**Index Card Literature Guide**

**2014 Update**

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**History of Endodontics**

**AAE**/**ABE**:

1943 AAE begins (Clyde Davis-1st president)

1963 Endodontic specialty recognized by ADA

1956 ABE begins

1965 1st ABE board exam

**1st in Endodontics**:

Hermann (1930): Introduced Ca(OH)2 - vital pulp cap; Frank: Apexification

Coolidge (1919): Introduced NaOCl as tissue solvent (Dakin’s Solution)

Nygaard Ostby (1957): Introduced EDTA for dentin softening

Imperial Chemical Industries (1940s).: Introduced CHX

Barnum (1864): Introduced concept of Rubber dam

Bowman: Introduced GP for obturation, Rubber dam forceps

Koller: Proposed Cocaine as anesthetic

Einhorn (1906): Procaine (novocaine) introduced

Maynard: Developed broach

Arthur: Introduced barbed broach

**1st in Endodontics**:

Harry B. Johnston: Created term “Endodontia”; 1st Endodontic Office

Clyde Davis: 1st president of AAE

Otto Walkhoff: 1st dental radiograph, CMCP as pulpal antiseptic

Ingle (1961): Standardization of GP and instruments

Miller; Hunter: Introduced Focal Infection Theory, Billings: Introduced Focal Infection Theory to USA

Pfaff: Introduced pulp capping

Codman: Concept of secondary dentin in pulp cap healing

Buckley: Developed Formocresol for pulpal antiseptic (“Buckley’s solution”)

Jasper: Silver points

Hudson: 1st to obturate canal (gold)

Hill: Introduced Hill’s stopping (GP, carbonated lime, quartz) for obturation

Perry: 1st carrier based obturation (Goldwire/GP for obturation), see also Wm Ben Johnson: Thermafil

Callahan: Introduced chloropercha technique for obturation

# Diagnosis

# AAE Terminology

**Pulpal**

**Normal pulp** – A clinical diagnostic category in which the pulp is symptom free and normally responsive to vitality testing.

**Reversible pulpitis** – A clinical diagnosis based upon *subjective and objective findings* indicating that the inflammation should resolve and the pulp return to normal.

**Irreversible pulpitis** – A clinical diagnosis based on *subjective and objective findings* indicating that the vital inflamed pulp is incapable of healing.

*Additional descriptions:*

**Symptomatic** – Lingering thermal pain, spontaneous pain, referred pain **Asymptomatic** – No clinical symptoms but inflammation produced by *caries, caries excavation, trauma*, etc.

**Pulp necrosis** – A clinical diagnostic category indicating death of the dental pulp. The pulp is non-responsive to vitality (sensibility) testing.

**Previously Treated** – A clinical diagnostic category indicating that the tooth has been endodontically treated and the canals are obturated with various filling materials, other than intracanal medicaments.

**Previously Initiated Therapy** – A clinical diagnostic category indicating that the tooth has been previously treated by **partial endodontic therapy** (e.g. pulpotomy, pulpectomy).

**NOTE: See AAE Glossary of Endodontic Terms for further definitions**

**Periapical**

**Normal apical tissues** – Teeth with normal periradicular tissues that will not be abnormally sensitive to percussion or palpation testing. The lamina dura surrounding the root is intact and the periodontal ligament space is uniform.

**Symptomatic apical periodontitis** – Inflammation usually of the apical periodontium, producing clinical symptoms including *painful response to biting and/or percussion or palpation.* It may or may not be associated with an apical radiolucent area.

**Asymptomatic apical periodontitis** – Inflammation and destruction of apical periodontium that is of pulpal origin, appears as an apical radiolucent area and *does not produce clinical symptoms*.

**Acute apical abscess** – An inflammatory reaction to pulpal infection and necrosis characterized by *rapid onset, spontaneous pain, tenderness of the tooth to pressure, pus formation and swelling of associated tissues*.

**Chronic apical abscess** – An inflammatory reaction to pulpal infection and necrosis characterized by *gradual onset, little or no discomfort and the intermittent discharge of pus through an associated sinus tract*.

# Radiological Exam

1. Vital case w/ AP – *Stashenko; Yamasaki; Byers* (CGRP sprouting)
   1. *Torbijork* – C fibers resistant to hypoxia/necrosis
2. Bone loss & AP – Avg 7.1% MBL, 12.5% CBL – *Bender 1982*
3. Digital Comparison studies
   1. Film vs digital – root length – NSD – *Pitt Ford*
   2. Film vs digital – PARL det. – NSD – *Mistak/Loushine*
   3. Digital (filtered) > D speed – Cortical lesion – ***Hadley/Replogle***
   4. Film vs digital – WL measurement – NSD – *Lamus/Katz*
   5. Digital (Kodak) > Film – WL (10,15 k) – ***Goodell/McClanahan***
   6. 80 – 90% radiation reduction w/ digital – ***Soh***
4. Lamina dura – Most consistent feature aiding dx of PAP – *Kaffe*
   1. *Strindberg* – PDL width/shape, Lamina dura aids dx of PAP
5. Radiographic Interpretation accuracy –
   1. *Brynolf* - accuracy increases with added films, 1=74%, 3=87%
   2. *Goldman* – 47% interobserver, 75% intraobserver (6-8 mo later)
   3. *Tewary/Hartwell* – digital PAs – 35% interobserver agreement
6. Technique - Paralleling > Bisecting Angle – WL – *Forsberg*

## CBCT

-Coneshaped beam captures variable size cylindrical/spherical volume data (FOV)

-*Isotropic cubic Voxels* (3-D pixels) comprise the image

-3 Orthogonal planes: Axial, Coronal, and Sagittal; Multiplanar reconstruction

-*Spatial Resolution is less (2 line pairs/mm) than digital PA (7-25 line pairs/mm)*

**Diagnosis:** *Hassan – Axial view best for VRF detection*

1. ***Patel 2007*** – Applications for CBCT & Endo:

* Detection of PA changes & Diagnosis (PRE-OP)
* Anatomical anomalies (i.e.: Dens), Resorptive lesions
* Treatment complications – Perforations, VRFs, sep instruments
* Pre-surgical planning
* Healing/Treatment Outcomes (POST-OP)

1. *Simon 2006* – Large PARL, Granuloma vs. Cyst; CBCT (grey scale) >Biopsy
2. ***Bernardes/Azevedo 2009; Ozer 2010*** – VRF detection: CBCT > Digital PA
3. *Ozuk 2011* – Accumoto 93% accuracy VRF (confirmed via Sx/RETX)
4. ***Ee/Johnson 2014*** – CBCT>Digital PA *Diagnosis: VRFs, Resorption, Perforations, PARLs*- 83% vs 36%. CBCT changed recomm. tx 60% of time
5. *Blattner 2010* – MB2 detection: CBCT = Sectioning (gold standard)

**CBCT**

**Radiation:**

1. ***Ludlow 2006/AAE 2011*** – Kodak: Effective dose (μSv): Max Ant: 4.7, Max Post: 9.8, Mand Post: 38.3; 1 dig. PA: 6, FMX: 171, Background (day): 7-8

## CBCT vs. PA Radiograph (PARL detection):

1. ***Velvart 2001*** – Mand. Molars, CBCT 100%, *E speed PA* 78%
2. *Lofthag-Hansen 2007* – CBCT 100%, F speed PA 62%
3. ***Bornstein/Von Arx 2011*** – Mand. Molars, CBCT 100%, *F speed PA* 75%
4. ***Tsai/Torabinejad 2012*** – Simulated lesions (0-1.4 mm), **CBCT > *Digital PA***

**CBCT vs. PA Radiograph (Mandibular canal detection):**

1. ***Velvart 2001*** – Mand. Molars, CBCT 100%, *E Speed PA* 61%
2. ***Bornstein/Von Arx 2011*** – Mand. Molars, CBCT 100%, *F speed PA* 35.3%

**Working length:**

1. *Jeger/Bornstein* *2012* – NSD using CBCT and Digital PAs for FWL

**Subjective & Objective Examination**

1. ***Reeves/Stanley*** – Irrev. pulpitis - caries w/in 0.5mm of pulp or reparative dentin
2. *Tronstad* – direct pulp cap is only 50% successful (= Irrev. Pulpitis)
3. ***Barthel*** – direct pulp cap (CaOH2) – 5 yrs: 40% fail, 10 yrs: 80% fail
4. *Cvek* – Cvek (partial) pulpotomy (trauma/2 mm/7 days) = 96% success
5. *Kretzschmar* – referred pain may come from Max. sinus (Rhinosinusitis)
6. Cold test
   1. *Rickoff/Trowbridge* – no pulp injury Cold CO2 5 min, Heat 10s
   2. ***Miller*** – RS(TFE) > CO2 Pulp temp red; PFM/All Ceram/Gold Crns
   3. *Jones/Rivera/Walton* – RS Faster response than CO2 snow
   4. ***Petersson*** – Overall accuracy: Cold 86%, EPT 81%, Heat 71%
      1. Sensitivity (test identifies disease): 83%, 71%, 86%
      2. Specificity (test identifies healthy): 93%, 93%, 41%
      3. Pos. Predictive Value (given test states diseased, probability subject is diseased): 89%, 88%, 48%
      4. Neg. Predictive Value (given test states healthy, probability subject is healthy): 90%, 84%, 83%
   5. *Villa-Chavez 2013* – Cold>Heat>EPT – Accuracy, Reproducibility
   6. *Trowbridge* – mode of action Cold test – hydrodynamic theory
7. Cold + EPT
   1. ***Peters/Baumgartner*** - Cold neg + EPT neg = True Necrotic
   2. ***Weisleder/Trope*** - RS + CO2 + EPT: All 3 tests positive (NPP): 97% vital, All 3 tests negative (PPP): 90% necrotic; Cold + EPT ↑ accuracy
8. Barodontalgia –pain caused by change in pressure (flying/diving)
   1. *Senia/Cunningham* – inflammed vital pulp tissue
   2. *Fenjensick* – 86% faulty restorations
9. Histopathologic correlation with testing & clinical symptoms
   1. ***Seltzer/Bender; Dummer*** – no correlation btw diagnostic tests (except PN) & histo pulp status and clinical signs/symptoms & histo pulp status
10. Localization of Pain:
    1. *Friend/Glenwright* –37% patients could localize symptoms to offending tooth, to 3 teeth 80% (EPT stimulation – Vital teeth)
    2. ***McCarthy/McClannahan*** –73% patients could localize symptoms to the offending tooth (*Irreversible Pulpitis*); 90% Perc + patients, 30% Perc - patients; 100% No midline cross; ↑pain (VAS) = ↓ localization
11. Mechanical Allodynia (percussion sensitivity):
    1. ***Khan/Hargreaves*** – 67% Irrev. Pulpitis, 57% Pulp Necrosis
    2. *Owatz/Hargreaves* – 57% Irreversible Pulpitis

### Vitality testing = Sensibility testing = pulpal nerve status only

### *\*\*\* Does not evaluate blood/vascular supply\*\*\**

Reliability:

1. ***Fulling/Andreasen; Fuss/Trowbridge*** – immature developing teeth, unreliable response to EPT, use CO2 snow/RS (Higher EPT thresholds/late innervation of plexus of rashkow)
2. ***Bhaskar* –** trauma cases - EPT, cold and heat tests – unreliable (due to nerve damage w/out altering blood supply)
3. *Fuss*/*Trowbridge* –EPT unreliable w/ large restoration

EPT:

1. *Narhi*- Mode of action – low threshold Aδ fibers (prepain sensation) – ionic fluid shift
2. *Abdel Wahab*- Technique - slowly increased current- more accurate
3. *Mumford* – No relationship between EPT value and pulp histopathology
4. *Bender* – test Incisal Edge in Anterior teeth
5. *Jacobson* – test Middle 1/3rd F -Incisors, Occlusal 1/3rd B -Premolars

Heat test:

1. ***Schindler*** – used on refractory (persistent AP) cases to identify *missed canals* or *late stage of irreversible pulpitis*

**Laser Doppler**

LDF = Use of infared beam of light – scatters by Doppler principle when interacting with moving RBCs – photodetector reads this Doppler shifted backscattered light = Index of pulpal blood flow

1. ***Yanpiset/Trope* *2001***– dog study, avulsion/reimplantation - detect return of pulpal blood flow by **4 wks**
2. ***Gazelius 1988*** – case report, Lateral luxation 4 lower incisors – detected blood flow **6 wks (partially), 9 mos (complete)**
3. *Chandler/Sundquist* *1999* – case report: LDF – Dx: Periapical COD
4. *Mesaros/Trope* *1997* – case report: 8 yr old, luxation #8,9 – LDF- vital

# *Tronstad 1994* - LDF 91% accurate, more accurate than EPT (64%)

**Pulse Oximeter**

Pulse Ox = 2 light emiting diodes at 2 wavelengths (red, infared) transmit light through vascular tissue, absorbed selectively by oxygenated and deoxygenated hemoglobin, *photodetector reads unabsorbed light* = Oxyhemoglobin (HgO2) Saturation of Arterial blood

1. ***Gopikrishna* *2007*** – Compared **Pulse Ox to EPT and Cold** tests for recently traumatized (*uncomplicated crown fractures, concussions, subluxations only*) maxillary incisors (day 0 to 6 months post trauma); Pulse Ox signficantly improved ability to detect pulp vitality (**intact vascular supply**) **day 0 – 1 month**
2. ***Setzer 2012*** – Evaluated Pulse Ox for determining pulpal conditions: Normal pulp, Reversible pulpitis, Irreversible pulpitis, and Pulp necrosis. Statistically significant differences in **mean pulp oxygenation levels** for each pulpal condition – possible method to determine pathological process within pulp

# ASA Classification

1. A normal healthy patient
2. A patient with *mild to moderate systemic disease*
3. A patient with *severe systemic disease* that limits activity but is not incapacitating
4. A patient with *severe systemic disease* and is a constant threat to life
5. A moribund patient not expected to survive 24 hours with or without an operation.

# Glickman – Classification of furcation involvement

Class I – Incipient lesion

Class II – Bone destroyed on one or more aspects of furca, *partial penetration of probe* into furcation

Class III – Interradicular bone absent but orifice of furca is *occluded by gingival* *tissue,* *complete penetration of probe* through furcation

Class IV – Furca opening visible

# Mobility – Miller Index Classification

Class I - barely perceptible

Class II - < 1 mm movement

Class III - > 1 mm movement/depressible in socket

# Does periodontal disease cause pulpal disease?

**YES**

1. *Seltzer* – Yes, bacteria can pass through lateral/accessory canals
2. *Rubach/Mitchell* – Yes, bacteria can pass through lateral/accessory canals
3. *Wong* – Pulpitis adjacent to areas of scaling/root planing (Perio tx)

**NO**

1. *Langeland* – Only when Apical Foramen involved
2. *Bergenholtz* - No
3. *Mazur/Massler* – found no relationship between pulpal & periodontal disease

*Gutmann* – 28% Molars – Furcation Canals (see also *de Deus* - 27% accessory)

AAE definitions:

Accessory Canal – Any branch of the main pulp canal or chamber that communicates with the external surface of the root

Lateral Canal – Accessory canal located in the coronal or middle third of the root

# Simon’s Classifications for Endo-Perio Lesions

# Primary Endo: Necrotic pulp, CAA drains into sulcus/furca = Narrow isolated PD; mimics VRF, Perio Abscess

# Primary Perio: Vital pulp, Wide PD defect, Angular/may involve several teeth; Prognosis depends on Perio Tx

1. **Primary Endo + Secondary Perio**: Necrotic pulp, CAA drains into sulcus/furca + Plaque/calculus at gingival margin = solitary, wider PD
2. **Primary Perio + Secondary Endo**: (Controversial) Wide PD defect extending to AF → IP/PN; Prognosis depends on Perio Tx
3. **True Combined**: PN + Perio = Endo lesion (apically) meets perio lesion (cervically); Extensive bony destruction, Wide defects, May involve multiple teeth; Prognosis depends on Perio Tx
4. **Concomittant Endo Perio Lesion (added later)**: Separate and Distinct Endo and Perio lesions w/ no influence on either; Prognosis depends on Perio Tx

**Biologic Width**

*Gargiulo* – 1. sulcus depth ~ 1mm

* + 1. epithelial attachment ~ 1mm
    2. connective tissue attachment ~ 1mm

**Calcific Metamorphosis (aka PCO)**

1. *Trowbridge/Kim* – caused by luxation injury, obliteration of pulp by mineralized tissue. Occurs in **immature teeth**, pulpal infarct, connective tissue from PDL proliferates and replaces pulp.
2. ***Gutmann*** –1-16% CM cases develop pulpal necrosis, do not treat cases of calcific metamorphosis unless AP or nonvital
3. ***Andreasen*** – **22% trauma cases → Calcific metamorphosis**
4. *Jacobsen*– 70-90% horiztonal root fractures develop Calcific Metamorphosis
5. ***Walton*** – No visible canal radiographically but ALWAYS present histologically

# Calcific Metamorphosis (aka PCO)

1. ***Trope*** – caused by luxation injury, uncontrolled reparative dentin or hemorrhage and clot formation act as a nidus for calcification, occurs in **immature** teeth.
2. ***Robertson/Andreasen*** – **8.5%** PCO cases → Necrosis; 50% PCO respond to vitality test (final recall: 16 years), 20 yr survival: 84%
3. *Holcomb/Gregory* – 7% PCO cases → Necrosis
4. ***Gutmann*** – **1-16%** CM cases develop necrosis, do not treat CM unless AP or nonvital

# Dystrophic Calcification

Diffuse foci of calcification frequently found in the aging pulp; usually described as being perivascular or perineural.

**Age & Pulp Stones / pulpal calcification**

1. ***Bernick*** – Age causes:
   1. Reduction in *vascular supply, innervation* (loss of plexus of rashkow), *cellularity* (odontoblasts deterioration), *and pulp chamber size* (due to deposition of dentin)
   2. Increase in *calcified masses and collagen* (relatively) within the pulp
2. *Hendricks-Klyvert*– incidence of calcifications 8-90%
   1. Pulp stones – calcifications
   2. Denticles – composed of dentin

**Types: Free, attached, embedded**

Characteristics: Normal or Inflamed, Old or Young, Occurs in 50% pulps, Asymptomatic, No detrimental effect on pulp

# What lines sinus tracts?

***Baumgartner***–

1. 100% of sinus tracts are lined with epithelium to the level of the rete ridges (epithelium/CT barrier)
2. ***67%*** *had granulomatous tissue lining the tract past the rete ridges*
3. 33% had epithelium lining the entire way

*Harrison/Larson* –

1. 10% lined with epithelium
2. ***90%*** *lined with granuation tissue*

***Gupta*** –

1. Overall Incidence of Sinus Tracts (Teeth with PARLs): **18%**

**Discuss the Cracked Tooth**

1. *Cameron* – coined term **Cracked tooth syndrome**, most commonly found in the mandibular second molar (#1- biting pain, #2-acute onset thermal sensitivity)
2. *Hiatt* – **Mand 2nd Molar**>Mand 1st Molar>Max PMs = Max 1st Molar; 70%: non-restored or minimally restored
3. *Abou-Rass* – Transillumination and Staining for diagnosis of Cracked Tooth
4. *Rivera* – Classified longitudinal tooth fractures
   1. Craze lines
   2. Cuspal fracture
   3. Cracked tooth
   4. Split tooth
   5. Vertical root fracture
5. *Ehrmann* – Ortho band – eval 2-4 wks → symptomatic →RCT/Crown

**Discuss the Cracked Tooth**

1. *Krell/Rivera 2006* – **20%** Reversible Pulptitis/Cracked teeth treated with full coverage crown required NSRCT by 6 mos due to IP or PN. 9.7% incidence.
2. *Berman/Kuttler* – Fracture necrosis - 27 teeth w/ PN and M-D crack (no or shallow rest.), ext, micro CT, cracks deep onto root surface, Rec. EXT
3. *Tan 2006* – 1st longitudinal crack retrospective outcome study (49 patients) – RCT/Crown – 85.5% survival at 2 years. Sign. Outcome factors: terminal tooth, multiple cracks, pre-op probing depth; Location/extent not sign. factor.
4. *Seo/Park JOE 2012* – Characteristics of Cracked teeth: 67% No restoration/Class I; Bite test #1 diagnostic test; Staining/transillumination

# *Abbott/Leow* *2009* – Cracked Tooth Syndrome is not a “syndrome”

1. *Opdam* – Cuspal coverage composite; *Signore* – Cuspal coverage amalgam

# Vertical Root Fracture

***\*\*Tamse/Fuss 1999****:*

1. *“J-shaped”* lesions on PA
2. Etiology- *Intraradicular Posts, Lateral condensation*
3. #1- Max 2nd PM, #2- Mesial root Mand Molar
4. *67% had isolated buccal perio defect; 34% sinus tract closer to g.m. than apex*

***Holcomb/Pitts;*** *Meister* – VRF from lateral condensation – 84% (wedging forces)

***Peters*** – VRF from occlusal loading of Posts

*Rud/Omnell* – *79.8%*VRFs had isolated narrow perio pocket

***Pitts/Natkin*** – Exploratory Sx: “Punched out” bony les. (dehisc/fenest) – granulomatous tissue; perio abscess, multiple sinus tracts (pathognomonic)

*Ross* – Carbon fiber, Parallel sided posts least likely to cause VRFs

***Cohen/Berman 2006*** – VRFs: Max PMs, Mand 1st Molars; RCT; >40 yo

**Periapical Index**

***Orstavik* *1986*** – 5 categories, modeled on diagrams/histological diagnoses by *Brynolf*, Radiographic size of periapical lesion, 1=healthy, 2, 3 = uncertain, 4, 5 = diseased; 1=normal, 2=small changes in bone, 3=changes in bone w/mineral loss, 4=AP w/well defined RL area, 5=Severe AP w/exacerbating features

***Estrella* 2008** – PAI for CBCT:

0 = Intact periapical bone structures

1 = Diameter of PARL > 0.5 – 1.0 mm

2 = Diameter of PARL > 1.0 – 2.0 mm

3 = Diameter of PARL > 2.0 – 4.0 mm

4 = Diameter of PARL > 4.0 – 8.0 mm

5 = Diameter of PARL > 8.0 mm

Score + E = Expansion of periapical cortical bone

Score + D = Destruction of periapical cortical bone

**Periapical Index (PAI) – Orstavik/Kerekes (1986)**

**Radiographic Assessment of Apical Periodontitis**:

1 – Normal – No Periapical bone loss evident

2 – Small bony changes periapically, ***not*** *pathognomic for AP*

3 – Bony changes with Mineral Loss, *characterisitic of AP*

4 – AP with well defined RL area

5 – Severe AP with radiating expansion of bony changes

**Cone Beam CT Periapical Index (CBCTPAI) – Estrella (2008)**

CBCT Assessment of Apical Periodontitis:

0 – Normal – No Periapical bone loss evident

1 – PARL: >0.5 – 1 mm E – Expansion of Periapical cortical bone

2 – PARL: >1 – 2 mm D – Destruction of Periapical cortical bone

3 – PARL: >2 – 4 mm

4 – PARL: >4 – 8 mm

5 – PARL: >8 mm

# Give a differential diagnosis for a periapical radiolucency

1. Periapical Granuloma, Cyst, Abscess
2. Periapical Scar
3. Keratocystic Odontogenic Tumor (KCOT) aka OKC (multilocular)
4. Central Giant Cell Granuloma (multilocular)
5. Ameloblastoma (multilocular)
6. Metastatic carcinoma (breast, prostate, kidney)
7. Nasopalatine duct cyst (Anterior Maxilla only- between centrals)
8. Benign fibro-osseous lesions (early stages): Periapical/Florid/Focal cemento-osseous dysplasia, Central Ossifiying Fibroma
9. Lateral periodontal cyst (Lateral RL demarcated RO border, premolars/max lat)
10. Traumatic Bone cyst (men, 1st/2nd decade, RL demarcated/scallops roots)
11. Brown’s tumor (HPT), Multiple Myeloma, LHC Histocytosis, FD/Paget’s

MACHO – Multilocular RLs – Myxoma, Ameloblastoma, Central giant cell granulomas, Hemangioma, OKC (KCOT)

\*Multiple KCOTs – Basal cell nevus syndrome

# Give a differential diagnosis for a periapical radiolucency

Histological Biopsy Reviews:

1. *Bhaskar 1966* – No distinction radiographically between cyst/granuloma
2. *Simon* – *“Bay cyst”;* CBCT able to differentiate Cyst/Granuloma – Grey value
3. ***Nair* *1996*** – Granulomas 50%, Abscesses 35%, Cysts 15% (True 9%, Pocket 6%)
4. ***Spattafore 1990*** – WVU (1659 biopsies); Granulomas 52%, Cysts 42%, Scars 2%, Other 4%, “rule of 2s”
5. ***Koivisto* *2012*** – U of Minnesota (9723 biopsies):
   1. *Granulomas 40%*
   2. *Cysts 33%*
   3. Others 20%: *KCOTs 8%, CGCGs 1.3%, Ameloblastomas 1.2%, Metastatic <1%*

# Give a differential diagnosis for a periapical radiopacity

1. Condensing Osteitis (Focal sclerosing osteomyelitis) – LEO
2. Idiopathic osteoscleroses (aka Dense Bony Islands)
3. Benign fibro-osseous lesions (later stages): Periapical/Florid/Focal cemento-osseous dysplasia, Central Ossifying Fibroma
4. Odontoma
5. Cementoblastoma
6. Osteoma
7. Central Ossifying Fibroma (COF)
8. Calcifying odontogenic cyst (COC) – mixed (Gorlin’s cyst)
9. Calcifying epithelial odontogenic tumor (CEOT) – mixed (Pinborg)
10. Adenomatoid odontogenic tumor (AOT) - mixed

Cemento-osseous dysplasia (cementoma) forms - Florid, Focal, Periapical

Odontomas - Compound, Complex (“little teeth”)

Condensing Osteititis - **85%** resolve following NSRCT (***Eliasson***)

###### Stressed pulp syndrome & effect of restorative dentisty

1. ***Abou-Rass*** – Stressed pulps = Multiple restorations, Slow test results; NSRCT before restoration
2. *Felton* – Full coverage restorations led to a higher incidence (10-18%) of PN
3. *Zach* – Heat is capable of causing Pulpal Necrosis (20 deg = 60% necrosis)
4. *Bergenholtz* – Pulpal Necrosis: abutments: 15%, non-abutments: 3%

# *Cheung 2005* –Pulp vitality: Full coverage crown @ 10 yrs- 84%, 15 yrs- 81%; FPD abutment: 10 yrs- 70%, 15 yrs- 66% (At 10 yrs- 15% PFMs/30% FPD/50% Anterior FPD abutments are Necrotic requiring NSRCT)

1. ***Murray/Smith*** – *Remaining Dentin Thickness = #1 Factor for pulpal vitality* (vs. type of restoration placed, drill speed, coolant, and preparation method)

**Effects of restorative dentistry on the pulp**

***Murray***

1. Preparations within **0.5 mm** of Pulp = **Odontoblast injury**
2. **Survival of Odontoblasts** is critical for Pulp vitality and dentin repair
3. Caries → host-derived MMPs (collagenases, etc) → *Soluble growth factors released from dentin* (i.e.: *TGF-β, BMP, NGF, VEGF*) → stimulation of odontoblastic reparative dentin formation

***Murray/Smith***

1. Pulpal inflammation was most severe when **RDT < 0.25 mm**
2. **Cavity RDT** and **Bacterial leakage** affect survival of odontoblasts/pulp
3. Pulpal inflammation was highly correlated with **bacterial leakage**!

***Pashley***

1. The number, size of lumen, and surface area of dentinal tubules ↑ closer to the pulp (18,000→45,000/mm2, 0.8→2.5 μm, 5→22%)

**Does heat damage the Pulp?**

***Zach/Cohen OOO 1965*** – Monkey study, effect of temp increase on pulp

1. **4 °C – 100% pulps recovered**
2. 10 °C – 85% recovered; 15% necrotic
3. 20 °C – 40% recovered; 60% necrotic
4. **>20 °C – none recovered**

* Critical increase of intrapulpal temperature:
  + **5.5 °C = Irreversible pulpal injury**
  + **11 °C = Pulpal necrosis**
* Without water coolant, Avg intrapulpal temperature rise > 5.5 °C

**Facial Space Infections:**

*Laskin; Hohl 1983*

**Mandible/Below:**

|  |  |  |
| --- | --- | --- |
| **Fascial Space** | **Source** | **Borders** |
| Buccal Vestibule | Any Mand. Tooth; B cortical plate; Apex lies above Mentalis (ant) or Buccinator (post) | Buccal cortical plate, alveolar mucosa, Mentalis (anterior), Buccinator (posterior) |
| Mental Space | Mand. Anterior; B cortical plate; Apex lies below attachment of Mentalis | Mentalis (superiorly), Platysma (inferiorly) |
| Submental Space | Mand. Anterior; L cortical plate; Apex lies below attachement of Mylohyoid m. | Mylohyoid (superiorly), Platysma (inferiorly) |
| Sublingual Space | Any Mand. Tooth; L cortical plate; Apex lies above attachement of Mylohyoid m. | Mucosa of Floor of Mouth (superiorly), Mylohyoid (inferiorly), Mandible (laterally) |
| Submandibular Space | Mand. Posterior; L cortical plate; Apex lies below attachment of Mylohyoid m. | Mylohyoid (superiorly), Platysma (inferiorly), Mandible (laterally) |

**Facial Space Infections (cont.):**

**Lateral Face/Cheek:**

|  |  |  |
| --- | --- | --- |
| **Fascial Space** | **Source** | **Borders** |
| Buccal Vestibule (maxillary) | Max Posterior; B cortical plate; Apex lies below Buccinator | Buccal cortical plate, alveolar mucosa, Buccinator (superiorly) |
| Buccal Space (max/mand) | Max Posterior:B cortical plate; Apex lies above Buccinator  Mand Posterior: B cortical plate; Apex lies below Buccinator | Buccinator (medial), Skin of cheek (lateral), Zygomatic arch/Buccinator attachment (superior), Mandible/Masseter attachment (inferior) |
| Submasseteric | Impacted 3rd Molar | Ramus (medial), Masster (lateral) |
| Temporal Space | Infection spread superiorly from the pterygomandibular or inferiorly submasseteric spaces | Deep Temporal: Skull (medial), Temporalis (lateral)  Superficial Temporal: Temporalis (medial), Fascia |

**Facial Space Infections (cont.):**

Mid-Face:

|  |  |  |
| --- | --- | --- |
| **Fascial Space** | **Source** | **Borders** |
| Palate | Any Max tooth; Apex near Palate | Palate (superior); Periosteum (inferior) |
| Base of Upper Lip | **Max C.I**.; B cortical plate; Apex above Orbiularis *oris* | Mucosa upper lip; Orbicularis *Oris* (inferior) |
| Canine Space (Infraorbital) | **Max Can.-1st PM**; B cortical plate; Apex above Levator Anguli Oris | Levator *A*nguli *O*ris (inferior); Levator *L*abii *S*uperioris (superior)  ***AOLS*** |
| Peri-Orbital Space | Infection *spread from Canine or Buccal spaces* | Lies deep to Orbicularis *Oculi* |

**Additional spaces:**

**-** Pterygomandibular space (Moderate – Severe Trismus): *2nd or 3rd molar* drains directly into space or *Infected IAN B*. Borders: Medial Ptergyoid (medial), Lateral Pterygoid (superiorly) Lateral surface of Ramus of Mandible (lateral)

**Fascial Space Infections**

**Complications:**

1. **Ludwig’s Angina = Sublingual + Submental + Submandibular spaces bilaterally** →pharyngeal/cervical spaces →Airway closure **(life threatening cellulitis)**
2. **Cavernous sinus thrombosis** = Infection of **midface** (spread from ***canine or periorbital*** **spaces**) → Inflammation/Pressure within the *Infratemporal space* →*Reverse direction* of *venous blood flow & stasis* within the *Cavernous sinus* →*Thrombi* formation within *cavernous sinus.* Symptoms: Unilateral periorbital edema, headache, bulging of eye, blindness, fever

**Signs of Infection:**

Extraoral: Swelling, Dysphagia, Trismus, Lymphadenopathy

Lymphadenopathy: *Submental (lower incisors), Submandibular (all other teeth)*

Intraoral: Swelling, Sinus Tract

**Treatment:**

Surgical (I&D, decompression), Antibiotics (Pen VK, Clindamycin), Supportive (Analgesics, Heat, Fluids)

**Morphology**

**Morphology**

|  |  |  |  |  |  |
| --- | --- | --- | --- | --- | --- |
| **Max Anterior Teeth** | **1** | **2** | **3** | **4** |  |
| Vertucci ‘84 | 100 |  |  |  | Dye: decalc/dye/clear |
| **Max 1st Premolar** |  |  |  |  |  |
| Hartwell/Belizzi ‘85 | 6 | 91 | 3 |  | Retrospective chart review: 514 1st PMs (over 13 yrs) |
| Vertucci ’79 | 25 | 70 | 5 |  | Dye: 400 1st PMs – decal/dye/clear |
| **Max 2nd Premolar** |  |  |  |  |  |
| Hartwell/Belizzi ‘85 | 40 | 59 | 1 |  | Retrospective chart review: 630 2nd PMs (over 13 yrs) |
| Vertucci ‘79 | 75 | 24 | 1 |  | Dye: 400 2nd PMs – decalc/dye/clear |

**Morphology**

|  |  |  |  |  |  |
| --- | --- | --- | --- | --- | --- |
| **Max 1st Molar** | **1** | **2** | **3** | **4** |  |
| Neaverth ‘87 |  |  | 23 | 77 | Retrospective chart review: 228 1st Molars  Type II: 36%, III: 61% |
| Stropko ‘99 |  |  | 27/7 | 73/93 | Retrospective chart review: 1096 1st Molars (↑ MB2 after 6 yr)  Type II: 45%, III: 55% |
| Wolcott ‘02 |  |  |  | 60 | Prospective clinical cases: 3578 RCT/Retxs (6 Endodontists) |
| **Max 2nd Molar** |  |  |  |  |  |
| Fogel/Peikoff/Christie ‘96 | 3 | 7 | 57 | 23 | Retrospective chart review: 520 2nd Molars (3 Endodontists) |
| Wiene/Eskoz ‘95 |  |  | 60 | 40 | In Vitro: 73 Ext teeth, Burs and files, Radiograph  Type II: 21%, III: 16%, IV: 3% |
| Wolcott ‘02 |  |  |  | 35 | Prospective clinical cases: 2038 RCT/Retxs (6 Endodontists) |

**Morphology**

|  |  |  |  |  |  |
| --- | --- | --- | --- | --- | --- |
| **Mand Incisors** | **1** | **2** | **3** | **4** |  |
| Henry/Rankine-Wilson ‘65 | 60 | 40 |  |  | Radiographs/Histo Sectioning  Type III: 13% |
| Benjamin/Dowson ‘74 | 59 | 41 |  |  | #15 kfile/Radiographs  Type III: 1.3% |
| **Mand Canine** |  |  |  |  |  |
| Vertucci ‘84 | 80 | 20 |  |  | Dye: decalc/dye/clear |
| **Mand 1st Premolar** |  |  |  |  |  |
| Cleghorn ‘07 | 76 | 24 |  |  | Literature Review: 6700 1st PMs |
| Trope ‘86 | 66/86 | 34/14 |  |  | Retrospective Radiographic Evaluation: 400 AA/400 White |
| **Mand 2nd Premolar** |  |  |  |  |  |
| Cleghorn ‘07 | 91 | 9 |  |  | Literature Review: 7700 2nd PMs |
| Trope ‘86 | 92/97 | 8/3 |  |  | Retrospective Radiographic Evaluation: 400 AA/400 White |

**Morphology**

|  |  |  |  |  |  |
| --- | --- | --- | --- | --- | --- |
| **Mand 1st Molar** | **1** | **2** | **3** | **4** |  |
| Hartwell/Belizzi ‘82 |  | 0.1 | 65 | 35 | Retrospective chart review: 846 1st Molars  M root: Type II: 36%, III: 62% |
| Skidmore/Bjorndal ‘71 |  | 7 | 63 | 28 | Plastic resin casts of internal anatomy  M root: Type II: 40%, *III: 60%*  D root: Type II: 60%, *III: 40%* |
| **Mand 2nd Molar** |  |  |  |  |  |
| Hartwell/Belizzi ‘82 | 1 | 4 | 89 | 5 | Retrospective chart review: 420 2nd Molars |
| Weine ‘88 | 1 | 4 | 81 | 11 | PA radiographs and k-files  (75 teeth, C shape = 2.7%)  M root: Type II: 52%, III: 40% |

**Morphology**

C-shaped canals:

***Fan*** *– Classifications:*

Type I: C

Type II: Semicolon

Type III: 2 or 3 dots

Type IV: No discernable canal

***Cook/Cox 1979*** (U of Washington) – **8%** Incidence

Middle-Mesials:

***Dean Baugh/James Wallace*** – **1-15%** Incidence, Mesial root Mand 1st Molars

Radix:

Radix Entomolaris: Distolingual Supernumerary Root in Mandibular Molars

Radix Paramolaris: Mesiobuccal Supernumerary Root in Mandibular Molars

***Calberson* –** **1-5%** (AA, Indian), 5-30% (Mongoloids – Chinese, Eskimo, AI)

**Morphology**

Classifications:

1. ***Weine***-Type I (1-1), II (2-1), III (2-2), IV (1-2)
2. *Vertucci* - Type I (1-1), II (2-1), III (1-2-1), IV (2-2), V (1-2), VI (2-1-2), VII (1-2-1-2), VIII (3-3)

MB2:

1. *Weine*- Classic histological study of canal morphology
2. *Neaverth*- Access should be extended from triangular to rhomboidal for MB2

Accessory/Lateral Canals:

1. ***De Deus*** – *27%* incidence of *lateral canals*, *19% Apical 1/3rd*
2. ***Gutmann*** – *28%* incidence of *furcation canals*, *10% extend to PDL*

Isthmuses

1. ***Weller*** – MB root Max 1st Molars, *4 mm- 100% incidence* isthmus (3-5 mm from apex highest)
2. ***Von Arx*** – M root Mand 1st Molars, *90% incidence isthmus 3 mm* from apex

**Morphology**

**Dens Evaginatus:**

1. Definition: Developmental anomaly resulting in an accessory cusp (*enamel tubercle*) on the occlusal surface of posterior teeth or lingual surface of anterior teeth; thin enamel layer covering dentin core with *slender pulpal extension*
2. Prevalence:
   1. ***Levitan/Himel* *2006*** – Review Article: Asian descent **0.5-4.3%, Lower Premolars/bilateral,** Higher in Chinese/Eskimo/Native Americans –Mongoloids (up to 15%, *see also Radix*)
3. Dental Treatment:
   1. *Augsburger 1996* – Pulpal management of Dens Evaginatus
   2. *Levitan/VanHimel* - Cover with composite. NSRCT (mature) or MTA Pulpotomy (vital/inflammed/immature), MTA apexification/REP (necrotic/immature)

**Morphology**

**Dens Invaginatus (Dens in Dente):**

1. Definintion: Developmental anomaly resulting in *invagination of an enamel-lined tract extending into the root*, *with or without exposure of the dental pulp*
2. Types: ***Oehlers 1957***: **Type I**: occuring within confines of crown, not extending beyond CEJ; **Type II**: invades root but remains confined as a blind sac w/in root canal, no separate foramen;**Type III**: penetrates thru the root and exits as separate AF, *No communication* with the pulp
3. Prevalence:
   1. ***Hulsmann IEJ 1997* (review)** – **0.3-10%;** *bilateral 43%*, **Max Lats**
   2. *Hovland 1977* – **0.4-10%** overall incidence, **Max Lateral incisors**
4. Dental Treatment:
   1. *Narayana/Hartwell JOE 2012* – Case report (11 yo #7), Type III, case specific, removal of dens in dente and pulpal revascularization
   2. *Hulsmann 1997* – RCT of dens tract when separate AF; WV obturat.
   3. *Alani/Bishop IEJ 2008* – Case specific; RCT of dens only (Type III) or both dens/pulp; Removal of dens tract and tx as 1 canal (Type III)

**Orifice Location**

***Krasner/Rankow 2002*** – Laws of Pulp chamber anatomy:

1. **Law of Centrality** – Floor of Pulp chamber located in center of tooth at CEJ
2. **Law of Concentricity** – Walls of Pulp chamber are concentric to external surface of tooth at CEJ
3. **Law of CEJ** – CEJ is most consistent landmark for locating pulp chamber
4. **Law of Symmetry 1** (Except Max Molars) – Orifices are equidistant from line drawn M-D direction through pulp chamber floor
5. **Law of Symmetry 2** (Except Max Molars) – Orifices lie on line perpindicular to line drawn in M-D direction through pulp chamber floor
6. **Law of Color Change** – Color of pulp chamber floor is darker than walls
7. **Law of Orifice location 1** – Orifices are located at junctions of floor-wall
8. **Law of Orifice location 2** – Orifices are located at angles of floor-wall junctions
9. **Law of Orifice location 3** – Orifices are located at terminus of root developmental fusion lines

*Wilcox/Walton 1989* – In vitro – related location of canal orifices to occlusal landmarks (cusp tips, grooves) – Max molars – Avoid mesial marginal ridges, Mand molar access too far lingual

**Tooth Developmental Anomalies**

1. Fusion: 1 crown/2 roots; joining of 2 developing tooth germs during root formation; may be complete or incomplete fusion w/ separate or joined pulps
2. Gemination: 2 crowns/1 root; single enamel organ attempts to create 2 teeth
3. Concrescence: Adjacent teeth are joined by cementum only (form of fusion)
4. Amelogenesis Imperfecta: 3 types: Hypoplastic, Hypocalcified, Hypomaturation, Hereditary defects in enamel formation/maturation; yellow-brown discoloraiton; Radiographically: Dentin thin/normal roots

**Tooth Developmental Anomalies (cont.)**

1. Dentinogenesis Imperfecta: Opalescent Dentin; Autosomal Dominant; 3 types: 1- Osteogenesis Imperfecta, 2- Dental only, 3- Brandywine: Type 2 + Huge pulp chambers/canals (shell teeth), Periapical Radiolucencies; Clinical features: Opalescent translucency, yellow-brown discoloration, easily fractured enamel, blunted roots
2. Dentinal Dysplasia: Autosomal Dominant; 2 types: 1- **Radicular**: Crowns Normal, *SHORT roots, Obliterated pulps*, *PARLs*; 2- **Coronal**: Crowns Normal, *SHORT roots, Large pulps*

**Anatomy Studies**

1. Classic Studies:
   1. *Hess* – 1920s – Original morphology studies (sectioning/dye studies)
   2. *Walton* - Canal not seen radiographically is present histologically on sectioning
   3. ***Kuttler*** ***1955*** – Distance from the major to the minor diameters:
      1. **0.525mm (18-25y/o)**
      2. **0.659mm (>55 y/o)**
   4. ***Burch*** ***1972*** – Measured from the occlusal aspect of the major diameter to the apex**. Average distance for all roots = 0.59 mm**
   5. *Vertucci* – Decalcification/Dye/Clearing studies on canal anatomy
   6. *Stein* – Measured from the minor diameter (CDJ) to the major diameter = 0.72mm average. Foramen width increases with age but CDJ width does not
   7. ***Dummer* *1984*** – 270 Ext. teeth - Mean A-F distance: 0.38 mm, *Mean A-C distance: 0.89 mm*; 4 types of ACs described

# Pulpal Pathophysiology & Inflammation

# Hydrodynamic Theory of Dentinal Sensitivity

***Brannstrom*** –

1. Heat causes inward fluid movement in tubules
2. Cold causes outward fluid movement in tubules
3. Concurrent distortion of odontoblastic process stimulates 1° neurons at the pulpo-dentinal junction.
4. Distortion leads to impulse conduction

**Pulpal Functions & Formation**

1. Functions: Induction, **Formation**, Nutrition, Defense, Sensation
2. Development: Ectomesynchymal cells proliferate to form Dental papilla which gives rise to odontoblasts/dentin and pulpal tissues

**A-delta and C-fibers**

1. ***Narhi*** -
   1. **A delta fibers**: Mechanoreceptors; respond to hydrodynamic stimuli (i.e.: cold, heat, air, hyper-osmotic sol; plexus of rashkow; dentinal pain
   2. **C fibers**: Polymodal nociceptors; respond to tissue injury, Capsaicin, prolonged heat, inflammatory mediators, acids; pulp proper; pulpal pain
2. ***Johnsen 1983*** -
   1. >2000 neural axons innervate human premolar
   2. **80% pulpal neurons are unmyelinated C fibers**
   3. *Myelinated A fibers are late to innervate pulp*, may take up to 5 years (EPT unreliable in young teeth – see *Fuss/Trowbridge, Fulling/Andreasen*)
   4. Threshold stimulus level decreases significantly with apical closure
3. ***Matthews* *1994***-
   1. **90% of myelinated A fibers in dental pulp are A-delta fibers**

**Sympathetic & Parasympathetic Innervation**

1. ***Trowbridge* *1986*** –
   1. Sympathetic efferent fibers originating from the superior cervical ganglion
   2. Innervate *smooth muscle cells of arterioles and precapillary sphincters*
   3. **Vasoconstriction and reduction of blood flow** to the pulp
2. *Edwall 1985* –
   1. Sympathetic fibers derived from the superior cervical ganglion form plexuses around pulpal arterioles
   2. Responsible for constriction of arterioles/decrease in blood flow to pulp
   3. Sympathetic neural terminals contain **NE and NPY**
3. ***Sassano* *1995*** –
   1. Absence of parasympathetic (VIP) vasodilation in the pulp

**Dentinal Tubules & Odontoblastic Processes**

1. ***Pashley*** – Diameter and density of tubules increases closer to the pulp:
   1. Density: DEJ – 18,000 tubules/mm2, Pulp – 45,000 tubules/mm2
   2. Diameter: DEJ – 0.8 μm, Pulp – 2.5 μm
   3. Occupied Space: DEJ – 1% surface area, Pulp – 22% surface area
2. ***Byers*** – Intratubular A fibers extend into dentinal tubules as far as **100 μm**, most numberous in the pulp horns (40% tubules), least numerous in root dentin (1% tubules)
3. *Sigel* – Odontoblastic process extends to DEJ
4. ***Holland*** – Odontoblastic process extends **½ way** through the tubule.
5. *Aubin* – Odontoblastic process extends to DEJ

## Trowbridge - Histopathology of foreign body reactions

**Type I Anaphylactic** - **IgE** **mediated** (Mast cells/Basophils → Histamine, C4 leukotrienes – ↑ vasodilation/vascular permeability and bronchial smooth muscle contstriction), **IMMEDIATE** ie: **drugs**, foods, **asthma**, bites, allergic rhinitis

**Type II Cytotoxic - IgG, IgM mediated** (complement or phagocytosis)- Transfusion rxn, autoimmune hemolytic anemia, idiopathic thrombocytopenia

**Type III Immune complex (Ag-Ab) rxn** – IgG form complexes w/ complement, **6-8 hrs**, ie: serum sickness, arthus, immune vasiculitis, lupus, **viral hepatitis**

**Type IV Delayed Hypersensitivity rxn** - **Cell mediated** (no Ab): **T cell** (Killer T cells, Memory T cells), **Macrophages**; antigen sensitized T cells/Macrophages. More important than anaphylaxis rxn. **24-48 hours DELAYED**

ie: 48 hours **contact dermatitis, poison ivy**

tissue graft rejection, **TB**, **autoimmune diseases**

**Granulomatous Inflammation (i.e.: Periapical granuloma)**

**What Immune cells are found in the *Healthy Pulp*?**

***Jontell/Bergenholtz 1998***

1. **T-lymphoctyes** (CD4+ and CD8+): B cell/Macrophage activation, mem.
2. **Macrophages**: Phagocytosis, APC
3. **Dendritic Cells**: APC, phagocytosis; Perivascular/Periodontoblastic
4. ***Lack of B-lymphocytes, Mast cells from healthy pulps*** (*see Suda*)

***Soh*** - Dendritic Cells present in pulp

***Byers*** – Fibroblasts are most numerous cell in pulp and produce **NGF** and **proinflammatory cytokines** during inflammation

***Hahn*** – T lymphocytes in normal pulp, T suppressor most predominant

***Suda*** – Mast Cells are NOT present in normal pulps

**Pulpal Changes as related to depth of bacteria** –

1. ***Baum*** – Found correlation between depth of penetration of bacteria within dentin and severity of inflammation
2. ***Brannstrom/Lind*** – Pulpal changes occur early in caries, even in *incipient* *lesions*. Impairment of odontoblast layer, accumulation of lymphocytes
3. ***Reeves & Stanley*** – *Irreversible pulpitis* detected when bacteria were **0.5mm** from the pulp, **little pathosis seen if >1.2 mm from pulp**. If bacteria **invade repairative dentin** **= irreversible pulpitis.**
4. ***Stanley*** – Rate of repairative dentin formation = 1.49 micrometers/day, *tertiary dentin begins 19 days after operative procedures.*
5. ***Seltzer/Bender* (classic)** – Described classic caries progression to pulp
6. ***Jontell/Bergenholtz*** – Macrophages/Dendritic cells (APCs) initiate pulpal response to caries. T lymphocytes present. B lymphocytes/mast cells later arrivals. Multiple immune responses: Antigen-Ab IgG (III), Delayed type Hypersensitivity w/T cell/Macrophages (IV), Vascular and Neurogenic interactions.

**Pulpal Reactions to Caries** (*See Baum, Reeves/Stanley, Branstromm/Lind)*

**Shallow - Moderate caries**:

1. 1st defense: **Sclerotic dentin (crystals) deposition**, ↓dentin permeability
2. 2nd defense: **Tertiary dentin formation**, Mild stimuli – Reactionary dentin, Aggressive stimuli (death of odontoblast) – Reparative dentin
3. 3rd defense: **Immune response**
   1. ***Chronic inflammatory response*** –Initially Odontoblasts, later dendritic cells as most peripheral cells contain PRRs to respond to PAMPs with innate/adaptive immune response
   2. *Release of Cytokines, Chemokines* – **phagocytosis, chemotaxis**, recruitment of lymphocytes, macrophages, and plasma cells
   3. ***Neurogenic inflammation*** – **SP, CGRP, NKA** released from pulpal nociceptors (PRRs – TLRs on nociceptors)

**Deep caries**:

1. **Acute excerbation** of chronic inflammatory response – Influx of PMNs
2. *Localized inflammation, Focal Microabscesses, Progressive necrosis*

**Inflammation: PAMPs & PRRs**

*Initiation of host response – Innate Immunity*

PAMP = Pathogen Associated Molecular Pattern (ie: LPS, LTA, Peptidoglycan)

PRR = Pattern Recognition Receptor (ie: Toll Like Receptors (TLRs), CD-14)

***Wadachi/Hargreaves 2006*** – TRPV1 (Capsaicin) C fiber nociceptors express TLR-4 and CD-14, subclasses of Pattern Recognition Receptors able to sense bacterial presence through PAMPs (i.e.: LPS), and able **to directly activate these nociceptors or influence their release of Neuropeptides**

***Farges 2008*** – **Odontoblast TLR-2** stimulation by bacterial PAMP results in release of proinflammatory cytokines, chemokines, other mediators

*Horst 2007* – **Odontoblasts** contain **TLR 2, TLR 8**

*Jiang 2006* – **Odontoblasts, Pulpal vascular endothelial cell**s contain **TLR4**

***Farges 2010* – Dendritic cells TLR-2** stimulation by LTA (PAMP)

**Pathogen Associated Molecular Patterns (PAMPs)**

**LPS**

1. *Dickerson 1998* - LPS activation of pulpal nociceptors – SP/CGRP release (**TLR-4**)
2. ***Akira 2001*** - LPS stimulation of TLRs (PRRs) on Dendritic cells

**LTA**

1. ***Farges 2013*** – LTA activation of **TLR2** on pulpal odontoblasts stimulates pro-inflammatory cytokine release
2. ***Farges 2010*** – LTA activation of **TLR2** on Dendritic cells stimulates production of TNF-α, IL-1β, and IL-8

**Peptidoglycans**

1. ***Adachi 2007*** – Peptidoglycan promotes chemokine production by pulpal fibroblasts

**Acute Inflammation (Innate Immunity)**

**1st 24 hrs**

1. **PAMPs bind PRRs** (TLRs, CD14) on resident immune cells (dendritic cells, macrophages), odontoblasts, and peripheral nociceptors (TRPV1 C fibers)
2. **Pro-inflammatory cytokines (i.e.: IL-1β, IL-6, IL-8, TNF-α) and neuropeptides (CGRP, SP, NKA) are released** to activate/recruit immune cells as well as sensitize/activate nociceptors, ↑ nerve sprouting, and effect vasodilatory responses
3. **Complement activation** via cytokines and neuropeptides → releasing C3a, C5a → vascular endothelium for chemotaxis, opsonization and killing of foreign antigens

**24hrs +**

1. **Cytokines (i.e.: IL-1, TNF-α) & C3a/C5a effect endothelial cells** for chemotaxis and migration of PMNs, Bradykinin, other pro-inflammatory cells
2. **C3a/C5a & neuropeptides (SP, CGRP) activate mast cells** to produce Histamine, Leukotrienes, Platelet Activating Factor (Vasodilation, ↑ Vascular Permeability)
3. **Sensitization/Activation of Nociceptors** → Hyperalgesia, Allodynia, Spont. pain
4. **Vasodilation, Vascular Permeability** → Edema, Inc. Tissue pressure
5. **Non-specific phagocytosis** – *PMNs (0-48hrs), Macrophages (48 hrs +)*
6. **Cytokines effect Osteoclasts** to promote RANK/RANKL binding and bone resorption

**Chronic Inflammation (Adaptive Immunity)**

**2 Components of Adaptive Immune Response (48 hrs +):**

1. **Humoral Immune Response** – Antibody mediated response
2. **Cellular Immune Response** – T cell mediated response – CD4+ T cells
3. **Antigen presenting cells (APCs),** i.e.: *Macrophages, Dendritic cells, B cells*, present to immature TH0 cells within lymph nodes via MHC II receptor, activating TH0 cells
4. **TH0 cells differentiate into TH1or TH2** cells via cytokine regulation: TH1 via *IFN-δ,* TH2 via *IL-4, IL-10* (suppresses IFN-δ and TH1 production)
5. **Cellular Immune Response**: TH1 → Maximizes killing by Macrophages, CD8+ cells (NKT cells, Cytotoxic T cells), Memory T cell production
6. **Humoral Immune Response**: TH2 → B cell activation → Plasma Cells → Antibody production (allows binding to antigen for complement killing or phagocytosis), Memory B cell production

# Inflammatory Mediators

Main objectives of pro-inflammatory mediators:

1. **Vasodilation** and **Increase Vascular Permeability**
2. **Recruit/Activate Inflammatory cells** (PMN, Mast cells, Macrophages, Lymphocytes, Bradykinin) & Complement system (C3a, C5a)
3. **Sensitize/Activate Nociceptors**

# Vasodilation

1. Histamine – Mast cells (stimulated by C3a, SP, CGRP), Platelets
2. Prostaglandins (Prostacyclin) – All leukocytes, Mast cells (COX pathwy)
3. CGRP – Pulpal nociceptors

**Increased Vascular Permeability**

1. Bradykinin – Plasma activated through kinin system cascade
2. Leukotrienes – All leukocytes, Mast cells (LIPOX)
3. C3a, C5a – Plasma complement system cascade
4. Substance P – Pulpal nociceptors

**Opsonization**

1. C3b, C5b – Plasma complement system cascade

# Inflammatory Mediators (cont.)

# Endothelial Adhesion Expression

# TNF (cytokine) – Macrophages

1. IL-1 (cytokine) – Macrophages
2. Chemokines – Macrophages, PMNs, Endothelial cells, Fibroblasts

**Leukocyte Activation and Chemotaxis**

1. C3a, C5a – Plasma complement system cascade
2. Leukotrienes – All leukocytes, Mast cells
3. Chemokines – Macrophages, PMNs, Endothelial cells, Fibroblasts
4. TNF – Macrophages

**Tissue Damage**

1. Lysosomal enzymes – PMNs, Macrophages - phagocytosis
2. Free oxygen radicals – Activated leukocytes
3. Nitric oxide – Macrophages

# What is the role of the neuropeptides?

**Neurogenic Inflammation**

1. Vasodilation (via CGRP & mast cells → histamine)
2. Increased Vascular Permeability (via SP & mast cells → Leukotrienes)
3. Nerve sprouting/Pain (via SP/CGRP → Fibroblasts → NGF) - *Byers*
4. Activate Macrophages/T lymphocytes → cytokines: IL-1, TNF-α, IL-6
5. Bone resorption and Immune regulation in AP development - *Byers*
6. ***Sessle*** – Neuropeptides mediate release of inflammatory mediators from immune cells (i.e.: macrophages, mast cells, platelets) resulting in inflammatory cascade
7. ***Byers***– injury leads to “sprouting” of sensory nociceptors: **Neuropeptides (SP/CGRP) → fibroblasts → NGF → Nerve sprouting** = ↑ Nociceptor receptive field + response
8. *Wakisaka 1990*– neuropeptides may help regulate pulpal blood flow + pain transmission as well as promote inflammatory response
9. *Hargreaves* – sympathetic transmission may modulate pain (capsaicin study)

**Neuropeptides**

**5 Major Neuropeptides**:

*Wakisaka 1990, Hargreaves 1994, Caviedes-Bucheli IEJ 2006*

**SP (Substance P):**

1. *Trigeminal 1° cell bodies (mainly C-fibers) – Trigeminal ganglion*
2. Interact with **Mast cells: ↑ Release of histamine & leukotrienes**
3. Activates **Macrophages/Lymphocytes: ↑ Release of inflammatory mediators (ie: Cytokines, PGs, Thromboxanes)**
4. Stimulate **Pulpal cells (ie: Fibroblasts, Odontoblast like cells): ↑NGF**

**CGRP (Calcitonin Gene Related Peptide):**

1. *Trigeminal 1° cell bodies – Trigeminal ganglion*
2. Interact with **Mast cells:** ↑ **Release of histamine & leukotrienes**
3. Stimulate **Pulpal cells (ie: Fibroblasts, Odontoblast like cells): ↑NGF**

**Neuropeptides**

**NKA (Neurokinin A)**:

1. *Trigeminal 1° cell bodies – Trigeminal ganglion*
2. Activates **Macrophages/Lymphocytes: ↑ Release of inflammatory mediators (ie: cytokines, PGs, Thromboxanes**)

**NPY (Neuropeptide Y)**:

1. *Sympathetic efferent derived* (co-localized with Norepinephrine) – Superior Cervical ganglion
2. Sympathetic vasoregulation - **↓ Vasodilation (↓ Pulpal Blood Flow)**

**VIP (Vasoactive Intestinal Peptide)**:

1. *Parasympathetic efferent derived* (co-localized with Acetylcholine)
2. **↑ Vasodilation (↑Pulpal Blood Flow)**

# Vascular response = LOCALIZED Inflammation

***Kim*** - Key components in pulpal inflammation:

1. Microcirculation – increased PBF by C fiber stimulation (neurokinin A, substance P, CGRP released from C fiber nerve terminals)
2. Sensory nerve activity – excitatory effect from increased pulpal blood flow *via increased tissue pressure* (effect on A delta fibers)

**VHK:**

1. ***V****an Hassel* – 1st to discuss **Localized Inflammation** – vascular collapse spreads incrementally from the site of injury, not by strangulation at the apex
2. ***H****eyeraas (Tonder)* – Pulpal lymphatics – drainage of interstitial fluid/proteins to ↓ Interstitial tissue pressure
3. ***K****im/Takahashi* – Pulpal Collateral Circulation circumvents blood flow around the area of injury/inflammation (**AV, VV shunts, Ushaped arteriole**)

# Who studied pulpal vasculature & localized inflammation?

1. ***VanHassel*** *–* **1st to discuss localized inflammation** in pulp – *vascular collape* spreads **incrementally** from site of injury (pressure differences)
2. ***Heyeraas (Tonder)*** – **localized increased tissue pressure** may persist in the inflamed area w/out a circumferential spread to the rest of the pulp. **Negative feedback system** prevents pulpal strangulation (**lymphatic drainage**)
3. ***Kim & Takahashi*** – discovered presence of **arteriovenous anastomosis and venous-venous anastomosis** and **u shaped arterioles** (unique feature of pulpal vascular network) = **collateral circulation** – circumvents blood flow around the area of inflammation (localized)

--Also found **sympathetic adrenergic vasoconstritor fibers (NPY) =** ↓ Vasodilation (↓ Pulpal Blood Flow)

**No Pulpal Strangulation Theory!**

**Are Mast Cells in the Pulp** ?

Yes:

*Farnoush* - Found in inflamed and non-inflamed pulpal tissue

No:

*Suda* – Mast Cells are NOT present in normal pulps

## Who found lymphatics in the pulp ?

1. *Bernick* – demonstrated lymphatics in the pulp
2. ***Heyeraas (Tonder)*** - Pulp may have a beneficial blood flow increase during inflammation in spite of simultaneously increased tissue pressure. This supports the concept of lymphatic drainage**. Localized increased tissue pressure without circumferential spread!** Lymphatic drainage of interstitial fluid/proteins prevents spread of tissue pressure

# Are antibodies present in the healthy pulp?

YES

1. *Langeland* *–* Antigens in the root canal system can initiate an immune response with antibodies
2. ***Hahn*** – IgG, major class of immunoglobulins in *normal and irreversible* groups

NO

1. ***Pulver***– **Normal pulps do NOT have immunoglobulins**-containing cells. In inflamed pulps, **IgG most common**, IgA, IgE, IgM containing cells are also seen.
2. ***Jontell/Bergenholtz*** – **B lymphocytes, mast cells, and Abs** are **NOT** present in **normal pulp**

# Describe the Compliment cascade

Activated by pro-inflammatory cytokines, neuropeptides, bacterial antigens

1. **Mediate vascular responses**: C3a, C5a → Mast Cells →Histamine (vasodilation), Leukotrienes (↑ Vascular Permeability)
2. **Leukocyte chemotaxis:** C3a, C5a → PMNs, T/B cells, Bradykinin
3. **Opsonizination** of targets for phagocytic cells (C3b, C5b)
4. **Directly damage** of target cells (C5-9, MAC)

Most important step is cleavage of C3

**Classical pathway**: activated by **Ab coated targets** or **Ag-Ab complexes (IgM, IgG – Type III foreign body rxn)**

Alternate pathway: activated by LPS, aggregated IgM or IgG, Ag-IgG complexes, plasmin

**Immune Modulators & Silent Pulpitis**

1. ***Holland/Michaelson*** – **Silent pulpitis: 40-60%**
2. ***Jaber/Dionne*** - **μ opioid receptors** present peripherally within the pulp on primary nociceptors
3. ***Narhi*** – **Endogenous opioids** and **somatostatin** released by inflammatory cells are capable of inhibiting firing of sensory nerve fibers
4. ***Mudie/Holland* *2006*** – **Endorphins** are located within lymphocytes in inflammed pulps

Endogenous Opioids (neuromodulators): Beta-Endorphins, Met- and Leu-Enkephalins, Dynorphins

Targets: Mu, Delta, Kappa receptors

# Periapical Pathophysiology

**Apical Periodontitis – Etiology - BACTERIA**

AP is caused by entry into PA tissues either by bacterial toxins, enzymes, or byproducts (TEBs) or direct invasion by microbes from the canal system

**Exogenous Factors**

I. Microorganisms:

1. ***Kakehashi 1965*** – Pulp exposure - germ free vs conventional rats - germ free rats: vital/hard tissue repair; conventional rats: pulp necrosis/AP
2. ***Sundqvist 1976***– Human study, evaluated necrotic traumatized teeth – w/o AP: no bacteria; w/ AP: Bacteria present (90% Anaerobes)
3. ***Moller/Fabricius* *1981***– Monkey study, Devitalized pulps/sealed 6-7 months: Sterile necrotic pulps – No AP changes; Infected necrotic pulps – AP inflammation/destruction

II. Bacterial Toxins/Bacterial Enzymes/Metabolic Byproducts (TEBs)

1. Toxins: LPS, LTA, Peptidoglycan
2. Enzymes: Hyaluronidases, Chondroitin sulfatases, Collagenases
3. Byproducts: Sulfur

**Apical Periodontitis – Etiology**

***Nair 1995***

III. Physical Insults (Foreign bodies)

1. Overinstrumentation of the canal system
2. Extrusion of obturating materials, dentin debris
3. Traumatic injury of the periapical tissues

IV. Chemical Insults (Foreign bodies)

1. Extrusion of Irrigants
2. Extrusion of Intracanal Medications
3. Extrusion of Root canal filling materials/sealers

**Endogenous Factors**

1. Cholesterol crystals

***Ricucci/Siqueira*** – Intraradicular biofilms are responsible for AP (CAGE)

**Periapical Pathology – Inflammatory Reactions**

1. Apical Inflammation: Periapical tissue reaction to *irritants emerging from the root canal system*, Resulting inflammation characterized by vasodilation, increased vascular permeability, and exudate
2. Apical Infection: Direct invasion of the periapical tissues by microorgansims that *establish an extraradicular infection*, evoke purulent inflammation, and subsequently produces tissue damage

**Pathogenesis**

1. Innate Immune Response (1st 48 hours)
2. Adaptive Immune Response (>48 hours)
3. Neurogenic Inflammation (24 hours +)

**Periapical Bone Destruction**

1. ***Stashenko/Wang*** - **Host immune response** mediates tissue destruction and bone resorption in response to bacterial infection. **TNF-α, IL-1β, IL-2, IL-6**
2. ***Byers 1990*** – **Sprouting** of CGRP containing nociceptors in the periapical tissues occurred *while vital pulp tissue remained coronally*, suggesting **neuropeptides may contribute to periapical lesion development**
3. ***Stashenko*** – Animal model (rats), **kinetics of bone resorption**:
   1. Bone destruction appeared radiographically~10 days (post p.e.)
   2. **Active phase 0-15 days**: rapid bone destruction, **TH > TS**
   3. **Stationary phase > 20 days**: slower bone destruction, **TS > TH**
   4. **T cells responsible for immunoregulation of bone destruction**

**RANKL: via IL-1, IL-6, TNF, PGs, Bradykinin, LPS =↑ osteoclast, ↑ bone dest.**

**Periapical Pathogenesis**

Granuloma vs. Granulation Tissue:

***Trowbridge 1990***

1. **Granuloma**
   1. Round, circumscribed inflammatory lesion consisting of an accumulation of *macrophages, lymphocytes, and a variable number of plasma cells, neutrophils, giant cells and mast cells all enclosed within a collagenous stroma.*
   2. **Periapical Granuloma** = Localized mass of chronic inflammatory tissue consisting of *macrophages, T lymphocytes, plasma cells, mast cells, giant cells and epithelium* formed in reaction to infection of the dental pulp which serves as a *constant source of antigenic material*.
2. **Granulation Tissue**
   1. Proliferative phase of wound repair consisting of *fibrin CT matrix* containing *endothelial cells and fibroblasts mixed with inflammatory cells of lymphocytes and macrophages*

# Periapical Pathogenesis

# *Marton JOE 2014 – Review of Immunological mechanisms in development of AP*

# Initial Inflammatory Response – Innate immune mechanisms (0-48 hrs)

* 1. Human PDL fibroblasts (PDLFs) – TLR response to Bacterial PAMPs (LPS, Peptidoglycan)
     1. *Upregulation of pro-inflammatory cytokines:* TNF-α, IL-6, PMN chemoattractant
     2. Upregulation of anti-inflammatory cytokines: TGF-β
  2. EBV, CMV, Herpes virus (*Sabeti*) contribute to stimulation of pro-inflammatory mediators – cytokines/chemokines
  3. *Vasodilation/Inc. vascular permeability, leukocyte chemotaxis/activation (PMNs, Macrophages), Phagocytosis (DCs, Macrophages), Cytokine/Chemokine production*

1. **Adaptive immune mechanisms (>48 hrs)**
   1. APCs initiate *differentiation of T cells*, *activation of macrophages/NKCs, specific antibody production*

# Periapical Pathogenesis

# *Marton JOE 2014 – Review of Immunological mechanisms in development of AP*

# Dynamics of Periapical Bone destruction and repair

* 1. *Early AP: TH1 cells predominant* (*Stashenko*) in early AP development and expansion. VEGF and angiogenesis associated with lesion expansion.
  2. *Late AP: TS cells*, *plasma cells/B cells/TH2 cell*s in late AP – lesion stabilization and healing (post 21 days – *Stashenko*)
  3. *MMPs*: *destruction of ECM by MMPs initiates bone resorption*
  4. ***Equilibrium of RANK-L and Osteoprotegrin (OPG)***
     1. RANKL expressed by osteoblasts, monocytes, DCs, fibroblasts, endothelial cells, PMNs, and activated T lymphocytes
     2. ***TNF-α, IL-1β, IL-6, IL-17, Bradykinin, PGE2 ↑ RANKL/Osteoclast expression***
     3. ***IFN-γ, IL-10, TGF-β ↓ RANKL/Osteoclast expression***
  5. Bone loss self limiting - *Equilibrium develops between destructive and repair mechanisms* – **PDL derived stem cells** adjacent to PA lesion may engineer bone, PDL, and cementum repair thru stimulation by growth factors such as TGF-β

# Cytokines and their activity

Cytokines are soluble polypeptide products of immune cells.

1. Modify behavior of other cells
2. Produce systemic effects
3. Act as growth factors
4. ***Stashenko*** – *IL-1β, TNF-α, PGE2, Bradykinin, and LPS* – stimulate resorption either alone or in synergistic combination
5. *Safavi* – TNF-α identified in periapical exudates from CAP
6. *Stashenko* – IL-1α, IL-1β, TNF-α and lymphocyte-derived lymphotoxin potentially stimulate resorption and inhibit reparative bone formation

# What cells are found in a periapical granuloma?

***Stern*** – (FMLP = Fibroblasts Macrophages Lymphocytes Plasma Cells PMNs)

Inflammatory Cells = 52% of all cells

1. **Macrophage = 24%**
2. Lymphocyte = 16%
3. Plasma cells = 7%
4. PMNs = 4%

Other Cells

1. **Fibroblasts = 42%**
2. Epithelial Cells = 5%
3. Vascular cells = 6%

***Stashenko*** – **T lymphocytes 50%**, PMNs 35%, Plasma cells, Macrophages 15%

*Perrini* – found mast cells in varying stages of activity

***Pulver*** – found **70% IgG**, 14% IgA, 10% IgE and 4% IgM (**GAEM**)

**Cysts** have **45% IgG, 45% IgA**, 5% IgE and 5% IgM (higher IgA)

*Torabinejad* – Granulomas & Cysts have T and B cells, T Cells were in greater quantity.

# Who studied LPS?

*Schilder* –

1. Pulpless teeth contain greater concentration than vital teeth
2. Symptomatic w/ AP contained greater concentrations than asymptomatic

*Bergenholtz* –

1. Endotoxin activity correlated with the presence and number of Gram – bacteria

***Horiba –***

1. Higher concentrations in symptomatic teeth than asymptomatic
2. Higher concentrations in teeth with radiolucencies
3. Higher concentrations in teeth with exudation than without

# Zones of Fish (NCIS)

Describes body’s way of isolating and localizing an infection in periradicular area

1. **Infection/ Necrosis** – Bacteria, PMNs (Innate Acute 0-48 hrs)
2. **Contamination** – Bacterial toxins, macrophages, lymphocytes (Innate Chronic 48hrs +)
3. **Irritation** – Macrophages, osteoclasts, lymphocytes, plasma cells (Adaptive)
4. **Stimulation** – Osteoblasts, fibroblasts (Proliferative phase)

**Symptomatic Apical Periodontitis**

*What is SAP?*

*Immediate Innate* *Immune response* to diffusion of inflammatory mediators, bacteria and bacterial toxins/enzymes/byproducts (TEBs) into the periapical tissues

*Cells Involved*

1. Mast Cells – Histamine, Leukotrienes, Cytokines, PGs released
2. Endothelial Cells – Vasodilation/Inc Vascular Permeability
3. PMNs – Principal cell of Acute immune response – *peak 24-48 hrs* – recrutied/activated by PAMPs, cytokines, chemokines → phagocytosis, release of inflammatory mediators, recruitment of leukocytes
4. Macrophages – 2nd wave in acute immune response – *peak 48-96 hrs* – activated by PAMPs, cytokines, chemokines – phagocytosis, release of inflammtory mediators, recruitment of leukocytes

*Inflammatory mediators*

Histamine, PGs, Cytokines/Chemokines, Complement, Bradykinin, Neuropeptides

*Outcomes* Abscess formation or Progression to Chronic apical inflammation

**Acute Apical Abscess**

*What is AAA?*

Inflammatory reaction to *direct bacterial invasion* of inflammed periapical tissues by specific *pyogenic* bacteria characterized by a *focal collection of purulent exudate surrounded by granulomatous layer*

*Cells Involved*

1. PMNs – Predominant cell in AAA – secretion of *lysozymal enzymes and free oxygen radicals* during phagocytosis of bacteria leads to connective tissue (ECM) destruction
2. Live/Dead Bacterial cells – *Pyogenic bacteria* resistant to phagocytosis

*Composition of Purulent exudate*

Dead/Live PMNs, Dead/Live bacteria/byproducts, Disintegrated ECM/CT, Lysozymal enzymes

*Inflammatory mediators*

Similar to SAP

**Asymptomatic Apical Periodontitis**

*What is AAP*?

*Adaptive immune response* characterized by persistence of inflammatory stimuli, adaptation of host immune response, *destruction of periapical tissues with formation of a granulomatous lesion* and initiation of repair process

*Cells Involved*

1. Macrophages – APCs, Activated for bacterial phagocytosis, ↑ cytokines
2. Lymphocytes – T lymphocytes – activation of Macrophages/NKCs, B cell differentiation; B lymphocytes – APCs, Antibody production
3. Dendritic Cells - APCs
4. Osteoclasts – Bone destruction – RANKL: IL-1, IL-6, TNF, PGs, BK
5. Fibroblasts – Chronic inflammation/wound healing – Collagen prod.

*Inflammatory Mediators*

Similar to SAP with different composition of adaptive cytokines present

*Key features*

Proliferation of *Fibrovascular Granulomatous lesion*, *Bone destruction*, External apical root resorption (cementum/dentin) – see *Filippe, Vier*

**Radicular Cyst**

*What is a radicular cyst?*

Pathologic cavity completely lined by *non-keratinized stratitifed squamous epithelium* of variable thickness and contained within a granulomatous AP lesion

*Types of cysts*

Pocket or Bay cyst – Attached to the AF of root, lumen opens to canal

True cyst – *NO attachment to the root structure*, completely enclosed by lining epithleium

*Cells Involved*

Same a AAP with Epithelial Cells most prominent cell type

*Inflammatory Mediators*

Similar to AAP

*Key Features*

Asymptomatic Cyst formation within Fibrovascular granulomatous lesion

**Extraradicular Infections**

*What is an extraradicular infection?*

Bacterial infection established outside the confines of the root canal space within the periapical tissues

*Types of E.I.*

1. Continuous with an intraradicular infection
2. Independent of an intraradicular infection

*Forms*

1. Biofilm
2. Planktonic

*Occurrence*

Rare: Usually associated with actinomycosis/proprionib. (*Nair, Siqueira*, *Sjogren*)

*Ricucci/Siquiera* – 6% Extraradicular bioflim presence in AP cases, *typically planktonic bacteria associated with abscess*

More common: See *Tronstad/Barnett, Sunde/Tronstad (35/36 AAP/CAA Sx)*

NOTE: Testing can only be done through surgical biopsy or microbiologic sampling during apical surgery which may be subject to contamination

**Periapical Pathology - Diagnosis**

**Granuloma** = *Localized mass of chronic inflammatory* tissue consisting of *macrophages, T lymphocytes, and a variable number of plasma cells, PMNs, mast cells, giant cells and epithelium* formed in reaction to infection of the dental pulp which serves as a constant source of antigenic material

**Abscess** = *Focal collection of purulent exudate* composed of dead and live PMNs, disintegrated cells, degraded ECM, lysozymal enzymes, dead and live bacteria and toxins *surrounded by a layer of viable PMNs and granulomatous tissue*

**Cyst** = *Pathologic cavity completely lined by non-keratinized stratiifed squamous epithelium* of variable thickness and *contained within a granulomatous AP lesion*

**Periapical Pathology - Diagnosis**

1. *Simon* – Granuloma 54.3%, Abscess 5.7%, Epithelialized Granuloma 22.9%, True Cyst 8.6%, Bay Cyst 8.6%
2. ***Nair*** – Incidence of Cyst, Abscess, and Granuloma:

Granuloma 50%, Abscess 35%, Cyst 15%, (True 9%, Pocket 6%) – need for

Serial biopsy to establish cyst vs. epithlialized granuloma

1. ***Spattafore 1990***– 1659 (~1700) *apical biopsies*: Granulomas 52%, Cyst 42%, Apical Scar 2%, Other 4% (rule of 2’s)
2. ***Koivisto* *2012***– 9723 (~9700) *jaw lesions* (excludes: angle/ramus mandible): Granulomas 40%, Cysts 33%, Others 20%: KCOT 8%, CGCG 1%, Ameloblastoma 1%, Metastatic Carcinomas <1%

# What are the current theories of cyst formation?

**STTT = S**eltzer, **T**en Cate, **T**oller, **T**orabinejad

1. **Epithelial proliferation (*Seltzer*)** - Epithelial rest cells of Malassez *activated by inflamm. mediators* to proliferate and line cavity
2. **Cavitational Breakdown Theory (*Ten Cate / Cohen*)** - Continuous growth of epithelial cells *removes central cells from nutrition* – innermost cells die and cyst cavity forms
3. **Osmotic Expansion of Cyst Lining (*Toller*)** - Osmotic pressure buildup due to *semi-permiable membrane* (Starlings Law)
4. **Immunological Theory (*Torabinejad*)** - Continued immune reaction to antigens – bacteria in infected root canal system. *Immune complex reaction (Type III) responsible for proliferation of epithelium.*

**Cyst Formation**

1. ***Lin/Huang/Rosenberg 2007*** – Proliferation of epithelial cell rests, formation of apical cysts, and regression of apical cysts after PA wound healing
2. ***Ten Cate* *1965***: Epithelial rest cells of Malassez remain dormant until activated to proliferate by inflammatory mediators
3. *Shear 1963*: Proliferating epithelial cells rely on surrounding CT for nutrients, innermost cells necrose, forming epithelial lined cystic cavity

**Do Cysts resolve following NSRCT?**

**Only Pocket Cysts:**

1. ***Nair* *1998***– Pocket Cysts heal, True Cysts do NOT heal due to self-sustaining nature
2. *Simon 1980* – Pocket Cysts may heal due to the continuity with the root canal space, True Cysts would not heal due to the independence from the root canal space and their self-sustaining nature

**All Cysts:**

1. ***Lin 2009*** – Apical true cysts may heal by *apoptosis mechanism* similar to pocket cyst when removal of inflammatory microbial etiology is satisfactory during NSRCT; *All cysts are inflammatory in origin*.
2. *Caliskan 2004* – Healing of large cyst-like lesions (7-18 mm diameter); + cholesterol crystals periapically; CaOH2 used- 74% complete/10% incomplete healing; **Size of lesion is not major determining factor in NSRCT vs. EMS**

# Pulpal & Periapical Microbiology

# Cause of Apical Periodontitis

Microorganisms colonizing the root canal system play an essential role in the pathogenesis of periradicular lesions:

*Miller 1894* – Light microscopy, detected bacteria species in teeth with AP

***Kakehashi 1965*** – Germ free vs conventional rats – germ free rats: vital/hard tissue repair; conventional rats: pulp necrosis/AP - directly linked AP to bacteria

***Sundqvist 1976***– Human study, evaluated necrotic traumatized teeth – w/o AP – no bacteria; w/ AP – Bacteria present (90% Anaerobes)

***Moller/Fabricius* *1981***– Monkey study, Devitalized pulps/sealed 6-7 months: Sterile necrotic pulps – no AP changes; Infected necrotic pulps – AP inflammation/destruction

***Ricucci/Siqueira*** – AP is an intraradicular biofilm disease – CAGE %: 95/83/71/6

# Bacteria involved in initial necrotic case – Mixed anaerobes

**PPTTDF (“Two Ps, Two Ts, a D and a F”)**:

Gram -: Gram +:

**P**revotella (intermedia) Lactobacillus

**P**orphyromonas (endodontalis/gingivalis) Streptococcus

**T**reponema (denticola) Peptostreptococci

**T**annerella (forsythia) Proprionibacterium (proprionicum)

**D**ialister (invisus) Actinomyces (israeli)

**F**usobacterium (nucleatum) Eubacterium

\***Mixed**, **polymicrobial 3-20 species**, symbiotic relationship

*Siqueira/Rocas 2006* – 55% of bacterial species yet to be cultivated

– polymicrobial, anaerobic community – initial invaders and late-comers

Bacteria involved in initial necrotic case – Mixed anaerobes

*Rocas/Siqueira 2008* – Bacterial species: AP: <5mm: 12.0, 5-10 mm: 16, >10 mm: 20, Sinus tracts: 17; Larger the PARL = Greater Bacterial Diversity

*Sundqvist* *1975*– Redirected understanding of canal flora – **predominantly obligate anaerobic (91.4%)** but mixed with facultative anaerobes

***Baumgartner/Falkler 1991*** – Carious pulp exp/AP teeth, extracted, cultured - **Apical 5 mm**, **predominantly Obligate anaerobes (68%)**. Most prevalent species: P. intermedia/nigrescens (BPB), Peptostreptococcus, Veilonella

***Fabricius 1982*** – Monkey study, **obligate anaerobes ↑ w/ time and apical position within the canal**; Early Pulpal Infection– More Facultative Bacteria

*Siqueira* – 400+ species poss. – 45% Molecular (qPCR), 32% Culture, 23% Both

Bacteria involved in previously treated cases – Gram +, facultative anaerobes – treatment resistant

E. faecalis:

*Rocas 2004* – E. faecalis 9x more likely in Persistent than Primary Infections

*Sedgley 2006* – E. faecalis 90% Persistent Infections (vs. 67% Primary) - qPCR

*Sundqvist* *1998*– 1st to find E. faecalis in persistent endo failures, frequently as a single species microorganism (9/24 cases); Retreatment success rate ~74%

*Moller* – high incidence of Enterococcus faecalis (Gr+, facultative) – few or mono species infection

*Haapasalo* – unsealed cases during treatment or multiple appts reveal higher frequency of E. Faecalis

*Zolleti/Siqueira* 2006 – E. faecalis present in Retx cases with and without AP – possibly may be present

Fungi and AP:

Candida albicans – most prevalent fungi involved in AP

*Siqueira 2004* – Review on Fungi and AP:

1. C. albicans most commonly identified fungi in primary/persistent AP
2. More commonly isolated from Persistent infections
3. Multiple virulence factors
4. Resistant to CaOH2 (*Waltimo/Orstavik/Haapasalo*)

*Nair* – found yeast-like microorganisms, therapy resistant

*Waltimo/Orstavik/Haapasalo 1999* – Candida (resistant to many medicaments), resistant to CaOH2, dentinal tubule infection

*Sen* – Candida – Most common fungi in Persistent Infections

Bacterial species associated with Refractory cases

PACES:

Pseudomonas aeurginosa

Pseudorambacter alactolyticus

Proprionibacterium Proprionicum

Actinomyces

Candida Albicans (yeast)

Enterococcus faecalis

Streptococcus

Prevotella, Fusobacterium, Lactobacilli – Persitent infection (@ obturation)

*Siqueira/Rocas 2009*

Persistent infections: 1-5 species, Gram + facultative anaerobes (*adequate RCT*), 2-30 species, Gram + facultative anaerobes (*inadequate RCT*)

Persistent/Secondary Intraradicular Infections

Secondary infection= Microorganisms not present in the primary infection but introduced in the root canal at some time after intervention

Persistent infection= Microorgansims that were members of primary or secondary infections that resisted intracanal procedures and survived post-treatment

*Sjogren 2003* – Prospective Human study, PA healing (5 years post-op), At time of root filling (1 visit): Negative culture: 94%, Positive culture: 68%; *Bacterial presence at time of root filling ↓ success of NSRCT; 2 visit necessary ↓ infection*

*Fabricius 2006* – Monkey study (175 root canals), PA healing (2.5 years post-op), At time of root filling: Negative culture: 72%, Positive culture: 21%; *Bacterial presence at the time of root canal filling decreased periapical tissue healing*

*Lin* – Failed NSRCT teeth harbored intraradicular infection

*Siquiera/Rocas 2004* – PCR analysis of non-healed NSRCT demonstrated intraradicular infection

*Sakamoto/Siqueira/Rocas 2008* – Long term surviving bacteria = failure

Nutrient Source, Oxygen Tension, and Bacterial Gradients

1. Nutrients Source: Carbohydrates vs. Peptides/AAs
2. Saccharolytic: Digest carbohydrates
3. Asaccharolytic: Digest proteins, amino acids (degradation products)
4. Later pulpal infection (more apical), More Asaccharolytic dominant
5. Oxygen Tension: Redox Potential of Canal
6. Initial infection (Coronal): Facultative (↑ O2), Higher Oxygen Tension
7. Later infection (Apical): Obligate Anaerobe (↓ O2), Lower Oxygen Tension
8. Bacterial Gradient/Interactions
9. Early colonizers – Facultative anaerobes
10. Latecomers – Obligate anaerobes

*Fabricius/Moller 1982* – Monkey study: devitalize/infect pulp space/seal canals, Clinical, Radiographic, Histo examination: Initial pulp infection (day 7): Facultative bacteria predominate; Later pulp infection (day 90, 180, 1060): Obligate bacteria

*Sundqvist 1979* – Saccharolytic (initial invaders) vs. Assacharoltyic (late comers)

Virulence Factors

*Sakamoto/Siqueira/Rocas 2007*

1. LPS - Gram – outer membrane, Lipid A moeity responsible for virulence
2. LTA - Gram + outer membrane
3. Peptidoglycans - Outer sheets of bacterial cell wall (Gram +: 80-100, Gram -: 2-3)
4. Secretory Products (Proteolytic Enzymes) - Collagenases, Hyaluronidases, Chondroitin sulfatases
5. Metabolic Byproducts - Polyamines, Sulfur
6. Coaggregation/Biofilms

Biofilms vs Planktonic Bacteria

Biofilm = community of microorganisms embedded in exopolysaccharide matrix

Planktonic = free-floating single microbial cells

1. *Socransky* – Dental biofilms can be up to 300 or more cell layers thick
2. *Mah* – Antibiotic concentration to kill biofilm bacteria is 100-1000x greater than concentration need to kill planktonic bacteria
3. *Ricucci/Siqueira* 2010– 106 roots w/ AP (42 treated) *Intraradicular biofilms present:* *Cysts 95%, Abscesses 83%, Granulomas 70%; Extraradicular biofilms 6%* (CAGE)
   1. No correlation between biofilm presence and clinical symptoms or sinus tracts. *Larger lesions had greater % of biofilm presence*.
   2. Intraradicular biofilms are responsible for AP
   3. *Extraradicular infections* in form of biofilms are *not common,* typically *planktonic bacteria in form of abscess with PMNs*
4. *Tronstad/Barnett* *1990* – Extraradicular Biofilms in persistent apical lesions

Dentinal Tubule Infection

1. *Love* – E.faecalis may invade dentinal tubules and remain viable by adhering to Type I collagen in the presence of human serum (Pathogenicity for persistent AP)
2. *Pashley* – Dentinal tubule infection
3. *Peters/Wesselink/Walton* 1995– Review – 70-80% teeth w/ AP have dentinal tubule infection; Failure of RCT appears to be unrelated to bacteria left within tubules after proper RCT
4. ***Haapasalo/Orstavik 1987*** – E. Faecalis survived w/in tubules 10 days w/out nutrients
5. ***Sen*** – Bacteria penetrate **10-150 μm** into the tubules
6. *Siqueira* – Bacteria invade dentinal tubules up to 300 μm
7. *Nagoaka 1995* – Dentinal tubules: ↑ Bacterial invasion rate in Non-Vital teeth (No dentinal fluid/odontoblast processes/immune cells to slow infection)

Causes of E. faecalis Resistance

*Stuart* 2006 – Review of E. faecalis and mechanisms of resistance

1. *Love 2001* – E. faecalis invades dentinal tubules/remains viable by adhering to Type I unmineralized collagen in the presence of human serum
2. *Distel* – E. faecalis form biofilms
3. *Evans* – E. faecalis have a proton pump – pulls H+ ions into cell – lowers pH within cell – survive CaOH2 (high pH)
4. *Sundqvist 1998* – E. faecalis can survive as single infection without dependence of nutrients from other microorganisms
5. *Sedgley 2005* – E. faecalis can survive long periods of nutrient starvation in obturated canals (gutta percha/ZOE) – 12 months Ex Vivo
6. *Orstavik/Haapasalo* – Ca(OH)2 – does not kill E. faecalis in dentinal tubules

# Black Pigmented Bacteria (BPB)

Bacteroides species – formerly Bacteroides melaninogenicus – 1980’s/90’s

Two genera (PP):

Porphyromonas (i.e.: P. gingivalis, endodontalis): Asaccharolytic BPBs

Prevotella (i.e.: P. intermedia, nigrescens, oris): Saccharoltyic BPBs

*Sundqvist; VanWinklehoff* – BPB (B. endodontalis/gingivalis) and AAA

***Sundqvist*** – Saccharolytic Bacteria - Early Pulpal Infection (coronal); Asaccharolytic Bacteria – Late Pulpal Infection (apical)

# Is HIV found in the root canal or apical lesion?

*Glick* – 1st to find HIV within the dental pulp

***Torabinejad 1994 JOE*** – Found HIV in the periradicular lesion w/ PCR

*Trope 1991 OOO* – Found HIV in pulp tissue fibroblasts w/ DNA hybridization

***Sabeti* 2004 JOE** – Found *Herpes simplex, Epstein-Barr & Human Cytomegalovirus**in periapical lesions (HSV, EBV, CMV*). Large lesions showed higher levels.

# Are certain bacteria taxa associated with symptoms?

# YES

1. ***Sundqvist 1976*** – Classic study – Bacteroides melaninogenicus (BPBs) associated with acute symptoms (AAA)
2. ***Griffee/Newton* *1980***– Black Pigmented Bacteroides (BPBs) are associated with pain, sinus tract and odor
3. *Van Winkelhoff 1985* – AAA: >90% contained Bacteroides species
4. *Yoshida 1987* – P. Magnus, Bacteroides in acute symptomatic cases
5. ***Jacinto/Gomes 2003*** – Specific Gram - anaerobes isolated from symptomatic cases (perc +, spontaneous pain, etc.), including BPBs (Por, Prev)
6. *Gomes 2006* – Fusobacterium alocis, T. forsythia (gram – anaerobic rods) & T. denticola (gram – anaerobic spirochete) *associated* with acute symptoms

# Are certain bacteria taxa associated with symptoms?

**NO**

1. ***Baumgartner 1999*** – Culture/qPCR; 55% Root canal samples positive for BPBs, No significant relationship between specific BPB and signs/symptoms
2. *Siquiera/Rocas 2005* – qPCR; no relationship of BPB w/ symptoms
3. ***Siqueira/Rocas* *2009*** – No strong evidence of any single species causing a specific sign or symptom of AP; **more likely *virulence factors/bacterial density/host resistance interactions***
4. ***Siqueira/Rocas 2013*** – No single bacterial species can be implicated in pathogenesis of AP – limited to inferring Cause-and-Effect relationships; **Different types and loads of the** **bacterial community (density/virulence factors)** may be responsible for the specific pathophysiology of symp vs. asymp. cases. *Bacterial community concept*

**Are more bacterial species found in symptomatic teeth?**

**YES**

1. *Sundqvis*t *1975* - >6 species = pain, 5 or less = no pain (culture-dependent study, less specificity)
2. ***Siqueira/Rocas 2004*** – **AAA cases: 12-18 taxa, AAP cases: 7-12 taxa** (culture independent, qPCR study, detects VBNC taxa)
3. ***Sakamoto/Siqueira 2006*** – 16s rRNA PCR sequencing:

**Symptomatic cases (AAA): 18 taxa, Asymptomatic cases (AAP): 12 taxa**, AAA greater bacterial diversity

1. *Horiba* – Higher levels of LPS endotoxin in symptomatic cases (along with lesions and exudate)

**Are bacteria found in periapical lesions? Controversial**

**YES**

**Persistent AP (Surgical Biopsies)**:

1. *Saber/Simon 2012* – 7/13 persistent AP contained extraradicular infections upon surgical biopsy. All 7 were symptomatic.
2. ***Gatti/Socransky 2000*** – DNA/DNA hybrid., Persistent AP (refractory) cases; Attempted to discount sample contamination during sx biopsy
3. ***Tronstad/Sunde; Tronstad/Barnett***– Extrarad Biofilms, Persistent cases

**Sinus Tracts/CAA:**

1. *Haapasalo* - Microbial presence within Sinus tracts (CAA)
2. ***Weiger 1995* –** Culture study, Sinus tracts contain bacteria; **9/12 sinus tracts contain species also found in the canal**

**AAA:**

1. ***Oguntebi/Langeland; Siqueira* –** culture; PCR, AAA contain bacteria (commonly accepted)
2. ***Siqueira/Rocas 2013*** – Review of AAA, *mixed obligate anaerobic infection – bacterial community as a pathogen concept*

**Are bacteria found in periapical lesions? Controversial**

**NO**

1. *Walton* – Inflammation resists spread of bacteria, confined to root
2. ***Nair* – Bacteria confined to root, except:**
   1. **Abscesses**
   2. **Therapy resistant cases – actinomyces (israeli)**
   3. Infected cysts
3. *Holland* – Bacteria are present when pushed out during RCT
4. *Langeland*
5. ***Siqueira/Rocas*** *–* Only in **Abscesses & Actinomycosis cases**
6. ***Ricucci/Siqueira*** – Extraradicular biofilms 6%, typically *planktonic bacteria in form of abscess with PMNs*

*Sjogren* – isolated P. propionicum extraradicularly

*Waltimo* – no candida in AP and is resistant to Ca(OH)2

**Discuss bacterial flora in Acute Apical Abscess**

1. *Oguntebi/Langeland* *1982* - Abscesses aspirated/cultured – Mixed gram +/- facultative and obligate anaerobic flora; Most common: Fusobacterium nucleatum, Streptococcus mitis
2. ***Baumgartner 2004*** – Periradicular abscesses are **polymicrobial infections** with *organisms similar to those found in infected root canals*
3. ***Siqueira/Rocas 2006*** - # bacterial species: Acute Abscess (12-18) > Chronic AP (7-12); **Mixed flora and dominated by Anaerobic bacteria**
4. *Santos/Siqueira 2011* – Pyrosequencing, Most abundant phyla:
   1. *Symptomatic Infections*: Firmicutes (Dialister, Strep, Filifactor), Fusobacteria, Bacteroidetes (P, P, T)
   2. *Asymptomatic Infections*: Firmicutes, Bacteroidetes, Actinobacteria
5. ***Siqueira/Rocas 2013*** – **AAA: Mixed anaerobic infection**, Great Diversity

Gram -: BPBs (Prevotella, Porphyromonas), Fusobacterium nucleatum, Treponema, Tannerella forsythia, Dialister invisus

Gram +: Peptostreptococci (Parvimonas), Streptococcus anginosus, Actinomyces

**Bacteremia from RCT**

1. ***Baumgartner* *1976***– Bacteremias & NSRCT: evaluated vital/necrotic cases; instrumentation & obturation; overinstrumentation of necrotic cases
   1. **Total incidence:** **3.3% (1/30)** – **overinstrumentation of necrotic tooth**
   2. *No bacteremia if instrumentation/obturation confined w/in canal*
2. *Debilian/Tronstad 1995* - **~25%** even when instrument is confined to canal
3. *Pallasch* - Endo tx is the **least likely** dental procedure to produce bacteremia
4. *Baumgartner 1977* – Bacteremias & Endo Surgery:
   1. Flap reflection: 83%
   2. Periapical curretage: 33%
   3. **Simple Tooth Extraction: 100%**
   4. Transient nature of bacteremias (83%-33% during surgery)

**Periodontal vs. Endodontic Bacterial Profiles**

1. ***Trope/Tronstad/Rosenberg 1988*** – Darkfield microscopy, Endodontic vs. Periodontal abscesses: Coccoid cells: Endo > Perio, Spirochetes: Perio > Endo; **Spirochete (i.e.: Treponema) ranges: Perio abscesses 30-60%, Endo abscesses 0-10%**
2. ***Socransky 1998*** – **RED Complex (PTT) = P. gingivalis, Tanerella forsythia, and Treponema denticola,** Periodontal pathogenic complex (13,000 plaque samples)
3. *Socransky 2005* – Periodontal microbial etiology review

# Antibiotic Susceptibility

***Baumgartner/Xia*** – (**98 strains isolated from 12 endo abscesses – aspirational**)

1. Pen VK *1st choice* - 85% effective – NARROW Spectrum
2. Amoxicillin - 91% effective (broader spect./rapid absorp/longer ½ life)
3. Amox + Clavulanic acid (Augmentin) - 100% effective
4. Clindamycin - 96% effective
5. Metronidazole - 45%\*
6. Metro + Pen V - 93%, Metro + Amox - 99% (↑ 8%)

\*Metronidazole - Effective only on obligate anaerobic bacteria

# Pen VK – Effective on Facultative & Obligate Anaerobes

# Clindamycin – More effective on *Gram +* Facultative & Obligate Anaerobes

Note: Amoxicillin or Augmentin used in *Immunocompromised patients* due to *broader spectrum, rapid absorption, longer ½ life, and higher serum levels*

# Antibiotic effect on Oral Contraceptives

*Hersch* – only effected by Rifampin, but still advise patient to use alternate BC due to legal issues.

# Are bacteria present in traumatized teeth with intact crowns?

**YES**

1. ***Bergenholtz*** - found bacteria **64%** of the time - **mixed anaerobic infection, penetrated thru tubules or cracks**
2. ***Tronstad/Langeland*** – Bacteria gaining access to necrotic pulp via **enamel/dentin cracks (trauma)** establish infection within **2-3 weeks**
3. ***Love 1996*** – **In vitro, traumatized incisors** - Bacterial penetration of trauma induced **enamel/denin cracks** → pathway for development of infections in devitalized pulps of intact crowns subjected to trauma

Does Anachoresis occur?

Anachoresis = bacterial infection of traumatized pulp via bacterial invasion of blood vessels from adjacent PDL/sulcus

**YES**

1. *Robinson/Bolling* – 2 requirements: *pulpal inflammation & bacteria*
2. ***Gier/Mitchell 1968***– Bacteria are attracted to inflamed pulps. Traumatized teeth – bacteria invade *lymphatics/vascularture via periodontium* – infect pulp
3. ***Tziafas 1989*** – Dog study; Pulp exposures/CaOH2/Induced bacteremia (α-streptococci) – 24/27 teeth infected, *↑ Inflamm. Zone,↑ Bacterial infection*; Non-inflammed pulp-no infection; did NOT I.D. bacteria!

**NO**

1. ***Delivanis/Doyle*** – Could NOT demonstrate bacteria in unfilled root canals after repeated intravenous injections of bacteria

## Focal Infection – Does it occur today?

*Wahl* - defines focal infection as “a *localized or generalized infection* caused by *dissemination of microorganisms* or toxic products *from a focus of infection*”.

**“Focal infection” term coined by *WD Miller*** ***1891*** - found gangrenous pulps could act as centers of infection causing alveolar abscesses

*William Hunter 1900*- Attributed a multitude of diseases to “oral sepsis” → EXT

*Billings 1912* - Introduced “focal infection” theory to USA

##### No definitive evidence bacteremia causes systemic disease!

*Pallasch; Ehrmann* – Focal Infection theory does not exist, NSRCT least likely dental treatment to produce significant bacteremia (*Pallasch*)

***Siqueira*** – No evidence that organisms from NSRCT cause disease in remote sites

***Torabinejad*** – *Chronic AP lesions* can not cause systemic diseases *via immune complexes*

***Premedicate those susceptible to infective endocarditis***

**Methods of Bacterial Identification**

1. Culturing: Aerobic, Anaerobic, excludes VBNC species
2. Molecular Methods:
   1. Broad Range PCR
   2. Pyrosequencing
   3. DNA-DNA hybridization (eg: Checkerboard arrays, microarrays)
   4. Species specific PCR
   5. Nested PCR
   6. Mulitplex PCR
   7. Real time Quantitative PCR (qPCR)

**PAIN**

**Peripheral Sensitization**

Peripheral Sensitization = Increased excitability of pulpal nociceptors due to inflammation and pro-inflammatory mediators

**Direct Activators of Peripheral Nociceptors**:

Serotonin

Histamine

Bradykinin

TNF-α

**Sensitizers of Peripheral Nociceptors**:

Prostaglandins

Leukotrienes

NGF – nerve growth factor

SP - neuropeptide

IL-1β

**Peripheral Sensitization (Triad)**

1. **Hyperalgesia** = Increased/Prolonged response to noxious suprathreshold stimuli, aka after-firing
2. **Allodynia** = Decreased threshold for AP → response to non-noxious stimuli
3. **Spontaneous pain**

***Sessle 2005*** – Peripheral sensitization of afferent nocicptive endings is associated with a *decreased threshold for generation of AP (Allodynia), increased responsiveness to noxious stimuli (Hyperalgesia), and spontaneous activity*

***Hargreaves 1991*** – Peripheral sensitization (Hyperalgesia) is mediated by inflammatory mediators. Results in *Triad* of *Spontaneous APs, Decreased threshold for AP (Allodynia), and Prolonged response to noxious stimuli (Hyperalgesia)*

***Narhi*** – *Arterial pressure following heartbeat* (non-noxious stimuli) stimulates sensitized nociceptors to fire = *“throbbing” pain*

**Central Sensitization**

***Sessle 2005/2011, Hargreaves 1991***

1. **Central sensitization** - Afferent C fiber barrage secondary to peripheral sensitization/activation may lead to an **Increased Receptive field** and **Recruitment of long range fibers** in Medullary dorsal horn; ***Plasticity*** of central neurons – ***Sprouting and Unmasking* of 2nd order neurons**
2. **Trigeminal brain stem complex** - somatotopically arranged: main sensory nucleus, **spinal trigeminal nuclear tract** (subnuclei: oralis, interpolaris, *caudalis aka Medullary dorsal horn*)
3. **Interneurons (local circuit neurons)** - inhibitory (GABA) or excitatory
4. **Decending pathways** - Down regulation by opioid pathways (Enkephalins, GABA) in Periaqueductal Gray (PAG)/Nuclear Raphe Magnus (NRM) of brainstem

**Pain Pathways**

Detection, Processing, and Perception of Pain – Trigeminal nociceptive afferents

**Detection:**

1st order neuron - Peripheral pulpal tissue -> Trigeminal ganglion (1º cell body) -> Medullary dorsal horn (Subnucleus Caudalis) of spinal trigeminal nuclear tract (brainstem)

**Processing: (Trigeminothalamic tract)**

2nd order neuron - Medullary dorsal horn of spinal trigeminal nuclear tract (2º cell body) -> Thalamus (contralateral due to crossing midline while ascending)

**Perception: (Thalamocortical tract)**

3rd order neuron – Thalamus (3º cell body) -> Cerebral cortx/higher centers

**Pain Pathways**

1. *Holland/Michaelson* – Silent pulpitis leads to pulpal necrosis (40-60%)
2. *Jaber/Dionne* - μ opioid receptors present peripherally within the pulp
3. *Wadachi/Hargreaves 2006* – Direct activation of peripheral nociceptors – TRPV1 C fiber pulpal nociceptors express TLR4 and CD14 receptors (PRRs = Pattern Recognition Receptors) which react to LPS
4. *Narhi* – peripheral inflammatory cells contain endogenous opioids and somatostatin that can inhibit nociceptor AP generation
5. *Mudie/Holland* – peripheral lymphocytes contain endorphins

Opioids mimic Endogenous Opioids – act centrally at level of spinal trigeminal nuclear tract and peripherally

**Referred Pain**

***Sessle 2005*** – **Convergence theory**: *Superficial and Deep* 1st order neurons synapse with the same WDR and NS 2nd order neuron cell bodies within the medullary dorsal horn of the spinal trigeminal nuclear tract. Afferent barrage leads to **increased receptive field/activation of long range neurons**/**central sensitization** (*central neuroplasticity – sprouting and umasking of 2nd order neurons*) and **inability of higher levels to determine pain specific location**

***Falace 1996*** – Referred pain:

1. **Pain severity is the most reliable predictor for referred pain**
2. ↑ intensity of nociceptive barrage, ↑ Referred pain
3. 89.9% of patients w/ severe pain have referred pain
4. Most common site = **Adjacent tooth (80%)**

C fiber stimuation:

See *Bender 2000* and *Van Hassel 1969*

**Deafferentation & Chronic Pain**

***Nixdorf* *2010***– Meta-analysis of Frequency of **Persistent tooth pain post Endodontic procedures***,* ie: Pulpectomy, NSRCT, Retx, Endodontic microsurgery, (6 months or >): **5.3% (>7% in well-controlled studies)**. **(**Pain = spontaneous pain or percussion, palpation, or bite induced pain)

***Holland*** – Deafferentationand chronic pain – Possible ***neuroma formation*** of periapical nerve fibers due to inflammation of periapical tissues at time of re-organization/healing following RCT

***Sessle/Hu*** – Cat study, post RCT pain - Deafferenation (pulpotomy) → ↑ Receptive Field + ↑ Spontaneous Firing = **Central *neuroplastic* changes & Chronic Pain. (**Neuroplastic changes = unmasking and nerve sprouting)

***Nixdorf 2010*** – Meta-analysis of Frequencey of **Persistent (> 6months) dento-alvoelar pain of non-odontogenic origin post Endodontic treatment**: **3.4%**

# Non-odontogenic pain *(Subramanian; Nixdorf – 3.4%*)

# Myofascial Pain Disorder

* 1. Pain of muscular origin, Triggered by contraction of masticatory muscle
  2. Trigger point – small foci of hyperexcitable muscle
  3. Pain – **Dull, aching**, diffuse, constant
  4. Muscles of mastication (**TMLA**)
     1. Temporalis – Max Teeth
     2. Masseter – Max/Mand Posterior Teeth, TMJ
     3. Lateral Pterygoid – TMJ
     4. Anterior Digastric – Mandibular Incisors
  5. Testing – Group of teeth typically positive to percussion/palpation; *Palpation of involved muscle group reproduces “toothache” like symptoms*
  6. LA injection – directly into affected muscle for relief, **local tooth block will not relieve pain**

# Non-odontogenic pain

# Neurovascular Pain – aka Headache Disorders

* 1. Pain – Severe, **Throbbing, Pulsatile**; *Episodic* w/ complete remission between episodes
  2. Location – Temples, Ocular, Sinuses, Jaws, Teeth

**Migraine**

1. **4-72 hours**, **Unilateral, Pulsatile**
2. Nausea, vomiting, photo/phonophobia
3. Tx: Sumatriptan (Imitrex), Amitryptiline - TCA

**Tension type**

1. Most common, short lasting

**TACs (Trigeminal Autonomic Cephalgias)**

1. **Cluster headaches**, Paroxysmal hemicranias, short lasting neuralgiform headache, more common males
2. Severe Unilateral headaches *with ipsilateral autonomic symptoms*
3. **15 mins – 2 hours**, **5-8 x/day**, clustered active periods
4. *10 mins 100% O2 pain relief = Cluster headache*

# Non-odontogenic pain

# Neuropathic Pain Disorders – Trigeminal Neuralgia

* 1. Pain – Intense, intermittent, sharp, **stabbing/shooting**/**electrical**
  2. Location – *Unilateral along any of 3 branches of Trigeminal nerve*
  3. Trigger point – external (i.e.: touch, cold, shaving) or internal (i.e.: lips or teeth*); Response to stimulus is NOT proportional to intensity of stimulus*
  4. LA of trigger zone may result in relief of symptoms, which may confuse the diagnosis if trigger is internal
  5. **Episodic** – **up to 50 times per day, lasting 60 seconds to minutes**
  6. Initial symptoms mimic odontogenic pain
  7. Under 40 yrs, *MS is a common etiology for Trigeminal neuralgia* (Brain MRI)
  8. Refer to Oral Pain – Carbamazepine (Tegretol), Pregabalin (Lyrica)
  9. Etiology: Abnormality of neural structures (gasserian ganglion pressure from carotid artery branches)

# Non-odontogenic pain

# Neurogenic Pain Disorders – Neuromas, Neuritis

**Neuromas**

* 1. *Traumatic neuroma* – proliferative mass of disorganized neural tissue at the site of a traumatically or surgically transected nerve
  2. Symptoms develop 10 days post trauma
     1. Sharp **electric pain** when **touching area of injury**
     2. *Zone of anesthesia peripheral to area of neuroma*
  3. Location – mental foramen, lip, tongue, ext site or post RCT (deafferentation – *Holland; Hu/Sessle*)

**Neuritis**

1. Inflammation of nervesecondary to injury or infection of viral or bacterial etiology (i.e.: pain associated w/ herpes zoster)
2. *May lead to post-infection neuropathy of infected nerve*
3. Localized trauma – chemical, mechanical (i.e.: *endo sealers, irrigating solutions, intracanal medicaments, overextended gp, implant placement*)
4. Pain - constant**, burning; parethesia/dysesthesia/anesthesia**; allodynia

# Non-odontogenic pain

# Neuropathic Pain Disorders – Atypical Facial Pain

* 1. **Localized, sustained, non-episodic pain secondary to an injury** or change in neural structure
  2. Pain is **chronic, present daily**, and most part of the day
  3. Pain – “Deep”, Sharp, throbbing, aching; **Hyperalgesia/Allodynia**
  4. **Central sensitization** due to injury/peripheral barrage induces central neuroplastic changes
  5. *Surgical approaches are not effective –* do not desensitize the nerve
  6. Pain may migrate to different quadrants or ipsilateral side
  7. *LA will NOT relieve pain*
  8. No Trigger points, One tooth or cluster of teeth may be involved
  9. **Symptoms are preceeded by traumatic event, i.e.: RCT, Ext**
  10. Management: Referral to pain specialist/ENT, Rxs: TCAs, Neurontin (gabapentin), SSRIs, topical desensitizers, Pain management therapy, Physical therapy

# Non-odontogenic pain

**Psychogenic**

1. *Somatoform disorder* – mental disorder
2. Undiagnosed pain with no apparent etiology and poorly characterized symptoms (i.e.: burning mouth syndrome)

**Cardiac (*Kreiner*)**

1. Cardiac pain (ischemia-angina, MI) refers to **left arm, shoulder, neck, face**
2. Anginal pain may solely refer to the **Lower Left jaw**
3. *Endo testing will be normal*, LA will not relieve pain
4. Stimulation of C fibers

**Sinus (***Kretzschmar 2003*) **- Rhinosinusitis**

1. Sinus pain can exhibit **fullness or pressure below the eyes**
2. **Multiple maxillary teeth with sensitivity to percussion, palpation**
3. Sensitivity to palpation of structures overlying sinues; **throbbing or increased pain when head is placed lower than heart**
4. Systemic signs of sinus infection; Rx Amoxicillin, ZPac

# Salivary Gland Pathology – pain present at time of eating, sialography, CT/MRI

**Neoplastic**

1. Primary SCC or metastatic tumors (mand.) – Numbness (#1), jaw pain

**What is a NICO lesion?**

Neuralgia Inducing Cavitational Osteonecrosis – aka – “Ratner’s Bone Cyst”

*Bouqout*:

1. Diagnosed by exclusion, technetium scan or multiple radiographs
2. Histology – **Ischemic Osteonecrosis**
3. Symptoms *– mimics: Atypical Facial Pain or Trigeminal Neuralgia*
4. Radiographic findings – subtle findings
5. History – possible history of trauma, extraction or infection
6. Treatment – decorticate & curettage (high incidence of recurrence)

AAE Position statement:

1. Suspected NICO lesion should be referred to Orofacial Pain specialist for evaluation, diagnosis, and treatment
2. Extraction of NSRCT tooth with suspected NICO lesion is unethical

# Non-odontogenic pain

P – Psychogenic – Manchausens

I – Inflammatory – Sinusitis

N – Neurovascular – Migraines, Cluster headaches; Neurogenic – Neuroma, Neuritis

S – Systemic – Myocardial Infarction, Neoplastic

M – Musculoskeletal – Myofacial pain disorder

Differential Dx:

Myofacial pain disorder, Neurovascular, Neurogenic (Neuroma, Neuritis), Neuropathic (Neuralgia, Atypical Facial Pain), Psychogenic, Cardiac, Sinus, Salivary or Primary/Metastatic (mandibular - numbness/jaw pain)

**Cranial Anatomy**

1. Arterial Supply:

R atrium→R ventricle→Pulmonary artery→Lungs→Pulmonary vein→L atrium→L ventricle→Aorta→Common Carotid artery→External Carotid artery→Maxillary artery→

* 1. Maxillary Posterior teeth: Ptergyopalatine artery→PSA artery
  2. Maxillary Anterior teeth: Pterygopalatine artery→PSA artery→MSA artery
  3. Mandibular Posterior teeth: Mandibular artery→Inferior Alveolar artery
  4. Mandibular Anterior teeth: Mandibular artery→Inferior Alveolar artery→Incisive artery

1. Venous Drainage:
   1. Mandibular Ant/Post teeth: Inferior Alveolar vein→
   2. Maxillary Anterior teeth: Infraorbital vein→
   3. Maxillary Posterior teeth: directly into the Maxillary vein→

→Maxillary vein→Ptergyoid venous plexus→Retromandibular vein→Internal Jugular vein→Brachiocephalic vein→Superior Vena Cava→Heart (via R. Atrium)

**Cranial Anatomy**

Cranial Nerves Supply:

Brain stem→Trigeminal nerve (C.N. V), 3 branches of Trigeminal:

1. Ophthalmic - sensory only:
   1. forehead, upper eyelid, nasal mucosa, frontal sinus
2. Maxillary (foramen rotundum) – sensory only
   1. Lower eyelid, cheek, upper lip, maxillary teeth/gingiva, palate, maxillary/ethmoid/sphenoid sinuses
   2. PSA - Maxillary Molars
   3. MSA - Maxillary Premolars, MB root of Max Molar (see *Walton*)
   4. ASA - Maxillary Canine, Incisors
3. Mandibular (foramen ovale) – sensory and motor
   1. Sensory - lower lip, mandibular teeth/gingiva, chin, lower face, tongue (lingual n) via: Buccal, Auriculotemporal, IAN, Lingual nerves (BAIL)
   2. Motor - *muscles of mastication*, mylohyoid, ant. Digastric
   3. IAN - Mandibular Molars, Premolars
   4. Incisive Branch - Mandibular Canine, Incisors

**Flare-ups/Post-op Pain**

**What is a flare-up?**

Definitions:

1. *Reader* – Post-operative acute exacerbation of symptoms resulting in moderate to severe pain and/or swelling
2. *Walton* – Severe pain and/or swelling within a few hours to few days requiring unscheduled visit and active treatment
3. *AAE* – Acute exacerbation of asymptomaticpulpal or periapical pathosis after the initiation or continuation of NSRCT
4. *Tsesis* – Strong pain/swelling occurring within 48 hours

**What are the causes of flare-ups?**

1. ***Seltzer/Naidorf 1985***
   1. **Extrusion of irritants into apical tissues** (medicaments, irrigating solutions)
   2. Changes in **periapical tissue pressure** (aspiration of bacteria/tissue fluids)
   3. Specific Bacterial species (*Sundqvist* and BPB)
   4. **Pro-inflammatory mediators** and inflammatory processes associated with acute inflammatory reaction incited by instrumentation of the canal
2. ***Siqueira 2003***
   1. **Extrusion of infected debris**
   2. **Changing microbial flora of canal**
   3. **Changing Redox potential of canal (introducing Oxygen)**

**What is the incidence of flare-ups?**

1. ***Walton/Fouad 1992*** – 946 visits, Flare up **Overall incidence: 3.2%,** **Severe pre-op pain: 19%,** Localized/diffuse swelling: 15%, AAA: 13%, Necrotic pulp: 6.2%, SAP: 5%, Vital pulp: 1.3%
2. ***Trope*** – **1.8% overall**, **13.6% Retx/AP/single visit**
3. ***Tsesis*** – Meta-Analysis - **8.4% overall**
4. ***Eleazer/Eleazer*** – **3% one visit, 8% two visit**

**What is the incidence of flare-ups? Continued**

1. *Torabinejad 1994* – Factors associated with endodontic interappointment emergencies of teeth with necrotic pulps
   1. Age
   2. Sex of patient
   3. Presence of preoperative pain
   4. Presence of allergies
   5. Absence of PA lesions
   6. Sinus tract
   7. Retreatment cases
   8. Those receiving prescribed analgesics

Factors that had no effect on the frequency of emergencies

* 1. Presence of systemic disease
  2. **Use of intracanal medications**
  3. Penetration of the foramen with small instruments during length determination

**Do prophylactic antibiotics decrease flare-ups? NO**

1. ***Walton/Chiapinelli 1993*** – **Pulp Necrosis/Chronic (Asymptomatic) Apical Periodontitis** – NSD between Prophylactic Penicillin (2 g at time of appt/ 1g 1 hr post op) and Placebo for post-treatment (Pulpectomy) incidence of *Flare-ups, Pain, Swelling, and Severity of pain* reported
2. ***Pickenpaugh/Reader 2001*** – **Pulp Necrosis/Asymptomatic Apical Periodontitis** – NSD between Prophylactic Amoxicillin (3 g 1 hr prior) and Placebo for incidence of *Flare up* post-op (Pulpectomy)
3. ***Fouad/Rivera/Walton 1996*** – **Localized** **AAA** – NSD between course of Pen VK 500 mg, Placebo, and No treatment following Pulpal debridement + I&D (if needed) + 600 mg Ibuprofen (pre-op and q6h post-op) for *Reduction of symptoms or Speed of recovery*. NO benefit from penicillin for treatment of localized acute apical abscess when local treatment measures are completed!

**Do prophylactic antibiotics decrease flare-ups? NO**

1. ***Nagle/Reader 2000*** – **Untreated Symptomatic Irreversible Pulpitis** – NSD between pre-treament course of Pen VK 500 mg (7 days) and Placebo for Reduction of *Pain, Percussion Pain, and Number of Analgesics* taken for the 7 days prior to treatment. Penicillin should NOT be prescribed to treat irreversible pulpitis as it does not reduce pain.
2. ***Henry/Reader 2001*** – **Pulp Necrosis/Symptomatic Apical Periodontitis** – NSD between course of Pen VK 500 mg (7 days) or Placebo for reduction of post-operative *Pain, Percussion Pain, and Number of Analgesics* taken. Post-op admin. of Penicillin does not reduce symptoms for symptomatic necrotic teeth with periapical radiolucencies.

**Do prophylactic antibiotics decrease flare-ups?** **YES**

1. ***Torabinejad 1994*** – Ibuprofen, ketoprofen, erythromycin base, *penicillin, and methyprednisolone plus penicillin* were more effective than placebo for reduction of flare-ups within the first 48 hours following pulpectomy
2. ***Morse 1987*** – 1 day of *high dose Pen VK* reduced flare-up incidence from *20% to 2%*

**What is the incidence of Interappt pain?**

1. ***Harrison/Baumgartner 1983*** – Incidence of Interappt pain (Asymptomatic pre-op): **No pain: 55.5%, Slight Pain: 28.8%, Moderate-Severe Pain: 15.7%**; *NSD between vital and necrotic teeth*
2. ***Georgopoulou 1986*** – Pain after chemomechanical debridement: **No pain: 57%, Mild pain: 21%, Moderate pain: 15%, Severe pain: 7%**
3. *Glennon/Ng IEJ 2004* – Prevalence of Post-preparation (necrotic w/AP) pain within 48 hours: 64.7%, Pain ↓ day 2 in 64%. Severe pain: 9.9% (day 1), 6.3% (day 2). Predictors: Pre-op pain, Pre-op swelling, Molars

# What is the incidence of Post-obturation pain?

1. ***Baumgartner/Svec 1983*** – **Risk factor for post obturation pain was extrusion of sealer or gutta-percha.**  No relationship with vitality, PARL, root # or level of obturation. **Pain rate w/in first 24 hrs. = 47.6% (14% severe)**
2. ***Ng IEJ 2004*** – NSRCT or Retx**, Prevalence: 40.2% (<12% Severe).** ***Predictors****: Females, Post-preparation pain, Post-preparation swelling, Single visit, Molars, < 3mm PARLs*
3. ***Figini* –** *Cochrane Review* **– More Post-op pain w/ Single visit**

# Would you leave a tooth open to drain?

**Yes**

1. ***August*** – Necrotic teeth left open to drain were filed and closed with minimal flare-ups.

**No**

1. ***Bence*** – Avoid leaving teeth open to prevent flare-ups when reclosing.
2. ***Simon*** – described *oral pulse granuloma* due to legumes.
3. *Weine* – When access is left open, a greater number of appointments were needed to complete treatment and more flare-ups occurred than when the tooth was kept sealed. “If you file, don’t close, if you close don’t file”

**Is routine trephination required?**

**No**

1. ***Moos*** – *Pulpectomy alone provided significantly better postoperative pain relief at 4 hours compared with pulpectomy /trephination*. At no time interval did the trephination group have less pain than the group without trephination.
2. ***Nist/Reader*** - *Trephination did NOT significantly decrease pain, percussion pain, swelling, or # of ibuprofen*. It was therefore determined not to be routinely recommended for symptomatic necrotic teeth with radiolucencies.
3. *Houck/Reader* – Short-term drainage upon access in symptomatic necrotic teeth with periapical radiolucencies did NOT reduce pain, percussion pain, swelling or the number of analgesic tablets taken compared to teeth that did not drain.

**Does reducing the occlusion decrease post-op pain?**

**YES**

1. ***Rosenberg 2009*** – LOE 1- Random assignment. 48 hour VAS. Occlusal reduction reduced postoperative pain in those patients whose teeth initially exhibited **pulp vitality, percussion sensitivity, preoperative pain and/or the absence of a periradicular radiolucency.** Post-instrumentation.

**NO**

1. ***Parirokh 2012*** – LOE 1 – Random assigment. 6 hrs – 6 days VAS. Sym. Irreversible Pulpitis patients with Percussion sensitivity. **Occlusal reduction did NOT significantly reduce pain levels in comparsion to placebo group**. Both groups had signficant ↓ pain post-instrumentation/CaOH2.
2. *Holland* – Preoperative pain did not influence the effectiveness of occlusal reduction. In fact occlusal reduction did not impact post operative pain.
3. *Walton* – Prophylactic occlusal reduction did not decrease post operative pain; relieve occlusion only as needed. Pre-op pain was related to post-op pain.

**Pain Management**

**Analgesic Strategy for post-op pain**

1. **Ibuprofen alone (pre-op admin/post-op pain)**
   1. ***Dionne 1983*** (NIH/VCU) – 3rd molar exts - Pre-op: *Ibu 800* mg sig. more effective than Aceto 600 mg for ↓ post-op pain; Post-op: *Ibu 400 mg* sig. more effective than Aceto 600 mg or Aceto 600 mg + Codeine 60 mg for ↓ post op pain
   2. ***Menke/Jackson 2000*** – **NSRCT** – Pre-op: *Ibu 600 mg* sig. more effective than Etodolac 400 mg or Placebo for ↓ post-op pain at 4 and 8 hours post NSCRT (37/42 1 visit)
2. **Ibuprofen + Acetominophen (post-op admin/post-op pain)**
   1. ***Brevik 1999*** – 3rd molar exts – Post-op: *Diclofenac (NSAID) 100 mg* + Aceto 1 g sig. more effective than Diclofenac 100 mg or Aceto 1 g or Aceto 1 g + Codeine 60 mg at ↓ post-op pain intesity
   2. ***Menhinick/Gutmann 2004*** – **NSRCT** – Post-op: *Ibu 600 mg* *+ Aceto 1000* *mg* sig. more effective than *Ibu 600 mg only* or placebo at ↓ post-op pain intensity from 0-8 hours post NSRCT (1 visit) w/ pre-op mod-severe pain

**Pain Management Regimens (Hargreaves)**

*Flexible strategies for pain management; See Oxford League Table*

**Max dosages: Ibuprofen: 3200 mg/day, Acetominophen: 3000 mg/day**

1. Aspirin-like drugs indicated (every 8 hours):
   1. Mild pain: 200-400 mg Ibuprofen
   2. Moderate pain: 600-800 mg Ibuprofen or 600 mg Ibuprofen + 1000 mg Acetominophen (*Menhinick*)
   3. Severe pain: 600 mg Ibuprofen + Acetominophen/Opiate (up to 10 mg oxycodone)
2. Aspirin-like drugs contra-indicated (every 8 hours):
   1. Mild pain: 650-1000 mg Acetominophen
   2. Moderate pain: 650-1000 mg Acetominophen + Opiate (up to 60 mg Codeine)
   3. Severe pain: 1000 mg Acetominophen + Opiate (up to 10 mg oxycodone)

**Pre-op Analgesics & Anesthesia**

**YES** (all LOE 1 studies)

1. ***Parirokh 2010*** -150 pts w/ Sym. Irreversible Pulpitis, IAN B Success: 600 mg Ibuprofen 78%, Indomethacin 62%, Placebo 32% - Preop (1 hr) *Ibu & Indo Signficantly ↑ Success of IAN B in Symptomatic IP patients*
2. ***Ianiro/Eleazer 2007*** - 40 pts w/ Sym. Irreversible Pulpitis, IAN B, Success: 600 mg Ibu/1000 mg Aceto 76%, 1000 mg Aceto 71%, Placebo 46%***.*** *NSD but trend towards more success with pre-op analgesic*

**NO** (all LOE 1 studies)

1. ***Simpson/Reader 2011*** – Combination of 1000 mg Acetominophen/ 800 mg Ibuprofen did NOT significantly improve success of IAN b in Symptomatic irreversible pulpitis cases; *Preop Ibu + Aceto did not Inc. success of IAN*
2. ***Aggarwal 2010*** – Sym. Irreversible Pulpitis, IAN B Success: 300 mg Ibu 27%, 10 mg Ketorolac 39%, Placebo 29% - NSD; *Preop (1 hr) Ibu or Ketorolac did NOT Inc. success of IAN B (pain during procedure*)
3. ***Oleson/Reader 2010*** – Sym. Irreversible Pulpitis, IAN B Success: 800 mg Ibuprofen 41%, Placebo 35% - NSD; *Preop Ibu did not Inc. success of IAN*

**Contraindications to ASA/NSAIDs**

1. Pregnancy (3rd Trimester only)
2. Asthmatics – blocks PGE2 → ↑ Leukotrienes → bronchoconstriction
3. Sickle Cell Anemia – vasoclusive crisis (acidosis)
4. Peptic Ulcer Disease – gi protection (blocking cox1), bleeding risk
5. Crohn’s Disease, Ulcerative Colitis – gi protection (blocking cox1), bleeding
6. Long-term Steroid use (gut mucosa – potential ulceration)
7. Uncontrolled Hyperthyroidism
8. Congestive Heart Failure
9. Liver Disease – bleeding risk/metabolism of drugs reduced
10. Chronic Kidney Disease/End Stage Renal Disease (GFR < 15 mL/min)
11. Previous MI (*Olson*) – NSAIDs only, Naproxen ok if limted to 7 days
12. Previous Stroke – Avoid NSAIDs only
13. Bleeding Disorders – Thrombocytopenia, Hemophilia, von Willebrand’s
14. Meds: Anti-platelet (Aspirin, Plavix), Anti-coagulants (Coumadin, Heparin), Valproic acid (Epilepsy), Lithium (bipolar), Sulfonylureas (diabetes), Methotrexate (cancer/AI)

\*Limit NSAIDs (< 2 wks) with Anti-HTN meds (Beta blockers, ACEI, Diuretics)

**Steroids**

***Marshall 2002 / Endodontic Topics***

Effect of glucocorticoids on Acute inflammation:

* Blocks release of membrane phospholipids (ie: **Arachadonic Acid**)
* Inhibit acute abscess metabolites by inhibition of phosopholipase A2
* Decrease transcription of **cytokines** IL-1,2,3,4,5,6,11,12,TNFα.
* Decrease iNOS
* Decrease **COX2** transcription by monocytes /macrophages
* Decrease **neurogenic inflammation** by inhibiting tachykinins
* Decrease **bradykinin** due to increase ACE synthesis

Widespread effects on many organ systems are typically seen only at supraphysiological doses given over a long-term period, usually more than 2 wks.

**Steroids**

***Marshall 2002* / Endodontic Topics**

1. Intraoral IM injection or an **intraosseous injection** is preferable over and extraoral IM injection. Intraoral injection of steroid is preferable as no assumption about patient compliance is required. A dose of **6-8mg of dexamethasone or 40mg of methylprednisone** appears from the literature to be appropriate.
2. If an **oral route** is chosen **48mg methyprednisolone/day for 3 days** and by extrapolation **10-12mg dexamethasone/day for 3 days** should provide significant **post treatment pain relief**

**Intracanal Steroids**

1. ***Chance/Lin 1987*** – Compared Intracanal corticosteroid (2.5% Meticortelone) vs. Saline for intracanal medicament. At 24 hrs, *Vital cases: Cortcosteroid sig. reduced post op pain,* Necrotic cases: NSD
2. ***Moskow/Morse/Krasner 1984*** – Corticosteroids (dexamethasone) vs. Placebo. At **24 hrs**: *Corticosteroid sig. reduced post-op pain compared to placebo. Only Vital teeth* were used for the study.
3. *Pierce/Lindskog 1987* – Ledermix (tetracycline/corticosteroid mix) is recommended as an intracanal medication to inhibit external inflammatory root resorption in traumatized teeth.

**Ledermix**

**What is Ledermix?** Corticosteroid antibiotic paste:

1. Triamcinolone Acetonide (1%) - **Corticosteroid**
2. Demeclocycline (3%) - **Antibiotic**
3. Water soluble cream: Triethanolamine, Calcium Chloride, Zinc Oxide, Sodium Sulphite Anhydrous, Polyethylene glycol
4. ***Ehrmann*** – LOE 1, 223 pts, significantly *less post-treatment pain* in patients after intracanal administration of *Ledermix compared with either CaOH2* or no intracanal dressing (*opposes Torabinejad, Walton*)
5. ***Bryson/Trope 2002*** – Dog study, premolars hemi-sected and extracted, 60 min dry time, replanted following instrumentation + 1) CaOH2 or 2) Ledermix. 4 month histological eval. *More favorable healing and less replacement resorption with Ledermix.* Possible anti-resorptive effects of tetracycline derivative + anti-inflammatory effects of steroid.

**Systemic Steroids:**

1. *Marshall/Walton 1984* –*IM Dexamethasone (4 mg)* reduced severity of pain at 4hrs & 24 hrs compared to placebo. ↑ Pre-op pain = ↑ Post-op pain.
2. ***Krasner/Jackson* *1986*** – *Oral Dexamethasone (0.75 mg*) significantly reduced post-NSRCT pain at **8 & 24 hrs** compared to placebo
3. *Glassman/Krasner 1989* – *Oral Dexamethasone (12 mg q4h)* significantly reduced post-NSRCT pain at 8 hrs but no difference at 24 & 48 hrs

**Injection Techniques for Steroids**

Intraosseous:

1. ***Gallatin/Reader 2000*** – Single dose of Intraosseous steroid – **methylprednisolone** (Depo-Medrol 40 mg*)* *sig. reduced pain, percussion pain, and number of analgesics* vs. placebo in patient with Symptomatic Irreversible Pulpitis. *Depo-Medrol can be used to temporarily alleveliate symptoms of irreversible pulpitis until definitive tx*.
2. *Isset/Reader 2003* – Compared Intraosseous DepoMedrol vs. Placebo for reduction of PGE2 and IL-8 in pts with Sym. Irreversible Pulpitis. *At day 1, Intraosseous DepoMedrol significantly reduced pulpal levels of PGE2.*

PDL:

1. *Kaufman 1994* – *Intraligamentary injection of methylprednisolone (Depo-Medrol)* reduced the frequency and intensity of post-operative pain in comparison to placebo and Mepivicaine plain

**Anxiolytic therapy - Benzodiazepines**

1. ***Hargraves & Dionne 1993 OOOO*** – **Triazolam (Halcion) 0.25mg** appears to be *safe, effective alternative to parenternal sedation* with a benzodiazepine for dental outpatients.
2. ***Hutter & Dionne 1997 JOE*** – Oral **Triazolam (Halcion) 0.25 mg** is safe and *more effective anxiolytic agent than diazepam (5.0 mg)* for endodontic patients.
3. *Dionne OOO 1997* – ***Sublingual Triazolam*** *results in greater anxiolytic activity and less pain perception than oral administration* as a result of greater plasma drug levels and may be useful as an alternative for nonprenternal outpatient sedation.

**Anxiolytic therapy – Nitrous Oxide**

1. ***Stanley/Reader*** – N2O significantly improved success of local anesthesia in *Symptomatic Irreversible patients:* w/**N2O: 50%, w/o N2O: 28%** (Sig. Difference)

**Post op pain control (pending NSAID contraindications)**

* Pre-op Ibuprofen 600 mg (*Menke/Jackson*)
* Post-op Ibuprofen 600 mg + 1000 mg Tylenol (*Menhinick/Gutman*)
* Flexible Management pain strategy (*Hargreaves*)
* Post-op Occlusal reduction: Vital, no AP, Perc +, Preop pain (*Rosenberg*)
* Post-op Steroids: Dexamethasone or Methylprednisolone (*Krasner*)

NOT Antibiotics, NOT Benedryl

**Should antihistamines be prescribed to reduce pain ?**

**NO**

*Nevins 1994 JOE* – Prophylactic use of Benedryl plays little or no role in abating post-operative pain after instrumentation of necrotic teeth.

Benedryl (*Diphenhydramine*): 1st generation Anti-Histamine with Anti-cholinergic (parasympathetic acetylcholine), Anti-emetic, and Sedative properties

**Anesthesia**

**Mechanisms of Action**

*L.A. alters the resting potential and ↑ excitation (A.P.) threshold* of the nerve by binding to sodium channels and preventing sodium influx

pKa of anesthetic determines amount of acid and base forms of anesthetic present:

**↓ pKa = ↑ Base form present extraneuronally = Faster Onset of L.A.**

*Base form of anesthetic crosses neuronal membrane* and converts to acid form

*Acid form (NH+) of anesthetic binds to sodium channels* to prevent AP transmission

The effect of properties of anesthetic on anesthesia efficacy

**Duration**: Protein binding affinity, Vasoconstrictor (decreases blood flow/absorption)

**Onset**: pKa (see above)

**Potency/Efficacy**: Lipid Solubility (membrane penetration), Tissue pH (↓ pH = ↑ acid form = ↓ efficacy)

**Determining Successful Anesthesia**

1. *Dreven/Reader 1987* – EPT as an indictor of pulpal anesthesia. Symptomatic IP patients with 80/80 may still be symptomatic on access.
2. *Certosimo/Archer 1996* – EPT as an indicator of pulpal anesthesia in normal pulps. Less than 80/80, patients symptomatic during treatment.
3. ***Cohen/Cha/Spangberg 1993*** - Lip anesthesia not reliable indicator of pulpal anesthesia. (Aβ fibers NOT Aδ). DDM (Endo Ice) reliable method of testing for pulpal anesthesia in Irreversible Pulpitis patients – **92% effective**

# Anesthetic Failures – Current Etiologies

#### Hargraves – Endodontic Topics Vol.2; Reader

1. Anatomic variations/Accessory innervation
2. Acute tachyphylaxis – reduced responsiveness due to repeated injection
3. Effect of Inflammation on local tissues (pH) - ↓ pH = less base form
4. Effect of Inflammation on blood flow – vasodilation/inc vasc. perm.
5. Effect of Inflammation on peripheral nociceptors - Peripheral sensitization
   1. *Gold; Wells* – 2-3x Inc in Nav1.8, 1.9 channels – TTX resistant – resistant to Lidocaine (*Roy*) – Neuronal plasticity
   2. *Fouad* – 6x Inc in TTX resistant Na channels in SIP cases
   3. *Byers* – Neuropeptides (SP/CGRP) → fibroblasts → NGF → Nerve sprouting = ↑ Nociceptor field
6. Effect of Inflammation on central nociceptors – Central sensitization -neuroplasticity(sprouting/unmasking) following afferent C fiber barrage
7. Psychological/Anxiety factors

**Reasons for local anesthetic failures**

1. Anatomic variations/accessory inervation
2. Acute Tachyphylaxis – reduced responsiveness due to repeated injections
3. Effect of inflammation on pH – ↓ pH, less base form to cross barrier
4. Effect of inflammation on blood flow – vasodilation/increased vascular permeability – carries away LA faster
5. Effect of inflammation on peripheral nociceptors – Nerve sprouting (*Byers*) and ↑ TTX-resistant Na channels (2-3x - 6x↑ Na channels) (*Gold, Wells, Fouad*) which are resistant to Lidocaine (*Roy*)
6. Effect of inflammation on Central sensitization – exaggerated CNS response to even gentle peripheral stimuli due to central neuroplasticity
7. Psychological factors – anxiety reduces pain threshold

**Approaches for managing failures:**

1. Reader – *Supplemental LA*: B infiltration, Intraosseous, PDL, Intrapulpal
2. Hargreaves – *Adjunctive drugs: NSAIDs* (reduced PGE2 decreases nociceptor sensitization and decreases TTX-R Na channel activity)

**How do you manage a Local Anesthetic Overdose?**

*Finder & Moore 2002 DCNA*

1. LA Toxicity –
   1. Initial symptoms - Tremors, *muscle twitching and convulsions*
   2. Later findings*– Respiratory depression*, lethargy and *loss of consciousness*.
   3. Final findings – *Cardiovascular depression and hypoxia* *secondary to respiratory depression* can rapidly produce serious outcomes including cardiovascular collapse, *brain damage and death.*
2. Vasoconstrictor Overdose –
   1. Initial signs - *Palpitations, increase heart rate and elevated BP*
   2. Anxiety, nervousness and fear are often found as well
   3. Severe overdose - *Arrythmia, stroke and MI are possible*

Prevention: Good technique, watch for drug interactions, avoid high doses, get good medical history

**How do you manage a Local Anesthetic Overdose? Continued**

1. Management from Little & Falace
   1. Protect patient during convulsive phase, consider IV Valium (Diazepam)
   2. Monitor and record vitals (BP, Pulse)
   3. Supportive therapy
      1. Supine position
      2. O2 10 L/min, Monitor O2 with Pulse Oximeter
      3. Maintain BP
      4. Treat Bradycardia w/ IV Atropine 0.4 mg
      5. EMS
   4. CPR if unconscious

*Haas 2002 DCNA* – Recommended Emergency drugs: O2, Epi Pen, Nitro, Injectable antihistamines (diphenhydramine or chlorpheniramine), albuterol, aspirin, oral carbohydrates, and corticosteroids.

**Methemoglobinemia**

*Wilburn-Goo & Lloyd 1999 JADA*

1. Caused by **metabolite of Prilocaine (MRD=4mg/lb) & Benzocaine – Met-hemoglobin** (selective affinity for O2)
2. Symptoms occur *1-3 hrs after treatment*
   1. **Cyanosis without respiratory distress** when met-Hgb reach 10-20%
   2. Vomiting and headache have been described
   3. **Dyspnea, seizures, coma and death** at levels higher than 20%
3. Patients at increased risk
   1. Heart disease
   2. **Anemia**
   3. G6PD deficiency
   4. Children < 2yo
   5. Elderly

**Allergic Reactions to local anesthetics**

Esters (benzocaine) – yes, **Amides** (Lido, Mepivicaine, Articaine) – **no**

1. Appearance –
   1. Urticaria - Hives
   2. Erythema/Edema – Redness/swelling
   3. Itching
   4. Angioedema & Respiratory depression (more severe reaction)
   5. Anaphylactic reaction
2. Sulfite antioxidant (EPI preservative) – allergic reaction – **Asthmatics**
   1. *Asthma-like signs* of **tachypnea, wheezing, bronchospasm**, dyspnea, tachycardia, dizziness, and weakness
   2. *Severe flushing, general urticaria,* ***angioedema*,** tingling, purities, rhinitis, conjunctivitis, dysphasia, nausea, and diarrhea
   3. No sulfite reaction in dental practice has ever been documented

**0.3-0.5 mL 1:1000 Epi Subcutaneous, 50-100 mg IV Diphenhydramine, O2**

**Local anesthetics**

**Maximum Dosages**:

1. Lidocaine: 4.4 mg/kg
2. Prilocaine: 4.4 mg/kg
3. Articaine: 7.0 mg/kg

*Finder/Moore* – Rule of 25 = 1 carp anesthetic for every 25 lbs of weight

**Normal Pulp vs. Irreversible Pulpitis**

**IAN B Success (1st Molar)**

Normal Pulp: 43-60% Success (*Vreedland/Reader, McLean/Reader*)

Irreversible Pulpitis: 25-33% Success (*Nusstein, Reisman/Reader, Aggarwal*)

**An Update on local anesthetics in dentistry**

*Hass 2002 J Can Dent Association*:

1. **Metabolism of Amide LA occurs in the liver**. *Reduced hepatic function* does not increase duration of anesthesia, but*predisposes the patient to toxic effects.* ***Use reduced dosages****!*
2. **Methemoglobinemia** is associated with **prilocaine and benzocaine**
3. **Articaine & Prilocaine (4%)** are associated with **increased paresthesia**
4. **Malignant hyperthermia** occurs with exposure to **inhalation anesthetics** (succinylcholine, volatile anesthetics), not local anesthetics
5. **Lidocaine** and **prilocaine** are pregnancy cat. **B**; others are C (Mep)
6. **7 mg/kg** is **max lido dose**. (4.4mg/kg is conservative)

**Trigeminal Nerve Injury associated with Local Anesthetics**

*Pogrel* - Mechanisms of neurosensory disturbance (NSD):

1. Mechanical injury – penetrating neural sheath with needle
2. Mechanical injury – intraneural hemorrhage, granulation tissue, scar formation
3. Neurotoxicity – axonal degeneration

*Hillerup*

1. **4% Septocaine** is most common anesthetic involved in NSDs (neurosensory disturbances) – 60/96 NSDs reported over 12 yr period in Denmark
2. **Mechanism of injury – Neurotoxicity of 4% Solution – Axonal degeneration.** Not mechanical due to under representation of other solutions.

**Contraindications to Epinephrine**

1. Untreated Congestive Heart Failure
2. Uncontrolled HTN (180/110 or >)
3. Severe Recalcitrant Arrythmias
4. Digoxin – Anti-Arrythmia/CHF drug
5. Unstable Angina
6. Recent MI (within 6 months)
7. Recent CABG (within 3 months)
8. Recent Stroke (within 6 months)
9. Uncontrolled Hyperthryoidism (Grave’s disease, Pituitary adenoma)
10. Pheochromocytoma – adrenal tumor ↑EPI/NorEPI
11. Sickle Cell Anemia (limit 2 carps in surgery) – vasoconstriction→Crisis
12. Severe Asthmatics – possible sulfite allergy – no reported incidents
13. Sulfite Allergy
14. Recent Cocaine or Methamphetamine use (within 48 hrs)
15. PRONJ – Post-radiation osteonecrosis of jaw; >6000 cGy radiation

**Limitations for Epinephrine (limit to 2 carps)**

1. Stable Angina, Non recalcitrant Arrythmias
2. Controlled HTN
3. Previous MI > 6 months
4. Previous Stroke > 6 months
5. Beta blockers (**non selective**) – Propranolol, Timolol, Nadolol, Cartelol, Sotalol, Penbutol – *unopposed alpha stimulation* (*HTN, bradycardia*)
6. Hepatic disease - ↓ Hepatic function = ↑ L.A. toxic effects -drugs/anesthetics
7. Tricyclic Antidepressant (TCA) – Amitryptiline, Doxepine
8. Monoamine Oxidase Inhibitor (MAOI) – Phenelzine, Tranylcypromine
9. Alpha adernergic blockers (non selective) – Chlorpromazine, Haloperidol, Clozapine
10. Anti-adrenergics – Guanethidine, Guanadrel
11. Levothyroxine (Synthroid)
12. Epilepsy/Seizures – Gabapentin (Neurontin), PreGabalin (Lyrica)
13. COMT Inhibitors (Parkinson’s) – Levodopa, Entacapone, Tolcapone

**Does accessory innervation affect anesthesia?**

***Frommer 1972 JADA*** – **mylohyoid nerve** occasionally innervates mandibular molars. **30%** of the population have separate canals for the mylohyoid nerve.

***Walton 1988 JADA*** – **5%** of maxillary 1st molars have innervation from both the **PSA and the MSA**.

**Topical Anesthesia**

***Nusstein/Beck 2003*** - Compared 20% topical benzocaine w/ no topical for *pain of needle insertion*: NSD for IAN B or Max. Post. Infilatration, **Maxillary Lat. Infiltration was significantly less painful with topical use**

***Martin/Ramsay 1994*** – 20% topical benzocaine, maxillary infiltrations – **psychological effect** of topical anesthetic responsible for success

**Anesthetic Techniques: Gow Gates (V3 Block)**

1. *Malamed OOO 1981* – Textbook pg 237 – Better success rates 95%, decreased positive aspirations 2%, fewer post injection problem however longer onset 5-10 min vs 3-5min IANB
   1. Technique: *anesthetized V3* – target – **lat side of condylar neck**
      1. Dry & apply topical for 1 minute
      2. 25 gauge needle (long)
      3. Insertion – mucous membrane on line from intertragic notch to corner of mouth, *distal to max 2nd molar at height of mesiolingual cusp max 2nd molar*.
      4. Slowly advance needle until bone is contacted (average depth 25mm) withdraw 1mm & aspirate (if positive it is usually the internal max artery, aim higher & repeat)
      5. Deposit 1.8cc over 60-90 seconds, may use up to 3ml
      6. Use rubber block 1-2 minutes for diffusion
      7. Return to upright and wait 5 minutes (due to diameter of nerve or greater distance to nerve trunk)

**Anesthetic Techniques: Akinosi (closed mouth)**

*Also known at the closed mouth mandibular block or Vazirani-Akinosi Block*

1. Malamed Textbook pg 242 – Indications for Akinosi technique are Trismus or inability to see landmarks for IANB (large tongue), lower aspiration rate (10%), successful for bifid alveolar nerve.
   1. Technique: *anesthetize same as IANB*, target – **medial lingual border of ramus** (**above IANB below Gow Gates**)
      1. Dry & apply topical for 1 minute
      2. 25 gauge needle (long)
      3. Insertion – *turn bevel of needle toward midline (deflects needle toward ramus*) soft tissue overlying medial border of ramus *directly adjacent to max. tuberosity at the height of the mucogingival junction adjacent to the max 3rd molar*.
      4. Advance needle 25mm (ave) from tuberosity, aspirate, deliver 1.8ml over 60 sec., wait 5 min. (motor nerve effect will reduce trismus)

**Anesthetic Techniques: Incisive**

1. *Nist/Reader JOE 1992* – Incisive block alone - **No pulpal effects**
2. Malamed Textbook Page 249 – “Pulpal, buccal soft tissue and bone anesthesia is readily obtained” with the Incisive nerve block. Lingual tissue is not anesthetized.
   1. Technique: no need to enter target mental foramen (traumatic)
      1. Dry & apply topical for 1 minute
      2. 25 gauge (short)
      3. Insertion – *orient bevel toward the bone*- have pt partially close, locate mental foramen (see x-ray), enter tissue at canine or 1st bi directing needle toward MF (approx 5-6mm), aspirate, deposit 0.6ml over 20 secs.
      4. *Maintain gentle finger pressure over site to increase volume of solution entering MF (intra or extra orally) for 2 minutes.*
      5. Wait 3-5 minutes to begin treatment.

**Compare different anesthetic studies**

**IAN B:**

1. ***Aggarwal 2012*** – 1 vs. 2 carps 2% Lidocaine w/1:200,000 epi – *Symp. Irreversible Pulpitis*, Success: **1 carp: 26%, 2 carps: 54% (Stat. Sig.)**
2. ***Fowler/Reader 2013*** – Retrospective. 1 vs. 2 carps 2% Lidocaine w/1:100,000 epi – *Symp. Irreversible Pulpitis*, Success: **1 carp: 28%, 2 carps: 39% (NSD)**
3. ***Vreeland/Reader***– Compared 1 carp 2% Lido w/1:100k epi vs. 2 carps 2% Lido w/1:200k epi vs. 1 carp 4% Lido w/1:100k epi, *Vital pulps*. **NSD in degree or duration of anesthesia**. **Molars** = **43-60% Success**
4. ***McLean/Reader JOE 1993*** – Compared 4% Prilocaine, 3% Mepivicaine and 2% Lidocaine w/ 1:100,000 epi. *Vital pulps*. **NSD in onset, success or failure between any of the 3 solutions used**. **Molars** = **43-57% Success**

**Compare different anesthetic studies**

**B Infiltration supplemental to IAN B:**

1. ***Aggarwal 2012*** – B/L Infiltration (2 carps) supplemental to IAN B – *Sym. Irreversible Pulpitis,* Success: No Inf.: **33%**, 2% Lido w/1:200,000 epi: **47%**, 4% Articaine w/1:200,000 epi: **67%**
2. ***Matthews/Reader* *2009* –** B infiltration supplemental to Failed IAN B – *Sym. Irreversible Pulpitis*, 4% Septocaine w/1:100k epi, **Success:** **58%**
3. *Haase 2008* –B infiltration supplemental to IAN B – *Normal Pulp*, Success: 4% Septocaine w/1:100k epi - 88%, 2% Lidocaine w/1:100k epi – 71%

**Maxillary Infiltration (normal pulp):**

1. ***Evans/Reader 2008***– 2% Lidocaine w/1:100k epi vs. 4% Articaine w/1:100k epi (1 carp) – B Infiltration Max Laterals/1st Molars: **Max Laterals: L: 62%, A: 88% (Sig. Difference), 1st Molars: L: 73%, A: 78% (NSD)**
2. ***Guglielmo/Reader 2011*** –2% Lidocaine w/1:100k epi – B inf (1.8 mL) + L inf (0.5 mL) vs. B inf (1.8 mL) only. **Success: NSD**. **Duration: B only – 21 mins, B+L – 57 mins**
3. ***Mikesell*** – 2 carps vs. 1 carp 2% lido w/ epi – **Inc duration lat, 1st pm, 1st m**

**Compare different anesthetic studies**

**Mandibular Infiltration:**

**Anterior:**

1. ***Nuzum/Reader 2010***- *Normal pulp* - 4% Articaine w/1:100k epi – B inf (1.8 mL) + L inf (1.8 mL) vs. B inf (1.8 mL) only. **Success: B+L – 98%, B only – 76%, Duration: B+L improved duration 4th-58th minute**

**Supplemental Buccal (Symptomatic Irreversible pulpitis)**

1. ***Aggarwal*** – IAN B only (2% Lido w/1:200 k epi): **33%**, vs. IAN B + *B/L infiltration (2% Lido w/1:200k epi)*: **47%**, vs. IAN B + *B/L infiltration**(4% Septocaine w/1:200 k epi)*: **67%**
2. ***Matthews/Reader 2009***– IAN B only (2% Lido w/1:100k epi): 33%, IAN B + *Supplemental B infiltration (4% Septo w/1:100k epi):* **58%**

**Gow Gates vs. Akinosi vs. IAN B:**

1. ***Goldberg/Reader*** – *No difference in anesthetic success* of IAN vs. Gow Gates vs. Akinosi. *Faster onset of pulpal anesthesia with IAN B*.

**Intraosseous Anesthesia**

Reader 1997, 1999 OOO; JADA 1999; JOE 199

1. ***Nusstein/Reader*** – Irreversible Pulpitis, IAN B only 19% success, **Intraosseous Stabident** (**2% Lidocaine w/1:100k epi**): **90% success** (Mandibular Molars), **88% Overall**
2. *Coggins/Reader 1996* – Stabident + 2% Lido w/1:100,000 epi – *Primary injection technique* - 75-93% success (Max/Mand teeth)
3. *Dunbar/Reader 1996* – Stabident+ 2% Lido w/1:100,000 epi – *Supplemental to IAN B* – 98% success
4. ***Reisman/Reader 1997*** – Irreversible Pulpitis (Mand. Posterior), IANB only 25% success. **Intraosseous injection** (**3% mepivacaine w/o epi**): **1st Injection**: **80%** **success, 2nd Injection 98% success**.

**Intraosseous Anesthesia**

Reader 1997, 1999 OOO; JADA 1999; JOE 1998 –

1. ***Replogle/Reader* *1999***– **Cardiovascular effects** of 2% Lidocaine w/1:100k epi vs. 3% Mepivicaine w/o epi: **2% Lido** - 67% of patients experienced **↑ HR (23-24 bpm) for ~ 4 mins**, **3% Mep** – **No ↑ HR**. No sig. difference in BP between the 2 groups. *Transient ↑ HR*.
2. *Nusstein/*Reader - Overall, the supplemental intraosseous inj was found to be **88%** successful in gaining pulpal anesthesia for endodontic therapy. In posterior teeth diagnosed with irreversible pulpitis, the supplemental intraosseous injection of 2% Lidocaine w/1:100k epi was successful when conventional therapies failed.
3. *Anderson 1998 JOE* – Stabident IOI was an effective supplemental anesthetic technique in 89%. More success in mandible than maxilla. (91% vs 67%)

**PDL injection**

*Kim 1986 JOE* – PDL is effective, painful, affects adjacent teeth, and doesn’t work via pressure. Pulpal blood flow is decreased when vasoconstrictor is used; don’t use for operative dentistry. Vasoconstriction is mechanism of action.

***Walton 1986 JOE*** – PDL is primarily intraosseous and required backpressure. Anesthetic spreads through cribiform plate. It is safe to the periodontium and pulp when used with operative procedures. *Can’t be used for differential diagnosis - anesthetizes adjacent teeth*. Widespread distribution. Supplemental only.

*Torabinejad & Peters et al OOO 1993* – PDL inj has no long-term deleterious effects on pulps of human premolars.

*Reader 1988 JOE* – 2% Lido w/1:100,000 epi is preferred for PDL and was more effective than anesthetic w/out epi. Average pulpal anesthesia = 20 min.

**Do additional measures increase success of IAN?**

Pre-op admin of Pain meds

1. ***Simpson/Reader*** – NO – 800 mg Ibu+1000 mg Aceto did NOT inc success of IAN B in Symptomatic Irreversible Pulpitis pts
2. ***Oleson/Reader*** *­*– NO – 800 mg Ibu alone did NOT inc success of IAN B in Symptomatic Irreversible Pulpitis pts
3. ***Parirokh*** – YES – *600 mg Ibu* or Indomethacin DID Inc success of IAN B in Symptomatic Irreversible Pulpitis pts (**78%**, 72%, **32%**)

Pre-op admin of Steroids

1. *Shahi JOE 2013* – YES – 0.5 mg Dextramethasone admin 1 hour prior to IAN B in *Asymptomatic Irreversible Pulpitis* pts Inc success of IAN B vs placebo

Pre-op admin of Benzodiazepines

1. *Khademi JOE 2012* – NO – 0.5 mg Alprazolam (Xanax) 1 hr prior did NOT inc success (53% vs. 40%) of IAN B in Sym Irrev. Pulpitis pts

Pre-op admin of N2O

1. ***Stanley/Reader*** – YES – Sym Irrev. Pulpitis pts admin 5 mins N2O prior to IAN B and procedure – Success of IAN B: w/ N2O: **50%**, w/o N2O: **28%**

**What about Oraverse?**

Oraverse = Phentolamine Mesylate

MOA: Reversible non-selective alpha adrenergic antagonist, Vasodilation due to α1 blockade

1. *Fowler/Reader* *2011* – Reversal of soft tissue anesthesia following admin of phentolamine in asymptomatic endodontic patients post IAN B and Maxillary Infiltrations. Statistically significant decreases in time for return of normal lip sensation: *Maxillary – 88 min decrease, Mandibular – 47 min decrease*.
2. *Elmore/Reader 2013* – Reversal of soft tissue anesthesia in normal patients following admin of phentolamine 30 min or 60 min post IAN block. Phentolamine significantly reduced duration of both pulpal and soft tissue anesthesia. *30 min post IAN admin was 24 mins faster (75 mins vs. 90-100 mins) for reversal effect than 60 mins post IAN admin*

**Resorption**

# Resorption

Internal

* Inflammatory
* Replacement

External

* Surface (transient)
* Inflammatory
  + Lateral (EIRR)
  + Sulcular (External Cervical or Subepithelial IRR)
    - Subepithelial external resorption
    - Invasive cervical resorption
    - Extracanal invasive resorption
    - Periodontal infection resorption
* Apical (AIRR due to AP/PARL)
* Pressure – “sterile” – ortho (*Mattison, Reitan*), tumors
* Replacement

# Classification of Resorption

*Trope 2002* – Root Resorption Classificaitons:

Progressive Inflammatory:

1. Non Infection related – PRESSURE – “sterile” inflammation, *ortho & tumors*, repair occurs after pressure removed
2. Infection related - PULPAL
   1. Apical external inflammatory - pulp necrosis/AP
   2. Lateral external inflammatory – trauma
      1. *Treat w/ CaOH2 long term (assess healing every 3 months)*
3. Infection related – SULCULAR
   1. External cervical aka Subepithelial Inflammatory
   2. Not related to pulpal environment

*Fuss* – classified root resorption according to stimulation factors:

1. Pulpal infection – Lateral/Apical
2. Periodontal infection - Sulcular
3. Orthodontic pressure resorption (OIRR)
4. Impacted tooth or tumor pressure resorption
5. Ankylotic resorption – *no bateria required* (Suda)

# Causes of Resorption – Theories

***Trope*** – **Two requirements for root resorption**:

1. *Loss or damage of the protective layer (pre-cementum or pre-dentin)*
2. *Inflammation must occur to the unprotected root surface*

**\*Osteoclasts will not adhere to/resorb unmineralized matrix (pre-dentin or pre-cementum layers) – lack of RGD amino acid sequence for osteoclast binding**

***Trope*** - *Cementum also inhibits the movement of toxins (TEBs) from root canal to periodontal tissues and visa versa* thereby inhibiting inflammatory response except where missing (lateral/accessory canals, apical foramen) or lost (scaling)

***Suda***– confirmed *correlation of bacteria and inflammatory resorption*, however determined that **ankylosis/replacement resorption can occur w/out bacterial infection present**. Germ free study

# Discuss Internal Root Resorption

***Wedenberg*/*Lindskog***:

1. Internal resorption is transient or progressive dependingon **bacterial contamination/inflammation to prolong activity of clastic cells**
2. Dentin contains a **resorption inhibitor, pre-dentin**
3. Internal resorption cannot develop unless normal pulp is replaced by a periodontal-like connective tissue.
4. **Multi-nucleated giant cells/macrophages** = clastic cells present

*Tronstad* **–** Tooth must be vital for Internal resorption to occur

***Heithersay*** – Two types of Internal Resorption*: Inflammatory and Replacement*

***Patel (Review)*** – *Damage to odontoblastic and pre-dentin layers resulting in exposure of mineralized dentin layer to odontoclasts*. Pulp tissue: Apical to resorption is vital, Coronal to the lesion is necrotic. 2 Types: Inflammatory and Replacement (resorption + deposition of bone/cementum-like tissues); May be symptomatic; *Differentiate between IRR and ECR with CBCT*

# Treatment of Internal Root Resorption

***Caliskan/Turkun 1997***-

1. **Etiologic factors: #1: Trauma, #2: Caries**
2. Most Common Location: *Middle 1/3rd of canal, Maxillary Anteriors*
3. RCT w/ 1 week CaOH2 and GP obturation (warm condensation) is the treatment of choice for non-perforating internal resorptive defects.
4. If perforated, CaOH2 (remineralization) should be attempted, but surgery may be necessary.
5. **Non Perforating Internal Resorption: 90% success**
6. **Perforating Internal Resorption: 25% success**

***Stamos*** – Use *Ultrasonics* to debride and *Warm gutta percha* obturation technique

# Discuss External Inflammatory Root Resorption

1. ***Tronstad*** ***1988***– Progressive External Inflamm Resorption: Damage (Trauma, root planing) to external root surface *denudes areas of precementum/cementoblasts* → **chemotactic for hard tissue resorbing cells** **(osteoclasts/odontoclasts**), *Pulpal infection sustains clastic cells*
2. ***Trope 2002 – Review Root Resorption –*** Pulp space infection – **bacteria/TEBs pass through dentinal tubules and stimulate an inflammatory response in the PDL** – Multinucleated giant cells bind/resorb the denuded root surface and *continues until the stimulus is removed*

***Gartner*** – *Buccal object rule* to differentiate external from internal resorption

***Patel*** – *CBCT* to differentiate external cervical from internal resorption

# Discuss Replacement Resorption and Dentoalveolar Ankylosis

# *Lindskog/Hammarstrom 1985* – Removal of the damaged PDL cells may inhibit dentoalveolar ankylosis and replacement resorption. Bone replacing the periodontal membrane grew from the alveolus towards the cementum.

1. ***Lindskog/Hammarstrom 1985*** – Necrotic PDL cells → Ankylosis between bone and cementum due to **repair confusion** (osteoblasts vs. cementoblasts). **2 Types:** Ankylosis w/o root resorption **(cementum-bone)** & Ankylosis following inflamm. root resorption (**dentin-bone)**
2. *Trope 2002* – Damage to the pre-cemental layer due to *traumatic injury + Inflammatory destruction of the cementum in response to dead PDL cells*
3. ***Andreasen 1975*** – Need damage of **>20%** of root surface for **progressive replacement resorption (**vs. transient replacement resorption)

# Discuss External Cervical Root Resorption

1. ***Heithersay*** – 257 teeth, ECR associated with: **orthodontic treatment (#1), trauma (#2), and intracoronal bleaching (#3)**, either alone or in combination. Recommends using *90% TCA* for treatment
2. ***Patel 2009*** – Review of External Cervical Resorption
   1. Damage to precementum or gaps at CEJ, “aseptic resorption”
   2. **Etiology: Ortho 24%, Trauma 15%, Internal bleaching 4%,** Surgery, Periodontal Therapy (Sc/Rp)
   3. **Most commonly: Maxillary Incisors/Canines, Mand Molars**
   4. Histology: **Hard base (**Caries = sticky) profuse bleeding due to **highly vascular granulomatous tissue** (*Pink spot in cervical area of crown, more common than in Internal root resorption*)
   5. Pathophys: Osteoclasts, resorption lacunae, no acute inflam.cells
   6. Often mistaken as Internal inflamm resorption**, outline of canal should be visible,** ECR follows SLOB. **Advocates use of CBCT.**
   7. Treatment – Based on Heithersay Classifications: **Treat Class I, II, III lesions** – **Curretage, 90% TCA, Glass ionomer restoration**

**Discuss External Cervical Resorption**

***Heithersay Classifications****:*

Class I: Small invasive lesion near cervical area w/ *shallow dentin penetration*

Class II: Well defined lesion, penetration *close to the coronal pulp* with *little or no extension* *into* *radicular dentin*

Class III: Deeper invasion, involving the coronal dentin and *coronal 1/3rd* of root

Class IV: Large invasion, extending *beyond the coronal 1/3rd of the root*

***Internal Bleaching and ECR:***

1. ***Rotstein*** – Internal bleaching & ECR – 30% H2O2 leakage through dentinal tubules at CEJ with no cemental layer (*Neuvald; Papadapolous* – 10%) – damages dentin, initiates inflamm/resorp.
2. *Heithersay EDT 1997* **–** *Hydroxyl radical* was generated after *thermocatalytic* bleaching w/ 30% H2O2. This radical may be one mechanism underlying PDL breakdown and resorption after bleaching.

# Orthodontic treatment, resorption and endodontics

1. *Mattison* – No difference was seen in *external root resorption* between endodontically treated teeth and vital teeth when subjected to orthodontic forces.
2. *Reitan* – Ortho movement too quickly = Pressure induced inflammatory Root Resorption (OIRR)
3. ***deSouza* *2006*** – dog study - Ortho movement (5 months) **delayed but did not prevent PA healing** in comparison to NSRCT (2 stage) teeth without ortho movement

**Discuss Apical Inflammatory Root Resorption (secondary to AP)**

1. ***Nair 2000; Trope 2002*** - Dental hard tissues (dentin/cementum) are resorbed in apical periodontitis by multinucleated giant cells (odontoclasts)
2. ***Felippe 2009*** – Apical Inflammatory root resorption produces irregular apical root surfaces and can *modify the AF, changing the working length* and resulting in *instrumentation beyond the AF (see Weiger – overinst. WL 0-2)*
3. *Malueg/Wilcox/Johnson 1996* – SEM of external apical root resorption. Necrotic teeth w/ AP had more apical root resorption than Normal or I.P.
4. *Laux/Abbott* *2000* – Radiographic/Histo correlation apical root resorption
5. ***Vier 2004*** –**75%** of teeth with PARLs had *apical internal inflammatory resorption, likely in conjunction with apical external inflammatory resorption of cementum*; Vital teeth had significantly less resorption

# CaOH2 & Intracanal Medicaments

# CaOH2 Mechanisms of Action

***Siqueira IEJ 2001*** –

* **pH (12.5) alters enzyme activity/cellular metabolism**
* **Hydroxyl (OH-) ions** created in aqueous environment: **Highly Oxidative Free Radicals (H.O.F.R.s)**:
  1. **Cell Membrane Damage**: OH- ions Remove H+ from Unsaturated Fatty Acids – *Generating free lipid radicals* and *destroying phospholipids –* key components of cell membrane
  2. **Protein Denaturation**: Alkalinization induces *breakdown of ionic bonds maintaining* *tertiary structure of proteins* = *Loss of activity of the enzyme* and Disruption of the cellular activity
  3. **DNA Damage**: OH- ions react with bacterial DNA and induce *splitting of strands*. *Inhibits DNA replication* and cellular activity.

# Does CaOH2 Kill Bacteria?

**Yes**

1. ***Sjogren*** – 7 day CaOH2 eliminated **100%** intracanal bacteria
2. ***Shuping/Trope* 2000 –** >7 day CaOH2 (avg 25 days) eliminated **92.5%** intracanal bacteria (vs. 62% with instrumentation S1-S4 + 1.25% NaOCl alone)
3. ***Safavi***– CaOH2 inactivates **LPS** (gram – endotoxin)
4. *Baik* – CaOH2 inactivates E. faecalis LTA (gram + endotoxin)
5. ***Bystrom/Sundqvist*** – Bacteria rapidly *repopulate* the canal without intracanal medicament. Negative culture following instrumentation + NaOCl + EDTA + CaOH2.

# Does CaOH2 Kill Bacteria?

**Yes**

1. *Law 2004* – Review - CaOH2 remains the best medicament available to reduce residual microflora beyond instrumentation effort.
2. *Mickel 2003 JOE* – Thin mix more effective antibacterial than thick mix
3. ***Vera/Siqueira 2012 JOE*** – 2 visit w/ CaOH2 = **↓ Bacterial counts** in *main canal,* ***d****entinal tubules,* ***i****sthmuses,* ***a****pical ramifications*, ***l****ateral canals* (**DIAL**)= Improved **histobacteriologic** status
4. ***Xavier/Martinho/Oliveria 2013 JOE*** – 2 visit w/CaOH2 were more effective at reducing **bacterial endotoxins (LPS)** than 1 visit protocols (**98% vs. 86%**)

# Does CaOH2 dissolve tissue?

**YES**

1. ***Hasselgren*** ***1988***– CaOH2 completely dissolved *necrotic porcine muscle* *tissue* in 12 days. Tissue **pretreated with CaOH2** prior to 0.5% NaOCl treatment **dissolved in 60-90 mins** vs. No complete dissolution at 12 days for tissue treated with NaOCl alone. CaOH2 causes **swelling of the tissue and increases surface area for dissolution (synergistic)**
2. *Turkun* – Pretreatment with CaOH2 enhanced tissue dissolving efficacy of 0.5% NaOCl to the level achieved with 5% NaOCl. CaOH2 causes tissues to swell and become more accessible to the NaOCl.
3. ***Wadachi 1998*** – *Bovine teeth*, *SEM analysis* of remaining pulpal tissue: **NaOCl >30 s, CaOH2 -7 days** showed significantly lower debris scores compared to shorter time intervals; Combination of **NaOCl + CaOH2** significantly enhanced the tissue dissolution effect compared to either NaOCl or CaOH2 alone (**Synergistic effect**)

## How do you place CaOH2?

1. *Sigurdsson/Madison 1992* – Compared CaOH2 placement techniques. Lentulo spiral > Calasept syringe + #25 finger plugger > #25 k-file CCW rotation for quality (density) and length of CaOH2 within canal (MB canal)

## Does CaOH2 have an effect on the apical seal?

1. *Porkaew 1990* – CaOH2 medicated teeth demonstrated *less apical leakage* (dye) than non-medicated teeth. This may be due to temporary occlusion of dentinal tubules by the CaOH2 paste or incorporation into the sealer.
2. *Kontakiotis/Wu/Wesselink 1997* – CaOH2 decolors methylene blue dye used in apical leakage studies. Compared apical leakage using fluid tranport model and dye leakage model. *CaOH2 had less leakage in only the dye leakage model,* calling into question the use of this model for analysis of leakage
3. ***Van der Sluis/Wu/Wesselink 2007*** – The negative impact of CaOH2 remnants on long term apical seal and leakage *has not been evaluated*

# What is the optimal time for CaOH2 mixture?

1. ***Hosoya 2001*** – **Optimal peak pH** (periapical tissues): Aqueous mixture (CaOH2 powder/distilled H2O) - **14 days**; CaOH2 powder alone - **49 days**; *Time required for opt. intracanal CaOH2 activity is at least 2 weeks*

# Does residual CaOH2 affect Sealer setting?

# YES

# *Margelos/Lambrianidis 1997* – Effect of CaOH2 on ZOE cement/sealers: *CaOH2 preferentially interacts w/ Eugenol* (rapid set), inhibiting ZnO-Eugenol chelate formation and *leaving eugenol in set product*; Brittle and Granular; Poor cohesion w/ destruction of sealer layer adj to CaOH2

1. ***Hosoya 2004*** – Effect of CaOH2 on various sealers: ZOE, Ketac-Endo, Sealapex. **Significant reduction in working time, Faster setting time, and Increased film thickness occurred**. Did not evaluate chemical rxns.

# Can CaOH2 be completely removed from the canal?

**NO**

1. *Lambrianidis/Margelos* *1997*– In vitro. Considerable amounts of CaOH2 remain on canal walls/apical region following irrigation/filing methods; *Filing/15% EDTA/2.25% NaOCl most effective at removing residual CaOH2*
2. ***Kenee 2006*** – In vitro. Mesial canals Mandibular molars; **MAF Rotary file or PUI (5.25% NaOCl)****significantly more effective at removing CaOH2 (3-4% remaining)** than Irrigation alone: 5.25% NaOCl; 5.25% NaOCl + 17% EDTA (19-20% remaining)
3. ***Van der Sluis/Wu/Wesselink 2007*** – In Vitro. CaOH2 + Artifical groove in apical canal. Compared removal techniques: PUI w/ 2% NaOCl, PUI w/ H2O, and syring delivery 2% NaOCl. **PUI w/ 2% NaOCl significantly more effective at removing CaOH2 (63%)** vs. PUI w/H2O (6.7%) or NaOCl only (16.7%).

# Can CaOH2 diffuse through dentin?

1. ***Tronstad*** – pH is decreased during resorption. Teeth filled with CaOH2 have *increased pH* in the surrounding dentin. (7.4-11) The pH of cementum/PDL is not effected by CaOH2 in the canal. *Increased dentinal pH may be the mechanism for stopping resorption.*
2. *Foster* – CaOH2 diffuses through root dentin to exterior surface, removal of smear layer may facilitate this diffusion.
3. ***Nerwich/Figdor/Messer*** – *hydroxyl ions* derived from a calcium hydroxide dressing diffuse through root dentin. 1-7 days elapse before pH began to rise in the outer root dentin, **peaking at pH 9.3 apically after 2-3 weeks**.
4. *Orstavik; Wang/Hume* – *Buffering capacity* of dentin inhibits OH- ion diffusion

**Does CaOH2 weaken Dentin?**

**YES**

1. ***Andreasen 2002*** – In vitro, Immature mandibular incisors (sheep), CaOH2 placed and sealed for ½, 1, 2, 3, 6, 9, or 12 months. *Significant ↓ in Fracture strength from 2 months – 12 months w/ CaOH2*.At 12 months, Dentin Fracture strength **50%** of original strength**.** **Limit use of CaOH2 to less than 30 days**, Fracture strength was not significantly reduced w/ 30 day CaOH2 period.
2. ***Rosenberg 2007*** – In vitro, maxillary incisors, CaOH2 placed 7, 28, or 84 days and compared with control (GP/Sealer). *Significant ↓ in Dentin Fracture strength from* ***28-84*** *days w/ CaOH2*. Long term use of CaOH2 decreases microtensile dentin fracture strength.

**What about CMCP?**

CMCP = Camphorated Paramonochlorophenol

1. *Messer 1984* – Antimicrobial action of CMCP sealed into pulp camber is of short duration (1-2 days)
2. *Harrison 1979* – CMCP and formocresol did not increase or decrease the incidence of interappointment pain.
3. *Madison 1992* – CMCP binds to cell membrane lipid and proteins. In addtion to being potent antimicrobial agents, this compound exhibits a high level of cytotoxicity with c.t. (severe inflammation/necrosis)
4. *Barbosa/Siqueira 1997* – Compared CMCP vs. CaOH2 vs. CHX for antibacterial effects clinically and agar diffusion tests (multiple obligate/facultative anaerobes). Clinically (1 wk) – neg cultures: CMCP = CaOH2 = CHX (69-77% red.). Agar: CMCP = CHX > CaOH2

**What about CMCP? Continued**

1. ***Haapassalo/Orstavik 1987*** – Studied the disinfection of dentinal tubules – smear layer removal facilitates bacterial invasion of dentinal tubules. *Calasept (CaOH2) failed to eliminate E. Faecalis in the tubules. CMCP was more effective*. E. Faecalis survived in tubules for 10 days without nutrient supply*.* Smear layer presence delayed pentration of irrigating solutions
2. ***Orstavik/Haapasalo 1990*** - Evaluated disinfection of infected bovine dentin sections. E. faecalis, S. sanguis, E. coli, and P. aeruginosa. Only E. faecalis survived 10 d. post withdraw of nutrients. *CMCP more efficient than Calasept at eliminating E. faecalis within tubules (60 mins vs. 10 days).* **This may be only a short term effect as CMCP evaportates rapidly!** Smear layer delays the penetration/action of medicaments.
3. *Ferguson 2002* – CaOH2 + CMCP when in direct contact were effective antifungal agents (against C Albicans)

**Do intracanal medicaments decrease pain?**

**NO**

1. *Hasselgren 1989* – The use of various dressings did not contribute to the relief of pain.
2. ***Trope 1990*** – No significant difference was found in the *flare-up rate* among the three intracanal medicaments (Ledermix, CaOH2, and CMCP)
3. ***Walton 1977*** – *Post-treatment pain is neither prevented nor relieved by medicaments* such as formocresol, phenolics (CMCP, Cresatin, eugenol, beechwood, creosote) iodine-potassium iodide, or calcium hydroxide.
4. *Torabinejad* – Flare up study – no effect on flare ups with intracanal medicaments

# Instrumentation

# Classic Instrumentation

# *Ingle 1955/61* – Standardization of instrumentation & obturation; Cites UW study for RCT failure: #1- incomplete obturation; ISO is created

1. *Seltzer/Bender 1968* – Histo studies of periapical tissue rxns to instrumentation short and beyond AF; **Long = Bad** (Inflamm/necrosis)

# *Schilder 1974* – Principles of C&S canal system; C&S most important part of RCT; Serial Preparation technique; Foramen Transportation: Zip or Apical Perf

**Design Objectives**:

1. Continously tapered funnel preparation from apex to access
2. Narrower at every point apically
3. Maintain original canal anatomy
4. AF should be maintained at same position
5. AF should remain as small as possible
6. *Wiene 1975* – **Instrumentation errors** (non-serial methods/no preflare): Elbow formation, *Apical Transportation* – *cuts outer wall below curve*; Zip AF

**Classic Instrumentation**

1. *Walton 1976* – Compared effectiveness of Filing vs. Reaming vs. Step-back Filing; Findings**: Step-back filing = highest % of pulpal walls planed**, *Reaming/Filing = more apical transportation*; White shavings ≠ Clean
2. *Caldwell 1976* – Evaluated **change in FWL before and after instrumentation** (step-back filing) – Largest change in canals with greatest curvature (MB canal Max Molars) – **NO statistically significant changes**
3. *Abou Rass 1980* – **Anti-curvature filing** – pre-curve files to instrument away from *“danger zones” – inner furcation areas*
4. *Holland 1980* – Apical plugging of infected dentin chips = inflammation/abscess; see *Tronstad* for apical plugging with non-infected dentin chips
5. *Morgan/Montgomery 1984* – Compared **crown down technique** (personal correspondence: *Marshall/Pappin*) to traditional circumferential filing – **Crown down significantly better in curved canals**

**Canal Preparation**

**Serial Preparation** (See also: *Schilder, Walton*)

1. *Coffae/Brilliant* *1975* – *serial preparations (step back + coronal flaring)* were more effective than non-serial preps (filing, reaming) in removal of tissue @ all 3 levels (1, 3, 5 mm)
2. *Walton* *1976* – tapering prep (step-back filing) permits better debridement of apical canal, reduces overinstrumentation of foramen and improves ability to obturate compared to filing or reaming only; ↓canal transport, ↑ walls planed

Clem Step back (Serial preparation)

Goerig Step down (Alternating serialization)

Torabinejad Passive step back (shaping waves w/passive instrumentation)

Abou Rass Anti-curvature filing

Marshall/Pappin Crown down pressureless (Morgan/Montgomery)

Roane Balanced force

**Discuss the benefits of the balanced force technique**

1. ***Roane/Sabala 1985*** – CW (180° or <)/CCW (120° or >) - *Restoring force of file (straightening force)* vs. *dentinal force* to allow instrumentation of curve without transportation
2. *Wu/Wesselink* – balanced-force technique produced a cleaner apical portion of the canal than the other techniques
3. ***Sepic*** – less apical transportation with balanced force technique in canals exhibiting curvature of more and less than 45 degrees.
4. *McKendry* – Balanced force technique extruded less debris
5. *Calhoun* – Using flex-R files balanced force produced more centered and round preps.

**How does tip design effect preparation?**

1. *Simon* – Tip modification (removing transitional angle) as in Flex-R, along with hand instrumentation, produced better control of preparation and less ledging.
2. ***Roane 1985*** – *Bi-beveled transitional angle tip* (Flex-R file) produced the least transportation and no ledges – minimized excessive cutting force on leading edge
3. *Moser* – Tip design contributes more to **cutting and efficiency** than flute design.

K3: Slightly + Rake Angle, Wide Radial Lands, Variable Pitch

**Why CW rotation for files?**

1. ***Seto/Nichols 1990*** – **Rotations to failure: CW > CCW**; CW: file must unwind, compression, reverse direction of flutes, tension, fracture vs. CCW: tension, fracture

**Preflaring, is it a good idea? How does it effect working length?**

1. ***Stabholz/Torabinejad 1995*** – Coronal Preflaring (Hedstroms/gates/US files) significantly **↑ Tactile detection of AC (75% vs. 32%)**
2. Baumgartner – When using SS files with GG burs, it is best to measure WL after coronal flaring. When using NiTi rotary instruments, little difference is noted whether WL is measured before or after flaring.
3. ***Ibarrola 1999*** – Pre-flaring (ProFile NiTi) allows working length files to more consistently reach FWL and **↑ efficacy of EAL** (14/16 vs. 13/16)
4. ***Roland* *2002***– Preflaring (vs. CD only) **↓ NiTi separation** (.04 Series 29)

**What about using a patency file?**

1. Paris – pass files through minor contriction to prevent dentin plug
2. ***Mullaney*** – Patency file is defined as *“a small flexible file that passively moves through the apical constriction without widening it” (Buchanan)* It is thought to **reduce the potential of forming a plug** of infected dentin/debris in the **apical 1mm**.
3. ***Goldberg*** – **Apical transportation** occurred when using a patency file (**61% - #25** vs **25% - #10**) Therefore *use small files* for patency.
4. ***Vera 2012*** – Maintaining apical patency (size #10, 1mm beyond AF) *improved irrigation penetration* in the ***apical 2 mm***
5. ***Ng 2011*** – Maintaining apical patency *improved success of NSRCT/RETX*

**Glide Path**

1. ***Varela-Patino 2005*** – Creation of glide path w/ *k-files #10-20* *↓ separation of NiTi rotary files* (K3, ProTaper, ProFile)
2. ***Peters*** – *No ProTaper .04 rotary file fractured* following glide path creation
3. *Beruti 2009* – Glide path: NiTi Path Files > SS files – less transp./canal

**NiTi**

1. ***Walia/Brantley 1988*** – *1st to study NiTi for use in endodontic files* – Compared #15 SS file with #15 NiTi (Nitinol ortho wire) file, Findings:
   1. NiTi is **2-3x more flexible** than SS
   2. NiTi **↑ Range of Elastic deformation** (bending at 90°)
   3. NiTi **↑ Resistance to fracture**, CW &CCW **torsional rotation**
2. *Wm. Ben Johnson* – 1st NiTi rotary file (Series 29)

**Compare hand stainless steel files with *hand* NiTi instruments**

1. *Esposito/Cunningham* *1995* – NiTi files (hand/rotary) were more effective in maintaining the original canal path of curved root canals when apical preparation was enlarged beyond #30
2. *Kuhn/Walker* *1997* – NiTi files (hand) remained significantly more centered and demonstrated less apical transportation than stainless steel files at size 25. When preparation continued to size 40 with step back, NSD in transportation apically or coronally
3. ***Zemner* *1995*** – NiTi files (hand) prepared **more centered** and **tapered** preparations than conventional K-files.
4. *Eldeeb* – SS files > Size #25 - cause apical transportation/zipping

**Compare hand stainless steel files with *rotary* NiTi instruments.**

1. ***Short/Baumgartner 1997***– Lightspeed and Profile NiTi files were **faster and stayed more centered** than stainless steel hand files (more pronounced at size #40 than #30)
2. ***Hata/Toda* *2002*** – Rotary files (ProFile/GT) were **faster** than hand files (Flex-R) and **decreased errors** such as **zip, elbow or ledge.**
3. *Chen/Messer* – Rotary instrumentation (Profile) may produce better canal shape versus stainless steel by reducing procedural errors.
4. *Tan/Messer* – Lightspeed NiTi files allowed greater apical enlargement w/ significantly cleaner canals, less apical transportation, and better canal shape than SS hand files
5. ***Zemner 1996*** – Profile Rotary files **more centered in canal** than SS or US Files, **more tapered preparation**
6. *Dalton 1998* – Profile Rotary files = SS k-files (stepback) in bacterial reduction

**Benefits of Pro Tapers**

1. *Berutti* – Pro Tapers are less elastic, can operate with higher loads without stress, is stronger than Profile. Pro Taper is idea for narrow curved canals.
2. *Yared* – Pro Tapers even in electric high torque control motor is safe with the experienced operator. NOTE – Inexperienced operators fractured Pro Tapers even with a low torque motor.
3. ***Peters*** – No Pro Taper instrument fractured when a patent glide path was present.

**Do rotary instruments remove bacteria?**

**YES**

1. ***Shuping/Trope JOE 2000*** *–*Profile rotaries and 1.25% NaOCl decreased bacteria **62%**. *Significant decrease in bacteria from S1 to S4 with larger file sizes.* 1 week + exposure to CaOH2 decreased bacteria 92.5%.
2. ***Bystrom & Sundqvist 1981*** – *Mechanical instrumentation reduced the number of bacteria 100 – 1000 fold* and bacteria persisted even after 4 visits
3. ***Law JOE 2004*** – Review of literature on CaOH2 effectiveness as Intracanal medicament. She noted that the *main component of antibacterial action is associated with mechanical instrumentation and irrigation w/NaOCl + EDTA*. Yet can not render canals 100% free of bacteria (DTs, ARs, LCs). CaOH2 necessary for **maximal microbial reduction** for successful healing
4. *Gupta 2013* – Outcome, PAI, 12 months. Canal 3 file sizes larger than FABF

**Does preflaring help with rotaries?**

1. ***Roland*–** Preflaring of the canal was far less likely to result in file separation.

**How much surface area does instrumentation clean?**

**MICRO CT STUDIES**

1. ***Peters*** – While instrumentation of canals increased volume and surface area, *all instrumentation techniques* *left* ***35% or more*** *of the canals’ surface area unchanged.*
2. ***Paque/Peters 2010*** – *Oval shaped canals* (Distal canals, Mand Molars), *left* ***60-80%*** *of canal surface uncleaned*. Instrumention of oval canal as 2 canals to improve surface area cleaned.

NOTE: May effectively debride only 20-40% of some canal shapes w/ files!!

**What are the properties of NiTi?**

***Shen/Haapasalo 2013***– NiTi has 2 phases: **Austenite & Martensite (3-D crystal lattice structure)**

2 properties:  **Superelasticity & Shape memory (Martensitic phase)**

1. The *ability to cycle between these two phases* results in properties
2. Phase transition occurs with rapid stress on the file, therefore use at a constant speed – *Stress-induced Martensite transformation*
3. Heating: Martensite → Austenite (Final Af temp)
4. Stress on file (during clinical use): Austenite → Martensite
5. **Martensite phase = super-elasticity, fatigue resistant**
6. *Traditional SE NiTi: Austenite; CM & M wire: Martensite (based on Af)*
7. **M wire** (Vortex, ProTaper Gold)**:** heat-patented proccess, *alteration of Af*
8. **R phase** (TF, K3XF)**:** heating/cooling patented process - R phase – twisted – cooled (TF), *No alteration of Af*
9. **CM:** thermochemical process, *alteration of Af,* Inc flexibility, **No shape memory**

**Discuss File Fatigue and Fracture**

***Haikel***:

1. Files are weakest during phase transition (cyclic fatigue)
2. **Radius of curvature** was found to be the **most significant factor** in determining thefatigue resistance of files.
3. **↓Radius of curvature, ↑ mass of metal, ↓ resistance to fatigue**
4. Cyclic fatigue is a major cause of instrument failure.

***Sattapan*: 2 types of file fatigue**

Cyclic Fatigue: result of *rotation around a curve,* repeated *longitudinal extension/compression* of the file with metal hardening and fracture

Torsional Fatigue: *tip of instrument binds while file continues to rotate*, ultimate strength of file is exceeded

*Lin* – To decrease the incidence of instrument separation utilize appropriate rotational speeds with a continuous pecking motion.

*Gambarini* – Low torque motor ↓ Cyclical Fatigue

*Gabel/Hoen* – Slower speeds, ProFiles at 333 rpm separate 4x more than at 167 rpm

*Yared/BouDahger* – Use rotary files up to 10 canals (3 Molars)

**Discuss M-wire, R-phase, and Controlled Memory NiTi Files**

***Shen/Haapasalo* JOE 2013** - REVIEW

**M Wire** (Dentsply – GTX, Vortex): Patented heat treatment of file → increased elasticity and resistance to fracture, alters Af

**R Phase** (Sybron - TF, K3XF): *Gambarini 2009;* NiTi wire (Austenitic) → Heat treatment → Cooling → R phase → Twisting process → Electropolish = TF w/ super-elasticity, shape memory, and resistance to fracture

**Controlled Memory Files** (Hyflex, Vortex Blue): Thermochemical process, Reduced shape memory for increased flexibility, Alters Af

1. *Johnson 2008* – Profile 25/.04 (M wire) vs. Profile 25/.04 (NiTi) – ↑ Cyclic fatigue w/ M wire
2. ***Gambarini 2009*** - TF (twisted R phase) > GTX (M wire) = K3 - **Cyc Fatigue**
3. ***Lopes 2013*** – R phase (K3XF) > M wire (Vortex Blue) > NiTi (K3/Revo): **Flexibility, Cyclic Fatigue, Torsional Fatigue**

**Discuss M-wire, R-phase, and Shape Memory NiTi Files**

1. *Celik 2013* – Compared Twisted Files (R phase) and GTX (M-Wire) with MTwo, ProTaper, and Race files (NiTi) and Flexo kfiles – TF = GTX = NiTi files > Kfiles
2. ***Ninan 2013*** – *Controlled memory files* (Hyflex, CM wire) showed **greater flexibility** compared to *conventional NiTi (*K3, ProFile) and *M-Wire* (Vortex, GTX) files

**Discuss Electropolishing**

1. *Herold 2007 JOE* – ProFile > EndoSequence – *Microfractures/File Separations* at 300 RPM. Electropolishing did not inhibit microfractures
2. *Ray 2007 JOE* – K3 > EndoSequence - *Cyclic Fatigue*, Electropolishing did not Inc resistance to cyclic fatigue. File design more important.

**Compare M-wire vs. R-phase vs. Traditional NiTi**

1. *Kim 2010* – Cyclic Fatigue Resistance: R-phase (TF) > Traditional (RaCe, ProFile, Helix)
2. *Al-Hadlaq 2010* – M wire greater flexibility and resistance to cyclical fatigue than Traditional NiTi
3. ***Gambarini 2009*** – R phase (TF) > M wire (GTX) = K3 (Traditional NiTi) for **Cyclic Fatigue**
4. ***Lopes 2013*** – R phase (K3XF) > M wire (Vortex Blue) > NiTi (K3/Revo): **Flexibility, Cyclic Fatigue, Torsional Fatigue**

**Cyclic Fatigue**

Rotations around curve, cycles of longitudinal tension/compression

1. ***Haikel 1999* -** *Radius of curvature = Most significant factor for cyclic fatigue*. Cyclic fatigue is a major cause of instrument failure**. ↓Radius of curvature, ↑ mass of metal, ↓ resistance to cyclical fatigue.**

**Discuss Instrument Fracture**

***Sattapan*** – 2 Methods of Failure: **Cyclic fatigue, Torsional fatigue**

***Parashos/Messer*** 2006 – Review of Rotary NiTi Fracture:

1. ***Spilli 2005*** – **3.3%** overall incidence of NiTi rotary fracture; *NSD in overall healing rate* of separated instrument vs. no instrument cases. **Only PARL affected healing rate.**
2. ***Pantivisai/Messer 2010*** – Meta-Analysis of instrument separation (Case control studies - *Crump/Natkin & Spilli* + Case Series): *Healing: w/o PARL 92.4%, w/ PARL 80.7%*; **No ↓ in Prognosis for Retained instrument alone.**

**Discuss Instrument Fracture**

1. *Crump/Natkin 1970 JADA* – No statistical difference between cases with separated instruments (81%) and control cases w/out separated instruments (73%)
2. ***Iqbal* *2006* (PennEndo)** – Incidence in Penn Grad Endo clinic: *Hand: 0.25%, Rotary: 1.68%*, **6x more likely in *apical 1/3rd*, *Mandibular molars most common (55.5%)***
3. ***Spilli*** – **3.3%** Overall Incidence, *NSD in overall healing* with presence of file. **Only PARL affected healing rate.**
4. ***Pantivisai/Messer 2010*** – Meta-analysis of Instrumentation separation (Case control studies: *Crump/Natkin & Spilli* + Case Series): *Healing: w/o PARL 92.4%, w/ PARL 80.7%*; **No ↓ in Prognosis for Retained instrument alone**

**Debris Extrusion: Instrumentation + Irrigation**

1. ***VandeVisse/Brilliant 1975***– Instrumentation + Irrigation = Debris exrusion; size 50 hand file = ↑ Apical debris extrusion
2. *Hinrichs 1998* – Rotary files (lightspeed, Profile, series 29) – no difference in debris extrusion (related to amount of irrigation)
3. ***Reddy 1998*** – K-files/flex-R files (hand files) with push-pull technique had *greater debris extrusion* than lightspeed/Profile Series 29 (rotary files)

**Does sterilization affect NiTi instruments?**

**NO**

1. ***Hicks*** – Heat sterilization of rotary NiTi files up to **10 times** does not increase the likelihood of instrument fracture.
2. ***Casper/Van Himel 2011*** – Steam heat autoclaving cycles up to **7 times** did not affect *torsional fatigue* properties of **Vortex, TF, and CM NiTi files**

**Prion Disease**

1. Misfolded proteins tranferred via contaminated instruments
2. Causes neurodegeneration (psychosis, seizures, death)
3. Sterilization may not eliminate prions – requires protein denaturation
4. Spongiform encephalopathies: Creutzfeldt Jakob Diesease (humans), “Mad Cow” (cows)
5. Reuse of NiTi rotary files comes into question due to Prions

**Steam Heat Sterilization**

Requirements:

* Time: 30 mins
* Temp: 121° C (250° F)
* Psi: 15 Psi
* Eliminates: Fungi, Bacteria, Viruses, Spores, NOT Prions

**How fast do you run Pro Tapers, Pro Files?**

1. *Martin* – Pro Tapers 350 rpm were more likely to fracture than those used at 250 or 150 rpm. A decrease in the angle of curvature of the canal also reduced the likelihood of fracture.
2. *Dietz* – Profiles .04 are less likely to break at lower rotational speeds
3. *Daugherty* – Profile .04 Series 29 rotary instruments should be used at 350 rpm, which nearly doubles the efficiency and halves the deformation rate when compared to 150rpm.
4. ***Gabel/Hoen*** – ProFiles at 333 rpm separate 4x more than at 167 rpm

# EAL

1. ***Suzuki 1942*** – Original study (Dogs), flow of current thru teeth, *electrical resistance between PDL & oral mucous membrane (lip) = constant value* ***6.5K* *ohms***, speculated could measure length of root canal using electrical resistance
2. ***Sunada 1962*** – *1st EAL* - applied Suzuki’s findings to humans, using *direct current*, *constant* ***resistance*** *between* *PDL & mucous membrane (lip)* = **6.5 K ohms**
3. ***Kobayashi 1995***– *3rd generation EAL* - developed the **Ratio Method**. **Root ZX** simultaneously measures the **impedance** **of 2 frequencies** *at 8 and 0.4 kHz* and calculates a ***quotient*** *of the impedance values* expressed as a location of file on the EAL
4. *Baumgartner* – Root ZX accurately locates minor diameter ~90% of the time

**EAL**

1. ***Ounsi/Naaman*** – *ex vivo*, Root ZX accurately locates (+/- 0.5 mm) the **AF (“Apex” mark): 84.2%, AC (“0.5” mark): 50%**. Use the AF mark only!
2. ***Shabahang*** – *in vivo/ex vivo*, Root ZX accurately locates (+/- 0.5 mm) the **AF: ~96% (using the “0.5” mark NOT “APEX” as indicator for AF**)
3. *Fouad/Krell 1990* – Eval of 5 EALs: 55-75% +/- 0.5 mm of AF; **± 0.5 mm** is clinically appropriate based on practice of subtracting 1 mm from EAL length

**How accurate is radiographic working length estimation?**

1. ***Weiger 2001*** – Radiographic WL *0-2mm short of apex* causes unintentional **overinstrumentation** in **51% of premolars** and **22% of molars**.
2. ***Fouad/Krell 1990*** – Radiographic WL Accuracy**: 53% (**eval of 5 EALs)
3. ***Hembrough 1993*** – Max DB/P canals, *Radiographic WL Accuracy:* ***88.5%****, EAL Accuracy (SonoMark III):* ***73.1%***. **Radiograph better than EAL**.

# Uses of EAL

1. *Katz* – root length measurement in primary dentition
2. ***Fuss*** - locate root perforations

# Does any solution effect the Root ZX?

**No**

1. *Meares* et al – Root ZX not adversely affected by presence of NaOCl (w/in 0.5 mm of AF 83%)
2. ***Jenkins* *JOE 2001***– No difference in length determination as a function of the seven irrigants used (w/in 0.31 mm of AF): 2% Lidocaine, 5.25% NaOCl, RC prep, EDTA, Peridex, Hydrogen Peroxide, Saline

# Does apical resorption affect the Root ZX?

**No**

1. ***Goldberg*** – Apical root resorption did not effect determination of FWL

# Does Pre-Flaring help with the Root ZX?

**Yes**

1. ***Ibarrola*** – Apical foramen could be reached more consistently by preflaring the canals before obtaining working length with EAL (13/16 vs 14/16)

# Does pulp status matter with the Root ZX?

**NO**

1. ***Dunlap 1998*** – In Vivo, *NO statistical difference in accuracy* for AF between between **vital and non-vital cases** with Root ZX (Vital= 0.2 mm, Necrotic= 0.5 mm, NSD). *Necrotic cases had more readings past the AF.*
2. *Pommer* – vital more accurate than non vital with non ratio units

# Can you use an EAL with a pacemaker patient?

1. ***Garofalo 2002*** – **In Vitro**, 4 of 5 EALs tested **did not** cause inhibition or interfere with normal pacemaker function
2. ***Wilson/Baumgartner 2006*** – **In Vivo**, 2 EALs and 1 EPT **did not** interfere with cardiac pacemaker or cardioverter/defibrillator in 27 patients
3. *Baddour JADA 2011* – Review – No antibiotics needed for pacemakers/cardiodefibrillators

**Irrigation**

**Is an Irrigant Necessary?**

1. ***Baker JOE 1975*** – **70% more debris** remaining in canals instrumented w/o irrigation; *Flushing action of solvent* *↓ debris*; **↑ volume, ↑ solvent effect**
2. ***VandeVisse/Brilliant JOE 1975*** – Instrumentation without irrigation – Files *bind/separate* and *block canals with debris*; **#50 file ↑ debris extrusion**
3. *Bystrom & Sundqvist 1981* – Mechanical instrumentation reduced the number of bacteria 100 – 1000 fold and *bacteria persisted even after 4 visits*
4. ***Peters*** – While instrumentation of canals increased volume and surface area, *all instrumentation techniques* left **35%** or more of the canals’ surface area unchanged (***Paque/Peters*** – **60-80% Oval shaped** canal surface undebrided)

Instrumentation w/o Irrigation: Debris, Files binding/separating, Inability to completely clean

**How large should the apical preparation be for irrigation?**

1. ***Salzgeber/Brilliant JOE 1977*** – Canal must be instrumented to **size 35** hand file (.02)/Gates #2 for irrigation to penetrate the *apical extent* of the canal (Mand. Molars & R.O. Dye)
2. *Abou-Rass/Piccinino 1982* – A 30 gauge irrigation needle can be placed in the *apical 1/3* of the canal when the **apex is size #30**.
3. ***Boutsioukis/Van der Sluis IEJ 2010*** *–* 3-D computational flow dynamics, canals instrumented to a **35/.06** or greater improved apical irrigation flow (*side vent needle – 30 g*)
4. *Chow 1983* – Size 40
5. *Ramz 1977*

**Discuss Irrigation Devices**

1. *Kahn/Rosenberg 1995* – The **Max-i-Probe** syringes (side-vented 2mm from closed end) were the most effective instrument used to clear dye from the simulated canals in both the mandibular and maxillary positions. Canals were instrumented to size 30 or 35 file. Compared with end-vented Monoject syringes, Cavi-Endo (ultrasonic), and Micromega (sonic) irrigation devices.
2. ***Siu/Baumgartner 2010*** – In vivo, EndoVac vs. Conventional side vented needle (30 g), evaluated **Tissue debris (cleanliness)**: 3 mm from WL: EndoVac = Conventional, *1 mm from WL: EndoVac > Conventional*
3. ***Desai/Himel 2009*** – In vitro, **EndoVac no debris extrusion**, *Endo Activator (sonic) insignificant debris extrusion*, Max-i-Probe = Ultrasonic = Rinsendo for significantly greater debris extrusion periapically

**Delivery of Irrigation**

**Side-Vented Needle**:

1. ***Boutsioukis/Van der Sluis IEJ 2010*** *– 3-D computational flow dynamics -* side vent needle (30 g) - irrigant flow **1.5 mm beyond tip**
2. ***Boutsiouskis/Lambrianidis JOE 2010*** – Side vent needle (30 g) achieved adequate **irrigant replacement** within **1.0 - 1.5 mm apical to tip**

**Passive Ultrasonic Irrigation (PUI)**:

1. ***Van der Sluis IEJ*  –** *3-D computational flow dynamics* **-** Irrigant flow **3.0 mm beyond ultrasonic tip**

**Irrigation and Dentinal Tubules**

1. ***Buck/Eleazer 1999*** – In vitro infection of dentinal tubules; **Irrigants can peneterate well into tubules***, effectiveness dependent on type of bacteria*
2. ***Orstavik/Haapasalo 1990*** – In vitro (dentin sections) infection of dentinal tubules. **Presence of smear layer impaired** **ability of irrigants and medicaments from penetrating into the dentinal tubules**. Resistance of E. faecalis infection to intracanal medicaments may be partly due to *density of tubular infection*.
3. *Pashley*

**Irrigation and Biofilms**

1. ***Clegg 2006***– *Polymicrobial biofilm* created on root dentin hemisections, *SEM analysis*: 6% & 3% NaOCl – Disrupt & Eliminate biofilm; 1% NaOCl & 1% NaOCl/EDTA – Disrupt but not eliminate biofilm; 2% CHX – No disruption of biofilm; **6% NaOCl only irrigant to render bacteria non-viable, disrupt and eliminate biofilm**
2. ***Dunavant 2006*** - *E. faecalis* *biofilm* grown in *flow cell system* – **% Bacteria Kill**: **6% NaOCl**>1%NaOCl>>Smear Clear>2%CHX>REDTA>MTAD
3. ***Wang/Shen/Haapasalo 2012*** – *E. faecalis biofilms* in *dentinal tubules*. Utilized Confocal Laser Scanning Microscopy (CLSM) for evaluation of biofilm disinfection. **Qmix = 6% NaOCl > 2% CHX or 2% NaOCl** for *antibacterial effects* on **1-day old biofilm (“young”)** but **6% NaOCl > QMix > 2% NaOCl or CHX** for **3-week old biofilm (“mature”)**

**Mechanisms of Action**

***Zehnder*** *–* Irrigation Review:

1. NaOCl: *Halogen releasing compound*, High pH, Chlorine oxid (HOFR)
2. CHX: *Bisguanide*, Strong base, no tissue disol., more effect. on *Gram +*
3. EDTA: *Polyprotic acid*, chelatingmetal ions (Ca+2), mild antibact.

***Estrella*** – **NaOCl Mechanisms of Action**:

1. **Tissue Solvent** – Hypochlorous acid releases chlorine, formation of **chloramines**, and leads to amino acid degradation and hydrolysis
2. **Antibacterial effects** – **HOFR = Chlorine**
   1. Inhibits bacterial enzymes/disrupts cellular metabolism thru **Chlorine oxidation**
   2. Strong Base – pH > 11 – **similar mechanisms to CaOH2**
      1. Interferes with cell membrane integrity – **phospholipid degradation**
      2. Inhibits enzyme activity through **protein denaturation**

**Discuss NaOCl. What concentration is best?**

1. *Harrison/Baumgartner 1978* – 5.25% is safe for clinical use, does not increase Inter-appt pain (measure of clinical toxicity)
2. *Hand/Harrison 1978* – Dilution of 5.25% NaOCl significantly inhibits the *Tissue dissolving ability* of NaOCl
3. ***Rosenfeld JOE 1978*** – 5.25% NaOCl dissolves **vital pulp tissue/**predentin (non-specific), does not dissolve calcified tissues or lateral canal tissues
4. *Harrison/Hand 1981*- Dilution of 5.25% NaOCl significantly inhibits the *Antibacterial properties* of NaOCl

**Discuss NaOCl. What concentration is best? Continued**

1. *Cunningham 1980* – 2.6% sodium hypochlorite solution at room temp was found to be equally effective as a collagen-dissolving agent when compared to 5.25% at body or room temp. **Inc. temp, inc. efficiency**.
2. ***Stojicic/Haapasalo 2010*** – NaOCl & Bovine muscle tissue dissolution: **↑ dissolution = ↑ Conc. NaOCl** (5.8%, 4%, 2%, 1%), **↑ Temperature** (45 vs. 37° C), **↑ Agitation** (UltraSonic, Sonic, Pipetting), **↑ Surface active agent**
3. ***Clegg* 2006**– *Polymicrobial biofilm* created on root dentin hemisections, *SEM analysis*: 6% & 3% NaOCl – Disrupt & Eliminate biofilm; 1% NaOCl & 1% NaOCl/EDTA – Disrupt but not eliminate biofilm; 2% CHX – No disruption of biofilm; **6% NaOCl only irrigant to render bacteria non-viable and eliminate biofilm from hemisections**

**Discuss NaOCl. What concentration is best? Continued**

1. ***Dunavant* 2006** - *E. faecalis* *biofilm* grown in *flow cell system* – **% Bacteria Kill**: **6% NaOCl**>1%NaOCl>>Smear Clear>2%CHX>REDTA>MTAD
2. ***Wang/Shen/Haapasalo 2012*** – *E. faecalis biofilms* in *dentinal tubules*. Utilized Confocal Laser Scanning Microscopy (CLSM) for evaluation of biofilm disinfection. **Qmix = 6% NaOCl > 2% CHX or 2% NaOCl** for *antibacterial effects* on **1-day old biofilm (“young”)** but **6% NaOCl > QMix > 2% NaOCl/CHX** for **3-week old biofilm (“mature”)**
3. *Bystrom & Sundqvist 1985* – No difference was noted between the antibacterial effect of 0.5% and 5% NaOCl. *The combined use of EDTA/NaOCl was more efficient, but did not eliminate all the bacteria*. Bacteria that survive the instrumentation and irrigation rapidly increase in numbers between appointments.

**Heating the NaOCl & Tissue dissolution**

1. ***Cunningham* *1980*** - ↑ Heat (37° vs 22°), ↑ Tissue disolution effect of NaOCl
2. ***Stojicic/Haapasalo 2010*** – NaOCl & Bovine muscle tissue dissolution: **↑ dissolution =** ↑ Conc. NaOCl (5.8%, 4%, 2%, 1%), **↑ Temperature** (**45 vs. 37° C**), ↑ Agitation (UltraSonic, Sonic, Pipetting), ↑ Surfactant

**Discuss Sodium Hypochlorite accident**

1. ***Becker 1974*** – Case report – *swelling, ecchymosis, severe pain, hemorrhage w/in canal* – Tx: Antibiotics, Corticosteroids, Anaglesics
2. ***Gluskin*** – Tx: long acting anesthetic (marcaine), irrigation with saline to dilute, antibiotics (Amoxicillin), analgesics, steroids, cold compress

**Discuss EDTA as an irrigant**

EDTA = Ethylene Diamine Tetraacetic Acid

* Act as metal ion chelator, strips Ca+2 ions from mineralized dentin (softens dentin)
* Polyprotic acid

1. *Nygaard-Otsby 1957* – 1st to discuss EDTA as root canal irrigant, softening dentin allows instrumentation of curved canals
2. ***Zehnder 2006*** – EDTA is effective demineralizing agent, highly biocompatible, *removes inorganic smear layer*, limited antiseptic capacity
3. ***Haapasalo 2010*** – Review – EDTA *dissolves inorganic material*, including *hydroxyapatite*. No effect on organic tissue. No antibacterial effect.

EDTA – a disodium salt solution that collects Ca ions and replaces with Na, making dentin softer – **Chelates Ca+2 ions (binds/strips from dentin)**

**RC Prep – introduced by Stewart**

Old formulation – 3.8% EDTA, Urea peroxide, propylene glycol, carbowax

New formulation – **3.8% EDTA, Urea peroxide, propylene glycol**

1. *del Rio 1975* - Old formulation remained after instrumentation
2. *del Rio 1976* – RC Prep caused increased apical leakage of radioactive iodine, less was noted in cases sealed with gutta-percha than with silver wires.
3. *Schafers 2002* - New formulation improved cleanliness of the root canal walls in the coronal and middle parts of the root canal
4. ***Peters IEJ 2005*** – **EDTA liquid > Glyde (EDTA) Paste** - ↓ Max. Torque values (ProFile), ↓ Full Torsional load over time (ProFile/ProTaper); **Glyde ↑ Apical directed force (ProFile)**

**What is the smear layer?**

1. ***McComb & Smith JOE 1975*** – **1st to describe the smear layer**: Smear Layer = surface layer completely obscuring the dentinal tubules composed of *superficial debris* and *embedded erythrocytes* scattered over the surface
2. ***Mader/Baumgartner 1984*** – *SEM analysis* of smear layer - found **2 components**:
   1. **Surface Layer** – *thin layer*, amorphous/irregular - **1-2 microns** thick
   2. **Dentinal Tubule Layer** – irregular *finger projections into dental tubules,* variable distances up to **40 microns**
3. ***Sen 1995*** – The smear layer is made up of **inorganic and organic debris**. (Pulpal tissues, Dentin debris, bacteria, bacterial TEBs: Toxins/Enzymes/Byproducts)

**Does the smear layer effect the apical or coronal seal?**

**YES**

1. ***Cergneux/Holz 1987*** – Better apical seal occurred when the smear layer was removed with EDTA
2. ***Jeansonne 1997*** – Less coronal leakage was seen when the smear layer was removed. AH-26 displayed less leakage than Roth’s 811sealer

**NO**

1. *Madison/Krell* *1984*– Apical seal (dye leakage) for obturation is not improved by irrigation w/EDTA. Compared NaOCl vs NaOCl+EDTA. **No difference in apical seal integrity with or without smear layer removal.**

**Should the smear layer be removed?**

**YES**

1. ***Torabinejad 2002*** – Infected cases - removal of smear layer to **decrease bacterial infection** and **improve adaptation of obturation materials**
2. ***White/Goldman 1984*** – **Penetration into dentinal tubules** by filling materials is possible *after* the smear layer removal
3. ***Wang/Shen/Haapasalo 2013*** – Smear layer *reduces the effectiveness of irrigants (6% NaOCl, 2% CHX, QMix)* – E faecalis biofilm/dent. tubules
4. ***Cergneux/Holz 1987*** – Better apical seal when smear layer removed
5. ***Jeansonne 1997*** – Better coronal seal when smear layer removed

**NO**

1. ***Drake 1994*** – do NOT remove smear layer, **smear layer inhibits bacterial penetration of dentinal tubules**

**How much/long should EDTA be used?**

1. ***Crumpton*** – In vitro, *1 mL/1 min of 17% EDTA = 10 mL/1 min of 17% EDTA* for smear layer removal; **Use** **1 mL for 1 min to remove smear layer**
2. *Saito/Webb* – In vitro, 1 min EDTA rinse
3. ***Calt/Serper 2002*** – *1 minute irrigation of EDTA is effective* in removing the smear layer. A **10 min** **application of EDTA** causes **excessive peritubular (intertubular) dentinal erosion**
4. ***Schilder*** *–* EDTA self-limiting at 7 hours

**Discuss EDTA/NaOCl combination for irrigation:**

1. ***Yamada/Goldman 1983*** – **1st to evaluate EDTA/NaOCl combination** as **a final flush to remove both inorganic and organic components**; For overall canal cleanliness (SEM), use final flush w/ 10ml of 17% EDTA (inorganic-smear layer) followed by 10 ml of 5.25% NaOCl (organic-debris)
2. ***Baumgartner/Mader 1987*** – 5.25% NaOCl and 17% EDTA more effective than NaOCl and saline for smear layer removal. **NaOCl/EDTA** combination **removed all smear layer as well as pulpal remnants/predentin on uninstrumented side**

**Discuss MTAD**

**Mix + Tetracycline + Acid + Detergent**

1. *Torabinejad 2003* – MTAD appeared similar to EDTA in solubizing effect on pulp and dentin. It had a high binding affinity of doxycycline for dentin.
2. ***Torabinejad 2003*** – MTAD killed E. Faecalis in human dentinal tubules in 5 minutes and was more effective than 5.25% NaOCl.
3. *Torabinejad 2003* – 1.3% NaOCl is recommended for irrigation to compliment MTAD (reduced antibacterial properties)
4. ***Torabinejad 2003*** – Components:
   1. Doxycycline – Prevents E. Faecalis in 100% of samples
   2. Acid – Removes smear layer w/out erosion of dentin
   3. Tween-80 (Detergent) – Reduces surface tension, increases dentin penetration

**Is Chlorhexidine an effective irrigant?**

1. ***Jeansonne 1994*** – Ex Vivo, **No difference in antimicrobial activity (ex vivo, pulpal pathosis**) between **2% CHX** and **5.25% NaOCl**, but NaOCl had added advantages of tissue dissolution. CHX is an excellent irrigating alternative for *NaOCl allergic patients, perforations and teeth with open apices*.
2. ***White 1997*** – Antimicrobial activity lasted **72 hours** after use with 2% CHX, 0.12% produced 6 –24 hrs. CHX *binds to dentin* and is *released over time* = **substantivity**
3. ***Haapasalo*** – CHX antibacterial effects: *Permeates cell wall/membrane, attacks cytoplasmic membrane, allowing* ***leakage of cellular contents***
4. *Siqueira 2001* – Only 2% CHX was able to eliminate most of both 1 & 3 day E. Faecalis biofilms (opposes *Dunavant* and *Wang/Shen/Haapasalo*).

**Is Chlorhexidine an effective irrigant? Continued**

1. *Hartwell 2003* – CHX 0.12% did not adversely affect the apical seal of Roths cement at 270 and 360 days when used as an endo irrigant.
2. ***Gomes 2003*** – **2% CHX** was more effective against **E. faecalis** than CaOH2 (disturbs bacterial cell wall osmotic equilibrium) see also *Orstavik/Haapasalo*
3. ***Baumgartner 2003*** – **CaOH2 + 2%CHX** was more effective **killing E. faecalis** in the dentinal tubules than CaOH2 + H2O.
4. *Basrani* – 2% CHX better than 0.12% for Antibacterial effects
5. *YuShen/Haapasalo* – CHX + US = Better antibacterial action

**QMiX**

*QMiX = EDTA + CHX + Detergent (Smear layer removal + Antibacterial)*

**Does not interact with NaOCl, Recommended as Final rinse following NaOCl**

1. ***Stoijicic/Haapasalo 2012*** – *QMiX and 2% NaOCl were equally effective* and superior to 1% NaOCl, 2% CHX, and MTAD at killing E. faecalis and plaque bacteria in *planktonic and biofilm (3 wk old) conditions*
2. ***Wang/Shen/Haapasalo 2012*** – E. faecalis biofilms in dentinal tubules. Utilized Confocal Laser Scanning Microscopy (CLSM) for evaluation of biofilm disinfection. **Qmix = 6% NaOCl** for antibacterial effects on **1-day old biofilm (“young”)** but **6% NaOCl > QMix** for **3-week old biofilm (“mature”)**

**Irrigant Interactions**

**NaOCl & CHX**:

*Basrani* – Parachloroanaline (PCA) brown ppt, mutagenic/carcinogenic

*Nowicki* – No parachloroanaline (PCA) in brown ppt

**NaOCl & EDTA**:

*Zehnder* – EDTA reduces free Cl-, NaOCl *ineffective as antibacterial/tissue solvent*

*Grawehr* – same as Zehnder, EDTA inhibits NaOCl’s antibacterial and tissue disolution properties through *reduction of free Chlorine ions* in solution

**EDTA & CHX**:

*Racemic* – white salt ppt

**Long Term EDTA**:

*Calt/Serper* – 10 min rinse of dentin bars resulted in *excessive intertubular dentin erosion*

**Final Rinse – EDTA → NaOCl** (See*Yamada/Goldman*):

*Qian/Haapasalo 2011* – Intertubular dentinal erosion due to NaOCl after EDTA

*Niu* – Same findings as *Qian/Haapasalo*

**Ultrasonics**

**What about ultrasonics? How do they work?**

***Cunningham 1982* –** Ultrasonic preps (endosonic file/irrigation) produced significantly cleaner canals and reduced smear layer better than hand instruments. **Ultrasonic energizes/activates/warms irrigating solution = ↑ solvent action**

***Goodman/Reader 1985*** – **1st to discuss PUI** – step-back vs. step-back + PUI: **Step-back+PUI** **greater cleaning ability** (isthmus 1, 3 mm/main canal 1 mm)

***Ahmad/Pitt Ford 1987*** – **Acoustic streaming**, not cavitation, exists with the Cavi-Endo and aids in debridement of large straight canals.

*Walmsley 1989* – If endosonic files are constrained (bind) near the tip, their motion and effectiveness is decreased. Use sonic files loose in the canals.

*Zeltner* – Temperature increase during use of ultrasonic instrumentation improves antibacterial effect

**Do ultrasonics remove bacteria?**

**Yes**

1. *Hoshino 1998* – Ultrasonic irrigation with 5.5% NaOCl eradicated bacteria from infected dentin.
2. ***Carver 2007*** – Hand/Rotary/PUI *Necrotic* Mandibular Molars (Mesial roots) – *1 min* – **7x more likely to have negative culture** – than Hand/Rotary only. Antibacterial effect

**No**

1. *Hicks 1989* – Cavi-endo and hand instrumentation were equally effective in removing bacteria form the root canal.

**Are Ultrasonics effective in canal cleaning?**

**Yes**

1. *Archer/Reader* 1992 – Combination step-back with ultrasonic instrumentation (3 min) resulted in a cleaner preparation than step-back technique alone in both the canals and isthmus.
2. *Hutter 1999* – 3 min passive activation of either sonic or ultrasonic produced significantly cleaner canals than hand instrumentation alone. Also, there was NSD in cleaning efficacy between sonic and ultrasonic activation.
3. ***Gutarts 2005*** – In Vivo, Hand/Rotary/PUI vs. Hand/Rotary only. *1 min* PUI *Vital* Mand Molars (Mesial roots) – Sig. Cleaner Main canal/isthmuses
4. ***Burleson 2007*** – In Vivo, Hand/Rotary/PUI vs. Hand/Rotary only. *1 min* PUI *Necrotic* Mand Molars (Mesial roots) – Significantly Cleaner Canal/Isthmuses (1, 2, 3 mm levels)

**Are Ultrasonics effective in canal cleaning?**

**No**

1. ***Walker*** – NSD between sonic, ultrasonic and hand instrumentation regarding debris removal and canal wall planing in *curved* canals.
2. *Holz 1989* – Ultrasound in association with EDTA did not enhance the dissolving capability of this chelating agent. Neither NaOCl nor EDTA successfully removed the smear layer in the apical portion of the canal.

**Ultrasonics & CaOH2**

1. ***Metzler/Montgomery***– In vitro, extracted teeth, ***PUI*** *(CaviEndo, 2 min)* and ***CaOH2*** *(7 days)* are **equal at cleaning main canals and isthmuses** and are **more effective than hand instrumentation + irrigation (2.6% NaOCl) alone** at debriding the **apical isthmuses (1 mm)**. Recommends 1 visit treatment – PUI irrigation (2 min), 2 visit treatment – CaOH2 (7 days).

**Ultrasonics & Cardiac pacemakers/Implanted defibrillators**

1. ***Roedig* 2010** - **Ultrasonic scalers** (*magnetostrictive)* **and battery operated curing light** DO interfere with pacemakers/defibrillators; EPTs, Electrosurge do not. See also: *Garofalo* (5 EALs), *Wilson/Baumgartner* (2 EALs, 1 EPT)
2. ***Gomez 2013*** – In vitro, ***Piezoelectric* ultrasonics** do NOT interfere with pacemakers/cardiodefibrillators

Obturation

Rational for filling 0.5 mm – 1.0 mm short of the radiographic apex

***Kuttler*** – distance from the major (AF) to the minor diameters (AC)

**0.525 mm** (18-25y/o)

**0.659 mm** (>55 y/o)

***Burch*** – measured from the occlusal aspect of the major diameter (AF) to the apex

Average distance for all roots = **0.59 mm**

1. ***Schaeffer*** – *Meta Analysis of Termination of Obturation*: Healed %: Group A (0-1 mm short of RA) > Group B (1-3 mm short) >> Group C (overextended past apex); Overall success: **Group A 2.7% better than B and 26.2% better than C**
2. ***Ricucci/Langeland*** – I/O to the **AC** to *prevent injury to the periapical tissues and foreign body rxn/tissue necrosis due to extrusion of sealer/gp*; I/O to 1 mm short of RA should NOT be used as this may end up overinstrumenting

**Discuss the Hollow Tube Theory**

***Richert & Dixon 1931*** – Hollow tube theory: the root canal must be filled to the end of the tooth to prevent *outward diffusion* of *circulatory elements* which *cause inflammation*.

**Disproved by: (TGW)**

***Torneck 1967*** – This study tested the reaction of rat connective tissue to polyethylene tube implants. **Best prognosis for repair was a sterile empty tube**; followed by a sterile

tube with sterile tissue. Worst prognosis was with sterile tube and infected tissue.

***Goldman 1965*** – Teflon rods were implanted in guinea pigs. An interchange of tissue fluids into and out of the tube occurred. There was **no evidence of inflammation** at the **open end** of the rods. **Disputes the “Hollow Tube” theory**

***Wenger 1978*** – Polyethylene tubes **obturated flush at one end and 1mm short at the opposite end** **with gutta percha and Grossman’s cement** were implanted in rat tibias. The Gutta-Percha, the set Grossman’s cement and the polyethylene implant were well tolerated by the rat intraosseous tissue. There was **no inflammatory response** at **either end** of the polyethylene tubes.

**Gutta-Percha**

**What is in gutta-percha?**

***Friedman 1975*** – *20% gutta-percha, 66% zinc oxide*, *11% heavy metal sulfates* (i.e.: Barium sulfate- radiopacifier), and *3% waxes and/or resins* (plasticizer).

**Does age affect gutta-percha?**

*Kolokuris 1992* – Moisture makes gutta-percha more plastic and workable. *Store in the fridge and at high humidity*.

***Sorin 1979*** – *Rejuvenate by alternating heating and quenching*. Immersion in hot tap water (above 55 degrees C) then remove and immerse in cold tap water or alcohol for several seconds and ready for use. Cones treated as such remain stable for months.

**Is Gutta-Percha biocompatible?**

1. ***Pascon/Spangberg 1990*** – In vitro, warmed/dissolved gp - *toxicity of gp is attributable to leakage of* ***Zinc ions*** *into the fluids*
2. *Sjogren & Sundqvist 1998* – Mouse peritoneal macrophages, when exposed to gutta-percha particles, release factors which have a bone resorbing activity that is primarily due to enhanced production of IL-1α.
3. ***Nair 1995*** – **Large pieces of GP** were **well encapsulated** by a collagenous capsule and the surrounding tissue was **free of inflammation**. **Fine particles** evoked an **intense, localized inflammatory response**, characterized by the presence of macrophages and multinucleated giant cells.

**Properties of Gutta-Percha:**

1. *Marciano 1993* – Both natural and commercial GP mainly have a 1-4 trans stereochemical structure (not altered by heating during GP fabrication process) and that the coloring agent is erythrosin
2. ***Schilder 1974*** – Thermomechanical Properties of Gutta Percha: Gutta percha exists in a **beta semicrystalline state**. It undergoes *2 phase transformations* upon heating from 0-100°C. The **Beta to Alpha** **transition** occurs at **42-49°C;** the **Alpha to Amorphous** at **53-59°C**. **GP is Compactable, NOT compressible**.

**β → 46° → α →56° → Amorphous → slowly cooled →→ α**

β → 46° → α →56° → Amorphous → normal cooling →→ β

**Is latex allergy a concern with Gutta Percha?**

1. ***Johnson JOE 2001*** – **No cross-reactivity to latex** was observed with any of the raw or clinically used gutta-percha products. The ***absence of gutta-percha proteins that can react with Hevea latex-specific IgE antibody*** supports the *minimal potential* for commercially available gutta-percha to *induce allergic symptoms in individuals sensitive to latex.*
2. *Hamann JADA 2002* – No detectable cross-reactivity between latex and commercial gutta-percha points. Gutta-percha alone is not likely to induce symptoms in patients with type I NRL allergy.
3. *Kleier JOE 1999* – Although no cross-reactivity w/GP DDS may take the following precautions
   1. Pre-test GP w/ latex sensitive pt by allergist
   2. consider premed w/ prednisone and diphenylhydramine
   3. prepare for the management of allergic rx w/ EpiPen

Latex allergy vs Gutta Percha

1. *Costa/Johnson JOE 2001* – Gutta-percha does not have the same allergenicity as latex

Gutta-percha and gutta-balata are derived from the ***Paliquium gutta*** and Mimusops globsa trees, respectively, that are in the *same botanical family as the rubber tree Hevea brasiliensis*. For this reason the potential for immunological cross-reactivity between the gutta-percha and gutta-balata used in endodontics and natural rubber latex (NRL) has been the subject of some controversy, because these products may be used in latex-allergic individuals. The objective of this study was to investigate the **potential cross-reactivity between gutta-percha, gutta-balata, and NRL.** Physiological extracts of seven commercially available gutta-percha products, raw gutta-percha, raw gutta-balata, and synthetic transpolyisoprene were each analyzed for cross-reactivity with NRL in a competitive radioallergosorbent test inhibition assay**. No detectable cross-reactivity was observed with any of the raw or clinically used gutta-percha products.** In contrast the **raw gutta-balata released proteins that were cross-reactive** with Hevea latex. We conclude that the **absence of gutta-percha proteins that can react with Hevea latex-specific IgE antibody supports the minimal potential for commercially available gutta-percha to induce allergic symptoms in individuals sensitized to NRL**. Because gutta-balata is sometimes added to commercial gutta-percha products caution should be exercised if products containing gutta-balata are used in endodontic care of latex-allergic individuals.

**History of Obturation Techniques**

1. *Schilder* – Warm vertical compaction
2. *Yee* – Injection thermoplasticized (warm) gutta percha
3. *Wm. Ben Johnson* – 1st Carrier based obturation (ss file + warm gp)
4. *Buchanan* – Continuous Wave Warm Vertical technique

**Is Obturation more important than Instrumentation/Irrigation?**

**NO**

***Sabeti 2006*** – Mixed German shepards, PN/AP, Instrumentation/Irrigation ± Obturtion. **Equal healing w/ and w/o obturation + coronal seal (@190 days).** *Healing depends on microbial elimination, host response, and bacteria tight seal coronally. Not Obturation*.

***Nair 2006*** – Microbial status of apical root canal system of Mandibular Molars with AP after ONE visit NSRCT. Histo sectioning/SEM analysis: 88% canals harbored intraradicular bacteria BIOFILMS within **DIALs**: Dentinal Tubules (**D**Ts), Isthmuses (**I)**, Apical Ramifications (**A**Rs), and Lateral Canals (**L**Cs). **Residual infection may communicate with the PDL/AP and elicit host response/derive nutrient source, promoting post-tx AP**. (***Vera/Siqueira***)

\*\**Entombing bacteria in GP and nutritional starvation* (*Peters/Wesselink*) **may NOT be effective**, may need maximal microbial killing through CaOH2(negative culture studies: Bystrom/Sundqvist, Sjogren, Shuping/Trope)

**What spreader is best for lateral compaction? How far should it be placed?**

1. *Schmidt JOE 2000* - Niti spreaders penetrate farther w/ less force than stainless, minimizing risk of vertical root fractures
2. *Joyce JOE 1998* – Niti spreaders induce stress patterns that spread out along the surface of the canals reducing the risk of vertical root fx.
3. ***Walton JOE 1981*** – Less leakage occurs with *deeper spreader penetration* (**w/in 1mm or 2mm of FWL)**
4. *Trope JOE 1991* – Dye study, less leakage with *finger spreaders* than D11T
5. *Messer JOE 1999* – Max loads and strain generated with finger plugger were lower than those generated with a hand spreader D11T. (even lower than the values at fracture). Therefore lateral compaction should not be a factor causing vertical root fracture.

**Compare lateral compaction and warm vertical compaction**

1. *Brothman JOE 1980* – Vertical vs Lateral – Veritcal filled more lat canals, was denser on radiograph but no difference was seen histologically, apical 1/3rd was filled equally well with both techniques.
2. *Hoskinson OOOO 2002* – Vertical vs Lateral – no difference in success, presence of AP was biggest factor, success decreased 18% for every 1mm in size of pre-operative periapical lesion.
3. ***Jacobson/Baumgartner JOE 2002*** – Compared post-obturation leakage in Lateral vs Continuous Wave Warm Vertical obturated canals *- Lateral condensation resulted in significantly faster coronal leakage; NSD in overall leakage between the two techniques at the end of the test period.*

**Compare lateral compaction and warm vertical compaction**

1. ***C. Reader JOE 1993*** – Lateral vs Warm Lateral vs Warm Vertical to fill simulated lateral canals – *NSD in quality of fill* between techniques but *more GP was found in lateral canals with warm techniques* (See *Riccuci/Siqueira*)
2. ***Peng JOE 2007*** – **Meta-Analysis Cold Lateral vs. WV** – *No difference: Post op pain, Long term outcomes, Obturation quality;* WV more overextensions
3. ***Dulac JOE 1999*** – In vitro, resin blocks w/ simulated lateral canals, Evaluated ability to obturate lateral canals: *CWWV and Carrier Based (CB) better than Cold Lateral for obturating lateral canals* in all 3 thirds of canal
4. ***De Chevigny (Toronto study)*** – NSRCT - WV vs. Cold Lateral – *Healed rate 10% higher w/ WV*

**Does obturation cause vertical root fractures?**

1. ***Holcomb/Pitts JOE 1987*** – **Excessive lateral force** can cause VRF. Suggested condensation forces **<2.5lb** as “safe limited load”. This corresponds with 70% of the minimum load resulting in fractures. Fractures usually occur facio-lingually.
2. *Lindauer/Hicks JOE 1989* – Described lateral forces acceptable for treatment of mesial roots of mandibular molars without causing vertical root fractures *(2.2 lbs to 10.8 lbs*)

NOTE: THIS DATA CONFLICTS WITH HOLCOMB’S FINDINGS

1. ***Meister 1980*** – Retrospective Chart review (32 cases): **Excessive lateral condensation forces caused VRFs: 84.38%**

**Do warm Gutta Percha techniques damage the PDL?**

1. ***Sweatman/Baumgartner JOE 2001*** – Ex vivo (max centrals), *thermocouples* @ levels 0, 2, 4, 6 mm from apex. Evaluated change in radicular temp. (internal, external) using *System B (200, 250, 300° C)* and *Obtura II (185° C)* to *4-5 mm from apex*. **At no level did external root temperature increase more than 10° C**.
2. ***Gutmann JOE 1987*** – In vitro/in vivo (dogs). Max temp change with Obtura thermoplast GP was 1.1° C. No inflamm/necrosis in PDL. **In Vivo, PDL microcirculation ↓ rise in external root surface**
3. *Lipski JOE 2006* – In vitro, Mand/Max Incisors, Obtura II (160°), *Thermocamera*; Outer root surface: Max: 8.5° C, **Mand: 22.1° C** (Caution in thin roots!)
4. ***Erikkson/Albrektson*** - **>10° C** is threshold for bony necrosis

**What do you know about Thermafil?**

1. ***Juhlin/Walton JOE 1993*** – In vitro (plastic blocks), curved canal preparations, *SEM/stereomicroscopic evaluation of fill* – **Most variations in apical 1/3rd – Stripping of gutta percha from carrier, lack of sealer, Length control problems**
2. ***Baumgartner JADA 1995*** – Lateral vs Thermafil – **Thermafil leaked more**, maybe due to **stripping of carrier** upon insertion.
3. ***Gutmann IEJ 1993*** – Thermafil looked better on radiograph than lateral condensation but caused ***more overextensions***.
   1. **Overfilling** was common problem with Thermafil
   2. Lat condensation had more leakage at 7 days, but no difference at 24 hrs and 5 months.
4. *Wilcox* – Retreatment of Thermafil

**How do you sterilize Gutta Percha points?**

1. ***Senia JOE 1975*** – Gutta percha points may be sterilized by a **one minute** immersion in 5.25% NaOCl.
2. *Frank JOE 1983* – NaOCl 5.25% killed spores in 1 minute
3. ***Subha JOE 2013*** – CHX for 1 minute rapid sterilization of GP cone

**How long for sealer to set?**

**ZOE:**

1. *Allan/Walton* – *Roth’s not completely set at 8 weeks*; 4 weeks (Tubliseal)

**Resin epoxy (hydrophobic):**

1. *Allan/Walton* - 4 weeks (AH26)

**CaOH2:**

1. *Allan/Walton* – 4 weeks (SealApex)

**Compare Resilon vs. Gutta Percha**

Resilon = Polyester based root canal filing material (*dimethacrylate polymer + Bioactive glass + CaOH2*)

*Tay/Pashley 2007* – Review of Monoblock theory

**Monoblock:**

**Yes**

1. ***Shipper/Trope 2004*** – Bacterial leakage model, Resilon/Epiphany superior to Gutta Percha/AH26 or GP/Epiphany

**No**

1. ***Gesi/Pashley 2005* –** compared interfacial strengths of Resilon/Epiphany and Gutta Percha/AH Plus using push out test. *GP/AH Plus group exhibited significantly higher interfacial strength than the Resilon group*. GP/AH Plus failed at the GP/sealer interface, Resilon failed at sealer/dentin interface. *Low interfacial strengths in both groups challenges the concept of strengthening root filled teeth with resin materials.*
2. ***Raina/Tay/Pashley 2007*** – Resilon/Epiphany sealed 17 mm root canals as well as GP/AH plus and **does not create a monoblock root filling**
3. ***Kim/Tay 2010* – Review of methacrylate resin sealers**. No benefit of methacrylate based sealers in conjunction with adhesive root filling materials

**Obturation materials**

1. Gutta Percha
2. Resilon/Real Seal – Polyester polymer (resin methacrylate, bioactive glass, CaOH2)
3. Carrier based
4. C-Point – Polyamide polymer w/nylon core – “self sealing”
5. Bioceramic – Calcium silicate/Calcium phosphate

**Sealers**

1. Calcium Hydroxide:
   1. Sealapex
   2. Apexit; Apexit-Plus
   3. Vitapex (CaOH2 + iodoform)
2. Resin epoxy (hydrophobic):
   1. AH-26; AH-Plus
3. Resin methacrylate (hydrophilic): Self-adhesive systems (no etching primer needed)
   1. Real Seal
   2. EndoREZ
   3. Epiphany
4. Zinc Oxide Eugenol:
   1. Roth’s (511/515- slow/med set silver, 801/811- slow/med set stainless)
   2. Tubliseal/Tubliseal EWT
   3. Kerr/Kerr EWT
5. Bioceramics (Calcium silicate/Calcium phosphate):
   1. BC Sealer
6. MTA Silicate Resin (Dicalcium silicate, Tricalcium silicate, Tricalcium aluminate):
   1. MTA Fillapex
7. Glass Ionomer:
   1. Ketac Endo

**Sealers**

1. *Brown JOE 1994* – Roth’s (ZOE) displayed less apical leakage than Ketac endo (glass ionomer) in a vacuum dye test.
2. *Weiss JOE 1997* – Ketac Endo possesses a short-acting very potent and diffusable antibacterial activity, whereas Roth’s extends its effect over 7 days after setting.
3. ***Mickel JOE 1999*** – Roth’s sealer had *better antimicrobial activity* than 3 CaOH2 sealers.
4. ***Kontakiotis/Wu/Wesselink IEJ 1997*** – Evaluated Sealer thickness. Compared 0.05 mm (thin) vs 0.3 mm (thick) before and after 2 yr water storage in root specimens. **Thick layers of ROTH and Pulp Canal Sealer EWT leaked more than thin layers**. No difference for AH-26, SealApex, Ketac-Endo. After 2 yrs, all sealers leaked more than before storage in water, with Pulp Canal Sealer EWT leaking more than others.

**Sealers**

1. ***Allan/Walton JOE 2001*** – *AH26, Sealapex, and Tubliseal were partially set after 1 wk and set was* ***complete after 4 wks****.* ***Roth’s was very slow, as none were completely set at 8 wks****.* Sealers on the glass slap set much more rapidly. In conclusison, under simulated clinical conditions, *sealers set slowly (particularly Roth’s)* and were more delayed than when tested in vitro.
2. *Hume 1984* – Cytoxic effects of ZOE as pulp capping agent: Eugenol is cytotoxic, neurotoxic. Diffuses through dentin. Fresh not set form.
3. ***Leyhausen JOE 1999*** – Genotoxicity and Cytotoxicity of resin-based sealers (AH-Plus, AH-26)
   1. **AH-Plus: Slight to no cellular injuries, No mutagenicity**
   2. **AH-26:Cytotoxic** due to ***formaldehyde*** release, not in AH Plus
4. *Langeland* – All sealers toxic when mixed – **reduced on setting**

**Is sealer extrusion a concern?**

1. *Bernath IEJ 2003* –
   1. Apex & Grossman’s (ZOE) = no periapical inflammation if confined to canal
   2. AH-26 & Endomethasone = + periapical inflam even if confined to canal
   3. All 4 initiated periapical inflammation if overfilled
2. ***Baumgartner/Svec JOE 1983*** - Extrusion of sealer or gutta-percha was associated with ***increased pain in vital cases w/o AP***. Overall incidence of post-obturation pain was 47.6% (14% Severe)
3. ***Augsburger/Peters JOE 1990*** – *Extruded sealer* *did NOT prevent healing*, was removed from apical tissues over the **6 year** follow-up period.

**Is sealer extrusion a concern? continued**

1. *Loushine 2011 –* All sealers tested (AH Plus, Endo Rez, BC Sealer) were initially cytotoxic. AH Plus became non-cytotoxic over 6 week period while BC Sealer remained moderately cytotoxic.
2. ***Ruparel/Diogenes 2014*** – Effect of Endo sealers on Trigeminal neuronal activity. *ZOE (fresh/set forms) and AH-Plus (fresh form) evoked significant CGRP release* while **EndoSequence BC and RealSeal Sealer reduced CGRP release**. Eugenol alone evoked 7.5x inc in CGRP release. \*\*Periapical extrusion of sealer (esp ZOE) may lead to **direct activation of nociceptors/neurogenic inflammation/central sensitization/chronic pain**
3. *Huang 2002* – *Calcium hydroxide sealers (SealApex)* were less cytotoxic than ZOE sealers and Resin epoxy sealers (AH-Plus, AH-26) for Human PDL cells

**Discuss Sargenti paste**

**AAE Position Paper** (Sargenti paste, N2, RC2B)

1. Sargenti technique – No rubber dam needed, Length – somewhere near apex, objective is *chemical fixation (not clean and shape)*, opposes irrigation, suggests trying to keep N2 paste in canals but it is “well tolerated” in PA tissues. **4-7% paraformaldehyde, contains lead tetroxide.**
2. ***Newton 1980*** – Monkey study, demonstrated at *6 mo and 1 yr* that teeth obturated with N2 technique and RC2B paste developed**severe apical pathosis** – **Granuloma, Abscess, or Osteomyelitis**
3. ***Spangberg 1974*** – **Formaldehyde** is responsible for **extensive tissue necrosis**. **Not resorbable** therefore must be surgically removed if expressed beyond apex
4. *Hata/Toda 1989* – *Systemic distribution* of 14C labeled formaldehyde: blood, lymph nodes, liver, kidneys, spleen
5. *AAE Paper* – Destruction of periapical tissues, paresthesia, dysathesia, pain

**What internal matrix material can be used when repairing a perforation?**

1. Hydroxyapetite - Lemon
2. DFDBA - Hartwell
3. Gelfoam – Walia, Hartwell
4. CaOH2 – Peterson, Frank & Weine
5. Collacote – Rosenberg
6. Calcium phosphate – Chau
7. Calcium sulfate – Alhadainey

*Torabinejad OOO 1996* – Case report: MTA used in furcation perforation, no internal matrix is recommended.

*Baumgartner JOE 1998*– Perforations repaired with MTA leaked less than amalgam

*Saunders JOE 2002* – MTA leaked less than those repaired with Vitrebond

**Discuss lateral root perforations**

1. ***Fuss/Trope 1996*** – REVIEW article on Root Perforations:

Prognostic Factors:

* 1. **Time** – Immediate repair ↑ success (*Seltzer 1970*)
  2. **Size** – Smaller the perf, ↑ success – ability to seal
  3. **Location** - *#1 factor* – *Critical zone = Level of crestal bone/connective tissue/epithelial attachment* – Lowest success due to bacterial infection/epithelial downgrowth/sulcular communciation → bone loss (perforation above or below this level is better!)

See also *Jew; Mente (86%), Krupp/Hulsmann (73%, 2 prognostic factors – Pre-op RL associated with perforation and Sulcular communication)*

Goals: Arrest inflammatory rxns, Preserve or promote regrowth of tissue attachment adjacent to perforation, Prevent bacterial infection (and subsequent downgrowth of epithelium)

**MTA & Perforation Repair**

1. ***Holland JOE 2001*** – **No inflammation**was seen and**cementum was deposited over MTA** in this dog study of lateral root perforations
2. *Main/Torabinejad 2004* – Retrospective, **Immediate** MTA & Perforation Repair – **16/16 (100%)** healed at 12-45 months post-repair with MTA: 5 lateral, 5 strip, 3 furcal, and 3 apical perforations
3. ***Mente JOE 2010*** *-* Retrospective, **Immediate** MTA root perf repair (*Furcal, Crestal, Mid root, Apical root*): Success: **18/21** (**86%**) healed at ≥1 year. No Predictors for success. Healed = No clinical/radiographic signs/symptoms, PAI ≤ 2.
4. ***Krupp/Hulsman 2013*** – Retrospective, MTA perf repair (90 teeth), Success: **73%** at ≥1 year. **Prognostic predictors: Pre-op RL associated w/ perforation and Sulcular communication with the perforation**

**Geristore & Perforation Repair**

1. ***Dragoo 1997***– Geristore (compomer) as a **subgingival root repair** material promoted healing with ***epithelial and connective tissue attachment***
2. *Hammad/Al-Omairi 2011* – In vitro (rat CT implentation), GMTA>Geristore for severity of CT inflammatory reactions at 1 wk – 1 month
3. ***Gupta* *2013***– In vitro (cell culture), Geristore >MTA for viability & attachment of **PDL fibroblasts**

**Post-Obturation**

**Should we use orifice sealers after obturation? What material ?**

1. ***Saunders IEJ 1997*** – *Vitrebond* is an effective barrier for preventing microleakage in the pulpal floor.
2. *Wolcott JOE 1999* – Pigmented Vitrebond glass ionomer cement fulfills the criteria for an ideal barrier better than Ketac-bond or GC America barriers
3. ***Wolanek JOE 2001*** – *Clearfil (composite) barrier* showed **no leakage**, No barrier group showed positive bacterial penetration in **15 to 76 days**. NOTE: Eugenol containing sealer had no effect on the bonding agent.

**Do posts cause VRFs?**

1. *Obermayr*
2. ***Peters*** – VRF from occlusal loading of posts
3. *Ross* 1980 – Carbon fiber, Parallel sided posts least likely to cause VRFs
4. ***Tamse/Fuss JOE 2001*** –**Etiology of VRFs**: **Post placement** (**Screw posts, tapered cast posts**), Lateral condensation
5. ***Dean/Jeansonne JOE 1998*** – *Carbon post/composite bu teeth* had NO root fractures while *parallel and tapered metal post* groups had root fractures upon 45 deg loading

**How much room should be left for a post space?**

1. *Neagley OOO 1969* & *Madison JOE 1984* - Agree that at least **4 mm** of apical gutta percha should remain following post space preparation.
2. *Mattison J Prosthetic Dentistry 1984* – **5-6 mm** of gutta percha is necessary for an adequate apical seal. Use rotary inst to remove GP.
3. ***Goerig J Prosthetic Dentistry 1983*** – Post should be **2/3 the length of the root** and **10-15 mm in length**, leaving at **least 4-5 mm of gutta percha** apically. Posts should be parallel and cemented, not screwed.
4. ***Baba/Goodacre 2009****:* Leave **4-5 mm** gutta percha for an apical seal

**Posts & Ferrule:**

1. ***Nissan 2008*** – When **ferrule is 2mm, Post length is non contributory**

**Should you place a post immediately to avoid leakage?**

**YES**

1. ***Wu/Wesselink 1998****;* ***Metzger JOE 2000*** –Both agree; Post prepared canals (apical 4mm gp) have inferior seal, *post and core should be immediately* completed after root canal treatment.
2. *Sato JOE 2002 & Fox IEJ 1997* - Both agree; Permanently cemented, prefabricated post and core produced the best seal; leakage was significantly greater with the temporary post/crown.
3. ***Barrieshi/Walton*** – Obturated/post spaced prepared canals – unsealed – apical penetration by ***90 days*** – *recommends immediate restoration*
4. *Fan/Wu/Wesselink 1999* – Immed. Post space leaked less than delayed post space preparation

**NO**

1. *Lemon JOE 1981* – NSD in apical leakage with immediate vs delayed post space preparation.

**What technique is best for making a post space?**

1. *Todd 1983 J Prosthetic Dentistry* – NSD between heat, Peeso, and Gates on apical seal. 4 mm apical seal is recommended for less leakage.
2. ***Mattison 1990 J Prosthetic Dentistry*** – Significantly less leakage was observed with the **heated plugger technique** at the 3 mm and 5 mm levels when compared to both the GPX and the Gates-Glidden groups.

**Does Ferrule affect root fracture?**

1. ***Sathorn* 2005** - Inadequate ferrule increases chance of root fracture
2. ***Tan 2005*** – Uniform **2mm** **ferrule** increases *root resistance to fracture*
3. ***Nissan 2008*** – When **ferrule is 2mm, Post length is non contributory**

**Does eugenol in sealer affect the retention of the post?**

**YES**

1. ***Nemetz 1992*** – *Residual eugenol* in the canal substantially decreased retention of Paraposts luted with *Panavia* composite resin cement. **Irrigation with Ethyl Alcohol or Etching 37% phosphoric acid restored the retention.**

**NO**

1. ***Hagge IEJ 2002*** – *Kerr EWT, AH-26 and Sealapex did not affect the retention of endodontic posts luted with Panavia cement*; therefore **eugenol avoidance is unnecessary** when selecting sealers.
2. ***Schwartz/Walker 1998 JOE*** – *Type of sealer (Roth’s or AH-26) had no effect on post retention with either cement* (ZnPO4 or Panavia). Post retention was significantly greater with the zinc phosphate cement than the resin cement.
3. *Boone 2001 JOE* – NSD in retention between types of sealer or post cementation times with Panavia. The mechanical removal of the sealer-impregnated dentin from the canal walls during post-space prepartation is a critical step in achieving optimum post retention when resin cement is used.

**Does a post put stress on the tooth or cause tooth fractures?**

1. ***Tamse/Fuss 1999*** – VRFs caused by posts, lateral condensation
2. ***Peters*** – VRF from occlusal loading of posts
3. ***Dean/Jeansonne JOE 1998*** – *Carbon post/composite bu* teeth had NO root fractures while parallel and tapered post groups had root fractures upon 45 deg loading

**What is in Cavit? What are it’s properties? Does it seal?**

1. ***Widerman JADA 1971*** – Cavit has **twice the linear expansion** and **half the compressive strength of ZOE**; Composition of Cavit =
   1. **Calcium sulfate**
   2. Glycol acetate
   3. Triethanolamine
   4. Polyvinyl acetate
   5. Polyvinylchloride acetate
   6. Red pigment
2. ***Webber 1978 OOO*** – A **3.5mm thickness of Cavit** should be used in order to prevent leakage.
3. *Bobotis/Pashley 1989 JOE* – Cavit, Cavit-G, TERM and glass ionomer cement provided leakproof seals during the 8 wk testing period. IRM did not.

**What is in Cavit? What are it’s properties? Does it seal? Cont.**

1. *Eleazer 2001 JOE* – Cotton trapped between the wall of the tube and the filling material dramatically reduced the sealing quality of the temporary restoration.
2. *Stark 1990 OOO* – Cavit had the best sealing ability, IRM showed the maximum dye penetration.
3. ***Beach/Hutter JOE 1996*** – Cavit provided a bacterial leakage-free seal for **3 wks**.
4. *Mayer JOE 1997* – Cavit showed less leakage in the dye penetration test and fewer marginal crevices.
5. *Deveaus JOE 1999* – In-vitro leakage test – cavit leaked less than TERM, IRM, and Fermit.

**Is Glass Ionomer superior to Cavit or IRM?**

**YES**

1. ***Barthel*** ***JOE*** ***1999*** – In vitro, *Bacterial leakage (turbidity) study* – Glass ionomer >> IRM > Cavit; **At 30 days only G.I. prevented bacterial leakage**
2. ***Seiler AGD 2006*** – In vitro, *Bacterial leakage (turbidity) study* – Glass Ionomer (GI) and Resin modified Glass Ionomer (RMGI) >> ZOE (IRM) for sealing capabilities

**Is TERM superior to Cavit or IRM?**

**YES**

1. ***Anderson/Pashley 1989*** – In vitro, *fluid filtration study,* **Multisurface temporary restorations**: **TERM** statistically superior seal compared with Cavit and IRM
2. *Bobotis/Pashley* – In vitro, 8 wks fluid filtration, TERM = Cavit > IRM

**Are Endodontically teeth more brittle ?**

1. ***Sedgley JOE 1992*** – *Vital dentin was 3.5% harder than endodontically treated dentin* however the biomechanical properties of endo treated teeth and their contralateral vital pairs indicates that **teeth do NOT become more brittle following endodontic treatment.**
2. *Huang 1991* – Neither NSRCT nor dehydration decrease the tooth’s physical properties
3. ***Papa/Messer*** - **Dentinal moisture content**: Vital teeth: 12.35%, Non-Vital teeth: 12.10%. **NSDs**
4. *Panitvisai* – NSRCT teeth have greater cuspal deflection than those without

**Is cuspal coverage important for endodontically treated posterior teeth?**

1. ***Aquilino/Caplan 2002*** – Chart review - Endodontically treated teeth NOT crowned after obturation were lost at a **6.0x greater rate** than teeth crowned after obturation
2. *Salehrabi/Rotstein* – 97% Survival, 3% that did not were more often not crowned
3. ***Linn 1994 JOE*** – Endodontically treated **molars** are considered *susceptible to bulk fracture because of loss of tooth bulk*. It is **more important to cover cusps than to preserve tooth structure** (including a marginal ridge)in endodontically treated molars**.**

**Effect of Coronal Restoration/Leakage**

1. ***Ray/Trope*** – *Coronal seal more important than quality of RCT*, Xray only,

Healed: GE/GR: 91.4%, PE/GR: 67.6%, GE/PR: 44.1%, PE/PR: 18.1%

1. ***Gillen 2011*** – *Meta-analysis* – ≥1 yr recall, Majority radiographic recall only. *Odds ratios*: AR/AE 2.8x more likely no AP than IR/AE, AR/AE 2.7x more likely no AP than AR/IE. *Equal odds for healing with both scenarios*.
2. ***Ng*** – Quality of Coronal restoration Sig. effects success of NSRCT/RETX
3. ***Swanson/Madison***– *dye leakage*, loss of coronal seal/coronal microleakage led to contamination of obturated canal w/in **3 days**
4. ***Khayat/Torbinejad*** – *bacterial leakage* – No coronal seal, obturated - complete contamination of entire length w/in **30 days** of loss of seal
5. ***Magura/Newton*** – dye leakage/**histo eval** - Recommends **retreatment if exposed (or IRM remains) for 3 months** (clinically significant leakage)
6. ***Barrieshi/Walton*** – Mixed anaerobic community developed/exposed to obturated/post space prepared canals – SEM analysis- **apical penetration by 90 days** - recommends immediate restoration of post space canal (*Wu/Wess.*)

**Non-vital Bleaching. Does it cause resorption? How?**

1. ***Spasser 1961;Nutting/Poe*** – Sodium perborate – **“walking bleach”** technique: seal cotton pellet soaked in a mixture of *Superoxol and Sodium perborate* in the access cavity of the tooth for a period of 4 to 7 days. Superoxol is a strong oxidizing agent which breaks down the darkly pigmented macromolecules into smaller lighter colored molecules.
2. ***Harrington/Natkin JOE 1979*** – 7 cases of external cervical root resorpiton. Theory: **Superoxol (+ heat)** seeps through patent dentinal tubules and initiates an inflammatory resorptive response in the cervical area.
3. *Lado 1983* – Theory: 30% H2O2 denatures dentin exposed at the cervical margin, inciting an inflammatory response by clastic cells within the PDL.

**Non-vital Bleaching. Does it cause resorption? How? Continued**

1. *Cvek EDT 1985* – Theory: damage to the periodontium, caused by the bleaching agent at the time of treatment, may heal or be followed by ankylosis. When the situation is complicated by bacterial contamination of the gingival sulcus, progressive inflammatory changes in the periodontium is possible.
2. *Pathways of the Pulp* **–** Walking bleach results in cervical root resorption 6-8%, if heat is used to activate the superoxol the rate rises to 18-25% (Pathways).
3. *Madison & Walton JOE 1990* – Theory: resorption occurs when heat is used by driving the Superoxol through the dentinal tubules, thereby directly altering the cementum.

**Non-vital Bleaching. Does it cause resorption? How? Continued**

1. ***Heithersay EDT 1997* –** *Hydroxyl radical* was generated after thermocatalytic bleaching w/ 30% H2O2. This radical may be one mechanism underlying PDL breakdown and resorption after bleaching.
2. ***Rotstein*** - Internal bleaching & ECR – 30% H2O2 leakage through dentinal tubules at CEJ with no cemental layer – exposed mineralized dentin – *damages dentin, initiates inflammation* and *external cervical root resorption*
3. ***Papadopoulos EDT 1996*** – All CEJ junction types showed leakage of H2O2 from the chamber, but teeth **with gaps (10%) at the CEJ** had *higher leakage values* compared to the other 2 types.
4. ***Neuvald JOE 2000*** – CEJ Configuration of Cementum/Enamel joint – 60% overlap, 30% butt joint, **10% Gap**

**Regression of Internal Bleaching**

1. ***Ho/Goerig 1989***– 4% Color Regression after 6 months
2. *Abbott 2009* – 87% Good color change, 13% Acceptable color change; 4% Regression after one year

**Success of Internal Bleaching**

1. ***Rotstein/Friedman JOE 1993*** – Compared Internal bleaching prognosis of Sodium perborate + H2O vs Hydrogen Peroxide. Ex Vivo study, Sodium perborate mixed with: 30% H2O2, 3%H2O2, or H20. 1 year follow up. Results: **Sodium perborate + H2O = Sodium perborate + 30% H2O2 (100% maintain color)**

**Preventing Resorption**

1. ***West JOE 1994*** - Proposed the location and shape of an intracoronal bleach barrier
2. ***Rotstein JOE 1992*** – Bovine/human teeth – evaluated radicular penetration of 30% H2O2 based on thickness and material for barrier. Recommmends placing a **2 mm protective base** (Glass ionomer) **to the CEJ level to avoid radicular penetration of H2O2**

**Can You Bleach tetracycline stained teeth? (Intrinsic stain)**

1. *Abou-Rass JOE 1982* – Found intentional RCT and internal bleaching is sometimes an effective treatment for tetracycline stains when other methods cannot be applied.
2. ***Walton JOE 1982*** – External bleaching is ineffective long term for tetracycline stains but internal bleaching is effective.

**How does bleaching affect restoration of the tooth?**

1. ***Torneck JOE 1993*** – Bond strength (resin polymerization) is adversely effected by bleaching
2. ***Silva IEJ 2001*** – Microleakage increased as a function of bleaching, short term use of CaOH2 after bleaching decreased microleakage

**Retreatment**

## Causes of Persistent AP

## *Nair 2006*

## Intraradicular Infection

## Extraradicular Infection

1. Foreign Body Rxn
2. Apical Cyst (True cyst)
3. Cholesterol Crystals
4. Apical Scar

## Persistent AP & Extraradicular Infection

**Extraradicular Infection Rare (outside of AAA/CAA):**

1. *Sjogren, Sundqvist et al 1988 IEJ* – **Propionibacterium propionicum** may be implicated in Extraradicular infection
2. ***Nair 1984 JOE*** – **Actinomyces israelii** is able to establish Extraradicular infection
3. *Ricucci/Siqueira* 2010– 106 roots w/ AP (42 previously treated) Intraradicular biofilms present: *Cysts 95%, Abscesses 83%, Granulomas 70%;* Extraradicular biofilms 6%. No correlation between biofilm presence and clinical symptoms or sinus tracts; Intraradicular biofilms are responsible for the AP (CAGE)

**Extraradicular Infection Common:** See also *Tronstad/Barnett (biofilms)*

1. ***Sunde/Tronstad JOE 2002***- Microbiota of Periapical lesions refractory to endodontic therapy: *AAP or CAA cases* – Sampling of Periapical lesion during surgery. **35/36 positive for extraradicular infection**. 51% Anaerobes, 79% Gram +, 148 microbial strains, Avg. 4.1 strains/case

## Retreatment

## *Van Nieuwenhuysen 1994* – Radiographic monitoring (6 years) of asymptomatic previously treated teeth (n=420) with radiographically deficient filling and no/small PA lesions: Stable: 94.8%, Healing: 2.4%, Failure: 2.8%

## *Gorni/Gagliani 2004* – 451 patients, 2 year recall, *Friedman* healing categories (Healed, Diseased), Healed + Healing = Success

## Root Canal Morphology Respected (RCMR) – calcifications, apical stop, broken instruments, short fill

## Root Canal Morphology Altered (RCMA) – transportation, perforation, stripping, internal resorption

Findings: Overall success: 69%, **RCMR: 87%, RCMA: 47%**

1. ***Hoen/Pink 2002*** – 337 Retx teeth, **89% of teeth with asymmetric obturation** had **additional canal located during Retx**

## How do silver points cause a problem?

***Seltzer et al*** –

1. Silver wires removed from failed endodontic cases showed ***corrosion products of silver sulfate*** products which are **cytotoxic**
2. Leakage from around the round wire within a non-round canal causes *washout of the cement* and *fluid contact with the silver wire*
3. ***Oxidation***of the wire leads to the ***corrosive byproducts – silver sulfate***

Leakage → oxidation → corrosion/byproducts (silver sulfate) → cytotoxic

**What are some techniques to remove separated instruments or silver points?**

1. *Hulsmann* 1993 EDT – Recommends using the needle sleeve tech, endo extractor, braiding Hedstrom files, Masserann kit, ultrasonics, Gonon post remover
2. ***Krell* 1984 JOE** – Recommends ultrasonic application and Hedstrom files for silver points
3. ***Ruddle*** ***JOE 2004*** – Staging platform w modified gg; ultrasonics; **never around curve**; if unable w/ ultrasonics, try IRS system, tube/glue system, or bypass w/files
4. ***Nevares JOE 2012*** – Success of instrument removal/bypass: **Fragment visualized: 85%, Fragment not visualized: 48%**
5. ***Iqbal* –** Staging platform most centered with lightspeed when prepared in the **apical 1/3rd**

**How do you remove posts?**

1. ***Berber*t *IEJ 1995*** - Reduced forces were necessary to remove the posts that were treated with an Ultrasonic device compared with posts which did not receive ultrasonic treatment.
2. ***Baumgartner* JOE 1997** – Takes longer to remove post with ultrasonic forces than with the Gonan system. Ultrasonic system induces more cracks than the Gonan system (but NSD)
3. ***Ruddle JOE 2004* – Review –** *Evaluate the curvature and circumferential diameter of root and post type* (parallel vs tapered, active vs non-active, metallic vs. non-metallic) in determining ability to remove post. Post removal with ultrasonics and Ruddle Post Removal System (trephine/tap/extractor pliers)
4. ***Dominici/Eleazer*** – High root temps with ultrasonic > 15 s --- use water coolant

**Is Chloroform safe for retreatments ?**

1. ***McDonald* 1992 JOE** – In vivo, Chloroform is **safe for the dentist/staff**. Air vapor levels were well below OSHA mandated levels (2 ppm/8 hrs).
2. ***Chutich* 1998 JOE** – In vitro, **No health risk to the patient**, amount of chloroform *expelled thru the apex* (0.32mg) is several orders of magnitude below the permissible toxic dose (49mg)
3. *Rotstein 1999 OOO* – chloroform may cause a significant softening effect on both enamel and dentin. This softening is already apparent after 5 minutes of treatment.

**Is Chloroform antimicrobial?**

1. ***Edgar/Baumgartner*** – *60% reduction in E. faecalis* using Chloroform in Retx (11/17 neg cultures Chloroform, 0/17 neg cultures Saline)

**Are other means available to remove gutta-percha?**

**Can it be removed completely?**

1. ***Kaplowitz* 1990 JOE** – tested 5 solvents (halothane, xylene) but found *chloroform* was the only one that completely dissolved the gutta-percha
2. *Schafer* 1987 OOO – tested chloroform vs eucalyptus oil, chloroform was far more effective.
3. *Krell 1987 JOE* – Evaluated 4 methods to remove root canal filling, AH26 was more difficult to remove than Roth’s. All methods left some debris on the canal walls.
4. *Wilcox 1991 JOE* – Retreating ones own failures is unlikely to debride areas previously undebrided because reinstrumentation usually enlarges in the *same directions as the first instrumentation*.

**Are other means available to remove gutta-percha?**

**Can it be removed completely?**

1. ***Hansen 1998 JOE*** – tested several solvents to remove different sealers. Only *chloroform* removed **AH-26** (epoxy resin sealer)
2. *Metzger* 1995 JOE – presented procedure for *removal of overextended root canal filling* – extend file 0.5-1.0 mm beyond apex/engage gp
3. ***Saad/Al-Hadlaq 2007 JOE*** *–* Compared K3 & ProTaper NiTi files to Hedstrom hand files for gp removal in retx procedure
   1. All techniques left some gp/sealer remaining
   2. *K3 & PT removed* ***significantly******more gp/sealer*** *than Hedstroms*
   3. *K3 & PT instrumentation took* ***significantly less time***
   4. No difference in apical extrusion among all groups

**How do you remove Thermafil ?**

Tulsa recommends: work down around carrier with small files and solvent until it is free. If Thermafil Plus was used, a rotary file can be used to engage the vent in the carrier. Ultrasonics may also be useful. Thermafil Plus has a groove in the core to vent GP during placement and to ease retreatment.

1. ***Bertrand* 1997 JOE** – Use *chloroform and hand files* (k-files, hedstroms) to remove Thermfil carriers
2. *Baratto 2002 IEJ* – 0.04 Pro Files were used to remove Thermafil plastic carriers at 300 rpm in a crown down manner. Unable to remove all gutta-percha from canals
3. ***Wolcott/Hicks* 1999 JOE –** Tested the application of a *System B at 225 degrees C (carrier melts at 300)*. Recommend heat and insert system B tip 10-15mm for 5-8 sec, then k-files (#30-50) on either side of the carrier with apical pressure and counter clockwise rotation.

**What is Resorcinol-formaldehyde resin “Russian Red”?**

1. ***Schwandt 2003 JOE*** – A material used is many foreign countries. Contains **two toxic components, resorcinol and formaldehyde**. Forms brick hard red material that has **no solvent**. Requires *no instrumentation, presumably “fixes” tissue and kills bacteria apical to paste level*.
2. ***Vranas/Hartwell*** *2003 JOE* – This study tested the effectivness of 0.9% NaCl, 5.25% NaOCl, chloroform, or Endosolv R on softening Resorcinol disks. Evaluated depth of probe penetration. *NaOCl was superior to all other groups after 5 minutes*. No solvent clinically effective.
3. ***Gambrel/Hartwell*** *2005 JOE* – In vitro – resorcinol/formalin paste fills and extracted teeth – no solvent (same as above) clinically effective at softening paste. *EndoSolv R* was stat. superior for probe penetration but this *did not translate into a clinical effect*.

**Surgery**

**Anatomical considerations during periapical surgery**

**SINUS**

1. ***Eberhardt/Torabinejad 1992 OOO*** – Distance of Apices of Maxillary posterior teeth and floor of maxillary sinus measured (CT scan): *MB root of Max 2nd molar is closest to the sinus (1.97mm avg)* but farthest from the buccal bony surface (4.45mm avg). *Max 1st bicuspid is closest to the B plate (1.63mm avg)* but farthest from the floor of the sinus (7.05mm). 5% of apicies protrude into the sinus.
2. ***Lin & Langeland 1985 JOE*** – Recommend the use of *nasal decongestant* in the event of a *sinus perforation (0.5% neosynephrine)*. Add antibiotics **only if acute sinusitis develops**, do **not** give prophylactically.
3. Rud 1998 JOE – Sinus perforations occur in half of all cases studied. Results of this study support the use of antibiotics based on case need, NOT prophylactically.

**Anatomical considerations during periapical surgery**

**SINUS**

1. ***Waztek/Bernhart DCNA 1997*** – *Sinus perforations* occurred in **28%** of maxillary posterior endodontic surgeries. *No difference in healing.*
2. ***Kretzschmar*** – Give 10 day course of *Nasal Decongestants/Oral Antihistamines* (*Neosynephrine/Pseudophedrine*) and *Amox 500 mg q6h* for iatrogenic sinus exposure (opposes ***Lin/Langeland*** for antibiotic coverage)
3. ***Mailet/McClanahan*** – ~**50%** of cases of Maxillary sinusitis identified on *CBCT were odontogenic in origin*. Avg. mucosal thickening = **7.4 mm**. Maxillary 1st and 2nd molars were **11x** more likely involved than Maxillary 1st or 2nd Premolars. *P root of Maxillary 1st Molars and MB root of Maxillary 2nd Molars* were most likely associated with Maxillary sinusitis.

**Mental Foramen**

1. *Moiseiwitsch* 1995 JOE – This study described 3 steps to minimize risk of damage to the neurovascular bundle exiting the mental foramen
   1. Take vertical periapical film
   2. Use triangular flap with the vertical releasing incision distal
   3. Make a groove in the bone superior to the foramen to prevent retractor slippage
2. ***Phillips* 1990 JOE** – Most common location of the mental foramen:
   1. Inferior to the mandibular second bicuspid
   2. ***60%*** *of the distance from the buccal cusp tip to the inferior border of the mandible*.
   3. It exits in a posterior and superior direction

**Mandibular Canal**

1. ***Denio/Torabinejad* 1992 JOE** – Anatomic relationship of Mandibular canal to Posterior teeth apices – 2nd Molar/2nd Premolar most problematic:

Distance from Root apex to Canal:

**2nd Molar: 3.7 mm**

1st Molar: 6.9 mm (~**7.0 mm**)

**2nd Premolar: 4.7 mm**

Canal Path:

***S-shaped****:* ***31%*** - Buccal to the distal root 2nd Molar, crosses to lingual below mesial root 2nd Molar, runs lingual to 1st Molar, then crosses to buccal below apex of 2nd Premolar

Lingual: 19%, Buccal: 17%, Directly below: 5%

1. ***Koivisto 2011 JOE*** – **CBCT** analysis of proximity to mandibular canal. Mandibular 2nd Molars closest to IAN. Mesial roots of Mandibular 2nd Molars closest to IAN in Females and young pts (< 18 y.o.).

**Discuss Flap Design for Surgery**

1. ***Kramper/Kaminski 1984 JOE*** – Dog study
   1. **Submarginal incision** is the flap of choice when not contraindicated by anatomical location of the lesion (**Incision should be placed over bone**) or by insufficient attached gingiva (**Attached gingiva must be 3 mm wide between base of sulcus and incision**)
   2. Intrasulcular: Greater Inflammation, *bone loss and recession*
   3. Semilunar: Greater Inflammation
2. ***Velvart 2002 IEJ*** – **Papilla based incision** – allows rapid and predictable *recession-free healing* following surgical exposure of the soft tissues. Soft tissue management in surgery.
3. ***Kim/Kratchman*** – Recommends *Intrasulcular or Submarginal (mucogingival)* flap design; Also discusses Velvart’s papillary based incision

**Discuss root end resection. How far should you resect? Bevel?**

1. Pathways of the Pulp 8th edition - When 3mm of the apex is resected 93% of lateral canals are removed. Additional resection reduced the percentage insignificantly as per Vertucci. A root resection of 3mm at a 0 degree bevel angle removes the majority of anatomic entities that are potential causes for failure.
2. ***Weller/Kim 1995 JOE*** – 50 **Maxillary Molars** **MB root** - **Incidence of Isthmus**: Highest in the **Apical 3-5mm levels (80-100%)**. In teeth with two MB canals the **4mm section** contained a complete or partial isthmus **100% of the time.** *Failure to treat the isthmus may be responsible for endodontic failures.*
3. ***Von Arx 2005 IEJ*** – **Mandibular Molars** – Frequency of **Isthmus**: **M root – 83%**, D root – 36%

**Discuss root end resection. How far should you resect? Bevel?**

1. ***Kim/Kratchman*** – **3 mm resection** – eliminates **98% apical ramifications** and **93% lateral canals (AR/LCs)**
2. ***Tidmarsh/Arrowsmith 1989*** – SEM analysis of resected root ends, due to dentinal tubule communication, **angle of bevel should be minimal** and **retroprep extend at least to coronal end of bevel**
3. ***Gilheany/Figdor 1994 JOE*** – Dye leakage study of depths/angles (0, 30, 45°) for retroprep/fill - Apical leakage may be reduced by resecting at a **0° bevel** and increasing the depth of the retrograde filling . Recommends **retroprep depth** to extend **coronal to pulpal termination of dentinal tubules (1.0 mm for 0° bevel)**
4. *Tetsch* – Round bur best for removing osseous tissue

**Discuss root end resection. How far should you resect? Bevel?**

1. *Morgan/Marshall* – Multipurpose bur best for Root Resection
2. ***Gagliani* 1998 JOE** – An **apical preparation** of **3mm** or more along the vertical axis can produce a safe and effective seal. The bevel should not be greater than the depth of the retropreparation: 1mm for 0° bevel, 2.5 mm for 45° bevel
3. ***Christiansen IEJ 2009*** – *Randomized Clincal Trial*: 44 patients: 2 groups: 1) 3 mm Retroprep/MTA retrofill, 2) Burnished GP w/*No retroprep/fill*. Healing (1 yr follow up): **MTA (1): 96%, GP (2): 52%**. Significance of Retroprep/Fill vs. No Retroprep/Fill.

**Compare ultrasonic and bur root end preparation**

NOTE – Richman was 1st to propose use of ultrasonics (root canal debridement and apicoectomy). Carr popularized it.

1. ***Carr 1997 DCNA*** – Ultrasonic technique satisfies all the major requirements for ideal retropreparations: a *class 1 preparation* at least *3mm into dentin* with *walls parallel* to and coincident with the anatomic outline of the pulpal space.
2. *Engel 1995 JOE* – This study suggests that the ultrasonic handpiece offers better control, preps were more centered on the canal and isthmus, and there was less gouging of the canal walls when compared to the microhandpiece.
3. ***Melhaff/Baumgartner* 1997 JOE** – Ultrasonic preps were **deeper, deviated less from the canal, required less bevel** and **smaller bony crypts**

**Does the ultrasonic tip cause cracks in the root end?**

**YES**

1. *Saunders 1994 IEJ* – No difference in the dye leakage studies until 7 months and then both leaked equally. Cracking was detected most often with the ultrasonically prepared roots
2. *Belini/Morgan/Marshall/Baumgartner 1999 JOE* – In vivo study that found no cracks after the root resection but one incomplete crack after the ultrasonic root-end preparation.
3. ***Abedi/Torabinejad 1995 OOO*** – *SEM/photomicrograph* comparison of ultrasonics vs. burs for root end resection and crack formation. **Higher incidence of crack formation with Ultrasonics**: Crack formation is a function of time, power and dentin thickness.

**Does the ultrasonic tip cause cracks in the root end?**

**NO**

1. ***Brent/Morgan/Marshall/Baumgartner 1999 JOE*** – **SS Ultrasonics – cracks noted, Diamond coated Ultrasonics – no additional cracks**, **eliminated several cracks**, but left heavily abraded, debris-coated cavosurface that may affect the apical seal. Root-end cracks NOT seen w/diamond US.
2. *Bakland 1996 EDT* – Use *medium setting with water spray* on ultrasonic for root-end preps to minimize infractions.
3. *Peters 2001 IEJ* – *No difference between stainless and diamond coated ultrasonic tips* regarding microcracks of root ends. Diamond tips were faster, only one microcrack was seen; incidence is low.

**Discuss hemostasis during surgery**

1. *Gutmann IEJ 1996* – 4 actions of hemostasis with collagen:
   1. Stimulation of platelet adhesion, aggregation, release
   2. Activation of factor XIII (Hagamen) and other clotting factors
   3. Mechanical tamponade
   4. Release of Serotonin (5-HT)
2. ***Kim/Kratchman*** – **Endodontic Microsurgery Review DCNA 1997**:
   1. ***Bone wax***

*Aurelio* **–** Acts mechanically via *tamponade effect*, foreign body reaction may occur if left in surgical site. (Bees Wax)

* 1. ***Chemical vasoconstrictors (epi)***

**Racellets and Epi**: Cotton impregnated w/ racemic epi. (immediate vasoconstriction) little systemic absorption. Placed on bone w/ another cotton pellet and pressure for 2-4 minutes.

**Discuss hemostasis during surgery**

b.***Chemical vasoconstrictors (epi)***

**Racellets and Epi**:

***Vickers/Baumgartner* -** Effective hemostasis**, No significant cardiovascular effects (#3 Racellet** = 0.55 mg racemic epi/pellet)

**Ferric sulfate**:

***Jeansonne/Lemon*** - Agglutination of blood proteins that **occlude capillary orifices**. Cytotoxic and causes tissue necrosis therefore it *must be removed to prevent delayed healing*

***Vickers/Baumgartner*** - Effective hemostasis, **No significant cardiovascular effects**

**Thrombin:**

Not studied for endodontic applications

**Discuss hemostasis during surgery**

c. ***Absorbable hemostatic agents***

**Calcium sulfate**: *Tamponade effect*, biocompatible, *resorbs in 2-4 weeks*

***Scarano 2012***

**Gelfoam**: Animal skin gelatin, promotes platelet disintegration. Stimulates thromboplastin release and thrombin formation. *Not applicable to endo surgery*.

**Collagen:** Causes *platelet aggregation & fibrin formation*. If applied directed w/ pressure = hemostasis in 2-5 mins. *Does not inhibit healing*. ***Vy/Baumgartner*** - **CollaCote+ 2.25% racemic epi – No significant cardiovascular effects,** effective hemostasis

**Surgicel**: Oxidized regenerated cellulose. Acts mechanically by forming a sticky mass when in contact with blood. **Inhibits healing & stimulates Inflammation.** **Not recommended.**

**How much blood is lost during periapical surgery?**

1. ***Messer EDT 1987*** – *Operating time was the biggest factor* influencing blood loss (increased loss with increased time) **Blood loss avg. = 9.5 mL**, range 1.2 - 48.4 mL. (comparable to single tooth extraction)
2. ***Buckley J Perio 1984*** – Study indicated significant reduction of blood loss using **1:50k vs 1:100k epi**. (**50% less blood loss**)
3. ***Lindorf OOO 1979*** – This study discussed the **rebound effect**  (**reactive hyperemia**) following injections with epi.

**Discuss CHX and Surgery**

1. ***Vaughan/Garnick 1989*** – **0.12% CHX** reduced **plaque and gingival inflammation up to 2 weeks** after Periodontal surgery

**Does removing the smear layer on the resected root end improve post surgical healing?**

**YES**

1. ***Craig/Harrison JOE 1993*** - Demineralizing the root end with citric acid **enhances cementogenesis and dentoalveolar healing** in dogs. It acts by exposing collagen fibrils of the resected cementum and dentin. (50% citric acid, pH 1, 2 min)

**NO**

1. ***Jeansonne JOE 2003*** – **NSD in healing or bone fill** when citric acid or tetracycline were used to remove the smear layer (at 9 or 18 days).

1. ***Zhu JOE 2000*** – **Cell adhesion** **of** **osteoblasts is NOT influenced by the existence of a smear layer** or the direction of the dentinal tubules (bevel angle) on the dentin surface.

**Discuss some different retrofilling materials besides SEBA and MTA**

1. *Andreasen JOE 1993* – *Studied composite retrofill*, produced reformation of periodontium including reformation of a lamina dura, inserting Sharpey’s fibers and cementum deposition.
2. *Chong IEJ 1997* – Studied Vitrebond vs ZOE vs Amalgam. Best results with *Vitrebond* in dog teeth.
3. ***Olsen JOE 1994*** – Materials study impanted in rats – IRM, Amalgam and EBA. IRM & amalgam had complete healing in 56 days. *EBA greater inflammation initially*, all had complete healing in 100 days.
4. *Gutmann IEJ 1997* – *Diaket superior seal to amalgam*, degree of bevel did not influence leakage, sonic prep larger than bur prep
5. ***Johnson OOO 1999*** – Problem with *amalgam* as a retrograde material – *toxicity, delayed expansion/ corrosion, tissue staining, and leakage*.
6. ***Zhu JOE 1999*** – *Amalgam more cytotoxic* to *human PDL cells* and *human osteoblast-like cells* than IRM or SEBA.

**Why use SEBA?**

Ease of handling, less washout, good seal, biocompatibility, proven track record

1. *Adamo IEJ 1999* – Comparative study: MTA, SEBA, Composite and Amalgam as a root end filling materials – results indicated NSD in leakage between all materials.
2. *Jeansonne JOE 2003* – *SEBA* & MTA *leaked less than amalgam*
3. ***Dorn JOE 1990*** – Results of study demonstrated greater success with use of **SEBA (95%) vs IRM (91%) vs amalgam (75%)**
4. ***Torabinejad JOE 1995*** – confirmed **biocompatablilty** of SEBA and MTA
5. *Trope OOO 1996* – SEBA superior to glass ionomer, amalgam, IRM and composite as retrofilling material

**Super EBA vs. MTA for Root End Filling**

**MTA > Super EBA:**

1. ***Von Arx 2012*** – Prospective, Apical Microsurgery, 170 teeth, ***Healing* @** ***5 year*** *follow up*: **MTA 86%, Super EBA: 67**%; Signficiant difference in healing between MTA & Super EBA
2. ***Tsesis 2013* –** Meta-Analysis (modern techniques), **MTA ↑ success** of Surgery **compared to SuperEBA, IRM, Amalgam** for retrofill
3. *Torabinejad 1993* – MTA > SEBA or Amalgam for root end filling

**MTA = Super EBA:**

1. ***Song 2012*** – EMS, 260 teeth, Prospective randomized clinical trial, MTA vs. Super EBA, ***12 month*** *follow up*, **MTA: 95.6%, SuperEBA: 93.1%**; No significant difference in healing (clinical/radiographic) … *does NOT account for* ***“Late Failures”***

**What is MTA?**

Components:

1. **Dicalcium silicate**
2. **Tricalcium silicate**
3. **Tricalcium aluminate**
4. Bismuth oxide
5. Tetra calcium aluminoferrite (not in white MTA)
6. Calcium sulfate hydrate (gypsum)

**Gray vs. White MTA**

1. ***Faraco/Holland*** – Both White and Grey MTA demonstrated complete *dentin bridge formation* in dog pulp capping
2. ***Parirokh*** – Both White and Grey MTA demonstrated *hard tissue barrier formation and no inflammation*
3. ***Camilleri*** – Both White and Grey MTA demonstrated comparable *biocompatibilities and osteogenic properties*

**Discuss studies using MTA for root end fillings –**

1. ***Torabinejad JOE 1993*** – **MTA showed less leakage than SEBA, IRM, or Amalgam**  (See also ***Fischer/Miller 1998***)
   1. Advantages of MTA:
      1. Easy to mix
      2. Dry field not required
      3. Excess is easy to remove
      4. Less *periradicular inflammation* than amalgam
      5. *Cementum* found on surface after healing
      6. Less *bacterial or endotoxin leakage* than amalgam, SEBA or IRM (90 days)
      7. Will not be effected by resection after it sets (cut thru material)
   2. Disadvantages of MTA
      1. Long setting time – 3 hours

**Discuss studies using MTA for root end fillings –**

1. ***Christiansen IEJ 2009*** – *Randomized Clincal Trial*: 44 patients: 2 groups: 1) 3 mm Retroprep/MTA retrofill, 2) Burnished GP w/No retroprep/fill. Healing (*1 yr follow up*): **MTA (1): 96%, GP (2): 52%**
2. ***Tsesis 2013*** – Meta-Analysis, MTA ↑ success compared to other material
3. *Wu/Wesselink* – 24 hrs: GI>Amalgam>MTA = SEBA; 1 year: MTA>GI>SEBA>Amalgam
4. ***Von Arx 2012*** – *5 yr apical microsurgery* outcome study: **MTA root end filling: 86%, Super EBA: 67%**
5. ***Baek* 2005** – Dog study, Root end retrofill: *MTA>SEBA>Amalgam* for Cementum attachment, cementum growth, degree/type of inflammation, and PDL reformation around the resected root end

**MTA**

1. *Parirokh 2006* – Review of MTA properties and clinical applications
2. *Yesilsoy* – 45 mins to set
3. ***Holland/de Souza 1999/2002*** – Histopathologic dog studies – **MTA** exhibited **↓ inflammation (vs. CaOH2**), **complete hard tissue barrier** formation, and **cementum** **attachment** directly to MTA
4. ***Baik*** – **PDL cells** closely attach to MTA
5. *de Souza/Costa 2008* – less cellular toxicity than CaOH2
6. ***Yasuda* *2007***– **MTA vs Dycal**, MTA ↑ Bone morphogenetic protein (BMP2) production, Dycal ↓ BMP2 production and ↑ cell death
7. ***Lovato/Sedgley 2011*** – **Antibacterial effect of MTA** against E. facaelis

**Discuss Bioceramics and EndoSequence Root Repair Material**

Only manufacturer and in Vitro studies to this point. **No outcome studies**!

***Damas/Hoen***:

1. Bioceramics = *Calcium silicate, Calcium Phosphate* bioactive material
2. Nanospheres and hydrophilic nature allow *dentinal tubule penetration*
3. Mechanical bond with dentin
4. *Biocompatibility = MTA* (*Damas/Hoen, Ma/Haapasalo*)
5. Exceptional dimensional stability (no shrinkage)
6. *Antibacterial: High pH (12.8), CaOH2 diffusion* (*Nasseh 2013*)
7. ***Hess*** – Sealer NOT retreatable with current methods – solvents, files, heat
8. *Ma/Haapasalo* – ESRR Putty/Paste similar cell viability as MTA (fibroblasts)
9. ***Damas/Hoen***– ESRR Putty/Paste similar cell viability as MTA (fibroblasts)
10. ***Modarezdeh*** – MTA > Geristore > ESRRM (odontoblastic viability and ALP expression)
11. ***Charland/Hartwell*** – MTA set at 36 hours, ESRRM NOT set at 48 hours!
12. *Lovato/Sedgley* – MTA and ESRRM have antibacterial properties - E. faecalis

**Discuss healing after surgery**

**Phases of Healing**:

1. **Inflammatory**: **1-3 days** – Fibrin clot, Epithelial seal/barrier, PMNs (24-48 hrs), Macrophages (48-96 hrs) – *Innate immune response*
2. **Proliferative**: **3-5 days** – Granulation tissue (day 4), Fibroblasts, Endothelial cells, variable number of macrophages and lymphocytes (T & B cells) – *Adaptive immune response*
3. **Maturation**: **5-14 days** – Collagen Formation, Angiogenesis, Re-organization, Fibrous CT replaces granulation tissue

**Discuss healing after surgery**

***Harrison/Jurosky 1991 & 1992 JOE*** – Monkey studies of surgical healing

**3 phases (CEC): Clotting/inflammation, Epithelial healing, CT healing**

1. Incisional wound – Intrasulcular incision leaves thin layer of connective tissue and epithelium attached to root surface after reflection. *Preservation of this tissue prevents epithelial down-growth* along root surface and loss of attachment.
   1. Day 1: **Fibrin Clot**, PMNs
   2. Day 2: **Epithelial seal,** Macrophages
   3. Day 3: **Epithelial barrier**, Fibroblasts, Macrophages
   4. Day 4: **Granulation tissue** w/collagen formation and Proliferation
   5. **Day 14**: **Replacement of granulation tissue by fibrous CT**

**CT heals by 14 days!**

Submarginal and intrasulcular flaps heal equally well (Refutes *Kramper/Kaminsky*).

**Discuss healing after surgery**

***Harrison/Jurosky 1991 & 1992 JOE*** –

**3 phases (CCB): Clotting/inflammation, CT healing, Bone Healing**

1. Dissectional wound – healing occurs ***slower*** *t*han incisional wound. Periosteum does not survive flap reflection. *Don’t curette cortical-retained periosteal tissues, they are a source of reattachment*. Crestal bone height is not altered (refutes *Wood, Kramper/Kaminsky)*
   1. Day 1: **Fibrin Clot**, PMNs, cortical necrosis of denuded areas
   2. Day 2: **PMNs**, Macrophages, Fibroblasts, No osteoclastic/osteoblastic activity
   3. Day 3: **Macrophages**, Fibroblasts, Collagen formation
   4. Day 4: **Granulation tissue**, Macrophages, collagen formation, **No osteoclastic/osteoblastic activity** on the cortical surface
   5. Day 14: Replacement of granulation tissue by **fibrous CT,** **new periosteum formed**, **osteoclastic/blastic activity!**
   6. Day 28: Flapped tissue normal, **continued osteoclastic/blastic activity** on the periosteal surface of crestal bone (PDL side heal)

**Discuss healing after surgery**

***Harrison/Jurosky 1991 & 1992 JOE*** –

1. Osseous wound – New bone matrix was *formed directly on devitalized bone surface*. Periosteum separates overlying mucosa from excisional wound site anddoes not function until excisional wound is filled with***woven (cancellous) bone of endosteal origin****.* 
   1. Day 1-3: **Coagulum** consisting of erythrocytes, some inflammatory cells, and tissue debris
   2. Day 4: **Proliferation of cancellous bone of endosteal origin into wound site,** Macrophages, PMNs, Fibroblasts within coagulum. Central core of coagulum devoid of vital cells
   3. **Day 14**: **Cancellous bone** occupied **4/5ths** of wound, **osteoblastic activity** throughout, **appositional growth** on both cortical and cancellous devitalized bone surfaces
   4. Day 28: Maturing trabecular bone predominates, delimiting membrane separates, osteoid deposited on inner endosteal and outer periosteal layers

**Discuss healing after surgery**

***Lin/Langeland* 1996 IEJ** – It is **NOT necessary to completely curette out all of the inflamed periradicular tissue during surgery,** but **removal of foreign objects is required** for resolution of a lesion.

***Corcoran 1984 JOE*** – Monkey study, Bone healing, demonstrated **16 week** results post op from apical surgery to be the same as undisturbed bone.

*(C.V. for bone healing = 16 weeks)*

***Von Arx*** – **Cementum:** Start: **10-12 days**, Completion: **28 days**; **Bone**: Start: **7 days (***Harrison/Jurosky – day 4)*, Completion: **16 weeks (4 months)**

***Wood 1972*** *–* **Full thickness mucoperiosteal flap** results in **loss of 0.5 mm** of **crestal bone height**

***Zimmerman/Velvart 2001*** – **Full thickness mucoperiosteal flap** healing results in **recession of marginal gingiva** and **papilla shrinkage**

**Discuss Suture Selection (LSK)**

1. ***Lilly 1968/69*** – Monofilament (ie: Nylon, Gut) produced *less inflammatory tissue* responses than Multifilament (ie: Silk, Polyester)
2. ***Selvig 1998*** – Monofilament sutures are *least traumatic/bacterial adhering* (Nylon, PTFE); Sutures should be removed at **48-72 hours**, Post 72 hours = Inflammation and Resealing of epithelium (epithelial barrier day 3)
3. *Torbejork* – Vicryl poly galactin 9/0
4. ***Kim*** – 5-0 or 6-0 **M**onofilament **N**ylon **O**r **P**ropylene i.e.: Vicryl (**MNOP**) to prevent *inflammation*

Monofilament = Nylon, Gut, Propylene (Vicryl, PTFE)

Multifilament = Silk, Polyester

**What is enamel matrix derivative ?**

Emdogain: a protein gel that has been shown to promote acellular cementum formation, which is the first step in regeneration of the attachment apparatus.

1. *Nakamura 2002 IEJ* – Pig study; Compared Emdogain and Dycal for pulpotomy – Emdogain had better pulpal healing and greater dentin formation than Dycal
2. *Iqbal/Bamaas 2001 Dent Traum* – Dog study; Evaluated Emdogain in replantation cases of extended extraoral dry time. Emdogain increased resistance to root resorption/ankylosis and stimulated healing of the PDL. (supported by Trope)
3. *Hamamoto 2002 Dent Traum* – EMD is accumulated in the cells at the root surface and promotes regeneration of the periodontal tissues and healing of root resorption

**Discuss GTR in endodontic surgery**

CaSO4 (Calcium Sulfate)

1. ***Suda 2002 IEJ*** – Dog study – CaSO4 was effective in *bone regeneration* on both large osseous defects and “through and through” osseous defects. It was less effective in osseous defects communicating with the gingival sulcus.
2. ***Pecora 1997 OOO*** – Rat study - CaSO4 barrier *impedes connective tissue ingrowth*, *allowing bone regeneration* during healing; Use in: >10 mm lesions, through and through lesions, endo-perio defects
3. *Apaydin/Torabinejad 2004 JOE* – Dog study – CaSO4 does NOT improve cementum deposition/osseous healing
4. *Dahlin* – Useful in through and through defects
5. *Oh/Fouad* – Useful in Pathologic dehiscence

**Discuss GTR in endodontic surgery**

GTR is beneficial

1. ***Pecora 1997 DCNA*** – Indications for GTR (Gortex membrane) in endodontic surgery include (same as for CaSO4):
   1. ***Large periapical lesion >10 mm***
   2. ***Through and through periapical lesion***
   3. ***Endo-perio defects***
      1. Periapical lesion *communicating with alveolar crest* (dehiscence)
      2. *Furcation involvement* as a result of perforation
      3. Root perforation with *bone loss to alveolar crest*

\*Periradicular healing occurred more rapidly with GTR membranes

1. Gutman 2001 JOE – Dog study w/Guidor – bioresorbable membrane enhances apical regeneration of bone, ct attachment and marginal bone
2. ***Tsesis 2011 JOE*** – *Sys Review/Meta-Analysis*, GTR favorable for ***large periapical lesions, through and through lesions***. Resorbable membranes.

**Discuss GTR in endodontic surgery**

GTR is not needed

1. ***Hartwell 2002 JOE*** – human study in vivo – NSD between rate of healing with or without GTR. **No beneficial effects on rate of healing.**
2. ***Torabinejad 1998 JOE*** – Cat study – GTR exhibited more inflammation, no positive effect on osseous healing or new cementum formation.

**Discuss Apical Decompression**

***Neaverth/Burg 1982*:**

Marsupialization = *Unroofing outer wall of cyst* by making an incision, evacuating contents and *establishing permanent opening by suturing cystic membrane to mucosal surface*; slower healing; not useful in radicular cysts

Decompression = *No exteriorization of cystic wall*; incision into cystic cavity, indwelling catheter (tubing) placed to *maintain patency of opening* and *allow irrigation, drainage of fluids/metabolic byproducts, and reduction of lesion*

Decompression is done to avoid:

* 1. Devitalization of adjacent teeth
  2. Damage to anatomic structures (IAN, Sinus)
  3. Loss of bony support
  4. Parasthesia
  5. Elderly pts where surgery is risky
  6. Enucleation and more extensive surgeries

**Discuss Apical Decompression**

1. *Freedland 1970* – Use polyvinyl tubing for maintaing patency and irrigation of large PA cystic lesions
2. ***Neaverth/Burg JOE 1982*** – Case reports; Use of *radiopaque catheter polyvinyl tubing* (size 8FR – lumen diameter: 1.5 mm, length: 2 inches)
3. ***Martin JOE 2007*** – Case report (13 yo/#9), large PARL, Protocol: NSRCT/CaOH2, GP obturation, return of sinus tract, Incision/Drain placement (size 10FR *radiopaque surgical latex tubing,* lumen diameter: 1.5 mm), sutures to close incision and stabilize drain, *Irrigation protocol: 0.12% CHX rinse with syringe once daily, Drain removed at 6 weeks.* Recalled every 3 months, complete healing at 2 years.

**Are root amputations an option to avoid extraction?**

YES

1. ***Smukler 1976*** – RCT prior to surgical root amp is treatment of choice but vital root amps are successful if RCT is done within 2 weeks of amputation.
2. *Blomlof 1997* – prognosis of root-resection is comparable to single-rooted teeth with an equal susceptibility to periodontitis, if endodontic conditions an maintenance care are optimal.
3. ***Basten 1996*** - **92%** of all resected molars survived an average of *12 years.*

**Vital Root Amputation**

1. ***Filipowicz*** – **Necrosis:** 12 months: 38%, **5 years: 87%**
2. *England/Hartwell*
3. *Langer* – 5 year survival: 18%

**Discuss intentional replantation. What is the prognosis?**

1. ***Kratchman 1997*** – Dental Clinics of North America – **Success rate 80-85%**
2. *Grossman* – 70% at 5 years
3. *Bender & Rossman* - 81% Success
4. *Yoshino* – 80% Success; ↑ Success for younger pts and Males

**Anti-platelet medications and Apical Surgery**

1. ***Napeñas*** – Do NOT take patient off of anti-platelet meds - plavix (clopidogrel) or aspirin - prior to apical surgery. Risks of discont. Meds prior to surgery far outweigh low risk of post-op complications with bleeding.

**TRAUMA**

**What is the incidence of traumatic dental injuries?**

1. ***Glendor 1996*** – **Avulsions** of permanent teeth seen **0.5 – 3%** of all dental injuries
2. ***Glendor 2008*** – **Maxillary central and lateral incisors** are most commonly avulsed
3. *Andreasen 1970* – **Avulsions** account for up to **16%** of traumatic injuries to permanent dentition
4. ***Ravn/Andreasen*** – **22%** schoolchildren experience at Traumatic Dental Injury
5. ***Kaba 2010*** – **11%** children (**6-18 yrs**) experience dental truama

**Discuss non-complicated crown fractures**

***Ravn 1981*** – Retrospective study of incisors w/ **enamel-dentin fractures**

1. **Pulp necrosis developed in 6.1%**
2. If concussion & mobility – pulpal necrosis in 30.1%
3. Prognosis depends not only on damage to tooth but damage to periodontium
4. Most **changes in vitality** occur in the **1st 6 months**.

***Kaba 2010*** – **12%** enamel-dentin fractures led to pulpal necrosis

Sensibility tests: +

Mobility: Normal, Percussion: - (if + evaluate for root fx or luxation)

Radiographs: Occlusal film (rule out root fx), 2 PAs: Mesial/Distal

**Discuss complicated crown fractures**

***Cvek 1982 JOE*** – *Monkey study* – Inflammatory reactions of pulp exposures from fractures or cavity preps at different times. **Crown-fractured teeth with vital pulp exposures** up to a period of **7 days, no more than 2 mm** of pulp beneath the exposure needs to be removed. **Partial Pulpotomy**.NOTE – foundation study for CVEK PULPOTOMY FOR TRAUMA CASES !! (96% success)

*Holland 2002 Dent Traum* – Dog study – success of treatment of traumatic fractures is partly dependant on how quickly therapeutic treatment is rendered.

*Fuks 1987 Endo Dent Traum* – Partial pulpotomy is treatment of choice in crown-fractured teeth with pulp exposure (including closed apicies)

Sensibility tests: +

Mobility: normal, Percussion: - (if + evaluate for root fx or luxation)

Radiographs: Occlusal film (rule out root fx), 2 PAs: Mesial/Distal

**Treatment for crown fractures**

*Crown/Root Fractures: Occlusal Film, 2-3 PAs; CBCT (root/alveolar fxs only)*

|  |  |
| --- | --- |
| Uncomplicated crown fracture | * Baseline pulp test * Smooth edges or restore with composite * Place Dycal base on exposed dentin * F/U at 6-8wks, 1 yr |
| Complicated crown fracture | * DPC (MTA) if *small, <24 hours, and open apex* * Cvek pulpotomy *if larger, >24 hours, or closed apex*   + Remove 2mm of pulp with diamond and H2O spray, then DPC * *Pulpectomy if necrotic or uncontrolled hemorrhage* * F/U at 6-8 wks, 1 yr |
| Crown-root fracture | 4 options after removing coronal fragment: F/U at 6-8 wks, 1 yr   * Gingival reattachment * Crown lengthening * Ortho extrusion * Extraction |

NOTE: *Transient Loss of Sensibility testing* – (up to 3 months), Need 1 add’l sign of necrosis – PARL, Vestibular swelling, dramatic color change

**Horizontal root fractures:**

1. *Mechanowitz* – Healing of root fracture occurs from the PDL
2. ***Jacobsen*** – **70-90% incidence of PCO**, **25% Necrosis**
3. ***Andreasen 2012*** – *10 yr survival root fractures*: Apical **89%**, Mid root: **78%**, Cervical/Mid root: **67%**; Cervical: **33%**; *CT healing only (8 yr survival)*: Apical, Mid root, Cervical/Mid root: **80%**, Cervical**: 25%**

**Healing mechanisms of root fractures:**

1. *Andreasen*/*Hjorting-Hansen 1966*:
2. Hard tissue fusion
3. PDL (C.T.) only
4. PDL (C.T.) + bone
5. Granulation tissue = Non-healing (*necrosis of coronal segment*)

**Horizontal root fractures:**

\*Root fractures/Alveolar fractures – Occlusal/2 PAs/CBCT

|  |  |
| --- | --- |
| Root fracture | * Clinical/Radiographic examination (*Occlusal/2 PAs/CBCT*) * Reposition, confirm position with PAs * Flexible splint **4 weeks or 4 months (cervical root fracture)** * F/U at 4 weeks, 6-8 weeks, 4 months\*, 6 months, 1 yr, then annually * RCT of coronal segment *if necrosis*, apical matrix may be needed * SX removal of necrotic apical segment if necessary * *Transient discoloration (grey) or False neg. pulp testing (up to 3 months)* |

NOTE: Alveolar fracture has same recall schedule as Root Fracture: 4 wks (remove splint), 6-8 wks, 4 mo, 6 mo, 1 year – 5 years

***Andreasen 1967 JOS*** – **4 types of healing**, mobility of coronal segment is important for healing

1. Calcified (callous) – hard tissue fusion
2. Connective tissue - PDL
3. Bone/Connective tissue – Bone/PDL
4. Granulation/inflammatory (non-union) – Necrosis of coronal segment

**Horizontal root fractures**

*Degering*; *Bender JADA 1983* – Recommend **3** radiographs with different **vertical angulations** to view horizontal fracture (See also *Brynolf*)

*Andreasen* – semirigid splint 2-4 weeks for horizontal root fractures, 4 months in cervical root fractures due to increased stability

***Jacobsen 1975*** – Long term prognosis of anterior teeth with root fractures

1. Location of the fracture influenced repair only slightly
2. Longevity (prognosis) of teeth was not shortened even when necrosis occurs; **Necrosis: 25%**; **PCO 70-90%**
3. Optimal treatment:
   1. reposition
   2. fixation – flexible/physiologic
   3. relief of occlusion

**What is the prognosis for luxation injuries ?**

**Closed Apex**

|  |  |  |
| --- | --- | --- |
| **Injury**  **(Pulpal Necrosis)** | ***Andreasen/Vesteergard-Pederson***  ***1985*** | *Dumsha* |
| Concussion | **3%** | 2% |
| Subluxation | **6%** |  |
| Extrusion | **26% (~30%)** | 98% |
| Lateral luxation | **58% (~60%)** | 77% |
| Intrusion | **94% (~90%)** | 100% |

**Trope, Pathways of the Pulp**: If sensibility testing indicates necrosis at the **2 wk f/u**, **CLOSED APEX ONLY (Luxation injuries – extrusive/lateral/intrusive)**, NSRCT should be performed due to high success of NSRCT in non-infected pulp versus risk of external inflammatory root resorption complication.

**Luxation Injuries**

IADT Guidelines 2012 – *de Angelis/Andreasen, AAE Guidelines 2014*

*2 PAs (M/D), CBCT (same as avulsions)*

|  |  |
| --- | --- |
| Concussion | * PDL injury only * Percussion pos., No mobility, No displacement * Vitality testing normal * No treatment needed – soft food 1 wk, CHX 0.12% 2 wks * Follow up: 2 wks, 4 wks, 6-8 wks, 6 months, 1 year |
| Subluxation | * PDL injury only * Percussion pos., Inc. mobility, No displacement * Vitality testing may be initially negative (transient) * Bleeding from sulcus * Flexible splint for **2 weeks** – patient comfort only * Follow up: 2 weeks, 4 weeks, 6-8 wks, 6 months, 1 year |

**\*Transient pulpal necrosis**: up to **3 months**, monitor for clinical/radiographic signs of necrosis (*At least* ***2 signs/symptoms*** *needed for pulpal necrosis*)

**Luxation Injuries**

IADT Guidelines 2012 – *de Angelis/Andreasen, AAE Guidelines 2014*

*2 PAs (M/D), Occlusal, CBCT*

|  |  |
| --- | --- |
| Extrusive Luxation | * *Tooth appears long, Excessive mobility* * Vitality testing are likely negative * *Radiographically: Increased PDL space apically* * Reposition w/ fingers, physiological splint for **2 weeks** * NSRCT if no response to vitality testing at 2 week f/u (closed apex) * Follow up: 2 weeks, 4 weeks, 6-8 weeks, 6 months, 1-5 years |
| Lateral Luxation | * Tooth is displaced in a palatal/lingual or labial direction, poss alv. fx (F) * *No mobility, high percussion sound (ankylotic)* * Vitality testing negative * *Possible alveolar fracture – palpable, Increased PDL space apically* * Reposition w/ forceps or fingers and physiologic splint for **4 weeks** * Monitor pulpal response * NSRCT if no response to vitality test at 2 week f/u (closed apex) * Recall: 2 weeks, 4 weeks, 6-8 weeks, 6 months, 1-5 years |

\*If pulpal necrosis (open apex) – attempt pulpal revascularization or MTA apexification

**Luxation Injuries**

IADT Guidelines 2012 – *de Angelis/Andreasen, AAE Guidelines 2014*

Occlusal, 2 PAs (M/D), CBCT

|  |  |
| --- | --- |
| Intrusion   * Tooth is displaced axially into alveolar bone – appears short * *Loss of PDL space apically* * *No mobility, high metallic percussion sound (ankylotic)* * Vitality testing negative * *CEJ apical to level of adjacent tooth,* may be apical to level of marginal bone * Recall: 2 weeks, 4 weeks, 6-8 weeks, 6 months, 1-5 years | **Open apex**:   * ≤ 7 mm: Allow spontaneous re-eruption, if no movement after 2-3 weeks, ortho reposition * > 7 mm: Ortho or surgical reposition * Monitor for pulp vitality   **Closed apex**:   * < 3mm/<17 y.o.: Allow spontaneous re-eruption, after 2-3 weeks, reposition ortho or surgically * 3-7 mm: Ortho or surgical repostition * > 7 mm: Surgical reposition * NSRCT at 2 week f/u * Splint **4 weeks** |

**Discuss luxation injuries**

1. ***Bergenholtz 1974*** – **64%** of the time, traumatized teeth with necrotic pulps have a mixed flora with anaerobes predominating. Aseptic necrosis was found in the other teeth.
2. ***Bhaskar JADA 1973*** – EPT, cold, heat testing are unreliable following trauma. Blood supply may still be functioning.
3. *Barnett 2002 Dent Trauma* – RCT is necessary when there are clinical and radiographic signs of pulpal infection.
4. *Siskos 1996 Endod Dent Traum* – Techniques to reposition intruded tooth include:
   1. Observation for spontaneous reeruption – minimal intrusion
   2. Surgical reposition – w/ early RCT to prevent anklyosis
   3. Orthodontic reposition – luxation of intruded tooth before applying ortho forces may prevent ankylosis.
5. *Feiglin 1996 EDT* – Histology – concussion and subluxation caused minimal damage. Lateral extrusion, intrusion caused major damage. Histology often not related to clinical symptoms

**Discuss storage media for avulsed teeth**

1. HBSS:
   1. ***Trope/Friedman*** - Provided **96h** storage- no replacement resorption
   2. ***Ashkenazi*** - *PDLF cells****:***  *HBSS=MILK > Viaspan*
2. Viaspan:
   1. ***Trope/Friedman***; ***Pettiete/Trope*** - Comparable storage times to HBSS (96h) (T/F), >HBSS (P/T)
3. Milk (low fat is better- ***Walker***):
   1. ***Blomlof***- pH and osmolality are compatible**;** better than Saliva - ↓ Infl. Root Resorption; **3 hours milk = Immediate replantation**
   2. Trope – Provided 6 hours of storage
4. Saline: ***Andreasen 1981*** – 0-120 mins – no replacment resorption
5. Saliva: ***Andreasen 1981*** – 0-120 mins – no replacement resorption
6. Water: ***Blomlof 1981*** - hypotonic – causes cell lysis; ***Andreasen 1981*** - ↑ Replacement resorption with tap water (0-120 mins)

***Andreasen 1986*** – Saline storage of a previously dry tooth has no positive effect on resorption or pulpal repair.

**Discuss splinting of avulsed teeth**

Physiologic (Flexible) Splint: up to 0.016” or 0.4 mm

1. ***Antrim 1982 JOE*** – describes a technique using 30lb monofilament nylon line and acid etch resin to splint traumatically luxated or avulsed teeth. The **non-rigid splint** stabilizes traumatized teeth and allows for physiologic movement.
2. ***Nasjleti/Casteli 1982 OOO*** – Replanted teeth splinted for **7 days – PDL repaired** w/ no resorption/ankylosis. Extended splinting periods (**30 days)** induced **further root resorption and ankylosis**.
3. *Berude/Hicks 1988 JOE* – Monkey study - No differences observed in healing in replanted teeth w/ physiologic splint, rigid splint, no splint.

**Discuss Effect of Diet on Healing**

1. ***Andersson 1985*** – Monkey study – Eval. Hard pellet vs. Soft diet post replantation (8 weeks) for replacement resorption. Significantly less replacement resorption in the **hard pellet group**– Normal Mastication

**How would you manage an avulsed tooth clinically?**

Management at site:

1. Gently wash if dirty, replant
2. If unable to replant, store in HBSS, milk, saline or saliva (no water!)
3. Proceed to office

***Andreasen 1981***– Monkey study, relationship exists between **extra-alveolar time, storage medium and root resorption.** *After 60 min of dry storage, Replacement resorption is very prominent. After 30 mins dry time, Inflammatory resorption is prominent. Saline, Saliva – no replacement resorption at 2 hrs. Water bad – hypotonic, cell lysis (Blomlof).*

General Adjuncts to trauma treatment:

1. tetanus booster – if tooth touches soil
2. chlorhexidine rinses – 0.12%, 2 wks
3. analgesics, antibiotics (Pen VK or Doxycycline)
4. recall for 5 years

**How would you manage an avulsed tooth clinically? Continued**

Avulsion**: OPEN APEX , Storage Media and/or < 1 hour extra-oral dry time**

**Rational: Promote Revascularization**

1. Rinse root surface/AF with Saline, Anesthesia, Irrigate socket
2. **Soak 5 mins in suspension 1mg Doxycycline in 20 mL Saline\***
3. **Coat with Minocycline microspheres (Arrestin)\*\*** IADT 2012
4. Replant, Verify position w/ PAs, Physiologic splint up to 14 days
5. Administer antibiotics, check tetanus booster, patient instructions (soft diet, CHX)
6. Recall, if necrotic, proceed with revascularization or MTA apexification

\****Cvek/Cleaton Jones 1990 EDT*** – monkey study, Showed a *decreased frequency of microorganisms* in the pulpal lumen and *less ankylosis or inflam resorption* as a result of the soaking in Doxcycyline (1mg in 20 mL saline). No revascularization in closed apex teeth. **Inc AF opening (> 1mm), Inc Revascularization** (*Kling*)

\*\****Ritter/Trope 2004*** – dog study, Minocycline (Arrestin) promoted *revascularization* of immature avulsed teeth (dry 5 mins), ***91% revas.*** (vital tissue)

**How would you manage an avulsed tooth clinically? Continued**

Avulsion: **OPEN APEX, >1 hour extra-oral dry time**

Poor prognosis for revascularization, PDL necrotic, Goal: **replant for esthetics, function and maintain alveolar bone contour** – *Outcome: Ankylosis/resorption*

1. Remove tissue tags with wet gauze
2. **Soak in 2% NaF for 20 mins**
3. **Complete RCT extraorally or No RCT/Monitor (esp wide open apex)**
4. Anesthesia, Replant, Verify position w/PAs
5. Physiologic splint 4 weeks, Antibiotics, Tetanus booster, Pt instructions (soft diet, CHX)
6. Recall, *Decoronation* necessary when *infraposition > 1mm*
7. Baseline: Weight/Height measurements to follow growth and need for decoronation

***Kling/Cvek 1986*** – immature teeth replanted >45 mins = ↓ revascularization

***Coccia 1980*** – human study; **5 min 2% NaF soak** (vs. saline) prior to replantation ↓ replacement resorption (esp in longer dry times); Fl binds with HAP to create FAP (resistant to resorption). **Delays replacement resorption (2x survival time)**

**How would you manage an avulsed tooth clinically? Continued**

Avulsion: **CLOSED APEX, Storage Media and/or < 1 hour extraoral dry time**

1. Rinse root surface/AF with Saline, Anesthesia, Irrigate socket, Replant
2. Verify position w/ PAs, Physiologic splint up to 2 weeks, Antibiotics, Tetanus booster, Patient instructions (soft diet, CHX rinse)
3. Initiate RCT **7-10 days** (Rationale: *prevent infection of canal that leads to external inflammatory resorption*), Remove splint at 14 days
4. Place CaOH2 for up to 1 month
5. Obturate canal when CaOH2 is removed

***Gregorio/Jeansonne 1994*** – dog study; **Immed. pulpectomy/CaOH2: ↑ replacement resorption vs delayed 18 days**; Delayed 4-18 days: NSD in resorption (surface, inflamm, replacement) – Supports waiting 7-10 days to initiate pulpectomy/CaOH2

***Trope/Yesilsoy 1992***– No difference in inflammatory & replacement resorption between 1 week and 8 weeks CaOH2 when **RCT initiated at day 14**

***Dumsha 1995*** – No difference in inflammatory resorption between avulsed teeth obturated with gutta-percha or long term CaOH2 (5 months). Perform RCT at 14-28 days and obturate with gutta-percha.

**How would you manage an avulsed tooth clinically? Continued**

Avulsion: **CLOSED APEX, > 1 hour extra-oral dry time**

Rational: *PDL is necrotic*, *prepare root to resist replacement resorption*

1. Remove tissue tags with wet gauze
2. **Soak in 2% NaF for 20 minutes**
3. Anesthesia, Irrigate socket, Replant, Verify position w/ PAs,
4. Physiologic splint for 2 weeks, Antibiotics, Tetanus booster, Patient instructions (soft diet, CHX rinse)
5. Initiate RCT **7-10** days (*or before replantation*)
6. Place CaOH2 for up to 1 month
7. Obturate canals when CaOH2 is removed

***Kling/Cvek*** – Mature teeth (AF < 1mm) exhibited no revascularization

***Coccia***– 2% NaF delays replacement resorption, 2X survival time expected

***Gregorio/Jeansonne*** Delay Pulp/CaOH2 for 7-10 days following replant to ↓ replacement resorption (damages PDL/prevents healing) – *Andreasen; Lindskog*

***Trope/Yesilsoy***: 1 wk = 8 wk CaOH2 for inflamm/replacement resorption

**What are some factors that affect healing of avulsed teeth?**

1. ***Andreasen 1966*** –
   1. PDL showed 4 types of healing:
      1. Normal
      2. Replacement resorption (Dentoalveolar ankylosis)
      3. Surface (transient) resorption
      4. Inflammatory resorption
   2. **90 % of teeth replanted w/in 30 min = no resorption**
   3. **Majority of teeth replanted after 90 min = resorption**
2. ***Lindskog/Hammarstrom 1985 EDT*** –Avulsion, if PDL damage, removal of the PDL with NaOCl to reduce resorption. **Destruction of >20% of the root surface is required for replacement resorption to occur**
3. ***Oswald/VanHassel 1980*** – monkey study, **all 90 minute dried teeth showed ankylosis and replacement resorption**. All saliva-stored teeth retained normal mobility, healing PDL space and no resorption.

**What are some factors that affect healing of avulsed teeth?**

***Andreasen/Borum 1995*** – Factors related to Pulpal and PDL healing:

1. Pulpal Healing (Revascularization)
   1. Pulp length: ↓ pulp length, ↑ revascularization
   2. Wet extra-oral period: <5 min, ↑ revascularization
   3. Dry extra-oral period: *↓ dry time, ↑ revascularization*
2. PDL Healing: *↑ Stage of root development, ↑Dry time = ↑ Replace. Resorp.*
3. Diagnosis of Resorption:
   1. External inflammatory: < 6 months (radiograph)
   2. Replacement: *1-2 months (clinical),* < 12 months (radiograph)

***Soder 1977*** – Effect of drying on the viability of PDLF cells, **>60 mins dry time = no viable PDLF cells**

*Andreasen 1981* – Effect of extra-alveolar dry time: 30 mins – inflammatory resorption, 60 mins – replacement resorption. Wet after dry does NOT help.

**Discuss Root Resorption in Avulsion Cases**

1. External Inflammatory Root Resorption (*Andreasen*, *Trope*)
   1. Surface/Transient
   2. Progressive
2. Replacement Resorption/Ankylosis (*Lindskog*, *Andreasen*)
   1. Initial Inflammatory Root Resorption (may or may not)
   2. Replacement with Osseous tissue (Osteoblasts faster than cementoblasts) – Transient or Progressive

* ***Lindskog/Hammarstrom 1985 EDT*** –Destruction **>20%** of root surface = progressive ankylosis/replacement resorption
* ***Andersson 1989*** - >60 min extraoral dry time, **rate of replacement resorption was age related**: Young patients – 3-7 yrs, Older patients – much slower
* ***Andreasen 1995*** – Diagnosis: External Inflammatory: <6 months (radiographically), Replacement: 1-2 months (clinically), <12 months (radiographically), Surface: <12 months

**Discuss Root Resorption in Trauma Cases**

**External Inflammatory Root Resorption:**

1. ***Tronstad*** ***1988***– Trauma to external root surface denudes areas of precementum/cementoblasts → *chemotactic for hard tissue resorbing cells* *(osteoclasts/odontoclasts*), **Pulpal infection sustains clastic cells**
2. ***Trope 2002 – Review Root Resorption –*** Pulp space infection – **Bacterial TEBs** pass through dentinal tubules and **stimulate/prolong** inflammatory response in the PDL – Multinucleated giant cells resorb *until the stimulus is removed*
3. *Andreasen 1995* – Ext. inflammatory resorption diagnosed w/in **1st 6 months**

**Replacement Resorption (Repair-related):**

1. ***Andreasen 1975*** – **>20% root surface damage** = progressive replacement resorption
2. ***Lindskog/Hammarstrom 1985*** – Necrotic PDL → Ankylosis between bone and cementum due to **repair confusion** (osteoblasts vs. cementoblasts). **2 Types**: Ankylosis w/o root resorption (cementum-bone) & Ankylosis following inflamm. root resorption (dentin-bone)

**What is the role of CaOH2 in replanted teeth?**

***Andreasen 1981 JOE, Dent Trauma 2002***

1. **CaOH2 used too soon may diffuse through apex, damage PDL/cementoblasts, and prevent repair**. Should be used **after initial PDL healing (7 days)** has progressed.
2. **Long term CaOH2** weakens dentin, **>30 days** (*Andreasen; Rosenberg*)

***Gregoriou/Jeansonne 1994 EDT*** – Dog study; **Immed. pulpectomy/CaOH2: ↑ replacement resorption vs delayed 18 days**; **Delayed 4-18 days**: NSD in resorption (surface, inflamm, replacement) – **Supports waiting 7-10 days**

***Trope 1995 EDT*** – Long term (**12 wk**) CaOH2 for **established inflam root resorption** or **RCT started >10 days post avulsion** is better than short term (1 wk) treatment.

***Tronstad*** – CaOH2 upregulates alkaline phosphatase, ↑ pH - inhibits collagenases, promotes hard tissue repair/formation

**What is the role of fluoride?**

1. *Shulman 1973 J Dent Res* – Demonstrated decreased resorption of avulsed teeth using sodium fluoride.
2. *Bjorvatn/Klinge 1989* ***–*** Dog study, Soaking in **1% SnF2** (45 mins) will decrease surface, inflammatory and replacement resorption
3. ***Coccia 1980 JOE*** – Human study, Treatment of the root with **2% NaF** **(5 mins)** before replanting makes it osteoclast resistant (**fluroapatite**), **delaying replacement resorption**. **Twice the survival time**can be expected.

**Would you recommend systemic antibiotics after replantation?**

Antibiotic Recommendations:

1. **Doxycycline 500mg q6h x 7 days ( > 12 yrs)**
   1. Static, acts on ribosomes (30s rRNA)
   2. *Avoid if <12 years old or pregnant*
2. **Pen VK 500 mg q6h x 7 days (< 12 yrs or Pregnant)**
   1. Use only if tetracycline is contraindicated

***Sae-Lim/Trope 1998 EDT*** – Dog studies, Avulsion/replantation 1hr, compared *Amox vs. Tetracycline vs. placebo* to inhibit inflammatory or replacement resorption. Findings: **Tetracycline has anti-resorptive properties in addition to the antibacterial properties. It inhibits Osteoclasts and Collagenase**. It could be considered as an alternative to Amoxicillin after avulsion injuries.

***Hammarstrom/Blomlof 1986 EDT*** – Use systemic antibiotic (amox) immediately after replantation to reduce inflammatory resorption. No effect seen on replacement resorption.

**IADT/AAE 2014 Guidelines: Recall Schedule**

Enamel-Dentin Fracture (Uncomplicated): 6-8 weeks, 1 year

Enamel-Dentin-Pulp (Complicated) Fracture: 6-8 weeks, 1 year

Horizontal Root Fracture (middle/apical 1/3rd): 4 weeks, 6-8 weeks, 4 months, 6 months, 1-5 years

Horizontal Root Fracture (cervical 1/3rd): 4 weeks, 6-8 weeks, 4 months, 6 months, 1-5 years

Concussion: 2 weeks, 4 weeks, 6-8 weeks, 6 months, 1 year – 5 years

Subluxation: 2 weeks, 4 weeks, 6-8 weeks, 6 months, 1 year – 5 years

Extrusive Luxation: 2 weeks, 4 weeks, 6-8 weeks, 6 months, 1-5 years

Lateral Luxation: 2 weeks, 4 weeks, 6-8 weeks, 6 months, 1-5 years

Intrusive Luxation: 2 weeks, 4 weeks, 6-8 weeks, 6 months, 1-5 years

Avulsions: 7-10 days (closed apex, NSRCT), 2 weeks, 4 weeks, 3 months, 6 months, 1 year, yearly to 5 years (Monitor growth: ht/wt – infrapositioning)

**Pulp Testing Post Truama (*Bhaskar* – inaccurate tests!)**

LDF: Infared light, Doppler scatter of moving RBCs, Pulpal Blood Flow

1. *Yanpisett/Trope -* dog study, avulsion/reimplantation - detect return of pulpal blood flow by **4 wks**
2. *Gazelius* – case report, Lateral luxation 4 lower anterior incisors – detected blood flow **6 wks (partially), 9 mos (complete)**

Pulse Oximeter: Red/Infared light, selective absorption by oxygenated/deoxygenated Hemoglobin, non-absorbed light = Oxy Sat.

1. *Gopikrishna* –Pulse Ox signficantly improved ability to detect pulp vitality *(intact vascular supply)* **day 0 – 1 month** in comparision with EPT and Cold tests for recently traumatized teeth (**concussions/subluxation only**)
2. *Setzer* – Pulse Ox able to differentiate mean pulpal oxygen saturation levels for pulpal conditions (ie: normal vs. reversible vs. irreversible vs. necrotic)

EPT:

1. *Pileggi* – Ferrets, Concussion, 10 days return of EPT response

**Decoronation**

**Technique**: Removal of crown and submergence of root, removal of obturating materials (if present), induction of blood clot, reposition flap over socket

**Indications**: *Replacement Resorption/Ankylosis of erupting tooth*; Post Trauma; ***Infrapositioning >1mm***

**Timing**: Pre-growth phase, during growth phase

1. ***Malmgren/Cvek* *1984*** – Prospective Case Series, *1st Study on Decoronation*. 24 reimplanted Maxillary incisors, ages 11-19, clincial/radiographic signs of **ankylosis and infrapositioning** – decoronated and followed for up to 18 months. Findings: *continued growth of alveolar bone and replacement resorption*
2. ***Filippi/VonArx 2001*** – Case report, 12 yr old, avulsion of C.I., ankylosis/replacement resorption. Decoronation resulted in *preservation of alveolar ridge with 1 mm vertical bone apposition* **over top of the decoronated root** and continued replacement resorption

**Success-Failure**

**When should you recall a patient?**

1. ***Reit 1987 EDT*** – Recall after 1 years and annually for min of **4** years
2. *Andreasen 1972 Int J Oral Surg* – Recall after 1 year and continue recall for 4 years. Wait 4 years before considering uncertain and incomplete cases a failure.
3. ***Orstavik 1996 IEJ*** – This study recommends follow-up at **1 year**. Peak incidence of healing or emerging persistent apical periodontitis occurred at 1 year. **88% showed signs of healing at 1 year**. Complete healing of preoperative AP in some instances required **4 years** for completion.
4. *Seltzer/Bender 1966* – Evaluation of success should occur after 6 months – 2 years. May take up to 2 years for radiographic healing or signs of persistent disease to present clinically or radiographically

**Outcome Studies**

**Initial treatment – NSRCT (83-89%)**

\* = # of studies meeting inclusion criteria for meta-analysis (strict/loose criteria)

|  |  |  |  |  |
| --- | --- | --- | --- | --- |
| Author | % Healed | Cases | Follow-up | Date |
| Strindberg | 87 | 529 | 4 years | 1956 |
| Seltzer | 80 | 2921 | 0.5 years | 1963 |
| Grossman | 86 | 432 | 1-5 years | 1964 |
| Ingle | 92 | 1229 | 2 years | 1965 |
| Sjogren | 91 | 356 | 8-10 years | 1990 |
| **Fristad/Molven** | **88** | 265 | **20-27 years** | 2002 |
| **Salehrabi/Rotstein (surv.)** | **97** | 1.46 mil | **≥8 years** | 2004 |
| Imura | 94 | 1376 | ≥0.5 years | 2004 |
| \*Ng (sys review/meta) | 75/85 | \*61 | ≥6 month | 2007 |
| **de Chevigny (Toronto)** | **86/95** | 510 | **4-6 years** | 2008 |
| **Ng (prospective)** | **83/89** | 702 | **≥2 years** | 2011 |

Ng: Strict = no signs/symptoms, no PARL/normal PDL, Loose = no signs/symptoms/reduc. of PARL

**Outcome Studies**

**Retreatment (80-86%)**

\* = # of studies meeting inclusion criteria for meta-analysis (strict/loose criteria)

|  |  |  |  |  |
| --- | --- | --- | --- | --- |
| Author | % Healed | Cases | Follow-up | Date |
| Allen/Newton | 84 | 667 | ≥0.5 years | 1989 |
| **Allen/Newton (2nd Retx)** | **47** | 41 | ≥0.5 years | 1989 |
| Sundqvist | 74 | 54 | 5 years | 1998 |
| Imura | 86 | 624 | ≥0.5 years | 2007 |
| **de Chevigny (Toronto)** | **82/94** | 229 | **4-6 years** | 2008 |
| \*Ng (sys review/meta) | 76/77 | \*17 | ≥0.5 years | 2008 |
| **Torabinejad (sys. review)** | **71, 83** | 1253 | **2-4, 4-6 years** | 2009 |
| **Ng (prospective)** | **80/86** | 750 | **≥2 years** | 2011 |

Ng: Strict = no signs/symptoms, no PARL/normal PDL, Loose = no signs/symptoms/reduc. of PARL

**Outcome Studies**

**Surgical Root Canal Treatment (88-94%)**

|  |  |  |  |  |
| --- | --- | --- | --- | --- |
| Author | % Healed | Cases | Follow-up | Date |
| **Rubinstein/Kim** | **92** | 59 roots | **5-7 years** | 2002 |
| Tsesis (sys review/meta) | 92 | \*11 | ≥1 year | 2009 |
| **Barone** (**Toronto**) | **74/94** | 134 | **4-6 years** | 2010 |
| **Setzer** (TRS vs EMS)  *Sys. review/Meta-analysis* | **59; 94** | 925; 699 | **≥ 0.5 years** | 2010 |
| **Setzer** (CRS vs EMS)  *Sys. review/Meta-analysis* | **88; 94** | 610; 699 | **≥ 1 year** | 2012 |
| **Song/Kim (prospective)** | **93** | 104 | **6-10 years** | 2012 |
| **Song (ReSx)** | **92** | 42 | **1 year** | 2011 |
| **Song (prospective)** | **88** | 115 | **4 years** | 2013 |
| **Tsesis (sys review/meta)** | **89** | \*18 | **≥1 year** | 2013 |

**Toronto Study – Outcome Predictors**

***de Chevigny/Friedman 2008* – Initial NSRCT**

Phase 4: *4-6 year* prospective outcome studies (cohort)

Teeth: 510, Healed = PAI ≤ 2, No symptoms or clinical signs other than Perc +

Healed: 439/510 (**86%**)

Functional: 479/506 (**95%**) No signs/symptoms, PAI not considered

Pre-operative Outcome Predictors (Overall):

1. **Presence of RL**: Absent 93%, Present 82%
2. **Number of Roots**: Single 93%, Multiple 84%

Pre-operative Outcome Predictors (Teeth w/AP):

1. **Intraoperative complications**: Absent 84%, Present 69%
2. **Root filling technique**: Vertical 87%, Lateral 77%

*Ingle (classic) 1965* - #1 Cause of Failure NSRCT = Incomplete obturation

*VonArx*  - 1 year follow up, pts who had pain at initial exam had ↓ healing rate

***Ng 2007*** – **PARL, Level of Obturation (Flush (0-2 mm short) > Short > Long), Quality of Obturation, Quality of Coronal Restoration**

**Ng Study – Outcome Predictors**

***Ng 2011*** – **NSRCT & Retreatment**, Prospective Clinical Study (cohort)

Recall period: *≥ 2 years*, Teeth: NSRCT 702 teeth, Retx 750 teeth

Success: Strict = No signs/symptoms/No PARL, Loose = No signs/symptoms/↓ PARL

**Strict Criteria: NSRCT 83%, Retx 80%**

**Loose Criteria: NSRCT 89%, Retx 86%**

Pre-operative Outcome Predictors:

1. **Presence of PARL**
2. **Size of PARL: < 5mm or ≥ 5 mm**
3. **Presence of Sinus Tract**
4. **Presence of Perforation**

Intra-operative Outcome Predictors:

1. **Apical Patency**: Maintenance of Patency ↑ Success
2. **Level of Obturation**: Overextended ↓ Success, Short of terminus ↓ Success
3. **Use of 0.2% CHX ↓ Success**, Use of **17% EDTA ↑ Success (RETX only)**
4. **Interappt Flare up**: ↓ Success

Post-operative Outcome Predictors:

1. **Quality of Coronal Restoration**

**Toronto Study – Outcomes**

***de Chevigny/Friedman 2008* – Orthograde Retreatment**

Phase 3/4: *4-6 year* prospective outcome studies (cohort)

Teeth: 229

Healed: 187/229 (**82%**)

Functional: 207/221 (**94%**)

Pre-operative Outcome Predictors (Overall):

1. **Root filling quality** (**voids, length of fill**): Inadequate 88%, Adequate 66%
2. **Perforation**: Absent 87%, Present 56%
3. **Presence of RL**: Absent 93%, Present 80%

Pre-operative Outcome Predictors (Teeth w/AP):

1. **# Treatment sessions**: One 100%, Two + 77%
2. **Root filling quality**: Inadequate 86%, Adequate 50%

*Imura 2007* – Outcome predictor for Retx: Pre-op AP

*Ng 2008* – Outcome predictors for Retx: **Pre-op PARL, Apical extent of root canal filling, and Quality of Coronal Restoration**

**Toronto Study – Outcomes**

***Barone/Friedman 2010* – Apical Surgery**

Phase 3-5: *4-10 year* prospective outcome studies (cohort)

Teeth: 134

Healed: 99/134 (**74%**)

Functional: 126/134 (**94%**)

Pre-operative Outcome Predictors (Overall):

1. **Age:** > 45 y.o. 84%, ≤ 45 y.o. 68%
2. **Pre-operative Root filling length**: Inadequate 84%, Adequate 68%
3. **Size of Surgical Crypt**: ≤ 10 mm 80%, > 10 mm 53%

Pre-operative Outcome Predictors (Teeth w/AP):

None

***Song*** *– 3 mm collar of bone* ↑ *success*

*Von Arx 2010* – Prognostic factors: *MTA vs. superEBA – 86% vs. 57%*

*Zuolo* – Apical surgery 10% Higher Healing Rate w/ Retx prior to Sx

***Tsesis 2013*** – Progn. Factors: **Use of Microscope, MTA Retrofill** (vs. EBA/IRM)

**Comparison Outcome Studies**

**Retx vs. Endodontic Surgery:**

1. ***Torabinejad 2009*** – Systematic Review of Retx (1998-2009) vs. Endodontic Surgery (1970-2008) Outcomes using Rud’s classification system: *Late Healing RETX, Late Failures Surgery*
   1. 2-4 years: Endodontic Surgery: 78%, Retreatment: 71%
   2. 4-6 years: Retreatment: 83%, Endodontic Surgery: 71%
2. *Kvist/Reit 1999* – Retx vs. Endodontic Surgery. 95 endodontic failed cases. *Max centrals/canines*. Random assignment. Endo surgery = resection, hedstrom gp removal apically or round bur retroprep, heated gp retrofill. *Recall 1-4 years*. Clinical/radiographic evaluation at recall:
   1. 1 year: Endodontic Surgery > Retx
   2. 4 years: Endodontic Surgery = Retx

**\*Both studies suggest** **“long term failures” in surgery cases, follow up > 4 yrs**!

**Comparison Outcome Studies**

**Traditional vs. Endodontic Microsurgery:**

1. ***Setzer 2012*** – *Systematic Review of EMS vs. CRS* (Contemporary Root end surgery – loupes/no magnification), EMS n = 699, CRS n = 610. **EMS = 94%, CRS = 88%.** Success based upon Rud or Molven’s classifications (radiographic/clinical healing) – Complete, Incomplete (scar), Uncertain (↓/same size RL), Unsatisfactory healing (↑ size RL)
2. ***Setzer 2010*** – *Systematic Review of EMS vs. TRS* (Traditional Root end surgery), EMS n = 699, TRS = 925. **EMS = 94%, TRS = 59%.** *EMS 1.58 x more successful than TRS*. Success based upon Rud or Molven’s classifications for surgery (radiographic/clinical healing).

EMS = microsurgical techniques, ultrasonic root end prep, IRM/SuperEBA/MTA retrofill, microscope (10X or greater)

CRS = same as EMS but with only loupes or no magnification (0-4X)

TRS = Bur for root end prep, amalgam retrofill, loupes or no magnification (0-4X)

**What are some reasons for failure of non-surgical treatment?**

The *most common cause is previous treatment falls short of accepted standards*:

POOR PAST AM = Perforation, Obturation, Overfill, Root canal missed Perio disease, Another tooth, Split tooth, Trauma, Anatomy complexities, Microleakage

*When treatment is consistent with high standards*, failure may occur due to:

**Intraradicular infection**:

1. ***Lin 1992 JOE*** – major factors associated with endodontic failures are *persistence of bacterial infection in the canal space* and the *presence of preoperative periradicular rarefaction.*
2. ***Nair 1990 JOE*** – In the majority of root-filled human teeth with therapy resistant periapical lesions, *intraradicular microorganisms may persist* and may play a role in treatment failures.
3. ***Fabricius 2006 (see also Sjogren 94% vs. 68%)*** – Monkey study, Bacteria surviving NSRCT significantly inc failure (72% vs. 21%)
4. ***Ricucci/Siqueira 2012*** – *Intraradicular biofilms responsible for AP*, CAGE – Cysts: 95%, Abscess: 83%, Granuloma: 70%, Extraradicular: 6%. Most extra-radicular infections are planktonic and AAA cases.

**What are some reasons for failure of non-surgical treatment?**

**Extraradicular Infections**: Actinomyces Israelii (Actinomycosis), Propionibacterium propionicum

1. ***Sjogren****,* ***Sundqvist et al 1988 IEJ*** – **Propionibacterium propionicum** may be implicated in Extraradicular infection
2. ***Nair 1984 JOE*** – **Actinomyces israelii** is able to establish Extraradicular infection
3. *Sjogren, Sundqvist & Nair 1992 OMI* – The pathogenicity of **A. israelii** is due to its ability to establish cohesive colonies of branching filamentous organisms that are enmeshed in an extracellular matrix. It seems that the organisms existing in such colonies can evade destruction and elimination by the host phagocytic cells.
4. ***Sunde/Tronstad JOE 2002***- Microbiota of Periapical lesions refractory to endodontic therapy: AAP or CAA cases – Sampling of Periapical lesion during surgery. **35/36 positive for extraradicular infection**. 51% Anaerobes, 79% Gram +, 148 microbial strains, Avg. 4.1 strains/case

**What are some reasons for failure of non-surgical treatment?**

**Foreign body reactions**: chronic inflammatory periapical tissue reaction due to extruded root canal filling materials or food debris (ie: cellulose)

1. ***Nair 1990 JOE*** – In the absence of microbial factors, root filling materials which contain irritating substances can evoke a foreign body reaction at the periapex, leading to the *development of aymptomatic periapical lesions*.
2. ***Simon 1982 JOE*** – Open teeth can trap food particles (ie leguminous seeds “pulses”) which may travel through the tooth into the periradicular space and induce a “**pulse” granuloma**. The cellulose component of the seeds is the causative component.
3. *Koppang et al 1992 J Dent Asso S Afr*. – Identification of common foreign material in granulomas:
   1. Black/brown fragments – amalgam
   2. Fine black/brown/yellow – sealer
   3. Basophilic fragments – CaOH2
   4. Elongated/rounded/oval/kidney-shaped, colorless – cellulose
4. ***Koppang 1978 Scan J Dent Res*** – Endodontic paper points material has been found as an etiologic factor in periapical inflammatory processes.

**What are some reasons for failure of non-surgical treatment?**

**Apical Cysts (true cysts)**

1. ***Nair 1996*** – Histologic examination of apical lesions were identified proportionally to be:
   1. 50% granulomas, 35% abscess
   2. **15% cysts**
      1. 61% - true cysts (9%)
      2. 39% - pockets cysts (6%)
2. ***Spattafore*** – 52% granulomas, **42% cysts,** 2% scar
3. ***Koivisto*** – 40% granulomas, **33% cysts**, 20% other (KCAM)

**Cholesterol Crystals**

1. ***Nair, Sjogren & Sundqvist 1993 IEJ*** – The *accumulation of tissue break-down products such as cholesterol crystals*, and the condition of the lesion itself, can *adversely affect the healing process* of the periapex following root canal therapy. Consequently, such apical lesions can remain refractory to conventional endodontic therapy for long periods of time.

**Do Periapical Cysts heal?**

**YES**

1. ***Lin/Ricucci/Rosenberg* *2009*** – Apical cysts can heal regardless of true vs. pocket cyst. Apoptosis following removal of source of inflammation (bacteria) within canal.
2. *Caliskan 2004* – Healing of large cyst-like lesions (7-18 mm diameter). *Size of lesion is not major determining factor in NSRCT vs. EMS (opposes Ng)*

**MAYBE**

1. ***Nair* 1998** - Pocket cysts may heal after NSRCT, True cysts are less likely to heal without surgical intervention
2. *Natkin* 1984 – Apical cysts are less likely to heal with NSRCT alone

**Does the presence of a radiolucency affect the prognosis?**

**YES**

1. ***deChevigny/Friedman*** – The presence of apical periodontitis decreases the success by 10-25%
2. Other studies finding a decreased success with radiolucencies:
3. *Strindberg*
4. *Seltzer*
5. *Sjogren*
6. *Lin*
7. *Molven*
8. ***Ng***

**Does a negative culture at the time of filling give better prognosis?**

**YES**

1. ***Sjogren, Sundqvist 1997 IEJ*** – Human study, 5 years post op. **Healed** (cultured at time of filling): **Negative culture: 94%, Positive culture: 68%.** Success appears dependent on eliminating bacteria from the root canal prior to obturation. *CaOH2 to eradicate infection – 2 stages*
2. ***Fabricius/Moller 2006*** – Monkey study, innoculated canals with combinations of bacterial species; 2.5 yr recall, **Healed** (cultured at time of filling): **Negative culture: 72%, Positive culture: 21%**; Bacterial combinations – more common than single species
3. *Molander/Kvist 2007 JOE* – Human study, PN/AAP, 2 year recall. No difference in 1 vs. 2 visit (65% vs. 75%). **Healed: Neg culture: 80%, Pos. culture: 44%.**

**Does a negative culture at the time of filling give a better prognosis?**

**NO**

1. ***Peters/Wesselink 2002 IEJ*** – Complete radiographic healing was observed in 81% of the cases treated in one visit vs 71% for those treated in two visits. (NSD in study) In addition no statistical difference was found whether cultivatable bacteria were present or not present prior to obturation (*opposes Sjogren and Fabricius/Moller*)
2. *Stromberg 1987 EDT* – Healing occurs in apical periodontitis even if bacteria are present in the canal during obturation.

**Does a negative culture at the time of filling give a better prognosis?**

**NO**

1. *Matsumoto 1987 JOE* – Multiple factors usually involve in failure, NSD noted between positive and negative cultures. Risk factors observed for treatment failure included:
   1. Periradicular radiolucency
   2. *Overextension – greatest inflammatory response*
   3. Deep perio pockets
   4. Occlusal trauma
   5. No adjacent teeth present
2. *Seltzer 1964 OOO* – This sudy compared obturation of teeth with positive and negative cultures. NSD was detected histologically. *Greatest inflammatory response was seen in overfilled canals.*

**Does the level of root canal filling affect success?**

**YES**

1. *Sjogren 1990 JOE* – Relationship between level of fill and success
   1. **Underfill >2mm – 68% success (least successful)**
   2. 0-2mm from apex – 94% success
   3. Overfilled – 76% success
2. ***Schaeffer*** – **Meta-Analysis (level of obturation)**: Success – 0-1 mm short of RA (A) > 1-3 mm short of RA (B) >> overextended past RA (C). **Overall success: Group A 2.7% > B and 26.2% > C**
3. ***Wu/Wesselink/Walton*** – Termination of instrumentation/obturation: **Vital cases: 2-3 mm from Apex (vital apical stump), Necrotic cases: 0-2 mm from apex (elimination of apical infection)**
4. *Seltzer/Bender 1963 JADA* – An **overextended fill decreases success** but underfilling had no influence.

**Does the level of root canal filling affect success?**

**YES (Cont.)**

1. ***Ng*** ***2007*** – Level of filling affected success , **Overall Long ↓ success**; w/o PARL: Short = Flush (0-2 mm) > Long, w/PARL: Flush > Short = Long
2. *Fristad/Molven 2002 IEJ* – Extruded material delayed healing, late periapical changes can occur more than 10 years after treatment.
3. ***Ricucci/Langeland 1998 IEJ*** – Apical Limit of Root canal instrumentation/obturation should be the **AC**. **Worst prognosis with I/O beyond the AC** as this causes **injury/larger tissue wound and introduces foreign bodies (ie: sealer, gp) into the periapical tissues**.

**NO**

1. *Lin 1992 JOE* – The apical extent of the root canal filling, ie underfilled, flush-filled or overfilled, seems to have no correlation to treatment failures.

**Is one visit NSRCT more successful than two visit NSRCT?**

**YES**

1. *Ashkenaz 1984 DCNA* – Review article: findings
   1. No increase in post op pain in single visit treatment
   2. High level of success w/ single visit treatment
   3. Eliminates inter-appt. contamination potential (leakage)
   4. Disadvantage – emergency drainage complicated by filling

**NO DIFFERENCE**

1. ***Ng 2007*** – No significant differnce in odds of success between 1 and 2 visit NSRCT (1 visit = more post obturation pain)

Problem: Inaccuracies w/culturing intratubular bacteria – false neg cultures

**Is one visit NSRCT more successful than two visit NSRCT?**

**NO DIFFERENCE (Cont.)**

1. *Kvist 2004 JOE* –microbio prospective, 2 visit no better than 1 visit
2. ***Weiger 2001* -** Prospective clinical trial, NSD in 1 vs. 2 (CaOH2), PN/AP
3. ***Penesis 2008* - Randomized clinical trial**, 12 month f/u, Pulpal necrosis/PARL present, NSD in 1 vs. 2 (CaOH2/CHX paste)
4. ***Figini 2008*** – **Cochrane Review** – PN/AP, No difference in success (radiographic) of 1 vs. 2, *1 appt – more pain/swelling (see also Ng)*
5. ***Su/Wang/Ye 2011*** – **Sys. Review/Meta-Analysis** – PN/AP, No difference in healing rate in 1 vs. 2 visit. Less short term pain in 1 visit.
6. ***Peters/Wesselink 2002*** – Necrotic/PARLs: 1 visit (18), 2 visit w/ culturing (21), 4.5 yr recall: 1 visit 81%, 2 visits 71% (NSD); 88% w/ positive culture @obturation healed. Conclusions:
   1. **NSD between 1 visit and 2 visit (w/CaOH2) for PARL healing**
   2. **Positive culture did NOT decrease success** (*refutes Sjogren, Fabricius*)

**Does 2 visit treatment with CaOH2 increase the chances of healing?**

**YES (goal is maximal microbial elimination from the canal system)**

* ***Law 2004*** *– CaOH2 (>7 days) is essential to help ↓ microbial load for healing*

1. *Sjogren 1997 IEJ* – Influence of infection on healing (1 visit): **94%** with negative culture, **68%** with positive culture at time of root filling. *Use intracanal medicament (2 visits) to maximize bacterial reduction.*
2. ***Trope/Orstavik 1999 JOE*** – Compared healing of PN/AP 1 vs. 2 visits w/ CaOH2 as intracanal medicament. **CaOH2 ↑ the rate of healing by 10%** (74% vs. 64%), *Not stat. significant but clinically important*.
3. ***Siqueira; Nair*** – Instrumentation/Irrigation protocols are not effective at reaching bacterial biofilms in anatomical complexities (dentinal tubules, apical ramifications, accessory canals); *CaOH2 necessary to reach these complexities and kill bacteria that may prevent healing of AP*

**Does 2 visit treatment with CaOH2 increase the chances of healing?**

**YES (goal is maximal microbial elimination from the canal system)**

1. ***Vera/Siqueira 2012 JOE***:
   1. In vivo, Mandibular Molars, Mesial roots, Histobacteriological study
   2. Compared 1 visit vs. 2 visit w/ CaOH2 (1 wk) for bacterial status
   3. **↓ Bacterial counts** (improved histobacter. status) in *main canal, isthmuses, dentinal tubules, apical ramifications* (DIALs)
   4. Current instrumentation/irrigation can NOT predictably (100%) disinfect the root canal system (*Anatomical complexities* & *Biofilms*) *Rocas/Siqueira****,*** *Shupping/Trope, Bystrom/Sundqvist, Nair*
   5. *“Entombed bacteria” by GP/Sealer* **(ARs, Lateral canals)**can derive nutrients from PDL or tissue remnants → persistent AP/non-healing
   6. 2 visit w/ medicament necessary to **maximize bacterial reducation** (↓ microbial load for periapical healing**)** before obturation
2. ***Xavier/Martinho/Oliveria 2013 JOE*** – 2 visit w/CaOH2 was more effective at **↓** bacterial endotoxins (LPS) (98% vs. 86%)

**Does 2 visit treatment with CaOH2 increase the chances of healing?**

**NO (goal is to eliminate some bacteria and entomb remaining by gp/sealer)**

1. *Weiger 2000 IEJ* – This study had comparable success results with both single and multivisit treatment (CaOH2) of necrotic teeth w/AP. (93% multi vs 92% single)
2. ***Penesis 2008* - Randomized clinical trial**, Pulpal necrosis/PARL present, NSD in 1 vs. 2 visit (CaOH2/CHX paste)
3. ***Figini 2008* – Cochrane Review** – 2 visit with CaOH2 did not increase the success of NSRCT compared with 1 visit treatment in PN/AP cases
4. ***Peters/Wesselink 2002*** – Necrotic/PARLs: 1 visit (18), 2 visit w/ culturing (21), 4.5 yr recall: 1 visit 81%, 2 visits 71% (NSD); 88% w/ positive culture @obturation healed. Conclusions:
   1. **NSD between 1 visit and 2 visit (w/CaOH2) for PARL healing**
   2. **Positive culture did NOT decrease success** (refutes Sjogren, Fabricius)

**Compare post operative pain / flare-ups between 1 & 2 visit Tx.**

1. ***Trope 1991 IEJ*** – Evaluation of specific preop conditions w/ flare-ups
   1. Single visit w/out AP – no flare-ups
   2. Single visit w/ AP – 1.4% flare-ups
   3. **Single visit ReTx w/ AP – 13.6% flare-up**– statistically the highest risk factor in the study
2. ***Eleazer/Eleazer 1998 JOE*** – Flare-ups: 1 visit: 3%, **2 visits: 8%**
3. ***Ng*** – **1 visit = More post-obturation pain** (along with molars, females, post instrumentation pain and/or swelling)
4. ***Figini*** *– Cochrane Review –* PN/AP, **1 visit > 2 visit for post-op pain/swelling**
5. ***Su/Wang/Ye 2011*** – *Systematic Review/Meta-Analysis* - Infected teeth, NSRCT: **Post-op pain: 2 visits > 1 visit**

**How often does the absence of a radiolucency correspond with histological success ? (BW)**

1. ***Brynolf 1967 Odontol Revy*** – Histologic and radiographic exam indicated complete healing following NSRCT only **7%**. *93% had inflammation despite no radiolucency*.
2. ***Walton 1997 OOO*** – Histologic and radiographic exam revealed *26% of specimens without radiolucencies had inflammation* and **74%** had complete healing. **This study disputes Brynolf’s findings !!!**

**Does Orthodontic movement affect the healing of PA lesions?**

1. ***deSouza* *2006*** – dog study - Ortho movement (5 months) **delayed but did not prevent PA healing** in comparison to NSRCT (2 stage) teeth without ortho movement

**How long does it take to heal?**

1. ***Murphy 1991*** – Retrospective study, Resolution of AP can occur **as early as 3 months,** average rate is **3.2 mm2/month**. **70% of lesions needed > 12 months** for healing.
2. *Bystrom, Sjogren, Sundqvist et al. 1987 EDT* – Failure of apical healing may be due to bacteria outside the canal. Most lesions heal within 2 years, some take as long as 4-5years before bone regeneration is completed.
3. ***Orstavik 1996*** – Prospective study, *Peak incidence of healing or emerging chronic apical periodontitis (PAI ≥ 3)* *occurred at* ***1 year***. **88%** showed **signs of healing by 1 year**. **76%** showed signs of **disease by 1 year**. Complete healing of preoperative AP in some instances required **4 years** for completion.
4. ***Strindberg; Reit*** – up to **4 years** for resolution of PARL

**What factors attect successful healing of a perforation ?**

1. ***Fuss/Trope* – Time, Size, Location. Time** – immediate repair ↑ success (*Seltzer 1970*), **Size** – Smaller the perf, ↑ success = sealing perf, **Location** - *#1 factor* – Critical zone – level of crestal bone/epi/ct att. –bacterial contamination/epithelial downgrowth/sulcular commun.
2. ***Jew 1982 OOO*** – Prognosis depends on **time** lapse since perforation, **location** relative to attachment, **size and sealability** of repair material. Best prognosis: apical or middle thirds. Contamination leads to failure.
3. ***Krupp/Hulsman JOE 2013*** – Retrospective, MTA perf repair (**90** Root perfs), Success: 73% healed at ≥1 year. **2 Prognostic Variables**: 1) **Pre-op RL** w/ defect, 2) **Sulcular commun.** w/ defect (*100% Failure*)
4. ***Mente JOE 2010*** *-* Retrospective, MTA root perf repair (*Furcal, Crestal, Mid root, Apical root*): Success: **18/21** (**86%**) healed at ≥1 year.

**Why do Retreatment instead of Apical Surgery?**

Better success will occur with Retx due to the *ability to determine and eliminate the etiology (missed canal, coronal leakage, incompletely debrided canal system, poor quality obturation – density/length…)*

1. ***Nair, Sjogren, Sundqvist 1990 JOE*** – In the majority of root-filled human teeth with therapy-resistant periapical lesions, microorganisms may persist **in the canal** and may play a role in treatment failures.
2. *Briggs 1997 Br Dent J* – Conventional ReTx is most appropriate first, providing access to the root canal is possible.
3. ***Trope 1998 OOO*** – Surgery should not be considered the primary treatment when root canal treatment or retx **may be readily achieved**.
4. *Lovdahl 1992 DCNA* – Conservative retx should be given priority over surgery in treatment planning.

**Why do Retreatment instead of Apical Surgery?**

1. ***Allen/Newton/Brown 1989 JOE*** – Retrospective study of 1200 cases:
   1. Overall success for retx 65.6%
   2. Surgical retx – 60%
   3. NS Retx – 73%
   4. ReTx of prior ReTx – 47%

Take home message – Try retx first but if unsuccessful next step is surgery.

1. ***Torabinejad*** – Early failures Retx, Late failures Sx
2. ***Zuolo*** – **10%** greater success of Apical surgery when *retreatment completed prior to surgery*
3. ***Riccuci/Siqueira*** – Intraradicular biofilms responsible for AP (NSRCT/RETX cases): CAGE – 6% Extraradicular biofilms. Retx over Sx to treat etiology if possible

**Does a separated instrument affect prognosis?**

Depends on when during treatment it occurs, location of separation, status of pulp and periapex.

1. ***Crump/Natkin 1970 JADA*** – No statistical difference between cases with separated instruments (81%) and control cases w/out separated instruments (73%)
2. *Strindberg 1956 AOS* – Separated instruments decreased success by 14%
3. ***Spilli* –** 3% incidence, NSD in cases with separated instruments or without, only PARL made significant difference
4. ***Pantivisai*** – *Systematic Review/Meta-analysis,* presence of separated instrument **does Not** affect success, PARL does affect success based on outcome studies available

**Implant vs. NSRCT**

1. ***Doyle* 2006** – Retrospective outcome study. Single tooth implant vs. NSRCT/restoration: Success: 73.5% vs 82.1%, **Survival with intervention: 18% vs. 4%.** **Failure: 6.1% vs. 6.1%***. Implants* ***>4x*** *incidence of post-operative complications requiring subsequent treatment intervention*.
2. ***Iqbal/Kim 2007*** – **Systematic review/Meta-analysis**. *No difference in the survival outcome between NSRCT/crown and single tooth implant*. Widely differing criteria are used to measure “success”. NO agreed definitions!!
3. ***Kim/Solomon* *2011*** – **Cost effectiveness**: EMS > Retx/Crown > FPD > Implant/Crown
4. ***Woodmansey* *2009*** – Compared Mand molar NSRCT/Crown & Implant/Crown vs. contralateral natural tooth for ***maximum bite force****,* ***chewing efficiency****, and areas* ***of occlusal contact****/near contact.* Findings: **Endo tx tooth = Contralateral > Implant** for more effective occlusal contact during masticatory function. Implant/crowns have reduced *masticatory function* (one goal of tooth restoration)

**Vital Pulp Therapy & Immature Teeth**

Vital Pulp Therapy: Requirements for Success

1. Treatment of Non-inflamed Pulp:
   1. *Tronstad* – Direct pulp capping of inflamed pulp tissues yields inferior success rates
   2. Pulp tissue must be removed to level of noninflamed pulp
2. Bacteria Tight Seal (see also: *Murray, Murray/Smith* – RDT/Microleakage)
   1. *Cox/Bergenholtz* - Successful healing will occur independent of pulp capping material if exposed pulp is effectively sealed from bacterial leakage
3. Pulpal Dressing:
   1. CaOH2 – *Schroeder; Holland* – liquefactive necrosis superficial layer, coagulative necrosis at junction of necrotic/vital tissues, mild inflammatory response, healing with hard tissue barrier
   2. MTA – *Torabinejad; Holland; Nair -* Healing w/MTA showed complete tubular dentin bridge formation and No inflammation in any of the pulps capped with MTA, Better healing than with CaOH2

Who described apexification of nonvital teeth and what are the possible outcomes? Al Frank

Nonvital immature teeth treated with CaOH2 developed 4 different types of barrier formations. Was the 1st to describe technique.

1. periapex closes with definite recession of the root canal
2. obliterated apex develops without any change in canal space
3. no radiographic evidence of development in canal or apex; an apical stop is evident clinically.
4. calcific bridge forms coronal to apex that is detectable radiographically.

# How long does apexification take?

***Cvek* – 18.2 months**; *Yates* – 9 months; *Kleirer* – 12 months

Considerations for Immature teeth to prevent fractures during apexification

***Trope*** – strengthen cervical portion of immature teeth with *composite* during apexification to prevent fractures.

*Goldberg* – use resin modified glass ionomer after apexification to increase resistance to fracture in immature teeth with total crown loss.

***Kerekes*** – 30% Fractures after long term CaOH2

# Materials used to form apical barrier in cases with an open apex

Dentin Chips

1. *Brady* – apical dentin plug promotes a severe periapical response and inhibits cementum/bone formation

Ca(OH)2

1. *Torabinejad* – CaOH2 induction of root end closure (apexification)
2. ***El-Meligy*** – CaOH2 = MTA for Apexification success

MTA

1. ***Andreasen*** – in a guide for traumatic injuries, he recommends:
   1. MTA apexification after *2-4 wks of CaOH2*, *MTA thickness* should be***4 mm (****Lawley****;****Al-Kahtani* 4 mm >2 mm-leakage)
2. *Torabinejad* – Apexification w/MTA, place CaOH2 for 1 wk in infected cases, place MTA, close w/wet cotton/cavit, obturate 4 hours. Induces formation of cementum.
3. ***Holland;Baek*** – MTA permits cementum attachment/growth over surface and reattachment of PDL (extrusion of MTA not an issue)
4. ***Andreasen***– Long term MTA does NOT ↓ dentin fracture strength

**MTA Artificial Barrier Technique (Open Apex)**

**4 mm** apical plug MTA (***Lawley; Al-Kahtani***), GP or composite coronally

1. ***Mente 2013*** – Cohort study- **252** open apex teeth treated with MTA apical plugs, Min follow up **12 months** (avg 21 months), Findings: **90% Healed** (85% w/AP, 96% w/o AP), *Presence of AP significantly ↓ Prognosis*. (Healed = PAI ≤2, No clinical signs/symptoms)
2. ***Jeeruphan/Hargreaves 2012*** – Pulpal regeneration vs. MTA apexification vs. CaOH2 apexification, Immature necrotic teeth. Minimum **6 month** Recall. Findings Root length: Revasc>MTA>CaOH2; Root width: Revasc>CaOH2>MTA; **Survival (Healed + Healing): Revasc (100%) >MTA Apex. (95%)>CaOH2 Apex. (77%)**
3. ***Witherspoon JOE 2008*** – Retrospective study, **144** open apex teeth treated with MTA apexification in 1 visit or 2 visit (w/CaOH2). Recall **1 year**. Success (Healed + Healing): **1 visit = 93.5%, 2 visit = 90.5% NSD**

See also *Holden/Schwartz*: 85% Healed at 12 months

**What is the prognosis for formocresol pulpotomy in Primary teeth? (Formocresol = Buckley)**

1. *Shelton 2000 Ped Dent.* – Success rates are: 93% for indirect pulp cap, 74% for formocresol pulpotomy
2. *Fuks 1997 Ped Dent*. – This study compared the use of ferric sulfate with formocresol for use in pulpotomy in primary teeth. NSD was found in the success rates between the two materials. 92% ferric sulfate vs 84% formocresol. Diluted Formocresol as Successful as Full Strength!
3. *Waterhouse 1995 EDT* – This study reviewed the success rates for pulpotomies in primary teeth with various medicaments.
   1. Formocresol 55-98% - cytotoxic, mustgenic, carcinogenic
   2. CaOH2 31-100%
   3. Glutaraldehye 82-98%
4. *Holan/Fuks* 2005 – Pulpotomy Success (16 mos): MTA: 97%, Formo: 83%

**Discuss the prognosis of direct pulp cap tx for carious exposures**

1. ***Barthel 2000 JOE*** – Retrospective study after 5 & 10 years of pulp cap success (CaOH2): *Time dependent failure of CaOH2 (see Mente)*
   1. **44.5% failures, 5 yrs // 79.7% failures, 10 yrs**
   2. 18.5% questionable, 5 yrs // 7.3% questionable, 10 yrs
   3. 37% successful, 5 yrs // 13% successful, 10 yrs
2. *Lovschall 2002 Endo Topics* – Vital pulp therapy highly successful with careful case selection and observation of intricacies of technique. (97% at 1 yr, 82% at 5 yr)
   1. Case selection – no clinical or radiographic signs of pulpitis
   2. Technique –
      1. Gentile
      2. No interference of blood clot between pulp and material
      3. Do not introduce infected dentin chips or material into pulp

**Discuss the prognosis of direct pulp cap tx for carious exposures**

1. *Langeland 1971 OOO* – Best tx for carious pulp exposure is teeth with complete roots is RCT, since enough toxic products remain in pulp to maintain inflammation.
2. ***Tronstad 1972 OOO*** – Direct pulp capping of carious pulps had less than a 50% chance of success. It **should be considered IP** and RCT provided.
3. ***Mente*** – Direct pulp capping with MTA vs. CaOH2, **1+ yr recall**, ***Caries or Mechanical, Immature/Mature.***  **MTA: 78%, CaOH2: 60%.** Teeth restored ≥ 2 days after pulp cap had significant ↓prognosis. **MTA better for direct pulp cap.**
4. ***Bogen*** – Young teeth (**OPEN apices**), **1-9 year** recall, ***Reversible Pulpitis and Carious pulpal exposures*,** **Success: 98%**

**What are the properties of the dentin bridge formed?**

1. *Pisanti 1964 Jdent Res*. – Calcium in the newly formed tertiary dentin comes from the pulp and not from the CaOH2 base.
2. ***Nair*** ***2008***: **MTA: @ 1 week – no inflammation, @ 3 months – compact barrier formation.** **Dycal: @ 3 months** – presence of **persistent inflammation** and hard tissue barrier with **tunnel defects**. **MTA > CaOH2 for inflammation and dentinal bridge formation**
3. *Goldberg 1984 JOE* – Dentinal bridge formed with CaOH2 is porous and permeable.
4. ***Holland/deSouza 1999*** – MTA reacts with tissue fluid to form CaOH2 resulting in **hard tissue formation in similar manner as CaOH2**. MTA also releases **soluble growth factors to stimulate hard tissue (reparative dentin) formation (*Smith*)***.* Source of Ca+2 is pulp.

**Discuss Indirect pulp capping pros and cons ?**

1. *Massler 1977 OOO* – Pain is the most important diagnostic tool in deciding vital pulp therapy. Deep carious lesions w/o exposure are AFFECTED and will repair themselves. Exposed lesions are INFECTED w/ bacteria.
2. *Stanley 1966 OOO* – Following operative procedures, the formation of tertiary dentin began at 19 days and the average formation rate was 1.49 micrometers/day.
3. ***Reeves/Stanley 1966 OOO*** – If bacteria were 1.1 to 2.4 mm from the pulp, little pulpal pathology was observed. **If bacteria were within 0.5mm or invaded reparative dentin, irreversible pulpal damage was observed.**
4. ***Langeland 1987 EDT*** – “Affected” hard dentin of cavity floor contains bacteria, therefore indirect pulp capping is not a good idea.
5. ***Murray/Smith*** – RDT <0.5 mm = injury to odontoblasts, microbial leakage into pulp

**Histology: MTA vs. CaOH2 for Direct Pulp Capping?**

1. ***Holland 2001 Dent Trauma*** – Comparison of CaOH2 vs. MTA for pulp capping in dogs. **Healing w/MTA** showed **complete tubular dentin bridge formation and no inflammation**. Mechanism of action believed to be similar to CaOH2. **MTA provided a superior bacteria-tight seal.**
2. ***Nair 2008*** – Human RCT, Compared MTA vs Dycal for iatrogenic pulp capping (healthy 3rd molars). **MTA: @ 1 week – no inflammation, @ 3 months – compact barrier formation.** **Dycal: @ 3 months** – presence of **persistent inflammation** and hard tissue barrier with **tunnel defects**. **MTA > CaOH2 for inflammation and dentinal bridge formation**
3. ***Smith*** - MTA stimulates the release of **soluble growth factors** (i.e.: DSPP, BMP, TGF) from dentin, **promoting reparative dentin formation**. Avoid RMGIs (i.e.: vitrebond), composite resins – intense inflammatory response, greater bacterial peneration and cytotoxic effects

**MTA vs. CaOH2 Pulp Capping**

1. *Pitt Ford/Torabinejad 1996* – Introduced MTA as pulp capping material
2. ***Farsi 2006*** - 30 Young Permanent Molars (73% open apices) ***Asymptomatic and Carious pulpal exposures***, MTA Pulp cap, 12-24 month Recall, Success (Clinical/Radiographic/Cold Testing**): 93% @ 24 months**
3. ***Bogen*** ***2008*** – 53 teeth (15 open apices, ages 7-45) w/ ***Reversible Pulpitis and Carious pulpal exposures*,** 10 min NaOCl/cotton pellet hemostasis, MTA Pulp cap (2 visits), 1-9 year Recall, Success (Clinical/Radiographic/Cold Testing): **98%**
4. ***Mente 2010*** – 108 teeth (C.C.), Direct pulp cap with MTA vs. CaOH2, 1+ yr recall, ***Caries or Mechanical, Immature/Mature.*** **MTA: 78%, CaOH2: 60%.** Teeth restored ≥ 2 days after pulp cap had significant ↓prognosis. No time dependent ↓ in success of MTA pulp cap (CaOH2 did ↓). **MTA appears to be superior to CaOH2 for pulp capping (Not statistically signficant)**

**MTA vs. CaOH2 Pulpotomy**

1. ***Barrieshi-Nusair 2006*** – Evaluated MTA for Partial Pulpotomy in *young*permanent molars with ***carious exposures*** and ***Reversible Pulpitis***/***Normal periradicular tissues***. 24 month Recall. Success: **28/28 (100%)** (Clinical/Radiographic). 78% responded to sensibility testing at recall.
2. *El-Meligy 2006* – Evaluated MTA as Pulpotomy agent in *young* permanent teeth, 1 year Recall, Success (Clinical/Radiographic): **CaOH2 13/15, MTA 15/15.** MTA is suitable for pulpotomy. **Better dentinal bridge with MTA** (See *Holland; Torabinejad; Nair*)
3. ***Witherspoon 2008*** – 19 *immature* vital teeth (ages 7-15) with ***carious or traumatically*** *exposed pulps* diagnosed with ***Symptomatic*** ***Irreversible Pulpitis*** were treated with MTA Pulpotomies (vital pulp therapy). 21 month Recall. **18/19 (95%)** were classified as **healed or healing** (cont. root growth, asymptomatic). 75% responded to sensibility testing at recall.

**Vital Pulp Therapy Review – Witherspoon JOE 2008**

1. Goals: Eliminate bacterial infection, Treat uninflammed pulp, Create bacterial tight seal, Allow continued root development and pulp vitality for function
2. Properties of Material: Antibacterial, Bacterial tight seal, Induce hard tissue formation, Non-cytotoxic to tissues
3. MTA:
   1. **Antibacterial** (facultative only - *Lovato/Sedgley*)
   2. **Resists bacterial leakage** (*Torabinejad; Fischer*)
   3. **Induces hard tissue formation** with more complete barrier formation/less inflammation of pulpal tissues (*Holland; Nair*)
4. Direct Pulp Capping: *Farsi (93%)*, *Bogen (98%), Mente (78%)*
5. Pulpotomy: *Barrieshi-Nusair* (100%)-PP, *Witherspoon* (95%)-FP
6. Technique: Affected pulpal tissue removed w/highspeed diamond bur, Flush with **6% NaOCl** for up to **10 mins** (*Cox*). Note: Avoid direct cotton pressure to pulp – traumatizes tissues, leaves fiber remnants. **2 mm MTA** layer over tissue. Flowable compomer/G.I./RMGI placed followed by bonded c.r.
7. Assessing health of pulp tissue: Ability to gain *hemostasis with 6% NaOCl!!*

**What is the effect of pulp disease/trauma in primary teeth? Does it affect the permanent tooth? Does Tx?**

1. *Andreasen 1978 Int J Oral Surg* – NO effect on odontogenesis of permanent teeth in monkeys after induced pulpal and periradicular inflammation
2. *Holan 1992 EDT* – Trauma of primary dentition treated with RCT induced enamel defects in permanent teeth as compared to extraction or no treatment. Despite findings RCT is still recommended as opposed to extraction to prevent speech problems, premature eruption and / or malalignment problems or affect the child’s self image.
3. *Sonis 1987 J Pedo* – Traumatized ant tooth which becomes necrotic w/out radiolucency or clinical pathosis will not effect developing succedaneous tooth. If pathology develops, ext will minimize potential effect to permanent tooth.
4. *Torneck 1982 DCNA* – Trauma to primary tooth may alter development of the permanent success

**Is Formocresol Safe?**

Formocresol is Buckley’s Formula 1:5 dilution

1. *Pashley 1980* – Dog study – formocresol was detected throughout the body (spleen, liver and kidney). Systemic spread is possible.
2. *Sipes 1986* – States that use is questionable due to **potential mutagenicity, carcinogenicities and humoral immune response**. Formo will cause tissue damage when not used carefully*.*
3. *Ribeiro 2004 JOE* – Formocresol, paramonochlorophenol and calcium hydroxide do not promote DNA damage in mammalian cells.

**Regenerative Endodontics**

**Regeneration vs. Revascularization**

**Regeneration**

Definition - Biologically based procedures designed to *replace damaged structures,* including dentin and root structures, as well pulp-dentin complex cells

Examples:

1. Tissue engineering – Placement of Stem cells, Scaffolds, and Growth factors into pulp space to form new pulp-dentin complex
2. Replication of embryonic tooth formation/artificial tooth germs

**Revascularization**

Definition – Restoration of the *vascular supply* to the pulp-dentin complex

Examples:

1. Triggering bleeding into an empty root canal space similar to blood clot in surgical wound healing (*no use of stem cells or growth factors*)

**Goal of Regenerative Endodontic Procedures:** Regeneration of theentire *pulp-dentin complex* (cellularity, vascularity, dentin/pulpal tissues)w/in exisitng tooth utilizing bioengineering principles of stem cells, scaffolds, and growth factors

**Tissue Engineering (Triad) – SSGs – Stem cells, Scaffolds, GFs**

**Postnatal Mesynchymal Stem Cells (5 types):**

1. SCAP – Stem Cells of Apical Papilla
   1. ***Nakashima*** – periapical tissues contain an enriched population of mesychymal stem cells; Triad of stem cells, scaffolds, growth factors
   2. ***Lovelace/Hargreaves*** – Evoked bleeding in canal released *600 fold increase of mesynchymal stem cell markers* into the canal system of immature teeth, possibly SCAP cells
2. SHED – Stem cells of Human Exfoliated Deciduous teeth (primary teeth)
3. DPSC – Dental Pulp Stem Cells
   1. ***Gronthos 2000*** – isolated DPSCs, implanted in mice, dentin-like, pulp-like, and odontoblast-like cells formed
4. DFPC – Dental Folicle Progenitor stem Cells
5. PDLSCs – Periodontal Ligament Stem Cells

*Postnatal stem cells are* ***multipotent***

*Embryonic stem cells are* ***omnipotent/totipotent or pluripotent***

**Tissue/Bio-Engineering (Triad)**

* **Scaffolds** – *3-D support for spatial positioning of stem cells/growth factors* and regulation of growth/metabolism, supports proliferation/differentiation

1. Natural
2. PRP – Platelet Rich Plasma
3. PRF – Platelet Rich Fibrin
4. Collagen
5. Blood clot (Endo Regen Procedure)
6. Synthetic
   1. PLA (polylactic acid), PGA (polyglycolic acid), PLGA, bioceramics, hydrogels, fibrin gels

* **Growth Factors** – bind to stem cells to *trigger proliferation/differentiation*

1. Fibroblastic Growth Factor
2. *TGF-β (Zhao)*
3. NGF
4. *BMP*

**Revascularization/Revitalization**

HISTORY:

***Nygaard Otsby*** **1961**

1. 1st to evaluate regenerative endodontic procedures – described revascularization procedure of lacerating the periapical tissues to form blood clot within the canal space (Based on surgical wound healing principles)
2. Found ingrowth of connective tissue into the canal space and cementum deposition on canal walls.

***Banchs/Trope 2004*** - Case report – 11 yo male/open apex/AP, #29, Revasc

(See also ***Iwaya 2001*** - 1st case report on revascularizaton)

**Is Regeneration considered Apexification or Apexogenesis?**

Apexogenesis: *Vital pulp therapy* aimed at continued physiologic root development with No loss of vascularity (no need to revascularize the canal space)

Apexification: Inducing calcific barrier in a root with an open apex or continued root development of an incompletely formed root in teeth with Necrotic pulp

* Regenerative Endodontics would be considered *Apexification*

**Currently in Regenerative Endodontics**

Source: AAE

Current Regenerative Endodontic Protocols rely on:

1. Irrigants to disinfect (1.5% NaOCl, TAP) and release of *growth factors* found in dentin – DSPP, TGF, NGF (17% EDTA)
2. Stimulate bleeding from the periapical tissues to promote influx of *stem cells (SCAP) and growth factors*
3. Blood clot and dentinal walls provide the *scaffold* for generation of new tissues

Future Regenerative Endodontics:

1. Autologous stem cells seeded on customized scaffolds and delivery of appropriate growth factors
2. Hydrogels and autologous stem cells

**Regenerative Endodonic Procedures (REPs)**

*Law JOE 2013* – Considerations for Regenerative Procedures

Case Selection:

## Tooth with necrotic pulp and an immature apex (*Kling/Cvek* AF = 1.1-5 mm)

## Pulp space not needed for post/core, final restoration

## No known allergies to antibiotics if intended for use

## Compliant patient (parent/guardian)

# Informed Consent

#### Two (or more) appointments

#### Use of antimicrobial(s)

#### Possible adverse effects: staining of crown/root, lack of response to treatment, pain/infection

#### Alternatives: MTA apexification, no treatment, extraction (when deemed non-salvageable)

#### Permission to enter information into AAE database (optional)

**Regenerative Endodontic Procedures**

## First Appointment

### Local anesthesia, rubber dam isolation, access

### Copious, gentle irrigation with 20ml 1.5% NaOCl using an irrigation system that minimizes the possibility of extrusion of irrigants into the periapical space (e.g., needle with closed end and side-vents, or EndoVac). The lower concentrations of NaOCl are advised, to *minimize cytotoxicity to stem cells* in the apical tissues. (*Essner/Eleazer*)

### Dry canals

### Place antibiotic paste or calcium hydroxide:

### Ca(OH)2 is antimicrobial at concentrations that do not induce stem cell toxicity and is widely available

### As an alternative, if the triple antibiotic paste is used:

### consider sealing pulp chamber with a dentin bonding agent (to minimize risk of staining)

### mix 1:1:1 ciprofloxacin:metronidazole:minocycline in a lower concentration (0.01-0.1 mg/ml) to avoid stem cell toxicity; these lower concentrations appear as a *liquid form* and are no longer a paste (*Ruparel*)

### 1:1 Cipro:Metro also eliminates the staining from minocycline.

#### Deliver into canal system via Lentulo spiral, MAP system or syringe

#### If triple antibiotic is used, ensure that it remains below CEJ (minimize crown staining). As an alternative, Ca(OH)2 does not cause staining.

#### Seal with **3-4mm Cavit**, followed by IRM, glass ionomer cement or another temporary material

### Dismiss patient for 3-4 weeks

*Kakoli* – infection of dentinal tubules is deeper and affects more tubules in young patients vs older patients … possible complication to disinfection protocol

**Regenerative Endodontic Procedures**

### Second Appointment

#### Assess initial treatment: If signs/symptoms of persistent infection, consider additional treatment with the antimicrobial, or an alternative antimicrobial. Recall the patient in about 3-4 weeks as before.

#### Anesthesia: 3% mepivacaine without vasoconstrictor, rubber dam

#### Copious, slow irrigation: 20ml 17% EDTA, followed by normal saline, using a similar closed end needle.

#### Dry with paper points

#### Create bleeding into canal system by over-instrumenting (endo file, endo explorer, or 17% EDTA dipped endo explorer)

#### Stop bleeding 3mm from CEJ and place CollaPlug/Collacote (Petrino)

#### Place 3-4 mm of MTA and reinforced glass ionomer and place permanent restoration. Glass ionomer may be an alternative to MTA in cases where discoloration of the crown is a potential concern (Giesler)

**Regenerative Endodontic Procedures**

Triple Antibiotic Paste:

1. ***Hoshino*** –1:1:1 Ciprofloxacin, Metronidazole, Minocycline

* Effective for eradicating bacterial infection from infected dentin
* Creating environment for ingrowth of vascularity and regenerative cells

1. ***Windley/Trope* –** dog study, infected canals, 1.25% NaOCl irrigation = 10% teeth bacteria free, 2 wks triple antibiotic paste =***70%*** teeth bacteria free
2. ***Law 2013* (review)** – 2-4 weeks for TAP medicament interappt

Irrigation: 1.25% NaOCl (1st appt), 17% EDTA (2nd appt), NO CHX

1. ***Essner/Eleazer*** – *Higher conc. of NaOCl are cytotoxic to stem cells*, tested .04% - .33% NaOCl
2. *Martin 2012* – Higher conc. of NaOCl ↓ DSPP capacity to *induce SCAP differentiation*
3. ***Trevino 2011*** – *17% EDTA best supported SCAP cell survival*, protocols with *2% CHX lacked any viable stem cells*

**Regenerative Endodontic Procedures**

Triple Antibiotic Paste vs. CaOH2: *Balance of antibacterial effects and stem cell survival*

1. *Chueh* - CaOH2 as intracanal medicament, no staining
2. ***Bose/Hargreaves*** – Case series,TAP > CaOH2 for *root wall width* growth
3. ***Law* *2013*** – TAP (*0.01-0.1 mg/mL*) or CaOH2; consider eliminating Minocycline or use Dentin bonding agent (*Kim*) to avoid staining
4. ***Ruparel 2012*** – TAP, DAP, and Augmentin at currently used clinical concentrations (thick slurry pastes >0.1 mg/mL) are cytotoxic to SCAP cells. CaOH2 supported SCAP survival at all concentrations

Anesthetic: 3% Mepivicaine w/o epinephrine

1. ***Petrino 2010*** – Use 3% Mepivicaine w/o vasoconstrictor – the use of a vasconstrictor may *impede the ability to induce bleeding* in the canal, the crucial step for attaining a scaffold and delivery of stem cells

**Regenerative Endodontic Procedures**

Irrigation: 17% EDTA (2nd appt), NO CHX

1. ***Trevino 2011*** – 17% EDTA best supported SCAP cell survival, protocols with 2% CHX lacked any viable stem cells
2. *Ring/Murray* – Irrigants effect stem cell adherence to dentin
3. ***Galler*** – EDTA promotes exposure of growth factors in dentin, differentiation of stem cells into odontoblast like cells and adhesion of those cells to dentin

Collagen matrix for MTA placement:

1. ***Petrino 2010***– Collagen matrix (Collagplug or Collacote) may be used as a matrix barrier for MTA placement

MTA Barrier: (3-4 mm) – (4 mm seal: *Al-Kahtani; Lawley*)

1. ***Holland; Nair*** – MTA promotes complete hard tissue barrier w/no infl.
2. ***Torabinejad/Parirokh****–MTA is biocompatible, osteoconductive/inductive*

**Regeneration Success**

*Hargreaves 2012, Law 2013*

Clinical criteria: No pain/swelling, no pain to percussion/palpation, no sinus tracts Radiographic criteria: Healing of AP, *Continued radiographic root development: Increased root width, Increased root length*

**Recall time**: **12-18 months** (*Cheuh, Bose/Hargreaves, Law)*

1. ***Bose/Hargreaves 2009*** – Retrospective, compared differences in root lengthening and root wall thickening for various intracanal medicaments (TAP, CaOH2, and Formocresol). **Root Length: TAP = CaOH2** >> Controls (NSRCT or MTA apexification), **Root Width: TAP >> CaOH2 >>** Controls. **12-18 month recall**.
2. ***Jeeruphan/Hargreaves 2012*** – Retrospective, 61 teeth, Pulpal regeneration vs. MTA apexification vs. CaOH2 apexification, Immature necrotic teeth. Findings Root length: Revasc>MTA>CaOH2; Root width: Revasc>CaOH2>MTA; **Survival: Revasc (100%) >MTA (95%)>CaOH2 (77%) – >6 months (avg 14 months) recall**

**Regeneration Outcomes – Histological Evaluation**

1. ***Wang/Thibodeau/Trope JOE 2010*** – Histological characterization of regenerated tissues in canal space following revascularization procedures in immature dogs teeth: ***Cementum-like, Bone-like, and PDL-like tissues*** *within the canal space*
2. ***Torabinejad/Faras JOE 2012*** – Histological evaluation of canal contents of immature human premolar treated with regenerative endodontic procedure and *PRP scaffold*: **Vital Pulp-like connective tissues**
3. ***Shimizu JOE 2013*** – Histological evaluation of *immature human maxillary incisor* treated with revascularization procedure (26 months): ***Cementum-like and Bone-like tissues*** *within canal space.* ***No pulp-like connective tissues observed****.* Need new regenerative protocols to promote regeneration of pulp tissues within canal space

**Medically Compromised**

**What is Sickle cell anemia, how does it effect the root canal?**

***Kaya IEJ 2004*** – SCA is a genetic and systemic disease which may cause *pulp necrosis without necessarily having an identifiable etiology*. SCA causes radiographically observable differences in jaw structure, especially in the mandible. The clinical problem is directly associated with the *defective RBC*. The patients are prone to infection because the *macrophages are involved in the phagocytosis of the RBC* and not available for destroying bacteria. The distorted cells may also occlude the Microvasculature and impede blood flow to an area. This mechanism is suspected by ***Ingle & Taintor 1985*** to be the cause of pulpal necrosis and repeated episodes of pain as described by ***Andrews/England 1983*** in sickle cell patients

***Costa JOE 2013*** – **8.33x higher incidence of PN with Sickle Cell patients**

Radiographic observation- **“stepladder”** appearance of the widening trabeculation due to increased marrow space (**increased hematopoiesis/RBCs**)

**What are characteristics of Vit D resistant rickets ?**

***Bender & Naidorf 1985 JOE –***

1. **Pulp horn extension into the DEJ** is pathognomonic for Vit. D resistant rickets
2. Clinically: *frontal bossing, bowing of legs*, short enlarged wrists and ankles
3. Dental: *hypoplastic/hypocalcified enamel*, draining sinus tract, gingival swelling, ***apical abscesses***
4. Radiographic*:* ***enlarged pulp chambers***, wide root canals, and loss of lamina dura, ***shortened roots***

**What are the characteristics of hyperparathyroidism ?**

Primary – caused by *adenoma (80%)*, carcinoma of the parathyroid or PTH release from ectopic malignant tumor.

Treatment – surgical removal of parathyroid

Secondary – caused by *Chronic kidney disease, Vit D deficiency, Calcium malabsorption states (↓ Ca, ↑ phosphate, K)*

Treatment – renal dialysis or transplant

**Classic signs = stones, bones, groans**

1. Ectopic calcifications – **kidney stones**
2. **Bone lesions**
   1. Lytic lesions (**brown tumors = central giant cell granuloma**)
   2. Ground glass appearance with decreased trabeculation
   3. Loss of lamina dura
3. **Vague abdominal pain**, **bone pain**, fatigue, weakness
4. Emotional liability, **psychoses**

**Diabetes**

***Bender 2003 JOE*** – Inherent factors of the disease:

1. Prone to **bacterial or opportunistic infections** (see below)
2. Vulnerability caused by a generalized circulatory disorder (**peripheral microvascular collapse)**
3. Blood vessels are damaged by the *accumulation of atheromatous deposits* (**macrovascular disease)**
4. Capillaries develop a *thickened basement membrane (***microvascular diesease)**
5. **Impaired leukocyte chemotaxis and immune cell delivery**
6. Decreased PMN microbicidal ability
7. **Due to limited pulpal/periapical circulation,** patients are **more prone to infection** and **impaired healing!!**

***Fouad et al 2003 JADA*** – Preoperative periradicular lesions and a history of diabetes have a significant reduction in successful outcome for endodontic treatment. **PARL + Diabetes = ↓Success of NSRCT (*impaired healing)***

***Segura 2005; Marota/Siqueira 2012*** – Type 2 Diabetics have an increased prevalence for AP *(****prone to infection****)*

**Diabetes**

1. Types:
2. **Type I** – loss of insulin producing beta cells of the islets of Langerhans in pancreas leading to insulin deficiency; immune-mediated – T cell attack; Prevalence: 10%; Sudden onset; Childhood; Ketoacidosis common
3. **Type II** – insulin resistance +/- reduced insulin secretion (insulin receptor); unknown defect; Prevalence: 90%; Gradual onset; Adult; Ketoacidosis uncommon
4. Other types: Gestational, Prediabetes, LADA
5. Signs/Symptoms: (Cardinal) Polyuria, Polydipsia, Polyphagia, Wt loss
6. Complications: *Diabetic retinopathy, Diabetic nephropathy, atherosclerosis/ischemic heart disease, peripheral vascular disease*
7. Testing: **HbA1c** (Glycosolated hemoglobin: measures 2-3 month prior glucose levels): **<6% Normal, <7% Well controlled diabetic**
8. Diagnostic criteria: **Fasting glucose: >126 mg/dL, 2 hr glucose: >200 mg/dL, HbA1c: >6.5%**

**Coronary Heart Disease/Coronary Atherosclerosis/Ischemic HD**

*Symptomatic* Coronary Atherosclerosis aka Ischemic Heart Disease:

* Angina, possible MI
* Oxygen deprivation due to reduced blood flow to myocardium

Stable Angina:

* Predictable, reproducible, ppt by physical effort, relieved by cessation of exercise/rest/Nitro. *Limit EPI, short appts, stress reduction, N2O2, vitals*

Unstable Angina:

* New, Increasing in frequency/intensty, ppt at rest, not relieved by Nitro
* Changing pattern, poorer prognosis, MI likely
* *Avoid elective care,* *consult phys.*,monitor ECG/Pulse ox/BP, N2O2, Nitro

**-Avoid NSAIDs** (excluding Aspirin) with **hx of MI or Stroke** (*Olson*)

**-Avoid EPI in Unstable Angina**/**recent** **MI, Limit** **2 carps in Stable Angina**

***Frisk*** – No significant association between coronary heart disease (CHD) and RCT treated teeth or teeth with AP

***Rodriguez JOE 2014*** – Suggests association between coronary heart disease and teeth with AP

**HTN**

Classifications of BP:

1. Normal: <120, <80
2. Pre-Hypertensive: 120-139, 80-89
3. Stage 1 HTN: 140-159, 90-99
4. Stage 2 HTN: ≥ 160, ≥ 100 – If Symptomatic: Emer tx only, Refer immed.

Systolic = Arterial pressure at peak ventricular contraction (more sign. >50 yrs)

Diastolic = Resting Arterial pressure

Treatment Considerations:

-**Avoid long term (>2 wks) NSAIDs** in pts taking *anti-hypertensive medications* (ACEIs, Beta blockers, Diuretics, Alpha/Beta blockers)

-**Limit Epi to 2 carps w/ Non-selective Beta blockers** (Propranolol, Coreg): *Block Beta 2* which maintains *normal peripheral vasodilatory tone* → *Unopposed alpha stimulation (peripheral vasoconstriction, ↑ BP, ↓ HR)*

-**Avoid EPI in Uncontrolled HTN, limit 2 carps in controlled HTN**

-**Defer *elective* dental tx if >180/110**

-Orthostatic Hypotension: Temporary **↓** in BP, Dizziness/light-headed/Fainting

**Medications**

**HTN/CAD/Angina/Arrythmias/CHF:**

1. **ACE Inhibitors (“PRILs”): *Enalapril, Lisinopril***

MOA: Acts on *Renin/Angiotensin/Aldosterone system*; Prevents Angiotensin I → II = ↓ arteriolar resistance, cardiac output, ↑ Na/H2O excretion, ↓ HTN

1. **ARBs (“SARTANs”): *Losartan, Valsartan, Olemsaran (Benicar)***

MOA: Blocks activation of Angiotensin II receptor = Vasodilation, ↓ HTN

1. **Beta Blockers (“LOLs”):**

**Selective (β1 only): *Metoprolol, Atenolol, Acebutolol, Bisoprolol***

**Non-Selective (β1 + β2): *Propranolol, Carvedilol (Coreg)***

MOA: Block action of epi/norepi (sympathetic) on β adrenergic receptors; β1-heart, kidneys; β2-lungs, gi, vascular smooth muscle, skeletal muscle

β1:↑ Cardiac output/contractility, Renin release (kidneys)

β2: Smooth muscle relaxation/Peripheral Vasodilation, Bronchodilation

**Medications**

**HTN/CAD/Angina/Arrythmias/CHF:**

1. **Calcium Channel Blockers (“VD-PINEs”): *Amlodipine (Norvasc), Nifedipine, Verapamil, Diltiazem***

MOA: Blocks calcium channels within vascular smooth muscle, cardiac muscle = Vasodilation, ↓ contractility (ionotropic), ↓ HR (chronotropic)

1. **Diuretics: (“IDES”)**

**Loop** (ascending loop)**: *Furosemide***

**Potassium Sparing** (collecting duct)**: *Triamterine, Spironolactone***

**Thiazide** (distal convoluted tubule)**: *Hydrochlorothiazide***

MOA: Prevent kidney resabsorption of Na, Cl, K, H2O = Inc fluid excretion

1. **Combo Drugs:**

***Lotrel*** (Amlodipine/Benazepril) = Ca channel blocker + ACE inhibitor

***Azor*** (Amlodipine/Olemasartan) = Ca channel blocker + ARB

**Medications**

***Anti-Platelet* – Prevention of thromboemboli (dislodged thrombus)**

1. **Aspirin: AcetylSalicylic Acid (ASA)**

* NSAID but more selective for Cox-1 and ***irreversibly*** inhibits Cox enzyme =↓TXA2(↓Platelet aggreg./production); Avoid <19 y.o. (Reye’s)

1. **NSAIDs: Ibuprofen, Naproxen, Indomethacin, Ketorolac, Diclofenac, Meloxicam, Celebrex (Cox-2 selective)**

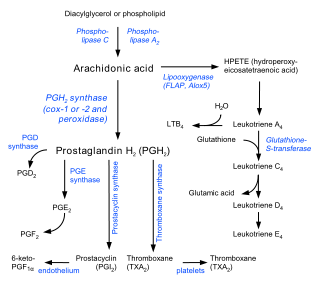
* Analgesic (↓ PGs), Antipyretic (↓ PGs), Anti-platelet (↓Thromboxane A2), Anti-inflammatory; ***reversibly*** inhibits Cox enzyme (AA → PGH2)

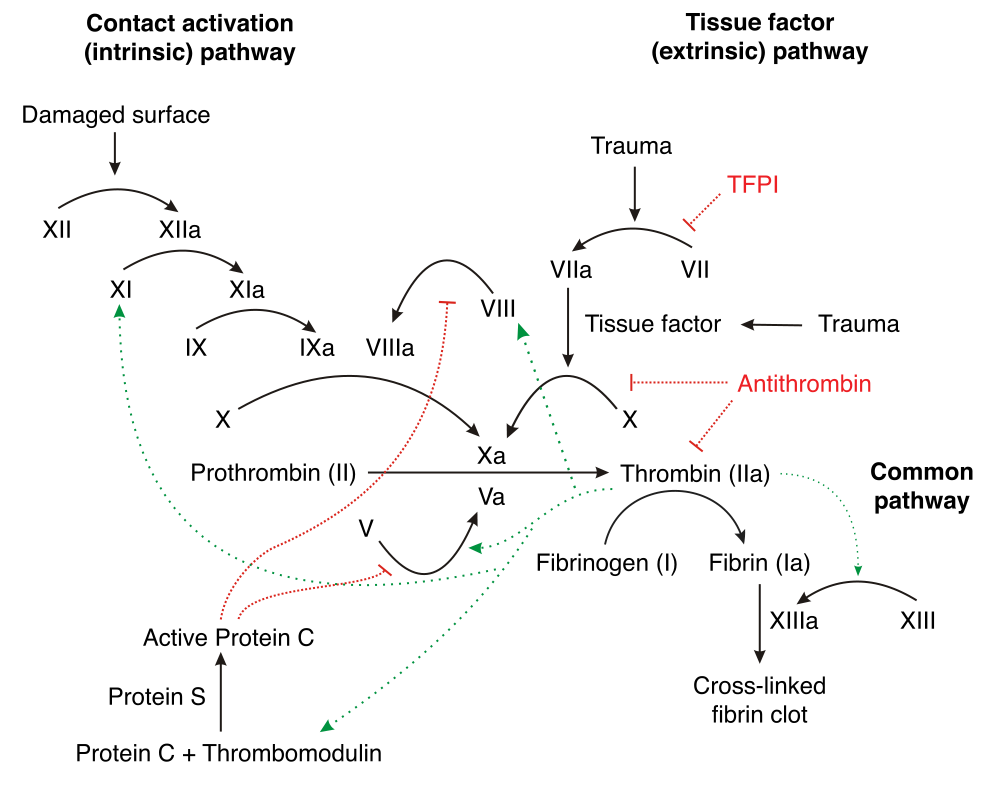
1. **Plavix (Clopidogrel)**

* Alters Platelet fx through ***irreversible*** inhibition of ADP chemoreceptor on *platelet surface*

***Anti-Coagulant* – Prevention of thromboemboli – stroke, MI**

1. **Coumadin (Warfarin)** – Inhibits Vit K-dep. (liver) synthesis of *clotting factors (II, VII, IX, X)*. INR(PT-extrinsic factor VII) **2.0–3.0** = normal range
2. **Heparin (pulmonary emobli)**– Activates Anti-thrombin, prothrom→throm
3. **Pradaxa (Dabigatran)** – Direct thrombin (Factor II) inhibitor , A Fib





**Cardiac Arrythmias**

\*\**Avoid elective dental care in with patients with severe arrythmias*: High grade AV blocks, Symptomatic Ventricular arrythmias, Supraventricular arrythmias

\*\***Avoid Epi in patients taking *Digoxin***

**Digoxin Toxicity**: Hypersalivation, Nausea/Vomiting, Drowsiness, Visual disturbance - YELLOW or GREEN appearance

**\*\*Avoid Epi in patients with severe arrythmias, limit to 2 carps in others**

**MED CONSULT**, Nitrous oxide, Oxygen and Nitro should be used

Atrial Fibrillation – Pradaxa (anti-coagulant/direct thrombin inhib. = factor II) – *check* ***aPTT (intrinsic/common pathways****), use local hemostatic measures*

**\*\*Antibiotics not recommended for Pacemakers/Atrial defibrillators**

***Roedig*** – *In vitro*, Electromagnetic interference with pacemakers – battery powered Curing Light and *Magnetostrictive* Ultrasonic units

***Wilson/Baumgartner*** – ***In vivo***, No Electromagnetic interference with pacemakers/cardio-defibrillators – 2 EALs (Root ZX), 1 EPT

***Gomez*** – *In vitro*, No Electromagnetic interference with pacemaker/defibrillators – *Piezoelectric* Ultrasonic units

**Congestive Heart Failure**

**CHF** = complex clinical syndrome that can result from any structural or functional cardiac disorder that **impairs the ability** of the **ventricle to fill with or eject blood**

1. Etiology: CAD/CHD, HTN, Cardiomyopathy, etc
2. Sequelae: MI/Cardiac arrest… peripheral edema, fluid retention, pulm. HTN
3. **Avoid: NSAIDs, EPI (also for Digoxin)**
4. Dental Tx: **MED CONSULT**
   1. Without limitations: Elective Tx
   2. With limitations/but ok at rest: Elective Tx with Med Consult
   3. Symptoms at Rest: No Elective Tx, Hospital based Tx for emergencies
5. **Medical Consultation**: Physical status, Lab values, Control of condition, Stability, Meds/Recommendations
6. **Nitrous Oxide: Ok to use**
7. **Semisupine/upright position, short/stress free appts/monitor vitals/pulse ox/N2O2 (stress reduction!)**

**Pulmonary Disorders**

**COPD: Chronic Bronchitis & Emphysema**

1. **Chronic Bronchitis (“Blue bloaters”): cyanotic w/chronic cough**
2. *Excessive trachobronchial mucus production*
3. Chronic cough *with* sputum production
4. **At least 3 months for 2 consecutive years**
5. **Emphysema (“Pink puffers”): barrel chested w/exertional dyspnea**
6. *Permanent enlargement of the airways distal to terminal bronchioles*
7. Destruction of the alveolar walls or septa *without* fibrosis

**Defer Tx**: Shortness of Breath at rest, Productive cough, URI, **O2 Sat < 91%**

**Avoid: Respiratory depressants (Sedatives** – Benzos/Flexaril, **Narcotics**, Barbituates**), Nitrous Oxide (*Severe COPD only*)**

Dental Tx: Semisupine position, **Pulse Ox monitoring, O2 (2-3L/min) <95%**

**\*Theophylline:**

1. MOA: inhibits TNFα, Leukotrienes; relaxes bronchial smooth muscle, ↑ HR, Contractility, BP (**non-selective beta agonist**)
2. **Toxicity:** *Nausea/Diarrhea (β2), ↑ HR, Arrhythmias (β1),* *CNS Exc/Seizures*
3. **Adverse Drug Interactions:** Ciprofloxacin, Macrolides (**CMT**)

**Pulmonary Disorders**

**Asthma**

* *Reversible episodes of airway hyperresponsivness*
* Recurrent episodes of wheezing, coughing, and dyspnea
* Extrinsic (smoke, seasonal), Intrinsic (drugs, foods), Exercise, Stress

**Avoid:**

* **NSAIDs/Aspirin** – **Bronchoconstriction** in 10% (40% w/pansinusitis, nasal polyps – triad asthmaticus) due to **build up of AA→Leukotrienes**
* **EPI - Sulfates in LA w/ EPI** – build up of sulfur dioxide – acute attack
* **Respiratory Depressants**–Barbituates, **Narcotics,** **Benzos**, Flexaril

**Nitrous Oxide: Okay to use**, not a respiratory depressant or irritant

If Attack occurs in office:

* **Short acting beta2 agonist (bronchodilator)**: **Albuterol q20 mins**
* **Subcutaneous** injection – **0.3-0.5 mL 1:1000 epi**
* **Oxygen** admin, Monitor **Vitals,** **Pulse Ox**, **EMS**

Treatment: Short acting Beta2 Agonists (albuterol), Long acting Beta2 Agonists, Corticosteroids (Advair), Corticosteroids (oral)

**G.I. Disorders (PUD/CUD)**

1. **Peptic Ulcer Disease**

* Ulceration of the stomach lining or first part of the small intestine (duodenum), *acid related*, *H. pylori* major factor
* **Severe abdominal pain,** bloating, **nausea, vomiting**, loss of apetite

1. **Crohn’s Disease**

* IBD, inflammatory ulceration of **any part of the G.I. tract (mouth – anus)**
* *Autoimmune mediated*, mucosal ulcerations
* Symptoms: **Abdominal pain**, diarrhea, fever, wt. loss, **anemia**, skin rash
* **No cure, Steroids, Immunosuppresants, Biologics, Methotrexate, Sx**

1. **Ulcerative Colitis**

* IBD, *similar to Crohn’s Disease*, intermittent ulcerations of the intestinal tract lining. Marked by **diarrhea, anemia.** Mucosal ulcerations.
* *Unlike Crohn’s* ***only affects the large intestine (colon)***
* Treatment: **Steroids, Biologics (Humira), Colectomy**

**\*\*AVOID: NSAIDs/Aspirin, Clindamycin** (C. dif assoc. pseudomem. colitis), **steroids**

\*\*Caution: Antacids, Proton pump inhibitors “ZOLEs” – ie Omeprazole (H+ pump of gastric parietal cells) interact with certain antibiotics, benzodiazepines, methotrexate

**Liver Disease**

Hepatitis:

* Inflammation of the liver, may be acute (<6 months) or chronic
* **Symptoms: Often asymptomatic, jaundice,** poor apetitie, **malaise**
* Self limiting or leading to *fibrosis/cirrhosis*, poss. Hepatocellular carcinoma
* Causes: Viral (Hepatitis A, B, C, D, E), alcohol, medications, autoimmune
* **Diagnosis: CBC, Liver enzymes (ALT/AST, ALP, Bilirubin), PT time**
* Chronic can have compromised: *liver fx, drug metabolism, hemostasis*

Dental Tx:

* **Acute hepatitis: Emergency only**, Hospital based, Avoid drugs metabolized in liver (LAs, Pain meds), check bleeding/*PT time* – *Extrinisic* pathway (VII)
* **Bleeding issues (Vit K dep clotting factors – Prothrombin(2), 7, 9, 10) – chk CBC, PT, Platelets (thrombocytopenia), Med Consult prior to Surgery!**
* Higher tolerance to drugs: LAs, sedatives, GA, **AVOID Tylenol, NSAIDs**

HCV needlestick: Blood to Blood transmission

* Baseline testing of patient – ***HCV antibody* *enzyme immunoassay (ELISA)***
* Baseline and follow up (6 months) – *HCV antibody* and *liver enzyme activity*
* HCV antibody ELISA postive results confirmed with HCV qPCR or RBIA

**Chronic Kidney (Renal) Disease**

Definition: Progressive loss in renal function, ↓ fluid/K/Cl/Na/H2O excretion

Signs/Symptoms:

* **HTN/CHF (fluid retention/overload)**
* **Hyperkalemia, Hyperphosphatemia, Hypocalcemia (Vit D3 deficiency)**
* Metabolic Acidosis (accumulation of sulfates, phosphates, uric acid)
* **Iron Deficiency Anemia (weakness/pallor/**↑ cardiac output)
* Accelerated atherosclerosis

Causes: **Diabetes Mellitus, HTN,** Chronic Glomerulonephritis, PKD

Diagnosis: **Creatinine (↑ levels),** urinalysis, adbominal ultrasound, **GFR**

Staging: *CKD = <60 mL/min (3 months),* >90/90-60/60-30/30-15/<15 mL/min

**Stage 5 (End Stage Renal Disease) = < 15 mL/min**

ESRD Hematologic Abnormalities:

* **Anemia**: ↓erythropoeitin/RBC production (weakness/pallor/breathless)
* **Leukocyte dysfunction -** ↑ risk of infection, *leukopenia*
* **Platelet dysfunction**: Abnormal platelet aggregation, *thrombocytopenia*
* **Coagulopathy**:Atherosclerosis, ***HTN/CHF***

**Chronic Kidney (Renal) Disease**

ESRD Renal Osteodystrophy & **Secondary Hyperparathyroidism**:

* ↓ Vit D3 production → ↓ Calcium absorption (gastric mucosa)
* ↓ Renal excretion of Phosphate and Potassium (binds free Ca+2)
* ↓ Serum Calcium → ↑ Parathyroid Hormone (PTH) secretion

PTH Secretion → **Secondary Hyperparathyroidism**:

* Inhibits tubular reabsorption of Phosphate
* Stimulates renal production of Vit D3 for calcium absorption
* Sustained levels of PTH → Bone remodeling, Calcium mobilization from bones, Renal and metastic calcifcations:
  + Ostomalacia – increased bone matrix
  + Osteitis fibrosa – **lytic lesions of bone**, marrow fibrosis
  + Osteosclerosis – enhanced bone density
* **STONEs (kidney stones), BONEs (lytic lesions), GROANs (pain)**
* Radiographic Changes: **Loss of Lamina Dura**, Demineralized bone (**Ground Glass**), **RL jaw lesions (CGCGs/Brown’s tumors)**, **Widened Trabeculations**, Metastatic calcifications within the skull

**Hemodialysis**

* Every 2-3 days (4-6 hrs), Heparin admin IV to prevent coagulation
* Antibiotic prophylaxis NOT needed for AV fistula for *routine dental care* but is necessary for I&D Abscess (leukopenia)
* Best day to Tx: Day after hemodialysis
* **Avoid Tx within 4-6 hours of hemodialysis** due to Heparin
* Avoid BP cuff or IV meds on arm with AV Shunt

**ESRD**

* Dental Tx: **Med consult**, Hospital based treatment w/comorbid conditions, **Aggressive management of orofacial infections** (leukocyte dysfunction)
* Surgery: Med consult, **Screening for Platelets, PT, aPTT, TT, bleeding time**, assess need for **Antibiotics** (leukocyte dysfunction)
* Medications/Drugs:
  + Reduce dosages – *eliminated slower*/reach toxic levels faster
  + Avoid most pain meds, antibiotics – **Tylenol, Clindamycin OK (liver)**

**Diabetes**

1. Types:
2. **Type I** – loss of insulin producing beta cells of the islets of Langerhans in pancreas leading to insulin deficiency; immune-mediated – T cell attack; Prevalence: 10%; Sudden onset; Childhood; Ketoacidosis common
3. **Type II** – insulin resistance +/- reduced insulin secretion (insulin receptor); unknown defect; Prevalence: 90%; Gradual onset; Adult; Ketoacidosis uncommon
4. Signs/Symptoms: (Cardinal) Polyuria, Polydipsia, Polyphagia, Wt loss
5. Complications: *Diabetic retinopathy, Diabetic nephropathy (microvascular) atherosclerosis/ischemic heart disease, peripheral vascular disease*
6. Testing: **HbA1c** (Glycosolated hemoglobin: 2-3 months prior glucose levels): **<6% Normal, <7% Well controlled diabetic**
7. Diagnostic criteria: **Fasting glucose: >126 mg/dL, 2 hr glucose: >200 mg/dL, HbA1C: >6.5%**
8. **↑ Risk of Infection:** Tx aggressively!! **I&D, Antibiotics (esp >200 mg/dL)**

**Diabetes**

1. Drug Interactions:
   1. **Sulfonylureas** (ie: Glypizide, Glyburide) and ASA/NSAIDs - ↑ Hypoglycemic effect of drug – **Avoid ASA/NSAIDs**
2. **Insulin Shock/Hypoglycemic Shock (Fast onset):**
   1. Mild: Hunger, Weakness, **Tachycardia, Pallor, Sweating**
   2. Moderate: **Incoherent, Uncooperative**, Beligerent, Poor orientation
   3. Severe: **Unconsciousness, Tonic/Clonic movements**, Hypotensive
   4. Tx: **Oral glycemic (*juice, cake icing*) or *IV glucose****, glucagon,* epi
3. **Hyperglyemic Crisis (Slow onset):**
   1. **Diabetic Ketoacidosis (DKA)** – Insulin insuffiency, *Excessive blood glucose*, and Dehydration. *Cells break down Fatty acids* into **ketone bodies/keto acids, blood pH drops**, and excessive excretion of fluids/glucose.
   2. Symptoms: **confusion**, **hunger**, dehydration, **“fruity”** smell breathe

**Diabetes**

***Bender 2003 JOE*** – Inherent factors of the disease:

1. Prone to **bacterial or opportunistic infections** (see below)
2. Vulnerability caused by a generalized circulatory disorder (**peripheral microvascular collapse)**
3. Blood vessels are damaged by the *accumulation of atheromatous deposits* (**macrovascular disease)**
4. Capillaries develop a *thickened basement membrane (***microvascular diesease)**
5. **Impaired leukocyte chemotaxis and immune cell delivery**
6. Decreased PMN microbicidal ability
7. **Due to limited pulpal/periapical circulation,** patients are **more prone to infection** and **impaired healing!!**

***Fouad et al 2003 JADA*** – Preoperative periradicular lesions and a history of diabetes have a significant reduction in successful outcome for endodontic treatment. **PARL + Diabetes = ↓Success of NSRCT (*impaired healing)***

***Segura 2005; Marota/Siqueira 2012*** – Type 2 Diabetics have an increased prevalence for AP *(****prone to infection****)*

**Adrenal Insufficiency**

1. Primary Adrenal Insufficiency: **Addison’s disease (Autoimmune)**
   1. Insufficent production of cortisol and/or aldosterone
   2. Symptoms: **Hypoglycemia,** dehydration**, wt loss, disorientation**, abdominal pains, vomiting, **fatigue**, depression, **hypotension**, kidney failure and shock (adrenal crisis)
   3. Treatment: Hydrocortisone, Prednisone
2. **Adrenal Crisis**: (initial symptoms similar to Insulin shock)
   1. Insufficient levels of cortisol to respond to stressful stiuation (SITS = **S**tress **I**nfection **T**rauma **S**urgery)
   2. Symptoms: **Sweating, hypotension**, weak pulse, lethargy, **confusion/pyschosis, convulsions**, **loss of consciousness**
   3. Untreated may lead rapidly to: ***Hypo*thermia, Severe *Hypo*tension, *Hypo*glycemia, and Circulatory Collapse**
   4. **Treatment: IV Hydrocortisone (SoluCortef), Fluids**
   5. Diagnosis: ACTH stimulation test, Cortisol level, Blood sugar

**Adrenal Insufficiency**

1. Secondary Adrenal Insufficiency:
   1. **Steroid-induced – most commonly**
   2. Pituitary Adenoma – results in↓ ACTH
   3. Rare to have Adrenal Crisis with 2° Adrenal Insufficiency
   4. Long term Steroids: Insomnia, Gastric Ulceration, Delayed wound healing
2. Supplemental Steroids & Surgery:
   1. **Only Addison’s disease requires supplemental steroid therapy**
   2. Minor Sx: **25 mg/day** **hydrocortisone** prior to Surgery
   3. Moderate Sx: **50-75 mg/day** **hydrocortisone** prior to Sx *and* **1 day post Surgery**
   4. Major Sx: **100 mg/day hydrocortisone** prior to Sx (*or GA procedure*) *and* **2-3 days post Surgery**
   5. 2° Adrenal Insufficiency receive normal daily dose of steroids **within 2 hours of Surgery**

**Hyperadrenalism**

1. **Cushing’s Syndrome (opposite of Addison’s)**
   1. Prolonged exposure to inappropriately high levels of cortisol
   2. Causes: **Exogenous glucocorticoids (ie: Prednisone),** Pituitary Adenoma (secondary hypercortisolism aka “Cushing’s Disease”, ↑ ACTH), or Adrenal tumor
   3. Symptoms:
      1. **Rapid wt gain – buffalo hump, moon facies**
      2. **Irritability, insomnia**
      3. Muscle/Bone weakness
      4. Memory/Attention dysfunction
      5. **Osteoporosis**
      6. **Diabetes Mellitus/Hyperglycemia**
      7. **Hypertension**
      8. Hirsutism – excessive hair growth (females), Acne
   4. Treatment: Taper off steroids, Removal of Tumor

**Thyroid Disease**

1. **Hyperthyroid**
2. Definition: Overproduction of thyroid hormones (T3, T4), aka thyrotoxicosis (hypermetabolic condition), leading to **overstimulation of metabolism** and **exacerbates the sympathetic nervous system** effects
3. Symptoms:
4. **Weight loss**
5. **Anxiety, Irritability**
6. **Heat intolerance**, Sweating
7. Weakness/Fatigue
8. **Exopthalmus**
9. **Tremors** – Epi/NorEpi Symp.
10. **Heart palpitations/Arrythmias** – Epi/NorEpi Symp.
11. Causes: **Grave’s Disease (autoimmune)**, Thyroid adenoma
12. Testing: Labs – *TSH (low), T4 (high);* Normal T4:T3 ratio = 20:1
13. Treatment: Anti-thyroidics, Surgical removal, Radioiodine tx

**Thyroid Disease**

1. **Thyroid Storm/Thyrotoxic Crisis**
2. Definition: Rare but life threatening form of hyperthyroid
3. **Precipitating Factors**: (similar to Adrenal Crisis) - **SITS**
4. **S**tress
5. **I**nfection
6. **T**rauma
7. **S**urgical Procedure
8. Clinical Manifestations: **Restlessness**, **Fever,** Pain, Delirium, **Psychosis**, **Tachycardia, Arrythmia**, Pulmonary edema, **Coma, Heart failure**
9. Treatment: **Call EMS**, **Cool patient** with cold towels, **Injection** of **100-300 mg hydrocortisone**

NOTE: **Avoid EPI and ASA/NSAIDs** in patients with **Uncontrolled Hyperthyroidism**

**Thyroid Disease**

1. **Hypothyroid**
2. Definition: Underproduction of thryoid hormones (T3, T4) – **Understimulation of metabolism** and **Underexaggeration of Sympathetic Nervous System effects**
3. Symptoms (opposite of Hyperthyroid):
   1. **Fatigue**
   2. **Cold Feeling**
   3. **Bradycardia**
   4. **Weight gain**
   5. Poor memory/concentration
4. Causes: *Hashimoto’s Thyroiditis (autoimmune)*, ↓iodine, Thyroidectomy
5. Testing: TSH (high), T4 (low)
6. Treatment: Levothryoxine (synthetic replacement) – **LIMIT EPI**

**Thyroid Disease**

1. **Myxedematous Coma**
2. Definition: Stressful event (SITS) in hypothyroidic patient (typically elderly) precipitates myxedma coma
3. **Precipitating Factors**: (same as Thyroid Storm + CNS depressants)
   1. Infection
   2. Trauma
   3. Surgical Procedure
   4. Stress
   5. CNS/Respiratory depressants – *Narcotics, Benzos, Flexaril*
4. Symptoms (Similar to Adrenal Crisis, Hypoglycemic Shock): **Hypothermia,** Bradycardia, **Hypotension**, **Seizures,** **Hypoxia**
5. Treatment: **Call EMS, Heat patient** with blankets, **Injection** of **100-300 mg hydrocortisone**, IV injections: T4, T3, levothyroxine

*NOTE: Avoid CNS/Respiratory depressants in Uncontrolled Hypothyroidism*

**Pregnancy**

Treatment Considerations:

1. Dental Tx: 2nd trimester or 1st half of 3rd trimester (weeks 14-32)
2. Analgesic: **Acetominophen (B)**, Ibuprofen (B, D3 – *constriction of ductus arteriosus, post-partum hemorrhage, delayed labor*)
3. Local Anesthesia: **Lidocaine (B)** or Etidocaine (B), Mepivicaine (C)
4. Antibiotics: **Amoxicillin, Penicillin, Clindamycin**, Metronidazole all class **B**
5. Narcotics/Anxiolytics: **AVOID** – Hydrocodone (C/D3), Triazolam (X)
6. Radiation exposure:
   1. *Danforth* – 9 in 1 billion chance of birth defect from FMX
7. Risk Classifications:
   1. Human studies – no risk
   2. Animal studies – no risk or Animal – risk/Human – no risk
   3. Animal studies – risk/Human studies – no studies or Animal/Human – no studies
   4. Human studies – risk, drug may be used despite risk (situational)

X. Human studies - Fetal harm outweighs possible benefit

**Pregnancy**

1. Nitrous Oxide (pregnant patient): **50% O2, <30 mins** admin.
2. Nitrous Oxide (pregnant employee):
   1. *Rowland* – Nitrous oxide exposure without scavenger (**3 hour/week or more**) increases *the risk of spontaneous abortion*
3. Maximum radiation exposure for pregnant worker = **5 mSv/year**

**Inferior Vena Cava Syndrome**

1. Definition:
   1. Prolonged supine position in late pregnancy places pressure on the inferior vena cava and drops blood return
2. Symptoms:
   1. *Intense pain* on the **R side**, *Muscle twitching*, ***drop of blood pressure***, and fluid retention
3. Treatment:
   1. **Turn patient on L side, monitor BP**

**HIV/AIDS**

HIV: Human immunodeficiency virus, AIDS: Acquired immunodeficiency syndrome

1. HIV: enveloped RNA retrovirus, infects most commonly **CD4+ cells** (**TH, macrophages**), entry-reverse transcription RNA-DNA-integration into host
2. Transmission: Exchange of infected bodily fluids (blood, seminal/vaginal fluids, tears, breast milk, CSF, urine
3. Stages:
   1. **Stage 1: Acute seroconversion syndrome**: **1-3 wks post-exp**, symptoms: fever, nausea/vomiting, lymphadenopathy, **CD4+ ≥500**.
   2. **Stage 2: Latent period**: **8-10 years**, asymptomatic, lymphadenopathy, steady decline in **CD4+ 200-499 cells/μL**
   3. **Stage 2: Early symptomatic stage**: **1-3 years**, Herpes Zoster, Oral hairy leukoplakia**,** Fungal infections. Signs/Symptoms increase as **CD4+** count declines/**approaches 200 cells/μL**, **Platelet count ↓**
   4. **Stage 3: AIDS**: opportunistic infections – Kaposis’s sarcoma, lymphoma, cancer. **High viral load. CD4+ count <200/μL**

**HIV/AIDS**

1. Laboratory Counts:
   1. Stage 1 (acute seroconversion): CD4+ T cell count: **≥500 cells/μL**
   2. Stage 2 (latent/early sym): CD4+ T cell count: **200-499 cells/μL**
   3. Stage 3 (AIDS): CD4+ T cell count: **<200 cells/μL**
2. Dental Treatment:
   1. Stage 1: No modifications, Monitor CD4+ and viral load
   2. Stage 2: No modifications, **Labs: CD4+, viral load, WBC/ CBC (est. immune status)**
   3. Stage 3: ***Antibiotic coverage*** for surgical/invasive treatment (**neutrophil count** <**500 cells/μL**)**, Emergency care only**
   4. **Avoid NSAIDs/ASA w/Thrombocytopenia**
3. Occupational Exposure:
   1. **Risk: 0.3%** (3 in 1000 sticks or sharps exposure)
   2. PEP: 4 weeks of 2 or 3-drug regimen ARTs
   3. Testing: *baseline, 3 months, 6 months, 12 months*
4. Effect of HIV on NSRCT:
   1. *Succhina; Shetty* – **No difference** in success of NSRCT

**Allergies & Anaphylaxis**

**Type 1: IgE Mediated – Anaphylactic:**

* **Immediate response – Mast cells, Bradykinin (Vasodilation, Inc vas. Permeability)**
* Antigens: Allergens – dust, pollen, *food, drugs*
* Symptoms: **Anaphylaxis - Asthma, Urticaria (hives), Angioedema (face, throat)**

**Type 2: Cytotoxic – IgG, IgM:**

* Antibody mediated – cytotoxic hypersensitivity
* **Ab bind to host cells recognized as foreign**
* Examples: Mismatch transfusions, Rh incompatibility

**Type 3: Immune complex mediated:**

* **Immune complexes (Ab-Ag)** lodge in vessel walls, incite inflammatory rxn, PMNs/macrophages, **Complement** activation
* Examples: SLE, Erythema multiforme

**Type 4: Cell-mediated – Delayed Type:**

* **T lymphocytes, No antibodies**
* Delayed response – **2 days after exposure**
* Examples: Contact dermatitis, Graft Rxn, Tb, Drug hypersensitivity

Treatment: Call EMS, Supine positiion, **0.3-0.5 mL 1:1000 EPI IM or SC**, **IV Diphenhydramine (Benedryl – Antihistamine) 50-100 mg,** Oxygen

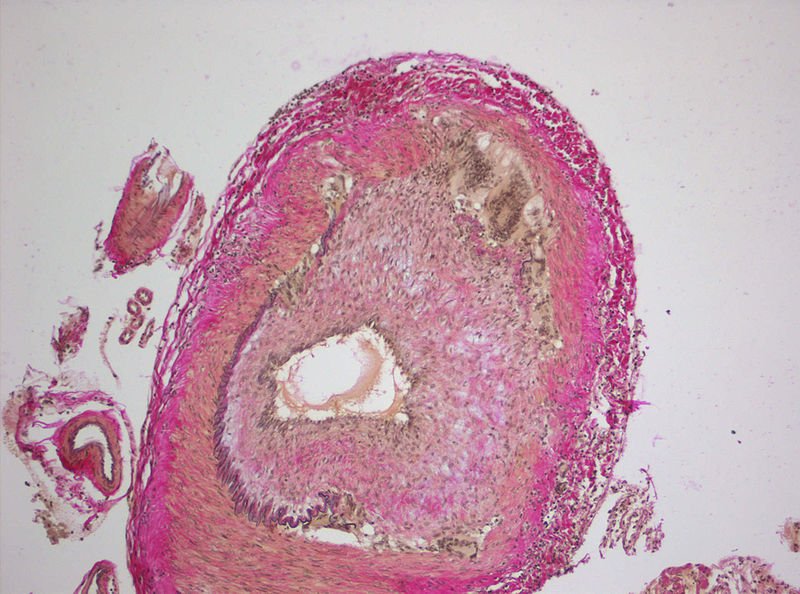
If angioedema/hives, w/ no throat or tongue swelling–50 mg Diphenhydramine oral 4x/day

**Rheumatological & Connective Tissue Disorders**

1. **Rheumatoid Arthritis**
   1. **Autoimmune disease** resulting in chronic systemic inflammation of the synovial joint spaces, destruction of the articular cartilage, and fusion of the joints, leading to severe loss of function/mobility
   2. Treatments: Methotrexate (anti-folate), Biologic agents (TNF-α blockers, IL-1 blockers), NSAIDs, Steroids
   3. Lab tests: **RF**, Imaging of hands/feet
   4. Possible *anemia, thrombocytopenia, secondary adrenal insufficiency*
2. **Giant Cell Arteritis**
   1. Inflammatory disease of the **arterial wall**, *T cells/macrophages* form **granulomatous lesion within vascular wall**
   2. Affects the **External Carotid** or **Superficial Temporal** **artery**
   3. Signs/Symptoms: **Jaw pain**, Fever, **Throbbing Headache** (unilateral/back of head), **Scalp Sensitivity, Blurred/loss of vision**
   4. Diagnosed by **Biopsy/Histopathology (gold standard)**
   5. Lab Tests: Sedimentation rate, C-reactive protein, ALP (liver)

**Giant Cell Arteritis**

**\*\*Commonly misdiagnosed as TMD!**



**Rheumatological & Connective Tissue Disorders**

1. **Systemic Lupus Erythematosus**
   1. Systemic autoimmune disease that attacks connective tissues throughout the body with a **Type III** Hypersensitivity rxn
   2. Involved organs: *heart, lungs, skin, joints, blood vessels, liver, kidneys, CNS*, may lead to athersclerosis and CAD
   3. Signs/Symptoms: Butterfly rash, Polyarthritis, Malaise, Fever
   4. Treatment: Immunosuppressants – cyclophosphamide, corticosteroids, DMARDs
   5. Lab tests: **ANA**, Immunofluorescence, **Liver/Kidney enzymes**, **CBC**
   6. Dental considerations:
      1. **Leukopenia**/corticosteroids – **post op antibiotics**
      2. **Secondary adrenal insufficiency – corticosteroids**
      3. **Thrombocytopenia** – platelet count; Anemia
      4. *Possible cardiac, liver, and kidney impairment*

**Prosthetic Joint Replacement**

1. Cover high-risk patients with prosthetic joints for invasive dental tx:
   1. **Immunocompromised/Immunosuppresed** *with inflammatory arthropathies:*
      1. Rheumatoid arthritis
      2. SLE
      3. MG
   2. **Disease, drug, or radiation** induced **immunosuppresion**
   3. Insulin dependent **Type I Diabetes**
   4. **Hemophilia**
   5. **< 2 years** post-joint replacement
   6. **Previous prosthetic joint infection**
2. Antibiotic regimen:
   1. No allergy to Penicillins: Amox, Cephalex 2 g 30-60 mins prior
   2. Allergy to Penicillins: Clindamycin 600 mg 30-60 mins prior
   3. IV meds only: Ampicillin 2 g IM/IV or Clinda 600 mg IM/IV

**Organ & Bone Marrow Transplantation**

1. Medical Consulation Pre-Surgery:
   1. Patient status
   2. Need for **Antibiotic prophylaxis**
   3. Need for **modification of drugs, anesthetics dosages**
   4. **Bleeding** precautions
   5. **CBC (platelets, WBC), INR/PT, aPTT, bleeding time**
2. Post-Transplantation Surgery:
   1. ***Defer elective tx for 6 months***
   2. Medical consultation:
      1. Antibiotic prophylaxis
      2. Modification of dosages of drugs, anesthetics
      3. Bleeding: INR, PT, aPTT, CBC (WBC, RBC, …)
      4. Supplemental Steroids
3. Heart transplant:
   1. Cover SBE for **heart transplant *with cardiac valvulopathy***

**Red Blood Cell Disorders**

1. **Sickle Cell Anemia**
   1. *Autosomal recessive* (singe nucleotide mutation of β-globin gene) inherited blood disorder resulting in abnormal sickling shape of RBCs (**HbS**) which have *decreased elasticity* and are *susceptible to breakdown* within capillaries
   2. Trait vs. Disease:
      1. **Trait (carrier - HbS)** = 1 allele (heterozygous), symptomatic *only* in oxygen deprivation/severe dehydration
      2. **Disease (SCA - HbSS)** = 2 alleles (homozygous), Anemic, intermittent vaso-occlusive crises
   3. Pathophysiology: low-oxygen tension, repeated sickling of RBCs, **decreased elasticity**, *inability to deform in capillaries*, leading to **vaso-occlusion** and **ischemia (pain, necrosis – tissues, pulps)**

**Red Blood Cell Disorders**

* 1. Lab Tests: **Sickledex test** (Initial test - detects HgS), **hemoglobin electrophoresis** (differentiates trait from disease); **CBC**, High reticulocyte count (↑ RBC production to compensate)

* 1. **Aplastic Crisis**: **(H.A.D.I.)**
     1. Signs/Symptoms: **5-7 days**, **severe anemia** → pallor, fatigue, tachycardia, reticulocytopenia
     2. **Provoked by: Hypoxia, Acidosis, Dehydration, Infection**
     3. Treatment: Blood transfusions
  2. **Vaso-occlusive Crisis**:
     1. Signs/Symptoms: Obstructed capillaries → **ischemia, pain, necrosis, organ damage**
     2. **Provoked by: Hypoxia, Acidosis, Dehydration, Infection**
     3. Treatment: NSAIDs, hospitalization

**Red Blood Cell Disorders**

* 1. Dental Considerations:
     1. **Medical consultation** for status
     2. **Drug modifications**:
        1. **Monitor O2** Saturation: Want **>95%**
        2. **Nitrous oxide**: Okay, **provide 50% O2**
        3. **Avoid Epi (non-surgical**) – vasoconstrict/hypoxia
        4. Limit Epi (2 carps) – Surgery
        5. **Avoid Narcotics, Barbiturates, Benzos, Flexaril** (Respiratory Depression) – Hypoxia = crisis
        6. **Avoid ASA/NSAIDs** – Acidosis = crisis
     3. **Aggressive management of infections** (prevent crisis):
        1. I&D
        2. Antibiotics
        3. Endo, Ext

NOTE: Prophylactic Abs for Sx – prone to infections, macrophages phagocytizing abnormal RBCs

**Red Blood Cell Disorders**

1. **Aplastic Anemia**
   1. Disease of bone marrow stem cells resulting in a **deficiency of RBCs, WBCs, and platelets aka Pancytopenia** (anemia, leukopenia, & thrombocytopenia)
   2. Diagnosis: Bone marrow biopsy
   3. Treatment**: Immunosupression (corticosteroids, cyclosporine),** Stem cell transplantation
   4. Dental Management: **Med Consult**, Severe pancytopenia – *Emergency care only*, **Aggressive management for oral infections** (antibiotics, supportive tx)

**White Blood Cell Disorders**

1. **Leukemia**
   1. Group of Tumors developing from malignant **immature abnormal leukocytes**
   2. Types: **AML, ALL, CML, CLL**; *Lymphocytic (B cell lineage)*, *Myeloid (undifferentiated lineage* – RBC, WBC, or platelet)
   3. Complications: **Crowding of immature leukocytes** impairs development of RBCs, WBCs, and Platelets. This leads to the following:
      1. Anemia, Pallor (**anemia**)
      2. Susceptible to infections (**neutropenia/leukopenia**)
      3. Bleeding/clotting dysfunction (**thrombocytopenia**)
      4. Fever/Flu-like symptoms
      5. Splenomegaly, Hepatomegaly
   4. Diagnosis: **CBC, Bone marrow biopsy**
   5. Treatment: Chemotherapy, Radiation, Bone marrow transplant

**White Blood Cell Disorders**

1. **Lymphoma**
   1. Group of tumors developing from malignant **Lymphocytes (B, T, NK cell)**, often times originating in lymph nodes, bone marrow, or spleen
   2. Major Types: **Hodgkin’s, Non-Hodgkins**, **Multiple Myeloma**
   3. Symptoms: **Lymphadenopathy, Night Sweats**, Fever, Fatigue, Weight Loss
   4. Diagnosis: **Lymph node biopsy**
   5. Histopathology: **Reed Sternberg cells** – “owls eyes” (**HL only**)
   6. Prognosis: HL (85%) > NHL (69%)
   7. Treatment: Chemotherapy, Radiation, Surgery

**White Blood Cell Disorders**

1. **Multiple Myeloma**
   1. Tumors developing from malignant **Plasma cells** (precursors to Abs), accumlate in the bone marrow and *interfere with normal production of RBCs, WBCs, and platelets*
   2. Signs/Symptoms: “**CRAB”** = **Calcium (elevated), Renal Failure, Anemia (↓RBCs), and Bone pain**; Infection (↓WBCs), Bleeding (↓ Platelets)
   3. Diagnosis: Serum electrophoresis, **Bone marrow biopsy**, **Bence-Jones proteins (Urinalysis)**
   4. Histopathology: Plasmacytoma
   5. Radiographic: **“Punched Out” resorptive bony lesions** (**similar to 2° HPT/ESRD**) throughout skeletal system (overexpression of RANKL in bone marrow)
   6. Treatment: Protease Inhibitors, **Bisphosponates**, Chemotherapy, Bone marrow transplant

**White Blood Cell Disorders**

Dental Management of WBC Disorders:

* **MED CONSULT**: Immune status, Bleeding/Clotting Issues
* **Antibiotics**: **Pen VK** 2g 1 hr prior + 500 mg 4x/day, 7 days
  + WBC < 2000 cells/μL
  + Neutrophil count < 500 cells/μL
  + 6 months post splenectomy (lymphoma patients)
* Platelet transfusion:
  + Platelets <50,000 cells/μL
* **Emergency care ONLY** for advanced WBC disorders
* Routine care for stable disease progression or state of remission

**Bleeding and Hypercoagulable Disorders**

AVOID ASA/NSAIDs in all bleeding/clotting disorders!

1. **Thrombocytopenia**
2. Definition: Abnormal decrease in platelet count **<50,000 cells/μL**
3. Clinical Signs/Symptoms:

* **Petechia, Purpura, Ecchymosis**
* **Bleeding –** nose bleeds, gingival bleeding
* Malaise, **Fatigue,** Weakness

1. Causes:

* ↓ Production: Vitamin B12 def. **leukemias, AA, liver disease**
* ↑ Destruction: **SLE, HIV,** Thrombotic Purpura
* Medication Induced: *Valproic Acid*, *Methotrexate*, *H2/PP inhibitors*

1. Lab Testing: Platelet count
2. Dental Considerations:

* Counts: Surgery **>50,000**, NSRCT >30,000
* **Avoid NSAIDs/Aspirin** - ↑ Bleeding due to ↓ Thromboxane A2
* Local hemostatic measures: *Gelfoam/Collagen, Thrombin***,** Amicar

**Bleeding and Hypercoagulable Disorders**

1. **Coumadin and Anti-coagulants**
2. Drugs: Coumadin/Warfarin, Heparin, Pradaxa
3. MOA:

* **Coumadin**: Inhibits **liver** synthesis of **Vit K-dependent clotting factors**: *Factors 2 (Prothrombin), 7, 9, 10*
* **Heparain, Pradaxa**: Anti-Thrombin (II), Direct thrombin inhibitor

1. Lab Values: INR (Coumadin only), PT time (Ext/Common)
2. Dental Considerations:

* **INR (within 48 hours): < 3.0 (surgery),** < 3.5 (NSRCT)
* **Med consult: bleeding control, INR, comorbid conditions**
* Delay treatment **2-3 days** for ↓ anti-coagulant
* **Avoid: Aspirin/NSAIDs, Metronidazole**, **Macrolides, Tetracyclines (CMT = C**oumadinavoid **M**etro**/M**acro**, T**etra**)**
* **Local hemostatic measures:** Gauze, gelfoam, Collagen (plug/tape), Thrombin, Amicar, Tranexamic acid

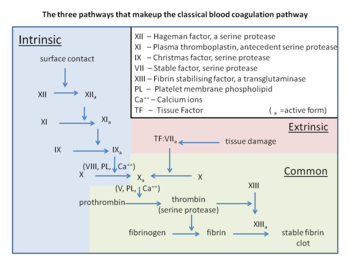
**Bleeding and Hypercoagulable Disorders**

1. **Hemophilia**
2. **Hemophilia A (Factor VIII)**

* **X linked recessive** trait, Males predominately
* Dysfunctional or ↓ Production of Factor VIII
* Signs/Symptoms: **Spontaneous or prolonged bleeding** episodes, **bruising**/ecchymosis, **internal bleeding**
* Tests: **Prolonged aPTT**, Normal PT/TT/Platelets
* Treatment: **IV infusion** **Factor VIII**, **Desmopressin** **(↑ VWF/VIII**)

1. **Hemophilia B (Factor IX/Christmas)**

* **X linked recessive** trait, Males predominately
* Deficiency of Factor IX gene due to **mutation**
* Signs/Symptoms: Same as Factor VIII defiicency
* Tests: Same as Factor VIII, Prolonged aPTT/Normal PT/TT/Platelets
* Treatment: **IV Infusion** **Factor IX only, NO Desmopressin!**



**Bleeding and Hypercoagulable Disorders**

1. **Von Willebrand’s Disease (vWD)**
2. Definition: Most Common inherited coagulaopathy (1 in 100), qualitative or quantitative deficiency in the vWF. Most Asymptomatic.
3. vWF: Large glycoprotein that **binds Factor VIII and Platelets**, promotes **platelet adhesion** to vascular endothelium at the site of injury
4. Types: 3 Forms: Hereditary, Acquired, Psuedo. **3 Types of Hereditary VWD**: *Type I (most common, 60-80%), II, and III* with mulitple subtypes (II A, B, N, M)
5. Clinical Signs/Symptoms: **Prolonged bleeding, Bruising**
6. Lab Tests: **vWF antigen assay** - Type I (vWF 10-45 IUs), **Factor VIII assay**, **aPTT time** (elevated due to VIII def.), CBC, PT
7. Treatment: **Desmopressin** (DDAVP = Syn. analog of **ADH** Vasopressin) – **Type I, IIA only** - **↑ vWF/VIII** (Intranasal/IV), **Infusion Factor VIII**, Blood transfusions (correct anemia and hypotension)

**Dental Considerations with Hemophilia and vWD**

* **Medical Consultation!**
* Lab Values: **aPTT, Factor VIII, IX, vWF assays, CBC, bleeding time**
* **Avoid Aspirin/NSAIDs!** - ↑ Bleeding due to ↓ Cox/Thromboxane A2
* Pre-op Clotting Aids:
  + **Desmopressin – Factor VIII, vWF only – releases vWF/VIII from vascular endothelium**
  + Amicar, Transexamic acid – clotting stimulators
  + **Factor VIII, IX, and vWF IV infusions**
* **Avoid infiltrations/block injections** for patients not on desmopression and/or IV Factor concentrates – **internal bleeding risk**
* **Local hemostatic measures –** Collagen, gauze, Thrombin**,** Amicar
* **vWF Type I and some Type II** may be managed **in-office** with **Desmopressin.** May requireInfusions **- Factor VIII/vWF**

|  |  |  |  |  |  |  |  |  |  |  |  |  |  |  |  |  |  |  |  |  |  |  |  |  |  |  |  |  |  |  |  |  |  |  |  |  |  |  |  |  |  |  |  |  |  |
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| **Laboratory findings in various platelet and coagulation disorders (**[**V**](http://en.wikipedia.org/wiki/Template:Bleeding_worksheet) **-** [**T**](http://en.wikipedia.org/wiki/Template_talk:Bleeding_worksheet)**)**   |  |  |  |  |  | | --- | --- | --- | --- | --- | | **Condition** | [**Prothrombin time**](http://en.wikipedia.org/wiki/Prothrombin_time)  **(PT)** | **Activated** [**Partial thromboplastin time**](http://en.wikipedia.org/wiki/Partial_thromboplastin_time)  **(aPTT)** | [**Bleeding time**](http://en.wikipedia.org/wiki/Bleeding_time) | [**Platelet count**](http://en.wikipedia.org/wiki/Platelet_count) | | [Vitamin K deficiency](http://en.wikipedia.org/wiki/Vitamin_K_deficiency) or [warfarin](http://en.wikipedia.org/wiki/Warfarin) | **Prolonged** | **Normal or mildly prolonged** | Unaffected | Unaffected | | [Disseminated intravascular coagulation](http://en.wikipedia.org/wiki/Disseminated_intravascular_coagulation) | **Prolonged** | **Prolonged** | **Prolonged** | **Decreased** | | Von Willebrand’s disease | Unaffected | **Prolonged or unaffected** | **Prolonged** | Unaffected | | [Hemophilia](http://en.wikipedia.org/wiki/Hemophilia) | Unaffected | **Prolonged** | **Prolonged** | Unaffected | | [Aspirin](http://en.wikipedia.org/wiki/Aspirin) | Unaffected | Unaffected | **Prolonged** | Unaffected | | [Thrombocytopenia](http://en.wikipedia.org/wiki/Thrombocytopenia) | Unaffected | Unaffected | **Prolonged** | **Decreased** | | [Liver failure](http://en.wikipedia.org/wiki/Liver_failure), early | **Prolonged** | Unaffected | Unaffected | Unaffected | | Liver failure, end-stage | **Prolonged** | **Prolonged** | **Prolonged** | **Decreased** | |

**Normal Lab Values**

Platelets:

* Normal: 150,000 – 450,000 cells/μL
* Surgery: > 50,000 cells/μL

PT (Prothrombin time):

* Tests **Extrinsic (VII)** and common pathway
* Normal: 11-15 s

aPTT (Activated partial thromboplastin time):

* Tests **Intrinsic (VIII, IX, XI, XII)** and common pathway
* Normal: 25-35 s

TT (Thrombin time):

* Tests ability to form **initial clot from fibrinogen** (end of common pathway)
* Normal: 9-13 s

INR Value:

* **Standardized Ratio** of patient’s PT to control PT, measures *Extrinsic (VII) and common pathways*, **measured index** for Coumadin therapy/risk of bleeding
* Normal: 0.8-1.2 (< 1.0), Coumadin: 2.0-3.0 (< 3.0)

**Normal Lab Values**

CBC:

* RBCs
* WBCs
* Hemoglobin
* Hematocrit
* Platelets

Sed rate: 1 – 40 mm/hour

WBC: 4,000-11,000 cells/μL

Neutrophil count: > 500 cells/μL

**Cancer & Oral Care**

**Chemotherapy & Dental Considerations**: *Myelosuppresion*

* Thrombocytopenia – Prolonged bleeding - Local measures, Platelets
  + Infusions – Platelets <50,000 cells/μL
* Leukopenia – Risk of Infection – Antibiotics (prophylactic + 7 d. course)
  + WBCs < 1000 cells/μL
  + Neutrophils < 500 cells/μL
  + Culturing/Ab sensitivity testing of exudate
* Anemia – Weakness, Hypotension – Blood transfusions
* **Emergency Tx ONLY during Chemotherapy!**

**H&N Radiation & Osteonecrosis**:

* **Avoid Sx** in patients who received **> 6000 cGy**
* **Avoid EPI, Avoid Lidocaine**
* Surgery - Pen VK – **Prophylaxis and 7 day regimen**
* **Hyperbaric Oxygen** prior to Sx/Invasive Tx – improve wound healing

**Cancer & Oral Care**

Common chemotherapeutic agents:

* Methotrexate:
  + MOA – *Anti-metabolite, Anti-folate*
  + Uses - cancer chemotherapy (high doses), autoimmune diseases – Rheumatoid Arthritis, Lupus, Crohn’s disease (low doses)
  + Side effects – Ulcerative stomatitis (oral ulcerations), **↓ WBC/↑ infection risk**, abdominal pain, nausea, acute pneumonitis
  + **Drug interactions: (PNPV)**
    - **Penicillins** - ↓ elimination, ↑ toxicity
    - **Nitrous Oxide** – hemo toxicity
    - Proton pump inhibitors (ie: Omeprazole) - ↑ toxicity
    - Valproic acid - ↑ toxicity
* Cyclophosphamide – alkylating agent, DNA replication, severe side effects
* 5-fluoruracil – Anti-metabolite, severe myelosuppression, CNS damage
* Doxorubicin – anthracycline antbiotic, intercalating DNA, cardiomyopathy, CHF

**Neurologic Diseases**

**Epilepsy/Seizures**

1. Definition: Abnormal brain activity resulting in depolarizing shift
2. Types: Convulsive (60% - focal/generalized), Non-convulsive (40%); Generalized (loss of conciousness): Tonic-clonic(rigidity/convulsions), clonic, myoclonic, absence, atonic
3. Aura: sensory(visual/hearing/smell), motor
4. Causes: brain trauma, stroke, tumors, idiopathic (60%)
5. Medications: *Phenytoin(Dilantin), Carbamezpine (Tegretol), Valproic Acid*

* **↑ GABA** = sedative, **CNS depression**
* **Avoid: Benzodiazepines, Barbituates, & Alcohol due to effect on CNS/GABA**

1. Dental Considerations:

* **Medical Consultation**: Type of Seizure, Last activity, PFs, Medications, Control
  + Well controlled: Routine Care
  + Poorly controlled: Med Consult, possible limitations
* **Avoid Aspirin/NSAIDs** (valpr)**, Benzodiazepines/Alcohol/Opioids** (CNS dep)
* **Limit Epi** to **2 carps**
* Use ligated mouth prop

**Neurologic Diseases**

**Epilepsy/Seizures**

* During Seizure:
  + Clear area, Protect patient, *Suppine Position, Turn patient to Side* (to avoid aspiration)
  + **No padded tongue blade**
  + *Passively Restrain*
* Testing: EEG, CT/MRI Brain scan

**Medications**: Anticonvulants- carbamazepine, valproic acid, pre-gabalin (lyrica), gabapentin (neurontin) – other uses include *neuropathic pain, neuralgia, migraines*

Valproic Acid, Carbamazepine (Tegretol) adverse effects:

* Nausea, blurred vision
* Xerostomia
* *Platelet dysfunction – bleeding (avoid ASA/NSAIDs)*
* Stevens-Johnson syndrome
* Drug interactions: macrolides
* **Potential CNS depression – avoid other CNS depressants**

**GABA & CNS Depression**

**GABA**: Major side-effect: *CNS/Respiratory Depression – coma/death*

* **Major inhibitory neurotransmitter** in the CNS – ***reducing neuronal excitability of CNS*** *(pre- and post-synaptic junctions)*
* Target of **Benzodiazepines, Barbituates, Anti-convulsants**
* Anxiolytic, Anti-convulsant, Sedative, Amnesia, Euphoria

**GABA Agonists**: Benzodiazepines, Barbituates, Alcohol, Anticonvulsants (Tegretol/Carbamazepine)

**GABA Analogues**: bind to Ca/Na channels, act similarly to GABA

* **Pre-gabalin (*Lyrica*)** – Neuropathic pain, generalized anxiety disorders, epilepsy
* **Gabapentin (*Neurontin)*** – Neuropathic pain, Diabetic neuropathy, Anti-convulsant (blocking Na channels)

**Flumazenil** – GABA receptor antagonist, Tx: Benzodiazepine overdose

NOTE:

Benzodiazepines’ CNS depressant effect is potentiated by: ***Barbituates, Opioids, Alcohol, Anticonvulsants* (other CNS depressants)**, *Cimetidine, Macrolides*

\*\***Avoid benzodiazepines** in **Narrow angle glaucoma**!

**Neurologic Diseases**

**Stroke**

1. Definition: Loss of brain fx due to disturbance of blood flow
2. Types: Hemorrhagic (leakage) or Ischemic (emboli)
3. Etiologies: Thrombus/Emboli (Ischemic), HTN/various factors (Hemorrhagic)
4. Symptoms: **FAST** – **F**ace droop, **A**rm weakness, **S**peech impaired, **T**ime
5. Dental Considerations: EMS, Immediate hospitalization

* **Past Hx of Stroke**:
  + *No elective care* for **currrent TIAs**
  + Delay elective care for **6 months**
  + **INR: <3.0** for invasive/sx
  + **Monitor Vitals/O2 Saturation**
  + Use N2O2
  + **Limit EPI:** **2 carps** (>6 months post stroke)
  + **Avoid NSAIDs**
  + IV heparin – no Sx until another antiocgulant is started

**Psychiatric Disorders – Anti-depressant Medications**

**TCAs:** ↑ Serotonin/NE, **Side Effects**: **Avoid respiratory despressants** (Benzos, Narcotics) – **potentiates respiratory depression**, Anti-cholinergic effects (dry mouth/nose, blurry vision)

* **Amitryptiline** (***Elavil*)** – Major depression, Migraines, Neuropathic pain

**MAOIs**: Prevents breakdown of mononamine neurotransmitters; **Drug interactions**!

**\*\*\*Drug Interactions of TCAs/MAOIs**:

* TCAs/MAOIs + **Epinephrine**: Limit to **2 carpules**
* TCAs/MAOIs + **Flexaril (cyclobenzaprine)** - **↑ Respiratory depression**
* TCAs/MAOIs + **Respiratory depressants** (Benzos, Barbs, Opioids, Alcohol, Anti-convulsants) – **↑ Respiratory depression**

**SSRIs**: ↑ Serotonin by ↓ reuptake synaptic cleft; Interact w/anti-coagulants

* Celexa, Lexapro, Prozac, Paxil, Zoloft (*Most Widely Used category*)

**SNRIs**: ↑ Serotonin/NE

* Effexor, Cymbalta

**Wellbutrin**: Atypical antidepressant, added to 1st line drugs

**Psychiatric Disorders – Bipolar (affective) Disorder**

1. Definition: Mental disorder characterized by episodes of Manic and Depressive characteristics
2. Symptoms:
   1. **Manic Episodes**: period of elevated euphoria, irritability, rapid speech, lack of sleep, and poor decision making
   2. **Depressive Episodes**: period of sadness, anxiety, guilt, anger, isolation
3. Treatment:
   1. Medications: **Lithium** (mood-stabilizer), Anti-convulsants (Valproic Acid, Carbamazepine)
4. Dental considerations:
   1. **Lithium interactions: ASA/NSAIDs,** Diuretics, ACE Inhibitors**, Narcotics**
   2. **Avoid ASA/NSAIDs, Narcotics**
   3. **Med consulation** – drug interactions, side effects

**Opioids**

1. Effects: **Analgesic, *Sedation, Respiratory depression*, Euphoria**
2. MOA: Bind to μ,κ,γ opioid receptors – CNS/Peripherally

* Endogenous opioids: *enkephalins, beta endorphins, dynorphins*

1. Semi-synthetic alkaloids: hydrocodone, oxycodone, hydromorphone
2. Side effects: Nausea, vomiting, drowsiness, **respiratory depression**, hallucinations, deliruim, brady/tachycardia
3. Interactions with **Benzodiazepines, Barbituates, Anti-convulsants, and Alcohol – CNS/Respiratory Depression**
4. ***Nalaxone (Narcotics)*** - μ opioid receptor antagonist, Tx: Opioid overdose – IV, IM, Subcutaneous

**CNS depressants (Breathing, H.R.):** Benzodiazepines, Barbituates, Anti-convulsants (GABA agonists), Alcohol, Opioids, Muscle Relaxants

**Muscle Relaxants**

**Cyclobenzaprine (*Flexaril*)** – muscle relaxant

* Side effects: CNS inhibition (drowsiness/sedation), Anti-cholinergic effects (xerostomia, fatigue, blurred vision), Tachycardia
* **Antagonistic effect:** *histamine***,** *serotonin, and muscarinic receptors*
* **Drug interactions**:
  + **CNS depressants**: Benzodiazepines, barbiturates, anti-convulsants (GABA), alcohol, opioids (**BBAAOs**) – Potential CNS/Respiratory depression
  + **Anti-depressants**: **TCAs, MAOIs** – Potential Serotonin Syndrome or Respiratory depression
  + **Psychotropic drugs/Anti-depressants**: SSRIs, SNRIs – Potential Serotonin Syndrome

**Bisphosphonate-associated Osteonecrosis of the Jaw (BONJ)**

AAE Statement: Retrospective studies, case reports/expert opinions only

-MOA: **Inhibits Osteoclastic activity, Induces Osteoclast Apoptosis, Inhibits Osteoclastic differentiation;** *may inhibit angiogenesis*

-Indications:

1. Resorptive Bone Diseases: Osteoporosis, Paget’s Disease, Fibrous Dysplasia
2. Hypercalcemia associated with certain diseases: Multiple Myeloma, Primary Hyperparathyroidism, Bone metastasis (prostate, breast)

-Drugs: BRADZPD – **B**oniva, **R**eclast, **A**redia/**A**ctonel, **D**idronel, **Z**ometa; non-bisphosphonates: **P**rolia, **D**enosumab (monoclonal antibody)

-Signs/Symptoms: **Mucosal ulceration + exposed bone >8 wks**, Pain/swelling, Infection, Altered Sensation (Numbness/Paresthesia)

-**Mandible>Maxilla**, CTX Test unreliable

-Treatment: Local debridement, Resection, Antibiotics, Hyperbaric Oxygen

-Risk Factors: **IV** bisphophonates (>1 year or Oral >5 years) - Bioavailibility, **Traumatic Dental injury**, **Nitrogen containing** bisphosphonates

*\*\*Avoid Exts, Endo Sx, Implants in IV bisphosphonate users*

**Infective Endocarditis/SBE - PPCC**

*-Streptococi* most common causitive organism, others: staph, enterococci, candida

-Most common complication/cause of death is heart failure (valvular dysfunction)

-Current AHA Guidleines for SBE coverage:

* **Prosthetic cardiac valve**
* **Previous IE**
* **Cardiac transplant patient who develop cardiac valvulopathy**
* **Congenital Heart Disease:**
  + **Unrepaired CHD**
  + **Completely repaired within the 1st 6 months**
  + **Repaired with residual defects at the site or adjacent site**

-Dental procedures requiring SBE coverage:

* Any manipulation of gingival tissues or periapical region
* Any perforation of oral mucosa, *excluding: routine anesthetic injections, radiographs, placement of oral appliences*

-Antibiotics (30-60 mins prior, up to 2 hrs post): Amox 2 g (50 mg/kg), Keflex 2 g (50 mg/kg), Clindamycin 600 mg (20 mg/kg), Azithromycin 500 mg (15 mg/kg)

**Antibiotic Classes**

**Bactericidal**: **PARQ** **M**y **V**ehi**C**le

***P****enicillins* – Beta Lactam Ring – *Inhibits the cross-linking of cell wall peptidoglycan* (not active against beta-lactamase secreting bacteria), Gram +/-

* Amoxicillin: moderate spectrum, better absorption, longer ½ life, Gram +/-

**A**minoglycosides – 30s Ribosomal

**R**ifampin - RNA

**Q**uinolones – Ciprofloxacin – DNA Gyrase

***M****etronidazole* – *Inhibits DNA nucleic acid synthesis* – Obligate Anaerobes

**V**ancomycin

***C****ephalexin* – Beta Lactam *– Inhibits cross-linking cell wall peptidoglycan*

**Bacteriostatic (Protein Synthesis Inhibitors**): **C**ountry **M**usic **T**elevision

***C****lindamycin (Lincosamide)* – *50s Ribosomal*, Gram + Anaerobes, C. dificile associated Pseudomembranous Colitis (nausea, diarrhea, vomiting)

**M**acrolides (i.e.: Erythromycin, Azithromycin) – 50s Ribosomal

**T**etracyclines (i.e.: Minocycline, Doxycycline) – 30s Ribosomal

**Medical Condition and related Emergencies**

1. **End Stage Renal Disease: 2° HPT – “Stones, Bones, Groans”**

* Lytic lesions (CGCG/Brown’s Tumor HPT), loss lamina dura, Ground glass appearance

1. **Sickle Cell Anemia: Aplastic/Vaso-occlusive crises**

* H.A.D.I. 🡪 Pain, Ischemia, Tissue necrosis

1. **Diabetes (Hypoglycemia): Insulin Shock (similar to Vasovagal Syncope)**

* Sweating/Pallor/Tachycardia🡪Uncooperative/Beligerent🡪Loss of consciousness/Convulsions/Hypotension

1. **Diabetes (Hyperglycemia): Diabetic Ketoacidosis**

* Hunger, Fruity smelling breath (blood glucose levels)

1. **Addison’s Disease: Adrenal Crisis AVOID SITS**

* Pyschosis, Loss of conscious, Convulsions, Bradycardia, Hypoglycemia, Hypotension, Hypothermia, Circulatory Collapse

1. **Epinephrine overdose: SEE EPI Contraindications Charts**

* Palpitations, Tachycardia 🡪 Arrythmias, MI, Stroke

**Medical Condition and related Emergencies**

1. **Hyperthyroid: Thyroid Storm AVOID SITS**

* Fever, Pyschosis, Tachycardia, Arrythmias, Hyperthermia

1. **Hypothyroid: Myxedematous Coma AVOID SITS/Resp. depress.**

* Bradycardia, Hypothermia, Hypotension, HYPOXIA

1. **Theophylline Toxicity (COPD)**: **AVOID Cipro, Macrolides (CMT)**

* Nausea, Vomiting, Arrythmias, Seizures

1. **Digoxin Toxicity:** **AVOID EPI**

* Hypersalivation, Nausea/Vomiting, Drowsiness, Visual disturbance - YELLOW or GREEN appearance

1. **L.A. overdose: CAUTION Chronic Hepatitis, Chronic Kidney Disease**

* Initial: Tremors/convulsions, Later: Loss of consiousness, Respiratory depression, CNS depression, Coma, Death

1. **Met-hemoglobinemia: AVOID Prilocaine, Benzocaine (spray)**

* Met-hemoglobin – metabolite of prilocaine and benzocaine, has Fe+3 instead of Fe+2, Selective affinity for bound O2, decreased O2 to cells = Cyanosis w/o respiratory distress 🡪 Respiratory dep/coma/death

**Antibiotic coverage for prophylaxis or management of orofacial infections:**

* Uncontrolled Diabetes – Microvascular disease (infection/delayed healing)
* End Stage Renal Disease – Leukopenia, consult physician
* Autoimmune disorders (immunosuppressant therapies):
  + Crohn’s disease/Ulcerative Colitis
  + Rheumatoid Arthritis
  + SLE
  + MS
* HIV: Stage 3: AIDS – *CD4+ <200 cells/μL (post tx/sx)*
* Organ/Bone Marrow Transplantation – Leukopenia, immunosup., consult physician
* Sickle Cell Anemia – Aggressive management of infections (vasoclusion)
* Aplastic Anemia – Med consult, Pancytopenia
* WBC tumors: Leukemia, Lymphomas, MM – consult physician
  + *WBC < 2000 cells/μL*
  + *Neutrophil count < 500 cells/μL*
  + 6 months post splenectomy (lymphoma patients)
* Chemotherapy – Bone marrow suppression, *WBCs < 1000 cells/μL, PMNs <500*
* H&N Radiation – Prevention of Osteonecrosis (Sx only, <6000 cGy)

**Rhinosinusitis (*Kretzschmar)***

Definition:

* Inflammation and infection of the mucous membranes of the nasal/paranasal sinuses (maxillary, frontal, ethmoid, sphenoid), Tissue edema, Non-patent ostia

Types:

* Acute <4 wks, Subacute 4-12 wks, Recurrent Acute 4+/1 yr, Chronic >12 wks

Etiology:

* Viral Infection, may progress to Bacterial infection, typically after 7-10 days

Symptoms:

* Major: *Facial pain, Facial pressure, Congestion*, Nasal obstruction, *Fever*
* Minor: Headache, Dental pain, Cough, Ear pain

Dental Findings:

* *Pain on palpation of Infraorbital region, Pain on percussion multiple Max. posterior teeth, Diffuse lingering pain in Max posterior, Cloudy sinus*

Treatment:

* Decongestants (*Systemic: Pseudophedrine 30 mg* q6h, *Nasal:* Oxymetazoline 0.05%, *Phenylephrine 0.125-1.0%,* *Neosynephrine 0.5%),* Amoxicillin 500 mg q6h 10 d or Clindamycin 300 mg q6h, Augmentin 500 mg q12h 14 days (if no response to Amox)