Semester 1

EXAM GIL

BDS2

ADA
SOUTH AUSTRALIA

Royal Australasian College
of Dental Surgeons
Let knowledge conquer disease

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INSURANCE

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EMBRYOLOGY– IMPORTANT INFO & ANATOMY

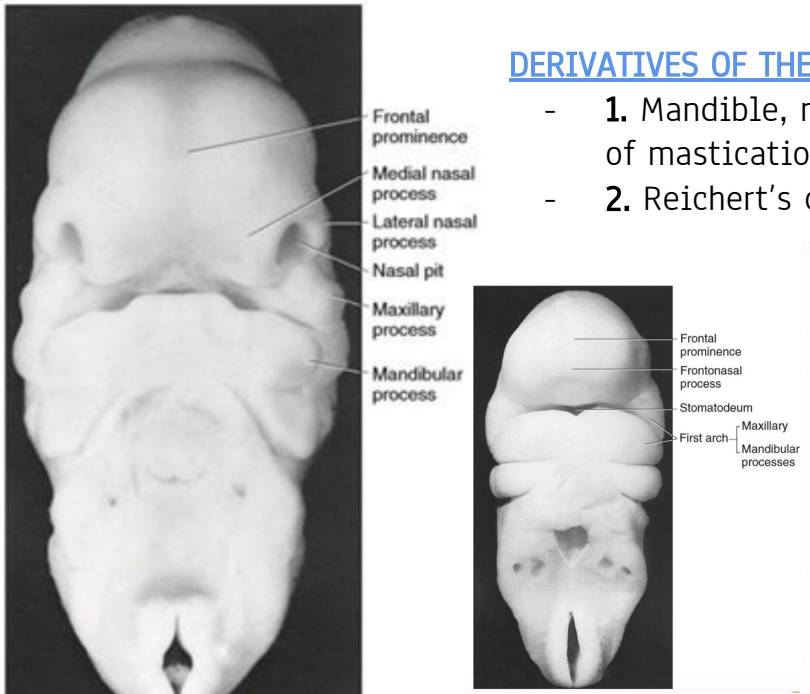
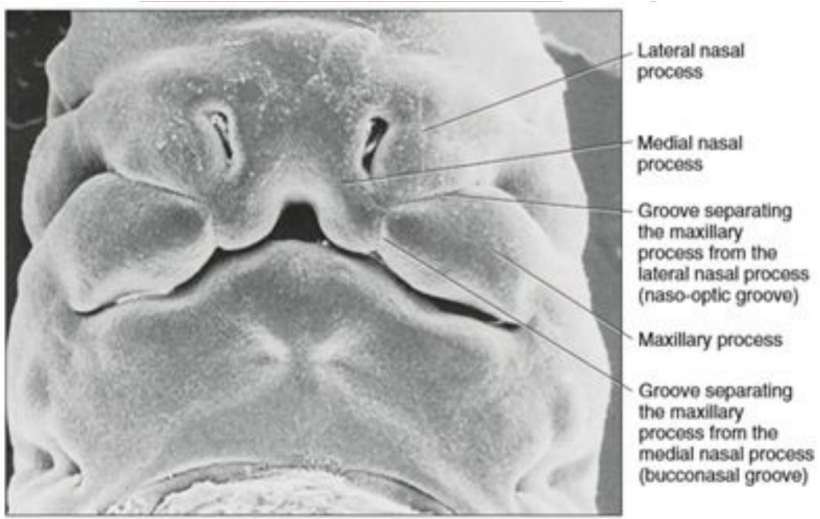


FIGURE 3-13 A 34-day-old embryo viewed from the front. The nasal pits have formed, thereby delineating the lateral and medial nasal processes. (Courtesy of H. Nishimura.)

DERIVATIVES OF THE BRANCHIAL ARCH SYSTEM:

- **1.** Mandible, maxilla, meckel's cartilage, trigeminal nerve, muscles of mastication
- **2.** Reichert's cartilage, facial nerve, muscles of facial expression



Scanning electron micrograph of a human embryo at around 6 weeks of development.

WHAT CAN GO WRONG? CLP

ENVIRONMENT

1. Infectious agents
2. X-Ray Radiation
3. Drugs
4. Hormones
5. Nutritional deficiencies

GENETICS

1. Syndromic
2. Non-syndromic

CLEFT PALATE:

1. Failure of shelves & septum to contact; either lack of growth or physical disturbance during shelf elevation
2. Failure of epithelium adhesion- doesn't breakdown/ resorb
3. Rupture after fusion of shelves
4. Defective merging and consolidation of mesenchyme

Describe different events- molecular/ cellular/ tissue/ organ

** will help you to fully understand the topic

Remember how to describe timing:

- <4wks= embryo death
- Susceptible during weeks 4-8

ODONTOGENESIS – Early Odontogenesis

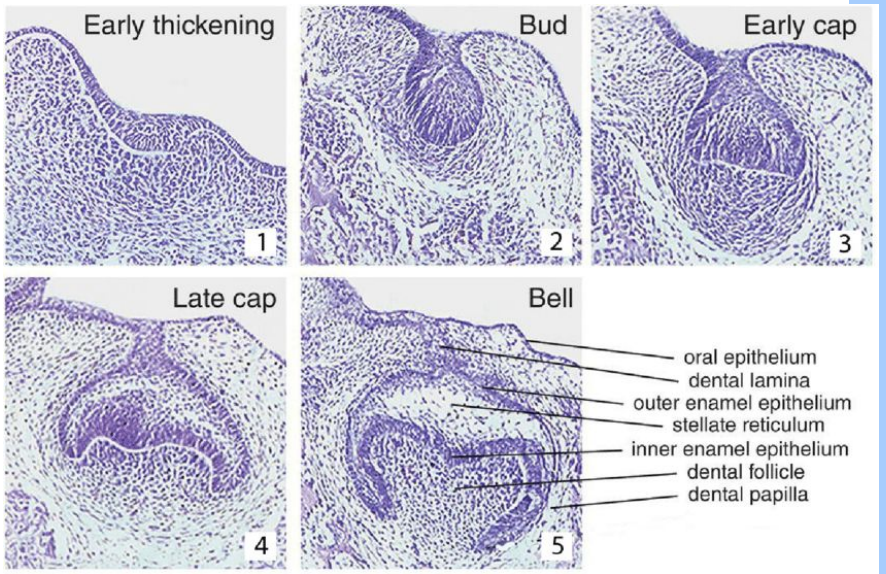
Process of odontogenesis:

Formation of tooth germ from primary epithelial band and dental lamina

Bud stage: Formation of epithelial bud surrounded by condensing ectomesenchyme

Cap stage: Formation of enamel organ and **initial** differentiation of enamel cell types (inner enamel epithelium, outer enamel epithelium, stratum intermedium and stellate reticulum). Dental sac and papilla form and begin genesis of dentine and PDL/cementum respectively

Bell stage: Occlusal shape is now formed by IEE cells, all cells of the enamel organ are now differentiated, communication with epithelial band is severed and tooth germ is embedded in ectomesenchyme



ODONTOGENESIS – Amelogenesis

- Stages of amelogenesis:

Morphogenetic: IEE cells gain polarity (nuclei move proximally, more RER)

Histodifferential: IEE cells differentiate into preameloblasts -> stimulate odontoblasts from DP cells -> differentiate into ameloblasts

Initial Secretory: Formation of initial layer of (aprismatic enamel) on dentine

Secretory: Formation of Tome's processes, secretion of enamel matrix. Proximal end of Tome's process forms interrod enamel, distal portion forms rod enamel

Ruffle and smooth-ended ameloblasts: Modulation cycles occur -> ruffle ended ameloblasts deposit inorganic material onto enamel matrix, smooth ended ameloblasts remove water and organic material

Protective: Ameloblasts lay dormant, 50% of the initial population has now apoptosed

ODONTOGENESIS - Developmental Anomalies

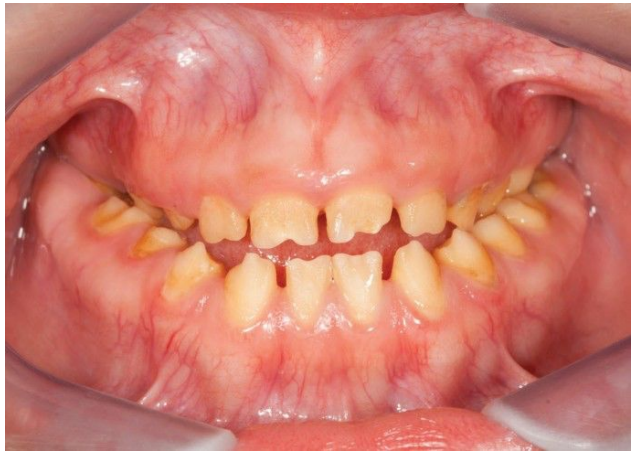
Amelogenesis Imperfecta:

Three types:

Hypoplasia - reduction in amount of enamel matrix produced -> presents as pitting, may cause sensitivity

Hypomineralisation - inability for sufficient organic material to be removed during maturation stage of amelogenesis -> presents as variation in colour from white-yellow-brown, teeth are highly vulnerable to staining and tooth wear

Hypocalcification - insufficient inorganic material deposition during maturative stage -> teeth adopt chalky, yellow appearance, highly vulnerable to staining and tooth wear



Gingivitis + Periodontitis

Definitions:

Gingivitis: Localised (10-30% BOP) & Generalised (>30% BOP)

Periodontitis:

Proximal CAL at ≥ 2 or more non-adjacent teeth

OR

Buccal/oral CAL of ≥3mm with ≥3mm pocketing at ≥ 2 teeth

Without any other attributable causes (trauma, caries, etc)

Gingivitis + Periodontitis

Aetiology of Gingivitis:

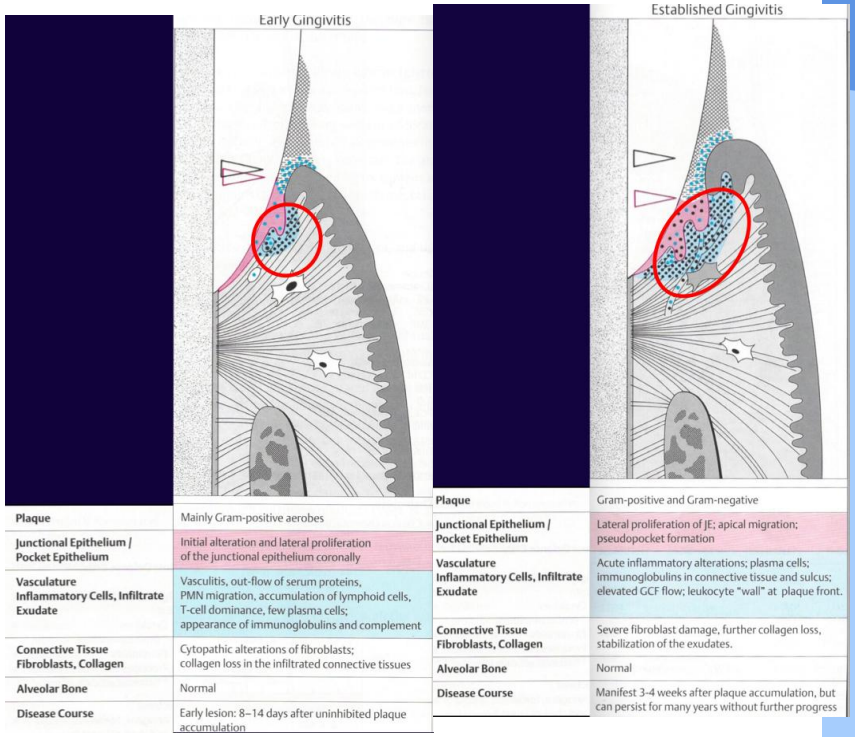
Influx of neutrophils to the gingival sulcus to form a palisade

Release of pro-inflammatory cytokines and enzymes -> influx of lymphocytes & macrophages to the area

Lateral proliferation + apical migration of the junctional epithelium causing the probe to go deeper

Formation of a pseudopocket due to oedema causes increased “pocket depth”

Damage to collagen in CT but no loss of periodontal attachment!! -> periodontitis



	Early Gingivitis	Established Gingivitis
Plaque	Mainly Gram-positive aerobes	Gram-positive and Gram-negative
Junctional Epithelium / Pocket Epithelium	Initial alteration and lateral proliferation of the junctional epithelium coronally	Lateral proliferation of JE; apical migration; pseudopocket formation
Vasculature Inflammatory Cells, Infiltrate Exudate	Vasculitis, out-flow of serum proteins, PMN migration, accumulation of lymphoid cells, T-cell dominance, few plasma cells; appearance of immunoglobulins and complement	Acute inflammatory alterations; plasma cells; immunoglobulins in connective tissue and sulcus; elevated GCF flow; leukocyte “wall” at plaque front.
Connective Tissue Fibroblasts, Collagen	Cytopathic alterations of fibroblasts; collagen loss in the infiltrated connective tissues	Severe fibroblast damage, further collagen loss, stabilization of the exudates.
Alveolar Bone	Normal	Normal
Disease Course	Early lesion: 8–14 days after uninhibited plaque accumulation	Manifest 3-4 weeks after plaque accumulation, but can persist for many years without further progress

Gingivitis + Periodontitis

Signs + Symptoms & Histological Rationale:

Sign/Symptom	Histology
Increase in BOP/bleeding on brushing	Dilation of blood vessels and increase in permeability to permit serum protein influx into CT
Erythema/redness	Increased concentration of erythrocytes in local area due to increased blood flow
Oedema/swelling	Increased blood flow to region to accommodate immune cell influx from chemotactic gradient
Halitosis/bad breath	Production of volatile sulfur compounds from bacteria

Gingivitis + Periodontitis

Predisposing factors (Local):

Calculus, overhangs, open contacts, orthodontic appliances, etc -> sites that harbour pathogenic bacteria

Oral dryness/xerostomia -> reduction in antimicrobial effect of saliva

Modifying Factors (Systemic):

Smoking - reduced immune function, reduced blood flow, shift from beneficial to destructive neutrophil function -> increased numbers of periodontopathogens

Diabetes - increased formation of Advanced Glycation End Products (AGEs) -> increased osteoclasts function & oxidative stress -> increased tissue destruction and reduced tissue repair

Gingivitis + Periodontitis

Treatment of Gingivitis:

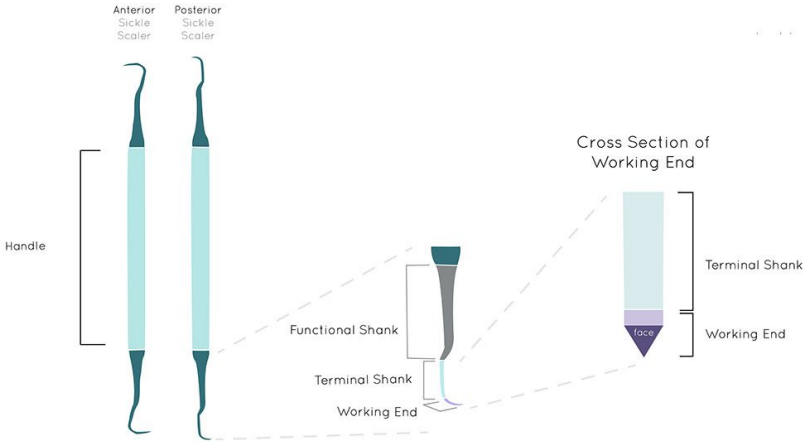
Debridement (Manual or Ultrasonic) - goal is to reduce bacterial load to resolve the inflammatory process and enable healing

Need to know the parts of a manual scaler, as well as optimal angulation (long axis of the tooth parallel to the terminal shank)

Know the contraindications of ultrasonic scaling (non-shielded pacemakers, **bioaerosols**)

Know the function of water in an ultrasonic (flushing debris, lavage, acoustic microstreaming, dissipating heat)

Know what steps follow if treatment is unsuccessful -consider gingivitis may not be biofilm induced, addressing systemic factors, ineffective treatment)



Endocrine - Diabetes Mellitus

Defined as ≥ 7% HbA1c test result

Type 1: Autoimmune condition causing destruction of beta cells in the pancreas and insufficient production of insulin. Requires replacement therapy.

Type 2: Acquired insulin resistance due to chronically high levels of serum glucose with or without secretory defect.

Most common signs and symptoms:

Polyuria, polydipsia, weight loss, retinopathy, neuropathy, nephropathy, reduced healing

Oral Manifestations:

Xerostomia, burning mouth, increased infections/delayed or impaired healing response, accelerated periodontitis or caries -

Management:

Medications, diet, exercise, replacement insulin (primarily T1),

Endocrine – Diabetes Mellitus

Insulin and glucagon are antagonistic hormones - work in tandem to stabilise serum glucose

Insulin promotes anabolic reactions:

- Glycogenesis
- Protein synthesis
- Lipogenesis

Glucagon promotes catabolic reactions:

- Glycolysis
- Proteolysis
- Lipolysis
- Gluconeogenesis

Important to remember other hormones assist glucagon in catabolism (adrenalin, cortisol)

Endocrine - Diabetes Mellitus

Syncope can occur in patients when insulin administration is mistimed

E.g., consider a patient that has come to an appointment in the morning without having breakfast after having administered insulin:

Blood glucose is moved into the insulin-dependent tissues (skeletal muscle, adipose), causing hypoglycaemia

Hypoglycaemia causes lack of glucose to the brain (glucose-dependent tissue) -> temporary loss of consciousness (syncope)

Signs of Syncope:

Lightheadedness Confusion

Sweating Slurred Speech

Nausea Blurry vision

Management:

To avoid:

Plan appointments in the morning

Ensure the patient has eaten before they come to an appointment/the appointment has not interrupted their schedule

In Emergency:

Stop tx

Lay patient in supine position

Provide glucose/refined CHO

Monitor/supervise patient

Endocrine – Aging

Oestrogen (W):

Decreased production upon completion of menstruation, leading to:

- Reduced apoptosis of osteoclasts
- Decreased IGF-1 formation leading to decreased formation of osteoblasts
- Reduced osteoprotegerin formation

Then in vasculature this deficiency causes:

- Reduced formation of coagulation factors
- Reduced function of platelets

Growth Hormone:

Decreased production causing:

- Reduced muscle mass
- Increased adiposity

Aldosterone:

Reduced secretion which may increase the risk of hyponatremia or hyperkaleamia

Melatonin:

Reduced secretion which causes advanced sleep phase syndrome (earlier sleep and waking)

Endocrine – Aging

Calcitropic Hormones:

Reduced amounts of vitamin D production + increased resistance leads to reduced calcium -> depletion of bone stores,

Increased secretion of PTH, further withdrawing calcium from bone stores

Risk factors for osteoporosis (reduced bone density)

Insulin:

Increased resistance, **increased risk of T2DM**

Thyroid Hormones:

No changes to hormone levels, however higher risk of thyroid dysfunctions:

Hyperparathyroidism:

Symptoms related to accelerated metabolism including:

Heat intolerance, sweating, fatigue, weight loss, muscle weakness, accelerated bone loss

Hypoparathyroidism:

Symptoms related to reduce metabolism including:

Weakness, cold sensitivity, memory less, cognitive decline

Microbiology – Cell Structure

Cell Component	Function
Capsules	Virulence factor for evasion
Flagella	Move to nutrients/environmentally favourable conditions
Fimbriae	Involved in attachment
Cell wall	Prevent bursting of the cell in hypotonic conditions
Granules	Store nutrients when they cannot be sourced from elsewhere
Endospores (not a true component)	Form of the cell which is highly resistant to thermal and chemical extremes

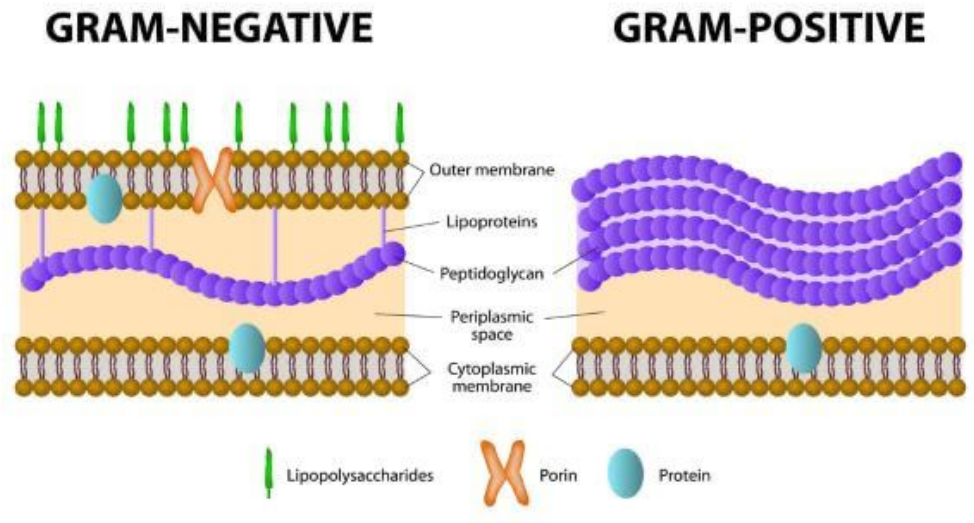
Microbiology - Gram +ve/-ve

Gram Positive vs Gram Negative Bacteria:

Positive: Peptidoglycan layer is the outermost layer of the cell

Negative: Peptidoglycan layer is protected by an outer membrane

Antibacterials that target the peptidoglycan layer are more effective on gram positives than negatives - more direct access to the peptidoglycan layer (lysozyme, penicillin)



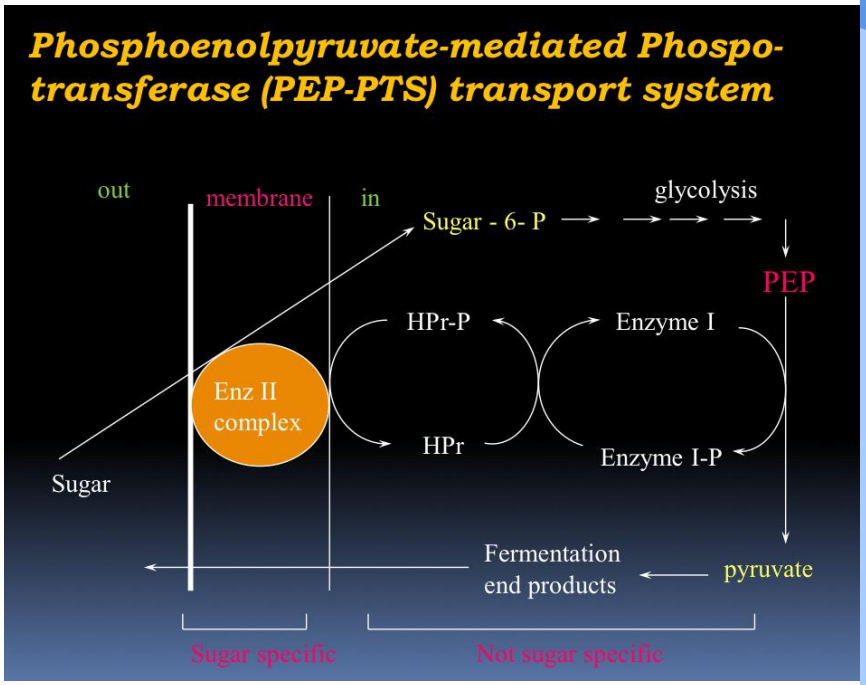
Microbiology - Virulence Factors

Mechanism	Type	Function
Siderophores	Growth	Iron sequestration
Exotoxins (proteases, cytolyins, haemolysins)	Growth	Release nutrients and enable infection to spread
Endotoxins	Growth/evasion	Promote host inflammation (dysregulated)
Quorum sensing/autoinducers	Growth/evasion	Keep bacteria away from immune cells until numbers are sufficient for virulence
Type 3 Secretion	Growth	Mechanism to “inject” virulence factors directly into a cell
Gingipains	Growth	Endopeptidase for nutrients Sequesters iron from the environment

Microbiology - Metabolism

PEP-PTS:

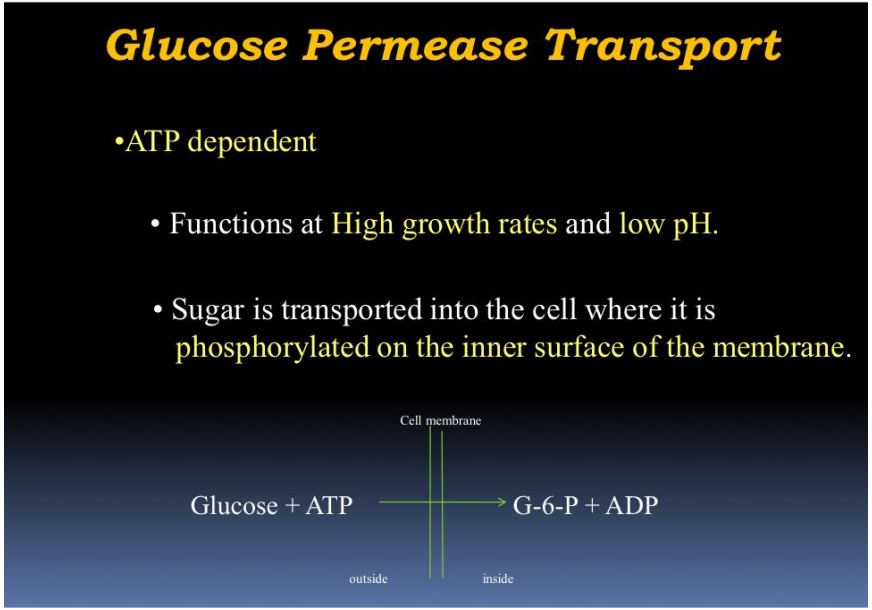
- High affinity system used in times of famine (low sugar higher pH)
- Does not require ATP
- Sugar is uptaken and via glycolysis becomes PEP
- PEP donates a phosphate to the Enz II complex
- Enz II Complex uses the phosphate to bring in a sugar
- Associated with the formation of **formic, acetic and ethanoic acid**



Microbiology - Metabolism

Glucose Permease:

- ATP dependent
- Low affinity and used in times of feast (high sugar low pH)
- Sugar is brought into the cell where it is phosphorylated on the membrane
- Associated with the formation of **lactic acid**

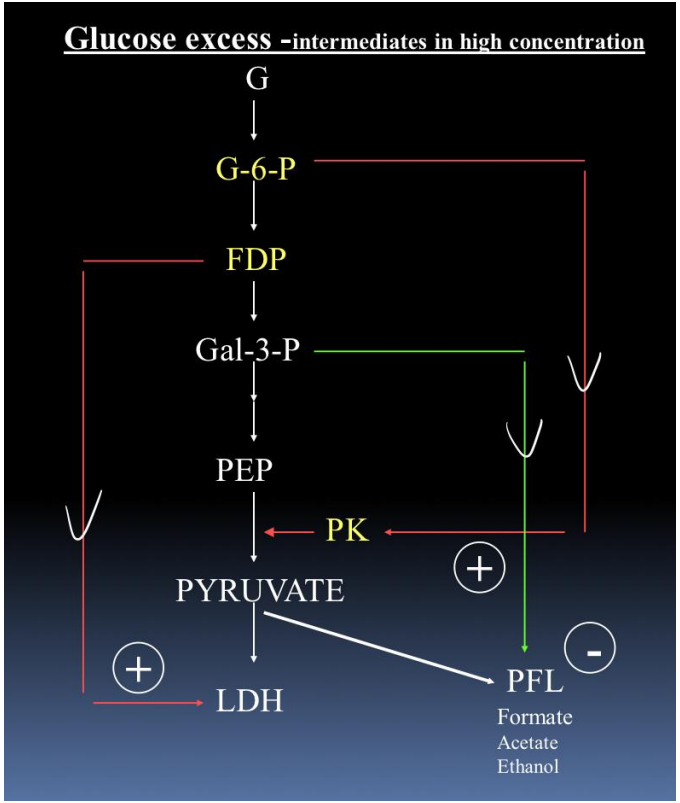


Microbiology - Metabolism

Controls of Glucose Metabolism:

In high sugar/low pH conditions there is:

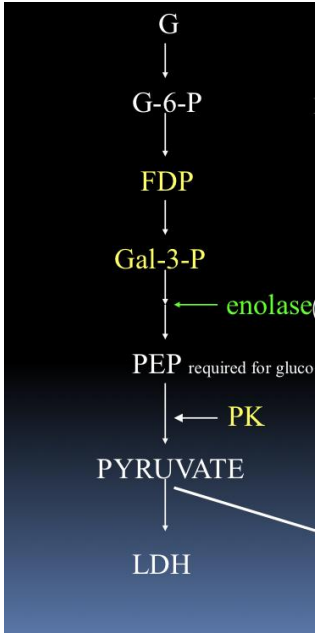
- Inhibition of pyruvate formate lyase
- Increased activity of lactate dehydrogenase
- Increased activity of pyruvate kinase (PEP -> pyruvate) - **no need for PEP-PTS system in times of feast**



Microbiology - Metabolism

Effect of Fluoride:

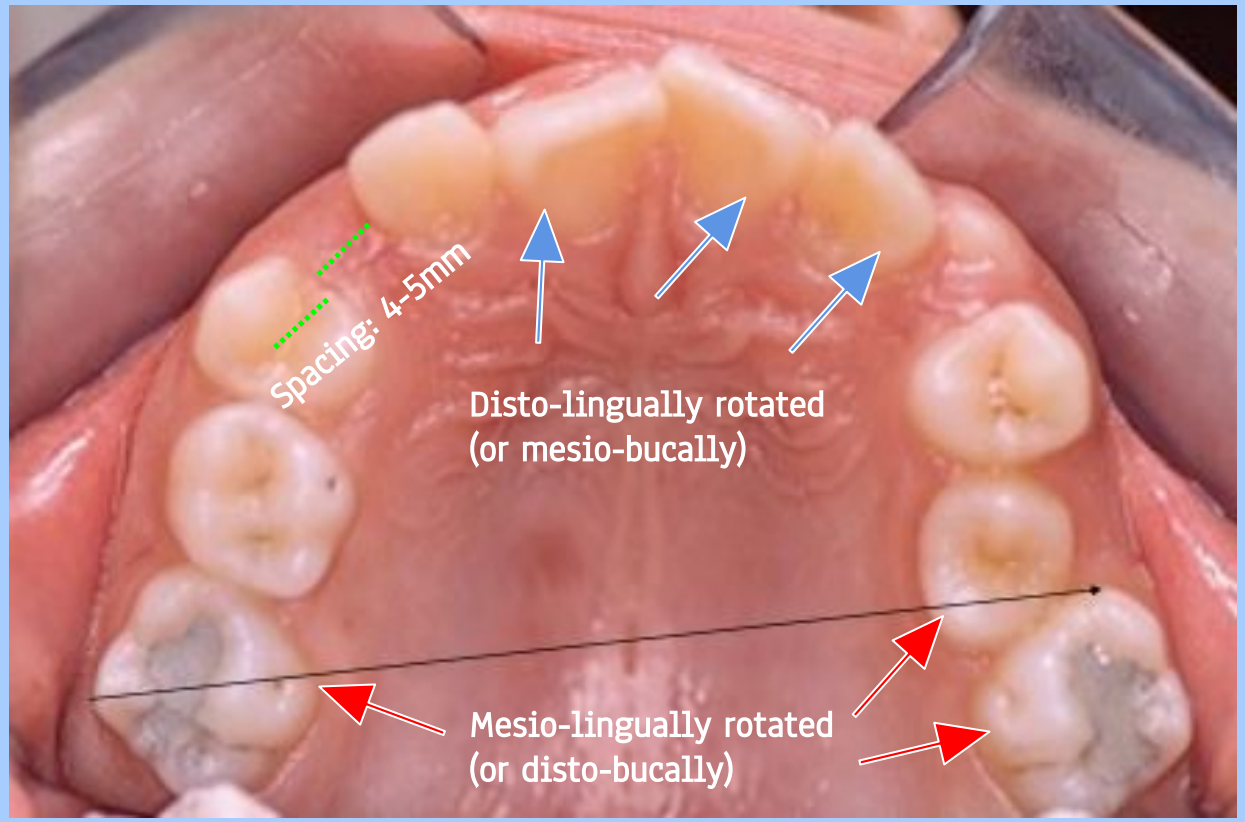
- Inhibits the enzyme enolase which is responsible for the formation for PEP
- This means that the PEP-PTS system is inhibited when fluoride concentrations are higher -> its more effective to brush at night than in the morning as the high affinity system is disabled when it is needed most -> bacteria perish



Occlusal Analysis - Template

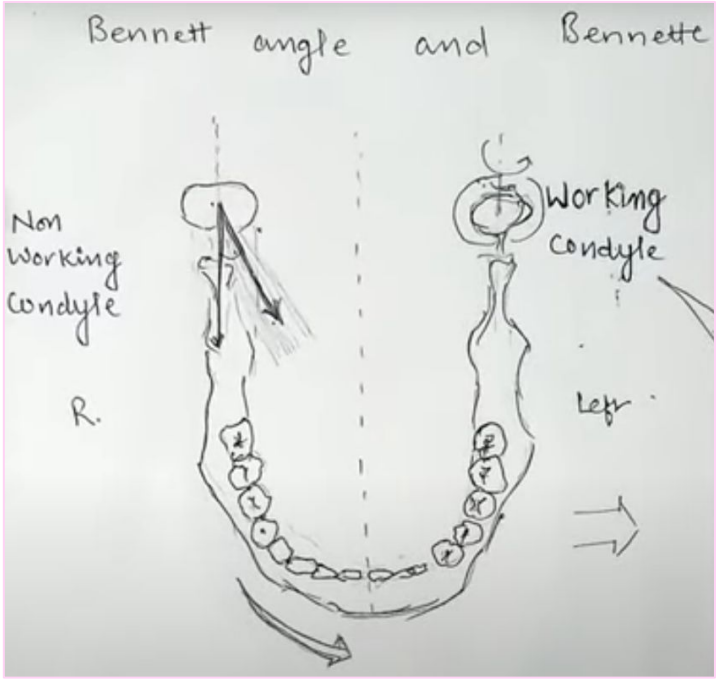
Being SYSTEMATIC is KEY

1. Teeth present/missing
2. Morphology of teeth (eg. cusp of carabelli, peg-shaped lateral incisors, macro/microdontia)
3. Wear (eg. attrition, erosion, abrasion -> assess extent of wear: mild, moderate, severe)
4. Crowding, spacing, rotations
5. Axial inclinations
6. Shape of dental arch (Mx + Md)
7. Curve of Spee + Monsoon's curve
8. Angle molar/canine classification
9. Overbite (%) / overjet (mm)
10. Mediolateral relationships (eg. midline of Md is shifted 2mm to the left, crossbite)


Spacing: 4-5mm
Disto-lingually rotated
(or mesio-bucally)
Mesio-lingually rotated
(or disto-bucally)

Occlusal Analysis – Key Terms

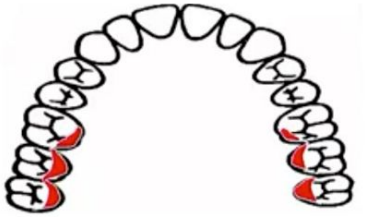
- **Bennett Angle:** angle formed between the sagittal plane and the non-working condyle during lateral excursion
- **Bennett Movement**: lateral shift of the Md along the working side of the condyle
- **Functional cusp** (Mx = lingual cusps | Md = buccal cusps)
- **Non-functional cusp** (Mx = buccal cusps | Md = lingual cusps)
- **Occlusal vertical dimension (OVD):** the distance between two selected anatomic or marked points (usually nosetip and chin) in maximum intercuspal position



Patterns of Erosion

What patterns of erosion are experienced during reflux *(GORD)*?

Palatal of Mx posteriors are mainly affected -> may spread further anteriorly overtime



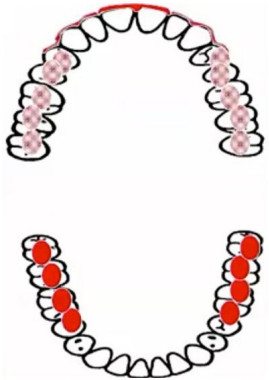
What patterns of erosion are experienced during vomiting?

- Palatal of Mx *(more evenly distributed between anteriors & posteriors)*
- Tongue covers Md teeth during vomiting *(but can settle on Md if not rinsed out)*

What patterns of erosion are experienced for acidic drinks?

- Lowers are more affected *(particularly occlusal -> due to liquid being held in FoM before being swallowed)*
- Labial *(entry of acid into mouth)*
- Anterior palatal uppers *(tongue pushes liquid upward -> flushed over both arches during swallowing)*

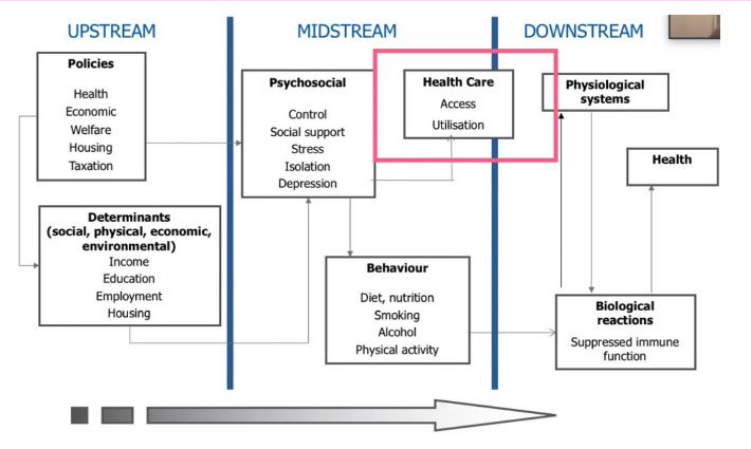
NOTE: if bottom has lip protection or drink is consumed with straw, anterior labial surfaces are generally unaffected)

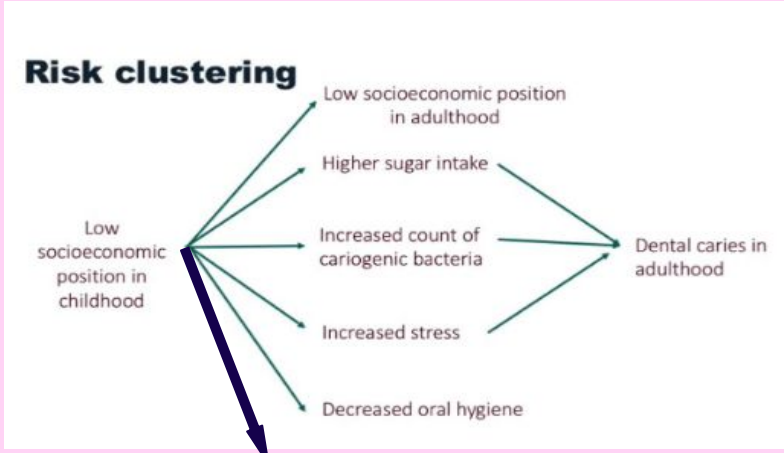


What patterns of erosion are experienced for mastication of acidic foods?

U & L occlusal surfaces are 'evenly' distributed

Social Determinants





Pt Scenario: Pt comes into the SADS clinics. They have a low SES background. Explain how the Pt may be at increased risk of developing dental caries?

- Possibly reduced parental education/guidance
- Possibly reduced access to F water supply
- Possibly limited access to dental services

Dental Anxiety

- Why is this important?
- Fear-inducing aspects (specific, adverse reactions, loss of control)
- 4A's (ask, assess, acknowledge, address)
- Management strategies (muscle relaxation, controlled breathing, tell-show-do, providing time-markers/rest-breaks, distractions, positive encouragement)

TRIM – Successful OHI Plan

Timing
- Good timing
- Poor timing

Relevance
- OHI-centred and catered towards Pt's expectations, values & beliefs
- Meaningful to the Pt

Involvement
- Ask Pt if they would like to alter any parts of your proposed OHI
- Ensure OHI is catered towards the Pt's SHx & daily lifestyle

Method
- Use Pt end goal as a motivation to pursue OHI
- Introduce gradual changes in small increments

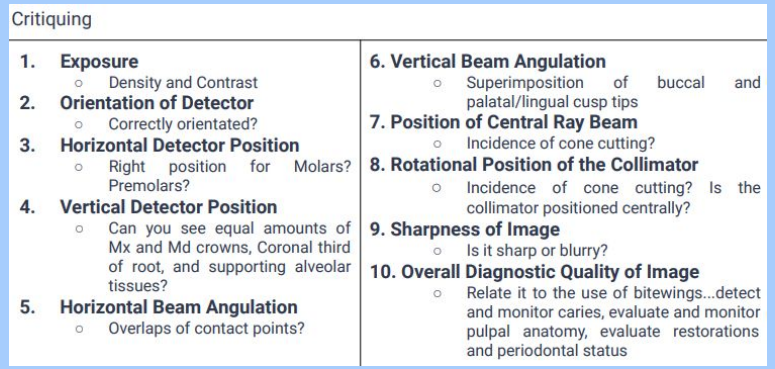
EBD

- Types of studies
- P values and confidence intervals (<0.05)
- Odds ratio : >1.0
- Incidence + prevalence

[Self revision]

Radiography

CRITIQUE THE RADIOGRAPH



Critiquing

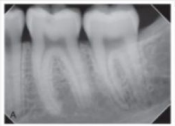
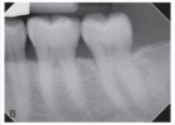
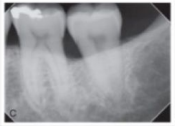
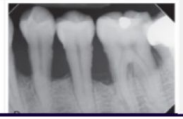
1. **Exposure**
   - Density and Contrast
2. **Orientation of Detector**
   - Correctly orientated?
3. **Horizontal Detector Position**
   - Right position for Molars? Premolars?
4. **Vertical Detector Position**
   - Can you see equal amounts of Mx and Md crowns, Coronal third of root, and supporting alveolar tissues?
5. **Horizontal Beam Angulation**
   - Overlaps of contact points?
6. **Vertical Beam Angulation**
   - Superimposition of buccal and palatal/lingual cusp tips
7. **Position of Central Ray Beam**
   - Incidence of cone cutting?
8. **Rotational Position of the Collimator**
   - Incidence of cone cutting? Is the collimator positioned centrally?
9. **Sharpness of Image**
   - Is it sharp or blurry?
10. **Overall Diagnostic Quality of Image**
    - Relate it to the use of bitewings...detect and monitor caries, evaluate and monitor pulpal anatomy, evaluate restorations and periodontal status

INTERPRET THE RADIOGRAPH or Provide a RADIOGRAPH **REPORT**

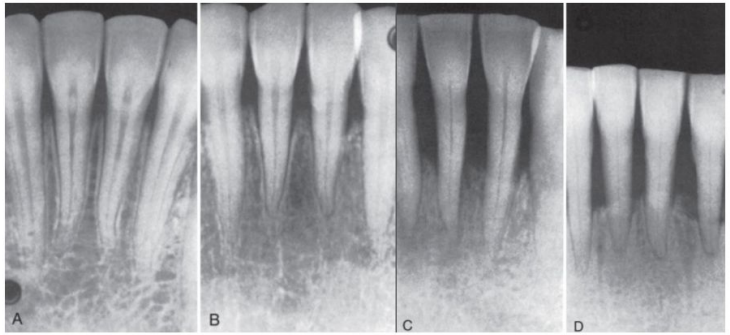
1. Identify teeth present, unerupted/missing, not imaged, restorations, R/L (start from Q1 -> Q2 -> Q3 -> Q4)
2. Identify abnormalities if present (eg. horizontal/vertical alveolar bone loss, calculus present, R/L near the tooth apex, artifacts, etc.)

In all scenarios, PLEASE PLEASE label the radiograph -> eg. LHS Molar BW, 12-22 PA, etc.

Radiography – Periodontitis

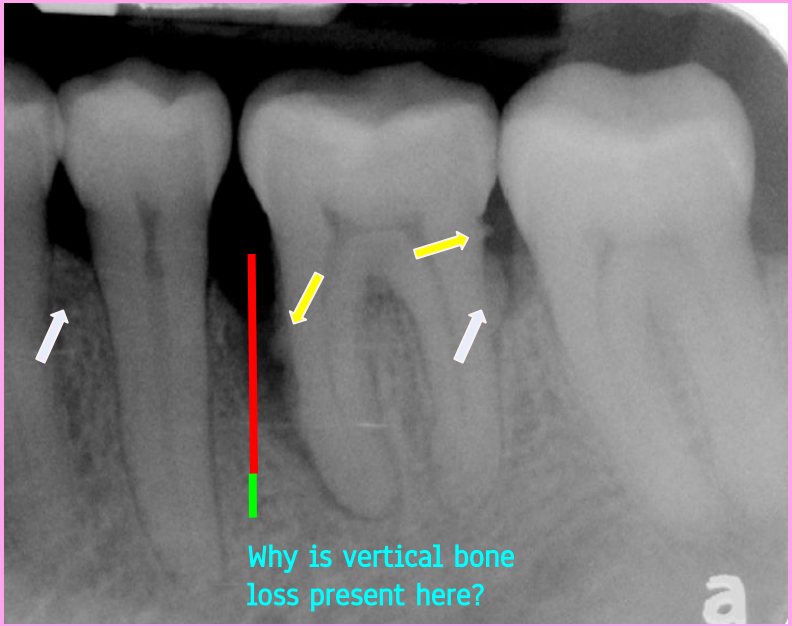
Severity of Periodontitis	Clinical Attachment Loss (mm)	Distance From Alveolar Crest to CEJ (mm)	Radiographic Findings	Radiographic bone loss	Periodontitis Stage
Health	0	<2		n/a	n/a
Mild	1–2	3–4		<15%	I
Moderate	3–4	5–6		15% - 33% coronal 1/3	II
Severe	≥5	≥7		middle to apical 1/3	III or IV

Outline the progression of radiographic changes for periodontitis in this PT



A: alveolar crest is 1-2mm below CEJ
B: break in lamina dura -> widening of PDL
C: height of interdental septums are reduced
D: bone crater around 32

Case Scenario



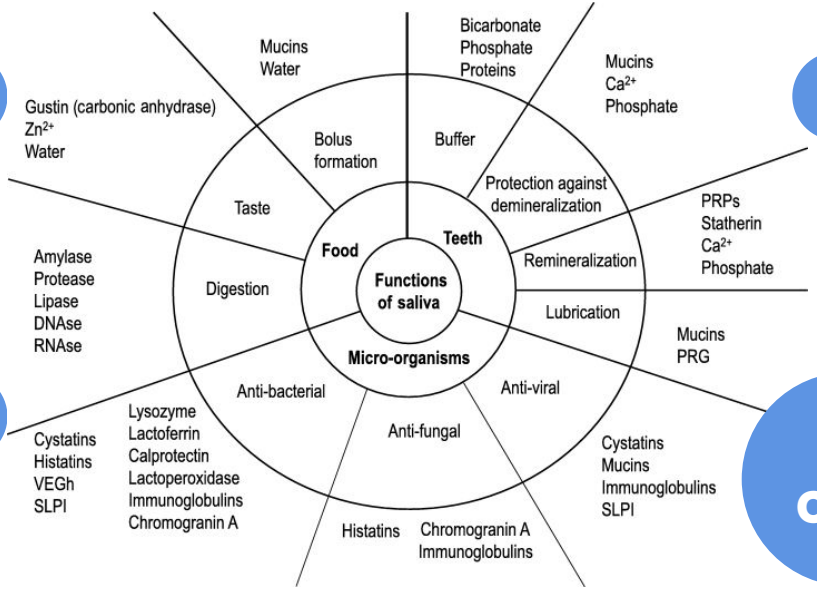
FUNCTIONS of SALIVA

DEFENCE

- Physico-chemical, non-immunological, immunological

DIGESTION

- Taste, bolus formation



BUFFERING

- pKa
- Three systems esp. Bicarbonate

LUBRICATION & COMFORTABILITY

Dental diagnosis

EROSION

- Understand how it begins + progresses
- Shiny/ clean lesions; scooped appearance
- Endogenous or exogenous source

CARIES

- Associated with areas of plaque accumulation
- Can be cavitated or non-cavitated (incipient)
- Know pathogenesis of caries
- Treatment: when MI can be used (remineralisation) vs intervention
- Pt risk factors (inc. Stephens curve, denim-remin cycle)

CALCULUS

- Associated with areas of plaque accumulation + high salivary flow
- Understand debridement, instruments, post-op instructions

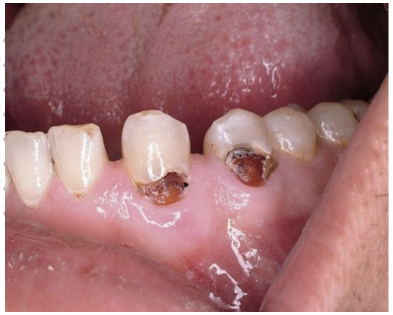
ATTRITION

- Often associated with incisal edges
- Tooth-tooth contact (clenching, grinding)

ABRASION

- Shiny or dull wear facets around cervical area of tooth (most likely from toothbrushing)
- Also may be due to habits such as pipe smoking, chewing seeds etc.

Treatment PLANNING



Name	Steven Smith-Jones		
Age	30 years old	**MHx**	Clear
Gender	Male	**CC**	Lower brown spots on teeth

TYPE OF Q.

<u>WILL be scenario based</u>
Provided with both a history; accompanied by either/ both intraoral photographs and radiographs

APPROACH SYSTEMATICALLY

- Summarise key findings (if it asks you too)
- Use subheadings
  - CC/ Additional tests/ Preventive case/ Periodontal/ Restorative
- If asked- rationalise your steps e.g. *why Tooth Mousse over neutrafluor 5000*?

Preventive products

AT-HOME PRODUCTS

- Regular toothpaste (1500ppm)
- NeutraFlour 5000 (5000 ppm)
- Sensodyne Rapid Relief (1450 ppm with SnF)
- Tooth Mousse Plus (900ppm NaF with 10% CCP-ACP)

IN- CHAIR FLUORIDE

- Duraphat (22600ppm NaF)
- MI Varnish (5%NaF + 2%CCP-ACP)
- **Remember post-op instructions + contraindications!!**

MECHANICAL REMOVAL

- Manual or electric toothbrush with a small, soft head
- Modified bass technique- 45 deg.
- Spit no rinse

INTERDENTAL

- Flossing; regular floss or super floss (thicker)
- Interdental brushes (especially if manual dexterity is low)
- - Consider including in subsequent appointments if compliance is low

+ JUSTIFICATIONS

Dental Materials

GIC

Pros:

- Fluoride releasing, quick application, biocompatible

Cons:

- long setting time, poor wear resistance, moisture sensitive first 24hrs, poor mechanical properties

Composite Resin

Pros:

- Highly aesthetic; with a high polish + finishing ability
- Micromechanical bond strength

Cons:

- Moisture sensitive
- Polymerisation shrinkage

Amalgam

Pros:

- Biocompatibility
- Longevity
- Corrosive marginal seal
- Low moisture control needed

Cons:

- Poor aesthetics
- Non-conservarive cavity preparation
- Macromechanical bonding

Comparison OF PROPERTIES

	CR	AMALGAM	GIC
HIGH BIOCOMPATIBILITY	✗	✓	✓
MOISTURE SENSITIVE	✓	✗	✓
LONGEVITY	✓	✓	✗

Restorative Steps

SHADE SELECTION

Don't forget to select your shade before rubber dam - why?

After conditioning the dentine- dry & do not dessicate- why?

DENTINE

REMEMBER!

Etching, light cure, check occlusion, patient satisfaction

Post-op instructions given; write what they will be

POIG