PROGNOSIS & TREATMENT PLAN

DEFINITION:

- □ The Prognosis is a prediction of the probable course, duration and outcome of a disease based on general knowledge of the pathogenesis of the disease and the presence of the risk factors for the disease.
- It is established after the diagnosis is made and before the treatment plan is established.

Prognosis is usually confused with the term RISK.

Difference between Prognosis & Risk

PROGNOSIS

- It is the prediction of the duration , course and the termination of the disease and its response to treatment.
- Prognostic factors are characteristics that predict the outcome of disease once the disease is present.

RISK

- Deals with the likelihood that an individual will get a disease in a specified period.
- Risk factors are those characteristics of an individual that put them at increased risk for getting a disease.

- Historically, prognosis classification schemes have been designed based on studies evaluating tooth mortality.
- Good prognosis: Control of etiologic factors and adequate periodontal support ensure the tooth will be easy to maintain by the patient and clinician.
- **Fair prognosis:** Approximately 25% attachment loss and/or Class I furcation involvement (location and depth allow proper maintenance with good patient compliance).
- **Poor prognosis:** 50% attachment loss, Class II furcation involvement (location and depth make maintenance possible but difficult).
- Questionable prognosis: >50% attachment loss, poor crown-to root ratio, poor root form, Class II furcations (location and depth make access difficult) or Class III furcation involvements; >2+ mobility; root proximity.
- Hopeless prognosis: Inadequate attachment to maintain health, comfort, and function.

Determination/Types of Prognosis

- Excellent Prognosis
- Good Prognosis
- ¬ Fair Prognosis
- Poor Prognosis
- Questionable Prognosis
- Hopeless Prognosis

Excellent Prognosis

■ No bone loss

Excellent Gingival Condition

Good Patient Cooperation

No systemic / Environmental Factors

Good Prognosis

- Adequate remaining bone support
- Adequate Possibilities to control etiologic factors & establish maintainable dentition
- Adequate Patient Cooperation
- No systemic/Environmental factors or if present are well controlled

Fair Prognosis

- Less than adequate remaining bone support
- Some tooth mobility
- Grade I furcation involvement
- Adequate maintenance
- Acceptable patient cooperation
- Presence of limited systemic/environmental factors

Poor Prognosis

- Moderate to advanced bone loss
- Tooth Mobility
- Grade I &II furcation involvements
- Doubtful patient cooperation
- Difficult to maintain areas
- Presence of systemic/Environmental factors

Questionable prognosis

- Advanced bone loss
- Grade II or III furcation involvement
- Tooth mobility
- Inaccessible areas
- Systemic/Environmental factors

Hopeless Prognosis

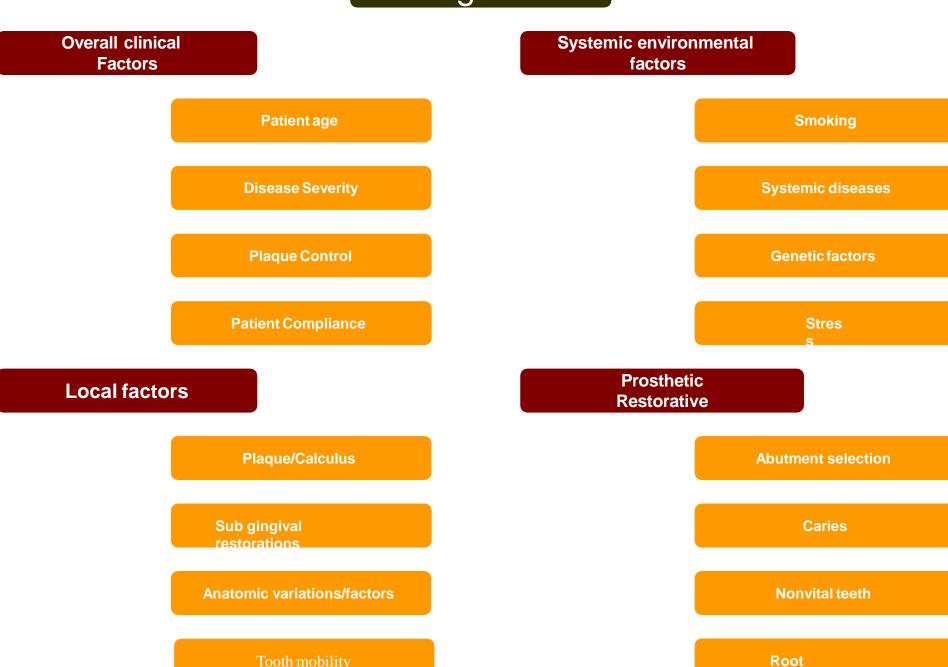
One or more of the following:-

Non maintainable areas

Extraction indicated- Grade III mobility

 Presence of uncontrolled systemic/environmental factors

Prognosis



restoration

Overall clinical factors

Patient age:-

Better in younger patients

Disease severity

Based on past history of periodontal disease

Following Factors must be recorded carefully:-

- Pocket depth
- Level of attachment
- Degree of bone loss
- 4. Type of bony defect

a) Pocket depth

Tooth with deeper pockets has better prognosis

b) Level of attachment

Poor prognosis if: - base of pocket is close to root apex

- Presence of apical disease

c) Degree of bone loss

Prognosis related to height of remaining bone loss

d) Type of bony defect

Prognosis depends upon:-

For Horizontal bone loss — height of existing bone

For Angular intrabony defects – contour of existing bone, no. of osseous walls

Plaque control

 Plaque is primary etiological factor for periodontal disese

 Effective removal of plaque is important for therapy and good prognosis

Patient compliance and co-operation

Prognosis depends upon

- patient's attitude
- 2. Desire to retain natural teeth
- Willingness and ability to maintain good oral hygiene

Systemic and environmental factors

- SMOKING:-
- -Most important environmental risk factor
- -Affects the healing of periodontal tissues
- -Prognosis improves with cessation of smoking
- Systemic disease:-
- Prognosis depends upon
- 1 Good control of diabetes
- 2 Correction of underlying systemic disorder

Genetic factors: -

several genetic variations are linked with periodontal disease Prognosis depends upon:-

Early detection and intervention/treatment/alteration in treatment regimens..

Stress:-

Alters patient's response to treatment
Includes:- .physical and emotional stress
.Substance abuse

LOCAL FACTORS

1. Plaque or calculus:-

- -most important local factor
- -prognosis depends upon removal of these etiological factors

2. Subgingival restorations:-

Contributes to plaque accumulation

increased inflammation

increased bone loss

-poorer prognosis than the supra-gingival margins

3. Tooth mobility:-

Prognosis improved by stabilisation of tooth mobility through the use of splint

4. Anatomic variations:-

factors predisposing to disease include

- 1. Short tapered roots with large crowns
- 2. Cervical enamel projection
- Enamel pearls
- 4. Intermediate bifurcation ridges
- Root concavities and proximities
- Developmental grooves
- 7. Furgation involvement

Short tapered roots have poorer prognosis because

- .disproportionate crown root ratio
- reduced surface area for periodontal support
- injured peridontium by occlusal forces
- Accessibility problems worsen the prognosis

PROSTHETIC/RESTORATIVE FACTORS

CONSIDERATION FOR PROGNOSIS INCLUDE:-

- 1- bone levels radiographically
- 2- attachment levels clinically
- Purpose of consideration:-
- To assess functional and aesthetic dentition
 - -- usefulness as abutment for prosthetic replacement

RELATIONSHIP BETWEEN DIAGNOSIS AND PROGNOSIS

FACTORS LIKE:-

- 1.patient's age
- 2. Severity of disease
- 3. Genetic susceptibility
- 4. Presence of systemic disease

ARE IMPORTANT CRITERIA IN
DIAGNOSIS AND ALSO IMPORTANTIN
DEVELOPING PROGNOSIS

PROGNOSIS FOR PATIENTS WITH GINGIVAL DISEASES

Plaque induced gingival disease

- 1- gingivitis associated with dental plaque only:-
- prognosis is good provided all local irritants are removed
- 2 diseases modified by systemic factors:-
- Prognosis depends upon control of bacterial plaque and correction of underlying systemic disorders
- 3 gingival diseases modified by medications:-
- Prognosis depends upon removal of offending drug/use of alternative medication to treat systemic disorders
- 4 -malnutrition
- Prognosis depends upon degree of severity and duration of deficiency

Non plequeeinduced gingiyav asionens:-

Seen in patients with bacterial, viral and fungal infections

Prognosis depends upon elimination of source of infectious agent

Prognosis for Patients with Periodontitis

Chronic Periodontitis

- Slowly Progressive disease associated with local environmental factors.
- 2) Can be localized or generalized.
- In case of not advanced attachment loss prognosis is generally good. But inflammation has to be controlled.
- In case of severe disease with furcations involvement and increasing clinical mobility, prognosis is fair to poor.

Aggressive Periodontisis

- Rapidly Progressive with minimal/no local factors.
- 2) Can be localized or generalized.
- Two Common features are observed.
 - a) Rapid Attachment loss and bon destruction
 - ы A familial aggregation
- 4) Hocalized type, which occurs around the age of puberty and is localized to first molars and incisors, if diagnosed early, prognosis is excellent. In advanced cases prognosis is still good. If the lesions are treated with local & systemic antibiotics.
- Generalized type, also seen in young patients with generalized interproximal attachment loss and poor antibody response. Aggrawated by cigarette smoking therefore prognosis is fair, poor or questionable.

Periodontitis as a manifestation of systemic diseases

Divided in to two categories:

- Periodontitis associated with hematologic disorders such as leukemia & acquired neutropenias.
- Periodontitis associated with genetic disorder such as familial and cyclic neutropenia, Down syndrome, Hypophosphatasia

Prognosis in these cases will be fair to poor.

Necrotizing Periodontal Dieseases

- In NUG the primary predisposing factors is bacterial plaque.
- With control of both the bacterial plaque and secondary factors Prognosis for the patient with NUG is good.
- Many Patients with NUP are immune compromised through systemic conditions. In these prognosis depend not only on inducing local and secondary factors but also on dealing with systemic problem.

Reevaluation of Prognosis after Phase I therapy

- A frank reduction in pocket depth and inflammation after phase I therapy indicates a favorable response to treatment and a better prognosis.
- If inflammatory changes can not be controlled by Phase I therapy, the overall prognosis may be unfavorable.

The lesion should be reanalyzed after completion of phase I therapy.

TREATMENT PLAN

After the diagnosis and prognosis have been established, the treatment is planned. The plan should encompass short and long-term goals.

• The treatment plan is the blueprint for case management.

■ It includes all procedures required for the establishment and maintenance of oral health and involves the following decisions:

- Need for emergency treatment (pain, acute infections).
- Teeth that will require removal.
- Periodontal pocket therapy techniques (surgical or nonsurgical).
- Endodontic therapy.

- The need for occlusal correction, including orthodontic therapy.
- The use of implant therapy.
- The need for caries removal and the placement of temporary and final restorations.
- Prosthetic replacements that may be needed and which teeth will be abutments if a fixed prosthesis is used.
- Decisions regarding esthetic considerations in periodontal therapy.
- Sequence of therapy.

MASTER PLAN FOR TOTAL TREATMENT

- Extracting or preserving a tooth
- Consideration of occlusal relationships
- Systemic conditions
- Supportive periodontal care

- THERAPEUTIC PROCEDURES
- EXPLAINING TREATMENT PLAN TO THE PATIENT

BOX 34-1 Phases of Periodontal Therapy

Preliminary Phase

Treatment of emergencies:

- Dental or periapical
- Periodontal
- Other

Extraction of hopeless teeth and provisional replacement if needed (may be postponed to a more convenient time)

Nonsurgical Phase (Phase I Therapy)

Plaque control and patient education:

- Diet control (in patients with rampant caries)
- Removal of calculus and root planing
- Correction of restorative and prosthetic irritational factors
- Excavation of caries and restoration (temporary or final, depending on whether a definitive prognosis for the tooth has been determined and the location of caries)
- Antimicrobial therapy (local or systemic)
- Occlusal therapy
- Minor orthodontic movement
- Provisional splinting and prosthesis

Evaluation of Response to Nonsurgical Phase Rechecking:

- Pocket depth and gingival inflammation
- Plaque and calculus, caries

Surgical Phase (Phase II Therapy)

- Periodontal therapy, including placement of implants
- Endodontic therapy

Restorative Phase (Phase III Therapy)

- Final restorations
- Fixed and removable prosthodontic appliances
- Evaluation of response to restorative procedures
- Periodontal examination

Maintenance Phase (Phase IV Therapy)

Periodic rechecking:

- Plaque and calculus
- Gingival condition (pockets, inflammation)
- Occlusion, tooth mobility
- Other pathologic changes

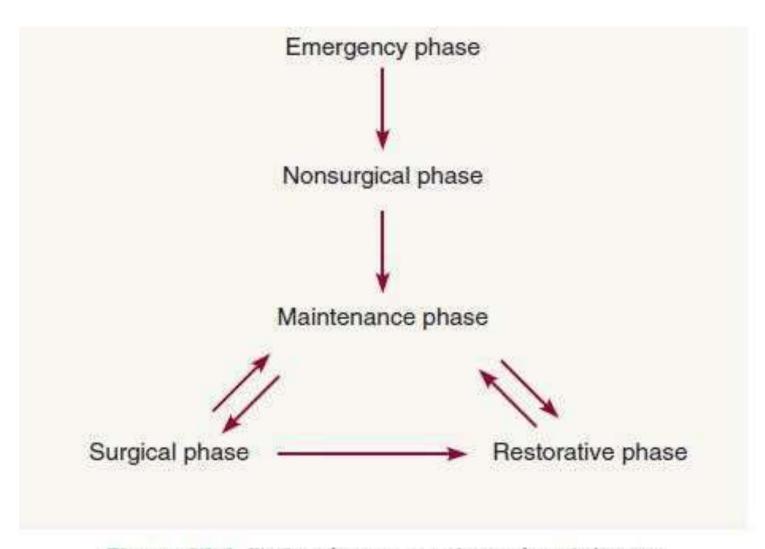


Figure 34-1 Preferred sequence of periodontal therapy.

Treatment planning should not result in just a list of procedures but should be phased.

The preliminary phase includes emergency treatment and is followed by the nonsurgical phase I, which leads to phase II, the surgical phase; then phase III, the restorative phase; and the final phase is the maintenance phase. Part of this approach to treatment planning is the incorporation of comprehensive reevaluation of periodontal status on the completion of each phase.

Patient involvement is essential; clinicians need to develop good communication skills so that patients are informed and motivated to optimize their periodontal status and have their lives enhanced by a healthy functional dentiition.

Advanced Diagnostic Aids

 Periodontitis: is characterized by a loss of connective tissue attachment that begins at, or just apical to, the cementoenamel junction and extends apically along the root surface.

Disease Activity?

Periodontal Disease Markers?

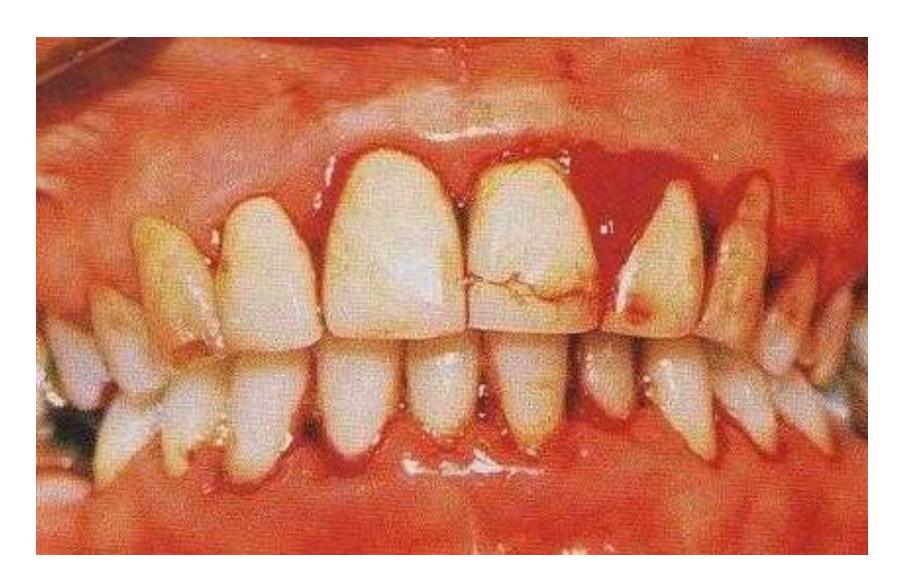
How are they Important??



Conventional clinical diagnostic tools. **A,** Visual diagnosis. **B,** Periodontal probing. **C,** Bleeding on probing. **D,** Intraoral periapical radiographic series.

- Advances in Clinical diagnosis
- Advances in Radiographic Assessment
- Advances in Microbiologic Analysis
- Advances in Characterizing the Host Response

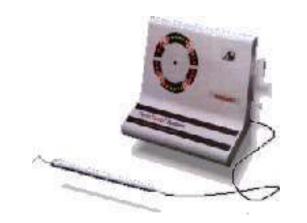
I. Advances in Clinical Diagnosis



Gingival Bleeding

 Gingival bleeding is related to the persistent presence of plaque on the teeth and is regarded as a sign of the associated inflammatory response.

 Gingival bleeding is a good indicator of the presence of an inflammatory lesion in the connective tissue at the base of the sulcus and that the severity of bleeding increases with an increase in size of the inflammatory infiltrate.



Gingival Temperature:

- → Subgingival temperature as a diagnostic tool (PerioTemp)
- → Subgingival temperature in increased at diseased sites. (increased cellular and molecular activity)
- → Variation in natural temperature gradient.
- →Smokers have differences in subgingival temperature and sublingual temperature.



Thermal periodontal probe system: PerioTemp electronic monitor. (Courtesy Abiodent, Danvers, Mass.)

Periodontal probing:

Gold standard is recording changes over period of time.

Problems with probing:

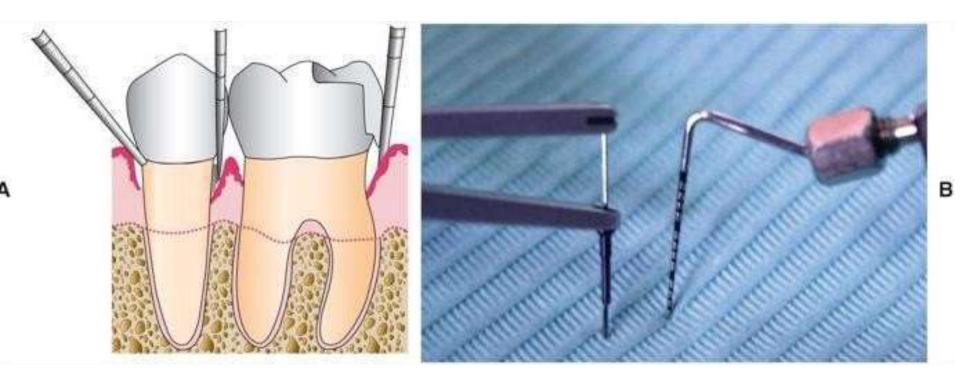
→ Lack of sensitivity and reproducibility.

Probing depends on: force, angulations, size of probe, precision of calibration, presence of inflammation.

{force to probe pocket: 30g}

{force to probe periodontal osseous defect: 50g}





A, Limitations in periodontal probing. B, Probing pressure caused by probe angulation, presence of subgingival calculus, and presence of overhanging restorations.

Florida probe:

- →Tip is 0.4mm
- →Sleeve- edge provides reference to make measurements



- → Coil Spring; provides constant probing force
- → Computer for data storage.



FP Handpiece tip as it enters the sulcus.

FP Handpiece tip with constant force in use (tip at bottom of sulcus) and sleeve properly positioned at the top of the gingival margin allowing the computer to measure the difference (3.0 mm).

- Disadvantages of Florida probe.
- → Lack of tactile sensitivity
- → Fixed probing force
- → Underestimation of deep periodontal pockets.

Other electronic probes:

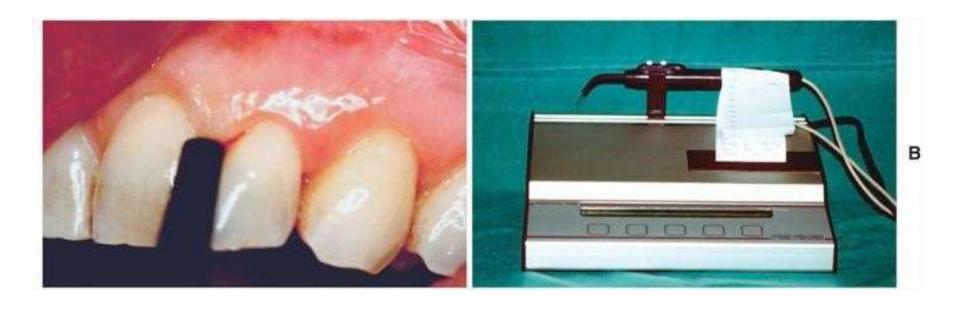
Improvised Florida PASHA probe

Interprobe

Perioprobe

Foster Miller probe

Toronto Automate (difficult to reproduce patient head position and in 2nd and 3rd Molar area.)



Periprobe electronic periodontal probing system. A, Measuring device inserted in the sulcus. B, Probing unit.

II. Advances in Radiographic Assessment

- Problems with conventional Radiography:
- → Variation in projection geometry
- → Variation in contrast and density
- → Masking by other anatomic structures.

Digital Radiography:

Advantage: digital storage, image enhancement, and radiation dose reduction.

Substraction Radiography:

→Serial radiographs → converted to digital images → superimposed → composite image—Quantitative changes

→ Limitations: needs paralleling technique and accurate superimposition.

Advantages:

- → High correlation between alveolar bone loss and CAL changes
- Increased detection of small osseous lesion
- → Both quantitative and qualitative visualization
- → More sensitive

Disadvantage:

→ Identical projection alignment during sequential radiographs

Digital SR: uses position device for films and software that corrects angular alignment discrepancy.

 Computer Assisted Densitometric Image Analysis. (CADIA)

Video camera measures the light transmitted through radiograph and the signals form the camera is converted to gray scale image.

Advantage:

- → Measures quantitative changes in bone density overtime.
- → Higher sensitivity, reproducibility and accuracy as compared to DSR.

Digital OPG



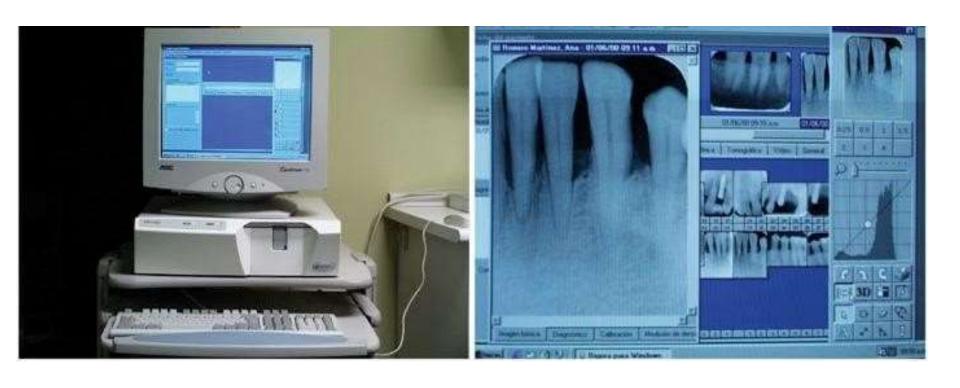
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How we play!



RVG





Digital radiographic system: Digora.

III. Advances In Microbiologic Analysis

- Subgingival microenvironment has 300+ species
- Only few organisms are thought to be involved with periodontal disease.
- Strong evidence for Aa, Pg, and Tf.
- Other organisms that are thought to have etiologic role are Camphylobacter rectus, Eubaterium nodatum, Fusobacterium nucleatum, Peptostreptococcus micros, Prevetolla intermedia and Prevetolla nigrescens, Td.

Uses of microbiologic analysis

- → Support diagnosis.
- → Treatment planning
- →Indicator for disease activity (absence is a better indicator)

Bacterial culturing

 Plaque samples are cultivated under anaerobic conditions using selective and nonselective media.

Advantage:

Relative and Absolute count of the cultured species.

Disadvantage:

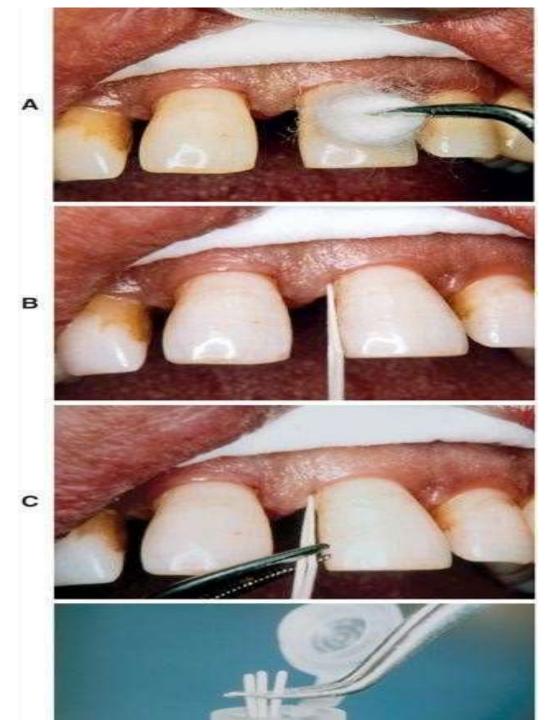
Strict sampling conditions

Difficulty in culturing most organisms

Low sensitivity: organisms lesser then 10³ is difficult to detect

Time consuming

Expensive equipment and experienced personnel

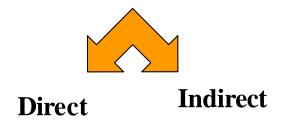




 Direct Microscopy: Most of the periodontal pathogens are non-motile so it is difficult to identify.

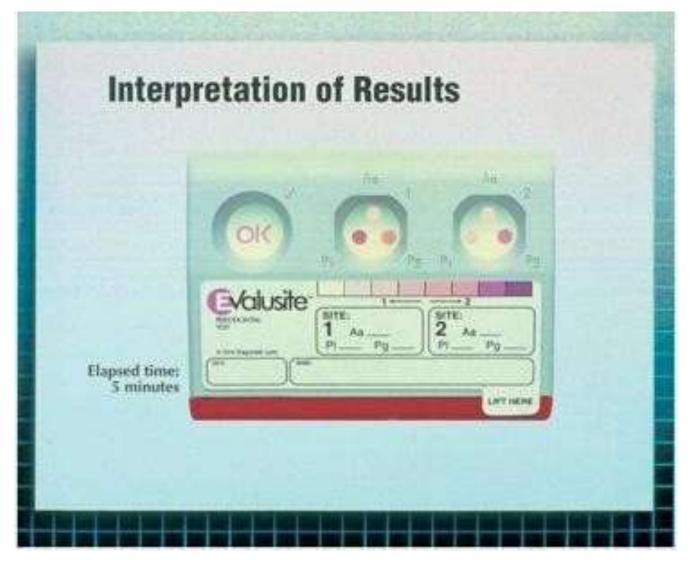
Immunodiagnostic Methods

Immunofloresence Assay (IFA):

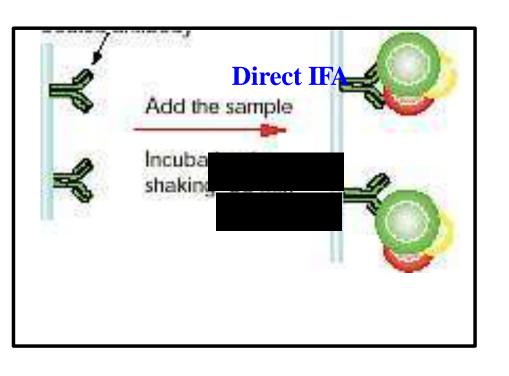


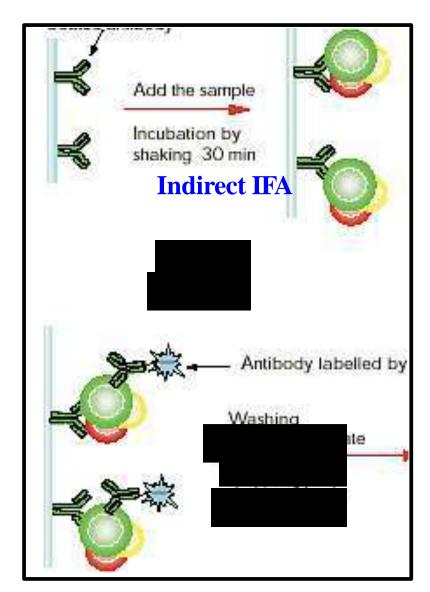
Direct IFA: AB conjugated with Fluorescein marker + Bacteria (Antigen) = Immuno complex

Indirect IFA: Primary AB + Bacteria= Immune Complex+ Secondary Fl conjugated AB



Enzyme-linked immunosorbent assay (ELISA, Evalusite) demonstrating positive results for *Aggregatibacter* actinomycetemcomitans.





Flow Cytometry

Bacterial cells+ species specific AB + Secondary FL Conjugated AB → Introduced in flowcytometer

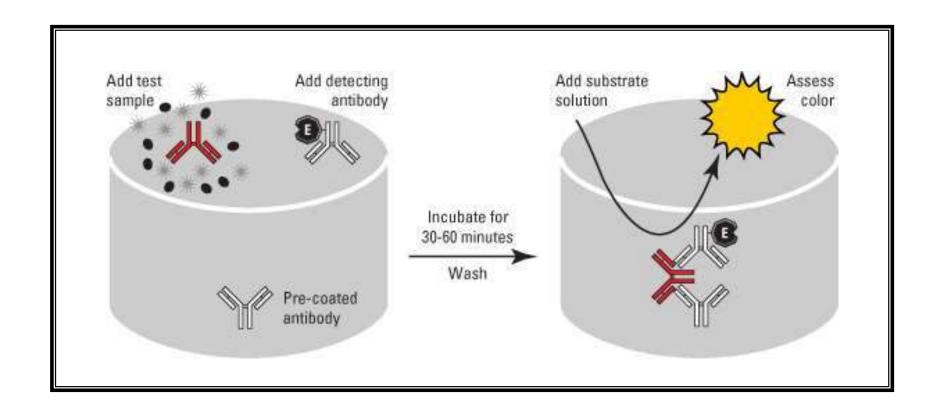
→Bacterial cells is separated into single cell suspension-→ passes through the tube → Cells identified by lasers.

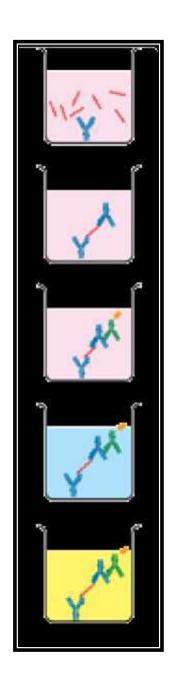
ELISA= Enzyme Linked Immunosorbent Assay

Similar AB and Antigen reaction, but the fluorescence is read using a photometer.

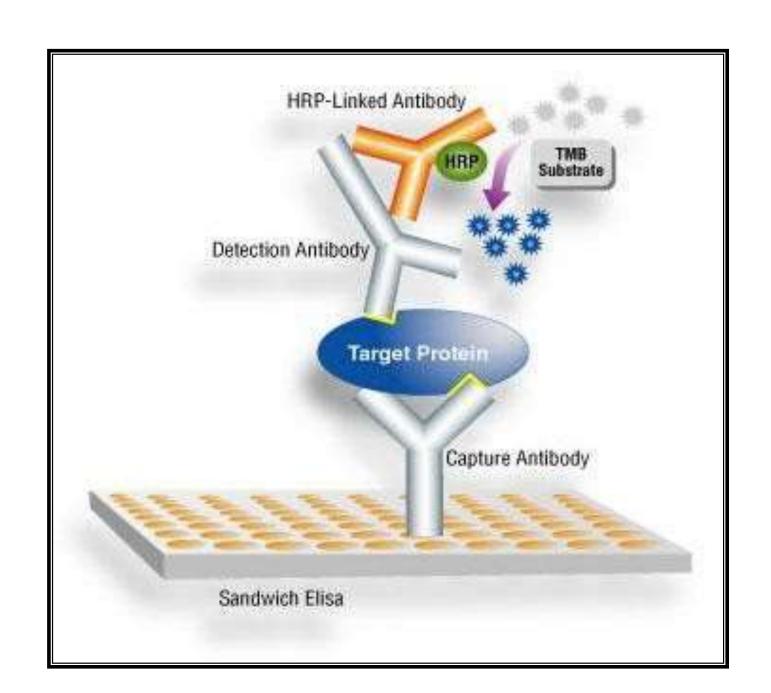
Evalusite: commercially available kit to detect Aa,Pg and Pi.







- → Well with precoated antibody + Sample to be tested= immune complex
- →Specific antigen bind to the antibody + Secondary antibody added.
- →Immunofloresence dye bound to secondary antibody
- → Substrate added which changes the color of the solution
 - →Amount of florescence checked by photometer (450nm)



Latex Agglutination Test

Latex beads coated with species specific AB→ when beads come in contact with specific species in sample they bind and agglutination occurs → clumping of beads is visible → test positive.

Advantages:

Simple and Rapid testing Higher sensitivity and specificity.

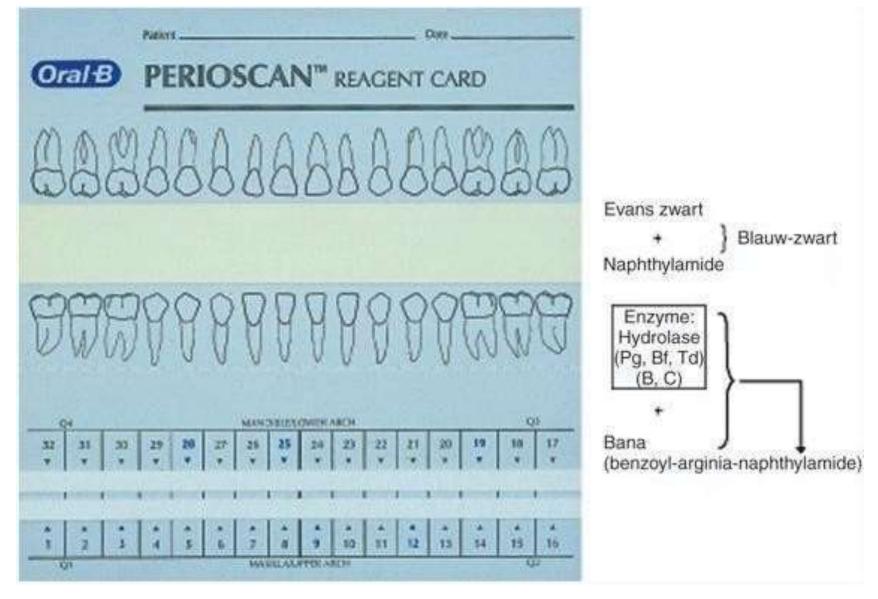
Enzymatic Methods

- Bacteria release specific enzymes. Certain group of species share common enzymatic profile.
- e.g. Tf, PG, Td, and Capnocytophaga species release trypsinlike enzyme.
- Enyme hydrolysis specific substrate (BANA) >> release cromophore (B-naphtalamide) >> Cromophore changes color on addition of a substance(fast garnet)

Perioscan is a popular diagnostic kit uses BANA reaction.

Disadvantage:

- → May be positive in clinically healthy site
- → Cannot detect disease activity
- → Limited organisms detected
- →Other pathogens may be present if it's negative.

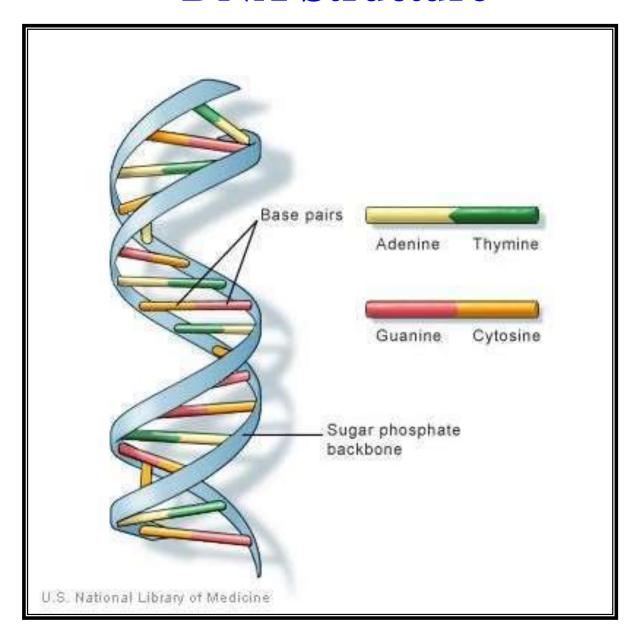


Diagnostic kit (Perioscan) developed for the identification of specific bacteria profiles using an enzymatic reaction from plaque isolates.

Molecular Biology Techniques

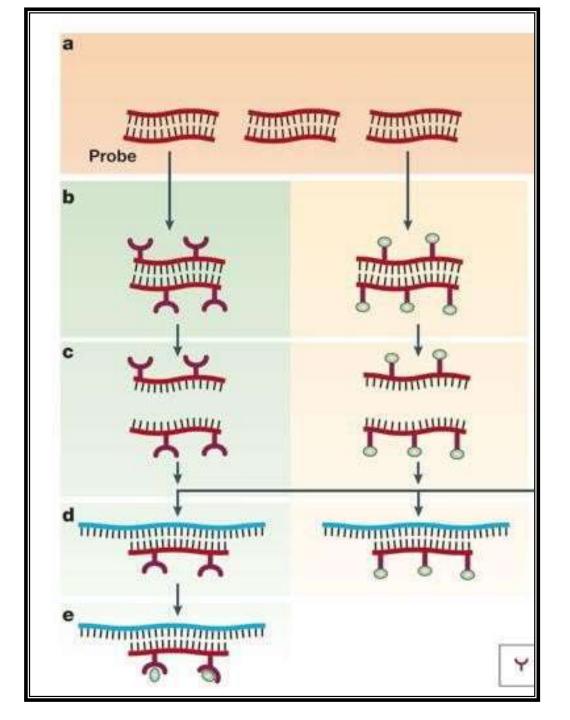
- Basic Principle: Analysis of DNA, RNA and protein structure.
- Hybridization: Pairing of complimentary strands of DNA to produce a double stranded DNA.
- **Nucleic acid probe:** is a known DNA/RNA which is synthesized artificially and labeled with a enzyme or a radioisotope for detection when placed in a plaque sample.

DNA Structure



DNA Probe: uses a segment of a single stranded DNA, labeled with a enzyme of a radio isotope, that is able to hybridize to a complimentary nuclei strand, and thus detect presence of target microorganism.

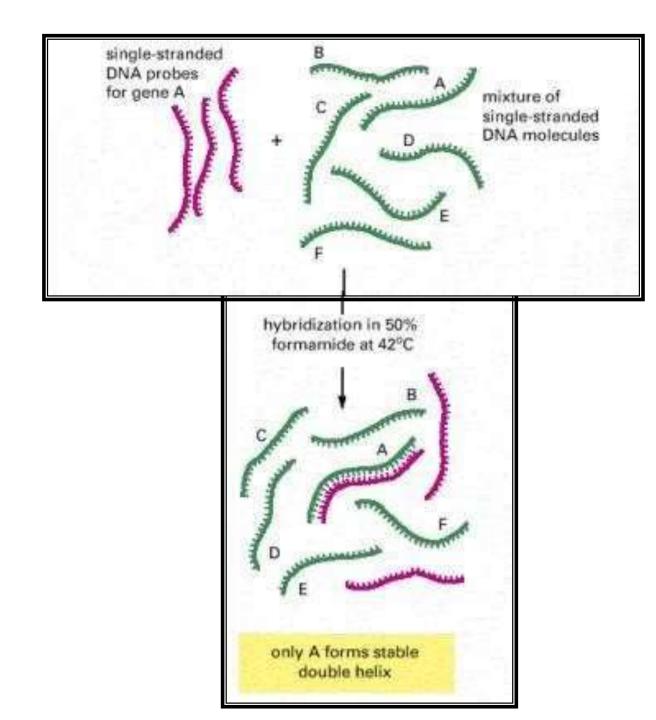
DNA Probe



DNA Probe

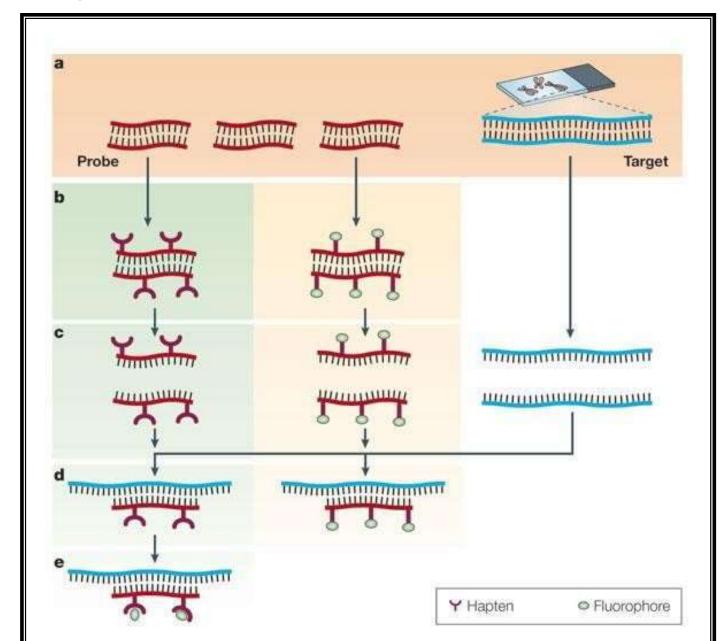
- Two types of DNA probes
- Whole genomic: Targets the whole DNA strand rather then a specific sequence or gene.
- → High chances to cross react with non target microorganism
- → Lower sensitivity and specificity.

Whole GenomicDNA Probe



- Oligonucleotide probes: target variable region of 16sRNA or a specific sequence in the DNA strand.
- → Higher sensitivity and specificity.

Oligonucleotide probes



 Checkerboard DNA-DNA Hybridization Technology:

Developed by Socransky et.al.

40 bacterial species can be detected using whole genomic digoxigenin-labeled DNA probes.

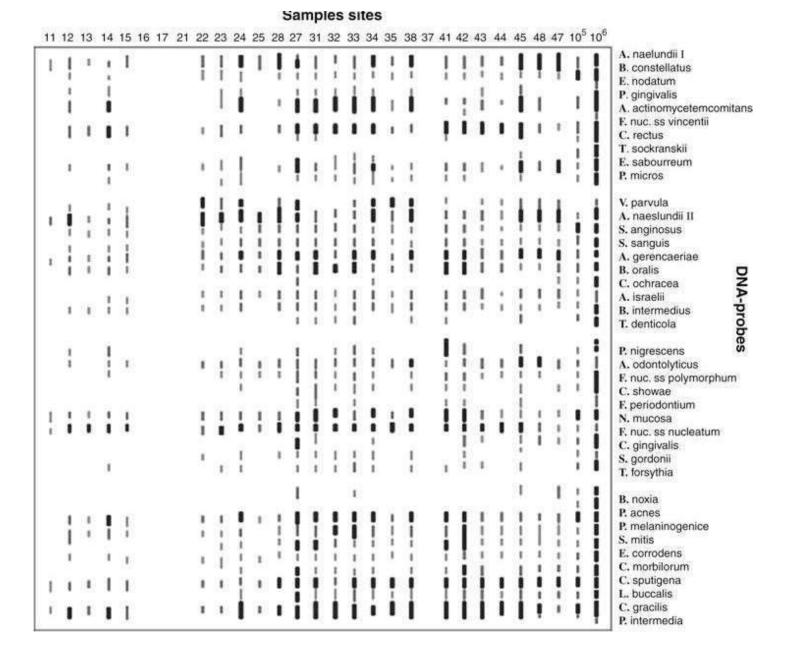
Large number of samples can be tested and upto 40 oral species detected with a single test.

Advantages of DNA probes as compared to bacterial culturing.

- 1. More sensitive and specific
- Requires as less as 10⁴ cells of each species to be detected.
- 3. Multiple species detected with a single test
- 4. Does not require viable bacteria
- 5. Large number of samples can be assessed.

Disadvantage:

- 1. Expensive
- 2. Expert personnel to carry out the test
- 3. Not easily available

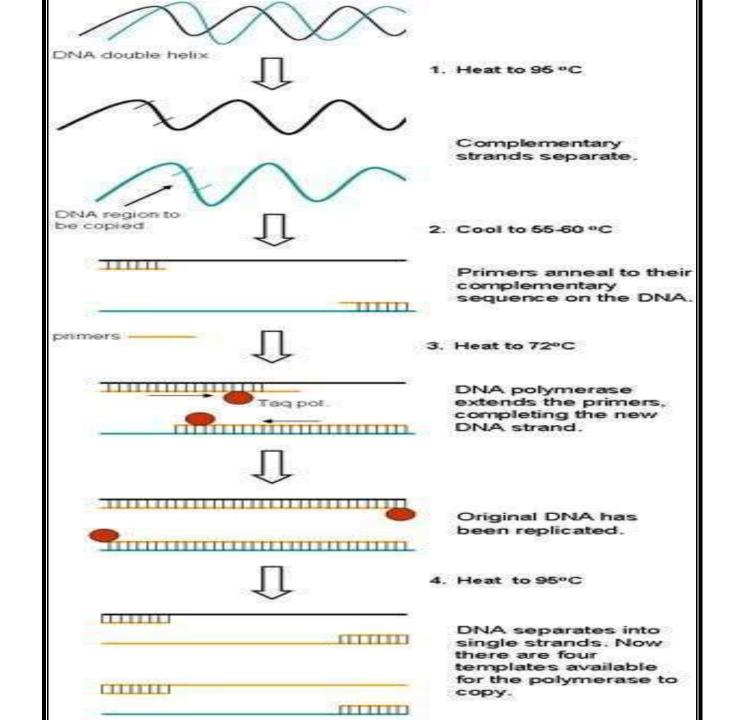


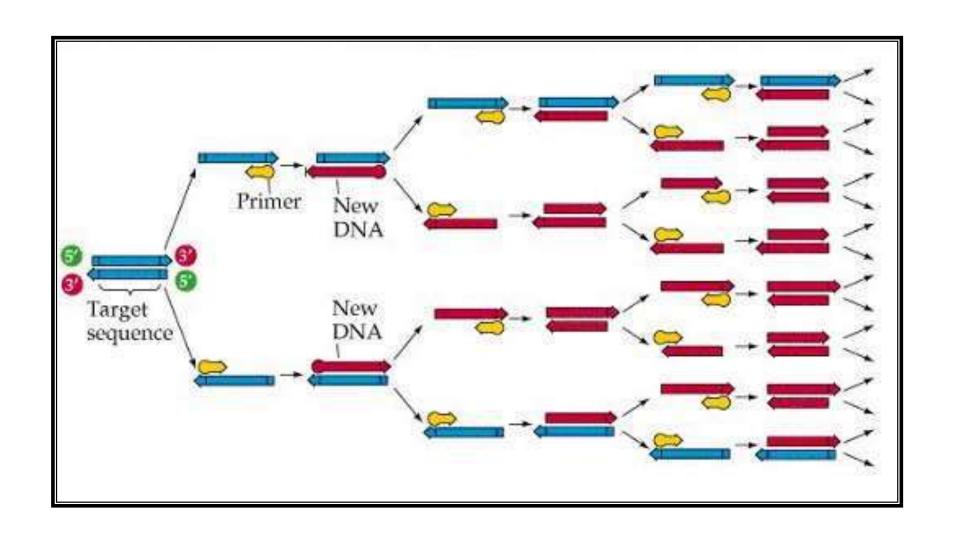
Checkerboard DNA-DNA hybridization technology for detecting 40 bacterial species often found in the oral cavity.

Polymerase Chain Reaction (PCR):

Involves amplification of a region of DNA by a primer specific to the target species.

If there is amplification then it indicates the presence of the target species in the sample.





Advantages:

- High detection limit. As less as 5- 10 cells can be amplified and detected.
- Less cross reactivity under optimal conditions
- 3. Many species can be detected simultaneously

Disadvantage:

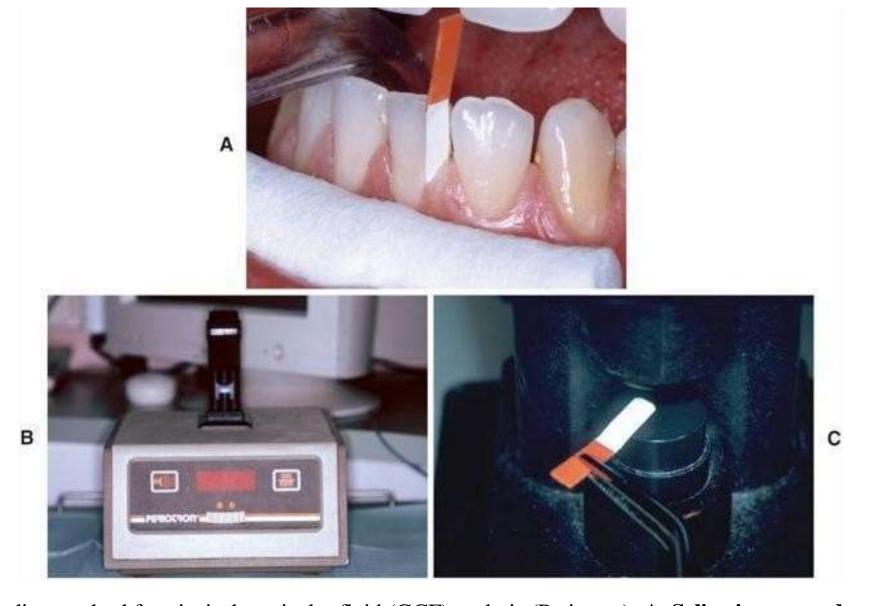
- 1. Small quantity needed for reaction may not contain the necessary target DNA
- 2. Plaque may contain enzymes which may inhibit these reactions.

IV. Advances in Characterizing Host Response

- Asses host response by studying mediators as a response to specific bacteria or local release of inflammatory mediators or enzymes as response to infection.
- Source of samples may be; GCF, Saliva, or Blood.
- GCF is most commonly used, where as saliva is been recently been researched recently.

• GCF:

- Most well studied, with almost 40 components in form of host-derived enzymes, tissue breakdown products, and inflammatory mediators.
- → Collected with paper strips, micro papillary tubes, micropipettes, microsyringes, plastic strips.
- →Paper strips commonly used, introduced in sulcus for 30 secs and volume is measured using Periotron 6000.
- → Periotron measures the capacitance across the wet paper strip, which is converted to digital reading.
- → Periotron reading have high correlation with clinical gingival indices.
- → Quickest and easiest way to measure GCF.



Sampling method for gingival crevicular fluid (GCF) analysis (Periotron). **A, Saliva is removed** from the tooth surface with cotton wool before placement of a periopaper strip into the pocket to collect GCF. **B, The moist paper strip is removed and placed between the jaws of the Periotron** for assessment of fluid content. **C, Periotron device.**

- Saliva: is the next most used after GCF
- easily collected
- contain both local and systemic derived markers for periodontal disease
- → Collected from parotid, sub-mand or sub lingual or as 'Whole saliva'
- → Whole saliva contains secretions of major and minor salivary glands, desaqumated cells, and GCF.
- →No diagnostic test available in the market although lot of research is in progress.
- → Markers to look for in saliva: proteins and enzymes from host, phenotypic markers, host cells, hormones, bacteria, bacterial products, volatile compounds, and ions.

Inflammatory Mediators and Products

Cytokines: are substances released by cells of the immune system.

Cytokines in GCF are: TNF-alpha, IL-1, IL-6, and IL-8

Have actions on immune cells and release of enzymes, including bone resorption.

Can be used to determine the disease activity.

Esp. Prostaglandin E in increased in GCF of periodontitis patients.

Can be used to determine disease activity

Host Derived Enzymes

Aspartate amino trasnferase (AST), Alkaline phosphatase, B-glucoronidase (BG), elastase, cathespins, and MMPs.

AST: derived from dead cells.

Elevated in periodontal disease.

Periogard is a commercially available colorimetric test.

Cannot differentiate active and inactive sites.

ALP: released from osteoblast, neutrophils, fibroblast..

BG and Elastase: found in Neutophils.

BG, Elastase, Neutral protease, and cathepsins

→all shown to be higher in diseased sites. May be used to predict severity of disease or to predict disease activity.

- Matrix metalloprotienases:
- → zinc and calcium dependent enzymes
- →constitutively formed in the body, secreted by fibroblast and macrophages.
- →Normally help in degrading and remodeling of extracellular matrices.
- →In chronic periodontitis they cause the degradation of the collagen fibrils in PDL and Alveolar bone.
- → MMPs 2,3,8 9 and 13 play important role.
- → MMP 8 level is associated with the attachment loss
- →Level reduces in response to treatment. (Chair side test kit)
- → Can be used to indicate present disease status and predictor of future disease.

Tissue Breakdown Products

The extracellular matrix of the periodontium is composed of a diverse number of macromolecules; the predominant one is collagen, and the other components include proteoglycan (versican, decorin, biglycan, syndecan) and non-collagen proteins (elastin, fibronectin, laminin, osteocalcin, osteopontin, bone sialoprotein, osteonectin, tenascin). All these matrix components are theoretically detectable and potentially informative in terms of their clinical diagnostic utility.

CONCLUSION

 Although there are many potential markers for periodontal disease activity and progression, numerous features still hamper the ability to use them as diagnostic tests of proven utility. There is still a lack of a proven "gold standard" of disease progression, and thus the correlation of these potential markers with proven clinical attachment loss may be a potential confounder in any proposed test.

Endodontic Periodontal Lesions

Anatomic Considerations

- There is an intimate relationship between the periodontium and pulpal tissues
- As the tooth develops and the root is formed, 3 main avenues for communication are created:
 - Apical Foramen
 - Lateral and Accessory Canals
 - Dentinal Tubules

Additional Avenues of communication between the Pulp and the Periodontium

- Developmental malformations such as palatogingival grooves of maxillary incisors. These usually begin in the central fossa, cross the cingulum, and extend apically with varying distances
- Perforations these may result from extensive carious lesions, resorption, or from operator error
- Vertical root fractures these can produce deep periodontal pocketing and localized destruction of alveolar bone. The fracture site provides a portal of entry for irritants from the root canal to the PDL

Endodontic Disease and the Periodontium

When the pulp becomes inflamed or necrotic, inflammatory byproducts may leach out through the apex, lateral and accessory canals as well as the dentinal tubules to trigger an inflammatory vascular response in the periodontium

Seltzer and Bender 1967

Periodontal Disease and the

Periodontal Disease and the Pulp

- The effect of periodontal inflammation on the pulp is controversial and conflicting studies exist:
 - It has been suggested that periodontal disease has no effect on the pulp, at least until it involves the apex (Czarnecki & Schilder, '79)
 - On the other hand, some studies suggest that the effect of perio disease on the pulp is degenerative in nature including an increase in calcifications, fibrosis and collagen resorption in the pulp (Langeland et al '74 and Mandi '72)
 - It has been reported that pulpal changes resulting from periodontal disease are more likely to occur when the apical foramen is involved (Langland et al '74)

Differential Diagnosis of Endo/Perio Lesions

- The following classification system was developed by Simon, Glick and Frank in 1972:
 - Primary Endodontic Disease
 - Primary Periodontal Disease
 - Primary Endo w/ Secondary Perio
 - Primary Perio w/ Secondary Endo
 - True Combined Lesions

- Typically, endodontic lesions resorb bone apically and laterally and destroy the attachment apparatus adjacent to a nonvital tooth
- It is possible for an acute exacerbation of a chronic periapical lesion on a tooth with a necrotic pulp to drain through the PDL into the gingival sulcus. This clinical presentation mimics the presence of a periodontal abscess, or a deep periodontal pocket

- When endodontic infection drains through the PDL, the pocket is very narrow and deep. In reality, it is a sinus tract of pulpal origin that opens through the PDL, and not breakdown due to periodontal disease
- A similar situation can occur where drainage from the apex of a molar tooth extends coronally into the furcation area. These cases resemble a "through-and-through" furcation defect (Grade III) of periodontal disease

 For diagnostic purposes, it is imperative to trace the sinus tract by inserting a guttapercha cone and exposing one or more radiographs to determine the origin of the lesion

The sinus tract of endodontic origin is readily probed down to the tooth apex, where no increased probing depth would otherwise exist around the tooth

 Primary endodontic disease will heal following root canal treatment

The sinus tract extending into the gingival sulcus or the furcation area disappears at an early stage once the necrotic pulp has been removed and the root canals are well sealed

Primary Periodontal Disease

- Caused by periodontal pathogens
- It is the result of progression of chronic periodontitis apically along the root surface

Pulp tests yield a clinically normal pulpal reaction

Primary Periodontal Disease

- Frequently accumulation of plaque and calculus are seen throughout the dentition
- Periodontal pockets are wider, and are generalized
- The prognosis depends on the stage of periodontal disease and the efficacy of periodontal treatment

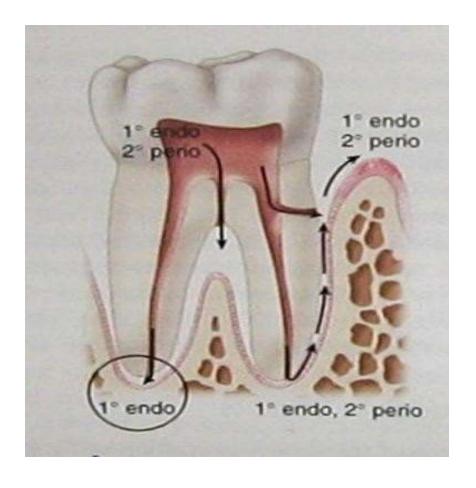
Primary Endo with Secondary

Primary Endo with Secondary Perio

- This happens with time as suppurating primary endodontic disease remains untreated, it may become secondarily involved with periodontal breakdown
- Plaque forms at the gingival margin of the sinus tract and leads to plaqueinduced periodontitis in the area

Primary Endo with Secondary Perio

 The pathway of inflammation into the periodontium is through the apical foramen, accessory and lateral canals



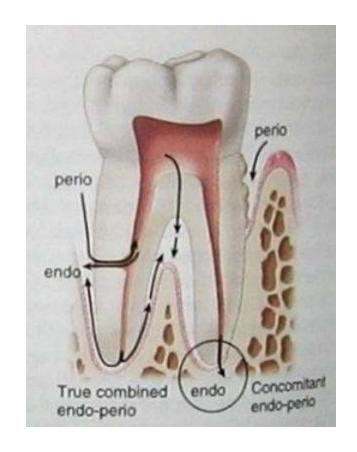
Primary Perio with Secondary Endo

 The progression of periodontitis by way of lateral canal and apex to induce a secondary endodontic lesion



True Combined Disease

Concomitant endoperio lesion is an additional classification that has been proposed to describe the presence of endo and perio disease as two separate and distinct entities



Diagnosis

- A thorough clinical and radiographic examination is imperative for developing a diagnosis
- Data Collected must include:
 - periapical radiographs
 - pulp vitality testing: cold, EPT, cavity test
 - percussion
 - palpation
 - pocket probing
 - sinus tract tracking
 - cracked tooth testing: transillumination, tooth-slooth, staining

Aggressive Periodontitis

Periodontitis

"Periodontitis is defined as an inflammatory disease of the supporting tissues of teeth caused by specific organisms or a group of specific organisms, resulting in progressive destruction of periodontal ligament and alveolar bone with pocket formation, recession, or both."

•The clinical feature that distinguishes periodontitis from gingivitis is the presence of clinically detectable attachment loss.

Classification of Periodontitis

- •According to AAP World Workshop in Clinical Periodontitis, 1989
 - 1. Adult Periodontitis
 - 2. Early onset Periodontitis(may be prepubertal, juvenile or rapidly progressing)
 - 3. Periodontitis associated with systemic disease
 - 4. Necrotizing Ulcerative Periodontitis
 - 5. Refractory Periodontitis

- According to European Workshop on Periodontology, 1993
 - 1. Adult Periodontitis
 - 2. Early Onset Periodontitis
 - 3. Necrotizing periodontitis
- According to AAP International Workshop for Classification of Periodontal Diseases, 1999
 - 1. Chronic Periodontitis
 - 2. Aggressive Periodontitis
 - 3. Periodontitis as manifestation of systemic disease

Types Of Aggressive Periodontitis:

- Localized Aggressive Periodontitis
- Generalized Aggressive Periodontitis

Localized Aggressive Periodontitis:

•In this there is localized first molar/incisor presentation with interproximal attachment loss on at least 2 permanent teeth, one of which is a first molar, and involving no more than 2 teeth other than first molars and incisors

Clinical Characteristics:

- Usually has an age onset around puberty
- •Localized first molar/incisor presentation with interproximal attachment loss on at least 2 permanent teeth, one of which is a first molar, and involving no more than the 2 teeth other than first molars and incisors

Reasons for the limitation of periodontal destruction to certain teeth have been suggested as:

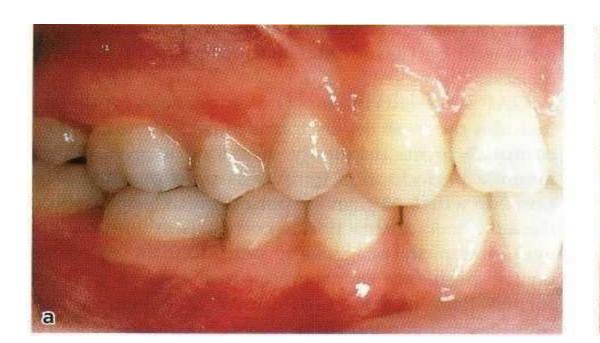
1. After initial colonization of the first permanent teeth to erupt(the first molars and incisors), Actinobacillus actionomycetemcomitans evades the host defenses by different mechanisms.

- Mechanisms including production of polymorphonuclear leukocyte chemotaxis -inhibiting factors, endotoxin, collagenases, leukotoxin, and other factors that allow bacteria to colonize the pocket and initiate the destruction of periodontal tissues.
- After this initial attack, adequate immune defenses are stimulated to produce opsonic antibodies to enhance the clearance and phagocytosis of invading bacteria and neutralize leukotoxic activity.
- In this manner, colonization of other sites may be prevented. A strong antibody response to infecting agents is one characteristics of LAP.

- 2. Bacteria antagonistic to A. actinomycetemcomitans may colonize the periodontal tissues and inhibit it from further colonization of periodontal sites in the mouth. This would localize A. actinomycetemcomitans infection and tissue destruction.
- 3. A. actinomycetemcomitans may lose its leukotoxin producing ability for unknown reasons. If this happens, the progression of the disease may become arrested or retarded and colonization of new periodontal sites averted.

4. The possibility that a defect in cementum formation may be responsible for the localization of the lesions has been suggested. Root surfaces of teeth extracted from patients with LAP have been found to have hypoplastic cementum. This was true not only of root surfaces exposed to periodontal pockets but also of roots still surrounded by their periodontium.

- •A Striking feature of LAP is the lack of clinical inflammation despite the presence of deep periodontal pockets
- •Although the quantity of plaque is minimal, it often contains elevated levels of A. actionomycetemcomitans, and in some patients, Porphyromonas gingivalis.
- •As the name suggests, the rate of bone loss is about 3 to 4 times faster than in chronic periodontitis.
- •There may also be distolabial migration of maxillary incisors with concomitant diastema formation.





Healthy gingival status of a patient of LAP



The interdental papilla between teeth 31 and 32 has been lost



LAP in a 22-year-old systemically healthy African-American female patient

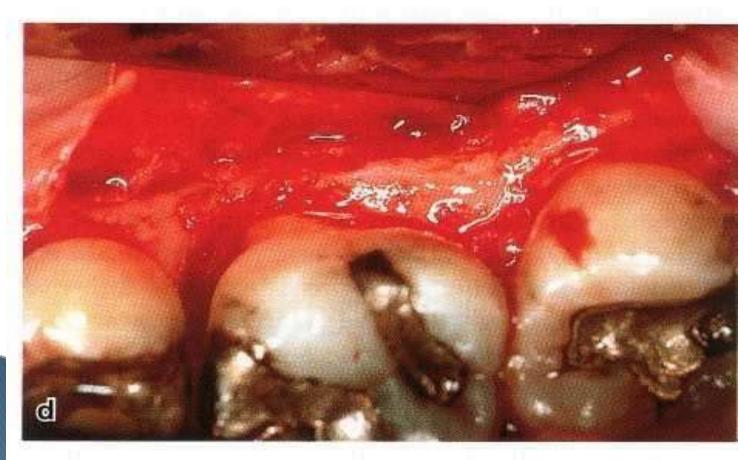
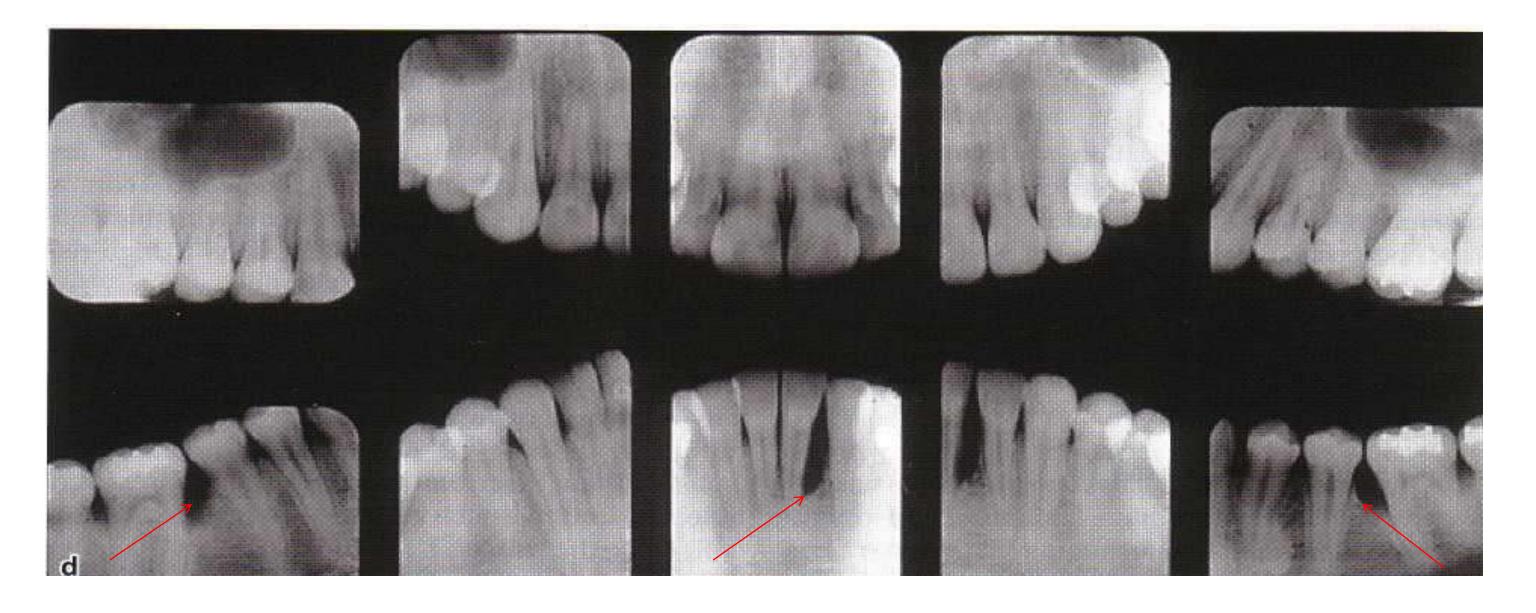


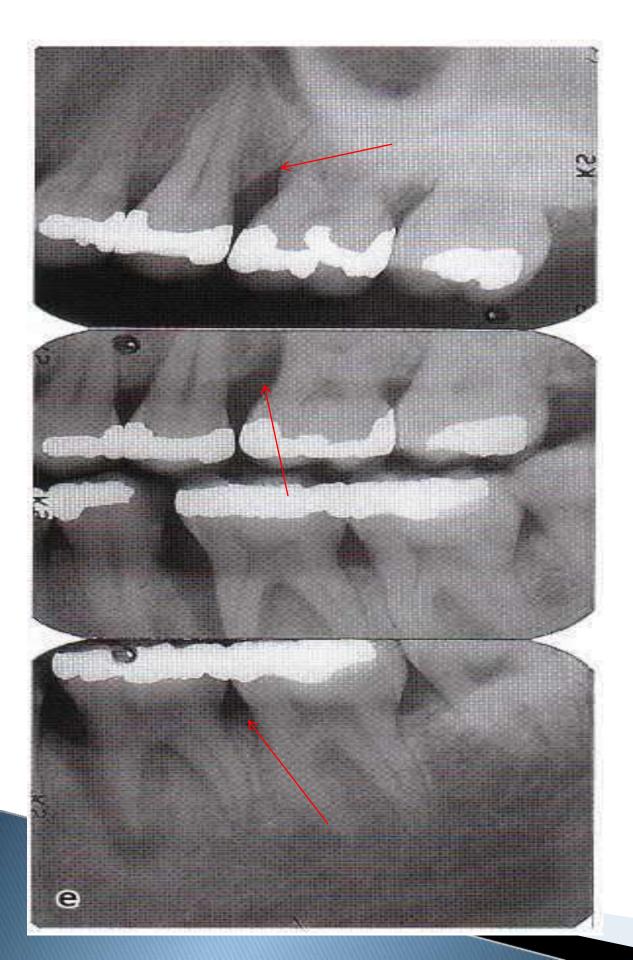
Fig. 9-11. (c-e).

Radiographic Findings:

- Vertical loss of alveolar bone around the first molars and incisors
- •Radiograph may include an arc shaped loss of alveolar bone extending from the distal surface of the second premolar to the mesial surface of the second molar



Intraoral radiographs show the presence of localized angular bony defects, associated with clinical attachment level loss, at the mesial aspect of tooth 46, 36 and at the distal aspect of tooth 31. No significant bone loss and/or attachment loss was detectable in other areas of the dentition



Bone Loss in localized areas suggestive of LAP



Prevalence & Distribution by Age and Sex:

Studies show that-

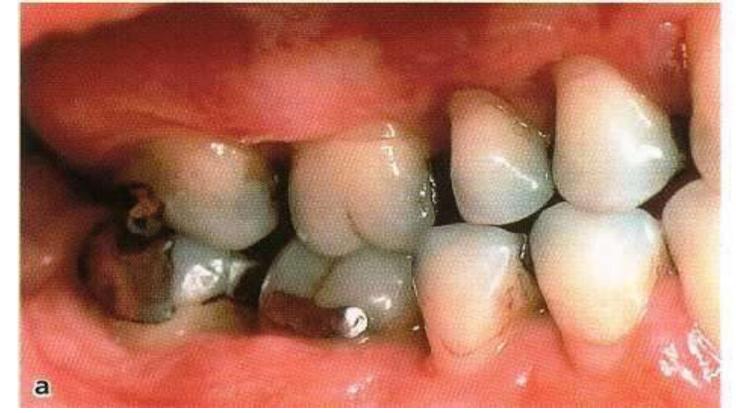
- Prevalence of LAP is estimated to be below 1%
- Blacks were reported to be at much higher risk
- •Black males were 2.9 times more likely to have the disease than black females
- •White females were more likely to have the disease than white males
- •Seen most frequently in the period between puberty and 20 yrs of age

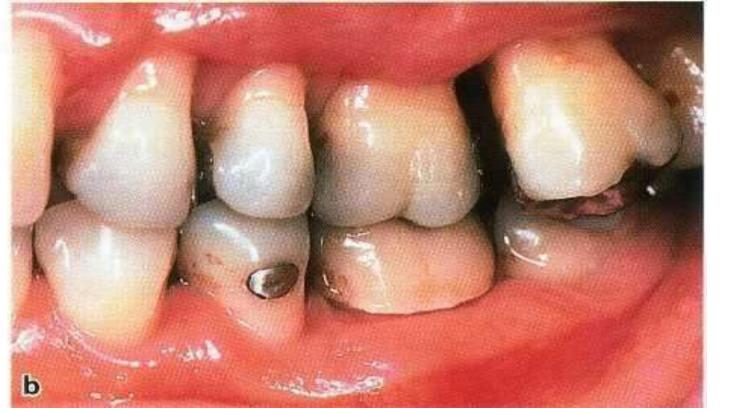
Generalized Aggressive Periodontitis

- •It is characterized by generalized interproximal attachment loss affecting at least 3 permanent teeth other than first molars and incisors
- •In contrast to LAP, evidence suggests that individuals affected with Generalized Aggressive Periodontitis(GAP) produce a poor antibody response to pathogens present
- •The destruction appears to occur episodically with periods of advanced destruction followed by stages of quiescence of variable length (weeks to months or years)

Clinical Characteristics:

- •As seen in LAP, patients with GAP often have small amounts of bacterial plaque associated with the affected teeth
- •Quantitatively, the amount of plaque seems inconsistent with the amount of periodontal destruction
- •Quantitatively, P. gingivalis, A. actinomycetemcomitans, Bacteriods forsythus frequently are detected in the plaque that is present







A32-year-old female with generalized severe bone loss and clinical attachment loss, recession of the gingival margin and presence of deep periodontal pockets. Presence of local factors, and intensingly inflammation and edema of the gingival margin are evident



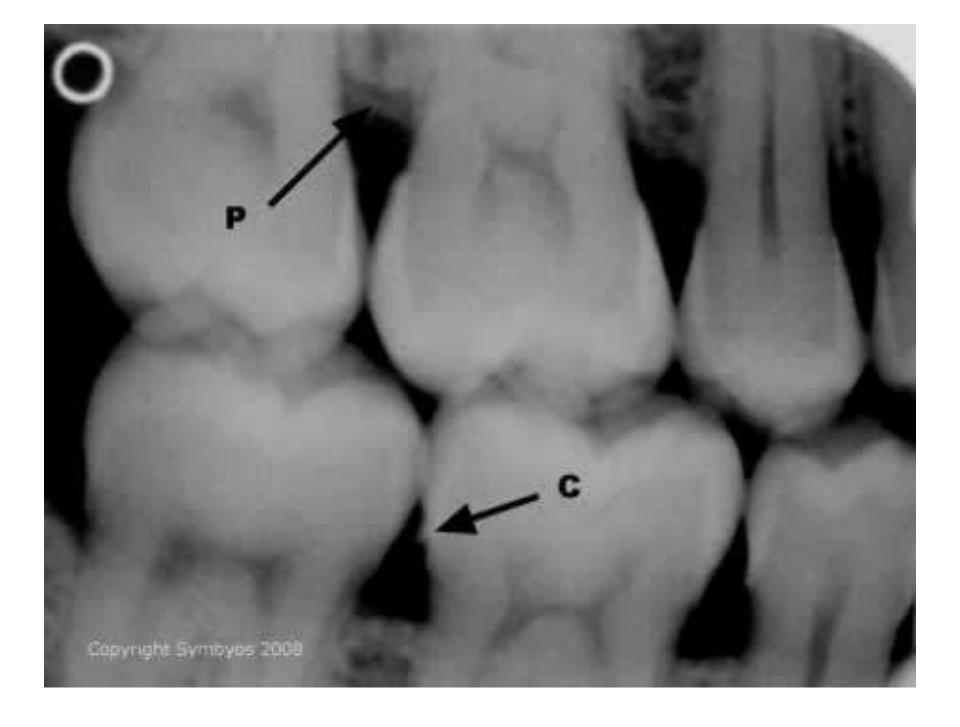
Generalized Aggressive Periodontitis



Generalized Aggressive Periodontitis

Radiographic Findings:

•The radiographic picture in generalized aggressive periodontitis can range from severe bone loss associated with the minimal number of teeth to advanced bone loss affecting the majority of teeth in the dentition



Generalized Bone Loss in a patient with GAP

Prevalence & Distribution By Age & Sex:

- •Study in Sri Lanka showed 8% of population had rapid progression of periodontal disease characterized by a yearly loss of attachment of 0.1–1.0mm
- Blacks were at much higher risk than whites
- Males were more likely to have GAP than females

Rise Factors for Aggressive Periodontitis:

- Microbiologic Factors
 - A. actinomycetemcomitans has been implicated as the primary pathogen associated with this disease

Not all reports support the association of A.a and LAP

Some studies found elevated levels of P.gingivalis, P.intermedia, Fusobacterium nucleatum, C.rectus and Treponema denticola

A.a often can be detected in periodontally healthy subjects, suggesting that this microorganism may be part of the normal flora in many individuals

2. Immunologic Factors:

- · Some immune defects have been implicated in the pathogenesis of aggressive periodontitis
- •Several investigators have shown that patients with aggressive periodontitis display functional defects of PMNs, monocytes or both

This hyperresponsive phenotype could lead to increased connective tissue or bone loss due to excessive production of these catabolic factors

Autoimmunity has been considered to have a role in GAP

3. Genetic Factors:

 Segregational analysis and linkage analysis of families with a genetic predisposition for LAP suggest that a major gene plays a role in this disease, which is transmitted through an autosomal dominant mode of inheritance

3. Environmental Factors:

- The amount and duration of smoking can influence the extent of destruction seen in young adults
- Patients with GAP who smoke have more affected teeth and more loss of clinical attachment than non-smokers with GAP

Diagnosis Of Aggressive Periodontitis:

A tentative clinical diagnosis of AgP is made based on the following criteria:

- Absence of significant systemic conditions—
 As indicated, the diagnosis of chronic, aggressive or necrotizing periodontitis implies presence of periodontal destruction in the absence of systemic diseases that may severely impair host defense.
- •Rapid attachment loss and bone destruction-

A rapid rate of destruction of the periodontium is a major criterion for the diagnosis of AgP. It is aimed at identifying subjects characterized by high virulence of the microflora and/or high levels of susceptibility.

- Familial aggregation of cases—
 Establishing the presence of familial aggregation of cases is based on a combination of history and clinical examination of family members of the affected individual
- •Lack of consistency between clinically visible bacterial deposits and severity of periodontal breakdown-
 - •It is maintained that in the majority of AgP cases the amount of periodontal destruction seems to be higher than that expected from the mere accumulation of local factors.

Treatment Modalities

- Conventional Periodontal Therapy
- Antimicrobial Therapy
- Host Modulation

Essential therapeutic considerations for the clinician are:

- To control the infection
- Arrest disease progression
- Correct anatomic defects
- Replace missing teeth
- •Help the patient maintain periodontal health
- •Educating family members as the disease is known to have familial aggregation.

Conventional Periodontal Therapy:

- •This consists of patient education, oral hygiene improvement, scaling and root planing, and regular recall maintenance.
- It may or may not include periodontal flap surgery
- •The response to conventional periodontal therapy alone has been limited and unpredictable
- •But patients who are diagnosed in the early stages may have a better outcome as compared to those who are diagnosed in advanced stage.
- •Teeth with moderate to advanced periodontal attachment loss and bone loss often have poor prognosis.

- •Some of these teeth should be extracted while some which may be pivotal to the stability of individual's dentition should be saved by treatment
- •Treatment options for teeth with deep periodontal pockets and bone loss may be
 - a) Non-surgical
 - b) Surgical.
- Surgery may be purely
 - a) Resective
 - b) Regenerative
 - c) Combination of both.

Surgical Resective Therapy:

- •It can be effective to reduce or eliminate pocket depth in patients with aggressive periodontitis.
- •However it may be difficult to accomplish if the adjacent teeth are unaffected as in case of LAP. This is because correcting the defect will often result in deep probing pocket depth around the affected tooth despite surgical efforts.
- •So its important to realize its limitations in some already mobile teeth which may become unmanageable after resective surgery.

Regenerative Therapy:

- •The use of regenerative materials including bone grafts, barrier membranes and wound healing agents are well documented and used.
- •Regenerative surgeries have been more successful in patients with chronic periodontitis and not much in aggressive periodontitis.
- •However these procedures have been successful in some of LAP patients.
- •Depending on the anatomy of the defect and teeth involved, potential for bone fill and periodontal regeneration may be poor, especially if the bone loss is horizontal and involves furcations.

• Recent advances in regenerative therapy have advocated the use of an enamel matrix protein to aid in periodontal regeneration but its success in patients with aggressive periodontitis have not been documented.

Antimicrobial Therapy:

- 1. Systemic Administration Of Antibiotics-
 - There is evidence that adjunctive antibiotic treatment frequently results in a more favorable clinical response than mechanical therapy alone.
 - Patients with deeper, progressive pockets seemed to benefit the most from systemic administration of adjunctive antibiotics.

•Many different antibiotic types and regimes were reviewed but definitive recommendations were not possible.

Systemic Tetracycline in Treatment of Aggressive Periodontitis:-

- •Systemic Tetracycline(250 mg of tetracycline hydrochloride 4 times daily for at least 1 week) should be given in conjunction of mechanical therapy
- •If surgery is indicated, systemic tetracycline should be prescribed and the patient instructed to begin taking the antibiotic approx 1 hour before surgery.

- •Doxycycline(100 mg/day) may be used instead of tetracycline.
- •Chlorhexdine rinses should be prescribed and continued for several weeks to enhance plaque control and facilitate healing.

Antibiotic Therapy for Aggressive Periodontitis

Associated Microflora	Antibiotic of choice
Gram positive organisms	Amoxicillin- clavulanate potassium (Augmentin)
Gram negative organisms	Clindamycin
Nonoral gram negative, Facultative rods Pseudomonas staphylococci	Ciprofloxacin
Black pigmented bacteria & spirochetes	Metronidazole
Prevotella intermedia, Porphyromonas gingivalis	Tetracycline
Actinobacillus actinomycetemcomitans	Metronidazole-Amoxicillin Metronidazole-Ciprofloxacin Tetracycline
P.Gingivalis	Azithromycin

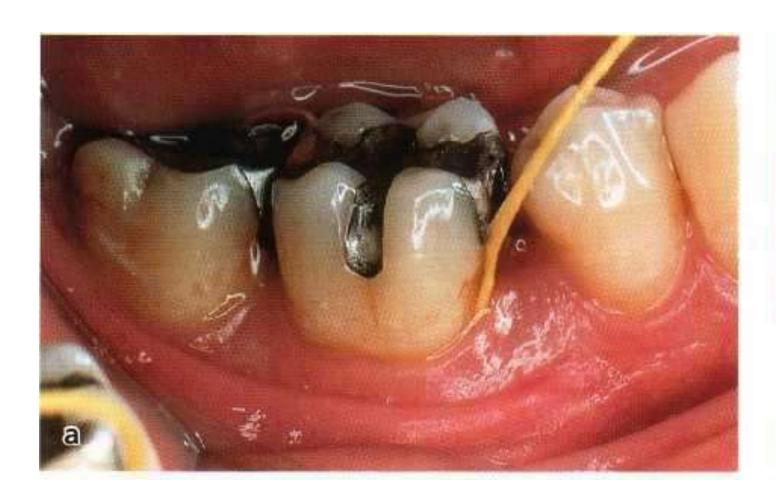
Local Delivery- The use of local delivery to administer antibiotics offers a novel approach to the management of periodontal localized infections.

- Primary advantage is that smaller dosage delivered inside pocket avoiding systemic side effects
- Increase the exposure of antibiotic to pathogen
- Many different forms have been formulated like solutions, gels, fibers and chips.



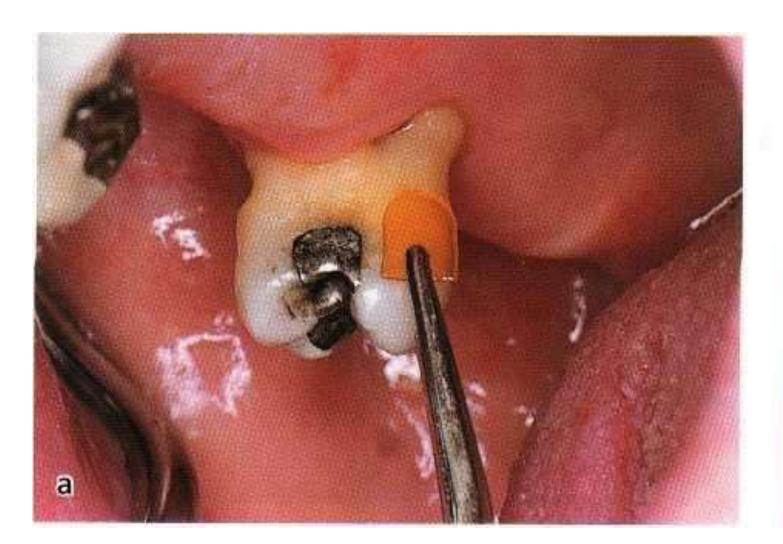


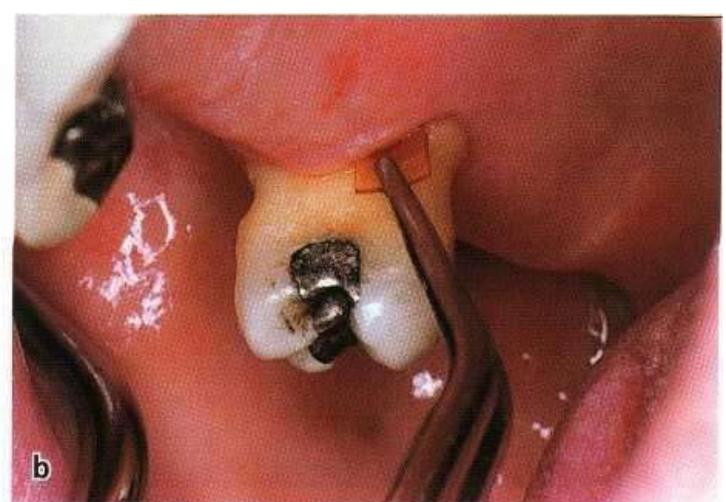
Antimicrobial Gel Application





Tetracycline fiber is packed into the periodontal pocket (a) Secured with a thin layer of cyanoacrylate adhesive (b) And left in place for 7 to 12 days.





Insertion of a chlorhexidin chip into a residual pocket

Full Mouth Disinfection:

- •This consists of full mouth debridement(removal of plaque and calculus) completed in 2 appointments within 24 hour period.
- •In addition to debridement, tongue is brushed with a chlorhexidine gel(1%) for 1 minute, the mouth is rinsed with chlorhexidine solution(0.2%) for 2 minutes and periodontal pockets are irrigated with a chlorhexidine solution(1%)

Host Modulation:

- •Several agents have been used. One such is subantimicrobial-dose doxycycline (SDD)
- •SDD may help to prevent the destruction of the periodontal attachment by controlling the activation of matrix metallo-protienases, primarily collagenases and gelatinases from neutrophiles.
- •20 mg of Doxycycline given twice daily is the subantimicrobial dosage.

- •Extensive fixed prosthesis should be avoided, and removable partial dentures should be planned in such a way so as to allow the addition of teeth.
- · When hopeless teeth are extracted, they need to be replaced

Use of Dental Implants

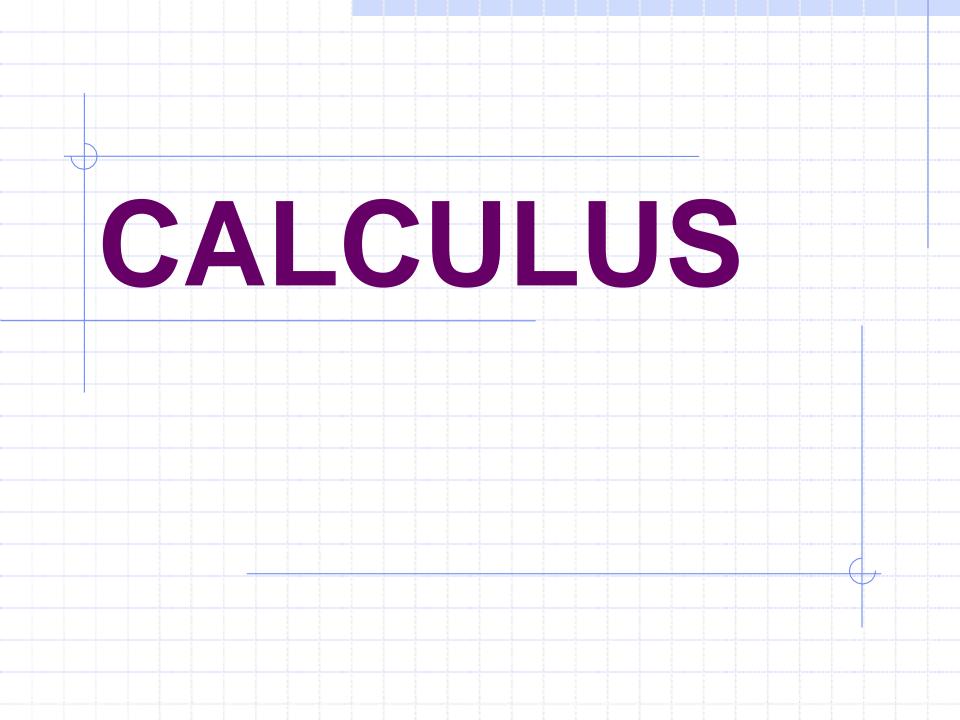
- •Dental implants can be considered for the replacement of lost teeth.
- •Initially with the use of implants there was a fear of bone loss resulting in failure of implants. But studies support the use of implants for replacement of lost teeth.

Periodontal Maintenance:

- Each maintenance visit should consist of
 - a) Medical history review
 - b) Inquiry about any recent periodontal problems,
 - c) Comprehensive periodontal and oral examination
 - d) Thorough root debridement and prophylaxis
 - e) Followed by review of oral hygiene instructions

Frequent maintenance visits appear to be one of the most important factors in the control of disease and the success of treatment in patients with Aggressive Periodontitis.

- •The duration between these recall visits is usually short during the first period after the patient's completion of therapy, generally no longer than 3 month intervals.
- •Monitoring as frequently as every 3-4 weeks may be necessary when the disease is thought to be active.
- •The rate of disease progression may be faster in younger individuals, and therefore the clinician should monitor such patients more frequently.
- •Over the time, the recall maintenance interval can be adjusted according to patient's level of oral hygiene and control of disease.



INTRODUCTION

- The primary cause of gingival inflammation is bacterial plaque, along with other predisposing factors.
- Calculus is one of the common predisposing factors.

Definitions



Calculus is defined as a deposit of inorganic salts composed primarily of calcium carbonate and phosphate mixed with food debris, bacteria and desquamated epithelial cells (Greene, 1967)

Calculus is calcified plaque. (Manson & Eley)

 Calculus consists of mineralized bacterial plaque that forms on the surfaces of natural teeth & dental prosthesis. (Carranza)

CLASSIFICATION

- According to location
- Supragingival calculus (Supramarginal, Extragingival, Coronal, Salivary)
- Subgingival calculus (Submarginal, Serumal)
- According to source of mineralization salivary calculus
 serumal calculus
- According to surface exogeneousendogeneous

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



TABLE 10.1: Differences between supragingival calculus and subgingival calculus

Supragingival calculus

- 1. Color
- 2. Shape
- 3. Consistency
- 4. Attachment
- 5. Location
- 6. Visibility
- 7. Composition

- 8. Source
- 9. Distribution

White or whitish yellow

Amorphous bulky, shape of calculus is determined by anatomy of teeth, contour of gingival margin and pressure of tongue, lips or cheeks

- Moderately hard
- Easily detached from tooth
- Coronal to the gingival margin (Fig. 10.2)
- Visible in the oral cavity
- More brushite and octacalcium phosphate.

Less Magnesium whitlockite.

Salivary proteins are present.

Sodium content is lesser.

- Derived from salivary secretions
- Symmetrical arrangement on teeth, more on facial surface of maxillary molars and lingual surface of mandibular anterior teeth due to openings of salivary glands ducts (Fig. 10.4).

Subgingival calculus

Dark brown/genuine black

Flattened to conform with pressure from the pocket wall.

May be crusty, shiny, thin, finger and fern like.

Flint like, brittle

Firmly attached to the tooth surface

Below the crest of the marginal gingiva (Fig. 10.3).

Not visible on routine clinical examination

Less brushite and octacalcium phosphate.

More Magnesium whitlockite.

Salivary proteins are absent.

Sodium content increases with the depth of pocket.

Formed from gingival exudate

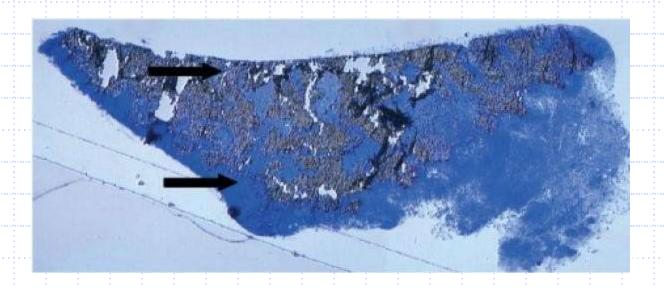
Related to pocket depth, heaviest on proximal surfaces.

Ultrastructure of calculus

Supragingival calculus contains nonmineralised areas and, by nature of its porosity may act as a reservoir for irritating substances such as endotoxins.

Light microscopy

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



Subgingival calculus

Mineralised component is uniform.

The calculus surface previously in contact with the tooth is usually flat and mineralised whilst the external/oral surface is fairly regular and covered by a non-mineralised plaque layer of variable thickness.

Transmission Electron Microscopy

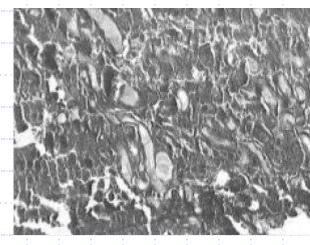
Supragingival calculus

- Contained predominantly small, randomly orientated needle-shaped/platelet-shaped crystals.
- Individual microorganisms present within the mineralised matrix showed varying degrees of calcification.
- Channels of organic matrix containing non-mineralised bacteria

Supragingival calculus





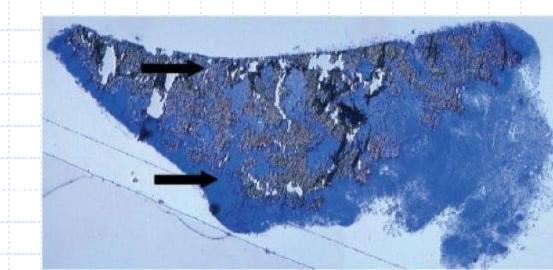


Subgingival calculus

- More homogeneous than supragingival calculus
- Mineralised bacteria were randomly arranged
- Throughout the body of the calculus there were non-mineralised areas resembling the shapes of single coccoid or rod-shaped bacterial cells.

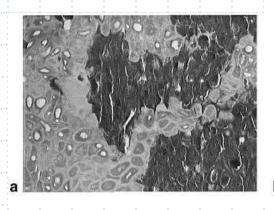
Light microscopy

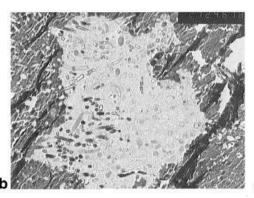
Contains many non-mineralised lacunae and, in some sections, the lacunae formed a continuous connection with the external bacterial plaque, and extended to the calculus/tooth interface

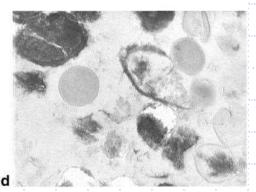


Transmission Electron Microscopy

- Channels of organic matrix containing non-mineralised bacteria were often observed. (Fig. 3a).
- Individual mineralised bacteria could also be observed within these generally non-mineralised areas (Fig. 3b and d).







x-ray beam diffraction analysis

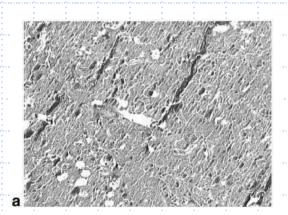
- Distribution of calcium phosphate compounds
- Octacalcium phosphate (OCP) was detected in the areas of supragingival calculus
- In the middle layer of the calculus HA was the main component.
- Supragingival calculus adjacent to the gingival and subgingival calculus contain WL.
- Bru in calculus is very rare and is found only in the supragingival calculus adjacent to the gingiva.

Sub gingival Light microscopy

The calculus surface previously in contact with the tooth was usually flat and mineralised whilst the external/oral surface was fairly regular and covered by a non-mineralised 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 (Fig.).



Scanning electron microscopy

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

x-ray beam diffraction analysis

- In subgingival calculus OCP is scarcely found.
- The main constituent is WL and HA.
- Bru is totally absent

Prevalence

Studies by Anerud and co-workers (1991)

- Periodontal status of a group of Sri Lankan tea labourers and a group of Norwegian academicians for a 15 year period were observed.
- The Norwegian population had ready access to preventive dental care throughout their lives, whereas the Sri Lankan tea labourers did not.

Composition

Inorganic Components 70-90%

Calcium phosphate (75.9%),

Ca-carbonate (3.1%) and Mg-phosphate (traces).

Principle components are:

Ca-39%, P-19%, CO₂-.9%,

Mg-0.8%

Trace elements are:

Na, Zn, Sr, Br, Cu, Mn, Au, Al, Si, Fe and F.

Organic components 20-30 %

Protein-polysaccharide complexes
Desquamated epithelial cells
Leukocytes
Micro-organisms

Crystal forms:

Hydroxyapatite – 58%

Mg-whitlockite – 21%

Octacalcium phosphate – 12%

Brushite – 9% (Mand ant)

Carbohydrates (1.9-9.1%)
Salivary Proteins (5.9-8.2%)
Lipids (0.2%)

Crystals

- Hydroxyapatite and octacalcium phosphate are detected most frequently and constitute the bulk of the specimen.
- Brushite is more common in the mandibular anterior region.
- Magnesium whitlockite is common in the posterior areas and subgingivally.

- Early calculus brushite
- Later octacalcium phosphate
- Mature calculus hydroxyapatite and whitlockite
- Exterior layers have OCP and inner layers have hydroxyapatite.

Organic content

- Carbohydrate: 1.9%
- Galactose
- Glucose
- Rhamnose
- Mannose
- Glucuronic acid
- Galactosamine

Sometimes:
arabinose
galacturonic acid
glucosamine

❖ Salivary proteins— 5.9% to 8.2% of organic component, include most amino acids.

- Lipids 0.2% of organic component, in the form of:
- Neutral fats
- Free fatty acids
- Cholesterol
- Cholesterol esters
- Phospholipids

Bacterial Content

- Percentage of Gram+ve and Gram-ve filamentous organisms is greater within calculus than in the remainder of oral cavity.
- Micro-organisms at the periphery are predominantly gram-negative rods and cocci.
- Most of the organisms within the calculus are nonviable.

Formation of calculus

- Mineralization: Soft plaque is hardened by precipitation of mineral salts which usually starts between 1st-14th day of plaque formation.
- ❖ 50% mineralization 2 days60-90% mineralization 12 days
- All plaque does not necessarily go mineralization.

Crystallization initially occurs in the intercellular matrix and the bacterial surfaces and finally within the bacteria.

Calculus formation begins with the deposition of kinetically favored precursor phases of calcium phosphate, Octacalcium phosphate and Brushite, which are gradually hydrolysed and transformed into less soluble Hydroxyapatite and magnesium whitlockite mineral phases

Rate of formation & accumulation

- Varies from person to person and in different teeth and at different times in the same person.
- 90% of all calculus occurs on the mandibular anteriors.
- Average daily increment in calculus formers is 0.10% to 0.15 % of dry weight

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







slight

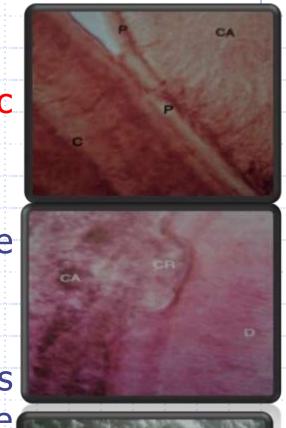
moderate

heavy

The decline from maximum accumulation (reversal phenomenon) may be explained by the vulnerability of bulky calculus to mechanical wear from food and the cheeks, lips and tongue.

Attachment mechanisms

- 1. Attachment by means of an organic pellicle on enamel.
- 2. Mechanical locking into surface irregularities.
- 3. Close adaptation of calculus undersurface depressions to the gentle sloping moulds of the unaltered cementum surface.





4. Penetration of the calculus bacteria into cementum.

5. Attachment of organic matrix of calculus into minute irregularities that were previously insertion locations of Sharpey's fibres.



Mineralization

- Two modes of mineralization :
- 1. 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.

Formation of Calculus

Attached plaque

Precipitation of mineral salts

Early plaque of heavy calculus formers contains more calcium, thrice more phosphorous and less potassium

Crystal formation intercellular matrix, bacterial surface

Calcification on inner surface

Coalescence of separate foci

Basophilic to eosinophilic

THEORIES OF MINERALISATION

Booster theory

- 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:
 - Salivary pH rise in pH causes precipitation of calcium phosphate
- 2. Colloidal proteins
- 3. Phosphatase
- 4. Esterase

Epitactic/ seeding theory

Acc to this concept, seeding agents induce small foci of calcifications, which enlarge and coalesce to form calcified mass

Inhibition theory

Another approach considers calcification as occurring only at specific sites because of the existence of an inhibiting mechanism at noncalcifying sites. The pyrophosphate inhibits calcification by preventing the initial nucleus from growing, possibly by poisoning the growth centers of the crystal.

Transformation

Amorphous noncrystalline deposits and brushite can be transformed to octocalcium phosphate and then to hydroxypatite (Eanes et al. 1970).

Bacteriological Theory

Oral microorganisms are the primary cause of calculus, and that they are invovled in its attachment to the tooth surface.

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 phospheric esters of the hexophosphoric group.

Etiological significance

- 1. Promotes retention of dental plaque.
- 2. Mechanical irritation.
- 3. Interferes with local self cleansing mechanisms.
- 4. Serve as reservoir
- Hampers oral hygiene
- 6. Periodontal pocket formation increases



Factors affecting calculus formation

- Diet and nutrition- coarse foods \ \
 - soft diet ,dietary Ca, P, Protein & Carohydrate ↑
- Age ↑ calculus with ↑age
- Habits regular oral hygiene decreases calculus formation
- Smoking- ↑ risk of supragingival calculus
- Salivary flow rate- ↑ salivary flow ↓ calculus
- Salivary Ca concentration- ↑
- High total salivary lipids- ↑
- Salivary pH- increase pH increases the calculus formation.

CRYSTAL GROWTH INHIBITORS-

- statherin is representative of salivary inhibitors of crystal growth.
- pyrophosphate and zinc ions act as crystal growth inhibitors.
- Immunoglobulins.

CALCIFICATION PROMOTERS

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.

Fluoride & calculus formation

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.

Examination

- SUPRAGINGIVAL EXAMINATION
 - direct examination
 - indirect examination with mouth mirror, with combination of retraction and light.
- SUBGINGIVAL EXAMINATION
 - -visual examination (dark edge of calculus beneath gingival margin)
 - tactile examination

CLINICAL MEASUREMENT

- two indices quantitating calculus depositscalculus component of OHIcalculus component of PI
- calculus surface index (Ennever 1961)
- marginal line calculus index (Muhlem 1967)
- Volpe manhold index (1965)
- Calculus surface severity index

CALCULUS DETECTION

Diagnodent TM Auto fluorescence Based			
DetecTar TM DetecTar TM Based Technology Spectro-Optical Technol Auto fluorescence Based		Calculus 1	Detection Only
Diagnodent TM Auto fluorescence Based	Perioscopy™		1 1
Diagnodent TM	DetecTar™		Spectro-Optical Technology
Technology	Diagnodent™		Auto fluorescence Based Technology

Calculus Detect	tion and Removal
Perioscan TM	Ultrasound technology
Keylaser3 TM	Laser Based Technology

Anticalculus agents

- 1st GENERATION
 - 1. Dissolution
- Chelating agents (EDTA, Na Hexa metaphosphate)
- Aromatic sulphuric acids (20% trichloroacetic acid)
- -Spring salts
- Alkalies
- Sodium Ricinolate

- 2. Altering Calculus Attachments
 - Silicon
 - Ion exchange resins
- 3. Plaque Inhibiton
- -Antibiotics (Triclosan 0.3 %)
- Polyvinylmethyl ether & maleic acid 2%
- -Antiseptics e.g. Chloramines

Matrix disruption

Enzymes - Mucinase, Trypsin, chymotrypsin,
 Carboxypeptidase, lipase, amylase

2nd GENERATION

- Inhibition of crystal growth
 - Vitamin C (By crystal poisoning mechanism)
 - Pyrophosphate
 - Pyrophosphate + Sodium fluoride
 - Zinc salts
 - Bisphosphonates
 - Polymers & Co Polymers

CHLORHEXIDINE

- It is second generation anti plaque agent.
- It is a cationic bisbiguanide which acts by being adsorbed onto the bacterial cell wall, leading to damage of the permeability barrier.

Cetylpyridinium Chloride(CPC)

It is a cationic quaternary ammonium compound. It was tested for anticalculus activity as a mouth rinse for 7 days but there were no differences in calculus level in CPC and controlled rinses.

VITAMIN C

- It is a surface active organophosphorus compound that has been shown to be effective in inhibiting the in vitro crystallization of calcium phosphate onto smears of supra gingival calculus.
- It has a characteristic taste which might have promoted saliva flow, thus reducing calculus levels.

Antibiotics PENICILLIN VANCOMYCIN ERYTHROMYCIN

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

CONCLUSION

Dental calculus represents mineralized bacterial plaque .It is always covered by unmineralized viable bacterial plaque ,and hence ,does not come in direct contact with the gingival tissues .Calculus ,therefore is the secondary etiological factor for periodontitis.

CHRONIC PERIODONTITIS

Introduction

It is inflammatory disease of supporting tissues of teeth caused by specific microorganism or group of specific micro-organisms resulting in progressive destruction of periodontal ligament and alveolar bone with pocket formation, recession or both.

"As an infectious disease resulting in inflammation within the supporting tissues of the teeth, progressive attachment loss and bone loss"
 (Flemmig TF 1999)

• Chronic –(Greek –Kronos means time) long lasting

• Chronic periodontitis, formerly known as "adult periodontitis" or "chronic adult periodontitis" is the most prevalent form of periodontitis.

Most commonly seen in adults.

• Age associated but not age relate.

Historical background

- "Pyorrhea Alveolaris"- Alphonse Toirac in 1823
- "Riggs Disease" John W Riggs in mid 19th century
- "Periodontitis"- Orban in 1942
- 5 types of periodontitis- Page & Schroder in 1982 & AAP in 1986
- "Chronic Periodontitis"- International workshop for classification of periodontal disease 1999.

CLASSIFICATION

1977

• Juvenile Periodontitis

 Chronic Marginal Periodontitis 1986

- Juvenile Periodontitis
- A. Prepubertal
- B. Localized juvenile periodontitis
- C. Generalized juvenile periodontitis
- Adult Periodontitis
- Necrotizing Ulcerative
- Gingivo-Periodontitis
- Refractory Periodontitis

1989

- Early-Onset Periodontitis
- A. Prepubertal periodontitis
- Localized
- Generalized
- B. Juvenile periodontitis
- Localized
- Generalized
- C. Rapidly progressive periodontitis
- Adult Periodontitis
- Necrotizing Ulcerative
- Periodontitis Refractory
- Periodontitis Periodontitis Associated with Systemic Disease

AAP 1999

- Chronic periodontitis
- ✓ Generalised
- ✓ Localised
- Aggressive periodontitis-
- ✓ Generalised
- ✓ Localised
- Periodontitis as a manifestation of systemic diseases

Clinical features

- Supra and subgingival plaque accumulation (frequently associated with calculus)
- Gingival inflammation
- Pocket formation
- Loss of periodontal attachment
- Occasional suppuration
- Poor oral hygiene –gingiva is typically may be slightly to moderately swollen 10



- Color- pale red to magenta
- Consistency soft or firm
- Surface topography loss of stippling
- Blunted or rolled gingival margin
- Flattened or cratered papillae.
- Furcation
- Tooth mobility



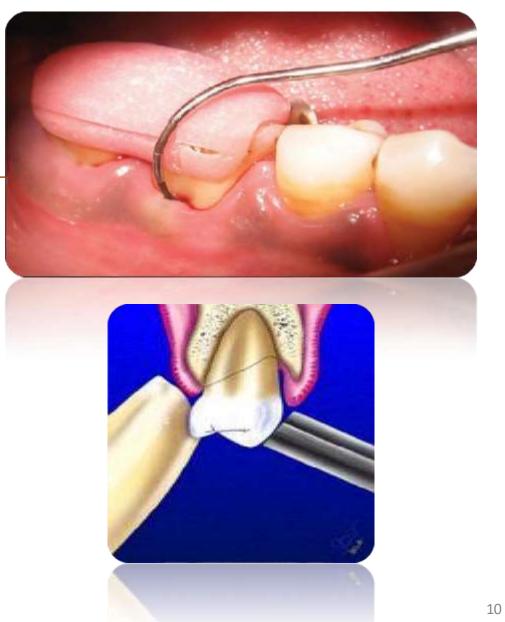
Attachment loss with and without deep PD



Pocket depths are variable, and both horizontal and vertical bone loss can be found

• Furcation involvement in the molars are common in advance cases of chronic periodontitis.

• Tooth mobility often appears in advanced cases when bone loss has been considerable.



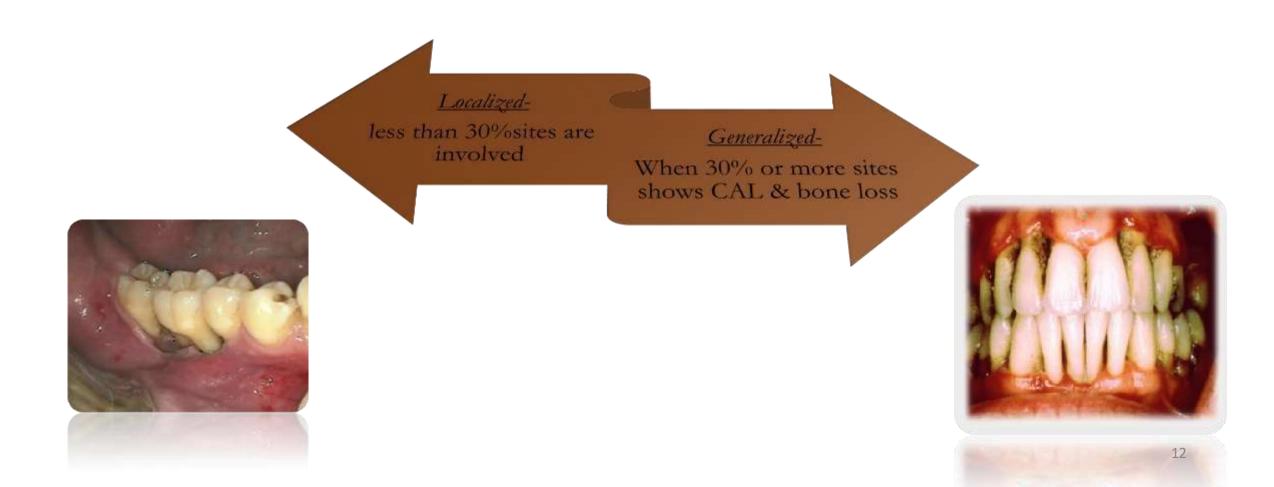
DISEASE DISTRIBUTION

• Chronic periodontitis is considered a site-specific disease.

• The clinical sign of Chronic periodontitis, namely inflammation pocket formation, attachment loss, and bone loss are considered to be due to the direct, site specific effect of subginigival plaque accumulation.

• It may occur on one surface and other may be free of symptom.

In addition to being site specific, chronic periodontitis may be described as being *localized when few* sites demonstrate attachment and bone loss or *generalized* when many sites around the mouth are affected.

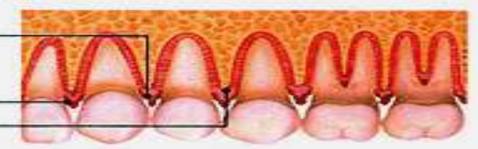


Disease Severity

- Slight periodontitis: When clinical attachment loss of 1 to 2 mm has occurred
- *Moderate periodontitis:* when clinical attachment loss of 3to 4mm has occurred
- Severe periodontitis: when clinical attachment loss >5mm

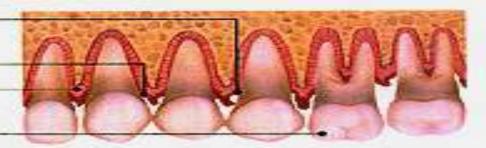
EARLY PERIODONTITIS

- Pockets form as gums separate from teeth —
- Tissue that anchors teeth to bone becomes inflamed —
- Slight bone loss -



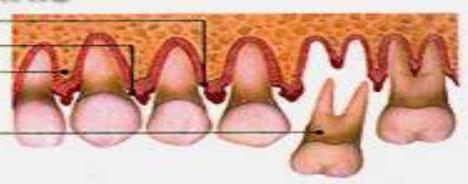
MODERATE PERIODONTITIS

- Gums may recede from teeth —
- Moderate bone loss
- Teeth may begin to separate and loosen



SEVERE PERIOD ONTITIS

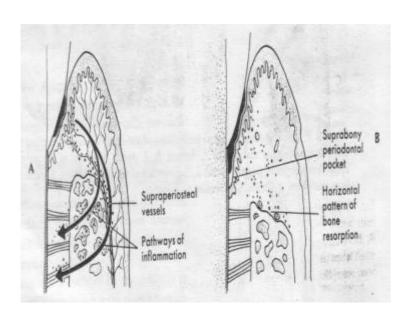
- Pockets deepen severely
- Pus may form in pockets +
- Advanced bone loss
- Teeth become very loose, and may fall out or need to be extracted



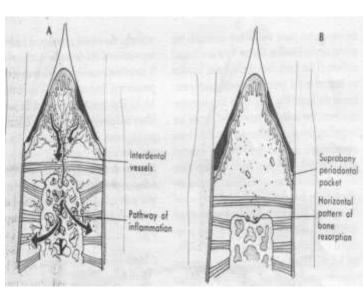
Disease Progression

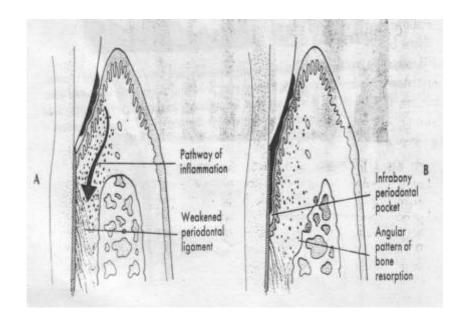
- The rate of disease progression is usually slow but may be modified by systemic and/or environmental and behavioral factors.
- Chronic periodontitis does not progress at an equal rate in all affected sites throughout the mouth.
- Rapidly progressive lesions occur most frequently in interproximal areas' and are usually associated with areas of greater plaque accumulation and inaccessibility to plaque control measures (e.g., furcation areas, overhanging margins, sites of malposed teeth, or areas of food impaction).

- Continuous model
- Random or episodic burst
- Asynchronous multiple burst model



Patterns of bone loss





RISK FACTORS FOR DISEASE

- Prior History of Periodontitis
- Local Factors
- Systemic Factors
- Environmental and Behavioral Factors
- Genetic Factors

Prior History Of Periodontitis

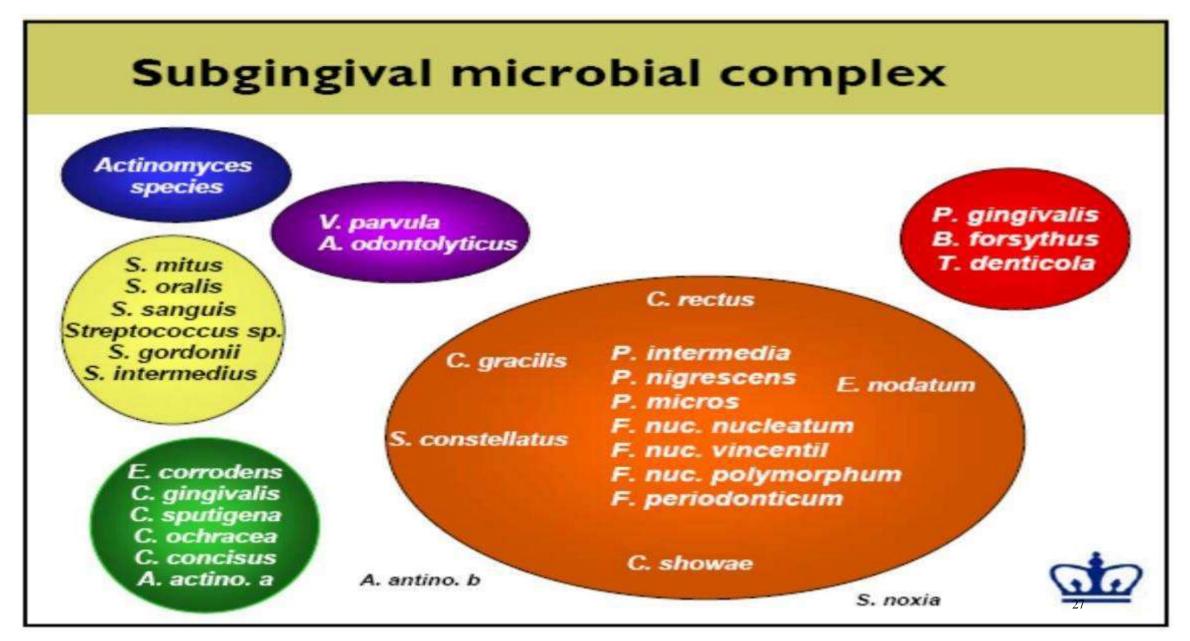
Although not a true risk factor for disease but rather a disease **predictor**, a prior history of periodontitis puts patients at greater risk for developing further loss of attachment and bone, given a challenge from bacterial plaque accumulation.

ROLE OF MICROBES

• Dental plaque is composed primarily of bacteria. One gram of plaque (wet weight)

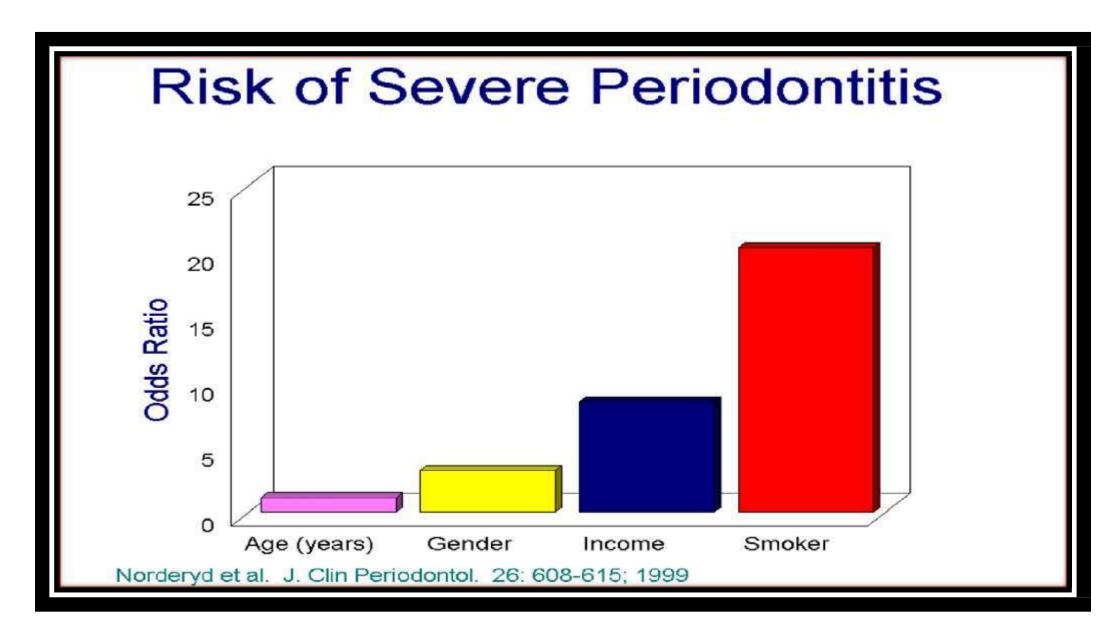
contains approximately 10¹¹ bacteria.

- In a periodontal pocket,
 - ➤ Healthy crevice 10³ bacteria.
 - ➤ Deep pocket 10⁸ bacteria.
- Nonbacterial microorganisms that are found in plaque include Mycoplasma species, yeasts, protozoa, and viruses.



Systemic and environmental risk factors

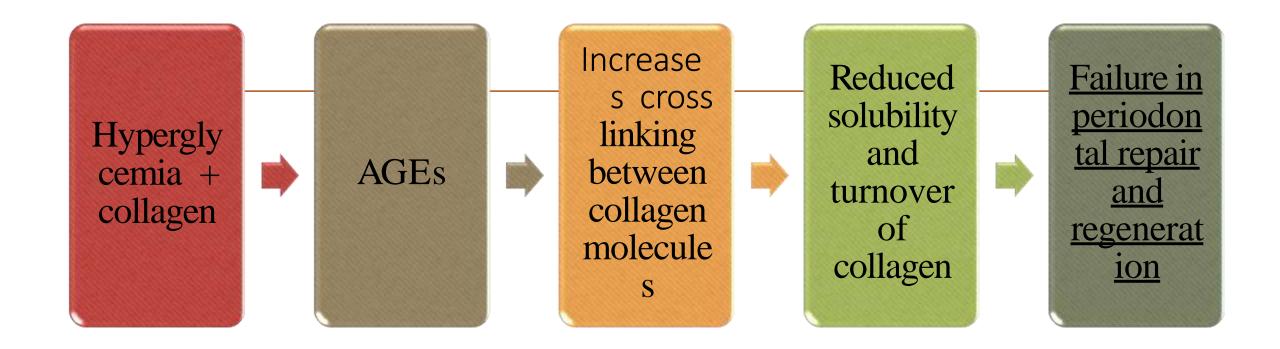
Uncontrolled diabetes mellitus (types I and II) Smoking **Emotional stress** Oral hygiene habit Environmental factor and Nutrition Osteoporosis HIV



DIABETES

- Diabetes mellitus is a disease of metabolic dysregulation.
- About 37-40million Indians have diabetes and is expected to double by 2025. India is having maximum number of diabetic patients.









- Undoubtedly one of the **main** and **most** prevalent, risk factors for chronic periodontitis, risk calculations suggesting 40% of the cases of chronic periodontitis may be attributable to smoking.
- It has been estimated that there are 1.1 billlion are smokers worldwide and 182 million (16.6%) of them live in India.

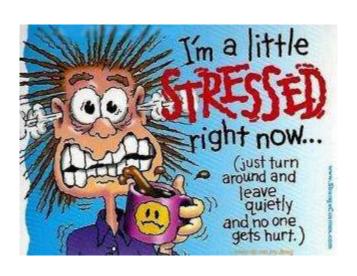
• The International Classification of Disease (ICD-10) has recognized that "Tobacco Dependence" is a disease.

• The negative effect of cigarette smoking on the Periodontium is Cumulative and Dose dependent. (Sreedhar, Shobha P 2006)

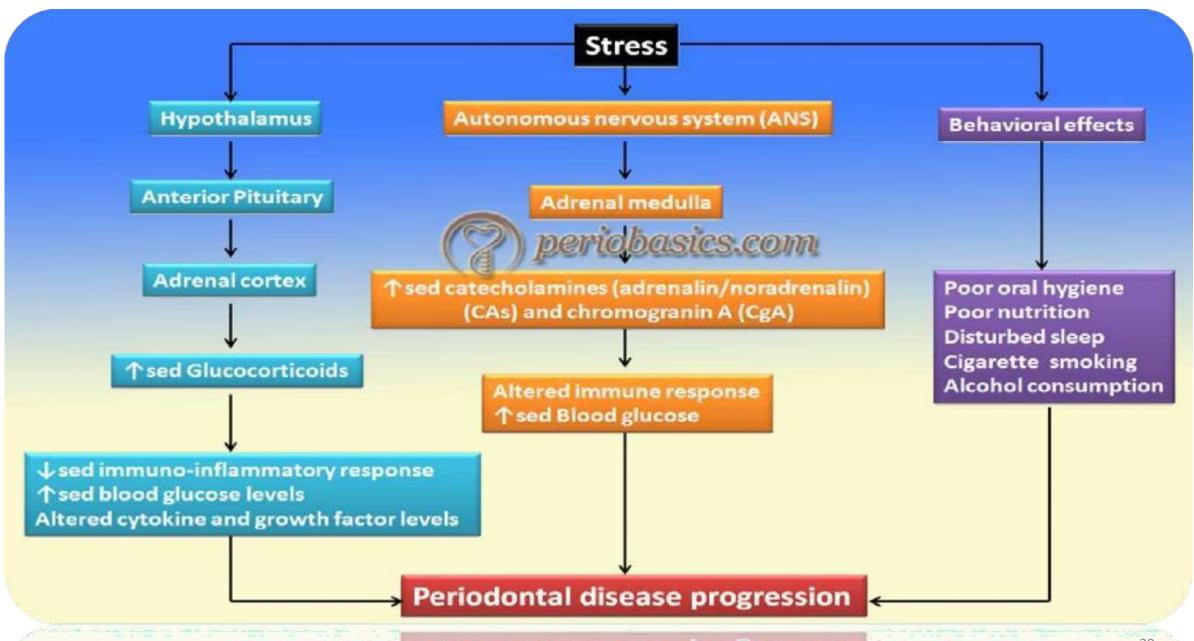
MECHANISM

Vascular alterations
Altered neutrophil function
Decreased IgG production
Decreased lymphocyte proliferation
Increased prevalence of periopathogens
Altered fibroblast attachment and function
Difficulty in eliminating pathogens by mechanical therapy
Negative local effects on cytokine and growth factor products

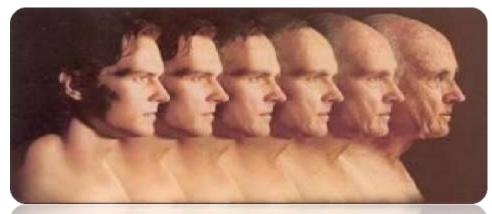
STRESS



Psychophysiological response of the organism to a perceived challenge or threat." (Breivik et al 1996)





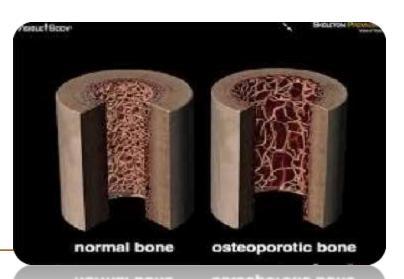


Both the prevalence and severity of periodontal disease increases with age.

(Burt 1994, Papapanou 1994, 1998).

- Lindhe (1991, 1992) —minimal loss of attachment in aging subjects enrolled in preventive programs throughout their lives.
 - ✓ Intake of medications,
 - ✓ Decreased immune function, and
 - ✓ Altered nutritional status interaction





- It is a disease characterized by low bone mass and deterioration of bone structure that causes bone fragility and increases the risk of fracture.
 - A direct association between skeletal and mandibular osteopenia and destructive periodontal disease as measured by loss of interproximal alveolar bone in postmenopausal women has been reported.

(Wactawski-Wende and coworkers 1996)

HIV

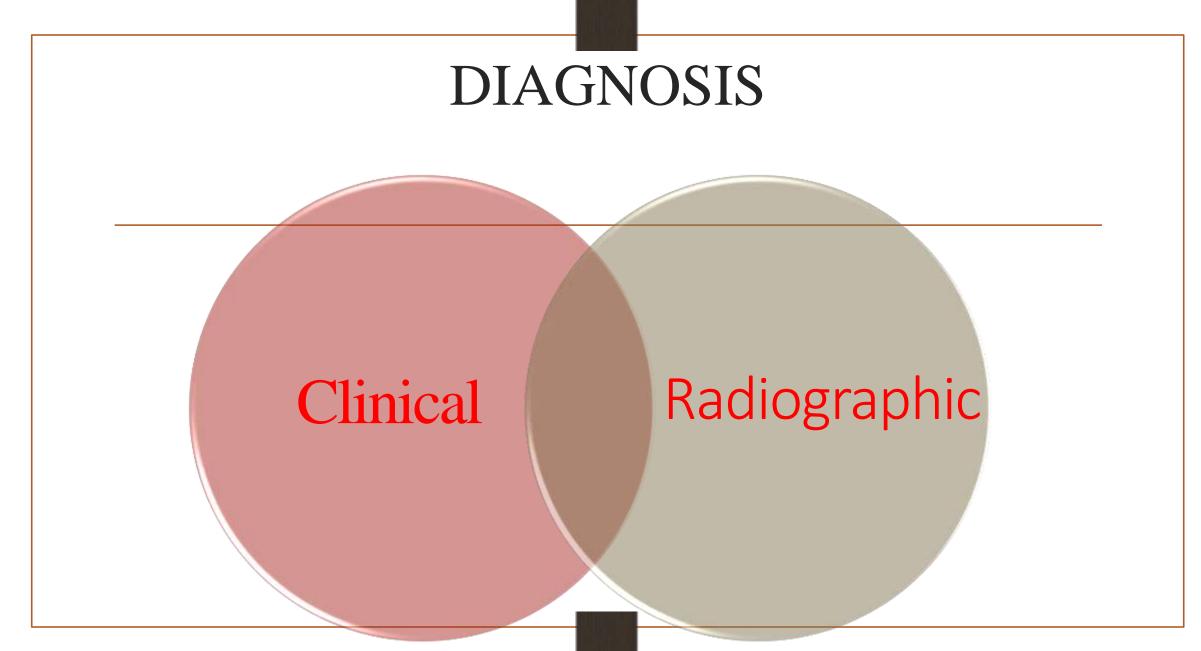
AIDS epidemics in US suggests HIV positive patients especially those with AIDS and low count of T Lymphocytes(CD4 <200 cells/ml) were at increased risk of chronic periodontitis.

Recent – HIV infection alone does not increases the risk for periodontitis.

(Smith et al 1995)

GENETICS

- Multifactorial disease....?
- Twin studies it has familial component but transmission of bacteria among family members and due to common environmental factors it is difficult to interpret.
- Polymorphism in genes encoding for IL-1alpha and beta is associated with aggressive form of chronic periodontitis in Northern America. (Korman1998)



Clinical diagnosis

• Clinical parameters, such as pocket probing depths, bleeding on probing (BOP) and suppuration (Badersten et al. 1985) or micro-biological parameters using dark-field microscopy (Listgarten & Levin 1981, Listgarten & Schifter 1982) with or without adjunctive culturing techniques (Rosling et al. 1984) as indicator tests for disease "activity".

DISEASE ACTIVITY

Consistent with the view of periodontitis as a highly localized infection of the periodontium, disease activity is perceived as the condition under which periodontal attachment loss increases abruptly at discrete sites over a relatively short period of time in a small percentage of sites (Socransky et al 1984).

How to Diagnose?

Probing pocket depth—walking of

probe

G.V.Black was first to describe systematic use of probe to explore periodontal pocket.

Periodontal probing is done on all surfaces of every tooth in the dentition. During probing, a thin periodontal probe should be used with gentle pressure and it should be "walked" around the entire circumference of each tooth.

 Increased probing depth and loss of clinical attachment are pathognomonic for periodontitis.

• Therefore, pocket probing is a crucial and mandatory procedure in diagnosing periodontitis and evaluating periodontal therapy.

• Reduction of pocket depth and gain of clinical attachment are the major clinical outcome measurements used to determine success of treatment.

Clinical attachment loss

- Clinical attachment loss is the distance from the cemento-enamel junction to the apical extent of the pocket and represents the best clinical measure of disease severity in terms of loss of support for the teeth.
- Clinical attachment level greater than 1 mm should be considered in establishment of periodontitis.
- Ramfjord et al. proposed that loss of attachment was considered the best measure of disease progression.

BLEEDING ON PROBING

- Gingival bleeding has universally been considered an indicator of gingival inflammation and by some investigation, an indicator of disease activity (Polson 1985).
- Although bleeding on probing alonemay serve as an excellent predictor for future loss of attachment.
- Lack of bleeding on probing does appear to serve as an excellent indicator of periodontal health.



SUPPURATION



- Gingival suppuration: weak predictor of active periodontal destruction, but better than bleeding.
- Suppuration upon probing is associated with probing attachment loss.(Anita Bmjersten 1985)

Journal of Clinical Periodontology 1985: 12: 432-40

SUBGINGIVAL TEMPERATURE

- Elevated temperature is one of 4 cardinal inflammatory signs.
- Subgingival temperature is thought to directly reflect the subgingival inflammatory state (Hoithius et al. 1981)
- In a study by Fedi and Killoy (1992), the temperature of pockets more than 5mm deep with bleeding on probing was 1.0°C to 1.8°C higher than that of pockets less than 3mm deep without bleeding.

MOBILITY

- Tooth mobility is a clinical expression of periodontitis.
- Many attempts have been made to develop mechanical or electronic devices for the precise measurement of tooth mobility.
- Mobility is graded clinically by holding the tooth firmly between the handles of two metallic instruments or with one metallic instrument and one.
- An effort then is made to move it in all directions. Abnormal mobility most often occurs facio-lingually.



FURCATION



• It is important to document furcation involvement because teeth with periodontal pockets in furcation have been shown to have increased loss of attachment and a poorer prognosis following periodontal therapy than teeth without furcation involvement. (McGuire MK, J Periodontol 1996)

• Furcation can be probed with Naber's probe to determine extension of pockets into areas between roots.

Pathological tooth migration

Pathological tooth migration is a characteristic sign of an advanced form of chronic periodontitis.

Microbial plaque-induced periodontal infection is considered to be the most common causative factor. Kim et al., In 2012.

He observed that no single factor is associated with PTM, but the primary factor is periodontal bone loss.



Radio graphical Diagnosis

Widening of PDL space

Loss of corticated interdental crestal margin

Localised or generalized loss of alveolar supporting bone.

Blunting of the alveolar crest due to beginning of bone resorption

Bone loss may be either horizontal or vertical.







• Numerous cross sectional and longitudinal epidemiologic studies have used radiographs as the principal method of determining the presence or absence of periodontal destruction.

• The primary criterion for bone loss in these studies was the distance from the cementoenamel junction (CEJ) to the alveolar crest, The threshold distance of bone loss has varied from 1 mm to 3 mm, although most of the studies have used > 2 mm as the criterion for bone loss.

PROGNOSIS

- Slight-to-moderate periodontitis, the prognosis is generally good, provided the inflammation can be controlled through good oral hygiene and the removal of local plaque-retentive factors.
- In patients with more severe periodontitis, as evidenced by furcation involvement and increasing clinical mobility, or in patients who are noncompliant with oral hygiene practices, the prognosis may be downgraded to fair to poor.

TREATMENT PLANNING

Treatment for periodontitis generally falls into two categories:

1) Procedures designed to halt the progression of disease.

2)Procedures designed to regenerate structures destroyed by disease.

0

Non surgical

Surgical

• <u>Successful periodontal therapy is dependent on anti-</u> infective procedures aimed at eliminating pathogenic organisms found in dental plaque associated with the tooth surface and within other niches in the oral cavity.

• Since periodontal disease is a plaque-induced infection and most patients are not skilled in mechanical plaque removal, professional cleaning is almost universally indicated to sustain long-term stability of the periodontium.

• Anti-infective therapy includes both mechanical and chemotherapeutic approaches to minimize or eliminate microbial biofilm (bacterial plaque), the primary etiology of gingivitis and periodontitis..

MECHANICAL APPROACH

• Mechanical therapy consists of debridement of the roots by the meticulous use of hand or power-driven scalers to remove plaque, endotoxin, calculus and other plaque-retentive local factors.

• The term mechanical therapy refers to both supra-gingival and sub-gingival scaling as well as root planing.

CHEMICAL APPROACH

Chemotherapeutic approaches include topical application of antiseptics or sustained-release local drug delivery agents that are designed to prevent plaque accumulation and to disinfect the root surfaces and adjacent periodontal tissues.

Rationale for adjunctive topical or systemic antimicrobial agents

Mechanical therapy alone may not effectively control infection, particularly in deep pockets.

Poor plaque control increases the rate of reinfection of the pocket

Root surface, tongue, tonsils and within other niches in the oral mucosa harbor pathogenic bacteria that recolonize the periodontal pocket and can act as sources for reinfection

Aggrebacter actinomycetemcomitans and other tissue-invasive organisms are not zeasily irradicated without concomitant antibiotic therapy

• Antimicrobial products such as mouthrinses containing essential oils, triclosan or chlorhexidine are also useful adjuncts to brushing and flossing in gingivitis and periodontitis patients and can reduce plaque accumulation and gingivitis by 0–75%.

Chemical agent	Plaque reduction	Gingivitis reduction	"References"	
Chlorhexidine	48–61%	27–67%	Grossman et al. (49) Löe & Schiott (70) Löe et al. (71)	
Essential oils	19–35%	15–37%	DePaola et al. (24) Gordon et al. (45) Lamster et al. (62)	
Triclosan	0–30%	20–75%	Cubells et al. (20) Denepitiya et al. (23) Garcia-Dodoy et al. (36) Svatun et al. (108) Svatun et al. (109)	· ·

• In disease sites that are more difficult to control, local drug delivery devices such as chlorhexidine chips (PerioChipTM) or 10% doxycycline gel (AtridoxTM) may be placed directly adjacent to the infected site.

• By placing an antibiotic or antiseptic in direct contact with the root surface, pathogenic organisms that were not accessible to mechanical removal by hand or power-driven instruments can be reduced or eliminated.

• Another new nonsurgical approach includes using a systemic subantimicrobial dose of doxycycline (Periostat) that targets tissue breakdown by blocking bacterial and host-derived enzymes associated with loss of alveolar bone and connective tissue.

• Ashley has reported in a summary of several studies that as an adjunct to either scaling or root planing or supra-gingival scaling and dental prophylaxis, subantimicrobial doses of doxycycline were shown to reduce collagenase levels in both gingival crevicular fluid and gingival biopsies.

SURGICAL THERAPY

• Nonsurgical therapy is performed prior to surgical treatment for periodontitis. Surgery is indicated where nonsurgical methods fail.

- Advantages of periodontal surgery:
- ✓ Improved visualization of the root surface
- ✓ More accurate determination of prognosis
- ✓ Improved pocket reduction or elimination
- ✓ Improved regeneration of lost periodontal structures
- ✓ An improved environment for restorative dentistry
- ✓ Improved access for oral hygiene and supportive periodontal treatment

• Pocket elimination procedures gave the greatest probing depth reduction.

• Pocket elimination was defined as gingivectomy or a flap procedure with or without osseous re-contouring.

Surgical techniques includes

Gingivectomy

Undisplaced flap

Modified Widman flap with and without osseous re-contouring

Apically positioned flap with and without osseous re-contouring

CONCLUSION

• Chronic periodontitis an infectious disease resulting in inflammation with in supporting tissues of the teeth, progressive attachment loss and bone loss". With all emerging technologies, a successful diagnosis and treatment will only be achieved through open sharing of ideas, research findings and thorough testing.

REFERENCES

Carranza. Clinical periodontology 9th, 10th and 11th edition

Outline of periodontics Manson, Eley, 4th edition.

Relationship between periodontal disease and systemic health Periodontology 2000, Vol. 25, 2001, 21–36

Periodontal risk assessment, diagnosis and treatment planning Periodontology 2000, Vol. 25, 2001, 37–58

Non-surgical periodontal therapy Periodontology 2000, Vol. 25, 2001, 77–88

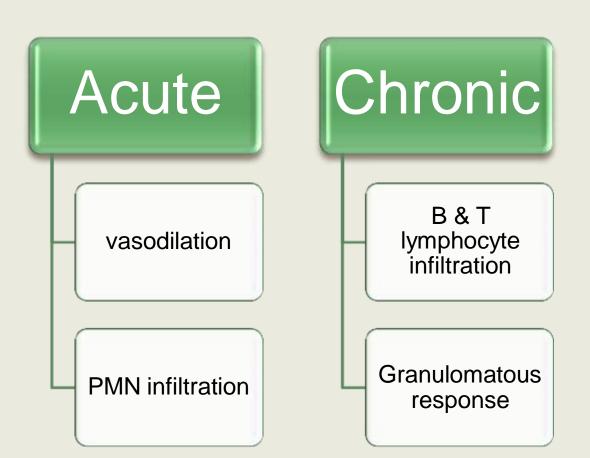
Surgical periodontal therapy Periodontology 2000, Vol. 25, 2001, 89–99

CLINICAL FEATURES OF GINGIVITIS

INTRODUCTION

Inflammation is defined as an observable alteration in tissues associated with changes in vascular permeability and dilation, often with the infiltration of leukocytes into affected tissues.

COMPONENTS OF GINGIVAL INFLAMMATION



PREVALENCE

■ More than 82% of U.S. adolescents have overt gingivitis and signs of gingival bleeding.

In children on average 6% of sites measured showed bleeding on probing.



COURSE & DURATION OF GINGIVITIS

Acute Gingivitis - Can occur with <u>sudden onset</u> and <u>short</u> duration and can be <u>painful</u>.

Recurrent gingivitis - reappears after having been eliminated by treatment or disappearing spontaneously.

Chronic gingivitis - is slow in onset and of long duration.

It is <u>painless</u>, unless complicated by acute or subacute exacerbations, and is the type most often encountered



Chronic gingivitis. The marginal and interdental gingivae are smooth, edematous, and discolored. Isolated areas of acute response are seen



Localized, diffuse, intensely red area wrt 12 and dark-pink marginal changes in remaining anterior teeth.

DISCRIPTION

- Localized gingivitis is confined to the gingiva of a single tooth or group of teeth
- 2. Generalized gingivitis involves the entire mouth.
- 3. Marginal gingivitis involves the gingival margin and may include a portion of the continuous attached gingiva.
- 4. Papillary gingivitis involves the interdental papillae and often extends into the adjacent portion of the gingival margin.
- 5. Diffuse gingivitis affects the gingival margin, the attached gingiva and the interdental papillae.

Localized marginal gingivitis is confined to one or more areas of the marginal gingiva

Localized diffuse gingivitis extends from the margin to the mucobuccal fold in a limited area

Localized papillary gingivitis is confined to one or more interdental spaces in a limited area

Generalized marginal gingivitis involves the gingival margins in relation to all the teeth. The interdental papillae are usually affected

Generalized diffuse gingivitis involves the entire gingiva.



Generalized marginal gingivitis in the upper jaw with areas of diffuse gingivitis.

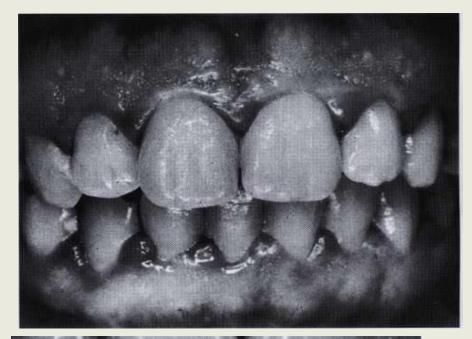


Generalized marginal and papillary gingivitis



Generalized diffuse gingivitis involves the marginal, papillary, and attached gingivae









CLINICAL FINDINGS

The two-earliest signs of gingival inflammation

(1)Bleeding from the gingival sulcus

(2)gingival crevicular fluid

BLEEDING ON PROBING

bleeding on probing is widely used to measure

- □ disease prevalence and
- progression,
- □ to measure outcomes of treatment, and
- □ to motivate patients with their home care.





Gingival bleeding on probing is an important diagnostic factor -

The presence of plaque for only 2 days can initiate gingival bleeding on probing.

The presence of bleeding - active gingival inflammation, and until not controlled - risk of continuing periodontal disease and tissue destruction.

Even though gingival bleeding on probing may not be a good diagnostic indicator for clinical attachment loss, its absence is an excellent negative predictor of future attachment loss.

Bleeding is a more <u>objective sign</u> that requires less subjective estimation by the examiner.

GINGIVAL BLEEDING CAUSED BY LOCAL FACTORS

Factors contributing to plaque retention:

- 1. Anatomic & developmental variations
- 2. Caries
- 3. Frenal pull
- 4. latrogenic factors
- 5. Malpositioned teeth
- 6. Mouth breathing
- 7. Overhangs
- 8. Recession
- 9. Partial dentures



Chronic and Recurrent Bleeding:

Provoked by mechanical trauma (e.g., from toothbrushing, toothpicks, or food impaction) or by biting into solid foods such as apples.

Vessel wall contracts, blood flow diminished

Platelets adhere to the edges

Fibrous clot formed



Histopathology

- dilation and engorgement of the capillaries
- thinning or ulceration of the sulcular epithelium.



Microscopic view of interdental space in a human autopsy specimen.Inflammatory infiltrate and thinned epithelium in area adjacent to the tooth, as well as collagenous tissue in outer half of the section.

7

ACUTE BLEEDING

Caused by:

- -Laceration of the gingiva by toothbrush bristles or by sharp pieces of hard food.
- Gingival burns from hot foods or chemicals.
- -Spontaneous bleeding or bleeding on slight provocation can occur in acute necrotizing ulcerative gingivitis.

GINGIVAL BLEEDING ASSOCIATED WITH SYSTEMIC CHANGES

Hemorrhagic disorders (Vascular abnormalities):

- Vitamin C deficiency or
- Allergy, e.g.,
 Schonlein Henoch purpura

Platelet disorders

- thrombocytopen ic purpura,
- hypoprothrombi nemia (vitamin K deficiency)

Other coagulation defects

- hemophilia,
- · leukemia,
- · Christmas disease,
- Deficient platelet thromboplastic factor (PF3) resulting from uremia

- Multiple myeloma and
 - Post rubella purpura.

The effects of **hormonal replacement therapy**, oral contraceptives, pregnancy, and the menstrual cycle are also reported to affect gingival bleeding.

Among pathologic endocrine changes, diabetes is an endocrine condition with a well-characterized effect on gingivitis.

Several medications have also been found to have adverse effects on the gingiva.

Aspirin is often prescribed for cardiovascular disease, rheumatoid arthritis, osteoarthritis, rheumatic fever, and other inflammatory joint diseases.

Anticonvulsants, antihypertensive CCBs immunosuppressant drugs are known to cause gingival enlargement, which secondary can cause gingival bleeding.

COLOUR CHANGES IN GINGIVA:

Normal gingival color- coral pink

The color of the gingiva is determined by several factors, including

- □ the number and size of blood vessels,
- □ epithelial thickness,
- quantity of keratinization,
- and pigments within the epithelium

Color Changes in Chronic Gingivitis:

The color becomes pale when vascularization is reduced (in association with fibrosis of the corium) or epithelial keratinization increases.

The gingiva becomes redish when there is increase in vascularisation. Venous stasis will add a bluish hue

The changes start in the interdental papillae and gingival margin and spread to the attached gingiva.



COLOUR CHANGES IN ACUTE DISEASES

In acute necrotizing ulcerative gingivitis, - marginal;

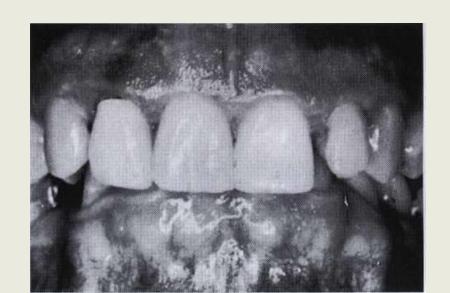
In herpetic gingivostomatitis, - diffuse; and

In acute reactions to chemical irritation, - patch-like or diffuse.

Color changes vary with the intensity of the inflammation.

METALIC PIGMENTATION

Heavy metals (bismuth, arsenic, mercury, lead and silver) absorbed systemically from therapeutic use or occupational or household environments may discolor the gingiva and other areas of the oral mucosa.



7

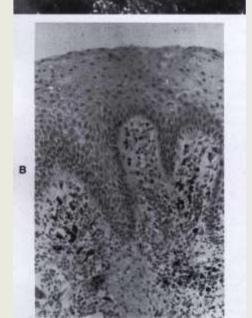
Lead - bluish red or deep blue linear pigmentation of the gingival margin(Burtonian line)

Silver - a violet line accompanied by a difuse bluish grey discoloration in throughout the oral mucosa(Argyria)



Bismuth gingivitis.linear black discoloration of the gingiva in a patient receiving bismuth therapy.







Discoloration of the gingiva caused by embedded metal particles (amalgam).

Colour changes assosiated with systemic diseases

- Addison's disease produces isolated patches of discoloration varying from bluish black to brown.
- Peutz-Jeghers syndrome melanin pigmentation in the oral mucosa and lips.
- Albright's syndrome (polyostotic fibrous dysplasia) and von RecLinghausen's disease (neurofibromatosis) produce areas of oral melanin pigmentation.
- The deposition of iron in hemochromatosis blue-gray pigmentation.
- Localized bluish black areas of pigment are often caused by amalgam implanted in the mucosa



CHANGES IN CONSISTENCY OF THE GINGIVA

Chronic gingivitis- both destructive (edematous) and reparative (fibrotic) changes coexist, and the consistency of the gingiva is determined by their relative predominance.





TABLE 22-1

Clinical and Histopathologic Changes in Gingival Consistency

Clinical Changes

Chronic Gingivitis

- 1. Soggy puffiness that pits on pressure.
- Marked softness and friability, with ready fragmentation on exploration with probe and pinpoint surface areas of redness and desquamation.

3. Firm, leathery consistency.

Acute Forms of Gingivitis

- Diffuse puffiness and softening.
- Sloughing with grayish, flakelike particles of debris adhering to eroded surface.
- 3. Vesicle formation.

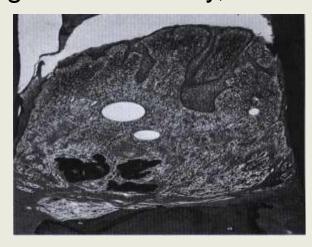
Underlying Microscopic Features

- 1. Infiltration by fluid and cells of inflammatory exudate.
- 2. Degeneration of connective tissue and epithelium associated with injurious substances that provoke the inflammation and inflammatory exudate; change in connective tissue–epithelium relationship, with inflamed, engorged connective tissue expanding to within a few epithelial cells of surface, thinning of epithelium and degeneration associated with edema and leukocyte invasion, separated by areas in which rete pegs are elongated to connective tissue.
- Fibrosis and epithelial proliferation associated with longstanding chronic inflammation.
- Diffuse edema of acute inflammatory origin, fatty infiltration in xanthomatosis.
- Necrosis with formation of pseudomembrane composed of bacteria, polymorphonuclear leukocytes, and degenerated epithelial cells in fibrinous meshwork.
- Intercellular and intracellular edema with degeneration of nucleus and cytoplasm and rupture of cell wall.



Calcified masses in the gingiva:

Chronic inflammation and fibrosis, and occasionally foreign body, giant cell activity, occur in relation to these masses.





- promotes keratinization of the oral epithelium,
- enhances capillary gingival circulation, and
- □ thickens alveolar bone.
- increases the proliferative activity.



CHANGES IN SURFACE TEXTURE OF THE GINGIVA



Stippling is restricted to the attached gingiva and is predominantly localized to the subpapillary area, but it extends to a variable degree into the interdental papilla. In chronic inflammation - the gingival surface is either smooth and shiny or firm and nodular, depending on whether the dominant changes are exudative or fibrotic.

Smooth surface texture is also produced by epithelial atrophy in atrophic gingivitis, and peeling of the surface occurs in chronic desquamative gingivitis.

Hyperkeratosis results in a leathery texture, and druginduced gingival overgrowth produces a nodular surface.

CHANGES IN POSITON OF GINGIVA

TRAUMATIC LESIONS

Traumatic lesions, whether chemical, physical, or thermal, are among the most common lesions in the mouth.

<u>Chemical injuries</u> - aspirin, hydrogen peroxide, silver nitrate, phenol, and endodontic materials.

Physical injuries - lip, oral, and tongue piercing, which can result in gingival recession.

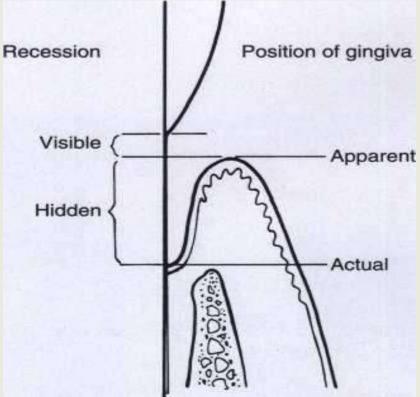
Thermal injuries - hot drinks and foods...

In <u>acute cases</u>, the appearance of slough (necrotizing epithelium), erosion, or ulceration and the accompanying erythema are common features.

In <u>chronic cases</u> - permanent gingival defects - gingival recession.

GINGIVAL RECESSION

Positions of the Gingiva: By clinical definition, *recession* is exposure of the root surface by an apical shift in the position of the gingiva.



7

ETIOLOGIC FACTORS

- Age
- Faulty toothbrushing technique (gingival abrasion)
- Tooth malposition,
- Friction from soft tissues (gingival ablation),
- Gingival inflammation,
- Abnormal frenum attachment,
- latrogenic dentistry
- Trauma from occlusion
- Orthodontic movement in a labial direction



This type of injury may present as lacerations, abrasions, keratosis and recession, with the facial marginal gingiva most affected.

Susceptibility to recession is also influenced by the

position of teeth in the arch,

the root-bone angle, and the

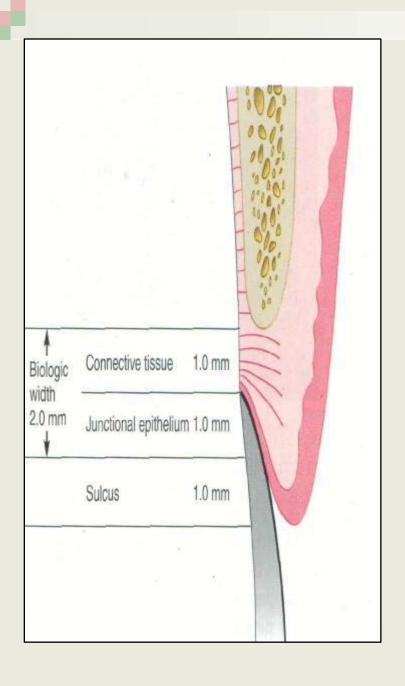
mesiodistal curvature of the tooth surface.

Restorations & recession

Pressure from a poorly designed **partial denture** can cause gingival trauma and recession.

Overhanging dental restorations - contributing factor to gingivitis because of plaque retention.

Placing restorative margins within the **biologic width** frequently leads to gingival inflammation, clinical attachment loss, and eventually, bone loss.



Rule 1: If the sulcus probes 1.5 mm or less, place the restoration margin 0.5 mm below the gingival tissue crest. This is especially important on the facial aspect and will prevent a biologic width violation in a patient who is at high risk in that regard.

Rule 2: If the sulcus probes more than 1.5 mm, place the margin half the depth of the sulcus below the tissue crest. This places the margin far enough below tissue so that it will still be covered if the patient is at higher risk of recession.

Rule 3: If a sulcus greater than 2 mm is found, especially on the facial aspect of the tooth, evaluat to see if a gingivectomy could be performed to lengthen the teeth and create a 1.5-mm sulcus. Then the patient can be treated using Rule 1.

Smoking and Recession:

The multifactorial mechanisms may include reduced gingival blood flow and altered immune response but are not, as yet, conclusive.

Clinical Significance.

Exposed root surfaces are susceptible to caries.

Abrasion or erosion of the cementum – dentinal sensitivity.

Hyperemia of the pulp and associated symptoms.

Interproximal recession creates oral hygiene problems and resulting plaque accumulation.

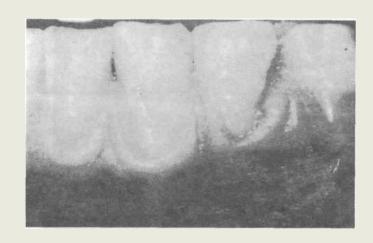
CHANGES IN GINGIVAL CONTOUR

 Changes in gingival contour are primarily associated with gingival enlargement, but such changes may also occur in other conditions.

Of historical interest are the descriptions of indentations of the gingival margin referred to as Stillman's clefts and the McCall festoons.







McCALL FESTOONS

E CHEST SATE	APPEARANCE IN HEALTH	CHANGES IN DISEASE CLINICAL APPEARANCE	CAUSES FOR CHANGES
Color	Uniformly pale pink or coral pink	Acute: bright red	Inflammation Capillary dilation Increased blood flow
	Variations in pigmentation related to complexion, race	Chronic: bluish pink, bluish red	Vessels engorged Blood flow sluggish Venous return impaired Anoxemia Increased fibrosis
		Attached gingiva: color change may extend to the mucogingival line	Deepening of pocket, mucogingival involvemen
Size	Not enlarged Fits snugly around the tooth	Enlarged	Edematous: inflammatory fluid cellular exudate vascular engorgement hemorrhage Fibrotic: new collagen fibers
Shape (contour)	Marginal gingiva: knife-edged, flat, follows a curved line about the tooth Papillae:	Marginal gingiva: rounded rolled Papillae:	Inflammatory changes: edematous or fibrous
	(1) normal contact: papilla is pointed and pyramidal; fills the interproximal area (2) space (diastema) between teeth; gingiva	bulbous flattened blunted cratered	Bulbous with gingival enlargement (see edematous and fibrotic, above) Cratered in necrotizing
	is flat or saddle shaped	and the second second	ulcerative gingivitis
Consistency	Firm Attached gingiva firmly bound down	Soft, spongy: dents readily when pressed with probe Associated with red color, smooth shiny surface, loss of stippling, bleeding on probing	Edematous: fluid between cells in connective tissue
		Firm, hard: resists probe pressure Associated with pink color, stippling, bleeding only in depth of pocket	Fibrotic: collagen fibers
Surface texture	Free gingiva: smooth Attached gingiva: stippled	Acute condition: smooth, shiny gingiva Chronic: hard, firm, with stippling, sometimes	Inflammatory changes in to connective tissue; edem cellular infiltration Fibrosis

1111111	APPEARANCE IN HEALTH	CHANGES IN DISEASE CLINICAL APPEARANCE	CAUSES FOR CHANGES
Position of Gingival Margin	Fully erupted tooth: margin is 1–2 mm above cementoenamel junction, at or slightly below the enamel contour	Enlarged gingiva: margin is higher on the tooth, above normal, pocket deepened Recession: margin is more apical; root surface is exposed	Junctional epithelium has migrated along the root; gingival margin follows
Position of Junctional Epithelium	During eruption along the enamel surface (Figure 12-7) Fully erupted tooth: the junctional epithelium is at the cemento-enamel junction	Position determined by use of probe, is on the root surface	Apical migration of the epithelium along the root
Mucogingival Junctions	Make clear demarcation between the pink, stippled, attached gingiva and the darker alveolar mucosa with smooth shiny surface	No attached gingiva: (1) Color changes may extend full height of the gingiva; mucogingival line obliterated (2) Probing reveals that the bottom of the pocket extends into the alveolar mucosa (3) Frenal pull may displace the gingival margin from the tooth	Apical migration of the junctional epithelium Attached gingiva decreases with pocket deepening Inflammation extends into alveolar mucosa
Bleeding	No spontaneous bleeding or upon probing	Spontaneous bleeding Bleeding on probing: bleeding near margin in acute condition; bleeding deep in pocket in chronic condition	Degeneration of the sukular epithelium with the formation of pocket epithelium Blood vessels engorged Tissue edematous
Exudate	No exudate expressed on pressure	White fluid, pus, visible on digital pressure Amount not related to pocket depth	Inflammation in the connective tissue Excessive accumulation of white blood cells with serum and tissue makes up the exudate (pus)

PERIODONTAL FLAP

INTRODUCTION

The successful periodontal therapy is based on total elimination of plaque, calculus and diseased cementum from the tooth surface. Whenever there is a pocket present, the plaque control becomes difficult and also deeper the pocket the more difficult the access creating problems.

Since long, flap is considered the basic surgical procedure of periodontal therapy. It not only provides access to underlying tissues, but also permits the surgeon to perform variety of regenerative procedures.

DEFINITIONS

A periodontal flap is a section of gingiva and/or mucosa surgically separated from the underlying tissues to provide visibility of and access to the bone and root surface (Carranza).

In general, flap procedures are indicated in cases of periodontitis with active (inflamed) pockets over 5mm deep, which do not <u>respond</u> satisfactorily to initial closed therapy.

Crown lengthening procedures

Infrabony pockets, defects.

Implants and osseous transplants into infrabony pockets.

Tooth extraction with immediate PDL t/t of adjacent PDL structures.

INDICATIONS

Guided tissue regeneration

Hemisection of a tooth, or root resection.

Pronounced thickening of the marginal bone.

Pronounced

enlargement/overgrowth , which can be treated more efficiently by means of GV/GP.

CONTRAINDICATIONS

Exposure of tooth preparations and restoration margins, usually better achieved by GV(exception: crown lengthening procedures).

ADVANTAGES

Pocket epithelium is removed by the inverse bevel incision

Optimal subgingival SRP to the base of the pocket can be performed with direct vision.

At the end of the surgery, the flaps can be replaced back, or repositioned apically, coronally or laterally.

The interdental bone or infrabony defects can be covered by the soft tissue flaps

No open wound persists post-operatively

DISADVANTAGES

With apical flap repositioning and due to shrinkage of the gingiva:

Coronal segments of the root surface are exposed, and this may be associated with

Poor esthetics,

Sensitive tooth necks

The risk of root surface caries.

CLASSIFICATION OF FLAPS

BASED ON BONE EXPOSURE AFTER FLAP REFLECTION

(Carranza, 1979):

Full thickness flap

Partial thickness flap

Combination Flap



Non-Displaced Flaps

Displaced Flaps

BASED ON MANAGEMENT OF PAPILLA

Conventional flap

Papilla Preservation flap

BASED ON THE MAIN PURPOSE OF THE PROCEDURE(Ramfjord, 1979)

Pocket
Elimination
Flap

Reattachment Flap Surgery Mucogingival Repair

DESIGN OF THE FLAP

The degree of access to the underlying bone and root surface necessary

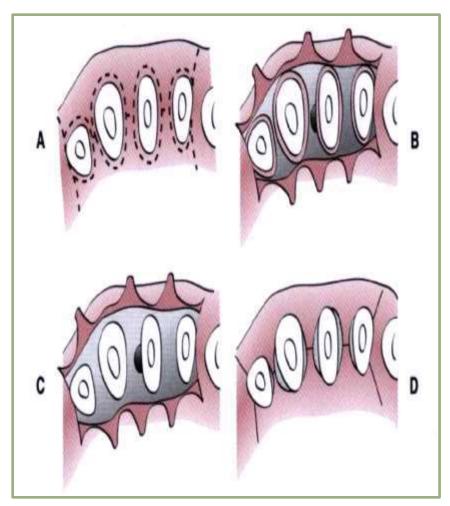
The final position of the flap

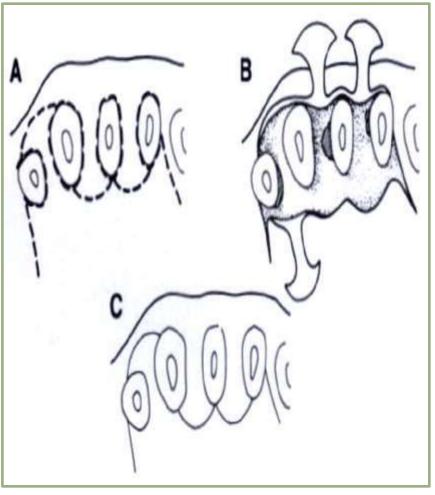
The following must be considered:

Width of attached gingiva

Preservation of good blood supply to the flap is very important

TWO BASIC DESIGNS:

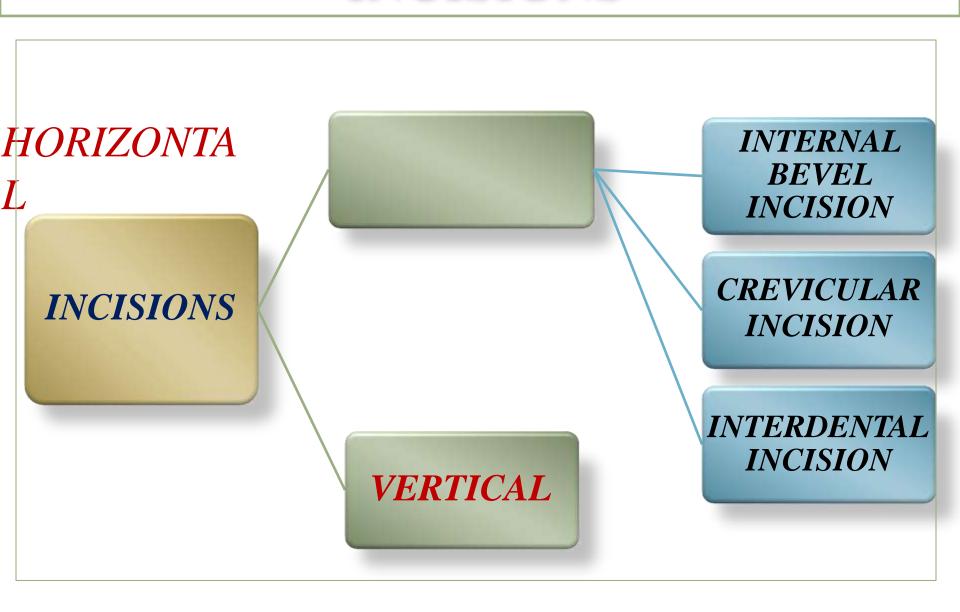




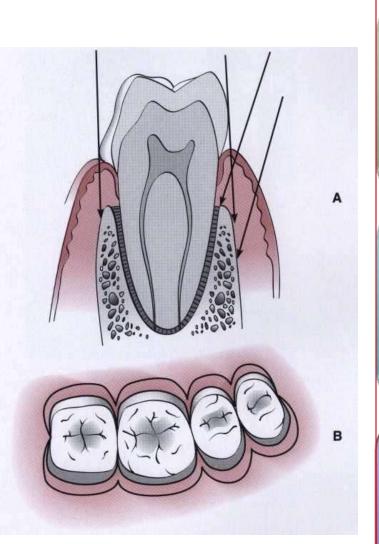
CONVENTIONAL FLAP

PAPILLA PRESERVATION FLAP

INCISIONS



INTERNAL BEVEL INCISION



It starts at a distance from the gingival margin and is aimed at the bone crest.

This incision has also been termed the first incision because it is the initial incision in the reflection of a periodontal flap.

Its called the reverse bevel incision, because its bevel is in reverse direction from that of the gingivectomy incision.

INDICATIONS

Primary incision of flap surgery if there is a sufficient band of attached gingiva

Desire to correct bone morphology (osteoplasty, osseous resection)

Thick gingiva (such as palatal gingva)

Deep periodontal pockets and bone defects

Desire to lengthen clinical crown

CREVICULAR INCISION

The crevicular incision, also termed the second incision, is made from the base of the pocket to the crest of the bone, with a #12 D blade.



INDICATION S

Narrow band of attached gingiva

Thin gingiva and alveolar mucosa

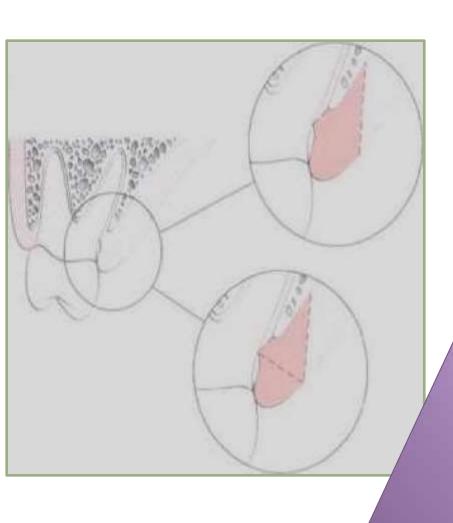
Shallow periodontal pocket

To \post-op gingival recession for esthetics in the max. ant. region.

As a second incision for flap surgery

Bone graft or GTR

INTERDENTAL INCISION



The 3rd or interdental incision separates the collar of gingiva that is left around the tooth.

The Orban knife is usually used for this incision.

VERTICAL INCISIONS

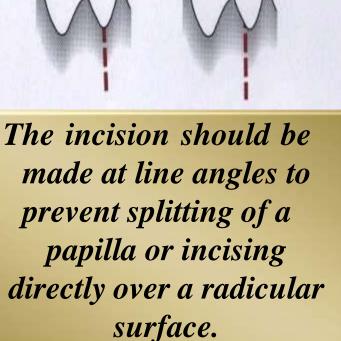
Vertical or oblique

• incisions can be used on one or both ends of the horizontal incision.

Incorrect

Vertical incisions must extend beyond the MGJ.

In general, vertical incisions in the lingual and palatal areas are avoided.



Correct

PERIOSTEAL RELEASING INCISIONS

These are used when coronal or lateral advancement of a flap onto the root or crown of the tooth is indicated.

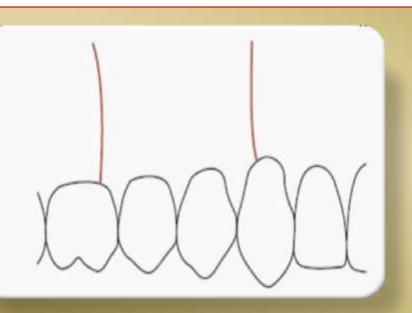
DIFFERENT TYPES OF FLAP TECHNIQUES

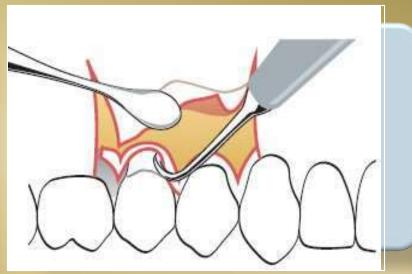
THE ORIGINAL WIDMAN FLAP

In 1918 Leonard Widman, described a mucoperiosteal flap aimed at removing the pocket epithelium and the inflamed C.T, thus facilitating optimal cleaning of the roots.

ADVANTAGES OVER GINGIVECTO-MY Less
Discomfort
for the
patient since
healing
occurred by
primary
intention.

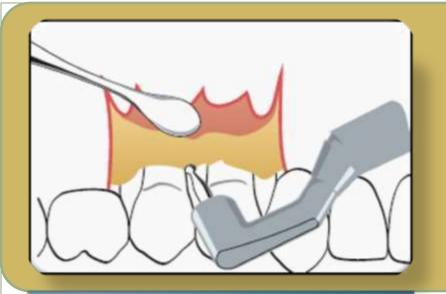
The possibility to re-establish a proper contour of alveolar bone in sites with angular bony defects.

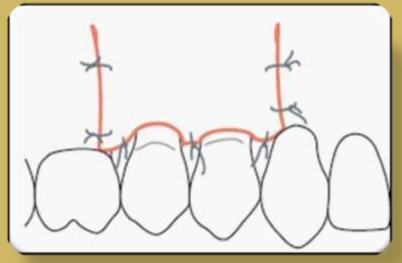




Sectional releasing incisions were first made to demarcate the area scheduled for surgery.

These were connected by a gingival incision, that separated the pocket epithelium and the inflamed connective tissue from the non-inflamed gingiva.



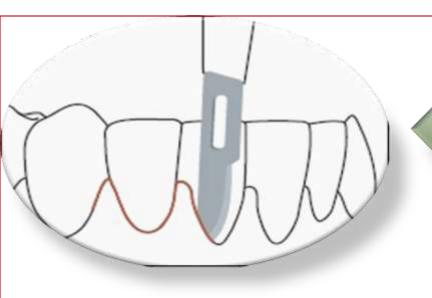


A mucoperiosteal flap was elevated to expose 2-3 mm of the marginal alveolar bone. After debridement, bone recontouring was done in order to achieve an ideal anatomic form of the underlying alveolar bone.

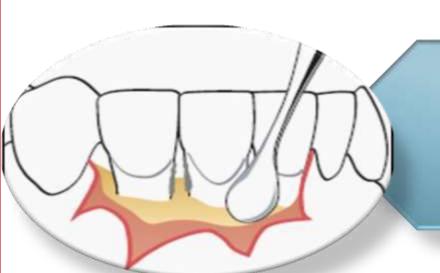
The flaps were laid back over the alveolar bone, such that the soft tissue margin was at the level of the alveolar bone crest, so that no pockets would remain.

MODIFIED FLAP OPERATION

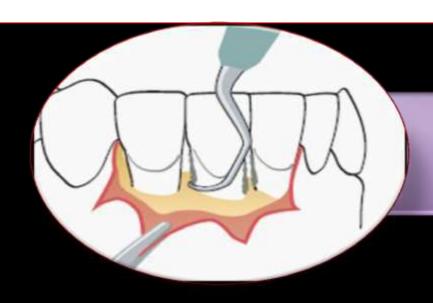
In 1931 Kirkland described a procedure for t/t of 'periodontal pus pockets'. The procedure was called the modified flap operation, and is basically an access flap for proper root debridement.



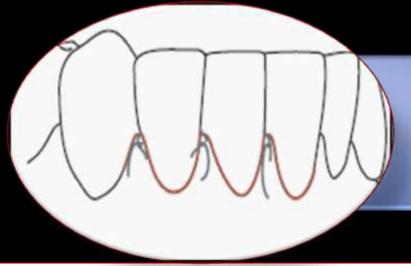
Intracrevicular incisions



The gingiva was retracted labially and lingually to expose the diseased root surfaces



Root surfaces were carefully debrided. Angular bony defects were curetted.



Flaps were replaced to their original position and secured by interproximal sutures.

There was not extensive sacrifice of non inflamed tissues

Apical
displacement
of gingival
margin was
not done

ADVANTAGES

Since roots
were not
markedly
exposed the
method could
be esthetic,
hence used in
anteriors.

The potential for bone regeneration in intrabony defects.

Apically Position ed Flap

Fullthickness Splitthicknes

If pocket reaches/ extends beyond the MGJ Gingiva is extreme ly thin

In case of osseous defects.

Thickness of marginal alveolar bone

Amt. of pocket to be eliminate

Clinical crown length reqd. for restorative/ prostho t/t & esthetics

Width and thickness of gingiva

POSITION
OF THE
FLAP
DEPENDS
ON:

Length of root trunk

MODIFIED WIDMAN FLAP

In 1965, Morris revived a technique and called it the unrepositioned mucoperiosteal flap.

Essentially the same procedure was presented in 1974 by Ramfjord and Nissle who called it the Modified Widman flap.

The modified widman flap is used for exposing the root surfaces for instrumentation and debridment and for removal of pocket lining. It is not intended to eliminate or reduce pocket depth, though the reduction does occur due to tissue shrinkage following healing.

Mild to moderate periodontitis, with pocket depths between 4-6mm. Can also be used in areas with deeper interdental defects.

Depending upon the pathology in an area, it can be combined with larger fully reflected flaps.

Less frequently,
it maybe
combined with
gingivectomy &
special
operations (e.g.
wedge excisions,
root resection,
hemisection etc

INDICATIONS

CONTRAINDICATIONS

(Practically none)

Very narrow attached gingival can render this technique difficult (mand. anteriors).

Also difficult in cases where the osseous recontouring has been planned for extremely deep defects and an irregular pattern of bone loss.

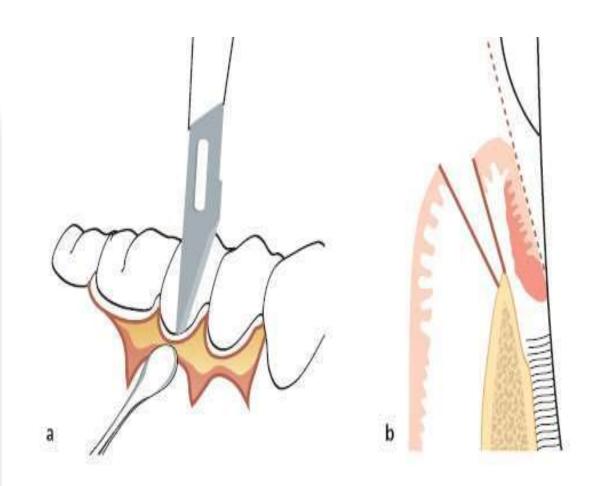
If the bony crest is particularly protuberant in some areas, this technique is difficult to perform.

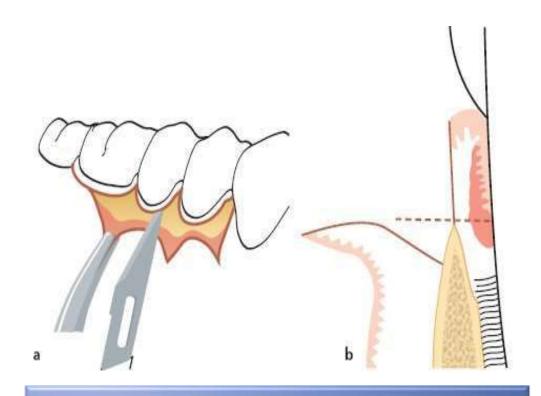


The initial incision should be parallel to the long axis of the tooth and placed approximately 1 mm from the buccal gingival margin in order to properly separate the pocket epithelium from the flap.

Buccal and palatal full-thickness flaps are carefully elevated.

An intracrevicular incision is made.





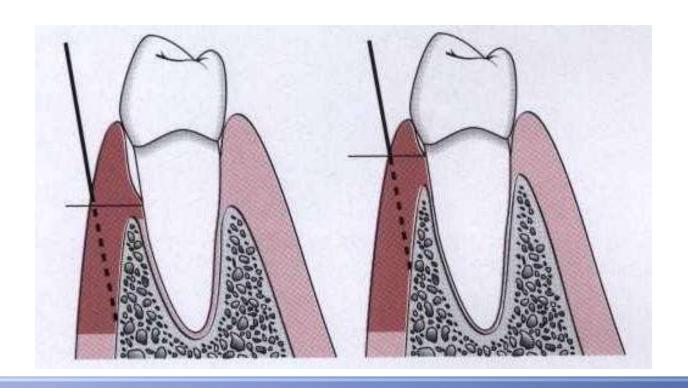
A third incision is made in a horizontal direction. This separates the soft tissue collar.

The pocket epithelium and the granulation tissues are removed by curettes. The exposed roots are carefully scaled and planed. Angular bony defects are carefully curetted.

The flaps are then trimmed and adjusted to the alveolar bone to obtain complete coverage of the interproximal bone. (Bone architecture is not corrected except if it prevents good tissue adaptation to the necks of the teeth.)

The flaps are sutured together with individual interproximal sutures.

THE UNDISPLACED FLAP



It differs from the modified Widman in that soft tissue pocket wall is removed with an initial incision, thus it may be considered an internal bevel gingivectomy.

Pockets are measured with PDL probe & bleeding points are produced to mark bottom of the pocket.



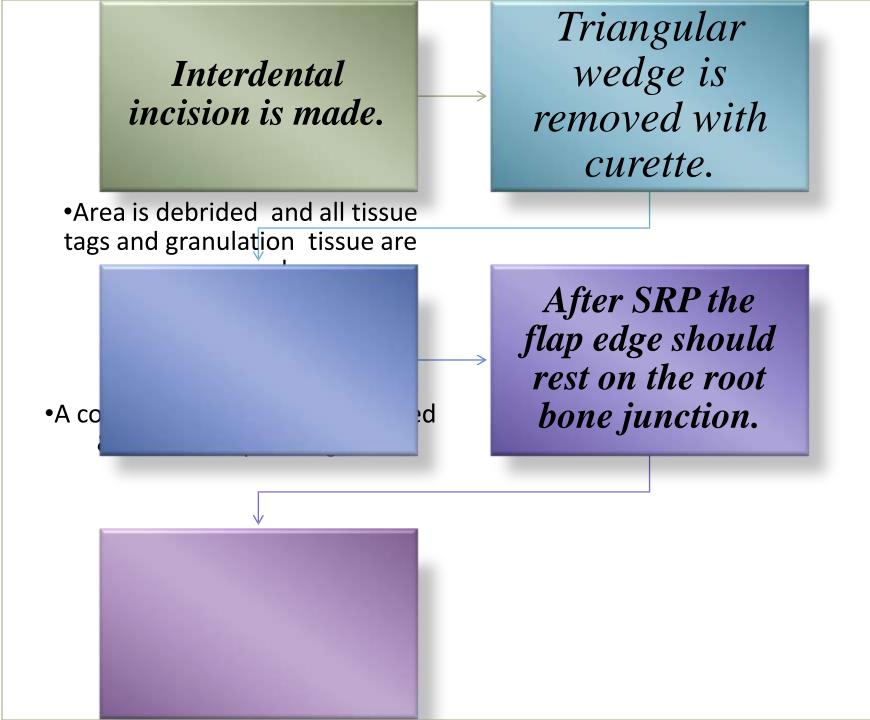
The initial, internal bevel incision is made after the scalloping of the bleeding marks on the gingiva.



Flap is reflected with a periosteal elevator (blunt dissection) from the internal bevel incision.



Crevicular incision is made.



PALATAL FLAPS

- The surgical approach to the palatal area differs from that for other areas.
- The palatal tissue is all attached, keratinized tissue and has none of the elastic properties associated with other gingival tissues.
- Therefore the palatal tissue cannot be apically displaced, nor can a partial (split) thickness flap be accomplished.
- Reduction of periodontal pocket in a thick gingival wall in the palatal aspect is uncommon because of minimal gingival shrinkage achieved by initial therapy.

FOLLOWING ARE CONSIDERED FOR THIS FLAP:

For debridement, the internal bevel incision is planned so that the flap adapts at the root-bone junction when sutured.

For osseous resection- the incision should compensate for the lowered level of the bone when the flap is closed.

The apical portion of the scalloping should be narrower than the line angle area.

Flaps should be thin to allow adaptation. This is done before complete reflection.

For vertical incisions- care is taken to avoid the numerous vessels located in the palate.

FLAPS FOR REGENERATIVE SURGERY Papilla Preservation

The flap design should allow maximum amount of gingival tissue & papilla to be retained to cover the material(s) placed in the pocket.

Flap

Conventional Flap with only crevicular incisions.

CONVENTIONAL FLAP FOR RECENTER ADVE **SURGERY** maintaining it as thick as possible.

Using a #12 blade, incise the tissue at the bottom of the pocket and to the crest of the bone, splitting the papilla below the contact point.

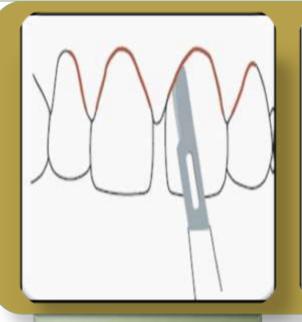
The maintenance of a thick flap is necessary to prevent exposure of the graft or the membrane due to necrosis of the flap margins

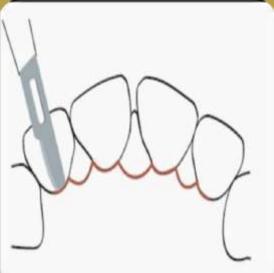
PAPILLA PRESERVATION FLAP (PPF)

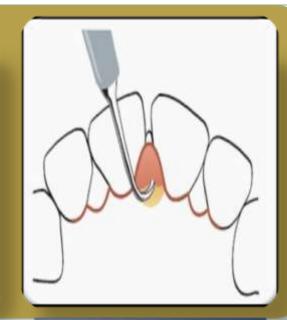
Takei et al. (1985) proposed this technique.

There must be adequate interdental space to allow the intact papilla to be reflected with facial and lingual flap.

For esthetic reasons, the papilla preservation technique is often utilized in the surgical treatment of anterior tooth regions.

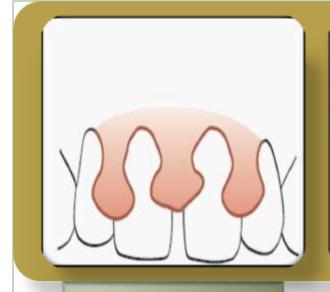




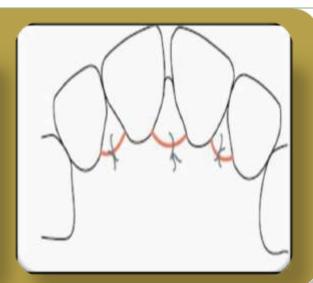


An intrasulcular incision is given without making incisions through the interdental papillae. Then an intrasulcular
incision is
made on the
lingual/palatal
side with a
semilunar
incision made
across each
interdental
area.

A curette or interproximal knife is used to free the interdental papilla carefully from the underlying hard tissue.







The detached interdental tissue is pushed through the embrasure with a blunt instrument.

A fullthickness flap is reflected and debridment is done. The flaps are repositioned and sutured.

HEALING AFTER FLAP SURGERY

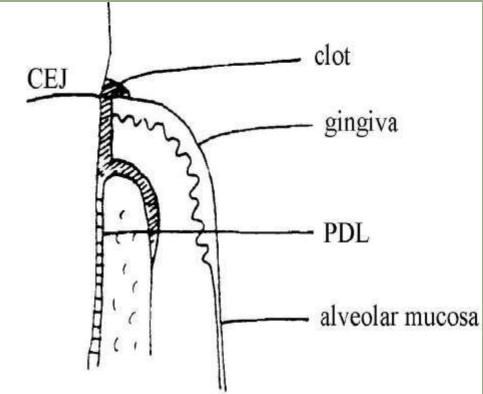
IMMEDIATELY AFTER SUTURING (0 – 24 Hrs.)

A blood clot is formed between the flap and the underlying bone. The clot contributes somewhat to maintaining the flap in position.

INITIAL HEALING (1 - 3 DAYS)

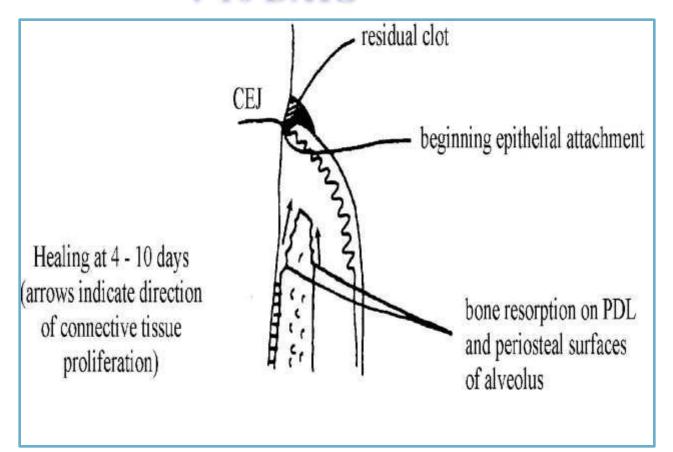
Space between flap and tooth or bone is thinner, epithelial cells migrate over the border of the flap.

Irreversible bone resorption occurs



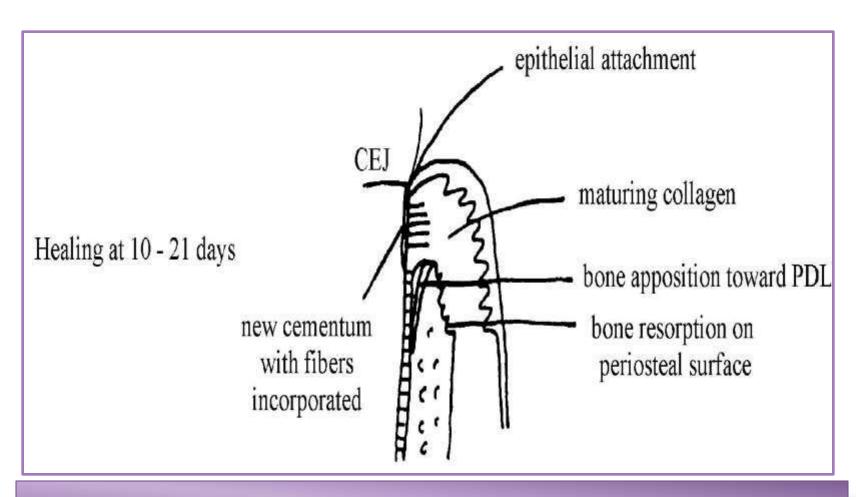
Healing at 0 - 4 days

4-10 DAYS



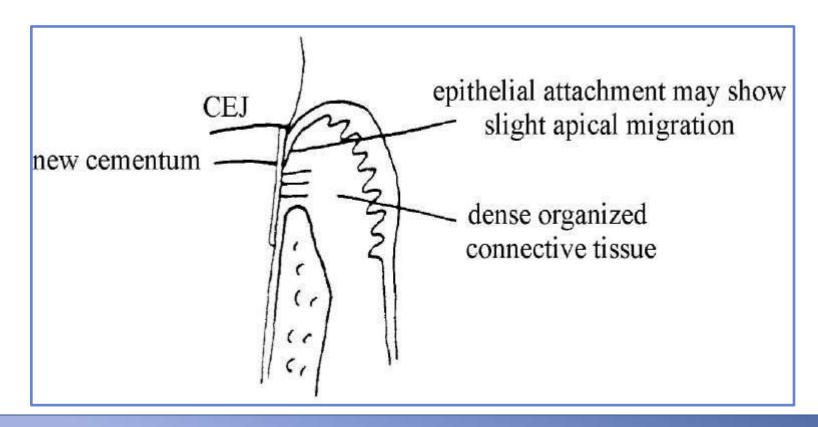
The fibrin clot starts undergoing resorption and is usually completely replaced by 6-7 days by granulation tissue.

Revascularization tends to follow the pattern of new connective tissue formation, which moves coronally as the clot is resorbed



Healing at 10-21 Days

21 DAYS



Cementum anchors periodontal fibers to the tooth. Maturation and repair of all tissues involved is occurring. Bone formation repairs most of the lost bone.

Epithelium of attached gingiva shows rete peg formation. The sulcular epithelium is thin and free from rete pegs.

ONE MONTH AFTER SURGERY

A fully epithelized gingival crevice with well defined epithelial attachment is present.

There is a beginning junctional arrangement of supracrestal fibers.

FAILURES ASSOCIATED WITH PERIODONTAL FLAP SURGERY

Improper incision

Reflection of the flap(over or under)

Incomplete Debridement

Improper Suturing

CONCLUSION

The scope of periodontal flap has increased over the past three decades from a philosophy based on access to underlying tissues to regeneration of lost tissues. Various techniques of periodontal flap surgery have come up since the introduction of flap. Choice of surgical procedure is dictated by a large number of variables and clinician should select appropriate technique for the successful outcome of therapy.



GINGIVAL INFLAMMATION

GINGIVAL INFLAMMATION

- Gingival inflammation around teeth is a general term for gingival diseases affecting gingiva.
- As generally used the term gingivitis refers to gingival inflammation induced by bacterial bio-films (plaque) adherent to tooth surfaces.





CAUSES

PLAQUE



CALCULUS



PREGNANCY

UNCONTROLLED DIABETES

ONSET OF PUBERTY

❖ ILL FITTING PROSTHESIS

Pathogenesis Behind It

Microorganisms attach to tooth in or near gingival sulcus

These Organisms capable of synthesizing Products like collagenase, hyaluronidase, Protease, endotoxins

Damage to epithelial and connective tissue cells and intercellular constituents

Widening of spaces between junctional epithelial cells

So, injurious agents gain access to connective tissue

Activate monocytes/macrophage to produce PGE 2, TNF and IL 1



SYMPTOMS

- Swollen Gums
- Mouth Sores
- Bright Red or Purple Gums
- Swollen Gums that emit pus
- Severe Malodor
- Gums That Bleed Easily Even With Gentle Brushing

Various Stages of Gingival Inflammation

- **SEarly Stage**
- **S** Established Stage
- Advanced Stage

Stage 1 Gingivitis: The Initial Stage

- Basically, Vascular Changes occur
- Dilated capillaries
- Hence, increased blood flow
- This occurs in response to microbial activation of resident leukocytes and stimulation of endothelial cells. Also known as subclinical gingivitis as initial response of gingiva to bacterial plaque is not apparent.

Microscopically,

Features of acute inflammation are seen

- Widening of small capillaries or venules
- Margination I.e. neutrophils to the vessel wall
- Margination mostly occurs within one week and sometimes as early as two days after plaque accumulation.

Diapedesis and emmigration I.e.leukocytes mainly PMNs leave capillaries by migrating through walls. Hence seen in increased quantity in connective tissue, junctional epithelium and gingival sulcus.

As these do not form an infiltrate so not considered as a pathological change.

Other changes seen are:

- Perivascular connective tissue matrix altered
- Exudation and deposition of fibrin in affected area
- Lymphocytes also accumulate
- Increase in migration of leukocytes into gingival sulcus
- Gingival fluid into the sulcus is increased.

EITHER THIS INITIAL LESION RESOLVES RAPIDLY WITH RESTORATION OF TISSUE TO NORMAL STATE

OR

EVOLVES INTO A CHRONIC INFLAMMATORY LESION

If latter occurs an infiltrate of macrophages and lymphoid cells appear within few days

STAGE 2 GINGIVITIS: THE EARLY LESION

- Evolves from initial lesion within 1 week after beginning of plaque accumulation
- Signs of erythema appear because of proliferation of capillary loops b/w rete ridges
- Bleeding on probing is evident
- Gingival fluid flow & no. of transmigrating leukocytes each max. b/w 6 - 12 days.

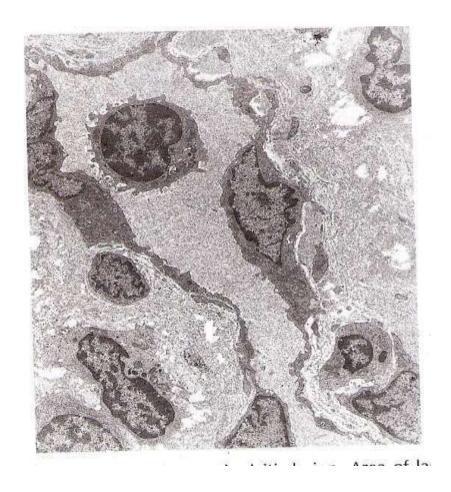


Marginal gingivitis and irregular gingival contour

MICROSCOPICALLY

- Leukocytic infiltration in connective tissue mainly of lymphocytes, neutrophils, macrophages, plasma cells, mast cells.
- All the changes seen in initial lesion intensify with the early lesion.
- J.E becomes densely infiltrated with neutrophils

- The amount of collagen destruction increases.
- The main fibre groups affected are circular and dentogingival.
- PMNs are found in epithelium emerging in pocket area.
- Fibroblasts show cytotoxic alterations with a decreased capacity for collagen production.



Area of lamnia propria subjacent to crevicular epithelium showing a capilary with several extra vascular lymphocytes and one lymphocyte within the lumen.

STAGE-III GINGIVITIS:THE ESTABLISHED LESION

CHARACTERISED BY:

- Plasma cells
- B Lymphocytes
- Gingival pocket formation
- IgG1,IgG3
- Blood vessels are engorged

- Venous return is impaired and blood flow becomes sluggish
- Extravasation of erythrocytes into connective tissue and breakdown of Hb into its component pigments, hence deepening the colour of inflamed gingiva.
- The established lesion can be described as moderately to severely inflamed gingiva.

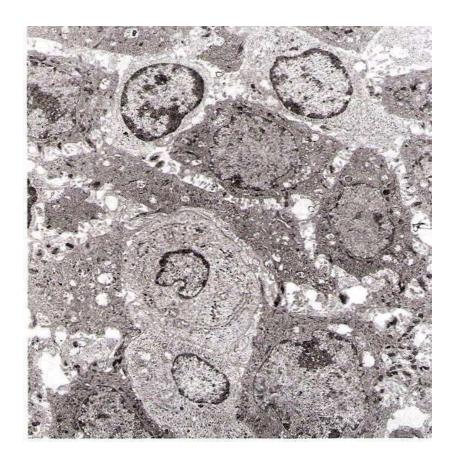


Marginal supragingival plaque and gingivitis

HISTOLOGICALLY

- An intense chronic inflammatory reaction is observed.
- Increased no. of plasma cells not only in C.T immediately below J.E but also deep into C.T
- J.E reveals widened intercellular spaces filled with granular cellular debris, including lysosomes.
- These lysosomes contain acid hydrolases that can destroy tissue components.

- J.E develops rete ridges that protrude into C.T
- Basal lamina is destroyed in some areas
- In C.T collagen fibres are destroyed.
- The predominance of plasma cells is primary characteristic of established lesion.
- Inflamed gingiva has elevated levels of acid &alkaline phosphatase, glucuronidase, esterase, aminopeptidase,
- Neutral mucopolysaccrides are decreased.



Area of crevicular epithelium ex. Enlarged intercellular spaces with microvilli and desmosomal junctions, lymphocytes are seen migrating through epithelial layer

- Established lesions of two types appear to exist:
- Some remain stable, & donot progress or months or years other seems to become more active & to convert to progessively destructive lesions.

STAGE-IV GINGIVITIS:THE ADVANCED LESION

- Extension of lesion into alveolar bone.
- o Periodontal Pocket formation
- O The most common cause of bone

destruction in periodontal disease is extension of inflammation from marginal gingiva into the supporting periodontal tissues

- The inflammatory invasion of bone surface and initial bone lose that follows mark the transition from gingivitis to periodontitis
- Periodontitis is always preceded by gingivitis, but not all gingivitis progresses to periodontitis
- The transition from gingivitis to periodontitis is associated with changes in composition of bacterial plaque



Figure 2. Advanced Chronic Periodontitis

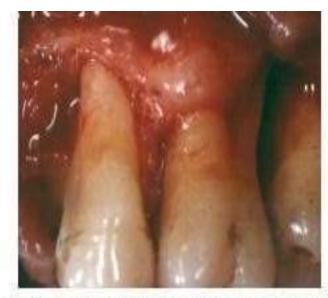
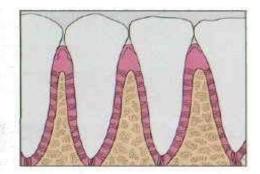


Figure 3. Severely damaged alveolar bone

1. Normal, Healthy Gingiva (Gums)
Healthy gums and bone anchor
teeth firmly in place.

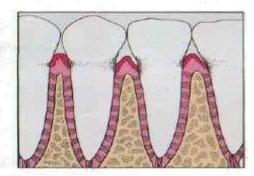




2. Gingivitis

Plaque and its byproducts irritate the gums, making them tender, inflamed, and likely to bleed.

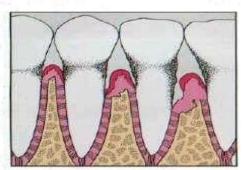




3. Periodontitis

Unremoved, plaque hardens into calculus (tartar). As plaque and calculus continue to build up, the gums begin to recede (pull away) from the teeth, and pockets form between the teeth and gums.

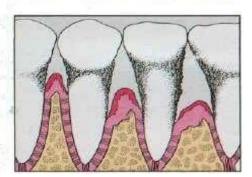




4. Advanced Periodontitis

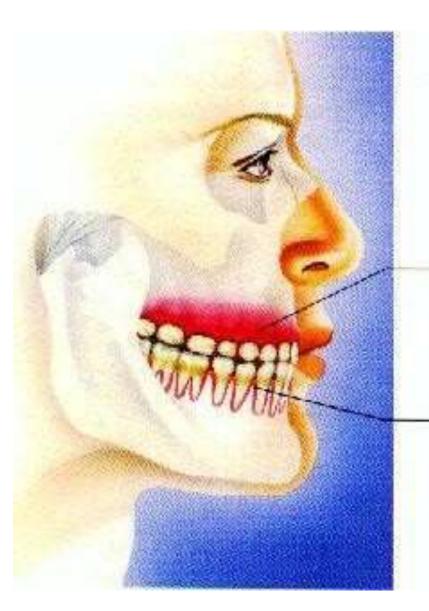
The gums recede farther, destroying more bone and the periodontal ligament. Teeth—even healthy teeth—may become loose and need to be extracted.





MICROSCOPICALLY

- Fibrosis of gingiva
- Inflammatory and immunopathologic tissue damage
- Plasma cell continue to dominate connective tisse
- Neutrophils continue to dominate junctional epithelium and gingival crevice

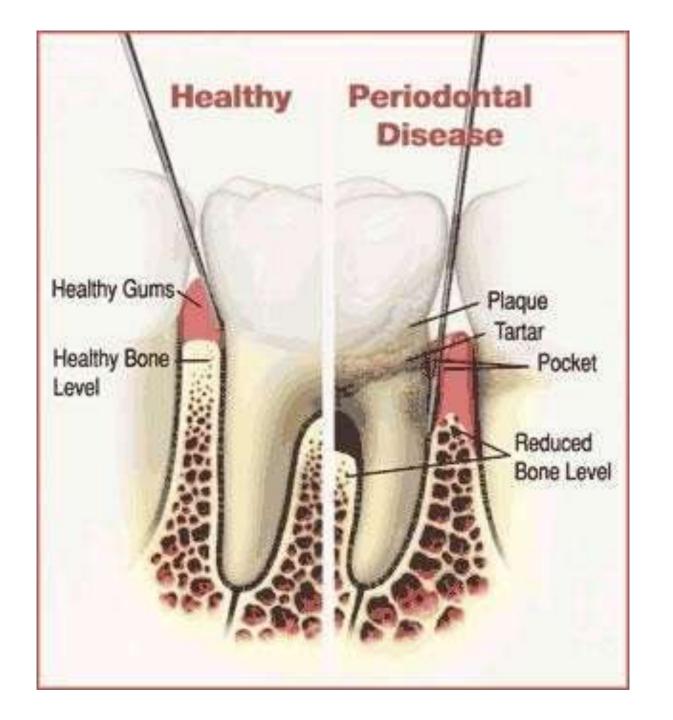


Gingivitis.

Inflamed gums, the early reversible stage of periodontal disease.

Periodontal diseases.

Affect gums, bones, and other supporting structures of the teeth.



STAGE	TIME (In Days)	BLOOD VESSELS	J.E AND SULCULR EPI.
I. Initial lesion	2 - 4	Vascular dilationVasculitis	Infiltration by PMNs
II. Early lesion	4 - 7	Vascular proliferation	Rete pegsAtrophic areas
III. Established lesion	14 - 21	Same as II , plus blood stasis	Same as II but advanced

STAGE	IMMUNE CELLS	COLLAGEN	CLINICAL FINDINGS
I.	PMNs	Perivascular loss	Gingival fluid flow
II.	Lymphocyt es	Increased loss around infiltrate	Erythema Bleeding on probing
III.	Plasma cells	Continued loss	Changes in colour size, texture

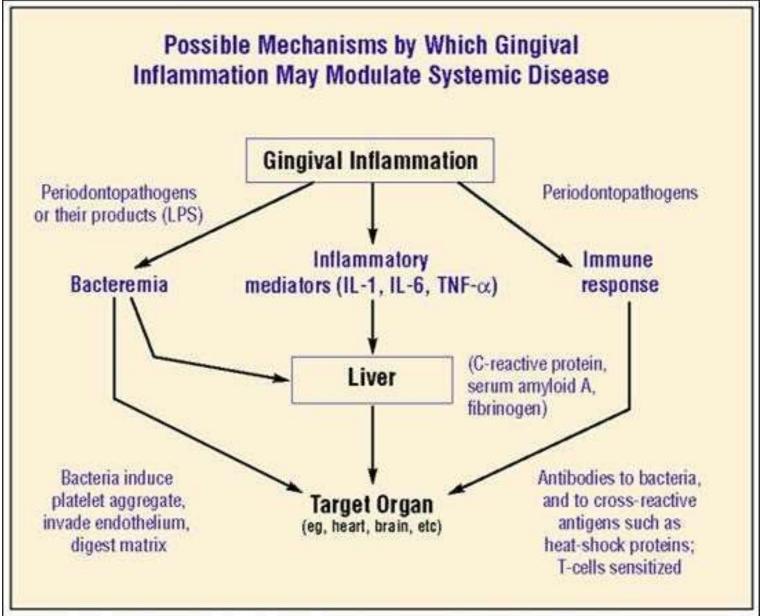


Figure 3: Model to explain oral inflammation-systemic disease association

From: Scannapieco, FA: Periodontal inflammation: from gingivitis to systemic disease? Compend Cont Educ Dent. 2004; 25(7) (Suppl1): 16-24.

PREVENTION

- Regular oral hygiene that is daily brushing and flossing
- Mouth wash is optional usually using a saline solution or chlorhexidine
- Plaque control programs along with perio scaling and curettage

RESEARCHERS ANALYSED DATA ON CALCIUM CONSUMPTION PERTODONTAL DISEASE AND **INDICATORS** IN NEARLY 13000 PEOPLE REPRESENTING US ADULTS. THEY FOUND HAD CALCIUM INTAKE 0F WOMEN WHO THEN 500MG OR ABOUT HALF WERE AS TWICE LIKELY TOHAVE GUM DISEASE AS MEASURED BY LOSS 0F GUMS ATTACHMENT 0F FROM TEETH. WAS PARTICULARLY FOR PEOPLE IN 20'S AND 30'S

RESEARCH SAYS THAT RELATIONSHIP BETWEEN CALCIUM AND GUM DISEASE IS LIKELY DUE TO CALCIUM'S ROLE IN BUILDING DENSITY IN ALVEOLAR BONE THAT SUPPORTS TEETH

DIAGNOSIS

- A dental hygienist or dentist will check for symptoms of gingivitis and examine amount of plaque in the oral cavity.
- A dentist will also look for signs of periodontitis using x-rays or periodontal probes.

TREATMENT

- Oral hygiene that is proper brushing and flossing
- **Gargling of brine water**
- Anti-bacterial rinses or mouthwash, chlorhexidine digluconate 0.2% may reduce swelling and local mouth gels which are antiseptic and anaesthetic can also help.

COMPLICATIONS

- **GRECURRENCE**
- **CS PERIODONTITIS**
- «INFECTION OF ABSCESS OF GINGIVA
- STRENCH MOUTH (BACTERIAL INFECTION AND ULCERATION OF GUMS)

CLASSIFICATION OF GINGIVITIS

Gingivitis can occur with sudden onset and short duration and can be painful

RECURRENT GINGIVITIS

 Reappears after having been eliminated by treatment or disappearing spontaneously

CHRONIC GINGIVITIS

- Slow in onset
- Long duration
- Painless, unless complicated by acute or subacute exacerbations
- Inflammation persists or resolves



Figure 22-1 Chronic gingivitis. The marginal and interdental gingivae are smooth, edematous, and discolored. Isolated areas of acute response are seen.

GINGIVITIS

- **S**Localized
- **G**eneralized
- **Marginal**
- **S**Papillary
- **S**Diffuse



Figure 22-3 Generalized marginal gingivitis in the upper jaw with areas of diffuse gingivitis.



Figure 22-2 Localized, diffuse, intensely red area facial of tooth #7 and dark-pink marginal changes in remaining anterior teeth.

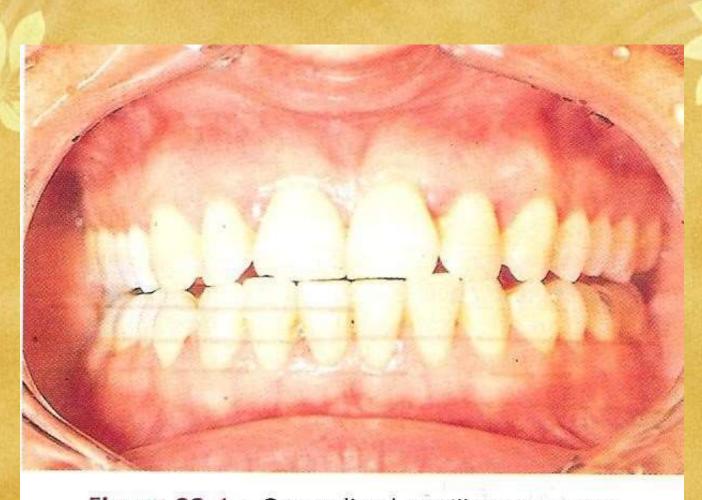


Figure 22-4 Generalized papillary gingivitis.



Figure 22-5 Generalized marginal and papillary gingivitis.



Figure 22-6 Generalized diffuse gingivitis involves the marginal, papillary, and attached gingivae.





THANKYOU

Peri-implantitis

 Peri-implant disease: An inflammatory reaction around the tissue surrounding an implant consist of two forms

Peri-implant mucositis

Peri-implantitis

Mombelli A. et al. *Periulintal* 2000 1998;17:63-76. The Sixth European Workshop on Periodontoloy 2008

Peri-implant mucositis

- Clinical findings
 - Bleeding on probing / gingival redness
 - Probing depth ≥ 4 mm
 - No radiographic bone loss

San M. et al. J. Clin Prinshnts/ 2012;39 Suppl.12):202-206.

Prevalence: 48% of implants

Roos-Jansaker A.M. J Cliv Periolantal 2006;33:290-295.

Peri-implant mucositis





CLASSIFICATION OF PERI-IMPLANTITIS

Early	PD ≥ 4 mm (bleeding and/or suppuration on probing*) Bone loss < 25% of the implant length**
Moderate	PD ≥ 6 mm (bleeding and/or suppuration on probing*) Bone loss 25% to 50% of the implant length**
Advanced	PD ≥ 8 mm (bleeding and/or suppuration on probing*) Bone loss > 50% of the implant length**

Peri-implantitis

- An inflammatory process
- Soft tissue inflammation, Bleeding on probing
- Probing depth ≥ 5 mm
- Suppuration

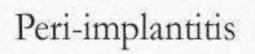
Peri-implantitis

- Progressive loss of supporting bone beyond biological bone remodeling
 - Mean crestal bone loss of 0.9-1.6 mm in first post-surgical year
 - Then annual bone loss of 0.02-0.15 mm
 - In case of no baseline radiograph, 2 mm vertical distance from expected marginal bone level

San M. et al. J Clin Paindont 2012;39 Suppl. 12):202-206.

Prevalence : varied from 11%-47% depending on the threshold used

Koldlands OC. et al. J Prindwad 2010;81:231-238.







Etiologies

- Formation of biofilm
- Gram-negative anaerobic bacteria: similar to natural teeth in periodontal disease
 - Peri-implant mucositis Gingivitis
 - Peri-implantitis Periodontitis : S.aureus could be found as the initiation of periimplantitis

Hen-Mayfield LJ, et al. Penakusi 2000 2010;53:167-181. Leohardt A. et al. Clin Oral Implanti Rei 1999; 10:399-345.

Factors associated Peri-implantitis

- History of periodontitis: two times
- Smoking: 3-4 times increased risk for peri-implantitis

Mombelli A. et al. Clis Ond Implants Ra 2012;23;5uppl.6):67-76.

Residual cement : Rough area beneath gingival margin → Bacterial attachment

Linkevicius T. et al. Gio Ond Implanti Res 2012 published online

Implant position and design : inability to clean

Treatment

- · Primary goals
 - Resolve inflammation
 - Arrest the progression of disease

Treatment

Non-Surgical treatment

- · Mechanical debridement
 - Ultrasonic scaler
 - * Hand instruments: Plastic curette
 - Rubber cup & pumice
- Plaque control

Non-Surgical treatment

- Effective in Peri-implant mucositis
 - carbon fibers curette, rubber cup, pumice
- In peri-implantitis, mechanical debridement alone was found not to be effective

Antibiotic

- Use in conjunction with mechanical debridement and chemical disinfection
- Local: high concentration, reduce side & adverse effect
 - Tetracyclin HCL (Actisite®)
 - Minocyclin
- Systemic : ornidazole 1000 mg daily, metronidazole, amoxicillin

Surgical approach

- Surgical approach
 - Access surgery: apically positioned flap, surface modification
 - * Resective
 - Regenerative : guided tissue regeneration, bone grafting
 - Surface decontamination

Surface decontamination

- Chemical agents
 - hydrogen peroxide, citric acid, 35% phosphoric acid
- Photodynamic therapy
 - Photosensitizer + high energy laser light -> destroy bacterial cells
- Laser treatment
 - combined with Chemical agents to archieve higher re-osseointegration

Anti-infective protocol

- Peri-implantitis VS Periodontitis
 - Eiology
 - · Treatment
- Anti-infective protocol have been adopted to treat peri-implantitis
 - Open flap debridement
 - Implant surface decontamination
 - Systemic antibiotic : Amoxicillin (500 mg) + Metronidazole (400 mg) 7-10 days

Conclusions

- Non-surgical treatment alone was found to be effective in peri-implant mucositis: carbon fibers curette, rubber cup, pumice
- Peri-implantitis with mild bone loss: Mechanical debridement,
 Antiseptic(CHX mouthwash), Systemic/Local Antibiotic, Resective surgery
- Peri-implantitis with moderate bone loss: Mechanical debridement, Antiseptic(CHX mouthwash), Systemic/Local Antibiotic, Open flap debridement, Surface decontamination, Regenerative surgery

- Bone fill & Re-osseointegraion
 - Regenerative procedure > Open flap debridement
 - Membrane did not improve treatment outcome in comparison to the use of autogenous bone alone
 - Systemic antibiotic (Amoxicilin plus metronidazole) and antiseptic mouthrinse(CHX): improved clinical outcomes

- No single method of surface decontamination(Chemical agents, air abrasive, lasers) was found to be superior
- Citric acid(40%,30-60 sec) has proved to be most effective agent for bacterial growth reduction on HA surfaces
- The simplest method of surface decontamination; gauze soaked alternately in CHX and saline, should be preferred when combined with membranecovered autogenous bone graft