PERIODONTOLOGY

Lec.1

Termsand definitionsused in periodontology

Periodontium: This term arises from the Greek word (pert=around,odontos=tooth)
Thus it can simply defined as: the tissues surround and support the teeth.
The periodontium is composed of the following tissues:
1.Periodental ligament.
2.Gingiva.
3.Root cementum.
4.Alveolar bone.

Periodotology: It is the clinical science that deals with the periodontium in health and disease.

Periodontics or periodontia: It is the branch of dentistry that conserned with prevention and treatment of periodontal disease.

Periodontist: Adental practitional who limits his or her practice to periodontics.

Periodontal diseases: those pathological processes affecting the periodontium most often gingivitis and periodontitis.

Gingivitis: Is the inflammation of gingiva with out attachment loss and it is a reversible condition.clinically manifested by bleeding upon probing, erythema and swelling.

Periodontitis: Is the inflammation of the supporting tissues of the teeth resulting in permenant destruction of the periodontal ligament and alveolar bone with pocket formation and loss of attachment it is irreversible condition.
There are two types of pockets:
1.periodontal pocket(true pocket).
2.pseudopocket(false pocket).

Periodontal pocket(true pocket):
It is a pathologically deepened gingival sulcus caused by apical migration of the junctional epithelium from the cementoenamel junction as loss of connective tissue attachment occur by periodontal disease process.(Fig.3)

Pseudopocket(false pocket):
It is deepening of the gingival sulcus resulting from gingival enlargement without apical migration of the junctional epithelium and without destruction of the underlying periodontal tissues (Fig.2)

A Periodontal ligament(PDL):-

It is a connective tissue surrounding the root and connecting it with the bone.
It is consistsprimarily of
1.Bundles of intermingling collagen fibers.
2.Cellular elements.
3.Ground substance.

Development of PDL:-

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

1 Fibers of PDL:-

((A)) Majority of fibers in PDL are collagen fibers and are called the "principal fibers of PDL" they are arranged in bundles and follow a wavy or S shaped course.
The development of principal fibers of PDL will be as follows :- (Fig.4)

1-Small,fine,brush like fibrils are detected arising from the root cementum and projecting into the PDL space. The surface of the bone is at this stage , covered by osteoblasts and small number of radiating thin collagen fibrils can be seen.

2-The number and thickness of fibers entering the bone increase and gradually become longer while the fibers originating from the cementum are still short.

3-The fibers originating from the cementum will increase in length and thickness and fuse with the fibers originating from the alveolar bone in the periodontal ligament space.
Following tooth eruption, the principal fibers become organized in bundles and run continuously from the cementum to the bone.

It has been suggested that in teeth undergoing active eruption, the fibers instead of being continuous, they consist of two separated parts, one is located toward the cementum and the other toward the alveolar bone and they spliced together in the mid way between cementum and bone to form what is called " intermediate plexus "

The terminal portions of the principal fibers that insert into cementum and bone are termed " Sharpey's fibers " (Fig:- 5)

The principal fibers of the PDL are arranged in five groups these are :-

1-Alveolar crest fibers (ACF):-

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

2-Horizontal fibers (HF):-

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

3-Oblique fibers(OF):-

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

4-Apical fibers(AF):-

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

5-Interradicular fibers(IF):- C → B

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

collagen

B Other fibers in PDL

((B))Elastic fibers:-These are relatively few, and associated with the blood vessels.

C ((C))Oxytalan fibers:-These immature forms of fibers are thought to regulate vascular flow.

Occlusal forces are absorbed primarily by the oblique fibers. The remaining (ACF,HF,AF,IF) bundles counteract tipping and rotating forces.

2-Cellular Elements of PDL include:-

(1)Synthetic cells

A- Osteoblasts.

B- Fibroblasts

C- Cementoblasts.

(2)Resorptive cells

A- Osteoclasts.

B- Fibroblasts.

C- Cementoclasts.

(3)Epithelial rests of Malassez cells.

(4) immune

(5) → neurovascular

(4)Immune system cells.

(5)Cells associated with neurovascular elements.

(1)Synthetic cells

A- Osteoblasts: These cells cover the osseous surface of the PDL and are responsible for the formation of the alveolar bone.

B-Fibroblasts: These are the most common cells in the PDL. The main function of fibroblast is the production of various types of fibers such as collagen fiber – oxytalan fibers and elastic fibers.

C-Cementoblasts: Are seen lining the cementum surface of the PDL and are responsible for cementum deposition .

(2)Resorptive cells

A-Osteoclasts: These are large multinucleated cells and responsible for bone resorption.

B-Fibroblasts: These cells synthesize collagen and also possess the capacity to phagocytose old collagen fibers and degrade them by enzyme hydrolysis. The process of fibers resorption occur either during disease or physiologic turn over.

C-Cementoclast: cementum is not remodeled as the alveolar bone and PDL but it undergoes continual deposition during life.

Resorptions of cementum occurs in certain conditions by cementoclasts.

(3)Epithelial rests of malassez

These are found close to the cementum. They are reminants of Hertwig's root sheath. They proliferate when stimulated and participate in the formation of periapical cysts and lateral root cysts.

(4)Immune system cells

Include mast cells, macrophages, lymphocytes, neutrophils. Mast cells contain histamine which play a role in the inflammatory reactions. Macrophages are capable of phagocytosis. So all these cells are consider as defense cells .

(5) → neurovascular

(3) Ground substance of PDL:-

The space between cells, fibers, blood vessels and nerves in the PDL space is filled by ground substance. It consists of two main components

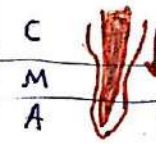
a. Glycosaminoglycans

b. Glycoproteins

It also has a high water content (70%)

Width of PDL:-

The width of PDL space varies with age, location of tooth, degree of stress to which the tooth was subjected. In compliance with the physiologic mesial migration of the teeth, the PDL is thinner on the mesial root surface than on the distal surface. A tooth in hypo function may have a narrow PDL space and a tooth in hyperfunction may have a wider PDL space. The width of PDL space is about 0.25mm in normal function. It is widest at the cervical and apical portions of the root and narrowest at the middle.



Elasticity of PDL:-

It is gained by

1- Wavy coarse of the principal fibers which allows for slight movement of the teeth despite the inelastic nature of the collagen fibers.

2- Intermediate plexus.

3- Presence of elastic & oxytalan

3-The presence of oxytalan and elastic fibers in the PDL but they are relatively few.

Functions of the PDL:-

1-Physical function

a-Transmission of occlusal forces to the bone.

b-Attachment of the teeth to the bone.

c-Resistance to the impact of occlusal forces(shock absorption)

d-Provision of a soft tissue 'casing' to protect the vessels and nerves from injury by mechanical forces.

2-Formative and Remodeling function

Cells of the PDL participate in the formation and resorption of cementum and bone which occur in physiologic tooth movement, in the accommodation of the periodontium to occlusal forces and in the repair of injuries. The PDL is constantly undergoing remodeling. Old cells and fibers are broken down and replaced by new ones. Fibroblasts form the collagen fibers and may also develop in to osteoblasts and cementoblasts.

3-Nutritive functions

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

4-Sensory functions

The PDL is supplied with sensory nerve fibers which transmit"①tactile-②pressure-③pain sensation by the trigeminal pathway,in addition the PDL is supplied with mechanoreceptors that transmit sense of ④localization which is done through proprioceptive nerve endings unlike the pulp do not have mechanoreceptors so the sense of localization is not present in the pulp. ✗ pulp

Blood supply of PDL:-

The blood supply to the supporting structures of the tooth is derived from the inferior and superior alveolar arteries to the mandible and maxilla respectively and reaches the PDL from three sources: Fig 8 - 9 - 10

(1) apical vessels supply the apical region of the PDL.

(2) the transalveolar vessels from the alveolar bone. (middle region)

(3) anastomosing vessels from the gingiva. (coronary region)

PERIODONTOLOGY

Lec -2-

Histology of the gingiva and oral mucosa

Oral mucosa:- all the soft tissues of the mouth , it consists of : Fig 1. Fig 2

1-Masticatory mucosa:

Which includes the gingiva and the covering of the hard palate. The boundries are from the free gingival margin to the mucogingival junction (MGJ) on the facial and lingual surfaces. (MGJ: is adistinct line between the attached gingiva apically and the alveolar mucosa).

On the palatal side there is no mucogingival junction because both gingiva and mucosa of the hard palate are of the same type which is masticatory mucosa. This tissue is firmly attached to the underlying bone and covered with keratinized epithelium to withstand the frictional forces of food during chewing and swallowing.

2-Specialized mucosa:

Which covers the dorsum of the tongue.

3-Lining mucosa:

This tissue is loosely attached to the underlying bone and covered with non-Keratinized epithelium. Examples of this type of mucosa are the tissues covering the lips, cheeks, floor of the mouth, inferior surface of the tongue, soft palate and the alveolar mucosa (**alveolar mucosa:** is located apical to the attached gingiva and extends into the vestibule of the mouth, it is darker red and moveable because there are more elastic fibers in the alveolar mucosa).

Gingiva:- Is that part of the masticatory mucosa which covers the alveolar process and surrounds the cervical portion of the teeth.

Clinically, the gingiva is divided into 3 parts: Fig 1 Fig 2

1-Marginal gingiva.
2-Attached gingiva.
3-Interdental gingiva.

1-Marginal gingiva :(Free or un attached)

Is the most coronal portion of the gingiva surrounding the teeth in collar like fashion but not attached to them and it is demarcated apically from the adjacent,attached gingiva,by the free gingival groove (which is a shallow linear depression of about 1 mm wide and is positiond at a level corresponding to the level of the cemento-enamel junction, it is only present in about 30-40% of adults). Free gingiva form the soft tissue wall of the gingival sulcus.

(**Gingival sulcus:** the space bounded by the free gingival margin, the tooth, and the most coronal attachment of the junctional epithelium, it is lined with non-keratinized sulcular epithelium. Clinically normal gingival sulcus measures from 1-3 mm deep, but ,in disease the space become deeper and is referred to as apocket.

2-Attached gingiva :

It is demarcated coronally from the free gingiva by the free gingival groove and extend apically to the mucogingival junction where it become continous with the alveolar mucosa. It is firm, resilient and tightly bound to the underlying teeth and periosteum of alveolar bone. The irregular surface texture of the attached gingiva (stippling) ,similar to the surface of an orange peel, found in 40% of adults.

The width of the attached gingiva differs in different areas of the mouth and is greatest on the facial surface the maxillary lateral incisors and narrowest on the facial surfaces of the mandibular canines and first premolars. On the lingual, it is widest near the first and second molars and narrowest adjacent to the incisors and canines.

3-Interdental gingiva :

It is located in the interproximal space beneath the area of teeth contact. It's triangular in shape from 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 cementoenamel junction.

In general, there are 2 shapes of the interdental gingiva:

1. Pyramidal shape:

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

2. Col-shape: Fig3

The interdental papillae between the posterior teeth are more flattened and there is a concave depression that connects a facial and lingual papilla and conforms to the shape of the interproximal contact surface.

If diastema or gingival tissue recession are present, no Col will be seen.

The significance of Col, it is covered by thin non-keratinized epithelium, hence represents the most frequent sit for initiation of disease process.

Clinical descriptive criteria of clinically healthy gingiva and inflammed one:

1. Gingival color:

The normal color of gingiva is coral pink with some variations depending on the amount of melanin in the tissues, the thickness of the epithelium, the degree of the keratinization and the vascularity of the connective tissue. Dark skinned people often exhibit dark blue or brown color.

The color of inflammed gingiva may vary from red to bluish red due to vasodilation which lead to bleeding tendency.

2. Gingival contour:

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

In inflammed gingiva, the contours are often rounded and enlarged because of vascular stagnation and increased formation of collagen fibers.

***3.Gingival consistancy*:**

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

In inflammed gingiva, the consistency may be soft and spongy because of the vascular stagnation and decrease in the amount of gingival collagen fibers or extremely firm because of excessive formation of collagen (fibrosis), this is in case of chronic inflammation.

***4.Gingival surface texture*:**

Gingiva may have either stippled or smooth and shiny sufrace, the attached gingiva is stippled, while the free gingva is smooth. In inflammed gingiva, reduction or lack of stippling is not an indicator of health nor is the absence of stippling an indicator of disease.

***Normal microscopic features*:** Fig 2 - Fig 4

The gingiva consists of fiberous connetive tissue known as lamina properia covered by stratified squamous epithelium.

Gingival epithelium may be differentiated as follows:

1.Oral epithelium: which faces the oral cavity.

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

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

***Oral epithelium*:**

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

The projection of epithelial cells into the connective tissue are known as Rete Pegs. The intervening connective tissue portions which project into the epithelium are called connective tissue papillae. Fig 4

C.T into epithelium

This alternating pattern of depression and protuberances of the connective tissue papillae and epithelial rete pegs is thought to give the attached gingiva the "stippled appearance".

The oral epithelium consists of four layers of cells: Fig 4

1-Stratum basale :Basal layer of cuboidal cells along the basement membrane.This is where epithelial cell replication& cell differentiation begins.
Melanocytes are formed in this layer

2-Stratum spinosum:The cells appear to have cytoplasmic spines .This is the thickest cell layer &Langerhans cells are found in this layer

3-Stratum granulosum:Keratohyaline granules may be seen in this layer.Cells appear to be flattened.

4-Stratum corneum:This is the layer where both para or the orthokeratinization occure .It is the most superficial layer.
The epithelial cells are formed as basal cells &gradually they undergo the process of keratinization,this is achieved by proliferation &differentiation of these cells(change to the characteristics of each of the cell layers)as they migrate toward the surface layer.

The Oral Epithelium contains the following types of cells :

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

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

3-Langerhans cells: These cells play a role in the defence mechanism of the oral epithelium. They have an immunological function by recognizing &processing antigens.

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

Under normal conditions, there is complete equilibrium between cell renewal &desquamation (cell turn over). It takes approximately 3-4 weeks for keratinocytes to migrate from basal layer until reach the outer epithelial surface, where it becomes desquamated from stratum corneum

The basal cells are found immediately adjecent to the connective tissue &are separated from this tissue by a basement membrane (basal lamina) → lamina propria [C.T]

The basement membrane consist of : Fig 5

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

2-Lamina densa: located beneath the lamina lucida, from this structure the anchoring fibers project into the connective tissue.

The epithelial cells are joined together by structure known as desmosomes, which is composed of two hemidesmosomes seprated from each other by granulated material.

A hemidesmosome composed from the following structures Fig 5

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(AP):*which represent granular &fibrillar material in the cytoplasm.

Sulcular Epithelium: Fig 4

It lines the gingival sulcus.It is a thin ,non keratinized stratified squamous epithelium without retepegs &extends from the coronal limit of the junctional epithelium to the crest of the gingival margin.

The sulcular epithelium is important because it is thin &may act as a semipermeable membrane through which tissue fluid from the gingiva seeps into the sulcus &makes easier for the bacterial products of dental plaque to penetrate into the connective tissue of the gingiva &stimulate inflammation &tissue destruction .That is why the sulcular epithelium is considered as apoor barrier against bacterial infection.

Periodontology رابع اسنان

Junctional epithelium(JE)

The epithelium that attaches the gingiva to thesurface of the tooth, it consist of stratified squamous non-keratinizing epithelium. It's 3-4 layers thick in early life but the numbers of layers increases with age to 10-20. It's thicker in coronal portion but become thinner toward cemento-enamel junction only a few cell layers.

The junctional epithelium cells can be groupedin two layers:the basal and the supra basal layer. This epithelium is continuously renewed through cell division in the basal layer and the cell migrate coronally to the base of the gingival sulcus from where they are shed (cell turn over)

The junctional epithelium is attached to the tooth surface by means of an internal basal lamina and hemidesmosomes and to the gingival connective tissue by an externalbasal lamina and hemidesmosomes, healthy JE exhibits no rete ridges where it contacts the connective tissue

The internal basal lamina consists of a lamina densa(adjacent to the enamel)and a lamina lucida to which hemidesmosomes are attached

The JE assumes a key role in maintenance of periodontal health, it creates the firm epithelial attachment that connect the soft tissue to the tooth surface. It is quite permeable and thus serve as a pathway for diffusion of the products plaque bacteria to the connective tissue. There is alsodiffusion in the

opposite direction moving towards the sulcus of host defense substances, this helps to mount an immune response

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

1.The size of the cells in the JE is relatively larger than the oral epithelium

2.The intercellular space in the JE is wider than in the oral epithelium. The intercellular space of the JE is preferred route for tissue fluids and inflammatory cells to migrate from the connective tissue to gingival sulcus

3.The number of desmosomes (intercellular junctions) is fewer in the junctional epithelium than in the oral epithelium, this may explain the JE susceptibility to tear during probing and its greater permeability to migrate cells and fluids

4.The sulcular and junctional epithelium are not as thick as the oral epithelium, because they are not keratinized and in health have no retepegs.

5.JE turnover rate is very high(4-6 days) compared to oral epithelium that has longest turn over rate (6-12days or up to 40 days)

6.JE forms the attachment of the gingiva to the tooth surface while oral and sulcular epithelium have no attachment to the tooth surface.

Gingival crevicular fluid:-GCF

The gingival fluid is continuously secreted from gingival connèctive tissue into the gingival sulcus through the sulcular epithelium.

In a strictly normal gingiva, little or no fluid can be collected but an increase in GCF flow is the first sign of inflammation

The GCF contain a variety of enzymes, cells and dectrolytes, proteins, antibodies

The functions of GCF

1.mechanical cleansing of the sulcus

2.possess antimicrobial properties

3.possess antibodies that enhance the resistance of the gingiva to inflammation

4.contain plasma proteins which may improve the adhesion of the epithelium to the tooth

Gingival connective tissue:CT

The connective tissue of the gingiva is known asthe lamina propira and consists of two layers.

1.The papillary layer: it consists of papillary projections between the epithelial retepegs

2.The reticular layer:it is contiguous with the periosteum of the alveolar bone

The major components of the CT are:-

1.collagen fibers 60%

2.cells 5%

3.ground substance, blood vessels, nerves and lymphatics (35%)

Gingival fibers:-

1.The most predominant type of fibers in the connective tissue of the gingiva are the collagen fibers

2.reticular fibers

3.oxytalan fibers

3.elastic fibers

Arrangement of gingival fibers

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

1.circular fibers:They run through the CT of the marginal and interdental gingiva and encircle the tooth in ring like fashion

2.dentogingival fibers:They project from the cementum in a fan shape fashion toward the free gingival

3.dentoperiosteal fibers:They extend from the cementum in apical direction to the periosteum of the alveolar bone and terminate in the attached gingiva.

4.transseptal fibers: Located interproximally, These are horizontal bundles that extend between the cementum of approximating teeth into which they are embedded.

The gingival fibers functions

1.To brace the marginal gingiva firmly against the tooth

2.To provide the rigidity to with stand the forces of mastication without being deflected away from the tooth surface.

3.To unite the free marginal gingiva with the cementum of the root and the adjacent attached gingiva

Cellular elements of the gingival CT:-

1.Fibroblasts:- The most predominant cells of the CT(65%), They synthesize collagen and elastic fibers as well as the CT matrix and regulate collagen degradation

2.Mast cells:- is responsible for the production of certain components of the matrix, they also produces vasoactive substances which may control the flow of blood through the tissue

3.Macrophages:- have a phagocytic function and are involved in the defense of the tissue against irritating substances.

4.Inflammatory cells:- Include various types such as poly morphonuclear leukocytes, Lymphocytes and plasma cells. These cells have different immunological functions.

Matrix of the connective tissue:-(ground substance)

The matrix fills the space between fibers and cells and has a high content of water. It's first produced by the fibroblasts although some constituents are produced by mast cells and other are derived from the blood, It is the medium in which the CT cells are embedded and it is essential for the

maintenance of the normal function of the CT, thus, The transportation of water, electrolytes, nutrients, metabolites, etc, to and from the C.T cells occurs within the matrix. Its main constituents 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.

The blood supply of the gingiva consists of

-supra periosteal vessels

-vessels from the PDL and bone

-These vessels coalesce in the gingival papilla as the gingival plexus.

Innervation of the gingiva:-

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

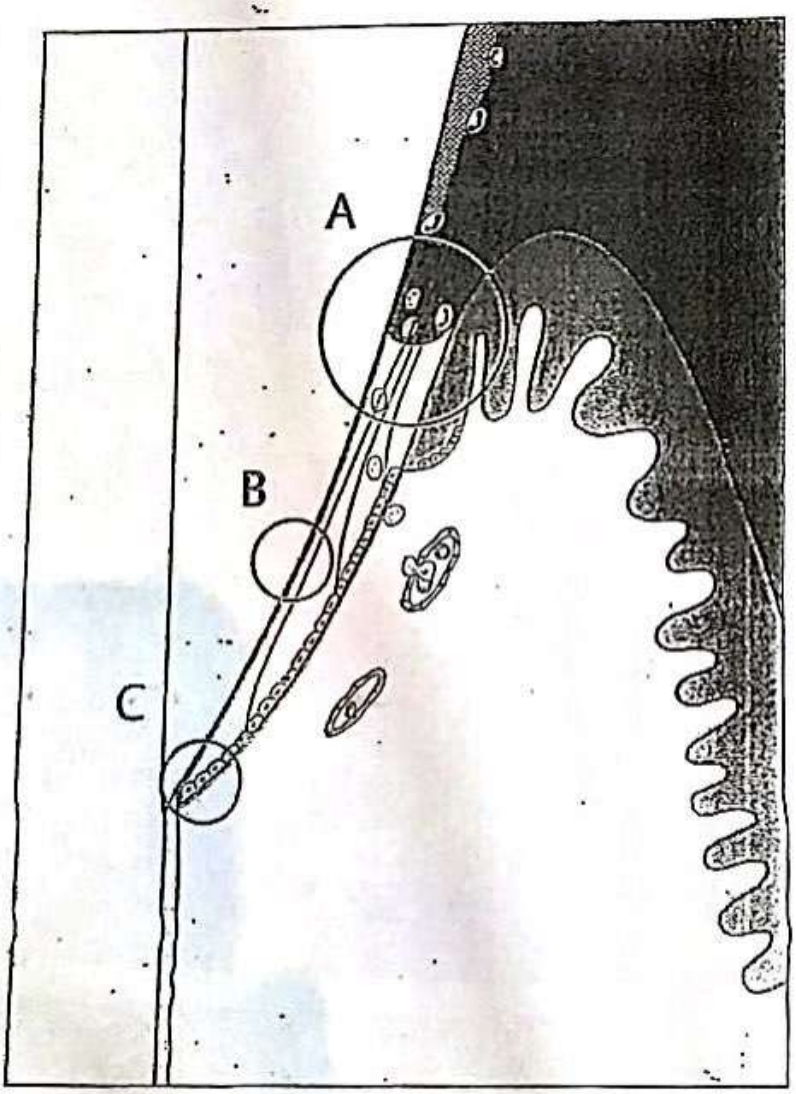
nctional Epithelium—Epithelial Attachment—ingival Sulcus

he marginal gingiva attaches to the tooth surface by means f the junctional epithelium, an attachment that is continously being renewed throughout life (Schroeder 1992).

nctional Epithelium

he junctional epithelium (JE) is approximately 1–2 mm in oronoapical dimension, and surrounds the neck of each ooth. At its apical extent, it consists of only a few cell layers; more coronally, it consists of 15–30 cell layers. Subjacent the sulcus bottom, the JE is about 0.15 mm wide.

The junctional epithelium consists of two layers, the ba (mytotically active) and the suprabasal layer (daugh cells). It remains undifferentiated and does not keratini The basal cell layer interfaces with the connective tissue hemidesmosomes and an external basal lamina. Healthy exhibits no rete ridges where it contacts the connective sue. JE turnover rate is very high (4–6 days) compa to oral epithelium (6–12 days, Skougaard 1965; or up 40 days, Williams et al. 1997).



7 Junctional Epithelium and Ingiva in Orofacial Section
he gingiva consists of three tis-ues:
Junctional epithelium
Oral epithelium
Lamina propria (connective tissue)
he *junctional epithelium* (JE) as-umes a key role in maintenance f periodontal health: It produces ne *epithelial attachment* and nerefore creates the firm connec-ion of soft tissue to the tooth sur-ace. It is quite permeable, and hus serves as a pathway for diffu-ion of the metabolic products of laque bacteria (toxins, chemo-actic agents, antigens, etc.). here is also diffusion in the oppo-ite direction, of host defense ubstances (serum exudates, anti-odies; etc.). Even when the gingi-ae do not appear inflamed clini-ally, the JE is constantly trans-nigrated by polymorphoneudear eukocytes (PMNs) moving to-vards the sulcus (p. 55, Fig. 109). *The red arrows depict the migra-ion of daughter cells from the* basal layer toward the gingival sulcus. The circled areas A-C are depicted in detail on page 11.

Structure of the Junctional Epithelium (JE)

Height: 1–2 mm
Coronal width: 0.15 mm

A **Gingival Sulcus (GS)**
Histologic
– Width: 0.15 mm
– Depth: 0–0.5 mm
Clinical
– Depth: 0.5–3 mm (dependent upon penetration of the probe into the junctional epithelium; Fig. 378)

B **Epithelial Attachment**
– Internal basal lamina (IBL)
Thickness: 35–140 nm
(1 nm = 10^{-9} m)
– Hemidesmosomes

C **Apical Extent**
of the junctional epithelium

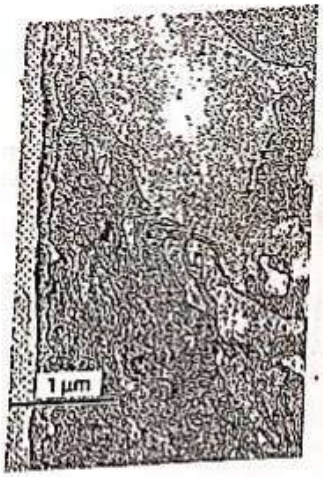
Epithelial Attachment

The epithelial attachment to the tooth is formed by the JE, and consists of an *internal basal lamina* (IBL), and *hemidesmosomes*. It provides the epithelial attachment between gingiva and tooth surface. This can be upon enamel, cementum or dentin in the same manner. The basal lamina nd the hemidesmosomes of the epithelial attachment are tructural analogs of their counterparts comprising the in-rface between epithelium and connective tissues.

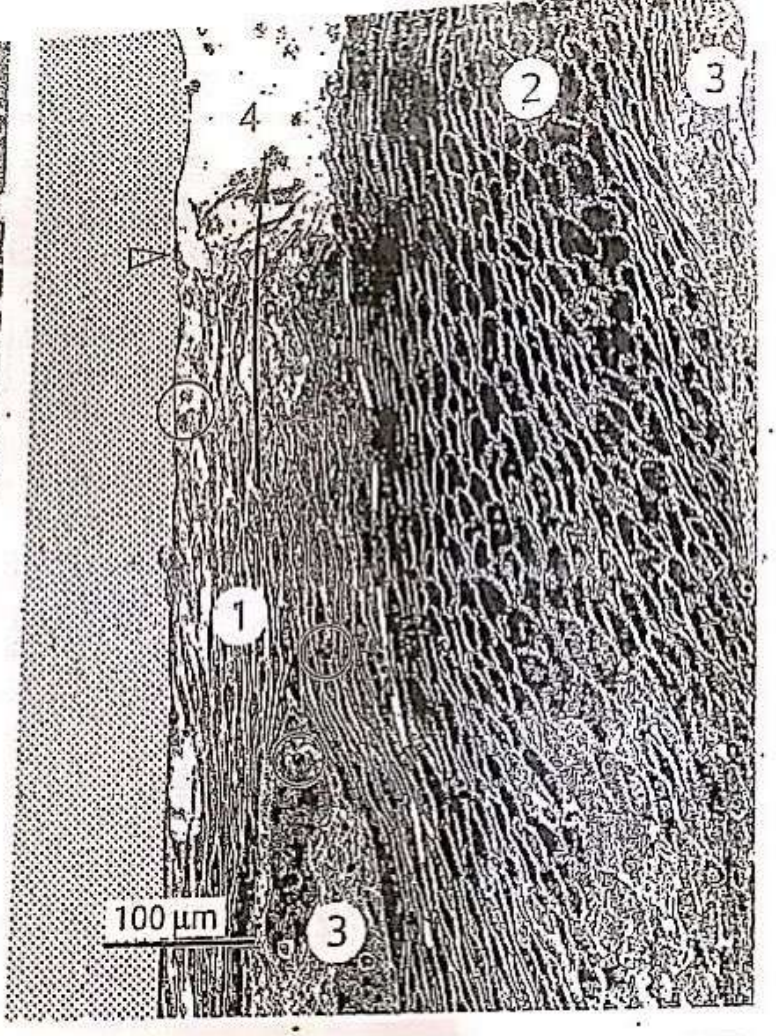
ll cells of the JE are in continual coronal migration, even ose cells in immediate contact with the tooth surface hemidesmosomal attachments. Between the basal lam and the tooth surface, a 0.5–1 µm thick "dental cuticle observed; this is possibly a serum precipitate or a secre product of the junctional epithelial cells.

Gingival Sulcus

The sulcus is a narrow groove surrounding the tooth, al 0.5 mm deep. The bottom of the sulcus is made up of most coronal cells of the junctional epithelium, which sloughed (exfoliated) in rapid succession. One lateral wa the sulcus is made up of the tooth structure.



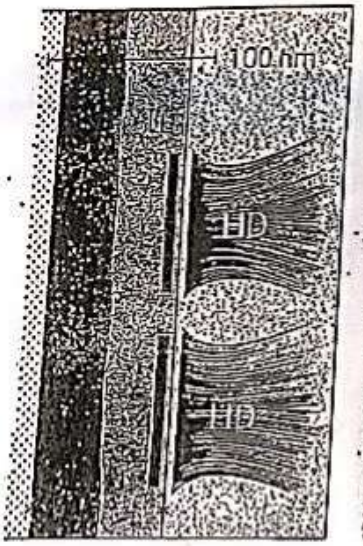
ınctional Epithelium	JE
ıral Sulcular Epithelium	OSE
onnective Tissue	CT
Ingival Sulcus	GS

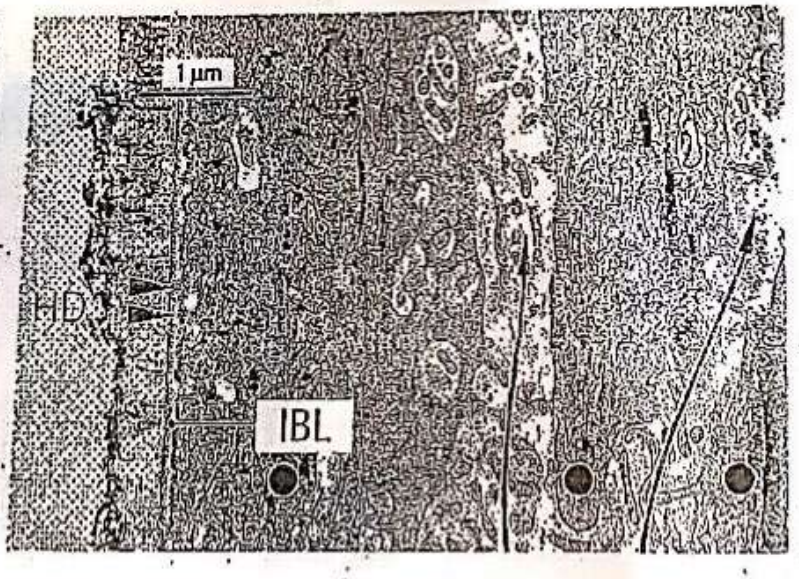


The junctional epithelial ce
are oriented parallel to the t
surface and are sharply demai
ed (broken line) from the more
deeply staining cells of the oral
sulcular epithelium (2). All of the
daughter cells that emanate from
the entire 1–2 mm length of the
basal layer of the junctional ep-
ithelium must transmigrate the
exceptionally narrow (100–150
µm) sulcus bottom (red arrow).
Note the polymorphonuclear
leukocytes (circled), which emi-
grate from the venule plexus in
the subepithelial connective tis-
sue (3) without altering it in any
way.

Left: In the enlargement, a portion of the most coronal JE cells (cf. empty black arrow in lower power view) is shown still manifesting hemidesmosomes and an internal basal lamina attached to the enamel surface.

Courtesy *H. Schroeder*

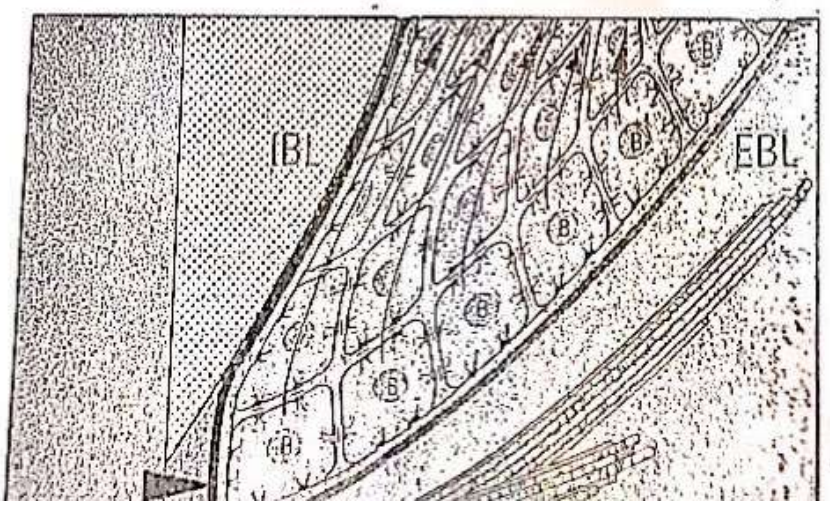




19 Internal Basal Lamina and Hemidesmosomes

Each JE cell adjacent to the tooth forms hemidesmosomes (HD) that enable these cells to attach to the internal basal lamina (IBL) and ultimately to the surface of the tooth. Remnants of enamel crystals are visible at the left. The long arrows indicate intercellular spaces between three JE cells (o).

Left: The basal lamina is comprised of two layers: the Lamina lucida (LL) and the Lamina densa (LD).



20 Apicalmost Portion of the Junctional Epithelium

In a young, healthy patient, the JE ends apically at the cemento-enamel junction. Daughter cells o the cuboidal basal cells (B) mi-grate toward the sulcus (red ar-rows). If a JE cell comes into con-tact with the tooth surface, it establishes the attachment mech-anism described above. The inter-nal basal lamina (IBL) is continu-ous with the external basal lamin

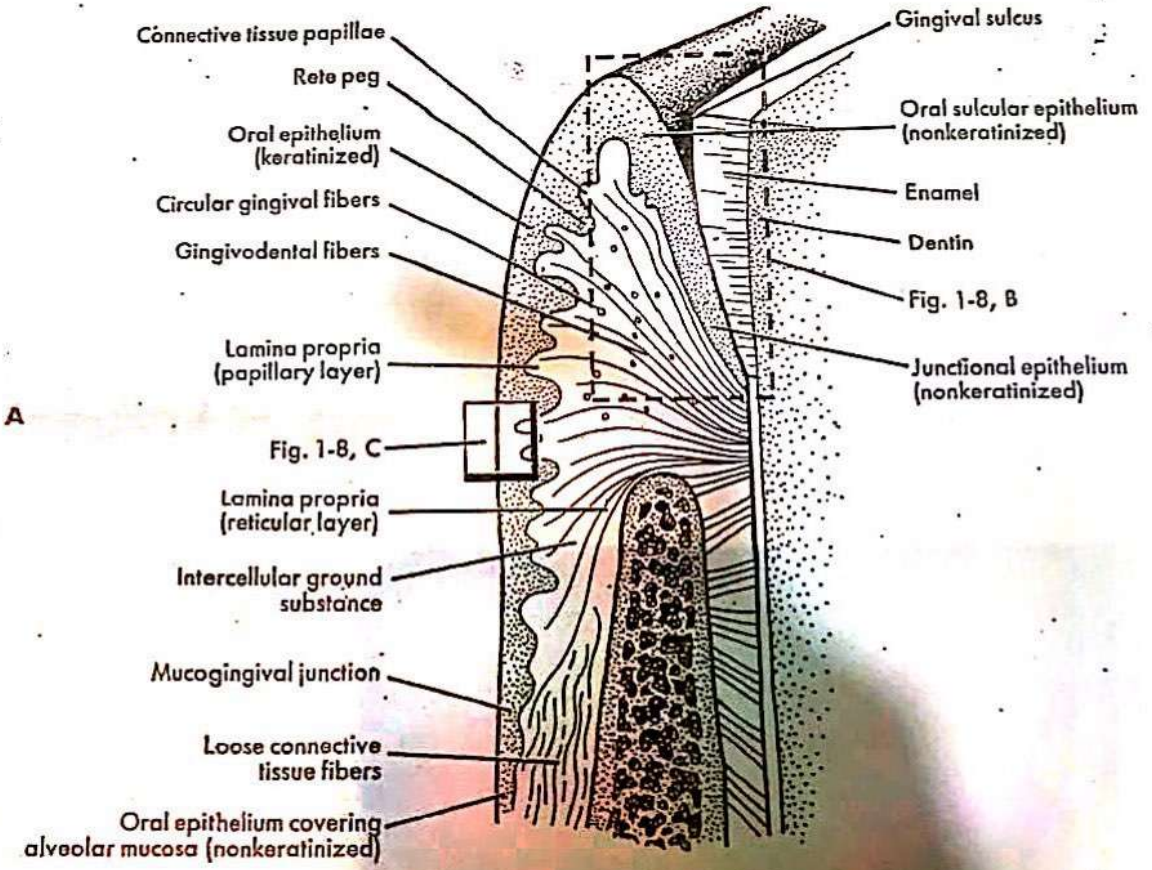


FIGURE 1-8. Histological characteristics of the gingiva. A, Faciolingual section of the periodontium.

Connective Tissue Attachment

Gingival and Periodontal Fiber Apparatus

The fibrous connective tissue structures provide the attachment between teeth (via cementum) and their osseous alveoli, between teeth and gingiva, as well as between each tooth and its neighbor. These structures include:

- Gingival fiber groups
- Periodontal fiber groups (periodontal ligament)

Gingival Fiber Groups

In the supra-alveolar area, collagen fiber bundles course in various directions. These fibers give the gingiva its resiliency and resistance, and attach it onto the tooth surface subjacent to the epithelial attachment. The fibers also provide resistance to forces and stabilize the individual teeth into a closed segment (Fig. 22). The periosteogingival fibers are also a component of the gingival fiber complex. These connect the attached gingiva to the alveolar process.

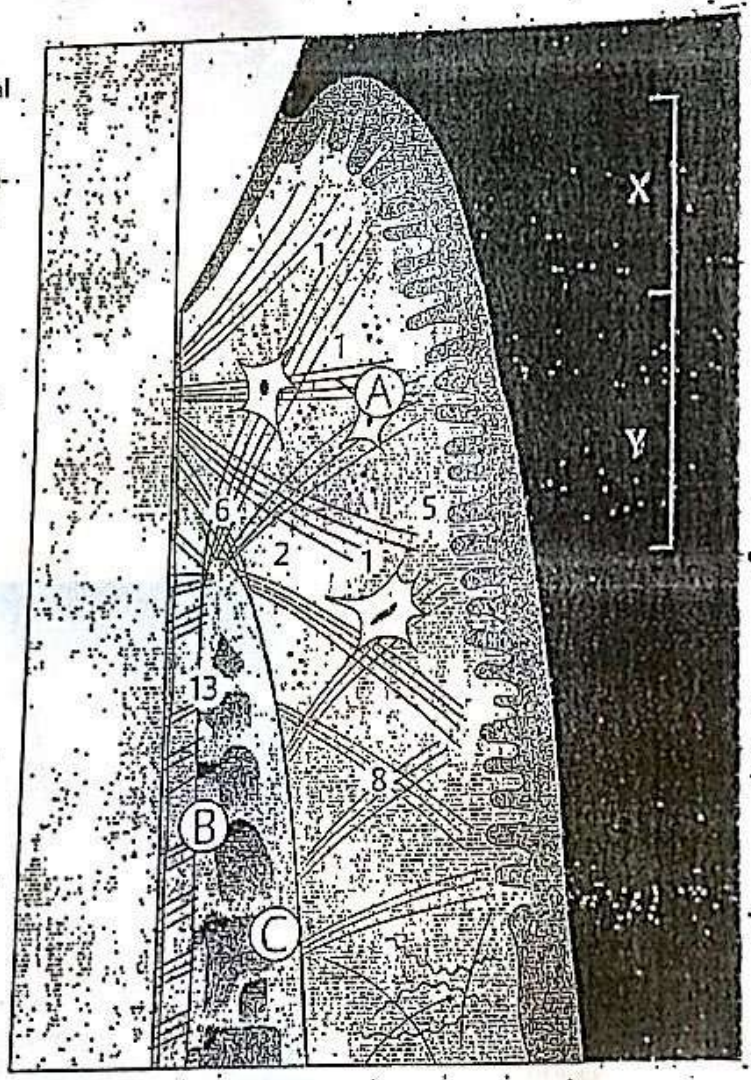
21 Localization and Orientation of Gingival and Periodontal Ligament Fiber Bundles (see also Fig. 22)

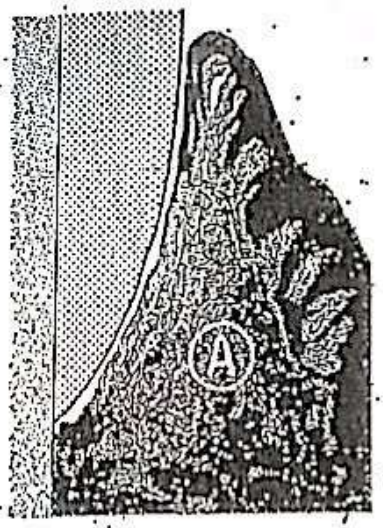
In the supra-alveolar region, within the free marginal gingiva and partially also the attached gingiva, the connective tissue compartment is composed primarily of collagen fiber bundles (A). These splay from the cementum of the root surface into the gingiva. Other fiber bundles course more or less horizontally within the gingiva and between the teeth, forming a complex architecture (Fig. 22). In addition to collagen fibers, one may also observe a small number of reticular (argyrophilic) fibers.

The periodontal ligament space (B) in adults is ca. 0.15–0.2 mm wide. About 60 % of the space is occupied by collagen fiber bundles. These fibers traverse from cementum to alveolar bone (C).

Right: Marginal gingiva. Fiber-rich connective tissue (A, blue), junctional epithelium and oral epithelium (reddish brown).

Histology courtesy N. Lang





A Gingival Fibers

B Periodontal Ligament Fibers

C Alveolar Bone

X Sulcus and Junctional Epithelium

Y Connective Tissue Attachment

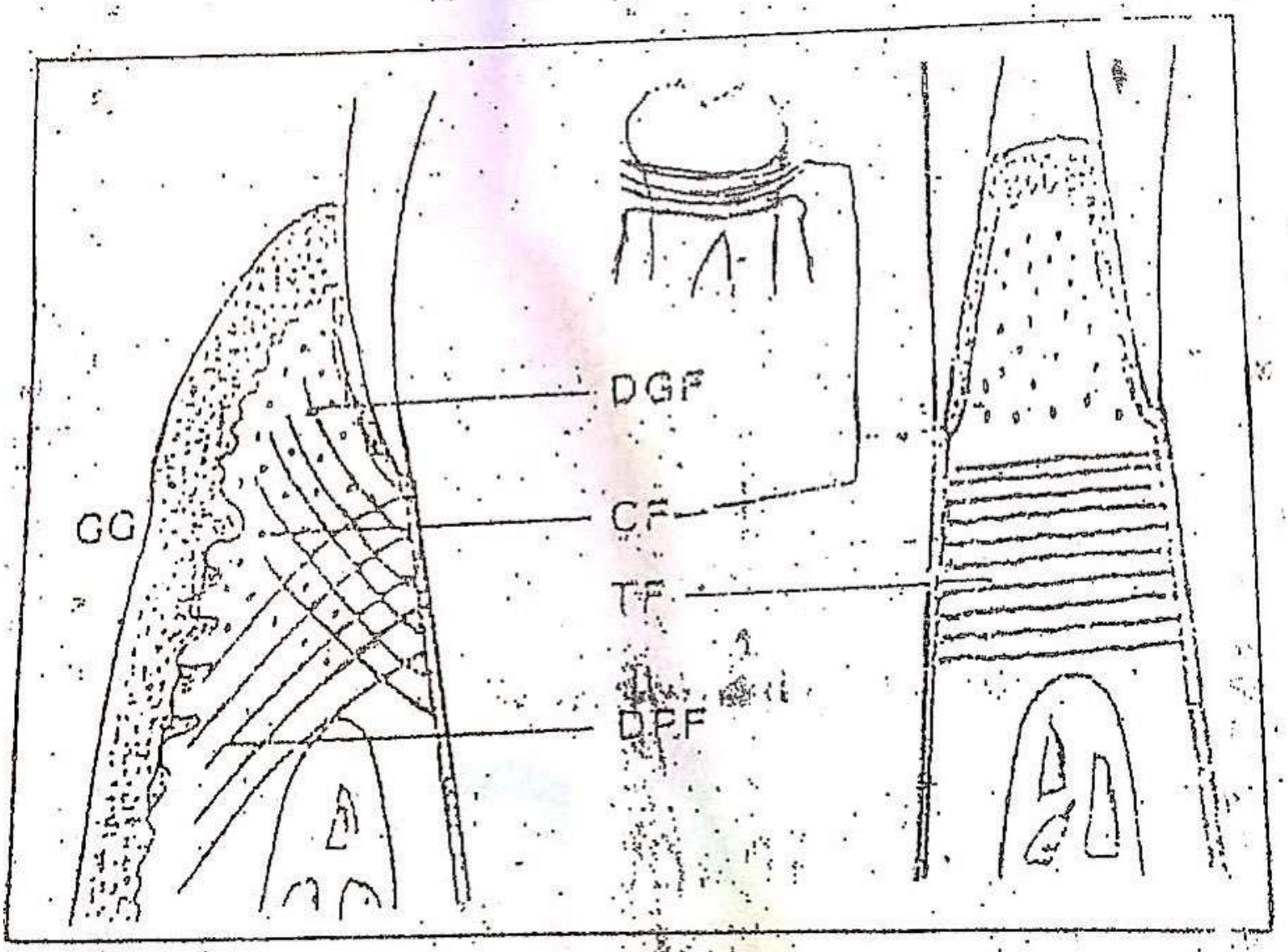
X+Y "Biologic Width" (BW) (cf. p. 319)

Periodontal Fiber Groups, Periodontal Ligament

The periodontal ligament (PDL) occupies the space between the root surface and the alveolar bone surface. The PDL consists of connective tissue fibers, cells, vasculature, nerves and ground substance. An average of 28,000 fiber bundles insert into each square millimeter of root cementum!

The building block of a fiber bundle is the 40–70 nm thick collagen fibril. Many such fibrils in parallel arrangement make up a collagen fiber. Numerous fibers combine to form collagen fiber bundles. These collagen fiber bundles (Sharpey's fibers) insert into the alveolar bone on one end and into cementum at the other (Feneis 1952). The most ubiquitous cells are fibroblasts, which appear as spindle-shaped cells with oval nuclei and numerous cytoplasmic processes of varying lengths. These cells are responsible for the synthesis and break-down of collagen ("turnover"). Cells responsible for the hard tissues are the cementoblasts and osteoblasts. Osteoclastic cells are only observed during phases of active bone resorption. Near the cementum layer, within the PDL space, one often observes string-like arrangements of epithelial rest cells of Malassez.

The periodontal ligament tissues are highly vascularized (p. 18) and innervated (p. 19).



" Arrangement of gingival fiber

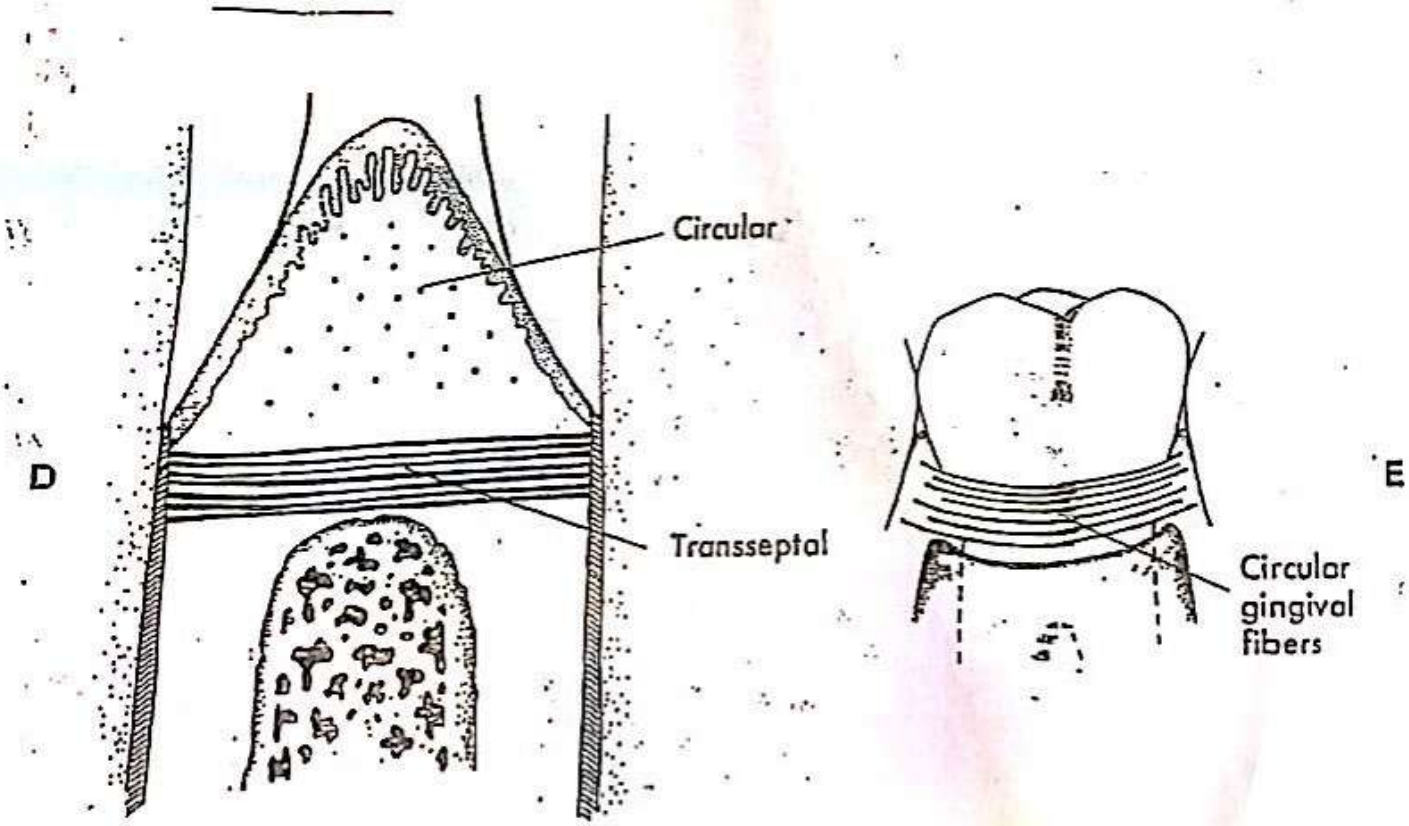


FIGURE 1-8, cont'd. C, Four layers (strata) of epithelial cells found in keratinized oral epithelium. D, Mesiodistal section of an interproximal area of the periodontium illustrating the transseptal and circular gingival fibers. E, Circular gingival fibers in the gingiva on the buccal aspect of a molar tooth.

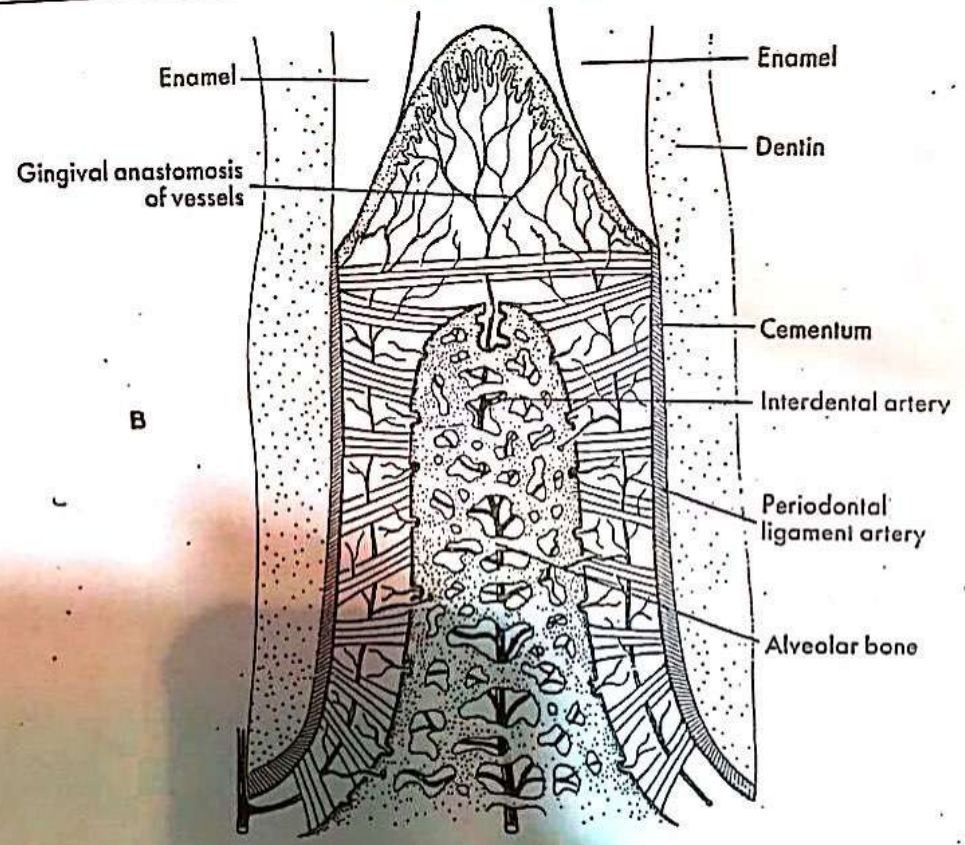


FIGURE 1-9, cont'd. B, Mesiodistal section of an interproximal area of the periodontium illustrating the interdental and the periodontal ligament arteries.

dark blue or brown color. Red to bluish-red changes are often characteristic of gingival inflammation.

Gingival consistency

In health the gingiva is usually resilient and firm because of the dense collagenous nature of

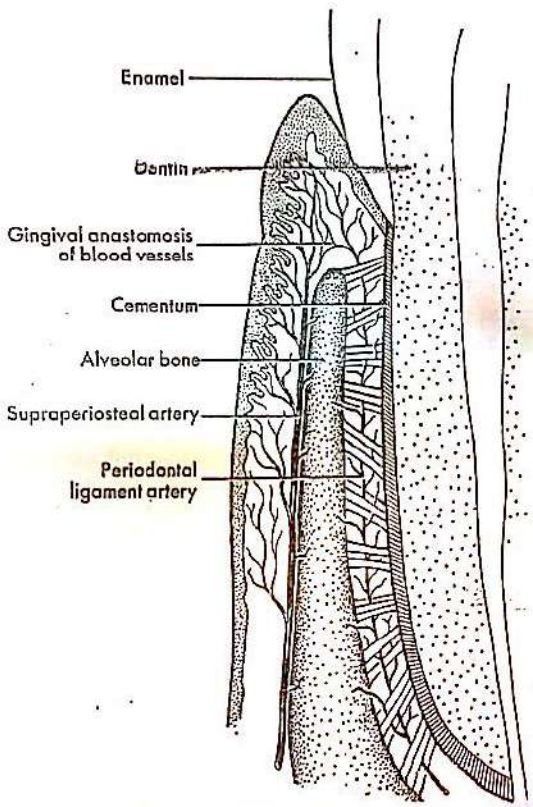


FIGURE 1-9. Blood supply to the periodontium. A, Supraperiosteal and periodontal ligament arteries on the facial or lingual surface of a tooth.

This fluid helps to mechanically clean the sul- to be an inflammatory exudate; however, small

Gingiv

FIGURE 1
illustrating

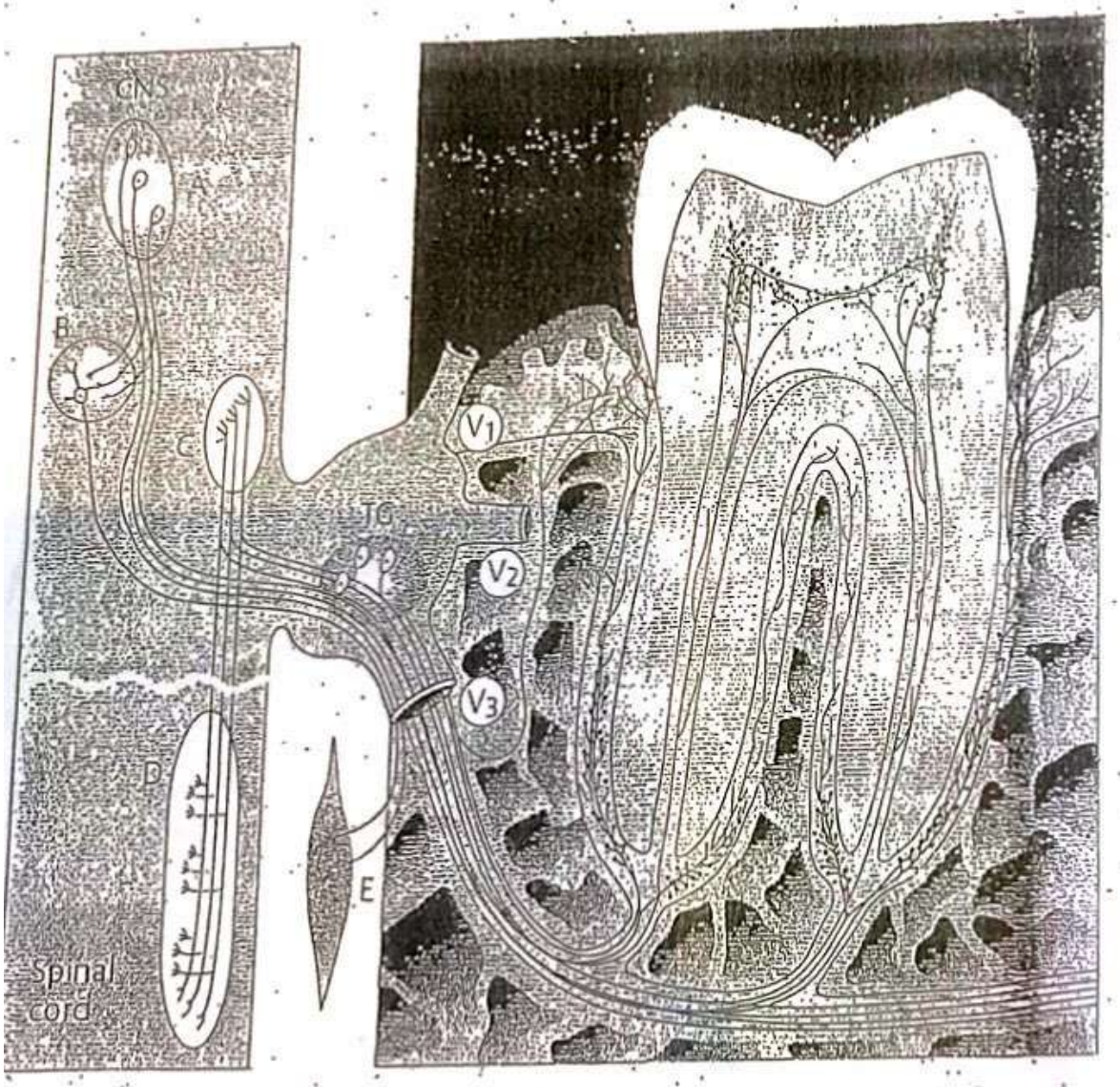
dark blue or br
changes are ofte
flammation.

Innervation of the Periodontium

The sensory innervation of the maxilla occurs via the second branch of the trigeminal nerve, and that of the mandible via the third branch. The following description of the neural distribution within the periodontal structures is based upon investigations by Byers (1985), Linden et al. (1994) and Byers & Takeyasu (1997).

The periodontium, especially the gingiva and periodontal ligament, contains "Ruffini-like" *mechanoreceptors* and *nociceptive nerve fibers*, in addition to the ubiquitous branches of the sympathetic nervous system.

The functions of these innervations are coordinated wi those of the dental pulp and the dentin. The stimuli threshold of the mechanoreceptors, which react to tac ti (pressure) stimulus as well as to the stretching of the per odontal ligament fibers, is very low. In contrast, the pain sensing nociceptive nerve endings have a relatively hig threshold. It is via these two separate afferent systems th "information" about jaw position, tooth movement speech, tooth contact during swallowing and chewin minor positional alterations (physiologic tooth mobility pain during unphysiologic loading, as well as injuries a



39 Innervation of a Mandibula Molar
Innervation of gingival and periodontal structures is via the mandibular nerve, the third branch of the trigeminal nerve.

Modified from *M. Byers*

- A Mesencephalic sensory neurons of the trigeminal nerve
- B Motor nucleus of the trigeminal
- C Sensory nucleus of the trigeminal
- D Spinal sensory trigeminal nucleus
- E Fibers of the masticatory musculature

TG Trigeminal ganglion (Gasserian ganglion) with its three branches:

- V_1 Ophthalmic
- V_2 Maxillary
- V_3 Mandibular

CNS Central Nervous System

ransmitted. In this way, various mechanoreceptors trans-
it "conscious reactions" via trigeminal ganglia to the sen-
ry nucleus of the trigeminal in the central nervous sys-
m, while unconscious reflexes transmit to mesencephalic
nsory neurons. These various receptors are localized in
ying regions of the periodontal structures: At the level of
middle of the root, one finds more receptors for up-take
'conscious reactions," whereas in the apical region there
more receptors for the unconscious reflexes whose sig-
transmit to the mesencephalic sensory neurons.

The junctional epithelium as well as the epithelia of the free and attached gingiva, neither of which are vascularized, are served by a dense network of nociceptive and tactile nerve endings. The same is true for the subepithelial, supracrestal gingival connective tissue.

Somatosensory perception in certain gingival diseases (e.g., ulcerative gingivoperiodontitis), as well as pressure and pain sensation during probing of the healthy gingival sulcus or periodontal pocket are the clinical manifestations of the innervation of gingival tissues.

Lec:- 4 periodontology

Cementum:-

It is a thin specialized calcified tissue covering the roots surfaces of the teeth.

It has many features similar to the bone tissue but differs from bone in the following aspects

It is microscopic organization.

Has no innervation

Has no blood or lymph vessels.

Doesnot undergo physiological remodeling

(resorption and deposition),but it is characterized by continuous deposition throughout life.

Functions of cementum:-

Anchorage of the tooth in the alveolus

To attach the PDL fibers to the teeth

To contribute to the process of repair after damage to the root surface and following regenerative periodontal surgical procedures.

Cemento-enamel junction (C.E.J)

Three types of relationships involving the cementum may exist at the C.E.J:-

Cementum overlaps the enamel (60%-65%)

Edge-to edge (butt joint (30%)

Cementum and enamel fail to meet (5%-10%)

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

There are two types of 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):-

Which 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 furcations of multirooted teeth . This type of cementum contains cells (cementocytes), but sharpey's fibers occupy a smaller portion of this type of cementum. Cellular cementum is less calcified than the acellular type.

Struetures of cementum:- cementum consist of

Fibrous elements (collagen fibers)

Cellular elements

Calcified interfibrillar matrix

Fibrous elements:-there are two types

a.Extrinsic fibers (sharpey's fibers): which are the embedded portion of the principal fibers of the PDL and are formed by the fibroblast cells . Sharpey's fibers make up most of the structure of acellular cementum and they are 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 are 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: The cells associated with

cementum are few and generally resides within the PDL.

a.cementoblast cells:responsible for the formation of both cellular and acellular cementum

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

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

d.cementoclast cells:these cells are responsible for extensive root resorption that lead to primary teeth exfoliation. Permenant teeth do not undergo physiologic resorption but

localized cemental resorption may occur which appears as concavities in the root surface and may be caused by local or systemic causes.local conditions include,trauma from occlusion, orthodontic movement, cyst and occur on mesial surfaces in association with mesial drift. Among systemic conditions are 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 cementoblast cells

Mineralization of cementum:occurs by the deposition of hydroxyapatite crystals,first within the collagen fibers,later upon the fiber surface and finally in the interfibrillar matrix.Cellular cementum is less calcified than acellular cementum and cementum mineralizalion is less than that of the bone, enamel and dentin

Development of cementum: Both cellular and acellular cementum are produced by cementoblast cells.Cementoid is first formed which is a non-calcified tissue containing collagen fibrils distributed in matrix.Cementum is characterized by continuous deposition and increase in thickness throughout life.A thin layer of cementum noted on recently erupted tooth will tend to increase thickness with age.Cementum formation is most 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 the cervical portion.Cementum is thicker in distal than in mesial surfaces because of functional stimulation from mesial drift over time.

Hypercementosis:refers to a prominant thickening of the cementum,it may be localized to one tooth e.g.tooth without antagonists or with periapical lesion , and sometimes affect the entire dentition that may occur in patients with 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 chronic periapical inflammation and occlusal trauma.

Alveolar process(AP)

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 the teeth and is gradually resorbed if the teeth are lost , thus it is tooth dependent structure

Functions of alveolar process:

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

2.provide the osseous attachment to the PDL fibers

3.distribute and resorb forces generated by mastication and other tooth contacts

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

Parts of the alveolar process:

1.Alveolar bone proper: it 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 blood vessels , lymphatics and nerves link the PDL with the cancellous bone thus it is called ((cribriform plate))

2.An external plate of cortical bone

3.Cancellous trabeculae or spongy bone: which is located in the space between the external cortical plate and alveolar bone proper, they meet and fuse to form the alveolar crest.cancellous bone, which act as supporting alveolar bone, with cortical bone surround the alveolar bone proper (APB)

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

Lamina dura:-the layer of ABP appears as white line surrounding the root of the tooth on radiographs.

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

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 2.81mm).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, e.g. teeth that are labially positioned in the arch will have thin labial radicular bone and thicker lingual radicular bone.

Bone marrow:- The cavities of all bones of new-born are occupied by red marrow while in the adult jaw occupied by fatty or yellow type of marrow , however foci of red bone marrow are seen in the jaw which may be visible radiographically as zones of radiolucency.

Common locations are the maxillary and mandibular molar and premolar areas.

Periosteum and Endosteum:

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

Endosteum:- the marrow spaces inside the bone are lined by endosteum, this tissue contains cells (osteoblasts)

Anatomical defects of bone:-

1.Fenestration(window):-This bony defect include isolated areas in which the root is not covered with bone and it does not extend to the marginal bone.

2.*Dehiscence:-This bony defect include the 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 these defects is not clear, but may be related to some factors such as, 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, consists of central canal called (Htaversian canal)whic in their center contains the blood vessel. These blood vessels surrounded by bone lamellae which arranged in concentric layers constitute the center of an osteon.The blood vessels in haversian canal are connected with each other by anastomoses running in the Volkmann's canals,so the nutrition of bone is secured by the incorporation of blood vessels in the bone tissue.

Bone cells:-

1.Osteoblast cells(bone forming cells):is responsible for the production of an organic matrix of bone which is consisting primarily of collagen fibers called (osteoid), this bone matrix undergoes mineralization by the deposition of minerals such as calcium and phosphate,which are subsequently transformed to hydroxyl apatite

2.Osteoclast cells:-These are large multinucleated cells found in concavities on the bone surface called (howship's lacunae) these cells responsible for bone resorption.

3.Osteocyte cells:- osteoblast cells that become trapped in the bone matrix and later on in the mineralized bone tissue , we call them osteocyte cells, they are located in the lacunae and are connected with the one another by extending processes into canaliculi through which they get nutrients and removes metabolic waste products.

Composition of the bone:-

Bone consists of 2/3 inorganic matter and 1/3 organic matrix.

The inorganic matter is composed principally of the minerals calcium and phosphate. The mineral salts are in the form of hydroxy apatite crystals.

The organic matrix consists mainly (90%) of collagen fibers

Remodeling of alveolar bone:-

Alveolar bone undergoes constant physiologic remodeling (resorption and formation)in response to external forces specially occlusal forces.

Teeth erupts and tend to move mesially throughout life to compensate for wearing in the proximal contact areas with age which become flat , this referred to as physiologic mesial migration , thus osteoclast cells and bone resorption occur in areas of pressure on the mesial surface and osteoblast cells with new bone formed in areas of tension on the distal surface.This process of resorption and formation of bone is called bone remodeling and it is important in the orthodontic treatment.

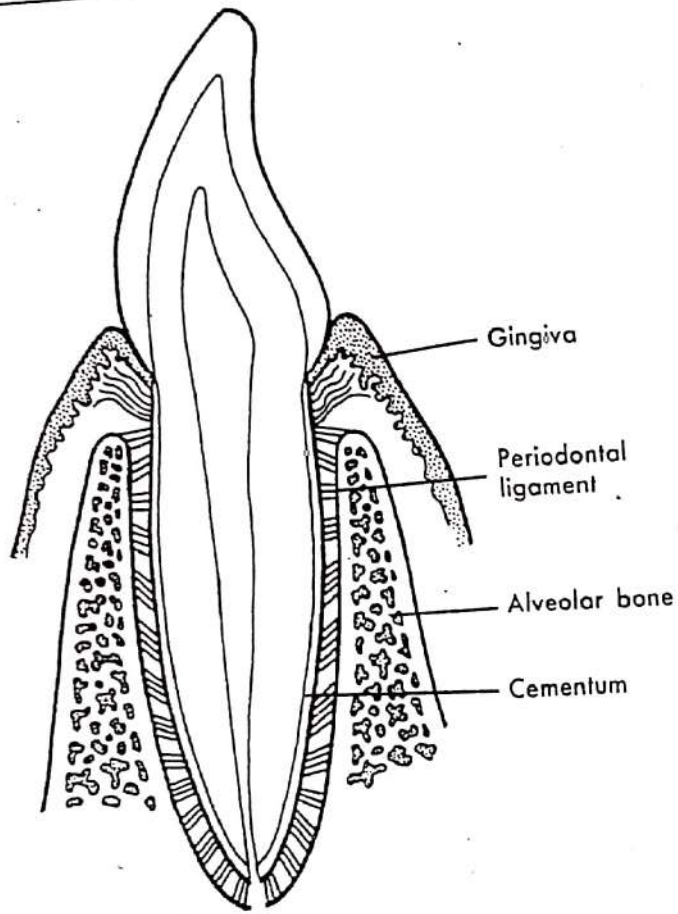
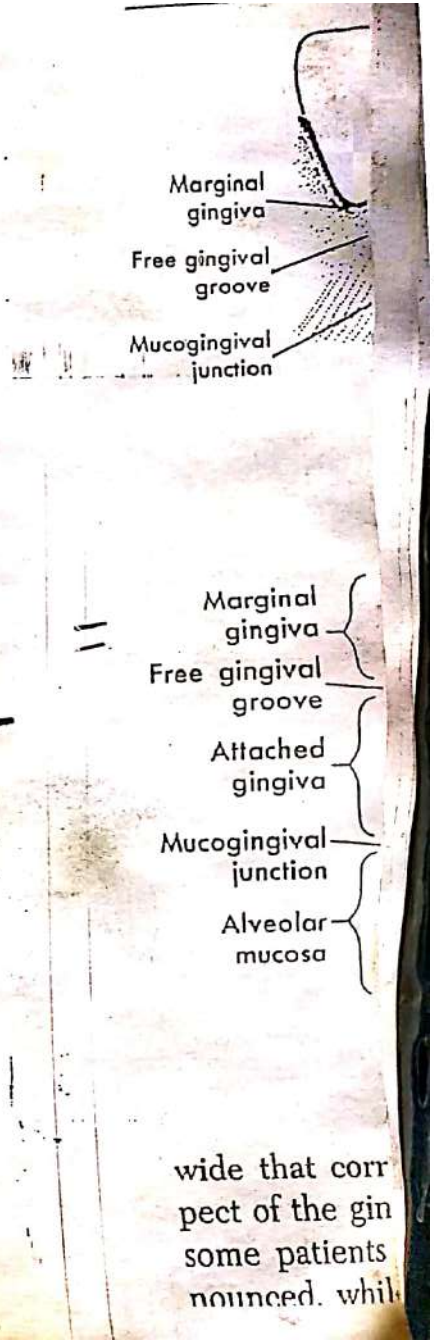
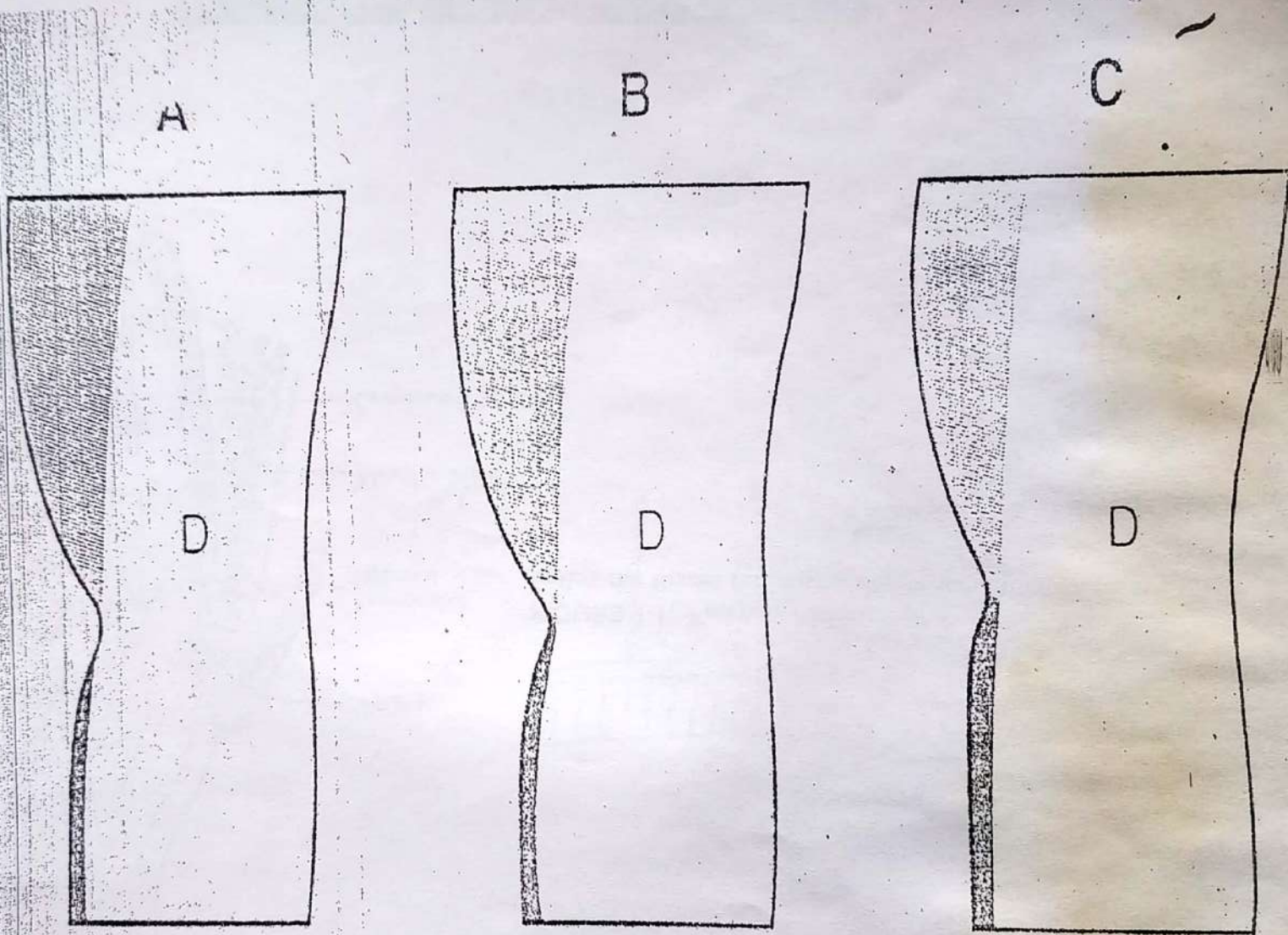


FIGURE 1-1. Faciolingual histological section illustrating the tissues comprising the periodontium.



wide that corr
pect of the gin
some patients
nounced, whil



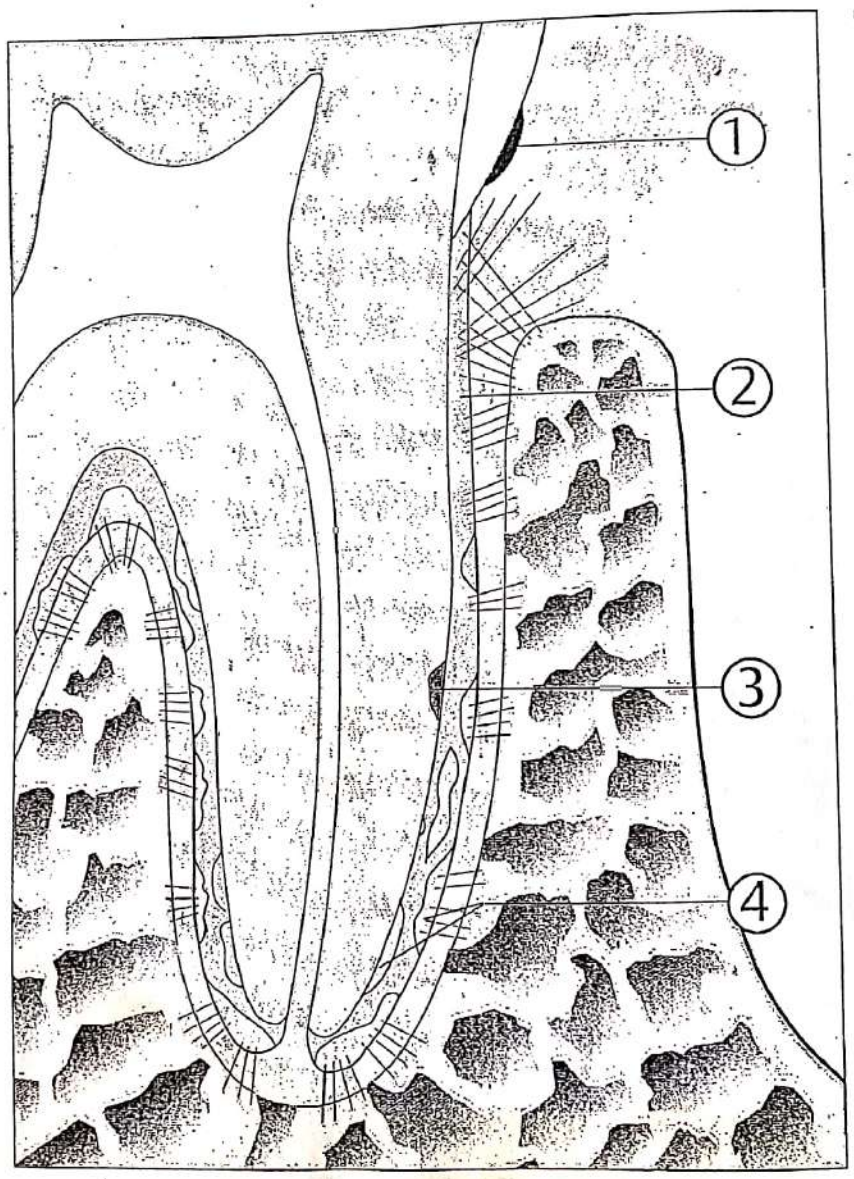
From a purely anatomic standpoint, root cementum is part of the tooth, but also part of the periodontium. Four types of cementum have been identified (Bosshardt & Schroeder 1991, 1992; Bosshardt & Selvig 1997):

1 Acellular, afibrillar cementum (AAC)
2 Acellular, extrinsic-fiber cementum (AEC)
3 Cellular intrinsic-fiber cementum (CIC)
4 Cellular mixed fiber cementum (CMC)

AEC and CMC are the most important types of cementum.

Fibroblasts and cementoblasts colla
of cementum. *Periodontal ligament*
lular extrinsic cementum. *Cementob*
trinsic cementum, and a portion of
cementum, and probably also acellu
Cementocytes evolve from the cen
come entrapped in cementum durir
result, cementocytes are observed
fiber cementum and frequently in c
tum (see also Cementum Formation

26 Types of Cementum—Structure, Localization and Development



1 **Acellular, Afibrillar Cementum** (AAC; red) AAC is formed at the most cervical enamel border following completion of pre-eruptive enamel maturation, and sometimes also during tooth eruption. It is probably secreted by cementoblasts.
2 **Acellular, Extrinsic-fiber Cementum** (AEC; green) AEC forms both pre- and post-eruptively. It is secreted by fibroblasts. On the apical portions of the root, it comprises a portion of the mixed-fiber cementum.
3 **Cellular, Intrinsic-fiber Cementum** (CIC; blue) CIC is formed both pre- and post-eruptively. It is synthesized by cementoblasts, but does not contain extrinsic Sharpey's fibers.
4 **Cellular, Mixed-fiber Cementum** (CMC; orange/green) CMC is formed by both cementoblasts and fibroblasts; it is a combination of cellular intrinsic-fiber cementum and acellular extrinsic-fiber cementum.

See p

Acellular Extrinsic Cementum (AEC)

The AEC is primarily responsible for the anchorage of the tooth in the alveolus. It is found in the cervical third of all deciduous and permanent teeth. The AEC consists of tightly packed and splaying fiber bundles (Sharpey's fibers), which are embedded in the calcified cementum.

The collagenous structures of cementum and dentin intertwine with each other during root formation and before calcification. This phenomenon explains the tight connection between these two hard tissues.

Cellular Mixed-fiber Cementum (CM

The CMC is also of importance for th
in its alveolus. But it is only the
cementum portion (AEC) within th
which the Sharpey's fibers secreted
therefore affix the tooth. CMC is la
horizontally to the root surface. The
mentoblasts contains high nun
(Fig. 30, left). The CMC is also tigh
because of the intertwining of th

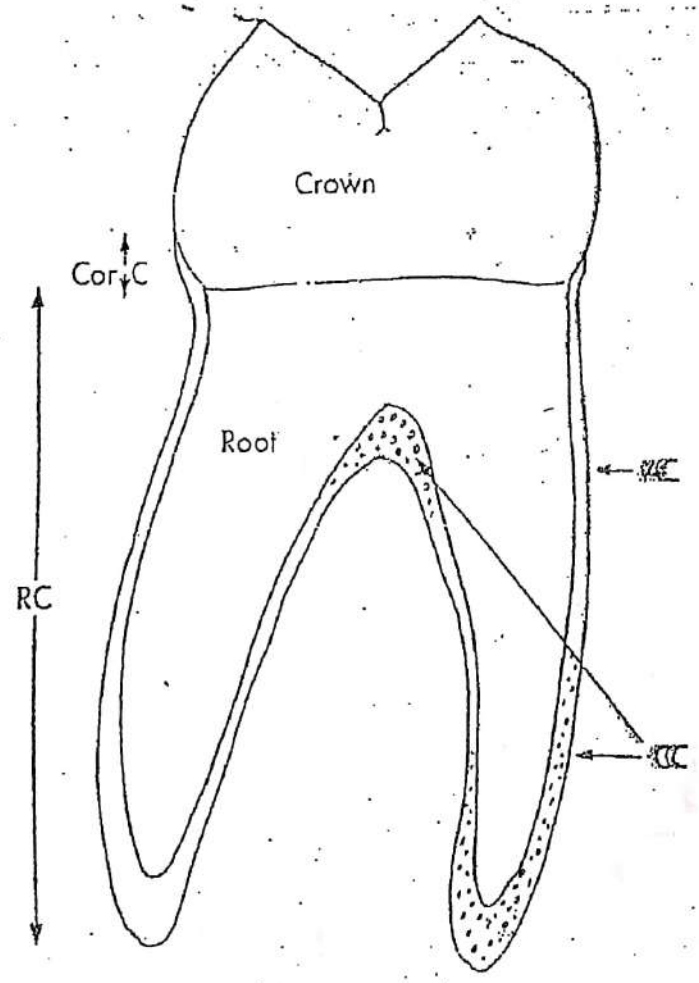
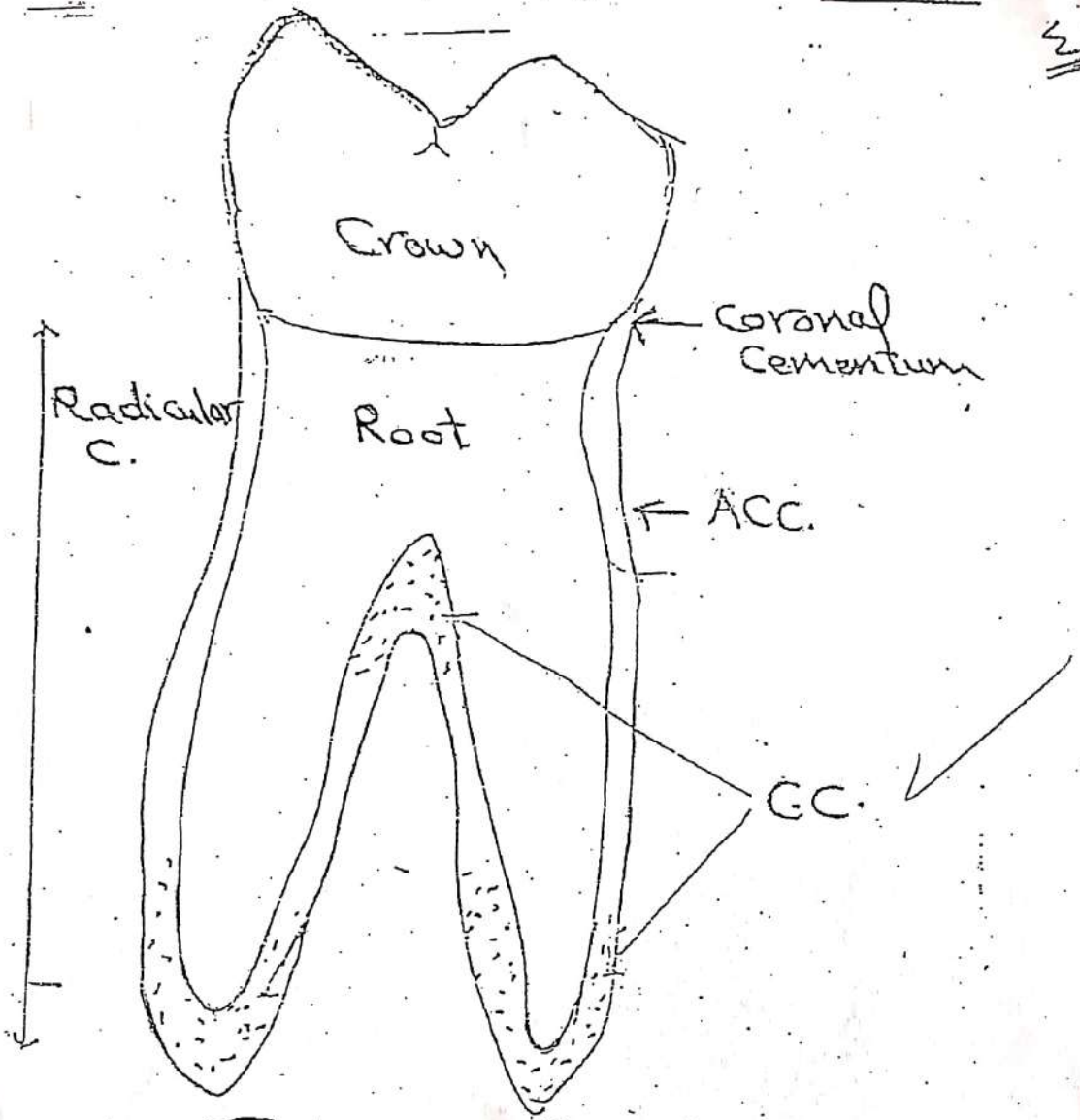
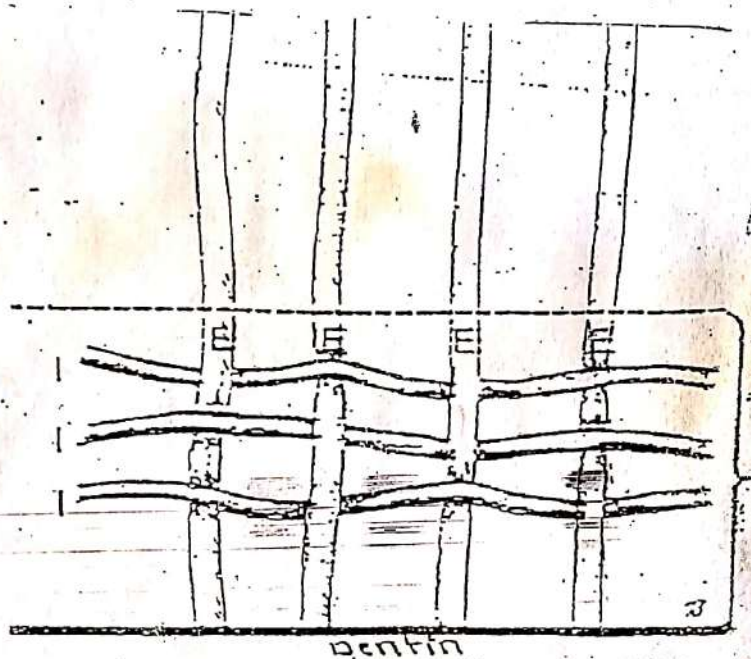


Fig. 4-1. Diagram illustrating the distribution of cementum on the tooth surface. The cementum covering the anatomic crown of the tooth is coronal cementum *(Cor C)*. The roots are covered with radicular cementum *(RC)*. Cellular cementum *(CC)* can be found on the apical third of the roots and within furcations. Acellular cementum *(AC)* covers the coronal two thirds of the root and extends over the cervical portion of the crown.



Types of C.



Fibers of C.

bers

l fibers run from the
alveolar bone in an api-
ontal fibers are located
ar crestal fibers and run
ne tooth to the alveolar
rs, which comprise the
coronal direction from
The apical fibers radiate
e tooth to the adjacent

ncipal fibers are embed-
ne tooth side and in the
n the opposite side. The
the principal fibers are
ers (Fig. 1-10).
lagen fibers is a looser
sue within which cells,
nd nerves are found.
ound in the periodontal
from three sources: the
ly the teeth, the vessels
eolar bone, and the gin-

principal cells found in
nt:
mesenchymal cells

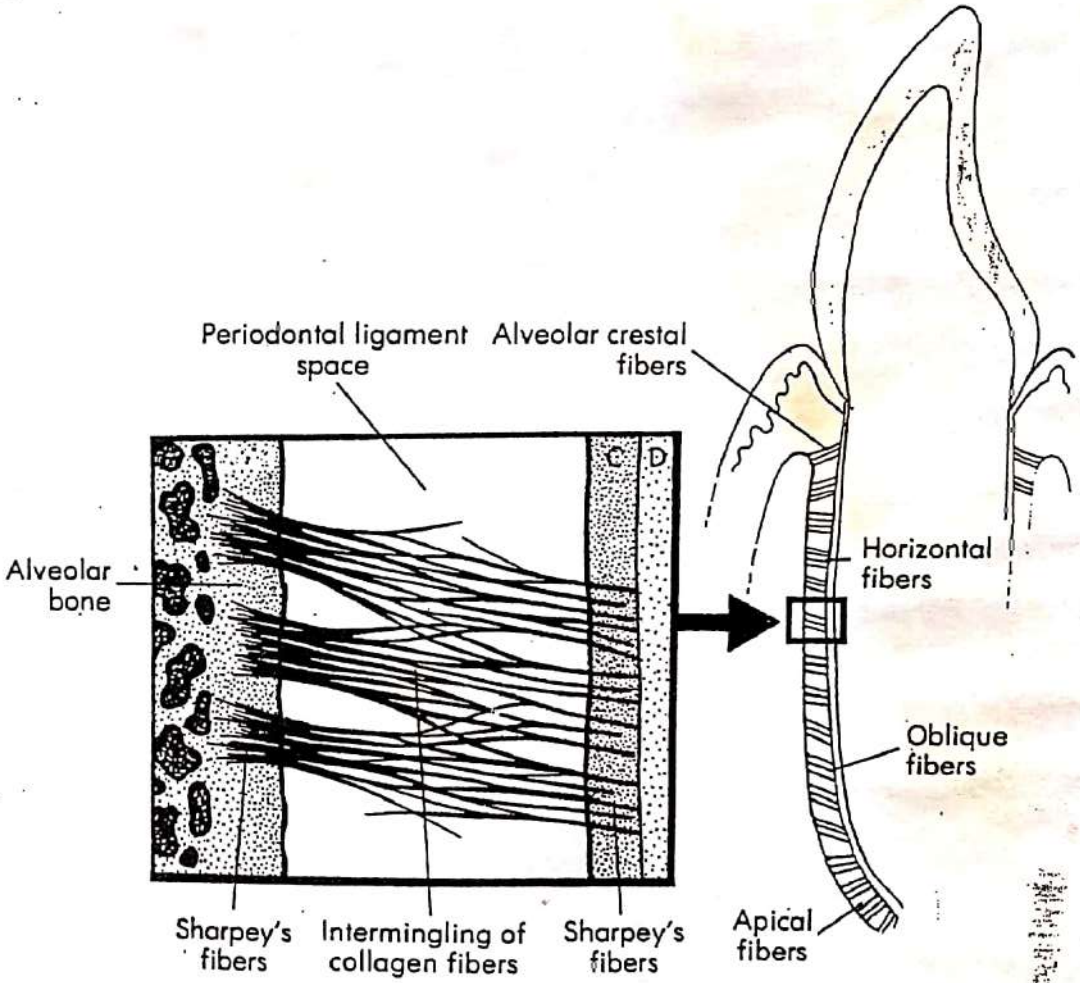


FIGURE 1-10. Principal groups of periodontal ligament fibers and their terminal portions, Sharpey's fibers.

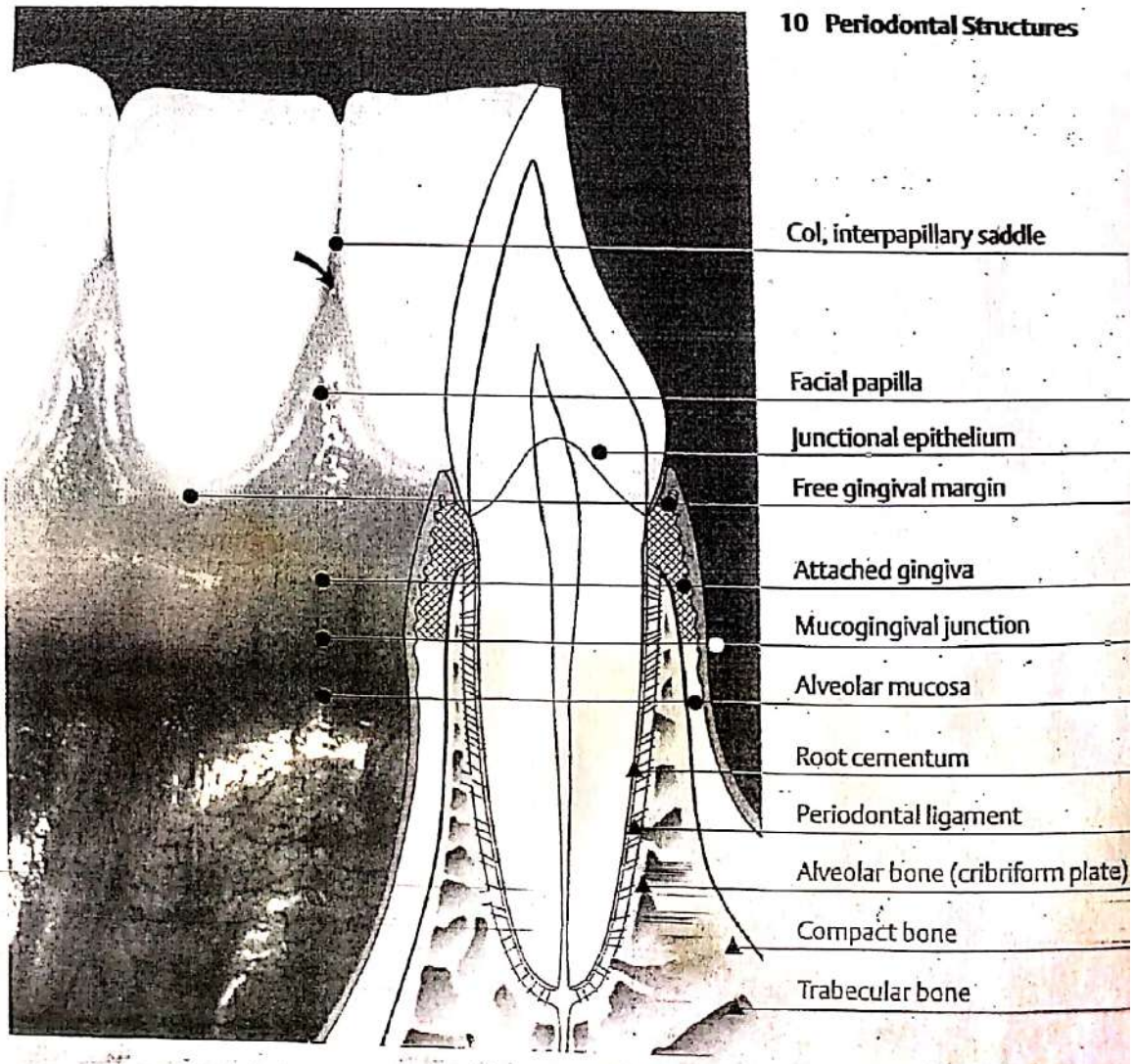
2. Formative function
3. Nutritive function
4. Sensory function

The nutritive function is performed by the blood supply of the periodontal ligament when it carries food products to the cells of the area

tructural biology" is a general term referring to the classical macromorphology and histology o
ssues, as well as their function, including the biochemistry of the cells and the intercellular sub
ances.

asic knowledge of the normal structural biology of periodontal tissues and their dynamics (medi
or-guided homeostasis, "turnover") is a prerequisite for full understanding of pathobiologica
anges in the periodontium, which can involve adaptations of the normal structures or an imbal
ce of otherwise normal functions (Schroeder 1992).

e term "periodontium" encompasses four different soft and hard tissues: gingiva, root cemen
um, alveolar bone, and the periodontal ligament, which attaches root cementum to bone. Each o
ese four tissues can be further differentiated in terms of structure, function and localization.



ft Side:

ansmission electron photomi-ograph (TEM) of root forma-n in humans (ca. 6-year-old)
s TEM depicts the growing de-arcation between dentin, ce-entum and periodontal liga-ent during root formation. tial mineralization of the "ce-entoid" directly apposed to the ntin, with penetrating collagen ers and fibroblast-like cemen-blasts, which are involved in the rmation of acellular exogenous er cementum.

Dentin
Cementoid
Radiating collagen fibers
Cementoblast (fibroblast-like) building acellular exogenous fiber cement

urtesy *D. Bosshardt, Schroeder*

The alveolar processes of the maxilla and the mandible are tooth-dependent structures. They develop with the formation of and during the eruption of the teeth, and they atrophy for the most part after tooth loss. Three structures of the alveolar process may be discriminated:

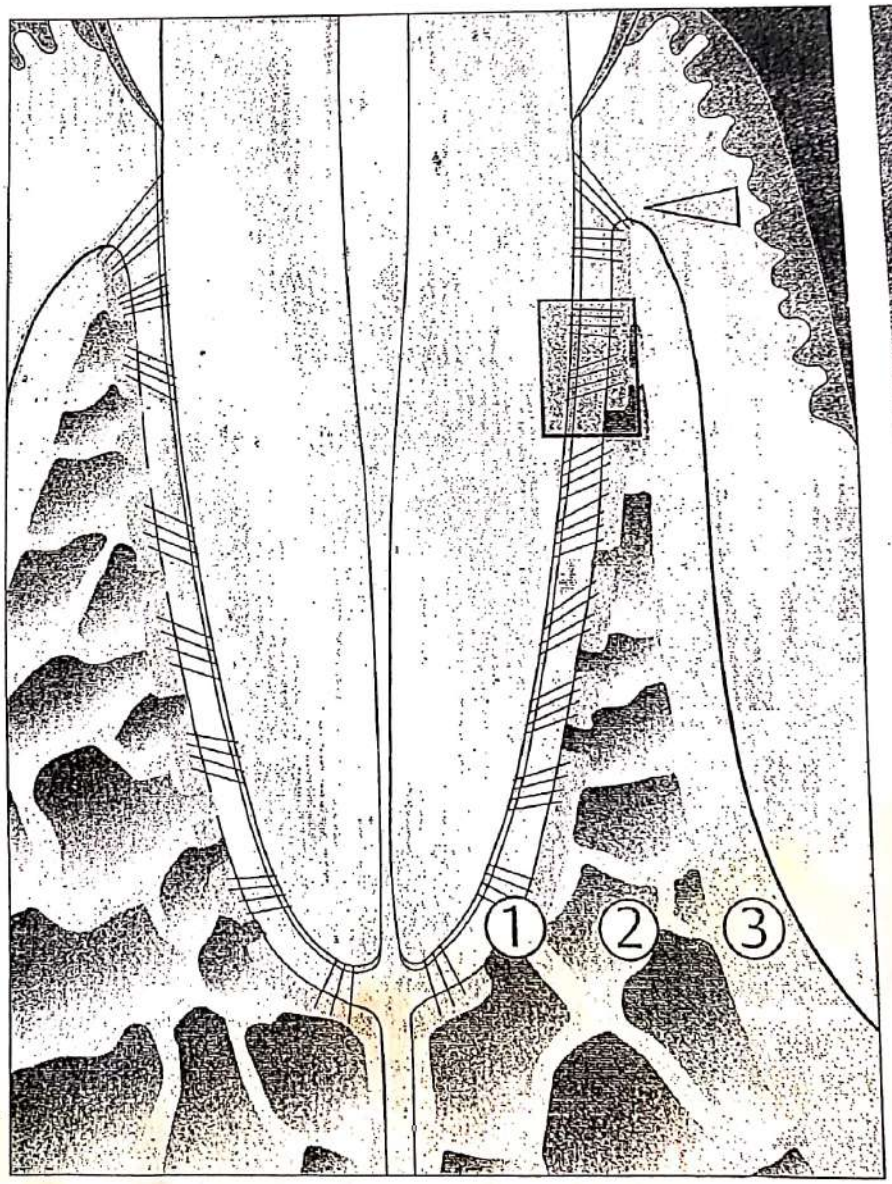
- Alveolar bone proper
- Trabecular bone
- Compact bone

the entrance to the alveoli, the alve
the cribriform plate, the alveolar b
the alveolar wall and is approxima
is perforated with numerous sr
canals) through which vessels as w
into and exit the periodontal ligam
bone occupies the space between c
lar bone proper. The distance betw
and the alveolar crest is referred tc
of 2–3 mm (BW; Gargiulo et al. 196

31 Osseous Support Apparatus
The tooth-supporting alveolar process consists of the alveolar bone (1), trabecular bone (2), and compact bone (3). Alveolar bone and compact bone join at the margin to form the alveolar crestal bone (arrow). In this region, the alveolar process is often extremely thin, especially on the facial aspect, and unsupported by trabecular bone (Fig. 36).

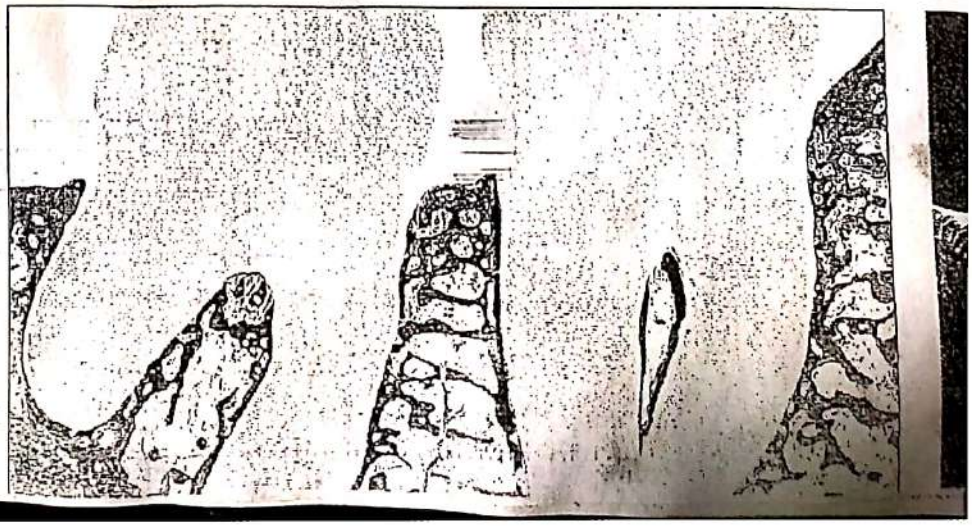
Right: Histologic section (HE, x10) through the periodontium (the location is indicated by the rectangle superimposed in the main figure). On the right side of the picture, the alveolar bone with its osteons and a Haversian canal is clearly visible. Bundle bone has been deposited adjacent to the structures on the periodontal aspect.
The periodontal ligament is cell-rich, and exhibits a thin layer of cementum-forming fibroblasts along the acellular, extrinsic-fiber cementum (left).

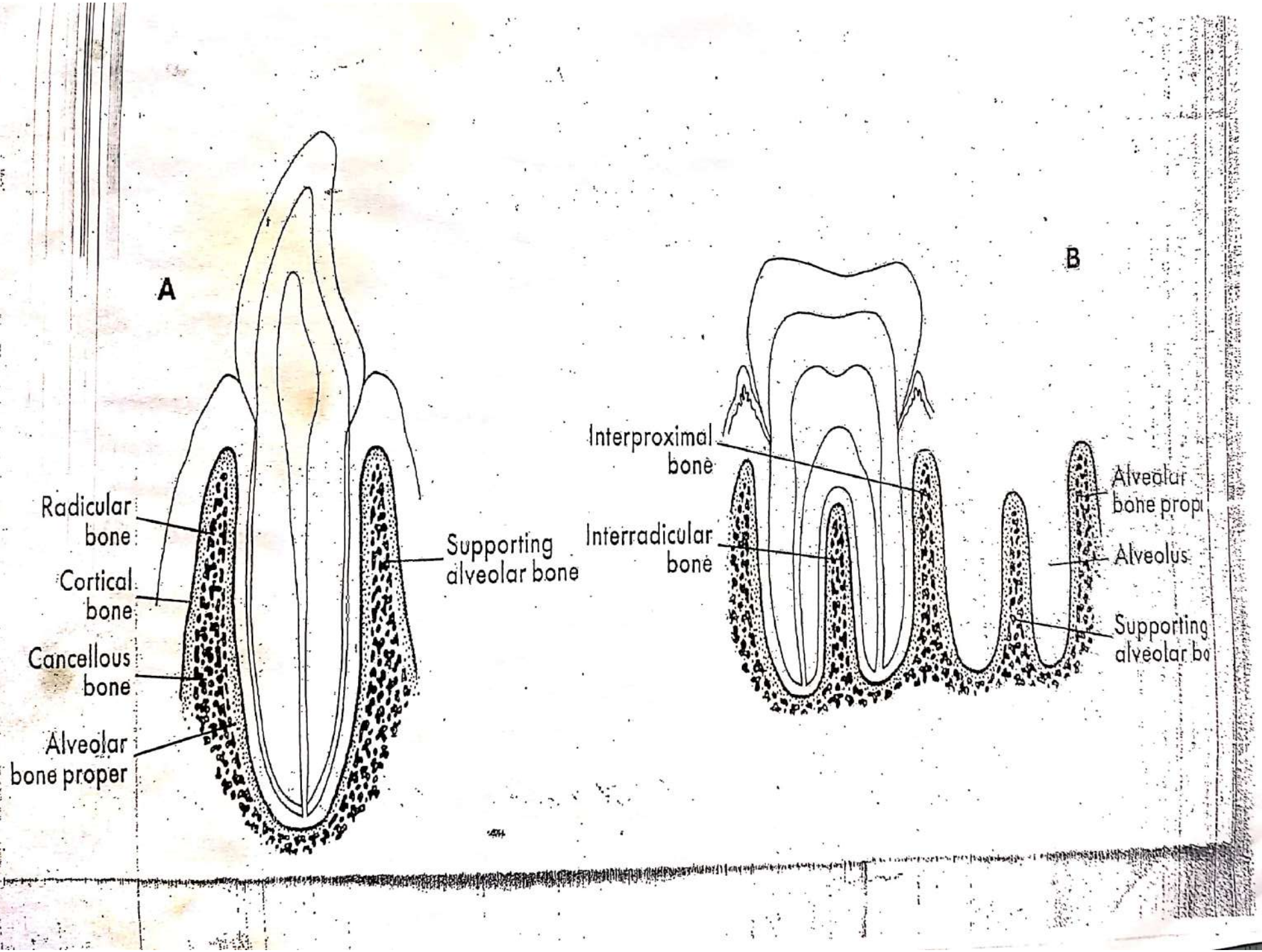
Histology courtesy *H. Schroeder*

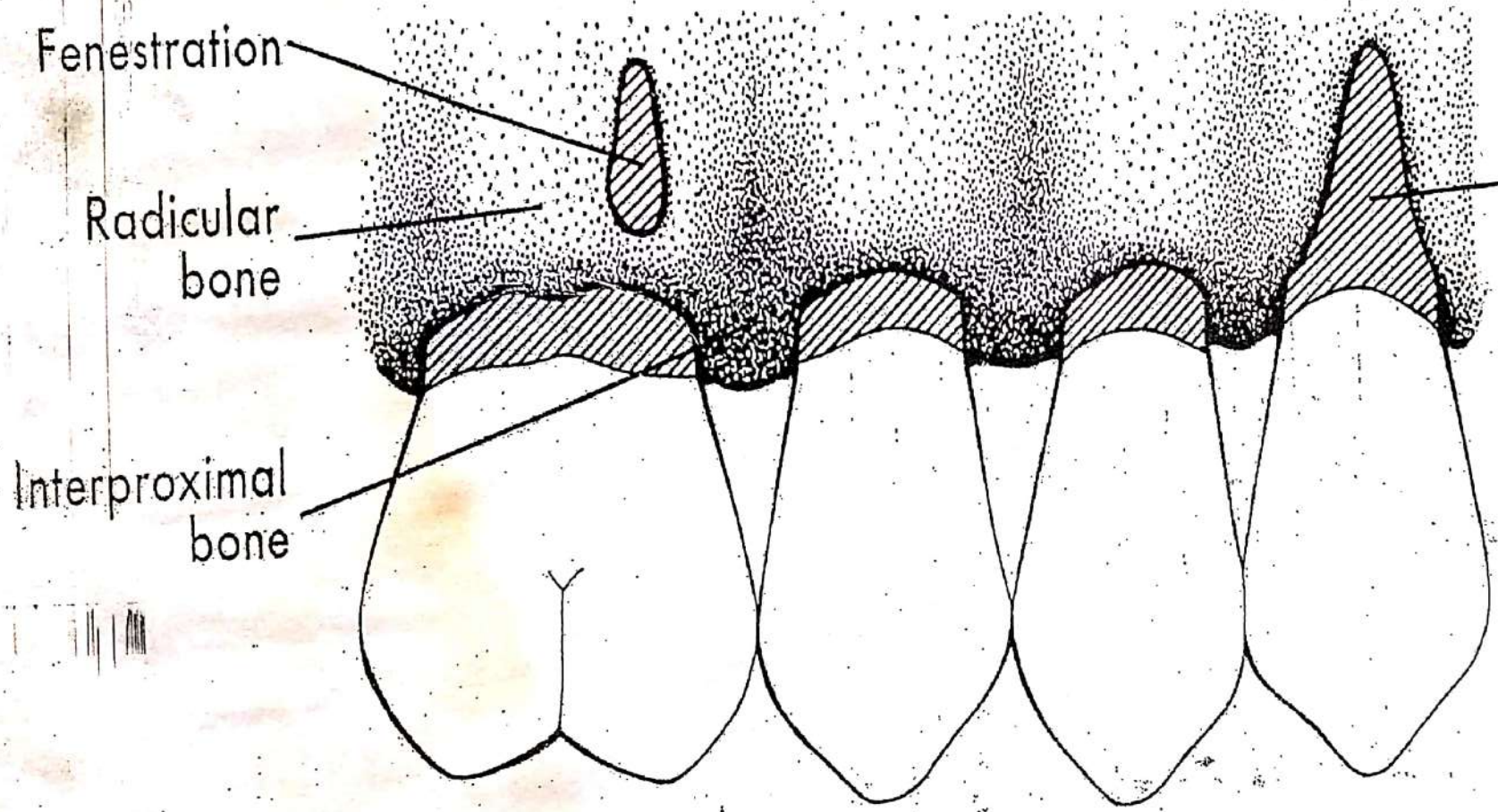


32 Mandibular Alveolar Process in Sagittal Section
In this histologic section (H and E, x1) the elegant structure of the trabecular bone and the more or less large marrow spaces are visible. The *alveolar bone proper* is depicted only as a very thin, often partially broken line.

Right: In this transilluminated bone preparation, it becomes clear that the alveolar bone is per-







A

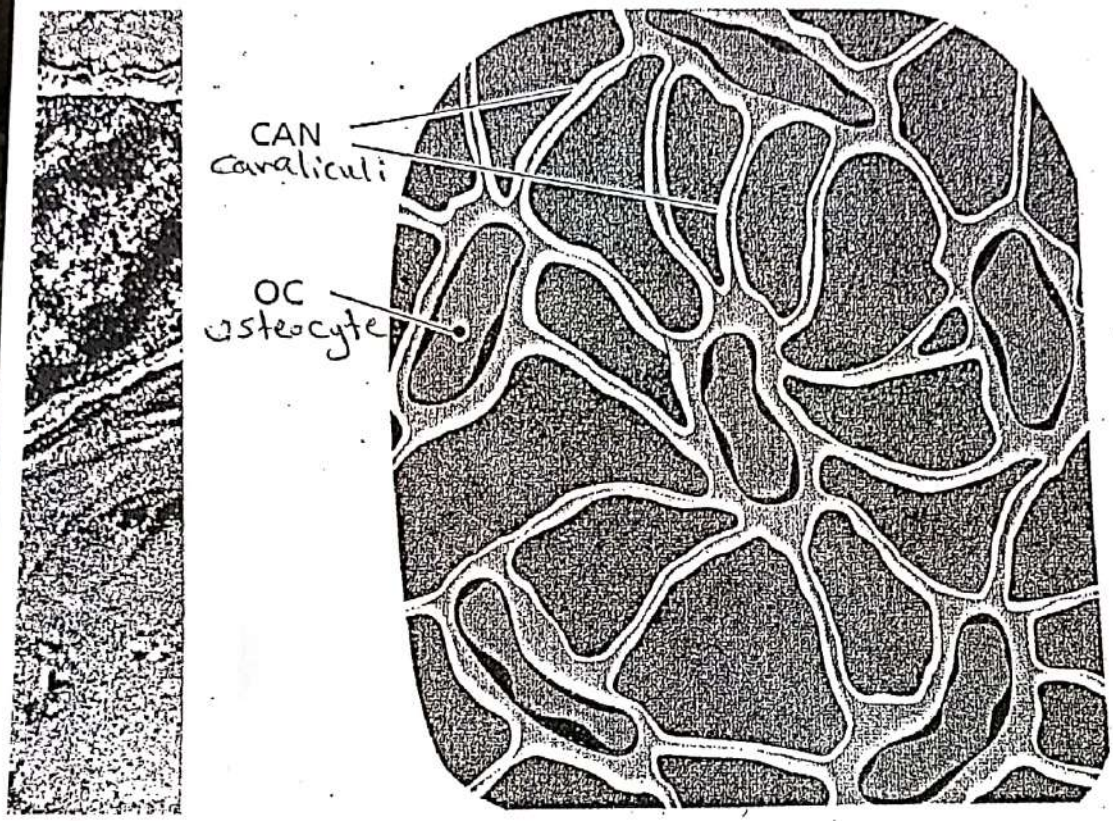
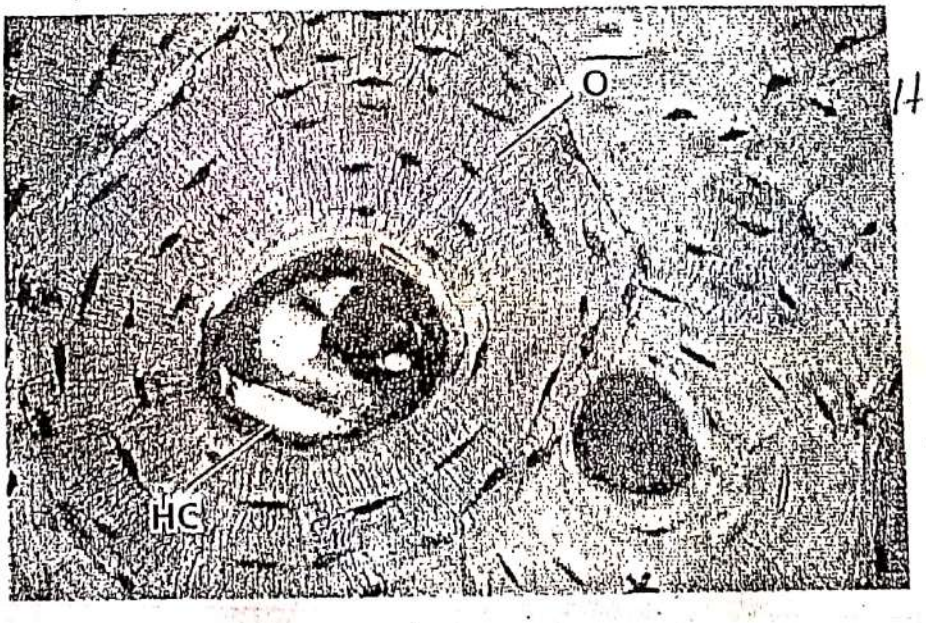


Fig. 1-82.



O: osteon
HC: Haversian canal

Fig. 1-83.

ıe surface
toplasmic
ineralized
has been
cells and
× 10 cm,
This enor-
regulator,
rum phos-
chanisms.

as osteons
l but also
ıe bone is
l vessels in
urrounded

22

movement of the teeth implies remodeling of

Lec: 5 محاضرة 5 بيريو

periodontics رابع اسنان

Dental Plaque Biofilms

Bacteria are the primary etiologic agents in periodontal disease. More than 500 distinct microbial phenotypes can be present in dental plaque. These bacteria have evolved to survive in the environment of the tooth surface, gingival epithelium, and oral cavity.

Macroscopic Structure and Composition of Dental Plaque

Dental Plaque is defined as a soft yellow –grayish deposits that form the biofilm adhering tenaciously to the tooth surface or other hard surfaces in the oral cavity including removable and fixed restorations. Dental plaque consist primarily of microorganisms + intercellular matrix along with scattering epithelial cells ,leukocytes and macrophages. The presence of tough extracellular matrix makes it impossible to remove by rinsing or the use of sprays.

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 a biofilm of complex and dynamic microbial community it contain areas of high and low bacterial biomass interlaced with aqueous channels of different sizes which are 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 is resistant to mechanical removal as well as antibiotics . A biofilm is a fascinating structure , which functions like multicellular organisms characterized by shedding of bacterial surface components (antigens, which can activate a host immune response) and release of various toxins (endotoxin, which activate a host inflammatory response) which cause host tissue damage . the biofilm also plays a major role in protecting the colonizing species from host defense mechanisms.

Structure of Biofilm: Biofilm are composed of microcolonies of bacterial cells (15%-20% by volume) that 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 "circulatory system " some of the functions of the biofilm are dependent on the ability of bacteria and microcolonies with in the biofilm to communicate with one another . this activity is called 'quorum sensing" in which bacteria secrete a signaling molecule that accumulates in the local environment and triggers a response such as a change in the expression of specific genes once they reach a critical threshold concentration. The threshold concentration is reached only at a high-cell density, and therefore bacteria sense that the population has reached a critical mass, or quorum. There is some evidence that intercellular communication can occur after cell-cell contact and in this case, may not involve secreted signaling molecules.

Plaque is differentiated 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 the organized structure of dental plaque and are easily displaced with a water spray.

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

Dental plaque is classified as supragingival or subgingival based on its position on the tooth surface .

Supragingival plaque is found at or above the gingival margin ; the supragingival plaque in direct contact with the gingival margin is referred to as ***marginal plaque.***

Subgingival plaque is found below the gingival margin between the tooth and the gingival sulcular epithelium . Morphologic studies indicate a differentiation of

tooth- associated regions and tissue – associated regions of subgingival plaque(epithelial associated subgingival plaque and connective tissue associated subgingival plaque) . The different regions of plaque are significant to different processes associated with disease of the teeth and periodontium . for example marginal plaque is of prime importance in the development of gingivitis. Supragingival plaque and and tooth- associated subgingival plaque are critical in calculus formation and root caries , whereas tissue-associated subgingival plaque is important in the soft tissue destruction that characterizes different form of periodontitis. Dental plaque is composed primarily of microorganism . one gram of plaque (wet weight) contains approximately 2×10^{11} bacteria. the number of bacteria in supragingival plaque on a single tooth surface can exceed 109 cells and its count can range from 10^3 bacteria in a healthy crevice to $>10^8$bacteria in a deep pocket. **Non bacteria microorganisms** that are found in plaque include yeasts, Protozoa and viruses. The microorganisms exist within an intercellular matrix that also contains a few host cells such as epithelial cells, macrophages and leukocytes.

The inter cellular matrix, estimated to account 20% to 30% of the plaque mass , consist of organic and inorganic materials derived from saliva, gingival crevicular fluid and bacterial products , organic constituents include polysaccharides, proteins, glycoprotiens and lipid material. The inorganic component of plaque 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 primarily saliva while for the subgingival plaque is derived from crevicular fluid which is a serum transudate.

Formation of Dental plaque:

Dental plaque may be visualized on teeth after 1 to 2 days with no oral hygiene measures. Movement of tissues and food materials over the teeth results in mechanical removal of plaque such removal is effective on the coronal two thirds of

the tooth surface . thus 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 colonization by bacteria
3. Secondary colonization 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 component of saliva and crevicular fluid as well as bacterial and host tissue cell products and debris. The pellicle on tooth surface consists of more than 180 pepides, proteins, and glycoproteins including keratins, mucins, histidine- rich proteins ,proline rich proteins, phosphoprotiens. The mechanisms involved in enamel pellicle formation include electrostatic, van der Waals and hydrophobic forces. Salivary pellicle can be detected on clean enamel surfaces within (1 minute) . By 2 hours , the pellicle is essentially in equilibrium between adsorption and detachment , although further pellicle maturation can be observed for several hours.

Pellicles functions

1. Protective barrier , providing lubrication for the surfaces and preventing tissue desiccations
2. They provide a substrate to which bacteria in the environment 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 peroxidases , amylase, may affect the physiology and metabolism of adhering bacterial cells.

Initial Colonization of the tooth surface

Tooth brushing removes most but not all bacteria from the exposed surfaces of teeth. However, recolonization begins immediately and bacteria can be detected within 3 minutes of introducing sterile enamel into the mouth. The

initial steps of transport and interaction with the surface are essentially non specific (they are the same for all bacteria). The proteins and carbohydrates that are exposed on the bacterial cell surface become important once the bacteria are in loose contact with the acquired enamel pellicle . it's a specific molecules termed (adhesin) on the bacterial surface that interact with receptors in the dental pellicle that determine whether a bacterial cell will remain associated with the surface. Only a small proportion of oral bacteria possess adhesins that interact with receptors in the host pellicle and these microorganisms are generally the most abundant bacteria in biofilms on tooth enamel shortly after cleaning . over the first 4 to 8 hours , 60%to 80% of bacteria present are members of the genus Streptococcus. Other bacteria commonly present at this time include species that cannot survive without oxygen(obligate aerobes) such as Haemophilus spp and Neisseria spp., as well as organisms that can grow in the presence or absence of oxygen (facultative anaerobes) including Actinomyces spp and Veillonella spp. These species are considered the **primary colonizers** of the tooth surface the primary colonizers provide new binding sites for adhesion by other oral bacteria. The metabolic activity of the primary colonizers modifies the local microenvironment in ways that can influence the ability of other bacteria to survive in the dental plaque biofilm. For example , by removing oxygen, the primary colonizers provide conditions of low oxygen tension that permit the survival and growth of obligate anaerobes.

Secondary colonization and plaque maturation

The primary colonizing bacteria adhered to the tooth surface provide new receptors for attachment by other bacteria , in process known as (coadhesion) together with growth of adherent microorganisms, coadhesion leads to the development of microcolonies and eventually to a mature biofilm. Different species or even different strains of a single spesies have distinct sets of coaggregation partners , secondary colonizers microorganisms include *Prevotella intermedia* , *Capnocytophaga spp.*, *Fusobacterium nucleatum* , and

Prophyromonas gingivalis . *Fusobacteria* coaggregate with all other human oral bacteria while *Veilloella* spp , *Capnocytophaga* spp. And *Prevotella* spp bind to *streptococci* 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- characterized interactions of secondary colonizers with early colonizers 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 primarly gram- positive organisms to high numbers of gram negative bacteria . therefore, in the later stages of plaque formation coaggregation between different gram- negative species is likely to predominate. Examples of these types of interaction are the coaggregation of *F. nucleatum with P. gingivalis or Treponema denticola*

Microscopic structure and physiologic properties of Dental plaque

Supragingival plaque typically demonstrates a stratified organization of a multilayered accumulation of bacterial morphotypes. Gram-positive cocci and short rods predominat at the tooth surface, whereas gram-negative rods and filaments, as well as spirochetes, predominate in the outer surface of the mature plaque mass. Highly specific cell-to-cell interactions are also evident from the "corncob" structures" often observed (Fig. 6-5). Corncob formations have been observed between rod-shaped bacterial cells (e.g., Bacterionema matruchotii or F. nucleatum) that form the inner core of the structure and coccal cells (e.g., streptococci or P. gingivalis) that attach along the surface of the rod shaped cell .

In general, the subgingival microbiota differs in composition from the supragingival plaque, primarily because of the local availability of blood products and a low reduction-oxidation (redox)potential, which characterizes the anaerobic environment. The environmental parameters of the subgingival region differ from those of the supragingival region. The gingival crevice or pocket is bathed by the

flow of crevicular fluid, which contains many substances which bacteria may use as nutrients. Host inflammatory cells and mediators are likely to have considerable influence on the establishment and growth of bacteria in the subgingival region. . Both morphologic and microbiologic studies of subgingival plaque reveal distinctions between the tooth-associated and soft tissue-associated regions of subgingival plaque(Epithelium -associated subginival plaque and connective tissue associated plaque)

The tooth-associated cervical plaque, adhering to the root cementum , does not markedly differ from that observed in gingivitis. . At this location, filamentous microorganisms dominate, but cocci and rods also occur. This plaque is dominated by gram positive rods and cocci, including S. mitis, S. sanguinis, Actinomyces oris. . However, in the deeper parts of the pocket, the filamen tous organisms become fewer in numbers, and in the apical portion they seem to be virtually absent. Instead, the microbiota is dominated by smaller organisms without a particular orientation.

The apical border of the plaque mass is separated from the junctional epithelium by a layer of host leukocytes, and the bacterial popultion of this apical tooth-associated region shows an increased concentration of gram-negative rods.

The layers of microorganisms facing the soft tissue lack a definite intermicrobial matrix and contain primarily gram-negative rods and cocci as well as large numbers of filaments, flagellated rods, and spirochetes.

Host tissue cells (e.g., white blood cells and epithelial cells) may also be found in this region Bacteria are also found within the host tissues, such as in the soft tissues and within epithelial cells, as well as in the dentinal tubules.

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

Sub gingial plaque resembles supragingival plaque but vary in types of microorganisms.

Lec:

Microorganisms of subgingival plaque (Summary)

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 sub gingival plaque and the tooth there is an electron dense organic material called Cuticle which may contain remnant of epithelial attachment lamina which connect the junctional epithelial to the tooth .

Biofilms also form on artificial surfaces exposed to the oral environment such as prostheses and implants. A large series of papers compared the microbiota in pockets around teeth with those around implants of partially edentulous patients. The similarities were striking.

Factors Affecting Supragingival Dental dental plaque formation

During the first 24 hours starting from a clean tooth surface, plaque Growth is negligible from a clinical viewpoint (<3% coverage of the vestibular tooth surface, which is an amount nearly undetectable clinically). This "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, coverage progresses rapidly to the point where it can detected clinically, after 4 days, on average 30% of the total coronal tooth area will be covered with plaque.

Several reports have shown that the microbial composition of the dental plaque will change with a shift toward a more anaerobic and a more gram-negative flora, including an influx of fusobacteria‹ filaments, spiral forms, and spirochetes. This was beautifully illustrated in experimental gingivitis studies. In this ecologic shift within the biofilm, there is a transition from the early aerobic environment characterized by gram-positive facultative species to a highly oxygen-deprived

environment in which gram-negative anaerobic microorganisms predominate. Bacterial growth in older plaque is much slower than in newly formed dental plaque, presumably because nutrients become limiting for much of the plaque biomass.

Topography of Supragingival Plaque

Early plaque formation on teeth follows a typical topographic pattern with initial growth along the gingival margin and from the interdental space (areas protected against shear forces). Later, a further extension in the coronal direction can be observed. This pattern may fundamentally change when the tooth surface contains irregularities that offer a favorable growth path.Plaque formation can also start from grooves, cracks, or pits.

By multiplication, the bacteria subsequently spread out from these starting up areas as a relatively even monolayer. Surface irregularities are also responsible for the so-called "individualized" plaque growth pattern , which is reproduced in the absence of optimal oral hygiene. This phenomenon illustrates the importance of surface roughness in plaque growth, which should lead to proper clinical treatment options.

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

Smoothing an intraoral surface decreases the rate of plaque formation. Below a certain surface roughness ($Ra < 0.2\ \mu m$).

Individual Variables Influencing Plaque Formation

The rate of plaque formation differs significantly between subjects, differences that might overrule surface characteristics. A distinction is often made between "heavy" (fast) and "light" (slow plaque formers).

A multiple regression analysis showed that the clinical wet ability of the tooth surfaces, the saliva-induced aggregation of oral bacteria, and the relative salivary flow conditions around the sampled teeth explained 90% of the variation. Moreover,

the saliva from light plaque formers reduced the colloidal stability of bacterial suspensions of, for example, S. sanguinis.

Variation within the Dentition

Within a dental arch large differences in plaque growth rate can be detected. In general early plaque formation occurs faster: in the lower jaw (when compared to the upper jaw); in molar areas; on the buccal tooth surfaces when compared to palatal sites (especially in the upper jaw); and in the interdental regions when compared to the buccal or lingual surfaces.

Impact of Gingival Inflammation and Saliva.

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

The Impact of Patient's Age

Although older studies were contradictory, more recent papers clearly indicate that a subject's age does not influence rate of plaque formation. The developed plaque in the older patient group resulted However, in a more severe gingival inflammation, which seems to indicate an increased susceptibility to gingivitis with aging.

Spontaneous Tooth Cleaning Many clinicians still believe that plaque is removed spontaneously from the teeth such as during eating. However, based on the firm attachment between bacteria and surface, this seems unlikely.

Even in the occlusal surfaces of the molars, plaque remains, even after chewing fibrous food carrots, apples, or chips

Metabolism of Dental Plaque Bacteria The majority of nutrients for dental plaque bacteria originate from saliva or GCF, although the host diet provides an occasional but nevertheless important food supply.

Overall, the total plaque population is more efficient than any one constituent organism at releasing energy from the available substrates Metabolic interactions occur also between the host and plaque microorganism.

Increases in steroid hormones are associated with significant increases in the proportions of P. intermedia found in subgingival plaque. These nutritional interdependencies are probably critical to the growth and survival of microorganisms in dental plaque and may partly explain the evolution of highly specific structural interactions observed among bacteria in plaque.

Microbiologic specificity of periodontal diseases

Nonspecific Plaque Hypothesis → غير مطلوب

In the mid1900s, periodontal diseases were believed to result from an accumulation of plaque over time, eventually in conjunction with diminished host response and increased host susceptibility with age. The nonspecific plaque hypothesis maintains that periodontal disease results from the "elaboration of noxious products by the entire plaque flora. According to this thinking, when only small amounts of plaque are present, the noxious products are neutralized

by the host. Similarly, large amounts of plaque would produce large amounts of noxious products, which would essentially overwhelm the host's defenses.

Several observations contradicted these conclusions. First, some individuals with considerable amounts of plaque and calculus, as well as gingivitis, never developed destructive periodontitis. Furthermore, individuals who did present with periodontitis demonstrated considerable site specificity in the pattern of disease. Some sites were unaffected, whereas advanced disease was found in adjacent sites. In the presence of a uniform host response, these findings were inconsistent with the concept that all plaque was equally pathogenic. Recognition of the differences in

plaque at sites of different clinical status (i.e., disease versus health) led to a renewed search for specific pathogens in periodontal diseases and a conceptual transition from the nonspecific to the specific plaque hypothesis.

Inherent in the nonspecific plaque hypothesis is the concept that control of periodontal disease depends on control of the amount of plaque accumulation. The current standard treatment of periodontitis by debridement (nonsurgical or surgical) and oral hygiene measures still focuses on the removal of plaque and its products and is founded on the nonspecific plaque hypothesis. Thus، although the nonspecific plaque hypothesis has been discarded in favor of the specific plaque hypothesis or the ecologic plaque hypothesis, much clinical treatment is still based on the nonspecific plaque hypothesis.

Specific Plaque Hypothesis → غير مطلوب

The specific plaque hypothesis states that only certain plaque is pathogenic, and its pathogenicity depends on the presence of or increase in specific microorganisms.This concept predicts that plaque harboring specific bacterial pathogens results in a periodontal disease because these organisms produce substances that mediate the destruction of host tissues.

Acceptance of the specific plaque hypothesis was spurred by the recognition of *A. actinomycetemcomitans* as a pathogen in localized aggressive periodontitis.

The association of Socranksy'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 >13,000 plaque samples. Nevertheless, 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 defined "pathogens," such as red complex bacteria, and conversely that "pathogens" may be present in the absence of disease.

Ecologic Plaque Hypothesis

In the 1990s, 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 the ecologic plaque hypothesis, 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 microflora is considered to be relatively stable over time and in a state of dynamic equilibrium or "microbial homeostasis." The host controls subgingival plaque to some extent by a tempered immune response and low levels of GCF flow. Perturbations to the host response may be brought about by the excessive accumulation of (nonspecific) dental plaque, or by plaque-independent host factors (e.g., the onset of an immune disorder, changes in hormonal balance such as in pregnancy), or environmental factors (e.g. smoking, diet). Changes in the host status, such as inflammation, tissue degradation, and/or high GCF flow, may lead to a shift in the microbial population in plaque, culminating in periodontal disease.

Bacterial adherence

***Characteristics of bacterial surfaces**

Most bacteria in nature are surrounded by highly hydrated matrices called "glycocalyces"; these are often made up of "heterolpolysaccharides", which bacteria can produce from any carbohydrate source, many bacteria bear long appendages at their surfaces which may extend beyond the surface of the glycocalyx, these appendages are called pilli or fimbriae.

Characteristics of bacteria adherence

An important characteristic of living cells is that they carry negative electric charge and thus tend to repel each other electrostatically, the tooth surface is also negatively charged and repels the cells, the cells are also influenced by electrodynamic forces (van der waal's force) which are attractive forces, the attractive and repulsive forces will create a gap between the bacteria and the tooth surface, this gap is influenced by the presence of ions, hydrogen ions and cations (+ve) charge will narrow the gap, the importance of the glycocalyx has extension beyond the highly charged surface of the bacterial cell and can bridge the gap between bacteria and tooth surface .

The bacterial pili or fimbriae are long enough to protrude beyond the glycocalyx and assist in bridging the gap and establishing the contact between the bacteria and the tooth surface, the adhesion of bacteria to tooth surface is highly specific mechanism,

there are molecules called "adhesins" on the bacteria recognize specific receptor molecules on the tooth surface, these adhesins located on the pili.

The carbohydrate groups of glycoproteins of the pellicle may serve as receptors for such bacterial adhesins, so the pellicle will facilitate adherence and serve as solid ground for long lasting microbial life.

On the other hand some of the salivary mucins (agglutinins and secretory immunoglobulin A) may react with the bacterial surface structure and block adhesins so in that way it will prevent the bacteria from adhering to the oral surfaces.

Some anaerobic sub-gingival bacteria:

1. *Prophyromonas gingivalis.*
2. *Prevotella intermedia.*
3. *Aggregatibacter actinomycetem comitans (A.a),* formally called *actinobacillus actinomycetem comitans.*
4. *Capnocytophaga* species.
5. *Actinomyces naeslundii.*
6. *Fusobacterium nucleatum.*
7. *Streptococcus sanguis*
8. *Tannerella forsythia.*

Microbial flora are associated with:

1. Clinically healthy gingiva

If the teeth are kept clean with proper oral hygiene measures, the gingiva remains healthy and few bacteria are found along the gingival margin. If the person with such gingiva stops cleaning his teeth, bacteria will be accumulated on his teeth within few hours. The most predominant bacteria are streptococcus (G+ve cocci) and actinomyces (G+ve rods) also G-ve rods and facultative anaerobic rods are found in small proportions.

2. Gingivitis: a. In mild to moderate gingivitis: for at least 2-3 months, streptococci account for around 25% of the microbial flora of subgingival plaque

(*streptococcus mitis & streptococcus sanguis*) are predominant species. Another 25% of subgingival bacteria is composed of *Actinomyces* species. Another 25% are G-ve anaerobic rods as *fusobacterium, bacteroids and campylobacter.* Other 25% are miscellaneous bacteria. The spirochetes are very difficult to be cultured so it can be identified by dark field microscopy. Their percentage is very small forming about 2%.

a. ***Pregnancy gingivitis*:** there is increase in the proportion of *prevotella intermedia* (or *black pigmented bacteroids* species) and *capnocytophaga.* The increase in these organisms is related to increased levels of estrogen and progesterone hormones in gingival fluid. These hormones are used by these organisms as growth factors.
b. **Acute necrotizing ulcerative gingivitis:** the microflora is composed primarily of fusiform bacteria & spirochetes to form fusospirochetal complex. These organisms 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% like *bacteroids* and *fusobacterium nucleatum.* Spirochetes form about 50% of the flora.

4. Aggressive periodontitis

Dominated by anaerobic G-ve rods which form about 60%, and 7% spirochetes. From the G-ve rods attention has been paid for *aggregatibacter actinomycetem comitans* (A.a) which is almost always present in aggressive periodontitis and less prevalent in chronic periodontitis

Experimental Gingivitis

In an investigation called "experimental gingivitis in man" (Loe et al 1965), the cause and effect relationship between dental plaque and gingival inflammation was demonstrated. In that study, the oral hygiene of a group of healthy individuals (12 patients who were 9 dental students, 1 instructor and 2 laboratory technicians) was improved during several weeks of intensive instruction in the use of tooth brush and tooth picks. This resulted in excellent gingival condition. Then, all 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 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 experimental period, plaque samples were obtained at regular intervals and subjects to bacteriological examination of gram stained smear. The bacteria present in the samples were classified according to their gram reaction and morphology.

With healthy gingiva, very few bacteria were present on the cervical surfaces of teeth. The removable deposit was dominated by desquamated epithelial cells between which few bacteria could be seen about 90% of these bacteria were G+ve cocci and rods. The remainder 10% was G-ve bacteria.

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

1st 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.

2nd phase:

Days 3 and 4 are characterized by proliferation of fusobacteria and filamentous bacteria.

3rd phase: Days 5-9 are characterized by the 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 the type of species of bacteria and it's an important and dependable experiment which proved clinically that dental plaque is the main etiological factor in the development of periodontal disease.

Why the bacterial composition of sub-gingival plaque is different from supra-gingival plaque?

1- The access to the oral cavity is limited, which favors anaerobic bacteria (growing only In the absence of oxygen)
2- Nutrients are readily available from gingival exudates, the volume of which is increased as a result of inflammation in the gingiva.
3- Detachment of already established micro-organisms is limited due to the protecting gingival tissue making it possible for the organisms without special adhesion mechanisms to survive.
4- The chance of arrival of additional bacteria from saliva is limited.

The **nutrients of sub-gingival bacteria are provided by the following:**

1- Gingival exudates or fluid which contains proteins, carbohydrates, minerals and vitamins and they form a good nourishment to the bacteria.
2- Dead cells of the periodontal tissue when periodontal lesions are initiated.
3- The metabolic products produced by one group of bacteria may serve as energy source for other bacteria.

Differences between bacterial deposits

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

Periodontology

Lec:- 6

Alveolar process(AP)

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 the teeth and is gradually resorbed if the teeth are lost , thus it is tooth dependent structure

Functions of alveolar process:

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

2.provide the osseous attachment to the PDL fibers

3.distribute and resorb forces generated by mastication and other tooth contacts

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

Parts of the alveolar process:

1.Alveolar bone proper: it 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 blood vessels , lymphatics and nerves link the PDL with the cancellous bone thus it is called ((cribriform plate))

2.An external plate of cortical bone

3.Cancellous trabeculae or spongy bone: which is located in the space between the external cortical plate and alveolar bone proper, they meet and fuse to form the alveolar crest.cancellous bone, which act as supporting alveolar bone, with cortical bone surround the alveolar bone proper (ABP)

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

Lamina dura:-the layer of ABP appears as white line surrounding the root of the tooth on radiographs.

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

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 increases with age to an (average of 2.81mm).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, e.g. teeth that are labially positioned in the arch will have thin labial radicular bone and thicker lingual radicular bone.

Bone marrow:- The cavities of all bones of new-born are occupied by red marrow while in the adult jaw occupied by fatty or yellow type of marrow , however foci of red bone marrow are seen in the jaw which may be visible radiographically as zones of radiolucency.

Common locations are the maxillary and mandibular molar and premolar areas.

Periosteum and Endosteum:

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

Endosteum:- the marrow spaces inside the bone are lined by endosteum, this tissue contains cells (osteoblasts)

Anatomical defects of bone:-

1.Fenestration(window):-This bony defect include isolated areas in which the root is not covered with bone but covered only by periosteum and overlying gingiva and it does not extend to the marginal bone.

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

Such defects occur on approximately 20% of the teeth,they occur more often on the facial bone than on the lingual bone are more common on anterior than on posterior teeth.

The cause of these defects is not clear, but may be related to some factors such as, 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, consists of central canal called (Haversian canal)which in their center contains the blood vessel. These blood vessels surrounded by bone lamellae which arranged in concentric layers constitute the center of an osteon.The blood vessels in haversian canal are connected with each other by anastomoses running in the Volkmann's canals,so the nutrition of bone is secured by the incorporation of blood vessels in the bone tissue.

Bone cells:-

1.Osteoblast cells(bone forming cells):is responsible for the production of an organic matrix of bone which is consisting primarily of collagen fibers called (osteoid), this bone matrix undergoes mineralization by the deposition of minerals such as calcium and phosphate,which are subsequently transformed to hydroxyl apatite

2.Osteoclast cells:-These are large multinucleated cells found in concavities on the bone surface called (howship's lacunae) these cells responsible for bone resorption.

3.Osteocyte cells:- osteoblast cells that become trapped in the bone matrix and later on in the mineralized bone tissue , we call them osteocyte cells, they are located in the lacunae and are connected with the one another by extending processes into canaliculi through which they get nutrients and removes metabolic waste products.

Resorption of bone:-

The sequence of events in the resorptive process as follows:

1.attachment of osteoclasts to the mineralized surface of bone

2.creation of a sealed acidic environment,which demineralizes bone and exposes the organic matrix

3.degradation of the exposed organic matrix to its constituent amino acids via the action of released enzymes(e.g.,acid phosphates, cathepsin).

4.Sequestering of mineral ions and amino acids within the osteoclast

Composition of the bone:-

Bone consists of 2/3 inorganic matter and 1/3 organic matrix.

The inorganic matter is composed principally of the minerals calcium and phosphate,along with hydroxyl,carbonate,citrate,and lactate trace amounts of other ions such as sodium,magnesium and fluorine. The mineral salts are in the form of hydroxy apatite crystals.

The organic matrix consists mainly of collagen type I fibers (90%),with small amounts of non collagenous proteins such as osteocalcin and osteonectin.

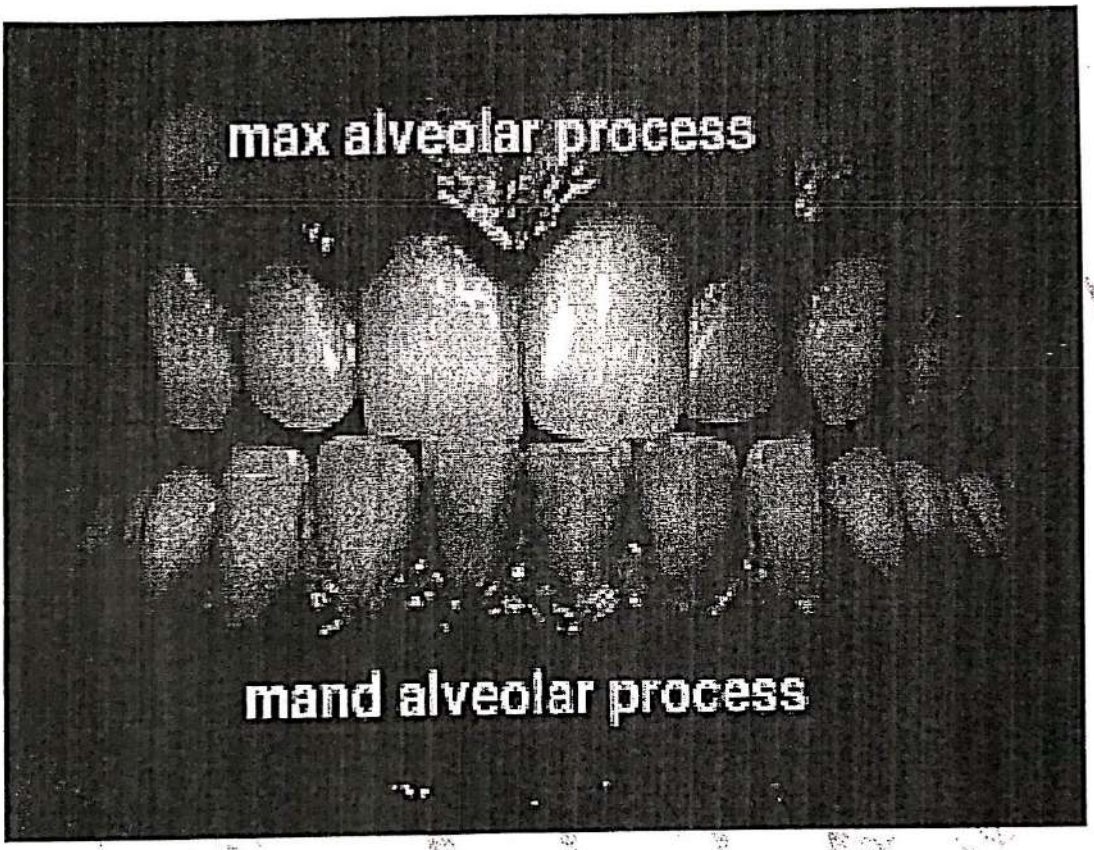
Bone contains 99% of the body's calcium ions and therefore is the major source for calcium release when the calcium blood levels decrease,this is monitored by the parathyroid gland.

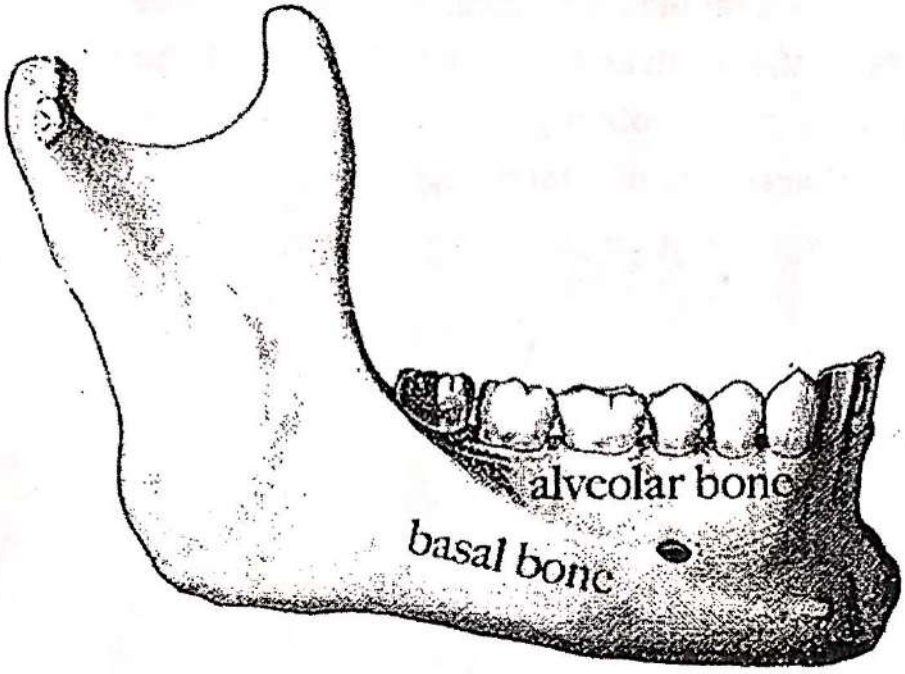
Remodeling of alveolar bone:-

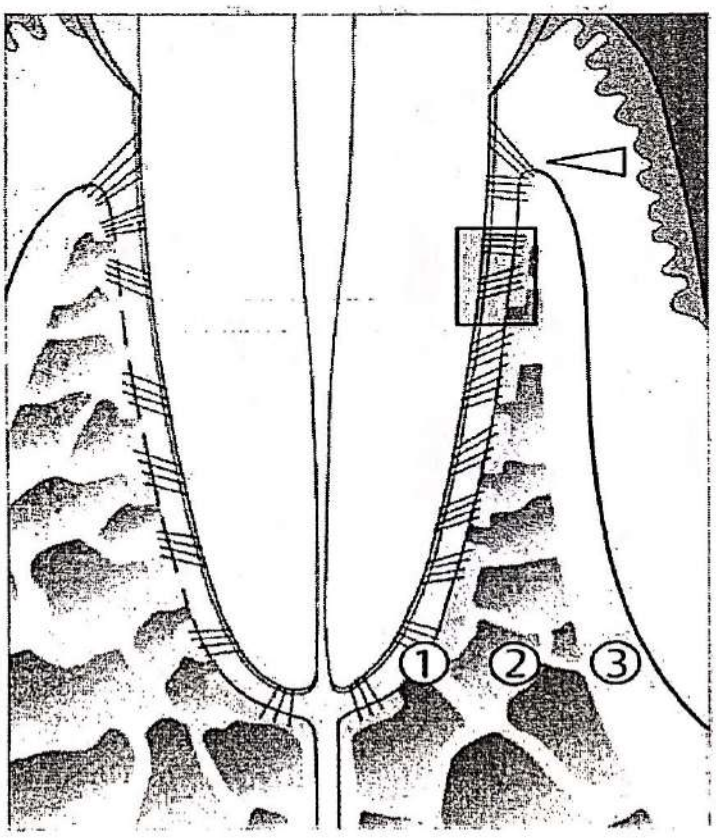
Alveolar bone undergoes constant physiologic remodeling (resorption and formation)in response to external forces specially occlusal forces.

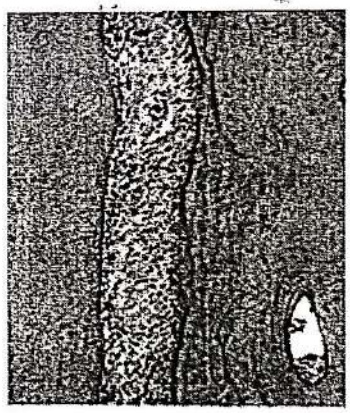
Teeth erupts and tend to move mesially throughout life to compensate for wearing in the proximal contact areas with age which become flat , this referred to as physiologic mesial migration , thus osteoclast cells and bone resorption occur in areas of pressure on the mesial surface and osteoblast cells with new bone formed in areas of tension on the distal surface.This process of resorption and formation of bone is called bone remodeling and it is important in the orthodontic treatment.

Remodeling of alveolar bone is regulated by local influences include functional requirements on the tooth and age related changes in bone cells while,systemic influences are probably hormonal(e.g.,parathyroid hormone,or vitamin D_3).









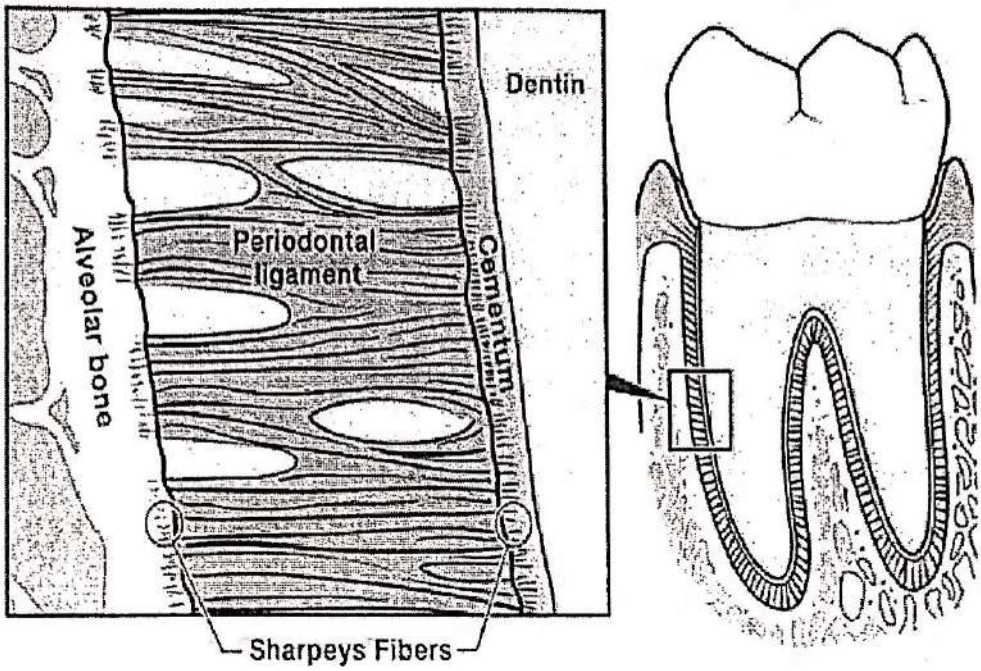
1 **Alveolar Bone**
Synonyms:

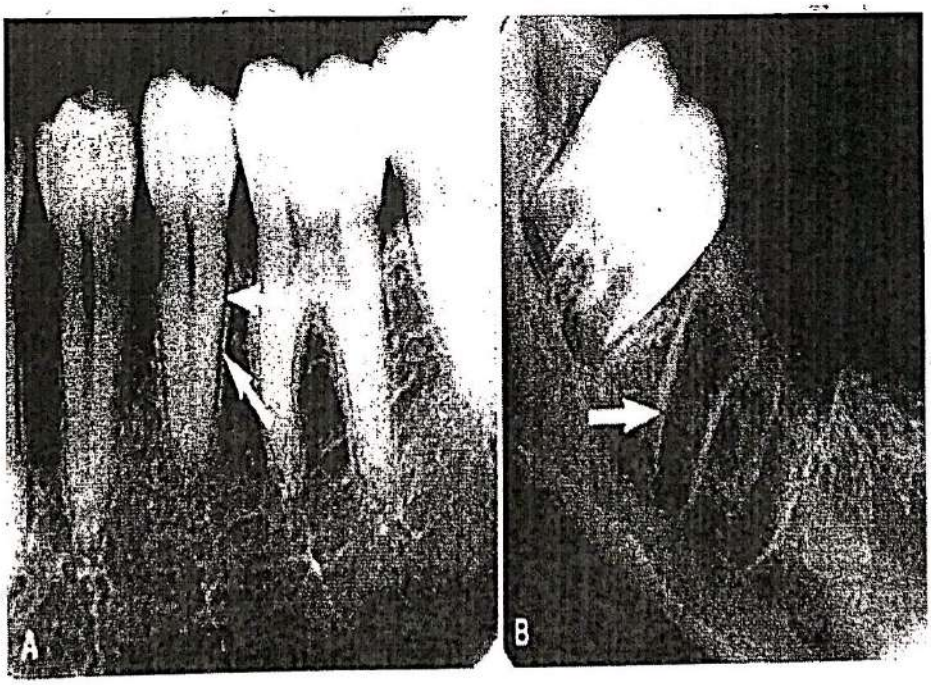
Anatomically
- Alveolar Wall
- Cribriform Plate

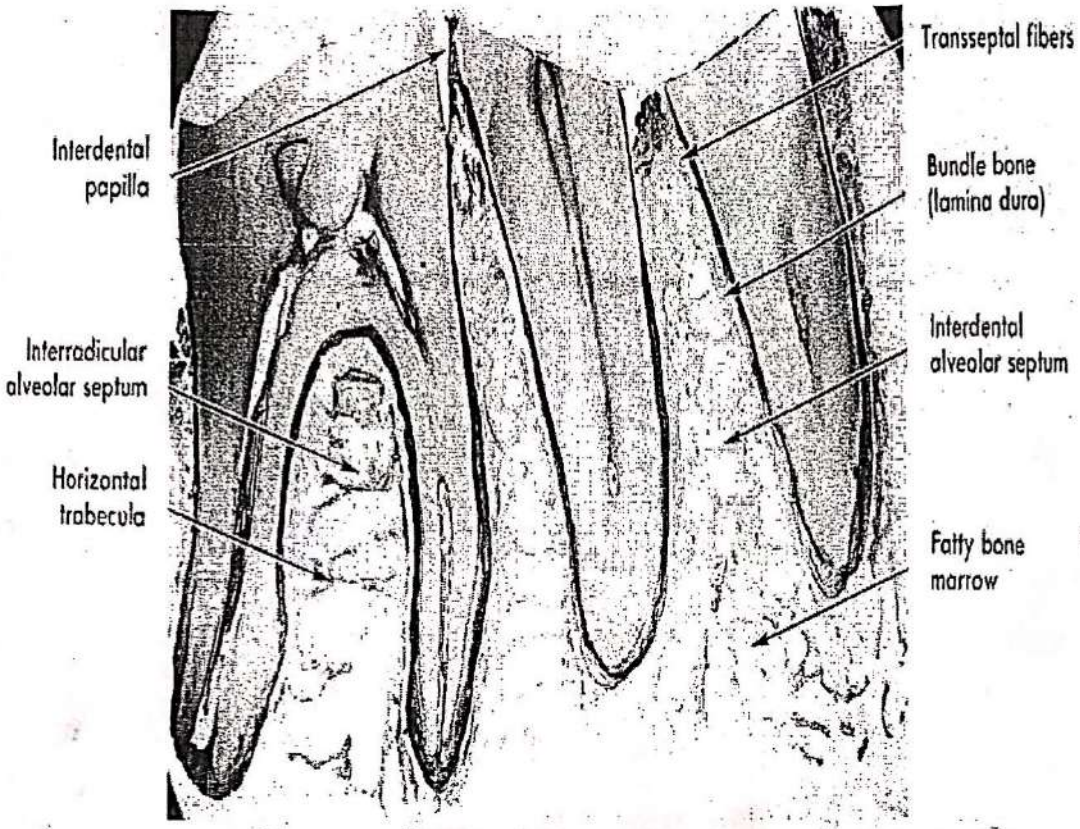
Radiographically
- Lamina dura

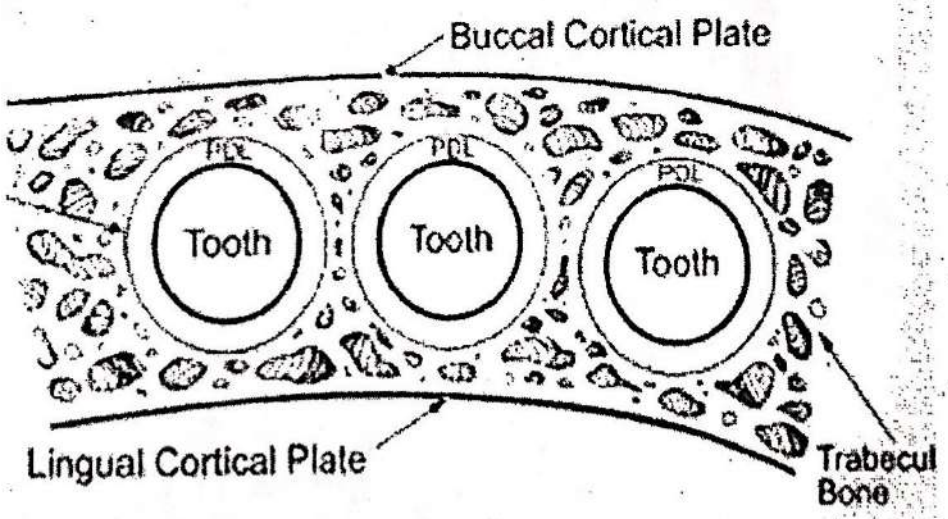
2 **Trabecular Bone**

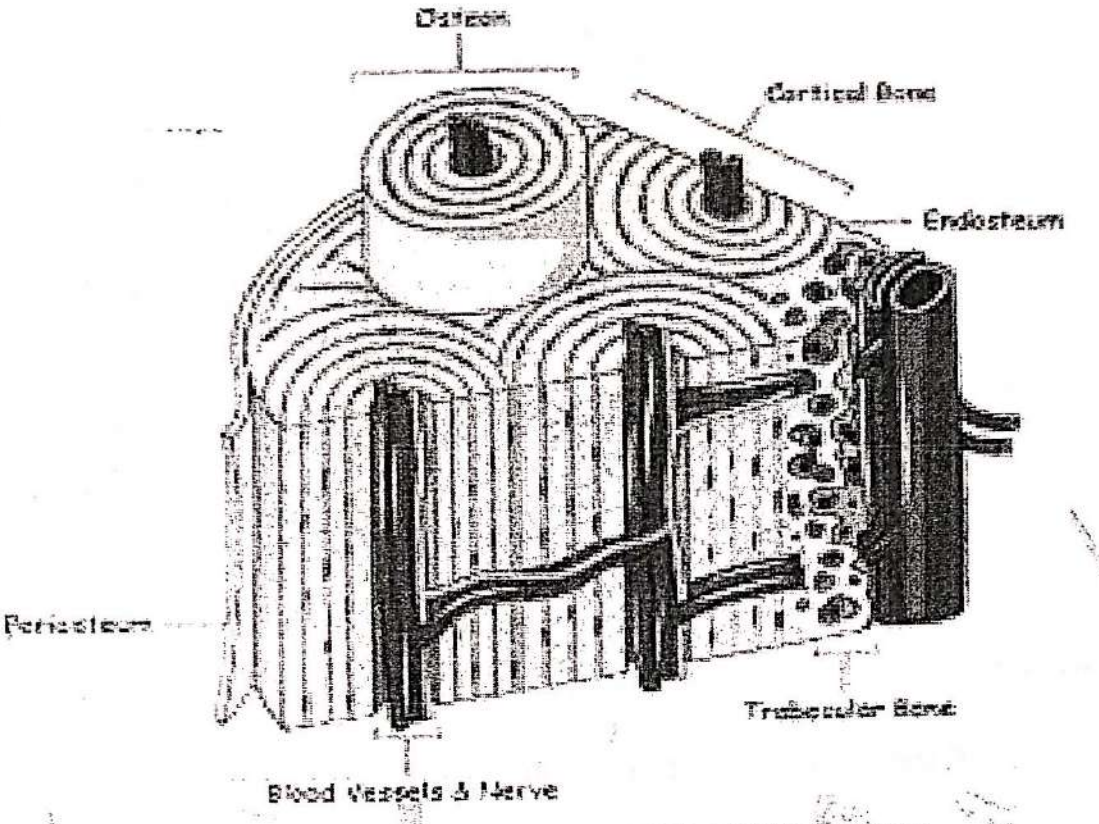
3 **Compact Bone**

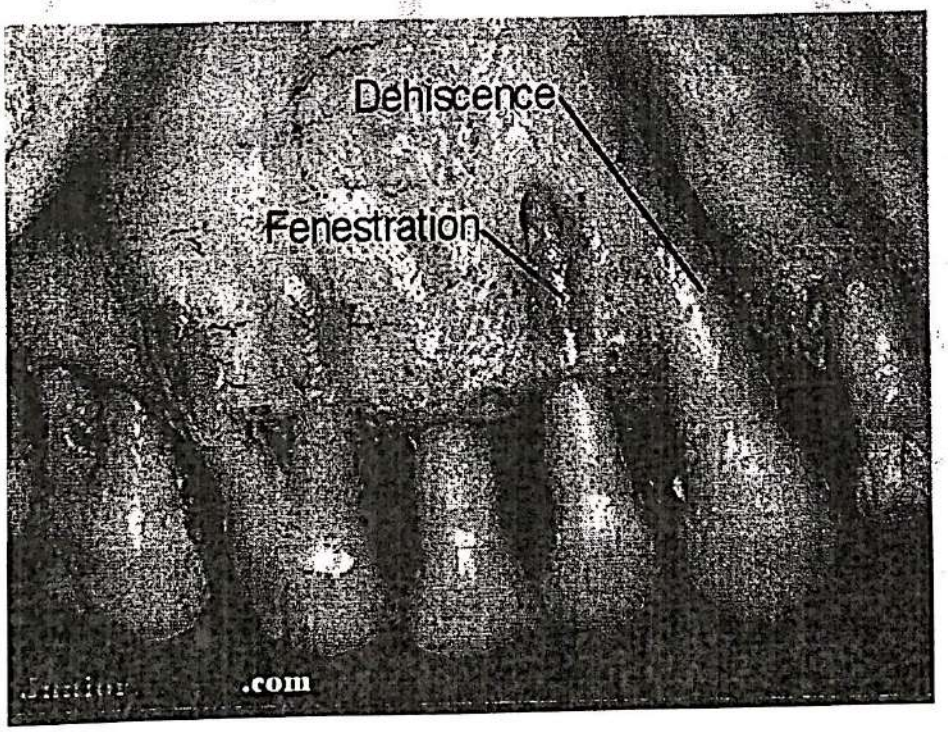


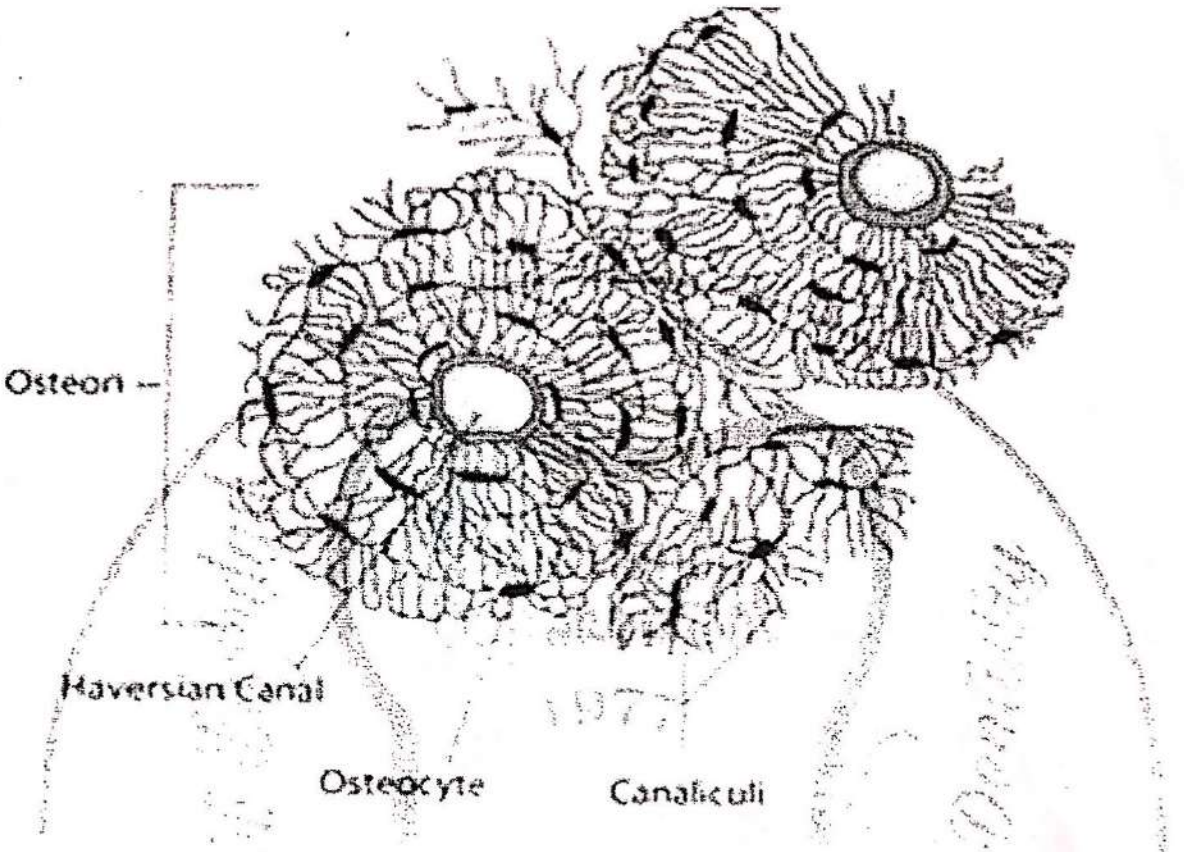


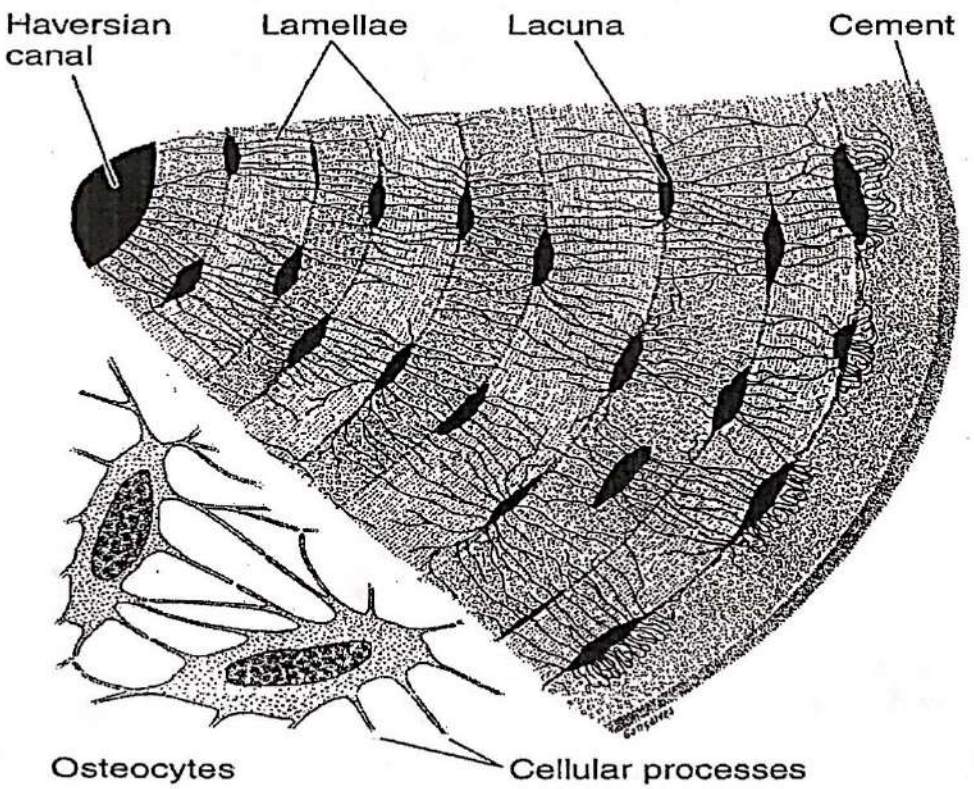


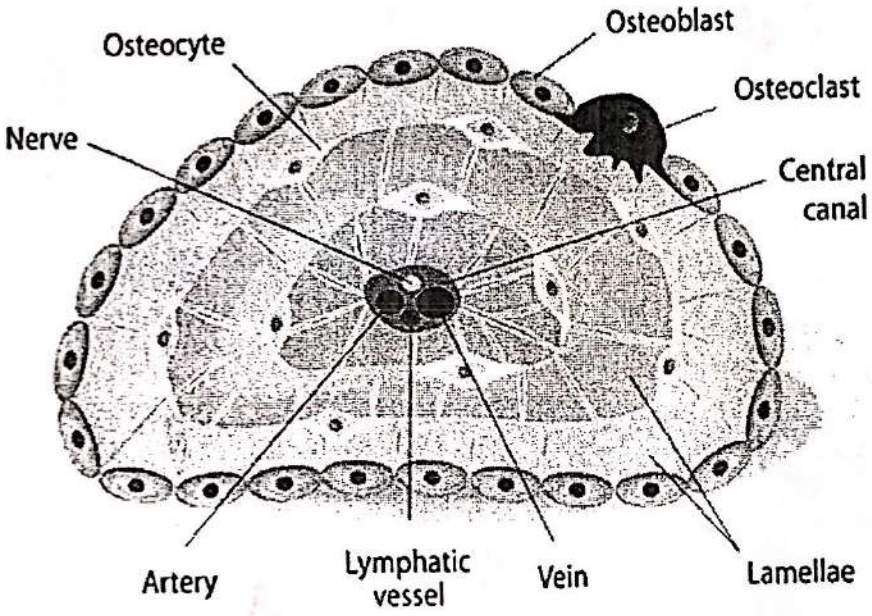




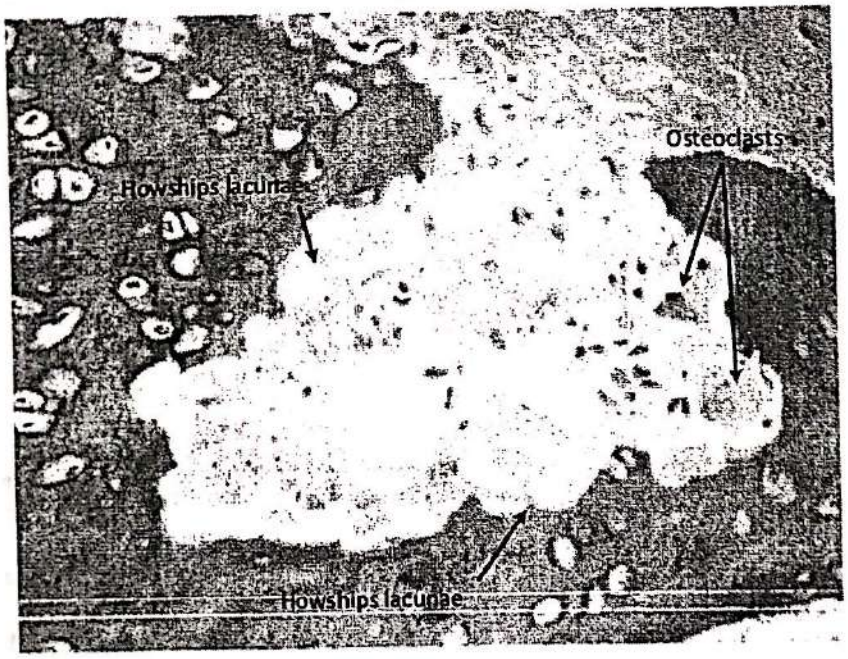


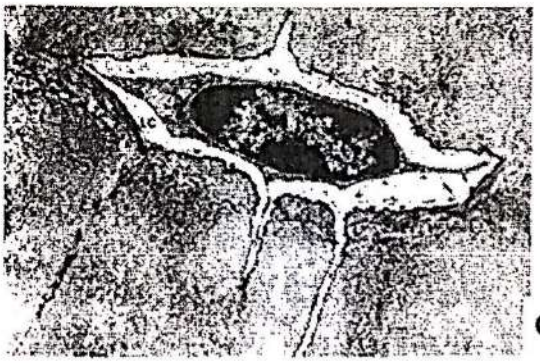


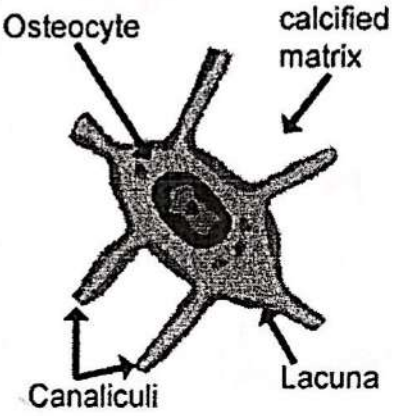


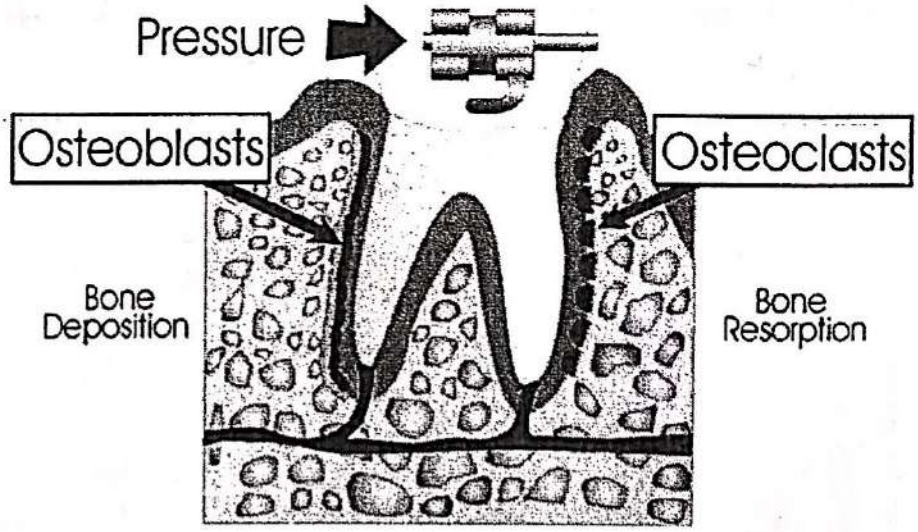


Bone Remodeling- Howships Lacunae









Tooth Movement

PERIODONTOLOGY

Lec 7

Dental Stain

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. 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 color is important for many aspects of restorative dentistry. Teeth are typically composed of a number of colors 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, 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. The science of color is important ir dentistry with regard to color perception and description, and can be improved with training. The viewing conditions are extremely important and variables such as the light source, time of day, surrounding conditions and the angle the tooth is viewed from affect the apparent tooth color. Light is composed of differing wavelengths and the same tooth viewed under different conditions will exhibit a different color, a phenomenon known as *metamerism.*

CLASSIFICATION OF TOOTH DISCOLOURATION

The coronal portion of the tooth consists of enamel, dentine and pulp. 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. discoloration of 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

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.

11. ***Pulpal hemorrhagic products:*** The discoloration of teeth following severe trauma was considered to be caused by pulpal hemorrhage.
12. ***Root resorption:*** Root resorption is often clinically asymptomatic; however, occasionally the initial presenting feature is a pink appearance at the cemento-enamel junction.
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.

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 mouth rinses 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.

INTERNALIZED DISCOLORATION

The stains taken up into the body of enamel or dentine are the same' as that causing extrinsic tooth discoloration, including in particular dietary chromogens and the byproducts 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 "intrinsic discoloration" either caused by increased enamel porosity, or the presence of enamel defects, extrinsic stains can penetrate into the enamel.
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

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.

7. ***Dentinal dysplasias:*** This reclassification allows separation of the inherited types of dentine defects from Dentinogenesis Imperfecta, with which they are often confused.
8. ***tetracycline's staining:*** Systemic administration of tetracycline during development is associated with deposition of tetracycline within bone and the dental hard tissues. Tetracycline is able to cross the placental barrier and should be avoided from 29 weeks *in uterus* until full term to prevent incorporation into the dental tissues. Since the permanent teeth continue to develop in the infant and young child until 12 years of age, tetracycline administration should be avoided in children below this age and in breast-feeding and expectant mothers. 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.
9. ***Fluorosis:*** This may arise endemically from naturally occurring water supplies or from fluoride delivered in mouth rinses, tablets or toothpastes as a supplement. The severity is related to age and dose, 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, while the color of enamel range from chalky white to a dark brown/black appearance.
10. ***Enamel hypoplasia:*** This condition may be localized or generalized. The

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, feces and teeth. A red-brown discoloration of the teeth is the result and the affected teeth show a red fluorescence under ultra-violet light.
3. ***Congenital hyperbilirubinaemia:*** The breakdown products of haemlysis will cause a yellow-green discoloration. 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 x-linked 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.
6. ***Dentinogenesis imperfecta:*** dentin defect may occur genetically or through

influence the colour of teeth.

a) *Tooth wear and gingival recession:* Tooth wear is usually considered to be a progressive loss of enamel and dentine due to erosion, abrasion and attrition. As enamel thins the teeth become darker as the color of dentine becomes more apparent. Once dentine is exposed the potential of chromogens to enter the body of the tooth is increased.

b) *Dental caries:* The various stages of the carious process can be recognized by changes in color as the disease progresses.

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 dentin. Some of polyantibiotic pastes used as root canal medicaments may caused darkening of the root dentin. Clinician are familiar with the dark grey to black color 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.

Perio

Classification of Periodontal Diseases

ATTEMPTS AT CLASSIFICATION Lec. 9+10

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 aetiology, histopathology and, where appropriate, genetics rather than age of onset and rates of disease progression. Over the last three decades there have been three major attempts to classify perio ntal disease. Although they all had obvious merits they all did not produce totally universally acceptable results mainly because of the imprecise nature of our knowledge on the specific bacterial etiology of periodontal diseases.

The first of these was by the 1st World Workshop in Clinical Periodontics in 1989 (American Academy of Periodontology 1989). This introduced the concept of periodontal diseases as distinct from periodontal disease and separated periodontitis into several categories: chronic periodontitis, rapidly progressive periodontitis and refractory periodontitis, on the basis of rate of progression and response to treatment. It also included separate entities of early-onset disease separating them into localized and generalized juvenile periodontitis and prepubertal periodontitis. Acute necrotizing gingivitis was also recognized as a separate entity.

The second attempt was made by the 1st European Workshop in Periodontics in 1993 (Attstrom & van der Velden 1994), which replaced chronic periodontitis with adult periodontitis, and introduced a broad category of early onset periodontitis, which contained localized and generalized juvenile periodontitis and prepubertal periodontitis.

The previous classification had many shortcomings including:

1) Considerable overlap in disease categories.

2) Absence of gingival disease component.

3) The age of onset of disease & rates of progression are not clear.

4) Unclear classification criteria.

The third attempt was started by the American Academy of Periodontology in 1997, who organized the International Workshop for a Classification of Periodontal Diseases and Conditions in 1999. At this workshop, a new classification was agreed upon (Armitage 1999). This attempted to develop a comprehensive classification of gingival diseases, periodontal diseases, necrotizing ulcerative gingivitis / periodontitis, periodontal abscesses, periodontitis associated with an endodontic lesion, developmental or acquired deformities and conditions mucogingival deformities and conditions and occlusal trauma. This classification includes both separate conditions and a number of other factors which may affect their severity or clinical presentation and is shown in Tables 1 and 2 The main changes in this classification arc:

1. The addition of a comprehensive section on gingival diseases.
2. The replacement of the term adult periodontitis with chronic periodontitis since epidemiological evidence suggests that chronic periodontitis may also be seen in some adolescents.
3. The elimination of separate categories of rapidly progressive periodontitis and refractory)" periodontitis because of the lack of evidence that they represent separate conditions but rather describe the rate of progression of chronic periodontitis or its response to treatment that result from differences in patient susceptibility.
4. Replacement of the term "early onset periodontitis" with 'aggressive periodontitis", largely because of the clinical difficulties in determining the age of onset in many of these cases. The authors of this new

classification also question the use of the term juvenile periodontitis for the same reasons. They have replaced them with the terms localized aggressive periodontitis and generalized aggressive periodontitis. They have also largely discarded the term prepubertal periodontitis and have included those cases which are not directly caused by systemic disease in the appropriate aggressive periodontitis category.

In summary aggressive periodontitis was previously termed as early onset periodontitis which classified as:

a) Prepubertal periodontitis (preteens) localized or generalized.

b) Juvenile periodontitis (teens) localized or generalized.

c) Rapidly progressive periodontitis.

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

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

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

I) Gingival diseases.

II) Chronic periodontitis:

A. Localized (<30% of involved sites)

B. Generalized (>30% of involved sites).

III) Aggressive periodontitis:

A. localized

B. Generalized.

IV) Periodontitis associated with systemic disease.

V) Necrotizing periodontal diseases:

A. Necrotizing ulcerative gingivitis

B. Necrotizing ulcerative periodontitis

VI) Abscesses of the periodontium.

VII) periodontitis associated with endodontic lesions.

VIII) Developmental or acquired deformities & conditions.

A Plaque induced gingival disease
1. Gingivitis associated with dental plaque only
a) without other locally contributing factors
b) with locally contributing factors
2. Gingival disease modified by systemic factors
a) associated with endocrine system
i) puberty-associated gingivitis
ii) menstrual cycle-associated gingivitis
iii) pregnancy-associated gingivitis or pyogenic granuloma
iv) diabetes mellitis-associated gingiivitis
b) associated with blood dyscrasias
i) leukaemia-associated gingivitis
ii) other
3. Gingival disease modified by drugs
a) drug-influenced gingival diseases
1)drug-influenced gingival enlargment
2) drug-influenced gingivitis
a) oral contraceptive-associated gingivitis
b) other
4.Gingival disease modified by malnutrition
a) ascorbic acid-deficiency gingivitis
b) other

B Non plaque-induced gingival lesion
1. Gingival disease of specific bacterial origin
a) Neisseria gonorrhea-associated lesions
b) Treponema pallidum-associated lesions
c) Streptococcal species-associated lesions
d) other
2. Gingival diseases of viral origin
a) Herpes virus infections
1) primary herpetic gingivostomatitis
2) recurrent oral herpes
b) oral Epstein-Barr virus lesions
c) Varicella-Zoster infections
3. Gingival disease of fungal origin
a) Candida species infections
i) generalized gingival candidiasis
b) linear gingival erythema
c) Histoplasmosis
d) other
4. Gingival diseases of genetic origin
a) hereditary gingival fibromatosis
b) other
5. Gingival manifestations of systemic conditions
a) mucocutaneous conditions
1) lichen planus
2) pemphigoid
3) pemphigus vulgaris
4) erythema multiformi
5) lupus erythematosus
6) drug-induced
7) other
b) allergic reactions
1) dental restorative materials
a) mercury
b) nickel
c) acrylic
d) other
2) reactions attributable to:
a) toothpastes/dentifrices
b) mouthrinses/mouthwashes
c) chewing gum additives
d) foods and food additives
6. Traumatic lesions (factitious,iatrogenic,accidental)
a) physical injury
b) chemical injury
c) thermal injury
7. Foreign body reactions
8. Not otherwise specified(NOS)

Table 1 Classification of gingival diseases:

d)others

Table 2 classification of periodontitis:

I) Chronic periodontitis
  a) Localized
  b) Generalized

Can be further divided according to severity on a tooth by tooth basis into:-

Early (mild) 1-2 mm CAL
Moderate 3-4 mm CAL
Advanced (severe) > or = 5 mm CAL

II) Aggressive periodontitis
  a) Localized
  b) Generalized

III) Periodontitis as a manifestation of systemic disease
  a) Associated with haematological disorders
    1) Acquired neutropenia
    2) Leukaemias
    3) Other
  b)Associated with genetic disorders
    1) Familial and cyclic neutropenia
    2) Down's syndrome
    3) Leukocyte adhesive deficiency syndrome
    4) Papillon-Lefevre syndrome
    5) Chediak-Higashi syndrome
    6) Histiocytosis syndrome
    7) Glycogen storage disease
    8) Infantile genetic agranulocytosis
    9) Cohen syndrome
    10) Ehlers-Danlos syndrome (types IV and VIII)
    11) Hypophosphatasia
    12) Other
  c) Not otherwise specified

IV) Necrotizing periodontal diseases
  a) Necrotizing ulcerative gingivitis
  b) Necrotizing ulcerative periodontitis

V) Abscesses of periodontium
  a) Gingival abscess
  b) Periodontal abscess

c) Pericoronal abscess
VI) Periodontitis-associated endodontic lesion
a) Combined periodontic-endodontic lesion
VII)Developmental or acquired deformities or conditions
A. Localized tooth-related factors that modify or predispose to gingivitis/periodontitis
1) Tooth anatomic factors
2) Dental restorations or appliances
3) Root fractures
4) Cervical root resorption or cemental tears

B. Mucogingival deformities or conditions around teeth
1) Gingival recession
a) Facial or lingual surface
b) Interproximal (papillary)
2) Lack of keratinized gingiva
3) Decreased vestibular depth
4) Aberrant fraenal or muscle position
5)Gingival excess
a. Pseudopocket
b. Inconsistent gingival margin
c. Excessive gingival display
d. Gingival enlargement
6)Abnormal color

C. Mucogingival deformities and conditions on edentulous ridges:

1. Vertical and/or horizontal ridge deficiency
2. Lack of gingiva/keratinized tissue
3. Gingival/soft tissue enlargement
4. Aberrant frenum/muscle position
5. Decreased vestibular depth
6. Abnormal color

D. Occlusal trauma

1. Primary occlusal trauma

2. Secondary occlusal trauma

Classification of Gingival Diseases

A. Dental plaque induced gingival diseases

There are four main types of plaque-associated gingival diseases :

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

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

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

1. Gingivitis associated with dental plaque only:-

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

- Without local contributing factors or
- With local contributing factors.

The local contributing factors can be defined as a local feature that may influence the presentation of the disease, such as

- overhanging restoration
- Dental calculus
- Prosthetic and orthodontic appliances.

Characteristics of plaque-induced gingivitis:

1. Plaque present at gingival margin

2. Disease begins at the gingival margin
3. Change in gingival color
4. Chang in gingival contour
5. Sulcular temperature change
6. Increased gingival exudate
7. Bleeding upon provocation
8. Absence of attachment loss
9. Absence of bone loss
10. Histological changes
11. Reversible with plaque removal.

2. Gingival diseases modified by systemic factors:-

A. Associated with the endocrine system:

1. Puberty-associated gingivitis
2. Menstrual cycle-associated gingivitis
3. Pregnancy-associated
    a) Gingivitis
    b) Pyogenic granuloma
4. Diabetes mellitus-associated gingivitis

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

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

3. a) pregnancy-associated gingivitis: It is pronounced inflammatory response of the gingiva to dental plaque and hormones usually occurring during the second and third trimesters.

b) pregnancy-associated pyogenic granuloma: 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.

4. Diabetes mellitus-associated gingivitis: It is inflammatory response

of the gingiva to plaque aggravated by poorly controlled plasma glucose levels.

B. Associated with blood dyscrasias:
   1) Leukemia- associated gingivitis
   2) Others

- Blood dyscrasia-associated gingivitis: gingivitis associated with abnormal function or number of blood cells.
- Leukemia-associated gingivitis: 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.

3. Gingival diseases modified by medications:
   a. Drug-influenced gingival diseases:
      1) Drug-influenced gingival enlargements
      2) Drug-influenced gingivitis:
         a) Oral contraceptive-associated gingivitis
         b) Others

- Drug-influenced gingival enlargement: Gingival enlargement resulting in whole or in part 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.

-Oral contraceptive-associated gingivitis:-

Pronounced inflammatory response of the gingiva to plaque and oral contraceptive. Oral contraceptive agents are one of the most widely utilized class of drugs in the world. 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.

4. gingival diseases modified by malnutrition: It is known that malnourished individuals have a compromised host defense system that may affect the susceptibility to infection.

a. Ascorbic acid-deficiency gingivitis
b. Others

- 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).

-

B. Non- plaque induced gingival lesions:

The origin of gingival inflammation in this group is different from that of the routine plaque-associated gingivitis. It is not caused by plaque and usually does not disappear after plaque removal.

1. gingival diseases of specific bacterial origin:
   a. Neisseria gonorrhea-associated lesions
   b. Treponema pallidum-associated lesions
   c. Streptococcal species-associated lesions
   d. Other

These conditions induced by exogenous bacterial infection other than common component of dental plaque.

2. Gingival diseases of viral origin:

These are acute manifestations of viral infections of oral mucosa, characterized by redness and multiple vesicles that easily rupture to form painful ulcers affecting the gingiva. These infections may be accompanied by fever, malaise, and regional lymphadenopathy.

a) Herpes virus infections:
   1) Primary herpetic gingivostomatitis
   2) Recurrent oral herpes
b) Oral Epstein- Barr virus lesions
c) Varicella- Zoster infections
d) other

- Primary herpetic gingivostomatitis:-

It is a viral infection of oral mucous membrane caused by herpes simplex virus type 1 (HSV-1). It is mostly occur in children and characterized by painful severe gingivitis with ulcerations and edema. A characteristic feature is the formation of vesicles which rupture, coalesce and leave fibrin-coated ulcers. The patient has fever and lymphadenopathy, the lesion will remain 7-14 days, recurrence is rare.

3. Gingival diseases of fungal origin:

These gingival manifestation of fungal infections are characterized by white, red, or ulcerative lesions associated with several predisposing conditions.

a) Candida species infections:

Generalized gingival candidiasis
b) Linear gingival erythema
c) Histoplasmosis
d) Others

In the generalized gingival candidiasis, the most common species that causes this condition is candida albicans. In otherwise healthy individuals, oral candidiasis rarely manifests in the gingiva, but in immunocompromized patients like HIV-seropositive, the infection show erythema of the attached gingiva and this condition is called linear gingival erythema which characterized by linear erythematous band limited to free gingiva and not respond to plaque removal.

4. Gingival diseases of genetic origin:
   a. Hereditary gingival fibromatosis
   b. Other

The hereditary gingival fibromatosis is genetically derived fibrotic gingival enlargement.

5. Gingival manifestations of systemic conditions:
   a. Mucocutaneous disorders:
      1) Lichen planus
      2) Pemphigoid
      3) Pemphigus vulgaris
      4) Erythema multiforme
      5) Lupus erythematosus
      6) Drug-induced
      7) Other

These oral manifestations of disorders of the skin and mucous membrane present as erosions, vesicles, bullae, ulcers, and desquamative lesions. The lesions may be erythematous, white, or striated in appearance.

   b. Allergic reactions:

These are gingival manifestations of immediate or delayed hypersensitivity responses.

1) Dental restorative materials:
   a) Mercury
   b) Nickel
   c) Acrylic
   d) Other

The allergy that is occur called contact allergy and there is clinical manifestations on the oral mucosa after a period of 12-48 hours following contact with the allergen. The lesions that affect the gingiva resemble oral lichen planus or leukoplakia. They are reddish or whitish and sometimes ulcerated but these lesions resolve after removal of the material.

2) Reactions attributable to:
   a) Tooth pastes/dentifrices
   b) Mouth rinses/mouthwashes
   c) Chewing gum additives
   d) Foods and additives

Tooth pastes and mouth washes:-

Contact hypersensitivity has been reported to occur after the use of tooth pastes and mouth washes but it is a rare condition and the allergic reaction may be due to the flavoring additives as carvone and cinnamon which also present in chewing gum. The gingiva will appear fiery red edematous and the lesions resolve after cessation of using the allergen-containing agent.

Foods:-

Some patients may be hypersensitive to certain type of food as kiwi, peach, apple, peanuts and pumpkin seed, red pepper resulting in gingivitis that resolve after removal of the allergen.

6. Traumatic lesions:

These are self-inflicted (factitious), accidental, or iatrogenic injuries. They may be present as localized gingival recession, abrasions, ulceration, and burns. The lesions may be edematous, erythematous, or white in appearance. Lesions may exhibit combinations of several of these clinical features.

a. Chemical injury
b. Physical injury

.. Thermal injury

-Traumatic lesions induced by chemicals:

These traumatic lesions can be caused by local application of certain chemicals such as aspirin, cocaine, pyrophosphates, detergents (e.g., sodium lauryl sulfate), smokeless tobacco, betel nut, and bleaching agents.

-Traumatic lesions caused by physical injury:-

These traumatic lesions may be accidental or result from inappropriate oral hygiene procedures, inadequate dental restorations, poorly designed dental appliances, and orthodontic bands and devices.

-Thermal trauma may occur from burns to the oral mucosa involving the gingiva. Common causes are hot coffee, pizza and melted cheese, dental treatment involving improper Handling of hot impression material, hot wax --- etc.

7. Foreign body reactions:

These lesions may present as acute or chronic gingival inflammation associated with entrance of foreign bodies or materials into gingival connective tissue. The foreign bodies could be of dental material origin or self-inflicted injury as chewing on sticks. The lesions can exhibit suppuration and may be red or red/white in appearance.

8. Not otherwise specifid (NOS): This mean that there may be some forms of gingivitis that do not fit under other items discussed previously.

Classification of periodontitis

I. Chronic periodontitis:

A. Localized

B. Generalized

The term chronic periodontitis replaced the previous name (adult periodontitis) since this form of periodontal disease can occur over a wide range of ages and can be found in both primary and secondary dentitions.

Chronic periodontitis: Is an infectious disease resulting in inflammation within the supporting tissue of the teeth, progressive attachment, and bone loss. It is characterized by pocket formation and/or gingival recession. It is recognized as the most frequently occurring form of periodontitis. Its onset may be at any age but is most commonly detected in adults. The prevalence and severity of the disease increases with age. It may affect a variable number of teeth and it has variable rates of progression. Chronic periodontitis is initiated and sustained by bacterial plaque, but host defence mechanisms play an integral role in its pathogenesis. The progressive nature of the disease can only be confirmed by repeated examination. It is reasonable to assume that the disease will progress further if treatment is not provided. Chronic periodontitis can be further 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;

- Can be modified by factors other than systemic diseases such as cigarette smoking and emotional stress.

There are recurrent and refractory (non-responsive) cases of periodontitis that is not considered as separate disease entity since any type of periodontitis can be recur and a small percentage of cases can be non-responsive to therapy.
Recurrent periodontitis represents a return of periodontitis.
refractory periodontitis represents some cases of periodontitis that do not have successful treatment outcome. Both these conditions are not separate disease entity.

II. Aggressive periodontitis:

A. Localized

B. Generalized

(A.P) The term aggressive periodontitis replaced the previous name early-onset periodontitis (prepubertal ,juvenile periodontitis & rapidly progressive periodontitis) because this term is too restrictive & features of this form of periodontitis can occur at any age and the classification for various forms of periodontitis should not based on the age of the patient at the time of presentation, but should based on clinical, radiographical, historical and laboratory findings.
The term localized aggressive periodontitis (LAP) replaces the older term localized juvenile periodontitis (L JP). Also generalized aggressive periodontitis (GAP) replaces generalized juvenile periodontitis (GJP).Rapidly progressive periodontitis replaced by either generalized aggressive periodontitis or chronic periodontitis.Prepubertal periodontitis replaced by localized or generalized aggressive periodontitis or periodontitis as a manifestation of systemic diseases.
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;
- Hyper-responsive macrophage phenotype, including elevated levels of PGE_2 and IL-1β;
- Progression of attachment loss and bone loss may be self-arresting.

The specific features of localized aggressive periodontitis (LAP):

- Circumpubertal onset;
- Robust serum antibody response to infecting agents;
- Localized first molar/incisor presentation with interproximal attachment loss on at least two permanent teeth, one of which is a first molar, and involving no more than two teeth other than first molars and incisors.

While the specific features of generalized aggressive periodontitis:

- Usually affecting persons under 30 years of age, but patients may be older;
- Poor serum antibody response to infecting agents;
- Pronounced episodic nature of the destruction of attachment and alveolar bone;
- Generalized interproximal attachment loss affecting at least three permanent teeth other than first molars and incisors.

III. Periodontitis as a manifestation of systemic diseases:

Systemic factors modify all forms of periodontitis through their effects on the normal immune and inflammatory defenses when there is reduction in number or function of polymorphonuclear leukocytes (PMNs) that may result in an increased rates and s..crity of periodontal destruction.

A. Associated with hematological disorders:

1. Acquired neutropenia
2. Leukemias
3. Other

B. Associated with genetic disorders:
1. Familial and cyclic neutropenia
2. Down syndrome
3. Leukocyte adhesion deficiency syndrome
4. Papillon-lefèvre syndromes
5. Chediak-higashi syndrome
6. Histiocytosis syndromes
7. Glycogen storage disease
8. Infantile genetic agranulocytosis
9. Cohen syndrome
10. Ehlers-Danlos syndrome (Types IV and VIII)
11. Hypophosphatasia
12. Other.

C. Not otherwise specified (NOS)

IV. Necrotizing periodontal diseases:

A. Necrotizing ulcerative gingivitis

B. Necrotizing ulcerative periodontitis

- 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.

V. Abscesses of the periodontium:

A. Gingival abscess: A localized purulent infection that involves the marginal gingiva or interdental papilla.

B. Periodontal abscess: A localized purulent infection within the tissue adjacent to the periodontal pocket that may lead to the destruction of periodontal ligament and alveolar bone.

C. Pericoronal abscess: A localized purulent infection within the tissue surrounding the crown of a partially erupted tooth.

Abscesses of the periodontium 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 radiolucency of the affected alveolar bone.

Gingival abscess is usually an acute inflammatory response to foreign substances forced into the gingiva. It starts as red painful swelling with smooth shiny surface. Within 24-48 hours, the lesion become fiuctuant and pointed with a surface orifice from which a purulent exudate may be expressed.

Periodontal abscess is usually associated with more advanced destruction of periodontal structures and it is located along the lateral surface of the root. The lesion may be acute or chronic. Acute abscess may progress to chronic if the purulent contents drain through a fistula into outer gingival surface.

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

A common cause for periodontal abscess formation is the incomplete removal of the calculus from periodontal pocket, shrinkage of the gingival wall will occur causing occluding of the pocket orifice and formation of the abscess. 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.

VI. periodontitis-associated endodontic lesion:

a) Combined periodontic-endodontic lesion:

lesions of the periodontal ligament and adjacent alveolar bone may originate from infections of the periodontium or tissues of the dental pulp. Pulpal infection may cause tissue destructive process that proceeds from the apical region of the tooth toward the gingival margin and this is called retrograde periodontitis to differentiate this from marginal periodontitis in which the infection spreads from the gingival margin toward the root apex. The periodontium communicates with pulp tissues through many lateral and accessory canals and foramina, so these channels will participates in extending pulpal infections to the periodontium and vice versa. Some periodontal-endodontic lesions are primarily of endodontic origin and in other cases, bacteria from chronic or aggressive periodontitis, gain access to the pulpal tissues through accessory canals and lead to pulpal infection. So in those cases where there is any coalescence of endodontic and periodontal lesions, the condition was termed Combined periodontal-endodontic lesions and this term is not based on the initial etiology of the lesion.

VII. Developmental or Acquired Deformities and Conditions

A. Localized tooth related factors that modify or predispose to plaque-induced gingival diseases & periodontitis:
   1. Tooth anatomic factors
   2. Dental restorations/appliances
   3. Root fractures
   4. Cervical root resorption and cemental tears.

Several conditions exist around teeth that may predispose the periodontium to disease. In certain cases these tooth-related 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. It should be emphasized that these tooth-related conditions are not separate disease entities, but may serve as localized predisposing and/or modifying factors in the onset or progression of plaque-induced gingival diseases and periodontitis.

- Tooth anatomic factors as cervical enamel projection and enamel pearls, palato-gingival grooves, open contacts ------ ect.
- Dental restorations and appliances as incorrect subgingival margin of restorations, crowns, orthodontic bands ------ ect.
- Cervical root resorption: the lesion located coronally on the root and this allows a communication between the area of resorption and the oral environment and bacteria can penetrate these areas and cause inflammation.

B. Mucogingival deformities or conditions around teeth:

1. Gingival/soft tissue recession
   a. Facial or lingual surfaces
   b. Interproximal (papillary)
2. Lack of keratinized gingiva
3. Decreased vestibular depth
4. Aberrant frenum/muscle position
5. Gingival excess
   e. Pseudopocket
   f. Inconsistent gingival margin
   g. Excessive gingival display
   h. Gingival enlargement
6. Abnormal color

C. Mucogingival deformities and conditions on edentulous ridges:

7. Vertical and/or horizontal ridge deficiency
8. Lack of gingiva/keratinized tissue
9. Gingival/soft tissue enlargement
10. Aberrant frenum/muscle position
11. Decreased vestibular depth
12. Abnormal color

Muc gingival: 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 recession: Is location of the gingival margin apical to the cemento-enamel junction.
The causes of gingival recession:

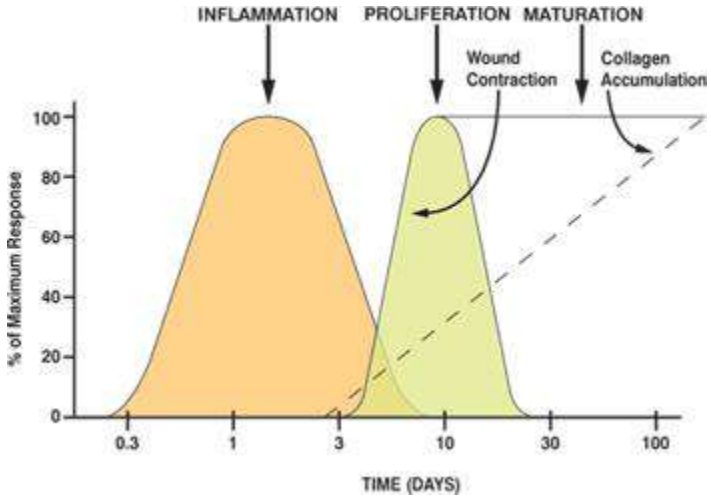
- Plaque accumulation will cause destruction of the junctional epithelia as a result of the inflammatory process.
- Traumatic gingival recession:
  - Fault tooth brushing
  - Tooth malposition
  - High frenal attachment
  - Excessive occlusal force
  - Overhanging fillings
  - Prosthetic appliances
  - Habits as nail biting.

D. Occlusal trauma
   3. Primary occlusal trauma
   4. Secondary occlusal trauma

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 excessive 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 excessive 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).

Healing of periodontal wounds

Phases of Wound Healing



Whether wounds are closed by primary intention, or left to heal by secondary intention, the wound healing process is a dynamic one which can be divided into three phases. Wound healing process is not linear and often wounds can progress both forwards and back through the phases depending upon intrinsic and extrinsic factors

The phases of wound healing are :

- Inflammatory phase
- Proliferation phase
- Maturation phase

The **inflammatory phase** is the body's natural response to injury. After initial wounding, the blood vessels in the wound bed contract and a clot is formed. Once haemostasis has been achieved, blood vessels then dilate to allow essential cells; antibodies, white blood cells, growth factors, enzymes and nutrients to reach the wounded area. This leads to a rise in exudate levels. It is at this stage that the characteristic signs of inflammation can be seen; erythema, heat, oedema, pain and functional disturbance. The predominant cells at work here are the phagocytic cells; 'neutrophils and macrophages'; mounting a host response and autolysing any devitalised 'necrotic / sloughy' tissue.

During **proliferation**, the wound is 'rebuilt' with new granulation tissue which is comprised of collagen and extracellular matrix and into which a new network of blood vessels develop, a process known as 'angiogenesis'. Healthy granulation tissue is dependent upon the fibroblast receiving sufficient levels of oxygen and nutrients supplied by the blood vessels. Healthy granulation tissue is granular and uneven in texture; it does not bleed easily and is pink / red in color. The color and condition of the granulation tissue is often an indicator of how the wound is healing. Dark granulation tissue can be indicative of poor perfusion, ischemia and / or infection. Epithelial cells finally resurface the wound, a process known as 'epithelialisation'.

Maturation is the final phase and occurs once the wound has closed. This phase involves remodeling of collagen from type III to type I. Cellular activity reduces and the number of blood vessels in the wounded area regress and decrease

Repair of wounds: include 2 responses

- ✓ **Epithelial response** which mean that mobilization and migration of epithelial cell at wound margin.
- ✓ **Connective tissue response**
  - Hemostasis
  - Inflammation
  - Proliferation
  - Synthesis (collagen synthesis)

Initial response to wounding:

A. Hemostasis
   - Hemorrhaging results in deposition of fibrin, aggregation of platelets and coagulation to form a clot within minutes of wounding. The clot aid in
     - Serves as a hemostatic barrier
     - Unites the wound margin
     - Provide a scaffold for subsequent migration of reparative cells

B. Inflammatory cell activation, migration and function. These inflammatory cells derived from 3 sources
   1. Cells normally present in the tissue.

2. Cells extravasated when blood vessels are damaged.
3. Cells carried in intact blood vessels.

The most important inflammatory cells:

- Neutrophil within few hours of injury to reach max. Concentration at 24 hours. Main functions: phagocytosis and mediate inflammatory changes.
- Macrophage which present after 24 hours and predominate at 5 days, these cells aid in phagocytosis and release of growth factors as IL-1

C. Proliferation of fibroblast (2 days and on) from undifferentiated perivascular cells.
D. Collagen synthesis from the new fibroblast.

Wound union:

A. ***Primary union:*** if wounds are produced surgically in sterile environments and their edges are brought closely, it is said that "primary union" or "healing by primary intent" has been achieved. Under these condition there is:
    a. Minimal trauma
    b. Little chance of secondary infection
    c. Heal quickly without complication.

    Steps of healing:

    1. Clot formation
    2. Inflammation
    3. Granulation
    4. Epithelization
    5. Cicatrization (scar formation).

B. ***Secondary union:*** it takes place when the edge of wound can't be brought together e.g: Gun shot exit wounds, free gingival graft, the wound produced at the donor site can't be sutured together and is left open, it termed "secondary union" or "healing by secondary intent" or "granulating-in".

    Steps of healing:

1. Clot formation
2. Inflammation
3. Granulation
4. Epithelization
5. Cicatrization (scar formation).

Wound healing after periodontal treatment:

Rationale for periodontal treatment:

1. Eliminate pain and gingival inflammation and bleeding.
2. Reduce periodontal pockets.
3. Eliminate infection and stop pus formation.
4. Arrest soft tissue and bone destruction and restore the tissue destroyed by the disease.
5. Reduce abnormal tooth mobility and establish optimal Occlusal function.
6. Re-establish the physiologic gingival contour necessary for the preservation of periodontal health.
7. Prevent recurrence of the disease and so reduce tooth loss.

Healing after scaling and blind root planing:

If scaling is done with overlapping strokes, it is technically possible to detach all the subgingival deposits. Immediately after conclusion of a successful subgingival scaling all plaque organisms are detached from the tooth, many of the bacteria are swimming in the exudates. However, the bleeding which follows will carry most of detached particles, including bacteria out of the pocket during and immediately after the debridement. The bleeding will stop in a few minutes, but a fairly profuse exudation from damaged blood vessels will continue for many hours. The exudate which is a mixture of water, serum protein and white blood cells will accumulate between the tooth and the soft tissue. This is called gingival fluid. Gingival crevicular fluid secretion per day of normal healthy gingiva is 0.5-2.4 ml/day. The gingival fluid contributes to the mechanical cleansing of pocket because it seeps out in a continuous flow.

Most of the detached plaque organisms are brought out of the pocket with the gingival fluid in a few minutes. Those organisms which may have been captured in the soft tissue are being eradicated by the PMNL. Finally, large numbers of bacteria enter the lymph and blood vessels to be brought to the regional lymph nodes or to spleen where they are destroyed.

Only a few hours after debridement, all the bacteria are removed (mostly with the gingival fluid). The secretion of gingival fluid will then subside and the epithelial reminant which may have been left in the pocket begin to proliferate.

The granulation tissue in the lateral wall of the pocket, in an environment free of plaque and calculus will be changed into connective tissue; there by minimizing shrinkage, this is regarded as an important advantage of blind root planing over radical surgery, i.e.: less trauma and hemorrhage will result in less gingival shrinkage during healing. This is very important for esthetic which is a major consideration of therapy, particularly in the anterior region.

Further more exposed cementum to a pathological pocket is cytotoxic to both epithelium and fibroblast by bacteria with their toxins penetrating this cementum.

Removal of exposed cementum through root planing eliminates undesirable surface contamination and provides a healthy surface to which fibroblasts adhere. Thus reduction of pocket probing depth following blind root planing is partly due to shrinkage of gingival wall of pockets (repair) forming long junctional epithelium in most of cases and partly from regeneration of lost attachment.

Healing following surgical procedure:

Gingivectomy: healing will be by 2nd intention (secondary union) which occurs by the formation of granulation tissue which grows from the base of the wound to fill the defect.

The vascular and fibroblastic proliferation which together make up the granulation tissue are much more abundant and healing taken much larger than when it occur by first intention.

The main problem after Gingivectomy is for the epithelial cells to cover the open wound. There is little if any regeneration necessary in the connective tissue because an incision at the bottom of the pocket usually will remove all the granulation tissue, leaving a clean connective tissue surface. Thus, the epithelial cells are the main actors in the healing of Gingivectomy.

Steps of healing:

1. The incision exposes many blood vessels of all sizes, when the pack is applied, blood clot is formed and the blood vessels are sealed with fibrin to stop further bleeding. The underlying tissue becomes acutely inflamed with some necrosis.
2. The blood clot below the pack contains large numbers of microorganisms. However, these are in most cases quickly phagocytozed by PMNs which are migrating into the area in large numbers. Therefore, the blood clot is likely to be free of bacteria within hours.
3. The next step in healing is the proliferation of macrophages which engulf RBC and disintegrating PMN. Within 1-2 days, epithelial cells start to migrate from oral mucosa. These cells migrate on a network of fibrin. Surface epitheliazation is completed after 5-14 days.
4. Under particularly favorable condition, epithelial cells can migrate as far as 2mm in 24 hours. After Gingivectomy, the speed must be considerably less and it may take 1-2 weeks before the oral epithelial cells reach the tooth surface.
5. If the regeneration occurred in a plaque free tooth surface, free gingival unit will form. This regeneration occurs in coronal direction and appear clinically as gain in marginal height “zero pocket”.
6. Complete epithelial repair taken about 4-5 weeks, while complete repair of connective tissue takes about 7 weeks.
7. The gingival fluid increases after Gingivectomy and diminished as healing progresses, because decrease in vasodilatation and vascularity. Also during the first 4 weeks after gingivectomy, keratinization is less than it was prior to surgery. Also pigmentation is diminished in the healed gingiva in patients with physiologic gingival melanosis.

Healing following a flap operation:

Healing will be by first intention and has many similarities with healing of an incision in the skin. It is more rapid than secondary intention and characterized by the formation of only minimal amounts of granulation tissue.

Steps of healing:

1. Immediately after suturing (0-24 hrs), a connection between flap and tooth or bone surface is established by the blood clot which consists of fibrin, PMNs, erythrocytes, debris from injured cells and capillaries at the edge of the wound, there are also bacteria and an exudates as result of tissue injury.
2. 1-3 days after surgery: the space between the flap and tooth or bone is thinner, epithelial cells migrate over the border of the flap to contact the tooth. When the flap is closely adapted to the alveolar process, there is minimal inflammatory response.
3. One week after surgery: epithelial cells are attached to the root by hemi desmosomes and a basal lamina. The blood clot is replaced by granulation tissue derived from gingival connective tissue, the bone marrow and the periodontal ligament.
4. Two weeks after surgery: appearance of collagen fibers parallel to the tooth surface. They are immature therefore union is still weak, although clinically may be normal.

One month after surgery: a fully epithelized gingival crevice with a well-defined epithelial attachment is present. There is a beginning of functional arrangement of supracrestal fibers.

Healing following grafting procedures

Thickness of any given graft range between 1-1.5 mm in optimum condition, Soft tissue grafts placed on bone shrink by about 25% while that placed on periosteum shrink by about50%.

Healing of soft tissue grafts placed entirely on a connective tissue recipient bed divided in to 3 phases (Oliver et al.1968)

1-.Initial phase: (from 0-3days)

Thin layer of exudates present between the graft and recipient bed during this period the graft survive with an avascular (plasmatic circulation) from recipient bed.

- The epi. Of free graft degenerate early and desquamated.
- In case of placing graft over a recession part of the bed will be a vascular root surface so the area of graft must receive nutrient from the connective tissue bed that surrounds the recession.

2-. Revascularization phase :(from 2-11days)

- anastomosis are established between the blood vessels of recipient bed and graft tissue (re-established of circulation)and capillary proliferation .also a fibrous union between graft and underlying C.T and re-epithelialization of the graft by proliferation of adjacent epi.
- **3-Tissue maturation phase** :(from 11-42days)

During this period the number of blood vessels in the transplant reduced and after 14 days vascular system appear normal with formation of keratin layer after epithelial maturation.

Periodontal wound healing after regenerative therapy

Regeneration of the periodontium must include the formation of new cementum with inserting collagen fibers on the previously periodontitis-involved root surfaces and the regrowth of the alveolar bone. However, whether regrowth of alveolar bone should always be considered a requirement for success following regenerative periodontal surgery is a matter of discussion. The basis for this discussion is that a fibrous attachment may exist without opposing bone in a normal dentition, not affected by periodontitis, in the presence of bone dehiscenses and fenestrations
After flap surgery the curetted root surface may be repopulated by four different types of cell:
1. Epithelial cells
2. Cells derived from the gingival connective tissue
3. Cells derived from the bone
4. Cells derived from the periodontal ligament

Regenerative capacity of tissue cells

The ability of newly formed tissue originating from different type of periodontal cells to produce a new connective tissue attachment was examined in many studies and, it was concluded that tissue derived from bone lacks cells with the potential to produce a new connective tissue attachment.

Gingival connective tissue also lacks cells with the potential to produce a new connective tissue attachment.Only cells in the periodontal ligament seem capable of regenerating lost periodontal attachment.

Factors affecting healing after perio treatment;

I. Local factors

a. Local factors improve healing

- Good debridement
- Immobilization of the healing area.
- Pressure on the wound
- An increase in O_2 consumption that increase cellular activity

b. Local factors delay healing

- Excessive tissue manipulation.
- Trauma and presence of foreign bodies.
- Repetitive treatment procedures that disrupt the orderly cellular activity in the healing process.
- Inadequate blood supply lead to impaired cellular activity ⟶ necrosis ⟶ delay healing.

II. ***<u>Systemic factors:</u>***

- ✓ Aging (diminishes capacity because of atherosclerotic vascular changes which reduce blood circulation).
- ✓ Patient with generalized infections.
- ✓ Diabetes.
- ✓ Patients with debilitating diseases.
- ✓ Malnutrition: Vit. C deficiency, Protein deficiency.
- ✓ Systemically administrated hormones as cortisone

Healing after implant placement

- A direct connection between bone and implant without interposed soft tissue layers termed as "osseointegration"
- Dr. branemark(1952) defined osseointegration as "*Direct structural and functional connection between ordered, living bone and surface of a load carrying implant*".

Stages of osseointegration

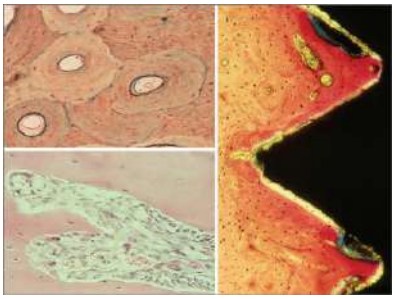
After 24 hours:

- Bone trabeculae in the apical portion of the implant dislocated into marrow space
- Blood vessels were severed and bleeding occurred
- Blood clot formation can be observed between the implant body and the host bone

After one week:

- There will be release of growth factors which activate fibroblast and formation of provisional connective tissue in the apical trabecular region and the inner part of the threaded region of implant
- After two weeks:
- Newly formed bone has been laid down on the implant

After 4 weeks :

- Newly formed woven bone lined most part of the implant surface which represents 1st stage of true osseointegration
- 

Final stage of osseointegration

- A stage of remodeling will occur during which woven bone is substituted with lamellar bone 

Factors influencing bone healing

1 --biological factors

- health condition of the recipient bone

-Surgical and operative procedures

-Infection control

2--Chemical factors: properties of the implant material should be:

-biotolerant

-Bioinert

-Bioactive

3--Physical factors include

-Size of implant-bone contact surface (interface)

-*Implant shape → retentive form to achieve primary retention and increase of implant surface.*

-loading

New attachment and guided tissue regeneration GTR

Aspects of periodontal healing:

1. Regeneration.
2. Repair
3. New attachment.

I. **Regeneration:** is the growth and differentiation of new cells and intercellular substances to form new tissues. It occurs by growth from the same type of tissue that has been destroyed or from its precursor.

In the periodontium:

1. Gingival epithelium is replaced by epithelium.
2. Connective tissue, PDL., bone & cementum all are derived from connective tissue, undifferentiated connective tissue cells develop into fibroblasts, osteoblast and cementoblasts.

Regeneration under normal conditions:

Regeneration of the periodontium is a continuous physiologic process, new cells and tissues are continuously being formed to replace mature and dead cells, this is termed "wear and tear repair".

Regeneration during destructive periodontal disease:

Most gingival and periodontal diseases are chronic inflammatory conditions, i.e, they are healing processes regeneration is part of healing. However, bacteria and bacterial products are injurious to the regenerating cells and tissues. They prevent the healing from proceeding to completion, but, when bacterial plaque is removed and prevented from new formation by periodontal treatment, the inherent regenerative capacity of tissues is established.

II. **Repair**: restoration of the continuity of the diseased marginal gingiva and re-establishment of a normal gingival sulcus at the same level as the base of a preexisting pocket, it is called (healing by scar), bone loss is arrested with mobilization of epithelial

and connective tissue cells into the damaged area with increase mitotic division to provide a sufficient number of cells.

III. **New attachment**: is the embedding of new PDL. Fibers into new cementum and attachment of epithelium to a tooth surface previously denuded by disease. The term reattachment was used in the past to represent the restoration of the marginal periodontium, but because it is not the existing fibers that reattach but new fibers that are formed and attach to new cementum, the term was changed to new attachment.

Reattachment: refer to repair in areas of the root not previously exposed to the pockets, but after surgical detachment of the tissues or after traumatic tears in cementum, tooth fractures, or treatment of periapical lesion.

Epithelial adaptation: close apposition of the gingival epithelium to the tooth surface without complete obliteration of the pocket, it may be as resistant to disease as true connective tissue attachments. 4-5mm. depth with absence of bleeding or secretion on probing post therapy may be acceptable. This may indicate that the "deep sulcus" persists in an inactive state.

Regeneration of PDL is the basis for new attachment because:

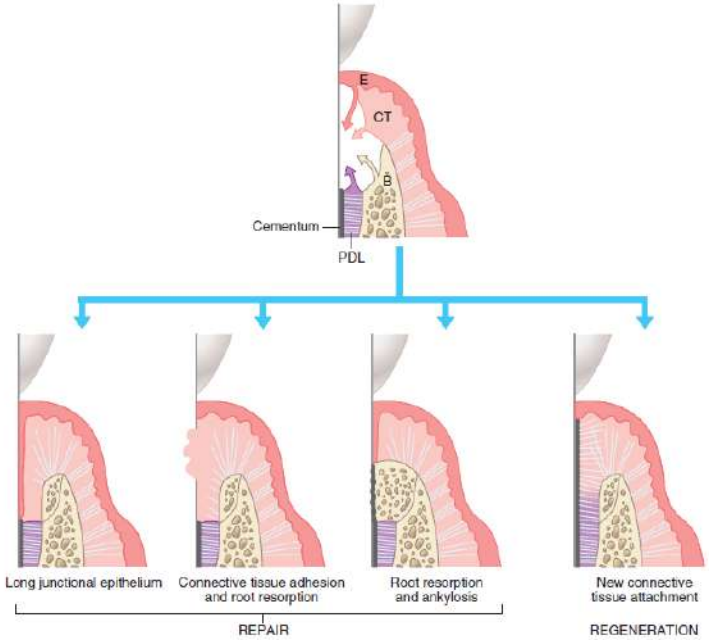
1) PDL provides continuity between the alveolar bone and cementum.
2) PDL contains cells that can synthesize and remodel the 3 connective tissues of the periodontium.

The possible outcomes of therapy:

During healing stages of a periodontal pocket, the area is invaded by cells from 4 different sources which modify the final outcome of pocket healing:

1. Oral epithelium: if epithelium proliferates along the tooth surface before the other tissues reach the area, the result will be a long junctional epithelium.
2. Gingival connective tissue: if the cells from the gingival connective tissue are the first to populate the area, the result will be fibers parallel to the tooth surface and remodeling of the alveolar bone, with no attachment to the cementum (recurrence of pocket).
3. Bone. If bone cells arrive first, root resorption and ankylosis may occur.

4. Periodontal ligament: when cells from PDL proliferate coronally, there is new formation of cementum and PDL (new attachment). Which is the ideal outcome of periodontal therapy as it will obliterate the pocket and reconstitute the marginal periodontium.



Evaluation of new attachment and bone regeneration:

1) **<u>Clinical methods:</u>** comparison of pre and post treatment records of:
    a. Pocket probing.
    b. Attachment level
    c. Gingival indices
    d. Bone level.

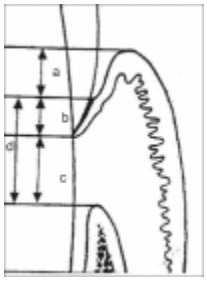
A-pocket probing

The periodontal pocket is a soft tissue change; therefore it is not detected by radiographic examination, but by careful exploration with a periodontal probe. The biologic depth is the distance between the gingival margin and the base of the pocket (the coronal end of the junctional epithelium). It differs from the probing depth which is the depth of penetration of a probe in a pocket(the distance from gingival margin to the apical extent of periodontal probe)

that depends on factors such as the size of the probe, the force with which it is introduced, the direction of penetration, the resistance of the tissues and the convexity of the crown. The probe tip penetrates to the most coronal intact fibers of the connective tissue attachment apical to the junctional epithelium about 0.3mm, reduction of this penetration after treatment may be a result of reduced inflammatory response rather than gain in attachment.

BIOLOGIC WIDTH ANATOMY

In the human body, ectodermal tissue serves to protect against invasion from bacteria and other foreign materials. However, both teeth and dental implants must penetrate this defensive barrier. The natural seal that develops around both, protecting the alveolar bone from infection and disease, is known as the biologic width. The biological width is defined as the dimension of the soft tissue, which is attached to the portion of the tooth coronal to the crest of the alveolar bone. This term was based on the work of Gargiulo *et al*., who described the dimensions and relationship of the dentogingival junction in humans. They established that there is a definite proportional relationship between the alveolar crest, the connective tissue attachment, the epithelial attachment, and the sulcus depth. They reported the following mean dimensions: A sulcus depth of 0.69 mm, an epithelial attachment of 0.97 mm, and a connective tissue attachment of 1.07 mm. Based on this work, the biologic width is commonly stated to be 2.04 mm, which represents the sum of the epithelial and connective tissue measurements



(a) Histological sulcus (0.69 mm), (b) Epithelial attachment (0.97 mm), (c) Connective tissue attachment (1.07 mm), (d) Biologic width (b+c)

B-Attachment level:

Is the distance between the base of the pocket and a fixed point on the crown, such as the cemento-enamel junction, it is measured by a periodontal probe. Clinical determinations of attachment level are more useful than pocket depths, because the latter may change due to displacement of the gingival margin and degree of inflammation, while changes in the level of attachment can be due only to gain or loss of attachment, this gives better indication for the degree of periodontal destruction. Shallow pockets attached at the level of the apical third

of the root represents more sever destruction than deep pockets attached at the coronal third of the roots.

- When the gingival margin is located at the level of CEJ. the loss of attachment equals the pocket depth.
- When the gingival margin is located apical to the CEJ. The loss of attachment will be greater than the pocket depth, and therefore the distance between the CEJ and the gingival margin should be added to the pocket depth to measure loss of attachment.
- When the gingival margin is located on the anatomic crown, the pocket depth will be greater than loss of attachment and therefore the distance between gingival margin and CEJ is subtracted from the pocket depth to measure level of attachment.

Measurement should be reproducible, this can be performed by the use of (a grooved acrylic stent)

C- Gingival indices:

the gingival index of Loe and Silness(1963) and the Sulcus Bleeding Index of Muhlemann and Son(1971) are the most useful in clinical evaluation of gingival health before and after treatment.

D-Alveolar bone level:

Is evaluated clinically by (trans-gingival probing) after anaesthetizing the area. It determines the height and contour of the facial and lingual bones obscured on the radiograph by the dense roots. The architecture of the interdental bone also can be evaluated.

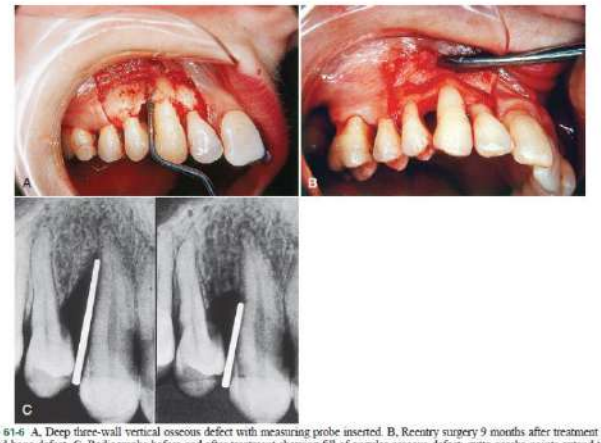
2-Radiographic methods:

standardized technique is needed for reproducible positioning of the film and the tube, even though, this technique is less reliable than clinical probing technique, because a sufficient loss should take place at the alveolar crest to be recognized radiographically (not sensitive).

3-Surgical re-entry:

evaluation can be performed by taking repeated impression. This can give a good view of the state of the bone crest that can be compared with the view taken during the initial surgical intervention. This method has 2 disadvantages:

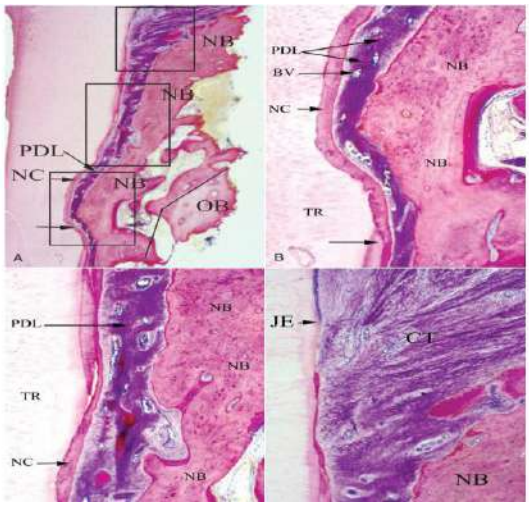
a. Requires unnecessary 2nd operation.
b. Does not show the type of attachment if it is new attachment or long junctional epithelium.



ıre 61-6 A, Deep three-wall vertical osseous defect with measuring probe inserted. B, Reentry surgery 9 months after treatment shows ared bone defect. C, Radiographs before and after treatment showing fill of angular osseous defect; gutta percha points extend to base

4-Histologic methods:

type of attachment can be determined only by histologic analysis of tissue blocks obtained from the healed area. Animal studies can be used because this method needs extraction of the examined tooth with its periodontium after successful treatment, therefore it's not used in humans.



Reconstructive surgical techniques:

Can be divided into two major approaches:

I. Non- bone graft associated new attachment.
II. Bone Graft associated new attachment or combination of both approaches.

I. Non-bone graft associated new attachment:

New attachment is more likely to occur when the destructive process has occurred very rapidly e.g after treatment of pockets with acute periodontal abscess, acute necrotizing ulcerative gingivitis ANUG.

Non–Graft-Associated Procedures

Removal of Junctional and Pocket Epithelium.
Junctional and pocket epithelium has been perceived as a barrier to successful therapy because its presence interferes with the direct apposition of connective tissue and cementum, thus limiting the height to which periodontal fibers can insert to the cementum. Several methods have been recommended to remove the junctional and pocket epithelium. These include curettage, chemical agents, ultrasonics, lasers, and surgical techniques.

Preventing or Impeding the Epithelial Migration. As with coronally displaced flap

Clot Stabilization, Wound Protection, and Space Creation.
Preservation of the root surface fibrin clot interface prevents apical migration of the gingival epithelium and allows for connective tissue attachment during the early wound-healing period. The importance of space creation for bone repair has long been recognized in orthopedic and maxillofacial surgery.

Laser-Assisted New Attachment Procedure. The
Role of laser in periodontal therapy remains controversial . Nevertheless, the use of neodymium : yttriumaluminum-garnet (Nd : YAG) to perform surgical LANAPs has been reported for the management of chronic periodontitis and can potentially result in new attachment and periodontal regeneration

Many questions remain about LANAP that need to be clarified. The first is the exact mechanism and parameter by which healing by new attachment versus regeneration occur with LANAP therapy. Additionally, a blinded split mouth study to compare LANAP protocol to other existing periodontal therapies is in progress and needs to be completed. This along with other randomized controlled trials will be needed for meta-analysis to determine if LANAP is equivalent or superior to other conventional therapy. As with all periodontal therapy, the long-term stability of the regenerationneed to be explored.

__Guided Tissue Regeneration.__ GTR is used for the prevention of epithelial migration along the cemental wall of the pocket and maintaining space for clot stabilization. this method is based on the assumption that periodontal ligament and perivascular cells have the potential for regeneration of the attachment apparatus of the tooth. GTR consists of placing barriers of different types (membranes) to cover the bone and periodontal ligament, thus temporarily separating them from the gingival epithelium and connective tissue. Excluding the epithelium and the gingival connective tissue from the root surface during the postsurgical healing phase not only prevents epithelial migration into the wound but also favors repopulation of the area by cells from the periodontal ligament and the bone.

Guided periodontal regeneration

- Generation 1= GTR(guided tissue regenerative membrane)
- Generation 2= Biomaterials as EMD (enamel matrix derivative protein), BMP(bone morphogenic protein), PRP(plasma rich protein).

Generation 3= Growth factors, stem cells, tissue engineering

Regenerative procedures

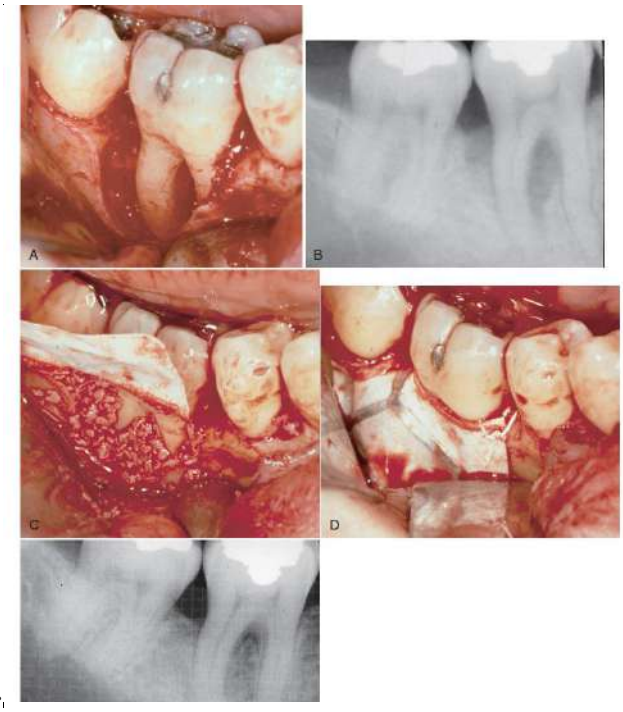
Periodontal regenerative procedures used to improve:

1. Local gingival architecture.
2. Function.
3. Prognosis of periodontitis involved teeth.

The intrabony defect (infrabony) could be classified according to the number of surrounding bone walls into:

1. One-wall defect.
2. Two-wall defect.
3. Three-wall defect.

The authors reported that new attachment had occurred in 2-wall and 3-wall defect but not in 1-wall defect.



- .

Principles of non-graft new attachment*: are based on*

1. Complete removal of all irritants with or without exposure of the area with a flap.
2. Occlusal adjustment may be indicated if there is trauma from occlusion.
3. Removal of the junctional and pocket epithelium: because it is a barrier to successful therapy due to interference with direct apposition of connective tissues and cementum limiting the height of insertion of periodontal fibers to cementum. Several methods have been recommended to remove junctional and pocket epithelia. These include curettage, chemical agents, ultrasonic methods,laser and surgical techniques. Because of lack of control over the first four methods, they are not currently use. Surgical techniques are recommended (the excisional new attachment procedure): consists of internal bevel incision with a surgical knife, which is performed either without flap but after carful scaling and root planning , an interproximal sutures are used to close the wound or it use with flap as now by the modified Widman flap operation.
4. Prevention of epithelial migration along the cemental wall of the pocket by guided tissue regeneration (GTR) which is the placement of barriers of different types to cover the bone and periodontal ligament excluding the epithelium and the gingival connective tissue from the root surface permit only PDL and bone cells to repopulate the area.

   Two types of membranes have been used:

A. Non-degradable (non resorbable): the one used clinically is the polytetra-fluoroethylene membrane (Gore-Tex) which can be obtained in different shapes and sizes to suit proximal spaces, facial and lingual surfaces of furcations, it must be removed after the initial healing stages (3-6 weeks).
B. Biodegradable (resorbable) membranes: are resorbed and therefore do not require a second intervention. These membranes include different resorbable materials: derived either from:

- Porcine collagen.

- Cecum of an ox.
- Polylactic acid.
- Synthetic skin (Biobrane).
- Freeze-dried dura mater.

The resorbable membranes resorbes at different periods as 4-18 weeks; 6-14 months.

Some studies use membranes with autogenic bone graft for better results specially in grade II furcations, or in interdental defect.

GTR disadvantages

- Non-resorbable
    A. 2nd surgery required after initial stage of healing 3-6 weeks
    B. Exposure to oral environment
    C. Bacterial contamination
    D. Failure of collapse.
- Resorbable
    A. Risk of exposure.
    B. Collapse into the defect area (bone filler is needed).
    C. Technical difficulties.
    D. Harmful degradation products of synthetic membranes.

5. Preparation of the root surface: changes in the root surface of periodontal pockets that interfere with new attachment are degeneration of remnants of sharpey's fibers, accumulation of bacteria and their product and disintegration of the cementum and dentin.

   These obstacles can be eliminated by thorough root planning but there are several substances can give better conditioning of the root surface for attachment of new connective tissue fibers, these include: 1.citric acid 2.fibronectin 3.tetracycline.

1. Citric acid: application of citric acid at Ph=1 for 2-3 or 5 minutes on planed root surfaces produced a surface demineralization that induced cementogensis and attachment of collagen fibers with prevention of apical epithelial migration along denuded roots.

2. Fibronectin: is a glycoprotein needed by fibroblasts to attach to root surface, addition of fibronectin locally but at the same level as that present in plasma may promote new attachment.
3. Tetracycline:(in vitro) it increases binding of fibronectin which in turn stimulates fibroblast attachment and growth while suppressing epithelial cell attachment and migration.

   Both citric acid and tetracycline remove the smear layer of microcrystalline debris that is formed on planed root surface. Thus exposing the dentinal tubules.

II. Graft new attachment:

Grafting procedure: to stimulate periodontal regeneration, the flap approach was combined with the placement of bone graft or implant materials into the curetted bony defect. These materials may actively induce bone formation or through it's own viability may deposit new bone. The various graft and implant materials used can be placed into four categories depend on their sources:

1. **Autogenous graft:** grafts transferred from one position to another within the same individual and are harvested either from intra oral or extra oral (iliac) donor site. It comprises:
   a. Cortical bone
   b. Cancellous bone of marrow. From max. tuberosity , edentulous areas ,and healing socket
   c. Bone blend which is combination of the previous two. bone is removed from predetermined site, triturated in a capsule to be workable ,plastic like mass and packed into bony graft
2. **Allograft:** a graft transferred between genetically dissimilar members of the same species (cadaver)
   a. **Viable cancellous bone and marrow.**
   b. Sterilized cancellous bone and marrow

   c.Freeze-deried bone.(FDBA).

Decalcified dried bone allograft is preferred because it has a higher osteogenic potential than freeze-dried bone graft. because the process of demineralization

followed by acid application exposed the component of bone matrix which are closely associated with collagen fibrils as bone morphogenic protein (BMP)

3. **Hetro- or xenografts:** grafts taken from a donor of another species (Calf ox bone).Bio-Oss is the most widely used, it's an inorganic bovine derived bone. in periodontology bio-oss used as graft material covered with resorbable membrane

Both allograft and xenograft are considered foreign, thus provoke an immune response, this antigenicity should be suppressed through radiation, freezing and chemical therapy.

4. **Alloplastic materials (non-bone graft synthetic material):** inert implant calcium phosphate bio materials which have been used as substitutes for bone grafts ex:

a. Hydroxyapatite: similar to that found in bone, it is non bioresorbable.
b. Tricalcium phosphate: is partially bioresorbable.
   Other alloplastic materials other than calcium phosphate are; sclera, cartilage, plaster of paris and bio active glass

The grafting materials introduce in treating periodontal disease may either:

- Contain bone forming cells (osteogensis) ex : DDB allograft.
- Serve as scaffold for bone formation (osteo conduction) ex BIO-OSS: Alloplastic materials.
  They induce bone formation when placed next to viable bone but not when surrounded by non-bone forming tissue such as skin.
- Induce bone formation (osteoinduction) because the matrix of bone graft contains bone-inducing substances.

GENERATION 2 (Bio active materials)

Enamel matrix Derivatives: Emdogain, enamel matrix protein mainly derived (Amelogenin) are secreted by Hertwigs epithelial root sheath during tooth development and induce acellular cementum formation. These proteins are believed to favour periodontal regeneration. The available derivative obtained from porcine teeth name's (emdogain) which is available as gel consisted in 90% is amelogenin with the rest are primarly proline-rich non amelogenin, tuftelin, tuft protein, etc.

Acellular dermal matrix allograft (ADMA): acellular human cadaver skin is a relatively new type of bioresorbable grafting material that has been obtained from tissue skin (Alloderm).

One of the major advantages of ADMA is that it's basically an immunologically inert avascular connective tissue. This is due to the fact that most of the targets of rejection response have been eliminated during the initial deepithelization and decellularization processes.

In periodontal surgery, the use of ADMA has been recommended in the management of ridge deformities , also in increasing keratinized tissue around teeth and dental implants and for root coverage.

It could be used in combination with EMD in treating gingival recession.

GENERATION3 (Growth regulatory factors for periodontal regeneration)

Growth factor is a general term to denote a class of polypeptide hormones that stimulate a wide variety of cellular events such as proliferation, chemotaxis, differentiation and production of extracellular matrix protein.

Proliferation and migration of periodontal ligament cells and synthesis of extracellular matrix as well as differentiation of cementoblasts and osteoblasts is a prerequisite for obtaining periodontal regeneration. Therefore, it is conceivable that growth factors may represent a potential aid in attempts to regenerate the periodontium.

The effect of various growth factors were studied in vitro, and a significant regeneration potential of growth factors was also demonstrated in animal models. These growth factors

primarily secreted by macrophage ,endothelial cells ,fibroblast and platelets.The important growth factors are:

- Platelet derived growth factors (PDGF).
- Insulin-like growth factor (IGF).
- Bone morphogenetic proteins (BMPs)
- Transforming growth factor(TGF)

Ideal requirements of Bio-Materials

- Biocompatibility.
- Enhancement of clinical attachment level.
- Reduction of probing depth.
- Hard tissue fill of the intrabony defects.

Factors influencing the success or failure of all regeneration techniques:

- Plaque control.
- Systemic status that affect the periodontium.
- Traumatic injury to teeth or tissues
- Root preparation
- Wound closure
- Soft tissue approximation
- Post operative and long term maintenance.