Bone Loss and Patterns of Bone Destruction

Bone loss is the ultimate and last result of the inflammatory process observed in periodontitis. In periodontitis, the inflammatory process, which occurs as a response to the bacterial biofilm insult, has damaging effects on the periodontal unit, which results in the destruction of periodontal ligament fibers and bone loss. The existing bone level is the consequence of past pathologic episodes, while 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. Periodontitis is always preceded by gingivitis, but not all gingivitis progresses to periodontitis. The factors responsible for the extension of inflammation to the supporting structures are not clearly understood and related to individual susceptibility to the bacterial biofilm insult or microbiologic changes that occur in the pocket environment and surrounding tissues.

The transition from gingivitis to periodontitis is associated with:

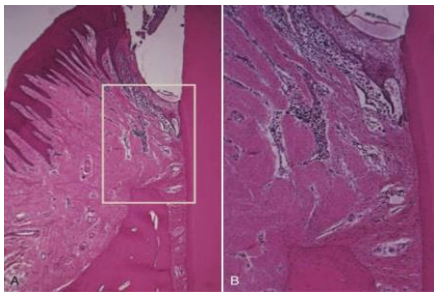
- Changes in the composition of the bacterial biofilm. In advanced stages of disease, the number of motile organisms and spirochetes increases, whereas the number of coccoid rods and straight rods decreases.

- Changes in the cellular composition of the infiltrated connective tissue with increasing severity of the lesion. Fibroblasts and lymphocytes predominate in stage 1 gingivitis, whereas the number of plasma cells and blast cells increases gradually as the disease progresses.

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. Some studies suggest that 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.

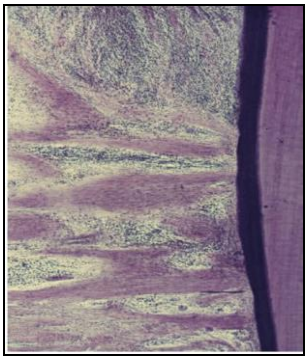
Histopathology

Gingival inflammation extends along the collagen fiber bundles and follows the course of the blood vessels through the loosely arranged tissues around them into the alveolar bone. Although the inflammatory infiltrate is concentrated in the marginal periodontium, the reaction is a much more diffuse one, often reaching the bone and eliciting a response before evidence of crestal resorption or loss of attachment exists. In the upper molar region, inflammation can extend to the maxillary sinus, resulting in thickening of the sinus mucosa. (Fig 1).



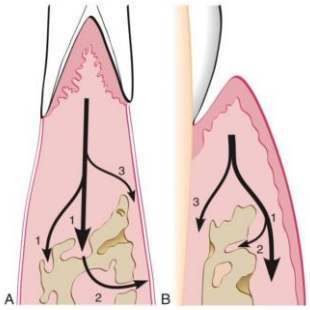
(Fig 1)

Inter proximally, inflammation spreads to the loose connective tissue around the blood vessels, through the fibers, and then into the bone through vessel channels that perforate the crest of the interdental septum at the center of the crest , toward the side of the crest, or at the angle of the septum. In addition, inflammation may enter the bone through more than one channel. Less frequently, the inflammation spreads from the gingiva directly into the periodontal ligament and from there into the interdental septum. (Fig 2)



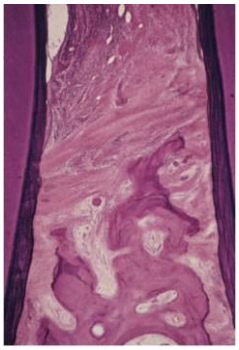
(Fig 2)

Facially and lingually, inflammation from the gingiva spreads along the outer periosteal surface of the bone and penetrates into the marrow spaces through vessel channels in the outer cortex. (Fig 3)



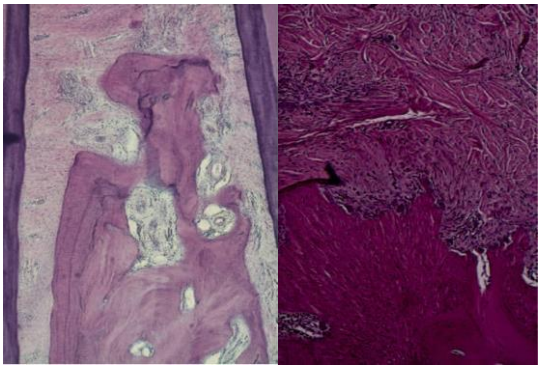
(Fig 3)

Along its course from the gingiva to the bone, the inflammation destroys the gingival and transeptal fibers, reducing them to disorganized granular fragments interspersed among the inflammatory cells and edema. However, there is a continuous tendency to recreate transeptal fibers across the crest of the interdental septum farther along the root as the bone destruction progresses . As aresult, transeptal fibers are present, even in cases of extreme periodontal bone loss. The dense transeptal fibers form a firm covering over the bone that is encountered during periodontal lap surgery after the super facial granulation tissue is removed. (Fig 4)



(Fig 4)

After inflammation reaches the bone via extension from the gingiva, it spreads into the marrow spaces and replaces the marrow with a leukocytic and fluid exudate, new blood vessels, and proliferating fibroblasts . Multinuclear osteoclasts and mononuclear phagocytes increase in number, and the bone surfaces appear to be lined with Howship lacunae. (Fig 5)



(Fig 5)

In the marrow spaces, resorption proceeds from within and causes a thinning of the surrounding bony trabeculae and an enlargement of the marrow spaces; this is followed by the destruction of the bone and a reduction in bone height. Normally, fatty bone marrow is partially or totally replaced by faibrous 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.

All bone present in areas with periodontitis is viable and live 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

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. Similar findings have been reported in experimentally induced periodontitisin animals.

Rate of Bone Loss

Löe and colleagues found the rate of bone loss averages about 0.2 mm per year for facial surfaces and about 0.3 mm per year for proximal surfaces when periodontal disease was allowed to progress untreated.

However, the rate of bone loss may vary, depending on the type of disease present. Löe and colleagues also identified the following three subgroups of patients with periodontal disease on the basis of the interproximal loss of attachment and tooth mortality (loss of attachment can be equated with loss of bone, although attachment loss precedes bone loss by about 6 to 8 months :

1. Approximately 8% of persons had a rapid progression of periodontal disease that was characterized by a yearly loss of attachment of 0.1 mm to 1 mm.

2. Approximately 81% of individuals had moderately progressive periodontal disease with a yearly loss of attachment of 0.05 mm to 0.5 mm.

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

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 and stimulate gingival cells to release mediators that have the same effect.

-This bacterial biofilm products and inflammatory mediators can also act directly on osteoblasts or their progenitors by inhibiting their action and reducing their numbers.

In addition, in patients with rapidly progressing diseases (e.g.,aggressive periodontitis), bacterial micro colonies or single bacterial cells have been found between collagen fibers and over the bone surface, suggesting a direct effect. Inflammatory cells released Several host factors which are capable of inducing bone resorption in vitro, and they play a role in periodontal disease. These include (prostaglandins, interleukin-1α, interleukin--, and tumor necrosis factor alpha).Prostaglandin E2 induces bone resorption in the absence of inflammatory cells, with few multinucleated osteoclasts. In addition, non steroidal anti-inflammatory drugs (e.g., ibuprofen) inhibit porstaglandin E2 production, there by slowing bone loss in naturally occurring periodontal disease. This effect disappears 6 months after the ending of drug administration.

Bone Destruction Caused by Trauma From Occlusion

Another cause of periodontal bone destruction is trauma from occlusion, which 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 and tooth structure. These changes are reversible and can be repaired if the wrong forces are removed. However, persistent trauma from occlusion results in funnel-shaped widening of the crestal portion of the periodontal ligament with resorption of the adjacent bone.**

When it is combined with inflammation**, trauma from occlusion may aggravate the bone destruction caused by the inflammation and results in bizarre bone patterns.**

Bone Destruction Caused by Systemic Disorders

Local and systemic factors regulate the physiologic equilibrium of bone. When a generalized tendency toward bone resorption exists, bone loss initiated by local inflammatory processes may be magnified and this systemic influence on the response of alveolar bone, envisioned by Glickman during the early 1950s, considers a systemic regulatory influence in all cases of periodontal disease.

There is possible relationship between periodontal bone loss and osteoporosis. Osteoporosis is a physiologic condition of postmenopausal women that results in the loss of bone mineral content as well as structural bone changes. Periodontitis and osteoporosis share a number of risk factors (e.g., aging, smoking, certain diseases, medications that interfere with healing).

Periodontal bone loss may also occur with generalized skeletal disturbances (e.g., hyperparathyroidism, leukemia, histiocytosis X) via mechanisms that may be totally unrelated to the more common biofilm-induced, inflammatory periodontal lesion.

Factors Determining Bone Morphology in Periodontal Disease

1-Normal Variation in Alveolar Bone

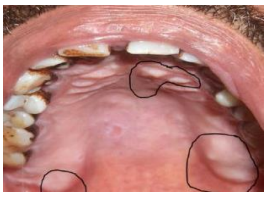
The anatomic features that substantially affect the bone-destructive pattern of periodontal disease include the following:

- **Thickness, width, and crestal angulations of the interdental septa**
- **Thickness of the facial and lingual alveolar plates**
- **Presence of fenestrations and dehiscences**
- **Alignment of the teeth**
- **Root and root trunk anatomy**
- **Root position within the alveolar process**
- **Proximity with another tooth surface**

For example, angular osseous defects cannot form in thin facial or lingual alveolar plates, which have little or no cancellous bone between the outer and inner cortical layers. In such cases, the entire crest of the plate is destroyed, and the height of the bone is reduced in a horizontal fashion.

2- Exostoses

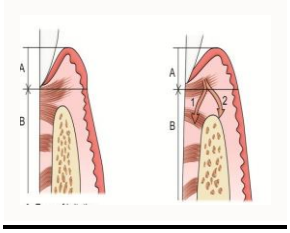
Exostoses are outgrowths of bone of varied size and shape. Palatal exostoses have been found in 40% of human skulls. They can occur as small nodules, large nodules, sharp ridges, spike like projections, or any combination of these examples. Exostoses have been described in rare cases as developing after the placement of free gingival grafts.(Fig 6)



(Fig 6)

3-Trauma From Occlusion

Trauma from occlusion may be a factor in determining the dimension and shape of bone deformities. It may cause a thickening of the cervical margin of alveolar bone or a change in bone morphology (e.g., funnel-like crestal bone, buttressing bone) on which inflammatory changes may later be superimposed.



4-Buttressing Bone Formation

Bone formation sometimes occurs in an attempt to buttress bony trabeculae that are weakened by resorption.

- *When this occurs within the jaw,* it is termed central buttressing bone formation. -*When it occurs on the external surface*, it is referred to peripheral buttressing bone formation and this may cause bulging of the bone contour, which sometimes accompanies the production of osseous craters and angular defects .

5-Food Impaction

Interdental bone defects often occur where the proximal contact is light or absent. Physical pressure and the additional collection of bacteria from food impaction contribute to interproximal resorption and the development of reverse bone architecture. In some cases, the poor proximal relationship may result from a shift in tooth position as a result of extensive bone destruction that precedes food impaction. In patients with this condition, food impaction is a complicating factor rather than the cause of the bone defect.

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.

-Horizontal Bone Loss

Horizontal bone loss is the most common pattern of bone loss in periodontal disease. The bone is reduced in height, but the bone margin remains approximately perpendicular to the tooth surface. The interdental septa and the facial and lingual plates are affected but not necessarily to an equal degree around the same tooth.

-Vertical or Angular Defects

Vertical or angular defects are those that occur in an oblique direction, leaving a hollowed-out trough in the bone along with the root; the base of the defect is located apical to the surrounding bone.

Angular defects mostly have accompanying infrabony periodontal pockets which must always have an underlying angular defect. Goldman and Cohen classified angular defects on the basis of the number of osseous walls and they may have one, two, or three walls.

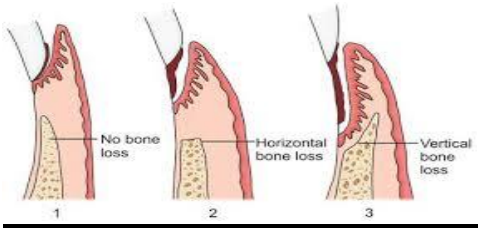
***Circumferential defects* are acontinuous defects that involved more than one surface of a tooth, in a shape that is similar to at rough.**

***Combined osseous defect* is the number of walls in the apical portion of the defect is often greater than that in its occlusal portion.**

Vertical defects that occur interdentally can generally be seen on the radiograph, although thick, bony plates may sometimes obscure them. Angular defects can also appear on facial and lingual or palatal surfaces, but these defects are more difficult to visualize on

radiographs. Surgical exposure is the only sure way to determine the presence and configuration of vertical osseous defects.

Vertical defects increase with age. Approximately 60% of people with interdental angular defects have only a single defect.Vertical defects detected radiographically have been reported to appear most often on the distal and mesial surfaces. However, three-wall defects are more frequently found on the mesial surfaces of the upper and lower molars.



-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.

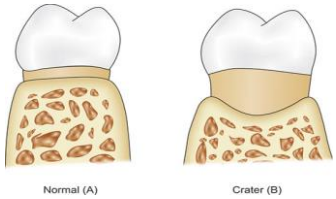
The heights of the facial and lingual crests of a crater have been found to be identical in 85% of cases, with the remaining 15% being almost equally divided between higher facial crests and higher lingual crests.

The following reasons for the high frequency of interdental craters have been suggested:

• The interdental area collects biofilm and is difficult to clean.

• The normal lat or even slightly concave buccolingual shape of the interdental septum in the lower molars may favor crater formation.

• Vascular patterns from the gingiva to the center of the crest may provide a pathway for inflammation.



-Bulbous Bone Contours

Bulbous bone contours are bony enlargements that are caused by exostoses , adaptation to function, or buttressing bone formation. They are found more frequently in the maxilla than in the mandible.

-Reversed Architecture

Reverse (or negative) alveolar bone design is the result of aloss of interdental bone, without loss of radicular (buccal or lingual/palatal) bone, thereby reversing the normal (or positive) architecture . Negative architecture is more common in the maxilla of patients with periodontitis.

- Ledges

Ledges are plateau-like bone margins that are caused by the resorption of thickened bony plates.

-Furcation Involvement

furcation involvement refers to the invasion of the bifurcation and trifurcation of multirooted teeth by periodontitis. The denuded furcation may be visible clinically or covered by the wall of the pocket. Furcation involvements have been classified as grades I through IV according to the amount of tissue destruction. It is simply the apical extension of the periodontal pocket along a multirooted tooth. Furcation involvement is a stage of progressive periodontal disease, and it has its same etiology. The difficulty and sometimes the impossibility of controlling biofilm in furcations are responsible for the presence of extensive lesions in this area. The diagnosis of furcation involvement is made by clinical examination and careful probing with a specially designed probe. Radiographic examination of the area is helpful, but lesions can be obscured by the angulation of the beam and the radiopacity of neighboring structures.