

GROUP & INDIVIDUAL LEARNING Exam Gil

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Gen info

- Read through the FAQs! (link at the end)
- Read through the Generic BDS1-5 link
- No set reading time
- If you think there is an error, note it down and work on the following question
- If there is an issue during the exam call the Online Exam support line +61 8 83133311
- Before the exam commences take note of the deadline and set a timer 5 or 10 mins before the end of the exam just to keep you on track

Treatment Planning

Treatment plan (what you want to do) vs Management plan (what you and pnt decided to do)

- CC
- Additional Tests
- Patient Education (not OHI)
- Preventative Tx
- Periodontal Tx
- Restorative TX
- Referral (complex Tx beyond scope)
- Review / Recall (long and short term)

Amalgam:

- Old school metallic material with greatest longevity potential of all current materials
- Moisture control not required (however may be for liners if used.....)
- Macro-mechanical retention (ability to use pins, <u>bonded amalgams</u> or retentive features for greater retention)
- Components: Liquid Hg and metal alloy mixture (Ag + Sn + Cu + others)
 - Zn (prevent material oxidation during manufacturing and prevents corrosion)
 - Indium (increase strength and creep reduction)
 - Others
- High Cu amalgams greater material properties
- Potential for mercury toxicity / hypersensitivity extremely low however popular population sentiment

Amalgam:

- Requirements:
 - 2 mm all dimensions (no thin coatings), no unsupported enamel, non aesthetics (aesthetics zone = 5-5), pulp proximity?
- Sim Clinic Use:
 - \circ High copper long setting type, 8 seconds tritute, 8-10 minute setting time*
 - Final setting takes place over 12-24 hrs (polishing protocol?)

• Setting Reaction:

• Ag3Sn + Hg = Ag2Hg3 + SnxHg + Ag3Sn Y (Strongest) Y1 (2nd strong.) Y2 (Weakest) Y

Amalgam chemical PO reactions:

- Electrochemical corrosion: Phase changes
- Galvanic corrosion: When in contact with metallic restorations
- Crevice Corrosion: Limited corrosion beneficial (reduced microleakage) unpolished surfaces leads to too much
- Stress corrosion: High stress areas leads to corrosion and reduced mechanical properties

RC:

• Components:

- Resin based oligomer matrix (BisGMA, UDMA)
- Inorganic filler: Silica
- Coupling agent
- Initiator
- Setting reaction:
 - Light cure activation free radicals produce which bind together monomers to chain polymers polymerization shrinkage (c - factor)
- Types:
 - Flowable: Reduced filler content with low viscosity
 - Bulk Fill: Additional initiators, reduced filler content with increased particle size, increasing cureable depth
 - Adhesive: No filler (or minimal)

GIC / RMGIC:

- Melbourne invention; limited use in North America
- True chemical bonding to underlying tooth structure (ion exchange forming hybrid layer)
- Components:
 - Polyalkenoid acid, glass and water
 - Glass: Silica, Alumina, Fluoride (Alumina and Calcium forms), Aluminum phosphate, trace elements
 - Chelating Additives: Tartaric acid and Citric acid delays setting reaction
- Setting Reaction:
 - GIC: Acid base reaction:
 - 1). Polyacid reacts with glass particle surface to release ionic components
 - 2). Ions incorporated into polyacid solution forming ionic crosslinks
 - 3). Post maturation of matrix continues to set

GIC / RMGIC:

- Role of water:
 - Solvent for polyalkenoid acid
 - Unbound water at material surface lost after initial placements presents as 'chalky' appearance with microscopic cracks (protect with vaseline or adhesive layer)
 - Within 1 month post placement water content increases with increasing strength and translucency associated

• Sim clinic types:

- Fugi II: RMGIC; good strength and FI release
- Fugu VII: (Dual cure) Most FI release, low strength, commonly used for erupting teeth with poor moisture control
- Fugi IX: High FI release, highest strength, useful for bases (high time setting requirements)
- Fugi Bond LC: Fugi II equivalent with modifiable ratio for liner, adhesive, bonded amalgam

RMGIC:

GIC material that contains RC monomers (HEMA) allowing for light polymerization and generally strengthened material properties

Dual setting reactions for free radical light cure and acid base reaction - light cure predominant form however acid-base form does take place

PRODUCT	IDEAL FOR	GREAT FOR	HANDLING	AESTHETICS	CLINICAL CASES
EQUIA* Forte Fil & Coat (Auto-cure GIC & LC Coat)	Restorations where occlusal wear resistance and aesthetics are priorities	 Occlusal, proximal and cervical restorations Sandwich restorations - closed and open Transitional restorations 	Manipulation time Moisture critical time 2 mm 30 we Finishing time 2 mm 30 we	 Shades: A1, A2, A3, A3.5, B1, B2, B3, C4 Great translucency 	Before After
Fuji* BULK (Auto-cure GIC)	Restorations where acid resistance, protection of surrounding surfaces and speed of bulk cure are priorities	Selected occlusal, proximal and cervical restorations Sandwich restorations - closed and open Transitional restorations	Manipulation time Moisture critical time 2 mo Finishing time 2 min	1 shade (A3 opaque)High opacity	Before After
Fuji* VIII op (Auto-cure, resin-reinforced GIC)	Cervical and sandwich technique restorations where acid resistance, fracture toughness and speed of placement are priorities	 Sandwich restorations - closed and open Cervical restorations Transitional restorations 	Manipulation time Moisture critical time J mm Finishing time 6 min	 Shades: A2, A3, A3.5 Very good translucency 	Before After Af
Fuji* II LC (Tri-cure, resin-reinforced GIC)	Cervical and sandwich technique restorations where acid resistance, fracture toughness and speed of set are priorities	 Sandwich restorations - closed and open Cervical restorations Transitional restorations 	Manipulation time Moisture critical time (Light-cure) 70 sec Finishing time 20 sec	 Shades: A1, A2, A3, A3.5, A4, B2, B3, B4, C2, C4, D2 Excellent translucency 	Before After
Fuji* VII EP/Fuji* VII (Auto-cure GIC)	Protection of "at-risk" surfaces, including erupting molars (fissure protection) and exposed root surfaces, and for caries stabilisation	Fissure protection Protection of root surfaces Endo access Caries stabilisation	Manipulation time Moisture critical time I min Finishing time Amin	 Shades: White and Pink High opacity 	Before After
Fuji* IX or EXTRA (Auto-cure GIC)	General purpose "all rounder" GIC where aesthetics are a priority	Selected occlusal, proximal and cervical restorations Sandwich restorations - closed Transitional restorations	Manipulation time Moisture critical time 2 min 30 sec Finishing time 2 min 30 sec	 Shades: A2, A3, A3.5, B1, B3, C4 Excellent translucency 	Before After
Fuji* IX @ FAST (Auto-cure GIC)	General purpose "all rounder" GIC	Selected occlusal, proximal and cervical restorations Sandwich restorations - closed Transitional restorations	Manipulation time Moisture critical time J min Finishing time J min	 Shades: A2, A3, A3.5, B2, B3, C4 	Before After

Material Indications

	Moisture Control Required	Mechanical Properties	Cavity Preparation	Bonding Type	Biocompatibility	Aesthetics	Additional Contraindicatio ns	Additional Indications
Resin Composite	High	Good	Conservative	Micromechanical	Poor	Good		
GIC	Low	Variable (type dependant), poor wear resistance	Conservative	Chemical ionic exchange	Good	Fair	Low pH, poor wear resistance, Xerostomia	Erupting teeth, limited pnt compliance, Remineralizing capabilities (reduce bacterial load), Asthma
RMGIC	Fair	Fair (type dependant), poor wear resistance	Conservative	Chemical ionic exchange	Fair	Fair	Low pH, Xerostomia	Remineralizing capabilities (reduce bacterial load)
Amalgam	Nil	Very good	2mm all dimensions, non conservative, no unsupported enamel	Macromechanical	Good	Poor		

Material Bonding

- Macromechanical: retention and resistance forms required, no unsupported enamel (amalgam)
- Micromechanical: Tooth structure demineralization allowing for adhesive resin infiltration and 'resin tags'. Allows chemical adhesive layer bonding to CR material
- Chemical: Ion exchange layer forming hybrid layer between two materials

- Etching: 37% orthophosphoric acid (10-15 secs enamel, 5 secs dentine)
- Conditioning: 20% polyacrylic acid for 10 secs

Direct Dentine Bonding

Dry Bonding

 Etch D 5 secs / E 15 secs, wash, dry thoroughly, apply HEMA primer to E / D, air dry, adhesive resin over E / D, light cure 20 secs, 2m CR increments

Etching dentine collapses dentine structure, HEMA based primer flows into tubules and breaks H peptide bonds -'rehydrating' - allows for adhesive bonding

Wet Bonding

 Etch D, wash, dry (dont dessicate), apply acetone based primer, air dry 10 secs, etch enamel, wash, dry thoroughly, apply adhesive resin over E / D, light cure 20 secs, 2mm CR increments

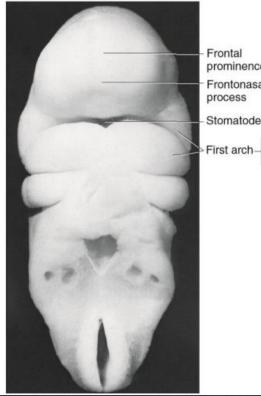
Theoretically keeps some moisture between collagen fibres thus does not collapse, acetone primer chases away water and adhesive then bonds to remaining structure Embryology

Branchial Arches and Grooves

TABLE 3-1

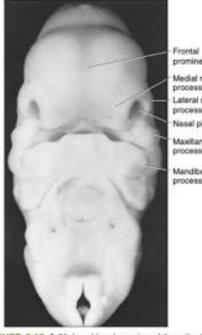
Derivatives of the Branchial (Pharyngeal) Arch System

	ARCH	GROOVE	POUCH
First	 Mandible and maxilla Meckel's cartilage: Incus and malleus of inner ear Sphenomalleolar ligament Sphenomandibular ligament 	1. External auditory meatus	 Tympanic membrane Tympanic cavity Mastoid antrum Eustachian tube
Second	 Reichert's cartilage: Styloid process of temporal bone Stylohyoid ligament Lesser horns of the hyoid bone Upper part of the body of the hyoid bone 	Obliterated by the down- growth of the second arch	 Largely obliterated Contributes to tonsil
Third	 Lower part of the body of the hyoid bone Greater horns of the hyoid bone 		Inferior parathyroid gland Thymus
Fourth	1. Cartilages of the larynx		Superior parathyroid gland Ultimobranchial body
Fifth	Transient	Transient	Transient
Sixth	Transient	Transient	Transient



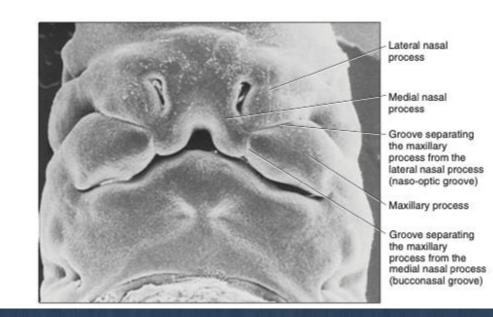
- Frontal prominence Frontonasal process Stomatodeum Maxillary

Mandibular processes



prominence Medial nasal process Lateral nasal process Nasal pit Maxillary process Mandibular process

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.)



What can go wrong?

Genetics (refer to your ILA maps)

Poorly understood, however appears to be associated with signalling molecules

Environment

- Infectious agents (e.g. rubella virus)
- X-ray radiation
- Drugs (nicotine, alcohol)
- Hormones
- Nutritional deficiencies

Cleft Palate

- 1. Failure of the shelves and septum to contact
 - 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 the mesenchyme of the shelves

CLP Treatment

In the 1st year

- Repair of cleft lip and unilateral anterior palate at 3 months
- Repair of cleft palate at 6 months
- Speech, ENT assessment, Dental, Orthodontics

2nd-5th years

- Speech assessment
- Dental review yearly
- If required, soft tissue revision (eg. lip, nose)
- From 5th year onwards, assess the need for nasendoscopy

6th-12th years

- Orthodontics
- Dental
- Possible nasendoscopy. If indicated pharyngoplasty.

13th-18th years

- Orthognathic surgery
- Soft tissue revision

Odontogenesis

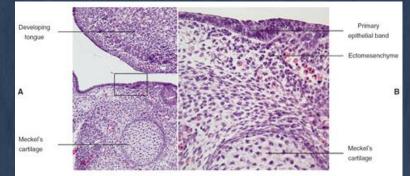
Initiation

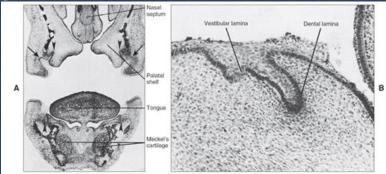
Wk 6: Primary Epithelial Band

• Primitive oral epithelium thickens into underlying mesenchyme

Wk 7: Dental Lamina / Vestibular Lamina

- Dental lamina forms tooth germ
- Vestibular lamina forms vestibular region





Morphogenesis / Histogenesis

Bud Stage:

- Wk 8-10: Enamel Organ forms
- Early Cap Stage:
 - Wk 11: Cap from of EO deep surface forms

Late Cap Stage:

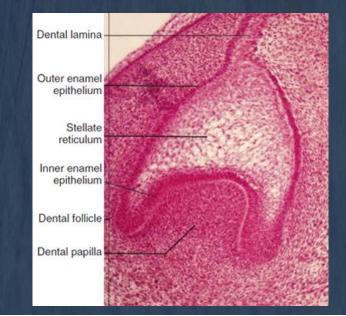
• Wk 12-13: EO begins histogenesis processes

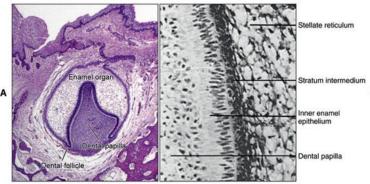
Early Bell Stage:

• Wk 14-16: IEE begins developing crown surface with high histodifferentiation occuring

Late Bell Stage:

• Wk 17-18: Rapid change in size / shape, permanent TG formation

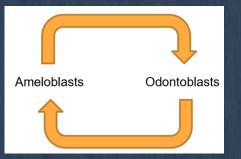




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Amelogenesis

1). Pre-Secretory Stage: Reciprocal Induction

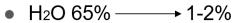


2). Secretory Stage: Ameloblasts secrete initial enamel matrix - initially mineralize immediately

3). Transition: Following initial enamel matrix, ameloblasts retract tomes processes and some undergo apoptosis

4). Maturation: Proteases and emelblasts mature matrix

5). Post Maturation: After eruption - Fluorapatite



● HA 15% -----> 96-98%

Dentinogenesis

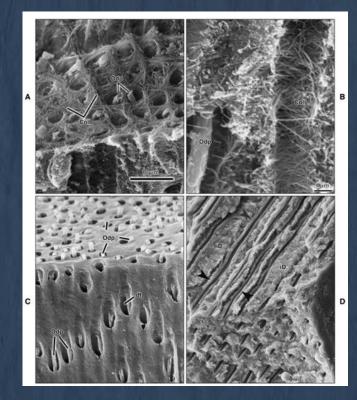
1). Odontoblast Differentiation: Odontoblasts (OD) differentiate with cellular processes towards IEE

2). Matrix Deposition: OD secrete initial matrix which <u>DOES NOT</u> mineralize immediately

3). Matrix Mineralisation: Lags behind deposition, DPP regulate mineralization

4). Peritubular / Secondary Dentine Formation: (both continue with age)

- Peri (intratubular) higher mineral content tubule narrowing
- Secondary (intertubular) pulp shrinkage
- **5). Tertiary Dentine Formation:**
 - Reactive / Reactionary: Mild stimuli, OD survive, tubule dentine
 - Reparative: Severe stimuli, OD die, atubular dentine



Tooth Eruption

Primary Teeth

Calcification pattern: A, D, B, C, E

Eruption pattern: A, B, D, C, E (Also shedding sequence)

Note:

- Teeth erupt when roots approx. ½ formed
- Generally female teeth eruption at younger age than males
- Primary teeth apex remains open during lifecycle

Teeth	А	В	С	D	E
Mx / Md Calcification	Mx before Md	Mx before Md	Md before Mx	Mx before Md	M before Md
Approx. calcification at birth	3/4	1/2	1/3	Cusps unified	Cusps isolated
Approx. age of crown completion	2.5 months	3 months	10 months	6 months	11 months
Approx. age of root completion	18 months	24 months	40 months	30 months	42 months
Approx. eruption age (months)	Mx: 10 Md: 7	Mx: 12 Md: 14	Mx: 20 Md: 20	Mx: 16 Md: 16	Mx: 29 Md: 28

Tooth Eruption

Permanent Teeth

Eruption pattern:

Mx: 6, 1, 2, 4, 3, 5, 7, 8 **Md:** 6, 1, 2, 3, 4, 5, 7, 8

Calcification pattern:

6,1, Mx 2, 3, Md 2, 4, 5, 7, 8

Notes:

- Crown development takes 4 years
- Root development: 4 years (incisors), 7-8 years remainder
- 1st molar: 3 years crown development, 6 years root development
- Teeth begin eruption when roots $\frac{1}{2} \frac{3}{4}$ formed
- Root apex closure indicates complete development

Teeth	Initial	Eruption Time (years)		
	Calcification Time	Мх	Md	
1	3 months	F: 7 M: 7	F: 6 M: 7	
2	Mx: 5 months Md: 1 year	F: 8 M: 9	F: 8 M: 8	
3	5 months	F: 11 M: 12	F: 10 M: 11	
4	1 ½ - 2 ½ years	F: 11 M: 11	F: 11 M: 11	
5	2 1/2 - 3 1/2 years	F: 12 M: 12	F: 12 M: 12	
6	Birth	F: 7 M: 7	F: 6 M: 7	
7	2 1/2 - 3 1/2 years	F: 12 M: 13	F: 12 M: 12	
8	7 – 12 years	NA	NA	

Dental Anomalies

Gemination vs Fusion

- Both appear as large fused tooth
- Gemination = normal tooth count (same roots, extra crown differentiated from TG)
- Fusion = tooth count minus 1 (separate roots, coronal fusion)

Hypoplasia vs Hypomineralisation

• What processes affect these anomalies?

Gingivitis and Periodontitis

Gingivitis - Histology and Clinical Appearance

Assessing	CLINICAL SIGN	HISTOLOGY		
gingivitis:	Colour change to red	Changes in gingival tissues		
gingivilis.		 Proliferation of blood vessels – inflammation 		
Colour				
	Changed contour and	 Tissue oedema (swelling) 		
Contour	consistency	 Inflammation 		
Consistency	Retractability	 Destruction of collagen/gingival fibre network 		
Texture	ВОР	Ulceration of JE		
		 Proliferation of blood vessels 		
Exudate		 Inflammation 		
	Exudate (GCF, pus)	Inflammation- PMNs		

CAL = Clinical attachment loss

- CAL DOES NOT always = Periodontal pocket depth (PPD)
- When CEJ is not detectable and max PPD equal to or less than 3mm, there is no CAL
- When CEJ is detectable on the height of the gingival margin, CAL = PPD
- For teeth with recession the CAL = recession + PPD

Periodontitis - Signs and Symptoms

Signs

- Periodontal pocketing
- Recession
- CAL
- Evidence of radiographic bone loss
- Increased mobility

Symptoms

- Receding gums
- Gaps between teeth
- Increased food debris
- Mobile teeth
- Change in teeth position
- Speech alteration
- Tooth loss
- Pain (acute conditions)

Periodontal Instruments

Sickle Scaler (supragingival calculus)

- Anterior teeth

McCall Scaler (supragingival calculus)

- Posterior teeth
- **Gracey Curettes**
 - 3/4 (anteriors)
 - 7/8 (posteriors true buccal or true lingual)
 - 11/12 (posteriors mesial)
 - 13/14 (posteriors distal)

Ultrasonic (supra and subgingival calculus)

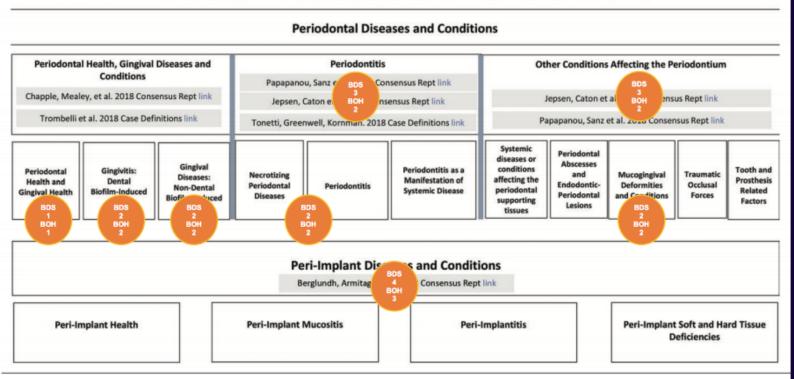
Periodontal Tx - Post op

Post op instruction	Reason
May feel sore gingival tissues post-op and to rinse with warm salty water several times a day if gums sore	During scaling procedure there is usually micro-trauma to the gingival tissues due to the manipulation of the instruments and the removal of the calculus. Normal and expected post- op inflammation that subsides within 24-48 hours. Patient need to be aware is normal response
May have some residual gingival bleeding but this subsides within 24 hours or so	There can be some minor bleeding post-op depending on how much initial inflammation and any underlying medical conditions that may affect clotting. Patient need to be aware is normal response. If prolonged bleeding >24hr to contact clinic as may be issue/other undiagnosed problems
May feel like there are gaps between the teeth. This is normal as thick calculus removed. This sensation should improve in a few days.	With the presence of calculus the interproximal spaces close up and the tongue gets use to this feeling. When calculus is removed then normal anatomy/shapes/contours are 'exposed' again and the tongue will feel this as different and sometimes patients may perceive we have done something wrong. Hence as part of consent and post op we need to remind them
May experience dentine hypersensitivity to cold or sweet foods – this may not occur. If it does, apply toothpaste or toothpaste for sensitive teeth to affected teeth	Depending on initial clinic situation, sometimes when doing subgingival calculus removal on root surface we may remove cementum from the dentine and thus exposed the underlying dentine causing open/patent tubules.
May have gingival recession as part of the healing process– this is difficult to predict if it will occur. Will need to be monitored	Often as part of healing process especially in periodontitis more so than gingivitis, part of healing response is gingival recession. How much occurs and if it occurs is unpredictable. Hence as part of consent and post op we need to inform patient.
Contact the clinic for follow up if any unresolved pain or any concerns.	Impt that patient is aware to contact clinic if have any concerns – part of good patient management and to follow up on patient concerns.

Classification of Periodontal Diseases

TABLE 1.

CLASSIFICATION OF PERIODONTAL AND PERI-IMPLANT DISEASES AND CONDITIONS 2017



Periodontitis Staging

PERIODONTITIS: STAGING

Staging intends to classify the severity and extent of a patient's disease based on the measurable amount of destroyed and/or damaged tissue as a result of periodontitis and to assess the specific factors that may attribute to the complexity of long-term case management.

Initial stage should be determined using clinical attachment loss (CAL). If CAL is not available, radiographic bone loss (RBL) should be used. Tooth loss due to periodontitis may modify stage definition. One or more complexity factors may shift the stage to a higher level. See **perio.org/2017wwdc** for additional information.

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

Periodontitis Grading

PERIODONTITIS: GRADING

Grading aims to indicate the rate of periodontitis progression, responsiveness to standard therapy, and potential impact on systemic health. Clinicians should initially assume grade B disease and seek specific evidence to shift to grade A or C. See perio.org/2017wwdc for additional information.

	Progression		Grade A: Slow rate	Grade B: Moderate rate	Grade C: Rapid rate
Primary criteria Whenever available, direct evidence should be used.	Direct evidence of progression	Radiographic bone loss or CAL	No loss over 5 years	<2 mm over 5 years	≥2 mm over 5 years
	Indirect evidence of progression	% bone loss / age	<0.25	0.25 to 1.0	>1.0
		Case phenotype	Heavy biofilm deposits with low levels of destruction	Destruction commensurate with biofilm deposits	Destruction exceeds expectations given biofilm deposits; specific clinical patterns suggestive of periods of rapid progression and/or early onset disease
Grade modifiers	Risk factors	Smoking	Non-smoker	<10 cigarettes/day	≥10 cigarettes/day
		Diabetes	Normoglycemic/no diagnosis of diabetes	HbA1c <7.0% in patients with diabetes	HbA1c ≥7.0% in patients with diabetes

The 2017 World Workshop on the Classification of Periodontal and Peri-Implant Diseases and Conditions was co-presented by the American Academy of Periodontology (AAP) and the European Federation of Periodontology (EFP).

Endocrine – Diabetic Patients

Uncontrolled Diabetes

- Uncontrolled glucagon release (no insulin) \rightarrow BG levels rise \rightarrow hyperglycemia
- Cells lack glucose entry due to lack of insulin (esp skeletal muscle cells with GLUT 4 transporters) → cells have no energy resource
- Fatty acids are used to produce ketone bodies for energy \rightarrow blood pH falls \rightarrow ketoacidosis \rightarrow syncope

Controlled Diabetics

- High initial levels of insulin \rightarrow drop in BG levels
- IF not managed correctly (i.e. eating breakfast in the morning) → BG levels continue to fall, unopposed → hypoglycemia
- Low BG → lack of glucose energy to transport oxygen → lack of oxygen transport to the brain → hypoxia

Managing Emergencies with Diabetic Patients

If patient is conscious:

- Cease dental treatment
- Give 20-25 g of glucose
- Followed by lower glycemic load carbohydrate meal
- Keep patient under watch, do not allow patient to drive home

If patient is unconscious:

- Cease dental treatment
- Call 000
- Begin instituting basic life support (DRSABCD)

Effects of Medications

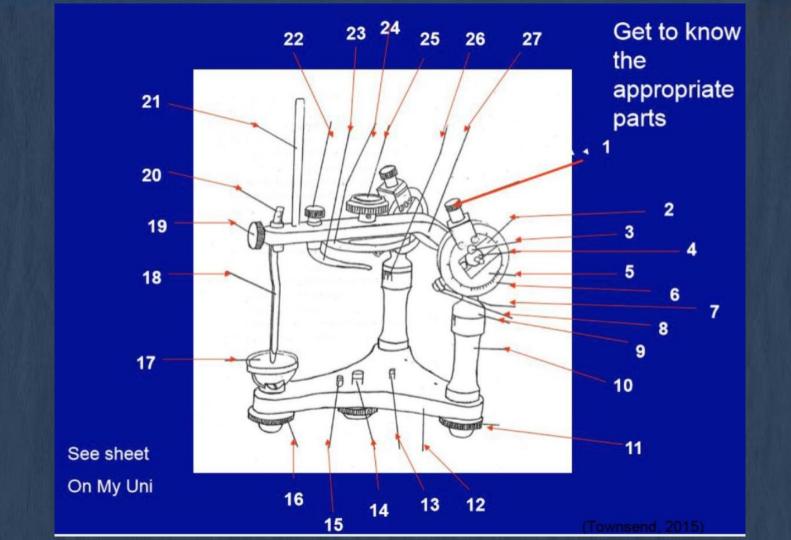
Diuretics:

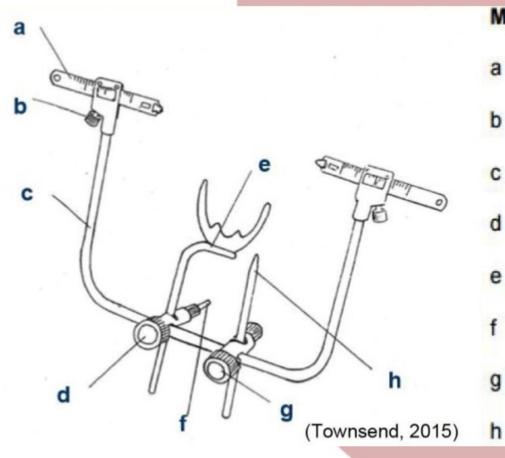
- Treat BP by elevating urine production and fluid loss
- Potassium: diuretics lead to a deficiency in potassium (hypokalemia)
- Saliva: increase in water loss is associated with a reduction in mean salivary flow rate \rightarrow xerostomia
- Lichen planus: Captopril can can induce lichenoid reaction in patients
- Orthostatic hypotension: ACE inhibitors associated with increased chance of OH

Hormonal Changes with Aging

- Menopause \rightarrow estrogen levels fall, progesterone production decreases
- Salivary issues saliva glands contain sex hormone receptors, stimulated by estrogen.
 - \circ Decrease in estrogen \rightarrow decreased salivary flow \rightarrow xerostomia
 - Salivary issues predispose patients to BMS, and reduction in antibacterial function (increased risk of periodontal disease, increased risk of candidal infection)
- Epithelium becomes thinner and more prone to inflammatory changes
- Periodontal issues estrogen receptors present in osteoblasts and fibroblasts of periodontal tissue; bone resorption decreases with higher levels of estrogen
 - Decrease in estrogen is associated with osteoporosis during menopause
 - Resorption of alveolar bone can occur
 - Patients are more susceptible to tooth loss due to loss of attachment

Biology of Occlusion





Main Components of Face-bows

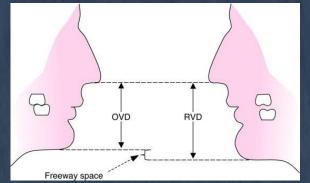
- a Calibrated condylar rod
- b Set screw for calibrated condylar rod
 - Face-bow frame
 - Locking clamp for bite fork
 - Bite fork
 - Anterior jack screw
 - Locking clamp for orbital pointer pin

Orbital pointer pin

Occlusion - Definitions

Bennett movement

- In lateral movement, the side that the md moves towards is called the working side and the condyle on this side is called the working (side) condyle. The working condyle shows a small lateral shift called the Bennett movement.
- Bennett angle
 - The Bennett angle is the angle traced by the non-working side. The angle is measured by the non-working side during lateral movement.
- Working side
 - In lateral movement, it is the side to which the mandible moves
- Balancing side (non-working side)
 - Opposite side to the working side
 - The condyle on the non-working side moves the most, such that it moves downwards, forwards and medially
- Interocclusal distance (IOD or freeway space)
 - IOD = RVD-OVD



Occlusion - Occlusal Exam

Morphology of teeth Attrition, erosion, abrasion Arch shape Rotations/crowding/spacing Angles Canine/Molar Class Midline Overjet (mm) Overbite (%) Crossbite Curve of Spee Curve of Wilson **Axial Inclination**



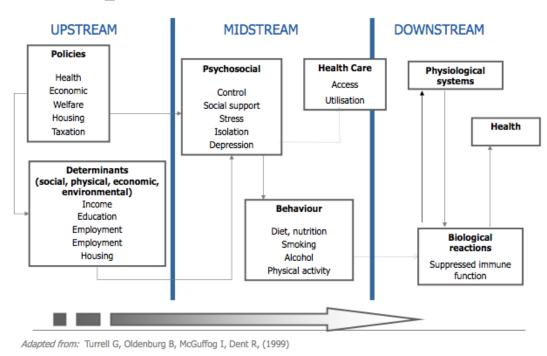
Social Determinants

- Extremely important for health disparities
- Impacts all levels and facets of health
- Especially important for Dentistry
- Pnt, community and geographical specific



PCC

Conceptual framework

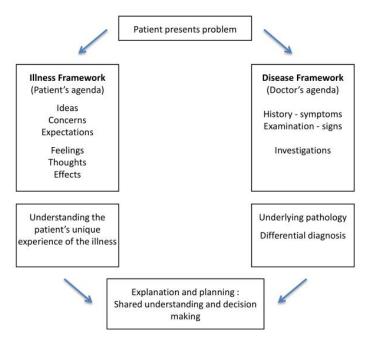


PCC



Disease – Illness Model

Communication Models



PCC - Theoretical explanations of anxiety/fears

1. Behavioural learning theories

- Learning is called 'conditioning'
- Learning involves association with behaviour
 - Classical conditioning = associate an involuntary response and a stimulus
 - Operant conditioning = associate a voluntary behaviour and a consequence
- 1. Social learning theory
 - Learning is the result of observing others
 - Observing leads to imitation
- 1. Cognitive learning theories
 - Learning is the result of thinking and evaluating
 - Thoughts about experiences produce fear and fearful behaviour (cognitive learning)

PCC - TRIM - giving patients information

Timing Relevance Involvement Method

Radiography

Assessing quality of radiographs

- Density
 - Increased mA = increased x-rays generated = increased density
 - Increased exposure time = increased x-rays striking receptor = increased density
- Contrast
 - Increased kV = increased energy = increased x-rays = decreased contrast
- Sharpness
- Horizontal detector placement
- Vertical detector placement
- Horizontal beam angulation
- Vertical beam angulation
- Position of central beam
- Position of rectangular collimator
- Overall diagnostic value of image would you retake? Why?

EBD

P(opulation)I(ntervention)C(comparison)O(utcome)

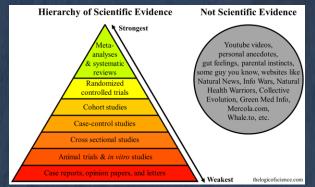
- Hypothesis: Drinking sugar leads to diabetes
- Null Hypothesis: No significant health change during time I drink coke only or pepsi only
- Alternative Hypothesis: Health improves when I drink coke only compared to drinking pepsi only

Reliability:

- Inter examiner error: reliability is determining whether two or more different examiners completing the tests gets the same results on repeated people/ things / etc
- Intra examiner error: reliability is when the same single examiner completing the tests, repeats and whether they get the same result or not

Validity:

- Sensitivity: Accuracy in diagnosis disease
- Specificity: Accuracy in diagnosing health





ILA

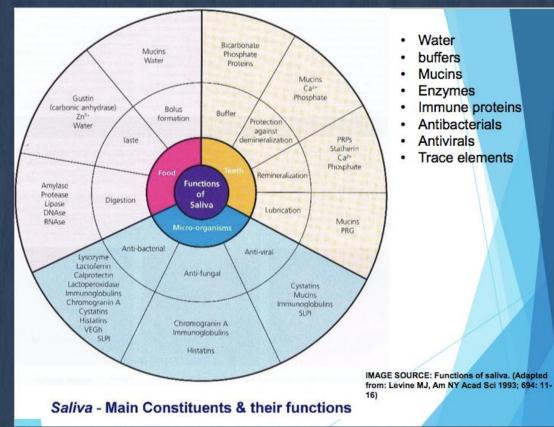
Know the ILAs in depth Be able to answer every single question

MI treatment

Know what these lesions look like clinically and how to distinguish between them:

- Erosion
 - Clean and shiny lesions
 - Scooped appearance
- Attrition
 - Associated on incisal edges (can be seen in posterior teeth too)
- Abrasion
 - Shiny or dull wear facets around the cervical area of the tooth
 - Wedged shaped
- Caries
 - Usually associated with areas of plaque accumulation

MI - Saliva



General info resource

FAQs - <u>https://www.adelaide.edu.au/covid-19/student-information/assessment-and-grades#what-if-i-have-an-issue-with-my-online-exam-should-i-contact-my-course-coordinator</u>