Early periodontitis

Radiographic Changes in Periodontal Disease:

- **Early periodontitis**: areas of localized erosion of the alveolar bone crest (blunting of the crest in anterior regions and a rounding of the junction between the crest and lamina dura in the posterior regions).

- **Moderate periodontitis**: the destruction of alveolar bone extends beyond early changes in the alveolar crest and may include buccal or lingual plate resorption, generalized horizontal erosion or localized vertical defects and possible clinical evidence of tooth mobility.

- **Advanced periodontitis**: the bone loss is so extensive that the remaining teeth show excessive mobility and drifting and are in jeopardy of being lost. There is usually extensive horizontal bone loss or extensive bony defects.

1. In gingivitis, the radiographic appearance of the bone will be normal.
2. The crest of the alveolar bone is affected in periodontal disease. In health, it lies 1-2 mm below the level of the CEJs of adjacent teeth.
3. A reduction of only 0.5 or 1.0 mm in the thickness of the cortical plate is sufficient to permit radiographic visualization of destruction of the inner cancellous trabeculae.

**Important**: Diabetes mellitus is an extremely important disease from a periodontal standpoint. It is a complex metabolic disease characterized by chronic hyperglycemia. Individuals with diabetes have a higher prevalence and severity of periodontal disease than do those without diabetes. Diabetes does not cause periodontal disease but studies show that it alters the response of the periodontal tissues to bacterial plaque. **Poorly controlled diabetics often have:**

- Enlarged gingiva, sessile or pedunculated gingival polyps, polypoid gingival proliferations, abscess formation, and loosened teeth
- Polymorphonuclear leukocyte deficiencies resulting in impaired chemotaxis, defective phagocytosis, or impaired adherence
- The chronic hyperglycemia adversely affects the synthesis, maturation, and maintenance of collagen and extracellular matrix. Numerous proteins and matrix molecules undergo a nonenzymatic glycosylation, resulting in accumulated glycation end products (AGEs). This increase in AGEs affects how collagen is normally repaired or replaced and may play a role in the progression of periodontal disease.