
GOOD MORNING

Dr. KANCHAN J

BONE LOSS AND PATTERNS OF BONE DESTRUCTION

CONTENTS:

Introduction:

- Height of the alveolar bone is normally maintained by equilibrium.
- Loss of alveolar bone support is one of the characteristic signs of destructive periodontal disease and is considered to represent the anatomical sequel to the spread of periodontitis.
- A variety of factors other than the main **local factors, ie plaque play a role in bone destruction.**
- Acting **singly or together**, these factors are responsible for bone destruction in periodontal disease and determine its severity and pattern.
- The level of bone is the consequence of past pathologic experiences, whereas changes in the soft tissue of the pocket wall reflect the present inflammatory condition.
- Therefore, the degree of bone loss is not necessarily, correlated with the depth of periodontal pockets, the severity of ulceration of the pocket wall, or the presence or absence of pus.
- Chronic inflammation is the most common cause of bone destruction in periodontal disease, as it results in the extension of the inflammatory process to the bone.
- The extension of inflammation from the marginal gingiva into the supporting periodontal tissues marks the transition from gingivitis to periodontitis

Factors Responsible for Bone Resorption:

Parathyroid Hormone (PTH):

- PTH affects bone cell function, may alter bone remodeling, and cause bone loss.
- PTH acts on **both bone-resorbing cells and bone-forming cells**. The net effect of the hormone depends on whether it is **administered continuously or intermittently.**
- When administered continuously, it **increases osteoclastic bone resorption and suppresses bone formation.** However, when administered in low doses **intermittently, its major effect is to stimulate bone formation, a response that has been called the anabolic effect of PTH.**

Mechanism In Bone Destruction:

- PTH **stimulates osteoclasts to resorb** bone. In **organ cultures, PTH increases osteoclast activity, with resultant degradation of bone matrix**.
- PTH **activates mature osteoclasts to resorb bone, whereas other agents exert their effects by increasing the formation of new osteoclasts.**
- It stimulates mature, multinucleated osteoclasts to form ruffled borders and resorb bone.
- However, the **precise molecular mechanism by which PTH exerts its effects on these cells is still not known.**

1, 25-Dihydroxycholecalciferol (I, 25 (OH) 2 D 3):

- The **active metabolites of vitamin D3 have complex effects on calcium homeostasis and bone regulation**.
- I, 25 (OH)2 D3 stimulates osteoclastic bone resorption in vitro and in vivo.
- **It has a very slow onset of action, with a shallow dose-response curve**.
- **It increases both osteoclast number and activity, with an increase in ruffled border size and clear-zone volume**.
- Mature osteoclasts do not have receptors for I, 25 (OH)2 D3. Thus, the effects of this hormone on mature osteoclasts are most likely mediated indirectly through other cells.
- The major effect of I, 25 (OH)2 D3 on osteoclastic bone resorption may be to stimulate the fusion of differentiation of osteoclast progenitors to form mature cells.
- **Use of I, 25 (OH)2 D3 influences and modulates cytokine production by immune cells**.

Estrogens:

- Estrogen clearly inhibits the increase in bone resorption associated with menopause. Following estrogen withdrawal, an initial increase in bone turnover can be observed.
- Later, bone resorption occurs faster than bone formation, with a net effect of bone loss.
- The effects of estrogen are mediated by a **combination of direct and indirect effects.**
- The **direct effect** is mediated by **specific receptors** found in cells of the **osteoblast and osteoclast lineages**. The effects of **estrogen on osteoclasts** are in **part direct** and in part **mediated through osteoblasts**.
- Some **indirect effects of estrogen** result from its enhancing the **expression of growth factors like insulin like growth factor (IGF-1) and transforming growth factor-β (TGF-β), and of cytokines,** others from its inhibition of prostaglandin production by bone cells.
- Thus, the **major effect of estrogen may be to inhibit bone resorption,** but it may also have the **additional effect of stimulating bone formation.**

Calcitonin:

- Calcitonin has been demonstrated to inhibit osteoclastic bone resorption. The effect of calcitonin on osteoclasts is mediated through cyclic Amp(adenosine monophosphate).
- **Calcitonin decreases osteoclast activity**. The effects of calcitonin on bone resorption are short-lived; however, **osteoclasts eventually lose their responsiveness to calcitonin after continuous exposure, a phenomenon referred to as escape.**
- One explanation for this **phenomenon may involve a decrease in receptor number after long periods of exposure**.
- Another possible explanation is **that, a second population of osteoclasts, which is not responsive to calcitonin, emerges**.
- It is believed that it **inhibits bone resorption transiently when bone turnover is not needed for calcium homeostasis**.

Host and Bacterial Factors Involved in Bone Resorption:

The substance that can induce bone resorption in periodontal disease come from 2 sources: **Bacterial Factors, Host Factors.**

Bacterial Factors:

- Capsular and surface associated material, Lipopolysaccharides, Lipoteichoic acids, Peptidoglycans, Muramyll dipeptide, Lipoprotein.
- Substances from bacteria include Lipopolysaccharides (LPS) from gram negative bacteria, lipoteichoic acid from Actinomyces viscosus, peptidoglycan.
- Muramyll dipeptide (MDP), bacterial lipoprotein and capsular or surface associated material (SAM) from gram- negative bacteria have the potency to cause resorption in vitro. It also varies with each source.

- LPS is 10 times more potent than lipoteichoic acid and capsular material is 1000 times more potent than the 3 materials above.
- There are also differences in effect from different bacterial sources of these materials. In this regard, LPS from Porphyromonas gingivalis is more active than, those from Actinobacllus actinomycetemcomitans, Capnocytophaga orchracea or Fusobacterium nucleatum.
- Capsular material or surface associated material (SAM) stimulates the production of PGE2 and collagenase from bone cells.
- The surface associated material from Prophyromonas gingivalis and Eikenella corrodens appear to achieve this by first releasing IL-1, which then stimulates the production of PGE2 and collagenase.

Host Factors:

- Inflammatory mediators: PGE2, Leukotrienes, Bradykinin, Cytokines, Interleukin-1, Interleukin– 6, Tumor Necrosis Factor.
- In recent years, it has become increasingly clear that many of the cellular events involved in bone resorption are modulated by a group of local factors .
- Effects of local factors on bone resorption appear to be overlapping and are seemingly redundant; for example, Interleukin-1 (IL-1), tumor necrosis factor-alpha (TNF-a), and lymphotoxin appear to affect bone resorption similarly.

Interleukin-1 (IL-1):

- **IL-1 is a powerful and potent bone-resorbing cytokine**. It has been found that IL-1α and IL-1 β are equally potent in stimulating bone resorption and probably exert their effects on bone-resorbing cells in several ways.
- They **stimulate proliferation of precursor cells, but also probably act indirectly on mature cells to stimulate bone resorption**.
- The **effects of IL-1 probably occur by two mechanisms**.
- One mechanism is the **stimulation of the production and release of PGE2, there by stimulating bone.**
- The second mechanism involves **direct action of IL-1 on osteoclasts independent of PGE2 synthesis by 80 kda receptor**.

Interleukin-6 (IL-6):

- In some experimental models, IL-6 appears to have no effects on bone resorption.
- However, in others, it stimulates bone resorption.
- IL-6 is also responsible for the formation of cells with an osteoclastic phenotype.
- Bone cells also have the ability to produce IL-6, which seems to be greater when the stimulus is by another cytokine.

Tumor necrosis factor (TNF) and Lymphotoxin:

- Lymphotoxin and TNF are two closely related cytokines that have equivalent effects on bone cells.
- They are both multi-functional cytokines produced by activated Lymphocytes, and they share the same receptor.
- Their major effect on bone is to stimulate osteoclastic bone resorption.
- It has been suggested that part of the effect of TNF is mediated by PGE2, as well as by IL-6.
- TNF also affects cells with osteoblast phenotypes and inhibits differentiated function and stimulates cell proliferation.
- Production of TNF in some tumors, like squamous cell carcinomas, may be responsible for paraneoplastic syndromes.

Gamma interferon (IFN-γ):

- Gamma interferon is a multi-functional cytokine which has effects similar to TNFα or IL-1 in most biological system.
- However, it has an effect on bone resorption that is opposite to that of IL-1 and TNF.
- Gamma interferon is more effective in inhibiting IL-1 or TNF-α than systemic hormones like PTH or 1,25-(OH)2 D3.
- Further, it has been found in long-term marrow cell cultures that gamma interferon inhibits the formation of cells with the osteoclast phenotype.

Colony stimulating factors (CSF3):

- CSF has ability to stimulate differentiation of osteoclast precursors info mature osteoclasts.
- Recently, it was found that there are a number of human and animal tumors associated with granulocytosis in which increased production of CSF3 is involved.
- In many of these tumors, hypercalcemia is associated with increased bone resorption.
- It is possible that CSF3 mediate their effects on osteoclast formation indirectly. For example, early studies showed that CSF stimulates IL-I production, which stimulates prostaglandin synthesis

Prostaglandin and other arachidonic-acid metabolites:

- A number of arachidonic-acid metabolites act as modulators of bone-cell function. These factors are produced by immune, marrow, and bone cells.
- PGEs are slow acting, but powerful mediators of bone resorption and affect both active mature osteoclasts, as well as differentiated osteoclast precursors.
- The effect of PGE is local and has been shown to mediate the effects of other factors like epidermal growth factor (EGF) and transforming growth factor-β (TGF-β).
- PGE is produced by osteoblasts and has effects not just on bone resorption, but on bone formation as well.
- In vitro, it has been found that high doses of PGE are inhibitory, while low doses stimulate bone formation.
- However, in vivo it appears that the effect of PGE is clearly associated with an increase in periosteal bone formation.
- Arachidonic acid can be metabolized by an alternative enzyme system, 5-lipoxygenase, which also produces metabolites capable of stimulating bone resorption.

Other Products of Inflammation:

- Heparin from mast cells can enhance bone resorption in tissue culture system induced by LPS and lipoteichoic acid, but cannot induce bone resorption on its own.
- Thrombin, an inflammatory mediator and end product of the blood coagulation cascade is a potent bone-resorbing agent.
- Another inflammatory agent, bradykinin, evokes similar effects and it is independent of prostaglandin production.

ETIOLOGY OF BONE LOSS:

INFLAMMATION:

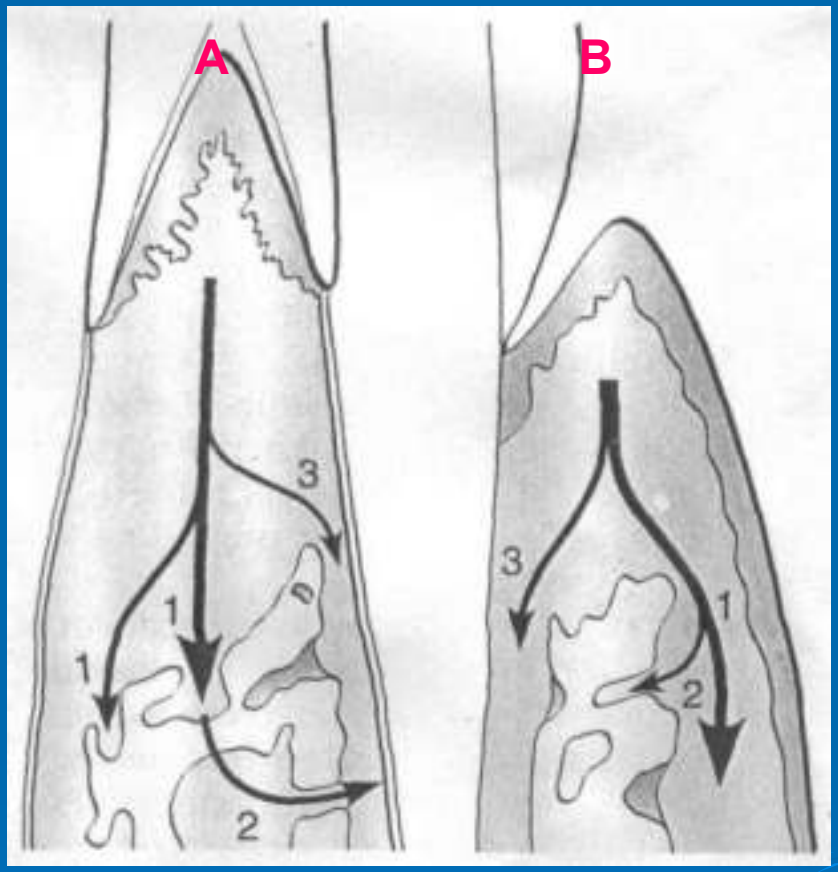
- The extension of inflammation to the supporting structures of a tooth may be modified by the pathogenic potential of plaque or by the resistance of the host.
- The latter includes immunologic activity and other tissue-related mechanisms, such as the degree of fibrosis of the gingiva, probably the width of the attached gingiva, and the reactive fibrogenesis and osteogenesis that occur peripheral to the inflammatory lesion.
- A fibrin- fibrinolytic system has been mentioned as "walling off' the advancing lesion.
- The pathway of the spread of inflammation is critical because it affects the pattern of bone destruction in periodontal disease.

Pathway of Inflammation:

Pathways of inflammation from the gingiva into the supporting periodontal tissues in periodontitis.

A, Interproximally, from the gingiva into the bone (1), from the bone into the periodontal ligament (2), and from the gingiva into the periodontal ligament (3).

B, Facially and lingually, from the gingiva along the outer periosteum (1), from the periosteum into the bone (2), and from the gingiva into the periodontal ligament (3).



- After inflammation reaches the bone by extension from the gingiva, it spreads into the marrow spaces and replaces the marrow with a leukocytes, fluid exudate, new blood vessels, and proliferating fibroblasts.
- Multinuclear osteoclasts and mononuclear phagocytes are increased in number, and the bone surfaces are lined with resorption lacunae.
- In the marrow spaces, resorption proceeds starts from within, causing first a thinning of the surrounding bony trabeculae and enlargement of the marrow spaces, followed by destruction of the bone and a reduction in bone height.
- Normally fatty bone marrow is partially or totally replaced by a fibrous type of marrow in the vicinity of the resorption.

- Bone destruction in periodontal disease is not a process of bone necrosis. It involves the activity of living cells along viable bone.
- When tissue necrosis and pus are present in periodontal disease, they occur in the soft tissue wall of periodontal pockets, not along the resorbing margin of the underlying bone.
- The amount of inflammatory infiltrate correlates with the degree of bone loss but not with the number of osteoclasts.
- However, the distance from the apical border of the inflammatory infiltrate to the alveolar bone crest correlates with both the number of osteoclasts on the alveolar crest and the total number of osteoclasts.

Radius of Action:

- Locally produced **bone resorption factors may have to be present in the proximity of the bone surface to be able to exert their action.** Page and Schroeder (1982), on the basis of
- Waerhaug's (1980) measurements made on human autopsy specimens, = postulated that there is a range of effectiveness of about 1.5 to 2.5 mm within which bacteria/ plaque can induce loss of bone.
- Beyond 2.5 mm there is no effect; **interproximal angular defects can appear only in spaces wider than 2.5 mm because narrower spaces would be destroyed entirely.**
- Tal (1984) corroborated this with measurements in human patients. Large defects far exceeding 2.5 mm from the tooth surface may be caused by the presence of bacteria in the tissues.

Mechanisms of Bone Destruction:

- Many investigations have been conducted and many explanations considered, but the mechanism by which inflammation and/or plaque-derived products destroy bone in inflammatory periodontal, disease have not yet been determined.
- There are several possible pathways by which products in plaque absorbed by periodontal tissues could cause alveolar bone loss (Hausman, 1974).
- 1. **Absorbable products from plaque** could **stimulate bone progenitor cells in the periodontium to differentiate into osteoclasts, which resorb alveolar bone.**
- 2. Absorbable products from plaque as, for example, complexing agents and hydrolytic enzymes could destroy alveolar bone through **non-cellular mechanism by dissolving bone mineral and hydrolyzing the organic matrix**.

- 3. a) Absorbable products from plaque could stimulate cells within the gingiva to release mediators, which in turn could trigger bone progenitor cells to differentiate into bone resorbing osteoclasts.
- b) Gingival cells in response to plaque products could release agents which by themselves have no effect on bone, but could potentate as co-factors other bone resorptive agents.
- c) Gingival cells could release agents, which destroy bone by direct chemical action without osteoclasts.

Periods of Destruction:

- Periodontal destruction occurs in an episodic, intermittent fashion, with periods of inactivity or quiescence. The destructive periods result in loss of collagen and alveolar bone with deepening of the periodontal pocket.
- 1. Bursts of destructive activity are associated with subgingival ulceration and an acute inflammatory reaction, resulting in rapid loss of alveolar bone.
- 2. Bursts of destructive activity coincide with the conversion of a predominately T- lymphocyte lesion to one with predominance of B lymphocyte-plasma cell infiltrate.
- 3. Periods of exacerbation are associated with an increase of the loose, unattached, motile, gram-negative, anaerobic pocket flora, and periods of remission coincide with the formation of a dense, unattached, non-motile, gram-positive flora with a tendency to mineralize.
- 4. Tissue invasion by one or several bacterial species is followed by an advanced local host defense that controls the attack.

BONE LOSS INPERIODONTITIS:

CHRONIC PERIODONTITIS:

- The characteristic findings in slowly progressive periodontitis are gingival inflammation, which results from the accumulation of plaque, and loss of periodontal attachment and alveolar bone, which results in formation of a pocket.
- **Pocket depths are variable**, and **both horizontal and angular bone loss can be found.**
- **Tooth mobility** often appears in **advanced cases** when bone loss has been considerable.
- **Radiographically bone loss is usually horizontal**

AGGRESSIVE PERIODONTITS:

- Clinically, there is a **small amount of plaque**, which forms a thin bio-film on the tooth and rarely **mineralizes to become calculus**.
- The most common initial symptoms are **mobility of the first molars and incisors, distolabial migration of the incisors.**
- Bone loss is about **3-4 times faster than in chronic periodontitis.**
- The progression of **bone loss and attachment loss may be self-arresting.**
- **Vertical loss of alveolar bone** around the first molars and incisors is seen. An **"arc-shaped" loss of alveolar bone extending from the distal surface of the second premolar to the mesial surface of the second molar**.

TRAUMA FROM OCCLUSION:

- Trauma from occlusion can produce **bone destruction in the absence or presence of inflammation**.
- In the **absence** of inflammation, the changes caused by trauma from occlusion **vary** from **increased compression and tension of the periodontal ligament and increased osteoclasts of alveolar bone, necrosis of the periodontal ligament and bone, and resorption of bone and tooth structure.**
- Trauma from occlusion results in **funnel-shaped widening of the crestal portion of the periodontal ligament.**

FOOD IMPACTION:

- Interdental defects often occur where **proximal contacts is abnormal or absent.**
- Pressure and irritation from food impaction **contributes to inverse architecture**.
- In some instances the **poor proximal relation ship** may be the result of shift in bone position because of extensive bone destruction preceding food impaction.
- In such cases food impaction is the complicating factor rather than the cause of bone destruction.

SMOKING:

- Various studies recently have advocated and proven the adverse effect of smoking on periodontium and subsequently on bone.

STRESS:

There are many forms of stress, such as trauma, drug intoxication, and muscular fatigue that may compromise the health of an individual.

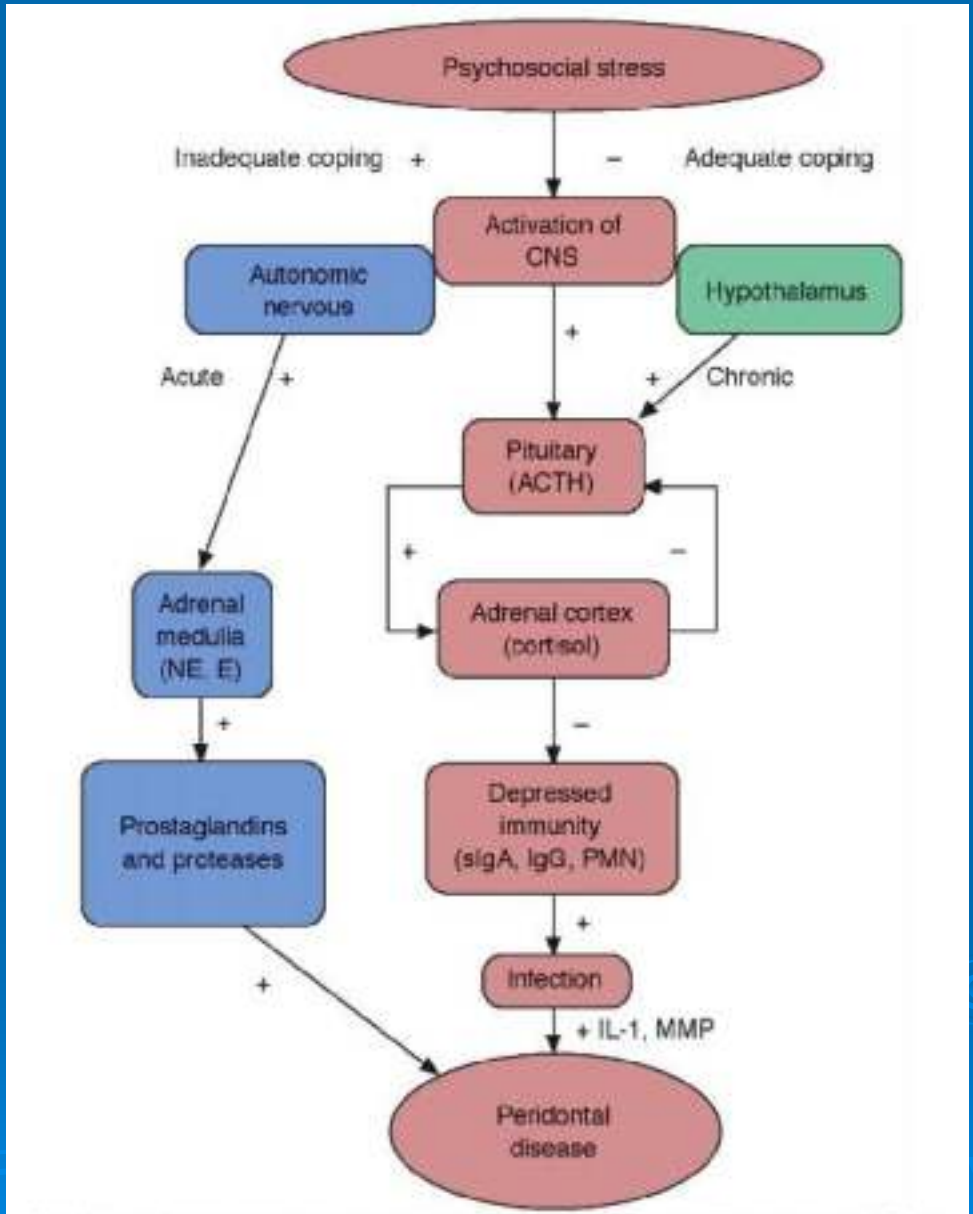
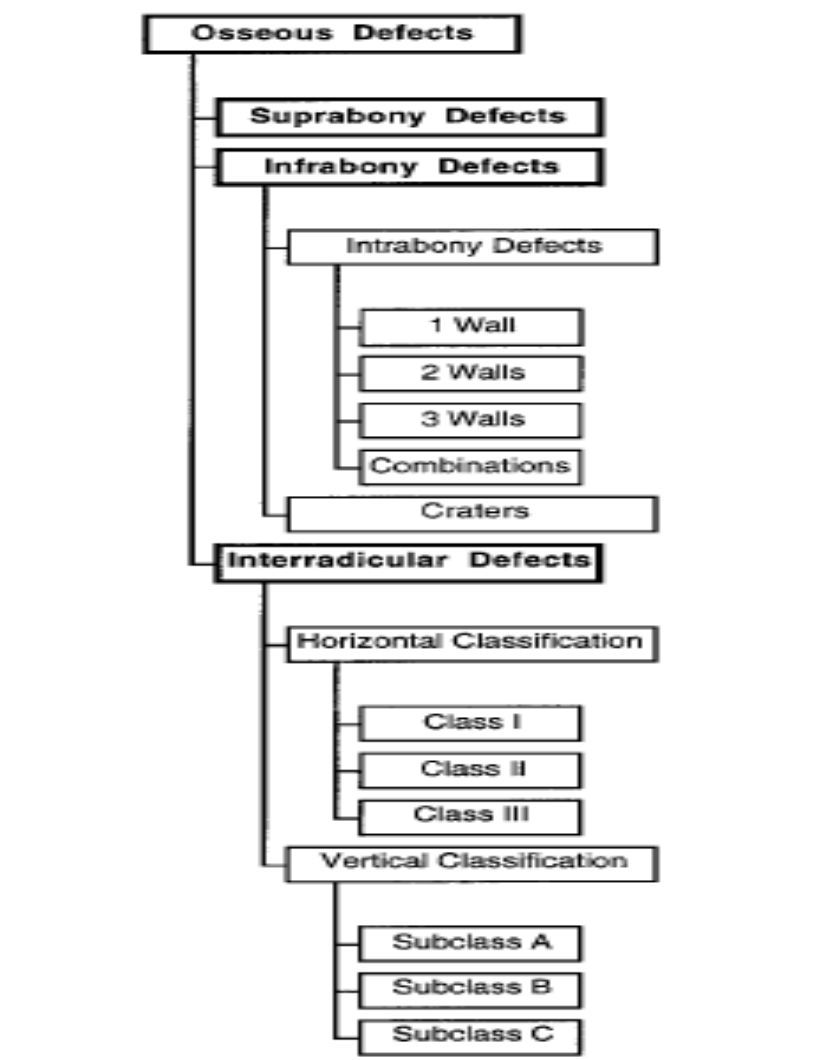


Fig. 3. Model 1. A physiological model for the effects of stress on periodontal diseases: CNS, central nervous system; CRH, corticotropic releasing hormone; ACTH, adrenocorticotropic hormone; NE, norepinephrine; MMP, matrix metalloproteinase; [reproduced with kind permission from ref. (24)].

BONE FACTOR CONCEPT:

- The **focal and systemic factors regulate the physiologic equilibrium of bone.**
- When there is general tendency toward bone resorption, bone loss initiated by local inflammatory processes may be magnified.
- This **systemic influence** on the **response of alveolar bone has been termed the bone factor in periodontal disease.**
- The bone factor concept, developed by **Glickman (1951), envisioned a systemic component in all cases of periodontal disease.**
- In addition to the **amount and virulence of plaque bacteria, the nature of the systemic component, not its presence or absence, influences the severity of periodontal destruction**.
- Although, the term bone factor is not in current use, the concept of a role played by systemic defense mechanisms has been validated, by various studies.

Classification of Periodontal OSSEOUS DEFECTS:



Classification of periodontal osseous defects

Pritchard (1965) classification:

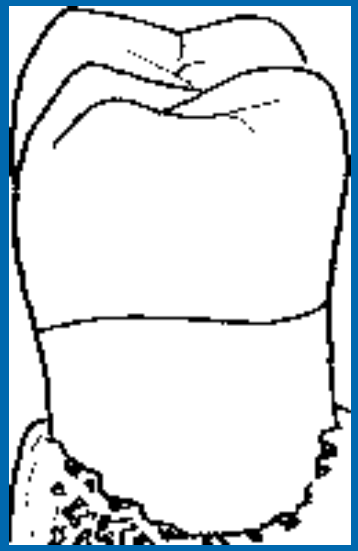
- Interproximal craters.
- Inconsistent margins.
- Hemisepta.
- Furcation involvement.
- Intra bony defect (with three osseous walls).
- Combination of above.
- Fenestration.
- Dehiscence.

Glickman (1964):

- Osseous craters.
- Intra bony defect.
- Bulbous bony contours.
- Hemisepta.
- Inconsistent margins.
- Ledges.

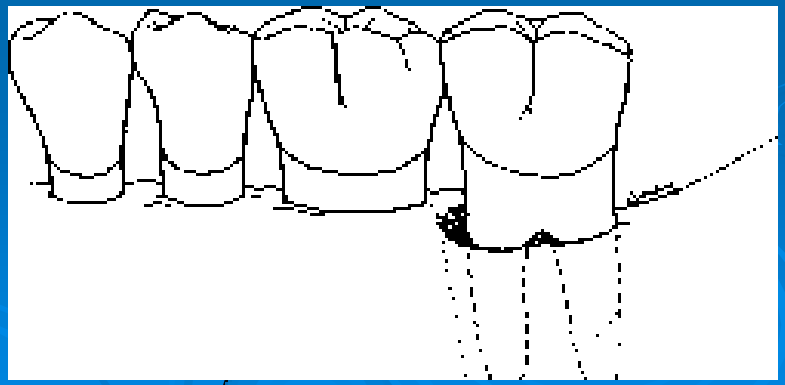
Nomenclature of deformities of the alveolar process:

The proposed system of nomenclature for bony deformities caused by non-uniform loss of bone is based on the following basic terms:

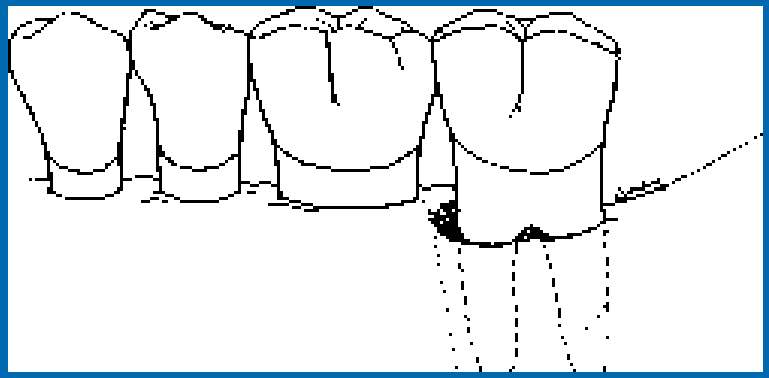


- ***OSSEOUS CRATERS:***

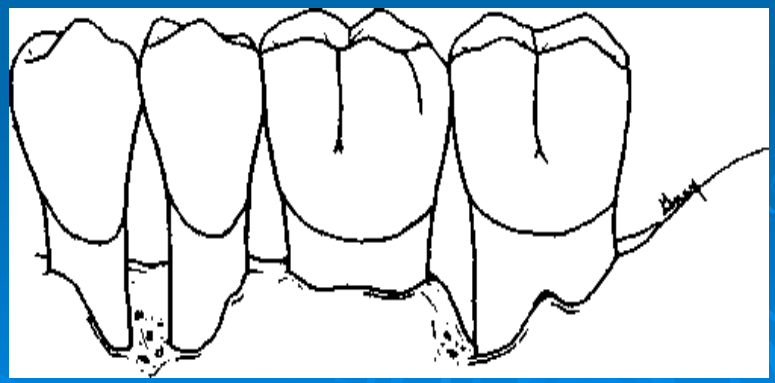
- ***TRENCH:*** This term is applied when such **bone loss affects two or more three confluent surfaces of the same tooth**.



- **MOAT:** When the previously described deformity **involves all four surfaces of tooth** it is described as a moat.

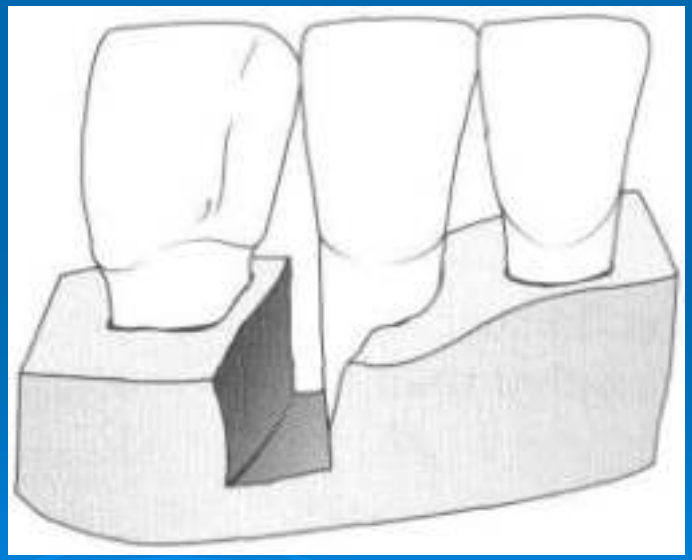


- **RAMP:** In its purest form the term ramp describes **deformity that results when both alveolar bone and its supporting bone are lost** to the **same degree** in such a manner that the **margins of the deformity are at different levels.**

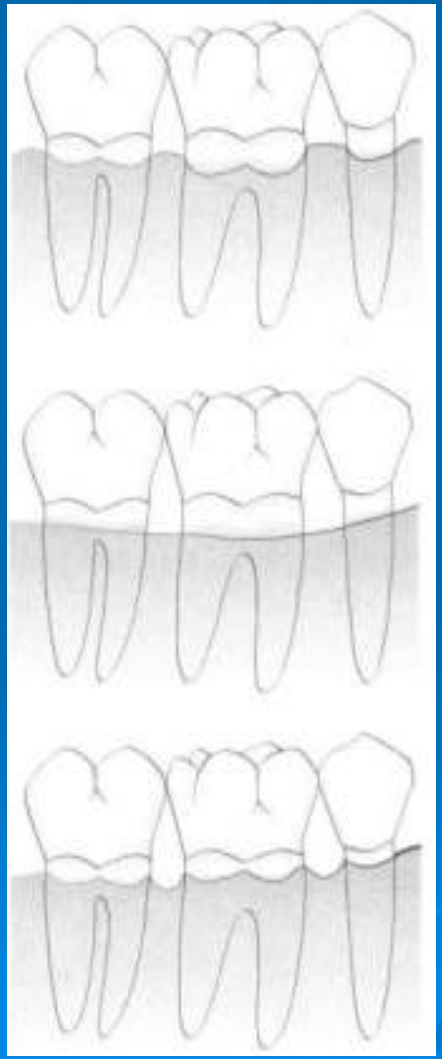


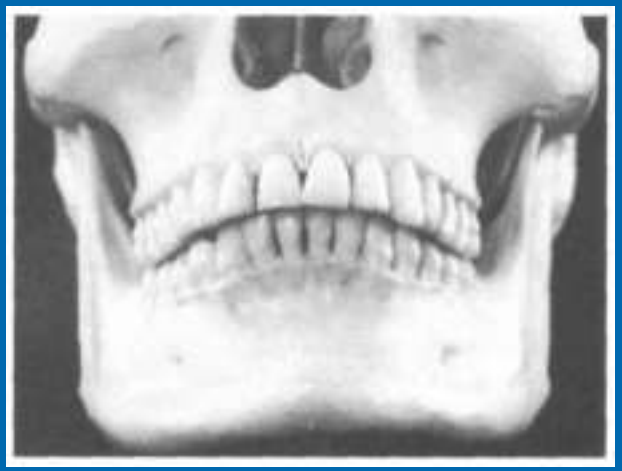
- **PLANE:** This term is applied **when both alveolar bone and supporting bone is lost to the same degree such that the margins of the deformity are at the same level.** It can be considered **horizontal bone loss about one tooth or portion of a tooth.**

- HEMISEPTA: A **hemiseptum often is associated with an inconsistent osseous margin.** Hemisepta occur between **anterior** as well as **posterior teeth, found in combination with all other types of bony deformities.**

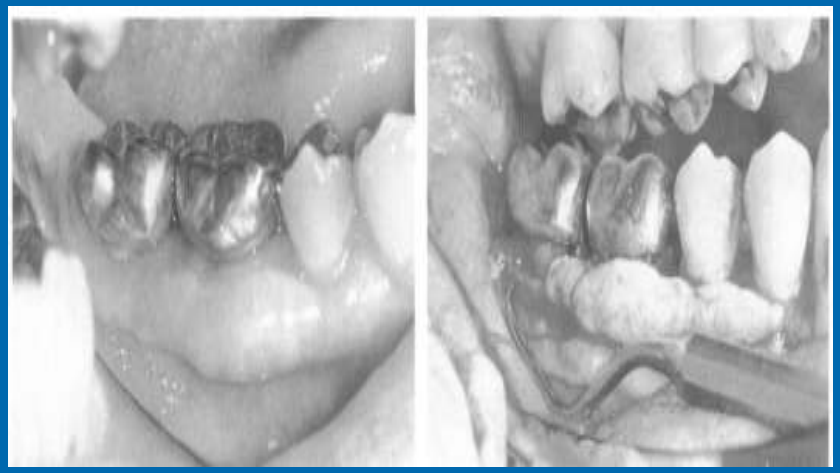


- **REVERSED ARCHITECTURE:** These defects are produced by **loss of interdental bone, including the facial and/or lingual plates**, without concomitant loss of radicular bone, thereby reversing the normal architecture. Such defects are more common in the maxilla.

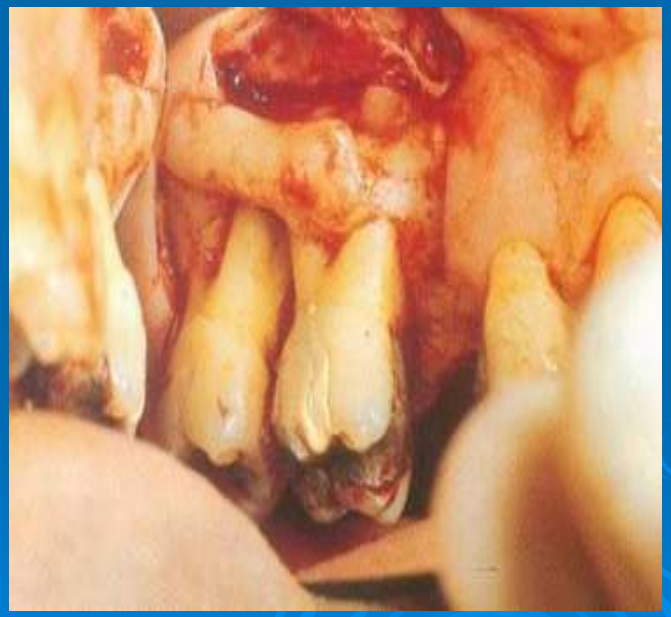




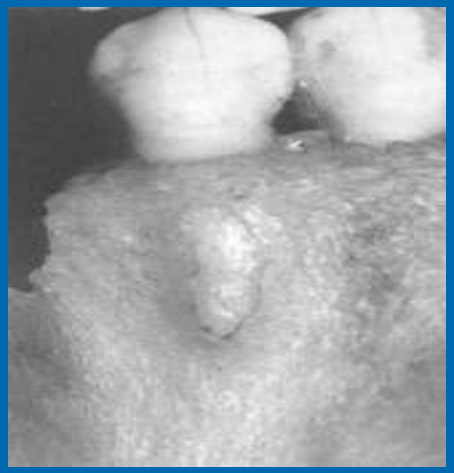
- **LEDGES:** **Ledges are plateau-like bone margins caused by resorption of thickened bony plates.**

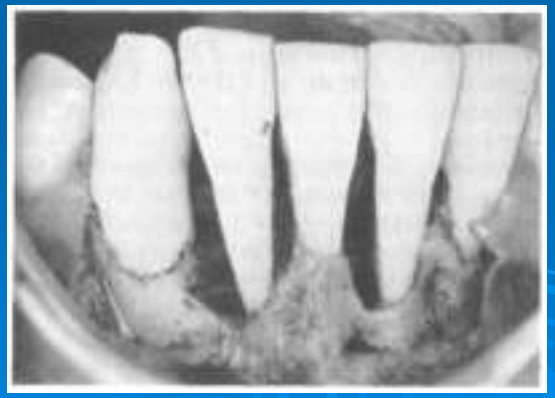


- **BULBOUS BONE CONTOURS:** These are bony enlargements caused by **exostoses, adaptation to function or buttressing bone formation.** They are found more frequently in the **maxilla** than in the mandible.



- **FENESTRATIONS AND DEHISCENCE:**





FURCATION INVOLVEMENT:

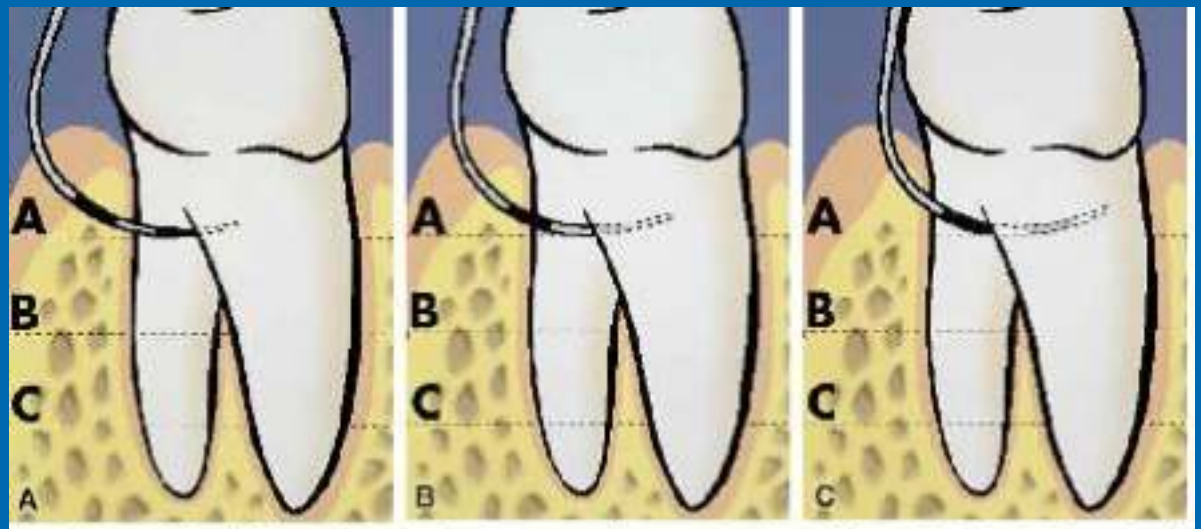


Fig. 3. Horizontal classification of furcation involvements. **A.** Class I, less than 3 mm of horizontal attachment loss. **B.** Class II, more than 3 mm of horizontal attachment loss but not through and through. **C.** Class III, through and through furcation involvement.

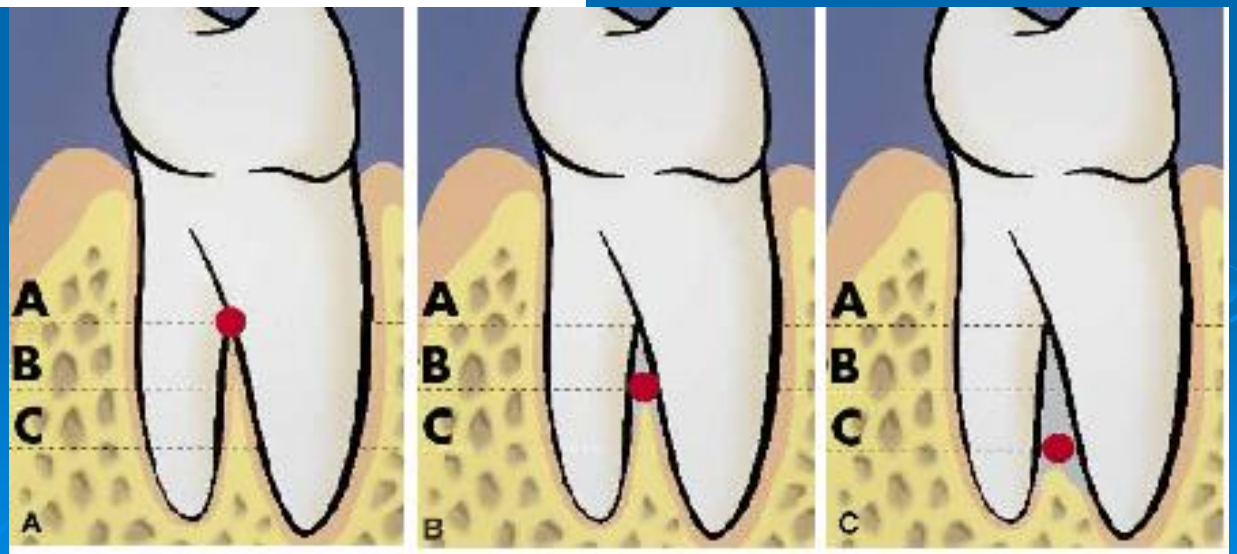
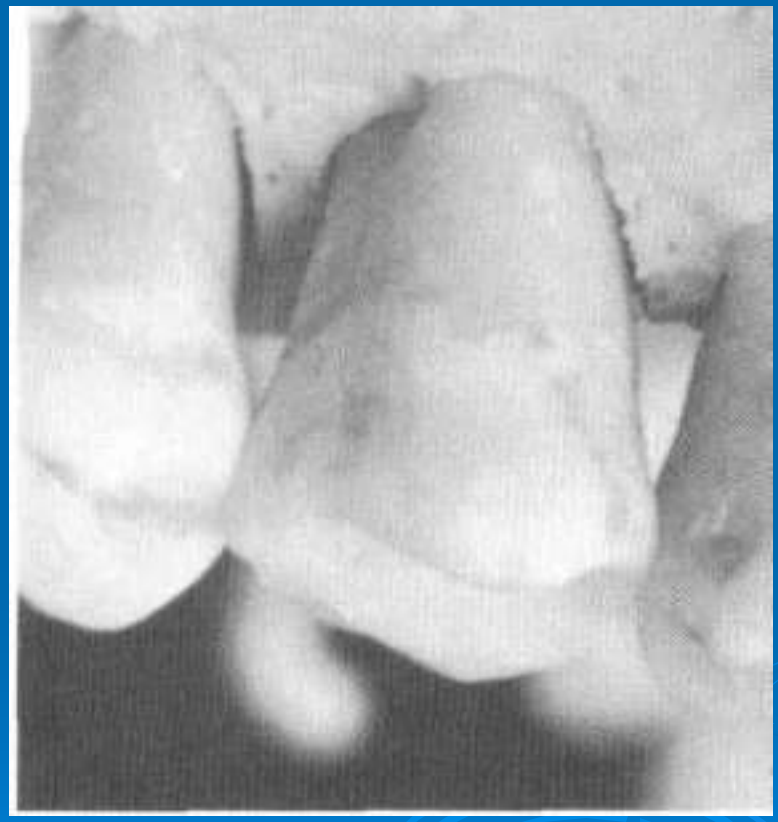


Fig. 4. Vertical classification of furcation involvements. **A.** Subclass A denotes furcation involvements with vertical bone loss of 3 mm or less. **B.** Subclass B denotes furcation involvements with vertical bone loss of 4 to 6 mm. **C.** Subclass C denotes furcation involvements with vertical bone loss from the fornix of 7 mm or more.

- **THICKENED MARGIN:** An **enlargement of facial or lingual marginal alveolar plate, instead of a thin, tapering, slightly rounded bony margin**. Irregular bone margin is seen where there are abrupt irregularities in the scalloped level of marginal bone and interdental septa.

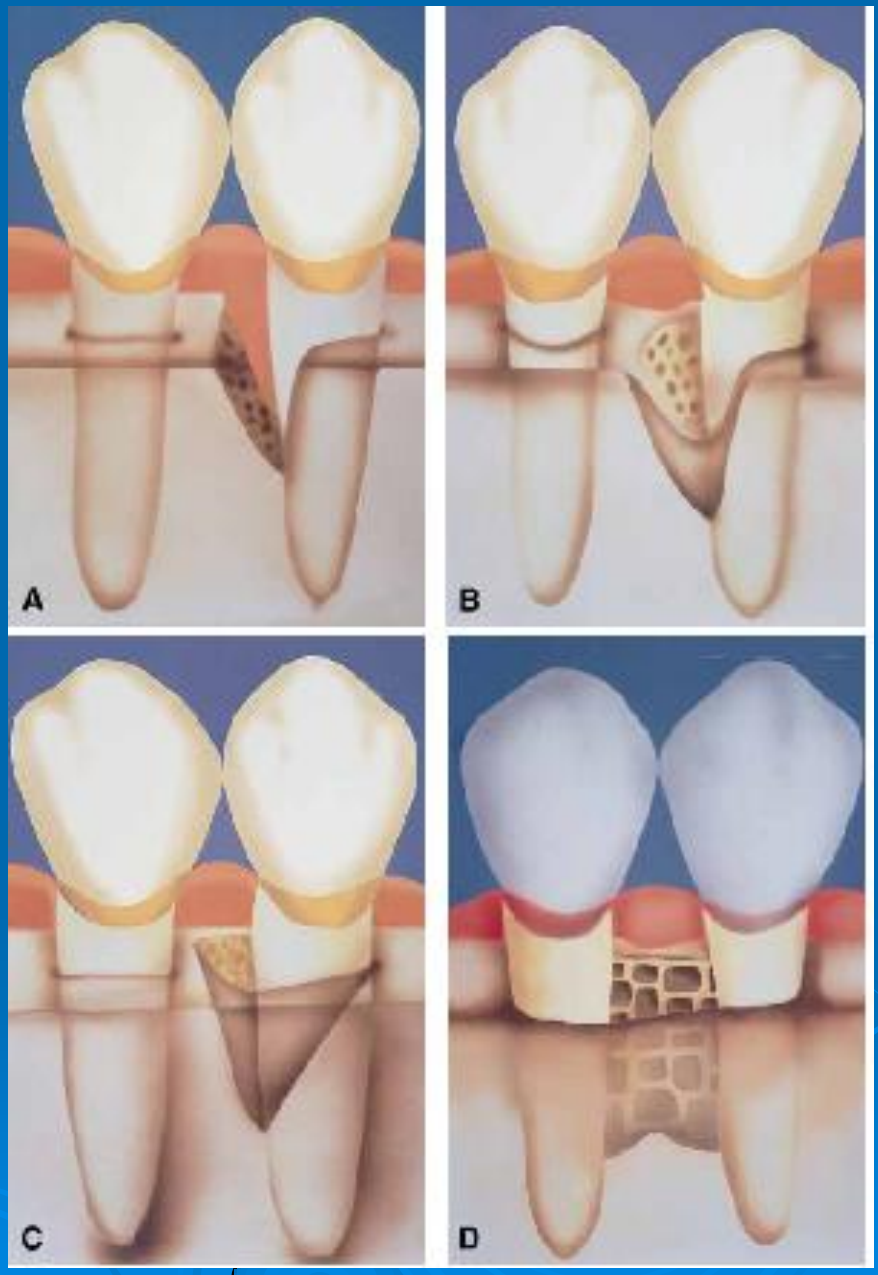
- **MARGINAL GUTTER:** A **shallow linear defect between marginal bone of the radical cortical plate or interdental crest, extending the length of one or more root surfaces, usually formed by resorption of the socket side of the plate and deposition on the facial surface.**

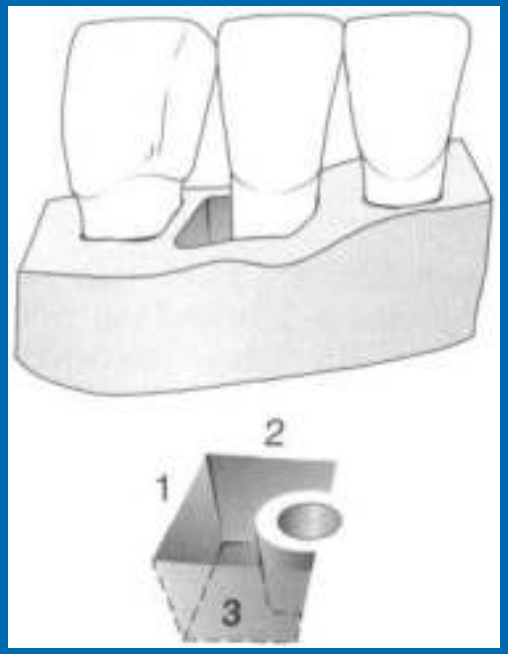
- **HORIZONTAL BONE LOSS:** This is the most common pattern of bone loss in periodontal disease the bone is reduced in height, but the **bone margin remains roughly perpendicular to the tooth surface.**
- The **interdental septa and facial and lingual plates are affected, but not necessarily to an equal degree around the same tooth**.



- **VERTICAL BONE LOSS OR ANGULAR DEFECTS:** Vertical or angular defects are those that occur in an oblique direction, leaving a hollowed-out through in the bone along side the root; the base of the defect is located apical to the surrounding bone.

Fig. 2. Infrabony defects. A. One-wall infrabony defect. B. Two-wall infrabony defect. C. Three-wall infrabony defect. D. Interproximal crater.

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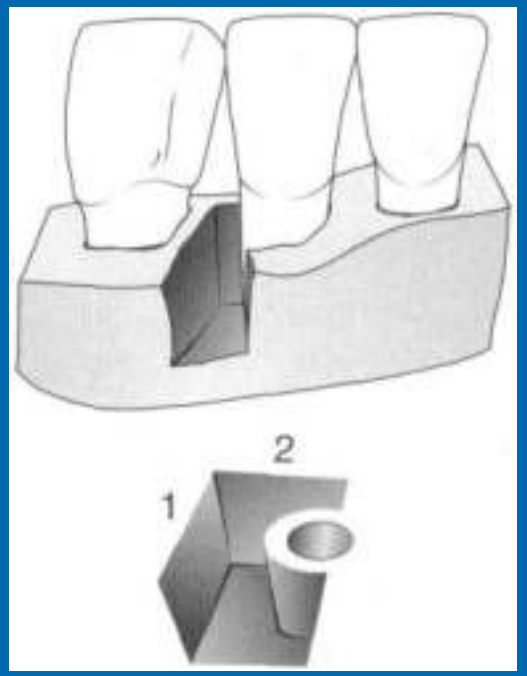
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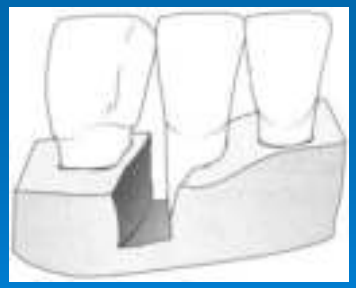

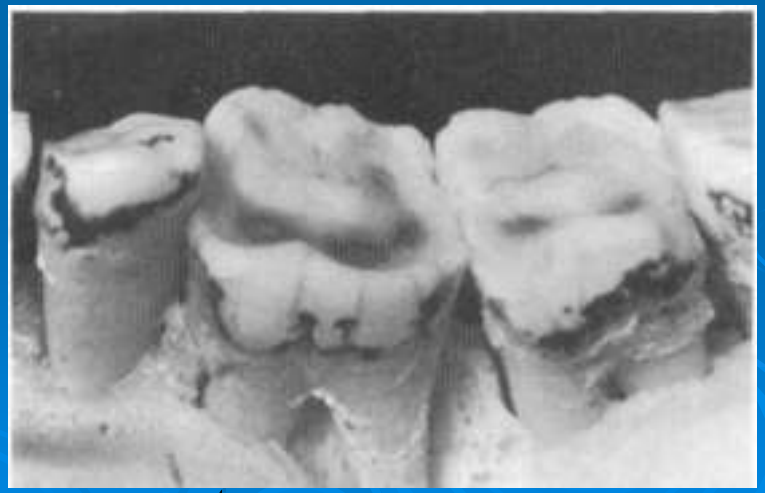

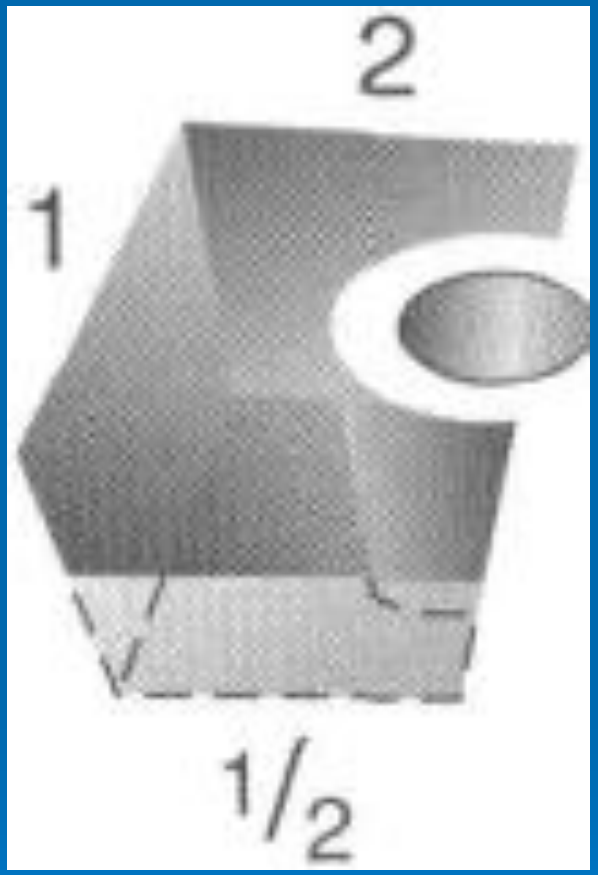
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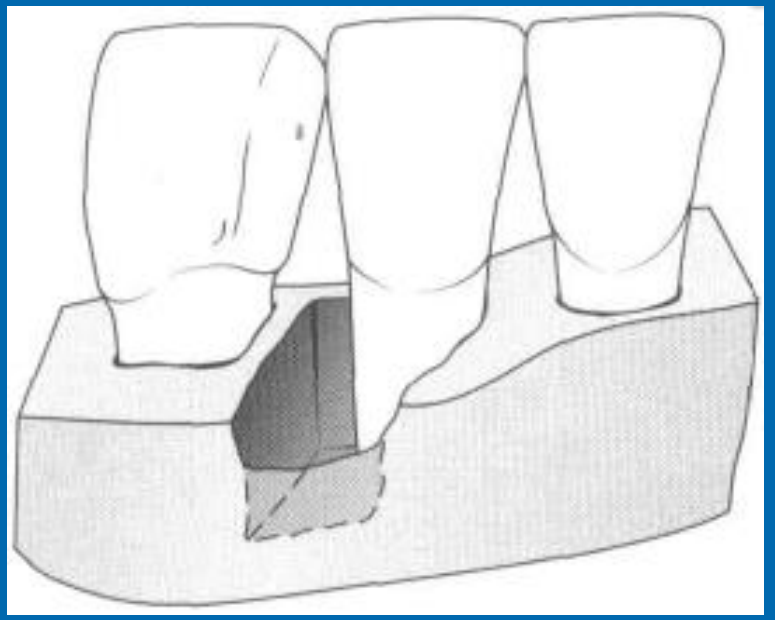

DIAGNOSIS:

CLINICAL:

- **Periodontal probing and exploration** are key aspects of clinical examination. Careful probing reveals the presence of pocket depth greater than that of a normal gingival sulcus.
- The **location of the base of the pocket in relative to the mucogingival junction** and **attachment level on adjacent teeth, the number of bony walls and the presence of furcation defects.**
- **Transgingival probing, or sounding, under local anesthesia confirms the extent and configuration of the intrabony component of the pocket or** of furcation defects.
- The probe should be **"walked"** along the tissue-tooth interface, so as to feel the bony topography.
- The probe may also be passed horizontally through the tissue to provide three-dimensional information regarding bony contours.

RADIOGRAPHIC DIAGNOSIS:

- Dental radiographs are the **traditional** method used to assess the destruction of alveolar bone associated with **periodontitis**.
- Although radiographs cannot accurately reflect the bony morphology buccal and lingual, they provide **useful information on interproximal bone levels**.
- However, even at this level, the exact topography of defects cannot be assessed accurately from radiographs.
- Various methods of radiographic diagnosis include photodensitometric analysis digital radiography, subtraction radiography, CADIA, , nuclear medicine.

Conclusion:

- "Prevention is better than cure" so there is a need for early diagnosis and identification of these factors such as local factors, TFO, Smoking, Stress, Systemic Disorders etc prevention of which would further prevent progression of the disease".

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