

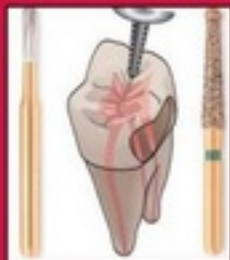
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PrettyDarnedQuick

Endodontics

Second Edition

John I. Ingle



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PDO ENDODONTICS

John I. Ingle, DDS, MSD

Lecturer, Department of Endodontics

School of Dentistry

Loma Linda University, Loma Linda, CA

*Formerly Dean and Professor of Endodontics
and Periodontics*

University of Southern California, Los Angeles, CA

*Formerly Professor of Endodontics and Periodontics
University of Washington, Seattle, WA*

Diplomate, American Board of Endodontics

Diplomate, American Board of Periodontology

People's Medical Publishing House—USA

2 Enterprise Drive, Suite 509

Shelton, CT 06484

Tel: 203-402-0646

Fax: 203-402-0854

E-mail: info@pmph-usa.com

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Japan

Tel: 03-5479-7251

Fax: 03-5479-7307

Email: kakimoto@ups.co.jp

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Dedication

This second edition of *PDQ Endodontics* is dedicated to two of the most remarkable persons I know, **Dr. Leif K. Bakland** and **Dr. J. Craig Baumgartner**, colleagues extraordinaire! Leif and Craig were the “backbone” of the remarkable resurgence of the sixth edition of *Ingle’s Endodontics*. Both enormously talented, we are all deeply indebted to them for their contributions to our knowledge and skills.

John I. Ingle, 2009



Left to right: J. Craig Baumgartner, John I. Ingle, Leif K. Bakland

Acknowledgments

My cup runneth over.

—Psalm XXIII

I have been so blessed! Blessed first of all with loving parents who provided me a fine education, and then with a loving and extraordinary family. I have also been blessed with wonderful teachers, brilliant students, and marvelous associates and fellow faculty members who have led me on a pathway of which I never dreamed. And through it all, I have been backed up by talented and dedicated nurses, assistants, secretaries, technicians, and publishers. How fortunate I have been!

In my course through life, I have been associated with a number of unusual universities, each one depositing special skills and knowledge. I am deeply indebted to them all: Washington State University, Northwestern University, University of Michigan, University of Washington, University of Southern California, Howard and Georgetown Universities, UCLA and, finally, Loma Linda University. My stint at the Institute of Medicine, National Academy of Sciences, was a humbling experience. I suddenly learned what I did not know.

And now to this text. The backup at Loma Linda University has been outstanding. Leif Bakland and Mahmoud Torabinejad were eager to help. How fortunate I am, at my age, to be a member of such a distinguished faculty.

Thanks to my computer guru and maven, Lynda Arnett, I was able to suffer through many failures, “viruses,” “worms,” and my own inadequacies. And to former student and former colleague, Tom Pallasch, a well deserved thank you for his assistance in formulating the section on focal infection. And at the AAE, Stacy Bogard, Jill Forister, and Jill Cochran were all a great help. And I must again thank my wife, Joyce, for her exceptional computer typing skills, far outclassing my “hunt and peck” abilities.

I'd be remiss if I failed to thank those countless associates who, as contributing authors, were so generous with their time and knowledge to make the text, *Ingle's Endodontics*, the success it has

been. I have borrowed unsparingly from their writings and research in producing this condensed version.

Finally, I must acknowledge those at the People's Medical Publishing House-USA, Martin Wonsiewicz, Tim Stanton, Jason Malley, and Lyn Spaulding. At Spearhead Global, Inc, Harjeet Singh, compositor, and his associates.

John I. Ingle, DDS, MSD
San Diego, CA, 2009

Introduction to Endodontics

Before one delves into *PDQ Endodontics*, a condensed version of the “mother text,” *Ingle’s Endodontics*, we should define **endodontics**, a.k.a. root canal therapy or endodontology. The following definition was “lifted” and modified from the American Association of Endodontists’ (AAE) version:

Endodontics is that branch of dentistry centered on the dental pulp and periapical tissues. The scope of endodontics deals with the diagnosis and treatment of pulp and periapical pathology and the prevention of pulp disease. It also deals with oral pains of pulpal and/or periapical origin, as well as extraoral referred pain; vital pulp capping or pulpotomy; pulp extirpation and cleaning and shaping of the root canal; its disinfection, and its obturation; surgical removal and biopsy of pathologic periapical tissue; root end resection and reverse (retro) obturation of the canals, as well as hemisection and full root resection of pathologic roots; endodontic and osseointegrated implants; management of traumatic injuries to the teeth and replantation of avulsed teeth; internal and external bleaching of discolored teeth; retreatment of previously treated teeth; and post and core placement involving the root canal space.

An endodontist is a dentist with two or more years of advanced training in endodontics who has received a certificate or advanced degree from an accredited program. Those who meet these qualifications have the privilege of appearing before the American Board of Endodontics; if they pass, they become diplomates of the ABE. These specialists stand ready to help any dentist with a pulpal, periapical, or pain problem. Count on them!

But make no mistake, endodontic specialists are only doing 20% of the root canal procedures in America, whereas general practitioners are doing 75% of the endodontics. The remaining 5% is done by other specialists (Table 1). On the other hand, endodontists

Table 1.

DISTRIBUTION OF ENDODONTIC PROCEDURES BY SPECIALTY

Type of Dentist	1990		1999	
General practitioners	15,758,100	76.1%	16,493,200	75.2%
Endodontists	3,860,700	18.6%	4,459,900	20.3%
Pediatric dentists	942,200	4.5%	721,300	3.3%
Oral and maxillofacial surgeons	108,800	0.5%	188,900	0.9%
Orthodontic and dentofacial Orthopedists	0	0.0%	0	0.0%
Periodontists	31,800	0.2%	50,700	0.2%
Prosthodontists	25,400	0.1%	18,800	0.1%
Total	20,754,000	100.0%	21,932,800	100.0%

Source: American Dental Association Survey Center, 1990 and 1999 Surveys of Dental Services Rendered

are treating more molars (61.9%) than the general practitioners, who in turn refer only 9.8% of the anterior teeth to the specialists (Table 2). The number of endo treatments done in a year varies from 17 million to 30 million, according to the ADA. The difference

Table 2.

SURVEY OF DENTAL SERVICES RENDERED.

Endodontic Procedures	General Practitioners		Endodontists	
Molar root canals	4,887,500	29.6%	2,761,900	61.9%
Bicuspid root canals	3,501,600	21.2%	908,500	20.4%
Anterior root canals	3,317,600	20.1%	438,000	9.8%
Pulpotomy	1,802,800	10.9%	158,700	3.6%
Pulp cap	1,609,200	9.8%	16,400	0.4%
Bleaching	1,237,600	7.6%	23,900	0.5%
Apicoectomy	136,900	0.8%	152,500	3.4%
Root amputation	0	0.0%	0	0.0%
Total	16,493,200	100%	4,459,000	100%

Source: American Dental Association, Survey Center, 1990 and 1999 Surveys of Dental Services Rendered.

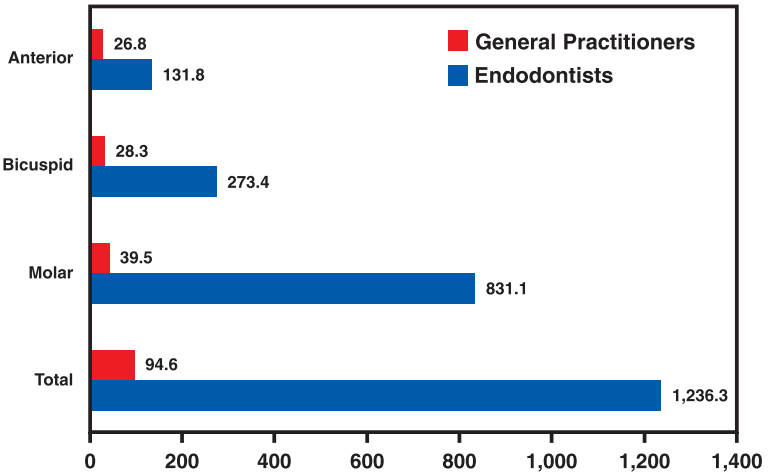


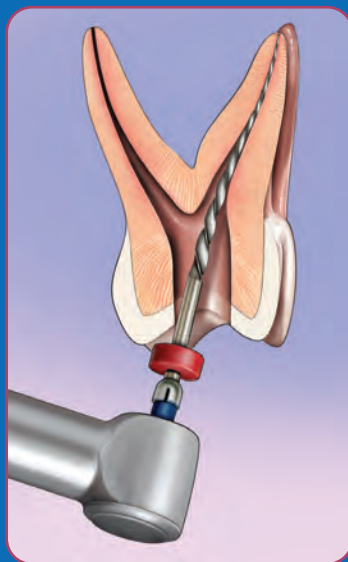
Figure A. Root canal treatments per dentist. ADA Survey Center. Survey of Dental Services Rendered.

is due to the fact that the 30 million **procedures** probably include radiographs, examinations, and so on, versus 17 million **filled** root canals (Figure A). In either event, that's a lot of root canals and is probably an even higher number today, ten years later.

Note to the reader: If you are seeking additional information over and beyond the contents of this book, please refer to the full text, *Ingle's Endodontics*, sixth edition, by J. I. Ingle, L. K. Bakland, and J. C. Baumgartner, published in 2008 by People's Medical Publishing House-USA.

*Ingle's***ENDODONTICS 6**

Ingle
Bakland
Baumgartner



ENDODONTIC CONTROVERSY

However, there is more to be said beyond success and failure. **Two present controversies** must be faced. The first deals with life and death: claims that endodontics contributes to myocardial infection, that root canal therapy is not to be trusted, that extraction is “the final solution” to periapical pathology.

This age-old canard leads to the second controversy—**endodontics versus extraction and implants**. Should pulpless teeth, with perfectly good and sound roots, be sacrificed on the altar of “focal infection?” Unfortunately, a covey of dentists seeking to

broaden their practices is listening to the **siren's call of focal infection** as an excuse to extract and implant. Those of this mind “leads to what *Jane's Defense Weekly* terms ‘incestuous amplification’: a condition of warfare where one only listens to those who are already in lock-step agreement.”¹

Both of these controversial subjects have built-in adherents—all the more reason why this should lead to discussion and debate. Pallasch notes that, “it is better to follow the observation of Harry Lime, in Graham Greene's *The Third Man*, regarding the **value of controversy**: 30 years of noisy, violent churning conflict under the Borgias in Italy produced Michelangelo, Leonardo da Vinci and the Renaissance, while 500 years of peace in Switzerland produced the cuckoo clock.”¹ So let's review each of these controversies before committing ourselves.

For those who have not lived through the devastating tooth loss from the “**theory of focal infection**” (TFC), a short evaluation should be in order. Years ago, Sicher referred to this debacle as “The mountain of needlessly extracted teeth that would put the skull pyramids of Ghengis Khan to shame.” In *Ingle's Endodontics* (sixth edition), Guttman reviews this sordid history.² Pallasch and Wahl point out that oral foci, postulated as the source of “**elective localization**” of TFC bacteria (principally viridans-group streptococci [VGS]) includes “pulpless teeth and endodontically treated teeth along with periodontitis.”¹ Previously, focal infection was thought to lead to arthritis, nephritis, asthma, indigestion, goiter, Hodgkin's disease, and stupidity, among many other conditions. And even today, cancer and neuropsychiatric disorders are blamed on focal infection.

Pallasch points out that “(T)he ‘first’ report of focal infection has been ascribed to Hippocrates, who attributed the cure of a case of arthritis to tooth extraction. In the early 1800s, Dr. Benjamin Rush ... a signer of the Declaration of Independence, also related arthritis cure to tooth extraction.”¹ Later, the **autointoxication theory** claimed that “bacterial stasis of the colon” caused systemic disease. Colonic purging became the treatment for gastric cancer, peptic ulcer, neuritis, headache, endocarditis, stupidity, and arthritis, among other disorders. One wonders how many bowels were perforated from these violent enemas.

In 1890, physician/dentist W.D. Miller published *Micro-Organisms of the Human Mouth: The Local and General Diseases Caused by Them*. Miller **did not** mandate removal of teeth as a focus

of infection, and suggested “treating and filling root canals.”¹ The next year, the term **focal infection** was first used in *Dental Cosmos*.

And it was in 1900 that William Hunter, the famous English physician, reported on *Sepsis as a Cause of Disease*, listing poor oral health and restorative dentistry as a toxic source of a list of diseases he thought was caused by focal infection. It was Hunter’s infamous lecture at McGill University in 1911 that “ignited the fires of focal infection.” It was his claim that dental restorations, “built on and about diseased tooth roots, formed a veritable mausoleum over a mass of sepsis,” and were leading to the worst cases of anemia, gastritis, obscure fever, nervous disturbances, lesions of the cord, chronic rheumatic infections, and kidney diseases, among countless other conditions. “A careful reading of Hunter’s lecture, however, reveals that he listed only a single case of dental sepsis: a patient told by his dentist never to remove his partial denture from his mouth under any circumstances.”¹

The era of focal infection truly began in 1912 when physician Frank Billings introduced the concept of **focal infection** via case reports ascribing distant organ infections, and claiming “cures” of these afflictions by tonsillectomies and dental extractions. This was the same year dental x-rays first revealed unsuspected periapical pathology. E. C. Rosenow, a pupil of Billings at Mayo’s, developed the theory of “**elective localization**,” that bacteria possessed affinities for certain organs, and that *Streptococcus viridans*, for example, could transmute into pneumococci or beta hemolytic strep. Many famous physicians, including Charles Mayo of the Mayo Clinic and Russell Cecil, the famous author of *Cecil’s Essentials of Medicine*, joined Hunter, Billings, and Rosenow, advocating focal infection and its remedy by surgery. Subsequently, “[m]illions of tonsils, adenoids and teeth were removed in an ‘orgy of extractions’ as described by Grossman.”¹

However, by 1935 Cecil had published “an analysis of 200 cases of rheumatoid arthritis that documented **no benefit from tonsillectomy or dental extractions**.” He became one of its harshest critics and commented that a focus of infection was “anything readily accessible to surgery.” (As an aside, years later Russell Cecil addressed the Annual Meeting of the American Association of Endodontists, and his entire thesis was debunking the theory of focal infection.)

During this time, pulpless teeth—treated or not—came under particular scrutiny, and many physicians and dentists (the “100-

percenters”) recommended **extraction of all pulpless and endodontically treated teeth**. Endodontic education was also eliminated in many United States dental schools.

However, C. Edmund Kells, the founder of dental radiology, was one of the few dissenting voices describing the indiscriminate extraction of teeth as “the crime of the age.” In the United States, surgery for focal infection was twice as common among the rich as among the poor. It is also of interest to note that bodily areas, superficially accessible to surgery, were listed as foci of infection, whereas deeper structures were conspicuously absent. Cecil noted in 1935 that “focal infection was elegant in its simplicity and offered quick and easy, albeit expensive solutions, to a multitude of diseases for **which medicine had no answer**.” He further concluded that “focal infection is a splendid example of a plausible medical theory that is in danger of being converted by its enthusiastic supporters into the status of accepted fact, and that the time has arrived for a complete reevaluation of the focal infection theory.” As Thomas Henry Huxley noted: “The great tragedy of science is the slaying of a beautiful hypothesis by ugly facts.” This, said more than 100 years ago needs revival today!

The three most documented, publicized, and **litigated examples** of focal infection are **bacterial endocarditis**, brain abscess, and orthopedic prosthetic joint infections.¹ In the United States, the annual incidence of infective endocarditis is about 11,200 cases a year with approximately 25% caused by strep viridans VGS. On the other hand, the majority of cases in the twenty-first century are **caused by staphylococci**. **Nosocomial** (hospital-acquired) **staphylococcal endocarditis** has reached 25% of the total number of cases of endocarditis.

In marked contrast, dental professionals have been routinely blamed for endocarditis due to VGS, even though these microorganisms are ubiquitous throughout the body. New case-control studies indicate that there may be **no association between dental treatment procedures and bacterial endocarditis**.¹ Even daily oral hygiene procedures and mastication can produce an incidence of bacteremia approaching that of dental treatment procedures.¹

Pallasch has developed a listing of the **absolute risk rate** for various focal infections from a **single dental treatment procedure**.¹ (Table 3). The incidence of metastatic infections of heart, joint, and brain appears miniscule, even as low as 1 case of VGS infection for every 14,258,714 dental procedures. Surprisingly, it has been demonstrated that “the risk of a bacteremia arising from normal daily

Table 3.

THE ABSOLUTE RISK RATE (ARR) FOR VARIOUS METASTATIC FOCAL INFECTIONS FROM A SINGLE DENTAL TREATMENT PROCEDURE

Brain abscess: 1 million to 1 per 10 million dental procedures
Prosthetic joint infection: 1 per 2.5 million dental procedures
Bacterial endocarditis:
1. If all general population VGS cases caused by dental treatment: 1 per 142,578 procedures
2. If only 1% of VGS cases caused by dental treatment: 1 per 14,258,578 dental procedures
3. Previous endocarditis: 1 per 95,058 dental procedures
4. Cardiac valve prosthesis: 1 per 114,069 dental procedures
5. Rheumatic heart disease: 1 per 142,258 dental procedures
6. Congenital heart disease: 1 per 475,290 dental procedures
7. Mitral valve prolapse with regurgitation: 1 per 1,096,824 dental procedures

Source: Pallasch TJ. *Endo Topics* 2002;4:32–54. Reproduced with permission.

living is 1,000–8,000 times more likely than from a dental treatment procedure. It is time for the **courts** to recognize the scientific facts regarding bacteremia and focal infections, and cease and desist from the **prosecution and persecution of dental professionals** for metastatic infections, particularly bacterial endocarditis.”¹

In addition, there is yet to be a scientifically determined case of **prosthetic joint infection** due to dental treatment procedures. Meanwhile, millions upon millions of antibiotics have been given to prevent this “infection” without cost/risk benefit determinations.¹ Moreover, no study exists that delineates the incidence/magnitude of spontaneous bacteremias from infected root canals with **chronic periradicular abscesses**.¹

On the other hand, Bender determined a 0% to 15% incidence of bacteremia from infected root canals—0% if the instrument remained within the canal and 15% if it **extended beyond the apex**. In a study that intentionally instrumented beyond the apex, Baumgartner detected a 34% to 54% incidence of bacteremia.³ If one suspects that instrumentation beyond the apex has happened, **prophylaxis** for endocarditic prevention would be appropriate if the best clinical judgment of the dentist is that a pure “yes or no” determination of length cannot be made.

In summary, nonsurgical endodontics may be the **least likely** of dental treatment procedures to produce significant bacteremias in either incidence or magnitude. Pallasch recommended “that studies must be performed to determine if endodontic treatment is inferior to alternate treatments such as implants, prosthetic replacement or no treatment other than extractions.... There is **no evidence** to support the theory that modern endodontic therapy is not safe or effective.”¹ These studies have been and are being done, which is a perfect segue to our next controversy. Pallasch’s treatise and references¹ are recommended for more detailed study.

ENDODONTICS VS. IMPLANTS

It goes without saying that root canal therapy has been very successful. But what of implants? A number of studies comparing success and failure rates between the two disciplines have concluded that both ventures have the same success rate. One of the most dramatic by Salehrabi and Rotstein involved **1,463,936 root canal treated teeth** from the files of the Delta Dental insurance company.⁴ They found that endodontic teeth demonstrated a **97% success rate over 8 years**. Moreover, analysis of a small subset of the **extracted teeth** revealed that 85% of this cohort had no full coronal coverage (Figure B). Another report from an insured cohort of 44,000 endodontic patients confirmed these results with a 94% success rate retained for 3 and a half years.⁵

Implant success rates are quite comparable. Reporting from Scandinavia, Creugers et al. demonstrated that 459 single-tooth implants achieved a 4-year survival rate of 97%.⁶ Lindh et al. reported an implant success rate of 97% after 6 to 7 years.⁷ Prior to these

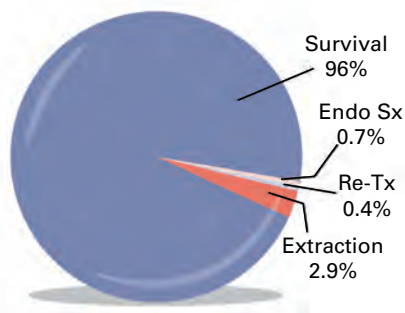


Figure B. Pie chart illustrating the outcomes of 1,463,936 root canal-treated teeth with an 8-year follow-up. Salehrabi R, Rotstein I. *J Endod* 2004;30(12):846–50. Reproduced with permission.

studies, endodontics judged success or failure based on clinical and radiographic improvement. Now the comparison must be made on **survival**, the criteria by which implant failure is judged—removal of failed implants and extraction of failed endodontics.

A good example of a comparative study was reported by Hannahan and Eleazer.⁸ There were **129 implant cases** in the study and they enjoyed a survival rate of **98.4%** over an average of 36 months. There were **143 endodontic cases** in the study and they enjoyed a survival rate of **99.3%** over an average of 22 months. (Figure C). The minor difference is not significant. If one adds the 26 “uncertain” cases to the cohort, the implant success drops to 87.6% and the endodontic success drops to 90.2%, which is still not significant.

However, what was significant was that **12.4%** of the implants required **further intervention**, whereas only **1.3%** of the endodontic cases required **intervention** (Figure D). All the cases were from endodontic and periodontic specialty practices.

In an outstanding review of this subject, Torabinejad pointed out that “[p]reviously, all efforts were made to save teeth with periodontal disease. Currently, the high success rates of implants have affected this concept, causing a **paradigm shift in periodontics**. Recent surveys by the American Academy of Periodontology show that while **63%** of the periodontists are placing their primary emphasis on periodontics, **27%** are **placing their primary emphasis on implants**.”⁹

If success rates between the two procedures are identical, what then are the differences in outcome? First, one must set a baseline

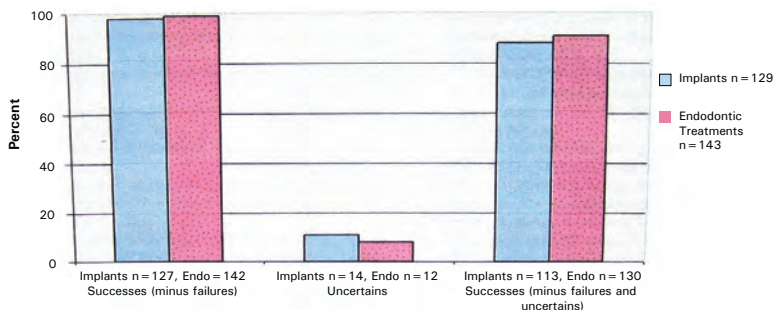


Figure C. Outcomes of implant surgery and endodontic treatment. No statistically significant differences between implants and endodontic treatments were found. Hannahan JP, Eleazer PD. *J Endod* 2008;34(11):1302–5. Reproduced with permission.

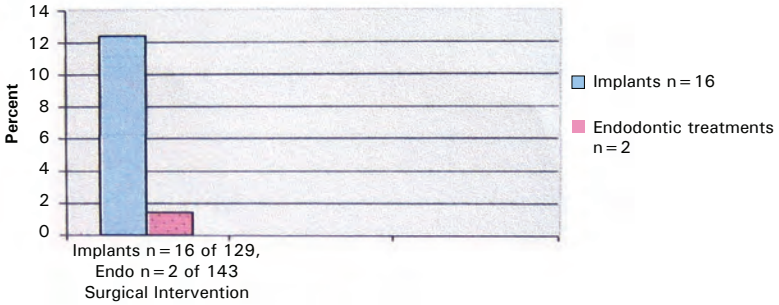


Figure D. Percentage of treatments requiring intervention after initial treatment. A statistically significant difference was found between implant surgery and endodontic treatment. Hannahan JP, Eleazer PD. *J Endod* 2008;334(11):1302–5. Reproduced with permission.

for what one hopes to achieve. In an AAE position paper quoting the American Dental Association: “The recommended treatment should be safe, predictable, cost-effective and respectful of patient preferences. It should also be aimed at preserving normal anatomy and function and based upon the best available scientific evidence.”

In view of similar success rates, the decision to treat a tooth endodontically or place a single-tooth implant should therefore be based on other criteria including restorability of the tooth, quality of the bone, esthetic demands, cost-benefit ratio, and systemic factors. There is a higher implant failure rate in diabetics, for example.

Endodontists have no argument with an implant in cases of non-restorable teeth, fractured or vertically split roots, or hopeless periodontally involved teeth. In fact, 6.6% of endodontists, well trained in implantology, are now doing single-tooth implants in their offices. A number of the university graduate endodontic programs have added implants to their curricula approved by the AAE and the ADA.

Iqbal and Kim have pointed out that 28% of the extracted teeth replaced with single tooth implants had already been endodontically treated (Figure E).¹⁰ They noted that “the presence of **apical periodontitis** [a granuloma for example] is increasingly being used to recommend tooth extraction and immediate replant replacement.” Also, teeth with horizontal root fractures constituted 16% of the teeth replaced with single-tooth implants, whereas many of these teeth do not require any intervention and heal naturally. Many others respond favorably to endodontic treatment.

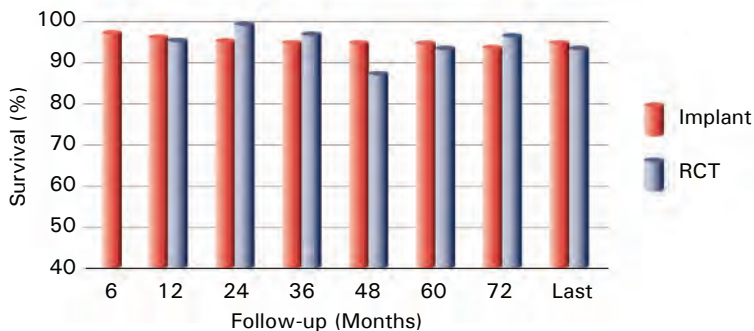


Figure E. A meta-analysis comparison of the survival rates of restored endodontically treated teeth and restored single-tooth implants. Iqbal MK, Kim S. Endodontics: Colleagues for Excellence, Summer 2007. American Association of Endodontists. Reproduced with permission.

Rather than an honest disclosure during informed consent, some dentists are raising the “old bugaboo” of focal infection and citing endodontic failure rates, especially **retreatment** “failure rates,” long ago repudiated. Many salvable teeth are being sacrificed on the “altar of insatiability”—a new version of “**Pull-and-Be-Damned Road.**” Placing single-tooth implants when the natural tooth could predictably be retained would be considered **malpractice**, and leaves the dentist vulnerable to litigation.

In an excellent editorial entitled “Is Endodontic Treatment Passe?” Spangberg recently pointed out that “[t]eeth with failed endodontic treatment are often candidates for some form of retreatment to achieve complete elimination of periradicular disease.”¹¹ He goes on to say that **the claim is often made** that these teeth, scheduled for retreatment, are at high risk of repeated failure. “This misunderstanding has resulted in a never-ending large pool of teeth being candidates for single tooth implants, thus identified by uncritical interests.”

“There are very few teeth that cannot be retained, if the full scope of endodontic treatment options is used. **An implant**, on the other hand, is an excellent treatment option for the replacement of a **missing tooth**. However, it should never be an option for the **replacement of an existing restorable tooth.**”¹¹

What are some other considerations? How about cost? Gordon Christensen, an icon of dental impartiality, stated in 2006: “In terms of costs, a recent analysis of 2005 insurance data concluded that restored single-tooth **implants cost about 75–90 percent**

more than similarly restored endodontically treated teeth.”¹² Here again, Torabinejad noted in 2007 that the initial cost of an extraction, endosteal implant, abutment, and crown is approximately \$2,850, whether a general practitioner, periodontist, or oral surgeon provides the surgical care.⁹ On the other hand, the costs of anterior root canal treatment provided by a general practitioner with a resin-composite restoration is approximately \$743. This simple analysis does not include consultation fees and preoperative radiographs to cone-beam tomography and CT scan for implant placement.

As far as **postoperative intervention** is concerned, it is noted previously in the study of about 400 patients that restored single-tooth **implants** required nearly **five times more postoperative interventions** than similarly restored endodontically treated teeth. These additional visits could account for unforeseen costs, lost wages, and so on. Moreover, Goodacre has pointed out that “[o]ther adverse events can occur during implant surgery, including paresthesia, hematoma, hemorrhage and devitalization of adjacent teeth.”¹³ Endodontic therapy has also been shown in multiple studies to dramatically and significantly reduce pain.

As far as chewing ability is concerned, Woodmansey and his colleagues recently examined the maximum bite force that could be exerted between restored implants and contralateral controls.¹⁴ They found that “dental **implants** were found to have significantly **lower** maximum bite force and reduced chewing efficiency due to a smaller occlusal contact area. Endodontically restored teeth, on the other hand, “were not statistically different than their contralateral controls” (Figure F).

The problem with **esthetics** has become a serious deterrent to implantation. Nothing surpasses the appearance of a natural tooth and a healthy attached gingiva. Due to unavoidable recession in some implant cases, ugly interproximal gingival spaces develop (Figure G-A). In other cases where insufficient bony support is present, unattractive acrylic “gingival” additions have been added for some semblance of natural appearance, or a serious dehiscence develops due to the fenestration of the buccal bony plate (Figure G-B). Also, it is very difficult for an implant replacement to exactly color-match the natural teeth (see Figure G-A).

Louis Rossman, President of the American Association of Endodontists, summarized it best when he said: “I’ve become increasingly disturbed by the speed with which many dentists are

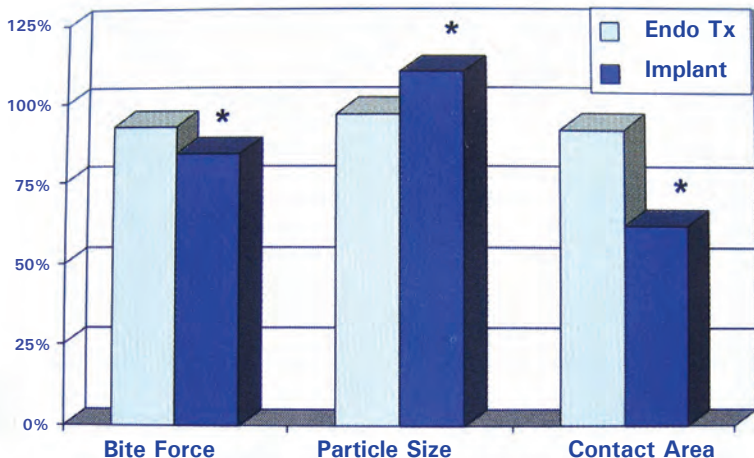


Figure F. Comparison of masticatory performance in endodontically treated teeth vs. implants. Woodmansey KF, et al. J Endod 2009;35(1):10–14. Reproduced with permission.

embracing implants as a replacement for natural teeth that could have been saved with endodontic treatment. Our goal as dentists is to make every reasonable attempt to maintain the natural dentition” (Figure I).¹⁵



Figure G. Rogue’s gallery of implant failures. **A,** Esthetic problems occurring with placement of dental implants in the “esthetic zone.” Notice as well the disparity in the color match between the implant restorations and the natural tooth color. Colleagues for Excellence, Summer 2007, American Association of Endodontists. Courtesy of David Cagna. Reproduced with permission.

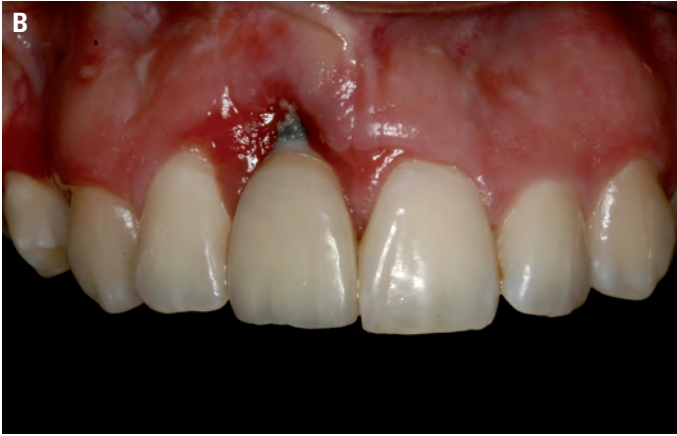
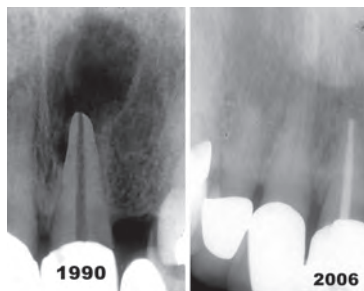


Figure G. Continued, B, Severe dehiscence developed when an implant was placed in a socket with a fenestration or very thin buccal bony plate. Courtesy of Bach Le, the “corrective dentist.”



Figure H. Male, age 44. Following an accident at age 23, totally avulsed teeth #s 7,8 & 9 were replanted, became ankylosed but remained in place for 10 years. After extraction, bone grafting and 3 implants were placed, but infection of the site forced their removal after a few years. Second and third bone grafting and implantation followed, but the tissue suffered continued apical migration due to lack of bone support. Patient declined treatment that included implant removal and settled for a removable partial denture with a labial flange covering the defect. **A,** Patient's present appearance including the advanced labial gingival recession on the canine, **B,** Radiograph reveals the severe bone loss around the implants at teeth 8 & 9. Courtesy of Adriana McGregor, periodontal microsurgeon, Santa Barbara, CA.

Figure 1. Pre- and postoperative radiographs of a central incisor treated with nonsurgical root canal therapy. Sixteen-year follow-up. Courtesy of Louis Rossman. Colleagues for Excellence, Summer 2007, American Association of Endodontists. Reproduced with permission.



REFERENCES

1. Pallasch TJ, Wahi MJ. Focal infection: new age or ancient history. *Endo Topics* 2003;4:32–45.
2. Gutmann JL. History of endodontics. In: Ingle's Endodontics 6th ed. 2008; Shelton, CT: People's Medical Publishing House; 36–85.
3. Baumgartner JC, et al. Incidence of bacteremia related to endodontic procedures. II surgical procedures. *J Endod* 1977;3:399–403.
4. Salehrabi R, Rotstein I. Endodontic treatment outcomes in a large patient population in the USA: an epidemiological study. *J Endod* 2004;30(12): 846–50.
5. Lazarski MP, et al. Epidemiological evaluation of the outcomes of non-surgical root canal treatment in a large cohort of insured dental patients. *J Endod* 2001;27(12):791–6.
6. Creugers NH, et al. A systematic review of single tooth restorations supported by implants. *J of Dent* 2000;28:209.
7. Lindh T, et al. A meta-analysis of implants in partial edentulism. *Clin Oral Implants Res* 1998;9:80–90.
8. Hannahan JP, Eleazer PD. Comparison of the success of implants versus endodontically treated teeth. *J Endod* 2008;334(11):1302.
9. Torabinejad M. Effects of dental implants on treatment planning for endodontics. In: Ingle's Endodontics, 6th ed. 2008; Shelton CT: People's Medical Publishing House; 105–18.
10. Iqbal MK, Kim S. A review of the factors influencing treatment planning decisions of single-tooth implants versus preserving natural teeth with non-surgical endodontic therapy. *J Endod* 2008;34(5):519–29.
11. Spangberg LSW. Is endodontic treatment passé? *Oral Surg Oral Med Oral Path Oral Radiol Endodont.* 2008;106(4):465–66.
12. Christensen GJ. Implant therapy versus endodontic therapy. *J Am Dent Assoc* 2006;137(10):1440–3.
13. Goodacre CJ, et al. Clinical complications with implants and implant prosthesis. *J Prosthet Dent* 2003;90(2):121–32.
14. Woodmansey KE, et al. Differences in mastication function in patients with endodontically treated teeth and single-implant supported prosthesis. *J Endod* 2009;35(1):10–14.
15. Rossman LE. Always aim to save natural teeth. *Endo Trib* 2009;4(1):1–4.

Endodontic Pathology and Successful Treatment

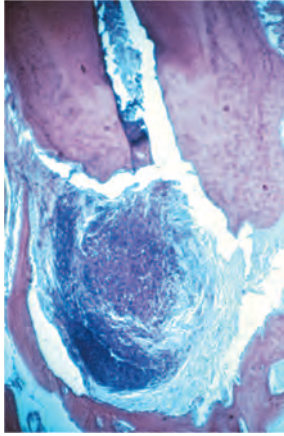


Figure J. "Endodontology deals with diseases of the pulp and periapical tissues, their anatomy, physiology and pathology as well as their diagnosis and treatment."

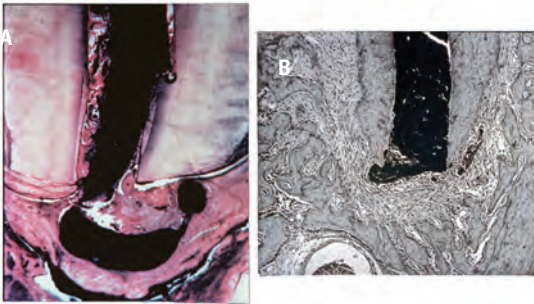


Figure K. "Elimination of bacteria and bacterial plaque as well as complete obturation of the properly prepared canal. **A**, Incomplete obturation of an improperly prepared canal leads to apical inflammation and infection. **B**, Canal properly prepared and obturated leads to complete healing and a return of the normal periapical structures."

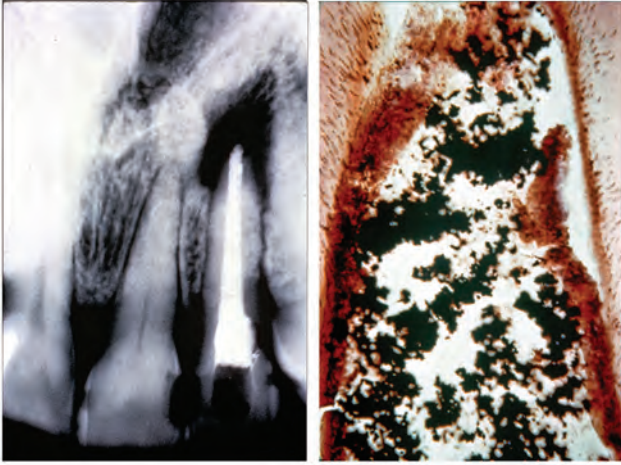


Figure L. *Left*, Apparently obturated root canal filling, yet with persistent apical lesion. *Right*, Amputated root tip shows the failure of compaction as well as debris and bacteria.



Figure M. *A*, Advanced cellulites related to an infected maxillary lateral incisor. *B*, Eight days following incision, drainage, and antibiotic therapy. Courtesy of B. Wolfson.

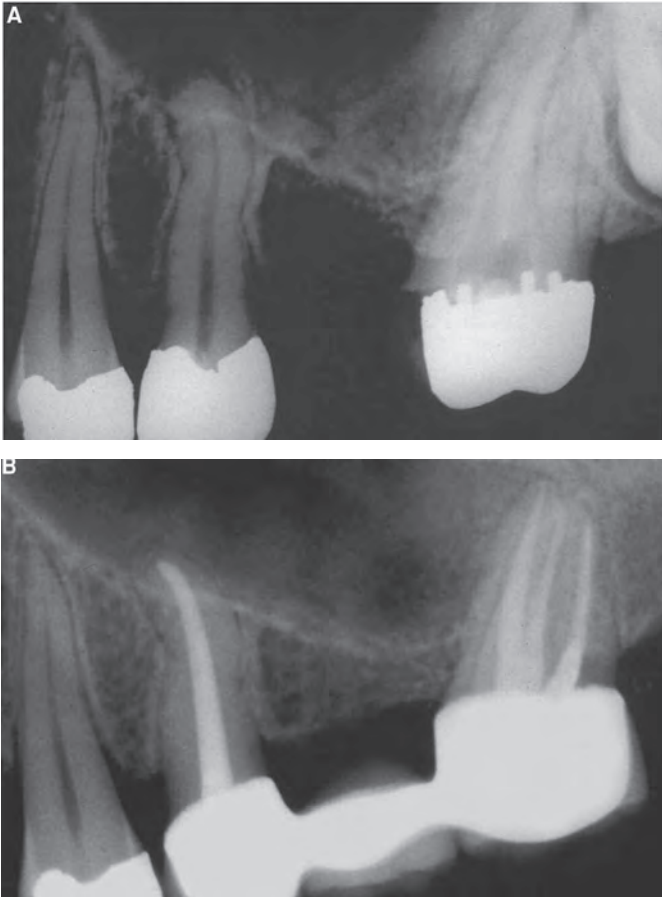


Figure N. Indication for root canal treatment and fixed prosthodontics rather than implant. Note the failing crown on the pulpless molar and the apparent perforation of the sinus floor by the premolar apical lesion. For an implant, there would have had to be a bony graft placed as well. Courtesy of N. Hertl.

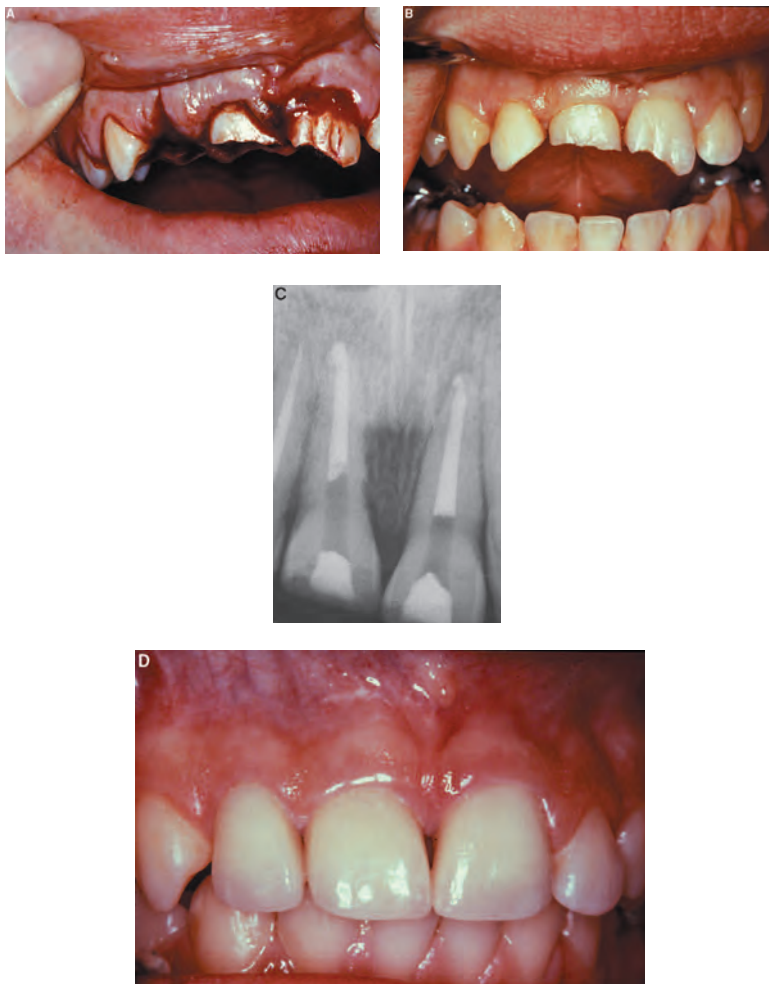


Figure 0. **A**, Two central incisors were fractured through the pulp, and the lateral incisor was totally luxated in a bicycle accident. **B**, The lateral incisor was treated “in hand” and replanted, and the injuries began to heal. **C**, Radiograph illustrates the root canal treatment and replanted lateral incisor. **D**, Final restoration with three porcelain jacket crowns.

Examination, Diagnosis, and Treatment

PULPAL CONDITIONS

Signs and Symptoms

One thinks of pulpalgia—toothache—as the most common symptom of pulp pathology. This may be true in the public's mind but is not necessarily so in dental practice. Dentists recognize discolored or fractured teeth, abscess stomas, and lesions spotted on a radiograph as likely candidates as well.

Nonetheless, toothaches still appear to be a common occurrence. Whether the incidence has increased or decreased over the years is open to question. The last survey available is 11 years old. At that time, the report estimated that 22 million people suffered from toothache that year.¹

Pulp Pain

Pulpalgia (that is, pulp pain) can be classified into three different categories: **hyperreactive**, **acute**, and **chronic**. Histologically, **pulpitis**, that leads to pulpalgia, is classified as **reversible** or **irreversible**. One hopes that pulps with reversible pulpitis can be saved. Pulps suffering irreversible pulpitis cannot be saved—it's that simple!

Dentinal Sensitivity

In the absence of inflammation, **hyperreactive pulpalgia** or **dentinal sensitivity** is the mildest form of pulp discomfort. The pain is often characterized as a short, sharp shock, and it is brought on by some stimulating factor—heat or cold, sweet or sour, acid or touch. **It is never spontaneous!** The reaction patients experience to heat or cold after a new restoration is quite typical of dentinal sensitivity.

Even biting down on a foil candy wrapper sends a shock between two dissimilar metals, gold and silver. Probably the most familiar example is the shocking pain up through the eye that occurs while eating ice cream. It is not pathologic, but rather involves fluid flow in the dentinal tubules that stretch or compress the nerve endings that pass alongside the tubular extensions of the pulp odontoblasts (Figure 1-1).

Dentinal sensitivity may develop when dentin is exposed from gingival recession or following periodontal surgery. The nerves in these exposed tubules respond not only to heat and cold and sweet and sour but also to scratching with an instrument or a fingernail and to tooth brushing. For this reason, patients often avoid brushing the area. The subsequent plaque build-up only worsens the situation.

Examination

Examination of dentinal sensitivity is quite simple: apply the irritant that sets off the painful reaction—heat or cold, sweet or sour, or scratching with an instrument. Radiographic results are generally

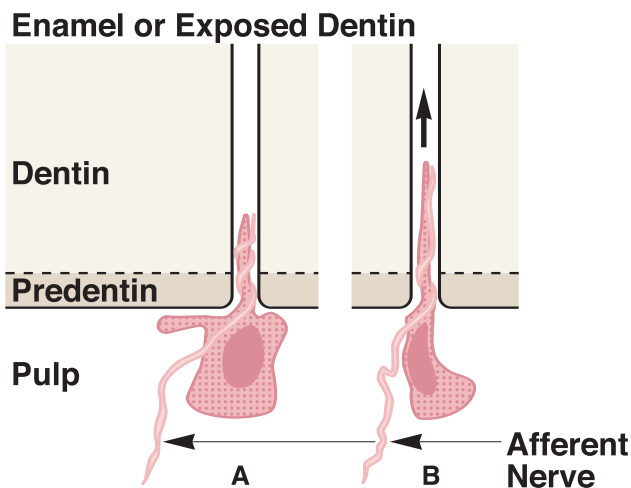


Figure 1-1. The effect of cold stimulus on the pulp. **A**, Cold is applied to the tooth, causing a contraction of the fluid in the tubule. **B**, Pulp capillary pressure forces replacement fluid into the tubule along with the odontoblast and the afferent nerve. Stretching the nerve (*thick arrow*) produces intense pain. Courtesy of M. Brannstrom.

normal, as are electric pulp test readings and percussion. **The pulp condition is totally reversible.**

Treatment

An insulating cement base under amalgam fillings prevents the shock of heat or cold to the pulp. Eventually, irritation dentin will build up to protect the pulp from thermal shock. Marginal micro-leakage around restorations may also lead to hypersensitivity owing to bacterial invasion and irritation. Acidic soft drinks may cause a washout of the smear layer that obstructs the dentinal tubuli. Removal of the smear layer before placing a restoration (Figure 1-2A and B) and coating the exposed dentin with a dentin bonding agent such as **Touch & Bond** (Parkell Co.; Figure 1-2C) protect the tubuli opened during preparation and even serve as insulation in place of a cement base.

Sealing dentinal tubules that terminate at the cementum but become exposed to the oral environment following periodontal surgery may be accomplished by applying potassium oxalate or strontium chloride, fluorides, or dentin bonding agents. Sodium fluoride (2%) applied by iontophoresis has also proved effective, blocking the dentinal tubules. There are also a number of toothpastes that contain 5% potassium nitrate that apparently do not block the tubules but

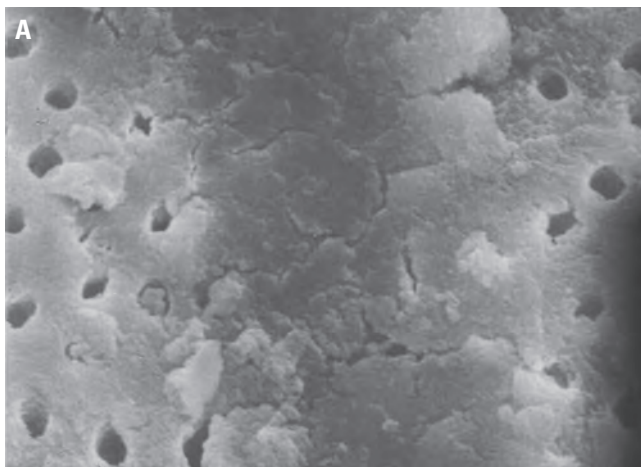


Figure 1-2. A, Smear layer produced by filing in the center of a canal wall formerly cleared of smear. **Continued on next page.**

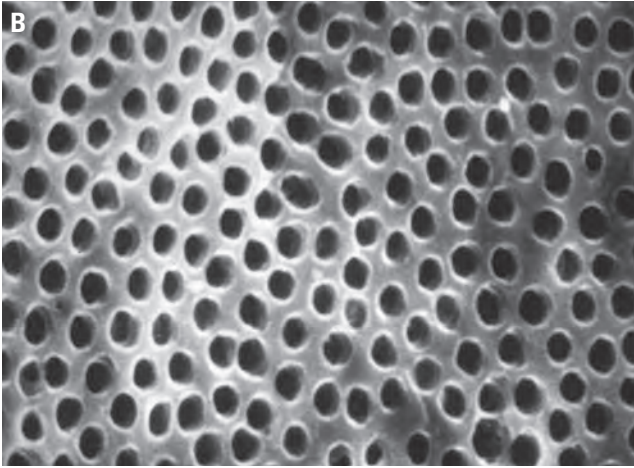


Figure 1-2. Continued. **B**, Removal of the smear layer using ethylenediaminetetraacetic acid (EDTA) and full-strength sodium hypochlorite rinses. **C**, Touch & Bond (Parkell Co.) dentin bonding agent used to close dentin tubuli and prevent hypersensitivity reaction

“numb” the nerve endings. These are sold in most drugstores as GUM (John O. Butler), Sensodyne and Aquafresh Sensitive (Glaxo-SmithKline), and Colgate Sensitive (Colgate-Palmolive). Although the fluid still flows in the tubules, the nerves are “unexcitable.” Finally, the application of dentin adhesives such as **Brush & Bond**



Figure 1-3. Brush & Bond (Parkell Co.) bonding agent contains a special tripolymer that creates a robust film that resists abrasion. When applied to a clean root surface, it alleviates hypersensitivity of exposed dentin.

(Parkell Co.) to the root surfaces of hypersensitive teeth has proved very effective. The roots must be scrupulously clean of dental plaque before the adhesive application (Figure 1-3). All of these modalities of root desensitization have a good “track record” of relieving dental sensitivity. Again, because **the condition is noninflammatory**, it is completely reversible.

Incipient Acute Pulpalgia

Acute pulpalgia is found in three stages of escalating discomfort: **incipient**, **moderate**, and **advanced**. All three stages are related to advancing degrees of inflammation.

Incipient acute pulpalgia should be completely reversible. It is characterized by mild discomfort such as that experienced following cavity or crown preparation. It may be gone by the next day. If one could study the cells of the pulp at this time, one would find a marginal increase in leukocytes and fluid pressure against the nerves that accompany the odontoblasts into the tubules. When pressure returns to normal, the discomfort disappears. Some patients may report slight discomfort from a carious lesion that has just broken through the enamel into the dentin (Figure 1-4). Constant trauma from a “high” filling may also start this mild discomfort.

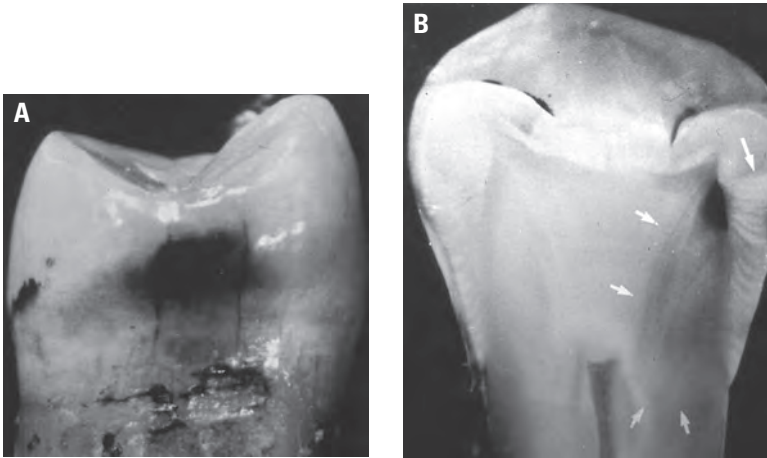


Figure 1-4. **A**, Early carious lesion that has not yet broken through the enamel surface. **B**, Same lesion shown invading the dentin (*arrows*) and already arousing a reaction in the pulp, enough to alert the patient to incipient pulpalgia. Courtesy of K. Langeland.

Examination

Only radiography discloses an incipient interproximal carious lesion. In cases of constant trauma, radiographs may exhibit periodontal membrane (PDM) thickening at the apex, and percussion may elicit a mild response. Pulp test readings are generally normal. Cold is the best stimulus to initiate the discomfort.

Treatment

Time is the best “cure” for the uncomfortable but normal reaction to cavity preparation. If **initial** caries is the cause of the discomfort, no bacteria have reached the pulp, so the obvious first step is to remove the caries and place a calcium hydroxide dressing and sedative zinc oxide-eugenol filling until the sensation returns to normal, then a permanent restoration can be placed. If the cause is trauma, relieving the high spot on the filling or its opposing tooth often brings immediate relief. In any event, these cases should all be reversible and endodontic therapy should be avoided.

Moderate Acute Pulpalgia

Moderate acute pulpalgia is a true but tolerable toothache, often described as “nagging” or “boring.” In such cases inflammation of the pulp is present, so the pulp may be **either reversible or irreversible**. This is extended pain, often diffuse and difficult to locate as it refers to other areas. The pain may start spontaneously or from a simple act such as lying down. Interestingly, cold may be the irritant that starts the pain but hot food or drink and biting down on the cavity are more common. If this pain has been mild and of short duration, the pulpitis may be **reversible** (Figure 1-5). The pulp may not yet be infected by bacteria but may be reacting to their acidic output by becoming inflamed and swollen. However, if the pain has been moderately severe and continuous for some time, the pulp is probably infected. This **pulpitis is irreversible**; the pulp must be sacrificed (Figure 1-6). This is a tough decision one is asked to make—to treat or not to treat. A warm rinse does not relieve the pain, and cold may make it worse. Acetaminophen or ibuprofen may alleviate mild but not severe discomfort.



Figure 1-5. Localized abscess formation (*arrows*) subjacent to dentin cavity (*C*) filled with dental plaque. The pulp may not yet be infected but is reacting to the acidic output from the plaque. Courtesy of G. Bergenholtz.



Figure 1-6. *Left*, Inflammatory changes in the pulp from an open cavity (C). *Right*, Higher power, the pulp is probably infected, and the pulpitis irreversible. Courtesy of I.A. Mjor and L. Tronstad.

Examination

Pinpointing the exact tooth involved in **moderate pulpalgia** is often difficult. Frequently, the results are clouded by the analgesics the patient has been taking for some time. The pain is diffuse, and two or three teeth may give similar responses to electric pulp testing. Percussion may reveal a slight difference in response between teeth. Pain may even be referred to the opposing arch. Radiographs may reveal large interproximal caries or a deep filling impinging on the pulp (Figure 1-7). There may be a thickening of the PDM at the apex. Thermal testing with cold should be attempted first. If pain response from the suspected tooth increases but then goes away, **stop!** Do not test other teeth, but wait for the rebound of pain that may occur. Then test the other suspected teeth to learn if they respond with the intensity of the prime suspect. When using the electric pulp tester, the involved tooth may respond a bit sooner than its neighbors, or the tooth may be a bit more sensitive to percussion. If there is pain in both arches, locally anesthetize the principal suspect. Pain elimination in both arches confirms the correct identification of the tooth and the pain referral. The anesthetic test is the last resort. Be patient; be shrewd. If in doubt, hesitate. One more day may be the panacea.

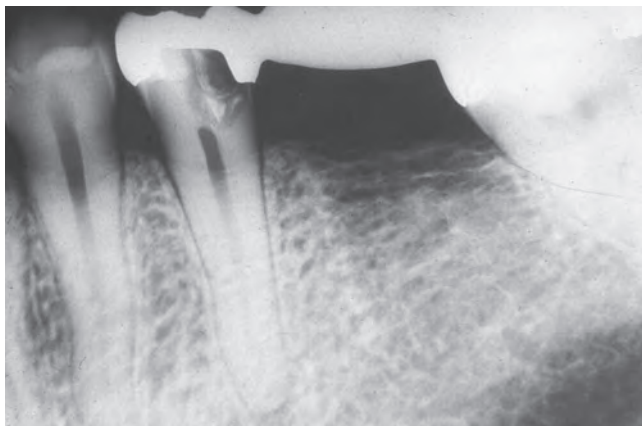


Figure 1-7. Undetected caries under a premolar bridge abutment. Irreversible pulpitis leading to moderate pulpitis.

Treatment

In the event moderate pulpitis is **mild and of short duration**, there is an outside possibility that the **pulpitis is not infected and is reversible**. In that case all the caries must be removed, and a calcium hydroxide dressing may be placed along with a thick, zinc oxide-eugenol, sedative, temporary filling. If the tooth remains symptom-free for some time, a more permanent filling may be placed with the proviso that root canal therapy may have to be done if symptoms return. On the other hand, **most pulps of moderate acute pulpitis are infected, inflamed, and irreversible**. Unless one opens into the pulp and discovers pus in the chamber, one is only guessing as to the state of the pulp. Playing the odds that any pulp that has been aching for some time is infected, one is wise to remove the pulp and clean, shape, and fill the root canal if the tooth is salvable. One can “play around” with a pulpotomy and calcium hydroxide dressing for some time, but the odds that the pulp will survive are less than 50:50. **This is irreversible pulpitis.**

Advanced Acute Pulpalgia

Advanced acute pulpalgia is the ultimate toothache, one of the most excruciating pains known to a human. **It is totally irreversible pulpitis.** The patient may be in exquisite agony, at the point of hysteria,

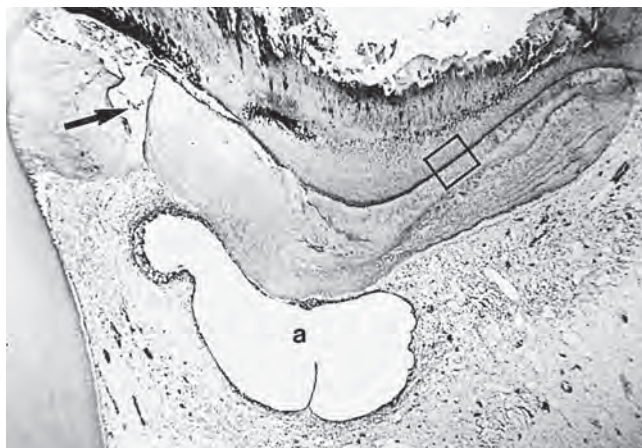


Figure 1-8. Although extensive irritation dentin has formed under carious lesion, it has not protected the pulp from the effects, as shown by the micro-abscess (*a*). The barrier is incomplete, as is evidenced by the opening (*arrow*) forming communication between the pulp and the carious lesion. Courtesy of K. Langeland.

crying and unmanageable. The relief of this pain is embarrassingly simple: cold water relieves the pain temporarily. The patient may report to the dental office with a jar of ice water. Histologically, one finds necrosis of the coronal pulp with vital remnants left (Figure 1-8). Gas has formed, and it is the contraction by this gas and excess fluid in response to the cold that gives 30 to 45 seconds of relief at a time.

Examination

There is usually not much question which tooth is involved. Testing with heat gives an immediate explosive response! One must be prepared to immediately bathe the tooth in ice water. The tooth will be responsive to percussion but diagnostic radiography at this point is irrelevant, as is the electric pulp tester. The symptoms are self-incriminating. **This is irreversible pulpitis!** An immediate block injection gives blessed relief.

Treatment

There is only one solution for this devastatingly painful experience—pulp removal either by pulpectomy or by extraction. If the tooth is

salvable, pulpectomy and root canal treatment is the preferred choice. Anesthesia may be difficult to achieve, and one may have to resort to intrapulpal anesthesia. The tooth may have risen in its socket, so the occlusion should be relieved. One must be careful in performing the pulpectomy that all the pulp is removed; otherwise, the patient may be back with continuing toothache when the anesthetic wears off.

Chronic Pulpalgia

Chronic pulpalgia deals with completely irreversible pulpitis. It is often described as a “grumble,” not severe but consistent discomfort. Patients have admitted withstanding the discomfort for weeks or even years, suppressing the pain with analgesics. On the other hand, they may not have had any overt symptoms that would alert them to seek a dentist. Finally, when the pulp starts to ache all night or flare up during an airplane flight, they come in for treatment. The pain is mild enough and diffuse enough to complicate its location. Moreover, chronic pulpalgia often refers pain to other teeth or the opposing arch. Pain may be precipitated by biting down on an open cavity. Cold has little effect, but heat may increase the discomfort. If one could see the pulp, one would find it mostly necrotic but with enough vital remnants to extend sensation. The barodontalgia during flight ascent is related to reduced cabin pressure allowing the pulp to “swell” more. Dull throbbing pain is the result. It may disappear during descent.

Examination

Oral examination often reveals the culprit. A huge carious lesion or fractured filling may pinpoint the involved tooth. The electric pulp test will record a very high reading, or the patient may be able to withstand the “full discharge.” Percussion often reveals one tooth to be more sensitive than others, but biting hard on a cotton roll or a hot water rinse may be even more indicative. Radiographic evidence is often the best. A huge interproximal cavity may appear, or there may be recurrent caries under an inlay. Teeth suffering chronic pulpalgia (and chronic pulpitis) may have a well-thickened apical PDM as well as external root resorption. **Condensing osteitis** of the surrounding cancellous bone is pathognomonic of chronic pulpitis (Figure 1-9A).

Interestingly, after the tooth is treated endodontically, the osteosclerosis disappears (Figure 1-9B). Molars inflicted with chronic

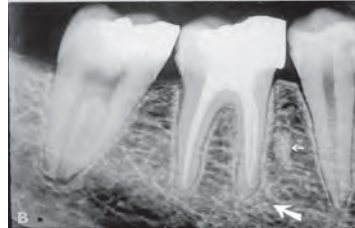
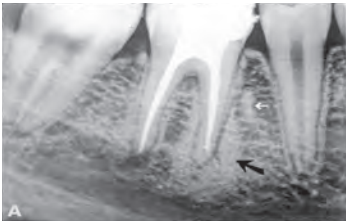


Figure 1-9. Top, Condensing osteitis, external resorption, and pulp stones associated with chronic pulpitis in a first molar. A & B Condensing osteitis disappears following successful root canal treatment. Note primary root tip does not change (*small arrow*).

pulpitis are notorious for referring pain to other regions, the opposing arch being the favorite.

Treatment

Chronic pulpitis is totally irreversible, and the tooth must be treated endodontically or extracted if not salvable.

Pulp Necrosis

Pulp necrosis should be completely devoid of pain in that all pulpal tissue is destroyed, including the sensory nerves (Figure 1-10). Pulp may die quietly over the years or may die a painful death, as described previously. It is virtually always infected. Of course, caries are the greatest source of bacteria. Some necrotic cases may be

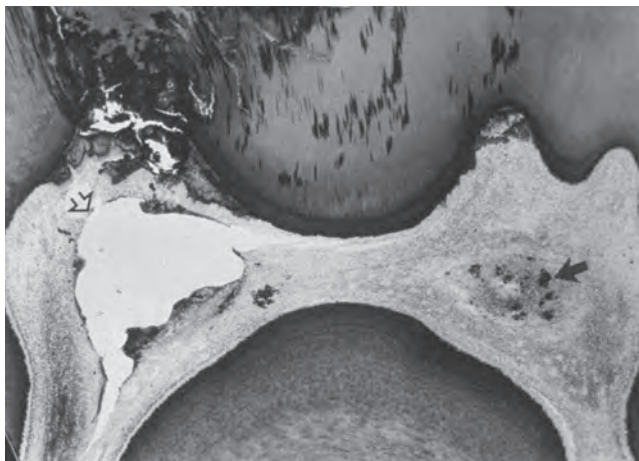


Figure 1-10. Massive, rapidly advancing pulp necrosis. Bacterial penetration leads to microabscess (*open arrow*). Bacterial masses are indicated by the *solid arrow*. Courtesy of S. Matsumiya and colleagues.

due to trauma in which the apical blood supply is cut off. So-called “quiet deaths” may be related to coronal leakage around fillings or crowns. Over time, bacteria invade an already traumatized pulp and slowly bring about its death. The process may be symptom-free but revealed to the dentist as discoloration under transillumination. Most quiet necrosis cases are noticed on radiographs by the appearance of root resorption or periradicular lesions.

Examination

Pulp testing, either thermal or electric, is an accurate way to prove necrosis. If a full crown is present, the necrotic pulp will not respond to heat or cold. There will also be no response to electric pulp testing unless one or two roots in a multirooted tooth are still partially vital and another is necrotic. This situation of partial necrosis leads to confusing results. The tooth responds as not totally necrotic but at a much higher scale than do comparable teeth. One learns soon enough not to trust radiography alone. A tooth can be involved in a condition known as osteofibrosis, in which the first stage appears as a periapical radiolucency (Figure 1-11); pulp testing reveals that the pulp is vital, and no treatment is advised. **Always test the pulp!**

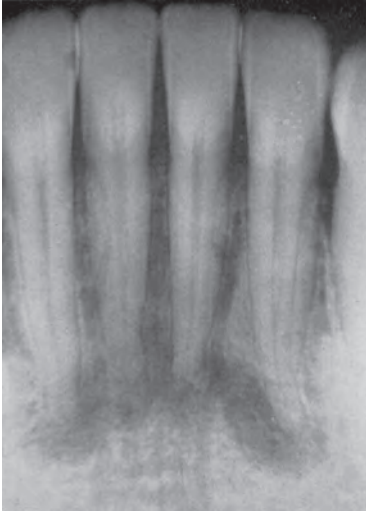


Figure 1-11. Initial stage of osteofibrosis, that can be mistaken for pulpal and periradicular disease. The pulps are all vital. Courtesy of S.N. Bhaskar.

Treatment

Again, as with chronic irreversible pulpitis, the solution is to eliminate pulp necrosis and its sequela by cleaning, shaping, disinfecting, and obturating the root canal, or by extraction if the tooth is not salvageable. In cleaning and irrigating the canal, one must be careful not to cause extrusion of infection out of the apical foramen (Figure 1-12).



Figure 1-12. Perforation of the apical foramen leads to extrusion of infected material during instrumentation. The result is often the excruciating pain of acute apical periodontitis.

ORAL AND EXTRAORAL PAIN REFERRALS

Pulpalgia is notorious for referring its pain to other teeth or regions—upper to lower premolars, maxillary to mandibular molars, upper incisors to a frontal headache, maxillary posterior teeth to the sinus area, lower molars to the angle of the mandible and even the ear and, quite commonly, lower third molars to the ear. Fortunately, pain usually does not cross the midline. If a patient insists a lower molar is aching and yet all testing and examination of that area is negative, test the opposing molars. If there appears to be **cause for pain** in that arch but no pain is felt there, anesthetize that area; if the pain is referred, it will disappear in the opposite arch.

There is also a condition called **atypical odontalgia**. It involves all the symptoms of an acute toothache: severe throbbing and continuous pain starting in one quadrant and even crossing the midline. This condition is also called “**phantom pain**” or “**dental migraine**” and is often associated with patients suffering from unipolar depression. Over 80% of the patients are female, and over 90% of the time the pain is in the teeth, jaws, or gingivae. In almost one third of cases, the pain is precipitated by dental procedures. Tricyclic or monoamine oxidase inhibitor anti-depression therapy relieves the pain in many cases.

The most common extraoral pain reference comes from the heart—**myocardial infarction**, **coronary thrombosis**, or **angina pectoris**. The patient may present with a typical toothache or a pain in the jaw, usually the left jaw. If testing for pulpalgia (usually with an ice water rinse) does not change the character of the pain, one should then question the patient about other areas that might be painful, such as a “smothering” feeling in the chest or reference pain in the left arm or even the right arm (Figure 1-13). Sometimes only the jaw pain is apparent, and it is these patients who are most likely to appear in a dental office. If a heart attack is suspected, great care must be exercised in getting the patient into proper hands.

Pain to the teeth and jaws can also be referred from the maxillary sinus or the thyroid gland, or from a cardiospasm, a spasm of the cardiac sphincter of the esophagus often associated with a hiatal hernia.

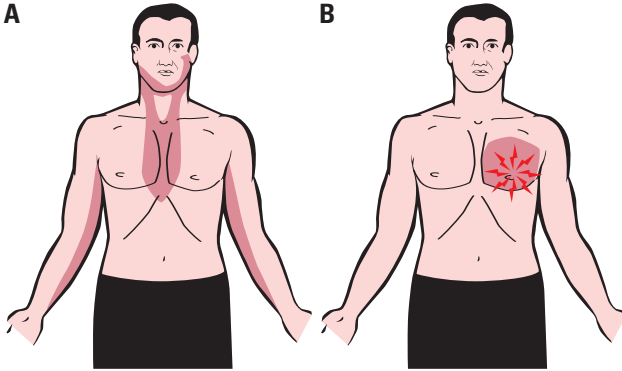


Figure 1-13. **A**, Pattern of referred pain emanating from a myocardial infarction. A central “necktie” pattern and greater pain in the left jaw and left arm than on the right side are typical. **B**, Harmless noncardiac pain in the left chest, often from a spasm of the esophageal sphincter. Courtesy of G.O. Turner.

Internal Resorption

Internal resorption is an insidious process because there may not be any symptoms involved. The only sign may be an unexpected radiographic discovery. However, in some cases internal resorption may mimic the mild pain of moderate acute pulpalgia. Another sign might be pink tooth of Mummery (Figure 1-14) if the crown has been thoroughly undermined by the resorption. The cause of internal resorption is usually thought to be impact trauma, and such



Figure 1-14. Pink tooth of Mummery, the result of internal resorption within the crown of a central incisor. Courtesy of L.K. Bakland.

may be the case. Iatrogenic damage to the pulp during overzealous crown preparation is another form of trauma (Figure 1-15). On the other hand, dental caries have been indicted as well. The pulp simply changes character, and instead of the odontoblasts producing dentin, dentinoclasts develop and destroy dentin.



Figure 1-15. Extensive internal resorption triggered by iatrogenic causes. **A** and **B**, Normal condition of teeth prior to crown preparation. **C**, Development of internal resorption from high-speed preparation without water coolant, seen 1 year later. Courtesy of D. H. Glick.

Examination

Pink teeth are an obvious sign of internal resorption. But if the resorption is confined to the root, only radiography will reveal the destruction. On the radiograph, the lesion has smooth walls and the pulp seems to disappear into the lesion (Figure 1-16). This is in contrast to the appearance of external resorption, in which the pulp seems to pass through the lesion. Thermal and electric pulp testing and percussion are only partially reliable when comparing a

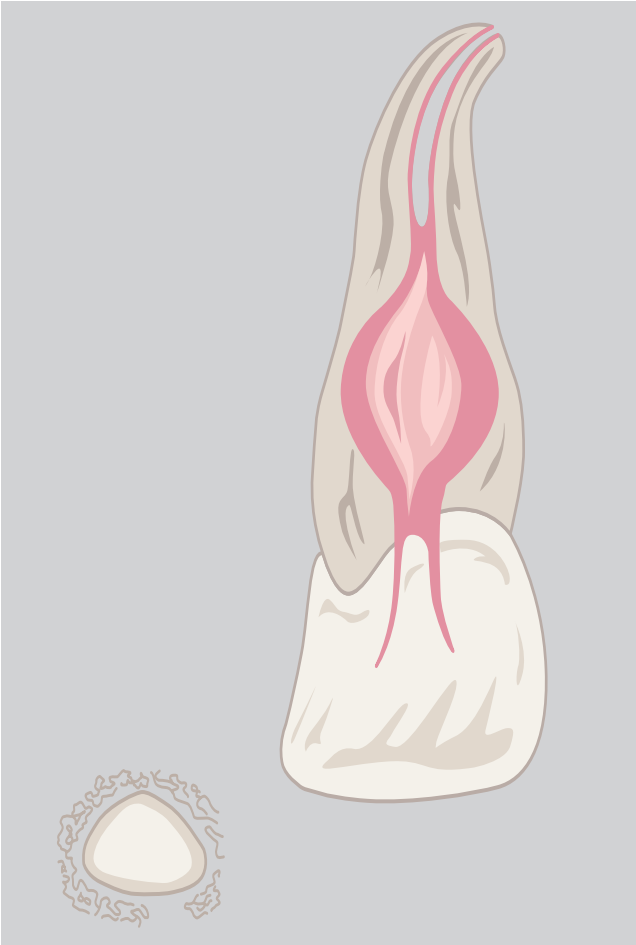


Figure 1-16. Drawing of internal resorption shows how the shadow of the pulp “disappears” into the huge lesion. Courtesy of F.H. Lepp.

resorptive tooth with neighboring teeth. The most difficult cases to diagnose are those with full coverage and internal resorption confined to the crown. Radiographically, nothing shows, so one must depend on vague symptoms and mild differences in thermal test results. Experience and “playing a hunch” contribute a great deal to the decision making. However, one thinks twice before cutting into a new gold crown.

Treatment

The only treatment for internal resorption is pulp extirpation followed by cleaning, shaping, and obturation. The pulp is the offending organ and must be removed before the resorptive process “eats its way” to the external root surface. Internal resorption is irreversible.

Tooth Infraction

Tooth infraction (often called cracked tooth or split tooth syndrome) involves a tooth that is split or cracked but the two parts are not yet separated. It presents bizarre symptoms ranging from constant unexplained hypersensitivity to constant unexplained toothache. Biting down on the tooth may bring on a sudden surge of pain as the dentin spreads along the fracture line. Many of these cases involve non-carious virgin teeth, so it is difficult to believe that anything is wrong with the tooth (Figure 1-17). As long as the crack does not extend into the pulp, the tooth is salvable by full crowning. Once it extends into the pulp, bacteria establish pulpal infection. At this point, one must decide if the tooth is salvable when the fractured cusp is removed.

A tooth crowned to prevent further fracture may give years of uneventful service only to again become painful. This usually indicates the fracture has extended into the root, and now the pain is coming from the inflamed or infected periodontal ligament. The best test is to bite down on a wet cotton roll. This tooth or root must be sacrificed.

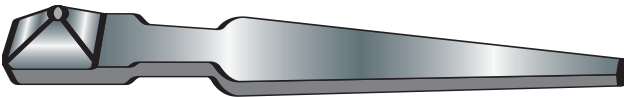
Examination

Sometimes the crack may be discerned if the tooth is dried and viewed under transillumination. Painting tincture of iodine on the surface and washing it off after 2 minutes may reveal a dark line at



Figure 1-17. Crown-to-root infraction that extends into the pulp. Courtesy of L.K. Bakland.

the fracture. The electric pulp test has little value unless the pulp is involved. Thermal testing with a stream of hot or cold water may be of help. Percussion is usually not helpful, yet biting on a wet cotton roll may cause the spreading action needed to elicit pain. To individually test each cusp, biting down on the **Tooth Slooth** (Professional Results, Inc.) is most helpful (Figure 1-18). Radiographs are meaningful only if the fracture line is from buccal to lingual, in line with the x-ray beam.



Tooth Slooth

Figure 1-18. The Tooth Slooth (Professional Results, Inc.), a fracture detector. The concave area at the peak of the pyramid is placed on the tip of the suspected cusp. The patient bites down on the plastic device, and the dentist watches for changes in facial expression or a verbal reaction. Several cusps should be tested.

Treatment

As stated previously, if the fracture does not extend to the pulp or into the periodontal ligament, the tooth may be prepared for a full crown. It should then be cemented temporarily with zinc oxide-eugenol cement until one is certain the tooth is symptom-free. If the infraction extends into the pulp, but not the root, root canal therapy is indicated, and the crown can later be restored with full coverage.

PERIRADICULAR CONDITIONS

Signs and Symptoms

Apical periodontitis is characterized in two ways: **acute** and **chronic**, as well as **symptomatic** and **asymptomatic**.

Symptomatic Apical Periodontitis

Symptomatic apical periodontitis (SAP) is comparable in pain intensity to advanced acute pulpalgia, but SAP lasts longer—24 hours a day, day after day. SAP is frequently caused by iatral blundering, perforating the apical foramen and forcing bacteria, necrotic debris, or caustic medicaments into the periapical tissues (Figure 1-19). Violent inflammation develops and, in the case of bacteria, apical infection.

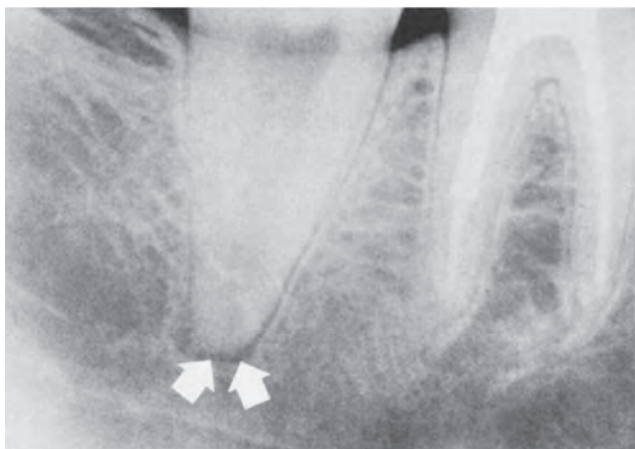


Figure 1-19. Radiographic features of symptomatic apical periodontitis. The periodontal ligament space is widened at the apex (*arrows*). Clinically, the tooth is exquisitely sensitive to percussion.

Because of the confining cortical bone and the paucity of cancellous bone, especially in the mandibular premolar region, there is no room for fluid expansion and the pressure becomes unbearable. The tooth is elevated in its socket, and at every mandibular closure the area is further traumatized. Pain is described as constant, gnawing, pounding, and throbbing.

Examination

The patient is in severe pain, and the involved tooth is exquisitely painful to touch, even the touch of the tongue! The tooth is in supra-occlusion. Radiographs should be taken for records' sake but are not particularly helpful. They will only show a widened PDM, no radiolucent area. Vitality testing is not indicated. **Don't even try percussion!**

Treatment

After examination is completed and before any treatment is undertaken, long-acting block anesthesia such as bupivacaine should be given. Bupivacaine injections should be repeated at least twice a day, preferably in the morning and at the last appointment of the day to tide the patient over the night. An evening phone call and the first appointment the next day are in order.

Be wary before undertaking any procedure. Approach the tooth gently to be sure it is numb. First the occlusion must be relieved, if at all possible, in the opposite arch. The involved tooth should be supported between the thumb and forefinger during any treatment. A rubber dam should be placed. Then carefully remove the temporary filling and gently use paper points to empty the canal of fluid. Radiographically check the tooth length that may be in error. Using a small reamer, set the exact tooth length and then gently perforate the apical foramen to relieve any trapped fluid. **Neo-Cortef** 1.5% eye/ear drops (Upjohn Co.) should then be placed in the canal and teased out the apex. This supplies both antibiotic and anti-inflammatory therapy. The procedure can be repeated that same day, if necessary. Place a small, dry cotton pellet in the chamber and a thin temporary filling. Recheck the occlusion. If the pain becomes unbearable at night, the patient should be instructed how to remove the filling, watching in a mirror and using a safety pin bent at a right angle. This may relieve the pressure. The patient should also be carried on antibiotics and a nonsteroidal anti-inflammatory drug

such as aspirin or acetaminophen. A narcotic drug such as codeine, hydrocodone bitartrate-acetaminophen, or meperidine hydrochloride may also be prescribed. SAP results in one of the most severe pains with which a dentist and patient must deal. It goes without saying that root canal treatment should not be undertaken until all the acute symptoms have subsided.

Acute Apical Abscess

Acute apical abscess (AAA) is a periapical extension of canal necrosis in which bacteria overwhelm the body's resistance and severe inflammation with pus formation develops. AAA carries a severe pain but is not as painful as SAP. The gross swelling itself is painful—a full, systolic, throbbing pain. As the abscess grows and becomes indurated (hardened), the pain increases. When it breaks through the bony plate and becomes soft (fluctuant), it becomes less painful. The abscess has grown from undetected swelling to gross cellulitis (Figure 1-20).

Examination

Here again, the signs of an abscess are readily apparent: the swelling, the redness, and later the “pointing” of the abscess. Gentle



Figure 1-20. Massive cellulitis from a lower molar involving the cheek and neck. Courtesy of J.F. Siqueira.

palpation reveals the early swelling. But which tooth is involved? The electric pulp test is very helpful. The tooth with the necrotic pulp and with no response to the electric pulp test is the obvious one. However, test other teeth in the region because more than one tooth may have a necrotic pulp, particularly after an accident. Very gentle percussion also confirms the involved tooth. No gross radiographic changes may be noted at first, but late in the process rapid bone loss shows up as a radiolucency.

Treatment

In its early stages, the abscess may be drained through the root canal that can be opened slightly with a reamer. If this does not work, the cavity should be closed and the abscess encouraged to “come to a head,” so to speak. It is usually wise to start the patient on an antibiotic, not to control the abscess—which is probably beyond being reversed—but to protect against bacteremia. The patient should be urged to use frequent hot rinses, which will speed up the process of pointing. Once the abscess becomes fluctuant (soft), incision and drainage is employed to drain the abscess (Figure 1-21). Once the whole area “quiets down,” root canal treatment may be completed.

Chronic Apical Abscess

Chronic apical abscess (CAA) is an abscess from low virulence bacteria, usually characterized by a long-standing fistulous stoma



Figure 1-21. Purulent drainage following the incision of a fluctuant acute abscess involving a pulpless lower anterior tooth.

draining the abscess into the oral cavity (Figure 1-22A) but occasionally extraorally—the **cutaneous fistula** (Figure 1-22B). It is relatively painless or asymptomatic, and for this reason patients often tolerate the condition for months or even years. This chronic abscess may also develop an acute exacerbation, the so-called **phoenix abscess**. When this happens, the patient suffers all the attendant problems of an acute apical abscess but “magnified.”

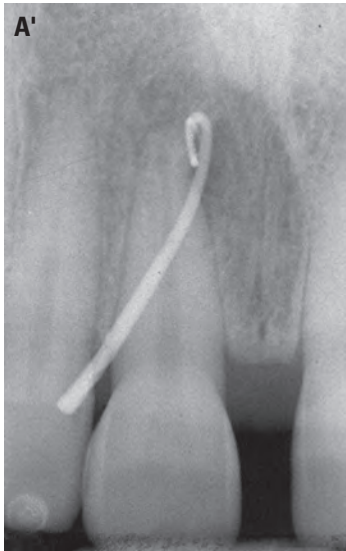


Figure 1-22. **A**, Chronic apical abscess and its intraoral stoma draining through a fistula from the abscess area. **A'**, Gutta-percha point inserted through the stoma indicates the tooth and chronic lesion. **Continued on next page.**

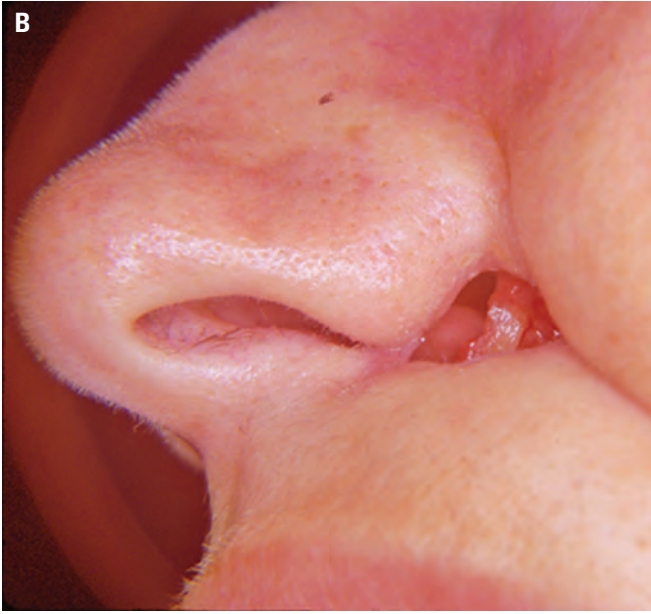


Figure 1-22. *B*, Draining extraoral fistula associated with infected pulpless incisor. *B'* Plastic surgery repair following successful root canal treatment. Courtesy of W.E. Harris.

Examination

If a draining fistula is present, the process is quite apparent. One should probe the fistulous track with a gutta-percha point, using a radiograph as a guide. Often what is thought to be the obvious tooth may not actually be the culprit. Radiographs reveal a diffuse radiolucency, sometimes of enormous size (Figure 1-23). Again, thermal and electric pulp testing reveals the necrotic pulp.

Treatment

Root canal treatment alone often solves the abscess problem. Often the fistula stops draining after the first appointment, and over a period of time the abscess area will heal and be filled in with new bone. Occasionally, surgery must be resorted to, particularly if the root apex has been “chewed-up” by inflammatory resorption (Figure 1-24). The open apex may be difficult to obturate, so surgery and a root-end filling may be in order. Removing all the chronic inflammatory cells from the region may also speed up healing.

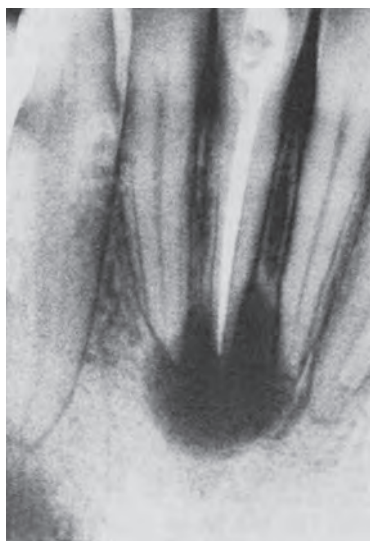


Figure 1-23. Chronic apical abscess related to improperly filled central incisor led to cutaneous fistulous drainage. Courtesy of L.K. Bakland.

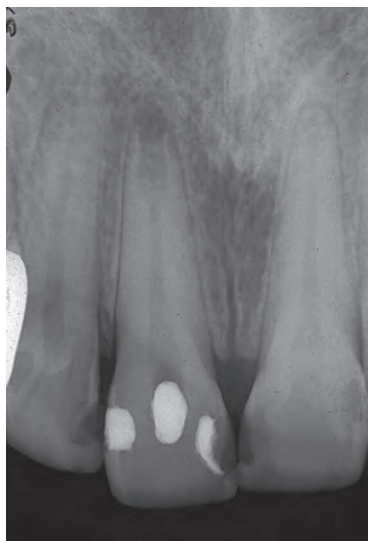


Figure 1-24. Apical inflammatory resorption has destroyed the “apical stop,” so periapical surgery and a root-end filling is recommended to complete treatment. Chronic inflammatory tissue is also removed.

Asymptomatic Apical Periodontitis

Asymptomatic apical periodontitis is a painless (or relatively painless) condition often referred to as a granuloma (Figure 1-25A). It is usually characterized by the radiographic appearance of a well-defined periapical lesion (Figure 1-25B). The patient may not even be aware of the situation. It is always associated with a tooth with



Figure 1-25. **A**, Asymptomatic apical periodontitis, also known as apical granuloma. The lesion is primarily made up of chronic inflammatory cells. No necrosis is present. Courtesy of S. Matsumiya. **B**, Radiographic appearance of asymptomatic apical periodontitis. Two distinct lesions are apparent at the apex of lower first molar with necrotic pulp.

a necrotic pulp that may have been devitalized by trauma or caries. Bacteria in the canal must be of low virulence, well controlled by layers of a periapical inflammatory lesion. It can spring into violence if virulent bacteria take over and a phoenix abscess develops, or it may permutate into an apical cyst.

Examination

Asymptomatic apical periodontitis is usually revealed in a routine radiograph. Both the patient and dentist are surprised to find the well-defined lesion. External resorption has usually not destroyed the root end. By transillumination, the involved tooth appears darker than its neighbors. Sometimes this is even apparent to the naked eye. Thermal and electric testing are negative. Percussion is of little value. If the lesion has been the result of an accident and has been present a long time, the patient often questions the necessity of further treatment. The logical argument is the possibility of a flare-up into a phoenix abscess and the possible dissemination of bacteria to a previously injured part such as a heart valve.

Treatment

Usually, this condition can be resolved by root canal treatment. Surgical intervention is usually not necessary. When it is, the granuloma can be curetted out to speed up healing.

Apical Cyst

An apical cyst is a condition of the lesion of apical periodontitis gone awry. The inflammatory process stimulates the epithelial resting cells of Malassez, and a cystic cavity filled with cholesterol and fluid develops around the apex. It may grow by expansion from the fluid, or it may become infected. In either case, it is pathologic (Figure 1-26A).

Examination

Like its precursor, CAA, the apical cyst is often found in a radiograph (Figure 1-26B). Unless it has grown to the point where it is moving teeth (which is pathognomonic of a cyst) or becomes infected and an abscess develops, the apical cyst may go unnoticed for years (Figure 1-26C). The involved tooth does not respond to thermal or electric stimulation, and may be slightly discolored in transillumination. Percussion should be negative, but if the cyst has grown to the size where it perforates the cortical plate, it may be palpated.

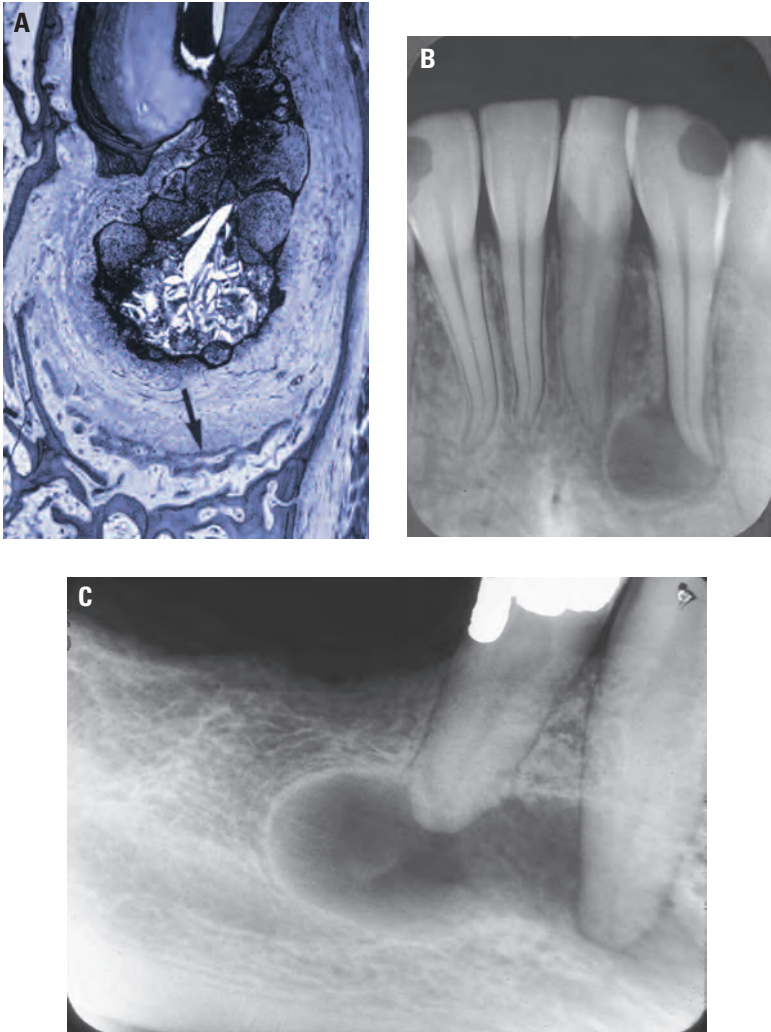


Figure 1-26. **A**, Apical cyst with marked inflammatory overlay. Spaces indicate where crystalline cholesterol has formed from the fluid within the cyst. New bone formation (arrow) is also apparent. Courtesy of S. Matsumiya. **B**, Apical cyst. Growth has caused the movement of associated teeth, which is pathognomonic of a cyst. A perfectly round radiographic appearance is also characteristic but may be confused with a granuloma, which is its precursor. **C**, Slow-growing, asymptomatic apical cysts may go unnoticed for years until revealed by radiography. Courtesy of C. Ames.

Treatment

Root canal treatment and periapical surgery are recommended for removing an apical cyst. Although evidence exists that a cyst may heal after the canal is disinfected and obturated, there is also evidence that some cysts do not heal when the causal irritant is removed. Because one does not know whether the cyst will resolve or continue to grow, it is recommended they be enucleated.

Differential Diagnosis

There are other lesions of non-pulpal origin that may be misdiagnosed as endodontic lesions: carcinoma and sarcoma, ossifying fibroma, cementoblastoma, central giant cell granuloma, nasopalatine duct cyst, enostosis, central ossifying fibroma, and ameloblastoma. Errors in diagnosis can be both health- and life-threatening. A good example is a case of ameloblastoma that went undiagnosed for 9 years as it proliferated across the midline from molar to molar. At one point, an endodontist performed root canal therapy on a premolar involved in the lesion that in no way resembled a typical periapical lesion (Figure 1-27). The patient, who lost the mandible from first molar to first molar, sued for and received over \$1 million (US).

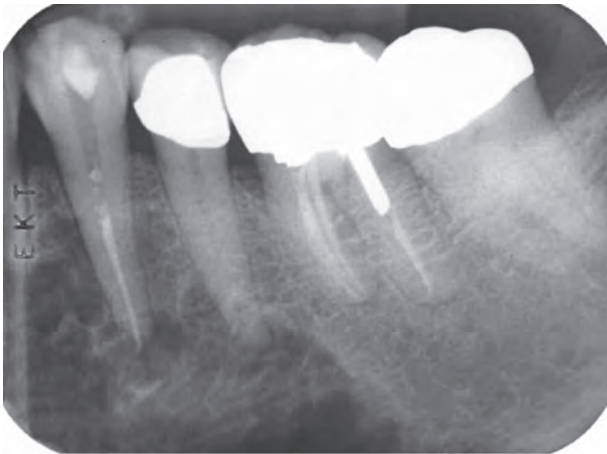


Figure 1-27. Ameloblastoma that originated at the apex of a mandibular first premolar. It was ignored for years and then diagnosed as an apical granuloma, at which time root canal treatment was done. The ameloblastoma grew from first molar to first molar, and this huge section of the mandible had to be amputated and restored later.

INSTRUMENTS, DEVICES, AND EQUIPMENT USED FOR DIAGNOSIS

Along with patience, visual and mental acuity are the greatest assets one must have in making a proper diagnosis. One must learn to “listen with a third ear,” as well. In addition to training, experience, and knowledge, a few simple instruments are also very helpful—an explorer, dental mirror, and periodontal probe. Do the simple things first—look, listen, probe, and explore. Beyond that, there are a number of devices that uncover or confirm the presence of dental disease: radiography, pulp testers (electric or thermal), bright illumination or transillumination, magnification, and local anesthesia.

Radiography: Film and Digital

It goes without saying that radiography is our most valuable tool in endodontics. Through it we discover the unknown, prove the known, determine length and curvature of roots, make decisions concerning the adequacy of our treatment, and follow-up to assess eventual healing or lack thereof. We would be “blind” without these unseen rays.

One must always be aware that a radiograph is a two-dimensional shadow of a three-dimensional situation. If in doubt, take another film from a different horizontal angle. Periapical radiolucencies may “move around” and “detach” from the apex, proving to be foramina rather than lesions (Figure 1-28). One canal can turn out to be two or even three canals. Learn to review one structure at a time. For example, follow around the **lamina dura** of each tooth in the film or the series before moving on to examine each crown in detail. Then follow the crest of the alveolar process all the way around. If the lamina dura at the apex disappears, look carefully under magnification to determine if resorption has taken place. This could open up the apical foramen, the “portal of exit” so to speak, signaling the loss of a normal “apical stop” so necessary during preparation and obturation (Figure 1-29). Anomalies should become apparent—dens evaginatus, dens invaginatus, palatogingival groove (Figure 1-30). Follow-up probing with a periodontal probe often confirms the radiographic diagnosis.

The peculiar patterns of internal and external resorption or orthodontic root resorption should warn of future trouble. As

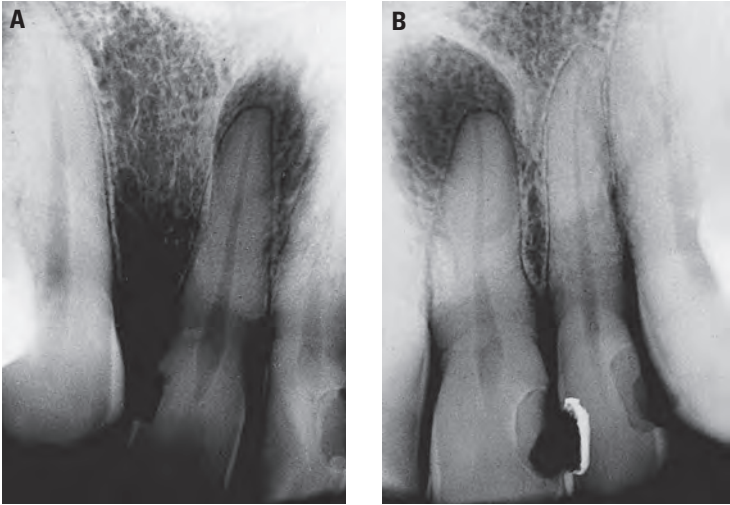


Figure 1-28. Method used to determine the relationship of a radiolucency to the periapex of a tooth. **A**, The nasopalatine foramen is superimposed over the apex of the **right** central incisor. The right lateral is missing. **B**, By changing the horizontal projection, the shadow of the nasopalatine foramen is now superimposed over the apex of the **left** central incisor, proving that the radiolucent area is some distance lingual to the apex of both teeth.



Figure 1-29. Apical root resorption of an infected pulpless tooth, opening up the foramen "portal of exit," thus destroying the "apical stop" so necessary during preparation and obturation. Courtesy of I. Brynolf.

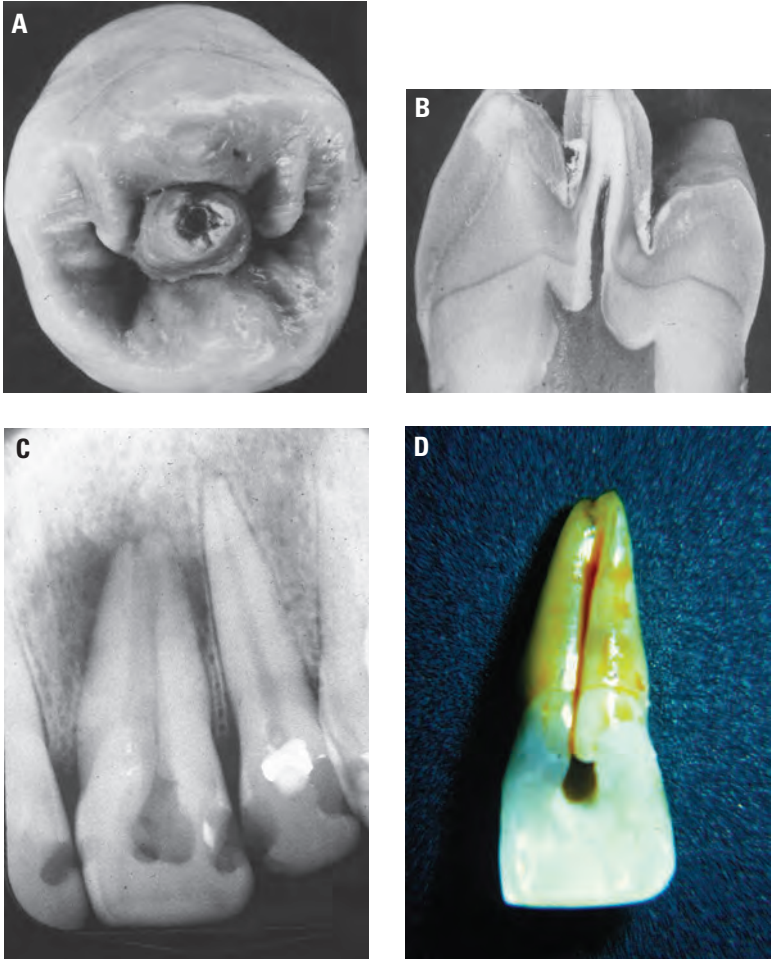


Figure 1-30. **A** and **B**, Dens evaginatus. **A**, Volcanic appearance of extra cusp. Direct access of bacteria to the pulp is possible. Courtesy of O. Carlson. **B**, Extension of pulp into evaginated defect. Courtesy of M.E. Palmer. **C** and **D**, Palatogingival groove, also known as radicular lingual groove. **C**, Bacteria had ready access down the groove to the apex to infect the pulp. **D**, Extracted tooth showing the length and depth of the lingual groove. Courtesy of D.S. August.

previously stated, root-end **condensing osteitis** indicates chronic pulpitis, not to be confused with the early stages of cemental dysplasia (**osteofibrosis**). Huge, circular alveolar lesions spell out chronic apical periodontitis, or the “granuloma” of asymptomatic apical periodontitis, or an apical cyst (Figure 1-31).



Figure 1-31. Unicystic ameloblastoma. This solitary lesion has displaced teeth much as an apical cyst would do. Courtesy of R.J. Melrose.

There are presently two methods that produce a radiographic image from x-ray exposure: the traditional intraoral film method and the so-called direct digital radiology. Both produce radiographic images of archival quality. In the first, unexposed film is placed in the mouth, sensitized by penetrating x-rays, and then chemically processed in the dark. In the second, a solid-state sensor is placed in the mouth and likewise sensitized by standard x-rays, but it is processed in a computer, from which it can be visualized on a monitor, stored, and printed.

In some offices, the traditional method may have a financial advantage, if only the cost of film, developing solutions, and a countertop developing hood (Figure 1-32) are considered. On the other hand, if one considers the staff time involved in developing and mounting films, the savings may disappear.

Completely portable direct digital radiography involves an intraoral sensor attached by a cord to an interface board (PC Capture Card) that plugs into the USB port of a laptop computer, into which the software program has been installed by CD-ROM. Direct digital radiography has a number of advantages. Images can be grossly enlarged on the screen to be viewed by both the dentist and patient, contrast can be changed to improve quality, and if a hard-copy is required the images can be printed from the computer, multiple times if necessary. Films are stored in the computer, not in file drawers. Another claimed advantage is less exposure time to radiation, a health improvement. The sensor can be left in the mouth while the



Figure 1-32. Countertop developing hood (Dentsply/Rinn). Using a rapid developer and fixer, films may be developed in seconds right in the operator.

image is reviewed on the monitor. If not satisfactory, the sensor may be moved and a new shot taken. There also is a savings in time over processing and mounting films. Moreover, the images can be shared by e-mail with a colleague or an insurance company.

Direct digital radiography began in France in 1982 with Dr. Francis Mouyen as the progenitor. Today, the three major competitors in the field are the original French **Trophy RVG** Ultimate Imaging, marketed in North America by Kodak, the **DEXIS 6** systems, developed in Germany and marketed in North America by ProVision Dental Systems, and **Schick Digital Radiography**, marketed in North America by Patterson Dental Supply (Figure 1-33).

Pulp Vitality Testing

Currently, there are two methods of testing for the vitality of the tooth pulp: **thermal** testing with heat or cold, and **electric** testing. Some swear by one method or the other. Both methods have their place and are often complementary to each other.

Thermal Pulp Testing

Cold testing can sometimes be made with a blast of cold air or with cold or ice water, with a stick of ice frozen in an anesthetic carpule, with a spray of ethyl chloride or **Endo-Ice**/tetrafluoroethane (Hygenic/Coltene/Whaledent; Figure 1-34) on a cotton swab or, better yet, a “stick” of CO₂ dry ice. The Endo-Ice swab is probably the easiest to use and ranks just below the CO₂ stick for efficacy. As noted



Figure 1-33. Completely portable direct digital radiography. **A**, PC Capture Card attached to an intraoral sensor. **B**, Capture card plugs into the USB port of the laptop computer, into which a software program has been installed from a CD-ROM. The image on the screen is the last scanned endodontic procedure. Total input is stored in the computer, where it may be modified, printed, retrieved at any time, enlarged on the screen and shown to the patient, and transmitted to the referring dentist or insurance company. **C**, Variety of CCD and CMOS Solid State Sensors.



Figure 1-34. Endo-Ice (tetrafluoroethane) (Hygenic/Coltene) evaporates so rapidly it provides a very cold application for testing the vitality of pulps. Sprayed on a cotton pellet until frosted, it is applied immediately to the suspected tooth.

previously, cold is best used to determine reversible from irreversible pulpitis or necrosis. When using the ice stick, one must be aware that the ice water may drip on other teeth, giving a false reading.

Hot testing is best performed with a ball of hot **baseplate** gutta-percha placed on the moistened tooth. Hot water can also be used and is best in testing porcelain-fused-to-metal crowns. Again, the tested tooth must be isolated. Remember, if an aching tooth is heat tested, one must be ready to immediately counteract the pain response with cold water. Thermal testing is necessary for testing any crowned tooth.

Electric Pulp Testing

There are a number of electric pulp testers on the market, all similar in action but with variation in price. The best known are **Vitality**

Scanner and **Endoanalyzer** (Sybron-Analytic) and **Digitest** and **Gentle Pulse** (Parkell Co.; Figure 1-35). All have lip electrodes to complete the circuit. For comparison, more than the suspected tooth should be tested. Records should be kept of the number at which certain teeth respond so that comparisons can be made later. Confused or malin-



Figure 1-35. Electric pulp testers. All have lip electrodes to complete the circuit. Both Vitality Scanner and Endoanalyzer (Sybron-Analytic) and Digitest Vitality Tester (Parkell Co.) function in the presence of NaOCl, blood, and pus. **A**, Vitality Scanner and Endoanalyzer. **B**, Digitest Vitality Tester with digital readout. **C**, Gentle Pulse (Parkell Co.) is an earlier model and is less expensive. *Continued on next page.*



Figure 1-35. Continued. C, Gentle Pulse analog pulp tester.

gering patients can be fooled by purposely not applying the discharge and questioning them about their reaction to the “electricity.”

In using the testers, the tooth must first be dried; toothpaste, as an electrical conductor, is then placed on the tip of the tester. False-negatives and false-positives may occur, so cross-checking with thermal testing and radiography is *de rigueur*.

FINAL NOTES

The various pulpal and periapical conditions that must be diagnosed and treated have been reviewed, along with the methods and devices used to establish a diagnosis. If one requires a more in-depth discussion of these matters, one is referred to *Ingle's Endodontics* by Ingle, Bakland, and Baumgartner, published by PMPH–USA Publishers, Shelton, CT.

REFERENCES

1. Lipton JA, Ship JA, Larach-Robinson D. Estimated prevalence and distribution of reported orofacial pain in the United States. *J Am Dent Assoc* 1993;124:115.

Rhinosinusitis and Endodontic Disease

Roderick W. Tataryn, D.D.S.

MAXILLARY RHINOSINUSITIS AS A SOURCE OF DENTAL PAIN

The most common **nonodontogenic** cause of dental pain arises from the maxillary sinus and associated pain-sensitive nasal mucosa.¹ Maxillary rhinosinusitis therefore deserves important consideration when one is developing a differential diagnosis for maxillary discomfort. Despite its prevalence, differentiating pain caused by rhinosinusitis from pain of pulpal origin can be one of the more difficult diagnostic challenges faced by the clinician. Results of a misdiagnosis in this area will lead to mistaken endodontic therapy, periapical surgery, and even multiple tooth extractions, with no effective pain relief for the patient.

To effectively distinguish rhinosinusitis from toothache, it is imperative that clinicians understand the anatomy and functions of the paranasal sinuses, both in healthy and diseased states, and to recognize the signs and symptoms associated with sinus inflammation. However, the diagnosis and treatment of a sinus condition is best left to an otorhinolaryngologist (ear, nose, and throat physician, or ENT).

Nevertheless, it is essential that endodontists have a thorough understanding of this system and its potential disease processes to effectively diagnose and treat or rule out pain of odontogenic origin, and to make an appropriate ENT referral if rhinosinusitis is suspected. Knowledge of these structures is also crucial to understanding the effects of periapical infection on the adjacent sinus tissues and the role of endodontic disease in acute and chronic rhinosinusitis.

PARANASAL SINUSES: ANATOMY AND FUNCTION

Sinuses are hollow air spaces or cavities located within the skull surrounding the nasal cavity. There are four pairs of paranasal sinuses: the maxillary, the frontal, the ethmoidal, and the sphenoidal (Figure 2-1). The sinuses are lined with a membrane of ciliated and non-ciliated pseudostratified columnar epithelium that is continuous with the nasal cavities. The columnar cells are interspersed with mucus-producing goblet cells that constantly produce a thin layer of mucinous fluid. The mucus is rich with immune cells, antibodies, and antibacterial proteins that serve as immune defense and air filtration by trapping and filtering particles such as dust, spores, and bacteria.

The cilia beat in a coordinated, rhythmic wavelike pattern moving the mucus toward a small opening, the ostium, into the nasal cavity, then on to the throat, where it is swallowed and finally dissolved by digestive acids. In addition to air filtration, the paranasal sinuses serve to humidify and warm inspired air, and are also important in giving the voice resonance, defining tonal quality, and voice amplification as well as reducing the weight of the skull.²

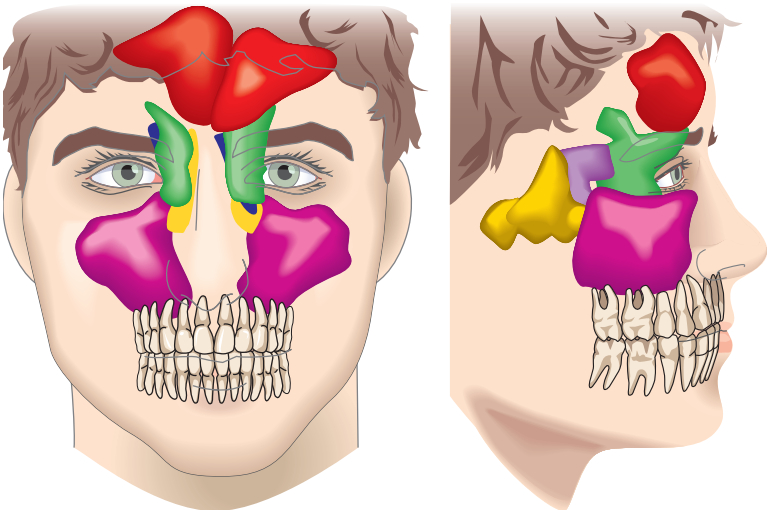


Figure 2-1. The paranasal sinuses. Maxillary sinuses (*red*), frontal sinuses (*orange*), anterior ethmoid sinuses (*green*), posterior ethmoid sinuses (*purple*), and sphenoid sinus (*yellow*).

In this abbreviated treatise, we will deal solely with the maxillary sinus, the only sinus in close proximity to the nasal structures and hence prone to “disease” originating from apical infections and inflammation. The maxillary sinus is the largest of the paranasal sinuses and most prone to infection. Also known as the antrum, it is somewhat pyramidal in shape, with the lateral wall of the nasal cavity forming the base and the apex extending into the zygoma. The floor of the maxillary sinus is the maxillary alveolar and palatine process, the roof is the floor of the orbit. The posterior wall is adjacent to the infratemporal fossa.

The ostium is located approximately two thirds up the medial sinus wall and is a small (1 to 3 mm diameter) passageway into the nasal cavity. The ostium is a highly innervated structure and is extremely sensitive when inflamed or stimulated during sinus-related pain episodes.³ Although the mucosa of the sinuses is rather insensitive to pain, the region surrounding the ostia is the most sensitive of any of the nasal structures.⁴

RHINOSINUSITIS: ETIOLOGY, EPIDEMIOLOGY, SYMPTOMS, AND TREATMENT

The sinus openings or ostia are the focal point for sinus disease. Blockage of the ostia initiates a cycle of events leading to rhinosinusitis. Obstruction is commonly caused by inflammatory edema of the nasal mucosa usually in response to a virus, allergen, or bacteria. Blockage can also occur from an anatomic obstruction such as septal deviation or polyp, or a foreign body obstruction such as dried mucus. When blockage occurs, mucous secretions, normally expelled through the ostium, accumulate within the sinus, where they stagnate and thicken. The lack of sinus ventilation plus stagnation results in a lowered oxygen tension and a decrease in pH, providing an excellent environment for bacterial pathogens to colonize.⁵ As the condition progresses, the cilia and the epithelium become damaged, preventing fluid movement. Further mucosal thickening creates more severe obstruction.

The condition of sinusitis has recently been redefined as rhinosinusitis due to the contiguous lining membrane of the paranasal and nasal cavities.⁶⁻⁷ Rhinosinusitis is the single **most common chronic**

medical condition in the United States, affecting approximately 16% of the population and accounting for up to 5% of all visits to primary care physicians.⁸ Rhinosinusitis is more prevalent in patients with congenital or acquired immunodeficiency, asthma, or allergic rhinitis. Symptoms may include nasal congestion, facial pain and pressure, purulent rhinorrhea, cough, headache, fever and fatigue, and pain in the maxillary posterior teeth.⁹ In a prospective study of 247 men, Williams et al. found that **the symptom with the highest specificity for maxillary rhinosinusitis was a maxillary “toothache” at 93%.**¹⁰

A diagnosis of rhinosinusitis is generally made through clinical examination and is based on the patient’s history and physical signs and symptoms.¹⁰ In recurrent acute or chronic cases, endoscopy and computed tomography (CT) scanning will be helpful in the diagnosis.⁶ Coronal viewed sections of a CT scan can reveal either ostial patency or blockage, soft tissue changes, or any retained fluids that may be indicative of sinus disease (Figure 2-2).

Mistakes can be made in reading a CT scan. More than 40% of asymptomatic patients and 87% of patients with common colds show sinus abnormalities on sinus CT.¹¹⁻¹² It is also important to note that sinus inflammation and symptoms can occur in the absence of CT findings. Thus the physician’s overall clinical impression is superior to any single physical or radiological finding.¹³ Treatment

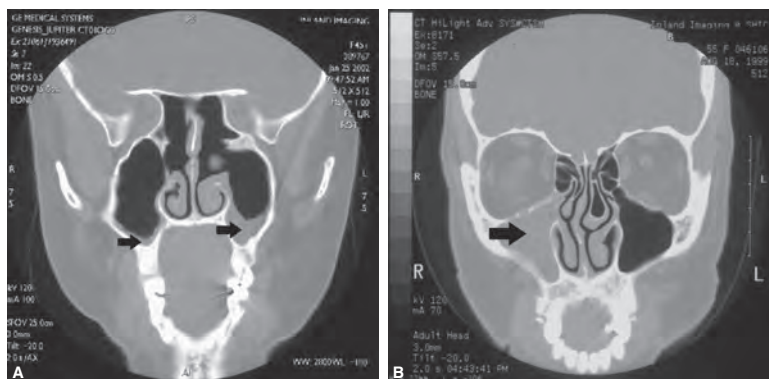


Figure 2-2. Maxillary rhinosinusitis. **A**, Coronal CT image of mucosal thickening and fluid accumulation in the maxillary sinuses (arrows). **B**, Coronal CT image of a completely obstructed right maxillary sinus (arrow).

of sinus disease is centered on re-establishing and then maintaining adequate drainage.

Rhinosinusitis is classified as acute, subacute, and recurrent acute or chronic according to the temporal course of the disease.^{6,9} Acute rhinosinusitis by definition lasts less than 4 weeks. Four or more episodes of acute rhinosinusitis within one year are defined as recurrent acute rhinosinusitis. Subacute rhinosinusitis is continuation of an acute form lasting between 4 and 12 weeks. Persistent signs and symptoms that continue unresolved for more than 12 weeks are classified as chronic rhinosinusitis.

Acute rhinosinusitis usually has a sudden onset, usually with severe symptoms, but 40% to 50% of patients will recover spontaneously and the remainder will respond fairly well to antibiotic or adjunctive treatments. The predominant bacterial flora isolated in acute rhinosinusitis includes *Streptococcus pneumoniae*, *Haemophilus influenzae*, and *Moraxella catarrhalis*. Amoxicillin or trimethoprim-sulfamethoxazole are the preferred antibiotics as a first-line treatment, although the role of antibiotics in **uncomplicated rhinosinusitis** has been questioned because of concern of their overuse.^{5,14-15}

The bacterial species isolated from acute rhinosinusitis has not changed in the last 50 years. On the other hand, antibiotic resistance in these organisms is a growing problem due to a significant increase in the production of β -lactamase. It has been estimated that up to 40% of *H. influenzae* strains and more than 90% of *M. catarrhalis* isolates are now resistant to amoxicillin.¹⁶

Chronic rhinosinusitis usually has less severe symptoms, yet is more difficult to resolve. Because symptoms are poorly localized and mild, this condition may be difficult to recognize. The bacterial flora of chronic rhinosinusitis tends to be a more mixed infection with *Staphylococcus aureus* and anaerobes being the more frequent isolates.¹⁷ Current treatment includes the use of broader spectrum antibiotics such as amoxicillin-clavulanate, clindamycin, or the combination of metronidazole and penicillin.¹⁸ Other adjunctive treatments include the use of daily steroid nasal sprays, decongestants, and saline irrigation. If antibiotic therapy and adjunctive treatments prove ineffective, then endoscopic nasal/sinus surgery is currently the recommended procedure to surgically remove any diseased tissue or blockage and open the natural ostium in an attempt to re-establish drainage.¹⁸

Referral of Maxillary Sinus Pain to the Teeth

The referral of pain from the maxillary sinus to the maxillary dentition is primarily due to the close anatomic relationship between the floor of the maxillary sinus and the roots of the posterior maxillary teeth. In a highly pneumatized antrum, the sinus floor will extend between adjacent teeth and between individual roots of the maxillary molars (Figure 2-3). In a study of adults, Eberhardt found the average thickness of the bony partition between the apices of the maxillary second molar teeth and the antral mucosa to be only 0.83 mm.¹⁹ Occasionally, no bony partition exists at all, with the root apices and the sinus separated only by the mucosal lining.³ With such an intimate relationship, increased sinus pressure during sinusitis can produce the sensation of pain and pressure in the maxillary alveolar process and teeth. Percussion of these proximate teeth produces acute tenderness due to the direct shock pressure to the inflamed sinus and the sensitive ostium. Often for some patients, the most sensitive trigger for their sinus pain is by manipulation of the maxillary posterior teeth, leading them to believe with near certainty that their teeth are the source of their pain.

Heterotropic or neurologically referred pain can also be responsible for a patient mistaking rhinosinusitis for odontalgia.²⁰ It has been demonstrated that stimulation of the maxillary sinus ostium



Figure 2-3. Radiograph of a highly pneumatized left maxillary sinus in close approximation to the roots of the maxillary posterior teeth. The dense cortical bone defines the inferior border of the antrum (*arrows*).

can induce dental pain in the maxillary posterior teeth.²¹⁻²³ It has also been demonstrated that a topical anesthetic applied to the maxillary sinus ostium can relieve perceived dental pain in the case of referred sinus pain.²⁴ However, anesthetic nerve blocks may not be reliable in differentiating maxillary rhinosinusitis from odontalgia due to mutual dental and antral innervations. Sensory innervation of the maxillary sinus is via the maxillary division of the trigeminal nerve with branches coming from the anterior, middle, and posterior superior alveolar nerves, the infraorbital nerve, and the anterior palatine nerve.⁴ These nerves travel along the floor of the maxillary sinus innervating the related teeth.²⁵

Differences Between Symptoms of Odontalgia and Sinus Pain

Proper diagnosis always starts with a complete medical and dental history. An important question to ask the patient during the endodontic exam as well as on the medical history form is, “Do you have chronic allergies, a current cold, congestion, or nasal drainage, or a history of sinus infections or problems?” Although many patients suffering with maxillary rhinosinusitis may perceive dental pain, they usually complain of a dull aching pain that is difficult to pinpoint or localize to a single tooth. In addition, the patients often feel pressure in their cheeks and below their eyes, which usually can be reproduced by external palpation of the cheeks. Positional changes can cause increased pain due to the movement of mucosal fluid over the sensitive sinus ostium.²⁶

Pain usually increases when patients are lying down, usually on one side more than the other, or if they bend over and place their head below their knees. In addition, patients with acute rhinosinusitis can have potentially severe symptoms, but will experience sudden and total relief if the sinus pressure is temporarily alleviated.

In contrast, pain of pulpal origin is more easily localized, unchanged with variations in position, and rarely intermittent in intensity. In cases of maxillary rhinosinusitis, all teeth that are proximate to the sinus floor often test positive to percussion, whereas with odontalgia usually only the offending teeth are percussion-sensitive.

The key diagnostic test in differentiating maxillary sinus etiology from pain of pulpal origin is the pulp vitality test. If the tooth in question responds to an electric pulp test (EPT) or ice, then pulpal etiology is effectively ruled out. Regardless of percussion sensitivity

or the patient's complaint of spontaneous dental pain, endodontic therapy is not indicated, as removal of a healthy pulp will surely not provide pain relief.

Although periapical radiographs are crucial to a thorough endodontic examination, the clinician must be careful not to establish the entire diagnosis on radiographic interpretation alone, particularly when evaluating the posterior maxillary area. Variation in bone density, the presence of the maxillary sinus and its bony septa, as well as the zygomatic and palatal processes, can impede important periradicular details necessary for accurate interpretation. Ambiguous radiographic conclusions are often the result, and multiple radiographic angles are highly recommended in combination with a thorough clinical examination.

PERIAPICAL MUCOSITIS

Maxillary posterior teeth with apical periodontitis can produce a localized mucosal thickening in the adjacent sinus mucosa. This localized mucosal response to apical inflammation has been termed **periapical mucositis**.²⁷ Periapical mucositis is the inflammation or swelling of the **sinus membrane** secondary to periapical inflammation, and will appear as a radiopaque soft-tissue dome directly above the offending tooth.²⁸

Mucositis is usually asymptomatic and can be expected to resolve following endodontic treatment. It is important not to confuse periapical mucositis with a mucous retention cyst or a mucocele on the floor of the maxillary sinus. Mucous retention cysts are seen in approximately 10% of the routine sinus CTs and are considered incidental findings. They do not require surgical intervention by the physician unless they are blocking the ostium.⁹ Careful pulp testing is imperative in distinguishing a routine mucosal abnormality on the floor of the sinus from a mucositis secondary to periapical inflammation.

Occasionally, apical periodontitis will not penetrate the antral floor but rather will displace the periosteum, which in turn will deposit a thin layer of new bone on the periphery of the disease process. This has been termed **periapical osteoperiostitis** or "halo" shadow and will resolve following endodontic treatment of the offending tooth (Figure 2-5).²

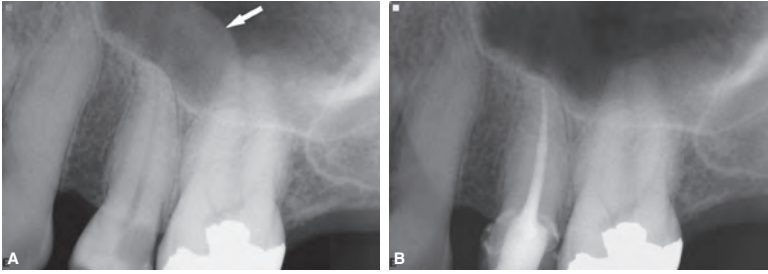


Figure 2-4. Periapical mucositis. **A**, Radiograph of a mucositis lesion, shown as a “dome” in the left maxillary sinus apical to tooth #13 (arrow). **B**, Follow-up radiograph taken 6 weeks after root canal treatment showing full resolution of the mucositis.

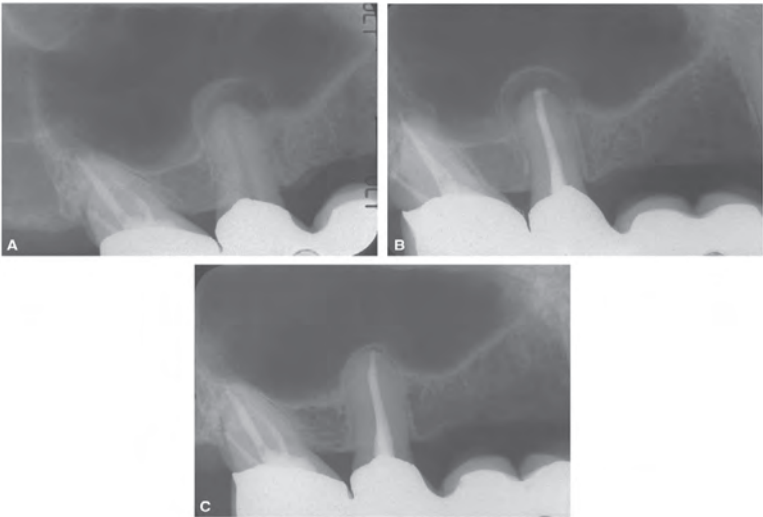


Figure 2-5. Periapical osteoperiostitis (“halo” lesion). **A**, Radiograph showing expansive bone formation resulting from displaced periosteum by the periapical infection from pulp necrosis in tooth #4. **B**, Immediate postoperative radiograph of RCT in tooth #4. **C**, Follow-up radiograph taken 1 year after RCT demonstrating full resolution of the halo lesion.

MAXILLARY SINUSITIS OF DENTAL ORIGIN

The link between dental and sinus pathosis is widely recognized in both the dental and the medical literature.^{28–39} This condition was first referred to by Bauer in 1943 as maxillary sinusitis of dental origin (MSDO) and occurs when a dental infection extends directly through the mucosal floor of the antrum, causing a secondary maxillary sinus infection.

The pathological extension of dental disease into the maxillary sinus has since been well documented. Abrahams et al. have reported that **apical infections of maxillary posterior teeth show maxillary sinus pathology in 60% of cases**, whereas Matilla found sinus mucosal hyperplasia present in approximately 80% of teeth with periapical osteitis.^{36–37} Sinus inflammation in response to dental infection is usually localized to the floor of the sinus; however, if obstruction of the ostium occurs, it can lead to bacterial colonization and a sinus infection. The reported frequency of MSDO varies considerably, between 4.6% and 47% of all sinus infections.⁴⁰ This wide variation may be due to the difference in criteria and definitions as well as the inherent difficulty in establishing an exact causal relationship in maxillary sinusitis.⁴¹

It is generally accepted that dental infections account for approximately 10% to 15% of the cases of acute maxillary sinusitis.^{9,40} However, this phenomenon may be much greater in chronic cases. In a study of 198 patients with 244 cases of chronic bacterial maxillary sinusitis, Melen et al. **found dental etiology in 40.6% of the cases.**³⁸ It is also well documented that the predominant anaerobic bacterial isolates frequently found in chronic sinusitis are *Prevotella* spp., *Porphyromonas* spp., *Fusobacterium nucleatum*, and *Peptostreptococcus* spp., the same bacterial species **found in endodontic infections.**^{17,42–44}

If a sinus infection is secondary to a dental infection, sinus healing cannot occur unless the offending tooth is endodontically treated or removed. Dental literature provides numerous case reports of the resolution of maxillary sinusitis following endodontic treatment or extraction.^{45–47} The challenge for patients with MSDO is that they often have primary symptoms of maxillary sinusitis yet are rarely able to localize pain to a specific tooth or sense any dental pain. These patients typically first seek care from their physicians

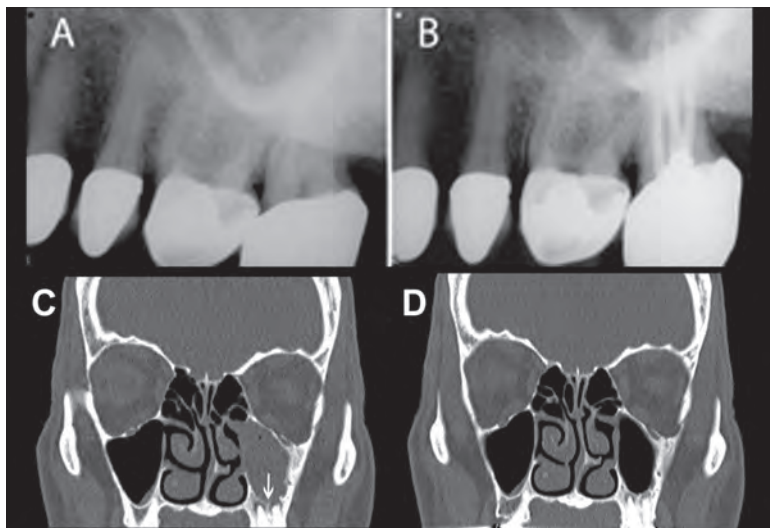


Figure 2-6. Maxillary sinusitis of dental origin. **A**, Preoperative periapical radiograph. Note the limited diagnostic value due to zygomatic and palatine processes obscuring the molar root apices. Periapical infection is not evident. **B**, Endodontic treatment completed on tooth #15. **C**, Coronal CT scan reveals periapical abscesses on both the buccal and palatal roots of tooth #15, and the extension of the dental infection into the floor of the left maxillary sinus via the broken cortical bone (*arrow*). Note the fully obstructed left maxillary sinus indicating an advanced sinus infection. **D**, Ten-week follow-up after RCT. CT scan showing full resolution of the sinus disease. All sinus symptoms have resolved, and the patient is free of antibiotics and adjunctive sinus regimens for the first time in 2 years.

or ENTs, who may diagnose and treat the condition as a primary sinus infection.

Dental infections are easily overlooked during routine ENT examinations.⁴⁸ Regrettably, some patients have undergone multiple antibiotic regimens and even sinus surgery before a dental condition is finally diagnosed as the primary etiology (Figure 2-6).⁴⁹

This unfortunately occurs despite strong suggestions in the medical literature for careful oral examination, including dental radiographs, in all patients with acute or chronic maxillary sinusitis.^{38,44,48,50} The importance of properly diagnosing and treating MSDO is heightened with reports in the literature of dental infections

spreading rapidly through the maxillary sinus, causing orbital cellulitis, blindness, meningitis, subdural empyema, brain abscess, and life-threatening cavernous sinus thrombosis.⁵⁰⁻⁵²

More recent medical literature on sinus management have emphasized the need to rule out and treat any possible dental etiology for both acute and chronic rhinosinusitis.^{16,41} However, to date none of the current medical guidelines for management of acute or chronic rhinosinusitis include the recommendation for referral to a dentist or an endodontist to rule out a possible dental etiology.^{18, 53-55} Improved communication between the endodontic and ENT communities is essential in providing improved patient care and resolving a greater number of cases of MSDO.

When diagnosing a possible dental etiology for a patient with maxillary rhinosinusitis, the endodontist must seek out any pulpal necrosis and periapical disease and must carefully evaluate all previous endodontic treatments for failure in the suspected quadrant. For a periapical infection to occur, bacteria must be present within the root canal system.⁵⁶ A vital pulp, whether healthy or inflamed, will not contribute significantly to any periapical or secondary sinus infection. Thus for MSDO to occur, the causative tooth must either have necrotic pulp tissue or have a failing endodontic treatment.

Properly angled periapical radiographs can offer helpful information regarding sinus pathosis. A generalized inflammatory reaction of the sinus mucosa will result in mucosal hyperplasia, which may be seen as a radiopaque band of tissue following the contours of the sinus floor.⁵⁷ Periapical and panoramic radiographs may also reveal the presence of significant mucosal fluid build-up in the sinus floor or complete opacification, which can be confirmed with a corresponding CT scan and through communication with the ENT physician (Figure 2-7).

If the sinus infection is determined to be secondary to periapical infection, endodontic therapy or extraction should resolve the problem. However, concomitant management of the associated rhinosinusitis may be necessary to ensure complete resolution of the infection and to prevent any complications.⁴³ The resolution should be confirmed with a follow-up radiograph or a CT scan and a positive patient report. The increased availability and resolution of digital CT imaging should be of great future benefit in the diagnosis of MSDO as well as for post-treatment follow-up confirming the resolution of the mucosal inflammation.

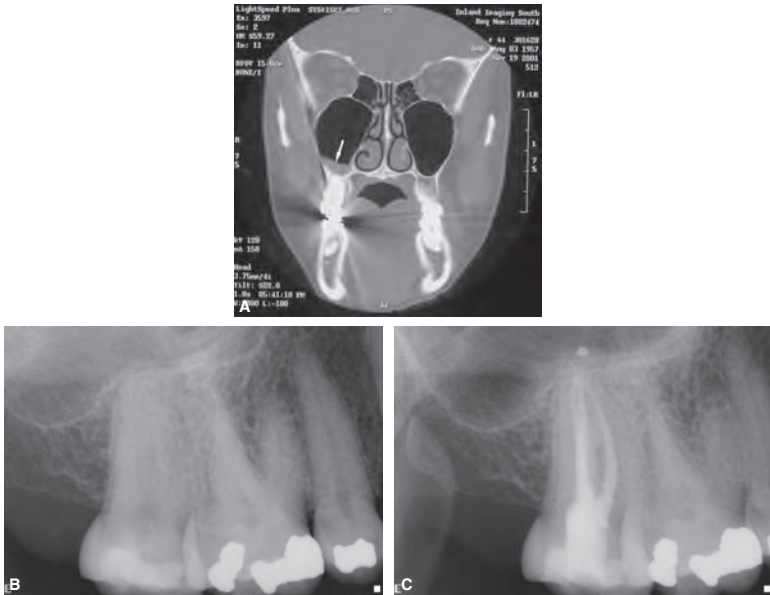


Figure 2-7. Maxillary sinusitis of dental origin (MSDO). **A**, Coronal CT image of a patient diagnosed with right maxillary sinusitis, (*arrow*). **B**, Periapical radiograph shows no evidence of periapical radiolucency; however, the right maxillary sinus appears clouded. Clinical tests reveal the pulp in tooth #2 to be necrotic. **C**, Follow-up radiograph taken 4 weeks after RCT in tooth #2 with patient reporting resolution of sinusitis symptoms. The sinus appears less clouded than preoperatively on the periapical radiograph.

CANCER AND THE MAXILLARY SINUS

Cancer of the maxillary sinus is not unusual. Because of the proximity of the sinus to the maxillary posterior teeth, confusion often reigns in a dentist's attempt to diagnose an unusual pain and other symptoms in the area. Errors are easily made, and teeth are either extracted or root canal treatment is instituted, in haste or desperation. The same applies to misdiagnosis in the mandible of benign lesions such as ameloblastomas.

A recent report out of Switzerland by Bornstein et al. presents an unusual case of Ewing's sarcoma in a 19-year-old female.⁵⁸ The first symptoms were loss of sensitivity and acute pain in the premolar and first molar area of the maxilla. The teeth did not react to



Figure 2-8. The swelling on the palate (*arrow*) appeared almost unchanged after incision and drainage. In addition, a soft buccal swelling in the area of teeth #14 through #16 was visible. Bornstein MM et al. J Endod 2008;34(12):1549–53. Reproduced with permission.

pulp testing so the referring dentist opened the three teeth and applied an “endodontic medication.” Because of the depth of palatal probing, the dentist suspected an acute perio/endo lesion. The pain decreased, but 6 weeks later it returned and a swelling in the palate had developed. Because of these unusual circumstances, the dentist referred the patient to The School of Dental Medicine in Bern. The palatal swelling (Figure 2-8) was confirmed and a cone-beam computed tomography (CBCT) of the region revealed a lesion that filled the right maxillary sinus fairly well (Figure 2-9). The involved teeth were root filled and the lesion was surgically exposed. Granulomatous tissue was curetted from the sinus (Figure 2-10) as well as “some orange foreign material” that proved on biopsy to be Ewing’s sarcoma (Figure 2-11).

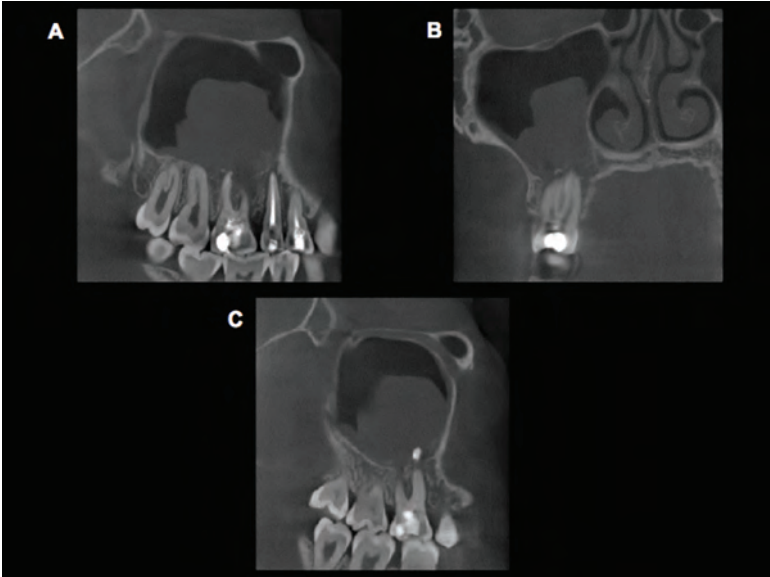


Figure 2-9. **A**, The sagittal CBCT plane exhibited a proliferation of soft tissue in the right maxillary sinus, ranging from the first premolar region back to the second molar area. The mesiobuccal and to a lesser extent the distobuccal roots of the first molar exhibited resorptive signs, and the floor of the sinus was not visible in that area. **B**, The coronal CBCT section showed a proliferation of soft tissue in the right maxillary sinus, occupying the basal third of the sinus. **C**, Another sagittal CBCT section shows a foreign, radiopaque material present at the tip of the mesiobuccal root. Bornstein MM et al. J Endod 2008;34(12): 1549–53. Reproduced with permission.

This not a typical case but it does prove the thesis that we tend to think along single tracks. We automatically think “apical granuloma or cyst,” the lesions most common in our mental vocabulary. But one must learn to think beyond the old saying, “When you hear the pounding of hoofs, think not of zebras but think of horses.” Unfortunately, there are “zebras” out there as well as horses, like Ewing’s sarcoma or ameloblastoma.

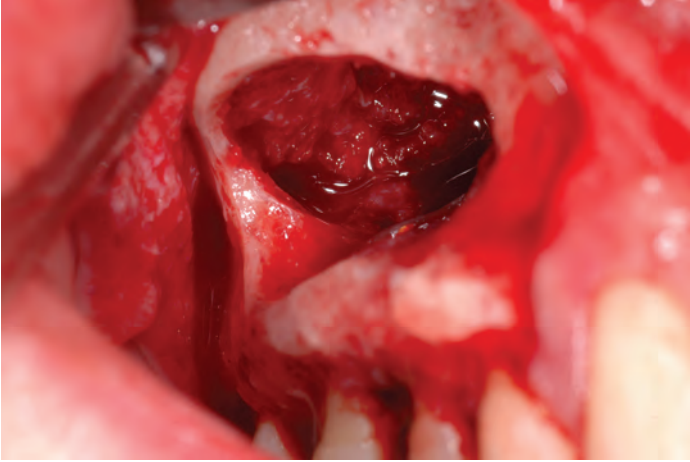


Figure 2-10. During the surgery, the buccal bone of teeth #14 through #16 appeared to be intact, and no resorption was visible. Granulomatous tissue was present in the area of the buccal swelling and was biopsied. The root tips of teeth #15 and #16 were resected, and the sinus was opened. Also here, granulomatous tissue was present with some orange foreign material, which was also biopsied. Bornstein MM et al. *J Endod* 2008;34(12):1549–53. Reproduced with permission.

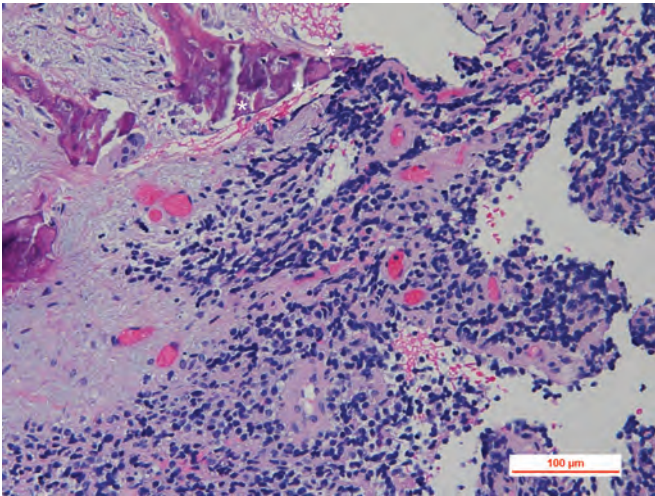


Figure 2-11. The tumor had an aggressive behavior, with infiltration and resorption of adjacent bone. H&E stain. Bornstein MM et al. *J Endod* 2008;34(12): 1549–53. Reproduced with permission.

REFERENCES

1. Ingle JI, Bakland LK. Endodontics. 5th ed. Hamilton, ON: BC Decker; 2002.
2. AAOMS Surgical Update. Maxillary sinuses, guidelines for diagnosis and treatment. *Am Assoc Oral Maxillofac Surg* 1986;2:4-6.
3. Alberti PW. Applied surgical anatomy of the maxillary sinus. *Otolaryngol Clin North Am* 1976;9:3-20.
4. Stammberger H, Wolf G. Headaches and sinus disease: the endoscopic approach. *Ann Otol Rhinol Laryngol* 1988;(Suppl)134:3-23.
5. Fagnan LI. Acute sinusitis: a cost-effective approach to diagnosis and treatment. *Am Fam Physician* 1998;58:1795-806.
6. Osguthorpe JD, Hadley JA. Rhinosinusitis. Current concepts in evaluation and management. *Med Clin North Am* 1999;83:27-41.
7. Hamilos DL, Lanza DC, Kennedy DW. Rhinosinusitis and the revised "sinusitis practice parameters," *J Allergy Clin Immunol* 2005;116:1267-8.
8. Benson V, Marano MA. Current estimates from the National Health Interview Survey 1992. *Vital Health Stat* 1994;189:1-269.
9. Lanza DC, Kennedy DW. Adult rhinosinusitis defined. *Otolaryngol Head Neck Surg* 1997;117:1-7.
10. Williams JW, Simel DL, Roberts L, Samsa GP. Clinical evaluation for sinusitis: making the diagnosis by history and physical evaluation. *Ann Intern Med* 1992;117:705-10.
11. Havas TE, Motbey JA, Gullane PJ. Prevalence of incidental abnormalities on computed tomographic scans of the paranasal sinuses. *Arch Otolaryngol Head Neck Surg* 1988;114:856-9.
12. Gwaltney JM, Phillips CD, Miller RD, Piker DK. Computed tomography study of the common cold. *N Engl J Med* 1994;330:25-30.
13. Benninger MS, Ferguson BJ, Hadley JA, et al. Adult chronic rhinosinusitis: definitions, diagnosis, epidemiology, and pathophysiology. *Otolaryngol Head Neck Surg* 2003;129(3 Suppl):S1-32.
14. Poole MD. A focus on acute sinusitis in adults: changes in disease management. *Am J Med* 1999;106:38S-47S.
15. Snow V, Mottur-Pilson C, Hickner JM. Principles of appropriate antibiotic use for acute sinusitis in adults. *Ann Intern Med* 2001;134:495-7.
16. Brook I. Microbiology and antimicrobial management of sinusitis. *J Laryngol Otol* 2005;119:251-8.
17. Brook I. Bacteriology of chronic maxillary sinusitis in adults. *Ann Otol Rhinol Laryngol* 1989;98:426-8.
18. Slavin RG, Spector SL, Bernstein IL, et al. The diagnosis and management of sinusitis: a practice parameter update. *J Allergy Clin Immunol* 2005;116: S13-47.
19. Eberhardt JA, Torabinejad M, Christiansen EL. A computed tomographic study of the distances between the maxillary sinus floor and the apices of the maxillary posterior teeth. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod* 1992;73:345-6.

20. Okeson JP, Falace DA. Nonodontogenic toothache. *Dent Clin North Am* 1997;41:367-83.
21. Reynolds OE, Hutchins HC, Werner AY, Philbrook FR. Aerodontalgia occurring during oxygen indoctrination in low pressure chamber. *US Naval Med Bull* June 1946;46:845.
22. Dalessio DJ. Wolff's headache and other head pain. 3rd ed. New York: Oxford University Press; 1972.
23. Ballenger JJ. Diseases of the nose, throat and ear. 11th ed. Philadelphia: Lea & Febiger; 1969.
24. Radman WP. The maxillary sinus: revisited by an endodontist. *J Endod* 1983;9:382-3.
25. Wallace JA. Transantral endodontic surgery. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod* 1996;82:80-4.
26. Hauman CHJ, Chandler NP, Tong DC. Endodontic implications of the maxillary sinus: a review. *Int Endod J* 2002;35:127-41.
27. Worth HM, Stoneman DW. Radiographic interpretation of antral mucosal changes due to localized dental infection. *J Can Dent Assoc* 1972;38:111-16.
28. Berry G. Further observations on dental caries as a contributing factor in maxillary sinusitis. *Arch Otol* 1930;11:55.
29. Bauer WH. Maxillary sinusitis of dental origin. *Am J Orthod Oral Surg* 1943;29:133-51.
30. Nenzen B, Welander U. The effect of conservative root canal therapy on local mucosal hyperplasia in the maxillary sinus. *Odontol Revy* 1967;18:295-302.
31. Maloney PL, Doku HC. Maxillary sinusitis of odontogenic origin. *J Can Dent Assoc* 1968;34:591-603.
32. Seldon HS. The interrelationship between the maxillary sinus and endodontics. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod* 1974;38:623-9.
33. Yoshiura K, Ban S, Hijiya T, et al. Analysis of maxillary sinusitis using computed tomography. *Dentomaxillofac Radiol* 1993;22:86-92.
34. Bertrand B, Rombaux P, Eloy P, Reychie H. Sinusitis of dental origin. *Acta Otorhinolaryngol Beig* 1997;51:315.
35. Connor SEJ, Chavda SV, Pahor AL. Computed tomography evidence of dental restoration as aetiological factor for maxillary sinusitis. *J Laryngol Otol* 2000;114:510-13.
36. Abrahams JJ, Glassberg RM. Dental disease: a frequently unrecognized cause of maxillary sinus abnormalities? *Am J Roentgenol* 1996;166:1219-23.
37. Matilla K. Roentgenological investigations of the relation ship between periapical lesions and conditions of the mucous membrane of the maxillary sinuses. *Acta Odontolog Scand* 1965;23:42-6.
38. Melen I, Lindahl L, Andreasson L, Rundcrantz H. Chronic maxillary sinusitis. Definition, diagnosis and relation to dental infections and nasal polyposis. *Acta Otolaryngol* 1986;101:320-7.
39. Mehra P, Murad H. Maxillary sinus disease of odontogenic origin. *Otolaryngol Clin North Am* 2004; 37:347-64.

40. Kretzschmar DP, Kretzschmar JL. Rhinosinusitis: review from a dental perspective. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod* 2003;96:128–35.
41. Legert KG, Zimmerman M, Stierna P. Sinusitis of odontogenic origin: pathophysiological implications of early treatment. *Acta Otolaryngol* 2004;124:655–63.
42. Brook I, Frazier EH, Foote PA. Microbiology of the transition from acute to chronic maxillary sinusitis. *J Med Microbiol* 1996;45:372–5.
43. Brook I. Microbiology of acute and chronic maxillary sinusitis associated with an odontogenic origin. *Laryngoscope* 2005;115:823–5.
44. Brook I, Frazier EH, Gher ME Jr. Microbiology of periapical abscesses and associated maxillary sinusitis. *J Periodontol* 1996;67:608–10.
45. Seldon HS, August DS. Maxillary sinus involvement—an endodontic complication. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod* 1970;30:117–22.
46. Seldon HS. The endo-antral syndrome *J Endod* 1977;3:462–4.
47. Dodd RB, Dodds RN, Hocomb JB. An endodontically induced maxillary sinusitis. *J Endod* 1984;10:504–6.
48. Lindahl L, Melen I, Ekedahl C, Holm SE. Chronic maxillary sinusitis. Differential diagnosis and genesis. *Acta Otolaryngol* 1982;93:147–50.
49. Kulacz R, Fishman G, Levine H. An unsuccessful sinus surgery caused by dental involvement within the floor of the maxillary sinus. *Opt Tech Otolaryngol Head Neck Surg* 2004;15:2–3.
50. Ngeow WC. Orbital cellulitis as a sole symptom of odontogenic infection. *Singap. Med J* 1999;40:101–3.
51. Wagenmann M, Naclerio RM. Complications of sinusitis. *J Allergy Clin Immunol* 1992;90:552–4.
52. Gold RS, Sager E. Pansinusitis, orbital cellulitis, and blindness as sequelae of delayed treatment of dental abscess. *J Oral Surg* 1974;32:40–3.
53. Gwaltney JM Jr, Jones JG, Kennedy DW. Medical management of sinusitis: educational goals and management guidelines. The International Conference on Sinus Disease. *Ann. Otol Rhinol Laryngol Suppl* 1995;167:22–30.
54. Benninger MS, Anon J, Mabry RL. The medical management of rhinosinusitis. *Otolaryngol Head Neck Surg* 1997;117:41–9.
55. Brook I, Gooch WM III, Jenkins SG. Medical management of acute bacterial sinusitis—recommendations of a clinical advisory committee on pediatric and adult sinusitis. *Ann Otol Rhinol Laryngol* 2000;109:1–20.
56. Kakehashi S, Stanley HR, Fitzgerald RJ. The effects of surgical exposures of dental pulps in germ-free and conventional laboratory rats. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod* 1965;20:340–9.
57. Van Dis ML, Miles DA. Disorders of the maxillary sinus. *Dent Clin North Am* 1994;38:155–66.
58. Bornstein MM et al. Loss of pulp sensitivity and pain as the first symptoms of a Ewing's sarcoma in the right maxillary sinus and alveolar process: report of a case. *J Endod* 2008;34(12):1549–53.

Endodontic Treatment Procedures

ROOT CANAL THERAPY

There has long been controversy over the number of appointments it should take to complete a root canal treatment. Years ago, dentists had a terrible reputation for extended treatment time—ten or twelve appointments to complete a single tooth. Over the years, this was moderated until, finally, three appointments became standard: one for examination, one for canal preparation, and one for obturation, that is, unless one kept encountering positive bacteriologic cultures. In that case, one kept medicating until a negative culture was obtained, or finally filled the canal anyway in disgust with the system.

Cases that started with vital pulp extirpation were the lone exception to multiple treatments. It was assumed that if the pulp was vital but inflamed, it was acceptable to fill the canal in a single visit because infection was thought not to be a problem. These patients sometimes complained that the tooth was “sore” for a couple of days, but that the soreness “wore off.” If treatment was done properly, follow-up radiographs usually revealed continued periapical health.

Encouraged by this success, single-visit treatment became popular. Some limited this approach to vital cases. Few dentists treated cases in one appointment that were acutely infected or abscessed.¹⁻² Others were daring and treated virtually every case in one appointment. No one took cultures, assuming the bacteria were being destroyed during cleaning and shaping with sodium hypochlorite. Reported success rates were comforting.³⁻⁶

However, disturbing reports started to appear. Sjogren and colleagues sounded a word of caution.⁷ At a single visit, they cleaned

and obturated 55 single canal teeth with apical periodontitis. **Following cleaning and shaping** with sodium hypochlorite and just **before obturation**, they cultured the canals. Using advanced **anaerobic** techniques, they found that 22 (40%) of the canals tested positive and 33 (60%) of the canals were negative.

Periapical healing was then followed up for **5 years**. Complete healing occurred in 94% of the 33 negative-culture cases. But in the 22 positive-culture cases, “the success rate had fallen to just 68%,” a statistically significant difference. In other words, if a canal is still infected before filling at a single sitting, there may be a 26% greater chance of failure than if the canal is free of bacteria.⁷ But who knows which canal is positive and which canal is negative?

In marked contrast, Figini et al. in Milan in a meta-analysis of twelve articles involving 657 patients comparing single versus multiple visits concluded that, “(n)o detectable difference was found in the effectiveness of root canal treatment in terms of radiologic success between single and multiple visits.”⁸ However, they did note that “(p)atients undergoing a single visit might experience a slightly higher frequency of swelling and analgesic use was significantly more common in the single-visit group.”

Baumgartner et al. found that the last 5 mm of the canal was absolutely teeming with bacteria, over 50 different species.⁹ Siqueira and colleagues recently examined the microbiota of infected root canals and found bacteria penetrating the dentinal tubuli as deeply as 300 microns (Figure 3-1).¹⁰ These are the bacteria that might easily avoid detection and eradication. Gutierrez and colleagues “found bacteria 250 microns deep within tubules **even after root canal instrumentation**.”¹¹ Bacteria harbored in the apical third of dentin could well be the source of refractory periapical infection following acceptable root canal treatment so well described by Barnett and colleagues and demonstrated by Leonardo and colleagues (Figure 3-2).¹²⁻¹⁴

To fill or not to fill? That is the question! Does single-appointment root canal treatment provide enough certainty of success to trust every case to one sitting?

Root canal treatment consists, in major part, of cleaning and shaping the root canal with files and reamers, either hand-driven or motor-driven, disinfecting the canal, and then obturating the canal space. On occasion, endodontists also prepare post space and place posts for the referring dentist. Bleaching discolored teeth

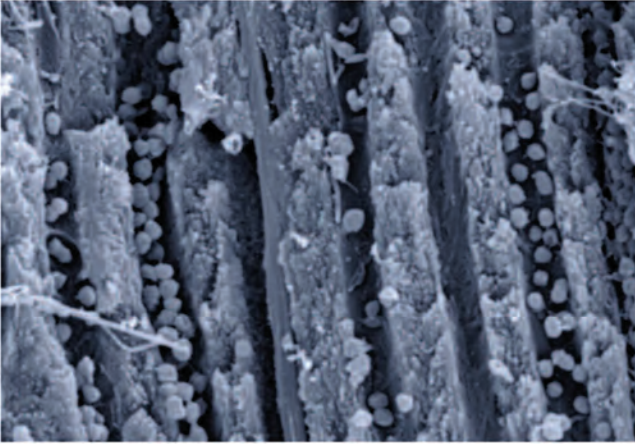


Figure 3-1. Cocci in dentinal tubules approximately 300 microns from the main root canal. Some cells are dividing ($\times 5,000$ original magnification). Reproduced with permission from Siqueira JF et al.¹⁰

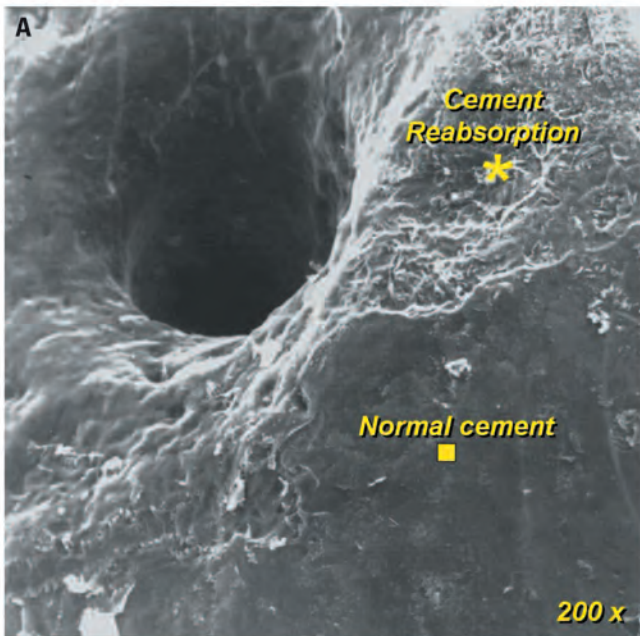


Figure 3-2. A, Root apex with morphologic changes in the apical cementum close to the apical foramen showing areas of intact cementum between areas of resorption. **Continued on next page.**

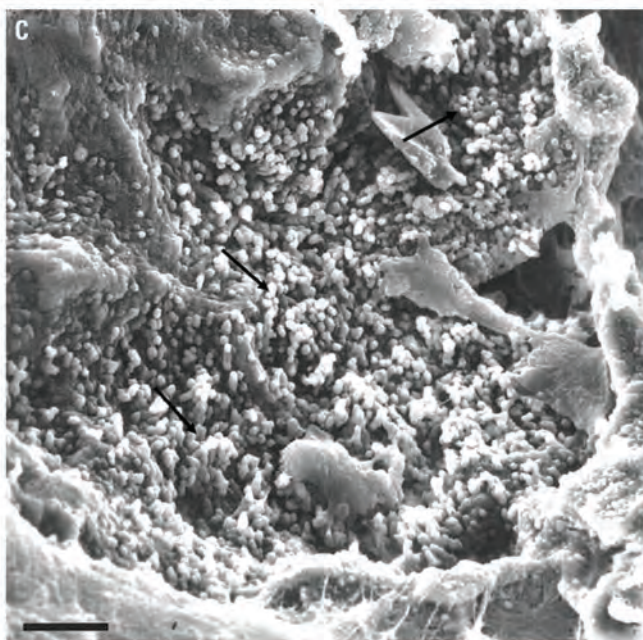
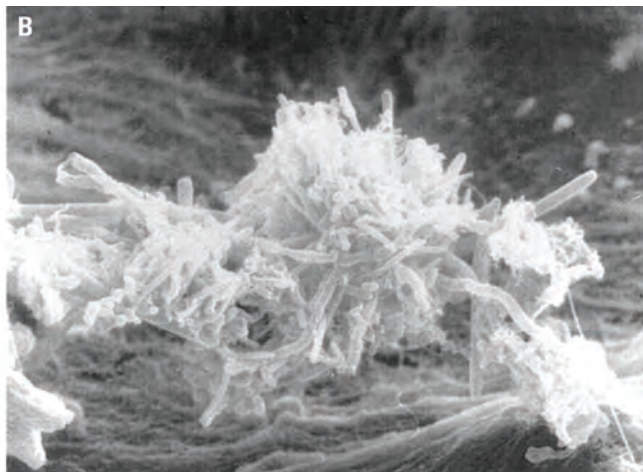


Figure 3-2. Continued. **B**, Apical biofilm on external root surface with the presence of filaments and bacilli. **C** Higher magnification of image **B** showing the presence of cocci forming apical biofilm. **A–C** reproduced with permission from Leonardo MR et al.¹⁴

(Figure 3-3), performing surgery to correct periradicular problems (Figure 3-4), re-implanting traumatically avulsed teeth as well as endosseous implants, treating traumatically damaged teeth, diagnosing and treating intraoral pain, and restoring endodontically treated teeth all fall under the purview of endodontics.

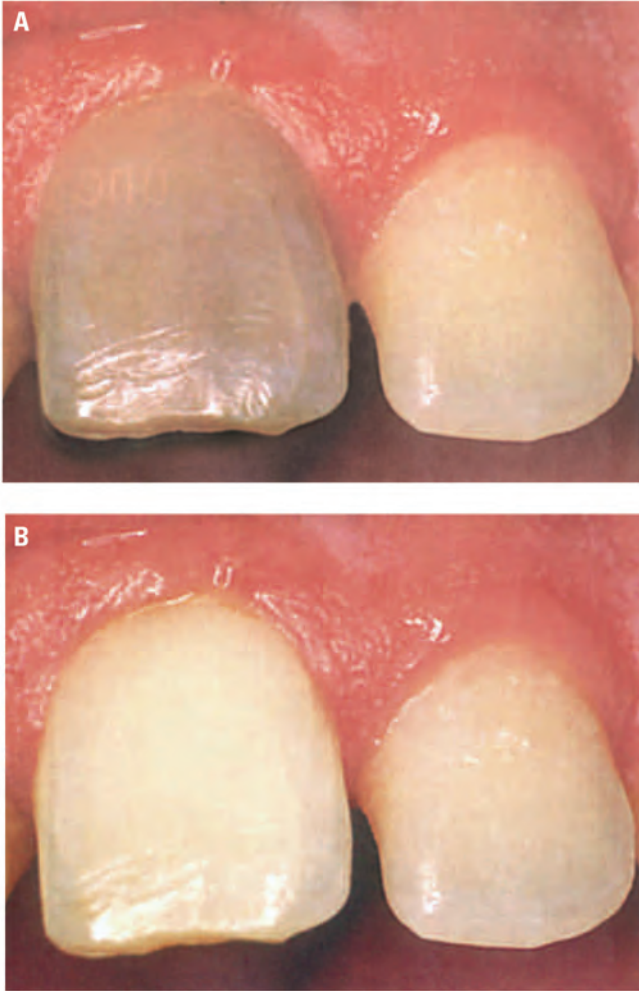


Figure 3-3. **A**, Post-traumatic discoloration of a maxillary left central incisor. **B**, A mixture of perborate and distilled water placed in the chamber two times over 3 weeks achieved a lightening of the tooth to its natural color. Courtesy of A. Claisse-Crinquette, Paris, France.

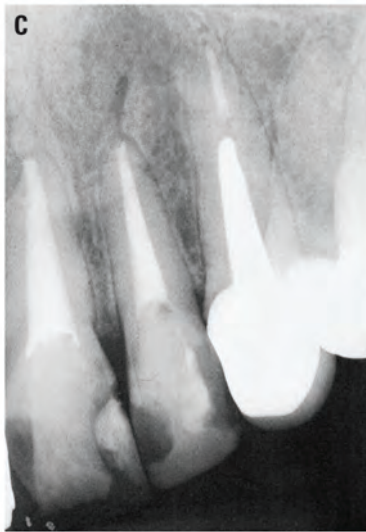
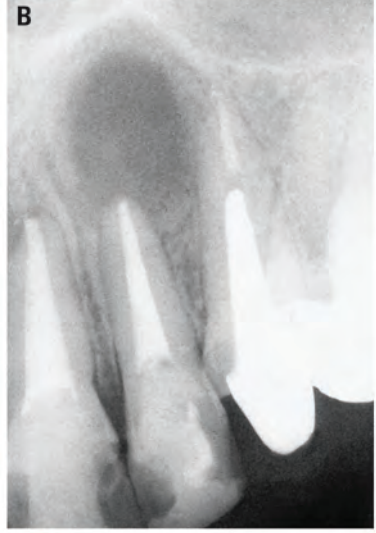
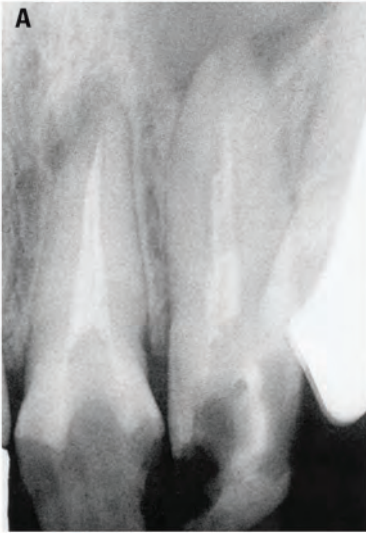


Figure 3-4. Root-end resection using laser energy. **A**, Pretreatment radiograph revealing failing endodontic treatments with periapical lesions. **B**, Radiograph following endodontic treatment and root-end resection of teeth #9 and #10 using an erbium: yttrium-aluminum-garnet laser. **C**, Twenty-six months postsurgical radiograph revealing good periradicular healing. Courtesy of S. Cecchini, Italy.

VITAL PULP THERAPY

There are occasions when one feels a pulp is salvable, its “sick” condition reversible. At these times, **vital pulp therapy** is employed. This might consist of the so-called **indirect pulp capping** procedure, **direct pulp capping**, and **coronal pulpotomy**, as well as **apexifica-**

tion in partially formed roots. Most of these latter procedures are carried out in children.

Indirect Pulp Capping

Indirect pulp capping has long been a controversial procedure. The technique is confined primarily to primary or young permanent teeth. It is defined as placing a medicament over a thin layer of carious dentin left after deep excavation, with **no pulp exposure**. The rationale is based on the fact that decalcification of the dentin precedes bacterial invasion and that the pulp may not be infected. It might be inflamed, but the inflammation is reversible if the cause (infected caries) is disinfected or removed and time allows healing. To properly determine the pulpal status of these teeth, the following criteria must be followed: no damage apparent radiologically, no history of spontaneous pain, no pain from percussion, no pain from mastication, and zero degree of mobility.

The most popular form of this procedure is the so-called **one-appointment technique**. Under anesthesia and the use of a rubber dam, the soft, necrotic, infected dentin is removed with a slow round bur. The remaining dentin is covered with a **hard-setting** calcium hydroxide cement, which is then protected with a reinforced zinc oxide–eugenol (ZOE) cement (IRM Dentsply-Caulk, York, PA) or glass ionomer. It is most important that the treatment be protected from microleakage of bacteria (Figure 3-5). Do not disturb for 6 to 8 weeks. Some dentists place a final amalgam restoration or stainless steel crown at this juncture. Success rates range from 62% to 98%; however, the success rate for adults is discouraging.¹⁵⁻¹⁶

Direct Pulp Capping

Direct pulp capping is carried out on healthy pulps that have been inadvertently exposed during caries excavation or from trauma. The treatment objective is to seal the pulp against bacterial leakage by placing a biocompatible agent on the exposure site that will encourage the formation of a dentin bridge. The criteria for treatment are the same as those listed for indirect pulp capping—any sign of pulp disease.

The protocol for direct pulp capping involves the following steps. A rubber dam should be placed, the bleeding controlled, the cavity wiped clean with a mild disinfectant agent such as chlorhexidine,

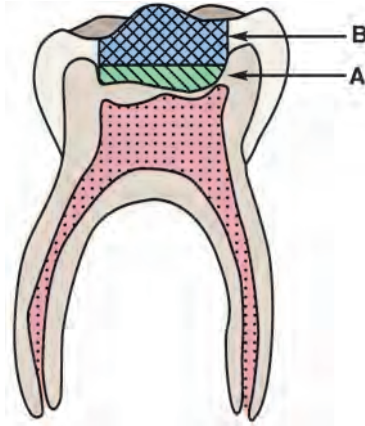


Figure 3-5. Indirect pulp-capping technique. **A**, Medicament of zinc oxide–eugenol cement, calcium hydroxide, or both is placed against the remaining caries. **B**, Lasting temporary restoration is placed. Following the repair, both materials are removed along with softened caries, and final restorations are placed.

and the pinpoint exposure covered with a capping agent such as $\text{Ca}(\text{OH})_2$ or MTA. This should then be sealed in place with a dentin bonding agent and covered over with a thick layer of hard-setting cement (Figure 3-6). Prevention of bacterial microleakage is imperative, so an amalgam restoration or a stainless steel crown should be placed in posterior teeth or a composite **sealed with dentin bonding**

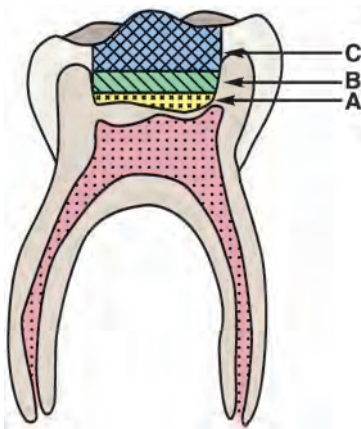


Figure 3-6. Direct pulp-capping technique. **A**, Capping material covers the pulp exposure and floor of the cavity. A protective base of zinc oxide–eugenol is placed, **B**, and covered with an amalgam restoration, **C**.

in the anterior. Months later, if there have been no overt signs of pathologic change, a permanent restoration may be placed in adults. Success is actually better in adults than in children.

The agents used to stimulate dentin formation are legion. However, the two favorites are calcium hydroxide and mineral trioxide aggregate (MTA; Dentsply/Tulsa). Zinc oxide–eugenol has not been successful. Recent success reports with MTA have been most encouraging.¹⁷ The sealant of choice could be C & B MetaBond (Parkell Co.).

Pulpotomy

Pulpotomy is the most widely used technique for carious pulp exposures in primary and young permanent teeth. It is also the most abused—wishful thinking, wishful waiting. Pulpotomy is the surgical removal of the entire coronal pulp presumed to be inflamed and/or infected. A germicidal medicament is then placed over the stumps of the pulp left in the roots. Again, there should be no signs of pathologic change in the radicular pulp or periradicular area. Bleeding that is profuse and unstoppable or absent, sluggish, or purulent is a contraindication, as is sensitivity to percussion.

A number of medicaments have been tried but there is no panacea. The favorite over the ages appears to be formocresol. The traditional one-appointment pulpotomy for primary teeth involves the following steps. The tooth is anesthetized, and a rubber dam is placed. All carious material is removed, and the coronal pulp is completely removed with a slow-speed, large, round bur. Bleeding is arrested, and diluted formocresol (1:5) is applied for 3 to 5 minutes on a large cotton pellet, squeezed out. A hard ZOE cement base is placed, and the tooth is restored with a stainless steel crown (Figure 3-7). A 3-year survival rate study showed a decreasing rate of success—91% success at 3 months and 70% success at 3 years, a decrease that may have been due to bacterial microleakage.¹⁸ Calcium hydroxide and zinc oxide–eugenol are **not** as successful as MTA and glutaraldehyde.^{19–20} Research has also been carried out using dentin adhesives to cover the stumps.

Pulpotomy in Young Permanent Teeth

Pulpotomy in young permanent teeth, particularly those with incompletely formed roots and open apices, has been recommended using

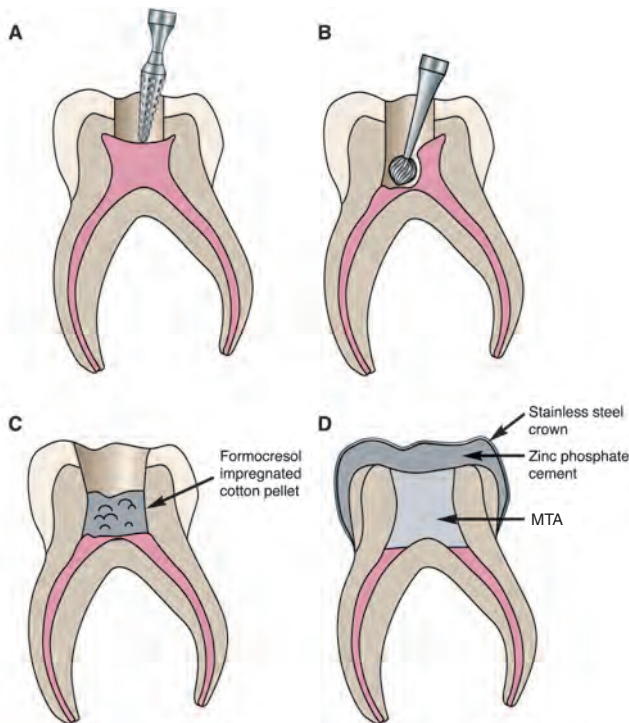


Figure 3-7. Step-by-step technique of one-appointment formocresol pulpotomy. **A**, Exposure of pulp by roof removal. **B**, Coronal pulp amputation with a round bur. Hemostasis with dry cotton or epinephrine. **C**, Application of formocresol for 1 minute. (Excess medicament is expressed from cotton before placement.) **D**, Following formocresol removal, an MTA base and stainless steel crown are placed.

calcium hydroxide as the agent of repair. The procedure is similar to that outlined previously, except that in young adult teeth calcium hydroxide or **MTA** is substituted for formocresol and is left in place (Figure 3-8). If infection is not allowed to intervene, apexification should take place to complete root growth and form and close the apical orifice. Amalgam fillings or stainless steel crowns are recommended to prevent microleakage that eventually leads to failure.

Apexification

Apexification of incompletely formed roots and apex, the so-called blunderbuss canal, can be accomplished by restoring root growth

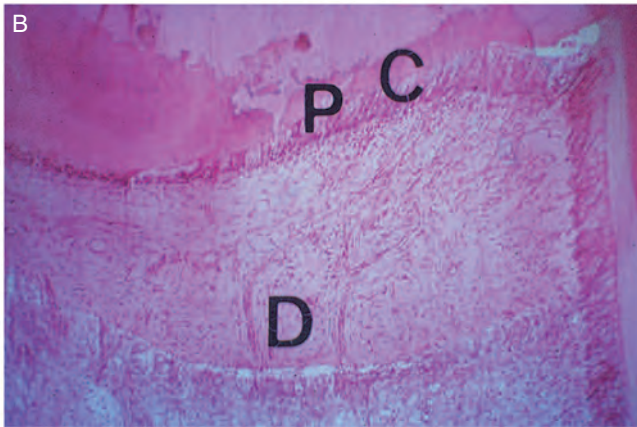
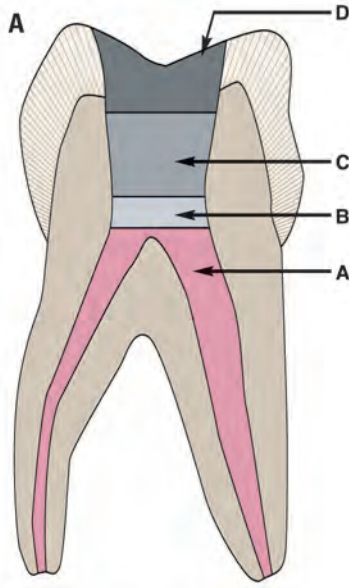


Figure 3-8. **A**, Calcium hydroxide or MTA pulpotomy in a young permanent molar. The cavity is prepared, caries and the chamber roof are removed, and the pulp is amputated to the canal orifices, (*A*). Following hemostasis, calcium hydroxide or MTA is placed (*B*) and protected with zinc oxide–eugenol (*C*) and an amalgam filling or stainless crown (*D*). **B**, MTA used as a covering following pulpotomy; (*P*) and (*C*) indicate the remnants of MTA at the pulpotomy site. Note the formation of odontoblasts immediately below and the formation of new dentin (*D*). Courtesy of M. Torabinejad and I.L. Soares.

and apical closure. The genetic potential is there if only the infection and inflammation can be arrested. It is also believed that this phenomenon can be initiated by introducing a stimulating agent such as calcium hydroxide or MTA. Unfortunately, CaOH_2 tends to make the dentin brittle; hence, there is the potential for later root fracture. Today, MTA is the preferred material.

A better way to bring about apexification is as follows:

1. The necrotic pulp and open divergent apex are gently cleaned with large blunted files, being careful not to reduce the thickness of the dentin. Plentiful irrigation with NaOCl removes debris and bacteria. The smear layer is removed using alternate irrigation with 6% NaOCl and 18% EDTA. A final flush of 2% chlorhexidine is applied before filling. This is the one-appointment treatment.
2. In the two-appointment treatment, the previous step is carried out but the canal is filled with CaOH as a medicament, and sealed with IRM for 2 weeks. This should effectively disinfect the canal but not damage the dentin.
3. On the return visit, the CaOH is removed and again the NaOCl and EDTA are used, the canal is dried and plugged with MTA using the MTA gun (MAP System, Roydent, Johnson City, TN), and ultrasonic plugs are used to fill the open apex.
4. The one-year recall revealed a 93.5% success for those cases completed in one appointment and a 90.5% success for those cases completed in two appointments (Figure 3-9).²¹

Pulpectomy

Pulpectomy, or pulp extirpation, is the removal of a vital pulp, diseased though it may be. **Total pulpectomy** is the removal of the pulp in fully formed roots, where the apical “portal of exit” is sufficiently closed to form an “apical stop” in obturation. **Partial pulpectomy** indicates that pulp extirpation terminates short of the apical orifice. It is used in those cases in which the apex has not fully formed and one hopes that the remaining pulp tissue will continue to fully form the root (Figure 3-10). Pulpectomy is indicated in all cases of irreversible pulp disease including internal resorption. In the event of acute pulpalgia, pulpectomy extends blessed relief to a suffering patient.

The following are the steps for performing pulpectomy:

1. Obtain regional anesthesia.
2. Open into the pulp and test for depth of anesthesia.
3. If necessary, inject anesthesia pulpally.
4. Extirpate the coronal pulp.
5. Extirpate the radicular pulp with barbed broaches.



Figure 3-9. Representative example of a case treated in one visit with MTA. **A**, Preoperative radiograph. **B**, Immediate post operative radiograph. **C**, Four-year recall radiograph showing completely healed periapical tissues.
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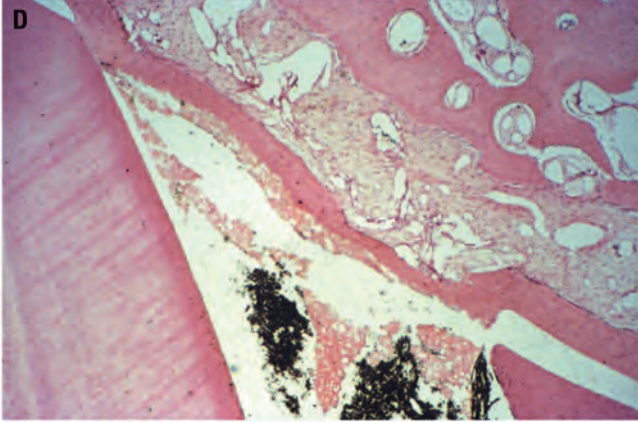


Figure 3-9. Continued. D, MTA used as a root-end filling material in a monkey. Note the band of cementum formed to close the root end. **A-C** From Witherspoon DE et al. J Endod 2008;34(10):1171–6. **D,** Courtesy of M. Torabinejad. Reproduced with permission.



Figure 3-10. Partial pulpectomy. An observation period of 6 months is needed. There is only a slight accumulation of lymphocytes adjacent to a plug of dentin particles and remnants of Kloropercha. Cell-rich fibrous connective tissue occupies the residual pulp canal. Large deposits of hard tissue (*H*) are present along the walls. Courtesy of P. Horstad and B. Nygaard-Ostby.

6. Control bleeding and clean and shape the canal.
7. Obturate the canal. In these cases, it is deemed appropriate to fill the canal at the same appointment because infection is not a problem. The patient may experience slight postoperative discomfort from the pulp amputation. Restoration of the crown may follow immediately.

Pulpectomy in a Large Canal

A pathway for the broach is formed by sliding a small anesthetic needle to the apical third and depositing a drop of anesthesia to control bleeding. A broach, small enough not to bind, is passed down the pathway to the apical third, twisted slowly to engage the fibrous pulp tissue, and slowly withdrawn. If fortunate, the entire pulp is removed (Figure 3-11). If not, the process is repeated. If the pulp is extra large, two or three broaches may be inserted simultaneously, twisted, and slowly withdrawn. Never lock broaches in the canal—they separate! They can be used to carefully “scrub” the walls of remaining fragments. Barbed broaches must be used with caution!

Pulpectomy in a Narrow Canal

In the case of pulpectomy in a narrow canal, broaches are not used. Small files are recommended, and the pulp is removed during cleaning and shaping. But be careful! Pulp fragments tend to pack at

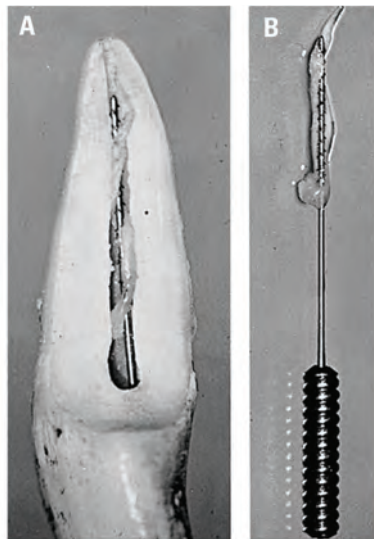


Figure 3-11. **A**, Total pulpectomy with a large broach that fits loosely in the canal. With careful rotation of the broach, the pulp has become entwined and will be removed with retraction. **B**, Total pulpectomy by a barbed broach. Young, huge pulps may require two or three broaches inserted simultaneously to successfully entwine the pulp.

the apex during cleaning and shaping and block access to the apical orifice. Fine instruments and copious irrigation with NaOCl must be used to remove the blockage.

Partial Pulpectomy

The site where the pulp is to be amputated is measured on a large, **blunted**, Hedstrom file. At this point, the file is gently “screwed” into the canal to engage the pulp, and is then slowly withdrawn. Bleeding is arrested. This should leave a stump of vital pulp tissue that may be necessary to complete root growth. (see Figures 3-9 & 3-10). The coronal portion of the canal is then cleaned and shaped, but it is probably not wise to obturate the canal at this appointment because the occasional patient continues to have pulpal pain. In this event, the patient must return and a total pulpectomy be performed. If the partial pulpectomy was successful, the remaining portion of the canal is gently obturated at a subsequent appointment. Without undue pressure and with blunted gutta-percha points, obturation is limited to the point of amputation.

APICAL PERIODONTITIS, GRANULOMA, OR CYST

It has long been known that some periapical lesions will heal following root canal obturation, but some will not. To assure healing and to speed recovery time, dentists have resorted to apical surgery, removing the chronic inflammatory lesion and allowing a blood clot to form. From this stage, healing begins immediately (Figure 3-12). Healing is much slower without surgery. However, surgery can be traumatic, and the American Association of Endodontists recommends performing surgery only in cases that cannot be treated otherwise.

There now is a way to remove the periapical lesion without surgery. An innovative procedure has been added to the endodontic armamentarium by Metzger and his associates in Israel and Romania.²² It is named the **Apexum procedure** (Apexum Ltd., Or-Yehyda, Israel) and consists of two devices, the Apexum Ablator and Apexum PGA Ablator, used sequentially. The entire procedure is carried out through the root canal after it has been cleaned and shaped. The apex is then perforated with a rotary no. 30 file (**ProFile .04**, Maillefer) creating a passage with about a 330- μ m diameter for

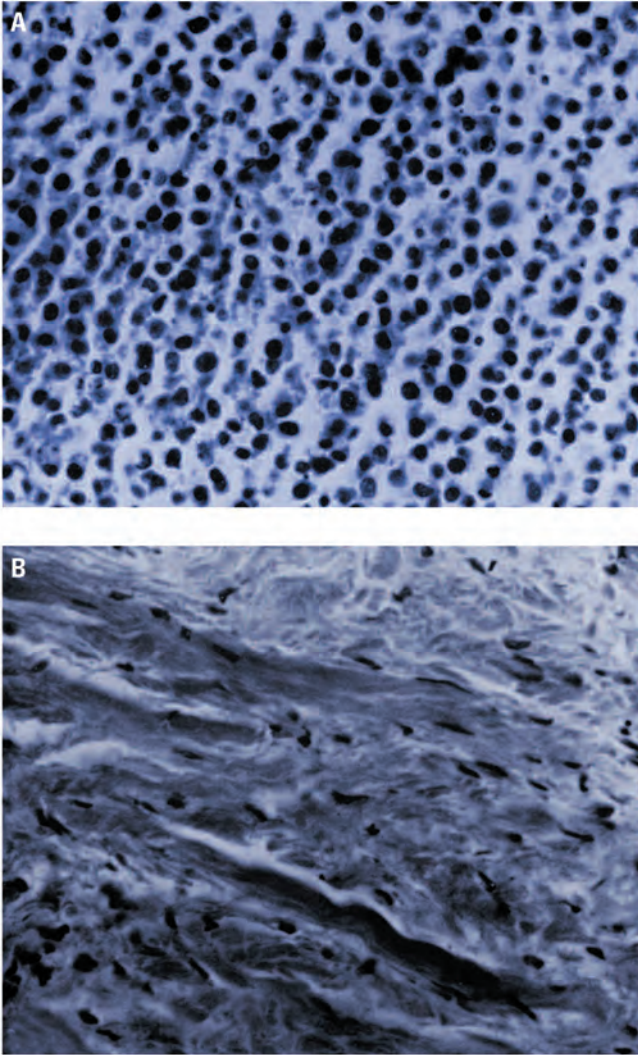


Figure 3-12. **A**, Biopsy of chronic inflammatory tissue removed from a chronic apical periodontitis lesion. **B**, Reentry biopsy taken 1 week later. Note the well-organized connective tissue fibers and cells that have developed in a remarkably short time. Healing is well on its way.

the Apexum Ablator to enter the apical lesion. The Ablator is a specially preshaped Nitinol wire, where one end is bent (Figure 3-13B,C) whereas the other end has a latch-type connector to allow its operation in a low-speed contra-angle handpiece.

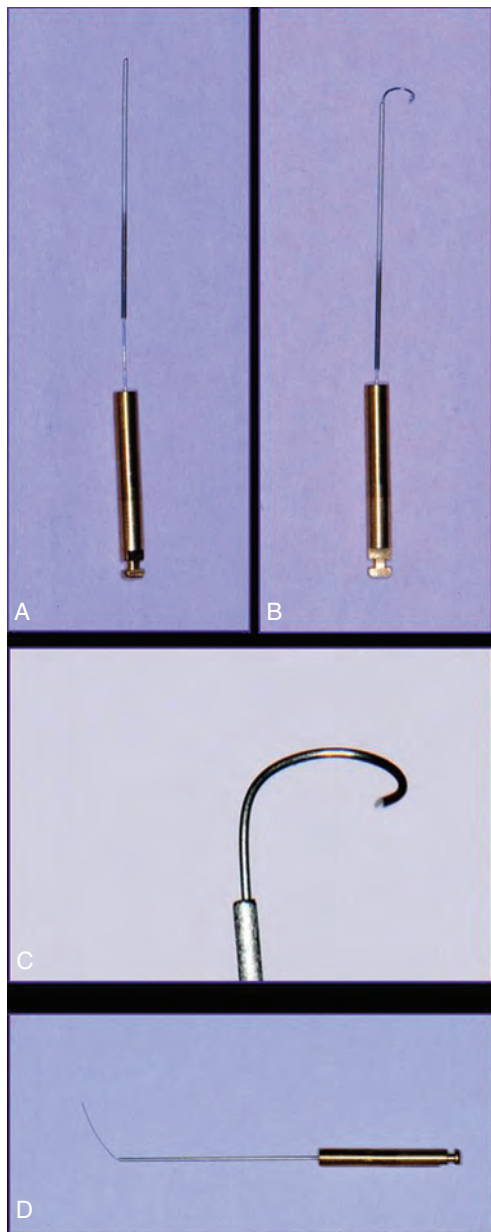


Figure 3-13. The Apexum devices. **A**, The Apexum NiTi Ablator in its sheath. **B**, The Apexum NiTi Ablator pushed in and extruded from its sheath. **C**, An enlarged view of the active part of the Apexum NiTi Ablator. **D**, The Apexum PGA Ablator. Metzger Z et al. J Endod 2009;35(2):155. Reproduced with permission.

The Ablator is carried to the apex concealed in a straight super-elastic Nitinol tube (Figure 3-13A). The wire Ablator is then inserted through the tube into the apical lesion and it opens up into its preformed curved shape (Figure 3-13C). With the handpiece, it is rotated for 30 seconds at 200 to 250 rpm to coarsely “mince” the contents of the lesion (Figure 3-14A).

The Ablator is then removed from the root canal with its sheath, the canal is rinsed with sterile saline, and the Apexum PGA Ablator, a bioabsorbable filament, is inserted into the periapical tissues (Figure 3-13D). It is rotated in place for 30 seconds at 5,000 to 7,000 rpm. This action turns the “minced” tissues into a thin suspension.

The Apexum PGA is then removed, and using a syringe with a 30G blunt needle, the suspension is now washed out with sterile saline. The opening in the apex is 3.4 times larger than the needle, allowing the suspension to flow readily. When the wash turns pale

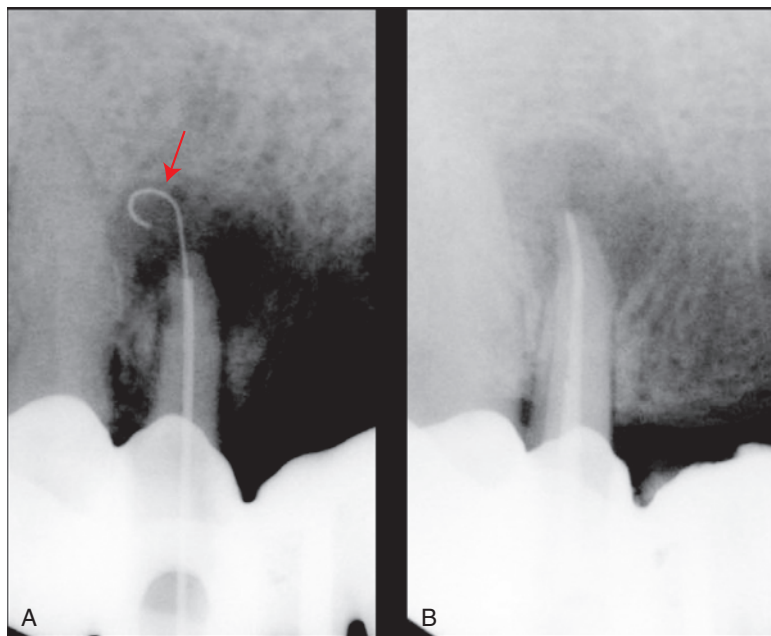


Figure 3-14. The Apexum NiTi Ablator in a periapical lesion. **A**, The Apexum NiTi Ablator fully extruded from its sheath and into the periapical lesion. **B**, Root canal filling performed immediately after the Apexum procedure. Metzger Z et al. J Endod 2009;35(2):155. Reproduced with permission.

and then clear, the removal of the lesion is complete. Dry with paper points and complete the obturation, starting with a blunted gutta-percha point (Figure 3-14B).

The Metzger group compared 39 teeth, with similar apical lesions, filled conventionally, against 48 similar teeth where the Apexum procedure was applied. Neither group had any adverse events.

Healing kinetics were significantly enhanced in the Apexum group ($p < 0.005$). “At 3 and 6 months, 87% and 95% of the lesions of the Apexum-treated group, respectively, presented advanced or complete healing, whereas only 22% and 39% of the lesions in the conventional treatment group presented this degree of healing at 3 and 6 months respectively.” (Figures 3-15 & 3-16).

“The Apexum procedure resulted in no adverse events, significantly less postoperative discomfort or pain than conventional root canal treatment or apical surgery, plus significantly faster healing ($p < 0.005$).”²³

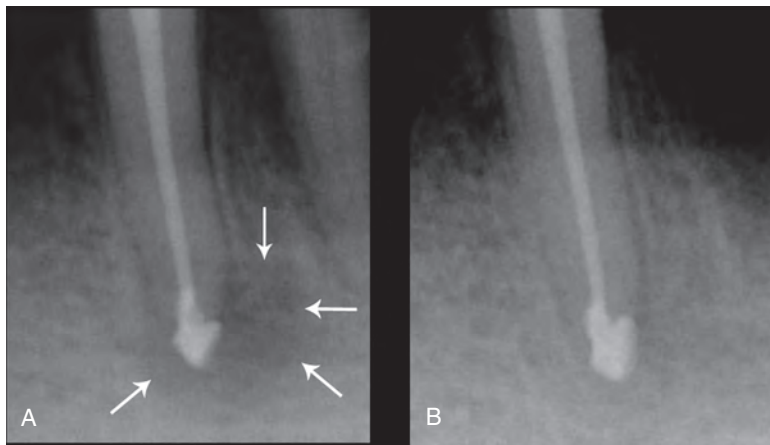


Figure 3-15. Healing of the periapical lesion after the Apexum procedure. **A**, The immediately postoperative radiograph; note the accidental major extrusion of the obturation material, a single case of its type in the whole study. Arrows indicate the extent of the lesion. **B**, Follow-up radiograph at 2 months. Metzger Z et al. J Endod 2009;35(2):158. Reproduced with permission.

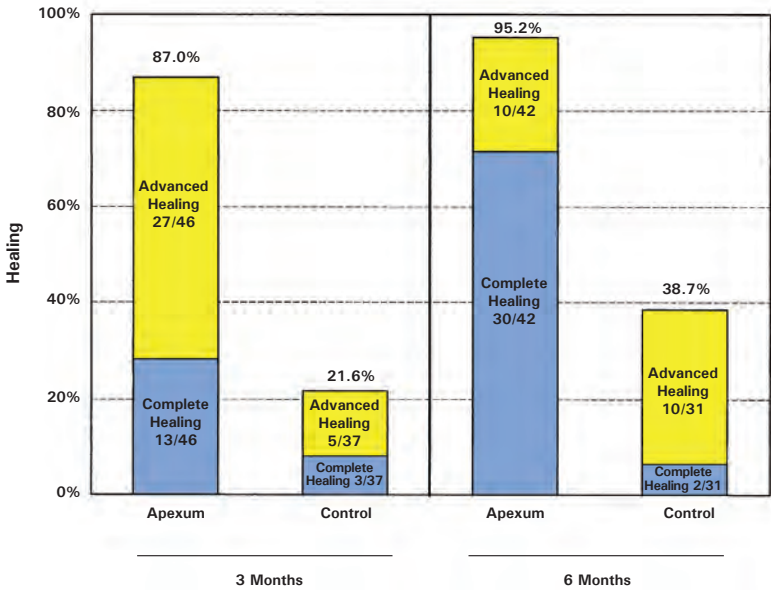


Figure 3-16. Healing in the Apexum and conventional (control) treatment groups; dichotomized data. Metzger Z et al. J Endod 2009;35(2):157.

FINAL NOTES

Additional endodontic procedures are covered in the following chapters: access (Chapters 4 and 5); cleaning, shaping, and medicating the canal (Chapter 6); obturation (Chapter 7); endodontic surgery (Chapter 8); mishaps (Chapter 9); emergencies, acute pain, dental trauma, and dental infection (Chapter 10); restoration of endodontically treated teeth (Chapter 11); and discoloration and bleaching (Chapter 12).

REFERENCES

1. Fox JL, Atkinson JS, Dinin PA. Incidence of pain following one-visit endodontic treatment. *Oral Surg* 1970;30:123.
2. Wolch I. The one-appointment endodontic technique. *J Can Dent Assoc* 1975;41:613.
3. Pekruhn RB. Single-visit endodontic therapy: a preliminary clinical study. *J Am Dent Assoc* 1981;103:875.
4. Roane JB, Dryden JA, Grimes EW. Incidence of post-operative pain after single- and multiple-visit endodontic procedures. *Oral Surg* 1983;55:68.
5. Trope M. Flare-up rate of single visit endodontics. *Int J Endodont* 1991; 24:24.
6. Eleazer PD, Eleazer KR. Flare-up rate in pulpally necrotic molars in one-visit versus two-visit endodontic treatment. *JOE* 1998;24:614.
7. Sjogren U. Influence of infection at the time of root-filling on the outcome of the endodontic treatment of teeth with apical periodontitis. *Int Endodont J* 1997;30:297.
8. Figini L, Lodi G et al. Single versus multiple visits for endodontic treatment of permanent teeth: a Cochrane systemic review. *J Endod* 2008;34 (9): 1041-7.
9. Baumgartner JC, Falkler WA, Jr. Bacteria in the apical 5mm of infected root canals. *J Endod* 1991;17(8):379-80.
10. Siqueira JF Jr, Rocas IN, Lopes HP. Patterns of microbial colonization in primary root canal infections. *Oral Surg* 2002;93:174.
11. Gutierrez JH, Jofre A, Villena F. Scanning electron microscope study of the action of endodontic irrigants on bacteria invading the dentinal tubules. *Oral Surg* 1990;69:491.
12. Barnett F et al. Demonstration of *Bacteroides intermedius* in periapical tissue using indirect immunofluorescence microscopy. *Endod Dent Traumatol* 1990;6:153.
13. Barnett F, Axelrod P, Tronstad L, et al. Ciprofloxacin treatment of periapical *Pseudomonas aeruginosa* infection. *Endod Dent Traumatol* 1988; 4:132.
14. Leonardo MR and Rossi, MA. EM evaluation of bacterial biofilm and microorganisms of the apical external root surface of human teeth. *JOE* 2002;28:815.
15. King J. Indirect pulp capping: a bacteriologic study of deep carious dentin in human teeth. *Oral Surg* 1965;20:663.
16. Jordan RE, Suzuki M. Conservative treatment of deep carious lesions. *Can Dent Assoc J* 1971;37:337.
17. Pitt Ford TR, Torabinejad M, Abedi HR. Using mineral trioxide aggregate as a pulp capping material. *J Am Dent Assoc* 1996;127:1491.
18. Rolling I, Thylstrup A. A three-year clinical follow-up study of pulp-otomized primary molars treated with the formocresol technique. *Scand J Dent Res* 1975;83:47.

19. Abedi HR, Ingle JI. Mineral trioxide aggregate: a review of a new cement. *Calif Dent Assoc J* 1995;23:36.
20. Kopel HM, Bernick S, Zachrisson E. The effects of glutaraldehyde on primary pulp tissue following coronal amputation: an in vivo histologic study. *J Dent Child* 1980;47:425.
21. Witherspoon DE, et al. Retrospective analysis of open apex teeth obturated with mineral trioxide aggregate. *J Endod* 2008;34(10):1171-6.
22. Metzger G, Huber R et al. Enhancement of healing kinetics of periapical lesions in dogs by the Apexum procedure. *J Endod* 2009;35(1):40-5.
23. Metzger G, Huber R et al. Healing kinetics of periapical lesions enhanced by the Apexum procedure: a clinical trial. *J Endod* 2009;35(2):153-9.

Access to the Root Canal System: Preparation for Treatment

ENDODONTIC RADIOGRAPHY

It is imperative that a good and current radiograph be available before root canal treatment is started. All too often, patients are referred for endodontic treatment along with a dated film or one of poor quality. The first order of procedures should be to take a new radiograph or, better yet, take additional films from a different horizontal angle. By this action, multiple roots or canals may be revealed (Figure 4-1), or what appears to be a periapical

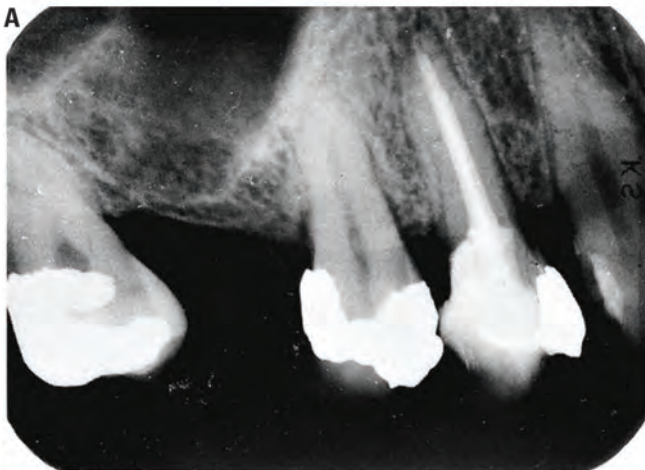


Figure 4-1. Maxillary premolars. **A**, Horizontal, right-angle projection produces the illusion that the maxillary molar has only one canal. *Continued on next page.*

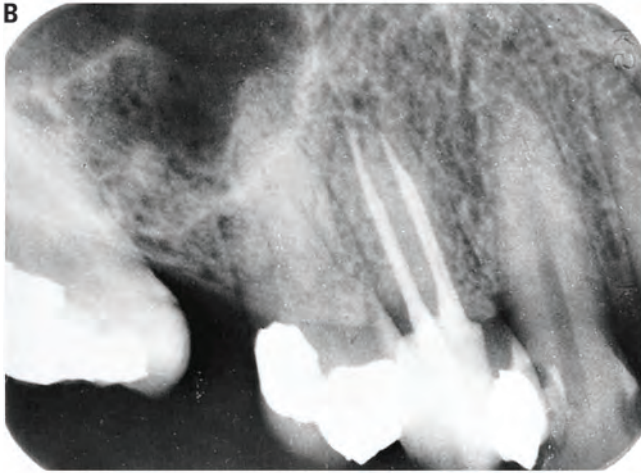


Figure 4-1. Continued. B, Varying the horizontal projection by 20° mesially separates the two canals. The lingual canal is toward the mesial. Courtesy of R. E. Walton.

lesion may be determined to be the mental or incisive foramen (Figure 4-2). Always check by pulp testing. Do not base your judgment on a single film. Remember, what one sees is a two-dimensional view of a three-dimensional object. Radiography, both traditional and digital, is discussed in Chapter 1.

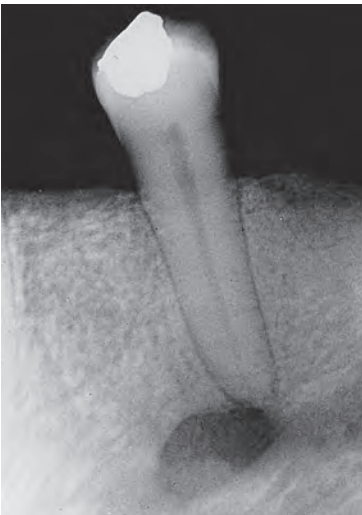


Figure 4-2. Mental foramen is superimposed exactly at the apex of the vital premolar, and may be easily mistaken for a periradicular lesion.

PULP TESTING

Pulp testing of the suspected tooth and adjacent teeth should follow radiography. Comparative tests on the opposite side and the opposite arch are often in order. Again, these procedures are covered in Chapter 1.

ANESTHESIA

Endodontic treatment should be painless. The old saw, “That’s worse than having a root canal,” should have long ago disappeared from our lexicon. However, some endodontic procedures can be painful unless special precautions are taken. On the other hand, some cases such as total pulp necrosis can be treated without anesthesia. In fact, during canal instrumentation as a file approaches the apical foramen, the patient often responds not in pain but in sensation. This is a warning that the working length had been reached. If anesthesia had been used, that length might have been exceeded. If the placement of a rubber dam clamp is likely to be painful, minimal local injections are in order. Block anesthesia is usually satisfactory for most cases.

Do not overlook the advantages of seldom-used block injection sites—for example, the mental foramen area, that blocks all mandibular teeth from second premolar to the midline and not the tongue; or the infraorbital injection, which blocks two premolars and the ipsilateral maxillary anterior teeth. This latter injection is made by inserting the needle into the buccal fold above the maxillary first premolar and aiming for the infraorbital foramen, found by palpation. A small deposit is made there and forced into the foramen by finger pressure. In the event that inflamed pulp tissue is not entirely anesthetized by blocks, there are additional injection techniques that can be used.

SUPPLEMENTAL INJECTION TECHNIQUES

Probably the most widely employed supplemental injection is the **periodontal ligament (PDL) injection**. It is used principally in the mandible when block anesthesia is not complete. The needle is

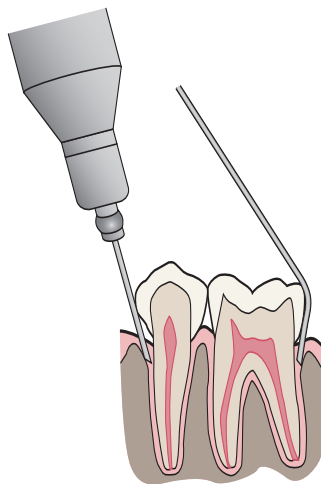


Figure 4-3. Insertion of the needle for periodontal ligament injection. Incorrect insertion of the sharp needle tip toward the root (*left*). Correct insertion of the bevel facing the root (*right*).

placed alongside the root of the tooth. **The bevel of the needle, not the sharp tip, faces the root!** The needle is then advanced down the PDL space, and 0.02 mL of solution is slowly deposited (Figure 4-3). Each root must be separately anesthetized. Onset is immediate; although limited, the duration should be long enough to enter the pulp and complete the pulpectomy. PDL injections should not be used on primary teeth or teeth with periodontal infection.

Intraosseous anesthesia is indicated in those cases of a “hot tooth” that seems refractory to being anesthetized. The anesthetic is injected directly into the bone surrounding the root. To do this, a small perforation must be made through the heavy cortical bone with a tiny dental bur. The needle is inserted through this hole, and 0.45 mL to 0.6 mL of anesthetic solution is deposited in the highly vascular cancellous bone.

The recent introduction of the **X-tip Intraosseous Anesthesia Delivery System** (Maillefer/Dentsply) has greatly improved the efficacy of this form of anesthesia. The X-tip comes in two parts: a tiny drill used in a slow-speed handpiece to perforate the cortical plate, and a very short 27-gauge needle that inserts into the guide sleeve left behind (Figure 4-4A). After the anesthetic is injected, the guide sleeve is withdrawn with a hemostat. Similar systems are also available.

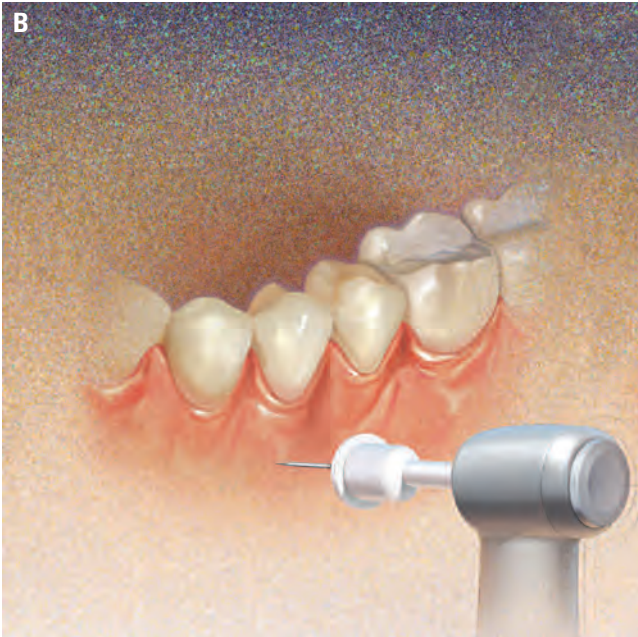
A**B**

Figure 4-4. Intraosseous anesthesia delivery system X-tip (Dentsply/Maillefer). **A**, The X-tip system comes in two parts: the drill and the guide sleeve with special injection needle. To use the system, first anesthetize the area with a few drops of anesthetic in the mucobuccal fold. Select a site 2 mm to 4 mm apical to the bony crest and between the roots. **B**, Place the X-tip drill and guide sleeve in a slow-speed (15,000–20,000 rpm) handpiece, and drill at maximum speed at 90° to the bone. In 2 to 4 seconds, the drill will perforate the cortical bone to the cancellous bone. *Continued on next page.*

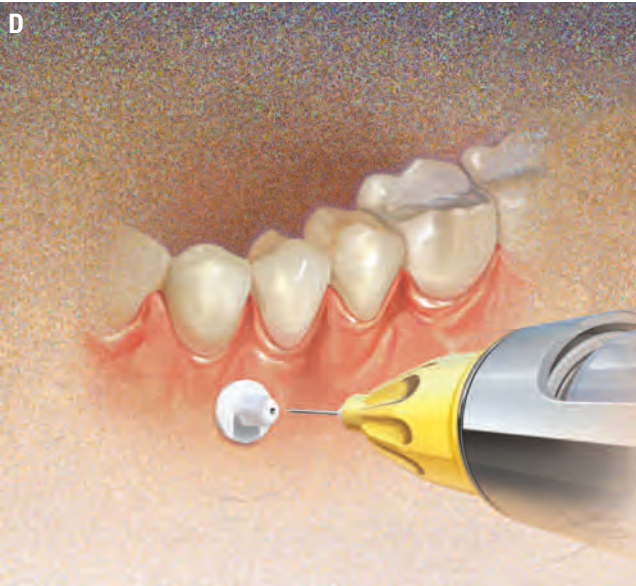


Figure 4-4. Continued. **C**, Hold the guide sleeve in place and withdraw the drill. **D**, Insert the special short needle into the tiny hole in the guide sleeve and slowly inject a few drops of anesthetic. In the event additional anesthesia may be needed, the guide sleeve may be left in place until the end of the appointment

However, a word of caution: Owing to rapid absorption, one must be careful not to inject too much anesthetic. A reaction to the anesthetic or the vasopressor may ensue. For this reason, a **nonepinephrine anesthetic should be used**. Anesthesia usually applies to one or two teeth. This injection has proven of great value when extirpating pulps with irreversible pulpitis refractory to block anesthesia.

Because it can be momentarily painful, **intrapulpal anesthesia** is the anesthesia of last resort. While injecting ahead, the needle is inserted into the pulp chamber and down a canal until it meets resistance. The anesthetic must be injected under pressure. A tiny amount of solution is deposited into the pulp tissue (Figure 4-5). Although it is painful at first, the discomfort should subside immediately and one may proceed with impunity.

When all else fails, concurrent administration of **inhalation sedation (N₂O-O₂)** or **intravenous midazolam** (versed) provides conscious sedation and negates any pain response.

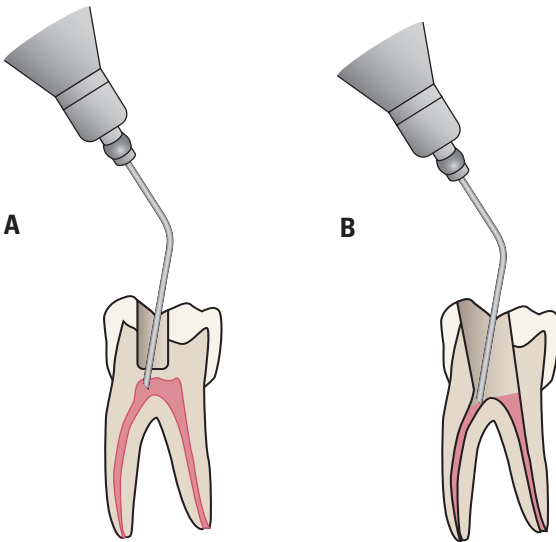


Figure 4-5. Intrapulpal pressure anesthesia with lidocaine. **A**, Coronal injection through a pinhole opening in the dentin. **B**, Pulp canal injection for each individual canal. The needle is inserted tightly, and one drop of solution is deposited. Courtesy of C. Lambert and G. Lambert.

RUBBER DAM APPLICATION

It is mandatory that a rubber dam be used during endodontic treatment to isolate the involved tooth and prevent further contamination of the root canal. Besides providing a dry, clean, and disinfected field, the dam protects the patient from swallowing or aspirating endodontic instruments or debris (Figure 4-6). Usually, only the sin-

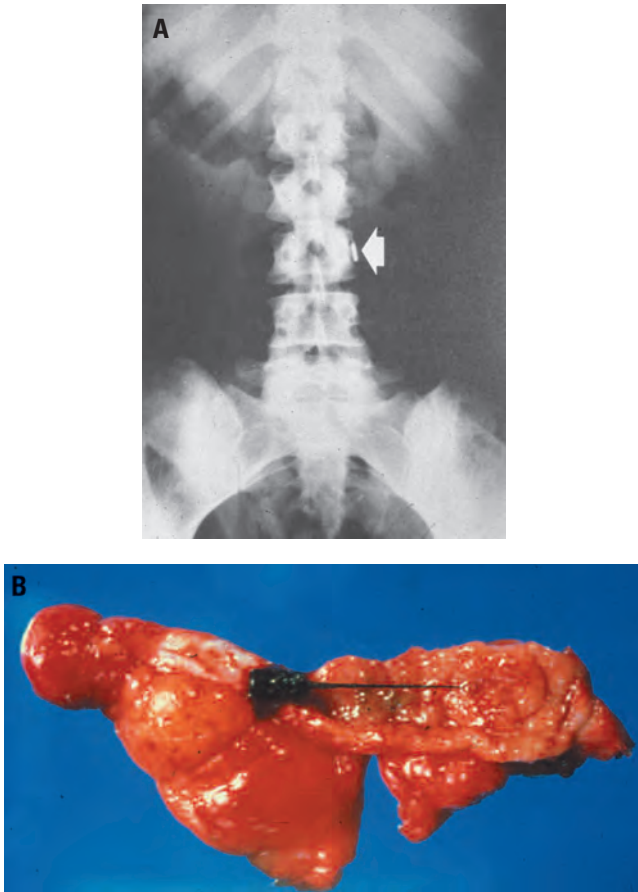


Figure 4-6. **A**, Abdominal radiograph of a swallowed endodontic instrument now caught in the duodenum. It must be removed surgically. Courtesy of J. Goultchin and B. Heling. **B**, Swallowed endodontic file that ended up in the appendix and led to acute appendicitis and appendectomy. The use of a rubber dam would have prevented this tragedy. Courtesy of L. C. Thomsen and colleagues.

gle tooth must be incorporated in the dam. The dam can be placed in less than a minute. The best dam sizes for endodontics are the 12.7 cm × 12.7 cm (5 in. × 5 in.) or 15.2 cm × 15.2 cm (6 in. × 6 in.) sheets; however, occasionally a small circular dam (Zirc) or the small oblong **HandyDam** (Dentsply) may be used for dressing changes or emergencies (Figure 4-7).

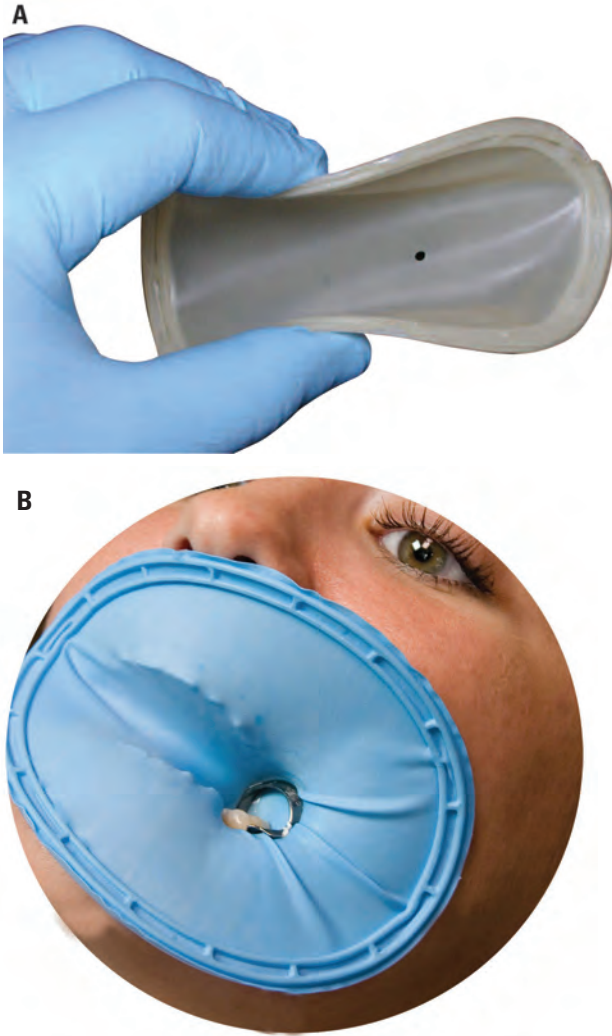


Figure 4-7. **A**, Instant, circular rubber dam with attached frame (Dentsply/Maillefer). **B**, Insta-Dam (Zirc Co.) used to isolate a single tooth for endodontic therapy.

“Rubber” dams come in latex and nonlatex materials—**silicone rubber** (Coltene/Whaledent/Hygenic)—for patients and dentists who are allergic to latex. Most popular are the nonmetal, **radiolucent rubber dam frames** that do not block important areas from x-rays. These include the shield-shaped **Nygaard-Ostby** frame (Figure 4-8A) (Coltene/Whaledent/Hygenic), the **Starlite VisuFrame** (Interdent), and the **Articulated Frame** (Dentsply/Maillefer), which may be folded back to better place x-ray films (Figure 4-8B). A new soft, metal frame that may be formed into shapes to fit the face has recently been introduced (**Derma Frame**, Ultradent Products; Figure 4-8C).

These frames also hold the dam away from the face so the patient may breathe more freely. The dam is best mounted high enough on the face to cover the nostrils; that way, the patient is breathing behind the dam and not exhaling bacteria down into the operating field (see Figure 4-8A).



Figure 4-8. A, Nygaard-Ostby Rubber Dam Frame (Coltene/Whaledent/Hygenic), developed in nylon by Nygaard-Ostby, is radiolucent and does not impede x-rays. The frame is curved to fit the patient’s face, and may be positioned so that the patient breathes behind the dam and not into the operating field. **Continued on next page.**

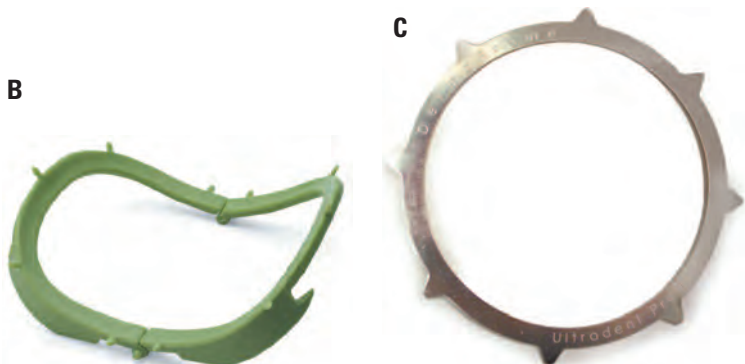


Figure 4-8. Continued. **B**, Articulated rubber dam frame (Dentsply/Maillefer). In the closed position, the frame is curved to fit the face. In the open position, it may be folded back, allowing the passage of a radiographic film holder. **C**, Derma Frame (Ultradent Products) is a soft, metal frame that may be formed to fit the patient's face. The frame retains its configuration but then may be reshaped.

Most dentists can get by with five to seven rubber dam clamps. However, unusual cases such as those involving rotated, malaligned, fractured, or partially erupted teeth will require additional clamps. The rubber dam clamp selection shown in Table 4-1 is a complete

Table 4-1

RUBBER DAM CLAMP SELECTION

Tooth	Rubber Dam Clamp
Maxillary teeth	
Central incisor	Ivory 00, 2, 212, or 9A; Hu-Friedy 27; Ash C
Lateral incisor	Ivory 00, 212, or 9A; Ash C
Canine	Ivory 2, 2A, 212, or 9A
Premolars	Ivory 2 or 2A; Hu-Friedy 27
Molars	Ivory 3, 4, 8A, 12A, 13A, 14, or 14A; Ash A
Mandibular teeth	
Incisors	Ivory 0, 00, 212, or 9A; Ash C
Canine	Ivory 2, 2A, 212, or 9A
Premolars	Ivory 2 or 2A; Hu-Friedy 27
Molars	Ivory 8A, 12A, 13A, 14, 14A, or 26, or fatigued Ivory 2A; Hu-Friedy 18, Ash A

list to cover any exigency. Clamps with “wings” are helpful in holding the dam down buccally and lingually.

Dam placement is started by punching a single hole in the dam in the proper location for the tooth in question. The punched hole, well off-center, can be positioned for any tooth by rotating the dam for an upper or lower tooth or to the right or left. It can also be placed on the frame ahead of time according to the tooth location. The clamp can be inserted into the punched hole with the bow to the distal side before the dam is positioned on the tooth. The wings are again useful in this instance. Much of this preparation can be done by the dental assistant. Incidentally, a good precaution is to mark the selected tooth with a felt marking pen to be sure the correct tooth is clamped (Figure 4-9).

The clamp is then spread with the **rubber dam forceps** and placed over the marked tooth. Mesially and distally, the dam is pulled through the contact points with dental floss. A blunted instrument is used to tuck the dam into the gingival crevice all around the tooth. In the posterior, adding clamps **over the outside of the dam** two or three teeth distant mesially and distally provides more working room (Figure 4-10A). The mesial clamp should be reversed with the bow to the mesial. A saliva ejector should be placed under the dam, not in a hole cut in the dam. In the event a leak develops, it may be stopped by applying Oraseal (Ultradent Products; Figures



Figure 4-9. Prior to dam placement the involved tooth is marked with a marking pen.

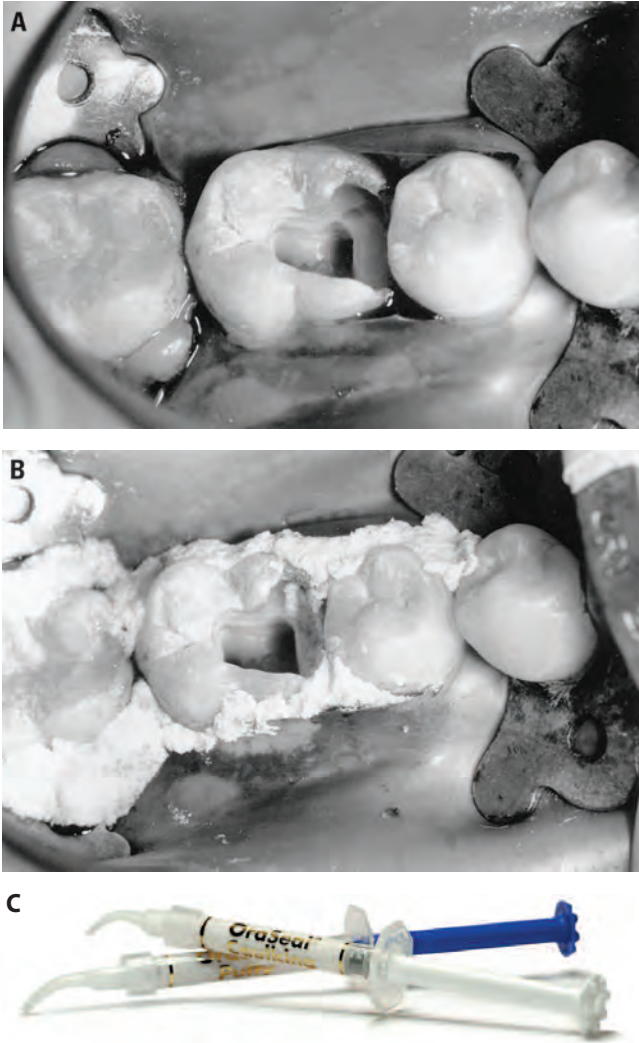


Figure 4-10. **A**, Four-tooth and two-clamp dam isolation in a patient with dilantin hyperplasia. **B**, Possible leakage toward the buccal and lingual aspects is controlled by Oraseal (Ultradent Products, Inc.). Courtesy of J.M. Coil. **C**, Oraseal injectable sealant.

4-10B and 4-10C). When the dam is removed, it is important it be inspected to be sure no interproximal dam septum was left.

Again, it is imperative that the rubber dam be used in all endodontic cases.

Access to the Root Canal System: Coronal Cavity Preparation

Careful cavity preparation and obturation are the keystones to successful root canal treatment. **As in restorative dentistry, the final restoration is no better than the initial cavity preparation.** Endodontic cavity preparation begins the instant the tooth is approached with a cutting instrument (Figure 5-1). Hence, it is important that adequate access be developed to properly clean and shape the canal system and obturate the space. When first approaching the tooth, one must have in mind the three-dimensional anatomy of the pulp chamber about to be entered, not the two-dimensional image revealed by the radiograph (Figure 5-2). It is this chamber outline that is to be “projected” out onto the occlusal or lingual surface of the crown (Figure 5-3).

Endodontic cavity preparation is separated into two anatomic entities: **coronal preparation** and **radicular preparation**. Coronal preparation is discussed in this chapter. In doing so one may fall back on Black’s principles of cavity preparation—**outline, convenience, retention, and resistance forms**—admittedly developed by Black for extracoronary preparation but just as applicable to intracoronary preparation (Figure 5-4). One may add **removal of remaining carious dentin (and defective restorations)** as well as **toilet of the cavity** (both of which are necessary) to make Black’s principles complete.

Outline form is often thought of as only the coronal cavity. But actually the entire preparation, from enamel surface to the apical terminus, is one long outline form (Figure 5-5). On occasion outline may have to be modified for the sake of **convenience** to

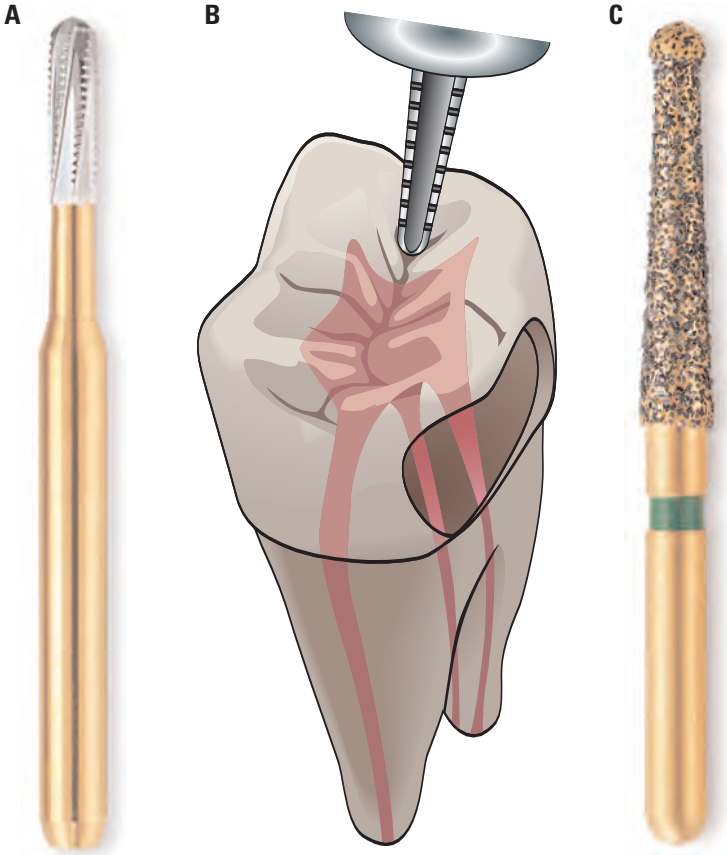


Figure 5-1. **A–C,** Entrance is always gained through the occlusal surface of posterior teeth and the lingual surface of anterior teeth, **B.** Specialized end-cutting fissure burs are used for the initial entrée through enamel or gold, **A,** and a special end-cutting amalgam bur is used to perforate amalgam fillings, **C.** The same burs and stones may be used to extend the walls to gain full access, bearing in mind that they are end-cutting and can damage the chamber floor.

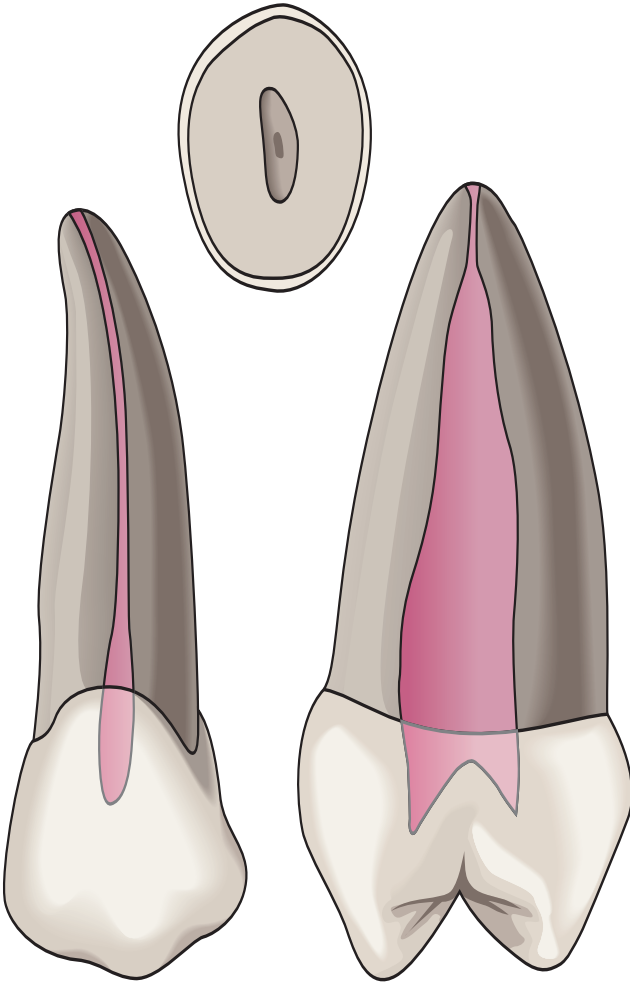


Figure 5-2. A standard radiograph (*left*) in buccolingual projection provides only a two-dimensional view of a three-dimensional problem. The maxillary second premolar is actually an ovoid ribbon, rather than a round thread.

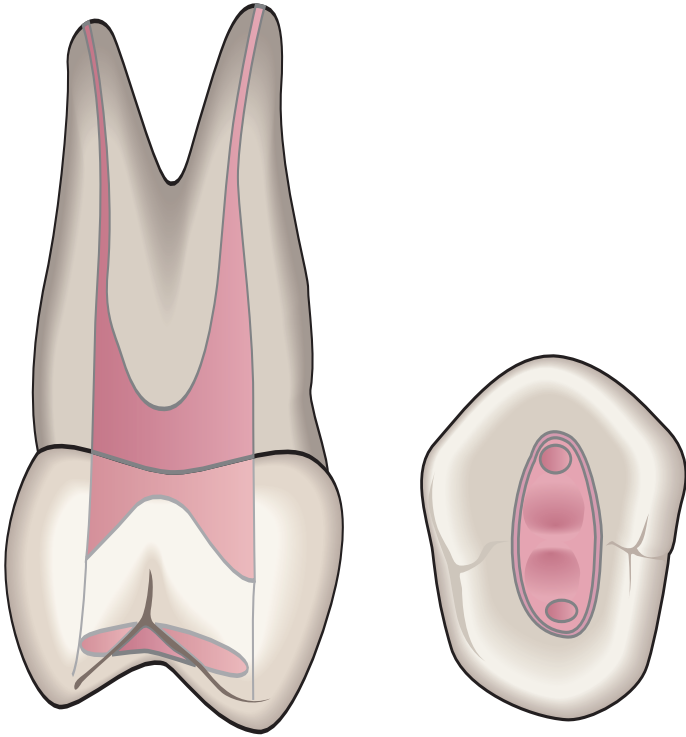


Figure 5-3. To gain adequate access to both canals, it is this broad ovoid outline that is projected out onto the occlusal surface, rather than a round “hole” in the central pit.

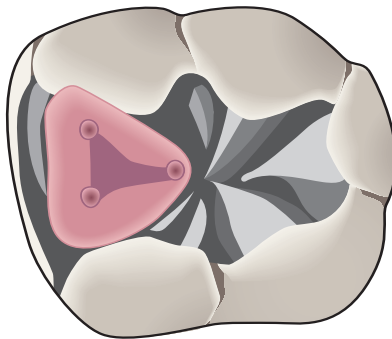


Figure 5-4. Black’s principles of cavity preparation—outline, convenience, retention, and resistance forms—apply to endodontic preparations as they do for coronal preparations: to the former to gain unlimited access to the canal orifices, to the latter as “extension for prevention.”

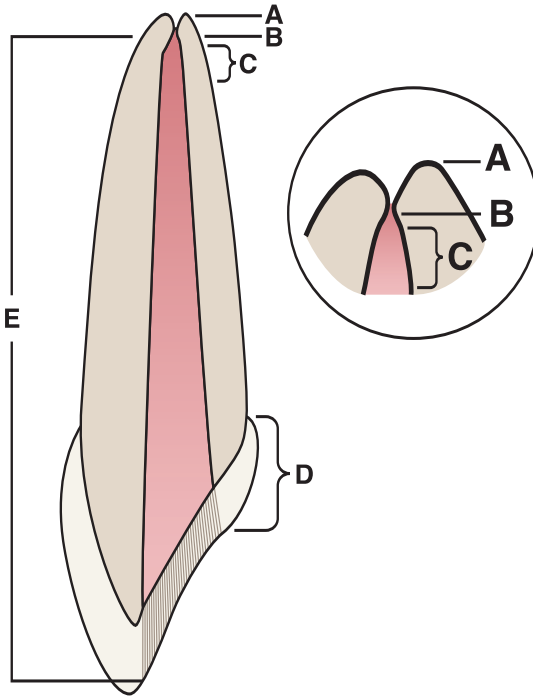


Figure 5-5. Concept of total endodontic cavity preparation, coronal and radicular as a continuum, based on Black's principles. **A**, Radiographic apex. **B**, Resistance form at the "apical stop." **C**, Retention form to retain the primary filling material. **D**, Convenience form subject to revision to accommodate larger, less flexible instruments. **E**, Outline form with basic preparation throughout its length, crown to apical stop.

accommodate the unstressed use of root canal instruments and filling materials (Figure 5-6). In other words, to reach the apical terminus without interference from overhanging tooth structure or ultra-curved canals, one must extend the cavity outline.

Also, on occasion the canal may be prepared for **retention** of the primary filling point (see Figure 5-5). However, more important is **resistance form**, to prepare an "apical stop" at the canal terminus against which filling materials may be compressed without overextending the filling (see Figure 5-5). Proper preparation of the apical one-third of the canal is crucial to success.

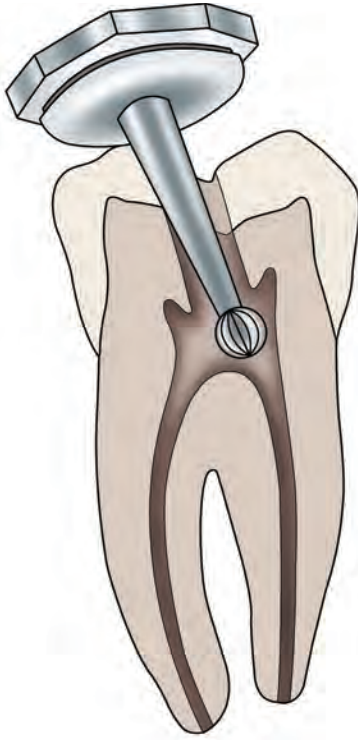


Figure 5-6. According to the size of the chamber, a no. 4 or no. 6 round bur may be used to remove the roof of the pulp chamber.

Power-driven rotary instruments are used to penetrate the crown. Round carbide burs commensurate in size with the chamber, as seen on a radiograph, or round-tip, end-cutting tapered burs (**Transmetal** - Caulk/Maillefer) or diamond stones (**EndoAccess**-Caulk/Maillefer, Tulsa, OK) are best for perforating enamel and entering the chamber (Figure 5-6). Enamel and precious metals are easily removed with carbide burs, but extra-coarse, round-tip diamonds are best for penetrating porcelain-fused-to-metal and all-porcelain restorations. Once the chamber is entered, the remaining tooth structure covering the chamber is removed with round or tapered burs or stones (Figure 5-7).

Initially, high-speed rotary instruments are used. But once the chamber is entered, **the neophyte** is advised to use slower-speed

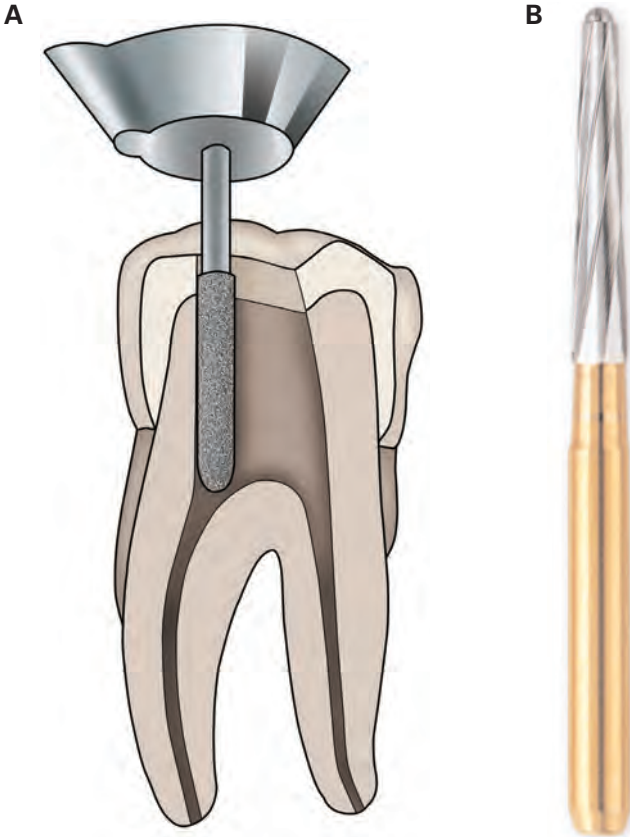


Figure 5-7. **A**, Final finish of the convenience form. In the case of a lower molar, the entire cavity slopes to the mesial aspect allowing easier access to the mesial canal orifices. **B**, Pulp-Shaper bur with noncutting tip (Dentsply/Tulsa) used to extend the access.

instruments so that tactile sensation may, in part, guide the removal of the remaining structure. To overcut the crown only weakens it more.

As soon as the canal orifices have been exposed, they may be entered with endodontic files to determine whether the instruments are either “under stress” or free of interfering tooth structure (Figure 5-8). If binding, convenience form dictates that the coronal outline form be extended to free up the shaft of the file. There should

be unobstructed access to the canal orifices and direct access to the apical foramen. This is done with high-speed tapered burs or stones, preferably with noncutting tips (Figure 5-9). Warning: fissure burs often “chatter,” distressing to the patient.

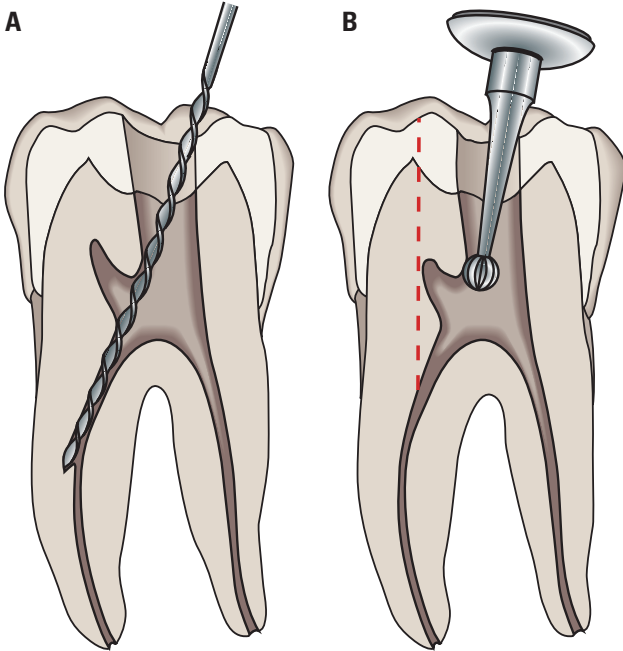


Figure 5-8. **A**, Obstructed access to a mesial canal. The overhanging roof misdirects the instrument mesially, with a resulting ledge formation. **B**, Completely removing the roof brings canal orifices into view and allows immediate access to each canal.

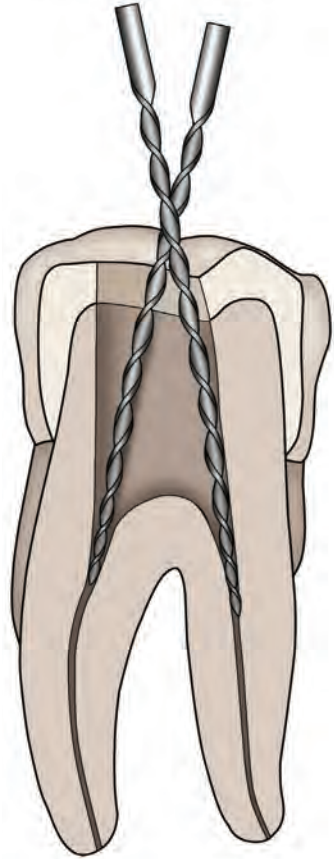


Figure 5-9. Unobstructed access to the canal orifices and down the canals to the “apical stop” area.

MAXILLARY ANTERIOR TEETH

Maxillary anterior teeth are entered from the lingual aspect. The enamel is perforated with a round carbide bur, an end-cutting, tapered bur, or a diamond stone held parallel to the long axis of the tooth (Figure 5-10). As soon as the pulp chamber is entered, the tapered bur is used to remove tooth structure incisally (Figure 5-11). This convenience removal allows adequate room for the shaft of burs that will enter deeper into the pulp chamber to remove its “roof” (Figure 5-12). Once the preparation is completed on the incisalabial surface, a tapered stone is used to remove the lingual “shoulder” (Figure 5-13).

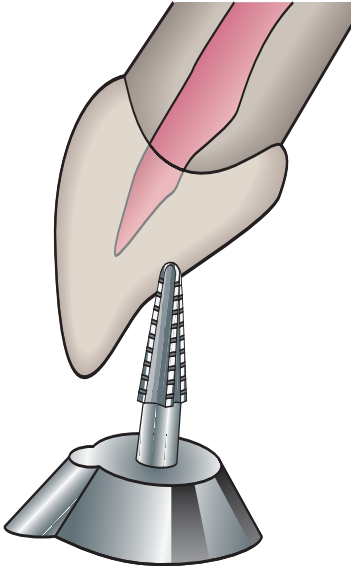


Figure 5-10. Maxillary anterior teeth. Entrance is always gained through the lingual surface. Initial penetration is made at the exact center of the lingual surface. The bur should be held approximately parallel to the long axis of the tooth. This initial cut may be made with high-speed instruments, but the neophyte is warned to use slower speeds in proceeding toward the canal.

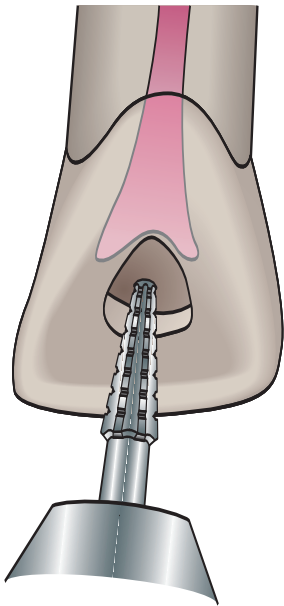


Figure 5-11. Maxillary anterior teeth. The preliminary cavity outline is funneled and fanned from the incisal edge to allow room for the shaft of the bur to follow and penetrate the chamber.

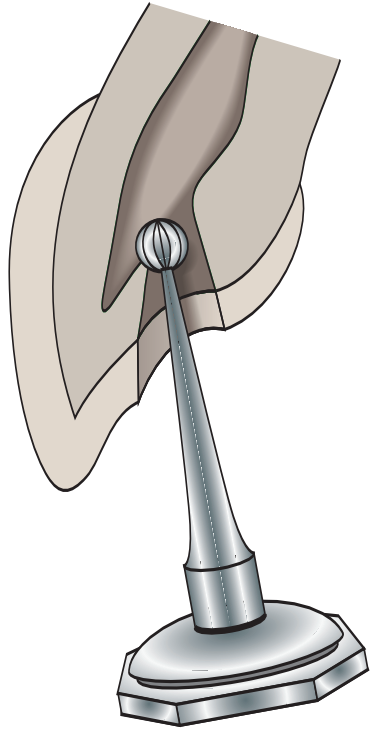


Figure 5-12. Maxillary anterior teeth. Use of a slower-speed round bur is suggested to enter the pulp chamber, keeping in mind the verticality of the tooth. The roof of the pulp chamber is removed toward the incisal surface in a convenience extension.

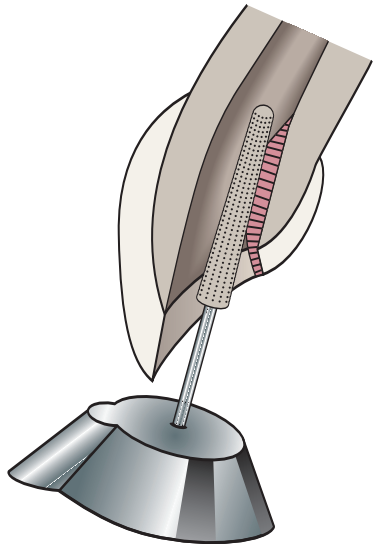


Figure 5-13. Maxillary anterior teeth. A fissure-style bur or diamond is used to remove the lingual shoulder and prepare the incisal extension to allow unobstructed access to the entire canal.

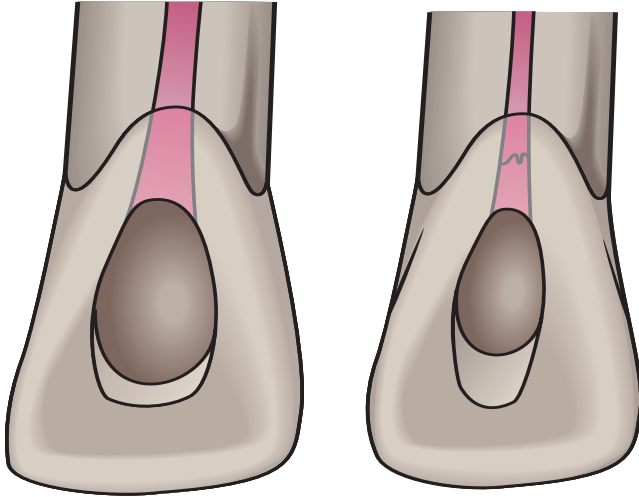


Figure 5-14. Maxillary anterior teeth. The lingual outline form reflects the size of the pulp chamber—a larger, fan-shaped outline in youngsters and a long, ovoid outline in older patients. Be sure to remove any pulpal remnants to the mesial or distal aspects to prevent future staining.

The final outline form on the lingual surface should reflect the size and shape of the pulp chamber, usually dictated by age (Figure 5-14). The entire outline form, from incisal to apex, must be free of any encumbrances that would interfere with cleaning, shaping, and obturation (Figure 5-15).

Operative Errors

The common error of perforating or badly gouging the gingivo/labial aspect (Figure 5-16) is usually due to two factors: not allowing adequate access toward the incisal aspect of the preparation (see Figure 5-11) or not properly aligning the bur vertically with the long axis of the tooth.

Another common failure is not providing adequate access or removal of the lingual shoulder (see Figures 5-11 and 5-13). Loss of control of the instrument results in a pear-shaped and inadequate preparation of the apical third (Figure 5-17).

A similar failure, the result of inadequate access, diverts instruments from the canal lumen, as illustrated here in a canine with a labial root curvature, undetectable in a standard labial-lingual radiograph (Figure 5-18).

Figure 5-15. Maxillary anterior teeth. Final preparation with the instrument in place. The instrument shaft clears the incisal cavity margin and the reduced lingual shoulder, allowing an unstrained approach, under the complete control of the clinician, to the apical third of the canal. **Virtually the same procedures and precautions apply to maxillary lateral incisors and canines as to central incisors.**

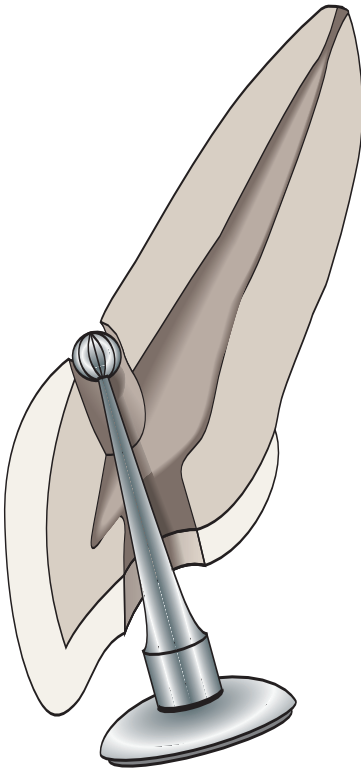
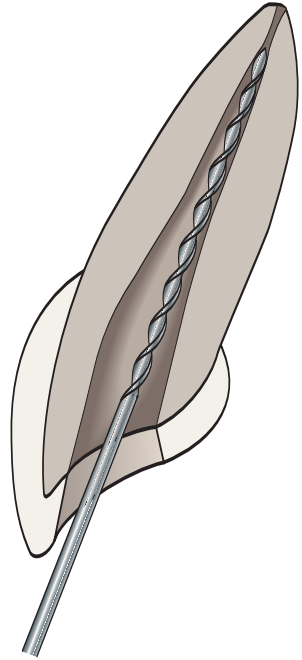


Figure 5-16. Operative error—maxillary anterior teeth. Perforation of the labiocervical aspect caused by failure to complete the convenience extension toward the incisal prior to entrance of the shaft of the bur. This also can be caused by a failure to align the bur parallel to the long axis of the tooth.

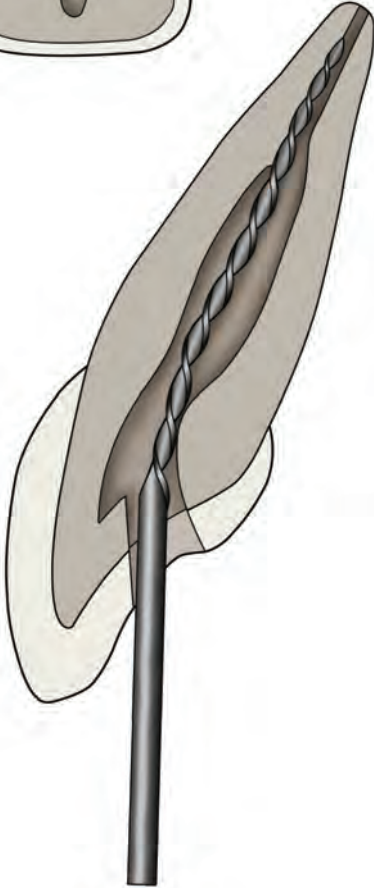


Figure 5-17. Operative error—maxillary anterior teeth. Pear-shaped apical preparation caused by a failure to complete the convenience extensions. The shaft of the instrument rides on the cavity margin and the lingual shoulder that direct the control of the instrument. Inadequate débridement and obturation ensure failure.

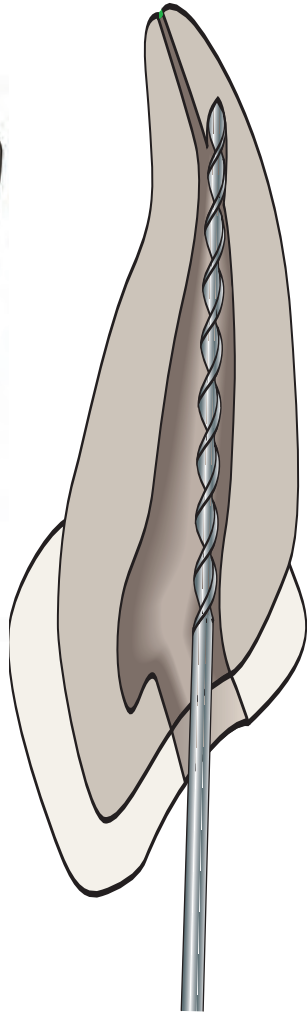


Figure 5-18. Operative error—maxillary anterior teeth. Ledge formation at the apicolabial curve (not discernible in a radiograph) caused again by a failure to complete the convenience extension at the incisal surface and the lingual shoulder.

MANDIBULAR ANTERIOR TEETH

Mandibular anterior teeth also are entered from the lingual surface. The enamel is perforated with a round carbide bur, an end-cutting tapered bur, or a diamond stone, held parallel to the long axis of the tooth (Figure 5-19).

As soon as the pulp chamber is entered, the bur/stone is used to remove tooth structure toward the incisal aspect (Figure 5-20).

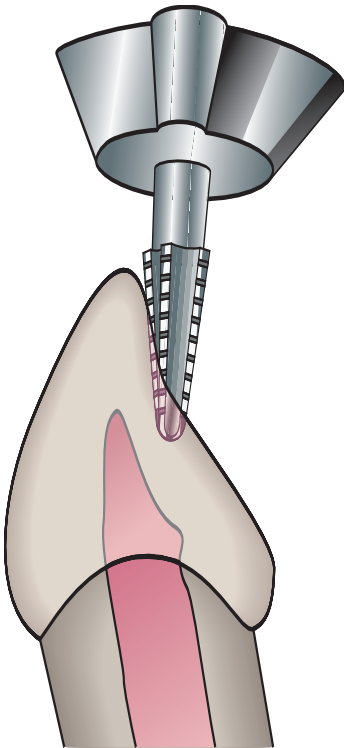


Figure 5-19. Mandibular anterior teeth. Entrance is always gained through the lingual surface. Initial penetration is made in the exact center of the lingual surface. An endcutting, fissure bur is turned to cut at right angles to the lingual surface.

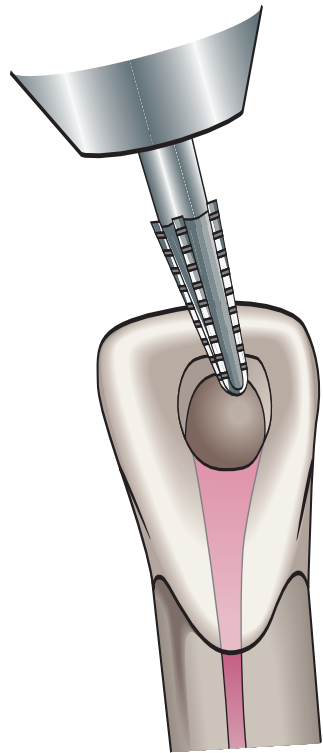


Figure 5-20. Mandibular anterior teeth. As soon as the enamel is penetrated, the bur is turned vertically, beginning the convenience cut toward the incisal. Once enough overhanging structure has been removed, the pulp chamber may be entered vertically with a no. 4 round bur in a slow handpiece.

This convenience removal allows adequate room for the shaft of burs that will enter deeper into the pulp chamber to remove its roof (Figure 5-21).

The final outline form on the lingual surface should reflect the size and shape of the pulp chamber, usually dictated by age (Figure 5-22). The entire outline form, from incisal to apex, must be free of

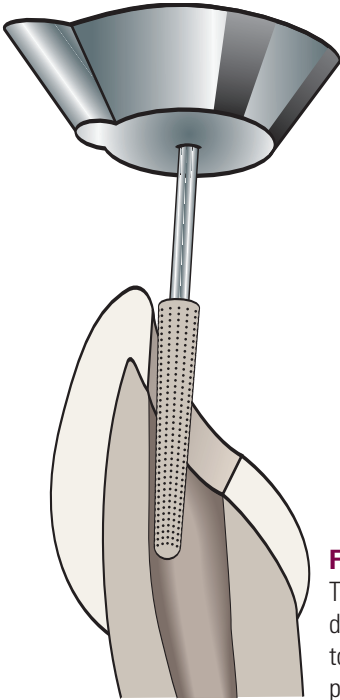


Figure 5-21. Mandibular anterior teeth. The orifice is widened with a fissure bur or a diamond toward the incisal and the shoulder toward the lingual to give a smooth-flowing preparation.

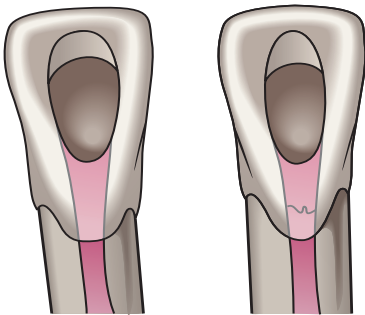


Figure 5-22. Mandibular anterior teeth. Again, the shape of the lingual outline form reflects the size of the pulp chamber, which in turn reflects the age of the patient.

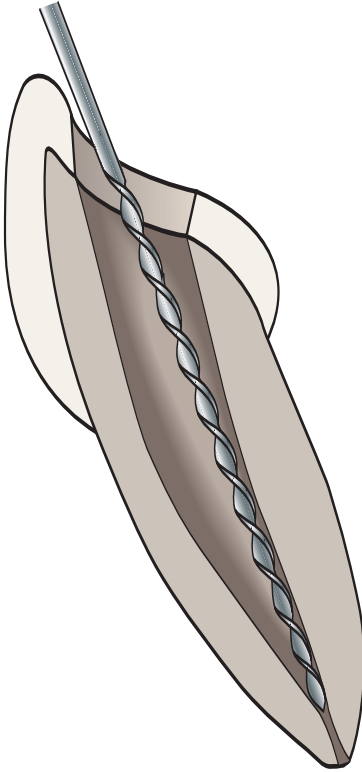


Figure 5-23. Mandibular anterior teeth. Final preparation with the instrument in place. The instrument shaft clears the incisal cavity margin and the reduced lingual shoulder, and penetrates unimpeded to the apex, under complete control of the clinician. Always search for a **second canal to the labial or the lingual** in lower incisors. Virtually the same procedures and precautions apply to mandibular lateral incisors and canines as to central incisors.

any encumbrances that would interfere with cleaning, shaping, or obturation (Figure 5-23).

Operative Errors

The common error of gouging or perforating at the incisogingival aspect (Figure 5-24) is usually due to two factors: not allowing adequate access toward the incisal of the preparation (see Figures 5-20 and 21) or not aligning the bur vertically with the long axis of the tooth (see Figure 5-19).

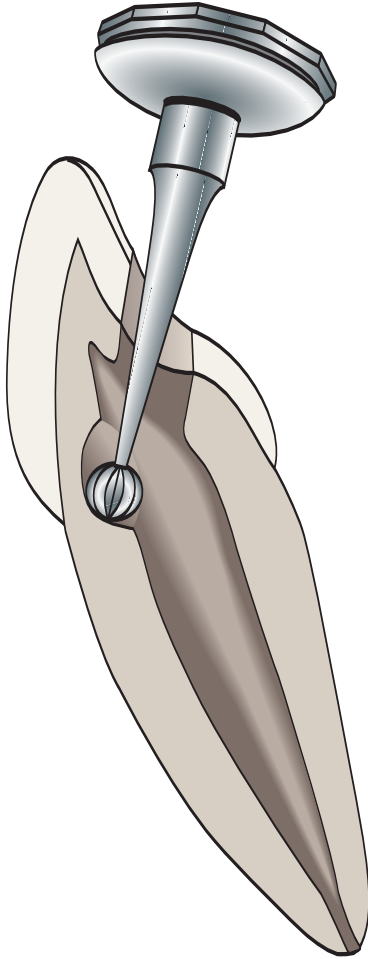


Figure 5-24. Operative errors—mandibular anterior teeth. Inadequate lingual access controls the shaft of the bur and misdirects it to the labial. Cervical perforation can result.

Inadequate access leads to the inability to explore, débride, and obturate the second canal, often not seen in a standard labiolingual radiograph (Figure 5-25).

Never enter the pulp chamber from a proximal surface (Figure 5-26). As inviting as it might appear in some situations, total loss of control of enlarging instruments is the result. Failure looms!

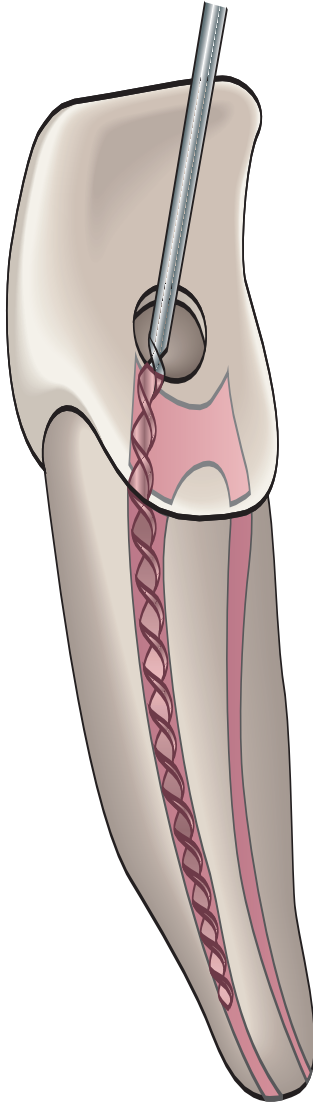


Figure 5-25. Operative errors—mandibular anterior teeth. Again, inadequate access prevents the exploration for a second canal toward the labial. Straight-on radiographs do not reveal this common error.

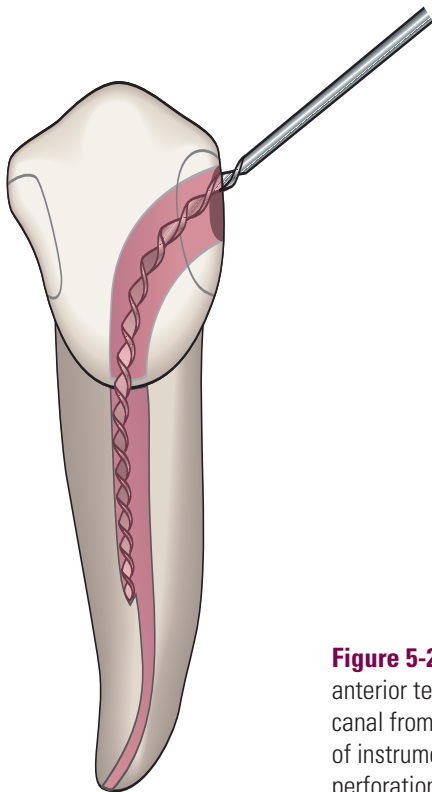


Figure 5-26. Operative errors—mandibular anterior teeth. **Never** attempt to enter the canal from the proximal surface. A total loss of instrument control leads to ledging and/or perforation.

MAXILLARY PREMOLAR TEETH

Entrance is always gained through the occlusal surface of all posterior teeth. The enamel or restoration is perforated in the exact center of the central groove with a round carbide bur, an end-cutting, tapered bur, or a diamond stone held parallel to the long axis of the tooth (Figure 5-27).

Once the chamber is entered, an explorer or endodontic file is used to explore the orifices of the labial and lingual canals of the first premolar or the central canal (or possibly additional canals) in the second premolar (Figure 5-28). From this exploration one learns the necessary extension of the buccolingual outline form. Always probe for the possibility of additional canals in either premolar.

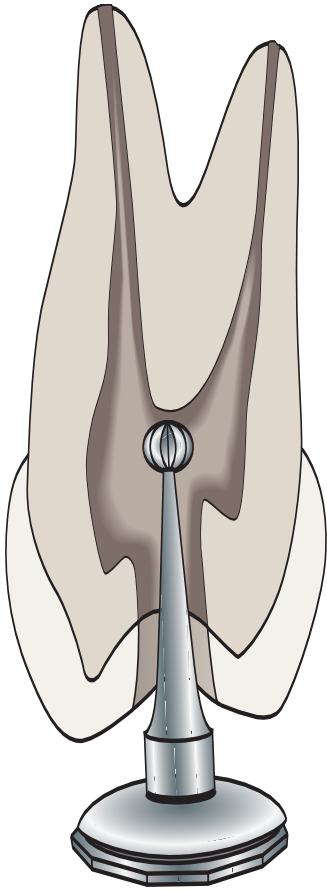


Figure 5-27. Maxillary premolar teeth. Access to all posterior teeth is through the occlusal surface. Initial penetration is made parallel to the long axis of the tooth in the exact center of the central groove. A no. 4 round bur may be used to penetrate into the pulp chamber. The bur will be felt to “drop” when the pulp chamber is reached. If the pulp is well calcified, the drop will not be felt, so the bur penetrates until the nose of the handpiece touches the occlusal surface—9.0 mm. The orifice is then widened to allow exploration for the canal orifices.

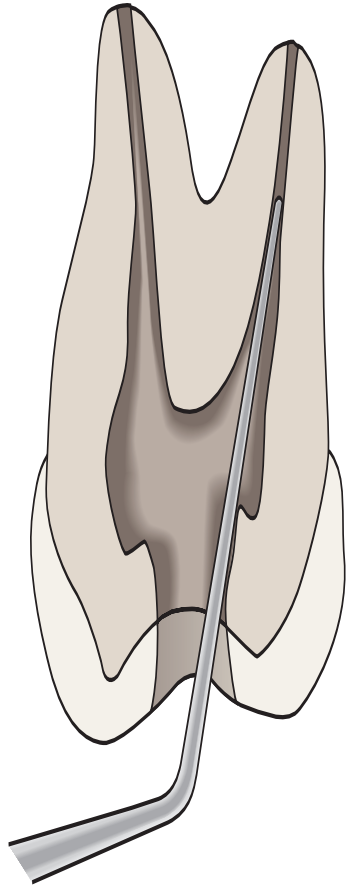


Figure 5-28. Maxillary premolar teeth. An endodontic explorer is used to locate the orifices of the buccal and lingual canals of the first premolar or the central canal of the second premolar. Always search for extra canals—the third canal in the first premolar or a second canal in the second premolar.

Buccolingual cavity extension is best done with tapering fissure burs or stones (Figure 5-29). Adequate buccolingual endodontic cavity outline form is in contrast to the mesiodistal restorative outline form (Figure 5-30).

The final preparation should provide adequate, unimpeded access to all canal orifices (Figure 5-31). Cavity walls should not impede complete authority over the enlarging instruments.

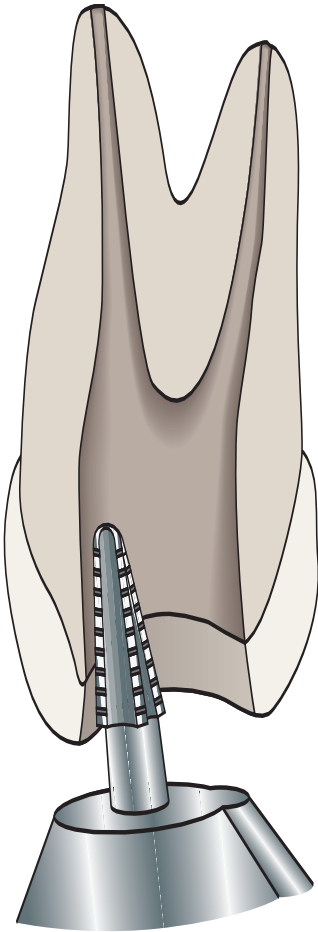


Figure 5-29. Maxillary premolar teeth. The cavity outline is then extended buccolingually with a tapered, fissure bur or diamond.

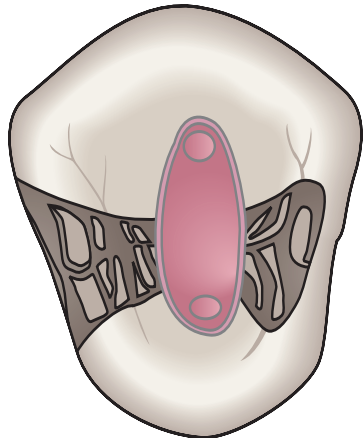


Figure 5-30. Maxillary premolar teeth. The buccolingual preparation reflects the internal anatomy of the pulp chamber and the entrance to the canal orifices.

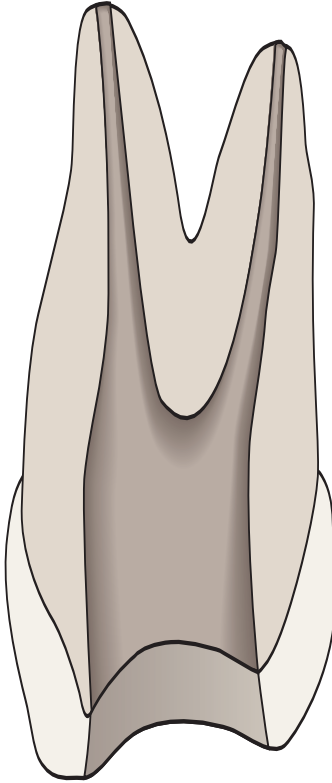


Figure 5-31. Maxillary premolar teeth. Final preparation should provide unobstructed access to the canal orifices.

Operative Errors

An error that occurs in maxillary premolar teeth is overextended preparation in a fruitless search for a receded pulp (Figure 5-32). The white color of the roof of the chamber is a clue that the pulp has not been reached. The floor of the chamber is a dark color.

Failure to observe the mesiodistal inclination of a drifted tooth leads to a gingival perforation owing to the misaligned bur. The receded pulp is missed completely (Figure 5-33).

An instrument can be broken or twisted off in a “crossover” canal (Figure 5-34). Failure may be obviated by extending the internal preparation to straighten the canals.

Inadequate occlusal access leads to the failure to explore, instrument, and obturate a third canal (Figure 5-35).

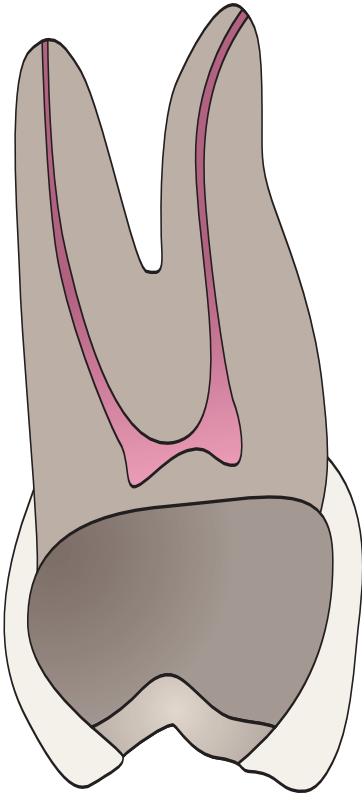


Figure 5-32. Operative errors—maxillary premolars. Overextended preparation from a fruitless search for a receded pulp. The enamel walls have been completely undermined and the tooth hopelessly weakened. Gouging relates to a failure to refer to the radiograph, that clearly shows the depth of pulp recession.

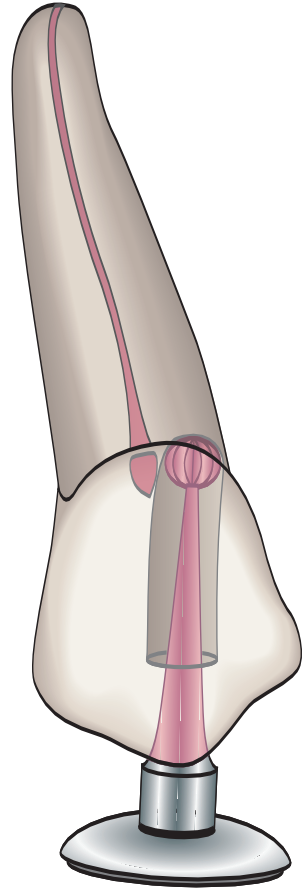


Figure 5-33. Operative errors—maxillary premolars. Perforation at the mesiocervical indentation caused by failure to observe the distoaxial inclination of the tooth. The receded pulp is also completely bypassed. The maxillary first premolar is one of the most frequently perforated teeth.

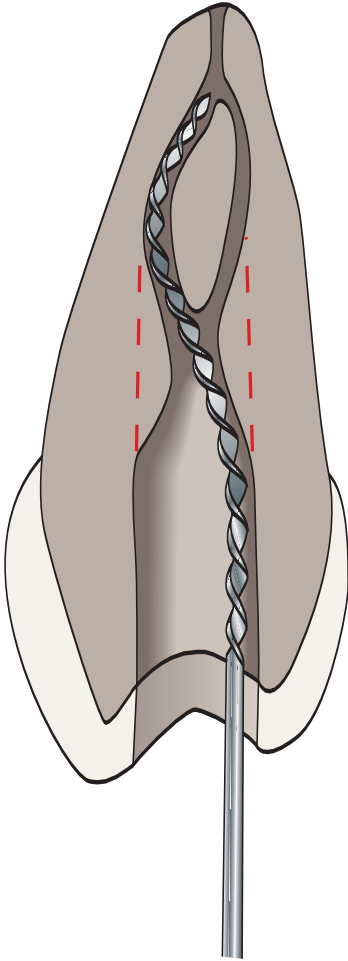


Figure 5-34. Operative errors—maxillary premolars. A broken instrument fractured in a “crossover” canal. This frequent occurrence may be obviated by extending the internal preparation to straighten the canals access (*dotted lines*).

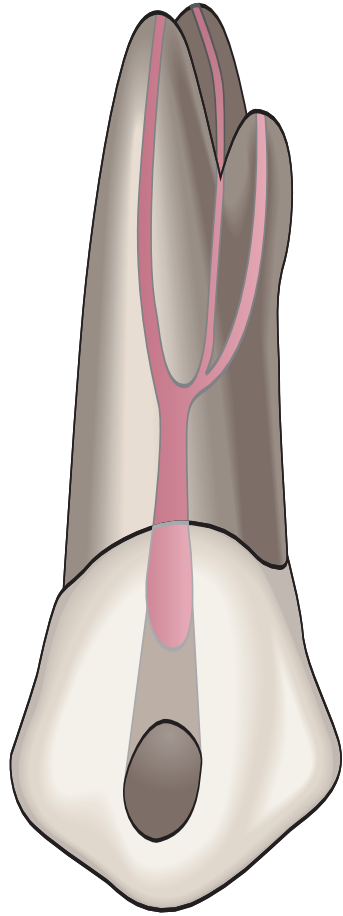


Figure 5-35. Operative errors—maxillary premolars. Inadequate occlusal access leads to the failure to explore, débride, and obturate the third canal (6% of the time), and to find and instrument the second canal in the second premolar (24% of the time).

MANDIBULAR PREMOLAR TEETH

Entrance is gained through the occlusal surface of all posterior teeth. The enamel or restoration is perforated in the exact center of the central ridge with a round carbide bur, an end-cutting, tapered bur, or a diamond stone held parallel to the long axis of the tooth (Figure 5-36).

Once the chamber is entered, an explorer or endodontic file is used to explore the canal and to search for a second canal (Figure 5-37). From this exploration one learns the needed extent

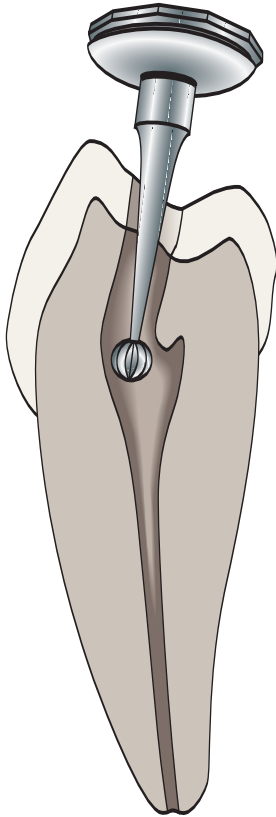


Figure 5-36. Mandibular premolar teeth. Initial penetration is made with a no. 4 round bur in the central groove of mandibular premolars. When the chamber is entered, the bur is felt to “drop” into the space. If the pulp has receded, the bur should cut vertically until the nose of the contra-angle touches the occlusal surface—9.0 mm. In removing the bur, the orifice is widened buccolingually.

of the outline form. Additional canals in lower premolars are more prevalent in African American patients.

Removal of the roof of the pulp chamber and expansion of the occlusal outline form is best done with tapering fissure burs or stones (Figure 5-38).

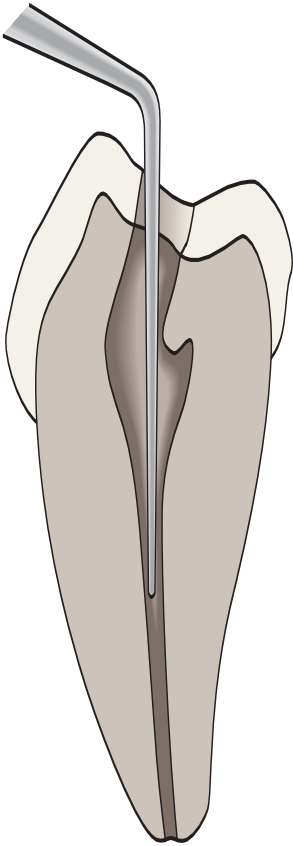


Figure 5-37. Mandibular premolar teeth. An endodontic explorer is used to locate the direction and the extent of the chamber and the central canal. One should also be cautious to search for additional canals, particularly in African American patients, in whom 32.8% of first mandibular premolars have two canals and 7.8% of mandibular second premolars have two canals.

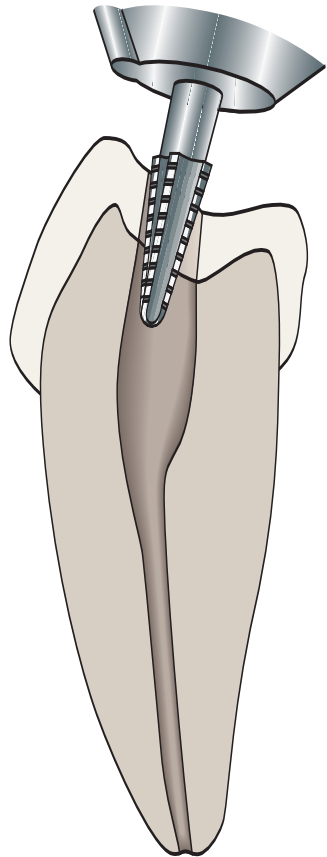


Figure 5-38. Mandibular premolar teeth. Buccolingual extension is completed with a tapered fissure bur or a diamond.

The ovoid outline form reflects the shape of the pulp chamber and must be extensive enough to accommodate instruments and filling materials (Figure 5-39). Search for a second canal, especially in the first premolar.

The final preparation should provide access from the occlusal surface to the apex (Figure 5-40). Cavity walls should not impede complete authority over the enlarging instruments.

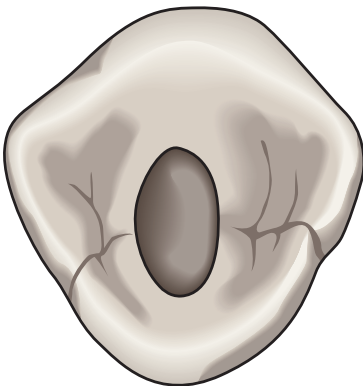


Figure 5-39. Mandibular premolar teeth. Buccolingual ovoid outline form reflects the internal anatomy of the pulp chamber and orifice to the canal.



Figure 5-40. Mandibular premolar teeth. Final preparation is a tapered funnel from the occlusal surface to the canal, providing unobstructed access to the apical third of the canal.

Operative Errors

An error that occurs in mandibular premolar teeth is perforation at the gingiva owing to the failure to recognize the distal tilting of the tooth that often follows extraction of the lower first molar (Figure 5-41). The pulp is missed entirely.

Never enter the pulp from the buccal aspect (Figure 5-42). Total loss of instrument control and imminent separation of the file follows.

Bifurcation of the canal is missed owing to the failure to thoroughly explore the canal in all directions (Figure 5-43).

Perforation at the apical curvature owing to the failure to recognize by exploration the curvature to the buccal aspect (Figure 5-44). A standard buccolingual radiograph does not reveal buccal or lingual curvatures.

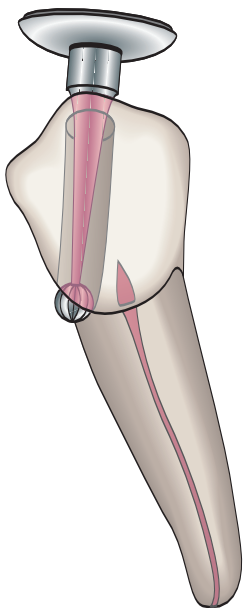


Figure 5-41. Operative errors—mandibular premolars. Perforation at the distolingual aspect caused by a failure to recognize that the premolar has tilted distally. The same error can occur with a mesial tilt.

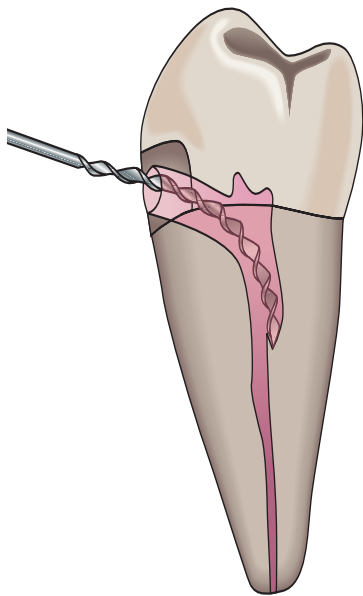


Figure 5-42. Operative errors—mandibular premolars. **Never** enter from the buccal aspect! The instrument is immediately under stress, will cause ledging, develop a pear-shaped preparation, or fracture.

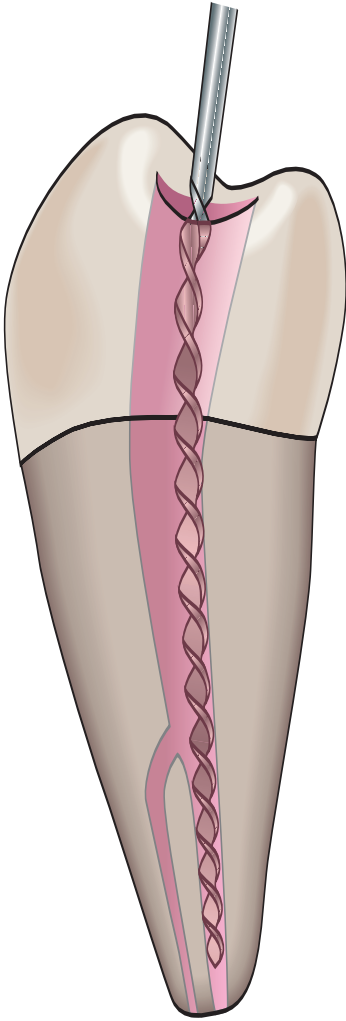


Figure 5-43. Operative errors—mandibular premolars. Bifurcation of the canal is completely missed owing to a failure to adequately explore the canal with a curved instrument.

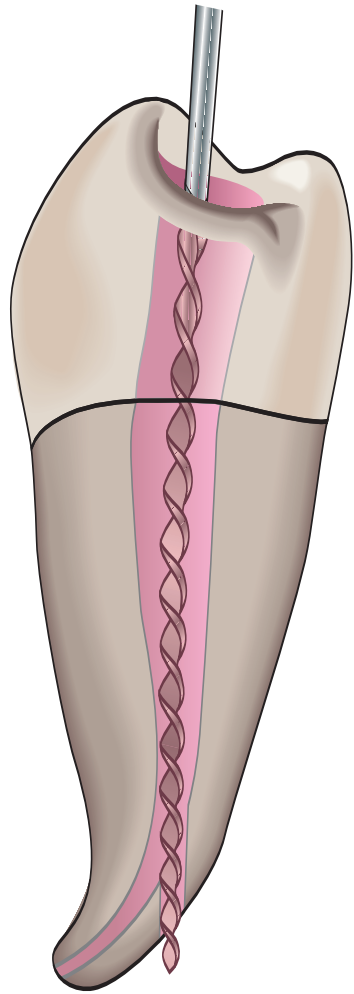


Figure 5-44. Operative errors—mandibular premolars. Perforation at the apical curvature is caused by a failure to recognize, by exploration, buccal curvature. A standard buccolingual radiograph does not show buccal or lingual curvature. One should also be wary of perforating the apical foramen in perfectly straight canals, a common cause of acute apical periodontitis.

MAXILLARY MOLAR TEETH

Molar teeth are always entered through the occlusal surface. Enamel or restorations are best perforated with a round carbide (no. 4) bur, an end-cutting, tapering fissure bur, or a diamond stone. The point of entrance should be the central pit, and the bur should be aimed at the orifice of the palatal canal, the largest canal (Figure 5-45). The same instrument can be used to remove the remaining roof of the pulp chamber.

Once the opening is large enough, the orifices of the canals should be explored with explorers or files (Figure 5-46). In this manner, convenience extension is established.

Final convenience extension is best done with a **non-end-cutting**, tapering fissure bur or stone so as not to nick the floor of the chamber (Figure 5-47).

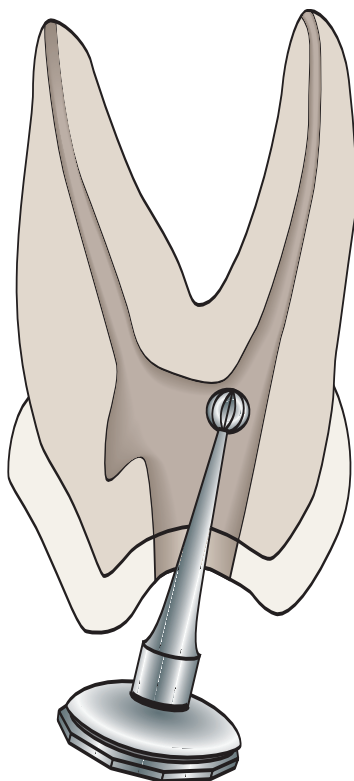


Figure 5-45. Maxillary molar teeth. Molar teeth should all be opened through the occlusal surface. The initial cut is made in the exact center of the central pit. After perforating the enamel or restoration, the bur should be directed toward the palatal canal orifice, the largest canal. Using a round or tapered fissure bur, the occlusal opening should be enlarged so that an endodontic explorer can be used.

The final occlusal outline form reflects not only the size and shape of the pulp chamber but the convenience extensions necessary to free the shafts of the enlarging instruments (Figure 5-48). Do not assume there are only three canals. **One must always probe for extra canals!** Keep in mind, however, that some maxillary second molars may have only two canals.

Outline form, from occlusal to apex, must be free of any encumbrance, allowing unimpeded use of the enlarging instrument (Figure 5-49).

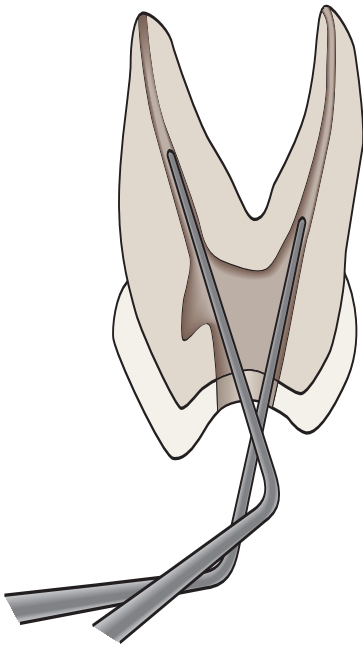


Figure 5-46. Maxillary molar teeth. Using the explorer the floor of the chamber is carefully explored to locate the canal orifices and the direction of the canals so that one knows in which direction to enlarge the outline form.

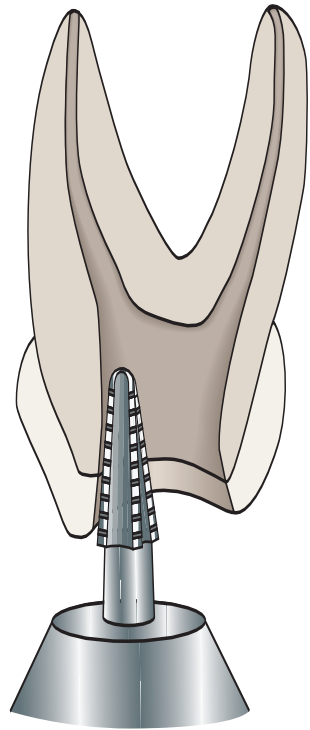


Figure 5-47. Maxillary molar teeth. A tapered fissure bur or diamond is used to remove all the overhanging roof of the chamber and extend the outline form so that enlarging instruments can be used unimpeded.

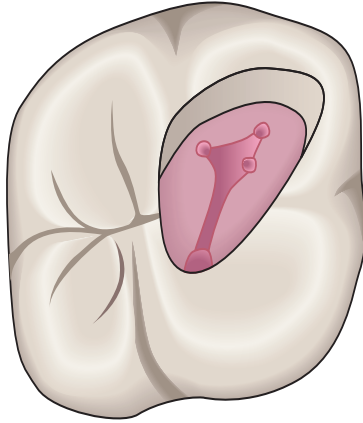


Figure 5-48. Maxillary molar teeth. The outline form can usually be confined to the mesial half of the occlusal aspect and should reflect the internal anatomy of the chamber and the direction of the canals. The preparation is sloped to the buccal aspect to give easier access. One must also search carefully for the possibility of a **fourth canal**, which is present in half of cases. On the other hand, a few second molars may have only two canals.

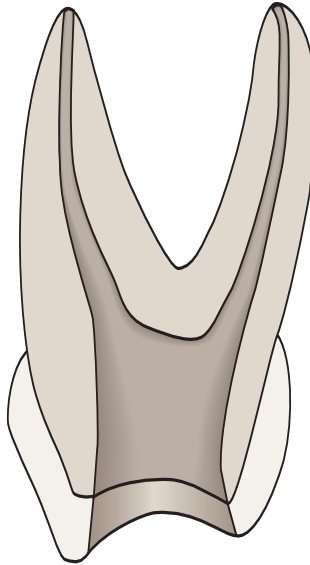


Figure 5-49. Maxillary molar teeth. Since all instrumentation is introduced from the buccal aspect, the cavity is sloped buccally for easier access. Each endodontic instrument, when inserted down the canal, must be unimpeded, free from any interfering wall.

Operative Errors

One of the most common errors that occurs in maxillary molar teeth is perforation into the furcation, using a surgical-length bur and unknowingly passing through the narrow pulp chamber (Figure 5-50). The depth to the chamber should be measured on the radiograph and marked with Dycal on the shaft of the bur.

In an underextended preparation, only the pulp horns are nicked. White-colored dentin is a clue to the underextension (Figure 5-51). The true floor of the chamber is marked by dark-colored dentin.

An imperfect vertical preparation can occur in a molar tipped to the buccal aspect (Figure 5-52). The preparation should be parallel to the true long axis of the tooth.

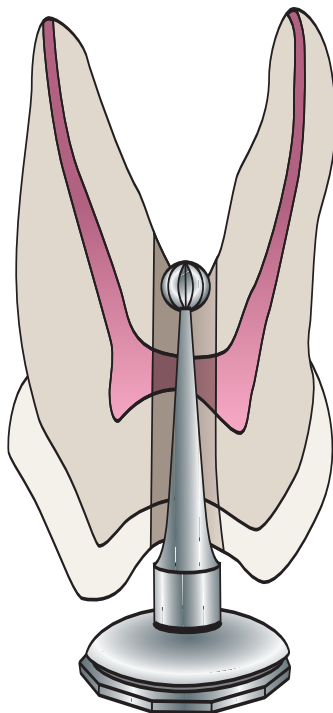


Figure 5-50. Operative errors—maxillary molars. One of the most common errors is perforation of the furcation while searching for a receded pulp with a surgical-length bur. Wider access helps prevent these accidents, as does measuring the depth on the radiograph. These perforations may be repaired with placement of mineral trioxide aggregate (MTA, Dentsply/Tulsa).

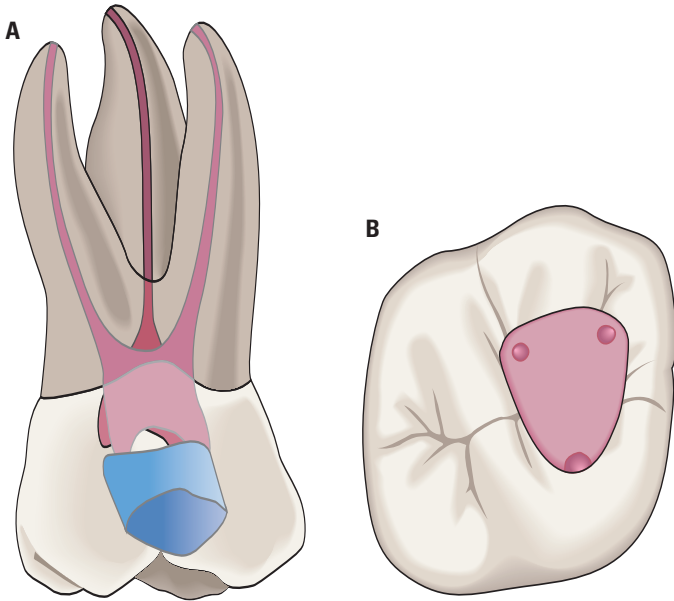


Figure 5-51. Operative errors—maxillary molars. **A**, Underextended preparation. The roof of the pulp chamber has not been removed. The white color of the dentin in contrast to the dark color of the dentin on the floor of the chamber should be the clue. The pulp horns have barely been nicked, and the clinician has assumed that the canal orifices have been located. Total control of the instruments will be lost if instrumentation proceeds through tiny orifices. **B**, Example of the failure to remove the roof of the pulp chamber. One can easily visualize how the interfering tooth structure will control the path of the instrument.

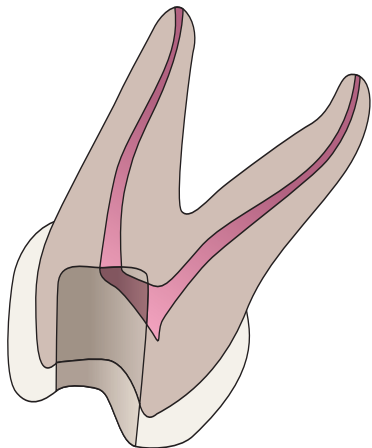


Figure 5-52. Operative errors—maxillary molars. Inadequate vertical preparation related to a failure to recognize the severe buccal inclination of an unopposed molar.

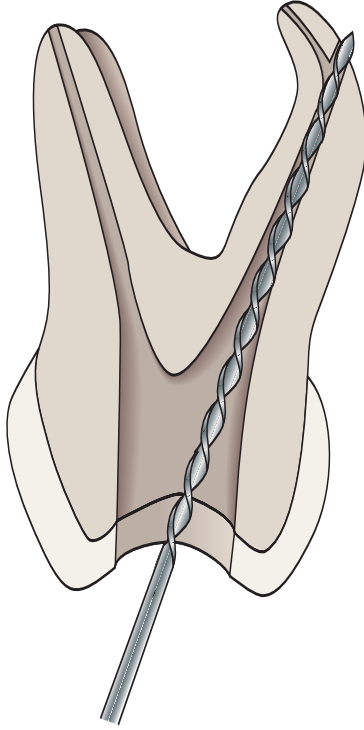


Figure 5-53. Operative errors—maxillary molars. Perforation of a palatal root is commonly caused when the clinician assumes the canal is straight and fails to explore and enlarge the canal with a fine, curved instrument. Remember, roots that curve buccally appear to be straight in buccolingual radiographic projections.

A final error in maxillary molars is perforation of the palatal canal, commonly caused by the assumption that the palatal canal is straight (Figure 5-53). In more cases than not, the palatal canal curves to the buccal aspect, a fact that does not appear in the standard buccolingual radiograph. Careful exploration with fine-curved files should reveal the presence and direction of the curve.

MANDIBULAR MOLAR TEETH

All mandibular molar teeth are entered through the occlusal surface. Initial penetration is made in the exact center of the mesial pit, aimed for the orifice of the distal canal, the largest canal. Round

carbide burs (no. 4), end-cutting, tapering fissure burs, or diamond stones are used to enter the chamber and to remove the roof of the chamber as well (Figure 5-54).

Endodontic explorers or files are used to locate the orifices of the canals and to determine the direction convenience extensions must be made (Figure 5-55). One must carefully explore for a possible fourth canal in the distal root.

Non-end-cutting, tapering fissure burs or stones are best used to expand the outline form to accommodate unimpeded use of the enlarging instruments (Figure 5-56).

The final outline form is dictated by the size and shape of the pulp chamber plus the convenience extensions needed to free the enlarging instruments from interference (Figure 5-57). Severe extensions to the mesial are not uncommon.

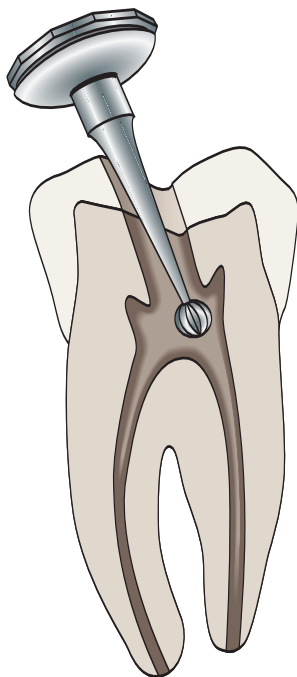


Figure 5-54. Mandibular molar teeth. Entrance is always gained through the occlusal surface of posterior teeth. Initial penetration is made in the exact center of the mesial pit, with the bur directed distally. Once the chamber has been entered, the bur is used to enlarge the opening orifice by cutting away the roof of the pulp chamber. This allows for the entrance of exploring instruments.

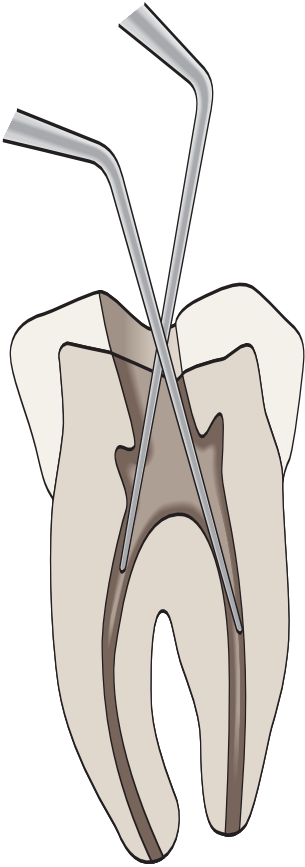


Figure 5-55. Mandibular molar teeth. An endodontic explorer is used to locate the orifices of the distal, mesiobuccal, and mesiolingual canals. Special care is taken to explore for a possible fourth canal in the distal root. Tension on the explorer indicates how much of the walls must be removed to gain unchallenged access to the canals' full length.

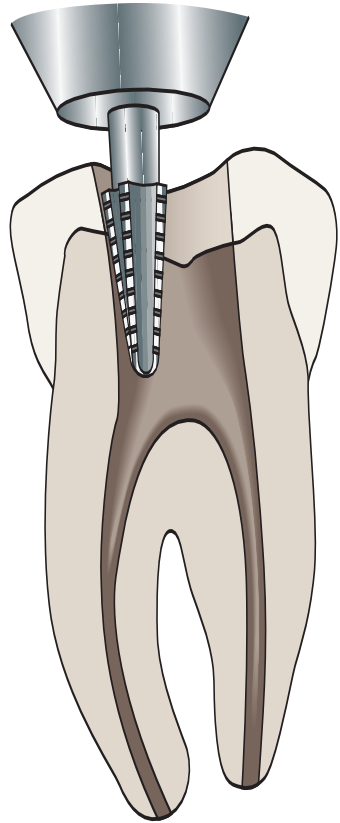


Figure 5-56. Mandibular molar teeth. Final finishing and funneling of the cavity walls are completed with a fissure bur or a diamond.

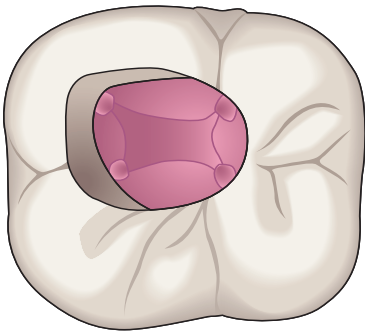


Figure 5-57. Mandibular molar teeth. The “square” outline form reflects the anatomy of the pulp chamber modified for convenience form, mostly flared to the mesial to allow easier access to the mesial canals. Four canals are shown in this illustration.

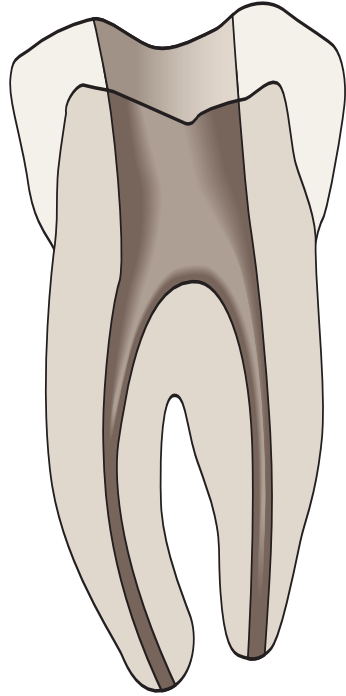


Figure 5-58. Mandibular molar teeth. The final preparation provides unobstructed access to the canal orifices and should not impede the enlarging instruments; there should be a free flow to the apical third of all canals.

The final outline form, from occlusal to apex, provides unobstructed access for all enlarging instruments and filling materials (Figure 5-58).

Operative Errors

Perforation into the furcation is commonly caused by using a surgical-length bur and unknowingly passing through the narrow pulp chamber (Figure 5-59). Depth to the pulp chamber should be measured on the radiograph and marked on the shaft of the bur with Dycal.

Perforation at the mesiogingival aspect is caused by the failure to orient the bur to the long axis of a mandibular molar severely tipped mesially (Figure 5-60).

Failure to locate a second distal canal occurs because of a lack of exploration for a fourth canal hidden by inadequate outline form (Figure 5-61).

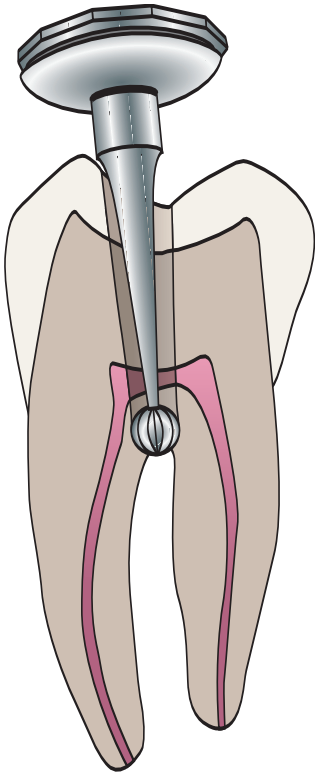


Figure 5-59. Operative errors—mandibular molars. Perforation into the furcation is caused by the use of too long a bur and a failure to recognize on the radiograph the depth of recession of the pulp. The bur should be measured against the radiograph and the depth marked on the shaft. This is a common error.

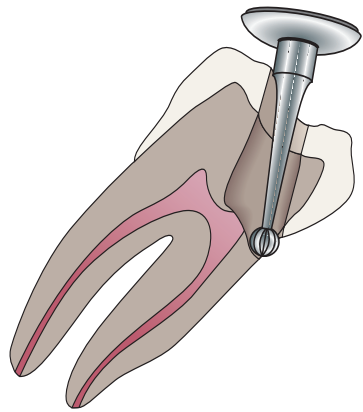


Figure 5-60. Operative errors—mandibular molars. A common error is perforation at the mesiogingival aspect caused by a failure to recognize the severity of the tilt of the lower molar. The bur must be oriented with the long axis of the tooth.

Perforation of a curved, distal root is caused by using a large, straight instrument in a severely curved canal (Figure 5-62). Such a curve should be observed in a radiograph, and fine, curved or flexible instruments should be used.

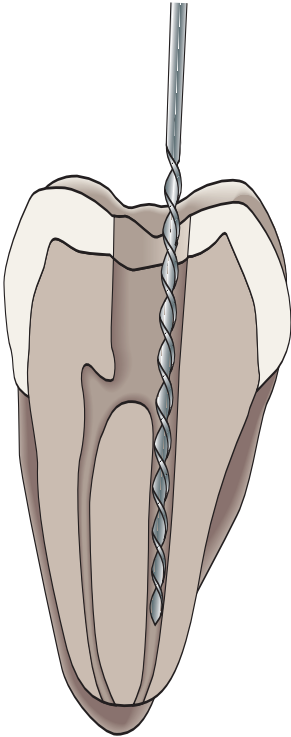


Figure 5-61. Operative errors—mandibular molars. Failure to locate a second distal canal is caused by the overhanging dentin wall and a lack of exploration for a second canal.

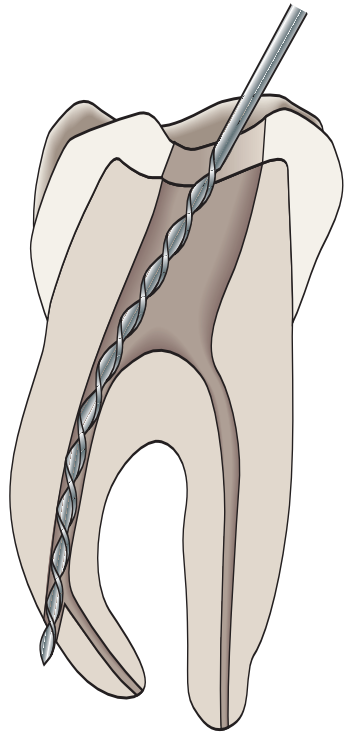


Figure 5-62. Operative errors—mandibular molars. Perforation of the curved, distal root is caused by the use of a large, straight instrument in a large but severely curved canal.

Root Canal Preparation

CLEANING AND SHAPING THE ROOT CANAL

Chapter 5 mentions that preparation of the root canal system is divided into two stages: coronal and radicular preparation. This chapter deals with **radicular preparation** of the root canal system: cleaning and shaping of the canal.

In Chapter 5, outline form was described as not only the access cavity outline on the occlusal or lingual surfaces, but the total shaping: from occlusal/lingual surfaces to the termination of the canal at the apical “portal of exit.” This concept applies to radicular preparation as well (Figure 6-1). Note that instruments pass through unimpeded access, ending at **resistance form**, or an “**apical stop**” (see Figure 6-1B), developed to prevent the passage of instruments beyond this point and to form a barrier against which filling materials may be compacted without overfilling.

The apical stop should be developed slightly short of the apical foramen; 0.5 to 1.0 mm has been suggested. This leaves a tiny remaining portion of the canal not properly cleaned of bacteria and/or debris. This section of the canal should finally be cleaned, not shaped, using fine instruments, no. 10 or no. 15, to the radiographic apex. This is known as **establishing apical patency**. In an adult patient the natural portal of exit is usually no larger than a no. 30 instrument. Final irrigation follows, and is most important.

Basic Endodontic Instruments

Before detailing the methods of cleaning and shaping the root canal, a discussion of the instruments to be used is in order. The tapered and twisted shape of today’s instruments was developed over a century ago (Figure 6-2). It was not until 1958, however, that standardized sizes and shapes of the basic files and reamers were

established.¹ The sizes of the instruments were all based on measurements at the tip in **hundredths** of a millimeter—10 through 100. The tapered shape was established as a 0.02 mm gain per millimeter advance, from tip to 16 mm of cutting blades, a 0.32 mm gain in diameter size overall (Figure 6-3). These sizes and shapes have since been accepted by the International Standards Organization (ISO). Since then, this 0.02 mm taper has been expanded in instruments

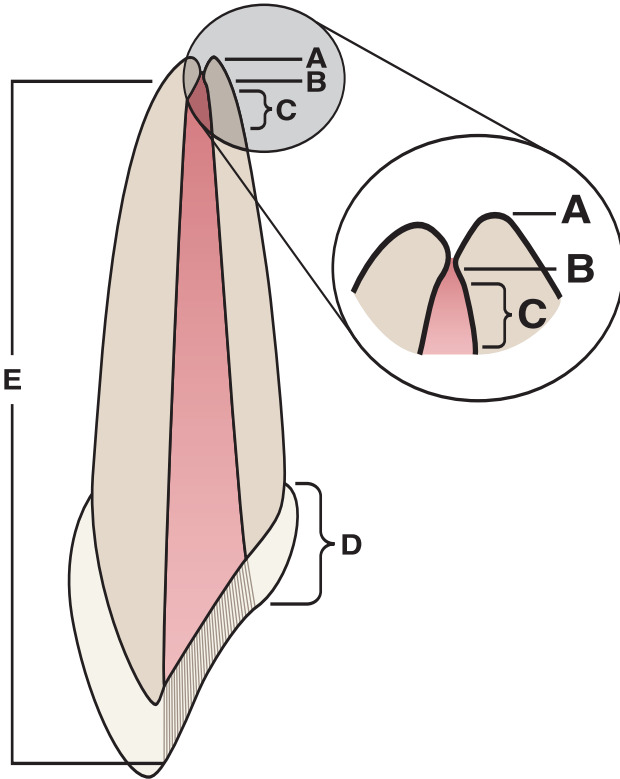


Figure 6-1. **A**, Concept of total endodontic cavity preparation, coronal and radicular as a continuum, based on Black's principles. Beginning at the apex: **A**, radiographic apex; **B**, **resistance form**—development of the “apical stop” at or near the cementodentinal junction; **C**, **retention form** to retain the primary filling point; **D**, **convenience form**, subject to revision to accommodate larger, less flexible instruments and to change the external **E**, **outline form**—basic preparation throughout its length dictated by the canal anatomy. **Continued on next page.**

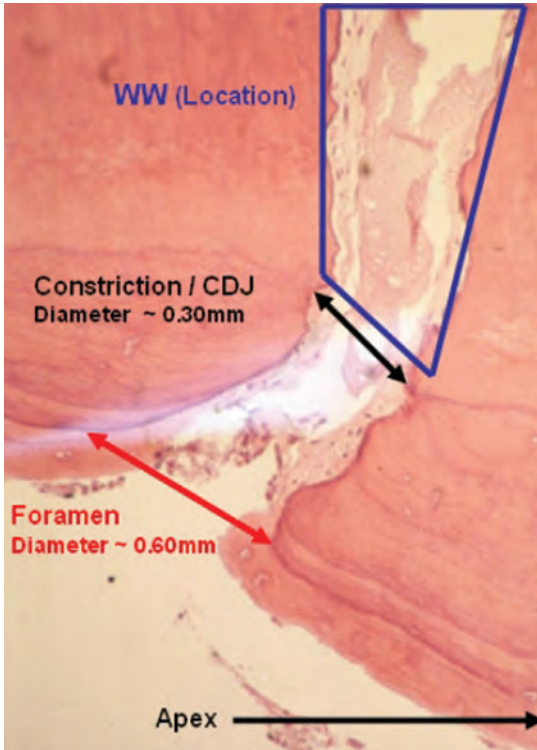


Figure 6-1. Continued. B, Excellent example of the apical anatomy with the minor terminus (ie, apical stop) and the radiographic terminus. The space between must be cleaned before filling. Courtesy, Dr. Steve Senia

Kerr Broach



Magnified Many times.
Note the triple cutting edge

Figure 6-2. Historical illustration, circa 1904, of the original Kerr reamer (titled a broach at that time), the origin of today's K-style instruments. Reproduced with permission from Kerr Dental Manufacturing Co. 1904 catalog.

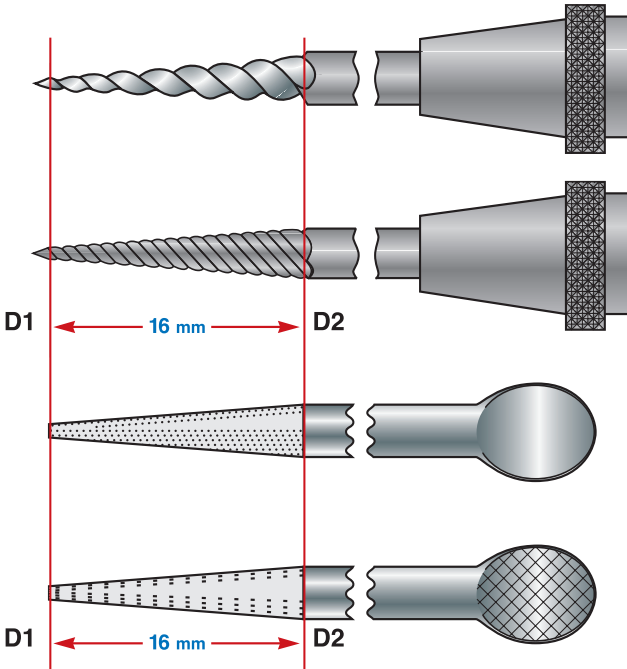


Figure 6-3. Original recommendation for standardized instruments. The number of the instrument is determined by the diameter size at D1 in hundredths of millimeters. Diameter 2 (D2) is uniformly 0.32 mm greater than D1, a gain of .02 mm/1 mm of cutting blades.

that gain 0.04, 0.06, 0.08 mm/1 mm of cutting blade. Handles of the instruments have also been color coded.

Most endodontic hand instruments can be divided into files or reamers. Files are usually used in an up-and-down rasping motion. Reamers are most effective when used in a twisting or drilling motion (Figure 6-4). Today many so-called files are actually reamers and are used in a reaming action, not as rasps. Both files and reamers are made by grinding sizes of stainless steel or nickel titanium piano wire into tapered square, triangular, or rhomboid cross-sections, and then twisting or grinding these pieces into files or reamers. “K” files have 33% more twists (blades) than reamers.

The traditional shape of files and reamers is the so-called K-style design. There have now been a number of variations of K-style

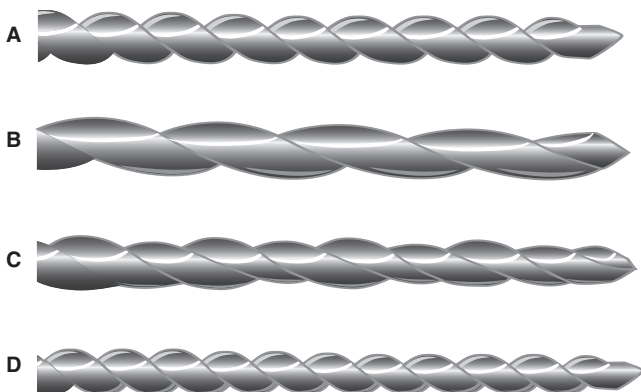


Figure 6-4. International Standards Organization Group I, K-style endodontic instruments. **A**, K-style file. **B**, K-style reamer. **C**, K-Flex file. **D**, Triple-Flex file with tip modification.

instruments—the diamond-shaped **K-Flex** and **Triple-Flex** files (Figure 6-5) (Sybron Endo, Orange, CA). The diamond shaped cross section of the K-Flex files presents 2 very sharp cutting blades (rakes) and 2 low non-cutting blades to collect debris. **Flex-R** files (Moyco Union Broach, York, PA). are much the same design with a non-cutting tip. The **H-style Hedstrom files** are an entirely different design. Shaped like a wood screw, they are used only in a rasping/filing motion (Figure 6-6).

Another popular cross-sectional instrument design is U-shaped and is found in **ProFiles** and **GT Files**. (Dentsply/Tulsa Dental, Tulsa, OK). Dr Steve Buchanan developed the GT series including the new GTX series. The GTX series uses the new **M-Wire** nickel-titanium (NiTi; Dentsply/Tulsa), created through an “innovative thermal-treatment process” that produces a “more flexible tougher product, with greater resistance to cyclic fatigue.” In comparison with the new **Twisted File**, (TF) (Sybron Dental, Orange, CA) (twisted NiTi rather than ground NiTi) and the **Endo Sequence** (ES) NiTi, (Brassler, Savannah, GA) the GTX with M-Wire was far less likely to fail due to cyclic fatigue (2) (Figure 6-7).

Recently Brassler introduced **EndoSequence files**, Sybron Endo introduced **NiTi Twisted files** and Tulsa Dental introduced **ProFile Vortex files**. All are relatively flexible, available in .04 and .06 **ta-pers** of triangular cross sectional shape.

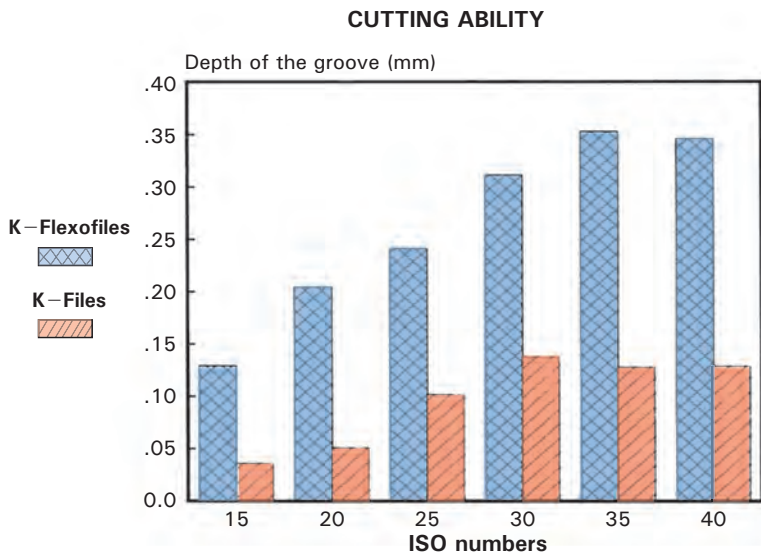


Figure 6-5. Comparison of two competing brands of endodontic instruments, showing widely different cutting ability related to the depth of the blade groove.

The three files differ in pitch and helical angle. The Sybron-Twisted file has a fixed pitch, that varies some, and a helical angle of 35 degrees. The Brassler Endo Sequence file also has a fixed pitch and a helical angle of 15 degrees, whereas the Tulsa ProFile Vortex has a **variable pitch** and a variable helical angle the length of the blade. It begins at 14 degrees at the “hip” and increases to 22 degrees at the tip. This one feature makes the Vortex different from the other two new instruments (Figure 6-8). The other feature is the improved M Wire NiTi (Tulsa) that provides more flexibility and a cyclic fatigue-life of 1.5 to 3.0 times that of the EndoSequence.²

Combining these two improvements makes for a smoother insertion to the apex for the Vortex, with very little vibration and greater efficiency.

The Vortex instruments were developed by Drs. David Clement and Ben Johnson and were introduced at the 2009 meeting of the AAE.

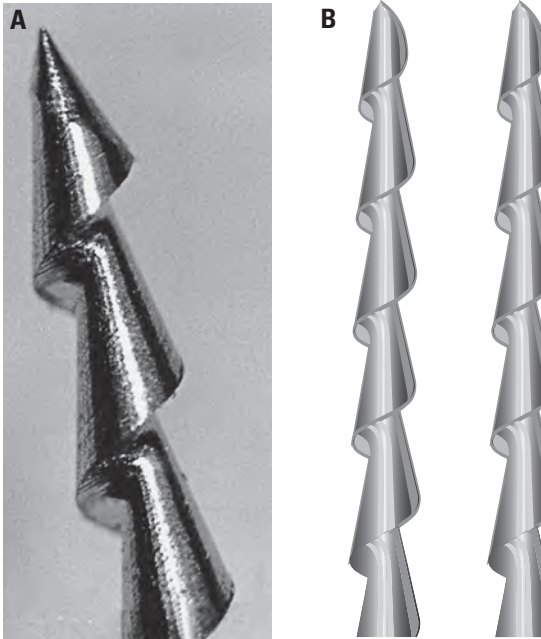


Figure 6-6. International Standards Organization Group I, H-style instruments. **A**, Mailefer Hedstrom file, resembling a wood screw. Courtesy of K.C. Keate and M. Wong. **B**, Modified Hedstrom file (*left*) with noncutting tip. "Safety Hedstrom" (*right*) with flattened noncutting side to prevent "stripping."

The **K3** (Sybron Dental) comes only in NiTi and as rotary instruments. In cross-section they are shaped with three blades, much like a wood router, each blade with a sharp positive rake angle and a shaft of variable flute angle (Figure 6-9).

NiTi has proved to be the most effective material for rotary instruments because of its flexibility, it is better able to conform to canal curvature and to resist fracture, and it wears less than stainless files.

Another nickel-titanium instrument of entirely different configuration is the **LightSpeed LSX** (Figure 6-10) (Discus Dental Culver City, CA). The LSX maximizes flexibility and allows larger apical preparations without unnecessary removal of canal dentin. It is noted for its ability to prepare an ovoid canal into a round shape.

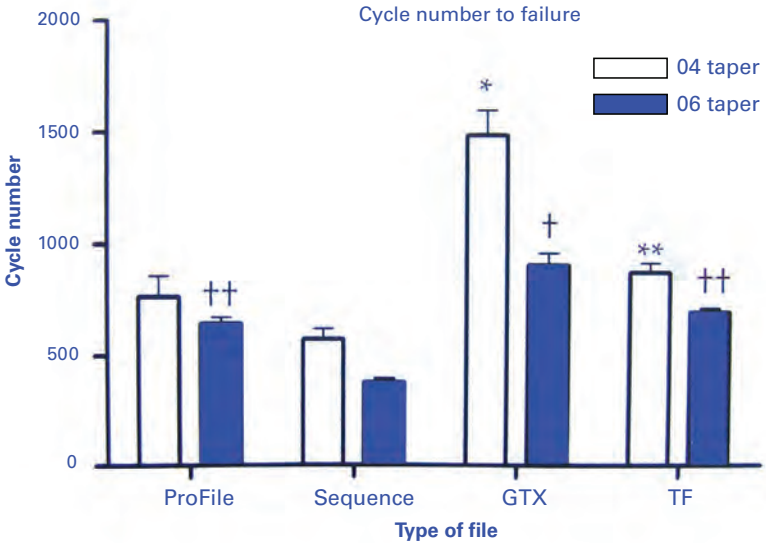


Figure 6-7. The number of rotations to failure for each instrument type. *Significantly higher than the other .04 file types. **Significantly higher than the .04 ES files. †Significantly higher than all the other .06 file types. ††Significantly higher than the other .06 ES files. Larsen CM, He J, Glickman GN, Watanabe I. J Endod 2009;35 (3): 401.



Figure 6-8. Comparison in pitch and helical angle in 3 comparable instruments: **A**, Sybron Twisted File (TF)—constant pitch and constant helical angle. **B**, Dentsply/Tulsa Profile Vortex—variable pitch and variable helical angle. **C**, Brassler-EndoSequence file—constant pitch and varied helical angle.



Figure 6-9. Cross-section of the K3 (SybronEndo) instruments that come only in nickel-titanium and as rotary instruments. The three blades are shaped much like a wood router, each blade with a sharp rake angle. The “tail” of two of the blades is ground short of the cavity wall, providing room for debris collection.

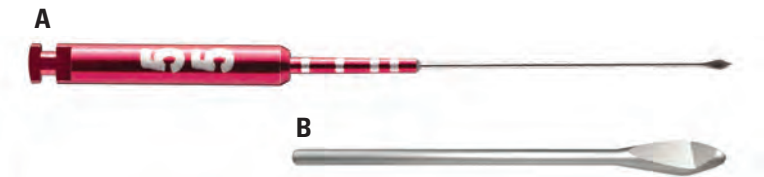


Figure 6-10. LightSpeed LSX apical instrument (Discus Dental). **A**, Note the long “unbladed” shaft and the small cutting head. **B**, Enlargement of the blade and unique cutting head. Courtesy, Discus Dental.

Gates-Glidden and **Peeso drills** are also power-driven endodontic instruments used primarily to enhance canal orifices and the coronal third of canals. Peeso drills are used as well to prepare canals for post placement. The new **LA Axxess burs** (SybronEndo) are used to improve access in straight canals, providing a widened file

path. Since all these burs and drills are rigid, they cannot be used in curved canals.

Contra-angle handpieces used to power NiTi instruments must be both speed and torsion controlled. Speeds vary from 300 rpm for **NiTi ProFiles** to 2,500 rpm for **LightSpeed** instruments.

An entirely new approach for rotary handpieces is the **Morita Tri Auto ZX** cordless endodontic handpiece and the newer **Dentaport ZX** (J. Morita, Irvine, CA) (Figure 6-11). Both have built-in apex locators that warn the clinician when the apex is being approached.

Irrigation

Before one begins using instruments in the necrotic root canal, it should be cleaned of the mass of gelatinous material comprised of pulp remnants, bacteria, and tissue fluids (Figure 6-12). Copious lavage with an irrigant eliminates most of this toxic infected material. The irrigant of choice is **sodium hypochlorite (NaOCl)**—plain, old household bleach (Clorox, Purex). NaOCl is not only a lubricant and disinfectant, it is a tissue dissolvent as well. Full-strength NaOCl (6.0%) dissolves both necrotic tissue and vital pulp remnants. Half-strength NaOCl (3.0%) dissolves only necrotic tissue. However, both are effective bactericides.

Irrigation should also follow the use of each instrument, and NaOCl should then be left in the canal to act as a lubricant for the next instrument. Most instruments cut better in a wet environment than a dry one. Sodium hypochlorite, combined with other irrigants, has often been recommended. NaOCl followed by **hydrogen peroxide** produces a débriding, foaming action. NaOCl followed by **chlorhexidine gluconate** destroys half again as many bacteria as NaOCl alone. They should not be mixed, however, because a brown precipitate forms that may stain the tooth.

Irrigation with NaOCl is accomplished with a syringe and specially designed needles. The concern with this approach is the possible extrusion of the liquid out the apical foramen. **The needle must always be loose in the canal.** Furthermore, the use of special needles designed to deflect the irrigant away from the foramen is imperative. **ProRinse needles** (Dentsply/Tulsa Dental) have been shown to be effective in canal cleansing as opposed to regular, sharp injection needles or notched needle tips (Figure 6-13).

Figure 6-11. A, The Tri Auto ZX cordless endodontic handpiece (J. Morita, USA) has a built-in apex locator, giving the clinician the capability to electronically monitor the canal, particularly its length, before, during, and after instrumentation. It has controllable torque settings and three automatic functions: auto start/stop, auto apical reverse, and auto torque reverse. **B,** The Dentaport ZX (J. Morita, USA) is the newest version of the electric handpiece with a built-in apex locator. The Dentaport is a low-speed, lightweight, compact handpiece connected to the easy-to-read Root ZX apex locator. As with the Tri Auto ZX, the rotary-powered instruments are monitored as they advance down the canal to a working length, thus avoiding apical perforation or underpreparation.





Figure 6-12. Gelatinous mass of necrotic debris should be eliminated from the pulp canal before instrumentation. Forcing this noxious and infected material through the apical foramen leads to acute apical periodontitis and/or an acute apical abscess.

Irrigation fluid should be in the canal during the use of all instruments, and copious irrigation should follow the use of each instrument. Hopefully, extended final irrigation should remove all loose debris from the canal. Removal of the smear layer just before filling enhances the final result.

All in all, irrigation is an important component of proper root canal preparation—**chemomechanical preparation**, if you will.

Exploration for the Canal Orifice

Locating the orifices to the canals can often be frustrating and time consuming, especially in older patients. Probing with an endodontic explorer in the areas where the orifices are expected to be and searching for extra canals, for example, the fourth canal in molars, is the first order of business. Bright illumination and magnification



Figure 6-13. ProRinse irrigating needles (Dentsply/Tulsa Dental) irrigate through a side vent. By rotating the needle in the canal, the douching spray reaches all regions of the canal. This eliminates the water-cannon effect of open-end needles. Courtesy of Dentsply/Tulsa Dental.

loupes, the surgical microscope, or a fiber optic **Orascope** or a rigid **Endoscope** (Jedmed, St Louis, MO) are invaluable (Figure 6-14).

Once located, fine instruments such as an .02 no. 10 should be inserted into the canals to establish their patency and the direction of curve. The cavity walls should then be extended to provide direct unimpeded access to the apex.

Flaring the orifice to provide easier access is often necessary. This can be done with hand instruments such as the **Micro-Opener** (Dentsply/Maillefer), rotary instruments such as Gates-



Figure 6-14. The **Endoscope** (Jedmed Co.) greatly magnifies images of either cavity preparations or apical surgery sites on a monitor screen. Intrachamber and intracanal views are gained with the 0.8 mm Orascope handpiece. Through this increased magnification and illumination, hard-to-find and fourth canals readily become visible. Surgical images can be captured with the 4.0 mm Endoscope and can be magnified up to 50 times. Illumination is provided by a 60 W halide (arc) lamp that issues 5,400° Kelvin. Screen images can also be captured with an attached camera.

Glidden drills or 0.04 or 0.06 mm taper files, the LightSpeed CRX and MRX instruments, (Figure 6-15), or sonic handpiece powered files. Once the orifice has been expanded, the entire canal may be explored.

Exploration of the Canal

Next, the full length of the canal should be explored with stainless steel files, following the curves shown in radiographs. The tip of the exploring instrument should be curved so that it progresses easily down the canal, past unknown curves and obstructions. Straight instruments catch at these points (Figure 6-16). Lubricants such as liquid soap, **ProLube** and **Glide File Prep** (Dentsply/Maillefer), and **RC-Prep** are helpful. The apex should be avoided in order not to force toxic debris out the exit. Once the extent and

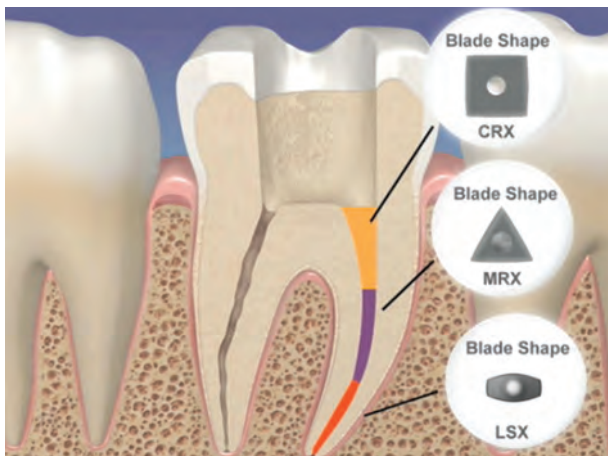


Figure 6-15. LightSpeed Trio. The LightSpeed CRX (*square cross section*) is used to prepare the coronal third of the canal. The LightSpeed MRX (*triangular cross section*) is used to prepare the middle third of the canal. The LightSpeed LSX (*oval cross section*) is used for final preparation of apical third of the canal into the round shape for which is noted. Courtesy of Discus Dental.

direction of the canal are established, initial irrigation should begin as cited above.

Determining the Working Length

Before cleaning and shaping are undertaken, the length of each canal must be established. This can be done radiographically or electronically. One must first establish in one's own mind just where the preparation and obturation of the canal should terminate. It has long been suggested that the **minor diameter** at the cementodentinal junction is often the narrowest site of the apical foramen, the **apical constriction**, and this is where the apical stop should be established. Measurements have shown this site to be from 0.5 to 1.0 mm from the **major diameter**, the radiographic apex (Figure 6-17).

So, if the full length of the canal is determined, 0.5 to 1.0 mm should be subtracted to stay within the confines of the canal and terminate at its narrowest point (Figure 6-18). This should be the **working length**. Some would argue that preparation and filling should extend past this site to the full length at the portal of exit or even beyond, a slight overfilling or apical "puff."

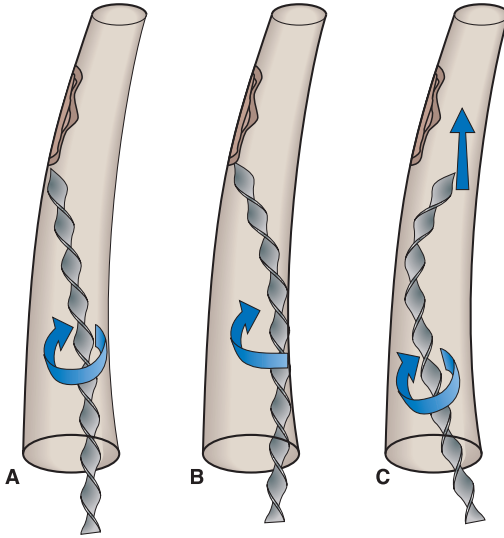


Figure 6-16. *A*, When a straight instrument catches on a curve or obstruction, turning the instrument merely drives the point deeper into the obstruction. *B*, and *C*, When a curved instrument catches on an obstruction, rotating the point of the instrument frees it from the obstruction, allowing the instrument to be moved up the canal

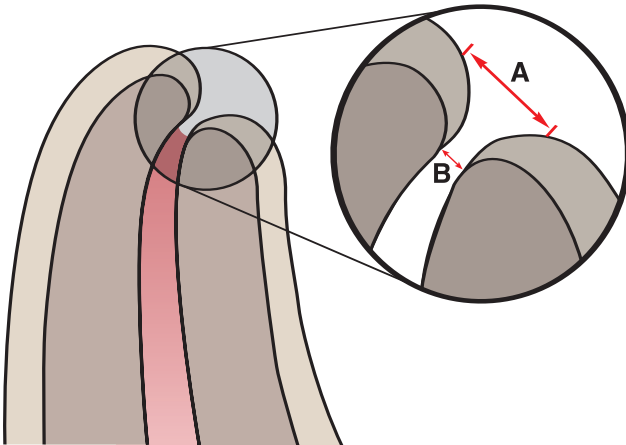


Figure 6-17. Diagrammatic view of the periapex. The importance of differentiating between the minor diameter *B* ("apical stop") and the major diameter (radio-graphic apex) *A* is apparent. Note that the minor diameter approximates but does not necessarily coincide with the cemento-enamel junction. Courtesy of S. Weeks.

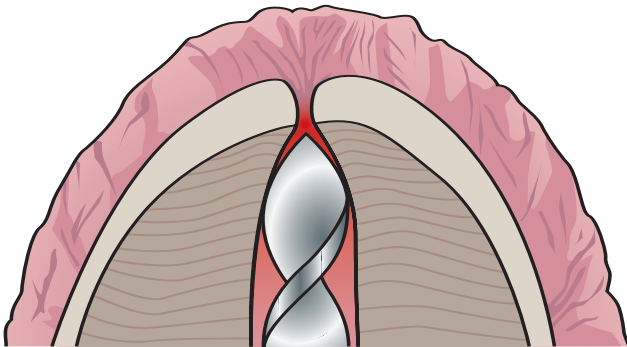


Figure 6-18. Apical limitations of instrumentation should be at the apical constriction that is about 0.5 to 1.0 mm from the end of the root.

Radiographic determination is done by measuring the length of the root on the radiograph and then, to be on the safe side, subtracting millimeters from this estimate (see Figure 6-19). A file, with a stop in place, is then set at this tentative length and inserted into the canal. A radiograph is taken and the measurement from the tip of the file to the apex is added to the known length of the file that was in the canal. These two numbers add up to the full length of the root; then 0.5 or 1.0 mm is subtracted from this total. This is the **working length** (Figure 6-19). During cleaning, shaping, and obturation, no instrument or filling material should exceed this length, except during the final cleaning beyond the apical constriction with a tiny instrument.

Weine has suggested that if **bone resorption** is radiographically apparent at the apex, 1.5 mm should be subtracted, because there is probably some unseen **root resorption**.³ If **both bone and root** resorption appear in the radiograph, 2.0 mm should be subtracted to ensure that one stays within the canal (Figure 6-20).³

Electronic determination is done with **electric apex locators**; there are a number on the worldwide market. The third- and fourth-generation locators all do essentially the same thing. They measure electric impedance, not frequency, as did earlier models. Hence, they function in the presence of electrolytes such as sodium hypochlorite. They all are battery powered. Price and warranty are often the only differences, ranging from \$549 to \$995 (US) and from a 1- to a 3-year warranty.

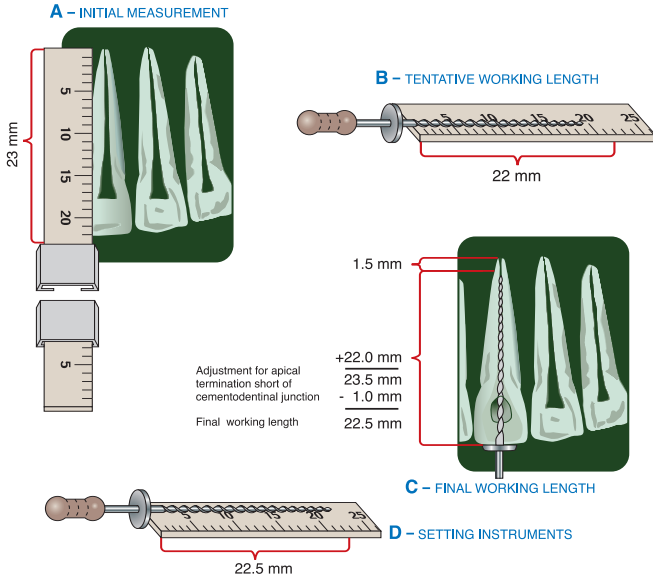


Figure 6-19. Radiographic method of establishing tooth length. **A**, The tooth is measured on a good radiograph. In this **illustrative** case the tooth appears to be 23 mm long. **B**, Tentative working length. As a safety factor, allowing for image distortion, subtract at least 1.0 mm for a tentative working length of 22 mm, and place an instrument stop. **C**, Final working length. The instrument is positioned in the canal to the stop attachment and is radiographed. The tip of the instrument appears to be 1.5 mm short of the apex. This 1.5 mm is added to the 22 mm for a total tooth length of 23.5 mm. **D**, A "safety factor" of 1.0 mm is subtracted, and the working length is 22.5 mm. The instrument stop is readjusted; nothing should exceed this length.

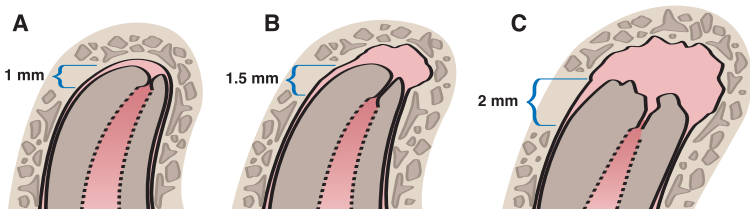


Figure 6-20. Weine's recommendation for determining working length based on radiographic evidence of root/bone resorption. **A**, If no root or bone resorption is evident, the preparation should terminate 1.0 mm from the apical foramen. **B**, If bone resorption is apparent but no root resorption, shorten the length by 1.5 mm. **C**, If both root and bone resorption are apparent, shorten the length by 2.0 mm. Courtesy of F. Weine.

The leaders in the field are **Elements Diagnostic Unit** and **Apex Finder A.F.A.** (SybronEndo), **Endex** and **Endex Plus** (Osada, Inc.), **Root ZX II Apex Locator** (J. Morita, USA), **Foramatron D10** (Parkell Co.), the **ProPex Unit** (Dentsply/Tulsa, and the tiny **Apex NRG** (Medidenta and/or EndoTechnic, San Diego) (Figure 6-21). Some models are combined with electric pulp testers, such as the **Sybron-Endo, Analyzer**, model 8500.

All of these third- and fourth-generation apex locators are accurate to within 0.5 mm in the high ninetieth percentile. They all are small and portable. All have highly visible screens showing the advance of the instrument down the canal, and they all have warning signals, either visual or auditory, that warn when the apex is being approached. All function in the presence of NaOCl, blood, pus, and saline. The locators function with a lip electrode and a canal electrode that is an attached endodontic file. One would be wise to shop for the best price, for one model is half the cost of the others and has a 3-year guarantee.

To use apex locators, a clip carrying up to 1 KHz of alternating current is placed on the patient's lip and an endodontic file carrying 5 KHz is inserted into the canal. As the file proceeds down the canal, the impedance between the two sites is converted to a readable scale, and when the minor diameter is reached, indicators—visual, sound, or light—announce the site. The instrument-stop on the file is then readjusted, the file is withdrawn, and working length (minus the 0.5–1.0 mm safety factor) is measured and recorded. This is then repeated with each canal.

All methods or devices for measuring root length admit to being slightly inaccurate, at least 0.5 mm plus or minus. For that reason it is recommended that length be cross-checked. A recent study at the Oregon School of Dentistry, compared the accuracy of the Morita Root ZX versus the SybronEndo Analyzer, model 8500.⁴ Investigators found that the Root ZX was able to predictably locate the minor diameter (± 0.5 mm) with 90.7% accuracy, whereas the Sybron Analyzer had 34.4% accuracy.

If the initial length is established on a radiograph, an apex locator should verify the length, and vice versa. One should not fall into the trap of believing that the radiographic length of the tooth is the actual length of the canal terminus. All too often the canal exits at the side of the root, short of the full length of the tooth (Figure 6-22).

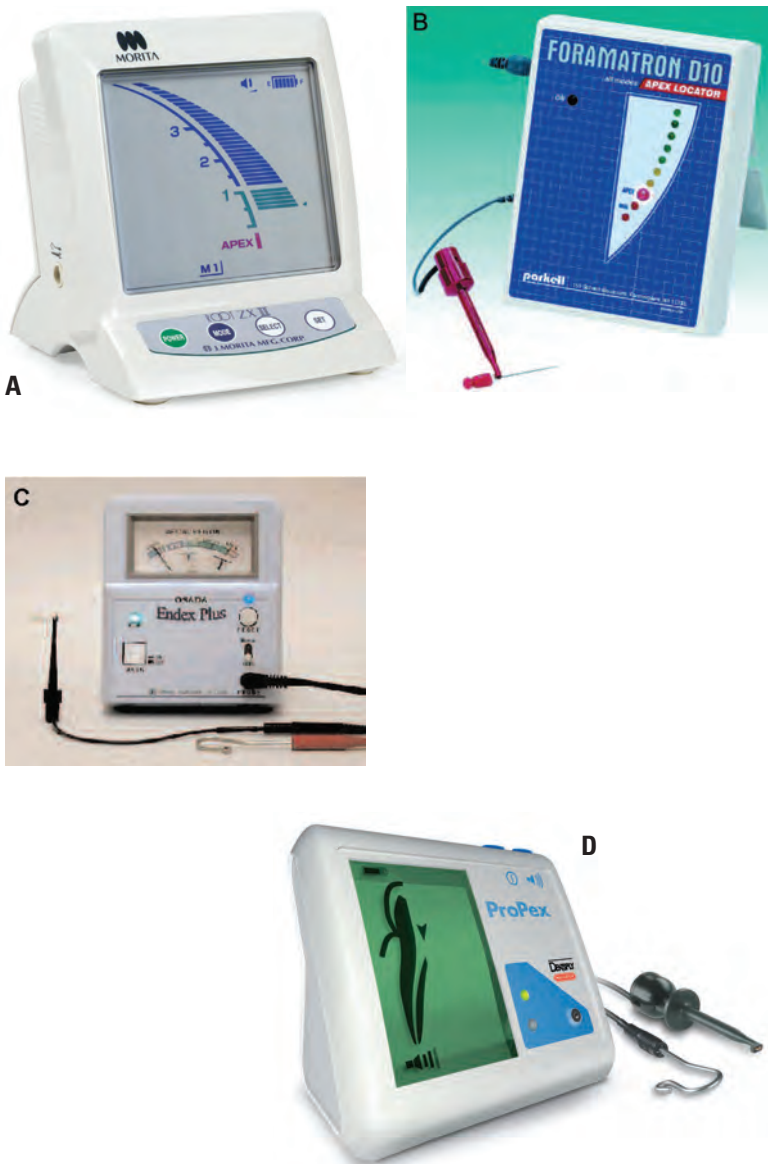


Figure 6-21. Apex locators. **A**, Morita ZX-II (J. Morita-USA). **B**, Foramatron (Parkell Co). **C**, Endex Plus (Osada, Inc.). **D**, ProPex (Dentsply/Tulsa). **Continued on next page.**

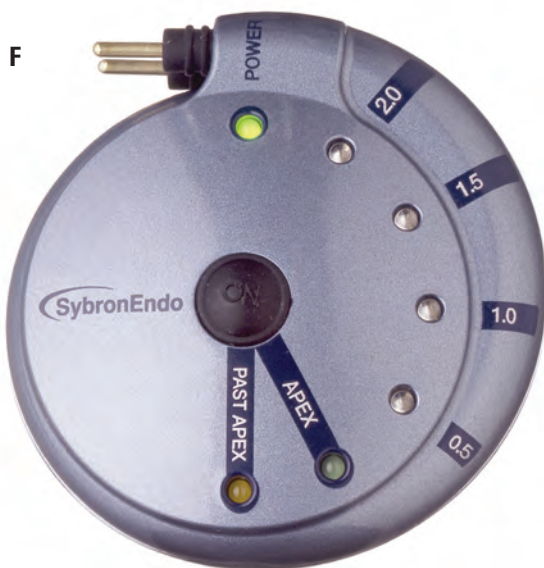


Figure 6-21. Continued. E, Elements Diagnostic Unit and Apex Locator (SybronEndo). F, Mini Apex Locator (SybronEndo). **Continued on next page.**



Figure 6-21. Continued. G & H, Miniature Apex NRG (Medidenta and/or Endo Tech).



Figure 6-22. The apical foramen is some distance from the radiographic apex; this stresses the importance of finding the actual apical orifice using multiple radiographs or an electronic apex locator. C = cementum; D = dentin. Courtesy of W.G. Skillen.

Achieving the Ideal Shape

The ideal shape of a finished root canal preparation is a gradually widening taper, from the apex to the crown. This may be achieved in one of two ways: by starting instrumentation at the apical termination and gradually enlarging the preparation toward the crown, the so-called **step-back** or **serial technique**, or by starting at the crown with a widening preparation and gradually working apically into a narrowing canal, the so-called **step-down** or **crown-down** preparation. Both approaches have their adherents and detractors, but with the emergence of power rotary preparations, the step-down approach seems to be emerging as the favorite.

Step-Back Preparation

The traditional approach to shaping the root canal was to start at the apex and work back up the canal to the crown with increasingly larger instruments, ever widening the canal. This worked well in straight canals. In curved canals, however, the preparation in the curved apical third often became hour-glass shaped and the apical constriction was destroyed. Weine termed this aberration a “zip,”

caused by transportation of the larger unyielding instruments.³ Sometimes perforation resulted (Figure 6-23).

The solution, it appears, is to clean and shape the canal to the apical constriction with the more flexible files, such as a no. 25, and then step back millimeter by millimeter with increasingly larger, stiffer instruments, for example, from a no. 30 up through a no. 60 (Figure 6-24). This prevents transportation in the apical third.

In hand instrumentation the instruments are precurved to match the curve of the canal and are then advanced in a moist canal until contact is made. The files are then rotated a quarter- to half-turn in a watch-winding motion and withdrawn. An irrigant is reintroduced, and the procedure is repeated. With each increase of instrument size, the working length is shortened by 1 mm (see Figure 6-24). The remainder of the canal, coronal to the curve, is then widened even more into the desired taper, often with Hedstrom files in a rasping motion or with a **Micro Mega 1500 sonic air handpiece** mounted with **RispiSonic** or **ShaperSonic** files. Introducing a lubricant in addition to the irrigant proves effective. Lubricants such as **R.C. Prep**, **Glyde**, and **ProLube**, or even liquid soap or **K-Y Jelly**, are often used. Removal of the cutting debris by irrigation between each instrument size is imperative. Otherwise the debris packs at the apex.

All in all the step-back technique has proved effective, and has led to a decided improvement in the cleaning and shaping, as well as the obturation of root canals.

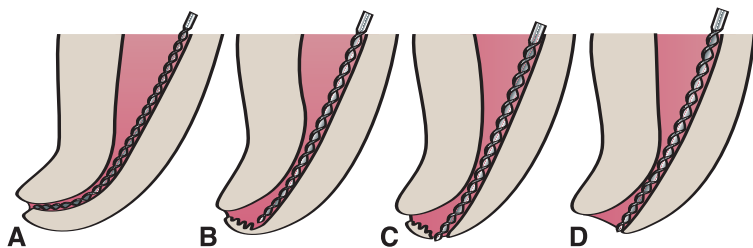


Figure 6-23. Hazards of over enlarging the apical curve. **A**, Small flexible stainless steel instruments (no. 10– to no. 25) readily negotiate the curve. **B**, Larger instruments (no. 30 and above) markedly increase in stiffness and cutting efficiency, causing ledge formation and cavitation. **C**, Persistent enlargement with larger instruments results in perforation. **D**, A “zip” is formed when the working length is fully maintained and larger instruments are used. Courtesy of R.E. Walton.

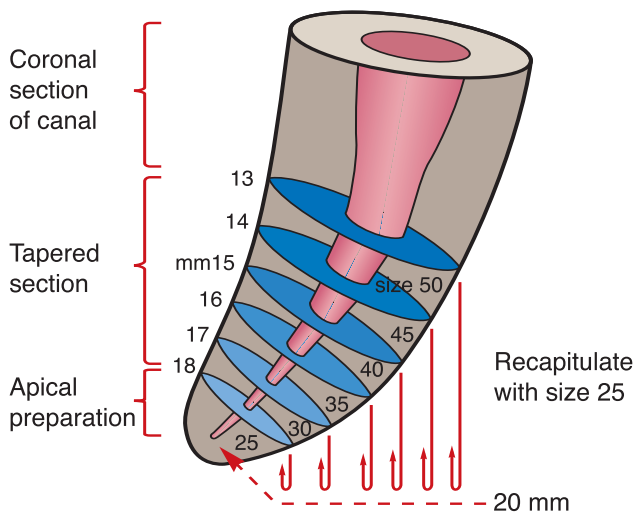


Figure 6-24. A stylized step-back preparation. A working length of 20 mm is used as an example. The apical 2 to 3 mm are prepared to at least a size 25. The next 5 mm are prepared with successively larger instruments, shortening the working length by 1.0 mm with each advance in instrument. Recapitulation with a no. 25 to the full length is performed between each step. When the curve is passed, the coronal part of the canal is further tapered with successive 0.04 and 0.06 mm/1 mm files and/or Gates-Glidden drills. Courtesy of B.G. Tidmarsh.

Step-Down (Crown-Down) Preparation

With the advent of rotary powered instrumentation and the introduction of NiTi instruments, the step-down preparation has become more popular. Flexible files rotating in a curved canal require unimpeded space, and widening this space from the crown down seems the logical solution.

As with the step-back technique, the first step is to explore the canal with a fine, curved stainless steel instrument. Once patency to the apical constriction has been confirmed, working length is established and irrigation and initial enlargement begin. Early in the technique's development, canal orifices were located and enlargement was done with Gates-Glidden drills. Starting with a size no. 1 instrument and drilling down canal for a short distance, the sizes of the instruments were increased to ever widen and flare

the beginning and straight portion of the canal. When 0.04 and 0.06 mm taper files were introduced, however, these became the instruments of choice for widening and tapering the canal.

Using hand instrumentation and starting with a size no. 50 file, for example, the instrument is used in a watch-winding motion and advanced down the canal for a short distance. It is then followed with increasingly smaller files down to a no. 25 to the apical constriction (ie, a no. 50 followed by a no. 45, a no. 40, etc, down to a no. 25), each instrument shaping the canal for 2 or 3 mm. Irrigation and lubrication are important. If a greater enlargement in the apical third is felt necessary, larger instruments can be worked down the canal in the same motion.

Similar step-down techniques employing different types of instruments have been promulgated. Using **ProFile Rotary instruments**, the step-down cleaning and shaping of the canal is done in the presence of a lubricant and irrigant.

ProFile instruments are U-shaped, and the set comes with **orifice shapers** that are first used to widen the coronal part of the canal. The cleaning and shaping of the remainder of the canal is done with a descending order of 0.04 mm taper instruments from ISO size 40 down to a 0.04 mm size 20 at the apical termination.

The ProTaperFile Rotary System employs rotary shaping files to widen access in the coronal two-thirds of the canal, to be followed by 1 and 2 shaping files and, finally, finishing files 1, 2, and 3 (Figure 6-25).

The newest **SybronEndo K3** instruments have a cross-sectional configuration more like a wood router (see Figure 6-9). They have a noncutting tip and three “lands” with positive rake angle blades. Two of the lands are broad and recessed, whereas the third land fully meets the wall. The recessed areas of the lands allow for chip collection and disbursement coronally. Access in the canal is gained by first using a 0.10 mm taper orifice opener at 250 rpm rotation. This is followed by a 0.08 mm opener for another 3 to 4 mm. At this point, working length is established and the K3 0.06 mm instruments are used at 200 to 250 rpm, starting with a size no. 35 and advancing down the canal with a descending order of 0.06 mm rotary files—30, 25, and 20 to working length. Apical patency is established with a no. 10 file. EDTA lubricants and copious irrigation are necessary throughout.

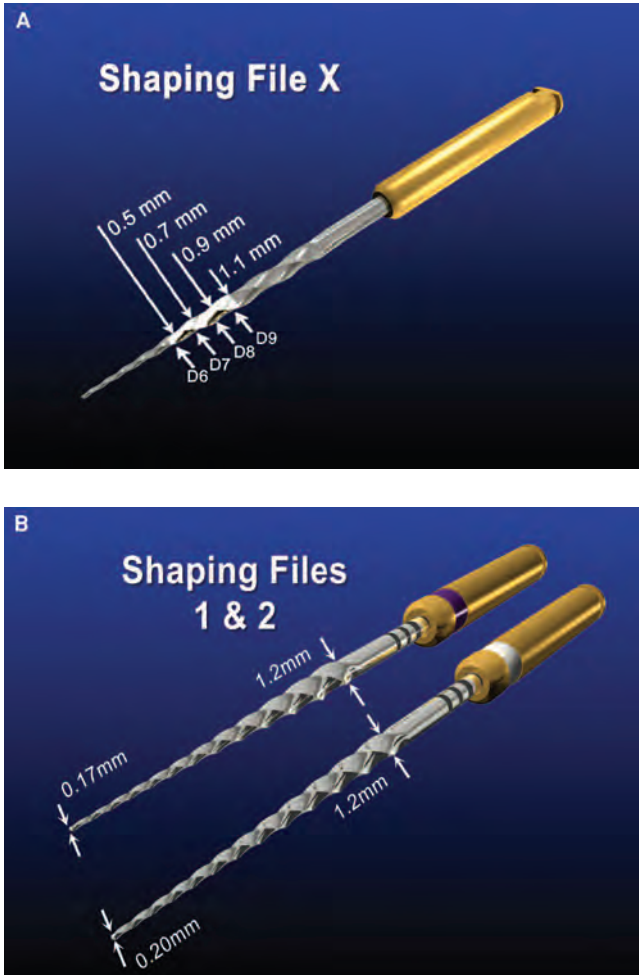


Figure 6-25. The ProTaperFile Rotary System. **A**, Shaping file X, an auxiliary instrument used primarily to extend canal orifices and widen access as well as create coronal two-thirds shaping in short teeth. **B**, Shaping files 1 and 2, used primarily to open and expand the coronal and middle thirds of the canal. Reproduced with permission from West J, Ruddle C, editors. Advanced Endodontics [videocassette]. *Continued on next page.*

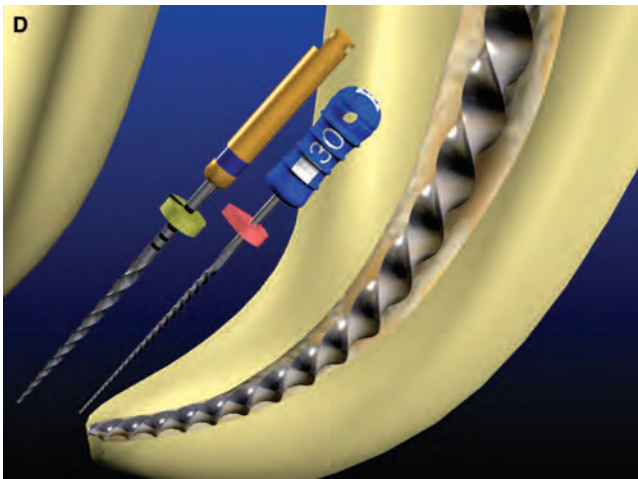


Figure 6-25. Continued. **C**, Finishing files 1, 2, and 3, used to expand and finish the apical third of progressively larger canals. **D**, Finishing file 3, used to finish the apical third of larger canals. The no. 30 file is used to gauge the size of the apical opening. Recapitulation with a regular no. 30 instrument, followed by liberal irrigation, is most important. Reproduced with permission from West J, Ruddle C, editors. *Advanced Endodontics* [videocassette].

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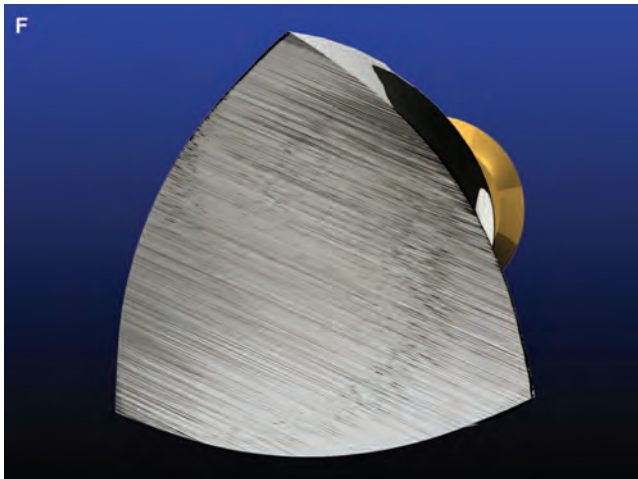


Figure 6-25. Continued. *E*, With the flexibility and cutting ability of nickel-titanium, ProTaper Rotary Files are assets in preparing curved constricted canals. *F*, Triangular cross-section presents three sharp cutting-blade edges that improve cutting ability and tactile sense. Reproduced with permission from West J, Ruddle C, editors. Advanced Endodontics [videocassette]

The **Balanced Force** technique using rotary **Flex-R** files is similar in concept. Initially the straight part of the canal is flared with Gates-Glidden drills, starting with the larger no. 6 and working down in 2 mm increments to a no. 1. Then the tapering of the remainder of the canal by balanced force begins, preloading the instrument with a clockwise motion and then shaping the canal with a counter-clockwise rotation. Increasingly larger instruments are used in **step-back reshaping** in the apical third, and the preparation extends to the radiographic apex (Figure 6-26).

LightSpeed rotary instruments (LightSpeed Technology) are like no other files or reamers. The LSX blade is very short, depending on the instrument size. The shaft is noncutting and has the flexibility to negotiate curves (Figure 6-27).

New additions to the LightSpeed rotary instruments include the CRX, used to prepare the coronal third of the canal, followed by the MRX to prepare the middle third of the canal. (see Figure 6-15). The apical third is finished by the LSX (Figure 6-28). Handpiece preparation is always with a gentle apical pecking motion. Instrumentation continues with sequentially larger sizes

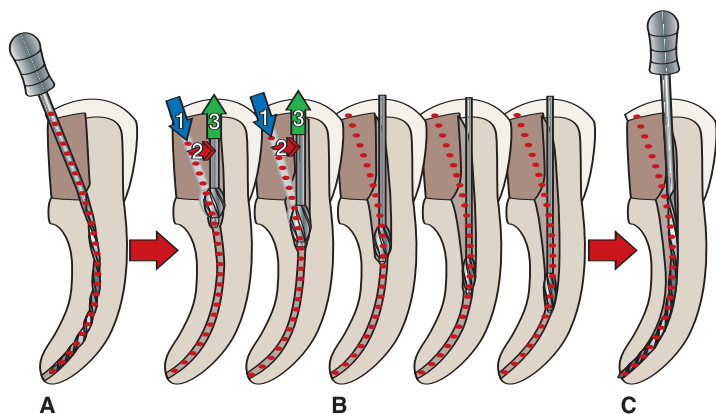


Figure 6-26. Balanced Force technique. **A**, File displays full curvature of the canal before radicular access is modified. **B**, Radicular access is completed with a descending series of Gates-Glidden drills progressing toward the apex in 2.0 mm or smaller increments. **C**, The dotted line indicates the original curvature, whereas the file displays the affective curvature after the radicular access is improved. The canal is now prepared for the enlargement by clockwise rotation of progressively larger instruments, followed by counterclockwise rotation and withdrawal. Courtesy of J.B. Roane.



Figure 6-27. The LightSpeed instrument (LightSpeed Technology). The cutting blades is U shaped in cross-section, and the tip is noncutting. The 16 mm shaft is noncutting. The instruments come in International Standards Organization (ISO) sizes 20 to 100, with half sizes between, and in .02, .04, and .06 mm/1 mm tip widths. They are nickel-titanium, very flexible, rotary instruments used at 1700–2000 rpm.

and ends when the apical terminus is round and all the walls are cleaned (Figure 6-29). The superb tactile feedback of LightSpeed's unique design makes this possible. When combined with tapered instruments in a hybrid technique the strengths of both systems are put to good use.

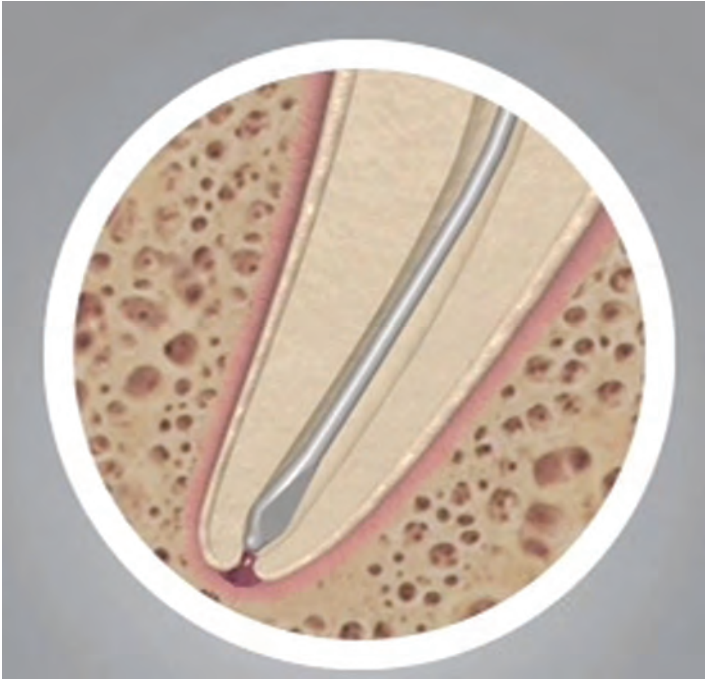


Figure 6-28. Note that only the cutting tip of the LSX LightSpeed contacts the canal walls.

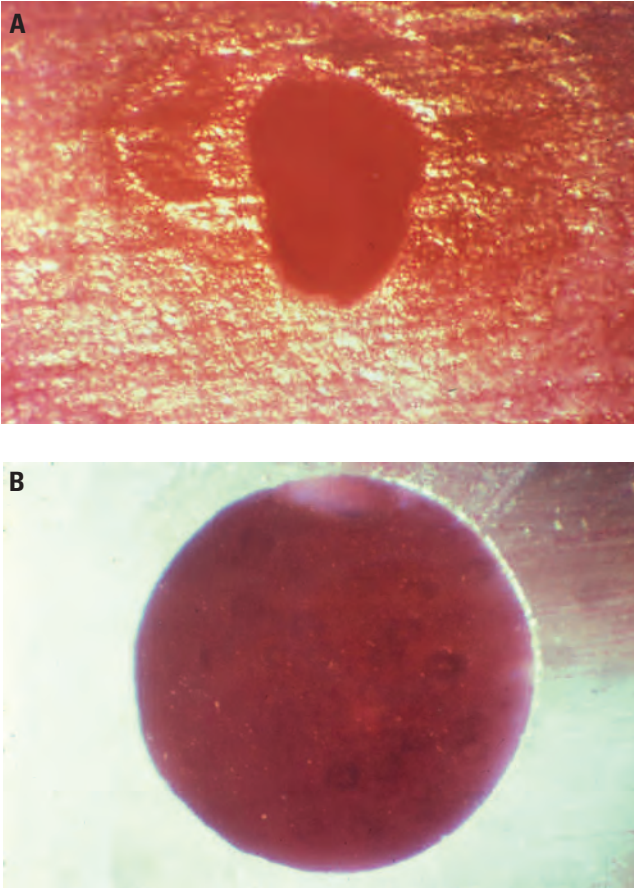


Figure 6-29. **A**, Natural shape of the canal before instrumentation with a LightSpeed instrument (Discus Dental). **B**, Shape of the canal prepared with a LightSpeed instrument. The apical third enlargement ensures the removal of more bacteria-laden dentinal tubuli (see Figure 3-1).

And again, the last 1mm or so of the canal, beyond the apical constriction, is last to be cleaned but not shaped, establishing apical patency with a size no. 10 or no. 15 instrument and copious irrigation.

The **MicroMega 1500 Sonic Air Endo System** (Medidenta/MicroMega, Woodside, NY) handpiece (Figure 6-30) attaches to the regular airline at a pressure of 0.4 Mpa. It has an oscillatory range of 1500 to 3000 cycles per second. Tap water irrigant/coolant is de-



Figure 6-30. Micro Mega 1500 Sonic Air handpiece (Medidenta/Micro Mega, Woodside, NY) is activated by pressure from the turbine air supply that produces oscillation. It can be mounted with special instruments that “rasp” the walls in enlargement and easily adjust to the length of tooth. Tap-water spray serves as an irrigant and coolant. Courtesy of Medidenta/Micro Mega Co.

livered through the handpiece to the preparation. The **MicroMega** employs two unusual endodontic instruments, the **RispiSonic** and the **Shaper Sonic**, resembling a barbed broach more than a K-file. As with ultrasonic preparations, these instruments must be free to oscillate in the canal and to rasp away at the walls. To accommodate the smallest instrument, size 15, the canal must be enlarged to working length, with hand instruments through size 20. The rasps have a 2.0 mm safe tip, and beginning this far from the apical stop, successively larger sizes are used as the instrument becomes loose in the canal. A flaring shape is developed. The sonic system has been shown to extrude the least amount of material out the apex.

The Canal Finder (EndoTechnic, San Diego, CA) is another system that has proved most effective, particularly in re-treatment cases and in **instrumenting fine canals**. The action of the Canal Finder handpiece is a vertical reciprocating motion, not a rotary motion (Figure 6-31). This tends to drive the instrument apically under continuous water irrigation. Avoiding rotary motion, one encounters less instrument breakage. The manufacturer recommends that **Master Files** (EndoTechnic), a modification of Hedstrom files, be used with the Canal Finder.



Figure 6-31. Canal Finder handpiece (EndoTechnic San Diego) operates from the turbine air supply in a vertical reciprocating motion, not a rotary motion. It has a continuous water irrigant and coolant feature that helps maintain canal patency. The Canal Finder uses Master Files, that resemble Hedstrom files. Because it has a vertical rather than a rotary motion, less or no file breakage is expected. It is especially good in opening very fine canals and in re-treatment cases.

REMOVAL OF THE SMEAR LAYER

Unfortunately, cleaning and shaping, either by hand or rotary instrumentation, leaves an ugly layer of dentin filings, tissue residue, fluids, necrotic material, and bacteria smeared against the walls of the preparation. This layer covers the orifices to the dentinal tubuli and the accessory canals and must be presumed to be infected. To place the final canal filling and seal the smear layer in place materially reduces chances of complete success. With the smear layer's removal, however, sealer is allowed to flow back into the dentinal tubuli and literally lock the final filling in place. This should prevent both the ingress and egress of any stray bacteria. For some years full-strength NaOCl (5.25%) followed by 17% ethylenediaminetetraacetic acid (EDTA) has been used to effectively remove the smear layer. An even more impressive system for smear layer removal has been developed

at Loma Linda University (Loma Linda, CA) and recently approved by the US Food and Drug Administration.⁴

The new irrigant solution, named **Bio Pure MTAD** (Dentsply/Tulsa Dental) (Figure 6-32), is made up of doxycycline (tetracycline isomer), citric acid, and a detergent—Tween-80. After a good deal of experimentation and reporting, it was determined that doxycycline was an ideal drug, owing to its bacteriostatic property as well as its propensity for chelating the inorganic components of the smear layer, the dentin calcium salts. The citric acid was added to the formula to potentiate this decalcification. The Tween-80 detergent improves the penetration of the other two components. The organic portion of the smear is removed with a rinse of NaOCl at a concentration of only 1.3% (4 parts H₂O and 1 part Clorox), which has been found to be as effective as half-strength and full-strength NaOCl.

Reports of the results of irrigation with this combination of bacteriostatic, chelating, and organic debris removal are most gratifying. After irrigation with a 1.3% concentration of NaOCl for 1 minute followed by 2 minutes of irrigation with MTAD, the smear layer is totally removed, the orifices of the dentinal tubuli grossly exposed, and the canal effectively disinfected (Figure 6-33). Results such as these greatly improve the chances of success following one-appointment root canal therapy.



Figure 6-32. Bio Pure MTAD (Dentsply/Tulsa Dental) is used to remove the smear layer and disinfect before obturation.

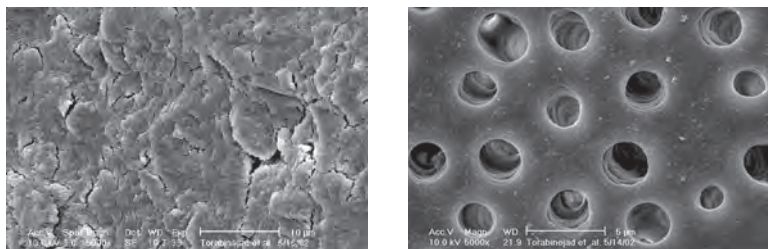


Figure 6-33. *Left*, Smear layer before removal with MTAD (Dentsply/Tulsa Dental). *Right*, Smear layer entirely removed with MTAD.

SmearClear (SybronEndo) is another solution used to remove the smear layer. It is composed of disodium and trisodium EDTA as well as polyoxyethylene ether and ATMA bromide. Its pH is 8.0: not acidic.

A new irrigant, as yet unnamed or marketed, is being developed at the University of British Columbia. Its action will be similar to MTAD.

Final Irrigation and Evacuation

In 2007, Nielsen and Baumgartner pointed out that “past studies have shown that the current irrigation methods are only effective in cleaning canals coronally but less effective apically.”⁵ Sixteen years before that, Baumgartner’s research also found that the last 5 mm of the canal is still teeming with fifty different strains of bacteria, and more importantly, 60% were strict anaerobes.⁶ Based on his previous research, he presently notes that to be effective, “endodontic irrigants should ideally be delivered near working length.” But at this time there is a danger of extruding debris and bacteria out the apex with irrigant pressure. And how thoroughly do our current methods of irrigation clean this last 5 mm?

In the past several years there has been a plethora of attempts to solve this problem. Today it has “boiled down” to a few. At the University of Tennessee, they tested various intracanal irrigation systems for their comparative safety by measuring the apical extrusion of irrigants.⁷ They tested the **EndoVac**, the **EndoActivator**, the **Rin-sendo**, and the **Ultra Sonic Needle Irrigation** systems against tradi-

tional manual irrigation with a **Max-I Probe** needle (Dentsply/Tulsa). They found that the Max-I-Probe, Ultrasonic with constant flow, and Risendo groups extruded a significantly greater amount of irrigant through the apex compared with EndoVac and EndoActivator.

Recognizing that the EndoVac system appears to be the safest irrigation evacuation unit, Nielsen and Baumgartner at Oregon set out to determine the effectiveness of the system in removing debris from the last 1 to 3 mm of a prepared canal.⁵ They noted that: “Ideally, root canal irrigants should flush out debris, dissolve organic tissue, kill microbes, destroy microbial byproducts, and remove the smear layer. To accomplish these objectives, there must be an effective delivery system to the working length.”

They found that the **EndoVac** system “showed significantly better debridement at 1 mm from working length compared with needle irrigation.” At 3 mm they were comparable. But it is those literally millions of microbes that remain in that last millimeter that could spell the difference between success and failure. It also stands to reason that a cleaner apical third should lead to a better apical seal. So what is the EndoVac system?

The **EndoVac** system (Discus Dental, Culver City, CA) was developed by Dr. John Schoeffel as a novel new irrigation system. “A delivery/evacuation tip is attached to a syringe of irrigant (such as NaOCl) and the high speed suction of the dental chair (Figure 6-34B). A small tube attaches either a macro-or a micro-cannula to the suction (Figure 6-34A,C). The delivery/evacuation tip places irrigant in the chamber and siphons off the excess to prevent overflow (Figure 6-34C). The macrocannula is plastic with an open end that measures ISO size 55 with an .02 taper (Figure 6-34A).

It is the macrocannula that cleans the upper two-thirds of the canal.

The microcannula is stainless steel and has twelve small laterally positioned, offset holes in four rows of three, with a closed end measuring ISO size 32 (Figure 6-34D). As these cannulas are placed in the canal, negative pressure pulls the irrigant from a fresh supply in the chamber, down the canal to the tip of the cannula, into the cannula and out through the suction hose. The microcannula can be used at working length in a canal enlarged to ISO size 35/.04 or larger.” By alternating 5% NaOCl with 17% EDTA, the smear later will be removed along with the other debris and bacteria (Figure 6-35).

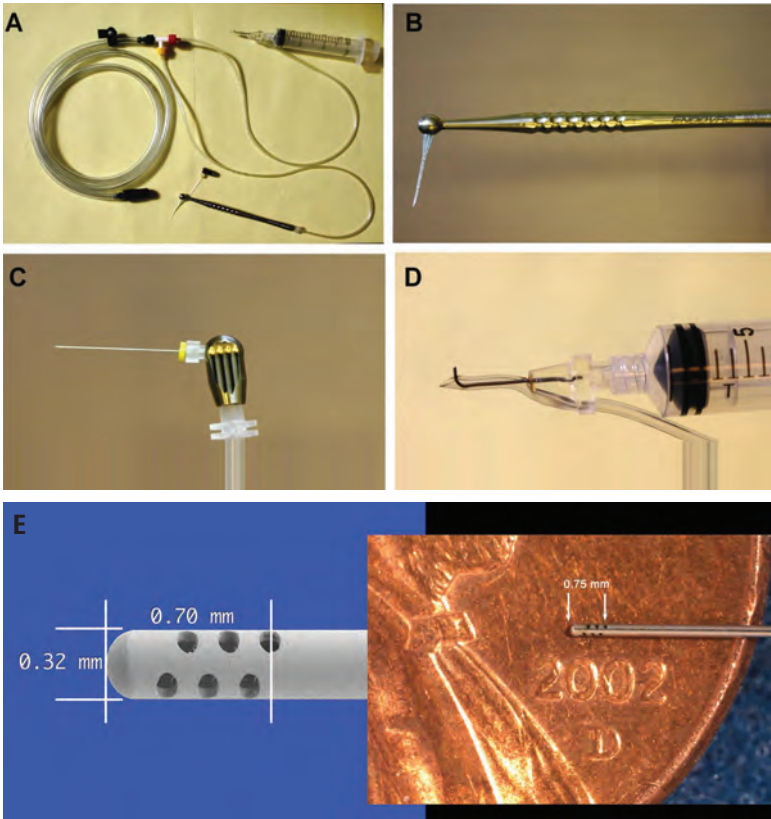


Figure 6-34. **A**, The complete EndoVac system. **B**, The macrocanula attached to its handle used for initial flushing of the coronal canal. **C**, The microcanula attached to its handle. This replaces the macrocanula and is used for irrigation of the apical portion of the canal to working length. **D**, The delivery/evacuation tip attached to a syringe. Irrigant is delivered to the pulp chamber by the metal needle. Any excess is immediately suctioned off through the plastic tubing surrounding the metal that is attached to the suction tubing. **E**, The microcannula, compared in size to a penny, is stainless, has twelve tiny offset holes and is no larger than a no. 32/.04 instrument. Nielsen B, Baumgartner, JC. J Endod 2007;33 (5): 611-615. Permission granted.

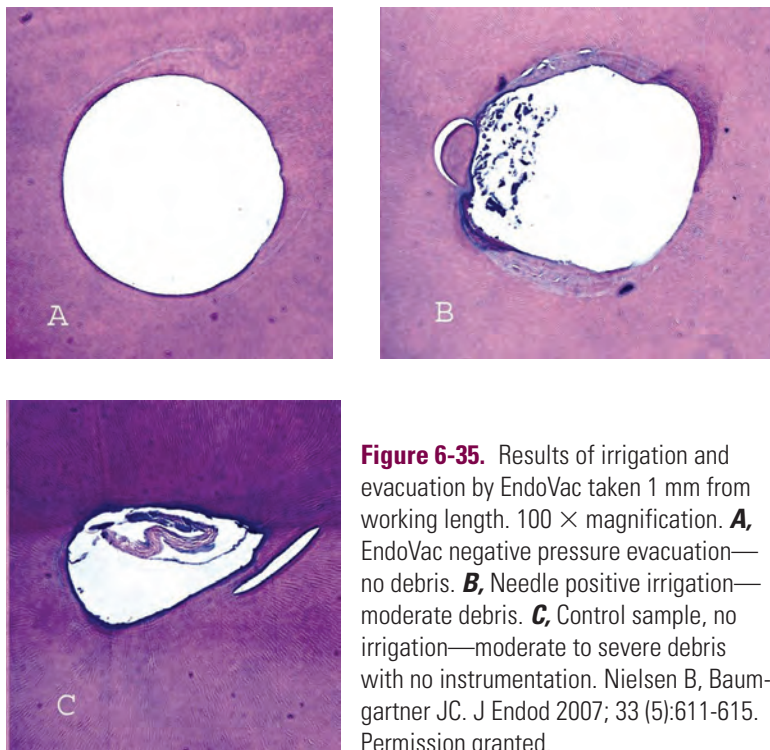


Figure 6-35. Results of irrigation and evacuation by EndoVac taken 1 mm from working length. 100 × magnification. **A**, EndoVac negative pressure evacuation—no debris. **B**, Needle positive irrigation—moderate debris. **C**, Control sample, no irrigation—moderate to severe debris with no instrumentation. Nielsen B, Baumgartner JC. J Endod 2007; 33 (5):611-615. Permission granted.

Schoeffel has pointed out that a “gas pocket,” an “apical vapor lock” if you will, develops at the very apex, and although this gas bubble can be penetrated by instruments, it does not collapse. It is this “bubble” that prevents most irrigants from reaching the apical 3 to 1 mm. It is the negative pressure of the EndoVac system, however, that removes the bubble and allows irrigants to reach the very apex.

Neilsen and Baumgartner observe that “Future studies should look into the effect of taper, apical size, safety and effect on the apical seal when the EndoVac is used for irrigation.” The University of Washington endodontists investigated some of these variables.⁸

Canals were prepared in either a tapered or nontapered configuration and contaminated for 30 days with *Enterococcus faecalis*. The canals were then prepared and were irrigated with 6% NaOCl and 17% EDTA. Irrigation and evacuation of the irrigant was done

with either the negative-pressure EndoVac system, or manually using positive pressure irrigation using side-vented Max-i-Probe needles (Dentsply/Tulsa Dental) and a 10-ml syringe.

The results were spectacular. All the specimens irrigated and evacuated with the EndoVac system rendered negative cultures after 48 hours (24 out of 24). On the other hand, eight specimens of the positive-pressure-with-the-vented-needle specimens rendered positive cultures after 48 hours (8 out of 24). The difference was significant (Figure 6-36).

Another finding; it made no difference whether the canals were tapered or not, nor whether the apical enlargement was with a size 35 or size 45 instrument. They did conclude, however, that “apical negative-pressure irrigation has the potential to achieve better microbial control than traditional irrigation delivery systems.”

Other irrigating systems have been reported. Burleson et al. at Ohio State used an ultrasonic irrigating device along with a constant flow pump (Figure 6-37).⁹ They reported 100% removal of bacteria in most cases.

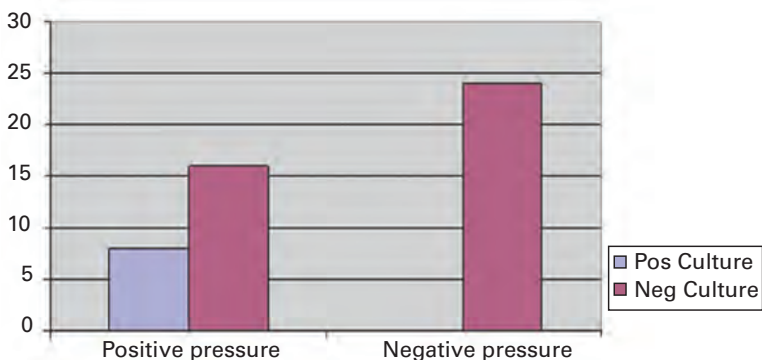


Figure 6-36. (Left) Eight specimens of the positive pressure irrigation groups rendered positive bacterial cultures at the end of the incubation period, versus total negative cultures from the 16 specimens where negative pressure of irrigation was used. Statistically significant difference ($p = 0.004$). (Right) All negative pressure irrigation specimens were culture negative. Hockett JL et al. *J Endod* 2008; 34-11:1374-1377. With permission.

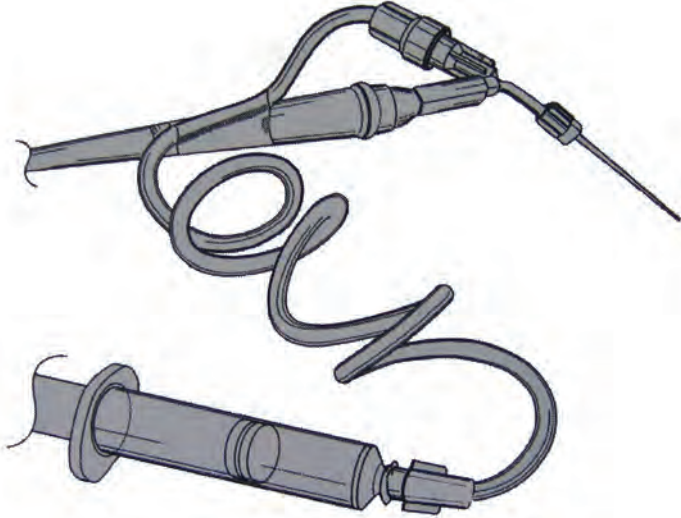


Figure 6-37. Diagrammatic representation of the ultrasonic irrigating device. Burleson A, Nusstein J et.al. J Endod 2007;33 (7):782-87. Permission granted.

FINAL NOTES

As stated at the beginning of Chapter 4, the proper cleaning and shaping of the root canal is as important to success as is the final filling itself. If debris and bacteria, or even the smear layer, are left lining the walls of the preparation, a reduction in success over the long run must be expected. There are many ways to “skin a cat,” as the old saying goes, and the method of cleaning and shaping canals that is the most effective for each individual is the method to pursue. That is not to say that if a simpler or better method comes along, it should not be considered.

In any event, proper preparation and irrigation is the *sine qua non* of continued success.

REFERENCES

1. Ingle JI, Levine M. The need for uniformity of endodontic instruments, equipment and filling materials. In: Transactions of the Second International Conference of Endodontics. Philadelphia: University of Pennsylvania Press; 1958. p. 123.
2. Larsen CM, Watanabe I, Glickman GN, He J. Cyclic fatigue analysis of a new generation of nickel titanium rotary instruments. *J Endod* 2009;35 (3): 401-403.
3. Weine F. Endodontic therapy. St. Louis: CV Mosby; 2002.
4. Welk AR, Baumgartner JC, Marshall JG. An in vivo comparison of two frequency-based electronic apex locators. *J Endod* 2003; 29:497.
5. Torabinejad M. A new solution for the removal of the smear layer. *J Endod* 2003; 29:170.
6. Neilsen BA, Baumgartner JC. Comparison of the EndoVac system to needle irrigation of root canals 2007;33 (11):611-615.
7. Baumgartner JC, Falkner WA. Bacteria in the apical 5mm of infected root canals. *J Endod* 1991;17: 380.
8. Desai P, Himel V. Comparative safety of various intracanal irrigation systems. *J Endod* 2007; 33(5):611-615.
9. Hockett JL, Dommisch JK, Johnson JD, Cohenca N. Antimicrobial efficacy of two irrigation techniques in tapered and nontapered canal preparations: An in vitro study. *J Endod* 2008;34(11): 1374-1377.
10. Burleson A, Nusstein J et al. The in vivo evaluation of hand/rotary ultrasound instrumentation in necrotic, human mandibular molars. *J Endod* 2007;33 (7): 782-787.

Obturation of the Radicular Space

A review of the Washington study of endodontic success and failure revealed that nearly 60% of the failures were due to incomplete obliteration of the radicular space.¹ The necessity of properly filling the canal seems obvious once cleaning, shaping, and disinfection are completed, yet ineffective obturation is often a prelude to eventual failure.

There are essentially three basic methods of filling the root canal system, and there are a number of minor permutations of these three methods. The first method is the **lateral condensation** technique, in which the canal is filled with “cold” gutta-percha points that are laterally condensed or compacted to fill the space. The second method is the **sectional** technique, in which small pieces of gutta-percha are stacked by compaction, one on top of the other, until the canal is filled. The third method is the **warm gutta-percha/vertical compaction** technique, in which gutta-percha is warmed in the canal, made plastic, and then compacted. The same filling techniques may also be applied to the new resin, Resilon. All these techniques are dependent on the addition of a cement-like sealer that adheres to the dentin walls and fills any space not filled by the compressible points.

SEALERS

Before proceeding with the actual filling techniques, a review of the sealers that are essential to a well-obtured canal is in order. First, it must be agreed that most of the sealers are soluble and subject to dissolution. Second, it must be conceded that most sealers are not

adhesive to the dentin walls, and that some of the sealers discolor teeth, especially those sealers that contain silver. So, it is best not to leave any sealer in the crowns of teeth. Another concession is irritability; that is, most of the sealers irritate periapical tissue if they escape the apex, some more than others.

For years, zinc oxide–eugenol (ZOE) has been the standard endodontic cement/sealer. The initial ZOE product, **Kerr Pulp Canal Sealer** (Kerr), contained powdered silver as a radiopaque mineral, but the silver stained teeth. Grossman then used essentially the same ZOE formula but added barium sulfate as a radiopacifier rather than silver. This became known as **Grossman's Cement** or **Roth's Sealer**. The adherents to the warm gutta-percha/vertical compaction technique have used the original Pulp Canal Sealer but with the added advantage of Extended Working Time (EWT) (SybronEndo; Figure 7-1). Other ZOE cements are **TubliSeal EWT** (SybronEndo; see Figure 7-1) and **Wach's cement** (Roth's Pharmacy).

Graduating from days of ZOE, the newest sealer formulas are resins. **RealSeal**, (SybronEndo) a methylmethacrylate resin, also known as **Epiphany** was among the first. It is a thermoplastic synthetic polymer with radiopaque fillers added. Its resin matrix is a mixture



Figure 7-1. **A**, Kerr Pulp Canal Sealer EWT (Extended Working Time) also known as Root Canal Sealer (SybronEndo/Kerr). **B**, Kerr Sealapex with Ca(OH)_2 (SybronEndo/Kerr). *Continued on next page.*



D



E



F



G



Figure 7-1. Continued. **C**, Kerr Tubli-Seal EWT (SybrionEndo/Kerr). **D**, Thermaseal Plus (Dentsply/Tulsa Dental). **E**, AH 26 (Dentsply/DeTrey). **F**, AH Plus (Dentsply/DeTrey). **G**, RealSeal, a.k.a. Epiphany (SybrionEndo).

of bisphenol-A glycidyl methacrylate (BIS-GMA; see Figure 7-1G). RealSeal attaches to the dentin walls, whereas ZOE does not. Another resin sealer is **EndoRez** (Ultradent). It is self-priming and is designed to work best with EndoRez, resin-coated gutta-percha points. A glass-ionomer sealer (Brassler) has also been introduced to accompany gutta-percha points coated with glass-ionomer particles.

Using the basic ZOE formula, a Swiss dentist added paraformaldehyde and called it **N2**. The American version was dubbed **RC2B**. Both were **condemned worldwide** and universally banned for their tissue destructive component—paraformaldehyde. The World Health Organization has recently declared formaldehyde “**a human carcinogen**.” European versions, **SPAD** and **Endomethasone**, are equally destructive.

Variations of ZOE sealers are **CRCS** (Coltene/Whaledent/Hygienic) and **Sealapex** (SybronEndo; see Figure 7-1), both of which contain **calcium hydroxide** based on the premise that CaOH exerts an apical osteogenic effect. Both are slow-setting and do not contain eugenol. **Apexit** (Vivadent) also contains calcium hydroxide. Whether the CaOH is released from these cements to exert its osteogenic action is under debate.

Sealers classified as plastics and resins have become popular. **Diaket** (3M/ESPE) is an earlier sealer, combining zinc oxide and a resin-reinforced chelate. It is quite sticky but has the advantage of adhering to dentin walls. Diaket was followed by the original AH26 (Dentsply/DeTrey), an epoxy resin sealer that became quite popular but had two drawbacks: it was toxic to periapical tissue and it stained teeth. The product was reformulated to alleviate these problems and renamed **AH Plus** (Dentsply International). It is also distributed as **ThermaSeal Plus** (Dentsply/Tulsa Dental; see Figure 7-1) as the sealer accompanying **ThermaFil** solid core fillers. Under both of these names, these sealers have become popular worldwide. A newer product, **Medicated Canal Sealer** (MediDenta), is essentially Grossman's Cement with 10% iodoform added. It is used with **Medicated Gutta-Percha**, containing either iodoform or tetracycline (MediDenta). The two combine to release iodoform and tetracycline to control residual bacteria.

As previously stated, it is imperative that the smear layer be removed before any of the cement/sealers are applied. The smear layer can plug up all the tubuli and may later dissolve out, allowing for

apical leakage. Removal of the smear layer allows the sealer to flow back into the uncovered dentinal tubuli and lateral canals, locking the sealer in place and preventing leakage between the sealer and the walls of the canal. It also may seal bacteria and entomb them in the tubuli.

However, one must remember that the root canal filling is no better than the restoration of the crown of the tooth. If leakage occurs around the coronal restoration, the oral acids and bacteria will slowly penetrate the root canal filling, and eventually reach the apex to set up periapical inflammation and infection. This may well account for root canal fillings that fail years after having appeared healed.

Sealers are mixed to a creamy consistency, not too thin and not too thick (Figure 7-2). They can be applied to the walls of the canal in a number of different ways. Some pump the sealer into the canal with a gutta-percha or paper point. Some carry it into the canal on a file or reamer, twirling it counterclockwise. Others use lentulo spirals, best rotated in the fingers, not in a handpiece. Sealer that is whipped sets too early. The canal must be liberally coated, and each auxiliary point added to the canal should be generously covered with cement.



Figure 7-2. Root canal cement should be mixed to a thick, creamy consistency that may be strung off the slab for 2.5 cm (1 inch).

Filling Points or Cones

For over 100 years, gutta-percha has been the preferred solid filling material for root canals. It now comes in various sizes and shapes. The original shape was contoured to fit the shape of the canal—cone-shaped—hence, the proper term for these should be gutta-percha cones. This was followed by a contour to more closely fit the shape of the instruments used in condensation—point-shaped—hence, the proper term, gutta-percha points. By common usage, both shapes are now called “points.” Gutta-percha has stood the test of time but was fated to be succeeded by a modern resinous plastic, in this case, **Resinol a.k.a. Real Seal**.

It is quite possible that after 150 years, gutta-percha will suffer its demise as an endodontic filling material, as it did for golf balls. In its place we may well see modern chemical compounds as the obturating material of the future. Today, **Real Seal** with a synthetic polymer-based material, methylmethacrylate, has been fashioned into obturation cones (Figure 7-3A). The points, that also contain bioactive glass and radiopaque fillers, are flexible, nontoxic, thermoplastic cones, soluble in chloroform but not in water. Pellets of **Real Seal** may even be heated and expressed through an **Obtura** gun. In addition, the polyester points are sealed to a resin-based composite sealer, **RealSeal** (SybronEndo) a.k.a. **Epiphany**, similar

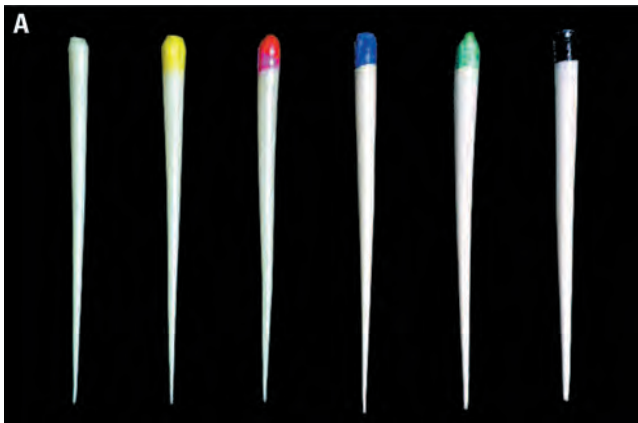


Figure 7-3. A, Color-coded Real Seal points (SybronEndo), a polyester that has been fashioned into endodontic obturation cones. The cones also contain bioactive glass and radiopaque fillers. **Continued on next page.**



Figure 7-3. Continued. B, RealSeal or Epiphany sealer (SybronEndo), a mixture of methacrylate and bisphenol-A glycidyl methacrylate urethane resin. RealSeal bonds chemically with Resilon points as well as with the dentin walls. Courtesy of SybronEndo.

to **EndoRez** (Ultradent), a mixture of methacrylate or BIS-GMA urethane resin. These sealers have the advantage of bonding chemically not only with the polyester cones but with the dentin walls as well (Figure 7-3B). If the smear layer is first removed and the dentin walls treated with a sulfonic acid primer, the resin sealer and the resin cones together form a monoblock that seals the canal and the tubuli (Figure 7-4A through 7-4C).

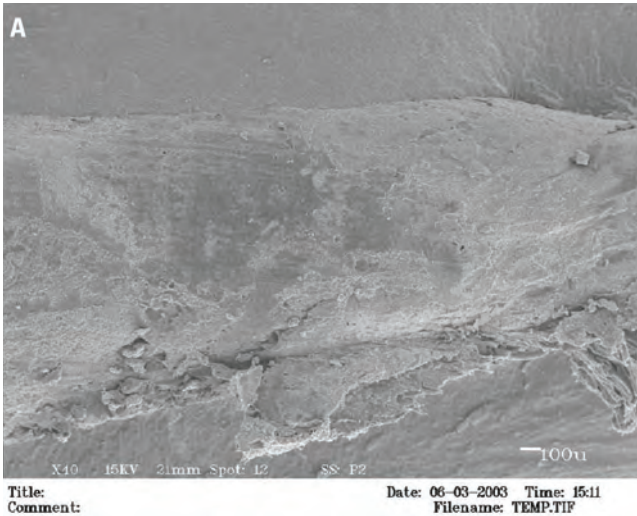
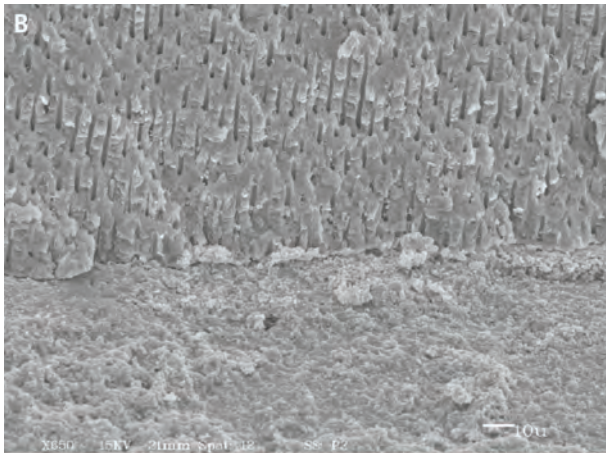


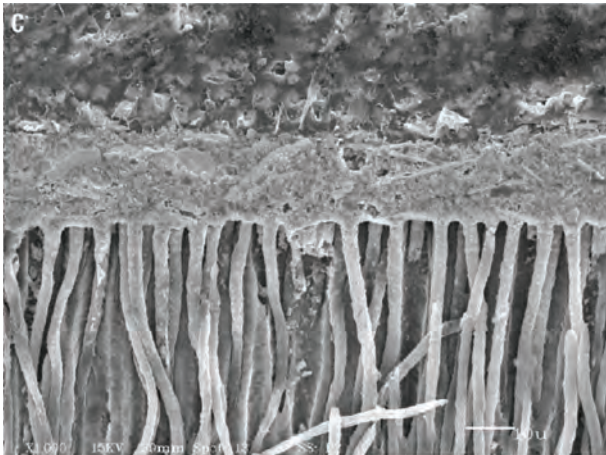
Figure 7-4. A, Monoblock of Resilon points and Epiphany sealer completely obturating the space and attaching to the dentin walls. **Continued on next page.**

Parkell has recently released **MetaSeal**, an endodontic version of their successful 4-meta hybrid bonding agents. MetaSeal cures best in a damp environment and bonds to dentin, gutta-percha and Resilon. First remove the smear layer. A curing light exposure assures an instant coronal seal.



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Figure 7-4. Continued. **B**, High-power view of the attachment of Resilon/RealSeal to the dentin wall. First, the smear layer must be removed and a sulfonic acid primer used. **C**, Penetration of the Resilon/Epiphany combination into the dentinal tubuli opened by the removal of the smear layer.



Figure 7-5. Obturation with Resilon/Epiphany demonstrates the radiopacity of the filling material and sealer. Courtesy of W.R. Watson Jr.

Microbial leakage studies over 30 days showed the synthetic filling to be superior to gutta-percha and a cement sealer.² Typical obturation techniques of lateral condensation or warm vertical compaction are used (Figure 7-5). Less pressure needed for compaction with these materials should lead to fewer vertical fractures later.³

Slowly, it appears endodontics is moving out of the nineteenth-century world of gutta-percha and zinc oxide–eugenol and into the twenty-first century of resin chemistry, something this author predicted nearly 40 years ago would happen.⁴

MULTIPLE-POINT LATERAL COMPACTION OBTURATION WITH GUTTA-PERCHA OR RESILON

First, a **spreader** is selected that will reach to the working length. Most spreaders are now numbered sizes 15 to 45, with the same system used in instrument standardization. Nickel-titanium spreaders have proved more effective than those of stainless steel. The spreader should be inserted in the canal to depth and then marked at the incisal/occlusal edge to be sure it does not overextend when later used to spread aside the additional points added after the primary point is in place.

A **primary point** must then be selected that matches the same size and number of the last file used at the apex. It may be slightly blunted if necessary and must extend to the “apical stop” (the working length) and fit well the apical third, even to the point that a bit

of “tug back” is felt. To be sure, it is best to radiograph the point in place to make certain it actually reaches the apical stop and that it generously fills the apical area. The point is then marked at the incisal/occlusal edge, removed, and set aside. Next, the canal is dried with paper points and the sealer is applied as noted previously.

The primary point is then coated with sealer and inserted into the canal to its full measured depth (Figure 7-6A). The excess at the crown should then be snipped off. This is followed by the act of spreading or lateral compaction.

The preselected spreader is inserted alongside the primary point and slowly rotated back and forth until it reaches its full predetermined depth (Figure 7-6B). It is important that it **fully reach this depth**. It should be held there for a minute or so to allow the movement of the sealer and gutta-percha, and then slowly removed with the same rotating motion. The pathway formed by the spreader is then filled with an additional point that is standardized to the same size and shape as the spreader. It, too, has been coated with sealer (Figure 7-6C). This point is immediately followed by the spreader rotated apically as far as possible (Figure 7-6D). When removed, its pathway is again filled by an additional coated point, followed again by the spreader (Figure 7-6E). This spreading/compaction action is continued until no more auxiliary points can be added to the canal (Figure 7-6F). To avoid fracturing teeth, one is warned not to use too much pressure in forcing the spreader, especially in lower incisors.

At this time, it is advisable to radiograph the fill to be sure it has reached the minor foramen and totally filled the canal without any discrepancies (Figure 7-7). Now is the time to make any adjustments. Once the filling is perfect, the excess points are seared off at the orifice of the canal at the base of the chamber. A very **large plugger**, such as size no. 140, or a small amalgam plugger is then selected to finally compact the filling with apical force. If satisfied with the result, the chamber should be thoroughly cleaned of all cement. An adherent sealer, such as C&B Metabond (Parkell Co.; Figure 7-8), should then be placed over the gutta-percha filling and the entire floor of the pulp chamber. This should discourage microleakage alongside the obturation from any coronal leakage that might develop in the future. The final coronal filling and/or crown may then be placed.

For years, lateral compaction has been the worldwide standard for obturation. If properly done, it has proved to be a very

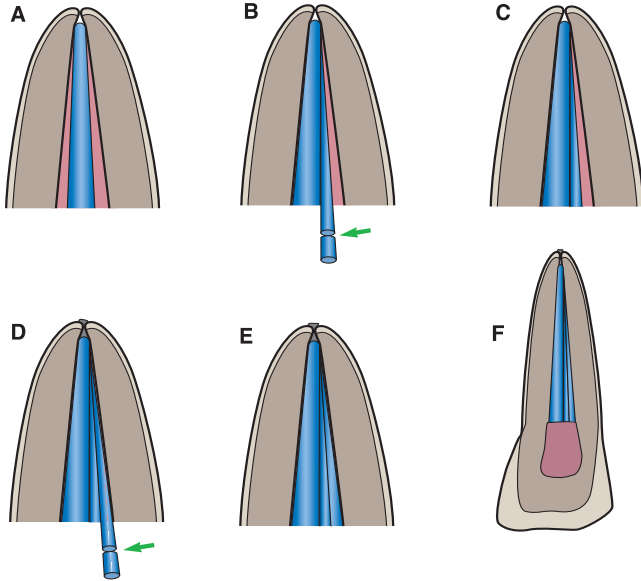


Figure 7-6. Lateral compaction, multiple-point filling procedure. The spreader has been tested previously to reach within 1 mm of the apical constriction. A thin layer of sealer lines the canal walls, and the tip of the primary point is coated with cement. **A**, The primary point is carried fully to place to within 1 mm of the "apical stop." Excess in the crown is severed at the cervical with a hot instrument. **B**, The spreader (arrow) is inserted to the full depth and allowed to remain one full minute as gutta-percha is compacted laterally and somewhat apically. **C**, The spreader is removed by rotation and immediately replaced by the first auxiliary point previously coated with sealer. **D**, The spreader (arrow) is returned to the canal to laterally compact the mass of filling. Secondary vertical compaction seals the apical foramen. **E**, The spreader is again removed, followed by the matching auxiliary point. The process continues until the canal is totally obturated. **F**, All excess gutta-percha and sealer are removed from the crown to below the free gingival level. Vertical compaction with a larger plugger completes root canal filling. After an intraorifice barrier and bonding sealer is established, a permanent restoration with adhesives is placed.

acceptable method to obturate most root canals. Some clinicians dip the primary point in chloroform for **only one second** before inserting it in the canal, claiming this makes for a more adaptive seal at the minor foramen.

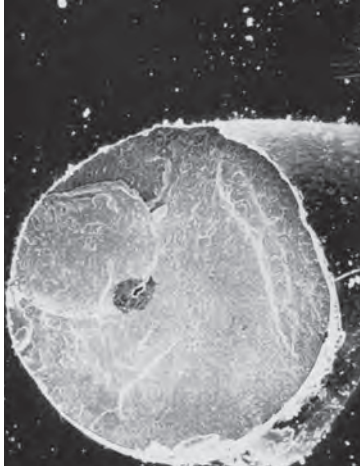


Figure 7-7. Scanning electron micrograph of a cross-section of lateral compaction illustrates how gutta-percha is condensed in the apical area with the placement of the initial spreader. Note how all the spreader tracts occur from the wall into the gutta-percha in a “wedging” fashion and adapt the gutta-percha to the opposite walls. Courtesy of C. W. Newton.



Figure 7-8. C & B Metabond adhesive bonding agent (Parkell Co.) is applied over the final root filling to seal it from any coronal microleakage that might occur later. All filling material and cement must be removed from the crown; then the area is etched and the C & B Metabond applied.

VERTICAL COMPACTION OF WARM GUTTA-PERCHA

The claim for this technique is that all the “portals of exit,” even lateral and accessory canals, are sealed when the warm gutta-percha is vertically compacted from above. Oftentimes, a minor flow of cement exudes from the apical foramen as well.

As with lateral compaction, the **pluggers** to be used rather than spreaders are first tested in the canal to be sure they extend as far as desired: wider pluggers for the coronal third of the canal and narrower pluggers for the apical third. The **master cone** used is the traditional cone shape rather than the slender, numbered points used in cold gutta-percha lateral compaction. The shape of these cones more closely fits the widened flare of this preparation. The primary cone is selected and inserted to the radiographic terminus (Figure 7-9). It should exhibit tug back. If satisfactory, it is **then marked at the incisal/occlusal edge** and withdrawn, **and 0.5 to 1.0 mm of the tip is removed**.

After the canal is dried and sealer applied, the coated primary cone is inserted to the incisal mark. This leaves it 0.5 mm short of the apex. The portion of the cone **in the chamber** is then severed with hot instruments (Figure 7-10A) such as **Touch ‘n Heat** (SybronEndo; Figure 7-10B) or the **Thermique Thermal Condenser** (Parkell Co.; Figure 7-10C), and vertical pressure is applied using the largest plugger. This should move the mass apically (Figure 7-11A). The heated instrument is again applied for 2 to 3 seconds,

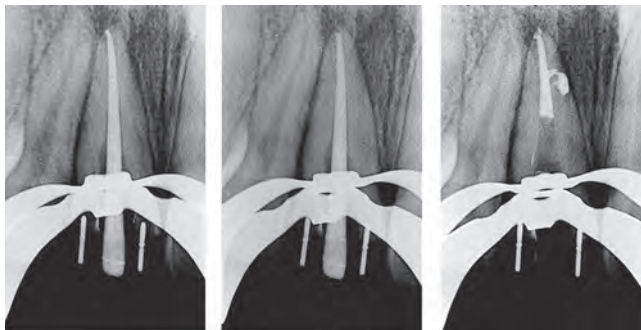


Figure 7-9. Vertical compaction of warm gutta-percha. Fitting the master gutta-percha cone. The cone is fit to the radiographic terminus. Courtesy of J. West.

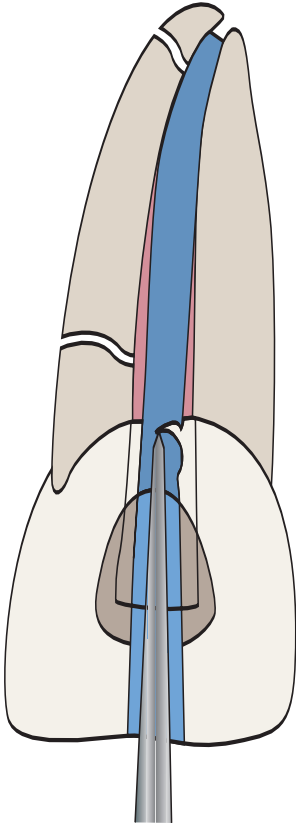
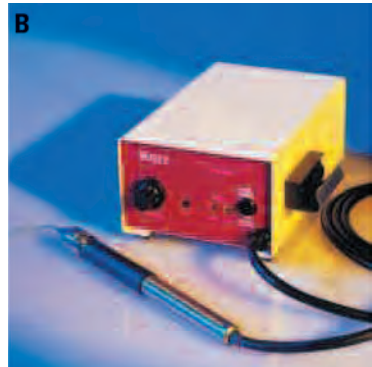
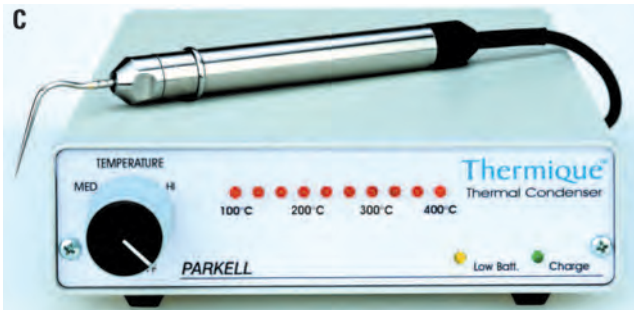
A**B****C**

Figure 7-10. Thermal compaction method. Surplus gutta-percha is removed with the heat carrier to the canal orifice **A**, Both Touch 'n Heat 5004 (Sybron Endo) **B**, and Thermique Thermal Condenser (Parkell Co.) **C**, Heat the spreader/plugger at the touch of a button to plasticize the gutta-percha in the canal. These devices are also used in re-treatment in the removal of gutta-percha. Both are battery-powered.

and some of the gutta-percha is removed (Figure 7-11B). Immediately, the mid-sized plugger is applied vertically and the warmed mass is compacted apically. This heat-and-compact procedure is repeated using the smallest plugger and then the mid-sized plugger until the apical third is completely obturated with the warmed gutta-percha (Figure 7-11C). If a post is to be placed, this is an adequate filling. If the canal is to be totally filled, it may be completed with sectional pieces of warmed gutta-percha or back-filled with a gutta-percha “gun” (Figure 7-11D) such as the **Obtura II** (Obtura/Spartan; Figure 7-11E) or the Discus **HotShot** (Discus Dental). Final compaction should be performed with a large plugger, the crown cleaned completely of all gutta-percha and sealer, the root fillings sealed off with an adhesive dentin sealant, and the final coronal restoration placed (Figure 7-11F). Do not be disturbed if a tiny “puff” of sealer exudes from the apex; it will gradually resorb.

Unquestionably, the warm gutta-percha/vertical condensation obturation renders the most compact filling to date, and also fills many of the patent accessory/lateral canals. However, it is slower and more painstaking than the lateral compaction method, and it requires additional heating and back-filling devices.

Another variation of the warm gutta-percha/vertical condensation technique is the **System B** Continuous Wave of Obturation developed by Buchanan and marketed by SybronEndo. The **System B Heat Source** (Figure 7-12A) monitors the heat at the tip of the heat-carrier pluggers. After the primary gutta-percha cone has been tested for fit, it is cemented into place and followed by the “hot-tip” plugger set for 200°C and driven into the gutta-percha for about 2 seconds. The heat switch is then released, and the cold plugger is held there for an additional 10 seconds. To remove the plugger, the heat is again activated for 1 second and the “cold” plugger is withdrawn. This should fill the apical third of the canal and any accessory canals. The remainder of the canal may be filled by the **Obtura** gutta-percha gun.

A hybrid technique of both lateral and vertical compaction was developed by Martin, who developed the **Endotech II Thermal Condensor** (MediDenta; Figure 7-13). It contains batteries that heat a spreader/plugger used in a similar manner of heating and laterally/vertically compacting the gutta-percha. When a button is pressed on the device, it heats up to warm the gutta-percha. At the release of the button, the spreader cools off and becomes a plugger. Martin has

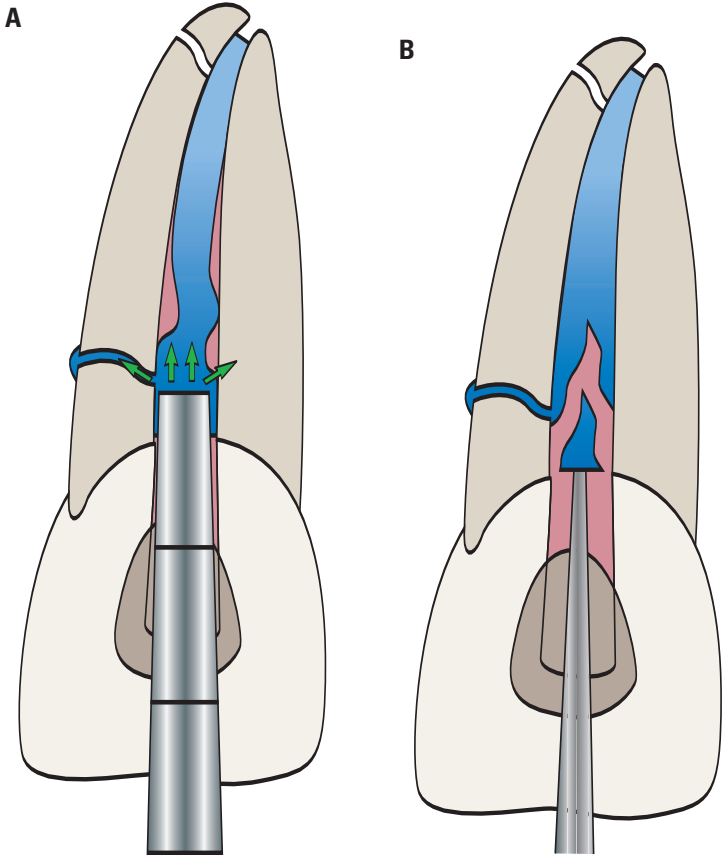


Figure 7-11. Warm gutta-percha/vertical compaction technique. Prior to this stage of the technique, the master gutta-percha point has been fitted to the canal (see Figure 7-9) and the three sizes of pluggers have been tested in the canal. Sealer has been applied, the master cone has been inserted into position, and the coronal excess has been removed. **A**, The largest plugger compacts warmed gutta-percha into a bolus. Note the midroot lateral canal being obturated. **B**, First selective gutta-percha heating and removal. **Continued on next page.**

C

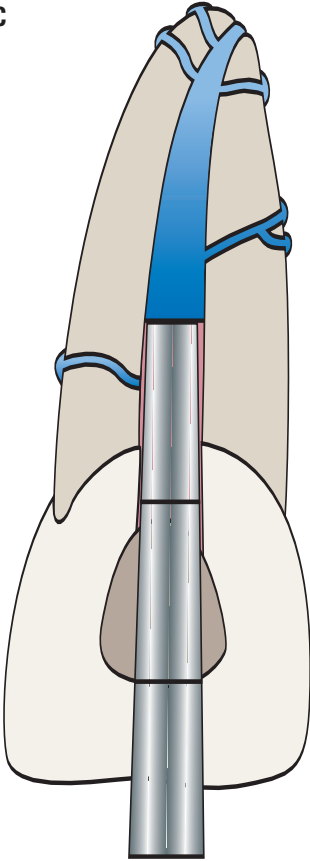


Figure 7-11. Continued. C, Warmed gutta-percha has been compacted to fill the apical half of the canal and lateral canals. **D,** If the gutta-percha “gun” (Obtura II, Obtura/Spartan) is used to back-fill the remainder of the canal, the gun’s needle is inserted to the apical segment and then backed out, leaving a deposit. Plasticized gutta-percha is compacted to complete the obturation of the canal orifice. **Continued on next page.**

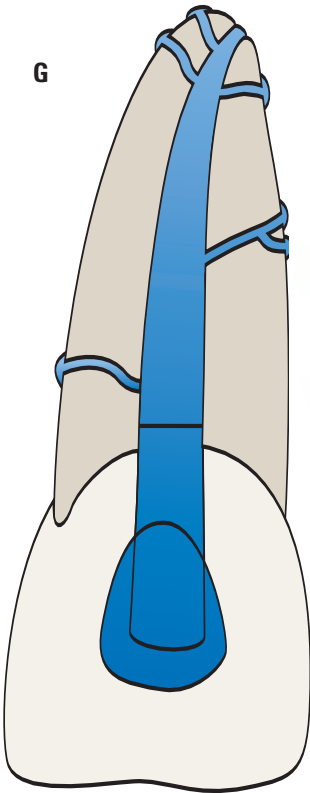
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F



Figure 7-11. Continued. **E**, HotShot warm gutta-percha "gun". **F**, Elements Obturation Unit (Sybron Endo), capable of numerous objectives in warm gutta-percha techniques, from System B to down-pack to backfill.

G, Gutta-percha and sealer are removed to below the free gingival level, the crown is thoroughly cleaned, an adhesive bonding agent is used to seal the root filling, and a final resin restoration is placed.



Figure 7-12. A, System B Heat Source (SybronEndo) monitors the heat at the tip of the heat-carrier pluggers. “Hot-tip” pluggers are set for 200°C to heat the apical gutta-percha. When the heat switch is released, the plugger becomes cold and is used to compact the apical third gutta-percha. **B, Discus HotTip** (Discus Dental, Culver City, CA) Cordless, with essentially the same characteristics as the System B.



Figure 7-13. Endotech II thermal condenser (MediDenta) is used as a heat source/cold source spreader/plugger in a hybrid obturation technique that encompasses both lateral compaction and warm gutta-percha vertical compaction.

also developed gutta-percha points embedded with either iodoform or tetracycline, that is gradually released as a disinfectant.

SOLID-CORE CARRIERS

First developed by Johnson in 1978, solid-core carriers developed from gutta-percha applied to a regular endodontic file (Figure 7-14A) to the modern version of the **ThermaFil Obturators**, (Dentsply/Tulsa Dental) **a.k.a. DensFil** (Dentsply/Maillefer; Figure 7-14B). These consist of a plastic central carrier coated with alpha-phase gutta-percha shaped like the tapered root canal. Canal preparation is much the same as for any obturating technique—clean and taper the shape and remove the smear layer. The ThermaFil Obturator, previously selected for the size to best fill the canal, is warmed and softened in the **ThermaPrep Plus** oven (Figure 7-14C). After the canal has been dried and **Thermaseal a.k.a. AH Plus**, sealer has been applied, the softened ThermaFil obturator is immediately positioned to full working length in the canal. When it has been determined radiographically that the obturator is fully in place, the shaft is severed about 2 mm above the canal orifice with a no. 37 inverted cone bur. The handle should be held during this operation to prevent dislodgment. It has been suggested that four or five small segments of gutta-percha should finally be compacted around the carrier. If the canal is especially wide, a spreader may be used to laterally condense additional gutta-percha points around the carrier. If a post is to be placed, a **Prepi Bur** (Dentsply/Tulsa) should be used to remove enough plastic carrier and gutta-percha for post space. The Prepi Bur is a smooth, round ball that removes the extra filling material by friction.

Initially, endodontic specialists condemned ThermaFil as ineffective. However, subsequent research proved these fillings to serve quite adequately, comparable to lateral compaction and System B

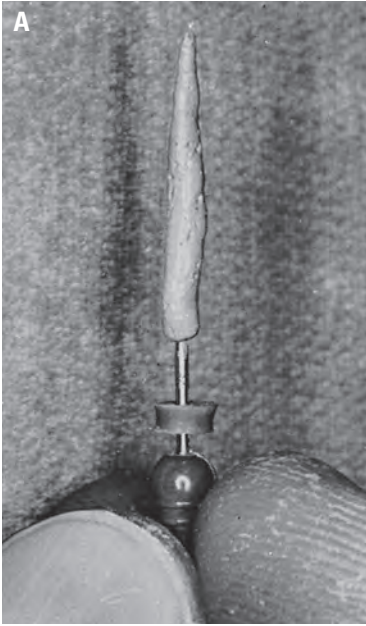


Figure 7-14. Solid core carriers.

A, Original handmade gutta-percha obturator mounted on a regular endodontic file. Courtesy of W. B. Johnson (circa 1978). **B**, Thermafil Carrier (Dentsply/Tulsa Dental), a modern, plastic, central-carrier coated with alpha-phase gutta-percha and shaped much like a tapered root canal. It comes in many ISO sizes. Courtesy of Dentsply/Tulsa Dental. **C**, Therma Prep Plus oven (Dentsply/Tulsa Dental) that thermostatically controls the plasticizing of the ThermaFil obturators. Courtesy of Dentsply/Tulsa Dental.



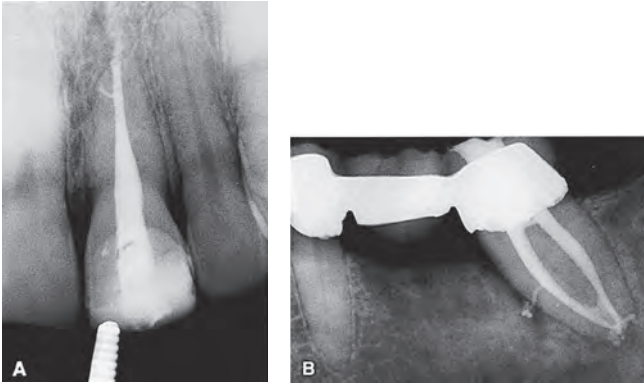


Figure 7-15. Obturation with TheraFil. **A**, Central incisor filled with a size 60 Therafil obturator-plastic carrier. Note the lateral canal near the apex. **B**, Both mesial and distal canals filled with size 45 TheraFil obturators. Note the apical fill and lateral canal. Courtesy of W.B. Johnson.

obturation. A small puff of cement was often extruded at the apex (Figure 7-15).

If “imitation is the highest form of flattery,” similar obturators, sealer, instruments, and devices have been modified from gutta-percha solid-core fillings into resin based fillings. **RealSeal SE** (SybronEndo) a.k.a. **Epiphany** as a sealer, coupled with **Resilon** coated solid-core obturators (Figure 7-16A) prepared in a Resilon Oven (Figure 7-16B), mimic the gutta-percha era with the methacrylate era.



Figure 7-16. RealSeal SE, (SybronEndo) Resilon methacrylate points used with RealSeal (a.k.a.) Epiphany sealer.



Figure 7-16. RealSeal thermo oven used to warm Resilon points for obturation.

APICAL THIRD FILLING

Long ago it was noted that dentin chips left in the periapical tissue often formed calcification around them. Taking up on this observation, the group at Oregon School of Dentistry advocated developing a dentin chip plug after the canal was cleaned, shaped, and disinfected. Chips were developed within the canal and plugged into the apex, filling the apical region with 1 mm to 2 mm of chips (Figure 7-17A). These would not only serve as stimulants for cementum formation but would also plug the apical foramen so that no cement or gutta-percha could escape (Figure 7-17B). Evidently, it is the hydroxyapatite in the dentin that acts as the stimulant for new cementum formation (Figure 7-17C).

Based on this finding, hydroxyapatite can be developed by introducing two aqueous calcium phosphate solutions, one acidic and one basic, at the apex. The two then form a hydroxyapatite plug. This has been shown to form a cemental cap over the apex. The same can be said for a calcium hydroxide apical plug. However, the most recent approach is to place **mineral trioxide aggregate (MTA)** as an apical plug (Figure 7-18). This technique has proved very successful. The remainder of the canal is then filled by any chosen method.

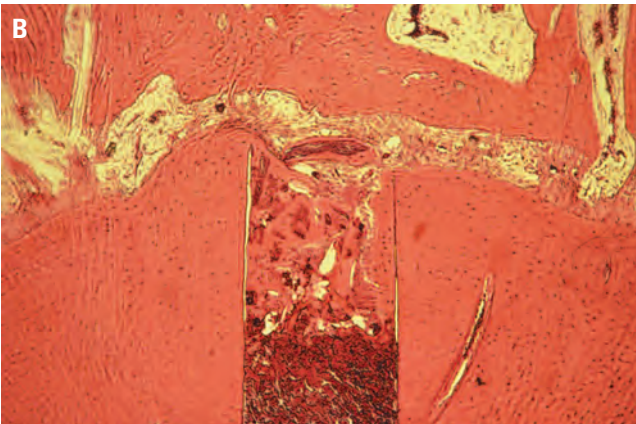
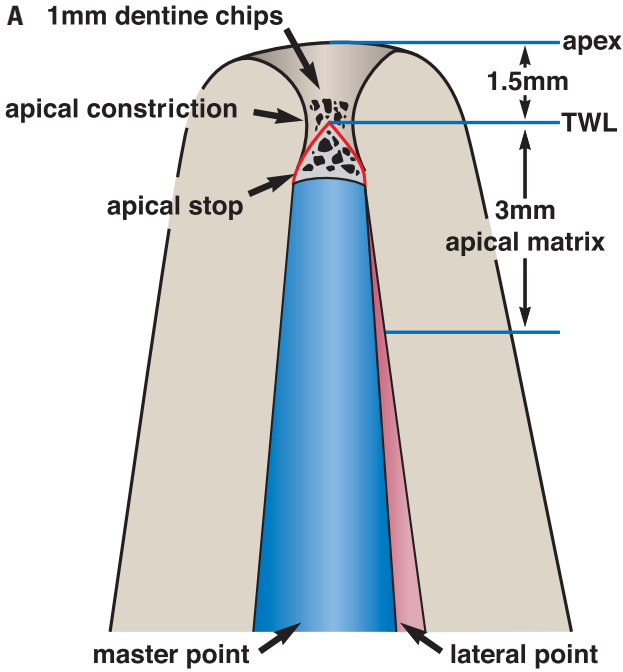


Figure 7-17. A, Dentine chips compacted into the last 2 mm of a preparation, spilling over from the true working length to stimulate cementogenesis. Gutta-percha and sealer complete the obturation. Courtesy of Oregon Health Sciences University. **B,** Low-power view of the periapex with acute inflammatory resorption of dentin and cementum following pulpectomy. The canal is filled with the calcium hydroxide mixture. New hard tissue forming across apex in response to $\text{Ca}(\text{OH})_2$. Courtesy of B. G. Jeansonne. *Continued on next page.*

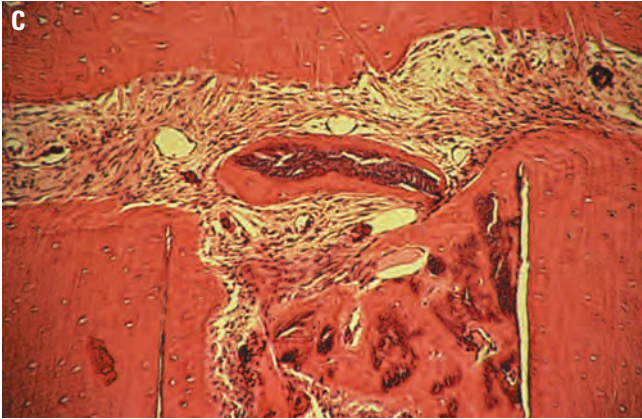


Figure 7-17. Continued. C, Higher-power view of the apical “cap” courtesy of B.G. Jeansonne.

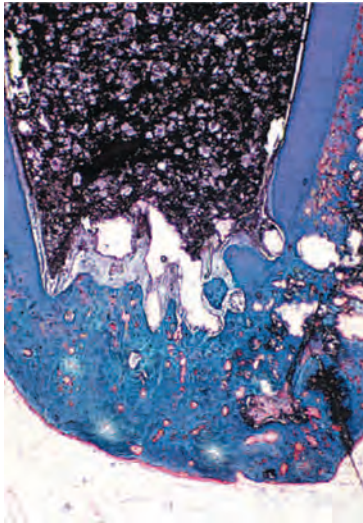


Figure 7-18. Cementum formation at the periapex following the introduction of MTA in the canal. Reproduced with permission from Shabahang S, Torabinejad M, et al. J Endod 1999;25:1–5.

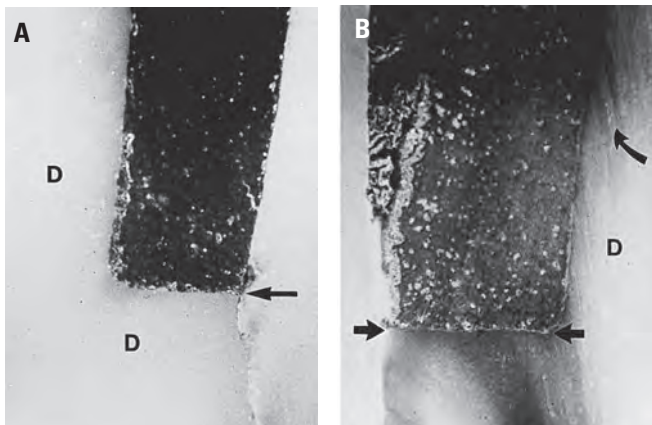


Figure 7-19. Comparative study in microleakage between resin adhesive and copal varnish. **left**, Amalgam filling bonded to dentin (*D*) and cementum (*arrow*) with Amalgambond (Parkell Co.): no microleakage. **right**, Amalgam filling lined with Copalite: gross microleakage at gingival floor (*arrows*) as well as from upper margin (*curved arrow*). Courtesy of A.H.L. Tjan and D.E. Tan.

Microbial leakage studies over 30 days showed the synthetic filling to be superior to gutta-percha and a cement sealer.² Typical obturation techniques of lateral condensation or warm vertical compaction are used (Figure 7-18). Less pressure needed for compaction with these materials should lead to fewer vertical fractures later on.³

If properly executed, any of the methods presented here will ensure successful obturation of the canal. However, one must be warned again that **long-range success** depends on the perfection of the final coronal restoration. Microleakage around a crown, inlay, amalgam, or composite may eventually lead to bacterial contamination as far away as the apex. Every precaution must be taken to make sure the final restoration is leak proof (Figure 7-19).

Slowly, it appears endodontics is moving out of the nineteenth-century world of gutta-percha and zinc oxide-eugenol and into the twenty-first century of resin chemistry, something I predicted nearly 40 years ago would happen.⁴

REFERENCES

1. Ingle JI, Bakland LK. Endodontics. 5th ed. Hamilton (ON): BC Decker; 2002.
2. Shipper G, Orstavik D, Teixeira, FB, Trope M. An evaluation of microbial leakage in roots filled with a thermoplastic synthetic polymer based root canal filling material (Resilon) or with gutta-percha. J Endod 2004;30:342.
3. Teixeira FB, Teixeira ECN, Thompson JY, Trope M. Fracture resistance of roots endodontically treated with a new resin filling material. J Am Dent Assoc 2004;135:646.
4. Ingle JI. Endodontics. 1st ed. Philadelphia: Lea & Febiger; 1965.

Endodontic Surgery

During the past 20 years, endodontics has seen a dramatic shift in periradicular surgery and the part it plays in the delivery of endodontic services. Previously, it was considered the treatment of choice when nonsurgical treatment had failed or there was the presence of large or intruding periapical lesions, overfilled canals, incomplete apical formation, or destruction of the apical constricture by overinstrumentation. Today, a more conservative approach solves many of these problems *sans* surgery (Figure 8-1). (See Chapter 3, Apexum)

INDICATIONS AND CONTRAINDICATIONS

Indications to endodontic surgery are outlined in Table 8-1, and some are illustrated in Figures 8-2 and 8-3. Contraindications are as follows:

Patient's medical status. If the patient reports any major system disorder—cardiovascular, respiratory, digestive, hepatic, renal, neural, immune, or musculoskeletal—a thorough medical history is mandatory and the patient's physician must be consulted. Surgery in the first trimester of pregnancy is also ill-advised, and in the third trimester is uncomfortable for the patient. Most miscarriages take place in the first trimester, and the endodontic surgeon may be blamed for the miscarriage. Before any surgery is undertaken, be sure the patient is not on aspirin, warfarin (Coumadin) or ximelagatran (Exanta), one of the newest anti-clotting drugs.

Anatomic considerations. Examine the nasal floor, maxillary sinus, mandibular canal and its neurovascular bundle, and mental foramen and its neurovascular bundle for adequate visual and mechanical access.

Limited skill and knowledge of the surgeon. One must know one's limitations. Endodontic surgical procedures are best done by

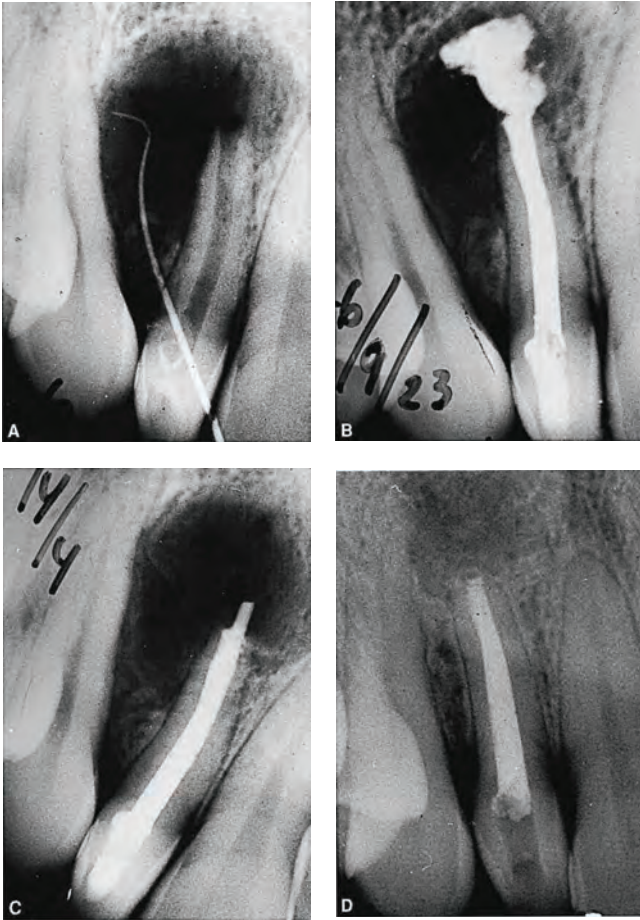


Figure 8-1. **A**, Dens invaginatus (dens in dente), with accompanying apical/lateral cyst. Gutta-percha sound is placed through a draining stoma. **B**, Calcium hydroxide and iodoform paste fills the débrided canal. **C**, The drainage ceased and $\text{Ca}(\text{OH})_2$ resorbed away in 8 months, at which time the canal was filled with gutta-percha and AH26 sealer (Dentsply/DeTrey) no surgery. **D**, Follow-up radiograph 15 years after obturation. Courtesy of C.C. Mexia de Almeida, Lisbon, Portugal.

a trained endodontic specialist. The standard of care in dentistry is that practiced by the region's specialists in any given dental discipline. Many oral surgeons practice endodontic surgery but fall far short in the intracanal preparation so necessary for a successful outcome.

Table 8-1

INDICATIONS FOR ENDODONTIC SURGERY

1. Surgical drainage
2. Failed endodontic treatment
 - a. Irretrievable filling material (see Figure 8-2)
 - b. Irretrievable post
3. Calcification of pulp space
4. Procedural errors
 - a. Instrument breakage (see Figure 8-2)
 - b. Non-negotiable ledging
 - c. Root perforation
 - d. Gross overfilling (see Figure 8-2)
5. Anatomic variations
 - a. Root dilaceration
 - b. Apical root fenestration (see Figure 8-3)
6. Biopsy
7. Corrective surgery
 - a. Root resorptive defects
 - b. Root caries
 - c. Hemisection
 - d. Bicuspidization
8. Replacement surgery
 - a. Intentional replantation
 - b. Post-traumatic replantation
 - c. Osseointegrated implant



Figure 8-2. **A**, Fractured instrument protrudes past the apical foramen. **B**, Overinstrumentation has led to apical perforation and a fractured root tip (arrow) that must be removed surgically. **Continued on next page.**



Figure 8-2. Continued. C, Overextended filling caused physical irritation with pain and inflammation.

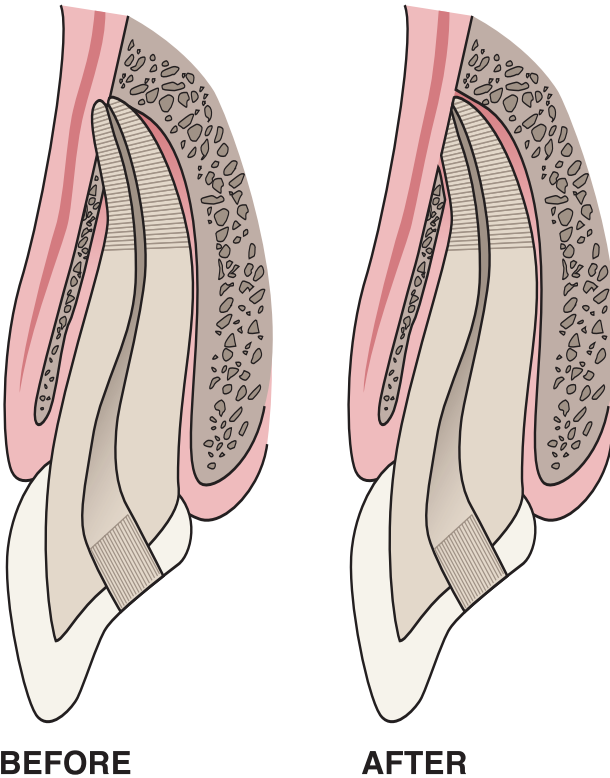


Figure 8-3. Beveling of the root apex to a subcortical level relieves facial peri/radicular tenderness.

CLASSIFICATION OF PROCEDURES

Endodontic surgery is performed to remove causative agents of periradicular pathosis (Figure 8-4) and restore the periodontium to a state of functional health. Classification of these procedures is presented in Table 8-2.

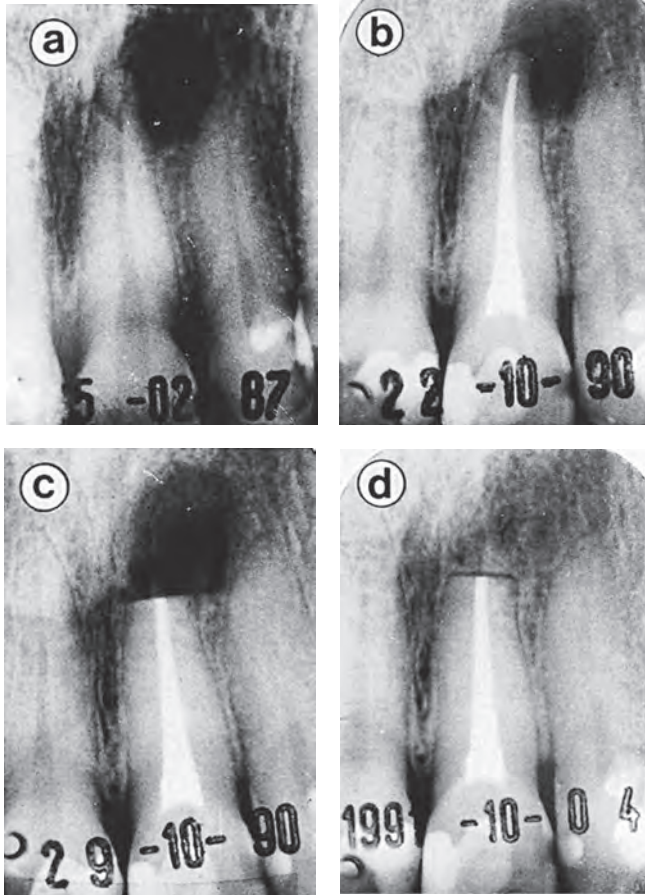


Figure 8-4. Cyst enucleation necessary to achieve healing. **A**, Pretreatment. **B**, No healing 44 months after the root filling. **C**, Apicoectomy and cyst enucleation. **D**, Complete healing 1 year later. Courtesy of P.R.N. Nair and colleagues.

Table 8-2

CLASSIFICATION OF ENDODONTIC SURGICAL PROCEDURES

1. Surgical drainage
 - a. Incision and drainage
 - b. Trephination (fistulization)
2. Periradicular surgery
 - a. Curettage
 - b. Biopsy
 - c. Root-end resection (see Figure 8-4), preparation, and filling (retrofill)
 - d. Corrective surgery: perforation repair (iatrogenic [mechanical]; resorptive [internal and external]); root resection; hemisection
3. Replacement surgery (extraction/replantation)
 - a. Endodontic implants
 - b. Osseointegrated implants

SURGICAL DRAINAGE

Surgical drainage is indicated when purulent or hemorrhagic exudate forms within the soft tissue or alveolar bone—an abscess. Pain is reduced and morbidity shortened following the surgical release of pressure and infection. This can be accomplished by either **incision and drainage** or **trephination** of the alveolar cortical plate.

Incision and Drainage

Initially, when a periradicular abscess forms the area appears and feels hard (indurated). This is when the lesion is the most painful. It is seldom productive to drain an abscess at this stage. The patient should be started on antibiotics and the abscess encouraged to “point” with the use of “hot holds,” a half-teaspoon of salt in 250 mL (8 ounces) of hot water, repeatedly held in the area, to speed the inflammatory process. The area becomes fluctuant (soft) once the purulent/hemorrhagic (pus) abscess breaks through the cortical bone. Timing is of the essence! If allowed to continue, the lesion could spread laterally and become full-blown cellulitis (Figure 8-5). It is at the fluctuant stage that the abscess should be **incised and drained**.



Figure 8-5. Massive cellulitis that developed from an infected abscessed lower molar. Incision and drainage as well as antibiotic therapy were prescribed. Courtesy of J.F. Siqueira Jr., Rio de Janeiro, Brazil.

After nerve block anesthesia has been established, the proper instruments for incision and drainage should be assembled (Figure 8-6). If complete anesthesia is not achieved, local infiltration with mepivacaine is in order, carefully avoiding the infected area. Failing all else, supplemental freezing of the area with ethyl chloride spray has been recommended. Avoid patient breathing the spray, an anesthetic.

The surgical area should be isolated with 5 cm × 5 cm (2 in. × 2 in.) gauze squares, and a **horizontal** incision with a no. 11 or no. 12 blade should be made at the lowest dependent base of the fluctuant area (Figure 8-7A). This is immediately followed by aspiration of the purulent material (Figure 8-7B). To ensure total evacuation, a curved hemostat may be inserted through the incision to the base of the abscess and the beaks spread to drain the last of the pus (Figure 8-7C). It has not been found necessary to insert a drain in the incision. Healing should occur within a week and, when comfortable for the patient, endodontic treatment may be instituted. Regular orthograde endodontics is the preferred treatment (Figure 8-8).

Likely candidates present themselves with apical pain and no swelling, but drainage of the immediate apical region seems necessary. However, apical trephination through the canal should be



Figure 8-6. A well-organized incision and drainage kit is essential for efficient accomplishment of drainage problems. Courtesy of Loma Linda University School of Dentistry, Loma Linda, CA.

attempted first. The exact tooth length is calculated and, with a fine file, the apical orifice is just perforated. This opening is then enlarged with up to a no. 20 or no. 25 file in the hope that drainage will occur through the canal. If this does not happen, then a surgical approach may be indicated. Another surgical indication may be the canal with a post present.

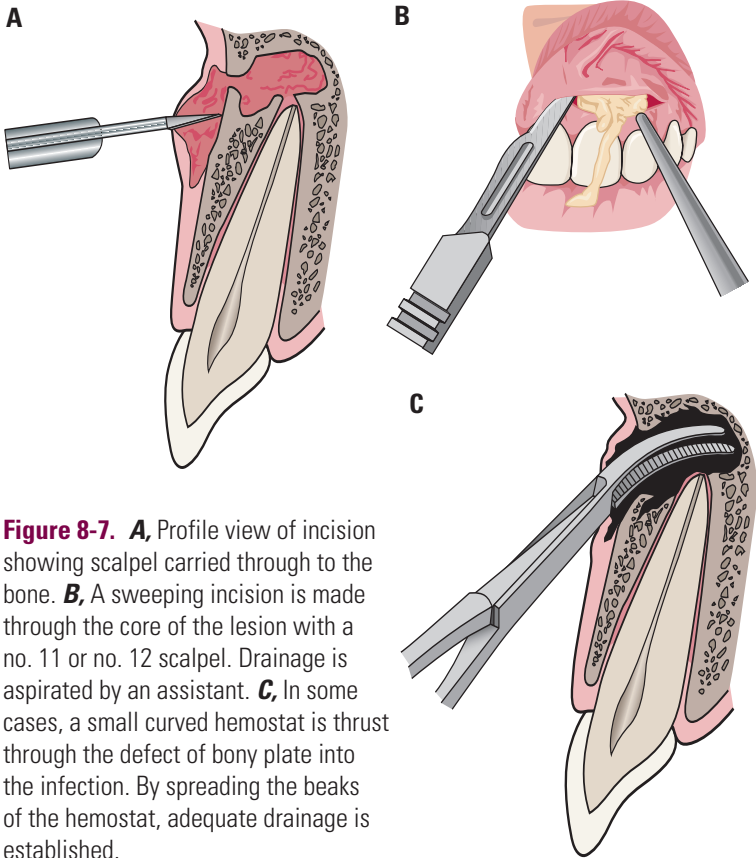


Figure 8-7. **A**, Profile view of incision showing scalpel carried through to the bone. **B**, A sweeping incision is made through the core of the lesion with a no. 11 or no. 12 scalpel. Drainage is aspirated by an assistant. **C**, In some cases, a small curved hemostat is thrust through the defect of bony plate into the infection. By spreading the beaks of the hemostat, adequate drainage is established.

Trephination Drainage

Trephination is a limited-use procedure and is fraught with peril. Cortical trephination involves making an incision through the mucoperiosteal tissues and then perforating with a No. 6 or no. 8 round bur through the cortical plate to the inflamed area (Figure 8-9). Once the cortical plate is perforated, a large reamer can often be used to open the cancellous bone through to the fluid collection. Hopefully, drainage ensues. Perfect radiographs, careful measurements, and tooth alignment in the arch are imperative. The floor of the nose, the maxillary sinus, and the mandibular canal must be avoided.

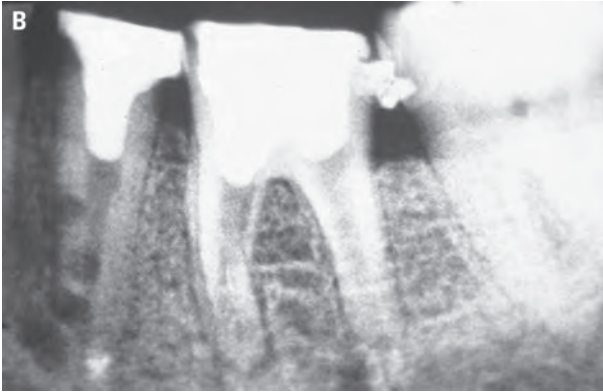
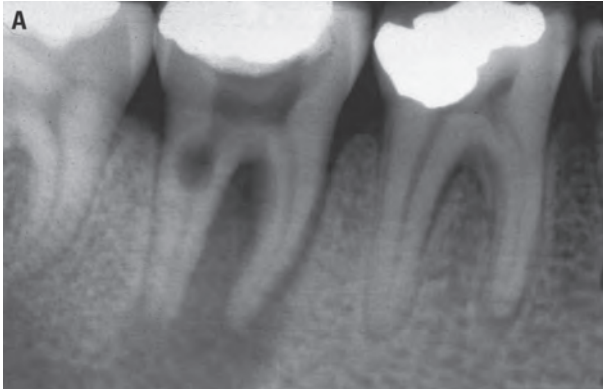


Figure 8-8. **A–C,** Examples of roots perforated during inaccurate trephinations for drainage.

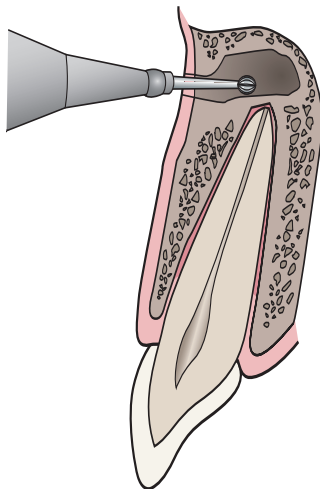


Figure 8-9. Surgical trephination of an intact labial cortical plate to relieve the liquid and gas pressure of an acute apical abscess. Accurate pinpointing of the lesion is done with the use of a radiograph.

PERIRADICULAR SURGERY

In the case of a **failed nonsurgical** endodontic treatment, the first choice of correction is **re-treatment** by the usual orthograde approach. Periradicular surgery may be the answer to retaining the tooth in the event other factors preclude an approach through the canal (see Figure 8-2), such as a perfect jacket crown should be preserved or a well-positioned post is in place. Surgery should not be considered the first approach in the event a periapical lesion is present. One exception might be the presence of an apical cyst, with the caveat that many so-called granulomas very much resemble a cyst on radiographs. In any event, some cysts continue to grow in spite of the fact a perfect root canal filling has been completed. To be on the safe side, if a cyst is suspected it should be removed surgically.

Informed Consent

Good patient communication is essential. Informed consent is mandatory, and a signed informed consent is advised. The risks involved, the short possibilities of pain and swelling, the probable outcome,



Figure 8-10. Suggested surgical instrument setup includes burs, sutures, hemostats, scalpels, curettes, retractors, mirrors, and sponges. Efficient offices have these setups covered, sterilized, and stored.

and the alternatives should all be explained. Use of a chlorhexidine (Peridex) mouth rinse prior to surgery is recommended.

Profound block anesthesia and local injections to control hemostasis are imperative. Lidocaine (Xylocaine) is the anesthetic of choice—1/100,000 epinephrine for the block and 1/50,000 epinephrine for the small local injections. The surgical procedure is preceded by placing 5 cm × 5 cm (2 in. × 2 in.) gauze squares between the posterior teeth for the patient to bite on.

A sterile surgical tray should contain all the required instruments for incision, retraction, curettage, resection, retrofilling, and suturing and should anticipate any contingency (Figure 8-10).

Flap Design

Vertical relaxing incisions are used to gain access to the area of the lesion (Figure 8-11). The vertical incisions must be made **over the solid interdental bone**. Horizontal incisions, such as the semilunar flap, are to be avoided. They may cross a bony fenestration that will later “heal” as a dehiscence (Figure 8-12). The **double vertical (rectangular) flap** is the preferred flap in most cases (see Figure 8-11C). In the posterior mouth, the triangular flap is recommended (see Figure 8-11A). Care must be taken that the termination of the incision at the papilla does not split the papilla; upon healing, the split would leave a periodontal defect (Figure 8-13). To avoid this

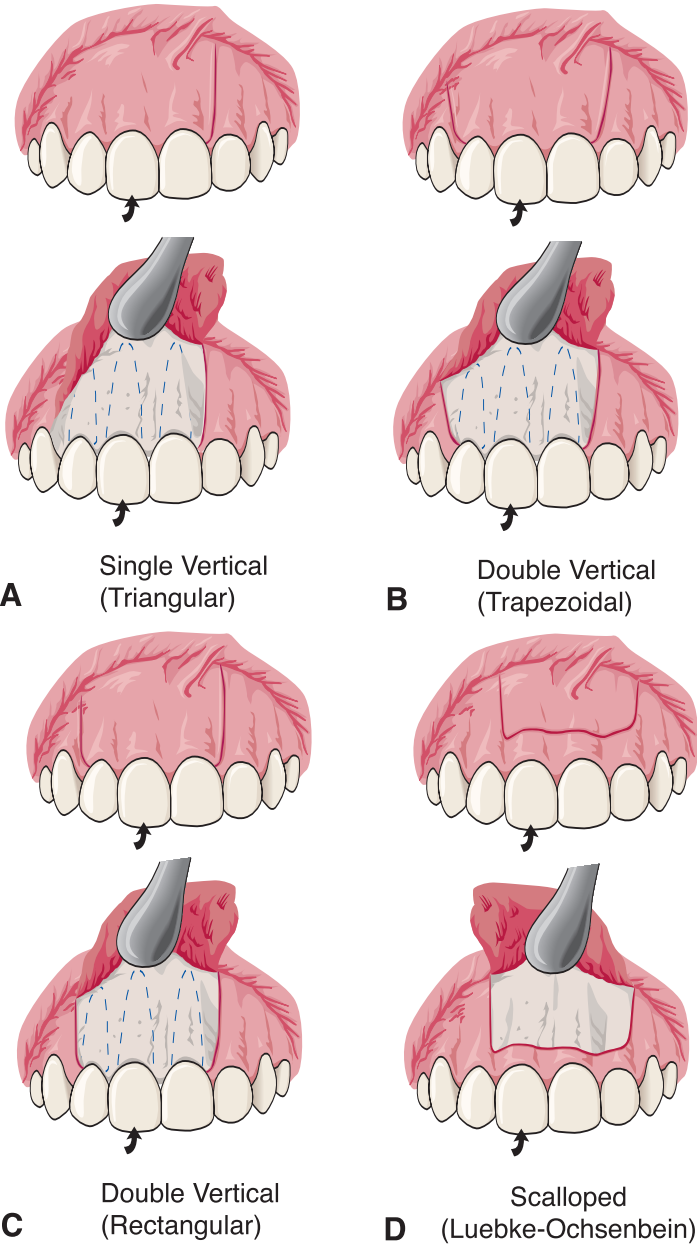


Figure 8-11. Surgical flap design and flap nomenclature. All vertical incisions must be on solid interdental bone. Horizontal incisions must not cross suspected bony fenestrations. **A**, Single vertical. **B**, Double vertical, trapezoidal. **C**, Double vertical, rectangular. **D**, Scalloped.

possibility, the **scalloped flap** can be used (see Figure 8-11D). The horizontal incision should be made just apical to the free gingival groove but should not be used if it will cross a radicular eminence with a possible bony fenestration (see Figure 8-12). The width of the flap should include at least two approximating teeth to allow plenty of freedom in retraction and vision (see Figure 8-11).

The **vertical incisions** must be on solid bone, well clear of the area where cortical bone is to be removed or has been destroyed



Figure 8-12. **A**, Development of a dehiscence following a horizontal incision across a bony fenestration of a lower canine owing to its labial displacement. **B**, An attempt at repair worsens the defect. *Continued on next page.*



Figure 8-12. Continued. C, Final dehiscence. Cemental caries later developed. Note the labial crossbite of the tooth.

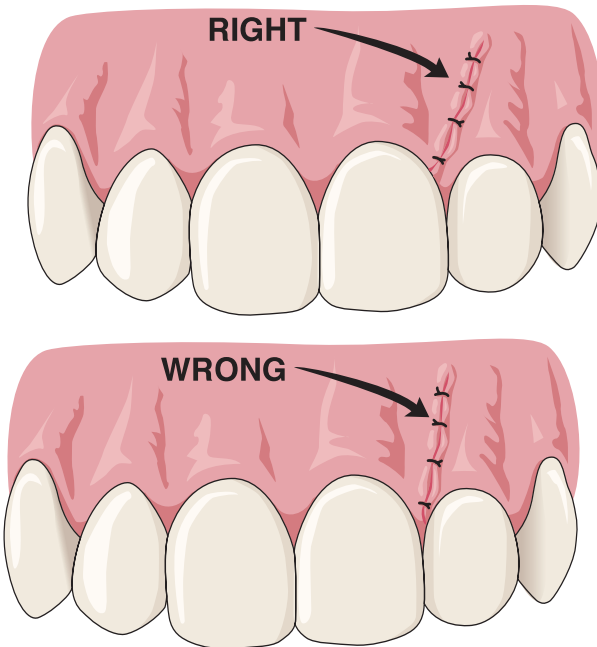


Figure 8-13. Incisions that split the papilla do not heal well and may leave a split-papilla periodontal defect.



Figure 8-14. Scalpel blades for surgical incisions. *From the top:* Microsurgical blade, no. 15, no. 12, and no. 11.

by the infection. The **horizontal incision** is made with a small scalpel blade (no. 15C) in the gingival sulcus of the involved teeth (Figure 8-14). This frees up the marginal gingivae and interdental papillae so that the flap may be retracted vertically. It should be a full-thickness flap involving the entire mucoperiosteum—marginal, interdental, and attached gingivae, alveolar mucosa, and periosteum. This flap carries the blood supply with it.

Flap reflection begins at the coronal termination of the vertical incisions. Undermining elevation follows along the gingival margins; then the entire flap is reflected apically. It is held in place with a broad retractor, on solid cortical bone, with light but firm pressure (Figure 8-15). The entire surgical area should be constantly bathed with water or, preferably, saline to prevent dehydration of the tissues. Aspiration is necessary to improve vision.

The periapical bone should now be explored with a sharp curette. Often the disease process has destroyed or materially thinned the cortical bony plate. If this is the case, the thinned bone can easily be removed with a curette exposing the granulomatous tissue beneath. In the event the cortical bone is intact and firm, it must be removed with burs (Figure 8-16). This is almost always true in the posterior mouth, especially in the mandible. The exact spot of entry is of major consideration. The radiographs must be carefully



Figure 8-15. Flap retractors. *From the top:* No. G3 (Hu Friedly); no. 3 (Hu Friedly); Rubinstein (JedMed Co.).

studied and the true tooth length noted. Beware of foreshortened or elongated images! Also, the angulation of the root, mesial to distal as well as labial to lingual, must be determined. One does not want to drill into the root itself or one of its neighbors (see Figure 8-8). Using a sterile millimeter ruler, the length of the tooth and its angulation are marked at the entry site. If in doubt, drill a small defect in the bone, place a tiny piece of film-packet foil in the defect, and radiograph it. The image will provide guidance to the apex.

Once this spot is determined, **bone removal** begins. It is best to use a large, sharp, round bur (No. 6 or No. 8) with light brush strokes in a high-speed handpiece (see Figure 8-16). It is **imperative** that copious **water coolant** be applied to the head of the bur, not only to cool the area but to remove debris from bur's flutes. It goes

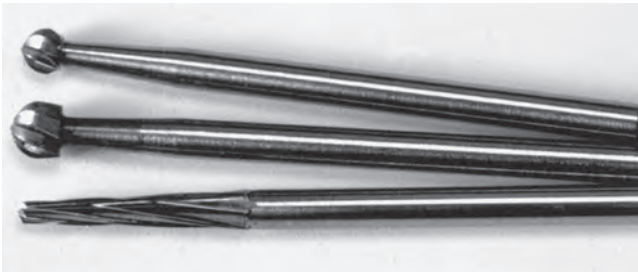


Figure 8-16. Burs for hard tissue removal. *From the top:* FG no. 6; no. 8; no. H267 (Brassler).

without saying that aspiration is necessary. Once the cortical plate is perforated, switch to a fissure bur to widen access to the lesion and the apex.

In the anterior mouth, a straight handpiece can be used. However, in the posterior mouth a contra-angled handpiece is necessary. The **Impact Air 45× handpiece** is recommended, not only for its unique angle but also for the fact that the air is exhausted to the rear of the turbine and not into the surgical site.

The diseased tissue is now removed using curettes, both surgical and periodontal. If discomfort is encountered during curettage, lidocaine (1/50,000 epinephrine) should be injected into the lesion. If possible, the granuloma or cyst should be removed intact and sent for biopsy. Once the body of tissue is detached, fine curettes are used to remove the tissue remnants and to curette the surface of the root.

In the event one is dealing with a large lesion that involves other roots or nears the floor of the maxillary sinus or the floor of the nose, great care must be exercised not to invade these tissues. A number of neighboring vital teeth have been devitalized when their blood supply has been destroyed during curettage.

At this point, if one is sure the previously placed root canal filling is optimal and that curettage was the single objective (as in the case of an apical cyst), closure of the surgical site is in order. On the other hand, if root-end resection or a root-end filling is contemplated, the next step is undertaken.

Root-End Resection

An often-treated but persistent periapical lesion is a perfect example of an indication for root-end resection. Investigation of root ends from cases refractory to healing have shown colonies of bacteria clinging to root surfaces (see Figure 3-2). Their removal, along with the root tip, combined with antibiotic therapy is often necessary to bring about healing. Persistent discomfort following “successful” endodontic treatment is another example of a situation that requires root-end resection or beveling (see Figure 8-3). Instances of root-end perforations, fractures, resorptions, unfilled curvatures or multiple unfilled canals, broken instruments through the apex, and gross overextension of filling material might also call for resection (see Figure 8-2).

How does one choose which bur to use in resection and how much root to remove? It appears that a plain fissure bur in a low-speed handpiece not only provides the smoothest resected surface but the least distortion of the previous orthograde gutta-percha filling. Remove as little root as necessary! Most lateral, accessory, and secondary canals lie within the last 2 mm to 3 mm of the root end, and these are best removed. And of course, any area damaged by a procedural error should be resected. But remember, the length of the tooth that is retained, the more retentive and functional the tooth will be. Most often following root-end resection, a **retrofilling** is necessary to ensure success.

Root-End Preparation

For years, root ends have been resected at a 45° angle facing labially, but recent research has shown that a beveled surface opens many more dentinal tubuli that may contain or retain bacteria than does the 90° resection. It is now *de rigueur* to amputate at a 90° angle. Of course, this puts one at a **sight disadvantage**: How does one see the end of the flat cut surface? Of course, the answer involves the miniature instruments and **mirrors** developed by such innovators as Carr and Rubinstein. Their use brings to mind the importance of needing loupes or a surgical microscope to magnify the root end, its orifices, isthmuses, and cracks (Figure 8-17). Although the microscope is ideal, similar results can be accomplished through increased illumination with a head-lamp and increased magnification with surgical telescopes (worn like glasses) that magnify in the range of 2.5 to 6.0 times (Figure 8-18).

Visual enhancement can also be achieved through the use of the **Endoscope**. Endoscopy consists of a tube and optical system with high-intensity light. The image captured on the endoscopic camera is projected onto a video monitor for viewing. One can visualize surgical sites as well as access openings, canal interior, fractures, and resorptive defects, all highly magnified (Figure 8-19).

To work in a blood-free environment, hemostasis is in order. This is best done by packing the cavity with Telfa Pads (Kendall) and then transferring epinephrine from a moistened Racellet cotton pellet No. 2 (1.15 mg epinephrine; Pascal) to the Telfa. It is best not to place cotton or gauze in the cavity, for if cotton fibers are inadvertently left behind they serve as a foreign body irritant and impair healing.

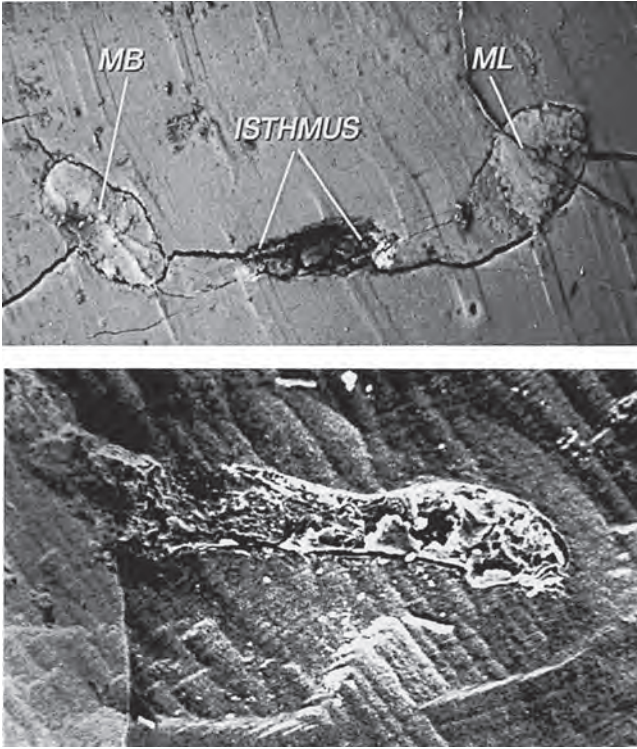


Figure 8-17. Endodontic failures revealed under surgical operating microscope following root amputation. **Top**, nondébrided isthmus connecting mesiobuccal (*MB*) and mesiolingual (*ML*) canals **Bottom**, low-power magnification reveals nondébrided unfilled portion of otherwise well-filled canal. Courtesy of G. Carr and the Pacific Research Foundation.

Instrumentation

Studies have shown that the act of root-end resection often disturbs the gutta-percha in the canal. Because one is working in micrometers, one has no way of knowing whether the gutta-percha has been freed from the walls of the preparation. It is therefore advisable to place a root-end filling each time a resection has been done. Carr and Bentkover have listed five requirements a retrofilling must fulfill:¹

1. The apical 3 mm of the canal must be freshly cleaned and shaped.



Figure 8-18. Surgeon using a fiberoptic headlamp system and $\times 2.5$ surgical telescopic loupes. Courtesy of R. Rubinstein.



Figure 8-19. The Endoscope (JedMed) greatly magnifies images of the apical surgical site on a monitor screen. The 4.0-mm endoscope can capture and magnify images by up to 50 times. The Endoscope is held on solid bone, like a retractor, so it stays focused and still. The surgeon learns to follow the action on the screen rather than on site. Screen images may also be captured with an attached camera.

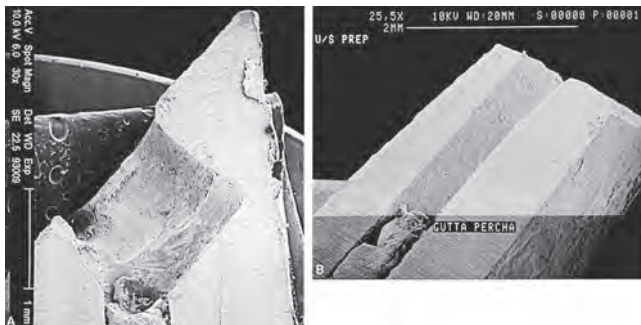


Figure 8-20. Comparison between a bur and ultrasonic root-end preparations. **Left,** Bur preparation shows a large cavity prepared oblique to the canal with a no. 33 inverted cone bur ($\times 30$ original magnification). **Right,** Ultrasonic preparation shows a clean preparation parallel to the canal ($\times 25.5$ original magnification). Courtesy of G. Wuchenich and D. Meadows.

2. The preparation must be parallel to the long axis of the pulp space (Figure 8-20B).
3. Adequate retention form must be created.
4. When present, all isthmus tissue must be removed.
5. Remaining dentin walls must not be weakened.

It is virtually impossible to make a parallel preparation in a flat-resected root-end with even a miniature handpiece (Figure 8-20A). The solution is to use the miniature ultrasonic tips (Figure 8-21) in an **ultrasonic handpiece**. Under magnification and irrigation, a parallel preparation 3 mm deep is produced. One must be careful to remove all the filling material and debris, especially toward the labial wall of the canal. Thorough inspection of the preparation with the **tiny mirror** (Figure 8-22) will ensure its cleanliness (Figure 8-23). It is probably best to use the ultrasonic instrument at the **low-power setting** to avoid the possibility of cracking the dentin during preparation.

In the event one wants to invest the time and money to purchase and learn to use a surgical microscope, it is well recommended. Its use will extend magnification from 2.5 to 30 times (Figure 8-24). The range of 2.5 to 8.0 times is best for a wide view and good depth of field. The best operating magnifications lie between 10 and 16 times. Higher-range magnifications, from 18 to 30 times, should be reserved for observing fine detail.

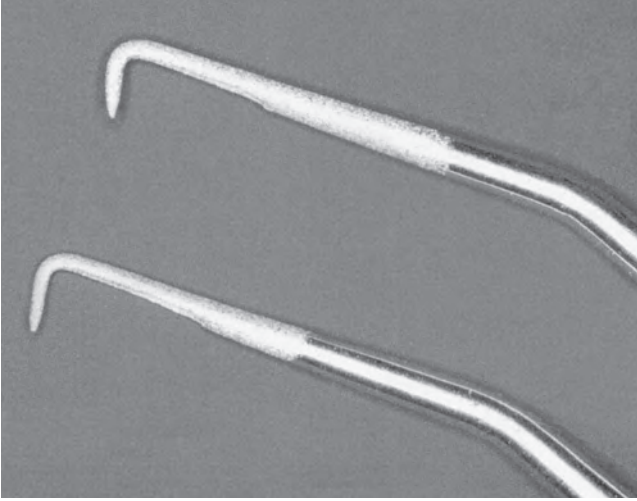


Figure 8-21. Close-up view of KiS Microsurgical Ultrasonic Instruments (Obtura Spartan). The tips are coated with zirconium nitride for faster ultrasonic cutting and are no more than 3 mm long.



Figure 8-22. Variety of tiny front-surface micromirrors for viewing a root-end resection and preparation through a microscope or surgical microscopic loupes.

Root-End Filling

Once the preparation is deemed perfect, it is best to remove the smear layer before filling. For this, MTAD (ProRoot MTAD, Dentsply/Tulsa Dental) and NaOCl are recommended. MTAD is a mixture of doxycycline, citric acid, and a detergent, Tween-80. To remove the smear layer, a mild solution of NaOCl (1.3%: one part NaOCl to four parts H₂O) is syringed into the preparation for 1 minute as it is being aspirated. The MTAD is then syringed into

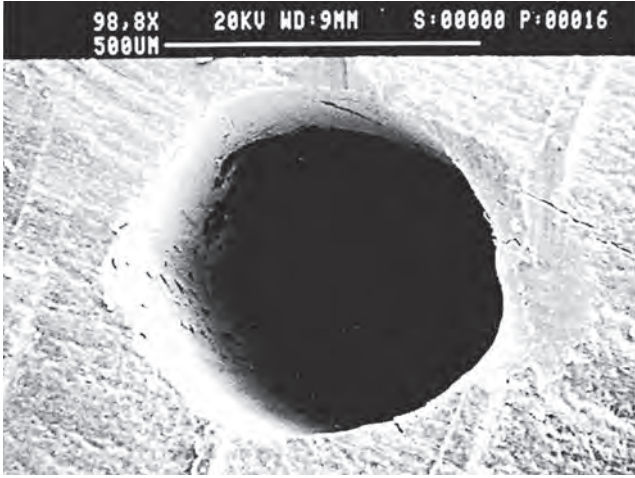


Figure 8-23. Eight-power dental operating microscopic view of root-end preparation using Carr ultrasonic tips ($\times 98.8$ original magnification). Preparation is 0.5 mm in diameter. Courtesy of G. Carr.

the preparation for 2 minutes and aspirated. Finally, the preparation is irrigated with water or saline and aspirated dry.

According to Gartner and Dorn, a suitable root-end filling material should be:²

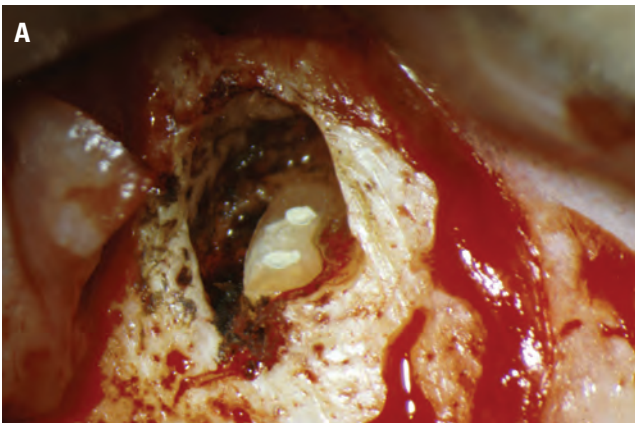


Figure 8-24. A. Final root-end fillings seen under a surgical operating microscope ($\times 8$ original magnification). Courtesy of R. Rubinstein. **Continued on next page.**

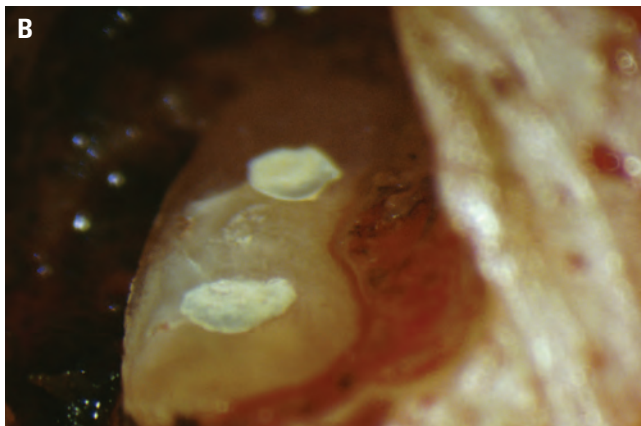


Figure 8-24. Continued. B, Final root-end fillings seen under a surgical operating microscope ($\times 26$ original magnification). Courtesy of R. Rubinstein.

1. Able to prevent leakage of bacteria and their byproducts
2. Nontoxic
3. Noncarcinogenic
4. Biocompatible with the host tissue
5. Insoluble in tissue fluids
6. Dimensionally stable
7. Unaffected by moisture during setting
8. Easy to use
9. Radiopaque.

One might add that it should not stain tissue (tattoo).

Numerous materials have been used over the years from amalgam to IRM to Super EBA to zinc oxide-eugenol. None measure up as well as **Mineral Trioxide Aggregate (MTA)** (Pro-Root MTA, Dentsply/Tulsa Dental), a dry powder. For retrofilling, it is mixed with water or saline into a dry mix, much like wet sand. It is carried to the root-end preparation with a small amalgam carrier, the **MAP System** (Roydent Dental Products). (Figure 8-25). It is then condensed with a fine plugger, and the excess is wiped away with a small cotton pellet moistened with saline. Finally, bleeding is induced and the newly placed MTA and the root-end are covered over with blood. Closure follows with flap suturing. MTA sets in 3 hours and 40 minutes. The MTA will be retained in the maxilla, but because of gravity it should be held in place with a formed clot until set in the mandible. One of its great advantages



Figure 8-25. **A**, Amalgam carrier kit (MAP System, Roydent Dental Products) with “syringe” and various size and shaped tips. **B**, Hook needle (Roydent Dental Products) used to flow mineral trioxide aggregate (MTA) into apical preparations comes in two diameters, 0.9 mm and 1.1 mm. The syringe is loaded with MTA, which is expressed through the hook needle into the apical preparation.

is its apparent stimulation of cementum production over the root end (Figure 8-26).

Soft Tissue Repositioning and Suturing

There must be a final inspection of the surgical site and removal of all excess filling materials and root fragments before suturing (Figure 8-27). Search under the surgical flap as well! The flap is then gently repositioned and held in place for a moment or two with a moistened gauze sponge. This tissue compression not only enhances intravascular clotting in the severed vessels but better approximates the wound edges as well. This also prevents a blood clot from forming **under the flap** (Figure 8-28).

The preferred suture material is gut, chromic gut, polyglycolic, or polyglactin 910 sutures. The preferred sizes are 000 and 0000. Silk

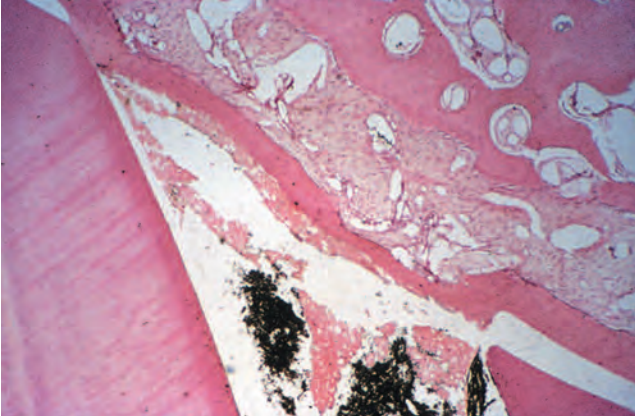


Figure 8-26. Effect of mineral trioxide aggregate (MTA) on the development of apical cementum in a monkey. MTA is the dark material in the canal at the bottom of the picture. The band of cementum formed diagonally seals off the apex, with normal connective tissue and bone above.



Figure 8-27. Radiograph taken after suturing and developed after the patient left the office. The patient had to return for the removal of the root tip.

sutures should be avoided because of their wicking action, transporting oral bacteria into the underlying tissues. Needles should be attached to the suture material, not tied. Half-curve needles are for the anterior mouth, and long-curve needles are for the posterior mouth (Figure 8-29).

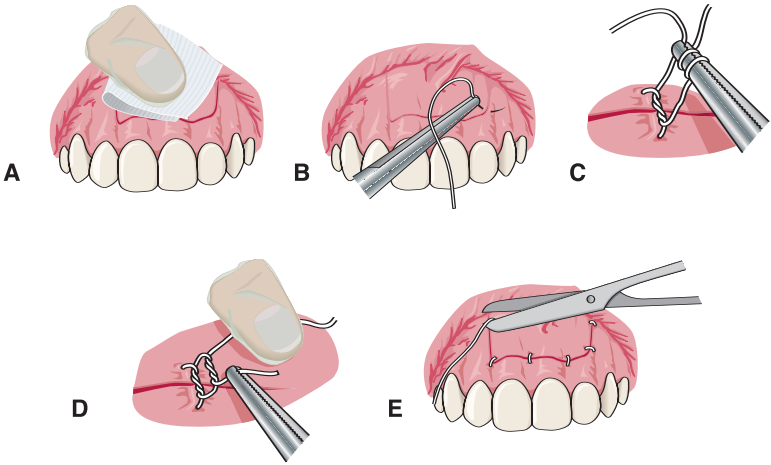


Figure 8-28. Suturing technique. **A**, Damp 5 cm × 5 cm (2 in. × 2 in.) gauze is used to smooth tissue into place. **B**, The unattached portion of the flap is sutured to the attached tissue. A 2-mm to 3-mm margin should be present between the puncture points and incision lines. **C**, Wound edges are approximated with the first tie. **D**, A surgeon's knot is used to tie the suture in place. **E**, Interrupted sutures are used to secure remainder of the flap.

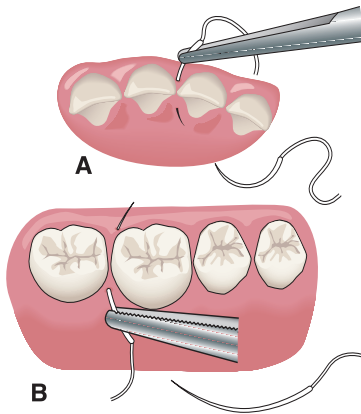


Figure 8-29. **A**, A relatively tightly curved needle such as a half-circle is best for suturing incision lines and anterior embrasures. **B**, A straighter and longer-curved needle is necessary for suturing posterior embrasures.

Interrupted suturing begins with the needle, grasped in the beaks of a hemostat, entering the labial side of the movable tissue (the flap) and then penetrating the under-surface of the fixed mucoperiosteal tissue (see Figure 8-28B). Be sure to include the periosteum to prevent tearing. The suture is pulled through with 5 cm (2 in.) left protruding, and the tie is made with a surgeon's knot.

The surgeon's knot is best made by wrapping two or three loops of the long end of the suture around the hemostat beaks and then grasping the short end of the suture and slipping the material off the beaks to pull the knot up to approximate the wound edges (see Figure 8-28C). To fix the knot, the long suture material is again looped around the closed beaks of the hemostat, but this time in the opposite direction, and pulled up tight to form the surgeon's knot—a square knot, not a “granny knot” (see Figure 8-28D). The knots should be tied to the side of the incision, not as seen in Figures 8-28C and 8-28D. Knots tend to collect debris and bacteria, that can enter the incision. The suture is then cut with very short ends. Interrupted suturing is then continued at each papilla, around the remainder of the flap (see Figure 8-28E).

Postsurgical Care

The patient must know that the dentist cares about his or her condition. A printed list of postoperative instructions is given to the patients and discussed by the dentist or an assistant (Figure 8-30).

The caring surgeon calls the patient that evening inquiring about his or her well-being. If minor bleeding persists, the patient should be instructed to place a moistened tea bag over the wound and apply slight pressure. This should stop the bleeding; if not, the patient should call the office. Injecting lidocaine 1/50,000 epinephrine into the area and applying pressure should stop the bleeding.

Before surgery, one should have made sure the patient was not taking blood thinners such as warfarin (Coumadin). Also, the patient should be warned not to take aspirin for pain relief or cardiac protection until healing is complete. Occasionally, a hematoma develops and the patient ends up with a “fat lip” or a “black eye” (Figure 8-31). The patient must be assured that, as unsightly as it appears, no permanent damage has been done—that a tiny vessel ruptured and, like a bruise, it will go away in a few days. All this should have been explained when informed consent was discussed.

Instructions for Postoperative Care following Endodontic Surgery

1. Do not do any difficult activity for the rest of the day. Easy activity is okay, but be careful and do not bump your face where the surgery was done. You should not drink any alcohol or use any tobacco (smoke or chew) for the next 3 days.
2. It is important that you have a good diet and drink lots of liquids for the first few days after surgery. Juices, soups, and other soft foods such as yogurt and puddings are suggested. Liquid meals such as “Ensure, Boost”, and Carnation Instant Breakfast can be used. You can buy these at most grocery stores.
3. Do not lift up your lip or pull back your cheek to look at where the surgery was done. This may pull the stitches loose and cause bleeding.
4. A little bleeding where the surgery was done is normal. This should only last for a few hours. You may also have a little swelling and bruising of your face. This should only last for a few days.
5. You may place an ice bag (cold) on your face where the surgery was done. You should leave it on for 20 minutes and then take it off for 20 minutes. You can do this for 4 to 6 hours. After 8 hours, the ice bag (cold) should not be used. The day after surgery, you can put a soft, wet, hot towel on your face where the surgery was done. Do this as often as you can for the next 2 to 3 days.
6. Discomfort after the surgery should not be bad, but the area will be sore. You should use the pain medicine you were given or that was recommended to you, as needed.
7. Rinse your mouth with 15 mL of the chlorhexidine mouthwash (Peridex) you were given or prescribed. This should be done two times a day (once in the morning and once at night before going to bed). You should do this for 5 days.
8. The stitches that were placed need to be taken out in a few days. You will be told when to return. **It is important that you come in to have this done!**
9. You will be coming back to the office several times during the next few months so we can evaluate how you are healing. These are very important visits, and you should come in, even if everything feels and looks okay.
10. If you have any problems or if you have any questions, you should call the office. The office phone number is _____.
If you call after regular office hours or on the weekend, you will be given instructions about how to page the doctor on call.

Figure 8-30. Postsurgical instructions for patients. This page may be enlarged, copied and given to patients.

Postoperative infection is rare. The patient should be instructed to rinse with chlorhexidine (Peridex) and to use a cotton swab with chlorhexidine or an antiseptic mouth rinse such as Listerine to cleanse the surgical area, and not to use a tooth brush in the area.



Figure 8-31. **A**, Hematoma in the upper lip following periradicular surgery. It resolved within the week. **B**, Unusual hematoma that developed below the eye following periradicular surgery. Note the ecchymosis in the lip as well. The patient was on high doses of aspirin.

Discomfort may be a patient complaint but it can usually be controlled with ibuprofen or acetaminophen. Severe pain is rare.

The **sutures should be removed in 3 or 4 days**. If left for a week, irritation develops around the sutures. Before the sutures are pulled through, they should be cleansed with a chlorhexidine swab. It is best to apply a topical anesthetic to the area. The sutures are clipped with sharp, pointed scissors, grasped by the knot, and pulled through. The area should once again be swabbed with

chlorhexidine. The patient may now be dismissed with the caveat that he or she should contact the office if anything unusual occurs in the surgical area. It is assumed that the orthograde endodontic filling has already been completed and that the final coronal filling is in place, or that the patient is being referred back to the referring dentist for the final restoration.

It is remarkable how rapidly these lesions heal after surgical intervention. Reentry into a surgical site one week later has revealed that connective tissue organization had already taken place (Figure 8-32).

Corrective Surgery

Corrective surgery may be necessary as a result of procedural accidents, repair of internal or external resorptive defects, root fracture, and periodontal disease. It may involve periradicular surgery, root resection (removal of an entire root from a multirooted tooth leaving the clinical crown intact), hemisection (the sectioning of a multirooted tooth and the removal of a root and a portion of the clinical crown), or intentional replantation (extraction and replantation of the tooth into its alveolus after the corrective procedure has been done).

Mechanical and Resorptive Perforation Repair

Procedural accidents can occur during root canal or post space preparation. Examples are “stripping” the distal aspect of mesial roots of lower molars, and perforation of the side wall of a premolar with a Peeso drill during post preparation. Midroot perforations such as these should immediately be attended to by applying intracanal calcium hydroxide and a substantial temporary filling. If the perforation is extensive, a vertical flap should be laid, the area of perforation exposed, and MTA placed in the defect (Figure 8-33). Final restoration should await healing. If the perforation is in the apical third, a root-end resection and retrofilling should be considered.

Root resorptive defects may also be treated surgically. If the defect lies just below the gingival crest and the pulp is not involved, a small gingivoplasty exposes the margins and following pulpectomy and root canal filling, a restoration may be placed in the defect. If the pulp is exposed, as in internal cervical resorption, root canal treatment is done first and then the defect is repaired (Figure 8-34).

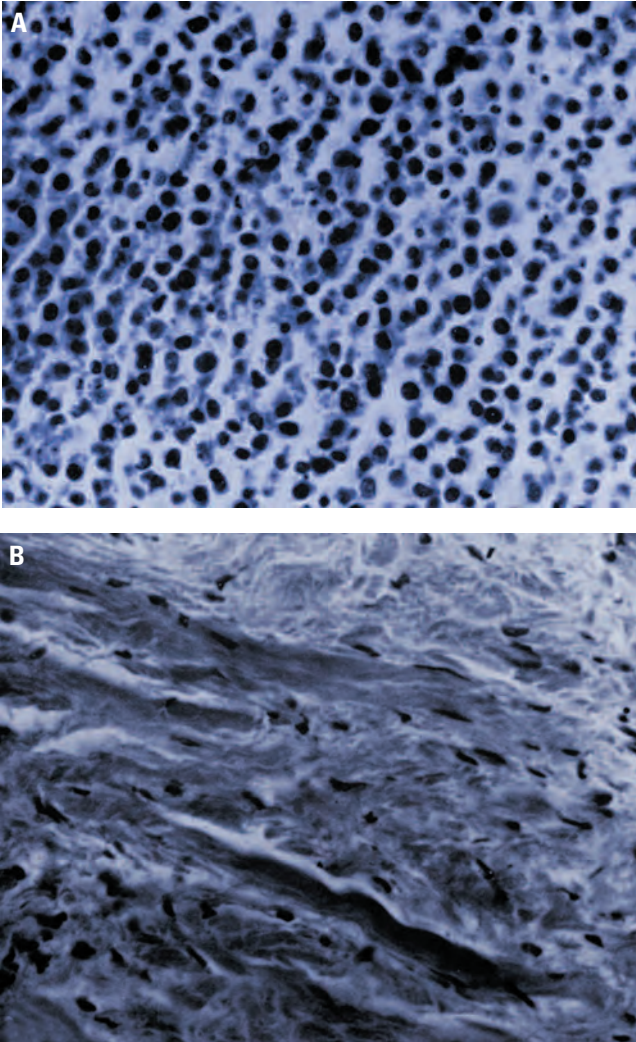


Figure 8-32. **A**, Biopsy of chronic inflammatory tissue removed from a chronic apical periodontitis lesion. **B**, Reentry biopsy taken 1 week later. Note the well-organized connective tissue fibers and cells that have developed in a remarkably short time. Healing is well on its way.

If the defect is midroot, it can be handled much like a perforation repair, as described previously. If the pulp is involved, the pulp may be removed and the canal filled; then the repair can be made. At one time, amalgam was used to repair these defects. An internal matrix

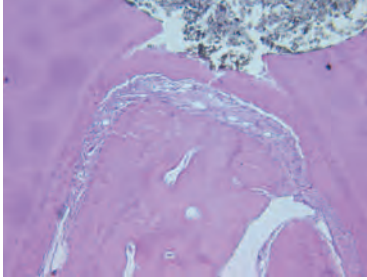


Figure 8-33. Furcation perforation in a dog. Mineral trioxide aggregate (Pro Root MTA, Dentsply/Tulsa Dental) has been placed in the perforation (*top*). Perfectly normal cementum has formed to block the perforation. Normal periodontal ligament and bone have developed below. Courtesy of M. Torabinejad.



Figure 8-34. **A**, Cervical resorption often mistaken for caries. In this case, the resorption had broken through into the pulp and symptoms had developed. **B**, Gingivoplasty gains entry to the lesion, the pulp is removed, and the canal cleaned, shaped, and obturated at the same appointment. A periodontal pack is placed as a temporary filling. **C**, After the gingiva has healed, a final amalgam filling is placed.

was needed to compact the root-defect filling, and the root canal filling was placed last (Figure 8-35).

In the event the resorption is coronal and much of the crown has been destroyed, one must resort to hemisection—half the crown is severed buccolingually, the attached root is extracted, and a root canal filling is placed in the remaining root, which then becomes an abutment for a fixed bridge (Figure 8-36).

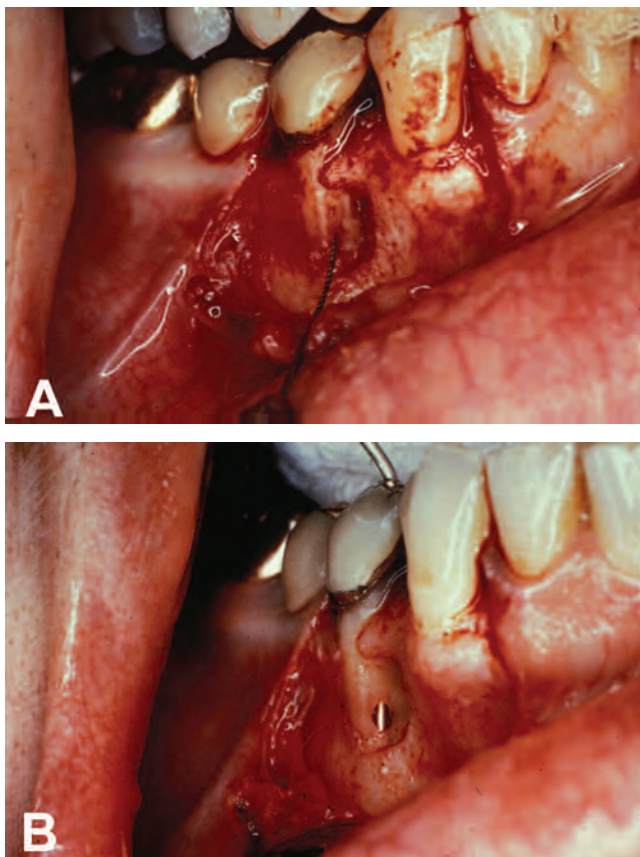


Figure 8-35. **A**, A buccal stoma indicated that midroot external resorption had broken through into the pulp and the area had become infected. A flap is raised to expose the lesion and the extensive bone loss. An endodontic file confirms the continuum of the resorption. **B**, Through a regular occlusal access cavity, the pulp is removed and the canal cleaned and shaped. A silver point is then placed in the canal to serve as an “internal matrix.” **Continued on next page.**

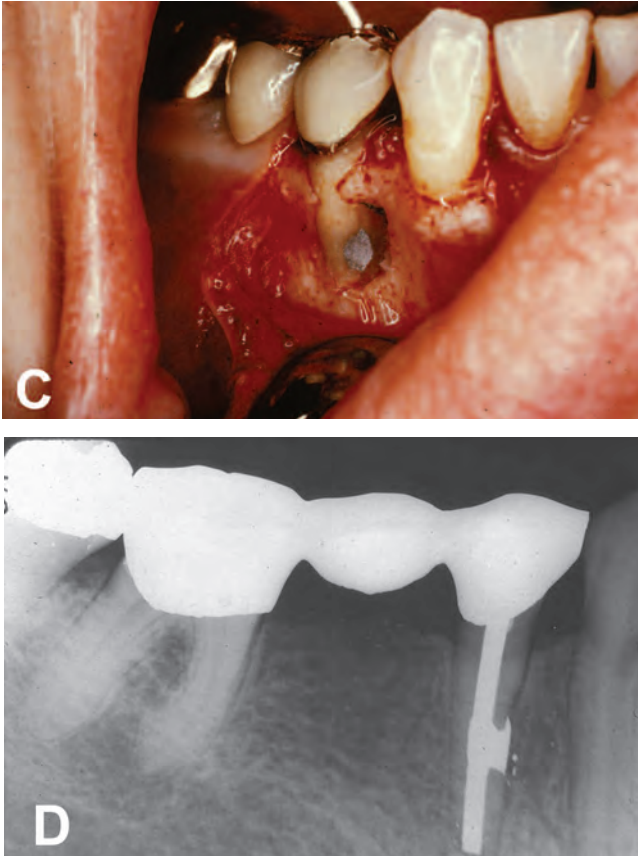


Figure 8-35. Continued. **C**, An amalgam filling is then placed in the resorptive defect. The silver point is removed, and the flap is sutured to place. Later a root canal filling and final occlusal restoration are placed. **D**, Radiograph taken 9 months later showing complete repair of the lateral bony lesion.

Whole Root Amputation

The indications for root amputation are as follows:

- Periodontal bone loss to such an extent periodontal therapy is inadvisable
- Destruction of the root through resorption, caries, or perforation
- Surgically inoperable roots that are calcified or contain separated instruments
- Fracture of one root without the involvement of another.

Contraindications are the following:

- Lack of osseous support for remaining roots
- Fused roots
- Remaining roots that are endodontically inoperable
- Lack of patient motivation to perform home care.

Most cases requiring root amputation involve huge periodontal lesions that completely denude one root of a multirooted tooth,



Figure 8-36. Technique for hemisecting and restoring mandibular molar. **A**, huge area of internal resorption had broken through into the pulp and the area had become infected. **B**, using an extra-long No. 559XL fissure bur, the tooth is sectioned, buccal to lingual, down to the furcation, with copious water and aspiration. **Continued on next page.**

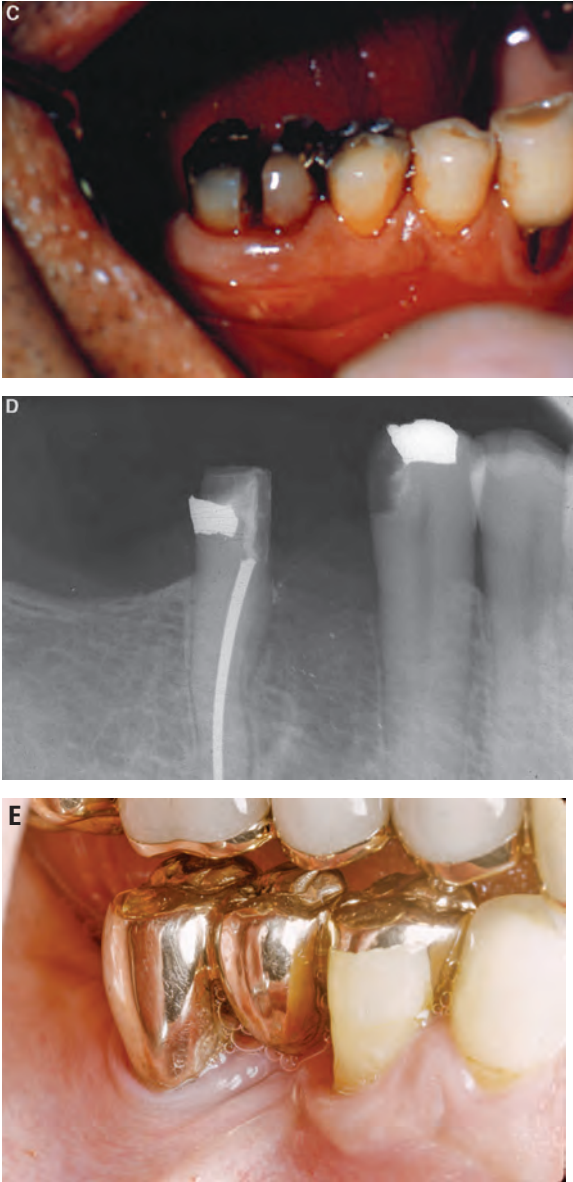


Figure 8-36. Continued. C, sectioning completed. Base of cut must terminate at the alveolar crest. **D**, after the mesial root is extracted, and at the same appointment, a rubber dam is placed and root canal treatment is completed on the distal root. **E**, importance of saving the distal half of the tooth as an abutment to maintain molar contact with the opposite arch. Restorative by M.S. Starks.

with the remaining roots well supported. In such a case, the root canal treatment is carried out on the roots that are to remain; the unsupported root, which is to be amputated at the crown, should have an enlarged, permanent, internal filling such as an amalgam filling placed in the canal from within the crown. The sectioning through the root and filling should be done after the filling material has set (Figure 8-37). In some cases, a simple tissue flap exposes

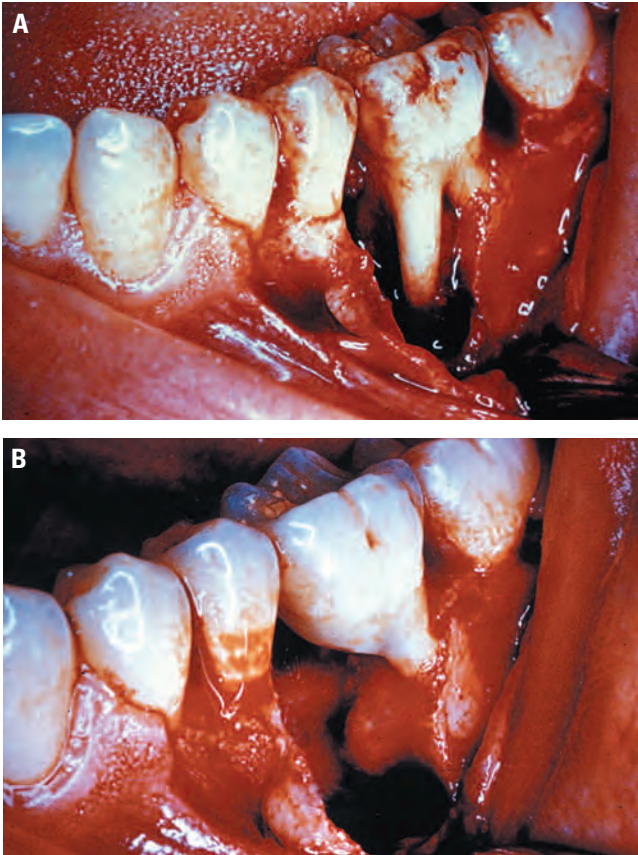


Figure 8-37. **A**, Huge, isolated periodontal lesion. The mesial root of the lower molar has lost total bony support. **B**, First, the root canal treatment is carried out on the distal root and cavity preparations are made with a bur in the canals of the two mesial roots. Amalgam fillings are then placed internally in the two mesial root canals. The mesial root is amputated across the two amalgam fillings and to the furcation. The flap is then sutured to place and the occlusal table is narrowed to relieve stress. *Continued on next page.*



Figure 8-37. Continued. C, Photograph taken at the 1-year follow-up.

the root at the furcation and amputation with a fissure bur is quite simple. Sometimes the furcations are exposed and no flap is necessary (Figure 8-38).

Occasionally, lower molar cases present that have periodontal destruction in the furcation area. These teeth can be hemisected buccolingually into two halves and treated as two premolars (Figure 8-39). Often rubber eraser wedges can be used to slowly separate the two halves. Meticulous home care using a Proxabrush (John O. Butler, Sunstar) maintains the new interseptal areas (Figure 8-40).

These cases of root amputation and hemisection have a remarkable success rate. To a great extent, success depends on the quality of both the root canal treatment and the final restoration. One can hardly argue with cases that last for nearly half a century (Figure 8-41).

Correction of the Radicular Lingual Groove

Another serious periodontal defect that may be corrected by a surgical approach is the radicular lingual groove, seen most frequently on maxillary central and lateral incisors. This developmental defect precludes the formation of cementum in the groove; hence, the peri-

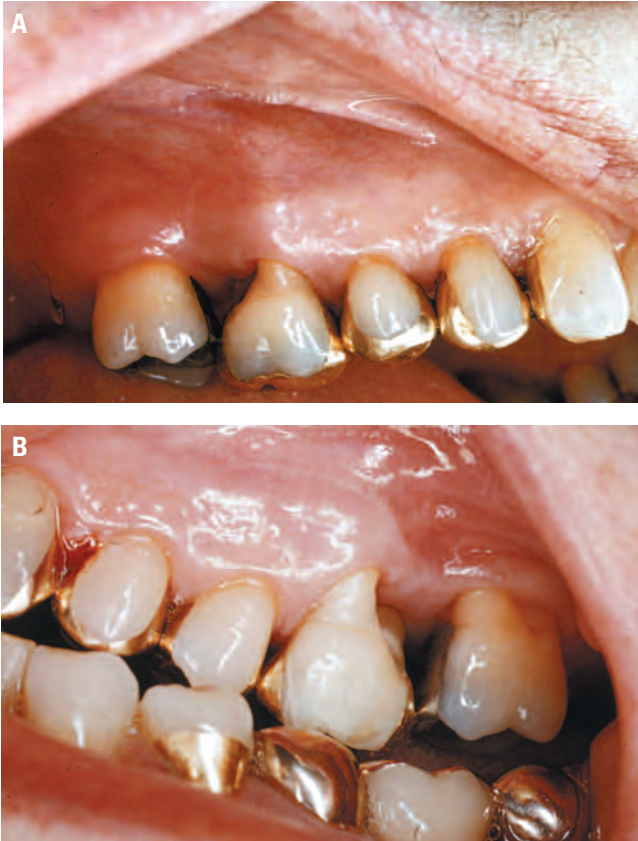


Figure 8-38. **A & B,** Bilateral amputation of the distobuccal roots of two maxillary first molars in the same patient. Root amputation was preceded by a root canal treatment of the two remaining roots in each tooth and an amalgam filling, from the crown, of the amputated roots. No flap was necessary.

odontal ligament cannot attach. The groove causes a narrow periodontal pocket and a bacterial pathway that often extends to the root apex allowing retroinfection of the pulp.

Following a **palatal surgical exposure** of the defect, the groove is eliminated by grinding it away with round burs or diamond points (Figure 8-42). If the lingual groove is so deep that it communicates with the pulp, the case is hopeless and the tooth must be extracted.

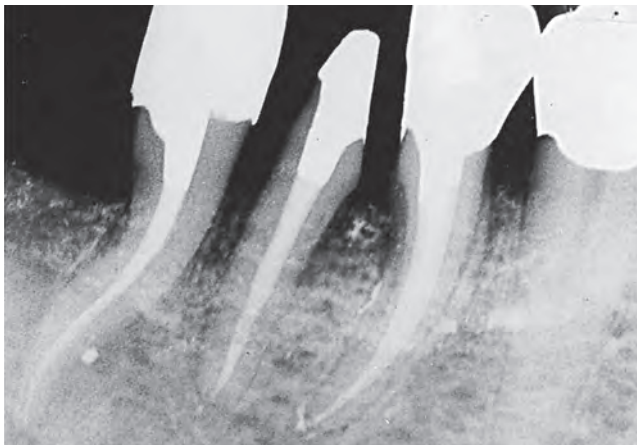


Figure 8-39. Hemisection of a second molar and bisection of a first molar provides three sturdy abutments for a terminal bridge. Courtesy of L.S. Buchanan.

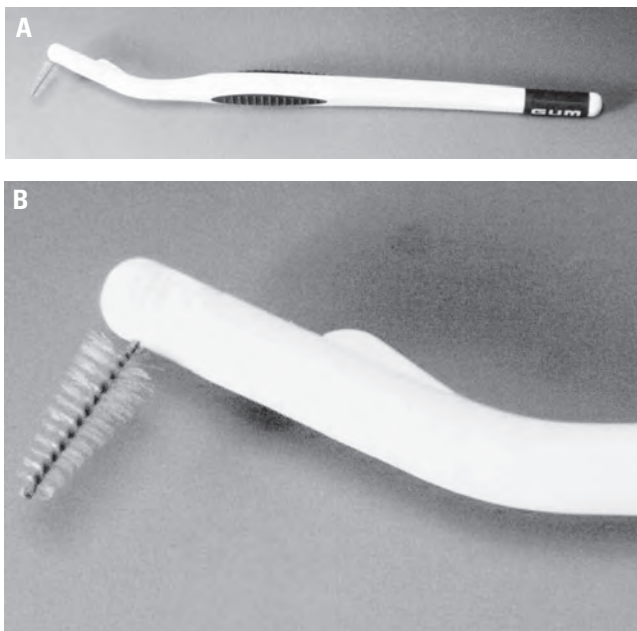


Figure 8-40. **A**, Cleaning aids such as the Proxabrush (John O. Butler Co.) are important for good maintenance of these compromised areas. **B**, Close-up of the Proxabrush head.

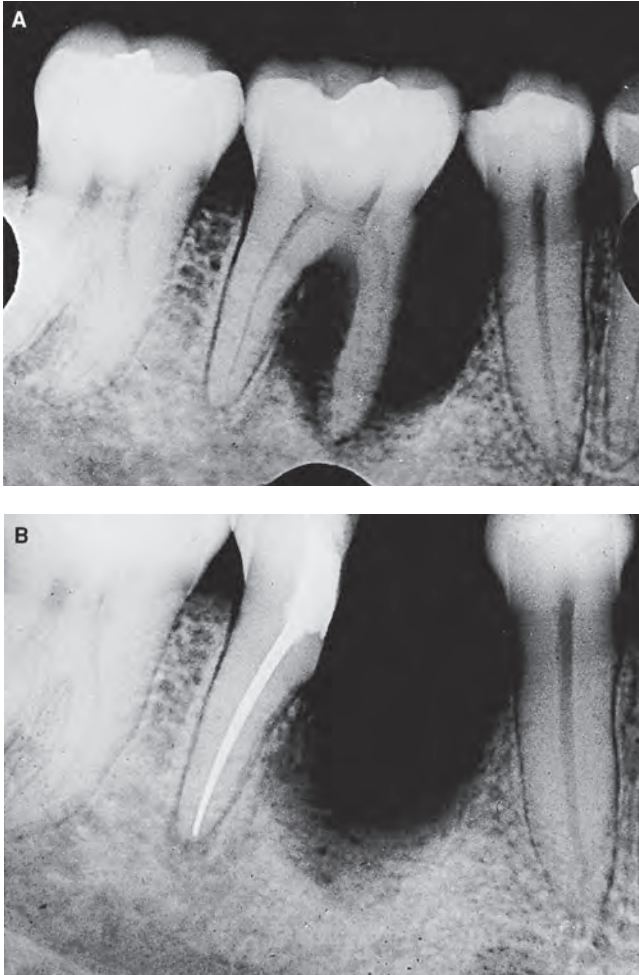


Figure 8-41. Hemisection of a mandibular molar. **A**, The decision to hemisect a molar and restore the space relates to the open contacts and future drifting that would encourage collapse of the remaining root. **B**, The tooth is hemisected through the furcation, and the pathologic root is removed. Root canal filling of the remaining root is done at the same appointment. Courtesy of D. Glick and J. McPherson. **Continued on next page.**

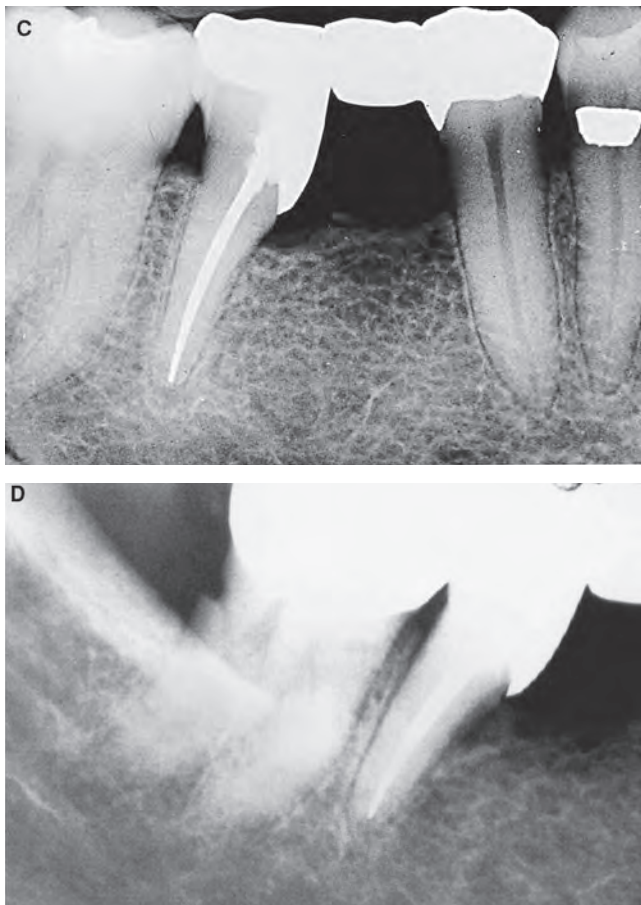


Figure 8-41. Continued. C, Final restoration of the space converts the first molar to a premolar. **D,** Radiograph taken at a follow-up **47 years later** attests to the meticulous therapy and long-range efficacy of this procedure. Courtesy of D. Glick and J. McPherson.

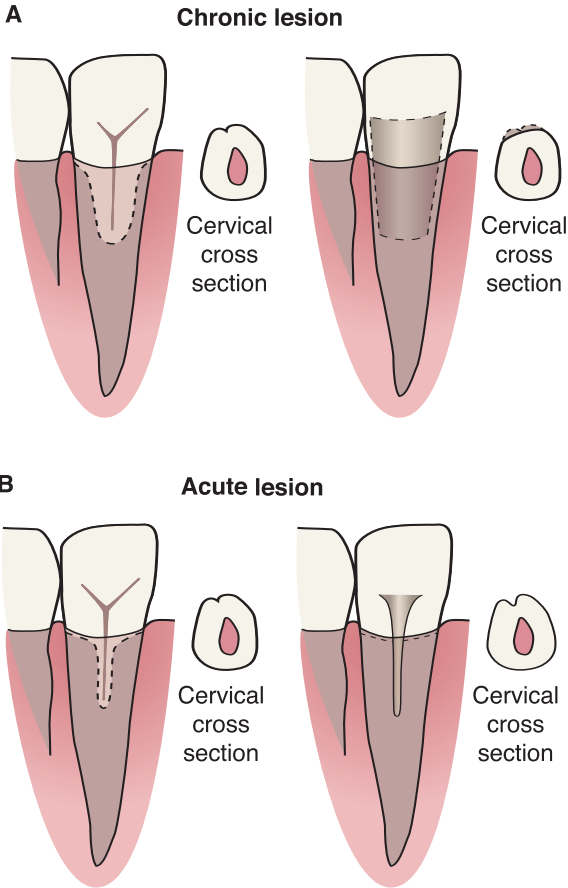


Figure 8-42. A, Surgical exposure of a shallow yet chronic palatogingival groove allows “saucerization” with diamond stones to remove the groove. **B,** An acute lesion may result in greater bone loss. In this case the groove may be eliminated with a bur to the depth of the groove. A perfect surface of exposed dentin sometimes allows a partial reattachment of the periodontal ligament.

OSSEOINTEGRATED IMPLANTS

Although implant surgery is within the scope of endodontics, it is a very technique-sensitive procedure requiring a relatively long learning curve. One is not encouraged to undertake implants unless one is well-versed and trained in the field.

FINAL NOTES

All in all, surgery is a very important aspect of endodontics. However, it should not be thought of as a cure-all or as a necessary procedure to remove all periradicular lesions. Indications for surgery are listed in Table 8-1. In most cases, surgery should be considered a final approach rather than a first approach.

REFERENCES

1. Carr GB, Bentkover SK. Surgical endodontics. In: Cohen S, Burns R, editors. Pathways of the pulp. 7th ed. CV Mosby: St. Louis, MO; 1997, 619.
2. Gartner AH, Dorn SO. Advances in endodontic surgery. Dent Clin North Am 1992;36:357.

Endodontic Mishaps

DETECTION OF MISHAPS

Endodontic mishaps or procedural accidents are those unfortunate accidents that happen during treatment, some owing to inattention to detail, others being totally unpredictable. Table 9-1 outlines a list of the most common mishaps.

Table 9-1

ENDODONTIC MISHAPS

- Access related
 - Treating the wrong tooth
 - Missed canals
 - Damage to existing restoration
 - Access cavity perforations
 - Crown fractures
- Instrument related
 - Ledge formation
 - Cervical canal perforations
 - Midroot perforations
 - Apical perforations
 - Separated instruments and foreign objects
 - Canal blockage
- Obturation related
 - Over- or underextended root canal fillings
 - Nerve paresthesia
 - Vertical root fractures
- Miscellaneous
 - Post space perforations
 - Irrigant related mishaps
 - Tissue emphysema
 - Instrument aspiration and ingestion

How can mishaps be prevented? Learn from experience! Unfortunately, someone—dentist or patient—must pay for this learning curve, but we do learn from our mistakes. However, there is **one cardinal rule**: If a mistake will affect the outcome of the case, the patient must be informed at once, and the mistake must be noted in the record along with a statement that the patient was informed of the mistake. Too many court cases are lost because of this neglect. Also, patients should be informed before treatment (informed consent) of any possible consequences such as the possibility of a porcelain crown fracture. In addition, if correction of the mishap is beyond the dentist's skill or knowledge, the case should be referred to someone better equipped to handle its correction, and the dentist should be liable for the correction fee. Don't argue, don't be stubborn, and don't try to cover it up. Remember Watergate! Admit your mistake and make every effort to have it corrected.

ACCESS-RELATED MISHAPS

Treatment of the Wrong Tooth

Treatment of the wrong tooth can be so easily prevented. One should make sure through inquiry, testing, examining, and radiography that one has confirmed which tooth requires treatment and then mark it with a felt-tip pen (Figure 9-1).

Missed Canals

Additional canals in the mesial roots of maxillary molars and the distal roots of mandibular molars are the most frequently missed. Second canals in lower incisors, and second canals and bifurcated canals in lower premolars as well as third canals in upper premolars, are also missed. One must be diligent and prepare adequate occlusal access! Always expect there will be an extra canal. Magnification with either telescopic lenses or a surgical microscope is indispensable (Figure 9-2).

Damage to an Existing Restoration

Porcelain crowns are the most susceptible to chipping and fracture. When one is present, use a water-cooled, smooth diamond point and do not force the stone. Let it cut its own way (Figure 9-3). Also,



Figure 9-1. Tooth no. 25 has been marked with a felt-tip pen in preparation for rubber dam placement. Marking the tooth prevents the incorrect placement of the dam.

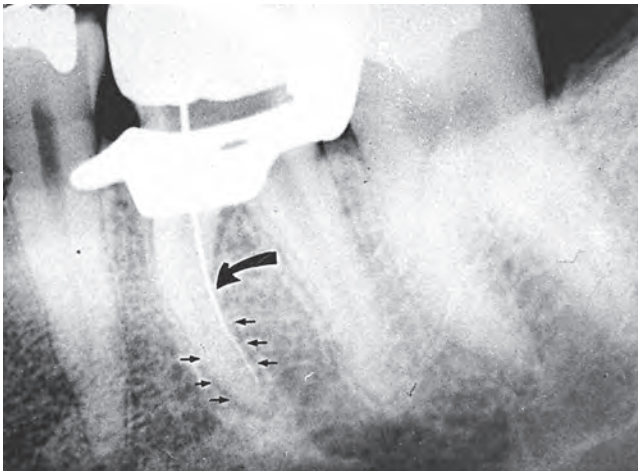


Figure 9-2. Radiograph used in search for a second, missed canal. By following the lamina dura of the root (*small arrows*), the eccentric position of the file (*large arrow*) suggests the possible presence of a missed canal. However, the file has actually passed through a perforation and is traversing the periodontal ligament. Look for bleeding in the pulp chamber.

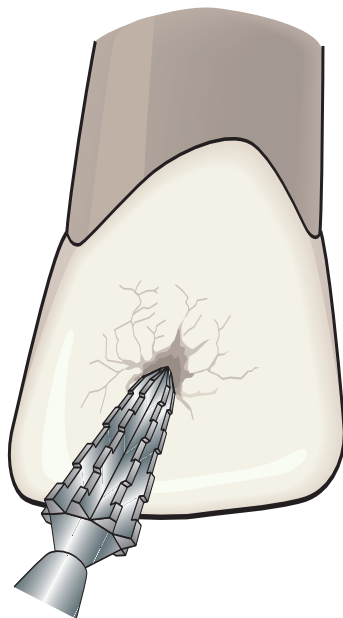


Figure 9-3. Forcing an accelerated tapered bur or diamond severely crazes the lingual enamel. If the crown had been a porcelain jacket, it would have fractured. The instrument should be allowed to cut its own way.

do not place a rubber dam clamp on the gingival of any porcelain or porcelain-faced crown.

Access Cavity Perforations

Often the first sign of an access cavity perforation is blood in the cavity or the patient complaining of a taste of NaOCl. This most frequently happens in the floor of molar preparations when one is searching for a third or fourth canal. The site of the perforation must be found, the floor of the preparation cleansed, the bleeding stopped, and mineral trioxide aggregate (MTA) applied to the perforation (Figure 9-4). Because it takes MTA more than 3 hours to set, it should be covered with a fast-setting cement. The other canal orifices should be protected by placing paper points or an instrument in the canals to prevent blockage. In the event MTA cannot be immediately applied, it is best to stop the bleeding, place calcium hydroxide over the “wound,” place a good temporary fill-

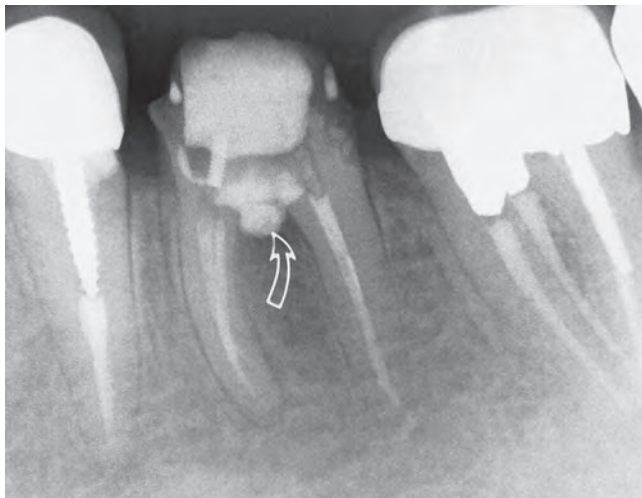


Figure 9-4. Peroration repair using mineral trioxide aggregate (MTA).

ing, and set an appointment with the patient, the sooner the better. The perforation area will be dry at the next appointment; then MTA can be applied and treatment continued.

Crown Fractures

Remember, preparing an endodontic access cavity in a tooth, particularly a molar or premolar with a large restoration, materially weakens the crown. Infrequently the crown fractures, either during preparation or at a subsequent appointment. One of the frequent causes is failure to relieve the occlusion. If the fracture is chisel-shaped and a cusp breaks off down to the periodontal ligament, the tooth can usually be salvaged. However, if the fracture extends through the pulp chamber and down into the root, the case is hopeless and the tooth should be extracted. (See Chapter 10, “Management of Endodontic Emergencies”)

INSTRUMENTATION-RELATED MISHAPS

Overzealous cleaning and shaping have led to many mishaps, zip-ping and perforations among them (Figure 9-5). Many of these iatral accidents begin with ledging during canal preparation.

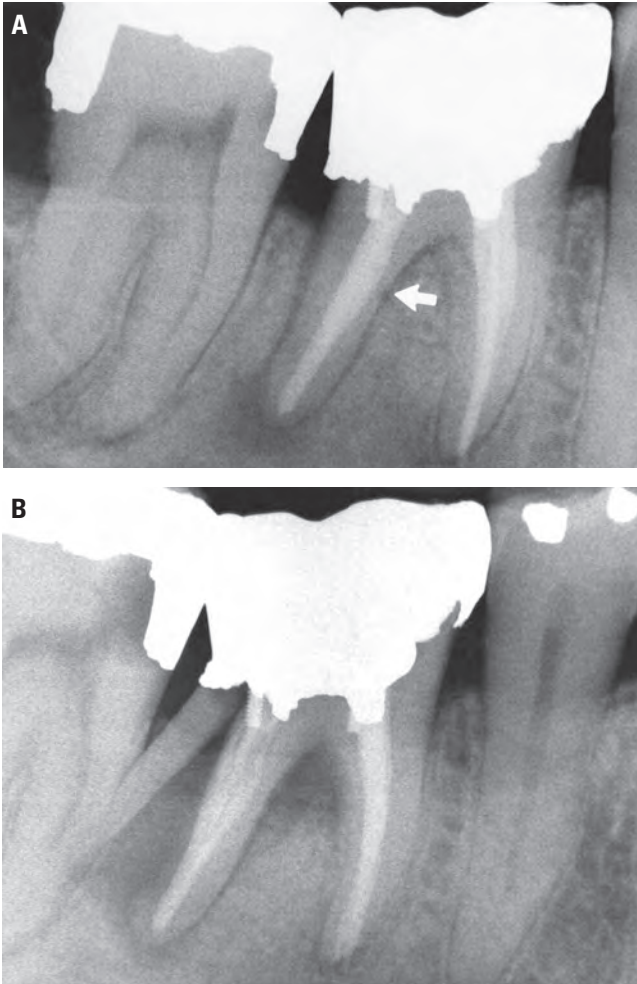


Figure 9-5. Vertical root fracture associated with overzealous canal preparation. **A**, Postoperative radiograph shows distal canals prepared and filled leaving a thin-walled root (*arrow*). **B**, Twelve months later, the patient reported with a vertical root fracture

Ledge Formation

Many ledges are caused from inadequate access. The operator does not retain complete control over the direction of the tip of the instrument and it gouges into the wall of the canal, starting a ledge (Figure 9-6A). Newer instruments with non-cutting tips have mate-

rially reduced this problem. The rounded tip does not cut into the wall but slips by it.

Curved roots are another impediment in canal preparation. This is especially true near the apex of maxillary lateral incisors and the palatal root of maxillary first molars. Small, flexible instruments with non-cutting tips negotiate these curves but larger, stiffer instruments start a ledge that can develop into a perforation.

If the instrument can no longer be inserted into the canal to full working length, one should suspect that a ledge has been formed. There also is a change in tactile sensation, a feeling that the instrument is no longer engaging the walls. Stop! Take a radiograph with the instrument in place.

To correct the ledge, return to a much smaller stainless steel file and curve the tip so that the file clings to the inner wall, away from the curve. Slip the file by the curve to the full working length, then file back against the ledge to remove the nick in the wall. Repeat the filing with the next size curved file and so on, until the ledge is removed and larger instruments slip by the ledge area (Figure 9-6B).

Be sure to use plenty of lubrication and irrigation to remove the dentin chips that tend to pack at the apex. Do not use EDTA for chelation because it tends to intensify the ledge. Precurved stainless steel instruments and nickel-titanium instruments obviate the start of these problems.

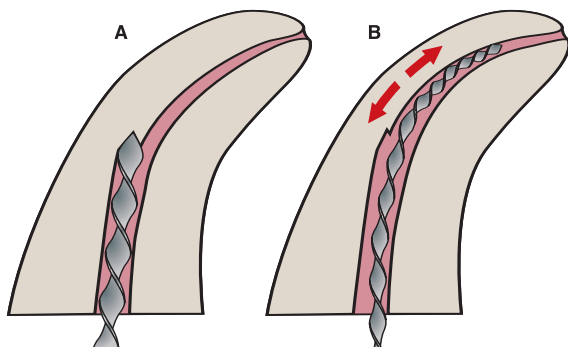


Figure 9-6. Cause and correction of ledge formation in a curved canal. **A**, A large, straight instrument used in a curved canal cuts a ledge at the curve. **B**, The ledge may be painstakingly removed with a severely curved stainless steel file rasping against the ledge (arrows) in the presence of NaOCl or a lubricant such as Prolube (Dentsply/Tulsa Dental). To bypass the ledge, the file must be severely curved and hug the inside wall at the curve.

Perforations

Canal perforations may be categorized by their location. Access cavity perforations, such as perforations through the floor into the furcation, have already been discussed. Root perforations can be identified as cervical, midroot, or apical. These are usually caused by three errors: creating a ledge and persisting until a perforation develops, wearing a hole in the lateral surface of the midroot by overinstrumentation (canal stripping), or using too long an instrument and perforating the apex.

Cervical perforations usually occur when too large an instrument is used to widen canal access, frequently a Gates-Glidden or Peeso drill. The first indication is the appearance of blood in the cavity. Again, the bleeding is stopped and MTA is applied to the perforation. Cotton should be placed in the chamber and a good temporary placed to allow time for the MTA to set (at least 3 hours). Preparation is continued at a subsequent appointment.

Midroot perforations are usually caused by zipping, frequently in the distal wall of a curved mesial root of a mandibular molar (Figure 9-7). Again, blood in the canal indicates that a perforation has occurred. By using paper points in the canal, the location of the perforation can be determined (Figure 9-8A). MTA is the material of choice to close the perforation (Figure 9-8B and 9-8C); an appointment is made with the patient for continued treatment.

Apical perforations are usually due to overeager instrumentation, just plain drilling out through the apical orifice. Again, this can usually be determined with paper points—they appear bloody at the tip (Figure 9-9). If a canal curves at or near the apex, using larger and larger instruments will cause zipping that hollows out this area and leads to perforation. Remember, a curved canal is gradually straightened through cleaning and shaping. A straight line is the shortest distance between two points; therefore, the cleaning and shaping actually shorten the working length! One must compensate for this change by shortening the working length on the shaft of the instrument. Maybe the difference will only be 0.5 mm. Confirm the new length by radiograph with an instrument in place.

Apical perforation destroys the resistance form. This means that the tip of the primary gutta-percha or Resilon point must be blunted and fit to place so that it does not extend beyond the orifice, even when compaction is exerted during obturation. A good method

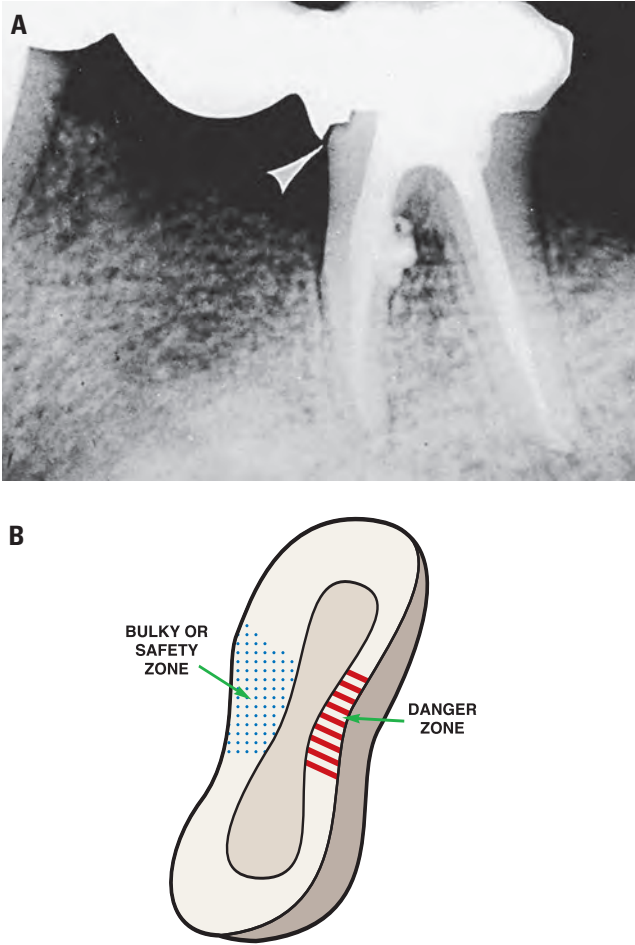


Figure 9-7. **A**, Lateral perforation by wearing through the thin distal wall of the mesial root. Note the open margin (*arrowhead*). **B**, Schematic cross-section of a curved root depicting the thickness of walls, safety zone, and danger zone.

is to deposit a tiny pack of MTA at the apex (see Figure 9-8B), checking its placement radiographically. The patient returns at a later appointment, after the MTA has set, and the apical foramen is plugged. At the subsequent appointment, this new plug (resistance form) allows compaction of the gutta-percha. Later, MTA will encourage cementum formation at the apex (see Figure 7-14).

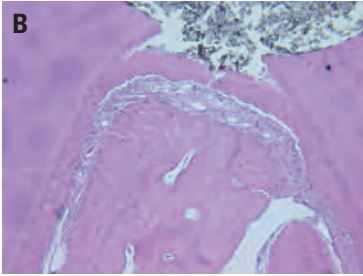
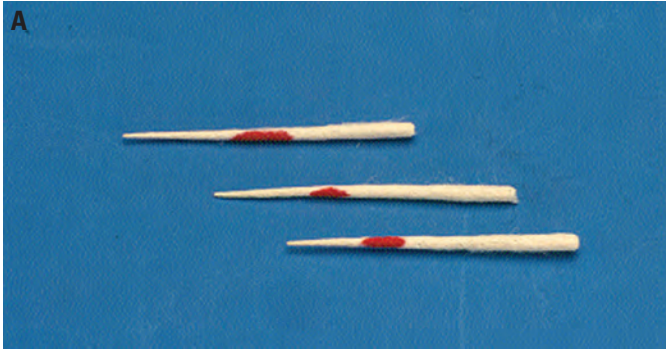


Figure 9-8. **A**, Paper points confirming strip perforation. The area of hemorrhage marked on the paper point indicates the area where the strip occurred. **B**, MTA repair of a furcation perforation in a dog. The MTA is shown at the top of the image, above the perforation. Complete cementum repair below the perforation lies above normal bone and the periodontal ligament of the two roots. Courtesy of M. Torabinejad. **C**, Curved needle and straight needle used with the MAP system syringe (Roydent Dental Products) for the placement of MTA in the canals to repair midroot and apical perforations.



Figure 9-9. Location of hemorrhage at the tip of a paper point suggests apical perforation.

Separated Instruments and Foreign Objects

Many objects have been reported to break or separate in the root canal—amalgam, files and reamers, lentulo spirals, Gates-Glidden drills, and burs. In addition, patient-placed foreign objects, such as nails, pencil lead, toothpicks, tomato seeds, and hat pins, have been reported in root canals. **Files and reamers** are the most frequent offenders, and since the advent of nickel-titanium rotary instruments, breakage (euphemistically called “separation”) has increased. Because these new instruments are so flexible, unrealistic strains are placed upon them, especially in severely curved canals and at greater rotary speeds.

The **first caveat is prevention**. Do not force any endodontic instrument, especially in a twisting or rotary motion. Advancement down the canal by a “pecking” or “watch-winding” motion, rather than a forceful advance, is *de rigueur*. Use of these new rotary instruments requires a learning curve, so one should practice their proper use on extracted teeth before starting in the mouth. Also, instruments should be inspected after their use to determine whether they have been stressed (Figure 9-10). A safe rule of thumb is to use rotary instruments only once. At first this seems costly, but all one has to do is break an instrument once and the cost in lost time and frustration would pay for dozens of instruments.

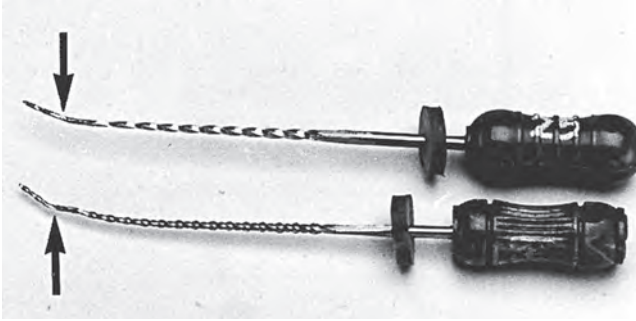


Figure 9-10. Instruments showing stress (*arrows*) from faulty instrumentation techniques. Instruments with these signs of potential fracture must be discarded.

If a broken instrument can be grasped, it may be removed with Stieglitz forceps. Failing that, using fine ultrasonic instruments to loosen and “float” out the broken piece has proven very successful. If all retrieval fails, broken instruments have been successfully bypassed and a successful root filling placed around them. If a broken instrument extends out the apex into medullary bone, it should probably be removed surgically and a retrofilling placed (Figure 9-11).



Figure 9-11. Treatment of a broken instrument past the apex. **A**, When the canals were re-treated, the broken file could not be removed. *Continued on next page.*

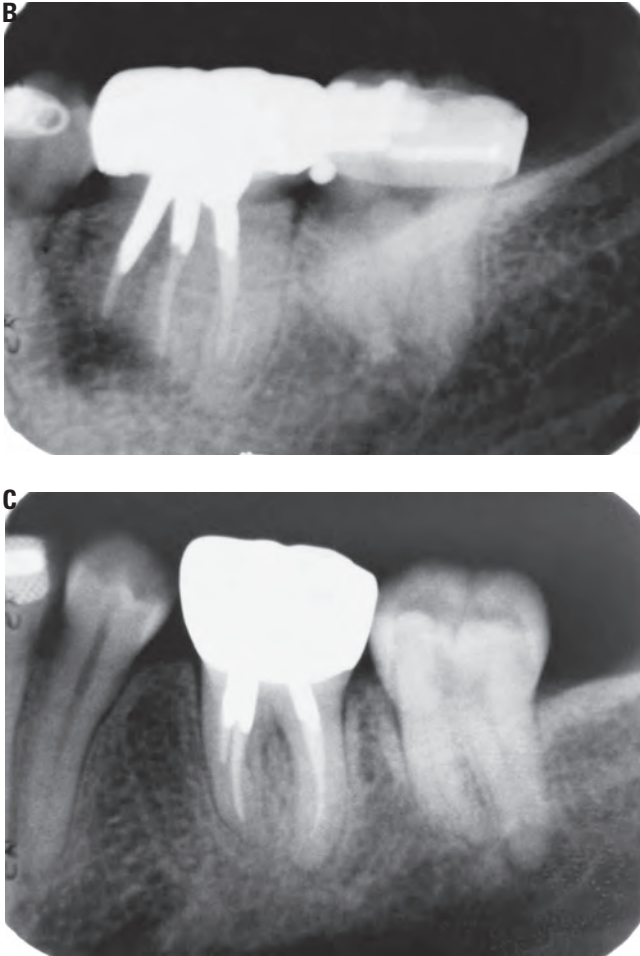


Figure 9-11. Continued. **B**, Apical resection of the mesial root to remove the file fragment. No retrofill was necessary in this instance. **C**, Reevaluation at 12 months shows satisfactory healing. Courtesy of D. Peters.

Canal blockage can occur during canal enlargement. Files are known to compact debris at the apex; even vital tissue can be compacted against the apical restriction. Suddenly, working length is shorter because the instruments are working against the packed mass at the apex. A radiograph will confirm this suspicion. Correction is made by recapitulation—starting with finer instruments used in a quarter-turn motion. Adding a chelating agent such as EDTA is helpful. However, one must be careful not to produce a ledge or

perforation. On the other hand, **sterile** dentin chips at the apex, formed during preparation, help to block the foramen and prevent over-compaction, and dentin chips have been shown to stimulate cementum formation at the apex. Infected chips, on the other hand contribute to further inflammation.

OBTURATION-RELATED MISHAPS

Over- and Underextended Root Canal Fillings

The most preferred location for apical termination of both the canal preparation and the root filling is the dentinocemental junction, often the most restrictive area of the canal. This point is just short of the radiographic apex. Slight extrusion of root canal filling material has been called a “puff” or “button,” and in some obturation methods is thought to be proof that the canal is now impermeable. Healing against these slight overfills is usually inconsequential. Loss of apical constriction by perforation is often the cause of overextension. There is no barrier against which to compact the root filling, but **gross overextension** may well deter healing and may even end up in the maxillary sinus or the mandibular canal (Figure 9-12A). Forceful spinning out the apex of sealers or paste-type fillers from a lentulo spiral may leave massive deposits of material that act as a neurotoxic agent against the sinus lining or the mandibular canal contents. Nerve paresthesia is often the result (Figure 9-12B). For massive overfills, removal by surgery followed by a retrofilling is often the only solution (Figure 9-12C).

Vertical Root Fractures

A sudden crunching sound (often referred to as “crepitus”) during obturation is a clear indication that the root has fractured. This may take place during compaction, more often during lateral than vertical compaction. Sometimes vertical fractures, called “silent fractures,” can happen months or years later. It is suspected that stresses built up during compaction are relieved later, following additional stress from mastication or clenching. This is especially true when overenlargement and thinning of the dentin walls has occurred.

Seating of posts, especially tapered posts, is another cause of vertical fractures. Also, when post preparations have removed so

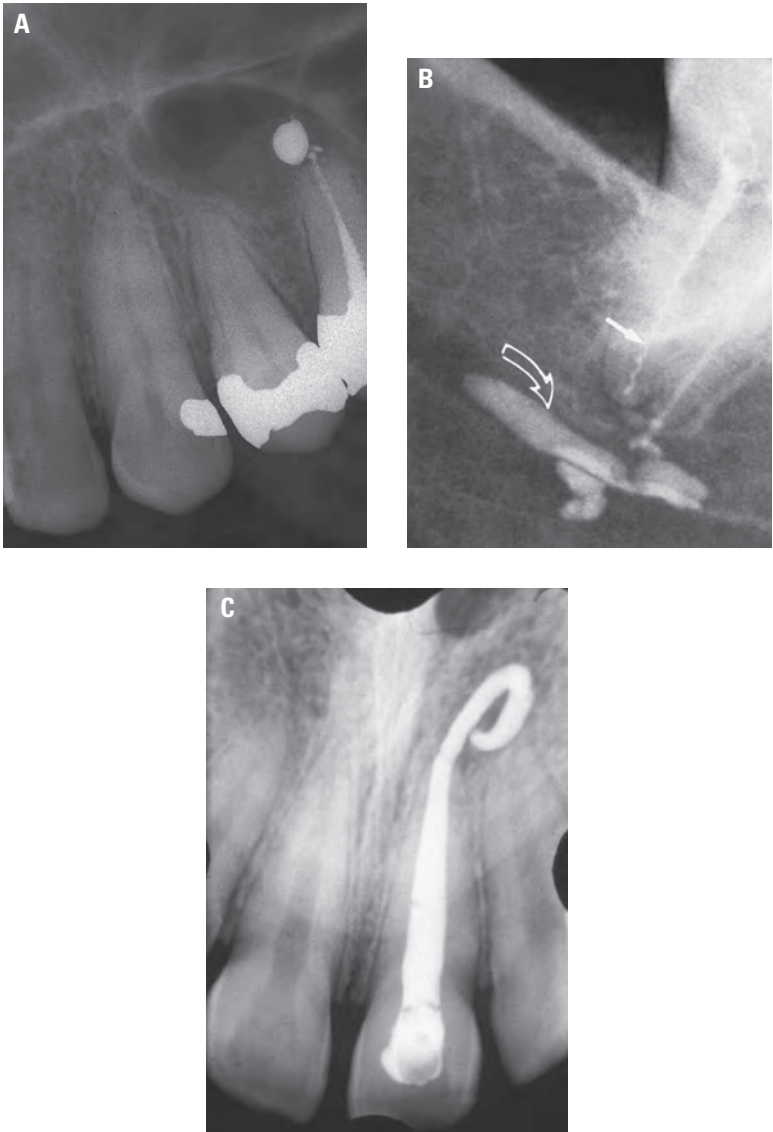


Figure 9-12. **A**, Apical perforation of a premolar and the floor of the maxillary sinus. Lentulo spiral paste filling overfilled into the sinus, leading to sinusitis. **B**, There is a risk in using a paste-type filling material and a lentulo spiral paste filler. Note that part of the spiral is still in the canal (*small arrow*). More serious is the presence of paraformaldehyde paste in the mandibular canal (*open arrow*). Nerve paresthesia is the result. **C**, Gross overfilling of an incisor leads to constant discomfort. The gutta-percha must be removed surgically.

much of the root structure that the tooth is materially weakened, the tooth is easily subject to fracture. There is no treatment if the fracture extends down the root.

Prevention, then, is the only alternative. Not overpreparing the canals to accompany large filling instruments or posts is of the first order. Then, avoiding excess pressure during compaction of gutta-percha fillings or the cementation of posts is necessary.

MISCELLANEOUS MISHAPS

Irrigant-Related Mishaps

The fear of the toxicity of sodium hypochlorite as an irritant of periradicular tissue has tended to deter its use, and for good reason! Forcibly injecting NaOCl or any other irrigating solution into the apical tissue can be disastrous (Figure 9-13). The same can be said for hydrogen peroxide (Figure 9-14). The patient may immediately complain of severe pain. Swelling can be violent and alarming. Of course, the concentration of the irrigant will be a major factor, for example, 5.25% versus 1.3% NaOCl. Antihistamines, ice packs, intramuscular steroids, even hospitalization and surgical intervention



Figure 9-13. Massive reaction to an injection of full-strength sodium hypochlorite out of the apical foramen. Demerol controlled the pain poorly. Swelling, pain, and discoloration disappeared within 10 days. Courtesy of C.L. Sabala and S.E. Powell.

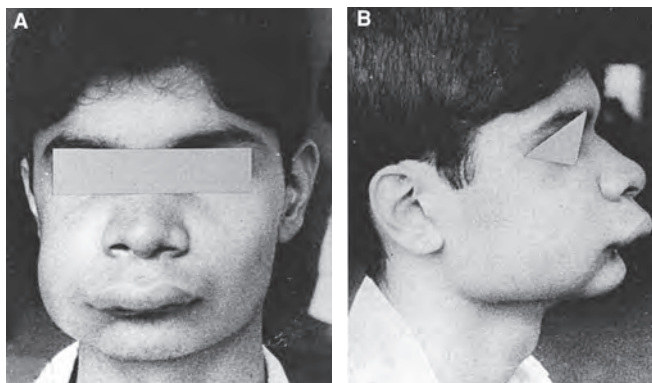


Figure 9-14. A & B, Severe tissue emphysema caused by injecting hydrogen peroxide irrigant into the tissues. Courtesy of K.S. Bhat.

may be needed. Paresthesia, scarring, and muscle weakness may follow. Ending up in court may be the least of one's problems.

Of course, **prevention** is the only solution! Inadvertent extrusion of irrigants past the apex can be avoided by using **passive placement of a modified needle**. The needle must not be wedged in the canal. The blunt-nosed, side-orifice **ProRinse needle** (Dentsply/Tulsa Dental) is the one recommended; it is carried down the canal until it stops, and then is withdrawn 1 mm or so (Figure 9-15). No great force is exerted on the plunger of the syringe. Moreover, if a patient warns the dentist of an allergy to household bleach, substitute chloramphenicol or Bio Pure MTAD (Dentsply/Tulsa Dental).

Tissue Emphysema

Although relatively uncommon, tissue emphysema should not be overlooked. Two actions may cause this to happen: a blast of air to dry a canal, and exhaust air from a high-speed drill directed toward the tissue and not evacuated to the rear of the handpiece during apical surgery. Emphysema from a blast of air down the canal is more likely to happen with youngsters, in whom the canals in anterior teeth are relatively large. The usual sequence of events is rapid swelling, erythema, and crepitus, the latter being pathognomonic of erythema. If the air pocket breaks through into the neck region, there is a sudden swelling of the neck, the voice sounds brassy, and the patient has difficulty breathing. If it breaks through into the mediastinum, a

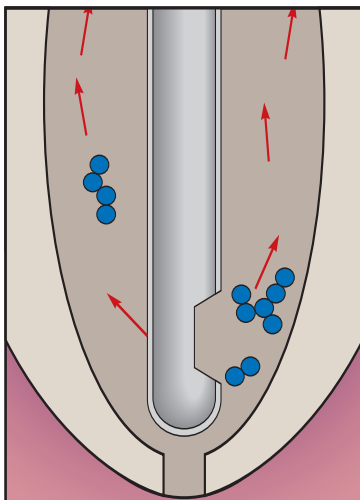


Figure 9-15. ProRinse needles (Dentsply/Tulsa) that irrigate through a side vent eliminate the possibility of puncturing the apical foramen or the “water cannon” effect from open-ended needles.

crunching noise is heard on auscultation. Death can follow! Although the problem should not be treated lightly, the majority of reported cases have followed a benign course to total recovery.

Prevention is simple: Use paper points. **Do not blow air directly down an open canal.** Use the Venturi effect: blow air **across the canal opening** to aid drying, and in surgery employ a handpiece that exhausts the spent air out the back of the handpiece rather than into the operating field.

Instrument Aspiration and Ingestion

Accidents of instrument aspiration and ingestion can be prevented so easily. **Always use a rubber dam!** There is no reason an endodontic instrument should ever be dropped down a patient’s throat. Long ago, endodontic instruments had a hole in the handle so one could tie a piece of dental floss to the instrument, supposedly to retrieve it. It is so unnecessary! If one insists on placing rubber dam clamps before the dam is placed, the clamp should probably be fitted with a long string of dental floss to aid in its recovery (Figure 9-16).

If instrument aspiration or ingestion is apparent, the patient must be taken immediately to a medical emergency facility for examina-

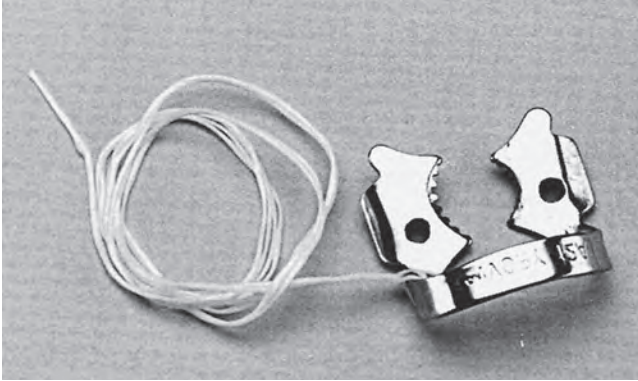


Figure 9-16. Routine placement of floss around the rubber dam clamp allows retrieval in the event the patient inhales it.

tion, and the dentist must accompany the patient. This examination should include radiography of the thorax and abdomen. It is helpful if the dentist takes a sample file along so the radiologist has a better idea of what to look for. The results may prove to be disastrous! The instrument may lie in the pharynx or the abdomen (Figure 9-17).



Figure 9-17. Swallowed endodontic instrument because a rubber dam was not used. The radiograph was taken 15 minutes after the broach was swallowed. Courtesy of B. Heling and I. Heling.

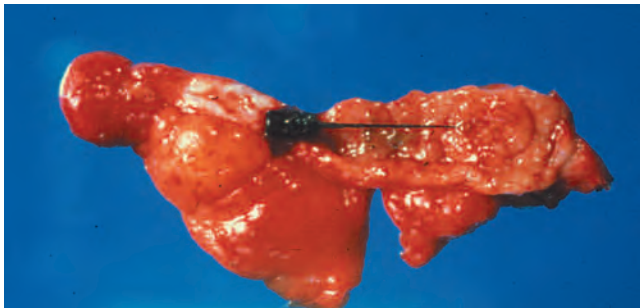


Figure 9-18. Appendix removed from a patient after an endodontic file ended up in the appendix. Rubber dam placement would have prevented this accident. Courtesy of L.C. Thomsen and colleagues.

Surgical intervention is the only solution (Figure 9-18). Be prepared for a session in court.

Management of Endodontic Emergencies*

Traumatic injuries to teeth can result in the fracture of hard tissues such as the teeth and bone, luxation of the teeth, and avulsion. See Table 10-1 for categories of dental trauma.

Table 10-1

CATEGORIES OF DENTAL TRAUMA

- Soft tissues
 - Lacerations
 - Contusions
 - Abrasions
- Tooth fractures
 - Enamel fractures
 - Crown fractures—uncomplicated (no pulp exposure)
 - Crown fractures—complicated (with pulp exposure)
 - Crown-root fractures
 - Root fractures
- Luxation injuries
 - Tooth concussion
 - Subluxation
 - Extrusive luxation
 - Lateral luxation
 - Intrusive luxation
- Avulsion
- Facial skeletal injuries
 - Alveolar process—maxilla/mandible
 - Body of maxillary/mandibular bone
 - Temporomandibular joint

*The author is indebted to Dr. Leif K. Bakland, Loma Linda University School of Dentistry, Loma Linda, CA, who contributed most of this chapter.

FRACTURES

Fractures of hard tissues include crown fractures, crown-root fractures, root fractures, and alveolar bone fractures.

Crown Fractures

Crown fractures may or may not include pulp exposure and are confined to the clinical crown of the tooth (Figure 10-1). If the fracture is confined to enamel, minimal effort is needed to manage the damage: smooth the fractured enamel edge or possibly repair with bonded resin material. An overlooked concomitant injury, tooth luxation, can result in pulpal injury; it is advisable to examine for possible pulp injury following all impact trauma to teeth.

If the crown fracture involves enamel and dentin but the pulp is not exposed, treatment can readily be done with current restorative techniques; possible pulpal damage should be monitored (see “Luxations,” following). Crown fractures resulting in pulpal exposure require attention to the pulpal injury. In an adult with a fully developed tooth, it is reasonable to consider root canal treatment prior to the restorative procedure. But it is also reasonable to consider the technique described for developing teeth—vital pulp therapy—as an option if the restoration can be accomplished with a bonded restoration or a rebonding of the fractured tooth fragment.



Figure 10-1. Crown fracture involving tooth no. 8 in a 9-year old girl. The goal of treatment in young patients is to protect the pulp for further root development. **A**, Clinical photograph of fractured tooth. *Continued on next page.*



Figure 10-1. Continued.

B, Radiograph showing fracture involving coronal pulp. **C**, Radiograph taken after shallow pulpotomy (see Figure 10-2 for procedure). **D**, After coronal restoration, which can be done either with composite build-up or by rebonding the fractured crown segment, if available. **E**, Radiograph taken 7 years later demonstrates continued root formation.

Crown fractures in developing teeth in young patients need special attention. If a developing tooth loses pulp vitality before it is fully formed, it is likely to be at risk for cervical root fracture within a few years. Every effort must be made to preserve pulp vitality in such teeth, and current techniques have been shown to be very successful in protecting pulp vitality.

Vital Pulp Therapy

The purpose of vital pulp therapy is to protect exposed pulp and allow it to continue its normal function—root development. Pulp capping and pulpotomy are both vital pulp therapies; the former is a technique in which the therapeutic dressing and restorative materials are placed directly on the exposed pulp, and the latter requires a small amount of pulp tissue removal prior to the placement of the materials. Pulpotomy is preferable to pulp capping because it results in a more secure placement of materials. The following describes pulpotomy treatment, first using calcium hydroxide (CH) and, second, using mineral trioxide aggregate (MTA).

Pulpotomy with Calcium Hydroxide

Figure 10-2 shows the steps in a pulpotomy with CH. After anesthetizing the tooth with crown fracture and pulp exposure, isolate the tooth with a rubber dam and disinfect the area with either sodium hypochlorite (NaOCl) or chlorhexidine. Next, remove granulation tissue that has developed from the pulp wound, and with the use of a round diamond stone (about the size of a No. 4 round bur) remove pulp tissue to a depth of about 2 mm into the pulp proper. It is advisable to create a bit of a dentin shelf around the pulp wound to support the CH dressing and restorative materials.

To control the bleeding from the pulpotomy site, place a cotton pellet soaked in saline on the wound and wait for the bleeding to stop. Then rinse away the formed blood clot with saline, and apply CH to the exposed pulp surface. The CH can be either a powder or in a mixed dressing such as **Dycal** (Dentsply/Caulk). Following the placement of CH, a flowable cement is applied over the CH and allowed to set, and the completion of the restoration can then be made by restoring the tooth with a bonded restorative material or by rebonding a crown fragment.

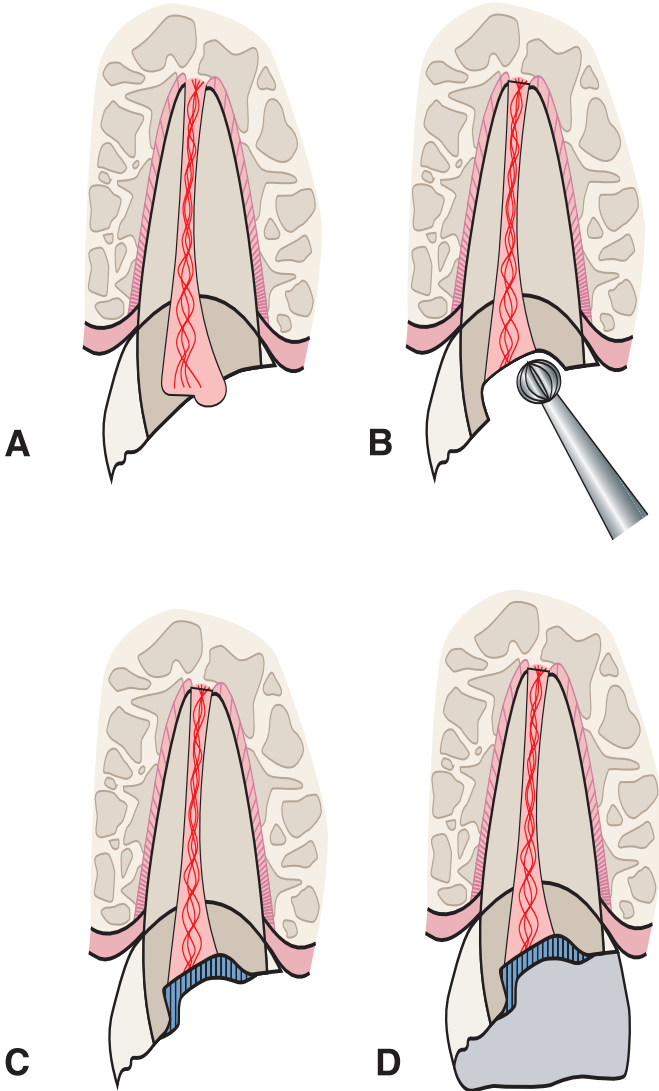


Figure 10-2. Shallow pulpotomy. **A**, Crown fracture exposes pulp. **B**, Remove pulp tissue with a round diamond bur to a depth of about 2 mm; use water spray to cool the diamond. **C**, After bleeding has stopped, wash the pulp wound with saline and apply calcium hydroxide liner, on top of which a base must be placed. The base can be glass ionomer cement. **D**, The lost tooth structure is replaced with acid-etched composite resin.

When using CH in vital pulp therapy procedures such as described here, it is important to recognize that CH will break down and leave a small space between the expected hard tissue forming subjacent to it and the restorative material next to it. If subsequently the restoration develops microleakage, the space previously occupied by the CH will be a suitable place for bacterial growth, resulting in injury to the underlying pulp. It is therefore recommended that after the development of a hard tissue barrier subjacent to the CH (usually after 3 to 6 months), the restoration should be removed and a new bonded restoration placed on top of the newly formed hard tissue.

Pulpotomy Using Mineral Trioxide Aggregate

The material, mineral trioxide aggregate (ProRoot MTA, Dentsply/Tulsa Dental), has been shown to have several advantages over CH when used for pulpotomy. First, MTA promotes a better-quality hard tissue across the pulp wound. Second, in contrast to CH, MTA does not disintegrate over time and does not need to be replaced. Third, MTA provides a very tight seal against adjacent dentin, providing a good barrier against microleakage.

The technique for using MTA in pulpotomy is similar to that for CH, with a few exceptions. Tooth preparation and pulp tissue removal is the same. However, it is not necessary to wait for a blood clot to form before placing the MTA on the tissue. Because MTA requires moisture for setting, it can be placed directly on the pulp, even if there is some minimal bleeding from the wound. The MTA should be allowed to set (4 hours) before completing the restoration of the tooth. The setting process can be allowed to take place in either of the following methods.

If the depth of pulpotomy is at least 2 mm, MTA can be used to fill in the entire cavity to the pulp proper, providing a layer of MTA at least 2 mm thick. The MTA is then set by being exposed to moisture from two sides: from the pulp and from saliva in the oral cavity. The patient should be advised not to use the tooth for biting or chewing for 4 to 6 hours. After the material has set, the definitive restoration of the tooth can be performed.

If the depth of MTA is less than 2 mm, there is a risk of flushing away MTA before it has set. In such a situation, it is advisable to cover the MTA placed on the pulpotomy wound with a moist cotton pellet, on top of which a temporary restoration must be placed. After

the MTA has set (4 to 6 hours), the temporary restoration and cotton pellet can be removed and the definitive restoration can be placed.

Crown-Root Fractures

As the name implies, crown-root fractures involve both coronal as well as radicular tooth structure, and may expose the pulp (Figure 10-3). Treatment is usually complicated by the severity of fracture.

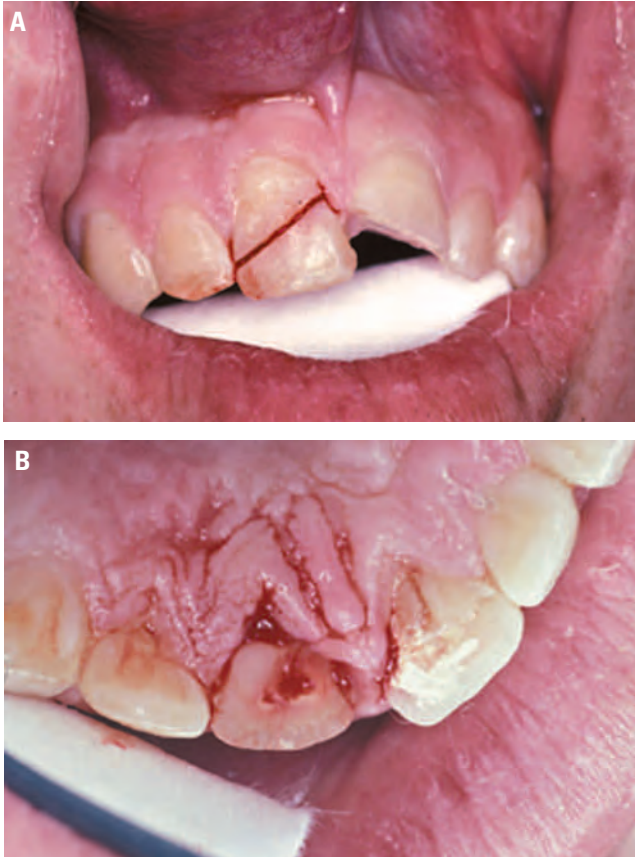


Figure 10-3. Crown-root fracture. **A**, Note the labial fracture line at midcrown level. **B**, After removing the fractured segment, one can see the palatal fracture level extends onto the palatal root surface. The reason the fragment stayed in place until physically removed is that it was still attached to periodontal ligament fibers.

Often both the crown and the root sustain a shattering injury, resulting in multiple fracture lines and necessitating extraction. A single tooth may be a candidate for an implant replacement, but one must be aware of the possibility that the labial bony plate may also have been damaged.

In fully developed teeth that are suitable for retention, it may be necessary to extrude or surgically expose the tooth in such a way that it can be restored. Developing teeth present an additional challenge in that it is desirable to protect the pulp for further root development. Failing that, crown-root fractured developing teeth have little chance for retention. When possible, pulp therapy procedures are similar to those performed when the fractures are confined to the crown of the tooth.

Root Fractures

Root fractures (Figure 10-4) are relatively uncommon compared to other traumatic injuries involving teeth. This has resulted in many misconceptions, for example, that root fractured teeth have poor prognosis (generally, the prognosis is good) and that these teeth require root canal treatment in most instances (the opposite is true). Management of root fractures is a two-phase procedure (Figure 10-5):



Figure 10-4. Radiograph showing midroot fracture (*arrow*) of tooth no. 8. Contrary to the belief of many dentists, root-fractured teeth respond very favorably to treatment.

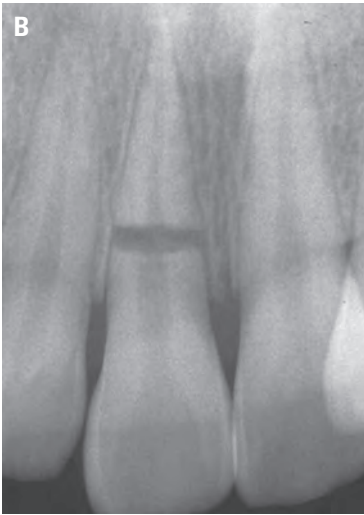


Figure 10-5. Management of root fractured tooth. **A**, Clinical photograph shows coronal part of tooth no. 8 displaced in an extrusive direction. **B**, Radiograph shows location of fracture and separation of the root at the line of fracture. **C**, Photograph shows the splinting completed after repositioning of the coronal part of the tooth. No root canal treatment was necessary.

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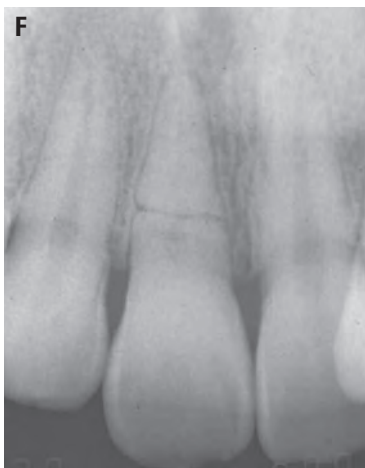


Figure 10-5. Continued. **D**, Radiograph taken immediately after the reduction of the fracture and splinting. **E**, Photograph taken after removal of the splint. **F**, Radiograph taken 2 years post-trauma. Note the healing by calcification within the root canal space. No root canal treatment was necessary.

1. As soon as possible after the injury has taken place, reposition the coronal segment of the tooth (if it has been displaced) and stabilize it with a nonrigid splint for 4 to 6 weeks.
2. Monitor the progress of healing and consider root canal treatment only if there is evidence of pulp necrosis (development of osteitis surrounding the fracture site).

If root canal treatment becomes necessary, as determined by the presence of pulp necrosis, it is usually only indicated for the coronal tooth fragment (Figure 10-6). Root canal treatment for teeth with root fractures that develop pulp necrosis includes one of the **following techniques**:

- Conventional root canal procedures—cleaning, shaping, and filling the canal—but only to the fracture line
- The use of CH to induce a hard-tissue barrier at the line of fracture before filling the canal
- Filling the coronal part of the canal to the fracture line with MTA.



Figure 10-6. Tooth with root fracture requiring root canal treatment. **A**, Pulp necrosis is present in the coronal segment; the lesion associated with the fracture line (*arrow*) supports the diagnosis of pulp necrosis. **B**, Root canal treatment is performed to the fracture line. The apical pulp tissue is vital and need not be removed.

When using a conventional root canal procedure for a root fractured tooth, it is usually not necessary to cross the fracture line when cleaning and filling the canal. If the pulp in a tooth becomes necrotic after the injury, the apical segment usually contains vital tissue, illustrated by the presence of bony lesions at the site of fracture and not at the apical area. However, this does not preclude both segments from being included in the treatment; if both segments are suitably aligned, canal cleaning and filling can be done in both segments—also an acceptable treatment choice.

When it is desirable to induce a hard-tissue barrier at the apical end of the coronal segment, for instance, in root fractured developing teeth with large-diameter pulp canals, CH can be used in the same manner as when apical closure (apexification) is performed. After the canal is cleaned, CH is placed in the canal and left until apical closure has occurred, at which time the canal can be filled without the risk of extruding filling material out the apical opening.

The new endodontic material, MTA, is also suitable for use in treating root fractured teeth with pulp necrosis. It can be used both in developing as well as fully developed teeth. In the case of root fractured developing teeth in need of root canal therapy, MTA has the added advantage that the procedure can be performed over a shorter period of time than when CH is used. The canal can be filled with MTA in one appointment and the coronal restoration on the next.

Alveolar Fractures

Fractures of the alveolar bone are considered here because of the potential that such fractures have to cause pulp necrosis in teeth, particularly in the bony fracture lines. Such teeth must be monitored for development of pulp necrosis and treated endodontically when indicated. Done in a timely fashion, root canal treatment in these teeth also promotes healing of the alveolar fracture (Figure 10-7).

LUXATIONS

Sudden impact blows to the dentition can cause injuries ranging from pulp concussion to tooth intrusion. Common to all these is trauma to the neurovascular supply to the pulp and supporting

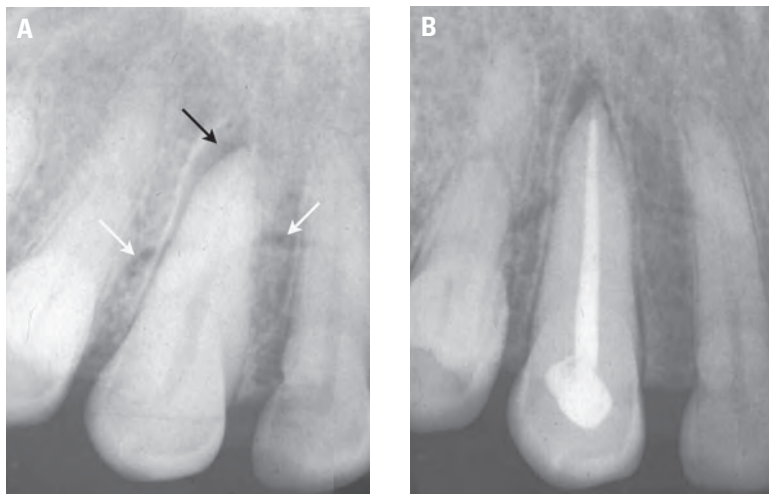


Figure 10-7. Alveolar fracture involving the tooth socket. **A**, Note the alveolar fracture (*white arrows*) and the fact that the tooth is displaced, as indicated by the apical radiolucency (*black arrow*). **B**, Root canal treatment for teeth in line of alveolar fracture is indicated if there is pulpal necrosis.

structures—periodontal ligament (PDL) and alveolar bone. The injury to the PDL can be either a separation or a crushing injury, the latter being the more destructive and taking longer to heal. Separation injury occurs when the tooth is displaced away from the bony socket wall (e.g., extrusive luxation), whereas a crushing injury results from the tooth being forced against the bone (e.g., intrusive luxation; Figure 10-8).

Affecting the outcome of traumatic injuries is the extent of damage to the neurovascular supply to the pulp. Severance of the blood supply leads to pulp necrosis (coagulation necrosis), which can become a fertile growth medium for bacteria, if present. The end result is infection-related resorption (inflammatory resorption) that involves both the tooth and the surrounding bone. In addition, if enough damage has involved the cementum and PDL, ankylosis-related resorption (replacement resorption) destroys the tooth. Thus, the combination of pulpal and PDL trauma during a luxation injury can have serious consequences for the injured tooth, and the treatment of such teeth is directed toward providing an environment in which the tooth may make complete recovery.



Figure 10-8. Luxation injuries. **A**, Extrusive luxation results in displacement out of the alveolar socket. **B**, Intrusive luxation forces the tooth against the bony wall, resulting in a disappearance of the periodontal ligament space.

Treatment of luxated teeth consists of repositioning the tooth, if necessary; stabilizing the tooth, if it is mobile, to promote PDL repair; and monitoring the pulpal condition and providing endodontic treatment if the pulp undergoes necrosis. The goal of treatment is to prevent root resorption. It has been shown that stabilization should be **nonrigid** to allow functional movement, which appears to reduce the risk of resorption. Further, the splint should be in place only long enough to allow reorganization of PDL fibers—2 to 4 weeks is sufficient.

Pulp survival in mature, fully formed teeth decreases with increasing luxation severity; the greater the injury to the pulp's blood supply, the more likely it is that pulp necrosis will result. Root canal treatment is indicated in cases of pulp necrosis. Pulp testing is in order.

Reduction in, or severance of, the blood supply to pulps in developing teeth is of more serious concern than in mature teeth. Pulp necrosis in an undeveloped tooth can result in a very weak tooth, prone to cervical root fracture. Apexification procedures in such teeth are done simply to induce a hard apical barrier to contain the root canal filling. The tooth will still be weak, and it appears that

long-term exposure to CH (as in apexification procedures) additionally weakens the tooth by making the dentin more brittle. A better approach is to use the CH for only a short time (less than 1 month) to disinfect the root canal, and then fill the canal with MTA.

An important consideration in **traumatized developing teeth** is that as unfavorable as pulp necrosis is in these teeth, by virtue of their large apical openings these teeth have the possibility of having a new blood supply enter the pulp tissue that was deprived of blood as a result of the injury. Such revascularization is not likely in a tooth with an apical opening of less than 1 mm. In a developing tooth, revascularization is an important opportunity that must not be overlooked; revascularization can result in continued, normal root development. Therefore, luxated developing teeth should be continually monitored for return of pulpal activity, typically seen as a continuing reduction in pulp lumen size.

Management

The goal in treating luxation injuries is to allow reestablishment of the PDL connection between the root and adjacent bone, promote revascularization of pulp in developing teeth, and prevent root resorption.

When damage has occurred to the PDL (separating or crushing injury), reestablishment of the PDL between the root and the bone can be enhanced by careful repositioning of the tooth, if displaced, and functional stabilization (by use of a semi-rigid splint) to promote PDL healing. Splinting is usually necessary for 2 to 4 weeks, depending on the severity of injury. Crushing injuries take longer to heal.

The splint used for dental injuries can be constructed by bonding unfilled resin to small etched areas of the involved teeth. If there is a significant space between teeth, this space can be spanned with the use of a thin, soft wire attached to the resin (Figure 10-9).

In young developing teeth in which damage has occurred to the pulp's blood supply, revascularization is a very desirable outcome. If bacteria can be prevented from entering the traumatized pulp (by protecting any exposed dentin in a concomitant crown fracture), the pulp has a reasonably good chance of recovering through revascularization. Careful and timely monitoring is essential. If revascularization does not take place, infection can lead to rapid, extensive



Figure 10-9. Splinting of luxated and replanted teeth. **A**, Bond unfilled resin to small, etched areas of the teeth. Avoid etching interproximally. **B**, If there is a diastema, or more than one tooth is involved, a thin wire can be used to bridge the gap.

(inflammatory) resorption. Monitoring the progress or lack thereof can be done by testing with an electronic pulp tester and by radiographic evaluation to check continued root development (sign of revascularization), and by watching for indications of a lack of continued development (pulp necrosis) or infection-related resorption (pulp necrosis with infection).

Root resorption as a sequela to trauma can be either infection-related (inflammatory) or ankylosis-related (replacement) resorption. The former can be prevented or arrested, if already started, by removing the tooth's infected pulp and completing the root canal treatment (Figure 10-10). However, the type of resorption associated with tooth ankylosis is currently not responsive to treatment. If ankylosis occurs after a traumatic injury, the outlook for the tooth is poor (Figure 10-11).

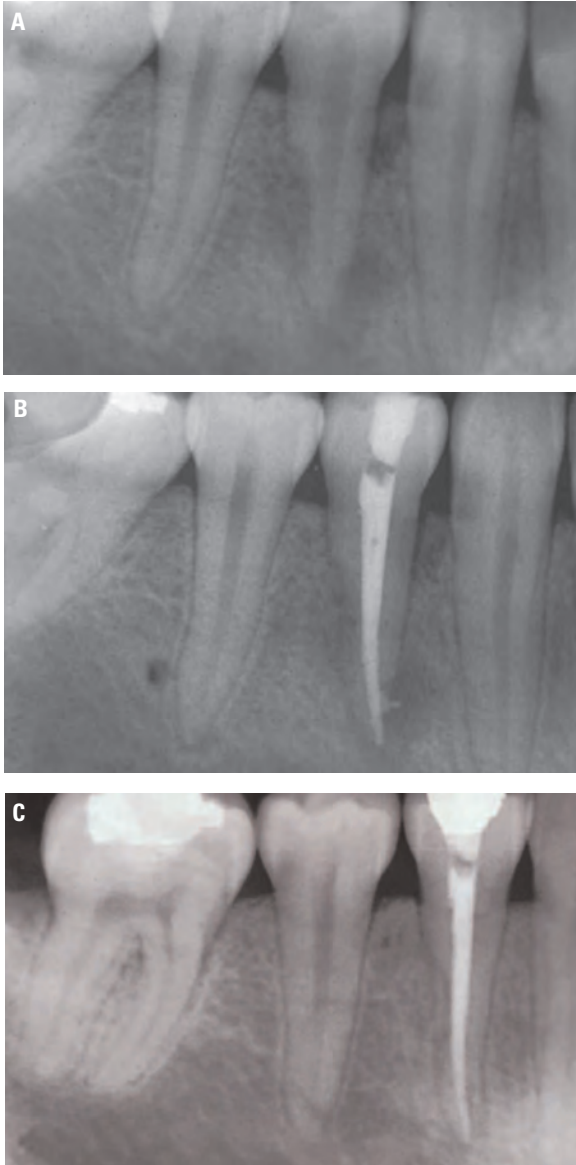


Figure 10-10. Infection-related (inflammatory) resorption. **A**, Radiograph taken 2 months after tooth no. 28 was traumatized. Note the resorptive defects in both the root and surrounding bone. Infection-related (inflammatory) resorption. **B**, Radiograph immediately after the root canal treatment. **C**, Follow-up radiograph shows healing of the bone and a reestablished periodontal ligament, the expected outcome when treating teeth with infection-related (inflammatory) resorption.

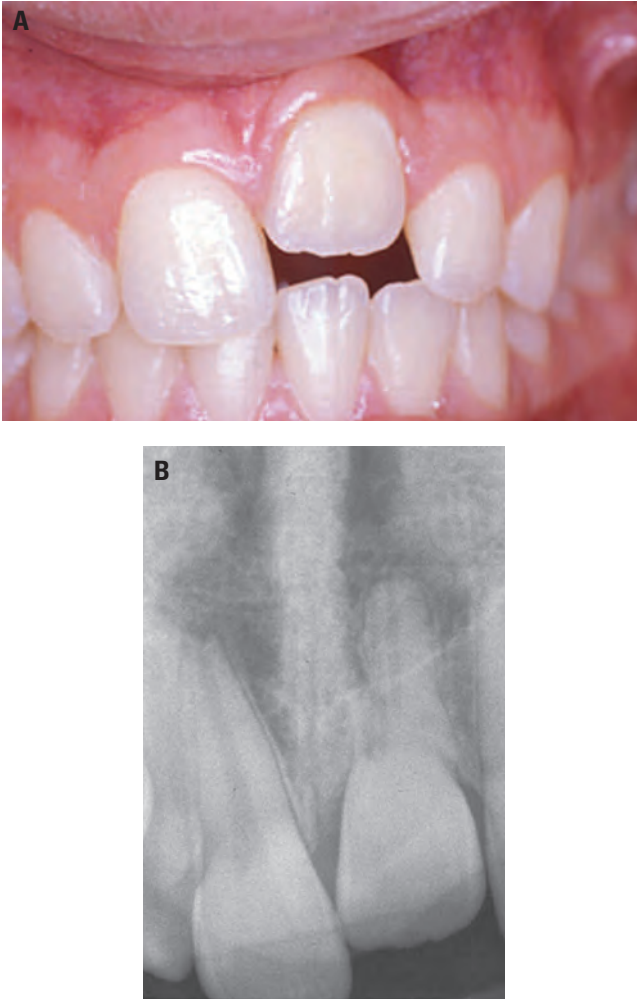


Figure 10-11. Ankylosis-related resorption, also called replacement resorption. **A**, Photograph shows tooth no. 9 in an infra-occlusal position as a result of trauma at an early age, causing the tooth to be ankylosed. **B**, Radiograph shows replacement of the root with bone, resulting in ankylosis.

Avulsion

A tooth that has been completely displaced from the alveolar socket is referred to as an avulsed tooth. Contrary to common belief, an avulsed tooth has a very good chance of being saved if the following two steps take place: the tooth is replanted back in its socket as soon

as possible (preferably within the first 15 minutes after avulsion) and root canal treatment is performed within 1 to 2 weeks. The only exception is very immature teeth with wide open apices in which revascularization is a possibility.

If the tooth cannot be replanted on site of injury, a simple method for preserving the important cells and fibers on the root surface is to place the tooth in a cup of milk for transport to the dentist (Figure 10-12). Other transport media (e.g., **Hank's Balanced Salt**

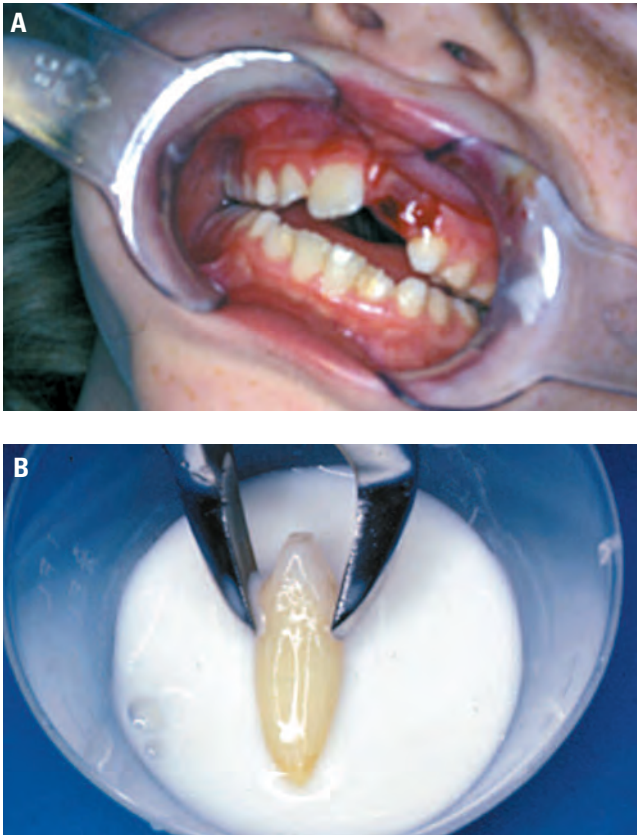


Figure 10-12. Management of tooth avulsion. **A**, Young girl who has lost a tooth in a traumatic accident. If the tooth can be found and replanted immediately, successful re-attachment of the periodontal ligament fibers can be anticipated. **B**, Milk Transport. If the tooth cannot be replanted on site, transporting it to the dentist in milk can also result in successful replantation.

Solution, Save-A-Tooth, 3M) may be more successful in supporting cell vitality, but none are more readily available than milk. If milk is not available, saliva is acceptable. (Keep the tooth in the mouth, next to the cheek, so it will not be swallowed.) Water storage is not recommended, being only slightly better than keeping the tooth dry.

Management of avulsions consists often of first advising the patient (or parent) over the phone to replant the tooth and come to the office for further care. Alternatively, the patient may bring the tooth in milk. Next, the tooth should be checked for position if already replanted, or replanted if brought in a storage medium.

After replantation, examine the gingival tissues and suture any concomitant lacerations. Splinting is necessary most of the time; use a semi-rigid splint. This must be in place only until the root canal treatment is started in 1 to 2 weeks. In most cases, it is more convenient to keep the splint in place while initiating root canal treatment—making the access opening and extirpating the pulp, followed by medicating the canal with CH. After closure of the access opening with a temporary restoration, the splint can be removed. In 1 to 2 weeks, the root canal can be filled and the coronal access restored with a bonded resin restoration (Figure 10-13).

Additional treatment of the avulsed tooth consists of administering antibiotics for the first week (e.g., penicillin 500 mg four times per day, with a reduced dosage for children) and rinsing the area



Figure 10-13. Replantation of an avulsed tooth. **A**, Photograph showing the avulsion of tooth no. 9. Courtesy of Dr. Mitsuhiro, Japan. **Continued on next page.**

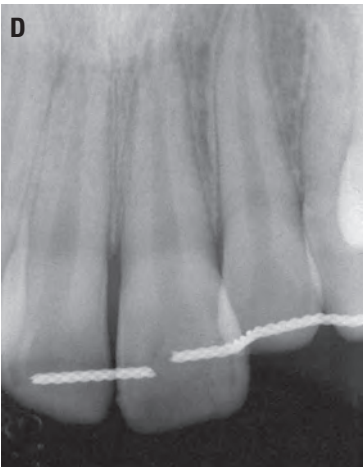


Figure 10-13. Continued. **B**, The tooth was brought to the dentist in milk. At the dental office, the tooth was examined and placed in saline during the examination of the avulsion site. **C**, The tooth has been replanted and splinted. Note the suturing of gingival tissues to improve adaptation. **D**, Radiograph of replanted tooth. Before removing the splint, root canal treatment is started, optimally 10 to 14 days post-replantation. **Continued on next page.**



Figure 10-13. Continued. **E**, Photograph taken after the removal of the splint, about 2 weeks post-replantation. **F**, Follow-up radiograph after root canal treatment and coronal restoration. The prognosis for the tooth is good. Courtesy of Dr. Mitsuhiro, Japan.

around the replanted tooth with chlorhexidine. Good oral hygiene promotes healing.

For avulsed, immature teeth with minimal root development (children less than 9 years old), it is desirable to reestablish vascularization of the pulp following replantation (Figure 10-14). This is unpredictable, and if the pulp does not survive clinical judgment must be used to decide whether endodontic therapy is preferable to extraction. It must also be recognized that even in cases of revascularization, ingrowth of bone into the pulp space can result in ankylosis and loss of the teeth.

For teeth that have been left to dry for more than one hour, replantation can still be done but the expected sequelae is ankylosis-related

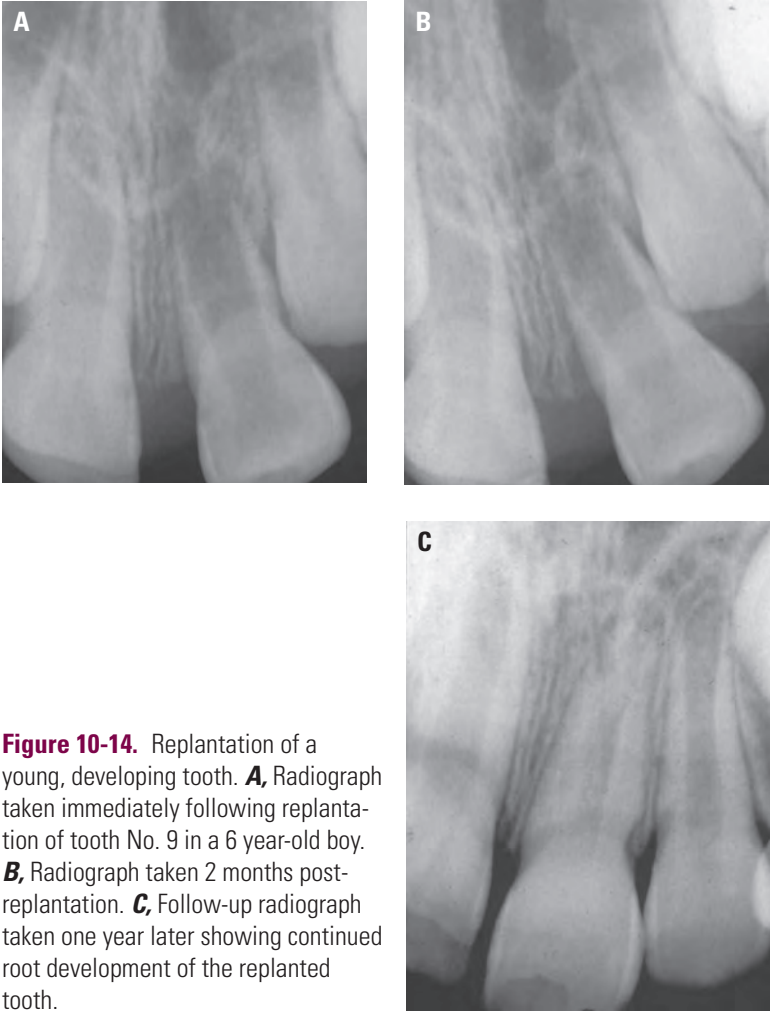


Figure 10-14. Replantation of a young, developing tooth. **A**, Radiograph taken immediately following replantation of tooth No. 9 in a 6 year-old boy. **B**, Radiograph taken 2 months post-replantation. **C**, Follow-up radiograph taken one year later showing continued root development of the replanted tooth.

resorption and eventual tooth loss (Figure 10-15). If it is decided that saving such a tooth for a limited time frame is still preferable to any other alternative, the procedure for replantation differs from that used for teeth with vital PDL attached to the root surface. The dry, necrotic PDL in the teeth with long extra-alveolar time periods should be carefully removed without damaging the cementum. Then the tooth is soaked in sodium fluoride solution for 5 to 20 minutes, after which root canal treatment can be done extra-orally and the



Figure 10-15. Ankylosed replanted tooth. The tooth had been left dry for several hours before the teenage patient was seen by a dentist. Before replantation, root canal treatment was done from the root apex. The radiograph was taken 5 years post-replantation, showing replacement resorption and ankylosis resulting in infra-occlusion because the adjacent teeth were still erupting. The tooth will be replaced with a bridge.

tooth replanted. The tooth is splinted for 6 weeks to allow bony apposition against the cementum; no PDL is expected to develop between the root and the bone. Eventually, osteoclasts gradually remove first the cementum and then the dentin until all that remains is the root canal filling. This process may take years and may be worth the effort in many instances.

ACUTE PAIN

Acute pupal and periradicular pain is discussed in Chapter 1. Here the discussion deals with pharmacologic intervention—what drugs can be used to abort, diminish, or prevent further pain.

Pain from pulpitis (pulpalgia) is best controlled by removing the inflamed pulp that is the source of the pain. However, to tide a patient over a weekend who is experiencing only moderate pain, one

would prescribe a moderate analgesic, such as acetaminophen two tablets of 500 mg q4h, ibuprofen two tablets of 200 mg q4h, or even aspirin two enteric-coated tablets of 325 mg q4h. Celecoxib and rofecoxib have no advantage in these cases over the drugs named here and are exceedingly more expensive and under suspicion of causing heart disease.

If the patient proclaims a “raging” toothache (**advanced acute pulpalgia**—pain that can be **controlled momentarily with an ice water rinse**), none of these moderate analgesics will have any effect, nor in some cases will narcotics help. The only answer is pulpectomy of the highly inflamed pulp. However, its removal may have to be followed with administration of acetaminophen, ibuprofen, or even aspirin. Antibiotics are not necessary.

On the other hand, periradicular pain often requires a much stronger analgesic. When a patient is developing a periapical abscess and it is in the developing stage with a hardened (indurated) appearance, the patient should be carried on codeine one or two tablets 30 mg q4h, for example, hydrocodone two tablets of 5 mg q4h, or oxycodone one tablet of 5 mg q6h. Sometimes pain at this stage of a developing abscess can be very severe and may require meperidine (Demerol) one tablet of 100 mg q4h or even morphine.

These recommendations are made with the following caveat: In prescribing narcotics, one must know exactly what one is doing. Never should they be given to a patient suspected of having or having had narcotic dependence. Also, dosage must be adjusted downward in prescribing to children, the elderly, or small adults. In addition, a federal license (and sometimes a state license) is required to prescribe narcotics.

Once an abscess has “pointed,” that is, become soft (fluctuant), pain is usually diminished. This is because it has broken through the cortical bone that encased it. When it is incised and drained (see Chapter 8), the trauma of the surgery may produce more pain, and some patients may need to be continued for a short time on these stronger analgesics, tapering off with the milder forms.

ACUTE INFECTION

Antibiotics are unnecessary and are **contraindicated for treating pulpitis** characterized by pulpalgia. On the other hand, antibiotics

are often indicated when one is dealing with periradicular disease. Treatment of acute infections periapically may require antibiotic administration, not so much to abort or control the emerging abscess but to prevent a **bacteremia** that may develop out of the infection. If the patient has an incipient heart lesion, the blood-borne bacteria **may colonize** there as bacterial endocarditis. These patients should routinely be premedicated prophylactically with antibiotics before invasive dental treatment.

Unless one is allergic to penicillin, it is usually the beginning antibiotic of choice to control acute infections: **penicillin VK** 5 mg qid, **ampicillin** 500 mg qid, or **amoxicillin** 500 mg tid/qid. All are effective, with penicillin VK being the least expensive. In the event penicillin does not appear to be effective, one might consider potentiating amoxicillin with **metronidazole** 250 mg to 500 mg qid, another antibiotic that works against obligate anaerobes. For those allergic to the penicillins, **clindamycin** 150 mg to 300 mg qid, as well as **doxycycline** 100 mg bid, are valuable antibiotics that are used to control bacteremias.

In Chapter 3, refractory periradicular infections were discussed, with anaerobes colonizing on the root ends of endodontically treated teeth. A long course of **metronidazole** has been found to be the antibiotic of choice for these chronic anaerobic infections.

Restoration of Endodontically Treated Teeth

In the year 1728, Pierre Fauchard in France was extolling the virtues of restoring pulpless teeth with post crowns (Figure 11-1). One hundred and twenty years later, Chapin Harris in the United States was proclaiming that “pivoting (*nee* posting) artificial crowns to natural roots... is the best that can be employed” (Figure 11-2). These **principles** have not changed in over 250 years of dentistry. However, post crowns have improved and root canal treatment is enormously better.

NEED FOR CROWNS

A question often posed is, do crowns need to be placed on endodontically treated teeth? The answer is that full coverage is generally not indicated for anterior endodontic teeth. Restoration with bonded resin is quite adequate. However, **for posterior teeth** there is considerable increase in success when **cuspal coverage** is placed. Unrestored **mandibular first premolars** might be the one exception to the rule. For these teeth, just as for anterior teeth, **bonded resin restorations** are the preferred treatment.

One also has to ask, do **posts** improve long-term prognosis or enhance strength? Again, the answer is that **posts fail to provide support** for endodontically treated teeth. This is especially true for anterior teeth. So posts need not be placed if the purpose of the procedure is to strengthen the endodontic tooth.

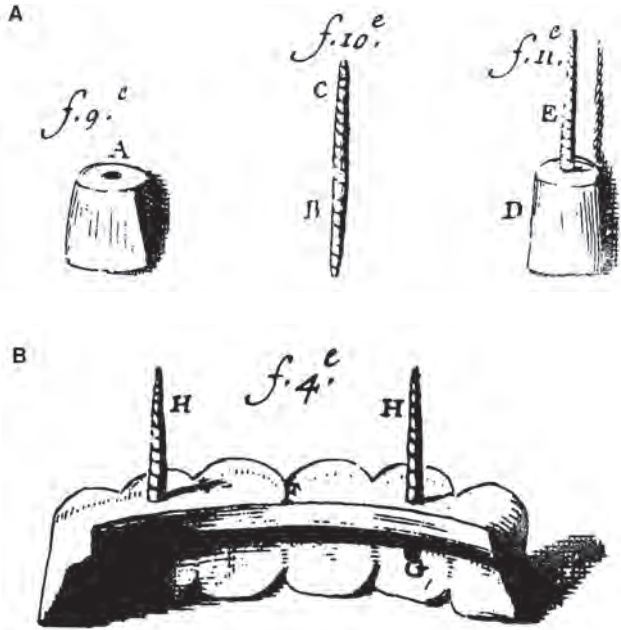


Figure 11-1. Fauchard's description, in the year 1728, of his attempts to restore single and multiple units. **A**, "Pivot tooth," consisting of a crown, post, and assembled unit. **B**, Six-unit anterior bridge "pivoted" in pulpless lateral incisors with the canines cantilevered. Crowns were fashioned from diverse materials: human, hippopotamus, and ox teeth, as well as ivory and leg bones. Posts were precious metal and were fastened to the crown with a sticky mastic. Nothing was said about treating the root canals before "cementation."

The purpose of **posts** is to **provide retention for a core**. Posts and cores do fail, with an average rate of 9%, but this is not too different from the endodontic failure rate. The two most common causes of post failure are **loss of retention** and **tooth fracture** (Figure 11-3). Posts bend and loosen or bend and fracture. However, posts that are too short, do not fit the canal and lose retention, or are far too large for the thin walls of dentin are much more apt to cause loosening and even root fracture.

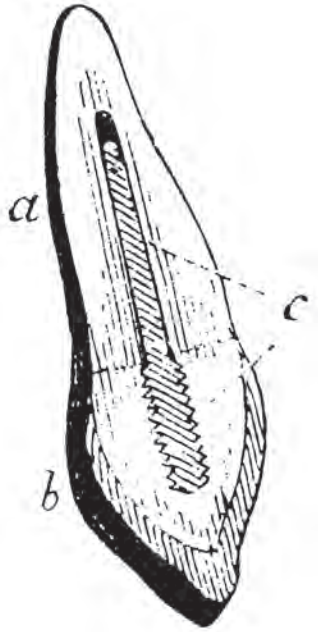


Figure 11-2. One hundred twenty years after Fauchard, Tomes in England was advocating a more sophisticated form of post-and-crown construction. Principles used today in selecting post length and diameter were taught by practitioners in the mid-1800s.



Figure 11-3. Fractured maxillary first premolar caused by a post with excessive diameter and insufficient length.

POST DESIGN

What, then, are the best post designs? What designs ensure the most retention? As one might suspect, it turns out that **threaded posts are the most retentive**, and tapered posts are the least retentive. On the other hand, threaded posts produce the greatest potential for root fracture. Threaded posts that cut their own threads into the dentin are the most threatening, even when the post is a split post. Threaded posts that fit into counterthreads prepared in the dentin are less threatening. **Parallel-sided posts with serrations** are more retentive than are smooth-sided parallel posts, and if they fit well in the canal they are the least likely to cause fracture. Posts that do not fit well and “float in a sea of cement” gradually lose retention. Also, caries attack the root facing if the post loosens and allows space to develop under the core.

POST LENGTH

The rule of thumb for the **proper length for a post** is **three quarters the length of the root** when treating long-rooted teeth. When an average root length is encountered, post length should be dictated by **retaining 5 mm** of apical gutta-percha and extending the post to the gutta-percha (Figure 11-4A). In curved roots, the post should terminate at the point where substantive curvature begins. **Posts too short** are not retentive enough to withstand the forces of mastication (Figure 11-4B), and **posts too gross do not increase retention and they materially weaken** the tooth, leading to root fracture (Figure 11-5). Post diameter should not exceed one third of the root diameter.

Laboratory testing and clinical observation have led to the conclusion that **4 mm to 5 mm of gutta-percha** should remain in the apical canal, 5 mm being preferable (see Figure 11-4A). It makes no difference if gutta-percha is removed at the time of canal filling completion or at a subsequent appointment. When 5 mm of gutta-percha is retained, a rotary or heated instrument appears to be acceptable in removing the excess gutta-percha.

To avoid perforations and because of their familiarity with the canal direction and size, endodontists are now being asked more frequently to prepare post space and even to cement the post to

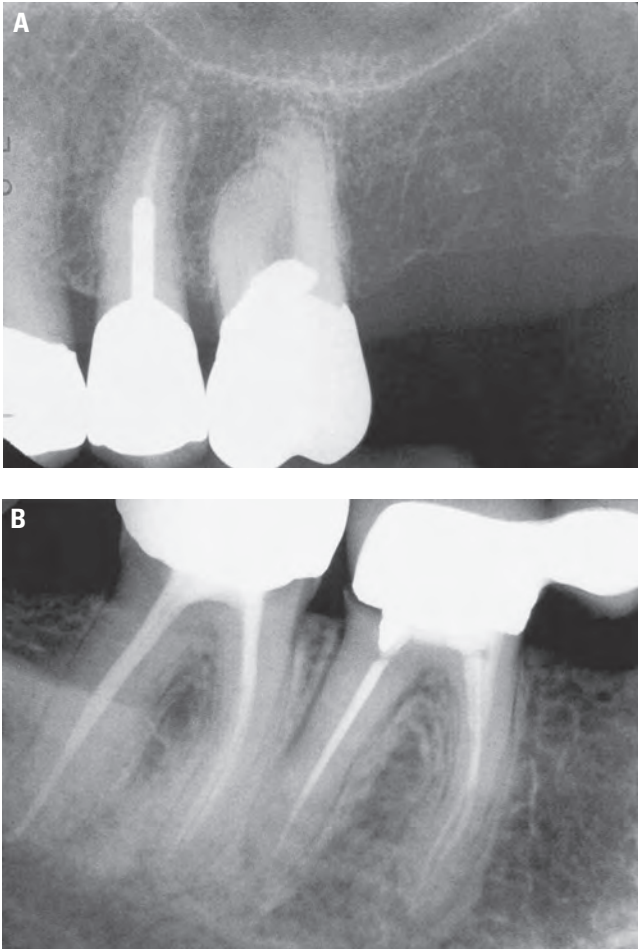


Figure 11-4. **A**, Five millimeters of gutta-percha root filling were retained in the maxillary premolar with a post extended to that point. **B**, Very short post in the distal root of the first molar has loosened and caused prosthesis failure.

place. In doing so, the endodontist is assuming part of the risk in the eventual failure of the case. It is therefore imperative that one knows well the capabilities of the referring dentist and has a definite understanding of the type of post to be placed. One caveat is that silver-point fillings should be removed and replaced with adequately condensed gutta-percha.

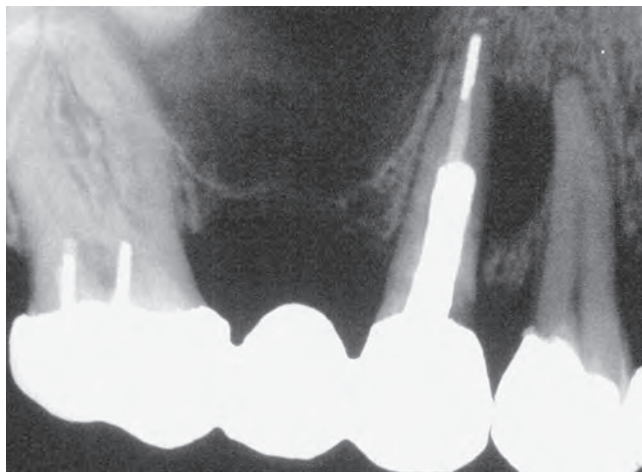


Figure 11-5. The excessive post diameter in the maxillary second premolar created a perforation in the mesial root concavity. Note the distinct border and round shape of the radiolucent lesion, a characteristic form indicative of root perforation. Pins in the molar (*arrows*) often perforate the pulp or set up fracture lines.

FERRULES

As stated previously, one of the principal causes of failure is **root fracture**. A principal factor in **preventing root fracture** is the placement of a **cervical ferrule**, a circumferential band of metal that surrounds the neck of the preparation. Two types of ferrules can be constructed: ferrules that are part of the post, that is, cast metal ferrules, and those that are part of the crown. It has been found that ferrules formed as part of the post are less effective than ferrules created when the overlying crown engages the tooth structure. In short, **crown ferrules are more effective than post ferrules**. It has also been noted that ferrules that grasp a larger amount of tooth structure are more effective than those that engage only a small amount of tooth structure (Figure 11-6).

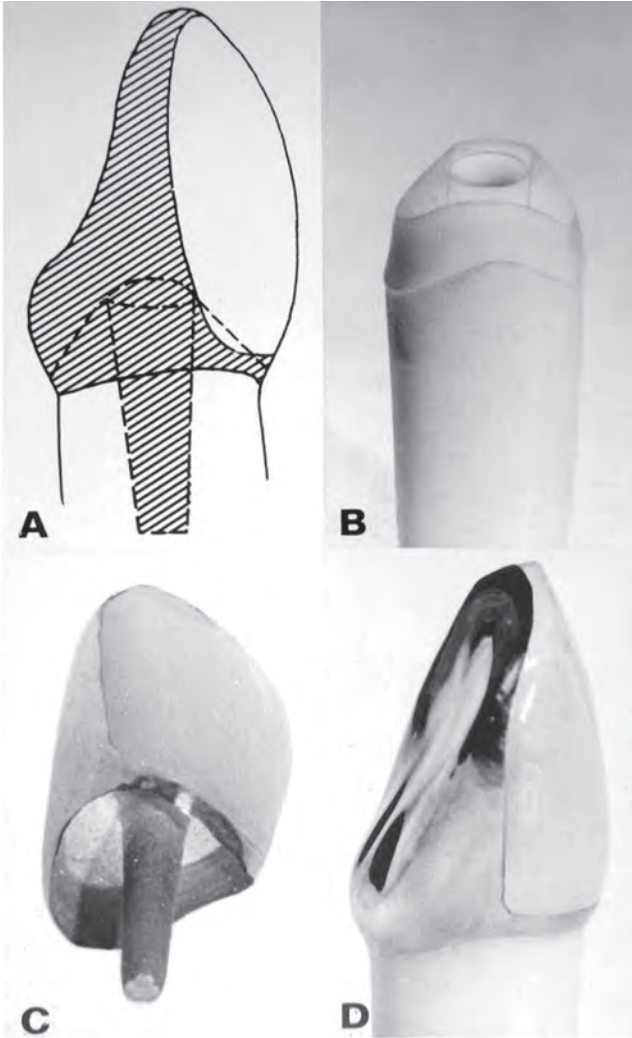


Figure 11-6. Excellent example of a crown ferrule that is extensive enough to strengthen the post crown in place. Courtesy of A.L. Frank.

TYPE OF POST AND CORE

Posts or dowels can generally be classified as cemented/bonded posts and threaded posts. For retention, cemented posts depend on their close proximity to the dentin walls and the cementing medium used. Variations are custom-made (cast) posts and cores, and prefabricated

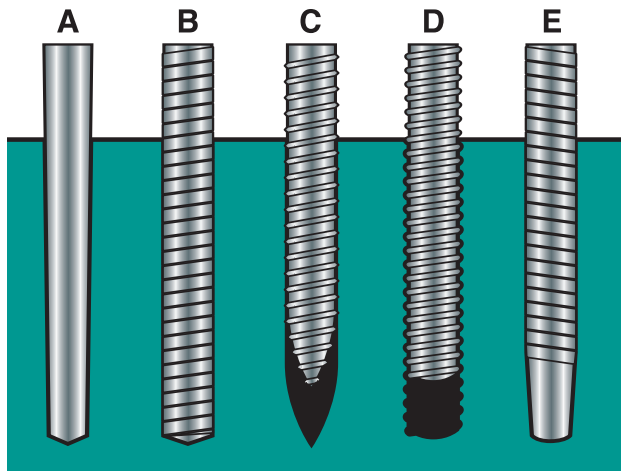


Figure 11-7. Prefabricated post designs. **A**, Tapered, smooth. **B**, Parallel, serrated. **C**, Tapered, self-threading. **D**, Parallel, threaded. Note that the post fits into pretapped threads cut into the dentin. **E**, Parallel, serrated, tapered end.

posts, either tapered, smooth metal posts (Figure 11-7A) or parallel-sided metal posts (Para-Post, Coltene/Whaledent; Figure 11-7B). Other than metal posts, there are also ceramic posts made of zirconium dioxide, posts made of carbon fibers and reinforced polymers, and white fiberglass/composite posts that do not show through translucent crowns (C-I White Posts, Parkell Co.; Figure 11-8).

There are two types of **threaded posts**: tapered, **self-threading posts**, that is, posts that are screwed into the dentin that cut their own threads into the dentin (Figure 11-7C); and posts that thread into **pretapped counterthreads** prepared in the dentin (Kurer posts; Figure 11-7D). In addition, there are tapered, finely threaded posts (Figure 11-7E) and tapered, coarsely threaded “split” posts (Figure 11-9).

Most of these threaded posts stress the dentin because they screw into it, much as a wood screw can stress and split wood. The least threatening of all these threaded posts is the **Kurer post** because it does not stress the dentin. For Kurer posts, the counterthreads are “tapped” into the dentin with a special instrument, and the threads of the post fit like a bolt into a nut, not like a screw into wood. In contrast, **tapered, threaded posts** add additional stress on root structure, and **tapered, split posts** have not been shown to provide any advantage over non-split types.



Figure 11-8. White C-I glass fiber/composite post (Parkell Co.) that does not show through metal-free translucent crowns.

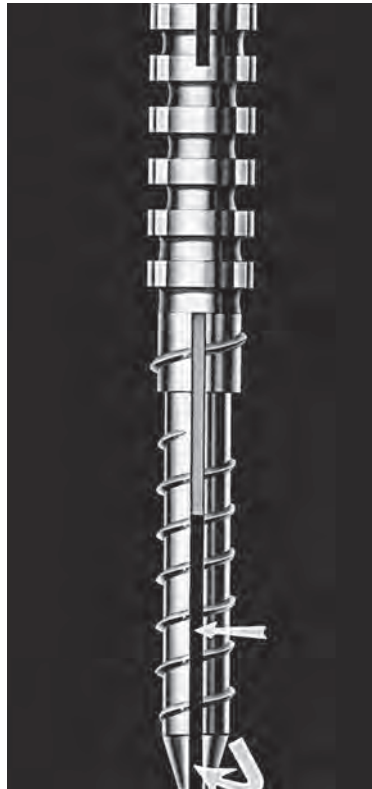


Figure 11-9. Flexi Post (EDS Co.). Note the "split" in the apical portion of the post that permits some flexion during placement.

PREPARATION OF A POST AND CORE

For illustration, one type of post-and-core restoration is outlined here: the metal, parallel, serrated Para-Post (Figure 11-10). It is assumed that the root canal filling has been completed.

First, the coronal tooth preparation is performed (Figure 11-11A). The amount of tooth structure that must be removed is related to the type of crown to be placed. Then, the chamfer for the ferrule is established. If some of the tooth structure is very thin, it is best to remove it and replace it with the core material.

Pulp Chamber Preparation

The best time to prepare the post space is when the root canal filling is completed. For this, Peeso drills and a twist drill matched to the size of the post to be placed are used (see Figure 11-10). First, the extra gutta-percha is removed with a warm plugger (Figure 11-11B).

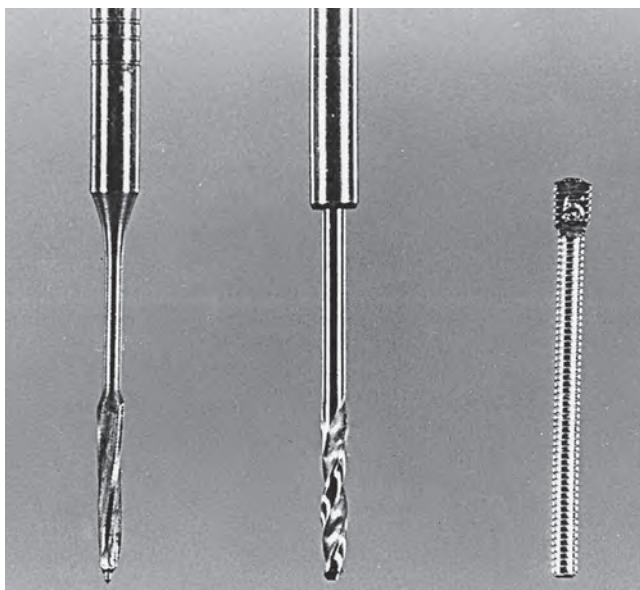


Figure 11-10. Para-Post (Coltene/Whaledent) is a parallel-sided, vented, serrated post (*right*). The canal is enlarged with a Peeso reamer (*left*), and the final channel preparation is made with a matched twist drill (*center*).

Remember, 5 mm of gutta-percha is to be left in the apical area, so any removal instruments should have a rubber stop placed so as to not exceed this depth. Next, advancing sizes of Peeso drills are used in a slow-speed handpiece until the required depth is reached and the dentin walls are enlarged to near the size of the post to be placed (Figure 11-11C). Then the Para-Post twist drill the same size of the post that is to follow is used to refine the space (Figure 11-11D).

Next is the trial placement of the post to verify approximation without binding (Figure 11-11E). It should be measured or radiographed to make sure it is at full depth, that is, up against the remaining gutta-percha. The post should then be shortened so that it does not interfere with closure and there will be enough space for the fabrication of the crown (Figure 11-11F). Before it is cemented to place, it has been suggested that the **smear layer be removed** from the dentin walls with a rinse of NaOCl and MTAD (ProRoot MTAD, Dentsply/Tulsa Dental). Finally, the post is cemented to place and the core is built up to receive the final restoration (Figure 11-11G).

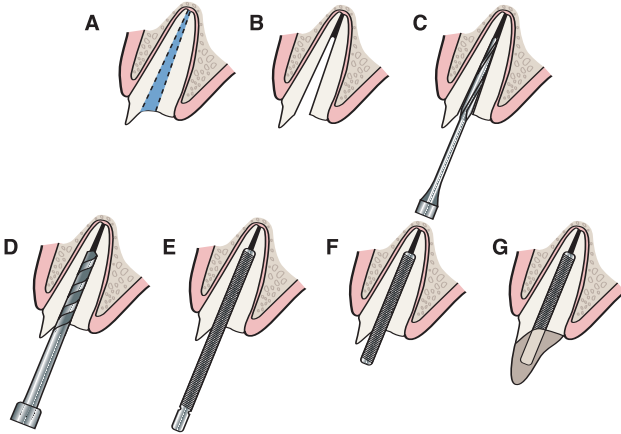


Figure 11-11. Placement of the parallel-sided Para-Post (Coltene/Whaledent) and composite resin core in an anterior tooth. **A**, Endodontic treatment completed. **B**, Gutta-percha removed. **C**, Post space formed with Peeso drill. **D**, Post space refined with Para-Post twist drill. **E**, Trial placement of the post. **F**, Shortening the post so it does not interfere with occlusal closure but with space for fabrication of the core. The post is cemented after shortening. **G**, The tooth is etched and the bonded-composite core is formed and shaped with rotary instruments.

OVERDENTURES

In 1789, George Washington's first lower denture, constructed of ivory by John Greenwood, was in part supported by a left mandibular premolar. In 1969, Lord and Teel coined the term "overdenture" and described the combined endodontic-periodontic-prosthetic technique applied thereto.¹ The concept of retaining roots in the alveolar process is based on the proven observation that as long as the root remains, the bone surrounding it remains (Figure 11-12). This overcomes the age-old prosthetic problem of ridge resorption (Figure 11-13). Ideally, retaining four teeth, two molars and two canines, one each at the four divergent points of the arch, should provide good balance and long life to a full overdenture (Figure 11-14).

If the abutment teeth are reduced to a short, rounded, or bullet shape—literally tucking the abutment inside the denture base—the crown-root ratio of the tooth is vastly improved, especially for periodontally involved teeth that have lost some bone support (Figure 11-15).



Figure 11-12. Overdenture abutment, well obturated and restored with amalgam. Note the excellent bony support. Courtesy of D.H. Wands.

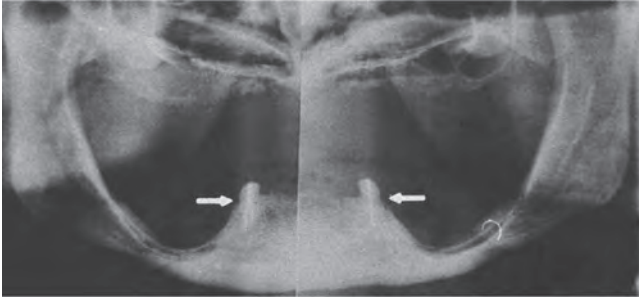


Figure 11-13. Dramatic demonstration of alveolar bone remaining around retained canines but badly resorbed under a full upper and a posterior lower partial. Courtesy of J.L. Lord and S. Teel.



Figure 11-14. Four retained abutments providing ideal support for an overdenture. Courtesy of A.A. Brewer and R.M. Morrow.

It goes without saying that root canal treatment is necessary for these abutment teeth. The crowns of the teeth are amputated 3 mm to 4 mm above the gingival level. The length of the remaining tooth is accurately established, and the pulps are removed. The canals are then cleaned and shaped, the smear layer is removed, and the canals are obturated with sealer and gutta-percha or Resilon. Next, the coronal 3 mm to 5 mm of the root canal filling is removed, the preparation is undercut, and an amalgam filling is placed with bonded sealer (AmalgamBond, Parkell Co.).

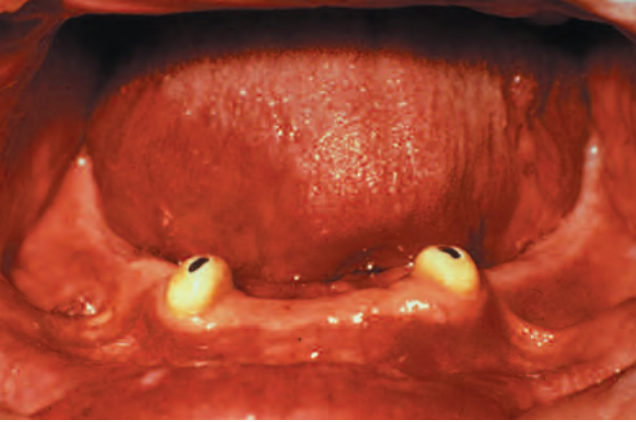


Figure 11-15. Mandibular canines that have served as overdenture abutments for years. Courtesy of A. Fenton and A.A. Brewer.

The abutments should then be properly bullet-shaped with a slope back from the labial aspect to accommodate the denture tooth to be set above it. The abutments must not be too short or tissue will grow over them as “lawn grows over a sidewalk.” The abutments, tooth, and amalgam are then highly polished. When the denture is ready to be seated, it is relieved over the abutment areas and a small amount of self-curing acrylic is placed in the relieved areas (Figure 11-16). This ensures a perfect fit over the abutments.



Figure 11-16. Soft, self-curing acrylic fills the depression prepared in the denture to receive the abutment. This may be replaced whenever necessary.

OVERDENTURE PROBLEMS

Most overdenture problems relate to poor patient care. Patients who have been neglectful of their teeth throughout their lifetime can hardly be considered prime candidates for good hygiene in the future. Recall appointments and bathing the teeth with sodium fluoride can be of some help. After all, two or four abutments should not be too hard to keep clean.

Another problem is **retention**. Fortunately, there is a solution to the retention problem. The **Locator Overdenture Attachment** (Zest Anchors) Escondido, CA has emerged as a remarkably clever solution to this age-old problem. The self-locating design allows patients to easily seat their overdenture. The post of the **female attachment** that fits in the endodontic tooth comes in three designs: a straight post and 10° and 20° posts to accommodate divergent roots (Figure 11-17).² The Locator Attachment is a parallel post that is notched for retention. It has a length of 6 mm but can be shortened to as little as 3 mm. It is stainless steel with a titanium-nitride coating and is passively cemented in place, not threaded or screwed into the root.

The canal is first prepared with a Locator pilot drill to the selected depth. This is followed by a countersink diamond bur that

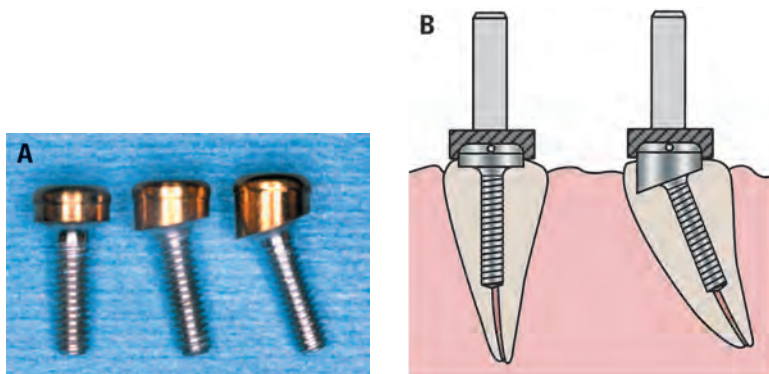


Figure 11-17. **A**, Locator Overdenture Attachments (Zest Anchors). Female attachments to be cemented into endodontically treated teeth: 0° (left); 10° (center); 20° (right). **B**, Illustration of how verticality and parallelism can be achieved with a 20° female attachment in a tooth abutment with divergent roots.



Figure 11-18. Four Locator Overdenture Attachments (Zest Anchors) ensure adequate retention for a mandibular full denture. Courtesy of A.L. Schneider.

forms a shallow recessed seat in the flat root surface. At least 1.5 mm of the female attachment must rise above the gingival crest to allow the male component in the denture to snap into place. The combined Locator attachment connection rises only 2.5 mm above the root face (Figure 11-18).³

The **male attachment**, processed in the denture, is stainless steel and fitted with a nylon insert that allows the metal cap to pivot up to 7° without contacting the surface of the root. This allows some flexibility in the denture movement, which allows the patient to easily self-align the denture over the attachments and snap it into position (Figure 11-19). The nylon insert snaps into the female attachment much like a clothes “gripper” or, as the British call it, a press-stud. The nylon inserts also come in three strengths: 2.3 kg (5 lb) for strong retention, 1.4 kg (3 lb) for medium retention, and 0.7 kg (1.5 lb) for light retention. When it comes to removing the denture, older or arthritic patients may find the light retention easier to manage. Husky males may prefer to have strong retention inserts. As the nylon inserts wear, they may easily be pried from position and a fresh insert snapped to place. Locator attachments are also made for osseointegrated implant overdentures.

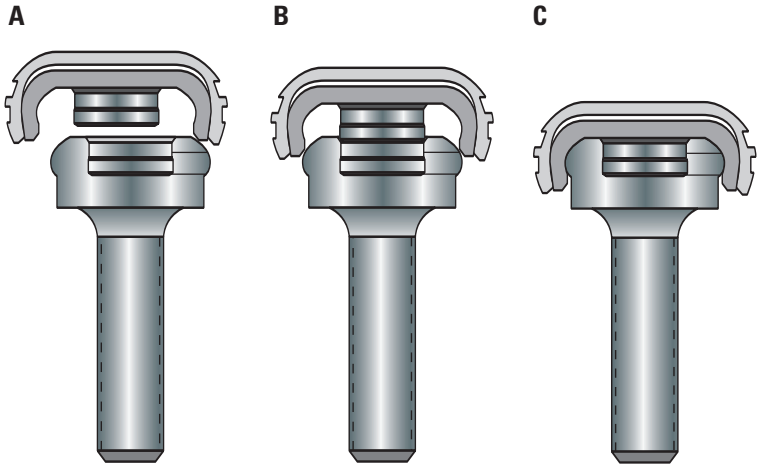


Figure 11-19. The male Locator Overdenture Attachment (Zest Anchors), which is implanted in the denture, is stainless steel with a snap-in plastic insert that, in turn, snaps into the female attachment below. **A**, The male attachment moving into place. **B**, The attachment positioned over the female receptacle. **C**, The male attachment snapped into place. When the plastic inserts wear, they may be pried out of the female attachment and replaced with new nylon inserts. The plastic allows movement in the denture.

REFERENCES

1. Lord JL, Teel S. The overdenture. *Dent Clin North Am* 1969;13:871.
2. Pavlatos J. The root-supported overdenture using the Locator overdenture attachment. *Gen Dent* 2002;Sept/Oct:448.
3. Schneider AL. The use of self-aligning, low-maintenance overdenture attachment. *Dent Today* 2000;19.

Discoloration and Bleaching

TOOTH DISCOLORATION

There are a number of causes for the discoloration of natural teeth. Some of them are caused by the patient, and some by the dentist.

Patient-Caused Discoloration

Pulp necrosis is one of the most common causes of discoloration. The longer the pulp has been necrotic, the more intense is the discoloration. Another cause is intrapulpal hemorrhage. A blow to the tooth bursts a vessel in the pulp, and the bleeding extends out into the dentinal tubules. If the pulp becomes necrotic from the blow, the blood remains in the dentin and the tooth gradually turns dark, presumably from the iron sulfides in the red cells (Figure 12-1). If the pulp retains its vitality, the discoloration may gradually fade and the tooth may regain its normal color.

A blow to the tooth may also stimulate the pulp to hyperactivity; as a result, dentin hypercalcification develops. The tooth becomes “old before its time” and stands out as being a different color from its neighbors. Along these lines, age is another cause of tooth discoloration. All the teeth become hypercalcified, and along with the discoloration from beverages plus the cracking and crazing of the enamel, the overall natural color gradually darkens.

Discoloration may be “built into” the teeth from birth. Excessive fluoride ingestion and tetracycline use, even by the mother during pregnancy, badly discolor the baby’s teeth (Figures 12-2 and 12-3). Fortunately, most physicians are now aware of this problem and no longer prescribe tetracycline or doxycycline during pregnancy or during early childhood.

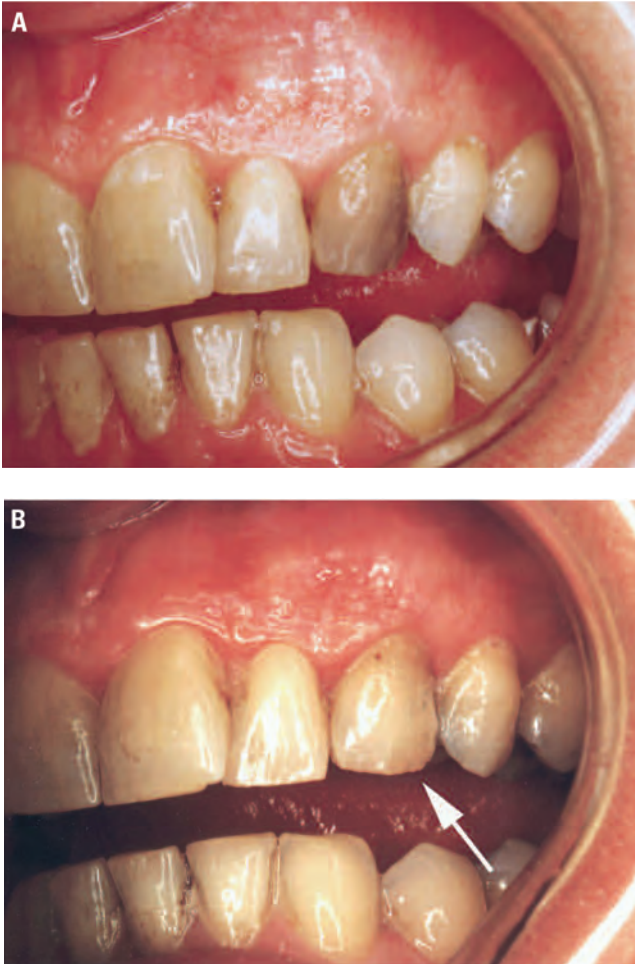


Figure 12-1. Traumatic pulp necrosis and dentin discoloration of a maxillary canine. **A**, Before treatment. **B**, Esthetic results following one treatment of “walking bleach” with sodium perborate and Superoxol. Courtesy of E.B. Nutting and G.S. Poe.

Dentist-Caused Discoloration

Most dentist-caused discoloration is avoidable. Pulp tissue or necrotic remnants left in the pulp chamber following root canal treatment is a cause of discoloration. The chamber must be scoured clean of debris before a final restoration is placed. Some intracanal medicaments, such as iodoform, and obturating materials, such as



Figure 12-2. Fluorosis of maxillary incisors treated by external bleaching. **A**, Before treatment. **B**, One month following bleaching treatment. Compare the treated maxillary incisors with the untreated mandibular incisors. Reproduced with permission from D. L. Meyers.

all sealers, must also be removed. Silver alloys have long been condemned for staining dentin a dark gray. Pins and posts can show through thin walls in the crown. (See Figure 11-8 for an example of a glass fiber/composite post that does not show through metal-free translucent crowns.) Composite resins not properly sealed by adhesives leave open margins that collect stains from ingested matter such as coffee and red wine as well as bacteria and yeasts. Leaking composites can be removed and replaced with better sealants.

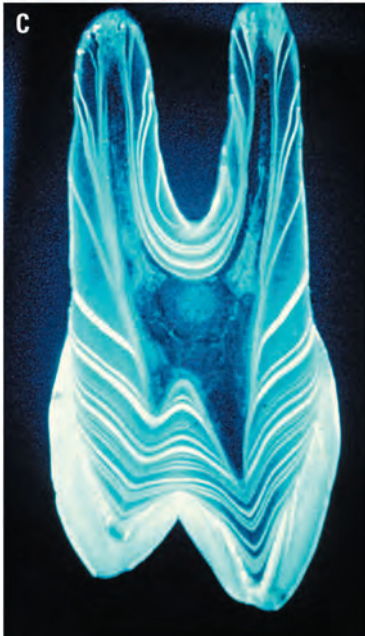


Figure 12-3. **A**, Fluorosis of central incisors treated by external bleaching. **B**, Bleaching results, 1 month later. Courtesy of D.L. Myers. **C**, Fluorescent photomicrograph of tetracycline-discolored tooth. Tetracycline deposition is seen as stripes caused by the start and stop ingestion. Courtesy of D.L. Myers.

BLEACHING AGENTS

Hydrogen peroxide in concentrations of 30% to 35% (Superoxol-Merck), sodium perborate, and carbamide peroxide are the presently used bleaching agents. **Hydrogen peroxide 35%** is caustic, and contact with tissue must be avoided. It must be kept refrigerated in a dark bottle. **Sodium perborate** is a dry powder, but when mixed with water becomes hydrogen peroxide and nascent oxygen (Figure 12-4). It is easily controlled and safer to use than 35% hydrogen peroxide. **Carbamide peroxide**, also known as urea peroxide, ranges in concentration from 3% to 45%. The most common form is 10%, which yields about 3.5% hydrogen peroxide. It is often combined with glycerin or propylene glycol and is used primarily for extra-coral bleaching, either in the dentist's office or at home.



Figure 12-4. Sodium perborate powder and water are mixed to the consistency of wet sand. Courtesy of I. Rotstein

INTRACORONAL BLEACHING

The so-called “**walking bleach**” is the preferred form for bleaching teeth that have had root canal treatment and have discoloration of the pulp chamber. It can be performed at the time of treatment or sometimes years later. The patient must first be informed of the possibility of failure or partial success. Also, any **root canal filling** of questionable quality must be redone before bleaching is undertaken. Defective restorations must also be replaced. Use of a shade guide and before and after photos are helpful.

Procedure

The steps for intracoronaral bleaching are as follows:

1. Isolate the tooth with a rubber dam.
2. Be sure the pulp chamber is free of all debris to below the labiogingival level (Figure 12-5). Remove any sealants with orange solvent or chloroform, and seal off the root canal filling with 2 mm of glass ionomer cement. This barrier should extend to the PDL attachment level. Seal the barrier and canal dentin (not the crown) with a bonding agent.
3. Pack the pulp chamber with an aqueous paste of sodium perborate (see Figure 12-4), dry it with cotton pellets, and cover it with a thick (3 mm) temporary filling, preferably of IRM cement. Inform the patient that it will probably be several days before an improvement is noticed.
4. Reevaluate the result after 2 weeks and, if necessary, repeat these treatments until a satisfactory result is achieved. If only partially successful, it may be necessary to add a drop of 35% hydrogen peroxide to the perborate paste.
5. When a satisfactory result is achieved, show the before and after photos to the patient (Figure 12-6).
6. Place a final restoration in the access cavity. Position white cement against the labial wall to ensure translucency, and then place an acid-etched, light-cured composite with a dentin adherent.

There can be some complications from intracoronaral bleaching. External root resorption at the neck of the tooth has been reported but usually in relation to the intracoronaral use of the strong 35% peroxide and the use of heat (Figure 12-7) The irritating chemi-

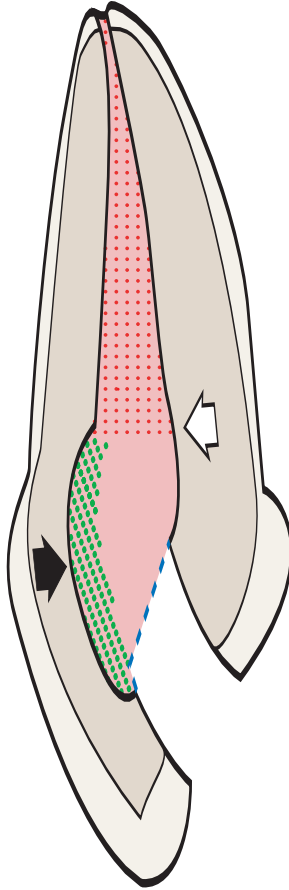


Figure 12-5. Gutta-percha, sealer, and dentin removal prior to bleaching. The *heavily dotted* area represents gutta-percha filling that is removed to a level below the attached gingiva (*open arrow*). The *lightly dotted* area represents the removal of dentin to eliminate heavy stain concentration and pulp horn material (*black arrow*).

cal evidently diffuses through the dentinal tubuli to destroy the cementum and periodontal ligament, eventually leading to external resorption—all the more reason to **place a cement layer over the obturation at the gingival level** (Figure 12-8). Hydrogen peroxide may also **break the bond** between composites and dentin, so the peroxide must be thoroughly removed from the crown before a final



Figure 12-6. Before, **A**, and after, **B**, photos demonstrating results of one internal bleaching treatment with Superoxol. The patient, a young medical student, recognized that the discolored tooth was off-putting and was overjoyed with the result.

composite restoration is placed.

VITAL BLEACHING

Bleaching **vital teeth** is usually not thought of as within the province of endodontics. However, it has become so because of endodontists' long experience in bleaching pulpless teeth. Applying oxidizers such as 35% hydrogen peroxide to the external enamel surface and enhancing the action by heat application ameliorate discoloration from a mild tetracycline stain, endemic fluorosis, or age-related

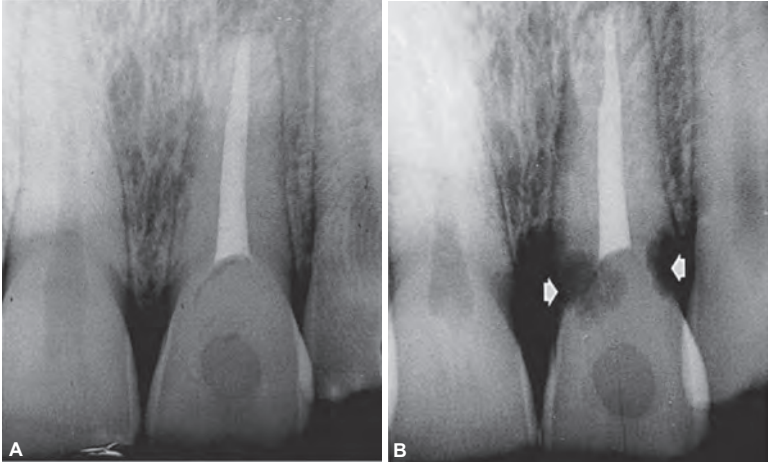


Figure 12-7. Post bleaching external resorption. **A**, Nine-year follow up radiograph of a central incisor devitalized by trauma and treated endodontically shortly thereafter. **B**, Radiograph taken 2 years following bleaching. Superoxol and heat were used first followed by “walking bleach” with Superoxol and sodium perborate. Reproduced with permission from D. Steiner and G. Harrington.

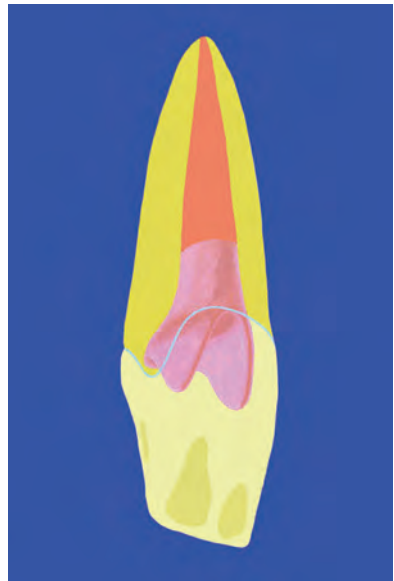


Figure 12-8. Schematic illustration of the intracorononal protective bleach barrier. The shape of the barrier matches the contour of the external epithelial attachment. Reproduced with permission from Steiner DR, West JD. J Endod 1994;20:304.

changes. However, hydrogen peroxide (35%) can be damaging to soft tissue, and heat application can injure vital pulps. Newer methods have generally replaced this caustic approach.

MOUTHGUARD BLEACHING

Mouthguard bleaching may be carried out in the dentist's office and at home by the patient. The product most often used is carbamide peroxide, and the percentage of peroxide ranges from 10% to 35%. There are presently 15 to 20 of these products on the market, including strips that contain the product and can be applied to the teeth. The most complete form involves confining the carbamide peroxide to the teeth with a **mouthguard** constructed in the dental office.

Office application of the bleach retained in the mouthguard may be for shorter periods of time but at a greater concentration of the peroxide, say, 35%. However, in home application the peroxide is usually in the 10% to 15% range, and application may extend for hours. Before and after photos should be taken so the patient can see the changes.

Procedure

The steps for mouthguard bleaching are as follows:

1. Take alginate impressions of both arches and pour up models. Mark the outline of the guards on the models, and paint two layers of die relief on the tooth surfaces. Fabricate a soft plastic matrix over the cast, and trim it to within 1 mm of the gingival margins (Figure 12-9A). Check the fit in the mouth.
2. If the product is being used in the office, the 35% carbamide peroxide gel is applied to the tooth indentations in the guard, which is then inserted into the patient's mouth for 30 minutes at a time (Figure 12-9B). When used at home, the 10% gel is used and the guard may be in place 3 or 4 hours at a time, with the gel replaced every 2 hours. Many patients wear the guard all night.
3. Treatment should take from 4 to 24 weeks, depending on the severity of the staining. The patient should return every

2 weeks so progress can be monitored, and to make sure that the soft tissues are not irritated.

Transient tooth sensitivity may be experienced as a result of bleaching. If so, discontinue the process for 2 or 3 days and suggest a less active approach for the patient. To date, there have been no reports of permanent pulp damage. However, there have been reports of mucosal irritation, usually mild and transient. The stronger

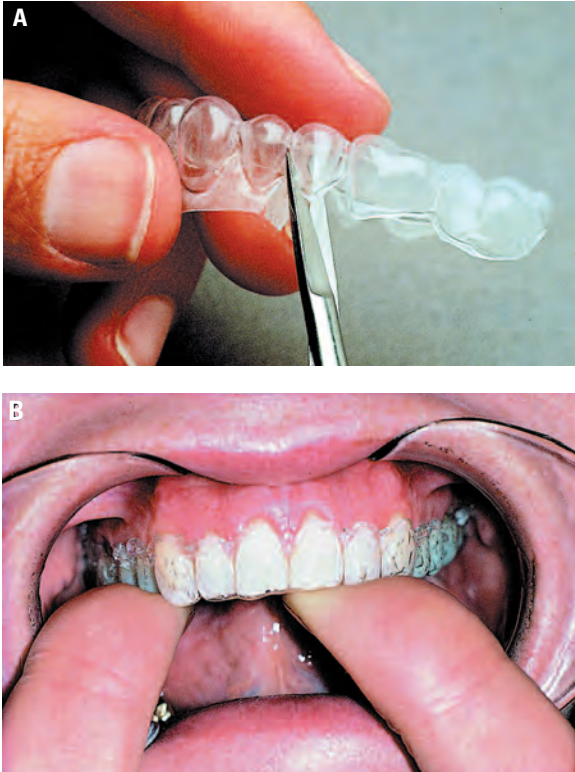


Figure 12-9. External bleaching. **A**, Soft plastic matrix of the patient's upper arch is trimmed to within 1 mm of the gingival margins. The guard can also be used at home by the patient, using 10% carbamide peroxide gel for longer periods of time. In the office the guard is loaded on the labial/buccal surfaces with 35% carbamide peroxide gel, Opalescence Tooth Whitening Gel (Ultradent Products, Inc.). **B**, The guard is then inserted into the patient's mouth for 30 minutes at a time. The guard can also be used at home by the patient, using 10% carbamide peroxide gel for longer periods of time.

(35%) carbamide peroxide is the product most likely to cause inflammation. There also have been reports that the peroxide may cause softening and cracking of composite resins. The patient must be warned that this may happen.

Home bleaching with drugstore products has become a fad with Americans. However, many patients are returning to their dentist for a more professional solution to what they perceive as a problem (Figure 12-10).



Figure 12-10. Professional result of external bleaching with mouthguards and carbamide peroxide gel. **A**, Before treatment. **B**, After two treatments. Discolored teeth that are not internally stained readily respond to external bleaching. Subsequently, the patient may maintain the color with an occasional repeat of the procedure with the 10% gel.

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