

Gingiva In Health And Disease

Third year
Batch 2020-21

Dr. Nilam Brahmhatt
Assistant Professor
Dept. Of Periodontology



Gingiva In Health

Introduction

The oral mucosa consists of three zones:

Masticatory mucosa - gingiva and covering of the hard palate.

Specialized mucosa - dorsum of the tongue.

Lining mucosa - lining the remainder of the oral cavity.

As far as periodontium is concerned consists of

Gingiva -main function is protection of underlying tissues.

Periodontal ligament

Cementum }--forms attachment apparatus

Alveolar bone

Definition

Gingiva: “Gingiva is the part of the oral mucosa that covers the alveolar processes of the jaws and surrounds the neck of the teeth.”

Clinical features

The gingiva is divided anatomically into

1. Marginal
2. Attached
3. Interdental areas.

Marginal Gingiva

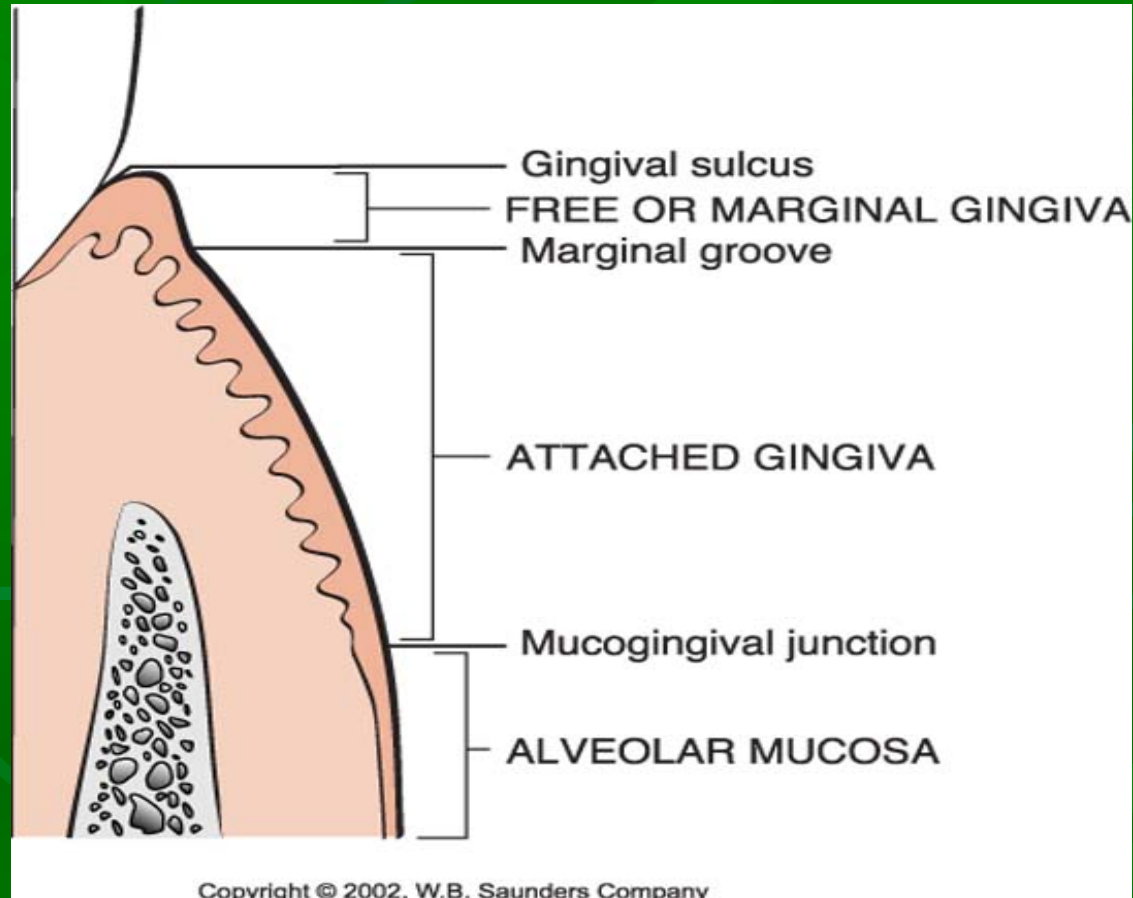
It is unattached gingiva, which is the terminal edge or border of the gingiva surrounding the teeth in collar like fashion.

Free gingival groove

-In about 50% cases, marginal gingiva is demarcated from the adjacent, attached gingiva by a shallow linear depression, runs parallel to & at a distance of 0.5 to 2mm from the margin of gingiva.

Gingival sulcus

“It is shallow crevice or space around the tooth bounded by the surface of the tooth on one side and the epithelium lining the free margin of the gingiva on the other.”



-It is “V” shaped and barely permits the entrance of a periodontal probe.

Histological depth- determined by histological sections

- 1.8mm with variations from 0 to 6mm,

Probing depth

- sulcus depth reading provided by periodontal probe
normal gingival sulcus is 2 to 3 mm.

Attached Gingiva

The attached Gingiva is continuous with the marginal gingiva. It is firm resilient and tightly bound to the underlying periosteum of alveolar bone.

Width of attached gingiva

“It is the distance between the mucogingival junction and the projection on the external surface of the bottom of the gingival sulcus or the periodontal pocket.”

Greatest in the incisor region - 3.5 to 4.5mm in maxilla
3.3 to 3.9mm in mandible

Least in first premolar area - 1.9 mm in maxilla
1.8 mm in mandible

As the mucogingival junction remains stationary throughout adult life, changes in the width of the attached gingiva are caused by modifications in the position of its coronal end.

Interdental gingiva

The interdental gingiva occupies the gingival embrasure, which is the interproximal space beneath the area of the tooth contact.

Shape

Anterior segment

Pyramidal because the tip of the papilla is located immediately beneath the contact point.

Posterior segment

Col shape because it represents the valley like depression that connects a facial & lingual papilla and conforms to the shape of the interproximal contact.

Shape depends on the contact point between the two adjoining teeth and the presence or absence of some degree of recession

Microscopic features

Gingiva consists of a central core of connective tissue covered by stratified squamous epithelium

Gingival epithelium

General aspects of gingival epithelium biology

Three different areas morphologically & functionally

1. Oral or outer epithelium
2. Sulcular epithelium
3. Junctional epithelium

Cell types

Principal cell- *keratinocytes*

Other cells, nonkeratinocytes or clear cells, which include

Langerhans cells

Merkel cells

Melanocytes

I. Keratinocyte

Function - Protecting deep structures.

- Allowing a selective interchange with oral environment,

II. Nonkeratinocyte cells_

Melanocytes

Dendritic cells located in the basal and spinous layer of the gingival epithelium synthesize melanin in organelles called premelanosomes or melanosomes.

Langerhans cells

Dendritic cells located among keratinocytes at all suprabasal levels belong to mononuclear phagocyte system (R.E system).

Merkel cells

located in the deeper layers of the epithelium, harbour nerve endings and have been identified as tactile receptors.

Basal Lamina

The epithelium is joined to the underlying connective tissue by a basal lamina 300-400 Å thick, lying approximately 400 Å beneath the epithelial basal layer.

It consists of

1. Lamina lucida
2. Lamina densa

- The basal lamina is permeable to fluids but acts as a barrier to particulate matter

Structural and metabolic characteristics of the different areas of gingival epithelium

Oral or outer epithelium

Covers crest & outer surfaces of marginal gingiva & surface of attached gingiva .

- Keratinized or parakeratinized or various combinations with prevalence toward parakeratinization.
- Keratinization varies in following order

Palate (Most keratinized)



Gingiva



Ventral aspect of tongue



Cheek (Least keratinized)

Sulcular epithelium

It is a thin, nonkeratinized stratified squamous epithelium without retepegs and extends from coronal limit of the junctional epithelium to the crest of the gingival margin.

It has potential to keratinize if - exposed to oral cavity
- bacterial flora is eliminated.

suggests that local irritation of sulcus prevents sulcular keratinization.

Importance

It may act as a semi permeable membrane through which injurious bacterial products pass into the gingiva and tissue fluid from gingiva seeps into the sulcus.

Junctional epithelium

The junctional epithelium consists of a collar like band of stratified squamous non-keratinized epithelium.

- It is 3-4 layers thick in early life, the number of layers increase with age to 10-20. The cells are grouped in two strata basal and suprabasal
- The length ranges from 0.25 to 1.35mm.
- It attaches to tooth surface by means of an internal basal lamina and to the connective tissue by an external basal lamina.
- The internal basal lamina consists of a lamina densa and lamina lucida to which hemidesmosomes are attached.
- The attachment of the junctional epithelium to the tooth is reinforced by the gingival fibers, which brace the marginal gingiva against the tooth surface.

Therefore the junctional epithelium and gingival fibers are considered a functional unit called as dentogingival unit.

Gingival Fluid (Sulcular Fluid)

The gingival sulcus contains a fluid that seeps into it from the gingival connective tissue through the thin sulcular epithelium.

The gingival fluid is believed to

- Cleanse material from the sulcus.
- Contain plasma proteins that may improve adhesion of the epithelium to the tooth.
- Possess antimicrobial properties.
- Exert antibody activity to defend the gingiva.

Gingival connective tissue

Known as the lamina propria

Consists of

Papillary layer subjacent to the epithelium.

- consists of papillary projections between the epithelial rete pegs.

Reticular layer contiguous with the periosteum of the alveolar bone

Connective tissue consists of

1. Cellular elements
2. Extra cellular compartment

1. Cellular elements

- Preponderant cell is fibroblast, synthesizes & degrade fibers.
- Glycoproteins & glycosaminoglycan is also formed by fibroblasts.
- Other cells
 1. Mast cells
 2. Fixed macrophages & histiocytes.
 3. Adipose cells
 4. Eosinophils
 5. Plasma cells
 6. Lymphocytes
 7. Neutrophils

2. Extracellular compartment

Fibers

Three types collagen, reticular & elastic.

- Collagen type-I forms bulk of lamina propria & provides tensile strength to gingival tissue.
- Collagen type IV-branches between collagen type-I bundles & is continuous with fibers of the basement membrane and blood vessel walls.
- The elastic fiber system is composed of oxytalan, elaunin, and elastin fibers distributed among collagen fibers.

Gingival Fibers

The connective tissue of the marginal gingiva is densely collagenous containing a prominent system of collagen fiber bundles called the gingival fibers.

They consist of type I collagen

Functions of gingival fibers

- 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.

Three groups of fibers

Gingivodental group

On the facial, lingual and interproximal surface embedded in the cementum just beneath the epithelium at the base of the gingival sulcus.

They extend externally to the periosteum of the facial and lingual alveolar bones and terminate in the attached gingiva or blend with the periosteum of the bone

Circular group

Course through the connective tissue of the marginal and interdental gingiva and encircle the tooth in ring like fashion.

Transseptal group

Form horizontal bundles that extend between the cementum of approximating teeth into which they are embedded.

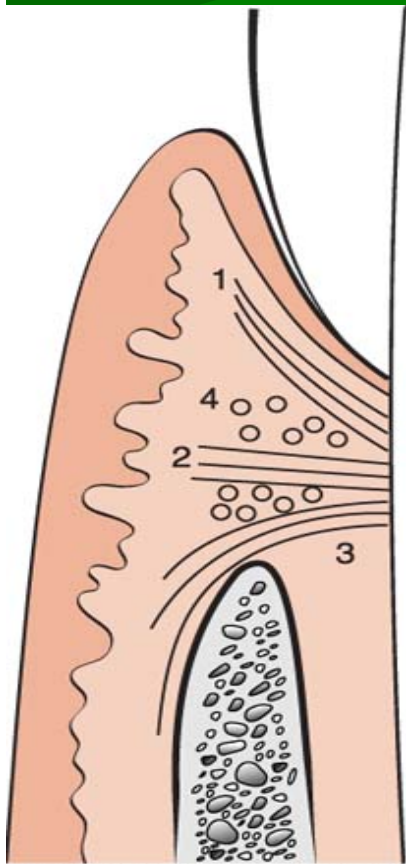


Diagram of the gingivodental fibers extending from the cementum

- (1) to the crest of the gingival
- (2) to the outer surface
- (3) external to the periosteum of the labial plate
- (4) circular fibers are shown in cross-section

Blood supply

Three sources of blood supply to the gingiva are as follows

1. *Supraperiosteal arterioles* along the facial and lingual surfaces of the alveolar bone, from which capillaries extend along the sulcular epithelium and between the retepegs of the external gingival surface.
2. *Vessels of periodontal ligament*, which extend into the gingiva and anastomose with capillaries in the sulcus area.
- 3 *Arterioles* which emerge *from the crest of the interdental septa* and extend parallel to the crest of the bone.

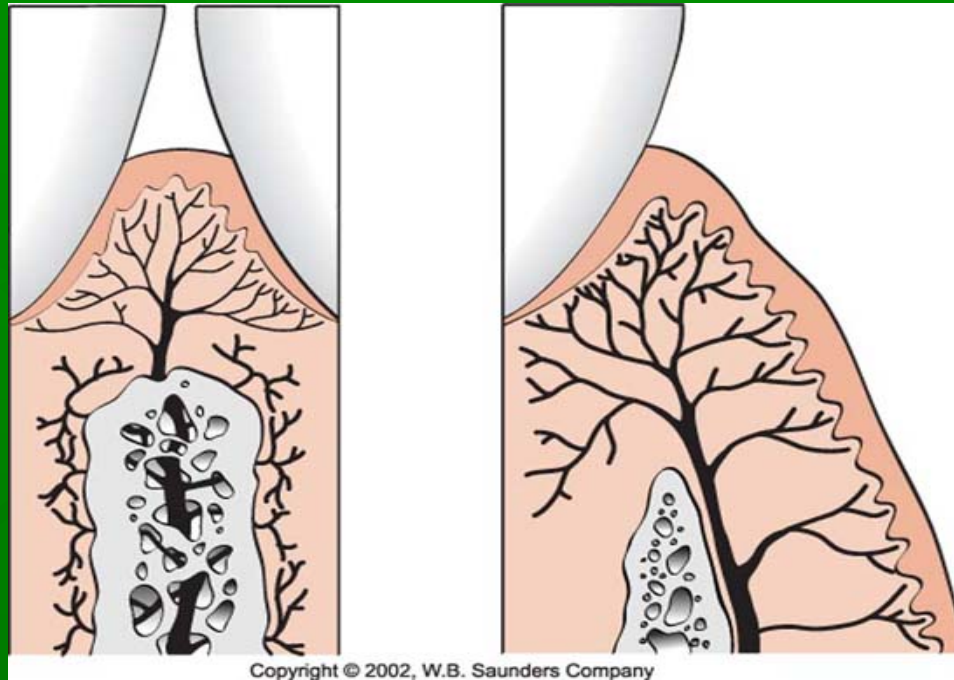


Diagram of arteriole penetrating the interdental alveolar bone to supply the interdental tissue (left)

Suprapariosteal arteriole overlying the facial alveolar bone sending branches to the surrounding tissue (right)

Lymphatic drainage

From connective tissue papillae into the collecting network external to the periosteum and then to regional lymph nodes.

Lymphatics beneath the junctional epithelium extend into the periodontal ligament and accompany the blood vessels.

Gingival innervation

It is derived from fibers arising from nerves in the *periodontal ligament* and from the *labial, buccal* and *palatal nerves*

The following nerve structures are present in the connective tissue.

Meissner type- tactile corpuscles

Krause-type end bulbs- temperature receptors

Encapsulated spindles.

Correlation of clinical and microscopic features of gingiva

An understanding of normal clinical features of the gingiva requires the ability to interpret them in terms of the microscopic structures they represent.

Color-Coral pink

varies among different persons and appears to be correlated with the cutaneous pigmentation, lighter in blond individuals with fair complexions than in swarthy, dark-haired individuals.

- It is produced by -Vascular supply.
 - Thickness & degree of keratinization.
 - Presence of pigment containing cells.

Physiologic Pigmentation (Melanin)

It is prominent in black individuals

Occurs as a diffuse deep-purplish discolouration or as irregularly shaped brown and light brown patches.

Size

- Sum total of bulk of cellular & intercellular elements and their vascular supply.
- Alteration in size is common feature of gingival disease

Contour

It depends on the

- Shape of the teeth and their alignment in the arch.
- Location and size of the area of proximal contact
- Dimensions of the facial and lingual gingival embrasures

The marginal gingiva follows a scalloped outline on the facial and lingual surfaces.

- Forms a straight line on teeth with relatively flat surfaces
- On teeth with pronounced mesiodistal convexity e.g. maxillary canines or teeth in labial version the normal arcuate contour is accentuated and the gingiva is located farther apically.
- On teeth in lingual version the gingiva is horizontal and thickened.

Shape

- The shape of the interdental gingiva is governed by the contour of the proximal tooth surfaces and the location and shape of gingival embrasures.
- Flat proximal surfaces , interdental gingiva is narrow mesiodistally.
- Proximal surfaces that flare away from the area of contact the mesiodistal diameter of the interdental gingiva is broad.
- The height of interdental gingiva varies with location of proximal contact.

Consistency

- Firm and resilient and with exception of the movable free margin, tightly bound to the underlying bone.
- The gingival fibers contribute to the firmness of gingival margin

Surface texture

- Gingiva presents a textured surface similar to orange peel referred to as being stippled best viewed by drying the gingiva.

- The attached gingiva is stippled, the marginal gingiva is not. The central portion of the interdental papilla is usually stippled but the marginal borders are usually smooth
- Stippling varies among individuals and different areas of same mouth, less prominent on lingual than facial surfaces and may be absent in some persons, also varies with age.
- *Microscopically*, produced by alternate rounded protuberances & depressions in the gingival surface. The papillary layer of connective tissue projects into the elevations covered by stratified squamous epithelium. The degree of keratinization and the prominence of stippling appear to be related.
- ● Stippling is a form of adaptive specialization or reinforcement for function, increases when the gingiva is stimulated by toothbrushing.
- It is a feature of healthy gingiva and reduction or loss is a sign of gingival disease.

Position

- The position of the gingival refers to the level at which the gingival margin is attached to the tooth.
- Marginal gingiva is 1 mm above the cemento enamel junction (CEJ)and junctional epithelium is at the level of CEJ.

The background is a solid green color with a faint, repeating pattern of stylized leaves and stems in a slightly darker shade of green. The leaves are simple, pointed shapes with visible veins.

Gingiva In Disease

Gingiva In Disease

Dental plaque induced gingival diseases

I Gingivitis associated with dental plaque only

- A. Without local contributing factors
- B. With local contributing factors

II 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
- B. Associated with blood dyscrasias
 - 1. Leukemia associated gingivitis
 - 2. Other

III 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. Other

IV Gingival diseases modified by malnutrition

- A. Ascorbic acid deficiency gingivitis.**
- B. Other.**

Non plaque induced gingival lesions

I. Gingival diseases of specific bacterial origin

- A. Neisseria Gonorrhoea**
- B. Treponema Pallidum**
- C. Streptococcal species**
- D. Other.**

II. Gingival diseases of viral origin

A. Herpes virus infections

1. Primary herpetic gingivostomatitis.
2. Recurrent oral herpes.
3. Varicella zoster

B. Other.

III. Gingival diseases of fungal origin

A. Candida species infections

-Generalized gingival candidiasis

B. Linear gingival erythema

C. Histoplasmosis

D. Other

IV. Gingival lesions of genetic origin

A. Hereditary gingival fibromatosis

B. Other

V. Gingival manifestations of systemic conditions.

A. Mucocutaneous lesions

1. Lichen planus
2. Pemphigoid
3. Pemphigus vulgaris
4. Erythema multiforme
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. Toothpaste or dentifrices
 - b. Mouth rinses or mouthwashes
 - c. Chewing gum additives
3. Other

VI. Traumatic lesions (Factitious iatrogenic or accidental)

- A. Chemical injury
- B. Physical injury
- C. Thermal injury

VII. Foreign body reactions

VIII. Not otherwise specified.

Dental plaque induced gingival diseases

Most common form of gingival diseases .Inflammation confined to gingiva without attachment loss.



Disclosed supragingival plaque covering one half to two thirds of the clinical crowns.



48-hour plaque growth. Generalized gingivitis at the margins of almost all teeth

I. *Gingivitis associated with dental plaque only*

A. Without local contributing factors

- As a result of an interaction between the micro-organism and the tissues & inflammatory cells of host.

B. With local contributing factor

Plaque host interaction can be altered by the effects of local factors

1. Calculus

-Calculus plays an important role in maintaining plaque in close contact with the gingival tissue & creating areas where plaque removal is impossible.



Supragingival calculus in a patient with gingival inflammation.

2. *Tooth anatomic factors*

i. Malformation of tooth development

- Enamel projections & enamel pearls -specifically in molar furcation area.
- Palatogingival groove
- Proximal root grooves
- maxillary incisors.
- incisors & maxillary premolars.

ii. *Tooth location*

- Tooth malalignment predisposes plaque accumulation & inflammation
- Open contact have been associated increased loss of alveolar bone ,most probably through food impaction.

3. *Iatrogenic factors*

Inadequate dental procedures contribute to deterioration of the periodontal tissues.

It includes -Margins of restoration.

-Contours/open contacts.

-Materials.

-Design of RPD

-Restorative procedures.

4. *Root fractures*

Causes periodontal involvement through an apical migration of plaque along fracture line.

5. Mucogingival deformities & conditions around teeth

A significant departure from the normal shape of gingiva & alveolar mucosa. It requires corrective surgery to restore form & function.

6. Occlusal trauma

Trauma from occlusion refers to the tissue injury not the occlusal force

Gingivitis

It is characterized by the presence of clinical signs of inflammation that are confined to the gingiva and associated with teeth showing no attachment loss.

- .

COURSE AND DURATION

Acute gingivitis is of sudden onset and short duration and can be painful.

A less severe phase of the acute condition has been termed subacute.

Recurrent gingivitis reappears after having been eliminated by treatment or disappearing spontaneously.

Chronic gingivitis is slow in onset and of long duration and is painless, unless complicated by acute or subacute exacerbations, most common.

DISTRIBUTION

Localized gingivitis- confined to the gingiva of a single tooth or group of teeth.

Generalized gingivitis- involves gingiva around all teeth.

Marginal gingivitis- involves gingival margin and may include a portion of the contiguous attached gingiva.

Papillary gingivitis involves the interdental papillae and often extends into the adjacent portion of the gingival margin.

- Papillae are involved more frequently than the gingival margin, and the earliest signs of gingivitis often occur in the papillae.

Diffuse gingivitis affects the gingival margin, the attached gingiva, and interdental papillae.



Chronic marginal gingivitis.



Generalized marginal inflammatory lesion.



Copyright © 2002, W.B. Saunders Company

Localized diffuse gingivitis



Copyright © 2002, W.B. Saunders Company

Generalized diffuse inflammatory lesion

Clinical findings

Gingival bleeding on probing

Two earliest symptoms of gingival inflammation.

1. Increased gingival crevicular fluid production rate.

2. Bleeding from the gingival sulcus on gentle probing

Bleeding on probing is easily detectable, appears earlier than a color change & more objective sign than color change

Chronic and Recurrent bleeding

Most common cause – chronic inflammation, followed by trauma.

Acute bleeding

- Can be due to laceration of gingiva by tooth brush, hard food, etc.
- Spontaneous bleeding-ANUG



Bleeding appears about 30 seconds after probing.

Colour changes in gingiva

Colour changes in chronic gingivitis

Red or bluish red colour, because of

- vascular proliferation
- reduction of keratinization due to epithelial compression by the inflamed tissue.
- Venous stasis will contribute a bluish hue.

Colour changes in acute gingivitis

The changes may be marginal, diffuse or patch like.

- In ANUG-marginal gingiva
- In herpetic gingivostomatitis- diffuse
- Acute reactions to chemical irritation- patch like or diffuse

The grey discoloration produced by tissue necrosis is demarcated from the adjacent gingiva by a thin, sharply defined erythematous zone.

Changes in consistency

In chronic cases consistency is determined by predominance of destructive edematous or reparative fibrotic changes.

Clinical changes

- Soggy puffiness that pits on pressure
- Marked softness & friability with ready fragmentation on exploration with probe and pinpoint surface areas of redness & desquamation
- Firm leathery consistency

Microscopic features

- Infiltration by fluid & cells of inflammatory exudates
- Degeneration of connective tissue & epithelium ,changes in connective tissue epithelium relationship, thinning of epithelium and degeneration associated with edema & leukocytic invasion separated by areas in which retepegs are elongated.
- Fibrosis & epithelial proliferation due to long standing chronic inflammation.



Edematous gingival inflammation: gingiva is red, smooth bright.



Fibrotic gingival inflammation: gingiva is firm and pink with marginal and papillary inflammation.

Changes in surface texture

- Loss of surface stippling is an early sign of gingivitis.
- In chronic inflammation the surface is either smooth and shiny or firm and nodular, depending on whether the dominant changes are exudative or fibrotic.

Changes in gingival contour

Mostly associated with gingival enlargement but such changes may also occur in other conditions e.g. Stillman's cleft
Mc Call's festoons.

Changes in the position of the gingiva

Recession is exposure of the root surface by an apical shift in the position of the gingiva.

Actual position is the level of the epithelial attachment on the tooth.

Apparent position is the level of crest of the gingival margin.

Severity of recession is determined by the actual position of the gingiva.

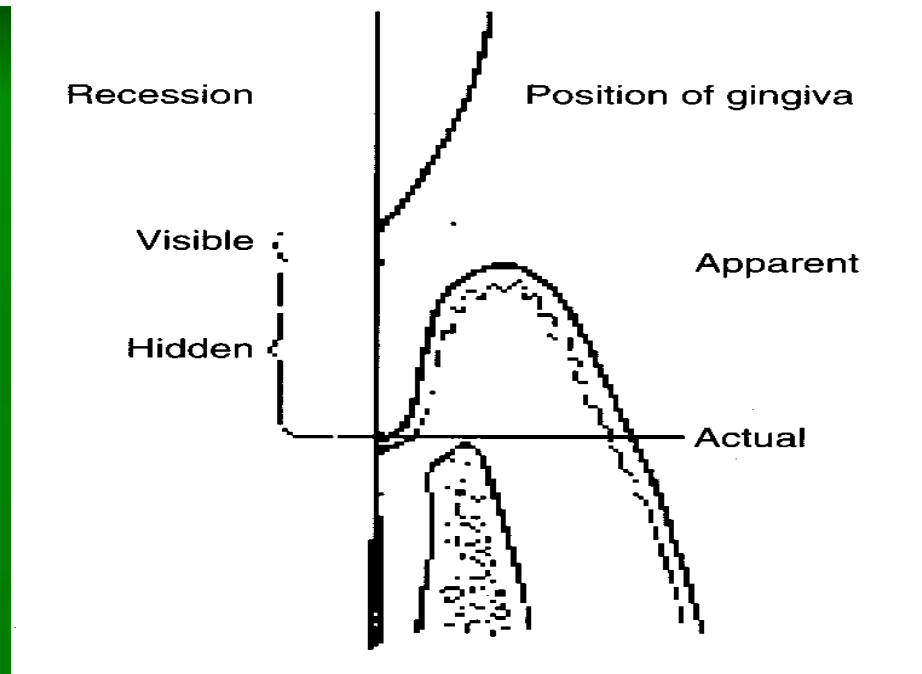


Diagram illustrating the apparent and actual positions of the gingiva and visible and hidden recession

II. *Gingival diseases modified by systemic factors*

A. *Associated with endocrine system*

1. Puberty associated gingivitis

-A higher prevalence & severity of gingivitis and gingival enlargements found in the circumpubertal period termed pubertal gingivitis.

Clinical features

-Increased in bleeding interdental sites .

-May induce gingival enlargement as a result of hormonal changes that magnify the tissue response to dental plaque .

After puberty the enlargement undergoes spontaneous reduction but does not disappear until plaque & calculus are removed .

2. Menstrual cycle associated gingivitis

-The exudate from inflamed gingiva is increased during menstruation, suggesting that existing gingivitis is aggravated by menstruation due to hormonal changes.

3. Pregnancy associated

a. Gingivitis

-Severity of gingivitis is increased during pregnancy beginning in the second or third month, becomes more severe by 8th month & decreases during the ninth.

-Partial reduction in severity of gingivitis occurs by 2 months postpartum & after 1 year the condition is comparable to normal. However gingiva does not return to normal as long as local factors are present.

b. Pyogenic granuloma

-It is an inflammatory response to bacterial plaque modified by the patient's condition, usually appears after the third month but may occur earlier.

Clinical features

-A discrete mushroom like, flattened spherical sessile or pedunculated mass that protrudes from the gingival margin or interproximal space.



Pregnancy gingival enlargement

- Expands laterally and the pressure from cheek & tongue perpetuates its flattened appearance.
- Dusky red or magenta, smooth, glistening surface, ordinarily not involving underlying bone.
- Consistency usually semi firm may be soft and friable.
- Usually painless unless its size and shape foster accumulation of debris under its margin or interfere with occlusion.

4. Diabetes mellitus associated gingivitis

-Diabetes alters flora of oral cavity, uncontrolled diabetes reduces body defense and increased susceptibility to infection leads to periodontal disease.

B. Associated with blood dyscrasias

1. Leukemia associated gingivitis

- - Patients with leukemia may have a simple chronic inflammation but usually shows gingival enlargement & increased bleeding tendency.
 - Acute painful necrotizing inflammatory involvement sometimes occurs in the crevice formed at the junction of enlarged gingiva & tooth structure.

2. Others.

-Agranulocytosis

Reduction in WBC count or granulocytes causes increased susceptibility to infection.



Leukemic gingival enlargement (Acute myelocytic leukemia).

III. *Gingival diseases modified by medications*

- A. *Drug influenced gingival diseases*

1. Drug induced gingival enlargement

-Caused by some anticonvulsants, immunosuppressant, calcium channel blockers.



Phenytoin gingival enlargement.



Cyclosporine gingival enlargement.

2. Drug influenced gingivitis

a. Oral contraceptive associated gingivitis

-Hormonal contraceptive aggravate the gingival response to local factors in a similar manner to pregnancy.

b. Others

-Corticosteroid

Systemic administration of cortisone and ACTH appears to have no effect on severity of gingival & periodontal disease. However renal transplant patients receiving same has less gingival inflammation.

IV. *Gingival diseases modified by malnutrition*

A. *Ascorbic acid deficiency gingivitis*

Gingivitis-common to all ages with enlarged, hemorrhagic, bluish red gingiva.

-Vit-c deficiency may aggravate gingival response to plaque and worsen edema ,enlargement & bleeding.

-Vit-c deficiency correction reduces severity, gingivitis will remain as long as local factors are present.

B. Others

B-complex deficiency

The gingivitis in B-complex deficiency is nonspecific, caused by plaque modified by deficiency. However, in niacin deficiency gingiva may be involved most commonly shows necrotizing ulcerative gingivitis.

Non-plaque induced gingival lesions

I. Gingival diseases of specific bacterial origin

A. Neisseria Gonorrhoea

-Causes primarily a venereal disease affecting genitourinary tract .

Oral manifestation

-Includes lip, tongue, buccal mucosa & palate.

-Gingiva may become erythematous with or without necrosis.

B. Treponema pallidum

-Causes syphilis, three stages-Primary, Secondary & Tertiary.

-In secondary stage gingiva shows characteristic mucous patches.

C. Streptococcal species

-Rare condition characterized by a diffuse erythema of the gingiva and other areas of oral mucosa called as streptococcal gingivostomatitis. Some cases shows marginal erythema with marginal hemorrhages.

-Bacterial smear shows group A beta hemolytic streptococcus.

D. Acute necrotizing ulcerative gingivitis (ANUG)

It is an inflammatory destructive disease of the gingiva, which presents characteristic signs and symptoms.



Acute necrotizing ulcerative gingivitis.

II. *Gingival diseases of viral origin*

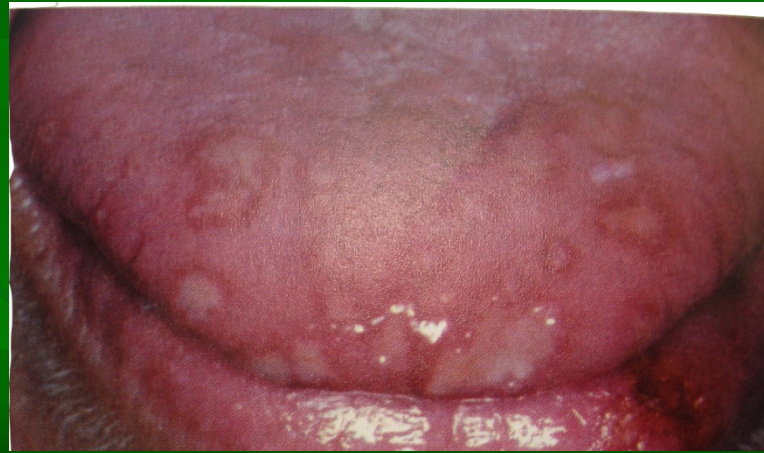
Herpes virus infection

1. Primary herpetic gingivostomatitis

- Caused by HSV type-1
- Most often in infants and children younger than 6yrs. of age.
In most patients primary infection is asymptomatic.
- After the primary infection, the virus ascends through sensory & autonomic nerves and persists in neuronal ganglia.



Acute herpetic gingivostomatitis



III. Gingival diseases of fungal origin

A. Candida species infections

Generalized gingival candidosis

-Caused by yeast like fungus *Candida albicans*, usually occurs as superinfection after use of antibiotics or immunosuppressive drugs.

Oral lesions in acute pseudomembranous candidiasis.

-Soft white slightly elevated plaque on buccal mucosa, tongue also on palate gingiva & floor of mouth, consists of fungal hyphae epithelium, keratin, fibrin, necrotic debris, WBC and bacteria.

B. Histoplasmosis

-Caused by *Histoplasma capsulatum* characterized by low grade fever, cough splenomegaly, hepatomegaly & lymphadenopathy, predilection for reticuloendothelial system.

Oral manifestation

-Nodular, ulcerative or vegetative lesions on the buccal mucosa gingiva, tongue, palate or lips covered by nonspecific gray membrane.

IV. Gingival lesions of genetic origin

Hereditary gingival fibromatosis

Etiology

- Cause is unknown, idiopathic, hereditary, may be autosomal recessive or autosomal dominant.
- Usually begins with the eruption of primary or secondary dentition & may regress after extraction.

Clinical features

- Affects attached gingiva, gingival margin & interdental papillae, facial and lingual surfaces of both the jaws are affected but may be limited to either jaw.
- The enlarged gingiva is pink, firm, and lathery in consistency with minutely pebbled surface.
- In severe cases teeth are almost completely covered & the enlargement projects into the oral vestibule.
- Secondary inflammatory changes are common at the gingival margin.

V. Gingival manifestation of systemic conditions

A. Mucocutaneous lesions

1. Lichen planus
2. Pemphigoid
3. Pemphigus vulgaris
4. Erythema multiforme
5. Lupus erythematosus
6. Drug induced
7. Other.



Copyright © 2002, W.B. Saunders Company

Desquamative lesions. Lichen planus.



Desquamative lesions. Mucous membrane pemphigoid.



Desquamative lesions. Pemphigus

6. Drug induced

An increased sensitivity to drugs noted to sulfonamides, barbiturates & other antibiotics.

- Eruptions in the oral cavity from drug sensitivity are termed *stomatitis medicamentosa* when taken by mouth or parenterally.
- Local application of medicament in the oral cavity causes local reaction called *stomatitis venenata* or *contact stomatitis*.

- Erosion, deep ulceration with pruritic lesions seen in different areas with the gingiva often affected. Desquamative gingivitis has been reported with use of tarter control toothpaste, includes intense erythema of attached gingiva.
- Elimination of offending agent leads to restoration of the gingival lesions within a week.

B. Allergic reactions

1. Dental restorative materials

- a. Mercury
- b. Acrylic
- c. Others

-A true allergy is rare, mostly due to monomer of acrylic causes generalized inflammation or denture sore mouth.

-

-Same reaction can occur to Co-Cr alloy FPD due to presence of Nickel as content.

2. Reaction attributable to

- a. Tooth paste or dentifrices
- b. Mouth rinses or mouthwashes.
- c. Chewing gum additives
- d. Foods & additives

VI. Traumatic lesions

A. Chemical injury

-Aspirin, Sodium perborate, Phenol, & Silver nitrate, when applied locally harmful causes burning sensation within few mins. Surface blanching produced by Aspirin, others causing reddened inflamed areas.

B. Physical injury

-May be factitial as in the case of toothbrush trauma resulting in gingival ulceration, recession or both.

-Iatrogenic as in case of preventive or restorative care that may lead to traumatic injury of the gingiva.

C. Thermal injury

- Can cause damage to gingiva through minor burns from hot foods & drinks.
- Sometimes iatrogenic injury by sealing gutta-percha points can cause thermal injury to gingiva.

VII. Foreign body reactions

-Lead to localized inflammatory conditions of the gingiva and are caused by the introduction of foreign material into gingival connective tissues through breaks in the epithelium.

E.g. introduction of amalgam into the gingiva during the placement of a restoration extraction of a tooth , leaving an amalgam tattoo or the introduction of abrasives during polishing procedures.



Thank You