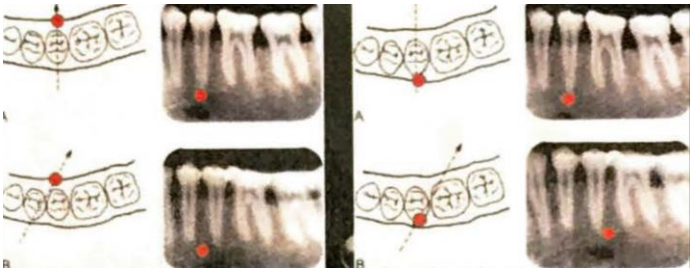
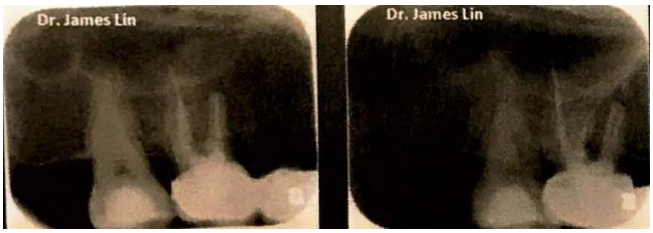
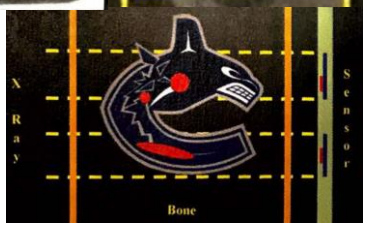
Radiography review

- Standard radiograph series for RCT
  - Pre treatment: 2 PA's (straight + off angle), sinus tract GP tracing, no rubber dam
  - Trial file: working length film with a minimum size 15 K file, add H files if multiple canals
  - Master apical file
  - Master cone
  - Post treatment: 2 PA's (straight + off angle), no rubber dam, access must be restored
- Adjunct radiographs, if needed
  - Bite wings
  - Cone beam
- Radiograph equipment
  - Paralleling: RINN
  - Bisecting: Snap A Ray, hemostat
- SLOB rule
  - "Same lingual, opposite buccal"
  - Object located lingually will move in the same direction as the source of the X ray beam
  - Object located lingually will move in the opposite direction as the source of the X ray beam
- Radiographic assessment criteria
  - Excellent: radiograph shows entire tooth, entire lesion, no distortion, and proper exposure
  - Slight~moderate: 1~2 retakes
  - Serious~critical: 3+ retakes, final PA taken with RD, unauthorized radiographs
- C shaped canal
  - This is a case of a C shaped canal that had a failure after first treatment
  - Since the radiograph is a 2D image, it failed to show the overlapping 2 canals
  - CBCT shows a missed canal
- Endodontic exam and diagnosis
  - Cold, hot, EPT
  - Percussion, palpation, bite test
  - Probing
  - Radiographs
  - Pulpal and periodontal diagnosis
  - Endodontic treatment classification







Standard of practice, case difficulty, referral

- Standard of practice
  - Minimal clinical quality and professional performance of a procedure without regard to the practitioner being a general dentist or specialist
  - 95% healing is expected under ideal conditions
  - Non healing of RCT can be due to misdiagnosis (perio for endo lesion, fracture), errors in treatment planning, or poor case selection
  - When a practitioner is faced with a case beyond their ability, they can:
    - Discuss limitations and ensure understanding from the patient. Receive informed consent and proceed with treatment
      - Does NOT absolve the practitioner from practicing below standard of practice
    - Refer patient for specialist consultation and treatment
    - Upgrade skills to meet standard of practice
- Case assessment
  - Tooth must be deemed restorable prior to treatment
  - Case assessment form can be used to quantify how difficult a case will be
  - Based on patient considerations, tooth considerations, and etc
  - Even if nothing is wrong, mark everything on the form
  - "Average risk" is 15~17 points, which is the only level undergraduates can perform
  - "High risk" is 18~25 points, which would be difficult for an experienced practitioner
  - "Very high risk" is 25+ points, which would be difficult even for the most highly skilled practitioner
- Why refer a patient?
  - Do what's best for the patient
  - Understand operator's capabilities
- Establishing a dentist – specialist relationship
  - Develop a relationship prior to referral
  - Refer in a timely manner
  - Inform patient for rationale for referral
  - Provide initial endodontic diagnosis
  - Indicate previous Txs (O&D, calcified canals, blocked canals, instrument fracture, perforation)
  - Include patient considerations (medical history, gag reflex, limited opening, anxiety)
  - Restorative treatment plan
  - Coordinate final restoration to within 1 month after endodontic treatment to avoid leakage
- If there is an iatrogenic pulp exposure
  - Assess if it is possible to stop the bleeding
  - If vital pulp with good isolation and no bleeding → direct pulp cap
  - Hyperemic → likely irreversible pulpitis already present → RCT or open and drain

Management of calcified canals

- Etiology of canal calcification
  - **Secondary dentin**: physiologic calcification due to aging. Organized dentin
  - **Tertiary dentin**: can be reactionary or reparative. Due to caries, trauma (canal obliteration), parafunction, or cavity preparation. Does not have a nice hydroxyapatite structure
- Calcified canals
  - Appears as faint or non visible canals on radiographs
  - Unable to penetrate the orifice with a size 6, may require dentin removal in coronal 1/3 to access canals
  - Unable to negotiate to WL with a size 10
  - Calcification starts coronally and extends apically
  - May need a drill to remove 2~3mm of dentin, and have a sudden drop to WL with a 10/15 file
  - When there is too much drilling to reach the canals, a surgical approach may be more conservative
- Locating canal orifices
  - Radiographic measurement
  - Laws of pulp chamber anatomy and symmetry
  - Dentin colour and chamber contents
    - Yellow: dentin above the pulp chamber
    - Grey: pulpal floor
    - White: wall of pulp chamber
    - Red: vital pulp tissue OR bleeding from a perforation
  - Anatomical assessment
    - Perio probe around the tooth to verify external anatomy
    - DG16 explorer to find canal orifices
    - 21mm stainless steel K files (stiffer than NiTi) to loosen calcifications
    - Irrigation to remove debris and visualize better
    - Allowing bleach to sit in the chamber will dissolve necrotic tissue and show champagne bubbling from the canal orifice. Useful technique when you're close enough to the canal but cannot find it
  - Microscope
    - Allows better visualization
    - Good for medicolegal documentation during procedure
  - Radiographic orientation
    - Place a radiopaque material (GP or cavit) in the canal, may need to remove rubber dam and temporize
    - Take 2 radiographs and apply SLOB rule
    - See how the current access prep is oriented to the canal

- Once the canal is found
  - Don't immediately pull the file out, may not be able to find it again
  - Gently stem wind the file to resistance
  - Stem wind back 2~3mm then back into the canal
  - File in and out 2~3mm several times
  - Once sufficiently enlarged, move up file sizes and continue until WL is reached
  - Enlarge canal to a size 20 and establish a guide path
  - Irrigants and lubricants
    - Bleach: dissolves organics, helps locate canals (champagne effect), and lubricates
    - EDTA (Prolube, RCPrep, Glyde): can be used once canal is located, as it softens and conditions the dentin. Also removes the smear layer
- Shaping the canal
  - Only use rotary NiTi once a 30 hand file can pass to mid root and a 20 hand file can reach WL
  - Never force the file down, press to resistance
  - MAF should be a minimum of size 30, like a non calcified RCT. Calcified canals are often just on the coronal, so the apical should be treated like a non calcified canal

- Prevention of perforation
    - Know anatomy
    - Probe and check orientation of bur to root angulation before drilling
    - Use low speed, small round burs
    - Must be able to see where the bur is drilling (magnification + lighting), never guess
    - Take films to check orientation
- Management of perforation
    - Disinfect and minimize contamination with a bleach rinse
    - Control bleeding by placing CaOH and pressing the perforation with a cotton pellet
    - Seal perforation with Cavit (temporary), MTA, GI, or composite
    - Make a timely referral
- Successful management depends on
    - **Size**: small perforation with an apex locator or radiograph
    - **Location**: apical has a better prognosis than crestal. Accessibility to seal the perforation is also a factor
    - **Time**: early intervention improves prognosis

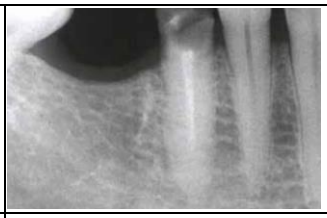
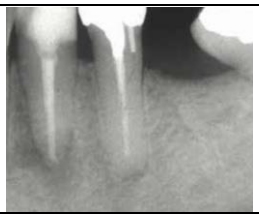
Problem solving in endodontics

Presentation	 	-Injured from a hockey puck -Pt complains of moderate pain
Diagnosis	-Lateral luxation and extrusion of 15 -Alveolar fracture	
Treatment	 	-Anesthetize the patient -Push tooth back into socket -Need to use lots of force to push away all the blood that has clotted at the apex of the tooth -This tooth has a closed apex, so all pulp tissues would be severed when pushed back → need to do RCT

Presentation	 	-21 avulsed for 3 hours: 30 mins in paper towel, 10 mins in milk, then in saline -Tooth replanted and splinted at hospital -11 + 21 not responsive to cold and palpation sensitive
Diagnosis	-21 linguoverted (lateral luxation), extruded, and has a history of avulsion -11 also linoverted (lateral luxation), but to a lesser extent	
Treatment		21: -RCT and splint for 2 weeks -Leaving tooth untreated would risk external root resorption 11: -Might be vital, but could take 1 year before cold response comes back -EPT would be the first test that would show vitality after trauma -Tooth turned out to be symptomatic, so was RCTed

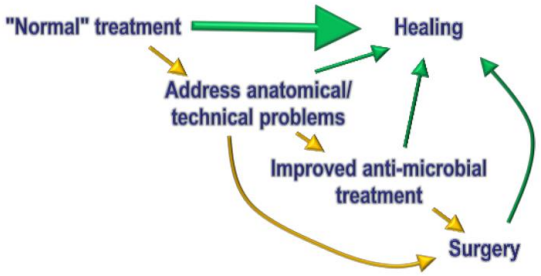
Presentation	  	-Swelling and discharge in the left nostril -No maxillary teeth sensitive to maxillary anteriors -All teeth respond to cold testing, but 11 and 21 are delayed
Diagnosis	-No PARL, teeth all respond to cold testing -Calcified canals may be the reason behind delayed cold response	
Treatment	-Refer back to ENT as pus is not dental origin	

Presentation		-26 sore to biting -Painful to percussion and bite -No response in EPT -Sinus tract on 26B -12mm and 9mm pocket on DL -Patient has cirrhosis of the liver
Diagnosis	-Necrosis with extensive bone loss	
Treatment	-Extraction -Liver cirrhosis → inability to metabolize lidocaine, carbocaine, articaine → could die with a few carps -Give ½ carp PSA, few drops on palate, few drops on buccal	

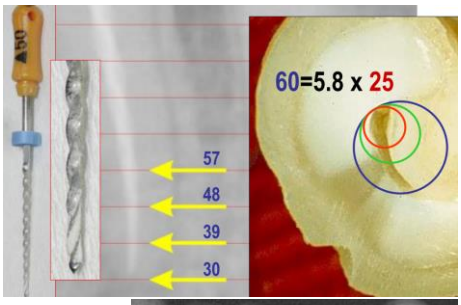
Presentation	 	-35 RCT with apical radiolucency -45 RCT done a year ago, but presented with a broken tooth
Diagnosis	-35 has a leaky restoration and is causing apical periodontitis -45 possible leakage	
Treatment	-Redo 35, 45 may need retx as well	

-Stopped here because I don't see how any of this is new, see slides for other random cases

Persistent endodontic infections



- Reasons for seeing a radiolucency on a root canal treated tooth
  - Healing in progress
  - Persistent infection
  - New 1 lesion
  - New 2 lesion
  - To diagnose what the reason is, must look at when tooth was treated, the symptoms, and the quality of the RCT
- Principles of long term success of RCTs
  - Eliminate infection
  - Prevent reinfection
  - Maintain or regain strong tooth structure
- Importance of irrigation
  - Mesial canals in molars and premolars have flattened canals, like seen in the picture
  - Debriding the whole canal with a file to make it round will remove excess dentin
  - 60 file has 5.8x the cross sectional area as a 25 file
  - Highlights the importance of irrigation, as it conserves tooth structure but also removes organic tissues



- 3 key things to prevent reinfection
  - 1. Correct length of filling, correct apical size, good filling
  - 2. 1~2mm of GP is removed coronally and placed with composite, GIC, or IRM to secure and seal off the root filling
  - 3. Long term success is better with a crown on molars
- Prognoses
  - Primary cases: 80~95%
  - Retreatment cases: 60~85%
  - 40% of endo treated teeth with root fillings will still have a lesion
- Reasons for persisting infections
  - **Biofilm in the canal**: poor quality of Tx, complex anatomy preventing removal, resistant microbes (E. faecalis)
  - **Biofilm on the root surface**: virulent microbes (actinomyces), root biofilm, foreign matter protecting biofilm
    - Would need apical surgery to address biofilm forming outside of the root
    - Actinomyces infections will need surgical intervention to treat
  - **Periapical tissues**
    - Type 1 cases: an abscess forms and will need an incision to drain and resolve
    - Type 2 cases: complete infection of periodontium, like osteomyelitis. Will require hospital treatment and possibly resection of the bone
- Oral pain
  - 90% of **chronic** oral pain is non odontogenic
  - **Dental causes**: pulpitis, apical periodontitis, resorptions (rare), hypersensitive dentin, dentin microfractures, cracked tooth, split tooth, gingivitis/marginal periodontitis, sinusitis
  - **Others**: trauma, primary herpes, viral infections, mucosal infections, neuritis, neuralgia, neuropathic pain, etc
  - Persistent endodontic infections are rarely symptomatic – have to make a call whether to leave the infection and keep the tooth, or retreat and risk losing the tooth
- Microbiology of apical periodontitis
  - Primary lesion
    - Anaerobes: gram – rods, gram + rods, gram + cocci
    - Facultative: gram + cocci, gram + rods
  - Secondary lesion
    - Facultative: *E. faecalis* (77%), Lactobacilli, Streptococci, gram + rods, gram – rods
    - Less anaerobes, more yeasts (*C. albicans* found in 9%)

Longitudinal fractures

	Presentation/symptoms	Diagnosis	Treatment
Craze line	**Fracture line in enamel** -Common in adults -Symptom free, and will not cause any problems	-Visible in good light	-No treatment indicated
	Fracture line in dentin -Usually symptom free, but may be painful in biting, pulp remains vital -Often under large fillings	-Visible in good light -Unknown whether an enamel craze line is necessary for a dentin craze line to form	-Stabilization of cusps may be needed, but has a good prognosis
Cuspal fracture	-In teeth with large caries/restorations or undermined cusps, but could also be seen in an unrestored tooth -Associated with a weak marginal ridge or excessive occlusal force -Could have pain initially (fluid movement in dentin tubules), but symptom free when cusp falls off -Can be supragingival or extend into epithelial attachment -Usually single cusp due to oblique shearing force -Usually no pulp exposure	-Not visible radiographically if broken piece is still attached -Clinical visualization -Transillumination -Dye staining -May need to remove filling to see **Pain assessment** -Pain with release -Pain to biting and cold is sharp and brief -No spontaneous pain	**Prevention** -Check for eccentric movements after doing a restoration -Expansive fillings like amalgam can stress the tooth on biting **Treating** -Pulp test and remove filling -Place cuspal restoration -Possibly CL or extrusion -RCT usually not indicated -Good long term prognosis -Catch fractures before catastrophic failure
Crack tooth	-Only seen on posterior teeth -Incomplete crown fracture, propagating along the long axis of the tooth -Vertical fracture extending M-D -No separate segments -Frequency: Md 7 > Md 6 > Mx 4/5 > Mx 6 -Frequency: class I = class II -Could occur on unrestored teeth -Due to strong bite, habits, steep cusps, tight cusp/fossa occlusion, inadequate canine guidance, dietary preference -No evidence RCT weakens tooth to increase risk of cracked tooth -Pulp may be involved if untreated	-75% tested vital pulp → <u>cracked tooth is not considered a pulpal diagnosis</u> -80% of radiographs were normal -Could have acute pain on biting (especially release), or asymptomatic -Since you don't know how far the crack propagates, stick a beaver tail between B/L halves and try to expand. Do this gently to see if there is mobility	-Prognosis worse if crack is more centered or is deeper -Deep probing = poor/hopeless -RCT first if needed **Stabilize cusps** -Ortho band and wait **If symptoms subside** -Temp crown +/- bonded core -Permanent crown or onlay -Tell pt crown may only delay Sx **If symptoms persist** -Pulpectomy while tooth is banded → RCT + crown when tooth is asymptomatic -Extraction
Split tooth	-Cracked tooth with a crack that has propagated apically and exits the tooth -Cracks M-D in a vertical direction -More centered the crack, more apically it extends -Like cracked tooth, only in posterior teeth -Visible fracture when restoration is removed -Often involves marginal ridges	-Typically pain on biting, but can vary -Usually deep pocketing adjacent to fracture -Bone loss may be seen on radiograph	-Extraction if split is severe -If fracture has one large piece and one small piece, can retain large segment -CL or extude if needed -Perio consultation for multi rooted teeth is recommended if not extracting
Vertical root fracture	-Crack in the root that continues coronally or apically -Thought to start in root canal and extends B-L, but not always through the whole root -Usually no separation of segments -More common in roots wide B-L, narrow M-D -Causes: inappropriate post placement, excessive obturation	-Fracture often invisible or poorly visible on radiograph -May see "halo" or "J" shaped radiolucency -Radiographically mimics failed RCT or perio -Deep localized probing -Usually symptom free or mild symptoms -May open flap to see	-Extraction, amputation, or hemisection of fractured root -Hopeless prognosis for the fractured root

Root perforations

- Etiology

Iatrogenic	Coronal 1/3	-While locating or opening canals, misidentifying canals -Treating calcified pulps -Significant tooth angulations
	Middle 1/3	-Excessive instrumentation -Instruments straightening curved roots (molars) -Instrumenting calcified canals
	Apical 1/3	-Poor technique can cause ledges, which deviate the instruments from the canal -Aggressive instrumentation through the apical constriction
	Post space	-Need good length and width of post -Poor post positioning, into dentin rather than canal
Pathological	-Root resorption (internal inflammatory RR, external inflammatory RR) -Extensive caries	

- Diagnosis
  - Clinical signs and symptoms
    - Profuse bleeding seen through the access prep or on a paper point inserted into the canal
    - Sudden pain during treatment (if LA is not being used)
  - Using instruments
    - **Apex locator**: will show "0" → indicates communication with PDL
    - **Microscope**: will be visible
    - **Radiograph**: may be hard to see as 2D image, but can take an off angle as well to help
    - **Cone beam**: increasingly used, but presence of GP/posts/cores will interfere with CBCT clarity
  - If not identified during treatment
    - Serous exudate from perforation
    - Sensitivity to percussion, localized pocketing, chronic gingival inflammation
    - Local osteolysis (radiolucency) on radiograph
- Sequelae of untreated perforations
  - Success of RCT drops by 56% due to a pathway for bacterial contamination
  - Initial acute inflammatory response
  - Destruction of PDL, bone, and formation of granulomatous tissue
  - If perforation is close to supra-crestal attachment, may cause epithelial proliferation and form a pocket
  - Ultimate loss of tooth

Table 1 The prognosis for success when considering site, size and time to repair of perforations

Prognosis	Site	Size	Time to repair
Favourable	Apical or supra-crestal	Small	Immediate
Unfavorable	Equi-crestal	Large	Delayed

- Prognosis of a perforation
  - Site
    - **Critical zone**: worst prognosis as it allows bacterial contamination, leads to a periodontal defect, and migration of epithelium into the perforation
      - A perforation in the furcation is also considered part of the critical zone
    - **Coronal to critical zone**: can get a good seal, has a good prognosis
    - **Apical to critical zone**: if accessible, it can be cleaned and sealed well. Has a good prognosis as it is less accessible for oral bacteria
  - Size: smaller is better
  - Time: immediate sealing is better
- Repair material
  - Historically: amalgam, ZOE cement, CaOH, GP, GIC, IRM, composite, SuperEBA cement
  - Now: mineral trioxide aggregate (MTA)
  - MTA is hydrophilic and sets in moisture, promotes repair/regeneration (cementum + periodontium), good seal
  - Disadvantages of MTA: hard to handle, takes 4 hours to set (may wash out if coronal), discolours tooth
  - Future: Biodentine

- Management principles
    - 2 choices: repair or extraction
    - If access is impossible without significant collateral risk, extraction may be the only option
    - Obtaining good visibility is essential
- Non surgical management

General principles	**Root canal treatment** -Obturation should be completed if possible -If not, protect canals with a removable material like Cavit, cotton, GP, or paper points so the restorative material doesn't end up blocking the canal **Timing** -If perforation is repaired immediately, it is non contaminated -If perforation is left for a while, must be decontaminated -Enlargement and cleaning of the perforation is best done with an ultrasonic -Site is copiously irrigated with 2.5% bleach (dangerous) or CHX (less dangerous) -If risk of irritating periradicular tissues is too high, can use saline but clinician must know that the perforation will not be decontaminated -Remove granulation tissue **Hemostasis** -Don't use agents like ferric sulfate as it can harm alveolar bone -Use collagen, calcium sulphate, or CaOH -If not possible to stop bleeding, pack Cavit and reattempt at another appointment
Coronal third perforations	-If isolation is possible, use composite or GIC -If close to the critical zone, use MTA or Biodentine -Placing MTA first and then a barrier of RMGIC is also a possibility
Middle third perforations	-Usually a strip perforation, which is very hard to isolate unless very small -Pre-bend all instruments to avoid instrumenting the perforation rather than the canal -2 options for treatment: 1. Hemostasis → place GP → sear off GP coronal to perforation → pack MTA into canal 2. Hemostasis → place GP/paper point → seal perforation → remove GP/paper point and perform proper obturation
Apical third perforations	-Access and visibility is the most difficult, and would require a straight + wide canal -Try to find the original canal → instrument → warm vertical GP and hope it will fill in defect -If original canal cannot be instrumented → warm vertical GP into perforation -Will leave uninstrumented tissue at the apex, and hence has worse prognosis

- Surgical management
    - Indications
        - Uncertainty of shape/nature of defect
        - Defect is subcrestal and associated with pathology or symptoms
        - Internal access not possible
        - Large defect
        - Apical third perforation with persistent disease
        - External cervical resorption
    - Technique
        - Raise a flap
        - Access perforation with a small round bur, piezo handpiece, or hand instrumentation with curettes
        - Achieve hemostasis
        - Pack material (MTA or resins)
    - Much poorer prognosis than non surgical

Root resorption

- Introduction
  - Progressive loss of dentin and cementum through osteoclastic resorption
  - Can happen internally or externally
  - Initiators: pulp necrosis, trauma, periodontal treatment, ortho, whitening agents
  - Irrespective of the cause, process is inflammatory in origin
  - Managed with endo, but may require surgical exposure and restoration
  - If ankylosis is suspected, decoronation or close monitoring are recommended
- Etiology

Caries	-Pulpal inflammation → clastic activity stimulated → internal resorption -Vital pulp apical to resorption site is necessary, with the coronal pulp being necrotic -Bacteria could also induce clastic activity -When pulp is fully necrotic, resorption ceases
Periodontal disease	-Apical migration of attachment → exposure of root surface + low grade inflammation → favourable site for clastic activity -Rarely seen clinically
Trauma	-Damage to cementum is most significantly associated to the onset of resorption -Loss of cementum means dentin tubules are exposed to all bone cell activity, and EIR may occur -Damage in the cervical area may cause ECR -Avulsion with extraoral time of >1h may result in extensive ERR -Ideally, healing will repopulate cementoblasts before bone cells. Will result in ESR -Apical intrusion is the most damaging to cementum, more than avulsion -Intrusion > avulsion > lateral luxation > subluxation > concussion
Orthodontics	-Resorption found in 19~31% of ortho, highest being mand and max incisors and least commonly molar and canines -Ankylosis does not occur -Well planned ortho moves teeth with even pressure on all PDL surfaces -If certain areas have excessive forces, local necrosis occurs → osteoclasts move in to remove necrotic tissue → attacks cementum after → can affect tooth -Apical pressure can cause blunting of roots or loss of root length, possibly due to EIR on apical cementum and IIR on apical predentine -Could also be a factor in developing external cervical resorption
Impacted teeth	-Follicle approaches periodontium of adjacent teeth -Pressure exerted by the follicle can induce resorption, much like orthodontics
Temperature	-Warm obturation and orthodontics can heat the tooth -Possible relation to resorption, as it upregulates clastic signalling mechanisms
Chemical	-Internal bleaching chemicals leak into dentin tubules at/below the CEJ -May provoke inflammation directly or denature dentin such that it initiates an immune response
Viral	-Found some cats with viral related resorption… ok
Systemic disease	-Hyperparathyroidism causes systemic decalcification -Process may involve dental hard tissues as well
Physiological	-Resorption of primary teeth is necessary to facilitate eruption of permanent dentition

	About		Treatment
External surface resorption	-Localized and limited injury to the root surface and surrounding periodontium -2~3 weeks of resorption followed by repair and reattachment of PDL -If isolated to cementum, will heal with no damage -If dentin affected, root surface may be damaged		-Subclinical -No treatment
External inflammatory resorption	-3 requirements: trauma to root surface, exposure of dentinal tubules, communication to an infected or necrotic pulp -Pulp may be vital if sterile -Immature/young teeth more frequently affected **Sterile** -Associated with orthodontics, impacted teeth, trauma -Will usually see rounding of apices **Infective** -Associated with trauma and/or pulpal necrosis -Bowl shaped resorption primarily on apex or lateral canal		**Prevention is essential** -Mouthguards should be used if patients participate in high risk activities -Replant avulsed teeth ASAP and manage according to guidelines -If apex is closed, endo should be done within 7~10 days **Damage limitation** -If sterile and due to ortho, reduce the amount of stress on the tooth -If infective, perform non surgical endo -If access is not possible or there is lack of patency, surgical approach is indicated
External cervical resorption	-Resorption below epithelial attachment -Unlike inflammatory resorption, it is associated with a vital pulp -May be significant tooth structure loss -May affect multiple teeth (multiple idiopathic cervical resorption) -Unknown etiology -Asymptomatic or mild discomfort -Cervical enamel may look pink as granulation fills the cavity, probing will cause profuse bleeding, thin and sharp cavity edges (but not carious)		Prevention when internal bleaching -Seal GP with >2mm of GI/composite, and up to the CEJ -Proceed with bleaching the crown Treatment -If suspected pulpal involvement, do RCT -Surgically open area → curette until no bleeding → condition cavity with 90% trichloroacetic acid or bleach + EDTA → fill cavity with GI, comp, or MTA
External replacement resorption	-Replacement of resorbed surface with bone, AKA ankylosis -Unknown etiology -May be transient (self limiting) or progressive (complete resorption and loss of PDL) -ERR is slower in adults, so a tooth may last ~20 yrs -3 signs are found when 10~20% of the root is affected: high pitched percussion, lack of mobility, tooth is in infraocclusion if ERR happened during growing age -On radiograph: loss of LD and moth eaten root surface		1. Accept infraocclusion and build up composite on incisal edge 2. Extraction (will be surgical) at the start of puberty 3. Extract and replant tooth 4. Extract and transplant a premolar to site (only in pts <14 yo) 5. Open flap → decoronate tooth → cover it up → preserves bone until more permanent tx
Internal surface resorption	-Associated with periapical pathology and is common -Advancement of external inflammatory resorption into the canal -Must be vital pulp apical to the osteoclasts to provide a nutrients and necrotic tissue coronally to stimulate osteoclasts	-Analogous to external cervical resorption -Clastic activity arrests and is self limiting	
Internal inflammatory resorption		-Ovoid or fusiform enlargement -Expansion in apical and lateral direction -May be analogous with EIR -Tooth may appear pink, as granulation fills and grows in the canal -On radiograph: well defined circular resorption in canal	-RCT -Check for perforation after accessing -Completely disinfect canal -Fill resorptive cavity with flowable GP
Internal replacement root resorption		-More irregular enlargement -Diffuse lucencies and opacities -May lead to obliteration -May appear as a pinkish crown	-More complex due to calcifications within canal -Surgical endo may be needed

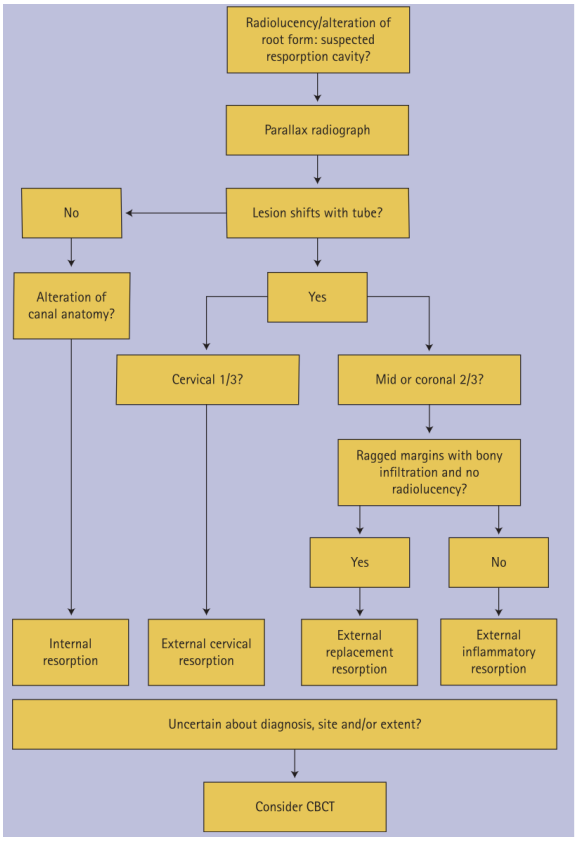
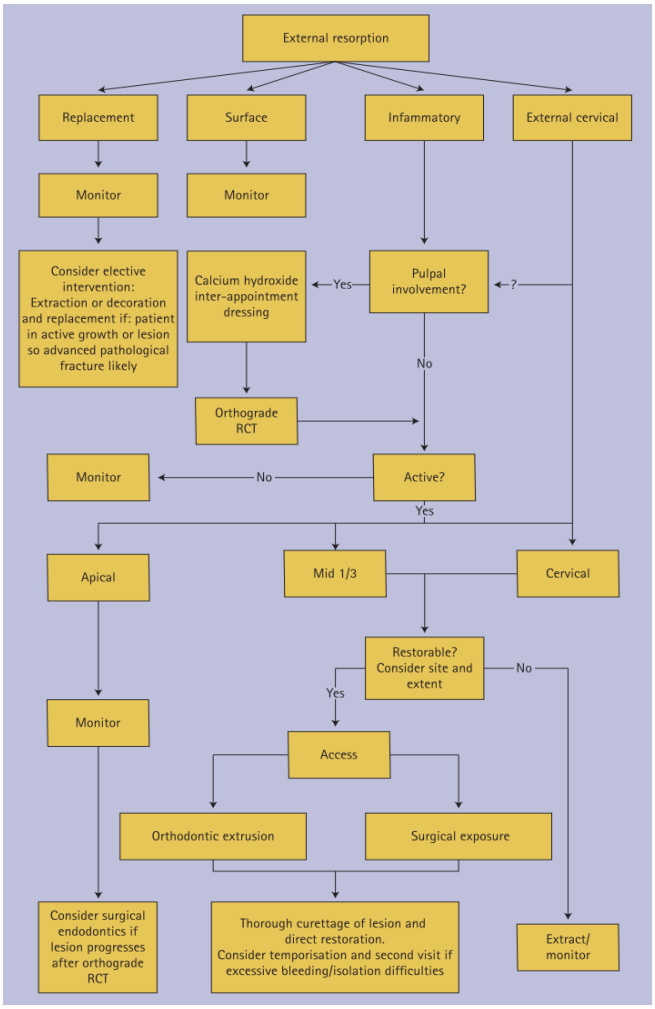
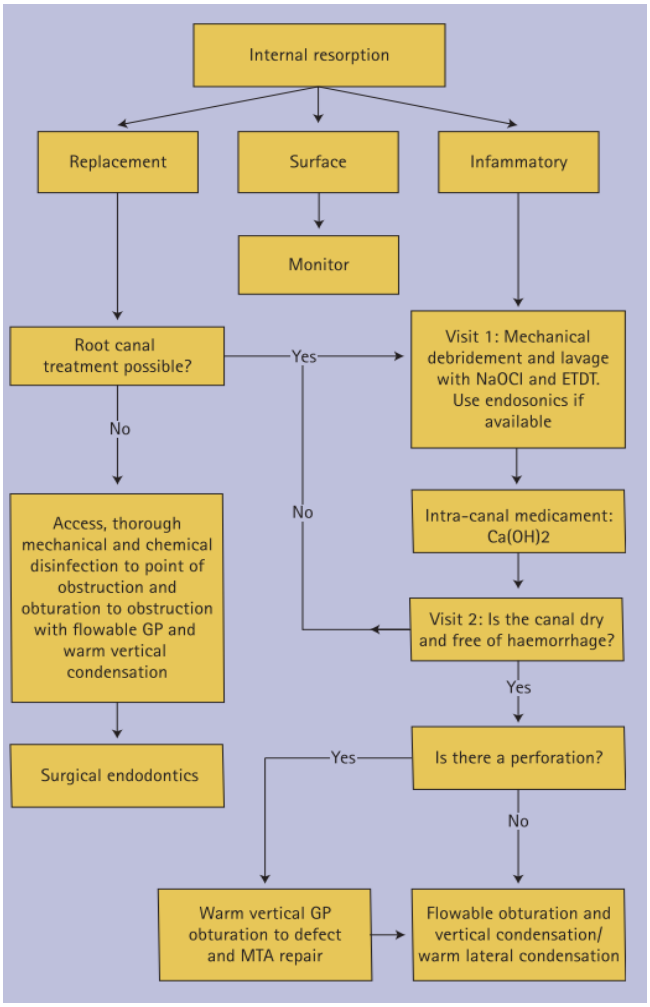


Table 1 Diagnostic features of resorption

Diagnosis	Mobility	Percussion	Colour	Vitality	Radiographic signs
External surface resorption	Normal	Normal	Normal	Normal	Minimal if at all: small cavity in the cementum and superficial dentine
External inflammatory resorption	Maybe increased	Normal	Normal, may change if pulpal involvement	Non-vital unless 'sterile' pressure resorption	Loss of lamina dura, irregular saucer shaped lesions with adjacent peri-radicular radiolucency
External cervical resorption	Maybe increased	Normal	Possible pink discolouration of cervical enamel	Normal, possibly increased response to vitality testing if lesion close to pulp. Absent if pulpal involvement.	Radiolucency of cervical third of root, may resemble class V cavities or internal resorption
External replacement resorption	Absent	Metallic sound	Normal, may change depending upon nature of trauma	Typically absent as sequelae of trauma	Irregular alteration to root form, loss of lamina dura, bony infiltration resorptive cavities with direct apposition of bone onto root surface. Absence of radiolucency of the periodontal ligament.
Internal inflammatory resorption	Normal	Normal	Possible pink discolouration if lesion is coronally situated	Mixed response: positive in active lesions, absent in extensive lesions	Expansion of canal, often symmetrical with sharp, well-defined margins
Internal replacement resorption	Normal	Normal	Possible pink discolouration if lesion is coronally situated	Mixed response: positive in active lesions, absent in extensive lesions	Irregular expansion of canal. Possible pulpal obliteration with mixed radio-opacities in the canal space





Mediators of acute and chronic periradicular lesions

- Introduction
  - Infection of the root canal can cause an inflammatory reaction at the exit of the main canal or an accessory canal
  - A periradicular lesion can form due to bone destruction secondary to the inflammatory process
  - This paper looks at the endogenous and exogenous compounds that are involved in this process and its possible connection to the pathologies seen

Mediator	Origin	Function	Dental relevance
Neuropeptides	-Proteins from somatosensory and autonomic nerve fibers -Stored in granules of nerves	-**Substance P**: vasodilation, vascular permeability, histamine release from mast cells. Potentiates inflammatory reactions -**Calcitonin gene related peptide:** potent vasodilator, may play a role in blood flow to bone and periosteum -Both SP and CGRP were found in dental pulp -Severing the IAN → complete disappearance of SP and CGRP → suggests they are made in the sensory fibers of the trigeminal ganglion -**Vasoactive intestinal peptide:** stimulator of bone resorption via a PGE_2 independent pathway. Has been found in dental pulp. VIP receptors are present on osteosarcoma cells. -Severing the IAN → VIP was still present, suggesting that it is of parasympathetic origin	-Role of NP in pathogenesis of periradicular pathosis has not been solved
Fibrinolytic peptides	-By-product of clot degradation	-Tissue injury → exposed collagen → platelets adhere (**primary hemostasis**) → coagulation cascade → **secondary hemostasis** -Factor XIIa or tissue factor will activate plasmin (AKA fibrinolysin) -Plasmin will begin to digest the clot, releasing fibrinopeptides and fibrin degradation products -These byproducts cause ↑ vascular permeability and chemotaxis of leukocytes	-Damage to blood vessels in PDL or bone during RCT can contribute to the inflammatory process
Kinins	-Liver, endothelium, platelets, neutrophils	-Kininogen → cleavage by kallikreins → activated **kinins** -Once released by cells, causes chemotaxis, contraction of smooth muscles, dilation of arterioles, increased permeability -Can cause pain by activating nerve fibers -Inactivated by peptidases	-Elevated kinins have been detected in human periradicular lesions (PRL)
Complement system	-Liver, circulates in blood	-Cell death: can form a membrane attack complex, or increase attraction to phagocytic cells -Other effects: ↑ permeability, chemotaxis for granulocytes and macrophages -Can be activated by the classic pathway (antigen-antibody complexes) or alternative pathway (direct interaction with carbohydrates on pathogen cell wall or plasmin) -Can cause destruction of bone or prevention of new bone formation via the production of prostaglandins	-C3 complement has been found in PR lesions -Most activators of complement are found in PRL
Vasoactive amines (histamine and serotonin)	-Mast cells, basophils, platelets	-Causes ↑ permeability, dilation, and smooth muscle contraction -Histamine release is triggered by physical/chemical injury, complement, activated T cells, membrane bound IgE antigens	-Mast cells have been found in PRL -RCT could cause mast cell degranulation
Lysosomal enzymes	-Lysosomes of PMN leukocytes, macrophages, platelets	-Group of proteolytic enzymes including: acid/alkaline phosphatases, lysozyme peroxidase, cathepsins, collagenases -Released if cell dies (lysis) or secretory release during phagocytosis -Effect: ↑ permeability, chemotaxis, cleavage of complement C5, activates bradykinin from plasma kininogen	-PRLs contain lysosomal hydrolytic arylsulfatase A + B
Arachidonic acid metabolites	-Cell membranes	-Oxidation of AA → produces prostaglandins, thromboxanes, and leukotrienes (see subsections below) -They are synthesized as a result of cell injury	

Mediator	Origin	Function	Dental relevance
Prostaglandins	-AA → COX pathway → PG	-PGE_2 and PGI_2 are associated with ↑ permeability and pain -PGE_2 caused bone resorption -Parathyroid hormone related proteins release PGE_2 → bone resorption -Cytokinins like IL1 can also stimulate PG production -Indomethacin blocked the effects of PGE_2	-High PG's found in inflamed gingival + pulpal tissues -PGs demonstrated PRL formation
Leukotrienes	-AA → LOX pathway → LT	-Causes chemotaxis (PMNs, eosinophils, macrophages), ↑ permeability, lysosomal release -Concentration of LT found to correlate with # of PMN leukocytes	-LT B4 has been found in PRLs
Cytokines	-Bone marrow, bone cells	-Stored in bone matrix to be release during remodelling -Also released during inflammatory processes -Cytokines implicated in bone resorption: IL1, 3, 6, TNF, CSF	
IL-1β, IL-1α	-Monocytes, macrophages	-IL-1β is the most active cytokine in stimulating bone resorption -15x more potent than IL-1α, 1000x more than TNF	-Seen in dental pulp -Implicated in bone resorption of PRLs
IL-3	-T lymphocyte	-Causes precursor cells to differentiate into osteoclast-like cells -Produced during resorptive diseases like RA and periodontitis	-Bone resorption in periodontitis
IL-6	-Many cells, one being osteoblasts	-Produced during resorptive diseases like RA and periodontitis	-Bone resorption in periodontitis
TNF	-α → from monocytes -β → from lymphocytes	-Similar to IL-1 -Effects on osteoclasts are indirect and are mediated through blasts -TNFα's resorption effect is dependent on PG synthesis	-Detected in diseased gingiva and periradicular tissues
CSF	-M-CSF → from blasts -GM-CSF → from T lymphocytes	-Causes growth and differentiation of many hematopoetic cells -M-CSF: directly ↑ osteoclast precursors, perhaps with IL1/3. IL1 and TNF enhances M-CSF production -GM-CSF: ↑ clasts and ↑ blasts. PTH or bacterial LPS stimulates production of GM-CSF	

- Immunologic reactions
  - Antigen-antibody complex reactions
    - Bacteria/byproducts interact with IgG or IgM → forms a complex → binds to platelets → release of vasoactive amines → ↑ permeability + PMN chemotaxis
    - Formation of immune complexes found to cause periradicular lesions in animals
    - Studies show immune complexes formed in chronic periradicular lesions are either minimal or confined within the lesions, and do not enter into systemic circulation
    - Patients with acute abscesses did demonstrate complexes found in the blood
  - Cell mediated immune reactions
    - B and T cells were found in periradicular lesions, with T cells heavily outnumbering B cells
    - Acute phase: T helper > T suppressor → stable phase: T helper < T suppressor
    - Appears that TH cells develop the lesion, and TS cells decrease immune reactivity
    - Natural killer cells were also found in PRLs
- Other findings
  - Pathogenesis of periradicular lesions is a multifactorial phenomenon, not just based on lymphocytes
    - Trauma, egress of antigens, various types of immunologic reactions
  - Pulpal necrosis extends coronally to apically
  - Periapical lesions extend mesiodistally (initially)

Biological perspectives on the non-surgical endodontic management of periradicular pathosis

- Introduction
  - Radiographs alone are not enough to differentiate between an apical granuloma or cyst
  - There is a suggestion that a lesion increasing in size with radiopaque borders is a cyst, but it is misleading because it prompts clinicians to perform surgical treatments which may be unnecessary
- Histopathology of periradicular tissues
  - >90% of periradicular lesions can be classified as granulomas, cysts, or abscesses
  - Study in mice showed that bony infections can be divided into 4 zones:
    - Zone of infection: microorganisms and neutrophils
    - Zone of contamination: round cell infiltrates
    - Zone of irritation: histiocytes and osteoclasts
    - Zone of stimulation: fibroblasts, new collagen, initial osseous deposition
  - Contents of a periradicular lesion in humans
    - Predominantly granulation tissue: angioblastic activity, fibroblasts, CT fibers, inflammatory infiltrates
    - Inflammatory infiltrates: plasma cells, lymphocytes, phagocytes, neutrophils
    - Often a connective tissue encapsulation
    - Occasionally cholesterol clefting or foreign body giant cells
    - If strands of epithelium or Rests of Malassez have been stimulated, an epithelial lining may form to create a cyst. The cyst can fill up with fluid or semi-solid material
    - It is difficult to know the incidence of cyst formation, as studies vary from 7~54%. This has to do with the variables in diagnostic criteria for a cyst
  - Simon's classification of periradicular cysts
    - Bay cyst: epithelium walls off irritants from the root canal. Cyst communicates with the root canal
    - True cyst: epithelium completely walled off. No communication to the root canal
    - Bay cysts may heal from non surgical endo, and true cysts need to be surgically removed
    - True cysts can sometimes heal via non surgical endo, as long as:
      - Source of coronal leakage is eliminated
      - Concomitant periodontal disease is managed
      - Tooth defects (fractures, resorption) are recognized and managed
    - Since we can't determine a bay cyst, true cyst, or granuloma on a radiograph, a conservative approach is recommended (non surgical RCT) in lesions perhaps <20mm
  - A periradicular lesion is in a constant state of repair and destruction
    - Unless the source of infection is removed, destruction will always be present
    - This is important for clinicians to know. Lesions removed with surgery only fix the issue temporarily, but fails to address the removal of the source of irritants
    - Non surgical RCT can treat these lesions
- Histopathology of periradicular tissue repair
  - After RCT on a granuloma or bay cyst: Inflammatory cell infiltration → removal of endogenous and exogenous irritants → fibroblastic proliferation → collagen deposition → bone formation → cemental apposition (if root resorption was present)
  - In a true cyst, non surgical RCT should in some way lead to local or generalized destruction of the epithelium. There are 2 proposed mechanisms as to how the epithelium is destroyed
    - A) RCT instruments are pushed 1mm beyond the root end 2~3 times, while preventing over-instrumentation. This causes acute inflammation → subepithelial hemorrhage → ulceration of epithelium
      - This mechanism is debated, as it could cause epithelial proliferation, not resolution
    - B) RCT instrument is pushed to the center of the radiolucency → establish drainage → reduce cystic pressure → cell lining collapses → fibroplasia → collagen deposition → ↓ vascularization of lesion
      - Theoretical, but clinically has shown success

- 5 criteria for histological repair
    - Absence of inflammation
    - Regeneration of periodontal fibers adjacent to or inserting into healthy cementum (Sharpey's fibers)
    - Layering or repair of cementum with new cementum into or across the apical foramen (rare)
    - Osseous repair is evident along with healthy osteoblasts surrounding the newly formed bone
    - No tooth resorption is present and previous areas of resorption demonstrate cemental deposition
- Clinical implications
    - Over-instrumentation technique
        - Pushing a file beyond the radiographic apex seems counter intuitive to conservative non surgical RCT, but it can be done using 10/15 K files without altering apical morphology much
        - File should be curved slightly at the apical 1~3mm and gently teased past the foramen
        - Barbed broaches or Hedstrom files should never be used for this (possible breakage past apex)
    - Calcium hydroxide placement
        - Filling canals with CaOH as an interim solution yielded 66.7% of lesions showed healing/complete heal in 12 months, 79.5% in 18 months
        - Pushing CaOH past the apex has also been advocated, due to 4 reasons
            - Anti inflammatory through its hygroscopic action, formation of CaOH proteinate bridges and inhibition of phospholipase
            - Neutralization of acid products (like acid hydrolases)
            - Activation of alkaline phosphatase
            - Antibacterial action
        - CaOH's necrotizing ability also may help it destroy the cystic epithelium, allowing healing
    - Phenolic resin mixture (not recommended for routine Tx)
        - Phenolic resin was placed in the canals after debridment
        - Resin infiltrated the canals, accessory canals, and dentinal tubules. Once set, this would entrap all residual debris and form a seal
        - 85.5% success rate, but not used. This study mostly emphasizes importance of good debridment
    - 9-aminoacridine periradicular lavage
        - An irrigant used in the 1940's due to its high spectrum of antimicrobial activity
        - Use not recommended due to unknown safety and efficacy
    - Decompression (marsupialization)
        - Lesions close to anatomical structures like the nasal cavity, sinus, or mandibular canal may benefit
        - A communication is created to the lesion, allowing cystic drainage → epithelial destruction → repair
        - Hopefully would make surgery unnecessary

Mid treatment flareups

- Introduction
    - Flareup is characterised as an episode of swelling and/or pain
    - Will discuss the management of pain and swelling after the initial appointment
- Pain
    - Pain in mid-treatment flareups is acute in nature, not chronic pain
    - As clinicians, we deal with patients presenting with a varying range of pain. It is hard to quantify exactly how much pain the patient is in, but there are ways to attempt objectively scoring pain
        - If over the counter aspirin, Tylenol, or Advil manages the pain, then it is mild
        - If the patient can sleep at night with the pain, then it is mild
        - If the pain needs to be managed by prescriptions like codeine, then it is moderate
        - Pain not controlled by codeine is severe, which necessitates immediate attention from the clinician
    - Pain in midtreatment flareups
        - Incidence of moderate ~ severe pain in mid-treatment flareups is 2.5~16%
        - Pts asymptomatic at initial appt and don't have a PA lesion have an incidence of 0~16%
        - Pts asymptomatic at initial appt with PA lesions with incidence of flareups have an incidence of 3.8~25%
        - Some studies, however, show now difference in incidence for teeth with/without PA lesions
        - Flareups are not related to irrigant used nor the intracanal medicament used
        - Flareups are mostly involved with maxillary laterals, mandibular premolars, and mandibular molars
        - All studies report the occurrence of moderate~severe pain during the course of RCT
    - Management
        - Routine management
            - Inform the patient of possible slight pain for several days after the appointment
            - Recommend 1~2 aspirin or its equivalent q2h for the remainder of the day, and next day PRN
            - Advise tooth will be tender to biting for 2 days
            - Offer contact information in case OTC analgesics do not manage the pain
        - Moderate to severe pain after initial appointment
            - Clinician should review diagnoses to ensure tooth is the source of the pain
            - Review pulpal status and radiographs to see if the pain is just an inflammatory response or an early acute infection
            - Management varies depending on whether pulp was initially vital or necrotic
        - Moderate ~ severe pain after removing vital pulp
            - Uncommon, no pain or only slight pain is to be expected
            - Most commonly due to incomplete removal of the pulp, especially multi rooted teeth
            - If the pulp is essentially intact on opening, biting tenderness is likely an extension of the inflammatory process
            - If tooth becomes tender to biting, the inflammatory process has involved periapical tissues. This can be mitigated with NSAIDs
            - **Accurate tooth length and ensuring complete instrumentation will reduce flareups**
        - Moderate ~ severe pain after removing necrotic pulp
            - Most studies show flareups is more common for teeth with necrotic pulps
            - Necrotic pulps without PA lesions = may be infected, with PA lesions = likely infected
            - Instrumentation may introduce bacteria into the PA space, causing pain or swelling
            - As stated above, **complete instrumentation of pulp is crucial to prevent flareups**
            - Could also be due to an acute abscess forming
                - Evaluate the patient for swelling palpation tenderness, tenderness to biting
                - Re-access the pulp chamber to check for pus flowing from the canal
                - **In the absence of swelling or pus in the canal, pain is not considered a reliable indication of an acute abscess**
            - Antibiotics can be given judiciously prophylactically after the appointment. Since 75% of patients will not have any pain or swelling, we shouldn't give abx to all patients with necrotic pulps

- Clinical protocol when a patient presents with pain in a necrotic pulp
    - Reaccess the pulp chamber
    - **No pus** → ensure canals are cleaned properly → irrigate copiously → medicament is placed → chamber is re-sealed
        - Pain subsides → routine treatment may proceed
        - Pain persists → likely a beginning abscess not yet broken to soft tissue → give antibiotics, give strong analgesics (since abx will take ~48h to kick in), take tooth out of occlusion
    - **Pus** without swelling → is an acute early abscess, and pain is severe as pus has nowhere to escape yet → same treatment as an acute abscess with swelling (coming up next)

- Swelling
    - Incidence of swelling after starting an RCT is low. Only 1.5% for first RCT's and 5.5% for retreatment RCTs
    - Treatment can vary greatly depending on clinician. Some may choose to allow the tooth to drain by leaving the access open, others seal off, some will give antibiotics, others wont
    - Swelling classification
        - **Local**: confined to oral cavity
        - **Diffuse**: spread into lip, chin, cheek, etc. Free to spread along the avascular fascial spaces. Appropriate appraisal and knowledge of anatomy is critical to initiate proper treatment. Could be life threatening
    - 3 ways to resolve swelling and infection of a pulpless tooth
        - Allow drainage of pus through the root canal
        - Allow drainage of pus through fluctuant soft tissue
        - Antibiotic treatment
    - Treatment methodologies
        - Use of antibiotics alone without attempts to establish drainage is not appropriate treatment
            - Antibiotics will not reach sufficient levels in tissues with pus
            - Antibiotics may even be ineffective until drainage is established
            - If drainage cannot be achieved, then antibiotics can be used as primary treatment
        - Localized swelling
            - Abscess is confined to the oral cavity, not as aggressive or dangerous
            - If adequate drainage is obtained, systemic antibiotic is not needed
            - Only give antibiotic when drainage could not be established
        - Diffuse swelling
            - Advanced infection that is potentially dangerous
            - Oral penicillin or erythromycin (if allergic to penicillin) is recommended
            - Penicillin covers the most common periapical abscess associated bacteria: staphylococci, streptococci, and some anaerobes
    - Types of swelling seen after initial treatment
        - Diffuse swelling is further broken down into **moderate** diffuse abscess and **extensive** diffuse abscess
            - Least severe moderate diffuse abscess will show swelling just beginning to spread to surrounding tissues like the lip, chin, etc
            - Very severe extensive diffuse abscess will have extreme characteristics like swelling in the FOM, soft palate, nose, eye, eyelid, neck, or a fever of >101F
            - There is a very wide range of abscess severity between these extremes
            - Clinicians treat all cases as if it were an extensive diffuse abscess, because it doesn't impose much hardship on the patient and clarifies treatment for the clinician
    - Types of treatment
        - Drainage via the pulp chamber
        - Incision of a fluctuant soft tissue swelling
        - Trephination of the apical foramen
        - Use of hot saline holds
        - Systemic antibiotic therapy
        - Taking a culture for antibiotic sensitivity testing
        - Prescription analgesics for pain control
        - Adjustment of occlusion if there is biting tenderness

- Treating acute abscesses – in detail

Drainage through the tooth	-In localized and moderate diffuse swelling, wait for drainage to stop→ irrigate→ dry→ medicate→ close -Milking the soft tissue can expedite flow of pus -Close the chamber if it can be dried. If pus continues to drain, it can be left open -In extensive diffuse swelling, chamber can be left open in all cases -Even if drainage could not be established, leave it open as it may spontaneously drain later
Incision and drainage	-In many cases, incising the swelling may not yield any pus. This is because pus only accumulates after swelling has been present for some time -The swelling needs to be fluctuant to yield any pus. If not fluctuant, then incision will not drain anything -Localized/moderate abscess → if draining through root canal, no need to incise soft tissue even if it is fluctuant -Extensive diffuse abscess → achieve drainage through all sources possible, with a drain inserted
Trephination of the apical foramen	-Ream the apical 2~3mm of the root canal up to a size 35~40 → gives a larger orifice for pus to drain -There are many disadvantages to this procedure: -Harder to obturate later -Not guaranteed to drain the pus, as it may not be present at the foramen yet -Mechanical trauma to the periapical tissues -Forcing additional bacteria and necrotic debris into the periapical area - Therefore, it is only done in the following circumstances: -Lesion is diffuse extensive, opening the chamber + incising soft tissue has yielded no pus, patient is in pain, and pain is not relieved by opening the chamber -Lesion is not extensive, but pain is a major problem -First attempt to resolve pain or swelling was unsuccessful and abscess is worsening -Precautions -Buccal canals of max molars, mesial canals of mand molars → size 35/40 may cause shelving or perforation since they are small canals → limit trephination to smaller files -Proximity of apex to mental foramen, mandibular canal, maxillary sinus
Hot saline holds	-Hot salt water applied to soft tissue will draw pus to the tissue surface -May cause a non fluctuant swelling to become fluctuant -Useful when canal drainage is not present and soft tissues are not fluctuant -Instructions -Place a teaspoon of salt in a glass of water as hot as can be comfortably tolerated -Hold water so it contacts swollen tissue until water cools -Repeat until whole glass is empty -Repeat all steps as frequently as possible for 4~8 hours -Avoid heat on the face as it can draw the pus there
Antibiotic sensitivity	-It is useful to test if which antibiotics will be effective, but there are some complicating factors: 1. Periapical abscesses have many types of bacteria. It's hard to know which bacteria is actually responsible for the abscess 2. Anaerobes causing periapical abscesses will die when they are exposed to air during sampling, preventing the possibility of testing -This is not done often due to complications listed above -Fortunately, empirical therapy and drainage procedures will handle most infections -Some instances where sensitivity testing is indicated are: 1. Abscess is extensive at initial appointment, or becomes extensive 2. 1st or 2nd antibiotics chosen empirically are ineffective -Technique -Sample pus from the root canal or soft tissue surface -Soft tissue pus is preferred (less contamination from other organisms) -If no pus draining, dry off a swollen area → incise → insert paper point into pus → place paper point in transport medium → submit to lab within 30 mins -Is pus is coming from root canal → place rubber dam → access tooth → insert paper point into pus as it fills the canal → place in transport medium -Transport medium just protects anaerobic bacteria, does not culture the bacteria
Analgesics	-Maximum swelling is associated with pain reduces markedly -For a patient with diffuse swelling, a reduction in pain is a good sign

- Patient monitoring
    - Extensive abscess → evaluate patient each day until improvement
    - Moderate diffuse abscess → contact patient by telephone on a daily basis until improvement
    - Educate patient about: antibiotics, analgesics, hot saline holds PRN. Also, patient should get at least 8 hours of sleep per day, maintain high protein + caloric diet, and high fluid intake
- After the initial emergency appointment
    - When a swelling reaches its maximum size, it will stay that way for 48 hours
        - There is some changes throughout the day (grows when laying down, shrinks when standing)
    - After 48 hours, swelling starts to improve dramatically
    - RCT can be continued when swelling has disappeared
    - If the pulp chamber was left open for drainage, patient should continue ABX at the time the chamber is closed
        - This is to prevent re-development of an abscess
    - If there is no improvement
        - Incise if the soft tissue is now fluctuant and incision has not been previously done
        - Trephine foramen if not done previously
        - Change antibiotic (more broad spectrum)
        - Culture for sensitivity test if not previously done

Postoperative pain incidence related to the type of emergency treatment of symptomatic pulpitis

- Previous studies have tried to correlate patient age, gender, tooth type, intracanal medicaments, preoperative pain, and analgesics to the incidence of post-operative pain
    - Found that **preoperative pain** is a good predictor for postoperative pain
    - No correlation was found between operator factors (instrumentation, irrigants) and postoperative pain
    - Analgesics preventing postoperative pain was controversial (some supported, some found no correlation)
    - **This study looked at the incidence of postoperative pain after emergency pulpotomy, partial pulpectomy, and total pulpectomy procedures**
    - These procedures were performed by dental students
- Methods
    - Patients with irreversible pulpitis +/- acute apical periodontitis were selected
    - Study also collected the gender, age, tooth type, and type of endodontic emergency procedure performed
    - Treatment choices
        - Anteriors and bicuspids → complete pulpectomies done only
        - Molars → pulpotomy, partial pulpectomy, complete pulpectomy
            - Time constraint or calcified canals → partial pulpectomy
            - Patent canals, but >30 degree curve → pulpotomy
    - Treatment details
        - Pulpotomy: removal of coronal pulp with a curette → irrigate with 2.5% bleach → wait for hemostasis
            - Cotton pellet with pressure was used if hemostasis was not obtained initially
            - Squeeze dried cresatin cotton pellet was placed and access was covered with ZOE temp cement
        - Partial pulpectomy: removal of coronal pulp (curette) and pulp from the most patent canal
            - Pulpotomy was performed first
            - Then, pulp from the largest canal was removed with a barbed broach → irrigated with 2.5% bleach → dried with sterile paper points → cresatin pellet → sealed off like pulpotomy
        - Complete pulpectomy: removal of all pulp, up to 1mm of radiographic apex
            - After removal, chamber was irrigated with 2.5% bleach → dried with paper points → cresatin pellet → sealed off like pulpotomy
            - WL was obtained by estimating on a pre-op radiograph and then checking (same as UBC)
    - Post treatment
        - Patients were prescribed 600 mg aspirin, 400 mg ibuprofen, or 650 mg acetaminophen q4h for any post treatment pain. Drug of choice was tailored to medical history
        - Patients were instructed to call within 24 hours of treatment if pain increased beyond analgesic control
        - 3 faculty clinical instructors performed total pulpectomy + occl adjustment if patient had increased pain

- Results
    - Overall, 7.14% of patients reported increased pain beyond control of prescribed analgesics
    - Some interesting findings regarding incidence of postoperative pain
        - Gender: 4% males, 9% females
        - Age: 11% for 10~30yo, 6% for older groups
        - Tooth: 9% molars, 6% anteriors, 2% bicuspids
        - Procedure: 13% partial pulpectomy 8% pulpotomy, 6% complete pulpectomy
- Keep in mind that this study only looked at teeth that were vital, not necrotic

Root canal morphology of maxillary permanent first molar teeth at various ages

- Difficulties with the maxillary first molar
  - Radiographs of the roots are superimposed with the zygomatic bone
  - Radiographs are usually taken in the same plane as the complex anatomy of the root canals
- Root canal morphology and age
  - Young teeth have single large canals, but aged teeth have secondary dentin which yields separation of canals and transverse connecting systems
  - Compressed or roots with external grooves have the most complex root forms
- Method
  - 216 maxillary first molars were collected. Most teeth were from Caucasians and most were carious
  - Only teeth verified to be first molars were collected. An adjacent 2nd molar had to be seen to verify the extracted tooth was a first molar
  - X rays were taken of the teeth in 4 angles
  - Pulp was removed and checked by flushing water through the canals
  - Iothalamate solution was mixed with gelatin and injected into the tooth with a syringe
  - 4 radiographs were taken again to capture the root canal anatomy
  - Iothalamate solution was rinsed off and the MB root was sectioned at various levels
  - Crown was ground down to 1mm away from roots, and the pulp chamber cross section was recorded
- Results
  - Anatomical findings
    - 12/216 maxillary first molars had 2 roots
    - Most max first molars had an immature MB root at age 10, but some appeared mature at age 9
    - MB root in young teeth had a slit-like apical foramen, despite looking normal radiographically
  - Root canal morphology (Vertucci's classification used)
    - Palatal root: 97.7% type I, with no differences in any age group
    - DB root: 95.7% type I, with no differences in any age group
    - MB root: 26.4% type I, 27% type II, 46.3% had two or more foramina, only 1.9% type IV
      - 6~10 year old age group had more type I, with some type VI
      - Other age groups had no differences
  - Morphology of the MB root canal and orifice
    - 8 years old: one large canal → 10 years old: 2 directional calcification visible
      - Calcification happens in a M-D direction (splits root canal into 2), and B-L direction (narrowing of resulting canals)
    - Most MB roots had one canal, wide B-L but narrow M-D. Separate orifices were not common
    - All types of orifices could occur in all age groups, except the youngest
    - No correlation between canal system and type of canal orifice
    - Occasionally, entrance to a second canal was concealed by a rounded dentinal ledge on the mesial surface
  - Pulp chamber
    - 81% of pulp chambers were trapezoidal, of which 62.2% had parallel M-D walls
    - 11.2% of pulp chambers were triangular
    - 7.8% were elliptical
- Discussion
  - If the calcification of the MB canal started in the mid root, it appeared to result in a type 3 canal
  - If calcifications happened throughout in partitions, then a type 6 canal was the result
  - Type 2 and 5 canals was the result of further calcifications
  - The rounded dentinal ledge should be removed on the mesial surface to check for MB2
  - The use of a trapezoidal access preparation may be more beneficial than a triangular form

Incidence and Position of the Canal Isthmus: MB Root of the Maxillary First Molar

- Introduction
  - An isthmus is a narrow, ribbon shaped communication between 2 root canals and contains pulp
  - Also called a corridor, lateral interconnection, transverse anastomosis
  - Isthmuses in the MB root were reported as 4.9~52%
  - Vertucci reported 75% of isthmuses were in the middle third and 15% were in the apical third
- Method
  - 50 maxillary first molars stored in 10% formalin
  - Ultrathin separating disk was used to remove the MB crown + root in one piece
  - Transverse sections were made at 1mm increments from the apex to 6mm and stored in 5.25% bleach for 24h
  - Apical side of each section was stained with 2% methylene blue
- Results

TABLE 1. Canal configurations in the mesiobuccal root of maxillary first molars

Canal Configuration	No. of Roots	%
Type I	20	40
Type II	10	20
Type III	17	34
Type IV	3	6
Total	50	100

TABLE 2. Level of convergence of type II canals

Level from Apex (mm)	No. of Roots	%
6		
5		
4	3	30
3	3	30
2	4	40
1		
Total	10	100

TABLE 3. No. of canals at each level

Level from Apex (mm)	No. with 1 Canal	%	No. with 2 Canals	%
6	23	46	27	54
5	21	42	29	58
4	25	50	25	50
3	29	58	21	42
2	30	60	20	40
1	37	74	13	26

  - Note: the canal configuration table is NOT Vertucci's classification. It is Weine classification
    - Type I: 1 canal at each level
    - Type II: 2 canals that merged short of the apex
    - Type III: 2 canals at each level
    - Type IV: 1 canal that divided short of the apex
  - 1 canal was found in 40% of MB roots, and 2 canals were found in 60%
  - All canals classified as type II converged into 1 canal within 2~4mm of the apex
- Discussion
  - If the entire root had been sectioned, different types of canal systems may have been present
  - In apical surgery, this study shows the important of checking the isthmus between 2 canals when retrofilling
  - Failling to prep and seal the isthmus will cause failure of the retrofill

Problems encountered in tooth isolation and access to the pulp chamber space

- When accessing the pulp, the conservation of tooth structure should never preclude the proper design and execution of the access opening
    - Clinician must be knowledgeable about pulpal anatomy and external root anatomy
    - Failure to find a curved canal may be attributed to problems in canal preparation, when in fact it may be due to errors in access opening
    - Failure to properly seat GP and obturate may also be due to poor access
- Tips in tooth isolation and access opening preparations
    - If the rubber dam can't be placed on the tooth, clamp the adjacent tooth → isolate tooth of interest with cavit, Oraseal, medical adhesives, a rubber base, or floss
    - If isolation can only be achieved by clamping the gums, then CL is necessary prior to RCT
    - Remove temporary crowns prior to isolation and access (unstable reference, may break, may contaminate)
    - Draw a line on crown indicating angulation of roots
    - Exercise care when using long shank or surgical length burs
    - In calcified pulp chambers, only use slow speed burs for penetration through roof of the chamber
- Major errors in access openings

Failure to establish proper access to the pulp chamber and root canal system	-Bitewings are necessary to see the chamber space, especially with large restos present -Angled radiographs are useful in rotated teeth or with abnormal root configurations -Enter the chamber in a coronal-apical direction, with mesial-distal parameters -This is more efficient and less conducive to bur misalignment/misdirection -Also minimizes failure to differentiate pulp horns from canal orifices **Anterior teeth** -Remove the lingual ledge and incisal edge to obtain straight line access -The lingual ledge removal may uncover extra canals in mandibular 1, 2, 3, 4, 5's **Posterior teeth** -Ensuring the complete removal of the pulp roof -Measure the size and depth of the pulp chamber space by holding a bur next to the image of the crown on a radiograph (this is old school…) -When some of the roof is exposed, stick a safe-ended side cutting bur to laterally unroof the dentin -Use a 17 or 23 explorer to check for proper roof removal, no dentin overhangs -Visually inspect the chamber for straight line access -Remove cervical ledges to gain straight line access and allow entry in curved canals -Commonly happens in MB canals of maxillary first molars -Failure to remove cervical ledge restricts first 1~2mm of the canal, increasing the chances of ledging **Perforation** -Should be prevented by using radiographs to see the pulp chamber prior to accessing -If the perforation is above the bone, in the sulcus, or above the gingiva: -Control hemorrhage with cotton pellet/paper point +/- 1:50k epi or hemostatic agent -Seal with temporary cement (cavit, ZOE) -Proceed with RCT -Restore perforated area separately or make restoration part of total tooth restoration -If the perforation is below the bone or into the furcation: -Prognosis is very poor, surgical repair is futile, and tooth will likely need extraction/hemisection -In single rooted teeth, perfs in the cervical 1/3 have a guarded prognosis, but may be managed with surgical repair (perio) or orthodontics (root extrusion) -Protect all patent canals by temporarily blocking it with a file/GP/silver cone -Control hemorrhage. Gelfoam is good because it will stop bleeding + can be used as a scaffold for sealing materials afterwards -If bleeding cannot be stopped, then pack CaOH powder -Cavit/ZOE/alloy/Super EBA is packed into the perforation for sealing -Try to avoid pushing sealing materials into periradicular tissues -Dye the sealing material with methylene blue or red food colouring for easy identification

Failure to excavate all caries and remove unsupported tooth structure or faulty restorations	-Allows clinician to see if tooth is actually restorable -Ensures that an uncontaminated environment can be established for aseptic RCT -Prevents potential future issues: -Salivary and bacterial contamination -Inadequate assessment of restorative needs of the tooth -Loosening and packing of amalgam/composite into the canals -Fracture of the tooth between treatments → loss of reference point, or even tooth -Technique -Blood or pus leaking from the pulp will block visualization → risk for perforation -Therefore, peripheral caries is removed first, then caries is removed toward the pulp -Then, remove unsupported tooth structure or restorations -A sound restoration can be kept, as long as the access is done with plenty of water (eliminate debris) and walls are flares for straight line access -If restorative debris falls into the canal, it can be removed with an ultrasonic -Temporary restorations should be removed, unless it opens up new avenues of leakage. Crown lengthening may be preferable to deep temporary restorations
Failure to identify the angle of the crown to the root and the angle of the tooth in the dental arch	-Possible issues that may arise due to misangulation -Misidentification of canals (thinking ML canal is MB) -Inability to locate canal or missing extra canals -Undermining and weakening the tooth, possibly even perforation (maxillary 2's and mandibular 4's have the most significant angulations. They are often perforated due to this) -Prevention of misangulation -Initial access without a rubber dam may be helpful in getting correct bur alignment -Confirm bur depth and angulation on radiographs -If tooth is severely misaligned, remove specific portions of the crown to facilitate an unconventional yet satisfactory access into the pulpal space
Failure to recognize potential problems in access openings through crowned teeth or large restos	-Considerations when RCT'ing a crown -Crowns must be assessed for potential leakage under margins -Bitewings can be taken, but often crown obscures pulp anatomy -Root structure can be misaligned with the crown position -Artificial anatomy on crown cannot be used as a guide -Crowns can have alloy or composite buildups that impede direct access -Visibility is limited as it is dark -PFM crowns can fracture or craze during access -Inadequate water may leave metallic deposits in pulp -Crowns may obscure fractures in the tooth structure, especially in proximal walls or floor -Recommendations when RCT'ing a crown -Thorough radiographic evaluation to identify angled roots -Use a fiberoptic system for increased visibility -Prepare initial access through the crown without a rubber dam -Evaluate the shape of the alveolar process over the root surface below the crown -Prior to accessing, measure the bur against radiographs -Use water and proceed slowly with a high speed diamond -Advise patient of a potential fracture -Cut porcelain in light, shaving manner with a diamond bur. Then, use carbide for tooth -Once in pulp, only move laterally or do an outstroke movement -Flare the walls (if posterior) or lingual (if anterior) so RCT instruments don't touch the crown -Probe for possible areas of leakage/fractures/caries -Do not hesitate to extend the opening – crown strength comes from the margins, not occlusal surface -Irrigate coronal access before entering canals (to prevent pushing metals/composite into canals)

Problems in locating and negotiating fine and calcified canals

- 3 pulpal responses to irritation
  - Rapid death with canal patency
    - Overwhelming bacterial invasion or trauma → no reparative dentin → canal space remains patent
  - Irritational response with pulpal demise
    - Irritate pulp starts a reparative response prior to death
    - Appears as extensive diffuse calcifications, isolated pulp stones, or irregular linear calcification characterized by gnarled atubular dentin
    - These cases give a hint of a canal space in varying positions in the root
  - Extensive irritational response and pulp closure
    - Result of long term low grade irritation
    - Complete calcification canal closure, except for a few small areas with tissue remnants
    - Only a few % of calcified/blocked canals are considered untreatable by RCT
- Normal root canal anatomy VS calcified root canal anatomy
  - The canal space is always in the center of the cross section of the root
  - The pulp chamber is always in the center of the crown (before calcification)
  - When accessing a calcified tooth, the access is opened to the same size as if the tooth was normal
    - Bitewing or PA film should be used to gauge distance from cusp tip to pulpal floor
  - Clinician must know the geometric pattern of canal orifices in multi canalled teeth
  - Access preparations for each tooth type:

Maxillary 1, 2, 3	-Root canal is on the cross sectional center of the root -Ideal access prep would be through the incisal surface, but would violate esthetic and structural integrity -Access is done on the lingual side, 45 degrees to the long axis of the tooth -3~4mm of bur penetration will intersect with the pulp chamber in average sized teeth **In a calcified tooth** -If the pulp is not reached after 3~4mm, re-angulate the bur to the long axis of the tooth and drill down -This is to prevent perforation through the buccal surface -Proceed with drilling down the lingual aspect with frequent checking with the DG16 explorer for the orifice
Maxillary 4, 5	-Start on the center of the occlusal surface and follow the long axis of the tooth -Pulp is wide B-L but narrow M-D, so access with that in mind
Maxillary 6, 7, 8	-Most common access design is a triangle connecting the 2 buccal canals and the palatal **In a calcified tooth** -Mental image of the geometric pattern of canals is invaluable -It is common to find the first 1~2 orifices, but no additional orifices -Assume all maxillary molars have 4 canals until each potential site has been excavated with a #2 round bur to a depth of 1mm -Clinician should use the visible canals to approximate position of calcified canal -For example, a slot/trench is excavated palatally from MB1 to find MB2 -MB2 could also be 1~2mm into the MB1 orifice or even palatal orifice -Negotiate initially with a #8 K file to 2~3mm into the root -Once negotiated, flare coronal 1/3 via anticurvature filing -Then, move on to a #10 K file then a #20 Hedstrom file using the same method -Copiously irrigate with 2.5~5.25% bleach -Enlarge orifices with 2, 3, 4 GG drills or Hedstrom files -#2 will penetrate to mid root, #3 to 2mm shorter, #4 only for flaring orifice
Mandibular 1, 2, 3, 4, 5	-Most commonly a single canal, but a second canal can be present (almost always lingually) -Widen the orifice lingually to probe for a 2nd canal with a 8 or 10 K file bent @ apical 1-2mm
Mandibular 6, 7, 8	-Most common access prep is a trapezoid (wide B-L on mesial, slightly narrower on distal) -Distal orifice is usually wide B-L, and is 2 separate canals 30% of the time -Sometimes, the 2nd molar will only have 1 canal in each root, but wide B-L prep should still be done to assess orifice symmetry and geometry -If the 2nd molar truly has 2 canals, it will be along the M-D midline -If the mesial canal is not along this imaginary line, then it likely has 2 canals -Like the maxillary molar, MB or ML canal may be 1~2mm into the mesial orifice

- Negotiating the calcified canal – start to finish
    - Location and penetration
        - Most important instrument is the DG16 explorer
            - Firmly press the explorer into the pulp floor, and feel for a "stick"
            - Be careful in angulation, as the "press" could fracture the tooth and cause perforation
            - Leave the explorer in place and take a radiograph to confirm it is in a canal, to avoid perforation
            - If perforation happens
                - Offers a hint as to where the canal is oriented (example: if perf happens on mesial root of a mandibular molar towards the furcation, the canal is likely on the furcation side)
                - Only water should be used as an irrigant (don't want bleach leaking to tissues)
                - Mix CaOH and water and place slurry in the tooth
                - Hard setting CaOH (DyCal) should be avoided if the canal has yet to be located
                - Only when the canal has been found and treated, should the perforation be sealed
                - Explorer tip sized perforation: seal with thin layer of hard setting CaOH
                - >1mm sized perforation: same treatment, poorer prognosis
                - Failure often manifests as a perio defect, which is managed with perio procedures
        - Use a #8 or #10 K file to negotiate the canal. #6 is too weak and curl with pressure
            - Alternatively, use Canal Pathfinder files with greater shaft strength
        - If the file cannot be negotiated into the orifice, drill 1~2mm into it with a #2 round bur
            - Then, use the DG16 to re-establish the canal orifice
            - Make sure the tooth is **dry during drilling** → will create white chips → serves as markers for exploration or further countersinking
            - Cervical ledges **must be removed** as they cause a sharp curvature in the coronal 1~2mm
            - Particularly useful for MB2 in max molars or separating MB/ML in mand 2nd molars
            - If the canals exit at a severe curve, drilling this area may cause perforation. Avoid use of drills in this case, and use a sharply curved file instead
            - ETDA, RC-prep, and canal plus are seldom of use in finding orifices, but useful for canal negotiation
    - Negotiating the calcified canal
        - Once orifice is located, 21mm #8 K file is usually used for negotiation
        - Curve the apical 1mm of the file, and place a pointed rubber stopper indicating direction of curve
            - The curve of the file is aligned with the curve of the canal
            - Canal is not rotated, but should be advanced with a "stem winding" motion
            - File is advanced 1~2mm into canal and filed until the instrument can slide freely to that level
            - A #6 file can be used in extremely narrow canals, as forcing a #8 can cause blockage
            - Any file showing fatigue should be discarded
            - A pathfinding instrument (non cutting) can be useful in negotiating too
        - Irrigation with a chelating agent is not usually needed, but NaOCl is important
            - Forceful irrigation keeps debris in solution +lubricates the file, but does not dislodge calcifications
            - Full irrigation should be done for every 1~2mm advancement
            - Apical penetration of irrigant is increased as coronal canal becomes more patent
        - Once the file reaches estimated WL, #8 is filed until a #10 can reach within 1mm of WL and take an image
        - Use of ultrasonics
            - Penetration to the apex needs to be already established for ultrasonics to be effective
            - Combination of bleach and ultrasonic dissolves organic material and removes calcified debris
            - Ultrasonics also enhance the penetration of bleach
            - Be cautious not to force ultrasonic file apically, as it may ledge or create a new canal
        - False canal
            - Vigorous probing, filling, and use of chelating agents can create a false canal
            - Radiographs should be taken regularly to confirm the file is still following the canal
            - If perforation occurs, luckily it will be very narrow and can be filled like a normal canal
            - There is little value in finding the true canal, unless false canal was detected prior to perf'ing
            - Tooth will likely need surgical intervention if RCT fails

- Completely calcified canals
    - It is common to find total blockage of the canal space at any level
    - However, it is seldomly completely calcified to the apex
    - If symptoms are absent and there is no periapical pathosis, it is acceptable to instrument and fill up to the level of negotiation and just observe
    - If tooth is symptomatic or there is a periradicular lesion, a surgical approach may be indicated
- Summary
    - Copious irrigation with bleach helps with dissolving organic debris, lubricating the canal, keeping dentin chips and calcified material in solution
    - Advance instruments slowly
    - Clean instrument on withdrawal and inspect before reusing
    - When a fine instrument has reached the apex, do not remove it and take a radiograph
    - Do not use acids or alkalis in canal penetration
    - Use chelating agents to assist in canal penetration
    - Use ultrasonics to loosen debris

Determination of the Minimum Instrumentation Size for Penetration of Irrigants to the Apical Third of Root Canal Systems

- Methods
    - Selection criteria: freshly extracted mandibular molars with 2 mesial canals, 20~23mm long, 15~25° curvature
    - Straight line access was achieved, and a #10 K file was instrumented 0.5mm short of the apical foramen
    - Apical foramen was covered in wax so operator couldn't see it
    - MB canals were instrumented with crown-down and rotary files
    - Apex was enlarged up to one of 5 sizes: #20 (group 1), #25 (group 2), #30 (group 3), #35 (group 4), #40 (+/- control)
    - Irrigation protocol
        - Group 1/2/3/4 and + control: 5.25% bleach after each instrument and a final irrigation of 17% EDTA x 5 mins, 5.25% bleach x 5 mins, distilled water x 5 mins
        - - control: no EDTA, just 5.25% bleach x 5 mins, distilled water x 5 mins
    - Imaging of the canal
        - Mesial root was split B-L into 2 long halves
        - Roots were incubated, dessicated, and coated in gold palladium and imaged under a scanning electron microscope
        - Root canal surface was graded from 1~8 (1 = no debris/smear layer, 8 = complete coverage of debris/smear layer)
- Results
    - **Positive control group**: all had no smear layer, significant erosion at the dental tubules → score = 1
    - **Negative control group**: all had a smear layer and debris → score = 8
    - **Group 1 (#20 file):** all had a smear layer and debris → score = 8
    - **Group 2 (#25 file):** 76% removal of debris and smear layer → score 3.4
    - **Group 3 + 4 (#30 + #35 file):** 100% removal of debris and smear layer → score 1
- Discussion
    - Various papers quote #25~#40 file being necessary for adequate tissue and debris removal
    - This paper recommends #30 is enough for adequate cleaning while conserving as much root structure as possible

Taxonomy, ecology, and pathogenicity of the root canal flora

Table II. Bacteria isolated from root canals of teeth with periapical lesions (% incidence)

Bacteria	*Percentage of incidence*
Fusobacterium nucleatum	48
Streptococcus sp.	40
Bacteroides sp.*	35
Prevotella intermedia	34
Peptostreptococcus micros	34
Eubacterium alactolyticum	34
Peptostreptococcus anaerobius	31
Lactobacillus sp.†	32
Eubacterium lentum	31
Fusobacterium sp.‡	29
Campylobacter sp.	25
Peptostreptococcus sp.§	15
Actinomyces sp.	15
Eubacterium timidum	11
Capnocytophaga ochracea	11
Eubacterium brachy	9
Selenomonas sputigena	9
Veillonella parvula	9
Porphyromonas endodontalis	9
Prevotella buccae	9
Prevotella oralis	8
Propionibact. propionicum	8
Prevotella denticola	6
Prevotella loescheii	6
Eubacterium nodatum	6

- Introduction
  - Obligate anaerobic bacteria dominated infected root canals and composed up to 90% of the flora
  - Difficult to isolate and culture root canal bacteria as old cultures were done in broth, which favoured fast growing bacteria. Was not a representative sample of the actual flora
  - Necrotic teeth due to trauma (without apical periodontitis) were found to have no bacteria
  - New organisms and taxa are continuously described and reclassified. An example would be *Bacteriodes melaninogenicus*, which was labelled for all black pigmented gram – anaerobic rods. Now, this species has been subdivided into 8 different species
- Taxonomy of root canal flora
  - *Fusobacterium nucleatum* was the most frequently isolated
  - Peptostreptococcus family was the most common family: *P. anaerobius* and *P. micros* were present in >1/3 of samples
  - Most common strep species: *S. anginosus* and *S. mitis*
  - Actinomyces *A. israelii* was implicated in RCT failure due to ability to infect periapical tissues. It was seen 11% of the time, and the actinomyces family was seen 15% of the time
  - In summary, root canal flora is dominated by anaerobic bacteria. Facultative anaerobic bacteria like streptococci also make up a significant part of the flora, especially if pulp is exposed to the oral cavity due to caries
  - Aerobic bacteria are very rarely found in initial infections, but may be introduced during treatment
- Microbial ecology in the infected root canal
  - Bacteria in root canal infections are a restricted subset of the bacteria found in the oral cavity
  - Most species found in infected root canals have also been found in the periodontal pocket
  - **During the course of infection, inter-relationships develop between microbial species and population shifts are produced as a result of these interactions**
    - Example: *P. oralis* cannot survive on its own in a root canal, but are found in established flora
    - Anaerobic bacteria gradually outnumber facultatively anaerobic bacteria in 3+ month long infections
    - Even when initial inoculation had equal numbers of bacteria, anaerobic always outnumbered in the end
  - Main selective pressures are: **availability of oxygen and nutrients**
    - Microorganisms use connective tissue disintegrates to make simple peptides and AA's for energy
    - *Peptostreptococcus micros* has a wide range of peptidases which can feed other bacteria as well
    - Bacteria reliant on fermenting carbohydrates will not be viable
    - A "food chain" of products can be established where some bacteria feed off the by-product of others
  - *"F. nucleatum*, which was the most prevalent species, was positively associated with *P. micros, P. endodontalis, C. rectus,* and *Selenomonas sputigena*."
- Pathogenicity of endodontic flora
  - Study was performed by inoculating monkey pulps with individual bacterial species, or a combination of them
  - Single strain inoculation caused a mild PA infection, whereas combinations caused severe PA infections
    - This highlights the power of bacterial synergy in its pathogenicity
    - Synergy can come from protection (obligate anaerobes interfere with phagocytosis of facultative anaerobes), nutrient "food chaining", or reducing the oxygen concentration
  - **All endodontic abscesses harbored one or more of the bacteroides (Prevotella + Porphyromonas) species**
    - *Prevotella intermedia (63%), Porphyromonas endodontalis (53%), prophyromonas gingivalis (12%)*
    - Other less major prevotella/porphyromonas species: *P. intermedia, P. loescheii*
    - These bacteria survive by preventing opsonization via degrading phagocytic plasma proteins
    - Also, *intermedia, gingivalis,* and *endodontalis* can resist phagocytosis via capsulation
  - *Actinobacillus actinomycetemcomitans* can directly kill leukocytes, but is very rarely present in infected root canals
  - *Actinomyces israelii* (+ actinomyces in general) and *Propionobacterium propionicum* can establish periapical infections, but are less acute than Prevotella/Porphyromonas. These infections are more smouldering and occasionally suppurative

Microbiologic factors in endodontology

- Enamel caries
  - Bacteria have been found under the enamel prior to cavitation, called white spot lesions
  - No inflammatory changes to the pulp were found in enamel caries though
- Dentinal caries
  - Teeth with dentinal caries and reversible pulpitis were found to have Prevotella and Porphyromonas (gram -)
  - Chronic inflammation was found in the pulps under these lesions
  - Many types of bacteria have been found in carious lesions: gram -, gram +, anaerobic strep, proteolytic bacteria
    - Shallow caries: facultative gram + rods
    - Pulp depth caries: facultative cocci and anaerobic rods
  - A pulp exposure doesn't need to be present for bacteria to be present in the pulp
    - Obligate anaerobes were found in pulp in teeth that did not have a pulp exposure
    - *Eubacterium, Propionibacterium,* and *Actinomyces* predominated
    - Other obligate anaerobes were lactobacilli, peptostreptococci, veillonella, and streptococci
  - Bacteria are primarily responsible for periapical lesions
    - A study showed that pulp exposures in germ free environments showed minimal inflammation
    - Pulp tissues even formed dentinal bridges over the exposed pulps
- Periapical infections
  - Vital pulp tissue acts as a barrier to periapical inflammation
  - Periapical lesions occur when the root canal becomes infected
  - Infected pulps start with mostly aerobes, but progresses to an obligate anaerobic dominated environment
  - Infection by aerobe or compromised blood supply in pulp → produces an environment of low O_2 → flora anaerobes infiltrate as secondary invaders → spreads out the periapex
  - **In dental abscesses, the most common pathogen is the Millieri group streptococci**
- Microorganisms in periapical lesions

Study	**Examined**	**Results**
Tronstead et al	-Type of bacteria present on the root tips of failed endos	-Root tips showed *Propionibacterium acnes, Bacteroides (Porphyromonas) gingivalis,* and *Porphyromonas endodontalis*
Nair	-Bacteria present on 30 granulomas and 1 radicular cyst	-All canals were infected with cocci, rods, filamentous organisms, spirochetes
Nair et al	-Therapy resistant periapical lesions	-Bacteria and yeast were found
Iwu et al	-Asymptomatic PA lesions	-2/16 samples had surface growth -14/16 samples contained microorganisms
Brown and Rudolph	-Spirochetes in pulp	-14% of pulp samples contained spirochetes, but could not be cultured
Hampp	-Spirochetes in pulp	-Small treponemes, the most anaerobic of oral bacteria were present
Trope et al	-Spirochetes in abscesses	-30~60% of periodontal abscesses had spirochetes -<10% of endodontic abscesses had spirochetes

- Survival mechanisms of pathogens

Adhesion of bacteria	-Laminin (non collagenous high MW protein) and fibronectin are involved in adhesion -Laminin is found in *Staph aureus*, the most common cause of severe infection
Sialic acid	-Sugar molecule coating that helps microorganisms gain entry into a human host -Viruses may live in cells without mounting any immune response
M protein	-M protein is on the bacterial cell surface, and enhances entrance + multiplication in eukaryotic cells -Pathogenic group A streptococci (*Strep* pyogenes) resist phagocytosis by PMNs due to M protein
Encapsulation	-Encapsulated strains, or even the purified capsule polysaccharide caused abscesses -Capsulated bacteria have a far less rate of being phagocytosed
Resistance to phagocytosis	-Endotoxins made by bacteria can resist ingestion by PMNs -Even after ingestion, intracellular killing is impaired. Pathogen could multiply in the phagocyte (staphylococci), or microbe may leave debris that can trigger chronic inflammatory sequelae -Once ingested, bacteria can inhibit the respiratory burst, shield itself with a capsule, inhibit fusion of the phagolysosome, etc
Alteration in neutrophils	-*Porphyromonas gingivalis* can alter the shape of neutrophils → cells are less polarized → inhibited motility
Resistance to antibiotics	-Resistance genes can be transferred from bacteria to other bacteria -Local root canal infections can develop resistance to antibiotic therapy

- Symptomatic root canal infections
    - There is a dominance of anaerobic bacteria in the root canals of necrotic pulps and periapical inflammation
    - In periapical abscesses, anaerobic gram negative bacteria predominate
    - Anaerobic bacteria such as porphyromonas, prevotella, and peptococcus are associated with clinical symptoms through the production of various cytokines
        - Various cytokines → various host tissue responses → formation of periapical lesion
    - Some studies suggest a relationship between root canal flora and clinical symptoms, but these studies did not contain a control sample
    - Other studies looked at certain microorganisms causing pain or odor, but that was inconclusive as well
- Lipopolysaccharides
    - Gram - bacteria (prevotella, porphorymonas, fusobacteria) have polysaccharides and phospholipids as a cell wall
    - The amount of lipid determines the endotoxic properties of the LPS cell wall
    - LPS is a major virulence factor
        - Activates complement without antibodies
        - Complement activation generates anaphylatoxins (C3a, C5a) which cause vasodilation and smooth muscle contractions
        - Neutrophils, mononuclear cells, platelets, mast cells, basophils, and endothelial cells are activated
        - B lymphocytes and macrophages are accelerated to maturity
        - Complement also activates kinins, which also mount an inflammatory response
        - LPS binds to endogenous LBP to form a complex → this complex binds to CD14 → binds to macrophage → macrophage releases IL1, IL6, IL8, TNFa, reactive oxygen, nitric oxide, interferons, PG's
    - Endotoxin in root canals
        - Endotoxins were found to be concentrated in the root canal in necrotic pulps
        - Even small amounts of endotoxin are capable of inducing a periapical inflammatory response
        - However, not all researchers agree that endotoxins play a major roll in inflammation
        - Endotoxins could possibly benefit the pulp by stimulating repair mechanisms by the immune system
        - There is also a degree of tolerance that forms with chronic exposure to endotoxins
        - *Porphyromonas endodontalis* has been specifically involved in odontogenic infections, and not other oral or non-oral infections
        - Porphyromonas/Prevotella could not induce abscesses by themselves, but they were significantly present in a mixture of other bacteria in teeth with purulent inflammation
        - Porphyromonas/Prevotella have also been associated with periodontitis, pregnancy gingivitis, and ANUG
    - Endotoxin and pain
        - Endotoxins could cause pain by increasing blood flow and neurotransmitter responses at nerve endings
        - Endotoxin activating Hageman factor → bradykinin → pain mediator
        - There was a significant association between *Prevotella melaninogenicus* (an anaerobic gram negative rod) and pain, foul odor, and sinus tracts. However, no relationship was found between this organism and periapical rarefaction
        - *P. gingivalis* and *P. endodontalis* was only found with acute infections
- Gram positive cells
    - Although gram - anaerobes dominate endodontic lesions, this does not negate the fact that gram + bacteria may also be involved in infections and flareups
    - Peptidoglycan
        - Gives rigidity to the bacterial cell wall, and makes up 40% of the cell mass
        - Induces IL1, TNFa, cytokines, oxygen radicals, nitric acid, eicosanoids, complement, B cell stimulator
        - Degraded by leukocyte hydrolases
    - Lipoteichoic acids
        - Can bind to lymphocytes and macrophages, and activate them
        - Also induces bone resorption and complement cascade
    - *Streptococcus mutans* have been shown to induce mild pulpal and periapical inflammation. However, only a mixture of organisms caused the full blown response seen in periapical lesions
- Current nomenclature for the black pigmented bacteria, the Bacteroides
    - Prevotella (saccharolytic) species: melaninogenicus, denticola, loescheii, intermedia, corporis
    - Porphyromonas (asaccharolytic) species: asaccharolyticus, gingivalis, endodontalis

Eradication of endodontic infection by instrumentation and irrigation solutions

- Introduction
  - There is no difference between root canals treated in one or two appointments
  - Some studies show that RCT prognosis does not change even though the root canal was positive for cultures
  - Nevertheless, there is a general agreement that causative agents have to be removed for success
- General strategy of infection control
  - Success in endodontic disinfection depends on: host defense, systemic antibiotics (occasionally), instrumentation, irrigation, intracanal medicaments, root canal filling, and a coronal restoration
- Composition of bacterial flora in infections
  - In primary apical periodontitis, strictly anaerobic bacteria are preferred
    - However, microaerophilic and facultative bacteria are often present as well. Examples are Actinomyctes, Lactobacillus, and streptococci
    - It is rare to see a mono-infection
  - In recurrent root canal treated infections, the most predominant species is *Enterococcus faecalis* (facultative)
    - *E. faecalis* is often found as a pure culture, but it is also found together with other facultative species like streptococci, lactobacilli, and even anaerobes
    - Gram negative enteric rods (coliforms, pseudomonas) and yeasts are almost entirely found in previously root filled teeth with apical periodontitis
  - Dentin invasion
    - Diameter of dentinal tubules large enough to cause bacterial penetration
    - Dentinal invasion was found in 50~80% of teeth with apical periodontitis
    - Primarily gram positive facultative/anaerobic cocci/rods, but gram negative also seen
    - Tubules are filled at random, some infected tubules are surrounded by several empty tubules
    - Invasion more effective at coronal and middle portion of the canal
    - Root cementum destruction in chronic apical periodontitis can cause bacteria to invade the full thickness of the dentinal tubules
    - Studies show that after instrumentation, the bacteria in the tubules and accessory canals remain
- Technical and biological goals of instrumentation
  - Goal is to remove all necrotic and vital organic tissue and some hard tissue
  - Give the canal system a shape that allows easy debridement and filling
  - However, instrumentation does not disinfect the canal
    - A 100,000 fold decrease in bacteria was found, but it was far from being disinfected
    - Only when an antibacterial irrigant (bleach) was added, was it acceptably clean
    - Another study found that instrumenting the apex to #40 vs #30 significantly reduced the numbers of cultivatable bacteria
- Irrigating solutions
  - Bleach
    - Active agent is hypochlorous acid, HOCl
    - Disrupts oxidative phosphorylation and other membrane associated activies
    - 2 bleach solutions were studied:
      - 5.25% bleach, pH 11, no buffer
      - 0.5% bleach, pH 9, bicarbonate buffer (AKA Dakin's solution)
      - No differences in antibacterial properties or removal of organic tissue were found
    - Bleach performance
      - *E. faecalis* and *C. albicans*: 5.25% killed them in 15~30s, 0.5% killed them in ~30min
      - *Porphyromonas gingivalis, P. endodontalis, P. intermedia*: 5.25 and 0.5% kill them in ~15s
      - However, these were *in vitro* results. *In vivo* tests show disappointing results
        - Mostly due to difficulty in the bleach to reach the apex
        - Also due to dentin shavings interfering with killing bacteria
  - Chlorhexidine
    - Permeates cell walls or outer membranes and affects bacterial/yeast membranes. In viruses, CHX is only effective in killing lipid enveloped ones
    - *In vitro* studies show CHX to be superior to bleach in killing *E. faecalis* and *Staph aureus*
    - Much less toxic to human tissues compared to bleach
    - However, activity is pH dependent and is greatly reduced in the presence of organic matter/dentin
    - 0.2~2% CHX is a good alternative for root canal irrigation, but additional studies are needed

- Chlorhexidine + peroxide
  - Hydrogen peroxide used in combination with CHX found that they killed *E. faecalis* in vitro in lower concentrations than if they were used together
  - Postulated that the CHX opens up pores in the microbe, allowing H_2O_2 to penetrate deep into the DNA to damage it, but not completely known
  - More studies needed regarding cytotoxicity of this combination
- H_2O_2
  - Available in 1~30%
  - Greater activity against gram positive than gram negative
  - Some bacteria can make enzymes (superoxide dismutase, catalase) to protect against H_2O_2
  - *Ex vivo* study showed that H_2O_2 + bleach was no more effective in irrigation than just bleach
  - As stated above, H_2O_2 alone was not as good as in combination with CHX
  - Has long been used in endodontics as a precursor to other antibacterial irrigants, but there are not enough studies yet to fully evaluate and assess its use
- Iodine compounds
  - Pure iodine compounds (mostly I_2) are less reactive than bleach, but rapidly germicidal
  - More stable forms of iodine (povidone, poloxamer) are longer lasting but less active against certain fungi and spores
  - Mechanism not fully understood
  - *In vitro* found that like other irrigants, it is hindered in the presence of organic matter. Dentin powder effectively made 0.2~0.4% iodine potassium iodine solution useless against *E. faecalis*
- EDTA
  - Found in 17% concentration, with a pH of 7
  - Little, if any antibacterial activity. Even if it was antibacterial, would need to be left for a long time
  - Removes smear layer by chelating inorganic material
  - Removing the smear layer improves the antibacterial effect of other agents
- Citric acid
  - Ranges from 1~50% concentration
  - 10% CA was more effective in removing the smear layer from apical root end cavities than ultrasound
  - Citric acid does a better job at killing bacteria than EDTA, but no studies have been done on its antibacterial effect
  - Some papers say EDTA is better, others say citric acid is better at removing the smear layer
  - Both citric acid and ETDA are food agents
- MTAD
  - Mixture of tetracycline (doxycycline), citric acid, and detergent (Tween 80). Combined pH of 2.15
  - New agent aimed at addressing the major weaknesses of all other irrigant solutions
  - When used after bleach, it effectively removes the smear layer without opening up the dentinal tubules, like acids do
  - EDTA caused more dentin erosion in middle and coronal 1/3 than MTAD
  - In apical 1/3, MTAD left a cleaner canal than EDTA
  - MTAD used alone left some organic components on the smear layer, hence the use of bleach prior
  - MTAD is less cytotoxic than eugenol, 3% H_2O_2, CaOH paste, 5.25% bleach, CHX, and EDTA

- Rotary instruments
  - Popularized due to less time instrumenting and claims for better pulp tissue removal
  - Studies show hand instruments are still better at pulp removal, but rotaries preserve the canal shape better
  - In dental students: zips and elbows occurred less frequently with rotary than manually, correct preparation length was achieved more often with rotaries
  - Frequency of file breakages were the same as hand instruments
- Molar canals vs single canal teeth
  - Molar roots have 2+ canals in a root, with complex network of anastomoses
  - Cross section of roots is often oval with narrow extensions
  - Most molar canals curve, sometimes severely

- Size of apical preparation
    - Operator has little control over how the file behaves when instrumenting the apex
    - Shaping of the apical canal can best be accomplished by reaming
    - Largest apical foramen was the distal root of mandibular molars, with an average of size #40
    - Wider apical prep (#50~#80) results in greater reduction in bacterial numbers
    - Apical cleanliness is more dependent on instrument size and less about canal length
- Working length vs apical foramen
    - If radiographic apex was the landmark for endo, overinstrumentation would be happen in majority of teeth
    - Teeth filled 2mm or more short of the correct length have the worse prognosis
- Sonic and ultrasonic preparation
    - Ultrasonics were found to energize the irrigant and cleaned the canal better than hand instrumentation
        - Steady streaming and cavitation microstreaming contributed to enhanced cleaning
        - Precurving the files decreased amount of debris, but not the smear layer
    - Sonic/ultrasonics were found to leave about the same amount of debris and smear layer as hand instrumentation
        - When EDTA was used with ultrasonics after bleach irrigation, smear layer was much reduced
        - Stepback instrumentation with ultrasonic preparation was superior to hand instrumentation alone
        - It is possible that in canals with complex anatomy, ultrasonics will do a superior job than normal irrigation
    - Risks of ultrasonics
        - Risk of transportation was greater with ultrasonics, and more severe in canals with curvatures >30°
        - Resulted in severe straightening, zipping, and strip perforations
        - Recommended to pre curve the file to reduce straightening forces
- Lasers in root canal disinfection
    - Nd:YAG laser was inferior to bleach irrigation in disinfecting a canal containing *E. faecalis*
    - CO_2 laser was inferior to bleach irrigation
    - Studies found lasers were good at being an antibacterial, but could not predictably cause sterility
    - Combination of lasers and sonic preparation produced the cleanest canals with open tubules
    - Despite smear layer removal, cleaning with a laser is difficult, and thermal damage is possible
    - Although proven to be effective, they are inferior to bleach and are less effective in complex anatomy
- New developments in root canal disinfection
    - Ozone, photoactivated disinfectants, electrochemically activated water are being studied
    - Bleach is the MVP
- Instrumenting techniques and amount of apical extrusion
    - Apical extrusion can cause symptoms due to stimulation of inflammatory mediators
    - Step preparation had the most extrusion, followed by standard technique > crown down > ultrasound
    - All methods caused extrusion out the apex
- Overinstrumentation
    - With the exception of #6 and #10 files, instrumenting out of the apex could cause:
    - Trauma to periapical tissues
    - Extrusion of necrotic canal contents, potentially causing a flare-up, bacteremia, periapical actinomycosis
    - Stimulate bleeding into the root canal and provide nutrients for intracanal bacteria
    - Increase foramen size and chances of bacterial infiltration
    - Chance of extruding irrigating solutions causing postoperative pain and discomfort
    - Extrusion of sealer or filling materials
    - Creation of an oval apical foramen
- Instrument fracture
    - 19% higher failure rate
    - Can be removed using an ultrasonic instrument or through apical surgery
    - If removal has a poor prognosis, 3 months of CaOH placed in the canal for disinfection can be done
    - As long as canals were adequately instrumented prior to breakage, prognosis is fine
- Interappointment medicament
    - Not needed when treating a vital pulp, because the canal should be sterile
    - CaOH is still recommended if Tx is in multiple appointments, to ensure canal remains sterile
    - In teeth presenting with apical periodontitis, there is undoubtedly bacteria after chemomechanical preparation. CaOH was effective in sterilizing the canals after 4 weeks, but results are debated (some show remaining bacteria, some show 7 days is enough)
    - CaOH also neutralizes bacterial LPS and makes necrotic tissue more soluble to bleach
    - It has been speculated that absolute disinfection may not be necessary, as a high quality endo will entomb remaining bacteria, and the filling materials will be antibacterial themselves

Rationale and efficacy of root canal medicaments and root filling materials with emphasis on treatment outcome

- Reversible pulpitis to apical periodontitis
  - Must know that the steps from reversible pulpitits to apical periodontitis is a continuum
  - When bacterial infiltration causes some necrosis, the pulp mounts an inflammatory response and is categorized as irreversible pulpitis
  - With continued aggravation, the pulp will try to continue to heal but will eventually die. This is called pulp necrosis and it may lead to apical periodontitis
  - Apical periodontitis happens as the body tries to prevent bacterial invasion into the bone
  - **It is possible to have bone resorption before total pulp necrosis if the process happens quickly enough**
    - This would explain why pulp vitality can test positive in some cases with an apical radiolucency
  - Some bacteria can colonize the root surface, but not cause bone resorption
  - Trauma can also kill the pulp, but not cause bone resorption because there is no bacterial infiltration
  - Since it is a continuum, it is possible that coronal pulp is severely diseased, whereas apical pulp is normal
  - This huge range of conditions and variables makes a proper diagnosis difficult
- Facts about treating apical periodontitis
  - Why are RCT's successful in primary apical periodontitis?
    - Primary apical periodontitis cases are usually colonized by obligate anaerobes
    - Anaerobic microflora is every sensitive to ecological changes
    - RCT seals off the supply of nutrients
    - Usually, bacteria are only in the canal and not out the apex, even when apical periodontitis is present
    - A zone of PMN's surrounds the apical foramen, preventing bacteria from spreading any further
  - What about recurrent apical periodontitis?
    - Primarily due to *Enterococcus faecalis*
    - If the bacteria can survive the treatment process, they are likely to be more resistant
  - Lateral periodontitis
    - Result of an infection in a lateral canal
    - Gram positive species (streptococci, enterococci, actinomyces, lactobacilli) can invade dentin tubules more readily than gram negative species
    - Are usually beyond the reach of mechanical preparation, and have to rely on irrigation
  - Treat as early as possible
    - At the beginning of the infection process, the pulp is sterile and bacteria only reside on the surface
    - At this stage, treatment can be done with a high degree of asepsis
    - As infection progresses, bacteria can invade the tubules
    - The further apical the infection spreads, the more difficult the disinfection will become and hence the chances of successful treatment
    - In order to achieve an optimal result, pulp inflammation should be diagnosed before necrosis, and necrosis should be diagnosed before apical periodontitis
- Pre-treatment preparation
  - Rubber dam tooth isolation
  - Tooth surface cleaned with 30% H_2O_2 and 5% iodine in alcohol. Alternatively, 0.5% CHX in alcohol works too
- Irrigation during treatment
  - Paired with effective suction, irrigation today is the most effective way of evacuating tissue from canals
  - A sterile canal can be irrigated with just saline, but necrotic pulps will need a better solvent like bleach
  - In the past, quaternary ammonium salts were popular irrigants
    - Is a detergent, so good at cleaning fatty deposits
    - Very effective with low toxicity during use in WWII, but less so in endodontic procedures
    - Tissue toxicity ended up being higher than expected
  - Today, commercial laundry bleach is mixed with water for patient care use
    - Does not substantially change the pH, but makes bleach less irritating
    - 1% sodium bicarbonate may also be used as the diluent
    - Bleach over 0.5% is considered unnecessary
  - Chlorhexidine, citric acid, and EDTA → see previous reading
  - Ultrasonics may better agitate the irrigant and improve cleaning, but only to the coronal part of the canal → see previous reading for more details

- Rotary instrumentation
  - Rotary instruments popularized Ni-Ti files over traditional stainless steel files
  - Rotary is much faster, but is not better than hand instrumentation at eliminating microbes
  - Originally shaped teeth via grinding (high torque, negative rake angles) at maximum of 300 RPM
  - Nowadays, rotary file design is different
    - LightSpeed: only the apical few mm are cutting, so speed can be up 1500~2000 RPM
    - Positive rake angles allow cutting rather than grinding and result in less friction. Speed can therefore be increased to 500~600 RPM
  - Rotary instruments used to have standardized 2% taper (0.02mm/1.00mm)
    - Was susceptible to "taper lock" → when the canal is instrumented with one file, there is a defined taper created in the canal
    - When the next file of the same taper is inserted, it locks in at every level of the canal
    - Because the file is fully engaged from coronal to apical, it can break easily
  - Nowadays, variable taper instruments and positive rake angles reduce the risk of taper lock
- Interappointment dressing
  - Idea is to reduce the bacterial content in the root canal between appointments
  - Traditionally used phenol or formaldehyde. Cotton pellets were soaked in these and left in the canals. The vapours were assumes to elicit the antibacterial effect
    - Phenols do not have antimicrobial vapours
    - Formaldehyde does have antimicrobial vapours, but questionable on its ability to reach the apex
    - Without direct contact, its effect on the apical region is unreliable at best
  - Calcium hydroxide
    - Intracanal medicament of choice
    - Generally safe to be extruded out the apex, but one reported case of spread in vascular bed and led to serious complications
    - CaOH is slow acting, must be left for at least 1 week. If well packed and left for sufficient time, it will completely disinfect the canal with high predictability
    - Found to interfere with bacterial LPS and lead to its degradation
    - CaOH + water
      - **Water must be present for the antimicrobial effect of CaOH**
      - 0.17% soluble in water (most of it forms a slurry)
      - Has a thixotropic behaviour (thick when idle, fluid when agitated)
      - This is why syringed applicators (CaOH is viscous) are worse than lentulospiral application (CaOH becomes less viscous)
    - Calcium hydroxide alternative methods of delivery
      - Glycerin: CaOH dissolves better, but does not yield OH ions → no antibacterial effect
      - Methylcellulose (PulpDent): delivers less OH ions than CaOH + water
      - Sealer cements: CaOH cannot raise pH enough in roots, so not valuable
      - CaOH with other antimicrobials (formaldehyde, phenol, CHX) makes CaOH less effective and adds unnecessary irritation
    - CaOH resistance
      - *Enterococcus faecalis* has a proton pump to control pH, and may confer resistance
      - 11% of retreatments had CaOH resistant bacteria
      - An additional medicament of 2% iodine potassium iodide may be needed to target the enterococci
- One visit treatment controversy
  - With proper instrumentation and irrigation, only 40~70% of canals were disinfected successfully
  - Theoretically, killing could continue due to the sealer and blocking nutrient access, but there are no studies
  - Vital pulps
    - Safe to assume the pulp is bacteria free, so the canal should be instrumented and sealed in one appt
    - Temporizing and medicating will only increase risk of contamination
  - Necrotic pulps
    - Although necrotic pulps don't always have bacteria, always assume it does
    - Asepsis is important, but antisepsis is the key to successful treatment outcome
    - There is no evidence that good instrumentation and the best antimicrobial use will predictably eliminate all bacteria in an infected root canal
  - Retreatments – one dressing is not enough, at least 2 dressings with CaOH are needed before obturation

- Root canal filling
  - Is a root canal filling even necessary?
    - Theoretically, it would not need to be obturated if the root canal can be completely sealed
    - Realistically, there is leakage through restoration margins, tooth, or circulation
    - Also, a hydraulic seal may be needed when placing a post
  - Desirable traits for root canal filling
    - A good predictable root filling material has not yet been developed
    - A hydraulic seal of the root canal space is the ideal outcome
    - Must withstand the repeated compression and flexure of the root
      - Gutta percha becomes brittle with age and can break the seal
  - Gutta percha filling method
    - Cold lateral condensation is the most common method
    - Warm vertical/3D filling is another popular technique
    - GP carriers (Thermafil) allow thermoplasticized GP to be introduced to WL to the same efficacy as lateral condensation. However, they frequently overfill patent canals
    - QuickFill and MicroSeal are rotary thermocompactor systems that extrude GP between 80~135°C
  - Gutta percha
    - Most common root canal filling material
    - 20% gutta percha, 70% zinc oxide (antibacterial), some additives
    - Gutta percha comes in α and β crystalline structures, but α is used for endodontics
    - Softens at 64°C
    - Low degree of toxicity, but small particles can stimulate immune activity
    - Must be used with a sealer to try to obtain hydraulic closure
  - Sealers
    - The most toxic of the materials used for obturation
    - Most commonly based on zinc eugenol cements and some resins
    - Hydrolyzes into zinc oxide (antimicrobial) and eugenol
    - **AH26 and AH sealer**: toxic when freshly prepared (due to formaldehyde), but lowest toxicity after 24h
    - **Diaket sealer**: adhesive sealer. Highly toxic, causes long term irritation, and tissue necrosis
    - **Endofill and RSA RoekoSeal**: silicone type sealers with remarkably low toxicity
    - **Sealapex and Apexit**: CaOH containing sealers, but not supported by any evidence
    - **KetacEndo**: glass ionomer sealer with low toxicity. Some evidence supporting use
    - **Rosin Chloroform**: contains 5~8% rosins that are toxic. After chloroform evaporates/absorbs, the leftover resins remain irritating
    - **Chloropercha**: white gutta percha and chloroform, toxic due to chloroform
    - **Kloropercha**: 20% gutta percha, 50% zinc oxide, balsam, and rosins. Balsam and rosins are irritating
  - Overfilling
    - Overfilling has generally been associated with lower chances of clinical success
    - However, overfilling in a **sterile canal** did not increase failures
    - Overfilling during retreatment resulted in a 60% failure rate
    - Appears that overfilling itself is not harmful, but the effect of pushing canal contents into the bone caused the adverse effects and failures
- Judging endodontic quality on a radiograph
  - 2D image cannot image the full circumference of the root canal filling
  - Defects/voids 50~100 um cannot be seen on a radiograph
  - Poor seal (visible voids in apical filling) was not associated with poorer prognosis in treatment of necrotic teeth with apical periodontitis
- Root end filling
  - Indications
    - Performed when orthograde access to the pulp is not available
    - Correction of perforations
    - Last tx option when orthograde treatment has failed
  - Filling materials
    - **Amalgam**: Known to have a poor seal. However, the fact that the infected apical portion is being removed and that amalgam has a mild antibacterial effect, it is associated with 60~80% success rate.
    - **IRM and SuperEBA**: good material with significant antimicrobial effects
    - **MTA (ProRoot)**: basically Portland cement. High pH = antimicrobial, can harden, and is non resorbable
    - No valid study saying one method is better than another

Persistent, recurrent, and acquired infection of the root canal system post-treatment

- This paper is basically a summary of all the papers listed before
- Dental caries
    - Usually the major pathway for bacteria to enter the pulp
    - Indicated that lactobacilli or Gram + pleiomorphic rods advance the front line of the caries lesion
        - Lactobacillus, Actinomyces, Propionibacterium, Bifidobacterium, Rothia, Eubacterium, Streptococci
        - Gram – anaerobes are also present, but to a lesser degree
    - Thought that the bacteria could be the etiology to pulpitis
        - However, first inflammatory responses in the pulp happen before bacteria enter the pulp
        - Also possible that metabolic end products of bacteria can initiate pulpitis
    - Bacterial infiltration will only affect the closes 2~3mm of pulp, and all underlying pulp can be normal
        - Gradually, bacteria invade deeper and diseased pulp grows
        - Not known how long this takes (few days ~ years)
    - Bacteria can enter the canal from caries, crown-root cracks, leaking fillings, lateral canals from a periodontal pocket, exposed dentinal tubules (post root planing), but **not** bacteremia
    - As long as there is vital pulp, there is only a limited number of bacteria in the root canal space
    - Therefore, the prognosis of RCT in teeth with pulpitis is excellent
- Bacterial flora
    - Several studies looked at microflora on teeth with primary apical periodontitis
    - Cases with closed pulps (no macroscopic communication with the oral cavity) had anaerobic organisms dominating at 70~100%
    - Cases with open pulps had much higher proportion of microaerophilic and facultative gram + bacteria
    - In dentinal tubules
        - Studies have shown 77~87% of necrotic teeth have bacteria in dentinal tubules
        - Streptococci and gram + rods (Actinomyces, Lactobacillus) appear to invade the dentinal tubules better than gram – species
        - Smear layer was seen to protect against tubular infection, but *E. faecalis* was able to penetrate through
        - Removal of root cementum (as seen in apical periodontitis) greatly facilitates invasion of bacteria into the tubules
        - NOT dependent on bacterial motility. Tubule infiltration is dependent on the bacteria's ability to attach to collagen
        - **Not associated with a poorer outcome of root canal treatment**
- Necessary components for eliminating an endodontic infection
    - Host defense system
        - Lack of circulation in a necrotic root canal means immune cells can only penetrate the root canal for the first few 100 micrometers
        - Can fight bacteria leaving the canal, but cannot eliminate the source
    - Systemic antibiotic therapy (not always needed)
        - Often given to minimize risk of post-operative symptoms, but evidence shows it does not reduce the incidence of flareups or acute problems after the start of treatment
        - Should only be given when there is a failure of host responses (spreading infection), and there is a risk of systemic involvement
    - Chemomechanical preparation and irrigation
        - Bleach is the most widely used, but concentrations over 0.5% can be caustic and irritate the patient
        - CHX has been proposed as an alternative, and found it similar in efficacy to bleach. However, CHX did not have the tissue dissolving ability
        - CHX + H_2O_2 has been proposed as a strong synergistic combination of irrigants against *E. faecalis*, *Strep sobrinus, and Staph aureus*
    - Local root canal disinfecting medicaments
    - Permanent root filling
    - Permanent coronal restoration
        - Coronal leakage is the main mechanism organisms can access the root canal after endo treatment

- Bacterial flora in previously treated teeth
    - Root filled teeth without apical periodontitis
        - Found *E faecalis*, streptococci, gram + facultative rods, *F nucleatum* (gram – anaerobic rod), *C albicans*
        - Number of bacteria were less than root filled teeth with apical periodontitis
        - May be residual flora from treatment, but more likely due to coronal microleakage
    - Root filled teeth with apical periodontitis
        - Predominantly *E faecalis*, being present in 53~64% of teeth
        - Facultative or microaerophilic species were also abundant: streptococci, *Actinomyces* spp, *Lactobacillus* spp, *Propionibacterium* spp
        - *Staphylococcus* spp were also found
        - Gram – rods were also present
        - Key point is that anaerobic bacteria were in the minority
- Protection against leakage
    - Bacterial penetration in filled canals is 50~100% in 2~12 weeks
    - Coronal coverage is essential in minimizing coronal leakage
    - 3mm of sticky wax on coronal part of root canal showed no bacterial leakage at the apex. However, this is not a suitable material
    - Cavit, IRM, and ZOE were shown to be good sealers
    - Glass ionomer cement alone or in combination with IRM provided an even better seal
    - IRM alone considerably delayed, but did not prevent bacterial penetration
    - **A stronger correlation was found between the presence of a periapical lesion and poor coronal restoration than poor quality of endodontic treatment**
    - **The combination of good restoration (GR) and good endodontic (GE) quality had the highest absence of periradicular inflammation (API) at 91.4%**
- Post operative healing
    - The healing of apical periodontitis can be observed for up to 4 years after the treatment (5 years after surgery), before taking a decision on further intervention
    - The prerequisite for this is that the tooth is symptom-free, and that the apical periodontitis lesion does not become enlarged in the control radiographs
    - Assess the possibility of a healing scar as well