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# Calculus

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***ABSTRACT:***

***Accumulation of dental plaque on teeth and restorative surfaces has been considered as the prime factor for periodontal disease. As the plaque gets mineralised, it ultimately leads to the formation of dental calculus. Calculus is harmful to the gingival health as it serves as niche for the increased plaque formation and retention.Thus, calculus along with other plaque retentive factors is referred to as a secondary etiological factor of periodontal disease.***

**DEFINITON OF CALCULUS:**

- Calculus is a hard deposit that is formed by mineralization of dental plaque on the surfaces of natural teeth and dental prosthesis, generally covered by a layer of unmineralized plaque.

  Carranza's clinical Periodontology (11th ed)

- Calculus is a hard concretion that forms on teeth or dental prosthesis through calcification of bacterial plaque. Glossary of periodontal terms(2001),4th edition.

**History:**

- Calculus was recognised to be associated with periodontal disease as far back as tenth century. [1]
- Van leeuwenhoek (1683)- described microorganisms in tartar ( ANIMALCULES)[2]
- Fauchard (1728) – termed it as TARTAR or SLIME .

**CLASSIFICATION:**

## 1. ACCORDING TO LOCATION:

**Supragingival Calculus:**

It is the tightly adherent calcified deposit that forms on the clinical crowns of the teeth above the free gingival margin and is clinically visible. It is also called as salivary calculus as it forms from the saliva[3]. The other names include:

- Supramarginal
- Extragingival
- Coronal

**Subgingival Calculus:**

It is the calcified deposit that is formed on the root surfaces below the free gingival margin. It is formed from the gingival exudates and hence called as serial calculus.[4] The other names include:

- Submarginal
- Hematogenetic

## 2. ACCORDING TO SURFACE:

**Exogenous And Endogenous**

## 3. According To Initiation And Rate Of Accumulation:

- Non calculus formers
- Slight calculus formers
- Moderate calculus formers
- Heavy calculus formers

**Composition:**

Dental calculus is comprised of 70-90% inorganic salts and 10-30% of organic components.[5,6]

The principal inorganic components include:

- Calcium phosphate -75.9%
- Calcium carbonate -3.1%
- Traces of magnesium phosphate and other metals

The organic components comprise of:

- Salivary proteins- 5.9-8.2%
- Lipids- 0.2%
- Carbohydrates- 1.9-9.1%

At least two thirds of the inorganic components is crystalline in structure. The four main crystal structures are:

1. Hydroxyapatite- 58% appears as sand grains or rod like crystals
2. Magnesium whitlockite-21% as hexagonal crystals in posterior areas
3. Octacalcium phosphate- 21% , platelet like crystals
4. Brushite- 9%, seen in mandibular anterior region[7,8,9,10]

**Supragingival Calculus:**

- Located coronal to the free gingival margin and visible in the oral cavity.
- Hard and clay like in consistency
- White or whitish yellow, which may be due to tobacco stain or food pigments
- Easily detached from the tooth
- The calcium phosphate ratio is less than that in subgingival calculus[11]

**STRUCTURE:**

1. **In Light Microscopy:**

- Calculus tooth interface is smooth
- External mineralized surface is irregular.
- Covered by non mineralized plaque
- Contain many non mineralized lacunae

2. **Transmission Electron Microscopy:**

- The mineralized intermicrobial areas of the body of the calculus contains predominantly small, randomly orientated needle-shaped/platelet-shaped crystals
- Areas containing crystals of larger columnar and roof-tile shapes were also observed [12,13]

**Subgingival Calculus:**

- Located below the crest of the free gingival margin.
- Found by tactile sensation
- Flint like in consistency
- Dark brown or greenish black due to blood and GCF production
- More of magnesium whitlockite and less of brushite and octacalcium phosphate seen.[14]

**STRUCTURE:**

1. **In Light Microscopy:**

- Unlike supra gingival calculus, lacunae of stained organic material were not seen within the body of sub gingival calculus.
- The calculus surface previously in contact with the tooth was flat and mineralised.
- The external surface was fairly regular and covered by a non- mineralized plaque layer of variable thickness.

2. **Transmission Electron Microscopy:**

- The calcification - more homogeneous and consisted of small randomly oriented needle and platelet shaped crystals.
- Areas with flat "bulk-shaped" crystals seen.[15]

**RATE OF FORMATION OF CALCULUS:**

Plaque mineralization begins within 24-72 hrs and takes average of 12 days to mature. Soft plaque is hardened by mineralization between 1$^{st}$ and 14$^{th}$ day. Calcification is reported to occur in 4-8 hours.

Calculus is formed in layers which is often separated by a thin cuticle, that becomes embedded in the calculus.[16,17]

**Phases Of Formation Of Calculus:**

1. **Pellicle Formation**: It consists of enzymes,glycoprotein,proline rich protein,histadin rich protein and phospholipids.
2. **Initial Adhesion And Attachment Of Protein:**
    - Initial transport to the surface
    - Reversible adhesion
    - Firm anchorage by specific interactions
3. **Colonization And Plaque Maturation:**
   Along with the firmly attached microorganism and the newly formed bacterial clusters, microcolonies develop.
   Gram positive coccidal organisms adhere to the formed enamel pellicle and then the filamentous bacteria dominate the mature plaque.
4. MINERALIZATION : The matrix is a form of mucopolysaccharide derived from bacteria or saliva.[18]

**Attachment Of Calculus:**

It includes 4 types:

- Attachment by means of organic pellicle
- Mechanical interlocking into surface irregularities
- Penetration of calculus bacteria into cementum
- Close adaptation of calculus to the unaltered cemental surface.[19]

**THEORIES REGARDING MINERALIZATION OF CALCULUS:**

## 1. Booster/Precipitation Theory:

Loss of carbon dioxide and formation of ammonia leads to the increase in the ph, which leads to the precipitation of calcium phosphate salts.

## 2. Epitactic/Nucleation Concept:

Seeding agents induce small foci of calcification that enlarge and unite together to form calcified mass. The seeding agents are CARBOHYDRATES- PROTEIN COMPLEX.

## 3. Inhibition Theory:

The inhibiting substance is PYROPHOSPHATE. Calcification occurs only at specific sites as there is an existence of inhibiting mechanism at noncalcifying sites. The pyrophosphate inhibits calcification by preventing the initial nucleus from growing, by affecting the growth centre of the crystals.[20]

**Diagnostic Aids :**

1.**Visual Examination:**

- Gentle air blast
- Transillumination
- Gingival tissue colour change

2.**Tactile Examination:**

Probe and explorers.

**Advanced Diagnostic Aids:**

- Fiberoptic endoscopy-based technology- PERIOSCOPE
- Spectro optical technology Novel LED probe- DETECTAR
- Autofluorescence based technology - DIAGNODENT
- Ultrasound technology -PERIOSCAN
- Laser based technology –KEYLASER[21]

**Control Measures:**

- Tooth brushing
- Scaling and root planning
- Lasers: semiconductor diode laser(gallium, aluminium), Nd-YAG lasers, carbon dioxide lasers.[22,23,24,25,26,27,30]

**CONCLUSION:**

While the bacterial plaque that coats the teeth is the main etiologic factor in the development of periodontal disease, the removal of sub gingival plaque and calculus constitute the cornerstone of periodontal therapy.

Calculus plays an important role in maintaining and accentuating periodontal disease by keeping plaque in close contact with the gingival tissue and creating areas where plaque removal is impossible.[28,29] Therefore the clinician must possess the clinical skill to remove the calculus and other irritants as a basis for adequate periodontal and prophylactic therapy.

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