	A Guide to the Endodontic Literature
	Success & Failure:
Authors	**Description**
European Soc. Endodontology (1994 IEJ):	**Definition of Success**: 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.
AAE Quality Assurance Guidelines	**Objectives of NSRCT (= nonsurgical root canal treatment)** • Prevent adverse signs or symptoms • Remove RC contents • Create radiographic appearance of well obturated RC system • Promote healing and repair of periradicular tissues • Prevent further breakdown of periradicular tissues
	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 1week (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 microganisms; 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 therapeutic 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

Green, Walton, 1997	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.
Ingle, Beveridge,	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&S).
Kerekes, Tronstad 1979	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.
Bergenholtz 1974	Retrospective study of 84 teeth with trauma and intact crowns and necrotic pulps. 64% had microorganisms present (primarily polymicrobial anearobic).
Akerblom, Hasselgren 1988	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.
Ray & **Trope** , 1995	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.

Augsburger, Peters 1990	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.
	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 mult-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)
Long-term Comparison Oliet 83 Pekruhn 86 Trope 99	**1-Step vs Multi-Step: Long-term Comparison** • 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

Freidman 95 Sjogren 97	or just short of the radiographic apex. • Pekruhn (1986): Evaluated failure with 1steps (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: 1week 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 regssion 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 1year. Therefore, 1yr follow-up predicts long-term success
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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 • Persistant Intraradicular infection (Nair 1990)

	• 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 tublues after 14-21 days • Enterococcus faecalis in 33% failed NSRCTs (Molander 1998 IEJ) & in 60% failed cases reported by Siren (1997) • Actinomycosis israelii found in two case reports of failed NSRCT. Had to be eliminated by Sx (Sundqvist 1981 OOO) • <u>Persisitent Extraradicular infection</u>, see Simon's review on POP for general info and nice figs • Nair (1984) Actinomyces isrealii . Also reported by Happonen (1986): 81% samples contained actinomyces, 62% contained arachnica • Sjogren (1988) Proprionibacterium proprionicum (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).
Grung 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 first 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 regssion 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
	Studies showing reduced success of NSRCT with apical periodontitis: Success (%): <u>No PARL</u> <u>PARL</u> <u>N</u> 1. Molvern & Halse (1988) 91% 68% 207 2. Akerblom, Hasselgren (1988) 98% 62% 64 3. Sjogren (1990) 96% 86% 471 4. Friedman (1995) 93% 69% 142
	The NSRCT success rate for <u>necrotic teeth vs vital</u> 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% Sources; Molvern & Halse 1988; Sjogren 1990 and Frideman 1995 (N = 569). • Bergenholtz (1979 Scan JDR): Classic on re-tx. Group being re-tx for prosth indication (ie, not failing) still 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 matieral: biocompatability, apical sealability and handling characteristics
- Briggs & Scott (1997): Re-tx is preferable over endo sx ("evidence based" analysis). Also, Moiseiwitsch & **Trope** 1998.
- Lin (1996 IEJ): Discussed periradicular curretage. 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:

1. Grossman (1982): 70% success at 5 yrs
2. Keller (1990): 91% success
3. Bender & Rossman (1993): 81% 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 IEJ	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.

	History and Rationale
Hudson 1862	Credited with performing some of the first NSRCT (obturated with gold) in the US (editorial in Dental Cosmos)
Price 1901	Discussed use of radiography in performing NSRCT and evaluating success-failure
Callahan 1914	Introduced a technique of filling root canals with a rosin-gutta-percha material as well as theorize on proper filling needs of a root canal.
Hatton 1922	He advocated confinement of instrumentation to the inside of canal. He also determined histologically, that repair was possible at the root apex
Blayney 1930	After his findings, the author offered the final conclusions: 1.) degenerative processes around root ends do not always indicate extraction, 2.) in many cases, repair promptly takes place after treatment and stays healthy for years and 3.) root canal treatment will only succeed when the practitioner is willing to adopt methods in accord with the biologic forces involved.
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	Implanted 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 therfore, 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 theroy 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 Immun* 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 *Stroke* 28:1724	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)	Examined 2400 teeth. 24-60% of teeth have lateral canals (highest in 2nd premolars and MB canals of max molars) Sudden narrowing of RC system on radiographic exam is good indication of canal bifurcation <u>MAXILLARY TEETH:</u> 1st Premolar 62% Type IV (2 canals), 18% Type II (2-1 canals),), [69% have 2 canals at apex) 2nd Premolar 48% Type I (1 canal), 22% Type II (2-1 canals), 11% Type IV (2 canals) 1st Molar: MB 45% Type I (1 canal), 37% Type II (2-1 canals), [18% have 2 canals at apex) <u>MANDIBULAR TEETH:</u> Central 70% Type I (1 canal), 22% Type III (1-2-1 canals) Lateral 75% Type I (1 canal), 18% Type III (1-2-1 canals) Canine 78% Type I (1 canal) 1st Premolar 70% Type I (1 canal) 2nd Premolar 98% Type I (1 canal), 1st Molar: M 12% Type I (1 canal), 22% Type III (1-2-1 canals), 43% Type IV (2 canals) 1st Molar: Distal 70% Type I (1 canal), 15% Type II (2-1 canals), 8% Type V (1-2 canals)
	Rationale for Instrumenting 0.5-1mm short of the radiographic apex: <u>Morphological Studies:</u> • 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 <u>Pulp – PA Pathology Studies:</u> • 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. <u>Outcome Studies:</u> • 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). THERFORE 71% OF MAX 1ST MOLARS HAVE TREATABLE MB2! This value splits the difference between *Weine* (50% incidence of MB2; CLASSIC: 1969 study) and *Kulid & 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 vertex 83% of the cases sometimes to a distance of 2-3 mm. Proves can't see curves on the radiograph.
Chohayeb 1983	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.
Wilcox & Walton 1989	When cutting access in crowned tooth, remember that pulp chamber is in center of crown
Leeb 1983	Remove cervical ledges over canal orifice during access prep to enhance straight-line access
Lowman, Burke, Pelleu 1973	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
Trope & Elfenbein 1986	Pts of African-American descent have 3X > incidence of 2 canals / 2 roots in mand premolars
	Dental Anomalies
Sabala , Benenati , Neas 1994	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 abberations are present, valuable information may be acquired through the evaluation of the contralateral tooth.
De Smit , Jansen & Demaut 1984	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.
Hulsmann 1997 IEJ	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” & 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&S difficult (consider Ca(OH)2, US files, thermoplasticized GP).
Froner 1999 EDT	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
Turell & Zmener 1999	Described NSRCT in fused mand molar
Rotstein, Stabholz, Heling, Freidman 1987	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.

Senia Regezi 1974 Also: Yip. 1974	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 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%). • 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.

Lovdahl & Gutmann 1980	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
Bramwell & Hicks 1986	Described use of oraseal or Cavit to seal leaky RD
Calcified Canals Gutmann Stamos Leeb Schindler	**Calcified Canals** • Wilcox & Walton (1989): Pulp chamber is in center of crown • Gutmann: 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) • Leeb (1983) Remove cervical ledge near orifice • RC Prep (Premier Dent Products) • Stamos (1985) Rec use of US files to gain access and file calcified canals; and to remove alloy or particles packing RC system • Schindler (1988): If cannot bypass calcification, then C&S & obturate to level of calcifiction; place on recall for potential Sx • Glyoxide = 10% carbamide peroxide in glycerol; Marion Labs • Flexofiles are available in 1/2 steps ("Flexofile Golden Mediums"; LD Caulk) • Weine (1970): Rec customize files by cutting 1mm from #10 to make #12 (However- cutting end vs pilot tip, etc) • EndoZ bur - safe ended carbide bur to enlarge access (LD Caulk) • Ngai (1986): Described use of US files to bypass separated instruments in canals • Weine (1975): 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).
Pliet & Sorm 1973	Triangular instruments cut more efficiently than square files
Powell, Simon and Maze 1986	A comparison of the effect of modified and nonmodified instrument tips on apical canal configuration, J Endod , 1986;12:293-300
Walia, Brantley, Gerstein 1988	1st description of NiTi (“nitinol”) files
Wildey , Senia 1989	1st description of Canal Master
	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)
Ingle 1961	Ingle JI, A standardized endodontic technique utilizing newly designed instruments and filling materials, Oral Surg Oral Med Oral Pathol , 1961;14:83-91
Short & Baumgartner 1997	Lightspeed and Profile were faster than hand filing and kept files centered in canal better that ss hand files
Pruett, Clement, Carnes 1997	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.
Dederick & Zakarriasen 1986	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).

Love 1996	Bacteria can invade up to 150-250 um into dentinal tubules. Confirmed by Sen (1995): bugs grow 150um into tubules. 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).
Klevant 1983 IEJ **	Chemomechanically debrided RC systems of 86 human teeth and left un-obturated for 2 years. Radiographic exam showed significant decrease in PARLs in C&S-unobturated and C&S-obturated teeth. Thus, reject "hollow tube" theory for breakdown of tissue fluid inducing PA lesion. (Should point out that even though C&S produced significant radiographic healing of AP, better healing was observed in C&S-obturated group. Also reported by Donnely 199, Weine, and others (see Klevant for refs)
Jahde & Himel 1987	A small amount of inflammation and localized bone necrosis occurs with file overextension .
	File cutting tips are responsible for ledges, zips and perforations (ie, the tip is an effective cutting region). This is consistent with study by Powell & Simon (1988) who showed that Flex-R produced less transportation regardless if used balanced-force or stepback
Roane & Sabala 1984	A CW rotation of a file has greater chance of separation than a CCW rotation. Confirmed by Seto & Harrington 1988
	Apex Locators • Suzuki (1942) reported that PDL and oral mucosa have a constant electrical resistance of ~6.5 kOhms • Sunada (1962): Classic! Applied Suzuki's idea to develop an apex locators • Old style = resistance (ex: NeoSono, Formatron) • Next generation = dual frequency (ex: Root ZX, Endex) • Pagavino (1998): Root ZX has 83% accuracy ± 0.5mm (includes teeth with lateral foramina) • Dunlap & 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). • Fouad (1993): Apex locaters ok on pts with a pacemaker (even though Root ZX manual says not to use it on pts with pacemakers) • Beach & Hutter (1996): Case report of using apex locator on a pt with a pacemaker • Fuss (1996): Describes use of Apex Locators to locate perforations • Ibarrola (1999 JOE): Preflaring canals permits WL files to reach apical foramen more consistantly with Root ZX.
Ahmad 1987	Most of the benefits of ultrasonics are due to acoustic streaming rather than cavitation.
Huque & Iwaku 1998 IEJ	Ultrasonics with 5.5% NaOCl is effective in eradicating bacteria from infected dentin (artifical smear layer infected with Actinomyces, Fusobacterium, Streptococcus)
Haikel 1998 JOE	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
Haikel 1999 JOE	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…
Walton 1976	Tapering preparation permits better debridement of apical preparation, reduces over-instrumentation of the foramen and improves ability to obturate

Abou-Rass, Frank & Glick	Classic: describes anticurvature filing. Defined danger and safety zones
Gambi & DelRio 1995	NiTi files may fxn best when used in reaming or rotary fashion (since less transportation and canal deviation)
Weine & Kelly 1975	Termed "apical zip", discussed elbow, teardrop apex and hourglass shape. Argued against reaming (before NiTi).
Mullaney DCNA 1979:	**Step-back (Telescopic Technique)** • Determine WL & develop apical stop to #25 • Step-back by shortening 30, 35, 40 in 0.5 or 1 or 2mm increments • Recapitulate with #25 • Coronal flare with #2 & 3 Gates-Glidden
Goerig JOE 1982	**Step-Down technique.** • Passively use #15, 20, 25 Hedstrom in coronal 2/3 of canal system; irrigate • Coronal flare with #2 & 3 Gates-Glidden • Establish WL and prepare apical seat with stnd serial filing • Step-back to blend apical and coronal segments • Recapitulate **The crown-down pressureless technique** (Morgan & 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
Roane & Sabala 1985 JOE	**Balanced force technique** (use FlexR files (Moyco Union Broach) or Flexofile for non-cutting pilot tips of triangular file) • Use Crown-Down to establish radicular access • Rotate straight file CW from 90-180° with light apical pressure to engage dentin • Shear dentin by 120° CCW rotation with apical force, flexing it to conform to canal curvature • Continue until get adequate apical enlargement at WL • Inspect files frequently; do not go beyond #35 in curved canals
Fava 1983 JOE	**Double-flared technique.** • Passively use larger-smaller files in coronal 2/3 of canal system; irrigate • Establish WL with small K file. Serial file to prepare apical stop and then step back to blend with coronal step-down flare • Circumferentially file with master K file
Torabinejad 1994 OOO	**Passive step-back technique:** • Establish canal patency with small K file at WL then passively instrument with larger K files • Coronal flare with #2, 3 and possibly #4 GG in coronal 1/3 • Confirm WL (since coronal flare and removal of curvatures often reduces WL) • Increase straight line access with careful re-work with GG • Serial file to prepare apical stop and then step back to blend with coronal step-down flare
Wilcox and Walton 1989	Studied access of molars: DB orifice is slightly distal to buccal groove. Rec start access prep centrally, and not at MMR.
Instrumentation and Removal of Bugs	**Instrumentation and Removal of Bacteria** • 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 & Trope '98 Siquerra '99	• Dalton and **Trope** (1998 JOE): n=48 MB canals of mand necrotic molars with apical periodontitis (AP defined as PARL) were found to be uniformally 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	**Infected Dentinal Tubules** • 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 tublues after 14-21 days. Presence of a smear layer delayed, but did not prevent, antimicrobial effects of medications. • Sen (1995): bugs grow upto 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 tublues (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	**NaOCl** • 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-anerobes, microaerophiles, obligate anerobes). 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	**NaOCL Accidents** • 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 & NaOCl** • 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 '97 Glickman '95	**Smear Layer:** • Ishley & ElDeeb (1983) Sealer was more important that the type of obturation used (McSpadeen vs 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 enchanced adaptation of thermoplasticized GP into dentinal tubules without smear layer • 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). ALSO by **Glickman** (1995 IEJ): SEM AH26 & LC ± 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 Safavi '93 **Trope '97** Messer '93 Fava '99	**Ca(OH)2 Properties:** • 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- • Sigurdsson (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 (aqeous, 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	**Chlorhexidine** • **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 (ie 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. • Lindskog & Blomlof (1998 EDT): Monkey study: Infected pulps, extract, scrape cementum. Intracanal application of chlorhexidine (10% soln for 4w) significantly reduced inflammatory resorption vs controls. • Leonardo &Ito (1999): 2% chlrohexidine 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% clearance 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 forman, 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 • **Hartwell** & Barbieri (1991): Found wide variations among finger spreaders and accessory GP cones. If one doesn't fit - grab another accessory cone. • Dang & Walton (1989): The hand spreader (D11) cased more root distortion and vertical fx then the B finger spreaders. Root fx may be delayed after obturation. Confirmed by Lertchirakarn & Messer (1999). • Joyce & West (1998): NiTi spreaders produces less stress during obturation than SS spreaders (NiTi distributed stress over larger area). May imply less risk of vertical root fracture during obturation. • 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 ~47C. 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) • Goldman & Schilder (1985): GP thermal study: beta to alpha phase at 46-48C and GP in alpha phase to amorphous phase at 56-62C. A small volume reduction occurs when cooling to 37C (so be sure to vertically condense). • **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 >65C 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:** • Tamse (1986): GP Solvents: chloroform > xylene >> Endosolv-E > orange turpene • 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, coherance, 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
Mickel '99 Shalhav '97 Brown '94	**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 hypersensitivy 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 • Kleirer (1988 EDT): Sargenti: painful dysethesia of the IAN after use of paraformaldehyde paste • 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 • Leyhausen (1999 JOE): AH26 cytotoxicity due to release of formaldehyde from the epoxy resin. Not seen with AH26 Plus. • Curson & Kirk (1968 OOO): ZOE sealers well tolerated by PA tissues ALSO: Augsberger & Peters (1990) • 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 paraformadehyde). 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 • Antrim (1978): Infection-related paresthesia: 2 case reports of mand molars necrotic & PARL: paresthesia resolved by NSRCT
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 Spandberg Allard Kleier	**Sargenti** • 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 • Cohler & Newton (1980): demonstrated short-term severe cytotoxicity of Sargenti paste in monkeys • 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 • Kleirer (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
<u>Ca(OH)2</u> Frank (1966) Cvek (1972) Hicks (1987) <u>MTA</u> FDA Indications	**Apexification / Apical Barriers** <u>Ca(OH)2</u> • 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 • Kleirer (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 <u>MTA</u> • FDA Indications for MTA: perforation repairs (non-communicating), apexification, root-end filling, pulp capping

Shabahang & Torabinejad '99 Torabinejad & Chivian Schwartz & Walker <u>Other Materials</u> Tronstad Yoshida <u>Other Issues</u> Katebzadeh & **Trope**	• 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 <u>Other Materials (dentin chips, FD dentin, bone)</u> • 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). • Yoshida (1998 JOE): Dog study: Histological eval of FD allogenic dentin powder & true bone ceramic (=incinerated bovine bone) both acceptable as apical barriers (obturated with LC) DM = TRAP activity (tartrate-resistant acid p'tase), marker for osteoclasts <u>Other Issues: Internal Bonding</u> • 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)
	Temporary and Final Restorations
Garguilo & Orban 1967	<u>Biologic width</u> 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 DelRio '78 Harris '76 **Cunningham '92**	**Cavit** • Anderson & Powell (1988) 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

	ridges) by 63%.
Sedgley & Messer 1992	Endo tx teeth are not more brittle than vital teeth
Randow & Glantz 1986	Demonstrated that a RCT-tx tooth could have twice as much force placed on it before pain as compared to vital tooth. THL: pulp may provide some protective sensory information when chewing to reduce risk of fracture.
Trope '85 Nayyar & Walton '80 Stockton '98 **Jeansonne '98** Kvist '89 Lemon '81 Kleier '99	**Posts:** • **Trope** (1985): argues that post space preparation weakens the tooth. AE composite in ant teeth strengthened them more than placing a post • Nayyar & Walton (1980): 4yr study (n=400) demonstrating succcess of amalgam core with extension into coronal 2-4 mm of root canal systems. This is a viable alternative over posts. This technique can be improved by use of amalgam bonding agents (= 4-META) such as Amalgambond (Parkell Inc) [Cooley 1991] • Stockton (1998 EDT): Rec that posts are no longer mandatory for restoring endo-tx teeth. Alternatives include resin-bonded cores. Problems include potential root perfs, costs, • Dean, **Jeansonne** & Sarkar (1998): In vitro central incisors posts & composite core: carbon posts (C-Post) had no root fractures vs parallel posts (50%; ParaPost) and tapered posts (50%; PD posts) • Kvist (1989): Examined 852 roots with about 50% containing posts. PARLs were present on 16% of teeth with posts and 13% teeth without posts (therefore, posts do not compromise periradicular healing). However, posts with only 3mm GP remaining have greater incidence of apical periodontitis. Need at least 4-5mm of GP. • Bourgeois & Lemon (1981): NS difference in apical leakage immediate vs delayed post preps using either ZOE or AH26 sealer • Kleier (Feb1999JOE): Titanium posts have similar radiodensity as GP, thus difficult to dx on radiograph. Hints: Look for straightened coronal RC system, slight canal enlargement at post:GP junction, slight radiolucent gap between post:GP, surface patterns of post (ie, serrated edges) • 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 • Mattison (1984): Leave 5-6 mm of GP when making post-space. Neagley (1969): recommends leaving 4mm of GP. • Haddix & Mattison (1990): Recommend remove GP with hot instrument. GG burs tend to "pull" GP and may disrupt apical seal. Conversely, Kwan & Harrington (1981) showed GG burs removal of GP immediately after obturation avoids the leakage problem • 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) • Tjan & Nemetz (1992): Eugenol from RCT sealers interferes with post bonding cements (eg., Panavia EX). • 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 • Sorensen & Martinoff (1984): Anterior teeth do not require post and crown. But premolars and molars require cuspal protection
	!

	Perforation, Resorption, Procedural Accidents, Re-TXs, Post Removal
Genl issues Weine '82 Fuss '96 Internal Matrix Lemon '92 **Hartwell '93** Petersson '85 Cavit Harris '76 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 OOO): 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 hydroxyapatitie [Lemon 1992], decalcified FD bone [**Hartwell** & England 1993], Ca(OH)2 [Petersson 1985] or CollaCote [Rosenberg 1995; IFEA Congress] 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 JOE): MTA superior to amalgam or IRM in perf repairs • Nakata & Baumgartner (1998 JOE):MTA better than amalgam in preventing leakage of *F. nucleatum* past furcal perf repairs • 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 adapation of MTA to perforation walls when first placed Gelfoam as an internal matrix Ca(OH)2 and other Materials • Balla & Lomonaco (1991): Monkey study on perf repair: neither amalgam, tricalcium phosphate nor Life induced hard tissue formation • 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 IEJ): Case report: used Ca(OH)2 as internal matrix and sealed perforation with EBA • Poi & Martin (1999 EDT): 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 • Aladiany & Himel (1994 JOE): Used plaster of paris as internal matrix to repair perfs with GI or amalgam
Crump & Natkin '70 Glick '86 Gutmann '79 Krell '84 & '86 Johnson '96 Stamos '88 Spriggs '90 Suter '98 Eleazor '99	**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 • Cauldfield 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; 1986) & Masserann (1959): Masseran kit: uses tubular trephine drills and tubular extractor with screw-down locking rod. Can increase force with this kit using impact-type crown-remover onto locking rod • Krell (1984) & Ngai (1986): 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 • Berbert & Filho (1995): Ultrasonics can reduce force needed to remove posts • **Johnson** & Leary (1996): Ultrasonic removal of Paraposts takes 16 min • Feldman & Solomon (1974): Describes use of Trephan 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 ultrsonic 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 Thermofill obturators • Metzger (1995 JOE): described removal of overextended GP cone by careful engagement with Hedstrom file
	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 Thermofill 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)
Burke 1976	Avulsion: Tx avulsed teeth with Ca(OH)2 after splinting in order to minimize inflammatory resorption
Gartner & Mack '76 Tronstad '88 Caliskan '98 Schwartz & Walker '99	**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. • Mattison & Gholston (1983): Pt with hx of external resorption due to ortho. Ca(OH)2 tx stopped resorption and mobility. • 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 peforated • 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 roor resorption caused by: trauma, periradicular inflammation, ortho, bleaching
Frank & 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).
	Pain, Endo Emergencies, Flare-ups
Absi & Addy 1987	Teeth with dentinal hypersensitivity have greater density and diameter of tubules

Ahlquist 1994	Human psychophysical study for A-delta fibers and C fibers. Confirmed by another psychophysical study: *Edwall* 1986.
Brannstrom 1967	Heat causes inward fluid movement in tubule and cold causes outward fluid movement. "Its cold out"
Fusayama 1988	Bonded composites can reduce dentinal hypersensitivity. Confirmed by Brannstrom JADA. And by Nordenvall & Brannstrom 1980
Mullaney & Howell 1970	Did not find nerves in necrotic pulps. Concluded that pain upon entering necrotic teeth may be due to apical compression.
Chen 1997 EDT	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
Negm 1994 JOMS	Intracanal diclofenac and ketoprofen effective analgesics for controlling endo pain
Rogers & Johnson 1999	Intracanal application of ketorolac (3mg) and dexamethasone (0.4 mg) were better analgesics than plbo at 12h; ketorolac also > plbo at 24h; Oral ibuprofen ns different from plbo. N=48 pts with vital cases NSRCT C&S. Pain by VAS.
	Air Emphysema • Shovelton (1957) reported 13 cases of air emphysema • Falomo (1984): Air syringe into root canal can cause emphysema . • Am Dent Assoc (1992): In 1992 ADA rec using remote exhaust handpieces when removing bone or tooth structure during sx. • 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 stnd 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

	• Replogle & Reader (1999): Stabident 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 **PDL Injection:** • Kim (1986): Proposed that PDL injection works by vasoconstriction of blood flow and therefore, should only be used for endo & extraction - not to be used for vital teeth for restorative treatment. • Pashley (1986): PDL is an intraosseous injection with significant CV effects. Confirmed by Walton (1986). • Walton & Garnick (1982): PDL injection does not harm the ligament, so it is not a ligament injection! Requires backpressure.
Ehrich, Dionne & Hutter 1997	Triazolam 0.25mg reduced anxiety better than diazepam 5mg or plbo in endo patients. Confirmed by Kaufman, Hargreaves & Dionne (1993).
Flath, Dionne & Hicks 1987	Pre-op flurbi reduced post-op pain. ALSO, Pulpectomy alone reduces post-op pain
Krasner '86 oral Marshall '84 im Rogers '99 intracanal Kaufman '94 PDL	**Are Steroids Effective?** • Glassman & Krasner (1989 OOO) Lg doses of dex (12 mg po) in vital asymptomatic cases are better than plbo for reducing post-appt pain • Chance & Lin (1987): Cortisone on paper point in vital cases reduced pain (but- did it induce bacteremia via paper point?) • De Deus & Han (1967): Placed [14C]-cortisone on pulp and found in liver & kidney. Pharmacokinetics show absorption from pulp • 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). • Rogers & Johnson (1999 JOE): Intracanal application of ketorolac (3mg) and dexamethasone (0.4 mg) were better analgesics than plbo at 12h; ketorolac also > plbo at 24h; Oral ibuprofen ns different from plbo. N=48 pts with vital cases NSRCT C&S. Pain by VAS. • Kaufman (1994 JOMS): Intraligamentary injection of slow-release methylprednisolone effective reducing post-endo pain • Krasner & Jackson (1986 OOO p187): randomized plbo study (n=50): oral dex > plbo for reducing post-endo pain • Marshall (1984 JOE & 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 • Moskow (1984 OOO): intracanal steroids reduced pain. Study used vital cases • Gallatin (JOE 1998 24:280): intraosseous injection of Depo-Medrol (1ml = 4-mg) sig reduced pain in irreversible pulpitis
Differential Dx <u>Referred Pain</u> Reeh & ElDeeb '91 Drinnan '87 Schwartz & Cohen '92 Sharav '84 <u>Neuropathic Pain</u> Dumas '99 Sigurdsson ' 95 Gutmann '96 & '99	**Differential Dx of Non-Odontogenic Pain:** <u>Referred Pain</u> • Reeh & ElDeeb (1991) Muscle trigger point referred to tooth and mimicked endo involvement. • Kleier (1985): Muscle trigger point referred to tooth and mimicked endo involvement • Drinnan (1987 DCNA): MI. ~10% MI cases have pain referred to mandible (Sandler (1995 JADA)). Coronary insufficiency referring to mandible Batchelder (1987) • Schwartz & Cohen (1992 DCNA): sinusitis. Need exclude possible endo-antral syndrome (Selden 1999) • Sharav (1984): Acute dental pain can be referred to opposite arch on same side (eg., Left Max = Left mand) <u>Neuropathic Pain</u> • 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 • Tidwell & Gutmann (1999 IEJ): Herpes with cc of toothache. Look for skin rash. Also: Sigurdsson 1995. Intense herpes infection-induced symptoms can lead to pulpal necrosis (Goon (1988 JADA). • Vickers (1998): Atypical odontalgia. Can be phantom tooth pain, RSD, psyhological • Drinnan (1987 DCNA): AFP

Cancer Glaser '97 Selden '98 Todd '87 Other: Ratner & Langer '86 Drinnan '87	• Battrum & Gutmann (1996): Phantom tooth pain. Claimed incidence of 3% of popln undergoing extirpation. Also: Marbach (1978) Cancer • Glaser (1997 Intl JOS): Intermittant tingling or numbness of lower lip. Numb lip most common feature of metastatic CA • Boyczuk & Solomon (1991): Metastatic breast cancer as mandibular pain. • 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 • Ratner & Langer (1986): Alveolar cavitational osteopathosis. Roberts (1984): NICO • Drinnan (1987 DCNA): Manchausen's syndrome
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
Bence & Meyers '80 Simon '82 Seltzer & Naidorf '84 August '82	**Endo Emergencies: Open vs Closed** Keep it Closed: • Bence & Meyers (1980): Recommended that leave tooth open only as last res ort (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 purlence 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 persent 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
Walton & Fouad '92 Torabinejad '88	**Predictors of Post-Endo Pain** • Walton & Fouad (1992): Best predictors are pre-op pain or swelling • Torabinejad (1994) n=588 Best predictors: Level of pre-op pain and anxiety • 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 • 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 • Genet (1987 IEJ): predictors of post pain: pre-op pain, necrotic, PARL, females • Imura (1995 IEJ): 1.5% incidence of flare-ups; most often with PARLs, pre-op pain, multiple appts • Maddox & Walton (1979): no intracanal med better than dry cotton pellet, no diff in pulpotomy, pulpectomy on post-endo pain. ALSO: Hasselgrein & Reit; Kleir & Mullaney (1980); Walton (1984 DCNA), Harrison (1979)
Flare-ups **Trope '90** **Trope '95** Morse '86 Walton '92 Torabinejad '88 Seltzer & Naidorf '85	**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 & lg PARLs • **Trope** (1990): Flare-up incidence = 2.6%. No difference between formocresol = Ledermix = Ca(OH)2 as intracanal medicaments • **Trope** (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 Trope '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] • Georgeopoulou (1993 IEJ): over-instrumentation may lead to flare-ups • Seltzer & Naidrof (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
Goerig	**Causes of Flare-Ups** Seltzer & Naidrof (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:

	• 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 unjustifed. • 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%! • Abbott, Koren & Morse OOO(1988)p722: Tx necrosis with PARL with Pen VK or Erythromycin. Found lower swelling & pain. • 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** <u>1. Adjust Occlusion to Reduce Post-Endo Pain:</u> • **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 • <u>Re-enter RC system and debride</u> • Open vs closed (Bence & Myers; Seltzer & Naidorf; August) <u>Establish drainage</u> • Apical penetration for intracanal drainage • Trephination (Elliot #3 spreader; Peters) <u>Evaluate for analgesics</u> • 3-D strategy - Hargreaves <u>Evaluate for antibiotics</u> • 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) <u>Evaluate for Steroids</u> • Marshall & Walton;

	Pulp Biology
Mark Bishop 1990	Pulp contains lymphatic vessels (cats). Also reported by Bernick 1977.
Stanley 1981	Irregular secondary dentin can be viewed as mineralized scar. Forms in humans at 1.5 um/day
Stenvik 1972	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).
	Cell Adhesion Molecules (CAMs) • Tasman (1999 JOE): CAMs expressed on vaculature 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
Heyeraas 1992	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
Fristad 1995	Denervation reduces influx of inflammatory cells into pulp
Kindlova 1965	Blood supply to mucosa is by vertical blood vessels. Therefore, horizontal incisions (for sx or IND) will bleed more than vertical incisions.
Garberoglio & Brannstrom 1976	Dentinal tubues density = 40-70,000/um2. At DEJ, 1% of surface = tubules. At PDJ, 22% of surface = tubules.
Papa & Messer 1994	Although previous studies believed that RCT dried out teeth, P&M reported that vital dentin contained 12.4% moisture whereas root filled teeth contained 12.1% moisture.
Sedgley & Messer 1992	Root-filled teeth are not more brittle than vital teeth.
Berneck & Nedelman 1975	Older pulps have reduced # blood vessels and nerve fibers
Johnson 1985	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 & Torabinejad 1991 (who found Ad during root development).

	DIAGNOSIS AND DIAGNOSTIC TESTS
Seltzer & Bender '63 Tyldesley '70	**There is a poor correlation between clinical symptoms and pulpal histopathology** • Seltzer & Bender (1963) • Dowden 1969; • Garfunkel 1973 • Tyldesley & Mumford (1970 Dent Prac): Classic: Examined 142 teeth with pain. No correlation between histology and clinical symptoms. • Baume (1970 OOO): impossible to determine histology from dx

White & Cooley '77 Fuss & Trowbridge '86 Petersson '99	**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 = -50C), CO2 and EPT are equally reliable as pulp tests in adults • Petersson (1999 EDT): 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 hydrodynamc theory) "Its cold out"
Fuss & Trowbridge '86 Anderson & Pantera Pantera '92 Myers '98	**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 +
Gutmann 1995 Compendium	When RC system is not centered in root, you should suspect a second canal. The classic study on this point is: Slowey (1974)
	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: Nielsoen (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	**Laser Doppler** • 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
Gutmann 1995 and Cox 1991	Rec take another WL film if file tip is >1mm from apex

Kelly 1988 JOE	Presented cases where sinus tract was tracked to adjacent teeth. Always track sinus tracts
Selden 1999	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
Ikeda & Suda 1998 JOE	Human microneurogaphy 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 "Pathological" 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 Interpret 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
Brynoff 1970	Dx accuracy is increased when take 2nd radiograph: 73% accurate with 2 radiographs and 87% accurate with 3 radiographs
Cameron 1993	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
Diaz-Arnold, Wilcox 1996	In vitro study to measure pulp vitality by oxygenated hemoglobin
Dodd, Dodds, Holcomb 1984	Case report of chronic sinusitis due to over-extended silver point. Sinusitis resolved when removed silver point.
Hill 1986	Describes transillumination as a useful adjunct

	Pulpotomy
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.
Mass 1993	Reported 91% success rate in doing Cvek partial pulpotomy in young, posterior, symptom-free teeth with carious exposures.
Schroder 1971	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.

	Coronal Microleakage
Saunders & Saunders 1994	Coronal microleakage is important cause of RCT failure.
Ray and **Trope** 1995	Retrospective study of 1,010 NSRCT, evaluating periradicular radiographic status. Failure was attributed more to technical quality of coronal

	restoration than to quality of RCT. ALSO: Allen (1989 JOE): teeth restored after NSRCT had better success rate
Cox 1987	Examined effects of materials placed against the pulp. Concluded that seal was more important than the material itself.
Swanson & Madison '87 Torabinejad '90 Khayat & Torabinejad **Trope** '95 Ray and **Trope** '95	**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. • Torabinejad (1990): Coronal microleakage with bacteria. Motile bacteria recontaminated obturated root canal systems in 49 days. Non-motile bacteria recontaminated obturated root canal systems in an average of 24 days. • 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 endodoxin. Endotoxin leaked faster than bugs (Means: 23 days vs 62 days) • **Trope** (1995): Endotoxin penetration of coronallly 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)

	ENDO-ORTHO
Heithersay 1973	1st description of vertical root extrusion. Note that Lemon 1982 recommends 1 month stabilization for every 1 mm moved.
Ingber 1975/76	Described forced eruption, tx perio defects, importance of biological width
Mattison & Delivanis 1984	Endo tx teeth can be moved orthodontically without increased risk of resorption.
	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:

Mazur & Massler '64 Bender & Seltzer '72 Torabinejad & Kigen '85	<u>Studies that found a correlation:</u> • Seltzer & Bender (1967 OOO): perio can be a factor contributing to failure of NSRCT • Wong (1989 EDT): Root planing & citric acid tx can cause pulpal inflammation & open dentinal tubules (small n, though) • <u>Studies that found no correlation between perio path inducing endo pathology</u> • Mazur & Massler (1964): Found no correlation between severity of periodontal disease and histological status of the pulp • Bender & Seltzer (1972): Found no association between pulp disease and 1) probing depths; 2) extent of bone loss; 3) extent ot perio disease • Torabinejad & Kigen (1985): found no correlation (ie, found teeth with perio path but normal pulpal responsiveness) • 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.
Simon 1972 J Perio	CLASSIC: Classification of endo-perio lesions: Great examples on pp 307-312 Problem Solving in Endo Primary pulpal lesion Primary pupal lesion / secondary perio lesion Primary perio lesion Primary perio lesion / secondary pulpal lesion Combined pupal & perio lesion Concomitant pulpal-perio
Trope 1988 JOE	Possible test for differential perio-endo dx: 30-60% Perio abscesses have spirochetes, but only 0-10% endo abscesses have spirochetes
Hiatt 1977 J Perio	Reviewed causes and differential dx of endo-perio. Prognosis of endo-perio lesion is ultimately the periodontal prognosis!
	BLEACHING
Dahlstrom, Hiethersay 1997	OH- radicals formed during thermocatalytic bleaching could be MOA for PDL breakdown and resorption.
Spasser 1961	Described walking bleach technique.
Madison & Walton 1990	Cervical resorption due mostly to heat in the thermocatalytic bleaching technique.
Smith & **Cunningham** 1992	Recommended seal canal orifices with 2mm Cavit prior to walking bleach.
Freccia & Peters 1982	Walking bleach produces same results regardless of use of heat.
Lewinstein 1994	Recommends use of sodium perborate instead of 30% H_2O_2 since H_2O_2 reduces microhardness of both dentin and enamel.
Walton & Odell 1982	Tetracycline stains are more in dentin than in enamel (therefore, vital bleaching is temporary).
Titley & Torneck 1993	H_2O_2 in walking bleach technique can inhibit composite polymerization and dentin bonding

	Surgery
Dorn & Gartner '90	**Success of Surgical Endo:** • Apical Sx: 59% • Re-Tx + Apical Sx: 80%

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-randomzed, 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. 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 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 matieral: biocompatability, apical sealability and handling characteristics • Briggs & Scott (1997): Re-tx is preferable over endo sx ("evidence based" analysis). Also, Moiseiwitsch & Trope 1998. • 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** Persistant 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 **Contrindications 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:

Gilheany '94 Chong '97 Gagliani '98	• Gilheany 1994: Studied relationship between bevel angle of root resection and required depth of retroprep to minimize leakage: 0 degree bevel: minimal retroprep depth of 1 mm 30 degree bevel: minimal retroprep depth of 2.1 mm 45 degree bevel: minimal retroprep depth of 2.5 mm 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) • Chong (1997): Bevelling root ends should be minimal since they open up dental tubules. • Tidmarsch (1989): Bevelling root ends should be minimal since they open up dental tubules. • Gagliani & Molinari (1998 JOE): 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°
Khoury 1987	Reported bony lid method for mandibular sx. Technique increases vision and access; AND, reduces bone loss and incidence of hematoma
Hirsch 1979	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.
	.
	US vs Rotary Handpiece • Richman (1957): First to propose US for root end resection • Wuchenich & Torabinejad (1994): Cadaver study: US produces deeper preps with smaller bevels, better centered in canal • Gutmann (1994): In endo sx, Ultrasonics remove bacteria better than burs. • Engle & **Steiman** (1995 JOE): US tips gave superior results compared to micro-rotary handpiece for retropreps • Gorman & **Steiman** 1995): Ultrasonic tip vibrating freely in completed prep may flush out remaining debris. Also reported by Gutmann (1994)
Morgan & Marshall 1998	Recommended use of the Multi-purpose bur (Caulk) for root resection to give smooth surface with least shattering and cracking
Saunders & Gutmann Abedi & Torabinejad Frank & Bakland Lin '99	**Ultrasonic Rootend Preparations and Crack Formation:** • Saunders & Gutmann (1994): US may produce cracks (at ENAC power setting = 10) =FIRST REPORT OF US & CRACKS ! Cracks may be due to impact of UC tip against dentin and heat formation • Layton (1996): In endo sx, ultrasonics may produce cracks at root end. (esp at high freq > low frequencies) • Min & Brown (1997) did report ultrasonics cause cracks in root end preps • Beling (1997): did not detect cracks. • Waplington (1997): Ultrasonic rootend preps does NOT cause cracks • Abedi & Torabinejad (1995): Crack formation by ultrasonics was a fxn of power, time, initial cracks, and thickness of remaining dentin • Frank & Bakland (1996): Ultrasonics on medium power with water spray reduces incidence of root infractions (= cracks) • Lin (1999): Used strain guages to measure root tip during US preps. US > rotary for rootend strains. But , didn't see any cracks • Brent & 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 pruduce heavy abrasion, lots debris & uneven preps and required CT-5 tip to make a pilot hole. Also, difficult using diamond tip to prepare isthmus groove • Morgan & Marshall (1999): Took in vivo polysiloxane impressions on 25 roots after root-end preps after smoothing with #ETUF-9 mulitfluted finishing bur and CT-5 US tip or diamond-coated 12S90D US tip. No cracks evident after root resection. 1 crack after diamond tip.
Harrison 1991	Overview of biological responses in wound healing

Harrison & Jurosky 1991	**Healing of incisional flaps in rhesus monkeys**. Sulcular incision leaves perio tissue attached to cementum which speeds up repair and prevents epithelial downgrowth. Rec using undermining elevation of vertical flap.
Harrison & Jurosky 1991	**Healing of dissectional wounds in rhesus monkeys.** 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 periosteum, no loss of alveolar crest Day 28: have completely normal tissue
Harrison & Jurosky 1991	**Healing of osseous excisional wounds in rhesus monkeys.** Days 1-3: coagulum of disorganized fibrin acts as barrier to inflam/repair cells Day 4: inflam cells in coagulum Day 14: proliferating granulation tissue replaces coagulum, woven bone, osteocytes, dense fibrous CT spearates flap from bone Day 28: trabeculae coalesce, lined with osteoblasts, in contact with devitalized cortical bone of wound edges
Andreassen & Rud 1972	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
	MTA: Major properties: excellent seal and biocompatable • 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): Biocompatable: Cementum forms over MTA retrofill with Sharpys fibers (Negative in mutagenicity tests • Composition (from 1999 web page Tulsa MSDS): tricalcium silicate, dicalcium silicate, tricalcium aluminate, 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, mnenomic "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 adapation 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
Oynick Dorn & Gartner '90 **Rubenstein & Kim** **Testori '99** **Steiman '97** **Hartwell '98**	**Super EBA** • Oynick (19xx): also shown Sharpy's fibers growing in super EBA • Dorn & Gartner (1990 JOE): Retrospective study in two endo offices (non-randomzed, 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. Isthmuses 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 fxnl 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 & Abdellatief 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?

Weller and Kim 1995	Assume an isthmus is present whenever a MB root of a max 1st molar is resected
Gutmann '93 & '96 Buckley '84 Milam '84 Roberts '87 Lindorf '79	**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 α1 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 ß1 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) & Stein (1995): Collacote is a non-woven pad of collagen. Acts similar to Avitene with similar healing properties • 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. • Lucas & Albert (1981): Tranexamic acid potent antifibrinolytic agent. MOA inhibition of plasminogen activation. Preinjection of tranexamic acid into sx site increased tensile strength of healing incision (Bjorlin & Nilsson 1988). Intraoral pre-op rinse effectively controls hemorrhage without altering pt's antocoagulation therapy (ALSO: Borea 1993; Souto 1996 JOMS). • 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 • Codben (1976): Topical thrombin indicated when wound bleeding from small capallaries & venules (J&J, 1990). Activates extrinsic and intrinsic clotting pathways (do not inject systemically!). Doesn't impede healing.
Evans '79 Gerstein '89 Witherspoon & Gutmann '96	
Lemon '93 Jeansonne '93 Lucas '81 Souto '96 Codben '76	

	Reimplantation and Transplantation
	Success in Intentional Reimplantation: 1. Grossman (1982): 70% success at 5 yrs 2. Keller (1990): 91% success 3. Bender & Rossman (1993): 81% 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 reimplantaion. 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-innoculated 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)) • Bystrom (1985): Importantly, E. faecalis is resistant to Ca(OH)2 tx. Confirmed by Reit & Dahlen (1988). • Estrella (1999 JOE): Ca(OH)2 demonstrated NO antimicrobial effect at 2, 3, & 7 days against E. faecalis, S. aureus in infected dentinal tublues (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-anerobes, 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. asacharolyticus, 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 persistant 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% curretage = 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)
Abou-Rass '98 Iwu '90 Wayman '92	**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. actinomyces (32%), Proprionibacterium (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 microoganisms). Found Veilonella (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!) • Molven (1991): Used SEM to find bugs on apices. Extraradicular infection. • 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 2° 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 persent 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 anerobic • Bacteroides melanogenicus (now: Prevotella melanogenicus) ocurred 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 mechansism 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)

Dwyer &Torabinejad '81 Horiba '91 Safavi '93 **Trope** '97 Alves '98 Darveau '95	**Endotoxin** • Dwyer &Torabinejad (1981): Endotoxin placed into canals caused severe pupal and periradicular inflammation. Concluded that endotoxin plays a significant role in periradicular disease. • Horiba (1991) found correlation between clinical symptoms, PARL and endotoxin content. • Schein & Schilder (1975) found positive correlation between endotoxin levels and necrotic, painful teeth with PARLs. • Safavi & Nichols (1993): Ca(OH)2 inactivates LPS in vitro • Barthel & **Trope** (1997 IEJ): Ca(OH)2 inactivates LPS in vitro • 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 • **Trope** (1995): Endotoxin penetration of coronallly unsealed NSRCT teeth <21 days. Emphasizes importance of coronal seal • Darveau (Infect Immun 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 disant 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" (see Bergman JOE '99 Nov).
Gharbia & Haapasalo 1994	At least some bacteria are site specific. P intermedia is associated with perio pockets whereas P nigrescens associated with root canals
Gomes, Drucker 1994; 1996	Pain is significantly associated with Prevotella m elaninogenica and Peptostreptococcus in infected root canals; especially specific combinations of Peptostreptococcus and Prevotella species (1996 study: 70 teeth from 60 consecutive pts)
Griffee & Patterson 1980	Found Bacteroides melanogennicus signficantly associated with foul odor, pain and sinus tracts. Also reported by Sundqvist 1976.
Iwu & MacFarlane 1990	Cultured PA lesions without sinus tracts. 88% had + cultures (100% contained strict or facultative anerobes). 96% sensitive to amoxicillin, tetracycline or clindamycin (but, only 45% were sensitive to metronidazole).
Namavar & Marian 1983	Good article to quote to support persistant infection even in presence of competent immune system: Get synergistic interaction between aerobic and anerobic bacteria for inhibiting PMN actions
Oguntebi 1994	Bacteria left in dentinal tubules may be a reservoir from which canal reinfection may occur. Conversely, Peters 1995 argues the opposite.
Reeves & Stanley 1966	No inflammation in pulp when bacteria at 1.1mm from pulp. Magnitude of inflammation increases when bugs are 0.5mm; abcesses found when bugs are at pulp.
Stabholz & Sela 1983	Do NOT need to have live bacteria to produce pulpal or periradicular inflammation. Cell fragments or supernatants can cause rxn.
Tronstad & Mjor 1972	Carious exposed pulps have <50% response to vital therapy. Therefore, carious exposures should be considered to be irreversible pulpitis.
Chirnside 1961	In contrast to vital pulps, bacteria can rapidly grow in dentinal tubules of necrotic teeth & reach pulp.
Matusow 1988	Believe endo cellulitis caused by aerobes & that prophlactic antibiotics unwise.
Giunta 1987	Discussed difference between swelling and erysipelas. Erysipelas = strep pyrogenes infection (redness / swelling)
Paonessa & Goldstein	Otolaryngol Clinics NA 1976 - excellent review of spaces with great diagrams. Also, Piecuch DCNA 1982
Tronstad & Olsen 1999 EDT	Reviews brain abscesses caused by odontogenic infections. Routes include 1) direct extension (fascial planes); 2) hematogenous (via facial, angular, opthalmic, 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 Diagnoisis" R. Langlais & M Kasle Exercises in Dental Radiology. Vol 1: "Intra-oral Radiographic Interpretation" Saunders 1978
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
Bender & Seltzer '61 Brynolf '67 Kaffe '88 Messer '98	**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 artifical 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 tangentally 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

	images fooled insurance company examiners. • JOE Sept 1999): Fabrication of Schick images
Tamse & Kafne 1980	Zygomatic arch interferes with the DB root of max 1st molar and 48% of MB roots of max 2nd 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 periodontits will show radiographic s igns 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 teet 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 **<u>Mandibular Premolars - Molars</u>** 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 Matastatic 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
Goerig	**Differential Dx of Periradicular Radiolucent lesions:** <u>1. Developmental PARLs:</u> Salivary gland depression Neurovascular canals Developmental Nasopalantine duct cyst: Gnanasekhar (1995) 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) <u>2. Infection-Related PARLs</u> LEO- granuloma: 50% cells are inflammatory cells (macrophages > lymphocytes > PMNs LEO- cyst: lymphocytes, plasma cells, macrophages , cholesterol clefts Actinomycoses TB <u>3. Metabolic PARLs</u> 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 (Shaeffer 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 Ricketts - multiple PARLs even on virgin teeth <u>4: Odontogenic PARLs</u> Botyroid odontogenic cyst = mulitlocular 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 Central odontogenic fibroma: Huey (1995): • 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? Il-1a & ß TNF a & ß 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-1ß (and ~60% of its bone resorbing activity is mediated by release of prostaglandins: Torabinejad & Kiger 1980; Dewhirst 1990). Thus, bacterial-induced release of IL-1ß and prostaglandins are destructive. • 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. • 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 innoculated microflora demonstrated important role of Ab in preventing infections spreading away from the RC sysem. 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 peridontitis 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 sensitization. (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 • Torabinejad (1980): Supports role of immunoglobulins in developing apical periodontitis. Induced PARL by placing antigen in RC system of previously sensitized cats. **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 sensitization.
	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:** • Intermittant 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.
Goerig	**Partial Loss of Lamina Dura:** • Hyperparathyroidism • Gauchers • Leukemia • Scleroderma • Pagets • Cushings • VD Resistant Rickets • Osteoporosis
	Granulomas vs Cysts • Bhaskar (1966) 48% granuloma 42% cysts • Lalonde & Leubke (1968): 45% granuloma 44% cysts • Nair (1996): n=256 50% granulomas 15% cysts (inc both pocket cysts and true apical cysts), 35% PA abscesses • Rubenstein & Kim (1998) 85% granuloma 15% cysts • Delzangles (1989): SEM study of root apices: granulomas produced more root resorption than cysts • Morse & Patnik (1973) PAGE can be used to differentiate cyst from granuloma by aspirate • **Trope** (1989): Used CT scan to differentiate cysts from granulomas • **Priebe** & Lazansky (1954): Can't tell if a lesion is a cyst or granuloma from an X-ray!
	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 cyst). (this approach seems to be sucessful even though epithelium is not curretted 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 & **Jeansonne** 1999 EDT	Cultured 13 necrotic teeth with AP: no ß-hemolytic strep were cultured. Staph epidermidis was most common bug isolated.
	Rubber Latex Allergies: • Gazelius & Olgart (1986): Pt with rubber allergy: NSRCT with GP caused pain/tenderness. Removed GP and symptoms stopped; replaced GP and symptoms recurred. • 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) • Boxer (1994): Reported allergic-like rxn (pain, swelling, urticaria) in pt with latex allergy who experienced overfill of GP into periradicular tissue. Reported immediate relief after removal of GP • 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
Cooper Porter Glick & Trope Shiboski Wahl	**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 workders exposed to HIV-infected blood thru percutanous 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 apthous 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 coagulaton 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 • Kelia (1990 OOO): Case report: uncontrolled bleeding during NSRCT in pt with un-dx von Willebrands disease • 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 • Galili & Findler (1994): Reviews oral and dental complications of diabetes mellitus • 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, numb-like sensation. CC #2 = numb lip
Herman JADA p327 1997	Good review of coumarin anticoagulant therapy
Jolly DCNA 38(3):361, 1994	Reviews importance of med hx review
Mealey DCNA 1994	Complications of head & neck radiation therapy
Muzyka and Glick JADA 128:1109, 1997	Reviews Hypertension pts
Brannon 1977	OKC needs to be removed entirely and biopsied. Hancock & Brown 1986 reported OKC as PARL remaining after NSRCT (possible zebra)
Clark & Allet 1980	Gorlin cyst. Mand molar/premolar Include in diff dx for PARL with opaque regions
Cohen 1984	Traumatic bone cyst can occur after extractions
Corio & Crawford 1976	Benign cementoblastoma = radiopaque lesion attached to roots causes resorption.
Donnelly & Koudelka 1986	Median palatal cysts with periradicular lesions. Need occlusal film. Also reported by Nip & Nguyen 1981
Yagiela 1999 JADA	**Vasoconstrictors are contraindicated in pts using :** • Tricyclic antidepressants (enhanced sympathetic effects) • Non-selective ß-blocker (eg, propranolol; may get hypertension) • Halothane (cardiac arrthymias) • 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 C02 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 (Schroder 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 anearobic).
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 Tziafas 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 <u>Dx vertical root fx is difficult because:</u> • 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). <u>Clinical findings of a vertical root fracture</u> <u>aka "Cracked tooth syndrome" (coined by Cameron (1964 JADA) and</u> <u>aka "split tooth" [coined by Silvestri 1976]:</u> • 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 transillumniation or by pulpal floor staining with methylene blue • Deep narrow periodontial 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 (hypoosmotic)
	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:** Concusion: 3% become necrotic Subluxation: 6% Extrusion: 26% Lateral luxation: 58% Intrusion: 85% In general, teeth with complete root development have poorer prognosis for pulp vitality (necrosis usually seen in 3 weeks) than teeth with incomplete root deveploment (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:**

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 avleolus and dentin (due to removal of PDL) • Inflammatory resorption = bowl shaped defects that penetrate dentin (dentin has Howships 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 • Hiltz & **Trope** (1991): Viaspan > HBSS > Milk • 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. Recommeded giving antibiotics when replanting avulsed teeth • Cvek & Cleaton-Jones (1990): Monkey study: Doxycycline tx of extracted teeth before replantation reduced inflammatory resorption & anykosis. • **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 cementrum, 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 tetracylcine 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 (NSCRT; extract, bench dry 1hr, replant; 3-4m follow-up): Controls & systemic amoxicllin ~11% healing. In contrast systemic tetracylcine (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) • Lindskog & Blomlof (1998 EDT): Monkey study: Infected pulps, extract, scrape cementum. Intracanal application of chlorhexidine (10% soln for 4w) significantly reduced inflammatory resorption vs controls. • Nishioka & Suda (1998 EDT): Performed 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 truama 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. Surveryed 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 repairative dentin in rats by 3 weeks and may have utility for direct pulp capping material. (No repairative dentin potential foudn for EGF, FGF, IGF, PDGF)
Rutherford 1993	Human osteogenic protein (= hOP-1 = BMP7) also reported to evoke repairative 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.
	Diagnositic 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 1° 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 • Longwill & Marshall (1982) report that pediatric use of formocresol does NOT sensitize the child. • 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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