Lec 9

Bone Loss and Patterns of Bone Destruction

Periodontitis is an inflammatory condition of the teeth and their supporting structures. The inflammatory process has detrimental effects on the periodontal unit, which results in the destruction of periodontal ligament fibers and bone loss.

The height and density of the alveolar bone are normally maintained by an equilibrium, which is regulated by local and systemic influences, between bone formation and bone resorption.

When resorption exceeds formation, both bone height and bone density may be reduced. Bone loss is the **ultimate and last** consequence of the inflammatory process observed in periodontitis. Therefore the existing bone level is the consequence of past pathologic :episodes, whereas changes present in the soft tissue of the pocket wall reflect the presence of the inflammatory condition

Thus the degree of bone loss does not necessarily correlate with the depth of periodontal pockets, the severity of ulceration of the pocket wall, or the presence or absence of suppuration. As an example, a reduced periodontium may exist in areas where bone loss occurred in the past but that currently present with periodontal health (i.e., following periodontal treatment).

Bone Destruction Caused by the Extension of Gingival Inflammation

The most common cause of bone destruction in periodontitis is the extension of inflammation from the marginal gingiva into the supporting periodontal tissues. The ,inflammatory migration of the bone surface and the initial bone loss that follows, mark the transition from gingivitis to periodontitis.

Periodontitis is always preceded by gingivitis, but not all gingivitis progresses to periodontitis. Some cases of gingivitis apparently never become periodontitis, and other cases go through a brief gingivitis phase and rapidly develop into periodontitis. The factors

responsible for the extension of inflammation to the supporting structures, thereby initiating the conversion of gingivitis to periodontitis, are not clearly understood and are likely to be related to individual susceptibility to the insult presented by the bacterial biofilm or microbiologic changes that occur in the pocket environment and surrounding tissues.

The extension of the inflammatory process into the supporting structures of a tooth may be modified by the pathogenic potential of biofilm and the susceptibility/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.

The quality of the host response to a similar bacterial insult varies, resulting in some individuals being more susceptible to the destructive aspects of periodontitis than others.

Bone destruction in periodontal disease is not a process of bone necrosis.

It involves the activity of living cells along viable bone. With the exception of necrotic bone that is visible in distinct pathogenic processes such as necrotizing ulcerative periodontitis and bisphosphonate-related osteonecrosis of the jaws,

all bone present in areas with periodontitis is viable, live bone. In periodontitis, bone resorption may be related to the analogy of the bone attempting to run away from the infectious/inflammatory process; this may be seen as a host protection mechanism.

Page and Schroeder, postulated a range of effectiveness of about **1.5 mm 2.5** mm in which bacterial biofilm can induce loss of bone

Rate of bone loss:

The rate of bone loss vary depending on the type of periodontal disease present. Loe and colleagues identified three subgroups of patients with periodontal disease based on the interproximal loss of attachment and tooth mortality

1. **8%** of persons had a **rapid progression** of periodontal disease with a yearly loss of attachment of **0. l- l mm**

2. **80%** of individuals had **moderately progressive** periodontal disease with a yearly loss of attachment of **0.05- 0.5 mm**

3. **11%** of persons had **minimal or no progression** of destructive disease with a yearly loss of attachment of **0.05-0.09mm**

Mechanisms of Bone Destruction

The factors involved in bone destruction in periodontal disease are bacterial and host mediated

- The bacterial biofilm products induce the differentiation of bone progenitor cells into osteoclasts.
  - bacterial biofilm products stimulate gingival cells to release mediators that induce the differentiation of bone progenitor cells into osteoclasts.
  - bacterial biofilm products and inflammatory mediators can also act directly on osteoblasts or their progenitors, thereby inhibiting their action and reducing their numbers.
  - In addition, in patients with rapidly progressing periodontal diseases bacterial micro- colonies or single bacterial cells have been found between collagen fibers and over the bone surface, suggesting a direct effect
  - Inflammatory cells release host factors capable of inducing bone resorption like prostaglandins and their precursors, interleukin-1α interleukin-β and tumor necrosis factor alpha.

Bone Destruction Caused by Trauma From Occlusion

Trauma from occlusion can occur 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 osteoclasis of alveolar bone to necrosis of the periodontal ligament and bone and the resorption of bone. These changes are reversible in that they can be repaired if the offending forces are removed.

* In the absence of inflammation (plaque), trauma from occlusion leads to angular defects without pocket formation.

* In the presence of existing periodontitis (local factors), trauma from occlusion may aggravate the bone destruction caused by the inflammation and results in bizarre bone patterns.

Bone Destruction Caused by Systemic Disorders

A possible relationship between periodontal bone loss and osteoporosis might be present. Osteoporosis is a physiologic condition of postmenopausal women that results in the loss of bone mineral content as well as structural bone changes. Some studies show a relationship between skeletal density and oral bone density; and among osteopenia and periodontitis, tooth mobility, and tooth loss.

Periodontal bone loss may also occur with generalized skeletal disturbances such as Hyperthyroidism, Leukemia, and Langerhans cell histiocytosis.

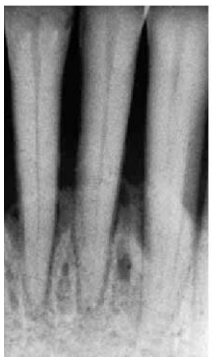
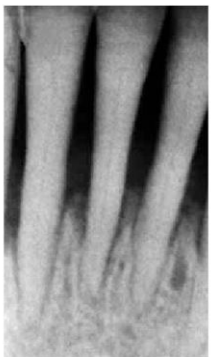
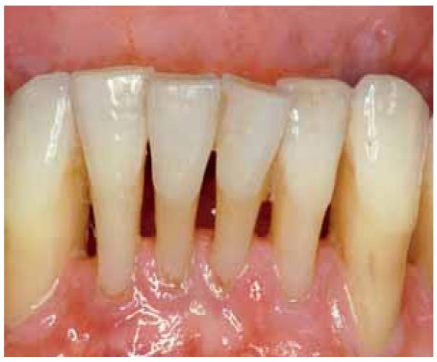
Bone Destruction Patterns in Periodontal Disease

Periodontal disease alters the morphologic features of the bone in addition to reducing bone height. An understanding of the nature and pathogenesis of these alterations is **essential for effective diagnosis and treatment**

1. Horizontal Bone loss

It occurs where progression of bone loss takes place at **an even rate leading to a symmetrical reduction in the height of the alveolar bone** .In this type of bone loss, the bony architecture appears relatively flat. It is the most common pattern of alveolar bone loss. It can be regularly seen at sites where the interdental bone is thin.

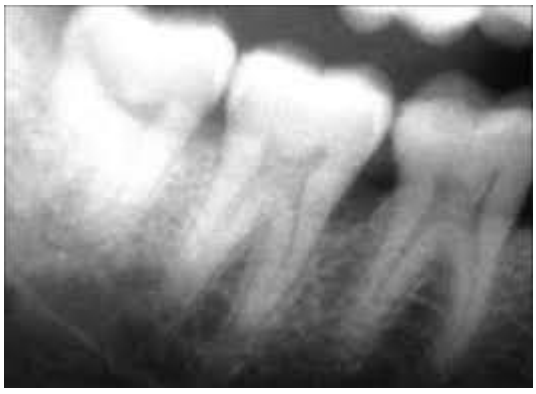
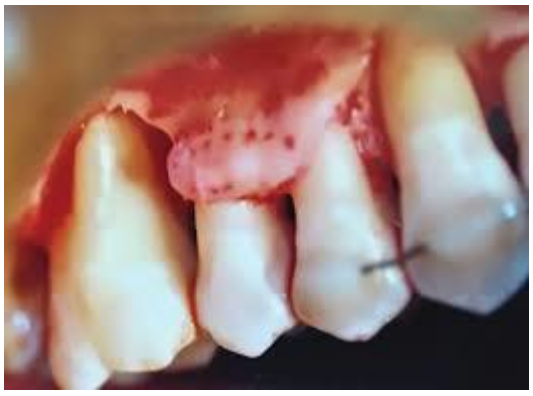
Supra-bony pockets accompany the horizontal bone loss. Horizontal bone loss can be seen on radiographs. Regenerative procedures with periodontal horizontal bone loss are not undertaken.

2. Vertical or Angular Defects

It occurs where bone loss develops **around adjacent teeth/surfaces at different rates**. It primarily affects a tooth and not it is adjacent. Bone loss takes a vertical angular-cuneiform when there is an adequate volume of alveolar bone surrounding the root/roots. They occur in an oblique-slanting direction with rapid bone resorption progressing into the bone adjacent to the root surface. They have a funnel shaped-trench like appearance hallowed trough-shaped presentation. Vertical defects can occur interproximally or on the radicular surface of tooth surface. Their presence may be suggested on radiographs, but careful probing and surgical exposure of the areas are required to determine their exact

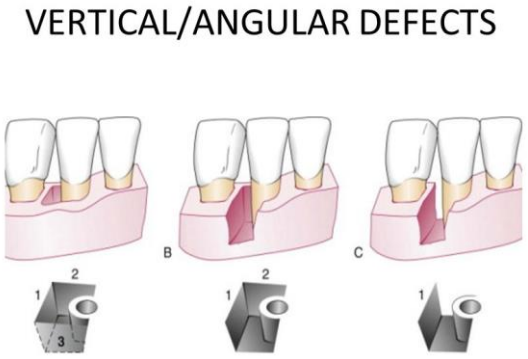
conformation and dimensions. They increase with age. An individual tooth can have combination bone loss consisting of both horizontal component and vertical component.

These vertical defects are classified based on the residual bony walls as follows:

❖ Three-wall bony defect

They are bordered by one tooth surface and three bony surfaces. It was originally called intra bony defect. It may be difficult to visualize this defect on the radiograph. It yields the best results with periodontal regeneration. They are frequently observed on the mesial surfaces of the molars.



❖ Two-wall bony defect

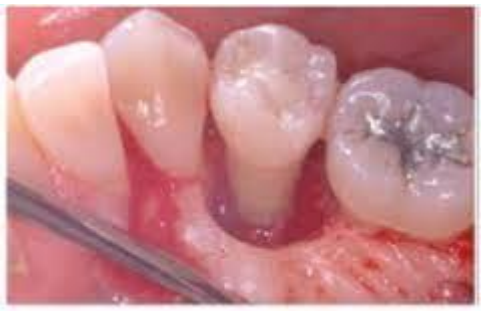
It is enclosed by two tooth surfaces and two osseous surfaces (buccal and palatal/lingual). The cortical bone is generally intact. It may be difficult to visualize this defect on the radiograph. It is located between adjoining posterior teeth.

❖ One-wall bony defect / Hemiseptum

Only one bony wall remains facing the involved tooth surface. It occurs generally when considerable amount of interdental septum is lost.

❖ circumferential defects

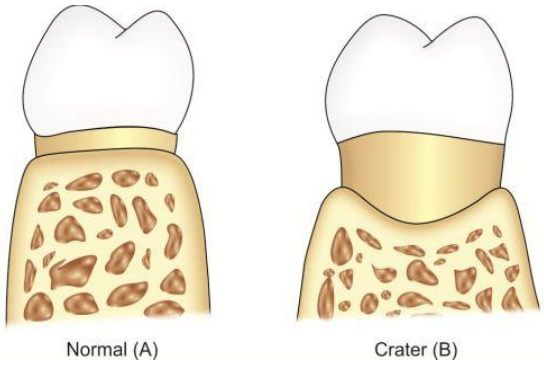
Continuous defects that involved more than one surface of a tooth, in a shape that is similar to a trough.



3. Osseous Craters

Osseous craters are a specific type of two-wall defect; they present as concavities in the crest of the interdental bone that is confined within the facial and lingual walls.

- Craters have been found to make up about one-third (35.2%) of all defects and about two-thirds (62%) of all mandibular defects.
- They occur twice as often in posterior segments as in anterior segments.
- The heights of the facial and lingual crests of a crater have been found to be identical in 85% of cases, with the remaining 15% being almost equally divided between higher facial crests and higher lingual crests.
- It is the most common bony lesion encountered in periodontal disease.



Ochsenbein divided osseous craters into three types based on the depth of the crater from the crest of facial/lingual bone as follows:

- Shallow crater:1-2 mm.
- Medium crater:3-4 mm.
- Deep crater: 5mm or more

Why are interdental craters so common?

-Plaque: The difficulty in removal of plaque from the interdental area.

-Morphology of interdental septum: The normal flat or even concave faciolingual shape of the interdental septum in lower molar may favor crater formation.

-Pathway for inflammation: Vascular patterns from the gingiva to the center of the crest.

4. Bulbous bone contours: .

-These are bony enlargement caused by exostoses .
-They occur as an adaptation to function or buttressing bone formation.
-Prevalence: Maxilla > Mandible.

5. Reversed architecture :

When the crest of the interdental gingiva or bone is located apical to its mid-facial and mid-lingual margins.

-These defects are produced by loss of the interdental bone crest, including the facial and/or lingual plates, without concomitant loss of radicular bone, thereby reversing the normal scalloped architecture.

- It is also called as inconsistent bony margin.
- It is more common in Maxilla.

6. Furcation Involvement

It refers to the invasion of bifurcation and trifurcation of multi-rooted teeth by periodontal disease.