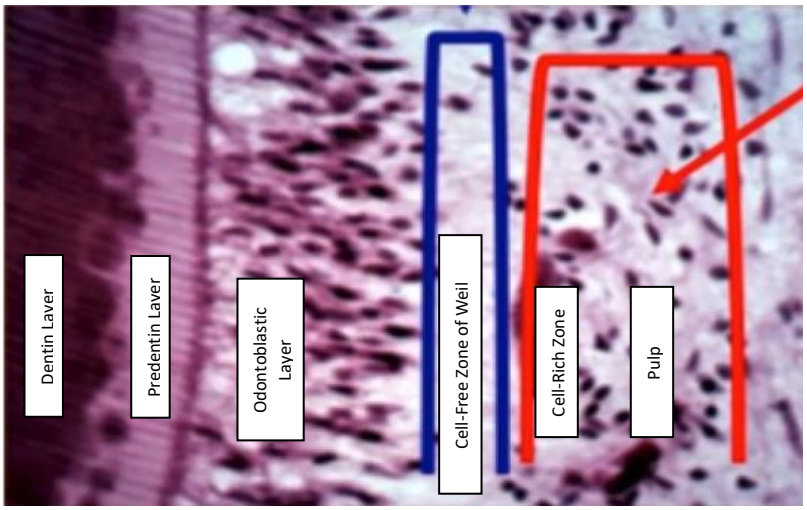
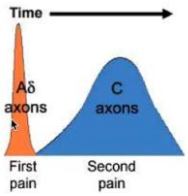
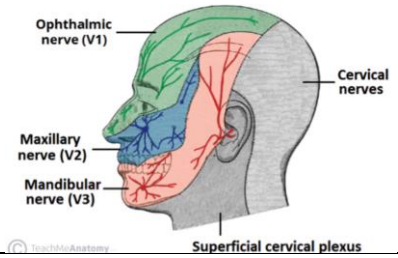
Pulp Biology and Tooth Pain

Pulp 	Contains loose fibrous CT w/ Nerves, Blood vessels, and Lymphatics **Cells**: - Fibroblasts (make the CT) - Odontoblasts (Make the dentin) - Undifferentiated mesenchymal cells (Makes tertiary dentin) Surrounded by hard dentin -> Limits its ability to expand (important when it comes to inflammation) Lacks collateral circulation -> Limits its ability to cope with infection
Dentin and Pulp Defence	**Different types of Dentin:** Sclerotic Dentin = Calcification of tubules in response to slowly advancing caries or aging Reactionary Dentin (2° dentin) = Reaction to *minor* damage - Stimulated by CaOH pulp capping if further away from pulp Reparative Dentin (3° dentin) = Repair for *major* damage - Stimulated by CaOH pulp capping if really close to the pulp Pulpal Necrosis = response to rapidly advancing caries or severe damage
Histology of the pulp	*Predentin* - Just inside the dentin, lighter in color because its not mineralized yet *Odontoblastic Layer* - Lay down the dentin on the outside of the pulp tissue just inside of the predentin *Cell-Free Zone of Weil* - Just inside the odontoblastic layer, no cells here BUT this is where the nerve bundles are found *Cell-Rich Zone* - Inside of the cell-free zone where the nuclei reappear *Pulp Core* 

Pain

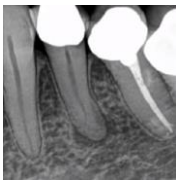


Dentinal Pain	***Aδ Fibers*** - Large myelinated afferent nerve - Courses coronally through the pulp, along the Pulpo-Dentinal complex/junction - Sharp transient pain "first pain" - Associated pain with Cold 
Pulpitis Pain	***C Fibers*** - Small unmyelinated afferent nerves - Course centrally through the pulp stroma - Dull, throbbing "second pain" - Associated pain with Heat 
Pain Sensitization	
Hyperalgesia	= Heighted response to pain - Presence of inflammatory mediators the ↑ sensitivity to pain
Allodynia	= Reduced pain threshold - Pain due to stimulus that does not normally provoke pain Memory trick: Sunburn -> Aloe-dynia - Usually touching your skin doesn't hurt…but when its burnt then it causes pain 
Referred Pain	Preauricular pain often refers from Mandibular Molars -> Because both share V3 innervation - You might think Max molars because they are closer…but the innervation is what matters 

Pulpal and Periapical Diagnoses

Every tooth (dead or alive) has 2 diagnoses: Pulpal and Periapical

Pulpal Diagnosis	
Normal Pulp	*Asymptomatic* - Mild to moderate transient response to thermal (Cold Test) and electrical stimuli (EPT) - Response is momentary and subsides when the stimulus is removed
Reversible Pulpitis	*Symptomatic* - Thermal (cold) stimulus -> Quick, Sharp, hypersensitive, transient (non lingering) response (Hyperalgesia) - No complaints of spontaneous pain - Caused by an irritant that affects the pulp *No RCT needed, just remove the irritant*
Symptomatic Irreversible Pulpitis	Symptomatic - Pulp has been irreversibly damaged beyond repair (will not heal even with removal of the irritant) - Spontaneous intermittent, or continuous pain - Thermal (Cold) stimulus causes lingering pain >10 seconds - Postural changes (bending over or lying down) ↑ BP to the head and may ↑ pain - Radiographs are generally insufficient - EPT is often not useful for Dx

Asymptomatic Irreversible Pulpitis	Asymptomatic - Microscopically similar to symptomatic irreversible - No clinical symptoms - Pulp is irreversibly damaged - Mostly a Dx of necessity -> Obvious clinical presentation that requires RCT (exposure of the pulp) but you still need a Dx in order to justify the Tx
Pulp Necrosis	Usually asymptomatic (not always though) - Can be partial or total (w/ or w/o symptoms) - Due to long term interruption of blood supply to the pulp - Crown discoloration may accompany pulp necrosis (Especially in anterior teeth) -> Tx with RCT and internal bleaching
Previously Initiated	= Tx was started (pulpectomy, pulpotomy) but not finished with full RCT
Previously Treated Pulp	= RCT was previous initiated or Tx
Periapical Diagnosis - Extension of pulpal disease into the apical tissues	
Normal Apical Tissues	Asymptomatic - No pain on percussion or palpation
Symptomatic Apical Periodontitis	Symptomatic - Painful inflammation around the apex - Pain on percussion with intense throbbing pain - Localized inflammatory infiltrate within PDL **If the tooth is vital, usually an occlusal adjustment is all that is needed. If tooth is necrotic, RCT is needed to prevent progression**
Asymptomatic Apical Periodontitis	Asymptomatic - Apical radiolucency found on radiograph - Confirmation of pulpal necrosis Periapical RL can be different based on histology - Radicular cyst - Periapical granuloma 
Acute Apical Abscess	Severe Pain - Rapid swelling - Purulent exudate (liquefaction necrosis) around the apex 
Chronic Apical Abscess	Usually asymptomatic - Draining sinus tract w/o discomfort - Insert GP cone into the tract and take a x-ray to find the source 
Condensing Osteitis	Response in bone due to long chronic low grade inflammation - RO surrounding the apex of affected teeth

Tests

Cold Test	Endo Ice = Dichlorodifluoromethane, -30° - Chilled pellet is applied immediately to the middle 3rd of the facial surface of the crown for 5 seconds (ensure the tooth is dried) - Intensity and duration of the response give info regarding the **pulpal diagnosis** 
Electric Pulp Test (EPT)	*Least reliable pulp vitality testing* - Indicates if there are vital ***sensory fibers*** in the pulp, but it does not provide any info on the ***vascular supply*** of the pulp - Lots of False positives and negatives - Tells you if the tooth is alive or dead, nothing in between **Contraindicated if the patient has a pacemaker 
Percussion	= Tapping on teeth with mirror handle - Vertical direction along the long axis of the tooth
Palpation	= Feeling gums around the apex of the tooth root - No swelling or pain on normal tissues

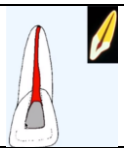
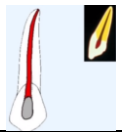
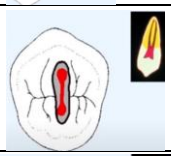
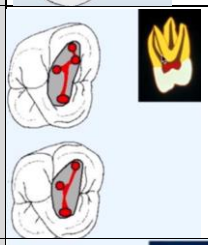
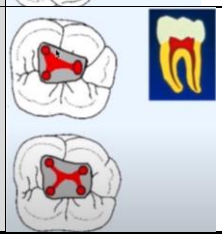
Root Canal Treatments

Access Preparations

Deroofing chamber to expose pulp horns and orifices

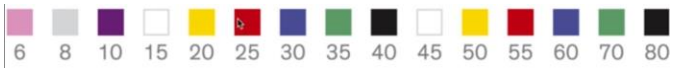
***Most important technical aspect of an RCT**
- Conservation of tooth structure is paramount
- Attain straight line access to orifice and apex

Also extremely important that a well sealed rubber dam is used to keep the canals clean of saliva contamination

Incisors		Triangle shape *U1 and U2* -> usually only have 1 canal, with triangular access *L2* -> sometimes has 2 canals, but can still use triangle or oval access
Canines		Ovoid shape U/L both usually only have 1 canal
Premolars		Narrow oval access *Upper 1st PM* usually has 2 roots and 2 canals
Maxillary Molars		Rhomboid/blunted triangle access Max. 1st molar Very frequently have 4 canals (MB1 and MB2, DB, P) - Important to get MB2. Missed canal is common for max molar RCT failure
Mandibular Molars		Trapezoidal Access Mostly have 3 canals, but can sometimes have 4

Instruments

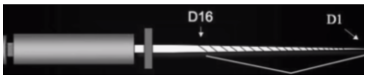
SS Hand Files (0.02 Taper)	NiTi Rotary (0.04 or 0.06 Taper)
K-File (Kerr)= Twisted square - Watch Winding method H-File (Hedstrom) = Spiral cone - Only cuts in retraction	More flexible files -> decrease ledging



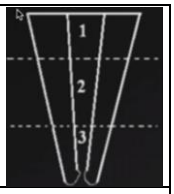
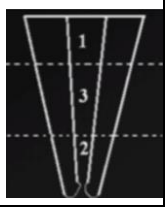
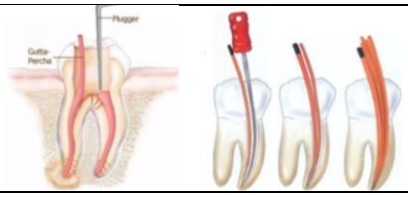
File Dimensions:

D1 = Diameter at the tip
- Size 15 = 0.15mm at tip

D2 (or D16) = Diameter 16mm from tip, where the cutting flutes end
- Size 15 K file = 0.15 + 0.02(16mm) = 0.47mm



Gates-Glidden drills	= Open orifice for straight line access
Barbed Broaches	= Entangle and remove pulp tissue or things that are stuck
Reamer	= Twisted Triangle

Cleaning and Shaping	*Aiming to clean and shape from 0-2mm from the apex (Avg 1mm)* **Crown Down**: Big – Small - Usually done with rotary 1. Start with orifice shaping (coronal 3rd) 2. Use successively smaller files to resistance to shape the middle and apical 3rds  **Step Back**: Small = Big - Usually done with hand instruments 1. Start with large file/gates glidden to open the orifice 2. Use small file to work up working length 3 sizes above initial binding 3. Use successively large files moving back 0.5mm at a time  **Irrigation and Medicaments** - ***Sodium Hypochlorite (NaOCl)*** = Irrigant, Dissolves organic material - ***Ethylenediamine Tetraacetic Acid (EDTA)*** = Lubricant, Chelating agent, Dissolves inorganic material (smear layer of dentin) - ***Chloroform*** = Dissolves GP in retreatment
Obturation	= Seal the canal system - Gutta-Percha + Sealer = Zinc-Oxide Eugenol - Techniques: Warm Vertical and Cold Lateral 

Endodontic Microbiology

1st Endodontic Infection	= Primarily Bacteroides - Gram –'ve obligate anaerobes
Failed Endodontic Infection	= Enterococcus Faecalis primarily - Gram +'ve facultative aerobic bacteria

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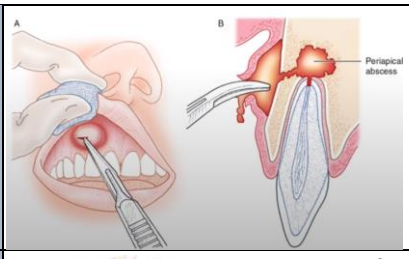
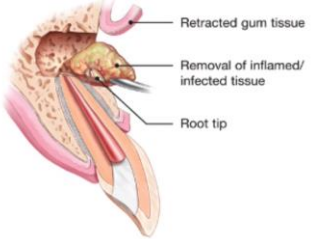
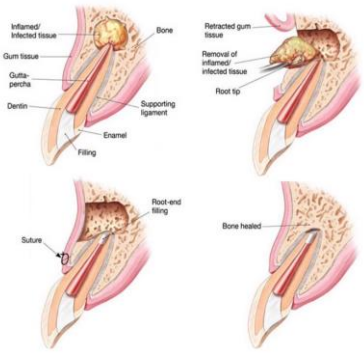
Surgical Treatment

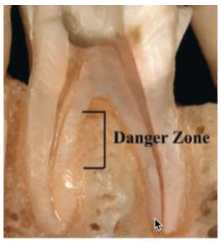
Endo Tx Planning:

1. RCT -> Orthograde (meaning its done from the normal direction…ie through the crown)
2. Retreatment -> Do if the RCT fails and the issue is in the canal
3. Surgical -> Persistent infection around the apex (not really an intra-canal problem)

Incision and Drainage	 = surgical opening in soft tissue to release pressure and exudate - Best for localized and fluctuant swelling - Done in soft tissue
Trephination	 = Surgical opening in hard tissue (bone) to release exudate and pressure

Periapical Microsurgery		Access is achieved through the bone at the apical aspect of the tooth - Coronal aspect is already well sealed…not we are trying to get a better seal apically 1. Raise ST flap and window the bone. Clean out all infected bone and granulation tissue 2. Remove 3mm of the apex of the infected tooth (Apicoectomy) with a 0-10^{o} bevel 3. Use an ultrasonic tip to instrument the apical portion of the Root Canal to 3mm deep 4. Retrofill with MTA 5. Suture the soft tissue and allow the bone to heal

Procedural Complications

Ledge Formation	= Artificial irregularity created on a surface of root canal wall - Creating a different shape of the canal as a result of the file attempting to create a straight canal **How**? - Inadequate straight line access (improper glide path) - Longer canals, smaller diameter canals, and curved canals more prone - Inadequate irrigation and lubrication - Transportation -> Tendency for a file to straighten the canal **Prevention** - NiTi flexible files are less likely to ledge - Bypass the ledge by using small instruments - Place a small bend in the file to bypass the ledge (need to know where the bend is and how to orient it in relation to the ledge) 
Instrument Separation	= Breakage of an instrument within the confines of a canal **How?** - Excessive force - Moving up file sizes too fast (Moving from #20 -> #30) - Inadequate lubrication and irrigation - File wear (too many rounds of sterilization and use) **Prevention** - Frequently replace files - SS files are less likely to fracture Vs. NiTi **Tx**: - Use smaller instruments to bypass the separated file - Usually leave it in place and chart **Later in the procedure that the instrument separates, the better the prognosis. Because more bacteria are removed* 
Perforation	**Coronal Perf** = Through the crown during the access - Occurs if you get lost through access prep. Always know the anatomy of the tooth and check your angulation **Furcal Perf** = Through the pulpal floor - Typical with molars and 1st max premolar **Strip Perf** = Due to excessive coronal flaring of the orifice - Particularly a risk for mandibular molars - Concavity exists on the Distal side of Mesial roots on mandibular molars. Danger zone! Always favor the mesial side of mesial roots on mandibular molars **Root Perforation** = Perforation of the root - Happens if you keep ledging all the way through the root - More apical the better the prognosis - Typical signs: Immediate hemorrhage +/- sudden pain - Tx: Internal repair with MTA  

Traumatic Injuries

Trauma Protocol:

T – Tetanus Booster w/i first 48hrs (Avulsions only when you want to replant)

R – Radiographs (At least 1 PA, possible need for Pan if fracture is suspected)

A – Antibiotics (Avulsions only when you want to replant)

V- Vitality Testing (Thermal testing, EPT etc) -> False negative results are common after trauma

M - More

A – Appointments (3wks, 3mth, 6mth, 1yr following the injury)

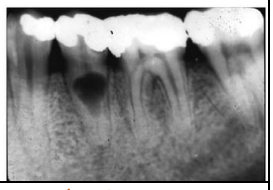
Ellis Classification	
Class I (1 layer)	Enamel Only
Class II (2 layers)	Enamel + Dentin
Class III (3 Layers)	Enamel + Dentin + Pulp
Class IV (Kills tooth)	Traumatized tooth that have become non-vital
Class V (Moves tooth)	Luxation
Class VI (Kicks tooth out of the socket)	Avulsion

Uncomplicated Fracture		= No pulp involvement *Tx:* - Enamel only -> Smooth edges - Enamel + Dentin -> Restore normally
Complicated Fracture		= Involves Pulp - Timing is important! *Tx:* - <24 Hrs: **Direct** Pulp Cap (for Permanent only) - ≥ 24 Hrs: **Cvek Partial** Pulpotomy - ≥ 72 Hrs: **Pulpotomy**
Horizontal Root Fracture		= Coronal segment is displaced, Apical segment is not displaced Investigations: - 3PA and 1 Occlusal Radiograph -> ↑ Radiographic angles the better Ideal Healing = Calcific Healing -> Reapproximate the pieces and a calcified callus holds the fragments together along the fracture line *Tx*: - *Vital* -> Splint ASAP - Coronal fracture -> Rigid splint 6-12 weeks - Mid-root Fracture -> Flexible splint for 3 weeks - Apical Fracture -> Flexible splint for 2 weeks max (to avoid ankylosis) - *Non-vital* -> Root Canal Therapy - 25% chance of necrosis of coronal segment, very rare to have necrosis of the apical segment
Concussion		= Booped the tooth. No displacement, no mobility, PDL is intact but sensitive Tx: - Let the tooth rest. Don't bite on the tooth for a few days

Subluxation	 = A harder boop. No displacement of the tooth, but ↑ mobility. PDL rips and you get sulcular bleeding. *Tx*: Flexible splint for 1-2 weeks - 6% chance of necrosis with closed apices, prognosis ↑ with open apex
Extrusion	 = A very hard boop. Tooth is partially extruded from socket *Tx*: - *Open Apex* -> Reposition, Flexible splint for 1-2 weeks, Monitor - *Closed Apex* -> Reposition, Flexible splint, RCT if tooth loses vitality - 65% chance of necrosis with closed apex
Lateral Luxation	 = Displacement of tooth in any direction (Except axially) - Usually crown is displaced palatally and the root apex is displaced labially *Tx*: Same as extrusion - *Open Apex* -> Reposition, Flexible splint for 1-2 weeks, Monitor - *Closed Apex* -> Reposition, Flexible splint, RCT if tooth loses vitality - 80% chance of necrosis with closed apex
Intrusion	 = Apical Displacement of tooth *Tx*: - *Open Apex* -> Allow to re-erupt -> This is a hot boards Q - *Closed Apex* -> Reposition, Flexible Splint, RCT - 96% chance of necrosis w/ closed apex
Avulsion	 = Complete separation of tooth from the alveolus **Extra-alveolar Dry Time (EADT) = the amount of time the tooth has been out of the mouth while dry** -> This is critical *Tx*: Reimplant ASAP, Flexible splint for 1-2 weeks - ***Closed Apex, EADT <60 minutes*** -> Reimplant, splint - ***Open Apex, EADT < 60 minutes*** -> Reimplant, Splint, Apexification at 1st sign of infected pulp (no RCT) - ***Closed Apex, EADT >60 minutes*** -> Reimplant, Splint, RCT - ***Open Apex, EADT > 60 minutes*** -> May or may not reimplant, splint, RCT, Plan for future implant **Storage Medium (from best to worst):** - Hanks Balanced Salt Solution (HBSS) - Milk - Saline - Saliva (aspiration and swallow risk) - Water -> least desirable 

Long Term Responses to Trauma

External Resorption	= Initiates in the periodontium due to damage to the cementoblastic layer in the PDL  **Replacement Resorption (RR)** -> Ankylosis, Replaces PDL with bone **Cervical Resorption (CR)** -> Subepithelial sulcular infection from trauma, or non-vital bleaching - Initiates at the CEJ, Presents as ragged moth eaten appearance - Clinically you can see a pink spot on the tooth **Inflammatory Root Resorption (IRR)** -> Bacteria and by-products from necrotic pulp travel through the dentinal tubules to affect the periodontium *Margins*: - Poorly defined, ragged, and move w/ different angled radiographs

Internal Resorption	= Initiates in the root canal system because damage to the odontoblastic layer - Inflammation due to necrotic pulp from caries, trauma etc but we get resorption from within Internal Resorption -> Tx w/ RCT *Margins*: - Sharp, well defined, does not move w/ angled radiographs 
Calcific Metamorphosis	= Trauma induced odontoblasts to rapidly form extensive amounts of reparative 3° dentin w/l pulp space - More likely w/ open apices, Intrusions, Severe crown fractures Appearance: - Yellow-orange tooth - Canal obliteration 

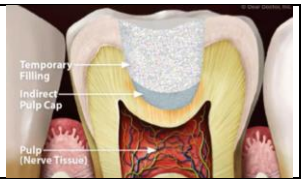
Adjunctive Endodontic Treatment

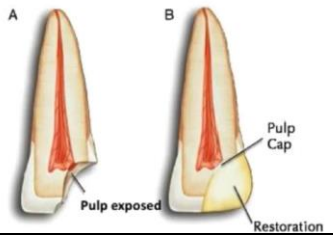
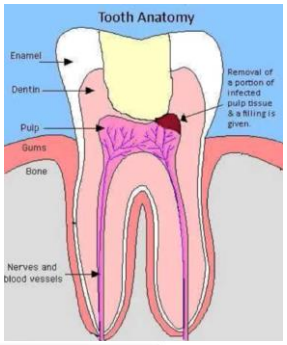
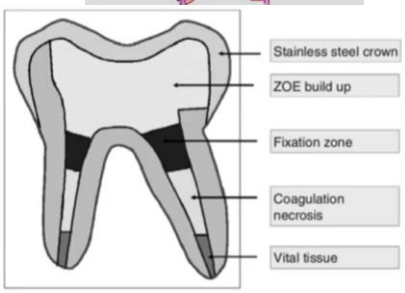
Important Materials in Vital Pulp Therapy

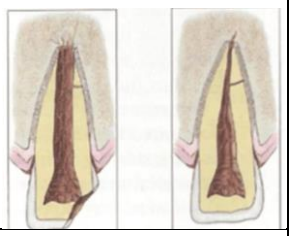
Calcium Hydroxide ($CaOH_2$)	Stimulates secondary odontoblasts to repair w/ dentinal bridge formation - Stimulates undifferentiated mesenchymal cells to become secondary odontoblasts, which then lay down tertiary dentin - High pH of 12.5 cauterizes tissue and kills bacteria 
MTA (Mineral Trioxide Aggregate)	Stimulates Cementoblasts to produce hard tissue **Contents**: 3 minerals - Calcium - Silicon - Aluminum **Opacifier**: Bismuth Oxide -> Can leak and stain the teeth (sketchy for **Setting Time**: Long, 3hrs ***Pros***: - Sets in the presence of moisture (isolation is a non-issue) - Antimicrobial - Nonresorbable + biocompatible. Great long lasting seal! ***The 3 3's:*** - 3 minerals - 3hrs to set - 3 Major pro's 

Vital Pulp Therapy

Idea is that the pulp is troubled, but still vital and we want to maintain that vitality

Indirect Pulp Capping (Vital)	= CaOH, or RMGIC is placed on a thin partition of remaining dentin which if removed might expose **Healthy** pulp *Indication*: Deep caries approximating the pulp 

Direct Pulp Capping (Vital)	= CaOH is placed directly on an exposure of a **healthy pulp** - Hard tissue barrier will hopefully form w/i 6 weeks - Less favorable prognosis vs Indirect Pulp Cap *Indication*: - Traumatic Exposure <24 hrs - Carious or mechanical exposure <2mm (pinpoint) 
Cvek Pulpotomy (Vital)	= Partial/shallow pulpotomy - Removal of small portion of coronal **diseased pulp** *Indications*: - Traumatic exposure ≥ 24 hrs - Carious or mechanical exposure >2mm 
Pulpotomy (Vital)	= Removal of coronal **Diseased Pulp** - ZOE buildup and formocresol to attain hemostasis - Formocresol placed on the canal orifices to create a Fixation zone. Renders it resistant to enzymatic breakdown - *Coagulation Necrosis:* Pulp tissue will die, but there is still some vital tissue in the apex *Indications*: - Traumatic exposure >72 hrs - Primary tooth that is restorable (SSC) with a pulp exposure but has no symptoms ***Buckley's Formocresol*** = Bactericidal + Fixative Agent - 19% Formaldehyde - 35% Cresol - 15% Glycerine - 31% Water **Its really toxic now though and not really indicated** 
Pulpectomy (Non-vital pulp therapy)	= Removal of coronal AND radicular **dead or dying** pulp tissue - ZOE buildup, CaOH in the root (resorbable by the erupting permanent tooth) *Indications*: - Often as a temp pain relief for irreversible pulpitis until full RCT can be done - Primary nonvital yet still restorable tooth w/ pulp exposure (Asymptomatic) 
Extraction (Non-vital pulp therapy)	= Removal of tooth w/ dead or dying pulp *Indications*: - Primary 1st molars (these teeth are too sketchy to do pulpectomy on) - Non-restorable teeth - Symptomatic root resorptions 
Root Canal Tx	= Pulp can be diseased or dead - Pulpectomy + Cleaning + Shaping + Filing 

Apexogenesis (Vital Tooth)	= Maintain pulp vitality in order to stimulate root development and allow the body to make a stronger (closed apex) root - CaOH or MTA placed on **<u>healthy or diseased</u>** pulp - Includes any IPC, DPC, Cvek, or PPTY performed in an immature permanent tooth -> Basically all of the Vital pulp therapies above…if they are done on an immature permanent tooth are considered Apexogenesis ***Contraindication***: - Avlused teeth - Non-restorable teeth - Revere Horizontal Fracture - Necrotic teeth 
Apexification (Non-vital tooth)	= Disinfection of root canal followed by induction of an acceptable apical barrier - CaOH or MTA is placed at the base of a canal after a dead or dying pulp is removed <u>*Includes*</u>: Pulpectomy performed in an immature permanent tooth