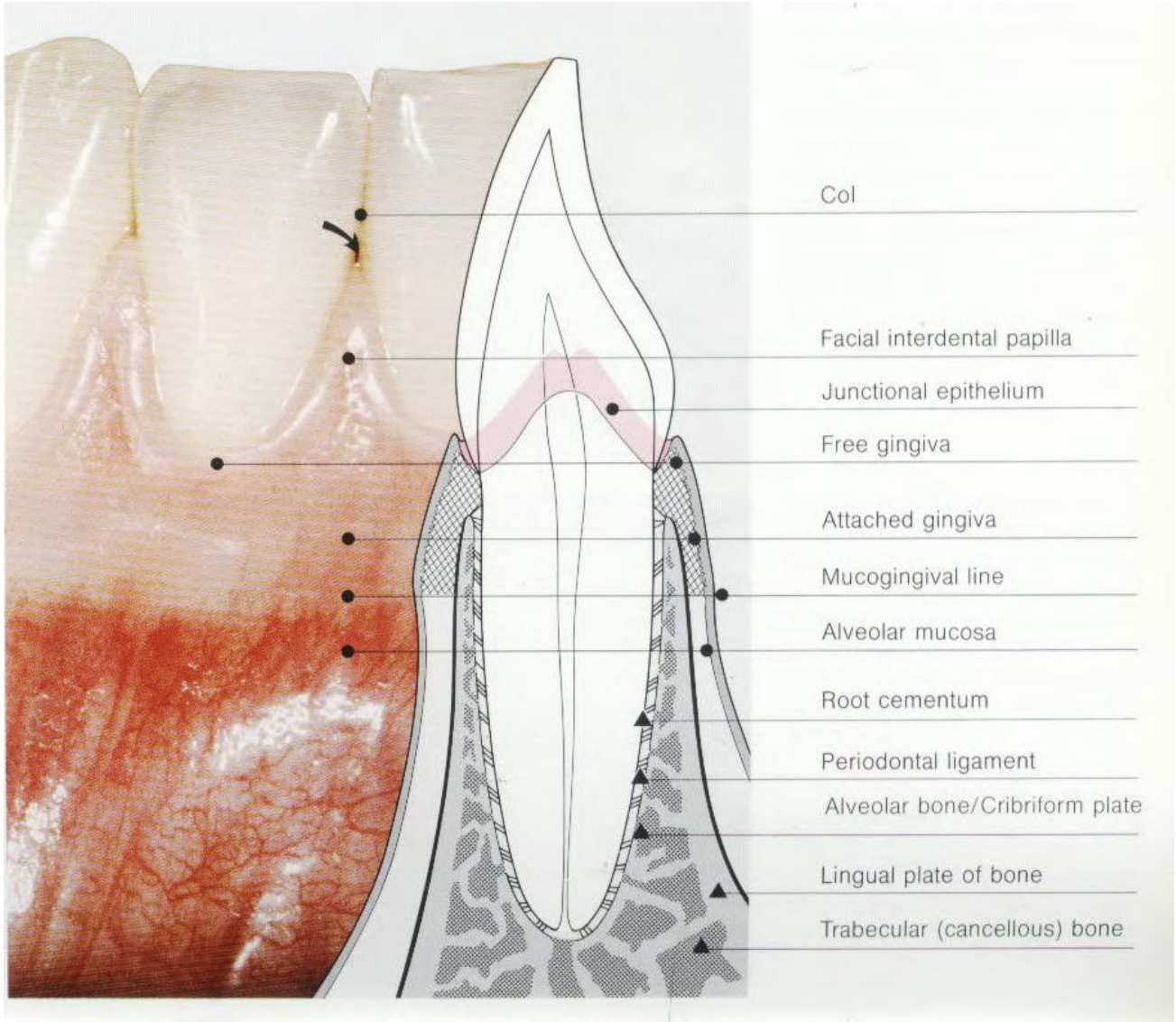
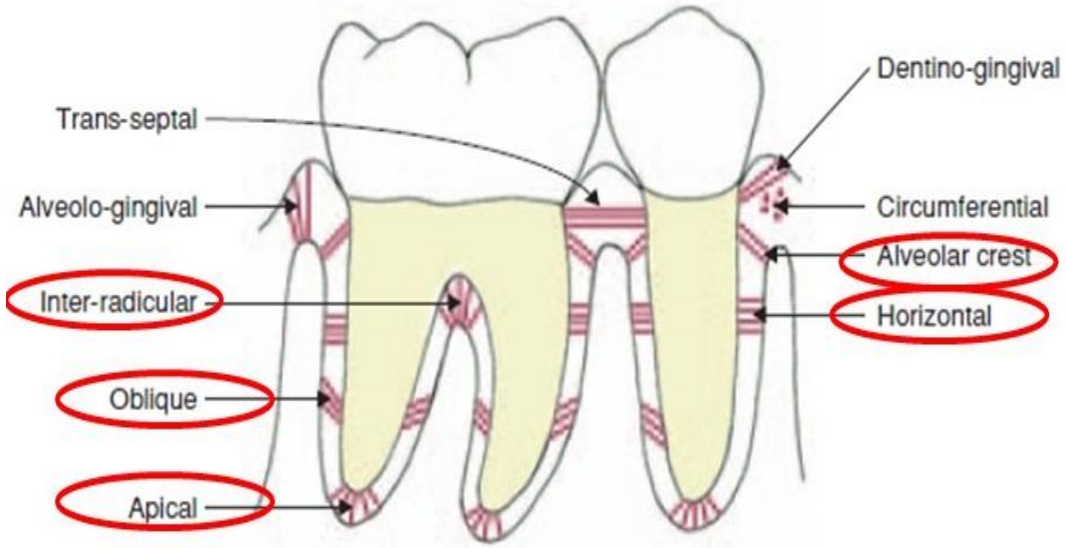
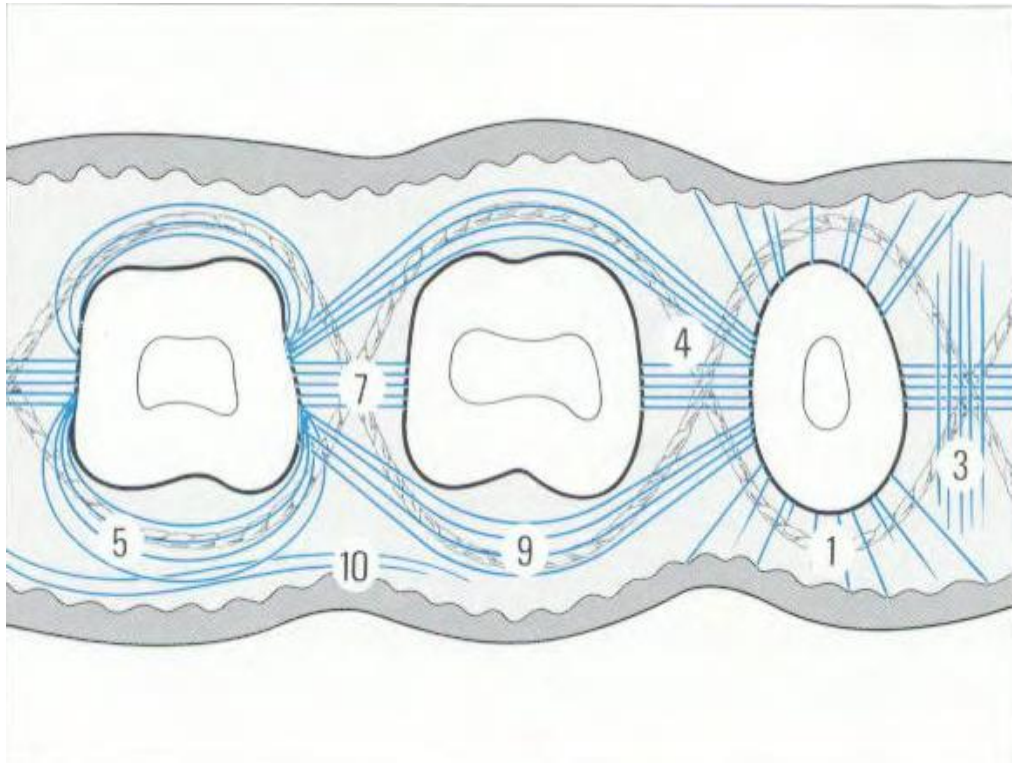
Periodontal tissue components

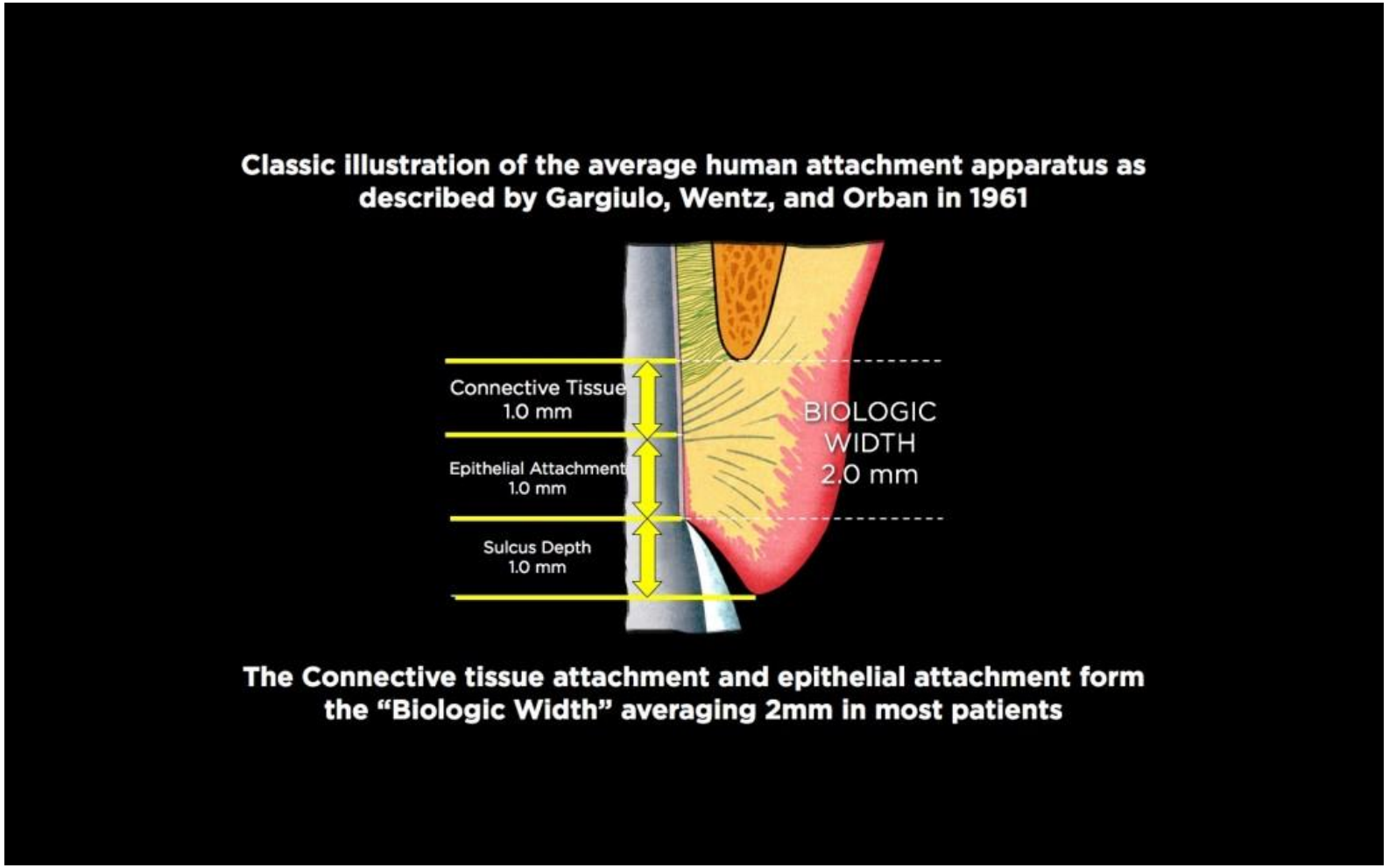


Periodontal ligaments

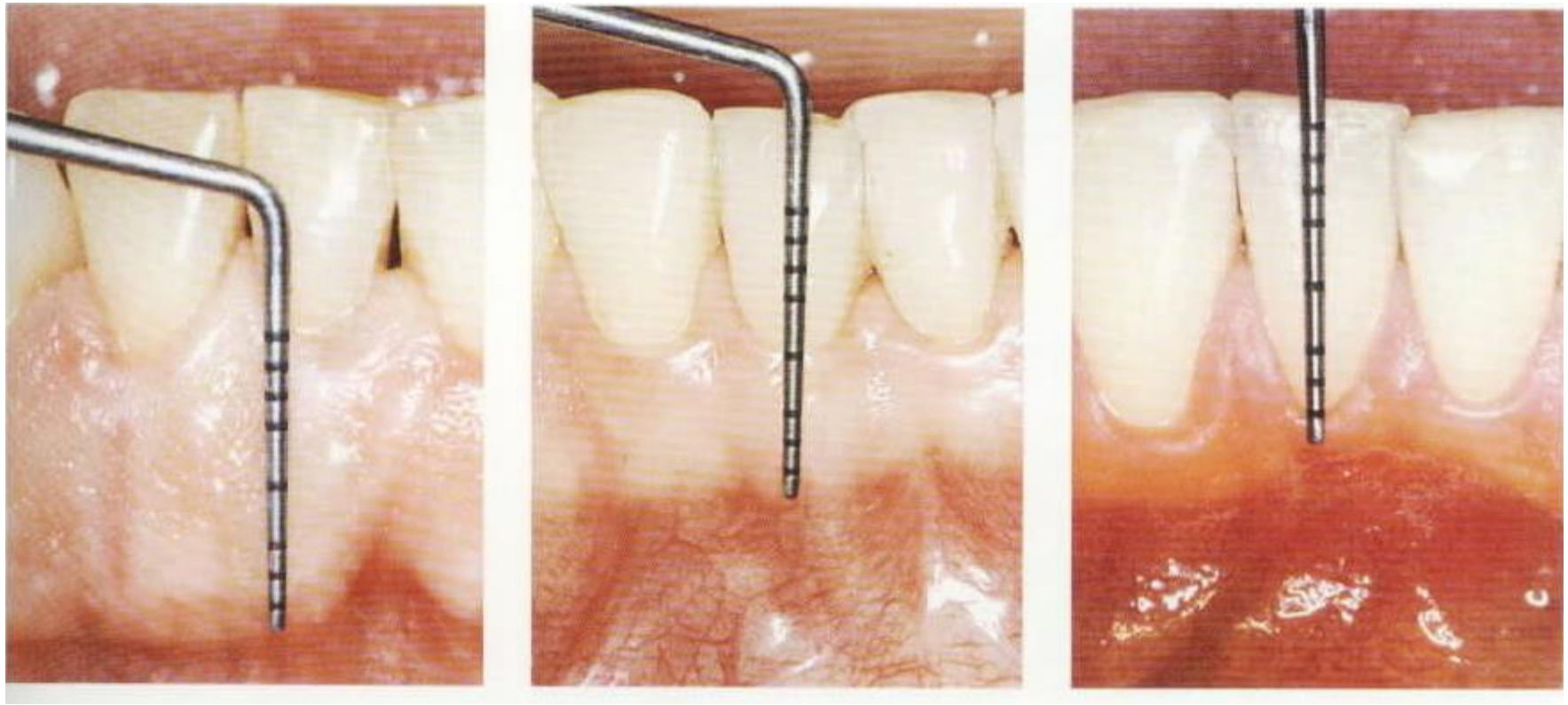




Biological width

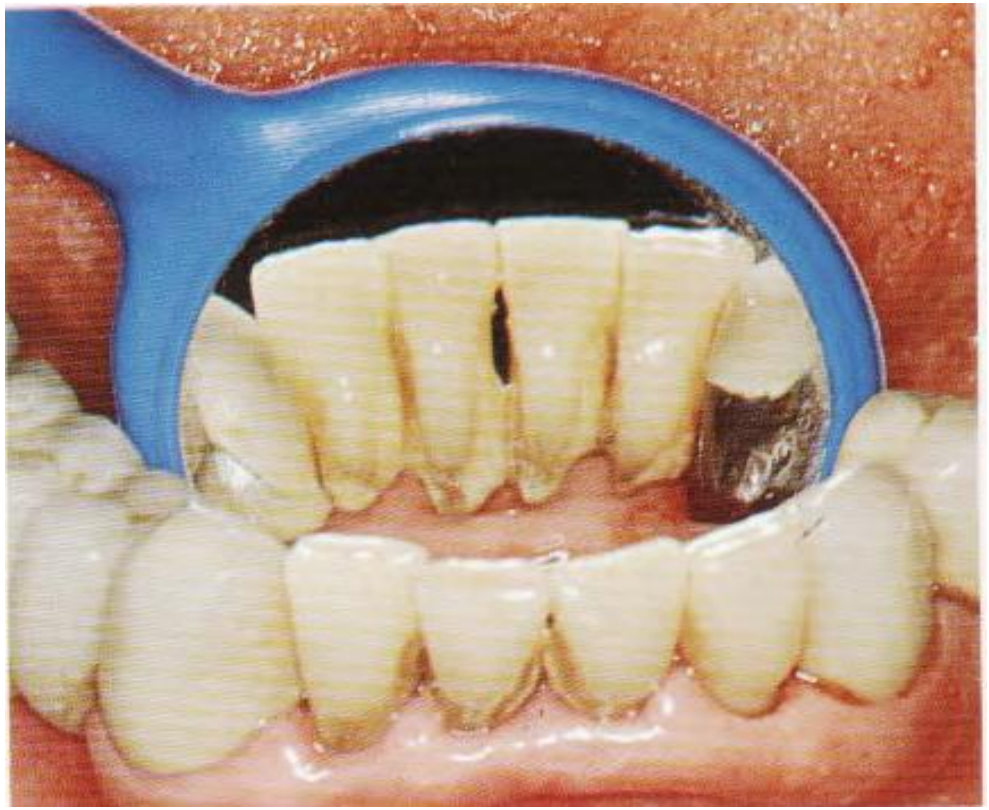


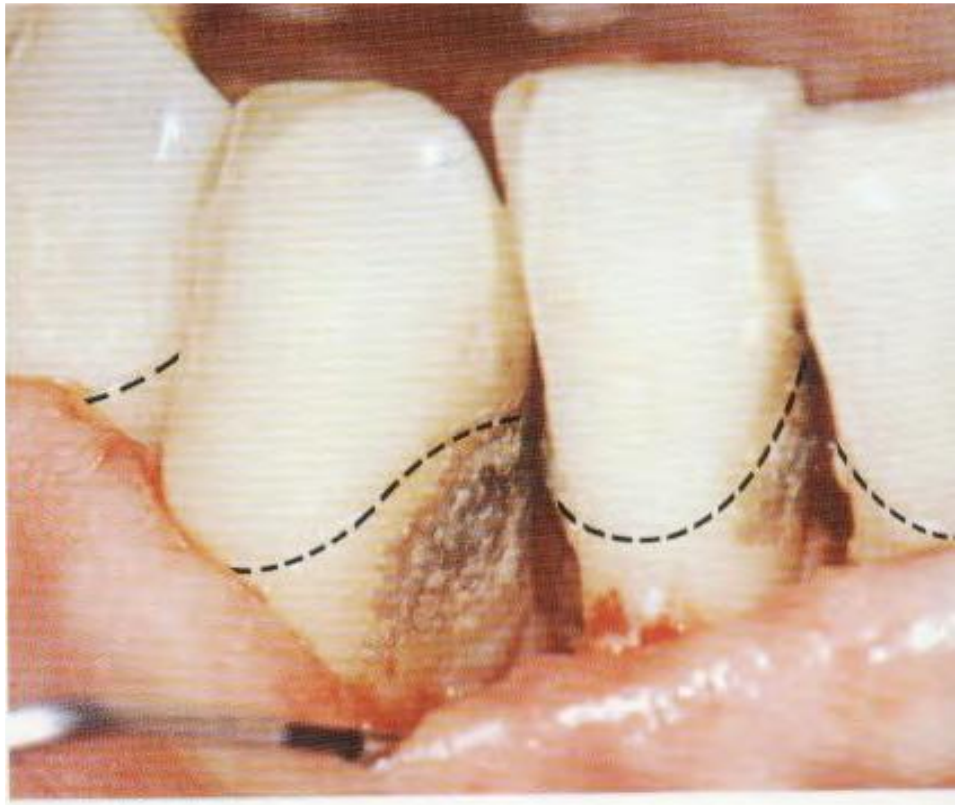
Variation between individuals



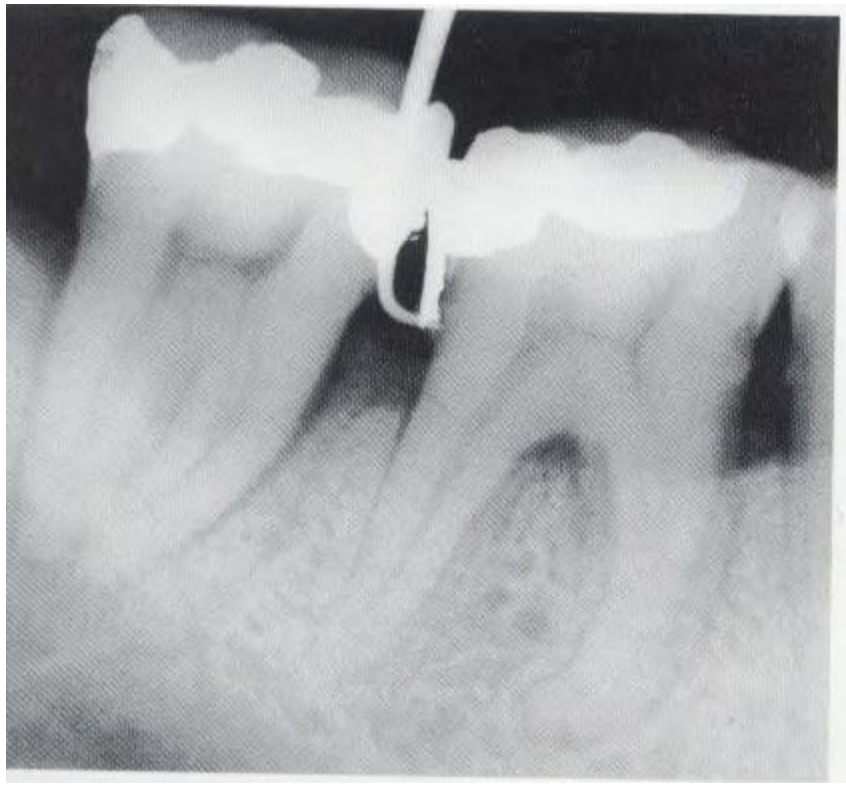
Plaque indicator

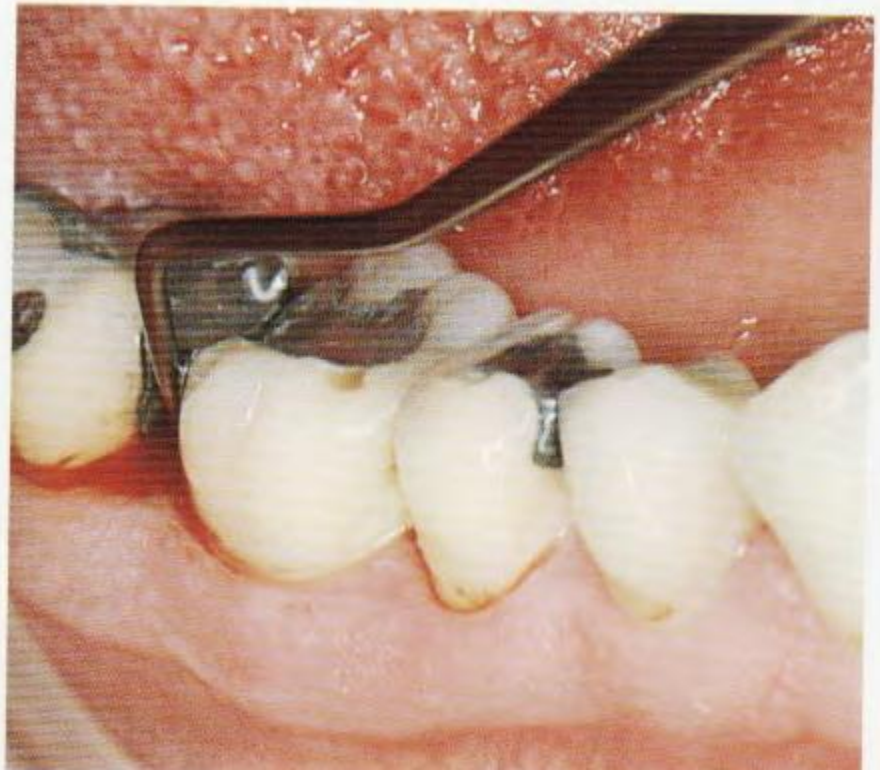
Dental Calculus





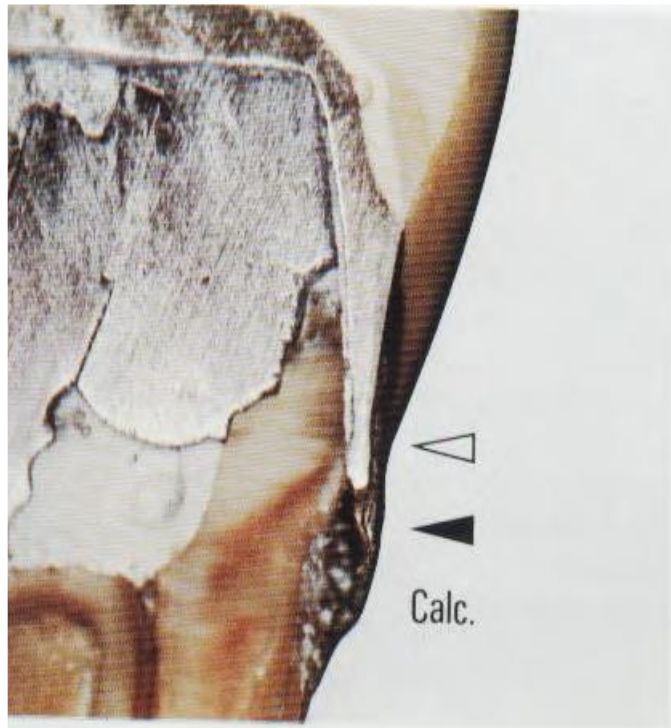
Plaque retentive factors

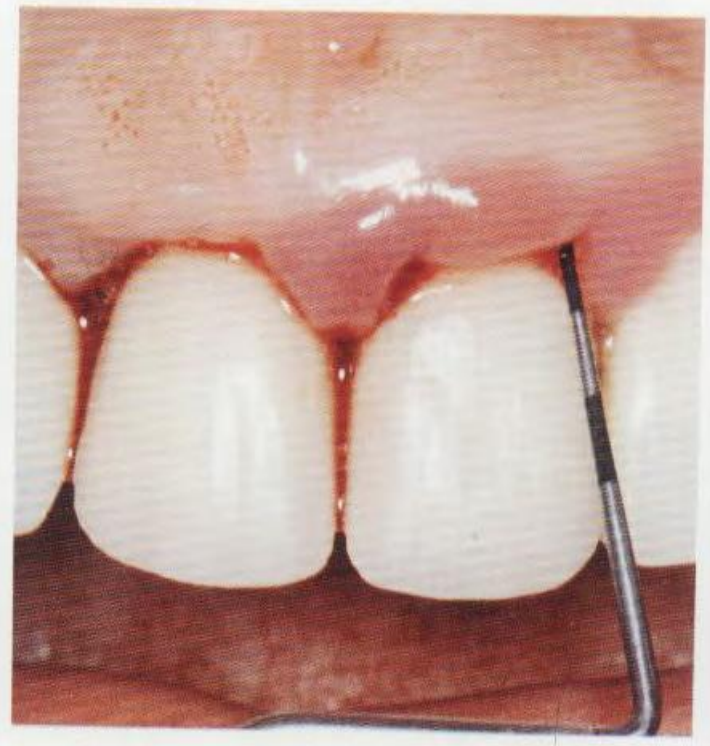




37 Amalgam restoration with overhang

Gross overhangs such as this, located subgingivally, invariably lead to plaque accumulation and to gingivitis (note hemorrhage). The plaque accumulated beneath an overhang changes in its composition: Pathogenic gram-negative anaerobes (e. g., Bacteroides species) increase markedly in number.





38 Crown margin overhang and open margins

Right: The cement that was used to cement this porcelain jacket crown has begun to extrude from the open margin. The massive retention of plaque between the crown and the prepared tooth led to severe gingivitis with establishment of a pathogenic bacterial flora.

Left: Section through a porcelain-fused-to-gold crown with a margin that is both overhanging (arrows) and open. Darkly stained calculus is observed apical to the poor crown margin.

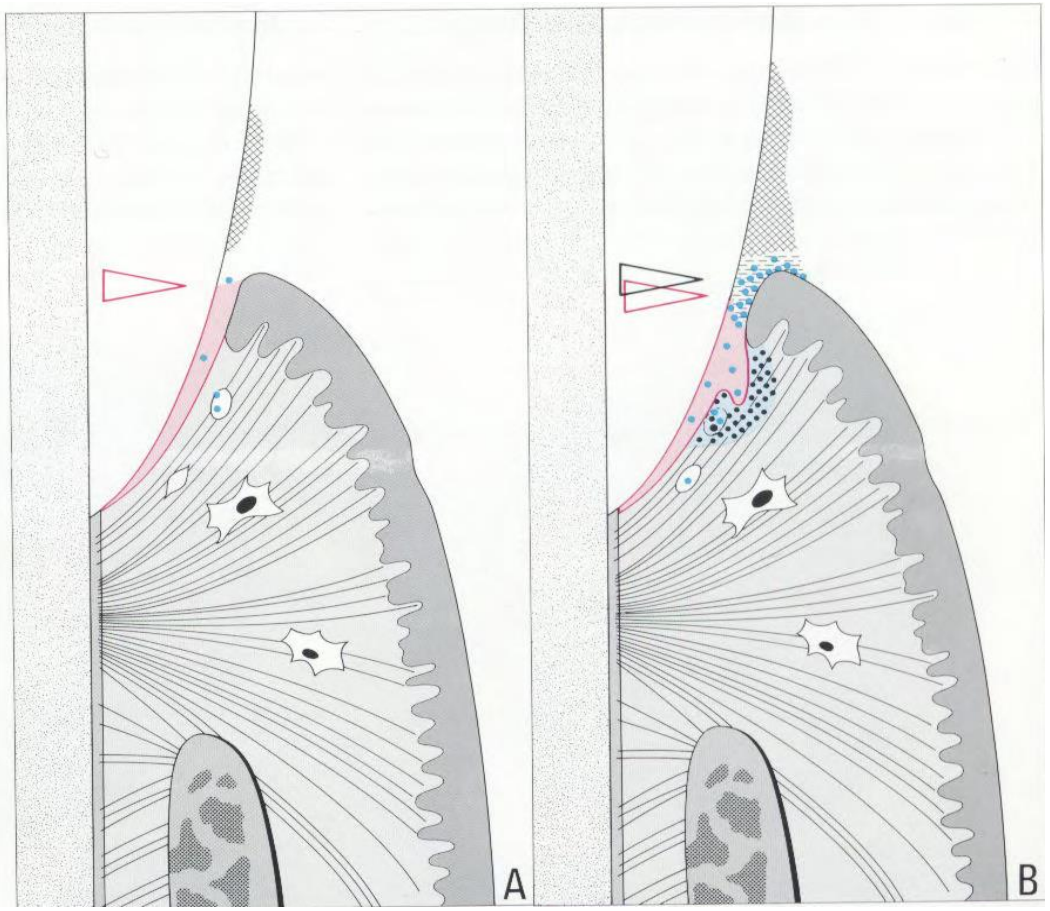
Pathogenesis of periodontal disease

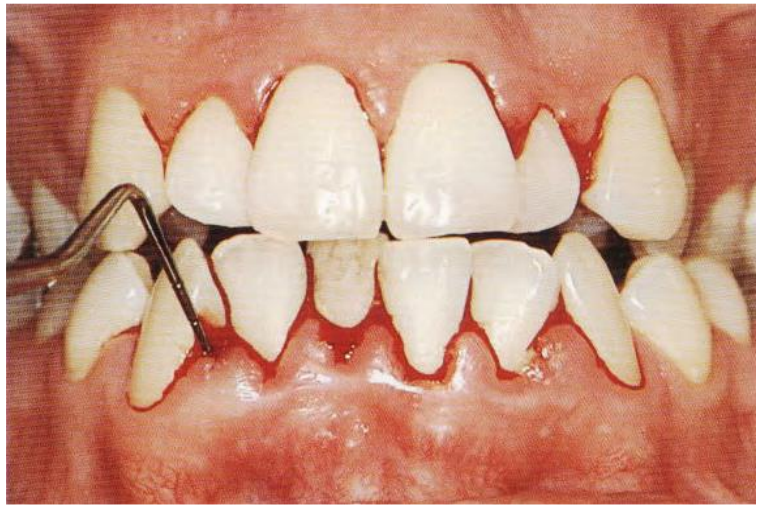
Healthy gingiva → Gingivitis

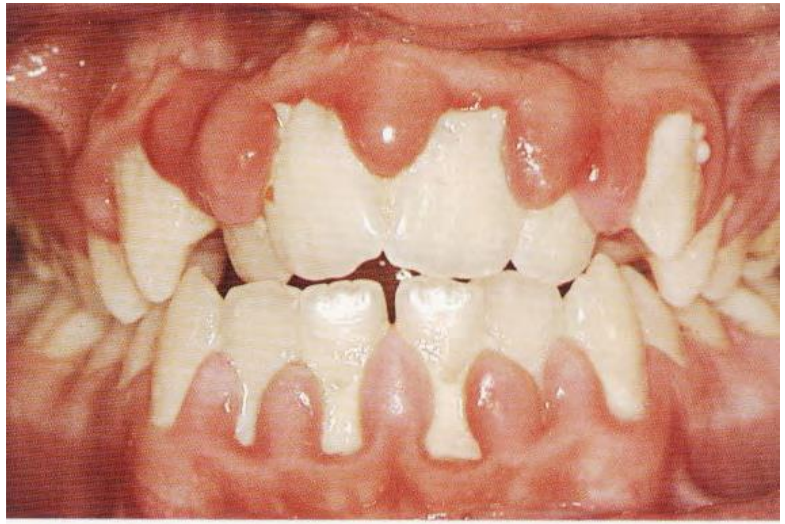
45 Healthy gingiva (A)
Absence of plaque or very little accumulation; normal junctional epithelium (pink); minimal sulcus depth (red arrow). A few PMNs (blue dots) transmigrate the JE in the direction of the sulcus bottom. Dense collagenous fiber system; intact fibroblasts.

46 Initial / Early gingivitis (B)
Early plaque accumulation. In the *initial* lesion, increased transmigration of PMNs (blue dots) within the JE.

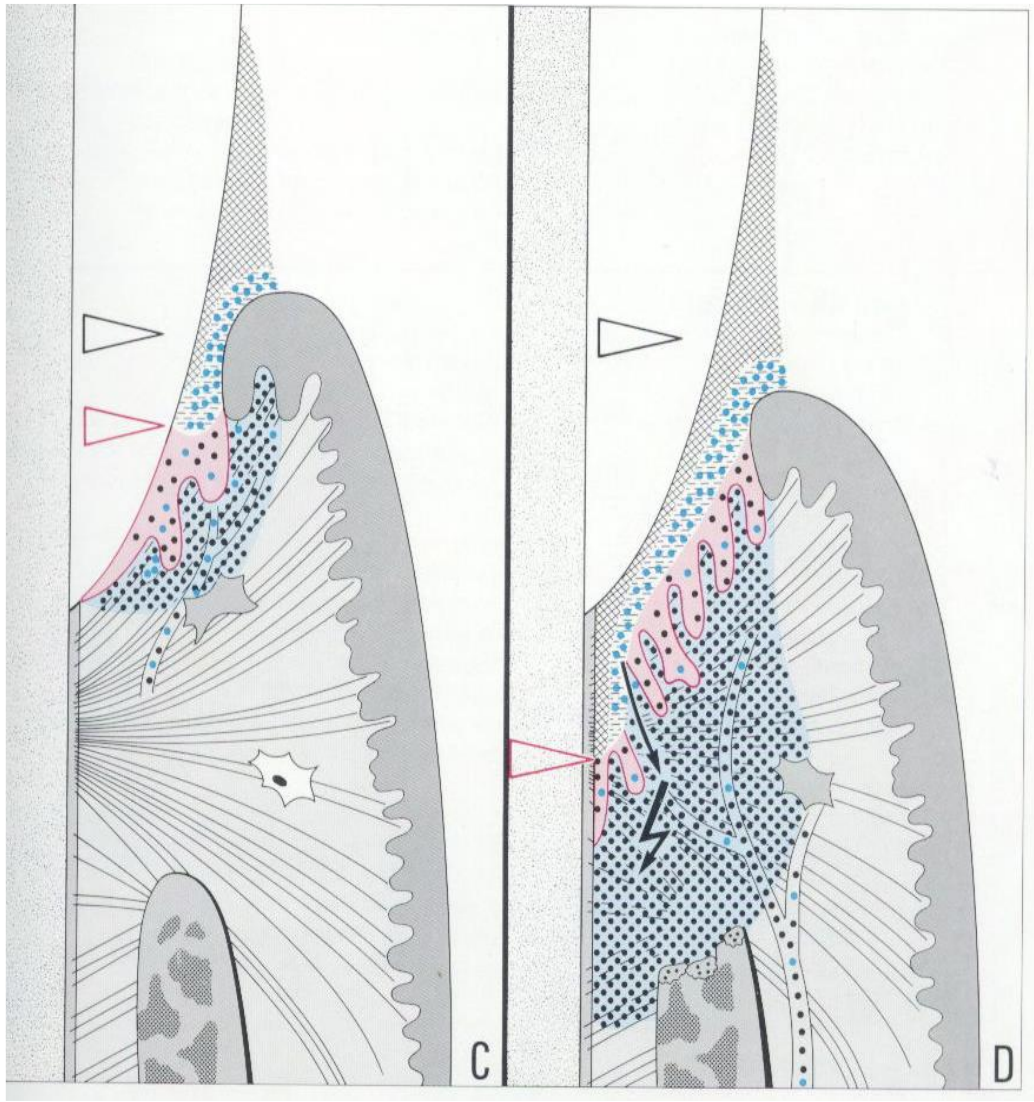
As the *early* lesion develops, the PMNs create within the slightly deepened sulcus (red arrow) a wall against the plaque bacteria. A lymphocytic infiltrate (black dots) occurs in the subepithelial tissues.







Gingivitis → Periodontitis

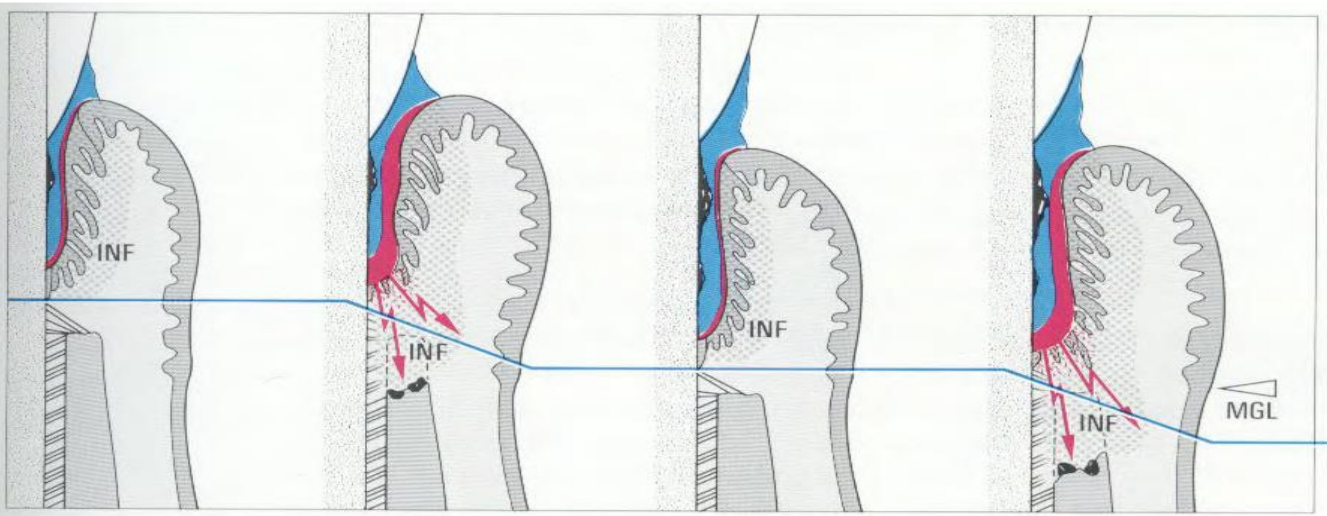


47 Established lesion (C)
The gingiva responds to a massive accumulation of plaque. All of the characteristics of gingivitis are manifest but may be more or less pronounced both clinically and histologically. The junctional epithelium, i. e., the epithelial attachment, may actually be displaced somewhat apically as a consequence of the advancing front of accumulating plaque, resulting in the formation of a gingival pocket (distance between red and black arrows). Nevertheless, at this stage there is no loss of connective tissue attachment. The differentiated inflammatory infiltrate protects the deeper structures of the periodontium.

48 Periodontitis (D)
The most important histological differences between gingivitis and periodontitis are bone resorption, apical proliferation and ulceration of the junctional (pocket) epithelium (red arrow indicates base of pocket), and progressive loss of connective tissue attachment.

In acute phases there may be bacterial invasion of the tissue, with resultant micro- or macroabscesses.

Cyclic Nature of Periodontitis – Attachment Loss – Bone Resorption



52 Cyclic nature
The increase of pathogenic bacteria in subgingival plaque, and the invasion of these into tissues causes an acute inflammatory reaction within the periodontium (red arrows). Bone resorption and loss of attachment ensue.

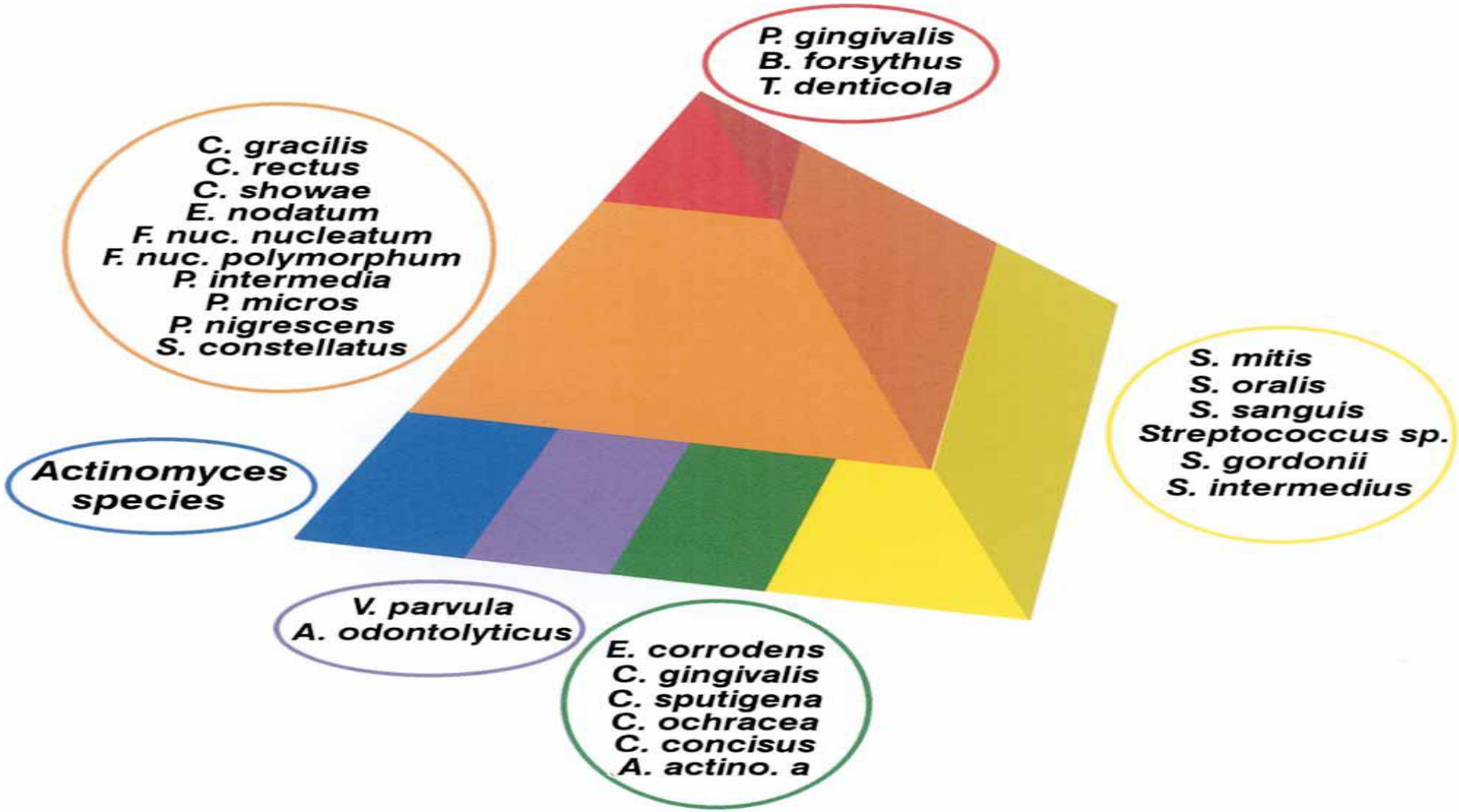
Clinical treatment or an enhanced host response can eliminate the bacterial insult and lead to stagnation of the acute exacerbation.

Attachment loss

Gram-negative anaerobic motile microorganisms
Invasion, infection

Pocket activity: Bleeding, exudation, PMNs, pus, ulceration of the pocket wall, infiltration

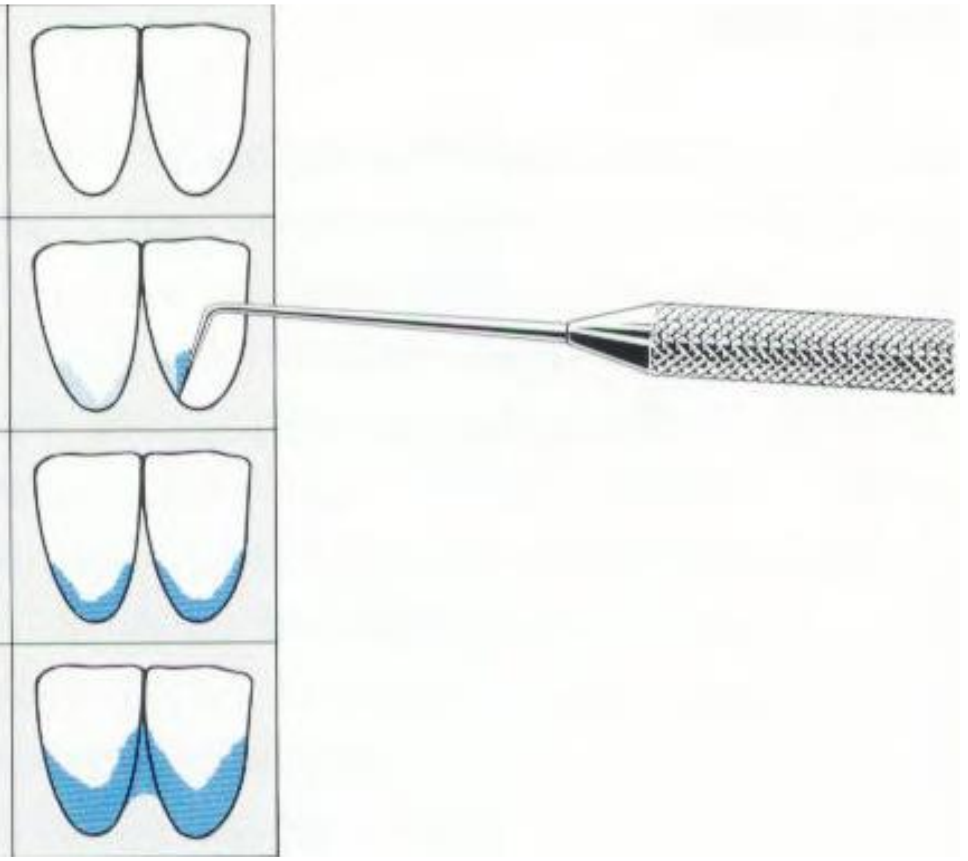
Bacterial plaque biofilm



Socransky & Haffajee 2002

Plaque index

Score	
0	No plaque
1	Thin film of plaque at the gingival margin, visible only when scraped with an explorer
2	Moderate amount of plaque along the gingival margin; interdental space free of plaque; plaque visible with the naked eye
3	Heavy plaque accumulation at the gingival margin; interdental space filled with plaque



Gingival index

Grade	
0	normal gingiva, no inflammation, no discoloration, no bleeding
1	mild inflammation, slight color change, mild alteration of gingival surface, no bleeding
2	moderate inflammation, erythema, swelling, bleeding on probing or when pressure applied
3	severe inflammation, severe erythema and swelling, tendency toward spontaneous hemorrhage, some ulceration

59 Gingival Index (GI)

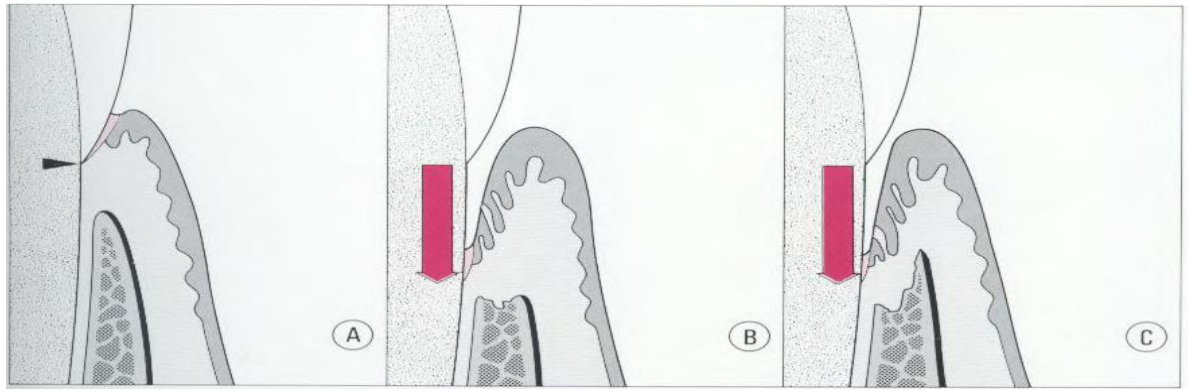
This index is used worldwide in epidemiological studies and scientific investigations. The GI scores gingival inflammation on the facial, lingual and mesial surfaces of all teeth. The symptom of bleeding comprises a score of 2.

The GI is recommended for epidemiological studies. It is less applicable for individual patients because the differences between the scoring levels are too gross.

Gingival recession

Table 1 Miller's classification of recession defects		
Class I		Recession that does not extend to the mucogingival junction with no periodontal bone loss in the interdental areas
Class II		Recession that extends to or beyond the mucovingival junction, with no interdental bone loss
Class III		Recession that extends to or beyond the mucogingival junction, with some periodontal attachment loss in the interdental area or malpositioning of the teeth
Class IV		Recession that extends to or beyond the mucogingival junction, with severe bone and/or soft-tissue loss in the interdental area and/or severe malpositioning of the teeth

Types of bone loss in Periodontal disease

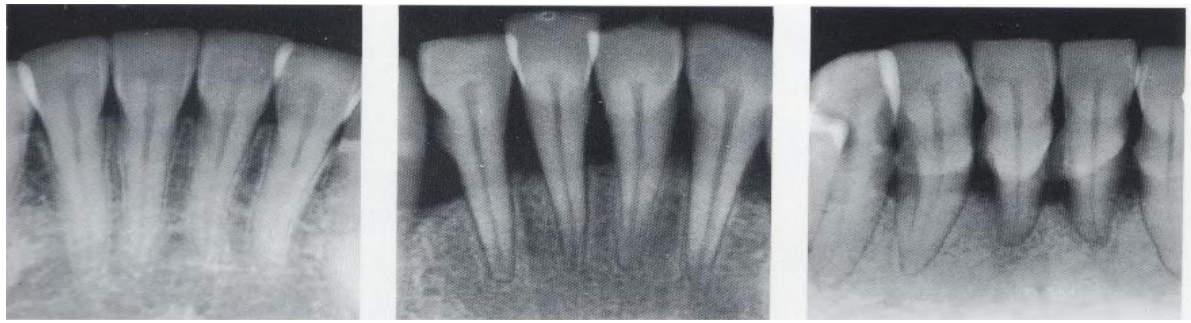


122 Types of pockets

A. Normal sulcus
Apical termination of the JE is at the cementoenamel junction (arrow).

B. Suprabony pocket
Proliferating pocket epithelium. A remnant of junctional epithelium persists (pink).

C. Infrabony pocket
Extends beyond alveolar crest.



123 No attachment loss, normal alveolar septa (left)
Lamina dura remains intact.

124 Horizontal bone loss (middle)
Up to 50% loss of interdental septal bone.

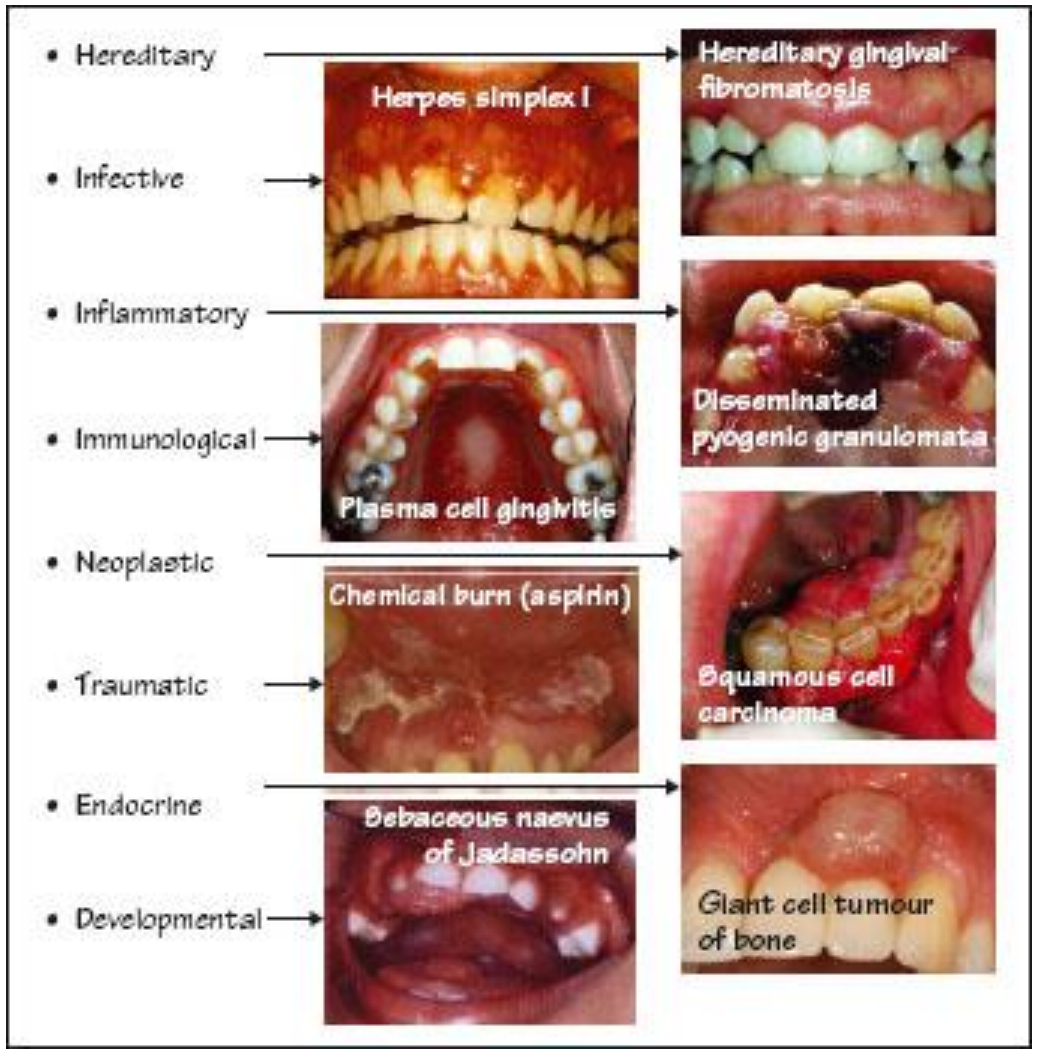
125 Severe horizontal bone loss (right)
Up to 80% bone loss. Heavy calculus accumulation.

126 Vertical bone loss – Craters (left)
Irregular pattern of destruction of alveolar septa. Mesial to the cuspid, evidence of undermining or cyst formation.

127 Vertical bone loss – Furcation involvement (right)
Severe bone loss distal to the first molar. On the same tooth the interradicular bone (furcation) is also involved in the destruction.

Classification of gingival disease

1. Plaque induced gingivitis
2. Non plaque induced gingivitis



Classification of periodontal disease

- Chronic periodontitis (adult periodontitis) localized or generalised
- Aggressive periodontitis (early onset, juvenile periodontitis) localised or generalised

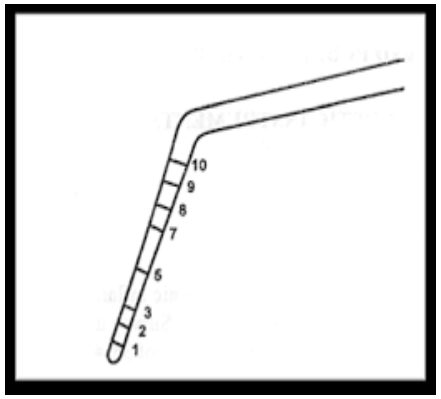
What are the aims of periodontal treatment?

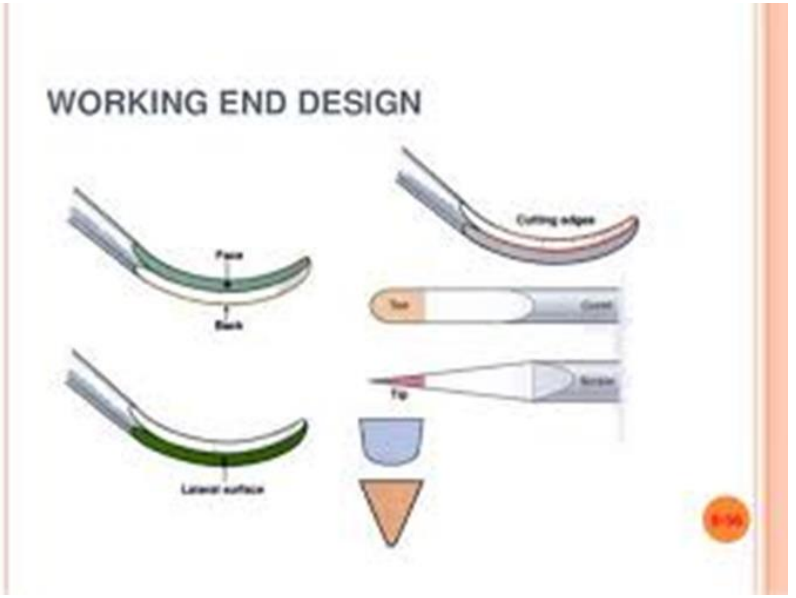
1. Reduction of gingivitis (bleeding on probing, halitosis).
2. Reduction of pocket depth.
3. Cessation (stopping) of pain
4. Individual satisfactory in terms of aesthetic and function

Periodontal treatment can be achieved through:

1. Patient motivation → ??? ???
2. Patient instruction → ??? ???
3. Scaling and polishing
4. Elimination of any plaque retentive factors (defective and overhang fillings, crowns)
5. Orthodontic treatment and occlusal therapy.

Some periodontal instruments





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- Colour Atlas of Dental Medicine and Periodontology