## A Guide to the Endodontic Literature

### Success & Failure:

<table>
<thead>
<tr>
<th>Authors</th>
<th>Description</th>
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<tbody>
<tr>
<td>European Soc. Endodontology (1994 IEJ)</td>
<td><strong>Definition of Success</strong>: Clinical symptoms originating from an endodontically-induced apical periodontitis should neither persist nor develop after RCT and the contours of the PDL space around the root should radiographically be normal.</td>
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<table>
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<tr>
<th>AAE Quality Assurance Guidelines</th>
<th><strong>Objectives of NSRCT (nonsurgical root canal treatment)</strong></th>
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<tr>
<td></td>
<td>• Prevent adverse signs or symptoms</td>
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<td></td>
<td>• Remove RC contents</td>
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<td></td>
<td>• Create radiographic appearance of well obturated RC system</td>
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<tr>
<td></td>
<td>• Promote healing and repair of periradicular tissues</td>
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<tr>
<td></td>
<td>• Prevent further breakdown of periradicular tissues</td>
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### The Mantra:

- Apical periodontitis (AP; periapical radiolucency = PARL) is caused primarily by bacteria in RC systems (Sundqvist 1976; Kakehashi 1965; Moller 1981)
- If bacteria in canal systems are reduced to levels that are not detected by culturing, then high success rates are observed (Bystrom 1987; Sjogren 1997)
- Best documented results for canal disinfection are chemomechanical debridement with Ca(OH)2 for at least 1 week (Sjogren 1991)
- Mechanical instrumentation alone (C&S) reduces bacteria by 100-1,000 fold. But only 20-43% of cases show complete elimination (Bystrom 1981; Bystrom & Sundqvist 1985)
- Do C&S and add 0.5% NaOCl produces complete disinfection in 40-60% of cases (Bystrom 1983)
- Do C&S with 0.5% NaOCl and add one week Ca(OH)2: get complete disinfection in 90-100% of cases (Bystrom 1985; Sjogren 1991).

### Problems with the Mantra

- Koch’s postulates cannot be applied to establishing a bacterial origin of AP (since polymicrobial – Baumgartner)
- Mantra misses host response contributions (eg: Stashenko’s P/E selectin knockout mice actually showed increased AP due to bacteria (thus, phagocytic leukocytes help to minimize AP via protection against microorganisms: implies host defenses regulate the development of AP)
- What is the clinical significance of a “non-cultivable” RC sample when organisms can reproduce in <12h?
- Implication: the “mantra” is focused on what the clinician can accomplish with current methods (eg., reduction-disruption of a bacterial ecosystem). It only provides general guidance for developing better therapeutical methods, and it cannot predict clinical success in cases where immunocompetence is altered.
- Given a polymicrobial etiology and a disease-modifying host capacity, it is (probably) overly simplistic to correlate one bug with given signs or symptoms. [Recall Sundqvist (1992) used odds ratio analysis & concluded that bacterial pairings in infected RC systems are not random, but appear to be due to forces such as ecological commensalism. Since pairings can occur, correlational analysis between bugs and signs-symptoms may be confounded if one bug is more easily cultivable than another]

### Penick, 1961

- NSRCT with GP. Still saw PARL at 14 months. Sx biopsy revealed healing by scar (no inflammation). THL - consider healing by scar when reviewing post-endo tx (and sx work-ups)

### Brynolf 1967

- This study was performed on human cadavers with X-rays taken of 320 upper incisors. Even though many radiographs appeared normal, complete histological healing after NSRCT occurred in only 7% of cases. Thus, radiographic success doesn't correlate with histological success
<table>
<thead>
<tr>
<th>Ref</th>
<th>Details</th>
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<tbody>
<tr>
<td>Green, Walton, 1997</td>
<td>Compared radiographic findings of NSRCT to histological exam of human cadavers. 74% of the teeth with normal radiographic findings showed NO inflammation. 26% with a normal periapex radiographically showed histologic signs of inflammation. The results of this study do not agree with those by Brynolf in 1967 who found inflammation in the majority of the teeth that had received root canal treatment.</td>
</tr>
<tr>
<td>Ingle, Beveridge</td>
<td>This study was done to evaluate treated endodontic cases and determine their rate of success. 33.41% of 3,678 patients returned for recall. 94.45% rate of success. The greatest cause of failure was interpreted to be obturation (but it may also be poor C&amp;S).</td>
</tr>
<tr>
<td>Kerekes, Tronstad 1979</td>
<td>Examined 333 patients treated by undergraduate students. Hand instrumentation with reamers and Hedstrom files was performed. EDTA and 5% chloramine-T was used for irrigation. Lat condensation with gutta percha points coated with Kloroperka N-O. Roots without periradicular radiolucencies prior to treatment showed better results than those with radiolucencies. No difference in success between vital and necrotic pulps, or in teeth with flare-ups during tx. Adequate seal and the apical level of the root filling were significant factors for the success of tx.</td>
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<tr>
<td>Bergenholtz 1974</td>
<td>Retrospective study of 84 teeth with trauma and intact crowns and necrotic pulps. 64% had microorganisms present (primarily polymicrobial anearobic).</td>
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<tr>
<td>Akerblom, Hasselgren 1988</td>
<td>Teeth with periapical radiolucencies had lower healing rates than those without a lesion. In teeth lacking lesions, 97.9% were judged successful. In the presence of a pre-operative lesion, only 62.5% teeth were deemed a success. 2-12 yr follow-up.</td>
</tr>
<tr>
<td>Ray &amp; Trope , 1995</td>
<td>Radiographic exam of 1010 endodontically treated teeth restored with a permanent restoration. The quality of the coronal restoration was significantly more important than the quality of the endodontic treatment for the presence of apical periodontitis.</td>
</tr>
<tr>
<td>Augsburger, Peters 1990</td>
<td>Radiographic evaluation of resorption of ZOE sealer/gutta-percha extruded into periradicular tissues. The rate of disappearance of the material did not differ with the presence or absence of radiolucent lesions, type of ZOE sealer used, or obturation technique. In no case did an irreversible lesion develop where sealer was expressed. Extruded material did not prevent radiographic repair of radiolucent lesions.</td>
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### 1-Step vs Multi-Step: Short-term Comparison

- **Pekruhn (1981):** Compared postoperative pain after single-visit and multiple-visit NSRCT. 1 shot = multi-appt (both had 16% popln with pain at 1day)
- **Oliet (1983):** Compared 1 step to multi-appt NSRCT (n=380). When pain occurred post-op, it typically presented within the first 24 hours; there was no difference between 1 shot vs multi-appt, or for vital vs necrotic cases. Also, no difference in healing at 18 months. A difference in healing was observed when comparing the quality of the obturation in single visit treated teeth. Teeth that were overfilled showed less healing than those filled to or just short of the radiographic apex.
- **Roane, Dryden & Grimes (1983):** Compared 1 step to multi-step NSRCT (n=300). No differences in pain different anatomic groupings or pulp status (necrotic vs vital). Pain after 1-step was about one-half of pain after multi-appt NSRCT
- **Mulhern and Patterson (1982):** 1 step NSRCTs does not increase post-op pain
- **Southard & Rooney (1984):** The article strongly supports the position that 1-step NSRCT is an acceptable method to treat an abscessed tooth. 0 of 19 patients had exacerbations of swelling or pain following treatment. 63% of pts with AAA were contacted 24hrs post-NSRCT with IND, and all reported no or reduced pain. Complete resolution of swelling resolved in 3-7 days. 58% of pts returned at 1 year and all were asymptomatic and showed radiographic signs of healing.
- **Eleazor & Eleazor (1998):** Retrospective study: Flare-ups: 1 step (3%) < 2-step (8%; p<.01). n=201 consecutive necrotic 1st & 2nd molars tx with 1-step had 3% flare-up vs n=201 consecutive necrotic 1st & 2nd molars tx with 2-visit (med= metacresylacetate) had 8% flare-up (p<.01)
<table>
<thead>
<tr>
<th>Author(s)</th>
<th>Study Details</th>
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| Friedman 95, Sjogren 97 | • Pekruhn (1986): Evaluated failure with 1 steps (n=925 @ 1 yr). The overall failure rate was 5.2%. Most of the failures had preexisting apical periodontitis. 18% of these had symptoms. Retreatment cases had the highest rate of failure at 16.6%. The teeth tx with 1-step showed 3 times the failure rate as those previously opened for emergency treatment. The higher failure rates of those teeth presenting with apical periodontitis may serve as contraindication for 1-step NSRCT.  
• Bystrom & Sundqvist (1981): One steps do not remove bacteria in necrotic cases. Ca(OH)2 is the best inter-appt medicament to kill residual bacteria. Simple mechanical debridement with saline is insufficient to remove all bacteria (although it does reduce bugs by 100-1,000 fold).  
• Sjogren (1997): Teeth with negative bacterial cultures prior to fill had 94% success rate whereas teeth with positive cultures had 68% success rate. Also demonstrated that could not reliably obtain negative cultures after just one appt. Others have also reported a simliar increase in prognosis when obturate canals with negative cultures: Engstrom (1964) and Oliet (1969).  
• Friedman & Trope (1995 JOE p386): n=378 eval Ketac-Endo for NSRCT. Multi-appt NSRCTs with Ca(OH)2 medicament tended (86% vs 76%; p=NS) to have better success and fewer failures than one-shots. 6-18m follow-up  
• Trope & Orstavik (1999 JOE): Randomized clinical trial evaluating 1 step vs 2step with or without Ca(OH)2 with 1yr follow-up. Ca(OH)2 had 74% healing > 1-step (64%; NS difference) > 2-step with no med (54% healing)  
• Katebzadeh & Trope (1999 JOE ): Dog study infected teeth with AP with 6m follow-up: C&S to size 45; 1 week Ca(OH)2 med gave better PA healing after 6m than 1-step with LC Roths. 1-step was better than no NSRCT (= open canals = positive control)  
• Weiger, Axman-Krcmar & Lost (1998 EDT): One-steps tended (p=0.13) to produce poorer healing than multi-steps using Ca(OH)2 over 18 month period. Used Cox regression analysis of raw data from Lost et al (1995; n=76): analysis showed that that one-steps tended (p=0.13) to produce poorer healing than multi-steps using Ca(OH)2 over 18 month period |
| Studies justifying 1 year Recall: | • Reit (1987): Best recall is at one year. Also rec recalls annually for minimum of 4 years (esp in questionable cases)  
• Rud & Andreasen (1972): If PARL healed at 1 year, then ok  
• Orstavik (1996): ~76% of apical periodontitis lesions developing post-tx are seen within 1 year. Therefore, 1yr follow-up predicts long-term success  
| Friedman 1998 Chap in Essential Endodontology by Pitt Ford & Orstavik | Meta-analysis of prior success-failure studies. For NSRCT: Apical periodontitis success rate is 10-25% lower than NSRCT performed in teeth with normal periradicular tissue (=83-100%). NSRCT Re-tx of teeth with AP = 56-84% healing.  
Reviewed 27 studies (from Strindberg 1956 to Ostravik 1996): 78% of studies demonstrated >10% reduction in success.  
| Orstavik 1986 | Proposed use of PAI (periapical index) to evaluate radiographic success by comparison to 5 standard images (healthy = 1; bad=2-5).  

Davis & Joseph 1971 **Classic!** Teeth that were fully instrumented, but filled short of the radiographic apex had best healing. ALSO: Seltzer & Bender 1963 & 67 (human and monkey study with healing eval at 3 months; overfill = persistent inflammation)  

Sjogren 1990 **CRITICAL STUDY.** Necrotic teeth without AP have 96% success, but necrotic with AP have only 86% success. Best success tx necrotic cases with apical periodontitis are when the obturation ends within 0-2 mm of radiographic apex = 94%); underfills are less successful (68% when filled > 2mm from apex) and overfills are less successful (76%). Also, re-tx of teeth with AP have low success (62%). Results are similar to Davis & Joseph (1971).  

**Causes for failure of NSRCT:** (see also: "Differential Dx of PARLs")

*If Dx is correct, bacterial infection is primary cause [Lin & Pascon (1991); Cheung (1996)].*

- **“POOR PAST”** (Crump 1979)  
P--perforation; O--obturation; O--overfill; R--root canal missed; P--periodontal disease; A--another tooth; S--split; T--trauma  
- Persistent Intraradicular infection (Nair 1990)
San Antonio Guide to the Endodontic Literature

- Sjogren (1997) reduced success when bacteria are present during obturation (94% vs 68%)
- Pitt Ford (1982) infected dentinal tubules
- Orstavik (1990): E. faecalis & Strep sanguis grew 300-400um into dentinal tubules after 14-21 days
- Enterococcus faecalis in 33% failed NSRCTs (Molander 1998 IEJ) & in 60% failed cases reported by Siren (1997)
- Actinomyces israelii found in two case reports of failed NSRCT. Had to be eliminated by Sx (Sundqvist 1981 OOO)

- Persistent Extraradicular infection. see Simon's review on POP for general info and nice figs
- Nair (1984) Actinomyces israelii. Also reported by Happonen (1986): 81% samples contained actinomyces, 62% contained arachnica
- Sjogren (1988) Propionibacterium propionicum (aka Arachnia propionica)
- Wayman (1992) evaluated 58 NSRCT failures in lesions with NO oral communication, 83% had bugs in lesion! (93% had bugs in lesions with oral communication). Similar to Iwu (1990) report of 88% lesions having cultivable bugs.
- Kirye (1994): found infected cementum. Also Tronstad (1990) reported bacterial plaque over apical foramen
- Holland (1980): infected dentinal chips expressed into periapex. Also reported by Yusuf (1982)
- Foreign body reaction (Nair 1990). Small particles of GP are extremely inflammatory [Sjogren (1995)]
- Cysts, esp true cysts (Nair 1993, 1996).

Grun 1990
Success of re-tx combined with endo sx is 24% higher than endo sx alone

Specialist vs Generalist
As defined ONLY by radiographs, success of NSRCTs is 83-94% (Grahnen 1961; Ingle 1985) in clinical trials and 61-77% (de Cleen 1993; Erckerborn 1989) in epidemiologic studies. The clinical trials represent optimal tx by specialists or well-supervised students, whereas the epidemiologic studies represent general practice. (From Ericksen in Essential Endodontology 1998).

Lavstedt 1978
(in Norwegian) Teeth with greatest prevalence for apical periodontitis are max laterals, max 1st premolars and mand 1st molars.

Weiger, Axman-Kcmar and Lost EDT 14:1, 1998
Reviewed predictors of success of NSRCT from statistical perspective. Based on metanalysis, probability of PARL healing after NSRCT within 3yr is 0.87-0.89. Used Cox regression analysis of raw data from Lost et al (1995; n=76): analysis showed that one-steps tended (p=0.13) to produce poorer healing than multi-steps using Ca(OH)2

<table>
<thead>
<tr>
<th>Success (%)</th>
<th>No PARL</th>
<th>PARL</th>
<th>N</th>
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<tbody>
<tr>
<td>1. Molvern &amp; Halse (1988)</td>
<td>91%</td>
<td>68%</td>
<td>207</td>
</tr>
<tr>
<td>2. Akerblom, Hasselgren (1988)</td>
<td>98%</td>
<td>62%</td>
<td>64</td>
</tr>
<tr>
<td>3. Sjogren (1990)</td>
<td>96%</td>
<td>86%</td>
<td>471</td>
</tr>
<tr>
<td>4. Friedman (1995)</td>
<td>93%</td>
<td>69%</td>
<td>142</td>
</tr>
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The NSRCT success rate for necrotic teeth vs vital appears equivocal
Smith (1993) reports reduced success with necrotic cases
Kerekes & Tronstad (1979) reports same success
Strindberg (1956) reports increased success with necrotic cases

Success of Re-Tx:
- No PARL: 89-100%
- PARL: 56-71%
- Bergenholtz (1979 Scan JDR): Classic on re-tx. Group being re-tx for prosth indication (ie, not failing) s till had 6% failure rate
• Allen (1989 JOE): Classic: Retrospective study of 1,300 cases. 65% success 16% uncertain. NSRCT Re-tx better success than sx (73% vs 57%).
• Sjogren (1990): re-tx teeth with AP has 62% success rate
• Briggs & Scott (1997): Re-tx is preferable over endo sx (“evidence based” analysis).
• Moiseiwitsch & Trope (1998) Re-tx is preferable over endo sx

Success of Surgical Endo:
- Apical Sx: 59%
- Re-Tx + Apical Sx: 80%
- Source: Friedman’s analysis in Essential Endo. (nice initial meta-analysis approach).
- Dorn & Gartner (1990 JOE): Retrospective study in two endo offices (non-randomized, etc): Success Super EBA 95%; IRM 91% and amalgam 75%

- Rubenstein & Kim (1999 JOE): CRITICAL: Using scope, ultrasonics and Super EBA: n=94 cases (2/3 posterior & 1/3 anterior): 97% radiographic success at 3-12m follow-up with mean healing of 7.2m (criteria = restoration of lamina dura). 85% granuloma and 15% cysts with no difference in time to heal. Isthmuses were found in 25% of the cases.
- Testori (OOO 1999): n=302 apices (181 teeth) with 5yr follow-up standardized radiographs with 2 observers: 85% complete healing with ultrasonic tips and super-EBA at 4.6yr versus 68% complete healing for rotary microhandpiece with amalgam. Saw reduced success when had poor or no prior NSRCT (see Danin below)
- Danin (1999 OOO): Did endo sx in necrotic cases without any NSRCT. 50% mod-complete success at 1yr (but used bur and glass ionomer for endo sx). But- 90% of these cases had cultivable bacteria in canals. Important point: cases may show radiographic success after sx even with bacteria in canals.
- Bradford (1999 OOO): defines sx success as 1) absence of symptoms; 2) absence of swelling, sinus tract, signs of infection; 3) radiographic evidence of healing; 4) continued normal functioning of the tooth. Summarized qualities of an ideal root-end filling material: biocompatibility, apical sealability and handling characteristics
- Lin (1996 IEJ): Discussed periradicular curettage. Remove for visibility. NEED NOT REMOVE ALL GRANULATION TISSUE FOR HEALING TO TAKE PLACE!

Moiseiwitsch & Trope (1998 OOO): Sx should not be considered primary tx when non-surgical re-tx (or even NSRCT for first time) can be done. ALSO: Briggs & Scott (1997): meta-analysis

Success in Intentional Reimplantation:
2. Keller (1990): 91% success
- Koenig (1988): n=192 Keep out of socket<15min, do not touch root, keep it moist, minimal splinting
- Dumsha & Gutmann (Compendium 6/95): reviewed clinical guidelines

Success with Separated instruments
- Strindberg (1956): found 19% higher incidence of failure with separated instruments
- Crump & Natkin (1970): No difference in failure rates with separated instruments. Location of instrument is important
- Tamse & Katz (1987 IEJ): Proposed using separated files to obturate a canal. Consider this tx only after all other techniques have been evaluated as impossible
Sjogren & Sundqvist 1997

Teeth with negative bacterial cultures prior to fill had 94% success rate whereas teeth with positive cultures had 68% success rate (p<.05). Also demonstrated that could not reliably obtain negative cultures after just one appt (only 40% cases were non-cultivable for bugs). In 3 of the failing cases, Actinomyces was found in the RC systems. Study was on 55 root canals with 5 yr follow-up. Most important point: The success rate of NSRCT is 26% higher if the RC system is free of bacteria at time of obturation.

Eriksen 1991

Prevalence of apical periodontitis increases with age.

Sundqvist 1976

CLASSIC: Apical periodontitis can only be detected in teeth with bacteria present in canal systems. Necrotic, but sterile traumatized teeth have no signs of PARL. In contrast, necrotic and infected teeth showed PARLs. Also, probability of pain increased with # bacterial species (esp when >6); suggests bacterial synergism is important virulence factor.

<table>
<thead>
<tr>
<th>History and Rationale</th>
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<tbody>
<tr>
<td>Hudson 1862</td>
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<tr>
<td>Price 1901</td>
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<tr>
<td>Callahan 1914</td>
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<tr>
<td>Hatton 1922</td>
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<tr>
<td>Blayney 1930</td>
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</table>
| Milas in: POP 1980 | • Harry B. Johnston - first endodontic practice was begun in 1928.  
• 1943 the AAE was formed in Chicago  
• 1963 the ADA recognized endodontics as a special area of dentistry.  
• Karl Koller introduced cocaine in 1884  
• Alfred Einhorn introduced Novocaine in 1905  
• Wilhelm Roentgen discovered x-rays and in 1896 the first dental apparatus was built by Rollins  
• Hall patented (1847) gutta-percha as canal filling material (was named "Hall's Stopping").  
• Elmer Jasper in 1930 discussed the use of silver points.  
• The rubber dam was first used in 1862 and 20 years later the first set of retainers were born  
• Bowman and Allen in 1873 developed the the rubber dam forceps  
• Coolidge 1919 Introduced NaOCl to endodontics  
• Nygard-Ostby 1957 Introduced EDTA to Endodontics  
• Hermann 1920 - introduced Ca(OH)2 as intracanal medicament for necrotic teeth |
Rickert & Dixon 1931 Implant materials and hollow needle in rabbits. The authors believed that when the tissue of the pulp has been destroyed, it must be filled to the very end in order to prevent “diffusion”. Therefore the filling material must come in contact with the surrounding vital tissue. This filling material must be tolerated by the tissue (biocompatible). Th authors also concluded that hollow tubes were not tolerated by the body and therefore, a root canal can not be filled short of the apex. = “hollow tube” theory - the idea that the body cannot tolerate an underfilled canal. DISPROVED BY: Torneck (1967) CLASSIC: Disproved the hollow tube theory with implanting sterile hollow needles and demonstrating minimal tissue response

Focal Infection & Systemic Responses to Oral Infection

Newman 1996 To re-present the idea that the human mouth is a focus of infection (originally proposed by W.D. Miller in 1890)

Fish 1939 Zones of Fish = early attempt to disprove focal infection theory
- Zone of infection (innermost zone which is necrotic and contains bacteria; center of abscess)
- Zone of contamination (cell destruction is evident; abscess wall; exudative)
- Zone of irritation (contains osteoclasts and histiocytes; granulomatous zone)
- Zone of stimulation (encapsulation)

Conclusion - Cotton wool + bugs implanted into guinea pig mandibles 4-40 days. Infection remained localized regardless of the duration or virulence of the organism.

Kawashima & Stashenko (1998 Immunnology) Used P/E selectin knockout mice (P/E ko’s lack rolling adhesion of PMNs and macrophages to endothelium): Saw significantly more PA bone destruction in ko’s. Thus, phagocytic leukocytes (PMNs and/or macrophages) protect against bacterial induced PA bone destruction in mouse model of AP.

Darveau Infect Immum 63:1311, 1995 Possible mech for oral bacteria (P. gingivalis) to influence distant sites of infection: LPS (only from oral bugs) down-regulates E-selectin expression in vascular endothelium. Get reduction in leukocyte diapedesis at distant sites of infection. “E selectin inhibition by bacterial LPS could explain the relative lack of inflammation and pain associated with periodontal pockets and root canals that harbor large numbers of bacteria” (quote from Bergman, below). Proposed that this is a possible mechanism of focal infection.

Bergman, Trope & Offenbacher 1999 JOE p747 Mouse model: Implanted two chambers sc in the R & L flanks: one contained E. coli (model of enteric infection) and the other contained either P. gingivalis (oral infection model) or Sham/Sham. Chronic administration of Pg delayed the time for 50% rejection (ie, sloughing) of Ec chambers (25 vs 19 days). Importantly, the reverse was not true (ie, Ec did not delay time for Pg rejection). Thus, oral microorganisms may alter infection at distant sites. Possibly due to Darveau mechanism of E selectin suppression.

Grau 1997 Stroke28:1724 Epidemiologic study which demonstrates positive correlation between endodontic infections and the incidence of stroke. “Raises new concerns regarding the role of untreated periapical infection”

Nair in: Essential Endodontology The concept of focal infection is built around the pathological effects of bacteremia. However, the significance of this proposal is weakened by the observation that bacteremia is found in healthy patients undergoing routine toothbrushing or flossing without adverse effects [see also: Baumgartner ’77; Hockett ’77 Arch Oral Biol].

Root Canal Anatomy
Vertucci 1984

Other anatomical studies: Bellizzi 1983 / 85

<table>
<thead>
<tr>
<th>Teeth Type</th>
<th>Number of Teeth</th>
<th>Canal Type Distribution</th>
<th>Maxillary Teeth</th>
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<tbody>
<tr>
<td>1st Premolar</td>
<td>62% Type IV (2 canals), 18% Type II (2-1 canals), 69% have 2 canals at apex</td>
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<tr>
<td>2nd Premolar</td>
<td>48% Type I (1 canal), 22% Type II (2-1 canals), 11% Type IV (2 canals)</td>
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<tr>
<td>1st Molar: MB</td>
<td>45% Type I (1 canal), 37% Type II (2-1 canals), 18% have 2 canals at apex</td>
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Mandibular Teeth:

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<thead>
<tr>
<th>Teeth Type</th>
<th>Number of Teeth</th>
<th>Canal Type Distribution</th>
</tr>
</thead>
<tbody>
<tr>
<td>Central</td>
<td>70% Type I (1 canal), 22% Type III (1-2-1 canals)</td>
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</tr>
<tr>
<td>Lateral</td>
<td>75% Type I (1 canal), 18% Type III (1-2-1 canals)</td>
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<tr>
<td>Canine</td>
<td>78% Type I (1 canal)</td>
<td></td>
</tr>
<tr>
<td>1st Premolar</td>
<td>70% Type I (1 canal)</td>
<td></td>
</tr>
<tr>
<td>2nd Premolar</td>
<td>98% Type I (1 canal)</td>
<td></td>
</tr>
<tr>
<td>1st Molar: M</td>
<td>12% Type I (1 canal), 22% Type III (1-2-1 canals), 43% Type IV (2 canals)</td>
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<tr>
<td>1st Molar: Distal</td>
<td>70% Type I (1 canal), 15% Type II (2-1 canals), 8% Type V (1-2 canals)</td>
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Rationale for Instrumenting 0.5-1mm short of the radiographic apex:

Morphological Studies:
- Kuttler (1955): Examined apices of extracted teeth distance from DCJ to radiographic apex 0.5mm (young pts) to 0.65mm (older pts).
- Burch & Hulen (1972): found apical foramen 0.59 mm short of radiographic apex
- Tamse & Littner (1988): apical foramen was positioned 0.8mm from the tip of the root
- Stein & Corcoran (1990): found apical foramen 0.72 mm short of radiographic apex and width of CDJ = 0.19mm
- BUT- Gani & Visvisian (1999 JOE): studied apical canal diameter in max 1st molars. At 2mm from apex, palatal systems are 60% circular and 30% ovoid regardless of age. At 2mm from apex, MB systems are 50-60% flat (ie, ribbon, tear-shaped) and 30% ovoid (no clear cut age effect). Interestingly, DB systems 30-60 circular. Problem is that if C&S in flat canal system for the long dimension, could perf in narrow dimension during instrumentation

Pulp – PA Pathology Studies:
- Malueg, Wilcox & Johnson (1996): SEM of teeth with varying external apical root resorption (n= 40). Apical resorption: pulpal necrosis > normal pulp, reversible pulpitis, or irreversible pulpitis. Teeth with periapical lesions had significantly more apical resorption than those without radiographically evident periapical lesions. Therefore, the status of the pulp and periapical tissues should be considered when determining length for preparation and obturation.
- Frank (1990) Also reported this finding (ie, necrotic teeth tend show more apical resorption).
- Trope & Chivian (1994) propose that CDJ at foramen is very thin (in some cases, absent) – exposing mineralized dentin to the resorptive clastic cells.

Outcome Studies:
- Sjogren (1990): Outcomes study: Best success for tx necrotic cases with apical periodontitis are when the obturation ends within 0-2 mm of radiographic apex (= 94%); underfills are less successful (68% when filled > 2mm from apex) and overfills are less successful (76%).
- Davis & Joseph (1971): Classic! Teeth that were fully instrumented, but filled short of the radiographic apex had best healing. ALSO: Seltzer & Bender 1963 &67 (human and monkey study with healing eval at 3 months; overfill = persistent inflammation)
- Ricucci (1998 IEJ): Review article and 100 case report series. Conclude that best results is to obturate at apical constriction which ranges 0.5-2mm short of radiographic apex.

Mandibular Incisors
- Benjamin & Dowson (1974): This radiographic study places the incidence of 2 canals in mandibular incisors at 41%, generally merging in the apical area. This value is higher than Vertucci's study (= 18-22%).
• Vertuci (1984): 70-75% 1 canal and 18-22% 2 canals
• Mauger, Schindler & Walker (1998): Determine the prevalence of two canals and an isthmus in mandibular incisors. An isthmus was present in 20% of the teeth at the 1mm level, 30% at 2mm, and 55% at 3mm. The width measurements indicate that a final apical prep size should > #35 file to debride most mand incisors. An isthmus may make it difficult to debride with rotary instruments alone without the risk of perforation proximally. Note that Benjamin & Dowson (1974) reported 41% incidence of 2 canals, but they used 2 files and did not section to look for isthmuses.
• Miyashita (1997) evaluated 1,085 mand incisors and recommended #40 MAF. 85% single canals with 99% foramina within 1mm.

Mandibular Molars
• Cooke & Cox (1979): Mandibular 2nd & 3rd molars can have "C" shape 8% of the time. MB joins D canal; can be difficult to debride and shape.
• Skidmore & Bjorndal (1971): When the mesial root of mand 1st molars contain 2 canals, they are 40% Weine type II (2 canals, 1 foramina) and 60% Weine type III (2 canals 2 foramina). When the distal root contains 2 canals (29% of the total) they can be classified as Weine type II 60% and Weine type III 40% of the time.
• Vertucci (1984): 1st Molar: M 12% Type I (1 canal), 22% Type III (1-2-1 canals), 43% Type IV (2 canals)
• Vertucci (1984): 1st Molar: Distal 70% Type I (1 canal), 15% Type II (2-1 canals), 8% Type V (1-2 canals)
• Reeh (1998 JOE): Reports 7 canal mand first molar MB1&2, ML1&2, DB, D, DL. Used Ca(OH)2 sealer for D canals due to large apical openings to reduce chance of sealer extrusion due to rapid setting time…..

Maxillary Premolars
• Carns & Skidmore (1973): Most important point: 85% max 1st premolars have 2 canals. Max first premolars showed five different morphologic categories of combinations of roots, canals, and foramina; (%); 2,2,2 (57%); 1,2,2 (15%); 1,2,1 (13%); 1,1,1 (9%); and 3,3,3 (6%). Remember to look for wider M-D width at CEJ as a predictor of a 3 canal premolar.
• Vertucci (1984): Max 1st Premolar: 69% have 2 canals at apex (Bellizzi (1985): 90% have 2 canals
• Vertucci (1984): Max 2nd Premolar: 82% have 1 canal at apex (Bellizzi (1985): 59% have 2 canals!

Maxillary Molars
• Kulild & Peters (1990): Max Molars: the incidence of 2nd canals in MB roots of 1st and 2nd molars is ~ 95% and this 2nd canal originates 1.82mm lingual to the MB canal.
• Gilles, Reader (1990): Found 90% Max 1st molars have MB2 and 70% max 2nd molars. ML canals exit the root an average of 2mm short of the anatomic apex in first molars and 1.45mm in second molars.
• Fogel and Peikoff (1994): Examined 208 Max 1st molars MB root: 29% Type I (1 canal), 39% Type II (2-1 canals), 31% Type III (1-2-1 canals). THEREFORE 71% OF MAX 1ST MOLARS HAVE TREATABLE MB2! This value splits the difference between Weine (50% incidence of MB2; CLASSIC: 1969 study) and Kulild & Peters (95% when sectioned tooth; 1990 study)
• Bone & Moule (1986): This study shows that the palatal root of the maxillary molar should always be assumed to curve. 85% of examined palatal roots displayed curvature > 10°. We need to bear this in mind when performing root canal therapy and when creating post space.

Hartwell & Bellizzi 1982
In vivo incidence of 4 canal cases (assessed by post-obturation film) is much lower than in vitro anatomical studies. For example, max 1st molar, only 18% had 4 canal systems obturated.

Stropko (1999 JOE)
Confirmed Hartwell & Bellizzi in cases series report: increasing #s MB2 was found with microscopic exam. Also: 1) make access more rhomboid, infringed MMR to access mesially inclined MB2. To test for MB1-MB2 communication, place paper point in MB2 and watch fluid level in MB1. Usually found MB2 mesial to line connecting MB1 to palatal canal

Pineda & Kuttler 1972
Examined 7,275 root canals; 85% of root canal curvatures are found in the apical third of the root. Foramina of the main root canal were located on one side of the apical foramen 10% of the time.
<table>
<thead>
<tr>
<th>Author</th>
<th>Text</th>
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<tbody>
<tr>
<td>Chohayeb 1983</td>
<td>This investigation demonstrates that the maxillary lateral incisors have a high tendency to dilacerate distolabially (52%) , and this could be related to the incidence of failure.</td>
</tr>
<tr>
<td>Wilcox &amp; Walton 1989</td>
<td>When cutting access in crowned tooth, remember that pulp chamber is in center of crown</td>
</tr>
<tr>
<td>Leeb 1983</td>
<td>Remove cervical ledges over canal orifice during access prep to enhance straight-line access</td>
</tr>
<tr>
<td>Lowman, Burke, Pelleu 1973</td>
<td>The purpose of this study was to determine, radiographically, the incidence of patent accessory canals in the coronal and middle thirds of the roots of molars. From this study, 59% of all the teeth had accessory canals (55% max and 63% man), therefore, one should not assume all furcal lesions are of periodontal etiology. Confirmed by Burch (1974) who reported that 76% of all molars have accessory canals in the furcation area</td>
</tr>
<tr>
<td>Trope &amp; Elfenbein 1986</td>
<td>Pts of African-American descent have 3X &gt; incidence of 2 canals / 2 roots in mand premolars</td>
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### Dental Anomalies

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<th>Author</th>
<th>Text</th>
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<tr>
<td>Sabala, Benenati, Neas 1994</td>
<td>This study determined the relative incidence of bilateral morphological aberrations (bifurcation, C-shaped, fused roots). Of the 221 unusual or aberrant situations, 60.2% were bilateral. Aberrations occurring less than 1% of the time were 90% bilateral. If dental aberrations are present, valuable information may be acquired through the evaluation of the contralateral tooth.</td>
</tr>
<tr>
<td>De Smit, Jansen &amp; Demaut 1984</td>
<td>The results support the hypothesis that morphogenesis of invaginated teeth occurs as an active apically directed proliferation of ameloblasts or as a local growth retardation of the inner enamel epithelium. Although only one case was seen to have a possible connection between the pulp and the invagination, after eruption this area of dens invagination may become a “weak spot where bacterial invasion” could occur.</td>
</tr>
<tr>
<td>Hulsmann 1997 IEJ</td>
<td>Review: Dens invaginitus due to infolding dental papilla during development. MOA unknown, but could include growth pressure of the arch buckling enamel organ, infection, trauma, fusion of two tooth germs. Clinically seen as deep infolding of enamel and dentin may extend deep into the root. Hallet (1953) proposed classification: Type I enamel-lined minor form; Type II enamel lined form that invade root but is still blind sac; Type III invades root and has 2nd foramen (opening). 1° max laterals; often “peg-shaped” &amp; bilateral. Frequently results in pulp necrosis. NSRCT difficult due to complex anatomy. First described by Ploquet 1794 in a whale’s tooth. Tx: Sealants applied to fissure, NSRCT described by Hovland 1977; C&amp;S difficult (consider Ca(OH)2, US files, thermoplasticized GP).</td>
</tr>
<tr>
<td>Froner 1999 EDT</td>
<td>Case report: Dens invaginitus (Dens in dente) Type III max lateral. Combined NSRCT (of main canal) and endo sx (retro-fill with GP-Roths) with good 3yr followup</td>
</tr>
<tr>
<td>Turell &amp; Zmener 1999</td>
<td>Described NSRCT in fused mand molar</td>
</tr>
<tr>
<td>Rotstein, Stabholz, Heling, Freidman 1987</td>
<td>Two categories for case selection of dens invaginitus: Category A – no pathosis, treated by prophylactic measures including sealing with composite. Category B – pathosis present, requiring pulpal therapeutic intervention. Clinical considerations include function and esthetics of invaginated teeth and complications associated with root canal therapy. Direct access may be difficult and may result in perforations. If this is the case, surgical therapy may be the treatment of choice.</td>
</tr>
</tbody>
</table>
Dens evaginatus is a coronal anomaly of premolar teeth with a reported incidence of 1-2%. It is rare in this country and affects mainly people of Mongoloid ancestry. It is composed of enamel and dentin, with a pulpal extension into it that may be detected radiographically. In this case a 32 year old Filipino woman was diagnosed with bilateral dens evaginatus with associated periapical involvement secondary to pulpal necrosis. Early recognition with appropriate therapy can prevent loss of these otherwise normal teeth. Apexogenesis should be the initial goal, followed by root canal therapy later if necessary.

Mellor, Ripa 1970
A talon cusp is characterized by a cusp-like projection arising from the cingulum area of a maxillary or mandibular incisor. Normal radiographic tooth structure, enamel, dentin and pulp tissue. At the junction of the cusp and the lingual surface of the incisor, there is a developmental groove, which creates a large niche to harbor bacteria. Recommended that prophylactic restorations be placed in these cases.

Cooke, Cox 1979
C-shaped canal configuration. Radiograph showed two-roots close together with one canal in each root. Upon access a normal pulp chamber with two canals centered in the buccolingual direction was found. Cleaning and shaping. A finding in all 3 cases was persistent hemorrhage and pain on instrumentation. They believe that C-shapes are impossible to dx from radiograph. Primarily mand 2nd molars, although Bolger and Schindler 1988 have reported C-shape mand 1st molar. Also: Yang & Yang (1988) reported that Chinese have 4.9% incidence of "C" shaped canals in max molars.

Canal Preparation: Access, Isolation, Instrumentation

Rationale for Instrumenting 0.5-1mm short of the radiographic apex:

Morphological Studies:
1. Kuttler (1955): 0.50 mm (young) to 0.65mm (old)
2. Burch & Hulen (1972): 0.59 mm
3. Stein & Corcoran (1990): 0.72 mm width of CDJ = 0.19mm
4. Tamse & Littner (1988): 0.80 mm

- BUT- Gani & Visvisian (1999 JOE): studied apical canal diameter in max 1st molars. At 2mm from apex, palatal systems are 60% circular and 30% ovoid regardless of age. At 2mm from apex, MB systems are 50-60% flat (ie, ribbon, tear-shaped) and 30% ovoid (no clear cut age effect). Interestingly, DB systems 30-60 circular. Problem is that if C&S in flat canal system for the long dimension, could perf in narrow dimension.

Pulp – PA Pathology Studies:
- Malueg, Wilcox & Johnson (1996): SEM of teeth with varying external apical root resorption (n= 40. Apical resorption: pulpal necrosis > normal pulp, reversible pulpalitis, or irreversible pulpititis. Teeth with periapical lesions had significantly more apical resorption than those without radiographically evident periapical lesions. Therefore, the status of the pulp and periapical tissues should be considered when determining length for preparation and obturation.
- Frank (1990) Also reported this finding (ie, necrotic teeth tend show more apical resorption).
- Trope & Chivian (1994) propose that CDJ at foramen is very thin (in some cases, absent) – exposing mineralized dentin to the resorptive clastic cells.

Outcome Studies:
- Sjogren (1990): Outcomes study: Best success for tx necrotic cases with apical periodontitis are when the obturation ends within 0-2 mm of radiographic apex (= 94%); underfills are less successful (68% when filled > 2mm from apex) and overfills are less successful (76%).
- Ricucci (1998 IEJ): Review article and 100 case report series. Conclude that best result is to obturate at apical constriction which ranges 0.5-2mm short of radiographic apex.
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<th>Author(s)</th>
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<tbody>
<tr>
<td>Lovdahl &amp; Gutmann 1980</td>
<td>Described gingivectomy (prefers scalpel over electrosurg) with reverse bevel for isolation indication: Dentin margin needs to be 3mm above crestal bone to give space for 1-2mm sulcus depth; want to preserve 4mm zone of attached gingiva</td>
</tr>
<tr>
<td>Bramwell &amp; Hicks 1986</td>
<td>Described use of oraseal or Cavit to seal leaky RD</td>
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### Calcified Canals

<table>
<thead>
<tr>
<th>Author(s)</th>
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<tr>
<td>Wilcox &amp; Walton (1989):</td>
<td>Pulp chamber is in center of crown</td>
</tr>
<tr>
<td>Gutmann:</td>
<td>Use long shanked #2 round; check orifices with sharp DG-16 endo explored. Initial stem-winding motion with #8 Pathfinder CS (Kerr) since it has a stiff shank (MUCH better than NiTi)</td>
</tr>
<tr>
<td>Leeb (1983)</td>
<td>Remove cervical ledge near orifice</td>
</tr>
<tr>
<td>RC Prep (Premier Dent Products)</td>
<td></td>
</tr>
<tr>
<td>Stamos (1985)</td>
<td>Rec use of US files to gain access and file calcified canals; and to remove alloy or particles packing RC system</td>
</tr>
<tr>
<td>Schindler (1988):</td>
<td>If cannot bypass calcification, then C&amp;S &amp; obturate to level of calcification; place on recall for potential Sx</td>
</tr>
<tr>
<td>Glyoxide = 10% carbamide peroxide in glycerol; Marion Labs</td>
<td></td>
</tr>
<tr>
<td>Flexofiles are available in 1/2 steps (&quot;Flexofile Golden Mediums&quot;; LD Caulk)</td>
<td></td>
</tr>
<tr>
<td>Weine (1970):</td>
<td>Rec customize files by cutting 1mm from #10 to make #12 (However- cutting end vs pilot tip, etc)</td>
</tr>
<tr>
<td>EndoZ bur -</td>
<td>safe ended carbide bur to enlarge access (LD Caulk)</td>
</tr>
<tr>
<td>Ngai (1986):</td>
<td>Described use of US files to bypass separated instruments in canals</td>
</tr>
<tr>
<td>Weine (1975):</td>
<td>Described zipping = elliptication = transportation of apical portion of the canal (eg., straightening a curved canal). The apical foramen becomes tear-dropped shaped due to excessive cutting of the outer portion of curved canal at file tip and inner portion of curved canal at more coronal portion of the file. Consider obturation with warm thermoplasticized GP to fill this unevenly prepared canal system. Use Sealapex in these cases (since contains Ca(OH)2; Kerr).</td>
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<tr>
<td>Pliet &amp; Sorm 1973</td>
<td>Triangular instruments cut more efficiently than square files</td>
</tr>
<tr>
<td>Walia, Brantley, Gerstein 1988</td>
<td>1st description of NiTi (&quot;nitinol&quot;) files</td>
</tr>
<tr>
<td>Wildey, Senia 1989</td>
<td>1st description of Canal Master</td>
</tr>
<tr>
<td>Profile: .02, .04, .06 mm taper. ISO sizes or Series 29: (Constant 29% increase in file size giving 13, 17, 22, 28, 36, 47, 60, 77, 100 sizes)</td>
<td></td>
</tr>
<tr>
<td>Short &amp; Baumgartner 1997</td>
<td>Lightspeed and Profile were faster than hand filing and kept files centered in canal better that ss hand files</td>
</tr>
<tr>
<td>Pruett, Clement, Carnes 1997</td>
<td>Cyclic fatigue testing of nickel-titanium endodontic instruments. NiTi instruments fracture within their elastic limit and without any signs of previous permanent distortion. Rotation subjects NiTi to both tensile and compressive forces in the area of the canal curvature; this produces a very destructive form of loading.</td>
</tr>
<tr>
<td>Dederick &amp; Zakariasen 1986</td>
<td>Axial movement during instrumentation may distribute stresses along the shaft and reduce risk of fracture. (Cite this along with the Pruett study on cyclic fatigue).</td>
</tr>
<tr>
<td>Author(s)</td>
<td>Summary</td>
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<tr>
<td>Love 1996</td>
<td>Bacteria can invade up to 150-250 um into dentinal tubules. Confirmed by Sen (1995): bugs grow 150um into tubules. Thus, Yared &amp; Bou Dagher 1994 advocate apical preparation to 0.3-0.5 mm larger than original size (and width of CDJ is often 0.19mm (Stein &amp; Corcoran 1990)).</td>
</tr>
<tr>
<td>Klevant 1983 IEJ</td>
<td>Chemomechanically debrided RC systems of 86 human teeth and left un-obturated for 2 years. Radiographic exam showed significant decrease in PARLs in C&amp;S-unobturated and C&amp;S-obturated teeth. Thus, reject &quot;hollow tube&quot; theory for breakdown of tissue fluid inducing PA lesion. (Should point out that even though C&amp;S produced significant radiographic healing of AP, better healing was observed in C&amp;S-obturated group. Also reported by Donnelly 199, Weine, and others (see Klevant for refs))</td>
</tr>
<tr>
<td>Jahde &amp; Himel 1987</td>
<td>A small amount of inflammation and localized bone necrosis occurs with file overextension.</td>
</tr>
<tr>
<td>Roane &amp; Sabala 1984</td>
<td>A CW rotation of a file has greater chance of separation than a CCW rotation. Confirmed by Seto &amp; Harrington 1988</td>
</tr>
<tr>
<td><strong>Apex Locators</strong></td>
<td></td>
</tr>
<tr>
<td>Suzuki (1942)</td>
<td>reported that PDL and oral mucosa have a constant electrical resistance of ~6.5 kOhms</td>
</tr>
<tr>
<td>Sunada (1962): Classic!</td>
<td>Applied Suzuki’s idea to develop an apex locators</td>
</tr>
<tr>
<td>Old style = resistance (ex: NeoSono, Formatron)</td>
<td></td>
</tr>
<tr>
<td>Next generation = dual frequency (ex: Root ZX, Endex)</td>
<td></td>
</tr>
<tr>
<td>Pagavino (1998): Root ZX has 83% accuracy ± 0.5mm (includes teeth with lateral foramina)</td>
<td></td>
</tr>
<tr>
<td>Dunlap &amp; Rauschenberger (1997 JOE): Root ZX used in teeth scheduled for extraction; cemented files and verified position. 82.3% accurate to 0.5mm of apical constriction. Mean distance from apical constriction was 0.21mm in vital cases and 0.49mm in necrotic cases (NS difference).</td>
<td></td>
</tr>
<tr>
<td>Fouad (1993): Apex locaters ok on pts with a pacemaker (even though Root ZX manual says not to use it on pts with pacemakers)</td>
<td></td>
</tr>
<tr>
<td>Beach &amp; Hutter (1996): Case report of using apex locator on a pt with a pacemaker</td>
<td></td>
</tr>
<tr>
<td>Fuss (1996): Describes use of Apex Locators to locate perforations</td>
<td></td>
</tr>
<tr>
<td>Ibarrola (1999 JOE): Preflaring canals permits WL files to reach apical foramen more consistantly with Root ZX.</td>
<td></td>
</tr>
<tr>
<td>Ahmad 1987</td>
<td>Most of the benefits of ultrasonics are due to acoustic streaming rather than cavitation.</td>
</tr>
<tr>
<td>Huque &amp; Iwaku 1998 IEJ</td>
<td>Ultras onics with 5.5% NaOCl is effective in eradicating bacteria from infected dentin (artificial smear layer infected with Actinomyces, Fusobacterium, Streptococcus)</td>
</tr>
<tr>
<td>Haikel 1998 JOE</td>
<td>NiTi – 2 phases: Austenite (= manufactured state) and Martensite. The ability to cycle between these two states is due to NiTi having properties of superelasticity and shape memory. Phase transition occurs with rapid stress on file (therefore, use at a constant speed). Files are weakest during phase transition and have highest probability of fx at this time</td>
</tr>
<tr>
<td>Haikel 1999 JOE</td>
<td>In vitro study with tempered steel canals: As radius of curvature decreased, fracture time decreased. Taper of files was also significant in determining fracture time (increased diameter = decreased time). ie, 06 taper will fracture sooner than 02 taper...</td>
</tr>
<tr>
<td>Walton 1976</td>
<td>Tapering preparation permits better debridement of apical preparation, reduces over-instrumentation of the foramen and improves ability to obturate</td>
</tr>
<tr>
<td>Author(s)</td>
<td>Description</td>
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<tr>
<td>Abou-Rass, Frank &amp; Glick</td>
<td>Classic: describes anticurvature filing. Defined danger and safety zones</td>
</tr>
<tr>
<td>Gambi &amp; DelRio 1995</td>
<td>NiTi files may fxn best when used in reaming or rotary fashion (since less transportation and canal deviation)</td>
</tr>
<tr>
<td>Weine &amp; Kelly 1975</td>
<td>Termed “apical zip”, discussed elbow, teardrop apex and hourglass shape. Argued against reaming (before NiTi).</td>
</tr>
<tr>
<td>Mullaney DCNA 1979</td>
<td><strong>Step-back (Telescopic Technique)</strong>&lt;br&gt;• Determine WL &amp; develop apical stop to #25&lt;br&gt;• Step-back by shortening 30, 35, 40 in 0.5 or 1 or 2mm increments&lt;br&gt;• Recapitulate with #25&lt;br&gt;• Coronal flare with #2 &amp; 3 Gates-Glidden</td>
</tr>
<tr>
<td>Goerig JOE 1982</td>
<td><strong>Step-Down technique.</strong>&lt;br&gt;• Passively use #15, 20, 25 Hedstrom in coronal 2/3 of canal system; irrigate&lt;br&gt;• Coronal flare with #2 &amp; 3 Gates-Glidden&lt;br&gt;• Establish WL and prepare apical seat with stnd serial filing&lt;br&gt;• Step-back to blend apical and coronal segments&lt;br&gt;• Recapitulate&lt;br&gt;The crown-down pressureless technique (Morgan &amp; Montgomery JOE 1984) is similar to the Step Down: Rotate straight file twice from larger to smaller sequence until reach 16mm. Coronal flare with GG. Establish provisional WL 3mm short of apex. Rotate straight file twice at WL. Finish apical prep at WL with file 2 sizes larger than first file to reach WL</td>
</tr>
<tr>
<td>Roane &amp; Sabala 1985 JOE</td>
<td><strong>Balanced force technique</strong> (use FlexR files (Moyco Union Broach) or Flexofile for non-cutting pilot tips of triangular file)&lt;br&gt;• Use Crown-Down to establish radicular access&lt;br&gt;• Rotate straight file CW from 90-180° with light apical pressure to engage dentin&lt;br&gt;• Shear dentin by 120° CCW rotation with apical force, flexing it to conform to canal curvature&lt;br&gt;• Continue until get adequate apical enlargement at WL&lt;br&gt;• Inspect files frequently; do not go beyond #35 in curved canals</td>
</tr>
<tr>
<td>Fava 1983 JOE</td>
<td><strong>Double-flared technique.</strong>&lt;br&gt;• Passively use larger-smaller files in coronal 2/3 of canal system; irrigate&lt;br&gt;• Establish WL with small K file. Serial file to prepare apical stop and then step back to blend with coronal step-down flare&lt;br&gt;• Circumferentially file with master K file</td>
</tr>
<tr>
<td>Torabinejad 1994 OOO</td>
<td><strong>Passive step-back technique:</strong>&lt;br&gt;• Establish canal patency with small K file at WL then passively instrument with larger K files&lt;br&gt;• Coronal flare with #2, 3 and possibly #4 GG in coronal 1/3&lt;br&gt;• Confirm WL (since coronal flare and removal of curvatures often reduces WL)&lt;br&gt;• Increase straight line access with careful re-work with GG&lt;br&gt;• Serial file to prepare apical stop and then step back to blend with coronal step-down flare</td>
</tr>
<tr>
<td>Wilcox and Walton 1989</td>
<td>Studied access of molars: DB orifice is slightly distal to buccal groove. Rec start access prep centrally, and not at MMR.</td>
</tr>
</tbody>
</table>

**Instrumentation and Removal of Bugs**<br><br>• Bystrom & Sundqvist (1981): One steps do not remove bacteria in necrotic cases. Ca(OH)2 is the best inter-appt medicament to kill residual bacteria. Simple mechanical debridement with saline is insufficient to remove all bacteria (although it does reduce bugs by 100-1,000 fold).
**Bystrom - Sundqvist '81**

- Dalton and Trope (1998 JOE): n=48 MB canals of mand necrotic molars with apical periodontitis (AP defined as PARL) were found to be uniformly infected (96% of teeth with AP had CFUs in MB canals; similar to 95% of Sundqvist (1976) and 96% of Orstavik (1991)). NiTi rotary (Profile) = SS files (step-back) for reducing CFU (saline irrigation). Saw progressive decrease in CFUs with progressive sampling during filing with larger files, regardless of NiTi or SS. Suggests that tx approach to infected teeth with AP may require additional antimicrobial measures than just instrumentation, irrigation and aseptic technique (ie, inter-appt Ca(OH)2).

- Siqueira (1999): Infected 35 mand premolars with E. faecalis; NiTi rotary & saline irrigation: (Profile 06, GT) reduced 94-99% bugs; Larger file sizes had greater reduction of bugs (but only looked up to #40)

## Intracanal Irrigants and Medicaments

### Infected Dentinal Tubules

- **Orstavik '90**
- **Estrella '99**

- **Perez (1993):** Strep sanguis grew 479um into dentinal tubules by 28 days
- **Orstavik (1990 EDT):** E. faecalis & Strep sanguis grew 300-400um into slabs of bovine dentinal tubules after 14-21 days. Presence of a smear layer delayed, but did not prevent, antimicrobial effects of medications.
- **Sen (1995):** bugs grow up to 150um into tubules
- **Love (1996):** bugs grow 150-250um into dentinal tubules
- **Estrella (1999 JOE):** Ca(OH)2 demonstrated NO antimicrobial effect at 2, 3, & 7 days against E. faecalis, S. aureus in infected dentinal tubules (suggests antimicrobial effectiveness is due to concentration of [OH] and time of exposure)
- Thus, Yared & Bou Dagher 1994 advocate apical preparation to 0.3-0.5 mm larger than original size (and width of CDJ is often 0.19mm (Stein & Corcoran 1990). However, remember Gani (1999 JOE) report on canal shape (ribbon) and instrumentation

### NaOCl

- **Bystrom '85**
- **D'Arcangelo '99**
- **Cunningham '80**
- **Ellerbruch '77**

- **Hand & Smith (1978):** 5.25% NaOCl has superior tissue dissolving properties.
- **Harrison & Hand:** diluting NaOCl can reduce antimicrobial effectiveness
- **Bystrom & Sundqvist (1985):** Antimicrobial effectiveness of 0.5% NaOCl = 5% NaOCl. 15% EDTA enhanced the effectiveness.
- **D'Arcangelo (1999):** 0.5% = 1% = 2.5% = 5% NaOCl for antimicrobial effectiveness (11 strains inc E. faecalis; in fac aerobes-aerobes, microaerophiles, obligate aerobes). IMPORTANT POINT: Best when use at least 10 min contact time
- **Ellerbruch & Murphy (1977 JOE):** Vapors of 5.25% NaOCl have strong antimicrobial activity
- **Cunningham & Joseph (1980):** 2.6% NaOCl is more effective in antimicrobial action at 37C.
- **Senia & Marraro (1975):** GP cones sterilized at chair-side by 1 min immersion in 5.25% NaOCl. Also reported by Frank & Pelleu 1983.
- **Siqueira (1998 EDT):** 5% NaOCl destroyed Bacillus subtilis spores from GP cones within 1 min of immersion

### NaOCL Accidents

- **Reeh & Messer '89**
- **Gatot '91**
- **Becker & Cohen '74**

- **Reeh & Messer (1989 EDT):** long term paresthesia (still present at 15months) after injection 1% NaOCL thru buccal perf of a maxillary incisor
- **Gatot (1991 JOE):** long term paresthesia can occur with NaOCL injection
- **Becker & Cohen (1974 OOO):** NaOCl injected beyond apex = PAIN! Tx with steroids iv and continue for 3 days
- **Recommendations for tx (from Gluskin, POP):** long acting LA, Amox X 5 days, analgesic, Steroid, cold compresses,

### EDTA & NaOCl

- **Baumgartner '87**
- **Yamada '83**
- **Margelos '97**

- **EDTA removes smear layer, but does not remove organic debris:** Baumgartner 1987; Garbergolio 1994
- **NaOCl is antibacterial and removes organic debris, but does not remove smear layer:** Shih 1970; Senia 1971; Baumgartner 1987
- **Alternating EDTA and NaOCl effectively removes smear layer, tissue, predentin and increases antimicrobial activity:** Baumgartner 1987; Goldman 1982; Bystrom 1985; Tatsuta & Baumgartner 1999
- **Yamada (1983 JOE):** The most effective way to remove organic and inorganic components of smear layer is 10ml 17% EDTA and then 10ml 5.25% NaOCl
- **Calt (1999 JOE):** Use both EDTA & NaOCl to maximally remove Ca(OH)2 dressing from canal system
• Patterson (1963): EDTA is self-limiting in its action
• Margelos (1997 JOE): Ca(OH)2 left in canals can accelerate setting of Roths. FTIR spectroscopy indicates that Ca evokes rapid sealer setting into a brittle and granular material with free eugenol in the set product. EDTA was best agent to remove RC systems tx with Ca(OH)2 medicament

Smear Layer:
ElDeeb ‘83
Evans & Simon ‘86
Jeansonne ‘96
Glickman ‘95

• Ishley & ElDeeb (1983): Sealer was more important that the type of obturation used (McSpaden versus lateral condensation)
• Evans & Simon (1986): Presence or absence of smear layer does not affect microleakage (dye leakage study eval both lateral condensaton of GP and Obtura system). The use of sealer is much more important in controlling leakage!
• Madison & Krell (1984): Presence or absence of smear layer does not make difference in leakage
• Takeda (1998): Er-YAG laser can remove smear layer
• Foster (1993) Removal of smear layers facilitates diffusion of Ca(OH)2 to kill bacteria (Bystrom: OH moiety is bactercidal)
• Gutmann (1993) Showed enhanced adaptation of thermoplasticized GP into dentinal tubules without smear layer
• Craig & Harrison (1993): Citric acid (50% X 2 min; pH=1) tx of resected root ends removes smear layer, exposes collagen and enhances cementogenesis

Irrigation and Needle Size / Location
• Ram (1977 OOO): Effective irrigation requires apical preparation. Rec prep size of #40 to get effective delivery of irrigants
• Salzgeber & Brilliant (1977): Irrigant reaches apex when canal systems are opened to file size 30
• Abou-Rass (1982): The closer the needle is to the apex, the better the irrigation (ie, needle does not irrigate much past the bevel tip)

Ca(OH)2
Bystrom ‘81-85
Sjogren ‘91
Saffey ‘93
Trope ‘97
Messer ‘97
Fava ‘99

• Bystrom & Sundqvist (1981; 1985): is antimicrobial
• Sjogren (1991): Ca(OH)2 applied for 7 days eliminated bacteria in canal systems - even up to 5 weeks later (Bystrom 1985 looked at one month of tx). 0.17% dissolves to form Ca++ and OH, requires at least 1 day to exert full effect
• Safavi & Nichols (1993): Ca(OH)2 inactivates LPS in vitro  Also reported by: Barthel & Trope 1997 (IEJ)
• McCormick (1983) Osteoclastic cells (osteoclasts & PMNs) prefer acidity. The high pH of Ca(OH)2 antagonizes their action
• Foster (1993) Removal of smear layers facilitates diffusion of Ca(OH)2 to kill bacteria (Bystrom: OH moiety is bactercidal)
• Segura (1997) Ca(OH)2 inhibits macrophage adherance (may contribute to Ca(OH)2 inhibition of resorption)
• Estrela (1995): antimicrobial action due to OH-
• Sigurdsinsson (1992) Lentulo spiral is most effective technique of carrying Ca(OH)2 to working length
• Nerwich & Messer (1993): Evaluated dentinal pH after Ca(OH)2 dressing. Inner dentin pH rapidly increases by OH diffusion (peaks 1 day), but takes 2-3 weeks to peak in outer dentin. Peak pH ~9-10 with cervical dentin peaking before apical dentin.
• Hasselgren, Olsson & Cvek (1988): Ca(OH)2 completely dissolves porcine muscle over time. Ca(OH)2 plus NaOCl QUICKLY dissolves muscle. May be clinically significant when use Ca(OH)2 as intracanal medicament and then rinse out with NaOCl. (not seen over 30min period by Morgan and Carnes 1991). To confirm Morgan & Carnes, Yang, Rivera, Walton (1996) showed that inter-appt NaOCl + Ca(OH)2 does not enhance debridement.
• Fava & Saunders (1999 IEJ): Reviewed Ca(OH)2 paste formulations and indications. Vehicle (aqueous, viscous, oily) plays important role in dissolution kinetics. Eg., Calisept is 56% Ca(OH)2
• Available in single dosage formations: Centrix syringe tips (=SteriCal®)

Chlorhexidine
Jeansonne ’94
Torabinejad ’93

• Jeansonne & White (1994): Antimicrobial properties of 2.0% chlorhexidine gluconate = 5.25% NaOCl.
• Ohara & Torabinejad (1993 EDT). Chlorhexidine effective antimicrobial against 6 strains of anerobes.
Heline '98
Martin '87
Lindskog '98
Leonardo '99

- Heline (1998 IEJ): Chlorhexidine is effective in dentin infected with E. faecalis (i.e. chlorhex = NaOCl)
- Martin & Nind (Br Dent J 1987): Chlorhexidine gluconate can be irrigated into apicoectomy sites to reduce flora 94% immediately and 78% even after 10 days! ? Effect on hemostasis & healing? If ok, something to consider for immunocompromised pts?
- White (1997 JOE): Intracanal chlorhexidine stills shows substantivity.
- Leonardo & Ito (1999): 2% chlorhexidine has good antimicrobial activity. Cultured RC systems (n=22 necrotic with AP) with 2%C as irrigant. Saw immediate reduction of bugs in canals with residual effects in RC system up to 48hr after tx.

Perez & Cardenas 1989

- EDTA is self-limiting since its efficiency is reduced during chelation. Clinically, this means that should replace EDTA during chelation and that inter-appt EDTA is efficient for only short period of time

Messer 1984

- CMCP loses about 90% of its effectiveness (active agent = parachlorophenol) in first 24hr. Moreover, CMCP clears bacteria from only 67% of RC systems, compared to 97% by 1 month tx with Ca(OH)2 (Bystrom 1985). Thus, CMCP is not useful as intracanal medicament.

Hoshino 1996

- Evaluated mixture of ciprofloxacin, metronidazole and minocycline to kill bacteria in infected human dentin, periapical lesions and infected pulps under strict anerobe conditions. None of the agents killed 100% when given alone; but the combo was 100% effective. Proposed as possible intracanal disinfectant. Also seen by Sato (1992) in infected RC systems.

Max Goodson & Stashenko 1999 JOE p722

- Evaluated clindamycin impregnated fibers as intracanal medicament. A 10mm fiber was effective in vitro against 12 organisms for 4 days. Zone of inhibition ranged from 10-100mm.

Obturation:

- Over-Fill = 3D obturation with some GP beyond apex
- Over-extension: Excess GP beyond apical foraman, BUT- no implication of a 3D obturation

Allard & Stromberg 1987

- Dog study: In microbiologically-induced PA lesions, got 4 month healing even when obturate with bacteria remaining in canal systems. Thus, can get healing even when canals are still infected. HOWEVER - Contrast with Sjogren 1997 who showed in humans that prognosis is reduced if bacteria are present at time of obturation.

Spreaders

- Allison & Walton (1981): Less leakage occurs if the spreader reaches within 1mm of the apex. Tugback of the master cone is NOT a good predictor
- Berry & Runyan: NiTi spreaders penetrate curved canals to significantly greater depth than SS spreaders
- Speier & Glickman (1996): Rec use of NiTi finger spreaders in apical compaction and SS spreaders for coronal 2/3 (to minimize buckling of NiTi spreaders)

If use hand spreaders:

D11T = normal cases
**D11T2** = small apical prep (max MAF = 25-35)
**GP3** = long canals (>23mm; HuFriedy)

**GP Properties:**
- Alpha phase is natural form (= 1,4-polyisoprene = dried juice of the thebaine tree), introduced by Jose D’Almeida, phase transition to beta phase at ~47°C. Examples include Thermafil, Successfil, Alpha Phase, Ultrafil
- Crystalline forms are alpha (slow cooling, natural) and beta (fast cooling)
- Spangberg (1969): Gutta percha has low tissue toxicity. But – REMEMBER that this is due in part to particle size (small GP pieces are extremely inflammatory = Sjogren 1995 Eur J Oral Sci).
- Kolokruis (1992): Store GP in refrigerator and at low humidity
- GP in beta phase will shrink after warm compaction technique, this is rationale for continued vertical compaction pressure
- Moore & Genet (1982 OOO): GP cones display slow acting (and weak) but significant antimicrobial action (may be due to ZnO)
- **Constituents of GP Cone:**
  - 59-75% ZnO - filler; antimicrobial
  - 19-22% GP - core material
  - 1-17% Heavy metal sulfates – radiopacity (eg., Barium sulfate)
  - 1-4% Waxes & resins - make more compactable; resins also antimicrobial
  - 0.1-0.3% Pigments
  - GP = trans isomer of isoprene (= poly trans 1,4-isoprene)
  - Alpha comes from tree
  - Beta made by heating alpha >65°C and slowly cooling

**Sunzel 1990 & 1995**
Zinc oxide has effective antimicrobial activity. Note that GP cones contain ~70% ZnO, 20% GP and rosins, waxes & metal sulfates. The rosins confer “stickiness” to dentin, reduce ZnO solubility and exert antimicrobial effects. The setting of ZOE forms ZnO crystals in a matrix of zinc eugenolate.
Friedman 1977 described composition of GP cones. The coloring agent in commercial GP is erythrosin (Marciano 1993).

**Jacobsen (1984)**
If cut GP cone with scissors, it leaves a flange that interferes with placement. To remove flange, cold roll GP between spatula and glass slab or use rolling cut with scalpel blade.

**GP Solvents:**
- Tamse '86
- Hicks '90
- McDonald '92
- Chutich '98
- Wilcox '87 & '89
- Rotstein '99
- Metzger '95

- **GP Solvents:**
  - Wourms & Hicks (1990): Reviews use of halothane as alternative GP solvent
  - McDonald & Vire (1992): Measured room air chloroform levels during endo. Air samples well below OSHA limits (8hr limit = 2 ppm)
  - Chutich (1998): The amount of chloroform, halothane or xylene exiting thru apical foramen during in vitro re-tx is 1,000 -15,000 times below permissible toxic dose.
  - Metzger (1995): Use solvent to soften coronal 1/2 of overextended GP, insert Hedstrom, let GP set hard then slow withdraw GP
  - Stamos (1988): Don’t let solvent get past apex! Pain!!
  - Rotstein (1999 JOE): chloroform, halothane & xylene: softened dentin & enamel (chloroform softened dentin by 29% after 15min)
  - Wong & Peters (1982 JOE): Chloroform dip technique shows 1.4% shrinkage (in contrast, chloropercha shows 12.4% shrinkage)
  - Wilcox (1987 & 1989 JOE): Examined RC walls after heat, files, chloroform, US for Roths 801 vs AH26. All techniques incompletely cleaned walls; AH26 more difficult to remove than Roths

**Grossman’s Sealer**
42% ZnO - filler, antimicrobial
27% Stabelite resin - gives body, coherence, good setting time
15% Bismuth Subcarbonate - accelerates setting time
15% Barium sulfate - radiopacity
1% Borax - retards setting time

EUGENOL – matrix (ZnO-eugenolate), antimicrobial, anti-PLA2, neuromodulator (capsaicin congener)

**Why use sealer?**
Binding agent for RC core filling material
Fills voids and discrepancies in canal walls
Prevents leakage
Acts as lubricant for fill

**Roth’s Sealer**
- Brown Jackson & Skidmore (1994): Apical seal with Roth’s 801 sealer better than Ketac-Endo
- Mickel & Wright (1999): Roths sealer has better antimicrobial activity vs Sealapex & CRCS (Ca(OH)2 containing sealers. Probably due to eugenol.
  In vitro evaluation using Streptococcus anginosus
- Abdulkader & Saunders (1996): In vitro antibacteria activity against anerobes: Roths > Sealapex
- Shalhav (1997) Roths exhibited 7day antimicrobial activity against E. faecalis (Ketac Endo was not as long-lived)
- Grossman (1976): Roths's 801 little shrinkage when sets

**Sealapex**
**Base:**
- Ca(OH)2 25%
- ZnO 6.5%

**Catalyst:**
- Barium sulfate 18.6%
- Titanium dioxide 5%
- Zinc stearate 1%

**AH26** (NB: AH26 PLUS - see Leyhausen JOE)
**Powder:**
- Silver Powder: 10%
- Bismuth Oxide: 60%
- Hexamethylenentetramine 25%
- Titanium Oxide 5%

**Liquid:**
100% Bisphenoldiglycidyl ether

**Torabinejad & Bakland 1979**
No Ab formation or delayed hypersensitivity to Grossman’s sealer

**Parasthesia After Obturation**
**NaOCl Irrigation**
- Reeh & Messer (1989 EDT): long term paresthesia (still present at 15months) after injection 1% NaOCL thru buccal perf of a maxillary incisor
- Gatot (1991 JOE): long term paresthesia can occur with NaOCL injection
- Recommendations for tx NaOCL induced paresthesia: (from Gluskin, POP): long acting LA, Amox X 5 days, analgesic, Steroid, cold compresses
**LA**
- Haas (1995): LA induced paresthesia (esp, Prilocaine, articaine) esp mand blocks

**Sealer & Core Material**
- Allard (1986): case report of N2 induced paresthesia
- Tamse (1982 JOE): Case report of paresthesia after AH26 overfill
- Nitzan & Stabholz (1983 JOE): 5 cases of paresthesia after AH26 overfill; 1 overfill with ZOE sealer but no paresthesia
- Serper (1998): Model of post-obturation paresthesia: Isolated rat sciatic recording of compound action potential. 50% inhibition occurred at CRCS (6.6 min: Ca(OH)2 containing sealer), Sealapex (9.2 min: Ca(OH)2 containing sealer), N2 universal (4 min: contains paraformaldehyde). IMPORTANTLY: After rinsing, Sealapex recovered fastest (6 min) then CRCS (55min) or N2 (60min). Similar to Kozman 1977 who reported eugenol inhibited frog sciatic activity.
- Morse (1997): 2 cases reports of paresthesia after NSRCT. Case 1: chloropercha overfill; tooth asymptomatic for 2.5yr; then PARL increased and swelling, pain and paresthesia developed; resolved after Sx removal of lesion. Case 2: Formocresol pulpotomy; paresthesia started at 1 day; resolved after 7 weeks of dexamethasone (0.75mg #4 stat then taper) antibiotics and irrigation. CC #1 = burning, painful, numb-like sensation. CC #2 = numb lip

**Non-Endodontic Causes of Paresthesia:**
- Cancer metastasis: Glaser (1997 Intl JOS): numb lip most common feature of metastatic CA. Also reported by Selden 1998 who found metastatic carcinoma as PARL on mand molar; later developed paresthesia.
- Dumas (1999): trigeminal sensory neuropathy. Sensory disturbance is ominous sign. MOA = CNS metastatic neoplasia (esp men>60), multiple sclerosis. Often rapid onset, ~50% report pain, differential of symptoms includes post-endo pain

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**Seltzer & Green 1972**
Silver points removed in failed cases have corrosion products of sliver amide hydrate which is cytotoxic. Corrosion is increased by bending, cracking or deforming the cones at obturation. However, this was challenged by Kerekes & Rowe (1982) who found corrosion products on successful silver cone cases (which were lost due to periodontitis).

**Senia & Marraro 1975**
GP cones sterilized at chair-side by 1 min immersion in 5.25% NaOCl. Also reported by Frank & Pelleu 1983.

**Siqueira 1998 EDT**
5% NaOCl destroyed Bacillus subtilis spores from GP cones within 1 min of immersion

**Blum 1998**
Measured "wedging" force (predictor of fracture force) during obturation: Thermafill << warm vertical = thermomechanical (McSpadden) < lateral condensation

**Cooke & Grower 1976**
GP gives better seal than silver points

**Economides & Kotsaki-Kovati 1995**
Inflammatory response with sealers was least with CRCS < Sealapex < Roths, AH-26 (AH26 had greatest inflammation)

**Leyhausen 1999 JOE**
AH26 cytotoxicity due to release of formaldehyde from the epoxy resin. This is NOT released from the new formulation (= AH26 Plus), which showed lower cytotoxicity, and no genotoxicity (umu test) or mutagenicity (Ames test).
Grossman 1976
Sealers: Roths 801 (little shrinkage when sets & flows well), AH26 (flows well), Tubliseal (sets fast - consider Tubliseal when doing sx right after completing NSRCT).

Horsted 1978
Reported good results in vital cases where hemostasis cannot be controlled by obturating 2-4 mm short of the wound area. Should not do this in necrotic cases due to concerns of remaining bacteria. Recall: Sjogren (1990) data about success in necrotic cases!

Brothman 1981
Vertical compaction demonstrated TWICE the number of lateral and accessory canals and denser fill. Also: Gutmann (1993) Showed enhanced adaptation of thermoplasticized GP into dentinal tubules without smear layer

Sargenti
Newton
Spangberg
Allard
Kleier
- Sargenti - no rubber dam needed, access not addressed, RCT length somewhere near apex, objective is chemical (not C&S), opposes irrigation, try to keep N2 in canals but it is "well tolerated" in PA tissues. 4-7% paraformaldehyde, lead oxides
- Newton (1980): Demonstrated 6m and 1yr cytotoxicity of Sargenti paste
- Spangberg (1974): The formaldehyde containing N2 formulation produces extensive tissue necrosis. Since the paraformaldehyde in N2 will not be resorbed, must sx remove Sargenti material expressed beyond apex.
- Allard (1986): case report of N2 induced paresthesia
- Kleier (1988 EDT): painful dysethesia of the IAN after use of paraformaldehyde paste (Sargenti)
- Serper (1998 JOE): Model of post-obturation paresthesia: Isolated rat sciatic recording of compound action potential. 50% inhibition occurred at CRCS (6.6 min: Ca(OH)2 containing sealer), Sealapex (9.2 min: Ca(OH)2 containing sealer), N2 universal (4 min: contains paraformadehyde).
  IMPORTANTLY: After rinsing, Sealapex recovered fastest (6 min) then CRCS (55min) or N2 (60min)

Tronstad 1978
CLASSIC: Evaluated periradicular tissue response to dentinal chips in monkeys. Showed little response, and actually saw cementum deposition (rationale for use of dentinal chips to prevent overfill).

Holland 1996
Endodontic filling materials can induce periapical inflammation. Recall also Sjogren (1990) study that overfills of necrotic cases had lowered success than fill 0-2mm short (76% vs 94%).

Pascon 1991
Overfill into periapical tissues induces a foreign body reaction. Recall Holland (1996) and Sjogren (1990) for effect of overfill on inflammation and success.

Campbell 1978
No systemic antibodies develop to endodontic filling materials

Ca(OH)2
Frank (1966)
Cvek (1972)
Hicks (1987)
- Frank (1966): Described apexification techniques with Ca(OH)2
- Cvek (1972): Tx necrotic teeth with incompletely formed apices with Ca(OH)2. Got 95% success for apical closure.
- Cotti (1998 IEJ): Case report mand 2nd molar with extensive apical external resorption: C&S and tx with Ca(OH)2 for 24 months for apexification followed by Obtura
- Kleier (1991 EDT): n=48 apexifications with Ca(OH)2; mean time closure = 1yr; Lg PARLs had more flare-ups
- Weisenseel & Hicks (1987 JOE): Dye study showing that need 2mm of Ca(OH)2 to create stop and prevent leakage

MTA
FDA Indications
- FDA Indications for MTA: perforation repairs (non-communicating), apexification, root-end filling, pulp capping
Shabahang & Torabinejad (1999): Infected immature dog teeth: MTA (delivered with Messing gun using Lux pluggers) had greater % roots with apical barriers (93%) vs osteogenic protein-1 (~40%) and Ca(OH)2 (~40%).

Torabinejad & Chivian (1998): Described clinical indications and techniques for using MTA. Indicated for pulp capping (Pittford 1996), apical barrier (after 1 w Ca(OH)2 in necrotic cases; close wet cotton/cavit; obturate >4h later), root perfs, root end fillings (rec small carrier=0.9mm RR Carrier, Chige Inc; don't rinse sx site after placing MTA), orifice plug seal.

Rick Schwartz, Bill Walker (1999 JADA): Case reports of MTA for vertical root fx (with post), apexification, perf repair, and repair of internal resorption.

Tronstad (1978): CLASSIC: Evaluated periradicular tissue response to dentinal chips in monkeys. Showed little response, and actually saw cementum deposition (rationale for use of dentinal chips to prevent overfill).


Katebzadeh & Trope (1998 JOE): Apexification cases often have narrow roots and may fx easily. Developed strengthening technique using internal resin bonded composites (clear posts for polymerization and removal to permit remedication).

Garguilo & Orban 1967
- Biologic width, is the dimension from the crest of the alveolar bone to the base of the sulcus and includes the connective tissue attachment (1 mm) and epithelial attachment (1 mm)

Inger 1977
- Because of decay or trauma that causes loss of tooth structure at or below the alveolar crest, surgical correction should include a minimum of 3 mm of tooth structure above the alveolar crest so that the Biological Width can be reestablished to prevent its impingement during restorative procedures.
- Biggerstaff (1966): can accomplish with orthodontic extrusion & crown lengthening.

Anderson ’88
- Cavit and TERM provided better seal than IRM. Also reported by Barkhorder 1990.
- Wilderman (1971): Cavit is composed of Zinc oxide, calcium sulfate, glycol acetate, triethanolamine, polyvinyl acetate, polyvinylchloride acetate, red pigment
- Deveaux (1999): Bacterial leakage study (S. sanguis) Cavit better than TERM & IRM
- Weber & DelRio (1978): To prevent leakage, Cavit must be at least 3.5mm thick!
- Harris (1976): Reported on 245 cases of perf repair with Cavit. 89% success; need to seal immediately.
- Smith & Cunningham (1992): Recommended seal canal orifices with 2mm Cavit prior to walking bleach.

Goldman 1992
- Most cast restorations leak. Thus, may wish to use ZOE inside access prep to seal crown:dentin margin.

Stanley 1981
- 1mm of dentin reduces toxic effect of material to 10% of the original level. 2mm of dentin completed blocks pulpal response to a toxic material.

Felton 1989
- Long term follow-up of 1,000 crowned teeth demonstrated that 11% became necrotic.

Reeh 1989
- NSRCT procedure only reduced cuspal stiffness by 5%. In contrast, class I prep reduced stiffness by 20% and MOD prep (which destroys marginal...
<table>
<thead>
<tr>
<th>Posts:</th>
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<tbody>
<tr>
<td>• <strong>Trope</strong> (1985): argues that post space preparation weakens</td>
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<td>the tooth. AE composite in ant teeth strengthened them more</td>
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<td>than placing a post</td>
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<td>• Nayyar &amp; Walton (1980): 4yr study (n=400) demonstrating</td>
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<td>success of amalgam core with extension into coronal 2-4 mm of</td>
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<td>root canal systems. This is a viable alternative over posts.</td>
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<td>This technique can be improved by use of amalgam bonding agents</td>
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<td>(= 4-META) such as Amalgambond (Parkell Inc) [Cooley 1991]</td>
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<tr>
<td>• Stockton (1998 EDT): Rec that posts are no longer mandatory</td>
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<td>for restoring endo-tx teeth. Alternatives include resin-bonded</td>
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<tr>
<td>cores. Problems include potential root perfs, costs.</td>
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<tr>
<td>• Dean, <strong>Jeansonne</strong> &amp; Sarkar (1998): In vitro central incisors</td>
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<td>posts &amp; composite core: carbon posts (C-Post) had no root</td>
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<td>fractures vs parallel posts (50%; ParaPost) and tapered posts</td>
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<td>(50%; PD posts)</td>
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<td>• <strong>Kvist</strong> (1989): Examined 852 roots with about 50% containing</td>
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<td>posts. PARLs were present on 16% of teeth with posts and 13%</td>
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<td>teeth without posts (therefore, posts do not compromise</td>
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<td>periapical healing). However, posts with only 3mm GP remaining</td>
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<td>have greater incidence of apical periodontitis. Need at least</td>
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<tr>
<td>4-5mm of GP.</td>
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<tr>
<td>• <strong>Bourgeois</strong> &amp; Lemon (1981): NS difference in apical</td>
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<tr>
<td>leakage immediate vs delayed post preps using either ZOE or</td>
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<tr>
<td>AH26 sealer</td>
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<tr>
<td>• Kleier (Feb1999JOE): Titanium posts have similar radiodensity</td>
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<td>as GP, thus difficult to dx on radiograph. Hints: Look for</td>
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<td>straightened coronal RC system, slight canal enlargement at</td>
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<td>post:GP junction, slight radiolucent gap between post:GP,</td>
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<td>surface patterns of post (ie, serrated edges)</td>
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- **Kurer** (1977): The primary function of a post is to retain the coronal restoration.
- **Standlee** (1978): For post retention: threaded > serrated > smooth sided (parallel > tapered). The longer the post, the greater the retention (should be at least the height of the crown or 9mm minimum).
- **Goerig & Mueninghoff** (1983): Recommend post be 2/3 root length with minimum of 10-15mm in length. Recommends parallel sided and cemented (not screwed). Gerstein 1964 proposed that post be no longer than 1-1.5 times crown height.
- **Guzy & Nicolls** (1979): Posts do not strengthen teeth.
- **Dickey & Harris** (1982): Ca45 leakage study. Should not prepare post space for at least one week.
- **Bachicha & Pashley** (1998): Both SS & carbon posts when cemented with dentin-binding agents (C&B Metabond or Panavia-21) exhibit less leakage than when cemented to non-dentin bonding cements (ZnPO4 or Fuji-I glass ionomer).
- **Schwartz & Walker** (1998): Eugenol containing sealer (Roths) did not differ from non-eugenol sealer (AH26) for post retention cemented by either Panavia 21 or ZnPO4. For both sealers, ZnPO4 was better than Panavia 21 (dentin bonding resin).
- **Sorenson & Martinoff** (1984): Clinical study: vertical root fracture is a common failure mode for threaded or tapered posts.
- **Funato** (1999 EDT): reports tx vertical root fx: removed post; re-tx, bond new post and root segments together with SuperBond (4-META/MMA-TBB resin). Showed reduced PARL. BUT- only 6 month follow-up.
## Perforation, Resorption, Procedural Accidents, Re-TXs, Post Removal

### General Issues

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<th>Author</th>
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<td>Fuss</td>
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### Internal Matrix

- **Lemon '92**
- **Hartwell '93**
- **Petersson '85**

### Cavit

- **Harris '76**: Reported on 245 cases of perf repair with Cavit. 89% success; need to seal immediately.

### MTA

- **Arens & Torabinejad Baumgartner '98**
- **Hartwell '98**

### Other Materials

- **Bogaerts '97**

### Perf Repair: General Factors

- Prognosis dependent upon time, size, location relative to attachment, sealability of repair material
- Jew & Weine (1982): Best prognosis for perf repair is in apical or middle thirds. Contamination with oral fluids = failure
- Fuss (1996): Apex locators are more reliable than radiographs for locating root perfs

### Internal Matrix:

- Lemon (1992) proposed the internal matrix concept for internal repair of perfs (internal matrix may consist of hydroxyapatite, decalcified FD bone, Ca(OH)2, or CollaCote)

### Cavit

- Harris (1976): Reported on 245 cases of perf repair with Cavit. 89% success; need to seal immediately.

### MTA

- Arens & Torabinejad (1996): MTA good for perf repair. Also: Pittford & Torabinejad showed MTA is good for furcal repairs
- Lee & Monsef (1993): MTA superior to amalgam or IRM in perf repairs
- Nakata & Baumgartner (1998): MTA better than amalgam in preventing leakage of *F. nucleatum* past furcal perf repairs
- Slayk & Hartwell (1998): MTA useful for furcation repair. MTA resisted displacement at 72hr better than 24hr (p<.05). No difference if MTA covered by wet or dry cotton pellet (moisture probably derived from furcal tissue). In this in vitro study, found better adaptation of MTA to perforation walls when first placed Gelfoam as an internal matrix

### Ca(OH)2 and Other Materials

- Frank & Weine (1973): Described Ca(OH)2 for perf repair due to internal resorption. But, Himel (1985) demonstrated in dogs that Ca(OH)2 caused the most inflammation
- Bogaerts (1997): Case report: used Ca(OH)2 as internal matrix and sealed perforation with EBA
- Poi & Martin (1999): Case report of perf with separated file in PDL. Did intentional reimplantation; repair, 4w stabilization with ortho wire; Ca(OH)2 medication for 1yr, then NSRCT. LC with Sealapex. Good healing with 8yr follow-up

### Removal of Posts, Ag Points, Separated Instruments:

- Review GP solvent studies (listed above)
- Crump & Natkin (1970): A separated file in a canal does not mean failure. It depends upon the location of the file and the prior debridement / shaping of the canal system
- Glick (1986): rec use of 3 Hedstrom files to braid around post
- Caulfield silver point remover (#35): looks like spoon with cut-out "V" shape to use as a crow-bar action
- Warren & Gutmann (1979): Rec Steiglitz forceps - can strengthen grip by simultaneous placement of hemostats onto Steiglitz beaks
- Krell (1984) & Ngai (1998): rec use of ultrasonic files to enlarge canal space around post/Ag points/separated instrument. Key point: often need to first start with 1/2 round bur (or Muller Pulp Chamber Bur with extra long shaft) or hand file prior to US files
- Stamos (1988): Use ultrasonic tips to remove ZnPO4 cement
- Berber & Filho (1995): Ultrasurions can reduce force needed to remove posts
- Johnson & Leary (1996): Ultrasonic removal of Paraposts takes 16 min
- Feldman & Solomon (1974): Describes use of Trephans burs, fiberoptics and hollow needles to remove separated instruments from the canal
- Fors & Berg (1986): reviews causes of iatrogenic canal obstruction and retrieval strategies
- Roig-Greene (1983): Describes the use of 0.14 diameter wire in a 25g needle to make a wire loop to retrieve instruments
- Spriggs & Gettleman (1990): Best way to use Endo Extractor (Brassler) is with at least 2mm overlap, snug fit, 5 min cyanoacrylate set
- Suter (1998) Use ultrasonics to create 1-2mm groove around instrument (or silver point). Place 21g tubing over instrument. Insert Hedstrom into tube and engage instrument with CW rotation. Pull on handle of the Hedstrom.
- Williams & Bjorndal (1983): Rec use Masserann trephin drill to 1/2 post length and then use ultrasonic vibrating tip for 30sec - 10 min to remove post
- Ibarrola (1993): Chloroform greatly facilitated removal of plastic Thermofil carriers in 19 of 20 cases
- Eleazor & O'Connor (1999 JOE March): Can use sharp bevel on hypodermic needle to enlarge canal openings and cut around broken instrument. Gave Table of needle guage #s corresponding to ISO sizes. Can bond with cyanoacrylate to retrieve files etc
- Bertrand (1997 JOE): Hand files with chloroform can remove Thermofil obturators

### Removal of Thermafill (Johnson 1978)
- Tulsa: Work down obturator as far as possible with small files and chloroform. And then insert #20 or #25 Profile .04 taper at 2,000 RPM to spin down further and hopefully withdraw the obturator
- Ibarrola (1993): Chloroform greatly facilitated removal of plastic Thermofil carriers in 19 of 20 cases
- Consider using Glick method of braiding Hedstrom files
- Wolcott, Himel & Hicks (1999 JOE p762): Used System B HeatSource (faster than chloroform): Use heated System B plugger (225C) to insert 10-15mm for 5-8 sec. Then insert #50 and #55 NiTi files on B & L sides of Thermofil obturator (while GP is still thermoplasticized). Apply firm apical pressure and CW rotration, remove files and carrier together. (Be careful: melting point of plastic carrier =300C)

<table>
<thead>
<tr>
<th>Reference</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>Burke 1976</td>
<td>Avulsion: Tx avulsed teeth with Ca(OH)2 after splinting in order to minimize inflammatory resorption</td>
</tr>
</tbody>
</table>
| Gartner & Mack ’76 | **Internal and External Resorption**
- Gartner & Mack (1976, JOE): Classic: Diff dx of Int vs Ext: Internal: sharp margins, canal not observed inside lesion, symmetrical, uniform density, doesn’t move on shift shot. External: irregular margins, can see canal thru lesion, asymmetrical, variable radiodensity, moves on shift shot
- Tronstad (1988 EDT): Internal resorption is maintained by RC infection coronal to site of the lesion
- Frank (1981): Describes the external-internal progressive resorptive lesion. Need to completely locate, debride & seal.
- Caliskan (1997 EDT): Reviewed 28 cases of internal resorption. >90% success with non-perforating resorption using 1w of Ca(OH)2 followed by single cone LC with CRCS sealer; resorptive space filled with thermoplasticized GP (or amalgam in some cases). In contrast only 25% success in perforating internal resorptive cases
- Wedenberg (1985 EDT): Internal resorption associated with pulpal inflammation and the presence infected RC systems
- England (1977 JOE): Rec use of Ca(OH)2 - Barium sulfate paste in canals to visualize whether internal resorption has perforated
- Rick Schwartz, Bill Walker (1999 JADA): Case reports of MTA for vertical root fx (with post), apexification, perf repair, and repair of internal resorption
- External root resorption caused by: trauma, periradicular inflammation, ortho, bleaching |
<p>| Frank &amp; Torabinejad 1998 | Describes extracanal invasive resorption. Radiographic appearance similar to external resorption, vital teeth, WNL percussion. Dx finding is irregular area of resorption separated from canal system. Tx depends on site of lesion (coronal vs mid vs apical 1/3s). |
| Absi &amp; Addy 1987 | Teeth with dentinal hypersensitivity have greater density and diameter of tubules |</p>
<table>
<thead>
<tr>
<th>Author(s)</th>
<th>Title</th>
<th>Notes</th>
</tr>
</thead>
<tbody>
<tr>
<td>Brannstrom 1967</td>
<td>Heat causes inward fluid movement in tubule and cold causes outward fluid movement. <em>It's cold out</em></td>
<td></td>
</tr>
<tr>
<td>Fusayama 1988</td>
<td>Bonded composites can reduce dentinal hypersensitivity. Confirmed by Brannstrom JADA. And by Nordenvall &amp; Brannstrom 1980</td>
<td></td>
</tr>
<tr>
<td>Mullaney &amp; Howell 1970</td>
<td>Did not find nerves in necrotic pulps. Concluded that pain upon entering necrotic teeth may be due to apical compression.</td>
<td></td>
</tr>
<tr>
<td>Chen 1997 EDT</td>
<td>Case report: spontaneous throbbing left max 2nd premolar thru upper frontal face to frontal parietal area. NSRCT did not resolve pain. MRI imaging revealed sinus turbinate hypertrophy and hazy material on floor of sinus. Final dx: pulpitis with max sinusitis and chronic rhinitis. Dx difficult due to multiple etiology. Referred pain</td>
<td></td>
</tr>
<tr>
<td>Negm 1994 JOMS</td>
<td>Intracanal diclofenac and ketoprofen effective analgesics for controlling endo pain</td>
<td></td>
</tr>
<tr>
<td>Rogers &amp; Johnson 1999</td>
<td>Intracanal application of ketorolac (3mg) and dexamethasone (0.4 mg) were better analgesics than plbo at 12h; ketorolac also &gt; plbo at 24h; Oral ibuprofen ns different from plbo. N=48 pts with vital cases NSRCT C&amp;S. Pain by VAS.</td>
<td></td>
</tr>
</tbody>
</table>

**Air Emphysema**
- Shovelton (1957) reported 13 cases of air emphysema
- Eleazor & Eleazor (1998): Air pressure applied to canals may produce emphysema. Esp when instrument to larger sizes. Stropko syringe produced 10% of pressure seen with std air syringe.

**Local Anesthesia Studies:**
- Denunzio (1998 JOE): Clinical aid: Mark suggests intracanal use of topical local anesthetic placed with files (topicals often contain 20% benzocaine)
- Buckley & Ciancio (1984): Perio flaps with 2% Lido with 1:100,000 epi had about twice as much blood loss as flaps tx with 2% Lido with 1:50,000 epi. Contrast this study to Bou Dagher (1997) (Different outcome measures: anesthesia vs blood loss)
- Bou Dagher & Yared (1997): Compared degree of anesthesia: 2% Lido with 1:50,000 = 2% Lido with 1:100,000 = 2% Lido with 1:80,000 epi. Contrast this study to Buckely 1984 (Different outcome measures: anesthesia vs blood loss)
- Dunsky & Moore (1984): Duration of etidocaine was the same as the duration of bupivacaine
- Birchfield & Rosenberg (1975): Pressure is the key for intrapulpal anesthesia
- Campbell & Mecuri (1979): Horner's syndrome (symp cervical block) LA into pterygomandibular space into lateral retropharyngeal spaces into danger space
- Sved & Wong (1992): Most common side effect of max nerve blocks = 36% diplopia
- Dryden (1993): Case report: Gow-Gates injection = twitching, burning, diplopia, ptosis. Due to retrograde flow into cavernous sinus. Resolved in 20 min
- Frommer & Mele (1972): Molar sensation after IAN could be due to separate foramen for mylohyoid nerve (occurs in 30% popln)
- Kleier & Deeg (1983): Extraoral infraorbital block: 90% effective and safe
- Loetscher & Melton (1988): PSA blocks 88% max 1st molars. Get addnl 5% blocked with mesial infiltration
- Lindorf 1979 (OOO): Can get rebound effect (reactive hyperemia) on blood flow after injection with vasoconstrictors

**Stabident Injection:**
- Parente & Weller (1998): Stabident effective as adjunct in 89% pts in pain after access prep (after IAN or max infiltration injections). Stabident was more successful in mandible vs maxilla (91% vs 67% of teeth) LA = 2% lido with 1:100k epi
<table>
<thead>
<tr>
<th>Replogle &amp; Reader (1999): Stabdent intraosseous injection of 2% Lido with 1:100,000 epi increased heart rate in 67% of pts, with mean increase from 69 to 97 bpm</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>PDL Injection:</strong></td>
</tr>
<tr>
<td>• Kim (1986): Proposed that PDL injection works by vasoconstriction of blood flow and therefore, should only be used for endo &amp; extraction - not to be used for vital teeth for restorative treatment.</td>
</tr>
<tr>
<td>• Pashley (1986): PDL is an intraosseous injection with significant CV effects. Confirmed by Walton (1986).</td>
</tr>
<tr>
<td>• Walton &amp; Garnick (1982): PDL injection does not harm the ligament, so it is not a ligament injection! Requires backpressure.</td>
</tr>
</tbody>
</table>

| Triazolam 0.25mg reduced anxiety better than diazepam 5mg or plbo in endo patients. Confirmed by Kaufman, Hargreaves & Dionne (1993). |

| Pre-op flurbi reduced post-op pain. ALSO, Pulpectomy alone reduces post-op pain |

<table>
<thead>
<tr>
<th>Are Steroids Effective?</th>
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<tbody>
<tr>
<td>• Glassman &amp; Krasner (1989 OOO) Lg doses of dex (12 mg po) in vital asymptomatic cases are better than plbo for reducing post-appt pain</td>
</tr>
<tr>
<td>• Chance &amp; Lin (1987): Cortisone on paper point in vital cases reduced pain (but- did it induce bacteremia via paper point?)</td>
</tr>
<tr>
<td>• De Deus &amp; Han (1967): Placed [14C]-cortisone on pulp and found in liver &amp; kidney. Pharmacokinetics show absorption from pulp</td>
</tr>
<tr>
<td>• Fava (1998 IEJ): N=60 teeth with AAP: Compared intracanal Ca(OH)2 vs solution of hydrocortisone-polymyxin-neomycin (Otosporin). Called at 48hr: NS difference in pain (no plbo group and no VAS assessment).</td>
</tr>
<tr>
<td>• Rogers &amp; Johnson (1999 JOE): Intracanal application of ketorolac (3mg) and dexamethasone (0.4 mg) were better analgesics than plbo at 12h; ketorolac also &gt; plbo at 24h; Oral ibuprofen ns different from plbo. N=48 pts with vital cases NSRCT C&amp;S. Pain by VAS.</td>
</tr>
<tr>
<td>• Kaufman (1994 JOMS): Intraligamentary injection of slow-release methylprednisolone effective reducing post-endo pain</td>
</tr>
<tr>
<td>• Krasner &amp; Jackson (1986 OOO p187): randomized plbo study (n=50): oral dex &gt; plbo for reducing post-endo pain</td>
</tr>
<tr>
<td>• Marshall (1984 JOE &amp; 1993 JOE): injectable dex better than plbo for reducing post-endo pain. No evidence of increased infections, fever. Must be confident that pain is due to inflammation and not due to infection</td>
</tr>
<tr>
<td>• Moskow (1984 OOO): intracanal steroids reduced pain. Study used vital cases</td>
</tr>
<tr>
<td>• Gallatin (JOE 1998 24:280): intraosseous injection of Depo-Medrol (1ml = 4-mg) sig reduced pain in irreversible pulpitis</td>
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<tr>
<th>Differential Dx of Non-Odontogenic Pain:</th>
</tr>
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<tbody>
<tr>
<td><strong>Referred Pain</strong></td>
</tr>
<tr>
<td>• Reeh &amp; ElDeeb (1991) Muscle trigger point referred to tooth and mimicked endo involvement.</td>
</tr>
<tr>
<td>• Kleier (1985): Muscle trigger point referred to tooth and mimicked endo involvement</td>
</tr>
<tr>
<td>• Drinnan (1987 DCNA): MI. ~10% MI cases have pain referred to mandible (Sandler (1995 JADA). Coronary insufficiency referring to mandible Batchelder (1987)</td>
</tr>
<tr>
<td>• Sharav (1984): Acute dental pain can be referred to opposite arch on same side (eg., Left Max = Left mand)</td>
</tr>
</tbody>
</table>

| **Neuropathic Pain** |
|• Dumas (1999 OOO): trigeminal sensory neuropathy. Sensory disturbance is ominous sign. MOA = CNS metastatic neoplasia (esp men>60), multiple sclerosis. Often rapid onset, ~50% report pain, differential of symptoms includes post-endo pain |
|• Francica & Brickman (1988): Trigeminal neuralgia (=tic douloureux) referring to endodontically treated teeth; lancating shooting pain; cabamazepine |
|• Vickers (1998): Atypical odontalgia. Can be phantom tooth pain, RSD, psychological |
|• Drinnan (1987 DCNA): AFP |
### Cancer
- Glaser '97: Intermittant tingling or numbness of lower lip. Numb lip most common feature of metastatic CA
- Kant (1989): Malignant mediastinal lymphoma as mandibular pain
- Selden (1998) who found metastatic carcinoma as PARL on mandibular molar; later developed paresthesia.
- Todd (1987 JOE) Hx of previous CA - reported metastasis occluding blood flow producing necrosis. Think of this when can find no obvious etiology

### Other:
- Aral (1997): Eagle's syndrome
- Drinnan (1987 DCNA): Manchausen's syndrome

### Other:
- Ratner & Langer ‘86
- Drinnan ‘87


### Seltzer & Bender 1985
- Pain on biting suggests PDL inflammation that is due to either necrotic pulp or irreversibly inflamed pulp.

### Seltzer & Bender 1963
- Hx of previous pain in tooth indicates moderate-to-severe pulpitis or necrosis 80% of the time. Therefore, should expect dx of irreversible pulpitis when this hx combined with vital testing responses

### Kier & Walker 1991
- 2 case reports of heat sensitivity after NSRCT. Critical point: reproduce pt's cc. Due to missed canals. NSRCT Re-Tx resolved cc. So, do not rule out NSRCT-tx teeth as source of pt's thermal discomfort.

### Trowbridge & Franks 1980
- Cold testing of teeth works by outward hydrodynamic fluid flow

### Endo Emergencies: Open vs Closed

#### Keep it Closed:
- Bence & Meyers (1980): Recommended that leave tooth open only as last resort (in contrast to August, B&M reported hat 46% had to be re-opened).
- Weine (1975 OOO): Teeth left closed had fewer exacerbations.
- Simon (1982 JOE) leaving teeth open can lead to foreign body reaction of material forced into periapical tissue
- Seltzer & Naidorf (1984 JOE): Reasons why not leave tooth open include additional bacterial contamination, contamination with food debris or blockage of canals, unnecessary follow-up appts to close the tooth
- Natkin (1974 DCNA): Regardless of how much purulence has drained tooth can be dried and safely closed if etiologic factors have been alleviated
- Sundqvist (1976): REMEMBER: Apical periodontitis can only be detected in teeth with bacteria present in canal systems. Open cases will become infected. Necrotic, but sterile teeth have no signs of PARL. In contrast, necrotic and infected teeth showed PARLs. Also, probability of pain increased with # bacterial species (esp when >6); suggests bacterial synergism is important virulence factor.

#### Open is OK:
- August (1982): Teeth left open can be completely instrumented and closed (95% remain closed).

### Chester & Selman ‘68
- Elliot ‘88
- Peters ‘80

### Trephination
- Chester & Selman (1968): Describes trephination to relieve pain.
- Elliot & Holcomb (1988). Trephination (#3 spreader) in pts with PARLs: 0% mod-severe pain vs 25% mod-severe in no-tx group
- Moos et al (1996) reports more pain after pulpectomy & trephination. Routine trephination is not justified
• Peters (1980 JOE): no need to prophylactically trephinate since incidence of post-endo pain is so low

### Predictors of Post-Endo Pain

- **Walton & Fouad (1992):** Best predictors are pre-op pain or swelling
- **Torabinejad (1988):** An incidence of 68% is related to the presence of periapical pathology
- **Harrison & Baumgartner (1981):** Post-endo pain: Most likely to occur in first 24hr. More likely to develop in pts with inter-appt pain
- **Creech & Walton (1984):** Magnitude of post-endo pain is related to magnitude of pre-endo pain
- **Jostes & Holland (1984):** Magnitude of post-endo pain is related to magnitude of pre-endo pain
- **Genet (1987 IEJ):** Predictors of post pain: pre-op pain, necrotic, PARL, females
- **Torabinejad & Kettering (1988):** Magnitude of post-endo pain is related to magnitude of pre-endo pain
- **Marshall & Walton (1984):** Magnitude of post-endo pain is related to magnitude of pre-endo pain

### Flare-ups

- **AAE Glossary:** an acute exacerbation of a periradicular pathosis after the initiation or continuation of NSRCT
- **Morse & Koren (1986):** Flare-up rate = 20%/! (1963-1970) Highest in females and pts<20. Esp max laterals, mand 1st premolar & Ig PARLs
- **Tropo (1990):** Flare-up incidence = 2.6%. No difference between formocresol = Ledermix = Ca(OH)2 as intracanal medicaments
- **Tropo (1995 IEJ):** Incidence = 13% (3 of 22) in 1-step re-tx with AP. 0% flare-up in teeth without AP. ALSO: Torabinejad ‘88 re-tx has sig more flare-up
- **Walton & Fouad (1992):** Incidence 3.1% (= unscheduled visit for severe pain/swelling). Pre-op pain = best predictor
- **Torabinejad (1988):** n=2,000 Predictors of flare-ups include cases with no or small PARL, re-tx cases, pts with hx pre-op pain or allergies, female, age 40-59 (*italics* = large significant difference). Lower risk of flare-ups: Large PARLs, sinus tracts, analgesics. Re-tx have significantly more flare-ups than initial NSRCT, and Tropo ‘95 argues that should re-tx in multiple appts.
- **Imura & Zoula (1995):** Flare-ups (1.58%) correlated with: PARL, pre-op symptoms, re-tx cases (NB: PARL in contrast to Torabinejad)
- **Kerekes & Tronstad (1979):** no change in prognosis for success if case has flare-up
- **Harrington & Natkin (1992 DCNA):** necrotic teeth more likely to have flare-ups; especially if debris is extruded. [NB: Crown-down (Ruiz-Hubbard & Gutmann 1987 JOE) and balanced-force (McKendry 1990 JOE) extruded less debris than step-back filing]
- **Georgeopoulos (1993 IEJ):** over-instrumentation may lead to flare-ups
- **Seltzer & Naidorf (1985 JOE):** Proposed 7 etiologic factors for flare-ups, including local adaptation syndrome (new factor exacerbates chronic inflammation); inc PA pressure, introduction certain bugs, immunological rxn, psych, etc
- **Rimmer (1993 JOE):** defined flare-up index to permit comparison across studies

### Causes of Flare-Ups

- **Seltzer & Naidorf (1985 JOE):**
  - Overinstrumentation
  - Overmedication
  - Debris forced into periapical tissue
  - Incomplete removal of pulp
  - Recrudescence of CAP
  - Over-irrigation
  - Hyperocclusion
  - Root fracture
  - Another tooth
  - Pasteur effect (ie, overgrowth of facultative anerobes (Naidorf, 1977 JOE))

### Evidence that prophylactic antibiotics have no significant benefit on post endo flare-ups or pain:

San Antonio Guide to the Endodontic Literature  version 2.34  Page 29
Walton & Chiapinelli (1993): n=80 Necrosis/CAP Pen VK (AHA regimen) = Plbo = No Tx for post-op pain & swelling
Torabinejad (1994) n=588 Obturation study: PLBO = Pen VK 500 mg = Erythromycin 500mg = (IBU + Pen VK) = (Methylpred + Pen VK) for reducing post-obturation pain;
Walton & Fouad (1992 JOE): 3% incidence of flare-ups. This prospective RCT found no benefits to prophylactic antibiotics for reducing flare-ups
Fouad, Rivera & Walton (1996): Pen VK = Plbo for reducing symptoms and recovery of localized AAA. Indiscriminate use of antibiotics is unjustified.
Ranta ('88 Scand J Infect Dis): Reported that neither short-term (1 week) nor long term (3 months!) tx with Pen VK had any effect on healing of periapical lesions!

Studies that seem to show an effect:
Morse & Furst (1987): (one of many articles by Morse on flare-ups) Necrotic with PARL 1-day tx with AHA protocol (high dose) Pen VK reduced flare-ups from 20% to 2%. Best results teeth with very large PARLs. But, baseline flare-up rate = 20%!
Mata & Morse (1985 OOO): necrosis & AP: pen 250 mg reduced flare-ups
Torabinejad (1994) n=588 Instrumentation study: Pts with Moderate-severe pain in first 48hr: Pen VK & Erythro & Methylpred + Pen VK were all better than plbo for reducing post-instrumentation pain

Cunningham
Rosenberg '88
Walton '84

Treating Flare-ups

1. Adjust Oclusion to Reduce Post-Endo Pain:
   - Cunningham: rec adj occlusion
   - Rosenberg (1998 JOE): Adj occlusion helps relieve pain (esp with + percussion pre-op)
   - Creech & Walton (1984 JADA): Adjust occlusion only as needed; prophylactic adjustment does not reduce pain
   - Jostes & Hikkabd (1984 JOE): Only adj occlusion when needed; adj occ on 50% teeth with "biting" pain but saw no benefit

Re-enter RC system and debride
   - Open vs closed (Bence & Myers; Seltzer & Naidorf; August)

Establish drainage
   - Apical penetration for intracanal drainage
   - Trephination (Elliot #3 spreader; Peters)

Evaluate for analgesics
   - 3-D strategy - Hargreaves

Evaluate for antibiotics
   - 4 General indications (Harrison; Baumgartner):
     - rapid increase S&S; anatomical danger zone; disease/drug that compromises immune status; systemic involvement of infection (eg., lymphadenopathy, fever, malaise)

Evaluate for Steroids
   - Marshall & Walton;
### Pulp Biology

<table>
<thead>
<tr>
<th>Author(s)</th>
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<tbody>
<tr>
<td>Mark Bishop 1990</td>
<td>Pulp contains lymphatic vessels (cats). Also reported by Bernick 1977.</td>
</tr>
<tr>
<td>Stanley 1981</td>
<td>Irregular secondary dentin can be viewed as mineralized scar. Forms in humans at 1.5 um/day.</td>
</tr>
<tr>
<td>Stenvik 1972</td>
<td>Pulpal hydrostatic (= tissue) pressure normally is 5-20 mm Hg and can increase up to 60 mm Hg locally in response to inflammation. Note that this increase is local and not pulp-wide. Also reported by Smulson (1984 DCNA).</td>
</tr>
<tr>
<td></td>
<td><strong>Cell Adhesion Molecules (CAMs)</strong></td>
</tr>
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<td></td>
<td>- Tasman (1999 JOE): CAMs expressed on vacuature act to regulate migration of leukocytes into inflamed tissue. BV in pulp with dx irrev pulpitis have significantly greater expression of CD102 CAM than blood vessels in normal pulp.</td>
</tr>
<tr>
<td>Heyeraas 1992</td>
<td>Proposed peptidergic afferents innervating tubules respond to trauma with axonal reflex leading to vasodilation leading to increased tissue pressure leading to increased outward (protective) flow of dentinal fluid</td>
</tr>
<tr>
<td>Fristad 1995</td>
<td>Denervation reduces influx of inflammatory cells into pulp.</td>
</tr>
<tr>
<td>Kindlova 1965</td>
<td>Blood supply to mucosa is by vertical blood vessels. Therefore, horizontal incisions (for sx or IND) will bleed more than vertical incisions.</td>
</tr>
<tr>
<td>Garberoglio &amp; Brannstrom 1976</td>
<td>Dentinal tubules density = 40-70,000/um2. At DEJ, 1% of surface = tubules. At PDJ, 22% of surface = tubules.</td>
</tr>
<tr>
<td>Papa &amp; Messer 1994</td>
<td>Although previous studies believed that RCT dried out teeth, P&amp;M reported that vital dentin contained 12.4% moisture whereas root filled teeth contained 12.1% moisture.</td>
</tr>
<tr>
<td>Sedgley &amp; Messer 1992</td>
<td>Root-filled teeth are not more brittle than vital teeth.</td>
</tr>
<tr>
<td>Berneck &amp; Nedelman 1975</td>
<td>Older pulps have reduced # blood vessels and nerve fibers.</td>
</tr>
<tr>
<td>Johnson 1985</td>
<td>Nerves terminate 100um in dentinal tubules. Reason why EPT prone for false negatives in developing teeth: C fibers innervate first and the A-delta come in later. This was challenged by Peckham &amp; Torabinejad 1991 (who found Ad during root development).</td>
</tr>
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### DIAGNOSIS AND DIAGNOSTIC TESTS

<table>
<thead>
<tr>
<th>Author(s)</th>
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<tbody>
<tr>
<td>Seltzer &amp; Bender '63 Tyldesley '70</td>
<td><strong>There is a poor correlation between clinical symptoms and pulpal histopathology</strong></td>
</tr>
<tr>
<td></td>
<td>- Seltzer &amp; Bender (1963)</td>
</tr>
<tr>
<td></td>
<td>- Dowden 1969;</td>
</tr>
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<td></td>
<td>- Garfunkel 1973</td>
</tr>
<tr>
<td></td>
<td>- Baume (1970 OOO): impossible to determine histology from dx</td>
</tr>
</tbody>
</table>
Cold Test
- White & Cooley (1977): DDM and Hot water gives largest temperature changes. 5-8 sec is enough to get a response
- Fuss & Trowbridge (1986): DDM (dichlorodifluoromethane = -50°C), CO2 and EPT are equally reliable as pulp tests in adults
- Petersson (1999 ETD): Compared cold test (ethyl chloride), heat test (GP) and EPT (Analytical Tech) vs Gold Stnd (access and eyeball) for vitality. N=59. Petersson's results are as follows:
  - Probability of negative test being necrotic pulp: 89% cold test, 88% EPT and 48% hot test
  - Probability of positive test being vital pulp: 90% cold test, 84% EPT and 83% hot test
- Augsberger & Peters: CO2 snow better than ice or skin refrigerant
- Jones (1999): Measured mand incisor temp when apply DDM with various applicators. Best reduction in temp occurred when spray on large cotton pellet (vs small cotton pellet, wooden stick cotton tip applicator or a cotton roll)
- Peters (1983 & 1986 JOE): CO2 snow does not crack enamel (even after 2 min) and is a safe test to use
- Rickoff & Trowbridge (1988 JOE): Tested teeth with hot GP and CO2 snow. No pathosis induced. Temp did not change much inside tooth (concluded hydrodynamic theory) "Its cold out"

EPT
- Seltzer & Bender (1963): Negative EPT = complete or partial necrosis 97.7% of the time
- Abdel Wahab & Kennedy (1987): Rate of electrical current increase affects sensation. Rec slow current increase.
- Anderson & Pantera: EPT gives same result when patient holds handle vs traditional non-gloved method.
- Pantera (1992 JOE): Can use dental instruments to contact under crown and bridge between tooth and EPT probe
- Fuss & Trowbridge (1986 JOE): EPT = DDM = CO2 for reliability. BUT- EPT not as reliable in young patients
- Fulling & Andreasen (1976 Scand JDR): EPT unreliable in developing teeth; CO2 snow more reliable in these teeth
- Dummer (1986 IEJ): Analytical technologies EPT easier to use; but still no consistent threshold value
- Myers (1998 JOE): EPT current can travel between adjacent teeth with amalgam contacts. Possible false +

Percussion Test
Klausen (1985):
Hargreaves (1994 OOO)

Stashenko 1995
PARL can develop before pulp totally necrotic in rats. May explain presence of + vitality test in teeth with PARLs. ALSO: Nielseno (1999) case report of carious lesion on vital first molar with PARL in 23yo pt (tooth was asymptomatic & tested vital). PARL healed after caries removal and IRM base and amalgam.

Trope '97 & '97
- Chandler & Sundqvist (1999): Case report of a pt with 2 teeth that responded to pulp testing but had PARLS. Laser doppler showed pulpal vitality. Dx periapical cemental dysplasia (cementoma). Also reported by Wilcox & Walton (1989) who did NSRCT on tooth with PARL- no response 2yr after re-tx; biopsy = cementoma.
- Mesaros, Trope, Maixner (1997 IEJ): Laser doppler (Moor DRT4 instrument) detected vital vs necrotic vs empty pulp chambers
- Mesaros & Trope (1997 EDT): Case report: traumatic injury to 8yo boy's two max centrals; only one weakly + to C02 ice at 76 days; but Laser Doppler indicated both were vital. Endo tx not performed and teeth developed normally
- Ingolfsson & Tronstad (1994 EDT): Laser doppler more accurate than EPT for necrotic pulps (91% vs 64%)
- Matthews (1993 IEJ): problems with Laser Doppler: not measured in absolute units, output may not linearly related to PBF

Trope '97 & '97
- Gutmann 1995 and Cox 1991
Rec take another WL film if file tip is >1mm from apex
<table>
<thead>
<tr>
<th>Author(s)</th>
<th>Year</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>Kelly 1988 JOE</td>
<td>Presented cases where sinus tract was tracked to adjacent teeth. Always track sinus tracts</td>
<td></td>
</tr>
<tr>
<td>Selden 1999</td>
<td>Also Selden 1970; 1974; 1977. Endo-Antral Syndrome: max post teeth producing sinusitis. Described 4 cases. Look for necrotic-PARL with faint radiopaque mass bulging into sinus space over apex. Most cases respond to NSRCT. Positive palpation in buccal vestibule</td>
<td></td>
</tr>
<tr>
<td>Ikeda &amp; Suda 1998 JOE</td>
<td>Human microneurography evaluation of pulp responsiveness: Recorded neurons at mental foramen (mand ant teeth). Normal teeth: Mean Laser Doppler = 42 units: EPT, cold (DDM), heat (GP @ 70-90C) all evoked discharges and painful sensation &quot;Pathological&quot; teeth: Mean Laser Doppler = 9 units (same value as for obturated teeth): EPT, cold (DDM), heat activated 80% units, BUT produced no sensation until instrumented canal with #10 Interpreted data to suggest that negative EPT/thermal can have false negative due to lack of perception. Lack of perception could be due to 1) destruction of Adeltas; inability of C fibers to follow EPT train (100Hz); Also reported that cold stimuli (DDM) was more useful to activate pulp nerves than heat test. Also reported that laser doppler will have false negative when coronal pulp is chronically inflamed and apical stump is vial</td>
<td></td>
</tr>
<tr>
<td>Brynoff 1970</td>
<td>Dx accuracy is increased when take 2nd radiograph: 73% accurate with 2 radiographs and 87% accurate with 3 radiographs</td>
<td></td>
</tr>
<tr>
<td>Cameron 1993</td>
<td>Review cracked tooth: Most common tooth = mand 2nd molar. Most common cc = pain upon chewing and pain with cold. Full coverage. Also reviewed by Johnson 1984</td>
<td></td>
</tr>
<tr>
<td>Diaz-Arnold, Wilcox 1996</td>
<td>In vitro study to measure pulp vitality by oxygenated hemoglobin</td>
<td></td>
</tr>
<tr>
<td>Dodd, Dodds, Holcomb 1984</td>
<td>Case report of chronic sinusitis due to over-extended silver point. Sinusitis resolved when removed silver point.</td>
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<tr>
<td>Hill 1986</td>
<td>Describes transillumination as a useful adjunct</td>
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</tbody>
</table>

### Pulpotomy

<table>
<thead>
<tr>
<th>Author(s)</th>
<th>Year</th>
<th>Description</th>
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</thead>
<tbody>
<tr>
<td>Cvek 1978</td>
<td>Advocated partial pulpotomy (removing 1.5-2 mm pulp) even 72hr after traumatic exposure. Remember that need remove blood clot (Schroder 1971). Cvek pulpotomy = 95% healing 3-15 yr follow-up.</td>
<td></td>
</tr>
<tr>
<td>Mass 1993</td>
<td>Reported 91% success rate in doing Cvek partial pulpotomy in young, posterior, symptom-free teeth with carious exposures.</td>
<td></td>
</tr>
<tr>
<td>Schroder 1971</td>
<td>Demonstrated that you need to remove the blood clot after a partial pulpotomy procedure, since it reduces healing success. With no clot, got 76% healing rate.</td>
<td></td>
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</tbody>
</table>

### Coronal Microleakage

<table>
<thead>
<tr>
<th>Author(s)</th>
<th>Year</th>
<th>Description</th>
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</thead>
<tbody>
<tr>
<td>Saunders &amp; Saunders 1994</td>
<td>Coronal microleakage is important cause of RCT failure.</td>
<td></td>
</tr>
<tr>
<td>Ray and Trope 1995</td>
<td>Retrospective study of 1,010 NSRCT, evaluating periradicular radiographic status. Failure was attributed more to technical quality of coronal</td>
<td></td>
</tr>
</tbody>
</table>
restoration than to quality of RCT. ALSO: Allen (1989 JOE): teeth restored after NSRCT had better success rate

<table>
<thead>
<tr>
<th>Cox 1987</th>
<th>Examined effects of materials placed against the pulp. Concluded that seal was more important than the material itself.</th>
</tr>
</thead>
</table>

**Coronal Microleakage Studies:**

- Swanson & Madison (1987): Demonstrated that it took only 3 days for coronal leakage of a tracer dye to reach apex.  
- Madison & Wilcox (1988): Emphasized importance of coronal seal. 3 day dye leakage  
- Magura (1991): Reported that it took 3 months for bacterial coronal leakage to percolate through RCT filling. Suggest need to re-tx if RCT tooth is not restored within 3months.  
- Khayat & Torabinejad (1993): Demonstrated recontamination of obturated root canal systems when bacteria placed in natural saliva within 30 days.  
- Alves (1998): Compared bacterial (Campylobacter rectus, Peptostreptococcus micros, Fusobacterium nucleatum, Prevotella intermedia) leakage of LC obturated canals with post-space prep to endodontin. Endotoxic leakage faster than bugs (Means: 23 days vs 62 days)  
- Torpe (1995): Endotoxin penetration of coronally unsealed NSRCT teeth <21 days. Emphasizes importance of coronal seal  
- Based on bacterial and endotoxin leakage studies of Torabinejad, Khayat, Alves & Trope, you should re-tx leaking NSRCT case if open > 3 weeks

**Orifice Sealers (to preserve NSRCT regardless of restoration’s microleakage):**

- Nayyar & Walton (1980): Reported on crown buildups using coronal 2-4mm of RC and packing with amalgam. 4yr follow-up of 400 teeth. Panavia EX or use of Amalgambond helps reduce microleakage.  
- Saunders & Saunders (1990): Suggested place glass ionomer material over canal orifice and pulpal floor to minimize coronal microleakage.  
- Wolcott, Hicks & Himel (1999 JOE): Rec use of Vitrebond as orifice sealer material  
- Torabinejad & Chivian (1998): Rec use of MTA to seal RC orifices

**Taylor & Jeansonne 1997**

Coronal leakage cumulatively reduced by removal of smear layer, use of AH26 and vertical compaction. Confirmed by Economides (1999) who showed that microleakage in AH26 was reduced by removal of smear layer (but that removal did not improve sealing ability Roths 801)

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**ENDO-ORTHO**

Heithersay 1973 1st description of vertical root extrusion. Note that Lemon 1982 recommends 1 month stabilization for every 1 mm moved.

<table>
<thead>
<tr>
<th>Ingber 1975/76</th>
<th>Described forced eruption, tx perio defects, importance of biological width</th>
</tr>
</thead>
</table>

Mattison & Delivanis 1984 Endo tx teeth can be moved orthodontically without increased risk of resorption.

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**ENDO-PERIO**

**Correlation Between Endo and Perio status is controversial:**

- In general, there is good consensus that endo pathology can lead to perio pathology  
- However, it is controversial whether perio path can induce endo pathology:
<table>
<thead>
<tr>
<th>Studies that found a correlation:</th>
</tr>
</thead>
<tbody>
<tr>
<td>- Seltzer &amp; Bender (1967 OOO): perio can be a factor contributing to failure of NSRCT</td>
</tr>
<tr>
<td>- Wong (1989 EDT): Root planing &amp; citric acid tx can cause pulpal inflammation &amp; open dentinal tubules (small n, though)</td>
</tr>
<tr>
<td>- Torabinejad &amp; Kigen (1985): found no correlation (ie, found teeth with perio path but normal pulpal responsiveness)</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Studies that found no correlation between perio path inducing endo pathology</th>
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<tbody>
<tr>
<td>- Mazur &amp; Massler (1964): Found no correlation between severity of periodontal disease and histological status of the pulp</td>
</tr>
<tr>
<td>- Bender &amp; Seltzer (1972): Found no association between pulp disease and 1) probing depths; 2) extent of bone loss; 3) extent ot perio disease</td>
</tr>
<tr>
<td>- Torabinejad &amp; Kigen (1985): found no correlation (ie, found teeth with perio path but normal pulpal responsiveness)</td>
</tr>
<tr>
<td>- Bergenholtz (1984 J Perio): perio-prosth: 15% of abutment teeth were necrotic (vs only 3% non-abutment teeth). Rec routine pulpal testing of prosth abutment teeth.</td>
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<tbody>
<tr>
<td>Primary pulpal lesion</td>
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<tr>
<td>Primary pulpal lesion / secondary perio lesion</td>
<td></td>
</tr>
<tr>
<td>Primary perio lesion</td>
<td></td>
</tr>
<tr>
<td>Primary perio lesion / secondary pulpal lesion</td>
<td></td>
</tr>
<tr>
<td>Combined pulpal &amp; perio lesion</td>
<td></td>
</tr>
<tr>
<td>Concomitant pulpal-perio</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Trope 1988 JOE</th>
<th>Possible test for differential perio-endo dx: 30-60% Perio abscesses have spirochetes, but only 0-10% endo abscesses have spirochetes</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hiatt 1977 J Perio</td>
<td>Reviewed causes and differential dx of endo-perio.  Prognosis of endo-perio lesion is ultimately the periodontal prognosis!</td>
</tr>
</tbody>
</table>

**BLEACHING**

<table>
<thead>
<tr>
<th>Dahlstrom, Hiethersay 1997</th>
<th>OH- radicals formed during thermocatalytic bleaching could be MOA for PDL breakdown and resorption.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Spasser 1961</td>
<td>Described walking bleach technique.</td>
</tr>
<tr>
<td>Madison &amp; Walton 1990</td>
<td>Cervical resorption due mostly to heat in the thermocatalytic bleaching technique.</td>
</tr>
<tr>
<td>Smith &amp; Cunningham 1992</td>
<td>Recommended seal canal orifices with 2mm Cavit prior to walking bleach.</td>
</tr>
<tr>
<td>Freccia &amp; Peters 1982</td>
<td>Walking bleach produces same results regardless of use of heat.</td>
</tr>
<tr>
<td>Lewenstein 1994</td>
<td>Recommends use of sodium perborate instead of 30% H2O2 since H2O2 reduces microhardness of both dentin and enamel.</td>
</tr>
<tr>
<td>Walton &amp; Odell 1982</td>
<td>Tetracycline stains are more in dentin than in enamel (therefore, vital bleaching is temporary).</td>
</tr>
<tr>
<td>Tittley &amp; Torneck 1993</td>
<td>H2O2 in walking bleach technique can inhibit composite polymerization and dentin bonding</td>
</tr>
</tbody>
</table>

**Surgery**

<table>
<thead>
<tr>
<th>Dorn &amp; Gartner ’90</th>
<th>Success of Surgical Endo:</th>
</tr>
</thead>
<tbody>
<tr>
<td>Apical Sx:</td>
<td>59%</td>
</tr>
<tr>
<td>Re-Tx + Apical Sx:</td>
<td>80%</td>
</tr>
</tbody>
</table>
Rubenstein & Kim '99
Testori '99
Briggs & Scott '97
Moiseiwitsch & Trope '98
Lin '96

- Source: Friedman's analysis in Essential Endo.
- Dorn & Gartner (1990 JOE): Retrospective study in two endo offices (non-randomized, etc): Success Super EBA 95%; IRM 91% and amalgam 75%
- Rubenstein & Kim (1999): CRITICAL: Using scope, ultrasonics and Super EBA: n=94 cases (2/3 posterior & 1/3 anterior): 97% radiographic success at 3-12m follow-up with mean healing of 7.2m (criteria = restoration of lamina dura). 85% granuloma and 15% cysts with no difference in time to heal. Isthmus were found in 25% of the cases.
- Testori (OOO 1999): n=302 apices (181 teeth) with 5yr follow-up standardized radiographs with 2 observers: 85% complete healing with ultrasonic tips and super-EBA at 4.6yr versus 68% complete healing for rotary microhandpiece with amalgam. Saw reduced success when had poor or no prior NSRCT (see Danin below)

- Danin (1999 OOO): Did endo sx in necrotic cases without any NSRCT. 50% mod-complete success at 1yr (but used bur and glass ionomer for endo sx). But- 90% of these cases had bacteria in canals. Important point: cases may show radiographic success after sx rct even with bacteria in canals.
- Bradford (1999 OOO): defines sx success as 1) absence of symptoms; 2) absence of swelling, sinus tract, signs of infection; 3) radiographic evidence of healing; 4) continued normal functioning of the tooth. Summarized qualities of an ideal root-end filling material: biocompatibility, apical sealability and handling characteristics
- Lin (1996 IEJ): Discussed periradicular curretage. Remove for visibility. NEED NOT REMOVE ALL GRANULATION TISSUE FOR HEALING TO TAKE PLACE!

Re-Tx is Preferable over Endo Sx:
- Moiseiwitsch & Trope (1998 OOO): Sx should not be considered primary tx when non-surgical re-tx (or even NSRCT for first time) can be done.
- Briggs & Scott (1997): meta-analysis
- Allen, Newton & Brown (1989): statistical analysis

Arens

Precautions for Sx
Persistive headache - get med consult
Convulsive disorders - tx pt when medication is at maximal effectiveness; no epi
Emphysema - AM appt; short appts; avoid sedation
Asthma- sedate, O2 available, bronchodilator, have epi available
Congenital heart disease- consult
HBP: systolic >150 or diastolic >90 no sx until pt is stabilized; do not exceed 0.2 mg epi

Contraindications for Sx
Uncontrolled HBP
Recent MI
Subacute bacterial endocarditis
Uncontrolled hematologic problems
Osteoradionecrosis
Uncontrolled diabetes
Root is excessively short
Tooth is non-restorable

Bradford (1999): If it is not possible to make root end prep to ideal depth (ie, large post to apex), then consider bonded composite

Bevel of the Root Resection:
<table>
<thead>
<tr>
<th>Author(s)</th>
<th>Title</th>
<th>Reference Year</th>
<th>Details</th>
</tr>
</thead>
<tbody>
<tr>
<td>Gilheany '94</td>
<td>Studied relationship between bevel angle of root resection and required depth of retroprep to minimize leakage.</td>
<td></td>
<td>0 degree bevel: minimal retroprep depth of 1 mm</td>
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<td></td>
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<td></td>
<td>30 degree bevel: minimal retroprep depth of 2.1 mm</td>
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<tr>
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<td></td>
<td>45 degree bevel: minimal retroprep depth of 2.5 mm</td>
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<tr>
<td></td>
<td>They conclude with a recommendation of 3.5 mm retroprep depths (thus, the retroprep depth should extend coronal to the pulpal terminus of the tubules)</td>
<td></td>
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<tr>
<td>Chong '97</td>
<td>Bevelling root ends should be minimal since they open up dental tubules.</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Tidmarsch (1989)</td>
<td>Bevelling root ends should be minimal since they open up dental tubules.</td>
<td></td>
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</tr>
<tr>
<td>Gagliani &amp; Molinari (1998 JOE)</td>
<td>The bevel of the root end should not be greater than the depth of the rootend prep.  3mm prep provides safe and effective seal even when the bevel is 45° or 90°</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Khoury 1987</td>
<td>Reported bony lid method for mandibular sx. Technique increases vision and access; AND, reduces bone loss and incidence of hematoma</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hirsch 1979</td>
<td>Should not do endo sx consisting of curretage without placing a root endo filling since not removing infected apex and sealing off potential RC microoganisms. Thus, curretage alone is not predictable.</td>
<td></td>
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</tr>
<tr>
<td>US vs Rotary Handpiece</td>
<td>Richman (1957): First to propose US for root end resection</td>
<td></td>
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<tr>
<td></td>
<td>Wuchenich &amp; Torabinejad (1994): Cadaver study: US produces deeper preps with smaller bevels, better centered in canal</td>
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<tr>
<td></td>
<td>Gutmann (1994): In endo sx, Ultrasonics remove bacteria better than burs.</td>
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<tr>
<td></td>
<td>Engle &amp; Steiman (1995 JOE): US tips gave superior results compared to micro-rotary handpiece for retropreps</td>
<td></td>
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<tr>
<td></td>
<td>Gorman &amp; Steiman (1995): Ultrasonic tip vibrating freely in completed prep may flush out remaining debris. Also reported by Gutmann (1994)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Morgan &amp; Marshall 1998</td>
<td>Recommended use of the Multi-purpose bur (Caulk) for root resection to give smooth surface with least shattering and cracking</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Saunders &amp; Gutmann</td>
<td>Saunders &amp; Gutmann (1994): US may produce cracks (at ENAC power setting = 10) =FIRST REPORT OF US &amp; CRACKS ! Cracks may be due to impact of UC tip against dentin and heat formation</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Abedi &amp; Torabinejad</td>
<td>Layton (1996): In endo sx, ultrasonics may produce cracks at root end. (esp at high freq &gt; low frequencies)</td>
<td></td>
<td></td>
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<tr>
<td>Frank &amp; Bakland</td>
<td>Min &amp; Brown (1997) did report ultrasonics cause cracks in root end preps</td>
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<tr>
<td></td>
<td>Waplington (1997): Ultrasonic root end preps does NOT cause cracks</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Abedi &amp; Torabinejad (1995): Crack formation by ultrasonics was a fxn of power, time, initial cracks, and thickness of remaining dentin</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Frank &amp; Bakland (1996): Ultrasonics on medium power with water spray reduces incidence of root infractions (= cracks)</td>
<td></td>
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</tr>
<tr>
<td></td>
<td>Lin (1999): Used strain guages to measure root tip during US preps. US &gt; rotary for root end strains. But , didn’t see any cracks</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Brent &amp; Baumgartner (1999): Diamond-coated US tips (= S12D/90°) did produced 2 cracks; CT-5 tip produced 5 cracks (neither produced root fracture). BUT- diamond coated tips produce heavy abrasion, lots debris &amp; uneven preps and required CT-5 tip to make a pilot hole. Also, difficult using diamond tip to prepare isthmus groove</td>
<td></td>
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</tr>
<tr>
<td>Harrison 1991</td>
<td>Overview of biological responses in wound healing</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Author(s)</td>
<td>Title</td>
<td>Summary</td>
<td></td>
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<tr>
<td>Harrison &amp; Jurosky 1991</td>
<td>Healing of incisional flaps in rhesus monkeys.</td>
<td>Sulcular incision leaves peri tissue attached to cementum which speeds up repair and prevents epithelial downgrowth. Rec using undermining elevation of vertical flap.</td>
<td></td>
</tr>
<tr>
<td>Harrison &amp; Jurosky 1991</td>
<td>Healing of dissectional wounds in rhesus monkeys.</td>
<td>Day 1: have clot present; Day 2-3: have PMNs, macrophages, fibroblasts. Day 4: Type I collagen in new BV, fibroblasts predominate, osteoclasts. Day 14: have fibrous CT, new perios, no loss of alveolar crest. Day 28: have completely normal tissue.</td>
<td></td>
</tr>
<tr>
<td>Andreassen &amp; Rud 1972</td>
<td>It is difficult to determine if a large PARL is scar tissue or inflammation in the healing periapical surgery area. Recall that Penick 1961 reported case of sx after NSRCT and found scar tissue.</td>
<td></td>
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</tbody>
</table>

**MTA: Major properties: excellent seal and biocompatible**
- Torabinejad (1993-5) MTA shows potential as root end filling (less leakage than Super EBA or amalgam, even in presence of blood). Less leakage due to dye tracer or bacteria (S. epidermidis for 90 days). Mix 3 parts powder to 1 part aqueous soln. MTA powder consists of hydrophilic particles that hydrates to form a colloidal gel with pH12.5; sets in ~4h with compressive strength (21d) similar to EBA & IRM. (NB: Oynick also shown Sharpy's fibers growing in super EBA).  
- Torabinejad (1995): Biocompatible: Cementum forms over MTA retrofit with Sharpys fibers (Negative in mutagenicity tests)  
- Composition (from 1999 web page Tulsa MSDS): tricalcium silicate, dicalcium silicate, tricalcium phosphate, bismuth oxide, tetracalcium aluminoferrite, calcium sulfate dihydrate (= gypsum) (sets in 3-4 hr). (NB: This is different from original formulation which was reported to be tricalcium silicate, tricalcium oxide, tricalcium phosphate, mnemonic “SOP”).  
- FDA Indications: perforation repairs (non-communicating), apexification, root-end filling, pulp capping  
- Sluyk & Hartwell (1998): MTA useful for furcation repair. MTA resisted displacement at 72hr better than 24hr (p<.05). No difference if MTA covered by wet or dry cotton pellet (moisture probably derived from furcal tissue). In this in vitro study, found better adaption of MTA to perforation walls when first placed Gelfoam as an internal matrix  
- Koh & Torabinejad (1999): Osteoblasts grow in vitro into contact with MTA (not seen with IRM); also saw elevated levels of cytokines from MTA-osteoblast cultures (IL1alpha, IL1beta and IL6).  
- Torabinejad & Chivian (1998): Described clinical indications and techniques for using MTA. Indicated for pulp capping (Pittford 1996), apical barrier (after 1 w Ca(OH)2 in necrotic cases; close wet cotton/cavit; obturate >4h later), root perfs, root end fillings (rec small carrier=0.9mm RR Carrier, Chige Inc; don't rinse sx site after placing MTA), orifice plug seal  
- Rick Schwartz, Bill Walker (1999 JADA): Case reports of MTA for vertical root fx (with post), apexification, perf repair, and repair of internal resorption  
- Arens & Torabinejad (1996): MTA good for perf repair. Also: Pittford & Torabinejad showed MTA is good for furcal repairs.  

**Super EBA**
- Oynick (19xx): also shown Sharpy's fibers growing in super EBA  
- Dorn & Gartner (1990 JOE): Retrospective study in two endo offices (non-randomized, etc): Success Super EBA 95%; IRM 91% and amalgam 75%  
- Rubenstein & Kim (1999): CRITICAL: Using scope, ultrasonics and Super EBA: n=94 cases (2/3 posterior & 1/3 anterior): 97% radiographic success at 3-12m follow-up with mean healing of 7.2m (criteria = restoration of lamina dura). 85% granuloma and 15% cysts with no difference in time to heal. Isthmus es were found in 25% of the cases.
### Trope '96

- **Testori (OOO 1999):** n=302 apices (181 teeth) with 5yr follow-up standardized radiographs with 2 observers: 85% complete healing with ultrasonic tips and super-EBA at 4.6yr versus 68% complete healing for rotary microhandpiece with amalgam. Saw reduced success when had poor or no prior NSRCT (see Danin below)
- **Fitzpatrick & Steiman (1997):** Finishing bur (Brassler ETUF9) gave better marginal adaptation of EBA & IRM to the retroprep as compared to ball burnisher or wet cotton pellet
- **Forte & Hartwell (1998):** 6 month Fluid filtration leakage study: super EBA/finishing bur (#556 cross-cut) leaked more at 1day, but was equal to super EBA/burnished at 1week to 6 months
- **Beltes (1988):** EBA < Amalgam < Ketac < hot burnished GP.
- **Bondra (199):** EBA < IRM << amalgam 2mm preps.
- **Trope (1996):** EBA < IRM << GI < Amalgam and light cured composite
- **Bogaerts (1997 IEJ):** Case report: used Ca(OH)2 as internal matrix and sealed perforation with EBA

### Chong & Pitt-Ford 1993
Glass ionomer is a poor retrofill (too much polymerization shrinkage), but in a thin layer, does a good job of sealing dentinal tubules exposed in the root end resection

### Craig & Harrison 1993
Citric acid (50% X 2 min; pH=1) tx of resected root ends removes smear layer, exposes collagen and enhances cementogenesis

### Eberhardt & Torabinejad 1992
CT scans of 38 pts. 5% had roots protruding into the sinus. 2nd molars closer to sinus than 1st molar

### Green 1986
Reviews hx, dx and tx of hemisection and root amp

### Javelet & Torabinejad 1985
Monkey study showing isobutyl cyanoacrylate is effective alternative to sutures. It causes a little more inflammation than sutures, but not much

### Pecora & Kim '95
**Jeanssone '99**
**Matisko & Zullo '99**

**Review of GTR (Guided Tissue Regeneration):**
- **Boyne (1961):** PA Lesions of 5-8mm healed with bone regeneration, but lesions 8-12mm diameter did not
- **Nyman (1991):** GTR procedures were developed to exclude epithelial proliferation
- **Kellert (1994):** 2 case reports. Indicated when LEOs are complicated by loss of marginal attachment. Can promote fxn PDL
- **Cortellini & Bowers (1995):** Comprehensive review of GTR. Best results for deep, narrow defects. Poor results when have poor plaque control, smoking
- **Pecora & Kim (1995):** Large PARLs healed more rapidly and better with GTR
- **Urbani (1997):** 3 case reports of exposed resorbable membranes. Left them in place & tx with antibiotics and chlorhexidine
- **Maguire & Simon (1998):** Cat study, neither hOP-1 nor resorbable membrane (Guidor) had positive effect on healing
- **Bohning & Jeanssone (1999):** Studies healing of rat 5mm cranial defects (Guidor). Saw NS difference vs control
- **Matisko & Zullo (1999):** GTR (Collagen Periodontal Barrier membrane by Integra Life Sci) improved healing rabbit maxillary sinus oroantral defect (5mm covered by flap). GTR produced complete healing by histology at 4weeks. Note also that Avitene has been used to close oroantral defects (Mitchell & Lamb 1983).

### Rud & Andreasen 1972
Recall: Watch periradicular area for 1yr. If ok at 1yr, then healing is ok. If not, then re-tx

### Saad & Abdellatif 1991
Used freeze-dried bone allograft to fill bony defect after removal of PA lesion. At 6-9 months follow-up, graft could no longer be identified as separate entity

### Wallace 1996
Discussed transantral sx of palatal root

### Martin & Nind Br Dent J 1987
Chlorhexidine gluconate can be irrigated into apicoectomy sites to reduce flora 94% immediately and 78% even after 10 days! Something to consider for immunocompromised pts?
### Hemostasis during Periradicular Surgery (see also: coagulation, hemorrhage, bleeding disorders)

#### General Issues
- Witherspoon & Gutmann (1996 IEJ): general review on hemostasis: 3 phases: vascular phase, platelet phase, coagulation phase
- Petruson (1974): Anxiety can increase bleeding via stress-induced inc BP and inc fibrinolysis
- Macphee & Cowley (1981): Vertical incisions bleed less since most vessels run parallel to long axis of tooth

#### Vasoconstrictors
- LA: need make deep anesthesia with slow injections of vasoconstrictor (Gutmann 1993; Curtis 1966)
- Gutmann (1996) rec use of 2% lido with 1:100k supplemented with 2% lido with 1:50k epi
- Gutmann (1993) & Buckley (1984) epi 1:50k produces better hemostasis than epi 1:100k. Vasoconstriction via \( \alpha_1 \) adrenoceptor.
- Gutmann (1996) recorded CV parameters in dog endo sx with no evidence of untoward effects (concludes that can use higher doses in endo sx if needed in healthy pts). Inject slowly
- Milam (1984): injection of epi into muscle increases bleeding via \( \beta_1 \) receptors
- Robers & Sowray (1987): Inject slowly (1-2 ml/min) to get better spread of LA and therefore better hemostasis
- Buckley & Ciancio (1984): Perio flaps with 2% Lido with 1:100,000 epi had about twice as much blood loss as flaps tx with 2% Lido with 1:50,000 epi
- Lindorf (1979 OOO): Can get rebound effect (reactive hyperemia) on blood flow after injection with vasoconstrictors

#### Collagen, Bone Wax
- Avitene (= microfibrillar bovine collagen. MOA: 1) increased platelet adhesion, aggregation & activation; 2) activates Hageman factor (XIII); 3) forms mechanical plug of collagen; 4) evokes release of 5HT. Avitene is effective hemostatic agent with minimal tissue rxn.
- Evans (1979): Avitene has been used to control bleeding from wounds of hemophiliacs.
- Haassch & Gerstein (1989): Avitene has been used in endo sx with minimal effects on wound healing.
- Decker (1991): Avitene sticks to surfaces, so may want to apply it with a spray technique.
- Witherspoon & Gutmann (1996): Bone wax interferes with healing, so it is contraindicated
- Finn & Schow (1992): Bone wax is not resorbed and inhibits new bone formation & causes foreign body rxn
- Sauveur (1999 IEJ): Suggested mix bone wax with fibers of calcium alginate into small ball. Place ball into crypt so that the root tip protrudes and is isolated from bony crypt. Easy complete removal after sx.

#### Chemicals, Drugs
- Lemon & Steele (1993 JOE): Ferric sulfate caused bone damage (wabbit sx) and delayed healing when left in-situ
- Lemon & Jeansonne (1993) report that ferric sulfate is a low pH (0.8-1.6) necrotizing agent which evokes intravascular coagulation (similar to cautery). Hemostatic control for 5min.
- Jeansonne & Lemon (1993): Ferric sulfate (Cut-trol) provides effective hemostasis and does not delay healing in rabbit PA sx, PROVIDED that crypt receives proper irrigation and the osseous defect is curretaged.
- Souto & Oliver (1996): Pts on coumadin tx were safe for oral sx using normal coumadin regimen and local tranexamic acid (a potent anti-fibrillar agent) post-op for 2 days
Reimplantation and Transplantation

**Success in Intentional Reimplantation:**
2. Keller (1990): 91% success

(Koenig (1988): n=192 Keep out of socket<15min, do not touch root, keep it moist, minimal splinting
Dumsha & Gutmann (Compendium 6/95): reviewed clinical guidelines
Kratchman (1997): Reviews intentional reimplantation. Describes technique based on Niemczyk's philosophy of keeping tooth in HBBS

Cohen 1995  Reported success autograft and allograft transplantations

**MICROBIOLOGY / ASEPSIS / MANAGEMENT OF INFECTIONS**

Fabricius 1982  RC infections are polymicrobial. When infected monkey teeth with bacteroides oralis (now = Prevotella oralis), could only recover it when co-innuculated with other bugs. Monkey teeth were left open for 1 week and then closed at various times. Relative # of obligate anerobes increased over time.

**E. faecalis**
- Molander & Siren
- Fabricius '82
- Bystrom & Estrella
- Siquera & D'Arcangelo
- Heline '98
- Fuss ‘97

*Enterococcus faecalis is a nasty organism:*
- Molander (1998): Found Enterococcus faecalis in 33% of 100 failed NSRCT cases.
- Siren (1997 IEJ): Found Enterococcus faecalis in 60% of failing NSRCT cases (cases tx by genl dentists in Finland).
- Fabricius (1982): Unlike most endo infections, Ef can survive in RC system as single organism (rather than polymicrobial community). Confirmed in germ-free mice (Sobrinho (1998))
- Estrella (1999 JOE): Ca(OH)2 demonstrated NO antimicrobial effect at 2, 3, & 7 days against E. faecalis, S. aureus in infected dentinal tubules (suggests antimicrobial effectiveness is due to concentration of [OH] and time of exposure)
- Fuss (1997 IEJ): In vitro study: Roths > CRCS or Salapex at killing Ef in 24hr old mixtures; sealapex active at 7days after mix.
- D’Arcangelo (1999): 0.5% = 1% = 2.5% = 5% NaOCl for antimicrobial effectiveness (11 strains inc E. faecalis; in fac aerobes-aerobes, microaerophiles, obligate anerobes). IMPORTANT POINT: Best when use at least 10 min contact time
- Siqueira (1997 IEJ): 4% NaOCl is effective at killing E. faecalis in vitro infected RC systems (with hand files, US files or H202)
- Heline (1998 IEJ): Chlorhexidine is effective in dentin infected with E. faecalis (ie chlorhex = NaOCl)

**BPB (Black Pigmented Bacteroides):**
- Found to be associated with necrotic symptomatic teeth (Sundqvist (1989): 73% of canals containing BPB had abscesses or pus
- Divided into Porphyromonas (asaccharolytic BPB): *P. asaccharolyticus, P. gingivalis, P. endodontalis
- Divided into Prevotella (saccharolytic BPB): *P. intermedia, P. nigrescens, P. melaninogenicus, P. denticola, P. loesscheii
- The most common BPB isolated is *Prevotella nigrescens* (Bae & Baumgartner 1997; Baumgartner 1999).
- Baumgartner & Faullker (1991): 84% of 90 PA lesions had IgG reactive to *P. intermedia
- Bogen & Slots (1999): Sampled 20 PA lesions with good controls for contamination; found only 1 lesion containing *Porphyromonas gingivalis* (pt had pain).
- Prev. melano is associated with pain (Yoshida [1987JOE]; Griffee [1980 OOO], Sundqvist [1989])

Waltimo & Haapasalo  Screened 967 samples taken for failing NSRCT cases. Found microorganisms in 71% failing cases. Fungi were found in 5% of the 967 cases. All
1997 IEJ isolates except one was from genus Candida (C. albicans was found most often). Also reported by Nair 1990. (EM description of yeast-like organisms in 22% (2 of 9) therapy-resistant failing NSRCT cases. Since initial flora of RC systems do not usually contain yeast (Sundqvist 1989), Waltimo concludes that yeast is more common in persistent RC infections.

Conrads 1997 Used PCR of 16S ribosome instead of culturing to identify actinomyces, F. nucleatum and, for the first time, Bacteroides forsythus in infected human pulp.

Bae & Baumgartner 1997 Used SDS PAGE to show that black pigmented Bacteroides collected from infected pulp consisted primarily of Prevotella nigrescens (73% of BPB isolated) and Prevotella intermedia (27% of BPB isolated).

Baumgartner 1999 Confirmed Bae & Baumgartner (1997): Cultured & PCR samples from 40 necrotic teeth with AP: 55% samples contained black pigmented Bacteroides pulp consisting primarily of Prevotella nigrescens (50% of BPB isolated) and Prevotella intermedia (36% of BPB isolated). 73% of the BPB-positive teeth were associated with purulent discharge from either RC system or sinus tract.

Anerobic infections occur when:
1. have necrotic tissue
2. compromised blood supply
3. by succession, infections by aerobes & facultative anerobes that lower the oxidation-reduction potential in the tissue.

Von Winkelhoff 1985 Reported that Porphyromonas intermedia & Porph. endodontalis were frequently found in periapical abscesses. Latter (1992) he argued that P. endodontalis is involved in anerobic mixed infections and may be essential in severe root canal infections.

Yamasaki 1999 Proposes that Porphyromonas endodontalis, Porphyromonas gingivalis and Fusobacterium nucleatum may participate in development PA lesions since sonicated bacterial extracts were cytotoxic to PA fibroblasts in vitro.

Sundqvist 1992 Demonstrates bacterial interactions are not random. Used odds ratio to show that some species tend to pair with others (eg., P intermedia and P anerobius).

Allard & Stromberg 1987 Dog study: In microbiologically-induced PA lesions, got 4 month healing even when obturate with bacteria remaining in canal systems. (Contrast with Sjogren 1997 who showed in humans that prognosis is reduced if bacteria are present at time of obturation)

Baumgartner 1976-77 NSRCT produced low bacteremia (same organisms in blood as in canal systems) whereas sx endo produced high bacteremia especially from flap resection. Extraction = 100% bacteremia
flap reflection = >80%
curette = 33%
NSRCT: 3.3% (instrument beyond apex)

Brook & Frazier 1991 Anerobic bacteria found in 94% of cases of aspirates collected from purulent abscesses (44% of cases were polymicrobial). Also reported by Farber & Seltzer 1988.

Tronstad 1987 Reported several species of bugs in PA lesions of teeth refractory to NSRCT. (but, they used cotton roll isolation which may have permitted contamination).

General cases where extraradicular infection is present:
1. Abscessed apical periodontitis (Gatti 1996)
2. Periapical actinomycosis (Sundqvist 1980; Happonen 1986)
3. Infected radicular cysts (esp Bay cysts= PA pocket cysts) Nair 1996
4. In association with extruded infected dentinal chips (Holland 1980)
5. Infected cementum (Kiryu 1994)

**Evidence supporting bacteria in periradicular lesions:**
- Abou-Rass (1998 IEJ): Well controlled clinical collection study of closed lesions. Found 13/13 lesions had bugs. 63% obligate anerobes and 36% fac anerobes. Inc. actinomycoses (32%), Propionibacterium (23%), Streptococcus (18%)
- Sundqvist (1980); Happonen (1986): Periapical actinomycosis
- Iwu (1990 OOO): 88% (14 of 16) periapical granulomas contained bugs (homogenized and then cultured granulomas for microorganisms). Found Veillonella (15%), Streptococcus (22%), Actinomyces (11%)
- Wayman (1992 JOE): 83% (51 of 58) granulomas contained cultivable bugs. (Importantly - also processed 1/2 of each sample for histology. Could only detect histological evidence of bugs in 14% of these samples!)
- Nair (1987): SEM bugs in PA lesions as evidence for extraradicular infection

**Evidence against bacteria in periradicular lesions:**
- Ok, they seem to be there for sinus tracts, but that could be 2nd to intraoral infection (REF?)
- Whose study isolated by cotton roll? Tronstad!
- Walton 1992: Dog study with infected RC systems: Block sections showed bugs in canals but not in periradicular lesions (DM = histology with gram stain) [BUT- See Wayman ’92 results]

**Sundqvist 1976**
CLASSIC: Microbiology study on 32 traumatized anterior teeth in 27 pts.
- Major Point: Apical periodontitis can only be detected in teeth with bacteria present in canal systems.
- Necrotic, but sterile teeth have no signs of PARL.
- In contrast, necrotic and infected teeth showed PARLs.
- 90% organisms isolated from intact, but necrotic teeth were anaerobic
- Bacteroides melanogenicus (now: Prevotella melanogenicus) occurred in all teeth with pain but in none of the other teeth
- Probability of pain increased with # bacterial species (esp when >6); suggests bacterial synergism is important virulence factor.

**Kakehashi, Stanley & Fitzgerald**
CLASSIC: Gnotobiotic rats demonstrated capacity of pulp for self-repair in the absence of bugs and no development of apical periodontitis. Infection of pulps in normal rats resulted in pulpal necrosis and AP (ie, supports bacterial origin of AP).

**Moller 1981**
CLASSIC: Bacteria are an etiologic factor for apical periodontitis: Aseptic pulp devitalization in monkeys had no PA lesions. BUT, necrotized and infected pulps produced PA lesions. Thus, no evidence that necrotic tissue (via toxic breakdown products or antigen altered proteins) per se induces lesions. Instead, you need bugs.

**Nair 1990**
Therapy-resistant bugs (inc fungi) may lead to failure. Also reported by Weiger 1995

**Gier and Mitchell ’68 Delivanis ’84**
Anachoresis
- Robinson (1941 JADA): states that anachoresis occurs in pulpal inflammation after bacteremia
- Gier and Mitchell (1968): Demonstrated anachoresis in traumatized pulps after systemic iv injection of bugs. This may explain Sundqvist’s (1976) observation of microbial infection in traumatized but intact teeth.
- Tziafas demonstrated that bugs given iv accumulate in pulp beneath Ca(OH)2 direct pulp cap (which induced pulp inflammation).
- Delivanis (1984 JOE): In order for anachoresis to occur, need some tissue in canals; unfilled canals do not become infected from the bloodstream
- POTENTIAL RESEARCH QUESTION: Is the mechanism of anachoresis possibly similar to mechanism of leukocyte chemotaxis to inflamed tissue? (ie, recall expression of the CAM CD102 on inflamed pulpal BV by Tasman (1999 JOE). Potential expt: do bugs bind to CD102? If so, then anachoresis and leukocyte chemotaxis have similar mechanisms…… (CHO cells transfected with CD102 gene, grow to confluency and use to pan for various microorganisms versus non-transfected CHO cells)
<table>
<thead>
<tr>
<th>Reference</th>
<th>Endotoxin</th>
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<tbody>
<tr>
<td>Dwyer &amp; Torabinejad '81</td>
<td>Dwyer &amp; Torabinejad (1981): Endotoxin placed into canals caused severe pulpal and periradicular inflammation. Concluded that endotoxin plays a significant role in periradicular disease.</td>
</tr>
<tr>
<td>Trope '97</td>
<td>Horiba (1991) found correlation between clinical symptoms, PARL and endotoxin content.</td>
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<tr>
<td>Alves '98</td>
<td>Schein &amp; Schilder (1975) found positive correlation between endotoxin levels and necrotic, painful teeth with PARLs.</td>
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<td>Darveau '95</td>
<td>Safavi &amp; Nichols (1993): Ca(OH)2 inactivates LPS in vitro</td>
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<td>Alves (1998): Compared bacterial (Campylobacter rectus, Peptostreptococcus micros, Fusobacterium nucleatum, Prevotella intermedia) leakage of LC obturated canals with post-space prep to endodoxin. Endotoxin leaked faster than bugs (Means: 23 days vs 62 days)</td>
</tr>
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<td>Trope (1995): Endotoxin penetration of coronally unsealed NSRCT teeth &lt;21 days. Emphasizes importance of coronal seal</td>
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| Darveau (Infect Immun 63:1311, 1995) | V.

**At least some bacteria are site specific.** P. intermedia is associated with perio pockets whereas P. nigrescens associated with root canals.

- **Gharbia & Haapasalo 1994**
- **Gomes, Drucker 1994; 1996**
- **Griffie & Patterson 1980**
- **Iwu & MacFarlane 1990**
- **Namavar & Marian 1983**
- **Oguntebi 1994**
- **Reeves & Stanley 1966**
- **Stabholz & Sela 1983**
- **Tronstad & Mjor 1972**
- **Chirnside 1961**
- **Matusow 1988**
- **Giunta 1987**
- **Paonesa & Goldstein**
- **Tronstad & Olsen 1999**

Reviews brain abscesses caused by odontogenic infections. Routes include 1) direct extension (fascial planes); 2) hematogenous (via facial, angular, ophthalic, via cavernous sinus); 3) local lymphatics; 4) indirectly by extraoral infection. 1° Streptococci, Bacteroides, Staph.
### Managing Infections
- Harrington (1992 DCNA): Key to managing fluctuant swelling due to infection is to obtain drainage
- Gatewood, Himel & Dorn (1990 JOE): antibiotics are an adjunct to establishing drainage. No consensus has been reached on IND of a cellulitis.

### Radiology and Oral Pathology

#### Great Reviews with lots of pictures:
- Miles DCNA Jan 1994 "The Clinical Guide to Radiologic Diagnosis"

- Hubar 1995 Gen Den
  - Film packets are disinfected by 1min immersion in 2.63% NaOCl (must blot film packs first)

- Danforth & Torabinejad 1990
  - In order to produce enough radiation to produce cataracts, a pt would need 10,900 endodontic surveys

- Ellingsen & Harrington 1995
  - D-speed radiographs are better than RVG and E speed radiographs in identifying tips of small files. RVG = D-speed in extracted teeth when using negative-to-positive and zoom mode

- Forsberg 1987
  - Paralleling technique was more accurate for working length films than bisecting angle

- Goerig JOE 13(12):1987
  - Describes simple explanation of buccal object rule with illustrations

#### Radiographic Evaluation of PARLs:
- Bender & Seltzer (1961): CLASSIC: Reported that had to remove cortical plate of bone in order to detect PARL (note that this misses subtle signs of apical periodontitis reported by Brynolf).
- Brynolf (1967): Correlated radiographic findings with histological findings: Radiographic signs of apical periodontitis: bone structural changes (= primary importance, but tough to see); widening PDL space; loss of lamina dura; mineral loss of bone
- Kaffe & Gratt (1988): Reviewed radiographic apex and found that the best predictors of pulpal disease are disruption and change in the size of the PDL space and bone trabeculation. Similar to report by Brynolf.
- Barbat & Messer (1998 JOE): Cadaver study: Compared conventional radiography to digital radiography for detection of artificial PARLs. Both could detect removal of lamina dura and cancellous bone. BUT lesions were largest and easiest to detect when removed cortical plate
- Schwartz (1981 JOMS): The lamina dura is a layer of compact bone (= cribiform plate or alveolar bone proper) that lines tooth socket; Xray beams passing tangentially through the socket passes many times the width of the adjacent alveolus and are attenuated by this greater thickness of bone; producing the characteristic "white line" radiopacity (From: Glickman, POP). Normal teeth may not have detectable LD due to angulation of central beam and tooth in socket

- Phillips & Weller 1992
  - When viewing panoramic radiographs, the mental foramen is 2.2 mm mesial and 2.4mm inferior to the apex of the 2nd premolar

- Shearer & Wilson 1996
  - Reviewed the use of contrast medium in dx and evaluation of canal systems

- Scarfe 1995
  - Used Hypaque (radiopaque contrasting media) to improve radiographic interpretations

#### Fraudulent Radiography
- Tseng (1999) scanned original film-based radiograph, manipulated, printed to 35mm film on slide maker, print to duplicating film. The altered
- JOE Sept 1999): Fabrication of Schick images

Tamse & Kafne 1980  Zygomatic arch interferes with the DB root of max 1\textsuperscript{st} molar and 48% of MB roots of max 2\textsuperscript{nd} molars. Need to shift xray beam to move arch off of the root

Forsberg 1994  Demonstrated advantages of parallel radiographic technique over bisecting-angle for interpretation of periapical lesions

**Transient Radiolucencies:**
- Orstavik (1990): Reported that can see transient increase in apical radiolucency over first few weeks after completion of NSRCT.
- Reit (1987): Confirmed Orstavik finding of a transient increase in PARL a few weeks after completing NSRCT
- Andreasen (1986): Trauma: 4% luxated teeth can develop transient apical breakdown (= PARL) which recovered over time.

Orstavik 1996 and Reit 1987  By one year after completing NSRCT, almost all teeth tx for chronic apical periodontitis will show radiographic signs of healing

Orstavik 1986  Developed standardized scoring system for evaluating apical healing on radiographs (= PAI = periapical index). This uses a visual reference scale using histology-verified case radiographs (from Byrnolf’s material).

**Periapical Pathology, Immunology and Inflammation**

**Quick Overview of Differential Dx of PARLs by Location:**

**Maxillary Incisors - Premolars**
- Granuloma
- Cyst
- OKC
- Ameloblastoma
- Sarcoma: early PDL widening 1 or more teeth; late floating roots
- Nasopalantine Canal Cyst (aka Incisive canal cyst): most common developmental cyst, often with swelling & tooth displacement
- Globulomaxillary Cyst: teeth are vital; can cause spreading of roots. Between lateral - canine
- Adenomatoid Odontogenic Tumor

**Maxillary Premolars - Molars**
- Granuloma
- Cyst
- OKC
- Ameloblastoma
- Sarcoma: early PDL widening 1 or more teeth; late floating roots

**Mandibular Incisors - Premolars**
- Granuloma
- Cyst
- OKC
- Ameloblastoma
Sarcoma: early PDL widening 1 or more teeth; late floating roots
Central Giant Cell Granuloma: most often mand ant region; women: <20yrs
Sublingual Salivary Gland Depression - usually associated with vital incisor. Mean: 1cm diameter & cover apical 1/3 tooth
Periapical Cemental Dysplasia (“cementoma”): early PARL and late PARO; teeth vital
Lateral Periodontal Cysts (usually lateral not PA): most often this region; may be from supernumary or epithelial rests of Malassez

**Mandibular Premolars - Molars**
Granuloma
Cyst
OKC: most often in mand molar region; recurrence, arises from dental lamina; can be unilocular or multilocular (esp larger ones)
Ameloblastoma: most often in mand molar region, though can also be anywhere; usually multilocular
Sarcoma: = malignant neoplasm, early: symmetrical PDL widening 1 or >teeth; late: floating roots & often pain-paresthesia
CEOT (calcifying epithelial odontogenic tumor aka Pindborg). Usually next to unerupted tooth; can be in mand ant or maxilla
Metastatic tumor: often mand molar region; (84% are carcinomas); majority are from lung, breast & kidney
Squamous cell carcinoma: =90% of oral cancers, often lat border of tongue; if intraosseous most often mand
Submandibular salivary gland depression (rarely PA site) - usually located between IAN and inferior border of mand

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**Goerig**

**Differential Dx of Periradicular Radiolucent lesions:**

1. **Developmental PARLs**
   - Salivary gland depression
   - Neurovascular canals
   - Developmental
   - Median palatal cyst (Donnelly 1986)
   - Lateral periodontal cyst: uncommon, may be primordial cyst of supernumary or from epithelial rests of Malassez, usually no S/S; Natkin 1994
   - JOE:Zebra. -- Found in mand canine-premolar 76% of time [Fantasia 1979 OOO]

2. **Infection-Related PARLs**
   - LEO- granuloma: 50% cells are inflammatory cells (macrophages > lymphocytes > PMNs)
   - LEO- cyst: lymphocytes, plasma cells, macrophages, cholesterol clefts
   - Actinomycoses
   - TB

3. **Metabolic PARLs**
   - Eosinophilic granuloma: heavily infiltrated with eosinophils; lots of cyst-like structures: "roots in air"; Lin & Wyman 1979
   - Hyperparathyroidism: 10% have no lamina dura; ground glass, inc serum Ca, osteoporosis, stones, bones, vague jaw pain, PARLs
   - Hyperthyroidism: rare bone lesions (Scheffer 1957 OOO)
   - Fibrous Dysplasia - diffuse on Xray; biopsy feels like cutting styrofoam. Slow expansion of bone in all directions, ground glass; Natkin 1994
   - JOE:Zebra
   - Gauchers: pt has no glucocerebrosides (Lipid Metabolism Disease), lots of foam cells, roots resorbed slowly; Bender
   - Pagets: considerable fibrosis, no NSRCT healing; 7:1 max; ground glass
   - Vitamin D resistant Rickets - multiple PARLs even on virgin teeth

4. **Odontogenic PARLs**
   - Botyroid odontogenic cyst = multilocular lateral periodontal cyst
   - Odontogenic keratocyst: Nohl & Gulabivala (1996); Hancock & Brown 1986
Ameloblastoma - multilocular; aggressive, arises from dental lamina, composed exclusively of epithelium
Cementoma: (periapical cemental dysplasia) Wilcox & Walton 1989; Chandler & Sundqvist 1999
Cementifying fibroma - benign neoplasm of PDL progenitor cells; 2:1 females; mandible, PARL-PARO

- Neoplastic
  Central giant cell granuloma: in young pts, 67% mand, swelling, displaced teeth, (Natkin 1994 JOE:Zebra)
  Hemangioma
  Desmoplastic fibroma
  Osteoblastoma
  Ossifying fibroma - distinct margins on Xray; solid lesion on biopsy, displacement of teeth
  Carcinoma - metastasis from breast to maxilla (Spott [1985 OOO])
  Sarcoma
  Malignant lymphoma: Spatafore (1989 JOE)
  Metastatic disease
  Multiple myeloma
  Adenoid cystic carcinoma (Burkes 1975)
  Carcinoma of antrum (Copeland 1980)

What Causes Apical Periodontitis?  II-1α & β TNFα & β and Prostaglandins

- Bacteria cause AP of odontogenic origin (Sundqvist 1976; Moller 1981; Kakehashi 1965). But, how do bugs induce AP?
- Stashenko argues that histological studies are largely descriptive since they tell you that a mediator/cell is present (or not), but do not distinguish between protective versus destructive actions. Instead, must review functional studies:
  - Wang & Stashenko (1993 JOE): Primary bone-resorbing cytokine in human PA lesions = IL-1β (and ~60% of its bone resorbing activity is mediated by release of prostaglandins: Torabinejad & Kiger 1980; Dewhirst 1990). Thus, bacterial-induced release of IL-1β and prostaglandins are destructive.
  - Kawashima & Stashenko (1998 Immunology): Used P/E selectin knockout mice (P/E ko’s lack rolling adhesion of PMNs and macrophages to endothelium): Saw significantly more PA bone destruction in ko’s. Thus, phagocytic leukocytes (PMNs and/or macrophages) protect against bacterial-induced PA bone destruction in mouse model of AP.
  - Stashenko (1995 JDR): If give agent which enhances PMN activity (=PGG-glucan), see 40% reduction in PA bone destruction. Suggests that PMNs are protective in mouse model of AP.
  - Hou & Stashenko (1999 : 2000): Proposed that immunosuppressed pts may be at greater risk for developing disseminated abscesses from RC infections. Expt = RAG-2 SCID mice passively immunized with antibodies against inoculated microflora demonstrated important role of Ab in preventing infections spreading away from the RC system. Thus, B-cell responses are probably more important in preventing systemic spread of PA abscesses than T-cell responses. In other words, B-cell responses (ie, Ab) help to restrict spread of PA infection. Neither T nor B seems critical for development of PARLs. (RAG-2 cannot generate fxnl Ab or T-cell receptors). The lack of increased AP in pts with HIV also suggests that T cells are not critical in development of AP (in contrast to their role in preventing systemic spread of infections).

Differential Dx of Periradicular Radiopaque lesions:

- Bender (1985): Consider condensing osteitis, cemental dysplasia, exostoses, fibrous dysplasia. Do pulp vitality testing!
- Hypoparathyroidism: potential for tetany, radiopacity, serum hypocalcemia

Torabinejad & Naidorf 1985 JOE

All portions of the cell mediated and humoral immunological reactions occur in periradicular lesions. T>B in chronic PARLs.

4 Classes of Hypersensitivity Reactions  refs = evidence for involvement in apical periodontitis

Type I: Anaphylaxis: immediate, mediated by IgE
Type II: Cytotoxic: antibody mediated (IGG, IgM, etc) via C’ cytotox, Opsonization-phagocytosis, NK cell activity (Kuntz 1977 JOE)
Type III: Immune Complexes: Arthus, serum sickness, immune vasculitis, (Torabinejad 1976 JOE)
Type IV: Cell Mediated: delayed (lymphocytes, macrophages), (Stabholtz 1978)
Studies on Cytokines & Prostanoids in PA Lesions
- Band & Henderson (1993): Human radicular cysts contain IL-1alpha, IL-1beta and IL-6. Originates from cystic epithelial cells
- Matsushita (1998): Prevotella melaninogenica evoked in vitro production of IL6 from blood of pts with >10 PARLS vs 1-2 PARLS vs controls. Thus, this bug is associated with PARLs in humans and appears capable of evoking systemic sensitzation. (IL6 induces bone resorption)
- Torabinejad (1979): Concluded that PMNs release PGs into PA lesions to induce bone resorption since resorption blocked by indomethacin

Studies on Immunoglobulins in PA Lesions:
- Kettering & Torabinejad: Immune complexes can cause disease. Chronic PARLs have localized immune rxns. IgGs found in PARLs bind to bugs associated with PARLs (A israeli, F nucleatum, P micros, S intermedias).
- Takahashi (1997): Human lesion material: IgG > IgA = IgM. Cysts = granulomas for immunoglobulins. IgG1 is the predominant immunoglobulin (similar to dental pulp studies by Hahn 1995). Suggest that B cells which express IgG1 "home" to antigens found in periapical lesions (since could find no proliferating plasma cells within the lesion).
- Baumgartner (1991 JOE): IN CONTRAST TO Takahashi, Craig cultured PA lesions and demonstrated that plasma cells in the biopsy were capable of producing IgG via a local site of action
- Dahlen (1982): Demonstrated that monkeys immunized against RC flora had less apical inflammation. Implies host defenses can inhibit bacterial invasion into apical area
- Greening (1980): Concentration of IgG in apical lesions was 5 times concentration in non-inflamed oral mucosa

Studies on Systemic Immunoglobulins in PA Lesions:
- Keudell & Powell (1982): Pts with pulpal or periradicular disease do NOT have increased circulating IgG or IgM
- Matsushita (1998): Porphyromonas gingivalis cross-reacted with Ab taken from blood of pts with >10 PARLS vs controls. Thus, both bugs may be associated with PARLs in humans and are capable of evoking systemic sensitzation.

Studies on T Cell vs B cells in PA Lesions
- Maron & Kiss (1993): Studies immune cells in 63 PR lesions: cell-mediated immunity is probably more important than anaphylactoid-mediated responses. Lots of T cells and macrophages
- Walstrom & Torabinejad (1993): Periradicular lesions are not strictly due to T cells since lesions still develop in animals without T cells
- Kontianen (1986): In human PA lesions: Lymphocytes (T>B) > macrophages. Lots PMNs also.

Pitts & Williams 1982
Endotoxins placed on dog pulp induce PA bone resorption

Serene & Vesely
GP activates C3 complement. May explain why over-extension with GP may induce bone resorption in some pts. Also recall Sjogren (1995) small particles of GP induce intense inflammatory response.

Akamine 1994
PMNs and macrophages are the first cells to respond to pulpal inflammation

Byers and Taylor
Innervated teeth have less pulp necrosis and PA destruction than denervated

Baumgartner 1984
Periradicular lesions with sinus tracts can be cysts, granulomas or abscesses. 100% S.T. lined with stratified squamous epithelium

Goerig
Factors Associated with Metastatic Carcinoma Mimicking PA Pathology:
- Intermittent tingling or numbness of lower lip - Glaser (1997 Intl JOS): numb lip most common feature of metastatic CA
- Hx of previous CA - Todd (1987 JOE) reported metastasis occluding blood flow producing necrosis. Think of this when can find no obvious etiology
- Pain and swelling that do not regress after NSRCT
- Higher predeliction for metastasis to mandible: especially mand post teeth
- PARLs are diffuse and poorly outlined
- Todd & Langeland 1987: Found pulp necrosis from neoplasm that disrupted blood flow. Think of this when pt has +hx cancer and there is no apparent reason for tooth needing RCT.
- Selden (1998) found metastatic carcinoma as PARL on mand molar; later developed paresthesia.

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<tr>
<th>Goering</th>
<th>Partial Loss of Lamina Dura:</th>
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<tr>
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<td>Hyperparathyroidism</td>
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<td>Gauchers</td>
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<td>Cushing</td>
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<td>VD Resistant Rickets</td>
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<td>Osteoporosis</td>
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<tr>
<th>Granulomas vs Cysts</th>
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<tr>
<td>Bhaskar (1966)  48% granuloma 42% cysts</td>
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<tr>
<td>Lalonde &amp; Leubke (1968): 45% granuloma 44% cysts</td>
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<tr>
<td>Nair (1996): n=256 50% granulomas 15% cysts (inc both pocket cysts and true apical cysts), 35% PA abscesses</td>
</tr>
<tr>
<td>Rubenstein &amp; Kim (1998) 85% granuloma 15% cysts</td>
</tr>
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<td>Delzangles (1989): SEM study of root apices: granulomas produced more root resorption than cysts</td>
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<tr>
<td>Morse &amp; Patnik (1973) PAGE can be used to differentiate cyst from granuloma by aspirate</td>
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<tr>
<td>Trope (1989): Used CT scan to differentiate cysts from granulomas</td>
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<tr>
<td>Priebe &amp; Lazansky (1954): Can't tell if a lesion is a cyst or granuloma from an X-ray</td>
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Theories of Cyst Development
- Epithelial proliferation Theory: Seltzer; Summers (1974): epithelial cells proliferate to line abscess cavity
- Cavitation Breakdown Theory: Ten Cate (1972) ; Cohen (1979): Continuous growth of epithelial cells (rests of Malassez) removes central cells from their nutrition; innermost cells die & cyst cavity forms (PROBLEM: no evidence for lack of BV)
- Breakdown theory of cysts: . Toller (1967): Osmotic pressure buildup due to semi-permeable membrane (remnants of cellular debris inside lumen leading increased osmotic pressure which expands lesions as get inc fluid movement due to Starling's law)
- Immunologic Theory: Torabinejad (1983 Int JOS): continued immune rxn to antigens -bacteria in infected RC systems. Immune rxn responsible for proliferation of epithelium
- Harris (Br Med Bull 1975): reviewed other theories (European view), including mural development, hydrostatic enlargement and bone resorption

Treatment of Cysts:
- Freedland (1970 OOO): Described decompression technique for tx large cystic lesions
- Rees (1997 IEJ): Case report large max cyst. Tx = NSRCT with decompression usng 3cm surgical tubing as drain. Pt removed drain daily and flushed cyst with 10ml saline. Resolved in this case in 5yr (LARGE cyt). (this approach seems to be successful even though epithelium is not curetted out). Also reported successes by Sommer(1964) and Freedland (1970). Main advantage of decompression for lg cysts is avoid sx-induced devitalization of adjacent teeth.
- Hoen & Labounty (1990): Tx 2 cases reports of cysts by inserting large gauge needle, aspirating the lesion, rinsing with saline. Bony healing observed at 18 months
- Kehoe (1986): Tx cysts with cystic decompression
- Simon (1980): Studied teeth with attached lesions: 8.6% bay cysts (=pocket cysts) and 8.6% true cysts
- Nair (1993): Periapical pocket (bay) cyst can heal after NSRCT since removed stimulus. However, “true” radicular cysts (esp those containing...
cholesterol crystals) are not likely to resolve after NSRCT since they are independent of the root. Recall that Simon 1980 says only ~10% apical lesions are true cysts. Cholesterol crystals are found in cysts (not granulomas).

White 1968  
Paper points in periapical tissue induces chronic apical periodontitis (cellulose is not digested).

Sjogren 1995  
Tissue reaction of gutta percha: well tolerated at large pieces (=encapsulated by collagen); but induced intense foreign body response when placed sc as fine pieces (similar to Proplast teflon issue).

Andreasen 1986  
4% luxated teeth can develop transient apical breakdown (= PARL) which recovered over time. Teeth recovered with time. This may be example of transient “sterile” inflammation secondary to trauma.

Heimdahl 1990  
Demonstrated transient bacteremia in 20% pts during endo tx of teeth with apical periodontitis.

Johnson & Jeansson 1999 EDT  
Cultured 13 necrotic teeth with AP: no ß-hemolytic strep were cultured. Staph epidermidis was most common bug isolated.

Rubber Latex Allergies:
- Knowles & Newcomb (1998 JOE): Case report of a latex allergy pt for NSRCT: Pts physician recommended pre-tx with prednisone, Benadryl and Claritin before each appt. No problems following obturation with GP (even though she later developed an anaphylactic attack following exposure to a rubber handgrip on a dental instrument)
- Safadi (1996): Reported that 12% of oral health care workers have latex allergies
- Monthly OSAP Focus (1997)): (OSAP = Office Safety & Asepsis Procedures): latex allergies show cross-reactivity with patients reporting allergies to chestnut, avocado and Kiwi fruit, bananas. Schedule the latex allergy pt in the morning before latex dust has accumulated in the office.
- Source of non-latex rubber dam: Hygenic company
- Spina (1999): reviewed latex allergy. Speculated that development of latex allergy may have been due to initial production of low-quality, high-allergenic gloves in mid-1980’s (see also Sussman 1995)

Longwill & Marshall 1982  
Pediatric use of formocresol does NOT sensitize the child

Morse 1972  
Look up J Brit Endo Soc 6:13. Location of abscess depends on location of the root apex relative to muscle attachments.

Pharmacology and Oral Medicine

Good General Reviews:
Mosby Dental Drug Reference by T. Gage et al.,
Drug Information Handbook for Dentistry R. Wynn et al
ePocrates (excellent free Palm software): www.epocrates.com [1500 drugs with side-effects and interactions]

Moore 1999  
Macrolide antibiotics Currently marketed erythromycin analogues:
- Eryth. Competes with theophylline, digoxin, warfarin, triazolam, terfenadine, cyclosporine, warfarin for metabolism by hepatic P450 CYP3A & CYP1A2 generating possible drug interactions
- Azithromycin & Dirithromycin (do not compete for same liver P450 as eryt.)
- Clarithromycin (possibly less N&V vs eryth.).
- Arith & Clarith can kill Helicobacter pylori.
- Clarith & Dirith both produrgs

**Clindamycin**
Vacek (1972 Chemotherapy): Clin has strong distribution to bone than many other antibiotics (bone can retain 30% of clin in serum)
Antagonistic to erythromycin
Dryden (1975 JOE): Clin effective against organisms isolated from RC systems

**Tetracyclines**
- Urist & Ibsen: Staining of teeth by formation of complex with Ca ions in hydroxyapatite
- Adverse rxn - liver damage. Outdated T can cause n&v, polydypisa, polyurea
- Doxycycline: t1/2= 20h. Completely absorbed across GI tract +/- food.

**Anamura 1988**
Phenol acts to inhibit arachidonic acid synthesis

**Beach & Hutter 1996**
Case report of using apex locator on a pt with a pacemaker

**HIV Patients:** Reviews: Barr (1994 DCNA), Chenowith & Gobetti (1997): Recommendations about postexposure prophylaxis for HIV exposure
- Cooper (IEJ 1993): Compared NSRCT in 40 HIV+ cases and 17 control cases. No difference in short-term (3mo) complications.
- Garfunkel & Glick (1992): Review article on HIV, related systemic conditions and medications used for tx
- Glick & Trope (1989): HIV DNA found in dental pulp fibroblasts
- Greenspan & Greenspan (1993): reviewed oral manifestations of HIV
- Silverman (1986): 99% HIV+ pts have white hairy leukoplakia. In contrast, only 33% have Kaposi's sarcoma
- Porter (1993 OOO): no difference in post-extraction wound healing in HIV vs control pts
- Meskin (1999): Risk of seroconversion in health care workers exposed to HIV-infected blood thru percutaneous route is <0.3%
- Shiboski (1994): HIV pts with low CD4 cell counts (<200 cells/mm3; and often high viral loads) are 9 times more likely to develop oral manifestations of HIV. Includes destructive periodontitis, oral candidiasis and hairy leukoplakia
- Glick (1994): HIV PTs: Presence of an oral lesion is highly predictive of CD4 <200 cells/mm3. More than 90% pts with major aphthous ulcers, Kaposi's Sarcoma, Herpes Simplex, Hairy leukoplakia and 70% pts with candidiasis had CD4 < 200 cells/mm3. CDC includes the commonly diagnoses "sentinal lesions" of oral hairy leukoplakia and oral candidiasis among its criteria for diagnosis of symptomatic HIV infection
- Gerner (1988): Granuloma from an HIV pt had dramatically lowered CD4 T-cells (Th) compared to healthy controls
- Shugars & Wahl (1999 JADA): Oral transmission of HIV is relatively uncommon due to low salivary HIV titers, low number of CD4 target cells, anti-HIV antibodies and endogenous salivary antiviral proteins

**Gilles 1997**
Estrogen deficiency increases PARL size due to bacteria or IL1 (in rats)

**Steinman & Patterson 1982 JOE**
NSRCT has no effect on CV system of healthy pts. However, pulpal pathosis (eg., pain) may produce CV changes. Thus, it is the condition of the pt and not the tx that produces these changes.

**Wilburn-Goo 1999 JADA**
Review on acquired methemoglobinemia
Methemoglobin = Hb that has been oxidized. Due to chemicals that can act as oxidants
Can be associated with benzocaine, ciprofloxacin, lidocaine, prilocaine, procaine
Risk factors: heart disease, anemia, elderly, genetic deficiency of glucose-6-phosphate dehydrogenase, excessive doses of an oxidant
Should consider this if pt becomes cyanotic and have been exposed to local or topical anesthetics or other oxidants

Blanchaert 1999
Review on ischemic heart disease (IHD).  IHD MOA = insufficient oxygen delivery.  Symptomatic IHD = angina (symptoms: heaviness, pressure, smothering, choking).  Duration usually <5min.  Stable angina occurs during incr cardiac work; unstable occurs without initiating event and requires urgent investigation.  Tx includes 1) med tx (eg., beta blockers & ACE inhibitors to reduce after-load; calcium channel blockers & nitrates to reduce cardiac work); 2) percutaneous transluminal coronary angioplasty; and 3) coronary artery bypass. Dental rec: minimize stress, use profound LA.  For any pt with MI: give O2, nitrates, ASA and quick transfer to EMS

Coagulation - Hemorrhage - Bleeding Disorders  (see also: Hemostasis during periradicular sx)
- Barr (1994 DCNA): Platelet counts ave 150-400k/ml; spontaneous gingival bleed: <15k; minimum for sx: 50k
- Jolly (1994 DCNA): Hematologic disorders: Med consult; Factor VIII & IX at least 50% of normal; platelets > 50k; NSRCT may be possible without factor replacement; local thrombin may be needed; should med consult on prophy antibiotic before sx
- Patton (1994 DCNA): Prothrombin time = extrinsic; measures V, VII, X, prothrombin, fibrinogen. Want at least 150% of control values. Use this for pts taking coumarins since VII, X and proth. are all vitamin K-dependent
- Patton (1994 DCNA): Partial thromboplastin time =intrinsic (- charge collagen). Measures all factors except VII. Want at least 150% of control values. Could be elevated due to liver disease
- Patton (1994 DCNA): Bleeding time measures efficiency of vascular and platelet phases of coagulation eg., Modified Ivy, Duke
- Patton (1994 DCNA): Pts with bleeding disorder may report epistaxis, easy bruising, gingival bleeding
- Evans (1978 JADA): NSRCT can generally be performed without bleeding complications
- Hemophilia A accounts for 85% of all hemophilias; Factor VIII
- Mulligan (1988 JADA): Coumarin -attacks vit K-dependent factors, t1/2 = 44hr, Rec med consult for stop for 1-2 d before sx (monitor until PT, PTT within normal range); alternative: for minor procedures no change coum; just local hemostatic techniques
- Petrover (1990 J Perio): 2 Cases reports of using desmopressin to manage pts with von Willebrands disease during perio sx

Heparin
Griffith (1965): Chronic heparin causes osteoporosis
Kuraner (1999): chronic heparin in rat did not cause dentin resorption (but they did observe increased fibrosis).

Diabetes:
- Type I = IDDM. Juvenile onset autoimmune against beta cell; 15% of DM;
- Type II = NIDDM. Gradual adult onset = 85% DM cases; often obese; impaired insulin fxn similar to fasting state
- Rees (1994) the dentist is often the first professional to encounter pt with uncontrolled DM. Use morning appts, confirm breakfast; avoid excessive epi in LA (elevates BSL; no more than 1:100k epi)
- Goerig: Diabetecs have slower healing of PARL
- Goldman (1987 MCNA): Reasons for poor wound healing & infection are hyperglycemia leading to impaired phagocytosis, chemotaxis, adherance and killing of bacteria. Also see reduced collagen synthesis, capillary in-growth, fibroblast proliferation
- Chronic complications of DM: Macroangiopathy (CV, HBP), microangiopathy (blindness, kidneys) & neuropathy

Antrim 1978
Infection-related paresthesia: 2 case reports of mand molars necrotic with PARL that had paresthesia resolved by NSRCT.

Holtzman 1998
Multiple neurofibromatosis (peripheral type aka Von Recklinghausen disease); schwann cell and fibroblasts; skin Café-au-lait spots: Multiple PARLs noted with all teeth testing positive to vitality .  No tx given, other than ext third molar with root resorption

Morse 1997
2 cases reports of paresthesia after NSRCT.  Case 1: chloropercha overfill; tooth asymptomatic for 2.5yr; then PARL increased and swelling, pain and
paresthesia developed; resolved after Sx removal of lesion. Case 2: Formocresol pulpotomy; paresthesia started at 1 day; resolved after 7 weeks of dexamethasone (0.75mg #4 stat then taper) antibiotics and irrigation. Cc #1 = burning, painful, numblke sensation. CC #2 = numb lip

<table>
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<tr>
<th>Reference</th>
<th>Summary</th>
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<tbody>
<tr>
<td>Herman JADA p327 1997</td>
<td>Good review of coumarin anticoagulant therapy</td>
</tr>
<tr>
<td>Jolly DCNA 38(3):361, 1994</td>
<td>Reviews importance of med hx review</td>
</tr>
<tr>
<td>Mealey DCNA 1994</td>
<td>Complications of head &amp; neck radiation therapy</td>
</tr>
<tr>
<td>Muzyka and Glick JADA 128:1109, 1997</td>
<td>Reviews Hypertension pts</td>
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<tr>
<td>Brannon 1977</td>
<td>OKC needs to be removed entirely and biopsied. Hancock &amp; Brown 1986 reported OKC as PARL remaining after NSRCT (possible zebra)</td>
</tr>
<tr>
<td>Clark &amp; Allet 1980</td>
<td>Gorlin cyst. Mand molar/premolar Include in diff dx for PARL with opaque regions</td>
</tr>
<tr>
<td>Cohen 1984</td>
<td>Traumatic bone cyst can occur after extractions</td>
</tr>
<tr>
<td>Corio &amp; Crawford 1976</td>
<td>Benign cementoblastoma = radiopaque lesion attached to roots causes resorption.</td>
</tr>
<tr>
<td>Donnelly &amp; Koudelka 1986</td>
<td>Median palatal cysts with periapical lesions. Need occlusal film. Also reported by Nip &amp; Nguyen 1981</td>
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**Yagiela 1999 JADA**

**Vasoconstrictors are contraindicated in pts using**:
- Tricyclic antidepressants (enhanced sympathetic effects)
- Non-selective 8-blocker (eg, propranolol; may get hypertension)
- Halothane (cardiac arrhythmias)
- Cocaine (hypertension and arrhythmias)

Data suggest minor interactions between vasoconstrictors and the following:
- Antipsychotic (chlorpromazine)
- Adrenergic neuronal blocker (guanadrel)
- Thyroid hormone
- MAO inhibitor (eg., phenelzine)

**Hersh 1999 JADA**

Reviewed Drug Interactions with Antibiotics
Potential Major Interactions:
- Metronidazole and lithium (elevated Li)
- Erythromycin or tetracyclines with Digoxin (elevated Dig via inhibition of GI flora)
- Erythro, Clarithromycin or metronidazole with warfarin (decrease metabolism of warfarin)

**Trauma**

**Review AAE Guidelines for avulsed tooth**
Andreasen (1991 EDT): For crown fx with dentin exposure: rec Ca(OH)2, ZOE, acid-etched composite

**Ellis Classification of Trauma**
Type 1: enamel fx
Type 2 enamel and dentin fx without pulp exposure
Type 3 crown fx with pulp exposure
Type 4 root fx
Type 5 tooth luxation
Type 6 Tooth avulsion

**Sensitivity (“pulp”) Testing of Traumatized Teeth**
- Ohman 1965): CLASSIC! Immature teeth can have pulp survival and regeneration of nerve function after replantation. Especially, if have wide apical foramen. Dx with EPT is not good indicator of pulpal function in these teeth
- Fulling & Andreasen (1976): EPT on developing teeth is unreliable until root development is complete. Dry ice more reliable than EPT in immature teeth.
- Bhaskar & Rappaport 1973): After trauma, teeth may not respond to pulp tests. Vitality is defined by intact blood supply
- Mesaros & Trope (1997 EDT): Case report: traumatic injury to 8yo boy's two max centrals; only one weakly + to CO2 ice at 76 days; but Laser Doppler indicated both were vital. Endo tx not performed and teeth developed normally

Simon 1999 EDT): Reports hx of “silent” trauma (devitalized incisors due to trauma during intubation of general anesthesia. During pt interview, if you suspect trauma, also ask about general anesthesia sx within the last several years

Cvek 1978 Advocated partial pulpotomy (removing 1.5-2 mm pulp) even 72hr after traumatic exposure. Remember that need remove blood clot (Schoeder 1971). Cvek pulpotomy = 95% healing 3-15 yr follow-up.

Bergenholtz 1974 Retrospective study of 84 teeth with trauma and intact crowns and necrotic pulps. 54/84 had microorganisms present (primarily polymicrobial anaerobic).

Gier and Mitchell 1968 Demonstrated anachoresis in traumatized pulps after systemic iv injection of bugs. This may explain Sundqvist's observation of microbial infection in traumatized but intact teeth. Confirmed by Tziatas who demonstrated that bugs given iv accumulate in pulp beneath Ca(OH)2 direct pulp cap (which induced pulp inflammation).

Andreasen 1986 4% luxated teeth can develop transient apical breakdown (= PARL) which recovered over time. Teeth recovered with time. This may be example of transient “sterile” inflammation secondary to trauma.

**Horizontal Root Fractures**
Andreasen (1967): Root fx occur most often in the middle 1/3. Location of fx does NOT determine success (in contrast to conclusions of Bender & Friedland 1967); degree of coronal mobility is significant factor
Four different types of wound healing after root fractures:
- Healing with calcific tissue - occurs 1° when segments are close; neg percussion, pulp viable
- Healing with connective tissue - comparable to fibrous healing of bone, fx edges appear smoothed, calcification of canal systems
- Healing with bone and CT - interposition of bone between segments, pulp viable
- Healing with granulation - non-union healing, coronal segment necrotic & mobile, apical segment viable, wide gap, sinus tract
- Zachrisson (1975): 75% of teeth with fractured roots had repair. Degree of coronal mobility is critical. Many of these teeth have fairly good prognoses with correct tx, fixation and follow-up
- Michanowicz & Abou-Raas (1971): Pulp is not needed for root repair. Fx of the apical and middle thirds have better prognosis that cervical fx.
- Bender & Friedland (1983 JADA): Rec taking 3 radiographs with differing vertical angulation to view horizontal fx. The more apical the fx line, the better the prognosis.
- Feiglin (1995 DCNA): Tx of horizontal fx depends on location of fx (coronal, middle, apical 1/3) and apposition of the fragments (See pp294 of Problem Solving in Endo (3rd ed) by Gutmann for nice summary of tx options); overview: ext coronal segment if fx communicates with gingival sulcus; otherwise splint if necessary and observe for possible NSRTC if necrosis occurs.

### Vertical Root Fractures

**Dx vertical root fx is difficult because:**

- Xrays often not diagnostic in initial stages (Matusow 1987 JADA),
- symptoms may mimic TMD, sinus, headaches ear ache (Schweitzer & Gutmann 1989 IEJ), and
- fx lines not always discernable (Cameron 1976 JADA).

**Clinical findings of a vertical root fracture**

- aka "Cracked tooth syndrome" (coined by Cameron (1964 JADA) and aka "split tooth" [coined by Silvestri 1976]:
  - Pain on biting
  - Pain on release of biting
  - Sensitivity to thermal changes
  - Persistant dull pain
  - Pain to selective cuspal percussion (eg., Tooth Slooth)
  - Presence of fracture lines by transillumination or by pulpal floor staining with methylene blue
  - Deep narrow periodontal pockets
  - "halo" PARL - more periradicular than just periapical

Andreasen 1981

Do NOT do Endo before implanting avulsed teeth. Keep root moist. Replant quickly!. Avulsed teeth should have the pulp removed after the PDL has healed a little. Ca(OH)2 stops inflammatory resorption. Saline is better than saliva and >> water (hypooosmotic)

### Splinting

- Andreasen (1985): Monkey study: recommends use of semi-rigid splint for replantation
- Antrim & Ostrowski (1982): Described technique for splinting with monofilament fishing line
- Burke (1976): Tx avulsed tooth after splinting with Ca(OH)2 to minimize inflammatory resorption
- Oikarinen & Gundlach (1987): Recommend shorter splinting (<42 days) to minimize external resorption and post-pone endo for 2 weeks. Replant ASAP to preserve the PDL.

Andreasen 1985

**Prognosis of luxated permanent teeth for developing necrosis:**

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<tr>
<th>Type</th>
<th>Proportion of Pulp Necrosis</th>
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<tr>
<td>Concussion</td>
<td>3%</td>
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<tr>
<td>Subluxation</td>
<td>6%</td>
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<tr>
<td>Extrusion</td>
<td>26%</td>
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<tr>
<td>Lateral luxation</td>
<td>58%</td>
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<tr>
<td>Intrusion</td>
<td>85%</td>
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In general, teeth with complete root development have poorer prognosis for pulp vitality (necrosis usually seen in 3 weeks) than teeth with incomplete root development (which showed 34% pulpal healing).

Andreasen 1989

In teeth left out of mouth for >60 min and received NSRCT within 3 weeks, developed replacement resorption of the root

Andresen 1989

**Four different types of root resorption after luxation injuries:**

San Antonio Guide to the Endodontic Literature version 2.34
| Also: Barrett & Kenny 1997 EDT | - External surface resorption = small resorptive cavities in cementum  
- Internal surface resorption  
- Internal tunneling resorption  
- Transient apical breakdown  
- Replacement resorption = ankylosis. Union of alveolus and dentin (due to removal of PDL)  
- Inflammatory resorption = bowl shaped defects that penetrate dentin (dentin has Howship's lacunae with occasional osteoclasts) |
| Transport medium for avulsed teeth: | - Blomlof & Lindskog (1983): Milk is ok (Skim milk is best of the milks: Harkacz and Walker (1997))  
- Andreasen (1981) Saline > saliva > water  
- Sae-Lim & Trope (1999 EDT): Viaspan + dexamethasone (16ug/ml) > Viaspan |
| Dumsha & Hovland 1982 | Extrusive injuries had pulpal necrosis in 98% of cases |
| Torneck 1982 | Trauma to primary teeth may alter develop of permanent teeth |

Avulsion & Inflammatory Resorption (IR) has a Bacterial Component: Tx Avulsions with Antibiotics!  
- Loe (1961): CLASSIC: PDL vitality is important in success of replantation and the presence of the rests of Malassez seem to be important in preventing ankylosis. Replant quickly without damaging the PDL. The PDL is the tissue to save, so do endo after replantation  
- Andreasen (1981 JOE): IR due to 5 factors: injury to PDL, initial external resorption exposing dentinal tubules, presence of necrotic & infected pulp communicating with resorbed area via dentinal tubules, possible presence of bacteria on PDL, age & maturation of tooth  
- Hammarstrom (1986): Monkey study: showed systemic amoxicillin reduced IR after avulsion. Recommended giving antibiotics when replanting avulsed teeth  
- Trope & Moshonov (1995): Long term Ca(OH)_2 tx is more effective that 1 week Ca(OH)_2 tx in teeth with established inflammatory root resorption  
- Sae-Lim & Trope (1998 EDT): Dog study (necrotic model; extract, shave cementum, replant; 6m follow-up): Controls had 72% inflammatory resorption. In contrast, systemic tetracycline (day of ext & 6d after) had 33% inflammatory resorption. Amoxicillin had 43% inflammatory resorption. Rec considering tetracycline as an alternative to amoxicillin after avulsion injuries. In Trope's dog model, only see IR if replant necrotic teeth with denuded cementum; no IR occurs if replant obturated uninfected teeth.  
- Sae-Lim & Trope (1998 EDT): Dog study (NSRCT; extract, bench dry 1hr, replant; 3-4m follow-up): Controls & systemic amoxicillin ~11% healing. In contrast systemic tetracycline (X6d) had 35% healing (5 of 11 teeth had >50% complete healing on surfaces; vs 1 of 8 for controls and 1 of 11 teeth for amoxicillin)  
- Nishioka & Suda (1998 EDT): Perform ed tooth replantation in Germ-free vs conventional rats. Conventional rats: necrotic pulps and inflammatory resorption was noted. Germ-free: Pulp filled in with "bone-like" material & roots had no inflammatory resorption (did get higher incidence of ankylosis). The Nishioka study is consistent with the studies by Andreasen, Hammarstrom, Cvek, Trope and Lindskog in implicating bacterial contribution to inflammatory resorption. On the other hand, ankylosis can occur even in strictly aseptic conditions.  

Should you tx teeth with Calcific Metamorphosis?  
- Holcomb & Gregory (1967 OOO): Only 7% of teeth with calcific metamorphosis develop problems, so no prophylactic tx is indicated  
- Robertson & Andreasen (1996 JOE): only 8.5% demonstrating pulp canal obliteration after trauma develop necrosis so prophylactic NSRCT not indicated  
- Akerblom & Hasselgren (1988 JOE): Performed NSRCT on teeth with calcific metamorphosis with PARL and got 62% success even when they could not C&S down to the apical area. Therefore, do NSRCT first and evaluate for success  
- Smith (1982 OOO): Review of lit on calcific metamorphosis. Surveyed endodontists; 50% stated that they would tx
## Endo-Pedo & Vital Pulp Therapy

### Andreassen and Riis 1978

**Monkey Study:** Induced pulpal and periradicular inflammation of primary teeth. No effect on developing permanent teeth (study only 6 weeks long)

### Torneck 1982

Trauma to primary teeth may alter develop of permanent teeth

### Direct Pulp Cap with Ca(OH)2

- Baume & Holz (1981): Direct pulp caps with Ca(OH)2 have 90% success IF teeth are asymptomatic and vital. Must have hermetic seal. But, Goldberg (1984) does point out that the dentinal bridge formed by Ca(OH)2 is porous and permeable.
- Hebling (1999): Human direct pulp caps: Ca(OH)2 produced initial coagulation necrosis then odontoblast-like cells organized underneath coagulation necrosis by 7d. Saw apparent complete dentin bridge by 60d. But, All-Bond2 showed no repair.

### Hu & Taum 1998

TGF-beta enhances formation of reparative dentin in rats by 3 weeks and may have utility for direct pulp capping material. (No reparative dentin potential found for EGF, FGF, IGF, PDGF)

### Rutherford 1993

Human osteogenic protein (= hOP-1 = BMP7) also reported to evoke reparative dentin

### Coll & Sadrian 1996

Primary teeth ZOE pulpectomies had 78% success rate; significantly better when <1mm root resorption

### Cvek 1978

Advocated partial pulpotomy (removing 1.5-2 mm pulp) even 72hr after traumatic exposure. Remember that need remove blood clot (Schroder 1971). Cvek pulpotomy = 95% healing 3-15 yr follow-up.

### Diagnostic Tests in Primary Teeth

- Bernick (1959): Nerve endings degenerate with onset of root resorption - predispose to false negative responses to EPT/ice
- Magnusson (1980): EPT is unreliable. Ice may be poor test. Reports that changes in eye contact or squinting may be best way to evaluate response to pulp sensitivity testing

### Glutaraldehyde Pulpotomies:

- Fuks 1990: Clinical study showing 2% glutaraldehyde in pulpotomies of 1st molars gave 2 year 72% success rate.
- Garcia-Godoy (1987): Glutaraldehyde is less toxic than formocresol
- Pearse (1980): major problem: glut is not stable; has short shelf-life

### Formocresol Pulpotomies

- Full (1979): Formocresol pulpotomies have good success (DM = pain) at 18 months on abscessed teeth
- Sipes & Brindley (1986) point out that formocresol is mutagenic and carcinogenic!
- Longwill & Marshall (1982): Pediatric use of formocresol does NOT sensitize the child
- Loos (1971): Rec diluting Buckey’s formocresol 1:5. Still see good clinical results with less cytotoxicity

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