DENTAL CALCULUS

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CONTENTS

- INTRODUCTION
- COMPOSITION AND STRUCTURE OF DENTAL CALCULUS
- THE DISTRIBUTION OF DENTAL CALCULUS IN THE DENTITION
- ULTRASTRUCTURE OF DENTAL CALCULUS AND ASSOCIATED BACTERIA
- FORMATION OF DENTAL CALCULUS
- THEORIES OF MINERALIZATION
    - BOOSTER MECHANISM
    - EPITAXIC THEORY
    - INHIBITION THEORY
    - TRANSFORMATION
    - BACTERIOLOGICAL THEORY
    - ENZYMATIC THEORY

- MECHANISM OF PLAQUE AND MICROBIAL MINERALIZATION
  - (A) ADSORPTION OF SALIVARY PROTEINS
  - (B) MICROBIAL ADHERENCE
    - (1) Surface proteins and bacterial adherence
    - (2) Microbial co-aggregation and co-adhesion
    - (3) New behavior pattern of attached bacteria
    - (4) Microbial adherence to implant surfaces
  - (C) THE DRIVING FORCE FOR PLAQUE MINERALIZATION
  - (D)THE INVOLVEMENT OF BACTERIA IN CALCULUS FORMATION
  - (E) MICROBIAL MINERALIZATION
  - (F) NUCLEATION INHIBITORS
  - (G) CRYSTAL GROWTH INHIBITORS
  - (H) ORGANIC ACID AND CALCULUS FORMATION
  - (I) ENZYMES DEGRADING CALCIFICATION INHIBITORS

(J) CALCIFICATION PROMOTERS
(1) UREA
(a) The effect of urea metabolism on plaque pH
(b) The effect of salivary flow rate on urea-dependent pH response in plaque
(c) Bacteria responsible for the ureolysis in dental plaque
(2) FLUORIDE AND CALCULUS FORMATION
(3) SILICON

- CALCULUS ATTACHMENT
- ANTI-CALCULUS AGENTS USED IN COMMERCIAL DENTIFRICES
  (A) TRICLOSAN WITH A PVM/MA COPOLYMER
  (B) PYROPHOSPHATE AND PVM/MA COPOLYMER
  (C) ZINC ION
  (D) PREVENTION OF CALCULUS FORMATION IN IMPLANT PATIENTS
- FUTURE RESEARCH ON CALCULUS
- REFERENCES

INTRODUCTION

- The oral cavity is the site of a variety of abnormal calcified deposits, the most common oral deposits however form on the teeth.
- The role of calcified and uncalcified plaque on teeth as primary etiologic factor in inflammatory periodontal diseases has been demonstrated by epidemiologic , experimental and clinical research.
- Dental plaque has been demonstrated to be the major initiating factor in the development of gingivitis and with these hard deposits playing a role in maintaining and promoting the formation of plaque.

DEFINITION

Calculus is an adherent calcified or calcifying mass that forms on the surface of natural teeth or dental prosthesis.

Calculus is essentially mineralized plaque covered on its external surface by vital tightly adherent non mineralized plaque.

HISTORY

- **Hippocrates (460 – 337 BC)**
  - association of oral deposits and disease
  - pituita (calculus)

- **Albucasis (936 – 1013 AD)**
  - Arabian physician and surgeon
  - Explained relationship between calculus and disease
  - A need for thorough removal of deposits

- **Paracelsus (1535)**
  - Swiss German physician and alchemist
  - Introduced term tartar as a designation for a variety of stony concretions that form in humans
  - Tartaric disease

CLASSIFICATION

- According to **location**
  - Supragingival calculus
  - Subgingival calculus

- According to **source of mineralization**
  - Salivary calculus
  - Serumal calculus (Jenkins, Stewart 1966)

- According to **surface**
  - Exogeneous
  - Endogeneous (Melz 1950)

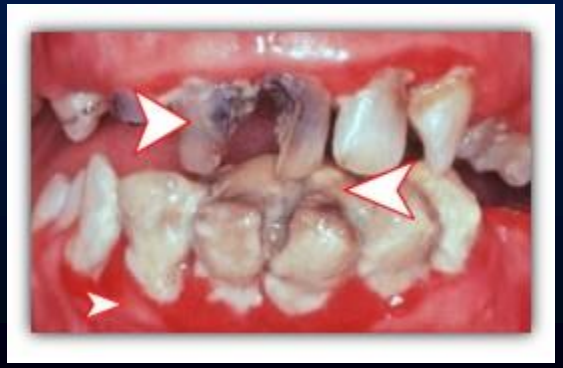
Subgingival calculus

Further subclassified into

- Crusty, spiny or nodular
- Ring like or ledge like encircling the tooth
- Veneer type
- Fingerlike or fern like extensions
- Individual islands or spots
- Combination of the above

SUPRAGINGIVAL CALCULUS

- **Location** -The tightly adherent calcified deposites that form on the clinical crowns of the teeth above the free gingival margin.(visible in oral cavity)
- **Color** -usually white-yellow in colour but can darken with age and exposure to tobacco
- **Texture and consistency** –hard clay like consistency. Easily detachable from tooth surface Salivary secretions are the main source of mineral salts.



Composition

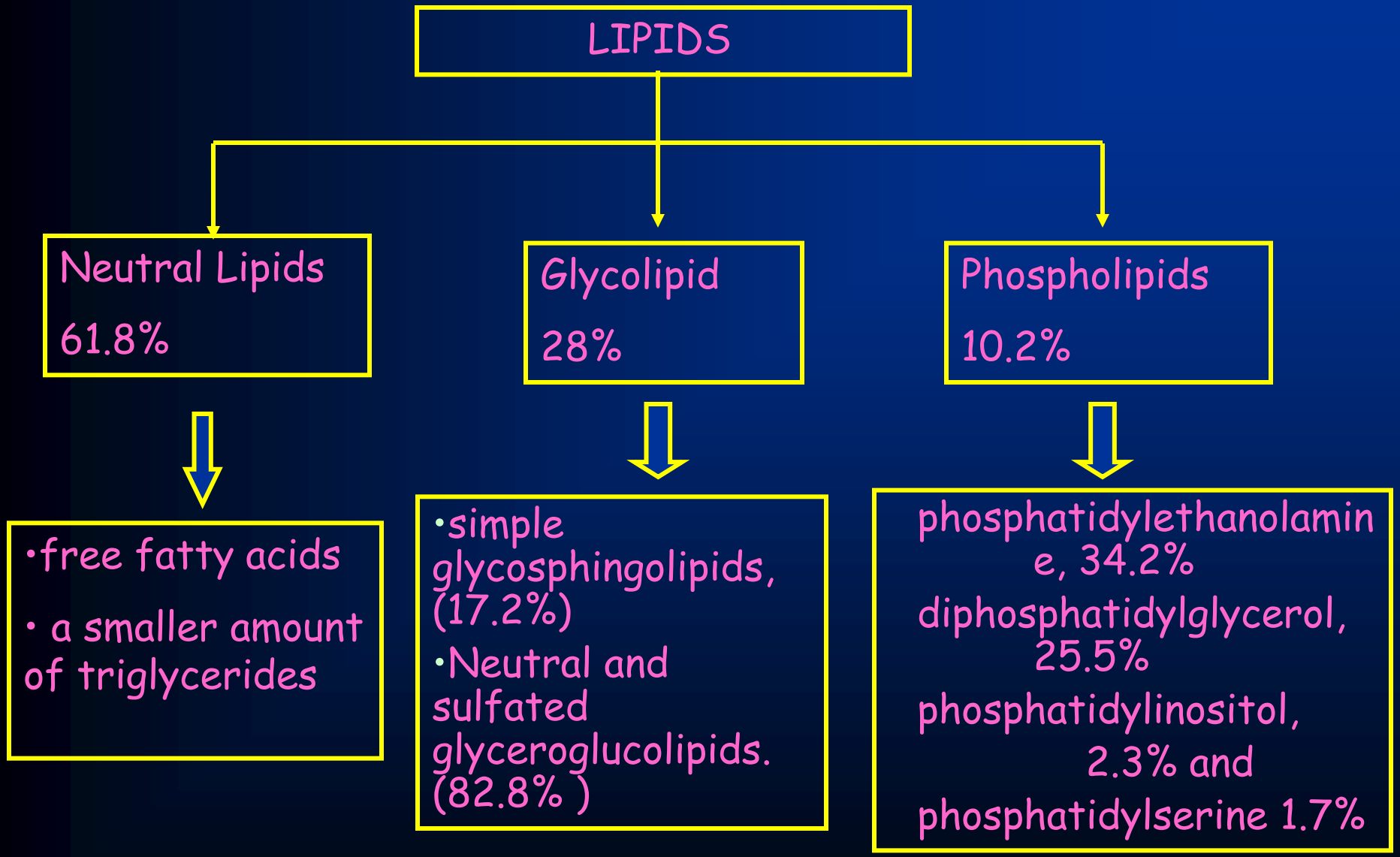
Inorganic :	**80% of dry weight**
Calcium	27 – 29%
Phosphorous	16 – 18%
Carbonate	2 – 3%
Sodium	1.5 – 2.5%
Magnesium	0.6 – 0.8%
Fluoride	0.003 –.04%

Traces of Na, Zn, St, Cu, Br, Mg, Fe, Al, Si, Tg, Au.
(Little et al 1963 Lundberg et al 1966 Schroeder 1969)

Organic:

Total Matrix 15-20%

contains 54.9% protein and 10.2% lipid.


LIPIDS
Neutral Lipids
61.8%
Glycolipid
28%
Phospholipids
10.2%
•free fatty acids
• a smaller amount of triglycerides
•simple glycosphingolipids, (17.2%)
•Neutral and sulfated glyceroglucolipids. (82.8%)
phosphatidylethanolamine, 34.2%
diphosphatidylglycerol, 25.5%
phosphatidylinositol, 2.3% and
phosphatidylserine 1.7%

Crystals

- Octacalcium phosphate [$Ca_8(PO_4)_4(HPO_4)_2 5HO$] (OCP),
- Hydroxyapatite [$Ca_{10}(PO_4)_6(OH)_2$] (HAP),
- ß-tricalcium phosphate or whitlockite [$Ca_{10}(HPO_4)(PO_4)_6$] (WHT) form the inorganic part of both supragingival and subgingival calculus.
- Brushite [$CaHPO_4$-$2H_2O$: dicalcium phosphate dihydrate] (DCPD) is present only in the early-stage supragingival calculus (Rowles, 1964)..

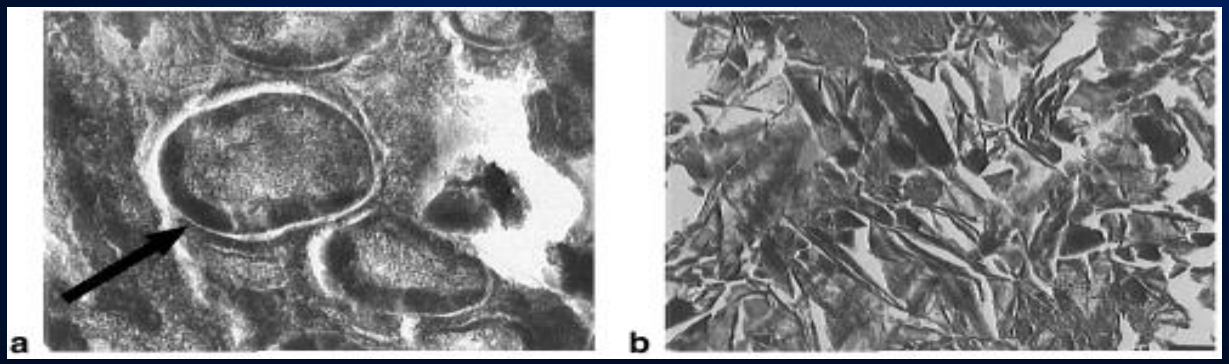
Light Microscopy

- The interface with the tooth surface was fairly smooth and slightly curved following the shape of the tooth whereas the external mineralized surface was generally irregular and covered by a non-mineralized plaque layer of variable thickness.

- containing many non-mineralized lacunae and, in some sections, the lacunae formed a continuous connection with the external bacterial plaque, and extended to the **calculus**/tooth interface

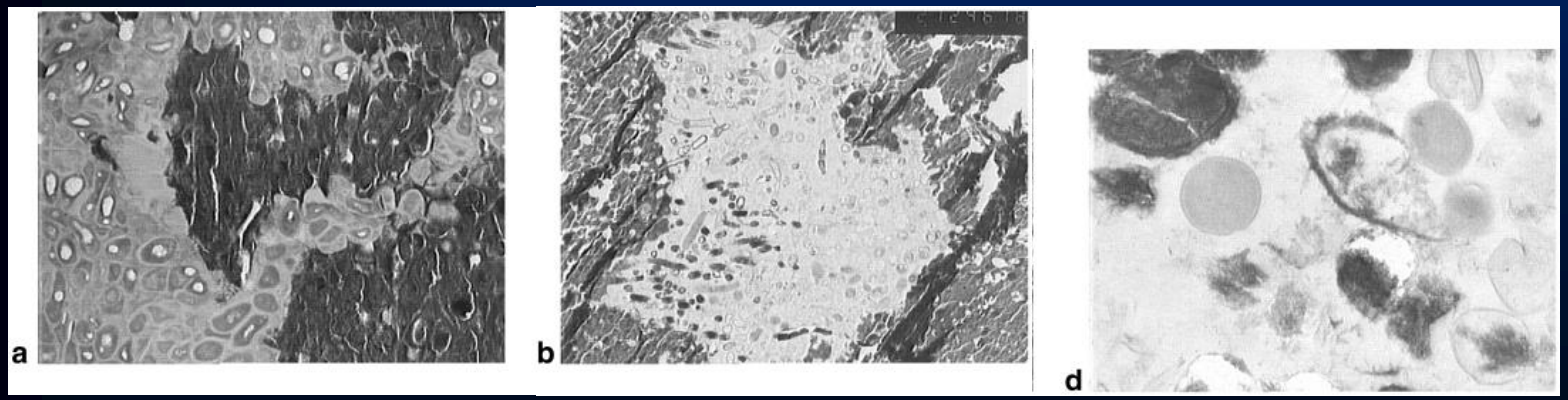
Transmission Electron Microscopy

- The ultra structure of young and mature supragingival **calculus** was similar. The mineralized inter-microbial areas of the body of the **calculus** contained predominantly small, randomly orientated needle-shaped/platelet-shaped crystals. Areas containing crystals of larger columnar and roof-tile shapes were also observed



- Channels of organic matrix containing non-mineralised bacteria were often observed extending into the **calculus** from the **calculus**/plaque interface.

- Individual mineralised bacteria could also be observed within these generally non-mineralised areas.



Scanning Electron Microscopy

- Supragingival calculus always appeared in one piece with a relatively smooth surface.
- Under low magnification the Supragingival calculus was thin at the incisal/occlusal part and thickened apically.
- Under high magnification the Supragingival calculus is covered with dense layer of filaments. These filaments were 1 micron thick with an estimated length of 25 to 100 microns.
- Fracture surface of Supragingival calculus generally had a smooth, crystalline appearance.

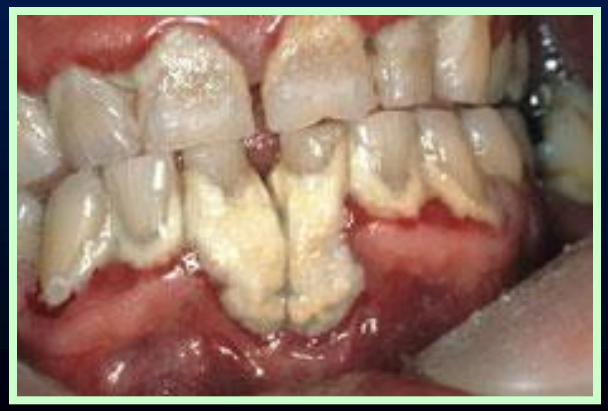
X-ray Beam Diffraction Analysis

Distribution of calcium phosphate compounds

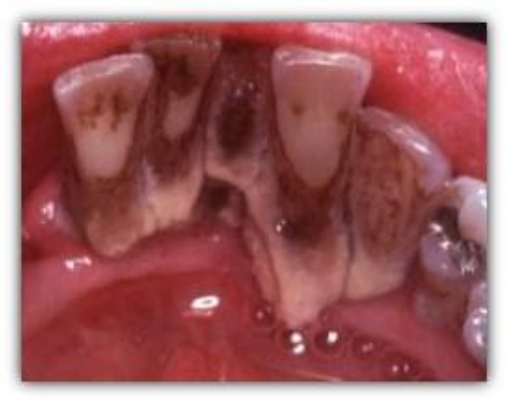
- Octacalcium phosphate was detected in the areas of supragingival calculus near the superficial layer always in contact with saliva
- In the middle layer of the calculus Hydroxyapatite was the main component .
- Supragingival calculus adjacent to the gingival and subgingival calculus contain Whitlockite.
- Brushite in calculus is very rare and is found only in the supragingival calculus adjacent to the gingiva.

Distribution

- **Vestibular surfaces**. Tooth 41 had significantly more supragingival calculus on its vestibular surface than had other teeth, with the exception of tooth 26. The mandibular central and lateral incisors had significantly more supragingival calculus on their vestibular surfaces than had maxillary central and lateral incisors. In contrast, maxillary first molars had a significantly higher mean grade for supragingival calculus on their vestibular surfaces than did mandibular first molars.



– **Lingual surfaces**. The lingual surfaces of the mandibular anterior teeth (33-43) had significantly higher grades of supragingival calculus than the lingual surfaces of other teeth. Of these lower anterior teeth, the lingual surfaces of the central incisors had higher mean calculus grades than those of the lateral incisors and both the central and lateral incisors had significantly greater amounts of supragingival calculus than did the canines.



Subgingival Calculus

- **Location**-below the crest of marginal gingival (not visible on routine examination)
- **Color**-dark brown or greenish black
- **Texture and consistency**-hard and dense crusty, spiny or nodular deposites
- Firmly attached to tooth surface.
- Crevicular fluid and inflammatory exudates are the main source of mineral salts.
- Found on any root surfaces with a periodontal pocket.

Inorganic/ Organic

Composition is similar to supragingival calculus except that it contains higher concentration of calcium,magnesium and fluoride then supragingival calculus,reflecting higher concentration of ions in GCF than in saliva.

Organically its devoid of salivary proteins, more from GCF and inflammatory exudate .

Crystals

- Octacalcium phosphate [$Ca_8(PO_4)_4(HPO_4)_25HO$] (OCP),
- Hydroxyapatite [$Ca_{10}(PO_4)_6(OH)_2$] (HAP), and
- ß-tricalcium phosphate or Whitlockite [$Ca_{10}(HPO_4)(PO_4)_6$] (WHT)

The WHT-to-HAP (Jensen and Danø, 1954) and calcium-to phosphorus ratios (Little and Hazen, 1964) in supragingival calculus are lower than those in subgingival calculus

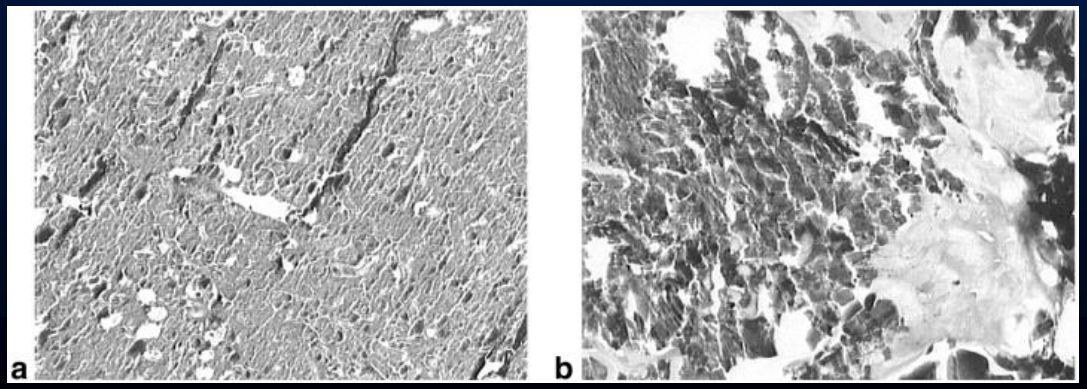
Light Microscopy

- Unlike supragingival **calculus**, lacunae of stained organic material were not seen within the body of subgingival **calculus**. The **calculus** surface previously in contact with the tooth was usually flat and mineralized whilst the external/oral surface was fairly regular and covered by a non-mineralized plaque layer of variable thickness

Transmission Electron Microscopy

- The calcification within the body of subgingival **calculus** was more homogeneous than supragingival **calculus** and consisted of small randomly orientated needle- and platelet-shaped crystals.

  There were also areas with flat "bulk-shaped" crystals, as described by Sundberg & Friskopp (1985), within which were fewer bacterial cell structures.



Scanning Electron Microscopy

- Subgingival calculus usually appeared in band like clusters of deposits with varying sizes and rough surfaces.

- under low magnification it has complex appearance. There are clusters of small deposits in the incisal/ occlusal part and larger deposits that tend to fuse in apical part.

- High magnification revealed that subgingival fracture surface were rougher than supra gingival grooves and holes that gave sub gingival fracture a 'worm eaten' look. Filaments did not dominate the surface of subgingival calculus and no pattern of orientations were seen

X-ray Beam Diffraction Analysis

- In subgingival calculus Octacalcium phosphate is scarcely found.
- The main constituent is Whitlockite and Hydroxyapatite .
- Brushite is totally absent

Distribution

- No significant differences were found for subgingival calculus between right and left sides of the mouth for a given site and a given tooth. For all teeth except 26, 27, and 41, the amount of subgingival calculus was higher on the lingual surface than on the vestibular surface

- Significant differences in the amounts of subgingival calculus among different teeth were detected. The results of the Duncan tests showed that for the vestibular surfaces of the 28 teeth, the mandibular anterior teeth and maxillary molar teeth had the greatest amount of subgingival calculus.

PREVALENCE

In mandible

- 100% mandibular anterior teeth had calculus
- decreasing posteriorly to 20% in mandibular third molar.

In maxilla

- 10% of anterior teeth
- 60% of first molars. (Sand 1949)
- Navajo Indians had more calculus then Caucasians . (Parfitt 1959)

- Subgingival calculus is found in interproximal surface and least on buccal surfaces.
- The first teeth to show calculus deposit were maxillary first molar and mandibular incisor.
- Maxillary incisors and bicuspids were least involved.
- Supragingival calculus starts forming with 6 years of eruption age while Subgingival at 8 yrs of age, Subgingival is least before 20 yrs of age.

- The formation of supragingival calculus was observed early in life in the Sri Lankan individuals, probably shortly after the teeth erupt.
- Deposition of supragingival calculus reaches maximal scores around 25 to 30 yrs of age. Calculus accumulation appeared to be symmetric, and by age 45 only a few teeth, typically the premolars were without calculus.
- By age 30, all surfaces of all teeth had subgingival calculus without any pattern of predilection.

- The Norwegian academicians had good oral hygiene,resulting in reduced accumulation of calculus. however, supragingival calculus still formed on facial surfaces of upper molars and lingual surfaces of mandibular incisors in 80% of individual,it did not increase with age and did not involved other teeth. (Anerud et al 1991)

Factors affecting the amount of calculus in a population

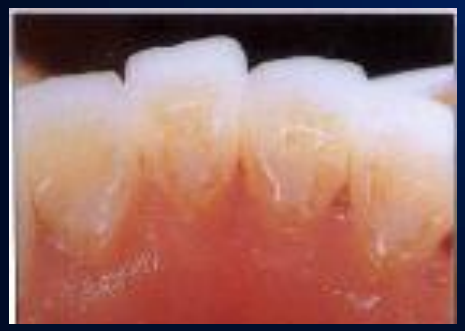
- Oral hygiene habits
- Access to professional care
- Diet (Macnill, 1956)
- Age
- Ethnic origin
- Habits eg tobacco, betel nut (Pindborg, 1947)
- Systemic disease
- Use of prescription medications
- Stress (Parodneck, 1937)

RATE OF FORMATION

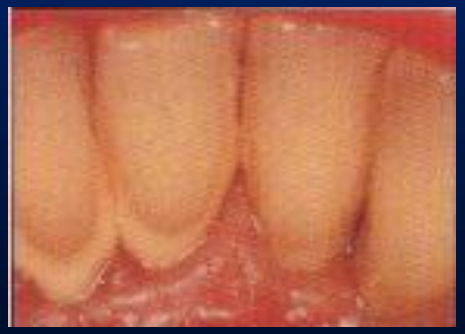
- Soft plaque is hardened by mineralization between 1st and 14th days of plaque formation.
- Calcification is reported to occur in as little as 4-8 hrs. Calcifying plaque may become 50% mineralized in 2 days and 60% to 90% mineralization in 12 days. All plaque does not necessarily undergo calcification.
- Plaque that does not develop into calculus reaches a plateau of maximal mineral content within 2 days.
- Calculus is formed in layers which are often separated by a thin cuticle that becomes embedded in calculus as calcification progresses
- Plaque has the ability to concentrate calcium at 2 to 20 times its level in saliva.

- The initiation of calcification and the rate of calculus accumulation vary from person to person for different teeth and at different times of same person

- According to this they are classified as **heavy, moderate or slight** calculus former. (Muhlar and Ennever 1962)



Slight



Moderate

Heavy

- Early plaque of heavy calculus former contains more calcium, three times more phosphorous and less potassium than that of non-calculus former

- Total protein and total lipid levels are elevated in Heavy calculus formers.

- Light calculus formers have higher levels of parotid pyrophosphate.

- Calcification of plaque is delayed in children. (Turesky, 1961)

- The average daily increment in calculus former is from 0.10% to 0.15% of dry weight. (Lobene et al, 1966)

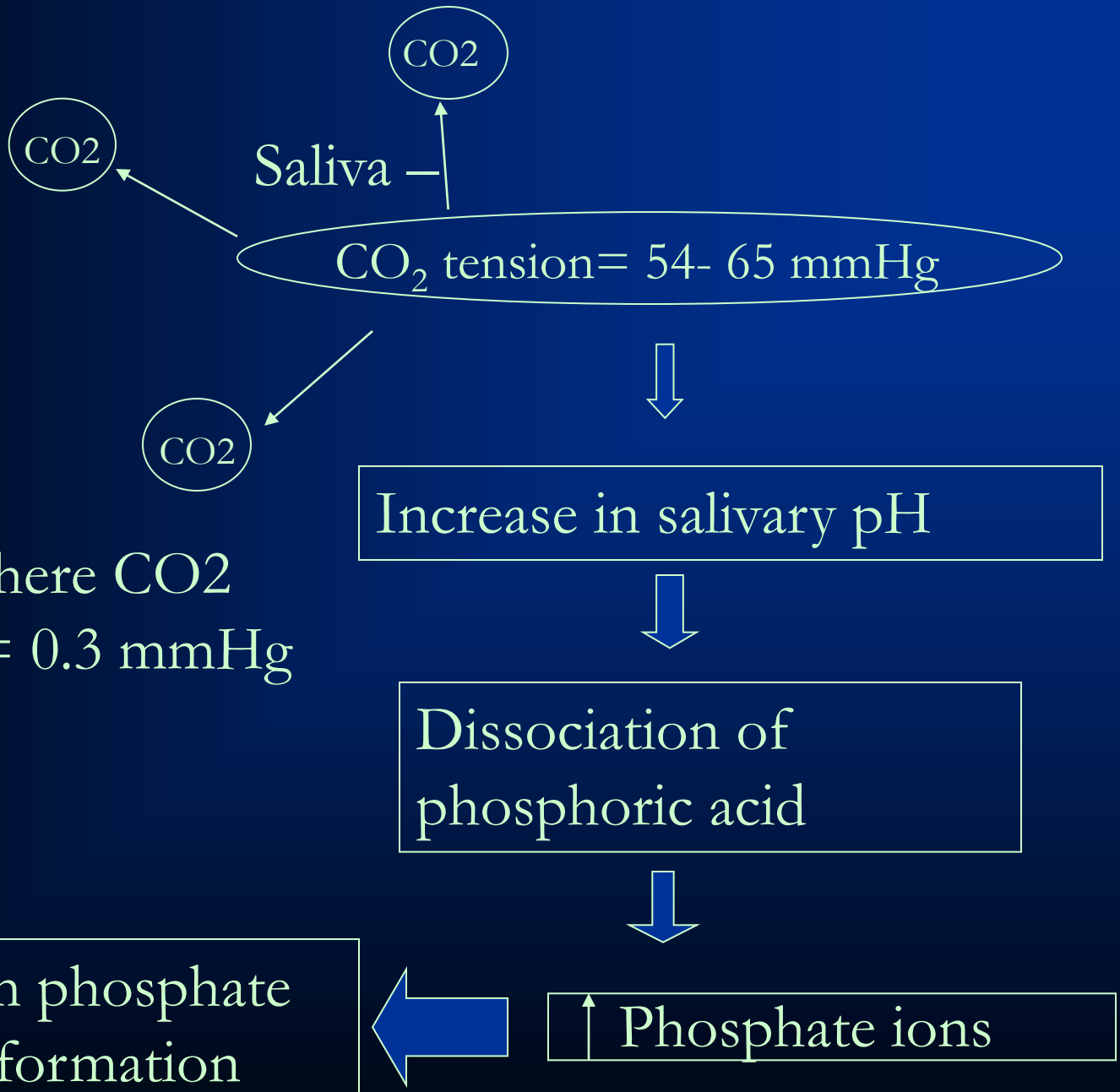
- Calculus formation continues until it reaches a maximum, after which it may be reduced in amount. The time required to reach the maximal level has been reported as 10 weeks and 6 months. The decline for maximal calculus accumulation, referred to as reversal phenomenon may be explained by the vulnerability of bulky calculus to mechanical wear from food and from the cheeks, lips and tongue.

- In the 10-15 yrs age group, 43% of normal children and 90-100% of children with cyctic fibrosis and asthma have calculus. This clinical observation may reflect elevation in salivary calcium and phosphorus in children with these diseases. (Wotman et al, 1973)

THEORIES OF MINERALISATION

- **Booster Mechanism**

According to this theory, calcification will occur in a particular locus when the local pH and calcium and phosphorous concentrations are high enough to allow for precipitation of a calcium phosphate salt. Factors such as loss of CO_2 and production of ammonia could account for an elevation in pH; acid or alkaline phosphatase activity could result in a higher phosphate concentration; liberation of bound or complexed calcium from the salivary proteins would produce higher calcium levels.


CO2
CO2
Saliva –
CO_2 tension= 54- 65 mmHg
CO2
Increase in salivary pH
Atmosphere CO2 tension= 0.3 mmHg
Dissociation of phosphoric acid
Calcium phosphate crystal formation
Phosphate ions

- **Epitactic Concept**

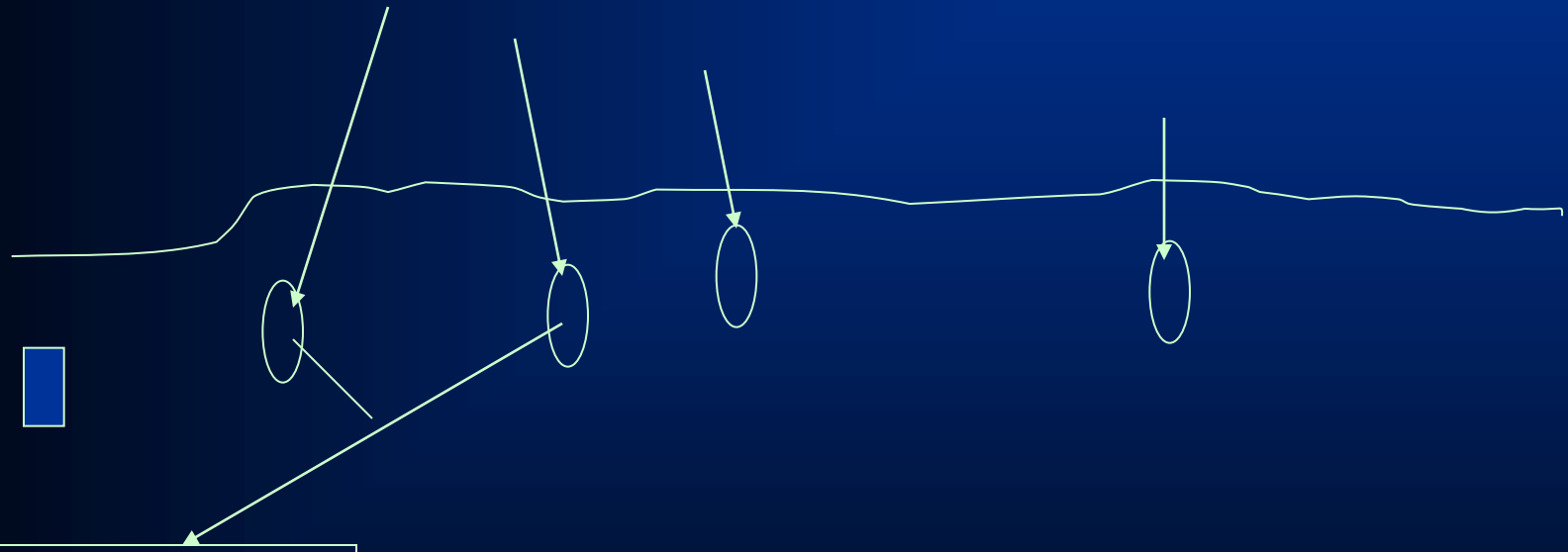
One of the most widely held theories has been the epitactic concept, which recognizes that the concentration of calcium and phosphate ions is not high enough in tissue fluids and saliva to precipitate spontaneously, but is sufficient to support growth of a Hydroxyaptite crystal once an initial seed or nucleus is formed. Tissue fluids and saliva are thus called metastable solutions. The formation of the initial crystal or nucleus is called nucleation and is thought to occur when a proper organic matrix is available on which the nucleus can crystallize in exact structural configuration.

(Boskey, 1981)

Epitactic Theory

" Metastable solutions":

saliva and tissue fluids



Initial organic seedings

- **Inhibition Theory**

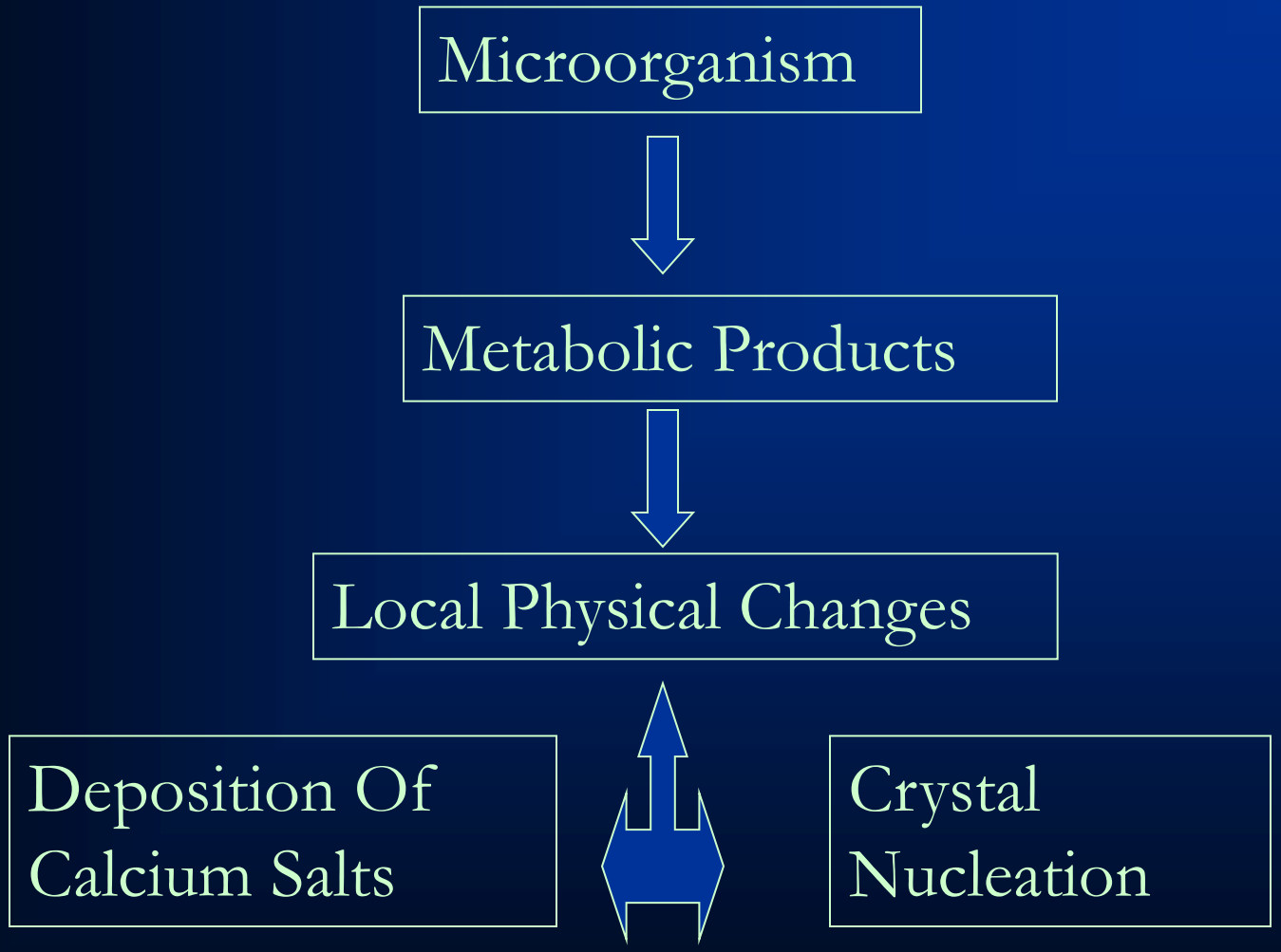
Another approach considers calcification as occurring only at specific sites because of the existence of an inhibiting mechanism at non-calcifying sites. One possible inhibiting substance is thought to be pyrophosphate (and possibly other polyphosphates) and among the controlling mechanisms is the enzyme alkaline phosphatase, which can hydrolyze the pyrophosphate to phosphate (Russsel and Fleisch. 1970). The pyrophosphate inhibits calcification by preventing the initial nucleus from growing, possibly by poisoning the growth centers of the crystal .

- **Transformation**

A most attractive hypothesis is the idea that hydroxypatite need not arise exclusively via epitaxy and nucleation. Amorphous non-crystalline deposits and Brushite can be transformed to Octocalcium phosphate and then to Hydroxypatite (Eanes et al, 1970). It has been suggested that the controlling mechanism in the transformation process may be pyrophosphate (Fleich et al 1968)

- **Bacteriological Theory**

  Oral microorganisms are the primary cause of calculus, and that they are invovled in its attachment to the tooth surface. Leptotrichia and Actinomyces have been considered most often as the causative microorganism.(Galippe.V., 1886)


Microorganism
Metabolic Products
Local Physical Changes
Deposition Of Calcium Salts
Crystal Nucleation

- **Enzymatic Theory**

  Calculus formation is the resultant of the action of phosphatases derived from either oral tissues or oral microorganism on some salivary phosphate containing complex, most probably phosphoric esters of the hexophosphoric group. (Adamson.K.T, 1929)

Mechanism Of Plaque And Microbial Mineralization

- Following tooth eruption or a dental prophylaxis, salivary proteins rapidly and selectively adsorb onto the enamel surface to form an acquired enamel pellicle. It is followed by the adherence of various oral micro-organisms. Gram positive coccoidal organisms are the first settlers to adhere to the formed enamel pellicle, and subsequently, filamentous bacteria gradually dominate the maturing plaque biofilm (Scheie, 1994).

- Plaque absorbs calcium and phosphate from saliva for the formation of supragingival calculus and from crevicular fluid for the formation of subgingival calculus

(A) Adsorption Of Salivary Proteins

Three types of time-dependent adsorption of salivary proteins have been observed (Lamkin *et al*., 1996)

- Salivary proteins, such as proline-rich protein–3 (PRP-3), PRP-4, and statherin, adsorb very fast onto HAP
- whereas the binding rate of -amylase, glycosylated proline-rich protein (PRG), and cystatins is slow.
- The third type of protein adsorption can be seen in the case of PRP-1, PRP-2, and histatins

(B) Microbial Adherence

- As Van Loosdrecht *et al.*(1990) and Bollen *et al.*(1996) have described, the microbial adhesion to solid surfaces (such as tooth surfaces and various implant surfaces) may proceed as a four-stage sequence
- initial approach of bacteria to a surface where random contact, such as Brownian motions and liquid flow, or active movement of micro-organisms may occur
- The attractive van der Waals' forces and repulsive electrostatic forces are responsible for the second stage of microbial adhesion, which is a reversible process

- The firm attachment of micro- organisms, "the third stage', is irreversible

- The fourth stage of adhesion, *i.e.*, bacterial colonization.

(1) Surface Proteins And Bacterial Adherence

- Appendages, such as fimbriae or pili
- Flagellum, another microbial surface protein
- Collectively, flagella, fimbriae (pili)
- *Streptococcus mutans* surface proteins have been most widely investigated.
- P1, a 117-kd and a 127-kd protein, and two glucosyltransferases (GTFase)

(2) Microbial Co-aggregation And Co-adhesion

Oral bacteria tend to associate with one another. In suspension, the association between different oral bacteria is termed "co-aggregation", while it is referred to as co-adhesion if one partner of the pair is attached to a surface.

- Lectin-sugar interaction
- Cation-dependent
- Non-cation-dependent (interfacial free energies)
- Extracellular vesicles
- Cell-surface lipoproteins

3) Microbial Adherence To Implant Surfaces

- Since Ti pellicle differs from enamel pellicle with regard to protein composition, it is understandable that the microbial adherence and the subsequent microbial colonization on a Ti surface may be different from that on an enamel surface (Edgerton et al., 1996)
- There is evidence that Ti implants harbour less mature plaque, which is composed of more coccoid cells and fewer motile rods, as compared with natural teeth (Quirynen and Listgarten, 1990)

- Higher levels of microbial adherence have been found on rough Ti surfaces than on smooth and polished surfaces in *in vivo* studies (Nakazato et al., 1989)

(C) Driving Force For Plaque Mineralization

- Calcium and phosphate are two salivary ions which are "raw materials" for dental calculus formation.

- In a recent study (Poff et al., 1997), however, it was reported that no significant correlation existed between calcium phosphate supersaturation in saliva and the rate of calculus formation for both unstimulated and stimulated saliva.

- However, calculus formation is influenced by a variety of factors, such as salivary flow rate, and inhibitors and promoters of calculus formation, other than salivary super saturation with calcium phosphate salts.

(D) Involvement Of Bacteria In Calculus Formation

- Although calculus can be induced in germ-free animals (Theilade et al., 1964),

- Microbial mineralization occurs even within "acidogenic" and cariogenic bacteria (Moorer et al., 1993).

- The filamentous micro-organisms (Diphtheroids and Veillonella sp)are predominant in supragingival calculus and calculus-associated plaque, whereas micro-organisms of various morphologies are found in the plaque adjacent to subgingival calculus (Friskopp and Hammarström, 1980). In addition to dead micro-organisms, live and degenerated ones can also calcify in synthetic calcifying media (Sidaway, 1980).

- Subgingival calculus deposits appeared to have a significantly greater percentage of coccoid forms and fewer motile rods (CM Brown et al., 1991).

- Plaque bacteria actively participate by forming phosphatases,changing the plaque pH or inducing minerialization

- Non-calculus sites are associated with a significantly higher level of *Actinobacillus actinomycetemcomitans* (*Aa*) and a lower level of black-pigmented anaerobic rods than sites presenting with calculus. *Aa* has, therefore, been proposed to exert an inhibitory effect on the colonization of plaque-producing and calcifiable bacteria (Listgarten, 1987).

(E) Microbial Mineralization

- Initial deposition of apatite in calcifying bacteria is associated with membrane or acidic membrane-associated components (Boyan and Boskey, 1984).

- The functions of acidic phospholipids in calcification depend upon their ability to bind calcium by their negatively charged groups.

- Calcium is bound to adjacent phospholipid molecules in the membrane through a two-point electrostatic attachment to form a stern layer which facilitates the interaction of calcium with inorganic phosphate ions in solution (Hauster *et al.*, 1969). The bound calcium ions can cause loss in water by dehydration due to the neutralization of electrostatic charge of membrane (Hauster *et al.*, 1975).

- Inorganic phosphate associates with the bound calcium to form a Ca-phospholipid-phosphate complex (CPLX). Once CPLX has formed, apatite deposition follows when sufficient calcium and phosphate ions are present and the concentration of inhibitors is low. The availability of CPLX exhibits bacterium-specificity; CPLX is always present in *Corynebacterium matruchotii*.

(F) Nucleation Inhibitors

- It has been demonstrated that magnesium (Mg) prevents apatite nucleation by *C. matruchotii*.

- Diphosphonates, such as ethane-1-hydroxy-1, 1-Diphosphonate (EHDP), inhibit both apatite nucleation (Fleisch et al., 1970) and crystal growth (Francis, 1969).

- Importantly, nucleation inhibitors should not be used clinically, because they have been found to interfere with normal mineralization of hard tissues (Schenk et al., 1973).

(G) Crystal Growth Inhibitors

- Negatively charged salivary proteins, statherin and PRP are two representatives of salivary inhibitors of crystal growth.
- Cystatins in saliva, such as the acidic cystatin and the neutral cystatin.
- Immunoglobulins present in dental plaque and calculus may also have an inhibitory effect on plaque mineralization.
- It was found that albumin could bind to Hydroxyapatite surfaces and inhibit crystal growth.
- In addition to these salivary proteins, pyrophosphate and zinc ions act as crystal growth inhibitors.

(H) Organic Acid And Calculus Formation

- Despite that fact, our knowledge of dental caries can lead one to assume the potential role of organic acids in calculus formation, since both the enamel and supragingival calculus contain apatites and have similar oral environments

- The importance of lactic acid in demineralization of enamel is well-known

- As the un-ionized organic acids diffuse, they continually dissociate partially into hydrogen ions and acid anions. The released hydrogen ions attack the enamel crystals. Collectively, the process of demineralization is composed of the following three steps: diffusion of the un- ionized acids into enamel, partial dissolution of acids, and diffusion of calcium and phosphate out of enamel (Featherstone and Rodgers, 1981).

(I) Enzymes Degrading Calcification Inhibitors

- According to a study by Watanabe et al.(1982), calculus level was positively correlated with protease activity in human saliva. protease in saliva and plaque can degrade calcification inhibitors such as statherin and PRP. Moreover, proteases may increase dental plaque pH through the production of ammonia, one of the proteolytic end- products of proteins (Frostell and Söder, 1970).

- Acid and alkaline phosphatases may promote crystal growth by degrading pyrophosphate, which is an inhibitor of crystal growth.

(J) Calcification Promoters

- **Urea**

  Urea is a product from the metabolism of nitrogen-containing substances. Urea can be secreted in normal saliva at concentrations of between 5 and 10 mmol/L (Macpherson and Dawes, 1991),

  The effect of urea metabolism on plaque pH

  Ammonia produced from ureolysis of urea contributes to an increased plaque pH that is an essential factor in natural calculus formation.

  The ureolytic pH response (an increase in plaque pH by the production of ammonia from urea) promotes calculus formation by increasing the saturation degree of calcium phosphate in plaque fluid.

- **The effect of salivary flow rate on urea-dependent pH response in plaque**

  In the oral cavity, urea available to supragingival plaque bacteria comes from both saliva and extra-oral factors such as urea-containing chewing gum and toothpastes. The effects of salivary flow rate on the pH responses induced by urea from these two sources are different. In the absence of exogenous urea, increased salivary flow rate promotes the urea-dependent pH response in plaque. Thus, the sites with higher salivary film velocity have an increased urea-dependent pH response in plaque (Shannon and Prigmore, 1960). That can partially explain the site-specificity of calculus distribution in the dentition.

- **Fluoride**

  - The caries-inhibiting ability of fluoride is well-known. Fluoride not only counteracts demineralization of hard tissue through the formation of lower-solubility fluorapatite by fluoride substitution for hydroxyl ions and adsorption onto apatite surfaces (Wong et al., 1987), but also contributes to re-mineralization by precipitation of a fluoride-enriched apatite or calcium fluoride.

  - fluoride may promote the maturation of spontaneous precipitated calcium phosphate at physiological pH by reducing the stability of OCP (Eanes and Meyer, 1978) Fluoride has also been demonstrated to affect the morphology of the apatite crystals as it converts them from thin plates to short and slender needles (Eanes and Meyer, 1978).

- **Silicon**

  - Silicic acid has been reported as a strong promoter of both spontaneous precipitation of calcium phosphates and the growth of seeded crystals (Damen and ten Cate, 1989). Silicic acid was also found to stimulate the transformation from amorphous calcium phosphate to HAP (Hidaka *et al.*, 1993).

  - Silica at a concentration of 0-2 mg/ml was reported to exert stimulating effects on calcium phosphate precipitation (Damen and Ten Cate, 1989). Silica was also reported to increase the rate of calculus formation 35 days after its incorporation into food.

– There is also epidemiological evidence for the effect of silicon on calculus formation. It has been reported that the rate of calculus formation is higher in the Indonesian than in the Norwegian population (Gaare et al., 1989). The difference in the silicon content of the food may partially explain the phenomenon. Indonesian people consume larger amounts of rice, which is enriched in silicon. The rice-based diet of Indonesian people contributes to the greater calculus formation in this population

– On the other hand, it is noteworthy that 3.0-30.0 mmol/L silicic acid and 10 mg/mL silica inhibit the rates of both amorphous calcium phosphate formation and HAP transformation. The inhibitory effects on calcium phosphate precipitation of silicic acid and silica at relatively high concentration may be due to their increased chelating effects

Mineralization

- Two modes of mineralization ,Type A and Type B centers ,have been distinguished ultra structally . Type A mineralization centers are formed only in presence and association of microorganisms ,Type B mineralization centers are apparently not related to microorganisms but have at least one common border with the Type A centers and included micro organisms .
- The crystals associated with Type A have been identified as hydroxyapatite ,where as
- Crystal patters associated with Type B are OCP, Whitlockite and Brushite.

Calculus Attachment

First study on modes of Calculus attachment

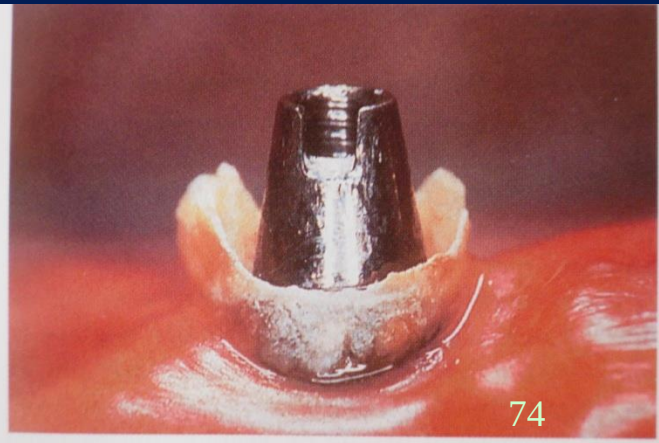
Zander in 1953

- A secondary cuticle interface between calculus and tooth surface
- Attachment of calculus matrix to irregularities of cementum surface corresponding to previous insertion locations of Sharpey's fibers
- Penetration of microbial organisms of calculus in cementum
- Attachment into areas of cementum resorption via mechanical locking into undercuts.

- Inorganic inter-crystalline forces may represent a significant factor for attachment. (Selvig, 1970)
- Attachment to pure titanium is less intimate than to root surfaces structure.

Smooth machined implants have less microporosities for retention.

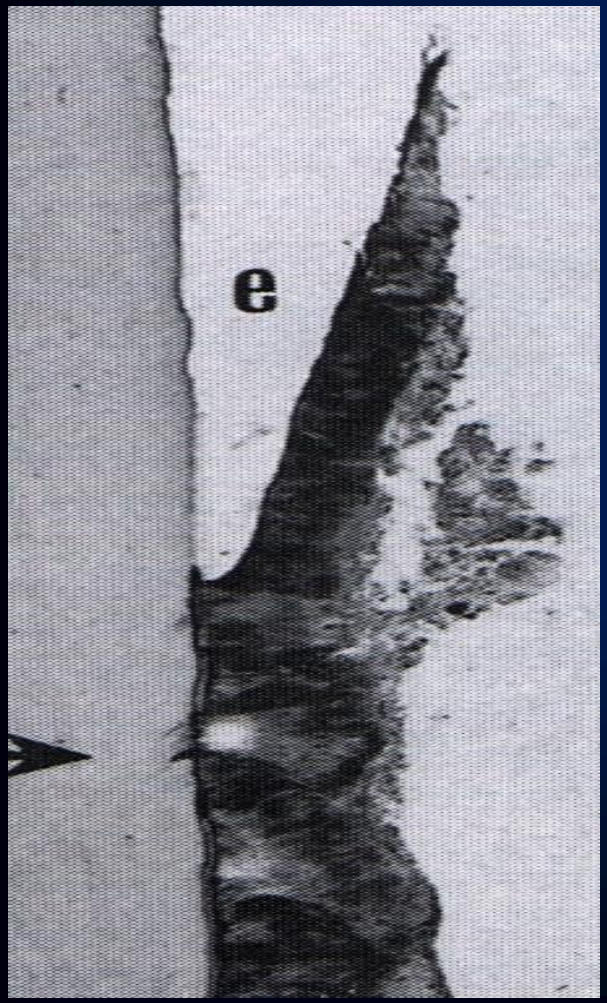
(this would mean that calculus may be chipped off from implants without affecting it) Matarraso etal 1996

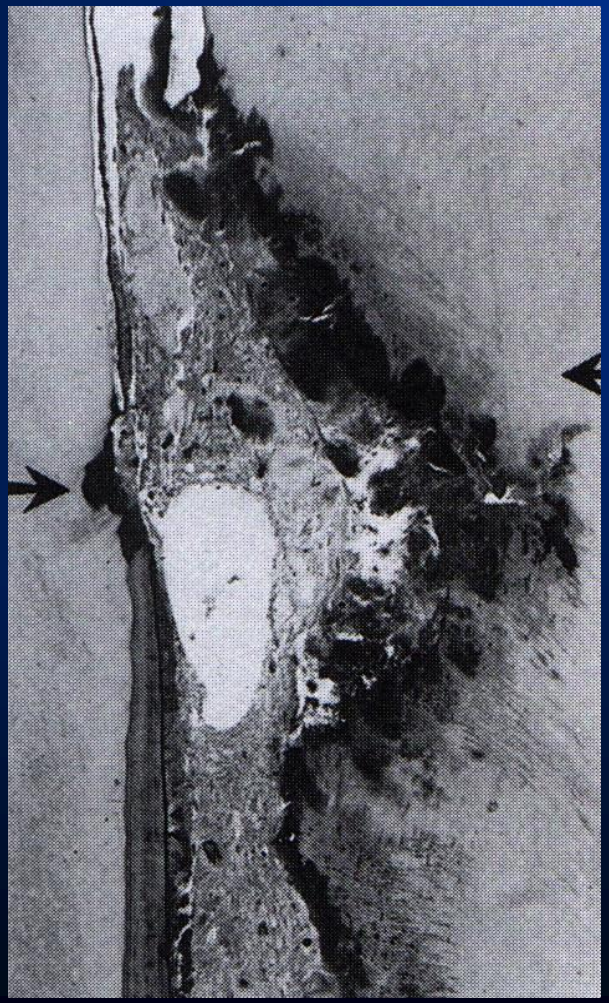


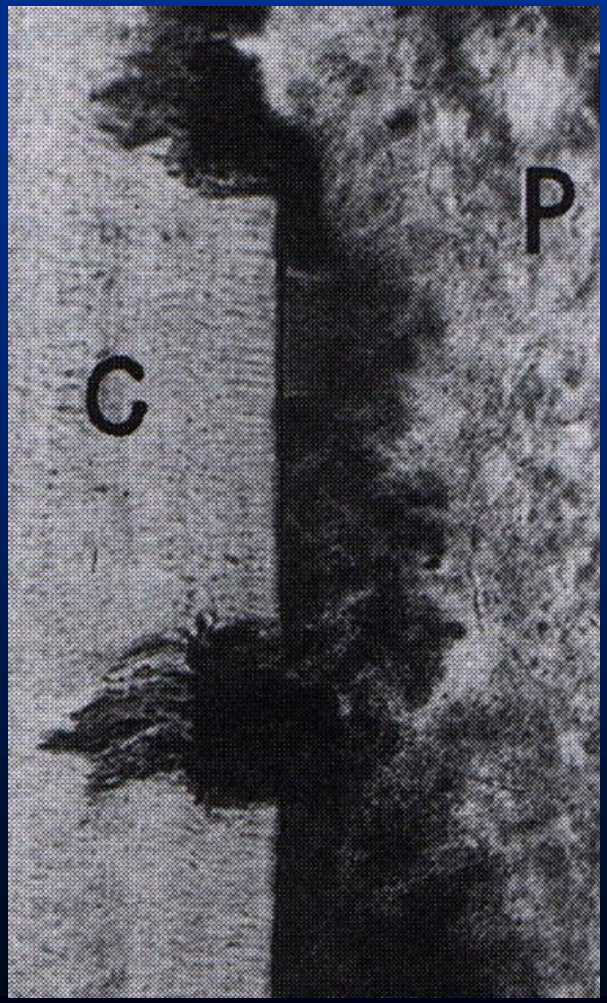
Pellicle

Dentin

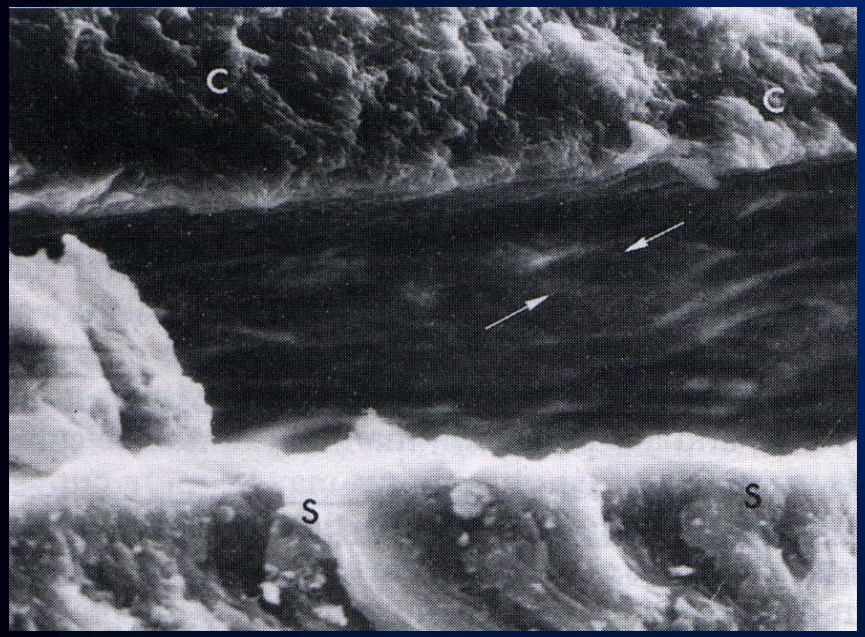
Cementum

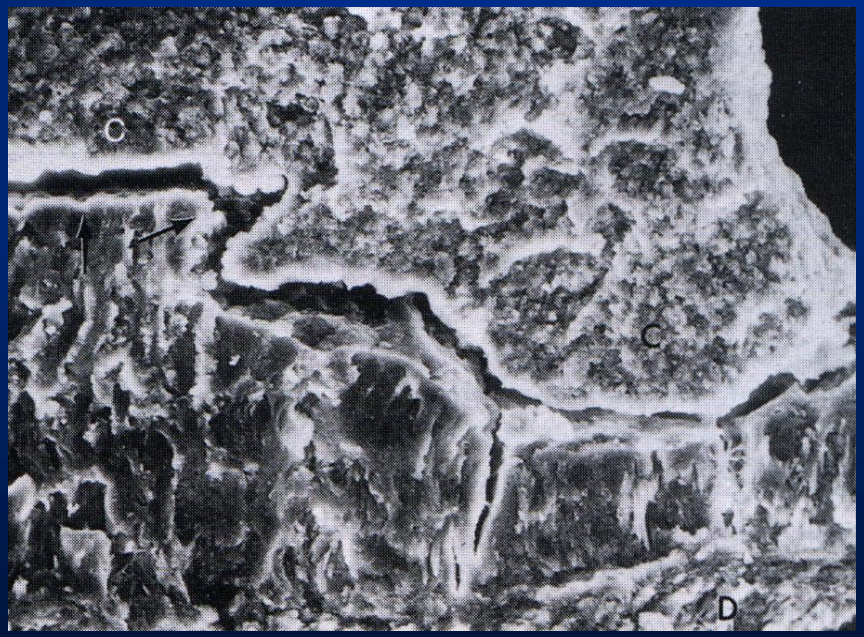






Close Adaptation Of Calculus To The Cementum





Clinical Significance

Until 1960 the prevailing view was that calculus was the major etiological factor in periodontal disease

- **Supragingival calculus**

  The process is enhanced by supragingival calculus which provides further retention and thus promotes new plaque accumulations it is still not clear whether calculus plus plaque provokes a greater reaction than plaque alone, though there is some suggestive evidence of the former. (Schwartz et al 1971)

  - bring the bacterial overlay closer to the supporting tissue
  - interfere with local self cleansing mechanism
  - make plaque removal more difficult for the patient.

- **Subgingival calculus**

  – Clinical studies attest the importance of frequent and thorough removal of root deposits by scaling and root planning to prevent attachment loss (Pihlstrom et al, 1983).

  – Morphologic studies show that calcified deposits are porous and act as a reservoir for irritating substances.

  – Experimental studies have established the permeability of subgingival calculus to endotoxin (Baumhammers and Rohrbaugh, 1970) and the presence in the deposits of high level of toxic stimulators of bone resorption and antigens from *Bacteroides gingivalis)* (Patters et al, 1982) when coupled with the increasing buildup of plaque on the surface of calculus the combination has the potential for increasing the rate of displacement of the adjacent junctional epithelium and extending the radius of destruction of bone beyond that of plaque alone (Mandel and Gaffar, 1986)

- Definite correlation between subgingival calculus and bone loss in adolescent was established (Matsson, 1990)

- Allen and Kerr in 1965 have shown, calculus after autoclaving to remove surface plaque, calculus still exerts a toxic effect on tissue cells.

- King in 1957 showed that retained calculus forms a nidus for calculus reformation proceeding more rapidly than that which forms on a smooth surface.

ANTI-CALCULUS AGENT

- Tooth brushing is relatively effective in dental plaque removal, but it is still inadequate for the maintenance of gingival health. Chemotherapeutic agents have been used to supplement the mechanical removal of dental plaque (Volpe et al, 1981).

- Early attempts with chemotherapeutic agents focused on the removal of dental calculus from teeth

Classification Of Anti- Calculus Agents

1st GENERATION

1)DISSOLUTION

- Ethylene diamine tetra acetate
  - Chelating Agents
- Sodium Hexa Metaphosphate
  - Acids –Aromatic sulphuric acid
- Nitro-muriatic Acid
- 20% Trichloroacetic Acid
- Spring Salts
- Sodium Ricinolate
- Alkalies

2)ALTERING CALCULUS ATTACHMENTS

- Silicones
- Ion exchange resins.

3.PLAQUE INHIBITION

- Antibiotics Example : Nidamycin
- Antiseptics Example :Chloramines

4.MATRIX DISRUPTION

- Enzymes Example : Mucinase
- Dehydrated Pancreas
- Trypsin, chymotrypsin
- Carboxypeptidase, lipase, amylase
- 30% UREA(solvent action on protein)

- **2nd GENERATION**
  - Inhibition of crystal growth
    - Vitamin C (By crystal poisoning mechanism)
    - Pyrophosphatase
    - Pyrophosphatase + Sodium fluoride
    - Zinc salts
    - Biphosphonates
    - Polymers & Co Polymers

Triclosan

- Triclosan is a broad-spectrum antibacterial agent active on both Gram-positive and -negative micro-organisms. The target is the cytoplasmic membrane.

- For triclosan to be effective, a delivery system is necessary to increase its residence time in the oral cavity.

PVM/ MA Copolymer

- PVM/MA copolymer promotes uptake of triclosan by enamel and buccal epithelial cells (Nabi et al., 1989).
- The copolymer is composed of two groups: an attachment group and a solubilizing group.
- The solubilizing group retains triclosan in surfactant micelles so that the attachment group can have enough time to react with tooth surfaces via calcium in the liquid adherent layer.

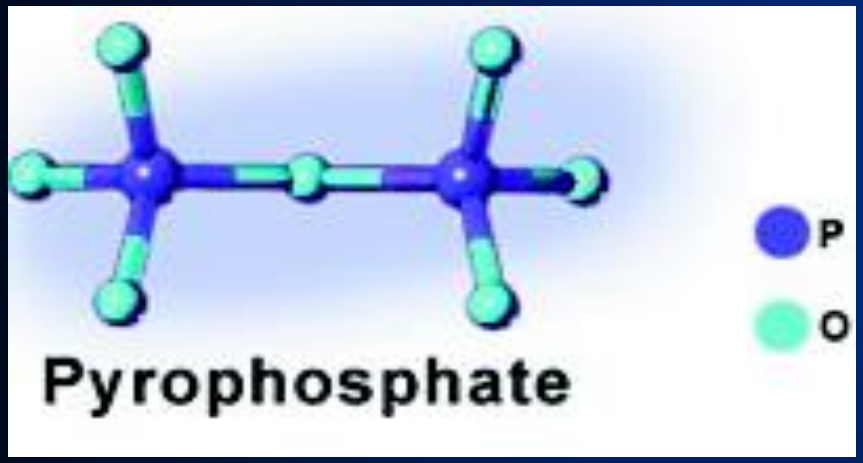
Crystal Growth Inhibitor

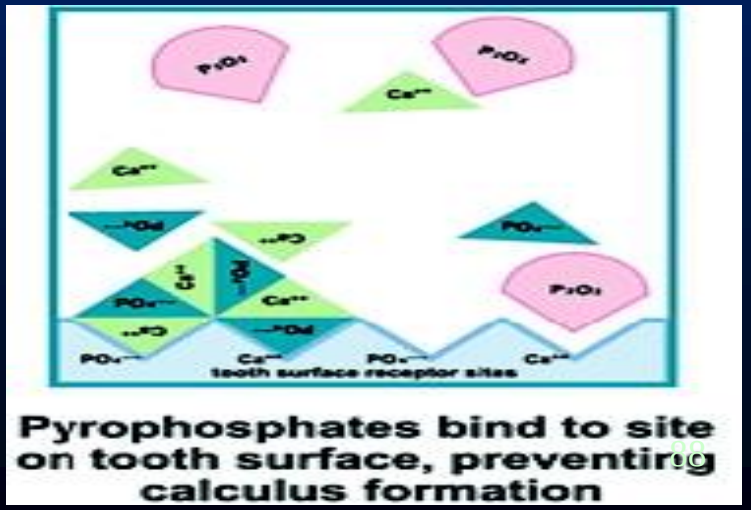
- Have Been Proven Effective Only For The Control Of Supragingival Calculus And Not For Subgingival Calculus.
- It Prevents Deposit Formation .
- It Decrease The Dissolution Rate Of Chemisorbed Surfaces (Fleisch, 1981).
- Prevents Re-mineralization And Prevents Demineralization

Pyrophosphate And PVM/MA Copolymer

- Pyrophosphate is a small molecule and has been reported to inhibit crystal growth by binding to the surface of crystal. Pyrophosphate binds to two sites on the HAP surface, and one of the two sites needs to be bound by phosphate ion to permit crystal growth to occur. If this site is bound by pyrophosphate, phosphate ion cannot absorb onto crystal, and thus crystal growth is inhibited.

- In addition to the inhibitory effect on crystal growth, pyrophosphate can also delay the initiation of conversion of DCPD to HAP by over three-fold (White et al., 1989) and reduce acquired pellicle formation


P
O
Pyrophosphate


tooth surface receptor sites
Pyrophosphates bind to site on tooth surface, preventing calculus formation

Zinc Ion

- Zinc salts are thought to reduce plaque acidogenicity (Oppermann et al., 1980) and plaque growth in vivo (Saxton et al., 1986) and to increase resistance of HAP to acid dissolution (Brudevold et al., 1963).
- Zinc can also inhibit crystal growth by binding to the surfaces of solid calcium phosphates (Gilbert and Ingram, 1988).
- Zinc ions also have an effect on the types and amounts of calcium phosphate crystals (Le Geros et al., 1999).

PREVENTION OF CALCULUS FORMATION IN IMPLANT PATIENTS

- Theoretically, various anti-calculus agents, which were discussed above, can also be active against calculus formation on implant surfaces in addition to tooth surfaces

CLINICAL MEASUREMENT

- Two indices – quantitating calculus deposits
  calculus component of OHI
  calculus component of PI
- Calculus surface index (Ennever, 1961)
- Marginal line calculus index (Muhlem, 1967)
- Volpe – manhold index (1965)
- Calculus surface severity index

EFFECT OF MEDICATION

- A quantitative recording of supragingival deposits among systemically medicated and non-medicated patients presenting for a routine dental examination and prophylaxis indicated the following: 1) a tendency for more plaque and less calculus to form among the older individuals (65 years and older) regardless of the use or non-use of medication;

- 2) a statistically significant reduction of calculus among individuals medicated with ß-blockers, diuretics, vitamin C anti-cholinergics, synthroid, or allopurinol despite the high quantity of plaque present.

- 3) neither the patient's sex nor the time interval between prophylaxis are important factors in the quantity of calculus or plaque formed in either medicated or non-medicated individuals

(Turesky et al, 1992)

Mycology of Dental Calculus

Study was carried out in view of increasing incidence of Oculomycosis in dentist. (Clayton and Fox, 1973)

Yeast Species

1. Candida albicans
2. C.guilliermondii
3. C.parapsilosis
4. C.zeylanoides
5. Cryptococcus albidus
6. Rhodotorula rubra
7. Torulopsis candida
8. Trichosporon cataneum

Filamentous Fungi

1. Aspergillus fumigatus
2. A.niger
3. A.ustus
4. A.versicolor
5. Penicillium spp
6. Aureobasidium pullulans
7. Cladosporium sp.
8. Epicoccum sp.
9. Phoma sp.
10. Scopulariopsis sp.
11. Mucor sp

DIAGNOSTIC AIDS

Visual examination

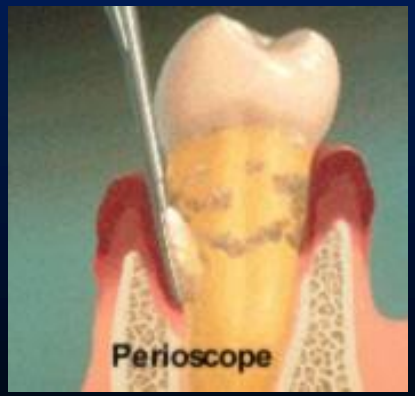
- Gentle air blast
- Transillumination
- Gingival tissue color change

Tactile examination

- Probe
- Explorer

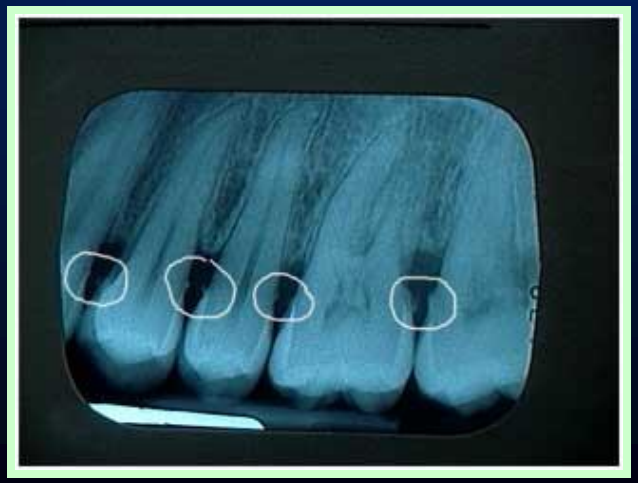
Advanced

- Modified ultrasonic scalers (Cad/Cam)
- Novel LED probe
- Fluorescence by InGaAsHg Diode laser
- Perioscope (endoscope)



RADIOGRAPHICALLY

- Under the best of the circumstances, conventional oral radiography was a poor diagnostic method for the detection of calculus. Radiographic analysis predicted calculus on less than half of the proximal surfaces where deposits were present visually, even when both investigators agreed on the radiographic diagnosis. (Buchanan et al, 1987)



FUTURE RESEARCH ON CALCULUS

- There are many questions left to be answered. The roles of macromolecules, such as PRPs, statherins, immunoglobulins, and other glycoproteins, in the initiation of biomineralizaiton remain incompletely understood. The potential effects of fluoride on calculus formation need to be clarified.

- Development of biofilm culture systems, notably the "artificial mouth", could lead to breakthroughs in our understanding of mineralization mechanisms.

- SofScale is a pre-scaling gel, containing disodium EDTA and sodium lauryl sulphate, which is claimed to soften calculus and therefore facilitate its removal.

Can calculus be used as a bone graft material… ???

- Calculus is made up of…
- Calcium phosphate is most biocompatible…
- Calculus is mineralized plaque…
- Readily available in patients…
- Non-viability of microbes…
- Rare chance of antigenicity…

- Its relative significance…
- Lack of bio-chemical & animal studies…
- Chances of infection & immune reaction…

- Conduct study's to determine the viability of microbes following AUTOCLAVING…

- Study's to determine antigenicity of microbes…

CONCLUSION

- 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 possess the clinical skill to remove the calculus and other irritants as a basis for adequate periodontal and prophylactic therapy.

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