Treatment plan guidelines

Phase 1 (behavior change, removal of supragingival dental

biofilm and risk factor control)

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Outline for the lecture

Self-performed supragingival biofilm control: 0

- i- Oral hygiene practices to control gingival inflammation
- ii- Behavioral change for oral hygiene improvement
- iii- Motivational interviewing and cognitive behavioral therapy
- o Adjunctive therapies for gingival inflammation
- o Professional supragingival dental biofilm control
- o Risk factor control:
- i- Local risk factor control
- ii- Tobacco smoking cessation interventions
- iii- Promotion of diabetes control interventions



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- The major goal of phase I therapy is to control the factors responsible for periodontal inflammation; this involves educating the patient in the removal of bacterial plaque or biofilm.
- Phase 1 or cause related therapy or non-surgical periodontal therapy – first in the chronologic sequence of procedures that constitute periodontal treatment
- □ The Objective of this phase is
- alter or eliminate the microbial etiology and factors that contribute to gingival and periodontal diseases
- halting the progression of the disease and returning the dentition to a state of health and comfort





Self-performed supragingival biofilm control:

1- Oral hygiene practices to control gingival inflammation

Plaque control instructions Essential step Explain the etiology of the disease to the patient Instruct the correct technique – targeted oral hygiene Instruct regarding the use of dental floss or interdental aids Multiple appointments to evaluate, reinforce and improve patient's oral hygiene







Self-performed supragingival biofilm control:

2- Behavioral change for oral hygiene improvement

The person must have the physical or psychological ability to change to the desired behaviour. This includes a person's knowledge of what the desired behaviour is and why it is important, the skills required to make the change and the self-control needed to start and maintain that desired behaviour over the long-term.

- improving oral hygiene
- optimising exposure to fluoride
- reducing sugar intake and healthier eating
- stopping smoking and tobacco use through very brief advice





Self-performed supragingival biofilm control:

3- Motivational interviewing and cognitive behavioral therapy

is an evidence-based psychotherapeutic approach that focuses on engaging, encouraging and facilitating intrinsic motivation within the individual in order to promote behaviour change

cognitive behavioral therapy is used to help patients actively change their behaviors





Adjunctive therapies for gingival inflammation

chlorhexidine mouthrinse used as an adjunct to mechanical oral hygiene procedures for the control of gingivitis Some cases need to prescribe antibiotic drug for treatment



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o Professional supragingival dental biofilm control

> It includes removal of dental plaque from all accessible surfaces using curettes and ultrasonic devices. It is followed by polishing with rubber cup and dentifrice/polishing agents. All interproximal surfaces are cleaned with dental floss





o Risk factor control:

i- Local risk factor control

calculus, caries, tooth position, anatomical features, iatrogenic factors, and trauma.

ii- Tobacco smoking cessation interventions

iii- Promotion of diabetes control interventions

Phase 2 (cause-related therapy) o Subgingival instrumentation:

Scaling

Root planing

- Removal of plaque-retentive factors
- Use of adjunctive systemically administered antibiotics to subgingival instrumentation
- Re-evaluation of the cause-related therapy
- o Decision to refer for specialist

Subgingival instrumentation:

- Scaling is the process by which biofilm and calculus are removed from both supragingival and subgingival tooth surfaces.
- □ When biofilm and calculus form on enamel, the deposits are usually superficially attached to the surface and are not locked into irregularities.
- □ Scaling alone is sufficient to completely remove biofilm and calculus from enamel, leaving a smooth, clean surface.
- □ Root surfaces exposed to biofilm and calculus pose a different problem. Deposits of calculus on root surfaces are frequently embedded in cemental irregularities.
- Subgingival calculus is porous and harbors bacteria and endotoxin and therefore should be removed completely



FIG. 50.7 The five basic scaling instruments. (A) Curette; (B) sickle; (C) file; (D) chisel; (E) hoe.

Ultrasonic and Sonic Scaling:

Ultrasonic scalers may be used for removing plaque and stain, scaling, root planing, curetting, and surgical debridement.

Ultrasonic scaler used for

- 1. S & RP.
- 2. Curetting.
- 3. debridement during p.d. surgery.
- 4. removal of stain.
- 5. overhanging restoration.
- Rough sur. left after use large tip

But such roughness not interfere with healing.

 \succ The vibrational energy produced by the ultrasonic instrument makes it useful for removing heavy, tenacious deposits of calculus and stain.

2 type of ultrasonic s.:

> Magnetostrictive:

vibration of tip is elliptical (all sides of tip are active). frequency range of 18,000 to 50,000 cycles per second.

Piezoelectric; vibration of tip is linear meaning, or back and forth, the 2 sides of tip are most active. frequency range of 18,000 to 50,000 cycles per second. Piezoelectric tips move, primarily in a linear pattern, giving the tip two active surfaces.

Magnetostrictive ultrasonic inserts generate heat and require water for cooling.
 Sonic and piezoelectric units do not generate heat but still use water for cooling of frictional heat and for flushing away debris.



Ultrasonic s.:

Its contraindicated for

1. Patient with artificial pacemaker

2- patient with communicable diseases that can be transmitted by aerosols should not be treated with ultrasonic or sonic scaling devices.

3-for implant ,porcelain ,bonded restoration which can be fractured or removed Advantages:

Less chair side time & less trauma so less postoperative discomfort

➤ Stain removal easier.

> Superior to hand instrument in cleaning furcation area.

Sonic scaler:

Sonic units work at a frequency of 2000 to 6500 cycles per second and use a high- or low-speed air source from the dental unit.

> Sonic units consist of a hand piece that attaches to a compressed air line and uses a variety of specially designed tips.

> A sonic scaler tip travels in an elliptical or orbital stroke pattern. This stroke pattern allows the instrument to be adapted to all tooth surfaces.



Blis-sonic K scaler kit, non swivel/fixed back end







Supragingival Scaling: For the treatment of interproximal spaces and for supragingival scaling





Remove Supragingival heavy calculus



Supragingival Scaling:

Remove calculus and bacterial Plaque on supragingival, neck of teeth and hard calculus on the teeth point.



Supragingival Scaling:

Remove calculus and bacterial plaque on supragingival, neck of teeth and hard calculus on the teeth point.



➢Root planing is the process by which residual embedded calculus and portions of Cementum are removed from the roots to produce a smooth, hard, clean surface.

 \succ R.P. done either by closed or opened technique:

<u>Closed:</u> mean without displacement of Gingiva. <u>Opened:</u> with displacement of Gingiva & exposure of Root surface & alveolar bone for access & visibility

<u>Instrument used</u>;

- \succ Hand Inst.
- ➤ ultrasonic & sonic scaler.
- \succ rotating Inst.



Scaling

Root Planing

Advantages & disadvantages:

- 1. Less trauma ,Gingiva shrinkage is less (esthetic).
- 2. Good result with maintenance.



2. Need manual dexterity.

Curettes:

- The curette is the instrument of choice for removing deep subgingival calculus, root planing, removed altered Cementum, and removing the soft tissue lining the periodontal pocket.
- \succ Each working end has a cutting edge on both sides of the blade and a rounded toe.

➤ Curettes are finer than sickle scalers and do not have any sharp points or corners other than the cutting edges of the blade. Therefore curettes can be adapted for and provide good access to deep pockets, with minimal soft tissue trauma.



> The two basic types of curettes:

- **Universal**.
- Gracey curette (area specific).
- The *cutting edge* of an instrument is formed by the angular junction of two surfaces of its blade.
- \succ The cutting edges of a curette, for example, are formed where the face of the blade meets the lateral surfaces.

Universal Curettes:

> 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.

> The blade size and the angle and length of the shank may vary, but the face of the blade of every universal curette is at a 90-degree angle (perpendicular) to the lower shank when seen in cross section from the tip.

> The blade of the universal curette is curved in one direction from the head of the blade toward the toe.

 \triangleright universal curettes have limited adaptability for the treatment of deep pockets in which apical migration of the attachment has exposed furcation, root convexities, and developmental depressions.



Area-Specific Curettes (Gracey Curettes):

 \succ a set of several instruments designed and angled to adapt to specific anatomic areas of the dentition.

> These curettes and their modifications are probably the best instruments for subgingival scaling and root planing because they provide the best adaptation to complex root anatomy.

> Gracey curettes because they are angled approximately 60 to 70 degrees from the lower shank term *offset blade*.









Double-ended Gracey curettes are paired in the following manner: Gracey #1-2 and #3-4. Gracey #5-6. Gracey #7-8 and #9-10. Gracey #11-12. Gracey #13-14.







Comparison of Area-Specific (Gracey) and Universal Curettes

	Gracey Curette	Universal Curette
Area of	Set of many curettes designed for specific	One curette designed for all areas
use	areas and surfaces	and surfaces
Cutting Edge		
Use	One cutting edge used; work with outer edge	Both cutting edges used; work with
	only	either outer or inner edge
Curvature	Blade curves from the shank toward the toe	Blade curves only from the shank
	and also appears to curve to the side	toward the toe, not to the side
Blade	Offset blade; face of blade beveled at 60	Blade not offset; face of blade
angle	degrees to shank	beveled at 90 degrees to shank

R.P. Procedure:

➤ Lower shank should be parallel with long axis of tooth , so cutting edge will be at 45 with tooth surface.

R.P. continue with controlled overlapping ,short, powerful strokes without withdrawing inst. from pocket (*working stroke*).

In proximal surface place with long axis neither tilted away from tooth nor tilted too far toward tooth.

Finishing stroke:

longer lighter with less lateral pressure





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strokes:

 \succ Three basic types of strokes are used during instrumentation: the exploratory stroke, the scaling stroke, and the root-planing stroke.

> Any of these basic strokes may be activated by a pull or push motion in a vertical, oblique, or horizontal direction.

- Vertical and oblique strokes are used most frequently. Horizontal strokes are used selectively on line angles or deep pockets that cannot be negotiated with vertical or oblique strokes.
- The direction, length, pressure, and number of strokes necessary for either scaling or root planing are determined by four major factors:
- \succ (1) gingival position and tone.
- \succ (2) pocket depth and shape.
- \succ (3) tooth contour.
- \succ (4) the amount and nature of the calculus or roughness.



 scaling stroke: is a short, powerful pull stroke that is used with blade instrument

for the removal of both supragingival and subgingival calculus.

- The scaling motion should be initiated in the forearm and transmitted from the wrist to the hand with a slight flexing of the fingers.
- The scaling stroke is not initiated in the wrist or fingers, nor is it carried out independently without the use of the forearm.

 Root-planing stroke: is a moderate to light pull stroke that is used for final smoothing and planing of the root surface. The design of the curette, which allows it to be more easily adapted to subgingival tooth contours, makes it particularly suitable for root planing in periodontal patients.

Removal of plaque retentive factors:

- 1. replacement.
- 2. Using scaler or file.
- 3. Flame shaped diamond bur mounted on H.P. for rotating movement.
- 4. Flat diamond stone bur mounted on H.P. for horizontal reciprocal movement (EVA system).
- 5. Metal strip with special handle.









403 Old amalgam restorations before and after recontouring Left: The rough, discolored surface of old restorations enhances plaque accumulation. Smoothing and polishing of such restorations will reduce the magnitude of microorganisms in the oral cavity. Care must be taken not to disturb functional contacts in centric and ateral occlusion during the smoothing and polishing of occlusal surfaces.

Right: Contoured and polished old amaigam restorations.

404 Amaigam overhang before and after removal Left: Tooth 46 exhibits a pronounced marginal overhang on the mesial aspect (massive plaque accumulation, arrow). A deep bony pocket is also located near this iatrogenic niche.

Right: The proximal overhang was removed and the restoration was polished. The margin is now perfect, discouraging any further plaque accumulation.

406 Subgingival overhanging crown margins This often observed iatrogenic irritant can be detected clinically using a fine pointed explorer. Plaque accumulates beneath the open margin, and gingivitis often persists for years. Such massive crown margin

overhangs may be removed with a diamond supragingivally, or a round bur may be used to remove the entire crown margin, as depicted here.

Right: The radiograph clearly reyeals that thecrown margin isoverhanging interproximally (arrows).

407 Overhanging crown margin removed Caries is often encountered following radical removal of an overhanging subgingival crown margin. Before initial therapy, this carious process could not be diagnosed either radiographically or clinically.

Right: The crown margin, removed in one piece!













405 Smoothing the proximal restoration surface with strips Left: LM strip holder with diamondcoated steel strip during smoothing of the distal surface of tooth 36. The contact area is protected.

Right: The interdental surface of the restoration is finally polished using fine, very mildly abrasive linen strips, which can also be used in the contact area.

Goal: Smooth proximal surfaces make possible perfect interdental hygiene with dental floss.

408 Crown margins now supragingival The old crowns serve nicely as "temporaries" following treatment of the cervical caries. Interdental hygiene can now be accomplished using Stimudents.

following completion of periodontal therapy.

Right: The radiograph depicts the removal of the major overhangs (metal residue in tissue; compare p.170).







After *1-3* m. of completion of phase I; Evaluation should be done for the:

- 1- Resolution of Gingival inflammation
- 2-Reduction of pocket depth & attachment loss
- 3- Reduction of tooth mobility
- 4- O.H. improvment
- 5- Rechecking presence of ca.,Root caries ,defective restoration

The results of Evaluation showed the following ➢ Good O.H. but still BOP, no reduction of P.D.; *p.d. surgery*

Good O.H., no G. inflammation, reduction of P.D. & CAL; <u>maintenance phase</u>

Poor O.H. lack compliance or ability for home care; <u>motivation &</u> <u>instruction</u>
Decision to Refer for Specialist Treatment

- It is fortunate that many periodontally involved cases do not require any further therapy beyond phase I therapy. Therefore, these patients can be seen by general dentists for routine maintenance therapy.
- It is critical to be skilled in determining which patients would benefit from specialist care and deciding when a patient should be referred.
- surgical treatment in deep pockets, those >6 mm, gained 0.6 mm more probing depth reduction and 0.2 mm more clinical attachment gain than did deep pockets treated with scaling and root planing alone.
- pockets of 4 to 6 mm probing depth, scaling and root planing resulted in 0.4 mm more attachment gain than surgical procedures, and shallow pockets of 1 to 3 mm had 0.5 mm less attachment loss compared with surgical results.
- The concept of the critical probing depth of 5.4 mm has been advanced to assist in making the determination to proceed to surgical intervention. This is the measurement above which therapy will result in clinical attachment gain and below which it will result in clinical attachment loss.



The 5-mm standard for referral to a periodontist is based on root length, probing depth, and clinical attachment loss. The standard serves as a reasonable guideline to analyze the case for referral for specialist care. CEJ, Cementoenamel junction.

In addition to the 5-mm probing depth criterion, other factors must be considered in the decision to refer to a periodontal specialist:

1. Extent of the disease and generalized or localized periodontal involvement. The amount of bone loss, even in localized areas, suggests the need for specialized surgical techniques.

2. Root length. Short-rooted teeth are jeopardized to a greater extent by the 5-mm clinical attachment loss criterion than teeth with long roots.

3. Hypermobility. Excessive tooth mobility suggests that contributing factors may be responsible for the mobility. The extent of mobility could mean that the prognosis for the tooth may be guarded to poor.

4. Difficulty of scaling and root planing. The presence of deep pockets and furcations makes instrumentation difficult, but the results can often be improved with surgical access.

5. Restorability and importance of particular teeth for reconstruction. Long-term prognosis of each tooth is important when considering extensive restorative work.

6. Age of the patient. Younger patients with extensive attachment loss are more likely to have aggressive forms of disease that require advanced therapy.

7. Lack of resolution of inflammation after thorough plaque or biofilm removal and excellent scaling and root planing. If inflammation and progressive deepening of the pocket continue, further therapy will be necessary. Such cases require an

understanding of the etiology to determine the best course of treatment.

Periodontology

Periodontal indices

Periodontal indices o Definition o Gingival index (Loe and Silness) o Plaque index (Silness and Loe) o Plaque index (O'leary) o Plaque index (Quigely Hein) o Probing pocket depth o Clinical attachment loss o Basic Periodontal Examination (BPE) o Modified Gingival Index o Bleeding on probing o Furcation involvement index o Calculus index o Recession index (Miller) o Recession index (Cairo)

INTRODUCTION

- Dental index or indices are devices to find out the incidence, prevalence and severity of the disease, based on which preventive programs can be adopted.
- An index is an expression of the clinical observation in a numerical value. It helps to describe the status of the individual or a group with respect to a condition being measured.

DEFINITION

An index is defined as 'A numerical value describing the relative status of the population on a graduated scale with definite upper and lower limits which is designed to permit and facilitate comparison with other population classified with the same criteria and the method' (- Russell A.L)

Oral indices are essentially set of values, usually numerical with maximum and minimum limits, used to describe the variables or a specific conditions on a graduated scale, which use the same criteria and method to compare a specific variable in individuals, samples or populations with that same variables as is found in other individuals, samples or populations.- ("George P Barnes" – 1985)

An index is an expression of clinical observation in numeric values. It is used to describe the status of the individual or group with respect to a condition being measured. The use of numeric scale and a standardized method for interpreting observations of a condition results in an index score that is more consistent and less subjective than a word description of that condition. – "Esther M Wilkins" - 1987

Gingival index (Loe and Silness)

Grade

0-3

	Grade O	Normal gingiva; no inflammation; no discoloration (erythema); no bleeding
Abbreviation Gl	1	Mild inflammation; slight erythema; minimal superficial alterations. No bleeding
	2	Moderate inflammation; erythema; bleeding on probing
	3	Severe inflammation; severe erythema and swelling; tendency to spontaneous bleeding; possible ulceration.

Grade O	No Plaque	\square		
1	Thin plaque layer at the gingival margin, only detectable by scraping with a probe	W		
2	Moderate layer of plaque along the gingival margin; interdental spaces free, but plaque is visible to the naked eye	\square	Abbreviation	Grade
3	Abundant plaque along the gingival margin; interdental spaces filled with plaque	$\overline{\mathbb{Q}}$	Pl	0-3



o Plaque index (Quigely Hein)

Score

- 0 No plaque at the cervical margin
- Separate flecks of plaque at the cervical margin of the tooth
- 2 A thin continuous band of plaque (≤1 mm) at the cervical margin of the tooth
- 3 A band of plaque wider than 1 mm but covering less than one-third of the crown of the tooth
- 4 Plaque covering at least one-third but less than two-thirds of the crown of the tooth
- 5 Plaque covering two-thirds or more of the crown of the tooth



The periodontal pocket is defined as a pathologically deepened gingiva sulcus. Deepening of gingiva sulcus may occur by coronal movement of the gingiva margin, apical displacement of gingiva attachment or combination of above.

o Probing pocket depth







CLIINICAL ATTACHMENT LOSS (CAL)



C. Periodontal health (pristine gingiva)







BASIC PERIODONTAL EXAMINATION (BPE)

Probing depth	Observation	BPE Score
Black band completely visible	No probing depths >3.5 mm, no calculus/ overhangs, no bleeding after probing	0
Black band completely visible	No probing depths >3.5 mm, no calculus/ overhangs, but bleeding after probing	1
Black band completely visible	No probing depths >3.5 mm, but supra- or sub- gingival calculus/overhangs present	2
Black band partially visible	Probing depth(s) of 3.5 – 5.5 mm present	3
Black band entirely within the pocket	Probing depth(s) of 6 mm or more present	4
N/A	Furcation involvement	*





The Modified Gingival Index (MGI).

Score	Inflammation	Appearance
0	Normal	None
1	Mild inflammation	Slight changes in color and texture, but not in all portions of gingival marginal or papillary
2	Mild inflammation	Slight changes in color and texture in all portions of gingival marginal or papillary
3	Moderate	Bright surface inflammation, erythema, edema, and/or hypertrophy of gingival marginal or papillary
4	Severe inflammation	Erythema, edema, and/or marginal gingival hypertrophy of the unit or spontaneous bleeding, papillary, congestion, or ulceration



bleeding on probing score







furcation involvement INDEX





Grade I

Grade II

Grade III





Calculus INDEX



Score Criteria

No debris or calculus present

Supra-gingival calculus covering not more than 1/3rd of the exposed tooth surface

Supra-gingival calculus covering more than 1/3rd but not more than 2/3rd of the exposed tooth surface or presence of individual flecks of sub-gingival calculus around the cervical portion of the tooth or both

Supra-gingival calculus covering more than 2/3rd of the exposed tooth surface or a continuous heavy band of subgingival calculus

Recession index (Miller)

Class I

The gingival recession does not extend to the mucogingival line, and there is no loss of interdental bone or soft tissue present. Complete root coverage can be achieved

Class II

Class

Class

IV

Ш



The gingival recession extends to or beyond the mucogingival line, and there is no loss of interdental bone or soft tissue present. Complete root coverage can be achieved



The gingival recession extends to or beyond the mucogingival line with bone or soft tissue loss in the interdental area or malpositioning of teeth. Partial root coverage can be achieved

The gingival recession extends to or beyond the mucogingival line with severe bone or soft tissue loss in the interdental area and/or severe tooth malpositioning. No root coverage can be expected

Recession index (Cairo)

RT1	Gingival recession with no loss of interproximal attachment. Interproximal CEJ was clinically not detectable at both mesial and distal aspects of the tooth
RT2	Gingival recession associated with loss of interproximal attachment. The amount of interproximal attachment loss was less than or equal to the buccal attachment loss
RT3	Gingival recession associated with loss of interproximal attachment. The amount of interproximal attachment loss was higher than the buccal attachment loss

RT: Recession type, CEJ: Cementoenamel junction

Recession index (Cairo)



Periodontology

Dental stains

Dental stain

- Color and color perception
- Classification of tooth discoloration:
- o Intrinsic discoloration
- o Extrinsic discoloration
- o Internalized discoloration
- The mechanisms of tooth discoloration
- Prevention
- Treatment approaches

Colour and colour perception

- Teeth are composed of a number of colours and a gradation of colour from the gingival margin to the incisal edge of the tooth.
 The gingival margin often has a darker appearance because of the close approximation of the dentine below the enamel.
- -In most people canine teeth are darker than central and lateral incisors.
- -Younger people have lighter teeth, particularly in the primary dentition.
- -Teeth become darker as a physiological age change by:-
- 1-the laying down of secondary dentine.
- 2- incorporation of extrinsic stains .
- 3-gradual wear of enamel allowing a greater influence on colour of the underlying dentine.
- -Tooth wear and gingival recession can directly or indirectly affect tooth colour.





Colour and colour perception

- The viewing conditions are extremely important and variables such as :-

light source, time of day, surrounding conditions and the angle the tooth is viewed.

Three sources of light in a dental surgery:

natural, fluorescent and incandescent.

Incandescent light will accentuate the red-yellow end of the spectrum and weaken the blue end.

Conversely, a fluorescent light source has more energy towards the blue-green end of the spectrum and accentuates these colours accordingly.





Colour and colour perception

Specially developed colour-corrected lights are available which help to reduce the effects of metamerism,by providing even colour distribution.

Natural sunlight varies in its colour, at noon the sky appears blue.

Early morning and late evening sunlight has a red-orange tinge as the shorter wavelength blue light is scattered by the atmosphere and only red and orange rays penetrate.

Disagreement between dentists in shade matching the same tooth has been documented.

Colour can be described terms :-.

Hue is the descriptive term to enable one to distinguish between different families of colour, for example reds, blues and greens.

Value is the relative lightness and darkness of a colour on a scale from black to white.

Chroma is the degree of colour saturation.

A fourth dimension in the form of **opacity/translucency**.



Stains

Pigmented deposits on tooth surface are called stains. Discoloration of teeth can occur in three different ways:

- Stains adhering directly to tooth surface.
- Stains contained within the calculus and soft deposits.

• Stains incorporated within the tooth structure or restorative material.

Classification of tooth discolouration

The coronal portion of the tooth consists of enamel, dentine and pulp. Any change to these structures is likely to cause an alteration in the outward appearance of the tooth .

The appearance of tooth colour is dependent

on the quality of the reflected light.

Classification of Stains

Based on location

A-Extrinsic stains:

They occur on the external surface of the tooth and may be removed by procedures like tooth-brushing, scaling and/or polishing.

B. *Intrinsic stains:*

They occur within the tooth substance and cannot be removed by scaling and polishing.

Classification of Stains

Based on source

A-Exogenous:

They develop or originate from sources outside the tooth.

It may be extrinsic and stay on the outer surface of tooth or intrinsic and become incorporated within the tooth.

B-*Endogenous*:

They develop or originate from within the tooth. They are always intrinsic and usually are discolorations of dentin reflected through enamel.

Intrinsic discolouration

It occurs following a change to the structural composition or thickness of the dental hard tissues.

The normal colour of teeth is determined by the blue, green and pink tints of the enamel and is reinforced by the yellow through to brown shades of dentine beneath.

A no. of metabolic diseases and systemic factors are known to affect the developing dentition and cause discolouration as a consequence.

Local factors such as injury are also recognised.

- 1. Alkaptonuria
- 2. Congenital erythropoietic porphyria
- 3. Congenital hyperbilirubinaemia
- 4. Amelogenesis imperfect
- 5. Dentinogenesis imperfecta
- 6. Tetracycline staining
- 7. Fluorosis

- 8. Enamel hypoplasia
- 9. Pulpal haemorrhagic products
- 10. Root resorption
- 11. Ageing

The mechanisms of tooth discolouration

Intrinsic tooth discolouration

1. Alkaptonuria:

This inborn error of metabolism results in incomplete metabolism of tyrosine and phenylalanine,which promotes the build up of homogentisic acid.

This affects the permanent dentition by causing a brown discolouration.

2. Congenital erythropoietic porphyria:

This is a rare, metabolic disorder in which there is an error in porphyrin metabolism leading to the accumulation of porphyrins in bone marrow, RBC , urine, faeces and teeth.

The affected teeth show a red fluorescence under UV light.



Congenital erythropoietic porphyria

Intrinsic tooth discolouration

3. Congenital hyperbilirubinaemia:

The breakdown products of haemolysis will cause a yellow-green discolouration.

Mild neonatal jaundice is relatively common.

Deposition of bile pigments in the calcifying dental hard tissues.

4. Amelogenesis imperfecta:

In this hereditary condition, enamel formation is disturbed with regard to mineralization or matrix formation .

The appearance depends upon the type of amelogenesis imperfecta, varying from the relatively mild hypomature 'snow-capped' enamel to the more severe hereditary hypoplasia with thin, hard enamel which has a yellow to yellow-brown appearance.



Amelogenesis imperfecta


5. Systemic syndromes:

Defects in enamel formation may also occur in a no.of systemically involved clinical syndromes such as vitamin D dependent rickets, epidermolysis bullosa and pseudo-hypoparathyroidism.

In epidermolysis bullosa there is pitting of the enamel possibly caused by vesiculation of the ameloblast layer.

The effect of these conditions depends on disease activity during the development of the dentition and is usually a minor element.

6. Dentinogenesis imperfecta:

Dentine defects may occur genetically or through environmental influences.

The genetically determined dentine defects may be in isolation or associated with a systemic disorder.

Dentinogenesis imperfecta II

Both dentitions are affected, the primary dentition more affected.

The teeth are usually bluish or brown in colour,.

The pulp chambers often become obliterated and the dentine undergoes rapid wear.

The enamel has chipped away to expose the amelo-dentinal junction.

Once the dentine is exposed, teeth rapidly show brown discolouration .

Dentinogenesis imperfecta I (ass. with osteogenesis imperfecta,)

The enamel is much less prone to fracture, the pulp chamber is seldom occluded by dentine

Dentinogenesis imperfecta (type III)

Multiple pulpal exposures occur in the primary dentition.

Radiographically, the teeth may take on the appearance of 'shell teeth'.



Dentinogenesis imperfecta





7. Dentinal dysplasias: .

It effects the primary and secondary dentition.

Both dentition are of normal shape and form but may have an amber translucency.

Radiographically the teeth have short roots with conical apical constrictions.

There is brown discolouration of the teeth.

8. Tetracycline staining:

Systemic administration of tetracyclines during development is associated with deposition of tetracycline within bone and the dental hard tissues.

Tetracycline have the ability to form complexes with calcium ions on the surface of hydroxy apatite crystals within bone and dental tissues.

Teeth affected by tetracycline have a yellowish or brown-grey appearance which is worse on eruption and diminishes with time.

Exposure to light changes the colour to brown, the anterior teeth dentine are particularly more susceptible to colour changes .



9. Fluorosis:

This may arise endemically from naturally occurring water supplies or from fluoride delivered in mouthrinses, tablets or toothpastes as a supplement.

The severity is related to age and dose, with the primary and secondary dentitions both being affected in endemic fluorosis.

The enamel is often affected and may vary from areas of flecking to diffuse opacious mottling.

The colour of the enamel ranges from chalky white to a dark brown/black appearance.

The brown/black discolouration is post-eruptive and probably caused by the internalisation of extrinsic stain into the porous enamel.

Fluoride only causes fluorosis in concentrations of greater than 1 ppm in drinking water.



Dental fluorosis



Fluorosis

10. Enamel hypoplasia:

It may be localised or generalised.

The most common localised cause of enamel hypoplasia is likely to occur following trauma or infection in the primary dentition.

Disturbance of the developing tooth germ may occur in a large no.of foetal or maternal conditions eg maternal vitamin D deficiency, rubella infection, drug intake during pregnancy and in paediatric hypocalcaemic conditions.

There may be pitting or grooving which predisposes to extrinsic staining of the enamel in the region of tooth disturbed, often then becoming internalised .

11-Pulpal haemorrhagic products:

The discolouration of teeth following severe trauma was considered to be caused by pulpal haemorrhage.

Haemolysis of the RBCs would follow and release the haem group to combine with the pulpal tissue to form black iron sulphide by combining with hydrogen sulfide.

Pink discoloration of crown is seen after immediate trauma which turns orange, then blue, then brown and finally brown to black.

In the absence of infection, the release of iron from the protoporphyrin ring is unlikely.

It has been shown that the pinkish hue seen initially after trauma may disappear in 2 to 3 months if the tooth becomes revascularised.



Haemorrhagic products in a non-vital central

12. Root resorption:

It is often clinically asymptomatic,

The initial presenting feature is a pink appearance at the amelo-cemental junction.

Root resorption always begins at the root surface, either from the pulpal or periodontal aspect, as internal or external root resorption respectively.

It can be difficult to locate a resorptive cavity on radiograph until it reaches a certain size .

13. Ageing:

The natural laying down of secondary dentine affects

the light-transmitting properties of teeth resulting in a gradual darkening of teeth with age.

14-Erythroblastic fetalis:-

(Rh incompatibility between mother and foetus) characterized by breakdown of an excessive number of RBC .

Degradation of RBC causes intrinsic staining of dentin of developing teeth.

Jaundice results in staining of dentin bluish green or brown primary teeth by bilirubin.



Extrinsic discolouration

1- compounds which are incorporated into the pellicle and produce a stain as a result of their basic colour.

2- compounds which lead to staining caused by chemical interaction at the tooth surface.

Tobacco smoking and chewing are known to cause staining as well as beverages such as tea and coffee.

The colour seen on the tooth is thought to be derived from polyphenolic compounds present in the food.

Indirect extrinsic tooth staining is associated with cationic antiseptics and metal salts.

The agent is without colour or a different colour from the stain produced on the tooth surface.

Extrinsic tooth discolouration has usually been classified according to its origin, whether metallic or non-metallic.

Extrinsic discolouration

Non-metallic stains:

The non-metallic extrinsic stains are adsorped onto tooth surface deposits such as plaque or the acquired pellicle.

The possible aetiological agents include dietary components, beverages, tobacco, mouthrinses and other medicaments.

Chromogenic bacteria have been cited in children.

Particular colours of staining are said to be associated with certain mouths, for instance, green and orange in children with poor oral hygiene and black/brown stains in children with good oral hygiene and low caries experience.

The staining effect of prolonged rinsing with chlorhexidine mouthrinses and quarternary ammonium compounds used in mouthrinses is of considerable interest to the dental profession.

Extrinsic discolouration

Metallic stains: Extrinsic staining of teeth may be associated with occupational exposure to metallic salts and with a number of medicines containing metal salts.

The characteristic black staining of teeth in people using iron supplements and iron foundry workers.

Copper causes a green stain in mouthrinses containing copper salts and in workers in contact with the metal in industrial circumstances.

A no. of other metals have associated colours such as potassium permangenate producing a violet to black colour when used in mouthrinses; silver nitrate salt used in dentistry causes a grey colour, and stannous fluoride causes a golden ,manganese causes black stain; mercury causes greenish black stain; nickel causes green stain and silver causes black stains brown discolouration.

Mechanism of stain production was related to the production of the sulphide salt of the particular metal involved.

Extrinsic Stains: by colour

1. *Brown stain*: It is thin, translucent, acquired, usually bacteria free, pigmented pellicle.

It is seen in individuals who do not brush adequately or who use dentifrice with inadequate cleansing action.

It is commonly seen on the buccal surface of maxillary molars and lingual surface of mandibular anterior teeth.

The brown color is due to presence of tannin.

2. *Tobacco stain*: It is tenacious dark brown or black deposit accompanied by brown discoloration of tooth substance.

It is commonly seen on the lingual surfaces of teeth.

Staining result from coal tar products and from penetration of pits and fissures, enamel and dentin by tobacco by-products.

The degree of staining is not necessarily proportional to amount of tobacco consumed, but depends to a considerable degree on pre-existent acquired coatings.

Extrinsic Stains

3. *Black stain*: It occurs as a thin black line on the facial and lingual surfaces of teeth along the gingival margin and as diffuse patches on proximal surfaces. -firmly attached and tends to recur after removal.

-seen more common in women, children and individuals with excellent oral hygiene.

-caused by chromogenic bacteria namely *actinomyces species*, *Prevotella melaninogenicus*.

4. Green stain:

Green or greenish yellow stain is commonly seen in children.

It is considered to be stained remnants of enamel cuticle.

-seen on gingival half of maxillary anterior teeth.

-more frequent in boys (65%) than girls (63%).

-caused by fluorescent bacteria and fungi such as penicillium and aspergillus.

Extrinsic Stains

5. *Orange stain*: It is seen on both facial and lingual surfaces of anterior teeth caused by chromogenic bacteria: *Serratia marcescens* and *Flavobacterium*.

6 *Chlorhexidine (CHX)stains*: Brown color staining of teeth and tongue were noted following the use of CHX mouthrinse.

Intensity of staining does not depend on concentration of CHX rinse. Mechanisms :

– Degradation of CHX resulting in the formation of parachloraniline

 Catalysis of browning reaction of carbohydrates and amino acids by CHX.

– Denaturation of proteins resulting in the formation of sulphides.

– Precipitation of anionic dietary chromogens.

Internalised discolouration is the incorporation of extrinsic stain within the tooth substance(enamel and dentin) following dental development.

It occurs in enamel defects and in the porous surface of exposed dentine.

The routes by which pigments may become internalised are:

1. Developmental defects

2. Acquired defects

- a) Tooth wear and gingival recession
- b) Dental caries
- c) Restorative materials

Developmental defects:

They are most important defects under the 'intrinsic tooth discolouration' **as seen in previous part**.

They create their own colour change in the tooth caused by influences on light transmission through the dentine and enamel.

Extrinsic stains can penetrate into the enamel post-eruptively, either by increased enamel porosity, or the presence of enamel defects.

Such examples would include fluorosis and other enamel conditions result in in enamel hypoplasia or hypocalcification.

Alternatively, developmental defects may expose dentine either directly or later caused by early loss of enamel as in dentinogenesis imperfecta.

Chromogens are then able to enter the dentine directly or facilitated almost certainly by the tubule system.

2. Acquired defects:

Tooth wear and tear occur throughout life.

a)Tooth wear and gingival recession:.

Tooth wear is usually considered to be a progressive loss of enamel and dentine due to erosion, abrasion and attrition.

enamel thins \rightarrow darker tooth as(the colour of dentine becomes more apparent).

Once dentine is exposed \rightarrow chromogens enter the body of the tooth.

Physical trauma of enamel (cracks or loss) facilitate internalisation of extrinsic stains.

Although tooth wear occurs at the cervical area of teeth(where enamel is most thin), exposure of dentine is more likely caused by gingival recession \rightarrow dentin exposure and the increased potential for the uptake of chromogen into the tooth .

b) Dental caries: advance carious process can be recognised by changes in colour.



Gingival recession with dentine discolouration

c) Restorative materials

Some of the materials used in restorative dental treatment may have an effect on the colour of teeth.

Eugenol and phenolic compounds used during root canal therapy contain pigments(Black stains caused by silver containing root canal sealers).

The dark grey to black colour of dentine following the removal of a longstanding amalgam restoration because mercury was penetrating the dentinal tubules and reacting with sulphide ions.

Other causes:

a) Failure to remove all pulpal remnants.

b) Leakage from dental materials.e.g breakdown of silicate cements, composite resins can result in grayer and discoloured teeth,yellowish discoloration is caused by volatile oils and iodine-creates brown, yellow or orange stains.

g) Pins causes blue grayish stains.

Treatment modalities for discolored teeth

Removal of surface stains:

Superficial stains caused by tobacco chewing can be removed by scaling and polishing of teeth.

• Micro abrasions:

Enamel micro-abrasion technique associated with dental bleaching is an excellent and successful clinical technique for reestablishing esthetics of severe case of enamel fluorosis eliminating the use of dental restoration.

The procedure involves removal of thin layer of enamel surface followed by polishing with a fluoride prophylactic paste provides better surface smoothness and better hardness of the enamel.

Macro abrasions:

This technique removes the superficial layer of fluoride that displays the most unaesthetic colour and defective structure.

The procedure can eliminate deepest stains in the enamel

Treatment modalities for discolored teeth

Veneering:

Treatment of deeply penetrated intrinsic discoloration defects or considerable tooth structure loss entails placement of porcelain veneers.

Porcelain crowns.

Bleaching:

Bleaching is the most common esthetic treatment for adults. It needs careful diagnosis, case selection and treatment planning. It is least invasive, simplest ,least expensive means to lighten the discoloration of non vital and vital tooth.

LEC 5

Classification of

periodontal diseases and

conditions (2017)



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2021-2022

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CLASSIFICATION OF PERIODONTAL AND PERI-IMPLANT DISEASES AND CONDITIONS 2017

Periodontal Diseases and Conditions

Periodontal Health, Gingival Diseases and Conditions			Periodontitis			Other Conditions Affecting the Periodontium				
			Papapanou, Sanz et al. 2018 Consensus Rept link							
Chapple, Mealey, et al. 2018 Consensus Rept link			Jepsen, Caton et al. 2018 Consensus Rept link			Jepsen, Caton et al. 2018 Consensus Rept link				
Trombelli et al. 2018 Case Definitions link			Tonetti, Greenwell, Kornman. 2018 Case Definitions link			Papapanou, Sanz et al. 2018 Consensus Rept link				
Periodontal Health and Gingival Health	Gingivitis: Dental Biofilm-Induced	Gingival Diseases: Non-Dental Biofilm-Induced	Necrotizing Periodontal Diseases	Periodontitis	Periodontitis as a Manifestation of Systemic Disease	Systemic diseases or conditions affecting the periodontal supporting tissues	Periodontal Abscesses and Endodontic- Periodontal Lesions	Mucogingival Deformities and Conditions	Traumatic Occlusal Forces	Tooth and Prosthesis Related Factors

Peri-Implant Diseases and Conditions Berglundh, Armitage et al. 2018 Consensus Rept link Peri-Implant Health Peri-Implant Mucositis Peri-Implantitis Peri-Implant Soft and Hard Tissue Deficiencies

Why Do We Need A New Classification System?

- 1. To facilitate an international language for clinical communication.
- 2. To enable proper diagnosis and prognosis for patient communication and education.
- 3. To ensure implementation of appropriate treatment.
- **4.** To facilitate international population surveys of disease prevalence.
- To enable research into the etiology, pathogenesis, natural history and treatment strategies.



Major changes from previous classification

- 1- removal of the Aggressive and Chronic Periodontitis terms and replaced by a single category "Periodontitis".
- 2-periodontal health in the intact periodontium which is as important in the maintenance phase of patient management was introduced
- 3- introduced peri-implant health, peri-implant mucositis, peri-implantitis, and soft- and hardtissue deficiencies around implants.
- 4- Introduction of the term "gingival pigmentation"
- 5- Identifying smoking and diabetes as the major potential risk factors that can alter the staging of periodontal disease.
- 6- Recognition of "periodontitis as a manifestation of systemic disease" such as Papillon Lefèvre Syndrome.
- 7- Systemic conditions affecting the periodontium when not related to dental plaque will be considered as "Systemic Diseases or Conditions Affecting the Periodontal Supporting Tissues".
- 8- Management protocol of gingival recession based on the inter proximal attachment loss.
- 9- The term periodontal phenotype replaced the periodontal biotype and supracrestal attachment is the new term replacing the biological width.

Periodontal Health and Gingivitis: Consensus Report Chapple, Mealey, et al. 2018 Active link to consensus report Gingival Diseases: Case Definitions and Diagnostic Considerations

Trombelli, Tatakis, et al. 2018 Active link to case definitions

PERIODONTAL HEALTH, GINGIVAL DISEASES/CONDITIONS

1. Periodontal health and gingival health

Lang & Bartold 2018 link

- a. Clinical gingival health on an intact periodontium
- b. Clinical gingival health on a reduced periodontium
 - i. Stable periodontitis patient
 - ii. Non-periodontitis patient

2. Gingivitis – dental biofilm-induced

Murakami et al. 2018 link

- a. Associated with dental biofilm alone
- b. Mediated by systemic or local risk factors
- c. Drug-influenced gingival enlargement

3. Gingival diseases – non-dental biofilm induced

Holmstrup et al. 2018 link

- a. Genetic/developmental disorders
- b. Specific infections
- c. Inflammatory and immune conditions
- d. Reactive processes
- e. Neoplasms
- f. Endocrine, nutritional & metabolic diseases
- g. Traumatic lesions
- h. Gingival pigmentation

Periodontal health and gingival health:

Clinical gingival health on an intact periodontium

- Clinical gingival health on an intact periodontium is characterized by:
- absence of bleeding on probing, erythema and edema, patient symptoms,
- Absence of attachment and bone loss.
- Physiological bone levels range from 1.0 to 3.0 mm apical to the cemento-enamel junction.





2-3 mm

в



Healthy alveolar crest, normal lamina dura, and periodontal ligament space on a periapical image.



clinical gingival health on a reduced periodontium

Stable periodontitis patient Non-periodontitis patient (e.g. recession, crown lengthening)

- Clinical gingival health on a reduced periodontium is characterized by
- an absence of bleeding on probing, erythema, edema and patient symptoms in the presence of reduced clinical attachment and bone levels.
- However, it should be recognized that successfully treated and stable periodontitis patients remain at increased risk of recurrent progression of periodontitis.
- In non-periodontitis patients, there is no current evidence for increased risk of periodontitis.

:dental biofilm induced gingivitis A-Associated with bacterial dental biofilm only

- Dental plaque biofilm-induced gingivitis is defined at the site level as "an inflammatory lesion resulting from interactions between the dental plaque biofilm and the host's immune inflammatory response, which remains contained within the gingiva and does not extend to the periodontal attachment (cementum, periodontal ligament and alveolar bone).
- Such inflammation remains confined to the gingiva and does not extend beyond the mucogingival junction and is reversible by reducing levels of dental plaque at and apical to the gingival margin".



:dental biofilm induced gingivitis

B- Mediated by systemic or local risk factors

- Local risk factors (predisposing factors)
- Local risk factors for gingivitis are those that encourage plaque accumulation at a specific site by either inhibiting its removal during daily oral hygiene practices, and/or creating a biological place that encourages increased plaque accumulation.
- These include:

1-retention factors (tooth anatomical factors and restoration

2-Oral dryness(xerostomia) drug or disease









:dental biofilm induced gingivitis Mediated by systemic or local risk factors

- Systemic risk factors (modifying factors)
- Systemic risk or modifying factors are those characteristics present in an individual, which negatively influence the immune-inflammatory response to a given dental plaque biofilm burden, resulting in exaggerated or "hyper" inflammation.
- Examples include:
- Smoking induce microvascular vasoconstriction and fibrosis. This can mask clinical signs of gingivitis, such as bleeding on probing, despite a significant underlying pathological inflammatory cell infiltrate
- Metabolic factors hyperglycemia in people with or without diabetes activate various proinflammatory mediator cascades
- Nutritional factors Severe Vitamin C deficiency, compromised antioxidant micronutrient defenses to oxidative stress and also negatively impacts collagen synthesis, resulting in weakened capillary blood vessel walls and a consequent propensity to enhanced gingival bleeding.
Systemic risk factors (modifying factors)

- Pharmacological agents This may include drugs that:
- reduce salivary flow,
- impact endocrine function
- that may induce gingival enlargement and pseudo-pocketing.
- Elevations in sex steroid hormones :
- at puberty,
- during pregnancy,
- or following medication with first generation oral contraceptives
- may modify the gingival inflammatory response. Complex biological reactions within the gingival tissues result from such elevated sex steroid levels and generate more than expected inflammation, in response to relatively small levels of plaque.
- Hematological conditions particular blood malignancies such as leukemia or pre-malignant conditions such as myelodysplasia associated with signs of excess gingival inflammation in the absence of excessive plaque biofilm accumulation
- Signs include swollen, purple or occasionally pale gingiva due to leukemic cell infiltration, gingival bleeding that is inconsistent with levels of dental plaque biofilm accumulation, due to thrombocytopenia and/or clotting-factor deficiencies



:dental biofilm induced gingivitis C-DRUG -INFLUENCED GINGIVAL ENLARG EMENTS

example of drug induced gingival enlargement

- antiepileptic drugs phenytoin and sodium valproate,
- certain calcium channel-blocking drugs (e.g., nifedipine, verapamil, diltiazem, amlodipine, felodipine),
- immunoregulating drugs (e.g., ciclosporine)
- For drug-influenced gingival conditions, plaque bacteria in conjunction with the drug are necessary to produce a gingival response

The common clinical characteristics of drug-influenced gingival enlargements include:

- a tendency to occur more often in the anterior gingiva,
- a higher prevalence in younger age groups,
- onset within 3 months of use, that is usually first observed at the papilla,

- it can be found in a periodontium with or without bone loss, it is not associated with attachment loss or tooth mortality

:Case definition of gingivitis A-Definition of gingivitis in a patient with an intact periodontium

- A patient with an intact periodontium is diagnosed as follows
- Localized gingivitis, defined as a patient presenting with a BOP score ≥10% and ≤30%, without attachment loss and radiographic bone loss. This case may be associated with patient perception of bleeding gums. if any, impact on quality of life
- Generalized gingivitis, defined as a patient presenting with a BOP score > 30%, without attachment loss and radiographic bone loss. This case is often associated with patient perception of bleeding gums, and a modest impact on quality of life.

:Case definition of gingivitis B-Definition of gingivitis in a patient with a REDUCED periodontium

- A patient with a reduced periodontium but without a history of periodontitis (e.g. gingival recession, crown lengthening) and a BOP score ≥10%
- A GINGIVITIS CASE can also be graded as localized (BOP ≥10% and ≤30%) or generalized (BOP > 30%).
- The same criteria may also be applied to a patient with a reduced periodontium who has been successfully treated for periodontitis (periodontally stable patient), provided that no BOP positive sites show a probing depth ≥4 mm.
- Both localized and generalized gingivitis should be managed by:
- patient motivation,
- oral hygiene instruction,
- professional mechanical plaque removal, and implementation of self-performed mechanical plaque control, which may be supplemented by adjunctive use of antimicrobial/ anti-inflammatory oral care products.
- Dietary advice and tobacco counseling are recommended when indicated

- several non- plaque-induced pathologic lesions, which may in some instances be manifestations of a systemic condition or a medical disorder.
- They may also represent pathologic changes limited to gingival tissues.
- Although these lesions are not directly caused by plaque, their clinical course may be impacted by plaque accumulation and subsequent gingival inflammation

1 I GENETIC/DE VELOPMENTAL ABNORMALITIES

- Hereditary gingival fibromatosis (HGF)
- Clinically, gingival fibromatosis may present gingival overgrowth in various degrees. Hereditary gingival fibromatosis is a rare disease which may occur as an isolated disease or as part of a syndrome
- 2 | SPECIFIC INFECTIONS
- 2.1 | Bacterial origin
- Gingivitis caused by a specific bacterial infection may, however, arise due to a loss of homeostasis between non-plaque related pathogens and innate host resistance.
- Necrotizing periodontal diseases (*Treponema* spp., *Selenomonas* spp., *Fusobacterium* spp., *Prevotella* intermedia, and others)(IN THE NEXT LECTURE)
- Neisseria gonorrhoeae (gonorrhea)
- Treponema pallidum (syphilis)
- Mycobacterium tuberculosis (tuberculosis)
- Streptococcal gingivitis (strains of streptococcus)

- 2.2 | Viral origin
- Coxsackie viruses
- Coxsackie viruses may cause herpangina and hand-foot-and-mouth disease (synonym: vesicular stomatitis with exanthema). While herpangina does not involve gingiva, hand-foot-and-mouth disease is a common contagious vesicular viral disease affecting skin and oral mucosa including gingiva. The lesions are primarily seen in children







Non-plaque-induced gingival diseases Viral origin | 2.2

- herpes viruses including herpes simplex virus types 1 (HSV-1) and 2 (HSV-2).
- HSV-1 usually causes oral manifestations, in contrast to HSV-2, which is primarily involved in anogenital infections and only occasionally in oral infections
- Primary herpetic infection typically occurs in infants and has an incubation period of 1 week. It may run an asymptomatic course in early childhood, but it may also give rise to gingivostomatitis with severe manifestations.
- A characteristic feature is the formation of few or many vesicles, which rupture, coalesce, and leave fibrin-coated ulcers often of irregular extension.
- Recurrent intraoral herpes simplex lesions typically occur in adults and have a much less dramatic course.
- As a result, they may remain undiagnosed or mistaken for aphthous ulcerations despite the fact that aphthous ulcers do not typically affect keratinized mucosa.



Non-plaque-induced gingival diseases Viral origin | 2.2

Varicella-zoster virus

- The primary infection of varicella-zoster virus causes varicella (chicken pox), which occurs mainly in children.
- Later reactivation of the virus in adults causes herpes zoster (shingles) with unilateral lesions following the distribution of an infected nerve.
- If the second or third branch of the trigeminal nerve is involved, skin lesions may be associated with intraoral lesions, including gingival lesions, and intraoral lesions may occur alone.
- Initial symptoms are pain and paresthesia, which may be present before lesions appear. The initial lesions are vesicles, which soon rupture and leave fibrin-coated small ulcers, often coalescing to irregular forms

- Human papilloma virus (HPV)
- More than 100 types of HPV have been identified, and at least the following 25 types have been detected in oral lesions
- The benign oral lesions, associated with HPV infection, include squamous cell papilloma, AND focal epithelial hyperplasia, and they appear to be associated with different distinct HPV subtypes. Oral benign HPV lesions are mostly asymptomatic, and may persist or regress spontaneously

focal epithelial hyperplasia

Non-plaque-induced gingival diseases Fungal origin | 2.3

- A number of fungi may give rise to oral infections, the most common candidosis, histoplasmosis, aspergillosis,
- Oral mycoses can cause acute, chronic, and mucocutaneous lesions.
 Candidosis is the most common mouth mycosis, while histoplasmosis and aspergillosis are less common
- The most common fungal infection of the oral mucosa is candidosis mainly caused by C. albicans. C. albicans is a normal commensal organism of the oral cavity but also an opportunistic pathogen.
- The most common clinical characteristic of gingival candidal infection is redness of the attached gingiva, often with a granular surface.

 3 | INFL AMMATORY AND IMMUNE CONDITIONS AND LESIONS

3.1 | Hypersensitivity reactions

- Contact allergy Oral mucosal manifestations of hypersensitivity (allergy) are very uncommon. reactions may be due to dental restorative materials, dentifrices, mouthwashes, and foods and are most often type IV hypersensitivity reactions.
- Plasma cell gingivitis is an uncommon inflammatory condition usually affecting the anterior maxillary gingiva and of uncertain etiology.
- Erythema multiforme (EM) is an uncommon, self-limiting, acute immune-inflammatory disorder of the oral mucosa. The etiology of EM is unclear in most patients, but it appears to be an immunologic hypersensitivity reaction mediated by T-lymphocytes

- 3 | INFL AMMATORY AND IMMUNE CONDITIONS AND LESIONS
- 3.2 | Autoimmune diseases of skin and mucous membranes

Pemphigus vulgaris (PV)

is an autoimmune vesiculo-bullous disease of skin and mucous membranes. Involvement of the oral mucosa is common, and in about 54% of cases, the oral cavity has been reported to be the primary site of involvement. The disease is characterized by intraepithelial bullae in skin and mucous membranes due to auto-antibodies directed against desmosome-associated protein antigens (desmoglein- 3). Oral mucosal lesions, including gingival lesions, may precede skin involvement. gingival localization of PV usually manifests as desquamative gingivitis and/or as vesiculo-bullous lesions of the free and attached gingiva; early lesions only rarely appear as extensive erythema and erosions

Pemphigoid

is a group of mucocutaneous disorders caused by autoantibodies toward antigens of the basement membrane, resulting in detachment of the epithelium from the connective tissue. If only mucous membranes are affected, the term mucous membrane pemphigoid (MMP) is often used. Any area of the oral mucosa may be involved in MMP, but the main clinical manifestation is desquamative lesions of the gingiva presenting as intensely erythematous areas.

Lichen planus

Lichen planus is a common mucocutaneous disease with frequent manifestation on the gingiva. Oral involvement alone is common, and concomitant skin lesions in patients with oral lesions have been found in 5% to 44% of the cases .**The major characteristic of this disease is an inflammatory reaction toward an unidentified antigen in the basal epithelial layer/ basement membrane zone**. The disease may be associated with severe discomfort. Because it has been shown to possess a premalignant potentialThe lesions, usually bilateral, often involve the gingiva and present as desquamative gingivitis causing pain and discomfort during eating and toothbrushing

Lupus erythematosus (LE)

is a group of autoimmune disorders characterized by autoantibodies to various cellular constituents, including extractable nuclear antigens and cytoplasmic membrane components. Two major forms are described: discoid LE (DLE) and systemic LE (SLE), which may involve a range of organ systems. DLE is a mild chronic form, which involves skin and mucous membranes, sometimes including the gingiva as well as other parts of the oral mucosa. The typical lesion presents as a central atrophic area with small white dots surrounded by irradiating fine white striae . Eight percent of patients with DLE develop SLE, and ulcerations may be a sign of SLE. The characteristic dark red "butterfly" skin lesions are photosensitive, scaly, erythematous macules located on the bridge of the nose and the cheeks. The systemic type may also include skin lesions located on the face, but they tend to spread over the entire body.

3 | INFL AMMATORY AND IMMUNE CONDITIONS AND LESIONS

• 3.3 | Granulomatous inflammatory conditions (orofacial granulomatosis)

Persistent enlargement of the soft tissues in the oral cavity as well as the facial region can occur concomitant with various systemic conditions like tuberculosis, Crohn's disease (CD), and sarcoidosis. These changes are also seen as a typical symptom of the Melkersson- Rosenthal syndrome

• 4 | REACTIVE PROCESSES

- 4.1 | Epulides
- is a term often applied to exophytic processes originating from the gingiva. The term is non-specific and histopathology is the basis of a more specific diagnosis. Several of these processes are reactive lesions, i.e., non-neoplastic proliferations with very similar clinical appearance to benign neoplastic proliferations
- They are classified according to their histology. True epulides include:
- Fibrous epulis
- Calcifying fibroblastic granuloma
- Pyogenic granuloma (vascular epulis)
- Peripheral giant cell granuloma (or central)

• Fibrous epulis

Fibrous epulides (focal fibrous hyperplasia, irritation fibroma) are common exophytic smooth-surfaced pink masses of fibrous consistency attached to the gingiva. The size varies from small to large tumorlike processes with a diameter of several cm.

Calcifying fibroblastic granuloma

Calcifying fibroblastic granuloma (ossifying fibroid epulis, peripheral ossifying fibroma) occurs exclusively on the gingiva. The lesion, although usually smaller than 1.5 cm in diameter, can reach a larger size and rarely cause separation of the adjacent teeth and resorption of the alveolar crest

• Pyogenic granuloma

 The pyogenic granuloma (telangiectatic granuloma, pregnancy granuloma, pregnancy tumor, vascular epulis) is rather common and shows a striking predilection for the gingiva, which accounts for 75% of all cases . When occurring during pregnancy, the influence of female sex hormones may result in a biologic behavior distinct from other pyogenic granulomas.

 Peripheral giant cell granuloma (giant cell epulis, peripheral giant cell reparative granuloma) usually develops from the marginal gingiva. The swelling may be sessile or pedunculated, sometimes ulcerated, and the appearance may resemble pyogenic granulomas

• 5 NEOPLASMS

- 5.1 | Premalignant
- Leukoplakia

refers to a white lesion of the oral mucosa that cannot be characterized as any other definable lesion. It is a clinical diagnosis arrived at by exclusion in that all other potential causes of a white lesion have been ruled out or addressed. Lesions are generally asymptomatic and cannot be rubbed off. Approximately 20% of leukoplakic lesions demonstrate some degree of dysplasia or carcinoma upon biopsy and most oral cancers are preceded by a long-standing area of leukoplakia. As a result, leukoplakia can be considered a premalignant condition. The prevalence of malignant transformation in leukoplakia ranges from 0.13% to 34%. Lesions occur most frequently on the buccal mucosa, mandibular gingiva, tongue, and floor of the mouth.

Erythroplakia

• is the red counterpart of leukoplakia in the sense that it is a red lesion, which cannot be diagnosed as any other disease. Erythroplakia usually has a higher

- 5.2 | Malignant
- Squamous cell carcinoma
- Squamous cell carcinoma of the gingiva represents about 20% of intraoral carcinomas and occurs most frequently in the mandibular premolar and molar regions. Lesions commonly occur in edentulous areas, but they may also occur at sites in which teeth are present. Mobility of adjacent teeth is common, and invasion of the underlying alveolar bone is apparent in approximately 50% of cases. Gingival squamous cell carcinoma may mimic other oral lesions affecting the periodontium, most of which are reactive or inflammatory in nature.
- Leukemia
- Leukemias can be classified as acute- or chronic-based on their clinical behavior, and lymphocytic/ lymphoblastic or myeloid depending on their histogenetic origin. Oral lesions occur in both acute and chronic leukemia but are more common in the acute form. The signs and symptoms are varied. Bacterial, viral, and fungal infections including candidosis, and herpes simplex infection may also be present
- Lymphoma
- Lymphoma is a general term given to tumors of the lymphoid system and represents the most common hematologic malignancy. Lymphoma may originate from B-lymphocyte and T-lymphocyte cell lines. There are two main types of lymphoma: Hodgkin lymphoma and non-Hodgkin lymphoma, the former being one-sixth as common as non-Hodgkin lymphoma. In contrast to non-Hodgkin lymphoma (Table 2), oral manifestations of Hodgkin lymphoma are extremely rare

6 | ENDOCRINE, NUTRITIONAL, AND METABOLIC DISEASES

- 6.1 | Vitamin deficiencies
- Vitamin C deficiency (scurvy) Ascorbic acid (vitamin C) is necessary for various metabolic processes in the connective tissue as well as in the formation of catecholamines.
- Clinically, scurvy is characterized by gingival bleeding and soreness, as well as by a depressed immune response. In gingival health, the concentration of ascorbic acid in gingival crevicular fluid is higher than in plasma

7 | TR AUMATIC LESIONS

• 7.1 | Physical/mechanical insults

- Frictional keratosis : Inappropriate toothbrushing can be injurious to the gingival tissues. Some patients believe they should actively brush the gingiva Limited physical trauma from brushing may result in gingival hyperkeratosis, a white leukoplakia-like lesion referred to as frictional keratosis
- Toothbrushing-induced gingival ulceration In cases of more violent trauma, toothbrushing damage varies from superficial gingival laceration to major loss of tissue resulting in gingival recession. Characteristic findings in these patients are extremely good oral hygiene, cervical tooth abrasion, and unaffected tips of the interdental papillae in the site of injury. The condition has been termed traumatic ulcerative gingival lesions. Inappropriate dental flossing may also cause gingival ulceration and inflammation primarily affecting the tip of the interdental papillae. The prevalence of such findings is unknown. Diagnosis of the lesion is based on clinical findings,
- Factitious injury (self-harm) Self-inflicted injury to the gingival tissue is usually seen in young patients, and the lesions may present unusual tissue damage in areas that can easily be reached by fingers and instruments

• 7.2 | Chemical (toxic) insults

- Etching
- Toxic chemical products may result in mucosal surface erosions, including reactions of the gingiva. Surface sloughing or ulceration may be related to the use of chlorhexidine, acetylsalicylic acid,cocaine, hydrogen peroxide,or to dentifrice detergents. These lesions are reversible and resolve after removing the toxic influence. Injury to the gingival tissue may also be caused by dentists' incorrect use of substances used for endodontic purposes that may be toxic to the gingiva, including paraformaldehyde or calcium hydroxide, which may give rise to inflammation, ulceration, and necrosis of the gingival tissue if the cavity sealing is insufficient.In most instances, the diagnosis is obvious from the combination of clinical findings and patient history

• 7.3 | Thermal insults

 Thermal burns of the gingiva may be prevalent due to a hurried lifestyle with intake of microwave-heated foods and drive-through coffee shops. Any part of the oral mucosa can be involved, including the gingiva. The lesion is erythematous with sloughing of a coagulated surface. Vesicles may also occur and sometimes the lesions present as ulceration, petecchia, or erosions, which may be painful. The clinical characteristics and the history are important for the correct diagnosis. Gingival injury due to cold has been described but appears to be very uncommon.

• 8 | GINGIVAL PIGMENTATION

• Gingival pigmentation/melanoplakia

• Oral pigmentation is associated with a variety of exogenous and endogenous factors including drugs, heavy metals, genetics, endocrine disturbances (Addison's disease), syndromes (Albright syndrome, Peutz-Jegher syndrome), and postinflammatory reactions.Physiologic pigmentation is usually symmetric, occurring on the gingiva, buccal mucosa, hard palate, lips, and tongue.

• Smoker's melanosis

• A primary etiologic factor in melanocytic pigmentation of the oral mucosa is cigarette smoking. Smoker's melanosis occurs most frequently on the mandibular anterior facial gingiva. Melanosis gradually improves or may completely resolve upon cessation of smoking.

Figure 4. Smoker's melanosis in a 30-year-old

• Drug-induced pigmentation (DIP)

DIP may be caused by the accumulation of melanin, deposits of drug or drug metabolites, synthesis of pigments under the influence of a drug, or deposition of iron following damage to the vessels. Quinine derivatives such as quinolone, hydroxyquinolone, and amodiaquine are antimalarial drugs that cause bluish grey or black mucosal pigmentation occurring most frequently on the hard palate including the palatal gingiva. Long-term use of minocycline is associated with pigmentation of the alveolar bone and teeth. When changes in bone are viewed through relatively thin overlying mucosa, the gingiva may appear grey and is seen primarily in the maxillary anterior region. True minocycline- induced soft tissue pigmentation is much less common and occurs primarily on the tongue, lip, buccal mucosa, and gingiva

Amalgam tattoo

• Pigmentation of the oral mucosa due to amalgam is frequently seen in the gingiva and alveolar mucosa. The lesion is a well-defined bluish, blackish, or greyish discoloration, which is not elevated Radiographic imaging may demonstrate underlying amalgam debris.

The end thanks

LEC 6

Classification of

periodontal diseases and

conditions (2017)

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CLASSIFICATION OF PERIODONTAL AND PERI-IMPLANT DISEASES AND CONDITIONS 2017

Periodontal Diseases and Conditions

Periodontal Health, Gingival Diseases and			Periodontitis		Other Conditions Affecting the Periodontium					
Conditions		Papapanou, Sanz et al. 2018 Consensus Rept link								
Chapple, Mealey, et al. 2018 Consensus Rept link			Jepsen, Caton et al. 2018 Consensus Rept link		Jepsen, Caton et al. 2018 Consensus Rept link					
Trombelli et al. 2018 Case Definitions link			Tonetti, Greenwell, Kornman. 2018 Case Definitions link		Papapanou, Sanz et al. 2018 Consensus Rept link					
Periodontal Health and Gingival Health	Gingivitis: Dental Biofilm-Induced	Gingival Diseases: Non-Dental Biofilm-Induced	Necrotizing Periodontal Diseases	Periodontitis	Periodontitis as a Manifestation of Systemic Disease	Systemic diseases or conditions affecting the periodontal supporting tissues	Periodontal Abscesses and Endodontic- Periodontal Lesions	Mucogingival Deformities and Conditions	Traumatic Occlusal Forces	Tooth and Prosthesis Related Factors

Peri-Implant Diseases and Conditions Berglundh, Armitage et al. 2018 Consensus Rept link Peri-Implant Health Peri-Implant Mucositis Peri-Implantitis Peri-Implant Soft and Hard Tissue Deficiencies

)STAGING AND GRADING(STAGING (SEVERITY OF THE DISEASE) EVALUATED BY CLINICAL ATTACHMENT LOSS OR BONE LOSS				
STAGE (1-2) MM ATTACHMENT LOSS		LESS THAN 15% BONE LOSS IN THE CORONAL 1/3		
STAG ii (3-4)MM ATTACHMENT LOSS		33%- 15% BONE LOSS IN THE CORONAL 1/3		
MM 5≥		BONE LOSS EXTEND TO THE MIDDLE 1/3 AND		

BYOND

BONE LOSS EXTEND TO

APICAL 1/3 AND BYOND

ATTACHMENT LOSS

MM 5≥

ATTACHMENT LOSS

WITH COMPLEXITY

GRADING RATE OF PROGRESSION EVALUATED BY BONE LOSS/ PT AGE

GRADE A 0.25<

GRADE B

)1- 0.25(

GRADE C

1>

Staging and Grading Periodontitis

The 2017 World Workshop on the Classification of Periodontal and Peri-Implant Diseases and Conditions resulted in a new Classification of periodontitis characterized by a multidimentional staging and grading system. The charts below provide an overview. Please visit perio.org/2017wwdc for the complete suite of reviews, case definition papers, and consensus reports.

Tables from Tonelli, Greenwell, Kornman. J Periodontal 2018; 89 (Suppl 1): \$159-\$172

MODERN PERIODONTICS Great Smiles Start with You

PERIODONTITS: STAGING

Staging intends to classify the severity and extent of a patient's disease based on the measurable amount of destroyed and/or damaged tissue as a result of Periodontitis and to assess the specific factors that may attribute to the complexity of long-term case management.

Initial stage should be determined using critical attachment loss (CAL). If CAL is not available, radiographic bone loss (RBL) should be used. Tooth loss due to periodontitis may modify stage definition. One or more complexity factors may shift the stage to a higher level. See **perio.org/2017wwdc** for additional information.

Periodontitis		Stage I	Stage II	Stage III	Stage IV	
	Interdental CAL (at site of greatest loss)	erdental CAL 1 - 2 mm		≥ 5mm	≥ 5mm	
Severity	RBL	Coronal third (<15%)	Coronal third (15-33%)	Extending the middle third of root and beyond	Extending the middle third of root and beyond	
	Tooth Loss (due to periodontitis)	No tooth loss	No tooth loss	≤ 4 teeth	≥ 5 teeth	
Complexity	Local	-Max. probing depth ≤ 4 mm -Mostly horizontal bone loss	-Max. probing depth ≤ 5 mm -Mostly horizontal bone loss	-Probing depths ≥ 6 mm -Vertical bone loss ≥ 3 mm -Furcation involvement Class II or III -Moderate ridge defects	Need for complex rehabilitation due to: - Masticatory dysfuntion - Secondary occlusal trauma (tooth mobility degree)	
Extent and Distribution	For each stage, desc Localized, Generalize	ribe extent as: ed; or Molar/ Incisor patterr				

Staging and Grading Periodontitis

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PERIODONTITS: GRADING

Grading aims to indicate the rate of periodontitis progression, responsiveness to standard therapy, and potential impact on systemic health.

Clinicians should initially assume grade B disease and seek specific evidence to shift to grade A and C. See **perio.org/2017wwdc** for additional information.

Progression			Grade A: Slow Rate	Grade B: Moderate Rate	Grade C: Rapid Rate
Primary criteria	Direct evidence of progression	Radiographic bone loss or CAL	No loss over 5 years	< 2mm over 5 years	≥2 mm over 5 years
Whenever available, direct evidence	Indirect evidence of progression	% bone loss / age	<0.25	0.25 to 1.0	> 1.0
snoula de asea.		Case phenotype	Heavy biofilm deposits with low levels of destruction	Destruction commensurate with biofilm deposits	Destruction exceeds expectations given biofilm deposits; specific clinical patterns suggestive of periods of rapid progression and/or early onset disease
Grade modifiers	Risk factors	Smoking	Non-smoker	<10 cigarettes/day	10 cigarettes/day
		Diabetes	Normoglycemic/no diagnosis of diabetes	HbA1c < 7.0% in patients with diabetes	HbA1c ≥ 7.0% in patients with diabetes

MODERN

Great Smiles Start with You

PERIODO

Periodontitis Consensus Report Papapanou, Sanz et al. 2018 Active link to consensus report

FORMS OF PERIODONTITIS

1. Necrotizing Periodontal Diseases

Herrera et al. 2018 link

- a. Necrotizing Gingivitis
- b. Necrotizing Periodontitis
- c. Necrotizing Stomatitis

2. Periodontitis as Manifestation of Systemic Diseases

Jepsen, Caton et al. 2018 Consensus Rept link Albandar et al. 2018 link Classification of these conditions should be based on the primary systemic disease according to the International Statistical Classification of Diseases and Related Health Problems (ICD) codes

3. Periodontitis

Fine et al. 2018 link	Needleman et al. 2018 link	Billings et al. 2018 link
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a. Stages: Based on Severity¹ and Complexity of Management² Stage I: Initial Periodontitis

Stage II: Moderate Periodontitis

Stage III: Severe Periodontitis with potential for additional tooth loss Stage IV: Severe Periodontitis with potential for loss of the dentition

- b. Extent and distribution³: localized; generalized; molar-incisor distribution
- c. Grades: Evidence or risk of rapid progression⁴, anticipated treatment response⁵
 - i. Grade A: Slow rate of progression
 - ii. Grade B: Moderate rate of progression
 - iii. Grade C: Rapid rate of progression

Staging and Grading of Periodontitis: Framework and Proposal of a New Classification and Case Definition Tonetti, Greenwell, Kornman 2018 *Active link to case definitions*

¹ Severity: Interdental clinical attachment level (CAL) at site with greatest loss; Radiographic bone loss & tooth loss

² Complexity of management: Probing depths, pattern of bone loss, furcation lesions, number of remaining teeth, tooth mobility, ridge defects masticatory dysfunction

 $^{^3}$ Add to Stage as descriptor: localized <30% teeth, generalized \geq 30% teeth

⁴ Risk of progression: direct evidence by PA radiographs or CAL loss, or indirect (bone loss/age ratio)

⁵ Anticipated treatment response: case phenotype, smoking, hyperglycemia

Necrotizing periodontal diseases

Necrotizing gingivitis Necrotizing periodontitis Necrotizing Stomatitis

- Necrotizing gingivitis (NG), necrotizing periodontitis (NP), and necrotizing stomatitis (NS) are the most severe inflammatory periodontal disorders caused by plaque bacteria. The necrotizing diseases usually run an acute course and therefore the term acute is often included in the diagnoses. They are rapidly destructive and debilitating, and they appear to represent various stages of the same disease process.
- The necrotizing periodontal diseases have been mentioned under several names, including: "ulceromembranous gingivitis", "acute necrotizing ulcerative gingivitis" (ANUG), "Vincent's gingivitis" or "Vincent's gingivostomatitis", "necrotizing gingivostomatitis", and "trench mouth"
Necrotizing gingivitis

UT

Clinical characteristics

 ulcerated and necrotic papillae and gingival margins, giving a punched-out appearance. The ulcers are covered by a yellowish–white or grayish slough, which has been termed a "pseudomembrane". It consists primarily of fibrin and necrotic tissue with leukocytes, erythrocytes, and masses of bacteria.

-The necrotizing lesions develop rapidly and are **painful**, but in the initial stages, when the necrotic areas are relatively few and small, pain is usually moderate. Severe pain is often the chief reason for patients seeking treatment.

-Bleeding is readily provoked on removal of the sloughed material and exposure of the ulcerated underlying connective tissue. Bleeding may also start spontaneously as well as in response to even gentle touch.

OTHER FEATURE

- Lymphadenopathy is infrequent & related to severity of disease
- fetor ex ore(Malodor) not always noted
- fever & malaise (common)

Episodes will usually resolve within few days after receiving









Etiology

- <u>Fusoform</u>
- Prevotella intermedia
- <u>Spirochetes</u>
- treated by elimination of plaque and with Antibiotic & mechanical debridment.

• Predisposing factors

1) Psychological stress High relation between NG & stress due to Reduction in O.H.measures, Nutrition, Tobacco smoking

2) AIDS seen with NG that progress to NUP & NUS as generalized Immune suppression lead to defects in Neutrophil function, AntiBody response & Lymphocytes mitogenesis

3)Malnutrition children related to poor nutritional status especially protein intake & 2ndary to viral infection as measles that progress to lethal infection named (NOMA)(CONCRUM ORIS) which is RARE and assoc.with elevated cortisol levels & reduced levels of zinc & amino acid in children previously infected with Herpes virus or measles

Other predisposing factors

Tobacco smoking

Pre-existing gingivitis

Trauma

Recurrence related to pericoronitis at impacted 1/3 molar & deep overbite in anterior teeth

Excessive work or following debilitating diseases



Smear of the pseudomembrane In addition to dead cells granulocytes (PMN) and fusiform bacteria (FUS), enormous numbers of Spirochetes (SPIR) are visible Electron micrograph showing a phagocytosing neutrophil (N) close to the surface of a sequestrum (C), covered by numerous microorganisms including .spirochetes (S) and rods (R). Bar = 1 μ m



Necrotizing periodontitis

- is an inflammatory process of the periodontium characterized by
- presence of necrosis/ulcer of the
 interdental papillae, gingival
 bleeding, halitosis, pain, and rapid
 bone loss.
- Other signs/symptoms associated with this condition may include pseudomembrane formation, lymphadenopathy, and fever.









Necrotizing stomatitis

- is a severe inflammatory condition of the periodontium and the oral cavity in which soft tissue necrosis extends beyond the gingiva and bone denudation may occur through the alveolar mucosa, with larger areas of osteitis and formation of bone sequestrum. It typically occurs in severely systemically compromised patients. Atypical cases have also been reported, in which necrotizing stomatitis may develop without prior appearance of necrotizing gingivitis/periodontitis lesions.
- Association b/w malnutrition, poor O.H. & NUG with progression to sever destructive dis., necrosis & sequestration of bone
- (noma or concrum oris)
- Similar to NUP in AIDS pt.due to alteration of I.R. through malnutrition or systemic disease



Treatment

- Perform debridement under local anesthesia, including gentle scaling.
- Remove pseudomembrane, using cotton pellet dipped in 0.12% chlorhexidine.
- Provide the patient with oral hygiene instructions and prescribe antibacterial mouthwash (0.12% chlorhexidine, b.i.d.) or peroxide hydroxyl mouth rinse (b.i.d.).
- Tell the patient to control pain with analgesics (ibuprofen 400–600 mg, t.i.d. or acetaminophen 750 mg, t.i.d.).
- Provide patient counseling: ensure proper nutrition, take vitamin supplements, avoid spicy foods, adopt appropriate fluid intake, increase sleep, decrease stress, and quit smoking, if possible.
- Prescribe antibiotics if signs of systemic involvement (e.g., fever, malaise, lymphadenopathy):

Amoxicillin: 500 mg, t.i.d for 7 days; or combination of amoxicillin 250 mg and metronidazole 250 mg one tab of each t.i.d. for 7 days

- If the patient is immunocompromised (e.g. AIDS, HIV-positive, leukemia, cyclic neutropenia), it is important to follow-up with their physician when you prescribe antibiotics. The risk of super infection with oral Candida must be considered when using systemic antibiotics in immunocompromised patients. In those cases antifungal medication might be needed.
- Follow Up
- Immunocompromised patients should be treated in close communication with their physician. Depending on the degree of immunosupression, it may be advisable to perform only conservative, minimally invasive procedures and monitor them closely.
- Assess treatment outcomes in 24 hours, then every other day until all acute signs and symptoms are controlled. Frequent periodontal maintenance visits and meticulous oral hygiene are necessary as NUP may continue to progress rapidly.
- After the first consult or control of the acute stage, referral to a periodontist or oral surgeon is advised.
- Follow up with a comprehensive periodontal evaluation.

Periodontitis as a manifestation of systemic disease

- Endocrine Disorders and hormonal changes
 - Hematologic disorders and immune deficiencies
 - Genetic disorders
 - Stress and psychosomatic disorders
 - Nutritional influences
 - Medications
 - Other Systemic Conditions.

Periodontitis as a manifestation of systemic disease

• 1) ENDOCRINE DISORDERS AND HORMONAL CHANGES.

1) DIABETES MELLITUS

- is a clinically and genetically heterogeneous group of metabolic disorders manifested by abnormally high levels of glucose in the blood.
- The hyperglycemia is the result of a deficiency of insulin secretion caused by pancreatic Beta cell dysfunction or of resistance to the action of insulin in liver and muscle or a combination of these.

CLINICAL SIGNS & SYMPTOMS AND DIABETIC COMPLICATIONS

- Polyuria (excessive urination)
- Polydipsia (excessive thirst)
- Polyphagia (excessive hunger)
- Unexplained weight loss
- Changes in vision
- Fatigue, weakness
- Irritability
- Nausea
- Dry mouth

COMPLICATIONS OF DIABETES MELLITUS

- Retinopathy
- Nephropathy
- Neuropathy
- Macrovascular disease
- Altered wound healing
- <u>Periodontal disease</u> is the sixth complication of diabetes mellitus
- Ketoacidosis (usually associated with severe hyperglycemia in Type I diabetes)
- Hyperglycemic hyperosmolar state
- Hypoglycemia

ORAL MANIFESTATIONS OF DIABETES

- Xerostomia
- Greater susceptibility of oral tissues to trauma
- More opportunistic infections (e.g., Candidiasis)
- Greater accumulation of plaque, greater risk of caries
- Greater susceptibility to periodontal disease
- Greater risk of developing periodontal abscesses when periodontitis is present
- Delayed healing
- Oral paraesthesia, including burning mouth or tongue
- Altered taste sensations

EFFECTS OF DIABETES ON THE PERIODONTIUM

GINGIVITIS

- •Erythema, oedema,
- •Bleeding on probing,
- •Gingival exudates.
- PERIODONTITIS
- •Greater attatchment loss & bone loss
- Multiple periodontal abscess
- Loosened teeth

PATHOGENESIS OF PERIODONTAL DISEASE IN DIABETIC SUBJECTS

- Bacterial flora Few differences in the subgingival microflora between diabetic and non-diabetic patients with periodontitis
- HOST DEFENSE ALTERATIONS :
- The function of immune cells, including neutrophils, monocytes, and macrophages, is altered in diabetes
- Neutrophil adherence, chemotaxis, and phagocytosis are often impaired
- Monocytes from diabetic subjects produce elevated levels of TNF-a in response to antigens from *Porphyromonas gingivalis*
- Subjects with HbA1c levels over 8% had crevicular fluid levels of interleukin-1 beta (IL-1β) almost twice as high as subjects with HbA1c levels <8%
- The net effect of these host defense alterations in diabetes is an increase in periodontal inflammation, attachment loss, and bone loss.

ALTERATIONS IN CONNECTIVE TISSUE METABOLISM

- Impaired osseous healing and bone turnover
- Inhibition of osteoblastic cell proliferation and collagen production -reduced bone formation poor newly formed bone
- Increased rate of apoptosis of fibroblasts & osteoblasts

SO More severe periodontal attachment loss

Impaired wound healing Elevated gingival crevicular fluid glucose levels in diabetic individuals .
 spreading of fibroblasts
 Impaired wound healing and tissue turnover

Inhibiting attachment and

ENDOCRINE DISORDERS AND HORMONAL CHANGES.

2) HORMONAL CHANGES

- FEMALE SEX HORMONES Gingival alterations during Puberty ,pregnancy and menopause are associated with physiologic hormonal changes in the female patient. Gingiva in PUBERTY • Pronounced inflammation,bluish-red discolouration,edema and enlarged gingiva. • There is increased prevalence of gingivitis,bleeding,exudation from inflammed gingiva,but the crevicular fluid is not affected.
- GINGIVAL DISEASES IN PREGNANCY Pronounced base of bleeding Gingiva is bright red to bluish red • Marginal and interdental gingiva is edematous,pits on pressure and sometime presents rasberry like appearance • There is depression of maternal T-lymphocyte response. • Aggravation of gingivitis has been attributed principally to increased levels of progesterone • Increased crevicular fluid,pocket depth,mobility.
- HORMONAL CONTRACEPTIVES Aggravate the gingival response to local factors in a manner similar to that seen in pregnancy and when taken for more than 1.5 years, increase periodontal destruction.
- MENOPAUSE Females can develop gingivostomatitis Oral mucosa is dry and shiny, vary in color from abnormal paleness to redness, and bleed easily. • Dry burning sensation throughout the oral cavity • Extreme sensitivity to thermal changes; abnormal taste sensations described as salty, peppery or sour.

3) HYPERPARATHYROIDISM

- 25% to 50% of patients hyperparathyroidism has associated oral changes
- Malocclusion
- Tooth mobility
- Radiographic evidence of alveolar osteoporosis with closely meshed trabeculae
- Widening periodontal space
- Absence of the lamina dura
- radiolucent cyst like spaces.

Periodontitis as a manifestation of systemic disease

2 HEMATOLOGIC DISORDES AND IMMUNE DEFICIENCIES

WBC: Involved in inflammatory reactions for cellular defense

• Proinflammatory cytokine release

RBC :Gas exchange •Nutritional supply to the periodontal tissues and platelets. •Normal hemostasis •Recruitment of cells during inflammations and wound healing

- Abnormal bleeding from the gingiva or other areas of the oral mucosa that is difficult to control is an important clinical sign.

- Petechiae
- Ecchymosis(Soft palate)
- Deficiencies in the host immune response may lead to severely destructive lesions





• LEUCOCYTE DISORDERS.

• Disorders that affect production or function of leukocytes may result in severe periodontal destruction.

Neutropenia: Results in low levels of circulating neutrophils.

Agranulocytosis: is more severe neutropenia involving not only neutrophil but also basophils and eosinophils.

• Gingival margin may or may not be involved. Gingival hemorrhage, necrosis, increased salivation, and fetid odor

• In cyclic neutropenia- Oral ulcerations, severe gingivitis, rapid periodontal breakdown and alveolar bone loss ,Most bone loss around incisors and first molars. Fever, malaise and sore throat

Leukocyte Adhesion Deficiency Syndrome (LAD)

PMNs cannot leave blood vessels and migrate to the infected area; PMN cells absent from gingival tissue.

Primary & permanent teeth.

Severe acute inflammation of the gingiva

Rapid destruction of bone

treatment rarely results in long-term retention of teeth.

More than 75% of children will die before the age of 5 years if they do not receive a bone marrow transplant

Leukaemia

- Malignant neoplasias of WBCs or their precursors
- Acute or Chronic
- Replacement of bone marrow with leukemic cells reduced the normal production of RBCs, Platelets, and WBCs.

The periodontium of leukemic patients

Bleeding:

- Can be an early sign of leukaemia
- Occur in the absence of gingivitis
- Oral ulcerations & Infections (Herpes, NUG)
- Gingiva:
 - Leukemic gingival enlargement (acute > chronic)
 - Clinically: Swelling, bluish red, cyanotic, roundness and tenseness of the gingival margin, spontaneous bleeding.
 - Areas of necrosis and pseudomembrane formation.
- Periodontium & Alveolar bone: localised areas of necrosis due to leukemic infiltration of the marrow.

Associated with genetic disorders-3

- Familial and cyclic neutropenia
- Down syndrome
- Leukocyte adhesion deficiency syndrome
- Papillon-Lefevre syndrome
- Chediak-Higashi syndrome
- Infantile genetic agranulocytosis
- Histiocytosis syndromes
- Ehlers-Danlos syndrome (Type IV and VIII)
- Hypophosphatasia
- Cohen Syndrome
- Marfan syndrome
- Other

Down syndrome

- Trisomy of Chromosome 21
- 1 out of 800 births
- Mental deficiency and growth retardation
- Periodontitis occurring in almost 100% of patients <30 years old
- Oral and periodontal disease-related features:
- ✓ Poor OH

- ✓ Local factors: Diastema, high frenal attachment, crowding, malocclusion
- Level of inflammation exceeds being explainable by plaque and local factors.
- ✓ Destructive periodontitis
- Generalised deep periodontal pockets, gingival inflammation
- ✓ NUG

Papillon-Lefevre Syndrome

Clinical features:

- Palmar-planter hyperkeratosis (sometimes knees and elbows)
- Severe periodontal disease
- Calcification of the dura

Papillon-Lefevre Syndrome

- Autosomal recessive inheritance.
- Onset: cutaneous and oral signs appear before the age of 4
- Primary teeth are lost by the age of 5-6
- Permanent teeth lost by the age of 15.
- No alteration in WBC

- Defect in Cathepsin C gene
- Systemic administration of <u>synthetic retinoids + non-surgical periodontal treatment</u> in addition to <u>topical</u> <u>antimicrobials</u> such as chlorhexidine, and <u>systemic</u> <u>antibiotic therapy</u>, may give the best chance for preventing progression of periodontitis and retaining teeth





Chediak Higashi Syndrome

- Rare, autosomal recessive disorder
- Abnormalities in the cytoplasmic granules
- fusion of phagosome and lysosome to form the bactericidal phagolysosome is impaired (large azurophilic inclusions within the cytoplasm)
- Primarily affects neutrophils, platlets and melanocytes.
- Average life span is 6 years.
- Clinical features:

- Partial albinism, mild bleeding disorders, recurrent bacterial infections, rapidly destructive periodontitis
- Bone loss is usually generalized and severe.
- Patients do not respond to periodontal therapy, leading to premature loss of both deciduous and permanent dentitions.
- BMT: treatment of choice.



Hypophosphotasia

- Rickets, poor cranial bone formation
- characterized by:
 - deficiency of serum alkaline phosphatase
 - increased urinary excretion of phosphoethanolamine
 - defective bone and tooth mineralization,
 - cementum hypoplasia or aplasia
 - premature exfoliation of the primary teeth
 - Localised aggressive periodontitis In permanent teeth



Figure 3 Model 2 Psychosocial stress and its effect on behavior as manifested by alterations



PSYCOSOMATIC DISORDERS

- It affects the oral cavity by
- development of habits that are injures to the periodontium as grinding or clenching the teeth, nibbing on foreign objects as pencils, nail biting of excessive use of tobacco.
- direct effect to the autonomic nervous system on the psychologic tissue balance.

NUTRITIONAL DEFICIENCIES

- There are no Nutritional defencies that by themselves may cause Gingivitis or Periodontitis (Carranza 11t edi)
- 2. There are Nutritional defencies that produce changes in oral cavity. (Carranza 11th edi)

Changes include alterations of tissue of lips, oral mucosa,gingiva and bone

MEDICATIONS

- <u>Bisphosphonates</u> –associated with Osteoradionecrosis of jaws.
- Corticosteroids-adverse effect by diminishing the immune response to periodontal bacteria.

Other systemic conditions

Metal intoxication: Nausea, vomiting, headaches, excessive salivation

- Bismuth: a narrow black- bluish discoloration of the gingival margin in areas with preexisting inflammation
- Lead: steel grey linear pigmentation of the gingiva "Bertunian line" peculiar sweetish sensation, ulcerartion
- Mercury: pigmentation, ulceration of gingiva and destruction of underlying bone

BISMUTH INTOXICATION

- GIT disturbances,
- Ulcerative gingivostomatitis with pigmentation.
- Metallic taste.
- Burning sensation of the mucosa.
- Inflamed sore tongue.
- Narrow bluish black discoloration of the gingival margin due to precipitation of bismuth sulfide associated with vascular changes in inflammation of the gingiva.

LEAD IN TOXICATION

- pallor of face and lips .
- Peripheral neuritis, psychologic disorders and encephalitis .
- Excessive salivation .
- Coated tongue.
- Sweetish taste.
- Gingival ulceration and pigmentation (Linear pigmentation in burtonian line, Steel gray)

MERCURY INTOXICATION

- Headache
- C.V.S. symptoms .
- Increase salivation .
- Metallic taste .
- Gingival pigmentation in linear pattern due to mercuric sulfide.
- Gingival ulceration and destruction of the underlying bone.
Other Systemic conditions

Osteoporosis:

- Low bone mass
- Females

- Bone mineral density
- Consequences: risk of fracture
- Limited evidence indicates increased risk of attachment loss, bone loss and tooth loss in osteoporosis.
- Less attachment loss and less gingival bleeding have been reported in postmenopausal women receiving estrogen replacement therapy (ERT) compared with estrogen-deficient postmenopausal women

Peri-implant Diseases and Conditions Consensus Report

Berglundh, Armitage et al. 2018 Active link to consensus report

PERI-IMPLANT DISEASES AND CONDITIONS

1. Peri-implant health

Araujo & Lindhe 2018 link

2. Peri-implant mucositis

Heitz-Mayfield & Salvi 2018 link

3. Peri-implantitis

Schwarz et al. 2018 link

4. Peri-implant soft and hard tissue deficiencies

Hammerle & Tarnow 2018 link

Renvert et al. 2018 Case Definitions link

PERI-IMPLANT DISEASES AND CONDITIONS

• PERI-IMPLANT HEALTH

- Visual absence of signs of inflammation
 - -Pink, firm tissue without swelling
- •Lack of profuse (line or drop) bleeding on probing
- •There should be no increase in probing depth over time
- Bone loss over time (following initial healing) should not be ≥ 2mm

PERI-IMPLANT DISEASES

Peri-Mucositis

• Equivalent to gingivitis with teeth

Peri-Implantitis

• Equivalent to periodontitis with teeth

PERI-IMPLANT MUCOSITIS

• Signs of inflammation may be present

Red, soft tissue with swelling

- •Presence of profuse (line or drop) bleeding on probing or suppuration
- •An increase in probing depth over time

•Absence of bone loss beyond crestal bone level changes resulting from the initial remodeling









PERI-IMPLANTITIS

- Evidence of visual inflammatory changes combined with BOP and/or suppuration
- Increasing probing depths as compared to baseline (supra-structure in place)
- Progressive bone loss compared to bone levels measured radiographically at 1 year post placement of supra-structure
- Radiographic evidence of bone loss ≥ 3mm and/or probing depths ≥ 6mm in conjunction with profuse bleeding represents peri-implantitis













THE END THANKS

LEC 6

Classification of

periodontal diseases and

conditions (2017)



Party III Dr.Ghayath Aljawady B.D.S, M.Sc., Ph.D in periodontics





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2021-2022

:Department of Oral and Maxillofacial surgery / Periodontics unit

• Other conditions affecting the periodontium

- - Periodontal abscess:
- o Periodontal abscess in periodontitis patients
- o Periodontal abscess in non-periodontitis patients
- - Endodontic periodontal lesions:
- o Endo-periodontal lesions associated with endodontic and
- periodontal infections
- o Endo-periodontal lesions associated with trauma and
- iatrogenic factors
- - Mucogingival deformity and conditions
- - Traumatic occlusal force
- - Tooth and prosthetic related factors

PERIODONTAL ABSCESSES

- PA can specifically be defined as a localized accumulation of pus located within the gingival wall of the periodontal pocket, with an expressed periodontal breakdown occurring during a limited period of time, and with easily detectable clinical symptoms
- Different etiological factors may explain the occurrence of abscesses in the periodontal tissues, such as pulp necrosis (endodontic, periapical or dentoalveolar abscesses), periodontal infections (gingival or periodontal abscess), pericoronitis (pericoronal abscess), trauma, surgery, or foreign body impaction. Together, they are referred to as odontogenic or dental abscesses, and when they are associated with EPL, they could also be considered odontogenic abscesses.

Pathophysiology

The first step in the development of a PA is bacterial invasion of the soft tissues surrounding the periodontal pocket, which will develop into an inflammatory process through the chemotactic factors released by bacteria that attract polymorphonuclear leukocytes (PMN) and other cells. This will trigger intensive release of cytokines; lead to destruction of the connective tissues; encapsulation of the bacterial infection and the production of pus. Once the abscess is formed, the rate of destruction within the abscess will depend on the growth of bacteria inside the foci; their virulence, and the local pH (an acidic environment will favor the activity of lysosomal enzymes).

Microbiology

Histopathology

- The most prevalent bacterial species identified in PA, by means of different techniques were
- Porphyromonas gingivalis (50-100%),
- Prevotella intermedia,
- Prevotella melaninogenica,
- Fusobacterium nucleatum,
- Tannerella forsythia,
- Treponema species,
- Campylobacter species,
- Capnocytophaga species,
- Aggregatibacter
 actinomycetemcomitans
- or gram-negative enteric rods

The histopathology of periodontal abscess lesions present with an acute inflammatory infiltrate; intense focus of inflammation, with presence of neutrophils and lymphocytes in an area of destroyed and necrotic connective tissue; and a destroyed and ulcerated pocket epithelium.

Etiology: risk factors

PA may develop in a pre-existing periodontal pocket (e.g., in patients with periodontitis) or in the absence of a pre-existing periodontal pocket.

Periodontal abscess in periodontitis patients

- In periodontitis patients, a PA could represent a period of disease exacerbation, favored by the existence of tortuous pockets, presence of furcation involvement or a vertical defect, in which the marginal closure of the pocket could lead to an extension of the infection into the surrounding periodontal tissues. In addition, changes in the composition of the subgingival microbiota, with an increase in bacterial virulence, or a decrease in the host defense, could also result in an inefficient capacity to drain the increased suppuration. Different subgroups could be distinguished
- Acute exacerbation:
- • In untreated periodontitis.
- • In "refractory" periodontitis.
- After different treatments:

 Scaling and root planing or professional prophylaxis: dislodged calculus fragments could be pushed into the tissues, or inadequate scaling could allow calculus to remain in deep pocket areas, whereas the coronal part would occlude the normal drainage.

• Surgical periodontal therapy: associated with the presence of foreign bodies such as membranes for regeneration orsutures.

• Systemic antimicrobial intake, without subgingival debridement, in patients with severe periodontitis could also cause abscess formation, probably related to an overgrowth of opportunistic bacteria.

• Use of other drugs: e.g., nifedipine.

Periodontal abscess in non-periodontitis patients

- PA can also occur in previously healthy sites, because of Impaction of foreign bodies: dental floss, orthodontic elastic, toothpick, rubber dam, or popcorn hulls.
- Harmful habits (biting wire, nail biting, clenching) could favor abscess formation because of subgingival impaction of foreign bodies or to coronal closure of the pocket.
- Orthodontic factors, such as inadequate orthodontic forces or across-bite, have been reported to favor PA development.
- Gingival enlargement.
- Alterations of the root surface, including:
- Severe anatomic alterations, such as invaginated tooth, dens evaginatus (grooves)

 \circ Minor anatomic alterations, such as cemental tears, enamel pearls or developmental grooves.

○ latrogenic conditions, such as perforations.

 $\circ\,$ Severe root damage: vertical root fracture or cracked tooth syndrome extending through the root.

• External root resorption.

DIAGNOSIS + SIGN AND SYMPTOM

• A series of symptoms have been reported by patients suffering from a PA, such as

pain, tenderness of the gingiva, swelling, or tooth "elevation."

The most prominent sign during the oral examination was the presence of an ovoid elevation in the gingiva along the lateral part of the root. Suppuration on probing or sampling was a common finding , . A PA was usually associated with a deep periodontal pocket , bleeding on probing , and increased tooth mobility . Bone loss was normally observed in the radiographic examination. Extraoral findings were uncommon, but could include facial swelling , elevated body temperature, malaise, regional lymphadenopathy

- Differential diagnosis is critical, because PA may be like other oral conditions:
- Other odontogenic abscesses (dento-alveolar abscesses, pericoronitis, endo-periodontal abscess), or other acute conditions(lateral periapical cyst and postoperative infection)
- • Tumor lesions, including metastatic tumoral lesions, odontogenic myxoma, non-Hodgkin's lymphoma, squamous cell carcinoma, metastatic carcinoma.
- • Other oral lesions: pyogenic granuloma, osteomyelitis, odontogenic keratocyst, eosinophilic granuloma.
- • Self-inflicted gingival injuries.
- • Sickle cell anemia.
- • Abscesses after surgical procedures.







:Endodontic periodontal lesions

• Pathophysiology and histological features

- The dental pulp and the periodontium have different communication pathways, such as the apical radicular foramina, accessory (or lateral) canals, and dentinal tubules. Accessory canals are more prevalent at the apical third of the roots, but they may be found in high numbers in other areas, such as in the furcation regions.
- Pathological communication between these structures, which includes the migration of microorganisms and inflammatory mediators between the root canal and the periodontium, may lead to the EPL.

:Endodontic periodontal lesions

Clinical presentation

EPL are clinical conditions involving both the pulp and periodontal tissues and may occur in acute or chronic forms. When they are associated with a recent traumatic or iatrogenic event (e.g. root fracture or perforation), the most common manifestation is an abscess accompanied by pain. However, EPL, in subjects with periodontitis, normally present slow and chronic progression without evident symptoms. The most common signs and symptoms associated with a tooth affected by an EPL are deep periodontal pockets reaching or close to the apex and negative or altered response to pulp vitality tests. The other signs and symptoms reported, in order of prevalence, are: bone resorption in the apical or furcation region, spontaneous pain or pain on palpation and percussion, purulent exudate, tooth mobility, sinus tract, and gingival color alterations



Endo-periodontal lesions associated with endodontic and periodontal infections

- They might be triggered:
- (1) by a carious lesion that affects the pulp and, secondarily, affects the periodontium;
- (2) by periodontal destruction that secondarily affects the root canal;
- (3) or by both events concomitantly. this type occurs less frequently and is usually referred to as a "true-combined" or "combined" lesion.
- These lesions may develop in subjects with periodontal health or disease
- .The periodontal condition has an important impact in the prognosis of the EPL because of the striking changes in the oral ecology of subjects with periodontal diseases. Converting this ecology back into a healthy state is challenging, especially in patients with severe periodontitis and in teeth with deep pockets, as in the case of EPL. Therefore, a detailed periodontal examination is a very important step for the accurate diagnosis and treatment plan of EPL.



Endo-periodontal lesions associated with trauma and iatrogenic factors

• These conditions usually have a poor prognosis as they affect the tooth structure. The most common lesions in this category were:

(1) root/pulp chamber/furcation perforation (e.g. because of root canal instrumentation or to tooth preparation for post-retained restorations)

(2) Root fracture or cracking (e.g., because of trauma or tooth preparation for post-retained restorations)

(3) external root resorption (e.g., because of trauma)or

(4) pulp necrosis (e.g., because of trauma) draining through the periodontium.









Risk factors

- The main risk factors for the occurrence of EPL were
- advanced periodontitis,
- trauma, and iatrogenic events.

Other reported risk factors were

the presence of grooves, furcation involvement, active carious lesions. Furcation involvement, high level of bone destruction around the affected tooth, and anatomic problems (e.g. the presence of grooves), could worsen the prognosis of EPL.Most of the single EPL in non-periodontitis patients were associated with palatal grooves. Periodontal Manifestations of Systemic Diseases and Developmental and Acquired Conditions: Consensus Report Jepsen, Caton et al. 2018 Active link to consensus report

PERIODONTAL MANIFESTATIONS OF SYSTEMIC DISEASES AND DEVELOPMENTAL AND ACQUIRED CONDITIONS

1. Systemic diseases or conditions affecting the periodontal supporting tissues

Albandar et al. 2018 link

2. Other Periodontal Conditions

Papapanou, Sanz et al. 2018 link

Herrera et al. 2018 link

- a. Periodontal Abscesses
- b. Endodontic-Periodontal Lesions

3. Mucogingival deformities and conditions around teeth

Cortellini & Bissada 2018 link

- Gingival phenotype
- b. Gingival/soft tissue recession
- Lack of gingiva
- Decreased vestibular depth
- e. Aberrant frenum/muscle position
- f. Gingival excess
- g. Abnormal color
- Condition of the exposed root surface

4. Traumatic occlusal forces

Fan & Caton 2018 link

- a. Primary occlusal trauma
- b. Secondary occlusal trauma
- c. Orthodontic forces

Prostheses and tooth-related factors that modify or predispose to plaqueinduced gingival diseases/periodontitis

Ercoll & Caton 2018 link

- Localized tooth-related factors
- Localized dental prostheses-related factors

Mucogingival deformity and conditions

- 1. gingival/soft tissue recession
 - 1. facial or lingual surfaces
 - 2. interproximal (papillary)
- 2. lack of keratinized gingiva
- 3. decreased vestibular depth
- 4. aberrant frenum/muscle position
- 5. gingival excess
 - 1. pseudo-pocket
 - 2. inconsistent gingival margin
 - 3. excessive gingival display
 - 4. gingival enlargement
- 6. abnormal color

Periodontal biotype and attached gingiva •

 A thin periodontal biotype, absence of attached gingiva, and reduced thickness of the alveolar bone due to abnormal tooth position in the arch are considered risk factors for the development of gingival recession. The presence of attached gingival tissue is considered important for maintenance of gingival health. The current consensus, based on case series and case reports (low level of evidence), is that about 2 mm of KT and about 1 mm of attached gingiva are desirable around teeth to maintain periodontal health, even though a minimum amount of keratinized tissue is not needed to prevent attachment loss when optimal plaque control is present

Thick periodontal phenotype



- Thick fibrotic gingival tissues
- Short, wide crowns
- Adequate amount of attached gingiva
- Thick underlying alveolar bone
- Resistant to gingival recession and interdental papilla loss

Thin periodontal phenotype



- Thin frible gingival tissues
- Long, narrow, conical crowns
- Minimal amount of attached gingiva
- Thin underlying alveolar bone
- Prone to gingival recession and interdental papilla loss

- A modern recession classification based on the interdental CAL measurement has been proposed by Cairo et al.
- **Recession Type 1 (RT1)**: Gingival recession with no loss of interproximal attachment. Interproximal CEJ is clinically not detectable at both mesial and distal aspects of the tooth.
- Recession Type 2 (RT2): Gingival recession associated with loss of interproximal attachment. The amount of interproximal attachment loss (measured from the interproximal CEJ to the depth of the interproximal sulcus/ pocket) is less than or equal to the buccal attachment loss (measured from the buccal CEJ to the apical end of the buccal sulcus/pocket).
- Recession Type 3 (RT3): Gingival recession associated with loss of interproximal attachment. The amount of interproximal attachment loss (measured from the interproximal CEJ to the apical end of the sulcus/pocket) is greater than the buccal attachment loss (measured from the buccal CEJ to the apical end of the buccal sulcus/pocket).



Traumatic occlusal force

- Occlusal trauma :- injury resulting in tissue changes within the attachment apparatus as a result of occlusal force (s).
- Primary occlusal trauma :- injury resulting in tissue changes from excessive occclusal forces applied to a tooth or teeth with normal support. It occurs in the presence of:
- 1- normal bone level
- 2- normal attachment level
- 3- excessive occlusal force (s).
- Secondary occlusal trauma :- injury resulting in tissue changes from normal or excessive occlusal forces applied to a tooth or teeth with reduced support . it occurs in the presence of
- 1- bone loss. 2- attachment loss. 3- normal / excessive occlusal force(s).

Histological studies

• The histological features of occlusal trauma

Alteration of the periodontium that have been associated with occlusal trauma will vary with the magnitude & direction of applied force and location (pressure versus tension). These changes many include:, widening / compression of the periodontal ligament (, bone remodeling (resorption /repair ,hyalinization-necrosis , increased cellularity ,vascular dilatation / permeability , thrombosis root resorption and cemental tears

Clinical studies

- Clinical indicators include
- 1- mobility (progressive). 2- occlusal prematurities. 3- thermal sensitivity. 4- wear facets. 5-muscle tenderness. 6- fractured teeth. 7migration of teeth.

Radioqraphic indicators :1-Discontinuity and thickening of lamina dura . 2- widened periodontal ligament space 3-evidence of root resorption and or bone loss

?How to diagnose trauma from occlusion

- 1-history: a-teeth which are sensitive (not related to recession, caries, or broken fillings). b-muscle pain c-problems with TMJ (clicking, limitation in opening, or deviation).
- 2-examination: a- attrition of teeth due to bruxism. b-decrease in vertical dimension. The functional attrition is normally compensated by continuous deposition of cementum in the apical area which differs from bone in that it has no resorption. c-some teeth with edges chipped. d-broken restorations. e-mobility of teeth(without periodontal disease or bone lesion). f-changes in position of teeth g-signs &symptoms of pulpal hyperemia or pulpitis without any obvious This could develop to pulpal necrosis then develop to peri apical lesion. cause.
- 3- x-ray; periapical x-ray Widening in space of pdl
- 4- occlsal analysis a-impression-cast-bite registration-mounted on fully adjustable articulater. the analyze occlusion and determine area of premature contact. b- occlusion indicater




Treatment

• Treatment; not every trauma from occlosion require treatment just keep these patient under observation. We should not change the pattern of occlusion unless we are sure that pattern is causing the problem. For example; sometimes the cause is the muscles[uncoordinated] so no matter how much we trim the teeth we wont solve the problem.

1-interfering hopeless tooth-extraction.

2-new restoration of interfering teeth.

3-diminshed occlusal table require placement [bridge,partial denture....]

4-bite plane, night guard, now called [inter-occlusal appliance]

a-prevent teeth from fully interdigitating.

b-help in preventing or minimizing isomeric contraction of muscles.

c-abolish the effect of mechanoreceptors.

5-exercise for more harmonious occlusion. 6-portable electromyography contain warning system[feed back] measures the electric potential on the muscles.

A. Localized tooth-related factors that modify or predispose to plaque-induced gingival diseases/periodontitis

Tooth anatomic factors. 1

Root fractures. 2

Cervical root resorption, cemental tears. 3

Root proximity. 4

Altered passive eruption. 5

B. Localized dental prosthesis-related factors

Restoration margins placed within the supracrestal attached. 1 tissues

Clinical procedures related to the fabrication of indirect. 2 restorations

Hypersensitivity/toxicity reactions to dental materials. 3

Treatment plan guidelines

- Phase 3 (corrective/surgical phase)
 - **Phase 4 (maintenance therapy)**



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Outline for the lecture

- Phase 3 (corrective/surgical phase)
- o Objectives of surgical therapy
- o Periodontal access surgery:
- i- Resective
- ii- Regenerative
- o Extraction of hopeless teeth
- o Periodontal plastic surgery:
- i- Mucogingival surgery
- ii- Aesthetic crown lengthening
- o Pre-prosthetic surgery:
- i- Crown lengthening
- ii- Implant site preparation

Phase 4 (maintenance therapy)

- o Clinical recommendations
- o Self-performed supragingival dental biofilm control
- o Adjunctive therapies for gingival inflammation
- o Professional supragingival dental biofilm control
- o Risk factor control





- D. p.d.surgery; specific surgical manipulation of p.d. soft tissue & bone.
- Early problems can be corrected with successful phase I therapy, consisting of biofilm removal by the patient on a daily basis, scaling, and root planing when necessary.
- moderate to advanced cases cannot be resolved without surgically gaining access to the root surface for root planing and reducing or eliminating pocket depth to allow the patient to remove biofilm.
- THIS phase of therapy is used to surgically treat residual periodontal pockets and bone defects remaining after phase I therapy.
- ❑ All patients treated surgically need to have a preoperative history of adequate plaque control with 20% or more of tooth surfaces free of plaque after oral hygiene procedure.

Objectives of surgical therapy





- **1-Accessibility and direct vision for proper S+ RP**
- 2-Reduction or elimination of plaque retentive area especially periodontal pockets that
- have not responded to initial therapy.
- 3-Eliminate inflamed periodontal tissue
- 4-Enhancing the regeneration of periodontal tissue
- 5-Create a physiologic morphology of the dentogingival area that will facilitate efficient self performed plaque control
- 6-Correct mucogingival defect and improve periodontal aesthetic
- 7-Provide access to correct bony defects



Periodontal Surgery

Pocket Reduction Surgery

- Resective (e.g., gingivectomy, apically displaced flap, undisplaced flap with or without osseous resection)
- Regenerative (e.g., flaps with grafts, membranes)

Correction of Anatomic or Morphologic Defects

- Plastic surgery techniques used to widen attached gingiva (e.g., free gingival grafts)
- Esthetic surgery (e.g., root coverage, recreation of gingival papillae)
- Pre-prosthetic techniques (e.g., crown lengthening, ridge augmentation, vestibular deepening)
- Placement of dental implants, including techniques for site development for implants (e.g., guided bone regeneration, sinus grafts)

Periodontal access surgery:



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<u>Resective Procedures:</u> It is the procedure that means to eliminate or reduce the pocket, by excising or amputating the tissue constricting the pocket wall. e.g; Gingivectomy, Gingivoplasty, Apically positioned flap with or without osseous surgery. New attachment procedures. It is the reunion of connective tissue by formation of new cementum with inserting collagen fibers on root surface that has been deprived of its periodontal ligament e.g; Closed curettage. Excisional new attachment procedure

(ENAP). Open flap curettage. Modified widman flap procedure.

<u>Regeneration procedures.</u> The use of regenerative materials, including bone grafts, barrier membranes, and wound-healing agents. Intrabony defects, particularly vertical defects with multiple osseous walls, are often amenable to regeneration with these techniques. Papilla preservation flap Conventional flap with regenerative procedures





Periodontal plastic surgery

is defined as the surgical procedures performed to correct or eliminate anatomic, developmental, or traumatic deformities of the gingiva or alveolar mucosa.



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Five objectives of periodontal plastic surgery are:
1. Problems associated with attached gingiva.
2. Problems associated with a shallow vestibule
3. Problems associated with an aberrant frenum.
4. Aesthetic surgical therapy.
5. Tissue engineering.

Problems Associated With Attached Gingiva



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- The ultimate goal of Mucogingival surgical procedures is the creation or widening of attached gingiva around teeth and implants.
- The width of the attached gingiva varies in different individuals and on different teeth of the same individual.
- The width of the attached gingiva is determined by subtracting the depth of the sulcus or pocket from the distance between the crest of the gingival margin and the Mucogingival junction.





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o Widening the attached gingiva accomplishes four objectives:

 Enhances plaque removal around the gingival margin.
 Improves aesthetics.
 Reduces inflammation around restored teeth.
 Allows gingival margin to bind better around teeth and implants with attached gingiva.





o Problems Associated With a Shallow Vestibule

- □ Another objective of periodontal plastic surgery is the creation of vestibular depth when it is lacking.
- □ Gingival recession displaces the gingival margin apically, reducing vestibular depth, which is measured from the gingival margin to the bottom of the vestibule. With minimal vestibular depth, proper hygiene procedures are reduced.
- □ The sulcular brushing technique requires the placement of the toothbrush at the gingival margin, which may not be possible with reduced vestibular depth.
- Minimal attached gingiva with adequate vestibular depth may not require surgical correction if proper a traumatic hygiene is practiced with a soft brush. Minimal amounts of keratinized attached gingiva with no vestibular depth benefit from Mucogingival correction. Adequate vestibular depth is also necessary for the proper placement of removable prostheses.



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Problems Associated With an Aberrant Frenum

- □ An important objective of periodontal plastic surgery is correction of frenal or muscle attachments that may extend coronal to the Mucogingival junction.
- □ If adequate keratinized, attached gingiva exists coronal to the frenum, it may not be necessary to remove the frenum.
- □ A frenum that encroaches on the margin of the gingiva can interfere with biofilm removal, and the tension on the frenum tends to open the sulcus. In these cases, surgical removal of the frenum is indicated.
- □ Frenotomy and Frenectomy



Crown lengthening

□ Indication

1-Short clinical crown require increased retention for placement of full coronal restoration (including cases of gross tooth wear requiring full mouth rehabilitation)

2-Deep subgingivally located crown preparation margins, resulting in difficulty finishing margins and taking impressions also encroachment on the biologic width

3-Sub gingival caries

4-Root fractures or root resorption in the cervical third of the tooth root

5-Aesthetic improvement of anterior teeth with short clinical crowns and high lip line





Pre-prosthetic surgery:

- After the loss of natural teeth, bony changes in the jaws begin to take - place immediately because the alveolar bone no longer respond to stresses placed in this area by the teeth and periodontal ligament.
- □ Bone begins to resorb the specific pattern of resorption is unpredictable in a given patient because great variation exists among individuals. This resorption tend to effect the mandible more severely than maxilla because of decreased surface area and less favorable distribution of occlusal of force.
- □ The objective of (P.S) is to create proper supporting structures for subsequent placement of prosthetic appliances.

Maintenance Phase

• Preservation of the periodontal health of the treated patient requires a supportive program that is just as important as the therapy used to treat the periodontal disease.

•After phase I therapy has been completed, patients are placed on a schedule of periodic recall visits for maintenance care to prevent the recurrence of the disease.

•Transfer of the patient from active treatment status to a maintenance program is a definitive step in total patient care that requires time and effort on the part of the dentist and staff.

• Patients who are not maintained in a supervised recall program subsequent to active treatment show obvious signs of recurrent periodontitis (e.g., increased pocket depth, bone loss, or tooth loss).

GOALS OF SUPPORTIVE PERIODONTAL TREATMENT

1. To prevent or minimize the recurrence and progression of periodontal disease in patients who have been previously treated for gingivitis, periodontitis and for peri-implantitis.

To prevent or reduce the incidence of tooth loss by monitoring the dentition and by any prosthetic replacement of the natural teeth.

To increase the probability and treating in a timely manner, other diseases or conditions found in the oral cavity.

- Motivational techniques and reinforcement of the importance of the maintenance phase of treatment should be considered before performing definitive periodontal surgery.
- The maintenance phase of periodontal treatment starts immediately after the completion of phase I therapy.
- □ While the patient is in the maintenance phase, the necessary surgical and restorative procedures are performed.
- □ This ensures that all areas of the mouth retain the degree of health attained after phase I therapy.

Maintenance Program

- The interval between visits is initially set at 3 months but may vary according to the patient's needs. Periodontal care at each recall visit comprises three parts:
- The first part involves examination and evaluation of the patient's current oral health.
- The second part includes the necessary maintenance treatment and oral hygiene reinforcement.
- The third part involves scheduling the patient for the next recall appointment, additional periodontal treatment, or restorative dental procedures. The time required for a recall visit for patients with multiple teeth in both arches is approximately 1 hour

Maintenance Recall Procedures

Part I: Examination

(Approximate time: 14 minutes) Patient greeting Medical history changes Oral pathologic examination Oral hygiene status Gingival changes Pocket depth changes Pocket depth changes Mobility changes Occlusal changes Dental caries Restorative, prosthetic, and implant status

Part II: Treatment

(Approximate time: 36 minutes) Oral hygiene reinforcement Scaling Polishing Chemical irrigation or site-specific antimicrobial placement

Part III: Report, Cleanup, and Scheduling

(Approximate time: 10 minutes) Write report in chart. Discuss report with patient.

Clean and disinfect operatory. Schedule next recall visit. Schedule further periodontal treatment. Schedule or refer for restorative or prosthetic treatment.

Examination and Evaluation

- □ The recall examination is similar to the initial evaluation of the patient. However, because the patient is not new to the office, the dentist or hygienist primarily looks for changes that have occurred since the last evaluation.
- □ Updating of changes in the medical history and evaluation of restorations, caries, prostheses, occlusion, tooth mobility, gingival status, and periodontal and peri implant probing depths are important parts of the recall appointment.
- The oral mucosa should be carefully inspected for pathologic conditions
- Radiographic examination must be individualized, depending on the initial severity of the case and the findings during the recall visit. These are compared with findings on previous radiographs to check the bone height and look for repair of osseous defects, signs of trauma from occlusion, periapical pathologic changes, and caries.





Thanks



Periodontology

Etiology of periodontal disease Risk factors for periodontal diseases:

Periodontology

Etiology of periodontal disease

- Risk factors for periodontal diseases:
- o Definitions of risk factors
- o Systemic risk factors:
- i- Modifiable risk factors
- ii- Non-modifiable risk factors
- o Local predisposing factors:
- i- Calculus
- ii- latrogenic factors
- iii- Margins of restorations
- iv- Malocclusion
- v- Associated with orthodontic therapy
- o Local anatomic risk factors

Risk factors for periodontal diseases:

- There is strong evidence available today suggesting a multifactorial etiology of periodontal diseases. It has been demonstrated that the susceptibility of different individuals to periodontitis for the same amount of local factors is variable. Some individuals demonstrate less periodontal breakdown, whereas other demonstrate significantly more, for a similar amount of pathogenic microflora.
- Periodontal disease is considered to have multiple risk factors. The term "risk factor refers to an aspect of personal behaviour or lifestyle, an environmental exposure, or inherited characteristics, which on the basis of epidemiological evidence is known to be associated with a health related condition".
- Risk factors therefore are part of the causal chain for a particular disease or can lead to an exposure of an individual to a disease and therefore the presence of risk factors implies a direct increase in the probability of the disease occurring.

Definitions

• Risk factor:

Risk factors may be environmental, behavioral or biologic in nature that when present increases the likelihood of a disease. These include poor oral hygiene and pathogenic microflora, tobacco smoking, diabetes, and psychological stress.

Risk determinant:

Risk determinants are those risk factors that cannot be modified. These include genetic factors, host response, age, gender and socioeconomic status.

• Risk indicators:

Risk indicators are probable or putative risk factors that have been identified in cross-sectional studies, but not confirmed through longitudinal studies. These include osteoporosis, HIV/AIDS, obesity, alcohol consumption, infrequent dental visits, drug intake associated periodontal disorders and iatrogenic factors.

• Risk marker:

A risk factor that can be used to predict the future course of the disease is known a risk marker. The risk markers for periodontal disease include the previous history of periodontal disease and bleeding on probing.

Risk factors for periodontal diseases:

• The risk factors for periodontal diseases can be classified broadly into two categories: modifiable risk factors and non-modifiable risk factors. Modifiable risk factors are usually environmental or behavioral in nature, whereas non-modifiable risk factors are usually intrinsic to the individual and therefore can not be easily changed. As already stated, the non-modifiable risk factors are also referred to as risk determinants.

- Poor oral hygiene and pathogenic microflora:-
- There is strong evidence in favor of the role of periodontal pathogenic microorganisms in the initiation and progression of periodontal diseases. Bacterial species, including (Porphyromonas gingivalis , Aggregatibacter actinomycetemcomitans , Bacteroides forsythus , Prevotella intermedia , Peptostreptococcus micros , Fusobacterium nucleatum and spirochetes) have been specifically found to contain the virulence factors responsible for evasion of host response and periodontal breakdown.
- The reduction of this pathogenic microflora has been found to be strongly associated with improvement in periodontal health
- Thus, reduction in the microbial load around the teeth is the cornerstone of periodontal therapy.

Tobacco smoking:

- The periodontal breakdown has been found to be more among smokers as compared to non-smokers. This finding establishes tobacco smoking as an important risk factor for the development of periodontitis. Although, smokers have reduced clinical signs of periodontal inflammation as compared to non-smokers but they have an accelerated periodontal breakdown
- A detailed description of the effect of smoking on periodontitis has been given in "Smoking as a risk factor for periodontitis" in next lecture

• **Diabetes Mellitus:**The association between diabetes mellitus and periodontitis has been found to be bi-directional. The poor glycemic control worsens periodontal disease status and poor periodontal health has been shown to worsen the glycemic control. Many studies and reviews have been published which support a relationship between periodontitis and diabetes. There are multiple mechanisms by which hyperglycemic state worsens the periodontal status of the patient. The most well-established mechanism is, altered collagen metabolism in diabetic patients. Other mechanisms include altered monocyte-macrophage function, altered wound healing, increased oxidative stress and effects of the hyperglycemic state on cellular functions. On the other hand, periodontitis results in increased pro-inflammatory cytokines locally and then systemically. IL-6 and TNF- α are two main chemical mediators which relate periodontitis to diabetes.

• Psychological stress:

- Psychological stress is another risk factor for periodontal diseases . It has been observed that stressed, anxious, or depressed persons tend to neglect their oral hygiene. Furthermore, smokers have been observed to smoke more under stress which further deteriorates their periodontal status. A stressed individual often takes inadequate diet, which is responsible for the overall deterioration of patient health. Stress has been shown to result in elevated levels of cortisols which further have been positively associated with the extent and severity of periodontitis .
- Stress management has been shown to improve the periodontal status of patients with periodontitis . Thus, stress can be considered as a modifiable risk factor that may influence the severity of periodontal diseases.

Nonmodifiable risk factors (Risk determinants)

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• Genetic factors:

- Present data strongly suggest that genetic factors are important risk factors for periodontal disease progression.
- Family studies, twin studies, population studies and single nucleotide polymorphisms (SNP) have been done to find out the genetic basis of periodontal diseases.
- Gene poly-morphisms have been shown to have a positive correlation with increased severity of periodontal disease. IL-1 and TNF-α gene polymorphism have been studied extensively and a positive correlation has been demonstrated between periodontitis and these polymorphisms
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• Host response:

- The present research on the etiopathogenesis of periodontal disease suggests that most of the tissue destruction caused by host-microbial interaction is primarily by host-derived chemical mediators.
- Further, it has been found that certain individuals mount an abnormal host response to the microbial challenge. The presence of hyperactive neutrophils and monocytes in individuals showing severe periodontal breakdown.
- Matrix metalloproteinases (MMPs) are responsible for the remodeling of connective tissue. Studies have shown that individuals demonstrating aggressive periodontal breakdown have prolonged and excessive activation of the latent MMPs resulting in the enhanced degradation of collagen, which is a primary component of the periodontal matrix .
- Because these altered immune responses are genetically determined and cannot be altered with our present strategies, the altered host response can be considered as a non-modifiable risk factor

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- Age:
- Various studies have demonstrated that with increasing age the severity of periodontal diseases increases .
- It is a general observation that periodontal attachment loss is more in elderly individuals than in younger individuals.
- Further-more, the advanced periodontal bone loss is less commonly observed in young individuals.
- However, it must be noted that the periodontal destruction observed in elderly individuals is a cumulative destruction over several years rather than a result of increased rates of destruction.

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• Gender:

- In general, males have been shown to have more overall periodontal destruction as compared to females.
- The reason suggested for this finding is the ignorance of oral hygiene, which is usually observed among males.
- However, it should be noted that this association is not reliable as it entirely depends on the maintenance of oral hygiene and not on gender

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• Socioeconomic status:

- The individuals with low socioeconomic status have been shown to have more periodontal destruction as compared to those having average or high socioeconomic status.
- However, the relationship between socioeconomic status and periodontitis is less direct as compared to gingivitis.
- The reason suggested for this difference is a better education level and more access to oral health care services by individuals with good socioeconomic status.

Local predisposing factors:

1-Calculus 2- Iatrogenic factors 3-Margins of restorations 4-Malocclusion 5- Associated with orthodontic therapy

• Calculus mentioned in previous lecture

• Iatrogenic factors and margins of restorations

IATROGENIC FACTORS

Deficiencies in the quality of dental restorations or prostheses are contributing factors for gingival inflammation and periodontal destruction.

a-Over hanging Margins of Restorations

Contribute to the development of periodontal disease by 1changing the ecology of the gingival sulcus to an area that favors the growth of disease associated gram-negative anaerobic microorganism 2- inhibiting the patient performed plaque control

b. Over contoured crown

Over contoured crowns and restorations tend to accumulate plaque possibly prevent the self- cleaning mechanisms of the adjacent cheek, lips and tongue.

c. Open Contacts

Food particles create a favorable environment for plaque accumulation. \Box Acts as a direct mechanical irritant to the tissue.

Malocclusion

- Irregular alignment of teeth or crowding as found in cases of malocclusion enhance retention of bacterial plaque and make plaque control more difficult .
- Several investigators have found a direct relation between crowding and periodontal disease
- Orthodontic Therapy may affect the periodontium by favoring plaque retention by :
- 1- directly injuring the gingiva as result of overextended bands
- 2- creating excessive forces , unfavorable forces or both on the tooth and supporting structures.
- □ Plaque retention and composition Orthodontic appliances modify plaque composition AND INCREASES anaerobic g-ve bacteria As (prevotella intermedia)

□ Gingival trauma Orthodontic bands shouldn't be placed beyond the level of epithelial attachment because This will result on apical proliferation of jun. epithelium THEN gingival recession

 Local predisposing factors: 1-Calculus
2- Iatrogenic factors
3-Margins of restorations 4-Malocclusion
5- Associated with orthodontic therapy

Local anatomic risk factors

Anatomic variation of soft tissue and teeth

• Cervical enamel projection

They develop more commonly on buccal surfaces of mandibular molars These projections are plaque retentive and can cause loss of periodontal attachment at furcation area \Box

• Enamel pearls

in furcation area of maxillary molars

- High frenum attachment
- become problem if the attachment is too close to the marginal gingiva , tension on frenum may pull the gingiva away from the tooth AND INCREASE plaque acc. & prevent proper tooth brushing & pocket and gingival recession.

Periodontology

Microbiologic Specificity of Periodontal Diseases

Microbiologic specificity of periodontal diseases

- Traditional nonspecific plaque hypothesis
- Specific plaque hypothesis
- Updated nonspecific plaque hypothesis
- Ecologic plaque hypothesis
- Keystone Pathogen Hypothesis

Dental calculus

- Clinical appearance and distribution (Supragingival and Subgingival Calculus)

- Calculus formation:

- o Theories of calculus formation
- Calculus composition:
- o Inorganic content
- o Organic content
- Attachment to tooth surfaces and implants
- Clinical significance

KOCH'S POSTULATES

Koch defined the criteria for attributing an organism as the cause of specific disease.

- 1. The organism must be isolated from every patient with the disease.
- 2. The organism must be isolated and cultured outside the body (in vitro) in pure culture.
- 3. The pure organism must cause the disease in healthy, susceptible animals.
- 4. The organism must be recovered from the inoculated animal.
- 5. The antibody to the organism should be detected in the patient's serum.



Non Specific Plaque Hypothesis

In the mid-1900s

-Periodontal diseases resulted from an accumulation of plaque over time, eventually with a diminished host response and increased host susceptibility with age.

-Periodontal disease resulted from the "elaboration of noxious products by the entire plaque flora.

-When only small amounts of plaque were present, the noxious products were neutralized by the host.

Similarly, large amounts of plaque would cause a higher production of noxious products, which would essentially overwhelm the host's defenses.

Non Specific Plaque Hypothesis

Objections to this theory:

Some individuals with considerable amounts of plaque ,calculus and gingivitis, never developed destructive periodontitis.

When periodontitis develops ,there is considerable site specificity.

Some sites were unaffected, whereas advanced disease was found in adjacent sites.

A uniform host response is incompatable with the concept that all plaque was equally pathogenic.

The control of periodontal diseases depended on the reduction of the

total amount of plaque.

Although the nonspecific plaque hypothesis has been discarded ,most of the therapeutic interventions are still based on the basic principles of the nonspecific plaque hypothesis.

Specific Plaque Hypothesis

In early 1960s

It focuses on the importance of the **qualitative composition** of the resident microbiota(presence of or an increase in specific microorganisms).

The plaque harbors specific bacterial pathogens may provoke periodontal disease by producing substances that mediate the destruction of host tissues.

Different bacterial morphotypes were found in healthy versus periodontally diseased sites .

Major advances were made in the techniques used to isolate and identify periodontal microorganisms.

Specific Plaque Hypothesis

Support of this hypothesis:

The recognition of *A.actinomycetemcomitans* as a pathogen in localized aggressive periodontitis.

The introduction of new molecular methods for bacterial identification increase the power of examining the microbiota associated with states of health and disease.

Example:

The association of **"red complex"** bacteria (*P. gingivalis*, *T. forsythia*, and *T. denticola*) with periodontal disease was based on the analysis of 40 different bacteria in more than 13,000 plaque samples.



Diagrammatic representation of selected species in the microbial complexes identified in the subgingival microbiota. Each defined grouping is identified by a color. The red complex species, as a group and individually, are found more often in periodontal sites demonstrating bleeding on probing.

Specific Plaque Hypothesis

Objections to this theory:

Disease association studies did not reveal whether the presence of specific bacteria **causes** or **correlates** with the presence of disease.

Periodontal disease can occur even in the absence of defined "pathogens," such as red complex bacteria, and conversely that "pathogens" may be present in the absence of disease.

Ecologic Plaque Hypothesis

During the 1990s

Both the total amount of dental plaque and the specific microbial composition of plaque can lead to the transition from health to disease.

The health associated dental plaque microbiota is relatively stable over time and in a state of dynamic equilibrium or "microbial homeostasis."

The host can control subgingival plaque to some extent by a tempered immune response and low levels of GCF flow.

Changes in the host response occur due to an excessive accumulation of nonspecific dental plaque, by plaqueindependent host factors (e.g., the onset of an immune disorder, changes in hormonal balance [e.g., during pregnancy]), or by environmental factors (e.g., smoking, diet).

Changes in the host status, such as inflammation, tissue degradation, and/or high GCF flow, may lead to a shift in the microbial population in plaque.



Ecologic plaque hypothesis in relation to periodontal diseases: gingivitis and periodontitis. The accumulation of plaque causes the inflammation of adjacent tissues (gingivitis) and other environmental changes that favor the growth of gram-negativeanaerobes and proteolytic species, including periodontopathogens. The increased proportions of such species result in the destruction of periodontal tissues (i.e., periodontitis). *Eh*, Redox potential; *GCF*, gingival crevicular fluid.

Ecologic Plaque Hypothesis

Changing of micro-environmental, the no. of beneficial spp. may decrease, whereas the no. of potentially pathogenic spp. increases.

This gradual shift in the entire microbial community, known as *dysbiosis*, may result in a chronic disease state such as periodontitis.

This hypothesis explain why disease-associated organisms are minor components of the oral microbiota in health; these organisms are compete with other spp. during microbial homeostasis.

Disease is associated with the overgrowth of specific members of the dental plaque biofilm when the local microenvironment changes, but it is not necessarily the same spp. in each case.

So,therapeutic intervention can be useful by:-

Eliminating the etiologic stimulus(microbial, host, or environmental)that can restore microbial homeostasis.

Targeting specific microorganisms may be less effective because the conditions for disease will remain.

Keystone Pathogen Hypothesis and Polymicrobial Synergy and Dysbiosis Model

The keystone pathogen hypothesis indicates that certain low abundance microbial pathogens can cause inflammatory disease by remodeling a normally benign microbiota into a dysbiotic one.

In other words, certain pathogens may trigger the disruption of microbial homeostasis, thereby leading to the development of periodontal disease, even when they are present only in low no..

For example, specific pathogen-free mice exposed to *P. gingivalis* developed periodontal bone loss even when the pathogen was present in less than 0.1% of the total microbiota.

P. gingivalis subverts the host immune system and changes the microbial composition of dental plaque, ultimately leading to periodontal bone loss.

On this basis, P. gingivalis was labeled a "keystone" pathogen.

Keystone Pathogen Hypothesis and Polymicrobial Synergy and Dysbiosis Model

Three "keystone pathogen" mechanisms are proposed for *P.gingivalis*.

1-*P. gingivalis* can manipulate the Toll-like receptor 4 (TLR4) response. *P. gingivalis* can act as an agonist of TLR4, activating the immune system, or as an antagonist of TLR4, lowering the immune response

Orally, the most important source of iron is hemin from the GCF. So, during inflammation, GCF increases, and *P. gingivalis* acts as antagonist of TLR4, thus inhibiting the immune response.

2-*P. gingivalis* can inhibit the synthesis of IL-8 (also in response to other bacteria). "local chemokine paralysis" and leads to delayed polymorphonuclear leukocytes transmigration, impairing the host to face the microbial challenge.

3-*P. gingivalis* is able to interfere with the complement system, a component of the innate immune.

Keystone Pathogen Hypothesis and Polymicrobial Synergy and Dysbiosis Model

This hypothesis can extended to include the concept of polymicrobial synergy and dysbiosis model of disease.

So, interspecies communication between keystone pathogens and other members of the community (known as *accessory pathogens*) is considered one important factor that leads to overgrowth of the more pathogenic microbiota and to a dysbiotic microbial community.

examples:

Oral streptococci and more pathogenic organisms including *P.gingivalis* and *A. actinomycetemcomitans*.

Complicating Factors

-The periodontal microbiota is a complex community of microorganisms, many of which are still difficult or impossible to cultivate or identify(specially during different phases of the disease progression).

-The chronic nature of periodontal disease has complicated the search for bacterial pathogens.

-It was previously thought that periodontal diseases progressed at a slow but steady rate.

-The disease progresses at different rates, with alternating episodes of rapid tissue destruction and periods of remission.

Dental Calculus

The primary cause of gingival inflammation is bacterial plaque.

Other predisposing factors include calculus, faulty restorations, complications associated with orthodontic therapy, self-inflicted injuries, and the use of tobacco.

Calculus consists of mineralized bacterial plaque that forms on the surfaces of natural teeth and dental prostheses.

It is of two types:-

Supragingival and Subgingival Calculus

Calculus

Supragingival calculus is located coronal to the gingival margin and therefore is visible in the oral cavity.

It is usually white or whitish yellow in color; hard, with a claylike consistency; and easily detached from the tooth surface

The color is influenced by contact with substances such as tobacco a food pigments.

It may be localized on a single tooth or group of teeth, or it may be generalized throughout the mouth.

The two most common locations for the development of supragingival calculus are the buccal surfaces of the maxillary molars and the lingual surfaces of the mandibular anterior teeth.

Saliva from the parotid gland flows over the facial surfaces of the upper molars via the parotid duct, whereas the submandibular duct and the lingual duct empty onto the lingual surfaces of the lower incisors from the submaxillary and sublingual glands, respectively.





Fig. 3-37. Abundance of supragingival calculus deposits. (a) Gross deposits as a result of long-term neglect of oral hygiene. Two mandibular incisors have been exfoliated. (b) Supragingival plaque usually covering the lingual aspect of mandibular incisors. Note the intense inflammatory reaction adjacent to the deposits. (c) Same patient and region as in Fig. 3-37b following removal of the calculus. The gingival tissues demonstrate healing.

SUBGINGIVALCalculus

Subgingival calculus is located below the crest of the marginal gingiva and therefore is not visible on routine clinical examination.

It's location and the extent may be evaluated by careful tactile perception with a delicate dental instrument such as an explorer.

Subgingival calculus is typically hard and dense; it frequently appears to be dark brown or greenish black in color.

It is firmly attached to the tooth surface.

Microscopically, subgingival calculus usually extend nearly to the base of periodontal pockets in individuals with chronic periodontitis but do not reach the junctional epithelium.



SI. no.	Feature	Supragingival calculus	Subgingival calculus	
1	Defined as	Tightly adhering calculus deposit that forms on the crowns of the teeth coronal to the gingival margin	Calcified deposit that forms on the tooth surface below the free margin of gingiva	
2	Location	Forms coronal to the gingival margin	Deposits present apical to the crest of marginal gingiva	
3	Source	Derived from the salivary secretions - salivary calculus	Derived from the gingival exudate - seruminal calculus	
4	Distribution	Symmetrical arrangement on teeth, more on facial surfaces of maxillary molars and lingual surfaces of mandibular anterior teeth	Related to pocket depth, heavier on proximal surfaces	
5	Color	It is white, yellow in color	Brown/greenish black in color	
6	Consistency	Hard and clay like	Hard and firm/flint or glass like	
7	Composition	More brushite and octa calcium phosphate Less magnesium whitelockite	Less brushite and octa calcium phosphate. More magnesium whitelockite	
8	Other contents	Sodium content is less	Sodium content increases with the depth of the pocket	
		Salivary proteins are present	Salivary proteins are absent	
9	Visibility	Clinically visible	Not visible on routine clinical examination	
10	Attachment	Easily detached from the tooth	Firmly attached to the tooth surface	



Composition of Calculus

Inorganic Content (70% - 90%) The organic components constitute the rest. The major inorganic proportions 76% calcium phosphate (Ca3[PO4]2) 3% calcium carbonate (CaCO3), 4% magnesium phosphate (Mg3[PO4]2) 2% carbon dioxide Traces of other elements such as sodium, zinc, strontium,

bromine, copper,manganese, tungsten, gold, aluminum, silicon, iron, and fluorine.

The percentage of inorganic constituents in calculus is similar to that of other calcified tissues of the body.



At least 2/3 of the inorganic component is crystalline in structure.

Composition of Calculus

The four main crystal forms are :

hydroxyapatite	58%	Ca5 (PO4)3 X OH
magnesium whitlockite	21%	$Ca_9(Mg\underline{Fe})(PO_4)_6PO_3OH$
octacalcium phosphate	12%	Ca4H (PO4)3 X 2 H2O
Brushite	9%	CaH (PO4) x 2 H2O

Hydroxyapatite and octacalcium phosphate are detected most frequently (i.e., in 97% - 100% of all supragingival calculus).

Brushite is more common in the mandibular anterior region, and magnesium whitlockite is found in the posterior areas.

The incidence of the four crystal forms varies with the age of the deposit.



Composition of Calculus

Organic Content

It consists of a mixture of protein–polysaccharide complexes, desquamated epithelial cells, WBC, and various types of microorganisms.

Between 2% -9 % is carbohydrate, which consists of galactose, glucose, rhamnose mannose, glucuronic acid, galactosamine, and sometimes arabinose, galacturonic acid, and glucosamine.

All of these organic components deriver from salivary glycoprotein, with the exception of arabinose and rhamnose.

Salivary proteins account for 6-8 % include most amino acids.

Lipids account for 0.2%.



Composition of Calculus

Organic Content

The composition of subgingival calculus is similar to that of supragingival calculus, with some differences.

It has the same hydroxyapatite content but more magnesium whitlockite and less brushite and octacalcium phosphate.

The ratio of calcium to phosphate is higher in subgingival calculus, and the sodium content increases with the depth of periodontal pockets.

The origin of subgingival calculus is plasma, whereas supragingival calculus is partially composed of salivary constituents.

Saliva is the primary source of mineralization for supragingival calculus, whereas the serum transudate called GCF furnishes the minerals for subgingival calculus.

Salivary proteins present in supragingival calculus are not found in subgingival calculus.

Dental calculus, salivary duct calculus, and calcified dental tissues are similar in inorganic composition.



Attachment to the Tooth Surface

Differences in the manner in which calculus is attached to the tooth surface affect the relative ease or difficulty encountered during its removal.

Four modes of attachment:-

(1) Attachment by means of an organic pellicle on cementum.

(2) Mechanical locking into surface irregularities, such as

caries lesions or resorption lacunae.

(3)Close adaptation of the undersurface of calculus to depressions or gently sloping mounds of the unaltered cementum surface.

(4) Penetration of bacterial calculus into cementum.

CALCULUS ATTACHMENT

THE FOLLOWING 4 MODES OF ATTACHMENT HAS BEEN DESCRIBED:-



Attachment by means of organic pellicle on enamel



Mechanical interlocking in emental resorption lacunae



Close adaptation of calculus undersurface depressions to gently sloping mounds on the unaltered cementum surface.



Penetration of calculus bacteria in cementum. But this mode of attachment was not acknowledged





Calculus Formation

Calculus is mineralized dental plaque.

The soft plaque is hardened by the ppt of mineral salts, which usually starts between the 1st and 14th days of plaque formation.

Calcification has to occur within 4 - 8 hours.

Calcifying plaques may become 50% mineralized in 2 days

60%-90% mineralized in 12 days.

All plaque does not necessarily undergo calcification.

Microorganisms are not always essential in calculus formation.

Saliva is the primary source of mineralization for supragingival calculus, whereas the *GCF* furnishes the minerals for subgingival calculus.

The calcium concentration in plaque is 2 - 20 times higher than in saliva.

Phosphorus may be more critical than calcium in plaque mineralization because early plaque of heavy calculus formers contains more calcium and **three** times more phosphorus than that of non-calculus formers.
Calculus Formation

The calcification of supragingival plaque and the attached component of subgingival plaque begins along the inner surface adjacent to the tooth structure.

Separate foci of calcification increase in size and coalesce to form solid masses of calculus

For the initial mineralization process to occur, calcium phosphate supersaturation, certain membrane-associated components, and regulation of nuclear inhibitors are required.Calcification may be accompanied by alterations in the bacterial content and staining qualities of the plaque.

As calcification progresses, the number of filamentous bacteria increases, and the foci of calcification change from basophilic to eosinophilic.

There is a reduction in the staining intensity of groups that exhibit a positive periodic acid–Schiff reaction.

Calculus Formation

Calculus is formed in layers, which are often separated by a thin cuticle.

The initiation of calcification and the rate of calculus accumulation vary among individuals, among tooth variety in the same dentition, and at different times in the same person.

So, persons may be classified as *heavy, moderate,* or *slight* calculus formers or as non-calculus formers.

The average daily increment in calculus formers is from 0.10% - 0.15% of dry wt..

Calculus formation continues until it reaches a maximum, after which it may be reduced in amount.

The time required to reach the maximal level is between 2.5 -6 months.

Reversal phenomenon is due to mechanical wear of bulky calculus due to food effects and action of the cheeks, lips, and tongue.

Theories of Calculus Formation

Plaque becomes mineralized by two categories.

1. **Mineral precipitation** results from a local rise in saturation of calcium and phosphate ions, done by:

• A rise in the pH of the saliva causes the precipitation of calcium phosphate salts.

The pH may be elevated by the loss of carbon dioxide and the formation of ammonia by dental plaque bacteria or by protein degradation during stagnation.

• Colloidal proteins in saliva bind calcium and phosphate ions and maintain a supersaturated solution.

• Phosphatase enzyme(liberated from dental plaque, desquamated epithelial cells, or bacteria) precipitates calcium phosphate by hydrolyzing organic phosphates in saliva, thereby increasing the concentration of free phosphate ions.

Esterase (present in the cocci and filamentous organisms and desquamated epithelial cells of dental plaque) may initiate calcification by :

hydrolyzing fatty esters \rightarrow free fatty acids \rightarrow soaps with calcium and magnesium \rightarrow the less-soluble calcium phosphate salts.

Mechanism 2



Theories of Calculus Formation

2. Seeding agents induce small foci of calcification that enlarge and coalesce to form a calcified mass.

This concept has been referred to as the *epitactic concept* or , *heterogeneous nucleation*.

The seeding gents in calculus formation are not known.

The intercellular matrix of plaque plays an active role.

The carbohydrate–protein complexes may initiate calcification by removing calcium from the saliva (chelation) and binding with it to form nuclei that induce the subsequent deposition of minerals.

Role of Microorganisms in Calculus

Predominant flora are cocci, bacilli and filaments (especially in the outer layers), and occasionally spiral organisms.

The bacteria near the enamel surface tend to have a reduced cytoplasm-to-cell wall ratio, (metabolically inactive).

Supragingival calculus contains more Grampositive organisms, whereas subgingival calculus tends to contain more Gramnegative species.

Role of Microorganisms in the Mineralization of Calculus

Mineralization of plaque generally starts extracellularly around both gram-positive and gram-negative organisms, but it may also start intracellularly.

Filamentous organisms, diphtheroids, and *Bacteriods* and *Veillonella* spp have the ability to form intracellular apatite crystals .

Mineralization spreads until the matrix and the bacteria are calcified.

Bacterial plaque may actively can mineralize calculus by forming phosphatases, which change the pH of the plaque and induce mineralization .

Most researchers think that bacteria are only passively involved and are simply calcified with other plaque components.

Factors increasing rate of calculus formation

- 1. Elevated salivary pH
- 2. Elevated salivary calcium conc.
- 3. Elevated bacterial protein and lipid concentration.
- 4. Elevated concentration of protein and urea in submandibular salivary gland secretion.
- 5. Higher total salivary lipid level.

Clinical Significance of Calculus

A positive correlation between calculus and the prevalence of **a** gingivitis exists, but this correlation is not as great as that between plaque and gingivitis.

The initiation of periodontal disease in young people is closely related to plaque accumulation, whereas calculus accumulation is more prevalent in chronic periodontitis found is older adults.

The incidence of calculus, gingivitis, and periodontal disease increases with age.

It is extremely rare to find periodontal pockets in adults without at least some subgingival calculus being present(even at microscopic levels). Calculus does not contribute directly to gingival inflammation, but it provides a fixed nidus for the continued accumulation of bacterial plaque and its retention in close proximity to the gingiva.

Periodontal pathogens such as *A.actinomycetemcomitans*, *P.gingivalis*, and *T.denticola* are present within the structural channels and lacunae of supragingival and subgingival calculus.

Clinical Significance of Calculus

Subgingival calculus is likely to be the product rather than the cause of periodontal pockets.

Plaque \rightarrow gingival inflammation \rightarrow pocket formation \rightarrow a sheltered area for plaque and bacterial accumulation.

The increased flow of GCF associated with gingival inflammation provides the minerals that mineralize the continually accumulating plaque, resulting in the formation of subgingival calculus.

The areas with detectable subgingival calculus were much more likely to have a loss of periodontal attachment than sites that did not have subgingival calculus.

So, removal of subgingival plaque and calculus are the cornerstones of periodontal therapy.

Calculus plays an important role in maintaining periodontal disease by keeping plaque in close contact with the gingival tissue and by creating areas where plaque removal is impossible.

Periodontal ligament



By: Dr.Ghayath Aljawady B.D.S, M.Sc., Ph.D in periodontics

2021-2022

DEFINITION:

it is the dense fibrous connective tissue that occupies the periodontal ligament space between the roots of teeth and alveolus. It is derived from the dental follicle above alveolar crest and is continuous with connective tissue of gingiva and the apical foramen which is further continuation with dental pulp.





SHAPE AND WIDTH OF PDL

Width variable = average 0.15 mm- 0.38mm.

• Thinnest around the middle third of the root & widens both apically and near the crest(WIDEST AREA).



deciduous teeth.

□ strong force on the teeth (within physiological limit).

While

Width

in

□ Non functional teeth.

Unerupted teeth.



HOUR GLASS



Development OF PDL



- As the crown approaches the oral mucosa during tooth eruption, the fibroblasts of dental follicle become active and start producing collagen fibrils. These fibers initially lack orientation, but they soon acquire an orientation oblique to the tooth.
- The first collagen bundles appear in the region immediately apical to the cemento-enamel-junction and give rise to the gingivodental fiber groups.
- As tooth eruption progresses, additional oblique fibers appear and become attached to the newly formed cementum and bone.
- The transseptal and alveolar crest fibers develop when the tooth merges into the oral cavity.
- Alveolar bone deposition occurs simultaneously with periodontal ligament organization.
- During eruption, cemental Sharpey's fibers appear first, followed by Sharpey's fibers emerging from bone.
- Sharpey's fibers of bone are fewer in number and more widely spaced than those emerging from the cementum.
- At a later stage, alveolar fibers extend into the middle zone to join the lengthening cemental fibers and attain their classic orientation, thickness and strength when occlusall function is established.

COMPOSITION OF PDL:

Histological structure The periodontal ligament is formed of :



Cells of PDL

The cells of PDL may be divided into

1) Synthetic cells

- FIBROBLASTS--- SYNTHSISE COLLAGEN
- OSTEOBLASTS
- CEMENTOBLASTS

2) Resorptive cells

- FIBROBLASTS---DEGRADE COLLAGEN
- OSTEOCLASTS
- CEMENTOCLASTS

3) **Progenitor cells**

- CAPABLE OF UNDERGOING MITOTIC DIVISION
- ADJACENT TO BLOOD VESSELS

4) Epithelial Rests of Malassez

REMNANTS OF HERTWIGS EPITHELIAL ROOT SHEATH IN
 PDL.

5) Defense cells

- MAST CELLS: REGULATE ENDOTHELIAL AND
 FIBROBLAST PROLIFERATION
- MACROPHAGES: PHAGOCYTOSE DEAD CELLS SECRETE GROWTH FACTORS TO REGULATE FIBROBLAST PROLIFERATION
- EOSINOPHILS: PHOGCYOSIS FUNCTION



EXTRACELLULAR STRUCTURES

Periodontal Ligament Fibers

- •Mainly collagen and oxytalan fibers.
- •Elastic fibers are present only in the wall of blood vessels
- •Oxytalan are immature elastic fiber they support the blood vessels of periodontal ligament.
- •Types I, III,V, VI, XII of collagen are present in periodontal ligament it is synthesized by fibroblasts, chondroblasts, osteoblasts, odontoblasts.
- •It is secreted in an inactive form called as procollagen which converted into tropocollagen then polymerized into collagen fibrils and aggregated into collagen bundles by the formation of cross-linkages.
- •There is rapid turnover rate of periodontal ligament collagen, with half life of only 10–15 days, which is about 5 times faster than gingival collagen.

EXTRACELLULAR STRUCTURES

GROUND SUBSTANCE:

COMPONENTS OF THE GROUND SUBSTANCE:

IT CONSISTS OF TWO MAIN COMPONENTS:

1) GLYCOSAMINOGLYCANS SUCH AS HYALURONIC ACID AND PROTEOGLYCANS

2) GLYCOPROTEINS SUCH AS FIBRONECTIN AND LAMININ

IT ALSO HAS A HIGH WATER CONTENT (70%).

PERIODONTAL LIGAMENT

FIBERS



 Alveolar crest group. Alveolar crest fibers extend obliquely from the cementum just beneath the junctional epithelium to the alveolar crest. Fibers also run from the cementum over the alveolar crest and to the fibrous layer of the periosteum covering the alveolar bone. The alveolar crest fibers prevent the extrusion of the tooth and resist lateral tooth movements. The incision of these fibers during periodontal surgery does not increase tooth mobility unless significant attachment loss has occurred.
 Horizontal group. Horizontal fibers extend at right angles to the long axis of the tooth from the cementum to the alveolar bone.

3) Oblique group. Oblique fibers, the largest group in the periodontal ligament, extend from the cementum in a coronal direction obliquely to the bone. They bear the impact of vertical masticatory stresses and transform them into tension on the alveolar bone.

4) Apical group. The apical fibers radiate in a rather irregular manner from the cementum to the bone at the apical region of the socket. They do not occur on incompletely formed roots.
5) Interradicular group. The interradicular fibers fan out from the cementum to the tooth in the furcation areas of multirooted teeth.

SHARPEYS' FIBERS

COLLAGEN FIBERS ARE EMBEDDED INTO THE CEMENTUM ON ONE SIDE OF THE PERIODONTAL SPACE & INTO THE ALVEOLAR BONE ON THE OTHER.

THE EMBEDDED FIBERS ARE CALLED SHARPEY'S FIBERS.

THESE ARE THE MOST NUMEROUS BUT SMALLER AT THEIR ATTACHMENT INTO CEMENTUM THAN ALVEOLAR BONE. SHARPEY'S FIBERS IN PRIMARY ACELLULAR CEMENTUM ARE MINERALIZED FULLY THOSE IN CELLULAR CEMENTUM AND BONE ARE MINERALIZED PARTIALLY AT THEIR PERIPHERY.

FEW SHARPEY'S FIBERS PASS UNINTERRUPTEDLY THROUGH THE BONE OF ALVEOLAR PROCESS TERMED (TRANSALVEOLAR FIBERS)TO CONTINUE AS PRINCIPAL FIBERS OF ADJACENT PERIODONTAL LIGAMENT OR MIXED BUCCALLY OR LINGUALLY WITH FIBERS OF PERIOSTEUM THAT COVER THE OUTER PLATES OF ALVEOLAR PROCESS.

THESE FIBERS PASS THROUGH THE ALVEOLAR PROCESS ONLY WHEN PROCESS CONSISTS ENTIRELY OF COMPACT BONE AND CONTAINS NO HAVERSIAN SYSTEM. ONCE EMBEDDED IN EITHER THE WALL OF ALVEOLUS OR THE TOOTH SHARPEY'S FIBERS CALCIFY TO CERTAIN DEGREE & ARE ASSOCIATED WITH ABUNDANCE OF NON COLLAGENOUS PROTEINS NAMELY OSTEOPONTIN AND BONESIALOPROTEIN.

INTERMEDIATE PLEXUSES :

It was believed that principal fibers frequently followed a wavy course from cementum to alveolar bone and are joined in the mid region of periodontal space giving rise to a zone of distinct appearance called INTERMEDIATE PLEXUSES

The plexuses was considered to be an area of high metabolic activity in which splicing and unsplicing of fibers might occur. Studies have indicated that once cemental fibers meet and fuse with the bone no such plexuses remains. **Functions of the periodontal ligament**

✓ Physical Function

✓ Formative and Remodeling Function

Nutritional and Sensory Functions

Physical Functions

- Provision of a soft tissue "casing" to protect the vessels and nerves from injury by mechanical forces.
- □ Transmission of occlusal forces to the bone.
- Attachment of the teeth to the bone.
- □ Maintenance of the gingival tissues in their proper relationship to the teeth.
- □ Resistance to the impact of occlusal forces (shock absorption)

- > Light forces are absorbed by intravascular fluid that is forced out of the blood vessels
- Moderate forces are also absorbed by extravascular tissue fluid that is forced out of the periodontal ligament space into the adjacent marrow spaces
- > The heavier forces are taken up by the principal fibers

TRANSMISSION OF OCCLUSAL FORCES TO THE BONE

THE ARRANGEMENT OF THE PRINCIPAL FIBRES IS SIMILAR TO A SUSPENSION BRIDGE OR HAMMOCK .

WHEN A HORIZONTAL OR TIPPLING FORCES IS APPLIED TWO PHASES OF TOOTH MOVEMENT OCCUR :

1 ST : WITH IN THE CONFINES OF THE PERIODONTAL LIGAMENT.

2 ND : PRODUCES A DISPLACEMENT OF FACIAL AND LINGUAL BONY PLATES IN SINGLE ROOTED TEETH THE AXIS OF ROTATION IS LOCATED IN THE AREA BETWEEN THE APICAL THIRD AND THE MIDDLE THIRD OF THE ROOT.

IN MULTIROOTED TEETH, THE AXIS OF ROTATION IS LOCATED IN THE BONE BETWEEN THE ROOTS

RESISTANCE TO THE IMPACT OF OCCLUSAL FORCES (Shock absorption):

Theories have been proposed to describe the mechanism of tooth support

A. TENSIONAL THEORY The tensional theory of tooth support describes to the principal fibers of the periodontal ligament. The major responsibility in supporting the tooth and transmitting forces to the bone.

WHEN

Force is applied to the crown THE

Principal fibers unfold and straightened AND Transmit the forces to alveolar bone

THEN Elastic deformation of the bony socket AND Finally when the alveolar bone has reached its limit, the load is transmitted to the basal bone

B.VISCOELASTIC Theory

It considers the displacement of the tooth to be largely controlled by fluid movements, with fibers having only a secondary role.

When forces are transmitted to the tooth, the extra cellular fluid passes from the periodontal ligament into the marrow spaces of bone through foramina in the cribri form plate.

After depletion tissue fluids, the fiber bundles absorb the slack and tighten. This leads to blood vessel stenosis. THEN Arterial back pressure causes ballooning of the vessels, and passage of blood ultra filtrates into the tissues, there by replacing the tissue fluids.

Formative and Remodeling Function OF PDL

Cells of the periodontal ligament participate in the formation and resorption of cementum and bone which occur:

in physiologic tooth movement;

in the accommodation of the periodontium to occlusal forces;

and

in the repair of injuries.

The periodontal ligament is constantly undergoing remodeling.

Old cells and fibers are broken down and replaced by new ones, and mitotic activity can be observed in the fibroblasts and endothelial cells.

Fibroblasts form the collagen fibers and may also develop into osteoblasts and cementoblasts.

Nutritional and Sensory Functions OF PDL

- Supplies nutrients to the cementum, bone, and gingiva by way of the blood vessels and provides lymphatic drainage.
- Supplied with sensory nerve fibers capable of transmitting tactile, pressure, and pain sensations by the trigeminal pathways.

Nerve bundles pass into the periodontal
ligament from the periapical area and through
channels from the alveolar
bone that follow the
course of the blood vessels.

HOMEOSTATIC MECHANISM

The resorption and synthesis are controlled procedures. If there is a long term damage of periodontal ligament, which is not repaired, the bone is deposited in the periodontal space. This results in obliteration of space and ankylosis between bone and the tooth.

The quality of tissue changes if balance between synthesis and resorption is disturbed

If there is deprivation of Vit. C which are essential for collagen synthesis, resorption of collagen will continue. So there is progressive destruction and loss of extra cellular substance of ligament. This occurs more on bone side of ligament. Hence, loss of attachment between bone and tooth and at last, loss of tooth.

Thickness of Periodontal Ligament

1- AGE, LOCATION OF THE TOOTH, AND DEGREE OF STRESS TO WHICH THE TOOTH WAS SUBJECTED.

- 2- THE MESIAL SIDE IS THINNER THAN DISTAL SIDE.
- 3- A TOOTH THAT IS NOT IN FUNCTION HAS A THIN PERIODONTAL LIGAMENT.
- 4- A TOOTH IN FUNCTIONAL OCCLUSION HAS A PERIODONTAL LIGAMENT SPACE OF APPROXIMATELY 0.25 MM, PLUS OR MINUS 0.10 MM.
- 5- A TOOTH SUBJECTED TO ABNORMAL STRESS HAS A CONSIDERABLY THICKER PERIODONTAL SPACE.

blood supply of periodontal ligament

THE BLOOD SUPPLY IS DERIVED FROM THE INFERIOR AND SUPERIOR ALVEOLAR ARTERIES AND REACHES THE PERIODONTAL LIGAMENT FROM 3 SOURCES:

(I) APICAL VESSELS

(II) PENETRATING VESSELS FROM THE ALVEOLAR BONE

(III) ANASTOMOSING VESSELS FROM THE GINGIVA

•THEY ARE PRESENT IN THE INTERSTITIAL SPACES OF LOOSE CONNECTIVE TISSUE BETWEEN THE PRINCIPAL FIBERS WHICH RUNS LONGITUDINALLY CONNECTED IN THE NET LIKE PLEXUS CLOSER TO THE BONE THAN CEMENTUM.

•THE CAPILLARIES OF PERIODONTAL LIGAMENT ARE FENESTRATED WHILE IN OTHER CONNECTIVE TISSUES THEY ARE CONTINUOUS. DUE TO FENESTRATION, THEY HAVE GREATER ABILITY OF DIFFUSION AND FILTRATION WHICH IS RELATED TO HIGH METABOLIC REQUIREMENTS OF PERIODONTAL LIGAMENT AND ITS HIGH RATE OF TURNOVER.

Clinical considerations of periodontal ligament

1) Restorative dentistry The supporting tissues of a tooth long out of function are poorly adapted to carry the load suddenly placed on a tooth by restoration e.g bridge abutments, teeth opposing bridges/dentures, teeth used as anchorage for removable bridges

• Therefore, the patient cannot use the tooth immediately

• After restoration supporting tissues take some time to adapt to new functional demands. Therefore after restoration an adjustment period should be allowed

• Gradually use the tooth first for soft food before the tooth is put to full use to bite hard food.

2) Orthodontic therapy • Orthodontic therapy also affects PDL similar to alveolar process

- On the side under tension, the PDL space will become wider
- With the side under pressure it will become narrower
- Interdental ligament responsible for memory of a tooth positioning within each dental arch
- Therefore a prolonged retention period must be allowed to reattach the interdental ligament fully to its new position TO ensure maintenance of clinical stability of tooth position
- Retainers used to maintain this desirable alignment

• 3) Periapical granuloma and cyst

- PDL in the periapical area of tooth often site of a pathologic lesion
- Inflammatory disease of pulp(pulpitis) progresses to the apical periodontal ligament and replace its fiber bundles with granulation tissue. This lesion is called periapical granuloma
- The periapical granuloma contain epithelial cells which undergo proliferation and produce a cyst

4) Chronic inflammatory periodontal disease

- Commonest pathology related to periodontal disease
- Dental plaque –toxins released from bacteria in plaque and metabolites of host defense mechanisms destroy PDL and adjacent bone very frequently LEAD TO tooth mobility and loss of tooth
- Repair of destroyed PDL challenging
- Involves limiting the disease process and to regenerate host tissues (pdl and bone to their original form so that reattachment of pdl to bone becomes possible

2021-2022

THANKS

Plaque biofilm control for the periodontal patient

Mechanical and chemical



By: Dr.Ghayath Aljawady B.D.S, M.Sc., Ph.D in periodontics







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2021-2022
Outline for the lecture

Plaque biofilm control for the periodontal patient

- The toothbrush:
- o Toothbrush design
- Powered toothbrushes
- Dentifrices
- Toothbrushing methods
- Interdental cleaning aids:
- o Dental floss
- o Interdental brushes
- o Other interdental cleaning devices
- Oral irrigation:
- o Supragingival irrigation
- o Subgingival irrigation
- Caries control



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- Mechanical plaque control is the most widely accepted and the most dependable mode of controlling microbial plaque.
- □Correct hygiene procedures for oral cavity can prevent dental caries and periodontal disease.
- □The aids that help in performing mechanical plaque control are the toothbrushes and the interdental cleansing aids. When properly performed in a systematic manner and in regular intervals, mechanical methods can control biofilm build-up.

Brushing:





• People brush their teeth for a number of reasons: to feel fresh and confident, to have a nice smile, and to avoid bad breath and disease

- ♦ Tooth brushing is currently the most commonly implemented measure in oral hygiene practices. when used properly, has no side effects, is easy to use, and is inexpensive
- Two types of brush: Manual tooth brush and electric.
- The American Dental Association (ADA) recommends that individuals should brush twice per day and use floss or other interdental cleaners once per day to effectively remove microbial plaque biofilms and prevent gingivitis.
- Toothbrushes vary in size and design, as well as in length, hardness, and arrangement of the bristles.



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The American dental association (ADA) has described the range of dimensions of acceptable brushes. These have

- A brushing surface from 1-1¼inches (25.4-31.8mm) long and 5/16 to 3/8 inch (7.9-9.5mm) wide 2-4 rows of bristles, and 5 to 12 tufts per row.
- Short-headed, relatively dense bristle bundles and soft to medium bristles with bristle thickness of 0.18 to 0.25 mm and bristle length ranging from 10 to 12mm are recommended.
- Rounded and soft bristles are recommended as they are less damaging than hard and stiff bristles.
- > The desired diameter of the bristle is 0.007inch (recommended by bass).
- \succ It is recommended to replace the brush every three months.





Ideal manual tooth brush:

- 1- Handle size appropriate to user's age and dexterity.
- 2- Head size appropriate to the size of the individual patient's requirements.
- 3- End-rounded nylon or polyester filaments.
- 4- Soft filament configurations.

5- Filament patterns that enhance plaque removal in the appropriate spaces and along the gum line.

6- In addition to: inexpensive, durability, imperviousness to moisture, and easy to use.

- Powered toothbrushes





They are electromechanical brushes and sonic brushes. The newer generation of powered toothbrushes removes plaque better than manual toothbrushes.

- ♦ They are especially useful when patient lack manual dexterity.
- They remove plaque faster than manual brushes.
- They have a rotating, oscillating or sonic action to remove plaque and reduce gingivitis. Other modes of action like side to side, counter-oscillation, circular actions are less consistent in plaque removal.
- ♦ Sonic brushes remove stains from teeth.
- ◆ The 2 minutes timer in the powered brushes helps the patient brush the teeth adequately which they seldom do when brushing manually.
- They can be used around implants for plaque control.





Powered toothbrushes

➢ Powered toothbrushes have been shown to improve oral health for the following:

- (1) children and adolescents.
- (2)people with physical or mental disabilities
- (3) hospitalized patients, including older adults who require the assistance of caregivers for hygiene.
- (4) patients with fixed orthodontic appliances



Dentifrice:

- \blacktriangleright dentifrices aid in cleaning and polishing tooth surfaces
- ➢ Facilitate removal of dental plaque& stain by increase mechanical friction due to presence of abrasive particles.
- > Appropriate size of abrasive particles; neither large nor small.
- ➤ The contents of abrasives:(silicone oxide or aluminum oxide and granular polyvinyl chlorides), water, soap, flavoring, sweetening agent & therapeutic agents (fluoride, pyrophosphates), coloring & preservatives
- ➤ Insoluble inorganic salts ;20-40%.
- > Tooth powder much more abrasive than paste that contain 95% abrasive materials.
- \succ Fluoride ion must be in a mount 1000 to 1100 (ppm) to achieve caries reduction effects.
- ➤ They are used mostly in the form of pastes, although powders and gels are also available.









o Toothbrushing methods:



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Bass /Sulcular brushing method:

1. Place the head of a soft brush parallel to the occlusal plane, with the brush head covering three to four teeth. This hygiene procedure begins at the most distal tooth in the arch and systematically proceeds mesially.

2. Place the bristles at the gingival margin, pointing at a 45- degree angle to the long axis of the teeth.

3. Exert gentle vibratory pressure using short, back-and-forth motions without dislodging the tips of the bristles. This motion forces the bristle ends into the gingival sulcus area, as well as partly into the interproximal embrasures. The pressure should be firm enough to blanch the gingiva



Place bristles along the gumline at a 45° angle. Bristles should contact both the tooth surface and the gumline.



Gently brush the outer tooth surfaces of 2-3 teeth using a vibrating back, forth & rolling motion. Move brush to the next group of 2-3 teeth and repeat.



Maintain a 45° angle with bristles contacting the tooth surface and gumline. Gently brush using back, forth & rolling motion along all of the inner tooth surfaces.

Tilt brush vertically behind the front teeth. Make several up & down strokes using the front half of the brush.



Place the brush against the biting surface of the teeth & use a gentle back & forth scrubbing motion. Brush the tongue from back to front to remove odor-producing bacteria.

o Toothbrushing methods:



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Modified Bass technique:

It is the most commonly taught technique in clinical lt practise. employs а circular vibratory motion on the buccal and lingual surfaces of the teeth instead of the back and forth motion of the bass technique.

Modified Bass Technique



Place bristles along the gumline at a 45° angle. Bristles should contact both the tooth surface and the gumline.



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o Toothbrushing methods:

Stillman's technique:

The bristles are positioned on the attached gingiva and directed apically at 45-degree angle to the long axis of the tooth. The brush is activated by short back and forth mini-scrub vibratory strokes.

♦ It stimulates the gingiva. It is used in cases of progressive gingival recession. It is sometimes suggested for use after periodontal plastic surgery.

Modified Bass Technique



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o Toothbrushing methods:

Charters technique:

The bristles are directed towards the occlusal surface at a 45-degree angle to the long axis of the tooth. The brush is activated with short back and forth strokes.
It is recommended for temporary cleaning in areas of healing after periodontal surgery. It is also advised in the presence of orthodontic appliances and fixed prosthesis.





o Toothbrushing methods:

Leonard technique:

 An "up and down" vertical brushing movement is employed.







o Toothbrushing methods:

Fones technique:

Bristles are placed perpendicular to the buccal and lingual surfaces and the brush is moved in circular motion.
 It is easy to learn and is

used in young children with primary teeth.







Targeted hygiene focuses brushing efforts on the critical cervical and interproximal areas of teeth.

- Brushing with either a manual or powered toothbrush requires a systematic routine to be effective.
- Patients will modify any technique to their needs, so emphasize brushing all the surfaces of the teeth effectively.





Interdental Cleaning Aids

- Brushing techniques can efficiently clean biofilm present on free surfaces of teeth like occlusal, buccal and lingual surfaces.
- However, most of the periodontal and carious lesions begin from interproximal regions and brushing is ineffective in cleaning those regions. Hence, flossing, rubber stimulators, proximal brushes, single-bristle brushes and irrigators may be needed to achieve adequate cleaning in proximal regions.



Interdental Cleaning Aids

Dental floss:

- Dental floss is the most widely recommended tool for removing biofilm from proximal tooth surfaces.
- Floss is made from nylon filaments or plastic monofilaments, and it comes in waxed, unwaxed, thick, thin, and flavored varieties. Some prefer monofilament floss made of nonstick material because they are slick and do not fray.
- ❑ Factors influencing the choice of dental floss include the tightness of tooth contacts, the roughness of proximal surfaces, and the patient's manual dexterity, not the superiority of any one product. Therefore, recommendations about type of floss should be based on ease of use and personal preference







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Interdental Cleaning Aids

Technique for the Use of Dental floss

The floss must contact the proximal surface from line angle to line angle to clean effectively. It must also clean the entire proximal surface, including accessible subgingival areas. Flossing technique requires the following:

1. 12 to 18 inches is usually sufficient to grasp securely. It may be wrapped around the fingers, or the ends may be tied together in a loop.

2. Stretch the floss tightly between the thumb and forefinger or between both forefingers, and pass it gently through each contact area with a firm back-and-forth motion.

3. Once the floss is apical to the contact area between the teeth, wrap the floss around the proximal surface of one tooth and slip it under the marginal gingiva. Move the floss firmly along the tooth up to the contact area and gently down into the sulcus again, repeating this up-and-down stroke two or three times. Then move the floss across the interdental gingiva, and repeat the procedure on the proximal surface of the adjacent tooth.

4. Continue through the whole dentition, including the distal surface of the last tooth in each quadrant. When the working portion of the floss shreds or becomes contaminated, move the floss to a fresh portion.





Interdental Cleaning Aids















Interdental brushes

Small cone-shaped or tapered brushes.

It is inserted interdentally and moved back and forth in facio-lingual direction.

- ♦ It is used in large open embrasures. It can be used to control plaque accumulation in root concavities, proximal surfaces and furcation areas.
- ♦ The interdental brush is the most effective method for inter-dental plaque removal and hence is the device of choice for interproximal plaque removal.
- They require little manual dexterity when compared with dental flossing.

As they can repeatedly be used they are cost effective.





Interdental Cleaning Aids

Other interdental cleaning devices Rubber Tips,

They are conical in shape and are mounted on a handle. They are used similar to wooden toothpicks.

Wooden Toothpicks,

Made from soft-wood and can be round or triangular (similar to the shape of the interdental area) in shape.

- Indications: used in open contacts, furcation areas, just under gingival margins and around fixed orthodontic bands.
- Toothpick moved in and out or up and down direction.

Tufted Brushes





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Oral irrigation:

o Supragingival irrigation

- □Oral irrigators for daily home use function by directing a pulsating stream of water through a nozzle to the tooth surfaces.
- □Most often, a device with a built-in pump generates the pressure
- □Oral irrigators remove nonadherent bacteria and debris from the oral cavity more effectively than toothbrushes and mouthrinses.
- They are particularly helpful for removing debris from inaccessible areas around orthodontic appliances and fixed prostheses. When used as adjuncts to toothbrushing and interdental cleaning, these devices can have a beneficial effect on periodontal health by reducing the accumulation of microbial plaque biofilm, as well as by decreasing inflammation and pocket depth

o Subgingival irrigation

Oral irrigation:

o Supragingival irrigation

Technique

1. The common home-use irrigator tip is a plastic nozzle with a 90-degree bend at the tip attached to a pump providing pulsating beads of water at speeds regulated by a dial. Patients should be instructed to aim the pulsating jet across the proximal papilla, hold it there for 10 to 15 seconds, trace along the gingival margin to the next proximal space, and repeat the procedure.

2. The irrigator should be used for both the buccal and lingual surface.

3. Patients with gingival inflammation should start at low pressure, and they can increase the pressure comfortably to about medium as tissue health improves. Some individuals like to use the device on the highest pressure setting, with no reported harm. Patient comfort should be the guide for pressure setting.





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Oral irrigation:

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o Subgingival irrigation

- Subgingival irrigation can be performed both in the dental office and at home by the patient.
- ❑ Home irrigation is performed by aiming or placing the irrigation tip or a blunt syringe tip at least 3 mm into the periodontal pocket.
- Irrigation performed in the dental office, also called *lavage* or *flushing of the periodontal pocket,* as a one-time treatment after scaling and root planning.
- Subgingival irrigation with an oral irrigator using chlorhexidine diluted to one-third strength performed regularly at home and after scaling, root planing, and in-office irrigation therapy produced significant gingival improvement compared with control subjects.
- Subgingival irrigation has been shown to disrupt more than half the subgingival plaque biofilm65 and reach about half the depth of pockets, up to 7 mm, which is much farther apically than a toothbrush or floss can reach.





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Gingival irrigation can be a useful adjunct for periodontal patients who have residual pockets and complex dental architecture to clean every day.

• Supragingival irrigation reduces gingival inflammation and is easy to perform with water irrigation devices.

• Subgingival irrigation with specialized tips or blunt syringes for deep pockets and furcation areas is effective for maintaining residual pockets and furcation areas when it is used as part of the daily home care routine.



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Caries Control

- Dental caries, particularly root caries, is a problem for periodontal patients because of attachment loss and exposed root surfaces associated with the disease process and periodontal therapy procedures.
- Periodontal patients should be aware of caries, and the use of fluoride products minimizes the risk for caries.
- All periodontal patients should be encouraged to use a fluoride-containing toothpaste daily.
- Patients at high risk for caries should use higher-concentration fluoride toothpaste or gel until the caries risk is controlled. A lower concentration can be used during maintenance therapy.
- A periodic chlorhexidine rinsing regimen to control cariogenic bacteria in the oral cavity is part of the caries risk management program for high-risk individuals.
- Other considerations in caries control, such as diet and reduced salivary flow, should be evaluated, and modifications should be made where possible.





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- Chemical plaque biofilm control with oral rinses
- o Chlorhexidine digluconate:
- i- Mode of action
- ii- Clinical use
- iii- Side-effects
- o Nonprescription essential oil rinse
- o Other products
- Disclosing agents
- Patient motivation and education:
- o Motivation for effective plaque biofilm control
- o Education and scoring systems:

i- Plaque biofilm control record (O'Leary Index) ii- Bleeding points index

o Instruction and demonstration

Plaque biofilm control for the periodontal patientChemical plaque biofilm control with oral rinses



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To date, the ADA has accepted two agents for treatment of gingivitis: prescription solutions of *chlorhexidine digluconate oral rinse* and nonprescription *essential oil mouthrinse.*

The agent that has shown the most positive antibacterial results to date is chlorhexidine, a diguanidohexane with pronounced antiseptic properties
 Several clinical investigations confirmed an initial finding that two daily rinses with 10 mL of a 0.2% aqueous solution of chlorhexidine digluconate almost completely inhibited the development of microbial plaque biofilm, calculus, and gingivitis in the human model for experimental gingivitis.



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- Chemical plaque biofilm control with oral rinses

Chlorhexidine:

Mechanism of action:

- It is a cationic biguanide that acts by rupturing cell membranes and precipitation of cytoplasm.
- It is a broad spectrum anti-microbial with effects against gram positive and gramnegative bacteria, yeasts including candida and some lipophilic viruses like HIV and HBV.
- > It has bacteriostatic, bactericidal and fungicidal activity. Prolonged exposure increases the bactericidal effect.
- > It binds covalently to cutaneous and mucosal proteins with limited systemic absorption.
- > It does not cause any bacterial resistance and suprainfection on long-term usage.
- > It forms a white coating layer over the damaged area due to substantivity of chlorhexidine, which coagulates salivary and serum proteins forming a protective



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- Chemical plaque biofilm control with oral rinses

Chlorhexidine:

Drug interactions:

- * Patient should be advised to allow at least 30 minutes to lapse between use of a dentifrice and rinsing with chlorhexidine.
- Interactions of chlorhexidine with sodium lauryl sulfate and sodium monofluorophosphate (fluoride) contained in the dentifrice may reduce its effectiveness. It is incompatible with soaps and other anionic materials.



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- Chemical plaque biofilm control with oral rinses

Chlorhexidine: Uses:

♦ Enhances wound healing; when chlorhexidine rinses are used before extractions and after scaling and root planing or periodontal surgery, 40 hours after surgery and not more than 10 days for each course.

♦ As a pre-rinse, it reduces the salivary bacterial load by approximately 90%. It minimizes the aerosol contamination associated with various dental procedures.

- ◆ After periodontal surgery.
- In handicapped patients.
- Implant dentistry
- Management of dental caries
- Patients with mandibular fixation
- Medically compromised patients
- Recurrent aphthous ulcers
- Orthodontic patients
- Control of bacteraemia and operatory contamination by oral bacteria
- Use of chlorhexidine gel could reduce the incidence

of alveolar osteitis due to its broad spectrum of activity against oral bacteria.

• Daily oral care with chlorhexidine can be used to control oral colonization of organisms causing ventilatorassociated pneumonia in critically ill patients.

- Chemical plaque biofilm control with oral rinses

Chlorhexidine: SIDE EFFECTS:



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- Bitter taste
- Brown staining of teeth, tongue, silicate and resin restorations.

Extrinsic tooth staining is a side-effect in 37% of the individuals who use chlorhexidine. Rinsing with chlorhexidine mouthrinse for 4 weeks or longer causes extrinsic tooth staining. This could be due to the result of a local precipitation reaction between tooth-bound chlorhexidine and chromogens found in food and drinks. It may be visible as early as one week after therapy. It is more pronounced in patients with heavy accumulation of plaque.

Hence, oxygenating agents are used along with chlorhexidine to reduce staining..

• Increased supragingival calculus formation. It may be due to precipitation of salivary proteins on the tooth surface, pellicle thickness and increased precipitation of inorganic salts on or in the pellicle layer.

- ♦ Taste alteration/ change of taste sensation.
- Oral itching and soreness
- Burning sensation
- Hypersensitivity
- Mucosal desquamation / mucosal lesions
- Anesthetized sensation
- Occasional parotid gland swelling

♦ As it is cationic in nature, it can be incompatible with anionic compounds like dentifrices that may neutralize its action.



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- Chemical plaque biofilm control with oral rinses

Nonprescription Essential Oil Rinse

- Essential oil mouthrinses contain thymol, eucalyptol, menthol, and methyl salicylate.
- □ These preparations have been evaluated in longterm clinical studies and have demonstrated plaque biofilm reductions of 20% to 35% and gingivitis reductions of 25% to 35%.
- □ This type of oral rinse has had a long history of daily use and safety since the 19th century, and many patients have used these products for decades. These products also contain alcohol (up to 24% depending on the preparation), which must be a consideration for some patients not to use these products.



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- Chemical plaque biofilm control with oral rinses

Other Products

- ❑ A preparation containing triclosan has shown some effectiveness in reducing plaque biofilm and gingivitis. It is available in toothpaste form, and the active ingredient is more effective in combination with zinc citrate or a copolymer of methoxyethylene. These preparations have been evaluated in long term clinical studies and have demonstrated plaque biofilm reductions of 20% to 35% and gingivitis reductions of 25% to 35%.
- □ This type of oral rinse has had a long history of daily use and safety since the 19th century, and many patients have used these products for decades.
- □ These products also contain alcohol (up to 24% depending on the preparation), which must be a consideration for some patients not to use these products.
- Other oral rinse products on the market have shown some evidence of plaque biofilm reduction, although long-term improvement in gingival health has not been substantiated. These include stannous fluoride, cetylpyridinium chloride (quaternary ammonium compounds), and sanguinarine.
Plaque biofilm control for the periodontal patient



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- Chemical plaque biofilm control with oral rinses
- The use of chemical agents helps mechanical plaque biofilm control procedures and reduces gingivitis and caries.
- Chlorhexidine rinses can be used to improve plaque biofilm control during phase I therapy, for patients with recurrent disease, after periodontal or oral surgery, and for caries management.
- Essential oil rinses are effective as irrigants, have fewer side effects, and are available without a prescription.
- The use of cosmetic oral rinses and prebrushing rinses should not replace proven mechanical and chemical means of plaque biofilm removal, but these rinses can be useful if patients perceive benefits from them.

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Plaque biofilm control for the periodontal patient *Disclosing Agents*

- Disclosing agents are solutions or wafers that stain bacterial biofilm on the surfaces of the teeth, tongue, and gingiva.
- Wafers are crushed and rinsed in the mouth for a few seconds before they are expectorated. They can be used as educational and motivational tools to improve the efficiency of plaque biofilm control procedures
- □ Solutions are applied to the teeth as concentrates on cotton swabs or diluted as rinses. They usually produce staining of plaque biofilm, gingiva, tongue, lips, and fingers. Either form, rinses or wafers, can be used for plaque biofilm control instruction in the office and dispensed for home use to help periodontal patients evaluate the effectiveness of their oral hygiene routine.

BEFORE PLAQUE DISCLOSING TABLETS



AFTER PLAQUE DISCLOSING TABLETS



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Plaque biofilm control for the periodontal patient *Patient Motivation and Education*

- In periodontal therapy, plaque biofilm control has two important purposes: to minimize gingival inflammation and to prevent the recurrence or progression of periodontal diseases and caries.
- Daily mechanical removal of plaque biofilm by the patient, including the use of appropriate antimicrobial agents, is the only practical means for improving oral health on a long-term basis. The process requires interest and compliance on the part of the patient and education and instruction from the dentist, followed by encouragement and reinforcement. Keeping records of patients' performance facilitates this process. provides an example of a plaque biofilm control record that permits comparison over time.



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Motivation for Effective Plaque Biofilm Control

- Motivating patients to perform effective plaque biofilm control is one of the most critical and difficult elements of long-term success in periodontal therapy. It requires commitment by the patient to change poor daily habits and return for regular visits for maintenance therapy.
- □ To be successful requires the following:
- (1)The patient must understand the etiology of the disease and its importance in treatment and maintenance; the patient must also be compliant and receptive and must understand the concepts of pathogenesis, treatment, and prevention of periodontal disease;

(2) the patient must be willing to alter harmful habits such as smoking;

(3) the patient must be able to adjust personal beliefs and values to eliminate risk factors; motivational interviewing and instruction techniques can assist in achieving these goals(4) manual skills must be developed to establish an effective plaque biofilm control regimen.

Education and Scoring Systems

- □ Many patients prefer to believe that treatment is a passive process, so it is mandatory on the dentist to educate and reinforce to each patient his or her personal role in the long-term success of therapy.
- Patients must also be informed of the importance of maintenance therapy and periodic assessment in the dental office to prevent recurrence of periodontal diseases and identify problems that may arise.
 The plaque biofilm control record and the bleeding points index are simple indices that are useful for patient education and motivation.



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Plaque Biofilm Control Record (O'Leary Index)

- Plaque biofilm is recorded on the appropriate box in a diagram for four surfaces on each tooth.After all teeth have been scored, the index number is calculated for the percentage of surfaces with biofilm by dividing the number of surfaces with microbial plaque biofilm by the total number of surfaces scored and then multiplying by 100
- □ reasonable goal is 10% or fewer surfaces with plaque biofilm. If biofilm is always present in the same areas, provide instructions to improve cleaning procedures in those areas.

Bleeding Points Index

- □ The bleeding points index provides an evaluation of gingival inflammation around each tooth.
- □ The percentage of the number of bleeding surfaces is calculated by dividing the number of surfaces that bled by the total number of tooth surfaces (four per tooth) and multiplying by 100 to obtain a percentage.
- □ This index is designed to demonstrate gingival inflammation characterized by bleeding rather than the presence of microbial plaque biofilm.
- □ a goal of 10% or fewer bleeding points is good, but 0 is ideal. If a few bleeding points repeatedly occur in the same areas, cleaning procedures for those areas should be reinforced or modified because usually a residual piece of subgingival calculus that was not noted during therapy is the cause of the localized gingival bleeding.



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Instruction and Demonstration

- □ Patients can reduce the incidence of plaque biofilm and gingivitis with repeated instruction and encouragement much more effectively than with self-acquired oral hygiene habits..
- □ Strategies that will assist in educating and motivating patients include:
 - Provide encouragement and positive reinforcement. Threats are not effective.
 - Demonstrate how devices work, and allow practice time.

• Provide samples so that patients do not have to stop and buy products on the way home because they will not do it.

• Show and reinforce improvements at subsequent appointments, even if changes are modest.





Thanks

