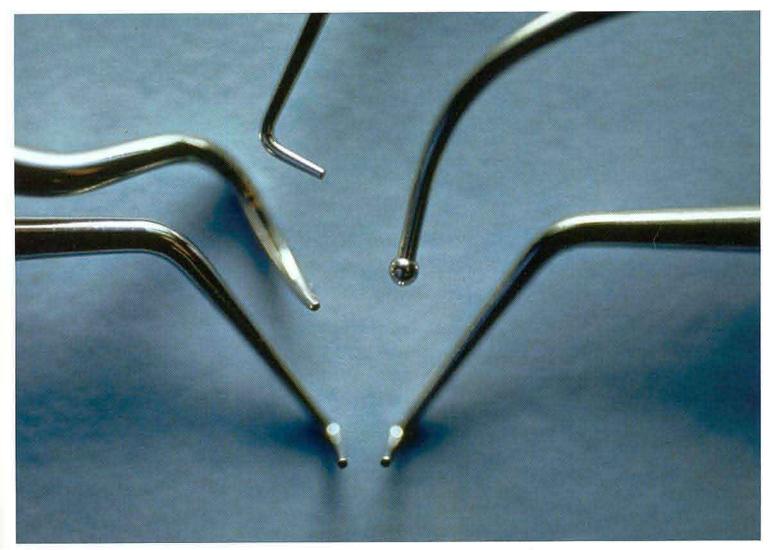
Nonsurgical Retreatment Therapy Success Contemporary Surgery

JOURNAL OF THE CALIFORNIA DENTAL ASSOCIATION VOL.32 NO.6

June 2004

ENDODONTICS



Mahmoud Torabinejad, DMD, MSD, PhD



DEPARTMENTS

- **442** The Associate Editor/Professional Hallmarks
- **445** Feedback/Readers Sound Off
- **447** Impressions/Teacher Motivates Dental Professionals with 'Real Life Story'
- **526** Dr. Bob/Fun With Gold Foil

FEATURES

456 NEW ADVANCES IN SCIENCE AND TECHNOLOGY OF ENDODONTICS

An introduction to the issue. Mahmoud Torabinejad, DMD, MSD, PhD

459 MICROBIOLOGIC ASPECTS OF ENDODONTIC INFECTIONS

An update on infections and treatment of endodontic disease. J. Craig Baumgartner, DDS, PhD

469 NEW ADVANCES IN THE MANAGEMENT OF ENDODONTIC PAIN EMERGENCIES

Exploring evidence-based approaches for managing endodontic pain emergencies and newly emerging pain management strategies based upon molecular, cellular and physiologic research into pain mechanisms. Kenneth Hargreaves, DDS, PhD, and Karl Keiser, DDS, MS

474 NONSURGICAL ENDODONTIC RETREATMENT

Identifying a variety of techniques to successfully retreat endodontically failing teeth. Clifford J. Ruddle, DDS

485 CONTEMPORARY ENDODONTIC SURGERY

With the introduction of enhanced magnification, periradicular ultrasonics and other associative technologies, teeth that might otherwise be extracted now have a chance for retention.

Richard Rubinstein, DDS, MS, and Mahmoud Torabinejad, DMD, MSD, PhD

493 THE SUCCESS OF ENDODONTIC THERAPY — HEALING AND FUNCTIONALITY

The most appropriate form of endodontic therapy should be attempted whenever feasible and is generally preferred over tooth extraction and replacement.

Shimon Friedman, DMD, and Chaim Mor, DMD

The Associate Editor

Professional Hallmarks

The end of all education should surely be service to others. — CESAR ESTRADA CHAVEZ



s I took a shortcut across the USC campus the other day, I discovered this plaque and quote. Being close to the school of dentistry, I paused for a moment to take in the

serene environment and ponder the words of Chavez. They seem particularly applicable to our profession because these two endeavors, education and service, are such integral components of dentistry. In fact, I firmly believe that by focusing our efforts in these two areas, we can overcome any challenges we face as individual dentists or as a united profession. Let's examine each and

"The end of all éducation should surely be service to others" "El próposito de toda educacion verdaderamente deberia servir a otros"

see what we're doing and what we could be doing to promote and improve our efforts.

When most of us think of dental education, we think of dental school. After all, it is where we all started our journey as dentists. I have previously shared comments on dental schools in this column. I would assert, however, that the most critical part of our education comes after we graduate. I heard Dr. Gordon Christensen, known and respected worldwide for his clinical skills and knowledge, say that when he graduated dental school, he was "just barely not dangerous." I've grappled with similar feelings. Many aspects of the dental procedures I perform on a daily basis were learned, not in dental school, but in continuing education courses and in practice through repeti-



tion. One such procedure that comes immediately to my mind is Class II composite restorations. A quick informal survey of my colleagues tells me I'm not alone. The road to placing excellent Class II composites has included mastering such techniques as rubber dam isolation, minimal preparations, bi-tine rings, sectional matrices, dentin bonding, sensitivity control with glass ionomer liners, flowable composite layers, incremental build-up techniques, directional and ramped curing, finishing and polishing. Several years ago, I was a master at none of these. I hated posterior composites so much I fell in love with them. The joy was in overcoming the challenge through education.

It is inspiring to see those who continue to further their dental training, not because they have to but because they love to. I hope I have as much passion about learning when I am as old as some of my colleagues and mentors who, even in retirement and even into their 80s, relentlessly pursue continuing education. Many may not be aware of CDA's efforts to be integrally involved in its members' lifelong education. Most visible is Scientific Sessions. Many of us have participated in these bi-annual events and are aware of the quality of speakers and programs that are offered. But CDA is also developing online learning and other avenues to promote education. CDA is continually making efforts to provide quality, reliable and relevant continuing education to its members.

What about service? I think of service as being divided into three categories: service to patients, service to the public, and service to the profession. By nature, dentistry is a service profession. We serve patients everyday. How and how well we serve them varies widely among individual dentists and is often the subject of debate. Much of the profession's service to the public is often performed by individual dentists with little or no public fanfare. These individuals form the grassroots of the profession's public service. Some examples of these efforts are dentists who participate in school screenings, partake in school educational talks, volunteer at small community clinics or travel to remote areas both in and outside of the country providing much needed dental care.

There is another arm of public service that occurs on a grander scale often with a higher degree of public visibility. These efforts are often carried out by concerted efforts of the tripartite of organized dentistry. The Give Kids a Smile program is one example, and the CDA Foundation is another. The former has received much press, I would like to briefly mention the latter. In a report to the Board of Trustees in February, CDA Foundation Chairman, Brian Scott, DDS, outlined no less than 20 strategic initiatives for 2004/2005 and six long-range goals and objectives through 2012! I'm sure staff would be happy to provide any member with details of these activities but suffice to say that your foundation is working hard to serve the public on every member's behalf.

Many of us are aware of countless individuals who serve the profession at many levels. If there is any doubt that dentists give back to their profession, one need look no further than a CDA or ADA House of Delegates meeting, take a roll call of the volunteer faculty at our dental schools, or attend a meeting of the International Association of Dental Research. The sum total of these efforts is a dental profession that continues to be strong in its leadership, that continues to effectively train tomorrow's professionals, and continues to discover new and better materials and techniques to treat our patients.

So may we rest on our current laurels in the areas of education and service? What type of grade would we receive in these two areas? I think Cesar Chavez would probably give us a B+. We certainly make noble efforts in these areas and outshine many other professions with our endeavors. But we can always do better. Dentistry is still pervaded by individuals who seek only minimal continuing education, and is even tainted by those who use dishonesty to obtain these minimal requirements. Several years ago, a program called QUIL3 (Quality Improvement through Life-long Learning) met with sharp criticism and unfortunately was not resoundingly accepted by the profession. Furthermore, the percentage of dentists who voluntarily serve the public and the profession are, by all estimations, a scarce minority. And much of the good that is done is undone by those who place their own financial interests and goals above the best interests of the patient, doing a disservice to everyone but themselves.

Perhaps our professional future will be enhanced by refocusing on the value of education and service. Perhaps we will have a new wave of leaders who promote and foster an environment that allows service and education to flourish far beyond current levels. And perhaps one day dentistry will be a showcase of individuals who not only believe that education and service are hallmarks of the profession but who, like Cesar Chavez, embody these virtues through their words and their actions. Much of the profession's service to the public is often performed by individual dentists with little or no public fanfare.

Readers Sound Off

To ignore or minimize this issue will erode our noble profession back into a trade.

Standard of Care?

In a recent article published in a dental journal (name not important), the author expounded on dental implants being the current standard of care.

I was both shocked and appalled at the idea of dental implants moving so quickly up the scale to standard of care.

Dental organizations throughout the U.S. must take great umbrage at a small segment, not representing the cross-section of dentistry, taking that issue to an undeserved position, a position backed primarily by the dental products industry.

Carefully analyzing this phenomenon should quickly raise concerns not the least of which is intimidation of general dentists to use/recommend implant therapy, to take fixed replacements (crown and bridge) out of his/her hands in deference to referrals for implants.

The legal implication of taking implants to that standard is frightening, costly and intimidating. Maybe that's the approach the dental products industry thinks will expand their business.

If we allow this escalation to occur unchallenged, we will soon be a trade dominated by manufacturers, third-party payers and other special interest groups. A frightening prospect.

Maybe you can spearhead a campaign to interpret this progression, to take control of our profession, expand your leadership muscle.

Theodore T. Fortier, DDS, FACD, PA, FADI

Assessing 'Real Science'

The paper Assessing 'Real Science': Poor Studies, Industry Ties Taking Toll is the most lucid, incisive essay describing the collective ethical "toothache" that has been troubling dentistry (and allied health professions) for quite some time; lately it's becoming more evident.

Ms. Janyce Hamilton is a gifted writer with the capacity to take a complex, multi-faceted subject and report it to our profession as a compelling cautionary tale. Every member of our profession should become conversant with this issue so we can take more preventative measures (described by Ms. Hamilton) to reduce the influence of money and favors on our hard, basic and clinical science. To ignore or minimize this issue will erode our noble profession back into a trade and betray the trust that was placed in us when we became doctors of dental medicine.

Stephen Cohen, DDS









Teacher Motivates Dental Professionals with 'Real Life' Story

by Debra Belt

hat do teachers and dental professionals have in common? Quite a bit, according to Erin Gruwell, who published *The Freedom Writers Diary* along with 150 high school students and became one of America's most famous teachers.

Gruwell recently addressed nearly 900 dental professionals at a dental team breakfast during Spring Scientific Session in Anaheim and emphasized the "power of one."

"As dental professionals, you have the power to touch, motivate and inspire the person in your chair," Gruwell said. "You can make people feel safe and can make them smile. You can make a difference with a simple question about how someone is feeling or how his day is going."

Even though dentists know this, they



enjoyed the reminder.

"I was encouraged by the teacher," said Michael De La Cruz, DMD, of Moreno Valley as he waited in line to meet Gruwell. "As a general dentist, I always talk to my patients, especially the students. I emphasize the priority of education."

De La Cruz also said that Gruwell's real life story caused him to "shed a tear."

It's fair to say that the entire audience was moved by Gruwell's testimony. She received three standing ovations.

James Pasternak, DDS, of San Fernando Valley said he was touched by Gruwell's presentation.

"It reminded me that as a dentist, I can make a patient's visit a nurturing experience," he said. "You never know when you can make a difference in someone's life."

Because the presentation was created for the entire dental team, the strength of Gruwell's message was multiplied as hygienists, dental assistants and office staff enjoyed a renewed vision of the potential each person carries with them every working day.

"Her message hit close to home," said Julie Daigh Pryor, RDH, who got up before dawn to drive from Ridgecrest to attend the breakfast. "I'm especially open to teenagers. I recently had a young patient and I could tell he was having a rough day. Once I started talking to him, he opened up. When he left, he left with a smile."

Throughout the course of the morning, Gruwell unfolded the story of *The Freedom Writers Diary*, the chronicle of how a newly minted teacher reached out to her "unreachable" students and motivated them to respond humanely and creatively, through writing, to the injustice and violence they experience in their lives.

The breakfast began with a screening of *Prime Time Live* with Connie Chung featuring Gruwell and her students at Wilson High School in Long Beach.

Gruwell followed the video with her personal account of being a "preppy" student teacher faced with a group of students

ith the motto,

"You cannot undo

the past, but you can

rewrite your destiny,"

she encouraged her

students to begin

writing their

own diaries.

the school's administration had termed "too stupid to make it."

She talked about the racial tension in Southern California following the Rodney King verdict, about her students who witnessed countless shootings, killings and incarcerations and about students who had never heard of the Holocaust.

The topic of the Holocaust came up in her classroom after Gruwell intercepted a note with a

crude racial caricature of a student.

"This is the type of propaganda the Nazis used during the Holocaust," she angrily told her students. She was met with blank stares.

When asked how many had heard of the Holocaust, not one student raised a hand. When Gruwell asked how many students had been shot at, nearly every student in the room raised a hand.

"If you don't learn from history, you're doomed to repeat it," she told her students. She decided to toss her meticulously planned lessons and opted for a curriculum focusing on tolerance.

She selected books such as *Anne Frank: The Diary of a Young Girl,* and *Zlata's Diary: A Child's Life in Sarajevo*; books about young people faced with violent situations. Gruwell did not have a budget for the books, so she moonlighted as a concierge

448 CDA.JOURNAL.VOL.32.NO.6.JUNE.2004

and worked in a department store. With the motto, "You cannot undo the past, but you can rewrite your destiny," she encouraged her students to begin writing their own diaries.

The students related the books to their own lives and dubbed themselves "The Freedom Writers" in homage to the civil rights activists "The Freedom Riders." Doubleday published *The Freedom Writers Diary* in 1999. In addition to appearing on *Prime Time Live*, The Freedom Writers were featured on National Public Radio's *All Things Considered*, *The Oprah Winfrey Show* and ABC's *Good Morning America*.

Throughout her presentation in Anaheim, Gruwell referred to adults who were an inspiration when she was a child, and one of her mentors was her dentist.

"My dentist would talk to me and tell jokes. He would tell me that I would grow into my very large teeth, and he made me feel safe," said Gruwell. Gruwell said that she often reminisced about adults who impressed her as a child and thought about what they had done, so she could do the same for her students.

"I thought about my dentist and how he talked to me so I was not afraid and how he had a treasure chest of things to choose from," she said. "I knew my students needed someone to help them feel safe, and thought maybe they would like a treasure chest."

After the presentation, Gruwell was asked the name of the dentist who left an impression on her life.

"I asked him if I could mention his name in my presentation to CDA, and he declined," she said. "He said he wanted to remain anonymous so all dentists could feel as if it had been him or her who had made a difference."

CDA thanks Essential Dental Systems for sponsoring the dental team breakfast at Spring Session. thought about my dentist and how he talked to me so I was not afraid and how he had a treasure chest of things to choose from."

Tips on Collecting Debts

One of the crucial factors in maintaining an effectively run dental practice is receiving timely payments said consultant Tom Stenklyft in the January issue of Today's FDA, the Florida Dental Association's newsletter. Five important factors small businesses should think about when trying to collect on past due accounts:

n The older a debt, the less inclined a debtor is to pay. The value of a service diminishes over time and chances of getting paid decrease also. Promptly addressing missed or late payments is good business.

n Lackadaisical collecting habits does not indicate compassion but may be a sign to the debtor that your willingness to let bills slide possibly suggests your profit margin is so high you don't collect on debts.

n Debtors typically pay creditors for urgent bills like utilities or secured loan lenders. Collection activities should convey

a sense of urgency.

n Using both the telephone and the mail can be the best way to make sure your collection requests are being heard.

How to Build a Web Site for Your Practice



Web sites are being utilized by more and more dental practices to supplement their marketing programs. In the December 2003 issue of the *Missouri Dental Association* magazine, practice consultant Mary Byers suggests that dentists pondering the idea of developing a Web site should ask themselves a few essential questions:

n What do I want my site to accomplish?

 $\ n$ What information do I want on my site?

n How often do I wish to update the information on the site?

n What is my budget for the design and setup of a site?

Eating Disorders Have Significant Effect on Oral Health

Bulimia nervosa, binge-eating and anorexia nervosa pose serious concerns to women as well as clinical challenges to dental professionals.

According to authors Drs. Bruce Glazer, Peter Gold and Wayne Wolfstadt in the November 2003 issue of Oral Health, the dental team should be aware that those suffering from eating disorders may return to their negative eating habits.

Individuals suffering from these disorders have a higher caries rate than those in a reference group. In a recent study, one group had erosive tooth wear involving dentine, and an estimated one-third have very low un-stimulated salivary flow rates with very high counts or streptococci mutans and lactobacilli. The degree of erosive tooth wear correlated with the number of years of binge eating.

From an ethical and legal standpoint, knowledge of the symptoms and clinical oral signs of bulimia is important since early diagnosis and treatment are directly related to successful dental care. Dental patients who

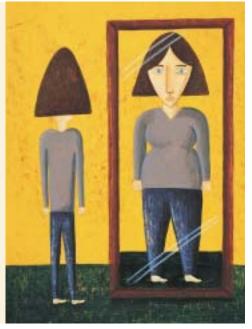
 $\ensuremath{\mathtt{n}}$ What is the cost of having the site hosted?

n Do I want to invest the time to oversee the site, or do I have a staff member able to assume this responsibility?

One critical step in establishing a Web presence is to take the time to surf the Internet and look at other dental practice sites, taking note of what you like and dislike, Byers said.

After figuring what it is that appeals to you about other dental office sites, you now must decide if you'll design the site yourself or use a professional. Not computer savvy? Byers said consider hiring a pro. While it may require an initial outlay, it likely ensures your site is well designed and easy to navigate. Byers also suggested determining whether you'll update the site yourself or rely on a designer for this. If opting for a designer, discuss this matter upfront and determine how you'll be charged.

After developing a site, Byers said it's important to periodically change it and add to it. Continue visiting other dental Web sites and stay updated on their sites. It's also a good idea to note what kind of information your patients request.



TAKE THE TIME TO SURF THE INTERNET AND LOOK AT OTHER DENTAL PRACTICE SITES, TAKING NOTE OF WHAT YOU LIKE AND DISLIKE.

Talking to Staff About Their Job Performance

One of the most difficult moments for dentists in private practice is to communicate to their employees on the subject of staff performance.

To make the inevitable easier, it is suggested dentists consider implementing a human resource system that includes a job description and a job evaluation, said Brian Pollard in the November 2003 issue of The College Connection, the College of Dental Surgeons of Saskatchewan's newsletter.

According to Pollard, every staff person should have a job description that was discussed with them upon hiring. An employer should periodically ensure employees agree with the duties outlined in their job descriptions. It also should be made clear to staffers that periodic evaluations, based on job descriptions, will occur in the future.

Fair job evaluations include two-way communication, giving employees the opportunity to discuss their concerns as well

as permitting them to tell their employers what they need to be successful, such as additional training, in their jobs, Pollard said.

Implemented properly, job descriptions and formal job evaluations can be important tools, providing employers opportunity to reward excellence at work as well as helping the dental staff improve its performance, Pollard said.

Fair job evaluations include two-way communication, giving employees the opportunity to discuss their concerns.

Eliminate Soda in Schools Pediatrics Academy Suggests

To safeguard against health problems that resulting from over-consumption of sweets by school-age children, the American Academy of Pediatrics has suggested that school districts restrict selling soft drinks. The association also stated pediatricians should educate patients, their parents and school authorities regarding the health consequences of soda consumption. Water, low-fat white and flavored milk, vegetable and real fruit juices are healthful alternatives, the association said.

Soft drink and other sugary beverage consumption has been associated with an increased risk of overweight and obesity, currently the most common medical condition of childhood. The association statement noted, "Additional health problems associated with high intake of sweetened drinks are dental cavities and potential enamel erosion."

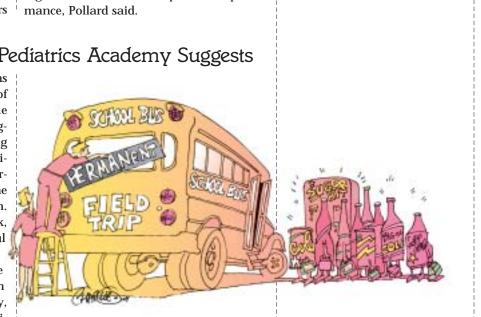
In recommending pediatricians work to eliminate sweetened soft drinks in schools, the policy also suggests that:

n Pediatricians advocate the creation of a school nutrition advisory council as a means of ensuring the health and nutritional interests of students form the foundation of nutritional policies in schools.

n Prior to making decisions to sign a vended food or drink contract, school districts should invite and encourage public discussion.

n Should a soft drink contract already be in place, it should be adapted so that it does not promote student over-consumption.

n Eliminate the advertising and prohibit the consumption of sweetened soft drinks within the classroom.





Studies Further Heart and Oral Health Link

A study of heart disease in women and another on serological factors related to periodontitis provide more evidence linking poor oral health with heart disease.

> Karolinska Institutet investigators first compared the oral health of 187 female patients with coronary heart disease with a control group in order to determine the connection between dental health and cardiovascular disease, according to an

article in ADA's *News Today.* Results showed heart patients had fewer remaining teeth and a higher rate of pathological periodontal pockets. Compared to the control group, more wore dentures and were apt to be edentulous.

Another study looked at plasma level markers for arteriosclerosis and indices of inflammation in 50 patients having severe periodontitis with those in 46 healthy subjects.

Although total cholesterol levels in the two groups were about the same, C-reactive protein levels were higher and high-density lipoproteins (HDL) levels were lower in the test patient group. Both low HDL and high C-reactive protein levels are known to be arteriosclerosis risk factors.

Investigators believe the release of bacteria, bacterial products or pro-inflammatory cytokines from the periodontal lesions into the blood stream may be the link between heart disease and periodontitis.

Upcoming Meetings 2004

June 24-26	ADA 18th annual New Dentist Conference, San Diego, (312) 440-2779, www.ada.org/goto/newdentconf			
Sept. 8-11	International Federation of Endodontic Association's sixth Endodontic World Congress, Brisbane, Queensland, Australia, www.ifea2004.im.com.au.			
Sept. 10-12	CDA Fall Scientific Session, San Francisco, (866) CDA-MEMBER (232-6362).			
Sept. 30-Oct. 3	ADA Annual Session, Orlando, Fla., (312) 440-2500.			
Nov. 7-13	U.S. Dental Tennis Association Annual Meeting, Palm Desert, (800) 445-2524, www.dentaltennis.org			
2005				
April 6-9	Academy of Laser Dentistry 12th annual Conference and Exhibition, New Orleans, (954) 346-3776.			
April 12-16	International Dental Show, Cologne, Germany, www.koelnmesse.de			
To have an event included on this list of parametic acceptation meetings, places cand the information				

To have an event included on this list of nonprofit association meetings, please send the information to Upcoming Meetings, *CDA Journal*, P.O. Box 13749, Sacramento, CA 95853 or fax the information to (916) 554-5962.

CORRECTION

The article "What is This Red Mark?" was reprinted from the January 2004 issue of the Sacramento District Dental Society's Nugget. The article "Do I Make the Call?" was reprinted from the Nugget's November 2001 issue. Both articles were authored by Kathleen Shanel-Hogan, DDS, MS, and appeared in the April Journal.



New Advances in Science and Technology of Endodontics

Mahmoud Torabinejad, DMD, MSD, PhD

ew advances in health care have changed the lives of millions of individuals around the globe. As a result, people in many parts of the world are living longer and

are enjoying a better quality of life. Dentistry, like medicine, has contributed significantly to this improvement. Two of the main objectives of dentistry have been the prevention of oral disease and the preservation of natural dentition. For decades, we have been fighting decay and periodontal disease, and restoring function and aesthetics to teeth affected by these maladies.

Root canal therapy has been a major contributing factor to the success of the dental profession. Many advances have occurred in endodontics within the last 10 years. These include new information in basic and clinical sciences, management of pain and anxiety, clinical practice, new techniques and instruments, retreatment, management of mishaps and endodontic surgery.

Studies have shown that pulpal and/or periradicular pathosis does not develop without the presence of bacteria. Depending on the stage of pulpal pathosis, various species of bacteria can be cultured from infected root canals. These bacteria are predominantly gramnegative anaerobes and they can infect the root canals by direct pulp exposures (caries or traumatic injuries) or by coronal micro-leakage. Because bacteria plays an important role in the development of pulpal and periradicular diseases, and the success of root canal treatment depends on the absence of bacterial contamination following root canal therapy, the main objectives of root canal therapy are cleaning and shaping, obturating the root canal system in three dimensions and preventing reinfection.

To effectively diagnose and treat pulpal and periradicular diseases, the clinician must be familiar with the new information on the nature and complexity of root canal infections.

New technological advances in the field of microbiology have allowed researchers in the field of endodontics to identify new species of bacteria and to develop a more elaborate description of microbial diversity. Histological and immunological studies of inflamed pulp and periradicular lesions have shown the presence of numerous immune competent cells capable of defending these tissues. The repair potential of the dental pulp is related to the presence of these cells. However, interaction of the irritants and the immune cells can also cause destruction of these tissues. Proper diag-



Author / Mahmoud Torabinejad, DMD, MSD, PhD, is a professor of endodontics and director of Graduate Endodontics at Loma Linda University School of Dentistry, and practices in Upland, Calif. He has coauthored two textbooks in nonsurgical and surgical

endodontics as well as numerous articles on endodontics and dental topics. He was certified as a diplomate of the American Board of Endodontists and taught at Harvard before joining Loma Linda University. He has received several awards including Ralph F. Somers, Louis I. Grossman, and the Philanthropist award of the AAE Foundation.

TO EFFECTIVELY DIAGNOSE AND TREAT PULPAL AND PERIRADICULAR DISEASES, THE CLINICIAN MUST BE FAMILIAR WITH THE NEW INFORMATION ON THE NATURE AND COMPLEXITY OF ROOT CANAL INFECTIONS.

nosis of pulpal and periradicular disease is the most important aspect of modern endodontic treatment. Cold testing with tetraflouroethaneethyl chloride or CO_2 snow is a relatively reliable method to assess the responsiveness of the pulp to cold substances. This testing is more reliable than using heat or electrical testing of the dental pulp. Considerable advances have been made in the management of pain, infection and anxiety in patients with pulpal and periradicular diseases.

The use of Nickel titanium (NiTi) files during cleaning and shaping of root canals has changed this procedure significantly. The flexibility of this metal allows practitioners to use rotary files during instrumentation. Many NiTi files with various tapers have been designed to shape root canals. Because of the flexibility of and presence of non-cutting tips in these files, the curvature of canals can be more easily maintained compared to stainless-steel files. Inappropriate use of these files can result in perforations and instrument locking, and separation.

New intra-canal medications are on the horizon to disinfect root canals. The use of gutta-percha in conjunction with a root canal sealer is still the method of choice for obturation of the root canal system. The main methods of obturation of root canals are lateral and vertical condensation techniques. There is no clinical study showing superiority of one technique over another. Mineral Trioxide Aggregate (MTA) has become very popular in plugging the apical third of roots with open apexes. The lack of a coronal seal has been recently identified as an important factor in the development of periradicular lesions following root canal treatment. Therefore, placement of a coronal seal following root canal treatment is critical in the outcomes of this procedure.

The introduction of the microscope to the field of endodontics has provided the clinician an opportunity to observe areas of interest at high magnification under constant illumination. The use of this device during root canal treatment can assist the clinician in locating and negotiating calcified canals, and performing surgical and nonsurgical root canal treatments. Endoscopes, orascopes and magnifying loupes with or without a supplemental light source are also useful tools to enhance visualization of the operative field. Ultrasonic devices are used to remove posts and separated instruments from the root canal system. These tools are also used to prepare a root-end cavity preparation during surgical root canal treatment. Determination of working length during cleaning and shaping using an apex locator is more accurate and is associated with less health hazard compared to the use of radiographs in endodontic patients. The introduction of new devices and techniques into the field of endodontics has significantly improved the success rate of retreatment. Root perforation during root canal treatment reduces the success rate of this procedure; the use of MTA to repair root perforations has significantly improved the

success of root canal treatment.

Nonsurgical root canal treatment is a highly successful procedure if practitioners are thorough with their diagnoses and proficient in performing their clinical treatments. The common belief that unresolved periradicular lesions should be corrected surgically is not always true. Although surgery is necessary in some situations to retain a tooth that would otherwise be extracted, retreatment can correct most persistent endodontic pathosis. Lesions of endodontic origin that have not healed can have a second chance with recent improvements in endodontic surgical instruments, materials and techniques. Current, relevant knowledge on the outcome of endodontic treatment is key to clinical decisionmaking, particularly when endodontic treatment is weighed against tooth extraction and replacement.

The purpose of this issue is to share new advances in some areas of endodontics. These areas are microbiological aspects of endodontic infection, management of endodontic emergencies, retreatment, endodontic surgery and the data on the success of endodontic treatment. I believe that the use of these new concepts and adherence to the principles of proper root canal treatment will result in the retention and rehabilitation of millions of natural teeth, as well as the satisfaction of patients throughout the world. Our goal continues to be that of working with all disciplines of dentistry promoting the best in oral health CDA care for our patients.



MICROBIOLOGIC ASPECTS OF ENDODONTIC INFECTIONS

J. Craig Baumgartner DDS, PhD

ABSTRACT

for

Our understanding of endodontic infections and treatment of endodontic disease has increased significantly over the last decade. This article is an update of those findings. Aspects that are reviewed include: portal of entry

microorganisms, virulence and pathogenicity of organisms, descriptions of primary and recurrent endodontic infections, and treatment of endodontic infections. irtually all endodontic disease (pulpal and periradicular) are either directly or indirectly related to the presence of microorganisms. To effectively diagnose and treat endodontic

ausease, clinicians must be familiar with the nature of infections of the root canal system and periradicular tissues. This fundamental relationship of microorganisms and endodontic disease has been established. Antony van Leeuwenhoek, the father of microscopy, described microorganisms observed from an infected root canal.¹ When he removed material from the hollow part of a tooth and examined it with his microscope, Leeuwenhoek saw "cavorting beasties."

It then took about 200 years for W.D. Miller, who was working in Robert Koch's laboratory in 1890, to make the correlation between microorganisms and pulpal/periapical disease.^{2,3} Using a microscope, Miller observed in teeth with exposed pulp chambers that the bacteria in the pulp chamber were different from those in the root canals. In addition, from culture experiments, Miller noted that only a few of the observed species of bacteria were cultivable.^{2,3} Even today many of the species remain uncultivable.⁴⁻⁷

In 1965, Kakehashi et al. showed that bacteria are etiological agents of both pulpal infection and the development of periapical lesions.⁸ Following mechanical pulp exposure, pulpal disease and periapical lesion formation occurred only in conventional rats with normal microflora but not in germ-free rats. In fact, in the absence of bacteria, the pulps of the germ-free rats formed bridges of reparative dentin demonstrating the potential for pulpal repair.⁸

Portal of Entry for Microorganisms

The most common portal of entrance for microorganisms to the pulp cavity is dental caries. In addition, microbes may find their way into the pulp cavity via mechanical or traumatic exposure and via exposed lateral/furcation canals or even exposed noncarious dentinal tubules. Once the pulp is necrotic, dentinal tubules become dead tracts that microorganisms can traverse with impunity. Passage of microbes through exposed dentinal tubules is likely the pathway when the pulp becomes necrotic following a traumatic



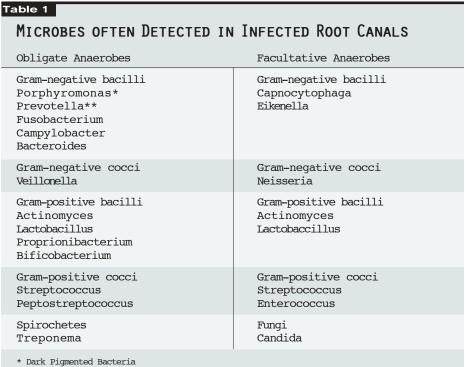
Author / J. Craig Baumgartner, DDS, PhD, is a professor, chairman of the Department of Endodontology and director of the Advanced Education Program in Endodontics at the School of Dentistry, Oregon Health & Science University. He is a diplo-

mate of the American Board of Endodontics and currently director of the American Board of Endodontics. He previously was director of the Advanced Education Programs in Endodontics at the U.S. Army Institute of Dental Research and Walter Reed Army Medical Center. Additionally, he is an associate editor of the Journal of Endodontics and on the editorial board of Oral Surgery, Oral Medicine, Oral Pathology, Oral Radiology. He maintains a clinical practice in the specialty of endodontics one day per week. injury to a tooth. Microorganisms may gain access to the pulp cavity via cracks in the enamel-dentin and periodontal exposure (treatment/disease) of dentinal tubules and accessory canals. Although it has been demonstrated in animals, a less likely route is via anachoresis in which microorganisms are transported in the blood to an area of inflammation where they establish an infection.⁹⁻¹²

Microbial Virulence and Pathogenicity

The dental pulp and periradicular tissues are normally sterile tissues. An endodontic infection results when microbes invade and multiply in the pulp periradicular cavity or tissues. Pathogenicity is the ability of microbes to produce disease while virulence is the degree of pathogenicity. Endodontic disease includes the response of these tissues to the microbes and their virulence factors. Virulence factors include capsules, pili (fimbriae) lipopolysaccharides (endotoxin), enzymes (collagenase, hyaluronidase, chondroitin sulfatase, proteases), extracellular vesicles and by-products such as polyamines, indole, hydrogen sulfide, methyl mercaptan, ammonia, butyrate and other organic acids.13-21 Virulence factors may vary from strain to strain even within the same species. The number of virulence factors relates to the degree of pathogenicity.

With few exceptions, studies that cultured the contents of infected pulp cavities and periradicular abscesses have shown them to be polymicrobial with 3-12 isolates. The majority of microbial isolates are a subset of organisms isolated from periodontal tissues which in turn are a subset of 400 or more species of bacteria in the oral cavity.^{5,22-25} Before the 1970s, very few strains of strict anaerobes were cultivated. Improved techniques for anaerobic culturing demonstrated that the vast majority of cultivable bacteria in root canal infections are anaerobic.²⁶⁻³⁰



** Dark Pigmented Bacteria and Non-pigmenting Bacteria

Primary Endodontic Infections

The dynamics of the ecosystem in infected root canals has been studied in monkeys.³¹⁻³³ Following the introduction of the monkey's indigenous oral flora into their canals, the canals were sealed but sampled over a period of 1,080 days.³² Although facultative bacteria predominate early in root canal infections, bacteria that are strict anaerobes displace them. After 1,080 days, 98 percent of the cultivable bacteria were strict anaerobes. A root canal containing a necrotic pulp becomes a selective habitat that allows some species of bacteria to grow in preference to others. The nutrients provided by the breakdown products of a necrotic pulp, tissue fluid, and serum from surrounding tissues along with low oxygen tension, and bacterial by-products support the growth of selected microorganisms (Table 1). The coronal portion of a root canal may harbor organisms different from those in the

apical portion including different strains of the same species.³⁴ When root canal walls were observed with a scanning electron microscope, patterns of colonization were not uniform even in the same specimen³⁵ (Figure 1). Clumps of bacteria were seen that appeared to be self-aggregating while other clumps had different morphologic types and appeared to be co-aggregating.35 Statistical odds ratios have been used to show a relationship between certain combinations of organisms in endodontic infections but the clinical significance is unknown.^{6,7,36-41} The polymerase chain reaction (PCR), a molecular method, has been used to show that geographical differences exist among endodontic infections.²² When PCR was used to detect microbes in endodontic abscess samples from patients either in Portland. Ore., or in Rio de Janeiro, Brazil, there was a significant difference in 5 of the 8 organisms tested.²²

Endodontic Infections

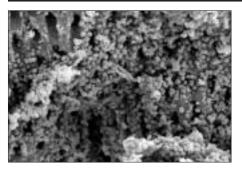


Figure 1. This scanning electron microscopic photo shows both cocci and bacilli on the surface of dentin and extending into the dentinal tubules. *(Courtesy of José Siqueira.)*

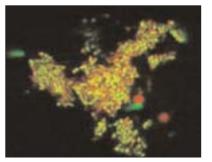


Figure 2. This confocal microscopic photo shows coaggregation of *Actinomyces* and *Eubacterium. (Courtesy of Saengusa Kheemaleelakul.)*

Table 2

NOMENCLATURE FOR PREVIOUS "BACTEROIDES" SPECIES

Porphyromonas - Dark-pigmented (asaccharolytic Bacteroides species)

- Porphyromonas asaccharolyticus (usually non-oral)
- Porphyromonas gingivalis*
- Porphyromonas endodontalis*

Prevotella - Dark-pigmented (saccharolytic Bacteroides species)

- Prevotella melaninogenica
- Prevotella denticola
- Prevotella loescheii
- Prevotella intermedia
- Prevotella nigrescens*
- Prevotella corporis
- Prevotella tannerae

Prevotella - Nonpigmented (saccharolytic Bacteroides species)

- Prevotella buccae*
- Prevotella bivia
- Prevotella oralis
- Prevotella oris
- Prevotella oulorum
- Prevotella ruminicola

* Most commonly isolated species of black-pigmented bacteria

Coaggregation of different species of bacteria or self aggregation of the same species may provide the organisms with protection from the host's defenses and provide nutrients from the surrounding bacteria^{19,42-46} (**Figure 2**). The combination of *Fusobacterium nucleatum* with dark-pigmented bacteria *Prevotella intermedia* and *Porphyromonas gingivalis* has been shown to be more virulent than when the bacteria are in pure culture.⁴⁷ This supports the concept that there is a synergistic relationship between bacteria in an endodontic infection. Numerous bacteria have been associated with various clinical signs and symptoms but no absolute correlation has been made.^{6,29,36,48-56}

Gram-negative bacteria, especially dark (black) pigmented bacteria, have received a great deal of attention. Depending on the species of dark-pigmented bacteria and the culture media, the color of the colonies may vary from

tan to black. Because of numerous taxonomic changes for dark-pigmented bacteria based on DNA studies, it is difficult to relate older studies to contemporary studies. Table 2 shows the current nomenclature of the 10 species of darkpigmented bacteria found in humans. For example, the species Porphyromonas endodontalis was separated from P. gingivalis and Prevotella nigrescens was separated from P. intermedia.6,57-59 The virulence potential of dark-pigmented bacteria has been studied in animals. Strains of cultivable bacteria have been shown to have the capability to resist phagocytosis, degrade immunoglobulins, and increase pathogenesis when in combination with other specific strains of bacteria (e.g. P. gingivalis and Fusobacterium nucleatum).^{13,14,47,60} Pathogenicity may be related to the presence of lipopolysaccharide (LPS) on the outer membrane of gram-negative bacteria. LPS has been demonstrated in root canals and periradicular tissues and related to the severity of disease.^{16,18,61-63} LPS (endotoxin) is released from gram-negative bacteria during multiplication and cell death. Once released bacterial endotoxin causes a series of biological effects, which produce inflammation and periapical bone resorption.^{64,65} The cytotoxic lipid A moiety in LPS is hydrolyzed by alkaline chemicals especially calcium hvdroxide.64-66

Gram-positive bacteria that are differentiated from gram-negative bacteria by a relative thick layer of peptidoglycan have also been associated with endodontic disease.^{6,29,36,48-56} Actinomyces may colonize periapical tissues. Aggregates of organisms may be recognized in periradicular biopsy sections as sulfur granules.^{52,67-69} Because some of the granules are yellow colored, older literature refer to them as sulfur granules.⁶⁷ The species A. israelli is often associated with cervical-facial actinomycosis. The cell surface of A. israelli has long pili believed to participate in attachment to other species of bacteria and other sur-

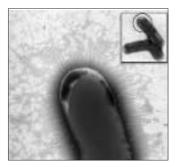


Figure 3. An electron microscopic view of hair-like fimbriae on the surface of *Actinomyces israelii*. Reproduced with permission from the Australian Dental Association, Figdor D, Davies J. Cell surface structures of *Actinomyces israelii*. *Australian Dent J* 42(2):125-8, 1997.

faces^{43,70} (Figure 3). A recent study using the PCR detected species of Actinomyces (israelii, naeslundii, viscosus) in 80 percent of the infected root canals and 46 percent of periradicular abscesses.71 The species A. israelli was detected in root canals 27 percent of the time and in periradicular abscesses 26 percent of the time.⁷¹ This suggests that our clinical methods of root canal debridement and surgical endodontics are successful even when Actinomyces is present. While cervical-facial actinomycosis may require long-term antibiotics, studies have shown that antibiotics may not be needed for endodontic infections with Actinomyces.72,73 If antibiotics are indicated because of continued clinical signs and symptoms, a short-term regimen should be adequate.^{72,73}

Other gram-positive bacteria often cultured from endodontic infections include *Peptostreptococci, Streptococcus, Enterococcus,* and *Eubacterium.*^{37,51,74,75} Using PCR, *Peptostreptococci micros* was detected in 28 percent of the infected root canals tested.⁷⁵

Spirochetes (treponemes) have been difficult to isolate in pure culture from endodontic polymicrobial infections. Recently, DNA hybridization and PCR have been used to screen for spirochetes.^{7,40,41,76-78} Using PCR, it was determined that 60 percent of samples from

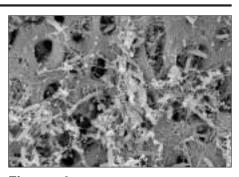


Figure 4. This scanning electron microscopic photo shows the surface of a root canal with cocci and bacilli on the surface of necrotic predentin. Some organisms are in the predentin openings to the dentinal tubules. The predentin openings were previously occupied by the cell bodies of the odontoblasts. *(Courtesy of José Siqueira.)*

abscesses/cellulitis contained spirochetes and 37 percent of asymptomatic teeth with periradicular lesions contained spirochetes.⁷ The spirochetes most commonly detected were *Treponema socranskii* (45 percent) followed by *T. maltophilum* (30 percent), *T. denticola* (29 percent), *T. pectinovorum* (14 percent), and *T. vincentii* (5 percent).⁷

Fungi have been cultivated and detected using molecular methods in infected root canal.⁷⁹⁻⁸³ When using PCR, *Candida albicans* was detected in 21 percent of infected root canals but not in any aspirates of purulence from endodontic abscesses.⁸³ Electron microscopy has detected fungi in infected root canals but not in periapical tissues.^{84,85}

Viruses may also be associated with endodontic disease. Bacteriocins are viruses that infect bacteria and carry DNA into the genome of the bacteria.^{86,87} The human immunodeficiency virus has been detected in the dental pulp and the periapex.^{151,152} Both the cytomegalovirus and Epstein-Barr virus have been associated with symptomatic endodontic infections.^{86,87} Future studies will likely show more relationships between viruses and endodontic disease.

Molecular methods such as DNA hybridization and PCR detect and identify many more microorganisms than the so-called "gold standard" of culturing. Molecular methods are much more sensitive and offer precise identification at the DNA level. A disadvantage of molecular methods is not knowing if the DNA was from an alive or dead organism. Currently, antibiotic susceptibility tests cannot be accomplished without viable organisms. In the future, probes may be used to identify antimicrobial resistant genes in the DNA samples without having to grow the organisms for susceptibility tests.

Periradicular Endodontic Infections

Once a necrotic pulp is infected, the root canal system becomes a sanctuary for microbes (Figure 4). They often seem to be contained by a layer of neutrophils or an epithelial plug at the apical foramen.⁸⁵ Plaque-like biofilms have been observed in the foramen and root-end of an infected canal.⁸⁸ Extraradicular bacteria are usually associated with acute symptoms, the presence of a sinus tract, an infected cyst, or in cases not responding to endodontic treatment. Both acute and chronic periradicular abscesses are polymicrobial infections with large numbers of bacteria.^{29,89} With the exception of species of Proprionibacterium and Actinomyces, it is controversial if asymptomatic lesions harbor extraradicular bacteria for very long beyond initial invasion of the tissue.52,68,90 Periapical inflammatory lesions (granulomas) contain macrophages, lymphocytes (T-cells and B-cells), plasma cells and neurophils.⁹¹⁻⁹³ Their function is to prevent microorganisms from invading periradicular tissues. Both the pulp and periradicular inflammatory tissues have been shown to mount cellular and humoral responses to the microorganisms.94-100 Microbial invasion of periradicular tissues results in production of an abscess or cellulitis. Patients with an abscess or cellulitis have significant clinical signs and symptoms as a result of both nonspecific inflammation and spe-

Endodontic Infections

Table 3						
COMPARISON OF	RETREATMENT	STUDIES	CULTURING E	NTEROCOCCUS	FAECALIS	
Study	Molander 1998	Sundqvist 1998	Piciuliene 2000	Handcock 2001	Piciuliene 2001	Pinheiro 2003
Canals Cultured	100	54	25	54	40	60
Positive Cultures	32	24	20	34	33	51
% Gm+	85	87	nd*	80	nd	83
% Facultative	69	58	nd	56	nd	5 7
Species in Canal	1.7	1.3	nd	1.7	nd	1.8
Canals with E. face * - not determined	calis 25%	16%	56%	16%	52% 4	5 %

cific immunological responses. In studies that examined periapical inflammatory lesions microscopically, extracellular bacteria were only seen when the associated teeth were symptomatic.^{84,85,101} However, several studies using culturing methods have isolated bacteria from "asymptomatic lesions."102-105 Some of the patients reported in these studies had a sinus tract (fistula) through the mucosa or the attachment apparatus. Sinus tracts are associated with chronic periradicular abscesses (suppurative apical periodontitis) that always have a polymicrobial infection. They are relatively asymptomatic because the sinus tract provides a pathway of drainage. It is possible that some specimens in these studies were contaminated with microbes from the root-end and apical foramen during curettement of the specimen. It is also possible that some samples were contaminated with salivary bacteria during the surgical procedure. Molecular methods have demonstrated the presence of microbial DNA in tissues removed during surgical treatment of asymptomatic periradicular lesions.^{106,107} Whether the microbial DNA is from viable or nonviable microbes cannot be determined and there is the possibility of sample contamination. Future studies should clarify these findings.

Invasion of periradicular tissues is related to the virulence of the microorganisms and the host's resistance. Periapical inflammatory lesions are dynamic inflammatory events and may contain an abscess with bacteria, a cyst (possibly infected), and surrounding inflammatory tissue simultaneously at the time of sample collection.¹⁰⁸ A major reason for persistent periradicular lesions is remaining intra-radicular infection caused by an incompletely debrided root canal space.84,109 This concept is supported by the high success rate attained when root-ends are resected and root-end fillings are placed to seal the apical canal system of teeth with failing nonsurgical endodontics.^{110, 111}

Bacteria Isolated after Unsuccessful Endodontic Treatment

The lack of periradicular healing following root canal treatment seems most likely to be related to the persistence of microbes in the root canal system. This would seem to be related to an inability to effectively shape, clean, and seal the complete root canal system. Interestingly, the microflora cultured from previously filled root canals with persistent apical lesions differs significantly for the microbes in untreated

necrotic canals.^{24,26,109,112,113} Bacteria isolated from canals previously obturated but still associated with radiographic lesions tend to have more facultative bacteria rather than strict anaerobes^{26,109,112-117} (Table 3). Instead of having approximately equal amounts of gram-negative and gram-positive bacteria, bacteria isolated from canals previously filled tend to have only one to two cultivable strains of mainly gram-positive bacteria. In several studies, previously filled canals had a relative increase in the presence of Enterococcus faecalis.^{26,109,112-117} This is likely the result of a change in selective pressures that differ in a canal filled with gutta-percha/sealer and a canal with necrotic pulp. The percentages of other species varied among the studies and there were many cases with no cultivable bacteria.

A recent study used PCR to detect the presence of bacteria in all 22 previously root-filled teeth with persistent periradicular lesions.¹¹⁸ *E. faecalis* was detected in 17/22 (77 percent) of the failing treatments. The next most detected organisms were *Proprionibacterium alactolyticus* (12/22), *P. propionicum* (11/22), *Filofactor alocis* (10/22), and *Dialiste pneumosintes* (10/22).¹¹⁹ The average number of species found in each canal was four organisms compared with a mean of less than two for studies

that culture for bacteria.^{26,109,112-117} This illustrates a relative increase in sensitivity in the ability of molecular techniques to detect organisms compared to cultivation of the organisms. Filofactor alocis and Dialiste pneumosintes have not been isolated from previously filled root canals with periradicular lesions.¹¹⁹ They are anaerobic bacteria recently shown to prevalent in untreated root he canals.^{119,120} The presence of bacteria in all of the failed root canals supports the assertion that the vast majority of endodontic treatment failures are caused by intraradicular infections.

The use of calcium hydroxide in canals may contribute to the selection of *E. faecalis* in failed endodontics. A recent study demonstrated that *E. faecalis* is resistant to calcium hydroxide at a pH of 11.1 but not pH 11.5. *E. faecalis* has a proton pump that can be used to decrease the pH to the level it needs for survival.¹²¹ Another study has shown that the addition of chlorhexidine to calcium hydroxide increases the efficacy of calcium hydroxide to inhibit the growth of *E. faecalis*.¹²²

Endodontic Abscesses and Cellulitis

The extent of endodontic infections beyond the root canal system is related to the virulence of the bacteria, host response, and associated anatomy. Infections may localize or continue to spread. An abscess is a cavity of purulent exudate (pus) consisting primarily of bacteria, bacterial by-products, inflammatory cells, lysed cells and their contents. The content of inflammatory cells includes enzymes, which are damaging to the surrounding tissues. Endodontic abscesses are polymicrobial infections with organisms similar to found those in infected root canals.^{29,49,123-127}

A diffuse erythematous cellulitis results if the infection spreads into surrounding tissues. The spread of infection into deep facial spaces may be life threatening.¹²⁸ Facial spaces are potential anatomic areas between fascia and underlying tissues and organs that provide a pathway for spread of an infection.¹²⁸ A diffuse cellulitis may have foci of purulence (abscess). The spread of infection and ensuing edema associated with inflammation often produces an indurated swelling. Over time, neutrophils accumulate and produce a fluctuant abscess. This concept supports the rationale for early incision for drainage

THE PRESENCE OF BACTERIA IN ALL OF THE FAILED ROOT CANALS SUPPORTS THE ASSERTION THAT THE VAST MAJORITY OF ENDODONTIC TREATMENT FAILURES ARE CAUSED BY INTRARADICULAR INFECTIONS.

to provide a pathway for the drainage of bacteria, bacterial by-products, and inflammatory mediators.¹²⁸ Drainage also improves circulation to the area so if antibiotics are prescribed they will more likely be delivered at a minimum inhibitory concentration.

Treatment of Acute Abscesses and Cellulitis

As with any disease process, diagnosis is of most importance. The correct diagnosis of an abscess or cellulitis of endodontic origin allows appropriate management. Chemomechanical debridement of the associated root canal system will remove the cause of the infection, however, drainage from the access opening does not significantly reduce postoperative pain or swelling.¹²⁹ Incision for drainage will usually allow rapid improvement in the patient's condition. Follow-up on a daily basis should be made to see if further treatment is indicated.

The prescription of adjunctive antibiotics is recommended in conjunction with appropriate endodontic treatment for progressive or persistent infections with the following signs and symptoms: fever (>100° F.), malaise, cellulitis, lymphadenopathy, and unexplained trismus.¹²⁸ Antibiotics are not recommended for an irreversible pulpitis, an acute apical periodontitis, a draining sinus tract, after endodontic surgery, or incision for drainage of a localized abscess (without fever, cellulitis, or lympadenopathy).¹³⁰⁻¹³⁴

Ideally, the choice of an antibiotic would be based on susceptibility testing of the organisms isolated from each patient's infection. Unfortunately, it takes days and sometimes weeks to culture, isolate each organism in pure culture, and do antibiotic susceptibility tests for anaerobic bacteria. Instead, recommendations based on clinical experience and previous susceptibility testing are made for normal healthy patients. It is prudent to get antibiotic susceptibility tests for immunocompromised patients if initial treatment is not successful. Patients must be made aware of benefits versus risks of taking antibiotics. Case reports have implicated antibiotics with reduction in the effectiveness of oral contraceptives; however, rifampin is the only antimicrobial shown to have such an effect.¹³⁵ Interactions of prescribed antibiotics with other medications are always of concern. For example, macrolide antibiotics should not be prescribed for a patient taking HMG-CoA reductase inhibitors such as Lipitor.

Antibiotics should be taken for two to three days after resolution of the major clinical signs and symptoms. To prevent selection of resistant organisms, a high dose of antibiotic for a short term is preferred to a low dose for a long term. A sixto seven-day around-the-clock regimen is adequate for the majority of patients once the source of the infections is removed. Recent antibiotic susceptibility

Endodontic Infections

tests have shown that penicillin VK is still the antibiotic of choice for endodontic infections.^{136,137} It is prescribed with a loading dose of 1,000 mg followed by 500 mg every four to six hours. Amoxicillin has a broader spectrum and is recommended for the most serious infections. Amoxicillin in combination with clavulanate (Augmentin) is effective against beta lactamase producing organisms. Amoxicillin or amoxicillin with clavulanate is prescribed with a loading dose of 1,000 mg followed by 500 mg every eight hours. Because metronidazole is only effective against anaerobes, it should not be prescribed by itself. Metronidazole may be prescribed in combination with penicillin. It is prescribed with a loading dose of 1,000 mg followed by 500 mg every four to six hours.

For patients allergic to penicillin, clarithromycin or aziththromycin may be considered instead of erythromycin. Erythromycin is not effective against the anaerobes commonly found in endodontic infections. For patients allergic to penicillin with serious infections, clindamycin is recommended. Clindamycin is prescribed with a loading dose of 600 mg and followed by 300 mg every six hours.

Metastatic Infection Associated with Endodontic Infections

Endodontic infections may be associated with metastatic infections by direct extension of the infection, via microbes carried through the blood (bacteremia), and by the release of bacterial products and inflammatory mediators. The direct extension of a periradicular abscess may reach the maxillary sinuses, cavernous sinus, orbit, brain, or via parapharyngeal pathways produce Ludwig's angina. The importance of removing the source of the infection, providing drainage, and adjunctive antimicrobial support has been discussed.

A bacteremia may be produced with both nonsurgical root canal instrumentation and surgical treatment.¹³⁸⁻¹⁴² In normal healthy individuals, bacteremias are usually of no consequence because the immune system rapidly eliminates the microbes. Bacteremias associated with the oral cavity occur with mastication, flossing, toothbrushing, and other daily activities. Bacteremia is considered a risk factor for the development of infective endocarditis in patients with congenital of acquired cardiac defects. Although dental procedures have not been confirmed

RECENT ANTIBIOTIC SUSCEPTIBILITY TESTS HAVE SHOWN THAT PENICILLIN VK IS STILL THE ANTIBIOTIC OF CHOICE FOR ENDODONTIC INFECTIONS.

as risk factors, the American Dental Association does recommend prophylactic antibiotics for patients susceptible to infective endocarditis or with artificial prosthetic devices.¹⁴³⁻¹⁴⁵

Metastatic infections are not the same as the Theory of Focal Infection which still receives some attention.146 Focal infection was defined as a localized or generalized infection caused by the dissemination of bacteria or their toxic products from a distant focus of infection.¹⁴⁷ In general, the support of this theory was anecdotal or flawed research.148 However, recent studies suggest a relationship between the elevated values of C-reactive proteins and other inflammatory proteins that accompany chronic periodontal infections to be associated with atherosclerosis, cardiovascular. cerebrovascular disease. or preterm low-birthweight.^{149,150} Future studies must determine if these relationships are causal or consequential.

Summary

In summary, this article reviewed the microbiologic aspects of endodontic infections with an emphasis on removal of the cause of the infection. In addition, recommendations for appropriate adjunctive use of antibiotics was presented. These recommendations are based on recent antibiotic susceptibility tests. Finally, the issue of metastatic endodontic infections versus the "theory of focal infection" was presented.

To request a printed copy of this article, please contact / J. Craig Baumgartner, DDS, PhD, Oregon Health & Science University, Mail Code 130B, 611 S.W. Campus Drive, Portland, Ore., 97239-3097.

References / **1.** Leeuwenhoek A, Leeuwenhoek's letter. Philosophical Transactions of the Royal Society of London 14(159):568-74, 1684.

2. Miller WD, Microorganisms of the human mouth. Philadelphia: The S.S. White Dental Mfg. Co.; 1890.

3. Miller W, Decomposition of the contents of the dentinal tubules as a disturbing factor in the treatment of pulpless teeth. *Ohio J Dental Sc* 10:288, 1890.

4. Xia T, Baumgartner JC, David LL, Isolation and identification of *Prevotella tannerae* from endodontic infections. *Oral Micro and Immun* 15:273-5, 1999.

5. Munson M, Pitt-Ford T, Chong B, Weightman A, Wade W, Molecular and cultural analysis of the microflora associated with endodontic infections. *J Dent Res* **81**:761-6, 2002.

6. Fouad A, Barry J, Caimano M, PCR-based identification of bacteria associated with endodontic infections. *J Clin Microbiol* 40:3223-31, 2002.

7. Baumgartner JC, Khemaleelakul S, Xia T, Association of spirochetes with endodontic infections. *J Endod* 29(4):290, 2003.

8. 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* 20:340-9, 1965.

9. Delivanis PD, Snowden RB, Doyle RJ, Localization of blood-borne bacteria in instrumented unfilled root canals. *Oral Surg Oral Med Oral Pathol* 52(4):430-2. 1981.

10. Delivanis PD, Fan VSC, The localization of blood-borne bacteria in instrumented unfilled and overinstrumented canals. *J Endod* 10(11):521-4, 1984.

11. Allard U, Stromberg T, Inflammatory reaction in the apical area of pulpectomized and sterile root canals in dogs. *Oral Surg Oral Med Oral Pathol* 48(5):463-6, 1979.

12. Tziafas D, Experimental bacterial anachoresis in dog dental pulps capped with calcium hydroxide. *J Endod* 15(12):591-5, 1989.

13. Sundqvist G, Bloom GD, Enberg K, Johansson E, Phagocytosis of *Bacteroides melaninogenicus* and *Bacteroides gingivalis in vitro* by human neutrophils. *J Periodont Res* 17:113-21, 1982.

14. Sundqvist G, Carlsson J, Herrmann B, Tärnvik A, Degradation of human immunoglobulins G and M and complement factors C3 and C5 by black-pigmented *Bacteroides*. J Med Microbiol 19:85-94, 1985.

15. Sundqvist G, Carlsson J, Hänström L, Collagenolytic activity of black-pigmented Bacteroides species. *J Periodont Res* 22:300-6, 1987.

16. Horiba N, Maekawa Y, Abe Y, Ito M, Matsumoto T, Nakamura H, Correlations between endotoxin and clinical symptoms or radiolucent areas in infected root canals. *Oral Surg Oral Med Oral Pathol* 71(4):492-5, 1991.

17. Horiba N, Maekawa Y, Yamauchi Y, Ito M, Matsumoto T, Nakamura H, Complement activation by lipopolysaccharides purified from gramnegative bacteria isolated from infected root canals. *Oral Surg Oral Med Oral Pathol* 74(5):648-51, 1992.

18. Dwyer TG, Torabinejad M, Radiographic and histologic evaluation of the effect of endotoxin on the periapical tissues of the cat. *J Endod* 7(1):31-5, 1981.

19. Kinder SA, Holt SC, Characterization of coaggregation between *Bacteroides gingivalis* T22 and *Fusobacterium nucleatum* T18. *Infect Immun* 57:3425-33, 1989.

20. Eftimiadi C, Stashenko P, Tonetti M, Mangiante PE, Massara R, Zupo S, et al. Divergent effect of the anaerobic bacteria by-product butyric acid on the immune response: suppression of Tlymphocyte proliferation and stimulation of interleukin-1 beta production. *Oral Microbiol Immunol* 6:17-23, 1991.

21. Maita E, Horiuchi H, Polyamine analysis of infected root canal contents related to clinical symptoms. *Endod Dent Traumatol* 6(5):213-7, 1990.

22. Baumgartner JC, Sequeira JJ, Xia T, Rocas I, Geographical differences in bacteria detected in endodontic infections using PCR. *J Endod* 28:238, 2002.

23. Kobayashi T, Hayashi A, Yoshikawa R, Okuda K, Hara K, The microbial flora from root canals and periodontal pockets of non-vital teeth associated with advanced periodontitis. *Int Endod J* 23:100-6, 1990.

24. Rolph H, Lennon A, Riggio M, Molecular identification of microorganisms from endodontic infections. *J Clin Microbiol* 39:3282-9, 2001.

25. Siqueira JF Jr, Taxonomic changes of bacteria associated with endodontic infections. *J Endod* 29(10):619-23, 2003.

26. Möller ÅJR, Microbiological examination of root canals and periapical tissues of human teeth. *Odontol Tidskr* 74(1), 1966.

27. Bergenholtz G, Micro-organisms from necrotic pulp of traumatized teeth. *Odont Revy* 25(4):347-58, 1974.

28. Sundqvist GK. Bacteriological studies of necrotic dental pulps [Odontological Dissertation No. 7]: University of Umea: Umea, Sweden; 1976.

29. Van Winkelhoff AJ, Carlee AW, de Graaff J, Bacteroides endodontalis and other black-pigmented Bacteroides species in odontogenic abscesses. Infect Immun 49:494-7, 1985.

30. Baumgartner JC, Falkler Jr WA, Bacteria in the apical 5 mm of infected root canals. *J Endod* 1991;17(8):380-3.

31. Möller AJR, Fabricius L, Dahlén G, Öhman AE, Heyden G. Influence on periapical tissues of indigenous oral bacteria and necrotic pulp tissue in monkeys. *Scand J Dent Res* 89:475-84, 1981.

32. Fabricius L, Dahlén G, Öhman AE, Möller ÅJR, Predominant indigenous oral bacteria isolated from infected root canals after varied times of closure. *Scand J Dent Res* 90:134-44, 1982.

33. Fabricius L, Dahlén G, Holm SE, Möller ÅJR, Influence of combinations of oral bacteria on periapical tissues of monkeys. *Scand J Dent Res* 90:200-6. 1982.

34. Dougherty W, Bae K, Watkins B, Baumgartner J, Black-pigmented bacteria in coronal and apical segments of infected root canals. *J Endod* 24(5):356-8, 1998.

35. Siqueira JF Jr, Rocas I, Lopes H, Patterns of microbial colonization in primary root canal infections. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod* 93(2):174-88, 2002.

36. Gomes B, Drucker D, Lilley J, Positive and negative associations between bacterial species in dental root canals. *Microbios* 80(325):231-43, 1994.

37. Gomes B, Lilley J, Drucker D, Associations of endodontic symptoms and signs with particular combinations of specific bacteria. *Int Endod J* 29:69-75, 1996.

38. Sundqvist G, Associations between microbial species in dental root canal infections. *Oral Microbiol Immunol* 7:257-62, 1992.

39. Peters L, Wesselink PR, van Winkelhoff A-J, Combinations of bacterial species in endodontic infections. *Int Endod J* 35:698-702, 2002.

40. Jung I-Y, Choi B-k, Kum K-Y, Yoo Y-J, Yoon T-C, Lee S-J, et al. Identification of oral spirochetes at the species level and their association with other bacteria in endodontic infections. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod* 92:329-34, 2001.

41. Jung I-Y, Choi B-K, Kum K-Y, Roh B-D, Lee S-J, Lee C-Y, et al. Molecular epidemiology and association of putative pathogens in root canal infection. *J Endod* 26(10):599-604, 2004.

42. Kolenbrander PE, Andersen RN, Inhibition of coaggregation between *Fusobacterium nucleatum* and *Porphyromonas (Bacteroides) gingivalis* by lactose and related sugars. *Infect Immun* 57:3204-9, 1989.

43. Nesbitt W, Fukushima H, Leung K-P, Clark W, Coaggregation of prevotella intermedia with oral actinomyces species. *Infect Immun* 61(5):2011-4, 1993.

44. Grimaudo NJ, Nesbitt WE, Coaggregation of *Candida albicans* with oral *Fusobacterium* species. *Oral Microbiaol Immunol* 12:168-73, 1997.

45. Noiri Y, Ehara A, Kawahara T, Takemurs N, Ebisu S, Participation of bacterial biofilms in refractory and chronic periapical periodontitis. *J Endod* 28(10):679-83, 2002.

46. Khemaleelakul S, Baumgartner JC, Pruksakorn S, Coaggregation of bacteria associated with acute endodontic infections. *J Endod* in press 2004.

47. Baumgartner JC, Falkler WA, Beckerman T, Experimentally induced infection by oral anaerobic microorganisms in a mouse model. *Oral Microbiol Immunol* 7:253-6, 1992.

48. Sundqvist G, Johansson E, Sjögren U, Prevalence of black-pigmented Bacteroides species in root canal infections. *J Endod* 15:13-9, 1989.

49. Baumgartner JC, Watkins BJ, Prevalence of black-pigmented bacteria associated with root canal infections. *J Endod* 20(4):191, 1994.

50. Griffee MB, Patterson SS, Miller CH, Kafrawy AH, Newton CW, The relationship of *Bacteroides melaninogenicus* to symptoms associated with pulpal necrosis. *Oral Surg Oral Med Oral Pathol* 50(5):457-61, 1980.

51. Yoshida M, Fukushima H, Yamamoto K, Ogawa K, Toda T, Sagawa H, Correlation between clinical symptoms and microorganisms isolated from root canals of teeth with periapical pathosis. *J Endod* **13**(1):24-8, 1987.

52. Happonen RP, Periapical actinomycosis: a follow-up study of 16 surgically treated cases. *Endod Dent Traumatol* 2:205-9, 1986.

53. Hashioka K, Suzuki K, Yoshida T, Nakane A, Horiba N, Nakamura H, Relationship between clinical symptoms and enzyme-producing bacteria isolated from infected root canal. *J Endod* 20(2):75-

7, 1994.

54. Heimdahl A, Von Konow L, Satoh T, Nord CE, Clinical appearance of orofacial infections of odontogenic origin in relation to microbiological findings. *J Clin Microbiol* 22:299-302, 1985.

55. Brook I, Frazier EH, Gher ME, Microbiology of periapical abscesses and associated maxillary sinusitis. *J Periodontol* 67(6):608-10, 1996.

56. Haapasalo M, Ranta H, Ranta K, Shah H. Black-pigmented *Bacteroides* spp. in human apical periodontitis. *Infect Immun* 53:149-53, 1986.

57. Gharbia S, Haapasalo M, Shah H, Kotiranta A, Lounatmaa K, Pearce M, et al. Characterization of *Prevotella intermedia* and *Prevotella nigrescens* isolates from periodontic and endodontic infections. *J Periodontol* 65(1):56-61, 1994.

58. Van Steenbergen TJM, Van Winkelhoff AJ, Mayrand D, Grenier D, De Graaff J, Bacteroides endodontalis sp. nov., an asaccharolytic black-pigmented Bacteroides species from infected dental root canals. Int J Syst Bacteriol 34:118-20, 1984.

59. Baumgartner J, Bae K, Xia T, Whitt J, David L, Sodium dodecyl sulfate-polyacrylamide gel electrophoresis and polymerase chain reaction for differentiation of *Prevotella intermedia* and *Prevotella nigrescens. J Endod* 25(5):324-8, 1999.

60. Sundqvist GK, Carlsson J, Herrmann BF, Höfling JF, Väätäinen A, Degradation in vivo of the C3 protein of guinea-pig complement by a pathogenic strain of *Bacteroides gingivalis. Scand J Dent Res* 92:14-24, 1984.

61. Schein B, Schilder H, Endotoxin content in endodontically involved teeth. *J Endod* 1(1):19-21, 1975.

62. Schonfeld S, Greening A, Glick D, Frank A, Simon J, Endotoxic activity in periapical lesions. *Oral Surg Oral Med Oral Pathol* 53(1):82-7, 1982.

63. Horiba N, Maekawa Y, Matsumoto T, Nakamura H, A study of the distribution of endotoxin in the dentinal wall of infected root canals. *J Endod* 16(7):331-4, 1990.

64. Nelson-Filho P, Leonardo MR, Silva LAB, Assed S, Radiographic evaluation of the effect of endotoxin (OPS) plus calcium hydroxide on apical and periapical tissues of dogs. *J Endod* 28(10):694-6, 2002.

65. Yamasaki M, Nakane A, Kumazawa M, Hashioka K, Horiba N, Nakamura H, Endotoxin and gram-negative bacteria in the rat periapical lesions. *J Endod* 18(10):501-4, 1992.

66. Safavi KE, Nichols FC, Effect of calcium hydroxide on bacterial lipopolysaccharide. *J Endod* 19(2):76-8, 1993.

67. Sunde P, Olsen I, Debelian G, Tronstad L, Microbiota of periapical lesions refractory to endodontic therapy. *J Endod* 28(4):304-10, 2002.

68. Sundqvist G, Reuterving CO, Isolation of Actinomyces israelii from periapical lesion. J Endod 6:602-5 1980.

69. Siqueira JF Jr, Rocas I, Souto R, de Uzeda M, Colombo A, Actinomyces species, streptococci, and enterococcus faecalis in primary root canal infections. *J Endod* 28(3):168-72, 2002.

70. Figdor D, Davies J, Cell surface structures of *Actinomyces israelii*. *Australian Dent J* 42(2):125-8, 1997.

71. Xia T, Baumgartner JC, Occurrence of actinomyces in infections of endodontic origin. *J Endod* 29(9):549-52, 2003.

72. Rush J, Sulte H, Cohen D, Makkaway H, Course of infection and case outcome in individuals diagnosed with microbial colonies morphologically consistent with actinomyces species. *J Endod* 28(8):613-8, 2002.

73. Barnard D, Davies J, Figdor D, Susceptibility of *Actinomyces israelii* to antibiotics,

Endodontic Infections

sodium hypochlorite and calcium hydroxide. *Int Endod J* 29:320-6, 1996.

74. Gomes BPFA, Drucker DB, Lilley JD, Association of specific bacteria with some endodontic signs and symptoms. *Int Endod J* 27(6):291-8, 1994.

75. Siqueira JF Jr, Rocas I, Andrade A, de Uzeda M, Peptostreptococcus micros in primary endodontic infections as detected by 16S rDNA-based polymerase chain reaction. *J Endod* 29(2):111-3, 2003.

76. Rôças I, Siqueira JF Jr, Andrade A, Uzeda M, Oral treponemes in primary root canal infections as detected by nested PCR. *Int Endod J* 36:20-6, 2003.

77. Alapati S, Brantley W, Svec T, Powers J, Mitchell J, Scanning electron microscope observations of new and used nickel-titanium rotary files. *J Endod* 29(10):667-9, 2003.

78. Siqueira JF Jr, Rôças JN, Souto R, deUzeda M, Colombo AP, Checkerboard DNS-DNA hybridization analysis of endodontic infections. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod* 89(6):744-8, 2000.

79. Sen B, Safavi K, Spangberg L, Growth patterns of candida albicans in relation to radicular dentin. Oral Surg Oral Med Oral Pathol Oral Radiol Endod 84(1):68-73, 1997.

80. Sen BH, Piskin B, Demirci T, Observation of bacteria and fungi in infected root canals and dentinal tubules by SEM. *Endod Dent Traumatol* 11(1):6-9, 1995

81. Waltimo TMT, Sirén EK, Torkko HLK, Olsen I, Haapasalo MPP, Fungi in therapy-resistant apical periodontitis. *Int Endod J* 30:96-101, 1997.

82. Siqueira JF Jr, Rocas I, Lopes H, Elias C, de Uzeda M, Fungal infection of the radicular dentin. *J Endod* 28(11):770-3, 2002.

83. Baumgartner JC, Watts CM, Xia T, Occurrence of *candida albicans* in infections of endodontic origin. *J Endod* 26(12):695-8, 2000.

84. Nair PNR, Sjogren U, Krey G, Kahnberg KE, Sundqvist G, Intraradicular bacteria and fungi in root-filled, asymptomatic human teeth with therapy-resistant periapical lesions: a long-term light and electron microscopic follow-up study. *J Endod* 16(12):580-7, 1990.

85. Nair PNR, Light and electron microscopic studies of root canal flora and periapical lesions. *J Endod* 13:29-39, 1987.

86. Sabeti M, Simon J, Nowzari H, Slots J,Cytmegalovirus and Epstein-Barr virus active infection in periapical lesions of teeth with intact crowns. *J Endod* 29(5):321-3, 2003.

87. Sabeti M, Valles Y, Nowzari H, Simon J, Kermani-Arab V, Slots J, Cytomegalovirus and Epstein-Barr virus DNA transcription in endodontic symptomatic lesions. *Oral Microbiol Immunol* 18:104-8, 2003.

88. Tronstad L, Barnett F, Cervone F, Periapical bacterial plaque in teeth refractory to endodontic treatment. *Endod Dent Traumatol* 6:73-7, 1990.

89. Weiger R, Manncke B, Werner H, Lost C, Microbial flora of sinus tracts and root canals of non-vital teeth. *Endod Dent Traumatol* 11(1):15-9, 1995.

90. O'Grady JF, Reade PC, Periapical actinomycosis involving *Actinomyces israelii*. J Endod 14:147-9, 1988.

91. Barkhordar RA, Desousa YG, Human Tlymphocyte subpopulations in periapical lesions. *Oral Surg Oral Med Oral Pathol* 65(6):763-6, 1988.

92. Stashenko P, Wang SM, T helper and T suppressor cell reversal during the development of

induced rat periapical lesions. *J Dent Res* 68:830-4, 1989.

93. Stern MH, Dreizen S, Mackler BF, Selbst AG, Levy BM, Quantitative analysis of cellular composition of human periapical granuloma. *J Endod* 7:117-22, 1981.

94. Hahn C, Falkler WAJ, Antibodies in normal and diseased pulps reactive with microorganisms isolated from deep caries. *J Endod* 18(1):28-31, 1992.

95. Jontell M, Bergenholtz G, Scheynius A, Ambrose W, Dendritic cells and macrophages expressing Class II antigens in the normal rat incisor pulp. *J Dent Res* 67:1263-6, 1988.

96. Jontell M, Okiji T, Dahlgren U, Bergenholtz G, Immune defense mechanisms of the dental pulp. *Crit Rev Oral Biol Med* 9(2):179-200, 1998.

97. Baumgartner JC, Falkler WAJ, Reactivity of IgG from explant cultures of perapical lesions with implicated microorganisms. *J Endod* 17(5):207-12, 1991.

98. Baumgartner JC, Falkler WAJ, Biosynthesis of IgG in periapical lesion explant cultures. *J Endod* 17(4):143-6, 1991.

99. Kettering JD, Torabinejad M, Jones SL, Specificity of antibodies present in human periapical lesions. *J Endod* 17(5):213-6, 1991.

100. Kuo M, Lamster I, Hasselgren G, Host Mediators in endodontic exudates. *J Endod* 24(9):598-603, 1998

101. Walton RE, Ardjmand K, Histological evaluation of the presence of bacteria in induced periapical lesions in monkeys. *J Endod* 18(5):216-21, 1992.

102. Tronstad L, Barnett F, Riso K, Slots J, Extraradicular endodontic infections. *Endod Dent Traumatol* 3(2):86-90, 1987.

103. Wayman BE, Murata SM, Almeida RJ, Fowler CB, A bacteriological and histological evaluation of 58 peripical lesions. *J Endod* 18(4):152-5, 1992.

104. Iwu C, MacFarlane TW, MacKenzie D, Stenhouse D, The microbiology of periapical granulomas. Oral Surg Oral Med Oral Pathol 69:502-5, 1990.

105. Abou-Rass M, Bogen G, Microorganisms in closed periapical lesions. *Int Endod J* 31(31):39-47, 1998.

106. Sunde P, Tronstad L, Eribe E, Lind P, Olsen I, Assessment of periradicular microbiota by DNA-DNA hybridization. *Endod Dent Traumatol* 16:191-6, 2000.

107. Gatti J, Dobrik J, Smith C, White R, Socransky S, Skobe Z, Bacteria of asymptomatic periradicular endodontic lesions identified by DNA-DNA hybridization. *Endod Dent Traumatol* 16:197-204, 2000.

108. Baumgartner JC, Picket AB, Muller JT, Microscopic examination of oral sinus tracts and their associated periapical lesions. *J Endod* 10(4):146-52, 1984.

109. Sundqvist G, Figdor D, Persson S, Sjögren U, Microbiologic analysis of teeth with failed endodontic treatment and the outcome of conservative retreatment. Microbiologic analysis of teeth with failed endodontic treatment and the outcome of conservative retreatment. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod* 85(1):86-92, 1998.

110. Rubinstein R, Kim S, Short-term observation of results of endodontic surgery with the use of a surgical operation microscope and super-EBA as root-end filling material. *J Endod* 25(1):43-8, 1999

111. Rubinstein R, Kim S, Long-term follow-

up of cases considered healed one year after apical microsurgery. *J Endod* 28(5):378-83, 2002.

112. Molander A, Reit C, Dahlen G, Kvist T, Microbiological status of root-filled teeth with apical periodontitis. *Int Endod J* 31:1-7, 1998.

113. Hancock HI, Sigurdsson A, Trope M, Moiseiwitsch J, Bacteria isolated after unsuccessful endodontic treatment in a North American population. Oral Surg Oral Med Oral Pathol Oral Radiol Endod 91(5):579-86, 2001.

114. Siren E, Haapasalo M, Ranta K, Salmi P, Kerosuo N, Microbiological findings and clinical treatment procedures in endodontic cases selected for microbiological investigation. *Int Endo J* 30(2):91-5, 1997.

115. Peciuliene V, Balciuniene I, Eriksen H, Haapasalo M, Isolation of enterococcus faecalis in previously root-filled canals in a Lithuanian population. *J Endod* 26(10):593-5, 2000.

116. Peciuliene V, Reynaud A, Balciuniene I, Haapasalo M, Isolation of yeasts and enteric bacteria in root-filled teeth with chronic apical periodontitis. *Int Endod J* 34:429-34, 2001.

117. Pinheiro E, Gomes B, Ferraz C, Sousa E, Teixeira F, Souza-Filho FJ, Microorganisms from canals of root-filled teeth with periapical lesions. *Int Endod J* 36:1-11, 2003.

118. Siqueira JF Jr, Rocas I, Polymerase chain reaction-based analysis of microorganisms associated with failed endodontic treatment. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod* 97(1):85-94, 2004.

119. Siqueira JF Jr, Rocas I, Detection of Filifactor alocis in endodontic infections associated with different forms of periradicular diseases. *Oral Microbiol Immunol* 18:263-5, 2003.

120. Rôças I, Sequeira JJ, Indentification of Dialister pneumosintes in acute periradicular abscesses of humans by nexted PCR. *Anaerobe* 8:75-8, 2002.

121. Evans M, Davies J, Sundqvist G, Figdor D, Mechanisms involved in the resistance of enterococcus faecalis to calcium hydroxide. *Int Endod J* 35:221-8, 2002.

122. Evans M, Baumgartner JC, Khemaleelakul S, Xia T, Efficacy of calcium hydroxide: chlorhexidine paste as an intracanal medication in bovine dentin. *J Endod* 29(5):338-9, 2003.

123. Brook I, Frazier E, Gher MJ, Microbiology of periapical abscesses and associated maxillary sinusitis. *J Periodontal* 67(6):608-10, 1996.

124. Lewis MAO, MacFarlane TW, McGowan DA, Quantitative bacteriology of acute dento-alveolar abscesses. *J Med Microbiol* 21:101-4, 1986.

125. Williams BL, Bacteriology of dental abscesses of endodontic origin. *J Clin Microbiol* 18(4):770-4, 1983.

126. Oguntebi B, Slee AM, Tanzer JM, Langeland K, Predominant microflora associated with human dental periapical abscesses. *J Clin Microbiol* 15:964-6, 1982.

127. Baumgartner JC, Watkins JB, Bae KS, Xia T, Association of black-pigmented bacteria with endodontic infections. *J Endod* 25(6):413-5, 1999.

128. Baumgartner JC, Hutter JW, Endodontic Microbiology and Treatment of Infections. In: Cohen S, Burns R, editors. Pathways of the Pulp. 8th ed. St. Louis: C.V. Mosby; 2001.

129. Nusstein J, Reader A, Beck M, Effect of drainage upon access on postoperative endodontic pain and swelling in symptomatic necrotic teeth. *J Endod* 28(8):584-8, 2002.

130. Nagle D, Reader A, Beck M, Weaver J, Effect of systemic penicillin on pain in untreated

irreversible pulpitis. Oral Surg Oral Med Oral Pathol Oral Radiol Endod 90:636-40, 2000.

131. Pickenpaugh L, Reader A, Beck M, Meyers W, Peterson L, Effect of Prophylactic amoxicillin on endodontic flare-up in asymptomatic, necrotic teeth. *J Endod* 27(1):53-6, 2001.

132. Walton RE, Chiappinelli J, Prophylactic

penicillin: effect on post-treatment symptoms following root canal treatment of asymptomatic periapical pathosis. *J Endod* 19(9):466-70, 1993.

133. Henry M, Reader A, Beck M, Effect of penicillin on postoperative endodontic pain and swelling in symptomatic necrotic teeth. *J Endod* 27(2):117-23, 2001.

134. Fouad A, Rivera E, Walton R, Penicillin as a supplement in resolving the localized acute apical abscess. Oral Surg Oral Med Oral Pathol Oral Radiol Endod 81(5):590-5, 1996.

135. Hersh EV, Adverse drug interactions in dental practice. *J Am Dent Assoc* 130(2):236-51, 1999.

136. Khemaleelakul S, Baumgartner JC, Pruksakorn S, Identification of bacteria in acute endodontic infections and their antimicrobial susceptibility. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod* 94(6):746-55, 2002.

137. Baumgartner JC, Xia T, Antibiotic susceptibility of bacteria associated with endodontic abscesses. *J Endod* 29(1):44-7, 2003.

138. Heimdahl A, Hall G, Hedberg M, Sandberg H, Detection and quantitation by lysis-filtration of bacteremia after different oral surgical procedures. *J Clin Microbiol* 28(10):2205-9, 1990.

139. Bender IB, Seltzer S, Yermish M, The incidence of bacteremia in endodontic manipulation. *Oral Surg Oral Med Oral Pathol* 13(3):353-60, 1960.

140. Baumgartner JC, Heggers J, Harrison J, The incidence of bacteremias related to endodontic procedures. I. Nonsurgical endodontics. *J Endod* 2:135-40, 1976.

2:135-40, 1976.
141. Baumgartner JC, Heggers JP, Harrison JW, Incidence of bacteremias related to endodontic procedures. II. Surgical endodontics. J Endod 3(10):399-404, 1977.

142. Debelian GF, Olsen I, Tronstad L, Bacteremia in conjunction with endodontic therapy. *Endod Dent Traumatol* 11(3):142-9, 1995.

143. ADA, Council on Scientific Affairs: Antibiotics use in dentistry. J Am Dent Assoc 128(5):648, 1997.

144. ADA, Antibiotic prophylaxis for dental patients with total joint replacements. *J Am Dent Assoc* 128:1004-7, 1997.

145. Strom B, Abrutyn E, Berlin J, Kinman J, Feldman R, Stolley P, et al. Dental and cardiac risk factors for infective endocarditis A population-based, case-control study. *Ann Intern Med* 129(10):761-9, 1998.

146. Grossman LI, Focal infection: are oral foci of infection related to systemic disease? DCNA 4:749, 1960.

147. Rosenow EC, The relation of dental infection to systemic disease. *Dental Cosmos* 59:485, 1917.

148. Cohen S, Burns R, Pathways of the Pulp. St. Louis: C.V. Mosby; 2001.

149. Williams R, Offenbacher S, Periodontal medicine: the emergence of a new branch of periodontology. *Periodontol* 23:9-12, 2000.

150. Mendall M, Patel P, Ballam L, Strachan D, Northfield T, Reactive protein and its relation to cardiovascular risk factors: a population based cross sectional study. *Br Med J* 312:1061-5, 1996.

151. Glick M, Trope M, Pliskin ME, Detection of HIV in the dental pulp of a patient with AIDS. *J Am Dent Assoc* 119(5):649-50, 1989.

152. Glick M, Trope M, Bagsara, O, Pliskin ME, Human immunodeficiency virus infection of fibroblasts of dental pulp in seropositive patients. *Oral Surg Oral Med Oral Pathol* 71(6):733-6, 1991.



New Advances in the Management of Endodontic Pain Emergencies

Kenneth M. Hargreaves, DDS, PhD; Karl Keiser, DDS, MS

A B S T R A C T

The development of more effective strategies for managing endodontic pain emergencies draws from the research of both clinicians and basic scientists. This review explores evidence-based approaches for managing endodontic pain emergencies and newly emerging pain management strategies based upon molecular, cellular and physiologic research into pain mechanisms. he successful management of the endodontic emergency patient represents a challenging test of clinical acumen, requiring skills in diagnosis, endodontic treatment, and

clinical pharmacology. Although this scenario is often perceived as stressful to both the patient and clinician, we believe that skills leading to the effective and efficient management of these cases provide a high point of satisfaction to many practitioners. This review will focus on current evidence-based reviews and basic research fronts likely to provide effective strategies for managing endodontic pain patients. One overall strategy that has gained rather broad acceptance is the use of a structured mnemonic approach for evaluation and treatment of these patients. This "3D" consists of Diagnosis, approach Definitive dental treatment and Drugs.

Diagnosis is clearly a critical first step in evaluating any pain patient (**Table 1**). Several factors must be kept in mind when examining the emergency pain patient.¹ First, the patient's chief complaint should be noted. Important characteristics of the pain (location, spontaneity, effect of temperature or chewing, pain quality, etc.) should be elicited from the patient. These features are important not only in establishing the

differential diagnosis, but also in confirming the suspected tooth. For example, a patient with a chief complaint of severe lingering pain to cold would be expected to report these same symptoms when the suspected tooth is selectively stimulated with cold. In some cases, this may require rubber dam isolation of individual teeth in order to selectively stimulate the suspected tooth. Although selective testing often consumes additional examination time, the failure to reproduce the patient's chief complaint is a risk factor for a mis-diagnosis and should prompt the prudent practitioner to consider alternative diagnoses. The clinical examination should evaluate for potential etiologic factors of odontogenic pain (eg. caries, open margins of

Authors / Kenneth M. Hargreaves, DDS, PhD, is a professor and chairman of the Department of Endodontics and professor of pharmacology at the University of Texas Health Science Center at San Antonio. He is a diplomate of the American Board of Endodontics. He and his colleagues have published more than 80 papers and 20 reviews of chapters. In addition to teaching and research, he has a practice limited to endodontics.

Karl Keiser, DDS, MS, is an assistant professor and director of the Advanced Education in Endodontics Program in the Department of Endodontics at the University of Texas Health Science Center at San Antonio. Prior to joining the university faculty in San Antonio, he was the director of Undergraduate Endodontics at the University of Tennessee College of Dentistry. He is a diplomate of the American Board of Endodontics and a fellow of the Academy of General Dentistry. He maintains a practice limited to endodontics.

Table 1

DIAGNOSTIC STEPS IN EVALUATING THE ACUTE ODONTOGENIC PAIN PATIENT

Establish the Chief Complaint Patient's words History of CC (When did it start? Stop? What makes it worse? Better? Location?) Nature of pain (dull, throbbing, sharp, shooting, burning, etc)

Review Medical and Dental History

Clinical Examination Visual inspection Periodontal probing Periapical percussion/palpation Pulpal tests

Radiographic exam

Differential Diagnosis Endodontic diagnosis as a dual diagnosis

restorations, crown-root fractures) as well as potential pathophysiologic sequelae of pulpal or periradicular disease (eg. sinus tract, intra/extraoral swelling, lymphadenopathy). Clinical examinations should include assessment of pulpal responsiveness (typically using cold or electrical stimuli), mechanical allodynia (eg. percussion or palpation of teeth and soft tissues), periodontal pocketing. The radiographic exam should include evaluation of both suspected teeth (coronal examinations focusing on potentially open margins, pulp stones, etc, and radicular examinations focusing on loss of the lamina dura, periradicular radiolucencies or opacities, resorptive processes) as well as examination of other visible structures. It is the total analysis of these data that form the basis for establishing the differential diagnosis. In rare cases, interventional tests (eg. anesthetic injection, test cavity) may be indicated to verify the differential diagnosis. However, most clinicians would agree that the practitioner's ears (eg. skills in listening and soliciting a history) often provide the most important diagnostic information.

The development of a differential diagnosis must include consideration of both odontogenic and non-odontogenic forms of pain (**Table 2**). Although space limitations preclude a comprehensive review on this topic, readers can refer to several recent and comprehensive reviews.²⁻⁷ These reviews provide important and practical information on differential diagnosis and management of a wide range of non-odontogenic pain disorders that have been reported to mimic odontalgia.

The rationale for developing a differential diagnosis is obvious, but its application is challenging. The vast majority of dental pain emergencies consist of disorders with relatively little diagnostic challenge (eg. pulpal necrosis with acute apical abscess). This dominant pattern of odontogenic pain disorders may lead to clinical shortcuts in which a differential diagnosis is never established. Under these conditions, the patient presenting with a rare but very real form of nonodontogenic pain may not receive appropriate therapy due to a misdiagnosis. Thus, the challenge is to maintain vigilance in evaluating patients and developing differential diagnoses.

Table 2

SELECTED DIAGNOSES THAT CAN MIMIC ACUTE ODONTOGENIC PAIN

Diagnosis

Odontogenic Pain* Dentinal Hypersensitivity Reversible pulpitis Irreversible pulpitis Acute apical periodontitis Acute apical abscess

Non-Odontogenic pain -Musculoskeletal Myofascial pain - TMD Bruxism

Non-Odontogenic pain -Neuropathic Trigeminal neuralgia Atypical odontalgia Glossopharyngeal neuralgia

Non-Odontogenic pain -Neurovascular Migraine Cluster headaches

Non-Odontogenic pain -Inflammatory Allergic sinusitis Bacterial sinusitis

Non-Odontogenic pain -Systemic disorders Cardiac pain Herpes zoster Sickle cell anemia Neoplastic disease

Non-Odontogenic pain -Psychogenic origin Munchausen's Syndrome

*Odontogenic pain may arise from the suspected tooth, or the pain may be referred from another tooth.

The second "D" in the "3D" algorithm is for definitive dental treatment. Too often the busy practitioner favors simple pharmacotherapy over definitive dental treatment as an efficient means of managing the unscheduled emergency. However, numerous studies have established clearly that the delivery of appropriate dental procedures offers significant and substantial reductions in



Table 3

COMPARATIVE EFFICACY OF ORAL ANALGESICS*

Analgesic	<pre>% Patients with 50% Pain Relief **</pre>	Sample Size (N)
Ibuprofen 800 mg	100%	7 6
Ibuprofen 600 mg	79%	203
Acetaminophen 650 mg + Oxycodone 10 mg	66%	315
Acetaminophen 1,000 mg + Codeine 60 mg	57%	197
Ibuprofen 400 mg	56%	4,703
Morphine 10 mg IM injection	50%	946
Acetaminophen 1,000 mg	46%	2,759
Ibuprofen 200 mg	45%	1,414
Acetaminophen 600/650 mg	38%	1,886
Tramadol 100 mg	30%	882
Codeine 60 mg	15%	1,305
Placebo	18%	>10,000

* Data from: The Oxford League Table of Analgesic Efficacy (URL: http://www.jr2.ox.ac.uk/bandolier/booth/painpag/Acutrev/Analgesics/lftab.html)

** Percentage of patients with moderate to severe pain who report at least 50 percent pain relief at four hours to six hours after taking medication. Data are from randomized, double-blind, placebo-controlled analgesic clinical trials.

odontogenic pain due to acute inflammatory processes.

Pulpotomies can be considered for treating cases of irreversible pulpitis even when the pain is severe in magnitude. The procedure consists of anesthesia, rubber dam isolation, access, removal of pulp chamber contents with a sharp spoon, and avoiding penetration of the root canal systems. In addition to its technical simplicity, pulpotomies have been shown to result in a significant and substantial reduction in pain by as little as 24 hours after the procedure.⁸⁻¹⁰ Thus, pulpotomies represent a viable approach for managing even moderate to severe odontogenic pain due to irreversible pulpitis.

Pulpectomies include debridement of the root canal systems and are typically performed on necrotic cases or in those vital cases where sufficient time is available for this longer clinical procedure. The pathophysiology of pain associated with pulpal necrosis is more complex than pain associated with irreversible pulpitis and therefore the mechanisms for the pain relief differ between pulpectomy and pulpotomy. Pulpectomies only indirectly influence periradicular nociceptors by their removal of irritants from necrotic root canal systems. In contrast, pulpotomies directly reduce pulpal nociceptor activity via the axotomy that occurs during removal of dental pulp in the pulp chamber space. Although the mechanisms for pulpectomy-induced pain relief is more complex, clinical trials often demonstrate a significant and substantial reduction in pain by 24 hours to 36 hours after pulpectomy treatment.^{9,11-16}

Other dental procedures have been shown to reduce odontogenic pain. For example, occlusal adjustment reduces post-endodontic pain, particularly in patients presenting with pre-operative pain, percussion sensitivity or vital teeth.¹⁴ An incision for drainage procedure is indicated for management of swellings due to an infectious process, but is useful both for control of infection and the related pain.

If the tooth has a hopeless prognosis, extraction of painful but nonrestorable teeth reliably reduces pain. In one study, patients with moderate to severe pre-operative pain reported pain as none to mild by two to three days after dental extraction.¹⁷ Of course, the purpose of endodontics is to save teeth, and as described above, endodontic treatment significantly reduces odontogenic pain.⁸⁻¹⁶

The third "D" in the "3D" algorithm is for drugs. Non-steroidal anti-inflammatory drugs (NSAIDs) have been demonstrated to be effective in treating odontogenic pain. Randomized placebocontrolled studies in endodontic pain

patients have reported significant analgesic benefit from ibuprofen (400 mg to 600 mg), flurbiprofen (50 mg to 100 mg) and ketorolac (30 mg to 60 mg), with comparatively little benefit from etodolac (400 mg).^{10,11,15,16,18-20} More recent studies have indicated that the COX-2 isozyme is upregulated in inflamed human dental pulp and that rofecoxib (50 mg) significantly reduced post-endodontic pain compared to placebo.^{20,21} However, the analgesic benefit of rofecoxib 50 mg was similar to that observed in the first six hours following administration of ibuprofen 600 mg. Thus, the Coxib class of NSAIDs appear effective in treating odontogenic pain, but not superior to the mixed COX1-2 inhibitors such as ibuprofen. For patients with contraindications for NSAIDs, acetaminophen (650 mg to 1000 mg) alone or with an opioid appear effective for treating post-endodontic pain.¹¹ A very useful Web site for comparing analgesic effectiveness for acute inflammatory pain is the Oxford League Table of Analgesic Efficacy.²² This group uses evidence-based analyses to develop comparison among analgesics for patients with moderate to severe pain. A summary of these data are presented in Table 3 and are the results of a large and ongoing meta-analysis of analgesic clinical trials across multiple acute pain conditions. A meta-analysis of clinical trials evaluating oral NSAIDs for postendodontic pain has been published recently with similar results.23

Recent studies indicate that NSAIDs such as ibuprofen appear to interfere with the anti-platelet effects of aspirin.²⁴ In this study, the administration of rofecoxib or acetaminophen had no effects on the anti-platelet effects of aspirin. Moreover, a retrospective study of more than 7,000 post-myocardial infarction patients taking low-dose aspirin therapy over several years found a significantly elevated hazard ratio for cardiac death in patients also taking NSAIDs.²⁵ This finding has lead to the suggestion that patients taking "baby" aspirin should not take certain NSAIDs under chronic conditions. It is not yet known whether a short-term course of NSAID treatment imposes a medically significant risk for cardiac death in certain patients. However, these data are consistent with the consideration of alternative analgesics such as acetaminophen combinations or COX2 inhibitors in patients taking low-dose aspirin for medical indications.

THE COXIB CLASS OF NSAIDS APPEAR EFFECTIVE IN TREATING ODONTOGENIC PAIN, BUT NOT SUPERIOR TO THE MIXED COX1-2 INHIBITORS SUCH AS IBUPROFEN.

NSAIDs are effective for treating the majority of endodontic pain patients who can tolerate this class of analgesics. However, a small subset of patients may still experience significant pain even after consuming maximal dosages of NSAIDs. Therefore, under certain conditions, the co-administration of NSAIDs with other analgesics may be indicated for short-term (one- to two-day) treatment of severe pain. For example, a randomized placebo-controlled clinical trial in post-endodontic patients demonstrates that the opioid tramadol (Ultram) significantly enhanced NSAID analgesia.16 In addition, randomized placebocontrolled clinical trials demonstrate that acetaminophen (650 mg to 1,000 mg) significantly enhances NSAID analgesia in both oral surgery and postendodontic pain trials.^{26,27} Given the substantial reduction in pain that occurs typically with pulpectomy or pulpotomy procedures, NSAID therapy alone is sufficient for the majority of patients who can tolerate this class of analgesics. However, opioids or acetaminophen co-treatment may serve an important adjunctive role when combined with NSAIDs role in managing severe odontalgia. When indicated, these adjunctive therapeutics are usually provided for one to two days after treatment due to the substantial reduction in pain that occurs due to the definitive dental treatment.

Many practitioners report difficulty in obtaining effective local anesthesia when treating the patient with severe odontalgia. Proposed mechanisms for local anesthesia failure have been reviewed recently and include upregulation of sodium channels resistant to local anesthetics (eg. NaV1.8, NaV1.9), central sensitization.²⁸ Clinical trials in endodontic patients indicate clearly that a positive "lip sign" does not predict effective pulpal anesthesia (see 28), and thus clinicians should not interpret a lip sign as a marker for effective pulpal anesthesia. Although some textbooks claim that lip sign predicts pulpal anesthesia, this is not supported by clinical research and recent basic research indicates that this disparity is likely due to the fact that unmyelinated nociceptors are more resistant to lidocaine as compared to the A delta fibers that mediate touch or proprioception.²⁹

One effective strategy for enhancing anesthesia in odontalgia patients is the use of the intraosseous route of injection.³⁰ Clinical trials in odontalgia patients demonstrate that intraosseous anesthetic injection significantly enhances anesthesia due to inferior alveolar nerve block.³¹ The use of vasoconstrictor-containing local anesthetics is associated with a transient tachycardia after intraosseous injection.³² The use of 3% mepivacaine without vasoconstrictor avoids tachycardia but has a shorter duration of action.³² Thus, the intraosseous route of injection represents an evidence-based adjunct for the difficult

New Advances

to anesthetize mandibular tooth.

Many randomized, double-blind, placebo-controlled clinical trials have evaluated the effectiveness of antibiotics for reducing endodontic related pain. Most, but not all have failed to detect any difference from placebo for postendodontic pain, swelling, flare-up or analgesic consumption.^{17,33-36} Thus, the preponderance of evidence does not support a beneficial role of antibiotics for odontogenic pain. This is clearly an area of continuing interest and continued research in this area is warranted.

While this represents the current state of the art in pain control, ongoing basic research offers the potential for new approaches for pain control.

Summary

Taken together, the "3D" mnemonic of Diagnosis, Definitive Dental Treatment and Drugs represents an effective, efficient, and evidence-based approach for managing the endodontic pain patient. This review has highlighted the continued development of more effective and predictable methods for pain control as a process that combines advancements in both the basic science and clinical arenas. This is most certainly an ongoing process, and emphasizes the need for life-long learning as epitomized by the evidence-based approach to therapeutics. It is hoped the reader has found this material both stimulating and clinically relevant, with the ultimate effect of improved care for patients in pain. CDA

To request a printed copy of this article, please contact / Kenneth M. Hargreaves, DDS, PhD, Department of Endodontics, Mail Code 7892, University of Texas Health Science Center at San Antonio, 7703 Floyd Curl Drive, San Antonio, Texas, 78229-3900.

References / 1. Keiser K, Hargreaves KM, Building effective strategies for the management of endodontic pain. *Endodontic Topics* 3:93-105, 2003.

2. Seltzer S, Hargreaves KM, Differential Diagnosis of Odontalgia. In: Hargreaves KM, Goodis HE, eds. Seltzer and Bender's Dental Pulp. Chicago: *Quintessence*, 449-68, 2002.

3. Schwartz S, Cohen S, The difficult differential diagnosis. *Dent Clin North Am* 36:279-923, 1992.

4. Aiken A, Facial pain — toothache or tumour? *Int J Oral Surg* 10 (Suppl 1):187-90, 1981.

5. Bavitz JB, Patterson DW, Sorensen S, Non-Hodgkin's lymphoma disguised as odontogenic pain. J Am Dent Assoc 123:99-100, 1992.

6. Eversole L, Nonodontogenic facial pain and endodontics: pain syndromes of the jaws that simulate odontalgia. In: Cohen S, Burns R, eds. Pathways of the Pulp. St Louis:Mosby, 51-9, 1994.

7. Holland GR, Differential diagnosis of orofacial pain. In: Walton R, Torabinejad M, eds. Principles and Practice of Endodontics. Philadelphia: Saunders, 520-32, 2002.

8. Hasselgren G, Reit C, Emergency pulpotomy: pain relieving effect with and without the use of sedative dressings. *J Endodon* 15:254, 1989.

9. Oguntebi BR, DeSchepper EJ, et al. Postoperative pain incidence related to the type of emergency treatment of symptomatic pulpitis. *Oral Surg Oral Med Oral Path* 73:479, 1992.

10. Penniston SG, Hargreaves KM, Evaluation of periapical injection of Ketorolac for management of endodontic pain. *J Endodon* 22:55, 1996.

11. Torabinejad M, Cymerman JJ, et al. Effectiveness of various medications on postoperative pain following complete instrumentation. *J Endodon* 20:345, 1994.

12. Marshall JG, Liesinger AW, Factors associated with endodontic post-treatment pain. *J Endodon* 19:573, 1993.

13. Moos HL, Bramwell JD, Roahen JO, A comparison of pulpectomy alone versus pulpectomy with trephination for the relief of pain. *J Endodon* 22:422, 1996.

14. Rosenberg PA, Babick PJ, et al. The effect of occlusal reduction on pain after endodontic instrumentation. *J Endodon* 24:492, 1998.

15. Rogers M, Johnson B, et al. Comparison of effect of intracanal use of ketorolac tromethamine and dexamethasone with oral ibuprofen on post treatment endodontic pain. *J Endodon* 25:381, 1999.

16. Doroshak A, Bowles W, Hargreaves K, Evaluation of the combination of flurbiprofen and tramadol for management of endodontic pain. *J Endodon* 25:660, 1999.

17. Al-Belasy FA, Hairam AR, The efficacy of azithromycin in the treatment of acute infraorbital space infection. *J Oral Maxillofac Surg* 61(3):310-6, 2003.

18. Menke ER, Jackson CR, et al. The effectiveness of prophylactic etodolac on postendodontic pain. *J Endodon* 26:712-5, 2000.

19. Curtis P Jr, Gartman LA, Green DB, Utilization of ketorolac tromethamine for control of severe odontogenic pain. *J Endodon* 20:457-9, 1994.

20. Gopikrishna, V, Parameswaran, A, Effectiveness of prophylactic use of rofecoxib in comparison with ibuprofen on postendodontic pain. *J Endodon* 29:62-4, 2003.

21. Nakanishi, T Shimizu, H Hosokawa, Y, Matsuo, T. An immunohistological study on cyclooxygenase-2 in human dental pulp. *J Endodon* 27:385-8, 2001.

22. http://www.jr2.ox.ac.uk/bandolier/booth/ painpag/Acutrev/Analgesics/lftab.html)

23. Holstein A, Hargreaves KM, Niederman R, Evaluation of NSAIDs for treating post-endodontic pain: a systematic review. *Endodontic Topics* 3:3-13, 2002

24. Catella-Lawson F, Reilly MP et al.

Cyclooxygenase inhibitors and the antiplatelet effects of aspirin. *New England Journal of Medicine* 345(25):1809-17, 2001.

25. MacDonald TM, Wei L, Effect of ibuprofen on cardioprotective effect of aspirin. *Lancet* 361: 573-574, 2003.

26. Cooper S, The relative efficacy of ibuprofen in dental pain. *Compend Contin Educ Dent* 7:578, 1986.

27. Breivik E, Barkvoll P, Skovlund E, Combining diclofenac with acetaminophen or acetaminophen-codeine after oral surgery: a randomized, double-blind, single oral dose study. *Clin Pharm Therap* 66:625-30, 2000.

28. Hargreaves KM, Keiser K, Local Anesthetic Failure in Endodontics: Mechanisms and Management. *Endodontic Topics* 1:26-39, 2002.

29. Huang JH, Thalhammer JG et al. Susceptibility to lidocaine of impulses in different somatosensory afferent fibers of rat sciatic nerve. *J Pharmacol Experimental Therap* 282(2):802-11, 1997.

30. Nusstein J, Reader A, Local anesthesia for endodontic pain. *Endodontic Topics* 3:14-30, 2002.

31. Nusstein J, Reader A, et al. Anesthetic efficacy of the supplemental intraosseous injection of 2% lidocaine with 1:100,000 epinephrine in irreversible pulpitis. *J Endodon* 24(7):487-91, 1998.

32. Replogle K, Reader A, et al. Cardiovascular effects of intraosseous injections of 2% lidocaine with 1:100,000 epinephrine and 3% mepivacaine. *J Am Dent Assoc* 130(5):649-57, 1999.

33. Fouad AF, Rivera EM, Walton RE, Penicillin as a supplement in resolving the localized acute apical abscess. *Oral Surg Oral Med Oral Path* 81:590, 1996.

34. Walton RE, Chiappinelli J, Prophylactic penicillin: effect on post-treatment symptoms following root canal treatment of asymptomatic periapical pathosis. *J Endodon* 19:466, 1993.

35. Henry M, Reader A, Beck M, Effect of penicillin on postoperative endodontic pain and swelling in symptomatic necrotic teeth. *J Endodon* 27(2):117-23, 2001.

36. Pickenpaugh L, Reader A, et al. Effect of prophylactic amoxicillin on endodontic flare-up in asymptomatic, necrotic teeth. *J Endodon* 27(1):53-6, 2001.



NONSURGICAL ENDODONTIC RETREATMENT

Clifford J. Ruddle, DDS

ABSTRACT

The purpose of this clinical article is to emphasize that although there is enormous potential for endodontic success, clinicians are, at times, confronted with posttreatment disease. A rationale for endodontic treatment is followed by the goals of nonsurgical retreatment. The focus of this article is to identify the various nonsurgical retreatment categories and provide an overview of the concepts, armamentarium and techniques available to disassemble roots, address deficiencies or repair defects that are pathological or iatrogenic in origin.

here has been a significant growth in endodontic treatment in recent years. This increase in clinical activity can be attributable to bettertrained dentists and specialists

alike. Necessary for this unfolding story is the general public's growing interest for root canal treatment as an alternative to the extraction.¹ Over time, patients have become more confident selecting endodontic treatment because of the changing perception that pain can be managed, techniques have improved and long-term success is achievable. During the last decade, significant procedural refinements have created greater promise for our profession to fulfill the public's growing expectations for long-term success. With all the potential for endodontic success, the fact remains clinicians are confronted with post-treatment disease. This article focuses on the concepts, strategies, and techniques that will produce successful results in nonsurgical endodontic retreatment.

Rationale for Retreatment

The root canal system anatomy plays a significant role in endodontic success and failure.²⁻⁴ These systems contain branches that communicate with the periodontal attachment apparatus furcally, laterally, and often terminate apically into multiple portals of exit.⁵ Consequently, any opening from the root canal system (RCS) to the periodontal ligament space should be thought of as a portal of exit (POE) through which potential irritants may pass.^{6,7} Improvement in the diagnosis and treatment of lesions of endodontic origin (LEO) occurs with the recognition of the interrelationships between pulpal disease flow and the egress of irritants along these anatomical pathways (**Figure 1**).⁸

Endodontic failures can be attributable to inadequacies in shaping, cleaning and obturation, iatrogenic events, or reinfection of the root canal system when the coronal seal is lost after completion of root canal treatment.⁹⁻¹² Regardless of the etiology, the sum of all causes is leakage and bacterial contamination.^{13,14} Except in rare instances, lesions of endodontic origin will routinely heal following the extraction of



Author / Clifford J. Ruddle, DDS, is founder and director of Advanced Endodontics, an international educational source, in Santa Barbara, Calif. He is an assistant professor of Graduate Endodontics at Loma Linda University, and University of

California, Los Angeles; an associate clinical professor at University of California, San Francisco; and an adjunct assistant professor of Endodontics at University of the Pacific, School of Dentistry. He also is the author of two chapters in the 8th Edition of Pathways of the Pulp: Cleaning & Shaping the Root Canal System and Nonsurgical Endodontic Retreatment. He is internationally known for providing endodontic education through lectures, clinical articles, training manuals, videos and DVDs. Additionally, he maintains a private practice in Santa Barbara, Calif. pulpally involved teeth because the extraction not only removes the tooth, but more importantly serves to eliminate 100 percent of the contents of the root canal system. Endodontic treatment can approach 100 percent success discounting teeth that are non-restorable, have hopeless periodontal disease or have radicular fractures.⁸

Goals of Nonsurgical Endodontic Retreatment

Before commencing with any treatment, it is profoundly important to consider all interdisciplinary treatment options in terms of time, cost, prognosis and potential for patient satisfaction. Endodontic failures must be evaluated so a decision can be made between nonsurgical retreatment, surgical retreatment, or extraction.¹⁵⁻¹⁷ The goals of nonsurgical retreatment are to remove materials from the root canal space and if present, address deficiencies or repair defects that are pathologic or iatrogenic in origin.¹⁸ Additionally, nonsurgical retreatment procedures confirm mechanical failures, previously missed canals or radicular subcrestal fractures. Importantly, disassembly and corrective procedures allow clinicians to shape canals and threedimensionally clean and fill root canal systems.^{19,20} Nonsurgical endodontic retreatment procedures have enormous potential for success if the guidelines for case selection are respected and the most relevant technologies, best materials and precise techniques are utilized.²¹⁻²³

Coronal Access

Clinicians typically access the pulp chamber through an existing restoration if it is judged to be functionally designed, well fitting and esthetically pleasing.²⁴ If the restoration is deemed inadequate and/or additional access is required, then it should be sacrificed. However, on specific occasions, it may be desirable to remove the restoration intact so it can be re-cemented following endodontic treatment.¹⁸ Several important technologies exist which facilitate the safe removal of a restoration. Coronal disassembly improves access, vision and the retreatment efforts.

The safe dislodgment of a restoration is dependent on several factors such as the type of preparation, the restorative design and strength, the restorative material(s), the cementing agent and knowing how to use the best removal devices. There are several important removal devices which may be divided into three categories: (1) Grasping instruments, such as K.Y. Pliers (GC America; Alsip, Ill.) and Wynman Crown Gripper (Miltex Instrument Company; Lake Success, N.Y.), (2) Percussive instruments like the Peerless Crown-a-Matic (Henry Schein; Port Washington, N.Y.) and the Coronaflex (KaVo America; Lake Zurich, Ill.), and (3) Passive-active instruments such as the Metalift (Classic Practice



Figure 1a. A pre-operative film shows the maxillary right first molar's remaining palatal root is endodontically failing.



Figure 1b. Nonsurgical retreatment demonstrates a mesiocrestal lateral canal, a loop and an apical bifidity.



Figure 2a. A photograph demonstrates removal of a crown utilizing the K.Y. Pliers. Note the grasping pads have been dipped in emery powder to reduce slippage.



Figure 2b. A photograph demonstrates bridge removal utilizing the Coronaflex. The air-driven hammer generates the removal force against various prosthetic attachment devices.



Figure 2c. A photograph demonstrates the removal of a PFM crown utilizing the Metalift. This system applies a force between the crown and the tooth.

Resources; Baton Rouge, La.), the Kline Crown Remover (Brasseler; Savannah, Geo.) and the Higa Bridge Remover (Higa Manufacturing; West Vancouver, BC, Canada). Clinicians must clearly define the risk versus benefit with patients before commencing with the safe and intact removal of an existing restoration (**Figure 2**).

Missed Canals

Missed canals hold tissue, and at times bacteria and related irritants that inevitably contribute to clinical symptoms and lesions of endodontic origin.9 Oftentimes, surgical treatment has been directed towards "corking" the end of the canal with the hopes that the rootend filling material will incarcerate irritants within the root canal system over the life of the patient.¹⁴ Although this clinical scenario occurs anecdotally, it is not as predictable as nonsurgical retreatment. Endodontic prognosis is maximized in teeth whose root canals are shaped and root canal systems cleaned and filled in all their dimensions (Figure 3).^{5,8}

There are multiple concepts, armamentarium and techniques that are useful to locate canals. The most reliable method for locating canals is to have knowledge regarding root canal system anatomy and appreciation for the range of variation commonly associated with each type of tooth.³ Frequently used methods for identifying canals include: radiographic analysis, magnification and lighting (microscopes), complete access, firm explorer pressure, ultrasonics, Micro-Openers (Dentsply Tulsa Dental; Tulsa, Okla.), dyes, sodium hypochlorite, color and texture, removing restorations, and probing the sulcus. However, if a missed canal is suspected but cannot be readily identified and treated, then an endodontic referral may be prudent to avoid complications. Caution should be exercised when contemplating surgery due to the aforementioned concerns.

Obturation Materials

There are four commonly encountered obturation materials found in root canals. These materials are gutta-percha, carrier-based obturators, silver points and paste fillers. Generally, it is necessary to remove an obturation material to achieve endodontic retreatment success or to facilitate placing a post for restorative reasons. The effective removal of an obturation material requires utilizing the most proven methods from the past in conjunction with the best presently developed techniques.

Gutta-Percha Removal

The relative difficulty in removing gutta-percha varies according to the obturation technique previously employed and further influenced by the canal's length, cross-sectional dimension, curva-

and internal configuration. ture Regardless of technique, gutta-percha is best removed from a root canal in a progressive manner to prevent inadvertent displacement of irritants periapically. Dividing the root into thirds, gutta-percha may be initially removed from the canal in the coronal one-third, then the middle one-third, and finally eliminated from the apical one-third. At times, single cones in larger and straighter canals can be removed with one instrument in one motion. For other canals, there are a number of possible guttapercha removal schemes.18 The removal techniques include rotary files, ultrasonic instruments, heat, hand files with heat or chemicals, and paper points with chemicals.²⁵ Of these options, the best technique(s) for a specific case is selected based on preoperative radi-



Figure 3a. A radiograph of a maxillary right second bicuspid reveals pins, a post, incomplete endodontics and an asymmetrical lesion.



Figure 3b. A photograph at 12x shows the post is out of the buccal canal, thread marks in the gutta-percha from the screw post, and evidence of a missed palatal canal.



Figure 3c. A photograph at 12x demonstrates complete access and identification of the orifice of palatal canal.



Figure 3d. A 10-year recall radiograph shows excellent osseous repair, the importance of good quality endodontics, and a well-designed and sealed restoration.





Figure 4a. A preoperative radiograph of a maxillary right central incisor demonstrates inadequate root canal treatment, resorption and an apical lesion.



Figure 4b. A photograph at 8x shows a 45 hedstroem mechanically removing the heat soft-ened single cone of gutta-percha.

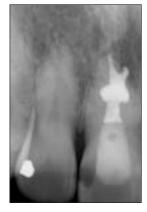


Figure 4c. A postoperative radiograph shows the nonsurgical retreatment has resulted in threedimensional obturation.

chronic leakage reduces the seal and hence, the lateral retention. Access preparations must be thoughtfully planned and carefully performed to minimize the risk of inadvertently foreshortening any given silver point. Initial access is accomplished with high speed, surgical-length cutting tools, then oftentimes ultrasonic instruments are used to brush-cut away remaining restorative materials and fully expose the silver point.

Different techniques have been developed for removing silver points depending on their lengths, diameters, and positions they occupy within the root canal space (Figure 5).^{23,26,27} Certain removal techniques evolved to address silver points that bind in unshaped canals over distance. Other techniques arose to remove silver points with large cross-sectional diameters, approaching the size of smaller posts. Finally, other techniques are necessary to remove intentionally sectioned silver points lying deep within the root canal space. The more effective methods for removing silver points include: grasping pliers utilizing the principles of fulcrum mechanics, indirect ultrasonics, files, solvents, chelators, the hedstroem displacement technique, tap and thread option using the microtubular taps from the Post Removal System kit (SybronEndo; Orange, Calif.), and microtube mechanics such as the Instrument Removal System (Dentsply Tulsa Dental).¹⁸



Figure 5a. A preoperative radiograph depicting an endodontically failing maxillary central incisor bridge abutment, a gutta-percha point tracing a sinus tract to a large lateral root lesion, and a canal underfilled and slightly overextended.



Figure 5b. Magnification at 15x reveals lingual access and restorative build-up around the coronal-most aspect of the exposed silver point.



Figure 5c. A working radiograph during obturation reveals complexity of the root canal system.



Figure 5d. A five-year postoperative radiograph demonstrates that three-dimensional endodontic treatment has resulted in complete healing.

ographs, clinically assessing the available diameter of the orifices after reentering the pulp chamber, and clinical experience. Certainly, a combination of methods are generally required and, in concert, provide safe, efficient and potentially complete elimination of gutta-percha and sealer from the internal anatomy of the root canal system (**Figure 4**).

Silver Point Removal

The relative ease of removing a silver point is based on the fact that



Figure 6a. A radiograph of a maxillary left first molar demonstrates "Coke bottle" preparations and carrier-based obturation of three canals.



Figure 6b. A post-operative radiograph reveals the retreatment efforts, including the identification and treatment of a second MB root canal system.



Figure 7a. A pre-operative radiograph of an endodontically failing paste-filled mandibular left second molar. Note the extra distal root.

Carrier Removal

Gutta-percha carriers were originally metal and file-like, yet over the past several years they have been manufactured from easier to remove plastic carriers that have a longitudinal groove. Metal carriers, although no longer distributed, are occasionally encountered clinically and can be more difficult to remove than silver points because their cutting flutes at times engage lateral dentin.²⁸ Successful removal is enhanced by recognizing that the carrier is embedded in hardened gutta-percha and sealer. The successful removal of carrier-based obturators utilizes the same techniques described for removing gutta-percha and silver points.18 However, successful removal poses additional challenges to the aforementioned obturation techniques in that the clinician must remove *both* the gutta-percha and the



Figure 7b. A five-year recall film shows the treatment of multiple apical portals of exit and excellent osseous healing.

carrier (**Figure 6**). Oftentimes, the biggest secret to remove a carrier is to appreciate the importance of first removing circumferential gutta-percha that will facilitate removing the carrier-based obturator.

Paste Removal

When evaluating a paste case for retreatment, it is useful to clinically understand that pastes can generally be divided into soft, penetrable and removable versus hard, impenetrable, and at times, unremovable. Fortuitously, the paste is denser in the coronal portion of the canal and the material is progressively less dense moving apically due to the method of placement (**Figure 7**). Before retreating a paste-filled canal, the clinician should anticipate calcifications, resorptions, and the possibility that the removal efforts may be unsuccessful. Importantly, patients should be advised there could be a higher incidence of flare-ups associated with these retreatment cases.

An excellent technique for the safe removal of hard, impenetrable paste from the straightaway portion of a canal utilizes abrasively coated ultrasonic instruments in conjunction with the microscope. To remove paste apical to canal curvature, hand instruments should first be utilized to establish or confirm a safe glide path. Pre-curved stainless-steel hand files may be inserted into this secured region of the canal and when attached to a "file adapter," may be activated utilizing ultrasonic energy.¹⁸ Other removal methods include heat, judicious use of end-cutting rotary NiTi instruments and small sized hand files with solvents such as Endosolv R and Endosolv E (Endoco; Memphis, Tenn.).²⁹ Additionally, Micro-Debriders (Dentsply Maillefer; Tulsa, Okla.) and paper points in conjunction with solvents play an important role in removing paste from canal irregularities.

Post Removal

Endodontically treated teeth frequently contain posts that need to be removed to facilitate successful nonsurgical retreatment.³⁰ Factors that will influence post removal are post diameter, length and the cementing agent. Other factors that will influence removal are whether the post is parallel versus tapered, stock versus cast, actively engaged versus non-actively retained, metallic versus non-metallic compositions, and the post head configuration. Additionally, other considerations include available interocclusal space, existing restorations and if the post head is supra- or sub-crestal. Over time, many techniques have been advocated for removal of posts.³¹ Before initiating any post removal method, all materials circumferential to the post must be eliminated and the orifice to the canal visualized (Figure 8).

Retreatment

Ultrasonic Option

The first line of offense to remove a post is to utilize piezoelectric ultrasonic energy. An ultrasonic generator in conjunction with the correct insert instrument will transfer energy, powerfully vibrate, and dislodge most posts. A frequent and intermittent air/water spray is directed on the post to reduce heat buildup and transfer during ultrasonic removal procedures. The majority of posts can be safely and successfully removed with ultrasonics in about 10 minutes.³²

PRS Option

The Post Removal System (PRS) is a reliable method to remove a post when ultrasonic efforts using the 10-Minute *Rule* prove unsuccessful.¹⁸ In this removal method, a trephine is used to machine down the most coronal aspect of the post 2 mm to 3 mm. The correspondingly sized tap is selected and an appropriately sized protective bumper is inserted onto this instrument. The tap is turned in a counter-clockwise direction to form threads and securely engage the post head. Once the tap is firmly engaged on the post and the protective bumper seated, then the extracting pliers are used to safely and progressively elevate the post out of the canal.

Separated Instrument Removal

Technological advancements have significantly increased the predictability in removing separated instruments. These advancements include the dental operating microscope, ultrasonic instrumentation, and microtube delivery methods.¹⁸ The ability to access and remove a separated instrument will be influenced by the cross-sectional diameter, length and curvature of the canal, and further guided by root bulk and form including the depth of external concavities. In general, if one-third of the overall length of an obstruction can be exposed, it can usually be removed. Instruments that lie in the straightaway portions of the canal or partially around the curvature can usually be removed if safe access can be established to its most coronal extent.^{33,34} If the entire segment of the separated instrument is apical to the curvature of the canal and safe access cannot be accomplished, then removal is usually not possible.

The techniques required to remove a separated instrument begin with establishing straight line coronal access. To create radicular access, hand files may be used serially small to large, coronal to the obstruction, to create sufficient space to safely introduce gates glidden (GG) drills. GG's are used like "brushes" and at a reduced speed of about 750 rpm. Importantly, in multi-rooted teeth, GG's may be used from small to large to cut and remove dentin on the outer wall of the canal and away from furcal danger. Each larger GG is stepped out of the canal to create a uniform tapered and smooth flowing funnel. The goal of radicular access is to optimally prepare a canal no larger than if there was no separated instrument.

Ultrasonic Option

In combination, microscopes and ultrasonics have driven "microsonic" techniques that have improved the potential, predictability and safety when removing separated instruments.¹⁸ When access and visibility to the head of the separated instrument are achieved then contra-angled, parallel-walled and abrasively-coated ultrasonic instruments (ProUltra Endo Tips No. 3, 4, 5) may be employed. When energized, these instruments may be used to precisely sand away dentin and trephine circumferentially around the obstruction. During the ultrasonic pro-



Figure 8a. A pre-operative radiograph of a mandibular right second molar bridge abutment demonstrates three posts, previous endodontics, and apical pathosis.



Figure 8b. Following coronal disassembly, the isolated tooth reveals the core sectioned and reduced.



Figure 8c. The pulpal floor is shown following three-dimensional cleaning, shaping, and obturation procedures. Note the displaced most lingual orifice.



Figure 8d. A mesially angulated postoperative radiograph confirms the disassembly and retreatment efforts.



Figure 9a. A pre-operative radiograph shows a strategic and endodontically failing mesial root of a mandibular left first molar. Note a short screw post, a separated instrument and amalgam debris from the hemisection procedures.



Figure 9b. A photograph shows the splint removed, the post out and an ultrasonic instrument trephining around the broken file.



Figure 9c. An eight-year recall film demonstrates three-dimensional retreatment, a new bridge and excellent periradicular healing.



10a. A preoperative radiograph of a maxillary canine demonstrates a temporized canal with an instrument separated deep in the apical one-third.

Figure



Figure 10b. A working film shows that the 21-gauge iRS has successfully engaged and partially elevated the deeply positioned file segment.



Figure 10c. A postoperative film demonstrates the retreatment steps and a densely filled system that exhibits three apical portals of exit.

cedure, the separated instrument will typically loosen, unwind and spin, then "jump out" of the canal (**Figure 9**).

IRS Option

When ultrasonic techniques fail, the fall-back option is to use the Instrument Removal System (iRS) (Dentsply Tulsa Dental). The iRS is composed of variously sized microtubes and screw wedges. Each microtube has a small handle to enhance vision and its distal end is constructed with a 45-degree beveled end and side window. The appropriately sized microtube is inserted into the canal and, in the instance of canal curvature, the long part of its beveled end is oriented to the outer wall of the canal to "scoop-up" the head of the broken instrument and guide it into its lumen. The screw wedge is then placed through the open end of the microtube and passed down its internal lumen until it contacts the separated instrument. Rotating the screw wedge handle tightens, wedges, and oftentimes, displaces the head of the file through the microtube's side window.¹⁸ With the separated instrument strongly engaged, it can generally be rotated counter-clockwise and removed (**Figure 10**).

Blocks, Ledges, Transportations and Perforations

Failure to respect the biological and mechanical objectives for shaping canals and cleaning root canal systems predisposes to needless complications such as blocks, ledges, external transportations and perforations. These iatrogenic events can be attributable to working short, the sequence utilized for preparing the canal, and the instruments and their method of use.¹⁹

Techniques for Managing Blocks

Techniques for managing blocked canals begin by confirming straight line access and then pre-enlarging the canal coronal to the obstruction.18 A 10 file provides rigidity and is pre-curved to simulate the expected curvature of the canal. The unidirectional rubber stop is oriented to match the file curvature. With the pulp chamber filled with a viscous chelator, efforts are directed toward gently sliding the 10 file to length. If unsuccessful, the file is used with an apically directed picking action while concomitantly re-orienting the unidirectional stop which serves to redirect the apical aspect of the pre-





Figure 11a. A pre-operative radiograph of a maxillary left second bicuspid reveals previous access and pre-enlargement of the canal in its coronal two-thirds.



Figure 11b. The post-operative radiograph provides an explanation as to the etiology of the original block. Note the canal bifurcates apically and this system has four portals of exit.



Figure 12a. A pre-operative radiograph shows an endodontically failing posterior bridge abutment. Note the amalgam in the pulp chamber and that the mesial root appears to have been ledged.

curved file. Short amplitude, light pecking strokes are best utilized to ensure safety, carry reagent deeper, and increase the possibility of canal negotiation. If the apical extent of the file "sticks" or engages, then it may be useful to move to a smaller sized hand file. A working film should be taken and the file frequently removed to see if its curve is following the expected root canal morphology. Depending on the severity of the blockage, perseverance will oftentimes allow the clinician to safely reach the foramen and establish patency (Figure 11). If the blocked canal is not negotiable, then the case should be filled utilizing a hydraulic warm gutta-percha technique. Regardless of the filling result, the patient needs to be advised of the importance of recall and that future treatment options



Figure 12b. A post-treatment film demonstrates ledge management with the obturation materials following the root curvature.

include surgery, re-implantation, or extraction.

Techniques for Managing Ledges

An internal transportation of the canal is termed a "ledge" and frequently results when clinicians work short of length and "get blocked." Ledges are typically on the outer wall of the canal curvature and are oftentimes bypassed using the techniques described for blocks.^{13,18} Once the tip of the file is apical to the ledge, it is moved in and out of the canal utilizing ultra-short pushpull movements with emphasis on staying apical to the defect. When the file moves freely, it may be turned clockwise upon withdrawal to rasp, reduce, smooth or eliminate the ledge. During these procedures, try to keep the file

coronal to the terminus of the canal so the apical foramen (foramina) is handled delicately and kept as small as practical. When the ledge can be predictably bypassed, then efforts are directed toward establishing patency with a 10 file. Gently passing a .02 tapered 10 file 1 mm through the foramen ensures its diameter is at least 0.12 mm and paves the way for the 15 file.²²

A significant improvement in ledge management is the utilization of nickeltitanium (NiTi) hand files that exhibit tapers greater than ISO files.¹⁸ Certain NiTi instruments have multiple increasing tapers over the length of the cutting blades on the same instrument (ProTaper, Dentsply Tulsa Dental). Progressively tapered NiTi files can be introduced into the canal when the ledge has been bypassed, the canal negotiated and patency established. Bypassing the ledge and negotiating the canal up to a size 15, and if necessary to a 20 file, creates a pilot hole so the tip of the selected NiTi instrument can passively follow this glide path. To move the apical extent of a NiTi hand file past a ledge, the instrument must first be pre-curved with a device such as Bird Beak orthodontic pliers (Hu-Friedy; Chicago, Ill.). Ultimately, the clinician must make a decision based on pre-operative radiographs, root bulk and experience whether the ledge can be eliminated through instrumentation or if these procedures will weaken or perforate the root. Not all ledges can or should be removed. Clinicians must weigh risk versus benefit and make every effort to maximize remaining dentin (Figure 12).

Techniques for Managing Apical Transportations

A canal that has been transported exhibits reverse apical architecture and predisposes to poorly packed canals that are oftentimes vertically overextended but internally underfilled.^{8,13} In these instances, a barrier/restorative can be selected to control bleeding and provide a backstop to pack against during subsequent obturation procedures. The barrier of choice for a transportation is generally mineral trioxide aggregate (MTA) (Dentsply Tulsa Dental), commercially known as ProRoot. MTA is an extraordinary material which can be used in canals which exhibit reverse apical architecture, such as in transportations or immature roots, nonsurgical perforation repairs, or in surgical repairs.^{18,35,36} Remarkably, cementum oftentimes grows over this nonresorbable and radiopaque material, thus allowing for a normal periodontal attachment apparatus.³⁷⁻³⁹ Although a dry field facilitates visual control, MTA is apparently not compromised by moisture and typically sets hard within four to six hours, creating a seal as good as or better than other materials.40-42

Techniques for managing apical transportations are facilitated when the coronal two-thirds of the canal is optimally prepared and radicular access is available for placing a barrier. ProRoot is easy to use and the powder is mixed with sterile water to a heavy cake-like consistency. MTA may be picked up and efficiently carried into more superficial regions of the tooth on the side of a West Perf Repair Instrument (SybronEndo). To more precisely introduce MTA deep into a prepared canal microtube carrying devices or the Lee carrier method (G. Hartzell & Sons; Concord, Calif.) are appropriately sized to accomplish this task.^{18,35,43} ProRoot is then gently tamped down the canal to approximate length using a customized nonstandard gutta-percha cone as a flexible plugger. In straighter canals, ProRoot can be gently vibrated, moved into the defect and adapted to the canal walls with ultrasonic instruments (ProUltra Endo Tips, Dentsply Tulsa Dental) Direct ultrasonic energy will vibrate and generate a wave-like motion which facilitates moving and adapting



Figure 13a. A preoperative film of the maxillary right central incisor bridge abutment depicts a post and an empty system that exhibits reverse apical architecture.



Figure 13b. A photograph shows tamping MTA into the apical one-third with a gutta-percha cone used as a flexible plugger.



Figure 13c. A photograph shows the ProUltra ENDO-5 ultrasonic instrument vibrating MTA densely into the apical one-third.

the cement into the apical extent of the canal. Prior to initiating subsequent procedures, a dense 4 mm to 5 mm zone of ProRoot in the apical one-third of the canal should be confirmed radiographically (**Figure 13**).

In the instance of repairing a defect apical to the canal curvature, ProRoot is incrementally placed deep into a canal then shepherded around the curvature with a flexible, trimmed gutta-percha cone utilized as a plugger. A pre-curved 15 or 20 stainless-steel file is then inserted into the ProRoot and to within 1 mm to 2 mm of the working length. Indirect ultrasonics involves placing the working end of an ultrasonic instrument, such as the ProUltra Endo Tip No. 1, on the shaft of the file. This vibratory energy will encourage ProRoot to move and conform to the configurations of the



Figure 13d. A six-year recall demonstrates a new bridge, post and excellent osseous repair.

canal laterally as well as control its movement to and gently against the periapical tissues. Again, the clinician should radiographically confirm that there is a dense 4 mm to 5 mm zone of ProRoot in the apical extent of the canal.

MTA needs moisture to set and become hard. Fluids are present external to the canal and will fulfill the moisture requirement for the apical aspect of the positioned MTA. However, a cotton pellet or paper point will need to be sized, moistened with water, and placed against the coronal most aspect of the MTA that is within the canal. The tooth is then temporized and the patient dismissed. At a subsequent appointment, the temporary filling and wet cotton pellet are removed so the MTA can be probed with an explorer to determine if







Figure 14a. A pre-operative radiograph of an endodontically involved mandibular left second molar bridge abutment. Note the previous access and possible floor perforation.

Figure 14b. A photograph demonstrates the identified orifices and a frank furcal floor perforation.



Figure 14c. This photograph shows the perforation repair utilizing a calcium sulfate resorbable barrier and a dual cured composite restorative.

it has set-up and is hard. Typically, the material is hard and the clinician can then obturate against this nonresorbable barrier. If the material is soft, it should be removed, the area flushed, dried, and a new mix of MTA placed. On a subsequent visit, when the inflammatory process has subsided, then a hard barrier should exist which will provide a backstop to pack against.

Techniques for Managing Perforations

A perforation represents a pathologic or iatrogenic communication between the root canal space and the attachment apparatus. The causes of perforations are resorptive defects, caries, or iatrogenic events that occur during and after endodontic treatment. Regardless of etiology, a perforation is an invasion into



Figure 14d. A five-year recall film shows a new bridge and osseous repair furcally and apically.

the supporting structures that initially incites inflammation and loss of attachment and ultimately may compromise the prognosis of the tooth. When managing these defects the prognosis will be impacted by the level, location and size of the perforation, and further influenced by its timely repair.¹⁸

Techniques and materials for managing perforation defects have been described earlier under the heading "Techniques for Managing Transportations." However, on occasion, toothcolored restoratives may be the material of choice for repairing certain perforations. Tooth-colored restoratives, such as a dual cured composite, require the placement of a barrier so the material is not contaminated during use. A barrier serves as a "hemostatic" and a "backstop" so a restorative material can be placed into a clean, dry preparation with control. Calcium sulfate is an excellent absorbable barrier material when using the principles of wet bonding because it is biocompatible, osteogenic, and following placement, sets brick-hard.⁴⁴⁻⁴⁶ When set, calcium sulfate is internally trimmed back to the cavo surface of the root. A dual cured, tooth-colored restorative can now be placed against the barrier and utilized to seal a root defect (**Figure 14**).

Conclusion

This article has identified a variety of techniques to successfully retreat endodontically failing teeth. It should be recognized certain endodontically failing teeth are not amenable to successful retreatment. In these instances, the various interdisciplinary treatment options can be thoughtfully considered to ensure each patient is best served. However, as the potential for health associated with endodontically treated teeth becomes fully appreciated, the naturally retained root will be recognized as the ultimate dental implant.

Summary

In the United States alone, tens of millions of teeth receive endodontic treatment annually. Regardless of the enormous potential for endodontic success, certain teeth exhibit post-treatment disease. Many endodontically failing teeth are either surgerized or extracted. This article emphasized the importance of case selection, interdisciplinary treatment planning and the role of nonsurgical endodontic retreatment in preserving strategic teeth. Properly performed, endodontic treatment is a cornerstone of restorative and reconstructive dentistry. CDA

References / **1.** Endodontic trends reflect changes in care provided, *Dental Products Report*, 30(12):94-8, 1996.

2. Scianamblo MJ, Endodontic failures: the retreatment of previously endodontically treated teeth, *Revue D'Odonto Stomatologie* 17(5):409-23, 1988.

3. Hess W, Zürcher E, *The Anatomy of the Root Canals of the Teeth of the Permanent and Deciduous Dentitions*, William Wood & Co, New York, 1925.

4. Ruddle CJ, Endodontic failures: the rationale and application of surgical retreatment, *Revue D'Odonto Stomatologie* 17(6):511-69, 1988.

5. Schilder H, Cleaning and shaping the root canal system, *Dent Clin North Am* 18(2):269-96, 1974.

6. Barkhordar RA, Stewart GG, The potential of periodontal pocket formation associated with untreated accessory root canals, *Oral Surg Oral Med Oral Pathol* 70(6), 1990.

7. DeDeus QD, Frequency, location and direction of the accessory canals, *J Endod* 1:361-6, 1975.

8. Schilder H, Filling root canals in three dimensions, *Dent Clin North Am* 723-44, November 1967.

9. West JD, The relation between the threedimensional endodontic seal and endodontic failure, *master thesis*, Boston University, 1975

10. Torabinejad M, Ung B, Kettering JD, In vitro bacterial penetration of coronally unsealed endodontically treated teeth. *J Endod* 16(12):566-9, 1990.

11. Alves J, Walton R, Drake D, Coronal leakage: endotoxin penetration from mixed bacterial communities through obturated, post-prepared root canals, *J Endod* 24(9):587-1, 1998.

12. Southard DW, Immediate core buildup of endodontically treated teeth: the rest of the seal, *Pract Periodont Aesthet Dent* 11(4):519-26, 1999.

13. Ruddle CJ, Nonsurgical endodontic retreatment, *J Calif Dent Assoc* 25(11):765-800, 1997.

14. Ruddle CJ, Surgical endodontic retreatment, *J Calif Dent Assoc* 19(5):61-7, 1991.

15. Stabholz A, Friedman S, Endodontic retreatment- case selection and technique. Part 2: treatment planning for retreatment. *J Endod* 14(12):607-14, 1988.

16. Allen RK, Newton CW, Brown CE, A statistical analysis of surgical and nonsurgical endodontic retreatment cases, *J Endod* 15(6):261-6, 1989.

17. Kvist T, Reit C, Results of endodontic retreatment: a randomized clinical study comparing surgical and nonsurgical procedures, *J Endod* 25(12):814-7, 1999.

18. Ruddle CJ, Nonsurgical endodontic retreatment. In Cohen S, Burns RC, editors: *Pathways of the Pulp*, Ch. 25, 875-929, 8th ed., Mosby, St. Louis, 2002.

19. Ruddle CJ, Ch. 8, Cleaning and shaping root canal systems. In Cohen S, Burns RC, editors: *Pathways of the Pulp* 231-91, 8th ed., Mosby, St. Louis, 2002.

20. Ruddle CJ, Three-dimensional obturation: the rationale and application of warm gutta-percha with vertical condensation, *Pathways of the Pulp*, Ch. 9, 243-7, 6th ed., Mosby Co., St. Louis, 1994.

21. Blum JY, Machtou P, Ruddle CJ, Micallef JP, The analysis of mechanical preparations in extracted teeth using protaper rotary instruments: value of the safety quotient, *J Endod* 29(9):567-5, 2003.

22. Ruddle CJ, Nickel-titanium rotary instruments: current concepts for preparing the root canal system, *Australian Endodontic Journal* 29(2):87-8, 2003.

23. Ruddle CJ, Microendodontic nonsurgical retreatment, in *Microscopes in Endodontics, Dent Clin North Am* 41(3):429-54, W.B. Saunders, Philadelphia, July 1997.

24. Machtou P, La cavité d'accès. In Machtou P, editor: *Endodontie - guide clinique*, Ch. 8, 125-37, Editions CdP, Paris, 1993.

25. Wilcox LR, Krell KV, Madison S, Rittman B, Endodontic retreatment: evaluation of gutta-percha and sealer removal and canal reinstrumentation, *J Endod* 13(9):453-7, 1987.

26. Goon WWY, Managing the obstructed root canal space: rationale and techniques, *J Calif Dent Assoc* 19(5):51-60, 1991.

27. Glick DH, Frank AL, Removal of silver points and fractured posts by ultrasonics, *J Prosth Dent* 55:212-5, 1986.

28. Bertrand MF, Pellegrino JC, Rocca JP, Klinghofer A, Bolla M, Removal of Thermafil root canal filling material, *J Endod* 23(1):54-7, 1997.

29. Cohen AG, The efficiency of solvents used in the retreatment of paste-filled root canals, *masters thesis*, Boston University, 1986.

30. Machtou P, Sarfati P, Cohen AG, Post removal prior to retreatment, *J Endod* 15(11):552-4, 1989.

31. Stamos DE, Gutmann JL, Survey of endodontic retreatment methods used to remove intraradicular posts, *J Endod* 19(7):366-9, 1993.

32. Altshul JH, Marshall G, Morgan LA, Baumgertner JC, Comparison of dentinal crack incidence and of post removal time resulting from post removal by ultrasonic or mechanical force. *J Endod* 23(11):683-6, 1997.

33. Ward JR, Parashos P, Messer HH, Evaluation of an ultrasonic technique to remove fractured rotary nickel-titanium endodontic instruments from root canals: an experimental study, *J Endod* 29(11):756-3, 2003.

34. Ward JR, Parashos P, Messer HH, Evaluation of an ultrasonic technique to remove fractured rotary nickel-titanium endodontic instruments from root canals: clinical cases, *J Endod* 29(11):764-7, 2003.

35. Castellucci A, L'uso del mineral trioxide aggregate in endodonzia clinica e chirurgica, *L'informatore Endodontico* 6(3):34-5, 2003.

36. Torabinejad M, Chivian N, Clinical applications of mineral trioxide aggregate, *J Endod* 25:197-205, 1999.

37. Torabinejad M, Pitt Ford TR, Abedi HR, Kariyawasam SP, Tang HM, Tissue reaction to implanted potential root-end filling materials in the tibia and mandible of guinea pigs, *J Endod* 24:468-1, 1998.

38. Torabinejad M, Hong CU, Lee SJ, Monsef M, Pitt Ford TR, Investigation of mineral trioxide aggregate for root-end filling in dogs, *J Endod* 21:603-8, 1995.

39. Torabinejad M, Pitt Ford TR, McKendry DJ, Abedi HR, Miller DA, Kariyawasam SP, Histologic assessment of MTA as root-end filling in monkeys, *J Endod* 23:225-8, 1997.

40. Torabinejad M, Watson TF, Pitt Ford TR, The sealing ability of a mineral trioxide aggregate as a retrograde root filling material, *J Endod* 19:591-5, 1993.

41. Torabinejad M, Higa RK, McKendry DJ, Pitt Ford TR, Dye leakage of four root-end filling materials: effects of blood contamination, *J Endod* 20:159-3, 1994.

42. Torabinejad M, Rastegar AF, Kettering JD, Pitt Ford TR, Bacterial leakage of mineral trioxide aggregate as a root-end filling material, *J Endod* 21:109-21, 1995.

43. Lee SL, A new mineral trioxide aggregate root-end filling technique, *J Endod* 26(12):764-5, 2000.

44. Sottosanti J, Calcium sulfate: a biodegradable and biocompatible barrier for guided tissue regeneration, *Compend Contin Educ Dent* 13(3):226-4, 1992. **45.** Himel VT, Alhadainy HA, Effect of dentin preparation and acid etching on the sealing ability of glass ionomer and composite resin when used to repair furcation perforations over plaster of Paris barriers, *J Endod* 21(3):142-5, 1995.

46. Alhadainy HA, Abdalla AI, Artificial floor technique used for the repair of furcation perforations: a micro-leakage study, *J Endod* 24(1):33-5, 1998.

To request a printed copy of this article, please contact / Clifford J. Ruddle, DDS, 227 Las Alturas Road, Santa Barbara, Calif., 93103.



CONTEMPORARY ENDODONTIC SURGERY

Richard Rubinstein, DDS, MS; and Mahmoud Torabinejad, DMD, MSD, PhD

ABSTRACT

During the past decade, endodontics has seen a dramatic shift in the application of periradicular surgery and the role it plays in endodontic treatment. With the introduction of enhanced magnification, periradicular ultrasonics and other associative technologies, teeth that might otherwise be extracted now have a chance for retention. This article describes the role of these advances in contemporary endodontic surgery. onsurgical root canal therapy is a highly successful procedure if diagnosis and technical aspects are carefully performed. There is a common belief that if root canal thera-

py fails, surgery is indicated for correction. This is not always true; most failures are best corrected by retreatment. Studies have shown that a majority of retreated cases are successful following retreatment.¹⁻⁵ There are, however, situations in which surgery is necessary to retain a tooth that would otherwise be extracted. The purpose of this article is to briefly describe indications and contraindications as well as the steps involved and new advances in periradicular surgery. The details for this procedure can be found in surgical and nonsurgical endodontic text books.⁶⁻⁸

Indications and Contraindications for Periradicular Surgery

The main indications for periradicular surgery are: complex root canal anatomy, procedural accidents (**Figure** 1), irretrievable materials in the root canal (**Figure 2**), symptomatic cases, horizontal apical fracture, biopsy (**Figure 3**) and corrective surgery (**Figure 4**). Contraindications are relatively few. There are four major categories: (1) anatomic factors, (2) medical or systemic complications, (3) indiscriminate use of surgery, and (4) unidentified cause of treatment failure.

Steps in Periradicular Surgery

The typical sequence of procedures used in periradicular surgery are flap design, incision and reflection, apical access, periradicular curettage, root-end resection, root-end cavity preparation, root-end filling, flap replacement and suturing, postoperative care and instructions, suture removal and evaluation.

Recent Advances in Endodontic Surgery

Many advances in surgical technique and instrumentation have occurred over the past decade. They include enhanced magnification and illumination, ultrasonic tips, micro-



Authors / Richard Rubinstein, DDS, MS, is adjunct assistant professor in the Department of Endodontics at the University of Pennsylvania School of Dentistry and an adjunct clinical professor at the Rackham School of Graduate Studies at the University

of Michigan School of Dentistry in Ann Arbor, Mich. He has written numerous scientific articles on the surgical operating microscope and endodontic microsurgical technique; a contributing author to *Endodontics*, fourth and fifth editions, and *Microscopes in Endodontics in The Dental Clinics Of North America*.



Mahmoud Torabinejad, DMD, MSD, PhD, is a professor of endodontics and director of Graduate Endodontics at Loma Linda University School of Dentistry, and practices in Upland, Calif. He has coauthored two textbooks in nonsurgical and surgical endodontics as

well as numerous articles on endodontics and dental topics. He was certified as a diplomate of the American Board of Endodontists and taught at Harvard before joining Loma Linda University. He has received several awards including Ralph F. Somers, Louis I. Grossman, and the Philanthropist award of the AAE Foundation.



Figure 1a. A separated file is present in the mesial root of the first mandibular molar.



Figure 1b. Presence of symptoms required to perform an apical surgery and placement of MTA as a root-end filling material.



Figure 1c. Complete periradicular healing is observed three years following surgery.



Figure 2a. A failing root canal treatment required surgery.



Figure 2b. The root end is resected, a cavity is prepared and is filled with MTA.



Figure 2c. A post-operative film after one year showing complete healing.



Figure 3. Periradicular radiolucency simulates a periapical lesion of pulpal origin. A biopsy showed presence of squamous cell carcinoma. instruments and newer root-end filling materials.

Enhanced Illumination and Magnification Magnification and Illumination

Perhaps the most important development in surgical endodontics in recent years has been the introduction of the surgical operating microscope (SOM). A few years ago, a handful of endodontists throughout the United States and Europe began experimenting with the SOM to determine whether any of its applications could be used in endodontic surgery.⁹⁻¹⁴ They believed they could achieve better results if they were able to further magnify and illuminate the surgical field beyond what was available with conventional loupes and surgical headlamps. After using the SOM, they realized cases that once seemed impossible became easier and more exciting to operate. Otologists were the first medical specialists to introduce the SOM in the early 1940s. Initially, loupes seemed adequate, and emphasis was placed on improving their function. At that time, many clinicians felt the SOM would make highly successful operations complicated and time consuming. Over time, they recognized advantages such as wider fields, variable magnification, better depth of focus, and coaxial illumination. Slowly, the use of the SOM was introduced into ophthalmology, and finally into neurosurgery in 1967, when UCLA's Dr. Peter Jannetta devised a microscopic procedure called micro-vascular decompression to treat trigeminal neuralgia.¹⁵

Most dentists have had clinical expe-

Endodontic Surgery



Figure 4a. An off-centered post has perforated the root and has caused a bony lesion. Internal and external perforation repairs with MTA resulted in complete repair of the bony lesion in three years (C).



Figure 4b. Internal and external perforation repairs with MTA resulted in complete repair of the bony lesion in three years (C).

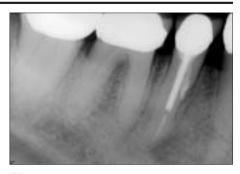


Figure 4c. Courtesy of Dr. Noah Chivian.

rience with conventional surgical telescopes or loupes and surgical headlamps, which are commonly available in a variety of configurations and magnifications. When a fiber optic headlamp system is added to the armamentarium, white light is projected coaxially with the line of sight into the surgical field, and surgical and nonsurgical endodontic procedures can be performed with less eyestrain and fatigue. Clinicians who have used surgical telescopes and surgical headlamps have benefited from the expanded use of magnification and illumination. But how much magnification is enough? Most microscopes have the ability to go to magnifications of x 40 and beyond (Figure 5). However, limitations in depth of field and illumination make this impractical. Therefore, magnifications in the range of x 2.5 to x 30 are recommended. The lower magnifications (x 2.5 to x 8) are used for orientation to the surgical field and allow a wide field of view. Midrange magnifications (x 10 to x 16) are used for operating. Higher range magnifications (x 20 to x 30) are used for examining fine detail.

Areas in which the SOM can have its greatest impact include the following: visualizing the surgical field; evaluating the surgical technique; reducing the number of radiographs needed; expanding patient education through video use; providing reports to referring dentists



Figure 5. JEDMED/ KAPS SOM 62 (Jedmed Instrument Company, St. Louis, Mo.) power zoom and power focus microscope, with 35 mm and video cameras.



Figure 6. Final retrofills on the beveled surface of a maxillary right first bicuspid (x 13).

and insurance companies as well as creating documentation for legal purposes.

The most significant advantage of using the SOM is in visualizing the surgical field. It is truly educational to enlarge the periradicular region before our eyes. Figure 6 shows SuperEBA retrofills in the buccal and lingual canals of the resected root surface of a maxillary right first bicuspid. They clearly illustrate the benefits of enhanced magnification and illumination achieved with an SOM. If you can see a task better, you can do it better, and performing endodontic surgery is no exception. Fractures, accessory canals, canal isthmuses, and fins can readily be observed and dealt with accordingly (Figures 7 and 8).

Microsurgical Technique

With the SOM, we can better locate surgical access entry sites and remove osseous tissue with more precision. Periapical curettage is facilitated because bony margins can be scrutinized for completeness of tissue removal (**Figure 9**). More efficient removal of granulomatous tissue can aid wound healing.

Root-end resection is performed with a 170L tapered fissure bur in an Impact Air 45 handpiece (SybronEndo; Orange, Calif.) (**Figures 10-12**). This handpiece was originally designed for oral surgeons who needed more access when sectioning third molars. Because the turbine is offset at 45 degrees, the operator can use this handpiece to gain better access to the



Figure 7. A microsurgical explorer pointing out a microfracture on the beveled surface of a maxillary left lateral incisor (x 20).

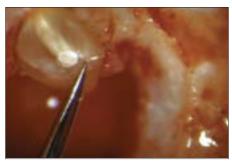


Figure 8. A microsurgical explorer pointing out an accessory canal on the beveled surface of a mandibular right first bicuspid (x 16).



Figure 9. Periapical curettage of a maxillary right first bicuspid (x 8).



Figure 10. 170L tapered fissure bur in an Impact Air 45 handpiece.

apices of maxillary and mandibular molars. When used in conjunction with the SOM, a long-shanked surgical bur can be placed with a high level of accuracy in the posterior regions of the mouth. When using the handpiece, the water spray is aimed directly into the surgical field but the air stream is ejected out through the back of the handpiece, thus eliminating much of the splatter that occurs with conventional high-speed handpieces. Because there is no pressurized air or water, the chances of producing pyemia and embolism are significantly reduced. The handpiece can be autoclaved, and a fiber optic system is available as an option.

After the root-end resection has been completed, the beveled surface can be easily examined for the presence of an isthmus, a very common finding. Recent studies^{16,17} have revealed presence of a complete or partial isthmus at

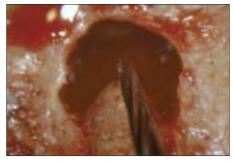


Figure 11. 170L tapered fissure bur in an Impact Air 45 handpiece placed against the apex of a maxillary left lateral incisor just before activation (x 16).

the 4 mm level of the mesiobuccal root of the maxillary first molar 100 percent of the time, and a complete isthmus in 90 percent of the time at the 3 mm level of the mesial root of the mandibular first molar (**Figure 13**).

Ultrasonic Tips

Apical preparations are now made with ultrasonic tips. Next to the SOM, the most exciting advancement in endodontic surgical technology has been in piezoelectric ultrasonic. Dentists are familiar with ultrasonic root scaling and ultrasonic cleaning and shaping of root canal systems. Piezoelectric ultrasonic units contain mechanisms that create ultrasonic vibrations in the range of 25 kHz to 40 kHz by exciting piezoelectric crystals in the handpiece. The units are selftuning regardless of changes in scaler



Figure 12. The beveled surface of the root of a maxillary left lateral incisor (x 16).

tip, file, or load, for maximum stability during operation. Water for cavitation and air pressure for activation are regulated by foot control. Continuous irrigation along the scaler or file cools the cutting surface while maximizing debridement and cleaning.

Various types of stainless-steel and diamond coated ultrasonic tips are available for apical preparation. The tips are 0.25 mm in diameter and about 3 mm in length. By comparison they are about one-tenth the size of a conventional micro-head handpiece (**Figure 14**).

A variety of tips are available to accommodate virtually all access situations. When used, they are placed in the long axis of the root so that the walls of the preparation will be parallel and encompass about 3 mm of the apical morphology. As the piezoelectric crystal in the handpiece is activated, the ener-

Endodontic Surgery



Figure 13. Isthmus on the beveled surface of the mesial root of a mandibular left first molar (x 16).



Figure 14. Ultrasonic tip and microhead handpiece with a No. 1 round bur.



Figure 15. Ultrasonic tip after activation, approximately 3 mm into the apical preparation (x 16).



Figure 16. Apical preparation of the root of a maxillary left lateral incisor using a 3 mm microsurgical mirror. Note the gutta-percha at the base of the preparation.

gy is transferred to the ultrasonic tip, which then moves forward and backward in a single plane. Typically, a 3 mm preparation takes about one to two minutes. The advantages of the ultrasonic tips over burs are:

- n Smaller apical preparations¹⁸
- n Cleaner apical preparations¹⁸

n Easier isthmus preparations between the exits of apical canals

n Easier access to the root tips

n Lesser strain and fatigue for the operator

 $\,n\,$ Better apical preparations (parallel walls in the long axis of the root)^{18}

The combination of the SOM and ultrasonic tips make previously challenging cases much easier. Apical preparation can be visualized and executed with a level of confidence that was previously unattainable. By combining magnification and ultrasonics, prepara-



Figure 17. One-half millimeter blunt Blue Micro Tip (Vista Specialty Products, Racine, Wisc.) mounted in a Stropko Irrigator on a tri-flow syringe.

tion of the apex can be conservative and can actually be viewed in the axial plane of the root (**Figure 15**).

Instrument Miniaturization

Magnification in endodontic surgery has led to miniaturization of endodontic surgical instruments. The entire armamentarium of the endodontic surgery has improved to facilitate precise treatment on tooth structures at magnifications of 15 to 30 times. Many standard operative and surgical dental instruments are no longer useful or appropriate for surgery when using the SOM.

Micro-Mirrors

Another development in endodontic microsurgery is the introduction of the surgical micro-mirror. These mirrors come in a variety of shapes and sizes and have diameters ranging from 1 mm to 5 mm. By using the SOM, it is now possible to look up into the apical preparation to check for completeness of tissue removal (**Figure 16**). Before using these mirrors, it was impossible to assess the thoroughness of apical preparation. Failure to completely remove old root canal filling material and debris from the facial wall of the apical preparation before placement of an apical seal may be the cause of surgical failures in the past.

Microsurgical Irrigation

Traditionally, practitioners dried apical preparations with paper points prior to placing root-end filling materials. A recently developed microsurgical irrigator now fits over the tri-flow syringe and allows for the directional micro-control of air and water (**Figure 17**). This instrument allows the apical preparation to be completely rinsed, dried and inspected with microsurgical mirrors before placement of a root-end filling material.

Today, practitioners can make apical preparations with a high level of confidence and accuracy with the combination of microscopic visualization, micro-mirrors, ultrasonic tips and microsurgical irrigators. Together with miniature carriers, condensers and pluggers, endodontists can now predictably identify and treat complex root canal anatomy, often overlooked in the past.



Figure 18. Placing retrofill material in the apical preparation of the root of a maxillary right lateral incisor with a No. 12 spoon excavator (x 16).

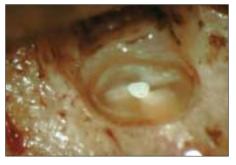


Figure 19. Finished retrofill of the root of a maxillary left cuspid (x 16).



Figure 20. Histological section of a monkey tooth filled with MTA as a root-end filling material demonstrates the regeneration of cementum over the resected root and MTA root-end filling material.



Figure 21. Condensing MTA with a P-1 plugger (x 16).



Figure 22. Micro-mirror view of finished MTA (x 16).

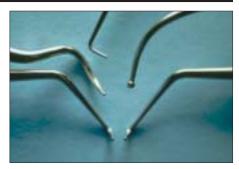


Figure 23. (Clockwise from top) Working ends of a P-1 plugger, the ball burnishing end of a B-3 plugger, opposite ends of a Stropko doubleended condenser, and working end of a P-2 (which is a "cowhorn" version of the P-1).

Root-End Filling Materials

After the apical preparation is thoroughly dried and examined, it is ready to be filled with a root-end filling material. The ideal root-end filling material should be biocompatible with periradicular tissues, resistant to absorption or breakdown by tissue fluids, and hermetically seal the canal.¹⁹

Cement consistency root-end filling materials such as SuperEBA and ProRoot MTA (Dentsply; Tulsa, Okla.) are currently the materials of choice. SuperEBA is mixed to a putty consistency and carried to the apical preparation in small, truncated cones 1 mm to 2 mm in size on a No. 12 spoon excavator (**Figure 18**). The cross-sectional diameter of this instrument is 1 mm; therefore, it does not block visual access to the apical preparation. The tip of the cone reaches

the base of the preparation as the sides of the cone contact the walls. Between each aliquot of material, a P-1 plugger is used to condense the SuperEBA. The P-1 plugger is a double-ended instrument with right-angled condensing surfaces of 0.25 mm and 0.50 mm on opposite ends. Additional aliquots of SuperEBA are added and condensed until there is a slight excess mound of material on the beveled surface of the root. Final compaction is accomplished with the ball burnishing end of a B-3 plugger (SybronEndo; Orange, Calif.). When the cement has set, an ET UF9 30-fluted finishing bur (Brasseler; Savannah, Ga.) or a smooth diamond is used to finish the procedure (Figure 19).

After the SuperEBA has been finished, a CX-1 microsurgical explorer is used to check for marginal adaptation and integrity. This is an extremely sharp instrument and caution must be exercised during its use. Final examination of the root-end filling is performed after the surface has been dried with a Stropko Irrigator, because it is more accurate to check the margins of the preparation when the beveled surface of the root is dry.

A new root-end filling material, mineral trioxide aggregate (MTA) is proving to be an impressive material for various endodontic uses including rootend filling and perforation repairs.²⁰ A tri-calcium compound, it provides excellent sealing properties and is extremely biocompatible with periradicular tissues. MTA provides a superior apical seal compared to other root-end filling materials and is not adversely affected by blood contamination. In

Endodontic Surgery

several studies, histological sections demonstrate the regeneration of new cementum over the MTA root-end filling,²⁰ a phenomenon that is not seen with other commonly used root-end filling materials (**Figure 20**).

ProRoot MTA is mixed with the supplied liquid to a very thick and dry consistency and carried to the apical preparation in a 0.90 mm diameter MAP System carrier (Roydent Dental Products; Johnson City, Tenn.). This carrier fits inside the apical preparation and allows for accurate placement of ProRoot MTA. The material is condensed with a P-1 plugger (**Figure 21**), and additional increments are added until the material is flush with the beveled surface. The ProRoot MTA is then finished with moist gauze and inspected with a micro-mirror (**Figure 22**).

The Effect Of New Advances On The Other Aspects Of Periradicular Surgery

As our surgical technique is re-evaluated, one of the natural consequences will involve modifying some of our surgical instruments. Many of the instruments now available will have to be redesigned with the endodontic micro-surgeon in mind. Conventional pluggers, curettes, and microamalgam carriers are just too big to work in small places. Several instrument companies have designed instruments for the various stages of endodontic microsurgery from flap design to flap closure (**Figure 23**).

The radiograph has been an essential tool for the endodontist. Although the SOM will not replace the radiograph in surgery, we can use fewer radiographs per procedure because we can see well by direct visualization. The bony crypt can be scrutinized for small particles of root-end filling material, lessening the need to stop the surgery and take a check radiograph. This decreases the patient's exposure to radiation.

Patient education can be a valu-

able result of videotaping through a video camera, which can be mounted on the surgical microscope. After surgery, the procedure can be viewed on a high-resolution monitor with the patient. The videotape can be viewed in its entirety or highlighted by using the fast-forward on the VCR. Most patients are quite receptive to reviewing the procedure post-surgically. This results in a better appreciation of the technology by the patient.

Most endodontists send their referring dentists a final radiograph of the completed case as part of the surgical report. An exciting addition to the final radiograph can be a video print of the completed case. Video printers have been available on the consumer market for some time and can easily be connected to a VCR or the video camera mounted on the microscope. A micro-computer inside the video printer automatically analyzes the image, and prints are created in 70 seconds by a high-density sublimation dye process. The video prints are 4 inches by 6 inches. Many different images can be digitized during the surgery and later recorded on a single print. Digital cameras can also be mounted to the microscope and images can be exported for use in computer presentations.

Most insurance companies require a radiograph of a completed surgical case before claim settlement. Often, the inclusion of a video print or a portion of videotape can help an insurance consultant get a better understanding of what occurred during the periradicular surgery. This can clarify and expedite claim processing.

Risk management is a familiar term to all of us. In addition to providing our patients with drawings and written surgical consents, videotaping certain highrisk procedures may provide the documentation necessary to prevail in a lawsuit. Videotaping has become a common practice in many medical specialties.

Does it Really Make a Difference?

A frequently asked question is, "Does it really make a difference?" A recent prospective study¹⁷ showed that the oneyear healing rates of endodontic surgery performed under the SOM in conjunction with microsurgical technique was 96.8 percent. A long-term follow up of these cases showed that 91.5 percent of these cases remained healed after five to seven years.²¹ Although it is impossible to tell whether the unusually high success rate resulted from the technique or the material, the clinical impression is that it is both the technique and the material with the emphasis on technique. Similar results are expected with ProRoot MTA.

Summary

Endodontic surgery is not "oral surgery" in the traditional sense. Rather, this procedure is actually "endodontic treatment-through a surgical flap." Simply cutting off the apex of a root and placing a filling in the vicinity of the canal does not accomplish the goals of surgical endodontic treatment. Endodontic surgery should result in sealing of all portals of exits to the root canal system and the isthmuses, eliminate bacteria and their byproducts from contaminating the periradicular tissues, and provide an environment that allows for regeneration of periradicular tissues. To accomplish these goals, endodontists have developed new techniques, materials and instruments. Enhanced illumination and magnification have greatly improved what practitioners can perform. Developments in root-end filling materials have increased both quality and biocompatibility of apical seals. Together, these advances have significantly improved the state of the art and science of endodontic surgery, giving a second chance to a tooth that was considered for extraction. CDA

References / **1.** Bergenholtz G, Lekholm U, et al, Retreatment of endodontic fillings. *Scand J Dent Res* 87:217-24,1979. 2. Allen R, Newton C, Brown C, A statistical analysis of surgical and nonsurgical endodontic retreatment cases. *J Endodon* 15(6):261-6, 1989.

3. Sjogren U, Hagglund B, et al, Factors affecting the long-term results of endodontic treatment. *J Endodon* 16(10):498-504, 1990.

4. Van Nieuwenhuysen J, Aouar M, D'Hoore W, Retreatment or radiographic monitoring in endodontics. *Int Endod J* 27:75-1, 1994.

5. Sundqvist G, Figdor D, et al, Microbiologic analysis of teeth with failed endodontic treatment and outcome of conservative retreatment. *Oral Surg* 85:86-3, 1998.

6. Gutmann JL, Harrison JW, Surgical Endodontics. Boston, Blackwell Scientific, 1991

7. Arens DE, Torabinejad M, Chivian N, Rubinstein R, Practical lessons in endodontic surgery. Chicago, IL, 1998, Quintessence Publishing Co., Inc.

8. McDonald NJ, Torabinejad M, Endodontic surgery In: Principles and practice of endodontics by: Walton RE and Torabinejad M. W. B. SAUN-DERS COMPANY, Philadelphia Penn., 2002.

9. Rubinstein RA, New horizons in endodontic surgery, Part I: The operating microscope. *Oak County (MI) Dent Review* 30(12):7, 1991.

10. Rubinstein RA, New horizons in endodontic surgery, part II: Periapical ultrasonics and more. Oak County (MI) Dent Review 30(13):9.1991.

11. Carr G, Microscopes in endodontics. *Calif* Dent Assoc J (11):55-1, 1992.

12. Pecora G, Andreana S, Use of dental operating microscope in endodontic surgery. *Oral Surg Oral Med Oral Pathol* 75:751-8, 1993.

13. Pecora G, Covani U, Giardino L, Rubinstein R, Valutazioni clinico-statistiche sull'uso dello stereomicroscopio in odontoiatria. *Riv Ital D Stoma* 8:425-1, 1993.

14. Izawa T, Kim S, Suda H, Pecora G, Rubinstein R: Microscopic endodontic surgery. *Quintessence Int* (Japan) 13:54-5, 1994.

15. Shelton M, Working in a Very Small Place: The Making of a Neurosurgeon. Ed 1, New York, Vintage, 1989.

16. Weller N, Niemczyk S, Kim, S, The incidence and position of the canal isthmus: part 1. The mesiobuccal root of the maxillary first molar. *J Endodon* 21:380-3, 1995.

17. Rubinstein R, Kim S, Short-term observation of the results of endodontic surgery with the use of a surgical operation microscope and SuperEBA as root-end filling material. *J Endodon* 25:43-8, 1999.

18. Wuchenich G, Meadows D, Torabinejad M, A comparison between two root-end preparation techniques in human cadavers and burs. *J Endodon*

20: 279-2, 1994.

19. Torabinejad M, Watson TF, Pitt Ford TR, The sealing ability of a Mineral Trioxide Aggregate as a retrograde root filling material. *J Endodon* 19:591-5, 1993.

20. Torabinejad M, Chivian N, Clinical applications of Mineral Trioxide Aggregate. *J Endodon* 1999; 25:197-205.

21. Rubinstein R, Kim S, Long-term follow-up of cases considered healed one year after apical microsurgery. *J Endodon* 28:378-3, 2002.

To request a printed copy of this article, please contact / Richard Rubinstein, DDS, MS, 31410 Northwestern Highway, Suite C, Farmington Hills, Michigan 48334.

Endodontic Therapy

THE SUCCESS OF ENDODONTIC THERAPY — HEALING AND FUNCTIONALITY

Shimon Friedman, DMD; and Chaim Mor, DMD

A B S T R A C T

Current, relevant knowledge on the outcome of endodontic therapy is key to clinical decision making, particularly when endodontic treatment is weighed against tooth extraction and replacement. Inherent to reviewing the outcome is a definition of "success" in relation to the goals of therapy. As the specific goal set out by the individual patient may either be healing/prevention of disease (apical periodontitis) or just functional retention of the tooth, the potential for both healing and functionality is reviewed. Based on selected follow-up studies that offer the best evidence, the chance of teeth without apical periodontitis to remain free of disease after initial treatment or orthograde retreatment is 92 percent to 98 percent. The chance of teeth with apical periodontitis to completely heal after initial treatment or retreatment is 74 percent to 86 per-

cent, and their chance to be functional over time is 91 percent to 97 percent. Thus there does not appear to be a systematic difference in outcome between initial treatment and orthograde retreatment. The outcome of apical surgery is less consistent than that of the nonsurgical treatment. The chance of teeth with apical periodontitis to completely heal after apical surgery is 37 percent to 85 percent, with a weighted average of approximately 70 percent. However, even with the lower chance of complete healing, the chance for the teeth to be functional over time is 86 percent to 92 percent. Considering the favorable outcome, conservative endodontic therapy, both nonsurgical and surgical,

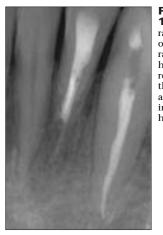


Authors / Shimon Friedman, DMD, is professor and head of Discipline of Endodontics and director of MSc program in Endodontics at the University of Toronto Faculty of Dentistry, Canada.

Chaim Mor, DMD, is a lecturer in the Department of Endodontics at Hebrew University — Hadassah Faculty of Dental Medicine in Jerusalem, Israel.



Figure 1a. Outcome classified as "healed." Pre-operative radiograph of a mandibular lateral incisor with apical periodontitis and associated apical external resorption.



(Reprinted with permission from Friedman S. Prognosis of initial endodontic therapy. Endodontic Topics 2:59-88, 2002)



2a. Outcome classified as "healed." Preoperative radiograph of a maxillarv second molar with apical periodontitis extending along the mesial root surface, and associated sinus tract (traced with a gutta-percha cone).

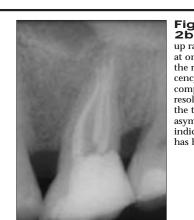
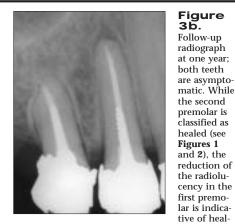


Figure 2b. Followup radiograph at one year; the radiolucency has completely resolved and the tooth is asymptomatic, indicating it has healed.

(Reprinted with permission from Friedman S. Prognosis of initial endodontic therapy. Endodontic Topics 2:59-88, 2002.)



Figure 3a. Outcome classification as "healed" vs. "healing.' Immediate post-operative radiograph of maxillary first and second premolars with apical periodontitis



ing in progress (see also **Figures 4** and **5**). Regrettably, both the restorations are inadequate.

(Reprinted with permission from Friedman S. Prognosis of initial endodontic therapy. Endodontic Topics 2:59-88, 2002.)

Figure 1b. Follow-up radiolucency has completely resolved and the tooth is asymptomatic, indicating it has healed. the tooth magine the following scenario. Mrs. L. K. suffered a complicated fracture of her left wrist. After receiving emergency care, she consulted with an orthopedic

surgeon who offered her two treatment options, each with its specific benefits and risks. The first option was to treat the broken wrist, with an 80 percent chance of "success" (complete healing of the fracture) but with the possibility of sporadic discomfort and some movement restriction, and the risk of re-fracture upon another impact. The second was to amputate the hand and replace it with a state-of-the-art prosthesis, with 97 percent chance of "success" (complete integration and functionality of the prosthesis) without discomfort or movement restriction. Although impressed by the prosthetic device's functionality and hand-like appearance, Mrs. L. K. selected the first treatment option without any hesitation.

The analogy of this scenario applies to recent articles¹⁻³ debating endodontic therapy of teeth versus implant-supported single tooth replacement, and comparing the "success" rates of both procedures. The main argument in that debate is the "success" rate of endodontic therapy — initial treatment, orthograde retreatment, and apical surgery. Often the quoted "success" rates are irrelevant to the debate, because they are outdated or derived from articles providing a low level of evidence. Importantly, the debate itself is often irrelevant, comparing a functional organ with an artificial prosthetic device, however perfect. While the latter problem concerns the balancing of ethics against pragmatism, the former is a matter of possessing the relevant knowledge regarding endodontic therapy and single-tooth implants, based on current information.

The objective of this article is to provide current, relevant review of the "success" of endodontic treatment pro-

Endodontic Therapy







Figure 4b. Immediate post-operative radiograph.



Figure 4c. Followup radiograph at one year; the tooth is asymptomatic but the radiolucency has not been reduced, indicating persistence of the disease.

(Reprinted with permission from Friedman S. Prognosis of initial endodontic therapy. Endodontic Topics 2:59-88, 2002.)

cedures, and thus the knowledge basis for case selection regarding options of endodontic therapy.

What is "Success"?

The non-specific term "success" is ambiguous - it has a different meaning when referring to different dental treatment procedures, such as endodontic therapy, periodontal therapy or implants.⁴ Undiscerning use of the term "success" confuses communication within the profession and it may misguide patients contemplating alternative treatments, particularly endodontic therapy versus extraction and tooth replacement. The definition of "success" and the related rates differ considerably for the various procedures in dentistry. The uninformed patient weighing one "success" rate against the other, may erroneously assume their definitions are comparable and select the treatment alternative that offers the "higher number" and thus appears to suggest a better chance of "success."⁴

Even for endodontic therapy, the definition of "success" has been ambiguous, with requirements ranging from stringent (radiographic and clinical normalcy) to lenient (only clinical normalcy).^{4,5} Clearly, the more lenient definition increases the "success" rate in comparison with the more stringent one. For example, in a follow-up study after endodontic initial treatment and retreatment, Friedman et al,⁶ report 78 percent complete healing (radiographic and clinical normalcy) and 16 percent incomplete healing (clinical normalcy) combined with reduced radiolucency). Their success rate can be interpreted as 78 percent using the stringent definition, or 94 percent using the more lenient definition.

To resolve this long-lasting dilemma, one should remember that "success" is invariably defined by the goal(s) established to be achieved. To use another analogy, one can reflect on two athletes preparing for the Olympic games - the first's goal is to attend the games, while the second's goal is to win. For the first athlete, just participating in the Olympics is a "success," irrespective of placement on the scoreboard. For the second athlete, only winning the gold medal is a "success" winning a silver medal may feel like a failure. Just like in this analogy, the confusion resulting from the ambiguity of the term "success" with regard to endodontic therapy can be easily avoided by defining the specific goals and expected outcomes of treatment.⁴

The usual goal of endodontic therapy is to prevent or heal disease, apical periodontitis.⁷ Accordingly, endodontic treatment outcomes should be defined in reference to healing and disease^{5,8,9} as follows:

n *Healed:* Both the clinical and radiographic presentations are normal (**Figures 1 and 2**).

n *Healing:* Because healing is a dynamic process, reduced radiolucency combined with normal clinical presentation can be interpreted as healing in progress (**Figure 3**).

n *Disease:* Radiolucency has emerged or persisted without change, even when the clinical presentation is normal (**Figure 4**), or clinical signs or symptoms are present, even if the radiographic presentation is normal.

Although curing of disease is the ultimate goal of therapy, patients are autonomous to set less demanding goals for therapy, such as prevention or elimination of symptoms, or retention of the tooth. The latter is particularly applicable when the patient is motivated to attempt therapy even though the projected prognosis is unfavorable because of complicating factors.



Figure 5a. Outcome classified as "functional." Pre-operative radiograph of a mandibular first molar with extensive apical periodontitis.



Figure 5b. Clinical view of the gingival recession on the buccal aspect of the tooth, coupled with probing depth apical to the root tips, suggests total loss of the buccal bone plate. The projected prognosis is poor.



Figure 5c.

Clinical view after reflection of a full-thickness flap reveals the extent of bone loss on the buccal aspect of the tooth. Advised of the poor prognosis, the patient decided to proceed with treatment in an attempt to retain the tooth in function as long as possible.

Accordingly, the endodontic treatment outcome can be defined as tooth retention,⁴ as follows:

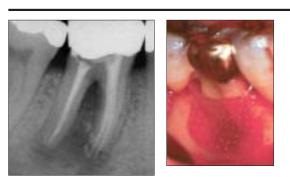
n *Functional retention:* The clinical presentation is normal, while radiolucency may be absent or present — newly emerged or persisting (**Figures 5 and 6**).

Considering the above, the outcome of endodontic therapy, or its "success," is usually defined as the healing of disease unless it is specifically defined as tooth retention in asymptomatic function.⁴

Relevant Information on Endodontic Treatment Outcomes

The potential for healing and functional retention of endodontically treated teeth can be gleaned from numerous follow-up studies of selected populations, exposed to initial treatment (Figure 7), orthograde retreatment (Figure 8) and apical surgery (Figure 9). Cumulatively, those studies include data from thousands of treated cases. Comprehensive reviews of those studies4,5 reveal inconsistencies and large variability in the reported outcomes,^{4,5} resulting from diversity in material composition, treatment procedures, and methodology.^{4,5} Because of that diversity, not all published studies are equally valuable as a source of valid and clinically relevant information. As suggested by one review,⁴ "undiscerning review of all the existing studies can be ineffective and even misleading."

The fact that clinical studies vary with regard to the level of evidence they



Figures 5d and e. Immediate post-operative radiograph after root canal therapy (root filling with vertical compaction of warm guttapercha), followed by placement of a resorbable guided tissue regeneration membrane.



Figures 5f and g. Follow-up radiograph and clinical view at six months; the radiolucency is considerably reduced and the gingival tissue appears to be healed. Although the prognosis remains poor, the tooth being functional achieves the goals of therapy as set by the patient, and should be considered as success.

(Reprinted with permission from Friedman S. Prognosis of initial endodontic therapy. Endodontic Topics 2:59-88, 2002.)

provide is well recognized in the current concept of evidence-based health care.¹⁰ Reviewed studies, therefore, must be appraised according to well-defined criteria to differentiate them according to the level of evidence.¹¹ Such appraisal criteria can also be applied to select those endodontic outcome studies that provide the best evidence.

In a review article on the prognosis of initial treatment of apical periodontitis, Friedman⁴ used the accepted guidelines for appraisal of studies.¹² The appraisal criteria were grouped into four general parameters, comprising the following: cohort at inception and end-point of study, exposure (treatment), outcome assessment, and analysis/reporting of data. Studies conforming to three out of the four parameters were selected for review, while others were excluded. A similar approach was subsequently used to review studies on orthograde retreatment¹³ and apical surgery.¹⁴

Tables 1-3 list the studies selected in the reviews as described above, for initial treatment,^{8,9,15-27} orthograde retreatment^{13,15,16,20,28,29} and apical

Endodontic Therapy



Figure 6a. Outcome classified as "functional." Preoperative radiograph of a mandibular lateral incisor with apical periodontitis and a palatal developmental groove associated with an extensive bone loss. Prognosis of this condition is recognized as hopeless: however. the patient decided to proceed with treatment in an attempt to retain the tooth in function as long as possible.



Figure 6b. Immediate postoperative radiograph after root canal therapy.



Figures 6c and d. Clinical view after reflection of buccal and palatal fullthickness flaps, revealing the extent of bone loss and the developmental groove.



Figure 6d.

Figure 6f.

Figures 6e and f. Immediate postoperative clinical view and radiograph after filling of the groove with varnish and amalgam.

Figure 6g.

Follow-up radiograph at four years; the api-

cal periodontitis has

healed, and the crestal

bone margin has stabi-

tooth being functional

achieves the goals of therapy as set by the

patient.

lized. Although the prognosis remains questionable, the



(Reprinted with permission from Friedman S, Goultschin J. The radicular palatal groove — a therapeutic modality. Endod Dent Traumatol 4:282-6, 1988.) surgery,^{14,29-33} respectively. The outcomes in the tables are interpreted from those reported by the original authors, as follows: (i) combined clinical and radiographic normalcy is classified as "healed"; (ii) reduced radiolucency combined with clinical normalcy is classified as "healing"; and (iii) the rate of teeth with no signs and symptoms is classified as "functional" — for several studies this is simply the sum of "healed" and "healing" (when both are available), while for others it also includes teeth where the radiolucency remained unchanged.

Treatment Outcome in Teeth Presenting Without Apical Periodontitis

Teeth that present without apical periodontitis may have irreversible pulpitis, pulp necrosis, or a dubious root filling.⁴ Accordingly, they undergo initial treatment or orthograde retreatment with the goal of preventing emergence of apical periodontitis. The outcomes of initial treatment and retreatment are presented separately in Tables 1 and 2, respectively. Consistently high percentages of teeth that remained healed after follow-up of up to 10 years can be seen in both tables. Excluding studies that appear to be outliers for initial treatment.^{16,25} it can be concluded that the chance of teeth without apical periodontitis to remain free of disease is 92 percent to 98 percent, both after initial treatment and orthograde retreatment. The rate of functional teeth is not indicated in these studies; however, it is likely to be even higher than the healed rate. Considering the generally asymptomatic nature of apical periodontitis.^{13,14,26,27} it can be assumed that only a few of the teeth with emerged disease are symptomatic.

Treatment Outcome in Teeth Presenting With Apical Periodontitis

Teeth that present with apical periodontitis may have a primary infection





Table 1

SELECTED FOLLOW-UP STUDIES ON THE OUTCOME OF ENDODONTIC INITIAL TREATMENT

			Teeth without Apical Periodontitis	Teeth with Apical Periodontitis				
Study	Follow-up (years)	Cases observed	Healed (%)	Healed (%)	Healing (%)	Functional ^a (%)		
Strindberg 1956	0.5-10	258	93	8 0	-	-		
Engtröm et al 1964	4 – 5	221	8 8	73	-	-		
Kerekes & Tronstad 1979	3-5	491	97	90	-	-		
Byström et al 1987	2-5	79	-	8 5	9	94		
Ørstavik et al 1987	1-4	543	9 5	-	-	-		
Eriksen et al 1988	3	121	-	8 2	9	91		
Sjögren et al 1990	8-10	471	9 6	86	-	-		
Ørstavik 1996	4	599	94	75	13	88		
Sjögren et al 1997	<5	5 3	-	83	-	-		
Trope et al 1999	1	76	-	8 0	-	-		
Weiger et al 2000	1-5	6 7	-	78	16	94		
Hoskinson et al 2002	4 – 5	200	8 8	74	-	97		
Peters & Wesselink 2002	1-4.5	38	-	76	21	97		
Friedman et al 2003	4-6	120	9 2	74	18	97		
Farzaneh et al 2004	4-6	242	94	79	-	9 5		
A Transmission of all tools, with and without animal anniholation								

^a Proportion of all teeth, with and without apical periodontitis

of the root canal system, or a residual or subsequent infection after endodontic treatment. Accordingly, they undergo initial treatment, orthograde retreatment, or apical surgery with the goal of healing of apical periodontitis. The outcomes of those treatment procedures are presented separately in **Tables 1**, **2** and **3**, respectively.

Even among the selected studies on the outcome of initial treatment (**Table 1**) and orthograde retreatment (**Table 2**), there is some variability in the reported results. The "healed" rates up to 10 years after therapy, range from 73 percent¹⁶ to 90 percent¹⁷ for initial treatment, and from 74 percent²⁸ to 86 percent¹³ for orthograde retreatment. This disparity is considerably smaller

than that observed across all studies for initial treatment (46 percent to 91 percent, Figure 7) and retreatment (43 percent to 86 percent, Figure 8). Because the selected studies are rather uniform in outcome assessment, this variability may be related to differences in case selection, in requiring a negative bacterial culture before root filling, and in restoration after treatment.⁴ Excluding studies that appear to be outliers, on initial treatment¹⁷ and on orthograde retreatment,^{20,29} as well as teeth with perforations before retreatment,¹³ it can be concluded that the chance of teeth with apical periodontitis to completely heal is 74 percent to 86 percent, after both initial treatment and orthograde retreatment. The fact that apical periodontitis has a similar potential to heal after initial treatment and orthograde retreatment challenges the historic perception, of the latter having a poorer prognosis than the former.

Seven of the studies on initial treatment (**Table 1**) and one study on retreatment (**Table 2**) reveal that over 88 percent of the teeth are "functional," with the disease mostly healed or healing. It can be assumed that in additional teeth disease persists without symptoms, as in 5 percent of the teeth included in the study by Friedman et al;²⁶ thus the rate of functional teeth probably approaches or even exceeds 95 percent.^{24,26,27} Excluding one study on initial treatment that appears to be an out-

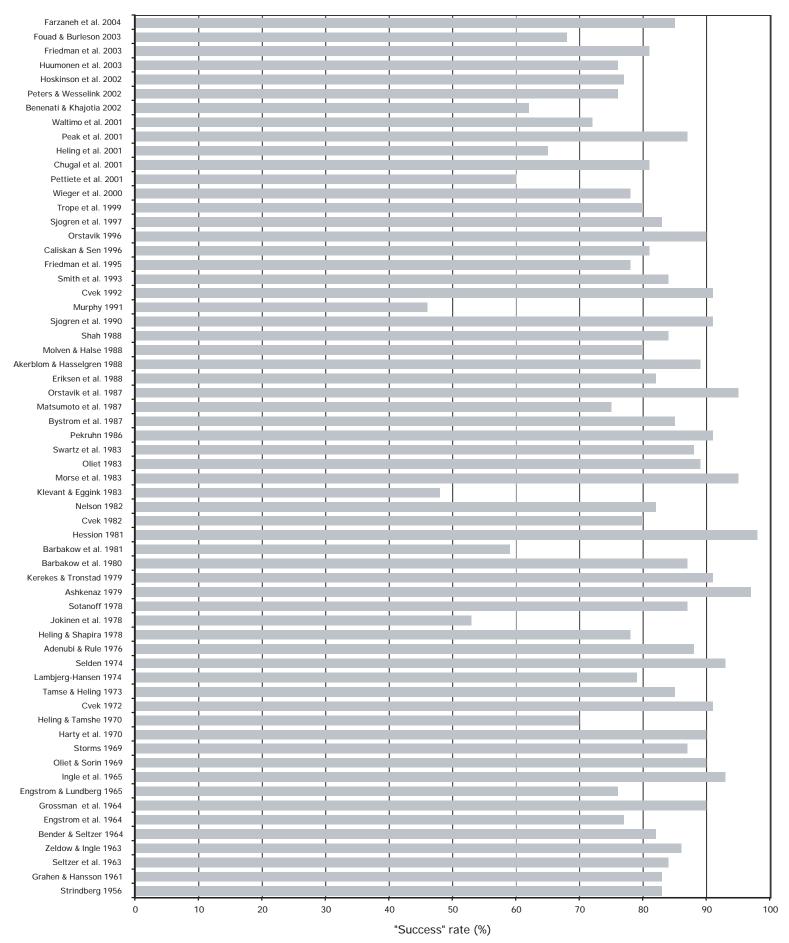


Figure 7. Graphic representation of the reported success rates in follow-up studies after endodontic initial treatment, from 1956 till 2004.

Continued from Page 498

lier,⁹ it can be concluded that the chance of teeth with apical periodontitis to remain in asymptomatic function is 91 percent to 97 percent, after both initial treatment and orthograde retreatment. These figures are certainly at par with the "success" rate reported for single-tooth implant-supported replacement.³⁴ Clearly, then, in teeth with apical periodontitis, a good restorative and periodontal prognosis and no pre-operative perforation, conservative endodontic therapy is definitely justified and should be attempted; tooth extraction and replacement should not be considered unless the patient is not motivated to retain the tooth.

Among the selected studies on the outcome of apical surgery (**Table 3**) the variability in the reported results

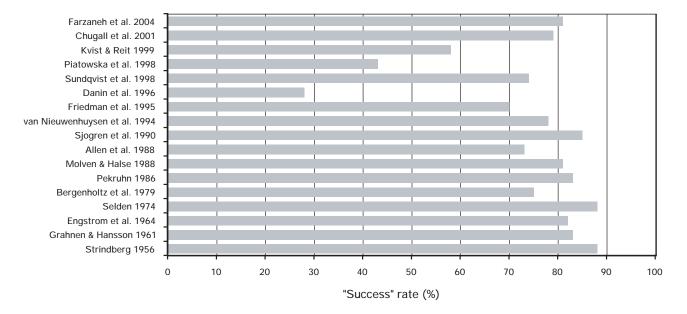


Figure 8. Graphic representation of the reported success rates in follow-up studies after endodontic orthograde retreatment, from 1956 till 2004.

Table 2

SELECTED FOLLOW-UP STUDIES ON THE OUTCOME OF ENDODONTIC ORTHOGRADE RETREATMENT

			Teeth without Apical Periodontitis	Teeth with Apical Periodontitis		
Study	Follow-up (years)	Cases observed	Healed (%)	Healed (%)	Healing (%)	Functional ^a (%)
Strindberg 1956	0.5-10	187	9 5	84	-	—
Engtröm et al 1964	4-5	153	93	74	-	-
Sjögren et al 1990	8-10	266	98	6 2	-	_
Sundqvist et al 1998	4	54	-	74	-	-
Kvist & Reit 1999	4	47	_	58	_	_
Farzaneh et al 2004	4	103	97	86 ^b	6	93

^aProportion of all teeth, with and without apical periodontitis

^bExcluding teeth with pre-operative perforations (78 percent healed with perforated teeth included)

Endodontic Therapy

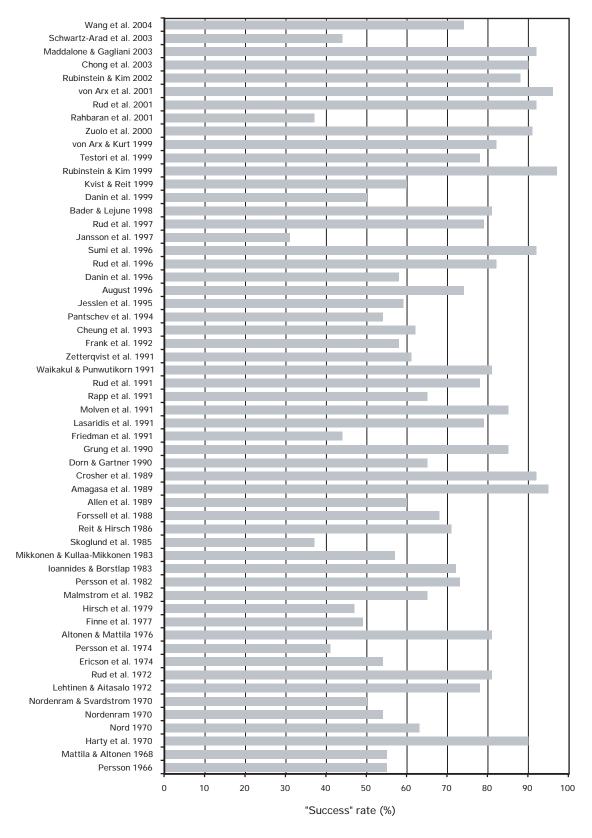


Figure 9. Graphic representation of the reported success rates in follow-up studies after apical surgery, from 1966 till 2004.

Table 3

SELECTED FOLLOW-UP STUDIES ON THE OUTCOME OF APICAL SURGERY

Study	Follow-up (years)	Cases observed	Healed (%)	Healing (%)	Functional (%)	
Molven et al 1991	1-8	222	8 5	17	92	
Jansson et al 1997	1-1.3	6 2	31	5 5	8 6	
Kvist & Reit 1999	4	4 5	6 0	_	-	
Zuolo et al 2000	1 - 4	102	91	-	-	
Rahbaran et al 2001	≥4	129	3 7	3 3	70	
Wang et al 2004	4-8	94	74	-	91	

remains large. The "healed" rates, up to eight years after surgery, range from 31 percent³¹ to 91 percent.³² This disparity is comparable to that observed across all studies for apical surgery (31 percent to 97 percent, Figure 9). In regard to apical surgery, this variability may be related to differences in case selection, in percentage of teeth undergoing repeat surgery,³⁵ in type (initial or retreatment) and quality of the previous endodontic treatment,^{5,14} and possibly, also in root-end preparation and filling techniques. Excluding two studies that appear to be outliers,^{31,32} the chance of teeth with apical periodontitis to completely heal after apical surgery appears to be 37 percent to 85 percent. To overcome this wide range and draw more definitive conclusions from the selected studies, a weighted average can be calculated.³⁶ Including the outlier studies^{31,32} the average is 66 percent, and excluding these studies, the average is 69 percent. It can be concluded, therefore, that the surgical treatment is less predictable than the nonsurgical treatment, with an approximate 70 percent chance for teeth to heal.

Three of the studies reveal that 70 percent to 92 percent of the teeth show the disease to be healed or healing (**Table 3**). Because in additional

teeth disease most likely persists without symptoms, the rate of "functional" teeth after apical surgery approximates 90 percent (Table 3). Excluding one study that appears to be an outlier,33 it can be concluded that the chance of teeth with apical periodontitis to remain in asymptomatic function after apical surgery is 86 percent³¹ to 92 percent.³⁰ These figures may be considered lower than those for nonsurgical endodontic therapy. Nevertheless, they suggest that, for teeth with apical periodontitis and a good periodontal prognosis, even apical surgery is justified and should be attempted rather than contemplating tooth extraction and replacement, unless the patient is not motivated to retain the tooth.

Case Selection

Selection of cases for endodontic therapy takes into consideration the prognosis of the endodontic, restorative and periodontal procedures, but also health and socio-economic factors. Contraindications to treatment include non-restorable and periodontally hopeless teeth, patients with extensive dental problems and restricted resources (that have to be utilized so as to benefit as many teeth as possible), and medically compromised patients at high-risk for infection.

Criteria used for case selection can influence the outcome of endodontic therapy. It can be generalized, however, that in teeth presenting without apical periodontitis, the chance to prevent disease in the long-term is excellent. Even in teeth presenting with apical periodontitis, the prognosis is good whether they are exposed to initial treatment, orthograde retreatment or apical surgery — the chance of complete healing is reasonably high, and the chance for the tooth remaining asymptomatic and functional over time is truly excellent, provided that the tooth is promptly and well restored. An asymptomatic functional state, although not a measure of healing, allows the tooth to be retained without necessitating extraction. This clear, even if not optimal benefit should be routinely communicated to patients when endodontic therapy is weighed against tooth extraction and replacement with a prosthetic device.

Summary

In summary, the concerns regarding the success of endodontic therapy are unsupported and misguided. The success of endodontic therapy, in terms of healing and functionality, is very good for both teeth without and with apical periodontitis. Therefore,

Endodontic Therapy

the most appropriate form of endodontic therapy should be attempted whenever feasible, and generally preferred over tooth extraction and replacement.

References / 1. Curtis DA, Lacy A, Chu R, Richards D, Plesh O, Kasrovi P, Kao R, Treatment planning in the 21st century: What's new? *J Cal Dent Assoc* 30:503-10, 2002.

2. Matosian GS, Treatment planning for the future: Endodontics, posts and core, and periodontal surgery - or an implant? *J Cal Dent Assoc* 31:323-5, 2003.

3. Somborac M, Implant treatment versus endodontic retreatment: A contemporary dilemma. *Oral Health* Oct: 8-15, 2003.

4. Friedman S, Prognosis of initial endodontic therapy. *Endodontic Topics* 2:59-88, 2002.

5. Friedman S, Treatment outcome and prognosis of endodontic therapy. Essential Endodontology: Prevention and Treatment of Apical Periodontitis. *Oxford: Blackwell Science*, 1998.

6. Friedman S, Löst C, Zarrabian M, Trope M, Evaluation of success and failure after endodontic therapy using glass ionomer cement sealer. *J Endodon* 21:384-90, 1995.

7. Ørstavik D, Pitt Ford TR, Apical periodontitis: Microbial infection and host responses. Essential Endodontology: Prevention and Treatment of Apical Periodontitis. *Oxford: Blackwell Science* 1998.

8. Byström A, Happonen RP, Sjögren U, Sundqvist G, Healing of periapical lesions of pulpless teeth after endodontic treatment with controlled asepsis. *Endod Dent Traumatol* 3:58-3, 1987.

9. Ørstavik D, Time-course and risk analyses of the development and healing of chronic apical periodontitis in man. *Int Endod J* 29:150-5, 1996.

10. Sackett DL, Richardson W, Rosenberg W, Haynes R, Evidence-based medicine: how to practice and teach EBM. *London: Churchill Livingstone* 1997.

11. Anderson JD, Need for evidence-based practice in prosthodontics. *J Pros Dent* 83:58-5, 2000.

12. Department of Clinical Epidemiology and Biostatistics, McMaster University Health Science Centre. How to read clinical journals. III. To learn the clinical course and prognosis of disease. *Can Med Assoc J* 124:869-2, 1981.

 Farzaneh M, Abitbol S, Friedman S, Treatment outcome in Endodontics: The Toronto Study. Phases I and II: Orthograde Retreatment. J Endodon 2004, in press.
 Wang NC, Knight K, Dao TT, Friedman S,

14. Wang NC, Knight K, Dao TT, Friedman S, Treatment outcome in Endodontics: The Toronto Study. Phases I and II: Apical Surgery. *J Endodon* 2004, submitted.

15. Strindberg LZ, The dependence of the results of pulp therapy on certain factors. An analytic study based on radiographic and clinical follow-up examination. *Acta Odontol Scand* 14:suppl. 21, 1956.

16. Engström B, Hard AF, Segerstad L, Ramstrom G, Frostell G, Correlation of positive cultures with the prognosis for root canal treatment. *Odontol Rev* 15, 257-70, 1964.

17. Kerekes K, Tronstad L, Long-term results of endodontic treatment performed with a standard-ized technique. *J Endodon* 5:83-90, 1979.

18. Ørstavik D, Kerekes K, Eriksen HM, Clinical performance of three endodontic sealers. *Endod Dent Traumatol* 3:178-6. 1987.

19. Eriksen HM, Ørstavik D, Kerekes K, Healing of apical periodontitis after endodontic treatment using three different root canal sealers. *Endod Dent Traumatol* 4:114-7, 1988.

20. Sjögren U, Hägglund B, Sundqvist G, Wing K, Factors affecting the long-term results of endodontic treatment. *J Endodon* 16:498-4, 1990.

21. Sjögren U, Figdor D, Persson S, Sundqvist G, Influence of infection at the time of root filling on the outcome of endodontic treatment of teeth with apical periodontitis. *Int Endod J* 30:297-6, 1997.

22. Trope M, Delano O, Ørstavik D, Endodontic treatment of teeth with apical periodontitis: Single vs. multivist treatment. *J Endodon* 25:345-50, 1999.

23. Weiger R, Rosendahl R, Löst C, Influence of calcium hudroxide intracanal dressings on the prognosis of teeth with endodontically induced periapical lesions. *Int Endod J* 33:219-6, 2000.

24. Peters LB, Wesselink PR, Periapical healing of endodontically treated teeth in one and two visits obturated in the presence or absence of detectable microorganisms. *Int Endod J* 35:660-7, 2002.

25. Hoskinson SE, Ng YL, Hoskinson AE, Moles DR, Gulabivala K, A retrospective comparison of outcome of root canal treatment using two different protocols. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod* 93:705-15, 2002.

26. Friedman S, Abitbol S, Lawrence HP, Treatment outcome in endodontics: The Toronto Study. Phase 1: Initial Treatment. *J Endodon* 29:787-93, 2003.

27. Farzaneh S, Abitbol S, Lawrence HP, Friedman S, Treatment outcome in endodontics: The Toronto Study. Phase II: Initial Treatment. *J Endodon* 2004, in press.

28. Sundqvist G, Figdor D, Persson S, Sjögren U, Microbiologic analysis of teeth with failed endodontic treatment and the outcome of conservative retreatment. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod* 85:86-3, 1998.

29. Kvist T, Reit C, Results of endodontic retreatment: A randomized clinical study comparing surgical and nonsurgical procedures. *J Endodon* 25:814-7, 1999.

30. Molven O, Halse A, Grung B, Surgical management of endodontic failures: indications and treatment results. *Int Dent J* 41:33-2, 1991.

31. Jansson L, Sandstedt P, Láftman AC, Skoglund A, Relationship between apical and marginal healing in periradicular surgery. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod* 83:596-1, 1997.

32. Zuolo ML, Ferreira MOF, Gutmann JL, Prognosis in periradicular surgery: a clinical prospective study. *Int Endod J* 33:91-8, 2000.

33. Rahbaran S, Gilthorpe MS, Harrison SD, Gulabivala K, Comparison of clinical outcome of periapical surgery in endodontic and oral surgery units of a teaching dental hospital: A retrospective study. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod* 91:700-9, 2001.

34. Creugers NHJ, Kreulen CM, Snoek PA, de Kanter RJAM, A systematic review of single-tooth restorations supported by implants. *J Dent* 28:209-7, 2000.

35. Peterson J, Gutmann JL, The outcome of endodontic resurgery: a systematic review. *Int Endod J* 34:169-5, 2001.

36. Hepworth MJ, Friedman S, Treatment outcome of surgical and nonsurgical management of

endodontic failures. J Can Dent Assoc 63:364-1, 1997.

To request a printed copy of this article, please contact / Shimon Friedman, DMD, Endodontics, University of Toronto Faculty of Dentistry, 124 Edward St., Toronto, Ontario, M5G 1G6, Canada.

Fun With Gold Foil

ike most dentists, I have a special place in my heart just between a prolapsed mitral valve and a partially occluded aorta for that great Italian professor, Giovanni Arcolani of the University of Bologna.

As we all know, Arcolani's scholarly treatise, Cirguria practica, published in Venice in 1483, dealt extensively with dentistry. If you are not conversant with Italian except for recognizing the first verse of Dean Martin's Amore and have difficulty following the subtitles in the early Gina Lollobrigida films, you might mistake his remarks for a recipe for pasta fagiola. The truth is, Arcolani offered the first documentation of the use of gold foil for filling diseased teeth. It was shortly after that Christopher "Giovanni" Columbus sailed off the edge of the earth in the Nina, the Pinto and the Corvair to discover India wasn't there, although historians have failed to find any connection between the two events.

Another authoritative surgical treatise authored by Giovanni da Vigo in 1514 also mentions filling cavities with "leaves of gold," leading some scholars to speculate he was poetically referring to autumn in Milan where it was common practice for local practitioners to stuff fallen leaves triturated with attar of roses as a protective pulpal base.

From that inauspicious beginning until the year 1983 when the California Dental Board mercifully dropped the requirement for demonstrating proficiency in gold foil placement, no dental student drew an easy breath working with this material. The term "technique sensitive" doesn't begin to cover the problems with gold foil. Even so, when it was discovered in the early 1850s that an alternative filling technique that involved pouring molten metals directly into prepared cavities was deleterious to the pulp, most reputable dentists used foil.

Fast forward to 1940 where at the College of Dentistry, University of Southern California, gold foil had become the Holy Grail for dental students. It was stated often enough to induce bruxism in the most placid of us that if you could place a good Class III foil, you could do anything. Implied was if you couldn't, a position as ribbon clerk at JC Penney's was about the most you could aspire to.

It pains me deeply to realize there is a generation of new dentists to whom gold foil is as familiar as red compound, vulcanite, silver nitrate, cocoa butter and

silicate cements.

Why these young whippersnapper dentists were spared the Sisyphean task of completing the dozens of required units of foils that my peers and I sweated out for graduation and licensure, is beyond understanding.

For many years an unholy alliance between the dental schools, the State Board of Dental Examiners and the International Gold Foil Cartel mandated that candidates for licensure demonstrate that

they could, by God, pound in a gold foil a sadistic examiner couldn't flip out after ______ Continued on Page 525 --

The term "technique sensitive" doesn't begin to cover the problems with gold foil.

Dr. Bob

Continued from Page 525

repeated tries. The requirement held even if the candidate was destined to become a member of one of the specialties, or a salaried employee at an Acme Smile & Breath Clinic franchise where gold foil was as foreign as hen's teeth.

There has never been a recorded instance of a patient actually requesting a gold foil be placed in his or her mouth, especially if the procedure had been explained beforehand. The gold foil discipline became an exercise in precision, of perfecting hand-to-eye coordination and a short cut to gastric ulcers. It was like boot camp or a fraternity initiation and we emerged the better for it, we lied for years after.

Our introduction to foil came in 1940 when we were issued a number of 5-inch by 5-inch sheets of extremely thin, delicate gold. We were to carefully roll these sheets into pellets about the size of a grain of rice. The gold was so fragile that if you breathed on it, or even stared at it too long, it would suddenly vaporize into individual atoms. It would take 5,000 of these sheets rolled up in a ball to be the size of a baby pea. Hitler's Panzer divisions were rolling through Europe, but that was a minor distraction. We were rolling through foil under the hooded, watchful gaze of a red-striped overseer.

After painstakingly concocting several thousand of these foil pellets, they were treated with ammonia fumes so they wouldn't stick together. Cold welding was a plus feature of gold foil, calculated to offset its other intractable frustrations. Those of us still displaying some visual acuity and able to withstand the orthopedic havoc resulting from prolonged pellet rolling, were allowed to learn the secret of cavity preparation.

There's one thing about gold foil it exhibits an almost manic determination to return to its natural state and decamp to Ney's or Sutter's Mill, or wherever it came from. You put it in a cavity, it falls out. You put it in again, it falls out again. So the drill was retention points, undercuts and, if the instructor wasn't looking, methylmethacrylate, commercially known as Krazy Glue.

All students were issued instruments (single-ended, of course) to accomplish this retention-making. The only other use for this armamentarium was inscribing the Lord's Prayer on the head of a pin. We were to learn later that there was a bigger market for the latter than for the foil restoration it was designed to facilitate.

In order to place a Class II or a Class III foil, it was necessary to spread teeth apart so when the task was done, interproximal contact would be intact. A device perfected by Tomas de Torquemada, CEO of the Spanish Inquisition, was used to separate teeth. The comfort level of this procedure was comparable to passing a largish kidney stone, only less fun.

Then came the actual foil placement. You needed to start this in the morning, because it was an all day job.

After approximately 50,000 condenser taps, the gold begins to take some sort of form. The instructor comes by followed by his entourage of crazed students. He demonstrates that your retention points are not up to snuff by taking a sharp explorer and flipping your handiwork out on the floor. retinue. Somebody titters.

This causes the patient to mumble through his rubber dam that he would like an aspirin or some morphine. The 50,000 blows at right angles to his tooth are beginning to take their toll.

Finally, the filling is complete. Sometimes this is the same day you started, which is desirable, otherwise you have to take the patient home and feed him via an eyedropper. "How's that?" you ask, handing the patient a No. 4 plain surface mouth mirror.

He lifts a slab of numb upper lip and peers at your masterpiece.

"Huh. Is it always going to look like that?" he demands.

"Like what?"

"Like I got a piece of spinach between my teeth. How come I can't have a white filling?"

You convince him that the eighthinch gap between his teeth will close up by summer and he'll hardly notice the new filling except when he smiles. You give him a referral to an endodontist.

Well, that was then and this is now. I confess I haven't put in a gold foil in 55 years. I have four foils in my own teeth deftly placed there by fellow students short on units and long on confidence. I almost died. I consider that if these were done under present day fees, I'd have over \$2,000 worth of work to show for the trauma. Each one is as good today as the day it was placed, which is more than I can say for myself.