WINSPERT

PERIODONTICS

H.O.T

HIGH-PRIORITY ORGANISED THEORY

NOTES

By Dr. Jigyasa Sharma

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Thank you for your understanding and continued dedication.

Best regards,
WINSPERT TEAM

PERIODONTICS

H.O.T TOPICS

1. Periodontitis staging grading
2. Gingivitis- drug induced, anug
3. Periodontal screening (BPE)
4. basic periodontal exam/ scoring / management
5. Periodontal problems - pregnancy
6. Perio - diabetes and obesity
7. Food impaction/ operative faults/ localised perio problems
8. Gingival defects/ localised recessions/ stillmans cleft/ mccalls festooning
9. Perio grafts, flaps, surgical perio.
10. Periodontal diagnosis
11. Periodontitis and smoking and smoking cessation

STAGING AND GRADING OF PERIODONTITIS

CLINICAL DEFINITION OF PERIODONTITIS:

Periodontitis is characterized by microbially-associated, host-mediated inflammation that results in loss of periodontal attachment. This is detected as clinical attachment loss (CAL) by circumferential assessment of the erupted dentition with a standardized periodontal probe with reference to the cemento-enamel junction (CEJ).

It should be noted that periodontal inflammation, generally measured as bleeding on probing (BOP), is an important clinical parameter relative to assessment of periodontitis treatment outcomes and residual disease risk post-treatment. However, BOP itself, or as a secondary parameter with CAL, does not change the initial case definition as defined by CAL.

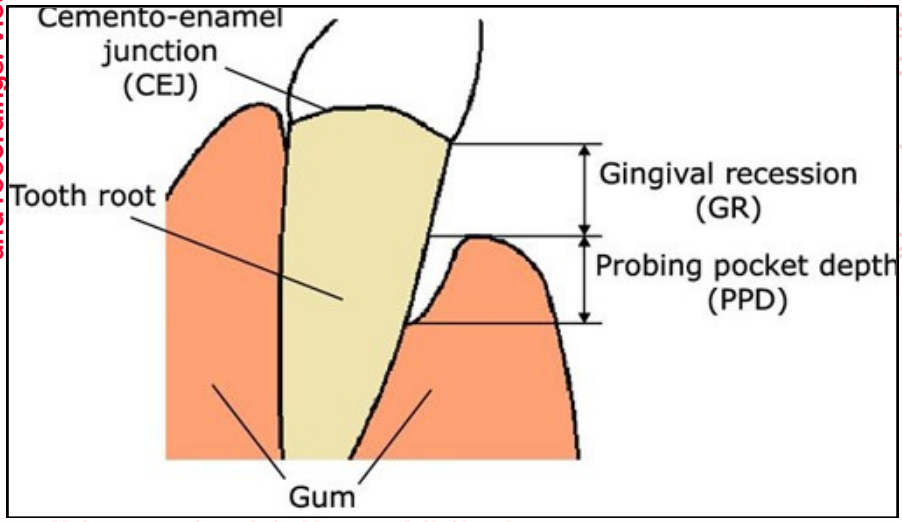
A patient is a periodontitis case in the context of clinical care if:

a) Interdental CAL is detectable at ⩾2 non-adjacent teeth
b) Buccal or oral CAL ⩾3 mm with pocketing >3 mm is detectable at ⩾2 teeth

WHAT IS CLINICAL ATTACHMENT LOSS?

Attachment loss = the distance from the cemento-enamel junction to the bottom of pocket.
Probing depth (PD) = the distance from the gingival margin to the bottom of the pocket.
Gingival recession = distance from the cemento-enamel junction to the gingival margin.
Therefore;

Attachment loss = Probing depth (PD) + Gingival recession



STAGING AND GRADING OF PERIODONTITIS

CAL cannot be ascribed to non-periodontal causes such as:

- gingival recession of traumatic origin
- dental caries extending in the cervical area of the tooth
- the presence of CAL on the distal aspect of a second molar and associated with malposition or extraction of a third molar
- an endodontic lesion draining through the marginal periodontium
- the occurrence of a vertical root fracture

Identification of the form of periodontitis:

Based on pathophysiology, three clearly different forms of periodontitis have been identified:

- Necrotizing periodontitis
- Periodontitis as a direct manifestation of systemic diseases
- Periodontitis

Necrotizing periodontitis is characterized by history of pain, presence of ulceration of the gingival margin and/or fibrin de- posits at sites with characteristically decapitated gingival papillae, and, in some cases, exposure of the marginal alveolar bone.

STAGING OF PERIODONTITIS

Staging relies on the standard dimensions of severity and extent of periodontitis at presentation but introduces the dimension of complexity of managing the individual patient.

Stage I to IV of periodontitis is defined based on severity and complexity of management, and additionally, described as extent (localized or generalized).

Severity

- primarily periodontal breakdown
- periodontitis-associated tooth loss

Complexity

- pocket depth
- infrabony defects
- furcation involvement
- tooth hyper-mobility
- masticatory dysfunction

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STAGING AND GRADING OF PERIODONTITIS

STAGING OF PERIODONTITIS

Periodontitis stage		Stage I	Stage II	Stage III	Stage IV
Severity	Interdental CAL at site of greatest loss	1 to 2 mm	3 to 4 mm	≥5 mm	≥5 mm
	Radiographic bone loss	Coronal third (<15%)	Coronal third (15% to 33%)	Extending to middle or apical third of the root	Extending to middle or apical third of the root
	Tooth loss	No tooth loss due to periodontitis		Tooth loss due to periodontitis of ≤4 teeth	Tooth loss due to periodontitis of ≥5 teeth
Complexity	Local	Maximum probing depth ≤4 mm Mostly horizontal bone loss	Maximum probing depth ≤5 mm Mostly horizontal bone loss	In addition to stage II complexity: Probing depth ≥6 mm Vertical bone loss ≥3 mm Furcation involvement Class II or III Moderate ridge defect	In addition to stage III complexity: Need for complex rehabilitation due to: Masticatory dysfunction Secondary occlusal trauma (tooth mobility degree ≥2) Severe ridge defect Bite collapse, drifting, flaring Less than 20 remaining teeth (10 opposing pairs)
Extent and distribution	Add to stage as descriptor	For each stage, describe extent as localized (<30% of teeth involved), generalized, or molar/incisor pattern			

- The initial stage should be determined using CAL; if not available then RBL should be used. CAL = clinical attachment loss; RBL = radiographic bone loss.
- Information on tooth loss that can be attributed primarily to periodontitis – if available – may modify stage definition. This is the case even in the absence of complexity factors.
- Complexity factors may shift the stage to a higher level, for example furcation II or III would shift to either stage III or IV irrespective of CAL.
- The distinction between stage III and stage IV is primarily based on complexity factors. For example, a high level of tooth mobility and/or posterior bite collapse would indicate a stage IV diagnosis.
- For any given case only some, not all, complexity factors may be present, however, in general it only takes one complexity factor to shift the diagnosis to a higher stage.
- It should be emphasized that these case definitions are guidelines that should be applied using sound clinical judgment to arrive at the most appropriate clinical diagnosis.
- For post-treatment patients CAL and RBL are still the primary stage determinants. If a stage-shifting complexity factor(s) is eliminated by treatment, the stage should not retrogress to a lower stage since the original stage complexity factor should always be considered in maintenance phase management.

STAGING AND GRADING OF PERIODONTITIS

GRADING OF PERIODONTITIS

Grade of periodontitis is estimated with direct or indirect evidence of progression rate in three categories: slow, moderate and rapid progression (Grade A-C). Risk factor analysis is used as grade modifier.

This relies on three sets of parameters:

- **rate of periodontitis progression**
- **recognized risk factors for periodontitis progression**
- **risk of an individual's case affecting the systemic health of the subject**

Periodontitis grade			Grade A: Slow rate of progression	Grade B: Moderate rate of progression	Grade C: Rapid rate of progression
Primary criteria	Direct evidence of progression	Longitudinal data (radiographic bone loss or CAL)	Evidence of no loss over 5 years	<2 mm over 5 years	≥2 mm over 5 years
	Indirect evidence of progression	% bone loss/age	<0.25	0.25 to 1.0	>1.0
		Case phenotype	Heavy biofilm deposits with low levels of destruction	Destruction commensurate with biofilm deposits	Destruction exceeds expectation given biofilm deposits; specific clinical patterns suggestive of periods of rapid progression and/or early onset disease (e.g., molar/incisor pattern; lack of expected response to standard bacterial control therapies)
Grade modifiers	Risk factors	Smoking	Non-smoker	Smoker <10 cigarettes/day	Smoker ≥10 cigarettes/day
		Diabetes	Normoglycemic / no diagnosis of diabetes	HbA1c <7.0% in patients with diabetes	HbA1c ≥7.0% in patients with diabetes
Risk of systemic impact of periodontitis[a]	Inflammatory burden	High sensitivity CRP (hsCRP)	<1 mg/L	1 to 3 mg/L	>3 mg/L
Biomarkers	Indicators of CAL/bone loss	Saliva, gingival crevicular fluid, serum	?	?	?

Grade should be used as an indicator of the rate of periodontitis progression.

The primary criteria are either direct or indirect evidence of progression. Whenever available, direct evidence is used; in its absence indirect estimation is made using bone loss as a function of age at the most affected tooth or case presentation (radiographic bone loss expressed as percentage of root length divided by the age of the subject, RBL/age).

Clinicians should initially assume grade B disease and seek specific evidence to shift towards grade A or C, if available. Once grade is established based on evidence of progression, it can be modified based on the presence of risk factors.

REFERENCE:
Staging and grading of periodontitis: Framework and proposal of a new classification and case definition- Maurizio S. Tonetti1 | Henry Greenwell2 | Kenneth S. Kornman

DRUG INDUCED GINGIVAL ENLARGEMENT & NUG

DRUGINDUCED GINGIVAL ENLARGEMENT

Gingival overgrowth or enlargement is a common side effect and unwanted outcome of certain systemic medication. Drug-influenced gingival enlargement refers to an abnormal growth of the gingiva secondary to use of systemic medication and is classified by the America Academy of Periodontology as a form of dental plaque–induced gingival disease modified by medication.

Currently three pharmaceutical categories of medication;

- Anticonvulsants
- Immunosuppressants
- calcium channel blockers

are associated with gingival enlargement.

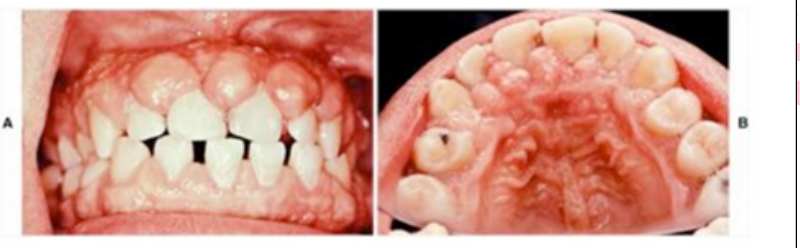
However, a strong association has been noted only with phenytoin (when used in a chronic regimen to control epileptic seizures), cyclosporine A (powerful immunoregulator drug primarily used in the prevention of organ transplant rejection), and nifedipine (commonly prescribed as antihypertensive, antiarrhythmic, and antianginal agent).

> Gingival enlargement is a well-known consequence of the administration of some anticonvulsants, immunosuppressants, and calcium channel blockers and may create speech, mastication, tooth eruption, and aesthetic problems
>
> CARRANZA

Clinical Features:

- The growth starts as a painless, beadlike enlargement of the interdental papilla and extends to the facial and lingual gingival margins (Figure 23-6)

Figure 23-6

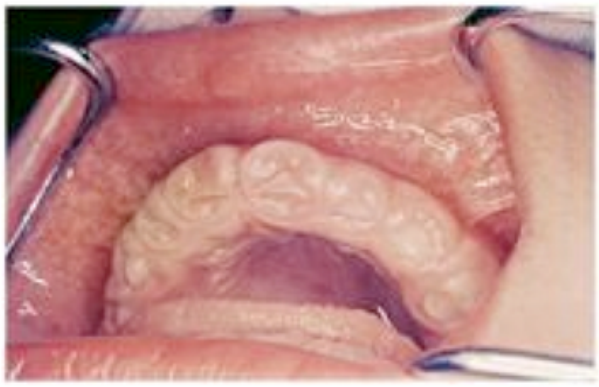


Gingival enlargement associated with phenytoin therapy. **A,** Facial view; note the prominent papillary lesions and the firm, nodular surface. **B,** Occlusal view of upper jaw.

DRUG INDUCED GINGIVAL ENLARGEMENT & NUG

- As the condition progresses, the marginal and papillary enlargements unite; they may develop into a massive tissue fold covering a considerable portion of the crowns, and they may interfere with occlusion (Figure 23-7).

Figure 23-7

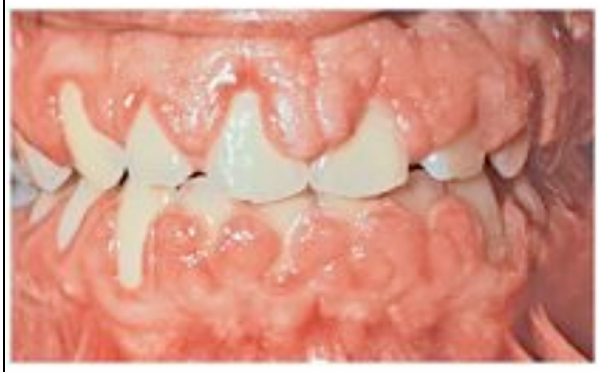


Gingival enlargement in a 5-year-old child covering most of the clinical crowns of teeth.

- When uncomplicated by inflammation, the lesion is mulberry shaped, firm, pale pink, and resilient, with a minutely lobulated surface and no tendency to bleed.
- However, the presence of the enlargement makes plaque control difficult, often resulting in a secondary inflammatory process that complicates the gingival overgrowth caused by the drug.
- The resultant enlargement then becomes a combination of the increase in size caused by the drug and the complicating inflammation caused by bacteria. Secondary inflammatory changes not only add to the size of the lesion caused by the drug, but also produce a red or bluish red discoloration, obliterate the lobulated surface demarcations, and increase bleeding tendency.
- The enlargement is usually generalized throughout the mouth but is more severe in the maxillary and mandibular anterior regions. It occurs in areas in which teeth are present, not in edentulous spaces, and the enlargement disappears in areas from which teeth are extracted. Hyperplasia of the mucosa in edentulous mouths has been reported but is rare.
- Drug-induced enlargement may occur in mouths with little or no plaque and may be absent in mouths with abundant deposits. Some investigators, however, believe that inflammation is a prerequisite for development of the enlargement, which therefore could be prevented by plaque removal and fastidious oral hygiene. Oral hygiene by means of toothbrushing or use of a chlorhexidine toothpaste reduces the inflammation but does not lessen or prevent the overgrowth.

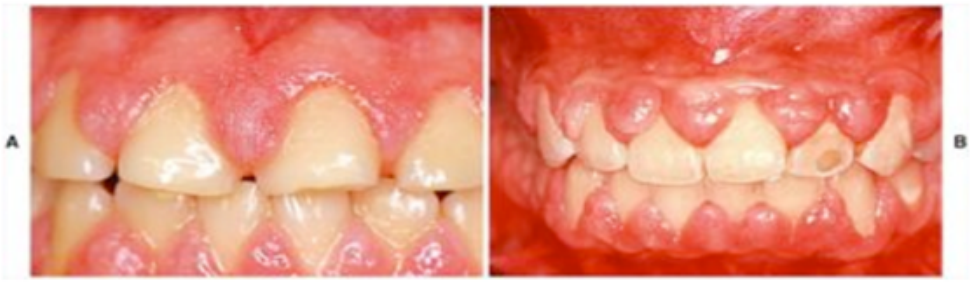
DRUG INDUCED GINGIVAL ENLARGEMENT & NUG

Figure 23-8



Combine gingival enlargement resulting from the inflammatory involvement of a phenytoin-induced overgrowth.

Figure 23-11



Cyclosporine-associated gingival enlargement. **A,** Mild involvement located particularly on papillae between teeth #9 and #10 and #10 and #11. **B,** Advanced generalized enlargement.

- Higher prevalence in children (due to phenytoin most often used in young patients and having the highest prevalence of all medication-induced gingival enlargement), onset within 1–3 months of drug use.
- Pocket depths >3–4mm may also be caused by the swelling of the gingiva without a concomitant apical migration of dentogingival epithelium from the cementoenamel junction (CEJ), as the case of gingival enlargement This increase in pocket depth is called a “pseudo-pocket” because it is not associated with bone loss or apical migration of the junctional epithelium.

DRUG INDUCED GINGIVAL ENLARGEMENT & NUG

The treatment of patients with drug-induced gingival enlargement consists of

- oral hygiene instructions
- supra- and subgingival scaling and polishing
- referral to the primary care physician for possible substitution of one medication for another (i e , phenytoin can be replaced with carbamazepine or valproic acid, cyclosporine with tacrolimus, and nifedipine with one of many dihydropteridines) not as strongly associated with gingival overgrowth
- If the previously described treatment does not result in significant resolution of gingival - enlargement, surgical excision of the excessive gingiva is performed using a classic external bevel gingivectomy or an internal bevel gingivectomy approach. The *internal bevel approach* provides primary closure and reduction of postoperative bleeding, discomfort, and infection.
- More recently, a carbon dioxide laser has been used for surgical excision and provides rapid haemostasis and compatibility with a host with underlying medical conditions. It also has been reported to reduce surgical time. To prevent postsurgical recurrence, a chlorhexidine rinse twice daily is recommended.
- Several medications have been shown to ameliorate gingival enlargement such as systemic or topical folic acid or a short course of metronidazole or azithromycin the latter drugs work particularly well for significant resolution of cyclosporine-induced gingival overgrowth.

NECROTIZING ULCERATIVE GINGIVITIS

Historical descriptions of the disease refer to it by many names, including “Vincent’s infection,” “trench mouth,” and the descriptive term “acute necrotizing ulcerative gingivitis” (ANUG). However, “acute” has been dropped from the latter nomenclature and description because it is understood that the disease has a sudden onset and that there is no chronic form.

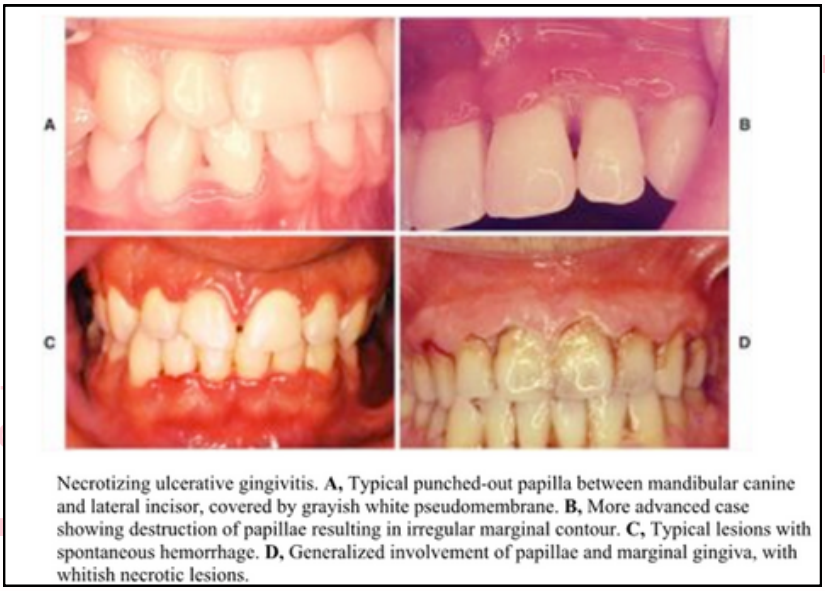
Necrotizing ulcerative gingivitis (NUG) is a microbial disease (The fusiform-spirochete bacterial flora found in NUG lesions) of the gingiva in the context of an impaired host response. It is characterized by the death and sloughing of gingival tissue and presents with characteristic signs and symptoms.

Clinical Features

- Areas of ulceration and necrosis of the interdental papilla, subsequently extending to the marginal gingiva and rarely to the attached gingiva and oral mucosa.
- covered by a whitish yellow soft layer, or pseudo-membrane, characterize gingival lesions of NUG.
- The ulcerated margin is surrounded by an erythematous halo.
- Lesions are typically painful and bleed easily, often without provocation.
- The clinical presentation of a patient with ulcerated, “punched out” papilla, pain, and bleeding are pathognomonic for NUG.
- Patients may also present with oral malodour, increased salivation, localized lymphadenopathy, fever, and malaise.

DRUG INDUCED GINGIVAL ENLARGEMENT & NUG

- The lesions are extremely sensitive to touch, and the patient often complains of a constant radiating, gnawing pain that is intensified by eating spicy or hot foods and chewing.
- Necrotising gingivitis most commonly occurs in young adult smokers and rarely occurs in children. Children thought to have necrotising gingivitis should be assessed for acute herpetic gingivostomatitis.



Necrotizing ulcerative gingivitis. **A,** Typical punched-out papilla between mandibular canine and lateral incisor, covered by grayish white pseudomembrane. **B,** More advanced case showing destruction of papillae resulting in irregular marginal contour. **C,** Typical lesions with spontaneous hemorrhage. **D,** Generalized involvement of papillae and marginal gingiva, with whitish necrotic lesions.

Management of necrotising gingivitis

Thorough debridement of plaque and necrotic debris is necessary for successful management of necrotising gingivitis. However, it may not be possible to complete debridement at the initial presentation because necrotising gingivitis can be associated with significant pain.

Begin management of necrotising gingivitis with:

- gentle removal of as much plaque and necrotic debris as possible, using local anaesthetics if necessary
- local irrigation with chlorhexidine 0.2% mouthwash or hydrogen peroxide 3% solution.
- Chlorhexidine mouthwash or hydrogen peroxide solution may also be used if pain limits the patient's ability to mechanically clean their teeth reduce plaque formation.

a) hydrogen peroxide 3% solution 5 mL, mixed with 5 mL of warm water, rinsed in the mouth for 1 minute then spat out, 12-hourly until pain has reduced	or	b) hydrogen peroxide 1.5% solution 10 mL, rinsed in the mouth for 1 minute then spat out, 12-hourly until pain has reduced
c) chlorhexidine 0.2% mouthwash 10 mL rinsed in the mouth for 1 minute then spat out, 8- to 12-hourly until pain has reduced*	or	d) chlorhexidine 0.12% mouthwash 15 mL rinsed in the mouth for 1 minute then spat out, 8- to 12-hourly until pain has reduced.

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DRUG INDUCED GINGIVAL ENLARGEMENT & NUG

- antibiotic therapy: metronidazole 400 mg orally, 12-hourly for 3 to 5 days.
- analgesics
- advice to stop smoking

Review the patient in 48 to 72 hours; perform a periodontal examination and provide the patient with advice on oral hygiene. Perform thorough debridement as soon as possible to prevent recurrence.

A poor response to treatment or recurrence of symptoms is usually due to Inadequate debridement or a lack of improvement in oral hygiene, rather than an ineffective antibiotic regimen. If the infection has not responded to appropriate management (complete debridement, antibiotic therapy, improved oral hygiene) within 2 weeks, refer for specialist management.

Profoundly immunocompromised patients or patients with severe cases of necrotising gingivitis require prompt referral for specialist management in addition to the management above.

Treatment failure is usually due to inadequate debridement or poor oral hygiene, rather than ineffective antibiotic therapy.

REFERENCE:
Carranza | Clinical cases in periodontics | Good practitioners guide to periodontology | Periodontal diseases -Bruce L Pihlstrom, Bryan S Michalowicz, Newell W Johnson | Therapeutic guidelines

BASIC PERIODONTAL EXAMINATIO (BPE), PERIODONTAL SRCEENING AND MANAGEMENT

The BPE is a simple and rapid screening tool that is used to indicate the level of further examination needed and provide basic guidance on treatment needed. The BPE guidelines are not prescriptive but represent a minimum standard of care for initial periodontal assessment. BPE should be used for screening only and should not be used for diagnosis.

Screening involves probing of the periodontal tissues to assess the presence of bleeding on probing, plaque and calculus deposits and the depth of any periodontal pockets which may be present.

How to record the BPE for adults:

1. **The dentition is divided into 6 sextants**
   - Upper right (17 to 14), upper anterior (13 to 23), upper left (24 to 27)
   - Lower right (47 to 44), lower anterior (43 to 33), lower left (34 to 37)
2. **All teeth in each sextant are examined**
   (except for 3rd molars unless 1st and/or 2nd molars are missing).
   - For a sextant to qualify for recording, it must contain at least 2 teeth.
3. **The probe should be "walked around" the sulcus/pockets in each sextant, and the highest score recorded for each sextant**
   - The WHO probe (often called a BPE probe) has a ball end 0.5mm in diameter, and a black band from 3.5 to 5.5mm. A light probing force of between 20-25 grams should be used.

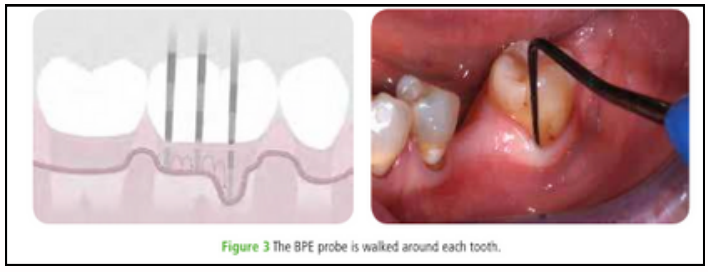

Figure 3 The BPE probe is walked around each tooth.

All sites should be examined to ensure that the highest score in the sextant is recorded before moving on to the next sextant. If a code 4 is identified in a sextant, continue to examine all sites in the sextant. This will help to gain a fuller understanding of the periodontal condition and will make sure that furcation involvements are not missed.

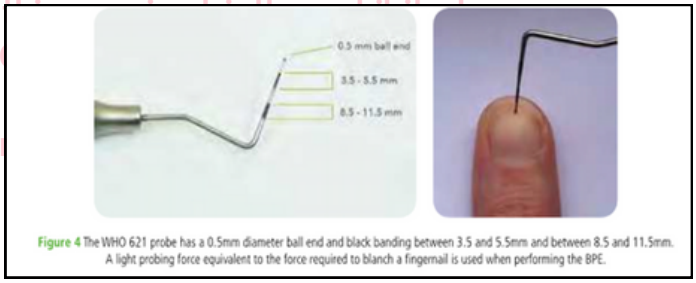

Figure 4 The WHO 621 probe has a 0.5mm diameter ball end and black banding between 3.5 and 5.5mm and between 8.5 and 11.5mm. A light probing force equivalent to the force required to blanch a fingernail is used when performing the BPE.

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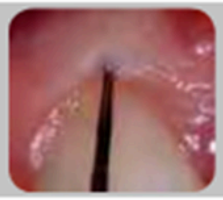
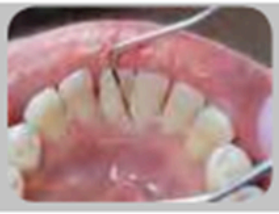
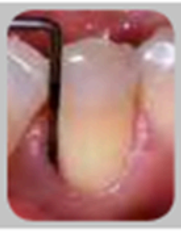
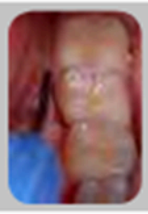
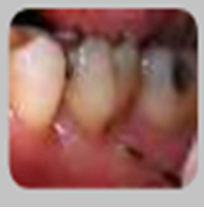
BASIC PERIODONTAL EXAMINATIO (BPE), PERIODONTAL SRCEENING AND MANAGEMENT

An example BPE score:

Both the number and the * should be recorded if a furcation is detected e.g. the score for a sextant could be 3* (e.g. indicating probing depth 3.5-5.5mm PLUS furcation involvement in the sextant).

BPE code	Probing depth	Observation	
0	Pockets < 3.5mm First black band completely visible	Healthy periodontal tissues No calculus/overhangs No bleeding on probing	
1	Pockets < 3.5mm First black band completely visible	Bleeding on probing No calculus/overhangs	 (Note the recession in this image is not accounted for in the BPE)
2	Pockets < 3.5mm First black band completely visible	Supra or subgingival calculus or plaque retention factor (overhang)	
3	Probing depth 3.5 - 5.5mm	First black band partially visible, indicating pocket of 4-5mm	
4	Probing depth > 5.5mm	First black band entirely within the pocket, indicating pocket of 6mm or more	
*	Furcation involvement	Detection of a furcation	

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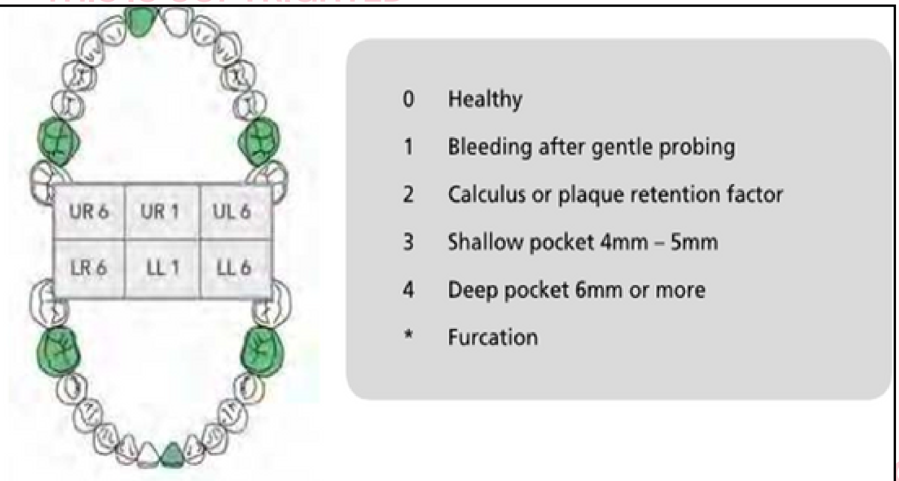
BASIC PERIODONTAL EXAMINATIO (BPE), PERIODONTAL SRCEENING AND MANAGEMENT

Recording the BPE for children:

Periodontal screening for children and adolescents assesses six index teeth (UR6, UR1, UL6, LL6, LL1 and LR6) using a simplified BPE to avoid the problem of false pockets. The ideal probe for this examination is a WHO 621 style probe, the second black at 8.5 – 11.5mm being useful if there is false pocketing.

BPE codes 0 - 2 are used in 7 to 11year-olds (during the mixed dentition phase) while the full range of codes 0, 1, 2, 3, 4 and * can be used in 12 to 17year-olds (when the permanent teeth erupt).



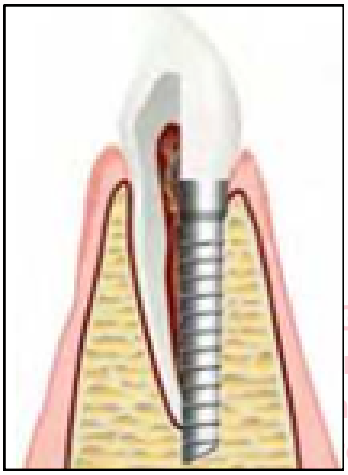
5 The simplified BPE for under 18 year olds. Examination of 6 index using BPE codes 0-2 for 7-11 year olds and the full set of codes for 12-17 year olds.

Examples of BPE scores

8-year old			15-year old		
1	1	2	2	2	3
1	2	2	2	2	2

BASIC PERIODONTAL EXAMINATIO (BPE), PERIODONTAL SRCEENING AND MANAGEMENT

Implants and the BPE



Like teeth, implants are susceptible to bacterial plaque leading to an inflammatory response in the peri-implant tissues. However, the tissues surrounding implants are not connected to the implant surface in the same way as those surrounding teeth and are less resistant to probing. This in combination with the anatomical position of the implant in relation to the bone and soft tissues may lead to deeper probing depths in healthy sites.

For this reason, the BPE is not appropriate for the assessment of implants. Detailed probing (four or six points) and the presence of any bleeding or suppuration should be measured around each implant.

Unlike natural teeth implants do not have a periodontal ligament connecting them to the underlying bone.

When to record the BPE

- All new patients should have a BPE recorded (both children and adults)
- For patients with codes 0, 1 or 2 on a previous BPE recording, the BPE should be recorded at every routine examination
- For patients with BPE codes of 3 or 4, more detailed periodontal charting is required.
  - Code 3: initial therapy including -care advice (oral hygiene instruction and risk factor control) then post-initial therapy, record a 6-point pocket chart in that sextant only.
  - Code 4: if a code 4 is found in any sextant, then detailed probing depths (6 sites per tooth) should be recorded for the entire dentition.

BPE cannot be used to monitor the response to periodontal therapy because it does not provide information about how sites within a sextant change after treatment. To assess the response to treatment, a 6-point pocket chart should be recorded pre- and post-treatment.

Once these patients reach the maintenance phase of care, then full probing depths throughout the entire dentition should be repeated and recorded at least annually. Your hygienist may be providing care for the patient at this stage, so it is important to check that the periodontal chart is kept up to date so that any persistent sites can be re-instrumented if needed.

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BASIC PERIODONTAL EXAMINATIO (BPE), PERIODONTAL SRCEENING AND MANAGEMENT

Radiographs

Radiographs should be available for all Code 3 and Code 4 sextants. The type of radiograph used is a matter of clinical judgement but crestal bone levels should be visible. Many clinicians would regard periapical views as essential for Code 4 sextants to allow assessment of bone loss as a percentage of root length and visualisation of the periapical tissues.

Guidance on interpretation of BPE scores

Interpreting the BPE score depends on many factors that are unique to each patient. As a general rule however, radiographs to assess alveolar bone levels should be obtained for teeth or sextants where BPE codes 3 or 4 are found. Information from the radiographs must be considered along with the BPE scores, to determine the level of attachment loss. The clinician should use their skill, knowledge and judgement when interpreting BPE scores.

Detailed periodontal charting

When a 6-point pocket chart is indicated it is only necessary to record sites of 4mm and above (although 6 sites per tooth should be measured). Bleeding on probing should always be recorded in conjunction with a 6-point pocket chart.

Indications for referring a child to a specialist include:

- Diagnosis of aggressive periodontitis
- Incipient chronic periodontitis not responding to treatment
- Systemic medical condition associated with periodontal destruction
- Medical history that significantly affects periodontal treatment or requiring multi-disciplinary care
- Genetic conditions predisposing to periodontal destruction
- Root morphology adversely affecting prognosis
- Non-plaque induced conditions requiring complex or specialist care
- Cases requiring diagnosis/management or rare/complex clinical pathology
- Drug-induced gingival overgrowth
- Cases requiring evaluation for periodontal surgery

BASIC PERIODONTAL EXAMINATIO (BPE), PERIODONTAL SRCEENING AND MANAGEMENT

Management

PERIODONTAL SCREENING

Code	Guidance	Special investigations	Periodontal reassessment
0	No need for periodontal treatment	None indicated	Repeat BPE at next check up appointment
1	Oral hygiene instruction (OHI)	Plaque and bleeding charts	Repeat BPE at next check up appointment
2	As for code 1, plus removal of plaque retentive factors, including all supra and subgingival calculus	Plaque and bleeding charts	Repeat BPE at next check up appointment
3	As for code 2 and OHI, root surface debridement (RSD) if required	• Plaque and bleeding charts • Radiographs should be considered (in order to establish if there is attachment loss)	Periodontal charting of sextants scoring 3, after initial therapy
4	OHI, RSD. Access the need for more complex treatment; referral to a specialist may be indicated	• Plaque and bleeding charts • Radiographs should be taken	Full periodontal charting before and after treatment
*	Treat according to BPE code (0-4). Assess the need for more complex treatment; referral to a specialist may be indicated	• Plaque and bleeding charts • Radiographs should be considered	Full periodontal charting before and after treatment

Reference: The Good Practitioner's Guide to Periodontology

PERIODONTAL PROBLEMS AND PREGNANCY

Recent epidemiological studies have suggested that periodontitis is a risk factor for other systemic diseases, including:

- Pneumonia (especially aspiration pneumonia) in the elderly
- Diabetes mellitus
- Atherosclerosis
- Pregnancy complications
- Male and female infertility (studies showed a positive association between chronic periodontitis and male infertility. On the other hand, there are few reports about the relationship between female infertility and periodontal diseases)

Periodontal diseases are considered a risk factor for adverse pregnancy outcomes (APO), including:

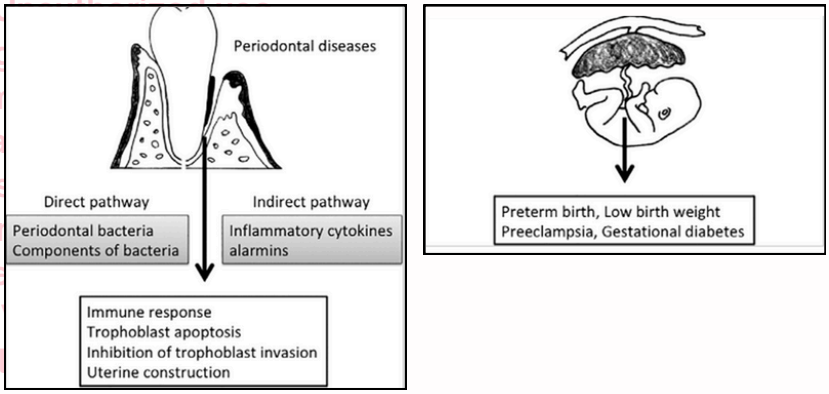
- preterm birth
- foetal growth restriction
- low birthweight
- pre-eclampsia
- gestational diabetes (periodontal disease is associated with an increasing risk of GDM among pregnant women compared to those without periodontal disease)

PATHOPHYSIOLOGY

Two pathogenic mechanisms might explain the potential effect of periodontal diseases on pregnancy outcomes.

- Periodontal bacteria originating in the gingival biofilm directly affect the feto-placental unit subsequent to bacteraemia.
- Inflammatory mediators secreted by the subgingival inflammatory site are carried to the feto-placental unit, where they then cause an inflammatory response.

Periodontal diseases are caused by multiple gram negative microaerophilic and anaerobic bacteria, such as Porphyromonas gingivalis, Fusobacterium nucleatum, Prevotella intermedia, Actinobacillus actinomycetemcomitans and Treponema denticola.



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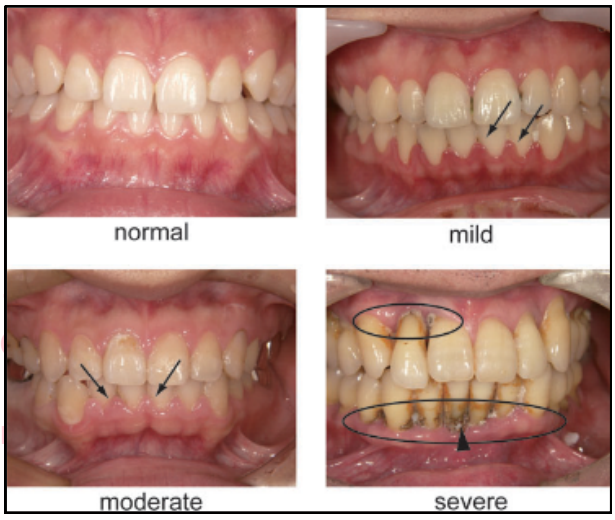
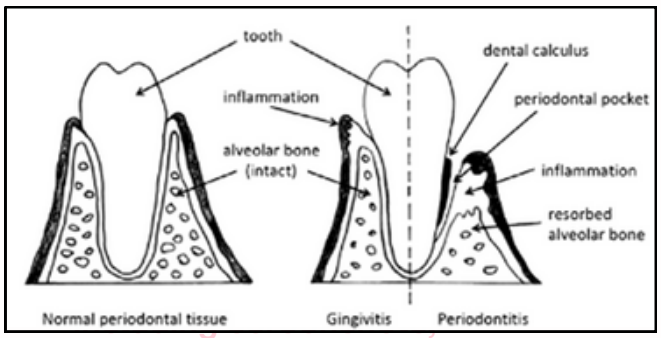
PERIODONTAL PROBLEMS AND PREGNANCY

Clinical appearance of healthy and periodontitis patients is explained in the below picture.

- **The gingiva of healthy subjects is located tightly against the teeth. (picture -normal)**
- **Swelling and redness (arrows) were shown in the gingiva of mild and moderate periodontitis patients. (picture- mild to moderate)**
- **In severe periodontitis patients, a regression of gingiva (circle) and dental calculus (arrow head) were observed. (picture-severe)**



Periodontal disease includes gingivitis and periodontitis. Gingivitis has gingival inflammation without the destruction of connective tissue and alveolar bone resorption. Periodontitis is the presence of gingival inflammation with a loss of connective tissue and alveolar bone. Dental calculus is observed in periodontal pockets.



DENTAL CARE DURING PREGNANCY

Periodontal bacteria, especially P. gingivalis, and their components can injure the trophoblast morphologically and functionally. Moreover, inflammatory mediators from periodontal pockets might elicit an inflammatory immune response at the feto-placental unit.

PERIODONTAL PROBLEMS AND PREGNANCY

However, periodontal treatment during pregnancy seems to have little effect on the prevention of APO incidence.

Trophoblast cells migrate into the uterine myometrium and reconstruct the uteroplacental sinus during the early period of pregnancy, and the placental structure is completed in the first trimester.

Therefore, although dental care is effective in curing periodontal diseases, dental care during pregnancy may occur too late to reduce pregnancy complications.

Despite knowing the importance of oral hygiene, obstetricians, dentists and pregnant women themselves have a tendency to avoid invasive dental treatment during pregnancy for possible side effects.

Therefore, oral health care and dental interventions before conception are strongly recommended.

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REFERENCE:
Periodontal diseases and adverse pregnancy outcomes- Shihoko Komine-Aizawa, Sohichi Aizawa, and Satoshi Hayakawa

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PERIO- DIABETES – OBESITY

The association between oral health and systemic health is bidirectional; systemic illnesses, especially metabolic disorders, affect oral health, and it appears that oral health may affect systemic health.

What is diabetes?

The term “diabetes mellitus” (DM) describes a group of disorders characterized by elevated levels of glucose in the blood and abnormalities of carbohydrate, fat and protein metabolism. DM is a metabolic disorder characterized by impaired action, secretion of insulin or both, resulting in hyperglycaemia presents with the classical triad of symptoms: Polydipsia, polyuria and polyphagia which are often accompanied by chronic fatigue and loss of weight thus DM is a true metabolic disorder and, thus, affects every tissue in the body.

Type 1 diabetes:

Type 1 diabetes is an autoimmune disease which results in the body destroying its own insulin-producing cells in the pancreas. The pancreas then is not able to produce insulin. People with this condition require daily insulin therapy to survive. Type 1 diabetes used to be called insulin dependent diabetes mellitus (IDDM) or juvenile-onset diabetes.

Type 2 diabetes:

Type 2 diabetes is marked by an inability of the pancreas to make enough insulin (insulin deficiency) and/or the inability of the body to use insulin properly (insulin resistance). Those at elevated risk of developing type 2 diabetes.

number of systemic diseases, especially diabetes, that can exaggerate the host response to the local microbial factors (for example, endotoxin), resulting in unusually destructive periodontal breakdown. Periodontal disease also is associated with hyperglycaemia; the poorer the control of DM is, the greater the risk of developing periodontal disease. In fact, aggressive periodontitis is recognized as the sixth complication of diabetes.

Periodontal disease–the sixth complication of diabetes

Research has confirmed that people with diabetes are more likely to have gingivitis and periodontal disease, particularly when diabetes is poorly controlled. Recent evidence suggests that periodontal disease may make it more difficult for people who have diabetes to control their diabetes.

Studies have provided evidence that control of periodontal infection has an impact on improvement of glycaemic control evidenced by a decrease in demand for insulin and decreased haemoglobin A--1c levels.

The extent to which diabetes is controlled is assessed by measuring the levels of blood glucose and glycosylated haemoglobin (HbA1c).

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PERIO- DIABETES – OBESITY

Recommended levels of blood glucose and HbA1c

Blood glucose level:

- Before meal - 4 – 6 millimoles/litre
- After meal- 4 – 7.7 Millimoles/litre

HbA1c (glycosylated haemoglobin) tested every 4 – 6 months:

- Type 1 diabetes 7.0 – 7.5%
- Type 2 diabetes <6.5%

In addition to periodontal infection and gingival inflammation, a number of other oral complications have often been reported in patients with diabetes.
These include:

- xerostomia
- dental caries
- Candida infection
- burning mouth syndrome
- lichen planus
- poor wound healing

There is also strong evidence that the presence of periodontal disease is associated with increased cardiovascular morbidity in patients with DM.

Diabetes and gingivitis

People with type 1 diabetes have a greater risk of developing gingivitis. More gingival inflammation and higher gingival bleeding scores are found in children with diabetes than children without diabetes after accounting for plaque scores. Type 2 diabetes is also associated with gingivitis that may be related to glycaemic control.

Diabetes and periodontitis

- Both gingivitis and periodontitis are more severe if the person with diabetes is a smoker. periodontal disease increasing markedly when diabetes is poorly controlled.
- The presentation of multiple and recurrent periodontal abscesses can often be pathognomonic for untreated and uncontrolled diabetes.
- Diabetes leads to more rapid and severe progression of destructive periodontitis.
- Patients with poor glycaemic control often present with severely inflamed gum tissues and evidence of loss of tooth support that is often seen as spreading of teeth result in open spaces between the teeth (diastemas).
- Despite similar plaque scores (bacterial deposits), patients with poorly controlled type 2 diabetes display more severe gingival bleeding compared to those with diabetes in good or moderate control.
- Patents with poorly controlled type 2 diabetes are at greater risk for periodontal disease progression than patients with well-controlled type 2 diabetes.

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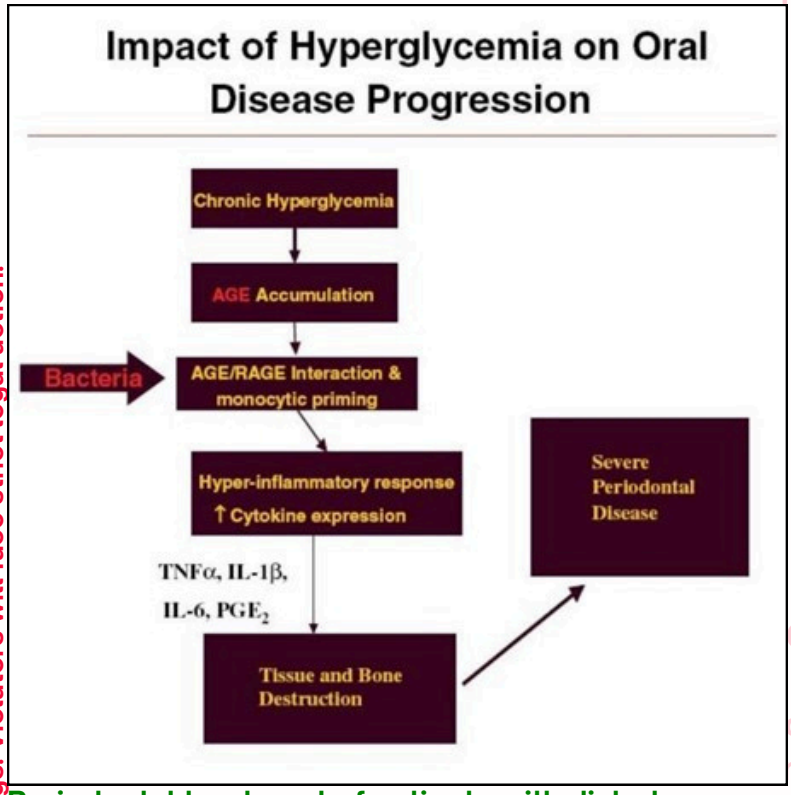
PERIO- DIABETES – OBESITY

Effect of Diabetes Mellitus on Periodontal Disease

Persisting hyperglycaemia leading to an exaggerated immuno-inflammatory response to the periodontal pathogenic bacterial challenge, resulting in more rapid and severe tissue destruction. Thus, local periodontal tissue destruction may be a consequence of an exaggerated monocytic inflammatory response induced by AGE accumulation and result in exaggerated secretion of local and systemic mediators to severe periodontitis.



Periodontal treatment of patients with diabetes

- **Treatment of periodontal disease in a patient with diabetes should be very similar to that of a patient without diabetes if the condition is well controlled. If the diabetes is poorly controlled or is difficult to control, some modifications of treatment may be necessary to allow for poorer response to therapy including wound healing. Use of systemic antibiotics in association with other treatment modalities may assist in achieving improved levels of glycosylated haemoglobin, the marker of diabetic control.**
- **The clinician should make sure that prescribed insulin or oral medication has been taken, followed by a meal.**
- **Morning appointments are appropriate because of optimal insulin levels.**
- **Refer to periodontist if periodontal condition deteriorates**

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PERIO- DIABETES – OBESITY

Complications of diabetes

Hypoglycaemia:
Hypoglycaemia can occur even in a person with well-controlled diabetes. Most patients recognise the symptoms – sweating, tremor/trembling, palpitations/pounding heart, anxiety, tiredness, pallor, headache, hunger, dizziness, irritability, blurred vision, aggressive behaviour, slurred speech, confusion, drowsiness, convulsions, coma.

Management:
Give sweets, liquid glucose or orange juice if the patient is feeling unwell. If the patient has more severe symptoms such as drowsiness, convulsions or coma, immediate medical assistance is required.

Hyperglycaemia:
Hyperglycaemia can be aggravated by infections and is more likely to occur in a person with undiagnosed diabetes. The symptoms are weakness, tiredness, frequent urination, increased thirst, blurry vision and itchy skin.

Management:
Refer the patient to a medical practitioner.

People newly diagnosed with diabetes should undergo a full oral examination including a full periodontal examination. Regular monitoring of the periodontal status of people with diabetes is advised.

The dental practitioner has an important role in the management of patients with diabetes. This role includes referral to a medical practitioner of patients with signs and symptoms indicative of diabetes, working closely with the medical practitioner in management of oral complications, specific monitoring of the oral environment, and treatment of conditions related to diabetes.

PERIODONTAL DISEASE AND OBESITY

According to the World Health Organization, obesity could be defined as “a systemic disease characterized by excessive body fat accumulation that can lead to adverse impacts on health conditions.

Obese individuals more susceptible to infectious diseases, like periodontal diseases. obese subjects tend to present with more unhealthy habits such as tobacco and alcohol consumption, physical inactivity, unhealthy diet including higher consumption of fat, carbohydrates and sugar. In addition, obese subjects are more likely to have neglected oral health habits, since low self-esteem and negative self-body image impact on the individual’s propensity to carry out health-promoting behaviours.

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PERIO- DIABETES – OBESITY

PERIODONTAL DISEASE AND OBESITY

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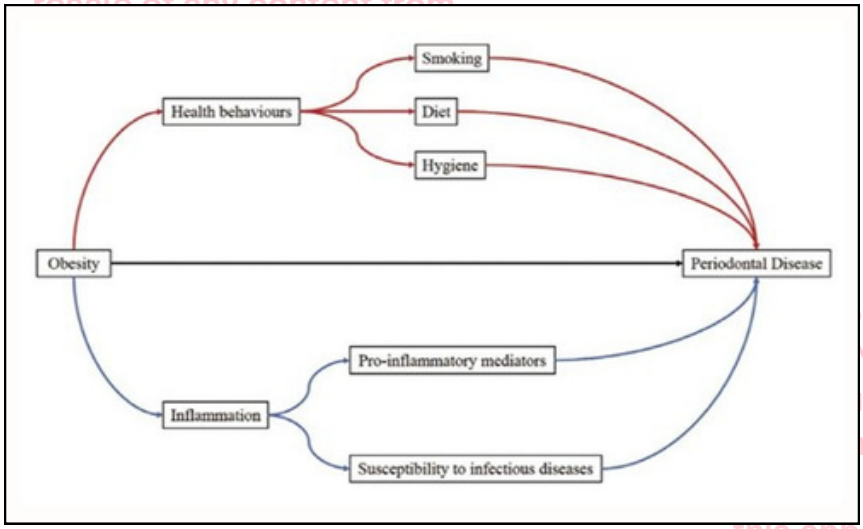
The below figure depicts the potential mechanisms underlying the relationship between obesity and periodontal disease:



According to the review conducted by Suvan and colleagues, the chance of developing periodontal disease in overweight and obese individuals, respectively, was 27% and 81% higher than in individuals with normal weight.

REFERENCE:
Diabetes and oral health | Periodontal disease and diabetes | The good practitioners guide to periodontology | Diabetes and periodontal disease- an update for health care providers | Periodontal disease and obesity | Periodontal disease- the 6th complication of diabetes-NIH article | Therapeutic guidelines

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FOOD IMPACTION/ OPERATIVE FAULTS/ LOCALISED PERIODONTAL PROBLEMS

DEVELOPMENTAL OR ACQUIRED DEFORMITIES AND CONDITIONS

In general, these factors are considered to be those local factors that contribute to the initiation and progression of periodontal disease through an enhancement of plaque accumulation or the prevention of effective plaque removal by normal oral hygiene measures. These factors fall into four subgroups.

Localized Tooth-Related Factors That Modify or Predispose to Plaque-Induced Gingival Diseases or Periodontitis

- Tooth anatomic factors
- Dental restorations or appliances
- Root fractures
- Cervical root resorption and cemental tears

Mucogingival Deformities and Conditions around Teeth

- Gingival or soft tissue recession
  a. Facial or lingual surfaces
  b. Interproximal (papillary)
- Lack of keratinized gingiva
- Decreased vestibular depth
- Aberrant frenum or muscle position
- Gingival excess
  a. Pseudo-pocket
  b. Inconsistent gingival margin
  c. Excessive gingival display
  d. Gingival enlargement
  e. Abnormal colour

Mucogingival Deformities and Conditions on Edentulous Edges

- Vertical and/or horizontal ridge deficiency
- Lack of gingiva or keratinized tissue
- Gingival or soft tissue enlargements
- Aberrant frenum or muscle position
- Decreased vestibular depth
- Abnormal colour

Occlusal Trauma

- Primary occlusal trauma
- Secondary occlusal trauma

Lets discuss about each topic in detail ...

FOOD IMPACTION/ OPERATIVE FAULTS/ LOCALISED PERIODONTAL PROBLEMS

LOCALIZED TOOTH-RELATED FACTORS THAT MODIFY OR PREDISPOSE TO PLAQUE-INDUCED GINGIVAL DISEASES OR PERIODONTITIS

a) Tooth anatomic factors

These factors are associated with malformations of tooth development or tooth location. Anatomic factors such as cervical enamel projections and enamel pearls have been associated with clinical attachment loss, especially in furcation areas.

Cervical enamel projections are found on 15% to 24% of mandibular molars and 9% to 25% of maxillary molars, and strong associations have been observed with furcation involvement. In descending order of occurrence, CEPs are most commonly seen in mandibular second molars, maxillary second molars, mandibular first molars, and maxillary first molars When CEPs are observed, they are usually seen on buccal aspects of molars.

Cervical enamel projections (CEPs) refer to the extension of enamel to the furcal area of the root surface CEPs may potentially predispose a furcation to attachment loss because they prevent connective tissue attachment at furcation As such, a periodontal pocket may form, leading to plaque accumulation and possibly furcation invasion Most clinicians agree there is a correlation between CEPs and the incidence of furcation invasion.

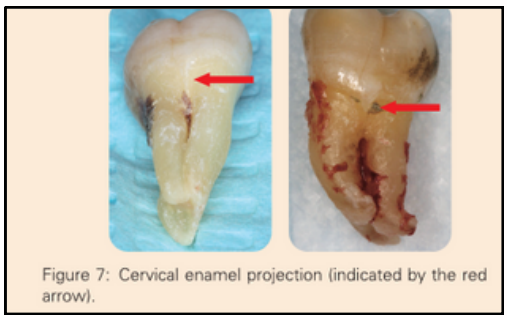

Figure 7: Cervical enamel projection (indicated by the red arrow).

Enamel pearls are ectopic globules of enamel and sometimes pulpal tissue that often adhere to the cementoenamel junction (CEJ) They are present in roughly 2.7% of the molars and are mostly found on maxillary third and second molars. Moskow and Canut suggested that enamel pearls may also predispose a furcation to attachment loss.

Figure 8: Enamel pearl (indicated by the red arrow).

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FOOD IMPACTION/ OPERATIVE FAULTS/ LOCALISED PERIODONTAL PROBLEMS

Proximal Contact Relation: open interproximal contacts or uneven marginal ridge relations may encourage food impaction between the teeth If proper oral hygiene is absent, food impaction can lead to inflammation, thereby potentially resulting in attachment loss in the interproximal area.

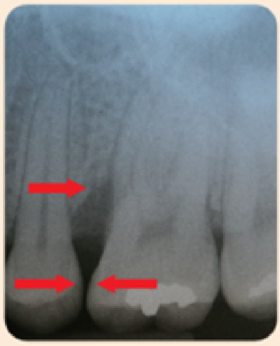

Figure 5: Interproximal open contact between #13 and #14 (indicated by the red arrows) and vertical bone loss on #14 mesial.

Root Proximity: Close root proximity between the two adjacent teeth will render oral hygiene difficult to maintain for both the patient and the dental professionals Hence without good oral hygiene there can be loss of attachment between the two teeth

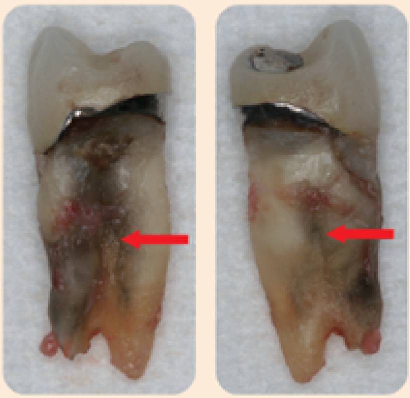

Figure 9: Mesial and distal root concavities of maxillary first premolar.

Root Divergence and Root Fusion: The degree of root divergence in a multirooted tooth will influence the ability of the patient and dental professionals to control plaque level.

Diverging roots allow easier instrumentation to the furcation area, whereas converging roots (e g , root fusion) render the access to the furcation area very difficult, resulting in poor plaque control and possible attachment loss

FOOD IMPACTION/ OPERATIVE FAULTS/ LOCALISED PERIODONTAL PROBLEMS

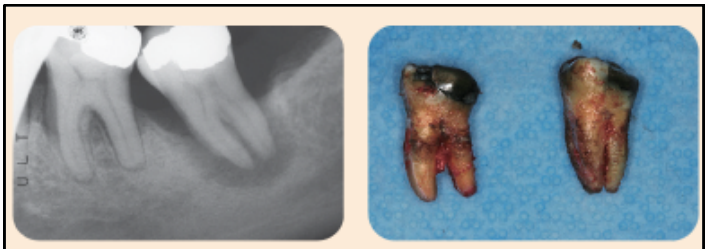

Figure 11: The root divergence of #19 is more prominent than #17.

Root Trunk Length: The length of root trunk affects attachment loss. The longer a given root trunk, the less likely a furcation will be predisposed to attachment loss. Teeth with Taurodontism usually have apically displaced furcation and longer root trunk length.

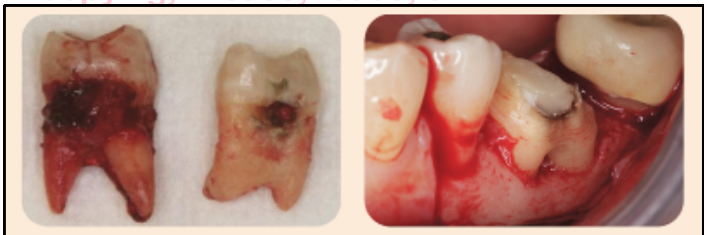

Figure 12: Long root trunk length (left) and short root trunk at #19 (right).

Intermediate Bifurcation Ridge: Intermediate bifurcation ridges are ridges spanning across the bifurcation of mandibular molars in the mesiodistal direction These ridges are present in 70–77% of the mandibular molars [8,9] Just like other anatomic structures, the presence of an intermediate bifurcation ridge may hinder effective plaque control and root preparation by both the patient and dentist.

Buccal Radicular Groove and Palato-Gingival Groove: Buccal radicular grooves and palato-gingival grooves are developmental phenomena that affect mainly the maxillary anterior teeth. These grooves run on the roots in the coronal-apical direction Due to their anatomy, the grooves frequently provide a plaque-retentive area that is very difficult to instrument, making teeth with these developmental grooves more prone to attachment loss.

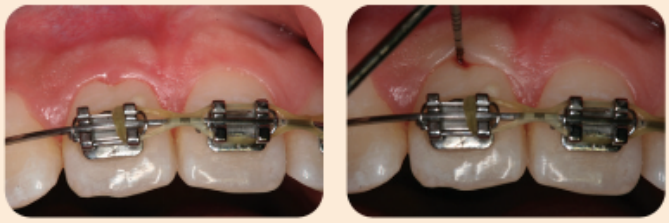

Figure 13: Buccal radicular groove present on #8 as indicated by the probe tip.

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FOOD IMPACTION/ OPERATIVE FAULTS/ LOCALISED PERIODONTAL PROBLEMS

Accessory Pulpal Canals: Accessory pulpal canals are small endodontic canals branching off from the main root canal that may furnish a communication between the pulpal chamber and the periodontal ligament. These accessory canals are usually located near the root apex; however, they can also be found anywhere along the root, including the furcation area. There is a theory that some periodontal infections can originate from endodontic sources, traveling through accessory/lateral canals located in the furcation areas In these cases there is periodontal involvement in the furcation, but the infection originated in the pulp Although still controversial, it has been proposed that periodontal disease can result from pulpal infection. An endodontic infection may be present at the furcation area when the infection travels through accessory canals that end at the furca. Vertucci and Williams reported that accessory canals at furcations are present in 46% of human lower first molar. Burch and Hulen observed accessory canals in 76% of maxillary and mandibular molars.

b) Dental restorations or appliances

Restorative Considerations: Dental restorations with overhangs or open margins are plaque-retentive areas that may result in gingival inflammation and attachment loss. Restorative margins are most compatible with the periodontium when located either supra-gingivally or at the level of gingival margin Should the restorative margin violate the biologic width, the resulting inflammatory process may lead to gingival recession, bone loss, and the exposure of the restorative margin. The restorative contour (e g, crown contour) should follow the root surface contour rather than accentuating the cervical bulge to support periodontal health. In the case of bridges, the design of the pontic can affect its ability to be cleaned and hence the periodontal health of the teeth.

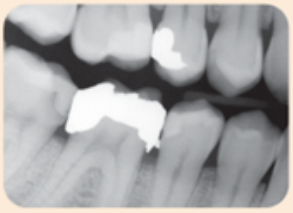


Figure 14: Overhangs on the mesial and distal of #30 that may eventually lead to bone loss on the mesial and distal of #30.

FOOD IMPACTION/ OPERATIVE FAULTS/ LOCALISED PERIODONTAL PROBLEMS

c) Root fractures

Root fractures caused by traumatic forces or restorative or endodontic procedures may lead to periodontal involvement through an apical migration of plaque along the fracture when the fracture originates coronal to the clinical attachment and is exposed to the oral environment.

d) Cervical root resorption and cemental tears

Cervical root resorption and cemental tears may lead to periodontal destruction when the lesion communicates with the oral cavity and allows bacteria to migrate subgingivally.

Mucogingival Deformities and Conditions around Teeth

Mucogingival is defined as “a generic term used to describe the mucogingival junction and its relationship to the gingiva, alveolar mucosa, frenula, muscle attachments, vestibular fornices, and the floor of the mouth.” A mucogingival deformity may be defined as “a significant departure from the normal shape of gingiva and alveolar mucosa” and may involve the underlying alveolar bone.

Mucogingival surgery is defined as “periodontal surgical procedures designed to correct defects in the morphology, position, and/or amount of gingiva”. The surgical correction of mucogingival deformities may be performed for aesthetic reasons, to enhance function, or to facilitate oral hygiene.

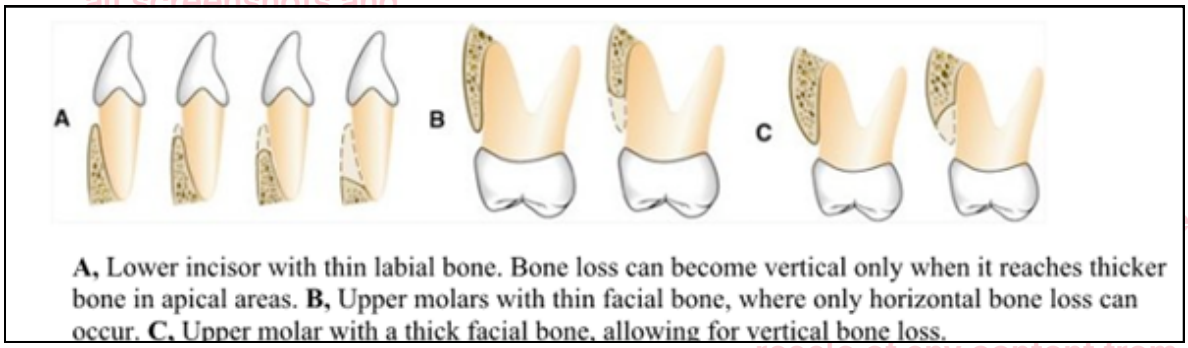
Mucogingival Deformities and Conditions on Edentulous Ridges

Mucogingival deformities and conditions on edentulous ridges usually require corrective surgery to restore form and function before the prosthetic replacement of missing teeth or implant placement.

FOOD IMPACTION/ OPERATIVE FAULTS/ LOCALISED PERIODONTAL PROBLEMS

Occlusal Trauma

Another cause of periodontal destruction is trauma from occlusion, which can produce bone destruction in the absence or presence of inflammation.
In the absence of inflammation, the changes caused by trauma from occlusion vary from increased compression and tension of the periodontal ligament and increased osteoclasis of alveolar bone to necrosis of the periodontal ligament and bone and the resorption of bone and tooth structure. These changes are reversible in that they can be repaired if the offending forces are removed. However, persistent trauma from occlusion results in funnel-shaped widening of the crestal portion of the periodontal ligament, with resorption of the adjacent bone.35 These changes, which may cause the bony crest to have an angular shape, represent adaptation of the periodontal tissues aimed at "cushioning" increased occlusal forces, but the modified bone shape may weaken tooth support and cause tooth mobility.
When combined with inflammation, trauma from occlusion aggravates the bone destruction caused by the inflammation and results in bizarre bone patterns.



A, Lower incisor with thin labial bone. Bone loss can become vertical only when it reaches thicker bone in apical areas. **B,** Upper molars with thin facial bone, where only horizontal bone loss can occur. **C,** Upper molar with a thick facial bone, allowing for vertical bone loss.

Trauma from occlusion may be a factor in determining the dimension and shape of bone deformities. It may cause a thickening of the cervical margin of alveolar bone or a change in the morphology of the bone (e.g., angular defects, buttressing bone) on which inflammatory changes will later be superimposed.

FOOD IMPACTION/ OPERATIVE FAULTS/ LOCALISED PERIODONTAL PROBLEMS

Acute and Chronic Trauma

Trauma from occlusion may be acute or chronic. Acute trauma from occlusion results from an abrupt occlusal impact, such as that produced by biting on a hard object (e.g., olive pit). Restorations or prosthetic appliances that interfere with or alter the direction of occlusal forces on the teeth may also induce acute trauma.

Acute trauma results in tooth pain, sensitivity to percussion, and increased tooth mobility. If the force is dissipated by a shift in the position of the tooth or by wearing away or correction of the restoration, the injury heals, and the symptoms subside. Otherwise, periodontal injury may worsen and develop into necrosis, accompanied by periodontal abscess formation, or may persist as a symptom-free, chronic condition. Acute trauma can also produce cementum tears.

Chronic trauma from occlusion is more common than the acute form and is of greater clinical significance. It most often develops from gradual changes in occlusion produced by tooth wear, drifting movement, and extrusion of teeth, combined with parafunctional habits such as bruxism and clenching, rather than as a sequela of acute periodontal trauma.

Primary and Secondary Trauma from Occlusion

Trauma from occlusion may be caused by alterations in occlusal forces, reduced capacity of the periodontium to withstand occlusal forces, or both. When trauma from occlusion is the result of alterations in occlusal forces, it is called "**primary trauma from occlusion**." When it results from reduced ability of the tissues to resist the occlusal forces, it is known as "**secondary trauma from occlusion.**"

Primary trauma from occlusion occurs if trauma from occlusion is considered the primary etiologic factor in periodontal destruction and if the only local alteration to which a tooth is subjected is from occlusion. Examples include periodontal injury produced around teeth with a previously healthy periodontium after the (1) insertion of a "high filling," (2) insertion of a prosthetic replacement that creates excessive forces on abutment and antagonistic teeth, (3) drifting movement or extrusion of teeth into spaces created by unreplaced missing teeth, and (4) orthodontic movement of teeth into functionally unacceptable positions. Most studies on experimental animals of the effect of trauma from occlusion have examined the primary type of trauma. Changes produced by primary trauma do not alter the level of connective tissue attachment and do not initiate pocket formation. This is probably because the supra-crestal gingival fibres are not affected and therefore prevent apical migration of the junctional epithelium.

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Secondary trauma from occlusion occurs when the adaptive capacity of the tissues to withstand occlusal forces is impaired by bone loss resulting from marginal inflammation. This reduces the periodontal attachment area and alters the leverage on the remaining tissues. The periodontium becomes more vulnerable to injury, and previously well-tolerated occlusal forces become traumatic.

REFERENCE:
Carranza | Clinical cases in periodontics

GINGIVAL DEFECTS/LOCALISED RECESSION/ STILLMAN'S CLEFT/ MCCALLS FESTOONING

Gingival Recession

Exposure of the tooth by the apical migration of the gingiva is called gingival recession, or atrophy. gingival recession may result in accentuated sensitivity because of exposed dentin. Cementum becomes exposed to the oral environment in cases of gingival recession and as a result of loss of attachment in pocket formation.

Recession may be localized to one tooth or a group of teeth, or it may be generalized throughout the mouth.

Etiologic Factors

Gingival recession increases with age; the incidence varies from 8% in children to 100% after age 50 years. This has led some investigators to assume that recession may be a physiologic process related to aging. However, no convincing evidence has been presented for a physiologic shift of the gingival attachment.

The gradual apical shift is most likely the result of the cumulative effect of minor pathologic involvement and repeated minor direct trauma to the gingiva. In some populations without access to dental care, however, recession may be the result of increasing periodontal disease.

The following etiologic factors have been implicated in gingival recession:

- faulty toothbrushing technique (gingival abrasion)
- tooth malposition
- friction from soft tissues (gingival ablation)
- gingival inflammation
- abnormal frenum attachment
- iatrogenic dentistry

Trauma from occlusion has been suggested in the past, but its mechanism of action has never been demonstrated. For example, a *deep overbite* has been associated with gingival inflammation and recession. *Excessive incisal overlap* may result in a traumatic injury to the gingiva. *Orthodontic movement* in a labial direction in monkeys has been shown to result in loss of marginal bone and connective tissue attachment, as well as in gingival recession.

Standard oral hygiene procedures, whether toothbrushing or flossing, may lead to a frequent transient and minimal gingival injury.18,57 Although toothbrushing is important for gingival health, faulty toothbrushing technique or brushing with hard bristles may cause significant injury. This type of injury may present as lacerations, abrasions, keratosis and recession, with the facial marginal gingiva most affected.59 Thus, in these cases, recession tends to be more frequent and severe in patients with clinically healthy gingiva, little bacterial plaque, and good oral hygiene.

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GINGIVAL DEFECTS/LOCALISED RECESSION/ STILLMAN'S CLEFT/ MCCALLS FESTOONING

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Susceptibility to recession is also influenced by the position of teeth in the arch, the root-bone angle, and the mesiodistal curvature of the tooth surface. On rotated, tilted, or facially displaced teeth, the bony plate is thinned or reduced in height. Pressure from mastication or moderate toothbrushing damages the unsupported gingiva and produces recession. The effect of the angle of the root in the bone on recession is often observed in the maxillary molar area. If the lingual inclination of the palatal root is prominent or the buccal roots flare outward, the bone in the cervical area is thinned or shortened, and recession results from repeated trauma of the thin, marginal gingiva.

The health of the gingival tissue also depends on properly designed and placed restorative materials. Pressure from a poorly designed partial denture, such as ill-fitting denture clasp, can cause gingival trauma and recession. Overhanging dental restorations have long been viewed as a contributing factor to gingivitis because of plaque retention. In addition, there is general agreement that placing restorative margins within the biologic width frequently leads to gingival inflammation, clinical attachment loss, and eventually, bone loss. Clinically, the violation of biologic width typically manifests as gingival inflammation, deepened periodontal pockets, or gingival recession.

A relationship may exist between smoking and gingival recession. The multifactorial mechanisms may include reduced gingival blood flow and altered immune response but are not, as yet, conclusive.

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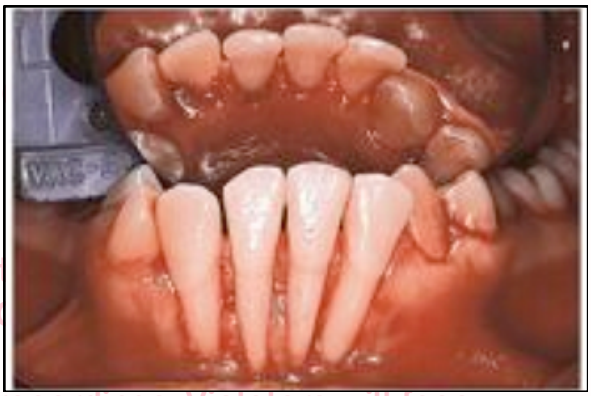
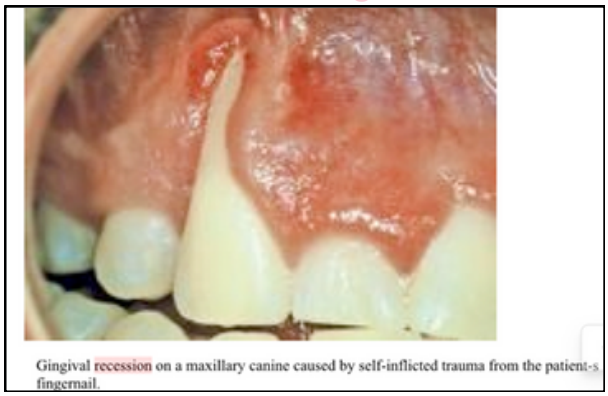
GINGIVAL DEFECTS/LOCALISED RECESSION/ STILLMAN'S CLEFT/ MCCALLS FESTOONING

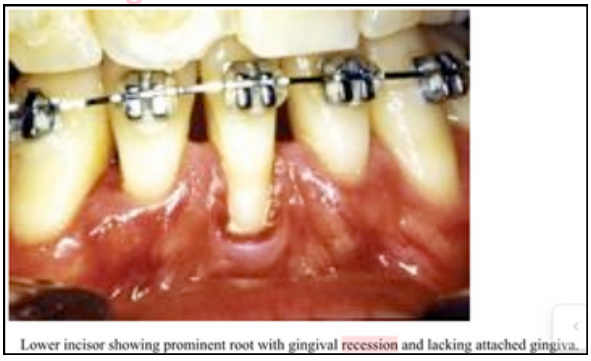
Clinical Significance

Several aspects of gingival recession make it clinically significant. Exposed root surfaces are susceptible to caries. Abrasion or erosion of the cementum exposed by recession leaves an underlying dentinal surface that can be sensitive. Hyperaemia of the pulp and associated symptoms may also result from excessive exposure of the root surface. Interproximal recession creates oral hygiene problems and resulting plaque accumulation.

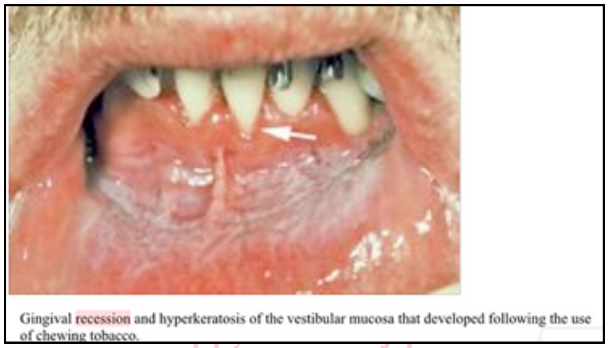




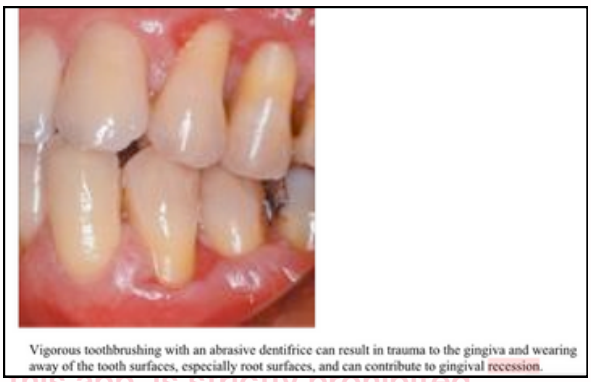
Gingival recession on a maxillary canine caused by self-inflicted trauma from the patient-s fingernail.



Lower incisor showing prominent root with gingival recession and lacking attached gingiva.



Gingival recession and hyperkeratosis of the vestibular mucosa that developed following the use of chewing tobacco.



Vigorous toothbrushing with an abrasive dentifrice can result in trauma to the gingiva and wearing away of the tooth surfaces, especially root surfaces, and can contribute to gingival recession.

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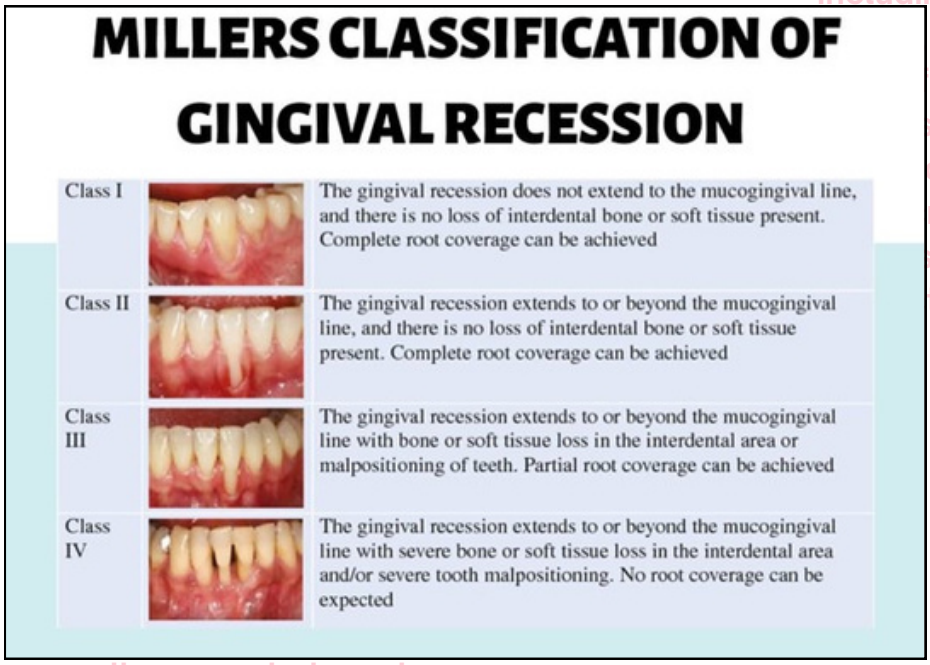
GINGIVAL DEFECTS/LOCALISED RECESSION/ STILLMAN'S CLEFT/ MCCALLS FESTOONING



Stillman's clefts and the McCall festoons

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

The term "Stillman's clefts" has been used to describe a specific type of gingival recession consisting of a narrow, triangular-shaped gingival recession. As the recession progresses apically, the cleft becomes broader, exposing the cementum of the root surface. When the lesion reaches the mucogingival junction, the apical border of oral mucosa is usually inflamed because of the difficulty in maintaining adequate plaque control at this site.

The term "McCall festoons" has been used to describe a rolled, thickened band of gingiva usually seen adjacent to the cuspids when recession approaches the mucogingival junction.

Initially, Stillman's clefts and McCall festoons were attributed to traumatic occlusion, and the recommended treatment was occlusal adjustment. However, this association was never proved, and these indentations merely represent peculiar inflammatory changes of the marginal gingiva.

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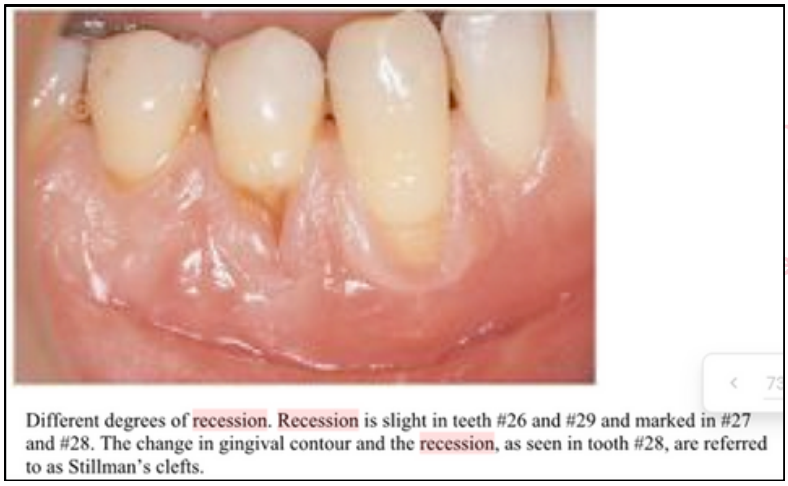
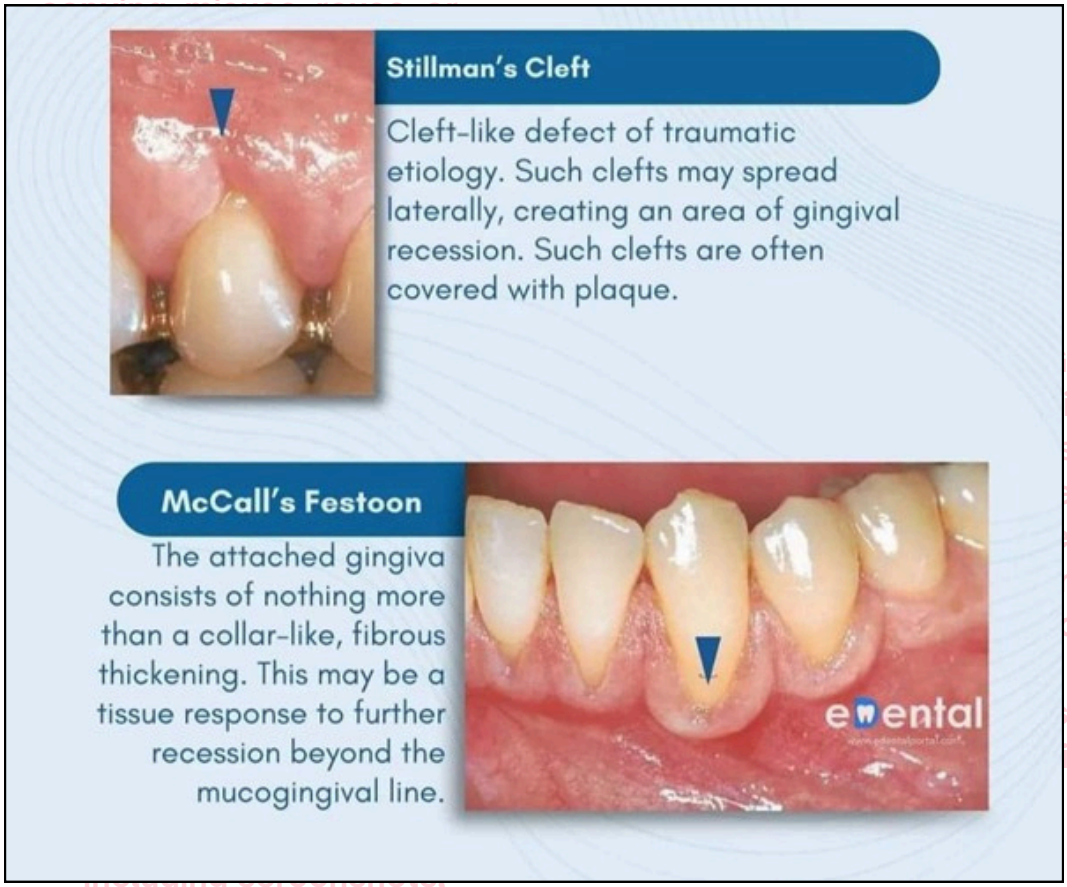


Different degrees of recession. Recession is slight in teeth #26 and #29 and marked in #27 and #28. The change in gingival contour and the recession, as seen in tooth #28, are referred to as Stillman's clefts.



REFERENCE:
Carranza

PERIO GRAFTS/FLAPS / SURGICAL PERIO

FLAP SURGERY

Periodontal flap surgery is almost invariably performed after a course of thorough non-surgical treatment. It should only be considered in highly motivated patients and the presence of optimal plaque and risk factor control. Following a course of non-surgical treatment in cases of moderate to advanced disease, and despite good plaque control, there may still be residual increased pockets and bleeding on probing. Most patients requiring periodontal surgery should be referred for specialist care unless you have the relevant expertise and experience.

The principle aims of periodontal flap surgery are:

a) Access for debridement

Removal of subgingival root surface deposits may be difficult where the pockets are deep or where access is poor, in particular molar teeth with complex root anatomy or furcation involvement. With a periodontal flap raised, the root surface can be visualised and cleaned until free of deposits. There may also be scope for pocket reduction or elimination by means of reshaping the bone and soft tissues during surgery. The aim is to achieve both shallow pockets and gingival tissues that are easier to access and clean, both by the patient and professional during maintenance.

ure 28 A: Periapical indicating an infrabony defect mesial LR6. B: The defect mesial to LR6 after thorough open flap debridement. C: Surgery complete and sutures placed. D: Two months post surgery.

Techniques for Access and Pocket Depth Reduction/Elimination

The modified Widman flap facilitates instrumentation but does not attempt to reduce pocket depth.

The *reduction or elimination of pocket depth* is the main purpose of two flap techniques: the *undisplaced flap and the apically displaced flap*. The decision of which to perform depends on two important anatomic landmarks:

- pocket depth and
- location of the mucogingival junction

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PERIO GRAFTS/FLAPS / SURGICAL PERIO

These landmarks establish the presence and width of the attached gingiva, which is the basis for the decision.

The modified Widman flap has been described for exposing the root surfaces for meticulous instrumentation and for removal of the pocket lining. 6 Again, it is not intended to eliminate or reduce pocket depth, except for the reduction that occurs in healing by tissue shrinkage.

The undisplaced (unrepositioned) flap, in addition to improving accessibility for instrumentation, removes the pocket wall, thereby reducing or eliminating the pocket. This is essentially an excisional procedure of the gingiva.

The apically displaced flap also improves accessibility and eliminates the pocket, but it does the latter by apically positioning the soft tissue wall of the pocket. 2 Therefore, it preserves or increases the width of the attached gingiva by transforming the previously unattached keratinized pocket wall into attached tissue. This increase in width of the band of attached gingiva is supposedly based on an apical shift of the mucogingival junction, which includes apical displacement of the muscle attachments. A study made before and 18 years after apically displaced flaps failed to show a permanent relocation of the mucogingival junction.

b) Regenerative surgery

Conventional periodontal flap surgery heals primarily with the formation of *a long junctional epithelium*. It is thought that this occurs as a result of epithelial cells being first to grow into the void left around the root surface following surgery.

Regenerative surgical procedures, in contrast, aim to promote the regeneration of the periodontal tissues that have been lost through the disease process. *The aim is thus to promote re-growth of cementum, periodontal ligament (PDL) and alveolar bone*. Such treatment can be more effective in achieving shallow pockets than conventional periodontal surgery in certain situations, such as deep narrow vertical bone defects.

Several regenerative approaches are currently in use and are termed *Guided Periodontal Tissue Regeneration (GPTR)*. The aim is to prevent the rapid down growth of epithelial cells into the void after periodontal surgery by introducing a membrane (resorbable or non-resorbable) and hence allowing a protected area for the slower turnover tissues, such as bone and PDL, to form.

The use of enamel matrix protein based regenerative materials, such as Emdogain®, may also be advantageous in terms of attachment gain and probing depth reduction. When applied to a defect, after open flap debridement and surface treatment, enamel matrix proteins aggregate to form a scaffold which promotes bone formation in the defect. Alternatively, the defect can also be directly filled with filler materials to ‘graft’ the defect (as illustrated below). These fillers may either be bone grafts from the patient or from human, animal or artificial sources.

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PERIO GRAFTS/FLAPS / SURGICAL PERIO

Figure 29 Intrabony defect mesial to LR6 after thorough open flap debridement. B: Filler covered with a membrane.

Figure 30 Before and after radiographs of a regeneration surgery.

FLAPS FOR RECONSTRUCTIVE SURGERY

In current reconstructive therapy, bone grafts, membranes, or a combination of these, with or without other agents, are used for a successful outcome (see Chapter 67). The flap design should therefore be set up so that the maximum amount of gingival tissue and papilla are retained to cover the material(s) placed in the pocket.

Two flap designs are available for reconstructive surgery:

- the papilla preservation flap and
- the conventional flap with only crevicular incisions.

The flap design of choice is the papilla preservation flap, which retains the entire papilla covering the lesion. However, to use this flap, there must be adequate interdental space to allow the intact papilla to be reflected with the facial or lingual/palatal flap. When the interdental space is very narrow, making it impossible to perform a papilla preservation flap, a conventional flap with only crevicular incisions is made.

c) Crown lengthening

Crown lengthening surgery involves the removal of the periodontal tissues to increase the clinical crown height for aesthetic reasons or to provide adequate sound tooth tissue for restoration. Crown lengthening surgery may be limited to the soft tissue when the thickness of the tissues are excessive. In such cases this can be performed using a scalpel, electrosurgery or soft tissue lasers. However, the dento-gingival anatomy and the position of the soft tissue are, to a large degree, dictated by the position of the underlying bone. In these cases, following removal of soft tissue alone, the gingival margin will rebound during healing to re-establish the soft tissue height above the bone crest, with loss of the amount of crown that was surgically exposed. In such cases a stable position can only be achieved by shifting the entire dento-gingival complex apically. This requires bone removal, and to access the bone a periodontal flap must be raised.

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PERIO GRAFTS/FLAPS / SURGICAL PERIO

Crown lengthening can be performed to facilitate restorative dentistry and allow access to subgingival restoration margins. Subgingival margins may result from oblique vertical fracture of a cusp or from the removal of extensive caries. In some cases, such as severe toothwear, there may not be adequate coronal tissue for mechanical retention of extracoronal restorations. Crown lengthening is a way to increase this.

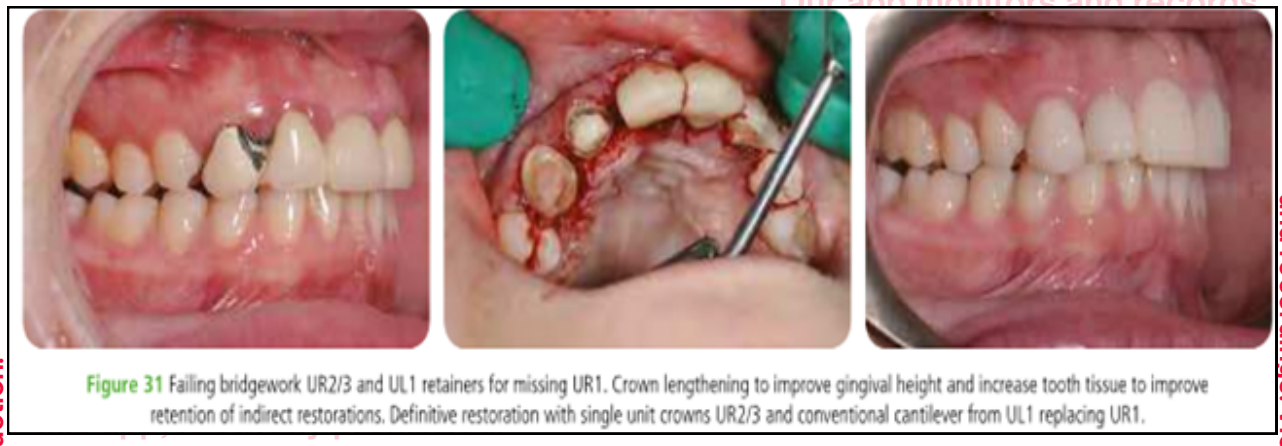


Figure 31 Failing bridgework UR2/3 and UL1 retainers for missing UR1. Crown lengthening to improve gingival height and increase tooth tissue to improve retention of indirect restorations. Definitive restoration with single unit crowns UR2/3 and conventional cantilever from UL1 replacing UR1.

Aesthetic crown lengthening uses the same techniques but applied to a different situation. In patients with a high smile line and where the anatomical crown is still partially hidden by an excess of soft tissues (as in cases with delayed passive eruption) a simple gingivectomy may be enough to achieve the desired result.

In patients where there is an excess of both soft and hard tissue (as in cases of tooth wear with compensatory over eruption) careful planning with diagnostic wax ups and a periodontal flap procedure with appropriate bone removal may be required to achieve correct tooth and gingival dimensions. If crowns or veneers are required as part of this treatment you will need to wait four to six months for the new gingival contour to stabilise before placement.

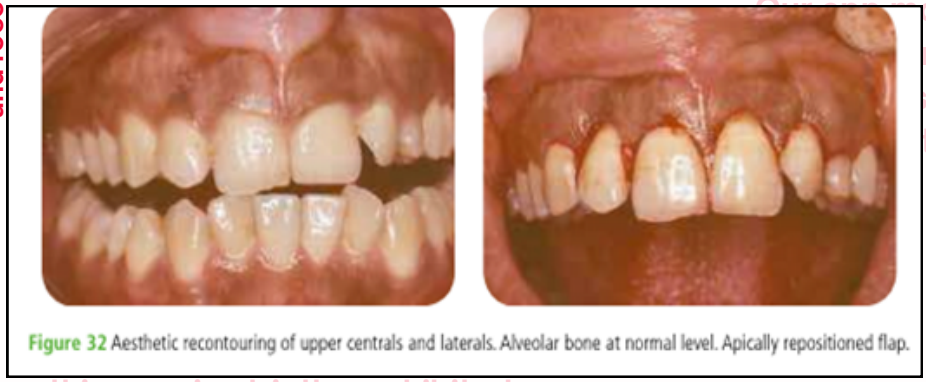


Figure 32 Aesthetic recontouring of upper centrals and laterals. Alveolar bone at normal level. Apically repositioned flap.

PERIO GRAFTS/FLAPS / SURGICAL PERIO

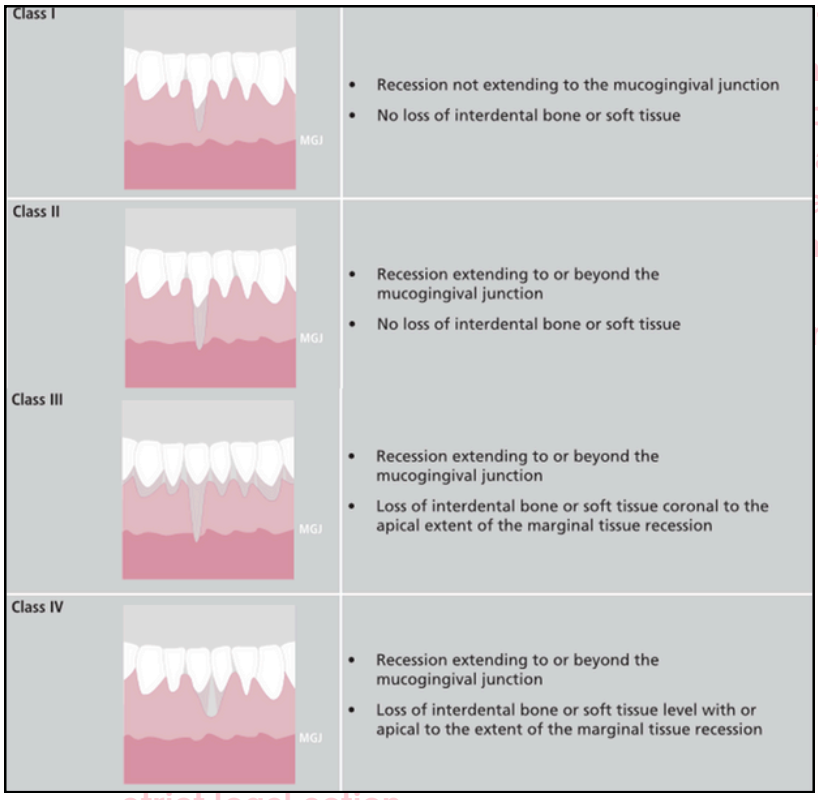
d) Management of recession (Mucogingival surgery)

There are a number of reliable periodontal surgical techniques available to manage mucogingival problems such as gingival recession. The key indication for such surgery is aesthetics. Whilst other complications of recession can include temperature sensitivity and root caries, these would normally be managed conservatively by appropriate care including dietary analysis, tailored oral hygiene instruction and use of high concentration fluoride preparations.

This type of surgery is technically demanding but can improve aesthetics and long-term stability. The likely extent of coverage can be assessed using the Miller's classification of the initial recession defect. As a rule surgical root coverage procedures should only be considered for Miller class I and II defects (i.e.periodontally healthy patients)

Figure 33 A: Class I recession defect on UL3. B: Defect following mucogingival surgery.

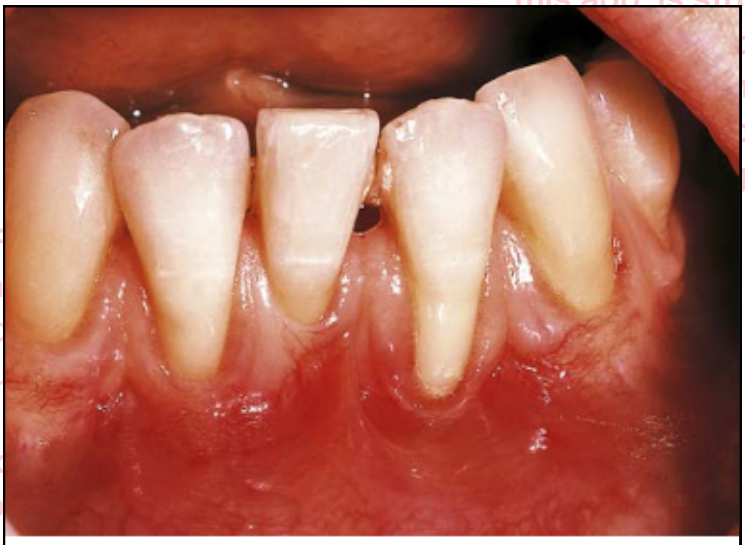
Miller's classification



PERIO GRAFTS/FLAPS / SURGICAL PERIO

To understand more about the mucogingival surgery, below clinical case has been discussed from the book ODELL.

A 30-year-old woman has gingival recession. Assess her condition, and discuss treatment options



• **Fig. 4.1** The labial view of the lower incisors on presentation.

DIAGNOSIS

What is Your Diagnosis, and What is the Likely Aetiology?

The patient has gingival recession. In this case, the assessment has not provided a diagnosis any more accurate than that given by the patient, but the features should give some clues to the possible aetiology.

Recession is probably multifactorial in aetiology. The most important factor is probably anatomical variations among patients. Some individuals have very thin gingival tissue buccally, both soft tissue and bone. When the buccal plate of the alveolus is thin, bony dehiscence or fenestrations below the soft tissue are more likely. For these reasons, there is more recession on the teeth that are prominent in the arch and least on slightly instanding teeth (see the more instanding central incisor in Fig. 4.1). When these predisposing factors are present, other insults become important. The most important is probably traumatic toothbrushing. Plaque-induced marginal inflammation will also destroy the thin tissue at this site relatively quickly. Traumatic occlusion may also contribute.

In this case, the patient is maintaining a very good standard of plaque control, and there is no cervical abrasion, which might be further evidence of toothbrush trauma.

PERIO GRAFTS/FLAPS / SURGICAL PERIO

Treatment	Effectiveness
Mucogingival surgery to correct the recession, using either coronally advanced or tunnel flaps. A lateral pedicle graft or a double papilla flap are also options. These may be used in conjunction with an interpositional (subepithelial) connective tissue graft in thin periodontal biotypes. These are essentially aesthetic procedures.	May be effective in carefully selected cases. The presence of adjacent interdental papillae and suitable donor sites is essential. Total root coverage is difficult to achieve and unpredictable, especially in the long term.
Mucogingival surgery to provide a wider and functional zone of attached gingiva. This therapeutic procedure provides a zone of thicker tissue that is more resistant to further recession and less prone to soreness with normal brushing. A free gingival graft is the treatment of choice.	Highly effective. Grafting palatal mucosa into the alveolar mucosa prevents the lip pulling the gingiva from the teeth. Even if the gingival margin has little attached gingiva, it can remain healthy if protected from displacement or other trauma.
Provision of a thin acrylic gingival stent or veneer.	Can provide an excellent cosmetic result if well made, but only considered for extensive recession in highly visible areas. The usual indication is to improve the appearance of the upper anterior region following treatment of advanced periodontitis resulting in the loss of papillae

Mucogingival surgery to correct the recession:

- **Lateral pedicle graft**
- **Double papillae flap**
- **Coronally positioned flap**

Mucogingival surgery to provide a wider and functional zone of attached gingiva:

- **A free gingival graft**

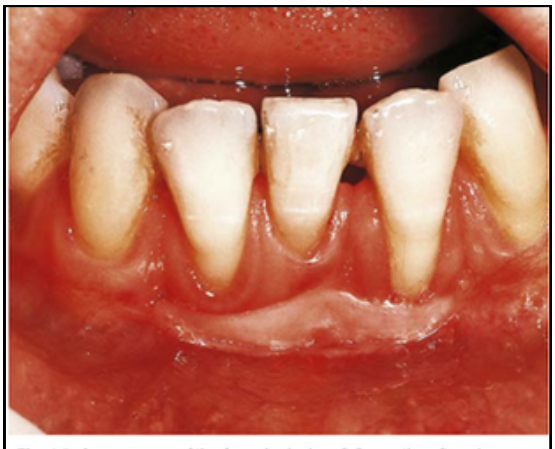


Fig. 4.2 Appearance of the free gingival graft 6 months after placement

PERIO GRAFTS/FLAPS / SURGICAL PERIO

What Do You See; is the Graft Successful?

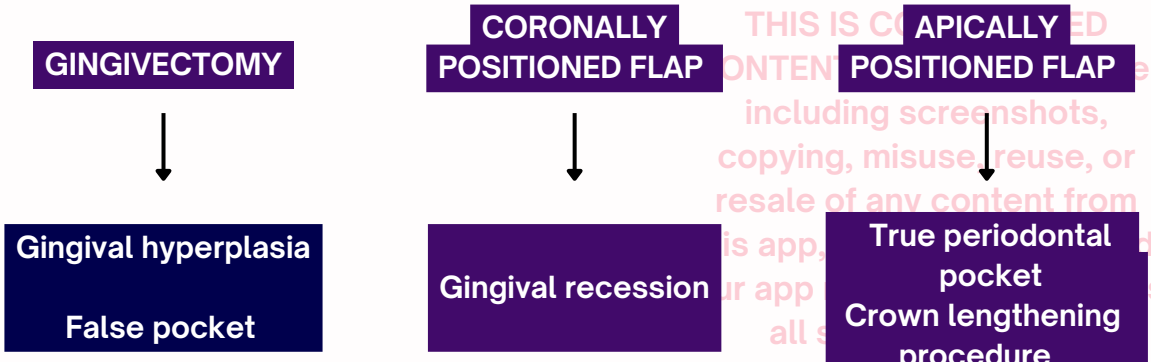
Yes, the graft appears successful. Palatal connective tissue and overlying epithelium have been placed apical to the lower incisor gingival margin to provide a wider zone of attached keratinized gingiva. Because the palatal connective tissue is transferred, the epithelium retains its keratinized palate structure.

Does the Graft Need To Lie At the Gingival Margin?

No. the graft forms the gingival margin on the lower left lateral incisor, but elsewhere, it lies below the margin. Provided the graft is firmly bound down to the underlying tissue, it will stabilize the gingival margin against displacement of lip movement.

Why Not Place the Graft Over the Root As Well and Correct the Recession?

Asnoted in Table 4.2, surgical correctionof the recessionitself is difficultto achieve andthe results unpredictable, especiallyin the longterm. The rootsurface does notprovide a nutrient bed on which the free graft can survive. Graft inthis situation wouldhave to bepedicled to ensuretheir nutrient supplyand also needto be placedsuch that theyreceive some nutrientsfrom an adjacentexposed connective tissuebed. A morepredictable result maybe obtained byusing an interpositional (subepithelial) connectivetissue graft andwould be requiredin cases withthin periodontal biotypes. Afree graft ismost unlikely tobe successful ifsim- ply placed over the root surface.



Graft Materials and Procedures

Numerous therapeutic grafting modalities for restoring periodontal osseous defects have been investigated. Material to be grafted can be obtained from the same individual *(autografts)*, from a different individual of the same species *(allografts)*, or from a different species *(xenografts)*.

PERIO GRAFTS/FLAPS / SURGICAL PERIO

CLASSIFICATION OF THE BONE GRAFTING MATERIALS

Autogenous Bone Bone from same individual	Allogenic Bone Bone from same species from another individual	Xenogenic Bone Material of biologic origin but from another species	Alloplastic Bone Material of synthetic origin
Block graft	Free frozen bone	Material derived from animal bone	Calcium phosphates
Bone mill Bone scraper Suction device Piezo Surgery	Freeze-dried bone allograft	Material derived from corals	Glass ceramics
	Demineralized freeze-dried bone allograft	Material derived from calcifying algae	Polymers
	Deproteinized bone allograft	Material derived from wood	Metals

There are 3 types of potentials in bone graft:

Bone graft materials are generally evaluated based on their osteogenic, osteoinductive, or osteoconductive potential.

- **Osteogenesis** refers to the formation or development of new bone by cells contained in the graft.
- **Osteoinduction** is a chemical process by which molecules contained in the graft (bone morphogenetic proteins) convert the neighbouring cells into osteoblasts, which in turn form bone.
- **Osteoconduction** is a physical effect by which the matrix of the graft forms a scaffold that favours outside cells to penetrate the graft and form new bone.

REFERENCE:
Carranza_10th edition | The Good Practitioner's Guide to Periodontology | Odell's Clinical Problem Solving in Dentistry, 4TH edition

PERIODONTAL DIAGNOSIS

Risk assessment

There are a number of risk factors for periodontal disease and the information collected during the history taking phase starts your periodontal risk assessment of the patient. On completing the periodontal screening, you should have a summary of the likely factors playing a role in their disease and together with their BPE score you can determine what further investigations are necessary.

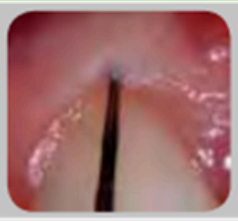
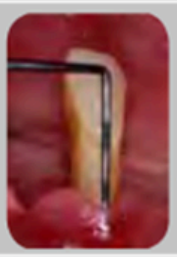
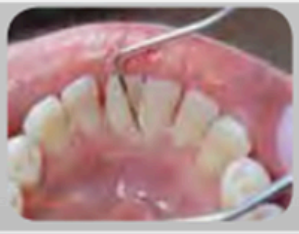
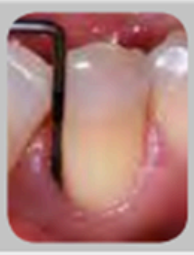
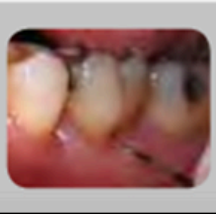
Visual inspection of gingival soft tissues

Prior to probing, you should assess and record the visual status of the gingival tissues for inflammation. Redness or change in contour of the gingival margins or interdental papillae indicates gingivitis and a need for oral hygiene modification.

BASIC PERIODONTAL EXAMINATION (BPE)

Code	Guidance	Special investigations	Periodontal reassessment
0	No need for periodontal treatment	None indicated	Repeat BPE at next check up appointment
1	Oral hygiene instruction (OHI)	Plaque and bleeding charts	Repeat BPE at next check up appointment
2	As for code 1, plus removal of plaque retentive factors, including all supra and subgingival calculus	Plaque and bleeding charts	Repeat BPE at next check up appointment
3	As for code 2 and OHI, root surface debridement (RSD) if required	• Plaque and bleeding charts • Radiographs should be considered (in order to establish if there is attachment loss)	Periodontal charting of sextants scoring 3, after initial therapy
4	OHI, RSD. Access the need for more complex treatment; referral to a specialist may be indicated	• Plaque and bleeding charts • Radiographs should be taken	Full periodontal charting before and after treatment
*	Treat according to BPE code (0-4). Assess the need for more complex treatment; referral to a specialist may be indicated	• Plaque and bleeding charts • Radiographs should be considered	Full periodontal charting before and after treatment

PERIODONTAL DIAGNOSIS

BPE code	Probing depth	Observation	
0	Pockets < 3.5mm First black band completely visible	Healthy periodontal tissues No calculus/overhangs No bleeding on probing	
1	Pockets < 3.5mm First black band completely visible	Bleeding on probing No calculus/overhangs	 (Note the recession in this image is not accounted for in the BPE)
2	Pockets < 3.5mm First black band completely visible	Supra or subgingival calculus or plaque retention factor (overhang)	
3	Probing depth 3.5 - 5.5mm	First black band partially visible, indicating pocket of 4-5mm	
4	Probing depth > 5.5mm	First black band entirely within the pocket, indicating pocket of 6mm or more	
*	Furcation involvement	Detection of a furcation	

PERIODONTAL DIAGNOSIS

Detailed periodontal charting

Codes of 3, 4 and * require further investigation and detailed periodontal charting should be performed. The following can be recorded; probing depth, bleeding on probing (recession, mobility and furcation involvement). The minimum requirement is to record all sites ⩾ 4mm and bleeding on probing. Periodontal charting forms an important part of your record keeping and should be accurate and kept up to date.

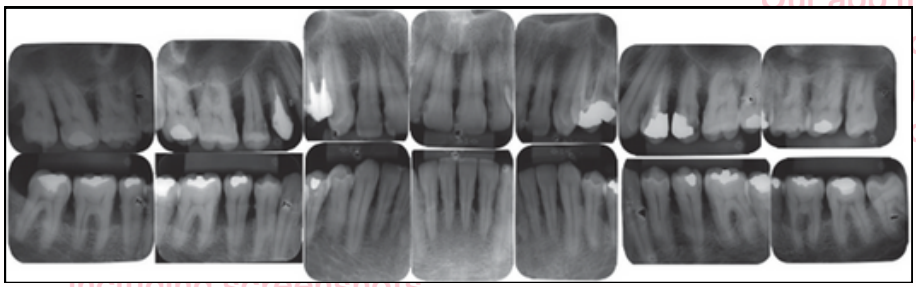


Figure 2: Periodontal chart, initial visit. Courtesy of Dr. Eduardo Sampaio and Dr. Marcelo Faveri.

Visit: Initial Exam

Surface	Buccal															
Tooth	2	3	4	5	6	7	8	9	10	11	12	13	14	15		
PD	8 3 4	5 3 4	7 2 5	4 2 4	4 2 3	5 1 3	3 1 4	4 1 2	3 1 3	4 1 4	4 1 6	5 2 6	6 2 6	2 7		
CEJ-GM	2 -2 -1	-1 -2 -1	1 -1 1	1 -1 1	2 0 0	1 0 0	0 0 1	2 -1 -1	0 0 0	1 0 2	2 -1 2	1 -1 2	2 -2 1	1 -1 2		
CAL	6 5 5	6 5 5	6 3 4	3 3 3	2 2 3	4 1 3	3 1 3	2 2 3	3 1 3	3 1 2	2 2 4	4 3 4	4 4 5	5 3 5		
BOP	1 1 1	1 1 1	1 0 0	1 0 1	1 0 0	1 0 0	0 0 1	1 0 0	0 0 0	0 0 1	1 0 1	1 1 1	1 1 1	1 1 1		
Surface	Palatal															
Tooth	2	3	4	5	6	7	8	9	10	11	12	13	14	15		
PD	8 2 7	7 2 6	7 2 5	5 2 5	4 3 4	5 4 4	3 2 3	3 3 3	4 3 4	3 3 3	3 2 4	5 2 7	6 3 7	7 3 8		
CEJ-GM	2 0 2	-1 2 2	0 2 2	0 2 2	1 1 1	1 1 1	1 0 1	1 0 0	0 0 1	1 0 1	1 1 1	1 0 2	2 0 2	1 2		
CAL	6 2 5	5 3 4	5 2 3	3 2 3	2 2 3	4 3 3	2 2 2	2 3 3	4 3 3	2 3 2	2 1 3	4 2 5	4 3 5	5 2 6		
BOP	1 1 1	1 1 1	1 1 1	1 1 1	0 0 1	1 1 1	1 1 1	1 1 1	1 0 1	1 1 1	1 1 1	1 1 1	1 1 1	1 1 1		
Surface	Buccal															
Tooth	31	30	29	28	27	26	25	24	23	22	21	20	19	18		
PD	3 2 3	5 1 1	3 1 3	1 1 2	2 1 1	1 1 1	1 1 2	2 1 2	1 1 1	1 1 1	1 1 1	2 1 2	2 1 3	3 1 3		
CEJ-GM	3 0 2	2 -1 0	1 0 1	0 -2 1	0 0 -1	-1 0 -2	-2 0 -2	-2 0 -2	-1 0 0	1 0 0	0 -1 1	1 -1 1	1 0 1	1 -1 2		
CAL	0 2 1	3 2 1	2 1 2	1 3 1	2 1 2	2 1 3	3 1 4	4 1 4	2 1 1	0 1 1	1 2 0	1 2 1	1 1 2	2 2 1		
BOP	1 0 1	1 0 1	0 0 0	0 0 0	0 0 0	0 0 0	0 0 0	0 0 0	0 0 0	0 0 0	0 0 0	1 0 1	1 0 1	1 0 1		
Surface	Lingual															
Tooth	31	30	29	28	27	26	25	24	23	22	21	20	19	18		
PD	4 2 6	6 4 5	5 4 4	4 1 2	3 1 3	3 2 2	2 1 2	2 3 4	4 3 3	4 1 3	4 2 5	6 3 5	4 2 6	6 3 2		
CEJ-GM	4 2 2	2 1 2	2 2 2	2 0 2	2 0 1	1 -1 -2	-2 -3 -3	-3 -3 -2	-2 -1 0	2 1 2	1 0 2	2 2 2	2 2 2	2 2 2		
CAL	0 0 4	4 3 3	3 2 2	2 1 0	1 1 2	2 3 4	4 4 5	5 6 6	6 4 3	2 0 1	3 2 3	4 1 3	2 0 4	4 1 0		
BOP	1 1 1	1 1 1	1 1 1	1 1 1	0 1 1	1 1 1	1 1 1	1 1 1	1 1 1	0 1 1	1 1 1	1 1 1	1 1 1	1 1 1		

A standard periodontal probe is necessary for the detailed data collection you need to enable you to monitor the progress of specific sites. Popular probes include the 10mm Williams probe and the 15mm UNC probe.

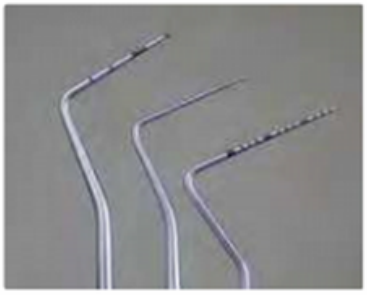


Figure 21 BPE probe, William's probe (1, 2, 3, 5, 7, 8, 9 and 10mm markings) and UNC 15 probe (1, 2, 3, 4-5, 6, 7, 8, 9-10, 11, 12, 13, 14-15).

The probing depth at any site dictates the patient's ability to maintain soft tissue health by optimal plaque control. Probing depths of 4mm or more are considered to be too deep to be controlled by tooth brushing and interdental cleaning alone. These sites should be considered for active periodontal therapy.

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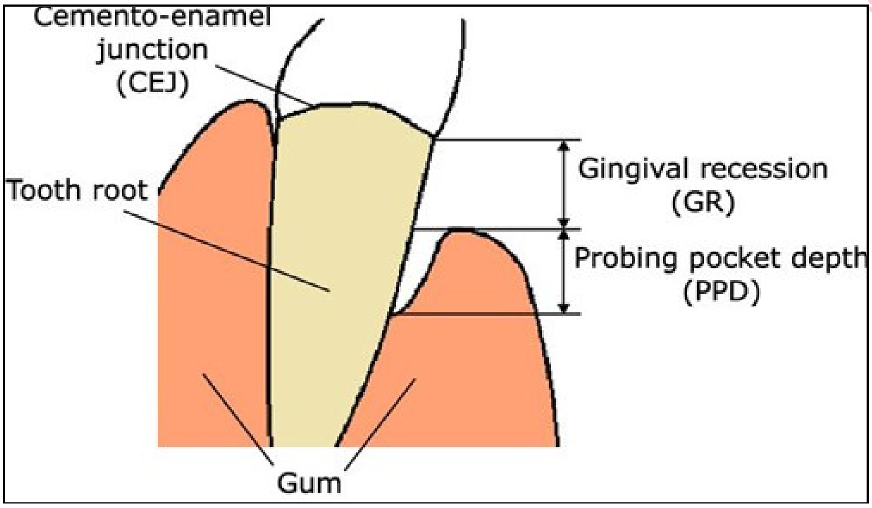
PERIODONTAL DIAGNOSIS

The periodontal probe answers two questions:

1. **Where is the base of the gingival crevice?**

Attachment loss = the distance from the cemento-enamel junction to the bottom of pocket.
Probing depth (PD) = the distance from the gingival margin to the bottom of the pocket.
Gingival recession = distance from the cemento-enamel junction to the gingival margin.
Therefore;

Attachment loss = Probing depth (PD) + Gingival recession



2. Does the tissue bleed on probing (BOP)?

This is a measure of inflammation, not necessarily active tissue destruction. No bleeding on probing means health (except in smokers, where it is hidden). Bleeding from the gingival margin is an indicator of gingivitis and will respond quickly to improvements in daily plaque removal. Bleeding from the base of the pocket represents periodontitis and is more reflective of the response to periodontal treatment such as root debridement. Bleeding should be recorded on the periodontal chart pre- and post-treatment to guide your therapy to the affected sites and monitor treatment. An absence of bleeding on probing (BOP) from the bottom of the pocket predicts periodontal stability and is a useful indicator at the reassessment stage.

PERIODONTAL DIAGNOSIS

Elements of a full diagnosis

The first question we need to ask is whether there is any attachment loss. If there is no attachment loss the patient is given a diagnosis of health or gingivitis (if there is inflammation present). If there is attachment loss and no other systemic condition, then the diagnosis will either be chronic or aggressive periodontitis. The patient's level of risk is important here.

Once the type of periodontal disease has been established, the next consideration is the pattern of the disease. Using the periodontal chart, if more than 30% of sites are involved then a diagnosis of generalised disease is given. If less than 30% of sites are involved, then a diagnosis of localised disease is given.

The final element of the diagnosis is an indication of the extent of the disease. This is deemed mild (1-2mm), moderate (3-4mm) or severe (⩾ 5mm) depending on the amount of attachment loss present.

Taking all this information together will permit you to provide a comprehensive diagnosis for your patient. Only once the diagnosis is formulated can the prognosis and treatment plan be considered.

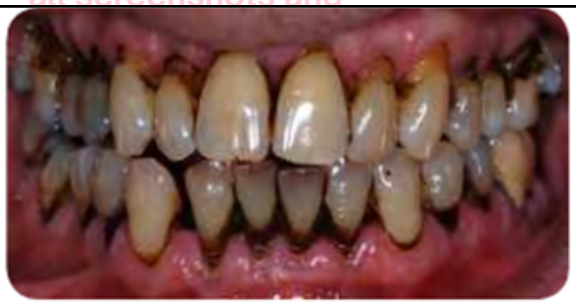


Figure 24 Diagnosis: Moderate generalised chronic periodontitis (smoking associated).

REFERENCE:
The Good Practitioner's Guide to Periodontology | Clinical Cases in Periodontics | Oral diagnosis and treatment planning: part 6. Preventive and treatment planning for periodontal disease

PERIODONTITIS AND SMOKING & SMOKING CESSATION

The main *etiologic factor*, which can cause initiation and progression of *periodontal disease*, is the infection produced by dental plaque. Althoughgingivitis and periodontitis are elicited by bacteria, smoking has been shown to represent a strong risk marker and probably *true risk factor* for the development and progression of *periodontal disease*.

> smoking is a risk factor even for individuals with good oral hygiene and the combined effect of smoking and plaque infection are likely to be more destructive than either factor alone.

Current smokers are 2.5 to 6 times more likely to have periodontitis than non-smokers. Former smokers are almost twice as likely to have periodontitis than people who have never smoked. Smokers are three times more likely to suffer from severe periodontitis compared to non-smokers. There is also evidence that smokers experience greater tooth loss than non- smokers.

Smoking causes irreversible damage to the periodontium; however, the progress of the disease can be stopped,and further damage prevented by cessation of smoking.

Smoking may also be an important factor in refractory periodontal disease as most individuals with refractory periodontitis are heavy smokers.

RISK FACTORS FOR PERIODONTITIS:

Modifiable Risk Factors:

- Smoking
- Diabetes mellitus
- Socio-economic status
- Psychological factors
- Stress
- Nutrition

Non-Modifiable Risk Factors:

- Genetic factors
- Osteoporosis
- Other systemics diseases (Chediak-Higashi syndrome, cyclic neutropenias, lazy leukocyte syndrome, agranulocytosis and leukocyte adhesiondeficiency and Down syndrome and
- Papillon–Lefevre syndrome)
- Ageing

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PERIODONTITIS AND SMOKING & SMOKING CESSATION

Smoking also increases the risk for:

- *osteoporosis*
- severe periodontitis in HIV-infected individuals
- *progressive periodontal attachment loss*
- *periodontal disease in diabetics*
- *root caries*
- oral leukoplakia
- cancer
- lung and cardiovascular diseases
- poor pregnancy outcomes
- implant failure

WHAT IS CLINICAL ATTACHMENT LOSS?

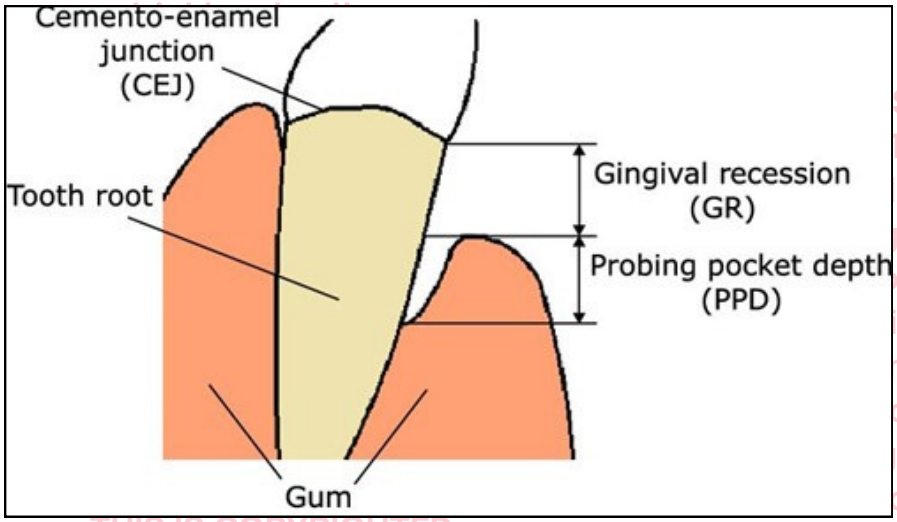
Attachment loss = the distance from the cemento-enamel junction to the bottom of pocket.

Probing depth (PD) = the distance from the gingival margin to the bottom of the pocket.

Gingival recession = distance from the cemento-enamel junction to the gingival margin.

Therefore;

Attachment loss = Probing depth (PD) + Gingival recession



EFFECTS OF SMOKING:

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PERIODONTITIS AND SMOKING & SMOKING CESSATION

EFFECTS OF SMOKING:

- Gingival bleeding tissue and all the signs of gingival inflammation were considerably *lesser* and gingival tissue has a thickened and fibrotic appearance in current cigarette smokers than the non-smokers.
- Tendency for higher prevalence supra and subgingival calculus and plaqueformation in smokers.
- Increased probingdepth, furcation involvement, gingival recession, missingteeth and tooth mobility is significantly greater in smokers than in non- smokers.
- radiographic examination shows mild to moderate bone loss in the smokers, regardless of the amount of local factor or level of inflammation.
- Nicotine can suppress the proliferation of cultured osteoblasts while stimulating osteoblast alkaline phosphatase activity. Therefore, influence of smoking on reduction in bone height is greater in adult smokers.
- clinical attachment loss is significantly greater in the smoker's groupthan non-smokers.
- Serum IgG antibodies to Provetella intermedia and Fusobacterium nucleatum also have been reported to be reduced in smokers.
- Inflamed sites in smokershave reduced vasculardensity and angiogenesis compared to inflamed sites in nonsmokers, thus impairing inflammatory response and wound healing.
- smoking has a strongnegative effect in response to periodontal treatment and other oral surgical interventions. Smoking is therefore a contraindication to periodontal therapy. 90% of cases not responsive to periodontal treatment occur in smokers.
- associated with a deficienthost immune function, causing an increased risk of disease and poor wound healing.
- Smoking also impairs the revascularisation of bone and soft tissues,further impairing healing.

PATHOPHYSIOLOGY:

Tobacco smoke contains many cytotoxic substances, such as nicotine,which can penetrate the soft tissue of the oral cavity, adhere to the tooth surface or enter to the blood stream.

local vasoconstriction

↓

reducing blood flow

↓

decreasing gingival inflammation, redness and bleeding

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PERIODONTITIS AND SMOKING & SMOKING CESSATION

The gingival vasoconstrictive episodes during repeated daily exposure to cigarette smoke might in long run result in vascular dysfunction in the gingival tissues.

Decreases blood flow

↓

reduced nutrients reaching cells

↓

decrease the ability of the body to fight against infection (reduced immunity)

Decreases saliva less buffering action

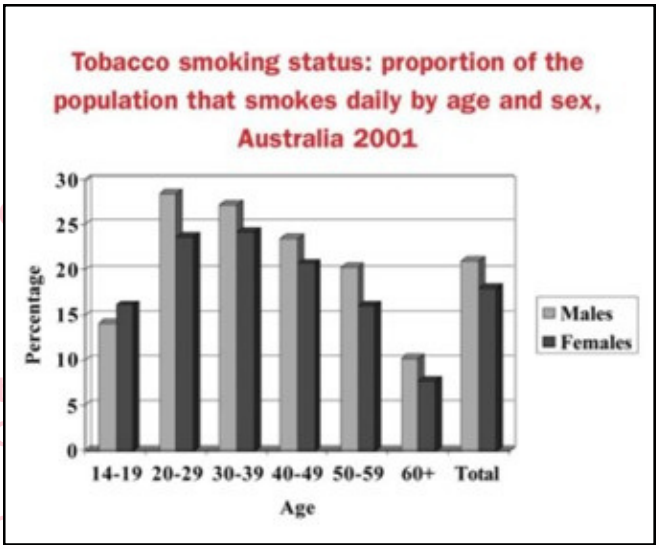
↓

more caries

↓

more plaque and calculus

- The highestrate of smoking is found in males aged 20-29years (28%)
- The highestrate for femalesis in the 30-39 years age group(24%)
- The lowest rate of smoking is found among those aged 60+ years
- It is disturbing that smoking amongadolescents (14-19 years) is greaterthan ever and is still rising, with more females (16%) taking up the habit than males (14%)
- The majorityof smokers (89%) start in theirteenage years; therefore, it is important to target this age group with some prevention strategies.



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PERIODONTITIS AND SMOKING & SMOKING CESSATION

NECROTISING PERIODONTAL DISEASE:

Necrotising periodontal disease is an acute painful condition characterised by gingival bleeding and necrosisor ulceration of the interdental papillae, which is often covered with a greyish pseudomembrane. It is usually associated with halitosis and can be associated with swollen glands (lymphadenopathy) and fever. Necrotising periodontal diseaseis classified by the extent of inflammation or necrosis.

NECROTISING GINGIVITIS:

Acute necrotizing ulcerative gingivitis (ANUG) occursmore frequently in smokers and rarely occurs in children. Children thought to have necrotising gingivitis should be assessed for acute herpetic gingivostomatitis
Possible explanations for the increased frequency of ANUG in smokers include the vasoconstriction of gingivalblood vessels, reduced activity of leukocytes and proliferation of anaerobic fusospirochaetal microorganisms.
Affects the interdental papillae and gingivae; if not managed appropriately, it can spreadto involve the bone.

NECROTSING PERIODONTITIS:

Affects the periodontium and results in bone loss. Promptly refer for specialist management.

NECROTISING STOMATITIS:

Affects the periodontium, bone and soft tissues of the oral cavity. Promptlyrefer for specialist management.

SMOKING CESSATION GUIDELINES:

New smoking cessation questions and recommendations:

Since the minor update in 2014, the field of smoking cessation has moved forward.It now includes more sophisticated pharmacology, technology in the form of quitting apps, and controversial nicotine delivery modalities such as electronic cigarettes (i.e. e-cigarettes). New topics identified by the Expert Advisory Group included questions on:

- combinations and dosage of pharmacotherapies
- relapse prevention
- use of nicotine replacement therapy (NRT) duringpregnancy
- nicotine containing e-cigarettes as a cessation aid.

PERIODONTITIS AND SMOKING & SMOKING CESSATION

Summary of recommendations:

The role of health professionals:

Recommendation 1
All people who smoke should be offered brief advice to quit smoking. Strong recommendation, high certainty.

Recommendation 2
A system for identifying all people who smoke and documenting tobacco use should be used in every practice or healthcare service.
Strong recommendation, high certainty

Recommendation 3
Offer brief smokingcessation advice in routine consultations and appointments, whenever possible.
Strong recommendation, high certainty

Recommendation 4
Offer follow-up to all people who are attempting to quit smoking. Strong recommendation, high certainty

Pharmacotherapy for smoking cessation:

Recommendation 5
In the absence of contraindications, pharmacotherapy (nicotine replacement therapy, varenicline or bupropion) is an effective aid when accompanied by behavioural support, and should be recommended to all people who smoke who have evidence of nicotine dependence. Choice of pharmacotherapy is based on efficacy, clinical suitability and patient preference.
Strong recommendation, high certainty.

Recommendation 6
Combination nicotine replacement therapy (NRT) (i.e. patch and oral form) accompanied by behavioural support is more effective than NRT monotherapy accompanied by behavioural support, and should be recommended to people who smoke who have evidence of nicotine dependence. Strong recommendation, moderate certainty

Recommendation 7
For people who have stopped smoking at the end of a standard course of nicotine replacement therapy(NRT), clinicians may consider recommending an additional course of NRT to reduce relapse.
Conditional recommendation for intervention, low certainty

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PERIODONTITIS AND SMOKING & SMOKING CESSATION

Recommendation 8

a) Nicotine replacement therapy (NRT) is safe to use in patients with stable cardiovascular disease.
Strong recommendation, high certainty

b) NRT shouldbe used with caution in patients who have had a recentmyocardial infarction, unstable angina,severe arrhythmias or recent cerebrovascular events.
Strong recommendation, moderate certainty

Recommendation 9

For women who are pregnantand unable to quit smokingwith behavioural support alone, clinicians might recommend nicotinereplacement therapy (NRT), compared with no NRT. Behavioural support and monitoring should also be provided. Conditional recommendation for intervention, low certainty

Recommendation 10

Varenicline should be recommended to people who smoke and who have been assessed as clinically suitable for this medication;
it should be provided in combination with behavioural support. Strong recommendation, high certainty

Recommendation 11

For people who have abstained from smoking after a standard course of varenicline in combination with behavioural support,clinicians may considera further course of varenicline to reduce relapse.

Recommendation 12

For people who are attempting to quit smoking using varenicline accompanied by behavioural support, clinicians might recommend the use of varenicline in combination with nicotine replacement therapy, compared with varenicline alone.
Conditional recommendation for intervention, moderatecertainty

Recommendation 13

Bupropion sustained release should be recommended
to people who smoke and who have been assessed as clinically suitable for this medication; it should be provided in combination with behavioural support. Bupropion is less effective than either varenicline or combination nicotine replacement therapy.
Strong recommendation, high certainty

Recommendation 14

Nortriptyline should be considered as a second-line smoking cessation pharmacotherapy agent because of its adverse effects profile.
Strong recommendation, moderate certainty

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PERIODONTITIS AND SMOKING & SMOKING CESSATION

Recommendation 15

Nicotine-containing e-cigarettes are not first-line treatments for smoking cessation. The strongest evidence base for efficacy and safety is for currently approved pharmacological therapies combined with behavioural support.

The lack of approved nicotine-containing e-cigarettes products creates an uncertain environment for patients and clinicians, as the constituents of the vapour produced have not been tested and standardised. However, for people who have tried to achieve smoking cessation with approved pharmacotherapies but failed, and who are still motivated to quit smoking and have broughtup e-cigarette usage with theirhealthcare practitioner, nicotine- containing e-cigarettes may be a reasonable intervention to recommend. This needs to be preceded by an evidence-informed shared decision-makingprocess, whereby the patient is aware of the following:

- no tested and approvede-cigarette products are available
- the long-term health effectsof vaping are unknown
- possession of nicotine-containing e-liquidwithout a prescription is illegal
- in orderto maximise possiblebenefit and minimiserisk of harms,only short-term use is recommended
- dual use (i.e. with continued tobaccosmoking) needs to be avoided. Conditional recommendation for intervention, low certainty

Behavioural and advice-based support for smoking cessation:

Recommendation 16

Referral to telephonecall-back counselling servicesshould be offered to all people who smoke.

Strong recommendation, high certainty

HEALTH BENEFITS OF QUITTING

20 minutes

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HEALTH BENEFITS OF QUITTING

20 minutes

- Blood pressure and pulse rate returns to normal.
- Temperature of hands and feet increases to normal.

8 hours

- Carbon monoxide and oxygen level in blood returns to normal.

24 hours

- Immediate risk of heart attack starts to fall.

14 days

- Circulation improves.
- Energy and fitness level improve.
- Lung function increases by up to 30%.

1 month

- Most nicotine withdrawal symptoms disappear.

3 months

- Cilia regrow in lungs, increase in their ability to handle mucus, clean themselves and reduce infection.

1 year

- Risk of coronary heart disease is half that of smoker.

10 years

- Risk of lung cancer is 30-50%that of continuing smoker.

15 years

- Stroke risk same as non- smoker.
- Risk of coronary heart diseasesame as a non-smoker.

DENTAL BENEFITS OF QUITTING

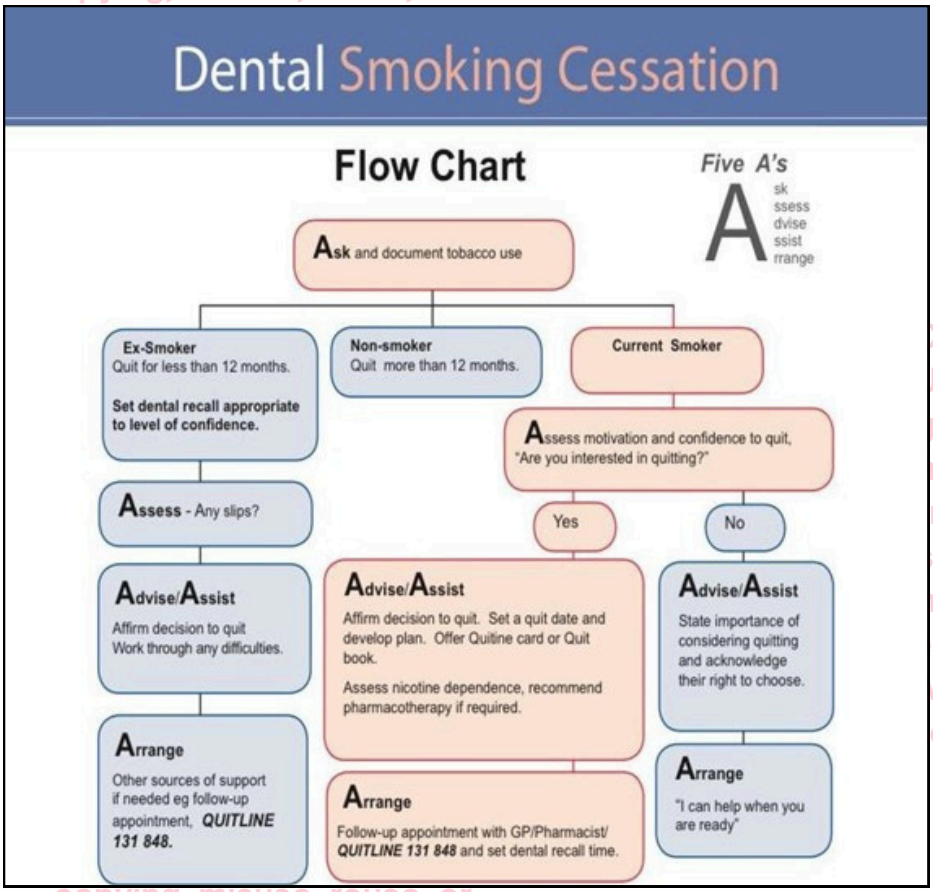
Initial and continuing benefits:

- Improved gingivaland oral tissue health.
- Improved taste sensation after 48 hours.
- Prevents bad breath.
- Minimises tooth staining.
- Smokers' palatedisappears shortly aftercessation of smoking.

PERIODONTITIS AND SMOKING & SMOKING CESSATION

Longer term benefits:

- Reduced risk of periodontal diseaseand tooth loss.
- Improved treatmentoutcomes for
  - Oral surgery
  - Periodontics
  - Implants
  - Prosthesis
  - Restorative and aesthetic dentistry
- Smokers' melanosis in heavy smokers reverses after a year and gingival colour returns to normal.
- Oral leukoplakia may regressor disappear following cessation.
- Diminished risk of mouth, throat and oesophagus cancer to half that of a smoker after 5 years.



PERIODONTITIS AND SMOKING & SMOKING CESSATION

SYMPTOMS OF QUITTING

Symptom	Effect on body	Coping strategy
Bleeding gums	Circulation improves, increased gingival bleeding may be evident.	Effective plaque removal.
Craving	Intense desire to smoke, declines over 4 weeks	Consider pharmacotherapy. Brief distractions eg: 4D's: drink water, deep breathe, do something else, delay urge to smoke. Ring the Quitline 131 848.
Coughing	Worse initially, body clearing respiratory tract.	Settles after first 2-3 weeks.
Hunger	Possibly intense, may persist.	Start regular exercise program. Eat sensibly, but no serious dieting until a less stressful period. Moderate alcohol consumption.
Bowel upsets	Possible constipation or diahorrea.	Settles over 2-3 weeks.
Sleep disturbances	Sleep patterns altered, insomnia or tiredness.	Settles over 2-4 weeks.
Dizziness	Caused by improved tissue oxygenation.	Passes spontaneously.
Mood alteration	Reflections of grief and (mainly) nicotine withdrawal on neuro transmitters.	Consider Pharmacotherapy. An old support system has been lost, find new ways to handle stress, eg: talk to a friend. Transient mood, returns to normal after 4 weeks.

PHARMACOTHERAPY FOR SMOKING CESSATION

SUITABLE ONLY FOR PATIENTS SMOKING 10 + CIGARETTES PER DAY

TYPES OF THERAPY	REFER TO
Nicotine sub-lingual tablet	Pharmacist
Nicotine lozenge	Pharmacist
Nicotine patch	Pharmacist
Nicotine gum	Pharmacist
Nicotine Inhaler	Pharmacist
Bupropion (Zyban)	General Practitioner

REFERENCE:
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