Gingival Crevicular Fluid

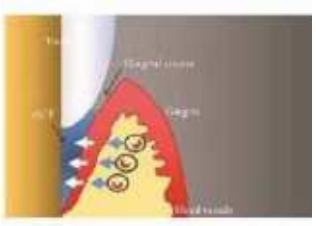
Dr Hadeel Mohammed Abbood

Sulcular fluid, or (GCF),

- contains an array of biologic mediators, cells, and bacteria.
- Recognized since the 19th century, its possible role in oral defense was first elucidated by the pioneering work of Waerhaug and Brill & Krasse during the 1950s.
- The latter investigators applied filter paper to the gingival sulci of dogs that had previously been injected intramuscularly with fluorescein; within 3 minutes, the fluorescent material was recovered on the paper strips.
- This indicated the passage of fluid from the bloodstream through the tissues and the exiting of fluid via the gingival sulcus.
- In subsequent studies, Brill confirmed the presence of GCF in humans and considered it as “transudate.” However, others demonstrated that GCF is an inflammatory exudate rather than a continuous transudate.
- In strictly normal gingiva, little or no fluid can be collected.
- Potential markers from crevicular fluid are now used as diagnostic tools for the activity of periodontal diseases and a return to homeostasis, with potential for the evaluation of systemic markers.

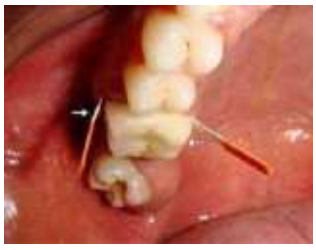
Methods of Collection

- The most difficult hurdle to overcome when collecting GCF is the scarcity of material that can be obtained from the sulcus. Many collection methods have been tried. These methods include the use of absorbing paper strips, the placement of twisted threads around and into the sulcus, and techniques involving micropipettes and intracrevicular washings.
- There are limitations to the techniques, including fluid collection, collection time, low rate, contamination, and reproducibility.



The absorbing paper strips

- Placed within the sulcus (intrasulcular method) or at its entrance (extrasulcular method).
- Placement of the filter paper strip in relation to the sulcus or pocket is important.
- The Brill technique involves inserting it into the pocket until resistance is encountered. This method produces some degree of irritation of the sulcular epithelium that by itself can trigger the flow of fluid.
- To minimize this irritation, Löe and Holm-Pedersen placed the filter paper strip just at or over the pocket entrance. In this way, fluid that seeps out is picked up by the strip, but the sulcular epithelium is not in contact with the paper.



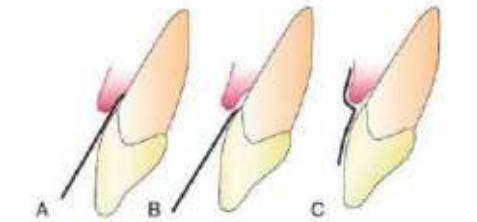
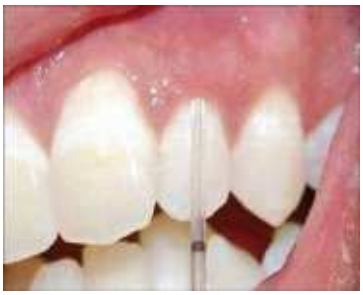
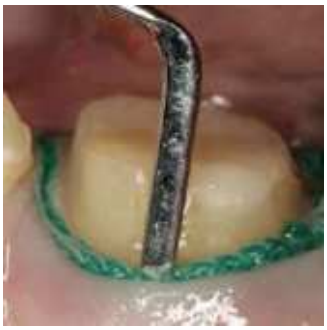


Fig. 16.1 Placement of a filter strip in the gingival sulcus for the collection of fluid. (A) Intrasulcular method. (B–C) Extrasulcular methods.

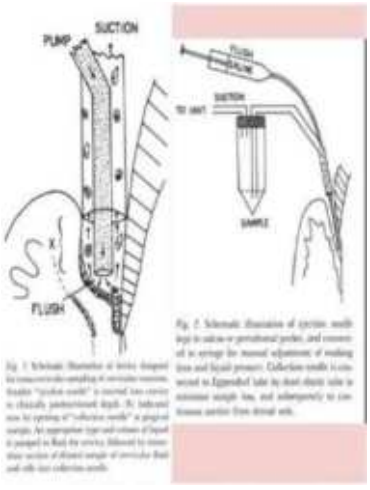
Other methods

- Weinstein and colleagues used preweighed twisted threads.
- The threads were placed in the gingival crevice around the tooth, and the amount of fluid collected was estimated by weighing the sample thread.
- The use of micropipettes permits the collection of fluid by capillarity. Capillary tubes of standardized length and diameter are placed in the pocket, and their contents are later centrifuged and analyzed.



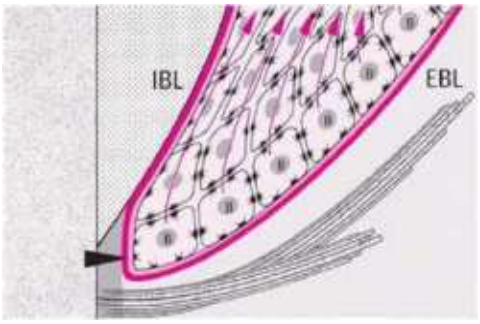


- Crevicular washings can be used to study GCF from clinically normal gingiva. One method involves the use of an appliance that consists of a hard acrylic plate that covers the maxilla, with soft borders and a groove that follows the gingival margins; it is connected to four collection tubes. Washings are obtained by rinsing the crevicular areas from one side to the other with the use of a peristaltic pump.
- A modification of the previous method involves the use of two injection needles that have been fitted one within the other so that, during sampling, the inside (ejection) needle is at the bottom of the pocket and the outside (collecting) needle is at the gingival margin.
- The collection needle is drained into a sample tube via continuous suction.



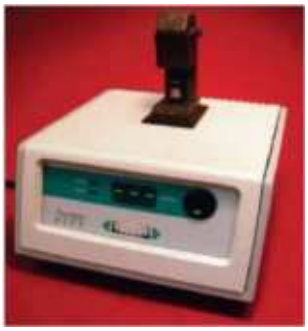
Permeability of Junctional and Sulcular Epithelia

- The initial studies by Brill and Krasse involving the use of fluorescein were later confirmed with substances such as India ink and saccharated iron oxide. Substances that have been shown to penetrate the sulcular epithelium include albumin, endotoxin, thymidine, histamine, phenytoin, and horseradish peroxidase.
- These findings indicate permeability to substances with a molecular weight of up to 1000 kD (kilodalton).
- In intact epithelium. The intercellular movement of molecules and ions along intercellular spaces appears to be a possible mechanism. Substances that take this route do not traverse the cell membranes.



Amount

- The amount of GCF collected on a paper strip can be studied in multiple ways. The wetted area can be made more visible by staining with Ninhydrin; it is then measured planimetrically on an enlarged photograph or with a magnifying glass or a microscope.
- An electronic method has been devised for measuring the fluid collected on a "blotter" (Periopaper) with the use of an electronic transducer (Periotron, Harco Electronics, Winnipeg, Manitoba, Canada). The wetness of the paper strip affects the flow of an electric current and provides a digital readout. A comparison between the Ninhydrin-staining method and the electronic method performed in vitro revealed no significant differences between the two techniques.
- The amount of GCF collected is extremely small. Measurements performed showed that a strip of paper 1.5-mm wide and inserted 1 mm within the gingival sulcus of a slightly inflamed gingiva absorbs about 0.1 mg of GCF in 3 minutes. The mean GCF volume in the proximal spaces from the molar teeth ranged from 0.43 to 1.56 μL in patients with mean GI less than 1.



Composition

- The components of GCF are characterized by individual proteins, metabolites, specific antibodies, antigens, and enzymes of several specificities. The GCF also contains cellular elements.
- Multiple research efforts have attempted to use GCF components to detect or diagnose active disease or to predict which patients are at risk for periodontal disease. So far, more than 40 compounds found in GCF have been analyzed, but their origin is not known with certainty. These compounds can be derived from the host or produced by bacteria in the gingival crevice, but their source can be difficult to elucidate; examples include β -glucuronidase, which is a lysosomal enzyme, and lactic acid dehydrogenase, which is a cytoplasmic enzyme. The sources of collagenases may be fibroblasts or polymorphonuclear leukocytes, or collagenases may be secreted by bacteria. Phospholipases are lysosomal and cytoplasmic enzymes, but they are also produced by microorganisms. The majority of GCF elements detected thus far have been enzymes, but there are nonenzymatic substances as well.

1- Cellular Elements

Cellular elements found in GCF include bacteria, desquamated epithelial cells, and leukocytes (i.e., PMNs, lymphocytes, and monocytes/ macrophages), which migrate through the sulcular epithelium.

2- Electrolytes

Potassium, sodium, and calcium have been studied in the GCF. Most studies have demonstrated a positive correlation of calcium and sodium concentrations with the sodium/potassium ratio seen with inflammation

3- Organic Compounds

- Both carbohydrates and proteins have been investigated. Glucose hexosamine and hexuronic acid are two compounds that are found in GCF. Blood glucose levels do not correlate with GCF glucose levels; glucose concentration in GCF is three to four times greater than that in serum. This is interpreted not only as a result of the metabolic activity of adjacent tissues but also as a function of the local microbial flora.
- The total protein content of GCF is much less than that of serum. No significant correlations have been found between the concentration of proteins in GCF and the severity of gingivitis, pocket depth, or extent of bone loss. Metabolic and bacterial products identified in GCF include lactic acid, urea, hydroxyproline, endotoxins, cytotoxic substances, hydrogen sulfide, and antibacterial factors. Many enzymes have also been identified.
- The methodology used to analyze GCF components is as varied as the diversity of those components. Examples include fluorometry to detect metalloproteinases, enzyme-linked immunosorbent assays to detect enzyme levels and interleukin-1β (IL-1β), radioimmunoassays to detect cyclooxygenase derivatives and procollagen III, high-pressure liquid chromatography to detect timidazole, and direct and indirect immunodot tests to detect acute-phase proteins.

Cellular and Humoral Activity in GCF

- Monitoring periodontal disease is a complicated task, because few noninvasive procedures can follow the initiation and progress of the disease. Analyzing GCF constituents in health and disease may be extremely useful as a result of GCF's simplicity and because GCF can be obtained with noninvasive methods.
- The analysis of GCF has identified cell and humoral responses in both healthy individuals and those with periodontal disease. The cellular immune response includes the appearance of cytokines in GCF, but there is no clear evidence of a relationship between cytokines and disease. However, IL-1α and IL-1β are known to increase the binding of PMNs and monocytes/macrophages to endothelial cells, to stimulate the production of prostaglandin E2 and the release of lysosomal enzymes, and to stimulate bone resorption. Preliminary evidence also indicates the presence of interferon-α in GCF, which may have a protective role in periodontal disease because of its ability to inhibit the bone resorption activity of IL-1β.
- Because the amount of fluid recoverable from gingival crevices is small, only the use of very sensitive immunoassays permits the analysis of the specificity of antibodies. A study that compared antibodies in different crevices with serum antibodies directed at specific microorganisms did not provide any conclusive evidence regarding the significance of the presence of antibodies in GCF among individuals with periodontal disease.
- Although the role of antibodies in the gingival defense mechanisms is difficult to ascertain, the consensus is that in a patient with periodontal disease, a reduction in antibody response is detrimental, and an antibody response plays a protective role

Clinical Significance

- As an exudate, GCF is a biologic fluid that has potential in diagnostics and disease management. Its presence in clinically normal sulci can be explained, because gingiva that appears clinically normal invariably exhibits inflammation when it is examined microscopically Commercially available kits for diagnosis are now available.
- The amount of GCF is greater when inflammation is present and it is sometimes proportional to the severity of inflammation.
- GCF production is not increased by trauma from occlusion, but it is increased by the mastication of coarse foods, toothbrushing and gingival massage, ovulation, hormonal contraceptives, prosthetic appliances, and smoking. Other factors that influence the amount of GCF are circadian periodicity and periodontal therapy.

TABLE 16.1 Gingival Crevicular Fluid Diagnostic Tests

Test Name	Target	References
Periocheck	Proteinases	Page RC. Host response tests designed for diagnosing periodontal disease. *J Periodontol* 63(4 Suppl):356–366, 1992.
Prognostik	Elastase	http://www.ncbi.nlm.nih.gov/pmc/articles/PMC3118084/
Biolise	Elastase	https://www.ncbi.nlm.nih.gov/pmc/articles/PMC3118084/
MMP dipstick	MMPs	Mäntylä P, Stenman M, Kinane DF, Tikanoja S, Luoto H, Salo T, Sorsa T: Gingival crevicular fluid collagenase-2 (MMP-8) test stick for chair-side monitoring of periodontitis. *J Periodontal Res* 38(4):436-439, 2003.
TOPAS	Bacterial toxins and proteases	http://www.ncbi.nlm.nih.gov/pmc/articles/PMC3118084/
Pocket watch	AST	Mäntylä P, Stenman M, Kinane DF, Tikanoja S, Luoto H, Salo T, Sorsa T: Gingival crevicular fluid collagenase-2 (MMP-8) test stick for chair-side monitoring of periodontitis. *J Periodontal Res* 38:436–439,|2003.

MMP, Mucous membrane pemphigoid.

- ***Circadian Periodicity***
- There is a gradual increase in the amount of GCF from 6 a.m. to 10 p.m. and a decrease thereafter.
- ***Sex Hormones***
- Female sex hormones increase GCF flow, probably because they enhance vascular permeability. Pregnancy, ovulation, and hormonal contraceptives all increase GCF production.
- ***Mechanical Stimulation***
- Chewing and vigorous gingival brushing stimulate the flow of GCF. Even minor stimuli represented by intrasulcular placement of paper strips increases the production of fluid.
- ***Smoking***
- Smoking produces an immediate transient but marked increase in GCF flow but, in the long term, a decrease of salivary and GCF flow.
- ***Periodontal Therapy***
- There is an increase in GCF production during the healing period after periodontal surgery.

- **Drugs in Gingival Crevicular Fluid**
- Drugs that are excreted through the GCF may be used advantageously in periodontal therapy. Bader and Goldhaber demonstrated in dogs that tetracyclines are excreted through the GCF; this finding triggered extensive research that showed a concentration of tetracyclines in GCF as compared with serum.
- Metronidazole is another antibiotic that has been detected in human GCF

Leukocytes in the Dentogingival Area

- Leukocytes have been found in clinically healthy gingival sulci in humans and experimental animals. The leukocytes found are predominantly PMNs. They appear in small numbers extravascularly in the connective tissue adjacent to the apical portion of the sulcus; from there, they travel across the epithelium to the gingival sulcus, where they are expelled.
- Leukocytes are present in sulci even when histologic sections of adjacent tissue are free of inflammatory infiltrate. Differential counts of leukocytes from clinically healthy human gingival sulci have shown 91.2% to 91.5% PMNs and 8.5% to 8.8% mononuclear cells.
- Mononuclear cells were identified as 58% B lymphocytes, 24% T lymphocytes, and 18% mononuclear phagocytes. The ratio of T lymphocytes to B lymphocytes was found to be reversed from the normal ratio of about 3:1 found in peripheral blood to about 1:3 in GCF.
- Leukocytes are attracted by different plaque bacteria, but they can also be found in the dentogingival region of germ-free adult animals. Leukocytes were reported in the gingival sulcus in non-mechanically irritated (resting) healthy gingiva, thereby indicating that their migration may be independent of an increase in vascular permeability. The majority of these cells are viable and have phagocytic and killing capacity. Therefore leukocytes constitute a major protective mechanism against the extension of plaque into the gingival sulcus.
- The main port of entry of leukocytes into the oral cavity is the gingival sulcus

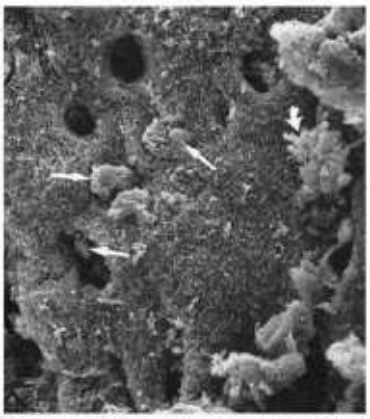


Fig. 16.3 Scanning electron microscope view of the periodontal pocket wall. Several leukocytes are emerging *(straight arrows)*, some of which are partially covered by bacteria *(curved arrow)*. Empty holes correspond to tunnels through which leukocytes have emerged.

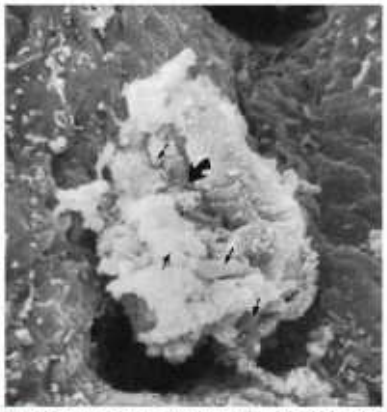
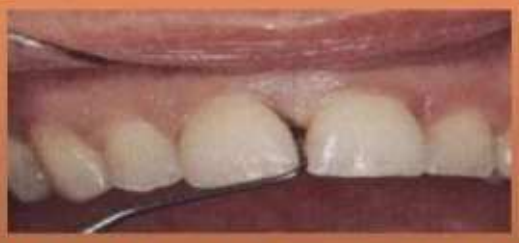


Fig. 16.4 Scanning electron microscope view at higher magnification than shown in Fig. 16.3. A leukocyte emerging from the pocket wall is covered with bacteria *(small arrows)*. The *large curved arrow* points to a phagosomal vacuole through which bacteria are being engulfed.

Thank you

PERIODONTAL EXAMINATION



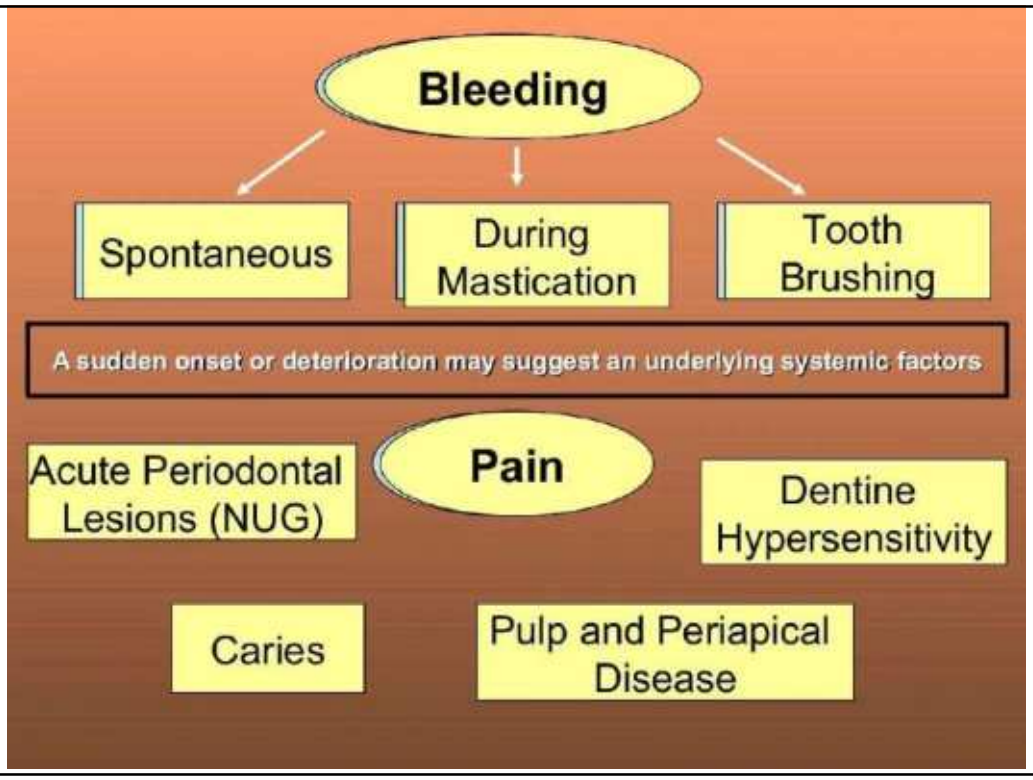
DIAGNOSIS AND PROGNOSIS

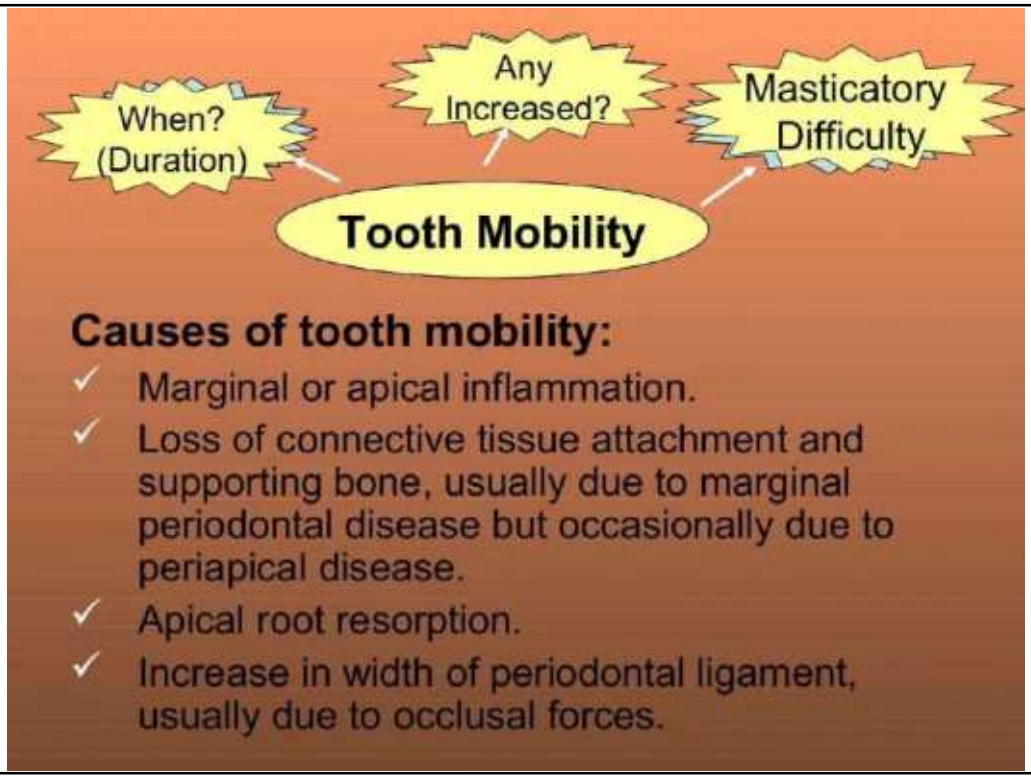
HISTORY

The main concerns of the patient

SYMPTOMS:

- ✓Gingival Bleeding, Pain and Swelling.
- ✓Tooth mobility.
- ✓Bad breath and taste.


Bleeding
Spontaneous
During Mastication
Tooth Brushing
A sudden onset or deterioration may suggest an underlying systemic factors
Pain
Acute Periodontal Lesions (NUG)
Dentine Hypersensitivity
Caries
Pulp and Periapical Disease


When? (Duration)
Any Increased?
Masticatory Difficulty
Tooth Mobility
Causes of tooth mobility:
✓ Marginal or apical inflammation.
✓ Loss of connective tissue attachment and supporting bone, usually due to marginal periodontal disease but occasionally due to periapical disease.
✓ Apical root resorption.
✓ Increase in width of periodontal ligament, usually due to occlusal forces.

MEDICAL HISTORY

OBJECTIVES:

1. To identify systemic factors which may help to account for the periodontal condition, ex: Pregnancy, Diabetes Mellitus.
2. To note the existence of systemic condition for which especial precautions (ex. Antibiotic prophylaxis) are required to safeguard the patient during the periodontal therapy.
3. To note the presence of any transmissible disease which may present a hazard to the clinician, dental surgery staff or other patients.

DENTAL HISTORY

- **Patient's attitude toward dental health.**
- **Date and nature of the last dental treatment.**
- **Regularity of previous dental treatment.**
- **Oral hygiene habits.**
  a. Tooth brush (type and frequency)
  b. Dental floss.
  c. Others.
- **Habits related to oral health or disease** (bruxism, smoking)

Examination

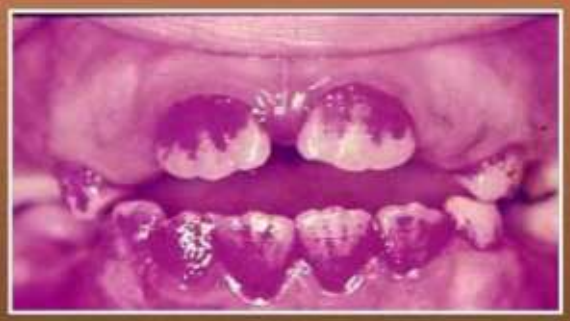
1. Gingival Inflammation, Plaque and Calculus

GINGIVITIS

- Changes of the colour
- Gingival Exudate
- Enlargement (edema or hyperplasia)
- Bleeding
- Supuration, Ulceration or Sweeling (acute inflammation)

PLAQUE RECORD

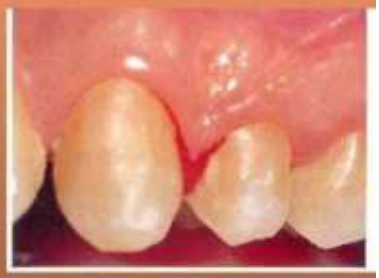
1. At the initial visit.
2. During the subsequent appointments to control the progress of the treatment.



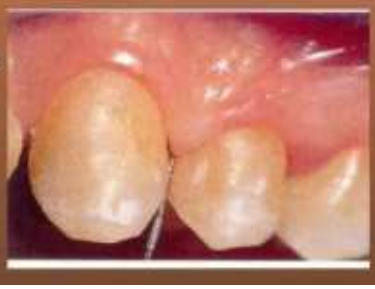
PERIODONTAL PROBING

- **Is used to:**

1. Identify pockets which bleed on probing.



2. To measures the pockets depth.



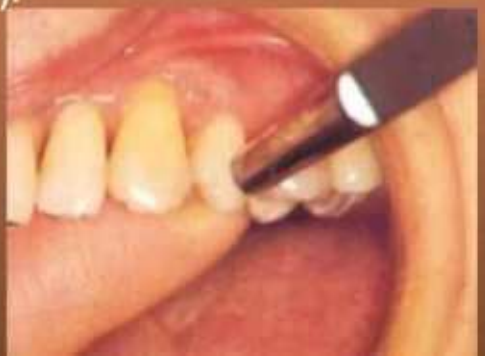
The depth to which the periodontal probe can penetrate beyond the gingival margins depends on:

1. The amount of gingival enlargement.
2. The extent of connective tissue attachment loss.
3. The resistance of the tissue to probing, determined by the extent to which gingival collagen has been replaced by inflammatory infiltrate.

4. The size, shape and tip diameter of the probe.
5. Use of the probe, angle of insertion and pressure applied.
6. The presence of obstructions such as subgingival calculus.
7. The patient's reaction to the discomfort on probing.

MOBILITY

- Each tooth should be rocked between an instrument handle and index finger in a buccolingual direction and mesiodistal direction (when the adjacent tooth is not present).



MOBILITY SCORES

- The amplitude of tooth movement of the crown tip from its most extreme buccal (or mesial) position to its most extreme lingual or distal position should be observed:

Grade 1- Visible horizontal mobility up to 1 mm.

Grade 2- Visible horizontal mobility between 1 and 2 mm.

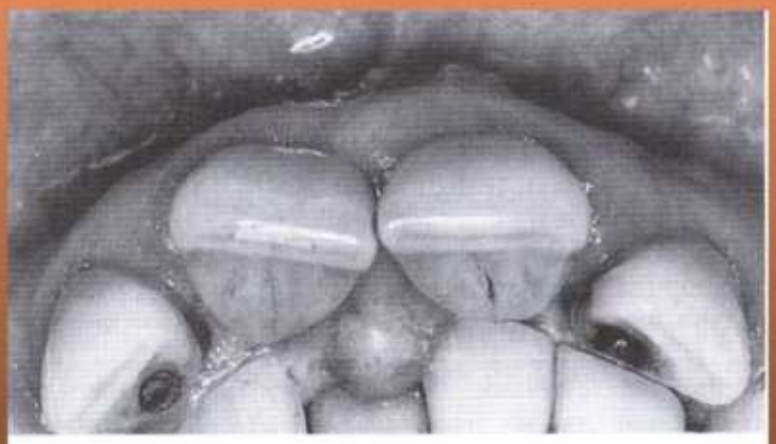
Grade 3- Visible horizontal mobility greater than 2 mm or rotation or vertical mobility (depression).

Occlusion

Parafunctional habits

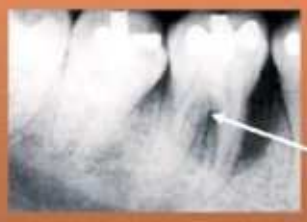
- The occlusion should be examined for detect premature or interfering contacts as contributory factors.
- Vertical bone destructive pattern is often associated with traumatic occlusion.

Occlusion



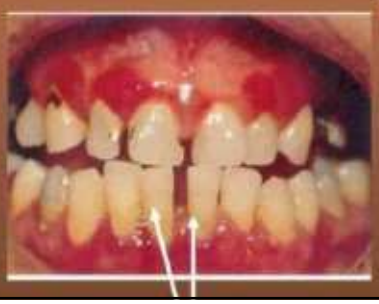
g. 18.3 Deep traumatic overbite.

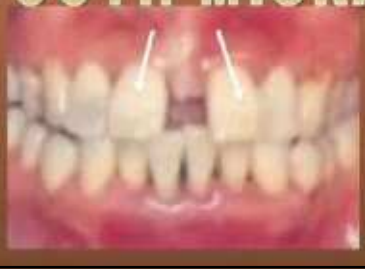
GINGIVAL RECESSION



FURCATION LESIONS

TOOTH MIGRATION

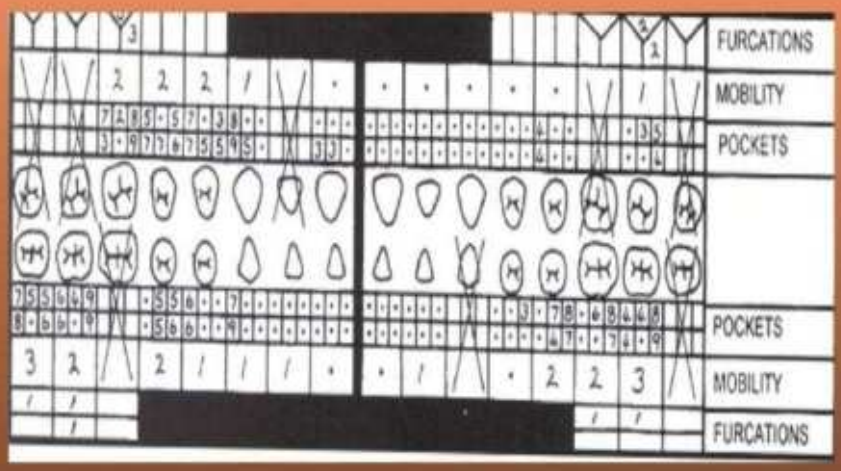




OTHER CLINICAL FINDINGS

- Overhanging Restorations
- Caries
- Missing Teeth
- Non- vital Teeth
- Unerupted teeth

PERIODONTAL CHART

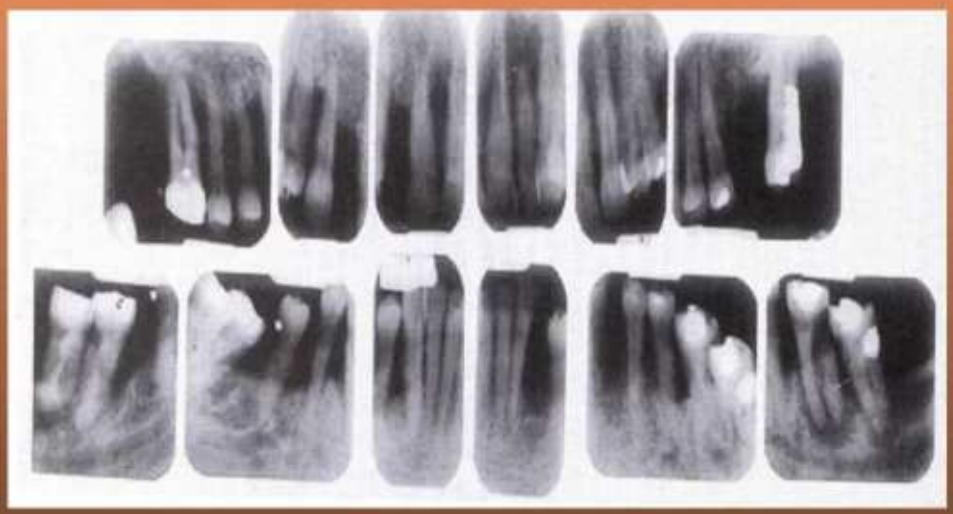


RADIOGRAPHS

- **Radiographs may:**

1. Show the proportion of support loss in relation to root length, the pattern of bone loss and the rate of destruction.
2. Reveal unerupted teeth, periapical pathology, inadequate endodontic treatment, proximal caries, overhanging margins, etc.

PRETREATMENT RADIOGRAPHS



SPECIAL TESTS

Pulp Vitality Test

Teeth associated with deep Periodontal pocket

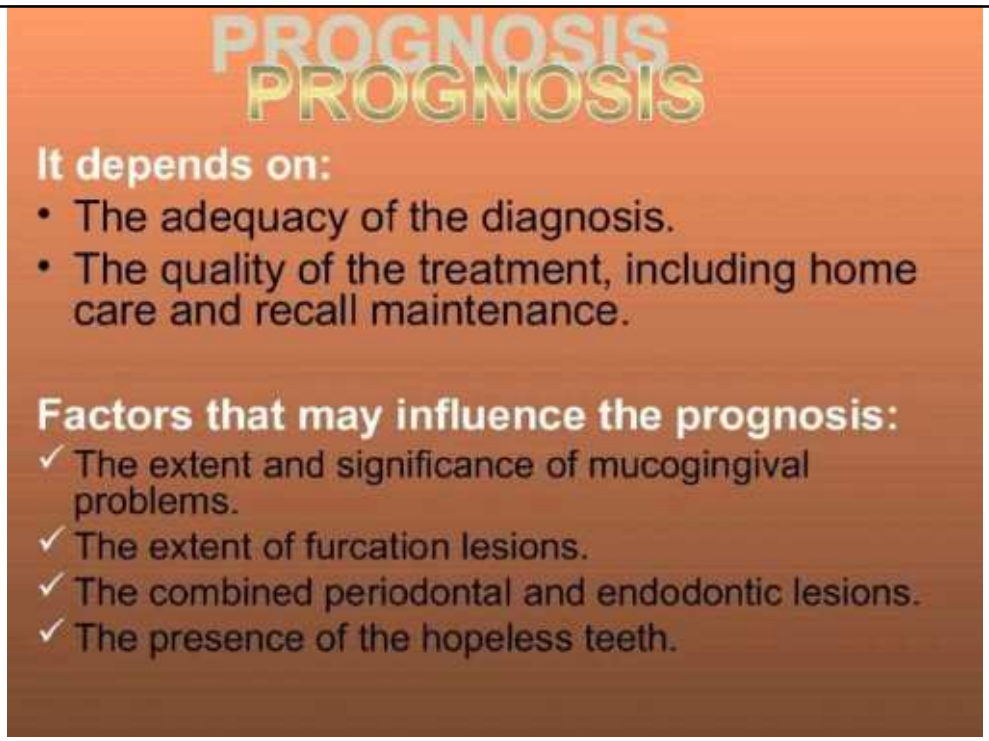
To distinguish a periapical from periodontal abscess

Haematological investigations, ex: full blood count in patients where blood dyscrasias are suspected(neutropenia, leukemia, etc)

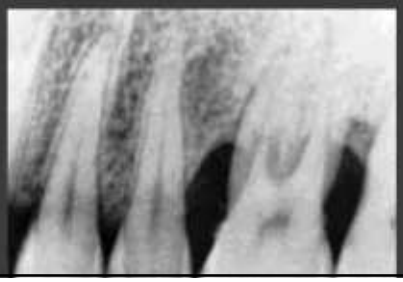
DIAGNOSIS

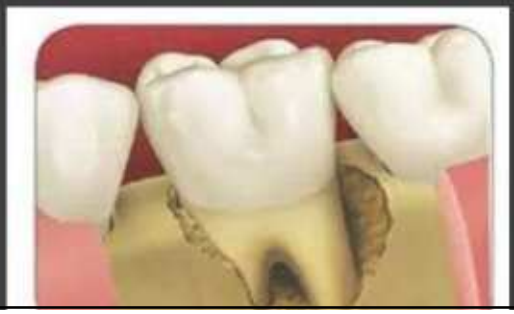
DIAGNOSIS

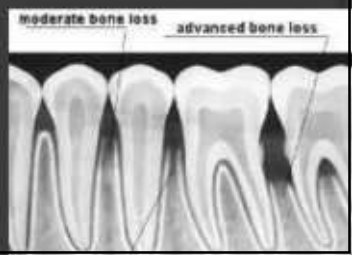
- ✓ **Tooth by tooth diagnosis.**
- ✓ **Whole dentition.**
- ✓ **For an individual patient.**


PROGNOSIS
PROGNOSIS
It depends on:
• The adequacy of the diagnosis.
• The quality of the treatment, including home care and recall maintenance.
Factors that may influence the prognosis:
✓ The extent and significance of mucogingival problems.
✓ The extent of furcation lesions.
✓ The combined periodontal and endodontic lesions.
✓ The presence of the hopeless teeth.

BONE LOSS AND PATTERNS OF BONE DESTRUCTION

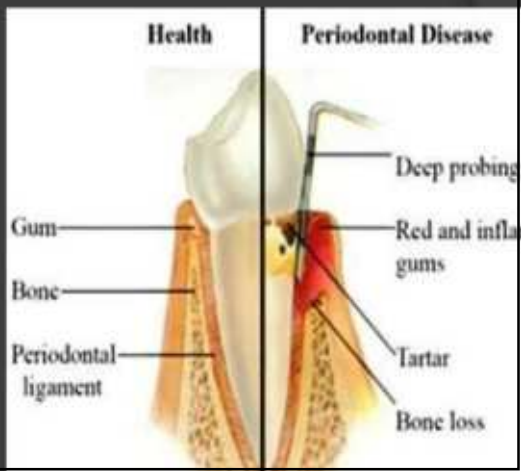






INTRODUCTION

- The bone that forms and supports the tooth is called ALVEOLAR PROCESS
- Destruction of the bone is responsible for tooth loss.
- As the tooth is shed this bone RESORBS



BONE RESORPTION

- The height and density of alveolar bone are normally maintained by an equilibrium.
- Regulated by local and systemic influences between bone formation and resorption.
- When resorption exceeds formation, both bone height and density is reduced

FACTORS CAUSING BONE DESTRUCTION ON PERIODONTAL DISEASE

1) Extension Of Gingival Inflammation

2) Trauma from occlusion(TFO)

BONE DESTRUCTION CAUSED BY EXTENSION OF GINGIVAL INFLAMMATION

- Most common cause
- The inflammatory invasion of bone surface and the initial bone loss marks the transition from GINGIVITIS to PERIODONTITIS

- Extension of inflammation from marginal gingiva to supporting tissues
- The transition from gingivitis to periodontitis is associated with changes in composition of bacterial plaque.

	GINGIVITIS	PERIODONTITIS
Composition of plaque	Coccid rods, straight rods	Motile organisms and

HISTOPATHOLOGY

- Area of inflammation extending from gingiva into suprabony area.
- course : along collagen bundle fibres, blood vessels, loosely arranged tissues
- Extension of inflammation into centre of interdental septum.
- Cortical layer at top of septum are destroyed and inflammation penetrates into bone marrow

RATE OF BONE LOSS

In individuals with no oral hygiene :

FACIAL SURFACE: 0.2mm/year

PROXIMAL SURFACE: 0.3mm/year

PERIODS OF DESTRUCTION

Periodontal destruction occurs in episodic and intermittent manner

↓

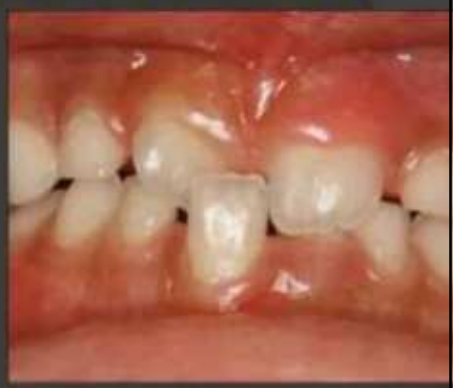
Periods of inactivity and destruction

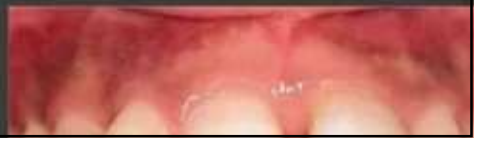
↓

Results in loss of collagen and alveolar bone resulting in deepening of periodontal pocket

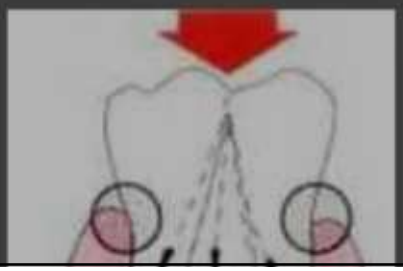
BONE DESTRUCTION CAUSED BY TRAUMA FROM OCCLUSION

- Periodontal response to the external force.
- TFO can occur in presence or absence of inflammation.
- In the absence, effects on alveolar bone ranges from resorption to necrosis





- When combined with inflammation- ZONE OF C
DESTRUCTION
- plaque induced inflammation entering into the zo
of trauma results in angular bone defects, BIZARRE
BONE PATTERN



BONE DESTRUCTION CAUSED BY SYSTEMIC DISORDERS

- Possible relationship between periodontal bone loss and systemic disorders.
- OSTEOPOROSIS : loss of bone mineral content and structural bone changes. Risk factors ageing, smoking, etc

FACTORS DETERMINING BONE MORPHOLOGY IN PERIODONTAL DISEASE

1) Normal variation of alveolar bone:

- thickness width, crestal angulations of interdental septa
- thickness of facial and lingual septa

2) Exostoses:

- Exostoses are outgrowths of bone in varied shapes and sizes
- They can occur as small nodules, sharp ridges, spike like projections, or a combination of these

3) Buttressing bone formation:

- bone formation sometimes occurs in an attempt to buttress bony trabeculae weakened by resorption.
- When this occurs within the jaw it is termed as central buttressing bone

4) Food impaction:

- interdental bone defects occur where proximal contact is abnormal or absent. In such areas food impaction results in inverted bone architecture

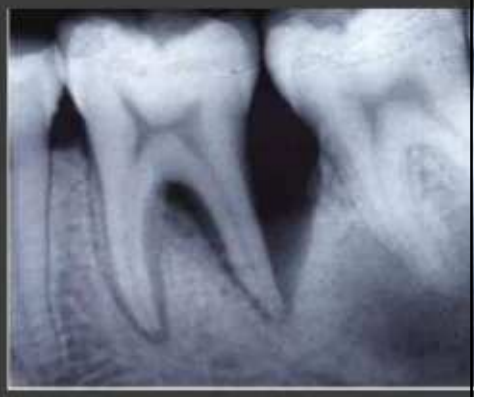
5) aggressive periodontitis:

HORIZONTAL BONE LOSS

- the most common pattern
- bone height is reduced, but bone margins remain perpendicular to tooth surface.
- interdental septa, facial and lingual cortical plates are affected

VERTICAL OR ANGULAR DEFECTS

- Occurs in an OBLIQUE DIRECTION
- leads to a HOLLOWED –OUT trough in the alongside bone
- classified on the basis of number of walls:
  - › one wall defect/one osseous wall



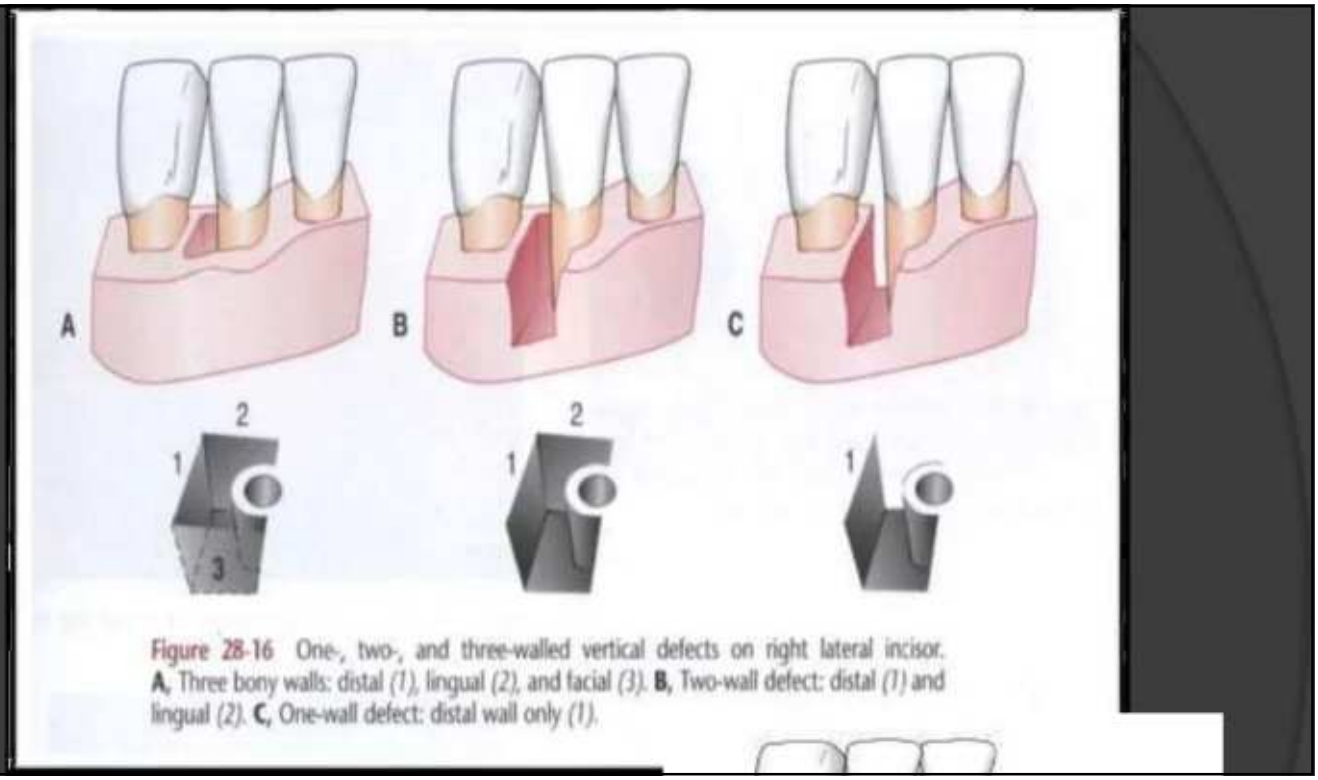


Figure 28-16 One-, two-, and three-walled vertical defects on right lateral incisor. **A**, Three bony walls: distal *(1)*, lingual *(2)*, and facial *(3)*. **B**, Two-wall defect: distal *(1)* and lingual *(2)*. **C**, One-wall defect: distal wall only *(1)*.

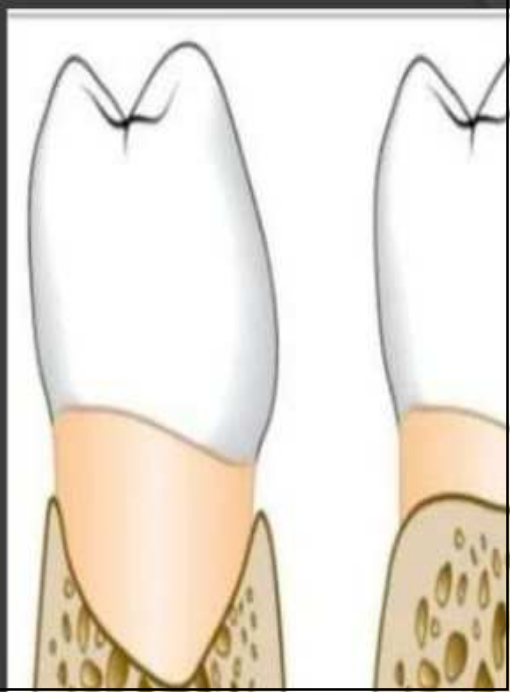
) concavities in the crest of interdental

one confined within faciolingal walls.

) Reasons :

plaque accumulation and difficulty to clean.

normal concavity in lower

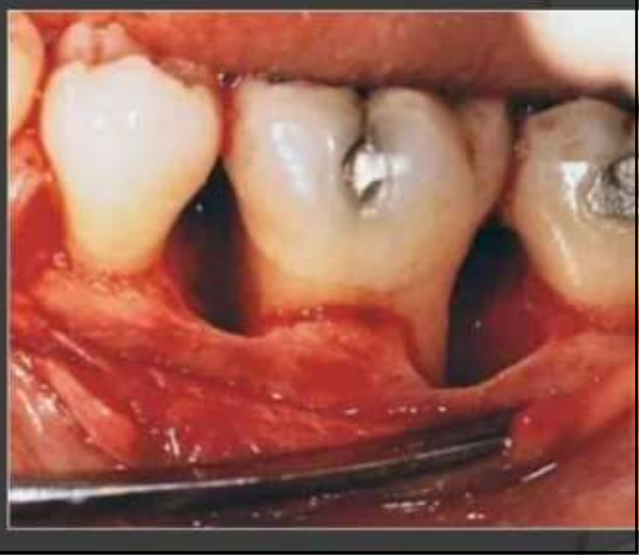


BULBOUS BONE CONTOURS

- bony enlargement
- an adaptation to Exostoses
- adaptation to function or buttressing bone formation.
- maxilla>mandible

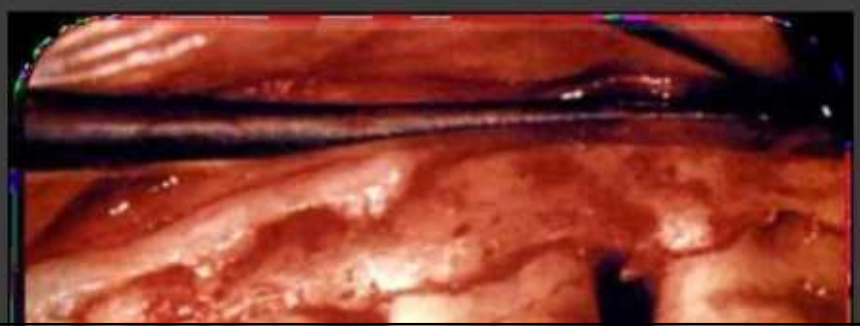
REVERSED ARCHITECTURE

- produced by loss of interdental bone, facial and lingual plates without concomitant loss of radicular bone
- maxilla more commonly affected



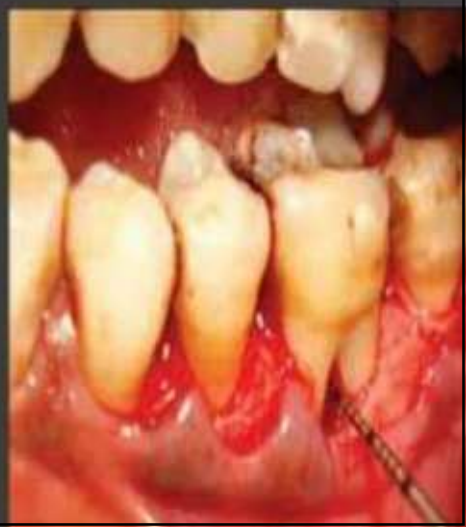
LEDGES

- plateau-like bony margins
- caused by resorption of thickened bony plates



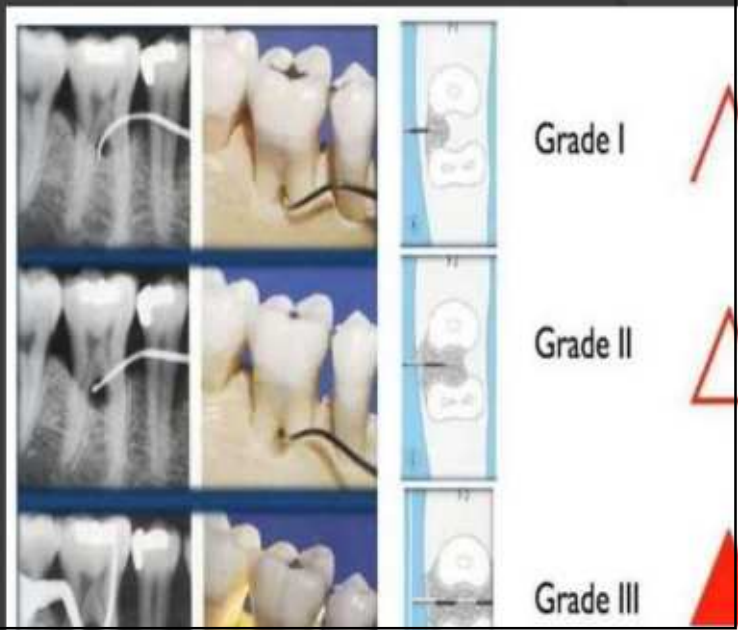
FURCATION INVOLVEMENT

- Involvement of bifurcation or trifurcation of multirooted teeth by periodontal disease.
- SITE: most common in mandibular molars, least common in maxillary premolar.

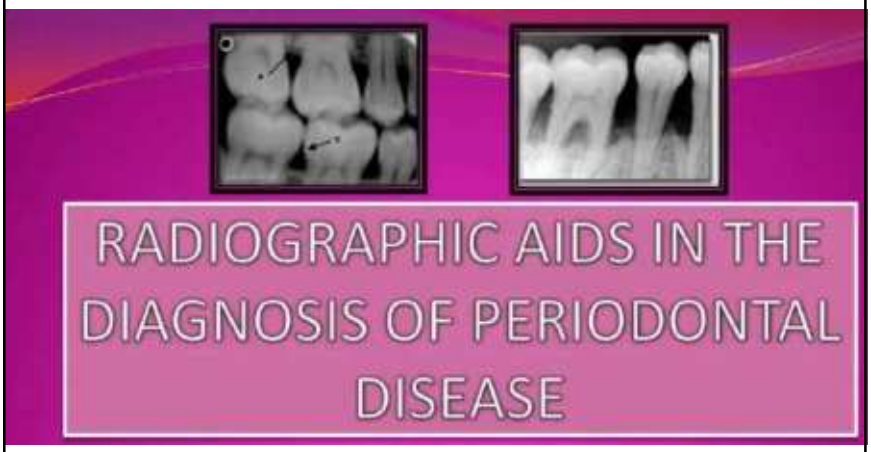


CLASSIFICATION -- GLICKMAN'S(1953) :

- GRADE I : Incipient bone loss, suprabony pocket involving soft tissue, no radiographic changes
- GRADE II : partial bone loss, bone destroyed in one or more surfaces of furcation, parts of PDL and alveolar bone remains intact
- GRADE III : total bone loss with through and thro ugh opening of furcation, facial or lingual or both orifices of furcation cannot be seen because of soft tissue coverage



- Although periodontitis is an infectious disease of the gingival tissue , changes that occur in bone are crucial because destruction of bone is responsible for tooth loss.
- Bone loss patterns associated with periodontal disease is varied and the type of management depends upon the type of loss.



RADIOGRAPHIC AIDS IN THE DIAGNOSIS OF PERIODONTAL DISEASE

INTRODUCTION

- The radiograph is **a valuable aid** in the:
  - ❑ Diagnosis of periodontal disease
  - ❑ Determination of the prognosis, and
  - ❑ Evaluation of the outcome of treatment.
- However radiograph is an adjunct to the clinical examination, not a substitute for it.
- The radiograph reveals alteration in calcified tissues
- It does not reveals current cellular activity but shows effects of past cellular experience on the bone & roots.

NORMAL INTERDENTAL SEPTA

- Radiographic evaluation of bone changes in periodontal diseases is based mainly on →**appearance of interdental septa**, because the relatively dense root structure obscures the facial and lingual bony plates.
- The interdental septum normally presents a thin radiopaque border that is adjacent to the PDL and at alveolar crest, termed as **Lamina dura**.

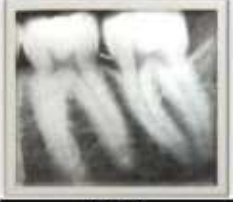


FIGURE-1.
Crest of interdental septum normally parallel to a line drawn between the CEJ of adjacent teeth (arrow). Also note the radiopaque lamina dura around the roots and interdental septum.

- Lamina dura appears radiographically as a **continuous white line**, but in reality it is perforated by
  - ❑ Numerous small foramina
  - ❑ Traversed by blood vessels, lymphatics & nerves, which pass between the PDL & bone.
- Because the lamina dura represents the bone surface lining the tooth socket, the shape and position of the root & changes in the angulation of the X-ray beam produce considerable variations in its appearance

- The angulation of crest of interdental septum is generally parallel to line between the CEJs of aproximating teeth.
- The interdental space and therefore the interdental septum between teeth with prominently convex proximal surfaces are wider antero-posteriorly than those between teeth with relatively flat proximal surfaces.
- The facio-lingual diameter of the bone is related to the width of proximal root surface.
- When there is a difference in the level of the CEJs, the crest of the interdental bone appears angulated rather than horizontal.

DISTORTION PRODUCED BY VARIATIONS IN RADIOGRAPHIC TECHNIQUE

- Variation in technique produce **artifacts that limit the diagnostic value** of the radiograph.

- Bone level
- Pattern of bone destruction
- Width of PDL space, as well as
- Radio-density
- Trabecular pattern

Are modified by altering exposure & development time, type of film & X-ray angulation.

- Marginal contour of interdental septum

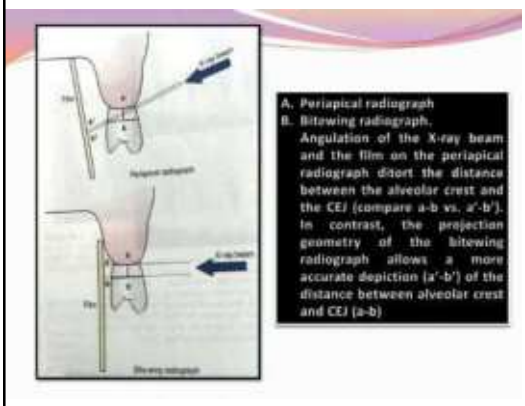
- *Long cone paralleling* technique → projects most realistic image of the level of the alveolar bone.
- *Bisecting of the angle technique* → increase the projection & make the bone margin appear closer to the crown.

- Shifting the cone mesially or distally without changing the horizontal plane projects the X-ray obliquely & change the:
  - Shape of the interdental bone on radiograph
  - Radiographic width of PDL space &
  - Appearance of lamina dura.
  - It also distorts the extent of furcation involvement.

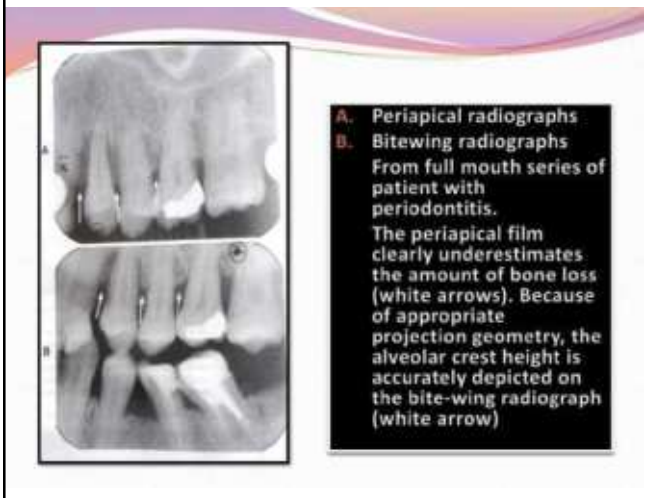
PRICHARD ESTABLISHED FOLLOWING FOUR CRITERIA TO DETERMINE ADEQUATE ANGULATION OF PERIAPICAL RADIOGRAPHS

1. The radiograph should show the tips of molar cusps with little or none of the occlusal surface showing.
2. Enamel caps and pulp chambers should be distinct.
3. Interproximal spaces should be open.
4. Proximal contacts should not overlap unless teeth are out of line anatomically.

- An additional intra-oral projection that can be used for evaluation of alveolar crest is the bitewing projection.
- For bitewing radiographs the film is placed behind the crowns of upper and lower teeth parallel to long axis of the teeth.
- The X-ray beam is directed through the contact areas of the teeth and perpendicular to the film.
- Thus projection geometry of bitewing films allows the evaluation of the relationship between the interproximal alveolar crest and CEJ without distortion.
- If bone loss is severe and bone level cannot be visualized on regular bitewing radiographs → film can be placed vertically to cover larger area of the jaws.



A. Periapical radiograph
B. Bitewing radiograph.
Angulation of the X-ray beam and the film on the periapical radiograph distort the distance between the alveolar crest and the CEJ (compare a-b vs. a'-b'). In contrast, the projection geometry of the bitewing radiograph allows a more accurate depiction (a'-b') of the distance between alveolar crest and CEJ (a-b)



A. Periapical radiographs
B. Bitewing radiographs
From full mouth series of patient with periodontitis.
The periapical film clearly underestimates the amount of bone loss (white arrows). Because of appropriate projection geometry, the alveolar crest height is accurately depicted on the bite-wing radiograph (white arrow)

BONE DESTRUCTION IN PERIODONTAL DISEASES

- The radiograph does not reveal minor destructive changes in bone.
- Therefore, slight radiographic changes in the periodontal tissues mean that "The disease has progressed beyond its earliest stages".
- The earliest signs of periodontal disease must be detected clinically.

BONE LOSS

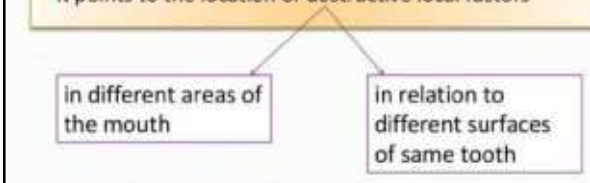
- The radiographic image tends to show less severe bone loss.
- Difference between the alveolar crest height and radiographic appearance **ranges from → 0 – 1.6 mm**, mostly accounted for by X-ray angulation.

Amount of bone loss:

- Radiographs → indirect method for determining the amount of bone loss in periodontal diseases.
- Shows amount of remaining bone rather than amount lost.
- **Amount of bone lost is estimated as =** difference between the physiologic bone loss of the patient and the height of the remaining bone.
- **Distance from CEJ to the alveolar crest:**
  - ✓ In adolescents = 2 mm.
  - ✓ May be greater in older patients.

DISTRIBUTION

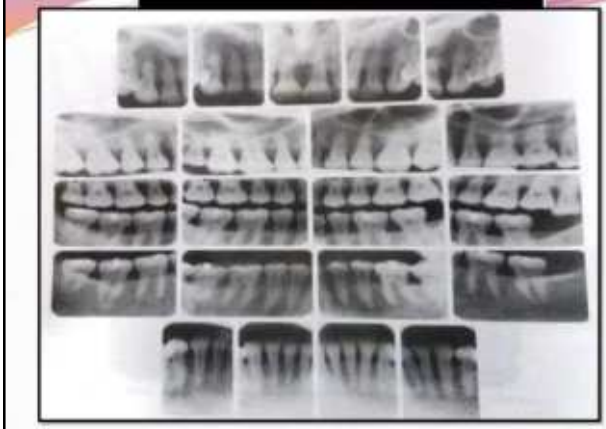
- Distribution of bone loss is an important diagnostic sign.
- It points to the location of destructive local factors

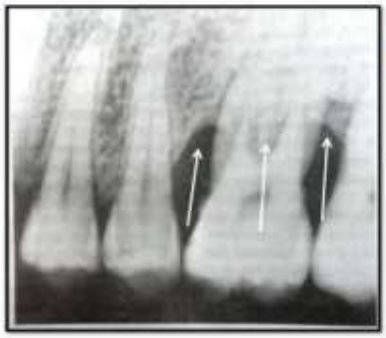


PATTERN OF BONE DESTRUCTION

- In periodontal disease the interdental septa undergo changes that affects:
  - ✓ Lamina dura
  - ✓ Crestal radiodensity,
  - ✓ Size and shape of medullary spaces &
  - ✓ Height & contour of the bone
- **HORIZONTAL BONE LOSS:** The interdental septa may be reduced in height, with the crest horizontal & perpendicular to the long axis of adjacent teeth.
- **VERTICAL/ANGULAR BONE LOSS:** Septa may have angular or arcuate defect.

- Generalized horizontal bone loss





- Angular bone loss on first molar with involvement of the furcation

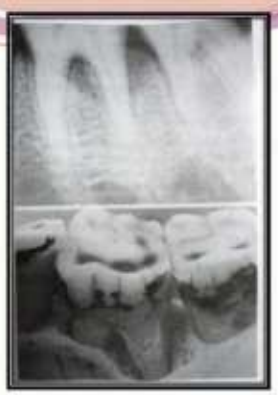
LIMITATIONS OF RADIOGRAPHS

1. Do not indicate the internal morphology or depth of the crater-like interdental defects, which appear as angular or vertical defects.
2. Do not reveals the extent of involvement on the facial & lingual surface.
3. Bone destruction on mesial & distal root surface may be partially hidden by dense mylohyoid ridge.

4. Dense cortical plates on the facial & lingual surface of the interdental space obscure destruction that occur in the intervening cancellous bone.

* Thus it is possible to have deep craters in the bone between facial & lingual plates without radiographic indications of its presence.

- A reduction of only 0.5 – 1.0 mm in the thickness of the cortical plate is sufficient to permit radiographic visualization of destruction of the inner cancellous trabeculae.
- Interdental vertical lesion in the posterior area with thick facial or lingual bone may not be isolated in interdental area.
- But it may continue facially & lingually to form a trough like defect that cannot be seen radiographically.
- These lesion may terminate on the radicular surface or may communicate with the adjacent interdental area to form one continuous lesion.



Interdental lesion that extends to the facial or lingual surfaces in a troughlike manner

- Figure on right shows - Two adjacent lesion connecting on the radicular surface to form one inter-connecting osseous lesion.
- Although radiopaque pointer placed in radicular defects will demonstrate the extent of bone loss.

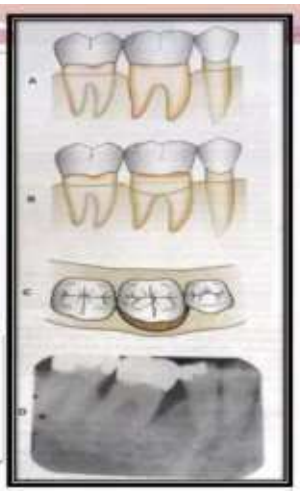


Figure:
A. Interdental mesial & distal lesions
B. Facial or lingual outlines of actual lesion
C. Occlusal view of lesion
D. Actual radiograph of mesial & facial lesions

- Gutta percha packed around the teeth increases the usefullness of radiograph, for detecting the morphologic changes of osseous craters & involvement of facial & lingual surfaces.

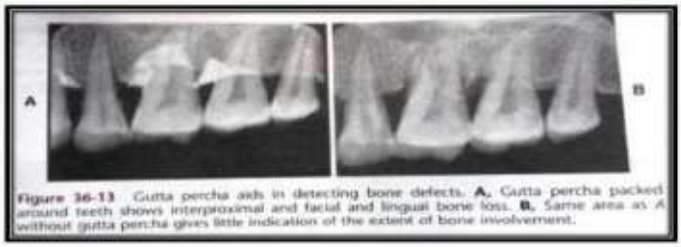


Figure 36-13 Gutta percha aids in detecting bone defects. **A,** Gutta percha packed around teeth shows interproximal and facial and lingual bone loss. **B,** Same area as A without gutta percha gives little indication of the extent of bone involvement.

RADIOGRAPHIC CHANGES IN PERIODONTITIS

Sequence of radiographic changes in periodontitis & the tissue changes that produce them :

1. **Earliest radiographic changes in periodontitis:**
   Fuzziness + a break in the continuity of the lamina dura at the mesial or distal aspect of crest of interdental septum.

   - These result from extension of gingival inflammation into the bone →causing widening of the vessel channels and a reduction in calcified tissue at the septal margin.

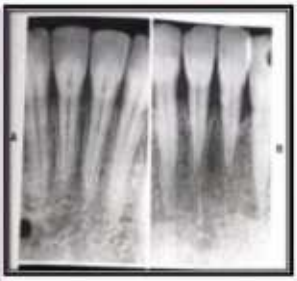


Figure: A. normal appearance
B. Fuzziness +break in continuity of lamina dura at the crest of bone distal to the central incisor (left).

2. A wedge shaped radiolucent area is formed at the mesial or distal aspect of crest of septal bone.

- The apex of the area is pointed in the direction of the root.
- This is produced by resorption of the bone of the lateral aspect of the interdental septum, with an associated widening of the periodontal space.

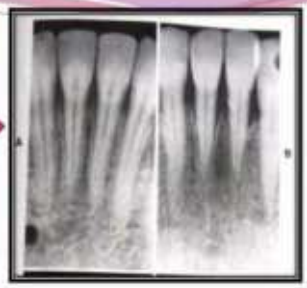


Figure: A. normal appearance
B. Fuzziness +break in continuity of lamina dura at the crest of bone distal to the central incisor (left). There are wedge shaped radiolucent areas at the crest of the other interdental septa.

3. The destructive process extends across the crest of the interdental septum & height is reduced.

- Fingerlike projections extend from the crest into the septum.
- The radiolucent projections into the septum are the result of deeper extension of inflammation into the bone.
- Inflammatory cells & fluid, proliferation of connective tissue cells, and increased osteoclasis caused increased bone resorption along the endosteal margins of the medullary spaces.

4. Height of the interdental septum if progressively reduced by the extension of inflammation and the resorption of bone. (figure D)

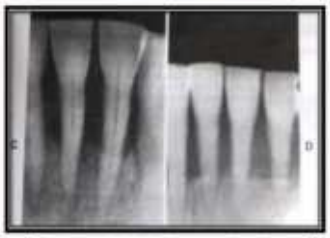


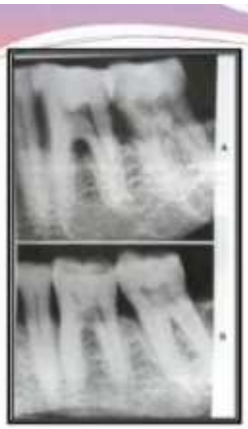
Figure: C. Radiolucent projections from the crest into the interdental septum indicate extension of destructive process
D. Severe bone loss.

RADIOGRAPHIC APPEARANCE OF INTERDENTAL CRATERS

- Interdental craters are seen as irregular areas of reduced radiography on alveolar bone crests.
- They are not sharply demarcated from the rest of the bone, with they bend gradually.
- **Radiographs do not accurately depict** the morphology or depth of interdental craters, which sometimes appear as vertical defects.

RADIOGRAPHIC APPEARANCE OF FURCATION INVOLVEMENT

- Definitive diagnosis of furcation involvement is made by clinical examination.
- This is diagnosed by specially designed probe called as Nabers probe.
- As a general rule bone loss is always greater than it appears in the radiograph.
- Variations in the radiographic technique may obscure the presence and extent of furcation involvement.
- A tooth may present marked bifurcation involvement in one film but appear to tbe uninvolved in another .



A. Furcation involvement indicated by triangular radiolucency in bifurcation area of mandibular first molar. The second molar presents only a slight thickening of the periodontal space in the bifurcation area.
B. Same area as A, different angulation. The triangular radiolucency in the bifurcation of the first molar is obliterated nd involvement of the second molar bifurcation is apparent

TO ASSIST IN THE RADIOGRAPHIC DETECTION OF FURCATION INVOLVEMENT FOLLOWING CRITERIA ARE SUGGESTED

1. The slightest radiographic change is the furcation area should be investigated. Clinically, if there is bone loss an adjacent tooth.
2. Diminished radiodensity in the furcation area in which outline of bony trabeculae are visible, suggests furcation involvement.

3. When there is marked bone loss in relation to a single molar root, it may be assumed that furcation is also involved.

Furcation involvement of the first molar, associated with bone loss on the distal root

Typical radiographic appearance of periodontal abscess on right central incisor

RADIOGRAPHIC APPEARANCE OF THE PERIODONTAL ABSCESS

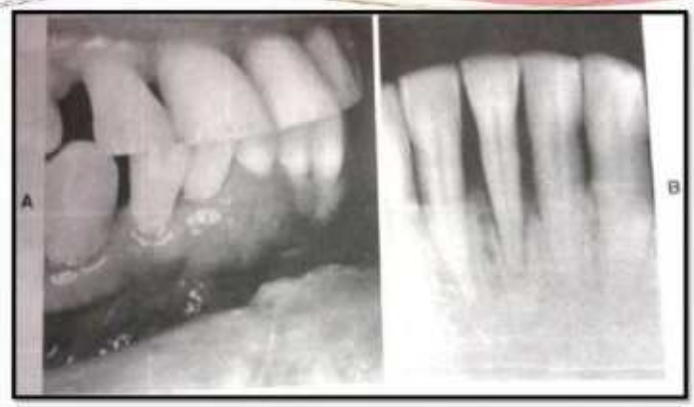
- The typical radiographic appearance of the periodontal abscess is the discrete area of radiolucency along the lateral aspect of the root.
- The radiographic picture is often not typical because of many variable such as following:

1. The stage of the lesion – in the early stages the acute periodontal abscess is extremely painful but presents no radiographic changes.
2. The extent of bone destruction & morphologic changes of the bone.
3. The location of the abscess.
   - Lesion in the soft tissue wall of a periodontal pocket are less likely to produce radiographic changes than those deep in the supporting tissues.
   - Abscess on the facial & lingual surface are obscured by the radiopacity of the root; interproximal lesions are more likely to be visualized radiographically.

- The radiograph alone cannot be relied on for the diagnosis of periodontal abscess.

Radiolucent area on lateral aspect of root with chronic periodontal abscess.

Typical radiographic appearance of periodontal abscess on right central incisor.

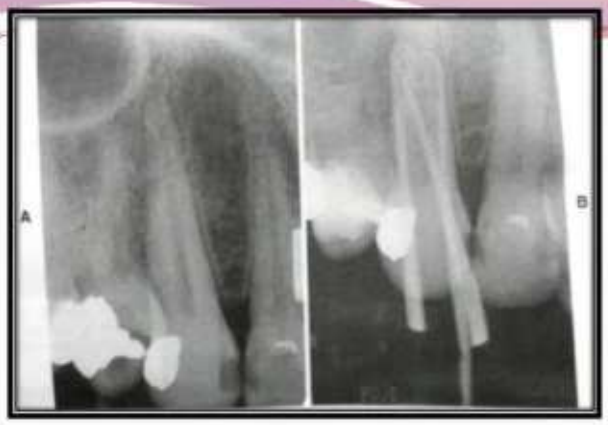


Chronic periodontal abscess

A. Periodontal abscess in the right central and lateral incisor area
B. Extensive bone destruction and thickening of the

CLINICAL PROBING

- Regenerative and resective flap designs and incisions require prior knowledge of the underlying osseous topography.
- Careful probing of these pocket areas after scaling and root planing often require local anesthesia and definitive radiographic evaluation of the osseous lesions.
- Radiographs taken with periodontal probes or other indicators (eg. Hirschfeld pointers) placed into the anesthetized pocket show the true extent of the bone lesion.
- As indicated previously, the attachment level on the radicular surface or interdental lesions with thick facial or lingual bone cannot be visualized in the radiograph.
- The use of radiopaque indicators is an efficient diagnostic aid for the clinician to better visualize every aspect of the defect.

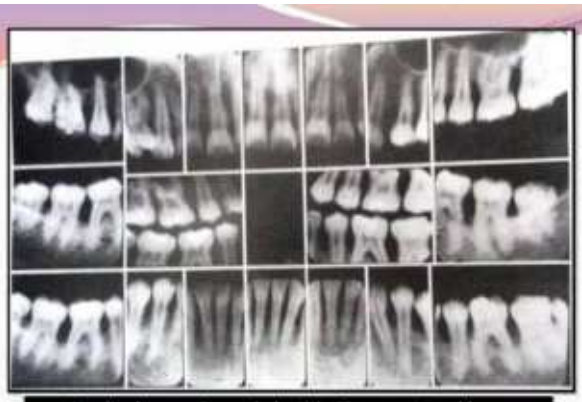


A. Radiograph of maxillary cuspid. This view does not show facial bone loss.
B. Radiograph of same maxillary cuspid as A, with guttapercha points placed in the facial pocket to indicate

RADIOGRAPHIC APPEARANCE OF THE LOCALIZED AGGRESSIVE PERIODONTITIS

- Localized aggressive (formerly "localized juvenile") periodontitis is characterised by combination of the following radiographic features:

1. Bone loss may occur initially in the maxillary and mandibular incisor and/or first molar areas, usually bilaterally, and results in vertical, arclike destructive patterns.
2. Loss of alveolar bone may become generalised as the disease progresses but remains less pronounced in the premolar areas.



Localized aggressive periodontitis. The accentuated bone destruction in the anterior and first molar areas is considered to be characteristic of this disease.

RADIOGRAPHIC APPEARANCE OF THE TRAUMA FROM OCCLUSION

- Trauma from occlusion can produce radiographically detectable changes in the lamina dura, morphology of the alveolar crest, width of PDL space, and density of the surrounding cancellous bone.
- Traumatic lesions manifest more clearly in faciolingual aspects, because mesiodistally the tooth has the added stability provided by the contact areas with adjacent teeth.
- Therefore, slight variations in the proximal surfaces may indicate greater changes in the facial and lingual aspects.
- The radiographic changes listed next are not pathognomonic of trauma from occlusion and must be interepreted in combination with clinical findings, particularly tooth mobility, presence of wear facets, pocket depth, and analysis of occlusal contacts and habits.

INJURY PHASE OF TFO:

- Produce a loss of lamin dura that may be noted in apices, furcations, and marginal areas, this loss of lamina dura results in widening of the PDL space.
- This change, particularly when incipient or circumscribed, may easily be confused with technical variations caused by X-ray angulation or malposition of the tooth; it can be diagnosed with certainty only in radiographs of the highest quality.

Widened PDL space caused by TFO. Note the increased density of surrounding bone formation in response to increased occlusal forces.

REPAIR PHASE OF TFO:

- Results in an attempt to strengthen the periodontal structures to better support that increased loads.
- Radiographycally, this is manifested bya widening of PDL space, which may be generalised or localized.
- Although microscopic measurements have determined that normal variations exist in the width of PDL space in the different regions of root, these are generally not detected in radiographs.
- When variations in width between the marginal area and midroot or between the midroot and apex are detected, it means that the tooth is being subjected to increased forces. Successful attempts to reinforce the periodontal structures by widening of the PDL space is accompanied by increased width of the lamina dura and sometimes by condensation of the perialveolar cancellous bone.
- More advanced traumatic lesions may result in deep angular bone loss, which combined with marginal inflammation, may lead to intrabony pocket formation. In terminal stages these lesions extend around the root apex, producing a wide, radiolucent periapical image (cavernous lesions)
- Root resorption may also result excessive forces on the periodontium, particularly those caused by orthodontic appliances.
- Although trauma from occlusion produces many areas of root resorption, these areas are usually of a magnitude insufficient to be detected radiographically.

ADDITIONAL RADIOGRAPHIC CRITERIA

- The following diagnostic criteria can be used as further aids in the radiographic identification of periodontal disease:

1. **RADIOPAQUE HORIZONTAL LINE ACROSS THE ROOTS:** This line demarcates the portion of the root where the labial or lingual bony plate has been partially or completely destroyed from the remaining bone-supported portion.

Figure 36-26

2. **VESSEL CANALS IN THE ALVEOLAR BONE:** Hirschfeld described linear and circular radiolucent areas produced by interdental canals radiolucent areas produced by interdental canals and their foramina respectively. These canals indicate the course of the vascular supply of the bone and are normal radiographic findings. The radiographic image of the canals is often so prominent, particularly in the anterior region of the mandible, that they might be confused with radiolucency resulting from periodontal disease.

3. **DIFFERENTIATION BETWEEN TREATED & UNTREATED PERIODONTAL DISEASE:** It is sometimes necessary to determine whether the reduced bone level is the result of periodontal disease that is no longer destructive (usually after treatment and proper maintenance) or whether destructive periodontal disease is present.

Prominent vessel canals in the mandible

SKELETAL DISTURBANCES MANIFESTED IN THE JAWS

- Skeletal disturbances may produce changes in the jaws that effect the interpretation of radiographs from the periodontal perspective.
- Destruction of tooth-supporting bone may occur in various diseases.

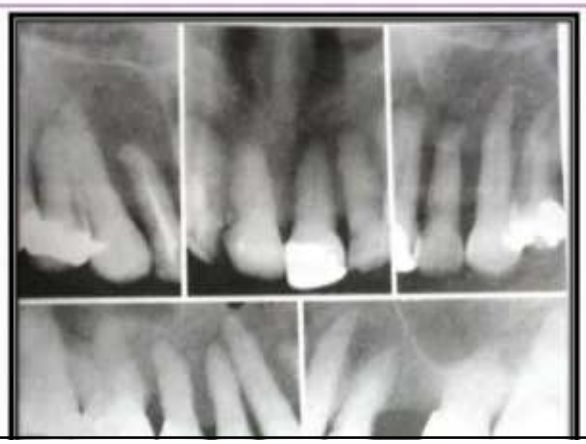
OSTEITIS FIBROSA CYCTICA/ RECKLINGHAUSEN'S DISEASE OF BONE

- Developed in advanced primary or secondary hyperparathyroidism and causes osteoclastic resorption of bone with fibrous replacement and hemorrhage with hemosiderin deposition, creating a mass known as brown tumor.
- The disease result in a diffuse granular mottling, scattered cyst like radiolucent areas throughout the jaws, and a generalized disappearance of lamina dura.

Fig 17.6

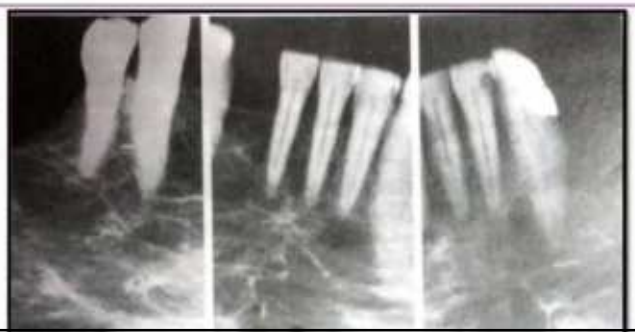
PAGET'S DISEASE

- The normal trabecular pattern is replaced by hazy, diffuse meshwork of closely knit, fine trabecular markings, with the lamina dura absent, or scattered radiolucent areas may contain irregularly shaped radiopaque zones.



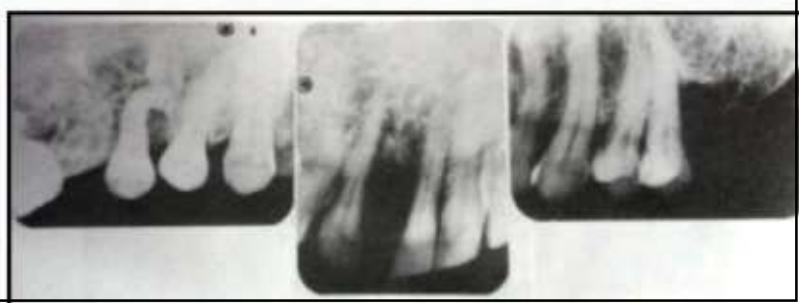
FIBROUS DYSPLASIA

- Appear as a small radiolucent area at a root apex or as an extensive radiolucent area with irregularly arranged trabecula markings.
- The cancellous spaces may be enlarged, with distortion of the normal trabecular pattern (ground glass pattern) and obliteration of the lamina dura.



LANGERHANS CELL HISTIOCYTOSIS

- Results from disturbances in immunoregulation, and its different forms comprise the diseases formerly called Hand-Schuller-Christia disease, Letterer-Siwe disease, Gaucher's disease, and eosinophilic granuloma.
- Appear as single or multiple radiolucent areas, which may be unrelated to the teeth or may entail destruction of the tooth supporting bone.



MULTIPLE MYELOMA

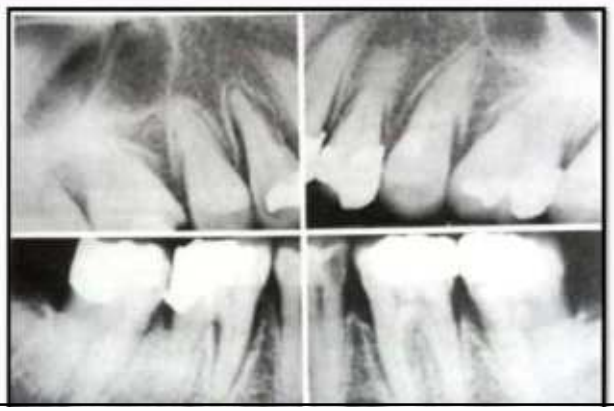
- Numerous radiolucent areas occur when the jaws are involved by multiple myeloma.

OSTEOPETROSIS (MARBLE-BONE DISEASE, ALBERS-SCHONBERG DISEASE)

- Outlines of root are obscured by diffuse radiopacity of the jaws.
- In less severe cases, the increased density is confined to the bone in relation to the nutrient canals and lamina dura.

SCLERODERMA

- The PDL is uniformly widened at the expense of the surrounding alveolar bone.



MALIGNANCY

- Both primary & metastatic, can affect the alveolar ridge and often presents as periodontal disease.
- A uniform widening of the PDL can be an early sign of osteosarcoma
- Irregular destruction of the periodontal bone without tooth displacement is frequently the result of squamous cell carcinoma or metastatic carcinoma.

Advanced Diagnostic Aids Lec4

Dr Mohammed Ibraheem Hazeem

CONTENTS

INTRODUCTION

DIAGNOSIS

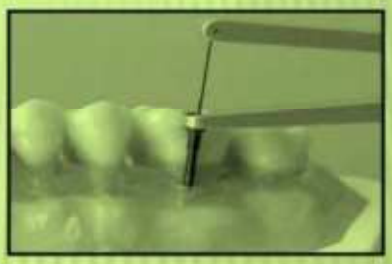
- WHO definition –it is the art of chronological organization and critical evaluation of the information obtained of patients history , lab investigations, clinical examination so as to identify the disease type and etiology.
- Greek word –
  - Dia =through
  - Gnosis =to know

- Proper diagnosis → rational treatment and preventive strategy
- Diagnosis Involves
  - Analysis of case history
  - Evaluation of clinical signs and symptoms
  - Results of tests (Probing, Mobility, Radiograph, blood test, biopsies)
- Diagnosis Determines
  - Presence of disease
  - Type of disease
  - Underlying disease process

PERIODONTAL DIAGNOSTIC TESTS

- Clinical methods
- Radiographic methods
- Microbial analysis
- Host response

ADVANCES IN CLINICAL DIAGNOSIS

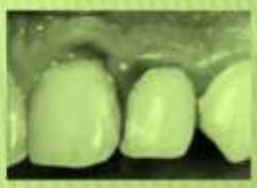


ADVANCES IN CLINICAL DIAGNOSIS

- Periodontal probes
- Non-Periodontal probes
  - Calculus detection system
  - Periodontal Disease Evaluation System
  - Gingival Temperature
  - Gingival bleeding
  - Tooth mobility

GINGIVAL BLEEDING

- Assessment of
  - Redness
  - Swelling
  - Gingival bleeding



- Inflamed periodontal tissues bleed when probed with a blunt instrument because there are frequently microulcerations in the epithelium that lines the soft tissue wall of a periodontal pocket.

- Gingival bleeding
  - Sensitive clinical indicator of early inflammation
  - More objective than change in colour
  - Severity is proportional to the size of inflammatory infiltrate
- **Lang et. al. 1986** → force > 0.25 newton evokes bleeding in sites with intact periodontium
- BOP is a good risk indicator of disease activity but not a good predictor

- It has also been shown that 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 severity of bleeding increases with an increase in size of the inflammatory infiltrate (Greenstein, Caton, Polson).

GINGIVAL TEMPERATURE

Inflamed tissues are usually warmer than core body temperature, because of

- Increased blood flow and a very high metabolic rate
- Haffajee et al in 1992 reported on a site basis, increased mean subgingival temperatures have also been associated with deeper probing depths and greater levels of clinical attachment loss. 2 different rationales support these relationships:
- Endotoxins of the infecting bacteria, especially the LPs of gram –ve organisms are exogenous pyrogens that stimulate macrophages to release endogenous pyrogens producing fever (Bencsics et al,1995)
- Bacteria respond to changes in environmental temperature with changes in their growth rate, metabolic activities and expression of virulence factors (Maurelli et al, 1989)

↑ Diseased sites

Posterior teeth

Mandibular sites

- Temp increases with probing depth
- **PerioTemp probe (Abiodent)**- sensitivity of 0.1° C
- 2 light indicating diodes:
- Red-emitting diode→ higher temp
- Green-emitting diode→ lower temp

PERIODONTAL PROBES

- Orban as the "*eye of the operator beneath the gingival margin*"
- Latin word "*Probo*", which means "*to test*".
- Gold standard
- **Simonton (1925) and Box (1928)** were among the first to advocate the routine use of calibrated probes
- locate calculus, measure gingival recession, width of attached gingiva and size of intraoral lesions, identify tooth and soft-tissue anomalies, locate and measure furcation involvements and determine mucogingival relationships and bleeding tendencies.

TYPES OF PROBE:

- **Pihlstrom (1992)** classified probes into three generations.
- **In 2000, Watts** extended this classification by adding fourth- and fifth-generation probes.

- *First-Generation (Conventional) Probes:*
- conventional hand-held instruments.
- Probes do not control for probing pressure and are not suited for automatic data collection.

1. **Willams' Periodontal probe:** 1936, Charles H.M. Williams

- Prototype/benchmark

2. Community Periodontal Index of Treatment Need (CPITN):

- Professor George S. Beagrie & Jukka Ainamo 1978
- Measurement of pocket depth
- Detection of subgingival calculus and other overhangs
- CPITN → 3.5mm,5.5mm,8.5mm & 11.5mm
- 5gm wt, ball tip 0.5 mm

3. **University of Michigan O probe:**

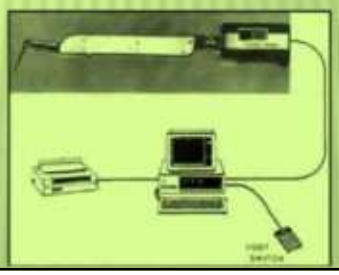
- 3mm, 6mm & 8mm

4. **University of North Carolina-15 (UNC-15):**

5. **Naber's probe:**

- Furcal areas

- **Gibbs et al. (1988)** developed the **Florida Probe® system** (Florida Probe Corp, Gainesville, FL):
- constant probing force, precise electronic measurement to 0.1 mm and computer storage of the data and sterilization of all system parts entering or close to the mouth

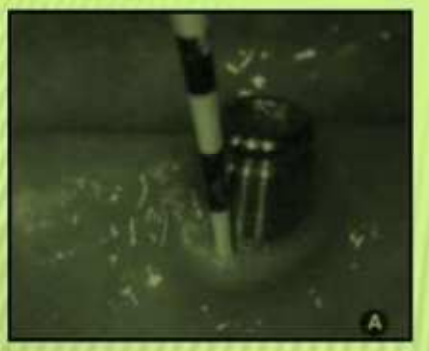


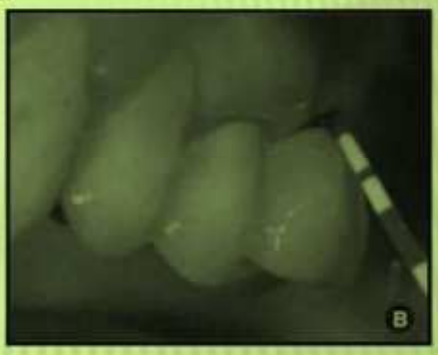
- **Birek et al. (1981) and McCulloch et al. (1981)** developed the **Toronto Automated probe**.
- It used the occlusal/ incisal surface to measure relative clinical attachment levels.
- **Goodson and Kondon (1988)** used fiber optic technology in their controlled-force ***Accutek probe***.
- The **InterProbe™** (The Dental Probe Inc, Glen Allen, VA), also known as the ***Perio Probe***, is a third-generation probe with a flexible probe tip, Jeffcoat 1991

- ***Fifth-Generation Probes:***
- 3D and non-invasive; an ultrasound or other device is added to a fourth-generation probe.
- aim to identify the attachment level without penetrating it.
- The only fifth-generation probe available, the ***Ultrasonographic (US) probe*** (Visual Programs, Inc, Glen Allen, VA), uses ultrasound waves to detect, image and map the upper boundary of the periodontal ligament and its variation over time as an indicator of the presence of periodontal disease.
- ***Hinders & Companion*** at the NASA Langley Research Center.

Probing around Implants :

To prevent scratching of the implant surface, and also of the implant abutments, Plastic Periodontal probes should be used instead of the normal steel probes





Scratchings act as niches for plaque accumulation

Plastic Periodontal probes

The Hu- Friedy black and yellow color coded replaceable plastic periodontal probe tip.

Reusable plastic perio probe

TOOTH MOBILITY

Schulte in collaboration with Siemens company developed an instrument designed to measure the mobility of the implants and natural teeth. This device rapidly percusses the tooth (16 times, 4 times a second) and then electronically records the rebound alteration pattern. The degree of attenuation (scale ranges from -8 to +50) is recorded digitally and acoustically then scaled into 4 degrees of tooth mobility.

- Goodson (1988) confirmed the correlation between PTV and clinical mobility index (MI). • The greater the alveolar bone height, the lower the periotest value.

Fig. 19. Control unit for Periotest device which audibly and visually indicates tooth mobility

-8 to +9 : clinically firm tooth
10-19 : palpable mobility
20-29 : visible mobility
30-50 : mobility in response to lip and tongue movements

Fig. 20. Periotest handpiece being applied to anterior teeth

II. ADVANCES IN MICROBIOLOGIC ANALYSIS

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

Bacterial plaque plays a primary role in the initiation and progression of periodontal diseases.

- A no. of assays have been developed for their detection and relative quantification in patient plaque samples.
- It should be known that these assays themselves are not diagnostic for periodontal diseases. They indicate that the presence of these organisms can increase a subjects risk for periodontal attachment loss.

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 10^3 is difficult to detect
Time consuming
Expensive equipment and experienced personnel

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.

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, 8000.

Periotron 8000

Perio Paper Strips

- 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.

- 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

CONCLUSION

- Although there are many clinical, radiographic and microbial techniques for determining the disease activity and progression, there is still a lack of a proven gold standard of disease progression.
- After all these years of intensive research we still lack a proven diagnostic test that has demonstrated high predictive value for disease progression, has a proven impact on disease incidence and prevalence and is simple, safe and cost effective.

REFERENCES

- **Newman MG, Takei HH, Klokkevold PR, Carranza FA**. 10th edition. Carranza's Clinical Periodontology. *Saunders Company 2006. 579-601.*
- **Ramachandra SS, Mehta DS, Sandesh N, Baliga V, Amarnath** J. Periodontal Probing Systems. A Review of Available Equipment. *Dentistry India 2009; 3(3). 2-10.*

REFERENCES

- Diagnostics: Perio 2000 vol 34
- Critical issues in diagnosis: Perio 2000 vol 39
- Comprehensive Dental Hygiene Care (4th Edition) –Irene R. Woodall
- Clinical Practice of Dental Hygiene–Wilkins
- Current Concepts in Periodontics –Varma and Nayak

PERIODONTAL RESPONSE TO EXTERNAL FORCES

MAGNITUDE *is increased*

- *thickening of the periodontal ligament*
- *increase in the number and width of the periodontal ligament fibers*
- *increase in the density of Al.bone*

changing the **DIRECTION** *causes*

- *Re-orientation of stresses and strains within the periodontium*
- *Lateral [horizontal] forces and torque[rotational] forces are more likely to injure the periodontium*

DURATION *AND* **FREQUENCY**

Constant pressure on the bone is more injurious than intermittent forces

TRAUMA FROM OCCLUSION

DEFINITION

When the occlusal forces exceed the adaptive capacity of the tissues, tissue injury results which is termed as

"TRAUMA FROM OCCLUSION"

Trauma from occlusion refers to the

TISSUE INJURY, NOT THE OCCLUSAL FORCE

An occlusion that produces such injury is

"TRAUMATIC OCCLUSION"

ACUTE

CHRONIC

ACUTE

ABRUPT CHANGE IN OCCLUSAL FORCE

such as biting on a

- HARD OBJECT
- RESTORATIONS *or* PROSTHETIC APPLIANCES

that interfere with direction of occlusal forces

resulting in TOOTH PAIN

SENSITIVITY TO PERCUSSION

INCREASED TOOTH MOBILITY

CHRONIC

MORE COMMON

develops from gradual changes in occlusion produced by :

- TOOTH WEAR
- DRIFTING MOVEMENT
- EXTRUSION OF TEETH *combined with parafunctional habits such as* BRUXISM *and*

CLENCHING

"MALOCCLUSION IS NOT NECESSARY TO PRODUCE TRAUMA"

Periodontal injury may occur when the occlusion appears normal

The dentition may be anatomically and esthetically acceptable but functionally injurious

Such traumatic occlusal relationships are referred as:

OCCLUSAL DISHARMONY
FUNCTIONAL IMBALANCE
OCCLUSAL DYSTROPHY

PRIMARY

SECONDARY

- *insertion of a "high filling"*
- *prosthetic replacement that creates excessive forces on abutment and antagonist teeth*
- *drifting movement or extrusion of teeth into spaces created by unreplaced missing teeth*
- *orthodontic movement of teeth into functionally unacceptable positions*

SECONDARY

When the adaptive capacity of the tissues to withstand occlusal forces is impaired by bone loss

Tissue response to increased occlusal forces occurs in 3 stages:

INJURY

REPAIR

ADAPTIVE REMODELLING

STAGE I : INJURY

SLIGHTLY EXCESSIVE PRESSURE *stimulates*

- *resorption of Al.bone, with resultant widening of the pdl space*
- *blood vessels are numerous and reduced in size*

- SLIGHTLY EXCESSIVE TENSION *causes*
- *elongation of the pdl fibers*
- *formation of Al.bone*
- *blood vessels are enlarged*

GREATER PRESSURE *produces a series of changes in the periodontal ligam*

- *within 30 mnts*
  - *retardation and stasis of blood flow occurs*
- *at 2-3hrs*
  - *blood vessels are packed with erythrocytes, which start to fragment*
- *within 7 days*
  - *disintegration of blood vessels and release of contents into the surrounding tissue*

SEVERE TENSION *causes :*

- *widening of pdl*
- *thrombosis*
- *hemorrhage*
- *tearing of pdl*
- *resorption of Al. bone*

The areas of periodontium most susceptable to injury from excessive occlusal forces are the "furcations"

STAGE II: **REPAIR**

Repair is constantly occurring in periodontium

When bone is resorbed by excessive occlusal forces, the body attempts to reinforce the thinned bony trabeculae with new bone

This attempt to compensate for lost bone is called "buttressing bone formation"

BUTTRESSING BONE FORMATION
occurs

within the jaw -- **CENTRAL BUTTRESSING**

on the bone surface - **PERIPHERAL BUTTRESSING**

STAGE III: ADAPTIVE REMODELING

When the repair process cannot keep pace with the destruction caused by the occlusion, the periodontium is remodeled by which the forces are no longer injurious to the tissues
This results in:

- *thickened pdl*
- *angular defects*
- *mobility of teeth*

After adaptive remodeling of the periodontium, resorption and formation return to normal

The marginal gingiva is unaffected by TFO because its blood supply is sufficient to maintain even when the vessels of the pdl are obliterated by excessive occlusal forces

RADIOLOGICAL SIGNS

- WIDENING OF PDL SPACE *often with thickening of lamina dura*
- "VERTICAL" *destruction of interdental septum*
- RADIOLUCENCE *and* CONDENSATION *of Al.bone*
- ROOT RESORPTION

CONCLUSION

Trauma from occlusion

DOES NOT INTIATE

GINGIVITIS

or

PERIODONTAL POCKETS

but it may affect the progress and severity of periodontal pockets started by local irritation

PATHOLOGICAL MIGRATION

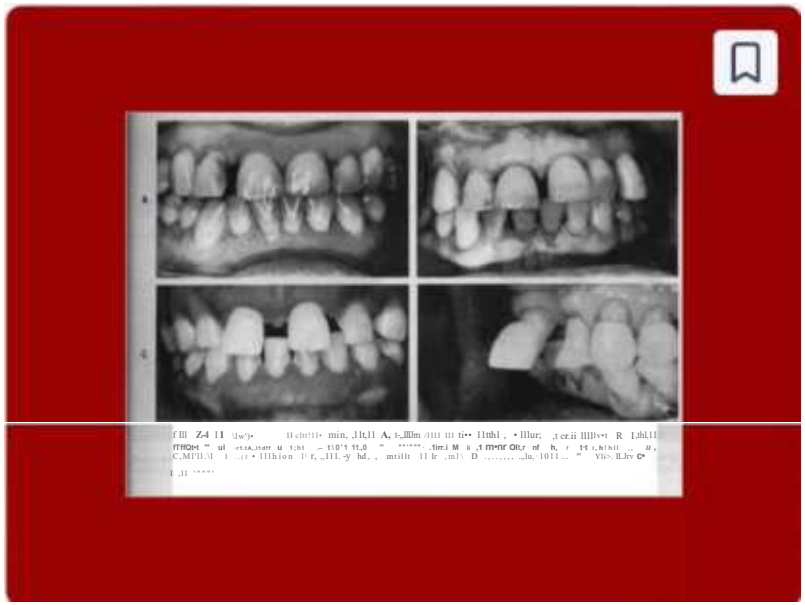
- *Occurs frequently in the* ANTERIOR REGION
- *The teeth move in* ANY DIRECTION
- *Accompanied by* MOBILITY *and* ROTATION
- *Pathological migration in the* OCCLUSAL *or* LINGUAL DIRECTION *is termed as* "EXTRUSION"

PATHOGENESI

TWO major factors play a role in maintaining the normal position of the teeth;

1. The health and normal height of periodontium

2. The forces exerted on the teeth such as
   -- the forces of occlusion
   -- pressure from the lips, cheeks, and tongue



WEAKENED PERIODONTAL SUPPORT

The tooth with weakened support is unable to maintain its normal position in the arch and moves away from the opposing force

The force that moves the weakly supported tooth may be created by factors such as

- *-- occlusal contacts*
- *-- pressure from the tongue*

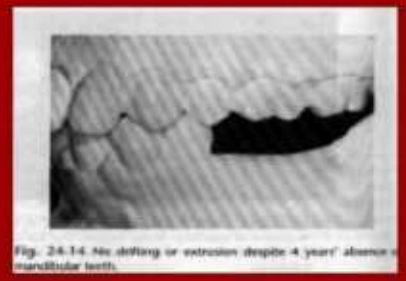


Fig. 24-14 No drifting or extrusion despite 4 years' absence of mandibular teeth.

fi 24 13 11.1a IMy t1ri1 i l f tilted .and o t r u d c d l m o t cu•..ated by a nit\\ n g mMICl hul•r 100th

FAILURE TO REPLACE FIRST MOLAR I

RESULTS IN:

i. The second and the third molars tilt, resulting in a decrease in vertical dimension

ii. The premolars move distally, and mandibular incisors tilt or drift lingually

iii. Anterior overbite is increased

iv. The maxillary incisors are pushed labially and laterally

v. The anterior teeth extrude

vi. Diastema is created by the separation of anterior teeth

The disturbed proximal contact relationships lead to :

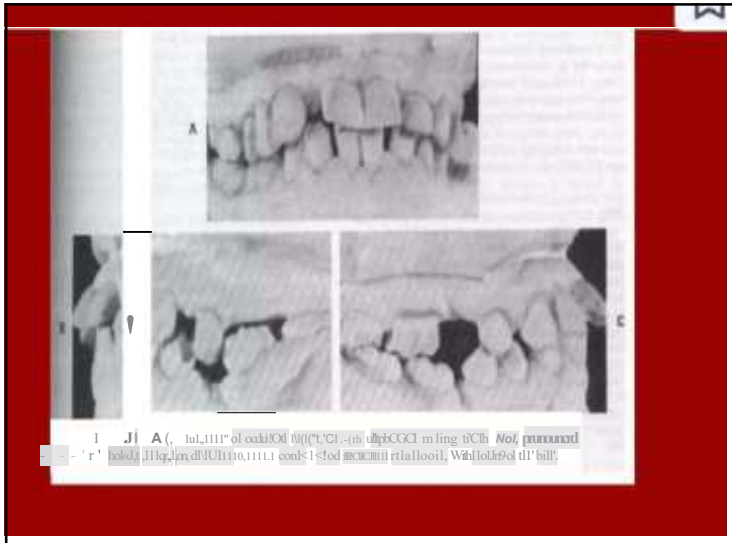
-- food impaction

-- gingival inflammation

-- pocket formation

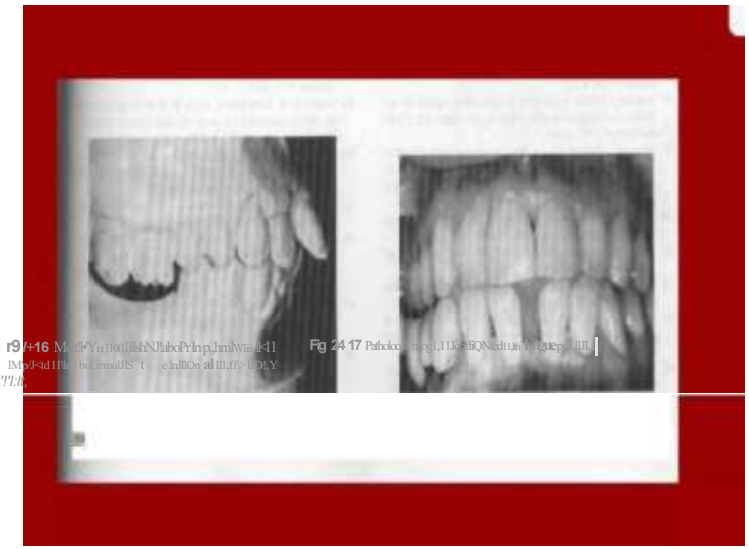
-- bone loss and

-- tooth mobility

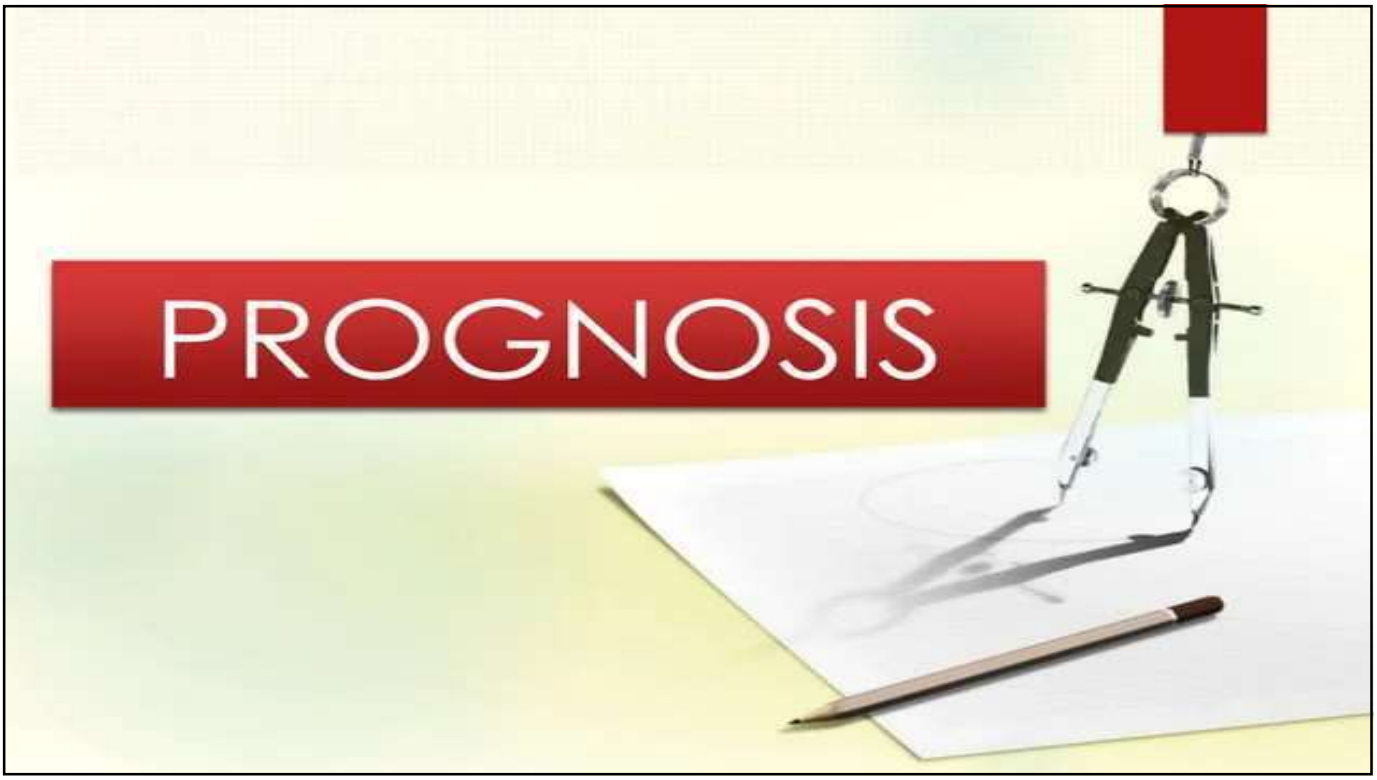


OTHER CAUSES

- *Trauma from occlusion*
- *Pressure from the tongue*
- *Pressure from the granulation tissue*




THERE IS NOTHING M
ATTRACTIVE THAN CONFIDEN
Good Morning
SANTABANTA.COM


PROGNOSIS

CONTENTS

Prognosis is the prediction of the probable course, duration, and outcome of a disease based on a general knowledge of the pathogenesis of the disease and the presence of risk factors for the disease.

Goodman et al

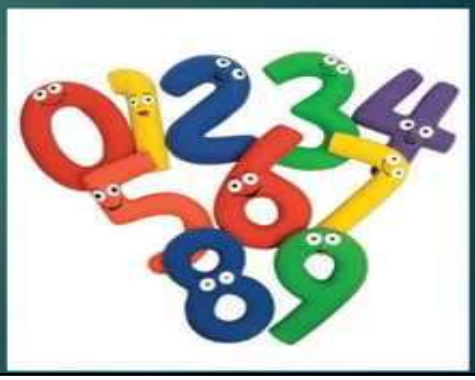
- Made before treatment plan is established
- Based on:
  - Specific information about disease
  - Previous experience

- **Confused with risk**
- **Risk : Likelihood that an individual will get a disease in a specified period**

DETERMINATION OF PROGNOSIS:

1> Excellent
2> Good
3> Fair
4> Poor
5> Questionable
6> Hopeless

(Mc Guire et al 1991)



EXCELLENT

- No bone loss
- Excellent gingival condition
- Good patient cooperation
- No systemic / environmental factors

GOOD

- Adequate remaining bone support
- Adequate possibilities to control etiologic factors and establish a maintainable dentition
- Adequate patient cooperation
- No systemic / environmental factors or if present well controlled

FAIR

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

POOR

- Moderate to advanced bone loss
- Tooth mobility
- Grade I and II furcation involvement
- Difficult to maintain areas
- Doubtful patient cooperation
- Presence of systemic / environmental factors

QUESTIONABLE

- Advanced bone loss
- Grade II and III furcation involvements
- Tooth mobility
- Inaccessible areas
- Presence of systemic / environmental factors

HOPELESS

- Advanced bone loss
- Non-maintainable areas
- Extractions indicated
- Uncontrolled systemic / environmental conditions

OVERALL CLINICAL FACTORS

1.PATIENT AGE

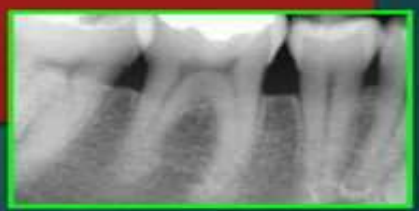
- Comparable CT attachment and alveolar bone – prognosis better for older
- Younger patient – shorter time – more periodontal destruction

2. DISEASE SEVERITY

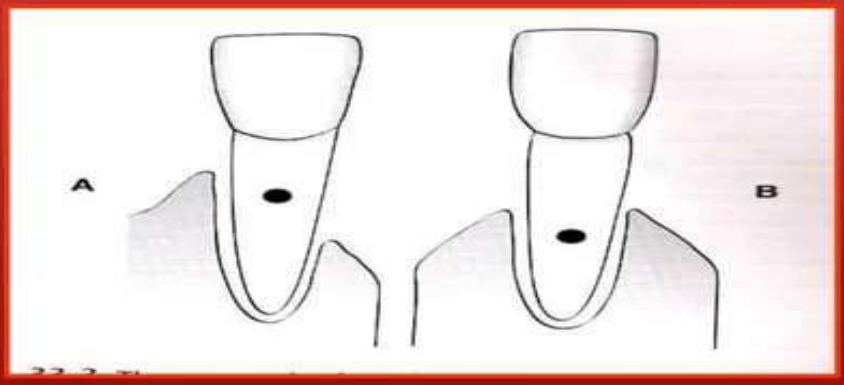
Determination of :

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

- Prognosis for horizontal bone loss depends on the height of the existing bone.
- Angular defects - if the contour of the existing bone & the number of osseous walls are favorable, there is an excellent chance that therapy could regenerate bone to approximately the level of the alveolar crest.



- When greater bone loss has occurred on one surface of a tooth, the bone height on the less involved surfaces should be taken into consideration when determining the prognosis.



3. PLAQUE CONTROL

- Bacterial plaque - primary etiologic factor associated with periodontal disease.
- Effective removal of plaque on a daily basis by patient.

4. PATIENT COMPLIANCE & COOPERATION

- Refuse to accept the patient for treatment
- Extract teeth with hopeless or poor prognosis and perform scaling and root planing on remaining teeth

SYSTEMIC/ ENVIRONMENTAL FACTORS

1.SMOKING

- Direct relationship - smoking and the prevalence and incidence of periodontitis
- Affects severity
- Affects healing
- Slight to moderate periodontitis - fair to poor
- Severe periodontitis - poor to hopeless

2. SYSTEMIC DISEASE/ CONDITION

- Prevalence and severity of periodontitis - significantly higher - type I and II diabetes
- Prognosis dependent on patient compliance relative to both dental and medical status
- Well controlled patients - slight to moderate periodontitis - good prognosis

4. GENETIC FACTORS

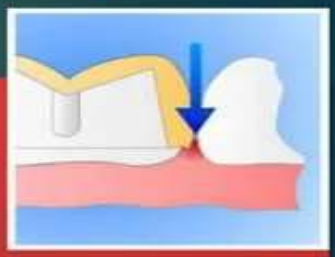
- Genetic polymorphism in IL-1 genes resulting in overproduction of IL-1β - associated with significant increase in risk for severe, generalized, chronic periodontitis.
- Genetic factors also influence serum IgG2 antibody titers and the expression of Fc-γRII receptors on the neutrophil - significant in aggressive periodontitis.

LOCAL FACTORS

1.PLAQUE AND CALCULUS

- Bacterial plaque and calculus - most important local factor in periodontal diseases.
- Good prognosis- depends on ability of patient and clinician to remove etiological factor.

2. SUBGINGIVAL RESTORATIONS



Contribute to

- Increased plaque accumulation
- Increased inflammation
- Increased bone loss

Subgingival margins - poor prognosis.

3.ANATOMIC FACTORS

- Short, tapered roots with large crowns, cervical enamel projections (ceps) and enamel pearls, intermediate bifurcation ridges, root concavities, and developmental grooves - predispose periodontium to disease
- Teeth with short, tapered roots and relatively large crown – Poor prognosis

4.TOOTH MOBILITY

Principal causes-

- Loss of alveolar bone — Non correctable
- Inflammatory changes in the periodontal ligament
- Trauma from occlusion.

Correctable

- stabilization by use of splinting - beneficial impact on the overall and individual tooth prognosis.

Prosthetic/Restorative Factors

The overall prognosis requires a general consideration of bone levels and attachment levels to establish whether enough teeth can be saved either to provide a functional and aesthetic dentition or to serve as abutments for a useful prosthetic replacement of the missing teeth.

When few teeth remain, the prosthodontic needs become more important, and sometimes periodontally treatable teeth may have to be extracted if they are not compatible with the design of the prosthesis.

Caries, Non-vital Teeth & Root Resorption.

- For teeth mutilated by extensive caries, the feasibility of adequate restoration and endodontic therapy should be considered before undertaking periodontal treatment.
- Extensive idiopathic root resorption or root resorption that has occurred as a result of orthodontic therapy, risks the stability of teeth and adversely affects the response to periodontal treatment.

RELATIONSHIP BETWEEN DIAGNOSIS AND PROGNOSIS

Factors such as patient age, severity of disease, genetic susceptibility, and presence of systemic disease are important in developing both diagnosis as well as prognosis.

PROGNOSIS FOR PATIENTS WITH GINGIVAL DISEASE

I. DENTAL PLAQUE INDUCED GINGIVAL DISEASES

a) **Gingivitis Associated With Dental Plaque Only-**

- **Reversible**
- **Prognosis - good provided all local irritants are eliminated & patient cooperates by maintaining good oral hygeine.**

b) Plaque induced gingival diseases modified by systemic factors

The inflammatory response to bacterial plaque can be influenced by systemic factors, such as endocrine related changes associated with puberty, pregnancy and diabetes.

Long term prognosis depends - control of bacterial plaque along with correction of the systemic factors.

c) Plaque induced gingival disease modified by medications

- Drug induced gingival enlargement often seen with phenytoin, cyclosporin, nifedipine and in oral contraceptive associated gingivitis.

- Plaque control alone does not prevent the development of lesions, and surgical intervention is usually necessary to correct the alteration of gingival contours.

d) Gingival diseases modified by malnutrition

- Exception - vitamin C deficiency (gingival inflammation and bleeding on probing independent of plaque levels present)
- Prognosis of these patients depend upon the severity and duration of the deficiency and on the likelihood of reversing the deficiency through dietary supplements.

II. Non plaque induced gingival lesions

- Seen in patients with a variety of bacterial, fungal and viral infections.
- Dermatologic disorders such as lichen planus, pemphigoid, pemphigus vulgaris, erythema multiforme, and lupus erythematosus can also manifest in oral cavity as atypical gingivitis.
- Allergic, toxic, and foreign body reactions, as well as mechanical and thermal trauma, can result in gingival lesions.

PROGNOSIS OF PATIENTS WITH PERIODONTITIS

Chronic periodontitis

- In cases where clinical attachment loss and bone loss are not very advanced (slight to moderate periodontitis) - prognosis - good.
- The inflammation - controlled through good oral hygiene and the removal of local plaque retentive factors.

AGGRESSIVE PERIODONTITIS

Poor prognosis

Localized aggressive periodontitis –

- Occurs around puberty
- Localized to first molars and incisors
- Patient exhibits strong serum antibody response to the infecting agent contributing to localization of lesions.

Diagnosed early - can be treated conservatively with oral hygiene instruction and systemic antibiotic therapy - excellent prognosis.

Advanced diseases, prognosis can be good if the lesions are treated with debridement, local and systemic antibiotics, and regenerative therapy

- Generalized form – fair, poor or questionable prognosis due to generalized interproximal loss, poor antibody response and thus poor response to conventional periodontal therapy.

PERIODONTITIS AS A MANIFESTATION OF SYSTEMIC DISEASES

- It can be divided into two categories:
  - periodontitis associated with hematologic disorders such as leukemia and acquired neutropenia.
  - periodontitis associated with genetic disorders such as familial and cyclic neutropenia, down syndrome and hypophosphatasia.
- Primary etiologic factor - bacterial plaque
- Systemic diseases affect the progression of disease and thus prognosis.

NECROTIZING PERIODONTAL DISEASES

- Necrotizing ulcerative gingivitis (NUG)
- Necrotizing ulcerative periodontitis (NUP).

In NUG - primary predisposing factor - bacterial plaque.

Disease - complicated by presence of secondary factors such as acute psychological stress, tobacco smoking, poor nutrition leading to immunosuppression.

- With control of both bacterial plaque and secondary factors prognosis (NUG) - good although tissue destruction is not reversible.
- NUP is similar to that of NUG, except the necrosis extends from the gingiva into the periodontal ligament and alveolar bone.
- Many patients presenting with NUP are immunocompromised through systemic conditions, such as HIV infection.

REEVALUATION OF PROGNOSIS AFTER PHASE I THERAPY

Reduction in pocket depth and inflammation after Phase I therapy indicates a favorable response to treatment and may suggest a better prognosis than previously assumed.

- If the inflammatory changes not controlled or reduced by phase I therapy- overall prognosis - unfavorable.
- In these patients the prognosis can be directly related to the severity of inflammation.

CONCLUSION

Prognosis help us in planning the customized treatment for each patient thus help in providing overall care to patient. So it should be given due importance in general clinical practice

REFERENCES

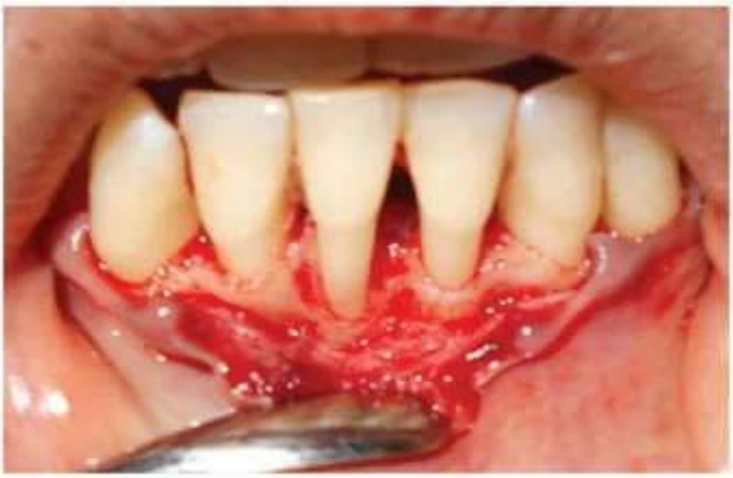
- Carranza's Clinical Periodontology 10th Edition.
- Lindhe- 5th edition
- Hart TC,Kornman KS. Genetic factors in pathogenesis of periodontitis. Periodontol 2000 1997;14:202


THANK YOU

Perio-Surgery

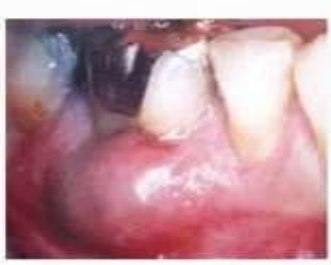
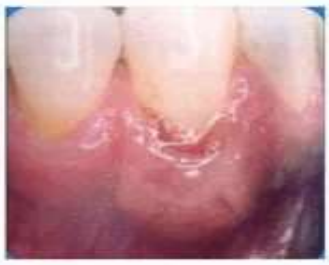
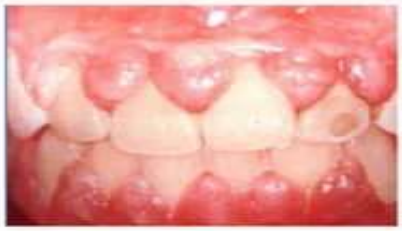
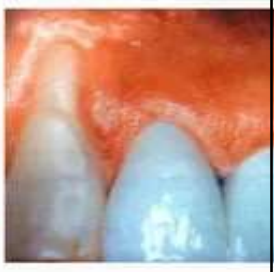
Perio-surgery

- An operative procedure used to treat disease or repair abnormalities in the tissue of the teeth and surrounding areas.

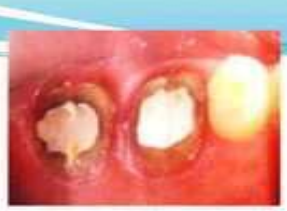
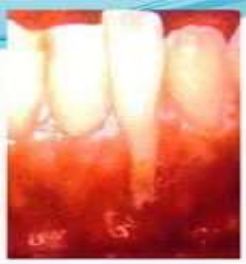
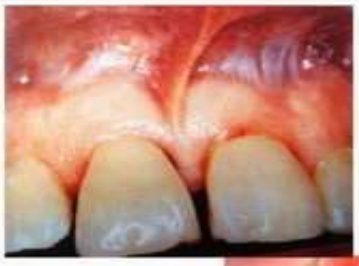
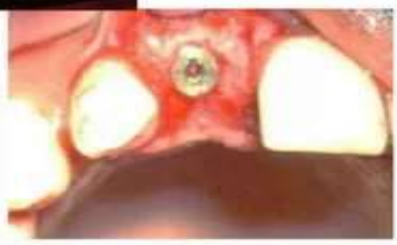


Objectives

- To eliminate all etiologic factors
- Reduction of pocket depth
- Elimination in gingival inflammation
- Establishing periodontal /gingival abccess drainage
- Prevent the recurrence of disease

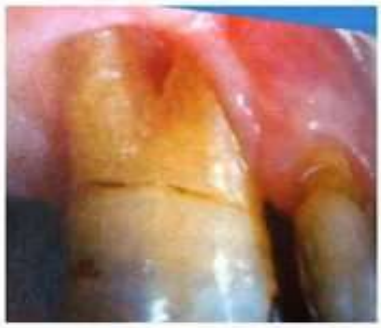





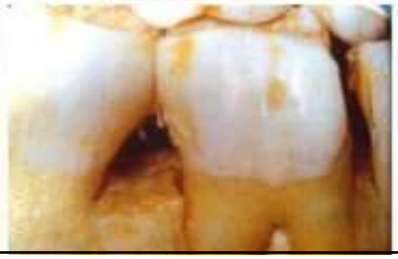
- Prevent further advancement of disease
- Create an oral environment conducive to plaque control
- Pre-prosthodontic treatment
- For osseous regenerative and guided tissue regeneration
- Aesthetic improvement (hyperplasia/recession of gingiva)

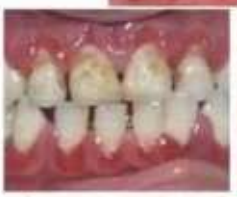
Indication

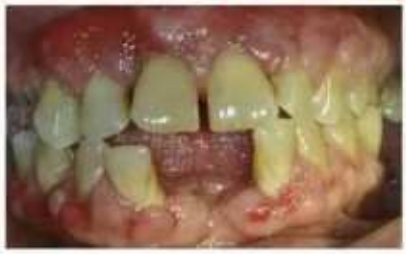
- Deep pocket when complete removal of root irritant is not possible, especially in inaccessible areas like incisors and premolars.
- In cases of grade II and III involvement.
- Persistent inflammation in areas with moderate and deep pocket.



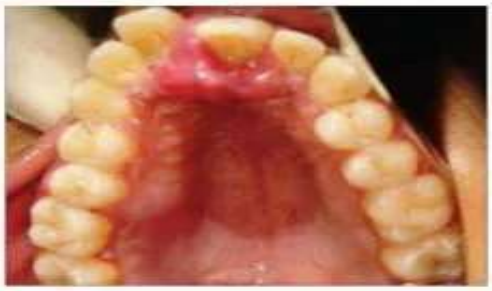


- Areas with irregular bony contours, deep craters, and other defects usually require surgical approach.
- Intrabony pockets on distal areas of last molars, frequently complicated by mucogingival problems, are usually unresponsive to nonsurgical methods.
- Impaired access for Scaling and root planning





- Presence of root fissures, root concavities, furcations and defective margins of restorations in the subgingival area
- Correction of gross gingival aberrations
- Impaired access for the self-performed plaque control
- To facilitate proper restorative therapy
- Loose teeth
- Pain on chewing
- halitosis



Contraindication

- All condition that could be considered contraindications for oral surgery concerning the age of the patient and general health problems, such as sever cardiovascular disease, hypertension, uncontrolled DM, Leukemia, bleeding disorders , hormonal and metabolic disorders tuberculosis etc.

- In patient of advanced age where teeth may last for life without resulting to radical treatment.
- When patient's motivation is in adequate.
- In the presence of infarction.
- When the prognosis is so poor that tooth loss is inevitable

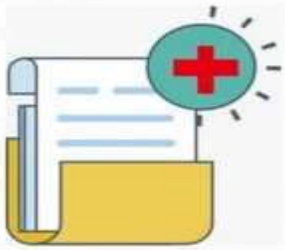
- Where thorough subgingival scaling and good home care will resolve or control the lesion.
- In the presence of infection.
- Smoking habit

Procedures

- Pre-operative evaluation
- Asepsis
- Anesthesia/sedation
- Adequate access
- Arrest of hemorrhage

- Tissue management
- Debridement/scaling root planning
- Closure of wound
- Management of post operative pain
- Post operative instruction

Pre-operative evaluation



- The patient should be prepared medically psychologically and practically for all aspects of intervention.(allergies etc..)
- All surgical procedures should be carefully planned and explained to the patient.
- Medical and dental history should be evaluated
- Premedication should be coordinated for medically compromised and patient w/ anxiety
- Cessation of tobacco product usage

Asepsis

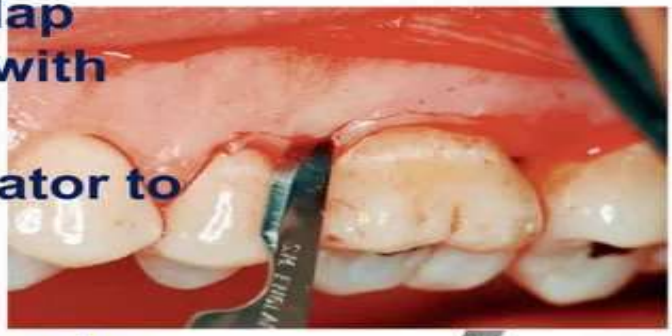
- PPE is worn w/ head cap and eye protector
- Sterilization of instruments
- Oral prophylaxis to surgical site
- Antiseptic mouthwash
- Asepsis of surrounding tissues
- No cross contamination
- Changing of gloves and face mask

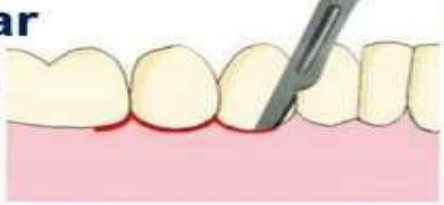
Painless surgery

- Use of correct anesthetic solution
- By administering proper anesthetic technique for Maxillary (ASA, MSA, PSA, Maxillary Nerve Block)
- For Mandibular (IAN, Buccal Nerve Block)
- Follow the correct maximum dosage

Adequate access

- By doing envelop flap with full thickness with blade #15
- Use periosteal elevator to deflect tissue
- Irrigate and arrest hemorrhage to have clear view in the working area





- Incision Technique
  - Horizontal Incision
    - **The internal bevel incision**
- Is basic to most periodontal flap procedures.
- It is the incision from which the flap is reflected to expose the underlying bone and root
- This incision has also been termed the first incision because it is the initial incision in the reflection of a periodontal flap, and the reverse bevel incision because its bevel is in reverse direction from that of the gingivectomy incision.

Arrest hemorrhage

- By applying digital pressure
- Burnishing the bone
- Using gel foam
- Using electro cuttery
- Bite on moist gauze
- Use of hemostat/suture for bigger blood vessels
- Continuous suction

Importance of haemostasis

1. Accurate visualization of the extent of the disease, pattern of bone destruction, anatomy and condition of roots
2. Provides clear view for debridement
3. Prevents excess loss of blood from the body

Tissue Management

- Operate gently and carefully
- Observe patient at all times
- Proper instrumentation
- Sharp instruments
- Don't blow air in the field of surgery (causes surface desiccation, emphysema and air emboli)

Debridement

- By using curettage and irrigate (with antiseptic solution and NSS)

- removal of deflected soft and hard tissue and calcular deposite. (use ultrasonic scaler/ manual scaler), clean also the root suface/s.

- irrigate with oral betadine or other irrigation solution.

Closure of wound

- By suturing

Technique: simple interrupted technique

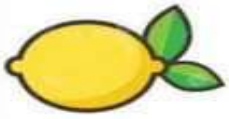
Post operative care

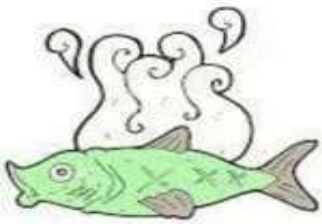
- Take medication like analgesic as needed to relieve the pain, and antibiotics to prevent infection(don't take aspirin)

- Apply ice, intermittently for alternative 20 minutes on and 20 minutes 0ff, on the face over the operated side on first day to reduce the swelling.

- Don't vigorously brush on the operated area use 12% chlorhexidine as a mouth rinse twice a day.

- Swelling is usual in extensive surgical procedure. It subsides in 3 or 4 days. Apply moist heat if it persists.
- Avoid hot and hard food for the first 24 hrs.
- Avoid alcohol, citrus, spicy and pungent foods
- No smoking



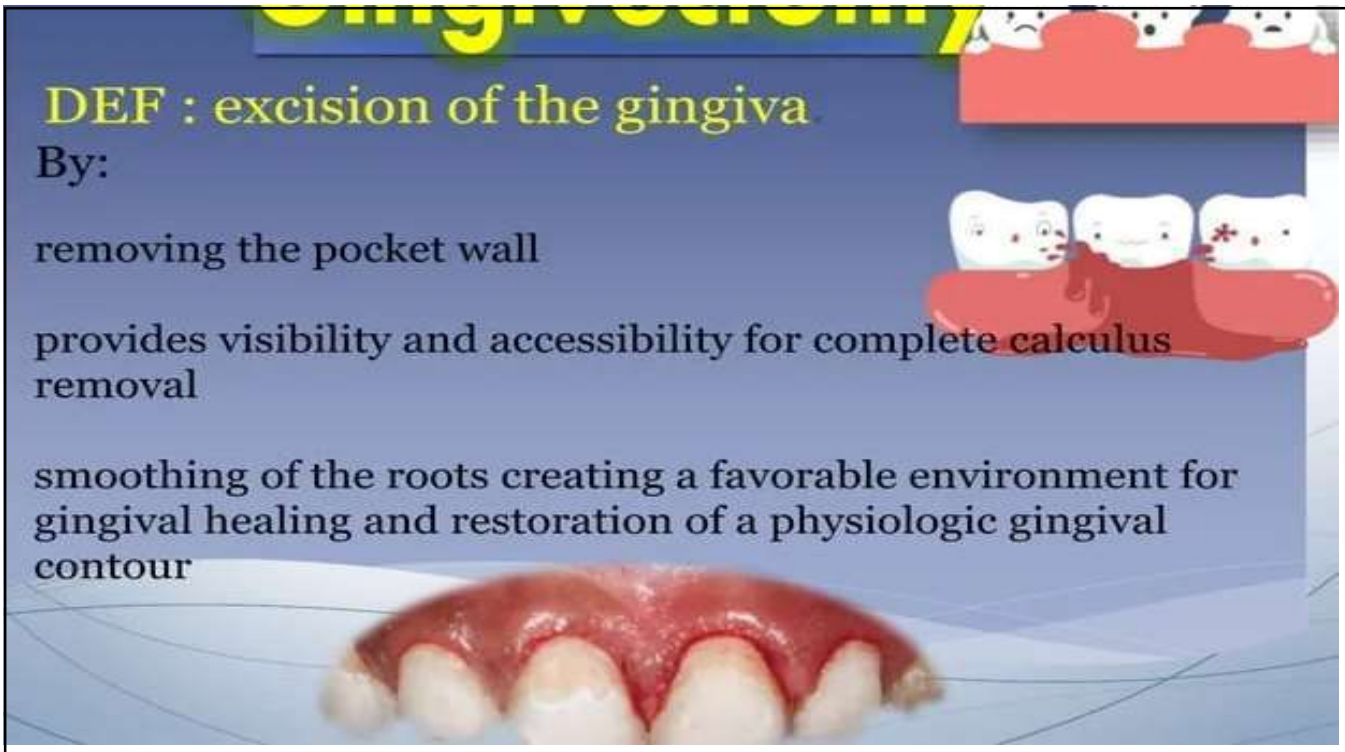


- Do not brush over pack.
- Avoid exertion.
- Do not try to stop bleeding by rinsing.
- Soft Diet.
- Chew on the non-operated side.

MEDICATION:

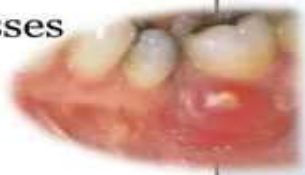
- Analgesics are used to relief the pain
- Rinse with 0.12% CHX for 3 days.
- Vitamin C supplementation 1000mg a day
- Amoxicillin 500mg every 8 hour for 7 days


Gingivectomy


DEF : excision of the gingiva
By:
removing the pocket wall
provides visibility and accessibility for complete calculus removal
smoothing of the roots creating a favorable environment for gingival healing and restoration of a physiologic gingival contour

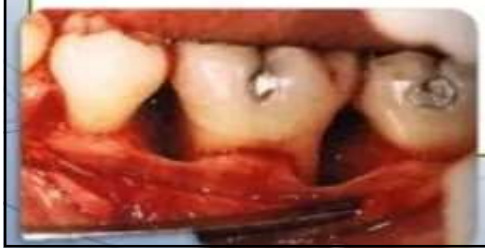
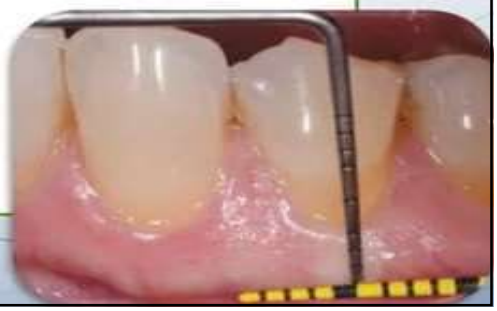
INDICATIONS

- Elimination of suprabony pockets
- Elimination of gingival enlargements
- Elimination of suprabony periodontal abscesses
- Crown lengthening
- Perio aesthetic


Contraindications

- bone defect can not be corrected
- the base of the pocket is apical to the mucogingival junction
- Esthetic considerations

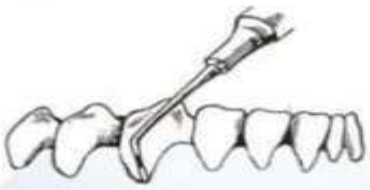



Types of gingivectomy

- SURGICAL GINGIVECTOMY
- GINGIVECTOMY by ELECTROSURGERY
- GINGIVECTOMY BY CHEMOSURGERY
- LASER GINGIVECTOMY

SURGICAL GINGIVECTOMY

Steps of surgical procedures:



Pocket of each surface is explored by periodontal probe and marked by pocket marker each pocket is marked in several area

Crane-Kaplan Pocket Marker

Steps of surgical procedures:

The incision started apical to the points marked course of pocket and directed coronally to apoint between base of pocket and crest of bone

Incision beveled e 45 degree to tooth surface as failure of beveling lead to broad fibrous plateau

It should be close to bone without exposing it..but if occur should cover by periodontal pack

Kirkland knife used for incision on facial & lingual surface
orban knife for interdental incision

Steps of surgical procedures:

Remove exist pocket wall, clean area . granulation tissue may be seen on excited soft tissue

Granulation tissue is curetted out and remove any remaining

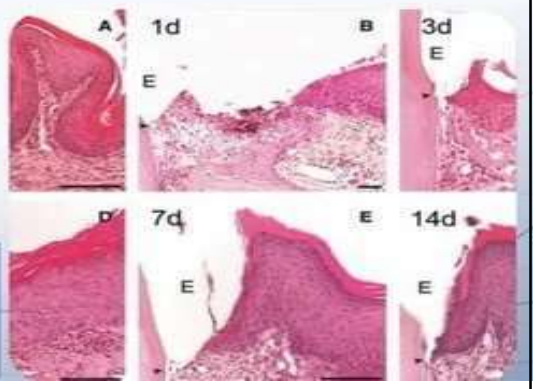
Cover the area with surgical pack.

Healing after surgical Gingivectomy

- The initial response is the formation of a protective surface blood clot , the underlying tissue becomes acutely inflamed with some necrosis
- The clot is then replaced by granulation tissue , by 24 hours there is an increase in new connective tissue cells mainly angioblasts.
- By the third day numerous young fibroblasts are located in the area
- The highly vascular granulation tissue grows coronally creating a new free gingival margin and sulcus.

Healing after surgical Gingivectomy

- During the first 2 weeks
  - granulation tissue forms within the clot
  - the epithelium forms the wound edge & migrates over this granulation tissue
- From about 10 days to about 30 days
  - organization of C.T
  - keratinization of epithelium



NOTE :
The time required for complete healing varies depending on the area of the cut surface and interference from local irritation and infection .

GINGIVECTOMY BY ELECTROSURGERY

Advantages

Permits an adequate contouring of the tissue and controls hemorrhage

Disadvantages

1) Cannot be used in patients with Cardiac pacemakers.
2) unpleasant odor .
3) damage can be done .
4) The heat generated by injudicious use can cause tissue damage.

GINGIVECTOMY BY ELECTROSURGERY

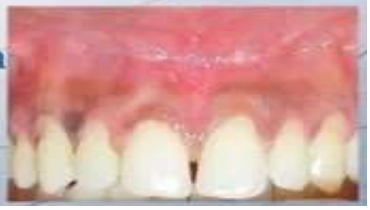
Indication (limited to superficial procedures)
- Removal of gingival enlargement
- Gingivoplasty
- Relocation of frenum
- muscle attachments

Contraindication
It should not be used for procedures that involve proximity to the bone such as flap operations or mucogingival surgery

Technique

* For removal of **gingival enlargements** and **Gingivoplasty** we use the needle electrode supplemented by the small ovoid loop for festooning , A blended cutting and coagulating current is used .

* **Frenum** and **muscle attachments** can be relocated to facilitate pocket elimination using a loop current .



LASER GINGIVECTOMY

The lasers most commonly used in dentistry are the carbon dioxide and the Nd:YAG .

The CO_2 laser beam

used for the excision of gingival growths although

healing is delayed when compared with healing after the conventional scalpel gingivectomy .

precautions to avoid reflecting the beam on instrument surfaces which could reflect into neighboring tissues and the eye of the operator.

It is **not recommended** to use laser for periodontal surgery as it is not supported by research .

GINGIVECTOMY BY CHEMOSURGERY

* Techniques to remove the gingiva using chemicals such as 5% paraformaldehyde or potassium hydroxide have been described in the past but are not currently used.

Gingivoplasty:

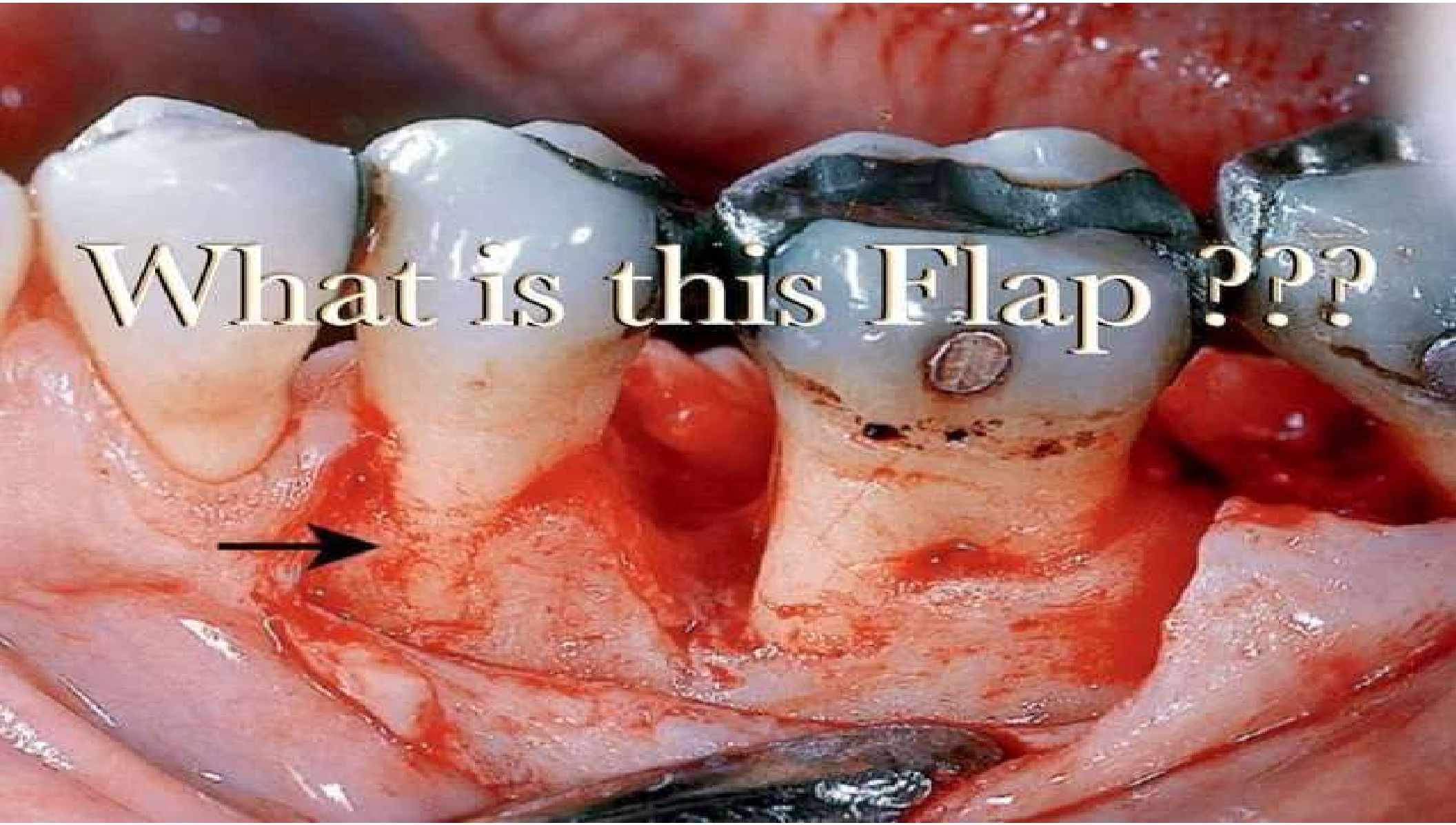
- is a reshaping of the gingiva to create physiologic gingival contours.

Indications:

- Gingival clefts and craters, shelflike interdental papillae caused by acute necrotizing ulcerative gingivitis.
- may be done with a periodontal knife, a scalpel, or electrosurgery.

Gingivectomy	Gingivoplasty
• Excision of the soft wall of p.p • It is performed to eleminate p.p • It includes reshaping of the gingiva as a part of the technique.	• Reshaping the gingiva to create a normal function form. • It is done with the sole purpose of recontouring the gingiva in the absence of p.p

The Periodontal Flap


What is this Flap ???

Definition

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

INDICATIONS

1. Irregular bony contours.
2. Deep craters.
3. Grade II or III furcation involvement.
4. Root resection / hemisection.
5. Intrabony pockets.
6. Persistent inflammation in areas with moderate to deep pockets.

CONTRAINDICATIONS

1. Uncontrolled medical conditions such as
    1. Un-stable angina
    2. Un-controlled diabetes
    3. Un-controlled hypertension
    4. Myocardial infarction / stroke within 6 months
2. Poor plaque control
3. High caries rate
4. Unrealistic patient expectations or desires

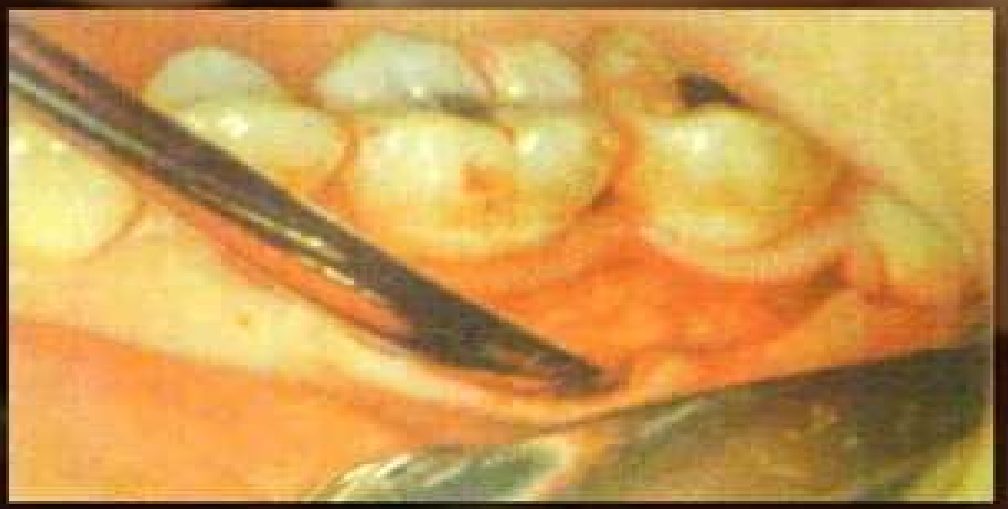

Classification

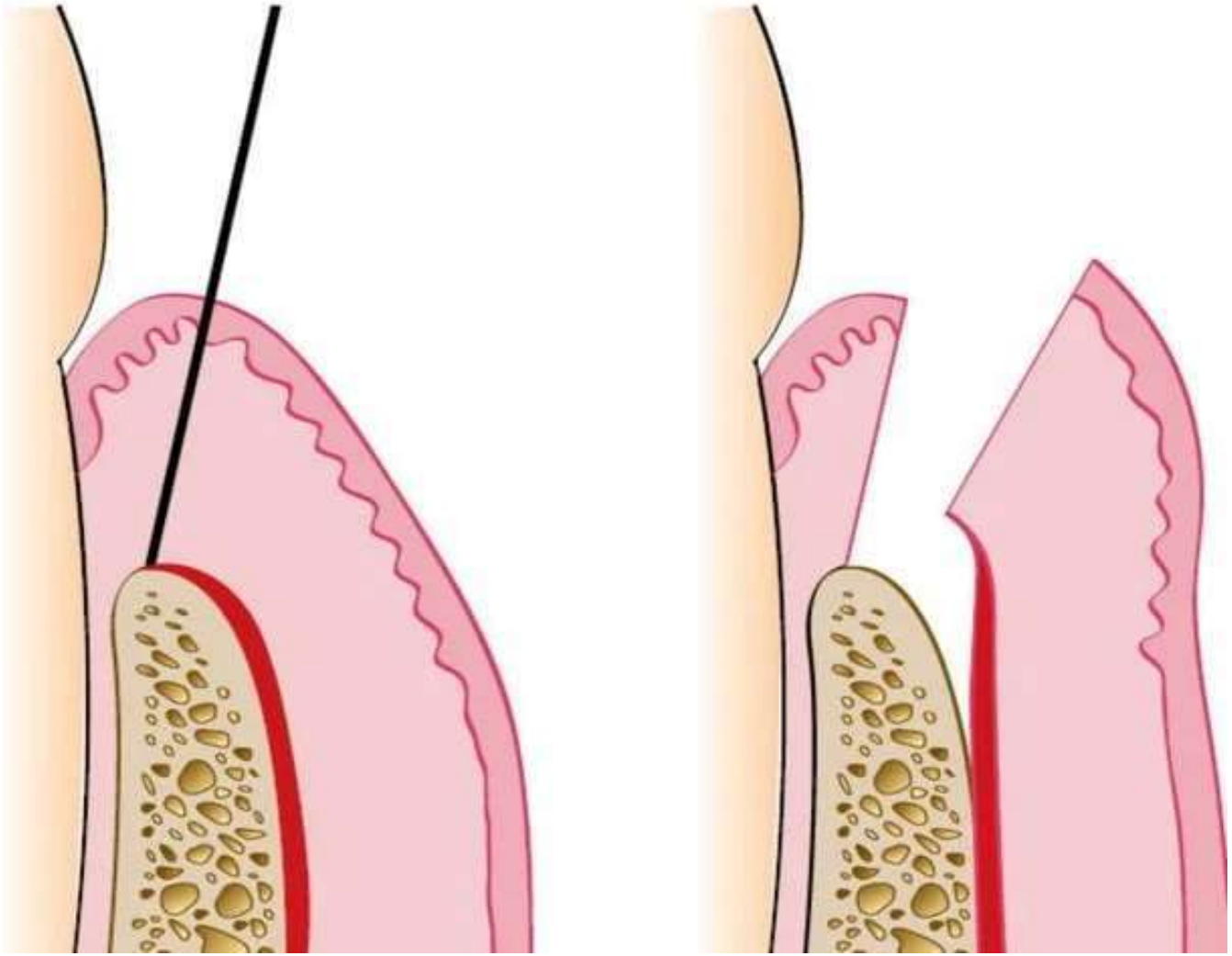
1. **Bone exposure after flap reflection**
    1. Full thickness (mucoperiosteal)
    2. Partial thickness (mucosal)
2. **Placement of the flap after surgery**
    1. Non displaced flaps
    2. Displaced flaps
3. **Management of the papilla**
    1. Conventional flaps
    2. Papilla preservation flaps

BASED ON BONE EXPOSURE AFTER REFLECTION

FULL THICKNESS FLAP

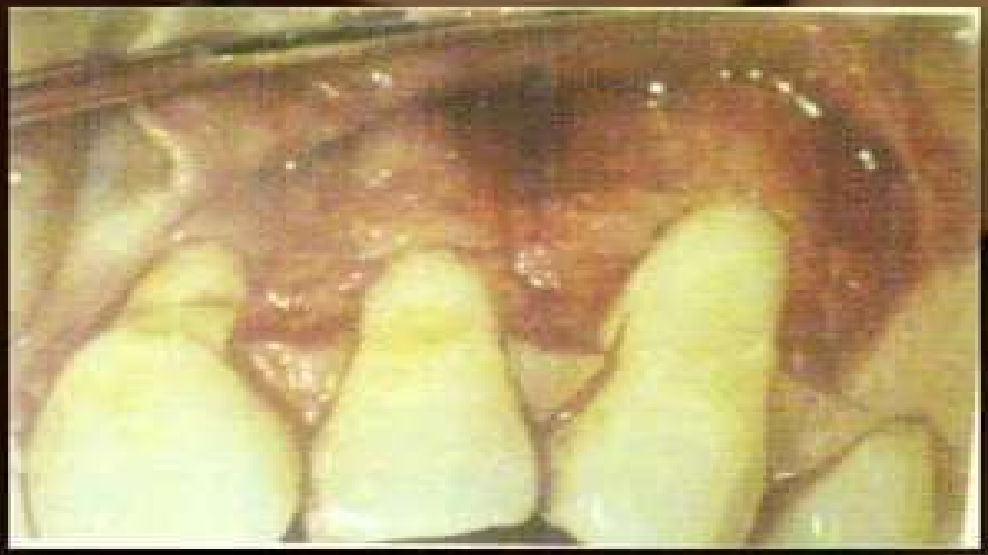
- Periosteum is reflected to expose the underlying bone.
- Indicated in resective osseous surgery.

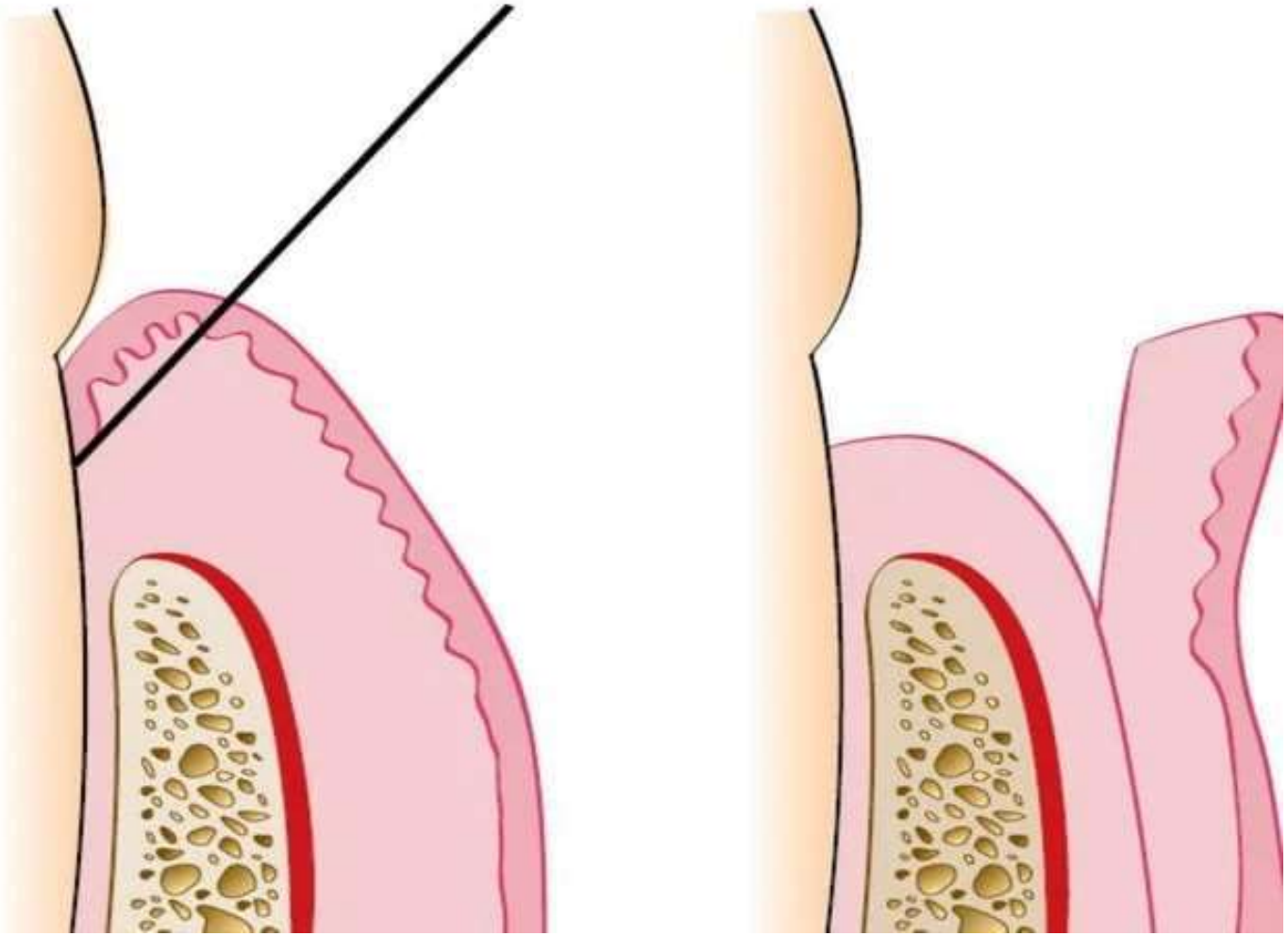




PARTIAL THICKNESS FLAP

- Split thickness flap.
- Periosteum covers the bone.
- Indicated when the flap has to be positioned apically.
- When the operator does not desire to expose the bone

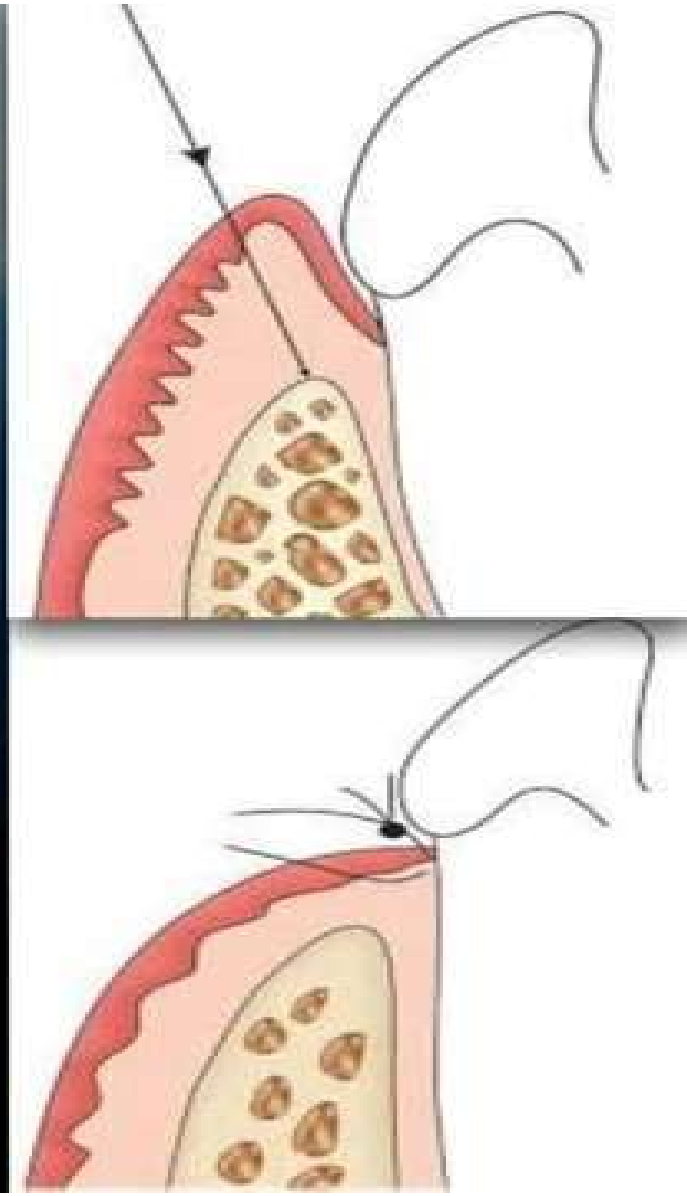





BASED ON FLAP
PLACEMENT AFTER
SURGERY

Non displaced flaps

When the flap is returned and sutured in its original position.



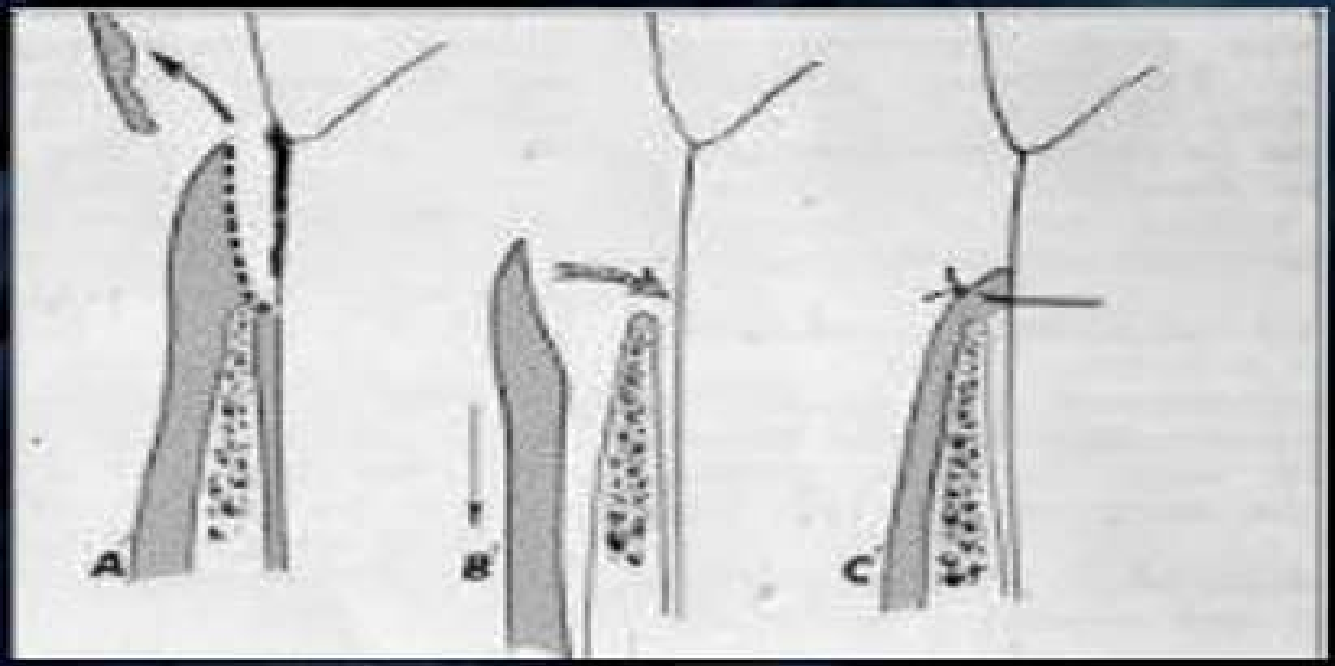
Displaced Flap

The flap is placed apically, coronally, or laterally to it's original position.

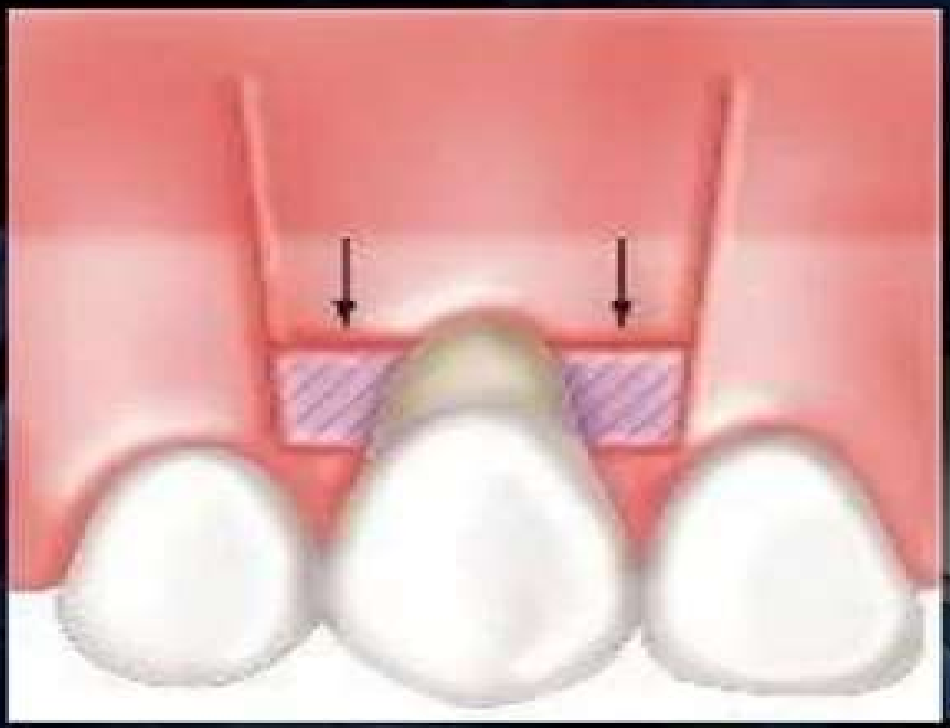
It can be a full-thickness or partial thickness flap.

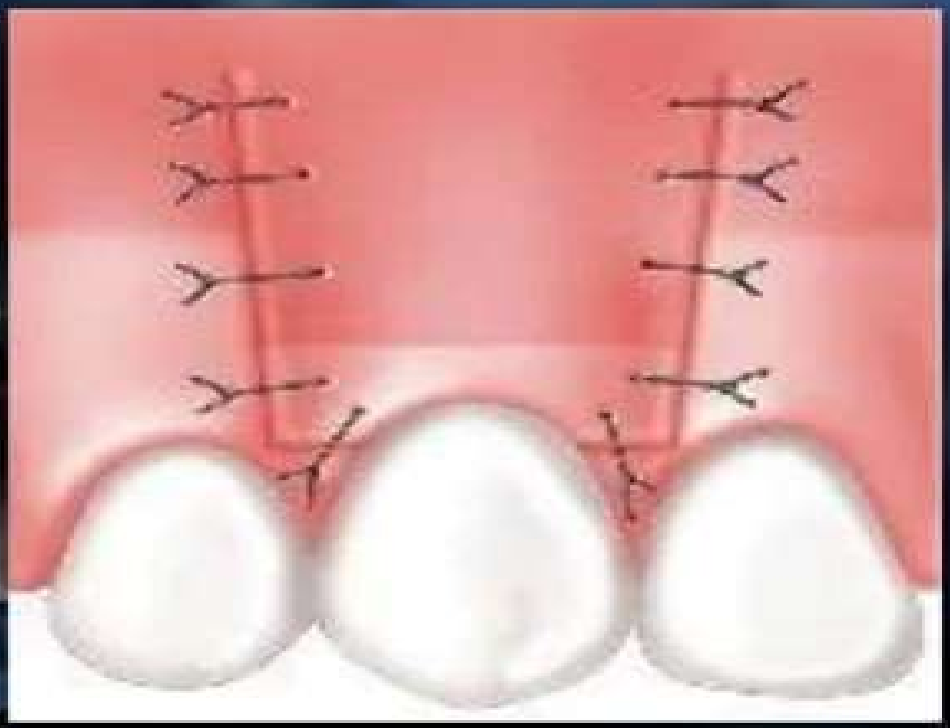
Note : The attached gingiva must be totally separated from the underlying bone.

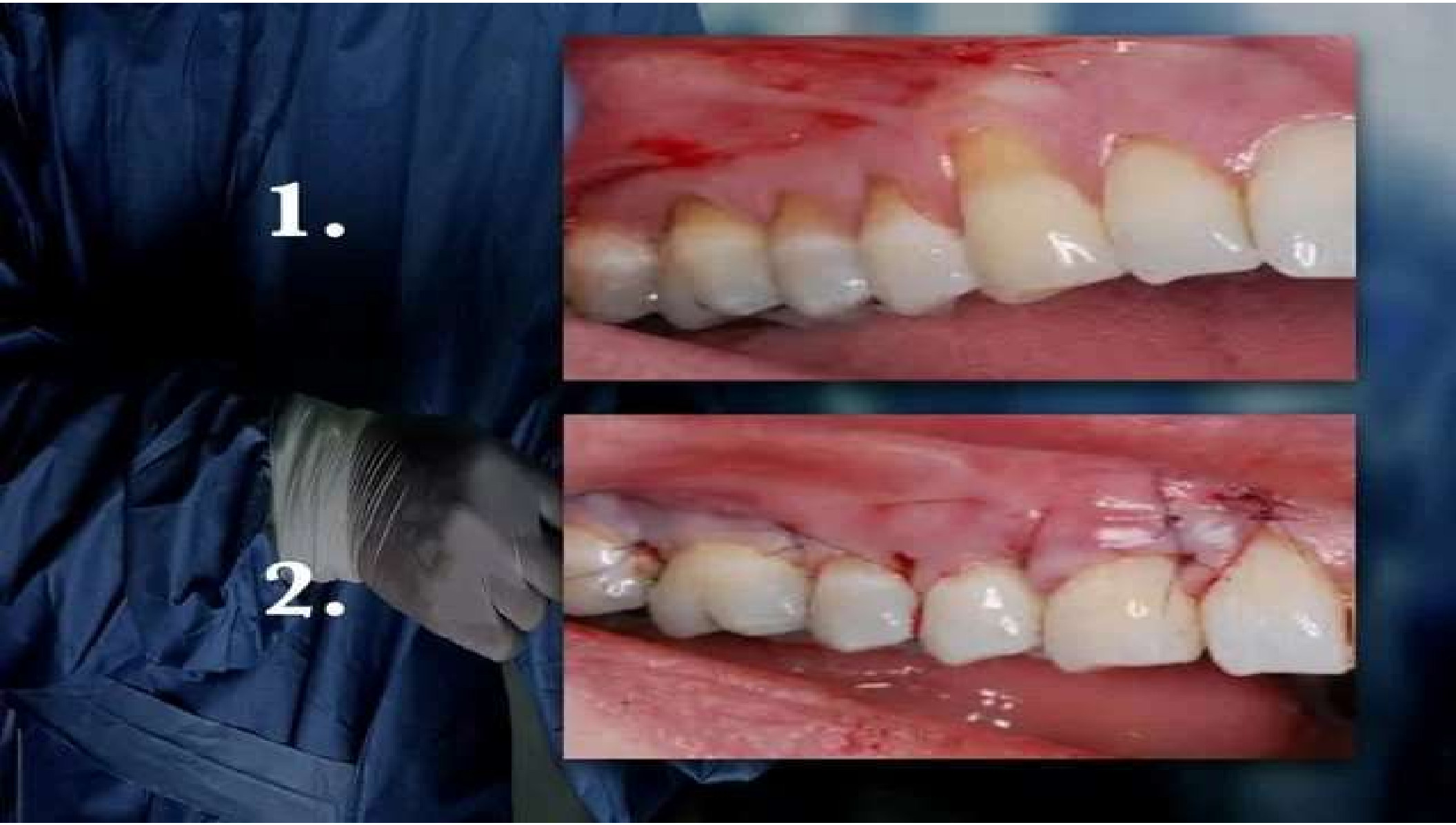
Apically Displaced Flap



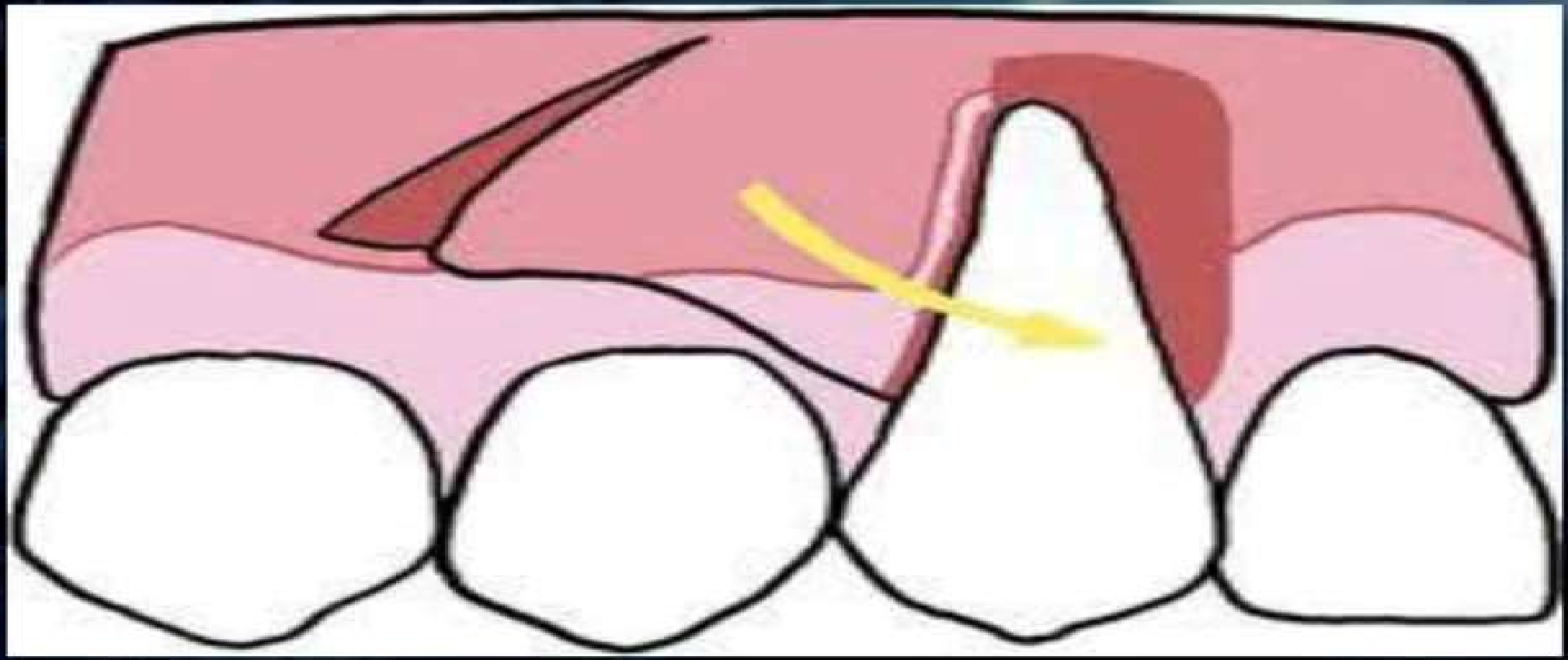
Coronally Displaced Flap



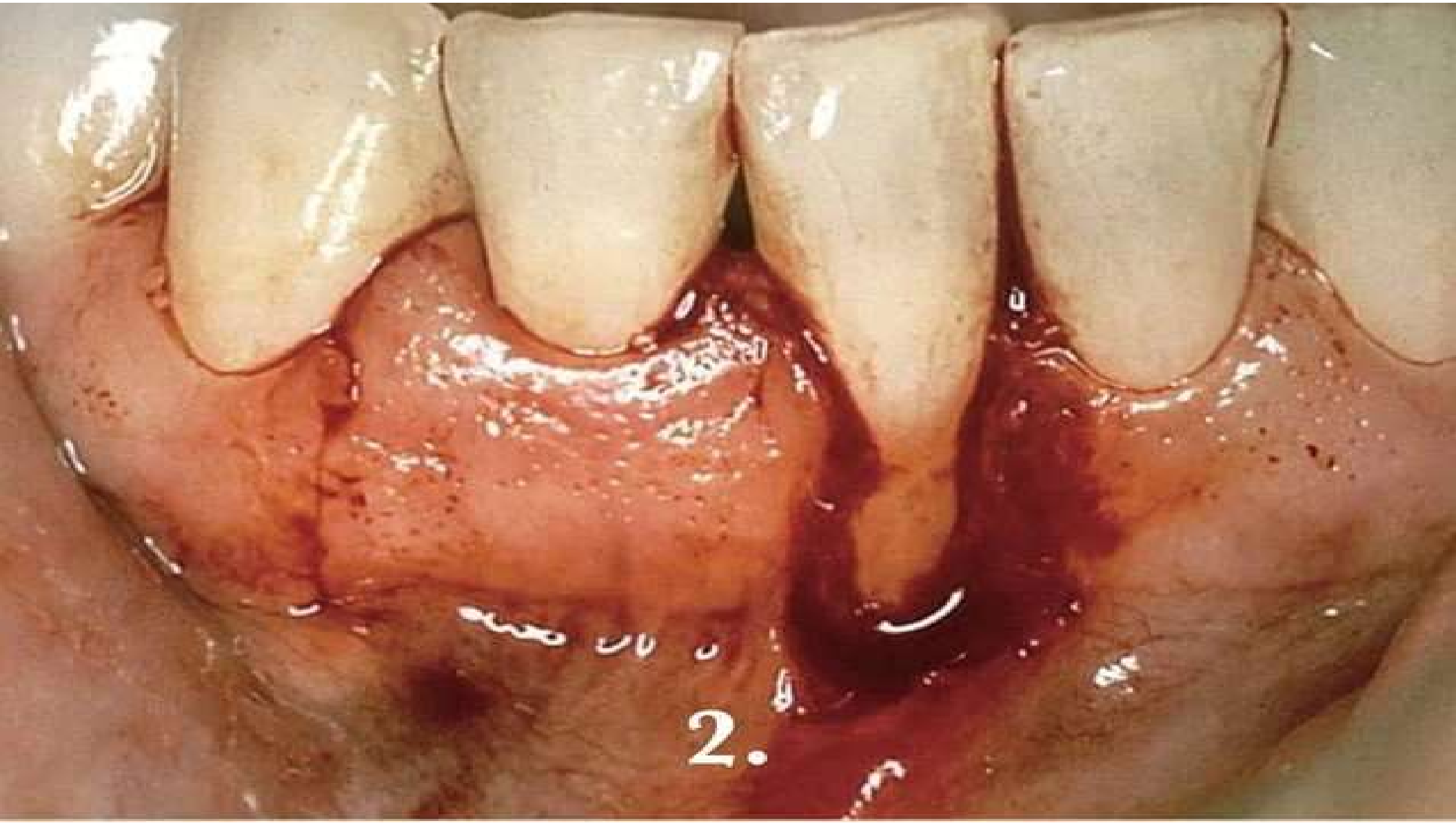


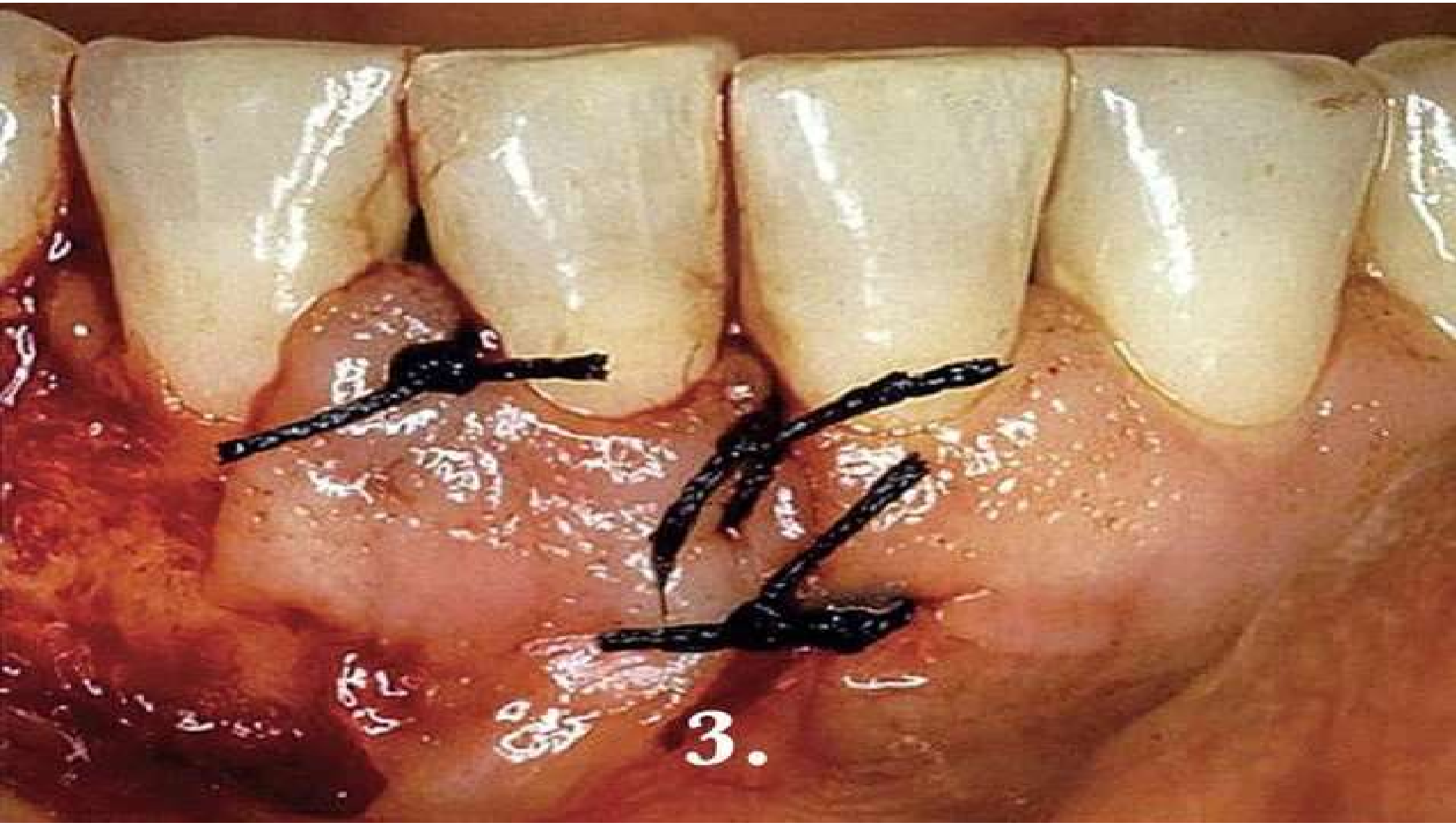

1.
2.

Laterally Displaced Flap



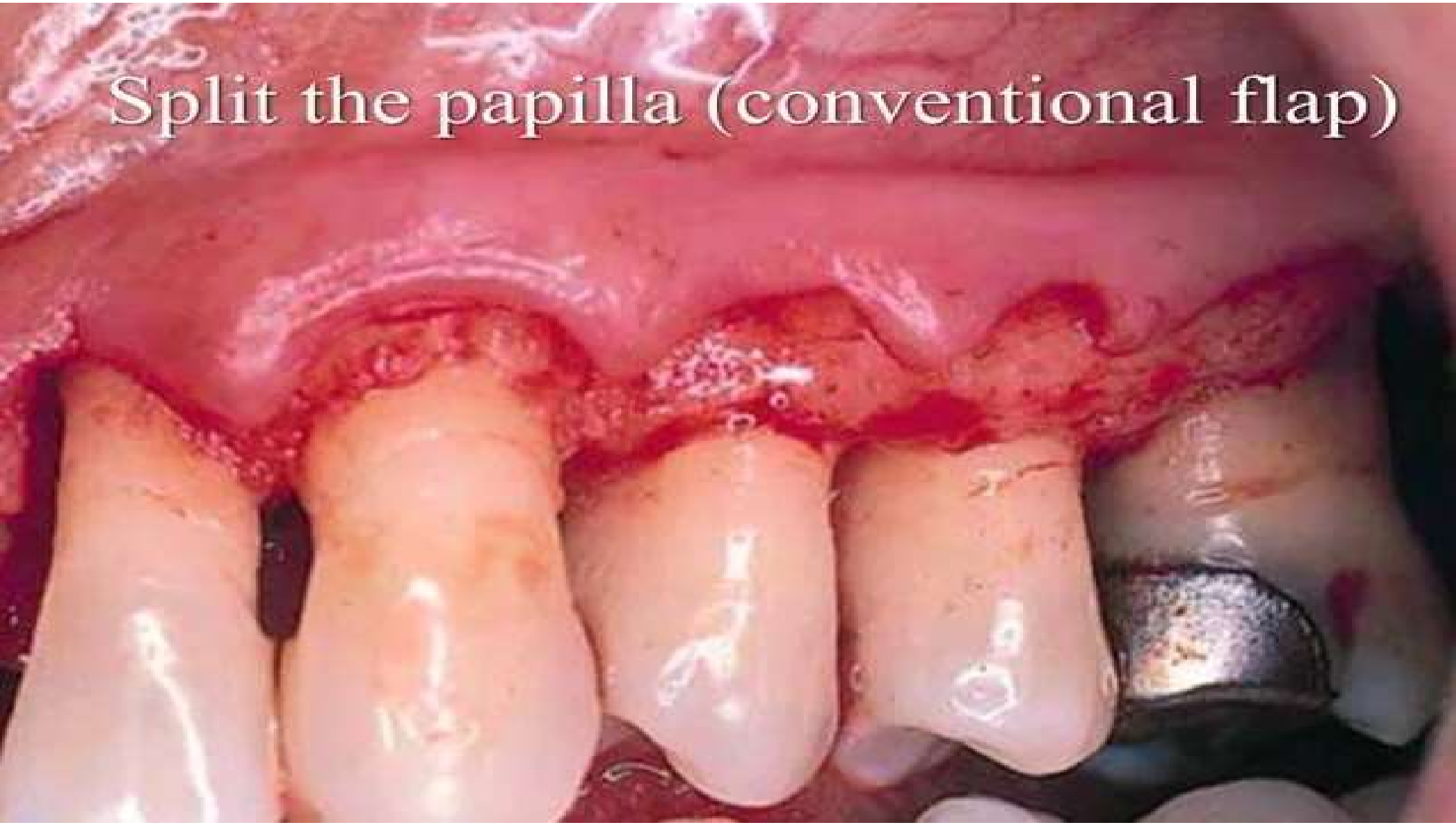

1. Pre-operative

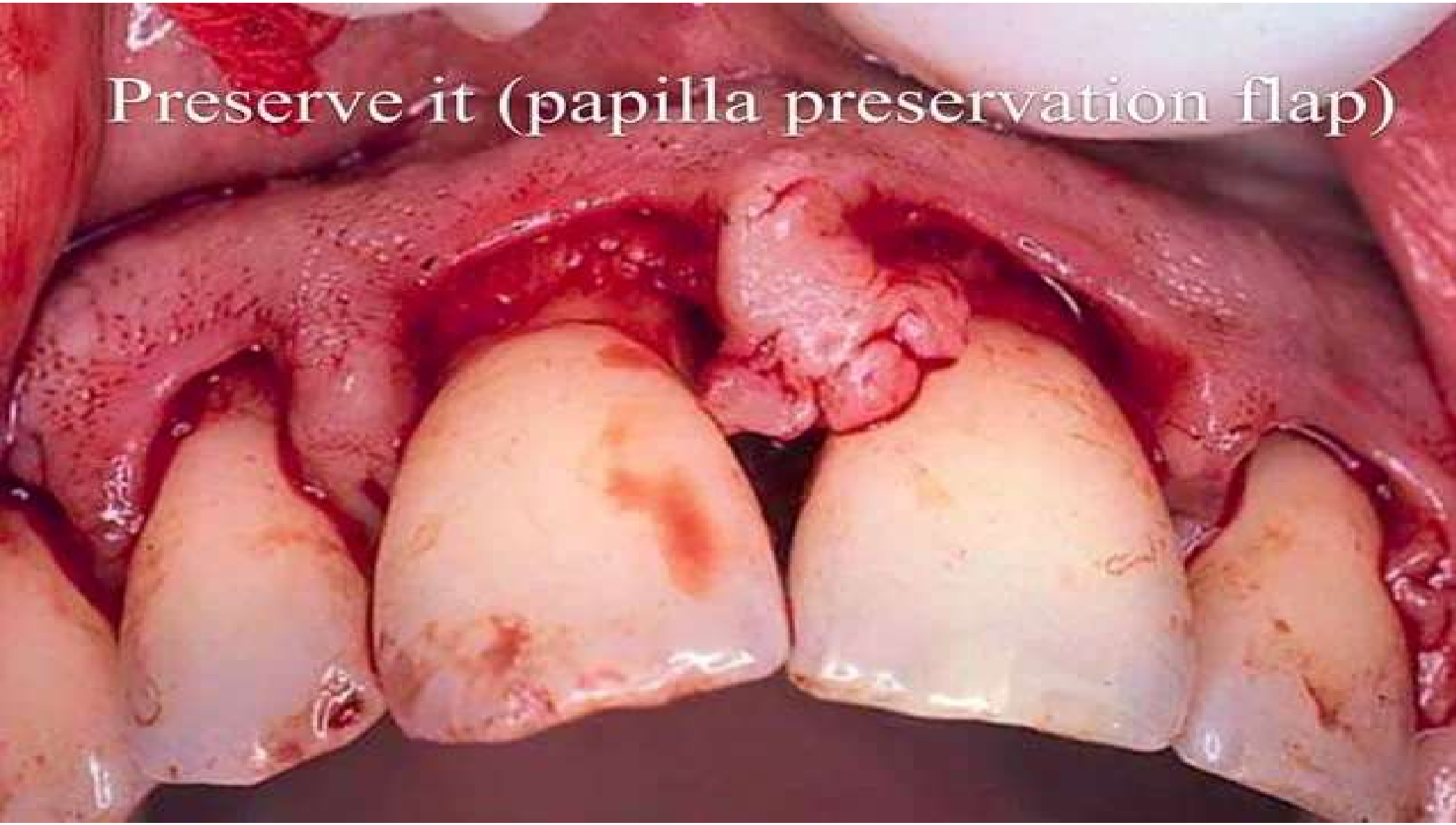

2.


3.


4. Post-operative


DESIGN
OF
THE FLAP


Split the papilla (conventional flap)


Preserve it (papilla preservation flap)

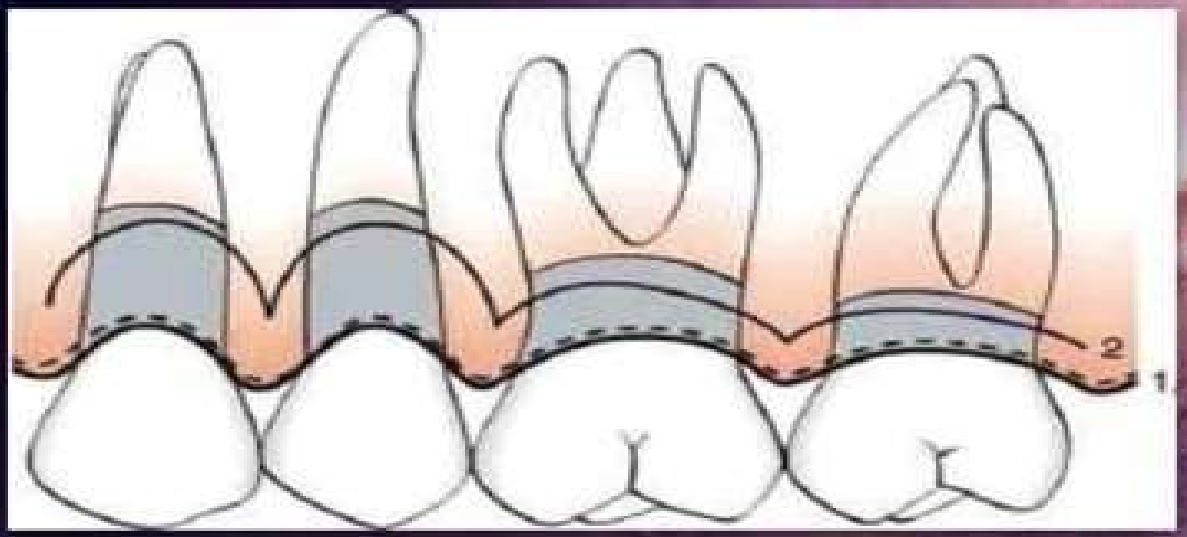

MODIFIED WIDMAN
FLAP
Presented by *Ramfjord* and *Nissle* in **1974**

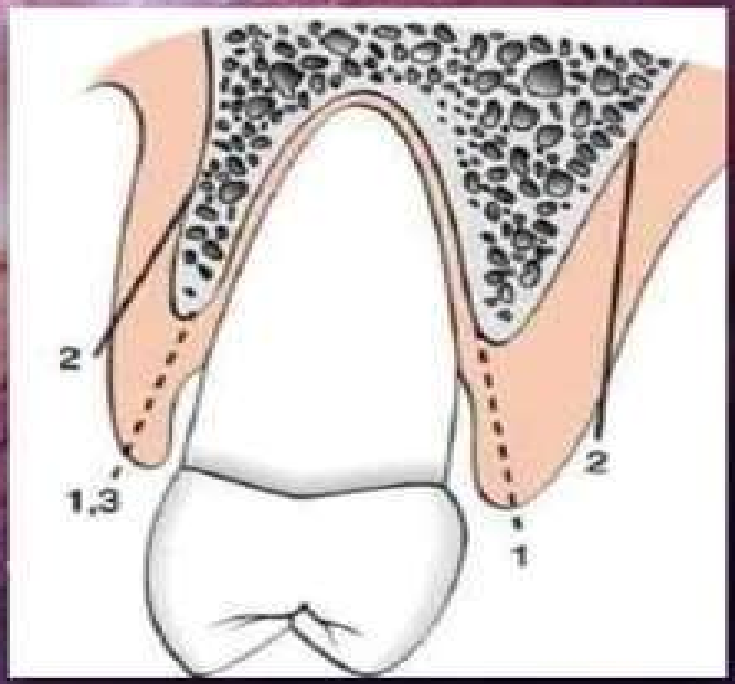

INDICATIONS:
Effective with pocket depths of 5-7 mm
CONTRAINDICATIONS:
Lack of or very thin and narrow attached gingiva can render the technique difficult, because a narrow band of attached gingiva does not permit the initial scalloped incision (internal gingivectomy).


ADVANTAGES
1.Root cleaning done with direct vision.
2.Healing by primary intention.
3.Minimal crestal bone resorption.
4.Lack of post operative discomfort.

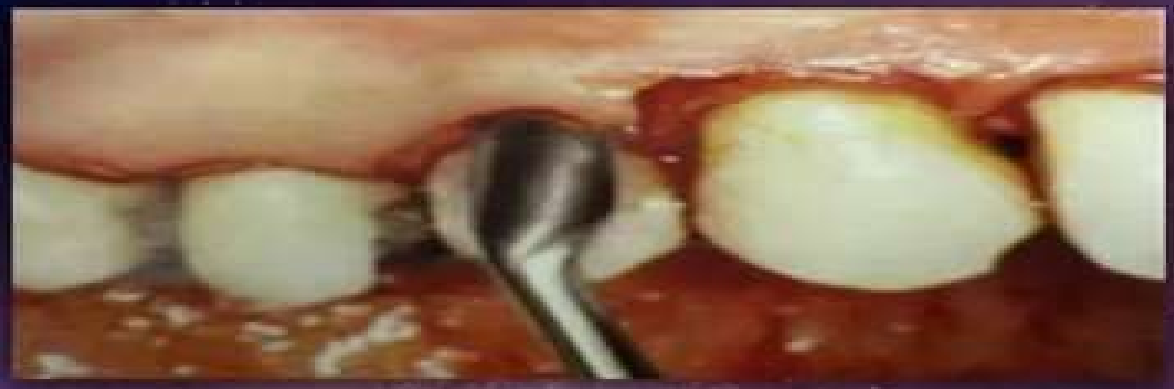
Procedure

Internal bevel incision should be made to the alveolar crest starting 0.5 to 1 mm away from the gingival margin.

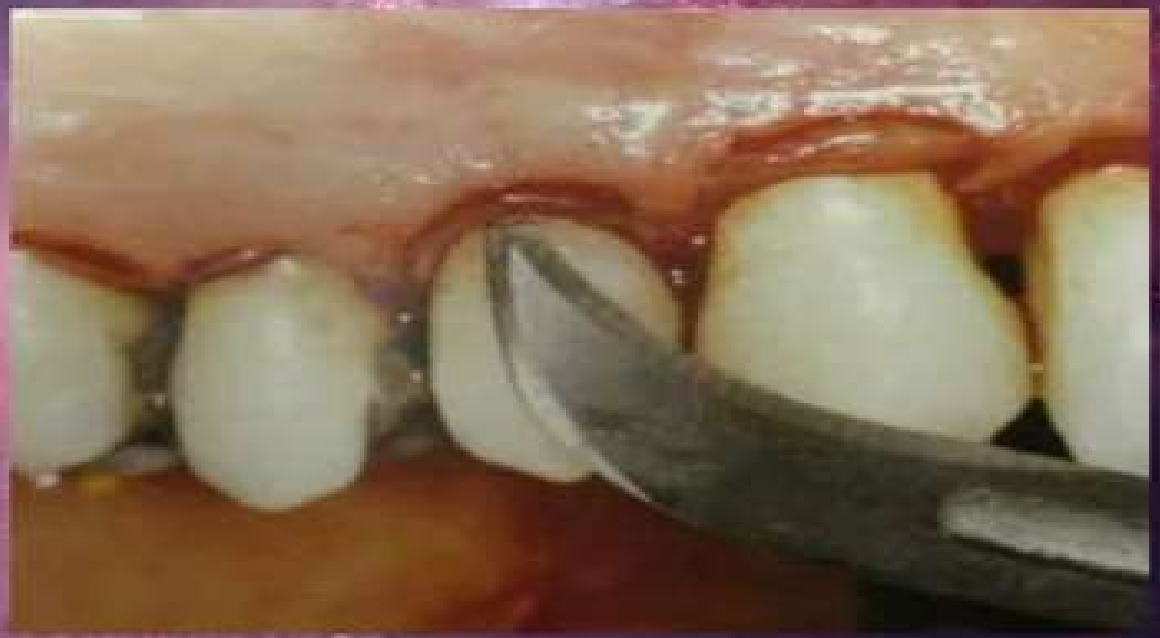




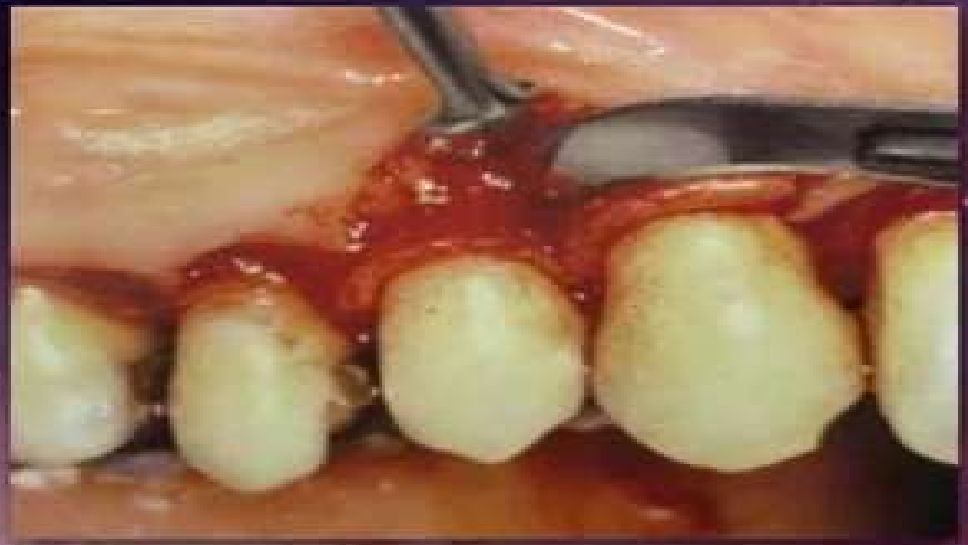
Flap is elevated



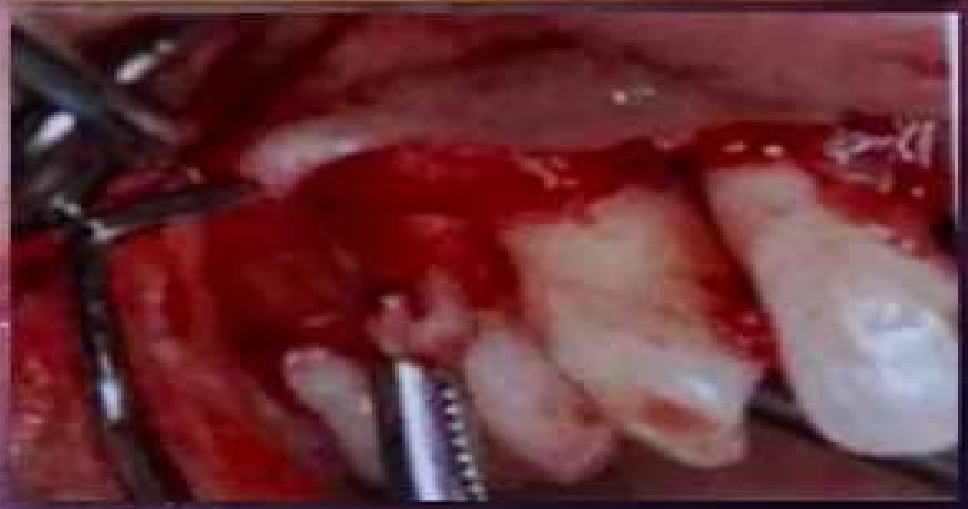
Crevicular incision is made from the bottom of the pocket to bone



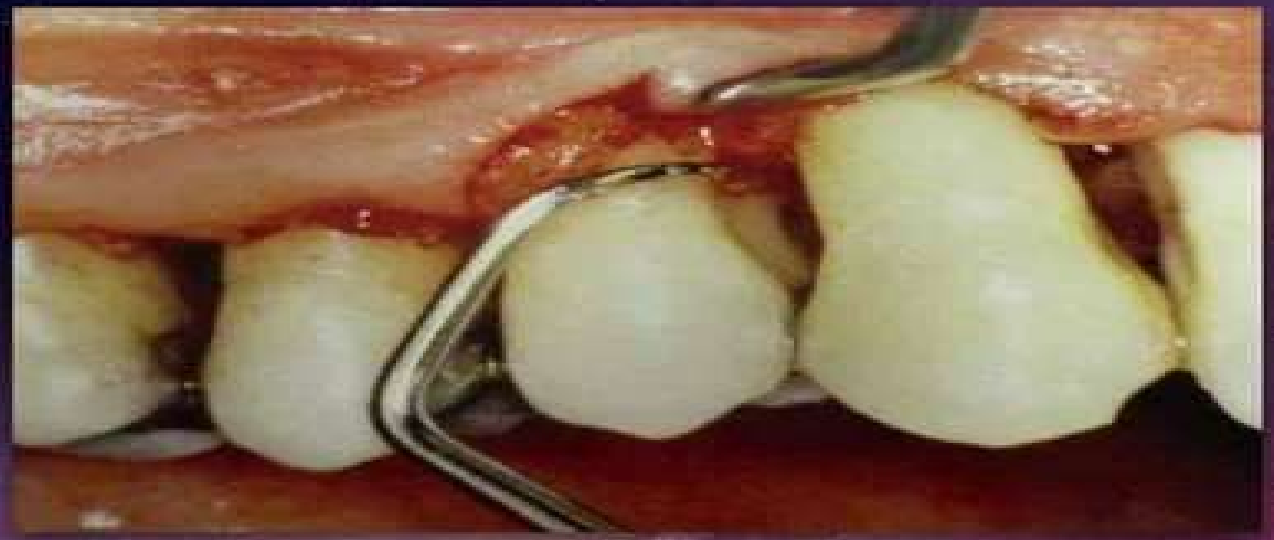
Interdental incision sectioning the base of the papilla.



Tissue tags and granulation tissue are removed.



Scaling
and
root planing
of
exposed root surfaces



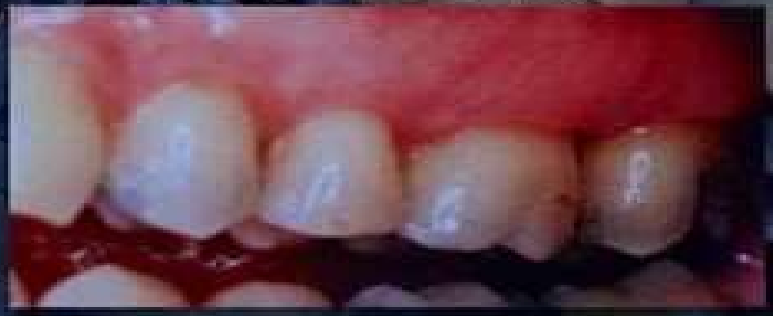
Suturing done and
covered with
tetracycline ointment
and with a periodontal
surgical pack

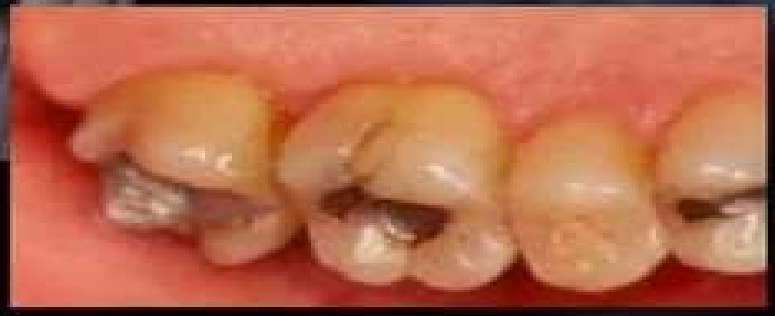
THE UN-DISPLACED FLAP

1. Most commonly performed type of periodontal surgery.
2. It differs from the modified Widman flap in that the soft tissue pocket wall is removed with the initial incision; thus it considered an internal bevel gingivectomy.

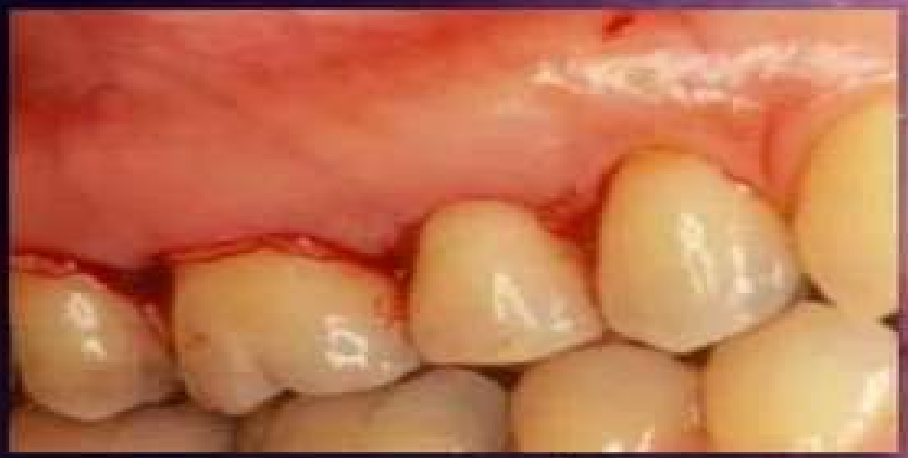
PROCEDURE

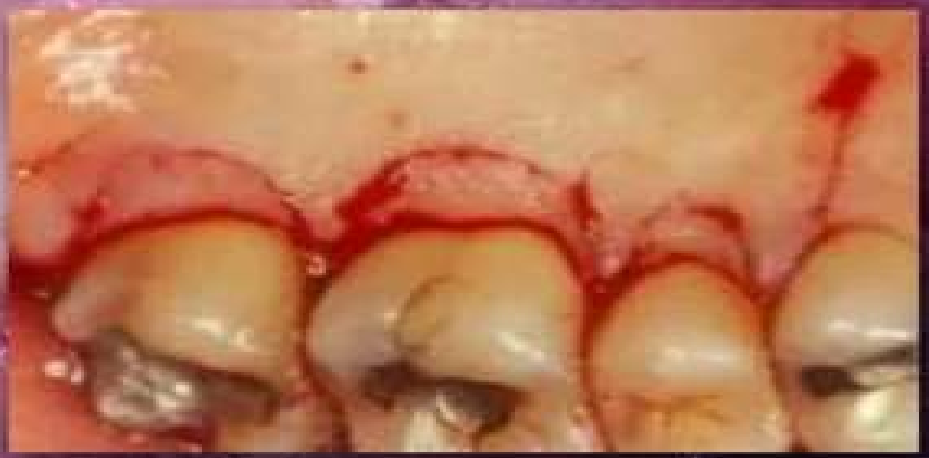
The pockets are measured with periodontal probe and a bleeding point is produced on the outer surface of gingiva to mark the pocket bottom.



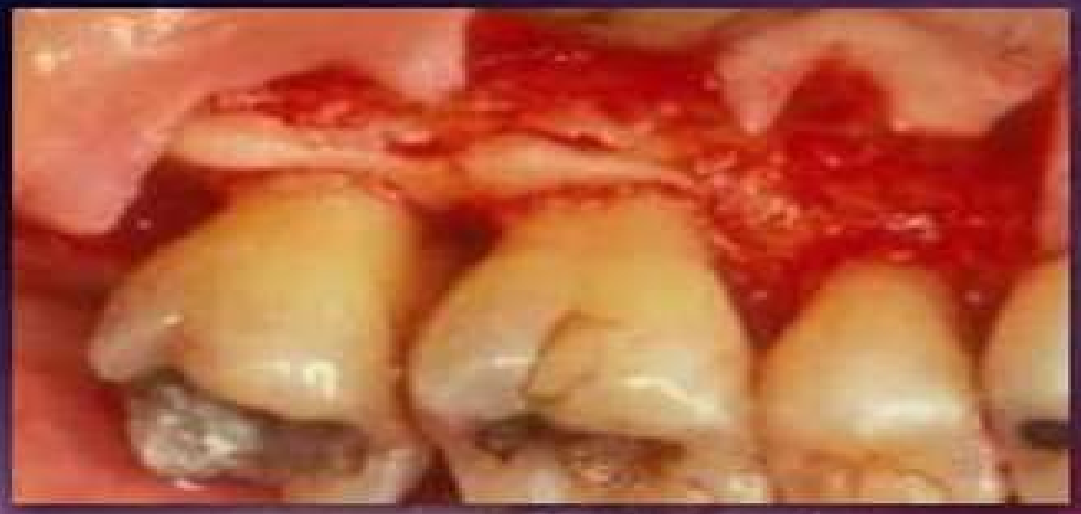


Internal bevel incision in the facial and palatal aspects

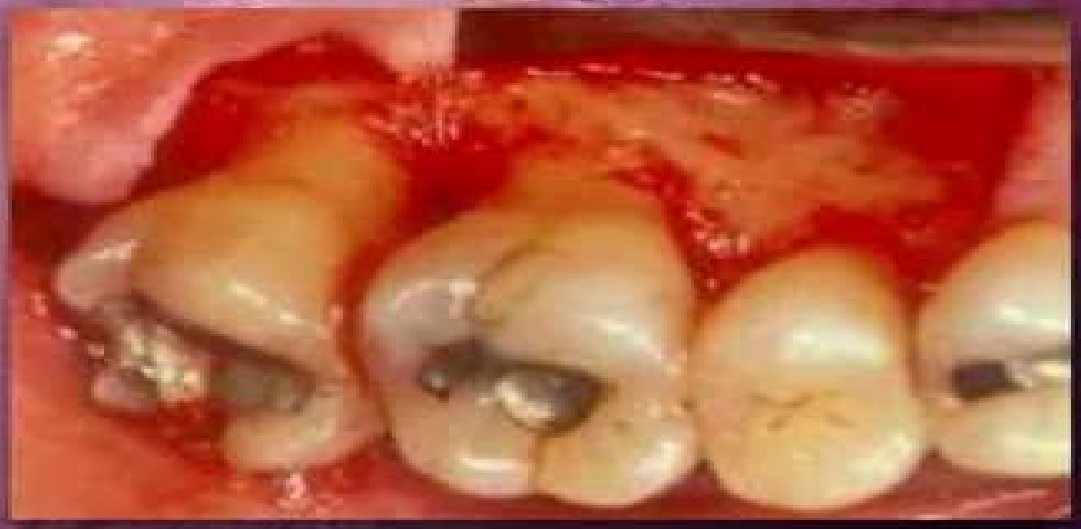




Interdental incision is made.



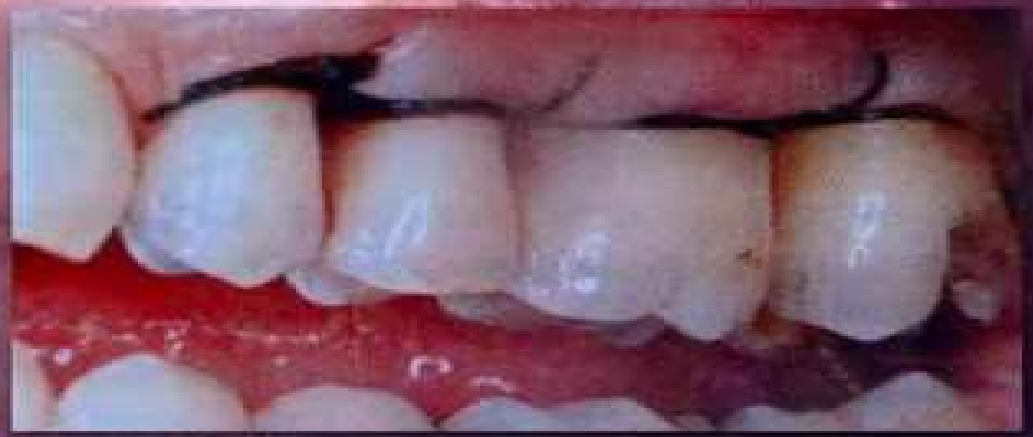
Triangular wedge of tissues is removed with curette.



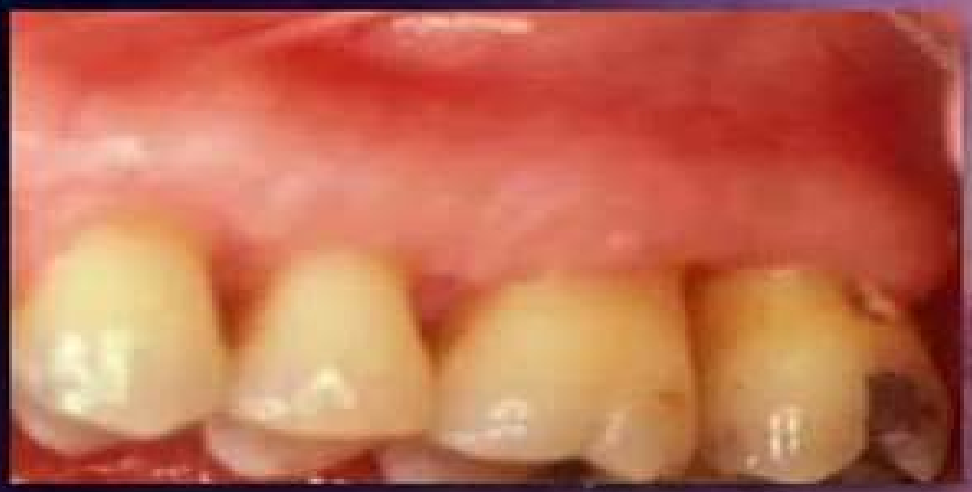
All tissue tags and granulation tissue are removed

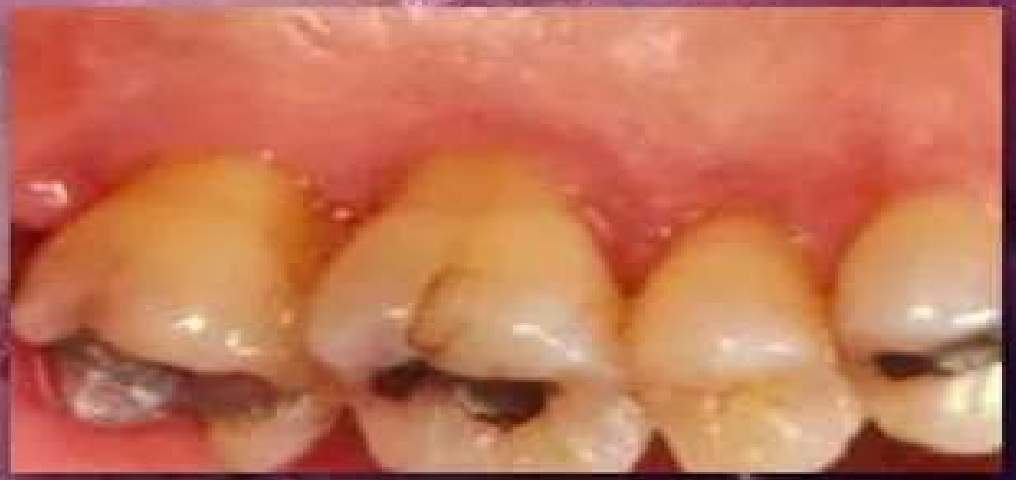
After the scaling and root planing the flap edge should rest on the root bone junction.

Flaps have been placed in their original site and Sutured.



Post-Operative Results





FLAPS FOR REGENERATIVE SURGERY

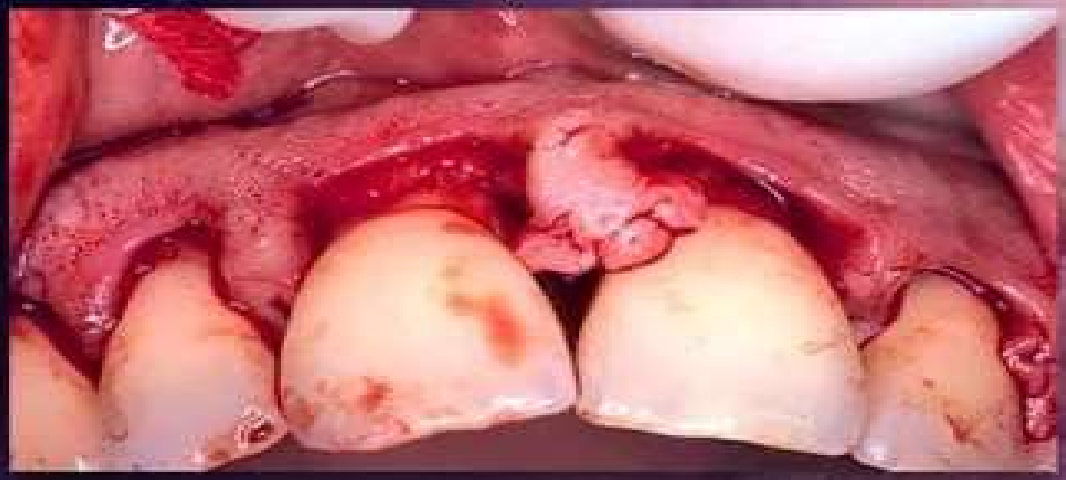
Two flap designs are available for regenerative surgery:

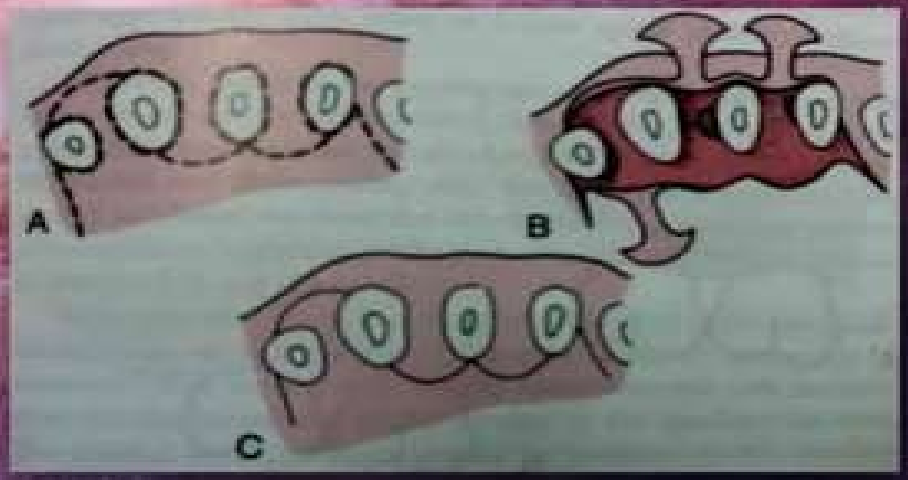
1. The papilla preservation flap &
2. The conventional flap with only crevicular incisions.

The papilla preservation flap

INDICATIONS:

- Where esthetics is of concern.
- Where bone regeneration techniques are attempted.



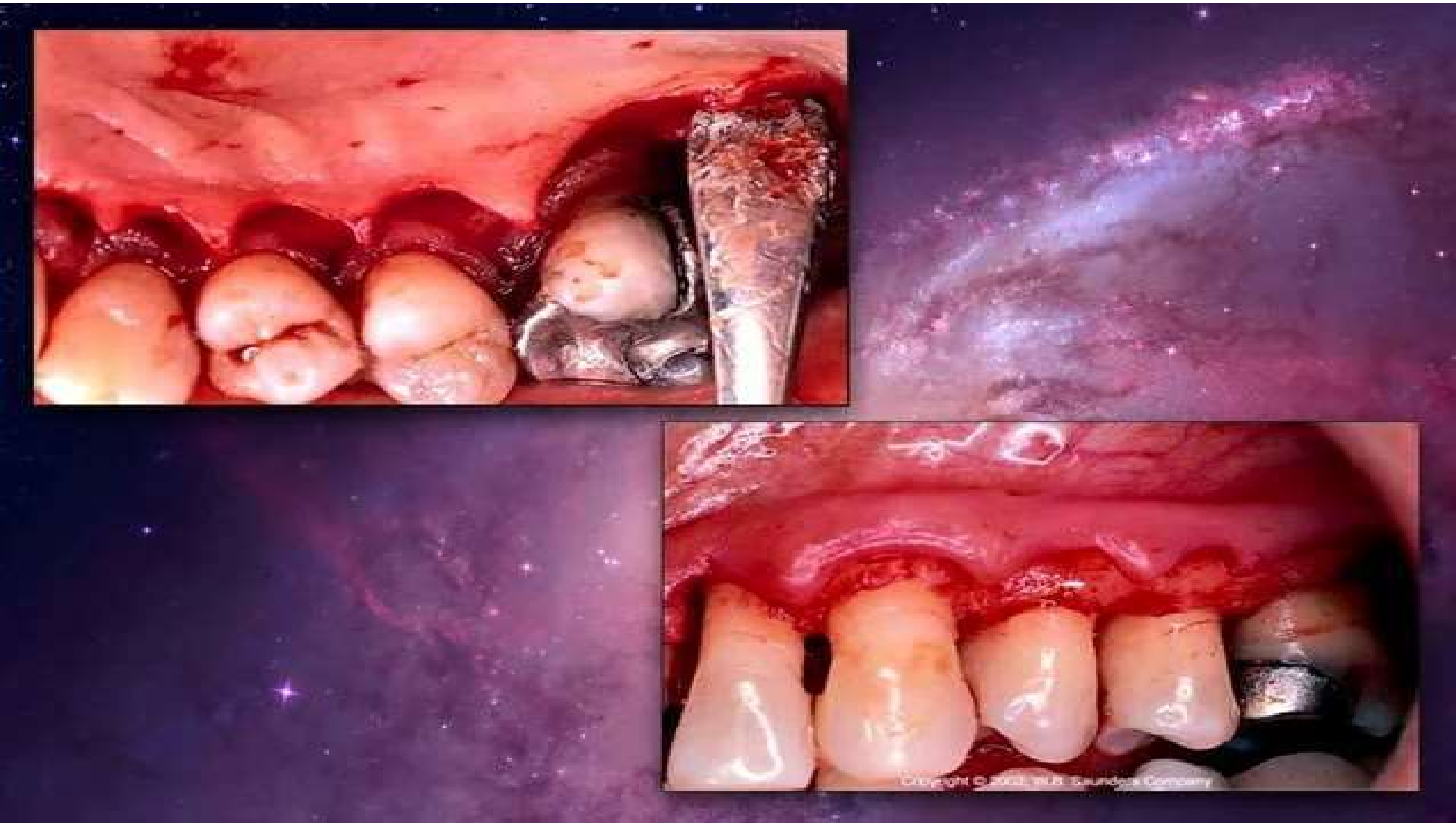


CONVENTIONAL FLAP FOR REGENERATIVE SURGERY

In the conventional flap operation, the incisions for the facial and the lingual or palatal flap reach the tip of the interdental papilla, thereby splitting the papilla into a facial half and a lingual or palatal half.

INDICATIONS:

1. When the interdental areas are too narrow to permit the preservation of flap.
2. When there is a need for displacing flaps.
3. The interdental papilla is split beneath the contact point of the two approximating teeth to allow for reflection of buccal and lingual flaps.



HEALING AFTER FLAP SURGERY

Immediately after suturing (0 to 24 hours), established by a blood clot, which consists of a fibrin reticulum with many polymorphonuclear leukocytes, erythrocytes, debris of injured cells, and capillaries at the edge of the wound. One to 3 days after flap surgery, the space between the flap and the tooth or bone is thinner, and epithelial cells migrate over the border of the flap.

One week after surgery– The blood clot is replaced by granulation tissue derived from the gingival connective tissue, the bone marrow, and the periodontal ligament.

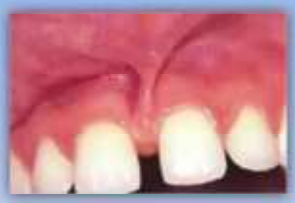
Time Interval	Healing Process
Up to 24 hours	Blood Clot
1 - 3 days	Epithelial cell migration on the tooth
7th Day	Epithelial attachment to the tooth surface. Blood clot replaced by granulation tissue derived from gingival C.T., bone marrow & PDL.
2 weeks	Immature collagen fibres : Parallel to the Tooth Surface.
1 month	Well defined epithelial attachment.

Mucogingival surgery (M.G.S)

- They are surgical procedures designed to correct defects in the morphology, position &\or amount of the gingiva &\or oral mucous membrane surrounding the teeth that complicate PD and may interfere with the success of periodontal treatment.
- Recently, it has been renamed as "Periodontal Plastic Surgery".

Indications of M.G.S

1. The change in the morphology will facilitate proper plaque control
   e.g. correction of high frenum attachment.



2. With localized soft tissue recession that creates esthetic or root sensitivity problems.

3. With a thin gingiva facial to a tooth planned for orthodontic movement when the final position expected to result in an alveolar bone dehiscence and fenestration.

Types of M.G.S

I. Techniques to increase width of attached gingiva & depth of vestibules:
   - A. Gingival augmentation coronal to the recession .
     1. Free gingival autograft.
     2. Subepithelial connective tissue graft:
     3. Pedicle autografts:
        - ✓ Laterally positioned flap.
        - ✓ Coronally positioned flap.
     4. GTR using allograft
     5. Pouch & tunnel technique
   - B. Gingival augmentation apical to the recession:
     1. Free gingival autograft.
     2. Free connective tissue autografts.
     3. Vestibular extension technique.
     4. Apically positioned flap.

II. Frenectomy & Frenetomy.

III. Techniques to improve esthetics:
   - A. Root coverage.
   - B. Papilla reconstruction.
   - C. Therapy to correct excessive gingival display.

IV. Tissue engineering.

I. Techniques to increase attached gingiva & depth of vestibules:

A- Gingival augmentation coronal to the recession (Root coverage):

If the width of the attached gingiva is adequate in the donor site, , includes:

1. Free gingival autograft: that consist of epithelium and a thin layer of underlying CT completely detached from one site and transferred to a remote site.

Advantages

- Increase keratinized tissue around teeth, implants or crowns and under removable prostheses.
- Increase vestibular depth.

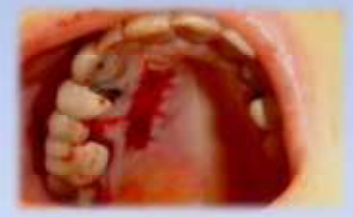
Disadvantages

- ❖ Difficult to achieve root coverage.
- ❖ High esthetic demand.
- ❖ Large, uncomfortable donor site.

Surgical technique

- **Step 1: Prepare the recipient site.**
- **Step 2: Root preparation:**
  - ➢ Root planing of exposed root to remove cementum and affected dentin.
  - ➢ Etch root surface with tetracycline (pH 2.0).

- **Step 3: Obtain the graft from the donor site:** The ideal thickness of a graft is 1.0 - 1.5 mm.



- **Step 4: Graft transferred to recipient site.**
- **Step 5: Protect the donor site.**

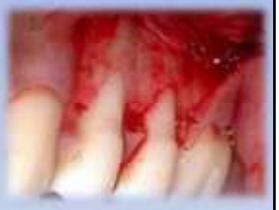
2. Subepithelial connective tissue graft:

A detached CTG that is placed beneath a partial thickness flap.

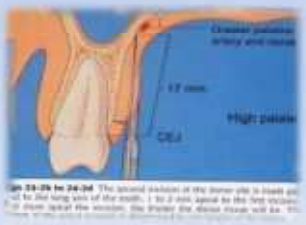
Surgical technique:

Step 1: Recipient site incision.

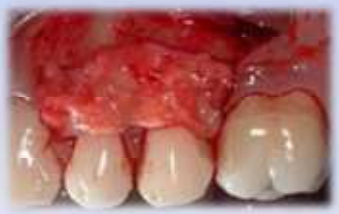
Step 2: Root preparation.



Step 3: Donor site incision.



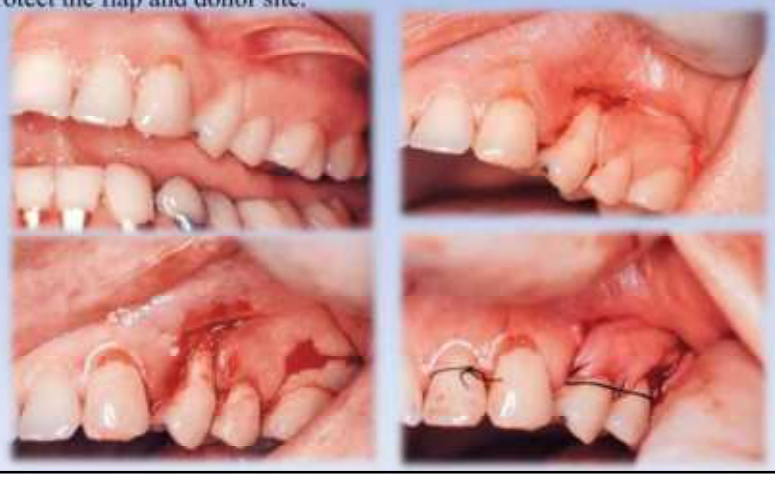
Step 4: Transfer the graft and suturing.



3. Pedicle autografts: A soft tissue graft that is not completely detached from one site and transferred to another site. There are connection with the donor site is maintained.

A. Laterally positioned flap:

Step 1: Prepare the recipient site
Step 2: Prepare the flap of the donor site.
Step 3: Transfer the flap.
Step 4: Protect the flap and donor site.



B. Coronally positioned flap:

First technique:

Step 1: With 2 vertical incisions.

Step 2: Root preparation

Step 3: Return the flap and suture it coronal to the pretreatment position.

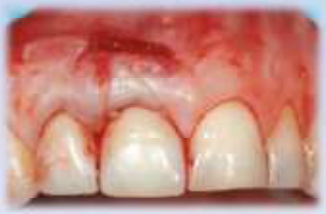
Step 4: Cover the area with a periodontal dressing.

Second Technique (Semilunar coronally positioned flap):

Step 1: Semilunar incision is made and ending about 2 to 3 mm short of the tip of the papillae.

Step 2: Perform a split-thickness dissection coronally from the incision, and connect it to an intrasulcular incision.

Step 3: The tissue will collapse coronally, covering the denuded root. then held in its new position for a few minutes with a moist gauze. Many cases do not require either sutures or periodontal dressing.



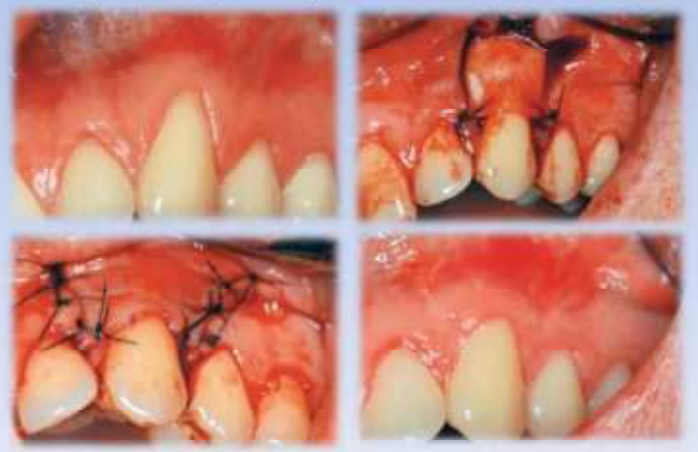
4. Guided Tissue Regeneration using allograft:

Step 1: A full-thickness flap is reflected to MGJ, continuing as a partial-thickness flap 8 mm apical to MGJ.

Step 2: Root preparation.

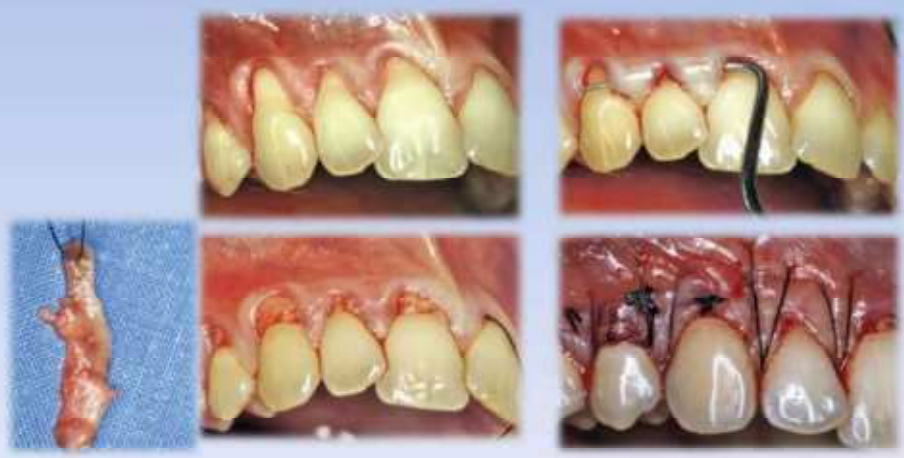
Step 3: A membrane is placed over the root surface and the adjacent tissue at least 2 mm of marginal periosteum.

Step 4: The flap is then positioned coronally and sutured.



5. Pouch and Tunnel technique:

- Create "pouch" using full thickness incision and maintain papilla for bilaminar blood supply.
- Extend incision to adjacent teeth and undermine flap beyond MGJ, which allows the coronal positioning of the flap.
- Insertion of CTG and suture.

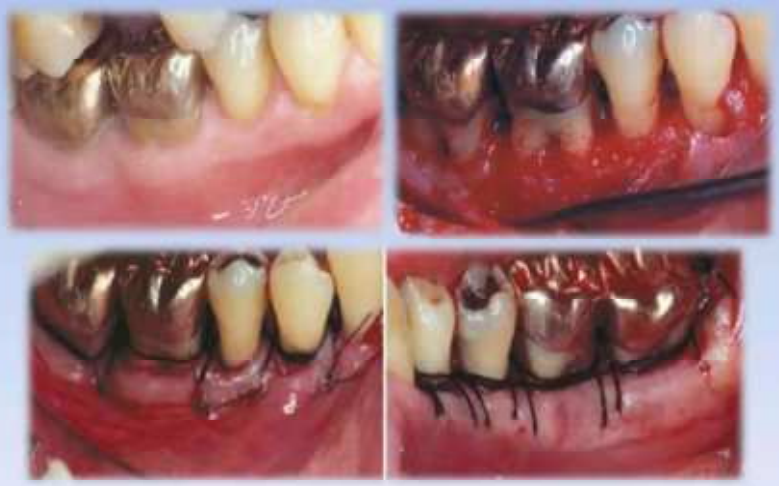


4. Apically positioned flap:

Step 1: The facial and lingual flaps have been elevated.

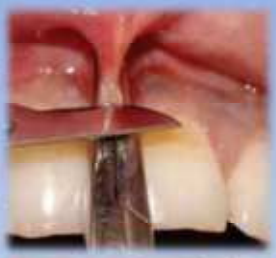
Step 2: Debridement of the areas.

Step 3: The sutures are in place.

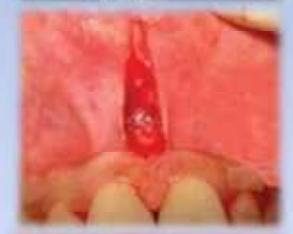


Surgical technique:

Step 1: Hold the frenum with a hemostat inserted to the depth of the vestibule.
Step 2: Incise along the uppersurface of the hemostat, extending beyond the tip and make a similar incision along the undersurface of the hemostat.



Step 3: Remove the triangular resected portion of the frenum with the hemostat. This exposes the underlying fibrous attachment to the bone.
Step 4: Make a horizontal incision, separating the fibers and bluntly dissect to the bone. Undermined for CT.

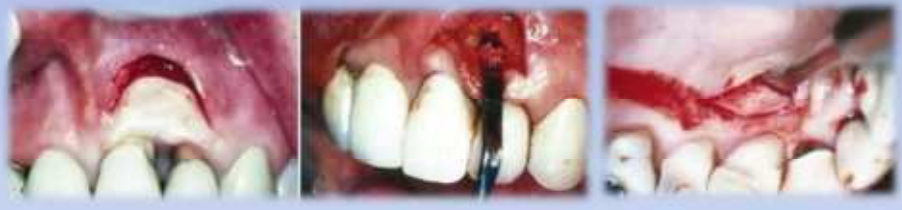


Step 5: Suturing the area.

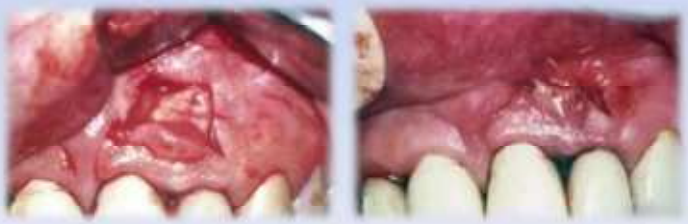
III. Techniques to improve esthetics:

1. Root coverage.
2. Papilla reconstruction:

- The semilunar surgical pouch and CTG.



- Then, suture to reconstruct the interdental papilla.

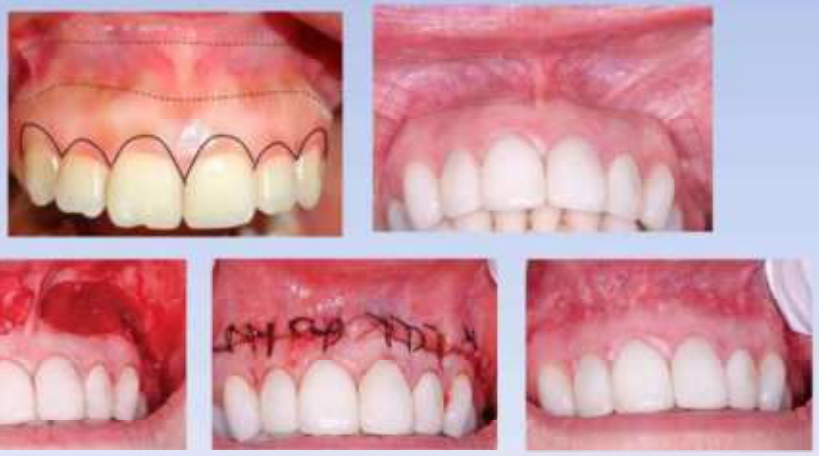


3. Therapy to correct excessive gingival display:

- Excision of marginal gingiva to expose full anatomic crown.
- Full-thickness flap elevation then ostectomy and osteoplasty completed.
- Flap repositioned and sutured with interrupted sling sutures.

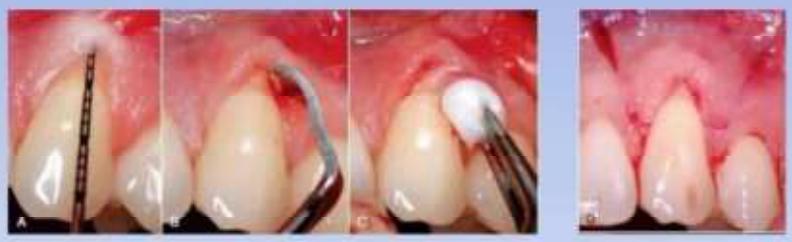
Correction of gummy smile by crown lengthening and lip repositioning

- The coronal and apical incisions met in the first or second bicuspid regions
  - The epithelium was dissected as a partial thickness flap.
  in a rounded fashion along the MG line.
  - The mucosal flap was advanced and sutured at the MG line using interrupte
- The frenum between the two centrals was left intact as a reference point so
  sutures.
  the incisions did not extend across the mid-line.

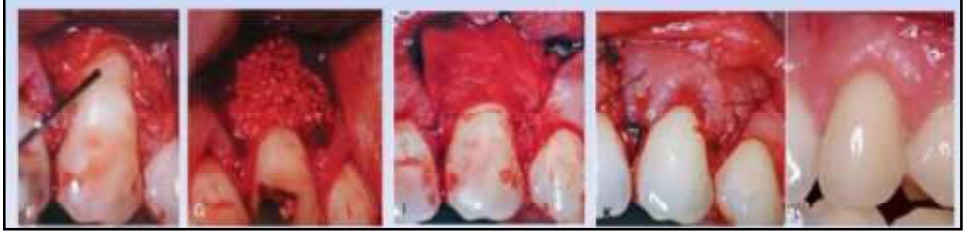


IV. Tissue engineering:(biologic mediator)

- Root surface preparations with a chisel & EDTA.
- Horizontal and vertical incisions.



- Root surface exposed to Recombinant Human Platelet-Derived (rhPDGF), then β-TCP and collagen wound dressing.
- Suture the area.



Furcation

Its Involvement and R_x

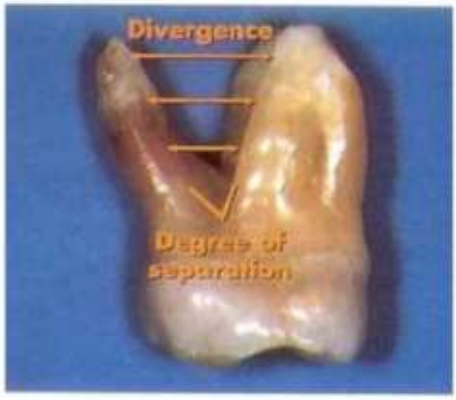
Dr Mohammed Hazeem

Why Furcation is an area of complex anatomic morphology ?

1. Difficult for routine periodontal instrumentation
2. Difficult to maintain by routine home care
3. clinical finding of furcation indicates advanced periodontitis and less favourable prognosis

Dr Mohammed Hazeem

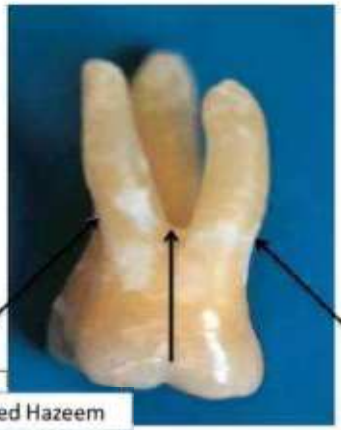
BASIC TERMINOLOGIES

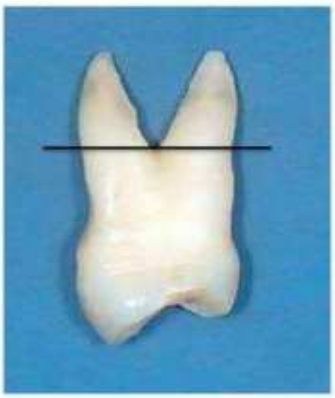


Dr Mohammed Hazeem

Brief about normal anatomy

* **Maxillary Molars & Premolars**



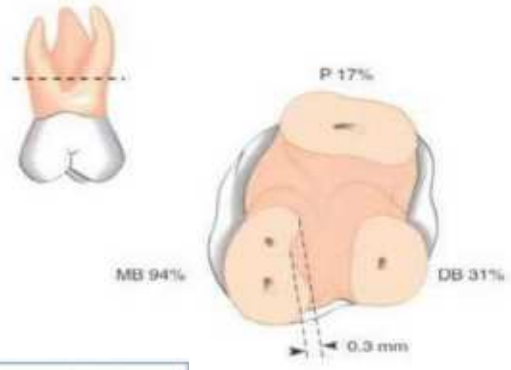


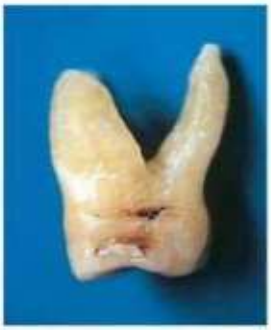
distal

mesial

Dr Mohammed Hazeem

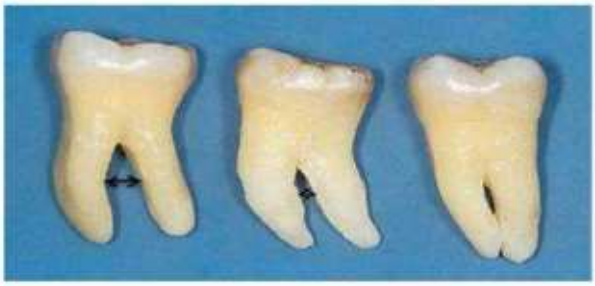
Complexity in Anatomy

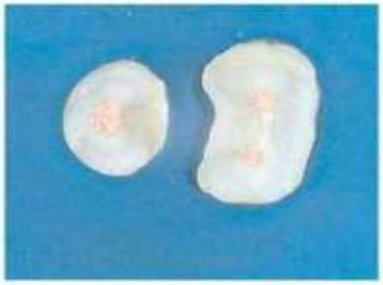




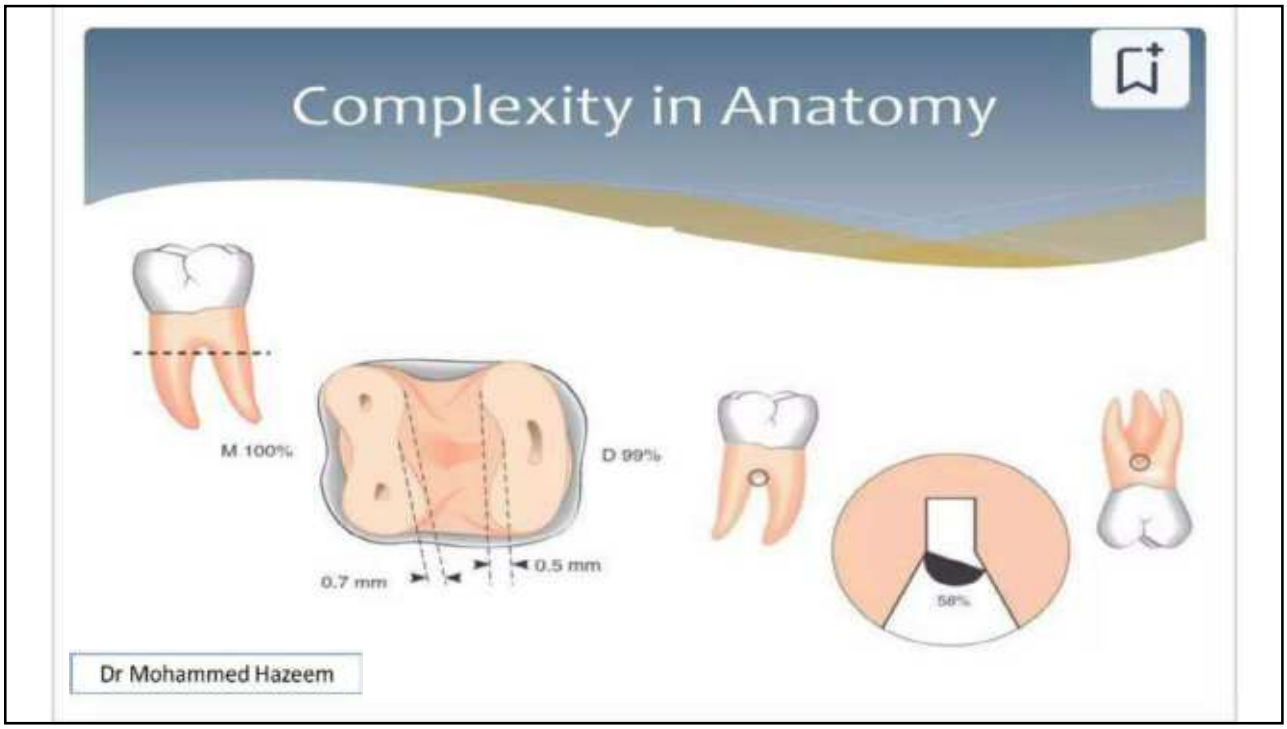
Dr Mohammed Hazeem

Mandibular Molars and other teeth





Dr Mohammed Hazeem

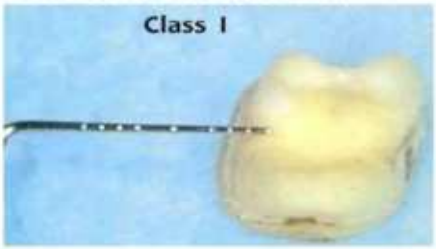

Complexity in Anatomy
M 100%
D 99%
0.7 mm
0.5 mm
58%
Dr Mohammed Hazeem


GR-III
GR-IV
Dr Mohammed Hazeem

Hamp's Classification (1975)

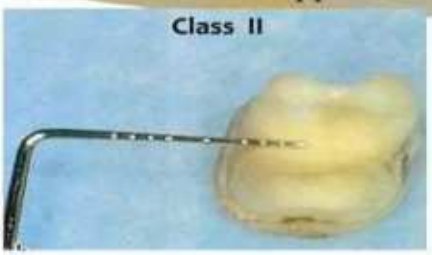
Horizontal loss ≤ 3 mm.

Class I



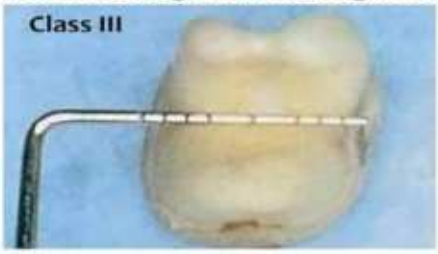
Horizontal loss of support > 3mm

Class II



Horizontal through and through destruction

Class III



Dr Mohammed Hazeem

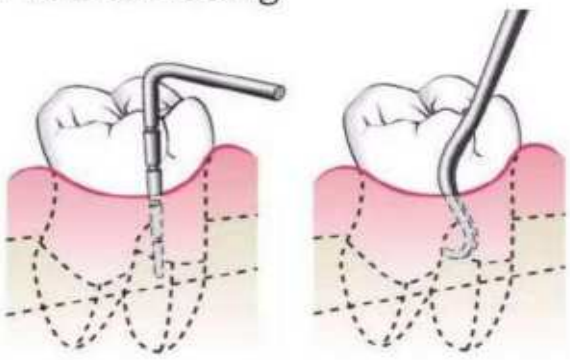
Tarnow and Fletcher (1984)

* Based on vertical component 3 subgroups:
  - Subgroup A: 1-3mm
  - Subgroup B: 4-6mm
  - Subgroup C: >7mm

Dr Mohammed Hazeem

Diagnosis

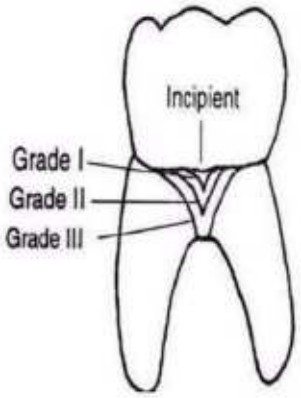
* Clinical Probing



- Naber's Probe
- No. 23 Explorer
- Each furcation entrance is classified.

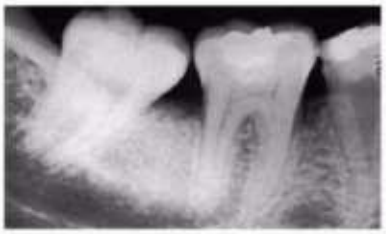
Dr Mohammed Hazeem

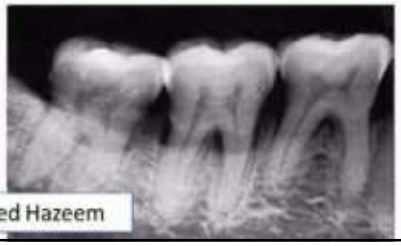
- Identification of Local anatomic factors:
  - Root trunk length
  - Root length
  - Interradicular dimension
  - Anatomy of furcation
  - Cervical Enamel Projections

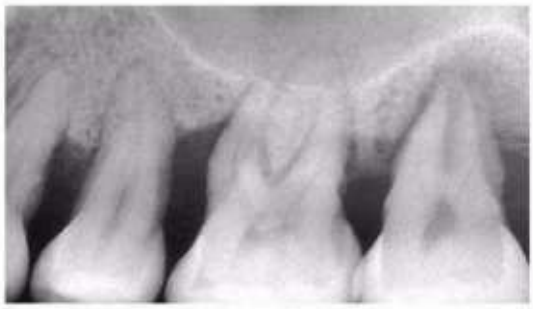


Dr Mohammed Hazeem

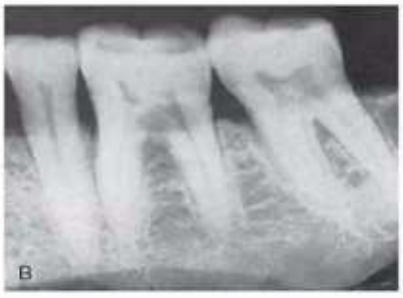
Radiographically



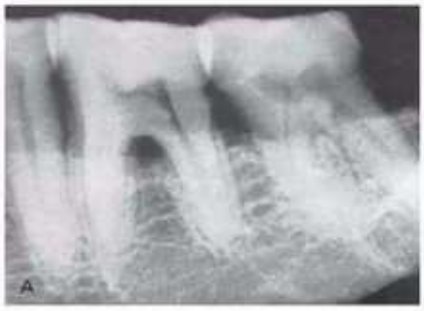




Dr Mohammed Hazeem



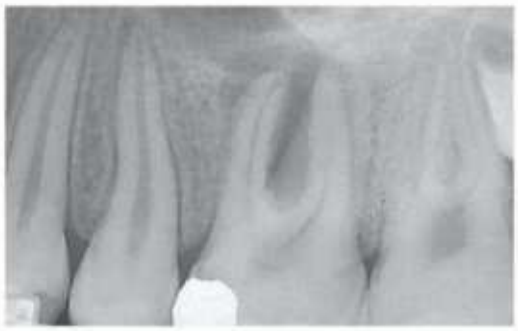
Different angulation

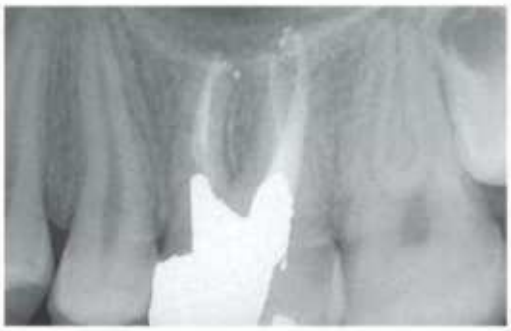


Dr Mohammed Hazeem

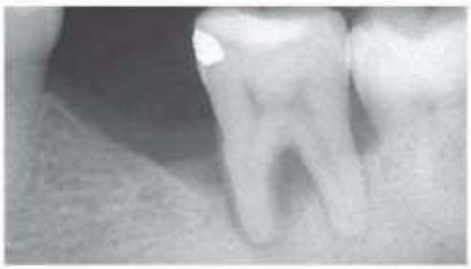
Differential Diagnosis

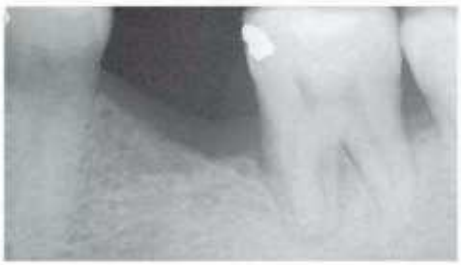
1. **Endodontic involvement**





2. **TFO**



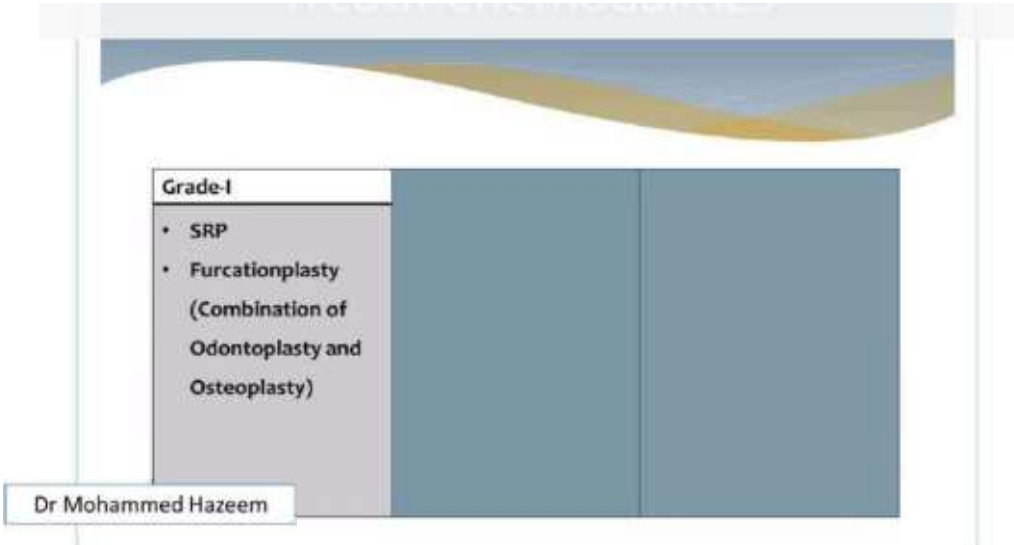


Dr Mohammed Hazeem

Treatment Aspect

- Main objectives are:

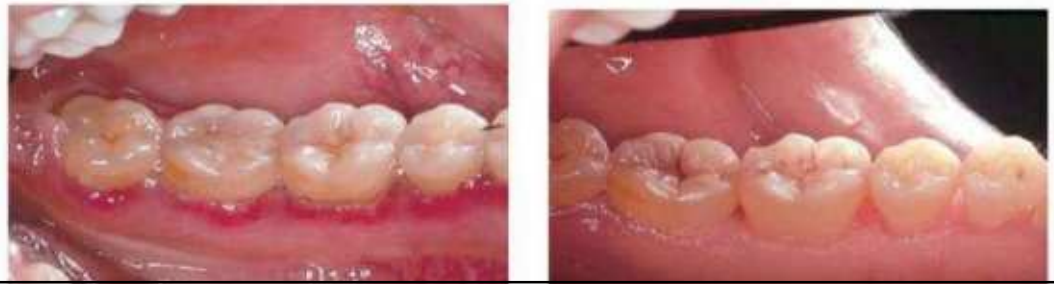
1. Elimination of the microbial plaque from root complex
2. Establishment of an anatomy to facilitates proper self-performed plaque control
3. Prevent further attachment loss



Non-surgical therapy

1. SRP

- Indicated for Grade- I and early grade- II



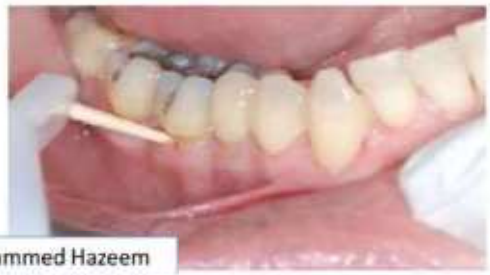
- **Advancements in non-surgical**- DeMarco curettes, diamond files, Quetin furcation curettes, and mini Five Gracey Curettes
- *Svärdström and Wennström (J Periodontol 2000)*

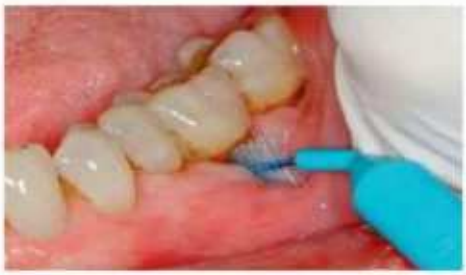
in the long term, furcations could be maintained over a 10-year period using NSPT.

Dr Mohammed Hazeem

Non-surgical therapy

2. **Oral Hygiene Procedures**
   - meticulous oral hygiene by the patient
   - rubber tips; periodontal aids; proxa toothbrushes.





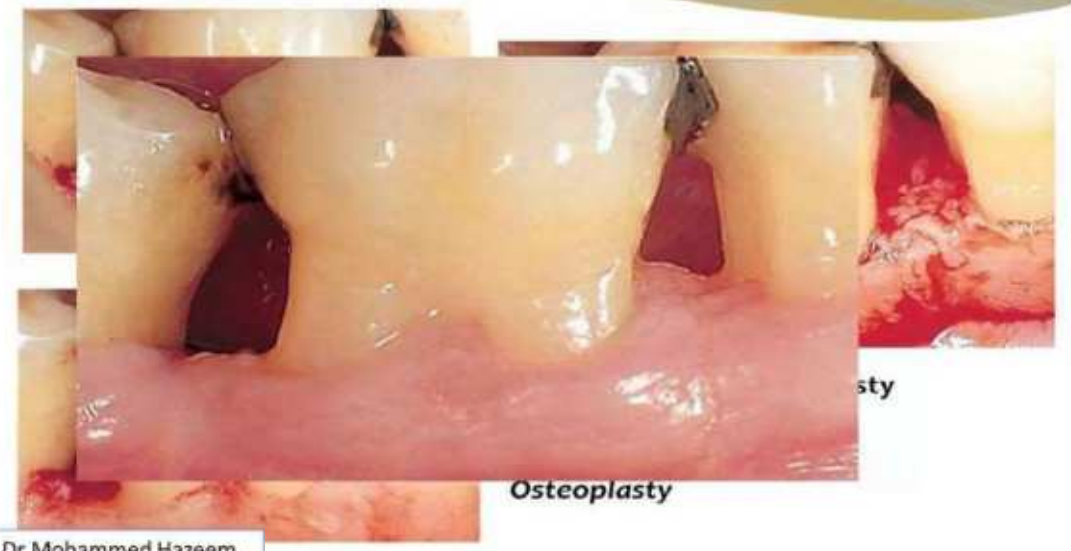
Dr Mohammed Hazeem

Surgical approach

1. **Furcation plasty**
   - First described by Hamp and colleagues (1975)
   - Early Grade-II
   - Result should be firm, well contoured papilla to cover the furcation defect.

Dr Mohammed Hazeem

Furcation plasty

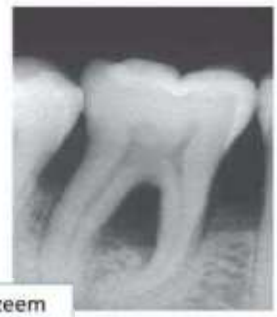


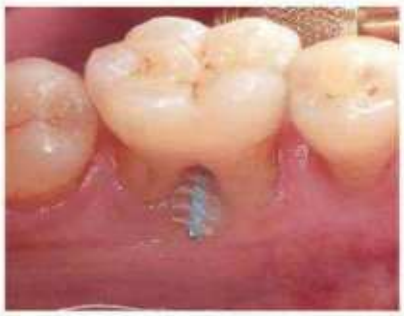
Osteoplasty

Dr Mohammed Hazeem

Tunnel preparati

- Indicated in deep grade- II and grade- III furcation defects in **mandibular molars.**
- Long and divergent roots (no possibility of regeneration)





Dr Mohammed Hazeem

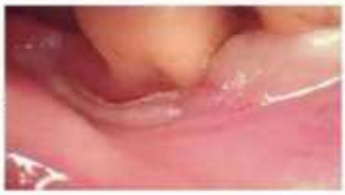
Regenerative procedures

- *Gottlow et al. (1986)* published first case rep. using GTR
- Most predictable results in grade- II *(Pontoriero et al. 1988; Lekovic et al. 1989; Caffesse et al. 1990)*
- Less predictable in grade-III and maxillary grade-II *(Pontoriero et al. 1989; Pontoriero & Lindhe 1995, Metzeler et al. 1991)*

Dr Mohammed Hazeem

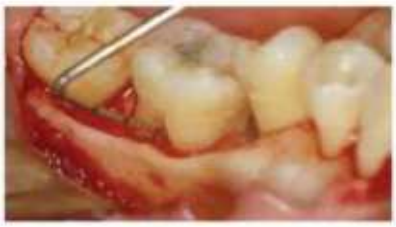
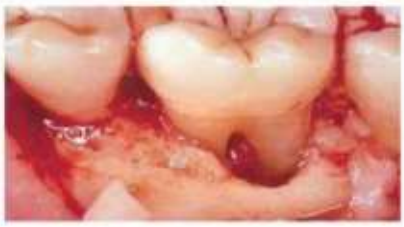
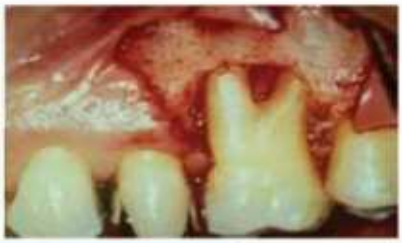
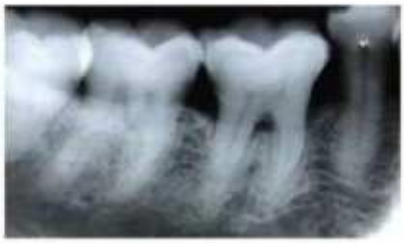
Why limited predictability ?

1. Horizontal type of furcation defects
2. Complex anatomy- poor debridement
3. Poor blood supply for graft material
4. recession of the flap margin and early exposure of both the membrane and fornix



Dr Mohammed Hazeem

GTR and grafti

Dr Mohammed Hazeem

Advancement in regeneration

Can Bone Lost from Furcations Be Regenerated?

Dent Clin N Am - (2015)

Joseph J. Zambon, DDS, PhD

Class II defects Maxillary and mandibular molars	Regenerative treatment predictable; periodontal regeneration demonstrated histologically and clinically
Class III defects Mandibular molars	One case report demonstrates periodontal regeneration histologically
Class III defects Maxillary molars	Regenerative therapy not predictable; shown only in clinical case reports

Dr Mohammed Hazeem

Root resection and separation

* ***Root resection***- involves the sectioning and the removal of one or two roots of a multirooted tooth.
* **Root separation**- involves the sectioning of the root complex and the maintenance of all roots.
* Indicated in deep grade- III and IV.

Indications

* By Bassarba et al.:

1. Teeth serving as abutments for prosthesis
2. Severe attachment loss on a single root
3. Teeth for which more predictable Rx is unavailable.
4. Teeth in patients with good oral hygiene and low caries activity

Dr Mohammed Hazeem

Contraindications

1. Poor C/R ratio on remaining roots
2. Unfavourable anatomy of retained roots
3. Long root trunks/ fused roots
4. Teeth in which Endo-Restorative Rx is not possible
5. Inability to perform oral hygiene
6. Splinting is not possible
7. Prosthetic factors

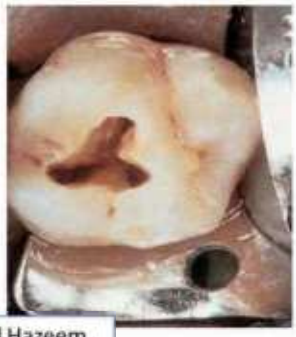
Which root to remove ?

1. root that will eliminate the furcation
2. with greatest amount of bone/attachment loss
3. Greatest number of anatomic problems:
   - Curvature, grooves, accessory canals
4. Least complicate the future periodontal maintenance

Dr Mohammed Hazeem

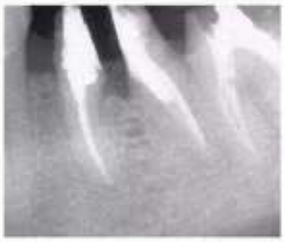
Sequence of treatment (carnevale 1981)

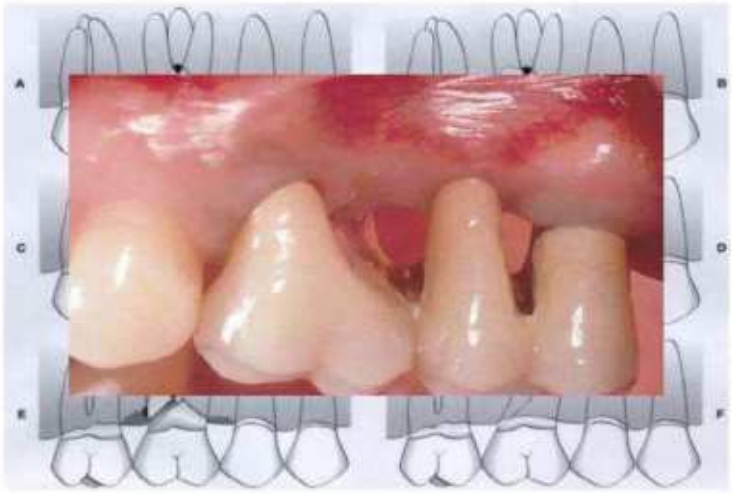
1. ***Endodontic treatment***
2. ***Provisional restoration***



Dr Mohammed Hazeem

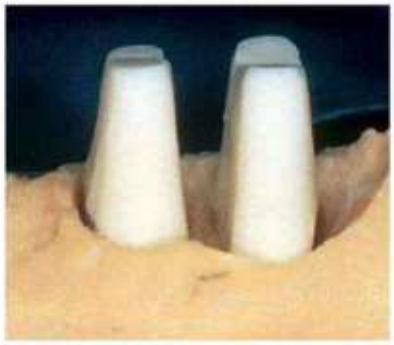
3. Root resection/ Hemisection

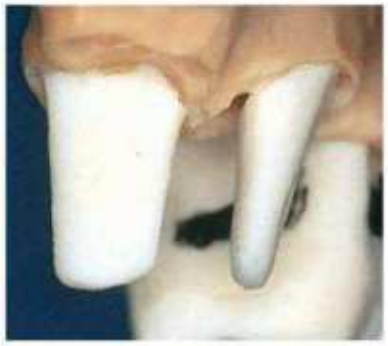




Dr Mohammed Hazeem

- performed as part of the preparation of the segment for prosthetic rehabilitation, that is prior to periodontal surgery (Carnevale *et al*. 1981).





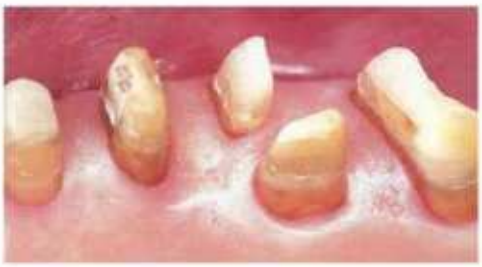
4. Periodontal surgery

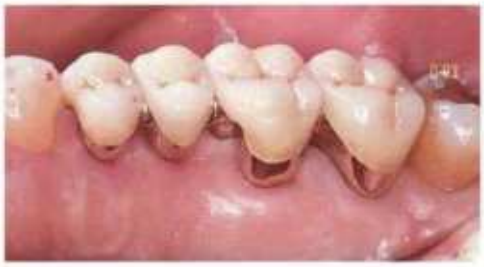
- osseous resective techniques are used to eliminate angular bone defects around the maintained roots.
- The provisional restoration is relined.
- The margins of the provisional restoration must end ≥3 mm coronal of the bone crest
- flaps are secured with sutures at the level of the bone crest.

Dr Mohammed Hazeem

5. Final prosthetic restoration

- After complete soft tissue and hard tissue healing (3months)





Dr Mohammed Hazeem

Extraction

- Extraction is better in grade- III and IV.
  - Inadequte plaque control
  - Can't commit to a maintenance programe
  - High caries activity
  - Poor socio-economic factor

Dr Mohammed Hazeem

Prognosis of Therapy

- In a 5-year study, **Hamp *et al.* (1975)** observed the outcome of treatment of 175 teeth with various degrees of furcation involvementOf
- 32 (18%) were treated by SRP alone, (12)
- 49 (28%) were subjected to furcation plasty (3)
- 87 teeth (50%), root resection (5)
- 7 teeth (4%) a tunnel had been prepared (4).

- **Hamp et al. 1992** 7-year study, 182 furcation- involved teeth.
- 57 had been treated by SRP alone
- 101 were treated by furcation plasty, and
- 24 were subjected to root resection or hemisection
- >85% of the furcations treated with SRP alone, or in conjunction with furcation plasty, maintained stable conditions

Dr Mohammed Hazeem

* *Carnevale et al. (1998)* in a 10-year prospective controlled clinical trial, demonstrated a 93% survival rate of root resected teeth similar to that of success rates of implants **(Fugazzato et al. 2001)**
* Greater than 65-70% rate of implants placed in poorer bone quality **(Engquist, Jaffin and Berman 1991)**

* Recently, ***Huynh-Ba et al. (2009)*** published a systematic review (22 publications)
* Reported tooth survival rates
* ***Non-surgical furcation therapy*:** 90.7–100% at the end of the observation period of 5–12 years.
* Grade- I : 99-100%
* Grade- II: 95%
* Grade- III & IV: 25%

Dr Mohammed Hazeem

- ***Surgical furcation therapy*** (i.e. flap with or without osseous resection, gingivectomy/gingivoplasty, but not including furcation odontoplasty): 43.1–96% at the end of an observation period of 5–53 years.
- ***Tunnel preparation***: 42.9–92.9% after 5–8 years of observation.

Dr Mohammed Hazeem

- ***Surgical resective therapy*** (i.e. root resection or root separation): 62–100% after an observation period of 5–13 years. Reported complications were mainly root fractures and endodontic failures.
- ***Surgical regenerative therapy*** (i.e. GTR, bone grafts): 62–100% after a period of 5–12 years.
- horizontal furcation depth reduction in most of the cases No complete furcation closure, especially in severely involved mandibular and maxillary molars.

Dr Mohammed Hazeem

Conclusion

- No clear scientific evidence that any given treatment modality is superior to the others.
- Treatment modalities are more predictable for grade- I and grade- II
- 4 keys for long term success

Dr Mohammed Hazeem

Thank You

LASERS IN DENTISTRY

[redacted]

[redacted]

Topics

- Introduction
- History
- Mechanism of Action
- Application of Lasers in Dentistry
- Advantages and Disadvantages of Lasers
- Safety Measures
- Examples of Dental Laser Therapies
- Video Presentations
- Conclusion

Introduction

AS

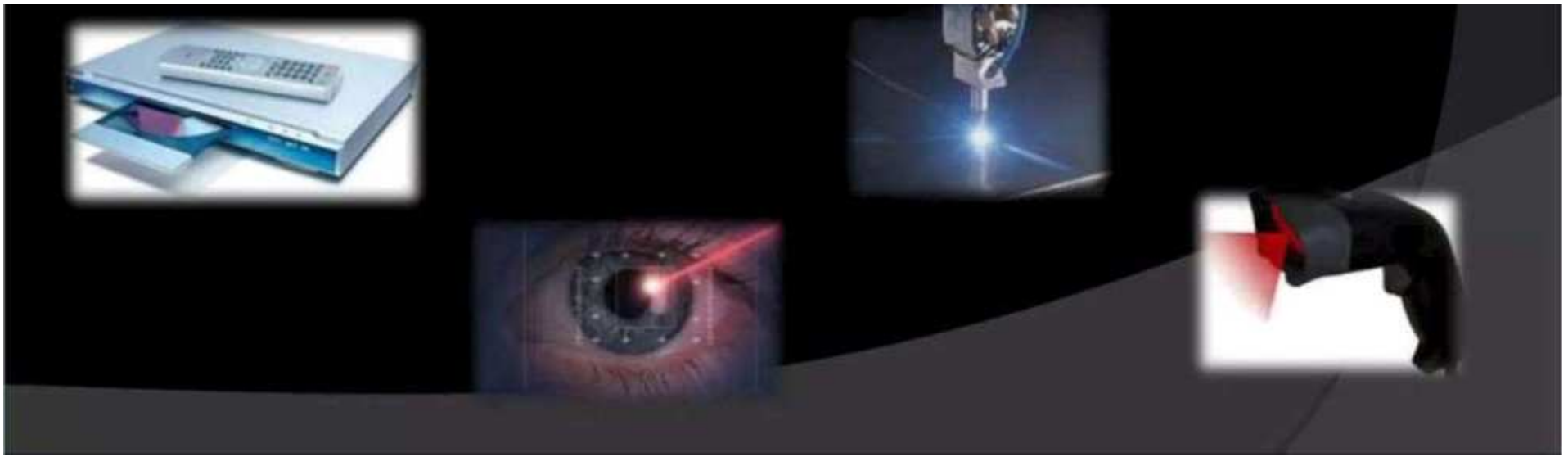
Light
mplification by
* timulated
Čmmission of
Radiation

- A device that emits light through a process of optical amplification based on the stimulated emission of electromagnetic radiation

Features: Spatially coherent, which means that either the light is emitted in a narrow, low divergence beam.

Many uses of lasers in daily life includes

- in consumer devices such as DVD players, laser printers, and barcode scanners
- in medicine for laser surgery and various skin treatments
- in industry for cutting and welding materials
- in military and law enforcement devices for marking targets and measuring range and speed



In Dentistry,

Laser dentistry	A precise and effecti\ie way to perform many dental procedures. The potential for laser dentistry to improve dental procedures rests by allowing for treatment of a highly specific area of focus without damaging surrounding tissues.

History

1917

Albert Einstein established the theoretical foundations for the laser *Quantum Theory of Radiation*.

1959

Gordon Gould published the term LASER in the paper *The LASER*.

May 16, 1960,

Theodore H. Maiman operated the first functioning laser, the Ruby laser at 694 nanometers wavelength

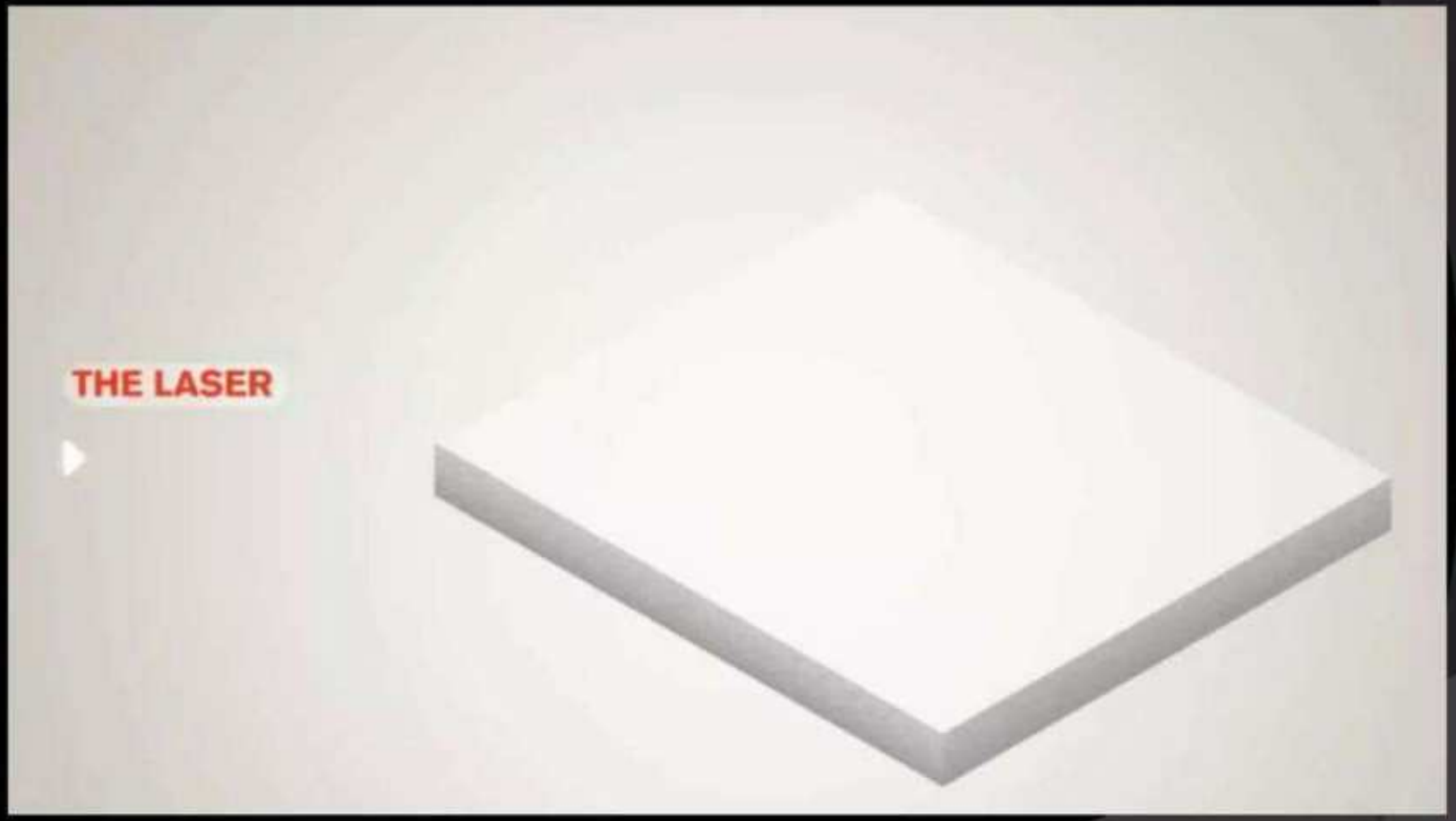
Dental Lasers

- **1993:** Nd:YAG Laser.
- **1994:** CO_2 Laser.
- **1996:** Laser welder.
- **1998** : Er:YAG Laser.

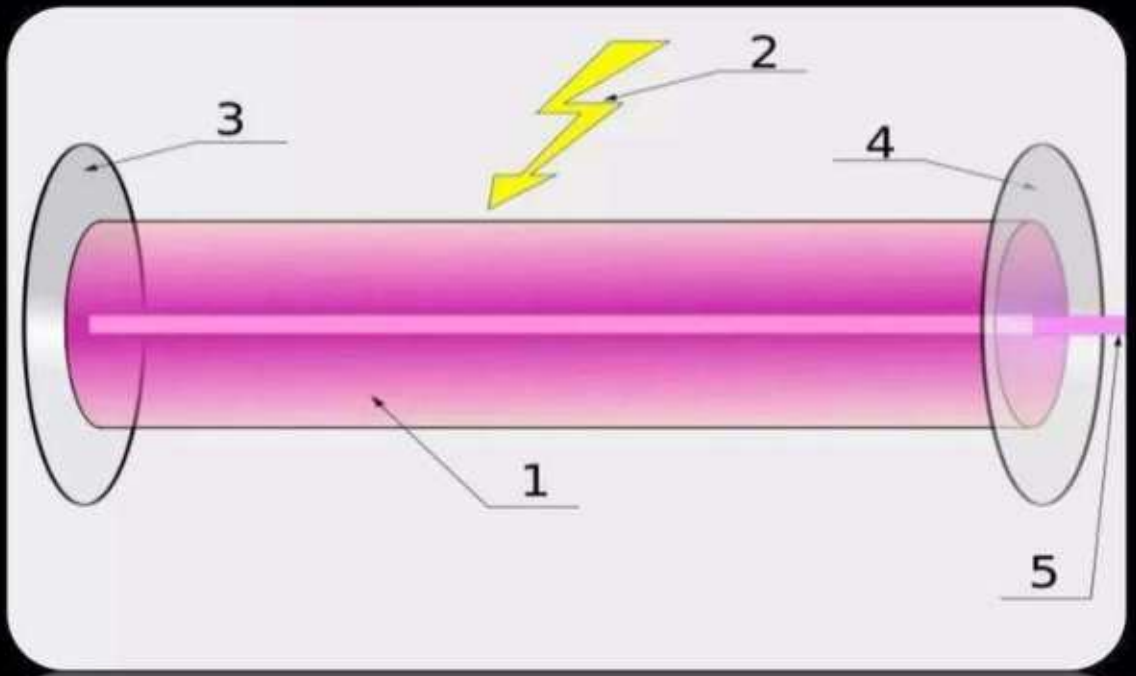
Mechanism of Action

- *Laser Physics*
- *Design of Laser Medium*
- *Tissue- Laser Interaction*

Laser Physics



Design of Laser Medium



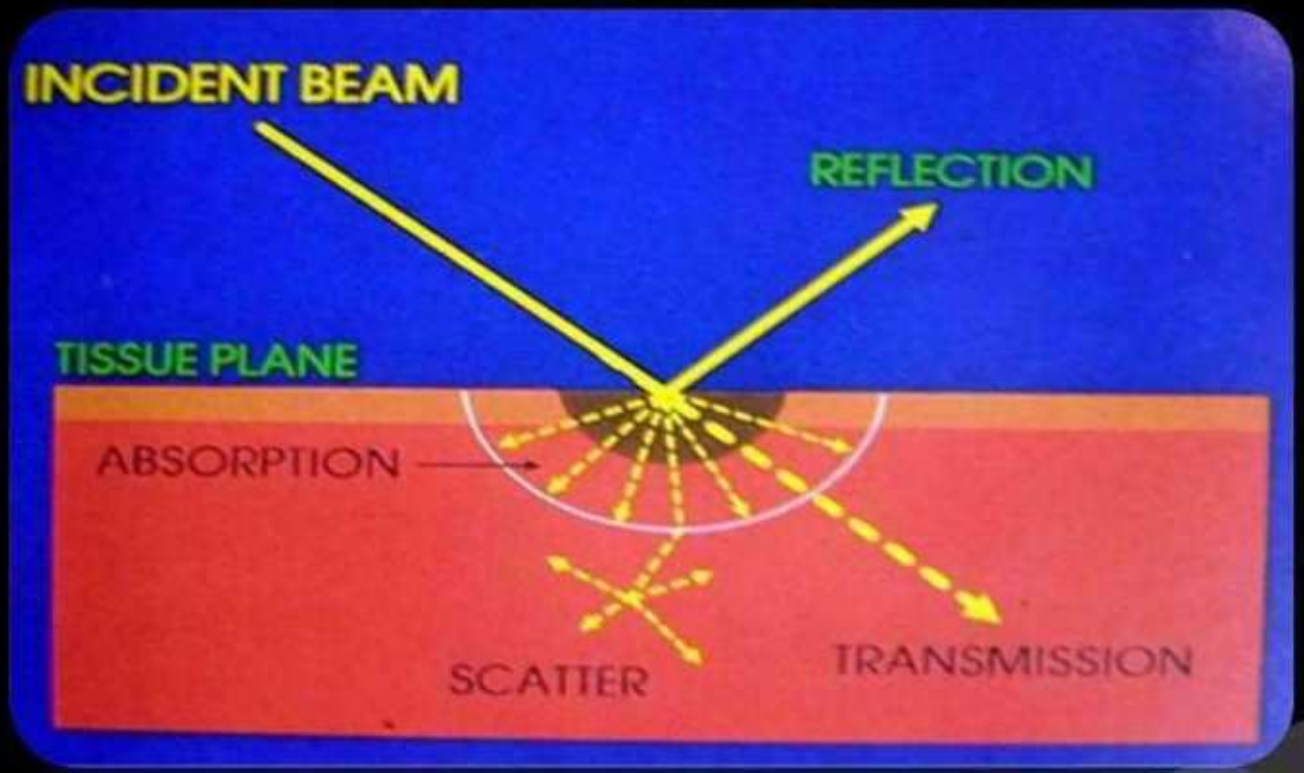
Components of a typical laser:

1. **Gain medium**
2. **Laser pumping energy**
3. **High reflector**
4. **Output coupler**
5. **Laser beam**

Tissue-Laser Interaction

Laser light can have four different interactions with the target tissue depending on the optical properties of that tissue

Different Laser-Tissue Interactions



- Using the principle of selective photothermolysis, these laser target different chromophores in the skin, which selectively absorb the laser o light energy as heat and yield the desired response.

Chromophores ubsorb the

Physical, mechanical, chem ical, temperature changes may occur

This energy travelsat different wavelcngth and is absorbed by a

Different Temperature Effects

Between 75°C and 100°C	Tissue shrinkage and dehydration → Vaporization and carbonization → Irreversible cell death
Between 55°C and 75°C	Increase of blood viscosity → Coagulation
Between 35°C and 55°C	Vasodilation and hyperthermia → Blood Supply

Applications of Lasers in Dentistry

The rapid development of laser technology has seen its introduction into various fields of den ist .

1. Diagnosis

- Detection of pulp vitality
- Doppler flowmetry
- Laser fluorescence- Detection of caries bacteria and dysplastic changes in the diagnosis Of cancer

2. Hard tissue applications

- Caries removal and cavity preparation
- Re-contouring of bone (crown lengthening)
- Endodontic (rOOt canal preparation ,sterilization and Apicectomy)
- Laser etching
- Caries resistance

3. Soft łissue applications

- Laser-assisted soft tissue curettage and peri-apical surgery
- Bacterial decontamination
- Gingivectomy and Gingivoplasty
- Gingival retraction for impressions
- Implant exposure

- Implant exposure

Soft tissue applications (cont.)

- · Biopsy incision and excision
- Treatment of aphthous ulcers and Oral lesion therapy
- Coagulation / Hemostasis
- Tissue fusion - replacing sutures
- Laser-assisted flap surgery
- Removal of granulation tissue
- Pulp capping, Pulpotomy and pulpectomy
- Operculectomy and Vestibuloplasty
- Incisions and draining of abscesses
- Removal of hyperplastic tissues and Fibroma

4. Laser-induced analgesia

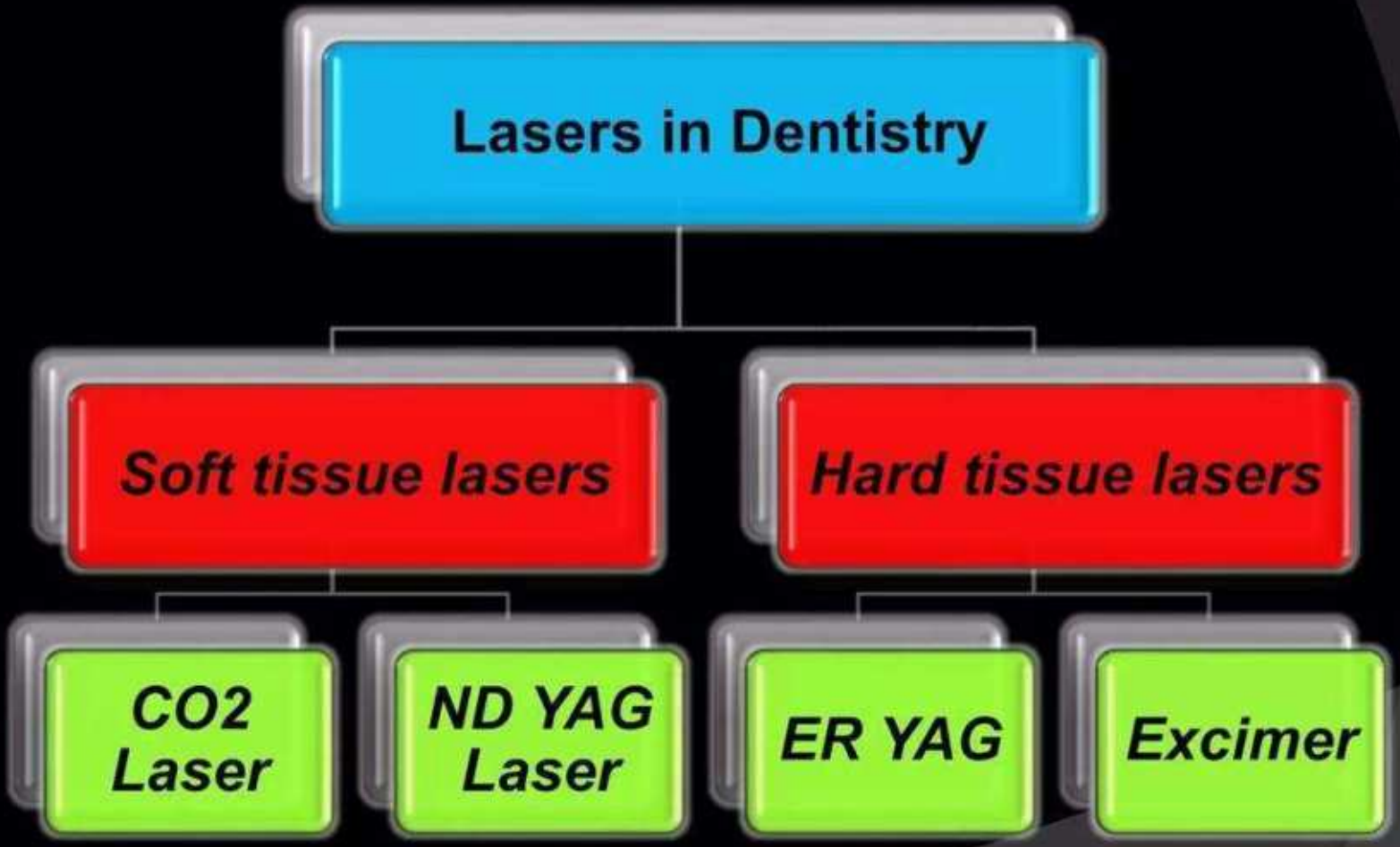
5. Laser activation

- Restorations (composite resin)
- Bleaching agents

6. Other

- Removal of rOOt canal filling material and fractured instrument
- · Softening gutta-percha
- Removal of moisture drying of canal

Types of Lasers Used in Dentistry



Carbon Dioxide Laser

Mode : vaporisatîon, cutting (>100 C)

a Specification : 10.6 micron wavelength

Used effectively in treating patients with oral lesions with blood dyscrasias.

a Oral indication:

a. Excision of pre-malignant lesions

b. Excision/biopsy

c. Hemiglossectomy

d. Adhesive microvascular/macro neural

Disadvantages :

-Cornea at risk

-Haemostasismay not be adequate on very vascular area (posterior tongue)

ND-YAG Laser (Neodymium doped-Yttrium Aluminium Garnet)

a Mode: coagulation (•60 c), central vaporisation

O Specification : 1.06 micron wavelength

Can be combined with CO2 (combo laser) or KTP

Oral indications:

a. Coagulation of very vascular lesions or near major blood vessel

b. Excision in vascular areas such as posterior tongue

d. Gingivectomy

e. Frenectomy

@ Disadvantages:

- Retina at risk

- Penetration could cause inadvertent spread

- Oedema more than CO2 laser

KTP Laser (Potassium Titanyl Phosphate)

a Modality : cutting with moderate coagulation

Specification : 0.53 micron wavelength

Can be combined with Nd-YAG laser

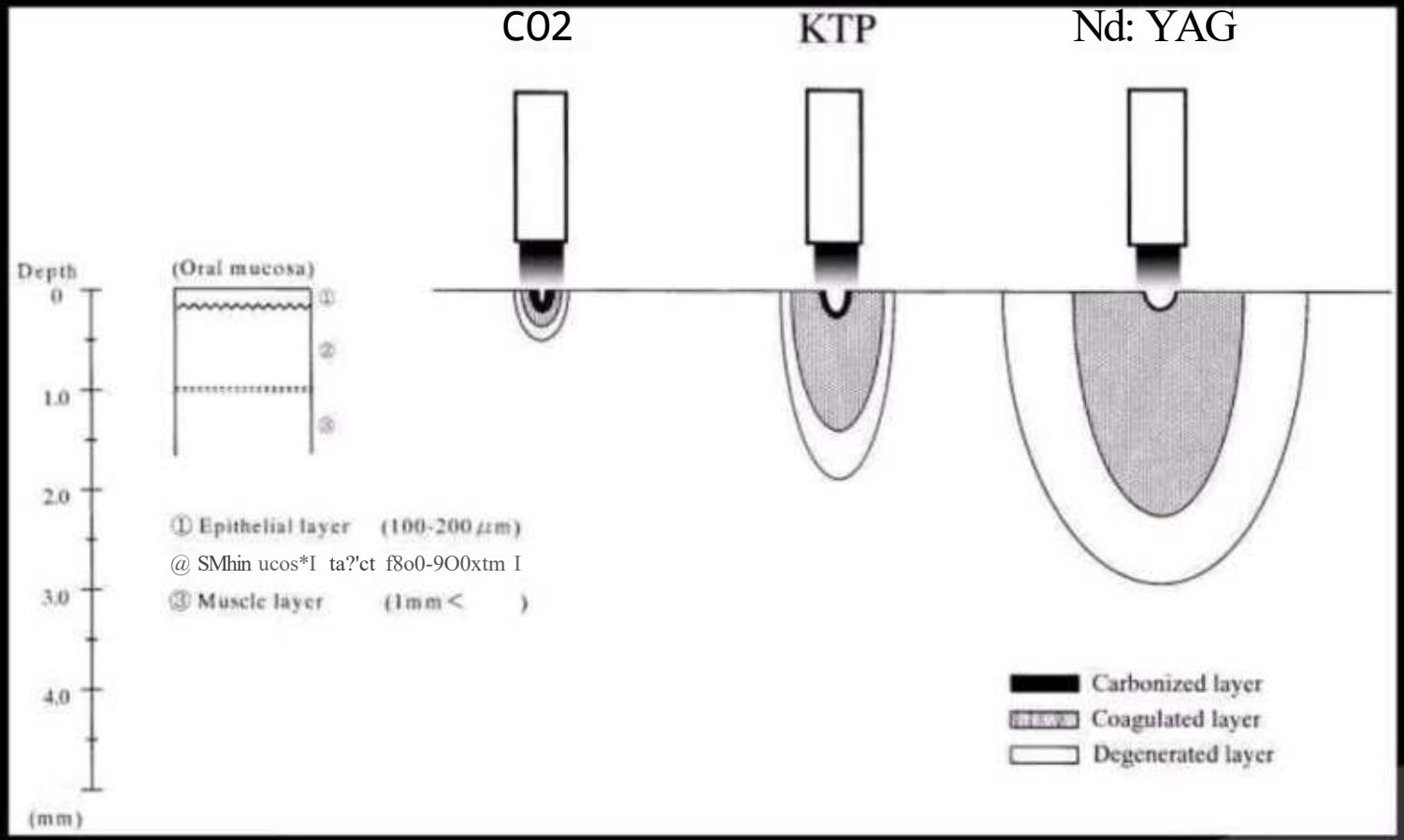
Oral indications

a. Excision in vascular areas, eg. Tonsillectomy

a Disadvantage

- Retina at risk

Comparison of The Lasers



Advantages and Disadvantages of Lasers

Advantages:

- Less pain in some instances (reducing the need for anesthesia)
- Reduce anxiety in patients uncomfortable with the use of the dental drill.
- Minimize bleeding (high-energy beam photocoagulation) and swelling.
- Reduce bacterial infections (sterilises the area being worked on)
- Preserve more healthy tooth during cavity treatment.

Disadvantages:

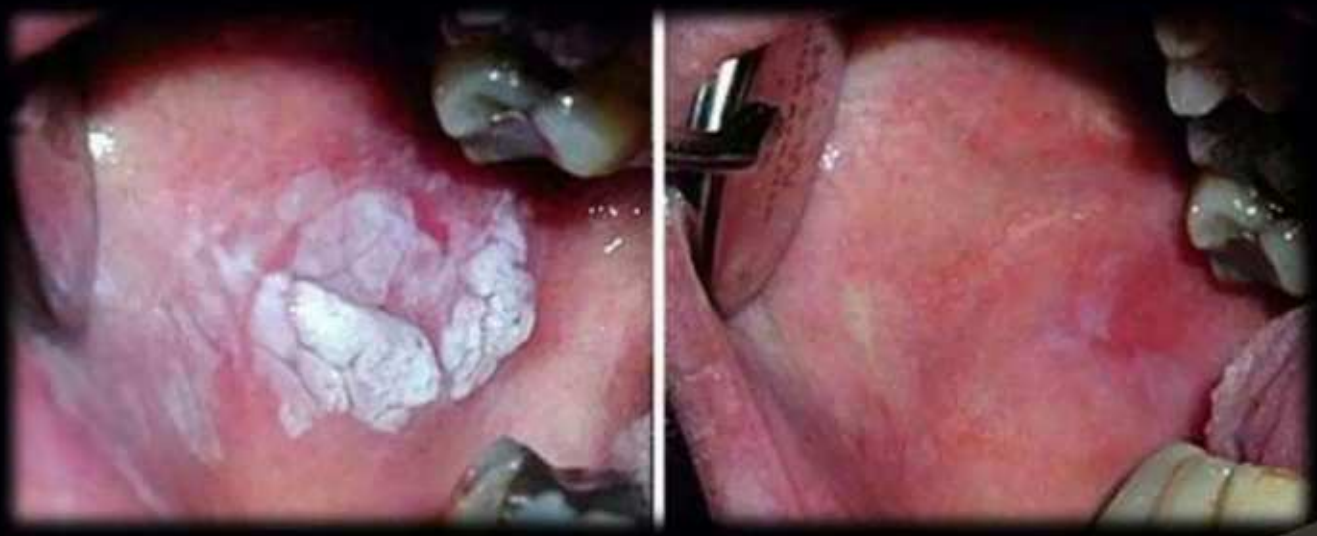
- Lasers can t be used on teeth with f flings that are already in place.
- Lasers can t be used n many commonly perfoi‘med dental procedures. Eg. lasers can t be used to 111 cavities located between teeth, cavit es around old f Il ngs, and large cavit es.
- Trad’t’onal dr’lls ray st11 be needed to shape the fi1l'ng, adjust the b te, and polish the Fll'ng evcn when a laser s used.
- Do not el'rn nate the need for anesthesia.
- More expens ve s nce the cost of the laser is much higher.

Precautions:

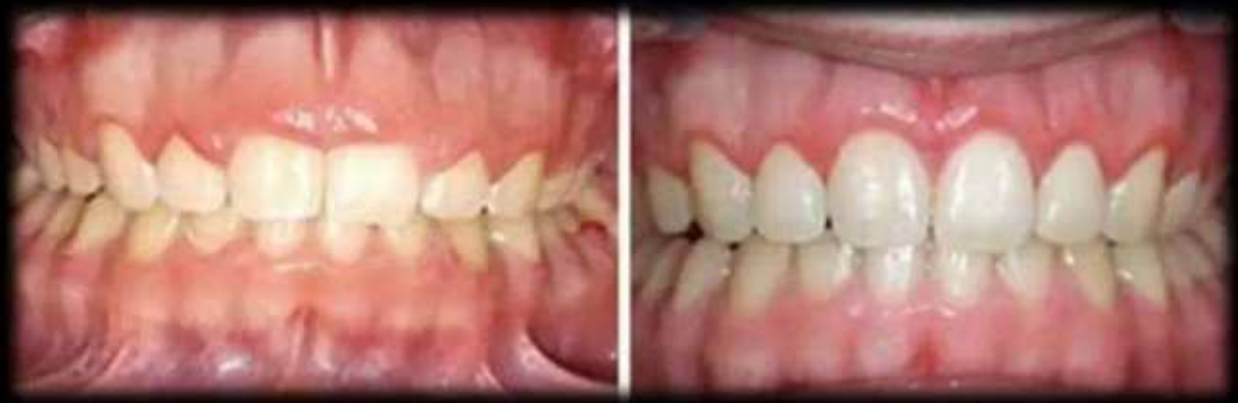
- Safety goggle (should be worn by patient and operator)
- Lock the door during the treatment
- Never look directly into the laser beam
- Never point the laser hand-piece at any person except at the treated area
- Never use the laser in the presence of flammable anaesthetics
- Never step on or abruptly bend the fibre optic cable
- Never move the laser machine during operation

Examples of Dental Laser Therapies

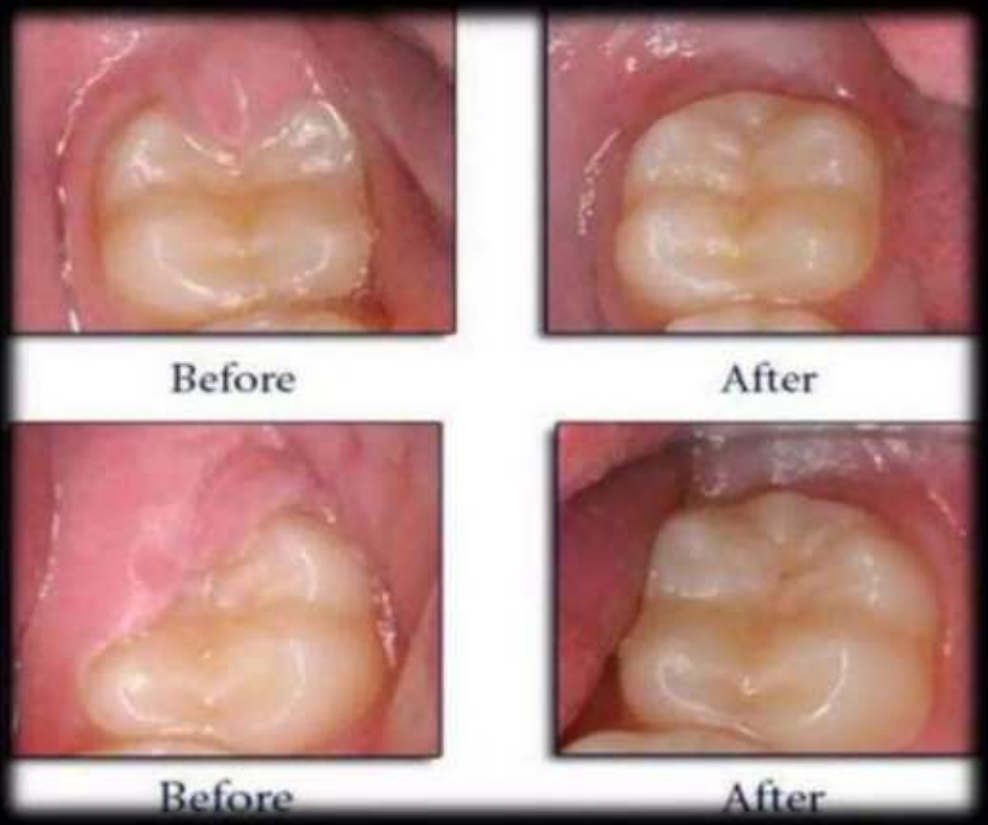
Leukoplakia



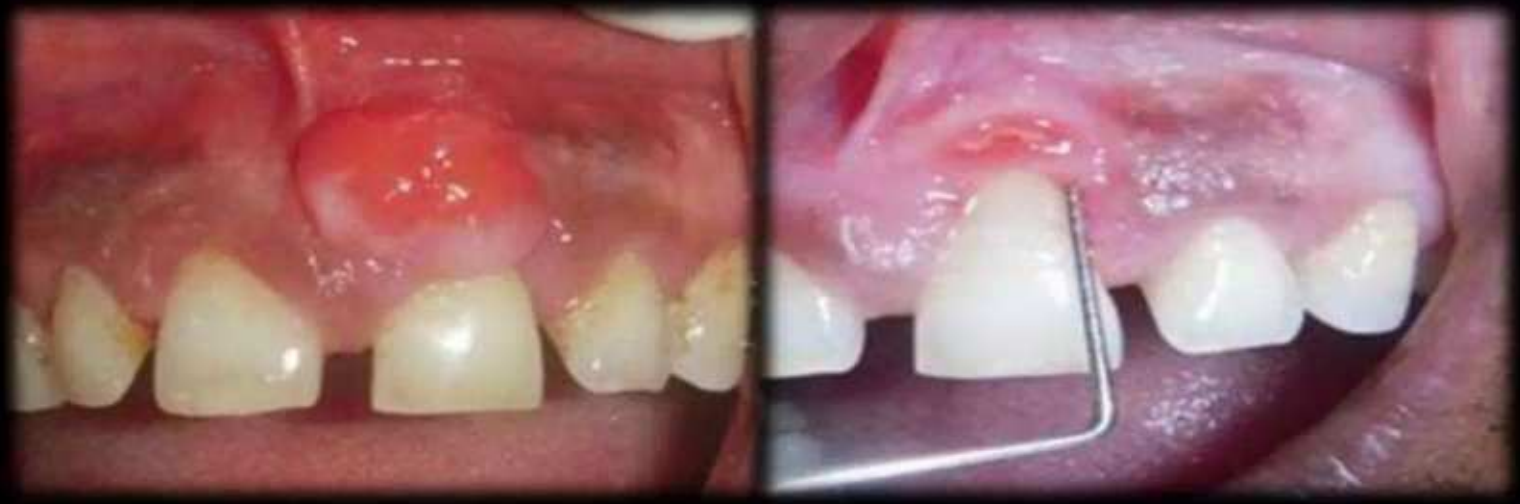
Gingivectomy



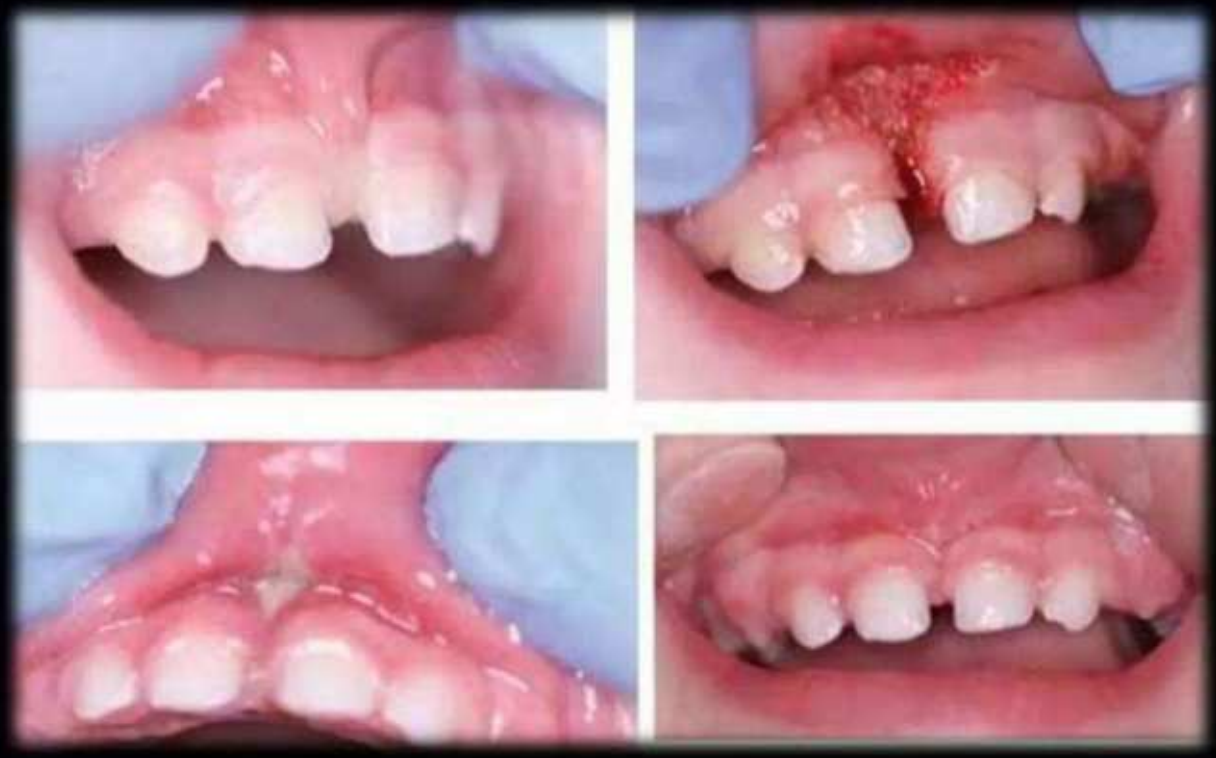
Operculectomy



Pyogenic Granuloma Excision



Frenectomy



Conclusion

Subject	Laser Surgery	Traditional Surgery
Anesthesia	No or mild	
Bleeding	No or minimal	Yes deoends on operate ng zone
Pain	Slight irritation	Sedation depended
Time	Less time	Time consuming
Suturing	No need	Yes in ii vasive *FOCe d u res*
Cost	Expensive	Less expensive
Postoperative complications	Minimal	

Subject	Laser Surgery	Traditional Surgery
Healing	Rapid	Slower
Method of pathology elimination	Dehydration and carbonization	Traumatic excision
Dentist comfort	Better	Slightly worse ‹depended›
Procedure	Complicated	More convenient
Team work	More	Less
Experience	Same	Same

Thank You.

Periodontal management of medically compromised patients

Periodontal management of medically compromised patients

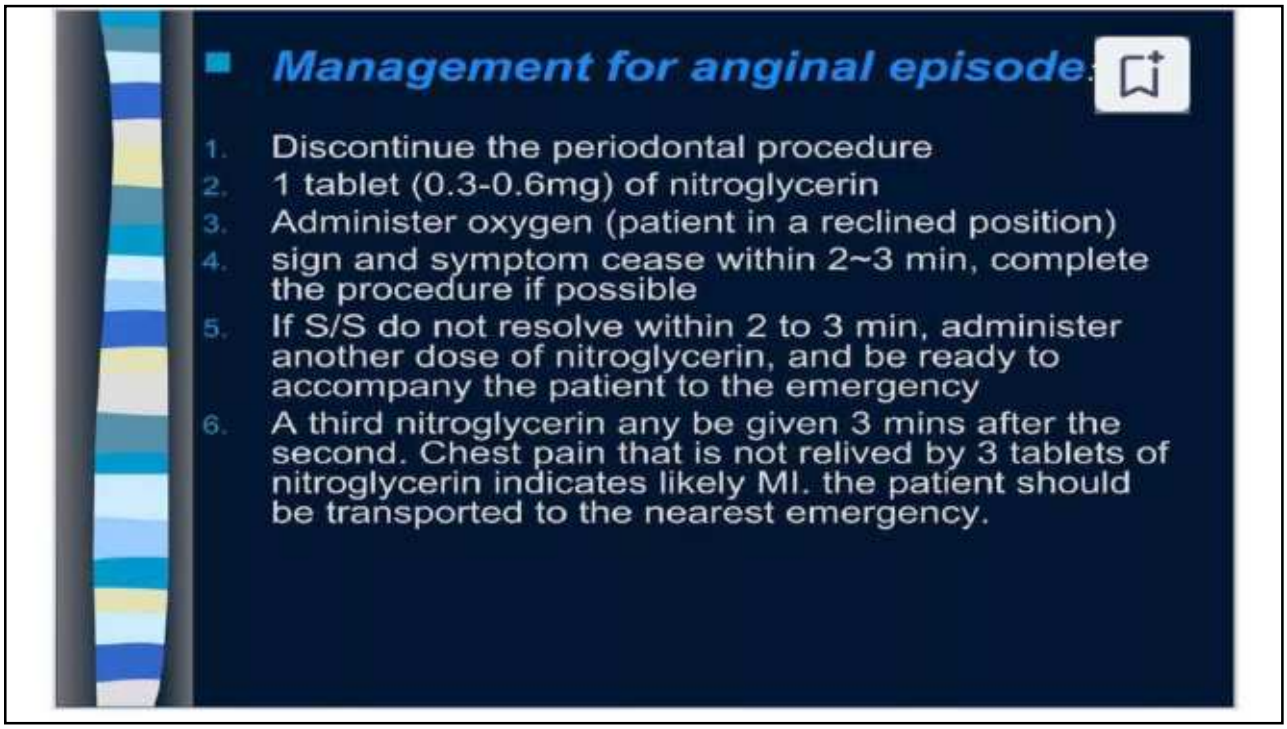
- CARDIOVASCULAR DISEASES
- RENAL DISEASES
- PULMONARY DISEASES
- IMMUNOSUPPRESSION AND CHEMOTHERAPY
- RADIOTHERAPY
- ENDOCRINE DISORDERS
- HEMORRHAGIC DISORDERS
- BLOOD DYSCRASIAS
- INFECIOUS DISEASES

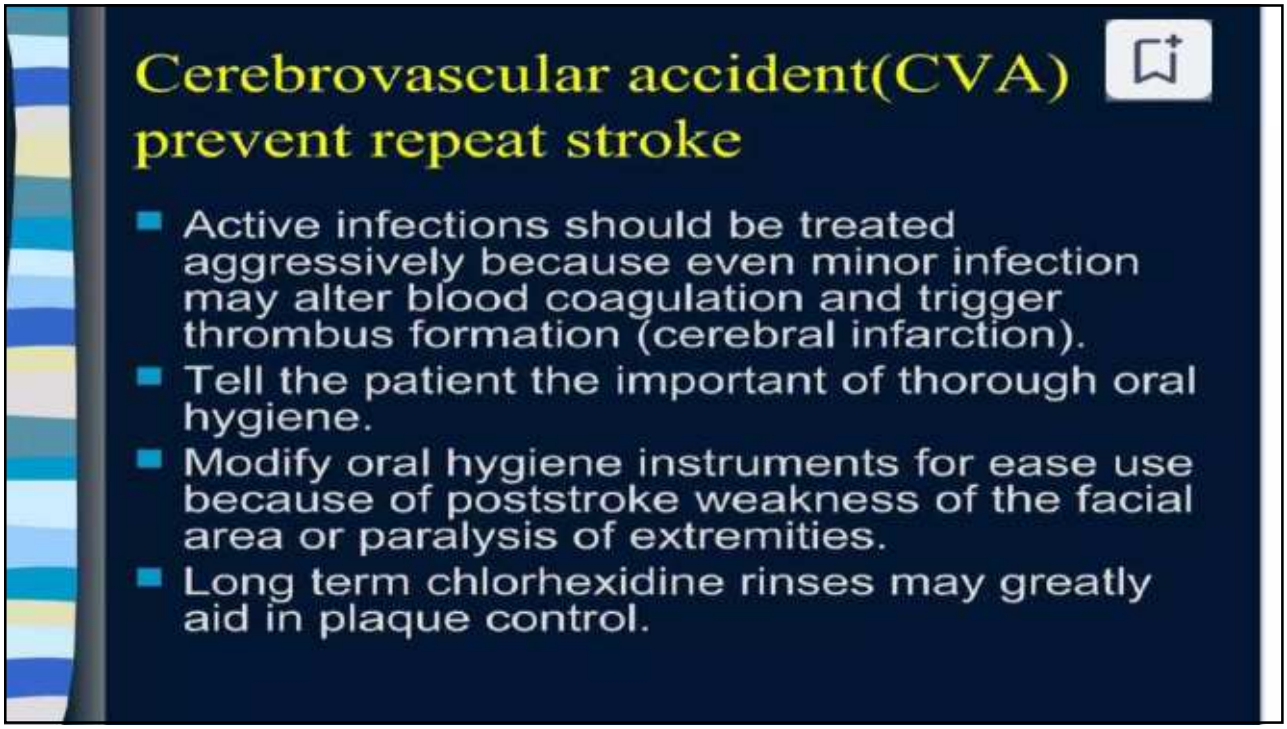
Cardiovascular disease

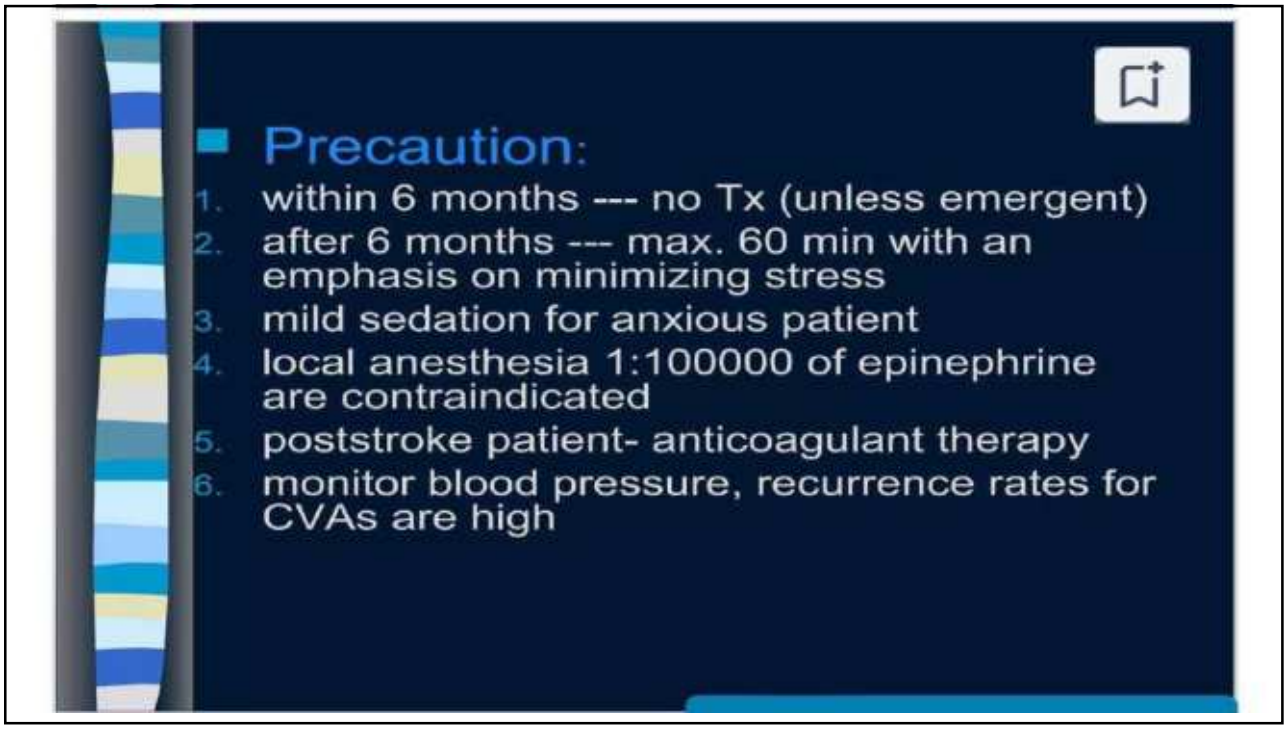
- Angina pectoris
- Arterial bypass
- Cerebrovascular accident (CVA)
- Congestive heart failure (CHF)
- Hypertension
- Infective (Bacterial) endocarditis

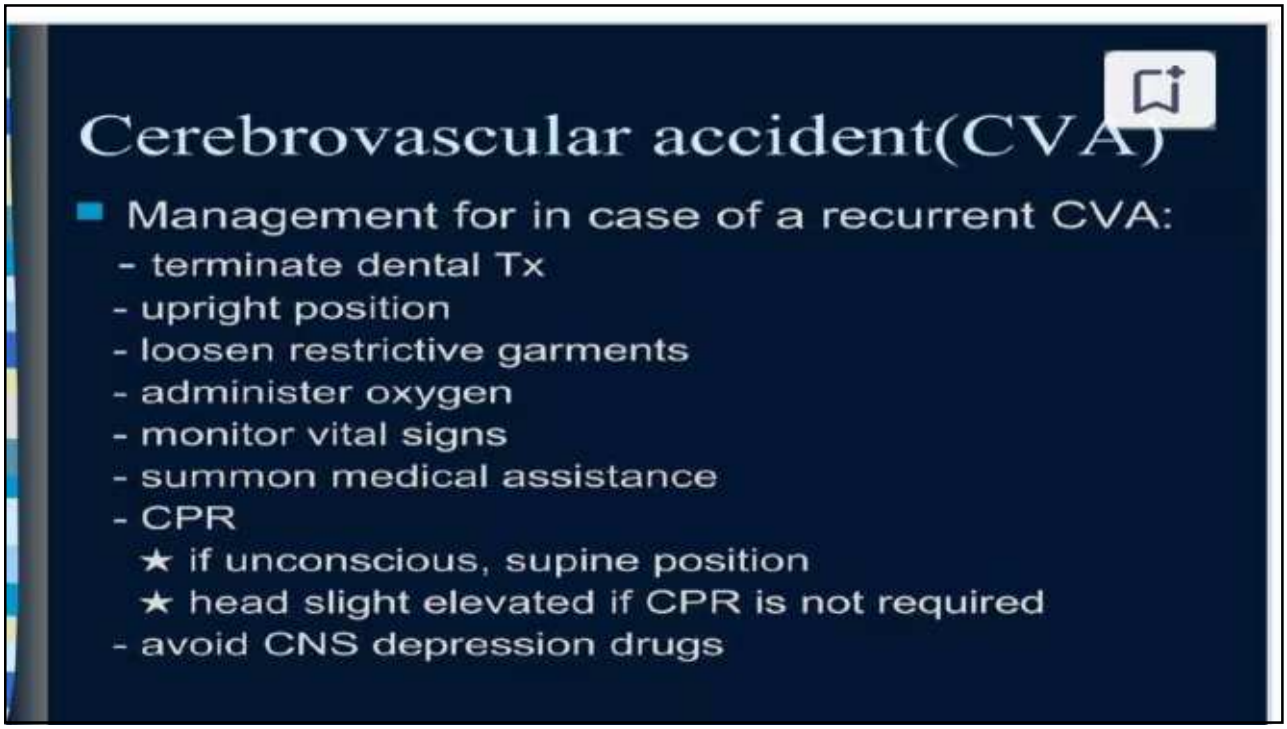
Angina pectoris

- *Unstable patient- emergencies only*
- *Stable patient- precaution for dental procedures:*
  - Premedication (conscious sedation)
  - Adequate anesthesia
  - Nitroglycerin.


Management for anginal episode:
1. Discontinue the periodontal procedure
2. 1 tablet (0.3-0.6mg) of nitroglycerin
3. Administer oxygen (patient in a reclined position)
4. sign and symptom cease within 2~3 min, complete the procedure if possible
5. If S/S do not resolve within 2 to 3 min, administer another dose of nitroglycerin, and be ready to accompany the patient to the emergency
6. A third nitroglycerin any be given 3 mins after the second. Chest pain that is not relived by 3 tablets of nitroglycerin indicates likely MI. the patient should be transported to the nearest emergency.

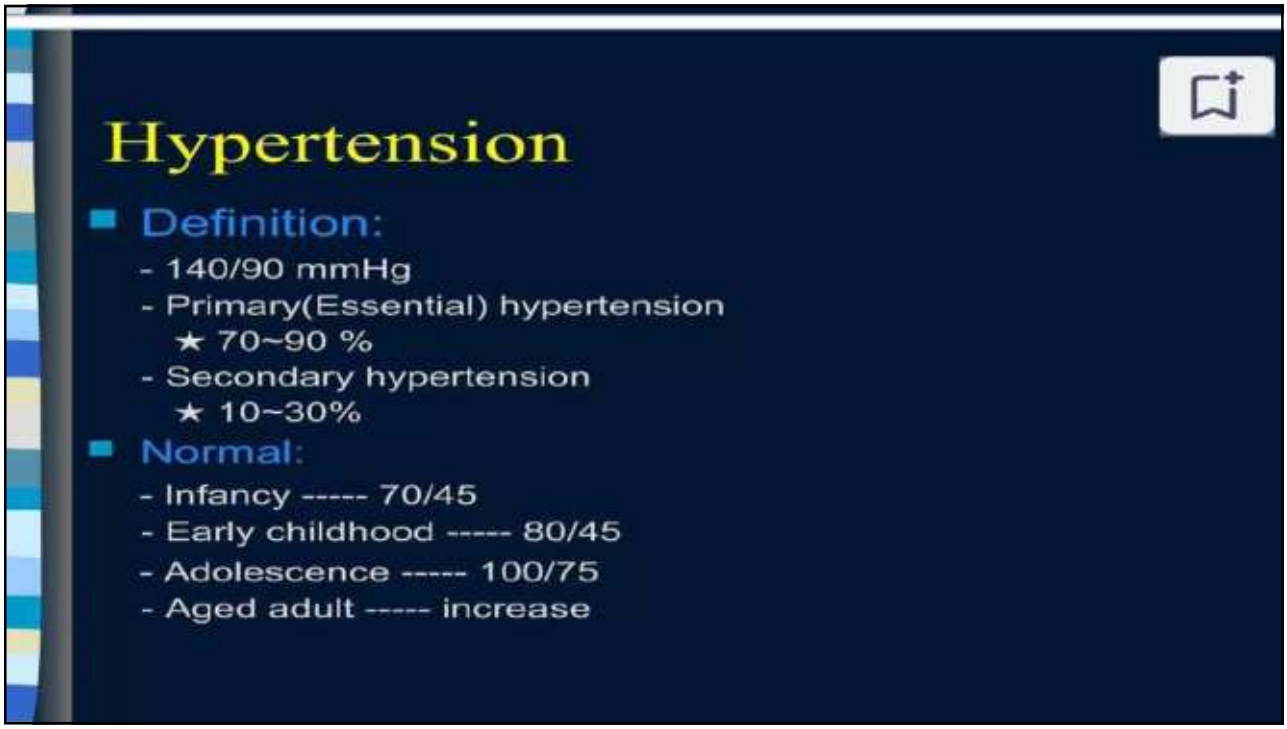

Cerebrovascular accident(CVA) prevent repeat stroke
Active infections should be treated aggressively because even minor infection may alter blood coagulation and trigger thrombus formation (cerebral infarction).
Tell the patient the important of thorough oral hygiene.
Modify oral hygiene instruments for ease use because of poststroke weakness of the facial area or paralysis of extremities.
Long term chlorhexidine rinses may greatly aid in plaque control.


Precaution:
1. within 6 months --- no Tx (unless emergent)
2. after 6 months --- max. 60 min with an emphasis on minimizing stress
3. mild sedation for anxious patient
4. local anesthesia 1:100000 of epinephrine are contraindicated
5. poststroke patient- anticoagulant therapy
6. monitor blood pressure, recurrence rates for CVAs are high


Cerebrovascular accident(CVA)
Management for in case of a recurrent CVA:
- terminate dental Tx
- upright position
- loosen restrictive garments
- administer oxygen
- monitor vital signs
- summon medical assistance
- CPR
★ if unconscious, supine position
★ head slight elevated if CPR is not required
- avoid CNS depression drugs


Congestive heart failure
CONGESTIVE HEART FAILURE IS THE CONDITION IN WHICH HEART IS UNABLE TO SUPPLY SUFFICIENT OXYGENATED BLOOD TO MEET THE BODY NEEDS.
MANAGEMENT
-100% OXYGEN
- Sitting upright position
- Record vital signs
- Rotating tourniquets high on four extremities
- reassure the patient
- Call for medical assistance


Congestive heart failure
Precaution for treated patient:
1. Medication- digitalis, diuretics, dicumarol, analgesics.
2. Presence of polycythemia, thrombocytopenia, leukopenia
antibiotics coverage and potential for bleeding problems
3. avoid dehydration
4. avoid flat reclining position
5. administer oxygen
6. stress reduction
7. avoid saline rinse- Na+ absorption


Hypertension
Definition:
- 140/90 mmHg
- Primary(Essential) hypertension
★ 70~90 %
- Secondary hypertension
★ 10~30%
Normal:
- Infancy ----- 70/45
- Early childhood ----- 80/45
- Adolescence ----- 100/75
- Aged adult ----- increase


Precaution:
- Avoid saline rinse
- Local anesthesia
---- Should not use greater than 1:100,000 epinephrine
------ No epinephrine - 30 mins
- Antihypertension drugs.
Non selective B blockers..
Carvedilol , Propanolol, Pindolol , Timolol .
Selective B blockers.
Atenolol, metoprolol, Bisoprolol.

- Dental treatment for H/T patient is generally safe as long as stress is minimized
- Morning appointment were once suggested. However, recent evidence indicated BP generally increases around awakening and peaks at midmorning. Lower BP levels occur in the afternoon; therefore, afternoon dental appointment may be preferred.

Infective Endocarditis

- *Definition:*
- ***Infective Endocarditis is the disease in which the micro organisms colonize the damaged endocardium or heart valves.***

Acute BE
- normal cardiac tissue
- non-hemolytic streptococci
- strains of staphylococci

Subacute BE
- damaged cardiac tissue
- pathogen organisms (α-hemolytic streptococci) (eg. Streptococcus viridans)

Infective Endocarditis

- Precaution:

1. define the susceptibe patient
2. poor oral hygiene- increase bacteremia: oral hygiene instruction (OHI) should begin with gentle procedure, as the gingival health improves, more aggressive OH may be initiated
3. During periodontal treatment, antibiotic prophylactic regimens should be practiced with all susceptibe patients

★antibiotic prophylaxis- it therefore is recommended that an alternate regimen be followed instead.

patient currently taking a penicilline agent after regenerative therapy may be placed on azithromycine before the next periodontal procedure

patient with EOP are also at risk for IE, Slot et al. suggested using tetracycline, 250mg, 4 times daily for 14 days to eliminated or reduce A.a.

Infective Endocarditis

- Management:

1. all periodontal procedures require antibiotic prophylaxis. Teeth with severe periodontitis and a poor prognosis may require extraction. Teeth with less severe involvement in a motivated patient should be retained, treated, and maintained closely.
2. pretreatment chlorhexidine rinses are recommended before all procedures, including probing.
3. allow at least 7 days between appointment (preferably 10-14 days). If this is not possible, select an alternative antibiotics regimen for appointment within a 7-day period.
4. If patient are placed on such regimens, the dosages are inadequate to prevent endocarditis during ensuing appointment. (if a patient was placed on 250mg of amoxicillin 3 times a day for 10 days after surgery and was returning for more treatment on the 7th day, ther patient would still require a full 2.0g dose of amoxicillin before that treatment
5. The need for antibiotic prophylaxis before suture removal is controversial. Chromic gut that resorbs in a short time may be indicated for patient at risk of IE.
6. regular recall on oral hygiene reinforcement


Renal disease
It is preferable to treat a patient dentally prior to rather than after transplant or dialysis.
Precaution:
1. Consultation
2. monitoring BP (end-stage are usually H/T)
3. check laboratory data:PTT, PT, bleeding time, platelet count.
4. eliminate oral infection to prevent systemic infection
Good oral hygiene
Periodontal treatment aim at eliminating inflammation and infection and providing easy maintenance, questionable teeth should be extracted
Frequent recall
5. nephrotoxic drugs :tetracycline, streptomycin, aminoglycoside antibiotics should not be given
acetaminophen may be used for analgesia and diazepam for sedation. Local anesthetics such as lidocaine are generally safe
6. hemodialysis
★ anemia
★ secondary hyperparathyroidism


Renal diseases
Management:
- screen serum hepatitis B, C antigens and antibody before treatment
- antibiotic prophylaxis endarteritis of the arteriovenous fistula or shunt
- prevent hypoxia
- provide the treatment the day following dialysis
★ 3 times/week
- long-term maintenance
- protect the dialysis shunt or fistula
- refer to physician if uremic problems


Immunosupression and chemotherapy
Leukopenia, alternations in cellular immunity and in inflammatory response may facilitate secondary infection.
Purpose:
- prevent oral complications that would be life threatening


Radiotherapy
Side effects:
- mucositis
- dermatitis
- xerostomia
- dysphagia
- gustatory alteration
- radiation caries
- vascular changes
- trismus
- TMJ degeneration
- periodontal changes

Radiotherapy

- Extraction indications: 10 days to 2 weeks prior to the initiation of radiation therapy
  - periodontal support
    less than ½ root length
  - nonrestorable
  - abscessed
  - oral hygiene and motivation are poor
  - furcation involement

Endocrine disorders

- Diabetes
- Thyroid disorders
- Parathyroid disorders
- Adrenal insufficiency
- Pregnanancy

Diabetes

- Precaution:
  - avoid periodontal treatment in uncontrolled DM
- Suspected patient:
  - consultation
  - analysis of laboratory tests
  - rule out acute orofacial infection or severe dental infection: only antibiotic and anagelsic care until diabetic control is attained. If a periodontal condition need immediate care, antibiotic coverage is required prior to I & D

Diabetes: Management

- Well-controlled DM: Tx as normal patient
  - **phase I therapy**

1. insulin and after a meal
2. morning appointment


Thyroid disorders
Hyperthyroidism:
- may cause tachycardia and other arrhythmias, increased cardiac output, and MI. medications such as epinephrine and other vasopressor amines should be given with caution
- treat so as to limit stress and infection
Hypothyroidism
- carefully given of sedatives and narcotics because of the potential for excessive sedation
- if surgery under GA
25% dose required for the euthyroid patient


Parathyroid disorders
Precautions:
- without medical care may be
★significant renal disease
★uremia
★hypertension
★cardiac arrhythmias
(hypercalcemia or hypocalcemia)
Properly treated medically routine periodontal therapy may be instituted.

Adrenal insufficiency

- Primary adrenal insufficiency (addison's disease) or secondary adrenal insufficiency (most often caused by use of exogenous glucocoticosteroid)
- Exogenous steroids may suppress the HPA axis (hypothalamic-pituitary-adrenal) and impair the patient's ability to response to stress, leading to the potential for acute adrenal crisis
- Such prophylaxis systemic steroids supplementation may not be required for many periodontal procedures. Adrenal crisis is rare in dentistry, especially when associated with secondary adrenal suppression caused by steroid use.
- Shapiro et al. found that patients taking 5 to 20mg/day prednisone maintained at least some adrenal reserve after immediate termination of steroid therapy.
- Use of a stress reduction protocol and profound local anesthesia may help minimize the physical and psycho logic stress associated with therapy and reduce the risk of acute adrenal crisis.

Adrenal insufficiency

- Suspected adrenal suppression: patient with a past history of steriod therapy-**Malamed's "Rule of twos"**
  - ★ 20 mg cortisone / day
  - ★ via oral or paraenteral route continuously over 2 weeks
  - ★ and within 2 years of dental therapy
- Minimum 12 months after the last dose before periodontal therapy is performed: regeneration of cortical function may occur within 9 to 12 months but 2 years has been reported.
  - ★ otherwise, steroid prophylaxis may be warranted

Adrenal insufficiency

- Management for patient is currently receiving steroid therapy:

  1988, Little and Falace:

  - no supplementation necessary
    - ★ low-dose(less than 20 mg cortisol/day)
    - ★ large-dose(more than 20 mg cortisol/day, for less than one month)
    - ★ topical steroids
  - more than 20 mg cortisol/day: 2 to 3 times of normal maintenance dose the morning of, and 1 hr before the procedure.

Pregnancy

- Precaution:
  - 2nd trimester
    - ★ the safest time for Tx
    - ★ only nonemergent periodontal treatment
  - 3rd trimester
    - ★ supine hypotensive syndrome of pregnancy, allow patient to change positions frequently

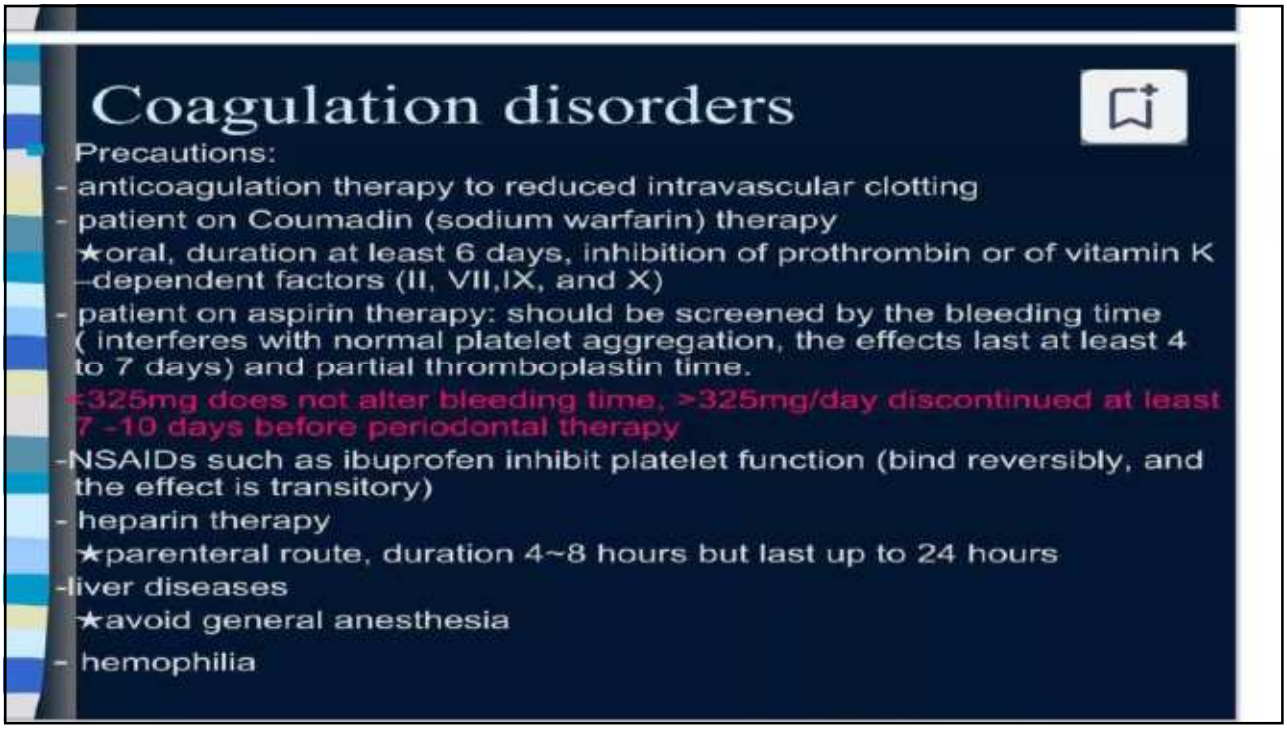
Pregnancy

- Management:
  - consultation if a drug
    - ★ could cross the placenta
    - ★ cause fetal respiratory depression
  - no medication or radiograph ideally

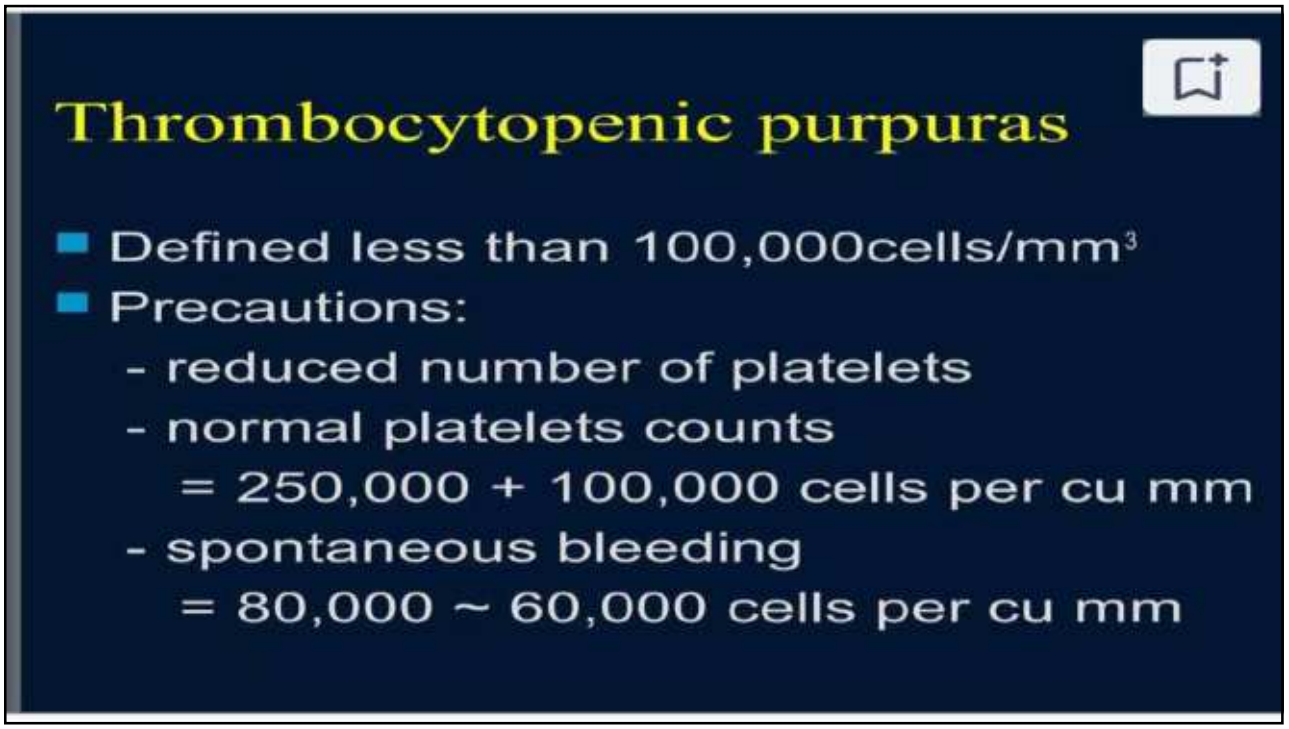
The ADA has stated that " normal radiographic guidelines do not need to be altered because of pregnancy" use of a properly positioned lead apron is an absolute requirement.

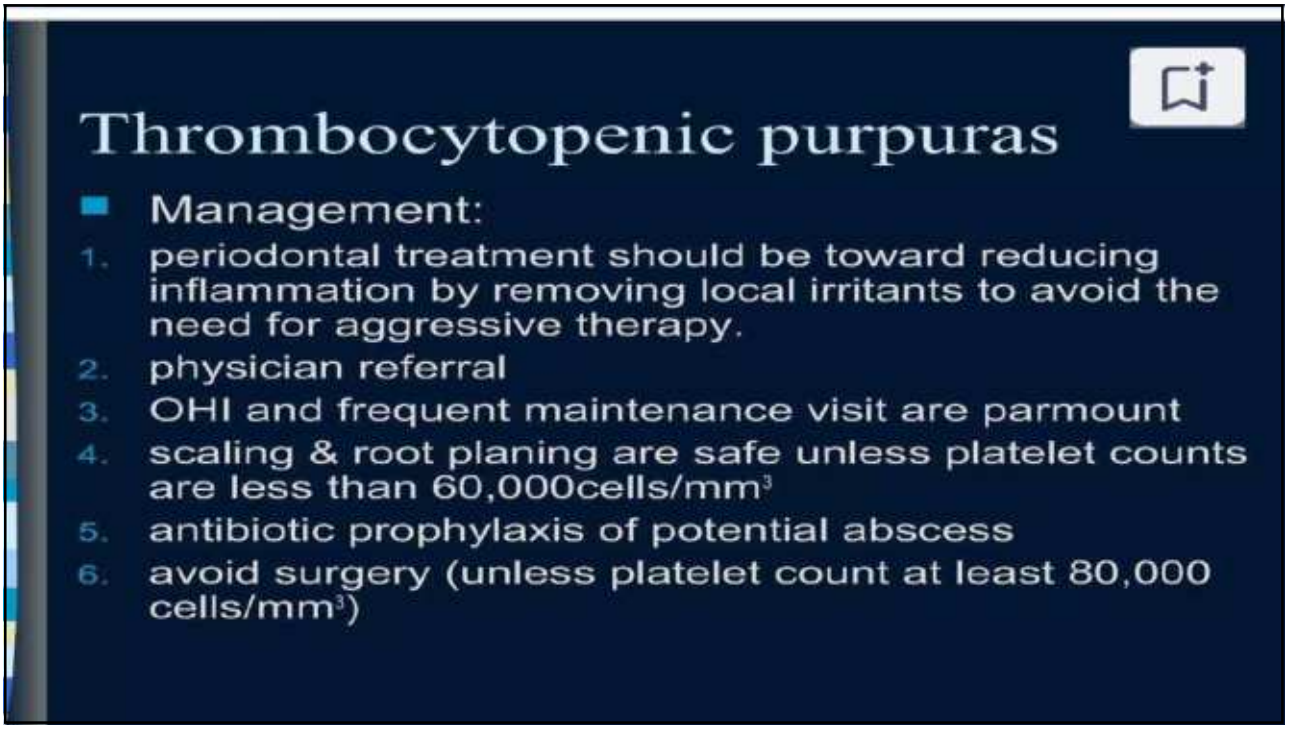
Hemorrhagic disorders

- Coagulation disorders
- Thrombocytopenic purpuras
- Nonthrombocytopenic purpuras


Coagulation disorders
Precautions:
- anticoagulation therapy to reduced intravascular clotting
- patient on Coumadin (sodium warfarin) therapy
★oral, duration at least 6 days, inhibition of prothrombin or of vitamin K –dependent factors (II, VII,IX, and X)
- patient on aspirin therapy: should be screened by the bleeding time (interferes with normal platelet aggregation, the effects last at least 4 to 7 days) and partial thromboplastin time.
≤325mg does not alter bleeding time, >325mg/day discontinued at least 7 -10 days before periodontal therapy
-NSAIDs such as ibuprofen inhibit platelet function (bind reversibly, and the effect is transitory)
- heparin therapy
★parenteral route, duration 4~8 hours but last up to 24 hours
-liver diseases
★avoid general anesthesia
- hemophilia


Coagulation disorders
Management: (continue)
- small segments may be treated in dental office
★ minimize trauma
★ antibiotic prophylaxis
★ use pressure hemostasis
★ no contraindication to local anesthesia with epinephrine --- caution of hematoma formation.
★ periodontal packing + cotton pellets


Thrombocytopenic purpuras
Defined less than 100,000cells/mm3
Precautions:
- reduced number of platelets
- normal platelets counts
= 250,000 + 100,000 cells per cu mm
- spontaneous bleeding
= 80,000 ~ 60,000 cells per cu mm

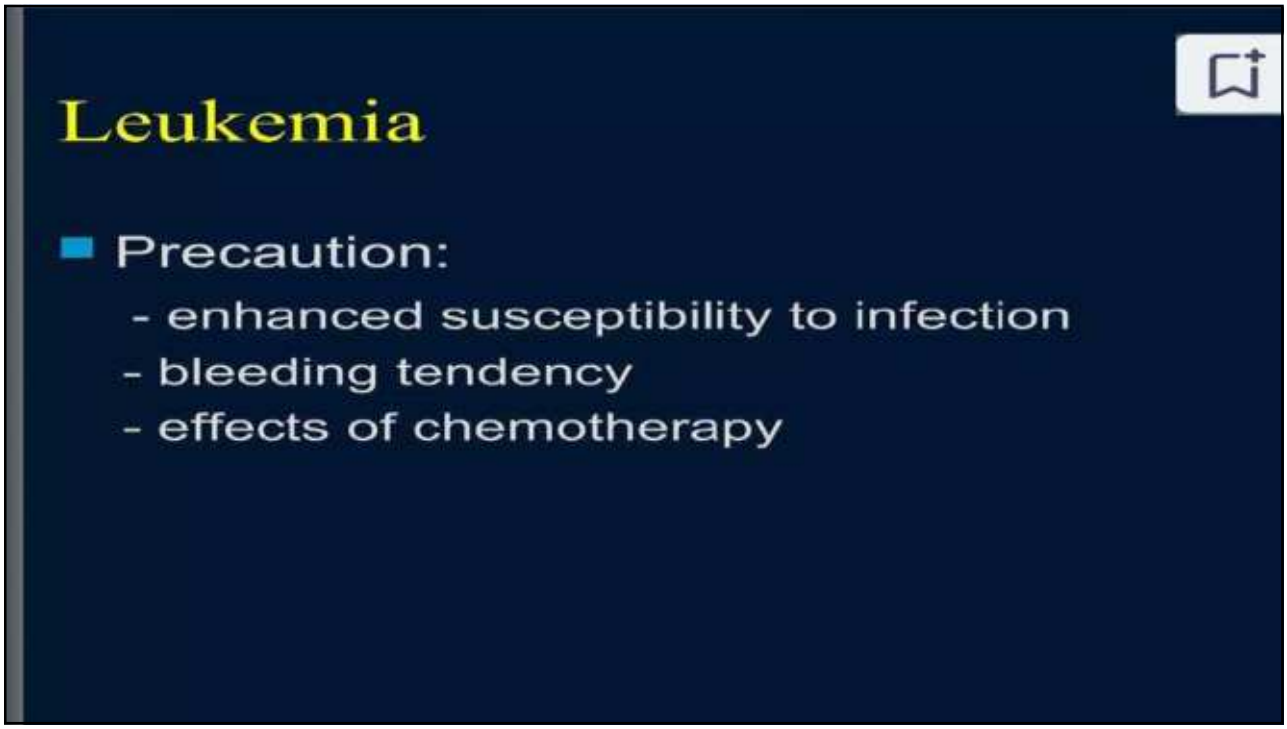

Thrombocytopenic purpuras
Management:
1. periodontal treatment should be toward reducing inflammation by removing local irritants to avoid the need for aggressive therapy.
2. physician referral
3. OHI and frequent maintenance visit are parmount
4. scaling & root planing are safe unless platelet counts are less than 60,000cells/mm3
5. antibiotic prophylaxis of potential abscess
6. avoid surgery (unless platelet count at least 80,000 cells/mm3)

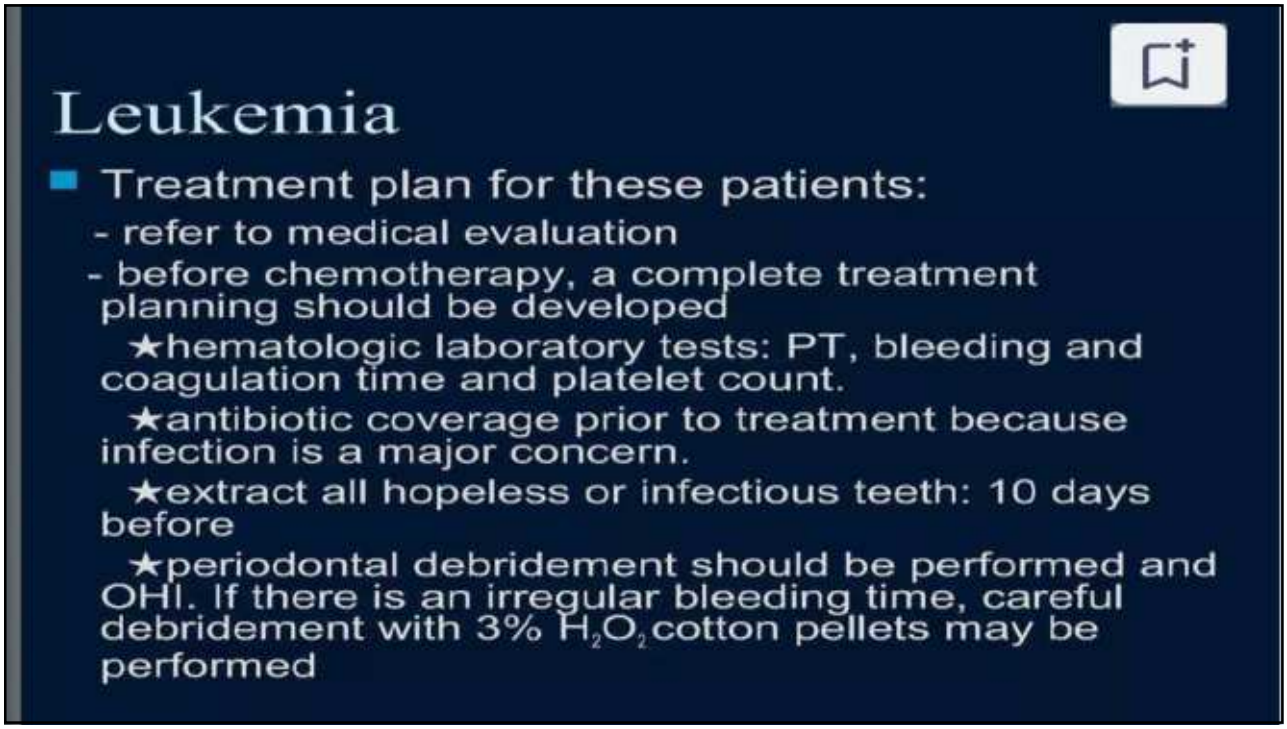
Thrombocytopenic purpuras

- Periodontal surgery treatment: transfusion of platelets prior to surgery
  - atraumatic
  - stents or thrombin-soaked cotton pellets
  - gentle H_2O_2 mouthwashes
  - close post-operation follow up 3~5 days

Blood dyscrasias

- Leukemia
- Agranulocytosis


Leukemia
Precaution:
- enhanced susceptibility to infection
- bleeding tendency
- effects of chemotherapy


Leukemia
Treatment plan for these patients:
- refer to medical evaluation
- before chemotherapy, a complete treatment planning should be developed
★hematologic laboratory tests: PT, bleeding and coagulation time and platelet count.
★antibiotic coverage prior to treatment because infection is a major concern.
★extract all hopeless or infectious teeth: 10 days before
★periodontal debridement should be performed and OHI. If there is an irregular bleeding time, careful debridement with 3% H_2O_2 cotton pellets may be performed

Leukemia

- Chronic leukemia

1. scaling and root planning can be performed without complication but periodontal surgery should be avoided
2. check bleeding time. If it is low, postpone the appointment and refer to a physician
3. plaque control and frequent recall

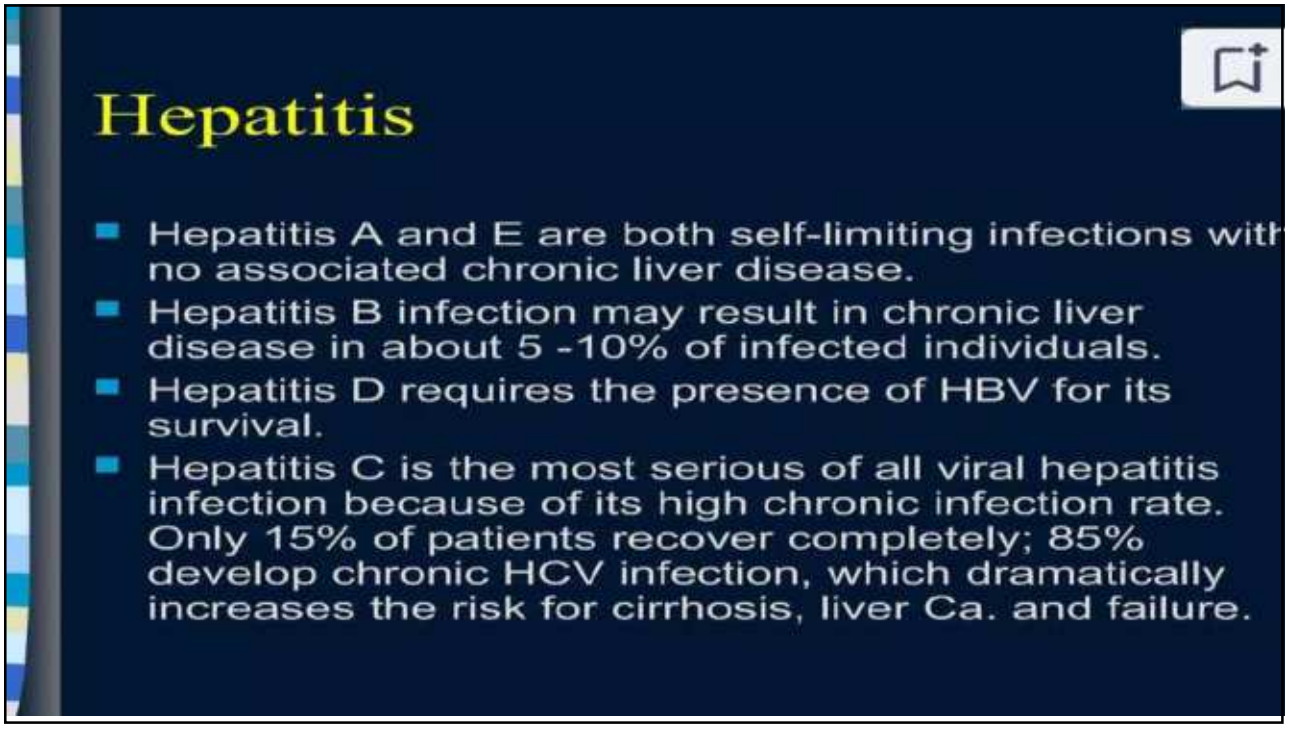
Agranulocytosis

- Precautions:

- reduction in total WBC or granular leukocytes
- more susceptible to infection
- scaling, root planning performed under antibiotic coverage which should be done during periods of disease remission
- severely affected teeth should be extracted aftr consultation
- OHI should include use of chlorhexidine rinses twice daily.

★ avoid aminopyines, barbiturates, chloramphenicol (potential causes of agranulocytosis)


Infectious disease
Hepatitis.
Sexually transmitted diseases.
Tuberculosis.


Hepatitis
Hepatitis A and E are both self-limiting infections with no associated chronic liver disease.
Hepatitis B infection may result in chronic liver disease in about 5 -10% of infected individuals.
Hepatitis D requires the presence of HBV for its survival.
Hepatitis C is the most serious of all viral hepatitis infection because of its high chronic infection rate. Only 15% of patients recover completely; 85% develop chronic HCV infection, which dramatically increases the risk for cirrhosis, liver Ca. and failure.

- If the disease, regardless of type, is active, do not provide periodontal therapy unless the situation is an emergency.
- For recovered HAV or HEV patients, performed routine periodontal care.
- For recovered HBV or HDV patients, consult with the physician and order HBsAg and antiHBs lab test.

If there are negative but HBV is suspected, order another HBs determination

HBsAg + carrier

if antiHBs + may be treated routinely

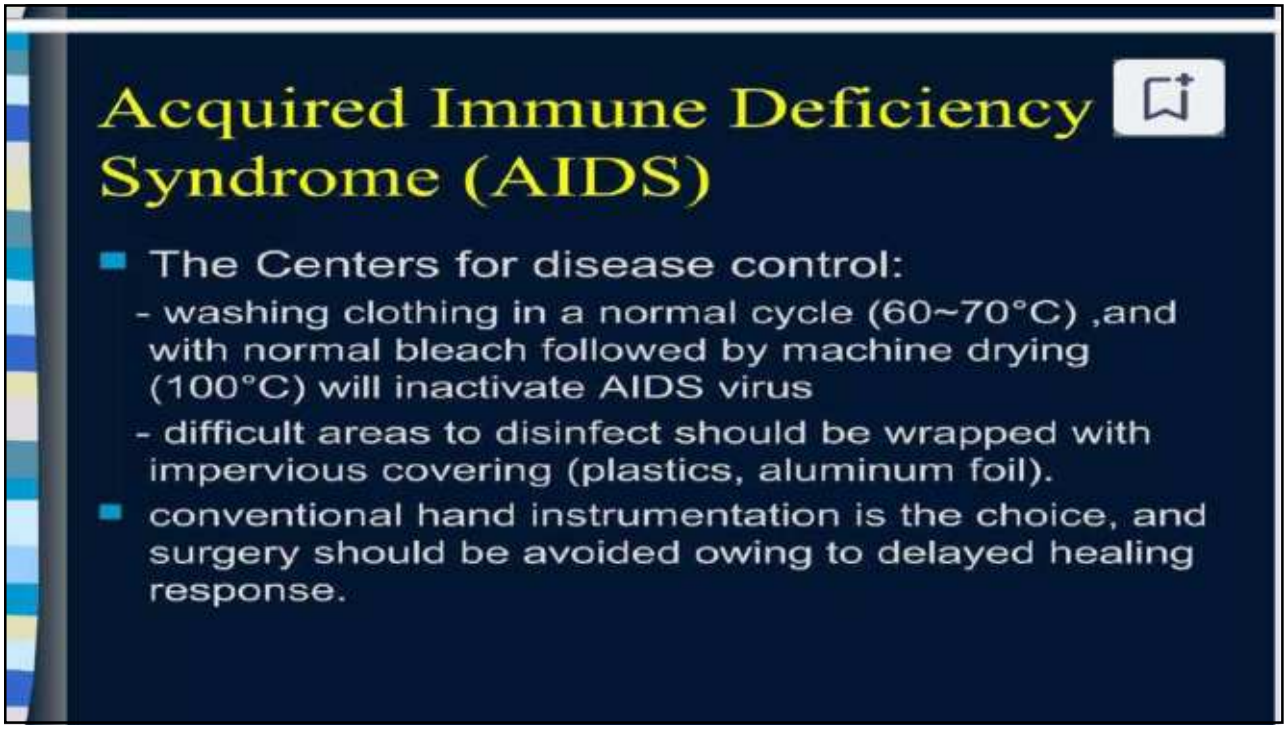
- For HCV consult with the physician to determine the patient's risk for transmissibility and current status of chronic liver

Hepatitis: if patient with HBsAg+ or active requires emergency treatment

- Management

- consultation
- if likely bleeding: checking PT, PTT
- wear mask, gloves, glasses, disposable gowns
- all instruments placed on a sheet of aluminum foil
- all disposable items placed in wastebasket
- minimize aerosol production
- all equipments should be scrubbed and sterilized
  - ★NaOCl (1:3) --- 10 min
  - ★handpieces --- autoclaved
- dental chair
  - ★NaOCl (1:3) --- 10 min
  - ★aseptic technique should be practiced


Acquired Immune Deficiency Syndrome (AIDS)
Precautions:
- barrier technique
- sharp instruments carefully
- proper sterilization technique
- chlorhexidine oral rinsing
- avoid surgery
★delayed healing response


Acquired Immune Deficiency Syndrome (AIDS)
The Centers for disease control:
- washing clothing in a normal cycle (60~70°C) ,and with normal bleach followed by machine drying (100°C) will inactivate AIDS virus
- difficult areas to disinfect should be wrapped with impervious covering (plastics, aluminum foil).
conventional hand instrumentation is the choice, and surgery should be avoided owing to delayed healing response.

Tuberculosis

- Management:
  - emergency care only: the guidelines listed under hepatitis.
  - adequate TB treatment requires a minimum 18 months
  - post-Tx follow up
    - chest films
    - sputum culture
    - patient's syndrome
  - recall at least every 12 months

Dentin Hypersensitivity

TIKRIT UNIVERSITY
College of dentistry
Branch of periodontology

Introduction

Definition:

Dentin hypersensitivity is a common condition of transient tooth pain caused by a variety of exogenous stimuli.

Characteristic

Short , sharp pain.

Most in cervical, then occlusal

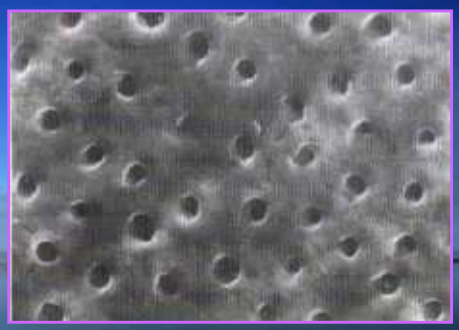
Stimuli

The exogenous stimuli include:

- Thermal (cold)
- Tactile (touch)
- Osmotic changes (sweets, drying the surface)

Etiology

The primary clinical cause is exposed dentinal tubules.



Two phases of development of dentin hypersensitity

- First, dentin has to be exposed.
  —lesion localization
- The dentinal tubules must be opened
  —lesion initiation

The most common clinical cause for exposed dentinal tubules is gingival recession.

Common Reasons for Gingival Recession

1. Inadequate attached gingiva
2. Prominent roots
3. Tooth brush abrasion
4. Pocket reduction periodontal surgery
5. Oral habits resulting in gingival laceration
6. Excessive tooth cleaning
7. Excessive flossing
8. others

Reasons for Continued Dentinal Tubular Exposure

1. Poor plaque control, acidic bacterial byproducts
2. Excess oral acids, sodas, fruit juice
3. Cervical decay
4. Toothbrush abrasion
5. Tartar control toothpaste

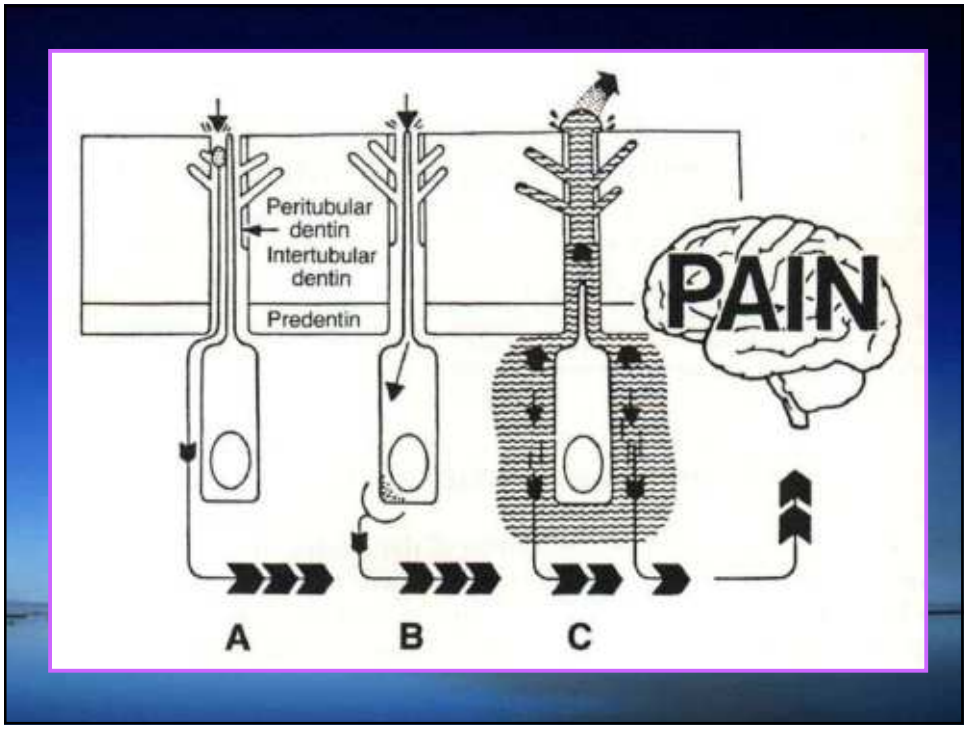
Mechanism

Hydrodynamic theory

—*M.Brännström* in 1967

The fluids within the tubule are disturbed either by temperature changes or physical osmotic changes.
These fluid changes stimulate a baroreceptor which leads to neural discharge (depolarization).

Baroreceptor:
a never receptor sensitive to pressure


Peritubular dentin
Intertubular dentin
Predentin
PAIN
A
B
C


Enamel
Dentin
Normal gum
Exposed nerve
Receded gum

Treatment

A challenge for both the patients and dentists.

- ✘ **It's difficult measuring/comparing different patient's pain.**
- ✘ **It's difficult for patients to change the habits.**

Treatment Strategies

- ✓ **Plug the dentinal tubules preventing fluid flow.**
- ✓ **Desensitize the nerve making it less responsive to stimulation.**

Nerve Desensitization

Potassium Nitrate
the only one approved by FDA and ADA

KNO_3 penetrates through the dentinal tubules to the nerve;
K^+ may depolarize the nerve and prevent it from repolarizing;
Thereby, Preventing it from sending pain signals to the brain.

Covering Dentinal Tubules

- **Composite or GIC restoration**
- **Crown placement**
- **Periodontal surgery**

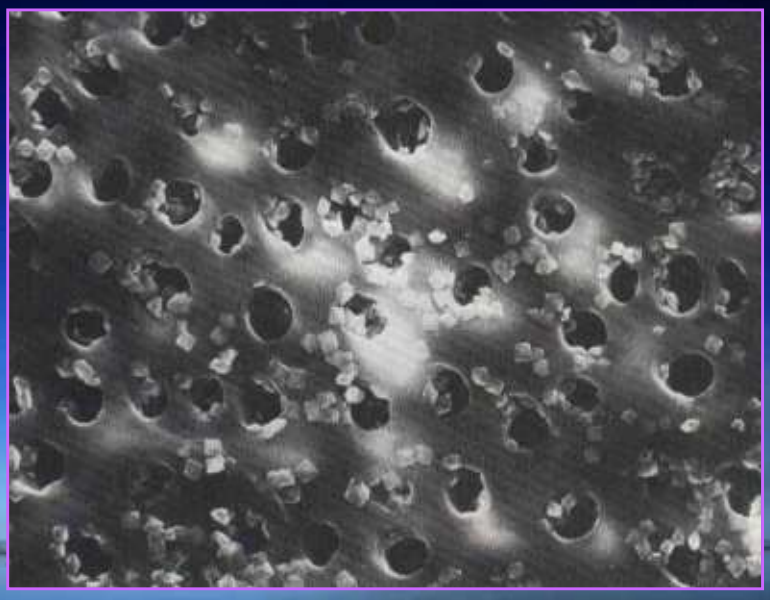
Occluding Dentinal Tubules

To plug the inside of the dentinal tubules

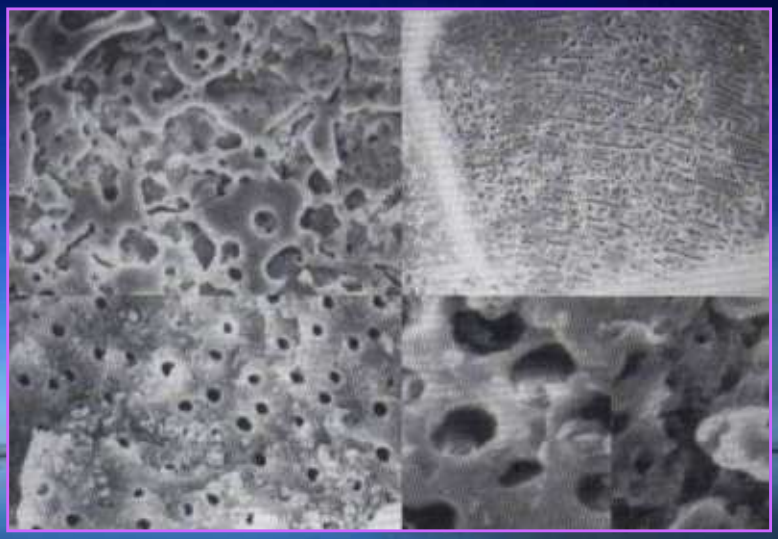
Ions or salts:
- **stannous fluoride, sodium fluoride, potassium oxalate, etc**

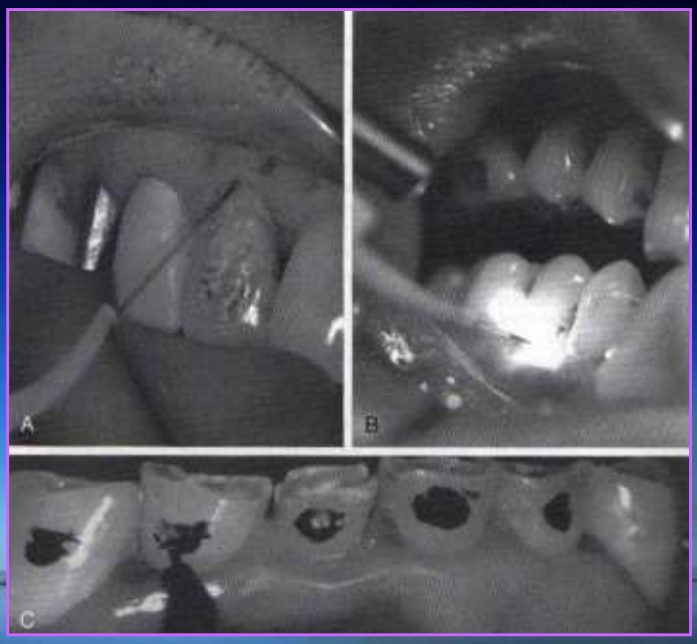
Precipitates:
- **glutaraldehyde**

Resin:
- **dentin sealers**



Laser: another choice





Treatment Steps

1. Thorough exam to identify etiology and eliminate tooth fracture and irreversible pulpitis.
2. Potassium nitrate containing product/toothpaste 2×day for at least 2 weeks.
3. Potassium nitrate containing product in a tight fitting dental tray.

4. In-office tubule occluding product.

5. In-office tubule sealer.

6. Dental restoration, or a periodontal surgery, that covers the exposed dentin.

7. Endodontic procedure to remove the pulp.

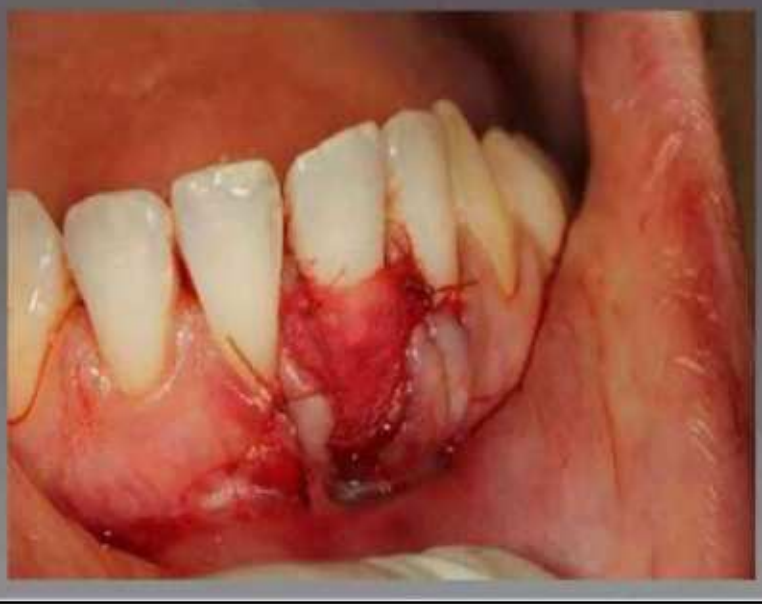
The patient should be informed of the series of steps that may be necessary to eliminate the problem.

Acknowledgements

PERIODONTAL REGENERATION

Dr Mohammed Hazeem

of lost periodontal structures

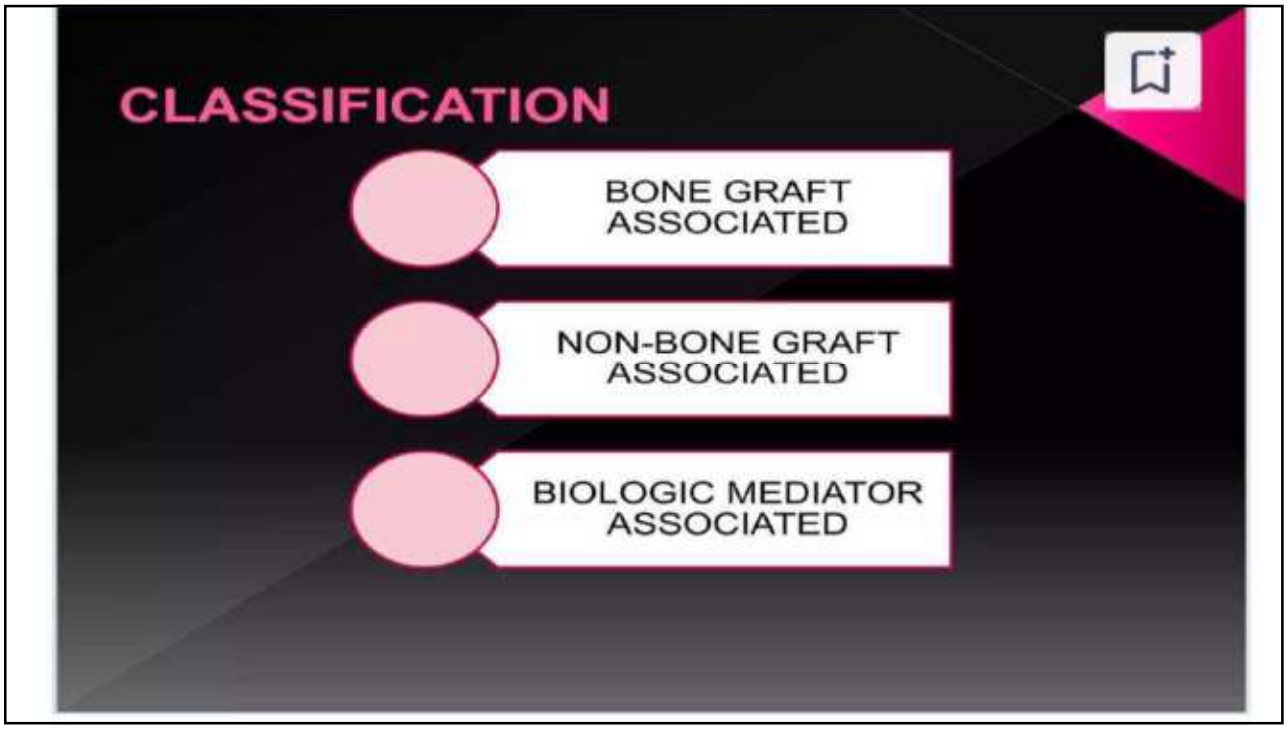


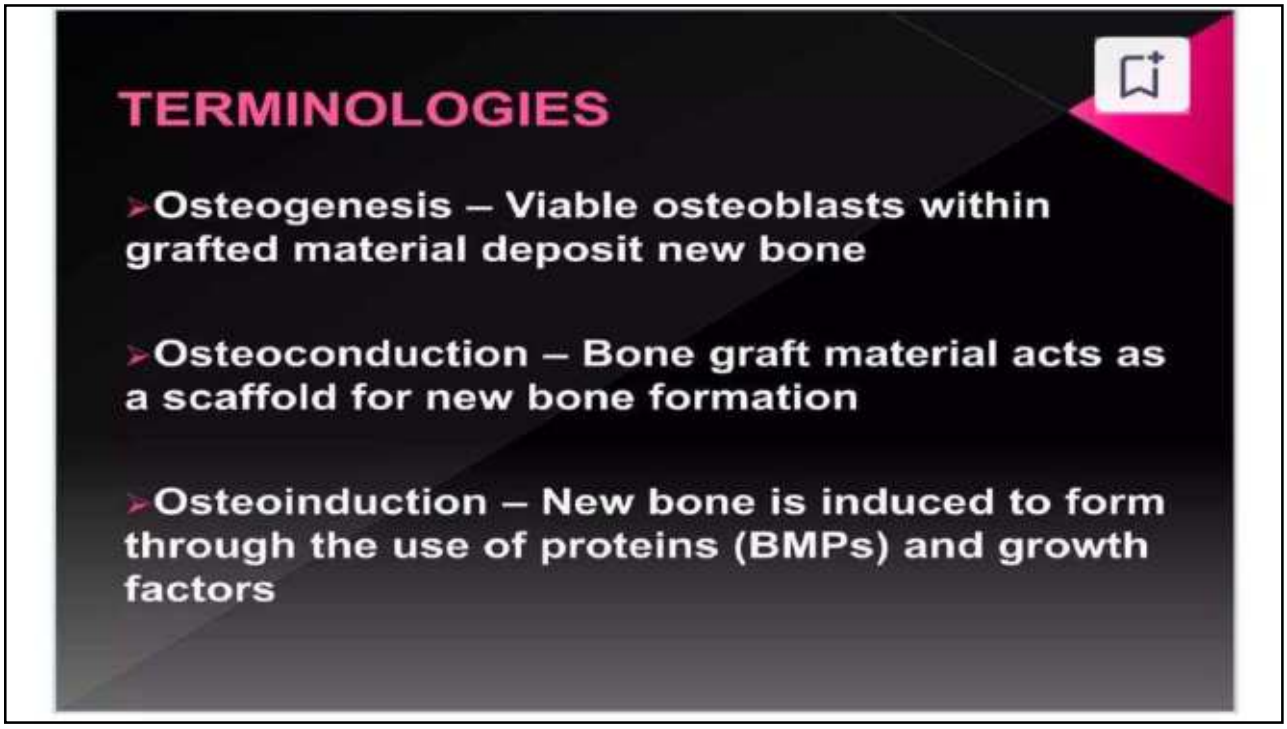
- Reattachment – Repair in areas of root not previously exposed to pocket ex. Surgical detachment of tissues, traumatic tears in cementum, tooth fractures, treatment of periapical lesions

- Repair – Biological process in which continuity of disrupted tissues is restored by new tissues which does not replace the structure and function of lost tissues ex. Healing by long junctional epithelium, scar tissue, fibrous adhesion, ankylosis, bone fill

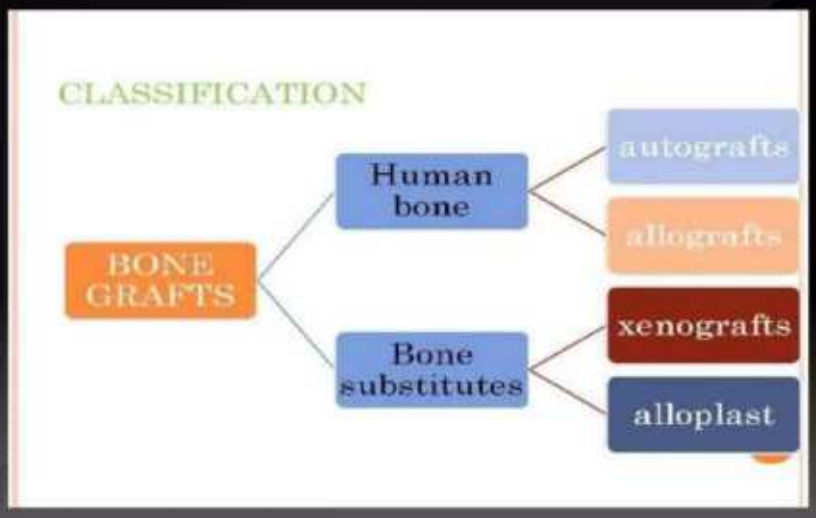
SOURCE OF PROGENITER CELL POPULATION (Melcher 1976)

- Epithelial cells
- Cells derived from connective tissue
- Cell derived from alveolar bone
- Cells derived from periodontal ligament – potential for regeneration (Nymen et al 1982 and Warrier et al 1993)


CLASSIFICATION
BONE GRAFT ASSOCIATED
NON-BONE GRAFT ASSOCIATED
BIOLOGIC MEDIATOR ASSOCIATED


TERMINOLOGIES
Osteogenesis – Viable osteoblasts within grafted material deposit new bone
Osteoconduction – Bone graft material acts as a scaffold for new bone formation
Osteoinduction – New bone is induced to form through the use of proteins (BMPs) and growth factors

BONE GRAFTS

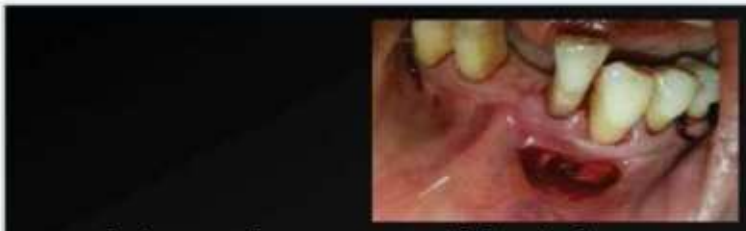


AUTOGRAFTS

- Harvested from patient's own body
- Osteoconductive + Osteoinductive + Osteogenesis (Source of osteoprogenitor cells)
- Advantage – no risk of disease transmission

- Cancellous – Revascularisation and contains viable cells
- Cortical – Scaffold hence provides stability
- Cortico-cancellous – Both

- 2 sources – intraoral and extraoral



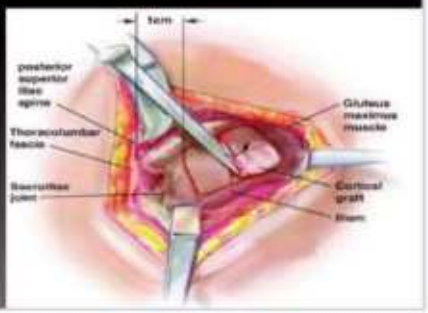
- Intraoral source – Edentulous areas, maxillary tuberosity, extraction sites, healing bone wound, mental and retromolar areas

- CORTICAL BONE CHIPS: Shavings of cortical bone removed by chisel during osteoplasty and ostectomy. Disadvantage – large size hence Increased chances of sequestration

- Extraoral source – Most suitable site is the iliac crest which shows greatest osteogenic capacity

Advantage – large amount of bone graft can be harvested

Disadvantage – Increased post-op complications, expense, time. Extra surgical procedure for procurement



ALLOGRAFTS

- Obtained from genetically dissimilar members of the same species
- 2 forms – FDBA – osteoconductive scaffold
- DFDBA – osteoconductive + osteoinductive
- Why need to decalcify or demineralise?
- Improves osteoinductive potential by exposing Bone Morphogenetic Proteins. Thus, increased bone formation

XENOGRAFTS

- Obtained from genetically dissimilar members of different species
- 3 sources – bovine origin, porcine origin and coralline calcium carbonate (derived from natural coral and resembles cancellous bone)
- Osteoconductive
- Processing : They are anorganic which means all inorganic content is removed to reduce chances of graft rejection

ALLOPLASTS

- Synthetic bone substitutes
- HYDROXYAPATITE
- Stoichiometry similar to bone mineral
- Produced from 3 sources
  - Natural reef building coral skeleton by hydrothermal exchange reacion (trabecular structure remains unchanged and calcium carbonate is converted into calcium phosphate)
  - Homogenisation of calcium phosphate powder with naphthalene particles. Thus macroporous material is left after naphthalene is removed
  - Hydrogen peroxide is used to generate porous structure

- BETA TRICALCIUM PHOSPHATE
- Derived from calcifying marine algae to produce micrperforations which are interconnected
- Good osteoconductive property

CALCIUM PHOSPHATE CEMENT

- Components are available in 2 phases – powder and liquid
- When mixed it produces a material with workable consistency and sets to a solid mass
- Disadvantage – Prolonged setting time and inability to set in the presence of blood

COMPOSITE GRAFTS

- They combine scaffolding properties and biological elements and thus stimulate cell proliferation and diffferentiation and hence improves osteogenesis
- So, they are considered as the close replacement of autogenous bone
- Ex. Beta TCP/ BMP/ Polyglycolic acid

FACTORS AFFECTING SUCCESS OF BONE GRAFTS

- **Defect size and topography** – Most predictable with 3 wall defect and deep narrow defect. Least with 1 wall defect

- **Presence of infection** – Low ph causes bone and graft material to get rapidly absorbed through solution mediated resorption and thus eliminates all infection

- **Graft stability** – Graft should be stable to facilitate biological response

- **Space maintenance** – If graft resorbs quickly then no sufficient time left for new bone formation and defect gets filled with CT

- **Healing period** – In a three wall defect – the healing period is shorter unlike a larger defect

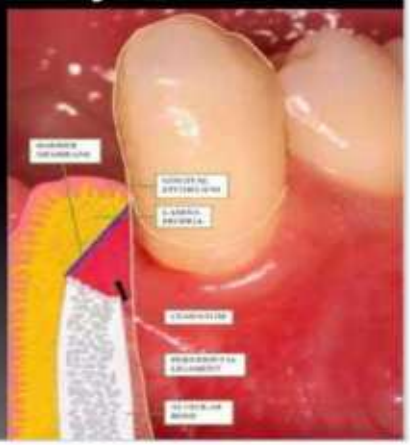
- **Adequate blood supply** – is of paramount importance. 2 sources – cortical/cancellous bone and soft tissue covering the defect

- **Effect of growth factors** – Presence of growth factors required for regeneration
- **Particle size** – 125-1000micromm. Less than 100micromm causes macrophage resorption and thus early loss. Hence must have size of 250-750micromm
- **Systemic factors and habits** – Includes DM, hyperparathyroidism, thyrotoxicosis, osteoporosis, Paget's disease and certain adverse habits which are known to cause adverse effects

GUIDED TISSUE REGENERATION

- **Rationale**
- **Selective growth of cells** derived from periodontal ligament by placing a physical barrier which prevents apical migration of epithelial and gingival connective tissue along root surface
- Provides a **physical barrier** to provide protection to the blood clot during early phases of wound healing and ensures space maintenance for ingrowth of newly formed periodontal apparatus

- Indications
- 2/3 walled vertical, interproximal intrabony defect
- CII and CIII furcation defect
- Treatment of gingival recession
- Ridge augmentation



- NON-RESORBABLE MEMBRANES:
- Milipore membrane – Made up of methylcellulose acetate. Disadv – tears easily during manipulation

- PTFE membrane – Biocompatible synthetic polymer with a long carbon backbone to which fluorine atoms are attached. No enzyme in the body to cleave carbon fluorine bond hence non-resorbable.
- Made up of 2 surfaces – outer surface retards the growth of the epithelium and inner surface provides space for new bone growth and prevents fibrous ingrowth

- ePTFE membrane – expanded PTFE
- For space maintenance material made more rigid by reinforcement with fluorinated ethylene propylene
- TiePTFE – Further enhancement of membrane rigidity by titanium reinforcement . The surface is rough – bacterial adhesion chances. Hence, no surface of the membrane should be exposed to the oral cavity

- Disadvantage – second surgical procedure required to remove the barrier - 4 to 6 weeks after implantation and trauma to the newly formed tissue during second exposure. Flap elevation causes crestal bone

- Titanium mesh – 4 properties
- Rakhmatia et al 2013
- Mechanical property (rigidity) – suitable for space maintenance
- Elasticity prevents mucosal compression
- Stability prevents graft displacement
- Plasticity prevents bending

- Disadvantage – Stiffness and complex surgery required to remove the mesh

- **BIORESORBABLE MEMBRANE**
- Advantage – Avoids the need for surgical removal
- Disadvantage – Unpredictable resorption time and degree of degradation

- **Collagen membrane**
- Made up of bovine and porcine Type 1 collagen
- Resorbed by collagenase in the body
- Weak immunogen – The immunogenicity is due to telopeptide non helical terminals - easily removed by enzyme – pepsin producing atelocollagen

- **Acellular dermal matrix allograft**
- Does **not contain cellular material**
- Hence, major histocompatibility antigen Class I and II are not present in the graft and no chances of graft rejection
- Advantage – Optimum colour matching, acceptable thickness of tissue and formation of additional attached gingiva

- SYNTHETIC
- They are organic aliphatic thermoplastic polymers
- Most commonly used – poly alpha hydroxy acids – either polyglycolic acid and polylactic acid
- Degradation results into end products of breakdown – CO_2 and H_2O
- Degradation may take 20 weeks depending upon polymeric composition

NON-BONE GRAFT ASSOCIATED

- REMOVAL OF JUNCTIONAL AND POCKET EPITHELIUM
- JE and PE interfere with direct apposition of connective tissue and cementum and limits the height to which periodontal fibres insert into the cementum

- Procedures for its removal involve:
- Curettage by ultrasonic, curettes, laser and rotary
- Disadvantage – not controlled due to lack of vision and tactile sense

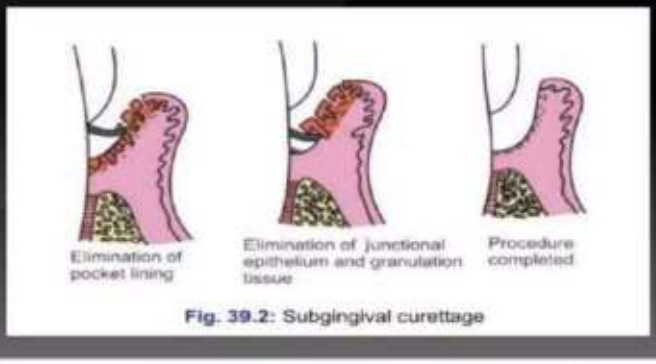
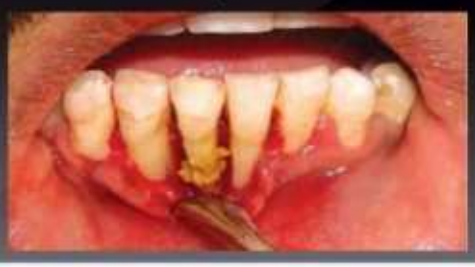


Fig. 39.2: Subgingival curettage

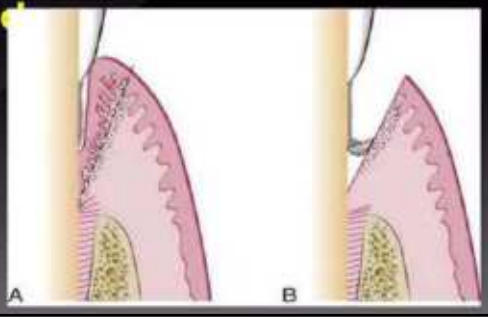
- Use of chemicals – Drugs like sodium sulfide, phenol, camphor, antiformin, sodium hypochlorite
- Disadvantage – depth of penetration not controlled

- **Root surface biomodification**
- Root surface has degenerated fragments of Sharpey's fibres, accumulation of bacteria and by-products, disintegration of cementum and dentin – This interferes with the new attachment
- Therefore, treating the root surface is important

- **Purpose** – Removes smear layer, exposes dentinal tubules, wider dentinal tubules with funnel shaped orifices, eliminates endotoxins and bacteria from diseased tooth surface and results in demineralisation – accelerated healing and new cementum formation
- Citric acid, fibronectin or tetracycline used



- Surgical technique
- ENAP (Excisional New Attachment Procedure)
- Internal bevel incision with removal of excised tissue followed by scaling and curettage
- Recently it is used with Nd:YAG laser – LANAP proce



- CLOT STABILISATION, WOUND PROTECTION AND SPACE CREATION
- Bone graft and barrier membrane with coronally displaced flap protects the wound and creates space for undisturbed and stable maturation of clot
- Prevents apical migration of gingival epithelium and allows connective tissue attachment

ENAMEL MATRIX PROTEINS

- Role in early tooth development and vital role in the formation of cementum, periodontal ligament and alveolar bone
- Commercially available – Emdogain
- Made up of freeze dried enamel proteins (amelogenin fraction) + PGA vehicle to carry biologically active proteins. They are mixed to make a syringeble gel

Obtained and purified from tooth buds of porcine origin

Mechanism of action

- EMD adsorbs to the hydroxyapatite and collagen on denuded root surface
- Insoluble spherical complex forms and remains at the site for 2 weeks
- Sufficient period for proliferation of periodontal ligament cells or undifferentiated cells

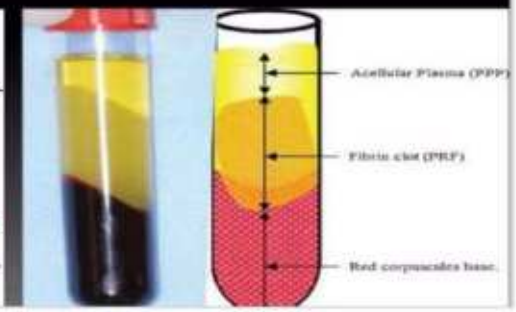
- EMD increases the attachment of periodontal ligament fibroblasts to diseased root surface , increase of growth factors, limits the epithelial downgrowth and increases matrix formation

- **PLATELET CONCENTRATES**
- Rationale – Provide high concentrations of growth factors at the healing area to promote healing and regeneration
- First generation – Platelet rich plasma (PRP)
- It is in the liquid state post centrifugation. For its easy handling platelet activator or agonist is added – topical bovine thrombin + 10% CaCl2 to activate the clotting cascade and produce platelet gel

- Second generation – Platelet rich fibrin. Found by Choukran 2000 and procedure by Dohan et al 2006
- Free of anticoagulant and biochemical modifications
- PRF is a dense fibrin network containing leukocytes, cytokines, structural glycoproteins and growth factors
- When placed over a healing area is a good source of growth factors released upto 7 days

- Procedure – Autologous blood is subjected to gradient density centrifugation. This results in sequestration and concentration of platelets and thus amplifies and accelerates the effect of growth factors
- Centrifugation produces three layers – bottom layer made up of RBC's, middle buffy coat of platelet rich fibrin and supernatent platelet poor plasma (PPP)

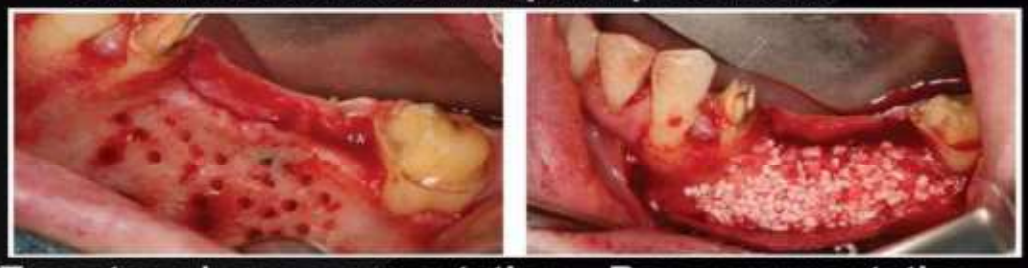
- ORIGINAL CHOUKROUN'S PRF PROTOCOL (STANDARD PROTOCOL): 3000 RPM / 10 MINUTES
- DOHAN EHRENFEST'S GROUP - LEUKOCYTE- AND PLATELET-RICH FIBRIN (L-PRF): 2700 RPM / 12 MINUTES
- CHOUKROUN'S ADVANCED PRF (A-PRF) ENRICHED WITH LEUKOCYTES: 1500 RPM / 14 MINUTES
- CHOUKROUN'S I-PRF (SOLUTION/GEL): 700 RPM/8 MINUTES
- TITANIUM PRF (T-PRF) : 2800 RPM /12 MINUTES (SAME AS L-PRF) ONLY TITANIUM TUBE IS USED



- GUIDED BONE REGENERATION
- Purpose – increase the bone volume in the area of bone resorption due to long standing loss of teeth
- Rationale - Based on the concept of guided tissue regeneration. Surgical placement of a membrane between gingiva and alveolar bone. Thus exclusion of faster healing tissues of the gingiva from entering the defect space. This allows cells of regenerative potential to populate the defect area and regenerate hard and soft tissues.

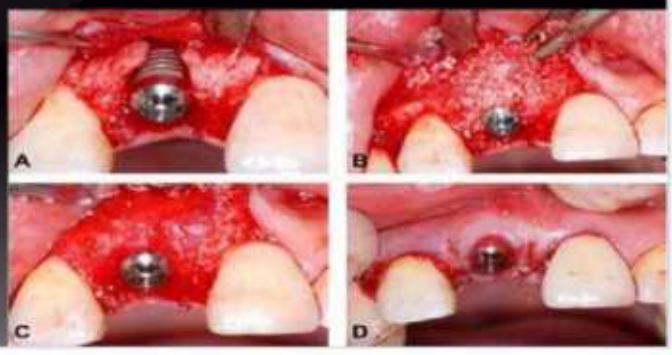
- Two types of GBR

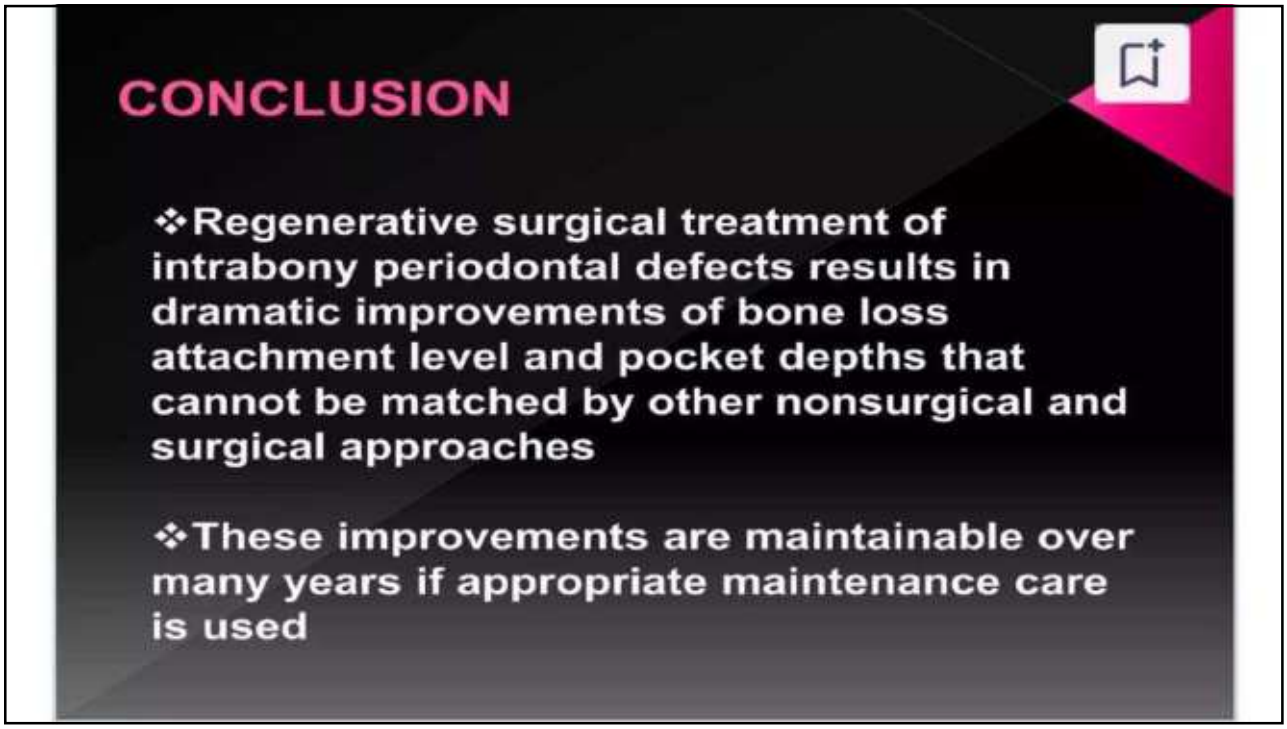
One stage lateral augmentation – dehiscence or fenestration defect in implant placement



Two stage bone augmentation – Bone augmentation followed by implant placement

- GBR with immediate implant placement – Implant if not in direct contact with the socket walls has a distance called as the "jumping distance"
- If this distance is more than or equal to 2mm then GBR is required



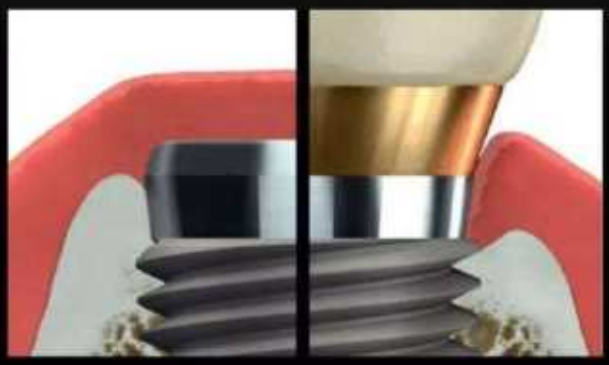

CONCLUSION
❖Regenerative surgical treatment of intrabony periodontal defects results in dramatic improvements of bone loss attachment level and pocket depths that cannot be matched by other nonsurgical and surgical approaches
❖These improvements are maintainable over many years if appropriate maintenance care is used

Definition

Peri-implantitis is a progressive and irreversible disease of implant-surrounding hard and soft tissues and is accompanied with bone resorption, decreased osseointegration , increased pocket formation and purulence.*

*Khammissa RAG, Feller L, Meyerov R, Lemmer J: Peri-implant mucositis and peri-implantitis: clinical and histopathological characteristics and treatment. SADJ 2012, 67(122):124–126

Comparison between physiologic periodontium and periimplant mucosa



Peri-implant Mucosa	Physiological Periodontium
1. Desmosomes and hemidesmosomes of epithelium and junctional epithelium (biological width) are linked with the contact surface	1. Desmosomes and hemidesmosomes epithelium and junctional epithelium (biological width) are linked with the contact surface
2. Direct bone-to-implant contact	2. Anchoring system of root cementum, alveolar bone and desmodontic fibers
3. Subepithelially more collagen fibers and less fibroblasts/vessels	3. Subepithelially more fibroblasts and vessels
4. Parallel collagen fibers in relation to implant surface	4. Dentogingival, dentoperiostal, circular and transseptal fiber orientation
	5. Biologic width 2.04–2.91 mm
5. Biologic width 3.08 mm (includes sulcus)	
6. Probing depth 2.5–5.0 mm (depending on previous soft tissue depth	6. Probing depth 3 mm in health
7. Vascularity is greater	7. Vascularity is greater
8. Connective tissue consist of Only two groups: parallel and circular fibers; no attachment to the implant surface ↑ collagen, ↓fibroblasts	8. Connective tissue consist of 12 groups: six insert perpendicular to tooth surfaces↓collagen, ↑ fibroblasts

*An Implant Is Not a Tooth: A Comparison of Periodontal Indices, Dental Implant Prosthetics, 2nd Edition, Carl.E.Misch

IMPLANT QUALITY SCALE: ICOI CONSENSUS PISA CONFERENCE, ITALY, 2008.

Group	Management	Clinical consideration
I.Success(optimal health)	Normal maintenance	a. No pain or tenderness upon function b. 0 mobility c. <2 mm radiographic bone loss from initial surgery d. Probing depth <5 mm e. No exudate history
II. Survival (satisfactory health)	Reduction of stresses, Shorter intervals between hygiene appointments, Gingivoplasty, Yearly radiographs	No pain 0 mobility 2–4 mm radiographic bone loss Probing depth 5–7 mm No exudate history
III. Survival (compromised health)	Reduction of stresses, Drug therapy (antibiotics, chlorhexidine) Surgical reentry and revision, Change in prosthesis or implants	a. No pain upon function b. 0 mobility c. Radiographic bone loss >4 mm d. Probing depth >7 mm e. May have exudate history
IV. Failure (clinical or absolute failure)	Removal of implant	Pain upon function Mobility Radiographic bone loss > 1 2 length of implant Uncontrolled exudate No longer in mouth

*An Implant Is Not a Tooth: A Comparison of Periodontal Indices, Dental Implant Prosthetics, 2nd Edition, Carl.E.Misch

ETIOPATHOGENESIS

Zitzmann et al. quantified the incidence of the development of peri-implantitis in patients with a history of periodontitis almost six times higher than in patients with no history of periodontal inflammation.

*Zitzmann NU, Walter C, Berglundh T: Ätiologie, Diagnostik und Therapie der Periimplantitis – eine Übersicht. Deutsche Zahnärztliche Zeitschrift 2006, 61:642–649

- Based on the Consensus Report of the Si European Workshop in Periodontology, Lindhe & Meyle reported an incidence of mucositis of up to 80% and of peri-implantitis between 28% and 56%
- The fact that bone remodeling processes often result in marginal bone loss during the first weeks after abutment connection which cannot be regarded as peri-implantitis.
- This led to the recommendation to take a radiograph after insertion of the suprastructure and to consider it as a basis for any future assessment of peri-implant bone loss.

*Lindhe J, Meyle J: Peri-implant diseases: consensus report of the sixth european workshop on periodontology. J Clin Periodontol 2008, 35:282–285.

- In contrast to periodontitis, peri-implantitis lesions harbor **bacteria that are not part of the typical periodontopathic microbiota.** In particular, *Staphylococcus aureus* appears to play a predominant role for the development of a peri-implantitis. This bacterium shows an high affinity to titanium and has according to the results of Salvi et al.

* Salvi GE, Fürst MM, Lang NP, Persson GR: One-year bacterial colonization patterns of Staphylococcus aureus and other bacteria at implants and adjacent teeth. Clin Oral Implants Res 2008, 19:242–248.

RISK FACTORS AND PREVENTION

- Implant loss may occur as "early implant loss" up to one year after implant insertion and "delayed implant loss" with a time period of more than one year after implant insertion -
- Smoking with additional significantly higher risk of complications in the presence of an positive combined IL-1 genotype polymorphism.**
- History of periodontitis.
- Lack of compliance and limited oral hygiene (including missing checkups).

- Charyeva O, Altynbekov K, Zhartybaev R, Sabdanaliev A: Long-term dental implant success and survival–a clinical study after an observation period up to 6 years. Swed Dent J 2012, 36:1–6.

** Gruica B, Wang H-Y, Lang NP, Buser D: Impact of IL-1 genotype and smoking status on the prognosis of osseointegrated implants. Clin Oral Implants Res 2004, 15:393–400.

- Systemic diseases (e.g. mal-adjusted diabetes mellitus, cardiovascular disease, immuno-suppression).
- Soft tissue defects or poor-quality soft tissue at the area of implantation (e.g. lack of keratinized gingiva).**
- History of one or more failures of implants.

- Heitz-Mayfield LJA: Peri-implant diseases: diagnosis and risk indicators. J Clin Periodontol 2008, 35:292–304.

** Lagervall M, Jansson LE: Treatment outcome in patients with peri-implantitis in a periodontal clinic- a retrospective study. J Periodontol 2012, 84:1365–1373.

ETIOLOGY OF RETROGRADE PERI-IMPLANTITIS*

- It is defined as clinically symptomatic peri-apical lesion that develops within the first few months after implant insertion while the coronal portion of the implant sustains a normal bone to implant interface. It can be an active or an inactive lesion.
- Overpreparation or overheating of osteotomy sites
- Presence of a foreign body Iatrogenic causes (e.g. "cementitis").
- Persistent or incompletely debrided periapical lesion.

*Quirynen M, Vogels R, Alsaadi G, Naert I, Jacobs R, van Steenberghe D,*et al.* Predisposing conditions for retrograde peri-implantitis, and treatment suggestions. Clin Oral Implants Res 2005;16:599-608.

SMOKING

- The presence of periodontitis or cigarette smoking increased the risk for peri-implantitis up to 4.7-fold as reported by Wallowy et al.
- Moreover, smoking has been shown to be a predictor for implant failure
- The extent of osseointegration as well as the oral hygiene around dental implants was found to be reduced among smokers

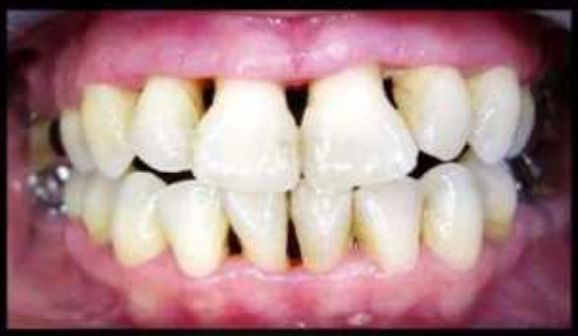
*Clementini M, Rossetti PH, Penarrocha D, Micarelli C, Bonachela WC, Canullo L: Systemic risk factors for peri-implant bone loss: a systematic review and meta-analysis. Int J Oral Maxillofac Surg 2014, 43:323–334.
**Kasat V, Ladda R: Smoking and dental implants. J Int Soc Prev Commun Dent 2012, 2:38–41

GENDER, AGE, MAXILLA vs MANDIBLE

- Evidence of predictors for implant success such as gender or age could not be found but for the jaw of treatment (maxillary versus mandibular implants).
- In a study by Vervaeke et al. maxillary implants were at a significantly higher risk for peri-implant bone loss compared to mandibular implants

HISTORY OF PERIODONTITIS

- The type of dentition (edentulous versus partially edentulous) may influence the colonization of peri-implant tissues with periodontal pathogens.

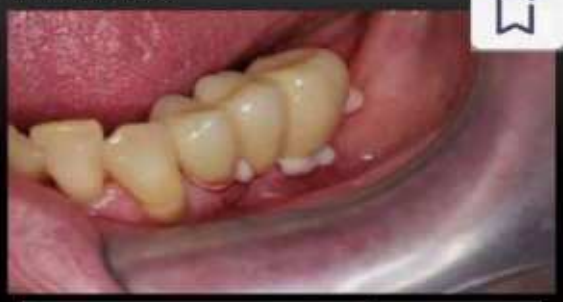


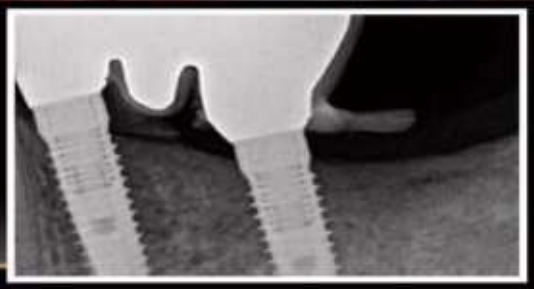
- Across an observation period of 10 years in a group of patients with periodontitis, the previously eliminated bacterial strains of *Aggregatibacter actinomycetemcomitans* and *Porphyromonas gingivalis* could again be detected in the oral mucosa.

* Malo P, Rigolizzo M, Nobre M, Lopes A, Agliardi E: Clinical outcomes in the presence and absence of keratinized mucosa in mandibular guided implant surgeries: a pilot study with a proposal for the modification of the technique. Quintessence Int 2013, 44:149–157.

CEMENTITIS

- "cementitis" may be regarded as the most important identifiable iatrogenic risk factor since its first description by Wilson et al. in 2009 .





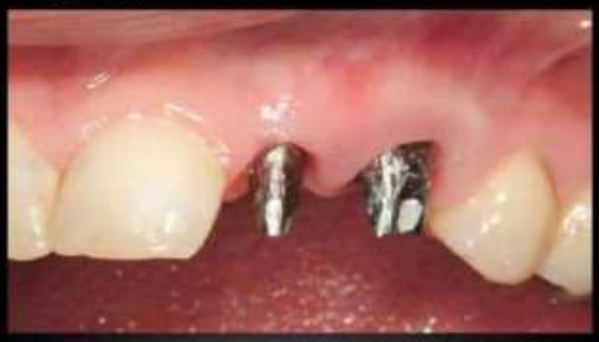
Wilson TG Jr: The positive relationship between excess cement and peri-implant disease: a prospective clinical endoscopic study. J Periodontol 2009, 80:1388–1392

- The latter group revealed that residual dental cement in a group of patients with clinical or radiographic signs of peri-implant disease was present in 81% of the sites. After its removal, clinical signs were absent in 74% of the affected sites.
- Korsch et al. found that the removal of cement remnants led to a decrease of the inflammatory response by almost 60%*

* Korsch M, Obst U, Walther W. Cement-associated peri-implantitis: a retrospective clinical observational study of fixed implant-supported restorations using a methacrylate cement. Clin Oral Implants Res 2014, 25:797–802.

LACK OF KERATINIZED GINGIVA

- Lang and Loe advocate a minimum 2 mm of keratinized gingiva and 1 mm of attached gingiva to maintain gingival health



* Lang NP, Loe H: The relationship between the width of keratinized gingiva and gingival health, *J Periodontol* 43:623–627, 1972.

a number of benefits are present wit keratinized mucosa-

a) The color, contour, and texture of the soft tissue drape should be similar around implants and teeth when in the esthetic zone.

b) The interdental papillae should ideally fill the interproximal spaces.

c) A high smile line often exposes the free gingival margin and interdental papillae zones.

d) The keratinized tissue is more resistant to abrasion. As a result, hygiene aids are more comfortable to use, and mastication is less likely to cause discomfort.

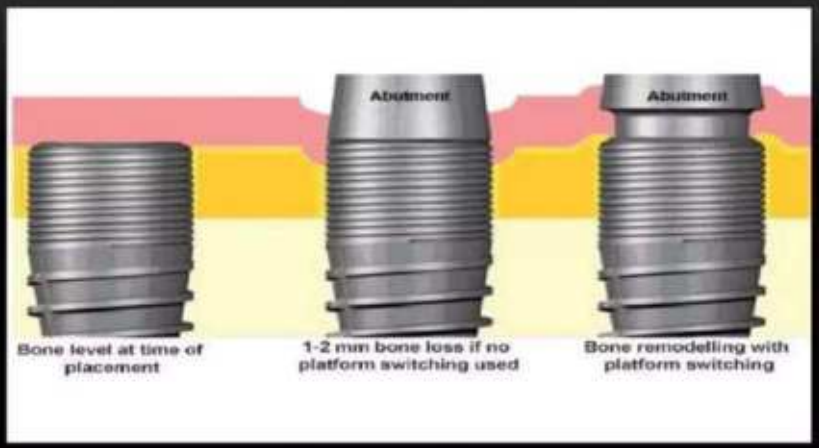
e) In a two-stage protocol, the implant is less likely to become exposed during the healing process. The formation of an interdental/implant papillae is completely unpredictable with mobile un-keratinized tissues.

- The presence of keratinized tissue next to an oral implant presents some unique benefits compared to natural teeth. Keratinized gingiva has more hemidesmosomes; thus, the JEA zone may be of benefit when in keratinized tissue. Whereas the orientation of collagen fibers in the connective tissue zone of an implant may appear perpendicular to the implant surface, these fibers in mobile, nonkeratinized tissue run parallel to the surface of the implant.

PLATFORM SWITCH

- Abutment is located horizontally between implant and crown) can complicate probing and, thus, hide the true extension of peri-implantitis.
- Nevertheless, studies have indicated that platform switch might be an important protective factor against peri-implant disease.*

* Vandeweghe S, De Bruyn H: A within-implant comparison to evaluate the concept of platform switching: a randomised controlled trial. Eur J Oral Implantol 2012, 5:253–262



Internal connections with inward located microgap, should be preferred

IMPLANT LOSS / IMPLANT FAILURE

Implant loss can be differentiated on the basis of the following additional factors-

- Overloading of the implant,
- Faults in material and techniques,
- Poor bone quality at the implant area,
- Systemic diseases and drug therapies, which inhibit bone modulations according to "Wolff 's law" (bone density and strength increase with stress - and vice versa).

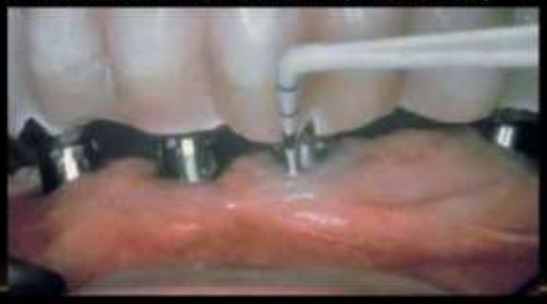
Steigenga JT, al-Shammari KF, Nociti FH, Misch CE, Wang H-L: Dental implant design and its relationship to long-term implant success. Implant Dent 2003, 12:306–317.

DIAGNOSIS OF PERI-IMPLANTITIS

- According to Mombelli and Lang, the parameters to be assessed include:

a) Peri-implant probing:

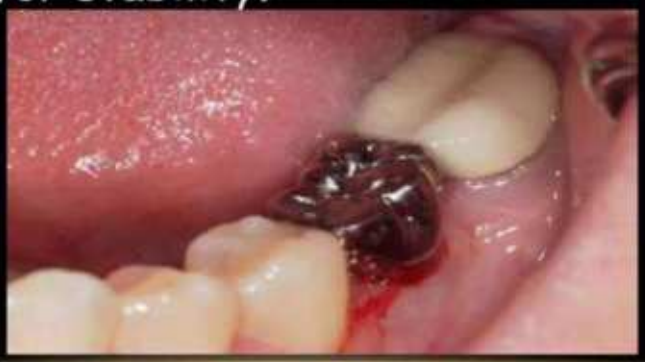
A rigid plastic probe is ideal.probing depth around the connective tissue attachment of 1-1.5 mm.



* Pulluri, *et al.*: Treatment for peri-implantitis, International Journal of Oral Health Sciences | Volume 7 | Issue 2 | July-December 2017

b) BOP:

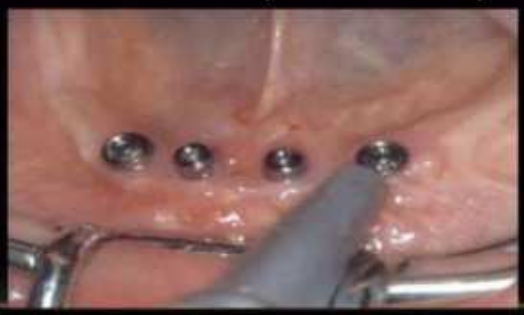
It has been shown that it is not a reliable predictor for progression of periodontal disease, instead its absence is a much better predictor for stability.



* Pulluri, *et al.*: Treatment for peri-implantitis, International Journal of Oral Health Sciences | Volume 7 | Issue 2 | July-December 2017 57

c) Mobility:

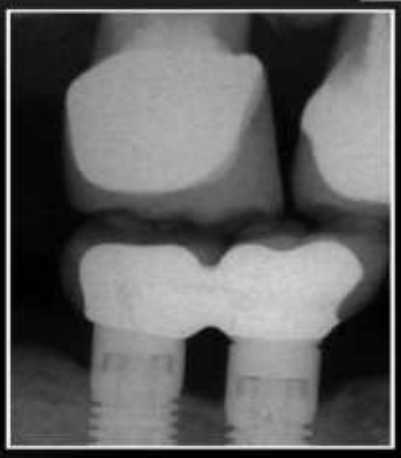
Implant mobility is an indication of lack of osseointegration, but it is of no use in diagnosing early implant disease, rather it shows the final stages of de-integration. Periotest or RFA can be used to assess the stability of an implant.



* Pulluri, *et al.*: Treatment for peri-implantitis, International Journal of Oral Health Sciences | Volume 7 | Issue 2 | July-December 2017

d) Radiography:

Bone loss is a definite indicator for peri-implantitis. The distance from implant shoulder to the alveolar bone crest is a reliable parameter providing the radiographs are properly standardized.

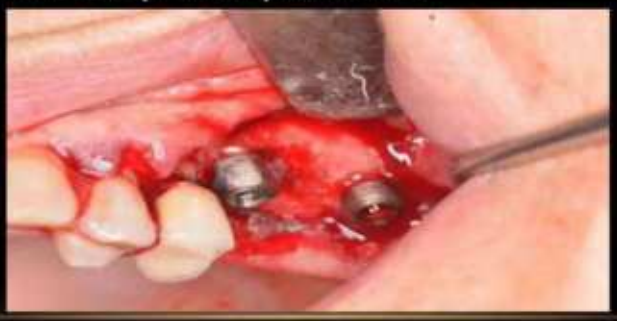


* Pulluri, *et al.*: Treatment for peri-implantitis, International Journal of Oral Health Sciences | Volume 7 | Issue 2 | July-December 2017

SURGICAL TREATMENT OF PERI-IMPLANT

A. Surface polishing/implantoplasty

The main objective of this therapy is to arrest the progression of the disease and to achieve a maintainable site by the patient.



* Pulluri, *et al.*: Treatment for peri-implantitis, International Journal of Oral Health Sciences | Volume 7 | Issue 2 | July-December 2017

- Implant topography should be altered with high-speed diamond burs and polishers to produce smooth continuous surfaces. Performed before any osseous resective therapy is initiated and with profuse irrigation

Pulluri, *et al.*: Treatment for peri-implantitis, International Journal of Oral Health Sciences | Volume 7 | Issue 2 | July-December 2017 57

GUIDED BONE REGENERATION

Pros

- In 2007, Roos-Jansaker *et al.* were able to show a good response to therapy (implants pocket reduction of 2.9-3.4 mm and new bone fill of 1.4-1.5 mm) for peri-implantitis sites treated with either bone grafts alone or bone grafts in conjunction with a resorbable collagen membrane.

Cons

- However, in a recent systematic review by Sahrmann *et al.* in 2011 have concluded that complete fill of the bony defect using guided bone regeneration (GBR) seems not to be a predictable outcome.

* Smeets et al. , Definition, etiology, prevention and treatment of peri-implantitis – a review Head & Face Medicine 2014, 10:34

- The mucosal health status is not considered in most studies. Well-controlled trials are further advocated to determine predictable treatment protocols for the successful regenerative treatment of peri-implantitis using GBR technique.

* Smeets et al. , Definition, etiology, prevention and treatment of peri-implantitis – a review Head & Face Medicine 2014, 10:34

Peri-implant Resective Therapy

- Resective surgery can be carried out for the **elimination of peri-implant lesions**, whereas regenerative therapies may be applicable for **defect filling**.
- Ostectomy and Osteoplasty: It is indicated in moderate to severe horizontal bone loss, moderate (<3 mm) vertical bone defects (1 and 2 wall bone defects), reduce the overall pocket depth and implant position in the un-esthetic area.

- Resective surgery has been shown to be effective in reduction of BOP, PDs and clinical signs of inflammation.
- Implantoplasty can be considered a useful adjunct along with resective surgery to smooth and decontaminate portions of an implant left exposed by peri-implantitis lesions.

TREATMENT OF RETROGRADE PERI-IMPLANTITIS

- Implant extraction,
- peri-apical surgery,
- debridement,
- regenerative procedure,
- local decontamination (antimicrobials/lasers), and
- use of antibiotics.

Pulluri, *et al.*: Treatment for peri-implantitis, International Journal of Oral Health Sciences | Volume 7 | Issue 2 | July-December 2017 57

CONSENSUS STATEMENT: NONSURGICAL INTERVENTIO THIRD EAO CONSENSUS CONFERENCE 2012:

- Peri-implant mucositis can be treated successfully.
- Peri-implantitis has limited success with mechanical debridement
- Clinical recommendations
- Patients should be monitored regularly for:
- Plaque control
- Signs of peri-implant inflammation
- BOP
- A regular maintenance program for the long-term management of peri-implantitis lesions

Pulluri, *et al.*: Treatment for peri-implantitis, International Journal of Oral Health Sciences | Volume 7 | Issue 2 | July-December 2017 57

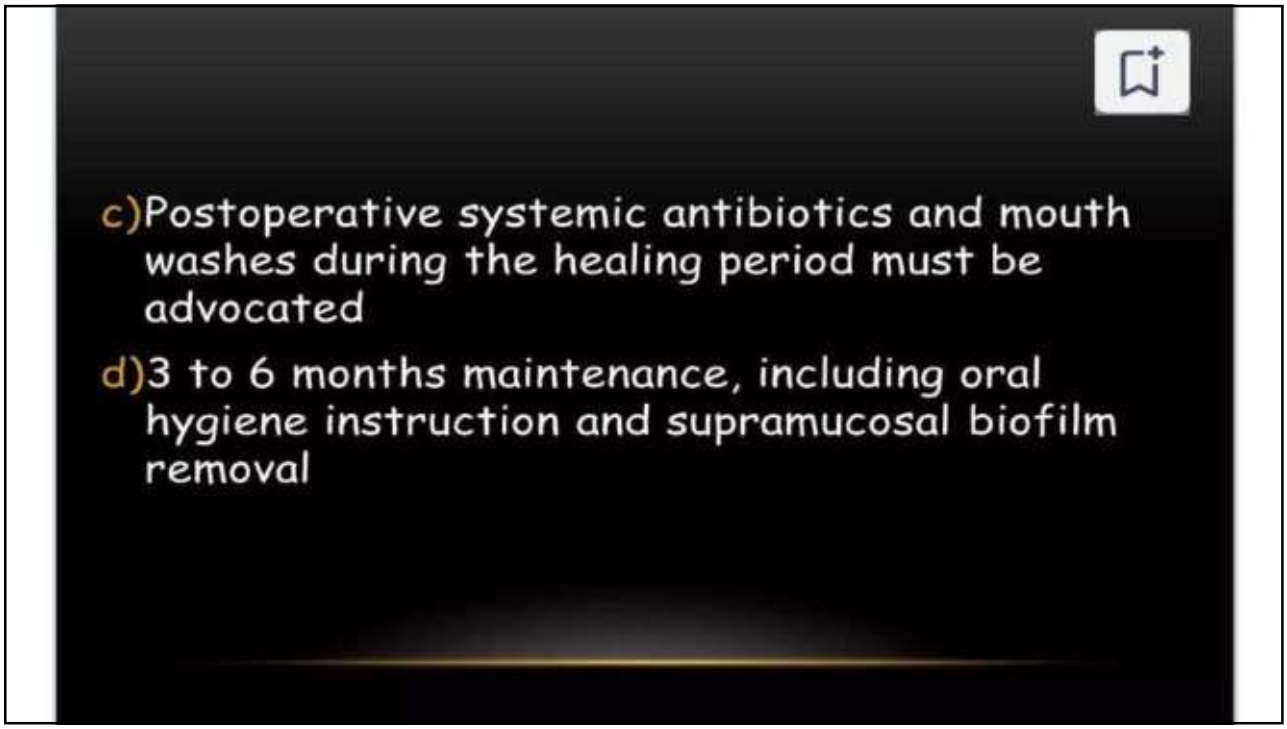
CONCLUSION

- A review by Heitz-Mayfield and Andrea Mombelli in 2014 showed that successful treatment outcomes 12 months following therapy of peri-implantitis could be achieved in a majority of patients.

Their recommendations include:

a) Pretreatment phase should include oral hygiene assessment, smoking cessation, and prosthetic evaluation

b) Surgical access when the resolution of peri-implantitis is not achieved with nonsurgical treatment


c)Postoperative systemic antibiotics and mouth washes during the healing period must be advocated
d)3 to 6 months maintenance, including oral hygiene instruction and supramucosal biofilm removal

Tooth mobility

Tooth mobility is a characteristic of loss of attachment. In case of periodontitis, tooth mobility is a sign of disease progression, and evaluation for treatment outcome.It might be the first symptom that the patient can feel. So, it is critical and it is important to plan whether to save the tooth or extract it.

Tooth mobility is described as the movement of the tooth within the socket. It is of two types: physiologic and pathologic mobility.

physiological mobility:

It is the tipping movement of the tooth in its socket exerted due to a force, when the surrounding periodontium is healthy and intact. This movement is within the periodontal space that is separating between the root and the alveolar bone. All teeth have a slight degree of physiologic mobility, as long as the tooth is not ankylosed. The degree of physiologic mobility is different based on the time of the day, early in the morning the teeth show high degree of physiologic movement due to the limited occlusion during sleeping that makes teeth extruded a little bit and within normal limits. During the day, mobility is reduced by chewing & swallowing forces which intrude the teeth in the sockets.

Increasing physiological tooth mobility can be seen in unilateral functioning of the dentition for longer period of time. A higher mobility demonstrates on the hypofunctional side than the other side. Increasing physiologic mobility can be also seen in pregnancy and orthodontic treatment.

Decrease in the height of the alveolar bone with intact periodontium: it can be seen in treated periodontitis. A horizontally directed force applied to the crown of the tooth in this case will result in a larger excursion of the crown than if a similar force is applied to a tooth with normal height of the alveolar bone and normal width of the periodontal ligament. Physically, this is due to low fulcrum line

Some still consider the above as physiologic mobility even with the clear mobility of the teeth. They consider this mobility as normal as long as there is no inflammation or progression of bone loss.

Pathologic mobility:

It is the progressive increasing tooth mobility, which may occur in conjunction with trauma from occlusion. It is characterized by active bone resorption which indicates the presence of inflammatory alterations within the periodontal ligament.

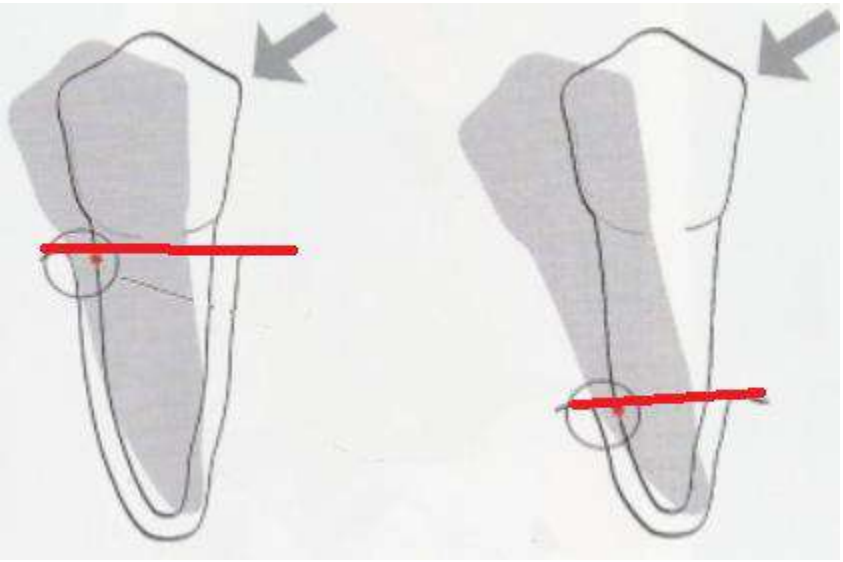
Tooth mobility could be in horizontal when the tooth moves in a faciolingual direction. It is assessed by putting the handles of two dental instruments on either side of the tooth and applying alternating moderate pressure in the faciolingual direction against the tooth first with one, then with the other instrument handle.

Vertical tooth mobility is the ability to depress the tooth in its socket. It is assessed using the end of an instrument handle to exert pressure against the occlusal or incisal surface of the tooth.

Causes of tooth mobility

Tooth mobility has several principal causes:

1. Advanced periodontal diseases & loss of supporting bone: Normally the crest of alveolar bone located about 1-2 mm shorter than CEJ. The amount of mobility depends on the severity of bone destruction & there will be reduction in the height of the bone. This reduction in bone height leads to lowering in the fulcrum line and reduce support around the teeth. This leads to increase tooth mobility.



2. Gingival & periodontal inflammation: Mobility will occur when the connective tissue infiltrated by inflammatory cells and the matrix begin to breakdown and fluids retains inside the tissue. This result in easy compressible tissue which allow tooth movement beyond normal. Healthy periodontium acts as a firm cushion surrounding the tooth. If this tissue become inflamed, it will lose its firm and fibrotic characteristics and become loose and fragile tissue.

3. Trauma from occlusion: It is injury to the tissue produced by excessive occlusal forces or occlusal habits as bruxism or clenching. It is a common cause for increased tooth mobility. It might be occurred due to imbalance occlusion for example a high filling. The pathologic changes that occur because of trauma from occlusion are widening of periodontal ligament space &/or destruction of the bone surrounding the root.

4. Immediately following periodontal therapy: May cause transient mobility for a short period of time because of the surgical trauma (physical trauma rather than bacterial) due to the use of surgical instruments. After 2-3 weeks, the mobility will disappear if the diagnosis, treatment plan & maintenance phase of therapy were done correctly.

5. Pulpal inflammation: Spread of inflammation from periapical area to the periodontal ligament may results in changes that increase tooth mobility.

Factors affecting tooth mobility:

1- the shape of the roots, long or short roots...etc.

2- Number of the roots, multirooted teeth have less mobility than single rooted.

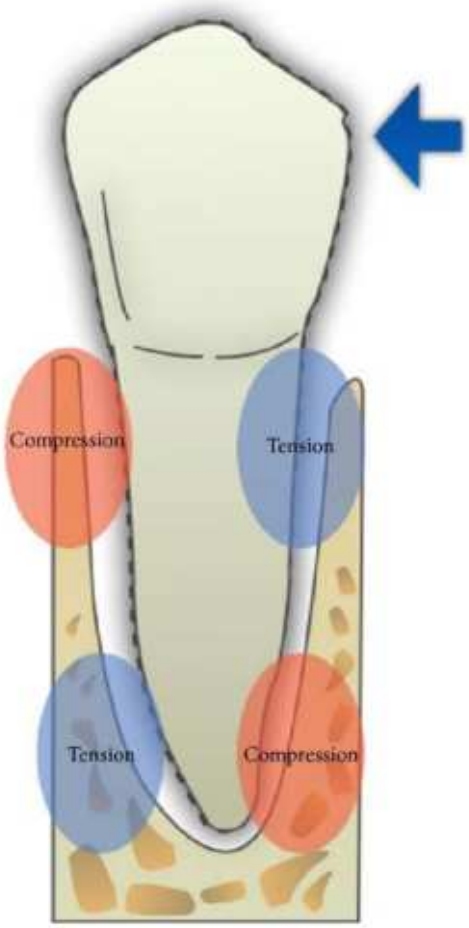
3- The periodontal space width. The normal width range between 0.15 to 0.38 mm.

4- The amount of alveolar bone support which decide the level of the fulcrum line (FL). FL is an imaginary line along which the tooth tends to rotate till it stopped by being in contact with the alveolar bone surrounding it. Normally the height of the alveolar crest is 1 to 2 mm below the CEJ.

5- The degree & duration of the applied force whether in normal or abnormal function.

Stages of tooth mobility:

Mobility of the tooth appears in two stages based on the force applied to the crown of the tooth. These stages are:

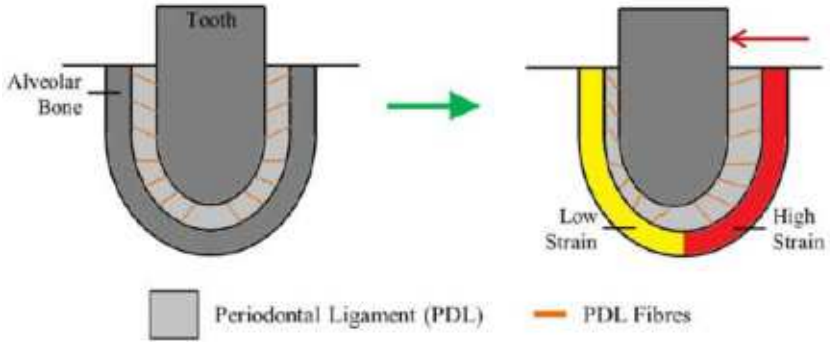


Initial tooth mobility (ITM):

It is the movement of the tooth within the periodontal space. It is also called intra socket stage. It occurs due to viscoelastic distortion of periodontal fluid, periodontal fibers and inter bundle content. The movement ranges from 50-100 µm, under a load of 100 lb. In this movement there is pressure & tension zone. In the pressure zone there is 10% reduction in the width of periodontal ligament & in the tension zone there is a corresponding increase. In the ITM, there is reorientation of the PD ligament fibers into a position towards tensile strength & it is different from individual to another & from tooth to tooth.

secondary tooth mobility

When a large force (500 Ib) is applied to the crown, the fiber bundles on the tension side cannot offer sufficient resistance to further root displacement. The additional displacement of the crown is called secondary tooth mobility (STM). In this stage there will be distortion & compression of the periodontium in the pressure side. Gradually, this distortion will affect the alveolar bone causing bone resorption. The displacement of the crown when a force of 500 pounds is applied varies between teeth: Incisors 0.1-0.12 mm Canines 0.05-0.09 mm Premolars 0.08-0.1 mm Molars 0.04-0.08 mm & it is larger in children than adults & is larger in females than males & increase during pregnancy.



Sign & symptoms of T.M

1) Patient awareness of mobility: Mobility is detected quite incidentally when patient's attention is brought to tooth by tenderness experienced on chewing.

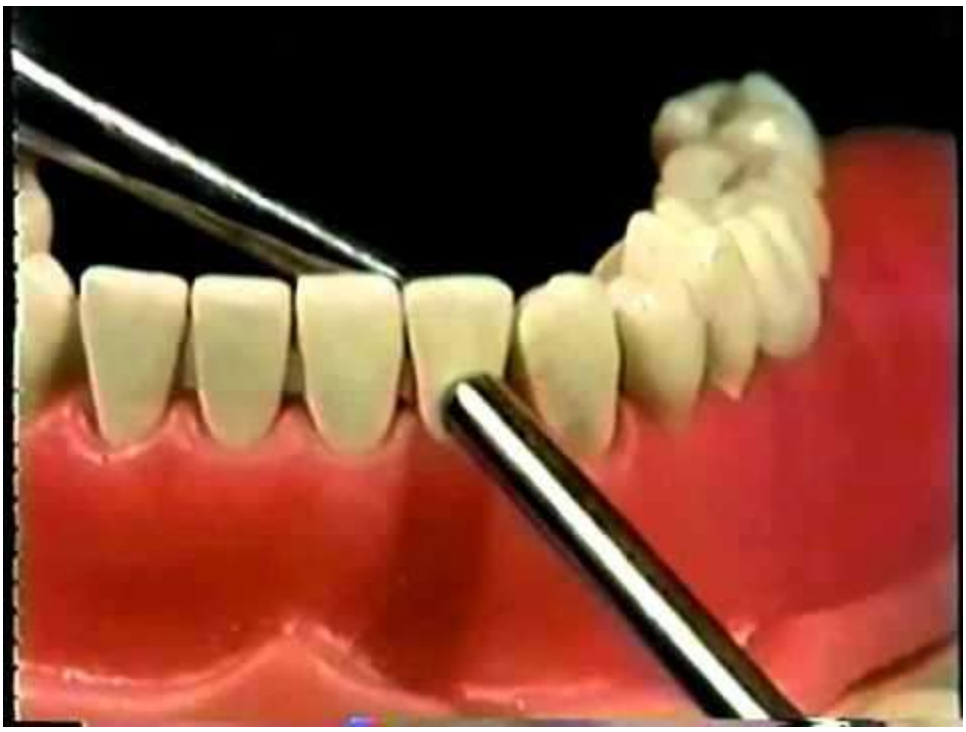
2) Functional discomfort: Pain may be expected following sudden tooth displacement when biting on hard foods or with inadvertent trauma.

3) Aesthetic: Anterior labial or lateral tooth displacement results in fanning & elongation of clinical crown with poor appearance.

Tooth Mobility

Measurement of tooth mobility

Basically, tooth mobility is assessed in clinics by observation. This procedure including putting the tooth between two metal instruments and exert an alternative light force from the instruments and observe and assess visually the degree of mobility. However, this technique is non-scientific, insufficient and do not assess mobility objectively as it is influenced by the clinician's tactile perception and individual interpretation.

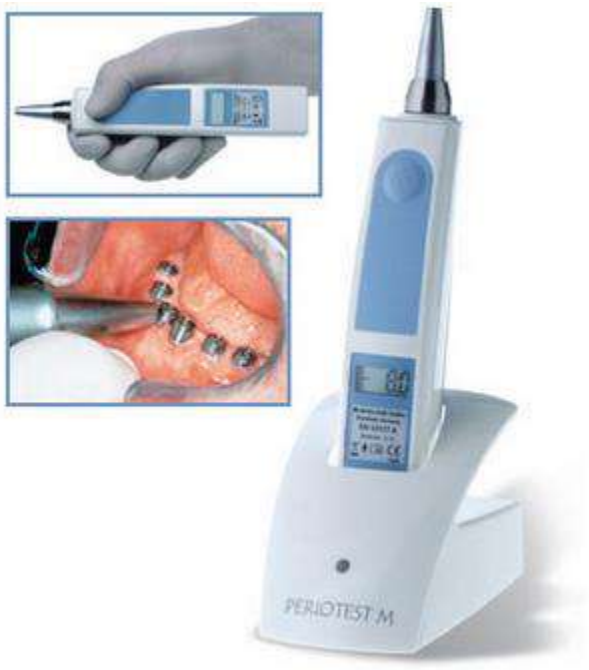


Several devices were invented to assess tooth mobility more scientifically. However, they were not sensitive enough to assess slight mobility less than 1mm. Such these devices as laser diodes, magnetic sensors, Doppler vibrometer, Manly's mobilometer, periodontometer...etc

Periotest (1992):

A popular method for determining tooth mobility was presented by Schulte 1987 and Schulte et al 1992. The value of 'periotest' depends mainly on the damping characteristics of the

periodontium. It has a pressure sensitive tip. When it is placed on the tooth surface it gives around 16 pulses in 4 seconds and measures the response of the tooth. The amount of tooth mobility is displayed by a value called periotest value (PTV) ranging from –8 to +50, which can be correlated to the 4 stages of tooth mobility reported by Miller. Now, it is widely used to assess the osteointegration of the implants.



NEVD- Non-Contact Electromagnetic Vibration Device (2016):

This device was evolved to objectively and accurately assess the overall periodontal tissue condition. This device analyses both tooth mobility as well as the periodontal tissue condition by using mechanical parameters (i.e., resonant frequency, elastic modulus and viscosity coefficient) and measuring the vibration of the tooth. Recently a Laser Displacement senser was added to NEVD to increase its accuracy.

Classification of tooth mobility:

TM classified into different classification and grading scores based on different researchers and views. The most widely used classification is Miller's classification which graded TM according to the following criteria:

Grade I: is the mobility of the crown 0.2-1 mm in horizontal direction.

Grade II: mobility of the crown of the tooth exceeding 1mm in horizontal direction.

Grade III: mobility of the crown of the tooth in vertical direction as well & the tooth becomes even depressed in its socket.

Others classified TM more precisely such as Nyman 1975:

Degree 1 – Horizontal or meso-distal mobility of < 0.2 mm

Degree 2 - Horizontal or meso-distal mobility of 0.2 – 1 mm

Degree 3 - Horizontal or meso-distal mobility exceeding 2 mm and/ or vertical mobility

While Glickman attempted to arrive to a diagnosis rather than a parameter of the status of PD:

Normal

Grade 1 – Slightly more than normal

Grade 2 – Moderately more than normal

Grade 3 – Severe mobility facio-lingually and/ mesio-distally combined with vertical displacement

Treatment of increased tooth mobility:

A number of situations will be described for the aim of reducing increased tooth mobility.

Situation I:

(increased mobility of a tooth with increased width of the periodontal ligament but normal height of the alveolar bone). This case is seen in teeth with improper filling or crown restoration (high spot). Occlusal interferences develop & the surrounding periodontal tissues become inflamed (trauma from occlusion). If a tooth is subjected to forces directed in buccal direction,

bone resorption will develop in the buccal-marginal & lingual-apical pressure zones with a resulting increase in the width of periodontal ligament in these zones. Such traumatizing forces in teeth with normal periodontium cannot result in pocket formation or loss of connective tissue attachment. The resulting mobility of the tooth is due to the imbalance distribution of the force on the surrounding alveolar bone. An adaptation of the periodontal tissue to the altered functional demands can occur. In such a case bone resorption is a reversible process and can be treated by elimination of occlusal interferences or occlusal adjustment.

Situation II:

(increased mobility of a tooth with increased width of PDL & reduced height of alveolar bone). This situation is developed due to periodontitis and reduced alveolar bone height. The tooth loss its support. This reduction in periodontal tissue support exposed the tooth to excessive horizontal forces (trauma from occlusion), inflammatory reaction develops in the pressure zone of the periodontal ligament with bone resorption& result in increased width of PDL & the tooth become hypermobile. In this case, periodontitis should be treated first and inflammation should be eliminated. A second step is to determine the mobility of the tooth whether it is disturbing the patient, ex. Interfering with mastication. Then, occlusal adjustment is performed to reduce excessive forces. So, bone apposition will occur & the PDL will regain its normal width & the tooth stabilized.

Situation III:

(increased mobility of a tooth with reduced height of alveolar bone & normal width of PDL)

In case of teeth with normal width of PDL, no further bone apposition on the walls of the alveoli can occur. So, occlusal adjustment will not cause any differences. However, it might lead to further problems.

To treat cases in situation III, we have to determine the patients main complain. If the patient is complaining of tooth mobility ex. discomfort, it can be treated by splinting. If the patient is not complaining and he/she is satisfied with this mobility, then no treatment is required.

Situation IV:

(progressive increasing mobility of a tooth (teeth) as a result of gradually increasing width of PDL in teeth with reduced height of alveolar bone).

This case seen in advanced periodontal disease, the tissue destruction may have reached a level where extraction of one or several teeth cannot be avoided. The remaining teeth that are not extracted may after therapy exhibit a progressively increasing mobility when force applied during function, which may mechanically disrupt the remaining PDL components& cause extraction of the teeth. This case is treated by fixed splint to stabilize the hyper mobile teeth & to replace missing teeth.

Situation V:

(increased bridge mobility despite splinting) In patient with advanced periodontal disease, following proper treatment for periodontitis that may include multiple extraction and splinting of the remaining teeth. In spite of the splinting, these teeth might show increase mobility, due to generalized bone resorption and loss of support. This cross-arch mobility or bridge mobility might be considered as acceptable if the patient is not complaining of any discomfort or interfering with mastication.

Occlusal adjustment

Also known as selective grinding is the modification of the occluding surfaces of teeth through grinding to create harmonious contact relationships between the maxillary and mandibular teeth. The aim of occlusal adjustment is to establish and maintain stable occlusal relationships and to restore an optimal occlusal function. One condition is applied for occlusal adjustment, is the elimination of inflammation first before planning for grinding. It is indicated in case of trauma from occlusion.

However, occlusal adjustment is contraindicated in these cases:

• Occlusal adjustment without careful pretreatment study, documentation, and patient education

• Prophylactic adjustment without evidence of the signs and symptoms of occlusal trauma

• As a primary treatment of plaque-induced inflammatory periodontal disease

• Treatment of bruxism based on a patient history without evidence of damage, pathosis, or pain

• When the emotional state of the patient precludes a satisfactory result

• Instances of severe extrusion, mobility, or malpositioning of teeth that would not respond to occlusal adjustment alone.

Splinting

It is the procedure joining the mobile tooth or teeth together with adjacent teeth in the jaw to increase resistance to an applied force. The purposes of splinting are mainly for protection of tissue, restoration of physiologic occlusion, distribution of force, and ensuring functional comfort during mastication. Splints could be Composit filling attaching the adjacent teeth, wired teeth, removable or fixed bridges, orthodontic appliances...etc. It could be temporary, provisional or permanent too.

Contraindications for splinting:

- In case of active, not treated periodontal disease
- In case of trauma from occlusion has not been treated by occlusal adjustment
- Insufficient number of firm non-mobile teeth
- Patient is not maintaining good oral hygiene

Locally delivered antibiotics in periodontal therapy

1 Hour lecture

Dr Hadeel Mohammed Abbood

Terms related to antibiotic (AB) therapy

- Antibiotic: Are medications that are used to kill or stop the multiplication of microorganisms at a concentrations that is relatively harmless to the host tissue. Therefore, it can used to treated infectious diseases.
- Can be classified into different classes according to different characteristics

Classification of Antibiotics

Antibiotics

- **According to the mechanism of action**
  - Bacteriostatic: stop the multiplication of MO
  - Bactericidal : kills MO
- **According the range of susceptible MO**
  - Narrow spectrum
  - Wide spectrum
- **According to the pathogen affected**
  - Antiviral
  - Antibacterial
  - Antifungal
- **According to the origin**
  - Extracted from living organisms
  - Synthetic

Rationale for Local Delivery and Controlled Release

- Chronic adult periodontitis is a multifactorial disease.
- The first requirement is a susceptible host, largely due to non-modifiable RF such as age and genetics.
- Disease risks that may be modifiable include pathogenic oral plaque microorganisms and behavioral or environmental factors such as tobacco use.
- Mediating the pathogenesis are host-derived elements, including immune and inflammatory responses that can affect soft and hard tissue metabolism and feed back on the pathogenic flora.

Periodontal diseases are bacterial infections; the requirement for bacteria to initiate the periodontal lesion is well recognized. The antibacterial effect of SRP or other mechanical therapy generally results from a reduction of the bacterial load or an alteration of the composition of the bacterial flora at the periodontal site, but the antibacterial effect of mechanical treatment alone is insufficient, providing the rationale for chemically augmenting the mechanical therapy.

- Many strategies have been used to deliver antimicrobial agents to the periodontal pocket at effective doses to reduce the bacterial microflora, including systemic administration or local administration by local irrigation of fluid formulations or placement of various gels or ointments.
- None of these strategies has proved as effective as controlled-release antimicrobials.

- A drawback of antimicrobials delivered to the pocket but not in controlled-release formulations results from the dynamics of the GCF. The GCF fills the periodontal pocket space, but the copious flow out of the pocket continuously moves GCF contents to the oral cavity. The flow rate can be markedly enhanced in the setting of inflammation.
- Antimicrobials delivered to the pocket are quickly washed out of the pocket by the GCF, rapidly reducing the concentration of drug locally to sub-antimicrobial levels.
- Locally delivered drug concentrations may need to be elevated above usual levels because microorganisms in the pocket can exist within a protective biofilm structure in the periodontal ecosystem.
- Bacterial biofilms can be highly resistant to penetration by fluids, emphasizing the critical need for high concentrations of active antimicrobials, which are achievable only with locally delivered, controlled-release agents but not possible with locally delivered antimicrobials not in controlled-release formulations or by systemic routes.
- Drug concentrations of systemically delivered antimicrobials in the GCF are orders of magnitude less than those achievable with controlled-release local delivery, and they cannot provide equivalent therapy.
- Drug that is transferred from the plasma compartment to the GCF in the periodontal pocket is quickly washed away by the GCF flow. It was previously thought that systemically delivered tetracyclines markedly concentrated drug in the GCF compared with plasma but the hypothesis was not supported by later studies. Some investigators have suggested that systemic therapies, rather than locally delivered agents, should be considered when there are multiple pocket sites for the sake of convenience.

Limitations of mechanical plaque control

- Mechanical plaque control is representing the essential therapy in periodontitis. However, this type of therapy is not free from limitations. These could be:
  1. Time consuming
  2. Required high level of manual skills
  3. Adequate motivation
  4. Cannot remove MO from all infected sites, such as concavities, lacunae, dentinal tubules and soft tissue
  5. Substantial hard tissue trauma may arise from repeated mechanical treatment in locally unresponsive sites or sites with recurrent disease.

Periodontal MO and AB

- Periodontitis is an infectious disease. However, it is different from other infections that affect the body in few important points, that should be considered in AB therapy:
  - Bacteria that cause periodontitis is commensally found in the oral cavity.
  - Lack of massive bacterial invasion to the tissue. Although, there is evidence for bacterial penetration in severely diseased periodontium, especially in acute periodontal abscess and necrotizing periodontal diseases.

  True bacterial invasion includes multiplication of the bacteria within the tissue. This is crucial in periodontal disease. In periodontitis, MO interact with the host immune system without direct contact or true invasion. Therefore, for an AB agent to be effective in periodontitis, it needs to be available in a sufficiently high concentration not only in the periodontal tissue, but also in the environment of the periodontal pocket.

Route of AB administration

Systemic

- Intramuscular and intravenous
- Orally (*per os, PO)*

Locally

- Ointments, drops, creams…etc.

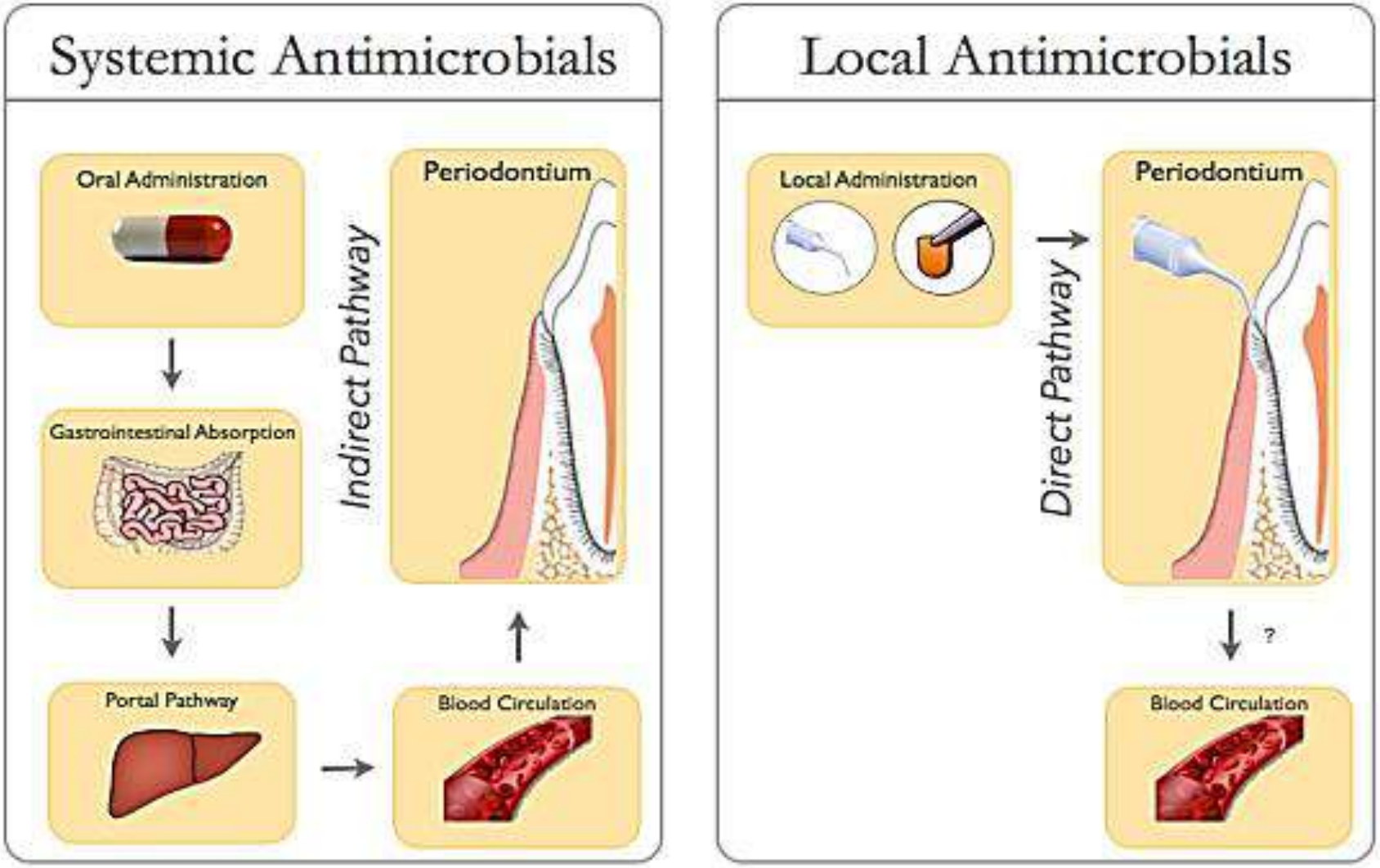
These routes can be used in periodontal therapy

In systemic route, only a small proportion of AB reaches the subgingival area, as the AB has dispersed over the whole body. In addition, adverse drug reaction are of great concern in oral route than using locally administered. It also requires good patient compliance.

In locally delivered route, <u>for the AB agent to be effective, it should be distributed allover the area especially the base of the pocket. In addition, it should be maintained in a sufficient concentration for some time.</u>

Locally delivered AB in periodontitis can include a wide range from simple pocket irrigation to installing a sophisticated devices for sustained release of the agent.

GCF has shown to be able to wash out agent brought into the pocket. The estimated half-time of non-binding agents is about 1 minutes. That is mean even a highly concentrated highly potent agent can be diluted below minimally inhibitory


Systemic Antimicrobials
Oral Administration
Gastrointestinal Absorption
Portal Pathway
Blood Circulation
Indirect Pathway
Periodontium
Local Antimicrobials
Local Administration
Direct Pathway
Periodontium
?
Blood Circulation

Indications for LABD

1. • **Isolated** periodontal pockets (>5mm), with successful phase-1 therapy (scaling and root planing)

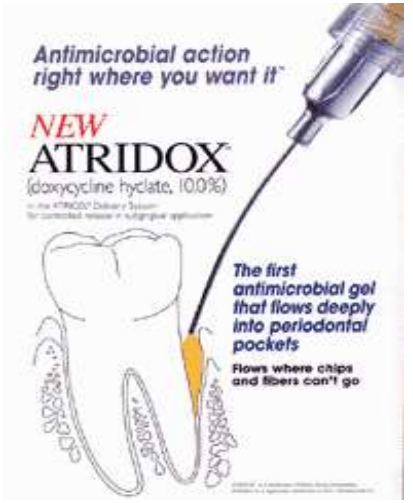
2. • Medically compromised patients where surgical therapy is contraindicated or not suggested

3. • In patients suffering from recurrent or refractory periodontitis

- Additionally, as an adjunct to periodontal regenerative procedures
- Periimplantitis
- Patients with risk factors

Local Drug Delivery Systems

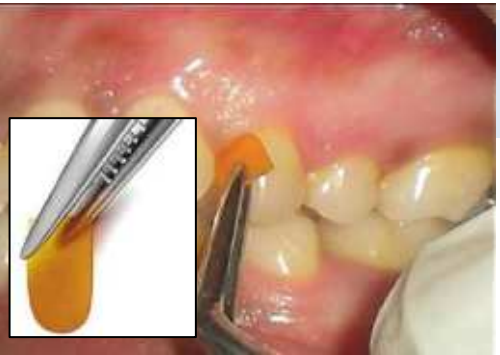
1. Actisite (Tetracycline)



2. Atridox (Doxycycline)

3. Periocline (Minocycline)

4. Elyzol (Metronidazole)



5. Periochip (Chlorhexidine)

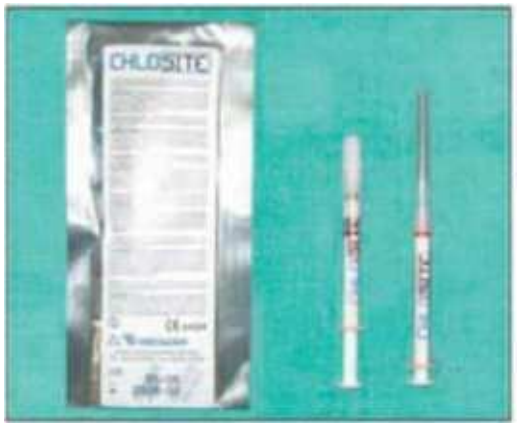
Chlorhexidine-based products:

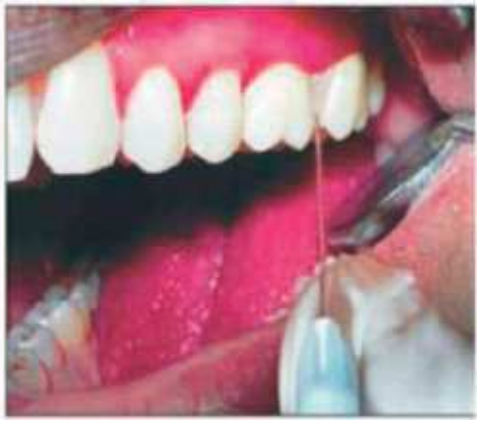
The chlorhexidine chip is a small chip (4.0 × 5.0 × 0.35 mm)

- contains 2.5 mg of the active ingredient chlorhexidine gluconate in a resorbable, biodegradable matrix of hydrolyzed gelatin that is cross-linked with glutaraldehyde and packaged in individual foil containers
- The chip is stored at 20° to 25° C.
- The chlorhexidine chip is placed into the pocket directly from the foil container using a forceps
- Indicated as an adjunct to SRP procedures for the reduction of pocket depth in adults with periodontitis, and it can be used as part of a periodontal maintenance program, which includes good oral hygiene and SRP.1
- After placement in the pocket, the chip has been reported to release chlorhexidine into GCF over 7 to 10 days.
- The chip is biodegradable and does not require removal, but dental floss should be avoided for 10 days to avoid dislodging it.

➢ PerioCol-CG

- PerioCol-CG is a small, 10-mg chip (4 × 5 × 0.25–0.32 mm) designed as a collagen matrix into which chlorhexidine gluconate (2.5 mg) is incorporated from a 20% chlorhexidine solution that is its active ingredient.
- The chip is designed for insertion into the periodontal pocket and resorbs after 30 days, but its coronal edge degrades within 10 days.
- It releases chlorhexidine *in vitro* at a rate of approximately 40% to 45% in the first 24 hours, followed by a linear release for 7 to 8 days.



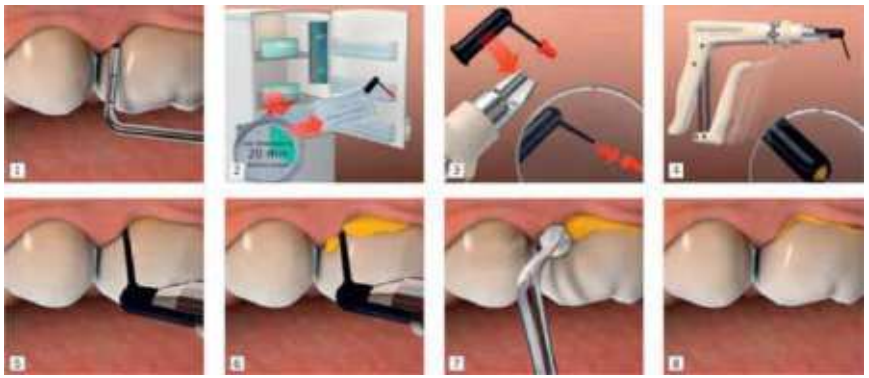


➢ Chlo-Site

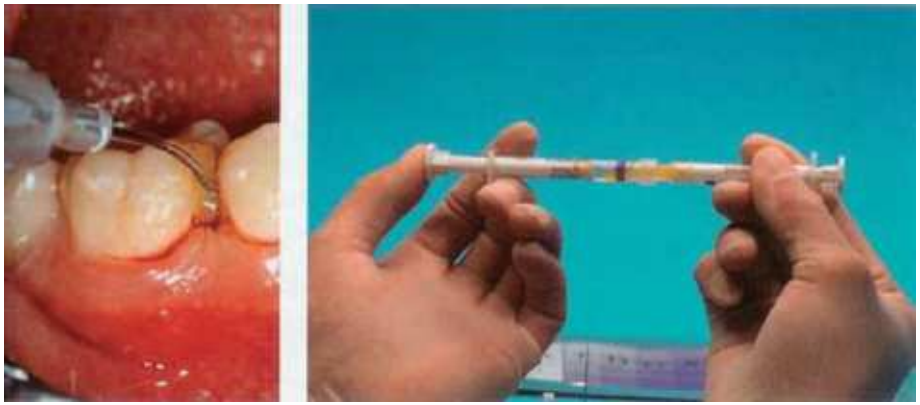
- is a xanthan gel, consisting of a saccharide polymer as a three-dimensional mesh containing 1.5% chlorhexidine in 0.5 mL of gel, which is injected into the periodontal pocket.
- The gel product is sterilized by gamma radiation and is individually packed for delivery in 0.25-mL prefilled syringes fitted with a blunt side-exit needle.
- The gel contains two types of chlorhexidine: a slow-release chlorhexidine digluconate (0.5%) and a rapid-release chlorhexidine dihydrochloride (1.0%).
- The gel is retained within the pocket and is not easily dislodged by the GCF or saliva.
- The gel disappears from the pocket in 10 to 30 days and is reported to achieve a chlorhexidine concentration in GCF of more than 1µg/mL for an average of 6 to 9 days and to maintain an effective concentration for at least 15 days.

Doxycycline-Based Products

- Doxycycline Gel
- Doxycycline gel is a subgingival, controlled-release delivery product
- composed of a two-syringe mixing system. Syringe A
- contains 450 mg of a bioabsorbable polymeric formulation.
- Syringe B contains 50 mg of doxycycline hyclate, equivalent to 42.5 mg of doxycycline.
- The two syringes are stored at 2° to 30° C.
- When mixed, the product is a viscous liquid of 500 mg, which contains 50 mg (10%) of doxycycline hyclate.

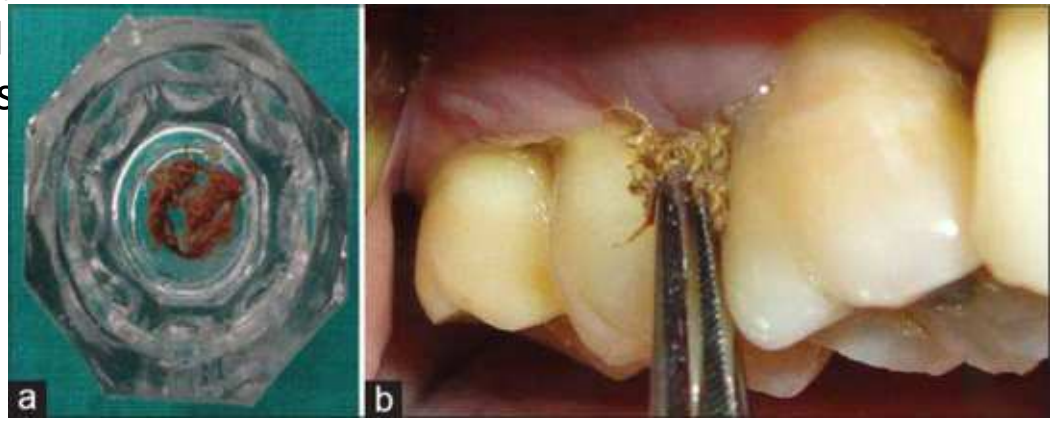


- Ligosan Slow Release
- Ligosan Slow Release is a 14% (w/w), resorbable doxycycline gel
- for periodontal application provided in a laminate pouch and stored
- under refrigeration. It contains 1, 2, 4, 8, 10, or 16 single-application
- cylinder cartridges, each containing 260 mg of Ligosan Slow Release.
- The product is used by inserting the cartridge into the caulking gun, opening the spray nozzle, and then discharging the gel to the bottom of the pocket.
- Concentrations in the GCF remained above 16 µg/mL for at least 12 days.
- Mechanical hygiene in the area should be avoided for 7 days.



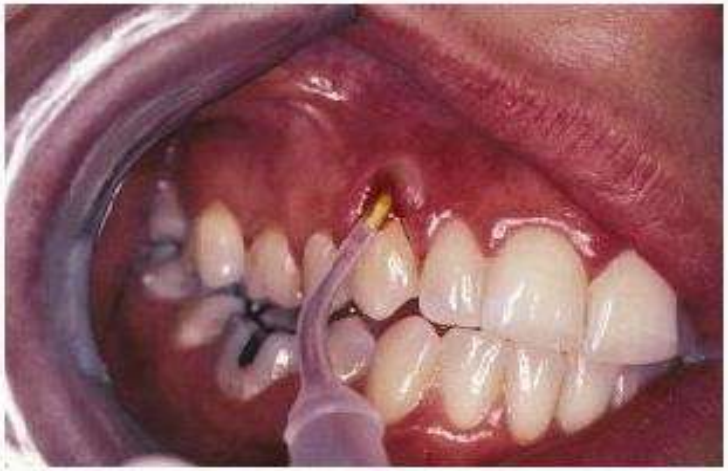
Periodontal Plus AB

- It is a bioresorbable tetracycline fiber. It is 25 mg of pure fibrillar collagen evenly impregnated with approximately 2 mg of tetracycline hydrochloride.
- The fibers are packaged as a strip containing four individually packed and separable sterile product packs.
- The fiber biodegrades in the periodontal pocket within 7 days.
- The fiber should be retained with a periodontal dressing or covered with a dental adhesive for 10 days



Minocycline microsphere

- The minocycline microspheres product is a subgingival, controlled release delivery system containing the antibiotic minocycline hydrochloride incorporated into a bioresorbable poly polymer in unit-dose cartridges.
- Each cartridge delivers minocycline hydrochloride equivalent to 1 mg of minocycline free base.
- Minocycline microspheres are indicated as an adjunct to SRP for the reduction of pocket depth in patients with adult periodontitis and as part of a periodontal maintenance program, which includes good oral hygiene and SRP.



minocycline microspheres (Arestin).

Clinical Use of locally delivered AB

- Additional dental indications for locally delivered, controlled-release antimicrobials have not been evaluated by the FDA. Examples include
- combination adjunctive therapy and adjuncts for surgical therapy or peri-implantitis. Preliminary data for these indications are available

Combination Adjunctive Therapy

- Locally delivered, controlled-release antimicrobials enhance the clinical efficacy of SRP. Similarly, adjunctive systemic therapy with low-dose (20 mg) doxycycline, given orally twice daily as a host modulating agent (later reported as a once-daily, modified-release formulation) also can enhance the clinical efficacy of SRP.
- A combination of local antimicrobial and host-modulating adjunctive therapies can provide greater clinical benefit than either adjunctive agent used alone.
- In a 6-month clinical trial, adjunctive therapies resulted in significantly greater improvements in probing depth and clinical attachment compared with SRP alone.More sites showed a probing depth reduction of 2.0 mm or more, and fewer sites had a residual probing depth 5.0 mm or more.
- The potential for combined adjunctive therapy to enhance clinical benefit is promising and warrants additional research.
- SRP plus adjunctive therapy could be considered a new standard for nonsurgical periodontal therapy. Available data also support the adjunctive efficacy of systemic, low-dose oral doxycycline to an extent that is numerically similar to that reported for locally delivered, controlled-release antimicrobials.
- In addition to the possibility of combination adjunctive therapy with a locally delivered agent and host modulatory therapy with low dose oral doxycycline, Maximal reductions in probing depth and gains in attachment level were further augmented by systemic antimicrobials and surgical treatment.

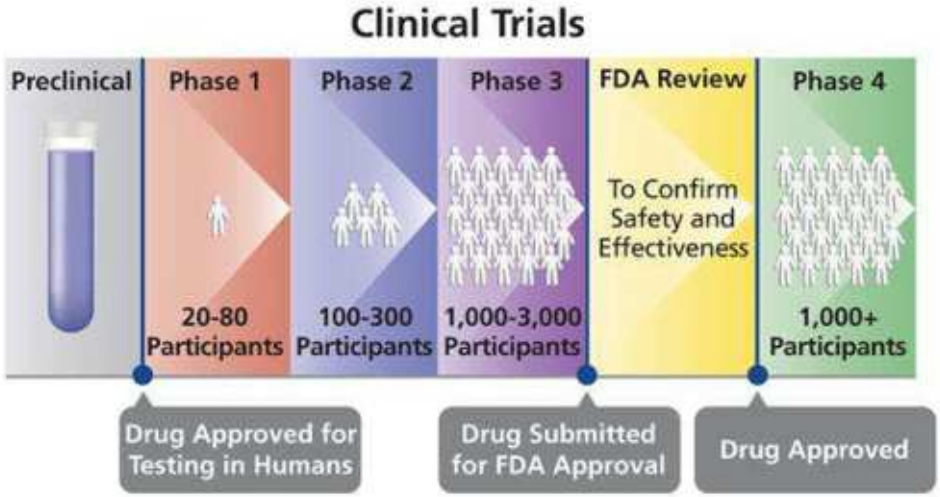
Surgical Therapy

- Pocket sites that do not seem to respond adequately to nonsurgical therapy and evidence residual probing depth with inflammation are often treated with follow-up surgical therapy.
- Locally delivered, controlled-release antimicrobials can augment surgical results.
- A specialized periodontal surgical procedure, regenerative surgery,
- has become a standard procedure in periodontal practice. It had been
- suggested that adjunctive locally delivered, controlled-release antimicrobials might improve outcomes after regenerative periodontal surgery. In a pilot trial, the adjunctive use of the chlorhexidine chip with regenerative surgery resulted in more than a 100% greater mean improvement from baseline in bone height and mass 9 months after surgical treatment compared with SRP alone and surgery.Both groups had also received prophylactic systemic antimicrobial treatment before surgery,
- A microbiologic rationale for the use of locally delivered antimicrobials (i.e., tetracycline fiber) in the regenerative setting was also supported by research.
- Some suggested that regenerative surgical procedures should include an adjunctive, locally delivered, controlled-release antimicrobial agent to provide a more consistent clinical benefit.
- These preliminary reports suggested that the adjunctive use of locally delivered, controlled-release antimicrobials can improve clinical outcomes after periodontal surgery in regenerative and nonregenerative settings.

Peri-implantitis

- Similar to periodontitis, peri-implantitis is an inflammatory disease process that is initiated by local microorganisms and that affects the tissues surrounding an implant. An opportunity exists to treat diseased implant sites chemically by targeting the local microflora. There is a potential rationale for the use of locally delivered, controlled-release antimicrobials for the treatment of peri-implantitis.
- Such as, adjunctive minocycline microspheres and doxycycline gel in combination with SRP with plastic instruments.

- Phase III trials are needed
- to test the hypothesis that locally delivered, controlled-release
- antimicrobials offer clinical benefit as part of a treatment regimen to manage peri-implantitis.



Tobacco Smoking

- Smoking is a well-known risk factor for the development or progression of periodontitis, and it can limit the effectiveness of periodontal therapy. The adjunctive use of locally delivered, controlled-release antimicrobials can enhance the efficacy of SRP in smokers.
- In a 3-month trial, SRP plus adjunctive doxycycline gel resulted in significantly greater probing depth reduction and clinical attachment gain compared with SRP alone about equally in smokers and nonsmokers.
- This result was consistent with subset analyses of current smokers, former smokers, and nonsmokers
- from two 9-month, multicenter trials of doxycycline gel and of microspheres smokers versus nonsmokers in studies of minocycline microspheres. Subset analyses of therapy with minocycline microspheres showed that results for smokers were consistent with overall trial results.
- Adjunctive therapy may lessen the adverse impact of smoking on the periodontium and improve treatment outcomes for patients who smoke.
- A systematic review of the doxycycline gel and minocycline microspheres found that available data are insufficient to conclude that adjunctive therapy significantly enhances SRP specifically in smokers, and it recommended additional clinical trials (i.e., adequate and well-controlled trials) to assess outcomes for smokers.

Cautions for Use

- Clinicians are cautioned about adverse effects of this class of drugs:

1. Potential for hypersensitivity reactions (i.e., not to be used in patients with a known sensitivity to any ingredient).
2. Potential for the overgrowth of nonsusceptible microorganisms, including fungi
3. Use in pregnancy
4. Potential for discoloration during tooth development (i.e., tetracyclines only)
5. Use in an acutely abscessed periodontal pocket or in extremely severe periodontal defects with little remaining periodontium
6. Use of local mechanical oral hygiene procedures (e.g., toothbrushing, interdental cleaning devices) for approximately 7 to 10 days after administration

Adverse Effects

- The most frequently reported adverse reactions reported in the clinical trials without mention of causality include headache, infection (including upper respiratory tract infection), flu syndrome, pain, tooth disorder and toothache, and various oral signs or symptoms.
- Toothache was the only adverse reaction that was significantly higher ($P = .042$) in the chlorhexidine chip group compared with placebo.

Thank you

Immunology

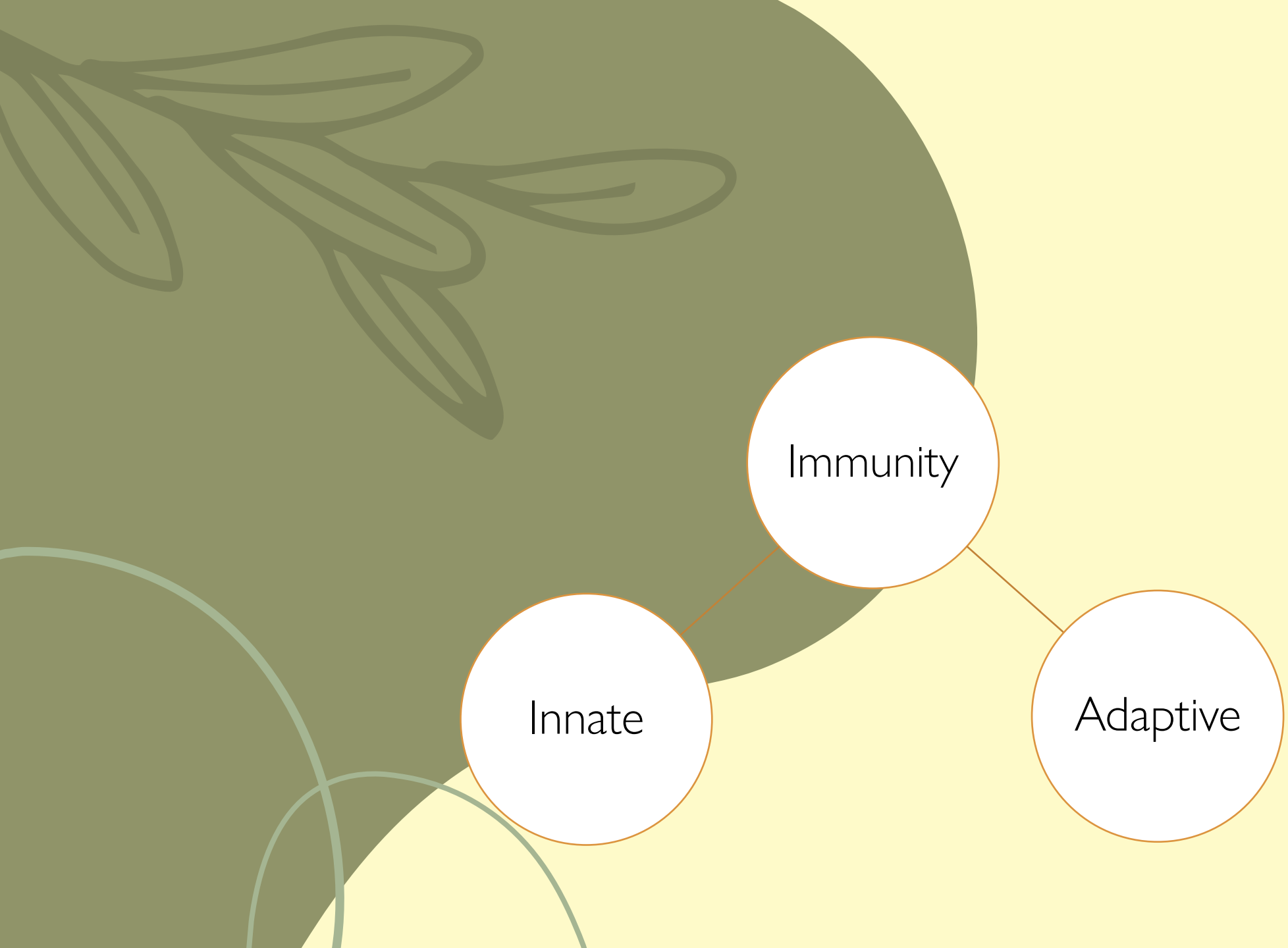
Dr Hadeel Mohammed Abbood

Year 5, Periodontology

College of Dentistry, Tikrit University

Immune System

- The immune system is essential for the maintenance of periodontal health,
- It is central to the host response to periodontal pathogens.
- If the immune response is dysregulated, inappropriate, persistent, or excessive in some way, then damaging chronic inflammatory responses
- The immune response to plaque bacteria involves the integration at the molecular, cellular, and organ level of elements


Immunity
Innate
Adaptive

Innate Immunity

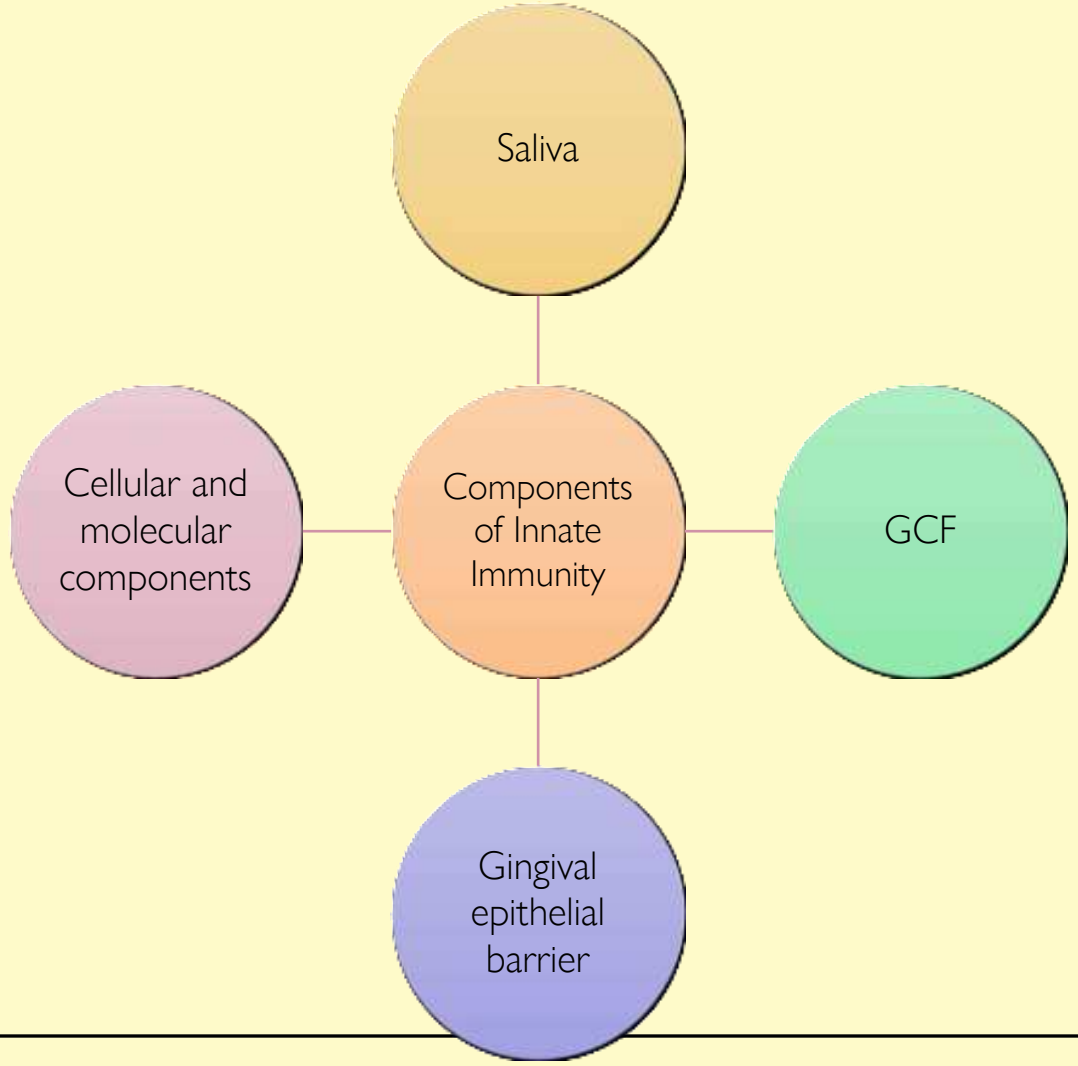
- Defenses against infection include a wide range of mechanical, chemical, and microbiologic barriers that prevent pathogens from invading the cells and tissues of the body.
- Saliva, GCF, and the epithelial keratinocytes of the oral mucosa all protect the underlying tissues of the oral cavity and the periodontium.
- The commensal microbiota (e.g., In dental biofilm) may also be important for providing protection against infection by pathogenic microorganisms through effective competition for resources and ecologic niches and also by stimulating protective immune responses.
- If bacterial products enter the tissues, then the cellular and molecular elements of the innate immune response are activated.

Characteristics of Innate Immunity

- The term innate immunity refers to the elements of the immune response that has 3 main characteristics:
- 1- these elements are determined by inherited factors (and therefore “innate”),
- 2- they have limited specificity,
- 3- they are “fixed” in that they do not change or improve during an immune response or as a result of previous exposure to a pathogen.

- The recognition of pathogenic microorganisms and the recruitment of effector cells (e.g., Neutrophils) and molecules (e.g., The complement system) are central to effective innate immunity.
- Innate immune responses are orchestrated by a broad range of cytokines, chemokines, and cell surface receptors,
- The stimulation of innate immunity leads to a state of inflammation.
- If innate immune responses fail to eliminate infection, then the effector cells of adaptive immune responses (lymphocytes) are activated.
- Immune response functions as a network of interacting molecular and cellular elements in which innate immunity and adaptive (antigen-specific) immunity work together toward a common purpose.

Components of Innate Immunity



Saliva

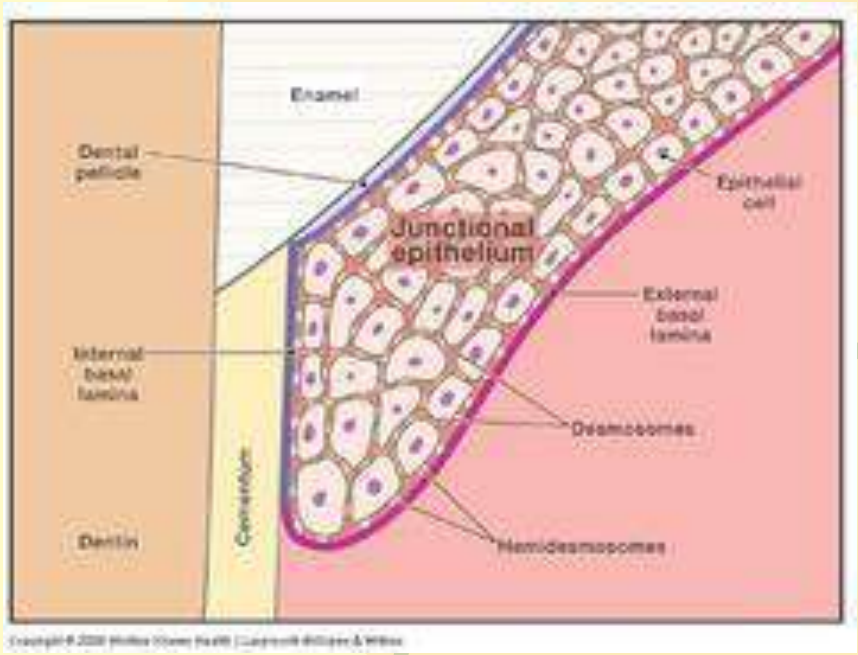
- Saliva that is secreted from the three major salivary glands (i.e., parotid, submandibular, and sublingual), as well as from the numerous minor salivary glands,
- It has an important role in the maintenance of oral and dental health.
- The action of shear forces associated with saliva flow is important for preventing the attachment of bacteria to the dentition and oral mucosal surfaces.
- Human saliva also contains numerous molecular components that contribute to host defenses against bacterial colonization and periodontal disease.

Saliva Constituent	Host Defense Function
Antibodies (e.g., immunoglobulin A)	Inhibit bacterial adherence, promote agglutination
Histatins	Neutralize lipopolysaccharides, inhibit destructive enzymes
Cystatins	Inhibit bacterial growth
Lactoferrin	Inhibits bacterial growth
Lysozyme	Lyses bacterial cell walls
Mucins	Inhibits bacterial adherence, promotes agglutination
Peroxidase	Neutralizes bacterial hydrogen peroxide

Gingival epithelial barrier

- The epithelial tissues play a key role in host defense because:
  - They are the main site of the initial interactions between plaque bacteria and the host,
  - They are also the site of the invasion of microbial pathogens.
- The keratinized epithelium of the sulcular and gingival epithelial tissues provides protection for the underlying periodontal tissue,
- Acting as a barrier against bacteria and their products.

Junctional epithelium



- The junctional epithelium has significant

intercellular spaces,

- It is not keratinized, and
- It exhibits a higher cellular turnover rate.
- These properties render the junctional epithelium permeable, thereby allowing for the inward movement of microbes and their products and the outward movement of GCF and the cells and molecules of innate immunity.
- The spaces between the cells of the junctional epithelium widen with inflammation, which results in increased GCF flow.

Stimulated epithelial cells can produce:

- Matrix metalloproteinases (MMPs), which contribute to a loss of connective tissue.
- Epithelial cells also secrete a range of cytokines in response to periodontal bacteria, which signal immune responses. These include:
  - Proinflammatory cytokines: IL-1β, TNF-α, and IL-6,
  - Chemokine IL-8 and the monocyte chemoattractant protein-1 (MCP-1), which serve to signal neutrophil and monocyte migration from the vasculature into the periodontal tissue.

Gingival crevicular fluid (GCF)

- GCF originates from the postcapillary venules of the gingival plexus.
- It has a flushing action in the gingival crevice,
- It also likely functions to bring the blood components (e.g., neutrophils, antibodies, complement components) of the host defenses into the sulcus.
- The flow of GCF increases in inflammation, and neutrophils are an especially important component of GCF in periodontal health and disease.

Pathogen recognition and activation of cellular innate responses:

Plaque bacteria and their products penetrate the periodontal tissues,

Specialized “sentinel cells” macrophages and dendritic cells recognize their presence and signal protective immune responses.

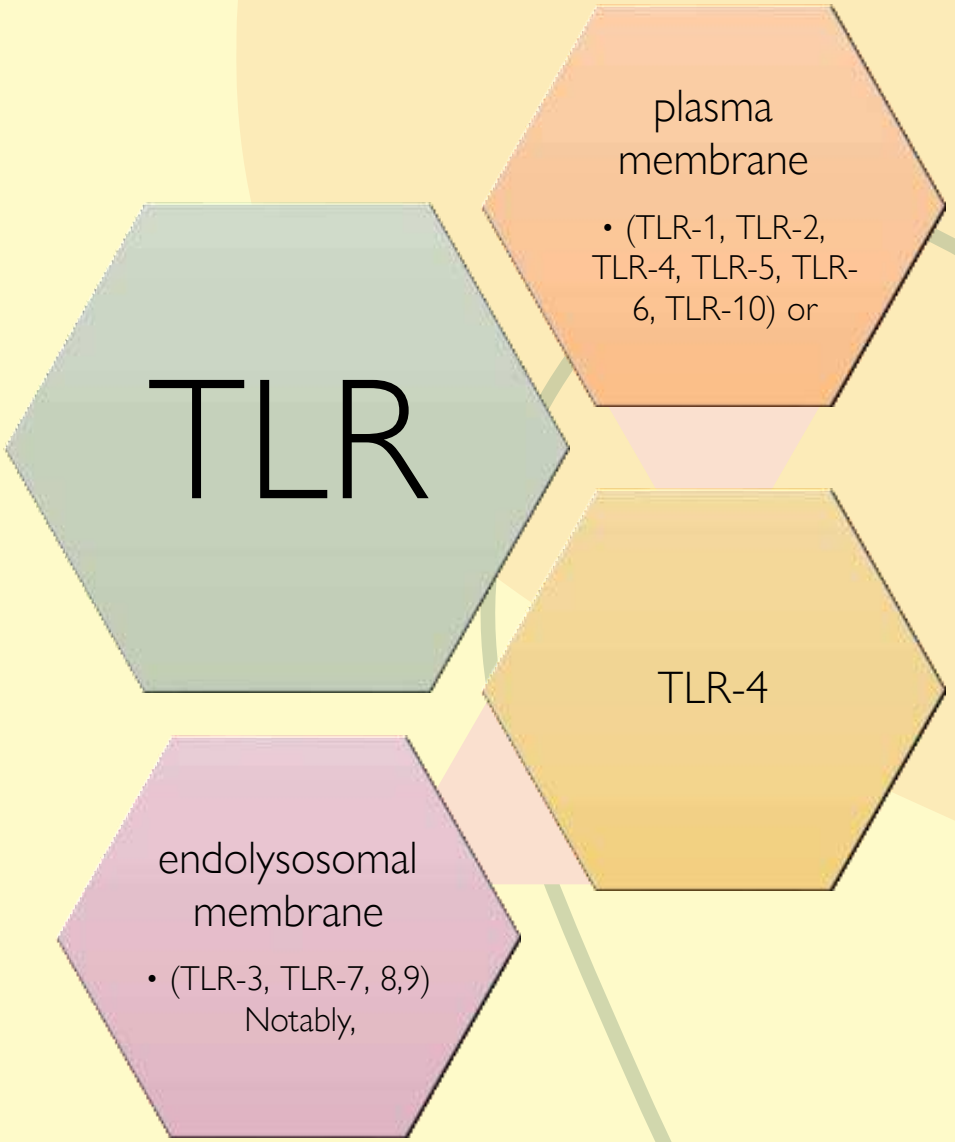
These cells express a range of pattern recognition receptors (PRRs) that interact with microbe-associated molecular patterns (MAMPs).

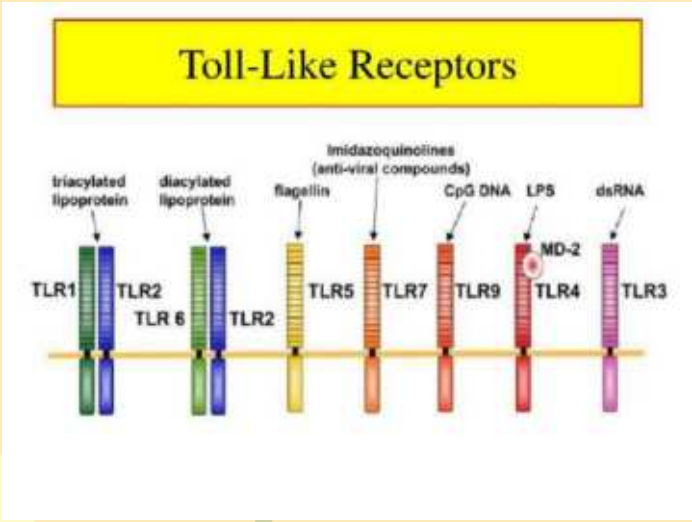
The activation of PRRs activates innate immune responses to provide immediate protection, and adaptive immunity is also activated with the aim of establishing a sustained antigen-specific defense.

- Excessive and inappropriate or dysregulated immune responses lead to chronic inflammation and the concomitant tissue destruction associated with periodontal disease.

Toll like receptors (TLR):

- The TLR family currently consists of 10 known functional TLRs in humans, of which TLR-10 is the only member having an unclear biologic role.
- Bacterial lipopolysaccharides (LPS) with TLRs:
- P. gingivalis, A. actinomycetemcomitans, and F. nucleatum all possess LPS molecules that interact with TLR-4 to activate myeloid immune cells.

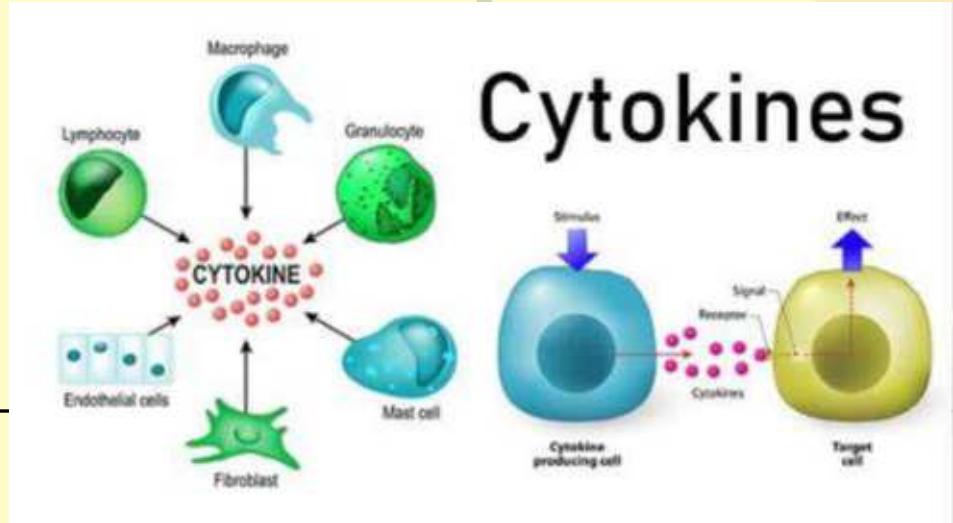




- Individual species of plaque bacteria have a wide variety of MAMPs, which may interact with PRRs.
- P. gingivalis:
  - LPS signals via TLRs (predominantly TLR-2),
  - Fimbriae, proteases, and DNA from P. gingivalis are all recognized by host cells through interaction with specific PRRs.
- Certain nonimmune cells in the periodontium (e.g., epithelial cells, fibroblasts) also express PRRs and may recognize and respond to MAMPs from plaque bacteria.

Pro inflammatory cytokines

- Although the signaling pathways activated by PRRs may be diverse, in general terms, they converge to elicit similar host cell responses in the form of:
  - The up-regulation of cytokine secretion
  - Cell differentiation that leads to enhanced signaling of the adaptive immune response.



The signaling of cytokine responses by PRRs influences:

- innate immunity (e.g., neutrophil activity),
- adaptive immunity (e.g., T-cell effector phenotype),
- The development of destructive inflammation (e.g., the activation of fibroblasts and osteoclasts).

IL-1β

- Activating other cells that express the IL-1R1 receptor (e.g., endothelial cells)
- Stimulating the synthesis and secretion of other, secondary mediators such as PGE2.
- IL-1β also stimulates the secretion of the chemokine IL-8, which stimulates neutrophil chemotaxis.

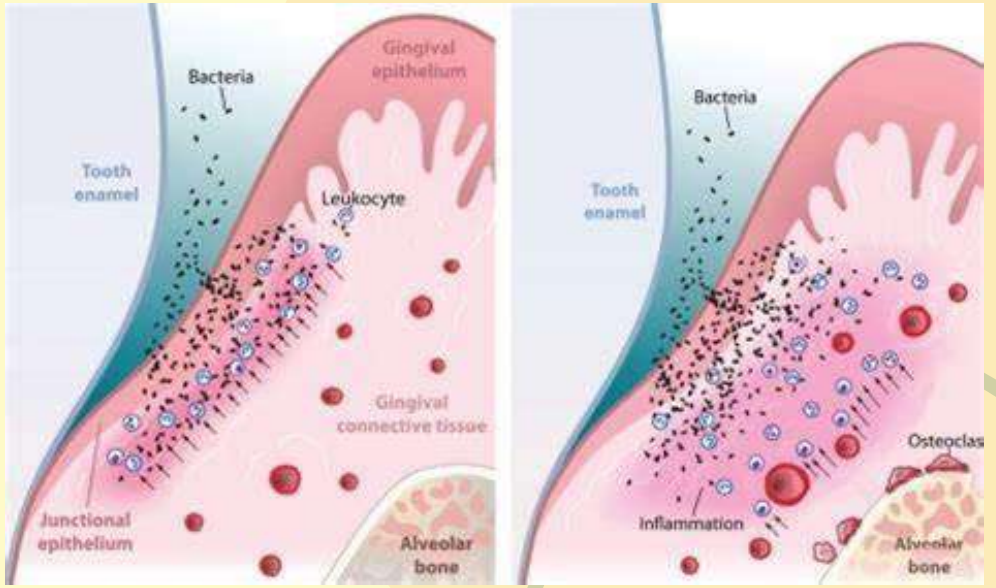
- IL-1β acts synergistically with TNF- α and stimulate migration of neutrophils to the periodontium.

- IL-1β and TNF-α also activate MMP secretion from fibroblasts and osteoclasts; this facilitates the movement of neutrophils through the connective tissues (and thus protective innate responses), but it also contributes to the tissue destruction associated with periodontal disease, along with MMPs from neutrophils.

Other cytokines

- Up-regulated as a result of the activation of PRRs include IL-6, which influences the development of a number of immune cells (e.g., B cells, dendritic cells) and stimulates osteoclast differentiation and thus bone turnover.
- Cytokines from T-cell subsets feedback to and modify innate immune responses; for example, IFN-γ from Th1 cells activates macrophages, and IL-10 and TGF-β suppress immune responses.
- Cytokines are pleiotropic (i.e., they have multiple effects).

Cells of innate immunity:

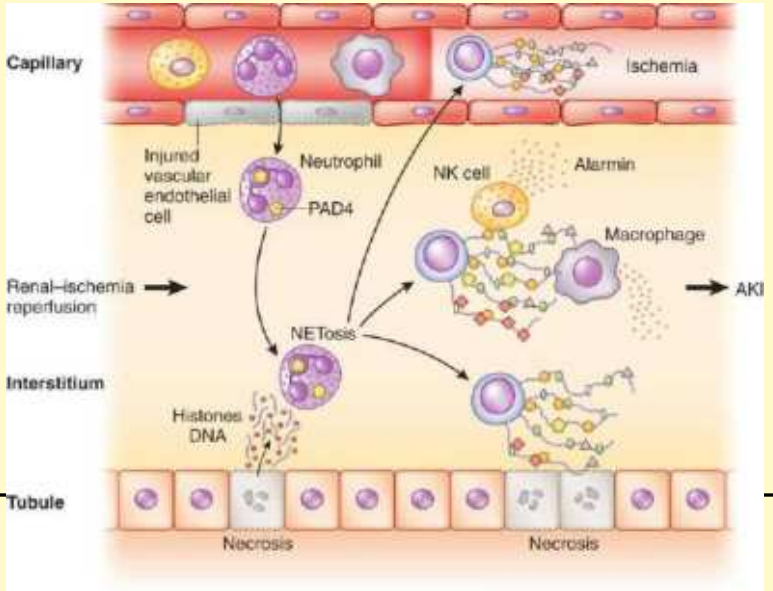


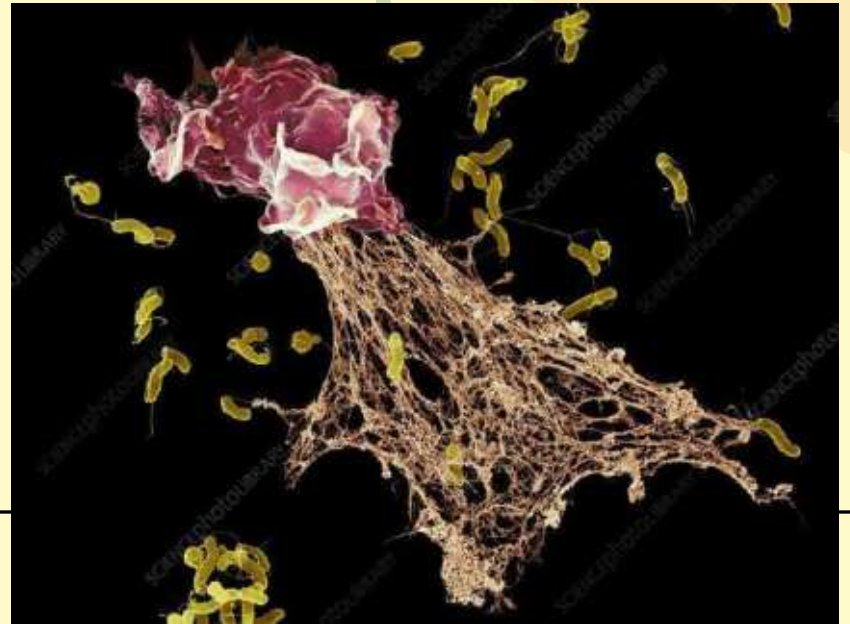
- **Neutrophils**
- Neutrophils are the “professional” phagocytes that are critical to the clearance of bacteria that invade host tissues.
- Present in clinically healthy gingival tissues, and they migrate through the intercellular spaces of the junctional epithelium into the sulcus. This is part of a “low-grade defense” against plaque bacteria, and it is necessary to prevent infection and periodontal tissue damage.
- The importance of neutrophils to the maintenance of periodontal health is demonstrated clinically by the observations of severe periodontitis in patients with neutrophil defects.

- A small proportion (1% to 2%) of the intercellular spaces in healthy junctional epithelium is occupied by neutrophils (and other leukocytes at various stages of differentiation),
- This can increase to 30% with even modest inflammation.
- In the inflammatory state, changes to the local vasculature occur in the gingiva: high endothelial venules develop from the postcapillary venules of the gingival plexus, which facilitates leukocyte emigration and increases the flow of GCF into the pocket.
- Neutrophils migrate from the gingival plexus to the extravascular connective tissue and then into the junctional epithelium through the basement membrane.
- The presence of a layer of neutrophils in the junctional epithelium forms a host defense barrier between subgingival biofilm and the gingival tissue.
- The migration of neutrophils contributes to the disruption of the junctional epithelium by the degradation of the basement membrane through protease release and the action of reactive oxygen species (ROS).

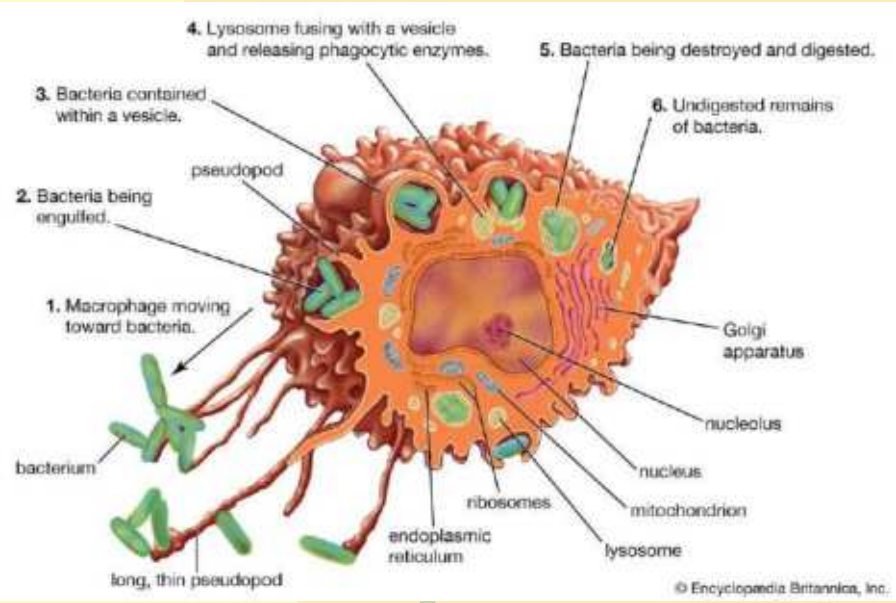
neutrophil extracellular traps (NETs).

- An aspect of neutrophil-mediated immunity
- NETs constitute a highly conserved antimicrobial strategy in which decondensed nuclear DNA and associated histones are extruded from the neutrophil, thus forming weblike strands of DNA in the extracellular environment.
- These strands, in conjunction with antimicrobial peptides (AMPs), facilitate the extracellular killing of microorganisms that become trapped within the NETs.





Macrophages



- One of the key elements of the innate immune system and are involved in the initiation, development, and resolution of inflammatory diseases, including periodontitis.
- In addition to immunostimulatory roles, they play pivotal roles in immunoregulation and tissue repair.
- Macrophages can be differentiated into osteoclast cells.
- They possess PRRs that can recognize invading bacteria and signals the immune response.
- They are important cells in innate and adaptive immune response. In the stage of repair and resolution of inflammation, Lipoxins signal macrophages to phagocytose the remnants of apoptotic cells at sites of inflammation without generating an inflammatory response.

Adaptive Immunity

Adaptive immunity

- Adaptive immunity has evolved to provide a focused and intense defense against infections that overwhelm innate immune responses.
- Adaptive immunity is particularly important as ecologic, social, and demographic changes—which alter susceptibility to existing and emerging infective microorganisms—outpace the natural evolution of biologic systems.
- The development of effective vaccination is, along with the identification of antibiotics, perhaps one of the greatest triumphs of medical science; this success is based on knowledge of the elements and principles of adaptive immunity.

Characteristics

- Adaptive immunity contrasts with innate immunity with regard to the dynamic of the underlying cellular and molecular responses:

1. adaptive immunity is slower
2. relies on complex interactions between APCs and T and B lymphocytes.
3. The antigen specificity of the responses of a diverse range of effector elements, including cytotoxic T cells and antibodies.
4. The ability of adaptive immune responses to improve during exposure to antigen and on subsequent reinfection events.

- Our current understanding suggests that the cellular and molecular elements of adaptive immunity are more diverse than those of innate immunity,
- Although a role for many of these factors in periodontal disease has been identified, our knowledge is far from complete.
- The importance of adaptive immune responses in periodontal pathogenesis is endorsed by histologic studies of established lesions in periodontal disease.

gingivitis and stable periodontal lesions *VS.* active (progressing) periodontitis

- Dominated by T cells, and these cells are clustered mainly around blood vessels.
- These cells are activated but not proliferating.
- A predominance of the helper T-cell subset (i.e., CD4-expressing T cells) over the cytotoxic T-cell subset (i.e., CD8-expressing T cells) is observed.
- These T cells are considered to be proactively maintaining tissue homeostasis in the presence of the microbial challenge of the plaque biofilm.

- B cells and plasma cells predominate and are associated with pocket formation and the progression of disease.

Cellular elements:
Antigen-Presenting Cells (APCs)

- Central elements of the activation and function of T cells and B cells are the presentation of antigen by specialized APCs to T cells and the development of a specific cytokine milieu that influences the development of T cells with particular effector functions.
- APCs detect and take up microorganisms and their antigens, after which they may migrate to lymph nodes and interact with T cells to present antigen.
- In periodontium: APCs are B cells, macrophages, and at least two types of dendritic cells (i.e., dermal dendritic cells and Langerhans cells).
- It is increasingly recognized that the engagement of PRRs (and in particular TLRs) by MAMPs from pathogenic microorganisms is not only central to signaling innate immunity in the form of cytokine up-regulation but also a critical element of the activation of APCs and the elaboration of T-cell effector function.
- Thus, TLR activation increases the expression of molecules on APCs, which are critical to the interaction of these cells with T cells. In addition, TLR activation enhances antigen uptake and processing.

T Cells

- Several different subsets of thymic lymphocytes (i.e., T cells) develop in the bone marrow and thymus and migrate to the peripheral tissues to participate in adaptive immune responses.
- The expression of the cell surface molecules (CD4 or CD8) or particular T-cell antigen receptors (αβ or γδ) broadly defines functional T-cell subsets that emerge from the thymus.
- CD4+ helper T cells are the predominant phenotype in the stable periodontal lesion, and it is thought that alterations in the balance of effector T-cell subsets within the CD4+ population may lead to progression toward a destructive, B-cell–dominated lesion.
- The best-defined functional subsets of CD4+ T cells are the Th1 and Th2 cells, and a dynamic interaction between Th1 and Th2 cells may provide, in part, an explanation for fluctuations in disease activity and the progression of periodontal disease.

stable periodontal lesion *vs.* destructive lesion

strong innate response results in IL-12 synthesis (e.g., by tissue macrophages)

That leads to a Th1 response that provides protective cell-mediated immunity.

Th1 cells secrete IFN-γ, which activates cell-mediated immunity (i.e., macrophages, NK cells, and CD8+ cytotoxic T cells) against pathogenic microorganisms.

Stable periodontal lesion

A poor innate response would lead to reduced IL-12

Permit the development of Th2 responses

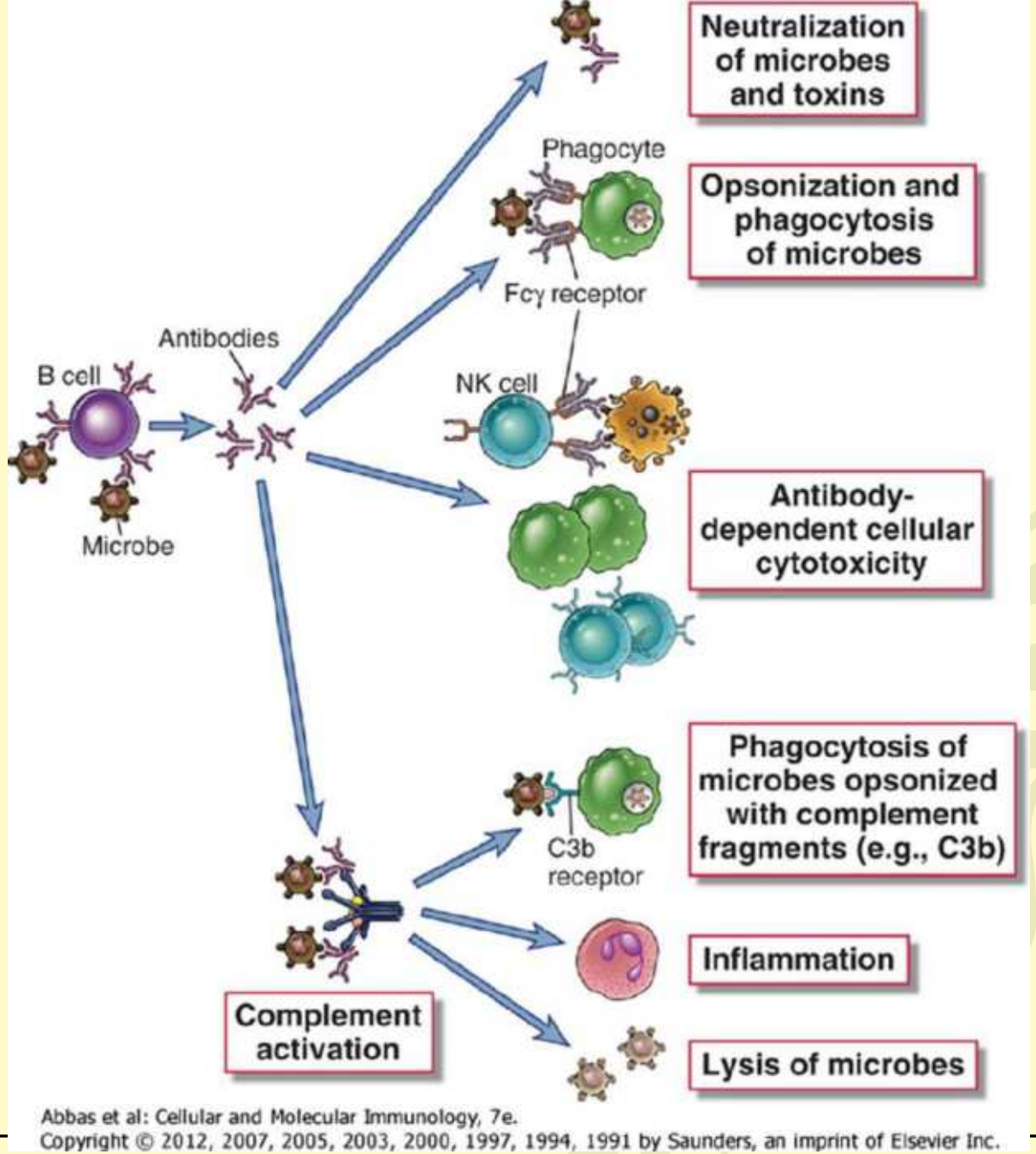
Lead to the activation of B cells;

enhanced B-cell– derived IL-1β.

Destructive lesion

B-cells:

- The production of specific antibodies that would serve to clear tissue infections through:
  - Interaction with the complement system
  - Enhancing neutrophil phagocytosis.
  - Neutralization of microbes and toxins
- B cells are also a source of proinflammatory cytokines that contribute to tissue destruction.

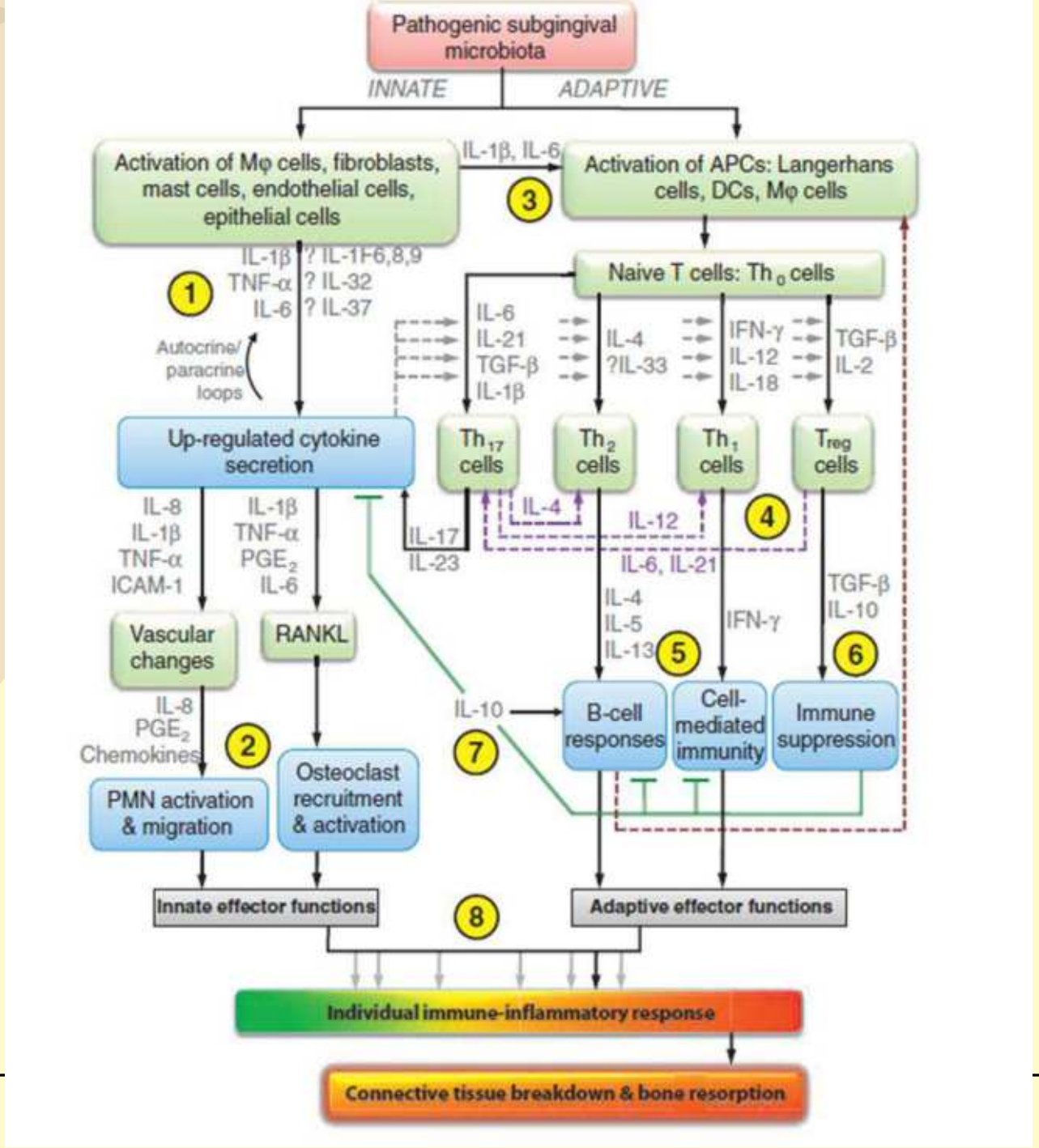


The humoral response to plaque

- Specific antibodies are produced in response to the bacterial challenge in periodontal disease and are the endpoint of B-cell activation.
- Differentiated plasma cells.
- High levels of antibodies appear in GCF (in addition to those in the circulation), and these are produced locally by plasma cells in periodontal tissues.
- Antibodies to periodontal pathogens are primarily IgG, with few IgM or IgA types produced.
- Many species of oral bacteria elicit a polyclonal B-cell response that augment responses against nonoral bacteria and may lead to the production of autoantibodies (e.g., antibodies against collagen and connective tissue proteins), which may contribute to tissue destruction in periodontal disease.

- Variations in the levels of specific antibodies to different species in different clinical presentations suggest differences in pathogenesis.
  - For example, antibodies to A. actinomycetemcomitans of the IgG2 subclass predominate in aggressive periodontitis.
  - Other P. gingivalis molecules (i.e., fimbriae and hemagglutinin) also act as antigens. Specific antibodies are also generated by serotype-specific carbohydrate antigens (e.g., capsular polysaccharide of P. gingivalis, carbohydrate of A. actinomycetemcomitans LPS).

- Individuals with aggressive periodontitis have monocytes that are hyperresponsive to LPS and that produce elevated quantities of PGE2. A. actinomycetemcomitans is commonly associated with aggressive periodontitis, these bacteria trigger cytokines release from monocytes and as a consequence, increase IgG2
- The significance of antibodies in periodontitis is not clear. It is not known whether these antibodies have a protective function or whether they participate in disease pathogenesis.


Pathogenic subgingival microbiota
INNATE
ADAPTIVE
Activation of Mφ cells, fibroblasts, mast cells, endothelial cells, epithelial cells
IL-1β, IL-6
3
Activation of APCs: Langerhans cells, DCs, Mφ cells
IL-1β
TNF-α
IL-6
? IL-1F6,8,9
? IL-32
? IL-37
1
Naive T cells: Th0 cells
Autocrine/ paracrine loops
IL-6
IL-21
TGF-β
IL-1β
IL-4
?IL-33
IFN-γ
IL-12
IL-18
TGF-β
IL-2
Up-regulated cytokine secretion
Th17 cells
Th2 cells
Th1 cells
Treg cells
IL-8
IL-1β
TNF-α
ICAM-1
IL-1β
TNF-α
PGE2
IL-6
IL-17
IL-23
IL-4
IL-12
4
IL-6, IL-21
IL-4
IL-5
IL-13
IFN-γ
TGF-β
IL-10
5
6
Vascular changes
RANKL
IL-10
7
B-cell responses
Cell-mediated immunity
Immune suppression
IL-8
PGE2
Chemokines
2
PMN activation & migration
Osteoclast recruitment & activation
Innate effector functions
8
Adaptive effector functions
Individual immune-inflammatory response
Connective tissue breakdown & bone resorption

Osteo-immunology in periodontal diseases

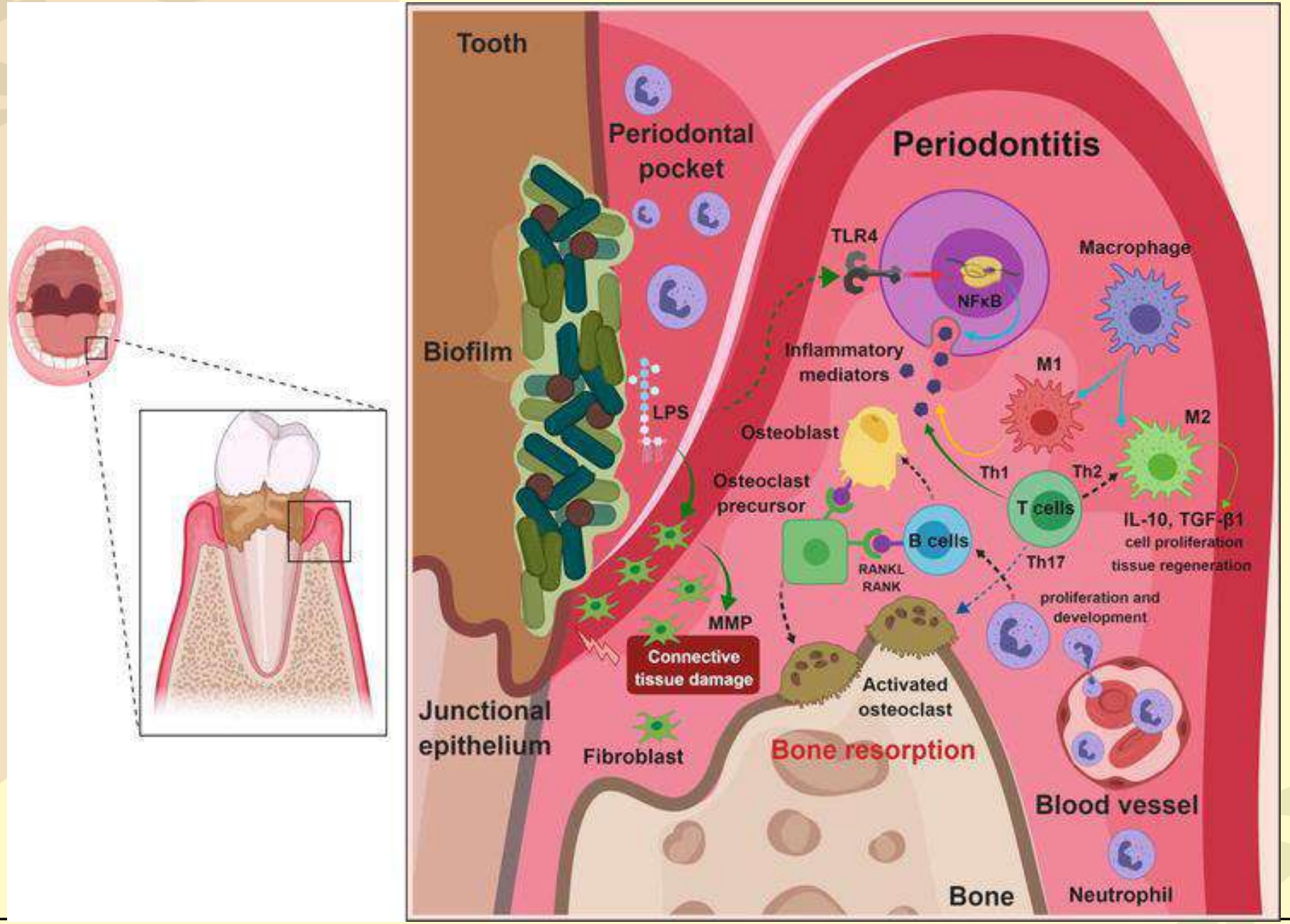
- As the advancing inflammatory front approaches the alveolar bone, osteoclastic bone resorption commences. This is a protective mechanism to prevent bacterial invasion of the bone, but it ultimately leads to tooth mobility and even tooth loss. The resorption of alveolar bone occurs simultaneously with the breakdown of the periodontal ligament in the inflamed periodontal tissues. Two critical factors determine whether bone loss occurs:
- (1) the concentration of inflammatory mediators in the gingival tissues must be sufficient to activate the pathways that lead to bone resorption.
- (2) the inflammatory mediators must penetrate to within a critical distance of the alveolar bone.

- Histologic studies have confirmed that the bone resorbs so that a width of non-infiltrated connective tissue of about 0.5 to 1.0 mm overlying the bone is always present. It has also been demonstrated that bone resorption ceases when at least a 2.5-mm distance is present between the site of bacteria in the pocket and the bone.
- Osteoclasts are stimulated by proinflammatory cytokines and other mediators of inflammation to resorb the bone, and the alveolar bone “retreats” from the advancing inflammatory front.
- Osteoclasts are multinucleated cells that are formed from osteoclast progenitor cells and macrophages. Osteoclastic bone resorption is activated by a variety of mediators (e.g., IL-1β, TNF-α, IL-6, PGE2).

Receptor Activator of Nuclear factor-Kappa beta (RANK)

- Osteoclast can be also activated by RANK which is expressed by osteoclast progenitor cells. This receptor is activated by RANK Legand (RANKL) which is a member of the TNF family.
- RANKL is produced by osteoblasts and stromal cells of the bone marrow. It is also produced by T-cells and other inflammatory cells. When RANKL is attached to RANK the process of differentiation of progenitor cells into active osteoclast will be initiated.
- Osteoprotegrin (OPG) is another member of the TNF family, however, when it binds to RANKL; it inhibits its activity.
- In patients with periodontitis, it is detected that the GCF contains reduced OPG levels and elevated levels of RANKL.
- The process of bone destruction is the imbalance between osteoclast activating and inhibiting cytokines

- TNF-α and IL-1 trigger bone resorption by controlling the osteoclast formation and keeping it in a continuous increase.
- IL-1 also affects the osteoclastogenic effect of TNF-α by reinforcing the differentiation of osteoclast precursors and the expression of RANKL.
- Cytokines like IL-1β play a vital role as it induces RANKL expression in various cells which include osteoblasts, periodontal ligament fibroblasts, and gingival fibroblasts.
- In the same way, IL-6 is produced by different cells such as osteoblasts and fibroblasts. It plays an extremely important role in inducing osteoclast formation and promoting bone resorption.
- In addition, the inflammatory mediator prostaglandin E2 is very necessary for this process. It triggers bone resorption by the upregulation of RANKL expression and the inhibition of OPG expression in osteoblastic cells.


Tooth
Periodontal pocket
Periodontitis
TLR4
NFκB
Macrophage
Biofilm
Inflammatory mediators
M1
LPS
Osteoblast
M2
Th1
Th2
Osteoclast precursor
T cells
IL-10, TGF-β1
cell proliferation
tissue regeneration
B cells
RANKL
RANK
Th17
proliferation and development
MMP
Connective tissue damage
Activated osteoclast
Junctional epithelium
Fibroblast
Bone resorption
Blood vessel
Bone
Neutrophil

How does a pocket develop?

- The bacterial biofilm causes inflammation in the gingival tissues that causes swelling, and therefore the sulcus deepens slightly (false pocket).
- The inflammatory response may subsequently spread to the deeper tissues and is characterized by infiltration by defense cells and breakdown of collagen in the connective tissues.
- The junctional epithelium migrates apically to maintain an intact epithelial barrier, and thus the sulcus becomes deeper again and is now referred to as a pocket.
- Bacteria in the biofilm proliferate apically, exploiting and perpetuating this environmental niche.
- The bacteria are never completely eradicated by the host response, and thus they continue to provoke an immune–inflammatory response, leading to progressing tissue breakdown, continued apical migration of the junctional epithelium, resorption of alveolar bone, and gradual deepening of the pocket

Resolution of Inflammation

- Inflammation is an important defense mechanism to combat the threat of bacterial infection, but inflammation also results in tissue damage associated with the development and progression of most chronic diseases associated with aging, including periodontal disease.
- Resolution of inflammation (i.e., “turning off” inflammation) is an active process that is regulated by specific mechanisms that restore homeostasis.
- It is possible that controlling or augmenting these mechanisms may lead to the development of new treatment strategies for managing chronic diseases such as periodontitis.
- It is mediated by specific molecules, including a class of endogenous, proresolving lipid mediators that includes the lipoxins, resolvins, and protectins. These molecules are actively synthesized during the resolution phases of acute inflammation;
    - they are antiinflammatory, and they inhibit neutrophil infiltration. They are also chemoattractants, but they do not cause inflammation. For example, lipoxins stimulate infiltration by monocytes but without stimulating the release of inflammatory cytokines.

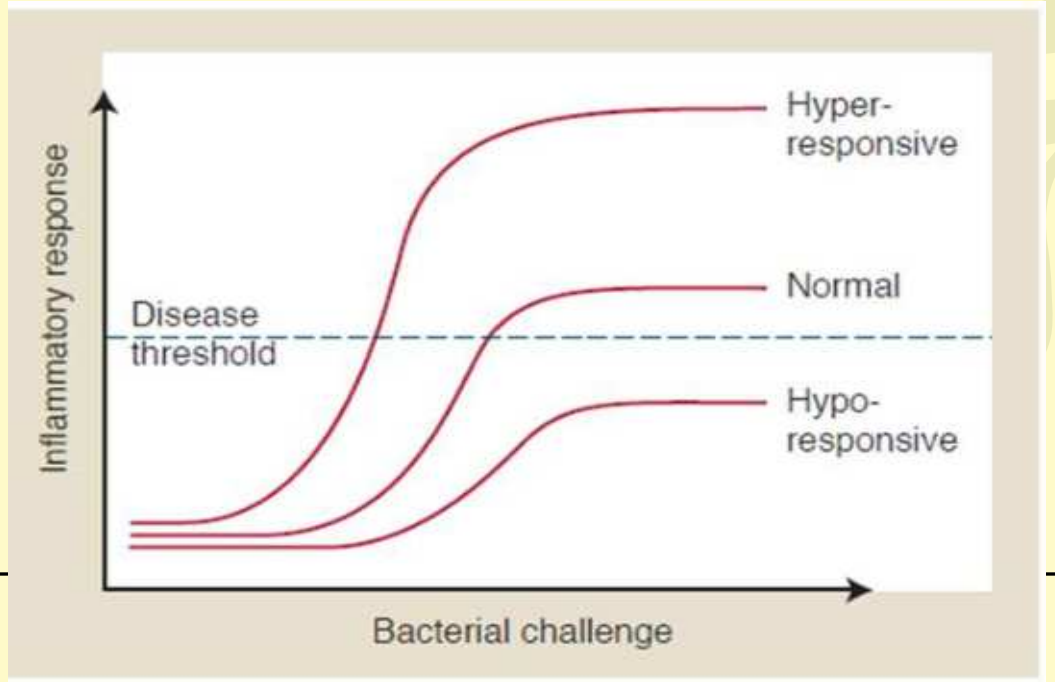
Individual variations in response to bacterial plaque

- Immune responses to the bacterial challenge do not occur in isolation, but rather take place in the context of other host and environmental factors that influence these responses and thereby determine the progression of disease.
- Certain risk factors increase susceptibility to periodontal disease, particularly smoking and diabetes.
- Quantitative and qualitative differences exist in immune responses among individuals. This could be due to infectious agents (e.g., bacteria) exert evolutionary selection pressures on the species that they infect.

“hyperinflammatory” or “hyporesponsive” trait

- Some studies have confirmed that immune cells from patients with periodontal disease secrete higher quantities of proinflammatory cytokines than do cells from persons who are periodontally healthy.
- Cytokine profiles are also different in those individuals with immune-mediated diseases as compared with healthy control subjects.
- These observations have led to the trait concept in which certain individuals possess a hyperinflammatory phenotype that accounts for their increased susceptibility to chronic inflammatory conditions such as periodontitis.
- Such a trait may also underpin shared susceptibility between conditions such as periodontitis and cardiovascular disease or diabetes.
- at present, it is not possible to identify with certainty those patients who are hyper-responders.

- It is likely that many reasons contribute to disease variations among individuals, such as variations in immune responses, pathogenesis, and the plaque biofilm
- This situation results in an uneven disease experience in the population.
- although plaque bacteria initiate the inflammatory response, most of the tissue damage results from the host response, which is influenced by genetic factors, as well as environmental and acquired risk factors.
- Risk factors such as smoking alter the progression of the immune–inflammatory response and shift the balance toward increased periodontal breakdown



Therapeutic Strategies

- Various treatment strategies have been developed to target the host response.
- Matrix metalloproteinase (MMP) inhibitors (e.g., low-dose formulations of doxycycline) have been used in combination with scaling and root planing or surgical therapy.
- High-risk patient populations (e.g., diabetic patients, patients with refractory periodontal disease) have benefited from the systemic administration of MMP inhibitors.
- Soluble antagonists of TNF-α and IL-1 can be delivered locally to periodontal tissues in nonhuman primates.
- Other therapeutic strategies are aimed at inhibiting the signal transduction pathways involved in inflammation. Inflammatory mediators including proinflammatory cytokines (e.g., IL-1, TNF, IL-6), MMPs, and others would be inhibited at the level of the cell-signalling pathways required for the transcription factor activation necessary for inflammatory gene expression or mRNA stability.
- Novel analogues of defensins have shown even higher antibacterial activity than the endogenous -defensins 1 and 3, without any cytotoxic effects on host cells, thus indicating the promise of this approach.

Thank you

Epidemiology of periodontal disease

Dr Hadeel Mohammed Abbood

The need for epidemiology

- Epidemiology is the study of the distribution and the determinants of health-related status or event in a population.

- It represents the basic science of public health. In public health clinics they look for the determinants of the disease in the whole population. This might be different from the determinants of the same disease in individuals that clinicians look for in clinical practices. For example, the determinants of periodontitis in individuals are dental plaque and calculus, whereas, public health practitioners might determine other causes for periodontitis in population, such as low socioeconomic status or lack of access to dental clinics.

- In epidemiology, the distribution of a given disease is usually described by percentage and the number of persons affected among subgroup, such as gender, socioeconomic status, ethnicity, education level or other characteristics in the population. The difference in distribution among subgroups is not random. It is because of physical, biological, behavioural, cultural, social or other factors that make this subgroup more susceptible to disease than other subgroups.

Why is it important to study epidemiology?

1. From studying the epidemiology of the disease, we can know the prevalence of the disease, and whether it is rare or prevalent. It can provide us information about the natural history of the disease too.
2. Epidemiologic studies can identify the causes and the risk factors of the disease
3. It can provide guidance and recommendations for prevention and control of the disease.
4. The new molecular epidemiologic studies can help to identify the perfect time of intervention in the continuum process of the disease
5. It can give us an idea about the prognosis of the disease under specific treatment.

Measuring the occurrence of conditions or diseases:

- Prevalence
- Risk
- The odds
- Incidence rate

Prevalence

- is the sum of all examined individuals or sites that exhibit the condition or disease of interest divided by the sum of the number of individuals or sites examined. The prevalence can range from 0 to 100%.
- As an example of prevalence, the CDCP reported about the prevalence of individuals with at least one periodontal pocket depth of 4 mm or deeper:
  - from 1988 to 1994, a little more than 1 in 5 Americans had such a condition, for a prevalence of a little more than 20%;
  - from 1999 to 2004, only 1 in 10 Americans fell into this category, for a prevalence of around 10%.
  - These findings suggested a 50% decline in the prevalence of ≥4 mm PD for adults aged 20 and 64 years, which occurred over approximately a decade.
  - Many countries do not have prevalence surveillance systems, which makes it difficult to determine whether these trends observed in the United States are isolated events or part of a more general trend.

Risk

- The *risk* is the probability that an individual or a site will develop a particular condition or disease during follow-up.
- The risk for a condition or a disease is a number that ranges between 0% and 100%.
- The simplest way to estimate risk is to have a fixed number of persons or sites at risk at some defining moment (i.e., time zero [t0]). After a follow-up period (i.e., from t0 to tn), the risk can be calculated as the proportion of persons or sites in which the clinical outcome of interest develops during the follow-up period.
- When a risk is reported, it should be accompanied by a specific time period to which it is applied. A 5% risk for death may be considered small when it refers to a 20-year period but large when it refers to a 3-month period.
- As an example, consider concerns about occupational human immunodeficiency virus (HIV) infection among dentists. It has been reported that the risk for developing an HIV infection within the year subsequent to an accidental needle stick with HIV-contaminated blood is 0.3%. Such a statistic has an intuitive appeal and can be related to patients or colleagues.
    - A risk of 0.003 (0.3%) indicates that for every 1000 individuals who have an accidental HIV contaminated needle stick, 3 are expected to develop an HIV infection within a year of the event.

The odds

- The odds for an event is the probability that an event occurred divided by the probability that an event did not occur.
- Whereas probability is a value that has to range between 0 and 1, odds values range from 0 to infinity. If the probability for observing an event is small, then the odds and the probability are almost identical. For example, if the probability for a vertical root fracture after an endodontic procedure is 0.001, then the odds are 0.001/0.999 or 0.001001.
- Odds are commonly reported in studies because they are often easier to estimate with statistical models than probabilities. For example, the odds for developing an HIV infection after an accidental needle stick with HIV-contaminated blood are 0.003 (0.003/0.997).

Incidence rates

- are an alternative measure to describe disease occurrence. In clinical trials or epidemiology, the rate reflects the number of disease occurrences per person-time or site-time. the disease rate is a ratio in which the numerator is the number of subjects or sites diagnosed with the disease of interest and the denominator is the sum of the time at risk overall subjects or sites in the population.

- Incidence rates—as opposed to the previously introduced measures of disease occurrence—imply an element of time. The denominator in the incidence rate has time as the dimension.

- Thus, the dimension of incidence rate is 1/time. This dimension is often referred to as “person-time” or “site-time” to distinguish the time summation from ordinary clock-time. The magnitude of the incidence rate can vary between 0 and infinity. When there are no new disease onsets during the study period, the incidence rate is 0. When every person observed dies instantaneously at the start of the study (and thus the sum of the time periods is 0), then the incidence rate is infinity.

- An example of the application of rates is provided in Fig. 6.1, in which the number of teeth lost per 1000 tooth-years is plotted as a function of the maximum probing depth at the start of follow-up. The plot suggests a nonlinear relationship between maximum pocket depth and tooth loss, with a substantial increase in tooth loss rate for teeth that have periodontal pockets of 7 mm or deeper.

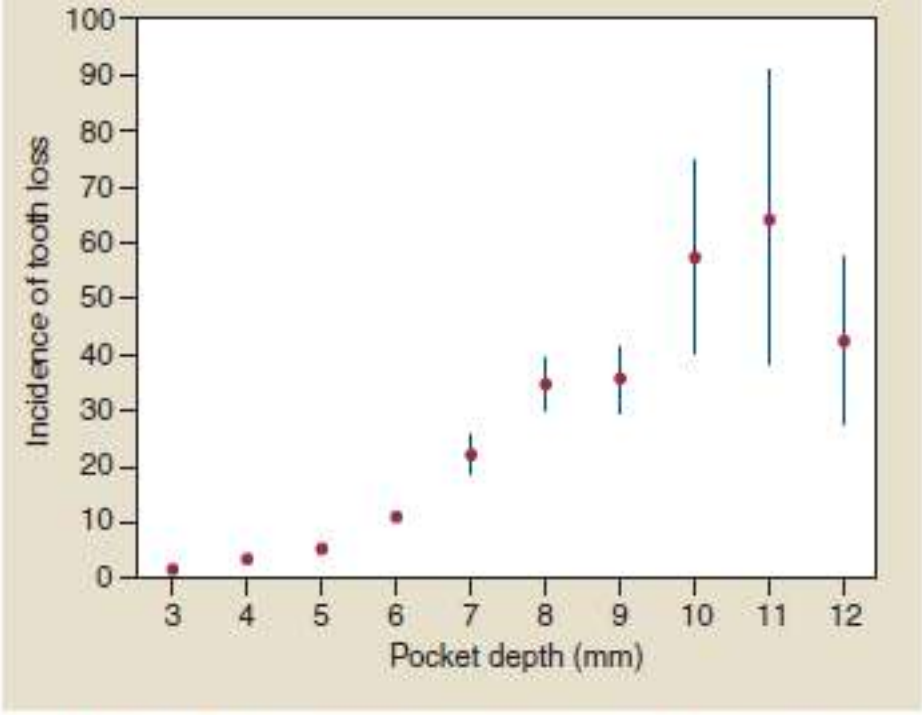


Fig. 6.1 Rate of tooth loss per 1000 tooth-years as a function of maximum probing depth per tooth in a cohort of 1021 patients between the ages of 40 and 65 years under periodontal specialist care for destructive periodontal disease. *(Data from Hujoel PP, Cunha-Cruz J, Selipsky H, et al: Abnormal pocket depth and gingival recession as distinct phenotypes.* Periodontol 2000 *39:22–29, 2005.)*

Typical measurement of periodontal disease

- A periodontal examination can measure various characteristics of the periodontium. Dental records of periodontal patients typically contain information about:
  - Present, missing or impacted teeth
  - clinical probing depth,
  - bleeding on probing,
  - gingival recession,
  - mobility of teeth,
  - furcation involvements .
  - Some clinicians may collect information about the presence of gingivitis by evaluating the color and form of the gingival tissues.
  - These measures can be complemented with radiographic examinations that may provide information about marginal bone levels.
- In research settings or in some selected private practices, additional periodontal measures may be collected, such as clinical attachment levels, microbiologic measures, gingival crevicular fluid volume, biomarkers in the GCF, and indices that measure the amount of gingival inflammation or dental plaque or debris accumulation.

Two common measures of gingival inflammation are:

1. Gingival Index (GI) and
2. Bleeding on probing.

- The GI was proposed in 1963 as a method for assessing the severity and quantity of gingival inflammation. With this particular index, only gingival tissues are assessed. Each of the four gingival areas of the tooth (i.e., facial, mesial, distal, and lingual) are assessed for inflammation and rated as normal gingiva (a score of 0) to severely inflamed gingiva with a tendency to spontaneously bleed (a score of 3). Gingiva that is mildly inflamed but without bleeding on probing is given a score of 1, whereas moderately inflamed gingiva with bleeding is given a score of 2. The scores can be averaged for each patient to provide patient means.

BoP

- The specific approach to obtain a bleeding measure can be done as follows, the examiner dried a quadrant of teeth with air. Then, starting with the most posterior tooth in the quadrant (excluding the third molar), the examiner placed a periodontal probe 2 mm into the gingival sulcus at the facial site and carefully swept the probe. After probing the sites in the quadrant, the examiner assesses the presence or absence of bleeding at each probed site. The same procedure was repeated for the remaining quadrant.

- Commonly used measures of periodontal tissue destruction include
  - mean probing depth,
  - mean attachment loss,
  - and mean recession level.
- The clinical protocols regarding how such mean values are collected and calculated can vary considerably.

True and Surrogate Measures of the Periodontal Condition

- *True endpoints* are tangible outcomes that directly measure how a patient feels, functions, or survives.

True endpoints include oral health–related quality-of-life measurements and self-reported problems, such as a positive answer to the following question: “When you brush or floss your teeth, do you notice bleeding that is both regular and that involves spitting blood-stained saliva?”.

- *Surrogate endpoints* are intangible to the patient. Surrogate endpoints in periodontal research include anatomic measures (e.g., probing depth), measures of inflammation (e.g., bleeding), microbiologic measures, and immunologic measures. Surrogate endpoints are often objective, because they can be measured by the clinician (rather than relying on self-report by patients) or by laboratory methods. Surrogate endpoints can be misleading when the goal is to provide reliable information about clinical decisions related to diagnosis, etiology, treatment, or prognosis.

TABLE 2.1 Examples of Potentially Misleading Surrogates[a]

Disease or Condition	Experimental Treatment	Control Treatment	Effect on Surrogate Endpoint	Effect on True Endpoint	Misleading Conclusion	Reference
AIDS	Immediate zidovudine	Delayed zidovudine	Significant increase of 30–35 CD4 cells/mm^3	No change in incidence of AIDS, AIDS-related complex, or survival	False-positive	80
Osteoporosis	Fluoride	Placebo	Significant increase of 16% in bone mineral density of lumbar spine	Nonvertebral fracture rates increased by 85%	False-positive	
Lung cancer	ZD1839 (Iressa)	Placebo	Dramatic tumor shrinkage in 10% of patients	No effect	False-positive	82
Aphthous ulcers	Thalidomide	Placebo	Although thalidomide expected to decrease TNF-α production, significant increase of 4.4 pg/mL in TNF-α production occurred, suggesting harm	Pain diminished and ability to eat improved	False-negative	32
Edentulism dentures	Implant-supported	Conventional dentures	No impact on chewing cycles	Improved oral health–related quality of life	False-negative	5
Prostate cancer	Radical prostatectomy	Watchful waiting	Substantial elimination of tumor mass	No effect on overall mortality risk	False-positive	78
Advanced colorectal cancer	5-FU + LV	5-FU	23% of patients had 50% or greater reduction in tumor volume	No effect on overall survival	False-positive	40
Periodontitis	Surgery	Scaling	Mean pocket depth reduced by 0.5 mm	Effect on tooth loss or quality of life unknown	?	30a

[a]For some examples, the experimental treatment led to improvements in surrogate endpoints, whereas the true endpoint was either unaffected or worsened (a false-positive conclusion). For other examples, the experimental treatment had no impact or worsened the surrogate endpoint, whereas the true endpoint improved (a false-negative conclusion).
AIDS, Acquired immunodeficiency syndrome; *5-FU*, 5-fluorouracil; *LV*, leucovorin; *TNF-α*, tumor necrosis factor-alpha.

Epidemiologic Study Designs

Randomized Controlled Trials (RCT)

- RCT in periodontics typically assign patients or some teeth within a patient randomly to a treatment. Patients are then monitored, and subsequent outcomes are assessed.
- Requirement of designing an RCT;

1. there needs to be a pretrial hypothesis that specifies the endpoint,
2. the treatments to be compared,
3. the patient population,
4. the degree of required precision.
5. A secure randomization process,
6. the masking of patients and clinicians,
7. the presence of an independent data and safety monitoring board,
8. Strict adherence to the pretrial hypothesis, which must include an intent-to-treat analysis.

- It is important to strictly follow the up mentioned factors in RCTs to reach to a reliable result. Otherwise, false positive results might be obtained.

Cohort Studies

Cohort studies can also be referred to as exposure-based study designs. Subjects who are free of the disease of interest are classified with respect to an exposure (e.g., cigarette smoking, diabetes) and followed longitudinally for the assessment of periodontal outcomes.

TABLE 6.4 Examples of Periodontal Cohort Studies

Periodontal Exposure	Outcome	Sample Size
Periodontal disease and tooth loss[25]	Coronary heart disease	51,529
Gingivitis[9]	Tooth loss	>500

Cohorts can be defined by a geographic area. Some natural disease history studies of destructive periodontal disease have been conducted on the basis of geographic location.

Examples include the Norwegian Longitudinal Study.

A cohort can be defined by records (e.g., schools, health insurance plans, unions, industries, professional organizations).

Many cohort studies of periodontal disease outcomes are performed in patients who belong to a particular dental insurance company or to a professional group.

Case–Control Studies

- Persons with a condition or outcome of interest (i.e., cases) are compared with persons without a condition of interest (i.e., controls) with respect to the history of the suspected causal factors.
- The primary goal of a case–control study is to find out what past exposures or factors are different between patients with a disease versus those without the disease.
- The case–control study is a challenging type of study to conduct. Trying to minimize the role of bias in case–control studies require careful planning, conduct, and analysis. Two important elements of the case–control study design are:

1. the definitions of the terms case and control. A case is a person in the population or study group who has been identified as having a particular disease, health disorder, or condition. The case definition should be rigorous to minimize bias and misclassification; it can be based on symptoms, signs, or the results of diagnostic tests.

- In a case–control study, the controls should be at risk for developing the investigated disease and come from the same population that generated the cases. For example, if the investigated disease is root caries, the controls should be at risk for developing root caries (i.e., have exposed root surfaces) and originate from the same population that generated the cases that have root caries.

Suspected Modifiable Causative Factors for Periodontal Disease

- **Tobacco Smoking**
- Tobacco smoking is recognized by several organizations as one of the primary drivers of periodontal disease epidemiology. Many criteria for causality have been satisfied, and smoking cessation has been shown to slow the progression of periodontal disease.
- The strong impact of tobacco smoking on periodontal disease has the potential to induce spurious causal associations in other suspected risk factors for periodontal disease. For example, smoking is a risk factor for both type 2 diabetes and periodontal disease, thereby making associations between type 2 diabetes and periodontal disease susceptible to biases.
- To obtain reliable inferences about causal factors other than smoking, studies of periodontal disease epidemiology may need to be restricted to those who have never smoked.

• Nutrition

• Several studies have demonstrated relationships between periodontal disease and a variety of medical conditions that center on carbohydrate metabolism, including intake of dietary carbohydrates, exercise, obesity, prediabetes, and diabetes.

• A systematic review of randomized controlled trials involving carbohydrates suggested that the increased intake of fermentable carbohydrates may cause an increase in gingivitis.

• Two systematic reviews suggested that diabetes was a risk factor for destructive periodontal disease.

- **Dental Plaque**

- Several systematic reviews have provided evidence that chemotherapeutic and mechanical plaque control will reduce gingival inflammation.

- Essential oils and cetylpyridinium-chloride–containing mouth rinses may reduce gingival inflammation. Interdental brushes may reduce dental plaque, bleeding, and probing pocket depth. Power-driven toothbrushes may be more effective than manual toothbrushes for removing plaque and reducing inflammation.

Thank you

Interrelationship between Periodontics and other parts of dentistry

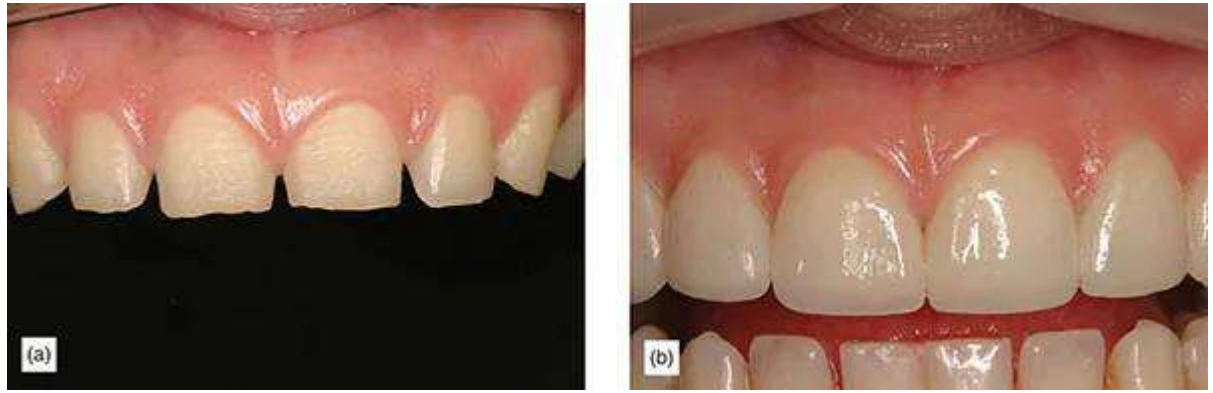
Dr Hadeel Mohammed Abbood

Restorative interrelationship

Biologic Considerations
Margin Placement and Biologic Width

- One of the most important aspects of understanding the periodontal–restorative relationship is the location of the restorative margin to the adjacent gingival tissue.
- Restorative clinicians must understand the role of biologic width:
  - in preserving healthy gingival tissues
  - controlling the gingival form around restorations.
- They must also apply this information in the positioning of restoration margins, especially in the aesthetic zone, where a primary treatment goal is to mask the junction of the margin with the tooth.
- A clinician is presented with three options for margin placement:
  - Supragingival,
  - Equigingival (even with the tissue),
  - And subgingival.

- The **supragingival margin** has the least impact on the periodontium. Classically,
- This margin location can be applied in unaesthetic areas because of the marked contrast in color and opacity of traditional restorative materials against the tooth.
- With the advent of more translucent restorative materials, adhesive dentistry, and resin cements, the ability to place supragingival margins in aesthetic areas is now a reality. Therefore, whenever possible, these restorations should be chosen not only for their aesthetic advantages but also for their favorable periodontal impact.



- The use of **equigingival margins** traditionally was not desirable because:
  - They were thought to retain more plaque than supragingival or subgingival margins and therefore resulted in greater gingival inflammation.
  - There was also the concern that any minor gingival recession would create an unsightly margin display.
- These concerns are not valid today, not only because the restoration margins can be aesthetically blended with the tooth, but also because restorations can be finished easily to provide a smooth, polished interface at the gingival margin.

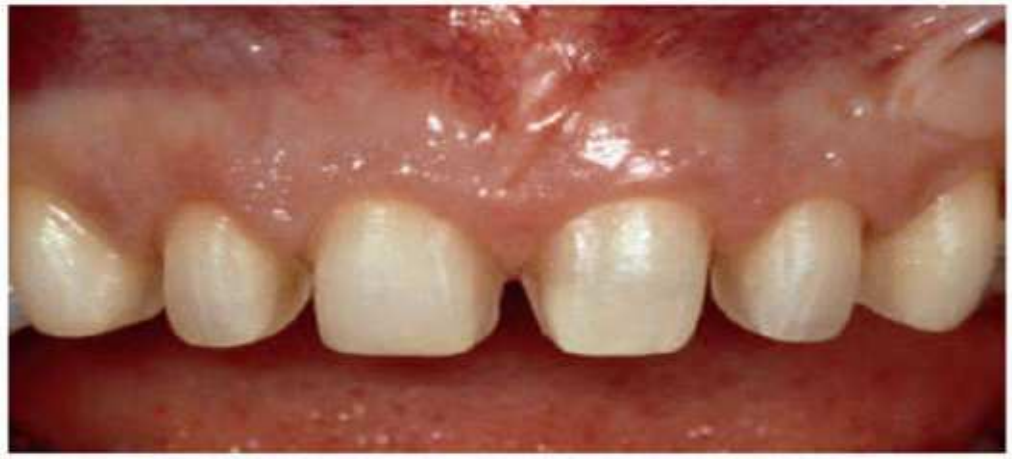


Fig. 70.1 With the advent of adhesive dentistry and ultrathin ceramic veneers, it now is possible to prepare restorations equigingival without visible margins. The preparations for six porcelain veneers with the margins placed at the level of tissue are shown.

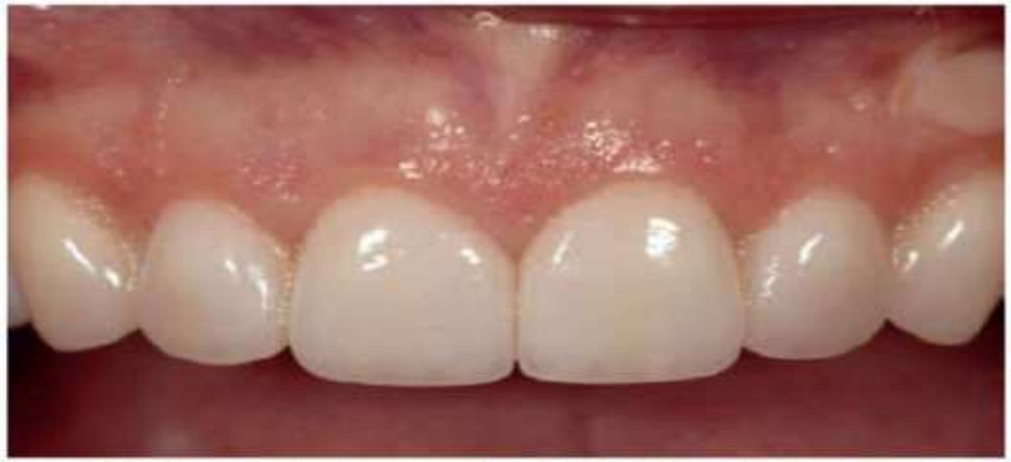


Fig. 70.2 The completed veneers from Fig. 70.1. Note the invisible gingival finish line, even though the margin has not been carried below tissue.

subgingival margins

- From a periodontal viewpoint, both supragingival and equigingival margins are well tolerated.
- The greatest biologic risk occurs when placing *subgingival margins.*

  Why?

  - These margins are not as accessible as supragingival or equigingival margins for finishing procedures.
  - In addition, if the margin is placed too far below the gingival tissue crest, it violates the gingival attachment apparatus.

Restorations may need to be extended gingivally:

- (1) to create adequate resistance and retentive form in the preparation,
- (2) to make significant contour alterations because of caries or other tooth deficiencies,
- (3) to mask the tooth–restoration interface by locating it subgingivaly, or
- (4) to lengthen the tooth for aesthetic reasons.

biologic width

- The dimension of space that the healthy gingival tissues occupy between the base of the sulcus and the underlying alveolar bone is composed of the junctional epithelial attachment and the connective tissue attachment. The combined attachment width is now identified as t*he biologic width.*
  - In the average human, the connective tissue attachment occupies 1.07 mm of space above the crest of the alveolar bone
  - The junctional epithelial attachment below the base of the gingival sulcus occupies another 0.97 mm of space above the connective tissue attachment.
  - The combination of these two measurements, averaging approximately 1 mm each, constitutes the biologic width.
- Clinically, this information is applied to diagnose biologic width violations when the restoration margin is placed 2 mm or less away from the alveolar bone and the gingival tissues are inflamed with no other etiologic factors evident.

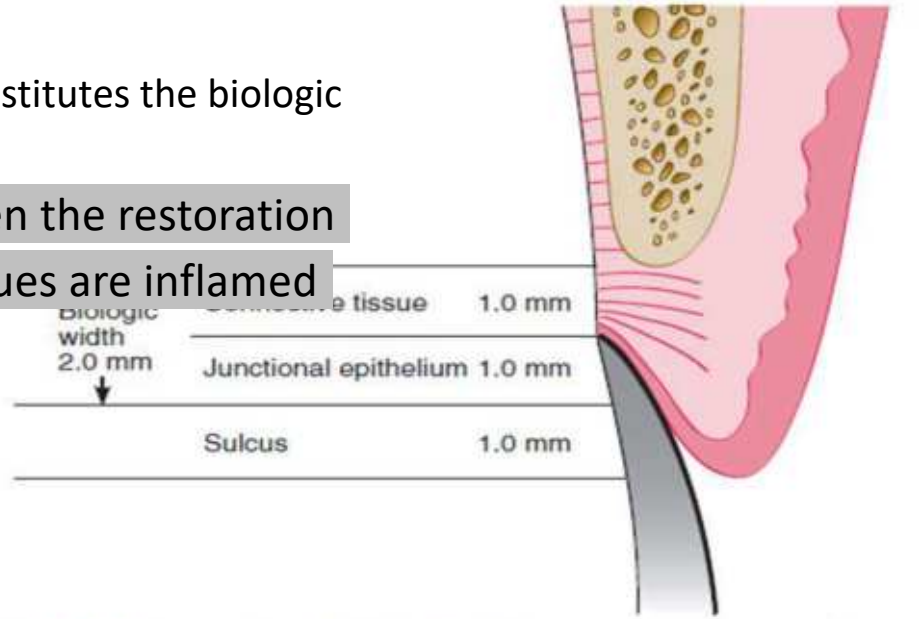
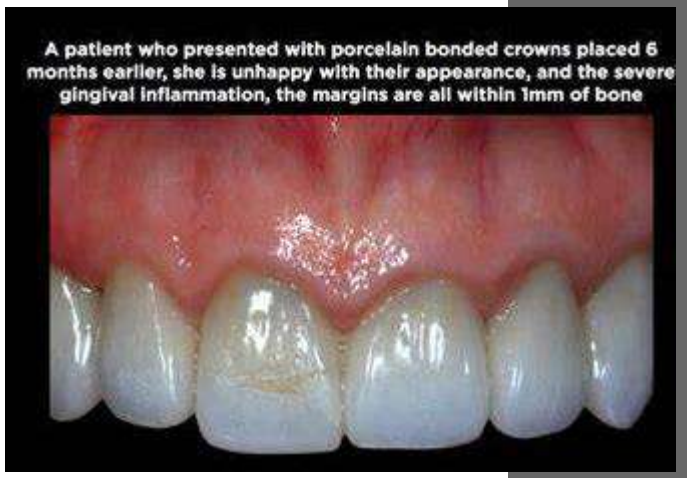
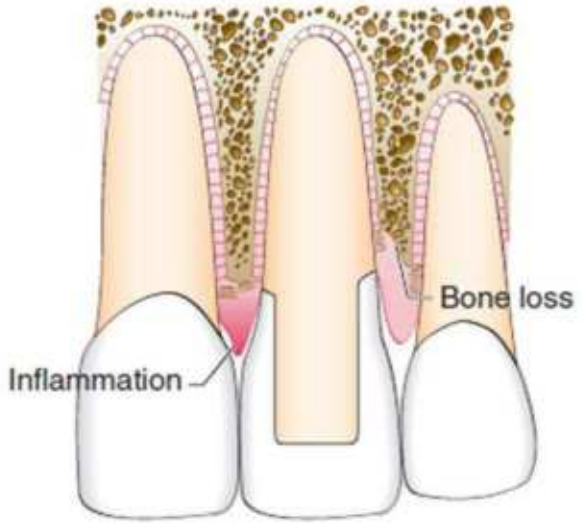


Fig. 70.3 Average human biologic width: connective tissue attachment 1 mm in height; junctional epithelial attachment 1 mm in height; sulcus depth of approximately 1 mm. The combined connective tissue attachment and junctional epithelial attachment, or biologic width, equals 2 mm.

violation of the biologic width

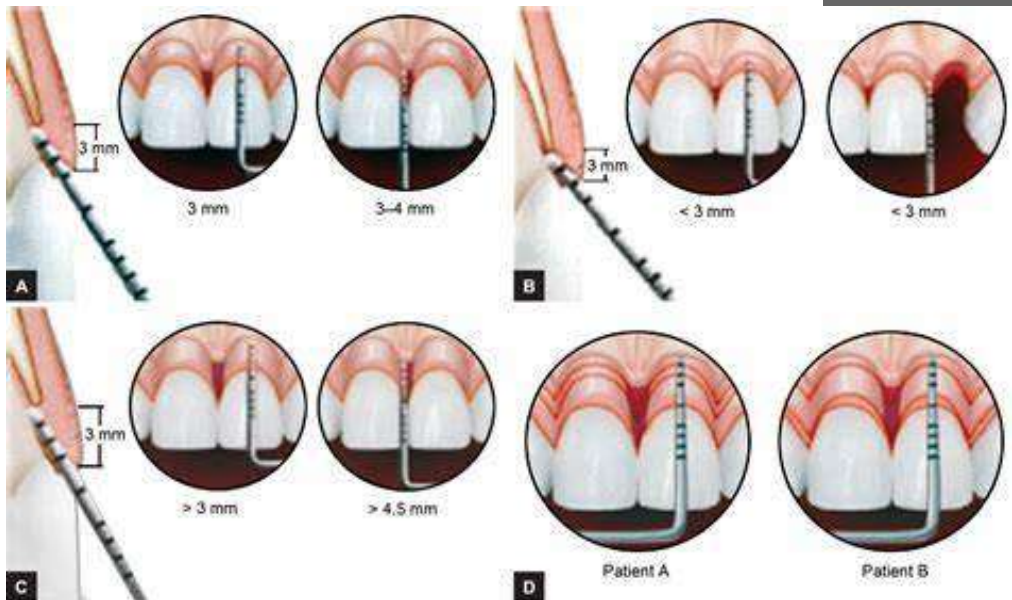


- When the restoration margin is placed too far below the gingival tissue crest, it impinges on the gingival attachment apparatus and creates a violation of biologic width.
- Two different responses can be observed from the involved gingival tissues.
  A. One possibility is that bone loss of an unpredictable nature and gingival tissue recession occurs as the body attempts to re-create room between the alveolar bone and the margin to allow space for tissue reattachment.
  B. Inflammation of the gingiva
- Factors that may impact the likelihood of recession include:
  I. The alveolar bone surrounding the tooth is very thin in width.
  II. whether the gingiva is thin and fragile
  III. whether the periodontium is highly scalloped



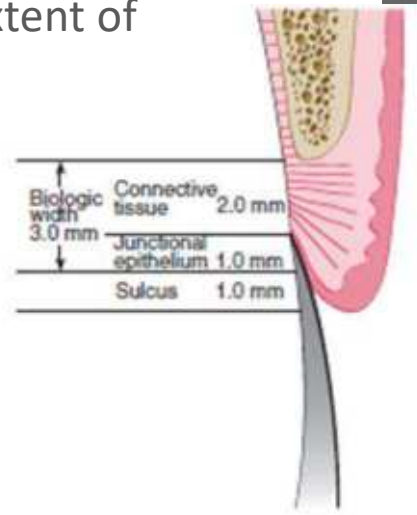
Biologic width evaluation

- **Radiographic** interpretation can identify interproximal violations of biologic width.
  - superimposition.
- **Clinical evaluation**
- If a patient experiences tissue discomfort when the restoration margin levels are being assessed with a periodontal probe, it is a good indication that the margin extends into the attachment and that a biologic width violation has occurred.
- A more positive assessment can be made clinically by measuring the distance between the bone and the restoration margin using a sterile periodontal probe. The probe is pushed through the anesthetized attachment tissues from the sulcus to the underlying bone. If this distance is less than 2 mm at one or more locations, a diagnosis of biologic width violation can be confirmed.
- Circumferentially around the tooth to evaluate the extent of the problem.



Normal variation

- Biologic width violations can occur in some patients in whom the margins are located more than 2 mm above the alveolar bone level.
- A range of different biologic width was reported; 0.75 mm to 4.3 mm.
- This information dictates that specific biologic width assessment should be performed for each patient to determine if the patient needs additional biologic width, in excess of 2 mm, for restorations to be in harmony with the gingival tissues.
- The biologic width can be identified for the individual patient by probing to the bone level (referred to as “sounding to bone”) and subtracting the sulcus depth from the resulting measurement.
- This measurement must be done on teeth with healthy gingival tissues and should be repeated on more than one tooth to ensure an accurate assessment.
- The information obtained is then used for definitive diagnosis of biologic width violations, the extent of correction needed, and the parameters for placement of future restorations.

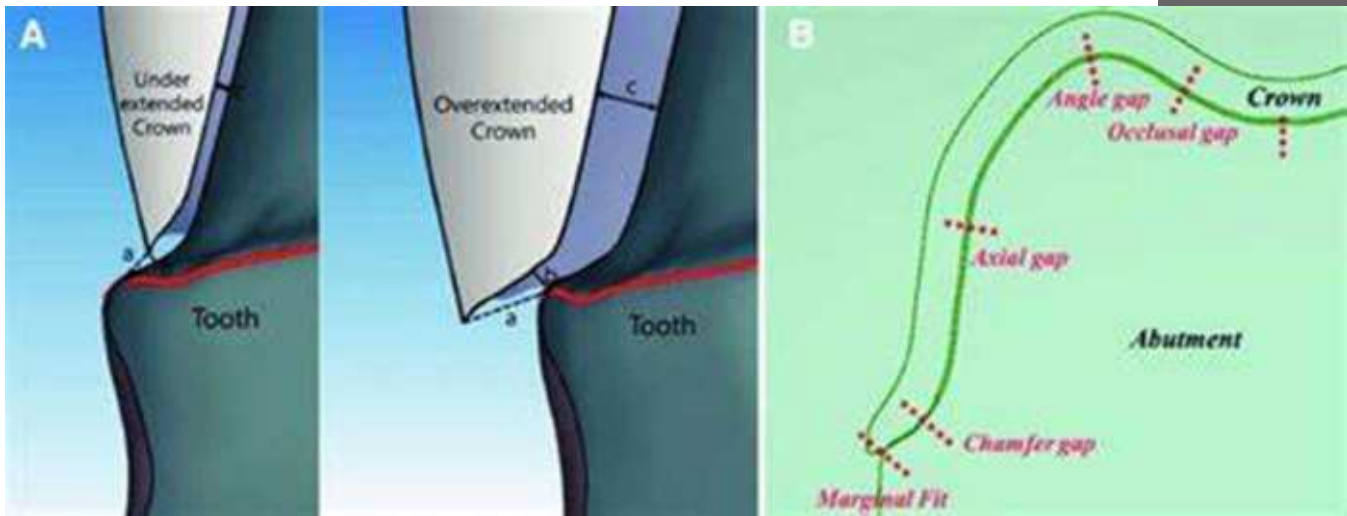


Margin Placement Guidelines

- It is recommended that the patient's existing sulcular depth be used as a guideline in assessing the biologic width requirement for that patient.
- The base of the sulcus can be viewed as the top of the attachment,
- ensuring that the margin is placed in the sulcus and not in the attachment. The variations in sulcular probing depth are then used to predict how deep the margin can safely be placed below the gingival crest.
- With shallow probing depths (1 to 1.5 mm), extending >0.5 mm subgingivally risks violating the attachment..
- With shallow probing depths, future **recession is unlikely** because the free gingival margin is located close to the top of the attachment.
- Deeper sulcular probing depths provide more freedom in locating restoration margins farther below the gingival crest. In most circumstances, however, **the deeper the gingival sulcus, the greater is the risk of gingival recession.**
- Locating the restorative margin deep subgingivally should be avoided:
  a. It increases the difficulty in making an accurate impression,
  b. finishing the restoration margins,
  c. Increases the likelihood of inflammation and recession.

Marginal Fit

- Marginal fit has clearly been implicated in producing an inflammatory response in the periodontium.
- It has been shown that the level of gingival inflammation can increase corresponding with the level of marginal opening. Margins that are significantly open (several tenths of a millimeter) are capable of harboring large numbers of bacteria and may be responsible for the inflammatory response seen.
- However, the quality of **marginal finish and the margin location** relative to the attachment are much more critical to the periodontium than the difference between a 20-µm fit and a 100-µm fit.



Crown Contour

- Restoration contour has been described as extremely important to the maintenance of periodontal health.
- Ideal contour provides access for hygiene, has the fullness to create the desired gingival form, and has a pleasing visual tooth contour in aesthetic areas.(Describe ideal crown contour?)
- overcontouring crowns leads to and gingival inflammation, whereas undercontouring produces no adverse periodontal effect.
- The most frequent cause of overcontoured restorations is inadequate tooth preparation by the dentist, which forces the technician to produce a bulky restoration to provide room for the restorative material.
- In areas of the mouth in which aesthetic considerations are not critical, a flatter contour is always acceptable.

Aesthetic Tissue Management

Managing Interproximal Embrasures

- Current restorative and periodontal therapy must consider a good aesthetic result, especially in the “aesthetic zone.” the interproximal papilla is an important part in creating this aesthetic result.
- The interproximal embrasure created by restorations and the form of the interdental papilla have a unique and intimate relationship.
- **The ideal interproximal embrasure** should house the gingival papilla without impinging on it and should also extend the interproximal tooth contact to the top of the papilla so that no excess space exists to trap food and to be aesthetically displeasing.
- **Papillary height** is established by the level of the bone, the biologic width, and the form of the gingival embrasure. Changes in the shape of the embrasure can impact the height and form of the papilla. The tip of the papilla behaves differently than the free gingival margin on the facial aspect of the tooth. Whereas the **free gingival margin averages 3 mm** above the underlying facial bone, **the tip of the papilla averages 4.5 to 5 mm** above the interproximal bone. This means that if the papilla is farther above the bone than the facial tissue but has the same biologic width, the interproximal area will have a sulcus 1 to 1.5 mm deeper than that found on the facial surface.

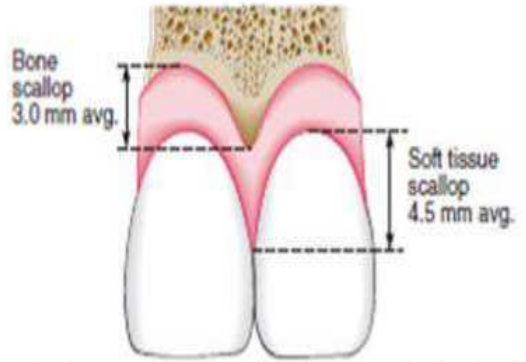


Fig. 70.6 Comparison of the behavior of the interproximal papilla relative to bone and the free gingival margin relative to bone in the average human. There is a 3-mm scallop from the facial bone to the interproximal bone. However, on average, a 4.5- to 5-mm gingival scallop exists between the facial tissue height and the interproximal papilla height. This extra scallop of 1.5 to 2 mm of gingiva compared with bone is the result of the extra soft-tissue height above the attachment interproximally.

CLINICAL CORRELATION

- If you create restorations with less than 5 mm from the contact to the bone, open gingival embrasures can be avoided. The downside to this approach is that the teeth will look square and blocky.
- Complete removal of healthy papillae to the bone level can routinely regenerated 4 to 4.5 mm of total tissue above bone, with an average sulcus depth of 2 to 2.5 mm.

Chance of complete papilla fill

- When the distance from the interproximal bone to the interproximal contact of the teeth measured 5 mm or less, 98% of these sites had complete papilla fill.
- When the distance was 6 mm, only 56% of the sites had complete papilla fill.
- When the distance was 7 mm, only 27% of the sites had complete papilla fill.

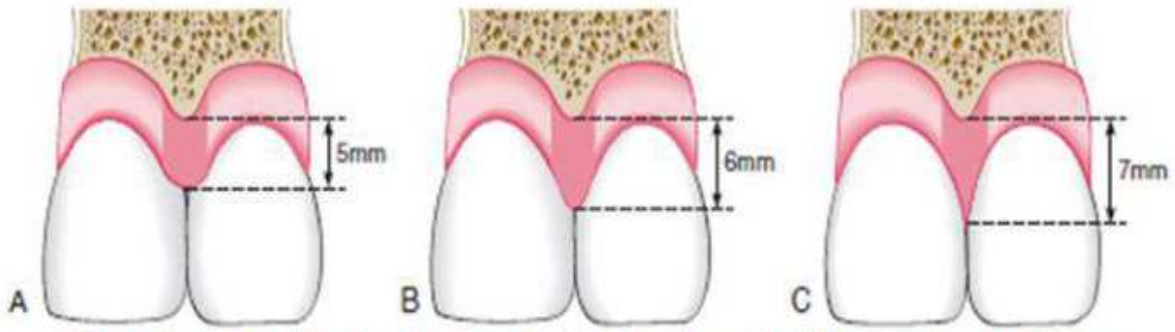


Fig. 70.7 The probability of complete fill of gingival embrasure by papilla. (A) With 5 mm from crest of bone to the apical contact point, there is a 98% chance of complete fill of the space. (B) At 6 mm from crest to contact, the chance of filled embrasure drops to 56%. (C) At 7 mm from crest to contact, the chance of complete fill drops to 27%.

Assessing open embrasure

- it is imperative that tissue be healthy and mature. Performing the analysis on inflamed or immature tissues will result in supragingival margins when the tissues heal.
- If the papillary sulcus measures greater than 3 mm, there is some risk of recession with restorative procedures.

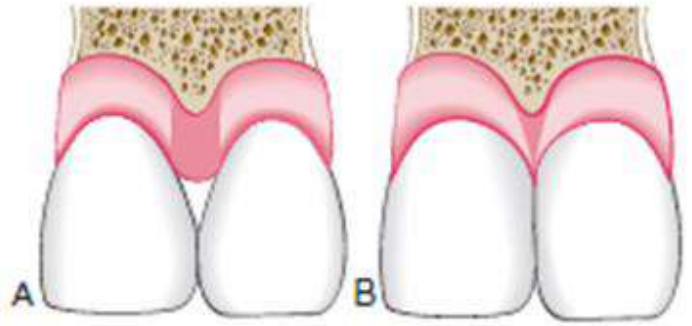
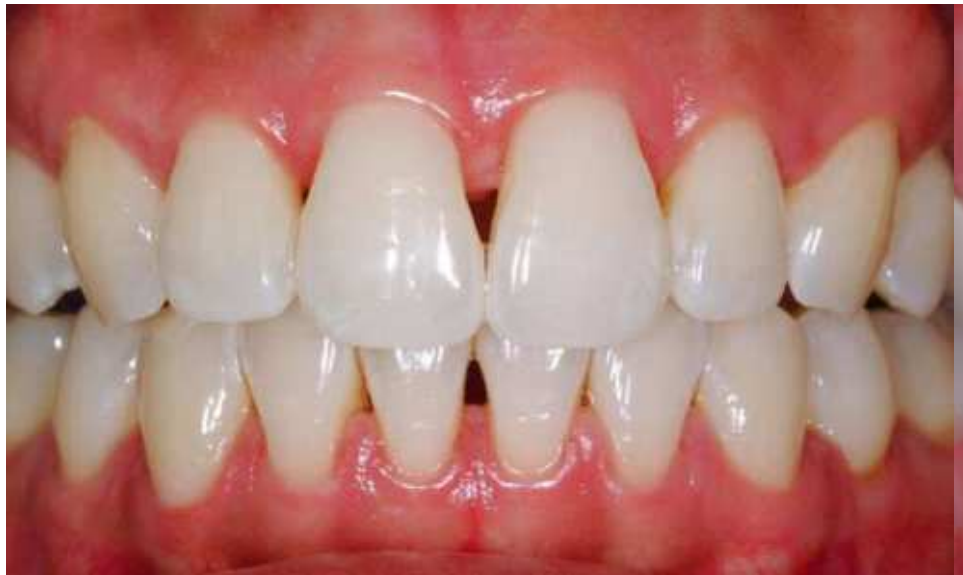


Fig. 70.8 Relationship between gingival embrasure volume and papillary form. (A) Gingival embrasure of the teeth is excessively large as the result of a tapered tooth form. Because of the large embrasure form, the volume of tissue sitting on top of the attachment is not molded to the shape of a normal papilla but rather has a blunted form and a shallower sulcus. (B) Ideal tooth form in which the same volume of tissue sits on top of the attachment as in part A. Because of the more closed embrasure form from the teeth in part B, however, the papilla completely fills the embrasure and has a deeper sulcus, averaging 2.5 to 3 mm. Note that the ideal contact position is 3 mm coronal to the attachment.

- The papilla in question is compared with the adjacent papillae. If the papillae are all on the same level, and if the other areas do not have open embrasures, the problem is one of gingival embrasure form. Restorative correction
- If the papilla in the area of concern is apical to the adjacent papillae, however, the clinician should evaluate the interproximal bone levels.
  - If the bone under that papilla is apical to the adjacent bone levels, the problem is caused by bone loss. Periodontal treatment
  - If the bone is at the same level, the open embrasure is caused by the embrasure form of the teeth and not a periodontal problem with the papilla. Restorative correction



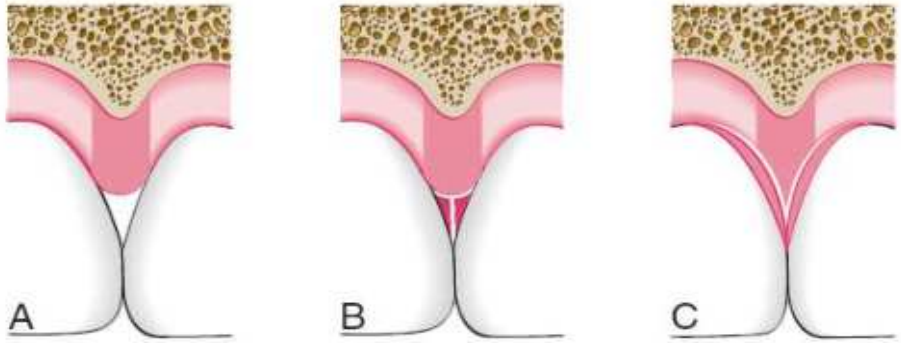
Open Gingival Embrasures

- This open space is usually caused by:
- (1) tooth shape,
- (2) root angulation, or
- (3) periodontal bone loss.
- The first step in the diagnosis of this problem is to evaluate a periapical radiograph of the central incisors. Correction of open gingival embrasure can be in different ways. Periodontal surgery, orthodontic treatment or restorations.

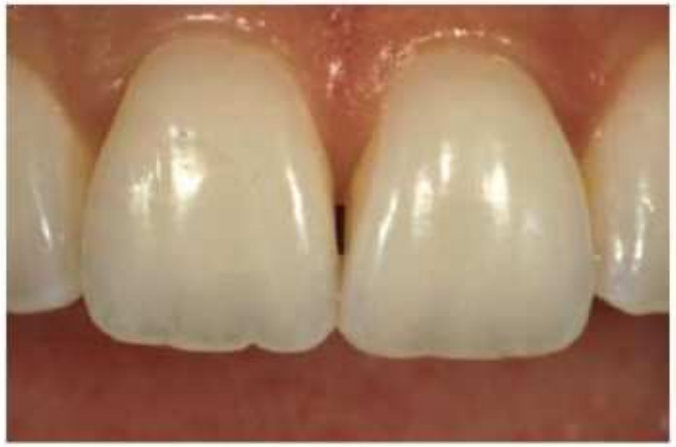
Correcting Open Gingival Embrasures **Restoratively**

Restorative dentistry can correct this problem by moving the contact point to the tip of the papilla. To accomplish this, the margins of the restoration must be carried subgingivally 1 to 1.5 mm, and the emergence profile of the restoration is designed to move the contact point toward the papilla while blending the contour into the tooth below the tissue.

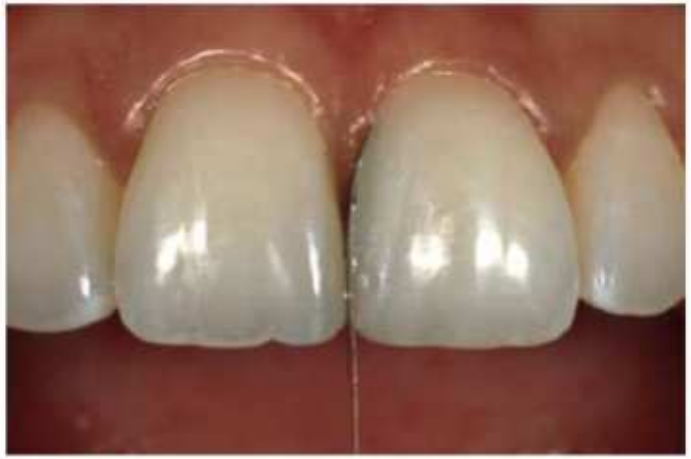
This can be accomplished easily with direct bonded restorations



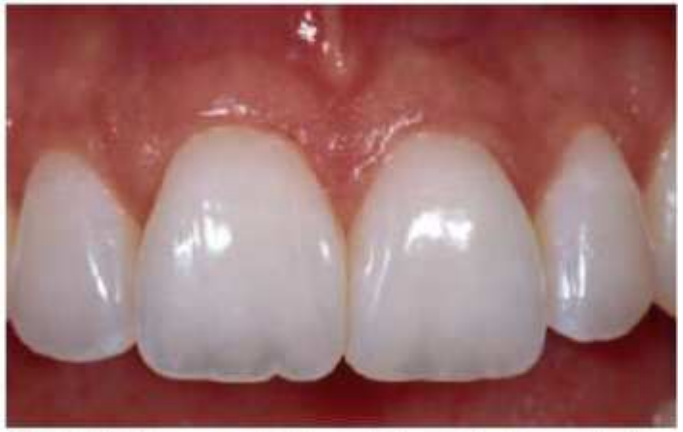
eFig. 70.26 Methods of altering gingival embrasure form. (A) Typical open gingival embrasure caused by excessively tapered tooth form. (B) Common method employed by restorative dentists to correct the embrasure, in which material is added supragingivally. This closes the embrasure by moving the contact to the tip of the papilla but results in overhangs that cannot be cleaned using dental floss. Removing these overhangs restoratively reopens the embrasure. (C) Correct method of closing the gingival embrasure, in which the margins of the restoration are carried 1 to 1.5 mm below the tip of the papilla. Note that this does not encroach on the attachment because the average interproximal sulcus probes 2.5 to 3 mm. This allows easy cleaning because of the convex profile. It also reshapes the papilla to a more pleasing profile aesthetically.



eFig. 70.27 This patient has parallel roots, has recently completed orthodontic therapy, and is unhappy with the open gingival embrasure between her central incisors. An evaluation of papillary height reveals that all are at an equal level. This can only mean that the open embrasure is the result of an overly tapered tooth form (see eFigs. 70.28 and 70.29).



eFig. 70.28 One method of correctly altering tooth form of the patient in eFig. 70.27. A metal matrix band has been shaped to the desired tooth form and placed 1 to 1.5 mm below the tip of the papilla. Restorative material then was added to the tooth against the matrix band, forming the new mesial surface of the left central incisor.



eFig. 70.29 One-year recall photograph after restoring the mesial surfaces of the right and left central incisors, moving the proximal contact to the tip of the papilla and extending the restorations 1 to 1.5 mm below the papilla, blending them into the tooth and making an easily cleaned area (see eFigs. 70.27 and 70.28).

Pontic Design

Classically, there are four options to consider in evaluating pontic design: hygienic, ridge lap, modified ridge lap, and ovate designs.

Regardless of design, the pontic should provide:

I. An occlusal surface that stabilizes the opposing teeth,

II. Allows for normal mastication

III. Does not overload the abutment teeth.

The area of the pontic interfacing with the gingiva can be porcelain, metal, zirconia, lithium disilicate, or some other material with no variation in the biologic response of the tissue provided it has a smooth surface finish.

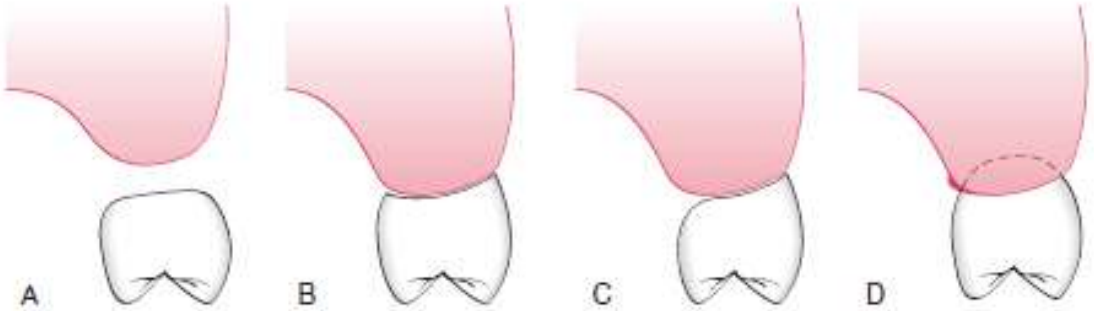


Fig. 70.9 Four options to designing the shape of a pontic. (A) Hygienic pontic. Tissue surface of the pontic is 3 mm from the underlying ridge. (B) Ridge-lap pontic. Tissue surface of the pontic straddles the ridge in saddle-like fashion. The entire tissue surface of the ridge-lap pontic is convex and very difficult to clean. (C) Modified ridge-lap pontic. Tissue surface on the facial is concave, following the ridge. However, the lingual saddle has been removed to allow access for oral hygiene. (D) Ovate pontic. The pontic form fits into a receptor site within the ridge. This allows the tissue surface of the pontic to be convex and also optimizes aesthetics.

- The key differences between the four pontic designs relate to the aesthetics and access for hygiene procedures.
- The primary method for cleaning the undersurface of pontics is to draw dental loss mesiodistally along the undersurface. The shape of this undersurface determines the ease with which plaque and food debris can be removed in the process. The hygienic and ovate pontics have convex undersurfaces, which makes them easiest to clean. The ridge lap and modified ridge-lap designs have concave surfaces, which are more difficult to access with the dental floss. though the hygienic pontic design provides the easiest access for hygiene procedures, it is much less aesthetic and objectionable by some patients.

The ovate pontic

- is the ideal pontic form, particularly in areas of aesthetic concern. It is created by forming a receptor site in the edentulous ridge with a diamond bur, electrosurgery, pressure, or wound healing.
- The site is shaped to create either a flat or a concave contour so that when the pontic is created to adapt to the site, it will have a flat or convex outline. The depth of the receptor site depends on the aesthetic requirements of the pontic.

- In highly aesthetic areas such as the maxillary anterior region, it is necessary to create a receptor area that is 1 to 1.5 mm below the tissue on the facial aspect.
- This creates the appearance of a free gingival margin and produces optimal aesthetics. This site can then be tapered to the height of the palatal tissue to facilitate hygiene access from the palatal side.
- In the posterior areas, a deep receptor site can complicate hygiene access. In these situations, the ideal site has the facial portion of the pontic at the same level as the ridge, and then the site is created as a straight line to the lingual side of the pontic.
- This removes the convexity of the ridge and produces a flat, easily cleanable tissue surface on the pontic

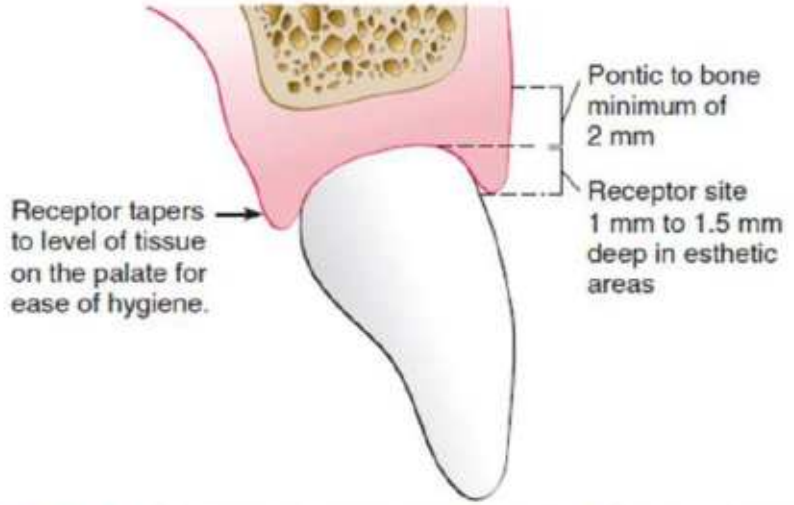


Fig. 70.10 Ideal shape and form of an ovate pontic in the aesthetic area. The receptor site has been created 1 to 1.5 mm apical to the free gingival margin on the facial aspect. This creates the illusion of the pontic erupting from the tissue. On the palatal side, the pontic is tapered so that the receptor site is not extended below tissue; this allows easier access for oral hygiene. Note that when the receptor site is created, the bone must be a minimum of 2 mm from the most apical portion of the pontic.

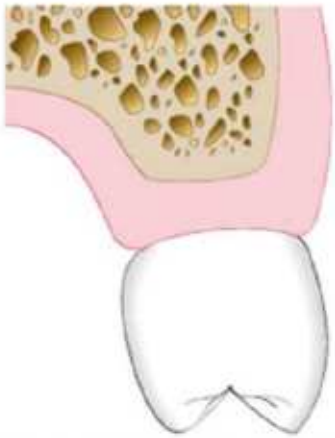
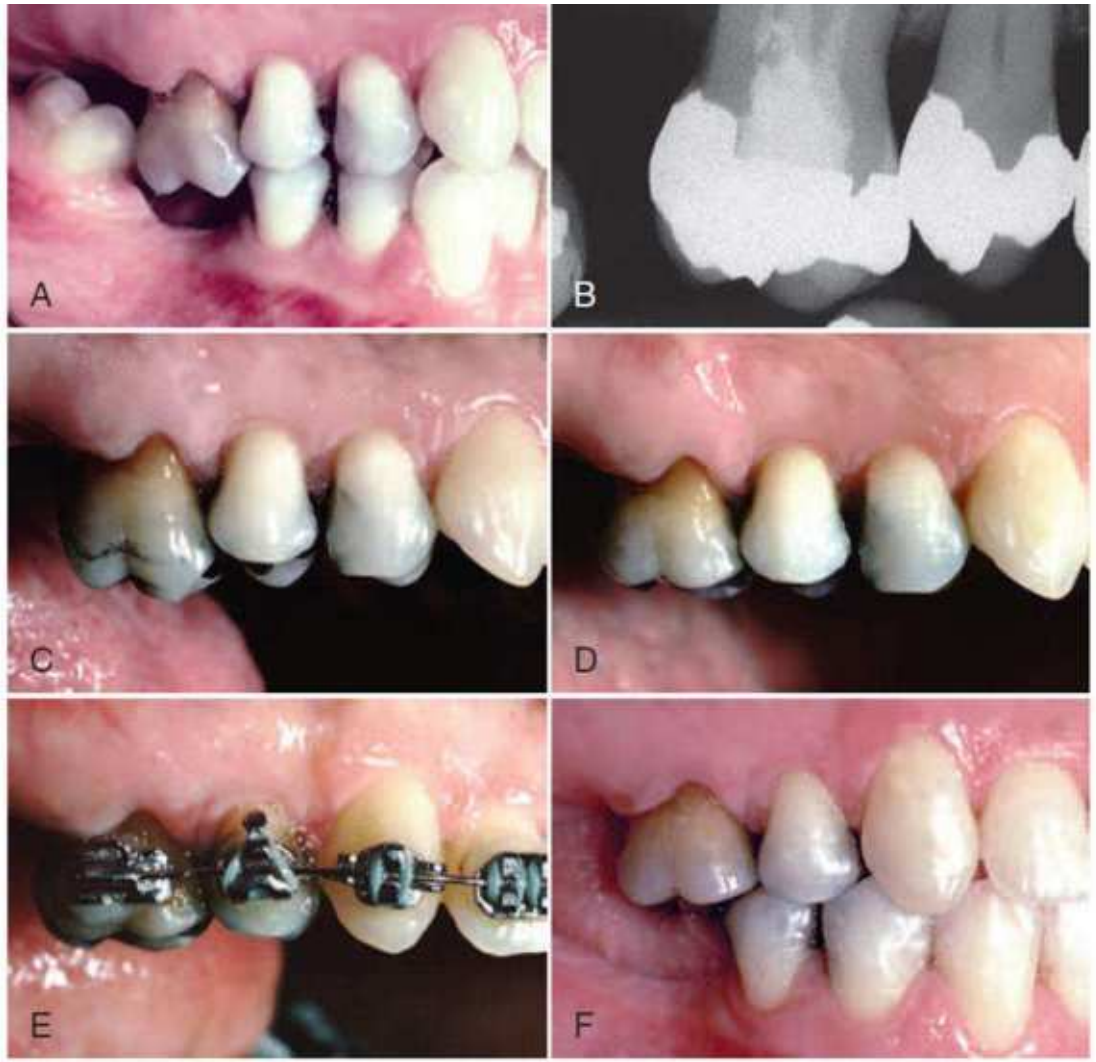


Fig. 70.11 Option for creating an ovate pontic receptor site in less aesthetic areas of the mouth. Rather than creating the receptor site so that the pontic extends into the ridge, it is possible to create a flattened receptor site in which the pontic sits flush with the ridge. This facilitates oral hygiene.

Periodontal-orthodontic interrelationship

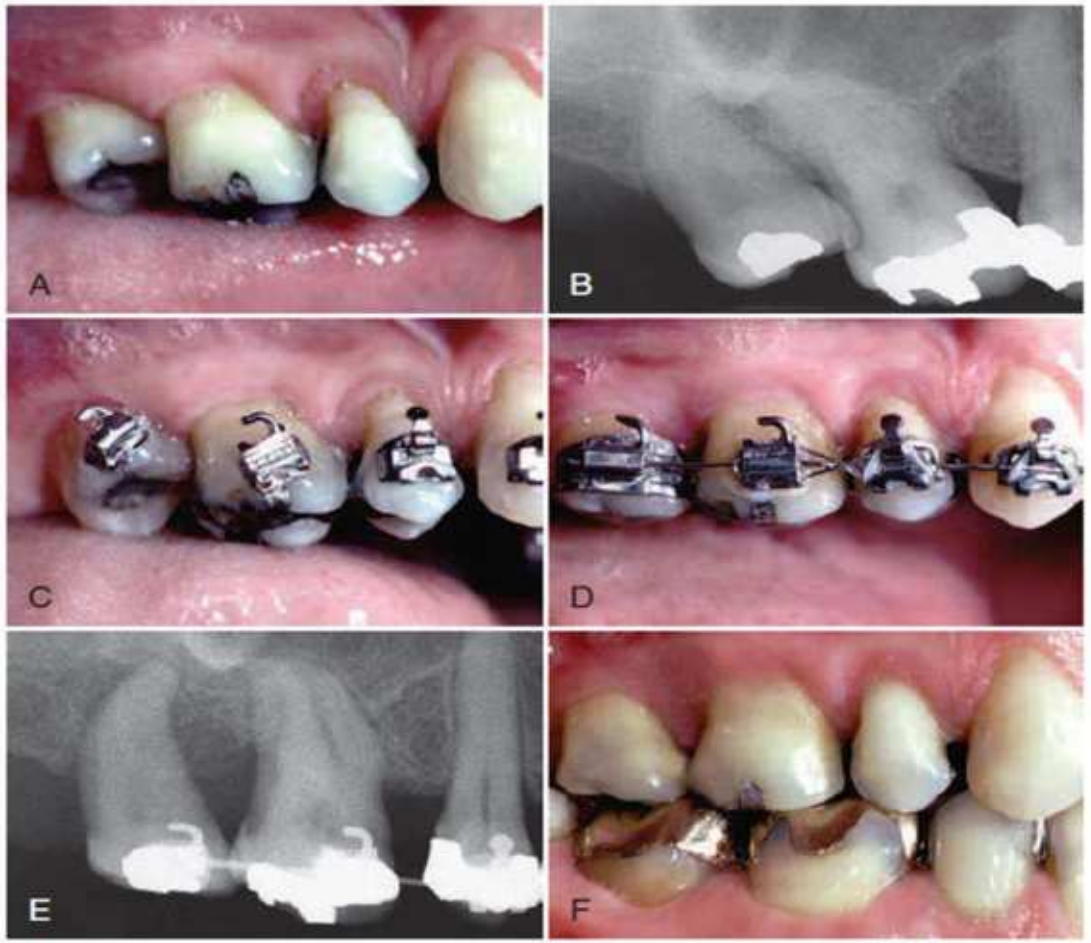
Orthodontic tooth movement in adults with periodontal tissue breakdown

- In the periodontally healthy patient, orthodontic brackets are positioned on the posterior teeth relative to the marginal ridges and cusps. However, some adult patients may have marginal ridge discrepancies caused by uneven tooth eruption. When marginal ridge discrepancies are encountered, the decision as to where to place the bracket or band is not determined by the anatomy of the tooth. In these patients, it is important to assess these teeth radiographically to determine the interproximal bone level.
- If the bone level is oriented in the same direction as the marginal ridge discrepancy, leveling of the marginal ridges will level the bone.
- If the bone level is flat between adjacent teeth and the marginal ridges are at significantly different levels, orthodontic correction of the marginal ridge discrepancy produces a hemiseptal defect in the bone. This could cause a periodontal pocket between the two teeth.
- If the bone is flat and a marginal ridge discrepancy is present, the orthodontist should not level the marginal ridges orthodontically.
- In these situations, it may be necessary to equilibrate the crown of the tooth.



eFig. 56A.6 This patient showed overeruption of the maxillary right first molar and a marginal ridge defect between the second premolar and first molar (A). The pretreatment periapical radiograph (B) showed that the interproximal bone was flat. To avoid creating a hemiseptal defect, the occlusal surface of the first molar was equilibrated (C and D), and the malocclusion was corrected orthodontically (E and F).

- For some patients, the equilebrated technique may require endodontic therapy and restoration of the tooth because of the required amount of reduction of the length of the crown. This approach is acceptable if the treatment results in a more favorable bone contour between the teeth.
- Some patients have discrepancies between both the marginal ridges and the bony levels between two teeth. However, these discrepancies may not be of equal magnitude; orthodontic leveling of the bone may still leave a discrepancy in the marginal ridges.
- In these patients, the crowns of the teeth should not be used as a guide for completing orthodontic therapy. The bone should be leveled orthodontically, and any remaining discrepancy between the marginal ridges should be equilibrated. This method produces the best occlusal result and improves the patient's periodontal health.



eFig. 56A.2 Before orthodontic treatment, this patient had significant mesial tipping of the maxillary right first and second molars, causing marginal ridge discrepancies and deep periodontal pockets (A). The tipping produced root proximity between the molars as well as a disruption of the normal gingival anatomy (B). To eliminate the root proximity, the brackets were placed perpendicular to the long axes of the teeth (C). This method of bracket placement facilitated root alignment and elimination of the root proximity, as well as leveling of the marginal ridge discrepancies (D through F).

Orthodontic treatment considerations for periodontal patients:

- Orthodontic therapy can provide several benefits to adult periodontal patients. The following seven factors should be considered:

1. Aligning crowded or malpositioned maxillary or mandibular anterior teeth permits adult patients better access to clean all surfaces of their teeth.
2. Vertical orthodontic tooth repositioning can improve certain types of osseous defects in periodontal patients. Often, moving the tooth eliminates the need for resective osseous surgery.
3. Orthodontic treatment can improve the aesthetic relationship of the maxillary gingival margins before restorative dentistry. Avoids gingival recontouring, entail bone removal and exposure of the roots of the teeth
4. Orthodontic treatment allows open gingival embrasures to be corrected to regain lost papillae.
5. Orthodontic treatment could improve adjacent tooth positioning before implant placement or tooth replacement. Drifting and tipping of the adjacent dentition.
6. A common tooth malalignment problem that results in periodontal pockets is the mesially tipped molar. Can be corrected orthodontically.

Periodontal surgery associated with ortho

- The extent of periodontal osseous surgery depends on the type of defect

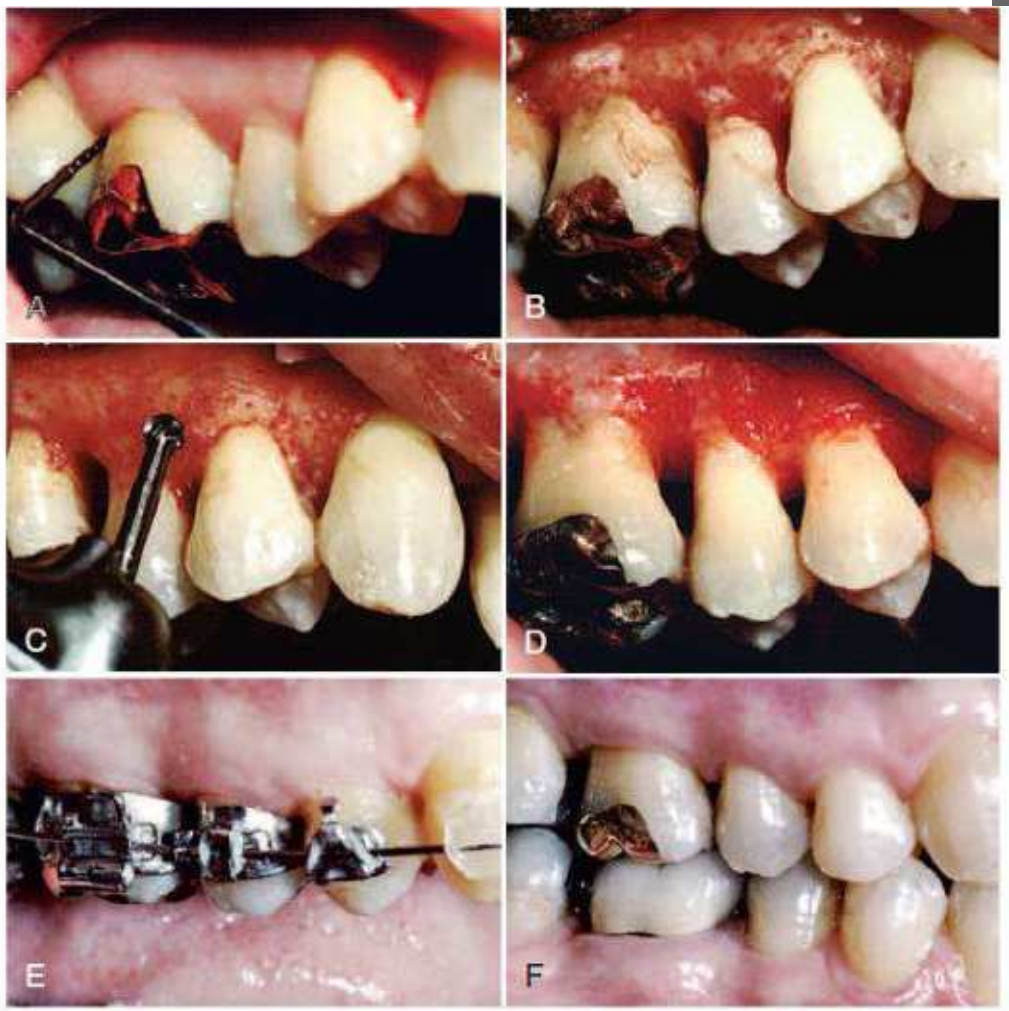
Osseous Craters

- An osseous crater is an interproximal, two-wall defect that does not improve with orthodontic treatment. Some shallow craters (4 to 5 mm pocket) may be maintained nonsurgically durin orthodontic treatment. However, if surgical correction is necessary, this type of osseous lesi can easily be eliminated by reshaping the defec and reducing the pocket depth. This in turn enhances the ability to maintain these interproximal areas during orthodontic treatment. The need for surgery is based on

I. The patient's response to initial root planing

II. The location of the defect,

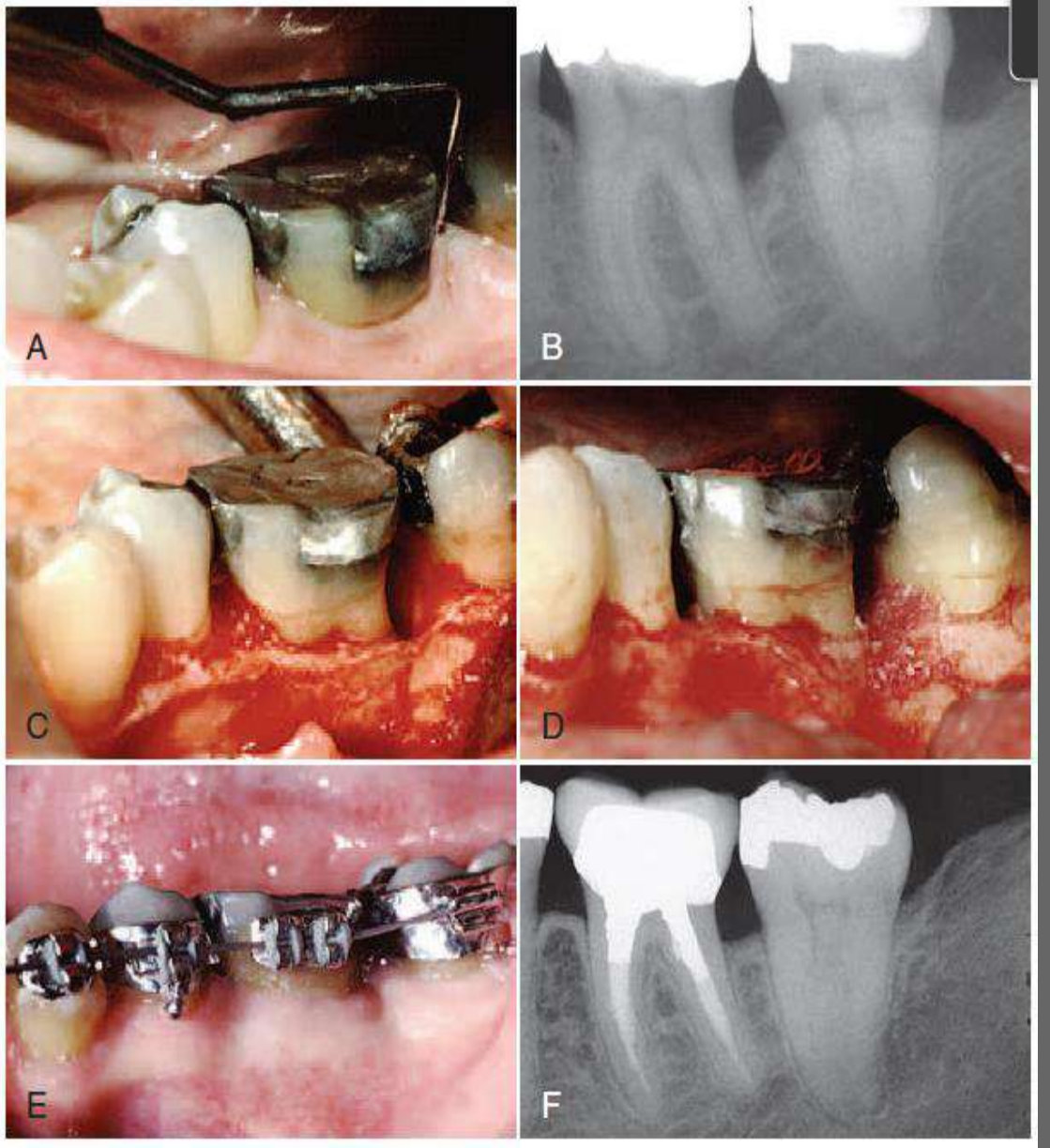
III. The predictability of maintaining defects nonsurgically, while the patient is wearing orthodontic appliances.



eFig. 56A.3 This patient had a 6-mm probing defect distal to the maxillary right first molar (A). When this area was flapped (B), a cratering defect was apparent. Osseous surgery was performed to alter the bony architecture of the buccal and lingual surfaces to eliminate the defect (C and D). After 6 weeks, the probing pocket defect had been reduced to 3 mm and orthodontic appliances were placed on the teeth (E). Because the crater was eliminated before orthodontic therapy, the patient could maintain the area during and after orthodontic treatment (F).

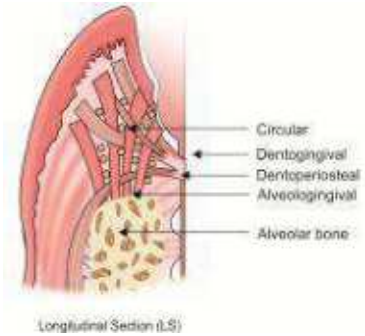
Three-Wall Intrabony Defects

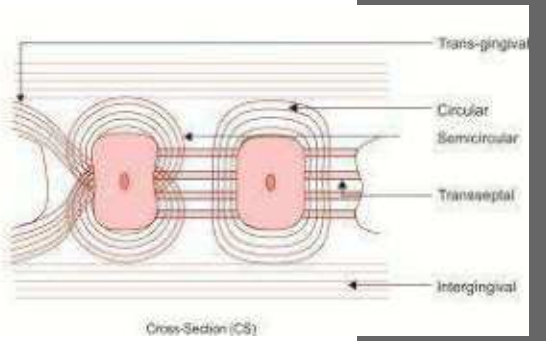
- Three-wall osseous defects are amenable to pocket reduction with regenerative periodontal therapy. Bone grafts using either autogenous bone from the surgical site or allografts, along with resorbable membranes, have been successful in regenerating three-wall defects.
- If the result is stable 3 to 6 months after periodontal surgery, orthodontic treatment may be initiated.



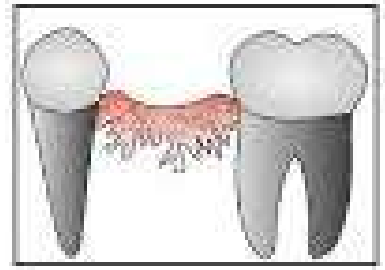
eFig. 56A.4 This patient had a significant periodontal pocket (A) distal to the mandibular right first molar. Periapical radiograph (B) confirmed the osseous defect. A flap was elevated (C), revealing a deep, three-wall osseous defect. Freeze-dried bone (D) was placed in the defect. Six months after the bone graft, orthodontic treatment was initiated (E). The final periapical radiograph shows that the preorthodontic bone graft helped regenerate bone and eliminate the defect distal to the molar (F).

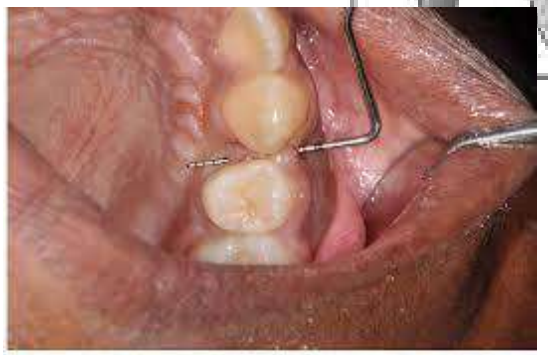
Minor surgery associated with Orthodontic treatment





- 1.Fiberotomy: trans-septal fiber. It includes non-elastics collagenous fibers, which seems to contribute to rotational relapse after orthodontic treatment.
- 2.Frenotomy: It is indicatedin case of very hyperplastic type of frenum, with a fan like attachment. It may obstruct diastema closure. Frenectomy is not favorable anymore as is considered as a destructive procedure. It might also lead to loss of interdental papilla.
- 3.Gingivectomy: it may be used to increase the clinical crown during or after ortho treatment and in case of gingival discrepancy is apparent
- 4. Removal of gingival invagination. This invagination is happen after closure of extracted spaceby ortho treatment. Sometimes thisinvagination is still in place for more than 5 years. It is advised sometimes to remove this invagination surgically.
- 5. Use of implants: in orthodontic treatment, osseointegrated implant may be used. Close cooperation between orthodontist, periodontist, and oral surgeon is important for optimal treatment planning and implant positioning.





Periodontal-Prosthodontic interrelationship

Preprosthetic Surgery

Management of Mucogingival Problems

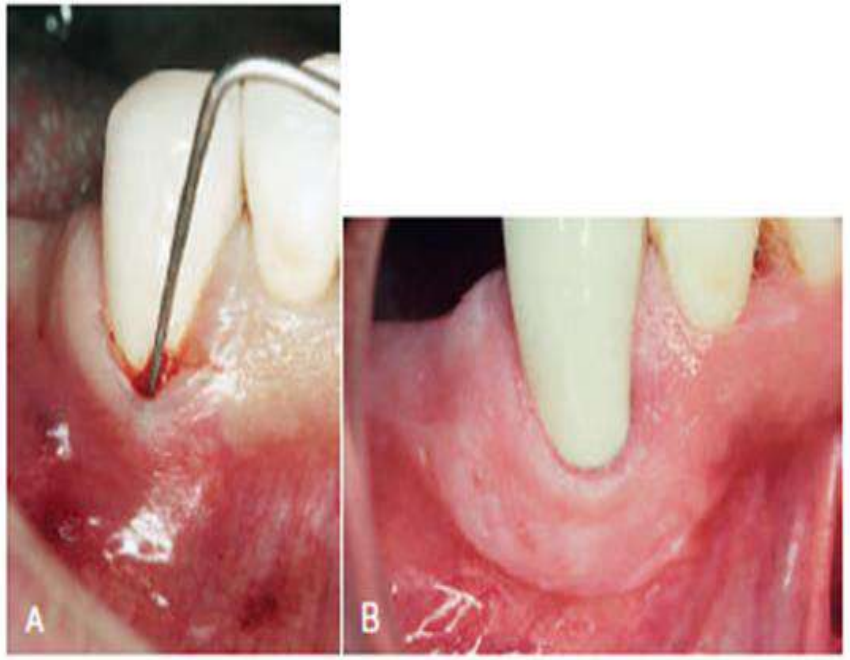
Periodontal plastic surgical procedures may be undertaken for a variety of reasons.[7] The most common techniques include those that increase the gingival dimensions and achieve root coverage. These procedures are often indicated before restoration for prosthetic reasons (eFig. 69.1) and in conjunction with orthodontic tooth movement.[34] Root coverage procedures may also be undertaken for purposes of comfort and aesthetics (eFig. 69.2).[7] At least 2 months of healing is recommended after soft tissue grafting procedures before initiating restorative dentistry[35] (see Chapter 65).

Preservation of Ridge Morphology After Tooth Extraction

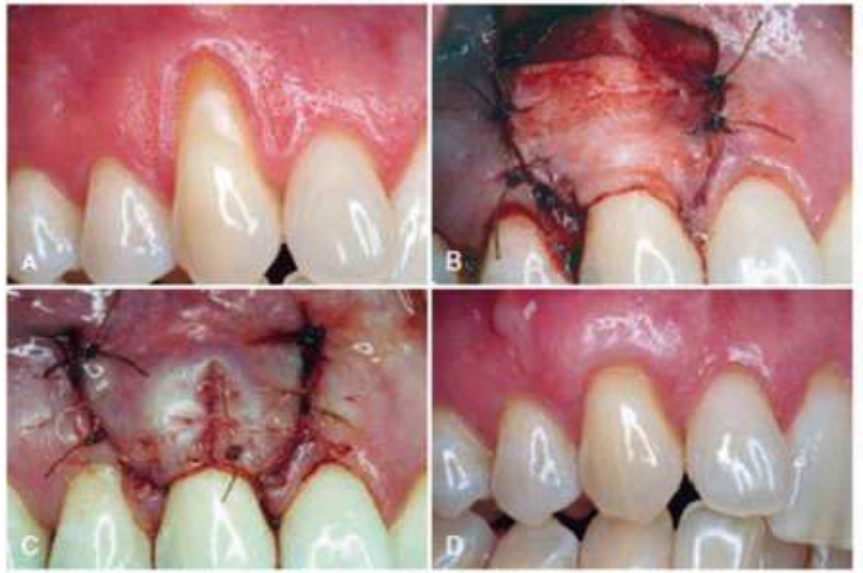
Alveolar ridge resorption is a common consequence of tooth loss.[1,2] Ridge preservation procedures have been shown to be useful in anticipation of the future placement of a dental implant or for pontics used for fixed bridges, as well as in cases where unaided healing would result in an unaesthetic deformity[16,26,27,33,36,42] (eFig. 69.3).

Crown-Lengthening Procedures

Surgical crown-lengthening procedures are performed to provide retention form to allow for proper tooth preparation, impression



eFig. 69.1 In preparation for a removable partial denture, this canine has received a gingival graft to increase attached gingiva and deepen the vestibule. (A) Before therapy. Note minimal attached gingiva. (B) After therapy, there is abundant attached gingiva and vestibular depth.



eFig. 69.2 Connective tissue graft placed under a double-papilla flap has been used to provide root coverage for a maxillary right canine. (A) Maxillary canine before therapy. (B) Connective tissue graft placed over denuded root surface. (C) Papilla placed over connective tissue. (D) Final result.

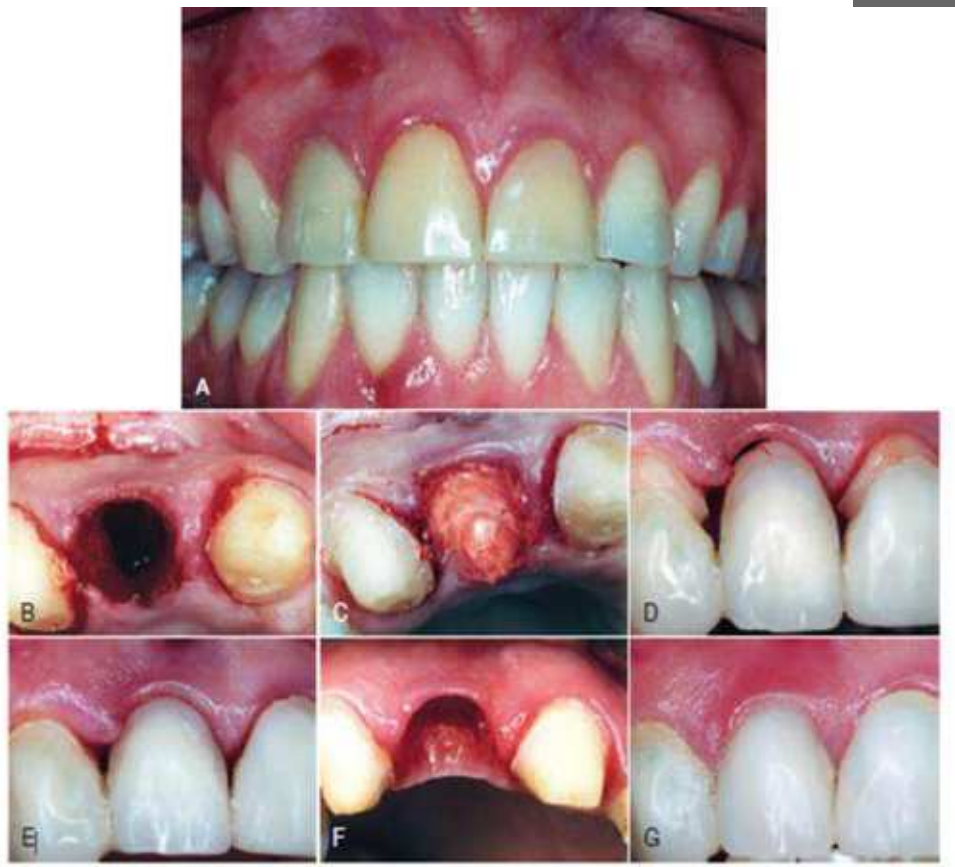
procedures,[23] and placement of restorative margins (eFig. 69.4),[23] and to adjust gingival levels for aesthetics.[35,50] It is important that crown-lengthening surgery is done in such a manner that the biologic width is preserved. The *biologic width* is defined as the physiologic dimension of the junctional epithelium and connective tissue attachment (see Chapter 70). This measurement has been found to be relatively constant at approximately 2 mm (±30%).[11] The healthy gingival sulcus has an average depth of 0.69 mm (eFig. 69.5).[21] It has been theorized that infringement on the biologic width by the placement of a margin of a restoration within its zone may result in gingival inflammation,[21] pocket formation, and alveolar bone loss[38] (eFig. 69.6). Consequently, it is recommended that there be at least 3 mm between the gingival margin and bone crest.[13,41,44,47] This allows for adequate biologic width when the restoration is placed 0.5 mm within the gingival sulcus[44,47] (eFig. 69.7).

Surgical crown lengthening may include the removal of soft tissue or both soft tissue and alveolar bone. Reduction of soft tissue alone is indicated if there is adequate attached gingiva and more than 3 mm of tissue coronal to the bone crest (eFig. 69.8). This may be accomplished by either gingivectomy or flap technique (see Chapter 60). Inadequate attached gingiva and less than 3 mm of soft tissue require a flap procedure and bone recontouring (eFig. 69.9). In the case of caries or tooth fracture, to ensure margin placement on sound tooth structure and retention form, the surgery should provide at least 4 mm from the apical extent of the caries or fracture to the bone crest (eFig. 69.10).

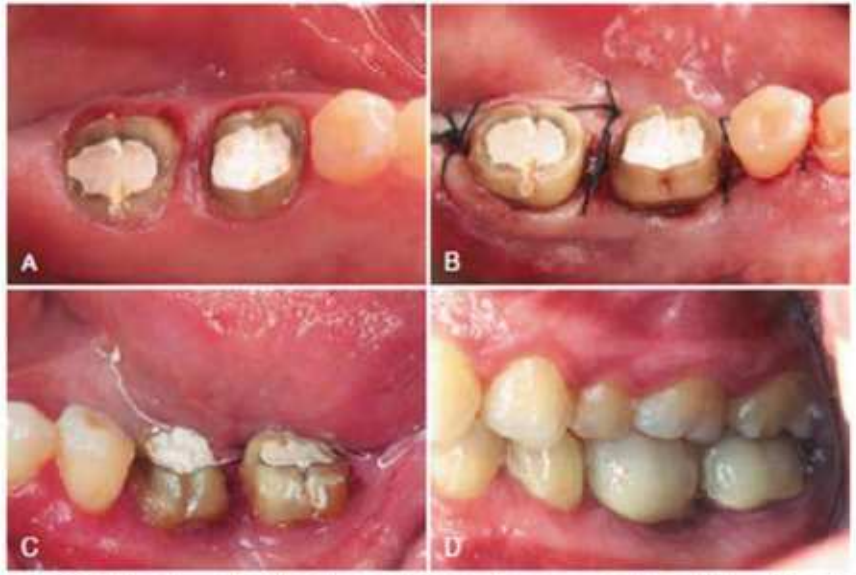
With the advent of predictable implant dentistry, it is important to carefully evaluate the value of crown lengthening for restorative therapy as opposed to tooth removal and replacement with a dental implant (eBox 69.1).

Alveolar Ridge Reconstruction

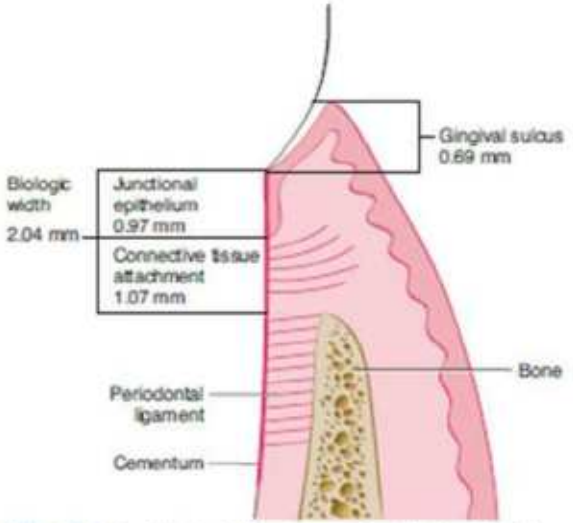
Patients are frequently seen with alveolar ridge resorption after tooth loss (see Chapter 75). To provide for adequate anatomic dimensions for the construction of an aesthetic pontic (see Chapter 70, or for a discussion on the placement of dental implants see Chapter 75), alveolar ridge reconstruction is undertaken.[42,43,45] In the case of aesthetic pontic construction, small defects may be treated with soft tissue ridge augmentation (eFig. 69.11). For larger defects and in those sites receiving dental implants, hard tissue modalities are used[43,45] (eFig. 69.12).



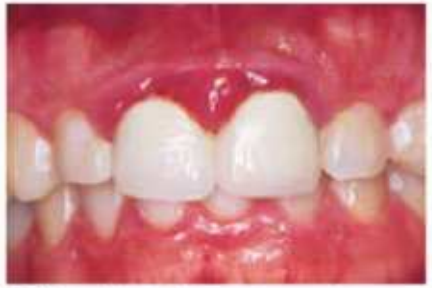
eFig. 69.3 (A) The maxillary right lateral incisor has failed endodontically, with a fistulous tract noted exiting from the attached gingiva. (B) The tooth is atraumatically removed and the socket debrided while maintaining the surrounding anatomic integrity. (C) In an effort to reduce ridge collapse, the socket is grafted with a combination of deproteinized bovine bone and calcium sulfate. (D) Provisional fixed partial denture is placed, with an ovate pontic extending 2 mm into the socket and supporting the surrounding tissues. (E–F) After 8 weeks, the socket has healed, preserving the gingival and papillary architecture, in preparation for an aesthetic final prosthesis. (G) Final restoration.



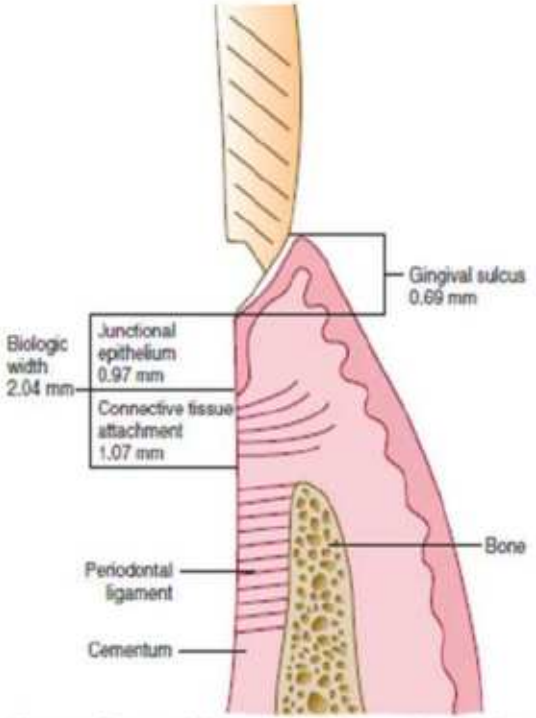
eFig. 69.4 Surgical crown lengthening has provided these otherwise unrestorable mandibular molars with improved retention and restorative access for successful restorations. (A) Before crown lengthening. (B) Crown-lengthening surgery completed. Note increased clinical crown. (C) Buccal view after surgery. (D) Final restorations.



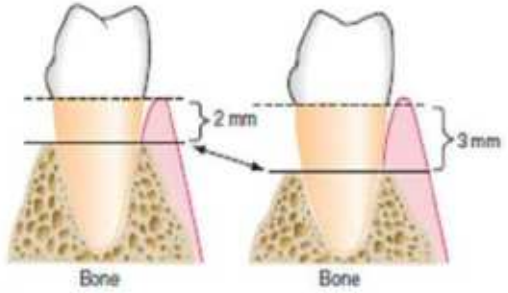
eFig. 69.5 The biologic width has been estimated to be about 2 mm. Efforts should be made to preserve its integrity.



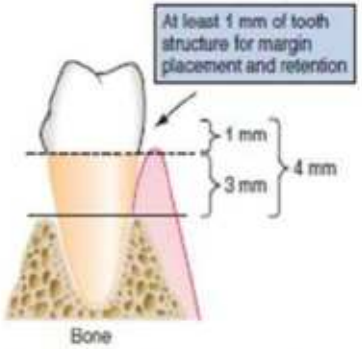
eFig. 69.6 Although gingival inflammation around crowns may have a variety of causes, infringement of biologic width must be considered.



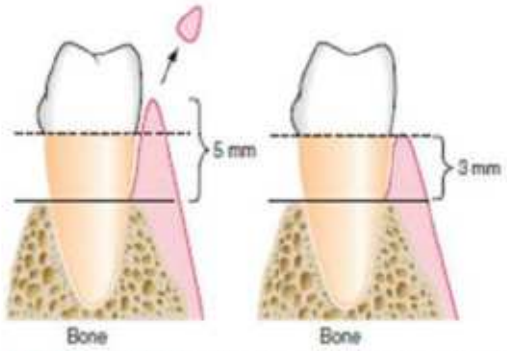
eFig. 69.7 Placement of the restorative margin 0.5 mm into the sulcus allows for the maintenance of the biologic width.



eFig. 69.9 With less than 3 mm of soft tissue between the bone and gingival margin, or less-than-adequate attached gingiva, a flap procedure and osseous recontouring are required for crown lengthening.



eFig. 69.10 In the case of caries or fracture, at least 1 mm of sound tooth structure should be provided above the gingival margin for proper restoration.



eFig. 69.8 Greater than 3 mm of soft tissue between the bone and gingival margin, with adequate attached gingiva, allows crown lengthening by gingivectomy.

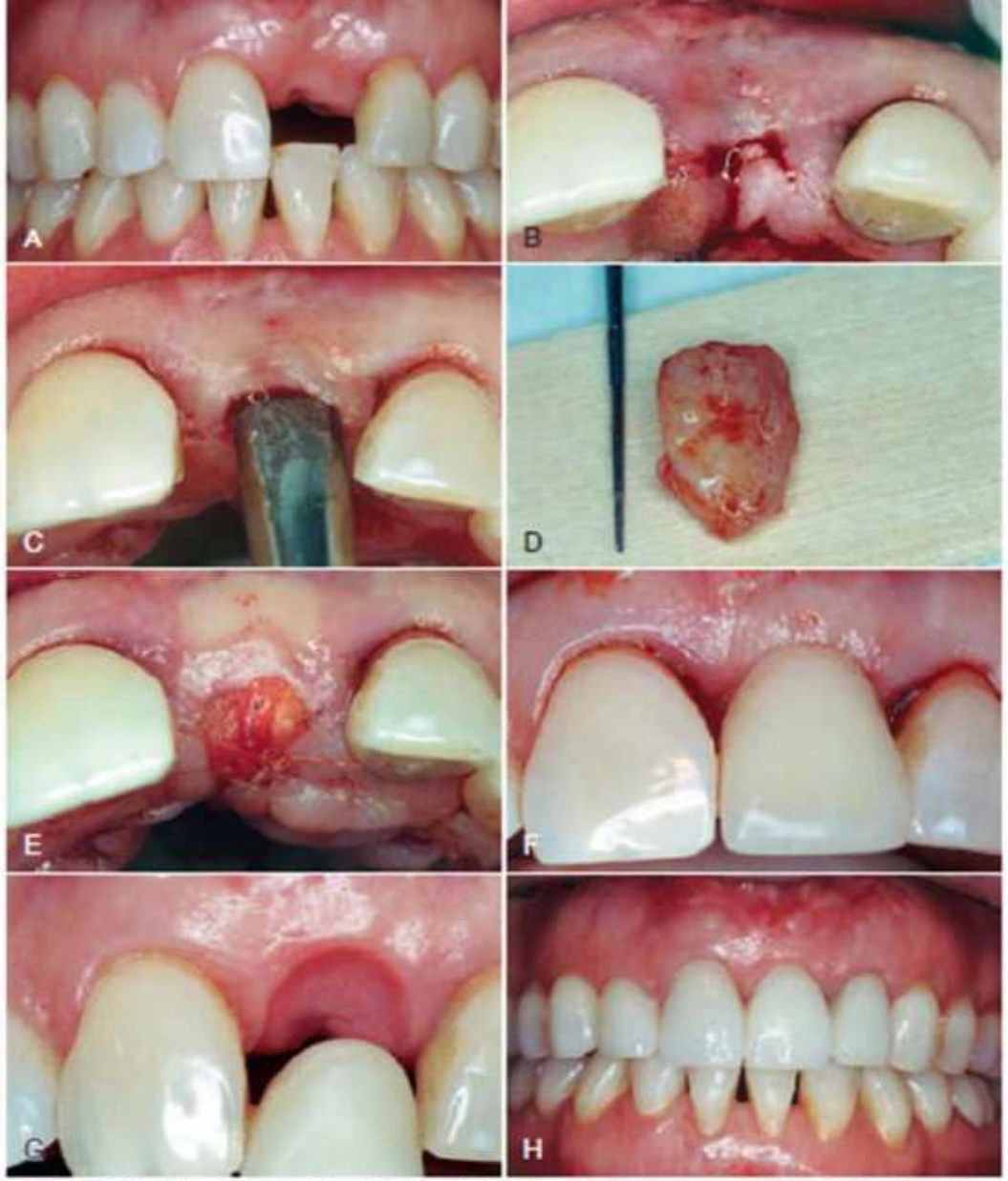
eBOX 69.1 Surgical Crown Lengthening

Indications

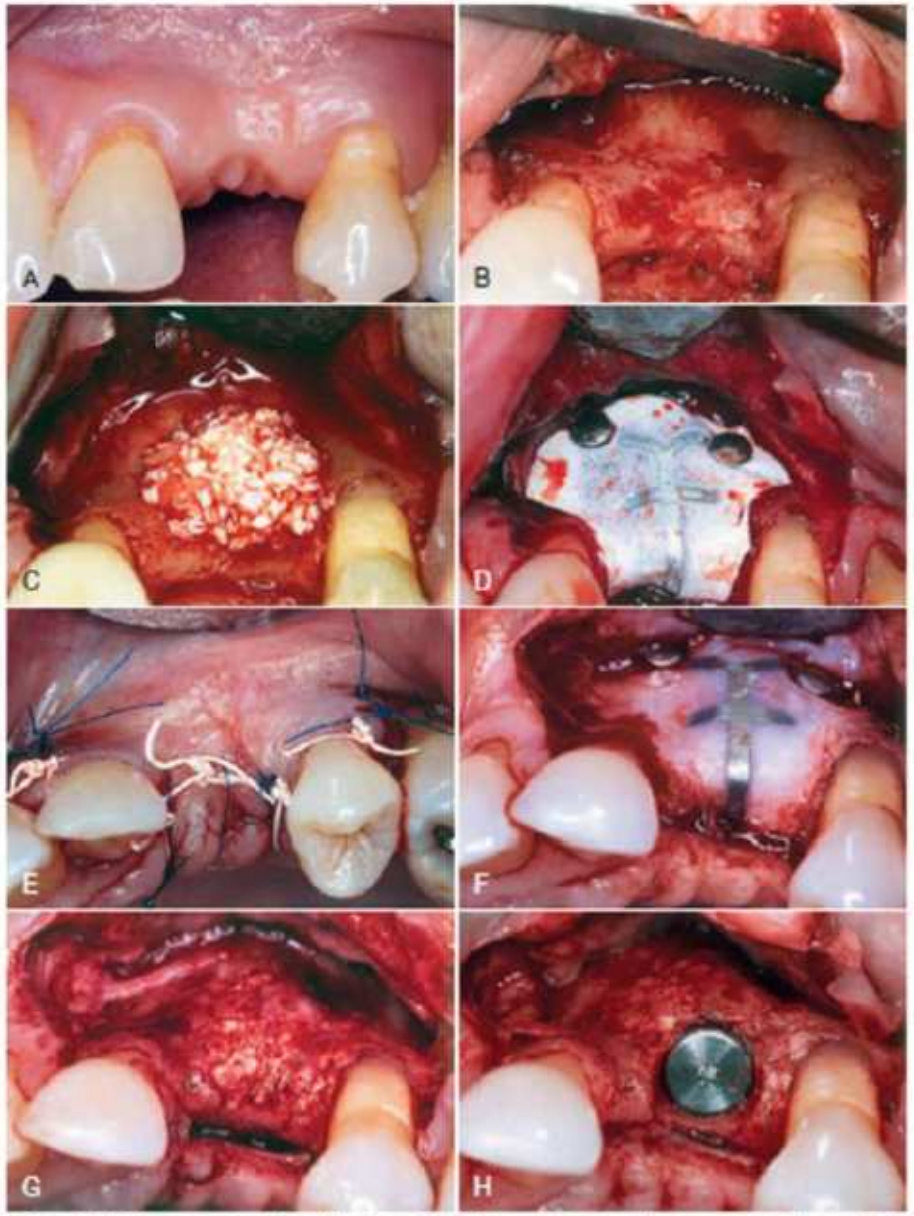
Subgingival caries or fracture.
Inadequate clinical crown length for retention.
Unequal or unaesthetic gingival heights.

Contraindications

Surgery would create an unaesthetic outcome.
Deep caries or fracture would require excessive bone removal on contiguous teeth.
The tooth is a poor restorative risk.



eFig. 69.11 (A) Loss of the maxillary left central incisor has resulted in an unaesthetic alveolar ridge defect. (B–E) An incision is made at the ridge crest, a pouch is created, and a soft tissue graft harvested from the palate is placed into the pouch. (F–H) A removable appliance with an ovate pontic is placed in light contact with the grafted site. Swelling around the pontic apex results in a tissue concavity from which the more natural-appearing final restoration emerges.



eFig. 69.12 Postextraction ridge defect is grafted with a combination of autogenous and deproteinized bovine bone and contained by nonresorbable barrier membrane.[52] After 8 months, the site is reopened and the membrane removed. A comparison of parts B and G shows significant reconstitution of hard tissue, in this case used for the installation of a dental implant. (A) Edentulous ridge before surgery. (B) Flap reflection to visualize defect. (C) Graft material placed over resorbed ridge. (D) Nonresorbable titanium-reinforced membrane placed over graft material. (E) Graft site sutured. (F) Surgical site reopened 8 months after surgery. (G) New bone over ridge. (H) Implant placed into augmented ridge.

Thank you

Non-surgical treatment Sonic and ultrasonic instrumentation

Dr Hadeel Mohammed Abbood

Non surgical treatment

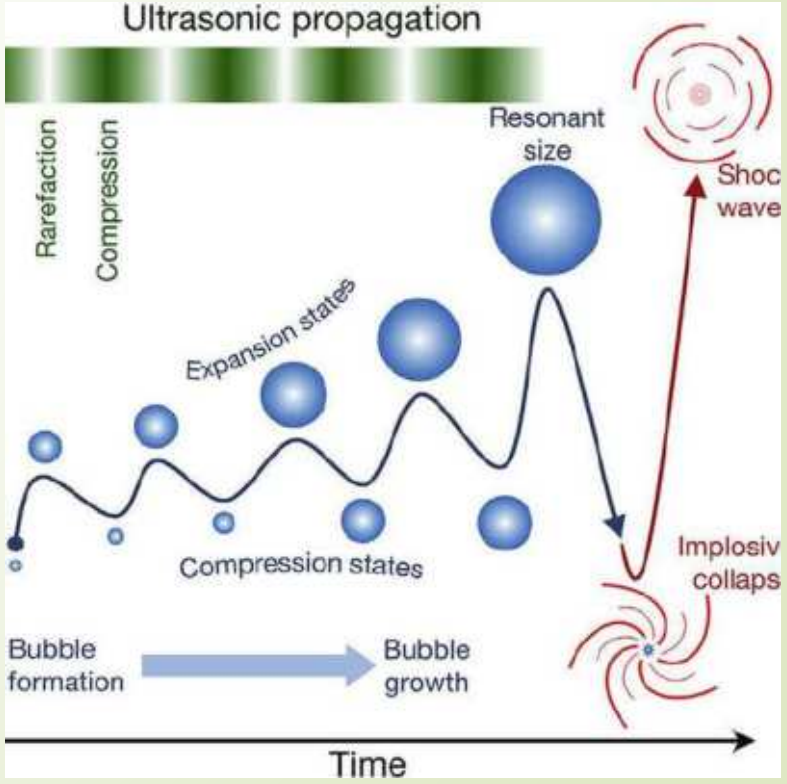
- Initial treatment
- Extended for benefits and less destructive
- Scalers and irrigators

Power driven instruments

- They may be used alone or combination with hand instruments.
- Evidence indicates that power-driven instruments provide clinical outcomes similar to those derived from hand instruments.
- Power instrumentation has the potential to make scaling less demanding and more time efficient.
- Potential hazards from using power-driven devices include:
  - Rough root surfaces,
  - Production of bioaerosols,
  - Interference with cardiac pacemakers.

Mechanism of Action of Power Scalers

- Various physical factors play a role in the mechanism of action of power scalers:
  - frequency, stroke, and water flow.
- Water contributes to three physiologic effects that play a role in the efficacy:
  - acoustic streaming, acoustic turbulence, and cavitation.
- Acoustic steaming is unidirectional fluid flow caused by ultrasound waves.
- Acoustic turbulence is created when the movement of the tip causes the coolant to accelerate, producing an intensified swirling effect. This turbulence continues until cavitation occurs.
- Cavitation is the formation of bubbles in water caused by the high turbulence. The bubbles implode and produce shock waves in the liquid, thus creating further shock waves throughout the water.
- In vitro, the combination of the 3 effects has been shown to disrupt biofilm.



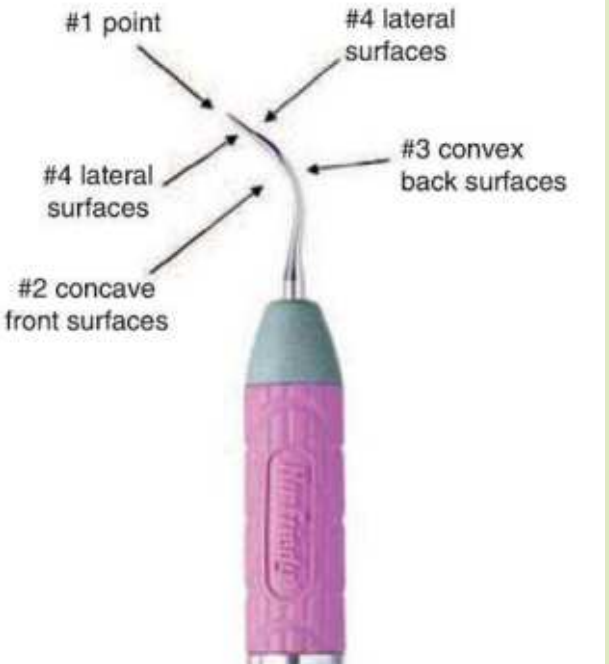
Type and Benefit of Power Instruments

Sonic units

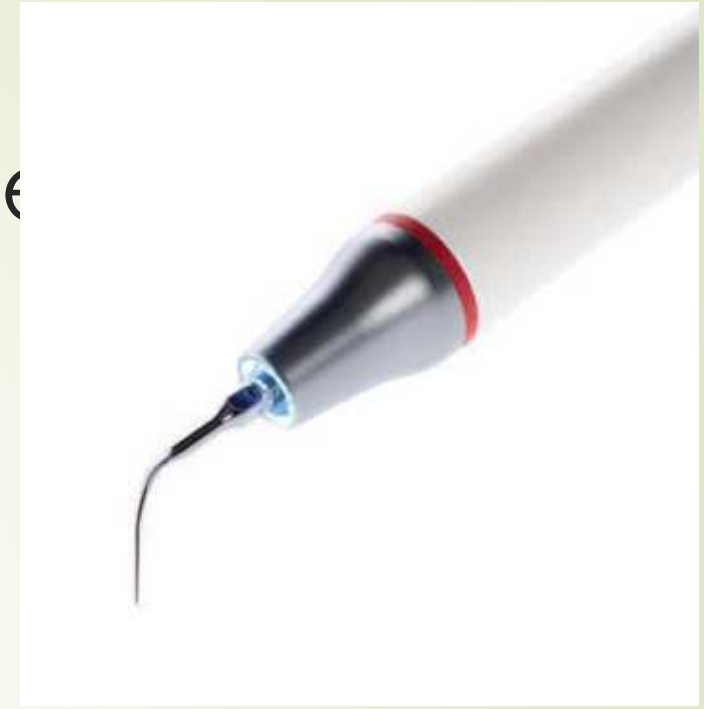
- work at a frequency of 2000 to 6500 cycles per second
- use a high- or low-speed air source from the dental unit.
- Water is delivered via the same tubing used to deliver water to a dental handpiece.
- Sonic scaler tips are large in diameter and universal in design.
- A sonic scaler tip moves in an elliptical or orbital stroke pattern. This stroke pattern allows the instrument to be adapted to all tooth surfaces.

Magnetostrictive ultrasonic scaler

- Work in a frequency range of 18,000 to 50,000 cycles per second.
- Electric power --Metal stacks change dimension magnetostrictive waves.
- Vibrations travel from the metal stack to a connecting body causes the vibration of the working tip.
- Tips move in an elliptical or orbital stroke pattern.
- This gives the tip four active working surfaces.



Piezoelectric ultrasonic scale



- work in a frequency range of 18,000 to 50,000 cycles per second.
- Ceramic disks located in the handpiece power the piezoelectric technology and change in dimension as electric energy is applied.
- Piezoelectric tips move primarily in a linear pattern, giving the tip two active surfaces.
- Various insert tip designs and shapes are available for use.


UDS-K
2
3
4
6
7
8
MIN
MAX

BOX 51.1 Advantages and Disadvantages of Mechanized Instruments Compared With Manual Instruments

Advantages

Increased efficiency
- Multiple surfaces of tip are capable of removing deposits
- No need to sharpen
- Less chance for repetitive stress injuries
- Large handpiece size
- Reduced lateral pressure
- Less tissue distention
- Water
- Lavage
- Irrigation
- Acoustic microstreaming

Disadvantages

More precautions and limitations
- Client comfort (water spraying)
- Aerosol production
- Temporary hearing shifts
- Noise
- Less tactile sensation
- Reduced visibility

From Darby ML, Walsh MM: *Dental hygiene*, ed 3, St. Louis, 2010, Saunders.

Efficiency

- Modified tip designs allow for improved access in many areas, including furcations.
- Newer, slimmer designs operate effectively at lower power settings, thus improving patients' comfort.

Tip Designs

- Some tips are designed to remove heavy supragingival calculus or debride periodontal pockets definitively.
- Large-diameter tips are created with a universal design and are indicated for the removal of large, tenacious deposits.
- A medium to medium-high power setting is generally recommended.
- Thinner-diameter tips may be site specific in design.
- The straight-tip design is ideal for use in treating patients with gingivitis and deplaquing maintenance patients.
- The right and left contra-angled instruments allow for greater access and adaptation to root morphology. These inserts are designed to work on a low-power setting (not available in our clinics).

Clinical outcomes

- The primary expected clinical outcomes from scaling and root planing are reductions in bleeding and probing depth and a gain in clinical attachment.
- Comparing power scalers with hand instruments, both types demonstrate similar outcomes
- Because the opening of a furcation is narrower than with conventional hand instruments, power scalers may be recommended as a means to improve access when scaling this type of defect.

Special Considerations

- Power-driven instruments must be used with some caution.
- Roots may be rougher post scaling than with hand instruments. Power driven instruments may increase the roughness of resin or glass ionomer restorative materials; therefore, repolishing post scaling is recommended
- Due to aerosol production, proper infection control procedures need to be implemented. Power-driven devices produce bioaerosols and splatter, which can contaminate the operator and remain in the air for up to 30 minutes. If patient has been diagnosed with contagious disease (droplet transmission), it is recommended to use hand instrument . Or using good infection control practices to minimize the hazard:
  - preprocedural rinsing with 0.12% chlorhexidine and
  - high-speed evacuation
  - A minimum of 30 min time period between patients is recommended

  These are the most efficient ways to reduce bioaerosols

Cardiac Pacemakers

- The use of ultrasonics on patients with cardiac pacemakers is somewhat controversial.
- Newer models of pacemakers often have bipolar titanium insulation that is believed to make ultrasonic and sonic instruments generally safe for use.
- An in vivo study supports this; 12 patients underwent continuous electrocardiogram monitoring during piezoelectric ultrasonic scaling and had no abnormal pacemaker functions. Conversely, an in vitro study found that ultrasonic scalers interfered with the activity of dual-system pacemakers.
- If in doubt, consult with the physician regarding any precautions or warnings from the manufacturer of the product.

BOX 51.2 Indications, Precautions, and Contraindications for Use of Mechanized Instruments

Indications

- Supragingival debridement of dental calculus and extrinsic stains
- Subgingival debridement of calculus, oral biofilm, root surface constituents, and periodontal pathogens
- Removal of orthodontic cement
- Gingival and periodontal conditions and diseases
- Surgical interventions
- Margination (reduces amalgam overhangs)

Precautions

- Unshielded pacemakers
- Infectious diseases: human immunodeficiency virus, hepatitis, tuberculosis (active stages)
- Demineralized tooth surface
- Exposed dentin (especially associated with sensitivity)
- Restorative materials (porcelain, amalgam, gold, composite)
- Titanium implant abutments unless using special insert (e.g., Quixonic SofTip Prophy Tips)
- Children (primary teeth)
- Immunosuppression from disease or chemotherapy
- Uncontrolled diabetes mellitus

Contraindications

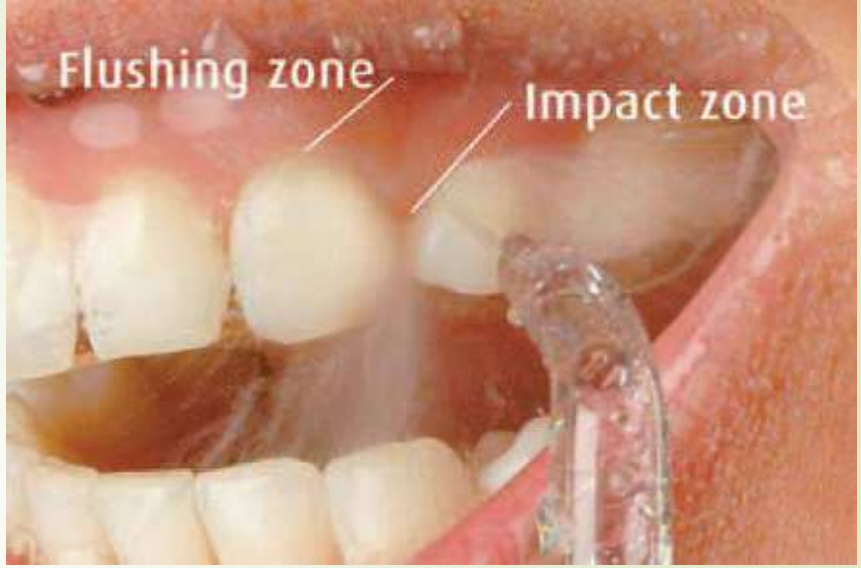
- Chronic pulmonary disease: asthma, emphysema, cystic fibrosis, pneumonia
- Cardiovascular disease with secondary pulmonary disease
- Swallowing difficulty (dysphagia)

- Instrumentation with the ultrasonic device is different from hand instrumentation.
- A pen grasp with light pressure is preferred, as is using an extraoral fulcrum.
- Deposits are removed coronally to apically.
- For deposits in the embrasure area, a horizontal or transverse stroke is recommended.
- A deplaquing stroke should be used when the focus is removal of biofilm and soft debris for the resolution of gingival inflammation. This stroke entails accessing every square millimeter

Home and Self-Applied Irrigation

- The oral irrigator (also called a dental water jet or water flosser) was introduced in 1962.
- Contrary to myth and misunderstanding,
- Emerging evidence indicates that the oral irrigator effectively removes biofilm
- As effective as dental floss when added to toothbrushing
- No adverse events have been reported

Mechanism of Action of Irrigation



- pulsation and pressure phase.
- Pulsation creates a decompression phase that allows the water or solution to penetrate subgingivally.
- Followed by a compression phase that expels bacteria and debris from the pocket.
- Physiologically, pulsation, along with pressure and water velocity, creates shear hydraulic forces that are capable of removing bacterial biofilm from treated areas.
- Clinical efficacy 1200 to 1400 pulses per minute set at a minimum of 60 psi.
- The oral irrigator is safe to use at higher pressure settings.
- Many types of oral irrigators are commercially available, but as with other self-care products, research available from one product brand should not be extrapolated to other brands, because they may have used different pressure settings and pulsation rates

- A variety of tips can be used with an oral irrigator.
- Tips placed above the gingival margin at 90 degree angle result in a pocket penetration of 50% on average
- Tips that are placed supragingivally are recommended for full-mouth irrigation or cleansing.
- These tips include a traditional jet tip, with bristles or filaments

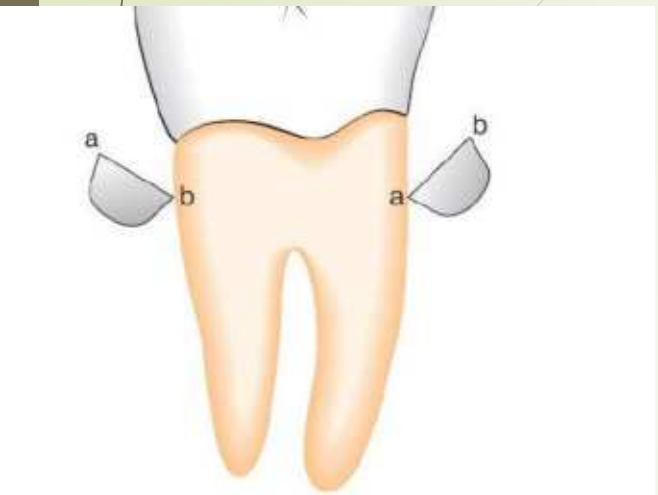
- The soft, site-specific subgingival tip penetrates to about 90% of the 6mm PD And 64% of the 7 mm PD
- The subgingival tip is generally used after full-mouth cleaning for localized irrigation of a specific site that is difficult to access, such as a deep pocket, a furcation, an implant, or a crown and bridge

- Evaluated outcomes include removal of plaque biofilm and reductions in calculus, gingivitis, bleeding on probing, probing depth, periodontal pathogens, and inflammatory mediators
- The use of an antimicrobial agent, such as diluted chlorhexidine, or an essential oil generally enhances reductions in gingivitis and bleeding.
- Irrigation for a period of 3 to 6 months results in reduced periodontal bacteria.
- Small improvements in probing depth.
- Incidence of bacteremia can range from 7% in people with gingivitis to 50% in those with periodontitis.
- In comparison, the incidence of bacteremia from string flossing has been shown to be 40% in people with periodontitis and 41% in periodontally healthy individuals.

Individuals With Special Considerations

- Both children and adults undergoing orthodontic therapy have shown significant benefits from using a dental water jet.
- For individuals with implants, a modified jet tip with filaments has been found to be both safe and effective. Patients who used the oral irrigator at 60 psi with warm water, had twice the reduction in bleeding around implants compared with patients who used floss. No adverse events were reported.
- The site-specific subgingival tip has also been shown to be safe and effective for use on implants.
- Oral irrigator has also been found to improve periodontal health in people with type 1 or 2 diabetes.

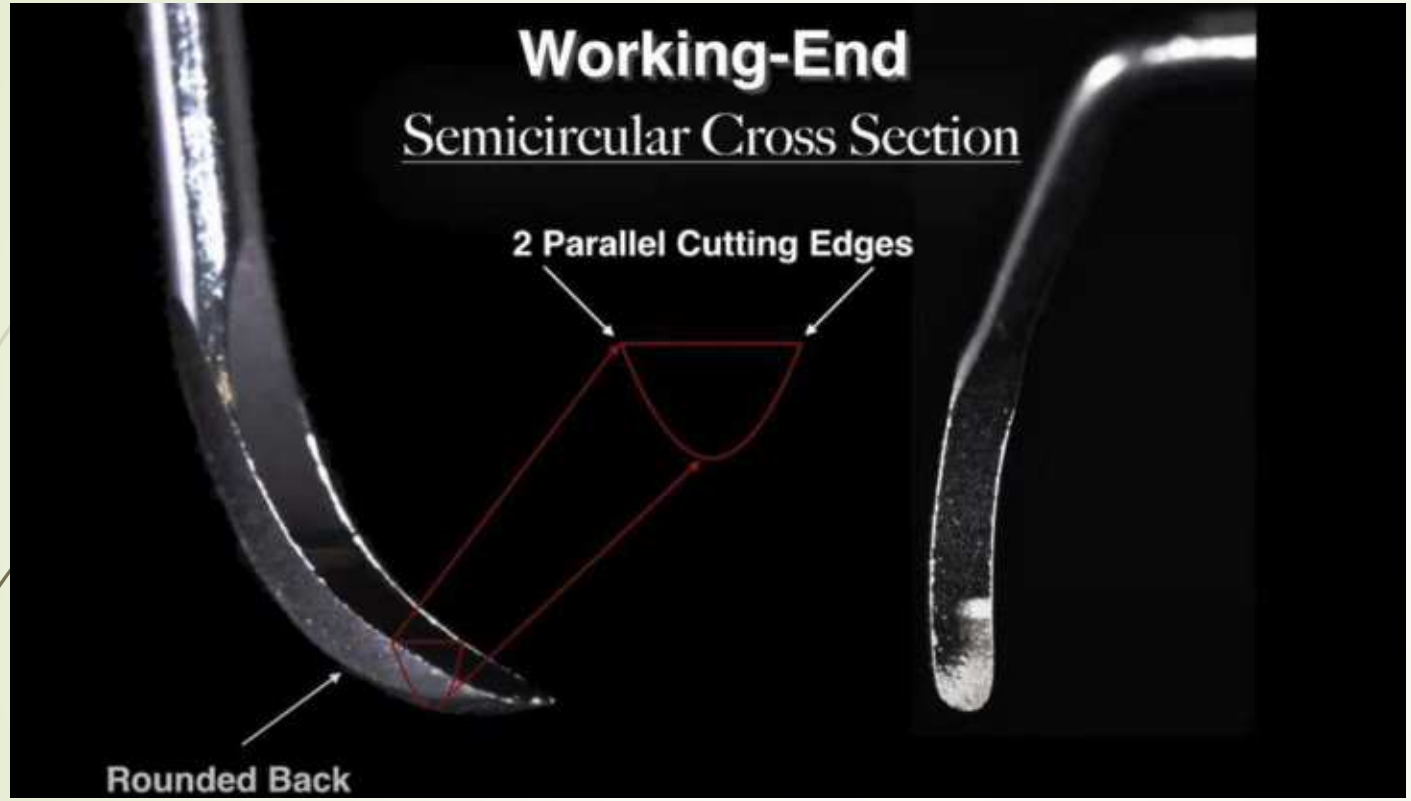
Root Planing *(Universal Curettes)*

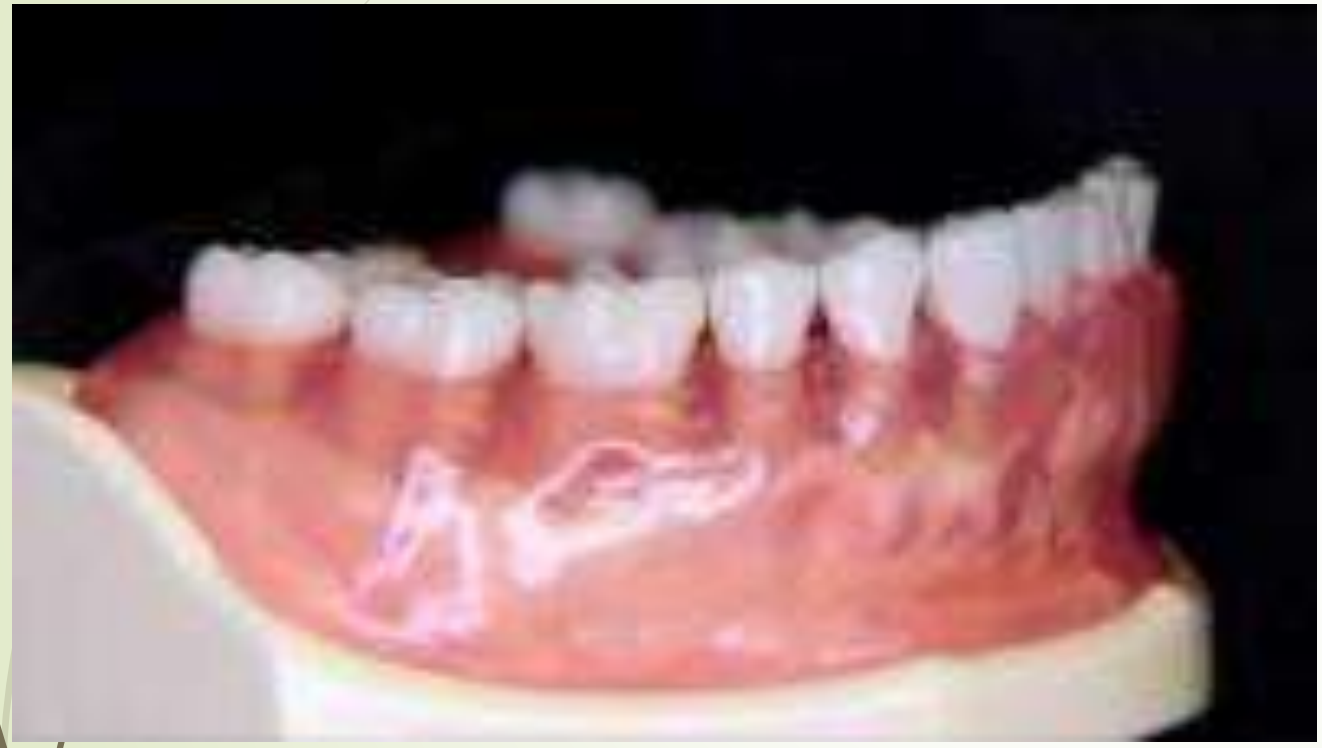


50.23 Adaptation of the universal curette on a posterior tooth.

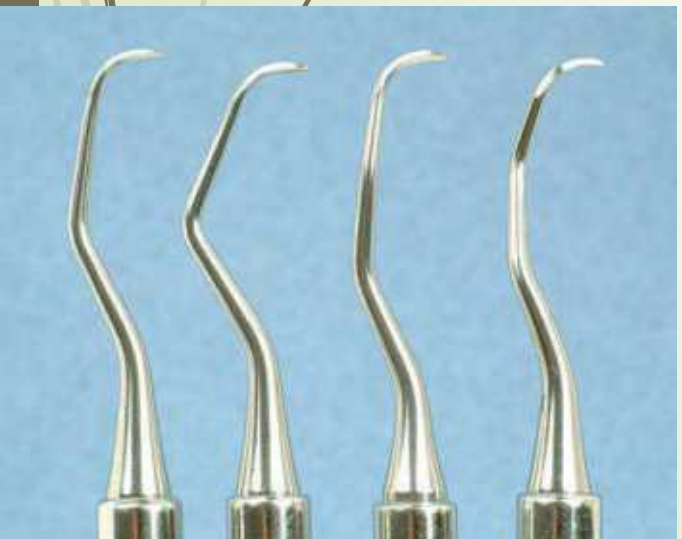
Fig. 50.14 Basic characteristics of a curette: spoon-shaped blade and rounded tip.

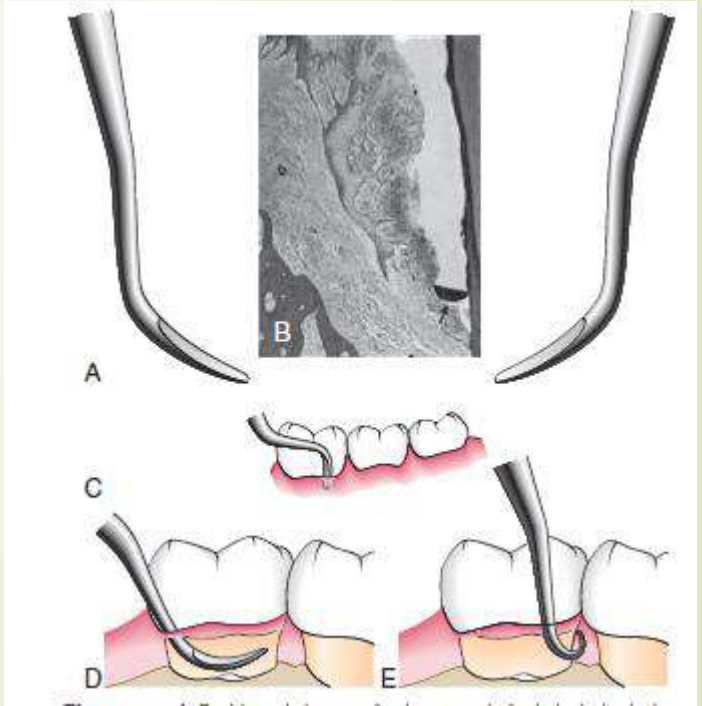
- The working ends of the universal curette are designed in pairs so that all surfaces of the teeth can be treated with one double-ended instrument or a matched pair of single-ended instruments.
- Both cutting edges of the universal curette blade are used.
- In any given quadrant, when approaching the tooth from the facial aspect, one end of the universal curette adapts to the mesial surfaces and the other end adapts to the distal surfaces. When approaching from the lingual aspect in the same quadrant, the double ended universal curette must be turned end for end because the blades are mirror images. This means that the end that adapts to the mesial surfaces on the facial aspect also adapts to the distal surfaces on the lingual aspect, and vice versa.
- Both ends of the universal curette are used for instrumentation of the anterior teeth.
- On posterior teeth, however, because of the limited access to distal surfaces, a single working end can be used to treat both mesial and distal surfaces by using both of its cutting edges. To do this, the instrument is first adapted to the mesial surface with the handle nearly parallel to the mesial surface. Because the face of the universal curette blade is honed at 90 degrees to the lower shank. the lower shank must be tilted slightly toward the tooth.
- The distal surface of the same posterior tooth can be instrumented with the opposite cutting edge of the same blade. This cutting edge can be adapted at proper working angulation by positioning the handle so that it is *perpendicular* to the distal surface.

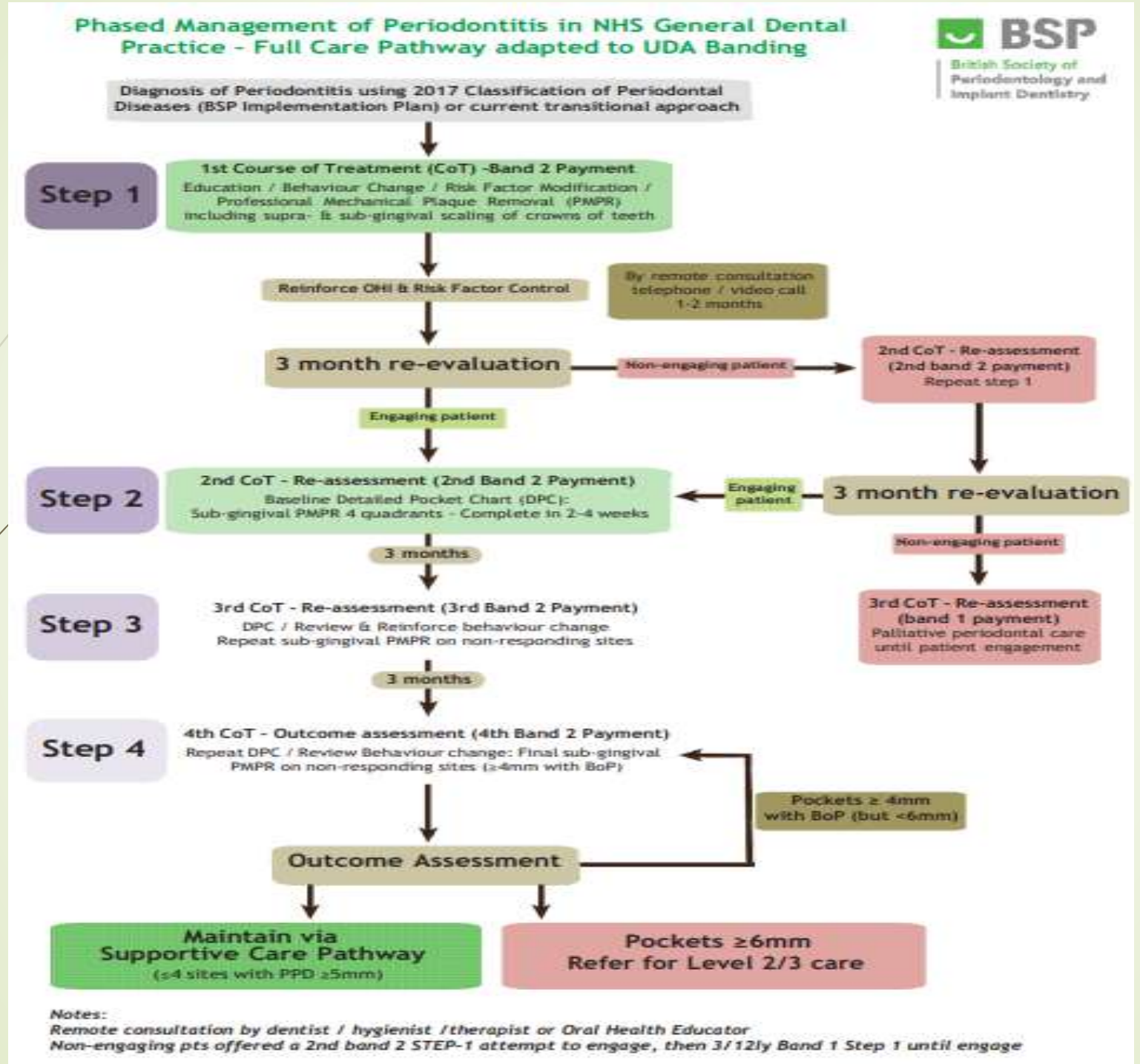

Working-End
Semicircular Cross Section
2 Parallel Cutting Edges
Rounded Back



- When adapting the universal curette blade, as much of the cutting edge as possible should be in contact with the tooth surface, except on narrow convex surfaces such as line angles.
- When the cutting edge is adapted to the tooth, pressure should be concentrated on the lower third of the blade during scaling and root planing strokes.
- The primary advantage of these curettes is that they are designed to be used universally on all tooth surfaces, in all regions of the mouth.
- However, universal curettes have limited adaptability for the treatment of deep pockets in which apical migration of the attachment has exposed furcations, root convexities, and developmental depressions.
- For this reason, many clinicians prefer Gracey curettes and the newer modifications of Gracey curettes, which are area specific and specially designed for subgingival scaling and root planing in periodontal patients.






Phased Management of Periodontitis in NHS General Dental Practice – Full Care Pathway adapted to UDA Banding
BSP
British Society of Periodontology and Implant Dentistry
Diagnosis of Periodontitis using 2017 Classification of Periodontal Diseases (BSP Implementation Plan) or current transitional approach
Step 1
1st Course of Treatment (CoT) -Band 2 Payment
Education / Behaviour Change / Risk Factor Modification / Professional Mechanical Plaque Removal (PMPR) including supra- & sub-gingival scaling of crowns of teeth
Reinforce OHI & Risk Factor Control
By remote consultation telephone / video call 1-2 months
3 month re-evaluation
Non-engaging patient
2nd CoT - Re-assessment (2nd band 2 payment) Repeat step 1
Engaging patient
Step 2
2nd CoT - Re-assessment (2nd Band 2 Payment)
Baseline Detailed Pocket Chart (DPC): Sub-gingival PMPR 4 quadrants - Complete in 2-4 weeks
Engaging patient
3 month re-evaluation
Non-engaging patient
3 months
Step 3
3rd CoT - Re-assessment (3rd Band 2 Payment)
DPC / Review & Reinforce behaviour change
Repeat sub-gingival PMPR on non-responding sites
3rd CoT - Re-assessment (band 1 payment) Palliative periodontal care until patient engagement
3 months
Step 4
4th CoT - Outcome assessment (4th Band 2 Payment)
Repeat DPC / Review Behaviour change: Final sub-gingival PMPR on non-responding sites (≥4mm with BoP)
Pockets ≥ 4mm with BoP (but <6mm)
Outcome Assessment
Maintain via Supportive Care Pathway
(≤4 sites with PPD ≥5mm)
Pockets ≥6mm
Refer for Level 2/3 care
Notes:
Remote consultation by dentist / hygienist /therapist or Oral Health Educator
Non-engaging pts offered a 2nd band 2 STEP-1 attempt to engage, then 3/12ly Band 1 Step 1 until engage

Thank you