

Diagnosis and Examination

Periodontics is one of the focuses on the INBDE and so a strong foundation is essential to your success. In this set of notes, we will review all of the periodontics concepts tested on the INBDE, including diagnosis and examination, classifications, plaque and biofilm, pathogenesis, treatment planning & prognosis, non-surgical therapy, surgical therapy, adjunctive therapy, and prevention and maintenance.



1 Diagnosis

Periodontics is a branch of dentistry dealing with the periodontium. The **periodontium** includes alveolar bone, periodontal ligament, cementum, and gingiva,

Anatomical Terms

- **Periodontal Ligament (PDL)** – fibers between cementum and alveolar bone
- **Alveolar Bone** – bone that supports teeth
- **Gingival Sulcus** – potential space between tooth and gingiva
- **Periodontal Pocket** – pathologically deepened gingival sulcus
- **Gingival Margin** – peak of gingiva
- **Free Gingival Margin** – gingiva border
- **Free Gingival Groove** – shallow linear depression marking transition from free gingiva to attached gingiva
- **Attached Gingiva** – keratinized gingiva firmly attached to underlying bone
- **Alveolar Mucosa** – mucosa not bound to bone, non-keratinized
- **Muco-gingival Junction** – junction between attached gingiva and alveolar mucosa
- **Vestibular Fold** – transition fold between alveolar mucosa of periodontium and labial/buccal mucosa near the lip or cheek

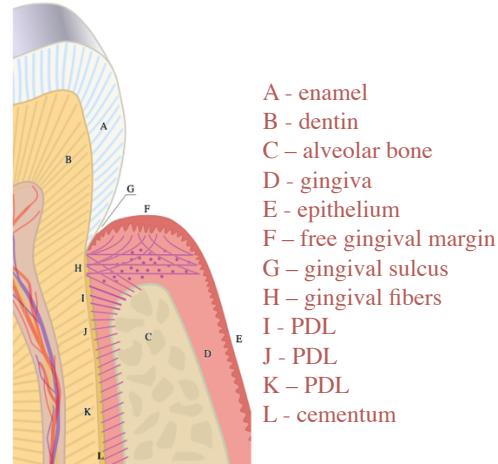


Figure 1.01 General dental anatomy



Figure 1.02 Anatomical terms of Gingiva

Periodontal Disease

Periodontal disease, also known as **periodontitis**, is a complex condition that plays on the interaction between bacteria and its host (the patient).

- **Microbial plaque** – the initiating factor of periodontitis, accumulation of bacteria on the tooth surface

There are 3 states of periodontal health

1. **Periodontal Health** – absence of inflammation, PDL destruction, and bone destruction
2. **Gingivitis** – presence of inflammation, absence of PDL destruction and bone destruction absent
3. **Periodontitis** – presence of inflammation, PDL destruction, and bone destruction

Pathogenesis of periodontal disease has a number of steps

1. Sub-gingival plaque bacteria present microbial challenge (LPS, antigens, other by-products)
2. Inflammatory response by up-regulated host immune system
 - Cytokines, prostaglandins, MMPs
 - Leads to gingivitis
3. Tissue destruction when microbial challenge is chronic or potent

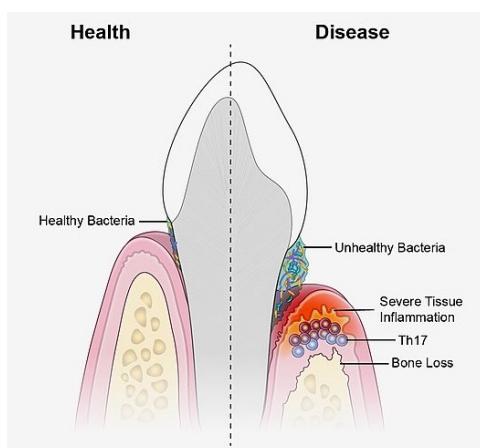


Figure 1.03 Gum recession

The above image depicts the body trying to protect itself by retreating its tissues from the plaque and bacteria

2 Examination

Tooth Examination

Teeth are examined for the following

1. **Erosion** – teeth eroded from acidic food/drink or stomach acid
2. **Abrasion** – mechanical wear (ex. aggressive tooth brushing) resulting in loss of tooth structure
3. **Abfraction** – flexural forces resulting in loss of tooth structure, usually in cervical areas
4. **Attrition** – occlusal wear from tooth-on-tooth contact
5. **Hypersensitivity** – exposed root surfaces

Periodontal Examination

The following are evaluated in the examination of the state of periodontal health

1. **Alveolar bone loss** – seen on radiograph but not as reliable
2. **Gingival recession** – shift of gingival margin apically to expose root from CEJ to gingival margin
3. **Tooth mobility** – indicates loss of periodontal support
4. **Suppuration** – sign of many neutrophils in periodontal pocket
5. **Furcation** – branching point of tooth roots; can be seen with enough periodontal bone loss

Periodontal Health can also be assessed with various measurements

1. **Probing Pocket Depth (PPD)** – gingival margin to base of pocket
2. **Clinical Attachment Loss (CAL)** – measures the position of the soft tissue to the CEJ (fixed point)
3. **Bleeding on Probing (BOP)** – used to measure inflammation of periodontal tissues
 - Seen when PPD or CAL is being taken

INBDE Pro Tip:

CAL = PPD + recession

- Rearrange this formula to calculate the unknown variable
- Recession can be a negative value (ex. when gingiva is swollen/high)

Furcation

A **Furcation** is branching point between tooth roots

- **Furcation involvement** – bone loss at the furcation area; can be achieved through ...
 - Shortened root trunk (CEJ to furcation) and roots
 - Narrow distance between roots (interradicular dimension)

Cervical enamel projection – enamel dips towards the furcation where there should be cementum; allows the periodontal apparatus to detach more easily.

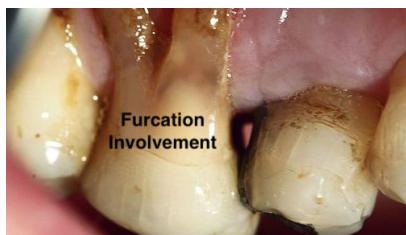


Figure 1.04 Cervical enamel projection

Classifications

1 Classifications

There are several methods of clinically classifying periodontal health:

Miller Classification (for Mobility)

- **Class 0** – normal tooth mobility, expect some give to the tooth due to the PDL
- **Class 1** – slightly more mobility than normal
- **Class 2** – moderate mobility ($\leq 1\text{mm}$)
- **Class 3** – severe mobility ($>1\text{mm}$), vertical mobility into tooth socket is possible

Hamp Classification (Furcation)

- **Class 0** – no furcation involvement
- **Class 1** – $<3\text{mm}$ horizontal furcation involvement
- **Class 2** – $>3\text{mm}$ horizontal furcation involvement
- **Class 3** – through-and-through furcation involvement (the probe can be it through)

Hamp classification uses a **Naber's Probe** for measuring.

- Each section represents 3mm
- Ability to dip probe into furcation indicates furcation involvement

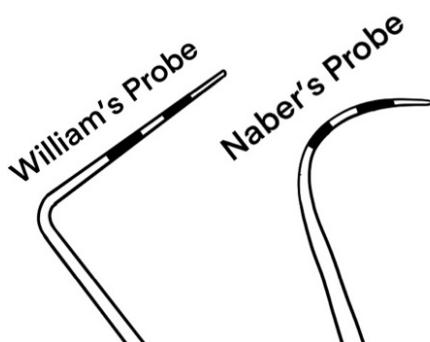


Figure 2.01 Probe types

Glickman Classification (Furcation)

- **Class 1** – pocket formation into the FLUTE, but not enough to expose furcation, incipient
- **Class 2** – pocket formation into the FURCA, also known as cul-de-sac furcation involvement
- **Class 3** – through-and-through furcation
- **Class 4** – through-and-through furcation, it displays a clear space that can be seen through

Alveolar Bone Loss

- The typical distance from the alveolar crest = 2mm
- The line connecting the alveolar crest should usually be parallel to line connecting CEJs
- Bone height is best measured with bitewing radiographs

Horizontal bone loss occurs when bone loss is still parallel to the line connecting CEJs

Vertical or angular occurs when bone loss is not parallel to CEJ line; and is classified by the number of bony walls remaining (infrabony defects).

- **1-wall** – hemiseptal shape wall
- **2-wall** – crater shape, most common
 - ▶ Loss of interseptal bone between two teeth, buccal and lingual walls remain
- **3-wall** – trough shape
- **4-wall** – bone remains around the circumference
 - ▶ Termed exclusively for extraction sockets

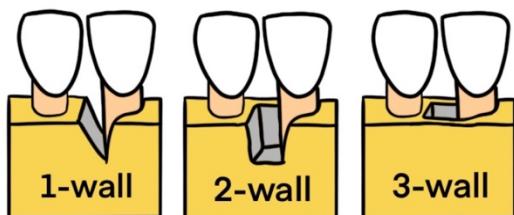


Figure 2.02 Vertical bone loss

INBDE Pro Tip: Terms for infrabony defects are very popular on the INBDE. Make sure you know them!

Miller Classification (Recession)

Miller classification indicates the probability of regaining root coverage through a connective tissue graft procedure. *Note that the Miller classification for mobility is a separate system.

- **Class I** – gingival recession does not reach mucogingival junction + loss of interdental bone or soft tissue not present
 - 100% likelihood of full coverage via graft since no interdental bone was lost
- **Class II** - gingival recession reaches or goes beyond mucogingival junction + loss of interdental bone or soft tissue not present
 - 100% likelihood of full coverage via graft since no interdental bone was lost
- **Class III** - gingival recession reaches or goes beyond mucogingival junction + loss of interdental bone or soft tissue/or root coverage prevented by tooth malpositioning
 - Partial likelihood of coverage via graft
- **Class IV** - gingival recession reaches or goes beyond mucogingival junction + loss of interdental bone or soft tissue/or root

3. **Consistency** – fibrosis if gingivitis is chronic

Plaque-Induced Gingival Disease

Plaque-Induced Gingival Disease is a common type of gingival disease that is caused by the interaction between plaque bacteria and host immune cells

Modified/influenced by

1. Medications
 - calcium channel blockers, dilantin and cyclosporine associated with drug induced **gingival enlargement**
 - oral contraceptives
2. Systemic factors
 - endocrine (diabetes, pregnancy)
3. Malnutrition

Non-Plaque Induced Gingival Disease

Non-Plaque Induced Gingival Disease is the less common type; and can be a response to ...

1. Allergies
 - restoration materials
 - food
2. Trauma
 - factitious
 - accidental
 - iatrogenic damage – damage from dentist
3. Infection
 - bacterial
 - viral
 - fungal
4. Hereditary Gingival fibromatosis – firm, non-hemorrhagic tissue

2 Gingivitis and Classifications

Gingivitis can be identified by the 3 Cs

1. **Color** – redder from increased blood flow
2. **Contour** – inflammatory exudate and edema

3 Periodontal Disease

Traditionally, periodontal disease is classified using 3 different terms: distribution, severity and type.

Distribution

Distribution describes how many sites are affected. Each tooth has 6 sites (disto-facial, mid-facial, mesio-facial, mesio-lingual, mid-lingual, disto-lingual). Therefore, each patient's total amount of sites is the number of total teeth x 6.

- 1. **Generalized** - ≥ 30% of sites
- 2. **Localized** - < 30% off sites

Severity

- 1. **Slight** – CAL = 1-2mm
- 2. **Moderate** – CAL = 3-4mm
- 3. **Severe** – CAL ≥ 5mm

Type

1. Chronic Periodontitis

- Slow, progressive bone destruction
- Destruction proportional to amount of microbial deposits (plaque)
- Patient is clinically unhealthy (ex. smoker, diabetic)
- Modified by systemic issues
- Tends to be older in age

2. Aggressive Periodontitis

- Fast bone destruction
- Destruction not proportional to amount of microbial deposits
- Patient is clinically healthy
- Molar/incisor pattern in local version where deeper pockets only seen in molars and/or incisors
- Familial aggregation
- Tends to be of younger age

3. Necrotizing

- Either ANUG or ANUP (acute necrotizing ulcerative gingivitis/periodontitis)
- Pseudomembrane, fever, fetid breath, blunted papillae
- Specific to individuals who smoke, have stress, smoking and immunosuppression are predisposed

Periodontitis can therefore be classified by a distribution-severity-type-disease, respectively.

- Ex. localized-moderate-chronic-periodontitis
- Ex. generalized-slight-aggressive-periodontitis

Epidemiology

The following are a few common facts on the INBDE:

- Most common to least common periodontitis: chronic > localized aggressive > general aggressive > refractory
- Most prevalent group to get periodontitis are Mexican Americans, non-Hispanic Black Americans, and those who regularly smoke.

4 New AAP Periodontal Classification

In 2017, the American Academy of Periodontology created a new periodontal classification system. The JCNDE recommends being familiar with **the old and new** classifications for the **INBDE** during this transition period.

The major change lies in the establishment of four main categories of disease processes.

1. Periodontal Health & Gingival Disease
2. Periodontitis
3. Peri-Implant Diseases and Conditions
4. Periodontal Manifestation of Systemic Diseases and Developmental and Acquired Conditions

After assessing the overall disease, its severity is determined by a process called **staging**. Lastly, the disease will be assessed for risk factors, therapeutic outcomes and rate of progression in a step called **grading**.

Periodontal Health & Gingival Disease

- Generally healthy, soft and hard tissue around teeth
- One or two sites may have some gingival inflammation (minimum BOP < 10% of sites and PD ≤ 3mm)
- **Intact Periodontium** (no attachment loss or bone loss)
- **Reduced Periodontium** (can have previous attachment loss or bone loss due to either: history of periodontitis however currently stable or no history of periodontitis if patient had for example a crown lengthening procedure)
 - ▶ Patient has minimal BOP and PD ≤3mm)

Gingivitis

- BOP, erythema, edema
 - ▶ Minimum BOP < 10% of sites
 - ▶ PD ≤ 3mm
- Periodontal stability (no progressive attachment or bone loss)

Periodontitis

- periodontal tissue is lost due to plaque which causes host-mediated inflammation
- often deeper PD
- **interproximal** attachment loss

Staging

Staging describes the **severity, complexity** and **extent** of the disease; and is measured from slight to severe in stages I through IV, respectively. It is a static measurement that measures the amount of destroyed or damaged tissue due to periodontitis. There can only be one stage per patient, so the most severe applicable option is used.

- **Severity** – usually based on worst interdental CAL site

1-2mm	Stage I
3-4mm	Stage II
≥5mm	Stage III or IV

- ▶ Radiographic bone loss (RBL) can also be considered

Coronal third	Stage I
Coronal third	Stage II
At/past middle third	Stage III or IV

- ▶ #of teeth lost from periodontitis can also be considered

No teeth lost	Stage I
No teeth lost	Stage II
≤4 teeth lost	Stage III
≥5 teeth lost	Stage IV

- Complexity** - based on PD

≤4mm + horizontal bone loss	Stage I
≤5mm + horizontal bone loss	Stage II
≥6mm + vertical bone loss + furcation	Stage III
≥6mm + occlusal trauma + bite collapse	Stage IV
Automatic Stage Placement	
Vertical bone loss ≥ 3mm	Stage III or IV
Furcation involvement class II or III	Stage III or IV
<20 teeth remaining	Stage IV

- Extent and Distribution** – based on teeth involved. This measure does not receive a stage, but a distribution pattern.

<30% of teeth involved	Localized
≥30% of teeth involved	Generalized
Molar/Incisor Pattern – molars and/or incisors are exclusively involved	Classic localized aggressive periodontitis

Grading

Grading is a measure based on changes overtime as well as risk factors and responsiveness to therapies. Each patient is given a grading from A to C based on several factors. Clinicians should initially assume grade B, then shift to A or C if applicable. The grading may change over time.

CAL or RBL	
No loss over 5 years	Grade A
<2mm loss over 5 years	Grade B
≥2mm loss over 5 years	Grade C
%RBL/Age	
<0.25	Grade A
0.25-1.0	Grade B
>1.0	Grade C
Phenotype	
Plaque exceeds bone loss	Grade A
Plaque consistent with bone loss	Grade B
Bone loss exceeds plaque	Grade C
Smoking	
Non-smoker	Grade A
<10 cigarettes/day	Grade B
≥10 cigarettes/day	Grade C
Diabetes	
No diabetes	Grade A
HbA1c < 7.0%	Grade B
HbA1c ≥ 7.0%	Grade C
C-Reactive Protein (marker of inflammation)	
<1 mg/L	Grade A
1-3 mg/L	Grade B
3 mg/L	Grade C

Peri-Implant Health

- No signs of inflammation or BOP around implants.
- Probing depths should be less than 5 mm

Peri-Implant Mucositis

- Visual signs of inflammation or BOP around implants.
- Probing depths greater than the baseline (compared to the previous year recording)
- No signs of progressive implant bone loss

Peri-Implantitis

- Visual signs of inflammation or BOP around implants.
- Probing depths greater than the baseline (compared to the previous year recording)
- Signs of progressive implant bone loss

Plaque and Biofilm

1 Plaque

As previously stated, the presence of microbial plaque is an indication of periodontal disease. However, dental plaque is not homogenous and has a complex composition depending on location and time.

Dental plaque is composed of the following:

1. Organic components
 - Polysaccharides, lipids, glycoproteins, proteins
2. Inorganic components
 - Phosphorus, sodium, calcium potassium fluoride
3. Supra-gingival Components
 - **Aerobic bacteria**
 - Gram positive on **early** layers near tooth
 - Gram negative on outer surface
 - Components from saliva
4. Sub-gingival Components
 - **Anaerobic bacteria**
 - Early layers near tooth are gram positive coronally and gram negative apically
 - Outer surface near epithelium are gram negative
 - Components from gingival crevicular fluid (GCF)

INBDE Pro Tip: Saliva and GCF influence the regions they are in proximity to. Hence, saliva influences supragingival components and GCF influences subgingival components.

Dental Plaque Formation

1. Pellicle Formation
 - Develops in seconds
 - **Pellicle** – proline-rich proteins, glycoproteins + other components that allow bacteria to attach
2. Bacterial Adhesion & Attachment
 - Occurs in minutes
 - Weak, reversible van der Waals and electrostatic forces are used for initial **adhesion**
 - Strong irreversible interaction between host pellicle receptors and adhesion molecules are used for firm attachment
 - **Primary (early) colonizers** mostly **streptococcus** and **actinomyces** bacteria
 - Feed on sugar and carbon from food eaten and saliva
3. Colonization and Plaque Maturation
 - Occurs within 1-2 days
 - Primary colonizers are strongly attached and allow other bacteria to attach by providing receptors via coadhesion
 - Facultative gram-positive switch to anaerobic gram-negative once the biofilm matures & bacteria grow
 - **Secondary (late) colonizers** like **P. Intermedia** and **T. Denticola** feed on amino acids more
 - **Fusobacterium nucleatum** binds primary to secondary colonizers; is a bridging micro-organism

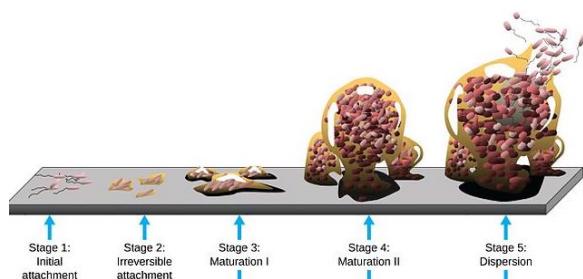


Figure 3.01 Plaque formation

Biofilm

Biofilm is an organized network of bacteria along with its associated nutrients, enzymes, by-products, oxygen etc.

- Consists of **fluid channels** that allow passage of nutrients through plaque to deeper bacteria
- **Quorum sensing** – bacteria communicate within biofilm to stimulate the growth of favorable species to inhibit that of unfavorable species
- Bacteria in the biofilm tend to be more resistant to antimicrobials compared to other bacteria such as free-swimming bacteria

2 Microbial Complexes

Bacteria can be arranged into groups or complexes based on their characteristics. In fact, different complexes correspond to different locations in plaque.

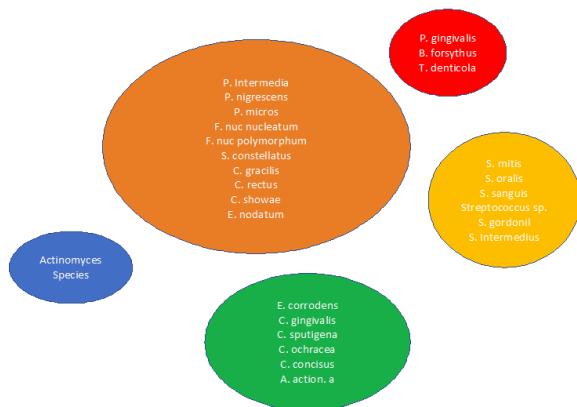


Figure 3.02 Microbial Complexes

- **Red complex** – linked to both BOP + deeper PD
 - ▶ *P. gingivalis*
 - ▶ *T. forsythia*
 - ▶ *T. denticola*

- **Orange complex** – precedes red complex in periodontal pocket
 - ▶ *Fusobacterium*
 - ▶ *C. rectus*
 - ▶ *P. intermedia*

Major Bacteria for INBDE

1. A. Actinomycetemcomitans (green complex)

- Gram-negative rod bacteria, immobile
- Capnophilic
- **Causes aggressive periodontitis**
- Contains lipopolysaccharide (LPS) which is an endotoxin located in outer membrane of all gram-negative bacteria
- Contains **leukotoxin** which kills leukocytes
- IgG cleaving protease
- Contains collagenase which breaks down collagen

2. P. Gingivalis (red complex)

- Gram-negative rod bacteria, immobile
- **Causes chronic periodontitis**
- Gingipain is a protease which cleaves host proteins
- Fimbriae is used for adherence
- Contains Capsule, Collagenase, Hemolysin

3. T. Denticola (red complex)

- Gram-negative spirochete bacteria, mobile
- **Causes ANUG/ANUP**
- Contain a protease that breakdown complement factors immunoglobulins & collagen
- Invades epithelium & connective tissue

4. T. Forsythia (red complex)

- Gram negative rod bacteria, immobile

5. P. Intermedia (orange complex)

- Gram-negative rod bacteria, immobile
- Causes **pregnancy gingivitis**

6. C. Rectus (orange complex)

- Gram-negative rod, mobile

7. F. Nucleatum (orange complex)

- Gram-negative rod bacteria, immobile
- Bridging micro-organism** (link between primary and secondary colonizers)

8. Other Bacteria

- S. salivarius** – most common oral bacteria & found in tongue
- S. mutans** – cause coronal caries
- Actinomyces** – found in healthy gingiva & cause root caries
- Pseudomonas & Staph** – cause peri-implantitis

Plaque Hypotheses

Over the course of history, different theories of plaque and disease have been hypothesized. Although the most accurate hypothesis is currently the ecological hypothesis, it is good to know all 3.

1. Nonspecific – amount of plaque correlates to disease severity (independent of which bacterial species is present)

2. Specific – disease is only caused by specific bacterial species

3. Ecological – certain bacteria in combination with specific host factors can shift the environment. This shift can favor pathogenic bacteria

3 **Local Factors**

In combination with the presence of plaque, local factors contribute to periodontal disease by promoting accumulation and retention of additional plaque bacteria.

1. Calculus

- Mineralized plaque
 - Takes 1-14 days for mineral salts to precipitate & form plaque
- Calculus does not irritate gingival tissues, but the layer of plaque on top of it is an irritant
- Supragingival calculus
 - White or yellow
 - Mineralized by saliva near salivary duct openings
- Subgingival calculus
 - Dark
 - Mineralized by GCF
- Identified by an explorer or visually
- Interproximal calculus can be visualized on radiographs



Figure 3.03 Calculus

2. Materia Alba

- White curd-like matter that is made up of unorganized bacterial accumulation, desquamated epithelial cells, food debris, salivary proteins



Figure 3.04 Microbial Complexes

3. Extrinsic Stains

Color	Association
Orange	Often associated with anterior teeth, Poor oral hygiene
Brown	Drinking dark beverages, Poor oral hygiene
Yellow-Brown	Usage of stannous fluoride or CHX
Dark brown/ Black	Tobacco use
Green & Yellow	Chromogenic bacteria cause this stain
Bluish-Green	frequent exposure of metallic dust
Black thin lines on cervical third	Found in healthy mouth and due to iron consumption

INBDE Pro Tip: Extrinsic tooth stains are mainly an esthetic concern and usually do not influence gingival inflammation. However, they are important to study as they are extremely popular on the INBDE.

4. Malocclusion

- Plaque retention by crowding
- Mesial drifting of teeth due to missing teeth can retain plaque and impact food

5. Faulty Restorations

- Overcontoured restorations can lead more plaque retention than undercontoured restorations
- Plaque retention from overhanging margins or rough surfaces

6. Sub-gingival Margins

- Associated with plaque accumulation and gingival inflammation
- Can be seen even when margins are ideal

7. Appliances

- Orthodontics
 - Can increase plaque retention
 - Can create excessive forces on periodontium

8. Oral jewelry

- Can lead to recession and bone loss

9. Removal Partial Dentures

- Increase abutment teeth mobility
- Increase plaque accumulation

10. Self-Inflicted Injury

- Excessive biting fingernails
 - Gingival tissue destruction
- Aggressively brushing horizontally

Pathogenesis

Immune cells and enzymes play a crucial role in the pathogenesis of periodontal disease. Ultimately, they are responsible for the destruction of periodontal tissue as a host response to the plaque bacteria. There are numerous cell types and proteins to consider.

1 Immune Cells

Neutrophils

- First immune cells to respond
- Control bacterial challenge & release chemicals that destroy periodontal tissue
- Phagocytize and kill bacteria via myeloperoxidase
- Migrate to periodontal pocket via chemotaxis to prevent invasion of periodontal bacteria
- MMP-8 (neutrophils collagenase)
 - Proteinase destroys periodontal tissue
 - Restricted by tetracycline
- Damaged neutrophil chemotaxis lead **aggressive periodontitis**
- Neutrophil activity too high and too low can lead to → tissue destruction

Macrophages

- Antigen presenting cells (APCs)
- Control immune response by releasing cytokines

Mast Cells

- IgE production
- Vascular permeability and dilation

Lymphocytes

- B-cells turn into plasma cells to produce antibodies
- T-helper (CD4) cells – coordinate immune response by activating other cells

- T-cytotoxic (CD8) cells – kill intracellular antigens
- NK cells – Like T cells, which detect & destroy cells infected by virus

2 Mediators

Pro-inflammatory - lead to tissue destruction

- IL-1 – mediate bone resorption
- IL-6
- **MMPs – break down collagen** (primary mediator in periodontal tissue destruction)

Anti-inflammatory - oppose tissue destruction

- IL-4
- IL-10
- TIMPs

3 Pathogenesis of Gingivitis

Pathogenesis of Gingivitis

1. Initial Lesion (2–4 days)
 - GCF increase
 - Neutrophil penetration
2. Early Lesion (4–7 days)
 - Increase collagen loss
 - T-lymphocyte penetration
 - BOP
3. Established Lesion (14–21 days)
 - B-lymphocytes penetration + plasma cells
 - Collagen loss, consistency change, contour change, and clinical color change
 - Gingivitis becomes more established at this stage
4. Advanced Lesion
 - Irreversible stage - the establishment of periodontal bone loss

Treatment Planning & Prognosis

1 Treatment Planning

Goals

Short-Term Goals:

- Decrease gingival inflammation by fixing the causes
- More focus on comfort and esthetics

Long-Term Goals:

- Stop hard and soft tissue destruction
- Stop pain
- Stopping recurrence
- Prevent tooth loss, but cannot always save all teeth
- Achieve occlusal function and stability
- More focus on function and health

Phases of Periodontal Treatment

Phase 0 – Preliminary Phase

- Prior to starting treatment
- Emergency purposes
- Extract **hopeless teeth**
 - ▶ Bone loss involving apex of tooth
 - ▶ Hardly any periodontal attachment that cannot be restored

Phase 1 – Non-Surgical Phase

- Plaque control, caries control, diet control and patient education
- Scaling & root planing, oral hygiene instruction, prophylaxis
 - ▶ Remove local factors
- Antibiotics (local or systemic)
- Fix restorative irritation
- Periodontal re-evaluation (PD, inflammation) 4-8 weeks after phase I therapy
 - ▶ Healing and formation of junctional epithelium

Phase 2 – Surgical Phase

- When non-surgical phases are unsuccessful
- Treat with endodontics
- Remove periodontal pockets and regenerate periodontal ligaments, fix soft and hard tissue defects
- Periodontal therapy such as implants

Phase 3 – Restorative Phase

- The stage at which periodontal disease is controlled
- Final restoration, fixed or removable prosthodontics

Phase 4 – Maintenance Phase

- Also known as:
 - ▶ Supportive periodontal therapy
 - ▶ Periodic ongoing evaluation of OHI and condition of periodontal tissue
- Continued along with phases 2 & 3 every 3 months during the first year

Risk Elements

Risk Factors – commonly result in disease:

- Pathogenic bacteria
- Diabetes
- Smoking
- Microbial tooth deposits

Risk Determinants – background

characteristics that cannot be changed & increase the possibility of disease

- Age – older = longer time exposed to etiological factors
- Genetic factors – periodontitis has high inheritability (severe chronic periodontitis result due to IL-1 gene polymorphism)
- Socioeconomic status – higher rate of smoking, less dental visits & awareness
- Gender – male CAL > female CAL

Risk Indicators – not commonly result in disease, but could potentially put one at a higher risk:

- HIV/AIDS – the immunocompromised often seen with ABUG/ANUP
- Stress – mental stress can inhibit the immune system
- Osteoporosis – decreased bone mass may progress of periodontal disease
- Low dental visits

Risk Marker or Predictors – disease that is associated quantitatively (previous history)

- CAL – most important indicator
- BOP
- History of periodontal disease

2 Prognosis

Prognosis is the anticipation of the probable course of a disease. In dentistry, the entire mouth needs to be looked at before prognosis of individual teeth can be done.

Clinical Factors

- Plaque control – poor oral hygiene instruction
- Disease severity – CAL > PD
- Vertical bone loss – more favorable prognosis than horizontal bone loss, since it has the potential to be treated with regenerative therapy
- Age – younger patients have more time and opportunity for the disease to get worse
- Low patient compliance

Systemic Factors

- Diabetes
 - ▶ Worse prognosis if poorly controlled and not controlled
- Smoking
 - ▶ increases periodontal disease frequency and severity
 - ▶ Reduced healing ability to nonsurgical and surgical therapies
- Parkinson's Disease
 - ▶ More difficult to perform oral hygiene (ex. reduced motor ability making it harder to brush)

Anatomic Factors

- Cervical enamel projections
- Tooth mobility – poorer response to therapy
- Root factors
 - ▶ Root proximity
 - ▶ Short, tapered roots
 - ▶ Root concavities
- Furcation involvement – more difficult to clean
- Bifurcation ridges

Prosthetic and Restorations

These factors are indirectly related to periodontal disease and can influence the prognosis of teeth:

- Non-vital teeth
- Root resorption
- Abutment selection
- Caries

3 Prognosis Classification

There are 6 categories of prognosis classification. A category could be assigned to the whole mouth or an arch.

	Bone Level	Clinical Factors	Local Factors	Systemic Factors	Cooperation
Excellent	No bone loss	None	Gingival health	No	Good
Good	Sufficient alveolar support	None	Can be maintained	No	Good
Fair	Insufficient alveolar support	Mobility, furcation I	Can be maintained	Limited	Adequate
Poor	Moderate bone loss	Mobility, furcation I or II	Difficult areas to maintain	Yes	Questionable
Questionable	Advanced bone loss	Mobility, furcation II or III	Inaccessible areas	Yes	Inadequate
Hopeless	Advance bone loss	Extraction is recommended	Cannot maintain	Uncontrolled	Inadequate

Non-Surgical Therapy

1 Scaling and Root Planing

Scaling and root planing aim to remove the etiologic factors of plaque and calculus in order to restore gingival health.

Scaling – removes supra-gingival and sub-gingival plaque and calculus

Root planing – removes residual embedded calculus and rough cementum

Scaler Types

1. Sickle Scalers

- Remove **supragingival** calculus
- triangle cross-section
- two cutting edges – either edge can be used to remove and scrape off calculus

2. Curettes

- Remove **subgingival** calculus
- rounded edge avoids trauma to gingival tissue
- **Universal curettes** – any area of the mouth
 - two cutting edges, semicircle cross-section
- **Gracey curettes** – specific areas of the mouth
 - One cutting edge, semicircle cross section
 - Gracey 1-2 & 3-4 → anterior
 - Gracey 5-6 → anterior + premolars
 - Gracey 7-8 & 9-10 → posterior, facial/lingual
 - Gracey 11-12 → mesial, posterior
 - Gracey 13-14 → distal, posterior

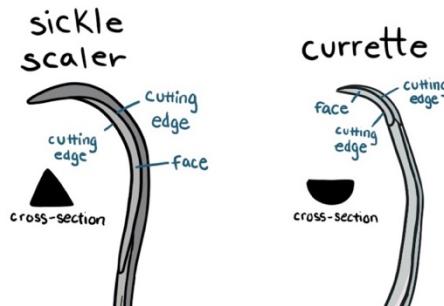


Figure 6.01 Scaler types

3. Ultrasonic Scalers

- i. Remove stubborn calculus
- ii. Contraindicated for patients with:
 - a. Pacemakers
 - b. Risk for respiratory disease
 - c. Infectious disease that can be transmitted via aerosols
- iii. Several functions to clean pocket
 - a. Acoustic turbulence creates agitation that can breakdown bacterial cell walls
 - b. Flush out with water
 - c. Vibration mechanically removes deposits
 - d. Cavitation
- iv. **Piezoelectric ultrasonics**
 - a. vibrate in linear pattern
- v. **Magnetostrictive ultrasonics (Cavitron)**
 - a. Vibrate in elliptical pattern

Strokes

1. Exploratory

- Light stroke for probes and explorers to detect CAL or pocket depth

2. Ultrasonics

- Light intermittent strokes
- Tip instrument parallel to tooth

3. Root planing

- Light to moderate pulling stroke used for smoothing

4. Scaling

- Stronger and shorter pulling stroke used to remove hard deposits

Curette Angulation

Curette angulation is specific and changes when you scale or root plane a tooth. The angle referenced is between the tooth surface and face of the instrument.

- 0 degrees (closed-angle)** – during initial insertion of curette into pocket
- 45-90 degrees (open-angle)** – when scaling and root planing the tooth

Essentially, you are facing the instrument parallel to the tooth surface when inserting, then angling when you start scaling.

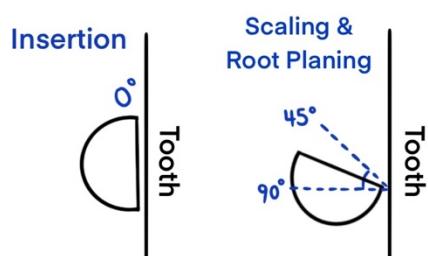


Figure 6.02 Curette Angulation

2 Prophy Cup and Brush

Prophylaxis paste

- Grittier than toothpaste
- Used to remove extrinsic stain

Prophy Cup

- Flexes on light pressure
- Helps remove extrinsic stains and pocket access

Prophy Brush

- Enables greater access to occlusal grooves and interproximal spaces

Prophy Jet

- Ejects water and sodium bicarbonate
- Removes soft deposits and extrinsic stains

Surgical Therapy

Surgical therapy is considered when non-surgical therapies do not resolve the issue. The flap technique is often used during surgery. After surgery, post-operative plaque control is essential.

1 Flap Incisions

Flap Design

There is a certain shape that soft tissues should be cut in order to access defective periodontal tissue safely.

The following rules should be adhered to:

- Wider base apically to allow blood flow down to coronal part of flap (narrow base would sever vasculature that feeds more coronal tissue)
- Avoid vital structures
- Incisions over intact bone
 - Avoid bony defect and eminences
- Round corners
- Flap margins contact tooth at line angle

Papilla Preservation

Papilla Preservation flap

- Incision that preserves the papilla
- Edges of flap contact line angles of tooth

Conventional flap

- Incision that splits the papilla
- Could result in loss of papilla and black triangle

3 options

- Lingual → facial line angle
- Lingual → lingual line angle
- Facial → facial line angle

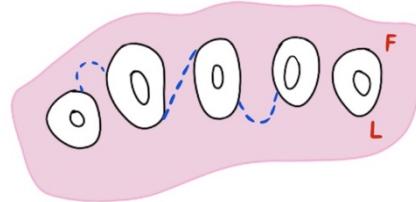


Figure 7.01 Papilla preservation

Flap Thickness

Split/Partial thickness (mucosal) flap

Includes gingiva/mucosa + submucosa

- **Mucogingival surgery**
 - No need to expose bone

Full thickness (mucoperiosteal) flap

Includes mucosa + submucosa + gingiva + periosteum

- Periodontal regeneration & osseous surgery
 - Allows primary closure and apically repositioned flaps
- Alveolar bone exposure normally lead 1mm bone resorption & remodeling

Involves 3 horizontal incisions:

1. Internal/reverse bevel

- ~1mm from gingival margin
- opens pocket lining while preserving outer gingiva

2. Sulcular/crevicular

- From base of pocket to alveolar crest

2. Interdental/interproximal

- Removes collar of tissue around the tooth that was made during the previous two incisions

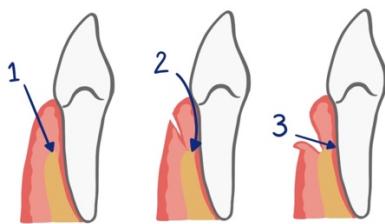


Figure 7.02 Horizontal incisions

Flap Types

- **Modified Widman flap**
 - ▶ Allows sub-gingival access for debridement so that new attachment can form
 - ▶ Uses the three horizontal incisions

- **Apically repositioned flap**
 - ▶ Includes vertical releasing incision beyond mucogingival junction to reduce periodontal pocket via moving gingival margin apically

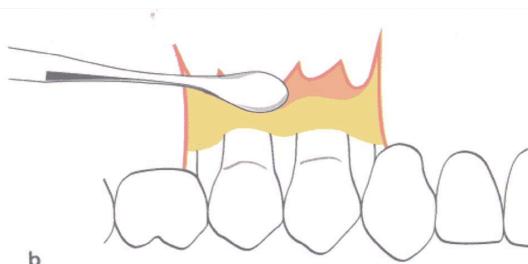


Figure 7.03 Repositioned flap

Periodontal Pack

- Used after invasive flap surgery
- Protects the surgical wound, maintains tissue placement, prevents bleeding, reduces discomfort
- Do not accelerate the healing
- Left in the mouth for 1 week

2 Bone Graft Materials

Some surgeries include bone grafts as part of the treatment. We need to review a few terms describing these bone graft materials before learning about specific surgeries.

Graft materials should carry out at least some of the following principles:

- **Osteoconductive** – scaffold (all bone grafts are osteoconductive)
- **Osteoinductive** – turns close by progenitor cells into osteoblasts
- **Osteogenic** – creates bone

There are 4 main categories of graft materials. All grafts are osteoconductive.

The type of graft is listed from most ideal to least ideal graft material:

- **Autograft** – from the patient
 - ▶ Osteoconductive, osteoinductive and osteogenic
- **Allograft** – from someone else, usually cadaver
 - ▶ Osteoconductive and osteoinductive
- **Xenograft** – from an animal, usually cow
- **Alloplast** - synthetic or inorganic

3 Categories of Surgery

Gingival Surgery

- Excision only involves gingiva and soft tissue above the mucogingival junction
- healing by secondary intention (open wound, walls do not suture together)

There are two types of gingival surgery:

- **Gingivectomy**
 - ▶ To remove supra-bony pocket or gingival enlargements (more aggressive therapy)
- **Gingivoplasty**
 - ▶ To reshape tissue deformities (less aggressive, addresses esthetic concerns)

Distal Wedge Surgery

- Used for pocket reduction distal to the terminal molars
- Specific type of gingival surgery
- Mandibular** – full thickness flap + V-shape incisions
- Maxillary** – full thickness flap + parallel incisions

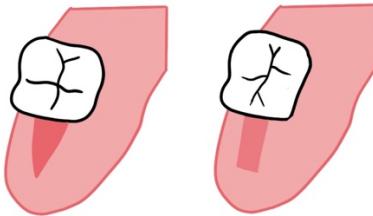


Figure 7.04 Distal wedge surgery

Connective tissue graft (CTG)

- Placing a piece of connective tissue to suture over the **exposed root surface**
- Donor site needs to have enough attached gingiva
 - Palate - most common donor site
- Harvested tissues is inner connective tissue only (not epithelium)
- Occurs coronal to gingival margin

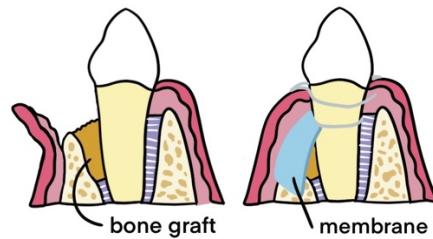


Figure 7.05 CT graft

Mucogingival Surgery

Mucogingival surgery involves operating on the gingiva and mucosa. There are several types

- Free gingival graft (FGG)**
 - Increase the amount of keratinized tissue **apical to gingival margin**
 - Attached gingiva enhances plaque removal and minimizes inflammation around abutment teeth and implants
 - Achieving 2mm of attached gingiva is recommended
 - Donor site usually from palate
 - Graft thickness ideally 1-1.5mm
 - Free graft = transplant without a nourishing blood supply
 - Thus, need to expose vascular bed at recipient site to supply newly grafted tissue

Vestibuloplasty

- Deepens the vestibule
- Frenotomy**
 - Incision of the frenum
- Frenectomy**
 - Removing the entire frenum

Osseous Surgery

This type of surgery deals with the bone, thus a specific assessment of the periodontal bony architecture is performed for the detection of bony defects and their severity.

Flat architecture

- Interproximal and radicular bone heights are the same

Positive architecture

- Interproximal bone is coronal to radicular bone
- ideal alveolar bone morphology

Negative architecture

- Interproximal bone is apical to radicular bone

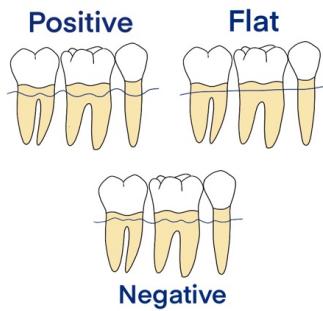


Figure 7.06 Osseous surgery

Osseous surgery has the aim of achieving positive architecture.

- **Osteotomy** – removes supporting bone
- **Osteotomy** – removes non-supporting bone
- **Clinical crown lengthening** – lowers bone (osteotomy) to expose more tooth
 - ▶ Combines with gingivectomy (when at least 2 mm keratinized tissue after gingivectomy) or with apically positioned flap

Periodontal Regeneration

- Guided Tissue Regeneration (GTR)
 - ▶ Regenerates PDL, bone & cementum

3 components

- **Barrier membrane** – placed over defect and allows hard tissue growth and prevents soft tissue from growing in
- **Bone graft** – osteoinductive, osteoconductive and /or osteogenic
- **Biologic agent** – promotes healing environment for tissue formation

INBDE Pro Tip: The various periodontal surgeries can be summarized into 2 methods.
Additive & Subtractive

- Periodontal regeneration (additive)
- CTG (additive)
- FGG (additive)
- Coronally advanced flap (additive)
- Gingivectomy (subtractive)
- Apically positioned flap (subtractive)
- Resective osseous surgery (subtractive)

4 Healing Process

Mechanisms of Healing

- **Regeneration**
 - ▶ Comeback to normal formation and function
- **Repair**
 - ▶ Not fully recovers formation and function
 - ▶ Healing via scar formation or formation of long junctional epithelium
- **Reattachment**
 - ▶ Epithelial and connective tissue reconnect with root surface after incision or injury
- **New Attachment**
 - ▶ Inserting new PDL fibers into new cementum in areas with insufficient attachment

Wound Healing Cells

From fastest to slowest, the following cells begin to populate the wound area:

1. Epithelial cells
2. Connective Tissue cells
3. PDL cells
4. Bone cells

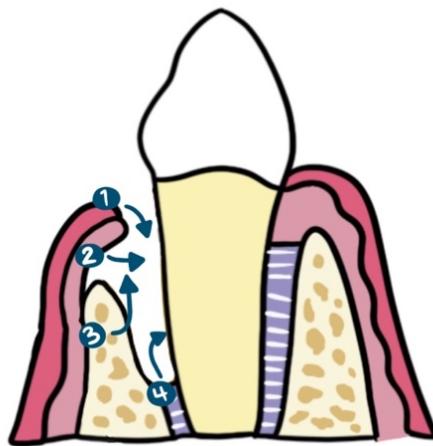


Figure 7.07 Wound healing cells

Root Surface Treatment

- Chelating agents like EDTA can be used to stimulate new attachment by exposing the collagen fibrils through demineralization.

Defects and Treatments

- 1 and 2-wall defects
 - **Resection** (ostectomy)
- 3 and 4-wall defects
 - **Regeneration**

Adjunctive Therapy

Adjunctive therapy is used alongside surgical or non-surgical therapies to help aid in the healing process of periodontal disease.

1 Antibiotics

Antibiotics are used to target bacteria in the periodontal pocket that are the initiating factor of plaque.

- Only use as an adjunct to phase I non-surgical therapy during mechanical debridement
- Most often used for localized aggressive periodontitis
- Do not take bactericidal (killing) and bacteriostatic (stopping growth) drugs at the same time
- Used for refractory periodontitis (disease not resolved despite undergoing treatment)

The following are a few antibiotics to take note of:

Tetracyclines

- In GCF
 - GCF is found inside each pocket, thereby targeting pocket bacteria
- Doxycycline can help with patient compliance
 - One dose per day

Amoxicillin (AMX) + Metronidazole (MTZ)

- Most common and effective antibiotic routine for periodontal disease
- AMX (500mg TID) + MTZ (200mg TID) for 14 days
- Then length of duration is more beneficial than the dose
- Do not mix MTZ and alcohol

Local Delivery Antibiotics (LDA)

- Used when conventional therapies still result in localized recurrent and/or residual PD $\geq 5\text{mm}$ + inflammation

Common Name	Antibiotic
Arrestin	Minocycline
Atridox	Doxycycline
PerioChip	Chlorhexidine gluconate

2 Host Modulation Therapy

As you know by now, tissue destroyed from periodontal disease comes from the host response to bacterial challenge. Thus, host modulation therapy aims to suppress the destructive nature of the host immune response

- Use for chronic periodontitis (not aggressive)
- Only use as an adjunct to phase I mechanical debridement

NSAIDs

- Inhibits prostaglandins = inhibits inflammation

Bisphosphonates

- Inhibit osteoclasts = inhibits bone destruction
- Side effect - Bisphosphonate related oral necrosis of the jaw (BRONJ)

Subantimicrobial Dose Doxycycline (SDD)

- Inhibits MMPs (collagenases) that destroy the PDL
- Used systemically and locally
- 20mg, twice/day for 3-9 months
 - Subantimicrobial dose – less than normal dose if one had a bacterial infection
- Only one from this list approved by FDA + accepted by ADA
- Should be an adjunct to removing plaque

3 Occlusal Correction

Sometimes periodontal disease stems from malocclusion. **Traumatic occlusion** can create occlusal forces that exceed the reparative ability of attachment apparatus. For instance, heavy contact or interference could injure the PDL. Occlusal indicating wax or articulating paper can be used to check for such occlusion.

Occlusal trauma can be divided into 3 categories

- **Primary** – too much pressure on normal periodontium
- **Secondary** – normal tooth-on-tooth contact but reduced periodontium
- **Fremitus** – teeth vibrate when closing

Occlusal therapies should be used as treatment only after inflammation has resolved

- **Interocclusal appliance (biteguard)**
 - Redistributions occlusal forces more evenly
- **Occlusal adjustment (coronoplasty)**
 - Reshapes occlusal surface
- **Splinting**
 - Immobilizes overly mobile teeth
 - Increases comfort and function

4 Furcation Correction

Furcation involvement is very difficult to clean sufficiently, often leading to periodontitis. Currently, there are 4 different treatment options for furcation involvement.

Furcation Plasty

- Opening furcation further up + smoothing so that there is more access for cleaning

Tunneling

- Removing bone and moving soft tissue apically to reveal more furcation for easier cleaning access

Root Amputation/Resection

- Cutting off and removing one root + smoothing → endodontic treatment → place crown that leaves open space for cleaning
- Commonly seen in removing distobuccal roots of maxillary first molar

Hemisection/Premolarization

- Cut molar in half → creates 2 premolar-like teeth

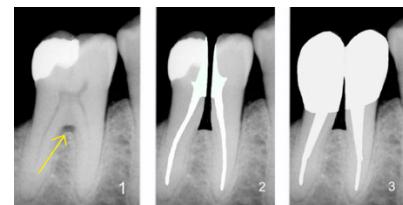


Figure 8.01 Furcation correction (1)

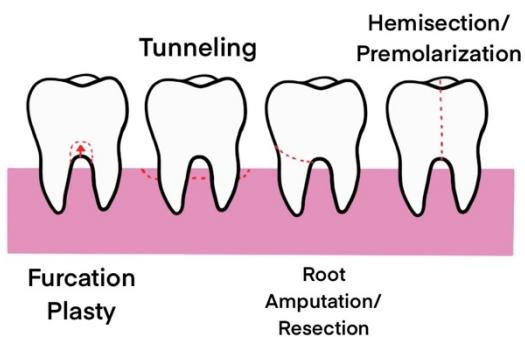


Figure 8.02 Furcation Correction (2)

INBDE Pro Tip: There is only one prognosis based periodontal question on the INBDE. If you only choose to remember one thing, know that CAL is the most important factor in determining prognosis

Prevention and Maintenance

1 Toothbrushing

Bass Method

- Regarded as best method of brushing amongst examiners
- Place bristles at gingival margins at 45 degrees to the tooth sulcus
- Extend slightly subgingival in order to breakup plaque in the cervical area

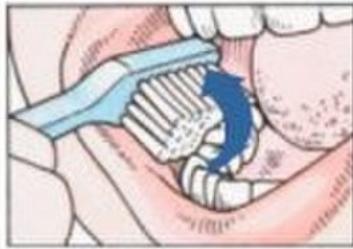


Figure 9.01 Toothbrushing method



Figure 9.02 Flossing method

3 Waterpik

Waterpik

- Made to flush out food debris with water
- Lowers bacterial load on gingiva
- Cannot remove biofilm on the tooth surface = does not prevent periodontal disease



Figure 9.03 Waterpik

2 Flossing

Flossing

- Wrap floss around middle fingers, hold between thumbs and index fingers
- Important to curve floss into C-shape against the surface of tooth
- Gently rub floss up and down tooth surface on each side
- Remember to floss the distal surface of the posterior-most teeth