Dental Calculus

The primary cause of gingival inflammation is bacterial plaque, other predisposing factors including calculus, malocclusion, faulty restoration, complications associated with orthodontic therapy, self-inflicted injuries, use of tobacco, and radiation therapy.

Calculus consists of mineralized bacterial plaque that forms on the surface of natural teeth and dental prosthesis and is classified as supragingival or subgingival according to its relation to the gingival margin.

➢ Classification of dental calculus :

- **According to location** :
  - Supragingival calculus
  - Subgingival calculus
- **According to source of mineralization** : (Jenkins, Stewart 1966)
  - Salivary calculus
  - Serumal calculus
- **According to surface** : (Melz 1950)
  - Exogenous
  - Endogenous
- **According to initiation and rate of accumulation**,:

  Calculus formers are classified as:

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

➢ Supra and sub-gingival calculus (distribution and appearance)

❖ **Supragingival calculus**: is located coronal to the gingival margin and therefore visible in the oral cavity, it's usually white to yellowish in color, hard with claylike consistency and easily detected from the tooth surface, after removal, it may rapidly recur, especially in the lingual area of the mandibular incisors, it's color is influenced by contact with such substances as tobacco and food pigments. It may localize on a single tooth or group of teeth or it may be generalized throughout the mouth.

The two most common locations for supragingival calculus to develop are the buccal surface of maxillary molars and the lingual surfaces of mandibular anterior teeth, saliva from the parotid gland flows over the facial surfaces of the upper molars via the parotid duct (**Stensen's duct**), while the submandibular and the sublingual glands empty onto lingual surfaces of lower incisors via the submandibular (**Warhtin's duct**) and sublingual ducts **(Bartholins'duct)** respectively.

❖ **Subgingival calculus:** is located below the crest of the marginal gingiva and therefore not visible on routine clinical examination, the location and extent of the subgingival calculus maybe evaluated by careful tactile perception with a delicate dental instruments such as a dental explorer, subgingival calculus is typically hard and dense, frequently appears dark brown or greenish black in color and firmly attached to the tooth surface.

When the gingival tissue recede, subgingival calculus becomes exposed and its therefore classified as supragingival. A reduction in the gingival inflammation and probing depths with a gain in clinical attachment can be observed after the removal of subgingival plaque and calculus.

Both supra and sub gingival calculus maybe seen by **radiograph**.

➢ Composition

- **Inorganic content :** supragingival calculus consist of inorganic (70% to 90%) and organic components, the major inorganic proportions of calculus have been reported as approximately 76% calcium phosphate $Ca_3(PO_4)$; 3% calcium carbonate $CaCO_3$; traces of magnesium phosphate $Mg_3(PO_4)$ and other metals (Na, Ba, Zn, Str, Br, Cu, Ag, Al, Fe, Fl).

The percentage of inorganic constituents in calculus is similar to that in other calcified tissue of the body, the principle inorganic components have been reported as approximately 39% calcium, 19% phosphorus, 2% carbon dioxide and 1% magnesium and trace amounts of sodium, zinc, strontium, bromine, copper, manganese, tungsten, aluminum, silicon.

At least two thirds of the inorganic component is crystalline in structure, the four main crystal forms and their approximate percentage are as follows: hydroxyapatite 58%, magnesium white-lockite 21%, octacalcium phosphate 12% and brushite 9%.

Generally two or more crystal forms are typically found in the sample of calculus hydroxyapatite and octacalcium phosphate are detected most frequently (i.e. in 97% to 100%of the supragingival calculus) and constitute the bulk of the specimen.

Brushite is more common in the mandibular anterior region and magnesium white-lockite is in the posterior areas, the incidence of the four crystals varies with the age of the deposit.

The composition of subgingival calculus is similar to that of supragingival calculus with some differences, it has the same hydroxyapatite content, more magnesium white-lockit, and less brushite and octacalcium phosphate than supragingival calculus.

- **Organic content:** the organic component of calculus consists of a mixture of protein-polysaccharide complexes, desquamated epithelial cells. Leukocytes and various types of microorganisms.

Salivary proteins present in supragingival calculus are not found subgingivally. The ratio of calcium to phosphate is higher subgingivally and the sodium content increases with the depth of periodontal pockets.

Differences between supragingival vs subgingival calculus			
No.	**Feature**	**Supragingival calculus**	**Subgingival calculus**
1	Defined as	Tightly adhering calculus deposit that forms on the crowns of the teeth coronal to the gingival margin	Calcified deposit that forms on the tooth surface below the free margin of gingiva
2	Location	Forms coronal to the gingival margin	Deposits present apical to the crest of marginal gingiva
3	Source	Derived from the salivary secretions – salivary calculus	Derived from the gingival exudate – serumal calculus
4	Distribution	Symmetrical arrangement on teeth, more on facial surfaces of maxillary molars and lingual surfaces of mandibular anterior teeth	Related to pocket depth, heavier on proximal surfaces
5	Color	It is white, yellow in color	Brown/greenish black in color
6	Consistency	Hard and clay like	Hard and firm/flint or glass like
7	Composition	More brushite and octa calcium phosphate Less magnesium whitelockite	Less brushite and octa calcium phosphate. More magnesium whitelockite
8	Other contents	Sodium content is less Salivary proteins are present	Sodium content increases with the depth of the pocket Salivary proteins are absent
9	Visibility	Clinically visible	Not visible on routine clinical examination
10	Attachment	Easily detached from the tooth	Firmly attached to the tooth surface

➢ Dental calculus attachment

❖ Attachment of calculus to the tooth:

1. By means of organic pellicle on cementum
2. Mechanical locking into surface irregularities such as resorption lacunae.
3. Close adaptation of calculus undersurface depressions to the gently sloping mounds of the unaltered cementum surface.
4. Penetration of calculus bacteria into cementum, calculus may embedded deeply in cementum may appear morphologically similar to cementum and thus termed calculocementum.
5. The access to the area of calculus removal, sub gingival area is more difficult and also the sub gingival calculus is invisible (visibility).

❖ Attachment of calculus on the implant :

Calculus attachment to pure titanium is less intimate than to root surfaces structure. Smooth machined implants have less micro porosities for retention. This would mean that calculus may be chipped off from implants without affecting it.

➢ Formation:

Calculus is a dental plaque that has undergone mineralization; the soft plaque is hardened by precipitation of mineral salts which is usually started between first and fourteenth days of plaque formation.

Calcification started as soon as (4-8 hours), calcifying plaque may become 50% mineralized in 2 days, 60%-90% mineralized in 12 days. However, the formation of dental calculus with the mature crystalline composition of old calculus may require months to years.

All plaque not necessarily undergo calcification, early plaque contains small amount of inorganic material which increase as the plaque develop into calculus.

Microorganisms are not always essential in calculus formation because calculus occurs readily in germ free rodents, saliva is the source of mineralization for supra gingival calculus whereas the serum transudate (Gingival crevicular fluid) is source of mineralization of sub gingival calculus.

Early plaque of patients who have heavy calculus formers contain more calcium, more phosphorus (three times) and less potassium than that of non-calculus formers, (i.e., phosphorus is critical in calculus formation).

Calcification entails the binding of calcium ions to carbohydrates-protein complex of organic matrix and the precipitation of crystalline calcium phosphate salts.

Crystals form initially in the intercellular matrix and on the bacterial surfaces and finally within the bacteria. Calcification begins along the inner surface of the supra gingival plaque and in the attached component of sub gingival plaque adjacent the tooth.

Separate foci of calcification increase in size and coalesce to form solid masses of calculus, the initiation of calcification and rate of accumulation vary among teeth in same individual, so person may be heavy, moderate or slight calculus former.

Calculus formation continues until it reaches maximum after which it reduced in amount due to mechanical wear from food and the cheeks, lip and tongue, also the use of anti-calculus (anti tarter) dentifrices reduce both quality and quantity of calculus.

➢ Theories regarding the mineralization of calculus:

The theoretical mechanisms by which plaque becomes mineralized can be stratified into two categories:

1. Mineral precipitation: results from a local rise in the degree of saturation of calcium and phosphate ions, which may be brought about in the following several ways:

• A rise in the pH of the saliva causes precipitation of calcium phosphate salts by lowering the precipitation constant. The pH may be elevated by the loss of carbon dioxide and the formation of ammonia by dental plaque bacteria or by protein degradation during stagnation.

• Colloidal proteins in saliva bind calcium and phosphate ions and maintain a supersaturated solution with respect to calcium phosphate salts. With stagnation of saliva, colloids settle out and the supersaturated state is no longer maintained, leading to precipitation of calcium phosphate salts.

• Phosphatase liberated from dental plaque, desquamated epithelial cells, or bacteria precipitates calcium phosphate by hydrolyzing organic phosphates in saliva, thus increasing the concentration of free phosphate ions. Esterase is another enzyme that is present in the cocci and filamentous organisms, leukocytes, macrophages, and desquamated epithelial cells of dental plaque. Esterase may initiate calcification by hydrolyzing fatty esters into free fatty acids. The fatty acids form soaps with calcium and magnesium that are later converted into the less-soluble calcium phosphate salts.

2. Seeding agents: induce small foci of calcification that enlarge and coalesce to form a calcified mass. This concept has been referred to as the epitactic concept or more appropriately, hetero- geneous nucleation. The seeding agents in calculus formation are not known, but it is suspected that the intercellular matrix of plaque plays an active role. The carbohydrate-protein complexes may initiate calcification by removing calcium from the saliva (chelation) and binding with it to form nuclei that induce subsequent deposition of minerals.

Other mineralization theories:

• Booster mechanism

• Epitactic concept

• Inhibition theory

• Transformation theory

• Bacterial theory

• Enzymatic theory

➢ Role of microorganisms in mineralization of calculus:

Mineralization of plaque generally starts extracellularly around both gram-positive and gram-negative organisms but may also start intracellularly. Filamentous organisms, diphtheroids and *Bacterionema and Veillonella* species have the ability to form intracellular apatite crystals. Mineralization spreads until the matrix and bacteria are calcified. Bacterial plaque may actively participate in the mineralization of calculus by forming phosphatases, which changes the pH of the plaque and induces mineralization but the prevalent opinion is that these bacteria are only passively involved and are simply the occurrence of calculus like deposits in germ-free animals supports this opinion.

• Etiologic Significance

The non-mineralized plaque on the calculus surface is the principle irritant for initiating gingivitis.

The underlying calcified portion is a significant contributing factor since it provides a fixed nidus for the continued accumulation of plaque and remains it close to gingiva.

Sub gingival calculus may be the product rather than the cause of periodontal pocket; dental plaque starts pocket formation which in turn provides a sheltered area for plaque accumulation. This formed plaque converted to calculus through the mineral precipitation from gingival fluid that increases during inflammation.

Removal of supra and sub gingival plaque and calculus constitute the cornerstone of periodontal therapy, calculus plays an important role in maintaining periodontal

diseases by keeping plaque in close contact with the gingival tissue and creating area where plaque removal is impossible unless we remove calculus. So, it is a secondary etiologic factor for periodontitis and it is the most prominent plaque retentive factor which has to be removed as a basis for adequate periodontal therapy and prophylactic activities.

➢ Other predisposing factors:

- **Iatrogenic Factors:**

Deficiencies in the quality of dental restorations or prostheses are contributing factors to gingival inflammation and periodontal destruction. Inadequate dental procedures that contribute to the deterioration of the periodontal tissues are referred to as iatrogenic factors. Iatrogenic endodontic complications that can adversely affect the periodontium include root perforations, vertical root fractures, and endodontic failures that may necessitate tooth extraction. Characteristics of dental restorations and removable partial dentures that are important to the maintenance of periodontal health include the location of the gingival margin for the restoration. the space between the margin of the restoration and the unprepared tooth, the contour of restorations, the occlusion, materials used in the restoration, the restorative procedure itself, and the design of the removable partial denture. These characteristics are related to the etiology of periodontal disease.

- **Margins of Restorations**

Overhanging margins of dental restorations contribute to the development of periodontal disease by (1) changing the ecologic balance of the gingival sulcus to an area that favors the growth of disease-associated organisms (pre- dominantly gram-negative anaerobic species) at the expense of the health-associated

organisms (predominately gram-positive facultative species)and (2) inhibiting the patient's access to remove accumulated plaque.

Subgingival margins are associated with large amounts of plaque, more severe gingivitis, and deeper pockets. Even high-quality restorations, if placed subgingivally, will increase plaque accumulation, gingival inflammation, and the rate of gingival fluid flow. Margins placed at the level

of the gingival crest induce less severe inflammation, whereas supragingival margins are associated with a degree of periodontal health similar to that seen with non restored interproximal.

- **Contours/Open Contacts**

Overcontoured crowns and restorations tend to accumulate plaque and possibly prevent the self- cleaning mechanisms of the adjacent cheek, lips, and tongue. Restorations that fail to reestablish adequate interproximal embrasure spaces are associated with papillary inflammation.

- **Design of Removable Partial Dentures**

Several investigations have shown that after the insertion of partial dentures, the mobility of the abutment teeth, gingival inflammation, and periodontal pocket formation all increase. This is because partial dentures favor the accumulation of plaque, particularly if they cover the gingival tissue. Partial dentures that are worn during both night and day induce more plaque formation than those worn only during the daytime. These observations emphasize the need for careful and personalized oral hygiene instruction to avoid harmful effects of partial dentures on the remaining teeth and periodontium. The presence of removable partial dentures induces not only quantitative changes in dental plaque but also qualitative changes, promoting the emergence of spirochetal microorganisms.

➢ Prevention:

The best way to prevent tartar is by **taking good care of your oral health**.

You can do this by:

- Brushing your teeth twice a day for two minutes at a time and flossing daily
- Using an electric toothbrush, replacing the bristles every 3 months
- Using a fluoride toothpaste
- Visiting your dentist every 6 months for routine checkups and cleanings
- Eating a healthy balanced diet, and limiting the consumption of sugars and starches
- Avoiding smoking or chewing tobacco.

➢ Removal

Tartar buildup bonds strongly to tooth enamel and must be removed by a dental professional. This can be done with a professionalcleaning, debridement, or scaling and root planning. If you do not have dense deposits and your teeth have not been affected by bone loss or periodontal disease, getting a regular dental cleaning every six months is recommended by the American Dental Association. At your regular dental checkup and cleaning, your dentist or dental hygienist will use a scaler to remove any dental plaque, tartar, and stains around your gums and between your teeth.

A debridement is recommended for people who have dense calculus on their teeth that interferes with the dentist's ability to perform a comprehensive oral evaluation. Ultrasonic instruments and hand tools are used to fracture tartar and remove it.

If you have pockets between your teeth and gums, or periodontal (gum) disease you may need scaling and root planning, which is a deep cleaning. This procedure is performed using local anesthesia, and plaque and tartar are removed above and

below the gumline, all the way down to the bottom of the pocket. Afterwards, the teeth roots are smoothed out to help your gums reattach to your teeth.