Radiological Exam

A. Vital case w/ AP – Stashenko / Yamasaki
B. Bone loss & AP – S/B 7.1 MBL / Root position to bone
C. Digital Comparison studies
    a. Film to digital – root length – NSD – Pitt Ford
    b. Film to digital – PARL – NSD – Newton
    c. D, E, F – film for measurement – NSD – Eleazer
    d. Film to digital – NSD – Lamus
    e. 80 – 90% radiation reduction – Soh
D. Lamina dura – consistent feature aiding dx – Kaffe
E. Interpretation accuracy –
    a. Brynholf – accuracy increases with added films, 3=87%
    b. Olsen – one film accuracy = 86%
    c. Goldman/Darzenta – 46% agreement on interpretation
F. Technique – best view, paralleling – Forsberg

Subjective & Objective Examination

A. Reeves – Irreversible pulpitis develops when caries is w/in 0.5mm of pulp
B. Tronstat – pulp cap is only 50% successful
C. Cvek – Vital pulp therapy 95% successful
D. Kretzchmar – referred pain may come from sinusitis
E. Cold test –
    a. Peters – no damage to tooth from CO2 snow or endo ice
    b. Jones & Miller 2004 – use large cotton pellet, CO2 snow = endo ice
    c. Peterson – cold test 90% accurate
    d. Trowbridge – mode of action – hydrodynamics
F. Heat test –Schindler – used on refractory cases to identify missed canals or late stage of an irreversible pulpitis
G. Barodontalgia – sensitivity or pain caused by change in pressure – Cunningham
H. Seltzer/Bender – no histologic correlation w/diagnostic tests
I. Friend – patient accuracy of etiology only 37%

Pulp Tests – vitality tests give information about pulpal nerve status only

Does not evaluate blood supply

Reliability –

a. Andreasen – immature developing teeth, unreliable response to EPT, use CO_2 snow
b. Bhaskar – trauma cases, EPT, cold and heat tests – unreliable (due to nerve damage w/out altering blood supply)
c. Fuss – unreliable EPT response in presence of large restoration, endo ice, CO_2 and EPT equally reliable in adult teeth.

EPT –

a. Mode of action – stimulate Aδ fibers
b. Technique – Wahab – slowly increased current is more accurate
c. Bender – test incisal edge in anterior teeth
d. Jacobson – test middle-third of incisors, occlusal-third premolars

Laser Doppler

A) Sundquist 99 Dx: pcd
B) Trope ’97- #8,9 dx’d w/ dopler revealed vital
C) Trondstad ’94: LD 91% accurate, more accurate than EPT (64%)

ASA Classification

I. A normal healthy patient
II. A patient with mild to moderate systemic disease
III. A patient with severe systemic disease that limits activity but is not incapacitating
IV. A patient with severe systemic disease and is a common threat to life
V. A moribund patient not expected to survive 24 hours with or without an operation.

Mobility – Modified Miller Classification

Class I - barely perceptible
Class II - < 1 mm movement
Class III - > 1 mm movement

Does periodontal disease cause pulpal disease?

Langeland – yes when all main apical foramina are involve by bact. Plaque
Seltzer/Bender – yes, bacteria can pass through lateral/accessory canals

Massler – found no relationship between pulpal & periodontal disease

Glickman system to classify 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
Class IV – Furca opening visible

Simon Classified Endo-Perio Lesions

Primary endo, primary perio, primary endo w/ secondary perio, primary perio w/ secondary endo, or true combined.

Biologic width

Gargiulo –
1. sulcus depth ~ 1mm
2. epithelial attachment ~ 1mm
3. connective tissue attachment ~ 1mm

Calcific metamorphosis

Trowbridge/Kim – caused by luxation traum, obliteration of pulp by mineralized tissue. Occurs in immature teeth, pulpal infarct, connective tissue from PDL proliferates and replaces pulp.

Gutmann – trauma causes 1-16% to develop pulpal necrosis, therefore do not automatically treat cases of calcific metamorphosis unless AP or nonvital.
Andreasen – 22% of traumatized teeth undergo calcific metamorphosis
Walton – no visible canal but always present histologically

Pulp canal obliteration

Trope – caused by luxation injury, uncontrolled reparative dentin or hemorrhage and clot formation act as a nidus for calcification, occurs in immature teeth.

Dystrophic Calcification

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

Cracked tooth syndrome

Cameron – coined term, most commonly found in the mandibular second molar

Rivera – Classified longitudinal tooth fracture

1. craze lines
2. cuspal fracture
3. cracked tooth
4. split tooth
5. vertical root fracture

Guthrie – treat with cast crown, banding and operative procedures do not protect against interocclusal forces.

Pashley 2004 JOE – Best method to identify cracks is transillumination and methylene blue dye.

Vertical Root Fracture

Pitts – identification requires direct visualization, transillumination, is a endo-perio lesion. Consider root resection, amputation and extrusion.

Testori – in endodontically treated teeth, occurs most often in premolars, usually observe narrow periodontal defect.

Cause of Apical Periodontitis

Microorganisms colonizing the root canal system play an essential role in the pathogenesis of periradicular lesion.

Kakehashi – germ free rat study directly linked AP to bacteria

Moller – monkey study repeated findings of Kakehashi

Sundqvist – human study further confirmed findings of Kakehashi

Host immunological mechanisms mediate tissue destruction and bone resorption in response to bacterial infection. IL 1,2,6 TNFά

In previously treated cases, bacteria may be present due to missed canal or coronal leakage.

Bacteria involved in initial necrotic case – Gram -, anaerobes

Provotella	Lactobacillus
Porphyromonas	
Fusobacterium	Peptostreptococci
Veillonella	Streptococci
Eubaterium	*mixed infection, polymicrobial
Propionibacter	3-17 species, symbiotic relationship
Actinomyces	

Sundqvist – redirected understanding of canal flora – predominantly anaerobic but mixed with facultative anaerobes.
Baumgartner – apical 5 mm, predominantly anaerobic, BPB found in both coronal and apical area, most common found was P. nigrescens.

Fabricus – number of anaerobes increases with time and apical position

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

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

Sundqvist – also found E. Faecalis, frequently as a single species microorganism.
Retreatment success rate ~74%

Nair – found yeast-like microorganisms, therapy resistant

Waltimo – Candida (resistant to many medicaments)

Gomes – Predominantly found same bacteria but also noted that symptomatic cases had anaerobes (pepto, porph., provet, fusos)

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

Species associated with refractory cases

Strep
Enterococci
Staph

Lactobacillus

Proprionibacter
Eubacterium
Actinomyces

Prevotella, Fusobaterium

Causes of E. Faecalis Resistance

Love – because the hide in the tubules

Distel – because they form biofilms

Evans – because the have a proton pump

Haapasalo – Ca(OH)2 – does not kill E. Faecalis / smear layer removal facilitates bacterial invasion into dentinal tubules.

Orstavik – dentin buffers Ca(OH)2

Baumgartner – 2% CHX kills E. Faecalis

Bacteroides Melanogenicus – new nomenclature

Bacteroides – ferment carbs

Porphyromas: asacrolytic

Prevotella: sacrolytic

Is HIV found in the root canal or apical lesion ?

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

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

Sebeti 2004 JOE – found Herpes simplex, Epstein-Barr & human Cytomegalovirus in periapical lesions. Large lesions showed higher levels.

Are Bacteria associated with symptoms?

YES

1. Newton – Bacteroides Melanogenicus are associated with pain, sinus tract and odor
2. Hahn – Gr+ cocci & Gr- rods = cold sensitivity
3. Sundqvist - >6 species = pain, 5 or less = no pain

NO

1. Baumgartner – no relationship of BPB w/ symptoms

Are bacteria found in periapical lesions? Controversy

YES

1. Iwu – homogenized study
2. Siqueira – Biofilm colonys
3. Sunde –

NO

1. Walton – inflammation resists spread of bacteria, confined to root
2. Nair – bacteria confined to root except
    a. Abscess
    b. Therapy resistant – actinomyces
    c. Infected cysts
3. Holland – bacteria are present when pushed out during RCT

Sjogren – isolated P. propionicum extraradicularly
Waltimo – no candida in AP and is resistant to Ca(OH)2
Torabinejad & Trope – found HIV in AP

Discuss bacterial flora in acute PA abscesses

Langeland – found facultative and anaerobic bacteria/ fuso & streptococcus

Siqueira – described flora as polymicrobial

Sundqvist – BPB's in abscess – associated with purulence

Bacteremia from RCT

Baumgartner – very low incidence (3.3%) / none if inst. kept w/in canal

Tronstat - ~25% even when instrument is confined to canal

Antibiotic Susceptibility

Baumgartner –

1. Pen VK 1st choice – 85% effective
2. Amoxicillin – 91% effective
3. Amox + Clavulanic acid 100% effective
4. Clindamycin 96% effective
5. Metronidazole – 45%*

*due to effect only on anaerobic bacteria

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 – Bergenholtz found bacteria 64% of the time/ mixed anaerobic infection, got in through tubules or cracks.

Do Bacteria grow into the tubules?

YES –
Haapasalo – E. Faecalis survived in tubules 10days w/out nutrients
Sen – bacteria penetrate 10-150 microns into the tubules
Oguntebi – bacteria in tubules is a reservoir for future infections

Does anachoresis occur?

YES

1. Robinson – 2 requirement – inflammation & bacteria
2. Gier – Bacteria are attracted to inflamed pulps

NO

1. Doyle
2. Moller

Coronal Leakage

1. Ray/ Trope – coronal seal more important than quality of RCT
2. Siqueira – loss of coronal seal led to contamination w/in 3 days
3. Torbinejad – complete contamination of entire length w/in 30 days of loss of seal.
4. Newton – recommends retreatment if exposed for 3 months
5. Walton – post prepared canals susceptible to endotoxin penetration even faster than bacteria / recommends immediate restoration

Ricucci – 3 year study – no effect from exposure to oral environment, questions role of coronal leakage.

Why do we get anesthetic failures?

Hargraves – Endodontic Topics Vol.2

1. Anatomic causes – variations in anatomy
2. Acute tachyphylaxis
3. Effect of Inflammation on local tissues (pH)
4. Effect of Inflammation on blood flow – vasodilation
5. Effect of Inflammation on nociceptors – allodynia
6. Effect of Inflammation on central sensitization
7. Psychological factors

Differential Diagnosis of non odontogenic pain

P – psychogenic – Manchausens

I – Inflammatory – Sinusitis

N – Neurovascular – Cluster headaches

S – Systemic – Myocardial Infarct

M – Musculoskeletal – Myofacial pain (TMD)

Give a differential diagnosis for a periapical radiolucency

1. Granulom
2. Cyst
3. Abscess
4. Scar
5. Foreign body reaction
6. OKC
7. Ameloblastoma (multilocular)
8. Central Giant Cell Granulom (multilocular)
9. Metastatic malignancy (breast, prostate, kidney)

Bhaskar – no distinction radiographically between cyst and granuloma

Nair – Incidence of Cyst, Abscess, and Granuloma
Cyst 15%, (True 61%, Pocket 39%), Granuloma 50%, Abscess 35%

Give a differential diagnosis for a periapical radioopacity

1. Periapical cemental dysplasia – cementoma
2. Focal sclerosing osteomyelitis
3. Idiopathic osteoscleroses
4. cementoblastoma
5. calcifying odontogenic cyst
6. calcifying epithelial odontogenic tumor
7. adenomatoid odontogenic tumor

What are the current theories of cyst formation?

1. Epithelial proliferation – Seltzer, epithelial cells proliferate to line abscess 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. Breakdown Theory of Cyst formation – 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 reaction responsible for proliferation of epithelium.

NAIR - POCKET CYSTS HEAL AFTER RCT / TRUE CYSTS DO NOT

Zones of Fish

1. Infection/ Necrosis – bacteria, PMSs
2. Contamination – bacterial toxins, lymphocytes, macrophages
3. Irritation – macrophages, osteoclasts, lymphocytes, plasma cells
4. Stimulation – osteoblasts, fibroblasts

Describes bodies way of isolating and localizing an infection in periradicular area

How do silver points cause a problem?

Seltzer et al – silver wires removed from failed endodontic cases showed corrosion products of silver sulfate products which are cytotoxic.

Leakage from around the round wire within not such a round canal causes washout of the cement and fluid contact with the silver wire. Oxidation of the wire leads to the corrosive byproducts.

Focal Infection – Does it occur today?

No – Correlation does not mean causation !

"focal infection" term coined by WD Miller 1890 found gangrenous pulps could act as centers of infection causing alveolar abscesses.

William Hunter attributed a multitude of diseases to "focal infection"
Billings 1912 introduced "focal infection" theory to USA
Rosenow, reported that streptococci present in diseased organs could establish an infection in a distant organ after traveling through the blood stream.

No evidence - does not pass scientific scrutiny

Siqueira – endodontic infections can cause bacteremia – no evidence that organisms from RCT can cause disease in remote sites.

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

Are antibodies present in the pulp?

YES
Langeland – antigens in the root canal system can initiate an immune response with antibodies

Martin – Immunoglobulins are present in the pulp which react with microorganisms

Hahn – IgG, major class of immunoglobulins in normal and irreversible groups.

Pulver – Normal pulps do NOT have immunoglobulins-containing cells. In inflamed pulps, IgG most common, IgA, IgE, IgM containing cells are also seen.

What is the role of the neuropeptides?

Byers – injury leads to "sprouting" of CGRP fibers

Wakisaka – neuropeptides may help regulate pulpal blood flow + pain transmission

Olgart – sensory nerves may play a role in instant (increase blood flow) defense reaction in the pulp.

Hargraves – sympathetic transmission may modulate pain (capsaicin study)

Vascular response to pulpal inflammation

Kim - key components in pulpal inflammation

1. microcirculation – increased PBF by C fiber stimulation (neurokinins, substance P, released from c fiber nerve terminals)

2. sensory nerve activity – excitatory/inhibitory effect from increased/decreased pulpal blood flow via increased tissue pressure.(A delta fibers)

What cells are found in a periapical granuloma?

Stern's study results –
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%

Perrini – found mast cells in varying stages of activity
Pulver – found 70% IgG, 14% IgA, 10% IgE and 4% IgM
Cysts have 45% IgG, 45% IgA, 5% IgE and 5% IgM
Torabinejad – Granulomas & Cysts have T and B cells, T Cells were in greater quantity.

Cellular Activity & their Mediators

1. Ito – Macrophages & Fibroblasts produce PGE2 which may contribute to the osteolytic resorption of periapical lesions.
2. Hamachi – Macrophages contribute IL –1, an important activator of osteoclastic bone resorption.
3. Torabinejad – NK Cells – defensive role in controlling root canal infections
4. Fouad – Pulpal and periapical pathosis were independent of the presence of functional T and B cells
5. Torabinejad – high concentrations of LTB4 in symptomatic human periapical lesions

Cytokines and their activity

Cytokines are soluble polypeptide products of immune cells.

Modify behavior of other cells
Produce systemic effects
Act as growth factors

1. Stashenko – IL1alpha – contributes to resorptive activity
2. Stashenko – IL1alpha, IL1beta, TNFalpha, PG, bradykinin, and LPS – stimulate resorption either alone or in synergistic combination.
3. Safavi – TNF identified in periapical exudates from CAP
4. Stashenko – IL1alpha and beta, TNF and lymphocyte-derived lymphotoxin potentially stimulate resorption and inhibit reparative bone formation

Describe the Compliment cascade

1. Mediate vascular responses
    a. Histamine release via C3a and C5a anaphylatoxins
2. Recruiting phagocytic leukocytes
3. Opsonizing targets of phagocytic cells (C3b)
4. Directly damaging target cells (C5-9 MAC)

Most important step is cleavage of C3

Classical pathway is activated by Ab coated targets or Ag-Ab complexes (IgM,IgG)
Alternate pathway is activated by LPS, aggregated IgM or IgG, Ag-IgG complexes, plasmin

Who studied LPS?

Schilder –

1. pulpless teeth contain greater concentration than vital teeth
2. symptomatic w/ AP contained greater concentrations than asymptomatic

Berganholtz –

1. endotoxic 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

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 nerves at the pulpo-dentinal junction.
4. distortion leads to impulse conduction

What lines sinus tracts?

Baumgartner –

1. 100% of sinus tracts are lined with epithelium to the level of the rete ridge
2. 67% had granulomatous tissue lining the tract
3. 33% had epithelium lining the entire way

Harrison –

1. Sinus tracts may be lined with epithelium 10%
2. lined with granulation tissue 90% of the time

LA complications:

1. stimulatory phase: talkativeness/agitation
2. Generalized convulsive state
3. CNS depression

LA toxicity treatment:

Protect patient
Monitor & record vitals
Provide supportive therapy
    Keep patient supine
    O2 w/ 10L flow/min
    Maintain BP
    Treat bradycardia (0.4 mg atropineIV)
Transport to hospital

<u>Trowbridge-Inflammation</u> - Histopatholoty of foreign body reaction

Type I Anaphylactic, IgE seconds-minutes Anaphylaxis (drugs, insect bite)

Type II Cytotoxic, IgM,G--Transfusion rxn, autoimmune

Type III Immune complex IgG form complexes w/ complement 6-8 hrs
ex: serum sickness, arthus, immune vasiculitis, lupus, viral hepitits

Type IV -Cell mediated immunity-Delayed Hypersensitivity rxn: more important than anaphylactoid, b/c lots of T cells and macrophages,
ex. 48 hours contact dermatitis
infectious granulomas (TB)
tissue graft rejection
chronic hepatitis

Lymphocytes (T>B), macrophages, lots of pmn's

Pulpal Changes as related to depth of bacteria –

1. Baum – found correlation between depth of penetration of bacteria and severity of inflammation
2. Brannstrom – 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 >1mm 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. Torneck – tissue from pulp exp = abscess components, nerves least effected
6. Langeland – Carious exp caused increased PMSs, chronic inflammatory cells cause most cell injury.
7. ***Classic*** **- Seltzer/Bender – described classic caries progression to pulp**

Are Mast Cells in the Pulp ?

Farnoush – yes, found in inflamed and un-inflamed pulpal tissue

Who found lymphatics in the pulp ?

Bernick – demonstrated lymphatics in the pulp

Heyerass - the pulp may have a beneficial blood flow increase during inflammation in spite of simultaneously increased tissue pressure. This supports the concept of lymphatic drainage.

Do Odontoblast process extend into the tubules?

1. Sigel – odontoblastic process extends to DEJ

2. Holland – Odontoblastic process extends ½ way through the tubule.

3. Aubin – Odontoblastic process extends to DEJ

4. Thomas – dentinal tubules are lined throughout their length by an organic structure, the lamina limitans, which can be mistaken for odontoblastic processes.

Who studied pulpal vasculature ?

Kim & Tekahashi – discovered presence of arteriovenous anastomosis and venous-venous anastomosis and u shaped arterioles (unique feature of pulpal vascular network)

Also found sympathetic adrenergic vasoconstritor fibers

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 self-strangulation (lymphatic drainage)

Pulp Stones / pulpal calcification

1. Bernick – age causes decreased vascularity, nerves, pulp chamber size and increased calcified masses in the pulp

2. Hendricks-Klyvert – incidence of calcifications 8-90%

    a. Pulp stones – calcifications
    b. Denticles – composed of dentin

Resorption

Internal
External

- Surface (transient)
- Replacement
- Inflammatory
    - Lateral
    - Cervical / sulcular
        - Subepithelial external resorption
        - Invasive cervical resorption
        - Extracanal invasive resorption
        - Periodontal infection resorption
    - Apical
    - Pressure

Classification of Resorption

Tronstat - classified root resorption
- Transient Inflammatory (surface)
- Progressive Inflammatory
  - Internal
  - External
    - Cervical
    - Replacement

Fuss – classified root resorption according to stimulation factors
- Pulpal infection
- Periodontal infection
- Orthodontic pressure resorption
- Impacted tooth or tumor pressure resorption
- Ankylotic resorption – *no bateria required* (Suda)

Gartner – discussed buccal object rule to identify ext from internal resorption

Causes of Resorption – Theories

Trope – two requirements for root resorption

1. loss or alteration of the protective layer (pre-cementum or pre-dentin)
2. inflammation must occur to the unprotected root surface

Osteoclasts will not adhere to or resorb unmineralized matrix

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

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

Causes of Internal Resorption

Wedenberg –

1. Dentin contains a resorption inhibitor, macrophages do not grow on pre-dentin.

2. Internal resorption cannot develop unless normal pulp is replaced by a periodontal-like connective tissue.

Treatment of Internal Resorption

Caliskan et al –

1. conventional RCT is the treatment of choice for non-perforating internal resorptive defects.
2. If perforated, CaOH2 (remineralization) should be attempted, but surgery may be necessary.
3. 90% success with non-perforating using 1 wk CaOH2 and warm condensation
4. 25% success with perforating resorption

Stamos – use ultrasonics to clean and warm gutta percha obturation technique

Discuss External Resorption

1. Kuperman – Inflammatory tissue resembling perio connective tissue grew into the canal from the defect
2. Trope – dog tooth study, long term CaOH2 (12 wks) more effective than short term (1 wk)

Discuss Invasive Cervical Resorption

1. Torabinejad – not well defined, only macrophages not other inflammatory cells. Treatment based on location of resorption (supraosseous, crestal or infraosseous)
2. Frank & Bakland – may be asymptomatic with normal pulp. Treatment may not involve RCT.
3. Heithersay – strong association exists between invasive cervical resorption and orthodontic treatment, trauma, and intracoronal bleaching, either alone or in combination. Recommends using TCA for treatment.

Orthodontic treatment, resorption and endodontics

Mattison – No difference was seen in external root resorption between endodontically treated teeth and vital teeth when subjected to orthodontic forces.

Stressed pulp syndrome – effect of restorative dentisty on the pulp

Abou-Rass - teeth with stressed pulps should be endo treated before restoring

Felton – full coverage restorations led to a higher incidence of pulp morbidity

Berganholtz – abutment teeth undergo necrosis more often (15%) than crowned non-abutments (3%)

Would you leave a tooth open to drain?

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

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"

Bence – Avoid leaving teeth open to prevent flare-ups when reclosing.

Simon – described oral pulse granuloma due to legumes.

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

Kuttler – distance from the major to the minor diameters
0.525mm (18-25y/o)
0.659mm (>55 y/o)

Burch – measured from the occlusal aspect of the major diameter to the apex
Average distance for all roots = 0.59mm

Stein – Measured from the minor diameter (CDJ) to the major diameter = 0.72mm average. Foramen width increases with age but CDJ width does not.

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

Conciderations 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.

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
2. Holland – ferret study – dentin + CaOH = 15% inflammatory response

Ca(OH)2

1. Hicks – CaOH 2mm thickness effective apical barrier
2. Torabinejad – CaOH has role in the induction of root end closure (apexification) than the presence of exogenous calcium.

MTA

1. Andreasen – in a guide for traumatic injuries, he recommends:
    a. MTA apexification after 2-4 wks of CaOH, MTA thickness should be 4 mm.
2. Torabinejad – Apexification w/MTA, place CaOH for 1 wk in infected cases, place MTA, close w/wet cotton + cavit, obdurate after 4 hours.

Does CaOH2 Kill Bacteria

Yes 1. Siqueira – pH (12.5) alters enzyme activity disrupting cellular metabolism
   a. Hydroxyl ions create free radicals – destroy cell membranes
   b. Free radicals react with bacterial DNA, inhibition

2. Sjogren – 7 day CaOH eliminated most bacteria which survived instrumentation

3. Trope – CaOH inactivates LPS

4. Peters – CaOH limit but does not totally prevent re-growth of endodontic bacteria

5. Law 2004 – CaOH remains the best medicament available to reduce residual microflora beyond instrumentation effort.

6. Joyce 2004 – Heat 46C enhanced the antibacterial action of 0.12% CHX and 10% CaOH2 against E. Faecalis w/out increasing toxicity.

7. Mickel 2003 JOE – thin mix more effective antibacterial than thick mix

Does CaOH dissolve tissue?

Maybe

1. Hasselgren, Cvek et al – CaOH completely dissolved porcine muscle tissue in 12 days. The addition of NaOCl after treatment with CaOH dissolved the tissue in as little as 60 minutes.

2. Morgan – CaOH solution was an ineffective solvent of pulpal tissue

3. Baumgarder – intracanal medication with CaOH or NaOCl for 1-7days to aid in dissolving tissue remnant was ineffective.

4. Turkun – Pretreatment with CaOH enhanced tissue dissolving efficacy of 0.5% NaOCl to the level achieved with 5% NaOCl. CaOH causes tissues to swell and become more accessible to the NaOCl.

How do you place CaOH?

Krell – describes using the Messing gun to place CaOH

Sigurdsson – compared CaOH placement – Found the lentulo is better than injection (Calasept) which is better than placement with a K file.

Does CaOH have an effect on the apical seal?

1. Porkaew – CaOH did not increase apical leakage
2. Wesselink – CaOH dressing does not effect the seal however study may be insignificant because CaOH decolorizes methylene blue (dye used)
3. Kim et al – CaOH groups showed significantly more dye leakage than the non-medicated control group.

CaOH mixture

Hosoya 2001 – aqueous mixture gave optimum peak pH change after 14 days
Therefore use for 14 days, powder alone – peak pH change 49 days.

Pacios 2003 – CaOH2 aqueous solutions in CHX, propylene glycol, anesthetic, CMCP, CMCP-PG all maintained alkaline environment.

Can CaOH be removed from the canal effectively?

YES

1. Margelos – Remove CaOH completely before using a ZOE sealer to avoid prolonged set time. EDTA is recommended for removal.
2. Baumgartner – Irrigants (H2O, NaOCl, EDTA) effectively removed CaOH dressing.

Can CaOH diffuse through dentin?

1. **Tronstad** – pH is decreased during resorption. Teeth filled with CaOH have *increased* pH in the surrounding dentin. (7.4-11) The pH of cementum /PDL is not effected by CaOH in the canal. Increased dentinal pH may be the mechanism for stopping resorption.

2. Foster – CaOH diffuses through root dentin to exterior surface, removal of smear layer may facilitate this diffusion.

3. Nerwich – 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.

Does CaOH weaken Dentin?

YES

Andreasen – limit use to a few weeks, strength was not reduced in study during period of 30 days

Cvek – longer term use in immature teeth weakens tooth structure.

EAL

1. Suzuki – Original research in dogs, electrical resistance between periodontium & mucous membrane = constant value 6.5K ohms
2. Sunada – applied Suzuki's findings to humans, contant = 6,500W (ohms)
3. Kobayashi – developed the ratio method of electronically determining working length. Root ZX measures impedance at 8 and 0,4kHz, and calculates a quotient of the impedance.
4. Baumgartner – Root ZX accurately locates minor diameter ~90% of the time
5. Shabahang – Root ZX is ~96% accurate to within 0.5mm of the apical foramen.
6. Fouad – EAL enhances length control throughout treatment, reduces x-rays

How accurate is radiographic working length estimation?

Weiger 2001 – x-ray determined WL 0-2mm short of apex causes unintentional overinstrumentation in 51% of premolars and 22% of molars.

Does any solution effect the Root ZX?

NO

1. Meares et al – Root ZX not adversely affected by presence of NaOCl
2. Schindler – No difference in length determination as a function of the seven irrigants used.

Does apical resorption affect the Root ZX?

NO

1. Goldberg – apical root resorption did not effect determination of working length

Does Pre-Flaring help with the ZX?

Yes

1. Ludlow 1999 – Apical foramen could be reached more consistently by preflaring the canals before obtaining working length.

Uses of EAL

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

Does pulp status matter with the root ZX?

1. Dunlap – no statistical difference in accuracy between between vital and non-vital cases with ratio unit
2. Pommer – vital more accurate than non vital with non ratio units

Can you use an EAL with a pacemaker patient?

1. Dorn – 4 of 5 locators tested did not cause inhibition or interfere with normal pacemaker function in vivo
2. Hutter - **Check with MD & pacemaker manufacturer !!!**

Canal Preparation

Serial Preparation
Brilliant – serial preparations were more effective than nonserial preps in romoval of tissue @ all 3 levels

Walton – tapering prep permits better debridement of apical canal, reduces overinstrumentation of foramen and improves ability to obdurate.

Clem	step back
Goerig	step down
Torabinejad	passive step back
Marshall	crown down pressureless
Roane	balanced force
Fava	double flare

Discuss the benefits of the balanced force technique

1. Wu – balanced-force technique produced a cleaner apical portion of the canal than the other techniques.

2. Sepic – less apical transportation with balanced force technique in canals exhibiting curvature of more and less than 45 degrees.

3. McKendry – Balanced force technique extruded less debris

4. 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 – Biconical tip files (Flex-R) produced the least transportation and no ledges.

3. Moser – Tip design contributes more to cutting and efficiency than flute design.

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

1. Torabinejad – The ability to determine the apical constriction by tactile sensation was significantly increased when the canals were pre-flared.

2. Walton – Changes in working length, although statistically significant, were very small (0.17mm) and clinically unimportant.

3. 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.

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.

Compare hand stainless steel files with hand NiTi instruments.

1. Cunningham – NiTi files were more effective in maintaining the original canal path of curved root canals when apical preparation was enlarged beyond #30.

2. Walker – NiTi files 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. Zmener – NiTi files prepared more centered and tapered preparations than conventional K-files.

Compare hand stainless steel files with rotary NiTi instruments.

1. Baumgartner – Lightspeed and Profile were faster and stayed centered better than stainless steel hand files.

2. Toda – Rotary files were faster and decreased undesirable outcomes such as zip, elbow or ledge.

3. Messer – Rotary instrumentation may produce better canal shape versus stainless steel by reducing procedural errors.

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 more bacteria?

NO

Trope & Orstavik –

JOE 2000

1. NiTi rotaries are NOT more effective for microbe elimination than hand instruments. Profile and 1.25% NaOCl decreased bacteria 62%, 1 week exposure to CaOH decreased bacteria 93%.

JOE 1998

2. There was no detectable difference in colony-forming unit count after NiTi rotary or stainless steel hand instrumentation.

Does preflaring help with rotaries?

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

How much surface area does instrumentation clean?

1. Peters – While instrumentation of canals increased volume and surface area, all instrumentation techniques left 35% or more of the canals' surface area unchanged.

NOTE – THAT MEANS THAT WE EFFECTIVELY ONLY CLEAN ABOUT 65% OF THE SURFACE AREA !!!!!!!

What are the properties of NiTi?

Haikel – NiTi has 2 phases: **Austentite & Martensite**

2 properties: **Superelasticity & Shape memory**

1. The ability to cycle between these two phases is due to its properties
2. Phase transition occurs with rapid stress on the file, therefore use at a constant speed.
3. Files are weakest during phase transition (cyclic fatigue)
4. Radius of curvature was found to be the most significant factor in determining the fatigue resistance of files.
5. Cyclic fatigue is a major cause of instrument failure.

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

Svec – Even the smallest NiTi instruments can be used multiple times unless there is a visible distortion of the instrument.

Does sterilization affect NiTi instruments ?

Hicks – Heat sterilization of rotary NiTi files up to 10 times does not increase the likelihood of instrument fracture.

Cunningham – Neither the number of sterilization cycles nor the type of autoclave sterilization affect the torsional properties, hardness and microstructure of stainless steel and NiTi files.

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 used at 333.3 rpm showed separation /distortion 4X as often as files used at 166.67.

3. Dietz – Profiles .04 are less likely to break at lower rotational speeds

4. Daugherty – Profile .04 Series 29 rotary instruments should be used at 350rpm, which nearly doubles the efficiency and halves the deformation rate when compared to 150rpm.

What about ultrasonics. How do they work?

Cunningham 1982 – Ultra sonic preps produced had cleaner canals and reduced smear layer better than hand instruments. Ultra sonics energizes irrigating solution by cavitation.

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.

Do ultrasonics remove bacteria?

Hoshino 1998 – Ultrasonic irrigation with 5.5% NaOCl eradicated bacteria from infected dentin.

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

Are Ultrasonics effective in canal cleaning?

Hutter 1999 – 3 min passive activation of either sonic or ultrasonic produced significantly cleaner canals than hand instrumentation alone. Also, there is NSD in cleaning efficacy between sonic and ultrasonic activation.

Reader 1992 – Combination step-back with ultrasonic instrumentation (3 min) resulted in a cleaner preparation than step-back technique alone in bothe the canals and isthmus.

Walker – NSD between soninc, ultrasonic and hand instrumentation regarding debris removal and canal wall planing in curved canals.

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.

Discuss the Hollow Tube Theory.

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

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 implants. **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 imflammatory response** at either end of the polyethylene implant.

Gutta-Percha

What is in gutta-percha?
Friedman 1975 – 20% gutta-percha, 66% zinc oxide, 11% heavy metal sulfates (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?

Gutta-percha is highly cytotoxic in cell culture experiments –

1. Spangberg 1990 – toxicity 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-1alpha.

3. Nair 1995 – Large pieces of GP were well encapsulated bya collagenous cacpsule, and the surrounding tissue was free of inflammation. The fine particles evoked an intense, localized tissue 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 and that the coloring agent is erythrosine.

2. Schilder 1974 – Gutta percha exists in a beta semicrystalline state. It undergoes 2 transformations upon heating from 0-100C. The beta to alpha transition occurs at 42-49C; the alpha to amorphous at 53-59C. Compactable not compressible.

What do you know about apical decompression?

1. Freedland 1970 – Use polyvinyl tubing for access and irrigation of large PA lesions

2. Burg 1982 – Decompression is an alternative to surgical enucleation

3. Decompression is done to avoid:

    a. Devitalization of adjacent teeth
    b. Damage to anatomic structures (IAN, Sinus)
    c. Loss of bony support
    d. Parassthesia
    e. Elderly pts where surgery is risky

Latex allergy vs Gutta Percha

1. Johnson – Gutta-percha does not have the same allergenicity as latex

Cross-Reactivity studies of gutta-percha, gutta-balata, and natural rubber latex (Hevea brasiliensis). J Endod. 2001 Sep;27(9):584-7.

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.

Horizontal root fracture –

Mechanowitz – healing of root fracture occurs from the PDL

Methods of healing of root fractures –

3 types as per Andreasen -

1. Fibrous connective tissue
2. Osseous
3. Cemental

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 anaverage of 12 years.

Is routine trephination required?

No

1. Moos – Pulpectomy alone provided signigicantly better postoperative pain relief at 4 hours compared with pulpectomy /trephanation. At no time interval did the trephination group have less pain than the group without trephination.

2. Reader - The study did not find that trephination significantly decreased pain, percussion pain or swelling. It was therefore determined not to be routinely recommended for symptomatic necrotic teeth with radiolucencies.

3. 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?

1. Rosenberg - Occlusal reduction should prevent postoperative pain in those patients whose teeth initially exhibit pulp vitality, percussion sensitivity, preoperative pain and/or the absence of a periradicular radiolucency.

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.,

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.

EDTA – a disodium salt solution that collects Ca ions and replaces with Na, making dentin softer.

What is the smear layer?

1. McComb & Smith 1975 – 1st to describe the smear layer.

2. Baumgartner 1984 – found two layers, frequency and depth varied
    a. A thin layer on the surface of the canal walls 1-2 microns thick
    b. A layer in the dental tubules up to 40 microns

3. Sen 1995 – The smear layer is made up of inorganic and organic debris. (pulp, bacteria, bacterial by-products)

Does the smear layer effect the apical or coronal seal?

Holz – a better apical seal occurred when the smear layer was removed with EDTA.
Krell – Apical seal of obturation is not adversely affected by irrigation w/EDTA

Jeansonne 1997 – Less coronal leakage was seen when the smear layer was removed. AH-26 displayed less leakage than Roth's 811sealer

Should the smear layer be removed?

1. Torabinejad 2002 – Suggests removal of smear layer to decrease bacteria and improve adaptation of obturation materials. MTAD (doxycycline, citric acid and Tween-80 detergent) will facilitate smear layer removal.

2. Yang 2002 – Given that the smear layer produced during root canal preparation promoted adhesion and colonization of P. nigrescens to the dentin matrix, it might also increase the likelihood of canal reinfection.

3. Gunday 1993 – Removal of the smear layer reduced leakage significantly.

4. White 1987 – Penetration into dentinal tubules by filling materials is possible after the smear layer removal.

5. Moss 2001 – controversy exists w/in endo community re smear layer removal

How long should EDTA be used?

1. Schilder 1974 – reports that excess EDTA will react with 73% of the available inorganic dentin component. EDTA works most rapidly during the 1st hour. An equilibrium forms within 7 hours. EDTA has self-limiting properties.

2. Calt 2002 – 1 minute irrigation of EDTA is effective in removing the smear layer. A 10 min application of EDTA causes excessive peritubular and intertubular dentinal erosion.

3. Baumgartner 1987 – EDTA and NaOCl used alternately as an irrigant is effective in removing organic and inorganic debris (removes smear layer)

4. Yamada 1983 – For removal of smear layer, use final flush w/ 10ml of 17% EDTA (organic) followed by 10 ml of 5.25% NaOCl (inorganic)

MTAD

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 – Component and value
    a. doxycycline – prevents E. Faecalis in 100% of samples
    b. Tween-80 – reduces surface tension, increases dentin penetration
    c. Removes smear layer w/out erosion of dentin

Do intracanal medicaments decrease pain?

Hasselgren 1989 – The use of various dressings did not contribute to the relief of pain.

Trope 1990 – No significant difference was found in the flare-up rate among the three intracanal medicaments.

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.

What about Steroids?

Marshall 2002 / Endodontic Topics

Effect of glucocorticoids on inflammation:

- 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.

Treatment of Endodontic Pain with 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 rout 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 Studies of Steroid usage:

1. Chance 1987 – Intracanal corticosteroids are recommended after intrumentation of vital pulps to reduce post-op pain. Pain was reduced significantly over saline.

2. Morse 1984 – Corticosteroids (dexamethasone) in canals reduce post-op pain. Vital teeth were used for the study

3. Pierce 1987 – Ledermix (tetracycline corticosteroids mix) is recommended as an intracanal medication to minimize inflammation associated with root resorption in traumatized teeth.

Systeminc Use of Steroids:

1. Marshall et al – PreTX Dx – IP/AP, IM Dexamethasone reduced severity of pain at 4hrs & 8hrs and 0.07 to 0.09mg/kg dosage alone reduced pain at 8 hrs.
2. Reader 1999 – Pre Tx Dx Necrotic/CAP, w/mod-sev pain, mild to mod swelling. Intraosseous injection of 40mg methyprednisone & clean and shape canals – Results = less pain during 7 days PO & less meds required.
3. Reader – same as above but no prior swelling, Oral admin of steroid, 48mg medthyprednisolone. Results = less post op pain for 3 days and less medications needed.
4. Morse 1989 – Inter appointment pain, oral dose reduced pain at 8 hours, 24 hours and 48 hours. Preop Dx = aymptomatic vital-inflamed pulps ??????

Injection Techniques for Steroids

Intraosseous-

Reader 2000 – single dose of IO steroid reduced pain over 7 days – Depo-Medrol, temporarily alleviates the symptoms of irreversible pulpitis until treatable.

PDL-

Kaufman 1994 – intraligamentary injection of methylprednisolone (Depomedrol) reduced the frequency and intensity of post-operative pain.

Do prophylactic antibiotics decrease flare-ups? NO !

Walton 1993 – Using penicillin prophylactically to control post-treatment symptoms is not recommended in cases of pulp necrosis and asymptomatic periapical pathosis. Placebo = Penicillin for post op pain.

Fouad 1996 – Patients with localized periapical pain or swelling recovered with local treatment. NO benefit from penicillin regarding decreased symptoms or quicker recovery. No justification for indiscriminated use of abx.

Reader 2000 – Pen VK did not significantly reduce pain, percussion pain, or the number of analgesic medications taken for patients with untreated irreversible pulpitis. Therefore, penicillin should not be prescribed to treat irreversible pulpitis.

Reader 2001 – The administration of penicillin postoperatively did not significantly reduce pain, percussion pain, swelling or the number of analgesic medications taken for symptoms in cases of symptomatic necrotic teeth.

Do prophylactic antibiotics decrease flare-ups? YES

Torabinejad 1994 – His study found that Ibuprofen, ketoprofen, erythromycin base, penicillin, and methyprednisolone plus penicillin were more effective than placebo within the first 48 hours following complete instrumentation.

Morse 1987 – 1 day of high dose Pen VK reduced flare-up incidence from 20% to 2%

What is a flare-up?

A flare-up is an acute exacerbation of periapical pathosis after initial or continuation of root canal treatment.

What are the incidence of flare-ups?

1. Walton 1992 – 946 visits resulted in an incidence of 3.17% flare-ups. Flare-ups increased to 19% with severe presenting symptoms, 7% with pulp necrosis, and 5% with acute apical periodontitis.

2. Imura 1995 – Factors associated with flare-ups: multiple appt., retreatment, periradicular pain prior to treatment, presence of radiolucent lesions, and patients taking analgesics. Study reported an incidence of 1.58% from 1012 teeth.

3. Tronstad 1979 – Study reported 91% success from RCT and NO decrease in success if flare-up occurs.

4. Baumgartner, Svec et al 1983 – Risk factor for post obturation pain was extrusion of sealer or gutta-percha. No relationship with vitality, apical lucency, root # or level of obturation. Pain rate w/in first 24 hrs.= 47.6% (14% severe)

What are the incidence of flare-ups? Continued

5. Torabinejad 1988 – Factors associated with endodontic interappointment emergencies of teeth with necrotic pulps
    a. Age
    b. Sex of patient
    c. Presence of preoperative pain
    d. Presence of allergies
    e. Absence of PA lesions
    f. Sinus tract
    g. Retreatment cases
    h. Those receiving prescribed analgesics

    Factors that had no effect on the frequency of emergencies
    a. Presence of systemic disease
    b. Use of intracanal medications
    c. Penetration of the foramen with small instruments during length determination

Discuss intentional replantation. What is the prognosis?

Kratchman 1997 – Dental Clinics of North America – Success rate 80-85%

Grossman – 70% at 5 years

Bender & Rossman - 81%

Keonig – 82%

Is irrigation with an antimicrobial necessary ?

Bystrom & Sundqvist 1981 – Mechanical instrumentation reduced the number of bacteria 100 – 1000 fold and bacteria persisted even after 4 visits.

How large should the apical preparation be for irrigation?

1. Brilliant 1977 – For proper irrigation apex preparation should be size #30

2. Abou-Rass 1982 – A 30 gauge irrigation needle can be placed in the apical 1/3 of the canal when the apex is size #30.

3. Rosenberg 1995 – The Maxi-Probe probes 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.

4. Teplitsky 1987 – Endosonics facilitate apical movement of irrigants, even with an apical preparation as small as 0.1mm. Syringe irrigation is effective when the apical preparation is at least 0.3mm.

Is Chlorhexidine an effective irrigant?

1. Jeansonne 1994 – No difference in antimicrobial activity 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 apecies.

2. White 1997 – Antimicrobial activity lasted 72 hours after use with 2% CHX, 0.12% produced 6 –24 hrs. (it binds to dentin and is released over time- "substantivity")

3. Weber – confirmed "substantivity" effect of CHX

4. Saunders 2002 – 1% CHX efficient in eliminating E. Faecalis from dentinal tubules. CaOH was also effective at 3 & 8 days but not at 14 days ??????????? IN VITRO STUDY

Is Chlorhexidine an effective irrigant? Continued

5. Siqueira 2001 – Only 2% CHX was able to eliminate most of both 1 & 3 day E. Faecalis biofilms.

6. 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.

7. Gomes 2003 – 2% CHX was more effective against E. Faecalis than CaOH.

8. Baumgartner 2003 – $CaOH_2$ + 2%CHX was more effective killing E. Faecalis in the dentinal tubules than CaOH + H_2O.

Discuss NaOCl. What concentration is best?

1. Baumgartner 1978 – 5.25% is safe for clinical use, does not increase PO pain

2. Harrison 1978 – Dilution of 5.25% adversely affects tissue dissolving ability

3. Baumgartner 1987 – NaOCl mixed with EDTA or H_2O_2 is safe to use in the canal; no chlorine gas was produced. NaOCl + H_2O_2 yeilds NaCl + H_2O + O_2

4. Cunningham 1980 – 2.6% sodium hypochlorite solution at room temp was found to be equally effective as a collage-dissolving agent when compared to 5.25% at body or room temp. Inc. temp inc. efficiency.

5. Raphael 1981 JOE – no direct relationship between temp & antibacterial effect (biased study due to culture reversals)

Discuss NaOCl. What concentration is best? Continued

6. Torabinejad 2003 – As pulp solubilizers 5.25% and 2.6% NaOCl were equal (>90%), and 5.25% NaOCl was capable of dissolving virtually the entire organic component of dentin.

7. Pashley 1985 – The antimicrobial efficacy of NaOCl is due to its abilty to oxidize and hydrolyze cell proteins and to osmotically draw fluids out of cells due to its hypertonicity.

8. 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.

What about CMCP?

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 connective tissue response ranging from severe inflammation to necrosis.

What about CMCP? Continued

4. Haapassalo & Orstavik 1987 – Studied the disinfection of dentinal tubules – smear layer removal facilitates bacterial invasion of dentinal tubules. Calasept (CaOH) 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.

5. Ferguson 2002 – CaOH + CMCP when in direct contact were effective antifungal agents (against C Albicans)

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.

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

Spasser 1961 – Sodium perborate – "walking bleach" the sealing of a pledget of cotton wool 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 broke down the darkly pigmented macromolecules into smaller lighter colored molecules. The technique results in cervical root resorption 6-8%, if heat is used to activate the superoxol the rate rises to 18-25% (Pathways).

Harrington JOE 1979 – Theory: Superoxol seeps through patent dentinal tubules and initiates an inflammatory resorptive response in the cervical area.

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.

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

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.

Papadopoulos EDT 1996 – All CEJ junction types showed leakage of H2O2 form the chamber, but the teeth with gaps (10%) in the CEJ had higher values compared to the other 2 types.

Preventing Resorption

Friedman JOE 1993 – Mixing sodium perborate with H2O produced equal bleaching results and minimized incidence of external root resorption.

West JOE 1994 - The external cervical root resorption associated with intracoronal bleaching of pulpless teeth can be a devastating lesion. It often cannot be repaired. To prevent this problem, increasing attention has been focused on placing a barrier between the pulp chamber and the endodontic filling material. The objective of this article is to propose a method for determining the location and shape of an intracoronal bleach barrier.

Rotstein JOE 1992 ?? - recommends placing a 2 mm protective base (??Glass ionomer) to avoid radicular penetration of H2O2. 14% of teeth have defects at the CEJ vs 10% as reported by Mjor.

Can You Bleach tetracycline stained teeth? (Intrinsic stain)

Abou-Rass JOE 1982 – found intentional RCT and internal bleaching is sometimes an effective treatment for tetracycline stains when other methods cannot be applied.

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?

Torneck JOE 1993 – bond strength is adversely effected by bleaching

Silva IEJ 2001 – Microleakage increased as a function of bleaching, short term use of CaOH2 after bleaching did not increase microleakage ?????

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 w/master cone)
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.

Tell us about 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 Emdo 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 that 3 CaOH sealers.

4. Pearson IEJ 2003 - 1. AH Plus showed the greatest stability in solution and Tubli-Seal EWT performed well, but Apexit and Endion had higher solubility

values. 2. The film thickness values in increasing order were: Tubli-Seal EWT<Apexit<Endion=Roth 801<AH Plus. 3. The flow rates for all sealers were similar. 4. The working times for all sealers were greater than 50 min. 5. Roth 801 did not set when incubated in volumes sufficient to fill the test matrices.

Tell us about sealers – continued

5. 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.

6. Wilcox JOE 1991 – This study evaluated and compared 4 methods of sealer placement: file, lentulo spiral, ultrasonic files, and master gutta-percha cone. No difference was seen.

7. Grossman JOE 1976 – Roth's sealer has little shrinkage when set

Is sealer extrusion a concern?

8. Bernath IEJ 2003 –
    a. Apex & Grossman's = no periapical inflammation if confined to canal
    b. AH-26 & Endomethasone = +periapical inflam even if confined to canal
    c. All 4 initiated periapical inflammation if overfilled

9. Baumgartner JOE 1983 - Extrusion of sealer or gutta-percha was associated with increased pain. Overall incidence of postobturation pain was 47.6%

10. Augsburger 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

11. Kim OOOO 1992 – Eugenol may cause extensive tissue damage. Keep exposure to a minimum. Forms Zinc Eugenolate when mixed with zinc, easily hydrolyzed by saliva and other liquids.

12. Shore JOE 1995 – Biocompatability of sealers
    a. Roth's 811 & Sealapex – mod-severe inflam rx
    b. CRCS - mild – moderate inflam rx
    c. AH-26 - most irritant

13. Leyhausen JOE 1999 – Genotoxicity and Cytotoxicity of resin-based sealers
    a. AH-Plus slight to no cellular injuries
    b. AH-Plus no genotoxicity or mutagenicity
    c. AH-26 is cytotoxic due to formaldehyde release, not in AHPlus

Should we use orifice sealers after obturation? What material ?

1. Saunders IEJ 1997 – Vitrebond is an effective barrier for preventing microleakage I 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 barrier showed no leakage, group w/out barrier showed bacterial penetration in 15 to 76 days. Eugenol containing sealer had no effect on the bonding agent.

Discuss Sargenti paste –

1. Sargenti technique – no rubber dam needed, access not addressed, length – somewhere near apex, objective is chemical (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 – demonstrated short-term, 6 mo and 1 yr severe cytotoxicity in monkeys

3. Spangberg 1974 – formaldehyde is responsible for extensive tissue necrosis. Not resorbable therefore must be surgically removed if expressed beyond the apex.

4. Allard 1986 – described case if N2 induced paresthesia

5. Kleier 1988 – described painful dysesthesia of IAN due to Sargenti paste

6. Serper 1998 JOE – Paraformaldehype pastes are neurotoxic

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 1/3 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. Camps JOE 2001 – System B vs other tech. – regardless of the technique the apical leakage increased after 1 month with fluid transport
4. Baumgartner JOE 2002 – Lateral vs Continuous Wave - NSD in bacterial leakage. Lateral did leak faster though.
5. Reader JOE 1993 – Lateral vs Warm Lateral, vs Warm Vertical to fill lateral canals – NSD in quality of fill between techniques but more GP was found in lateral canals with warm techniques.

Does obturation cause vertical root fractures?

Holcomb JOE 1987 – excessive force can cause vertical root fracture. 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.

Hicks JOE 1989 – Described 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

Hatton JOE 1988 – High condensation forces are not needed. 1 kg force produced equal seal up to 2.5kg force.

Do warm Gutta-Percha techniques damage the PDL?

1. Zach OOO 1965 – Monkey study, effect of temp increase on pulp
    a. 4 degree – pulps recovered
    b. 10 degree – 85% recovered; 15% necrotic
    c. 20 degree – 40% recovered; 60% necrotic

d. >20 degree – none recovered

2. Baumgartner JOE 2001 – This study tested the change in radicular temperature associated with use of System B and Obtura. At no time did the external root temperature increase more than 10 degrees C.

3. Gutmann JOE 1987 – Confirmed Baumgartner's study and found maximum temperature change from placement of GP with Obtura was 1.1 degrees C. Noted that 10 degree C change will damage PDL cells. Temps never reached this high.

What do you know about Thermafil ?

1. Baumbardner JADA 1995 – Lateral vs Thermafil – Thermafil leaked most, maybe due to stripping of carrier upon insertion.

2. Gutmann IEJ 1993 – Thermafil looked better on radiograph than lateral condensation but caused more overextensions.

3. Gutmann IEJ 1993 –
    a. overfilling was common problem with Thermafil
    b. Lat condensation had more leakage at 7 days, but no difference at 24 hrs and 5 months.

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

Is lates 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
    a. Pre-test GP w/ latex sensitive pt by allergist
    b. consider premed w/ prednisone and diphenylhydramine
    c. prepare for the management of allergic rx w/ EpiPen

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. Torabinejad JOE 1993 – MTA had significantly less leakage following repair of experimentally created root perforations than IRM or amalgam.

2. Holland JOE 2001 – No inflammation was seen and cementum was deposited over MTA in this dog study of lateral root perforations

How does intrapulpal anesthesia work?

1. Rosenberg JOE 1975 – Intrapulpal anesthesia produces effect via pressure

What are other techniques available besides IANB? Gow Gates

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
    a. Technique: anesthetized V3 – target – lat side of condylar neck

i. Dry & apply topical for 1 minute
ii. 25 gauge needle (long)
iii. 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.
iv. 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)
v. Deposit 1.8cc over 60-90 seconds, may use up to 3ml
vi. Use rubber block 1-2 minutes for diffusion
vii. Return to upright and wait 5 minutes (due to diameter of nerve or greated distance to nerve trunk)

What are other techniques available besides IANB? Akinosi

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

2. 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.

a. Technique: anesthetize same at IANB, target – med. lingual border of ramus (above IANB below Gow Gates)
    i. Dry & apply topical for 1 minute
    ii. 25 gauge needle (long)
    iii. 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.
    iv. Advance needle 25mm (ave) from tuberosity, aspirate, deliver 1.8ml over 60 sec., wait 5 min. (motor nerve effect will reduce trismus)

What are other techniques available besides IANB? Incisive

3. Reader JOE 1992 – Incisive block alone didn't give pulpal anesthesia.

4. Malamed Textbook Page 249 – "Pulpal, buccal soft tissue and bone anesthesia is readily obtained" with the Incisive nerve block. Lingual tissue is not anesthetized.
    a. Technique: no need to enter target mental foramen (traumatic)
        i. Dry & apply topical for 1 minute
        ii. 25 gauge (short)
        iii. 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.
        iv. Maintain gentle finger pressure over site to increase volume of solution entering MF (intra or extra orally) for 2 minutes.
        v. Wait 3-5 minutes to begin treatment.

Compare different anesthetics?

1. Spangberg JOE 1993 – Lip anesthesia not reliable indicator of pulpal anesthesia. (Aβ fibers NOT Aδ) DDM (endo ice) reliable method of testing pulpal anesthesia. 3% mepivacaine is = to 2% Lidocaine.

2. Dagher JOE 1997 – NSD in success and failure of anesthesia by varying concentrations of epinephrine.

3. Fortsch OOO 1992 – Injections w/out epi had higher rate of failure. Epi increased duration of pulpal anesthesia, no effect on onset time. NSD between 1:100,000 & 1:50,000

4. Reader JOE 1993 – Compared IANB w/ 4% Prilocaine, 3% Mepivacain and 2% Lidocaine w 1:100,000 epi. NSD in onset, success or failure.

Reasons for local anesthetic failures

1. Anatomic/accessory inervation

2. Acute Tachyphylaxis – reduced responsiveness of receptor to drug
3. Inflammatory effect on pH – lowers pH, blocks Na channels
4. Inflammatory effect on tissue blood flow – increases flow – carries away LA faster
5. Inflammatory effect on nociceptors – TTX-resistant Na channels (TTX resistant Na channel activity doubles after exposure to PGE2-inflammatory mediator)
6. Inflammatory effect on Central sensitization – exaggerated CNS response to even gentle peripheral stimuli
7. Psychological factors – apprehension causes reduced pain threshold

Approaches for managing failures:

1. Reader – supplemental LA: 2nd block with 3% mepivacaine, PDL, intraosseous, intrapulpal
2. Hargraves – Adjunctive drugs: NSAID (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 –
    a. Initial symptoms - Tremors, muscle twitching and convulsions
    b. Later findings – Respiratory depression, lethargy and loss of consciousness.
    c. Final findings – Cardiovascular depression and hypoxia secondary to respiratory depression can rapidly produce serious outcomes including cardiovascular collapse, brain damage and death.
2. Vasoconstrictor reactions
    a. Initial signs – palpitations, increase heart rate and elevated BP
    b. Anxiety, nervousness and fear are often found as well.
    c. Severe overdose, arrythmia, stroke and MI are possible

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

How do you manage a Local Anesthetic Overdose? Continued

3. Management from Little & Falace
    a. Protect patient during convulsive phase, consider IV Valium
    b. Monitor and record vitals
    c. Supportive therapy
        i. Supine position
        ii. O2 10L/min
        iii. Maintain BP
        iv. Treat Bradycardia w/ 0.4mg atropine IV
        v. EMS
    d. CPR if unconscious

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

Methemoglobinemia

1. Caused by metabolites of Prilocaine (MRD=4mg/lb) & Benzocaine
2. symptoms occur 1-3 hrs after treatment
    a. cyanosis without respiratory distress when met-Hgb reach 10-20%
    b. vomiting and headache have been described
    c. dyspnea, seizures, stupor, coma and death at levels higher than 20%
3. Patients at increased risk
    a. Heart disease
    b. Anemia
    c. G6PD deficiency
    d. Children < 2yo
    e. Elderly

Allergic Reactions to local anesthetics

1. Appearance –
    a. Urticaria
    b. Erythema
    c. Intense itching
    d. Angioedema & respiratory distress (more severe reaction)
    e. Anaphylactic reaction
2. Sulfite antioxidant reactions
    a. Asthma-like signs of tachypnea, wheezing, bronchospasm, dyspnea, tachycardia, dizziness, and weakness
    b. Severe flushing, general urticaria, angioedema, tingling, purities, rhinitis, conjunctivitis, dysphasia, nausea, and diarrhea

Hass 2002 J Can Dent Association –

1. Biotransformation 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. Methehoglobinemia is associated with articaine and benzocaine.

3. Articaine and prilocaine are associated with increased paresthesia

4. Malignant hyperthermia occurs with exposure to inhalation anesthetics, not local anesthetics.

5. Lidocaine and prilocaine are pregnancy category B; others are C

6. 7mg/kg is max lido dose. (4.4mg/kg is Malamed's max/conservative)

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.

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.

Anxiolytic therapy, what do you use ?

1. Hargraves & Dionne 1993 OOOO – Triazolam (0.25mg) appears to be safe, effective alternative to parenternal sedation with a benzodiazepine for dental outpatients.

2. Hutter & Dionne 1997 JOE – Orally administered Triazolam (0.25mg) is safe and more effective anxiolytic agent than diazepam (5.0mg) 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.

Discuss the 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 to anesthetize one tooth; adjacent teeth are affected.

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.

Is intraosseous anesthesia effective ?

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

1. NSD was noted between 2% Lido/100 epi and 2% Mepivacaine/20 levo. Both increased anesthetic success to 100%. 80% of pts experienced a mean increase in heart rate of 23-24 bpm.

2. IANB only 25% successful with irreversible pulpitis. Intraosseous injection w/ 3% mepivacaine increased success to 80%. Second intraosseous injection increased success to 98%. **CONSEPT -** If med cond precludes epi mepivacaine although not as efficacious as Lido/100 will be effective with 2 carpules.

3. 67% of patients had heart rate increases (23-24 bpm) with intraosseous anesthesia. Increase is not clinically significant in most healthy patients. In patients whose medical condition, drug therapies or epinephrine sensitivity suggests caution, 3% mepivacaine is a good alternative.

Is intraosseous anesthesia effective ? Continued

4. 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/100 was successful when conventional therapies failed.

5. Anderson 1998 JOE – Stabident IOI was an effective supplemental anesthetic technique in 89%. More success in mandible than maxilla. (91% vs 67%)

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 =
    a. Calcium sulfate
    b. Glycol acetate
    c. Triethanolamine
    d. Polyvinyl acetate
    e. Polyvinylchloride acetate
    f. Red pigment

2. Webber 1978 OOO – A 3.5mm thickness of Cavit should be used in order to prevent leakage.
3. Balto JOE 2002 – IRM leaked after 10 days, Cavit and Dyract leaked after 2 wks
4. Pashley 1998 JOE – Cavit, Cavit-G, TERM and glass ionomer cement provided leakproof seals during the 8 wk testing period.

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

5. Eleazer 2001 JOE – Cotton trapped between the wall of the tube and the filling material dramatically reduced the sealing quality of the temporary restoration.

6. Stark 1990 OOO – Cavit had the best sealing ability, IRM showed the maximum dye penetration.

7. Hutter JOE 1996 – Cavit provided a bacterial leakage-free seal for 3 wks.

8. Mayer JOE 1997 – Cavit showed less leakage in the dye penetration test and fewer marginal crevices.

9. Deveaus JOE 1999 – Invitro leakage test – cavit leaked less than TERM, IRM, and Fermit.

How much room should be left for a post space?

1. Neagley OOO 1969 & Madison JOE 1984 - agree that at least 4mm 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 10-15mm in length, leaving at least 4-5mm of gutta-percha

4. apically. Posts should be parallel and cemented, not screwed.

5. Standlee 1978 J Prosthetic Dentistry – Posts should be at least the height of the crown or 9mm minimum.

Should you place a post immediately to avoid leakage ?

YES

1. Metzger JOE 2000 & Wu J Prothetic Dent 1998 –Both agree; Post prepared canals 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.

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. 4mm 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 eugenol in sealer affect the retention of the post ?

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YES

1. Nemetz 1992 – The findings of this study demonstrated a substantial decrease in retention of posts luted with Panavia composite resin cement in the presence of eugenol.

NO

1. Hagge IEJ 2002 – Eugenol containing sealer, 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. Walker 1998 JOE – The 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. Schindler 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.

Dose a post put stress on the tooth or cause tooth fractures?

Stein 1992 – Beveled preparations provided an increased resistance to root fractures. Vertical fractures occurred twice as often with nonbeveled preparations. (ferrule-effect is important for Fx resistance)

Chan 1982 – Cast posts reduced force before tooth fracture

Jeansonne JOE 1998 – Carbon post had no root fractures

Akin JOE 1992 – Stress patterns within the root are altered as a result of post insertion. (non uniform along root, maximum bending stresses are associated with the apical termination of the post)

Randow 1986 – Patients detect pressure earlier in vital teeth vs non vital; vital intradentinal nerves are able to register pressure. Helps explain higher incidence of cracks in root filled teeth.

Are Endodontically teeth more brittle ?

Sedgley JOE 1992 – Vital dentin was 3.5% harder than endodontically treated teeth however the biomechanical properties of endo treated teeth and their contralateral vital pairs indicates that teeth do NOT become more brittle following endodontic treatment.

Douglas JOE 1989 – Endodontic procedures reduce stiffness by 5%.

Schilder JOE 1992 – The results of this study do NOT support the theory that dehydration after endodontic treatment per se weakens dentin structure in terms of compressive and tensile strengths

Is cuspal coverage important for endodontically treated posterior teeth ?

1. Caplan 2002 – Endodontically treated teeth not crowned after obturation were lost at a 6.0 times greater rate than teeth crowned after obturation.

2. Sorensen 1984 – Coronal coverage improved success of molar and premolar endodontic treated teeth.

3. Linn 1994 JOE – Endodontically treated molar teeth are considered susceptible to 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 moar teeth.

4. Tidmarsh JOE 1976 – Endodontically treated posterior teeth should be protected by overlaying cusps or placement of a well-retained core with a crown

What is a NICO lesion?

Neuralgia Inducing Cavitational Osteonecrosis – aka – "Ratner's Bone Cyst"

Bouqout et al –

1. Diagnosed by exclusion, technetium scan or multiple radiographs
2. Histology – iscemic 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 reoccurrence)

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.

3. Suter 1998 JOE – describes placing a 21 gauge needle over a separated instrument, then pushing a Hedstrom file thru the needle in a clockwise direction to interlock the separated instrument with the needle and the Hedstrom file. All three parts can then be removed coronally.

How do you remove posts?

1. Berbert 1995 IEJ – 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 1997 JOE – 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. Abbott 2002 IEJ – post removal is a predictable procedure with good case selection. Incidence of root fracture is rare.

4. Seto 1994 JOE – Ultra sonic units are more effective than sonic units.

Is chloroform safe for retreatments ?

1. Kaminski 1998 JOE – No health risk to the patient, amount expelled thru the apex (0.32mg) is several orders of magnitude below the permissible toxic dose (49mg/m)

2. McDonald 1992 JOE – Chloroform is safe for the dentist and staff. Air vapor levels were well below the OSHA mandated levels.

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.

Are other means available to remove gutta-percha ?
Can it be removed completely ?

1. Kaplowitz 1990 JOE – tested 5 solvents but found chloroform was the only one that totally 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 debrie 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 ? continued

5. Hansen 1998 JOE – tested several solvents to remove different sealers. Only chloroform removed AH-26

6. Metzger 1995 JOE – presented procedure for removal of overextended root canal filling.
    a. Soften gutta-percha and remove to a distance of 2-3mm short of apex
    b. Remaining gutta-percha is removed by a Hedstrom file
    c. Extend file 0.5-1.0mm beyond the apex
    d. Firmly engage the gutta-percha and slowly remove.

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.

Bertrand 1997 JOE – used chloroform and hand files to remove Thermafil

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

Hicks 1999 JOE – this study tested the application of a System B at 225 decrees C (carrier melts at 300). It recommends heat and insert system B 10-15mm for 5-8 sec, then instrument by hand 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, formaldehyde and resorcinol, polymerizes when 10% NaOH is added. Forms brick hard red material that has no solvent. Requires no instrumentation, presumably "fixes" tissues and kills bacteria.

2. Hartwell 2003 JOE – This study tested the effectivness of 0.9% NaCl, 5.25% NaOCl, chloroform, or Endosolv R on softening Resorcinol. NaOCl was superior to all other groups after 5 minutes.

What are some of the anatomical considerations during periapical surgery ?

SINUS

1. Torabinejad 1992 OOO – MB root of Max 2nd molars is closest to the sinus (1.97mm ave), farthest from the buccal bony surface. (4.45mm ave). Max 1st bicuspid is closest to the lateral boney surface (1.63mm ave) but farthest from the floor of the sinus (7.05mm). 5% of apicies protrude into the sinus.

2. Lin & Langeland 1985 JOE – This study recommends the use of antihistamines in the event of a sinus perforation (0.5% neosynephrine). Add antibiotics only if acute sinus develops, do **not** give prophylactically.

What are some of the anatomical considerations during periapical surgery ?

SINUS – continue

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.

4. Bernhart DCNA 1997 – This study found that sinus perforations occurred in 28% of maxillary posterior endodontic surgeries.

5. Torabinejad 1997 OOO – Sinus perforations tend to repair (regardless of size) with limited bony covering and fibrous scar. Furthermore, resorbable collagen membranes do not improve osseous repair.

MENTAL FORAMEN/NERVE –

1. Mioseiwitsch 1995 JOE – This study described 3 steps to minimize risks of damage to the neurovascular bundle exiting the mental foramen.
    a. Take Vertical periapical film
    b. Use triangular flap with the vertical releasing incision distal
    c. 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:
    a. Inferior to the mandibular second bicuspid
    b. 60% of the distance from the buccal cusp tip to the inferior border of the mandible.
    c. It exits in a posterior and superior

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. Kim 1995 JOE & Gomes 2003 IEJ/ Agreed that: – Incidence of an isthmus was highest in the apical 3-5mm levels. In teeth with two canals the 4mm section contained a complete or partial isthmus 100% of the time. Not treating the isthmus may be responsible for endodontic failures.

3. Gilheany 1994 JOE – Apical leakage may be reduced by resecting at a 0-degree bevel and increasing the depth of the retrograde filling . Recommend 3.5mm retroprep depth to extend coronal to pulpal termination of the tubules.

Discuss root end resection. How far should you resect ? Bevel ?

4. 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.

5. Gutmann 1993 IEJ – Root end resection is indicated for the following:

    a. Removal of anatomical variations or operator errors
    b. To enhance removal of a lesion
    c. To gain access to the apical canal
    d. To evaluate adaptation of the root canal filling
    e. To reduce fenetrated apicies
    f. To explore aberrant anatomy or fractures

Compare ultrasonic and rotary root end preparation:

NOTE – Richman was 1st to propose use of ultrasonics, 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. Torabinejad 1994 JOE & Gorman 1995 JOE / Agreed that: – This study compared root end preparations and found ultrasonic preps were deeper, more parallel and cleaner. (SEM study)
3. 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.
4. Baumgartner 1997 JOE – Ultrasonic preps were deeper, had less bevel, deviated less from the canal and less boney crypt was required.

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. Morgan 1999 JOE – Invivo study that found no cracks after the root resection but one incomplete crack after the ultrasonic root-end preparation.

3. Torabinejad 1995 OOO – This study found a higher incidence of cracks in the walls of ultrasonic root-end preparations as those prepared by a bur. Crack formation is a function of time, power and dentin thickness.

Does the ultrasonic tip cause cracks in the root end ? continue

NO

1. Baumgartner 1999 JOE – The use of the diamond-coated instrument resulted in a heavily abraded, debris-coated cavosurface that may affect the apical seal. Root-end cracking was NOT seen.

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 and should be used with care. Only one microcrack was seen; incidence is low.

4. Lumpley 1997 OOO – Ultrasonic root end preparations did not produce root face cracking

Discuss hemostasis during surgery –

1. Gutmann IEJ 1996 – 4 actions of hemostasis with collage:
    a. Stimulation of platelet adhesion, aggregation, release
    b. Activation of factor XIII (Hagamen) and other clotting factors
    c. Mechanical tamponade
    d. Release of Serotonin (5-HT)

2. Kim DCNA 1997 –
    a. Bone wax – **Aurelio:** acts mechanically via tamponade effect, foreign body reaction may occur if left in surgical site. (Bees Wax)
    b. Chemical vasoconstrictors (epi)
        i. Racellets and Epidri – cotton impregnated w/ racemic epi. (immediate vasoconstriction) little systemic absorption. Placed on bone w/ another cotton pellet and pressure for 2-4 minutes.

ii. Ferric sulfate – **Jeansonne:** agglutination of blood proteins that occlude capillary orifices. Cytotoxic and causes tissue necrosis therefore it must be removed to prevent delayed healing. **Baumgartner:** no significant cardiovascular effects
iii. Thrombin – not studied for endodontic applications

c. Absorbable hemostatic agents
i. Calcium sulfate – tamponade effect, biocompatible, resorbs in 2-4 weeks
ii. Gelform – animal skin gelatin, promotes platelet disintegration. Stimulates thromboplastin release and thrombin formation. Not applicable to endo surgery.
iii. Collagen – causes platelet aggregation & fibrin formation. If applied directed w/ pressure = hemostasis in 2-5min. Does not inhibit healing.
iv. Surgicel – oxidized regenerated cellulose. Acts mechanically by forming a sticky mass when in contact with blood. Inhibits healing & stim. Inflam. 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 ave. 9.5 ml, range 1.2-48.4ml. (comparable to single tooth extraction)

2. Buckley J Perio 1984 – Study indicated significant reduction of blood loss using 1:50 vs 1:100 epi. (50% less blood loss)

3. Lindorf OOO 1979 – This study discussed the rebound effect (reactive hyperemia) after injections with epi.

Does removing the smear layer on the resected root end improve post surgical healing?

YES

1. Harrison JOE 1993 - Deminieralizing the root end with citric acid enhances cementogenesis and dentoalveolar healing in dogs. It acts by exposing collagen fibrils in 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.

2. Zhu JOE 2000 – Cell adhesion and spreading 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 EBA and MTA ?

1. Andreasen JOE 1993 – Studied composit retrofill, produced reformation of periodontium including reformation of a lamina dura, inserting Sharpey's fibers and cementum deposition.
2. Chong IEJ 1997 – Studies Vitrebond vs ZOE or 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 material – result 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 greated 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 – This study supported SEBA as best retrofilling material vs glass ionomer, amalgam, IRM and composite

What is MTA ?

Components:

1. Tricalcium silicate
2. Dicalcium silicate
3. Tricalcium aluminate
4. Bismuth oxide
5. Tetra calcium aluminoferrite (not in white MTA)
6. Calcium sulfate hydrate (gypsum)

Discuss studies using MTA for root end fillings –

1. Torabinejad JOE 1993 – MTA showed less leakage than SEBA or amalgam.
    a. Advantages of MTA:
        i. Easy to mix
        ii. Dry field not required
        iii. Excess is easy to remove
        iv. Less periradicular inflammation than amalgam
        v. Cementum found on surface after healing
        vi. Less bacterial or endotoxin leakage than amalgam, SEBA or IRM (90 days)
        vii. Will not be effected by resection after it sets (cut thru material)
    b. Disadvantages of MTA
        i. Long setting time

Discuss healing after surgery –

Harrison 1991 & 1992 JOE –

1. Incisional wound – connective tissue and epithelium remains attached to root surface after reflection. Preservation of this tissue prevents epithelial down-growth along root surface and loss of attachment. Reattachment of epithelial seal occurs in 1 day, connective tissue attachment in 2 days. Submarginal and intrasulcular flaps heal equally well. Use undermining technique to reflect flap.
2. The dissectional wound – healing occurs slower than incisional wound, occurs by day 4, completed by day 14. Granulation tissue replaces fibrin clot (day 4). Periosteum does not survive flap reflection. Don't curette cortical-retained periosteal tissues, they are a source of reattachment. Crestal bone levels should reduce following endo surgery.
3. The osseous wound – New bone matrix was formed directly on devitalized bone surface. Periosteum (deliminating membrane) separates overlying mucosa from excisional wound site. Does not function until

excisional wound is almost filled with woven bone of endosteal origin. Osteoblastic activity in excisional wound site is observed in 14 days post surgery.

Kaminski 1984 JOE – Submarginal incision is the flap of choice in periapical surgery when not contraindicated by anatomical location of the lesion or by insufficient attached gingival. Healing, bone loss and recession were greater with intrasulcular design. (Attached gingival must be 3 mm wide between base of sulcus and incision for submarginal design)

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 displayed 16 week results post op from apical surgery to be the same as undisturbed bone.

Velvart 2002 IEJ – Papilla based incision – allows rapid and predictable recission-freee healing following surgical exposure of the soft tissues.

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 JOE – applied emdogain to pulp cap procedure – produced wound healing and dentin formation greater than dycal in pigs.

2. Bamaas 2001 Dent Traum – applied emdogain in replantation cases of extended extraoral dry time – makes root more resistant to resorption and stimulates new formation of PDL from the socket. (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 promotes healing of root resorption.

Discuss GTR in endodontic surgery –

CaSO4

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 exclude connective tissues, allowing bone regeneration during healing.

Discuss GTR in endodontic surgery – continued

GTR is beneficial

1. Pecora 1997 DCNA – Indications for GTR in endodontic surgery include:
    a. Through and through periapical lesion
    b. Large periapical lesion
    c. Endo-perio lesion
        i. Periapical lesion communicating with alveolar crest
        ii. Furcation involvement as a result of perforation
        iii. Root perforation with bone loss to alveolar crest

Gutman 2001 JOE – Dog study w/Guidor – The use of a bioresorbable membrane enhances apical regeneration of bone, connective tissue attachment and marginal alveolar bone significantly over time.

Discuss GTR in endodontic surgery – continued

GTR is not needed

1. Hartwell 2002 JOE – human study invivo – NSD between rate of healing with or without a Guidor membrane. 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.

What is the prognosis for luxation injuries ?

Injury	Andreasen FM, Pedersen	Crona-Larsson	Dumsha
Concussion	3% necrosis		2%
Subluxation	6%		
Extrusion	26%	15%	98%
Lateral luxation	58%	33%	77%
Intrusion	85%	44%	100%

Ravn 1981 – Retrospective study of incisors w/ enamel-dentin fracture

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.

What would you do for a crown fracture and why ?

Uncomplicated crown fracture	❑ Baseline pulp test ❑ Smooth edges or restore with composite ❑ Place Dycal base on exposed dentin ❑ F/U at 2mos
Complicated crown fracture	❑ DPC if small, <24 hours, and open apex ❑ (dry CaOH2 on exposure, then Dycal, then restore) ❑ Cvek pulpotomy if larger, >24 hours, or closed apex ❑ (remove 2mm of pulp with diamond and H2O spray, then DPC). ❑ Pulpectomy if necrotic, uncontrolled hemorrhage ❑ F/U at 3, 6, 12mos, then annually
Crown-root fracture	4 options after removing coronal fragment: ❑ Gingival reattachment ❑ Crown lengthening ❑ Ortho extrusion ❑ Extraction

What would you do for a crown fracture and why ? continue

Cvek 1982 JOE – Monkey study – this study examined the inflammatory reactions of pulp exposures from fractures or cavity preps at different times. Findings indicated that in crown-fractured teeth with vital pulp exposures up to a period of 7 days, not more than 2mm of pulp beneath the exposure needs to be removed. NOTE – foundation study for CVEK PULPOTOMY FOR TRAUMA CASES !!

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)

De Blanco 1996 OS – Cvek pulpotomy is successful and permanent treatment for crown fractures w/pulp exposures regardless of size of exposure, maturity of the root or inteval between accident and dental treatment.

What would you do for a root fracture and why ?

Root fracture	❑ 3 radiograph angles ❑ Reposition ❑ Rigid splint 2-4 months ❑ Adjust occlusion ❑ F/U at 3, 6, 12mos, then annually ❑ RCT of coronal segment if necrosis, apical matrix may be needed ❑ SX removal of apical segment if possible or necessary

Andreasen 1967 JOS – 4 types of healing/ location of fx did not determine success, mobility of coronal segment is important

1. Calcified (callous)
2. connective tissue
3. bone/connect tissue
4. granulation/inflammatory (nonunion)

What would you do for a root fracture and why ? continued

Barnett & Tronstad et al 1988 Endo Dent Trauma – Rigid splinting for 2-4 months is recommended for horizontal root fractures

Bender JADA 1983 – Recommends 3 radiographs with different vertical angulations to view horizontal fracture

Cvek & Andreasen 2001 Dent Trauma – A positive effect of splinting (various methods) could NOT be demonstrated. In conclusion, the findings from this study have cast doubt on the efficacy of splinting for root fracture healing; hard tissue consolidation of a root fracture may take place more advantageously under functional stress.

What would you do for a root fracture and why ? continued

Keller 2002 Dent Trauma – Clinical and epidemiological study of traumatic root fractures

1. 34% had no complications
2. 40% developed pulpal necrosis
3. 6% had root resorption
4. 20% developed pulpal canal obliteration
5. 54% no treatment needed

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
3. optimal treatment –
    a. reposition
    b. fixation
    c. relief of occlusion

What would you do for a luxation injury and why ?

Lateral Luxation	❑ Reposition and physiologic splint for 2-3 weeks ❑ Adjust occlusion ❑ Complete RCT if no response to vitality test in 3 weeks in closed apex ❑ Frequent F/U in open apex to R/O necrosis
Extrusion	❑ Same as lateral
Intrusion	❑ Open apex: loosen tooth with forceps, then allow to spontaneously ❑ Re-erupt, intervene with ortho if needed ❑ Closed apex: ortho repositioning is preferred ❑ Repositioning should be completed by 3 weeks ❑ RCT at 3 weeks

Trope, Pathways of the Pulp: if vitality testing indicates necrosis at the 3 wk f/u, RCT should be performed due to high success of RCT in noninfected pulp versus risk of external root resorption complication. Why does RCT reduce risk of external resorption if factors described by Trope himself indicate a) break in protective layer and b) inflammation, as the predisposing factors for resorption?

What would you do for a luxation injury and why ? continued

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. Bhaskas 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:
    a. Observation for spontaneous reeruption – minimal intrusion
    b. Surgical reposition – w/ early RCT to prevent anklyosis
    c. 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.

Storage materials:

1. HBSS - **Trope**
    a. **Provided 7 hrs of storage**
2. Viaspan - **Trope**
    a. Comparable storage times to HBSS
3. Milk (low fat is better- **Walker**) pH and osmolarity are compatible **Blomlof & Trope**
    a. **Provided 6 hours for storage**
4. Saline
5. Saliva - **Blomlof**
    a. **provided 2 hours for storage**
6. water – hypotonic – causes cell lysis

Andreasen 1986 – saline storage of a previously dry tooth has no effect on resorption or pulpal repair. A delay of replantation is acceptable if the teeth are placed in saline.

Discuss splinting of avulsed teeth;

Physiologic Splint

1. Antrim 1982 JOE – describes a technique using 30lb monofilament nylon line and acid etch resin to splint traumatically luxated or avulsed teeth. The splint stabilizes traumatized teeth and allows for physiologic movement.

2. Castelli et al1982 OOO – Replanted teeth splinted for 7 days recovered uneventfully. Extended splinting periods (30 days) induced root resorption and ankylosis.

3. Hicks 1988 JOE – Monkey study - No difference observed in healing in replanted teeth w/ physiologic, rigid or no splint.

4. Blomlof 1985 EDT – This study examined the effect of diet texture on healing after replantation. The hard diet induced less ankylosis.

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, Viaspan, milk, saline or saliva
3. Proceed to office

Andreasen 1981 Int J OS – a relationship exists between extra-alveolar time, storage medium and root resorption. After 30 min of dry storage, resorption is very prominent.

General Adjuncts to trauma treatment:

1. tetanus booster
2. chlorhexidine rinses
3. analgesics
4. recall for 5 years

How would you manage an avulsed tooth clinically? Continued

Avulsion, open apex - < 1 hour dry

1. Rational: attempt to promote revitalization
2. Soak 5 minutes in a suspension of 1mg doxycycline in 20ml physiologic saline*
3. HBSS rinse, replant
4. Physiologic splint 7-10 days
5. Recall every 3 weeks for up to 18 months to monitor for root formation or necrosis
6. If necrotic, proceed with apexification

*Cvek 1990 EDT – This study showed a decreased frequency of microorganisms in the pulpal lumen and less ankylosis or inflammation as a result of the soaking. Frequency of revascularization related to size of open apex. >4mm = 33%, >1mm=18%, less revascularization if replanted after 45 minutes.

How would you manage an avulsed tooth clinically? Continued

Avulsion open apex >1 hour dry

1. Poor prognosis for revascularization, consider alternate treatment plan
2. Rational if replanting: PDL is dead, prepare root to resist resorption
3. Remove tissue tags with scaler
4. Soak in 2% stannous fluoride for 5 minutes (**Trope – Pathways)**
5. Complete RCT extraorally, due to open apex, no time constraint (PDL is already dead)
6. Physiologic splint 7-10 days

Klinge 1989 – Soaking in SnF2 will delay remodeling of the root into bone (replacement resorption)

Coccia 1980 JOE – Treatment of the root with Fl before replanting makes it osteoclast resistant, delaying replacement resorption. 2X survival time expected.

How would you manage an avulsed tooth clinically? Continued

Avulsion with partially or fully closed apex, < 1 hour dry

1. Rinse with HBSS, replant
2. Physiologic splint 7-10 days
3. Clean and shape canal at 7th day, remove splint (rational: prevent infection of canal that leads to inflammatory resorption) Canal should not be infected to warrant 6 months dressing
4. Place CaOH2 for 7-10 days
5. Obturate canal when CaOH is removed

Dumsha 1995 IEJ – 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 obdurate with gutta-percha.

Trope 1992 JOE – confirmed Dumsha's study and recommended initiating RCT 14 days after replantation.

How would you manage an avulsed tooth clinically? Continued

Avulsion partially –fully closed apex, > 1 hour dry –

1. Rational: PDL is dead, prepare root to resist resorption
2. Remove tissue tags with scaler
3. Soak in 2% stannous fluoride for 5 minutes
4. Physiologic splint 7-10 days
5. Clean and Shape canal at 7 days, remove splint
6. Place CaOH2 for 7-10 days
7. Obturate canals at 7-10 days

Same supporting articles as previously listed by
Klinge re: SnF2 to delay replacement resorption
Coccia re: Fl to delay replacement resorption, 2X survival time expected
Dumsha re: NSD in inflammatory resorption when filled with GP vs CaOH2
Trope re: short 1 wk vs long 8 wk CaOH2 tx, similar healing patterns

What are some factors that affect healing of avulsed teeth?

1. Andreasen 1966 –
    a. PDL showed 4 types of healing:
        i. Normal
        ii. Replacement resorption
        iii. Surface resorption
        iv. Inflammatory resorption
    b. 90 % of teeth replanted w/in 30 min = no resorption
    c. majority of teeth replanted after 90 min = resorption

2. Blomlof 1985 EDT – In avulsion cases, if the PDL is damaged, removal of the damaged PDL with NaOCl may reduce resorption. Destruction of >20% of the root surface is required for replacement resorption to occur.

3. VanHassel 1980 – all 90 minute dried (monkey) teeth showed ankylosis and replacement resorption. All saliva-stored teeth retained normal mobility, healing PDL space and no resorption.

What is the role of CaOH2 in relanted teeth?

Andreasen 1981 JOE, Dent Trauma 2002 -

1. CaOH2 used too soon may diffuse through apex and damage PDL. Should be used after initial PDL healing (7 days) has progressed.
2. CaOH2 weakens dentin (Cvek) when placed for periods longer than 30 days.

Dumsha 1995 IEJ – NSD in resorption between avulsed teeth filled with GP or CaOH2 therefore in light of Cveks observation regarding weakening the root, perform RCT at 14-28 days and obdurate with GP.

Jeansonne 1994 EDT – delay pulpectomy after avulsion for 18 days to decrease chances of ankylosis w/out increasing risk of inflammatory resorption.

Trope 1995 EDT – 12 wk CaOH2 Tx for established inflam root resorption is better than short term (1 wk) treatment.

What is the role of fluoride ?

1. Shulman 1973 J Dent Res – showed decreased resorption of avulsed teeth using sodium fluoride.

2. Klinge 1989 – Soaking in SnF2 will delay remodeling of the root into bone (replacement resorption)

3. Coccia 1980 JOE – Treatment of the root with Fl before replanting makes it osteoclast resistant, delaying replacement resorption. Twice the survival time can be expected.

Would you recommend systemic antibiotics after replantation?

Antibiotic Recommendations: Use from initial visit until splint is removed at 7 days

1. Tetracycline 500mg QID x 7 days
    a. Static, acts on ribosomes
    b. Avoid if <9 years old or pregnant
2. Pen VK 500 mg QID x 7 days
    a. Use only if tetracycline is contraindicated

Trope 1998 EDT – 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.

Blomlof 1986 EDT – Use systemic antibiotic (amox) immediately after replantation to reduce inflammatory resorption. No effect seen on replacement resorption.

What is Sickle cell anemia, how does it effect the root canal?

Kaya 2004 IEJ – 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 1983** in sickle cell patients.

Radiographic observation- "stepladder" appearance of the widening trabeculation due to increased marrow space (increase hematopoiesis). This increased marrow space is accompanied by thinning cortical plate and irregularities in density.

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 writsts and ankles.

3. Dental: hypoplastic/hypocalcified enamel, draining sinus tract, ginigival swelling, apical abscesses

4. Radiographic: enlarged pulp chambers, wide root canals, and loss of lamina dura, rg band that surrounds region of DEJ.

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 renal disease, vit d deficiency, Ca malabsorption states
Treatment – renal dialysis or transplant

Classic signs = stones, bones groans

1. Ectopic calcifications – kidney stones
2. Bone lesions – lytic lesions (brown tumors, a giant cell granuloma)
    a. Ground glass appearance with decreased trabculation
3. Vague abdominal pain, fatigue, weakness
4. Emotional liability, psychoses

Discuss Diabetes –

Bender 2003 JOE – Inherent factors of the disease:

1. particularly prone to bacterial or opportunistic infections
2. vulnerability caused by a generalized circulatory disorder
3. blood vessels are damaged by the accumulation of atheromatous deposits
4. capillaries develop a thickened basement membrane
5. impaired leukotactic response capability
6. decreased PMN microbicidal ability
7. failure to deliver the humoral & cellular components of the humoral system.
8. Due to limited pulpal circulation, it is more prone to infection

Fouad et al 2003 JADA – Preoperative periradicular lesions and a history of diabetes have a significantly reduced risk for a successful outcome for endodontic treatment.

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. The peak incidence of healing or emerging chronic apical periodontitis occurred at 1 year. Complete healing of preoperative CAP in some instances required 4 years for completion.

Outcome Studies –

Initial treatment – NS RCT

Author	% Healed	# Cases	Follow-up period	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
Kerekes	91	501	3-5 years	1979
Swartz	88	1007	1 years	1983
Sjogren	91	356	8-10 years	1990
Smith	84	821	5 years	1993
Molven	86	265	20 years	2002

Outcome Studies –

Re-treatment

Author	% Healed	# Cases	Follow-up period	Date
Allen	73	1300	0.5 years	1989
Sundqvist	74	54	5 years	1998
Sjogren	98 without AP 62 with AP	356	8 years	1990

Outcome Studies –

Surgical Root Canal Treatment

Author	% Healed	# Cases	Follow-up period	Date
Dorn	95	488	6mos-10years	1990
Friedman	44	136	6mos-8 years	1991
Frank	58	104	15 years	1992
August	63	39	10 years	1996
Testori	85	302	6 years	1999
Rubinstein	92	59	5 years	2002

Toronto Study – Outcomes

Initial treatment – confirmation that preexisting apical periodontitis and treatment technique were the main predictors of outcome in initial treatments.

a. overall healed rate for Phase I & II was 85%
b. if based on only radiographic measure it would be 95%
    i. therefore, it should be noted that the absence of symptoms is insufficient as a measure of healing.
c. W/out preop AP healing rate reached 94%
d. With preop AP healing rate 79%
    i. 45% showed reduced size from original
e. 10% higher healing rate noted for those cases treated with flared canal preparations and vertical compaction of warm gutta-percha than step-back preparation and lateral compaction of gutta-percha
f. Other factors were noted but not statistically significant – tooth location, preop symptoms, perio condition, flare-ups, final restoration.

Toronto Study – Outcomes

Orthograde Retreatment – This study suggests that apical periodontitis, although a strong predictor, was secondary to preoperative perforation and root filling quality, and to postoperative restoration, in predicting the outcome of retreatment.

a. overall healed rate was 81%
b. 93% were asymptomatic and fully functional at the 4-6 yr f/u (radiographically not healed)
c. w/out preop AP healing rate 97% (elective procedure)
d. overall with preop AP healing rate 78%
e. with preop AP & w/out perf healing rate 86%
f. with preop AP & with perf healing rate 36%*
g. with preop AP & shorter than 2 mm fill 82%
    i. able to renegotiate short treatment in 74% of cases
h. with preop AP & adequate fill healing rate 67%
    i. these cases may be treatment resistant cases

*Trope suggests that 3 factors effect outcome of perf treatment

1. size, 2. locationl 3. time since perforation

Toronto Study – Outcomes

Surgical Retreatment – This study suggests that preoperative lesion size and root-filling length were significant predictors of outcome of apical surgery.

a. overall healing rate was 74%
b. 91% of the teeth were asymptomatic and functional
    i. despite radiographic findings
c. 60% demonstrated reduction of lesion size, & asymptomatic
d. if reduced lesion size were included in healing overall = 87%
e. healing rate for long or short fills = 86%*
f. healing rate for adequate fills = 68%
g. healing rate for lesion < 5mm = 86%
h. healing rate for lesion > 5 mm = 65%
i. healing rate w/ orthograde retx = 84% (NSD)**
j. Retrograde retreatment healing rate 94%

*Lustmann suggests etiology removed therefore healing

** Zuolo et al – 90% success for surgical cases previously retreated

What are some reasons for failure of non-surgical treatment?

The most common cause is previous treatment falls short of accepted standards. 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, microorganisms may persist and may play a role in treatment failures.

What are some reasons for failure of non-surgical treatment? Continued –

Extraradicular Infections – Actinomyces Israelii, Propionibacterium propionicum

1. Sjogren, Sundqvist et al 1988 IEJ – Propionibacterium propionicum may be implicated in Extraradicular infections

2. Nair 1984 JOE – Actinomyces israelii is able to establish Extraradicular infections.

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.

What are some reasons for failure of non-surgical treatment?

Foreign body reactions – of periapical tissue due to excess root canal filling material or food

1. Nair 1990 JOE – In the absence of microbial factors, root filling materials which contain irritating substances can evoke a forein 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:
    a. Black/brown fragments – amalgam
    b. Fine black/brown/yellow – sealer
    c. Basophilic fragments – CaOH
    d. 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? Continued

Cysts

1. Nair 1996 Oral Surg. – Histologic examination of apical lesions were identified proportionally to be:
    a. 50% granulomas
    b. 35% abscess
    c. 15% cysts
        i. 61% - true cysts
        ii. 39% - pockets cysts

2. Nair, Sjogren & Sundqvist 1993 IEJ – The accumulation of tissue break-down products such as cholesterol crystals, and the cystic 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.

Does the presence of a radiolucency affect the prognosis?

YES

1. Friedman – the presence of apical periodontitis decreases the success by 10-25%
2. Other studies finding a decreased success with radiolucencies:
    a. Strindberg
    b. Seltzer
    c. Storms
    d. Swart
    e. Matsumoto
    f. Sjogren
    g. Lin
    h. Smith
    i. Ackerblom
    j. Molven

Does a negative culture at the time of filling give a better prognosis?

YES

1. Sjogren, Sundqvist et al 1997 IEJ – 94% success with negative cultures, 68% success with positive cultures. Success appears to be dependant on eliminating bacteria from the root canal system before obturation. It is believed that this cannot be achieved in one-visit treatment because it is not possible to eradicate all infection from the root canal without the support of an inter-appointment antimicrobial dressing.

NO

1. Peters 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 cultivable bacteria were present or not prior to obturation.

Does a negative culture at the time of filling give a better prognosis? Continued –

2. Stromberg 1987 EDT – Healing occurs in apical periodontitis even if bacteria are present in the canal during obturation.

3. Matsumoto 1987 JOE – Multiple factors usually involve in failure, NSD noted between positive and negative cultures. Risk factors observed for treatment failure included:
    a. Periradicular radiolucency
    b. Overextension – greatest inflammatory response
    c. Deep perio pockets
    d. Occlusal trauma
    e. No adjacent teeth present

4. 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
    a. Underfill >2mm – 68% success
    b. 0-2mm from apex – 94% success
    c. overfilled – 76% success

2. Davis 1971 OOO – (dog study) Healing was least successful in overfilled teeth. Unfilled portion of the canal does not preclude healing.

3. Seltzer/Bender 1963 JADA – An overextended fill decreases success but underfilling had no influence.

4. Swartz 1983 JOE – Overall success 88%, overfilling decrease success, other risk factors for failure included: Preop AP, inadequate restorations

5. Molven 2002 IEJ – extruded material delayed healing, late periapical changes can occur more than 10 years after treatment.

Does the level of root canal filling affect success ? continued –

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 treatment more successful than two visits?

YES

1. Oliet 1983 JOE – Symptoms and healing 18 months after treatment indicated no significant difference between 1 or 2 visit treatment.
2. Pekruhn 1986 JOE – No greater failure rate for single vs multiple visit treatment. Retreatment had more failures.
3. Ashkenaz 1984 DCNA – Review article: findings
    a. No increase in post op pain in single visit treatment
    b. High level of success w/ single visit treatment
    c. Eliminates inter-appt. contamination potential (leakage)
    d. Disadvantage – emergency drainage complicated by filling

MAYBE –

1. Kvist 2004 JOE – from a micobiologic view, treatment of teeth with apical periodontitis performed in two appointments w/inter-appt dressing of $CaOH_2$ was not more effective than one –visit with 10 minute exposure to 5% IKI.

Compare post operative pain / flare-ups between 1 & 2 visit Tx.

1. Trope 1991 IEJ – Evaluation of specific preop conditions w/ flare-ups
    a. Single visit w/out AP – no flare-ups
    b. Single visit w/ AP – 1.4% flare-ups
    c. Single visit reTx w/ AP – 13.6% flare-ups – statistically the highest risk factor in the study.

2. Eleazer 1998 JOE – This study found a greater incidence of flare-ups in multi-visit treatments. (8% multi vs 3% single)

3. Roane 1983 OOO – This study found a 2 to 1 higher frequency of pain following treatment completed in multiple visits as compared to those completed in a single visit.

4. Southard 1984 JOE – One visit endo with I&D in patients with periapical abscess was not associated with high flare-up rate (0/19)

5. Johnson 2002 OOO – NSD in pain between 1 & 2 visit tx. Minimal PO pain w/in 24-48 hrs of treatment in both groups.

Does 2 visit treatment with CaOH2 increase the chances of healing?

YES

1. Sjogren 1997 IEJ – This study implys that healing would increase from 68% to 94% if accomplished in 2 visits with inter-visit medication was used (CaOH2).
2. Trope 1999 JOE – This study evaluated the healing of teeth with AP in one or two visits with or without CaOH2 as a intracanal medicament. Results indicated that CaOH2 increased the rate of healing by 10%.
3. Trope 2000 IEJ – Dog study – 1 wk CaOH2 in infected dog teeth increased healing after 6 months when compare to single visit tx.
4. Tanomaru 2002 JOE – Dog study – greater success with use of CHX than NaOCl, CaOH important for cases with CAP.
5. Holland 2003 JOE – CaOH2 helps achieve better results than single visit treatment in DOG teeth with AP.

Does 2 visit treatment with CaOH2 increase the chances of healing? Continued –

NO –

1. Weiger 2000 IEJ – This study had comparable success results with both single and multivisit treatment of pulpless teeth w/AP. (93% multi vs 92% single)

2. Peters 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 cultivable bacteria were present or not prior to obturation.

How often does the absence of a radiolucency correspond with histological success ?

1. Brynolf 1967 Odontol Revy – Histologic and radiographic exam indicated complete healing following RCT occurring only 7%. 93% had inflammation despite no radiolucency.

2. Walton 1997 OOO – Histologic and radiographic exam revealed 26% of specimines without radiolucencies had inflammation and 74% had no inflammation. **This study disputes Brynolf's findings !!!**

How long does it take to heal ?

1. Murphy 1991 OOO – Resolution of AP can occur as early as 3 months, average rate is 3.2mm/month. 70% of lesions require >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 IEJ – Recommends follow-up at 1 year. The peak incidence of healing or emerging chronic apical periodontitis occurred at 1 year. Complete healing of preoperative CAP in some instances required 4 years for completion.

What factors attect successful healing of a perforation ?

1. Jew 1982 OOO – Prognosis depends on time lapse since perforation, location relative to attachment, size and sealability of repair material. Best prognosis is in the apical or middle third of the root. Contamination with oral fluids leads to failure.
2. Seltzer 1970 J Dent Res. – Healing is dependtant on location and time lapse since perforation. Best prognosis is when perf is repaired immediately. The more attachment coronal to the perf the better.
3. Roane & Simon et al. 1986 JOE – Overall success for root perforation was 54%. Avoid extruding repair materials. Better success with amalgam than GP.
4. Oswald 1989 IEJ – Post-preparation caused perforations 53%. Success rate over 50%, Combined surg/orthograde approach best success.

Why not do SRCT instead of NSRCT retreatment?

Better success will occur with NSRCT due to the ability to determine and eliminate the etiology (missed canal, coronal leakage, incompletely debrided canal system…)

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 rot 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 not do SRCT instead of NSRCT retreatment? Continued

5. Newton 1989 JOE – Retrospective study of 1200 cases:
    a. Overall success for retx 65.6%
    b. Surgical retx – 60%
    c. NS Retx – 73%
    d. ReTx of prior ReTx – 47%

Take home message of Newton's study – Try retx first but if unsuccessful next step is surgery.

Does a separated instrument affect prognosis ?

Depends on when during treatment it occurs, location of separation, status of pulp and periapex.

1. Crump 1970 JADA – No statistical difference between cases with separated instruments (81%) and control cases w/out separated instruments (73%) ????? Something seems wrong with this data !!!!!!!!

2. Strindberg 1956 AOS – Separated instruments decreased success by 14%

What is the prognosis for formocresol pulpotomy in primary teeth?

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.

3. Waterhouse 1995 EDT – This study reviewed the success rates for pulpotomies in primary teeth with various medicaments.

    a. Formocresol 55-98% - cytotoxic, mustgenic, carcinogenic
    b. CaOH2 31-100%
    c. Glutaraldehye 82-98%

What is the prognosis after vital pulp therapy in young permanent molars? For apexogenesis

1. Mass 1993 Dent – Partial pulpotomy in young permanent molars w/carious exposures had a 91.4% success rate.

2. Holland 2001 Dent Trauma. – This study compared the success of pulp capping of dog teeth to CaOH or MTA. Significantly better results appear with the use of MTA. Healing w/MTA showed complete tubular dentin bridge formation and no inflammation in any of the pulps capped with MTA. Mechanism of action believed to be similar to CaOH2 – Calcite crystals attract fibronectic, which is responsible for cellular adhesion and differentiation. MTA provided a superior bacteria-tight seal.

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:
    a. 44.5% failures, 5 yrs // 79.7% failures, 10 yrs
    b. 18.5% questionable, 5 yrs // 7.3% questionable, 10 yrs
    c. 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)
    a. Case selection – no clinical or radiographic signs of pulpitis
    b. Technique –
        i. Gentile
        ii. No interference of blood clot between pulp and material
        iii. Do not introduce infected dentin chips or material into pulp

Discuss the prognosis of direct pulp cap tx for carious exposures. Continued

3. 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.
4. Stanley 1978 JADA – Direct pulp capping with CaOH2 powder ro PCN crystals was successful after 11.7 years in 87.3% of 356 cases, with major failures occurring after 1.65 yrs on average.
5. Tronstad 1972 OOO – Direct pulp capping of carious pulps had less than a 50% chance of success. It should be considered IR and RCT provided.
6. Hebling 1999 JOE – Studies histologic events during pulp cap
    a. 7 days – CaOH exhibited odontoblast-like cells organized underneath coagulation necrosis.
    b. 60 days – repair evolved into apparent complete dentin bridge
    c. All Bond 2 did not appear to allow any pulp repair

What are the properties of the dentin bridge formed?

1. Pisanti 1964 Jdent Res. – Calcium in the newly formed secondary dentin comes from the blood stream and not from the CaOH2 base.

2. Holland 1982 JOE – Dog study – This study looked at the pulpal reaction to Ca, Barium and Strontium hydroxides. All had similar reactions, w/necrotic zone, precipitation of large granules and tiny granuoles. **Inference is that source of Ca is from capping material.** Conflicts with Pisanti's study.

3. Goldberg 1984 JOE – Dentinal bridge formed with CaOH2 is porous and permeable.

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 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. Jordan 1978 JADA - <50% resolution w/indirect pulp caps on permanent teeth w/periapical lesions. Confirms Yamasaki's theory that AP can occur on vital cases. Radiographs showed that healing can occur after indirect pulp cap.

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 successor.