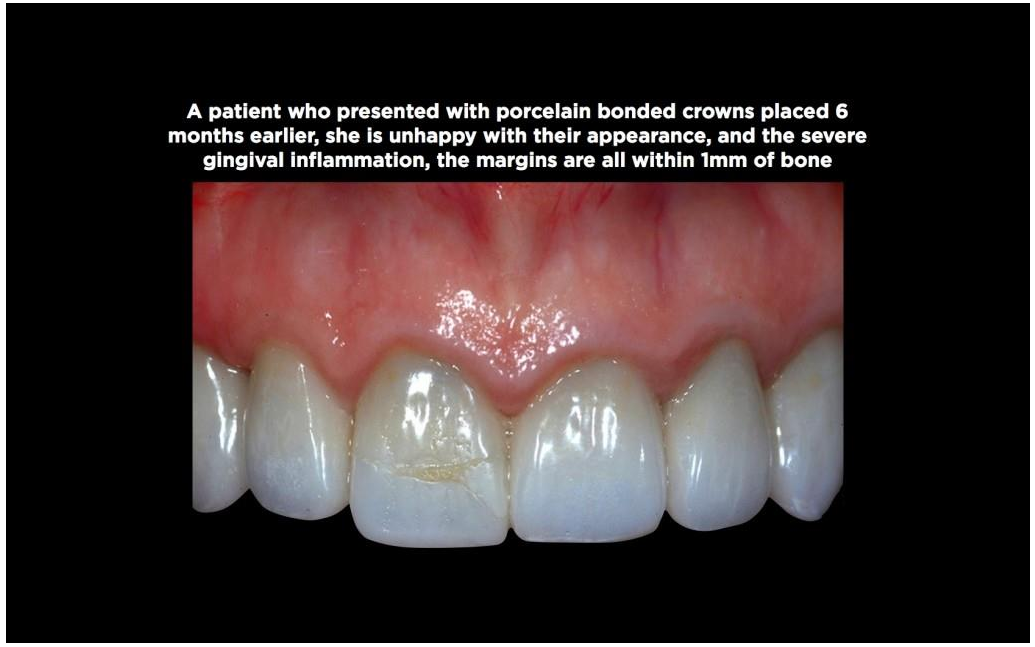
-The importance of sulcular epithelium is coming from its thin consistency and may act as a semipermeable membrane through which the fluid can seep from the gingiva into the sulcus, make it easier for bacterial products of dental plaque to penetrate into the connective tissue, stimulating inflammation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

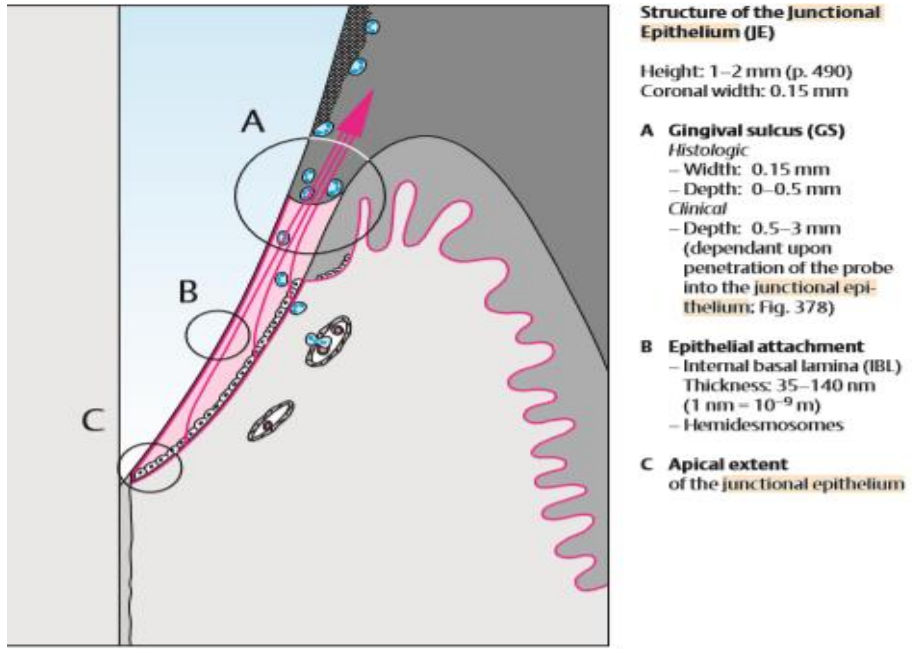




Junctional epithelium (JE)

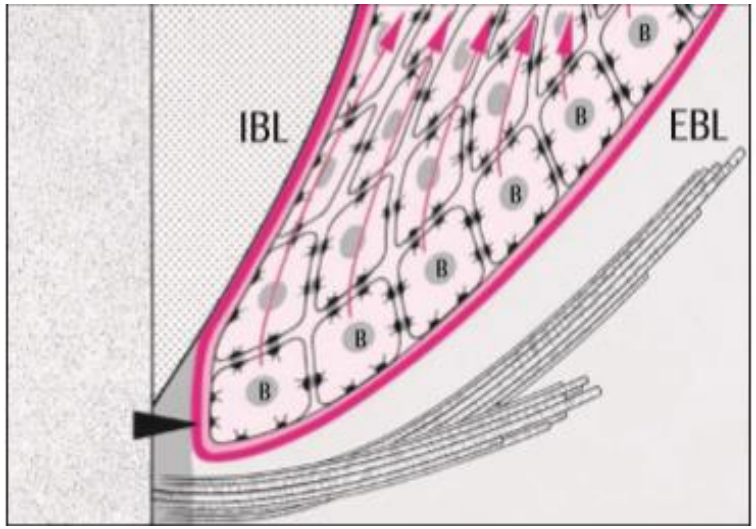
The type of epithelium that attached the gingiva to the tooth surface. It consists of stratified squamous non-keratinizing epithelium. It is usually consisted of 3-4 of thick layers in early life, however, the number of layers increases with age to 10-20. It is thicker in the coronal portion but become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

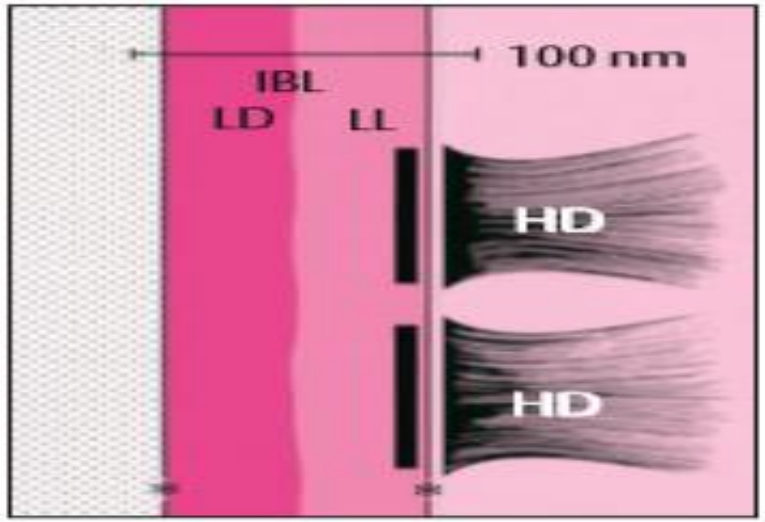
Junctional epithelial cells can be grouped in two layers:
1- The basal layer
2- the supra-basal layer
It is continuously renewed through cell division in the basal layer and the cell migrate coronally to the base of the gingival sulcus, where the cells can shed (cell turn over).



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

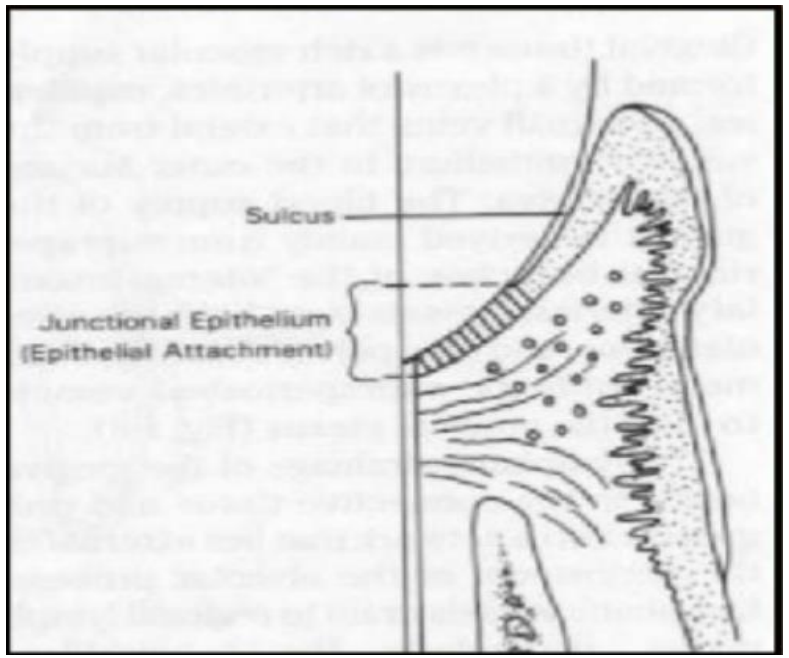
The functions of GCF

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

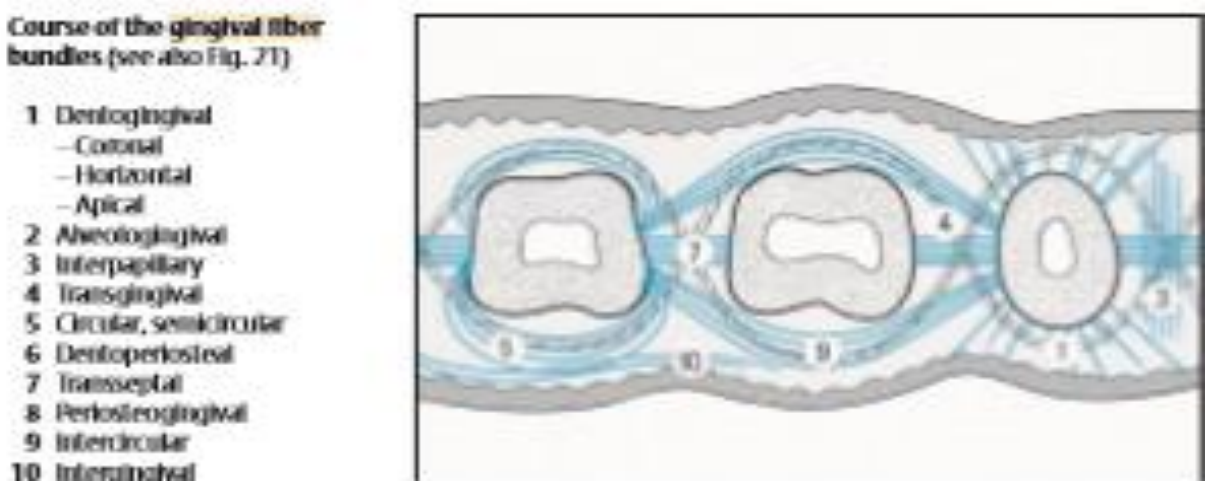
The supra alveolar crest fibers are arranged in groups of bundles according to their insertion and orientation in the tissue

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



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

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 though 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

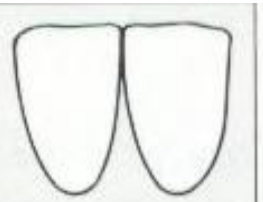
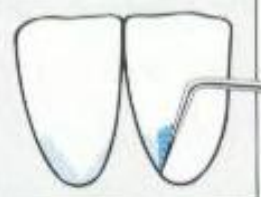
Gingival tissue has rich vascular supply which arise from the terminal branches of the internal maxillary artery

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

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

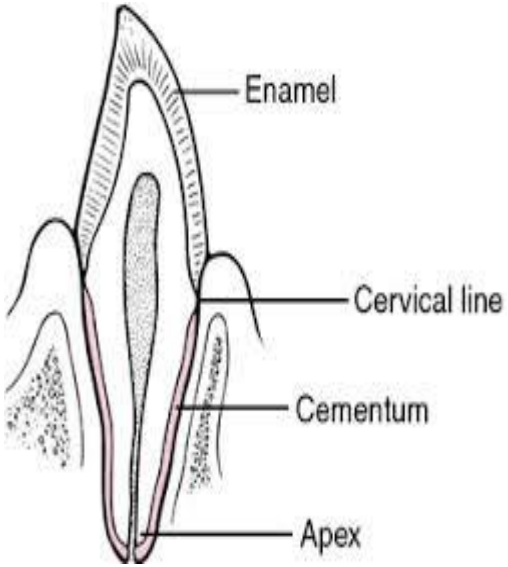
59 Gingival Index (GI)

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.

Cementum

It is a thin specialised calcified tissue covering the root surfaces of the teeth



It has many features similar to the bone tissue however it differs from bone by:

1- Microscopical organisation
2- Has no innervation
3- Has no blood or lymphatic vessels
4- Does not undergo physiological remodeling (deposition and resorption), but it is characterised by continuous deposition throughout life

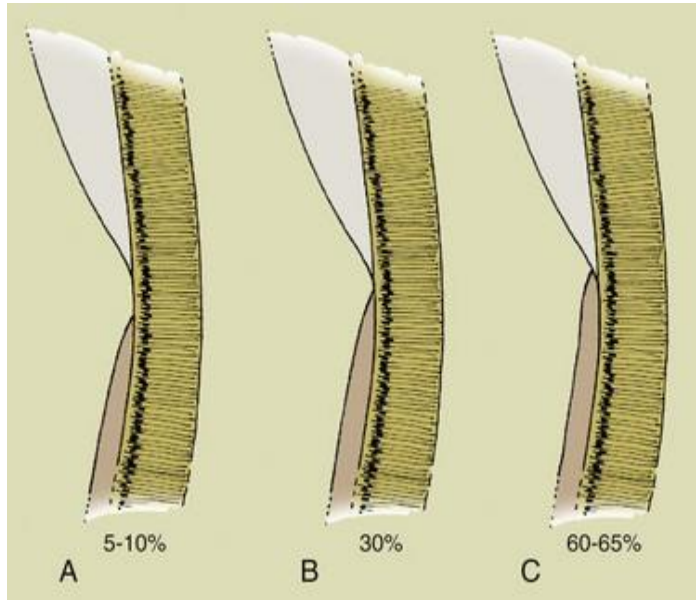
Cementum functions

- Anchorage the tooth to the alveolus
- Attach the PDL fibers to the teeth
- Contribute in the process of repair after damage to the root surface and following regenerative periodontal surgical procedures

Cemento-enamel junction (CEJ)

There are three types of relationships exited between the cementum and the enamel

First: Cementum overlaps the enamel (60-65%)
Second: edge to edge (30%)
Third: Cementum and enamel fail to meet (5-10%), in this condition, the possibility of tooth sensitivity is high due exposed dentin in case of gingival recession



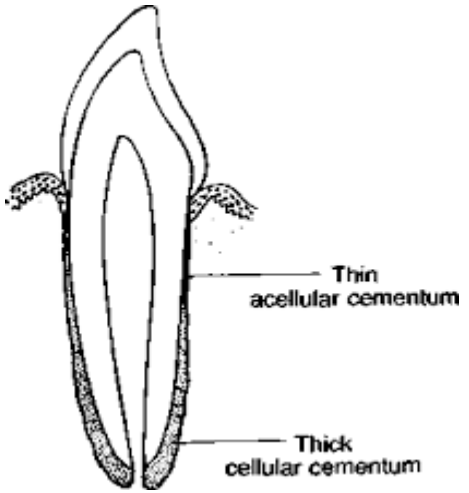
Types pf Cementum

1- Primary (acellular cementum):

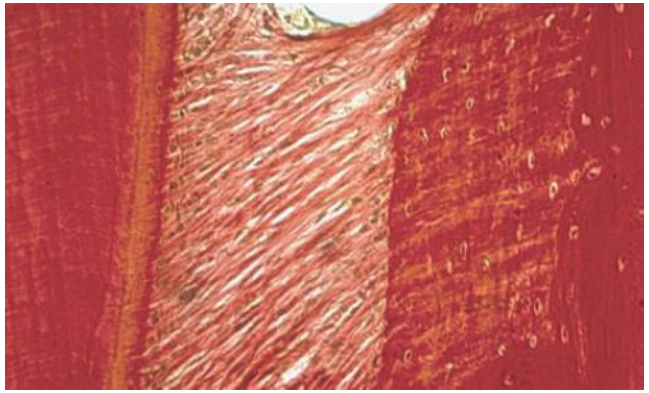
- Is the first to be formed in conjunction with root formation and tooth eruption
- It does not contain cells and sharpey's fibers make up most of its structure
- Generally it covers the cervical third of the root

2- Secondary (cellular cementum)

- It is formed after tooth eruption and in response to functional demands, therefore, it grows faster and over a thin layer of acellular cementum at the apical third of the root and furcation area of the multirooted teeth.
- It contains cells (cementocyts)
- Sharpey's fibers occupy only a smaller portion of this type of cementum
- It is less calcified than acellular (primary cementum)



Cementum's structure



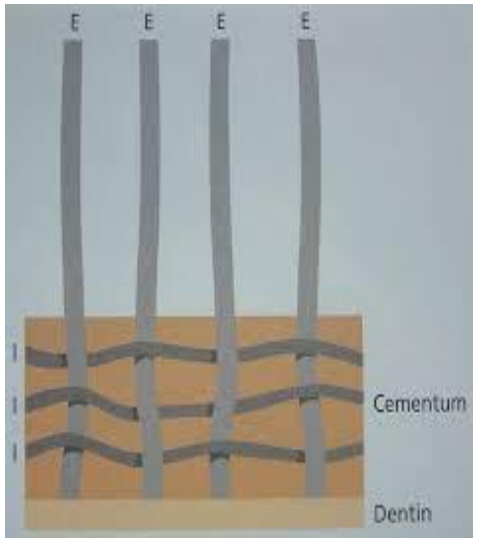
- Fibrous elements (collagen fibers)
- Cellular elements
- Calcified interfibrillar matrix

1- Fibrous elements

a. Extrinsic fibers (sharpey's fibers): they are embedded portion of the principal fibers of PDL and are formed by fibroblast cells.

- Sharpey's fibers make up most of acellular cementum structure and inserted at right angles to the root surface and penetrate deep into the cementum

b. Intrinsic fibers: these fibers are produced by cementoblast cells and oriented more or less parallel to the long axis of the root and form a cross banding arrangement with sharpey's fibers

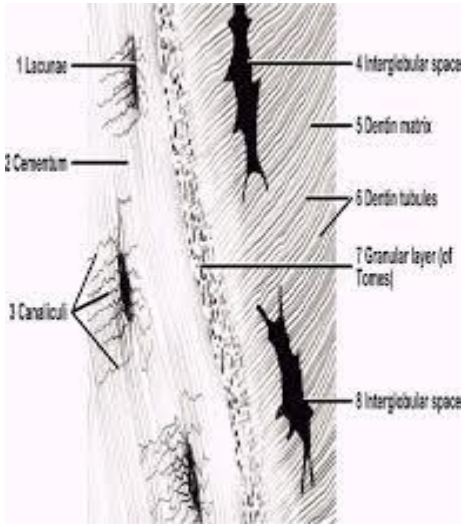


2- Cellular elements:

-Cells that associated with cementum are few and generally reside within PDL such as:

a. Cementoblast cells are responsible for the formation of cellular and acellular cementum

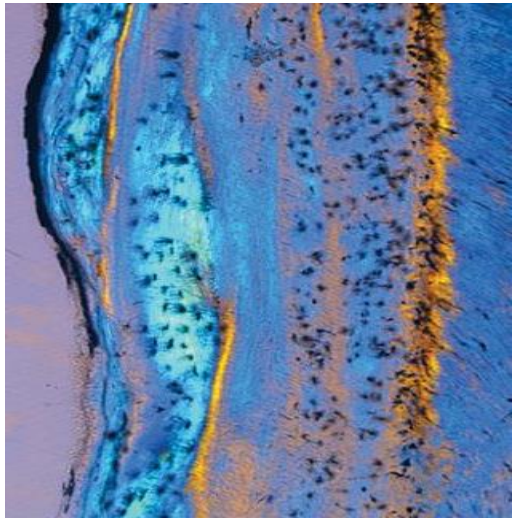
b. Cementocyte cells: are found only in cellular cementum. They are located within spaces called lacunae which communicate with each other through canaliculi that assist in transportation of nutrients and contribute to the maintenance of the cementum vitality



c. Fibroblast: cells are belong to the PDL where they are responsible for synthesis of principle fibers, but since these fibers become embedded in cementum, fibroblasts indirectly participate in the formation cementum

d. Cementoclast cells: are responsible for extensive root resorption which lead to primary teeth exfoliation, while permenant teeth do not undergo physiologic resorption but only localised cementum resorption, which may occur as concavities in the root surface and may be caused by local or systemic causes.

- Local conditions such as trauma from occlusion, orthodontic movement, cyst and occur mostly on the mesial surfaces in association with mesial drift
- Systemic conditions such as calcium deficiency and hypothyroidism



Reversal line: the newly formed cementum is demarcated from the root by a deeply staining irregular line which delineates the border of the previous resorption

Trauma from occlusion: forces that exceed the adaptive capacity of the periodontium and produce injury

Interfibrillar matrix: these are proteoglycans, glycoproteins and phosphoproteins formed by cementoblasts

Development of cementum

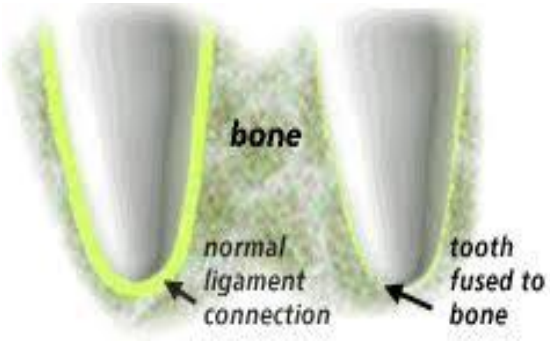
- Both cellular and acellular cementum are produced by cementoblasts
- Cementoid is the first formed as a non-calcified tissue containing collagen fibrils distributed in matrix
- Cementum is characterised by continuous deposition and increase in thickness throughout life
- A thin layer of cementum recognised on recently erupted tooth will tend to increase thickness with age
- Cementum formation is very rapid in the apical regions to compensates for tooth eruption and attrition
- The thickness of cementum is more pronounced in the apical third and in the furcation areas than in the cervical portion
- Cementum is thicker in distal than in mesial surfaces because of functional stimulation from mesial drift overtime

Mineralisation of cementum occurs by the deposition of hydroxyapatite crystals, first within the collagen fibers, and later upon the fiber surface and finally in the interfibrillar matrix

- Cellular cementum is less calcified than acellular once
- Cementum mineralisation is less than that of the bone, enamel and dentin

Hypercementosis: refers to a prominent thickening of the cementum, it may be localised to one tooth for example tooth without antagonists or with periapical lesion. Sometimes affects the whole dentition such as that occur in paget’s disease

Ankylosis: fusion of the cementum and alveolar bone with obliteration of the PDL. It results in resorption of the cementum and its gradual replacement by bone tissue and it may develop after a chronic periapical inflammation or occlusal trauma

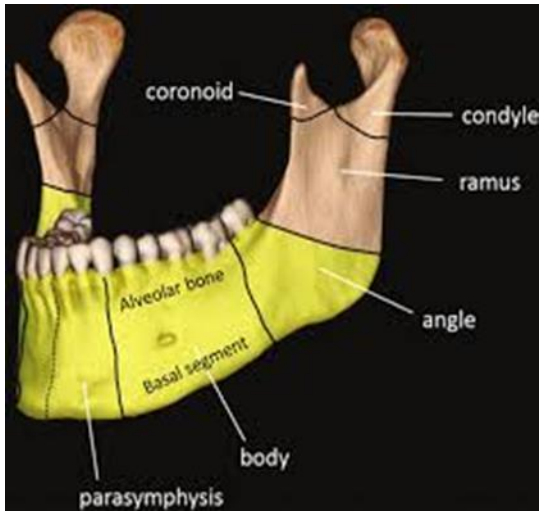


Alveolar process (AP)

- It is the portion of the maxilla and mandible that forms and supports the tooth sockets (alveoli)
- It develops in conjunction with the formation of and during the eruption of teeth and is gradually resorbed in case of teeth lost, thus it is a tooth dependent structure

Functions of the alveolar process

1. Comprises the attachment apparatus and the supporting tissue of the teeth together with root cementum and PDL fibers
2. Provide the osseous attachment to the PDL fibers
3. Distribute and resorb forces generated by mastication and other tooth contacts

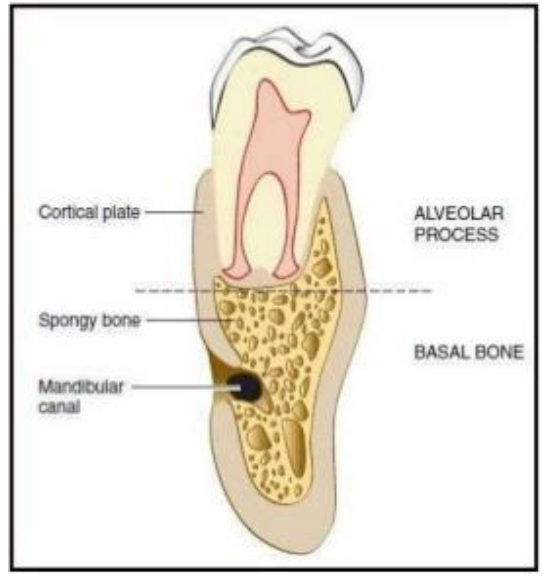


Alveolus: is the space within the alveolar bone that accommodates the roots of teeth

Parts of the alveolar process

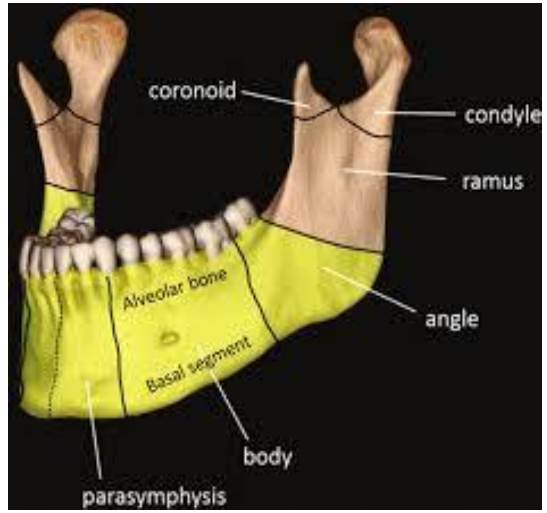
1. Alveolar bone process: is a thin layer of compact bone forming the inner socket wall (lines the alveolus), which is seen as the lamina dura in radiographs

- A great number of sharpey's fiber bundles are embedded into this layer of bone which is adjacent to the PDL, therefore it is called (bundle bone)
- Histologically, this bone contains many small holes or openings called (Volkmann's canals) through which the blood vessel, lymphatics and nerves link the PDL with the cancellous bone. So for this reason it is called cribriform plate



2. The external plate of cortical bone

3. Cancellous trabeculae or sponge bone: is located in the space between the external cortical plate and alveolar bone proper. They meet and fuse to form the alveolar crest.



Basal bone: is the portion of the jaw located apically but is unrelated to the teeth

Lamina dura: the layer of alveolar bone proper which radiographically surrounding the root of the tooth

The alveolar processes are subdivided according to their anatomical relationships to the teeth as following:

1. Interproximal bone (interdental septum): the bone located between the roots of adjacent teeth

2. Inter radicular bone: the bone located between the roots of multirooted teeth

3. Radicular bone: the alveolar process located on the facial, lingual or palatal surfaces of the roots of teeth

- The distance between the crest of the alveolar bone and the cementoenamel junction (average 2.8 mm)
- The thickness of alveolar process varies from one region to another depends on the position of the teeth in the arch and their relationship to one another
- For example teeth that are labially positioned in the dental arch will have thin labial and thick lingual radicular bone

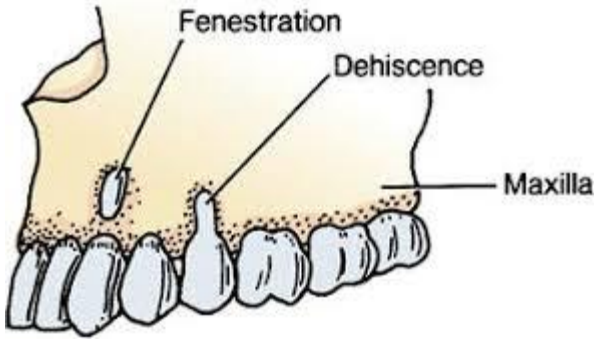
Bone marrow: are cavities which occupied by red marrow in the new born, whereas occupied by fatty or yellow marrow in adults

- The foci of bone marrow can be visibly seen as radiolucency in radiograph
- The common locations are the maxillary and mandibular molar and premolar areas

Periosteum and endosteum

Periosteum: is a layer of tissue covering the outer surface of bone, it contains collagen fibers, osteoblasts, fibroblasts, blood vessels and nerves

Endosteum: lined the marrow spaces inside the bone. It contains osteoblast



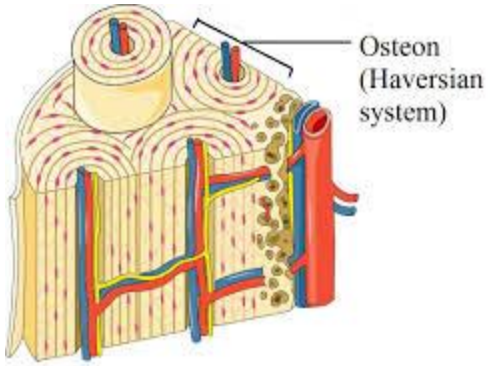
Anatomical defects of alveolar bone

1. Fenestration (window): it is an isolated area in which the root is not covered with bone and does not extend to the marginal bone

2. Dehiscence: a bone defect include denuded areas, which extend to the bone margin, exposing the root surface. The defects may extend to the middle of the root or farther

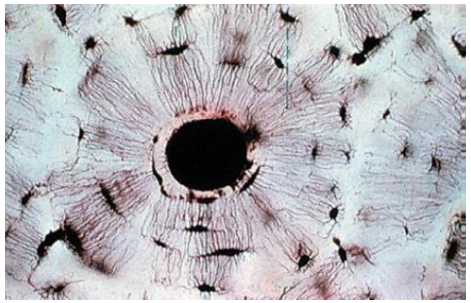
The cause of the defects is still not clear, but may be related to some factors such as shallow vestibule which cause pulling, prominent root, malposition or labial protrusion of the root with thin bony plate

Haversian system or osteon



It is an internal mechanism that bring a vascular supply to bones. It consists of central canal called Haversian canal, which contains blood vessels in their centre

- These blood vessels are surrounded by bone lamellae, arranged in concentric layers which constitute the centre od an osteon
- Blood vessels in this system are connected with each other by anastomoses running in the Volkmann's canals
- So the nutrition of bone is secured by incorporation of blood vessels in the bone tissue



Bone cells

1. Osteoblasts are bone forming cells, responsible for production of an organic matrix of bone, which consisting primarily of collagen fibers called Osteoid

- Osteoid undergoes mineralisation by the deposition of minerals such as calcium and phosphate which are subsequently transformed into hydroxyapatite crystals

2. Osteoclasts: are large multinucleated cells found in concavities on bone surface called howship's lacunae
- These cells are responsible for bone resorption

3. Osteocytes: are osteoblasts that become trapped in the bone matrix and later on mineralised in bone tissue
- They are located in lacunae and connected to each other by extending processes into canaliculi though which they get nutrients and remove metabolic waste products

Bone constituents

- Bone is consisted of 2/3 inorganic matter such as calcium and phosphate
- 1/3 of organic matrix, which comprised 90% of collagen fibers

Alveolar bone remodeling

- Alveolar bone undergoes a constant physiological remodeling (resorption and formation) in response to external forces such as occlusal forces
- Teeth tend to move mesially throughout life to compensate for wearing in the proximal contact areas.
- This refer to a physiological mesial migration
- Thus bone resorption occur in area of pressure on the mesial surface
- New bone will be formed on the distal surface
- This process of bone resorption and bone formation is called bone remodeling, which is very important in orthodontic treatment

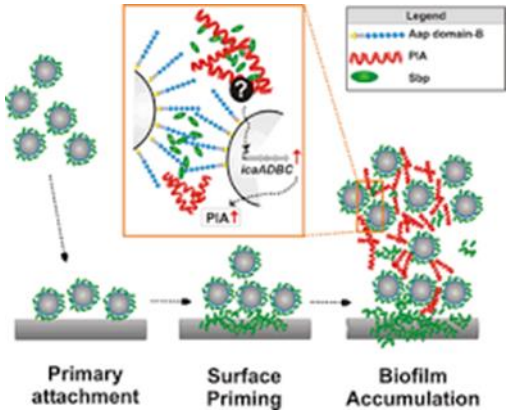
Dental plaque biofilm

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 spays



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

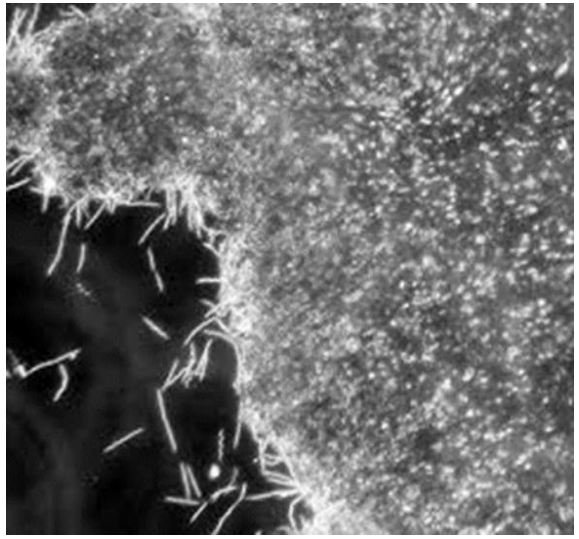
Dental plaque as a biofilm

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

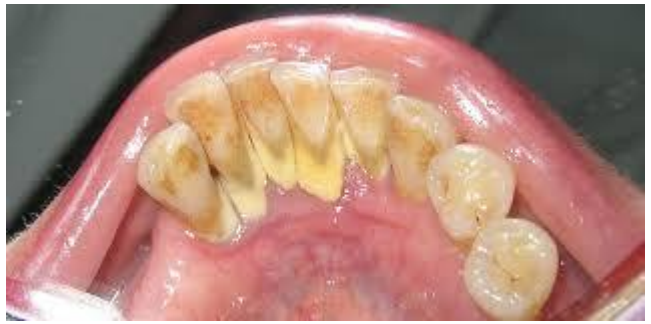
- Biofilm is composed of microcolonies of bacterial cells (15-20% by volume), which are distributed in matrix or glycocalyx (70- 80% by volume)
- Thick biofilms have demonstrated presence of water channels between the microcolonies.
- These water channels permit the passage of nutrient and other agents 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

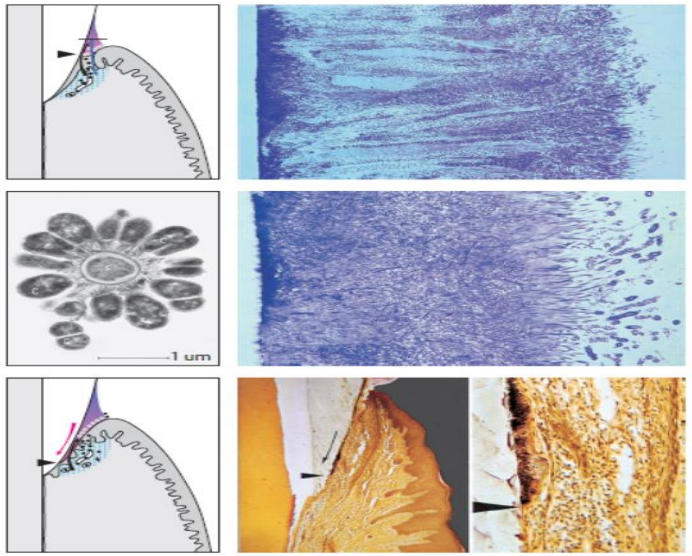


Classification of dental plaque

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 $2x10^{11}$ 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

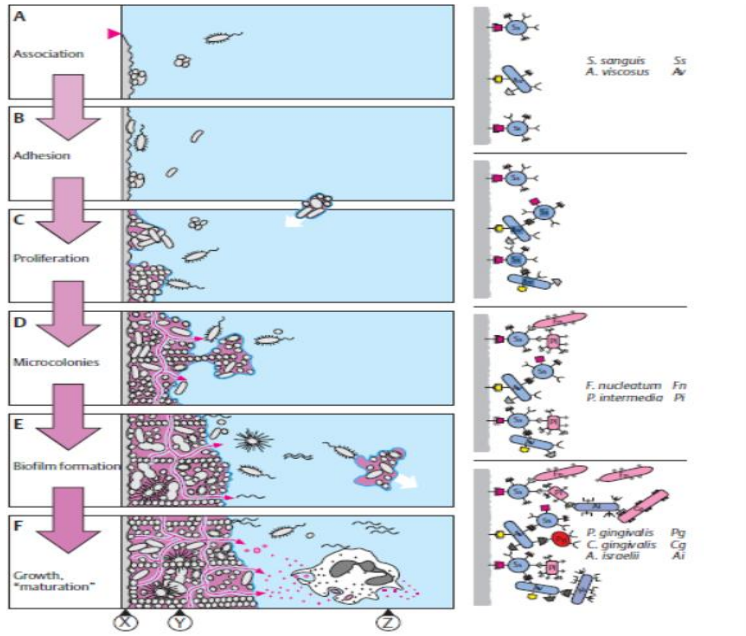
The **process of plaque formation** can be divided into three phases

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

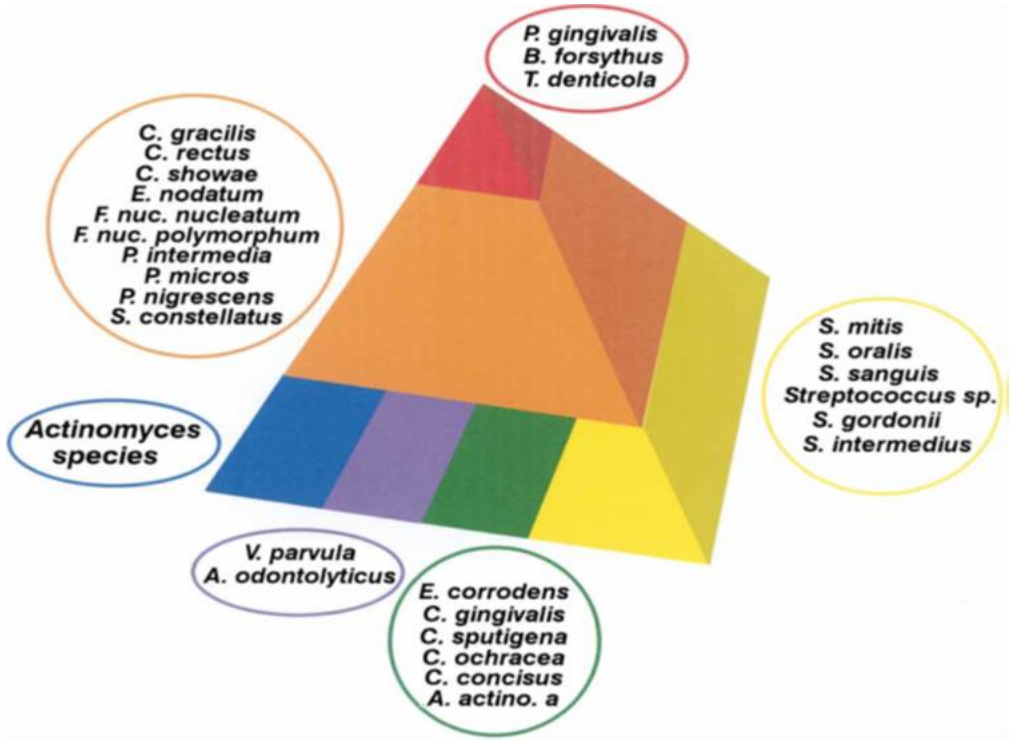
- However, recolonization begins immediately and bacteria can be detected within 3mins of introducing sterile enamel into he mouth
- Ther is a specific molecules on the bacterial surface called **adhesin**, which interact with receptors present in the dental pellicle
- This can determine whether or not 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 Actiomyces 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-charachterised 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

SIGMUND S. SOCRANSKY & ANNE D. HAFFAJEE

Periodontal diseases are infections caused by microorganisms 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*

Microorganisms of subgingival plaque

1- It resembles supragingival plaque, particularly with respect to plaque associated gingivitis without pocket

2- The bacteria comprise Gram-positive and Gram-negative cocci, rods and filamentous organisms

3- Spirochetes and flagellated bacteria are especially found in the apical extension of plaque

4- The layers of microorganisms facing the soft tissue lack definite inter-microbial matrix

Between subgingival plaque and the tooth, there is an electron dense organic material called **Cuticle**, which may contain remnant of epithelial attached lamina, connecting the junctional epithelium to the tooth

Factors affecting supragingival dental plaque formation

During the first 24h after cleaning tooth surface, plaque growth is very low from a clinical viewpoint.

- The lag time is due to the fact that the microbial population must reach a certain size before it can be easily detected by the clinician
- During the following 3 days, the plaque coverage can be detected clinically
- After 4 days an average of 30% of the total coronal tooth area will be covered with plaque

- The microbial composition of the dental plaque will change with a shift towards more an aerobic and Gram-negative flora, such as fusobacterium, filaments, spiral forms and spirochetes.
- Bacterial growth in older plaque is much slower than in newly formed dental plaque, presumably because nutrients become limited for much of the plaque biomass

Individual variables influencing plaque formation

The rate of plaque formation differs significantly between subjects, these differences might overrule surface characteristics

- There is a distinct difference can be recognised between **heavy (fast)** and **light (slow)** plaque formers

Variation within the dentition (dental arch) may show a difference in plaque formation

- Plaque formation in the lower jaw is much more that in the upper jaw
- Plaque formation in the interdental regions is more than that on the buccal or lingual surfaces

Microbiologic specificity of periodontal diseases

Nonspecific plaque hypothesis

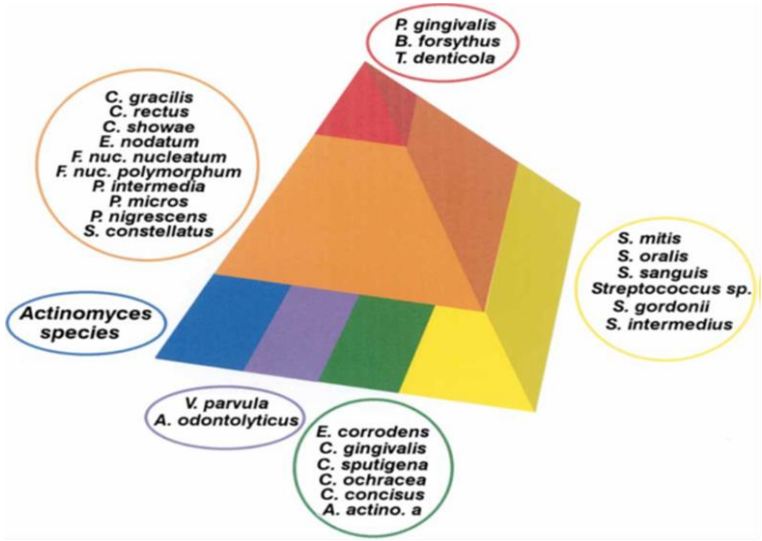
- In the mid 1900, periodontal diseases were believed to result from an accumulation of plaque over time
- In this case large amount of plaque would produce large of noxious products, which would essentially overwhelm the host's defences
- However, several observations contradicted these conclusions:
- First some individuals with considerable amounts of plaque, calculus, and gingivitis never developed destructive periodontitis
- In addition, individuals with periodontitis demonstrated considerable site specificity in the pattern of disease
- Some sites were unaffected, whereas advanced disease was found in adjacent sites
- In the presence of a uniform host response, these findings were inconsistent with the concept that all plaque was equally pathogenic

Specific plaque hypothesis

- It states that only certain pathogenic bacteria can cause the disease
- This concept predicts that plaque harbouring specific bacterial pathogens results in a periodontal disease, as they produce substances that mediate the destruction of host tissues

The association of Socransky's 'red complex' bacteria which are***, P. gingivalis***, ***T. forsythia*** and ***T. denticola*** with periodontal disease was based on the analysis of 40 different bacteria in >13000 plaque samples

- However, disease association studies do not reveal whether the presence of specific bacteria causes or correlates with the presence of disease
- In addition, these studies have shown that periodontal disease can occur even in the absence of these defined **pathogens**



Ecologic plaque hypothesis

In 1990's, Marsh and co-workers developed the **ecologic plaque hypothesis** as an attempt to unify the existing theories on the role of dental plaque in oral disease

- According to this theory, both the total amount of dental plaque and the specific microbial composition of plaque may contribute to the transition from health to disease
- The health-associated dental plaque is considered to be relatively stable overtime and in a state of dynamic equilibrium **(homeostasis)**
- Perturbations to the host response may be brought by the excessive accumulation of nonspecific dental plaque or by plaque-independent host factors such as changes in hormonal balance in pregnancy

Microbial flora which are associated with

1- Clinically healthy gingiva: If teeth are kept clean with proper oral hygiene measures, the gingiva remains healthy and few bacteria are found along the gingival margin

- In case of stop cleaning, bacteria will be accumulated on teeth within few hours
- The most predominant bacteria are streptococcus (G+ve cocci) and actinomyces (G+ve rods)
- In addition to small number of G-ve rods and facultative anaerobic rods

2. Gingivitis

a. Mild to moderate gingivitis for at least 2-3 months

- Streptococcus account is around 25% of the microbial flora of subgingival plaque *(Streptococcus mitis & Streptococcus sanguis)*
- Subgingival bacteria is composed of 25% Actinomyces. Another 25% are G-ve anaerobic rods such as, *fusobacterium, bacteroids* and *campylobacter*

b. Pregnancy gingivitis

- Increase in the proportion of *prevotella intermedia* (black pigmented *bacteriods species*)
- This increase is related to increased levels of estrogen and progesterone hormones in gingival fluid
- These hormones can be used by these organisms as growth factors

C. Acute necrotising ulcerative gingivitis

- The microflora is composed primarily of fusiform bacteria & spirochetes to form fusospirochetal complex
- These microorganisms are capable of invading the epithelium and the connective tissue of the gingiva

3. Chronic periodontitis

- The microflora is dominated by anaerobic microorganisms
- G-ve rods form about 75% such as, *bacteroids* and *fusobacterium nucleatum*
- *Spirochetes* form about 50% of the flora

4. Aggressive periodontitis

- Dominated by anaerobic G-ve rods which form about 60%
- Spirochetes form 7%
- *Aggregatibacter actinoycetem comitans* which was previously called *Actinobacillus actinomycetemcomitans*) *(A.A)* is found to be present in almost always in aggressive and less prevalent in chronic periodontitis

Experimental gingivitis

It is an investigation was carried out by (Leo et al 1965) to show the cause and effect relationship between dental plaque and gingival inflammation

- In this study the oral hygiene of a group of healthy individuals (12 patients, 9 dental students, 1 instructor and 2 laboratory technicians) were improved during several weeks of intensive instruction in the use of tooth brush and tooth picks, resulting in an excellent gingival condition
- Then the oral hygiene measures were withdrawn allowing plaque to reaccumulate along the gingival margin
- All subjects developed gingivitis within 10-21 days
- The mean gingival index score increased from 0.27 at the base line to 1.05 at the end of the no brushing period
- Gingival inflammation resolved in all subjects within 1 week of resuming hygiene measures
- During the experiment period, plaque samples were obtained at regular intervals and subjects to bacteriological examination of gram stained smear

In a health gingiva, very few bacteria were present on the cervical surfaces of teeth

- The removal deposit was dominated by desquamated epithelial cells between few bacteria
- About 90% of these bacteria were G+ve cocci and rods
- The remaining bacteria were G-ve

When all oral hygiene measures were stopped, the following phases of plaque development occurred:

First phase

- Initial 2 days of the experiment, not only all types of bacteria increase but their proportional distribution change as well
- G+ve cocci and rods forming a greater proportion of the flora

Second phase

- Day 3 and 4 are characterised by proliferation of *fuso*- and *filamentous* bacteria

Third phase

- Day 5-9 are characterised by appearance of *spirilla* and *spirochetes*

- After about 7 days, G+ve cocci and rods which initially predominated, now only form about 50% of the complex flora and up to 3 weeks , no further major changes in the bacterial distribution occur

- The experiment only gives information about G+ve and G-ve bacteria but not other types of bacterial species
- - However, it is a dependable experiment which proved clinically that plaque is the main etiological factor in the development of periodontal disease

Supra and sub-gingival calculus

- **Supra gingival calculus** is located coronal to the gingival margin and therefore it is visible in the oral cavity
- It is white to yellowish in colour, hard with claylike consistency and easily detected from the tooth surface
- After removal it may rapidly recur, especially in the lingual area of the mandibular incisors
- It's colour is influenced by contact with such substances as tobacco and food pigments
- It may localize on a single tooth or group of teeth or it may be generalised throughout the mouth
- The second most popular side of supragingival plaque is the buccal surface of maxillary molars
- Saliva from parotid gland flows over the facial surfaces of the upper molars via the parotid duct
- While submandibular and sublingual glands empty onto lingual surfaces of lower incisors via their ducts

Subgingival calculus is located below the crest of the marginal gingiva and therefore, it is not visible on routine clinical examination

- The location and extent of this calculus may be evaluated by careful tactile perception with a delicate periodontal probe
- It is hard, dense and frequently appears dark brown or greenish black in colour and firmly attached to the tooth surface
- Both supra and subgingival calculus can be seen by radiograph

Inorganic content: supragingival calculus consists of inorganic (70% to 90%)
- The major inorganic proportion of calculus is reported as approximately 76% calcium phosphate $Ca_3(po_4)$, 3% calcium carbonate, trace amount of magnesium phosphate $Mg_3(po_4)$ and other materials

- At least 2/3 of inorganic component is crystalline in structure
- There are four main crystal forms which are hydroxyapatite 58%, magnesium white-lockite 21% , octacalicium phosphate 12% and brushite 9%
- In general 2 or more crystal forms are typically found in the calculus sample of hydroxyapatite and **octacalcium phosphate** are detected most frequently in 97% of supragingival calculus

Brushite is more common → in the mandibular anterior region

Magnesium whitelockite is common → in the posterior area

Organic content consists of a mixture of protein-polysaccharide complexes, desquamated epithelial cells and leukocytes and various types of microorganisms

Attachment of calculus to the tooth

1- By means of an inorganic pellicle on cementum
2- Attachment on enamel
3- Mechanical locking into surface irregularities such as carious lesions
4- Close adaptation of the undersurface of calculus to depressions or gently sloping surfaces of the unaltered cementum surface and penetration by bacterial calculus into cementum
5- Penetration of calculus bacteria into cementum, calculus may embedded deeply in cementum may appear morphologically similar to cementum and thus termed calculocementum
6- The access to the area of calculus removal, subgingival area is more difficult as it is invisible

Formation of calculus

- Calculus is a dental plaque that has undergone mineralisation
- Plaque is hardened by precipitation of mineral salts which is usually started between day one and day fourteen of plaque formation
- The formation of dental calculus with mature crystalline composition of old calculus may require months to years
- Microorganisms are not always essential in calculus formation because it occurs readily in germ free rodents
- Saliva is the source of mineralisation for supragingival calculus
- Whereas, gingival crevicular fluid (GCF) is the source of subgingival calculus

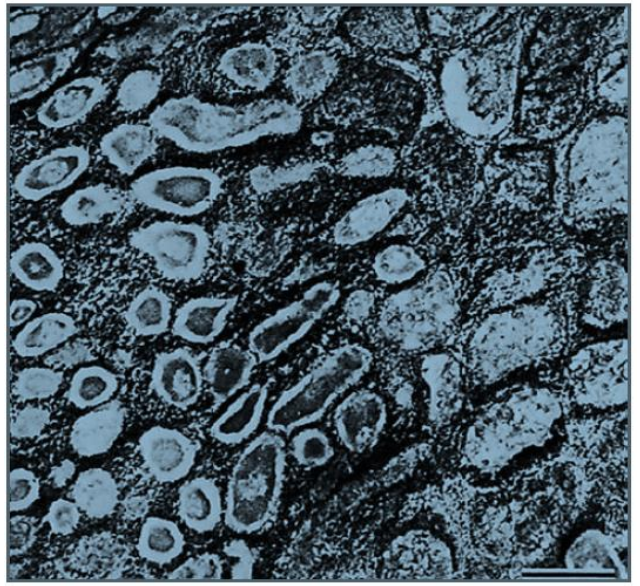


Fig. 9-8 Thin section of old mineralizing plaque. The intermicrobial matrix is totally calcified, and many microorganisms show intracellular crystal deposition. ×9500. Bar: 1 µm. (Source: Theilade 1964.)

- Crystals form initially in the intercellular matrix and on the bacterial surfaces and finally within the bacterial surfaces
- Calcification begins along the inner surface of the supra gingival plaque and in the attached component of subgingival plaque adjacent to the tooth
- The initiation and rate of accumulation vary among teeth in the same individual
- Calculus formation continues until it reaches maximum after which it reduced in amount due to mechanical wear from and cheeks, lip and tongue, also the use of anti-calculus (tartar) dentifrices would reduce both quality and quantity of calculus

Theories regarding the mineralisation of calculus

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

- A rise in pH of saliva causes precipitation of calcium phosphate salts. The pH may be elevated by the loss of carbon dioxide and formation of ammonia by dental plaque bacteria or by protein degradation during stagnation
- Colloidal proteins in saliva bind calcium and phosphate ions and maintain a supersaturated solution with respect to calcium phosphate salts, leading to its precipitation
- Phosphatase liberated from dental plaque, desquamated epithelial cells or bacteria precipitates calcium phosphate by hydrolysing organic phosphates in saliva, thus increasing the concentration of free phosphate ions

2- Seeding agents induce small foci of calcification that enlarge and coalesce to form a calcified mass

Role of microorganisms in mineralisation of calculus

- Mineralisation of plaque generally starts extracellular around both G+ve and G-ve organisms and may also start intracellular
- Filamentous organisms, *diphtheriods* and *bacterionema* and *veillonella* species have the ability to form intracellular apatite crystals
- Mineralisation spreads until the matrix and bacteria are calcified
- Bacterial plaque may actively participate in the mineralisation of calculus by forming phosphatases, which changes the pH of the plaque and induces mineralisation

Other predisposing factors

Iatrogenic factors

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

Margins of restorations

Overhanging margins of dental restorations contribute to the development of periodontal disease by
1- changing the ecologic balance of the gingival sulcus to an area that favours the growth of disease-associated organisms (pre-dominantly G-ve anaerobic species) at the expense of the health-associated organisms (predominantly G+ve facultative species)
2- Inhibiting the patient's access to remove accumulated plaque

Subgingival margins are associated with large amount of plaque, leading to severe gingivitis and deeper pockets, whereas **supragingival margins** are associated with a degree of periodontal health similar to that seen with non restored interproximal area

Contours/open contacts

Overcontoured crown and restorations tend to accumulate plaque and possibly prevent the self-cleansing mechanisms of the adjacent cheek, lips and tongue
- Restorations that fail to reestablish adequate interproximal embrasure spaces are associated with papillary inflammation

Design of removable partial dentures

Several investigations have shown that after the insertion of partial dentures, the mobility of the abutment teeth, gingival inflammation and periodontal pocket formation all increased

- This is because partial dentures favour the accumulation of plaque
- Partial dentures that are worn during both day and night induce more plaque formation than that worn only during daytime
- Thus it needs for careful and personalised oral hygiene instruction to avoid harmful effects of the partial denture

Lec. Dr. Nuha Hamed

Classification of periodontitis:

In 1999 the periodontitis were classified in to:

I. Chronic periodontitis:

Chronic periodontitis can be characterized by extent and severity. Extent is the number of the sites involved and can be described as localized or generalized. As a general guide, extent can be characterized as localized if ≤30% of the sites are affected and generalized if >30% of the sites are affected. Severity can be described for the entire dentition or for individual teeth and sites. As a general guide, severity can be categorized on the basis of the amount of clinical attachment loss (CAL) as follows:

Slight =1-2 mm CAL , moderate =3 - 4 mm CAL, and severe = ≥5 mm CAL.

The clinical features and characteristics of chronic periodontitis can be summarized as follows:

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

II. Aggressive periodontitis: (A.P.)

A. Localized (confined to molars and incisors)

B. Generalized

The term aggressive periodontitis replaced the previous name early-onset periodontitis (prepubertal, juvenile periodontitis & rapidly progressive periodontitis).

The common features of localized and generalized forms of aggressive periodontitis:

 Except for the presence of periodontitis, patients are otherwise clinically healthy;

 Rapid attachment loss and bone destruction;

 Familial aggregation;

 Amounts of microbial deposits are inconsistent with the severity of periodontal tissue destruction;

 Elevated proportion of aggregatibacter actinomycetemcomitans and, in some populations, porphyromonas gingivalis, may be elevated;

 Phagocyte abnormalities

 Progression of attachment loss and bone loss may be self-arresting.

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

1. Periodontitis.

2. Periodontitis as a direct manifestation of systemic diseases

3. Necrotizing periodontitis

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

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

Periodontitis groups are defined as (Tonetti et al., 2018):(1) Interdental CAL is detectable at ≥2 non-adjacent teeth, or (2) Buccal or oral CAL ≥3 mm with pocketing >3mm is detectable at ≥ 2teeth

Staging:

Stage I periodontitis: Stage I periodontitis is the borderland between gingivitis and periodontitis and represents the early stages of attachment loss, the severity of this stage associated with clinical attachment loss about 1-2mm and radiographic bone loss less than 15% in the coronal third, however, no tooth loss due to periodontitis associated with this stage .

Stage II periodontitis: represents established periodontitis. The severity of this stage associated with clinical attachment loss about 3-4mm and radiographic bone loss more than 15% and less than 33%in the coronal third.

Stage III periodontitis: This stage associated with clinical attachment loss ≥5mm and radiographic bone loss extending to the middle third of the roots.

Advanced stage IV periodontitis: This stage is characterized by the presence of deep periodontal lesions that extend to the apical portion of the root and/or history of multiple tooth loss. This stage associated with clinical attachment loss ≥5mm and radiographic bone loss extending to apical third of the roots.

Grades of periodontitis:

Irrespective of the stage at diagnosis, periodontitis may progress with different rates in individuals, may respond less predictably to treatment in some patients, and may or may not influence general health or systemic disease. Grading or rate of progression can be estimated by measurement of percentage of radiographical bone loss divided by the age of patient

Grade A periodontitis: is assigned if the maximum amount of radiographic bone loss in percentage terms is less than half the patient's age in years (for example, less than 30% in a 60-year-old or less than 40% in an 80-year-old)

Grade C periodontitis: is assigned if the maximum amount of bone loss in percentage terms exceeds the patient's age in years (for example, more than 30% in a 28-year-old or more than 50% in a 49-year-old)

Grade B periodontitis: all other situations

II. Periodontitis as a manifestation of systemic diseases :

A variety of systemic diseases and conditions can affect the course of periodontitis or have a negative impact on the periodontal attachment apparatus.

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

I. Genetic disorders

a. Diseases associated with immunologic disorders: like Down syndrome, Leukocyte adhesion deficiency syndromes, Papillon-Lefèvre syndrome

b. Diseases affecting the oral mucosa and gingival tissue: like Plasminogen deficiency

c. Diseases affecting the connective tissues: Ehlers-Danlos syndromes (types IV, VIII)

d. Metabolic and endocrine disorders: like Glycogen storage disease and Hypophosphatasia

II. Acquired immunodeficiency diseases : including Acquired neutropenia and HIV infection

III. Inflammatory diseases : Inflammatory bowel disease

2. Other systemic disorders that influence the pathogenesis of periodontal diseases

Diabetes mellitus ,Obesity ,Osteoporosis ,Arthritis (rheumatoid arthritis, osteoarthritis), Emotional stress and depression , Smoking (nicotine dependence)

3. Systemic disorders that can result in loss of periodontal tissues independent of periodontitis

a. Neoplasms:

-Primary neoplastic diseases of the periodontal tissues like Oral squamous cell carcinoma

-Secondary metastatic neoplasms of the periodontal tissues

b. Other disorders that may affect the periodontal tissues like Langerhans cell histiocytosis, Giant cell granulomas Hyperparathyroidism

III. Necrotizing periodontal diseases:

A. Necrotizing ulcerative gingivitis

B. Necrotizing ulcerative periodontitis

C. Necrotizing stomatitis

Necrotizing ulcerative gingivitis: This is an infection characterized by gingival necrosis presenting as 'punched-out' papillae, with gingival bleeding, and pain. Fetid breath and pseudomembrane formation may be secondary diagnostic features. Fusiform bacteria, prevotella intermedia, and spirochetes have been associated with gingival lesions. Predisposing factors may include: emotional stress, poor diet, cigarette smoking, and HIV infection.

Necrotizing ulcerative periodontitis: This is an infection characterized by necrosis of gingival tissues, periodontal ligament, and alveolar bone. These lesions are most commonly observed in individual with systemic conditions including HIV infection, severe malnutrition, and immunosuppression.

Necrotizing stomatitis : is a very severe and aggressive form of necrotizing periodontal disease showing extensive oral cavity tissue and bone destruction. In necrotizing stomatitis, after the oral mucosal membranes are destroyed, the entire mouth is involved due to spread of deep infection.

Other condition affecting the periodontium:

a. Periodontal abcesses and endodontic periodontal lesion

Periodontal abscesses (PA) :

Periodontal abscesses represented approximately 7.7–14.0% of all dental emergencies, being ranked the third most prevalent infection demanding emergency treatment, after dentoalveolar abscesses and pericoronitis.

I. Periodontal abscess in periodontitis patients In periodontitis patients, a PA could represent a period of disease exacerbation, favored by the existence of tortuous pockets, presence of furcation involvement or a vertical defect, in which the marginal closure of the pocket could lead to an extension of the infection into the surrounding periodontal tissues. In addition, changes in the composition of the subgingival microbiota, with an increase in bacterial

virulence, or a decrease in the host defense, could also result in an inefficient capacity to drain the increased suppuration. Different subgroups could be distinguished

a. Acute exacerbation:

○ In untreated periodontitis.

○ In “refractory” periodontitis.

b. After different treatments:

○ Scaling and root planing or professional prophylaxis

○Surgical periodontal therapy

○Systemic antimicrobial intake, without subgingival debridement

○Use of other drugs: e.g., nifedipine.

II. Periodontal abscess in non- periodontitis patients

(previously called gingival abscess), PA can also occur in previously healthy sites because of

• Impaction of foreign bodies: dental floss, orthodontic elastic, toothpick, rubber dam, or popcorn hulls.

• Harmful habits (biting wire, nail biting, clenching) could favor abscess formation because of subgingival impaction of foreign bodies or to coronal closure of the pocket.

• Orthodontic factors, such as inadequate orthodontic forces or a cross-bite, have been reported to favor PA development.

• Gingival enlargement.

• Alterations of the root surface, including:

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

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

○ Iatrogenic conditions, such as perforations.

○ Severe root damage: vertical root fracture or cracked tooth syndrome

extending through the root.

○ External root resorption.

PA may be associated with various combinations of the following clinical features: Pain, swelling, color change, tooth mobility, extrusion of teeth, purulence, sinus tract formation, fever, lymphadenopathy, and there may be a radiolucency of the affected alveolar bone.

The acute periodontal abscess characterized by slight discomfort to severe pain and swelling. Chronic periodontal abscess is usually a symptomatic or with dull pain with a history of intermittent exudate.

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

Periodontal abscess

Periapical abscess

1.

The tooth is vital.

Tooth is not vital.

2.

The lesion lateral to the root surface.

The lesion is most likely periapical.

3.

X-ray finding shows area of radiolucency along the lateral surface of the root.

X-ray finding shows apical radiolucency.

4.

The tooth is tender to lateral percussion.

Tooth tender to vertical percussion.

Endodontic periodontal lesions:

Are clinical conditions involving both the pulp and periodontal tissues and may occur in acute or chronic forms. When they are associated with a recent traumatic or iatrogenic event (e.g. root fracture or perforation), the most common manifestation is an abscess accompanied by pain. However, endo-

periodontal lesions, in subjects with periodontitis, normally present slow and chronic progression without evident symptoms. The most common signs and symptoms associated with a tooth affected by an endo-periodontal lesions are deep periodontal pockets reaching or close to the apex and negative or altered response to pulp vitality tests. The other signs and symptoms reported, in order of prevalence, are: bone resorption in the apical or furcation region, spontaneous pain or pain on palpation and percussion, purulent exudate, tooth mobility, sinus tract, crown, and gingival color alterations

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

(1) by a carious lesion that affects the pulp and, secondarily, affects the periodontium.

(2) by periodontal destruction that secondarily affects the root canal.

(3) or by both events concomitantly.

2. Endo-periodontal lesions associated with trauma and iatrogenic factors

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

(1) root/pulp chamber/furcation perforation (e.g. because of root canal instrumentation or to tooth preparation for post retained restorations)

(2) root fracture or cracking (e.g., because of trauma or tooth preparation for post-retained restorations)

(3) external root resorption (e.g., because of trauma)

(4) pulp necrosis (e.g., because of trauma) draining through the periodontium.

b. Mucogingival deformities or conditions around teeth:

1. gingival biotype

a. Thin scalloped

b. Thick scalloped

c. Thick flat

2. Gingival/soft tissue recession

a. Facial or lingual surfaces

b. Interproximal (papillary)

c. Severity of recession

d. Gingival thickness

e. Gingival width

3. Lack of keratinized gingiva

4. Decreased vestibular depth

5. Aberrant frenum/muscle position

6. Gingival excess

a. Pseudopocket

b. Inconsistent gingival margin

c. Excessive gingival display

d. Gingival enlargement

7. Abnormal color

Mucogingival: Term used to describe that portion of the oral mucosa that covers the alveolar process including the gingiva (keratinized tissue) and the adjacent alveolar mucosa.

Gingival biotype, which includes in its definition gingival thickness (GT) and keratinized tissue width (KTW);. A recent systematic review using the parameters reported previously, classified the “biotypes” in three categories:

• Thin scalloped biotype in which there is a greater association with slender triangular crown, subtle cervical convexity, interproximal contacts close to the incisal edge and a narrow zone of KT, clear thin delicate gingiva, and a relatively thin alveolar bone.

• Thick flat biotype showing more square-shaped tooth crowns, pronounced cervical convexity, large interproximal contact lo- cated more apically, a broad zone of KT, thick, fibrotic gingiva, and a comparatively thick alveolar bone.

• Thick scalloped biotype showing a thick fibrotic gingiva, slender teeth, narrow zone of KT, and a pronounced gingival scalloping.

Gingival recession: Is location of the gingival margin apical to the cemento-enamel junction.

The causes of gingival recession:

 Plaque accumulation will cause destruction of the junctional epithelia as a result of the inflammatory process.

 Traumatic gingival recession:

- Fault tooth brushing

- Tooth malposition

- High frenal attachment

- Overhanging fillings

- Prosthetic appliances

- Habits as nail biting.

c. Tooth and prosthetic related factors :

A. Localized tooth-related factors that modify or predispose to plaque-induced gingival diseases/periodontitis

1. Tooth anatomic factors

2. Root fractures

3. Cervical root resorption, cemental tears

4. Root proximity

5. Altered passive eruption

B. Localized dental prosthesis-related factors

1. Restoration margins placed within the supracrestal attached tissues

2. Clinical procedures related to the fabrication of indirect restorations

3. Hypersensitivity/toxicity reactions to dental materials.

Several conditions exist in teeth that may predispose the periodontium to disease. In certain cases these factors may contribute to the initiation of periodontal disease. While the etiology of periodontal disease is bacterial,

factors that enhance bacterial accumulation or allow ingress of bacteria into the periodontium should be considered in the classification of periodontal diseases.

d.Traumatic occlusal force

1. Primary occlusal trauma

2. Secondary occlusal trauma

3. Orthodontic force

Occlusal trauma: Injury resulting in tissue changes within the attachment apparatus as a result of occlusal force(s).

Primary occlusal trauma: Injury resulting in tissue changes from traumatic occlusal forces applied to tooth or teeth with normal support. It occurs in the presence of:

1) Normal bone levels, 2) Normal attachment levels, and 3) Excessive occlusal force(s).

Secondary occlusal trauma: Injury resulting in tissue changes from normal or traumatic occlusal forces applied to a tooth or teeth with reduced support. It occurs in the presence of:

1) Bone loss, 2) Attachment loss, And 3) "Normal"/excessive occlusal force(s). e.Peri-implant diseases and conditions 1. peri-implant health In health, the peri-implant site is characterized by absence of erythema, bleeding on probing, swelling and suppuration. 2. peri-implant mucositis: the diagnosis of peri-implant mucositis requires: Visual inspection demonstrating the presence of periimplant signs of inflammation: red as opposed to pink, swollen tissues as opposed to no swelling. Presence of profuse bleeding and/or suppuration on probing, an increase in probing depths compared to baseline; and absence of bone loss beyond crestal bone level changes resulting from the intial remodeling. 3. peri-implantitis: the diagnosis of peri-implantitis will involve radiographic bone loss associated with gingival recession or increased probing depth in addition to signs associated with peri-implant mucositis

Lec. Dental Stain Dr. Nuha Hamed

Pigmented deposits on the tooth surface are called dental stains.
The appearance of the dentition is of concern to a large number of people seeking dental treatment and the color of the teeth is of particular cosmetic importance.
There has been a recent increase in interest in the treatment of tooth staining and discolorations as shown by the large number of tooth whitening agents appearing on the market. Some of these agents are sold as 'over-the-counter' products and have no professional involvement in their application. The correct diagnosis for the cause of discoloration is important as, invariably, it has a profound effect on treatment outcomes. It would seem reasonable, therefore, that dental practitioners have an understanding of the etiology of tooth discoloration in order to make a diagnosis and enable the appropriate treatment to be carried out.

COLOUR AND COLOUR PERCEPTION

A basic understanding of the elements of tooth colour is important for many aspects of restorative dentistry. Teeth are typically composed of a number of colours and a gradation of color occurs in an individual tooth from the gingival margin to the incisal edge of the tooth. The gingival margin often has a darker appearance because of the close approximation of the dentine below the enamel. In most people canine teeth are darker than central and lateral incisors and younger people characteristically have lighter teeth, particularly in the primary dentition. Teeth become darker as a physiological age change; this may be partly caused by the laying down of secondary dentine,

incorporation of extrinsic stains and gradual wear of enamel allowing a greater influence on color of the underlying dentine.

CLASSIFICATION OF TOOTH DISCOLOURATION

The coronal portion of the tooth consists of enamel, dentine and pulp. Any change to these structures is likely to cause an alteration in the outward appearance of the tooth caused by its light transmitting and reflecting properties. The appearance of tooth color is dependent on the quality of the reflected light and is also, as a consequence, dependent on the incident light.

Historically, tooth discoloration has been classified according to the location of the stain, which may be either intrinsic or extrinsic. It may also be of merit to consider a further category of internalized stain or discoloration.

INTRINSIC DISCOLORATION

Intrinsic discoloration occurs following a change to the structural composition or thickness of the dental hard tissues. The normal color of teeth is determined by the blue, green and pink tints of the enamel and is reinforced by the yellow through to brown shades of dentine beneath. A number of metabolic diseases and systemic factors are known to affect the developing dentition and cause discoloration as a consequence. Local factors such as injury are also recognized.

1. Alkaptonuria
2. Congenital erythropoietic porphyria
3. Congenital hyperbilirubinaemia
4. Amelogenesis imperfecta
5. Dentinogenesis imperfecta
6. Tetracycline staining

7. Fluorosis

8. Enamel hypoplasia

9. Pulpal haemorrhagic products

10. Root resorption

11. Ageing

EXTRINSIC DISCOLORATION

Extrinsic discoloration is outside the tooth substance and lies on the tooth surface or in the acquired pellicle. The origin of the stain may be:

1. Metallic

2. Non-metallic

INTERNALIZED DISCOLORATION

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

1. Developmental defects

2. Acquired defects a) Tooth wear and gingival recession b) Dental caries c) Restorative materials

THE MECHANISMS OF TOOTH DISCOLORATION

INTRINSIC TOOTH DISCOLORATION

The formation of intrinsically discolored teeth occurs during tooth development and results in an alteration of the light transmitting properties of the tooth structure. There are a number of metabolic disorders which affect the dentition during its formation, unlike the inherited disorders in which only the hard tissue forming at the time may be involved. These disorders will now be discussed individually.

1. Alkaptonuria: This inborn error of metabolism results in incomplete metabolism of tyrosine and phenylalanine, which promotes the buildup of homogentisic acid. This affects the permanent dentition by causing a brown discolouration.

2. Congenital erythropoietic porphyria: This is a rare, recessive, autosomal, metabolic disorder in which there is an error in porphyrin metabolism leading to the accumulation of porphyrins in bone marrow, red blood cells, urine, faeces and teeth. A red-brown discolouration of the teeth is the result and the affected teeth show a red fluorescence under ultra-violet light. King George III was said to have suffered with acute intermittent porphyria but with the later onset of this disorder his teeth are unlikely to have been affected

3. Congenital hyperbilirubinaemia: The breakdown products of haemolysis will cause a yellow-green discolouration. Mild neonatal jaundice is relatively common, but in rhesus incompatibility massive haemolysis will lead to deposition of bile pigments in the calcifying dental hard tissues, particularly at the neonatal line.

4. Amelogenesis imperfecta: In this hereditary condition, enamel formation is disturbed with regard to mineralization or matrix formation and is classified accordingly. There are 14 different subtypes; the majority is inherited as an autosomal dominant or xlinked trait with varying degrees of expressivity. The appearance depends upon the type of amelogenesis imperfecta, varying from the relatively mild hypomature 'snow-capped' enamel to the more severe hereditary hypoplasia with thin, hard enamel which has a yellow to yellow-brown appearance

5. Systemic syndromes: Defects in enamel formation may also occur in

a number of systemically involved clinical syndromes such as Vitamin D dependent rickets, epidermolysis bullosa and pseudohypoparathyroidism. It had been reported areas of hypoplastic enamel, irregularities in the region of the amelo-dentinal and the cemento-dentinal junctions in Ehlers-Danlos Syndrome. In epidermolysis bullosa there is pitting of the enamel possibly caused by vesiculation of the ameloblast layer. However, the effect of these conditions depends on disease activity during the development of the dentition and is usually a minor element.

6. Dentinogenesis imperfecta: Dentine defects may occur genetically or through environmental influences. The genetically determined dentine defects may be in isolation or associated with a systemic disorder. The main condition related to the dentine alone is Dentinogenesis imperfecta II (hereditary opalescent dentine). Both dentitions are affected, the primary dentition usually more severely so. The teeth are usually bluish or brown in color, and demonstrate opalescence on transillumination. The pulp chambers often become obliterated and the dentine undergoes rapid wear, once the enamel has chipped away, to expose the amelo-dentinal junction. Once the dentine is exposed, teeth rapidly show brown discoloration, presumably by absorption of chromogens into the porous dentine Dentinogenesis imperfecta I (associated with osteogenesis imperfecta, a mixed connective tissue disorder of type I collagen) may show bone fragility and deformity with blue sclera, lax joints and opalescent dentine. The inheritance may be dominant or recessive, the recessive being more severe and often fatal in early life. Opalescent teeth are more common in the dominant inheritance

pattern, the primary teeth bear a strong resemblance to the teeth in Dentinogenesis imperfecta type I whereas the appearance of the secondary dentition is much more variable. The enamel is much less prone to fracture, the pulp chamber is seldom occluded by dentine (this may help to radiographically differentiate between types I and II), and the overall prognosis for the dentition is improved.

A third type of Dentinogenesis imperfecta (type III, brandywine isolate hereditary opalescent dentine) was described by Wiktop.In this condition, the teeth may be outwardly similar to both types I and II of Dentinogenesis imperfecta; however, multiple pulpal exposures occur in the primary dentition. Radiographically, the teeth may take on the appearance of 'shell teeth' as dentine production ceases after the mantle layer has formed. This type of Dentinogenesis imperfecta is thought to be related more closely to type II.

7. Dentinal dysplasias: Shields reclassified the inherited dentine defects in a review of the literature in 1973 and introduced the term dentinal dysplasias. This reclassification allows separation of the inherited types of dentine defects from Dentinogenesis imperfecta, with which they are often confused.

In type I dentine dysplasia the primary and secondary dentition are of normal shape and form but may have an amber translucency. Radiographically the teeth have short roots with conical apical constrictions. The pulp is commonly obliterated in the primary dentition, leaving only a crescentic pulpal remnant in the adult dentition parallel to the cemento-enamel junction. There are characteristic periapical radiolucencies in many, otherwise healthy, teeth. The condition is inherited as an autosomal dominant trait.

Type II dentine dysplasia is described through a small number of case reports in Shields review, the main characteristic is that of a thistleshaped pulp chamber with numerous pulp stones. The teeth had brown discoloration.

8. Tetracycline staining: Systemic administration of tetracyclines during development is associated with deposition of tetracycline within bone and the dental hard tissues. Tetracycline and its homologues have the ability to form complexes with calcium ions on the surface of hydroxy apatite crystals within bone and dental tissues. Dentine has been shown to be more heavily stained than enamel. Tetracycline is able to cross the placental barrier and should be avoided from 29 weeks *in uterus* until full term to prevent incorporation into the dental tissues. Since the permanent teeth continue to develop in the infant and young child until 12 years of age, tetracycline administration should be avoided in children below this age and in breast-feeding and expectant mothers. The color changes involved depend upon the precise medication used, the dosage and the period of time over which the medication was given. Teeth affected by tetracycline have a yellowish or brown-grey appearance which is worse on eruption and diminishes with time. Exposure to light changes the color to brown, the anterior teeth are particularly susceptible to light induced color changes. The various analogues of tetracycline produce different color changes, for instance chlortetracycline produces a slate grey color and oxy-tetracycline causes a creamy discoloration. Since tetracycline fluoresces under ultraviolet light so do affected teeth, giving off a bright yellow color. There have been recent reports of adults experiencing change in tooth

colour with the use of long term tetracycline therapy. Minocycline, a synthetic compound of tetracycline antibiotics, is also implicated in causing discoloration in an adult patient, following its long term use for treatment of acne. This phenomenon was described in a single case report in the literature by Cale *et al*. When the appearance of the dentine had altered following the long-term use of minocycline for acne, it was postulated that calcium-minocycline complexes were deposited in the dentine

9. Fluorosis: The association between fluoride intake and its effect on enamel was noted by Dean as long ago as 1932.This may arise endemically from naturally occurring water supplies or from fluoride delivered in mouth rinses, tablets or toothpastes as a supplement. The severity is related to age and dose, with the primary and secondary dentitions both being affected in endemic fluorosis. The enamel is often affected and may vary from areas of flecking to diffuse opacious mottling, whilst the color of the enamel ranges from chalky white to a dark brown/black appearance. The brown/black discoloration is post-eruptive and probably caused by the internalization of extrinsic stain into the porous enamel.

These features are often described as being pathognomonic of fluorosis, but care should be taken not to confuse the condition with the hypomaturation type of ameolgenesis imperfecta. Fluoride only causes fluorosis in concentrations of greater than 1 ppm in drinking water and is not distinguishable, clinically or histologically, from any other type of hypo plastic or hypo mineralized enamel.

10. Enamel hypoplasia: This condition may be localized or generalized. The most common localized cause of enamel hypoplasia is likely to

occur following trauma or infection in the primary dentition.Such localized damage to the tooth-germ will often produce a hypoplastic enamel defect, which can be related chronologically to the injury. Disturbance of the developing tooth germ may occur in a large number of fetal or maternal conditions eg maternal vitamin D deficiencies, rubella infection, drug intake during pregnancy and in pediatric hypocalcaemic conditions. Such defects will be chronologically laid down in the teeth depending on the state of development at the time of interference; the effect is directly related to the degree of systemic upset. There may be pitting or grooving which predisposes to extrinsic staining of the enamel in the region of tooth disturbed, often then becoming internalized.

11. Pulpal hemorrhagic products: The discoloration of teeth following severe trauma was considered to be caused by pulpal hemorrhage. Haemolysis of the red blood cells would follow and release the hem group to combine with the putrefying pulpal tissue to form black iron sulphide. Grossman asserted in 1943 that the depth of dentinal penetration determines the degree of discolouration; there was little if any scientific investigation of this hypothesis. *In vitro* studies have recently shown that the major cause of discoloration of non-infected traumatized teeth is the accumulation of the hemoglobin molecule or other haematin molecules. In the absence of infection, the release of iron from the protoporphyrin ring is unlikely. This greater understanding of the nature of tooth staining following trauma to teeth may be of importance if the manufacture of bleaching agents, with specific activity, becomes possible. For instance, with further analysis it may become possible to develop a bleaching agent for use

on teeth stained specifically by blood pigments. Incidentally, it has been shown that the pinkish hue seen initially after trauma may disappear in 2 to 3 months if the tooth becomes revascularized.

12. Root resorption: Root resorption is often clinically asymptomatic; however, occasionally the initial presenting feature is a pink appearance at the amelo-cemental junction. Root resorption always begins at the root surface, either from the pulpal or periodontal aspect, as internal or external root resorption respectively. It can be difficult to locate a resorptive cavity on radiograph until it reaches a certain size

13. Ageing: The natural laying down of secondary dentine affects the light-transmitting properties of teeth resulting in a gradual darkening of teeth with age.

EXTRINSIC DISCOLORATION

The causes of extrinsic staining can be divided into two categories; those compounds which are incorporated into the pellicle and produce a stain as a result of their basic color, and those which lead to staining caused by chemical interaction at the tooth surface.

Direct staining has a multi-factorial etiology with chromogens derived from dietary sources or habitually placed in the mouth. These organic chromogens are taken up by the pellicle and the color imparted is determined by the natural color of the chromogen. Tobacco smoking and chewing are known to cause staining, as are particular beverages such as tea and coffee. The color seen on the tooth is thought to be derived from polyphenolic compounds which provide the color in food.

Indirect extrinsic tooth staining is associated with cationic antiseptics

and metal salts. The agent is without color or a different color from the stain produced on the tooth surface. Extrinsic tooth discolouration has usually been classified according to its origin, whether metallic or non-metallic.

Non-metallic stains: The non-metallic extrinsic stains are adsorbed onto tooth surface deposits such as plaque or the acquired pellicle. The possible etiological agents include dietary components, beverages, tobacco, mouth rinses and other medicaments. Chromogenic bacteria have been cited in children. Particular colors of staining are said to be associated with certain mouths, for instance, green stain caused by penecillium and Aspergillus species, orange in children with poor oral hygiene and black/brown stains in children with good oral hygiene and low caries caused by Actenomyces species.

The most convincing evidence for the extrinsic method of tooth staining comes from the differing amount of stain found in a comparison of smokers and non-smokers. The staining effect of prolonged rinsing with chlorhexidine mouth rinses and quaternary ammonium compounds used in mouthrinses is of considerable interest to the dental profession.

Metallic stains: Extrinsic staining of teeth may be associated with occupational exposure to metallic salts and with a number of medicines containing metal salts. The characteristic black staining of teeth in people using iron supplements and iron foundry workers is well documented. Copper causes a green stain in mouth rinses containing copper salts and in workers in contact with the metal in industrial circumstances. A number of other metals have associated

colours such as potassium permangenate producing a violet to black colour when used in mouth rinses; silver nitrate salt used in dentistry causes a grey colour, and stannous fluoride causes a golden brown discolouration.It was previously thought that the mechanism of stain production was related to the production of the sulphide salt of the particular metal involved. This is perhaps not surprising since the extrinsic stain coincided with the colour of the sulphide of the metal concerned. However, those proposing the hypothesis appeared not to consider the complexity of the chemical process necessary to produce a metal sulphide.

chlorhexidine or iron sulphate followed by tea rinse produced immediately the characteristic brown and black discoloration of the teeth and tongue reported for chlorhexidine and iron respectively.

INTERNALIZED DISCOLORATION

The stains taken up into the body of enamel or dentine are the same as that causing extrinsic tooth discolouration, including in particular dietary chromogens and the by-products of tobacco smoking. Dental defects permitting the entry of chromogenic material can be classified under the headings of 'developmental and acquired'.

1. Developmental defects: The most important defects are considered under the 'intrinsic tooth discoloration' section of this review. As described these developmental defects create their own colour change in the tooth caused by influences on light transmission through the dentine and enamel. Post-eruptively, however, either caused by increased enamel porosity, or the presence of enamel defects, extrinsic stains can penetrate into the enamel. Such examples would include fluorosis and other enamel conditions resulting in enamel

hypoplasia or hypocalcification. Alternatively, developmental defects may expose dentine either directly or later caused by early loss of enamel as in dentinogenesis imperfecta. Chromogens are then able to enter the dentine directly or facilitated almost certainly by the tubule system.

2. Acquired defects: Wear and tear and disease of the teeth and supporting tissues occur throughout life, all of which can lead directly or indirectly to tooth discolouration. Additionally, repairs on restorations of teeth can influence the colour of teeth.

a) Tooth wear and gingival recession: Both conditions appear to have multifactorial etiologies but to date are poorly understood, there being limited research on the topics. Tooth wear is usually considered to be a progressive loss of enamel and dentine due to erosion, abrasion and attrition. As enamel thins the teeth become darker as the color of dentine becomes more apparent. Once dentine is exposed the potential of chromogens to enter the body of the tooth is increased. Physical trauma can also result in bulk loss of enamel or enamel cracks, both of which facilitate internalisation of extrinsic stains. Although tooth wear occurs at the cervical area of teeth, where enamel is most thin, exposure of dentine is more likely caused by gingival recession. Again, the net result is dentine exposure and the increased potential for the uptake of chromogens into the tooth.

b) Dental caries: The various stages of the carious process can be recognized by changes in colour as the disease progresses. For instance, the initial lesion is characterized by an opaque, white spot. The white spot lesion differs in colour from the adjacent enamel by virtue of its increased porosity and the effect this has on the refractive index. Enamel has a

Refractive Index of 1.62, compared with 1.33 for water and 1.0 for air. Air-drying removes water from the pores in partially dematerialized enamel leaving air and makes the 'white spot lesion' conspicuous by the alteration in its light transmitting properties. The hard, arrested lesion is black having picked up stain from exogenous sources. Early investigation into the change in color with the carious process centered around the amino-acids released during proteolysis, as a result of the proteolysischelation theory of cavity formation.

c) Restorative materials including amalgam: Some of the materials used in restorative dental treatment may have an effect on the color of teeth. Eugenol and phenolic compounds used during root canal therapy contain pigments which may stain dentine. Some of the poly antibiotic pastes used as root canal medicaments may cause a darkening of the root dentine. Clinicians are familiar with the dark grey to black colour of dentine following the removal of a long-standing amalgam restoration. It was previously thought that mercury was penetrating the dentinal tubules and reacting with sulphide ions. Electron microscopic studies have shown that this discoloration is caused by the migration of tin into the tubules.

HOW CAN WE PREVENT TEETH DISCOLORATION?

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

WHAT TREATMENT OPTIONS ARE AVAILABLE TO WHITEN TEETH?

Dental treatment of tooth discoloration involves identifying the etiology and implementing therapy. Medical treatment also may be warranted, depending on the etiology of the tooth discoloration.

・Diet and habits: Extrinsic staining caused by foods, beverages, or habits (e.g., smoking, chewing tobacco) is treated with a thorough dental prophylaxis and cessation of dietary or other contributory habits to prevent further staining.

・Tooth brushing: Effective tooth brushing twice a day with a dentifrice helps to prevent extrinsic staining. Most dentifrices contain an abrasive, a detergent, and an anti tartar agent. In addition, some dentifrices now contain tooth-whitening agents.

・Professional tooth cleaning: Some extrinsic stains may be removed with ultrasonic cleaning, rotary polishing with an abrasive prophylactic paste, or air-jet polishing with an abrasive powder. However, these modalities can lead to enamel removal; therefore, their repeated use is undesirable.

・Enamel microabrasion: This technique involves the rotary application of a mixture of weak hydrochloric acid and silicon carbide particles in a water-soluble paste. The resultant surface is smooth and has a glazed appearance. Enamel microabrasion is indicated for the removal of superficial intrinsic tooth discoloration, including that caused by fluorosis and decalcifications secondary to orthodontic brackets or bands. Enamel microabrasion may be used in conjunction with bleaching.

・Bleaching (tooth whitening): Early bleaching techniques were developed almost a century ago, and all of the techniques involved a process of oxidation. Today, with proper patient selection, bleaching

is a safe, easy, and inexpensive modality that is used to treat many types of tooth discoloration. Usually, bleaching is not indicated for the treatment of discoloration of the primary teeth. Bleaching includes 2 types of techniques: vital and nonvital.

oVital bleaching

・Bleaching of vital teeth is indicated primarily for patients with generalized yellow, orange, or light brown extrinsic discoloration (including chlorhexidine staining), although it may be helpful in ameliorating mild cases of tetracycline-induced intrinsic discoloration and fluorosis.

・Currently, the bleaching agents most commonly used are carbamide and hydrogen peroxide.When applied in higher concentrations, the agents produce more significant bleaching than they do without these measures.

・In office "power" bleaching involves the use of a 15-40% hydrogen peroxide solution and must be performed by a dental professional because careful isolation of the teeth is required to protect the soft tissues from the caustic effects of the bleaching agent.

・The use of home bleaching systems is currently popular; they may be used alone or in combination with in-office bleaching. The systems must be used under the careful supervision of dentists or dental hygienists. Patients apply a 10-22% carbamide peroxide solution into a custom-made mouth guard. After repeated daily and/or nightly (often while patients sleep) applications for 2-6 weeks, the teeth are gradually bleached. Whitening strips, using a 5.3% hydrogen peroxide–impregnated polyethylene strip, offer an at-home alternative to the above methods and can be recommended for

maintaining already whitened teeth.

· Whitening toothpastes, containing 1% or less peroxide, are minimally effective.

· With darker stains, the best results are achieved by using a combination of office and home bleaching systems. Most patients also require periodic re-treatment.

· Clinicians should be aware of potential adverse reactions and contraindications for bleaching. Approximately two thirds of patients have short-term, minor tooth sensitivity to cold and/or gingival irritation. Tooth surfaces, particularly exposed roots or enamel surfaces with defects secondary to incomplete amelogenesis, are porous to the bleaching agent and are more likely to develop cold sensitivity. Gingival irritation usually is related to improper fitting of the custom-made mouthguard.

· Allergic reactions to the bleaching agent are exceedingly rare.

· No adverse reactions are documented in pregnant or breastfeeding women or in patients who smoke; however, bleaching is not advised in these patients.

oNon vital bleaching

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

· For non vital bleaching, a tooth with an un restored crown is ideal.

Periodontics

Phase II Surgical Therapy

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

1-Controlling or eliminating periodontal disease (surgical pocket therapy)

2-Correcting anatomic conditions that favor periodontal disease,impair aesthetics or impede placement of prosthetic appliances (plastic surgery,aesthetic surgery,pre prosthetic techniques)

3-Placing implants to replace lost teeth and improving environment for their placement and function

Periodontal surgery

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

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

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

Objectives of periodontal surgery

1-Accessibility and direct vision for proper S+ RP

2-Reduction or elimination of plaque retentive area especially periodontal pockets that have not responded to initial therapy.

3-Eliminate inflamed periodontal tissue

4-Enhancing the regeneration of periodontal tissue

5-Create a 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

Surgical treatment include

1-Gingivectomy for the removal of the over growth gingival tissues

2-Flap surgery

3-Distal wedge procedure

4-Mucogingival surgery for correction of mucogingival and aesthetic defect

5-Crown lengthening to increase clinical crown length

6-Guided tissue regeneration (GTR) to regenerate periodontal supporting structures

Gingivectomy

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

Indication

1-Gingival enlargement or over growth

2-Idiopathic gingival fibromatosis.

3-Shallow suprabony pocket

4-Minor corrective procedure

Contraindication

1-Infrabony pocket

2-Thickening of marginal alveolar bone and the need for bone surgery

3-Attached gingiva is narrow or absent

Advantage

1-Technically simple, good visual access

2-Complete pocket elimination

3-Restoration of a physiologic gingival contour

Disadvantage

1-Gross wound, post operative pain

2-Healing by secondary intention

3-Danger of exposing bone

4-Loss of attached gingiva

5-phonetics and aesthetic problem in the anterior area with sensitivity due to exposure of the cervical area of tooth

Goldman gingivectomy procedure

-giving local anesthesia

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

Primary beveled incision which carried out 1 mm apical to bleeding points by Kirkland knife.

Continuous incision or interrupted , straight or scalloped is made.

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

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

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

Use Kirklaned knife for gingivoplasty(minor alterations in gingival

morphology without tissue excision)by shaving wound margin to create thin margin.

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

Flap surgery

Indications

1-In treatment of infrabony pockets

2-When the gingivectomy will lead to an unacceptable aesthetic results

3-Osseous recontouring (elimination of bony defect)

The Modified Widman flap Advantages

1-good access to root surface to facilitate S+ RP as well as the removal of the pocket epithelium and the inflamed connective

tissue.

2-width of keratinized gingiva is maintained

3-replacement of the flap at presurgical location leads to less exposure of the root surfaces thus minimizes problem of

aesthetic (especially anteriorly) and root hypersensitivity.

4-cause minimal amount of trauma to the periodontal tissues and discomfort to the patient.

5-the possibility of obtaining a close adaptation of the soft tissues to the root surfaces.

6-provides better access to re-establish proper contour of the alveolar bone as well as the potential for bone regeneration in

sites with angular bony defect.

7-furcation areas can be exposed.

Following flap procedures and the removal of plaque, calculus and chronically

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

Blades

Periosteal elevator

Hemostatic forceps, tissue forceps

Needle holder

Suture

Three incisions of Modified widman flap

Modified widman flap ; reported in 1974 by Ramfjod and Nissle,it is a replaced flap.There are three incisions in this flap,it is usually conducted as following:

Primary incision:

a:First incision-scalloping

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

Flap retraction:

b:Flap reflection

An elevator is used to raise a full thickness mucoperiosteal flap as atraumatically as possible.The flap is reflected only to permit direct

visualization of the root surface and the alveolar crest.In most cases it is possible

to stay within the boundaries of the attached gingiva,without extending beyond the mucogingival line.

Secondary incision:

C:Second incision-crevicular

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

Third incision:

d:Third incision-horizontal

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

Direct root planing:

e:Root planing with direct vision

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

Suturing:

f:Complete coverage of interdental defects

The labial and palatal flaps are closed over the interdental areas without tension,using interrupted sutures.The flaps should be adapted to the underlying bond and the necks of the teeth.New papillae where created by the scalloped

form of the initial incision.These make it possible to cover interdental defects(e.g.bony defects)even when the interdental space is wide.For this reason,placement of a periodontal dressing is not absolutely necessary.

Periodontics

Mucogingival surgery

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

Grafting procedures include

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

Free gingival graft

Free gingival graft

Sub epithelial connective tissue graft

Sub epithelial connective tissue graft

Lateral repositioned graft

Lateral repositioned graft

Bone graft (GBR)

GTR + GBR graft

Guided tissue regeneration GTR

Following periodontal surgery, the instrumented root surface is

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

Crown lengthening

Indication

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

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

3-Sub gingival caries

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

5-Aesthetic improvement of anterior teeth with short clinical crowns

and high lip line

Distal wedge procedures

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

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

Techniques for the removal of the frenum

A frenum is a fold of mucous membrane, usually with enclosed muscle fibers, that attaches the lips and cheeks to the alveolar mucosa and/or gingiva and underlying periosteum.A frenum becomes a problem if the attachment is too close to the marginal gingiva.Tension on the frenum

may pull the gingival margin away from the tooth.This condition may be conducive to plaque accumulation and inhibit proper brushing of the teeth with pocket formation.Also may tend to open the sulcus and gingival recession.

Frenectomy or Frenotomy

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

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

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

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

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

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

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

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

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

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

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

8.Cover the area with periodontal pack.

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

Periodontal dressing: are mainly used :

I· To protect the wound post surgically

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

3-For the comfort of the patient

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

5-Prevent the formation of excessive granulation tissue

Periodontal dressing should have the following properties:

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

2-Should harden within a reasonable time

3-After setting should be sufficiently rigid Io prevent fracture and dislocation.

4-Should have a smooth surface after setting to prevent irritation to the check and lips

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

6-Not detrimentally interfere with healing

Types of dressing

I-Zinc-oxide eugenol pack: eugenol in this type may induce an allergic reaction

2-Non eugenol pack: e.g. Coe pack; one tube contain zinc oxide and lorothidol (Fungicidal) and the second tube contain non ionizing carboxilic acids and chlorothymol (bacteriostatic agent)

3-Light cured dressing

Maintenance phase(supportive periodontal therapy SPT)

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

The primary goal of maintenance therapy include

I-Maintenance of oral health (cancer screening)

2-Prevention of new infection

3-Prevention of re-infection and disease recurrence

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

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

year

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

There are three parts to an SPT appointment:

1.examination

2.treatment

3.report,clean up and scheduling

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

Recurrence of Periodontal Disease

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

Other causes for recurrence include the following:

1.Inadequate or insufficient treatment that has failed to remove all of the potential factors favoring biofilm accumulation. Incomplete calculus removal in areas of difficult access is a common source of problems.

2. Inadequate restorations placed after the periodontal treatment was completed.

3. Failure of the patient to return for periodic maintenance care . This may be a result of the patient’s conscious or unconscious decision not to continue treatment or the failure of the dentist and staff to

emphasize the need for periodic supportive therapy.

4. Presence of some systemic diseases that may affect host resistance to previously acceptable levels of biofilm.

A failing case can be recognized by the following:

1. Recurring inflammation revealed by gingival changes and bleeding of the sulcus on probing.

2. Increasing depth of sulci, leading to the recurrence of pocket formation.

3. Gradual increases in bone loss, as determined by radiographs.

4. Gradual increases in tooth mobility, as ascertained by clinical examination.

The decision to retreat a periodontal patient should not be made at the preventive maintenance appointment but should be postponed for 1 to 2 weeks. Often, the mouth appears improved at that time because of the resolution of edema and the resulting improved tone of the gingiva.

Table summarizes the symptoms of the recurrence of periodontal disease and their probable causes.

Chemical plaque control

The action of these chemical could fit into four categories:

1. Anti_adhesive.
2. Antimicromicrobial.
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 chlohexidine 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 abroad 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 prolong 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. Sever necrotizing ulcerative gingivitis.
2. Multiple o sever periodontal abscesses with involvement of regional lymph nodes.
3. Some cases of aggressive periodontitis.
4. Refractory periodontitis.

Rout of administration:

It is impossible to mechanically remove plaque completely from narrow grooves, narrow furcation's 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 can invade periodontal tissues, making mechanical therapy alone in-effective.

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 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 microorganisims.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 time 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 actinomycetem comitance (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 indicted 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 create 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 prophyromonas 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).

Efficacies 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.

Nonsteriodal 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.

Lec. 15

د.نهى عكاب

Etiology and Risk factors of Periodontal diseases

- Periodontal diseases: are the most prevalent and multifactorial diseases that involved hard and soft dental tissues .Plaque, being the primary etiologic agent. Gingivitis & periodontitis, are the two basic form of periodontal disease.

- **Risk factors may be modifiable or non-modifiable**

Modifiable risk factors are:

a. Specific microorganisms
b. Tobacco Smoking
c. Diabetes Mellitus
d. Psychological factors
e. Obesity
f. Socioeconomic status
g. Pregnancy, puberty
h. Medications

a. Specific microorganisms:

- Aggregatibacter actinomycetemcomitans(*A.a*).
- Porphyromonas gingivalis (*P.gingivalis*).
- Prevotella intermedia (*P.intermedia*).
- Tannerella forsythia (*T.Forsythia*).

b. Tobacco Smoking: increase 2.5-7 times greater than nonsmoker.

The smokers appear have less gingival inflammation & less bleeding in the gingiva may explained by decreased gingival vascularity, which includes decreased vascular density, reduced lumen area of gingival vessels (increased vasoconstriction). Studies suggested that nicotin increases rate of proliferation of gingival epithelium which can contribute to the reduction of inflammatory clinical signs in the gingival tissues. These are the physiological effects of smoking on the etiology of periodontal disease. With decreased gingival crevicular fluid flow.

The microbiological effect of smoking:

- Smoker may have higher level of Tannerella forsythia, P.gingivalis & Treponema denticola. It has been found that smoke derived aryl hydrocarbons & bacterial Lipopolysaccharides (LPS) may act additively to inhibit bone formation, which may explain why periodontal bone loss is greater &bone healing is less successful in smokers than nonsmokers with periodontal infections.

The immunological effect of smoking :

1. Nicotine causes decrease immune response & impair PMNs chamotaxis &phagocytosis.

2. Increase the production of TNF-alpha, IL-1 Alfa & IL6 these immune mediator are known to lead to more sever destructive inflammation in the periodontal tissue.

3. Reduction in the serum concentration of immunoglobulin as IgG2 which is essential in the protection against periodontal infection.

Also smoking decrease the level of salivary IgA antibodies.

c. Diabetes mellitus: does not cause gingivitis or periodontal pockets, but it alters the response of periodontal tissues to local factor. Diabetic patients with poor oral hygiene may have very sever gingival inflammation, deep periodontal pockets, rapid bone loss &frequent periodontal abscesses .

It is likely to be related an increased susceptibility to infection , an impaired immune response ,poor wound healing &increase collagenase activity.

d. Psychological factors:

- Studies have demonstrated that under psychological stress are more likely to develop clinical attachment loss and loss of alveolar bone.
- ↑glucocorticoid secretion results in ↓immune function,↑insulin resistance &increases in production of IL-6.

e. Obesity:

- One of the most significant health risks of modern society. Obesity reduce blood flow to the periodontal tissues promoting the development of periodontal disease.

f. Socioeconomic status:

People who are better educated, wealthier and live in more desirable circumstances enjoy better health status than the less educated and poor society.

g. Pregnancy, puberty &menopause (hormonal);

- Pregnancy gingivitis is inflammation accompanied by increase in steroid hormones in crevicular fluid &increase in levels of (Prevotella intermedia microorganism) which use the steroid as growth factors. The increase in sex hormones may exaggerate the inflammatory response to dental plaque which means small amount of plaque may lead to gingivitis. During menopause, estrogen deficiency will reduce bone mineral density some women may develop menopausal gingivostomatitis.

h. Medications:

- anticonvulsants (phenytoin or Dilantin), immunosuppressant (cyclosporine) & Ca channel blockers (antihypertensive drugs).
- the uses of drug for along period of time (duration) which may result in recurrence of the lesion even if it is treated surgically.

Non-Modifiable risk factors are:

a. Hematological Disorders
b. Genetic factors
c. Aging
d. Sex
e. Ethnicity
f. HIV/AIDS
g. Osteoporosis

a. Hematological Disorders

- Hemorrhagic gingival overgrowth with or without necrosis is a common early manifestation of acute leukemia. Patients with chronic leukemia may experience similar but less sever periodontal changes.

b. Genetic factors

- Genetic factors may play an important role in determining the nature of the host response & may affect the function of phagocytic immune cells or the structure of the epithelia or connective tissue. One of these disease is papillon _lefevere syndrome which is a rare hereditary disease characterized by hyperkeratotic skin lesion in the palms, soles, knees, elbows & sever destruction to the periodontium with early loss of primary & permanent teeth.

c. Aging: with aging a number of changes take place in the periodontal tissues.

a. Arteriosclerosis (reduction in arterial blood supply).

b. the gingiva become more fibrotic &less keratinized.

c. the periodontal fiber bundles become thicker with decrease in cellularity.

d. Osteoporosis of alveolar bone.

Age by itself no influence on the periodontal tissues

d. Sex

- Studies reported higher periodontal destruction among males compared to female population. The reasons for these sex differences are not clear, but they are thought to be related to the ignorance of oral hygiene, which is usually observed among males.

e. Ethnicity

- Certain racial /ethnic groups, particularly subjects of African and Latin American background, have a higher risk of developing periodontal tissue loss than other groups.

f. HIV/AIDS

- It has been stated that the immune dysfunction (immunosuppression) associated with HIV infection & AIDS increased susceptibility to periodontal disease, they often had sever periodontal destruction characterized by necrotizing ulcerative periodontitis.

g. Osteoporosis

- postmenopausal osteoporosis may result in dental osteopenia involving the jaws, particularly the mandible.

Periodontal disease as a risk factor for other diseases

- Periodontal infections as risk factors for systemic diseases has recently attracted special attention.
- Heart disease has been reported to be the condition most commonly found in periodontitis patient,
- Periodontal infections induce low level bacteremia, elevated white blood cell counts.
- Bacteremia from periodontitis and dental disease is known to be the primary cause of infective endocarditis .
- Periodontal infections as a risk factor for pre-term low birth weight and poorly controlled diabetes mellitus.


Thank You!

Lec.19

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HALITOSIS

❖ Halitosis Classification:

1) Genuine halitosis:

a) Physiologic halitosis: morning breath odour, tobacco smoking, certain foods & medications.

b) Pathologic halitosis

2) Pseudo halitosis

3) Halitophobia

❖ Causes of halitosis:

1) Systemic causes 10%
2) Intra oral causes 90%
3) Iatrogenic / idiopathic cause
4) Psychosomatic cause

Systemic causes:

1-Diabetes 2-Cirrhosis 3-Renal failure 4-Leukemia

5-Respiratory system: sinusitis, tonsillitis, bronchiectasis, lung infection.

6-Gastrointestinal system: reflux, carcinoma, gastric ulcer

7-Genetic disorders.

Intra oral causes: poor oral hygiene, cancer, decaying food particles, cellular & nutritional debris,

Plaque coated tongue, caries, bleeding gums, periodontal disease, xerostomia, postsurgical state, improperly cleaned denture.

- **The gram negative bacteria associated with halitosis are:**

1. Treponema denticola
2. Porphyromonas gingivalis
3. Tannerella forsythensis
4. Porphyromonas endodontalis
5. Prevotella intermedia
6. Eubacterium

Iatrogenic / idiopathic smells:

- Gauze pad left after cleft palate surgery
- Foreign objects inserted up the nose in young children & developmentally disabled if in detected may lead to odor in adults.

Idiopathic odors detectable by others, no apparent oral or non oral cause.

❖Psychomatic smells:

Detectable only by patient, no apparent cause, patient often refuse to accept objective findings, mostly associated with anxiety or depression, can be confused with genetic disorders.

❖Diagnosis smells:

History

1. Onset, duration
2. Constant or intermittent
3. Self report or reported by others

4. Dietary factors, smoking, alcohol use.
5. Systemic disease & medication.
6. Neurological problems, test & smell function.
7. Currently under stress.
8. Comprehensive oral examination.

Treatment of halitosis:

- Mechanical reduction of intraoral nutrients and microorganisms
- Chemical reduction of oral microbial load
- Rendering malodorous gases nonvaltile
- Masking malodor

- **Mechanical reduction of intraoral nutrients and microorganisms: (Management)**

a) Tongue cleaning
b) Tooth brush-interdental cleaning
c) Professional periodontal therapy
d) Chewing gum

- **Chemical reduction of oral microbial load: (Management)**

a) Chlorhexidine
b) Essential oils
c) Chlorine dioxide
d) Water rinse
e) Aminefluoride/Stannous fluoride
f) Hydrogen peroxide
g) Triclosan

- **Masking malodor:**

Treatment with rinses, mouth sprays, and lozenges containing volatiles with pleasant odor.

Lecture: 1 Periodontics Dr: Nuha Hamed

Diagnosis of periodontal diseases

Proper diagnosis is essential to intelligent treatment. Periodontal diagnosis should first determine whether disease is present then identify its type, extent, duration, distribution and severity.

Periodontal diagnosis is determined after careful analysis of the case history and

evaluation the clinical signs and symptoms, as well as the result of various tests (probing, mobility assessment, radiographs, blood test, and biopsies).

The following is a recommended sequence of procedures for the diagnosis of periodontal diseases.

Overall Appraisal of the patient. This includes consideration of the patient's

mental, emotional status, attitude and physiologic age.

☐ Medical History

The importance of the medical history should be explained to the patients because patients omit information that they cannot relate to their dental problems. The patient

should be made aware of:

1. The presence of conditions that may require special precautions or modifications in

treatment procedure.

2. The possible role that some systemic diseases, conditions, may play in the cause of

periodontal disease

3. The possibility that oral infections may have a powerful influence on the occurrence and severity of certain systemic disease

The Medical history should include reference to the following:

☐ Is the patient under the care of a physician, and if so what is the problem? Its duration

and nature.

☐ Details on hospitalization and operation including diagnosis, kind of operation, and complications.

☐ Medical problem hematologic, endocrine, infectious, cardiovascular

☐ The medications taking with special inquiry should be made regarding the dosage and duration of therapy with anticoagulant and corticosteroids.

☐ History of allergy recorded like fever, asthma, sensitivity to food.

☐ Family medical history including bleeding disorders and diabetes or others.

☐ Abnormal bleeding tendencies such as nose bleeding, abnormal ecchymosis, prolonged bleeding from minor cut and excessive menstrual bleeding.

Dental History

Current illness some patients may be unaware of any problem but many may report bleeding gum; loose of teeth; spreading of the teeth with the appearance of spaces where none existed before, foul test in the mouth.

Sensitivity when chewing, sensitivity to cold &hot, and extreme sensitivity to inhaled air.

A preliminary oral examination is done to explore the source of the patient's chief complaint and to determine if emergency treatment is required.

The Dental History should include reference to the following:

☐ A list of visit to the dentist, frequency, date of the last visit, nature of treatment and cleaning by a dentist.

☐ The patient's oral hygiene regimen including tooth brushing (frequency, method, type of

tooth brush and dentifrices) mouth wash, interdental brush, water irrigation and dental floss.

☐ Any orthodontic treatment, duration & termination date.

☐ Pain in the teeth or in the gingiva (nature, duration& how its relieved)

☐ Gingival bleeding (spontaneously , on brushing or eating)

☐ A bad test in the mouth.

☐ Do the teeth feel "loose" or insecure? Is there difficulty in chewing? Any tooth mobility

should be recorded.

☐ The patient's general dental habits such as grinding or clenching of the teeth during the

day or at night. Do the teeth or jaw muscles feel "sore" in the morning? Are there other

habits such as tobacco smoking or chewing, nail biting, or biting on foreign objects?

☐ History of previous periodontal problems, including the nature of the condition and if

previously treated, the type of treatment received (surgical or nonsurgical) and

approximate period of termination of previous treatment

☐ Does the patient wear any removable prosthesis? Does the prosthesis enhance or is it a

detriment to the existing dentition or the surrounding soft tissues?

☐ Does the patient have implants replacing any of the missing teeth?

Intraoral Radiographic Survey

The radiographic survey should consist of a minimum of 14 intraoral films and four posterior bite-wing films. Panoramic radiographs are a simple and convenient method of obtaining a survey view of the dental arch and surrounding structures. They are helpful for the detection of developmental anomalies, pathologic lesions of the teeth and jaws, and fractures as well as dental screening examinations of large groups. They provide an informative overall radiographic picture of the distribution and severity of bone

destruction in periodontal disease, but a complete intraoral series is required for

periodontal diagnosis and treatment planning.

Radiographic image tend to underestimate the severity of bone loss, the difference

between the alveolar crest height and the radiographic appearance range from 0-1.6mm mostly accounted for x-ray angulation.

Casts

Casts from dental impressions are useful adjuncts in the oral examination. They indicate

☐ The position of the gingival margins (recession)

☐ The position and inclination of the teeth

☐ Proximal contact relationships, and food impaction areas.

☐ They provide a view of the lingual-cuspal relationships.

Casts are important records of the dentition before it is altered by treatment. Finally, casts

also serve as visual aids in discussions with the patient and are useful for pretreatment

and posttreatment comparisons, as well as for reference at recall visits. They are also helpful to determine the position of implant placement if the case will require their use.

Clinical Photographs

Color photographs are useful for recording the appearance of the tissue before and after treatment.

Oral Examination

Oral Hygiene

The extent of accumulated food debris, plaque, and tooth surface stains. Disclosing solution may be used to detect plaque that would otherwise be unnoticed. The amount of plaque detected, however, is not necessarily related to the severity of the disease present. For example, aggressive periodontitis is a destructive type of periodontitis in which plaque is minimal.

Qualitative assessments of plaque are more meaningful, and their value in diagnosis.

Oral Malodor

Oral malodor, also termed fetor ex ore, fetor oris, or halitosis, is foul or offensive odor

emanating from the oral cavity. Mouth odors may be of diagnostic significance, and their origin

may be either oral or extraoral. It may indicate patient with systemic diseases (Liver disease,

DM, tonsillitis ,oropharynx& stomach)

Examination of Lymph Nodes

1. Because periodontal, periapical, and other oral diseases may result in lymph node

changes, the diagnostician should routinely examine and evaluate head and neck lymph nodes.

2. Lymph nodes can become enlarged and/or indurated as a result of an infectious episode,

malignant metastases, or residual fibrotic changes.

3. Inflammatory nodes become enlarged, palpable, tender, and fairly immobile. The overlying skin may be red and warm.

4. Patients are often aware of the presence of "swollen glands." Primary herpetic

gingivostomatitis, necrotizing ulcerative gingivitis (NUG), and acute periodontal

abscesses may produce lymph node enlargement.

Examination of the Teeth and Implants

☐ The teeth are examined for caries, poor restorations, developmental defects, anomalies of

tooth form, wasting, hypersensitivity, and proximal contact relationships.

☐ The stability, position, and number of implants and their relationship to the adjacent

natural dentition is also examined.

Periimplantitis

☐ Can create pockets around implants. Probing is important in diagnosis

☐ To prevent scratching the implant surface we should use plastic instrument.

Dental Plaque & Calculus

+ Supragingival plaque and calculus can be directly observed.

+ Detection of subgingival calculus each tooth surface is carefully checked to the level of

gingival attachment

+ Warm water is useful to deflect the gingiva and aid in visualization of calculus.

Wasting Disease of the Teeth

Wasting is defined as any gradual loss of tooth substance characterized by the formation

of smooth, polished surfaces. The forms of wasting are:

erosion, abrasion, attrition & Abfraction.

☐ Erosion:

☐ also called corrosion, is a sharply defined wedge-shaped depression in the cervical area of

the facial tooth surface

□ .The surfaces are smooth, hard, and polished. Erosion generally affects a group of teeth.

□ In the early stages, it may be confined to the enamel, but it generally extends to involve

the underlying dentin, as well as the cementum.

□ The etiology of erosion is not known. Decalcification by acidic beverages, or citrus

fruits, combined with the effect of acid salivary secretion are suggested causes.

□ Abrasion :

□ Rrefers to the loss of tooth substance induced by mechanical wear other than that of

mastication.

□ Abrasion results in saucer-shaped or wedge shaped indentations with a smooth, shiny

surface.

□ Abrasion starts on exposed cementum surfaces rather than on the enamel and extends to

involve the dentin of the root. A sharp "ditching" around the cemento-enamel junction

appears to be the result of the softer cemental surface, as compared with the much harder

enamel surface.

Tooth brushing with an abrasive dentifrice, Aggressive tooth brushing and hard

tooth brush are the most common causes.

Horizontal brushing at right angles to the vertical axis of the teeth results in the

severest loss of tooth substance.

Attrition:

□ Is occlusal wear resulting from functional contacts with opposing teeth. Such physical

wear patterns may occur on incisal, occlusal, and approximal tooth surfaces.

☐ A certain amount of tooth wear is physiologic, but accelerated wear may occur when

abnormal anatomic or unusual functional factors are present.

☐ Occlusal or incisal surfaces worn by attrition are called facets.

☐ When active tooth grinding occurs, the enamel rods are fractured and become highly

reflective to light. Thus shiny, smooth, and curviplanar facets are usually the best

indicator of ongoing frictional activity.

☐ If dentin is exposed, a yellowish brown discoloration is frequently present

☐ Facets vary in size and location depending on whether they are produced by physiologic

or abnormal wear. Facets are usually not sensitive to thermal or tactile stimulation.

☐ Attrition has been correlated with age when older adults are considered.

The angle of the facet on the tooth surface is potentially significant to the

periodontium. ** Horizontal facets tend to direct forces on the vertical axis of the tooth,

to which the periodontium can adapt most effectively. **Angular facets direct occlusal

forces laterally and increase the risk of periodontal damage.

Abfraction:

** Results from occlusal loading surfaces causing tooth flexure and mechanical

microfractures and tooth substance loss in the cervical area.

Examination of the Periodontium

The periodontal examination should be systematic, starting in the molar region in either

the maxilla or the mandible and proceeding around the arch. This prevents overemphasis

of unusual findings at the expense of other conditions that although less striking, may be

equally important. It is important to detect the earliest signs of gingival and periodontal

disease.

Gingiva

The gingiva is the keratinized mucosa that surrounds the teeth. It forms a collar around each tooth. The gingiva is typically coral pink in color and can be readily distinguished from the adjacent dark red alveolar mucosa by its lighter pink color. In dark-skinned persons the gingiva may contain melanin pigment to a greater extent than the adjacent alveolar mucosa. Localized gingival inflammation is confined to the gingiva in relation to a single tooth or group of teeth. Generalized gingival inflammation involves the entire mouth.Features of the gingiva to consider are: color, size, contour, consistency, surface

texture, position, ease of bleeding, and pain .

Color changes in the gingiva.

The normal gingival color is —coral pink‖. Gingiva becomes redder when there is an increase in vascularization or the degree of epithelial keratinization becomes reduced or disappears. Thus, chronic inflammation intensifies the red or bluish red color; this is caused by vascular proliferation and reduction of keratinization owing to epithelial compression by the inflamed tissue

Changes in the size of the gingiva.

The normal size depends on the sum of the bulk cellular and intercellular elements, and

their vascular supply. In disease, the size is increased, which can be termed as gingival

enlargement. The factors responsible for this are increase in fibers and decrease in cells

as in the non-inflammatory type. Whereas in the inflammatory type there will be increase

in cells and decrease in fibers

Changes in the consistency of the gingiva.

Both chronic and acute inflammations produce changes in the normal firm, resilient

consistency of the gingiva. In chronic gingival inflammation both destructive

(edematous) and reparative (fibrotic) changes coexist, and the consistency of the gingiva

is determined by their relative predominance

Gingival Index (GI) (Loe, 1967) measures the degree of gingival iflammation. Tissues

surrounding each tooth divided into 4 gingival scoring units: distal facial papilla, facial

margin, mesial facial papilla, lingual gingival margin.

Score of gingival index

Score 0 Normal gingiva

Score 1 Mild inflammation — slight change in color, slight edema. No bleeding on

probing

Score 2 Moderate inflammation — redness, edema and glazing. Bleeding on probing

Score 3 Severe inflammation — marked redness and edema. Ulceration. Tendency to

spontaneous bleeding

The GI may be used for the assessment of prevalence and severity of gingivitis in

populations, groups and individuals.

Gingival bleeding. Gingival bleeding varies in severity, duration and the ease with which

it is provoked. Bleeding on probing is easily detectable clinically and therefore is of great

value for the early diagnosis and prevention of more advanced gingival inflammation.

Gingival bleeding on probing is one of the earliest visual signs of inflammation. It can

appear earlier than color changes or any other visual signs of inflammation. It also

provides an additional advantage, by being a more objective sign that requires less

subjective estimation by the examiner. Gingival bleeding on probing also helps us to

determine whether the lesions are in an active or inactive state.

Bleeding on probing (BOP). A periodontal probe is inserted to the —bottom‖ of the

gingival/periodontal pocket by applying light force and is moved gently along the tooth

(root) surface. If bleeding is provoked upon retrieval of the probe, the site examined is

considered —BoP — positive and, hence, is inflamed.

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Plaque Index:

Clinical plaque indices are used to evaluate the level and rate of plaque formation on tooth

surfaces, and to test the efficacy of oral care products for removal and prevention of plaque

deposits from these surfaces. A number of different indices have been described

• which was introduced by Silness and Loe in 1964

☐ Used on all teeth (28, wisdom teeth are excluded) or selected teeth (6 teeth) .

☐ No substitution for any missing tooth.

☐ Used on all surfaces (4)(M, B, D, L).

☐ This index measures the thickness of plaque on the gingival one third of the teeth.

• 0 No plaque

• 1 A film of plaque adhering to the free gingival margin and adjacent area of the tooth, which can not

be seen with the naked eye. But only by using disclosing solution or by using probe.

• 2 Moderate accumulation of deposits within the gingival pocket, on the gingival margin and/ or

adjacent tooth surface, which can be seen with the naked eye.

• 3 Abundance of soft matter within the gingival pocket and/or on the tooth and gingival margin.

•

Calculus Index (CI)

Calculus is mineralized material on the tooth surface. The calculus index refers to the amount of

calculus on a tooth.

☐ CI 0 — No observable calculus.

☐ CI 1 — Supragingival calculus covering not more than 1/3 of the exposed tooth surface.

☐ CI 2 — Supragingival calculus covering more than 1/3 but not more than 2/3 of the

exposed tooth surface or presence of flecks of subgingival calculus.

☐ CI 3 — Supragingival calculus covering more than two-thirds of the exposed tooth

surface or a continuous heavy band of subgingival calculus around the cervical portion of

the tooth.

Depth of sulcus. The normal sulcus depth usually 1–3 mm

Pockets: is defined as pathologically deepened of gingival sulcus may occur by coronal

movement of the gingival margin (gingival pocket), or apical displacement of gingival

attachment (periodontal pocket) or combination of the above.

pockets are generally painless but may give rise to symptoms such as localized or

sometimes radiating pain or sensation of pressure after eating, which gradually

diminishes. A foul taste in localized areas, sensitivity to hot and cold, and toothache in

the absence of caries is also sometimes present. a "rolled" edge separating the gingival

margin from the tooth surface; or an enlarged, edematous gingiva, may suggest their

presence. The presence of bleeding, suppuration, and loose, extruded teeth may also

denote the presence of a pocket

Gingival pocket : Also known as pseudopocket or false pocket, seen in gingivitis formed

by gingival enlargement (increased gingival bulk) without apical migration of the

junctional epithelium

Periodontal pocket : true pocket seen in periodontitis, occurs with apical migration of

junctional epithelium and destruction to the supporting periodontal tissues. It can classify

into:

Suprabony pocket : bottom of the pocket is coronal to the underlying alveolar bone.

Infrabony pocket: bottom of the pocket is apical to the crest of the alveolar bone

Detection of Pockets

The only accurate method of detecting and measuring periodontal pockets is careful

exploration with a periodontal probe. Pockets are not detected by radiographic

examination. The periodontal pocket is a soft tissue change. Radiographs indicate areas of bone loss in which pockets may be suspected, but they do not show pocket presence or depth

Assessment of probing pocket depth (PPD). For effective treatment planning, the

location, topography, and extent of periodontal lesions must be recognized in all part of

the dentition. It is, therefore, mandatory to examine all sites of all teeth for the presence

or absence of periodontal lesions. The probe should be inserted parallel to the vertical

axis of the tooth and “walked” circumferentially around each surface of each tooth to

detect the areas of deepest penetration.This turn means that single-rooted teeth have to be

examinated at four sites at least (e. g. mesial, buccal, distal, and oral) and multirooted teeth at six sites at least (e. g. mesiobuccal, buccal, distobuccal, distooral, oral, and mesio-oral) The probing depth, that is the distance from the gingival margin to the

bottom of the gingival sulcus/pocket, is measured to the nearest millimetre by means of

a graduated periodontal probe.

Clinical Attachment Level (CAL) : is amore accurate indicator of the periodontal

support around the tooth than probing depth alone

CAL is measured from a fixed point on the tooth that doesn't change, the CEJ.

To calculate CAL, two measurements are needed:

☐ In recession : probing depth + gingival margin to the CEJ (add)

☐ In tissue overgrowth : probing depth – gingival margin to the CEJ(subtract)

. Changes in the level of attachment can be the result of gain or loss of attachment and afford

a better indication of the degree of periodontal destruction or gain

Lecture Perio: **Dr.Nuha Hamed**

Smoking and Periodontal diseases

The smoking is highly prevalent and can be considered an epidemic in both developed and developing nations. tobacco users among men and women are 42.4% and 14.2%, respectively. According to the Global Adult Tobacco Survey 2 (GATS 2), every third adult in rural areas and every fifth adult in urban areas use tobacco in some form or the other.

The gas phase of tobacco smoke contains carbon monoxide, ammonia, formaldehyde, hydrogen cyanide, and toxic compounds such as benzo(a)pyrene and dimethylnitrosamine. · The particulate phase of tobacco smoke includes nicotine, tar, benzene · Nicotine is quickly absorbed in the lungs, and it reaches the brain within 10-19 seconds. · Nicotine causes a rise in blood pressure, increased heart and respiratory rates, and peripheral vasoconstriction. · Smokers have reduced gingival inflammation and bleeding on probing. · In smokers, even shallow periodontal pockets are colonized by periodontal pathogens.

When is a Smoker or Not a Smoker? · Smokers have smoked ≥100 cigarettes in their lifetime and currently smoke. · Former smokers have smoked ≥100 cigarettes in their lifetime and do not currently smoke. · Nonsmokers have not smoked ≥100 cigarettes in their lifetime and do not currently smoke.

Smoking is harmful to almost every organ in the body and is associated with multiple diseases that reduce life expectancy and quality of life. Diseases associated with smoking include lung cancer, heart disease, stroke, emphysema, bronchitis, and cancers of the oral cavity, bladder, kidney, stomach, liver, and cervix. Approximately half of long-term smokers will die early as a result of smoking, and those who die before the age of 70

years will lose an average of 20 years of life. Most deaths from smoking are due to lung cancer, chronic obstructive pulmonary disease, and coronary heart disease

Smoking is a major risk factor for periodontitis, and it affects the prevalence, extent, and severity of disease. In addition, smoking has an adverse impact on the clinical outcome of nonsurgical and surgical therapy as well as the long-term success of implant placement.

Effects of smoking on the prevalence and severity of periodontal diseases

Gingivitis

Controlled clinical studies have demonstrated that, in human models of experimental gingivitis, the development of inflammation in response to plaque accumulation is reduced in smokers as compared with nonsmokers . In addition, cross-sectional studies have consistently demonstrated that smokers present with less gingival inflammation than nonsmokers. These data suggest that smokers have a decreased expression of clinical inflammation in the presence of plaque accumulation as compared with nonsmokers.

Periodontitis Although gingival inflammation in smokers appears to be reduced in response to plaque accumulation as compared with nonsmokers, an overwhelming body of evidence points to smoking as a major risk factor for increasing the prevalence and severity of periodontal destruction. Multiple cross-sectional and longitudinal studies have demonstrated that pocket depth, attachment loss, and alveolar bone loss are more prevalent and severe in patients who smoke as compared with nonsmokers. On average, smokers were four times as likely to have periodontitis as compared with persons who had never smoked.

Former smokers were 1.7 times more likely to have periodontitis than persons who had never smoked.

Smoking has also been shown to affect periodontal disease severity in younger individuals. Cigarette smoking is associated with increased severity of generalized periodontitis in young adults, and those who smoke are 3.8 times more likely to have periodontitis as compared with nonsmokers. In addition, smokers are more than six times as likely as nonsmokers to demonstrate continued attachment loss

Former smokers have less risk for periodontitis than current smokers but more risk than nonsmokers, and the risk for periodontitis decreases with the increasing number of years since quitting smoking.

Effects of smoking on the etiology and pathogenesis of periodontal disease The increased prevalence and severity of periodontal destruction associated with smoking suggests that the host-bacterial interactions normally seen with periodontitis are altered, resulting in more extensive periodontal breakdown . This imbalance between bacterial challenge and host response may be caused by changes in the composition of the subgingival biofilm (e.g., increases in the numbers and virulence of pathogenic organisms), changes in the host response to the bacterial challenge, or a combination of both

Effects of Smoking on Microbiology:

*Increased complexity of the microbiome and colonization of periodontal pockets by periodontal pathogens

***Immune- inflammatory response**:

- Altered neutrophil chemotaxis, phagocytosis, and oxidative burst.
- ↑ Tumor necrosis factor-α and prostaglandin E2 in gingival crevicular fluid.

- ↑ Neutrophil collagenase and elastase in gingival crevicular fluid.
- ↑ Production of prostaglandin E2 by monocytes in response to lipopolysaccharide.

***Physiology effect of Smoking:**

↓ Gingival blood vessels with ↑ inflammation.

↓ Gingival crevicular fluid flow and bleeding on probing with ↑ inflammation

↓ Subgingival temperature with ↑ Time needed to recover from local anesthesia

Microbiology

A study sampled subgingival biofilm from all teeth with the exception of third molars in 272 adult subjects, including 50 current smokers, 98 former smokers, and 124 nonsmokers. screen for 29 different subgingival species, it was found that members of the orange and red complex species–including Eikenella nodatum, Fusobacterium nucleatum ss vincentii, Prevotella intermedia, Peptostreptococcus micros, Tannerella forsythia, Porphyromonas gingivalis, and Treponema denticola–were significantly more prevalent in current smokers than in nonsmokers and former smokers. The increased prevalence of these periodontal pathogens was caused by an increased colonization of shallow sites (pocket depth ≤4 mm), with no differences among smokers, former smokers, and nonsmokers in pockets 4 mm or greater. In addition, these pathogenic bacteria were more prevalent in the maxilla than the mandible. These data suggest that smokers have a greater extent of colonization by periodontal pathogens than nonsmokers or former smokers, which may increase the risk of periodontal disease progression.

Immune-inflammatory responses

The immune response of the host to biofilm accumulation is essentially protective. In periodontal health, a balance exists between **the bacterial challenge of the biofilm and the immune-inflammatory responses in the gingival tissues, with no resulting loss of periodontal support**.

By contrast, **periodontitis** is associated with an alteration in the host-bacterial balance that may be initiated by changes in the **bacterial composition of the subgingival biofilm, changes in the host responses**, **other environmental changes, or a combination of these**. Smoking exerts a major effect on the immune-inflammatory response that results in an increase in the extent and severity of periodontal destruction. The deleterious effects of smoking appear to result from alterations in the immune-inflammatory response to bacterial challenge.

The neutrophil is an important component of the host response to the bacterial challenge, and alterations in **neutrophil number or function** may result in localized and systemic infections. Critical functions of neutrophils include chemotaxis (directed locomotion from the bloodstream to the site of infection), phagocytosis (internalization of foreign particles such as bacteria), and killing via oxidative and nonoxidative mechanisms.

Neutrophils obtained from the peripheral blood, oral cavity, or saliva of smokers or exposed in vitro to whole tobacco smoke or nicotine have demonstrated functional alterations in **chemotaxis, phagocytosis, and the oxidative burst.** In vitro studies of the effects of tobacco products on neutrophils have shown detrimental effects on cell movement as well as on the oxidative burst.

In addition, **levels of antibody to the periodontal pathogens** essential for phagocytosis and killing of bacteria, specifically **immunoglobulin G2** , have

been reported to be reduced in smokers as compared with nonsmokers with periodontitis, thereby suggesting that smokers may have reduced protection against periodontal bacteria. By contrast, **elevated levels of tumor necrosis factor-α** have been demonstrated in the gingival crevicular fluid of smokers, and elevated levels of **prostaglandin E2** , **neutrophil elastase, and matrix metalloproteinase-8** have also been found. These data suggest that smoking alters the response of neutrophils to the bacterial challenge such that there are increases in the release of tissue-destructive enzymes, causing increased periodontal tissue destruction.

Physiology: Previous studies have shown that certain clinical signs of inflammation (e.g., gingival redness, gingival bleeding) are less pronounced in smokers than in nonsmokers. This may result from alterations in the vascular response of the gingival tissues. Although no significant differences in the vascular density of healthy gingiva have been observed between smokers and nonsmokers, the response of the microcirculation to biofilm accumulation appears to be altered in smokers as compared with nonsmokers. With developing inflammation, gingival crevicular fluid flow, bleeding on probing, and gingival blood vessels are lower in smokers than in nonsmokers. In addition, the oxygen concentration in healthy gingival tissues appears to be lower in smokers than in nonsmokers, although this condition is reversed in the presence of moderate inflammation. Subgingival temperatures are lower in smokers than nonsmokers, and recovery from the vasoconstriction caused by local anesthetic administration takes longer in smokers. These data suggest that significant alterations are present in the gingival microvasculature of smokers as compared with nonsmokers and that these changes lead to decreased blood flow and decreased clinical signs of inflammation. This explains the long-observed phenomenon of a transient

increase in gingival bleeding when a smoker quits.

Effects of smoking on the response to periodontal therapy:

Nonsurgical therapy: Numerous studies have indicated that current smokers do not respond as well to periodontal therapy as nonsmokers or former smokers do. Most clinical research supports the observation that probing depth reductions are generally greater in nonsmokers than in smokers after nonsurgical periodontal therapy. In addition, gains in clinical attachment as a result of nonsurgical treatment are less pronounced in smokers than in nonsmokers. When a higher level of oral hygiene was achieved as part of nonsurgical care, the differences in the resolution of 4- to 6-mm pockets between nonsmokers and smokers became clinically less significant.

Therapy Effects of Smoking

Nonsurgical ↓ Clinical response to root surface debridement ↓ Reduction in probing depth ↓ Gain in clinical attachment levels ↓ Negative impact of smoking with ↑ level of plaque control

Surgery and implants: ↓ Probing depth reduction and ↓ gain in clinical attachment levels after access flap surgery ↑ Deterioration of furcations after surgery, ↓ bone fill, ↑ recession, and ↑ membrane exposure after guided tissue regeneration ↓ Root coverage after grafting procedures for localized gingival recession . ↑ Risk for implant failure and peri-implantitis

Maintenance care ↑ Probing depth and attachment loss during maintenance therapy ↑ Disease recurrence in smokers ↑ Need for retreatment in smokers ↑ Tooth loss in smokers after surgical therapy

It can be concluded that smokers respond less well to nonsurgical therapy than do nonsmokers. With excellent plaque control, these differences may be minimized, but the emphasis is on truly excellent plaque control. When

comparing current smokers with former smokers and nonsmokers, the former and nonsmoking subjects appear to respond equally well to nonsurgical care, thereby reinforcing the need for patients to be informed of the benefits of smoking cessation.

Surgical therapy and implants:

The less favorable response of the periodontal tissues to nonsurgical therapy that is observed in current smokers is also observed after surgical therapy. In a longitudinal comparative study of the effects of four different treatment modalities (coronal scaling, root planing, modified Widman flap surgery, and osseous resection surgery), **smokers (with "heavy" defined as ≥20 cigarettes/day and "light" defined as ≤19 cigarettes/day) consistently showed less pocket reduction and less gain in clinical attachment as compared with nonsmokers or former smokers**. These differences were evident immediately after the completion of therapy and continued throughout 7 years of supportive periodontal therapy. During the 7 years, deterioration at furcation areas was greater in heavy and light smokers than in former smokers and nonsmokers. Smoking has also been shown to have a negative impact on the outcomes of guided tissue regeneration and the treatment of infrabony defects by bone grafts. By 12 months after guided tissue regeneration therapy at deep infrabony defects, smokers demonstrated less than half the attachment gain that was observed in nonsmokers (2.1 mm versus 5.2 mm). In a second study, 73 smokers also showed less attachment gain than nonsmokers (1.2 mm versus 3.2 mm), more gingival recession, and less bone infill of the defect. Similarly, after the use of bone grafts for the treatment of infrabony defects, smokers showed less reduction in probing depths as compared with nonsmokers.

In patients who had undergone implant therapy, smoking increases the risk

of implant failure. Overall, the risk for implant failure in smokers appears to be approximately double the risk for failure in nonsmokers, and the risks appear to be higher in maxillary implants and when implants are placed in poor-quality bone. Smoking has also been shown to be a risk factor for periimplantitis.

With a majority of studies showing a significant increase in periimplant bone loss as compared with nonsmokers. Collectively, these data indicate that implant failure is more common among smokers than nonsmokers. Given the current evidence, all patients who are considering implant therapy should be informed about the benefits of smoking cessation and the risks of smoking for the development of peri- implantitis and implant failure.

Maintenance therapy: The detrimental effect of smoking on treatment outcomes appears to be long lasting and independent of the frequency of maintenance therapy. After four modalities of therapy (scaling, scaling and root planing, modified Widman flap surgery, and osseous surgery), maintenance therapy was performed by a hygienist every 3 months for 7 years. Smokers consistently had deeper pockets than nonsmokers and less gain in attachment when evaluated each year for the 7-year period. Even with more intensive maintenance therapy given every month for 6 months after flap surgery,

*smokers had deeper and more residual pockets than nonsmokers, although no significant differences in plaque or bleeding on probing scores were found. These data suggest that the effects of smoking on the host response and the healing characteristics of the periodontal tissues may have a longterm effect on pocket resolution in smokers, possibly requiring more intensive management during the maintenance phase.

*Smokers also tend to experience more periodontal breakdown than

nonsmokers after therapy.

*Tobacco smoking was positively associated with tooth loss even when regular recall maintenance care was performed (overall, smokers had a risk of losing their teeth that was up to 380% higher than that of nonsmokers). Similarly, smoking has a detrimental effect on peri-implant tissue status, even when patients are under strict periimplant preventive maintenance care. It is clear from these studies that (1) smokers may present with periodontal disease at an early age, (2) they may be difficult to treat effectively with the conventional therapeutic strategies; (3) they may continue to have progressive or recurrent periodontitis; and (4) they may be at an increased risk of tooth loss or peri-implant bone loss, even when adequate maintenance control is established.

For these reasons, smoking cessation counseling must be a cornerstone of periodontal therapy in smokers.

Effects of smoking cessation on periodontal treatment outcomes

Smoking cessation positively influenced periodontal treatment outcomes. When patients received nonsurgical therapy as treatment for their periodontitis, in addition to smoking cessation counseling for a period of 12 months, those individuals who successfully quit smoking for the entire 12 months of the study had the best response to the periodontal treatment. The benefit of smoking cessation on the periodontium is likely to be mediated through various pathways, such as a shift toward a less pathogenic microbiome, the recovery of the gingival microcirculation, and improvements in certain aspects of the immune- inflammatory responses.

In conclusion, smoking is a major risk factor for periodontitis, and smoking cessation should be an integral part of periodontal therapy among patients who smoke. Smoking cessation should be considered a

priority for the management of periodontitis in smokers.

DR. Nuha Hamed

Impact of periodontal infection on systemic health

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

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

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

Subgingival Environment as a Reservoir for Bacteria

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

The mechanisms by which periodontal infections may influence systemic health

have been described as follows:

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

organs.

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

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

organs

Periodontal Disease and Coronary Heart Disease/Atherosclerosis/stroke

To further explore the association between periodontal disease and

CHD/atherosclerosis, investigators have studied specific systemic disorders and

medical outcomes to determine their relationship to periodontal status. CHD related events are a major cause of death. MI has been associated with acute systemic bacterial and viral infections. Traditional risk factors such as smoking, dyslipidemia, hypertension, and diabetes mellitus do not explain the presence of coronary atherosclerosis in a large number of patients. Localized infection that results in a chronic inflammatory reaction has been suggested as a mechanism

underlying CHD in these individuals.

It has been found that Gram negative bacteria & the associated lipopolysaccharide (LPS) plays an important role in atherogenesis because it has the ability to trigger the release of interleukin 1, tumor necrosis factor- alfa & thromboxane, initiate platelet aggregation, promote the deposition of cholesterol & enhance atheroma formation.

The role of LPS as a systemic trigger for the development of atheromas

has led the investigators to search for an infections site which would

provide a source for LPS.

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

1- Periodontal infection may also promote increased blood viscosity and thrombogenesis leading to an increased risk for central and peripheral vascular

2-Periodontal disease may predispose the patient to an increased incidence of bacteraemia, including the presence of virulent gram-negative organisms associated with periodontitis .

3-Platelets selectively bind some strains of *Streptococcus sanguis*, a common Component of supra gingival plaque, and *Porphyromonas gingivalis*, a pathogen closely associated with periodontitis –thrombosis

4-Gram-negative bacteria and associated PS cause infiltration of inflammatory cells into the arterial wall, proliferation of arterial smooth muscle, and intravascular coagulation Atheromatosis

Periodontal disease and diabetes mellitus

The role of diabetes in periodontal disease is bi-directional, that is diabetes is a known risk factor for periodontitis and periodontitis in turn affects the glycemic control in individuals with diabetes.

Periodontal infections result in an elevation of serum pro-inflammatory markers.

These may adversely affect metabolic control, may result insulin resistance which in turn over time can lead to hyperglycemia and type 2 diabetes. Chronic gram negative periodontal infections in individual with diabetes may also worsen glycemic control. Patients that harbor periodontal pathogens have significantly higher markers of systemic inflammation like C-reactive protein (CRP), IL-6 and fibrinogen than patients without these pathogens.

Periodontal disease and pregnancy outcome

Infants born before the completion of 37 weeks of gestation are referred to as preterm infants. Preterm infants usually weigh lower at birth (< 2500 gm) and prematurity is associated with increased perinatal mortality.

The most significant factor for preterm delivery is maternal infections attributing to about half of the preterm births. Bacteria from the maternal genital tract infections

elicit a pro inflammatory response in the mother, which ultimately results in release of prostaglandins and matrix metalloproteinases. This in turn causes smooth muscle contraction and membrane weakening respectively and triggers premature cervical ripening. This bacterial- host inflammatory response is considered to be the association between maternal periodontal disease and adverse pregnancy outcomes. In addition to the above pathway, bacteremia associated with periodontal disease may reach the uterus thus exposing the maternal-fetal unit to the bacteria and their products. This may elicit the above mentioned inflammatory response leading to preterm delivery.

Periodontal infection and respiratory disorders.

The oral cavity plays an important role in infections acquired in hospitals and nursing homes, especially infections of the respiratory tract. Several studies have demonstrated that the teeth of patients in the intensive care unit (ICU) became colonized by respiratory pathogens such as *Pseudomonas aeruginosa, Enteric species* and *Staphylococcus aureus*. Similar studies have shown that the teeth of nursing home residents can also serve as reservoirs for respiratory infection. An association between oral conditions such as periodontal infections and respiratory conditions such as pneumonias and chronic obstructive pulmonary disease has been found. Evidence has suggested the oropharyngeal region as a likely source of bacteria implicated in respiratory infection.

Oral periodontopathic bacteria can be aspirated into the lung to cause aspiration pneumonia. Respiratory pathogens have been shown to colonize the dental plaque of hospitalized intensive care and nursing home patients. Once established in the mouth, these pathogens can be aspirated into the lung to cause infection. Cytokines originating from periodontal tissues may change respiratory epithelium to promote infection by respiratory pathogens. A systematic review of the epidemiologic and clinical evidence found that poor periodontal health increases the risk of developing chronic obstructive pulmonary disease (COPD).

Periodontal disease and asthma

Asthma is a chronic disease of the airways characterized by inflammation and bronchoconstriction that occurs in people of all ages.

Children with chronic medical disorders, like asthma, who require long-term medication have an increased susceptibility to dental diseases in three ways:

frequent use of sugar containing syrups, use of sedatives causing a decreased saliva secretion, and use of corticosteroids.

The possible interactions between medications used for asthma and the induction of periodontal changes have also been positively correlated in the literature .Data from some studies suggest that inhalers can lead to changes in pH and a decrease in saliva production and therefore increase biofilm accumulation and calculus. In addition, the immunosuppressive effect of corticosteroids may have some influence on the response of the periodontal tissues. These agents act by inhibiting the host response, thus hampering the clinical expression of gingivitis.

The association between periodontal disease and respiratory/lung diseases has been shown previously. Once installed, gingival diseases (Gingivitis / periodontitis) may contribute indirectly through recurrence or worsening of respiratory attacks. Some mechanisms for the interrelationship between diseases have been proposed, such as aspiration of biofilm and hematogenous dissemination or inflammatory chemical mediators from the periodontal pockets. Thus, treatment and maintenance of gingival health can improve pulmonary function and decrease the frequency of respiratory attacks.

Conclusions

There is sufficient evidence to suggest that periodontal disease and systemic health have a two- way relationship, in that, the periodontal disease can cause adverse systemic conditions and that certain systemic diseases cause periodontal disease. It is vital that the physicians and other health care providers educate the patients about this association and to recommend dental care facilitate restoration of oral health in these individuals. The evidence suggests that treatment of one disease could lead to better outcomes for the other. This knowledge should be used to attain better patient outcomes in future.

Lecture: **Dr: Nuha Hamed**

ATTEMPTS AT CLASSIFICATION:

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

Major changes were made in the 1999 classification of periodontitis, which has been in use for the last 19 years. Periodontitis was reclassified as chronic, aggressive (localized and generalized), necrotizing and as a manifestation of systemic disease.

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

Classification of periodontal diseases and conditions (2017):

1. Periodontal health and gingival diseases and condtions

a. Periodontal health and gingival health

b. Dental biofilm induced gingivitis

c. Non-dental biofilm induced gingival disease

2. Periodontitis

a. Periodontitis

b. Necrotizing periodontal diseases

c. Periodontitis as a manifestation of systemic disease

3. Other conditions affecting the periodontium

a. Periodontal abscess and endodontic periodontal lesions

b. Mucogingival deformity and conditions

c. Traumatic occlussal force

d. Tooth and prosthetic related factors

4. Peri-implant disease and conditions

a. Peri- implant health

b. Peri-implant mucositis

c. Peri-implantitis

d. Peri-implant soft and hard tissues deficiency

1. Periodontal health and gingival diseases and conditions

a. Periodontal health and gingival health: is defined by absence of clinically detectable inflammation. There is a biological level of immune surveillance that is consistent with clinical gingival health and homeostasis. Clinical gingival health can be restored following treatment of gingivitis and periodontitis. However, the treated and stable periodontitis patient with current gingival health remains at increased risk of recurrent periodontitis, and accordingly, must be closely monitored.

Based on available methods to assess gingival health and inflammation, which can be simply, objectively and accurately defined and graded using a bleeding on probing score (BOP%), assessed as the proportion of bleeding sites when stimulated by a standardized (dimensions and shape) periodontal probe with a controlled (~0.25 N) force to the apical end of the sulcus. So gingival health can be classified in to:

- **Clinical gingival health on an intact periodontium**

Clinical gingival health on an intact periodontium is characterized by the absence (or minimum) of bleeding on probing (less than 10%), absence of patient symptoms, and attachment and bone loss.

- **Clinical gingival health on a reduced periodontium that include:**
  - **Non-periodontitis patient (e.g. recession, crown lengthening).**

While clinical gingival health on a reduced periodontium is characterized by an absence (or minimum) bleeding on probing (less than 10%), *with possible presence of reduced clinical attachment and bone levels*. With probing pocket

depth ≤3. In non-periodontitis patients, there is no current evidence for increased risk of periodontitis.

○ **Stable periodontitis patient:** the clinical gingival health in stable periodontitis patients is characterized by an absence (or minimum) bleeding on probing (less than 10%), *in the presence of interproximal clinical attachment loss*. while probing pocket depth ≤4 provided that there is no pseudo pockets and no bleeding on probing at site with 4mm pocket depth. However, it should be recognized that successfully treated and stable periodontitis patients remain at increased risk of recurrent progression of periodontitis.

A. **Dental biofilm induced gingivitis**

Dental plaque biofilm-induced gingivitis is defined at the site level as "an inflammatory lesion resulting from interactions between the dental plaque biofilm and the host's immune-inflammatory response, which remains contained within the gingiva and does not extend to the periodontal attachment (cementum, periodontal ligament and alveolar bone). Such inflammation remains confined to the gingiva and does not extend beyond the mucogingival junction and is reversible by reducing levels of dental plaque at and apical to the gingival margin". A patient diagnosed as gingivitis as follows: **localized gingivitis**, defined as a patient presenting with a BOP score ≥10% and ≤30%, or **generalized gingivitis**, defined as a patient presenting with a BOP score > 30%. Depending on whether dental biofilm-induced gingival inflammation occurs on an intact or reduced periodontium, or in a patient diagnosed with periodontitis, gingivitis can be further classified as:

• **Gingivitis on an intact periodontium**

Gingival inflammation associated with BOP score ≥10%, probing pocket depth ≤ 3mm assuming no pseudo pocket , no attachment loss and no radiographic bone loss.

• **Gingivitis on a reduced periodontium in a non-periodontitis patient (e.g., recession, crown lengthening)** A patient with a reduced periodontium but without a history of periodontitis (e.g. gingival recession, crown lengthening) and a BOP score ≥10% would be diagnosed as a "gingivitis on a reduced periodontium , probing pocket depth ≤ 3mm assuming no pseudo pocket , with possible presence of attachment loss and radiographic bone loss.

• **Gingival inflammation on a reduced periodontium in a successfully treated periodontitis patient (remission periodontitis)**

Gingival inflammation associated with BOP score ≥10%, probing pocket depth ≤ 4mm assuming no pseudo pocket , with presence of attachment loss and radiographic bone loss**,** the patient will be diagnosed as remission periodontitis (Note that recurrent periodontitis cannot be ruled out in this case).

The classification of dental biofilm induced gingivitis:

A. Associated with bacterial dental biofilm only

B. Mediated by systemic or local risk factors

1. Systemic conditions

a) Sex steroid hormones 1) Puberty 2) Menstrual cycle 3) Pregnancy 4) Oral contraceptives

b) Hyperglycemia

c) Leukemia

d) Smoking

e) Malnutrition

2. Oral factors enhancing plaque accumulation

a) Prominent subgingival restoration margins

b) Hyposalivation

C. Drug-influenced gingival enlargements

There are common characteristics to all gingival diseases associated with Dental plaque induced gingival diseases:-

1. Gingivitis is a clinical diagnosis. While emerging technologies are starting to shed light on the microbiological, molecular, and pathophysiological characteristics of gingivitis, definitive knowledge is not sufficient to supersede current clinical parameters.
2. The clinical signs of inflammation are erythema, edema, pain (soreness), heat, and loss of function, these may manifest clinically in gingivitis as: a. Swelling, seen as loss of knife-edged gingival margin and blunting of papillae b. Bleeding on gentle probing c. Redness d. Discomfort on gentle probing
3. The symptoms a patient may report include: a. Bleeding gums (metallic/altered taste) b. Pain (soreness) c. Halitosis d. Difficulty eating e. Appearance (swollen red gums) f. Reduced oral health–related quality of life
4. Radiographs cannot be used to diagnose gingivitis.
5.Reversibility of the disease by removing the etiology .

1. Gingival disease associated with dental biofilm only:-

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

2. mediated by systemic or local risk factors:-

1. Systemic factors

a) Plaque-induced gingivitis exacerbated by sex steroid hormones

 Puberty: It is pronounced inflammatory response of gingiva to dental plaque and hormones during the circumpubertal period (11-16) years.

 Menstrual cycle: It is pronounced inflammatory response of the gingiva to plaque and hormones immediately prior to ovulation.

 pregnancy: It is pronounced inflammatory response of the gingiva to dental plaque and hormones usually occurring during the second and third

trimesters. During pregnancy, the prevalence and severity of gingivitis has been reported to be elevated and frequently unrelated to the amount of plaque present, The features of pregnancy-associated gingivitis are similar to plaque-induced gingivitis, except the propensity to develop frank signs of gingival inflammation in the presence of a relatively small amount of plaque during pregnancy. Pregnancy may also be associated with the formation of pregnancy-associated pyogenic granulomas.

 pregnancy-tumor: It is a localized, painless, protuberant, exophytic gingival mass that is attached by a sessile or pedunculated base from the gingival margin or more commonly from an interproximal space resulting from dental plaque and hormones during pregnancy.it is more common in the maxilla and may develop as early as the first trimesters, and may regress or completely disappear following parturition.

 Oral contraceptive:- Pronounced inflammatory response of the gingiva to plaque and oral contraceptive. The features of gingivitis associated with oral contraceptive in premenopausal women are similar to plaque-induced gingivitis, except for the propensity to develop signs of gingival inflammation in the presence of relatively little plaque in women taking these hormones. The condition is reversible following discontinuation of the drug.

b. Hyperglycemia : It is inflammatory response of the gingiva to plaque aggravated by poorly controlled plasma glucose levels.

c. Leukemia: Pronounced inflammatory response of the gingiva to plaque resulting in increased bleeding and enlargement subsequent to leukemia. Gingival bleeding is a common sign in patients with leukemia and it is the initial oral sign and/or symptom in 17.7% and 4.4% of patients with acute and chronic leukemia respectively. Gingival enlargement initially begin at the interdental papilla followed by marginal and attached gingiva.

d. **Smoking** : is one of the major lifestyle/behavioral risk factors for periodontitis, but which also has profound effects upon the gingival tissues. Systemic circulatory uptake of components of cigarette smoke as well as local uptake are reported to induce microvascular vasoconstriction and fibrosis. This can mask clinical signs of gingivitis, such as bleeding on probing, despite a significant underlying pathological inflammatory cell infiltrate.

e. **Gingival diseases modified by malnutrition**: It is known that malnourished individuals have a compromised host defense system that may affect the susceptibility to infection. Ascorbic acid-deficiency gingivitis: Inflammatory response of the gingiva to plaque aggravated by chronically low ascorbic acid levels. The classic clinical signs of scurvy describe the gingiva as being bright red, swollen, ulcerated and susceptible to hemorrhage. It is common in certain population, with restricted diets (e.g. infants from low socio economic families and institutionalized elderly).

2. Oral factors enhancing plaque accumulation.

a) The local contributing factors can be defined as a local feature that may influence the presentation of the disease, such as prominent subgingival restoration margins, orthodontic appliance.

b) Hyposalivation. Oral dryness is a clinical condition often associated with symptoms of xerostomia. Oral dryness manifesting as a lack of salivary flow, availability, or changes in quality of saliva, leading to reduced cleansing of tooth surfaces is associated with reduced dental plaque biofilm removal and enhanced gingival inflammation. Common causes include medications that have anti-parasympathetic action, Sjögrens syndrome when the salivary acini are

replaced by fibrosis following autoimmune destruction, and mouth breathing in people who may have enhanced gingival display and/or an incompetent lip seal.

3.**Drug-influenced gingival enlargement**: Gingival enlargement resulting in whole or in part of gingiva from systemic drug use. Drugs that may cause gingival overgrowth include anticonvulsant (e.g. phenytoin), immunosuppressant (e.g. cyclosporine A), and calcium channel blockers (e.g. nifedipine, verapamil).

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

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

The classification of Non-dental biofilm induced gingival lesions:

1 Genetic/developmental disorders

Hereditary gingival fibromatosis

2 Specific infections

a. Bacterial origin
Neisseria gonorrhoeae (gonorrhea)
Treponema pallidum (syphilis)
Mycobacterium tuberculosis (tuberculosis)
Streptococcal gingivitis (strains of streptococcus)
b. Viral origin
Coxsackie virus (hand-foot-and-mouth disease)
Herpes simplex 1/2 (primary or recurrent)
Varicella-zoster virus (chicken pox or shingles affecting V nerve)

4 Reactive processes

Epulides
Fibrous epulis

5 Neoplasms

a. Premalignant
Leukoplakia
Erythroplakia
b. Malignant
Squamous cell carcinoma
Leukemia
Lymphoma

6 Endocrine, nutritional, and metabolic diseases

Vitamin deficiencies

7 Traumatic lesions

a. Physical/mechanical insults
Toothbrushing-induced gingival ulceration
Factitious injury (self-harm)
b. Chemical (toxic) insults
Etching

c. Fungal
Candidosis

3 Inflammatory and immune conditions and lesions

a. Hypersensitivity reactions
Contact allergy
Plasma cell gingivitis
Erythema multiforme
b.Autoimmune diseases of skin and mucous membranes
Pemphigus vulgaris
Pemphigoid
Lichen planus
Lupus erythematosus
c.Granulomatous inflammatory conditions (orofacial granulomatosis)
Crohn's disease
Sarcoidosis
Chlorhexidine
Acetylsalicylic acid
c. Thermal insults
Burns of mucosa

8 Gingival pigmentation

Smoker's melanosis
Amalgam tattoo