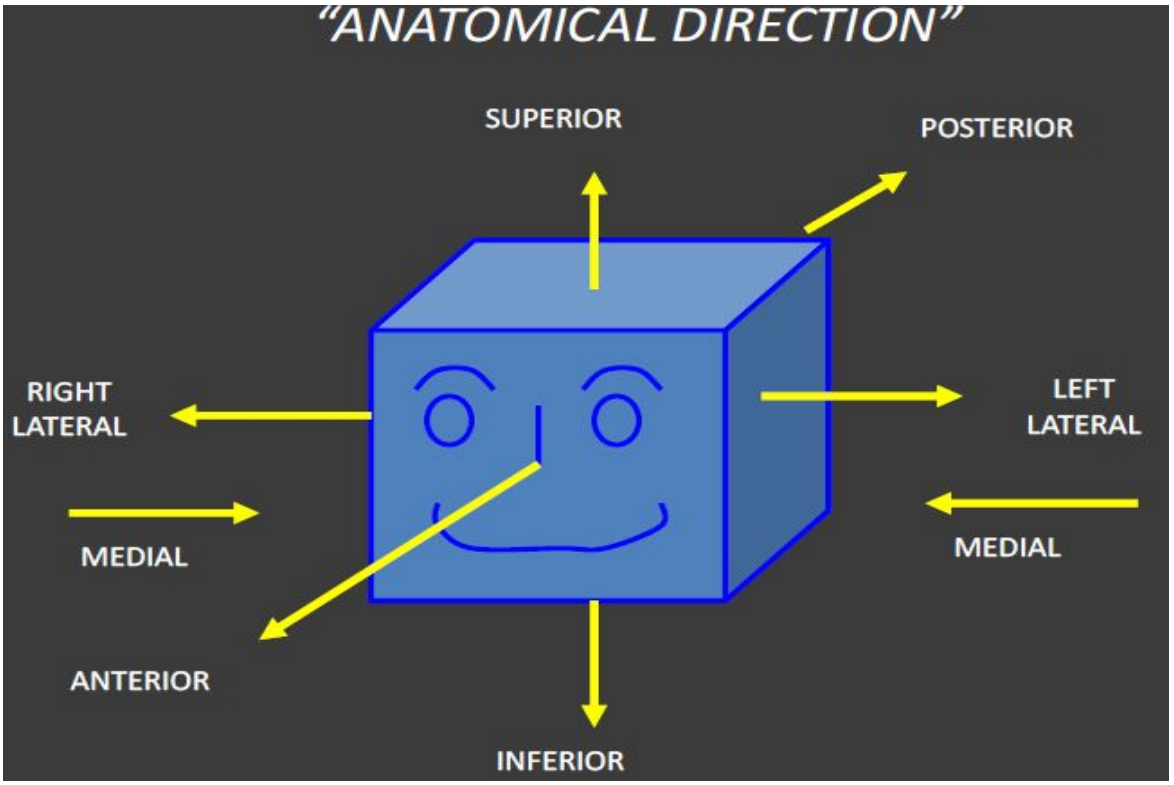
Russell Lee

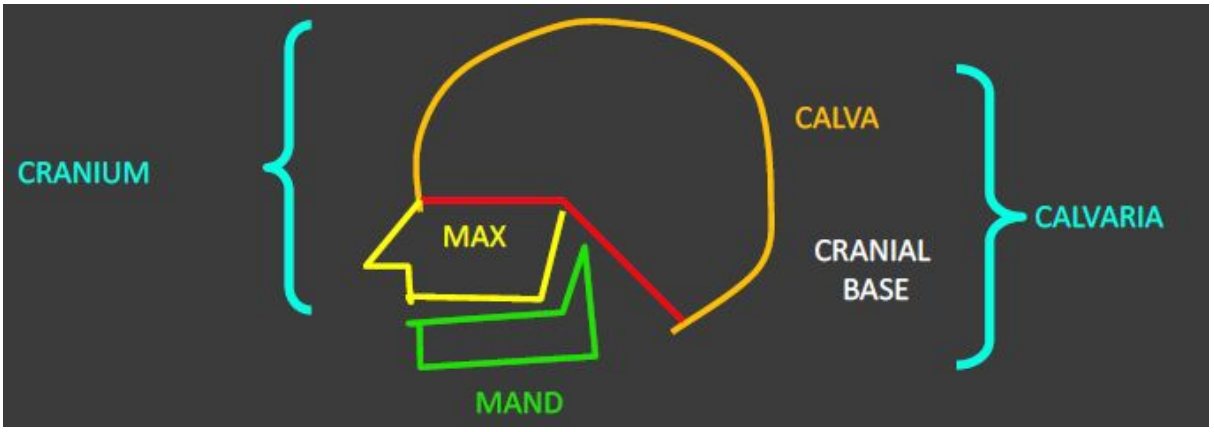
Kaidonis

1. Osteology of the skull

- Anatomical Direction



- Components of the skull
    - Calva + Cranial base → Calvaria
    - Calvaria + Maxilla → Cranium
    - Cranium + mandible → Skull



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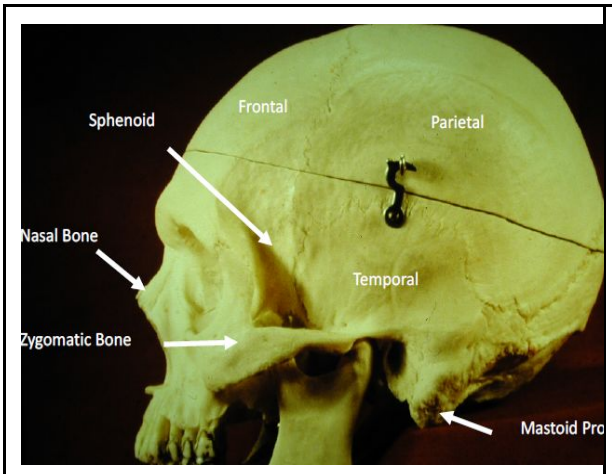
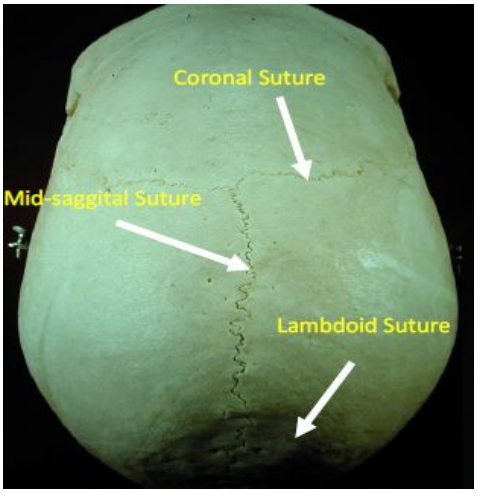
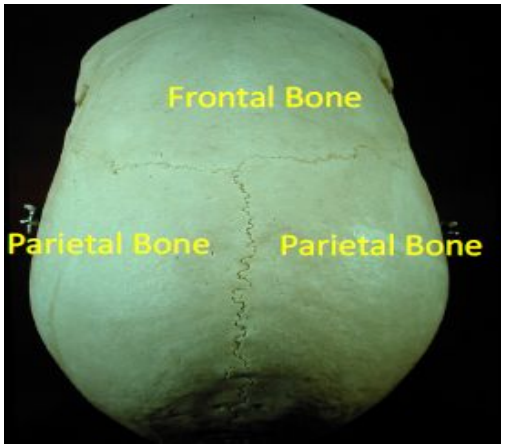
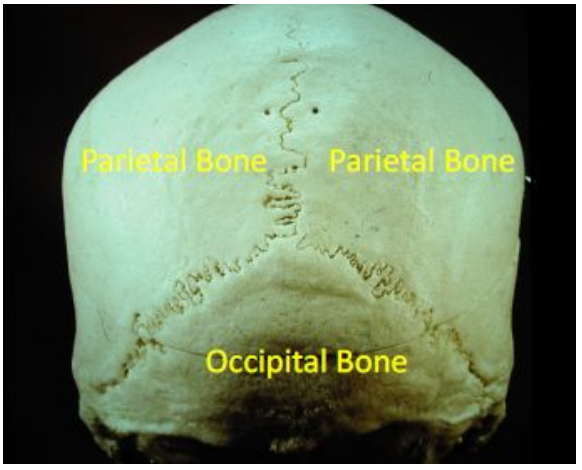
- Bones of the Skull
    - Calva (can remember them as FTOP)
        - Frontal (Most anterior)
        - Temporal (Lateral)
        - Occipital (Posterior)
        - Parietal (Superior)
        - These are all joined by fibrous joints known as Sutures, Good to know the following names (pictured below)









    - Cranial Base (can remember as NESO)
        - Nasal
        - Ethmoid
        - Sphenoid
        - Occipital

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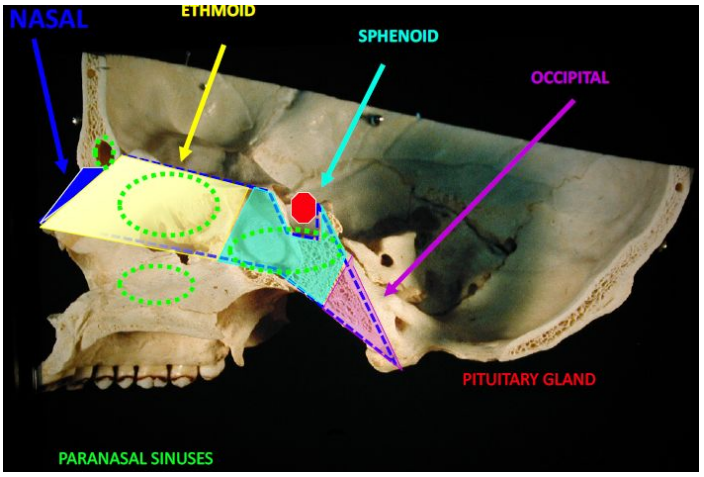
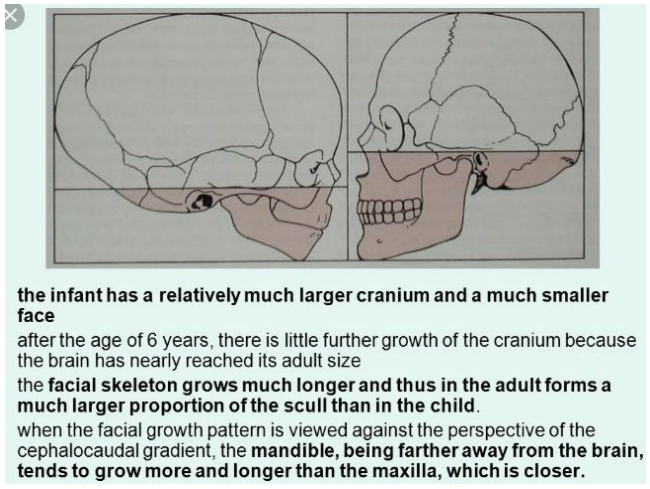


- Causes of variation in skulls
    - Age changes
        - Variation due to rate of growth of facial skeleton vs cranium
        - Cranium does not grow at the same rate as the facial skeleton over one's lifetime

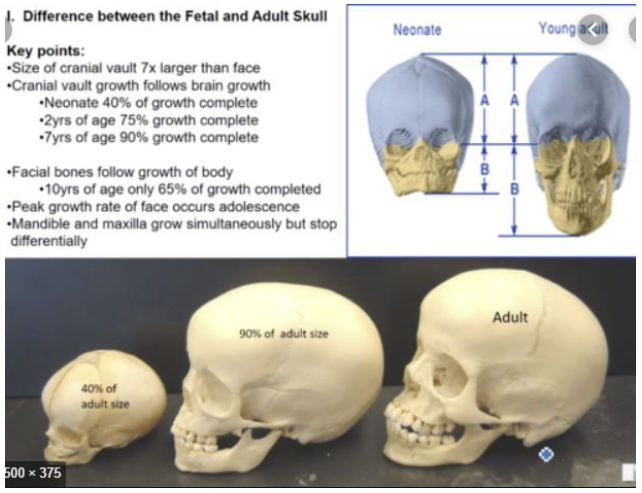


the infant has a relatively much larger cranium and a much smaller face

after the age of 6 years, there is little further growth of the cranium because the brain has nearly reached its adult size

the **facial skeleton grows much longer and thus in the adult forms a much larger proportion of the scull than in the child**.

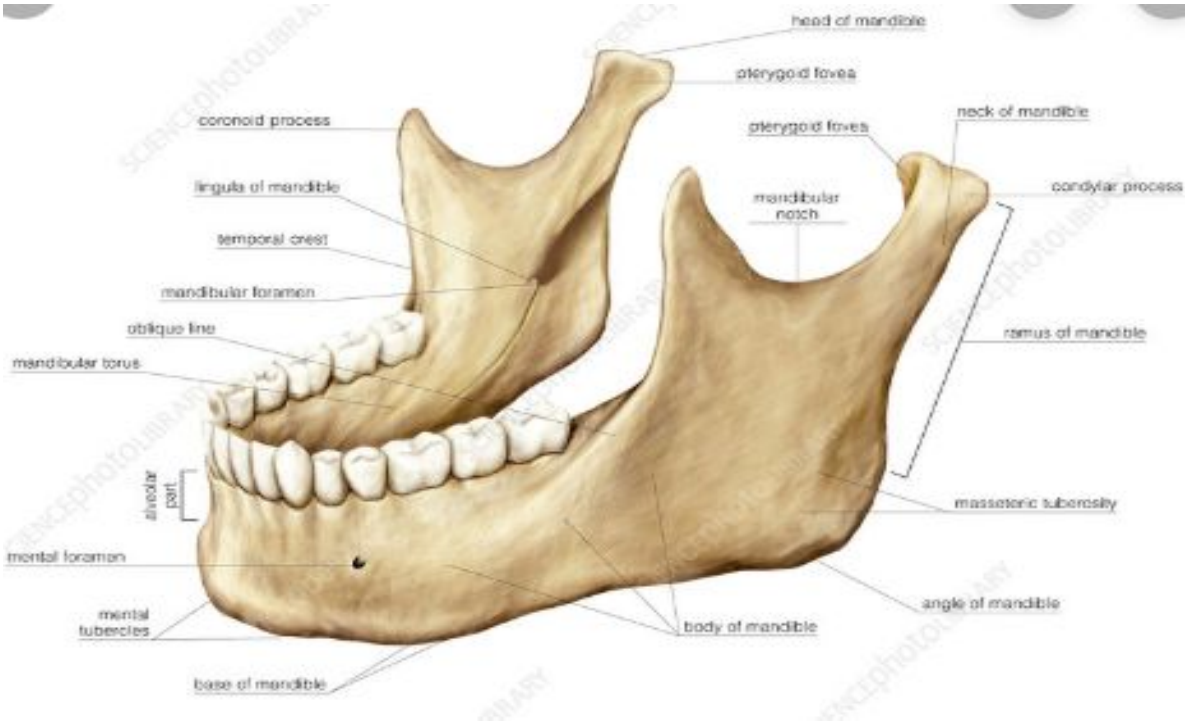
when the facial growth pattern is viewed against the perspective of the cephalocaudal gradient, the **mandible, being farther away from the brain, tends to grow more and longer than the maxilla, which is closer.**

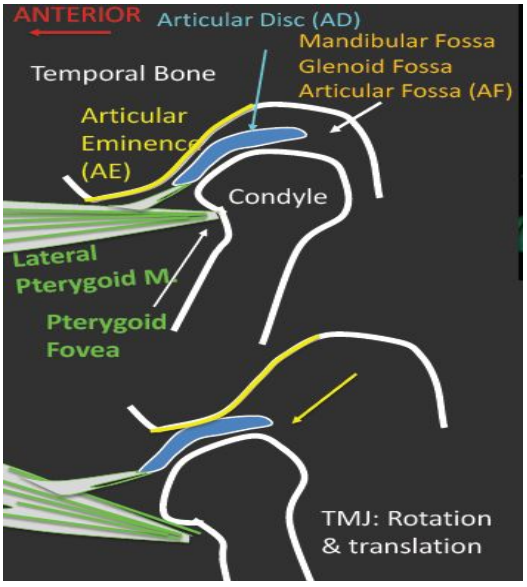


    - Ethnicity
        - Variation due to genetic racial makeup
    - Sexual Dimorphism
        - Males → larger + more pronounced features
        - Females → Smaller + more delicate features
    - Evolutionary changes
        - Change due to time

- Mandible
    - Components of the mandible
        - Condyle
        - Pterygoid Fovea (muscle attachment point, a very small process)
        - Neck

- Ramus
- Angle
- Body



- Tempro-Mandibular Joint/TMJ
  - The synovial joint between the Cranium and the mandible
  - Consists of
    - Articular eminence
    - Articular disk (made of fibrocartilage)
    - Condyle (head of condyle coated in Fibrocartilage)
    - Mandibular Fossa, also known as (lined with fibrocartilage)
      - Glenoid Fossa
      - Articular Fossa
      - 

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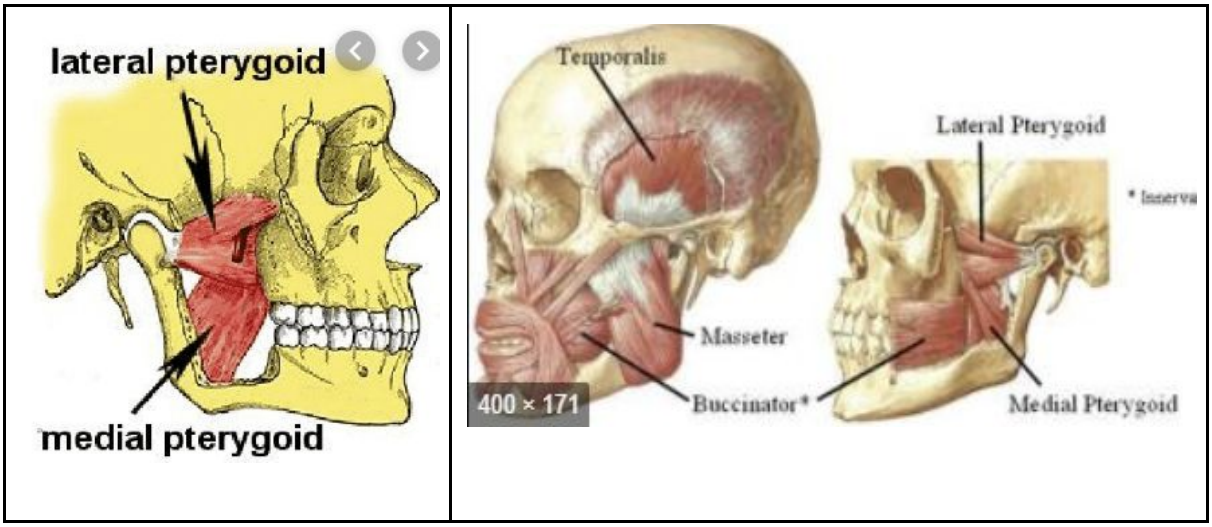
- Muscles of Mastication

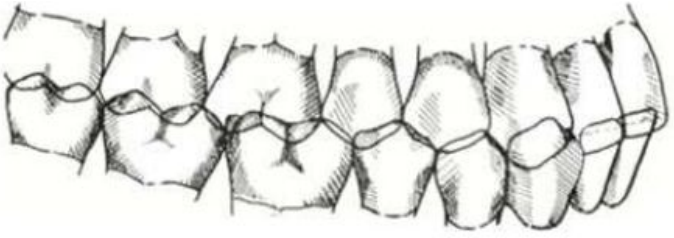
Muscle	**Origin**	**Insertion**	**Action**
Temporalis	Floor of Temporal fossa	Coronoid process	JAW CLOSING Elevates & retracts mandible
Masseter	Maxillary process (inferior border) of zygomatic arch	ramus of the mandible	JAW CLOSING Elevates mandible
Digastric	Anterior: Digastric fossa of Mandible Posterior: mastoid notch	Intermediate tendon attached to hyoid bone	JAW OPENING Depress the mandible
Lateral pterygoid	Infratemporal surface of greater wing of sphenoid Lateral surface of lateral pterygoid plate	Joint capsule of TMJ attached to fibrocartilaginous disc Condyloid process of mandible/ neck of the mandible	JAW OPENING Acts bilaterally to protract mandible & depress chin Act unilaterally to swing jaw contralaterally Assists in depressing mandible by protracting the mandible to allow it to rotate to a greater extent
Medial pterygoid	Medial surface of lateral pterygoid plate Tuberosity of maxilla	Medial surface of angle of mandible inferior to mandibular foramen	JAW CLOSING Acts with masseter to protract & elevate mandible

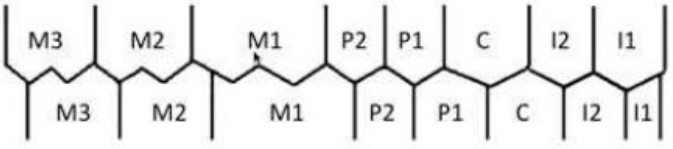


2. Function of masticatory system + Occlusion

Dental Occlusion

1. Maximum occlusal tooth contact between opposing teeth
    a. Maximum Intercuspal Position also known as,
    b. Maximum Intercuspation
    c. Maximum interdigitation
    d. Intercuspal occlusion
    e. Every Tooth Opposes 2 teeth, except Md Central Incisors

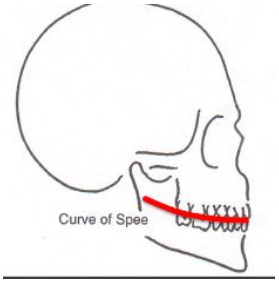


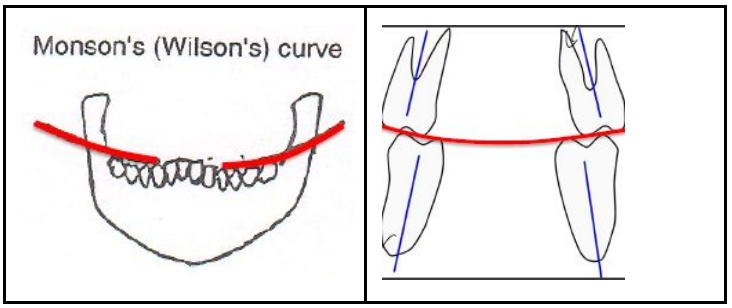


    f.
    g. MIP determines Occlusal Vertical Dimension/OVD
    h. When sitting upright with lips together and muscles relaxed, Md drops under own weight → known as Rest position/RP
    i. RP - OVD = Interocclusal Distance/IOD/freeway space
        i. Required for speech and comfort

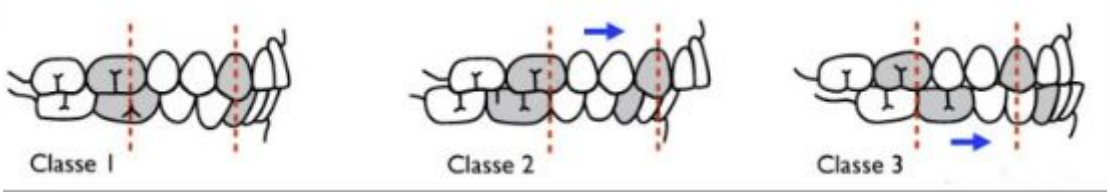
2. Tooth Contacts
    a. All other teeth except for Molars occlude along marginal ridges
    b. Mx Molars MP cusp occlude into central fossa of Md molar
    c. Md Molars DB cusp occlude into central fossa of Mx Molar

3. Axial Inclinations
    a. Proclination
        i. Tooth tilted anteriorly
    b. Retroclination
        i. Tooth tilted posteriorly
4. Overjet + Overbite
    a. Overjet
        i. Degree of vertical overlap between Mx and Md incisors
    b. Overbite
        i. Degree of horizontal protrusion between Mx and Md incisors

5. Occlusal curvatures
    a. Curve of Spee/Smile line
        i. Either
            1. Flat
            2. Moderate
            3. Present
            4. 

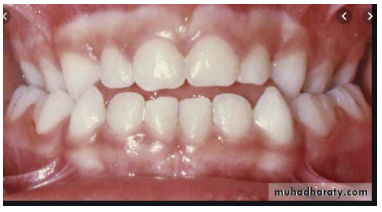
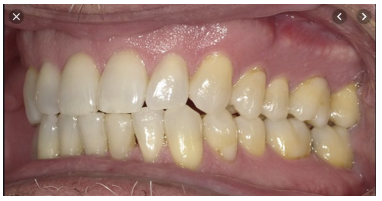
    b. Curve of Monson/Wilson
        i. Either
            1. Flat
            2. Present
            3. 

            4.
6. Angles Classification
    a. Molar
        i. Class 1 Buccal groove lies in line with MB cusp
        ii. Class 2 Buccal groove lies posterior to MB cusp
        iii. Class 3 Buccal groove lies anterior to MB cusp



b. Canine
    i. Class 1 Distal Surface of Md canine within 1 premolar width of mesial surface of Mx canine
    ii. Class 2 Distal surface of Mx canine is distal to mesial surface of Mx canine by at least 1 premolar
    iii. Class 3 Distal Surface of Md canine is mesial to mesial surface of Mx canine by at least 1 premolar

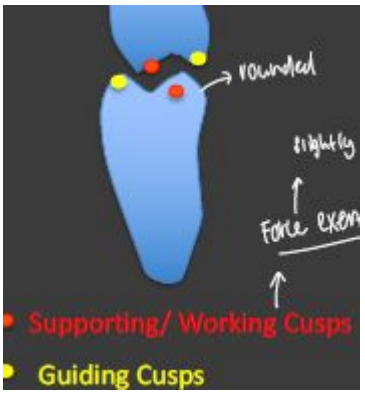
7. Occlusion
    a. Crossbite Absent
    b. Crossbite Unilateral
    c. Crossbite Bilateral
    d. Edge to edge

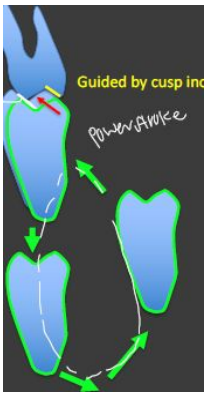
Unilateral	**Bilateral**	**Edge to Edge**
		

8. Arch shapes
    a. Parabolic
    b. Hyperbolic → Wider than parabolic
    c. U shaped → parallel sides and slightly curved front
    d. V shaped → Usually have rotated Anterior teeth
    e. Trapezoidal → flat anteriors
    f. Ovoid → molars curve in
    g. Omega → constricted at pre molar
    h. Asymmetrical

Mastication

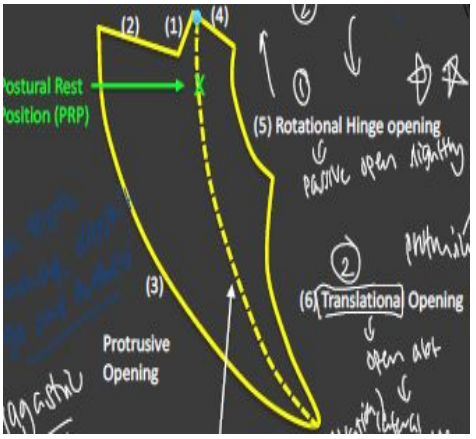
- Mastication occurs in a cyclical tear drop shaped motion, not purely up and down but includes lateral movement
- On the power stroke (Jaw closing) The Md tooth is guided into position by the cuspal incline
- With increasing degree of wear, the broader the tear drop shaped motion = greater lateral excursion

- 



- Border movements of the Md

1	Edge to edge incisal occlusion
2	Protrusion
3	Opening of jaw while Protruded
4	Retrusion
5	Rotational action of TMJ when opening
6	Translational action of TMJ when opening (where the Md moves forward) (occurs after the rotational movement)



3. Oral Ecosystem: Biofilms + Tooth Deposits

Acquired pellicle

1. Structure
    a. Thin coating of glycoproteins → mucins of salivary origin
    b. Found on all surfaces of exposed teeth
    c. Forms on artificial surfaces(restorations)
    d. 2 layers, surface forms first, sub surface forms later→ rich in protein, fills surface defects of enamel
    e. Amorphous(no set pattern, Homogenous (uniform throughout)
    f. Molecules can diffuse through AP into superficial enamel
2. Appearance
    a. Translucent

   b. Stains positive for CHO, Proteins, lipids
3. Formation
   a. Spontaneous
   b. Selective adsorption of salivary glycoproteins, Ca ions interact with GP via electrical charges
   c. Forms within seconds, but takes a week to mature
4. Functions
   a. Reservoir of ions, Ca, PO4, F → remin
   b. Semi permeable membrane allows for ion exchange in remin/demin
   c. Restricts access of acid to teeth in minor acid exposure
   d. Lubricant
   e. Antibacterial → Lysozyme, IgA
      i. Highest titre of IgA in body
5. **KEY TAKEAWAYS**
   a. Semi permeable membrane
   b. Protects teeth from acid and wear
   c. Reforms immediately after removal → remove and have good Moisture control when bonding adhesive materials
   d. Influences biofilm formation

Dental Plaque → soft, unmineralised deposit, forms on teeth/prosthetics when not adequately cleaned

1. Structure
   a. Matrix enclosed bacterial population, adherant to each other and tooth surface, an ecological community that evolved to permit survival of whole community
   b. Dental plaque → bacteria + plaque matrix
   c. Heterogenous → not uniform
   d. Bacteria in organised communities, each with own environment
   e. Composition of plaque alters with maturation
2. EPS → extracellular polysaccharides
   a. Sucrose metabolism by bacteria → EPS
   b. Allow bacteria to adhere and aggregate
   c. Source of CHO for bacteria
   d. Thicker, stickier → harder to remove by saliva/water
   e. Gives white colour to plaque
   f. Gelatinous → acids formed in plaque close to tooth surface
3. Maturation of Biofilm
   a. As plaque matures, more resistant to mechanical removal
   b. Materia alba → plaque that can be removed by water
   c. Poor OH → incorporation of food debris, human cells
   d. Prone to staining
   e. Bacterial by-products can trigger inflammatory response of gingival tissues
   f. Type of bacteria > amount of biofilm
4. Detection
   a. Adequate retraction

   b. Good lighting
   c. Dry site
   d. Good vision
   e. Instrument usage → floss IP, Side of probe/explorer, Tri-plaque Gel → dark blue staining = plaque present, light blue = cariogenic plaque
5. Ecological niches → favourable location, sheltered from clearance
   a. Supragingival → gingival margin, malaligned, tilted, rotated, recessed gingiva, IP, occlusal pits/fissures, ortho brackets
   b. Subgingival → thin, restricted by size of suclular pocket
   c. Protected areas
      i. No mechanical clearance by tongue/cheeks
      ii. Hard to clean/access
      iii. Can thicken

Dental Calculus

1. Formation
   a. Low pH, Ca released from amorphous state, reacts with PO4, precipitation → mineralisation of dental plaque
   b. Smoking also increases calculus formation
2. Significance
   a. Rough
   b. Produces ledges/spurs
   c. Creates an ecological niche for bacteria/plaque
3. Removal
   a. Needs to be mechanically removed by dental instruments → ultrasonic scaler/hand scaler
   b. Difficult to remove all calculus
   c. When handling subgingival calculus, risk of dmg to softer root cementum
4. Detection
   a. Supragingival
      i. Occurs on gingival margins
      ii. Opposite ducts of parotid/sublingual papillae
      iii. Yellow-cream, prone to staining
      iv. Rough and chalky
   b. Subgingival
      i. Gingival sulcus
      ii. Ca and PO4 from blood serum/GCF
      iii. Flattened
      iv. Grey-black due to iron in blood
      v. More mineralised than supragingival
   c. Conditions
      i. Dry field
      ii. Good lighting
      iii. Look out for colour differences
      iv. Using side of tip of explorer, feel for catching, roughness → TACTILE SENSITVITY

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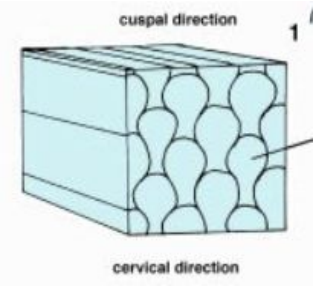
4. Oral Ecosystem: Homeostasis of ecosystem and remin/demin

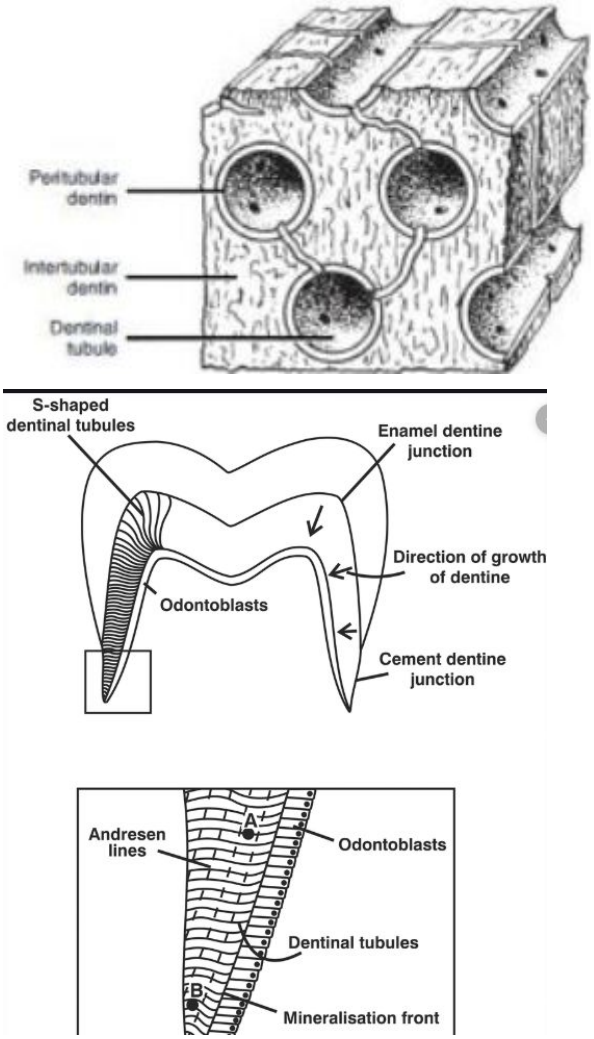
1. Structure of tooth tissues and their clinical significance

a. Evolution of healthy oral environment → historically had biofilm and no OH, however they had balanced diet with low sugar → hence no/little dietary acids → change in diet over time to a high sugar diet → increased dietary acids → increased erosion → Buffer system evolved to buffer dietary acids

b. Enamel → 95% mineral 5% water, some proteins. aprismatic/amorphous surface enamel, followed by Enamel rods. Laminar pores present between rods, H2O (ultrafiltrate) and proteins present in it. Acid enters laminar pores, affecting walls of Enamel Rods.



c. Dentine → 70% inorganic 30% organic. Consists of peritubular dentine(lining tubules which are highly mineralized), intertubular dentine(in between tubules mineralized collagen matrix), and dentinal tubules(contains outward pressure, forcing contents towards enamel, and can close with age). Dentinal tubules exist, with odontoblastic processes and nerve tissue within. Dentinal tubules fuse closer to pulp due to radiating structure (radiate from pulp out to the enamel). When dentine is cut (it is live tissue), pain experienced due to nerve fibres and odontoblastic processes being exposed



d. Tooth Moisture → Water responsible for chemical interactions (remin/demin), Dehydration of tooth → brittle + change in refractive index

e. Anisotropy → Protein and moisture between enamel rods → help to dissipate compressive stress, however not able to dissipate tensile loads (bending) → hence enamel and dentine work together to dissipate compressive and tensile stress

2. Saliva

a. Major salivary glands and respective ducts → Parotid + Stensen's duct → Submandibular + Wharton's duct → Sublingual + gland openings on the sublingual folds

b. Functions of saliva → Lubrication and clearance, Buffering and protection, Maintenance of integrity of tooth, Antibacterial/Fungal/viral, Taste and digestion (LBMAT)

c. How saliva coats teeth → Saliva from parotid papillae coat Mx teeth, Saliva from Sublingual and submandibular pool in buccal, floor of mouth → When mouth is closed tongue moves up creating a suction and bathing all surfaces of teeth in saliva

d. Unstimulated saliva and stimulated → Stimulated saliva triggered by Visual, olfactory, Mechanical stimulation, medications and drugs, pathology

e. Signs of Salivary gland hypofunction
    i. Mucosal dryness and soreness
    ii. Burning sensation of mucosa and tongue
    iii. Dysphonia (can't speak)
    iv. Dysphagia (cannot swallow)
    v. Dysgeusia (Cannot taste)
    vi. Difficulty wearing dentures → not able to form suction
    vii. Thirst
    viii. Dry lips + angular cheilitis (comissures cracked or sore)
    ix. Atypical caries pattern → root surface, smooth surfaces, cusp tips

f. Causes of Hypofunction/hyposalivation
    i. Medications
    ii. Autoimmune diseases → sjogrens disease
    iii. Radiation → radiotherapy
    iv. Dehydration
    v. Age
    vi. Gland stones
    vii. Eating disturbances
    viii. Depression

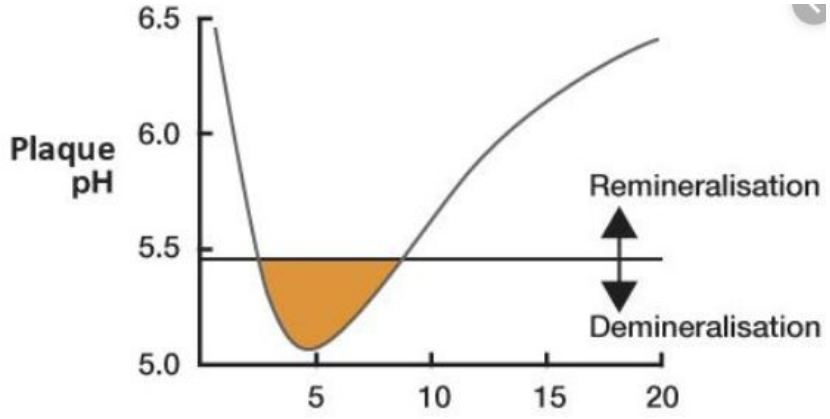
3. Oral environment in Health → Balanced ecosystem

a. Biofilms
    i. 10% human cells, 90% micro-organisms (bacteria)

b. Normal Oral Environment consists of
    i. Biofilm → several days to mature, high biodiversity with a symbiotic relationship with each other, roughly neutral pH. Each community of bacteria in biofilm have differing gradients of O2, nutrients and metabolic byproducts
    ii. Saliva → antibacterial, buffering, contain amorphous Ca2+ and PO4 for remin
    iii. Acquired pellicle →Glycoprotein layer bound to tooth enamel. Ion reservoir of Ca2+/PO4-/OH- bound to statherin in amorphous state
    iv. Tooth enamel

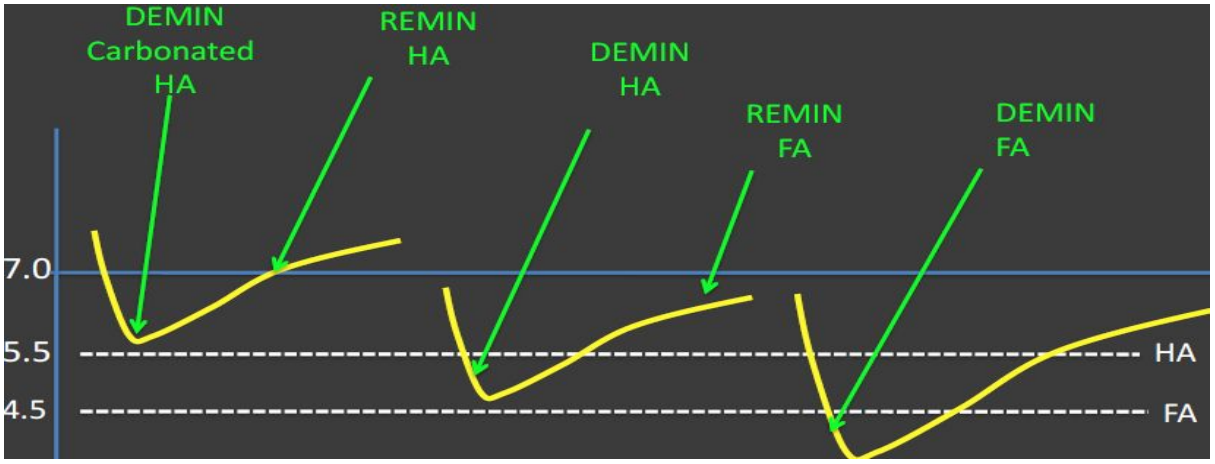
c. Plaque

i. Extracellular polysaccharides (EPS), Mucopolysaccharides, bind to biofilm, making it sticky and prone to staining, has a yellow-creamy appearance
ii. High concentrations of Ions in biofilms
iii. Saliva deposited on top of Biofilms
iv. In thick biofilms, ion transfer from surface to enamel may not be able to occur, requires removal of thick biofilm
v. Forms in ecological niches, places of protection from clearance or hard to reach by saliva, with abundance of food, such as Pits/fissures, interproximally, sub gingival, crowding

d. Good bacteria
    i. Produce ammonia as part of metabolic products, contribute to alkaline environment
    ii. Proliferate in pH band between 6.7 - 7.4
e. Stephan curve
    i. 

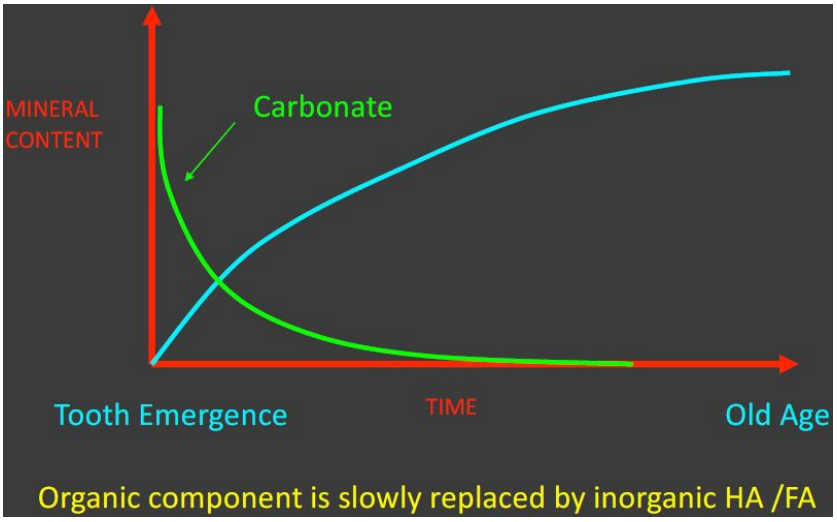
    ii. Acidic food can contribute to decrease in pH
    iii. Metabolic acids produced by oral bacteria can result in decreased pH
    iv. When pH is low, acid can remove statherin, resulting in Ca2+ and PO4- precipitation → formation of calculus
f. Chemistry of Remin/Demin
    i. Hydroxyapatite (HA) → Ca10(PO4)6(OH)2
    ii. In **<u>unsaturated conditions</u>** (low Ca2+/PO4- conc)
        1. Favours backward rxn, where HA breaks down
        2. Occurs in acidic conditions below critical pH
        3. H+ reacts with OH- and PO4- forming H2O and HPO4, along with removal of ions, this favours the backward rxn
        4. Ca2+ can still react with PO4- but forms calculus
    iii. In **<u>Saturated Conditions</u>**
        1. HA at eqm with saliva and oral environment
        2. No loss of HA, as remin=demin
    iv. In **<u>Supersaturated conditions</u>**
        1. Saliva/oral environment supersaturated with Ca2+ and PO4-, favours forward rxn
        2. Results in precipitation → remin occurs

v. Remin
   1. When undergoing remin, presence of impurities (F-,CO3,Mg,Zn) can result in it being incorporated into apatite
   2. All variants except fluorapatite result in a higher KSP/solubility
      a. More soluble → higher critical pH, more easily demin
      b. FA → Ca10(PO4)6F2



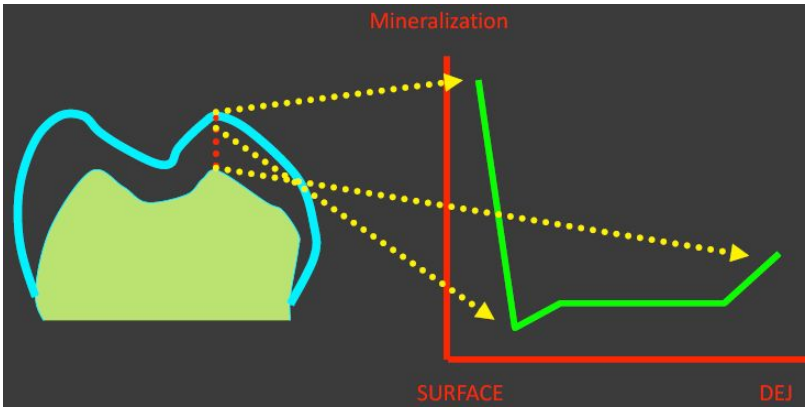
4. Homeostasis and the demin/remin Balance

a. Maturation of Enamel
   i. Newly erupted teeth tend to have more impurities in enamel, namely carbonated HA → Ca10(PO4)5**CO3**(OH)2
   ii. Impurities in enamel are more soluble → demin at a higher critical pH → more prone to caries
   iii. Maturation of enamel starts at surface of enamel
   iv.

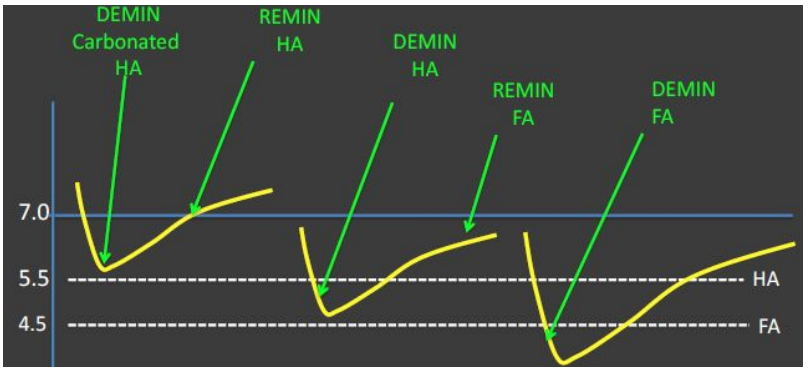


   v. Blue line is Pure, inorganic HA/FA
   vi. As enamel matures from the surface, enamel on surface is most Mineralized, followed by a decrease in the middle and an increase in Mineralization close to the DEJ. This is due to surface enamel exposure to

Saliva and oral environment allowing for demin/remin making it mineralize, and Enamel close to DEJ due to dentinal fluid containing Ions for mineralization

vii. 

b. Demin/Remin Cycle
   i. Has to demin for enamel to mature and strengthen (when it remins), refer to graph below

ii. 

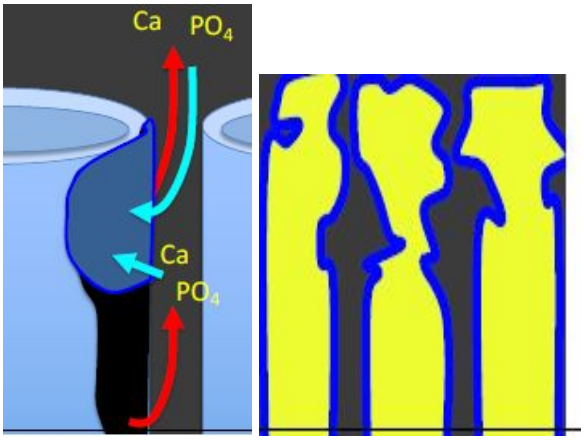
c. Biofilms and Soft Tissue health
   i. Epithelial cells Function as 1) Physical barrier to bacteria 2) Active part of immune system → produce molecules that keep biofilms in check as well as langerhans cells 3) Turnover of Epithelial cells → results in shedding of cells → Dead cells + attached biofilm enter saliva
d. Sialo-Microbial-Dental Complex
   i. Fluctuations in pH → normal
   ii. Demin/Remin Cycle → normal, as long as no net loss of mineral
   iii. Symbiotic relationship between **Biofilms, Saliva, Teeth**
      1. Symbiotic relationship between Biofilm and Host
      2. Symbiotic relationship within communities of biofilm
      3. evolved together, Hence in balance with each other

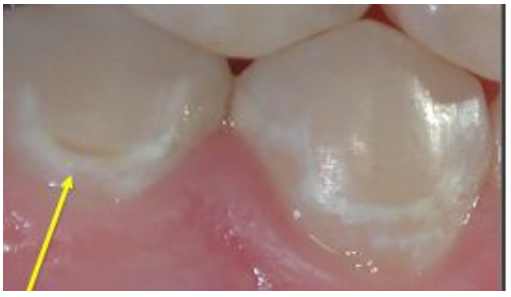
5. Oral environment in Disease → imbalanced ecosystem

a. The development of an imbalanced Ecosystem
   i. Through evolution, we have a changing diet. Currently have a refined, processed, high sugar diet
   ii. Today's diet → Refined soft food(sticks to ecological niches), refined CHO (sucrose), High sugar diets, Fermented foods (acids), high acid intake (soft

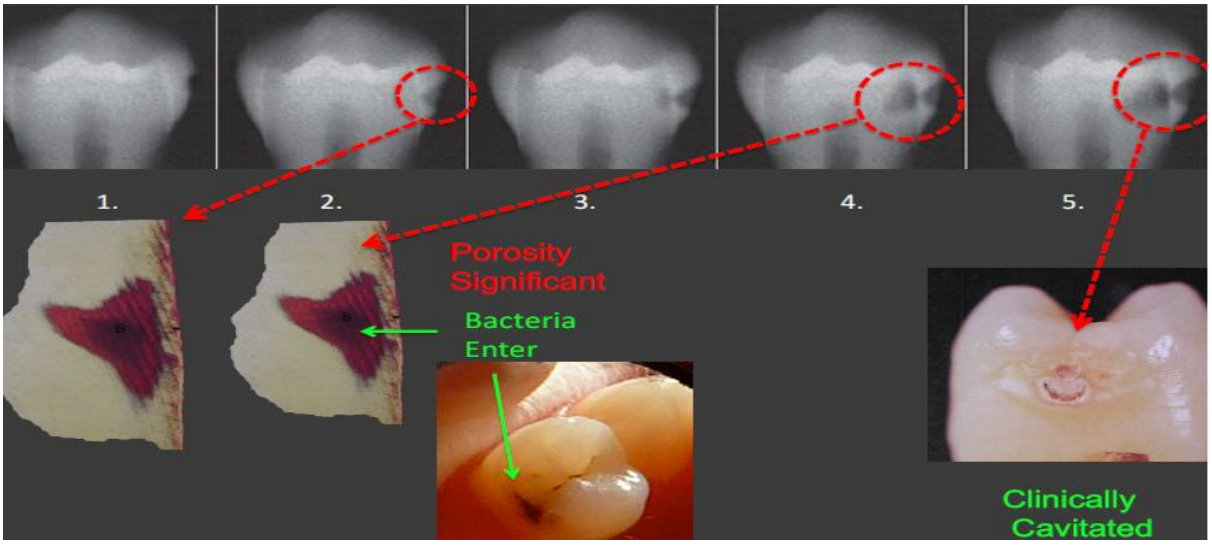
drinks) → Lead to high acid exposure (exogenous and endogenous → stomach acid/bacterial by products) → Caries risk

b. Acidification of the Biofilm
   i. How it occurs → excessive exposure to sugar (allows bacteria to metabolise → produce acid) and soft drink exposure (acidic) → Good bacteria that produce alkalis which maintain neutral pH die off, Acidogenic(producing acid) and aciduric(surviving in acid) bacteria thrive → resting pH becomes acidic (known as **Ecological Plaque hypothesis)**
   ii. When There is a lack of saliva (Hyposalivation/Xerostomia) → lack of buffer system (carbonic acid/bicarbonate, phosphates) and minerals (Ca2+, OH-, PO4-) → uncontrolled acidification of biofilm + Lack of Remin due to lack of available minerals
c. Sub-surface Enamel Lesions
   i. Upon acid exposure, surface enamel is demineralized, following which, acid percolates down laminar pores, reaching sub surface enamel
   ii. As the ions from lower regions escape, it moves upwards, allowing for superior regions to remin + Saliva only reaches the surface of enamel
   iii. Surface enamel undergoes remin but Sub surface portion remains demin → creates Porosities
   iv. Porosities allow for water to take place and light is refracted differently compared to unaffected enamel → appears white when dried
   v.





6. Pathogenesis of Caries → 5 stages

a. 

   i. Acidic waste products from bacteria → Demineralisation of surface enamel
   ii. **<u>Triangular demineralisation</u>** of enamel up to the Dentino-Enamel Junction → appears triangular Due to direction of enamel rods, Remin of surface enamel → subsurface lesion
   iii. Progression of acid **<u>along the DEJ</u>** (ballooning) → follows the path of least resistance, easier to travel along the DEJ as compared to traveling towards pulp hence triangular shape → porosities increase in size till bacteria can enter
   iv. Dentine clearly affected with several zones, Demin/Affected front, Bacterial/Infected Front
   v. Demineralization worsens and a cavitation develops → Clinically cavitated
      1. → clinical cavitation occurs due to the loss of dentine → loss of ability to bear tensile loads → enamel can only bear compressive loads well → enamel breaks → clinical cavitation

b. The various zones of caries (from coronal to apical)
   i. Infected dentine
      1. Demin and staining occurs

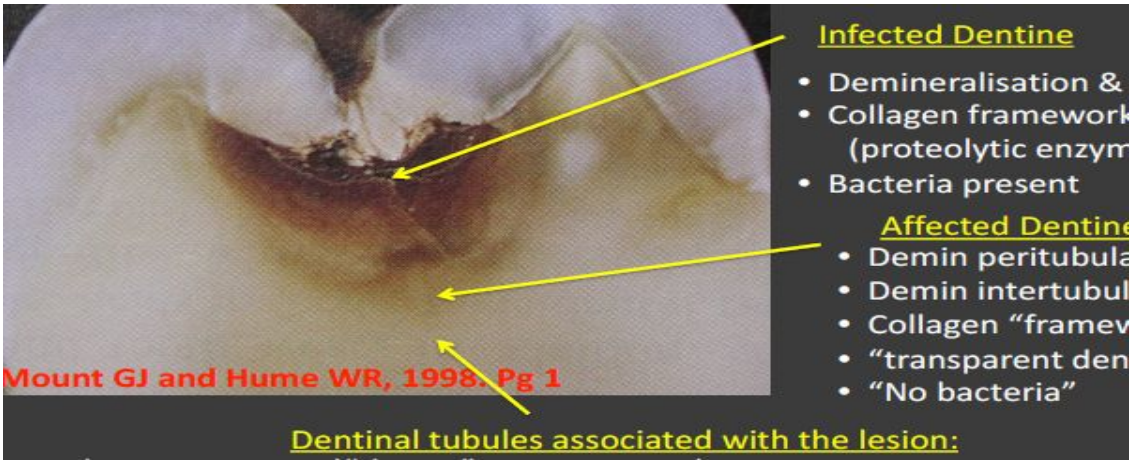
2. Collagen network denatured by proteolytic enzymes
3. Bacteria is present → infected

ii. **Affected dentine**

1. Demin of Peri/intertubular dentine
2. Collagen framework not affected
3. Appears transparent/translucent
4. No bacteria

iii. Associated Dentinal tubules

1. **Sclerotic** (slow caries) → can take several years
    a. Hypermineralisation occurs, Causing dentinal tubules to close → slows progression due to acid not being able to travel as fast
    b. Response to acids and bacterial bi-products
    c. Dentinal tubules closed → Cytoplasmic processes and pulp are isolated from bacterial bi-products and acid → no pain
2. **Dead tracts** (fast caries) → several months
    a. Open dentinal tubules → acid and bacterial byproducts clear out odontoblastic processes → open tubules allow acid to travel through dentine faster → faster progression
    b. Acids and bacterial byproducts have access to pulp → Can be painful

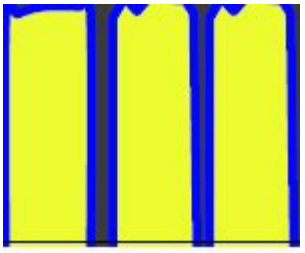
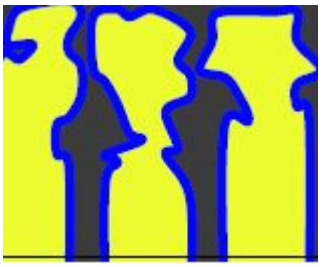


7. Corrosion/Erosion

a. Acid exposure → dissolves and removes biofilm, acquired pellicle → hence it removes any ion reservoir or buffer system available
b. Acid now in direct contact with tooth surface enamel (~pH 2.5 for coke) → as pH of acid is below critical pH → demineralisation of enamel occurs + unsaturated conditions, undergoes immediate demin
c. Demineralisation occurs → loss of ions to acid → acid is now swallowed → loss of ions
d. With the lack of ions → teeth cannot undergo remin process → remain demin → **KNOWN AS AN OPEN SYSTEM DUE TO LOSS OF IONS**
e. Appear as scooped or dished lesions, NOT AS WSL → **appears scooped due to differential rates of progression of acid through Enamel and dentine,**

progresses faster through dentine as less mineral content as well as the cusp and trough shape

f. Image below shows difference in enamel rods, in Corrosion and plaque acid, **Corrosion exhibits loss in rod height (vertical dimension loss) which cannot be gained back**, while **plaque acid exhibits loss in surface enamel which can be gained back except for Subsurface porosities**

g. Corrosion  WSL 

5. Minimal Intervention

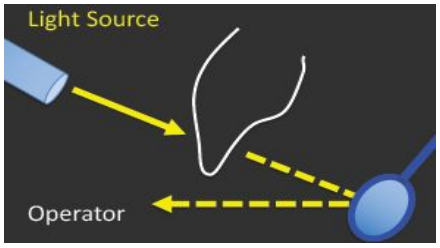
Steps to MI

1. Identify	1. Identification if disease is **present** 2. Identification if disease is **active** a. Arrested caries tends to be black (not absolute, could still be active) b. Active caries → stains should not exist as acid dissolves stains 3. Why does problem exist → risk factors of Pt
2. Prevent	4. Focus on tackling PT's risk factors → incite change in the oral ecosystem → stop disease
3. Heal	5. Remineralisation
4. Monitor	6. Monitor remineralisation (Pt's and your responsibility) → recall needs to be implemented into treatment plan

Identification of caries

- Visual
  - Mirror, air, explorer
  - Dry tooth using air
  - Use mirror for light/vision
  - Explorer for tactile sensitivity to sense changes in tooth structure/surface → do not jab and poke → can cause damage to WSL → not able to remin
  - Used for WSL/Cavitated lesions/Restorations done in last 2-3 years(detect overhangs or secondary caries)
    - If WSL can be seen through saliva → advanced WSL, almost through enamel

        - Sudden need for restorations → indicate change in lifestyle factors

- Transillumination
    - Fibre optic light/overhead light, Mirror
    - 

    - Transmission of light through teeth makes caries appear as shadowing/change in colour
    - Can only be used on anterior teeth as posterior teeth are too thick → no through transmission of light through teeth → little diagnostic ability unless serious caries

- Bitewing radiographs adjunct with clinical examination
    - Clinical examination with suspected areas/risk factors
    - Usually used for Interproximal caries (not so much for occlusal)
    - BW taken to aid/confirm diagnosis

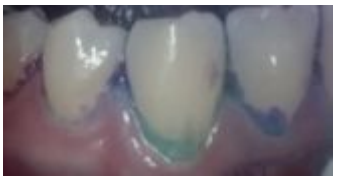
- Electronic caries detector
    - Laser emission on tooth and sensor picks up reflection
    - Reference number on Non cariogenic tooth → Cariogenic teeth have different reading

<u>Identification of risk factors</u>

Saliva	• Saliva acts as a buffer for acids → PO4-/HCO3- • Ion reservoir for remin → closed system • Lack of saliva → lack of buffering capacity/Ion supply for Remin → caries risk
High sugar diet	• High refined sugar diet(usually acidic) → **<u>BAD</u>** bacteria in biofilm proliferate (aciduric/acidogenic) + Good bacteria die → acidic/cariogenic biofilm → Bacterial byproducts - plaque acid demin tooth → caries risk
High acid diet	• High acid diet → removal of Biofilm+acquired pellicle → removal of ion reservoir → Direct exposure of acid to teeth → demin → loss of ions to acid which is ingested → Open system + no chance to remin due to low pH and lack of Ions available for remin → Erosion

- Testing the health of biofilm
    - Tri-plaque ID Gel →

Red	Immature biofilm
Dark blue/purple	Mature biofilm
Light blue	Cariogenic biofilm pH <4.5

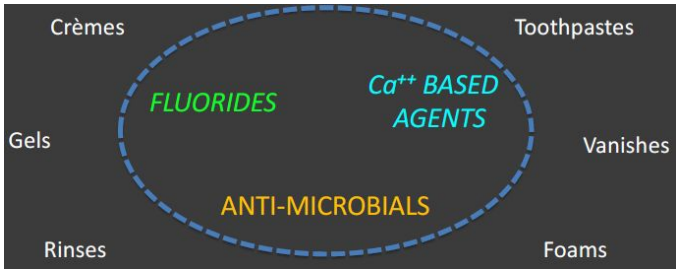
-  

- Saliva
    - Suspected issue
        - Root caries
        - Cusp tip caries
        - Dry mucosal tissues
        - Dry/cracked lips
        - Pt complain of dry mouth
    - Things to know
        - Medications → if Pt is on a lot of meds → likely to have impaired saliva flow
        - Medical conditions → sjogren's syndrome
        - Radiotherapy → xerostomia
    - Conduct saliva tests
        - Saliva check buffer
        - Do subsequent visit to see consistency

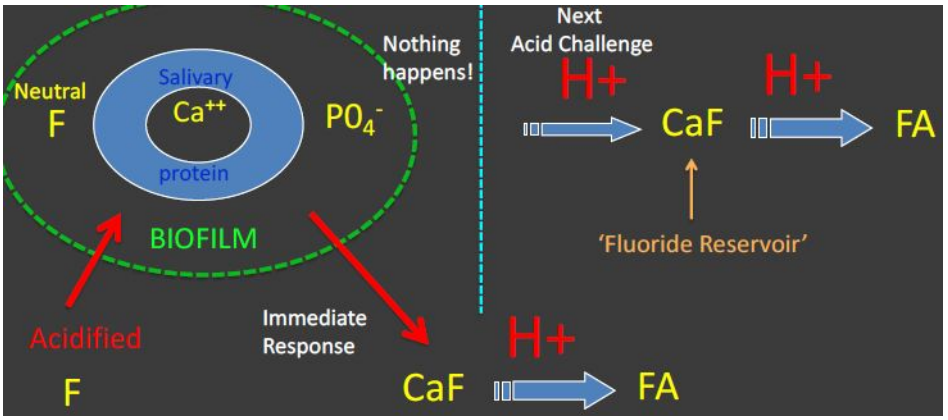
- Lifestyle change
    - E.g death of family member/change of house/migration
    - With a lifestyle change, there is an interval before disease (time for disease to take action)
    - From Good OH → lifestyle change (e.g change in diet) → Cariogenic biofilm ← Secondary risk factors (poor OH/fluoride history/occlusion-crowding/morphology)

<u>Preventive measures and Healing</u>

- Patient acceptance → pt must
    - Understand their problem → explain in layman terms
    - Understand consequences if problem is not addressed
    - Understand how you (dentist) can help

- Multitude of available products for treatment → ask pt how they would prefer to manage problem (Pt motivation) → accounting for beliefs and preferences
- 

- Fluoride
  - Enhancing remin + inhibition of Demin
    - Demin tooth surface → Super saturated conditions promote FA formation, remin can also occur at a lower pH than remin of HA → remin occurs → FA formed is more resistant to demin due to lower critical pH → inhibiting demin
    - Healthy tooth surface → Super saturated conditions prevent demin
  - Effects on bacteria
    - Bactericidal effects at high concentrations → F inhibits metabolism of bacteria/membrane inhibition
    - Bacteriostatic effects at low concentrations → Bacterial metabolism slowed → less acid produced
  - Different forms of Fluoride
    - NaF
    - Amine F
    - APF → acidulated phosphate fluoride ~pH 3.0
      - Good for PTs with xerostomia/hyposalivation → Acid demins tooth → available Ca2+ ions for remin + F → formation of FA /CaF2
    - SnF → stannous fluoride → antibacterial + stain
    - AgF → Silver Fluoride → Antibacterial + stain
  - Mechanism of Fluoride action
    - Neutral fluoride → Fluoride present in biofilm → on acid exposure, formation of CaF2 (ion reservoir for slow release of F) → subsequent exposure to acid, formation of FA
    - Acidulated fluoride (APF) → Acidic environment allows for formation of CaF2 → CaF2 forms FA immediately due to acidic environment
    - Only need to remove biofilm if biofilm is too thick, such that it prevents ions from reaching tooth surface



- Calcium products
  - Usually present as CPP-ACP/ACPF
  - CPP → casein phosphopeptide keeps Calcium in amorphous state → prevents ppt formation
  - High concentration of product → high osmotic pressure → drawn in by plaque fluid and enters carious lesion → subsequent acid exposure removes CPP and make Ca2+ available for remin
    - With F in product → FA can form
  - Clinpro tooth creme → Layer protecting Ca2+ dissolves on contact with mouth → makes Ca2+ available for remin
  - Effective in Xerostomic conditions due to lack of saliva hence lack of Ca2+

- Antibacterial product function
  - Inhibition of biofilm growth
  - Reduce number of bacteria → kills good bacteria too
  - Reduced virulence factor
  - Reduces acid and plaque formation by bacteria
- Antibacterial Products
  - Chlorhexidine
    - Long period of action
    - Absorbed by biofilms/tooth surfaces → can stain
    - 0.2% → bactericidal
    - 0.02% → Bacteriostatic
  - Sugar alcohols
    - Xylitol → when metabolised by bacteria, does not produce acid
  - Fluoride
    - As stated above

- Steps to take to re-establish healthy oral environment → need to change ecology of biofilm
  1. Increase pH
  2. Anti bacterial products

    a. Chlorhexidine
    b. gum
    c. Fluoride products → limited by amount of Ca, if excess F, it is just ingested
3. Improve OH → customise based on PT's preferences and current practice
4. Change diet → hardest to achieve, reduce sugar/acid intake
5. Sealing of cavities → reduce bacterial load
6. Improve saliva
    a. Hydration
    b. Products which contain Ca/PO4-/F → promote remin
    c. Gum → xylitol + stimulate saliva flow
    d. Salivary substitutes

- In erosion → Remin → place barrier on tooth (FS/SnF/Varnish)