Mental Dental Periodontics

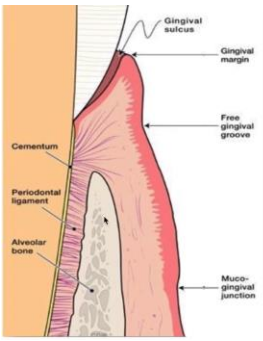
Diagnosis & Periodontal Exam

Periodontium

= Tissues that surround and support the tooth

Involves:
- Alveolar bone
- PDL
- Cementum
- Gingiva

Healthy

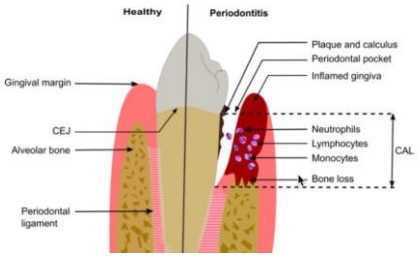


Gingival Sulcus	Natural space between tooth and gingiva. Becomes a perio pocket when it become pathologically deep
Gingival Margin	Peak height of the gingiva
Free gingival groove	Shallow depression on the surface that demarcates the "Free gingiva" from the "Attached Gingiva"
Mucogingival Junction	Junction between attached gingiva and alveolar mucosa
Free Gingiva	Not bound or attached to underlying bone
Attached gingiva	Bound to underlying bone
Alveolar Mucosa	Not attached either
Keratinized Tissue	Free Gingiva + Attached Gingiva
Non-Keratinized Tissue	Alveolar Mucosa

Periodontal Disease

Microbial Plaque is the initiating factor

- ***Periodontal health*** = no inflammation + no PDL or bone destructions
- ***Gingivitis*** = Inflammation + no PDL or bone destruction
- ***Periodontitis*** = Inflammation + PDL and Bone destruction (CAL, Clinical Attachment Loss)



Pathogenesis (simplified)

**All about the interplay between bacteria and the host

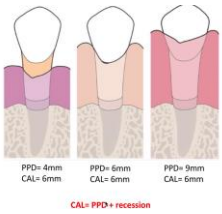
1. Microbial challenge (LPS, antigens etc) presented by subgingival plaque bacteria
2. Upregulation of host immune-inflammatory response (Cytokines, prostaglandins, MMPs)
3. Tissue Destruction if the inflammation persists

Tooth Exam

Erosion = Caused by acidic foods/drinks/gastric acid
Abrasion = Loss of tooth structure by mechanical wear (usually toothbrush)
Attrition = Occlusal wear from functional contacts w/ opposing arch (Bruxism)
Abfraction = Loss of tooth structure in cervical area due to tooth flexure
Hypersensitivity = result of exposure of dentinal tubules in root surfaces

Periodontal Exam

- *Probing Pocket Depth (PPD)*: Distance from gingival margin to the base of the pocket
- *Clinical Attachment Loss (CAL)*: Measured from CEJ to the base of the pocket
  - (PD + Recession, or PD – Overgrowth)
- *Bleeding on Probing (BOP)*: Best measure of inflammation in periodontal tissues
- *Gingival Recession*: From CEJ to gingival margin. Exposure of root surface due to apical shift of gingival margin
- *Alveolar Bone Loss*: Radiographic measure.
- *Suppuration:* Indicates large number of neutrophils in the pocket
- *Mobility:* Due to loss of periodontal support, traumatic occlusion or a combo
- *Furcation Involvement*: Bone loss at the branching point of a tooth root



Oral Exam

Home Care: Plaque and calculus measure. Techniques and frequency of brushing and flossing
Inflammation: Redness, Swelling, BOP
Destruction of Periodontal Tissues: PPD, CAL, Alveolar bone loss, mobility, furcation involvement

Classification Systems

Mobility

Miller Classification	
Class 0	Normal physiologic mobility
Class I	Slightly more that normal
Class II	Moderately more than normal (≤ 1mm B-L)
Class III	Severely more than normal (>1mm B-L) and can be vertically depressed in the socket

Furcation

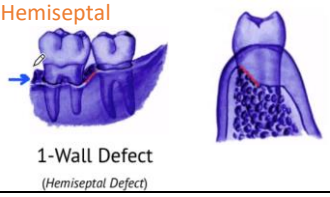
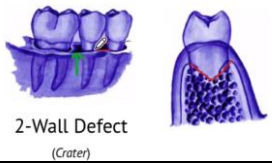
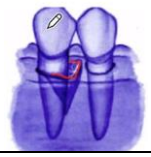
Factors that predispose furcation involvements:

- Short root trunk
- Short roots
- Narrow interradicular distance
- Cervical Enamel Projections

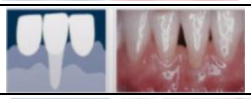
Hamp Classification	
Class 0	No furcation involvement
Class I	Horizontal involvement <3mm
Class II	Horizontal involvement >3mm (but not all the way through)
Class III	Through-and-through involvement
Glickman Classification	
Class 1	Pocket formation into the FLUTE area
Class 2	Pocket formation into the FURCA (cul-de-sac)
Class 3	Through-and-through furcation lesion
Class 4	Through-and-through furcation lesion that you can SEE through

Alveolar Bone Loss

- Always best measured with BW rads
- Normal distance from CEJ to alveolar crest = 2mm
- Crest should be parallel to a line connecting the CEJ of adjacent teeth
    - Horizontal Bone loss -> Stays parallel to this line
    - Vertical Bone Loss -> Classified by the number of bony walls remaining

Infrabony Defects (Vertical bone loss)	
1 Wall	Hemiseptal 
2 Wall	Crater (Most common) 
3 Wall (Intrabony)	Trough 
4 Wall	Circumferential defect -> Extraction socket

Recession

Miller Classification	
Class 1	= Recession does NOT extend to the mucogingival junction - No loss of interdental bone of soft tissue *Tx Prognosis*: 100% coverage 
Class 2	= Recession to or beyond the mucogingival junction - No loss of interdental bone or soft tissue *Tx Prognosis*: 100% root coverage 
Class 3	= Recession extending to or beyond the mucogingival junction - Interprox. Bone or soft tissue loss present *Tx Prognosis*: Partial root coverage 
Class 4	= Recession at or beyond the mucogingival junction - Severe Interprox. Bone or soft tissue loss *Tx Prognosis*: No root coverage 

Gingivitis (1999 Classification)

3 C's:

- *Colour*: ↑ blood flow causes redness
- *Contour*: Inflammatory exudate and edema
- *Consistency*: Chronic gingivitis leads to fibrosis (lack of stippling)

Plaque-Induced Gingival Diseases - Most common - Result of interaction between plaque bacteria and inflammatory host cells	
Modified by Systemic Factors	- Endocrine changes (puberty, pregnancy, diabetes) - Blood dyscrasias (Leukemia)
Modified by Medications	Drug Induced Gingival Enlargement - Nifedipine (CCBs) - Phenytoin (Dilantin) - Cyclosporine - Oral contraceptives
Modified by Malnutrition	- Vitamin C deficiency (Scurvy)
Non-Plaque-Induced Gingival Diseases - Less common	
In response to Infection	- Bacterial Infections (Neisseria gonorrhoeae, Treponema pallidum) - Viral Infections (Herpes Zoster) - Fungal Infections (Candidiasis)
In response to Allergy	- Food - Restorative materials - Toothpastes (SLS)
In response to Trauma	- Factitious (Unintentionally produces) - Iatrogenic (Caused by a doctor) - Accidental (through burns from hot foods and drinks)
Genetics	Hereditary Gingival Fibromatosis - Non-hemorrhagic and firm

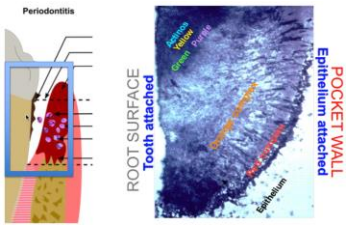
Periodontal Disease (1999 Classification)

Severity	
Slight	1-2mm CAL
Moderate	3-4mm CAL
Severe	≥ 5mm CAL
Distribution	
Localized	<30% of all sites
Generalized	≥ 30% of all sites
Type	
Chronic	- Pt is clinical not healthy usually - Slower, progressive bone destruction - Microbial deposits are consistent w/ extend of destruction - Modified by systemic issues (Diabetes and Smoking are major ones) - Pts tend to be older

Aggressive	- Clinically Health - Rapid bone destruction - Familial aggregation - Microbial deposits are not consistent w/ extend of destruction - Localized version has 1st molar/incisor presentation
Necrotizing	Acute Necrotizing Ulcerative Gingivitis (ANUG) and Acute Necrotizing Ulcerative Periodontitis (ANUP) - **Predisposing factors**: Stress, Smoking, Immunosuppression **Key Characteristics:** - Pseudomembrane/Necrotic tissue - Fetid Breath - Blunted Papilla - Fever

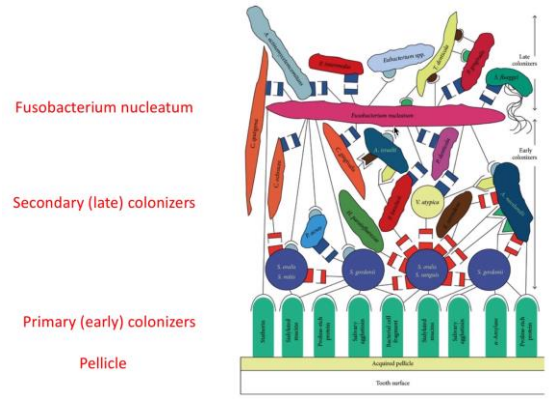
Plaque

- As plaque grows out from the tooth surface towards the epithelium the composition changes



Plaque Composition	
Supragingival	*Aerobic Microbes* - Tooth layer (*early* layers): Gram +'ve aerobes - Outer surface of plaque (more *mature* plaque): Gram –'ve aerobes
Subgingival	*Anaerobic* - *Tooth surface*: Gram +'ve coronal, Gram –'ve apical - *Epithelium*: Gram –'ve
Constituents	*Suprag. Plaque gets these constituents from the Saliva, but Subg plaque gets them from the Gingival Crevicular Fluid* *Organic*: - Polysaccharides - Proteins - Glycoproteins - Lipids *Inorganic*: - Calcium - Phosphorus - Sodium - Potassium Fluoride

Dental Plaque Formation	
1. Pellicle Formation	Forms within Seconds - Consists of glycoproteins, proline-rich proteins and other molecules that serve at attachment sites for bacteria
2. Adhesion + Attachment of Bacteria	Occurs within Minutes - Initial adhesion is from ***weak reversible*** van der Waals and electrostatic forces - Firm attachment is due to ***strong irreversible*** interactions between specific bacterial adhesin molecules and host pellicle receptors
3. Colonization and Plaque Maturation	Occurs within 24-48 hours - Firmly attached primary colonizers provide new receptors for attachment of other bacteria -> Co-adhesion - As bacteria grow and the biofilm matures: Shifts from Facultative Gram +'ve to Anaerobic Gram –'ve microbes



Pellicle: Provides attachment sites for microbial adhesion

Primary (early) Colonizers: Mostly Streptococci and Actinomyces

- Feed off of carbs and sugars

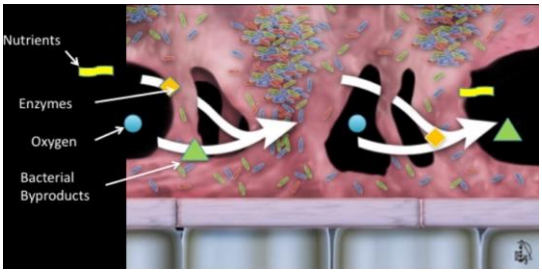
Secondary (late) colonizers: P. Intermedia, T Denticola etc

- Feed off of amino acids

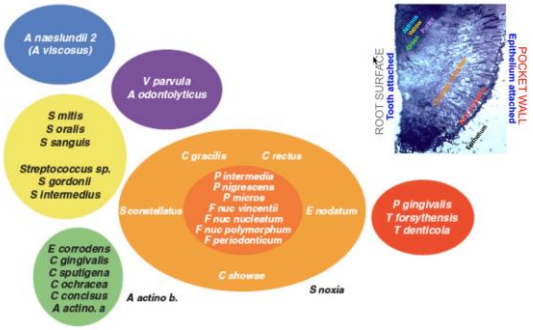
Fusobacterium nucleatum: Bridging Microbe. Can coaggregate 1º and 2º colonizers

Biofilm

- *Fluid Channels* -> Run through the plaque mass and permits the passage of nutrients and flush out waste
- *Quorum Sensing* -> Communication among bacteria in a biofilm to encourage growth of beneficial species and discourage the growth of competing species
- Biofilm bacteria are more resistant to antimicrobials than free swimming bacteria



Microbial Complexes



Red Complex	Associated with BOP and Deep Pockets - P. gingivalis - T. denticola - T. forsythia
Orange Complex	Precedes the Red complex and supports sequential nature of plaque maturation - Fusobacterium - P. intermedia - C. rectus

Agrebacter Actinomycetemcomitans (A.A)	= Causes Aggressive Periodontitis - Nonmotile Gram –'ve rod - Grows well in CO_2 environments (Capnophilic) - Releases Leukotoxin -> kills human neutrophils, monocytes, and lymphocytes - LPS Endotoxin -> a component of all Gram –'ve outer membranes - Collagenase - Proteases that cleave IgG
P. Gingivalis (Red complex)	= Causes Chronic Periodontitis - Nonmotile, gram –'ve rod - Fimbriae -> important in adherence - Capsule - Gingipain -> a protease that cleaves host proteins - Collagenase - Hemolysin
T. Denticola (Red Complex)	= Causes ANUG/ANUP - Motile, gram –'ve spirochete - Penetrates the epithelium and connective tissue - Protease that can degrade collagen, immunoglobulins and complement factors
T. Forsythia (Red Complex)	= A little boring compared to the rest - Nonmotile, Gram –'ve rod - Protease that cleaves IG and complement factors
P. Intermedia (Orange Complex)	= Causes Pregnancy Gingivitis - Nonmotile, Gram –'ve rod - Becomes darkly pigmented when grown on blood agar plates

C. Rectus (Orange Complex)	= Boring, but a funny name - Motile gram –'ve rod - Polar Flagellum
Fusobacterium Nucleatum (Orange Complex)	= The glue! Bridge microbe that co-aggregates everyone - Nonmotile gram –'ve - Induces apoptosis of leukocytes and release of tissue-damaging substances from leukocytes
Other Bacteria to know	
Actinomyces	Found in normal flora -> but is associated with Root Caries
S. Mutans	Coronal Caries
S. Salivarius	Most common oral bacteria -> Found mostly on the tongue
Psuedomonas and Staph.	Implant surfaces

Local Factors

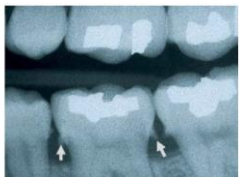
Calculus

= Mineralized dental plaque
- Precipitation of mineral salts into plaque -> within 1-4 days
- Doesn't serve as a mechanical irritant to tissue -> BUT is it covered in plaque which does irritate

Supragingival:
- White/Yellow.
- Mineralized from Saliva
- Occurs near salivary gland duct openings -> Lingual of mandibular anterior teeth is a common spot

Subgingival:
- Dark colour
- Mineralized via GCF

Detection:
- Visual detection (even easier when air dried)
- Subg. with an 11/12 Perio explorer
- Interproximal contacts radiographically



Materia Alba

= Soft white cheeselike material
- Unorganized accumulation of bacteria, salivary proteins, desquamated epithelial cells and occasional food debris

Easily removed with water spray

Extrinsic Stains

Don't actually contribute to Perio or Gingivitis, and is mostly an esthetic issue

Colours:

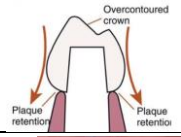
Orange	Usually anterior teeth, related to poor OHE
Brown	Drinking dark beverages (coffee) + Poor OHE
Dark brown/black	Tobacco
Yellow-Brown	CHX + Stannous Fluoride
Black	Thin lines on cervical 3rd in healthy mouths comes from Iron consumptions
Green-Yellow	Usually anterior teeth, from poor OHE + Chromogenic bacteria
Blue-green	Occupational exposure of metallic dust

Malocclusion
- Crowding can contribute to plaque retentive areas
- Prominent roots and high frenum attachment -> Causes gingival recession
- Mesial drift or extrusion associate w/ missing teeth can lead to food impaction and plaque retention

Fault Restorations

= Overhanging margins, open margins, rough surfaces, open contacts

Overcontoured restorations are much worse for gingival health than undercontoured



Subgingival Margins
- Even when perfect, they are associated w/ plaque accumulation, gingival inflammation and deep pockets

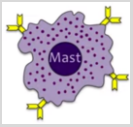
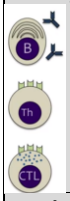
Appliances
- RPD may ↑ mobility of abutments and ↑ plaque accumulation
- Ortho therapy ↑ plaque retention and can create excessive force on the periodontium
  - Perio health must be established before ortho Tx
- Oral jewelry can result in recession, pocket formation and bone loss

Self-Inflicted Injury

= Aggressive horizontal brushing -> Recession, tooth abrasion
- Improper use of toothpicks, fingernail biting, stiff bristled brushes

Pathogenesis

Immune Cells

Neutrophils (PMN's) 	= First line defense - ***Most important cells*** involved in controlling the bacterial challenge…and in destroying the periodontal tissue with release of destructive molecules 1. Migrate from subepithelial vascular plexus into the periodontal pocket via chemotaxis -> forms a barrier to protect the body from the invading periodontal bacteria 2. Internalize bacteria via phagocytosis and kill them w/ myeloperoxidase and oxygen radicals *__***MMP-8 (neutrophil collagenase)***__ = most important proteinase involved in the destruction of periodontal tissues* - Inhibited by tetracycline antibiotics __Neutrophil abnormalities:__ - Defensive neutrophil chemotaxis = Aggressive periodontitis - Lose-Lose: ↑ neutrophil activity = self inflicted tissue destruction, but ↓ neutrophil activity = unchecked microbial challenge and tissue destruction - Ex: Neutropenia, Chediak-Higashi syndrome, Papillon-Lefevre syndrome, LAD-1, LAD-2
Macrophages 	= Antigen presenting cells (APC), like monocytes and dendritic cells - Regulates immune response via cytokine release (IL-8)
Mast Cells 	= Causes vascular permeability and dilation of blood cells - Produce IgE
Lymphocytes 	__B-cells:__ - Become plasma cells to make antibodies __T-Cells:__ - CH4 T_{helper}: Help in communication - CD8 $T_{Cytotoxic\ killer}$: Kill intracellular antigens - T_{NK} (natural killer): Recognize and kill tumor and virally infected cells
Proinflammatory mediators	IL-1: Bone resorption IL-6 PGE2 TNF α = Macrophage activation MMPs (Matrix Metalloproteinase) = Collagen destruction
Anti-inflammatory Mediators	IL-4 IL-10 TIMPs

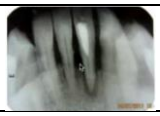
Pathogenesis of Gingivitis

Stage 1 Initial Lesion	__Timeline__: 2-4 days - Neutrophil infiltration - ↑ GCF
Stage 2 Early Lesion	__Timeline__: 4-7 days - T-Lymphocyte infiltration - ↑ collagen loss - BOP
Stage 3 Established Lesion	__Timeline__: 14-21 days - B-cell infiltration (plasma cells) - Collagen loss - Clinical change in color, contour, consistency
Stage 4 Advanced Lesion	= Transition to irreversible damage of periodontitis

Treatment Planning

Short Term Goals	↓ Gingival Inflammation - Correct the etiologic factors
Long Term Goals	- Eliminate Pain - Arrest hard and soft tissue destruction (CAL) - Establish occlusal stability and function - ↓ tooth loss (Sometimes some teeth are hopeless though) - Prevent disease recurrence

5 Phases of Perio Tx Planning

0 – Preliminary Phase	- Tx of emergencies - Extract hopeless teeth 
1 – Non-surgical Phase	=Plaque control and Pt education - Diet control - Caries control *Tx*: - Prophylaxis, SRP, OHI to remove local factors - Correct restorative irritational factors (overhang etc) - Local or systemic Antibiotics *Periodontal re-eval* - 4-8 weeks after completion of Phase I therapies to allow for healing and formation of Junctional Epithelium
2 – Surgical Phase	= Reduce or eliminate Perio pockets, Correct Soft and hard tissue defects, Regenerate perio tissues, Place Implants - ***Only needed if Phase 1 was unsuccessful*** *Tx*: - Periodontal Therapy, Placement of Implants - Endodontic Therapy
3 – Restorative Phase	****Not until Perio is fully under control**** *Tx*: - Final Restorations - Fixed and removable prostheses
4- Maintenance Phase	= Supportive Periodontal therapy - Periodic ongoing evaluation of OHI and perio tissue health *Tx*: - Maintenance in continuum with Phase II and III q3m for the 1st year

Risk Elements

Risk Factors	= Causally associated w/ disease - Smoking - Diabetes - Pathogenic Bacteria - Microbial tooth deposits
Risk Determinant	= Unchangeable background characteristics that ↑ chance of disease - *Genetics*: Polymorphisms in IL-1 genes may contribute to severe chronic perio - *Age*: Due to prolonged exposure to etiologic factors - *Gender*: Males have more CAL than females - *Socioeconomic Status*: ↓ dental awareness, ↓ frequency of dental visits, ↑ smoking
Risk Indicator	= Not causally associated w/ disease - *HIV/AIDS*: ANUG/ANUP ↑ in immunocompromised people - *Osteoporosis*: ↓ bone mass may have impact on progression of perio - *Infrequent dental visits* - *Stress*: Emotional stress interferes w/ normal immunologic function
Risk Marker/Predictor	= Quantitative association w/ disease - Previous Hx - BOP - CAL

Prognosis

= Prediction of the outcome of the disease. Prognosis of individual teeth must be considered in the context of the entire dentition

CAL is the most important factor in determining the prognosis

Clinical Factors	*Age*: - Younger patient w/ same level of disease as an older pt has ↓ prognosis *Severity*: - CAL is more important than PPD *Plaque Control:* - Poor OHE ↓ prognosis *Patient compliance:* - Non-compliant and uncooperative ↓ prognosis *Type of bone loss:* - Vertical bone loss has ↑ prognosis because it can potentially be treated w/ regenerative therapy (3-wall defect is best)
Systemic Factors	*Smoking*: - Smokers have ↑ prevalence and severity of perio and ↓ healing response to both non-surgical and surgical *Diabetes*: - Poorly controlled diabetes has ↓ prognosis than well-controlled diabetes *Parkinson's Disease:* - Compromises the patients ability to perform adequate OHE
Local Factors	- Plaque and Calculus - Subgingival restorations - Inadequate restorations
Anatomic Factors	- Short Tapered roots - Cervical enamel projections - Enamel pearls - Bifurcation ridges - Root concavities (Mesial of Maxillary 1st Premolar) - Developmental grooves - Close root proximity - Furcation involvement -> More difficult to clean - Tooth mobility -> ↓ response to therapy
Prosthetic and Restorative Factors	- Abutment selection - Caries - Non-vital teeth - Root Resorption

	BONE LEVEL	CLINICAL FACTORS	LOCAL FACTORS	SYSTEMIC FACTORS	PATIENT COOPERATION
EXCELLENT	No bone loss	None	Gingival health	No	Good
GOOD	Adequate alveolar support	None	Potential to maintain	No	Good
FAIR	Inadequate alveolar support	Mobility, furcation I	Potential to maintain	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	Advanced bone loss	Extraction indicated	Unable to maintain	Uncontrolled	Inadequate

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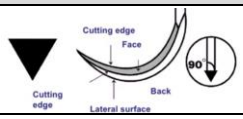
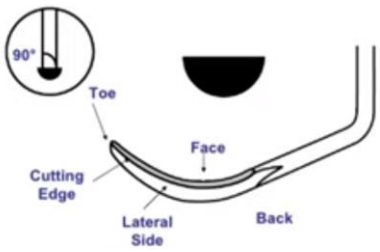
Non-Surgical Periodontal Therapy

Scaling and Root Planing	
Scaling = removal of both supragingival and subgingival plaque and calculus Root Planing = removal of embedded calculus and rough cementum	
Sickle Scaler	**Only used for supragingival calculus -> sharp tip can lacerate gingiva** - Has 2 cutting edges - Triangular cross section 

Curette

Used for subgingival calculus -> Rounded tip



Universal curettes:
- can be used in any area of the mouth
- Two cutting edges
- Semicircle cross section

Gracey Curettes:
- Adapt for specific areas of the mouth
- Only 1 cutting edge
- Semicircle in cross Section

Gracey 1-2 and 3-4	Anterior Teeth
Gracey 5-6	Anterior and Premolars
Gracey 7-8 and 9-10	Posterior (Facial and lingual)
Gracey 11-12, 15-16	Posterior Mesial
Gracey 13-14	Posterior Distal

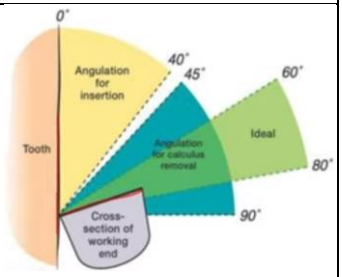
Ultrasonic Scalers

Used for Tenacious Calculus
- Contraindicated for patient w/ pacemakers, infectious diseases spread by aerosols and at the risk for resp. disease

Magnetostrictive Ultrasonics (Cavitron)	Vibrates in an elliptical pattern
Piezoelectric Ultrasonics	Vibrates in a linear pattern

Functions:
- Lavage (Flush with water)
- Cavitation (Vacuum air bubbles collapse and release energy to flush debris)
- Vibration (Mechanically remove deposits and debris)
- Acoustic turbulence (Agitation observed in fluids by mechanical vibrations that disrupts bacterial cell walls)

Strokes



Exploratory
- Light feeling stroke used w/ probes and explorers

Scaling
- Short, strong pull stroke to remove hard debris (60^0-80^0)

Root Planing
- Light-Moderate pull stroke used for final smoothing

Ultrasonics
- Light intermittent strokes w/ tip parallel to the tooth surface and in constant motion

**When initially inserting a curette into a pocket the angle between the blade and the tooth should be 0 degrees (Closed angle). When scaling the angulation though be 45-90^0 (Open angle)

Prophy

- Smooths and polishes the tooth surface to prevent plaque attachment and remove stains

Cups and Brush

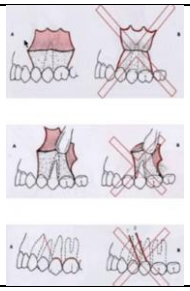
Cup:
- Flexes on slight pressure to the contours of teeth to help extrinsic stain removal and pocket access

Brush:
- Enables better access to select occlusal grooves and interproximal areas

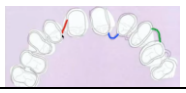
Prophy Jet

= Delivers slurry of water + Sodium Bicarbonate to remove extrinsic stains and soft deposits

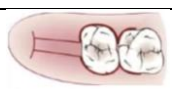
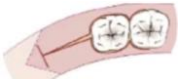
Surgical Therapy and Flap Design

Flap Design	Base of the flap should be wider than the top of the flap - Ensures adequate blood supple Incisions should be made over intact bone - Avoid bony defects of prominences Rounded corners Vertical releases at the *line angles*! Avoid vital structures *Post-op plaque control is the most important procedure after perio surgery* 
Flap Thickness	*Split/Partial thickness (Mucosal) Flap* - Gingiva/mucosa + Submucosa - Exposed for mucogingival surgery b/c exposing the bone is unnecessary *Full Thickness (Mucoperiosteal) Flap* - Gingiva/mucosa + Submucosa + Periosteum - Used for osseous surgery and periodontal regeneration to allow 1° closure and apically repositioned flaps ***Whenever alveolar bone is exposed (full thickness flaps), expect 1mm of bone resorption***  

Full Thickness Flap - 3 Incisions	
Internal/Reverse Bevel	= 1mm from the gingival margin. - Removes pocket lining, but preserves the outer gingiva 
Sulcular/Crevicular	Through the base of the pocket to the alveolar crest 
Interdental/Interproximal	Removes the collar of tissue around the tooth that you would have created with the first 2 incisions 
Modified Widman Flap	= Combination of all 3 of the above incisions - Provides access to the subgingival area for debridement w/ the goal of new attachment
Apically Repositioned Flap	= Requires an additional vertical releasing incision made beyond the mucogingival junction in order to attain pocket reduction

Papilla Preservation	*Conventional old school flaps* = Splits the papilla in the middle - Leads to papillary recession *Papilla Preservation Flap* = Preserves the Papilla. 3 Different options 
Periodontal Pack	= Consists of ZOE - Leave it in place for 1 week *Function*: - Protect surgical wound - Minimize discomfort - Maintain tissue placement - Help prevent post-op bleeding - DO NOT enhance healing **Contains peanut proteins -> Avoid in patients with peanut allergy**

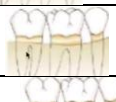
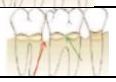
Gingival Surgery

Gingivectomy	= Excision of gingiva to eliminate supra-bony pockets or gingival enlargements - More aggressive, to remove tissues
Gingivoplasty	Excision of gingiva to reshape tissue deformities - More for esthetics
Distal Wedge	= For pocket reduction distal to terminal molars  *Maxillary:* - Full thickness flap w/ parallel incisions  *Mandibular*: - Full thickness flap with V-shaped incisions

Mucogingival Surgery

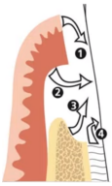
Free Gingival Graft	= Widens band of KT - ↑ KT to surround implants, crowns, and teeth. Stronger and more resistant tissue type. - KT helps prevent accumulation of plaque - Happens below the gingival margin No minimum width of attached gingiva is established -> 2mm is considered adequate though - Ideal thickness of graft is 1-1.5mm "Free" grafts = transplanted without a nourishing blood supply so it must undergo re-vascularization from the recipient bed
Connective Tissue Graft	= ↑ Root Coverage - CT usually comes from the **palate** and is sutured over an exposed root surface - Happens above (coronal) to the gingival margin Inner CT harvested only and not the epithelium -> ↓ pain during healing - Donor sites should always have enough attached gingiva
Frenectomy	= Complete removal of frenum
Frenotomy	= Incision of frenum
Vestibuloplasty	= Deepens the vestibule - To ↑ Support for dentures

Osseous Surgery

Bony Architecture	
Positive Architecture	Interproximal bone is coronal to radicular bone - Normal and ideal morphology 
Flat Architecture	Interproximal and radicular bone are the same height 
Negative Architecture	Interproximal bone is apical to radicular bone 

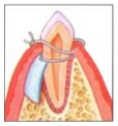
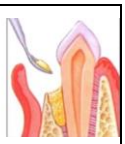
Ostectomy	= Removal of supporting Bone - More aggressive, bone is directly supporting the tooth (contact with PDL)
Osteoplasty	= Removal of non-supporting bone - Reduction of bone away from the tooth (no contact with tooth or PDL) Widow's peaks of bone may remain after ostectomy -> Remove these and ensure everything is smoothed out to prevent issues

Periodontal Regeneration



From Fastest -> Slowest cells that repopulate a wound:

- Epithelial Cells -> CT Cells -> PDL Cells -> Bone Cells

Guided Tissue Regeneration (GTR)	Regenerate Bone, Cementum and PDL   <u>*3 B's*</u> - Barrier membrane is the "Tank" -> Prevents ST downgrowth and permits hard tissue ingrowth - Bone Graft is the "Damage" -> Osteoconductive, Osteoinductive and or Osteogenic that does the work to make new bone - Biologic Agent is the "Healer" -> Creates an environment conductive to tissue formation
Root Surface Treatment	Chelating Agents (EDTA and Citric Acid) -> Expose collagen through demineralization and can improve new attachment

Bone Grafting Materials	
Autograft	= From Yourself - Osteoconductive, Inductive, and -Genic
Allograft	= From another human (usually cadaver) - Osteoconductive and inductive (not genic though)
Xenograft	= From another animal (usually a cow) - Osteoconductive
Alloplast	= Synthetic material - Osteoconductive
Osteoconductive	= Physical scaffold for bone to fill in
Osteoinductive	= Converts nearby progenitor cells into osteoblasts
Osteogenic	= Graft itself makes bone

Regeneration	= Completely restoring architecture and function
Repair	= Not completely restoring architecture and function - Involves healing by scar or formation or Long JE
Re-attachment	= Reunion of epithelial and CT w/ root surface after incision or injury
New Attachment	= Embedding of new PDL fibers into new cementum that has been previous deprived of its original attachment - As in Regenerative surgery

Issues and their Treatments

1 and 2 walled Defects	Osseous Resection -> Recontour the bone to restore positive architecture
3 and 4 walled defects	Regeneration -> Better blood supply and cell source is nearby - Deep, Narrow 3 walled Defect is ideal for regen
Hamp Class II	Regeneration within the furcation defect
Miller Class I (Thick phenotype and wide KT)	Regeneration for recession defects - CTG

Adjunctive Therapy

Antibiotics

Goal: ↓ # of bacteria in the perio pocket
- Only used as an adjunct to mechanical debridement during Phase 1
- **Do not give Bactericidal and Bacteriostatic drugs should NOT be administered at the same time**

Indications:
- Aggressive Perio
- Refractory Perio

Tetracyclines	Concentrates in the GCF -> Pretty good feature as this is where the bacteria are bathing in the sulcus **Doxycycline**: One 1 dose per day is needed, good for Pt compliance
Amoxicillin + Metronidazole	**This combo is the best combination drug therapy for combatting Perio** - Duration is more important than dose *Rx:* - Amoxicillin (500mg TID) + Metronidazole (250mg TID) for 14 days **Avoid Alcohol with Metronidazole**
Local Delivery Antibiotics (LDA)	= When localized recurrent and/or residual PD ≥ 5mm + inflammation that persist after conventional therapy Arrest**in** = Mi**n**ocycline Atri**dox** = **Dox**ycycline Perio**Ch**ip = **Ch**lorhexidine gluconate

Host Modulation Therapy

= Down regulate the destructive aspects of the host response
- Should be used as an adjunct during mechanical debridement during phase I

Perio Etiology is biofilm plaque -> but the cause of the damage is the body immune system…so this is what HMT is focused on

Indications:
- Chronic Perio

Systemic Modalities	
NSAIDS	Inhibit Prostaglandins - Long term use has side effects (↑ Bleeding, GI Ulcers etc) **Not really used**
Bisphosphonates	Inhibit Osteoclasts - Consider that you are putting the Pt at risk of BRONJ! **Not really used**
Subantimicrobial Dose Doxycycline (SDD)	**less than the dose prescribed to battle microbes** - 20mg 2x daily for 3-9 months (Periostat) = Inhibits MMPs (Collagenases) to ↓ the periodontal destruction 😊 *Only HMT therapy that is actually approved by the FDA and ADA
Local Modalities	
Emdogain	= Enamel Matrix Proteins - Amelogenins
PDGF	= GEM 21S - Growth Factor

Occlusal Correction

= Sometimes the root of the issue is malocclusion

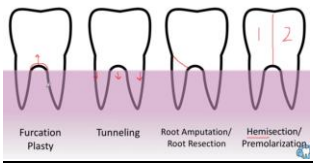
Traumatic Occlusion = Injury to the periodontium resulting from occlusal forces that exceed the reparative capacity of the attachment apparatus
- Widened PDL is the initial radiographic sign

Fremitus = Vibration of teeth upon closing

Primary Occlusal Trauma	Excessive forces on a normal periodontium
Secondary Occlusal Trauma	Normal forces on a reduced periodontium

Tx: -> Delay these treatments until inflammation is under control
- Coronoplasty = Selective reshaping of occlusal surfaces
- Interocclusal appliance (Bite guard) = Redistributes occlusal forces to minimize excessive force on individual teeth

Furcation Correction



Furcation Plasty	Reshape and move the furcation slightly more coronally to make OH easier
Tunneling	Remove bone and tissues apically to make the furcation a Glickman Class IV (Through-and-through) to make OH easier
Root Amputation/Root Resection	= Remove a root completely (providing the other roots can provide adequate support). Have to do endo of course as well - Commonly the DB root of a Maxillary 1st molar. This is the smaller root, and Palatal + MB can give adequate support. Otherwise it is really hard to clean a furcation in a 3 rooted tooth
Hemisection/Premolarization	= Endo a molar and then cut it in half (crowning both halves to form smaller teeth that you can floss in between)

Prevention and Maintenance

- Re-eval: 4-8 weeks after Phase 1 non-surgical therapy
- Maintenance = Every 3 months for the 1st year…then after the year can move up to 6 months etc based on patient health

Tooth Brushing	
Obviously - Soft brushes - Replace brush every 3 – 6 months	
Bass Method	 = Sulcular brushing - Bristles placed at gingival margin at 45° to the tooth -> This allows them to extend about 0.5mm into the sulcus. Flick down to disrupt plaque buildup in the cervical area - Prevents recession and horizontal brushing tendencies
Flossing	
Technique	= C-shape against the side of the tooth - Rub the floss gently up and down along the side of each tooth - Floss behind your last tooth also -  
Waterpik	
What is it?	= Home irrigation system. - Designed to flush out food debris and ↓ bacterial load **on the gingiva…not on the tooth surface** - Doesn't actually stop Periodontal Disease…because this comes from plaque on teeth 

Epidemiology and Facts

- Chronic > Localized Aggressive Perio > Generalized Aggressive Perio > Refractory Perio
- Most prevalent in males of African descent (This is also the most common for Diabetes)

New Classification System (2017)

4 Main categories:

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

Periodontal Health and Gingival Diseases	
Periodontal Health	= 1 or 2 sites with some evidence of clinical gingival inflammation - Minimal BOP (<10% of sites) - PD ≤ 3mm May be found with: - Intact Periodontium (no attachment or bone loss) - Reduced Periodontium (Previous attachment and or bone loss) - No Perio Hx (from abrasion or CL surgery) - Perio Hx but currently stable with no inflammation
Gingivitis	= Erythema, Edema, BOP - BOP (≥ 10% of sites) - PD ≤ 3mm Stable Periodontium (no progressive attachment/bone loss)

Periodontitis

= Loss of periodontal tissue support due to microbially associated, host mediated inflammation

- Typically associated with deeper PD and the presence of **Interproximal** CAL

Staging

= Severity and extent of disease at presentation

- Primarily based on Interdental CAL at the ***Worst site*** in the mouth

Staging				
	Stage I	**Stage II**	**Stage III**	**Stage IV**
Interdental CAL or RBL (at worst site)	1-2mm < 15% RBL	3-4mm 15-33% RBL	≥ 5mm >33% RBL	≥ 5mm >33% RBL
Complexity Modifiers				
Probing Depth (Complexity, can upgrade stage)	≤ 4mm Horizontal BL	≤ 5mm Horizontal BL	≥ 6mm Vertical loss Furcation Involvement	≥ 6mm Occlusal Trauma Bite Collapse
Special Factors (Auto boosts stage)			Vertical BL ≥ 3mm Furcation Class II/III	Vertical BL ≥ 3mm Furcation Class II/III < 20 teeth remaining
Extent/Distribution				
Localized <30% of teeth are involved (<10 teeth if Pt has wisdoms – 32 total) (<8 teeth if Pt has no wisdoms = 28 teeth)		**Generalized** ≥ 30% of teeth involved		**Molar/Incisor Pattern** Classic Localized Aggressive Perio

	Stage I	Stage II	Stage III	Stage IV
Interdental CAL	1-2mm	3-4mm	≥5mm	≥5mm
RBL	coronal third (<15%)	coronal third (15-33%)	extending to/past middle third (>33%)	extending to/past middle third (>33%)
Tooth Loss	no teeth lost	no teeth lost	≤4 teeth lost	≥5 teeth lost
Local Factors	• maximum PD ≤4mm • mostly horizontal bone loss	• maximum PD ≤5mm • mostly horizontal bone loss	• PD ≥ 6mm • vertical bone loss ≥3mm • furcation class II/III • moderate ridge defects	• masticatory dysfunction • secondary occlusal trauma • severe ridge defects • bite collapse, drifting, flaring • <20 remaining teeth

Grading

= Rate of progression, responsiveness to therapy and assessment of risk

- Rate of CAL/RBL
- Smoking
- Diabetes

Automatically assume Grade B, and adjust based on the other factors

	Grade A	Grade B	Grade C
CAL or RBL	no loss/5 years	<2mm loss/5 years	≥2mm loss/5 years
% RBL/Age	<0.25	0.25 – 1.0	>1.0
Case Phenotype	↑ plaque ↓ bone loss	plaque = bone loss	↓ plaque ↑ bone loss
Smoking	non-smoker	<10 cigarettes/day	≥10 cigarettes/day
Diabetes	no diabetes	diabetes HbA1c < 7.0%	diabetes HbA1c ≥ 7.0%
CRP	<1 mg/L	1-3 mg/L	>3 mg/L

Peri-Implant Diseases

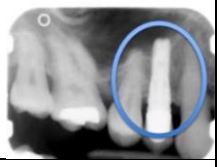
Risk Factors:

- Smoking
- Diabetes
- Poor OHE
- Poor compliance and maintenance
- Excess cement
- Lack of KT around implant
- Hx of previous Perio around teeth

Peri-Implant Health

= Absence of visual signs of inflammation and BOP

- Can't define healthy probing depths around implants because of natural healthy bone loss around the implant. ***Generally ≤ 5mm is ok though***
- Always compared to Baseline (measured upon crown delivery + 1 year after crown delivery)

Peri-implant Mucositis	= Akin to gingivitis - Presence of inflammation and BOP - ↑ PD compared to baseline (likely because of puffy gums - Absence of progressive marginal peri-implant bone loss	 
Peri-implantitis	= Akin to periodontitis - Presence of inflammation and BOP - ↑ PD compared to baseline (1 year after the crown placement) - If no Baseline: PD ≥6mm, RBL ≥ 3mm - Presence of progression marginal peri-implant bone loss	 

	Health	Mucositis	Peri-Implantitis
Inflammation	×	✓	✓
BOP	×	✓	✓
Increased PD	×	✓	✓
Progressive Bone Loss	×	×	✓