Department of Periodontology **Dr. Zaid Mohannad Yasser**

Dental Calculus

The primary cause of gingival inflammation is bacterial plaque. Other predisposing factors include calculus, faulty restorations, complications associated with orthodontic therapy, self-inflicted injuries, and the use of tobacco. **Calculus** consists of mineralized bacterial plaque that forms on the surfaces of natural teeth and dental prostheses.

☒ **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

☒ **According to its location divided into supragingival and subgingival calculus.**

1-Supragingival calculus is located above the gingival margin and therefore is visible in the oral cavity. It is usually white or whitish yellow in color; hard, with a claylike consistency; and easily detached from the tooth surface. After removal, it may rapidly recur, especially in the lingual area of the mandibular incisors. The color is influenced by contact with substances such as tobacco and food pigments. It may be localized on a single tooth or group of teeth, or it may be generalized throughout the mouth.

The two most common locations for the development of supragingival calculus are the buccal surfaces of the maxillary molars **(Fig.1)** and the lingual surfaces of the mandibular anterior teeth **(Fig.2)…**……why ?

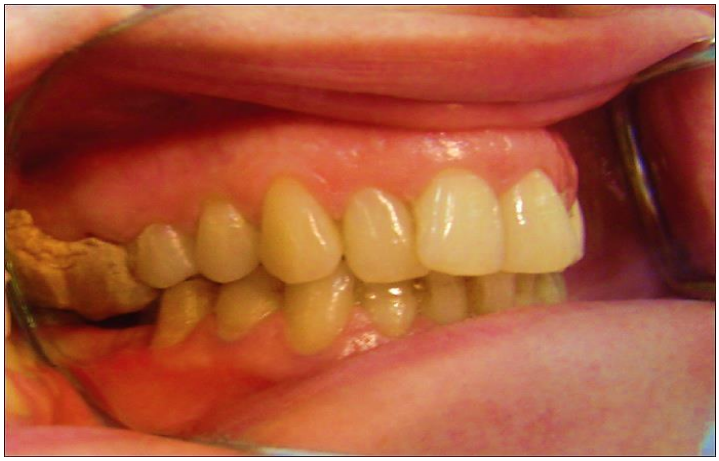

Fig.1:- Supragingival calculus is represented on the buccal surfaces of maxillary molars.

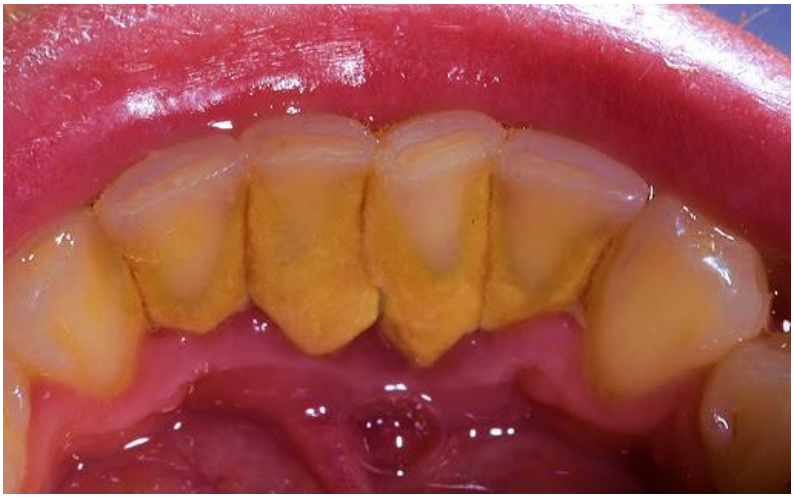

Fig.2:- supragingival calculus is present on the lingual surfaces of the lower anterior teeth.

The saliva from the parotid gland flows over the buccal surfaces of the upper molars via the parotid duct **(Stensen's duct)**, whereas the submandibular duct **(Wharton's duct)** and the lingual duct **(Bartholin duct)** empty onto the lingual surfaces of the lower incisors from the submandibular (submaxillary) and sublingual glands, respectively **(Fig.3).**

In extreme cases, calculus may form a bridge-like structure over the interdental papilla of adjacent teeth or cover the occlusal surface of teeth that are lacking functional antagonists.

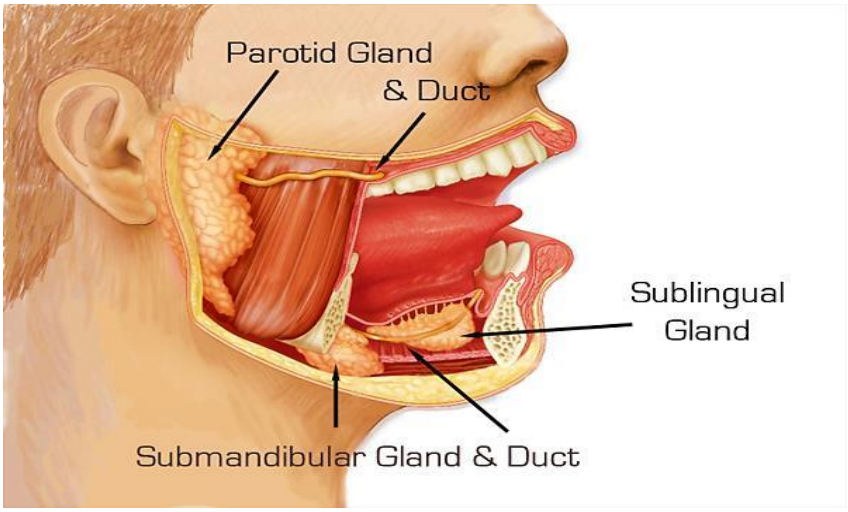

Fig.3:- Anatomy of the parotid & submandibular glands & ducts

2-Subgingival calculus is located below the gingival margin and therefore is not visible on routine clinical examination. The location and the extent of subgingival calculus may be evaluated by careful tactile perception with a delicate dental explorer. Subgingival calculus is typically hard and dense; it frequently appears to be dark brown or greenish black in color, and it is firmly attached to the tooth surface **(Fig.4).**

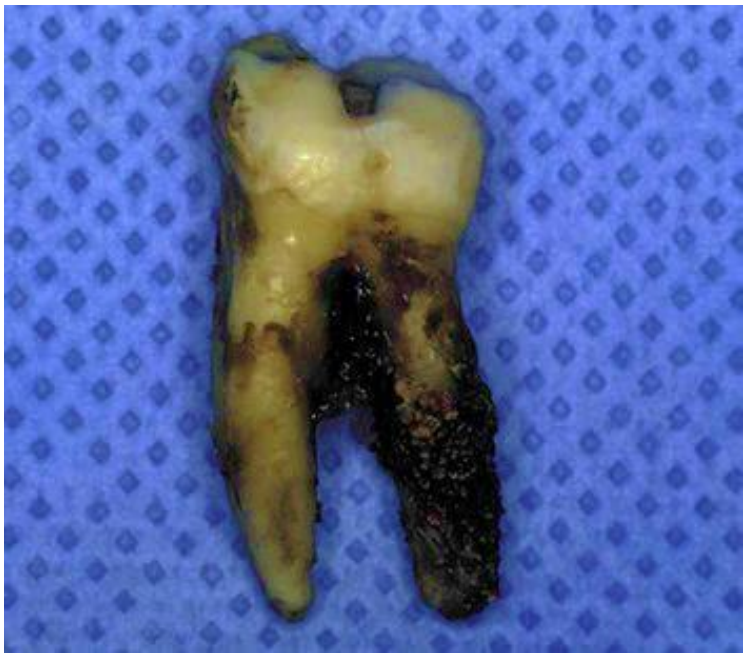

Fig.4:- Dark pigmented deposits of subgingival calculus are shown on the distal root of an extracted lower molar.

When the gingival tissues recede, subgingival calculus becomes exposed and is therefore **reclassified** as supragingival. Thus, supragingival calculus can be composed of both the initial supragingival calculus and previous subgingival calculus. A reduction in gingival inflammation and probing depths with a gain in clinical attachment can be observed after the removal of subgingival plaque and calculus.

Note :-Both supragingival calculus and subgingival calculus may be seen on radiographs.

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 octacalcium 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

Composition of the dental calculus

Inorganic Content

Dental calculus is primarily composed of inorganic components **(70% - 90%)** and the rest is organic components. The major inorganic proportions of calculus are approximately **76%** calcium phosphate (Ca3[PO4]2), **3%** calcium carbonate (CaCO3), **4%** magnesium phosphate (Mg3[PO4]2), **2%** carbon dioxide, and traces of other elements such as sodium, zinc, strontium, bromine, copper, manganese, tungsten, gold, aluminum, silicon, iron, and fluorine. The percentage of inorganic constituents in calculus is similar to that of other calcified tissues of the body **(Table 1).**

Table 1:- Calculus versus other oral hard tissues.

Structure	Inorganic Content (%)*
Dental Calculus	**70–90**
Enamel	**96**
Dentin	**45**
Bone	**60–70**

***Organic components and water constitute the rest.**

At least two-thirds of the inorganic component is crystalline in structure. **The four main crystal forms** and their approximate percentages are as follows: **(hydroxyapatite 58% ; magnesium whitlockite 21%; octacalcium phosphate 12%; and brushite 9%).** 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).Brushite is more common in the mandibular anterior region, and magnesium whitlockite is found 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, desquamated epithelial cells, leukocytes, and various types of microorganisms. Between 1.9% and 9.1% of the organic component is carbohydrate. All of these organic components are present in salivary glycoprotein, 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 but more magnesium whitlockite and less brushite and octacalcium phosphate. The ratio of calcium to phosphate is higher in subgingival calculus, and the sodium content increases with the depth of periodontal pockets. These different compositions may be attributed to fact that the origin of subgingival calculus is plasma, whereas supragingival calculus is partially composed of salivary constituents. Salivary proteins present in supragingival calculus are not found in subgingival calculus.

Attachment to the Tooth Surface:- Four modes of attachment have been defined:-

(1) Attachment by organic pellicle on cementum **(Fig.5).**
(2) Attachment on enamel **(Fig.5).**
(3) Mechanical locking into surface irregularities, such as caries lesions or resorption lacunae **(Fig.6).**
(4) Close adaptation of the undersurface of calculus to depressions or gently sloping surfaces of the unaltered cementum surface and penetration by bacterial calculus into cementum.
(5) Penetration of calculus bacteria into cementum, calculus may embedded deeply in cementum may appear morphologically similar to cementum and thus termed **calculocementum**.

- **The access to the area of calculus removal, subgingival area is more difficult and also the subgingival calculus is invisible (visibility).**

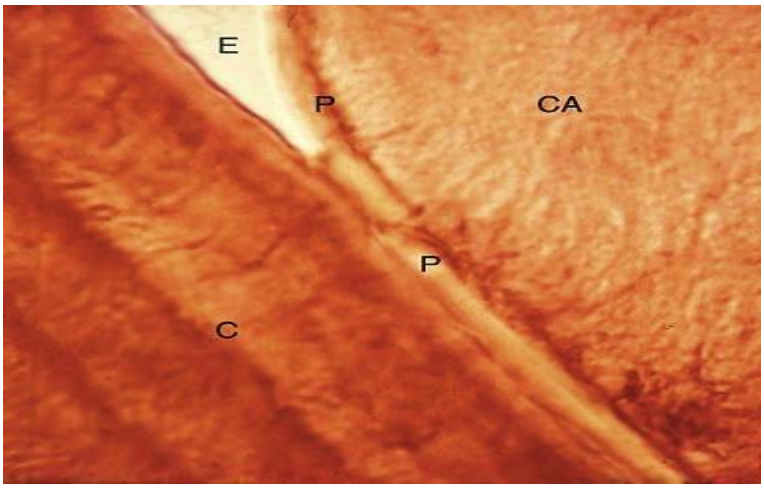

Fig.5:- Calculus attached to the pellicle on the enamel surface and the cementum.

enamel (E). Cementum (C). calculus (CA), pellicle (P).

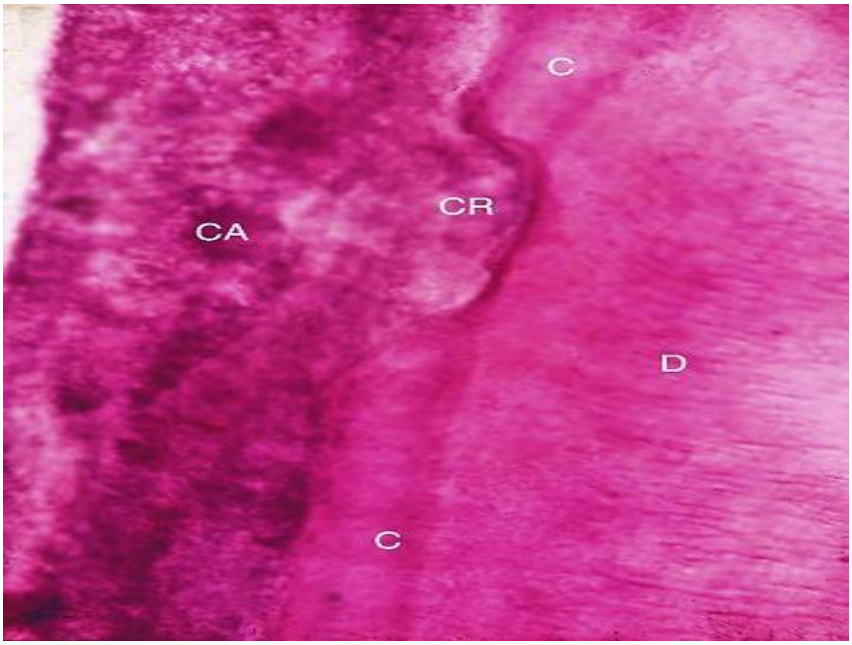

Fig.6:- Calculus (CA) attached to a cemental resorption area (CR) with cementum (C) adjacent to dentin (D).

Formation of the dental calculus

Calculus is mineralized dental plaque. The soft plaque is hardened by the precipitation of mineral salts, which usually **starts** between the 1st and 14th days of plaque formation. Calcification has been reported to occur within 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.

Saliva is the primary source of mineralization for supragingival calculus, whereas the **serum transudate called gingival crevicular fluid** provides the minerals for subgingival calculus. The calcium concentration or content in plaque is **2 to 20** times higher than 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 for plaque mineralization. **Calcification involves** 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. The calcification of supragingival plaque and the attached component of subgingival plaque begins

along the inner surface adjacent to the tooth structure. Separate foci of calcification increase in size and combine to form solid masses of calculus.

The initiation of calcification and the rate of calculus accumulation vary among individuals, among tooth variety in the same dentition, 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. 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 to be between 10 weeks and 6 months. The decline from maximal calculus accumulation, which is referred to as the **reversal phenomenon**, may be **explained** by the susceptibility of bulky calculus to mechanical wear from food and from the cheeks, lips, and tongue movement. Also the use of anti-calculus (anti tarter) dentifrices reduce both quality and quantity of calculus.

Theories of Calculus Formation

It can be explained mainly under 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 several ways:

a. Booster mechanism: According to this theory, precipitation of calcium phosphate salts results from a local rise in the pH of the saliva. Factors such as loss of carbon dioxide and production of ammonia could lead to rise in pH .

b. Colloidal proteins in saliva bind to calcium and phosphate ions thus producing a super-saturated solution. When saliva stagnates in the oral cavity, colloids settle and result in the precipitation of calcium and phosphorous salts.

c. Phosphatase liberated from dental plaque, desquamated epithelial cells, or bacteria precipitate calcium phosphate by hydrolyzing organic phosphates in saliva, thus increasing the concentration of free phosphate ions.

2. Seeding agents induce small foci of calcification that enlarge and combine to form a calcified mass. This concept has been referred to as the **epitactic concept** or, more appropriately, **as 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 **(chelation)** and binding with it to form nuclei that induce the subsequent deposition of minerals.

Other mineralization theories:

- ☒ **Booster mechanism**
- ☒ **Epitactic concept**
- ☒ **Inhibition theory**
- ☒ **Transformation theory**
- ☒ **Bacterial theory**
- ☒ **Enzymatic theory**

Role of Microorganisms in the Mineralization of Calculus

Mineralization of plaque generally starts extracellular around both gram-positive and gram-negative organisms, but it may also start intracellular. Filamentous organisms, diphtheroids, and Bacterionema and Veillonella species have the ability to form intracellular apatite crystals. Mineralization spreads until the matrix and the bacteria are calcified. Bacterial plaque may actively participate in the mineralization of calculus by forming phosphatases, which change the pH of the plaque and induce mineralization, but the **prevalent opinion** is that 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.

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. Subgingival 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.