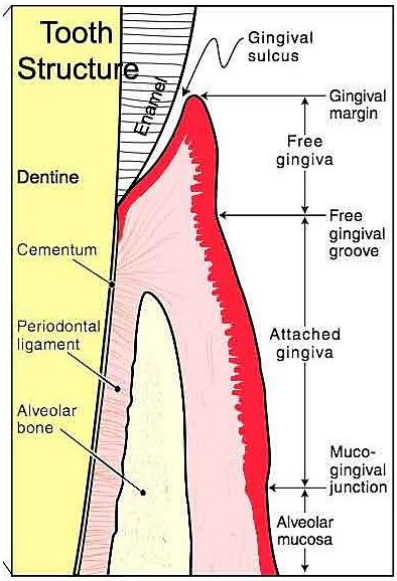
Gingival and periodontal pocket

Tooth gingival interface

The interface between a tooth and the surrounding gingival tissue is a **dynamic structure**. The gingival tissue forms a crevice surrounding the tooth, resemble fluid-filled moat, wherein food debris, endogenous and exogenous cells, and chemicals float. The depth of this crevice, known as a **sulcus**, is in a constant state of flux due to microbial invasion and subsequent immune response. Located at the depth of the sulcus is the **epithelial attachment**, consisting of approximately **1 mm of junctional epithelium** and another **1 mm of gingival fiber attachment**, comprising the **2 mm of biologic width** naturally found in the oral cavity. The sulcus is literally the area of separation between the surrounding epithelium and the surface of the encompassed tooth.



A **healthy sulcular** depth is **3 mm or less**. Through much investigation and research, it has been determined that sulcular depths of 3 mm or less are readily **self-cleansable** with a properly used toothbrush or the supplemental use of other oral hygiene aids.

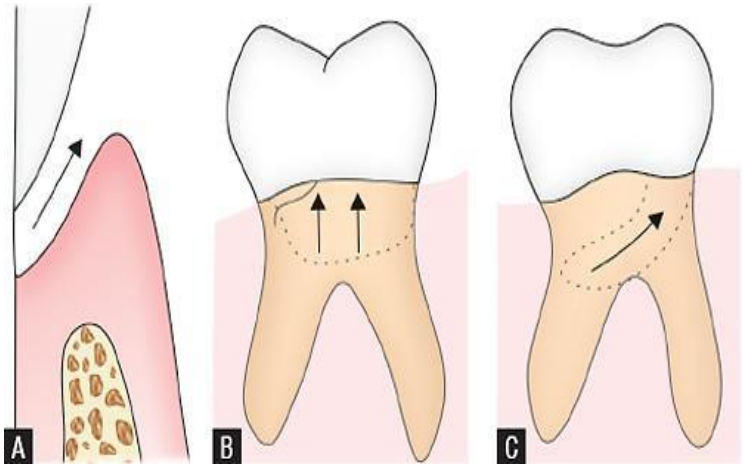
When the sulcular depth is chronically in **excess** of 3 mm, regular home care may be insufficient to properly cleanse the full depth of the sulcus, allowing food debris and microbes to accumulate, **forming dental biofilm**. This poses a danger to the periodontal ligament (PDL) fibers that attach the gingiva to the tooth. If accumulated microbes remain undisturbed in a sulcus for an extended period of time, they will penetrate and ultimately destroy the delicate soft tissue and periodontal attachment fibers. If left untreated, this process may lead to a deepening of the sulcus, recession, destruction of the periodontium, including the bony tooth socket, tooth mobility, and tooth loss. **Both the gingival and periodontal pocket** are extensions of the gingival sulcus, which exists in health. Gingival and periodontal pockets are dental terms indicating the presence of an abnormal depth of the gingival sulcus.

The periodontal pocket :- It's an inflammatory changes and apical migration of junctional epithelium; it is **also defined as** a pathological deepening of the gingival sulcus, is one of the **most important clinical features** of periodontal disease. All types of periodontitis, share histopathologic features, such as tissue changes in the periodontal pocket, mechanisms of tissue destruction, and healing mechanisms.

Classification

1. According to the involved tooth surface:

A. **Simple pocket:** involve one surface
B. **Compound pocket**: involve more than one surface
C. **Complex or spiral pocket:** originating on one surface and twisting around the tooth to involve one or more additional surface (but it opens into the oral cavity on the surface of its origin). These types of pockets **are most common** in furcation areas.



2. According to its location:

a) Gingival pocket: (also called **False or "pseudo-pocket**") which is formed by gingival enlargement without destruction of underlying periodontal tissue. The sulcus is deepened because of increased bulk of the gingiva. A gingival pocket presents when the marginal gingiva experiences an **edematous reaction**, whether **due to localized irritation** and subsequent inflammation, **systemic issues**, or **drug induced gingival hyperplasia**. In a gingival pocket, **no destruction** of the connective tissue fibers

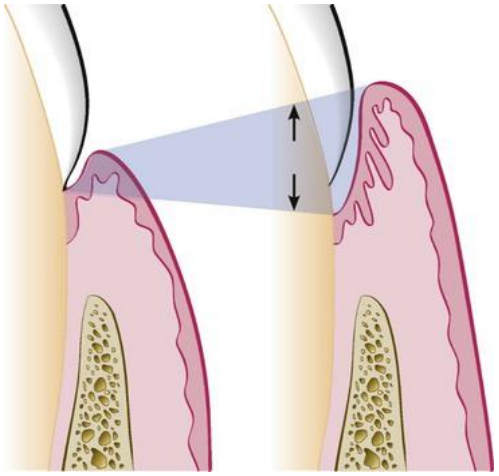


Fig.:- Gingival pocket.

(gingival fibers) or alveolar bone occurs. This early sign of disease in the mouth is **completely reversible** when the etiology of the edematous reaction is eliminated and frequently occurs without dental surgical therapy. However, in certain situations, **gingivectomy** is necessary to reduce the gingival pocket depths to a healthy 1– 3 mm.

b) Periodontal pocket: associated **with destruction** of underlying supportive tissues. periodontal pocket, which is defined as a pathologically deepened gingival sulcus, is one of the most important clinical features of periodontal disease.

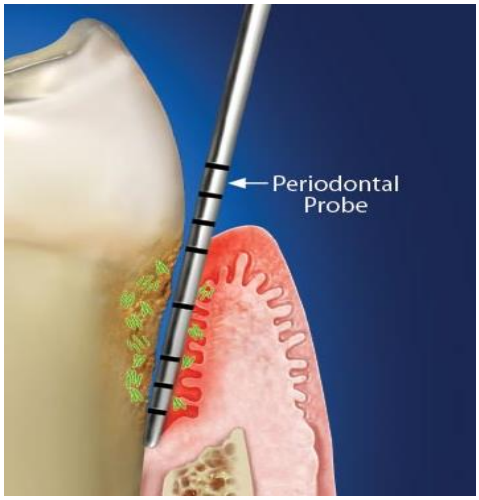


Fig.: Periodontal pocket (True pocket)

3. According to its relation to alveolar crest:

a) Suprabony pocket: also called supra crestal or supra alveolar. The base of the pocket is coronal to the level of underlying bone. The **bone loss is horizontal.**

b) Infrabony pocket: also known as sub crestal or intra alveolar pocket. The base of the pocket is apical to the level of adjacent bone. The **bone loss is vertical**.

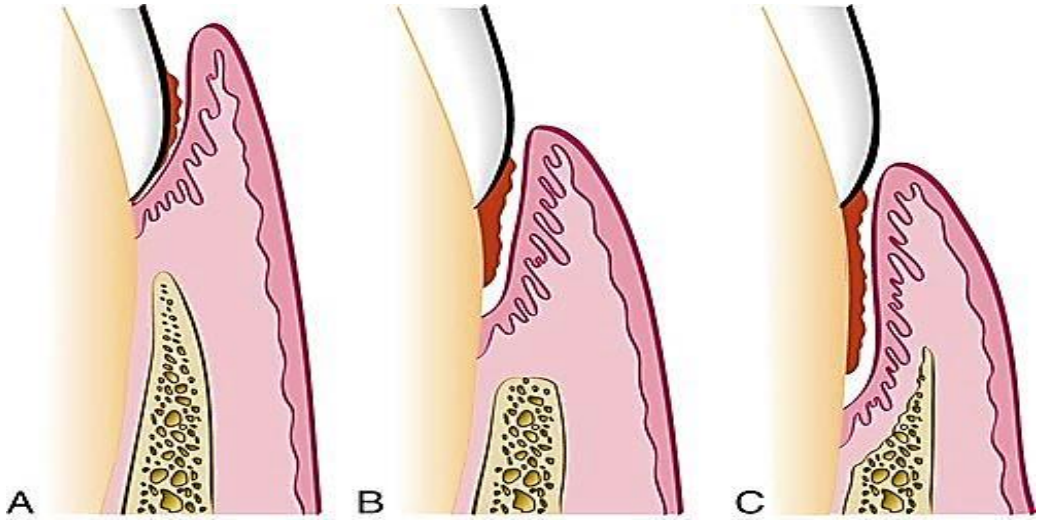


Fig. :- A. Gingival pocket, B. Suprabony pocket, C. Infrabony pocket.

Classification of Infrabony pocket according to the depth and width of the defect:

1. Narrow deep pocket
2. Wide deep pocket
3. Narrow shallow pocket
4. Wide shallow pocket

The number of the remaining osseous walls:

- ❖ Three osseous wall infrabony pocket
- ❖ Tow osseous wall infrabony pocket
- ❖ One osseous wall infrabony pocket
- ❖ Combined osseous defect

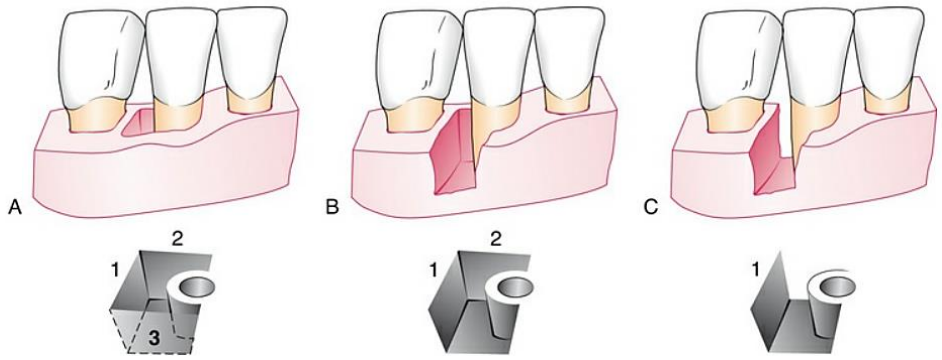


Fig.:- Diagrammatic representation of one-, two-, and three-walled vertical defects.

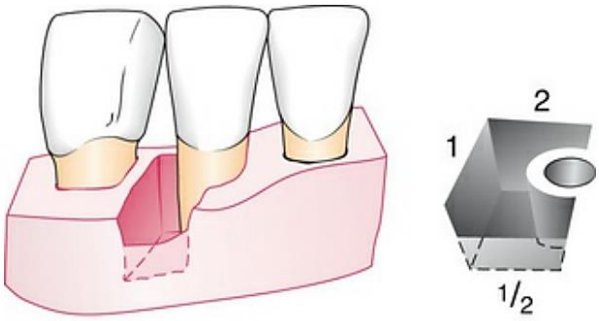


Fig.:- Combined type of osseous defect. Because the facial wall is half the height of the distal (1) and lingual (2) walls, this is an osseous defect with three walls in its apical half and two walls in its occlusal half.

Diagnosis/ detection of pockets

1. Careful exploration with periodontal probe (this method is accurate).

2. Radiographic: pockets are not detected by the radiographic examination because pockets are soft tissue changes. A calibrated silver points or gutta percha points can be used with radiographic to assist in determining the level of attachment of periodontal pocket.

Clinical Features

Clinical signs & symptoms that suggest the presence of periodontal pockets **include:-**

- ☒ Bluish red thickened marginal gingiva
- ☒ Bluish red vertical zone from the gingival margin to the alveolar mucosa
- ☒ Gingival bleeding and suppuration
- ☒ Tooth mobility
- ☒ Diastema formation
- ☒ Localized pain or pain "deep in the bone.

The only reliable method of locating periodontal pockets and determining their extent is careful probing of the gingival margin along each tooth surface.

Table :- Correlation of Clinical and Histopathologic Features of the Periodontal Pocket

Clinical Features	Histopathologic Features
1. The gingival wall of the pocket presents as bluish red discoloration; flaccidity; a smooth, shiny surface; and pitting on pressure.	1. The discoloration is caused by circulatory stagnation; the flaccidity by the destruction of gingival fibers and surrounding tissues; the smooth, shiny surface by atrophy of the epithelium and edema; and the pitting on pressure by edema and degeneration.
2. Less frequently, the gingival wall may be pink and firm.	2. In such cases, fibrotic changes predominate over exudation and degeneration.
3. Bleeding is elicited by gently probing	3. Ease of bleeding results from increased vascularity, the

the soft tissue wall of the pocket.	thinning and degeneration of the epithelium, and the proximity of engorged vessels to the inner surface.
4. When explored with a probe, the inner aspect of the pocket is generally painful.	4. Pain on tactile stimulation is caused by the ulceration of the inner aspect of the pocket wall.
5. In many cases, pus may be expressed with the application of digital pressure.	5. Pus occurs in pockets with suppurative inflammation of the inner wall.

Pathogenesis of the periodontal pocket

The initial lesion in the development of periodontitis **is the inflammation** of the gingiva in response to a bacterial challenge. Changes involved in the transition from the normal gingival sulcus to the pathologic periodontal pocket are associated with different proportions of bacterial cells in dental plaque. **Healthy gingiva** is associated with few microorganisms, **mostly coccoid cells and straight rods. Diseased gingiva** is associated with increased numbers of **Spirochetes and motile rods**. However, the microbiota of diseased sites cannot be used as a predictor of future attachment or bone loss, because their presence alone is not sufficient for disease to start or progress.

- **Accumulation of microorganisms on the supra gingival tooth surface and its extension into gingival sulcus.**
- **Inflammatory changes in the connective tissue wall of the gingival sulcus.**
- **Cellular and fluid inflammatory exudate causes degeneration of the connective tissue including the gingival fibers.**
- **Collagen fibers gets destroyed apical to the junctional epithelium and the area becomes occupied by the inflammatory cells and edema.**
- **The coronal portion of the junctional epithelium detaches from the root as the apical portion migrates.**
- **Polymorphonuclear neutrophils invade the coronal end of the junctional epithelium in increasing number.**
- **With continued inflammation the gingiva increase in bulk and the crest of the gingival margin extends coronally.**
- **The junctional epithelium continues to migrate along the root and separate from the root.**

There is two mechanisms associated with collagen loss:-

1- **Collagenases and other enzymes** secreted by various cells in healthy and inflamed tissue, such as fibroblasts, polymorphnuclear leukocyte (**PMNs**), and macrophages, become extracellular and destroy collagen, these enzymes that degrade collagen into small peptides are called matrix metalloproteinases (**MMP**).
2- **Fibroblasts** phagocytize collagen fibers by extending cytoplasmic processes to the ligament cementum interface and degrade the collagen fibrils.

Pathogenesis of bone destruction and periodontal pocket

The most common cause of periodontal disease **is the extension of inflammation from marginal gingiva into the supporting periodontal tissues**. The inflammatory process that occurs in periodontitis results in permanent destruction to the tissues of the periodontium, including the destruction of gingival connective tissue, PDL, alveolar bone & formation of periodontal pocket. The **pattern of bone destructions** that occurs in periodontitis **depends on** the pathway of inflammation as it spreads from the gingival into the alveolar bone it is important to understand the changes that occur in the alveolar bone because it is the reduction in bone height that eventually results in tooth loss.

Changes in alveolar bone height:

A. In health & gingivitis: The crest of the alveolar bone is located approximately 2 mm apical to (below) the CEJs of the teeth.

B. In periodontitis: Bone destruction may be marked as periodontal disease progresses.

Patterns of bone loss in periodontal disease, there are two types of bone loss are:

A. Horizontal Bone Loss

l. Is the most common pattern of bone loss, results in fairly even, overall reduction in the height of alveolar bone.

2. The bone is reduced in height, but the bone margin remains roughly perpendicular to the tooth surface

3. This type of bone loss produces a suprabony pocket

B. Vertical Bone Loss

1. Is a less common pattern of bone loss, results in an uneven reduction in the height of the alveolar bone.

2. Bone resorption progressing more rapidly in the bone next to the root surface, also **known as angular bone loss**. This uneven pattern of bone loss leaves a **trench like area** of missing bone alongside the root.

3. This type of bone loss produces an infrabony pocket.

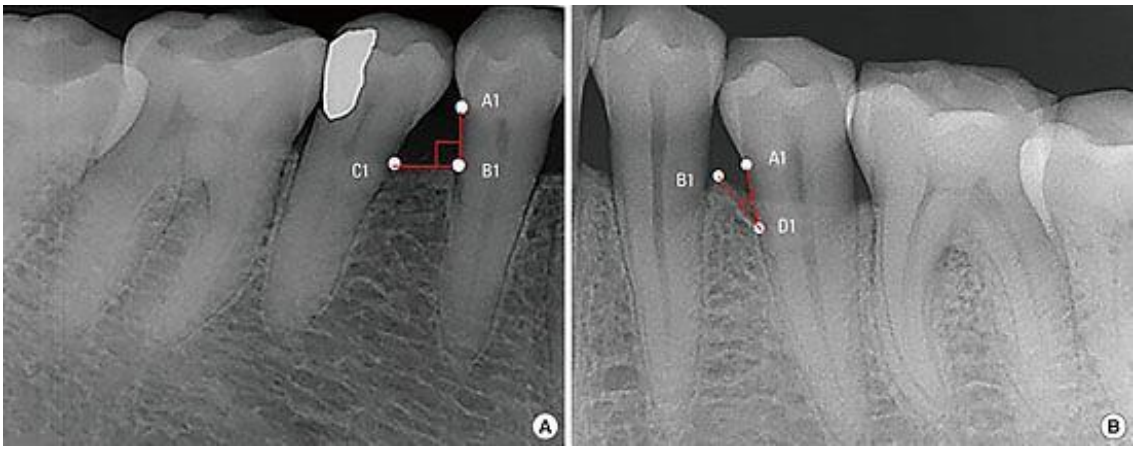


Fig.:- A. Horizontal bone loss , B. Angular bone loss

Pathways of inflammation into bone:

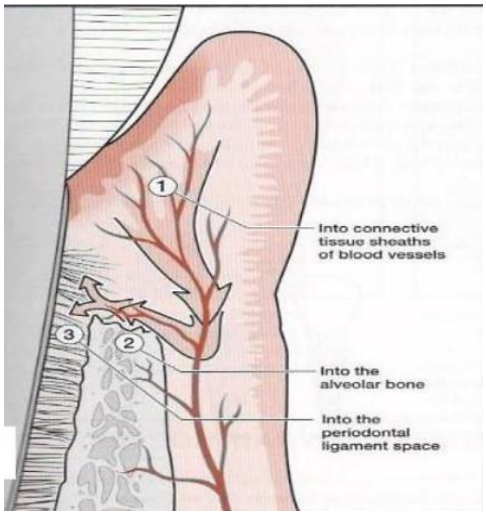
1. Pathway of Inflammation in (Horizontal Bone Loss)

A. inflammation spreads:

(i) Within the gingival connective tissue surrounding the blood vessels.

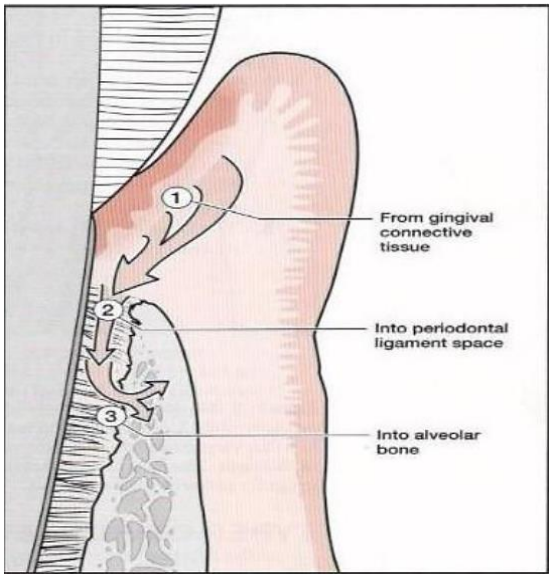
(ii) Into the alveolar bone.

(iii) Finally, into PDL space.



B. Inflammation usually spreads in this manner because it is the path of least resistance. **PDL fiber** bundles act as an **effective barrier** to the spread of inflammation. Thus, the inflammation spreads into the alveolar bone and then into P DL space.

2. Pathway of Inflammation in (Vertical Bone Loss)



A. Inflammation spreads:

(i) Within the gingival connective tissue

(ii) Directly into PDL space

(iii) Finally, into the alveolar bone.

B. Inflammation spreads in this manner when the crestal PDL fiber bundles are weakened & no longer represent an effective barrier. Prior events such as occlusal trauma can be responsible for the weakened condition of fiber bundles.

Histopathology:

A- Epithelial changes

The epithelium of the lateral wall of the pocket presents striking proliferative and degenerative changes. Epithelial buds or interlacing cords of epithelial cells project from the lateral wall into the adjacent inflamed connective tissue, and they may extend farther apically than the junctional epithelium

- Epithelium becomes degenerated and atrophied **(The severity of the degenerative changes is not necessarily related to pocket depth).**
- Inner aspect of the lateral pocket walls becomes ulcerated due to progressive degeneration and necrosis of the epithelium.
- Pus occurs in the pocket with suppurative inflammation of the inner wall.
- The epithelium at the gingival crest of a periodontal pocket is generally intact and thickened, with prominent rete pegs

B- Connective tissue changes

- The connective tissue is edematous and densely infiltrated with plasma cells(approximately 80%) ,lymphocytes and a scattering of PMNs.
- Blood vessels are increased in number , dilated and engorged particularly in sub epithelial connective tissue layer.

- ❖ The connective tissue exhibits varying degrees of degeneration. Single or multiple necrotic foci are occasionally present. In addition to exudative and degenerative changes,
- ❖ Proliferation of endothelial cells .with newly formed capillaries, fibroblasts, and collagen fibers

C- Root surface wall of the pocket

Root surface forms the medial wall of the pocket. As the pocket deepens, collagen fibers embedded in the cementum are destroyed, and cementum becomes exposed to the oral environment.

The root surface that get expose to the oral environment as a result of periodontal attachment loss, undergoes following changes **(structural, chemical, cytological)**.

Structural changes:

Exposure of cementum to the oral environment ⟶ Minerals present in saliva tend to get deposited on cementum surface (Ca, F,P,Mg) ⟶ area of hypermineralization.

Root surface is exposed to oral fluids and bacterial plaque ⟶ proteolysis of embedded remnants of Sharpey's fibers ⟶ Area of demineralization root caries (yellowish or brown patch) soft and lethargy on probing ⟶ patient feels hypersensitivity to the thermal changes and sweets ⟶ pulp exposure may occur in sever forms. (demineralization is related to dental carries).

Chemical changes

Cementum exposed to saliva may absorb calcium, phosphorus, magnesium and fluoride. This increase in mineral content of the root surface alters the chemical composition of the cementum, making it resistant to dental caries.

Cytotoxic changes

Histologic studies of periodontally involved cementum have shown the presence of bacteria in the cementum or endotoxins in the cementum. Collagenous remnants of Sharpey fibers in the cementum undergo degeneration, thereby creating an environment favorable to the penetration of bacteria. Bacterial penetration into the cementum can be found as deep as the cemento dentinal

junction, and it may also enter the dentinal tubules. Penetration and the growth of bacteria leads to fragmentation and breakdown of the cementum surface and results in areas of necrotic cementum that are separated from the tooth by masses of bacteria.

In addition, bacterial products (e.g., endotoxins) have also been detected in the cementum wall of periodontal pockets , diseased root areas induce an inflammatory response, and prevent fibroblast attachment These changes manifest clinically as softening of the cementum surface; this is usually asymptomatic, but it can be painful when a probe or explorer penetrates the area. They also constitute a possible reservoir for reinfection of the area after treatment. During the course of treatment, these necrotic areas are removed by root planing until a hard, smooth surface is reached. Cementum is very thin in the cervical areas, and scaling and root planing often remove it entirely, exposing the underlying dentin. Sensitivity to cold may result until secondary dentin is formed by the pulp tissue.

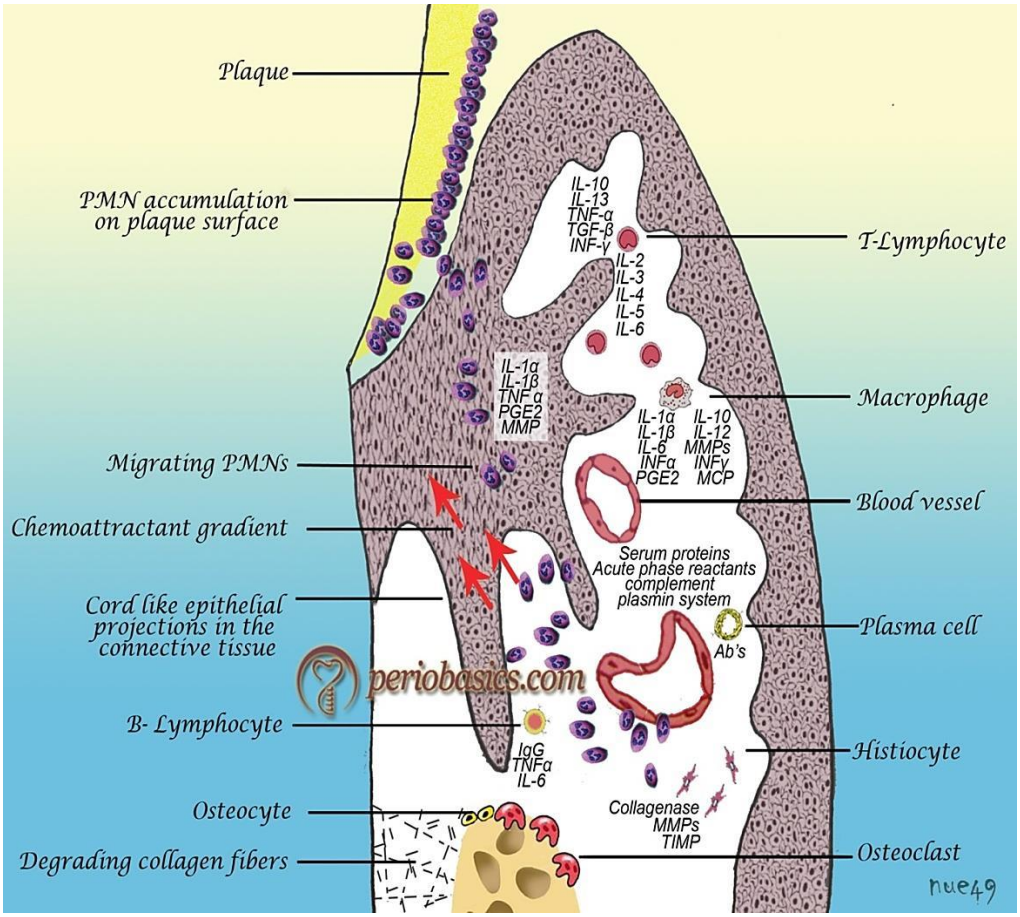


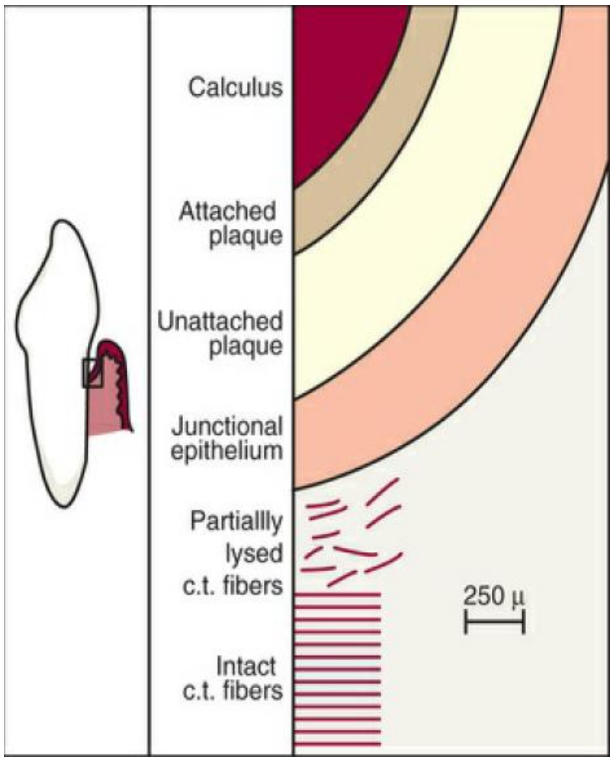
Fig. :- Pathogenesis of the periodontal pocket.

Content of the pocket:

- Microorganisms .
- Bacterial products (enzymes and endotoxins).
- GCF.
- Remnants of food
- Salivary mucin.
- Desquamated epithelial cells.
- Leukocytes
- Plaque covered calculus usually projects from the tooth surface
- Purulent exudates may be present and this is a **secondary feature** because deep pocket may have little or no pus and shallow pocket may have extensive pus formation s**o pus is not an indicator** of the depth of pocket.

Surface Morphology of Tooth Wall

The following zones can be found in the bottom of a periodontal pocket:-



1. Cementum covered by calculus, in which all of the changes can be found.

2. Attached plaque, which covers calculus and which extends apically from it to a variable degree (typically 100 to 500 µm).

3. The zone of unattached plaque that surrounds attached plaque and extends apically to it.

4. The zone of attachment of the junctional epithelium to the tooth. The extension of this zone, which in normal sulci is more than 500 µm, is usually reduced in periodontal pockets to less than 100 µm.

5. A zone of semidestroyed connective tissue fibers may be apical to the junctional epithelium. Zones 3, 4, and 5 make up the "plaque-free zone" seen in extracted teeth.

Micro-topography of gingival wall

Scanning electron microscopy has permitted the description of several areas in the soft tissue (gingival) wall of the periodontal pocket in which different types of activity take place. These findings suggest that the pocket wall is constantly changing as a result of the interaction between the host and the bacteria. The following areas have been noted:

1. Areas of relative quiescence showing a relatively flat surface with minor depressions and elevations and occasional shedding of cells .

2. Areas of bacterial accumulation, which appear as depressions on the epithelial surface, with abundant debris and bacterial clumps penetrating into the enlarged intercellular spaces.

3. Areas of emergence of leukocytes, in which leukocytes appear in the pocket wall through holes located in the intercellular spaces .

4. Areas of leukocyte-bacteria interaction, in which numerous leukocytes are present and covered with bacteria in an apparent process of phagocytosis.

5. Areas of intense epithelial desquamation, which consist of semi attached and folded epithelial cells, sometimes partially covered with bacteria .

6. Areas of ulceration, with exposed connective tissue .

7. Areas of hemorrhage, with numerous erythrocytes.

The transition from one area to another could result from bacteria accumulating in previously quiescent areas and triggering the emergence of leukocytes and the leukocyte-bacteria interaction. This would lead to intense epithelial desquamation and finally to ulceration and hemorrhage.

Periodontal Pockets as Healing Lesions

Periodontal pockets are chronic inflammatory lesions and thus 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. The condition of the soft tissue wall of the periodontal pocket results from the interplay of the destructive and constructive tissue changes. If

the inflammatory fluid and cellular exudate predominate, the pocket wall is bluish red, soft, spongy, and friable, with a smooth, shiny surface; at the clinical level, this is generally referred to as an **edematous pocket wall**. If there is a relative predominance of newly formed connective tissue cells and fibers, the pocket wall is more firm and pink, clinically referred to as a **fibrotic pocket wall** .

Edematous and fibrotic pockets represent opposite extremes of the same pathologic process not different disease entities. **Fibrotic pocket walls may be misleading because they do not necessarily reflect what is taking place throughout the pocket wall.** In some cases, inflammation and ulceration on the inside of the pocket are walled off by fibrous tissue on the outer aspect . Externally the pocket appears pink and fibrotic, despite the inflammatory changes occurring internally.

Periodontal disease activity

Periodontal pockets go through periods of exacerbation and quiescence, resulting from episodic bursts of activity followed by periods of remission.

Periods of quiescence are characterized by a reduced inflammatory response and little or no loss of bone and connective tissue attachment. A buildup of unattached plaque, with its gram-negative, motile, and anaerobic bacteria , starts a **period of exacerbation** in which bone and connective tissue attachment are lost and the pocket deepens. This period may last for days, weeks, or months and is eventually followed by a period of remission or quiescence in which gram-positive bacteria proliferate and a more stable condition is established.

These periods of quiescence and exacerbation are also known as **periods of inactivity and periods of activity**. **Clinically,** active periods show bleeding, either spontaneously or with probing, and greater amounts of gingival exudate.

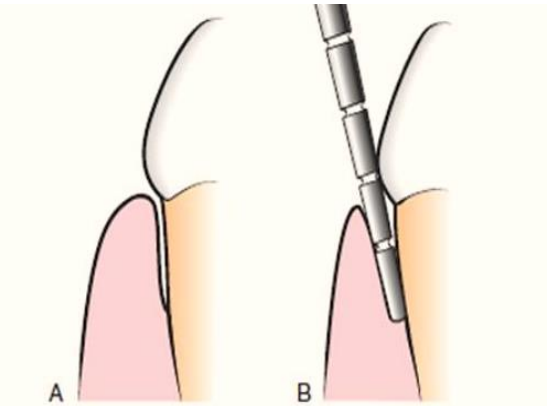
Pocket depth

There are two different pocket depths:

1) <u>Biologic or histologic depth:</u> distance between gingival margin and base of the pocket. measured histologically (accurate measurement but not used routinely).

2) <u>Clinical or probing depth:</u> distance to which a probe penetrates into the pocket. The standardized force used for penetration of probe is 25 pounds or 23 grams (0.75 N).

<u>Probing Pocket depth PPD:</u> Distance between base of pocket and gingival margin.

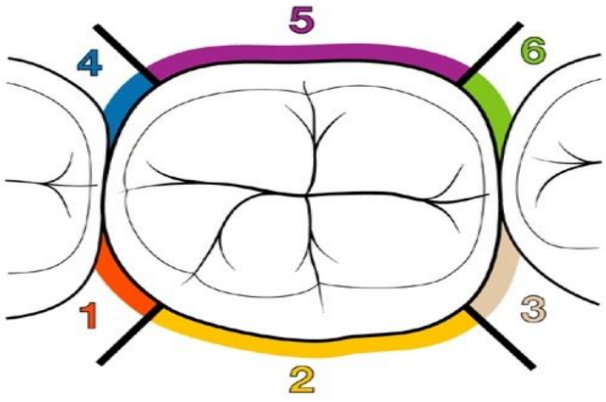


(A) The biologic or histologic pocket depth is the actual distance between the gingival margin and the attached tissues (i.e., the bottom of the pocket). (B) The probing or clinical pocket depth is the depth of prob penetration.

Extent

The "**extent**" of disease refers to the proportion of the dentition affected by the disease in terms of percentage of sites.

Sites are defined as the positions at which probing measurements are taken around each tooth and, generally, six probing sites around each tooth are recorded, **as follows:**



1. Mesiobuccal 2. Mid-buccal 3. Distobuccal 4. Mesiolingual 5. Mid-lingual 6. Distolingual

If up to **30%** of sites in the mouth are affected, the manifestation is classified as **"localized";** for **more than 30%,** the term **"generalized"** is used.

Probing techniques this can reveals the extent of the disease

1- **Occlusal view:** six surfaces measured in periodontal probing. The probe should be **inserted parallel** to the vertical axis of the tooth and walked circumferentially around each tooth to detect the area of deepest penetration.
2- **In multirooted teeth**, the possibility of furcation involvement should be carefully explored with specially designed probe (eg. **Nabers probe**).
3- **To detect internal crater:** the probe should be **placed obliquely** from both facial and lingual surfaces, so as to explore the deepest point of the pocket located beneath the contact point. Periodontal probes are used to locate, measure, and mark pockets, as well as determine their course on individual configuration.

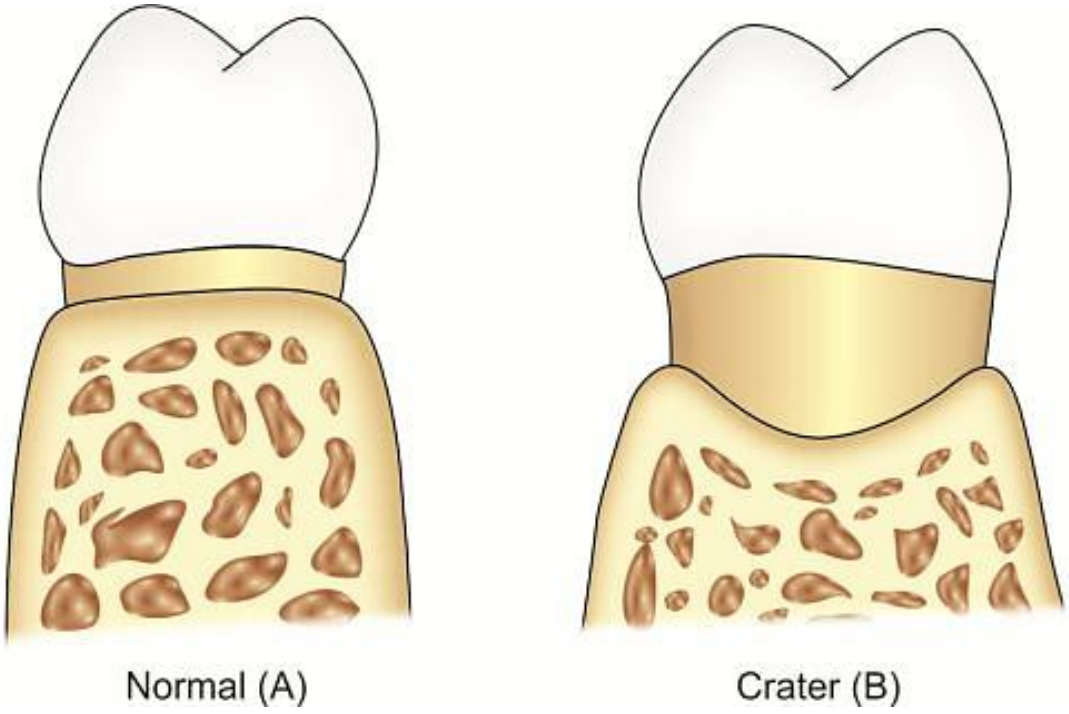


Fig.:- Interdental crater

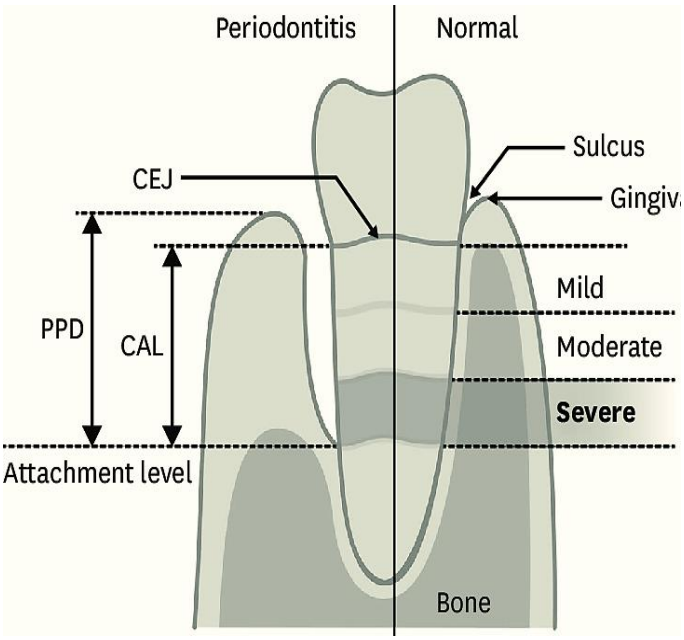
Severity

The **"severity"** of disease refers to the amount of periodontal ligament fibers that have been lost, termed **"clinical attachment loss".**

Level of attachment loss CAL: Distance between base of pocket and a fixed point on the tooth such as CEJ.

According to the American Academy of Periodontology, the classification of severity is as follows:

- **Mild: 1–2 mm of attachment loss**
- **Moderate: 3–4 mm of attachment loss**
- **Severe: ≥ 5 mm of attachment loss**



Classification of periodontal probes

Periodontal probes may be divided into:

First generation probes are conventional , and hand held probes , e.g. conventional periodontal probes. The typical probe is a tapered, rod-like instrument calibrated in millimeters, with a blunt rounded tip.

Second generation probes are pressure –sensitive probes . It has been shown that, with forces up to 30gms the probe tip remains within junctional epithelium and forces up to 50gms are necessary to diagnose osseous defects. This probe did solve many problems of the conventional probes, but lacked tactile sensitivity.

Third generation probes are computerized probes . Gibbs et al designed **Florida probe**. E.g.- **Foster Miller Probe**, **Toronto Automated Probes**, which can detect the cementoenamel junction

Fourth generation probes are the three dimensional probes in which sequential probe positions are measured .They are three dimensional probes

Fifth generation probes are ultra-sonographic probes which provides painless probing to the patient. The guidance path is predetermined in these probes.

Periodontal Probes have blunt, rod shaped working ends that may be circular or rectangular in cross-sections

Types of Calibrated Periodontal Probe

• **Marquis color-coded probe** Calibrations are in 3mm sections, markings are 3,6,9,12mm

• **University of Michigan 'O' probe, with Williams markings**

• **Michigan 'O' probe** Markings include 1,2,3,5,7,8 and 9mm with 4mm and 6mm missing

• **The UNC-15 Probe** 15mm long and markings are at each mm and coding at the **5th ,10th and 15th** mm. Millimeter markings at 1,2,3,4,5,6,7,8,9,10,11,12,13,14 and 15 millimeters.

World Health Organization (WHO) probe Prescribed in 1978. The probe was designed for two purposes: 1- Measurement of pocket depth. 2- Detection of sub gingival calculus. **Used in** the assessment of CPITN (Community Periodontal Index for Treatment Needs).

Naber's Furcation Probe :- It is used to determine the extent of furcation involvement on a multi rooted teeth. It has a curved working end for accessing the furcation area. The end is blunt so that it will not harm soft tissues. Most of the nabers probe do not have markings. The depth of insertion of the probe into the furcation area determines the degree of furcation involvement.

Peri-implant Probing ----- Advantages: Can measure the level of mucosal margin relative to a fixed position on the implant. Measure the depth of tissue around the implant. Periimplant probing depth is often a measure of the thickness of surrounding connective tissue and correlates most consistently with the level of surrounding bone. The probing depth around implants presumed to be **"healthy"** has been **about 3mm** around all surfaces. The results obtained with periimplant probing cannot be interpreted same as the natural teeth ? **Because:** Differences in the surrounding tissues that support implanted teeth. , Probe inserts and penetrates differently. Around natural teeth, the periodontal probe is **resisted by** the insertion of **supra-crestal connective tissue fibers** into the cementum of root surface. **There is no** equivalent fiber attachment around implants

Treatment:

Non-surgical treatment:

1) Oral hygiene instruction.

2) Scaling and root planning

✓ Use curettes for subgingival scaling, root planing and removal of the soft tissue lining the pocket.

✓ Root planing stroke should be moderate to light.

✓ Pull stroke for final smoothing and planing of root surface.

✓ Continuous series of long, overlapping shaving strock is achieved.

✓ To avoid over instrumentation, a delicate transition from short, powerful scaling strokes to longer, lighter root planning strokes must be made as soon as calculus and initial roughness have been eliminated.

✓ Periodontal medication as application of tetracycline's

Surgical treatment

Pocket depth reduction through different surgical procedures :-

1-Gingival curettage

2-Gingivectomy

3-Periodontal flap procedures

4-Osseous surgery & periodontal regeneration procedures

Relationship of attachment loss and bone loss to pocket depth

Clinical attachment loss is the distance between the base of the pocket and cemento-enamel junction ,the severity of the attachment loss is generally but not always correlated with the depth of the pocket. This is because the degree of attachment loss depends on the location of the base of the pocket on the root surface, whereas **pocket depth** is the distance between the base of the pocket and the crest of the gingival margin. Pockets of the same depth may be associated with different degrees of attachment loss , and pockets of different depths may be associated with the same amount of attachment loss .

Extensive attachment and bone loss may be associated with shallow pockets if the attachment loss is accompanied by recession of the gingival margin, while slight bone loss can occur with deep pockets (**gingival pocket**) . **So clinical attachment loss better than pocket depth** in reflection of the degree periodontal destruction.

Area Between Base of Pocket and Alveolar Bone

Normally, the distance between the apical end of the junctional epithelium and the alveolar bone is relatively **constant**. The distance between the apical extent of calculus and the alveolar crest in human periodontal pockets is most constant, having a mean length of **1.97 mm** (±33.16%). The distance from attached plaque to bone is never less than **0.5 mm** and never more than **2.7 mm.**

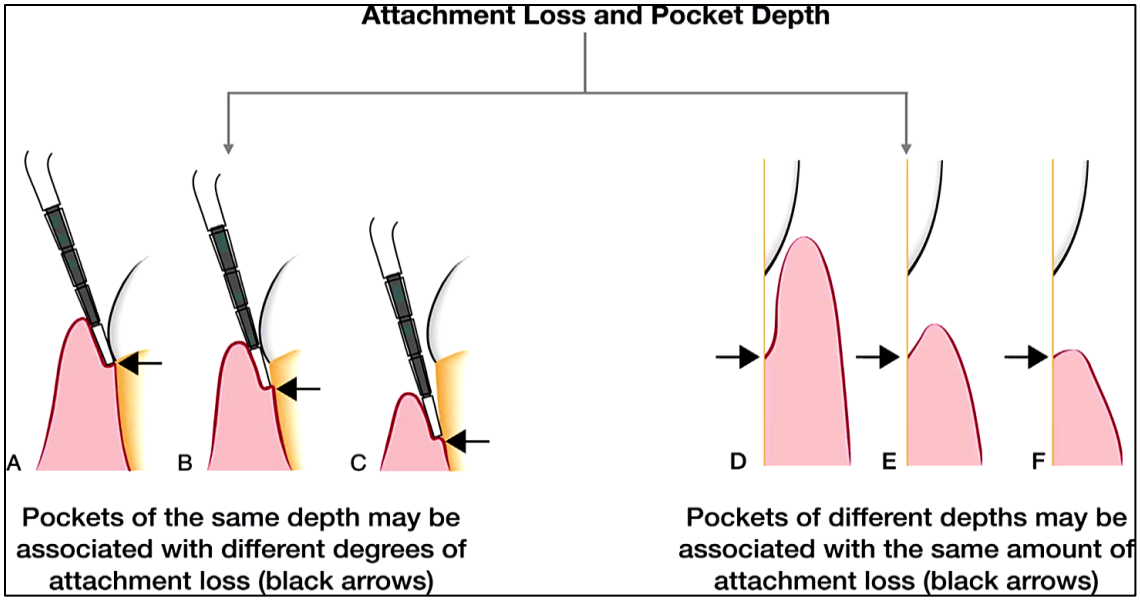


Fig. :- Relationship of attachment loss and bone loss to pocket depth.