Sheet#8

Refer to slides no. 7

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Etiology factors of periodontal disease are 2 groups:

- Primary factor and usually its plaque
- Secondary factors (systemic, environmental, smoking, medications, calculus and local predisposing factors)

Plaque and calculus they go hand in hand, so if the doctor ask you about etiology you are never wrong if you say plaque and calculus.

In this lecture we will talk about:
- Calculus.

-Other local predisposing factors.

❖ Calculus

Definition

Mineralized bacterial plaque that forms on the surfaces of natural teeth and non-shedding surfaces whether it's orthodontic appliance or prosthetic appliance removable or fixed.

Two types:

1. Supra-gingival calculus (above gingival margin)
2. Sub-gingival calculus (below gingival margin)

 # Supra-gingival calculus

Located coronal to the gingival margin

White or whitish yellow in color, but also influenced by other substances.

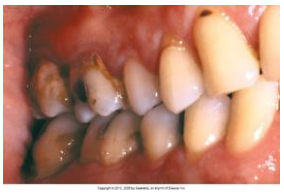
Hard with claylike consistency, easily detached from the tooth surface

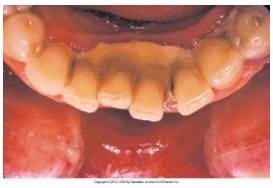
May rapidly recur after removal; you may do scaling for a patient and after 2 weeks you start see calculus accumulation again, there are some differences between patients; some patients form calculus more rapidly compared to other

patients, these patients you can consider for them to use toothpaste that has anticalculus agent (as; sodium hypophosphate) or practice more frequent plaque control as brushing 3 times per day rather than 2 and so on.

Most common places for accumulation:

Lingual side of lower anteriors and the **buccal** side of maxillary molar (due to the opening of salivary glands).





 # Sub-gingival calculus

Location and extent of subgingival calculus:

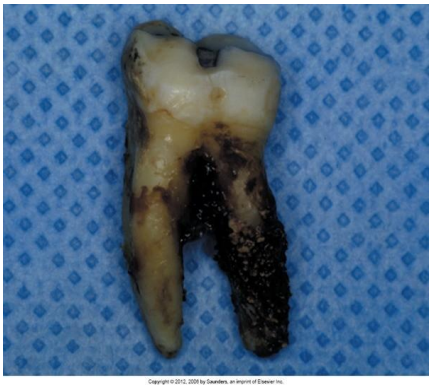
Usually it's detectable visually by retraction the gingival margin or by tactile sensation with a delicate dental instrument such as an explorer.

Hard and dense, and firmly attached to the tooth surface.

Dark brown or greenish black in color. Why?

- Because of the staining of heme from blood

Usually extend nearly to the base of periodontal pockets in chronic periodontitis but does **NOT** reach the junctional epithelium. This is important clinically, on the practical level you have never to invade the junctional epithelium to get to the most apical portion of the sub-gingival calculus, you always have a zone doesn't have calculus but may have sub-gingival plaque not calcified plaque!



This is sub-gingival calculus covering the entire of root.

Always after extraction inspect and see how much there is calculus.

Sometimes when tooth need extraction we do root planning and try to remove as much as we can from the sub-gingival calculus then after we took the tooth out we see how much calculus still there so it's very often to leave some sub-gingival calculus on it. They do this just for practice and to know how much calculus still there after root planning.

- Most common sites again are:

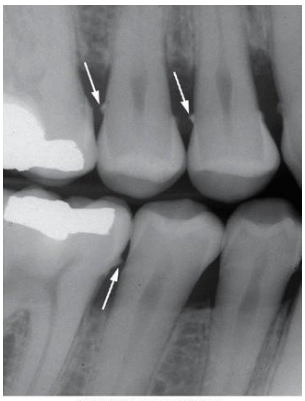
Buccal of maxillary molars and lingual of mandibular incisors.

- Appears early in life (I can see it in children) and continues to accumulate in absence of OH,mean it continues to increase in size.
- Stays localized in the presence of OH,
- What prevent calculus from accumulation?

Accumulation of plaque is countered by removal, even without brushing there is always part that doesn't have plaque because of the balance between cleansing action of chewing, mastication and speech and the accumulation of plaque (the same is applied for calculus)

Supra- and sub-gingival calculus may be also detectable radiographically

We cannot differentiate if it's supra or sub unless the shadow of the soft tissue is very clear. Sometimes we can see the bone profile.



"The right way to describe this finding by seeing these projections (arrows) on the cervical area, this is an evidence on radiographic calculus (sub or supra)"

If we don't see calculus in radiograph this mean x-rays are not sensitive but specific so clinically maybe there is calculus or not.

"Not calcified enough to show readily on the x ray but clinically it may be there."

And if you see calculus in x-ray, it's defiantly there.

Composition:

- Inorganic (supra-gingival): (most of the calculus)

70% - 90%

Ca (39%), **P** (19%), CO_2 (2%), Mg (1%) & other

elements in trace amounts (Na, Zn, Br, Cu, Au, F, ...)

- crystalline in structure:

Hydroxyapatite (58%), Mg Whitlockite (21%),

Octacalcium phosphate (12%), Brushite (9%)

- Organic (supra-G): it resemble the plaque content

Protein-polysaccharide complexes

Desquamated epithelial cells

Leukocytes

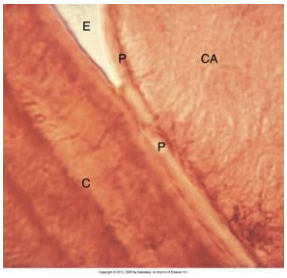
Microorganisms, are they viable/pathologic or not???

Usually they are non-viable, the core of calculus it's sterile (no bacteria)

But there is bacteria on the surface

Attachment to Tooth

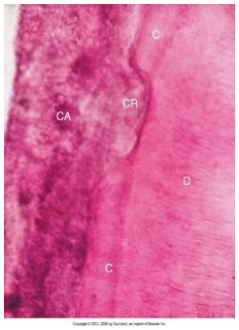
We have 4 classical explanations:



1- Organic pellicle
Remember it has role in plaque formation

"Calculus attached to pellicle on enamel and cementum surfaces"

2- Mechanical locking
which mean on the surface of the tooth there is cementum which has undercuts and irregularities giving us good mechanical locking with calculus.

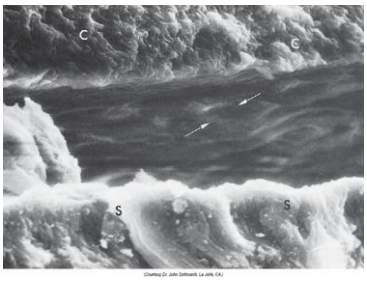


"Calculus attached to cemental resorption area"

3- Fusion
it means the crystalline structure of the cementum fuses with crystalline structure of the calculus.

4- Bacterial penetration; actually you have bacteria penetrating the cementum then they become calcified provides way for attachment of calculus to the tooth.

** Undersurface of sub-gingival calculus attached ultimately to the cementum surface



Formation

Mineralized/calcified dental plaque

- Calcification entails binding of Ca to the protein-carbohydrate complexes of the organic matrix and the precipitation of crystalline calcium phosphate salts.
- Calcification starts after 4-8 hrs of plaque formation (remember plaque start formation after 3 mins of brushing)

  So when we do scaling for patient, after 1 week we will start finding traces of calculus, we are talking about microscopic level.

  60%-90% calcified in 12 days

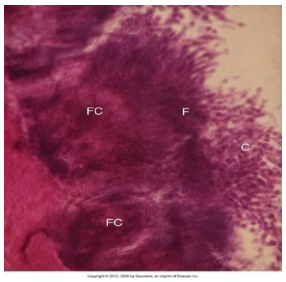
  Some studies showing that microorganisms are not necessary and organic matrix mainly come from saliva.

- The initiation and rate of calcification varies among individuals
  Trigger (2 theories): **mineral precipitation** OR **seeding agents**
  But what exactly trigger calcification we don't know.

  - **mineral precipitation:**
    We have super saturated environment of Ca and phosphate after reaching the threshold they can't stay at soluble stable state anymore so they start becoming crystals
    So usually we have crystalline structures (soluble Ca and phosphate trigger crystalline formation) within the matrix of plaque that what start the calcification.

- **Seeding agent:**

Other trace elements that can initiate the calcification of Ca and phosphate.



"Plaque and calculus on tooth surface"

Significance

- Calculus is considered secondary etiology factor (remember plaque is the primary causing factor)

  *But in any exam you should mention both plaque and calculus as etiology factors.

- Sub-gingival calculus is likely the <u>product</u> and <u>not</u> the cause of pocket formation
  So the cause of pocket formation and attachment loss is sub-gingival plaque.

  Calculus is always covered with a viable bacterial plaque; rough surface that's the role of calculus in initiation the periodontal disease.

  Calculus is sterile but we remove it because it covered with viable bacteria.

  It Keeps plaque in close contact with the tissues and creating areas where plaque removal is impossible.

  Its <u>specific</u> disease (certain types of bacteria that cause the periodontal disease) but the management scaling and planning is <u>not specific</u> we remove everything.

  Still we don't have tool that target the causative bacteria of periodontal disease.

  There are studies showing that when the pocket depth increases our ability to remove the calculus goes down.

That means when we remove calculus from 5 mm pocket and 8 mm pocket the percentage calculus that we leave behind the deeper pocket will be higher.

Even though when we do non-surgical therapy in a 7-8 mm pocket we still have clinical improvement and pocket reduction/shrinkage that's goes to what we said about the role of calculus is a 2^{o} factor not 1^{o} .

So even if you have residual calculus you still have clinical improvement.

*Remember that the core of calculus is sterile.

- Removal of subgingival PLAQUE & CALCULUS is the cornerstone of periodontaltherapy.

❖ Other Local Predisposing Factors

- ✓ Iatrogenic factors
- ✓ Anatomic factors
- ✓ Open cavities
- ✓ Malocclusion
- ✓ Orthodontic Tx
- ✓ Extraction of impacted 3rd molars
- ✓ Habits & self-inflicted injuries
- ✓ Smokeless tobacco
- ✓ Radiation

✓ *Iatrogenic Factors*

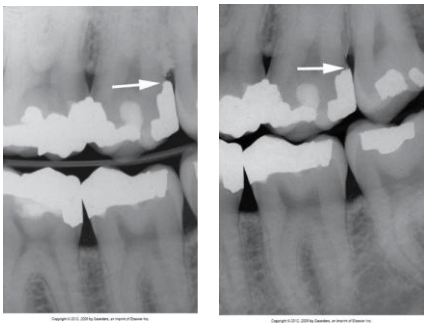
Inadequate dental procedures that contribute to the deterioration of the health of periodontal tissues (we are including the whole periodontal space not just the periodontitis)

They are all **secondary** factors of periodontal disease.

Examples:

1. Endodontic complications:
   - root perforations
   - vertical fracture(diagnosis is by founding a very deep pocket in one surface of the tooth along the line of the fracture and this is cause periodontal destruction so the treatment is extraction)
   - endodontic failures
2. Margins of restorations:
   - Overhanging margins, why??

[There is a study that has been done on dental students need MOD restorations on both sides, they put for them inlays; the contours were perfect in one side and in the other side theymade 1 mm overhang.



They asked the students not to brush their teeth for 21 days and along these 21 days they were sampling the bacteria all around the teeth and chickening the clinical condition of the gingiva.

They found more gingival inflammation around the inlays that have overhang margins ☺

So it's not only about that overhangs is an obstacle, it also changes the ecology of that side that mean the situation of the environment will favor accumulation of pathogenic bacteria.

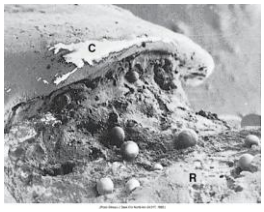
And that is one reason why overhang margins are bad.

It's very difficult to correct overhang margins so the best way to avoid it is by putting good wedges.]

So overhang margin:

- Changes the ecologic balance of gingival sulcus
- Hinders the patient's access for proper plaque removal
- marginal defects associated with reduced bone height
- removal of overhangs permits better plaque removal and reduced gingival inflammation

- Location of restorative margin:
    - **Subgingival** margins are associated with more plaque, more severe gingivitis and deeper pockets.
    - Also more Invasion of “the biologic width” which will cause persistent inflammation and periodontal bone loss (we will talk about it more next year)
- Roughness in the subgingival area:
    - facilitates plaque buildup so it’s a secondary factor
    - sources: restorative material, marginal gap, cement wash-out
    - gap gets colonized by bacteria



“Craters formed after dissolution of luting material”

3. <u>Materials</u>:
    - The best material on tissues is the most highly polished so the least irritating one is theglazed porcelain.
    - highly polished surfaces allow for adequatecleaning ifaccessibleto oral hygiene

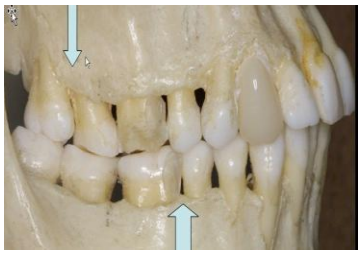
4. <u>Design of RPD:</u>
    - Greater plaque accumulation
    - Careful and personalized OHI
    - You have to have enough clearance

Lingual bar vs. lingual plate?

If you have lingual bar with a very little space between the margin of lingual bar an margin of gingival you will have plaque accumulation, swelling and inflammation of gingiva that’s why you are changing to lingual plate.

✓ *Anatomical factors*

- Root proximity:



In **lower anteriors** especially if we have crowding.

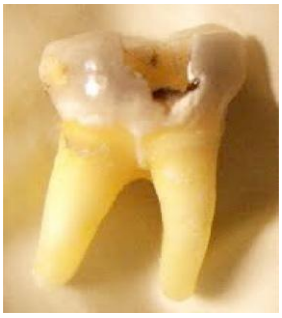
Another place is **maxillary molars** between (6 & 7) and (7 & 8)

When roots are too close to each other there is no bone between

Just cementum and periodontal ligaments.

That's make plaque control difficult and make therapy difficult.

- Cervical enamel projections &enamel pearls



Projection of enamel that extend as cementum into furcation area

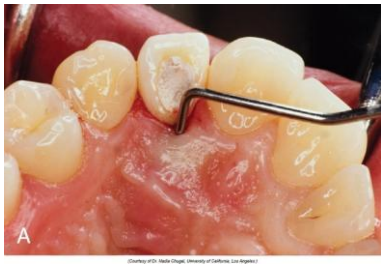
Normally we have dentino-gingival fibers between cemntum and gingiva.

Here we don't have fibers instead we have junctional epithelium attached to the enamel

Now we have two schools of thought one said it's significant to have junctional epithelium in the initiation of furcation attachment loss and the other school said it's not significant.

There is classification for cervical enamel projection called [Masters and Hoskins]

- Palato-radicular grooves

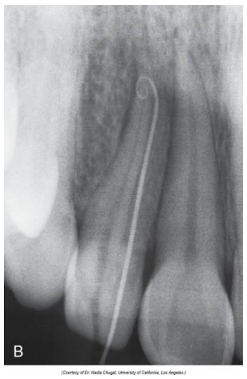


Very commonly seen on maxillary**lateral** incisors and may be seen in the centrals too.

Normal anatomy of the lateral is having cingulum and groove.

In this case the groove is extend and become very deep on the palatal root of lateral incisor.

The presence of this groove make it easier for plaque to accumulate and difficult for us to instrument and this could be the cause of attachment loss on the palatal side of laterals and so the centrals.



"x-ray showing that the pocket is go all the way to the apex."

**Student asked if this localized attachment loss stay localized or will surround the whole surfaces of the tooth ?

- It's hard to predict but usually these cases stay localized
- And remember that the periodontitis is a site specific disease not even tooth specific disease.
- It may spread laterally but this is not fast

✓ *Malocclusion*

- Crowding (it makes plaque control difficult) there is study shows that event if we have crowding with a good plaque control this is not a risk factor for periodontal disease
- Prominent roots especially canine because forces will go mainly to the most prominent root; so will have risk for recession.
- Anterior openbite and mouth breathing (conflicting evidence)

  They thought it's a very important factor for gingivitis.

  Patients with incompetent lips and breath from their mouth, the gingiva becomes dry and so become more susceptible to gingivitis.

  It's true with true mouth breathers.

  You can find demarcation line on the gingiva in the dry area hidden under the lip

  But what is the actual evidence that this dryness cause more gingivitis we don't know. It's only empirical

  Also it doesn't make plaque control difficult as crowding.

But if you have bad plaque control you will have gingivitis whatever you breathe from your mouth or nose.

So the most important thing is **plaque control**

- Occlusal discrepancies

  occlusal trauma – two schools of thought; one school saying if you don't have plaque induced inflammation and you have occlusal trauma you won't have attachment loss BUT if you have **existing** periodontal infection and added to it occlusal trauma this will faster progression of periodontal attachment loss,the other school saying maybe you don't faster progression of periodontal attachment loss.

✓ *Orthodontic Tx*

- Inappropriate placement of bands (cements of band might go subgingival)
- Periodontal disease should be controlled before initiation of orthodontic Tx.
- Root resorption due to excessive forces of ortho Tx and the result in the amount of periodontum to support the tooth.

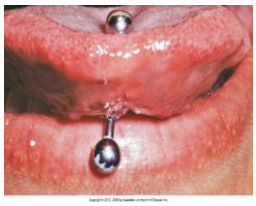
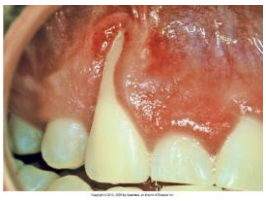
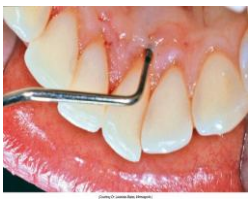
✓ *Extraction of Impacted 3rds*

- Two schools of thought one said that impacted 3rds cause late lower incisors crowding, other school said not relating.
- Often results in the creation of vertical defects on the distal of the 2nd molars so you take them out.
- more often when extracted >25 yrs of age
- other factors considered to extract 3rd molars : BOP on distal surface of 7, visible plaque, pathologically widened follicle because it might be cystic, root resorption, proximity to 2nd molar

important note :

If you have pocket on the distal of the 2nd molar and after you take out 3rd molar you have to doscaling and root planning on the distal of the 2nd molar in order to have healing with good chance of pocket reduction.

- ✓ *Habits and Self-Inflicted Injuries*
  - Trauma associated with oral jewelry
  - Toothbrush trauma
  - Chemical irritation


- ✓ *Smokeless Tobacco as 2ry local factor*

  Snuffand chewing tobacco

  Associated with Leukoplakiaand gingivalrecession and possibly greater susceptibility to periodontitis

- ✓ *Radiation*
  - Xerostomia
  - greater periodontal attachment loss and tooth loss inpatients treated with high-doseunilateral radiation

Reference for this lecture: ((very easy chapter in Carranza)).

11th edition: chapter 22 “The Role of Dental Calculus and Other Local Predisposing Factors”

DONE ☺