Periodontal Pocket

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- ***Periodontal pocket*** is defined as a pathologically deepened gingival sulcus.
- Deepening of gingival sulcus may occur by

  1. coronal movement of the gingival margin,

  2. apical displacement of the gingival attachment or

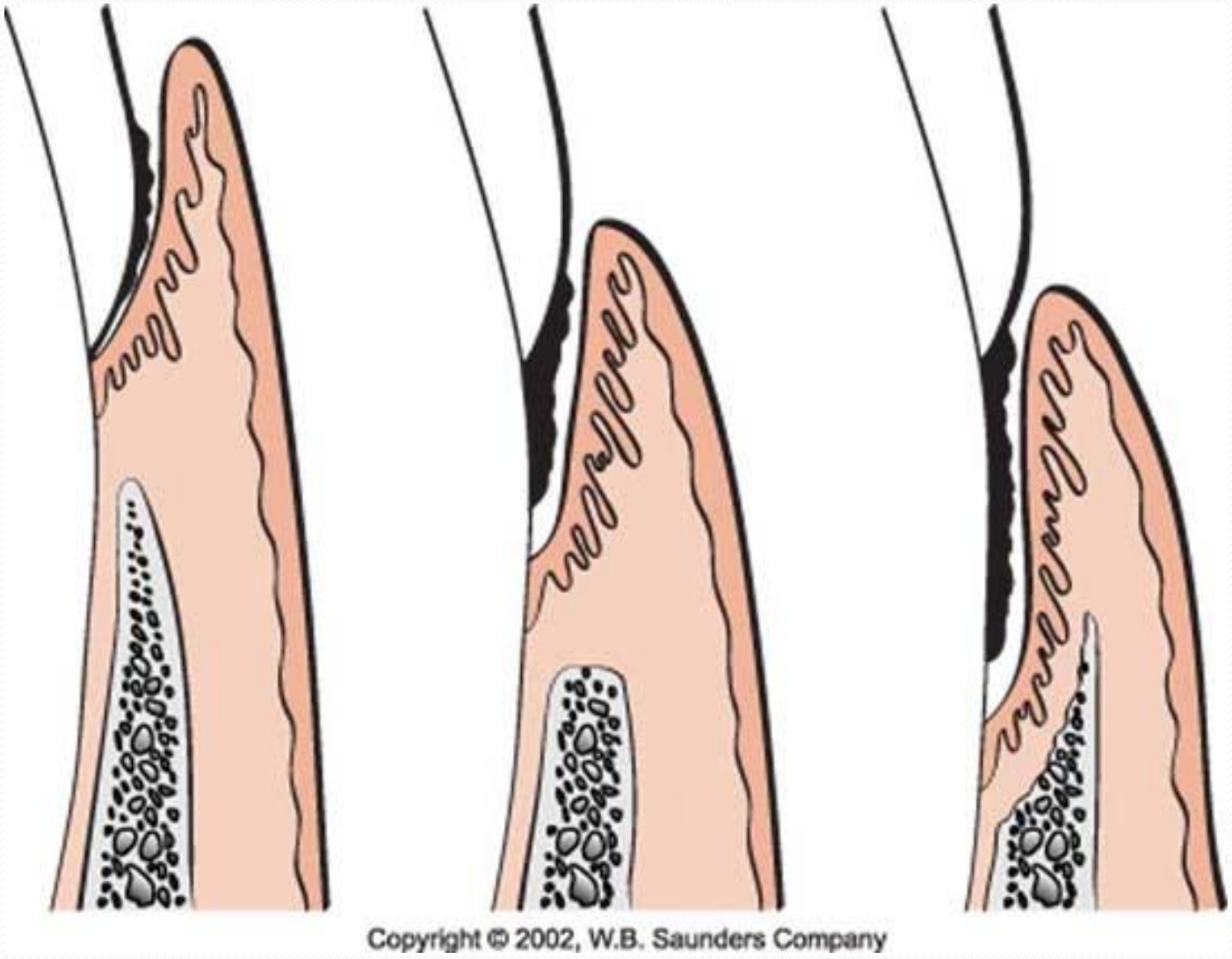
  3. the combination of the two processes.

- ***Gingival pocket (Relative or False)***
- ***Periodontal pocket (Absolute or True)***

CLASSIFICATION OF PERIODONTAL POCKETS

- **I Depending on the relationship of the alveolar crest with the base of pocket**
  - Suprabony pocket (Supracrestal or supra alveolar)
  - Infrabony pocket (Intrabony, subcrestal or intra alveolar)
- **II According to the involved tooth surfaces :**
  - Simple pocket
  - Compound pocket
  - Complex pocket
- **III Depending upon the depth of the pocket**
  - Shallow < 5 mm
  - Deep > 5 mm

Different types of periodontal pockets



Gingival pocket

Suprabony pocket

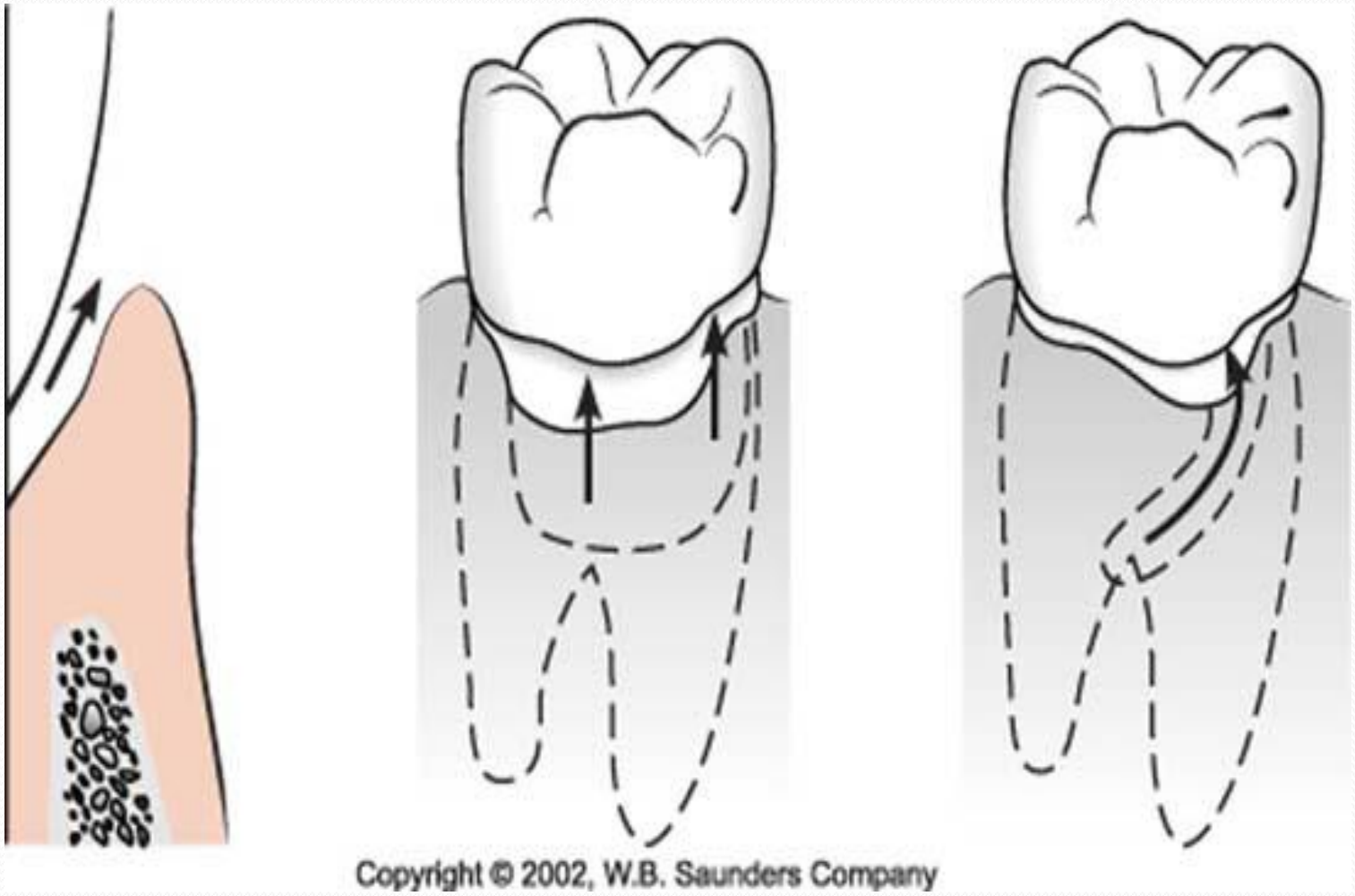
Intrabony pocket

1. Suprabony (Supracrestal or supraalveolar): the bottom of the pocket is coronal to the underlying alveolar bone.
2. Infrabony (intrabony or subcrestal or intraalveolar): the bottom of the pocket is apical to the level of the adjacent alveolar bone.

- In this second type, the lateral pocket wall lies between the tooth surface and alveolar bone.

- Pockets can involve one, two or more tooth surfaces and can be of different depths and types on different surfaces of the same tooth and on approximating surfaces of the same interdental space.
- Pockets can often be spiral (that is originating on one tooth surface and twisting around the tooth to involve one or more additional surfaces).
- These types of pockets are most common in furcation areas.

Classification of pockets according to involved tooth surfaces



Simple pocket

Compound pocket

Complex pocket

Suprabony pocket	Infrabony pocket
The base of the pocket is coronal to the level of the alveolar bone.	The base of the pocket is apical to the crest of the alveolar bone so that the bone is adjacent to the soft tissue wall.
The pattern of bone destruction is horizontal.	The pattern of bone destruction is vertical (angular).

Suprabony pocket	Infrabony pocket
Interproximally, the transseptal fibres that are restored during progressive periodontal disease are arranged horizontally in the space between the base of the pocket and the alveolar bone.	Interproximally, the transseptal fibres are oblique rather than horizontal. They extend from the cementum beneath the base of the pocket along the bone and over the crest of the cementum of the adjacent tooth.

On the facial and lingual surfaces, the PDL fibres beneath the pocket follow their normal horizontal-oblique course between the tooth and the bone.	On the facial and lingual surfaces, the PDL fibres beneath the pocket follow the angular pattern of the adjacent bone. They extend from the cementum beneath the base of the pocket along the bone and over the crest to join with the outer periosteum.

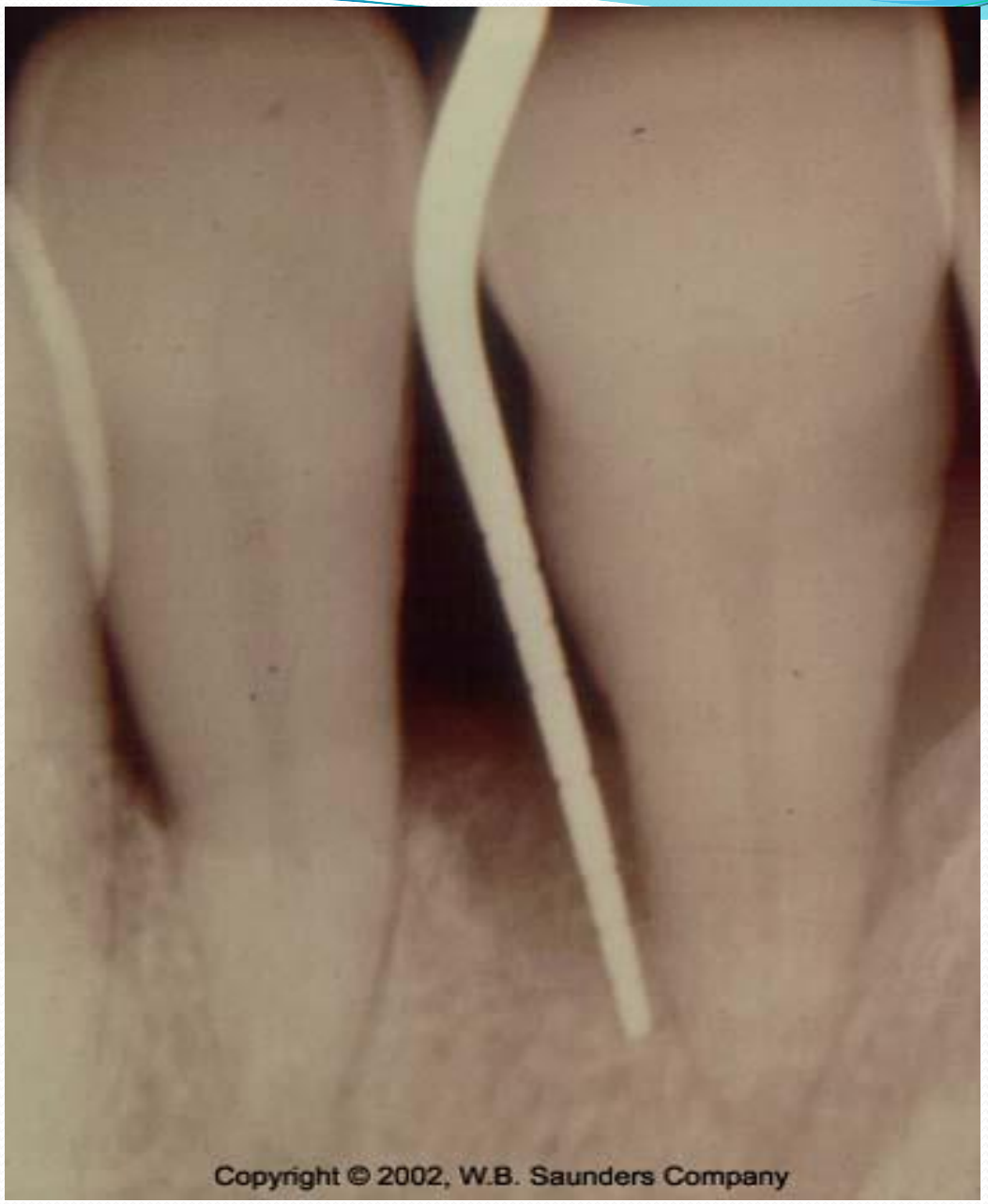
Signs :

1) Enlarged bluish red marginal gingiva with a rolled edge separated from the tooth surface.
2) A reddish blue vertical zone extending from the gingival margin to the attached gingiva and sometimes into the alveolar mucosa.
3) A break in the faciolingual continuity of the interdental gingiva.
4) Shiny, discolored and puffy gingiva.
5) Gingival bleeding.
6) Purulent exudate of the gingival margin.
7) Looseness, extrusion and migration of teeth.
8) Development of diastema where none had existed.

Symptoms :

1) Localized pain or a sensation of pressure after eating which gradually diminishes.
2) Foul taste.
3) Tendency to suck material from interproximal spaces.
4) Radiating pain.
5) Gnawing feeling or feeling of itchiness in the gums.
6) Urge to dig a pointed instruments into the gums with relief obtained from resultant bleeding.
7) Complaints that food sticks between the teeth, teeth feel loose, preference to eat on the other side.
8) Sensitivity to heat & cold, toothache in the absence of caries.

Extent of pocket revealed by periodontal probe



Examination must include consideration of the following

1) The presence and distribution on each tooth surface.
2) Pocket depth and level of attachment on the root.
3) Type of pocket (i.e. suprabony or infrabony, simple, compound or complex).

The only accurate method of detecting and measuring periodontal pockets is careful exploration with a periodontal probe. Pockets are not detected by radiographic examination.

Clinical / Probing depth :

Distance to which the periodontal probe enters into the pocket.

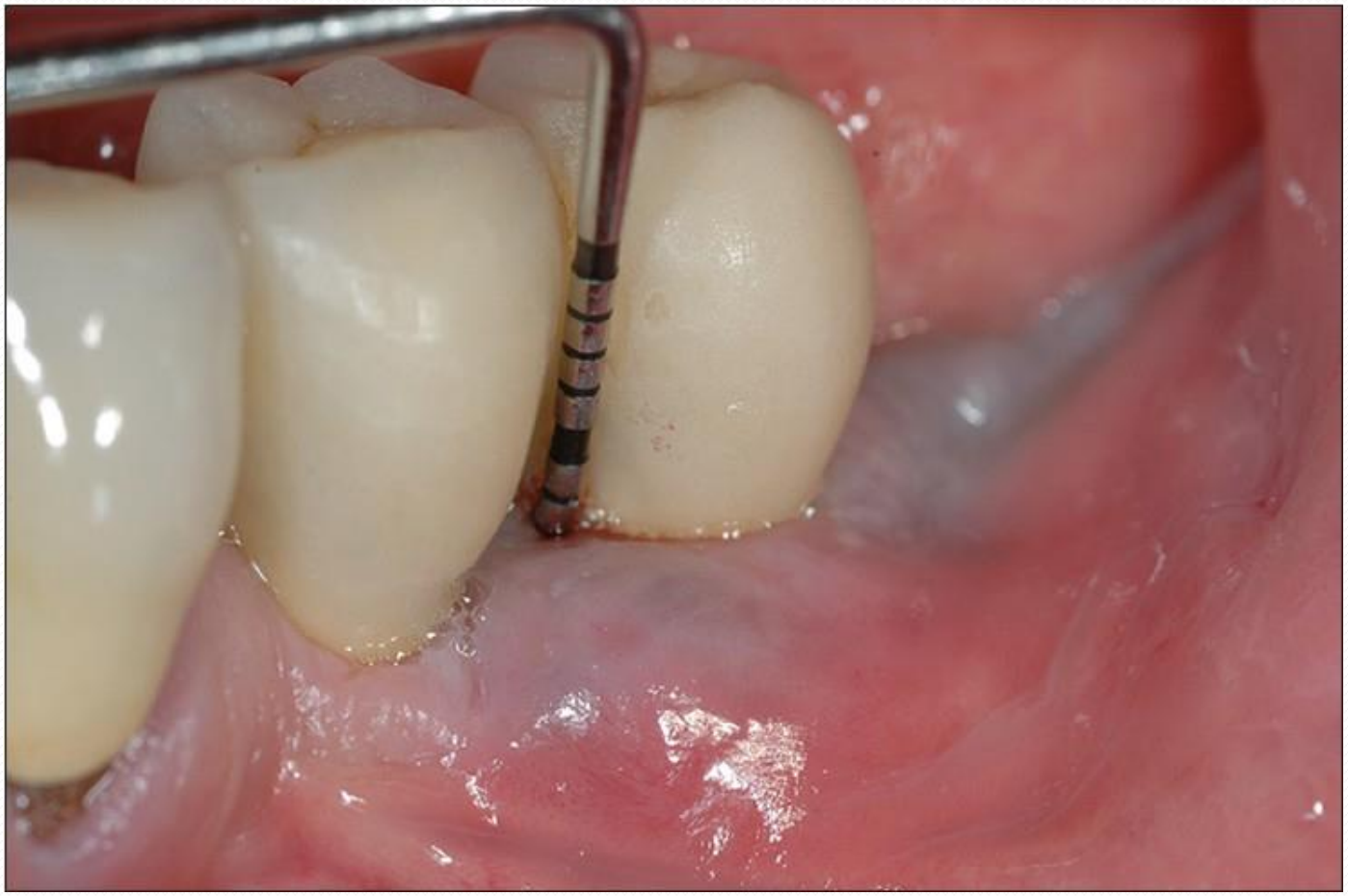
Level of attachment at the base of the pocket is of greater diagnostic significance than the depth of the pocket.

Bleeding on probing :

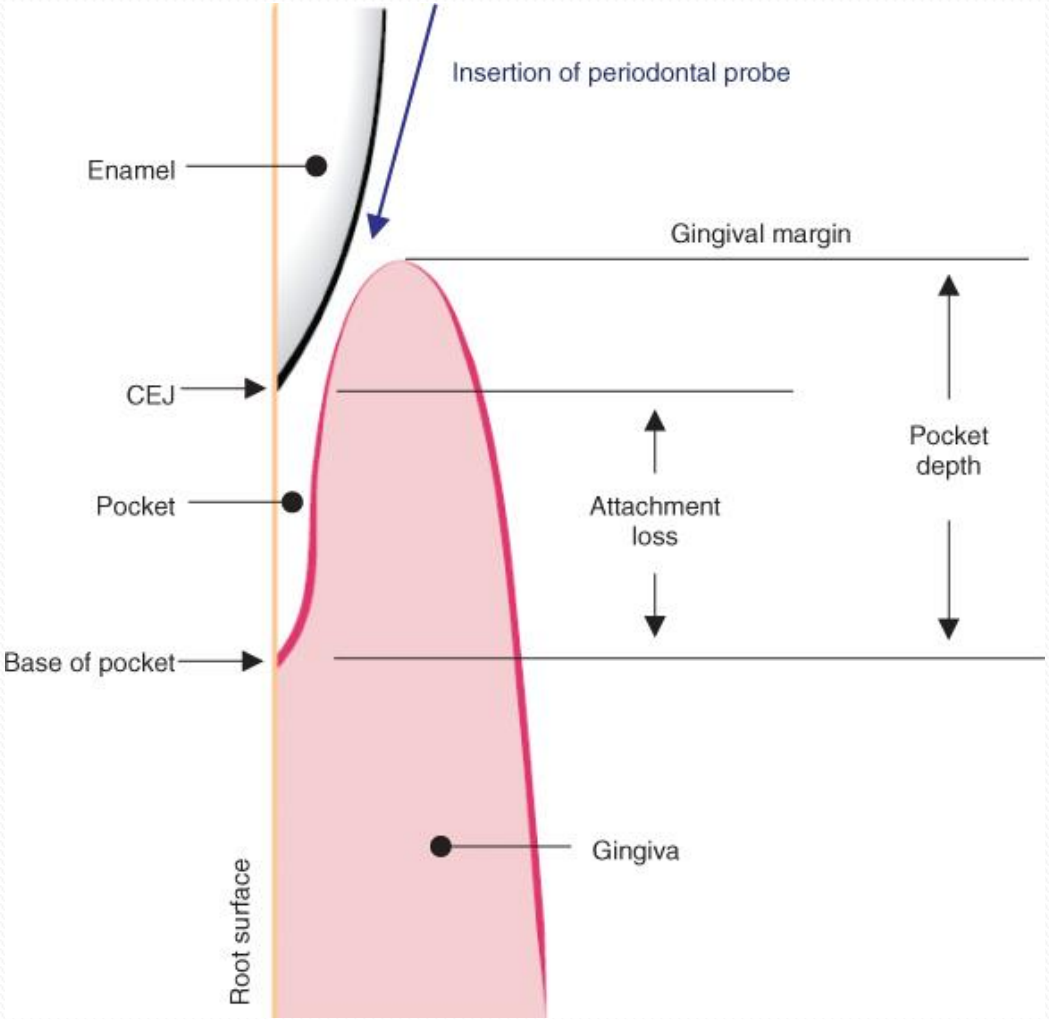
Insert probe carefully to bottom of the pocket and gently move laterally along the pocket wall. Recheck for bleeding 30-60 sec. after probing.

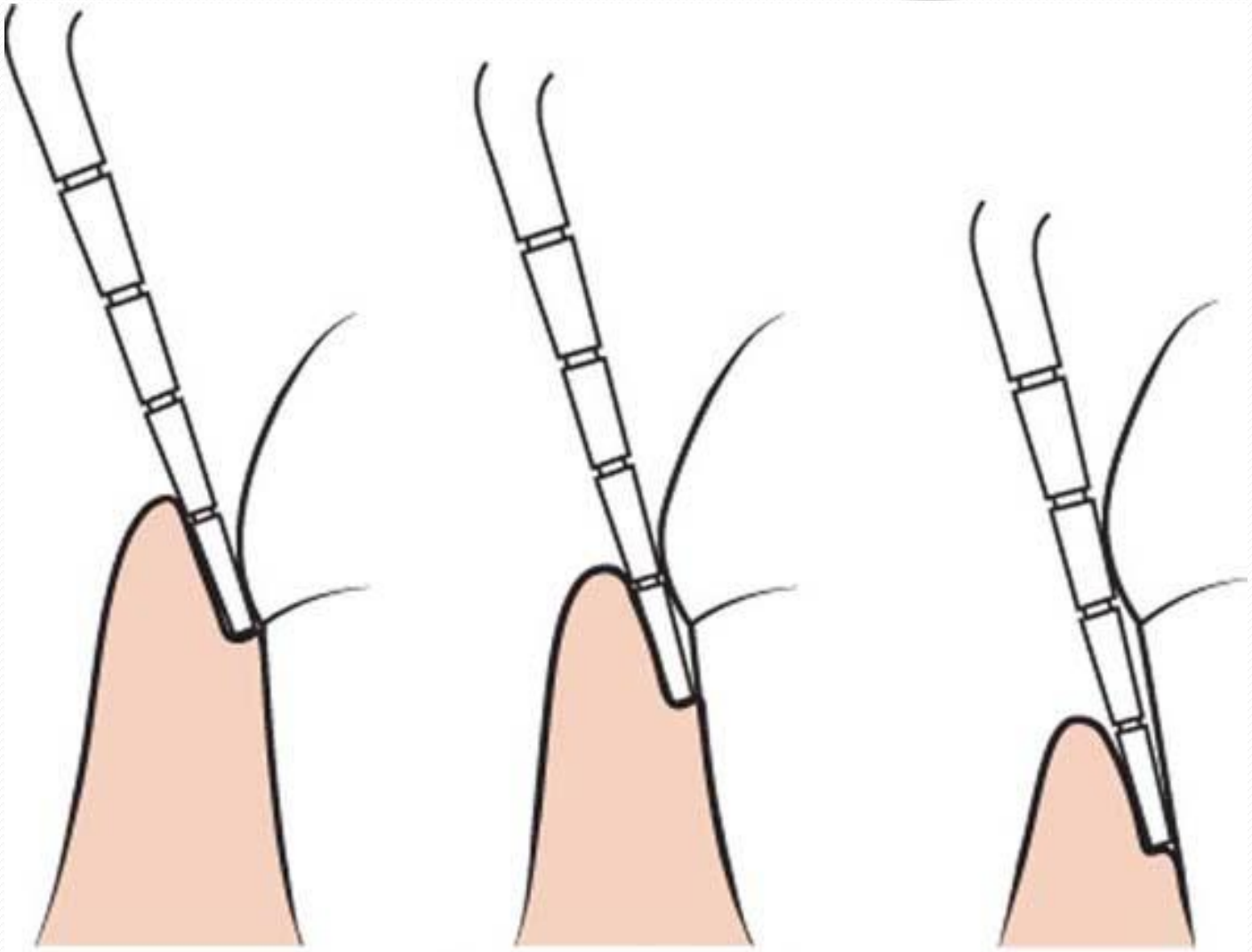
Transgingival probing :

Performed after the gingiva is anesthetized. More accurate method of evaluation and provides more information on bone architecture. Provides information about thickness, height and shape of the underlying base. Recommended immediately prior to surgical intervention.



Clinical Attachment Loss (CAL)





Correlation of clinical & histopathologic features of periodontal pocket

Sl. No.	Clinical features	Histopathologic features
1	Bluish red discoloration Flaccidity Smooth shiny surface Pitting on pressure	Circulatory stagnation, destruction of gingival fibers & surrounding tissues. Atrophy of epithelium Pressure by edema & degeneration
2	Pink & firm gingival wall	Fibrotic changes predominate over exudation & degeneration particularly in relation to outer surface of the pocket wall.

3	Bleeding is elicited by gentle probing the soft tissue wall of pocket	Due to increased vascularity, thinning & degeneration of the epithelium & proximity of engorged vessels to the inner surface.
4	On probing periodontal pocket is generally painful	Due to the ulceration of the inner aspect of the pocket wall.
5	Pus may be expressed by applying digital pressure	This occurs in pockets with suppurative inflammation of inner wall.

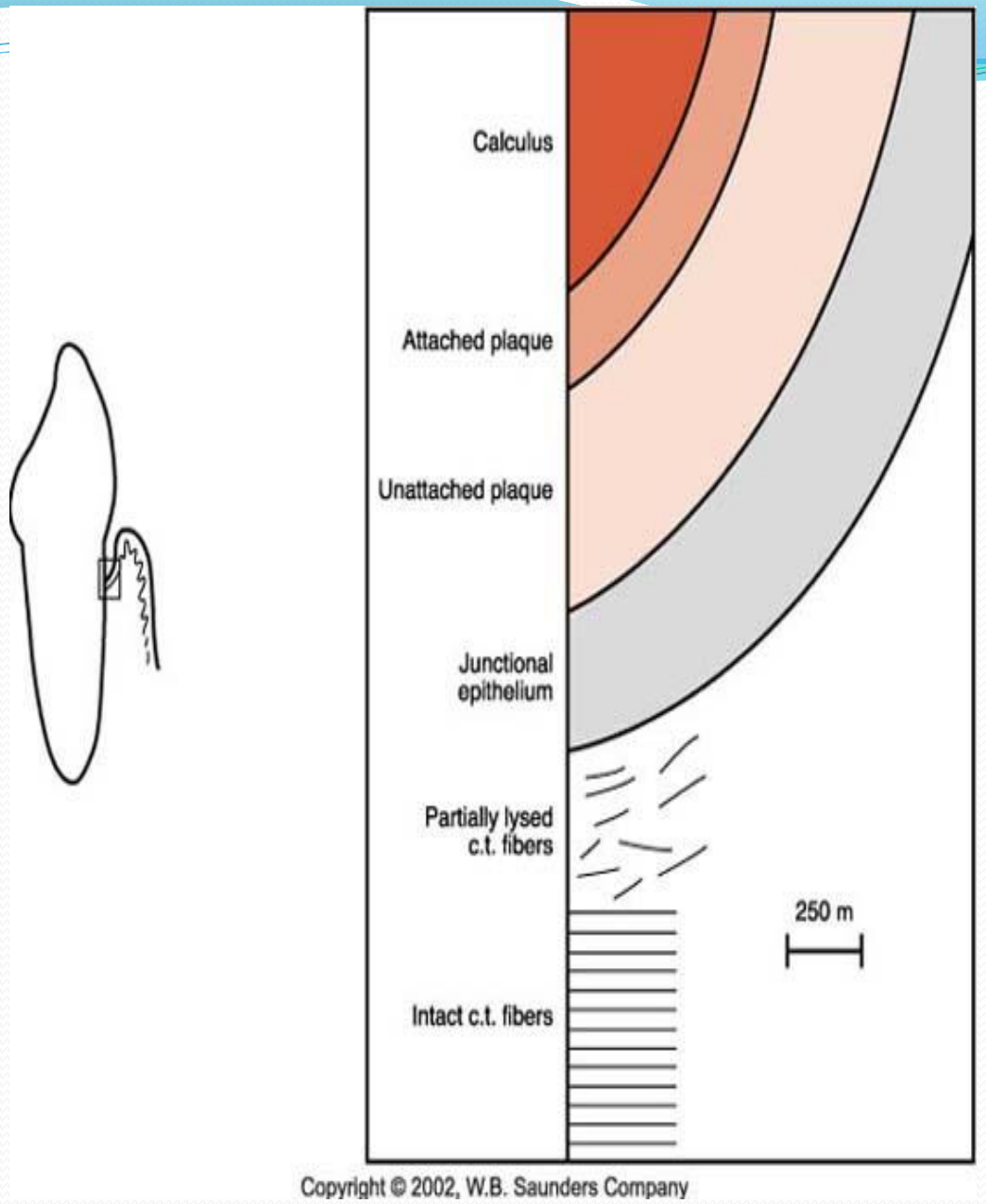
Pathogenesis of Periodontal pocket :

Pocket formation starts in connective tissue of the gingival sulcus.

The cellular and fluid inflammatory exudates causes degeneration of the surrounding connective tissue, including the gingival fibers

Just apical to the J.E. ⟶ Area with destroyed collagen & inflammed cells & oedema.

Beyond that zone of partial destruction then an area of normal attachment.



- Two hypothesis – Collagen loss

1) Collagenases (MMPs)
2) Fibroblasts phagocytoze collagen fibres

Pathogenesis

- As a consequence of the loss of collagen, the apical cells of the junctional epithelium proliferate along the root extending finger like projections two or three cells in thickness.
- The coronal portion of the junctional epithelium detaches from the root as the apical portion migrates.
- As a result of inflammation PMNs invade the coronal end of the junctional epithelium in increasing numbers.

- When the relative volume of PMNs reaches approximately **60%** or more of the JE, the tissue loses its cohesiveness and detaches from the tooth surface.
- Thus the sulcus bottom shifts apically, and the oral sulcular epithelium occupies a gradually increasing portion of the sulcular lining.

- ***Extension of the JE along the root requires the presence of healthy epithelial cells.***
- ***Marked degeneration and necrosis of the JE retards rather than accelerates pocket formation.***

Pathogenesis of periodontal pocket

Plaque → Gingival inflammation

↓

Pocket formation

↓

More plaque formation

Theories regarding the microscopic tissue changes in the initiation of pocket formation

1. Destruction of the gingival fibres is a prerequisite for the initiation of pocket formation

Proliferation of the epithelial attachment along the root can take place only if the attachments of the underlying gingival fibres into the cementum are destroyed – they act as barrier

2. The initial change in pocket formation occurs in the cementum

The continuous deposition of new cementum acts as a barrier - So long as continuous cemental deposition is not disturbed, migration of the JE, at a pathologic rate, will not occur.

3. Stimulation of the epithelial attachment due to infection or trauma is the initial histological change in pocket formation

- Stimulated by inflammation, the epithelium will migrate along the root without preceding destruction of the gingival fibers - In such instances, although the underlying fibres may remain intact, the epithelial cells will burrow between the fibres and attach themselves further apically along the cementum

4. Pathologic destruction of the epithelial attachment due to infection or trauma is the initial histological change in pocket formation

- The epithelial attachment is an area of low resistance, which is subject to infection.
- In experimental animals, pocket formation occurs because of pathologic destruction of the epithelial attachment due to infection or trauma or both.

5. The periodontal pocket is initiated by the invasion of bacteria at the base of the sulcus or the absorption of bacterial toxins through the epithelial lining of the sulcus

“pus-pocket” - an initial invasion of bacteria at the base of the sulcus - inflammation in the underlying connective tissue - ulceration at the base of the crevice, - sloughing of the epithelium, and loss of attachment to the cementum - progressive loss of connective tissue, and penetration of the pocket into the deeper tissues.

6. Pocket formation is initiated in a defect in the sulcus wall

7. Proliferation of the epithelium of the lateral wall, rather than the epithelium at the base of the sulcus, is the initial change in the formation of the periodontal pocket:

- Lateral wall proliferation – nutrition to cells of the sulcus is deprived – undergo degeneration and necrosis

8. Two stage pocket formation

- The first stage is proliferation of the subgingival epithelium.
- The second stage is loss of the superficial layers of the proliferated epithelium at the base is such that it precedes the destruction of the superficial epithelium, and the pocket is therefore always lined with epithelium.

9. Inflammation is the initial change in the formation of the periodontal pocket:

- *Periodontal lesions of purely local origin have their beginnings*
- Inflammation in the connective tissue - Increased mitotic activity in the basal epithelial layer, and sometimes in the prickle cell layer - Increased production of keratin with desquamation.
- It is the cellular desquamation adjacent to the tooth surface, which tends to deepen the pocket.

10. Pathologic epithelial proliferation occurs secondary to non-inflammatory degenerative changes in the periodontal membrane

Non-inflammatory degenerative changes in the periodontal membrane has been described as **“periodontosis”** – under such conditions the normal barrier afforded by the gingival fibres is diminished, this facilitates the migration of the epithelial attachment along the root and pocket formation, in the presence of local irritation.

Pocket contents :

- Microorganisms & its products (enzymes, endotoxins etc.)
- Gingival fluid
- Food remnants
- Salivary mucin
- Desquamted epi. cells
- Leukocytes
- Plaque covering calculus
- Purulent exudate etc.

Histopathology

Conversion of Junctional Epithelium to Pocket Epithelium

- Significant changes occur in the junctional epithelium at an early stage of gingivitis.
- These include an increase in the amount of extra cellular space; probably reflecting increased permeability, a loss of intercellular junctions, beginning proliferation of the basal cells with rete ridge formation, and loss of the attachment between the epithelium and the tooth surface via the attachment lamina.

Soft tissue wall

- The most severe degenerative changes in the periodontal pocket occur along the lateral wall

Epithelium :

- Presents proliferative & degenerative changes.
- Epithelial buds or interlacing cords of epithelial cells project into C.T.
- Epithelium projections are infiltrated by leukocytes & edema from inflamed C.T.
- Cells undergo vascular degeneration & rupture to form vesicles.
- Progressive degeneration & necrosis of lateral wall – ulceration exposure of inflamed C.T. & suppuration.

Junctional epithelium :

- Is much shorter than that of a normal sulcus (50-100 μm). Cells may show slight or marked degenerative changes.

Connective tissue :

- Edematous & densely infiltrated with plasma cells (80%), lymphocytes & scattering PMN's.
- Blood vessels – increased in number, dilated & engorged.
- Single or multiple necrotic foci.
- Also shows proliferative changes of endothelial cells, newly formed capillaries & collagen fibers.

Microtopography of the gingival pocket wall SEM shows the following areas

1) **Areas of relative quiescence :**

   Flat surface with depressions & mounds & shedding of cells.

2) **Area of bacterial accumulation :**

   Depressions on epi. surface with debris & bacterial clumps penetrating intercellular spaces.

3) **Area of emergence of leukocytes :**

   Leukocytes appear in the pocket wall through holes located in intercellular spaces.

4) **Area of leukocyte – bacterial interaction :**

   Numerous leukocytes are present & covered with bacteria in process of phagocytosis.

Bacterial invasion of apical & lateral areas of pocket can occur in human chronic periodontitis.

Filament, rods & coccoid org with predominent gram –ve cell walls found in intercellular spaces of exfoliating cells, deeper epi. cells, basement lamina, subepi. C.T.

The significance, if any, of bacterial invasion in the pathogenesis of P.D. is not yet clarified.

5) Areas of intense epithelial desquamation :

Epithelium covered with bacteria.

6) Areas of ulceration :

With exposed C.T.

7) Areas of hemorrhage :

With numerous erythrocytes.

- The transition from one area to other could result from bacterial invasion in the previously quiescent area and leukocyte - bacteria interaction

- The condition of the soft tissue wall of the periodontal pocket results from the interplay of the destructive and constructive tissue changes.
- The balance between destructive & constructive changes determines clinical features such as color, consistency etc of the pocket wall.
- If inflammatory fluid and cellular exudates predominate, the pocket wall is bluish red, soft, spongy, and friable, with a smooth, shiny surface.
- If there is a relative predominance of newly formed connective tissue cells and fibers, the pocket wall is more firm and pink.

Root surface wall :

Root cementum shows structural, chemical & cytotoxic changes.

Structural changes :

1) **Presence of pathologic granules :**

Represent areas of collagen degeneration or areas where collagen fibrils have not been fully mineralized initially.

2) **Areas of increased mineralization :**

Due to exposure to the oral cavity of minerals & organic components at the cementum – saliva interface. The hypermineralized zones are 10-20 μm thick upto 50 μm.

Areas of demineralization :

•Commonly related to root caries.

•Exposure to oral fluid & bacterial plaque results in proteolysis of sharpey's fibers,

•the cementum may be softened & may undergo fragmentation and cavitation.

•R.S. caries spreads around than into the tooth. Active lesions are yellowish or light brown, covered with plaque & soft or leathery consistency.

•Inactive lesions darker lesions with smooth surface & hard consistency on probing.

- The dominant org. of root caries is *Actinomyces viscosus*
- Involvement of cementum is followed by bacterial penetration of dentinal tubules resulting in destruction of dentin.

Chemical changes :

- Mineral content increased by absorption from local environment. (Ca, Magnesium, Phosphorus & Fluoride).
- A highly calcified layer is formed resistant to decay.
- Microhardness is unchanged.

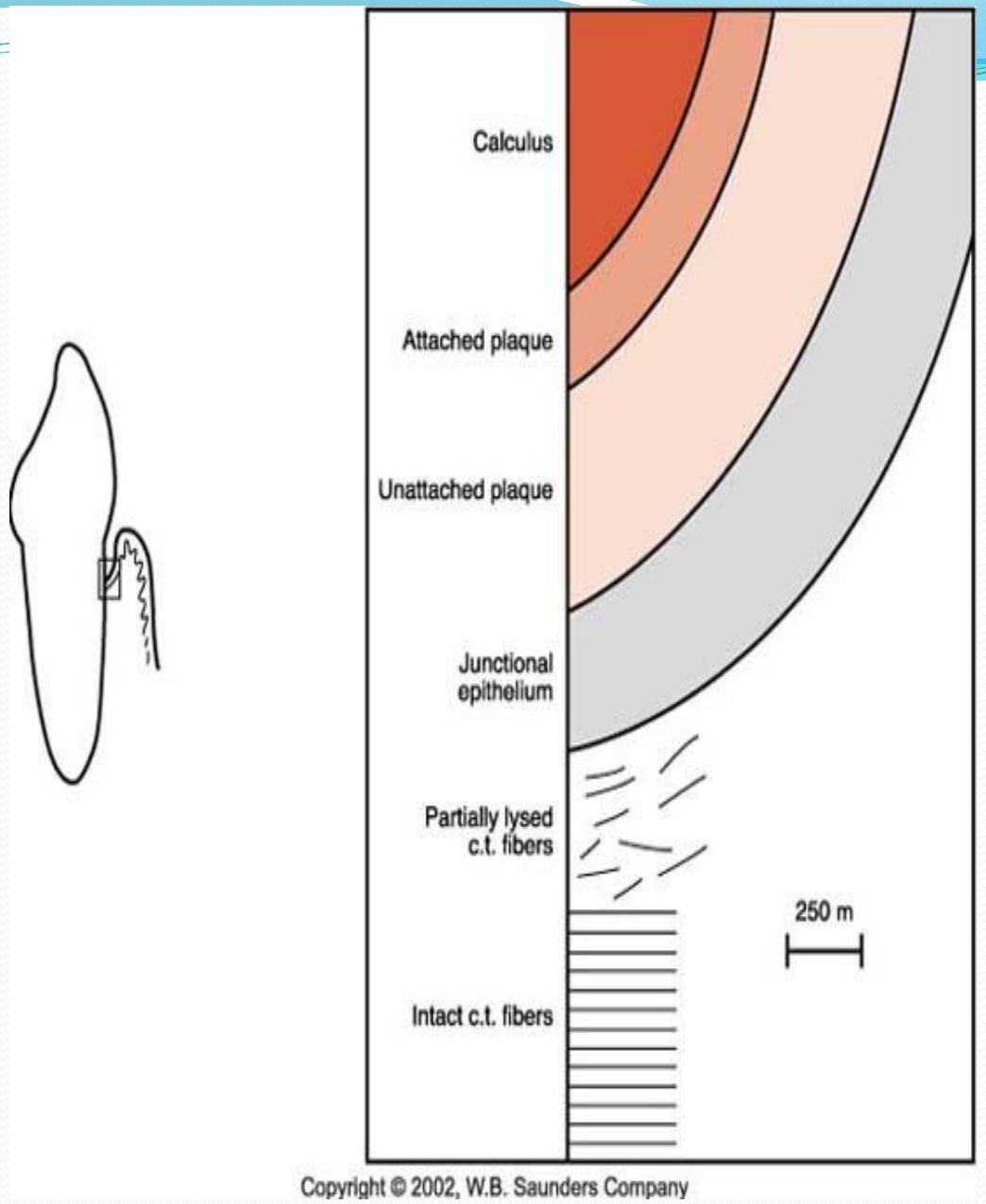
Cytotoxic changes:

- Bacteria & bacterial products like endotoxic can be found as deep as cementodentinal junction.
- Diseased root fragments bring about irreversible morphologic changes in the cells, prevent in vitro attachment of human gingival fibroblasts.
- They also induce an inflammation response even if autoclaved reimplantation.

Surface morphology of tooth wall

- Following zones are found at the bottom of a periodontal pocket

1. ***Cementum covered by calculus***
2. ***Attached plaque*** which covers calculus and extends apically 100 to 500 µm
3. ***Zone of unattached plaque*** which surrounds attached plaque
4. ***Zone of attachment of the junctional epithelium to the tooth*** – in normal sulci 500 µm but reduced to 100 µm in periodontal pockets
5. ***Zone of semidestroyed connective tissue fibres*** apical to junctional epithelium



Periodontal disease activity :

Periodontal pocket go through periods of quiescence & exacerbation.

Period of quiescence :

- Reduced inflammation response, little or no loss of bone & C.T. gram +ve, non-motile org, aerobic. Clinically no bleeding or exudate (Period of inactivity).

Period of exacerbation :

- Bone & C.T. attachments are lost, pocket deepens. Gram –ve, motile org & anaerobic org. Clinically bleeding on probing, exudation present.

Site specificity:

- Periodontal destruction does not occur in all parts of the mouth at the same time but rather on a few teeth at a time or even only some aspects of some teeth at any given time.
- Sometimes pulp changes may be associated with periodontal pockets.

Periodontal pockets as healing lesions :

- Pockets are chronic inflammatory lesions & are constantly undergoing repair.
- Complete healing *does* not occur because of the persistence of the bacterial attack, which continues to stimulate an inflammatory response, causing degeneration of the new tissue elements formed in the continuous effort at repair.

Significance of pus formation :

- Consist of living degenerated & necrotic leukocytes, living & dead bacteria, serum, fibrin.
- It is a common feature of periodontal disease but it is only a secondary sign.
- It merely reflects the nature of inflammation changes in the pocket wall, not an indication of depth of pocket or severity of periodontal disease.

THANK YOU