The Role of Dental Calculus
CALCULUS

- Calculus consists of mineralized bacterial plaque that forms on the surfaces of natural teeth and dental prostheses.
- Calculus is classified as supragingival or subgingival, according to its relation to the gingival margin.
Supragingival and Subgingival Calculus
Supragingival Calculus

- Supragingival calculus is located coronal to the gingival margin and therefore is visible in the oral cavity.
- It is usually white or whitish yellow in color, hard with clay-like consistency, and easily detached from the tooth surface. The 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. After removal, it may rapidly recur, especially in the lingual area of the mandibular incisors.
• The **two most common locations** for supragingival calculus to develop are the **buccal surfaces of the maxillary molars and the lingual surfaces of the mandibular anterior teeth**.

• Saliva from the parotid gland flows over the facial surfaces of upper molars via Stensen's duct, whereas Wharton's duct and Bartholin's duct empty onto the lingual surfaces of the lower incisors from the submaxillary and sublingual glands, respectively.
Subgingival Calculus

- Subgingival calculus is located below the crest of the marginal gingiva and therefore is not visible on routine clinical examination. The location and extent of subgingival calculus may be evaluated by careful tactile perception with a delicate dental instrument such as an explorer.
• Subgingival calculus is **typically hard and dense** and frequently appears **dark brown or greenish black** in color while being firmly attached to the tooth surface.

• Suprangingival calculus and subgingival calculus generally occur together, but one may be present without the other.

• Microscopic studies demonstrate that deposits of subgingival calculus usually extend nearly to the base of periodontal pockets in chronic periodontitis but do not reach the junctional epithelium.
• When the gingival tissues recede, subgingival calculus becomes exposed and is therefore reclassified as supragingival. Thus supragingival calculus can be composed of both supragingival calculus and previous subgingival calculus.

• A reduction in gingival inflammation and probing depths and a gain in clinical attachment can be observed after the removal of subgingival plaque and calculus.
Inorganic Content.

- Supragingival calculus consists of inorganic (70% to 90%) and organic components.
- The inorganic portion consists of 75.9% calcium phosphate, Ca$_3$(PO$_4$)$_2$; 3.1% calcium carbonate, CaCO$_3$; and traces of magnesium phosphate, Mg$_3$(PO$_4$)$_2$, and other metals. The percentage of inorganic constituents in calculus is similar to that in other calcified tissues of the body.
- The principal inorganic components are calcium, 39%; phosphorus, 19%; carbon dioxide, 1.9%; magnesium, 0.8%; and trace amounts of sodium, zinc, strontium, bromine, copper, manganese, tungsten, gold, aluminum, silicon, iron, and fluorine.
At least two thirds of the inorganic component is crystalline in structure. The four main crystal forms and their percentages are as follows:

- Hydroxyapatite, approximately 58%
- Magnesium whitlockite, approximately 21%
- Octacalcium phosphate, approximately 12%
- Brushite, approximately 9%
• Generally, two or more crystal forms are typically found in a sample of calculus.
  • Hydroxyapatite and octacalcium phosphate are detected most frequently (i.e., in 97% to 100% of all supragingival calculus) and constitute the bulk of the specimen.
  • Brushite is more common in the mandibular anterior region and
    • magnesium whitlockite in the posterior areas.
• The incidence of the four crystal forms varies with the age of the deposit.
Organic Content.

- The organic component of calculus consists of a mixture of protein-polysaccharide complexes, epithelial cells, leukocytes, and various types of microorganisms.

- Between 1.9% and 9.1% of the organic component is carbohydrate, which consists of galactose, glucose, glucuronic acid, galactosamine, and sometimes, galacturonic acid, and glucosamine.
• All these organic components are present in salivary glycoprotein with the exception of arabinose and rhamnose.

• Salivary proteins account for 5.9% to 8.2% of the organic component of calculus and include most amino acids.

• Lipids account for 0.2% of the organic content in the form of neutral fats, free fatty acids, cholesterol, cholesterol esters, and phospholipids.
• The composition of subgingival calculus is similar to that of supragingival calculus, with some differences. It has the same hydroxyapatite content, more magnesium whitlockite, and less brushite and octacalcium phosphate.

• The ratio of calcium to phosphate is higher subgingivally, and the sodium content increases with the depth of periodontal pockets.

• Salivary proteins present in supragingival calculus are not found subgingivally. Dental calculus, salivary duct calculus, and calcified dental tissues are similar in inorganic composition.
Attachment to the Tooth Surface

- Four modes of attachment have been described:
  - attachment by means of an organic pellicle;
  - mechanical locking into surface irregularities such as resorption lacunae and caries;
  - close adaptation of calculus undersurface depressions to the gently sloping mounds of the unaltered cementum surface;
  - penetration of calculus bacteria into cementum.

- However, not all investigators universally acknowledge this forth mode of attachment. Calculus embedded deeply in cementum may appear morphologically similar to cementum and thus has been termed calculocementum.
Calculus. A, Calculus attached to pellicle on enamel surface (e). The enamel was removed in the preparation of the specimen. Also note calculus attached to dentin and associated penetration of dental tubules (arrows). B, Interproximal area with early and advanced root caries of adjacent teeth and with calculus attached to carious surfaces (arrows).
Calculus on tooth surface embedded within the cementum (C). Note the early stage of penetration shown in the lower portion of the illustration. D, Dentin; P, plaque attached to calculus.
Undersurface of subgingival calculus (C) previously attached to the cementum surface (S). Note impression of cementum mounds in calculus (arrows)
Subgingival calculus (C) embedded beneath the cementum surface (arrows) and penetrating to the dentin (D), making removal difficult.
Formation

- Calculus is dental plaque that has undergone mineralization. The soft plaque is hardened by the precipitation of mineral salts, which usually starts between the 1st and 14th days of plaque formation. However, calcification has been reported to occur in as little as 4 to 8 hours.

- Calcifying plaques may become 50% mineralized in 2 days and 60% to 90% mineralized in 12 days.
• All plaque does not necessarily undergo calcification. Early plaque contains a small amount of inorganic material, which increases as the plaque develops into calculus. Plaque that does not develop into calculus reaches a plateau of maximal mineral content within 2 days. 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 called **gingival crevicular fluid** furnishes the minerals for **sub**gingival calculus.

• **Plaque** has the ability to **concentrate calcium at 2 to 20 times** its level in saliva.

• Early plaque of heavy calculus formers contains more calcium, three times more phosphorus, and less potassium than that of non-calculus formers, suggesting that phosphorus may be more critical than calcium in plaque mineralization.

• Calcification entails the binding of calcium ions to the carbohydrate-protein complexes of the 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 supragingival plaque and in the attached component of subgingival plaque adjacent to the tooth.

• Calculus is formed in layers, which are often separated by a thin cuticle that becomes embedded in the calculus as calcification progresses.
Calcification may be accompanied by alterations in the bacterial content and staining qualities of the plaque. As calcification progresses, the number of filamentous bacteria increases and foci of calcification change from basophilic to eosinophilic. There is a reduction in the staining intensity of groups exhibiting a positive periodic acid-Schiff reaction. Sulfhydryl and amino groups also are reduced and instead stain with toluidine blue, which is initially orthochromatic but becomes metachromatic and disappears.
• The initiation of calcification and the rate of calculus accumulation vary from person to person, for different teeth, and at different times in the same person. On the basis of these differences, persons may be classified as heavy, moderate, or slight calculus formers or as non-calculus formers. The average daily increment in calculus formers is from 0.10% to 0.15% of dry weight. Calculus formation continues until it reaches a maximum, after which it may be reduced in amount. The time required to reach the maximal level has been reported as 10 weeks and 6 months.

• The decline from maximal calculus accumulation, referred to as reversal phenomenon, may be explained by the vulnerability of bulky calculus to mechanical wear from food and from the cheeks, lips, and tongue.
Theories Regarding the Mineralization of Calculus

- The *theoretic mechanisms* by which plaque becomes mineralized can be stratified into two principal 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 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, epithelial cells, or bacteria precipitate 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 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, heterogeneous 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 and binding with it to form nuclei that induce subsequent deposition of minerals.
Role of Microorganisms in the Mineralization of Calculus

- Mineralization of plaque starts extracellularly around both gram-positive organisms and gram-negative organisms; it may also start intracellularly. Filamentous organisms, and Bacterionema and Veillonella species have the ability to form intracellular apatite crystals. Calculus formation 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 calcified with other plaque components.

• The occurrence of calculus-like deposits in germ-free animals supports this opinion. However, other experiments suggest that transmissible factors are involved in calculus formation and that penicillin in the diets of some of these animals reduces calculus formation.
Etiologic Significance

- Distinguishing between the effects of calculus and plaque on the gingiva is difficult because calculus is always covered with a nonmineralized layer of plaque.

- A positive correlation between the presence of calculus and the prevalence of gingivitis exists, but this correlation is not as great as that between plaque and gingivitis. In young persons, periodontal condition is more closely related to plaque accumulation than to calculus, but the situation is reversed with age. The incidence of calculus, gingivitis, and periodontal disease increases with age. *It is extremely rare to find periodontal pockets in adults without subgingival calculus,* although subgingival calculus may be of microscopic proportion in some cases.
• The nonmineralized plaque on the calculus surface is the principal irritant, but the underlying calcified portion may be a significant contributing factor. It does not irritate the gingiva directly but provides a fixed nidus for the continued accumulation of plaque and retains it in close proximity to the gingiva.

• Subgingival calculus may be the product rather than the cause of periodontal pockets. Plaque initiates gingival inflammation, which starts pocket formation, and the pocket in turn provides a sheltered area for plaque and bacterial accumulation. The increased flow of gingival fluid associated with gingival inflammation provides the minerals that convert the continually accumulating plaque into subgingival calculus.
• Over a 6-year period, Albander et al observed 156 teenagers with histories of aggressive periodontitis. They noted that areas with detectable subgingival calculus at the initiation of the study were much more likely to experience loss of periodontal attachment than sites that did not initially exhibit subgingival calculus.
• While the bacterial plaque that coats the teeth is the main etiologic factor in the development of periodontal disease, the removal of subgingival 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. Therefore the clinician must not only possess the clinical skill to remove the calculus and other irritants that attach to the teeth but also be very conscientious about performing this task.
Any Questions ?