

THIS IS JUST A SUPPLEMENTARY MATERIAL TO HELP IN YOUR EXAMINATIONS; IT SHOULD NOT REPLACE YOUR TEXT BOOKS. KINDLY GO THROUGH YOUR TEXTBOOKS FIRST FOR BETTER UNDERSTANDING OF THESE NOTES.

GINGIVA:

It is the part of the oral mucosa that covers the alveolar processes of jaws and surround the neck of teeth. It is made up of stratified squamous epithelium which may be keratinized or non keratinized but is mostly para keratinized.

Gingiva is anatomically divided into:

Marginal gingiva

Attached gingiva

Interdental gingiva

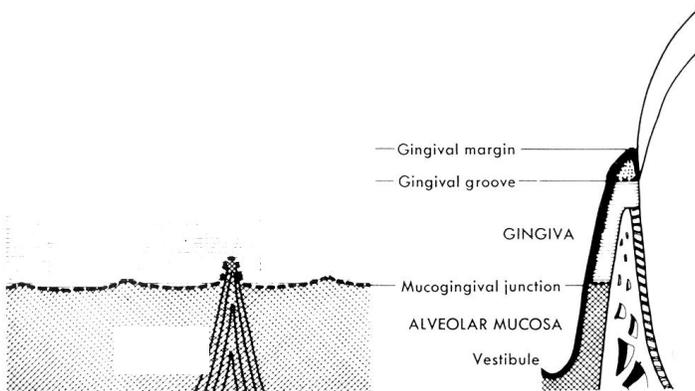
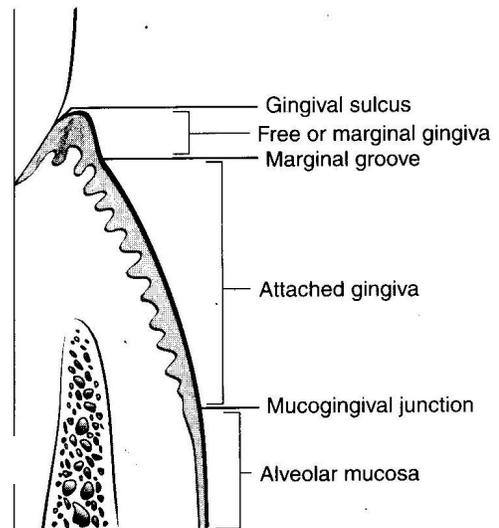


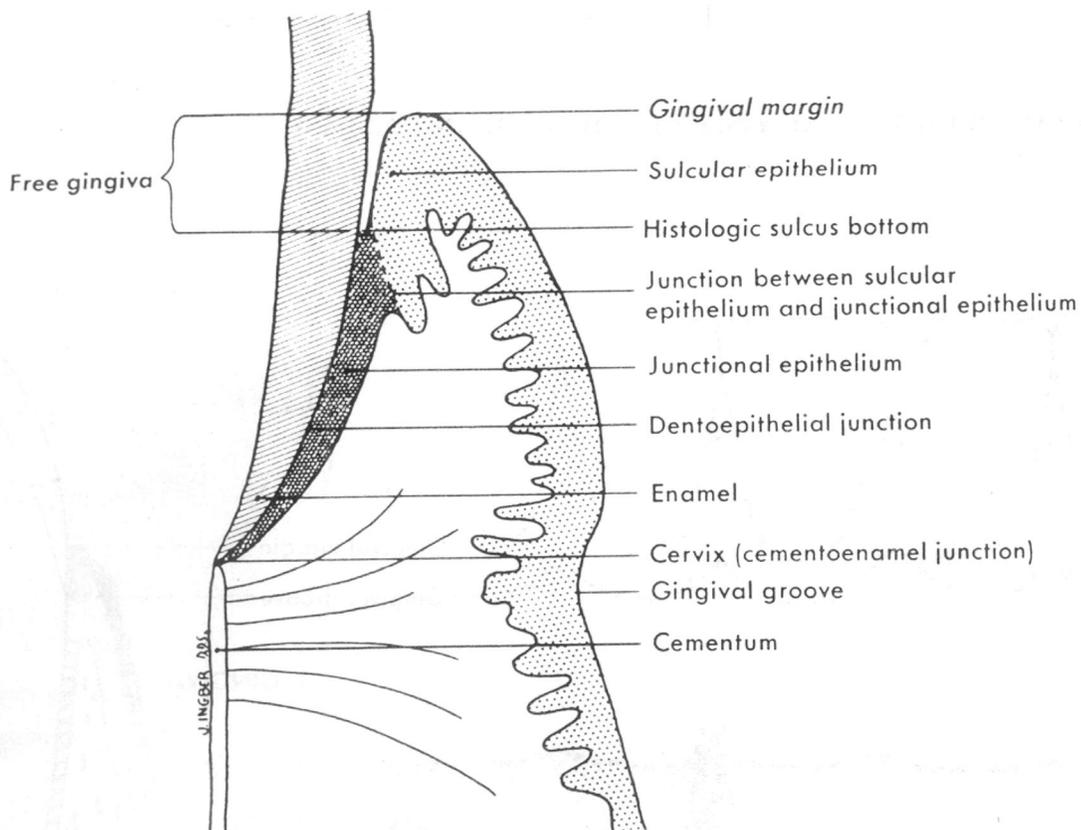
Fig. 1-1. Anatomic relationships of normal gingiva.



2 Diagram showing anatomic landmarks of the gingiva.

MARGINAL GINGIVA:

- It is terminal unattached border of gingiva surrounding the teeth in collar like fashion.
- In about 50% of cases, it is demarcated from attached gingiva by a shallow linear depression called the Free Gingival Groove.
- Usually about 1mm wide, it forms the soft tissue wall of the gingival sulcus.
- It may be separated from the tooth surface with a periodontal probe.



Histologic relationships of marginal gingiva.

Free gingival groove is located at approximate level with the bottom of gingival crevice.

Microscopic investigation suggest that it's absence or presence as well as its location is dependent upon special arrangement of supra-alveolar fibres running from the cementum into free attached gingiva.

GINGIVAL SULCUS:

- It is the shallow crevice or space around the tooth bounded by the surface of the tooth on one side and epithelium lining the free margin of gingiva on other.
- It is V-shaped space and rarely permits the entrance of a periodontal probe.
- Under absolutely normal condition the depth of gingival sulcus is zero or about zero. These conditions can be produced only in animals or after intense prolonged plaque control.

Variations in depth :-

1.8 mm (0-6 mm) - Orban & Kohler

1.5 mm -Weski O

0.69 mm -Garguilo

Probing depth clinically is: 2-3 mm

ATTACHED GINGIVA:

- It is continuous with marginal gingiva. It is firm, resistant and tightly bound to underlying periosteum of alveolar bone.
- The facial aspect extends to the relatively loose and moveable alveolar mucosa from which it is demarcated by mucogingival junction.
- The width of attached gingiva is an important clinical parameter. It is distance between the mucogingival junction and projection of external surface of the bottom of the gingival sulcus or the external surface of the bottom of the gingival sulcus or the periodontal pocket.

- **Ainamo and Loe in 1966** found that it is greatest in incisor region (3.5 mm – 4.5 mm in maxilla and 3.3 – 3.9mm in mandible) and has least width in premolar region i.e. is 1.9mm in maxilla and 1.8mm in mandible.
- The width of attached gingiva increase with age and in supraerupted teeth.

Significance of Attached Gingiva for the maintenance of Periodontal health :

- i. To protect the periodontium from injury caused by frictional forces encountered during mastication.
- ii. To dissipate the pull on the gingival margin created by the muscles of the adjacent alveolar mucosa.
- iii. Gives support to the marginal gingiva
- iv. Provides solid base for movable alveolar mucosa
- v. Helps to prevent soft tissue recession and attachment loss
- vi. Helps in connective tissue attachment

INTERDENTAL GINGIVA:

- It occupies the gingival embrasures which is the interproximal space beneath the area of tooth contact.
- It can be Pyramidal in shape (tip of the one papilla is located immediately beneath the contact point) or have a ‘col’ shape (it presents a vally like depression that connects facial and lingual papilla and conforms to the shape of the interproximal contact).
- The shape of the gingiva in a given interdental space depends on the contact point between the two adjacent teeth and the presence or absence of some degree of recession.

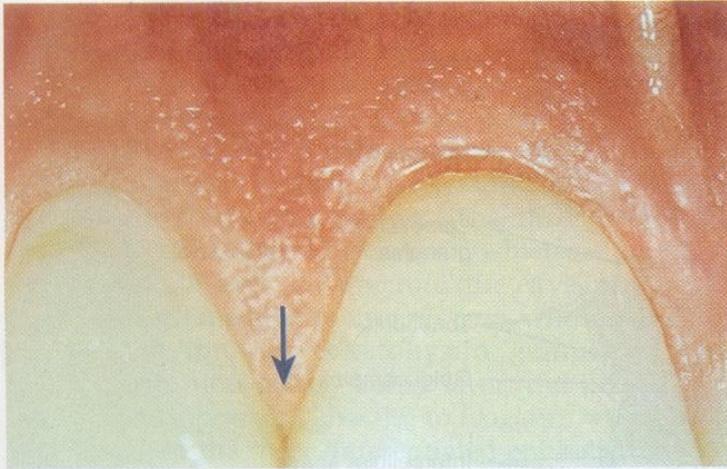
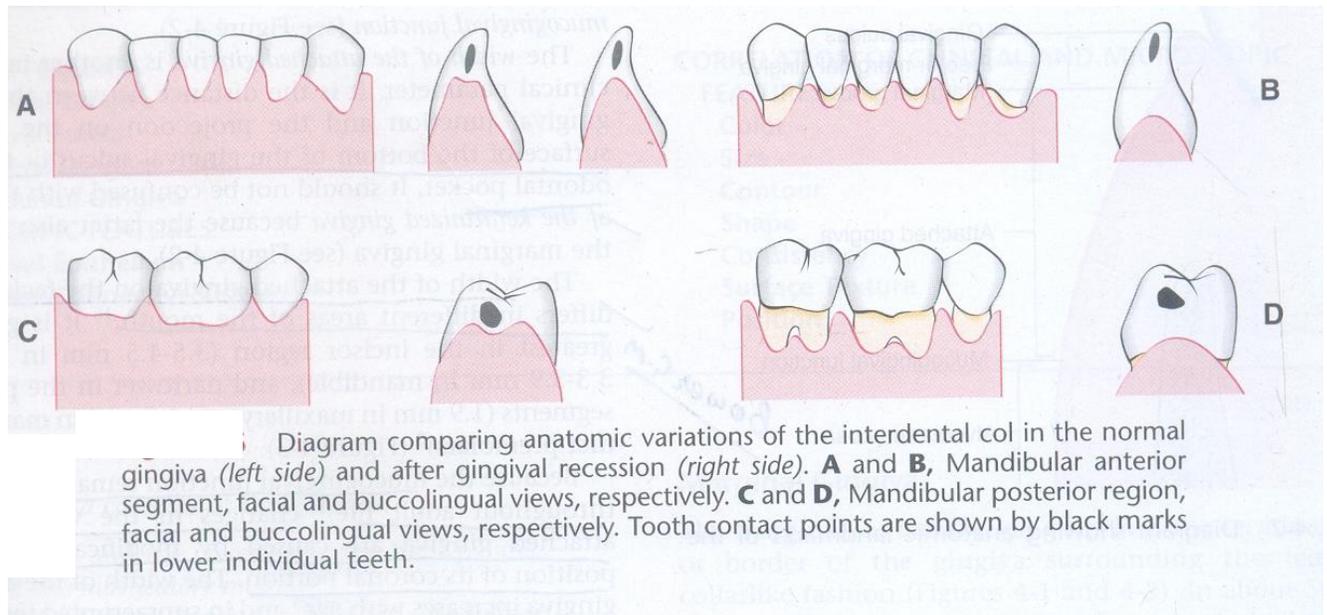


Figure 4-7 In papillae (*arrow*) with central portion formed by attached gingiva. The shape of the papillae varies according to the dimension of the gingival embrasure. (Courtesy Dr. Osvaldo Costa.)



Figure 4-8 All interdental papillae and col where proximal tooth contact is missing. (Courtesy Dr. Osvaldo Costa.)

- The facial and lingual surfaces are tapered and the mesial and distal surfaces are slightly concave. If a diastema is present the gingiva is firmly bound over interdental bone and interdental papilla is absent.



MICROANATOMY:

EPITHELIUM:

- Gingiva is covered with stratified squamous epithelium with architectural characteristic specific for areas related to teeth.
- Gingival epithelium can be divided into:
 - Oral epithelium
 - Sulcular epithelium
 - Junctional epithelium

ORAL EPITHELIUM:

- Gingival oral epithelium faces the oral cavity.
- Extends from gingival margin to mucogingival junction.
- Covers the clinically visible part of free and attached gingiva.
- Principal cell → keratinocyte
- Completely matured cell is called corneocyte.
- Have the ability to produce cytoplasmic keratin filament (tonofilaments).
- Function is to provide strength to epithelial sheet.
- Surface epithelia are constantly renewed tissues characterized by surface sloughing of mature keratinocytes and cell renewal by mitotic division of keratinocytes in the basal layer.
- 10 days are required for new cells to transverse epithelium and reach stratum corneum. This interval is called as epithelium turn over time.
- Genetically determined sequent of even during movement of keratinocytes from basal cell layer toward the surface is called as terminal differentiation.

The hallmark features of keratinization are:

- Increase in the number of tonofibrils
- Cells lose their nucleus and other cell organelles
- Progressive flattening of cells
- Cells lose their capacity to divide

If nuclei is lacking in cornified layer of keratinized cells, epithelium as well as stratum corneum itself is orthokeratinized.

If cells contains pyknotic nuclei-parakeratinized

Cell layers of keratinized epithelium:

- Stratum basale
- Stratum spinosum

- Stratum granulosum
- Stratum corneum

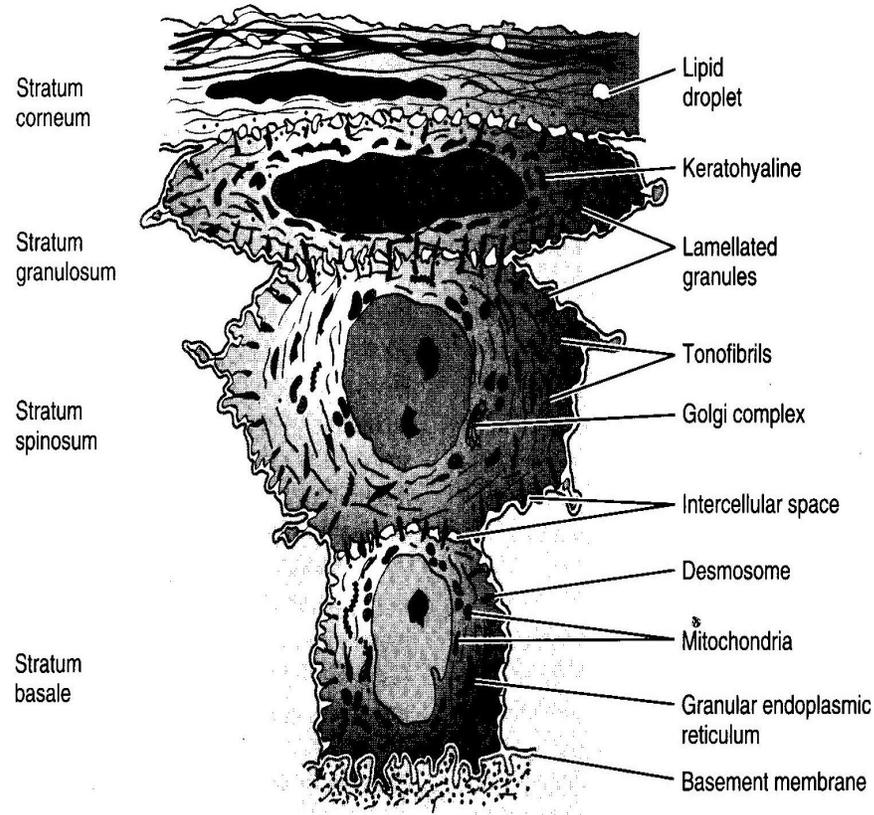


Fig. 1-9 Diagram showing representative cells from the various layers of stratified squamous epithelium as seen by electron microscopy. (Modified from Weinstock A. In: Ham AW: Histology, ed 7. Philadelphia, JB Lippincott, 1974.)

Stratum basale:

- Germinative layer
- cells are cuboidal / columnar
- Appears about 1 micrometer wide in electron microscope.
- Positive to periodic acid Schiff stain indicating carbohydrate presence.
- Contain bundles of tonofibrills.

Stratum spinosum:

- Several rows of large elliptical or spherical cells above basal layer.
- Large intercellular spaces with short cytoplasmic processes.
- Consists of 10-20 layers.
- Contains tonofibrils bundles.
- Membrane coating granules appears in upper part of this layer.
- These are called odland bodies / keratinosomes.
- They are modified lysosomes, contain acid phosphatase required for destruction of organelle membrane.

Stratum granulosum:

- Flattened cells
- Contain keratohyaline granules associated with tonofibrils.
- Membrane – coating granules fuse with cells membrane in upper part.
- Internal membrane thickening also occurs.
- Granules stain intensely with acid dyes such as haematoxylin (basophilic).
- Keratohyalin granules contain protein fillagrin, which forms the matrix of fully matured corneocyte.

Stratum corneum:

- Extremely flattened and dehydrated cells.
- All organelles are lost.
- Cells filled with packed fibrillar material.
- Stain pink with eosin (eosinophilic).

Thus, a fully differentiated corneocyte contains a matrix of fillagrin with increased number of tonofibrils and a resistant envelop that surrounds the cell.

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Tonofilaments are fibrous proteins synthesized by ribosomes and have approx diameter of 8mm. Belong to class of intercellular filaments called intermediate filaments.

Chemically, filament present a class of intercellular proteins known as “cytoperatin” when they become aggregated to form bundles of filaments called “tonofibrills”.

Keratins present 30 different proteins of differing molecular weight, those with lowest molecular weight (40 KD) found in glandular and single epithelium. Those of intermediate molecular weight in stratified epithelium and those with highest molecular weight (67 KD) is keratinized stratified epithelium.

All stratified oral epithelium possess K5 and K14 but difference emerge between keratinized oral epithelium which contain K1, 4, 10 and 16 and non keratinized epithelium which contains K4, 13, 19.

K1 K2 K10 -12 -> Oral epithelium
K6 K16 are highly proliferative but are exception in Junctional epithelium as Junctional epithelium is highly proliferative but K6 K16 are absent. K5 K14 K19 present in Junctional epithelium.
K1 K6 K10 K16 -> keratinized oral epithelium
K4 K13 K19 -> Non – keratinized oral epithelium (sulcular epithelium)

ALL STRATIFIED ORAL EPITHELIUM CONTAIN K5 AND K14.

BASAL LAMINA:

Basement membrane is composed of two quite distinct components.

- A thin finely febrile layer, the basal lamina, associated closely with the cells surface.
- A more distinct variable reticular lamina of larger fibrils and ground substance.
- Is usually about 80 nm thick.
- Consist of a fibrillar layer, the lamina densa (20-50 nm wide) separated from the plasma membrane of the adjacent cells, by a narrow electron – lucent zone, lamina lucida.
- The lamina lucida shows granular or fibrillar features representing the presence of various proteoglycans, the extra cellular domains of integrins and other small glycoproteins.
- Although all basal lamina have similar form, their detailed organization and sizes vary with their tissue sites.
- At the epidermal – dermal junction, special glycoproteins are present in the lamina lucida, and hemidesmosomes also anchor the neighbouring keratinocytes to lamina densa, and in various other features associated with the specialized filling functions of their structures.

FUNCTION:

- They form selectively permeable barriers between adjacent tissues in some cases.
- They have mechanical function forming anchoring intermediaries between epithelial and connections tissue, assisting to stabilize and orientate the tissue layers.
- They also have profound route of cell division, metabolism, movements and repair.
- They act as pathway for the migration and routing of growing cells process, for example in regeneration in the peripheral nervous system injury when

basal lamina components guide the outgrowth of axons and the reestablishment of neuromuscular junction.

- Changes in basal lamina thickness are also often associated with pathological conditions.

NON KERATINOCYTES

- Melanocytes
- Langerhan's cells
- Merckel cells

MELANOCYTES:

- Located in basal layer.
- have no desmosomes or tonofilaments.
- Have dendritic process.
- Color of oral mucosa is due to presence of melanin.
- Melanin is produced by pigment cells called melanocytes.
- Arise embryologically from neural crest ectoderm.
- enter epithelium at 11 weeks of gestration.
- Melanin is synthesized within Melanocytes as small structures called Melanaosomes.
- Lightly and darkly pigmented individuals have same number of melanocytes but color differences results from relative activity of melanocytes producing melanin.
- Melanosomes contain enzyme tyrosinase which hydroxylates tyrosine to DOPA which is converted to melanin.
- Melanin is phagocytosed and contain within other cells of epithelium and connective tissue called melanophage or menalophores.

FUNCTION:

To prevent light from reaching adjacent cells.

LANGERHANS CELLS:

- Lacks desmosomal attachment.
- Seen above basal layer i.e. among keratinocytes.
- Appears as clear cell.
- Modified monocytes derived from bone marrow and lymphocytes.
- Contain elongated granules and have antigenic properties.
- Play important role in immune reaction as antigen presenting cells for lymphocytes i.e. immunocompetent presentation cells.
- Contain small rod or flask shaped granules called BIRBECK'S GRANULES.
- Can migrate from epithelium to regional lymph nodes.
- Found in normal oral epithelium and small amount in sulcular epithelium, absent in Junctional epithelium.
- They have marked adenosine triphosphatase activity.

MERKEL CELLS (TACTILE RECEPTORS)

- Situated in basal layer.
- Not dendritic but possesses keratin tonofilaments and desmosomes linking to adjacent cells.
- Contain small membrane – bound vesicles in the cytoplasm, situated adjacent to nerve fibres associated with the cell.
- Act as tactile receptors.
- Cells may arise from division of an epithelial cells (keratinocytes)

DEVELOPMENT OF GINGIVAL SULCUS:

After enamel formation is complete, enamel is covered with reduced enamel epithelium which is attached to tooth by basal lamina and hemidesmosomes.

When tooth penetrates oral mucosa, reduced enamel epithelium unites with oral epithelium and transforms to Junctional epithelium. As tooth erupts, this united epithelium condenses along the crown and ameloblasts, which form inner layer of reduced enamel epithelium becomes squamous epithelial cells.

Transformation to Junctional epithelium takes places in apical direction.

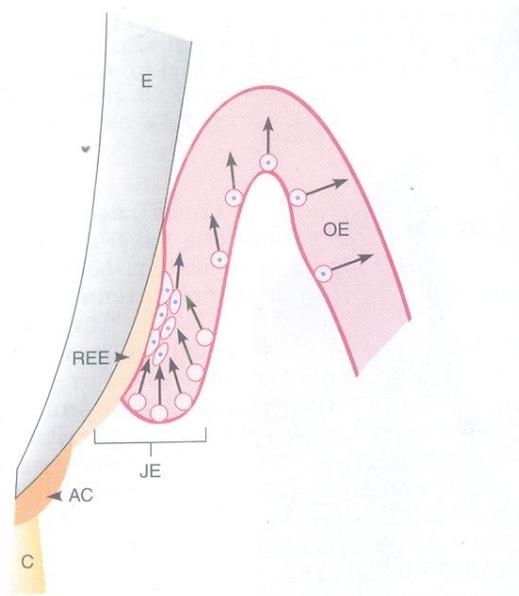


Figure 4-18 Junctional epithelium on an erupting tooth. The junctional epithelium (JE) is formed by the joining of the oral epithelium (OE) and the reduced enamel epithelium (REE). AC, Afibrillar cementum, sometimes formed on enamel after degeneration of the REE. The arrows indicate the coronal movement of the regenerating epithelial cells, which multiply more rapidly in the JE than in the OE. E, Enamel; C, root cementum. A similar cell turnover pattern exists in the fully erupted tooth. (Modified from Listgarten MA: *J Can Dent Assoc* 36:70, 1970.)

According to Schroeder and Listgarten, this process takes, 1-2 years.

Gingival sulcus is formed when tooth erupt into oral cavity. Then Junctional epithelium and reduced enamel epithelium form a band attached to tooth surfaces near tip of crown to cemento-enamel junction.

SULCULAR EPITHELIUM:

This lines the gingival sulcus. It is thin, non keratinizing stratified squamous epithelium without retepegs and extends from the coronal limit of Junctional epithelium to the crest of gingival margins. It is not keratinized however it has potential to keratinize if:

- If it is reflected and exposed to oral cavity.
- If bacterial flora of sulcus are totally eliminated.

It contains K4, K13, K19 keratins.

The sulcular epithelium is extremely important because it may act as a semipermeable membrane through which tissue fluid from gingiva seeps into the sulcus and bacterial products pass in the reverse direction.

JUNCTIONAL EPITHELIUM:

- This consists of a collar like band of stratified squamous non-keratinizing epithelium. It is 3-4 layers thick but the number of layers increases with age to 10 or even 20. Its length ranges from 0.25mm – 1.35mm.
- The Junctional epithelium is attached to tooth surface (epithelial attachments) by means of a basal lamina which consists of a lamina densa and lamina lucida. The Junctional epithelium attaches to afibrillar cementum when it is present on crown and to root cementum in similar fashion.
- Junctional epithelium expresses K5, K14, K19 keratins
- Junctional epithelium lacks K6, K16 as an exception.

GINGIVAL CONNECTIVE TISSUE:

The connective tissue of the gingiva is known as the lamina propria and consists of

- Papillary layer subjacent to the epithelium that consists of papillary projections between the epithelial rete pegs.
- Reticular layer contiguous with the periosteum of the alveolar bone.

Function of Gingival Connective Tissue :

- The gingival connective tissue serves primarily to protect the root surface and alveolar bone from the external oral environment.
- It aids in the support and fixation of teeth within their alveolar housing and provides adequate support for the epithelial tissues.
- In carrying out its protective role, the gingival tissues provides the stage upon which the host response acts out its role of surveillance, interception and removal of foreign materials.
- Under healthy conditions, there is a delicate balancing act played out in the gingival tissues involving both tissue repair and tissue destruction.

Connective tissue consists of :

Cellular compartment

- Fibroblasts
- Mast cells
- Macrophages
- Inflammatory cells

Exrtra cellular compartment

- Fibers
- Ground substance
- Nerves and vessels

Cellular Elements of Connective Tissue :

Fibroblasts :

- The principal function of fibroblasts is to synthesize and maintain the components of the extracellular matrix of the connective tissue.

- They are also involved in a number of regulatory process necessary for maintenance of tissue hemostasis. Fibroblasts are specifically involved in these process through phagocytosis and the secretion of collagenases.
- Fibroblasts (including gingival fibroblasts) synthesize a wide range of matrix metalloproteinases capable of degrading collagens, proteoglycans and other matrix components.

Mast Cells :

- Numerous in the connective tissue of the oral mucosa and gingiva.
- These are responsible for the production of certain component of matrix.
- Also produce vasoactive substances which can affect the function of the microvascular system and control the flow of blood through the tissue.

Fixed Macrophages and Histiocytes :

Are present in the gingival connective tissue as components of the mononuclear phagocyte system (reticuloendothelial system) and are derived from blood monocytes.

Inflammatory Cells :

- Neutrophils can be seen in relatively high numbers in both the gingival connective tissue and the sulcus.
- Small foci of plasma cells and lymphocytes are found in the connective tissue near the base of the sulcus.
- These inflammatory cells usually are present in small amounts in clinically normal gingiva.

Ground Substance :

Fills the space between fibers and cells, is amorphous, and has a high content of water.

Its main constituents are protein carbohydrate macromolecules which are divided into :

- i. Proteoglycans: glycosaminoglycans as carbohydrate unit eg. Hyaluronic acid and chondroitin sulfate.
- ii. Glycoproteins : Fibronectin and Laminin.
 - Fibronectin binds fibroblasts to the fibers and many other components of the intercellular matrix, helping mediate cell adhesion and migration.
 - Laminin found in basal lamina serves to attach it to the epithelial cells.

Ground substance matrix is necessary :

- As a medium in which connective tissue cells and fibers are embedded.
- For maintenance of the normal function of connective tissue
- For transposition of water, electrolytes, nutrients, metabolites to and from the cells.

Fibres of Connective Tissue :

Are produced by fibroblasts and are collagen, reticular, and elastic.

Collagen Fibers :

- Predominate in gingival connective tissue
- Collagen type I forms the bulk of the lamina propria and provides the tensile strength to gingival tissue.
- Type IV fibers branches between type I bundles and is continuous with fibres of the basement membrane and blood vessel walls.

The gingival fibers have the following functions :

- To brace the marginal gingiva firmly against the tooth

- To provide the rigidity necessary to withstand the forces of mastication without being deflected away from the tooth surface.
- To unite the free marginal gingiva with the cementum of the root and the adjacent attached gingiva.

The gingival fibers are arranged in bundles with a distinct orientation :

1. **Dentogingival fibers** : arise from the cementum of the root immediately apical to the base of the epithelial attachment, generally near the CEJ and splay out into the gingiva.
2. **Alveologingival fibers** : arise from the crest of the alveolus and course coronally, terminating in the free and papillary gingiva.
3. **Circular fiber:** group passes circumferentially around the cervical region of the tooth through the connective tissue of marginal and interdental gingiva.

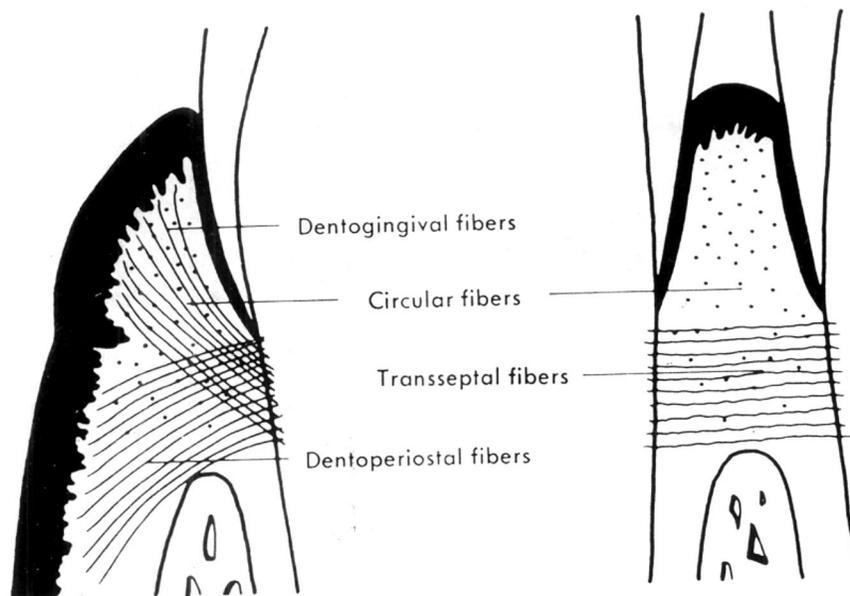


Fig. 1-26. Fiber arrangements of gingival connective tissue.

4. **Semicircular fibers:** arise from the cementum of the proximal root surface, just apical to circular fiber group, inserted into and traverse the facial and

- lingual free gingiva and insert into a comparable position on the opposite side of the same tooth
5. **Transgingival fibers:** arise from the cementum in the region of the CEJ of the one tooth and extend into the free marginal gingiva of the adjacent tooth.
 6. **Transeptal fibers:** arise from the cementum surface just apical to the base of the epithelial attachment, traverse the interdental bone and insert into a comparable region of the adjacent tooth.
 7. **Intergingival fibers:** extend along the facial and lingual marginal gingiva from tooth to tooth.
 8. **Interpapillary fibers:** runs in buccolingual direction through the interdental tissue.

HISTO CHEMISTRY

It gives the chemical composition & Enzyme systems of normal gingiva

- Connective tissue –PAS positive heteropolysaccharide intercellular ground substance & Glycogen
- In epithelium glycogen is inversely related to keratinisation
- PAS positive thin basement membrane
- RNA more in basal cells
- RNA &DNA activity seen more in gingival margin & Junctional ep
- Other components – sulfhydryls, disulphides , phospolipids & cholestral

ENZYMES

- Alkaline phosphatase – keratinised & parakeratinised layers
- Acid phosphatase – surface & prickle cell : related to keratinisation
- Nucleotide reductases – all layers except in keratin layers
- Acetylcholinesterase – Connective tissue
- Esterase – basal & granular layers

- Glucose 6 phosphate dehydrogenase – increased from basal to superficial layers
- Succinic dehydrogenase – increased in superficial layers
- Cytochrome oxidase – Junctional epithelium & sulcular epithelium

BLOOD SUPPLY TO THE GINGIVA :

Gingival connective tissue is highly vascular. The vessels have their origins in the periodontium and extend into the lamina propria of the gingiva.

Three sources of blood supply to the gingiva are :

- **Supra-periosteal arterioles:** Along the facial lingual surface of the alveolar bone, from which capillaries extend along the sulcular epithelium and between the retepegs of the external gingival surface. Occasional branches pass to the periodontal ligament through alveolar bone or run over the crest of the alveolar bone.
- **Vessels of the periodontal ligament:** which extend into the gingiva and anastomoses with capillaries in the sulcus area.
- **Arterioles emerging from the crest of the interdental septa:** They extend parallel to the bony crest to anastomose with the capillaries in the gingival crevicular areas and with vessels that run over the alveolar crest.

Capillaries extend into the papillary connective tissue between the epithelial retepegs in the form of terminal **hairspin loops** with afferent and efferent branches, spiral and varices. There are also flattened capillaries which serve vessels when the circulation is increased in response to irritation. Capillaries are arranged as flat, anastomosing plexus that extend parallel to the enamel from the base of the sulcus to the gingival margin. In the col area there is a mixed pattern of anastomosing capillaries loops.

GINGIVAL LYMPHATIC DRAINAGE :

- Brings in the lymphatics of the connective tissue papillae.
- It progresses into the collecting network external to the periosteum of the alveolar process and then to the regional lymphnodes (particularly the submaxillary group).
- In addition lymphatics just beneath the junctional epithelium extend into the periodontal ligament and accompany the blood vessels.

GINGIVAL INNERVATION :

- Derived from fibers arising from nerves in the periodontal ligaments and from the labial buccal and palatal nerves.
- Receptors are seen as free endings within the papillary layer of the lamina propria.
- The following nerve structures are present in connective tissue, a meshwork of terminal argyrophilic fibers, some of which extend into the epithelium.
 - Meissner type tactile corpuscles – Touch receptors
 - Krause type of end bulbs - Temperature receptors seen as coiled terminals.
 - Fine fibers in the papilla pain receptors.

All are found in the free and attached gingiva.

CLINICAL FEATURES OF GINGIVA:

COLOR

Of marginal and attached gingiva is generally described as cord pink and is produced by:

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- Vascular supply
- Thickness
- Degree of keratinization
- Melanin content

Colour is lighter in blond individuals with fair complexion and darker in brunettes. The alveolar mucosa is red, smooth and shiny.

PHYSIOLOGIC PIGMENTATION:

Melanin is responsible for the normal pigmentation of the skin, gingiva and remainder of oral mucous membrane.

SIZE:

Of the gingiva, correspond to the sum total of bulk of cellular and intercellular elements and their vascular supply. Alteration in size is a common feature of gingival disease.

CONTOUR:

(Knife edged, deflecting and scalloped) depends upon the shape of teeth and their alignment in the arch, the location and size of area of proximal contact and dimension of embrasures.

The marginal gingiva envelops the teeth in a collar like fashion and follows a scalloped outline on facial and lingual surface. It forms straight line and flat surfaces and arcuate contour is accentuated in teeth with pronounced mesiodistal convexity.

SHAPE:

When interproximal space is less, the interdental gingiva is narrow mesiodistally and vice – versa. The shape of interdental gingiva depends on the contour of proximal surfaces and location and shape of gingival embrasures.

CONSISTENCY:

The gingiva is firm and resilient and with exception of movable free margins, it is tightly found to the underlying bone.

SURFACE TEXTURE:

Gingiva presents a textured surface like that of orange peel and is referred to as being stippled. The attached gingiva is stippled whereas marginal gingiva is not. It is less prominent on lingual surface and may be absent in some people. It is absent in infancy, appear at about 5 years of age, increases until adulthood and begins to disappear in old age. It is also absent in inflamed gingiva.

Microscopically stippling is produced by alternate rounded protruberances and depressions in the gingiva surface. Papillary layer of connective tissue projects into elevation and elevated and depressed areas are covered by stratified squamous epithelium.

Stippling is formed of adaptive specialization or reinforcement for function. It is a feature of healthy gingiva and absence of stippling is common sign of gingival disease.

POSITION:

Refers to the level at which gingival margin is attached to tooth. When tooth erupts in oral cavity, margins and sulcus are at tip of crown, as eruption progresses they are closure to root.

CONTINUOUS TOOTH ERUPTION :

According to the concept of continuous tooth eruption, eruption does not stop when teeth meet their functional antagonist.

TWO PHASES OF CONTINUOUS TOOTH ERUPTION :

ACTIVE. 2. PASSIVE PHASE.

Active phase is the movement of the teeth in the direction of the occlusal plane.

Passive phase is the exposure of the teeth by apical migration of the gingiva.

This concept distinguishes between anatomic crown (portion of the tooth covered by enamel) and anatomic root (portion of the tooth covered by cementum) and between the clinical crown (part of the tooth that has been denuded of its gingiva and projects into the oral cavity) and clinical root (portion of the teeth covered by periodontal tissues.

Active eruption is coordinated with attrition; the teeth erupt to compensate for tooth substance worn away by attrition. Attrition reduces the clinical crown and prevents it from becoming disproportionately long in relation to the clinical root, thus avoiding excessive leverage on the periodontal tissues

Although originally thought to be a normal physiologic process, passive eruption is now considered a pathologic process. Passive eruption is divided into the following four stages.

- **Stage 1:** The teeth reach the line of occlusion. The Junctional epithelium and base of the gingival sulcus are on the enamel.
- **Stage 2:** The Junctional epithelium proliferates so that part is on cementum and part is on the enamel. The base of the sulcus is still on the enamel.
- **Stage 3:** The entire Junctional epithelium is on the cementum, and the base of the sulcus is at the cemento-enamel junction.

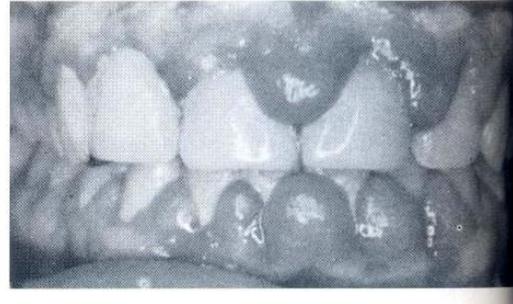
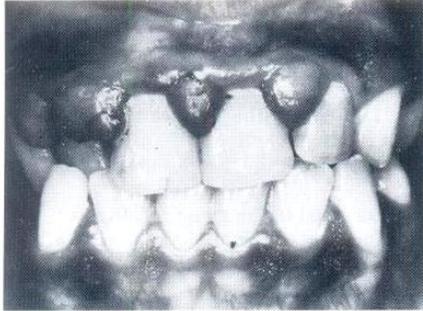
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- **Stage 4:** The Junctional epithelium has proliferated farther on the cementum, a portion of which is exposed. Proliferation of the Junctional epithelium onto the root is accompanied by degeneration of gingival and periodontal ligament fibers and their detachment from the tooth.

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GINGIVAL ENLARGEMENT



Increase in size of the gingiva is a common feature of gingival disease (Color Fig. 18-1). Accepted current terminology for this condition is gingival enlargement and gingival overgrowth.

The many types of gingival enlargement can be classified according to etiologic factors and pathologic changes as follows:

I. Inflammatory enlargement

A. Chronic

B. Acute

II. Drug-induced enlargement

III. Enlargements associated with systemic diseases

A. Conditioned enlargement

1. Pregnancy

2. Puberty

3. Vitamin C deficiency

4. Plasma cell gingivitis

5. Nonspecific conditioned enlargement (granuloma pyogenicum)

B. Systemic diseases causing gingival enlargement

1. Leukemia

2. Granulomatous diseases (Wegener's granulomatosis, sarcoidosis, and so on)

IV. Neoplastic enlargement (gingival tumors) A. Benign tumors

B. Malignant tumors

V. False enlargement

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Using the criteria of location and distribution, gingival enlargement is designated as follows:

Localized: Limited to the gingiva adjacent to a single tooth or group of teeth

Generalized: Involving the gingiva throughout the mouth

Marginal: Confined to the marginal gingiva Papillary: Confined to the interdental papilla

Diffuse: Involving the marginal and attached gingivae and papillae

Discrete: An isolated sessile or pedunculated tumorlike enlargement

The degree of gingival enlargement can be scored as follows' ⁶:

Grade 0: No signs of gingival enlargement

Grade I: Enlargement confined to interdental papilla Grade II: Enlargement involves papilla and marginal gingiva

Grade III: Enlargement covers three quarters or more of the crown

INFLAMMATORY ENLARGEMENT:

Gingival enlargement may result from chronic or acute inflammatory changes. The former is by far the more common. In addition, inflammatory enlargements commonly are a secondary complication to any of the other types of enlargement, creating a combined gingival enlargement. In these cases it is important to understand the double etiology and treat them adequately.

Chronic Inflammatory Enlargement:

Clinical Features:

Chronic inflammatory gingival enlargement originates as a slight ballooning of the interdental papilla and/or the marginal gingiva.

In the early stages it produces a life preserver-shaped bulge around the involved teeth.

Dr. Prerna Agarwal

This bulge can increase in size until it covers part of the crowns. The enlargement may be localized or generalized and progresses slowly and painlessly, unless it is complicated by acute infection or trauma.

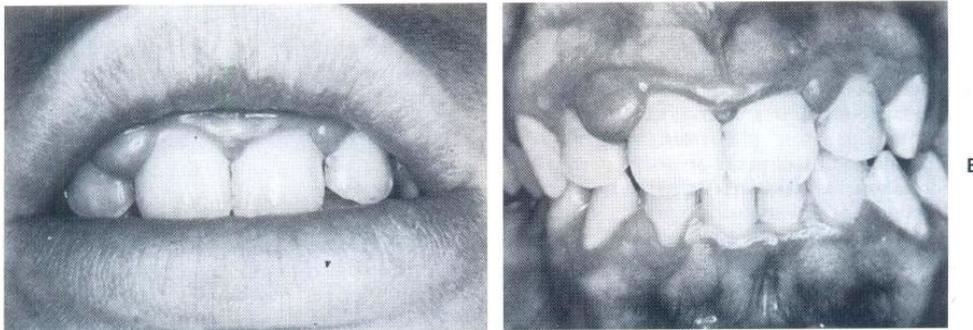
Histopathology: Chronic inflammatory gingival enlargements show the exudative and proliferative features of chronic inflammation.

Lesions that are clinically deep red or bluish red are soft and friable with a smooth, shiny surface, and they bleed easily. They also have a preponderance of inflammatory cells and fluid with vascular engorgement, new capillary formation, and associated degenerative changes.

Etiology: Chronic inflammatory gingival enlargement is caused by prolonged exposure to dental plaque. Factors that favor plaque accumulation and retentions include poor oral hygiene as well as irritation by anatomic abnormalities and improper restorative and orthodontic appliances.

Gingival Changes Associated with Mouth Breathing: Gingivitis and gingival enlargement are often seen in mouth breathers. The gingiva appears red and edematous with a diffuse surface shininess of the exposed area. The maxillary anterior region is the common site of such involvement. In many cases the altered gingiva is clearly demarcated from the adjacent unexposed normal gingiva.

Its harmful effect is generally attributed to irritation from surface dehydration.



Acute Inflammatory Enlargement

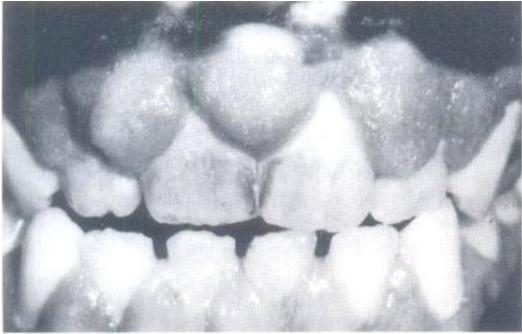
Gingival Abscess: A gingival abscess is a localized, painful, rapidly expanding lesion that is usually of sudden onset. It is generally limited to the marginal gingiva or interdental papilla. In its early stages it appears as a red swelling with a smooth, shiny surface. Within 24 to 48 hours, the lesion usually becomes fluctuant and pointed with a surface orifice from which a purulent exudate may be expressed. The adjacent teeth are often sensitive to percussion. If permitted to progress, the lesion generally ruptures spontaneously.

ETIOLOGY: Acute inflammatory gingival enlargement results from bacteria carried deep into the tissues when a foreign substance such as a toothbrush bristle, a piece of apple core, or a lobster shell fragment is forcefully embedded

into the gingiva. The lesion is confined to the gingiva and should not be confused with periodontal or lateral abscesses.

DRUG-INDUCED GINGIVAL ENLARGEMENT:

Gingival enlargement is a well-known consequence of the administration of some anticonvulsants, immunosuppressants, and calcium channel blockers and may create speech, mastication, tooth eruption, and aesthetic problems.



GENERAL INFORMATION

Clinical Features: The growth starts as a painless, beadlike enlargement of the interdental papilla and extends to the facial and lingual gingival margins. As the condition progresses, the marginal and papillary enlargements unite; they may develop into a massive tissue fold covering a considerable portion of the crowns, and they may interfere with occlusion.

The enlargement is usually generalized throughout the mouth but is more severe in the maxillary and mandibular anterior regions.

A genetic predisposition is a suspected factor-10,⁹⁶ in determining whether a person treated with phenytoin will develop gingival enlargement or not.

The enlargement is chronic and slowly increases in size. When surgically removed, it recurs. Spontaneous disappearance occurs within a few months after discontinuation of the drug.

Drug-induced enlargement may occur in mouths with little or no plaque and may be absent in mouths with abundant deposits. However, the presence of the enlargement makes plaque control difficult, often resulting in a secondary inflammatory process that complicates the gingival overgrowth caused by the drug. The resultant

Dr. Prerna Agarwal

enlargement becomes then a combination of the increase in size caused by the drug and the complicating inflammation, caused by bacteria.

Anticonvulsants

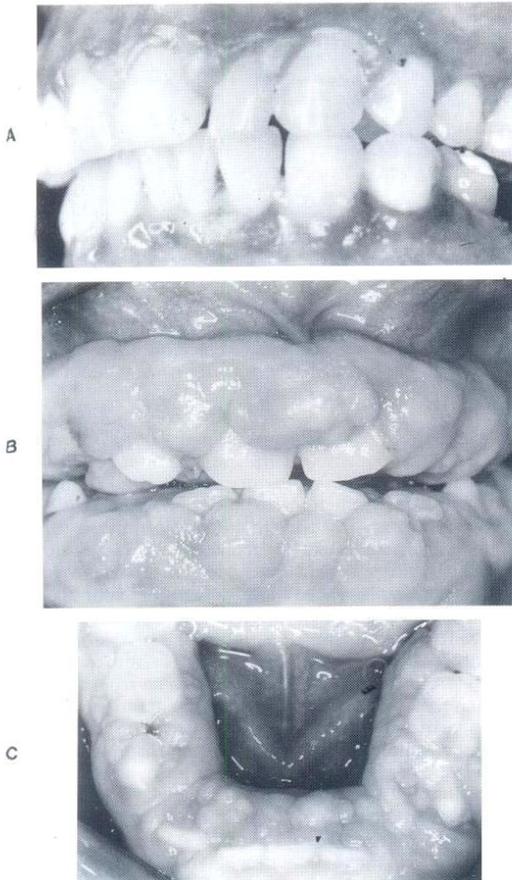
The first drug-induced gingival enlargements reported were those produced by phenytoin.

Other hydantoins known to induce gingival enlargement are ethotoin (Paganone), and mephenytoin (Mesantoin). Other anticonvulsants that have the same side effect are the succinimides (ethosuximide [Zerontin], methsuxinimide [Celontin]), and valproic acid (Depakene).

Tissue culture experiments indicate that phenytoin stimulates proliferation of fibroblast-like cells as epithelium. Two analogues of phenytoin (1-allyl-phenylhydantoinate and 5-methyl-5-phenylhydantoinate) have a similar effect on fibroblast-like cells.

Phenytoin may induce a decrease in collagen degradation as a result of the production of an inactive fibroblastic collagenase.

IMMUNOSUPPRESSANT



Cyclosporine is a potent immunosuppressive agent used to prevent organ transplant rejection and to treat several diseases of autoimmune origin.

Cyclosporine A (Sandimmune, Neoral) is administered intravenously or by mouth, and dosages greater than 500 mg/day have been reported to induce gingival overgrowth .

Cyclosporine-induced gingival enlargement is more vascularized than the phenytoin enlargement, occurs in approximately 30% of patients receiving the drug, is more frequent in children, and its magnitude appears to be related more to the plasma concentration than to the patient's periodontal status.

Calcium Channel Blockers

Calcium channel blockers are drugs developed for the treatment of cardiovascular conditions such as hypertension, angina pectoris, coronary artery spasms, and cardiac arrhythmias.* They inhibit calcium ion influx across the cell

membrane of heart and smooth muscle cells, blocking intracellular mobilization of calcium. This induces direct dilation of the coronary arteries and arterioles, improving oxygen supply to the heart muscle; it also reduces hypertension by dilating the peripheral vasculature.

Diltiazem, felodipine, nitrendipine, and verapamil also induce gingival enlargement.

ENLARGEMENT IN PREGNANCY:

During pregnancy there is an increase in levels of both progesterone and estrogen, which, by the end of the third trimester, reach levels 10 and 30 times the levels during the menstrual cycle, respectively.

These hormonal changes induce changes in vascular permeability leading to gingival edema and an increased inflammatory response to dental plaque.

MARGINAL ENLARGEMENT: Marginal gingival enlargement during pregnancy results from the aggravation of previous inflammation, and its incidence has been reported as 10%³ and 70%.

The gingival enlargement does not occur without the presence of bacterial plaque.

Clinical Features: The clinical picture varies considerably. The enlargement is usually generalized and tends to be more prominent interproximally than on the facial and lingual surfaces. The enlarged gingiva is bright red or magenta, soft, and friable and has a smooth, shiny surface. Bleeding occurs spontaneously or on slight provocation.

TUMORLIKE GINGIVAL ENLARGEMENT:

The so-called pregnancy tumor is not a neoplasm; it is an inflammatory response to bacterial plaque and is modified by the patient's condition. It usually appears after the third month of pregnancy but may occur earlier. The reported incidence is 1.8% to 5%."

Dr. Prerna Agarwal

Clinical Features: The lesion appears as a discrete, mushroomlike, flattened spherical mass that protrudes from the gingival margin or more commonly from the interproximal space and is attached by a sessile or pedunculated base.

Dusky red or magenta, it has a smooth, glistening surface that often exhibits numerous deep red, pinpoint markings.

It is usually painless unless its size and shape foster accumulation of debris under its margin or interfere with occlusion, in which case, painful ulceration may occur.

ENLARGEMENT IN PUBERTY: Enlargement of the gingiva is sometimes seen during puberty. It occurs in both male and female adolescents and appears in areas of plaque accumulation.

Clinical Features: The size of the gingival enlargement far exceeds that usually seen in association with comparable local factors. It is marginal and interdental and is characterized by prominent bulbous interproximal papillae.

Often only the facial gingivae are enlarged, and the lingual surfaces are relatively unaltered.

ENLARGEMENT IN VITAMIN C DEFICIENCY:

Acute vitamin C deficiency does not of itself cause gingival inflammation, but it does cause hemorrhage, collagen degeneration, and edema of the gingival connective tissue.

The combined effect of acute vitamin C deficiency and inflammation produces the massive gingival enlargement in scurvy.

Clinical Features: Gingival enlargement in vitamin C deficiency is marginal; the gingiva is bluish red, soft, and friable and has a smooth, shiny surface. Hemorrhage, occurring either spontaneously or on slight provocation, and surface necrosis with pseudomembrane formation are common features.

SYSTEMIC DISEASES CAUSING GINGIVAL ENLARGEMENT:

Several systemic diseases may, by different mechanisms, result in gingival enlargement. These are uncommon cases and will be only briefly discussed.

LEUKEMIA



Clinical Features: Leukemic enlargement may be diffuse or marginal, localized or generalized.

It may appear as a diffuse enlargement of the gingival mucosa (Fig. 18-20), an oversized extension of the marginal gingiva, or a discrete tumorlike interproximal mass.

The gingiva is generally bluish red and has a shiny surface.

GRANULOMATOUS DISEASES

WEGENER'S GRANULOMATOSIS:

Wegener's granulomatosis is a rare disease characterized by acute granulomatous necrotizing lesions of the respiratory tract, including nasal and oral defects.

The initial manifestations of Wegener's granulomatosis may involve the orofacial region and include oral mucosal ulceration, gingival enlargement, abnormal tooth mobility, exfoliation of teeth, and delayed healing response."

Clinical Features: The granulomatous papillary enlargement is reddish purple and bleeds easily on stimulation.

SARCOIDOSIS:

Sarcoidosis is a granulomatous disease of unknown etiology. It starts in individuals in their twenties or thirties, affects predominantly blacks and can involve almost

Dr. Prerna Agarwal

any organ, including the gingiva, where a red, smooth, painless enlargement may appear.

NEOPLASTIC ENLARGEMENT (GINGIVAL TUMORS):

BENIGN TUMORS OF THE GINGIVA

Epulis is a generic term used clinically to designate all discrete tumors and tumorlike masses of the gingiva.

Neoplasms account for a comparatively small proportion of gingival enlargements and make up a small percentage of the total number of oral neoplasms. In a survey of 257 oral tumors approximately 8% occurred on the gingiva.

Carcinoma, 11.0%; fibroma, 9.3%; giant cell tumor, 8.4%; papilloma, 7.3%; leukoplakia, 4.9%; mixed tumor (salivary gland type), 3.5%; angioma, 1.5%; osteofibroma, 1.3%; sarcoma, 0.5%; melanoma, 0.5%; myxoma, 0.45%; fibropapilloma, 0.4%; adenoma, 0.4%; and lipoma, 0.3%.

FIBROMA:

Fibromas of the gingiva arise from the gingival connective tissue or from the periodontal ligament. They are slow-growing, spherical tumors that tend to be firm and nodular but may be soft and vascular. Fibromas are usually pedunculated.

PAPILLOMA:

Papillomas are benign proliferations of surface epithelium associated with the human papillomavirus (HPV).

Viral subtypes HPV-6 and HPV-11 have been found in most cases of oral papillomas.

Dr. Prerna Agarwal

Gingival papillomas appear as solitary, wartlike or "cauliflower"-like protuberances and may be small and discrete or broad, hard elevations with minutely irregular surfaces.

CENTRAL GIANT CELL GRANULOMA:

These lesions arise within the jaws and produce central cavitation. They occasionally create a deformity of the jaw that makes the gingiva appear enlarged.

LEUKOPLAKIA:

Leukoplakia is a strictly clinical term defined by the World Health Organization as a white patch or plaque that does not rub off and cannot be diagnosed as any other disease.

It is associated to the use of tobacco.

Leukoplakia of the gingiva varies in appearance from a grayish white, flattened, scaly lesion to a thick, irregularly shaped keratinous plaque.

Histopathology: Leukoplakia exhibits hyperkeratosis and acanthosis.

Inflammatory involvement of the underlying connective tissue is a commonly associated finding.

GINGIVAL CYST:

Gingival cysts of microscopic proportions are common.

They appear as localized enlargements that may involve the marginal and attached gingiva.

They occur in the mandibular canine and premolar areas, most often on the lingual surface.

They are painless, but with expansion, they may cause erosion of the surface of the alveolar bone.

Which arises within the alveolar bone, adjacent to the root, and is developmental in origin.

MALIGNANT TUMORS OF THE GINGIVA:

CARCINOMA:

Oral cancer accounts for less than 3% of all malignant tumors in the body but is the sixth most common cancer in males and the twelfth in females. The gingiva is not a frequent site of oral malignancy (6% of oral cancers).

Squamous cell carcinoma is the most common malignant tumor of the gingiva. It may be exophytic, presenting as an irregular outgrowth, or ulcerative, which appear as flat, erosive lesions.

It is often symptom free, often going unnoticed until complicated by inflammatory changes that may mask the neoplasm but cause pain; sometimes it becomes evident after tooth extraction. They are locally invasive, involving the underlying bone and periodontal ligament of adjoining teeth and the adjacent mucosa.

FALSE ENLARGEMENT:

False enlargements are not true enlargements of the gingival tissues but may appear as such as a result of increases in size of the underlying osseous or dental tissues.

Underlying Osseous Lesions:

Enlargement of the bone subjacent to the gingival area occurs most commonly in tori and exostoses, but it can also occur in Paget's disease, fibrous dysplasia, cherubism, central giant cell granuloma, ameloblastoma, osteoma, and osteosarcoma.

The gingival tissue can appear normal or may have unrelated inflammatory changes.

Underlying Dental Tissues

During the various stages of eruption, particularly of the primary dentition, the labial gingiva may show a bulbous marginal distortion caused by superimposition of the bulk of the gingiva on the normal prominence of the enamel in the gingival half of the crown.

Dr. Prerna Agarwal

This enlargement has been termed developmental enlargement and often persists until the junctional epithelium has migrated from the enamel to the cementoenamel junction.