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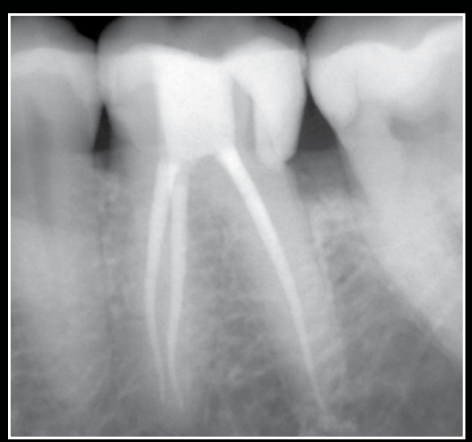

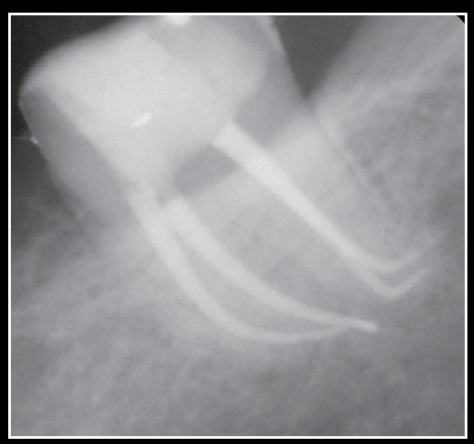

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AAE annual session: Time to come together and learn

Fred Weinstein, DMD, MRCD(C), FICD, FACD

If you are like me, you enjoy dental meetings because they bring so many of us together under one roof to learn. The American Association of Endodontists annual session is definitely something to look forward to. It's certainly one of the highlights of my year.

Perhaps you picked up this copy of *roots* at AAE15 in Seattle — or maybe at one of the many other spring meetings — and you are reading this on the plane home. That's good, because this issue includes many helpful articles.

Dr. Steven G. Morrow offers a report on the use and abuse of antibiotics in endodontic treatment. Dr. Rich Mounce shares his knowledge of the new Mani Silk files for canal shaping. Dr. Brett E. Gilbert, in an interview, discusses his experience using the new Sonendo GentleWave system in clinical practice. There are also articles about some other new product offerings.

The article by Dr. Morrow, which originally appeared in AAE's *ENDODONTICS: Colleagues for Excellence* newsletter, is being made available in this issue of *roots* with the permission of the AAE. By reading this article, and then taking a short online quiz at *www.DTStudyClub.com*, you will gain one ADA CERP-certified C.E. credit. Keep in mind that because *roots* is a quarterly magazine, you can actually chisel four C.E. credits per year out of your already busy life without the lost revenue and time away from your practice.

To learn more about how you can take advantage of this C.E. opportunity, visit *www.DTStudyClub.com*. You need only register at the Dental Tribune Study Club website to access these C.E. materials free of charge. You may take the C.E. quiz after registering on the DT Study Club website.

You can also access the vast library of C.E. articles published in the AAE's clinical newsletter by visiting *www.aae.org/colleagues*.

I know that taking time away from your practice to pursue C.E. credits is costly in terms of lost revenue and time, and that is another reason *roots* is such a valuable publication. I hope you will enjoy this issue and that you will take advantage of the C.E. opportunity.

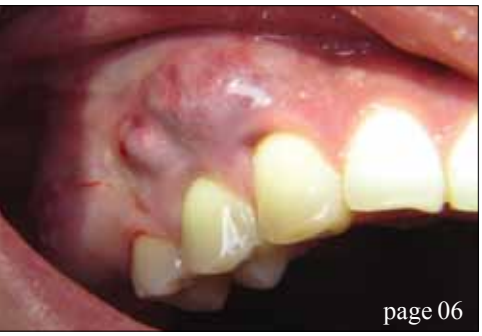
For those of you attending the AAE meeting this spring in Seattle, be sure to say hello in person. I'll also be at several other meetings this spring.

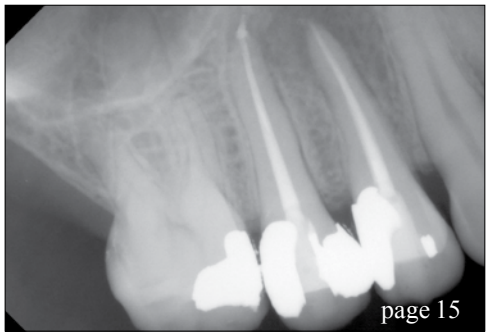
As always, I welcome your comments and feedback.

Sincerely,

Fred

Fred Weinstein, DMD, MRCD(C), FICD, FACD
Editor in Chief


page 06


page 15


page 21

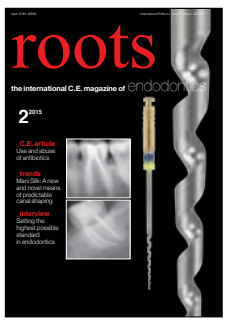
C.E. article

technique

interview

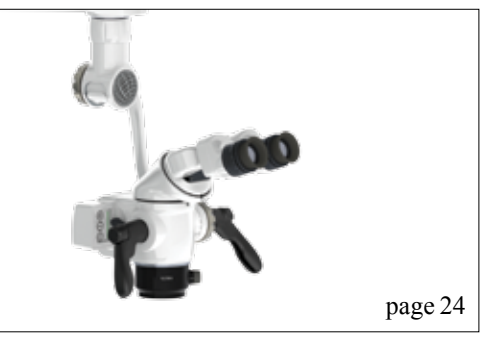
industry

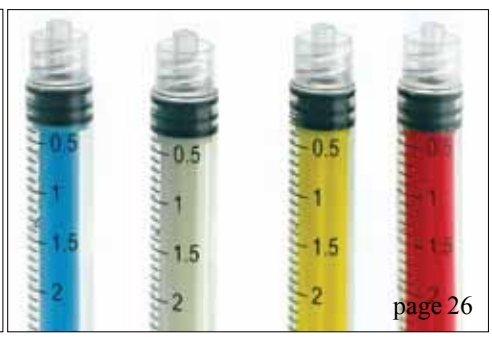
about the publisher

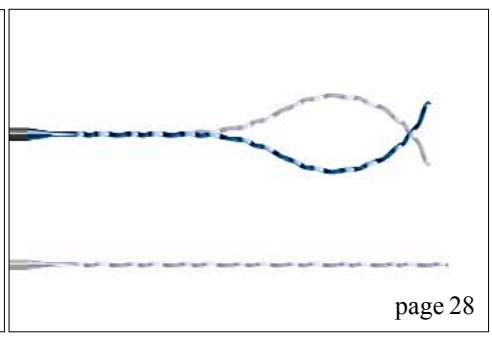


on the cover

Mani Silk files and clinical cases. (Photos/Provided by Mani Inc. and Rich Mounce, DDS)


page 24


page 26


page 28

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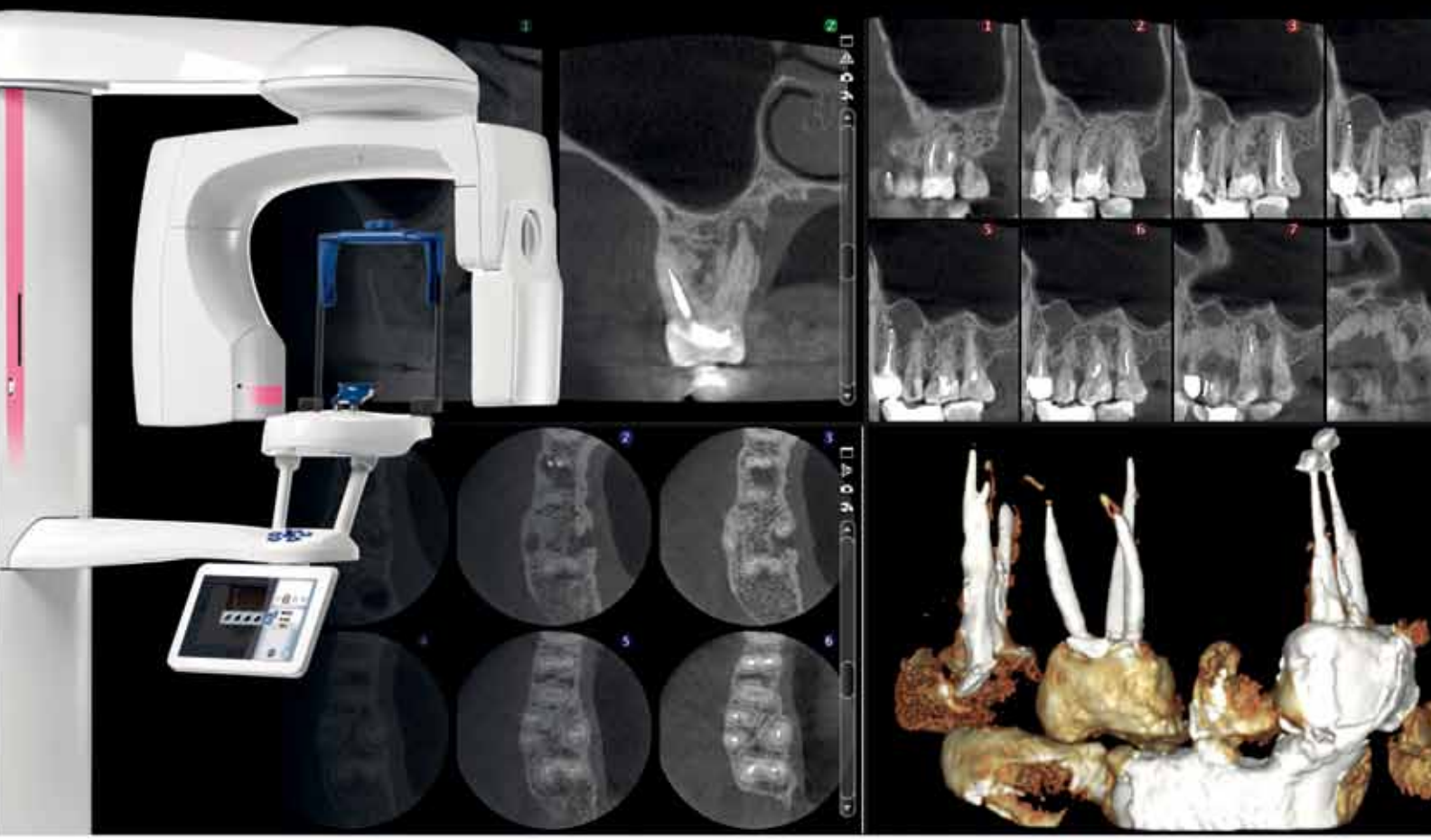

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Use and abuse of antibiotics

Author_Steven G. Morrow, DDS, MS

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This article qualifies for C.E. credit. To take the C.E. quiz, log on to *www.dtstudyclub.com*. Click on 'C.E. articles' and search for this edition (Roots C.E. Magazine — 2/2015). If you are not registered with the site, you will be asked to do so before taking the quiz. You may also access the quiz by using the QR code below.

_For the past 80 years, antibiotic therapy has played a major role in the treatment of bacterial infectious diseases. Since the discovery of penicillin in 1928 by Fleming and sulfanilamide in 1934 by Domagk, the entire world has benefited from one of the greatest medical advancements in history. The discovery of safe, systemic antibiotics has been a major factor in the control of infectious diseases and, as such, has increased life expectancy and the quality of life for millions of people.

According to the Centers for Disease Control and Prevention, life expectancy of individuals in the United States born in 1900 was 47 years, while those born in 2005 is projected to be 78 years.[1] At the beginning of the 20th century, the infant (< 1 year) mortality rate in the United States was 100/1,000 live births compared to 6.7/1,000 in 2006.[2] The major reason for these phenomenal achievements has been the ability to control infectious diseases.[3]

_Development of antibacterial drug resistance

Along with the dramatic benefits of systemic antibiotics, there has also been an explosion in the number of bacteria that have become resistant to a variety of these drugs. The problem is not the antibiotics themselves. They remain one of medicine's most potent weapons against diseases. Instead, the problem is in the way the drugs are used. The inappropriate overuse of antibiotics has resulted in a crisis situation due to bacterial mutations developing resistant strains.

Many worldwide strains of *Staphylococcus aureus* exhibit resistance to all medically important antibacterial drugs, including vancomycin; and methicillin-resistant *S. aureus* has become one of the most frequent nosocomial, or hospital-acquired, pathogens. The rate at which bacteria develop resistance to antibacterial drugs is alarming, demonstrating resistance soon after new drugs have been introduced. This rapid development of resistance has contributed significantly to the morbidity and mortality of infectious diseases, especially nosocomial infections.[4]

A nosocomial infection is a hospital-acquired infection that develops in a patient after admission. It is usually defined as an infection that is identified at least 48 to 72 hours following admission, so infections incubating, but not clinically apparent at admission, are excluded. Nosocomial infections are costly, resulting in increased morbidity, requiring longer periods of hospitalization and limiting access of other patients to hospital resources. The direct costs of hospital-acquired infections in the United States are estimated to be $4.5 billion per year. Nosocomial infections also contribute to the emergence and dissemination of antimicrobial-resistant organisms. Antimicrobial use for treatment or prevention of infections facilitates the emergence of more resistant organisms. Patients with infections caused by antimicrobial-resistant organisms are then a source of infection for hospital staff and other hospitalized patients. These drug-resistant infections may subsequently spread to the community.[5]

The British Society for Antimicrobial Chemotherapy published a review in the Journal of Antimicrobial Chemotherapy. This review examined the contributions antibiotic prescribing by general dentists in the United Kingdom has made to the selection of antibiotic resistance in bacteria of the oral flora.[6] The review concluded that inappropriate antibacterial drug prescribing by dental practitioners

Fig. 1_Asymptomatic apical periodontitis. (Photos/Provided by American Association of Endodontists)

Fig. 2_Chronic apical abscess.

Fig. 3_Acute apical abscess with intraoral localized swelling.

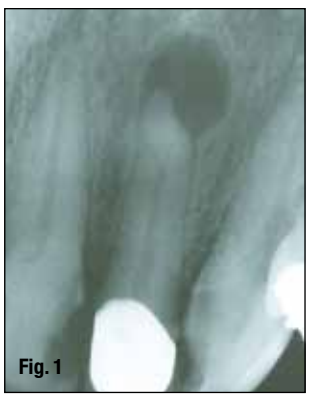
Fig. 1

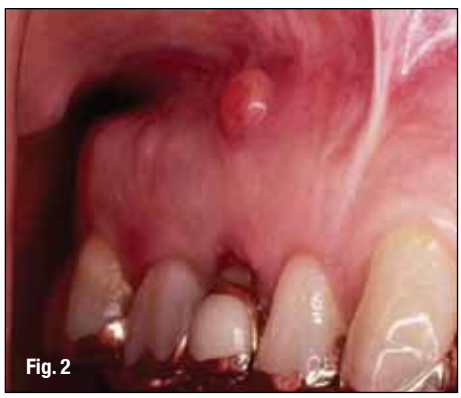
Fig. 2

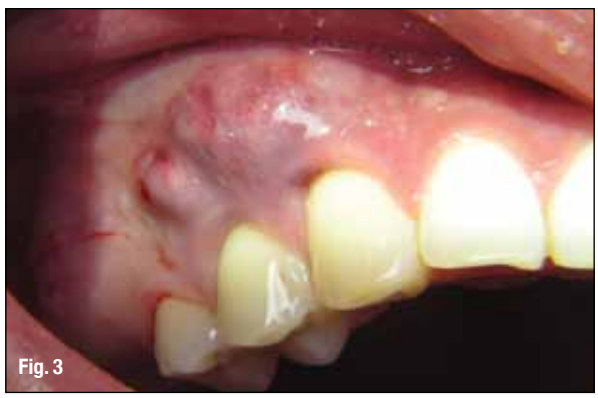
Fig. 3

is a significant contributing factor in the selection of drug-resistant bacterial strains.

The American Dental Association reported the results of a survey of antibiotic use in dentistry in the November 2000 Journal of the American Dental Association.[7] The authors surveyed all licensed dentists practicing in Canada and found that confusion about prescribing antibiotics and inappropriate prescribing practices were evident, and that inappropriate antibiotic use, such as improper dosing, duration of therapy and prophylaxis are all factors that may affect development of antibiotic resistant microorganisms.

_There is a glimmer of hope

A report from Aker University in Oslo, Norway, strongly suggests that bacterial resistance to antibacterial agents can be reversed.[8] While dangerous and contagious staph infections kill thousands of patients in the most sophisticated hospitals in Europe, North America and Asia, there is virtually no sign of this "killer superbug" in Norway. The reason? Norway stopped using so many antibiotics.

"We don't throw antibiotics at every person with a fever. We tell them to hang on, wait and see, and we give them a Tylenol to feel better," said Dr. John Haug, infectious disease specialist at Aker University Hospital.[8] In Norway's simple solution, there is a glimmer of hope.

_The proper clinical use of antibacterial drugs

In 1997, the ADA Council on Scientific Affairs issued a position statement on Antibiotic Use in Dentistry.[9] The Council stated: "Microbial resistance to antibiotics is increasing at an alarming rate. The major cause of this public health problem is the use of antibiotics in an inappropriate manner, leading to the selection of dominance of resistant microorganisms and/or the increased transfer of resistance genes from antibiotic-resistant to antibiotic-susceptible microorganisms."[9]

The council's position statement further identified that "Antibiotics are properly employed only for the management of active infectious disease or the prevention of metastatic infection, such as infective endocarditis, in medically high-risk patients."[9]

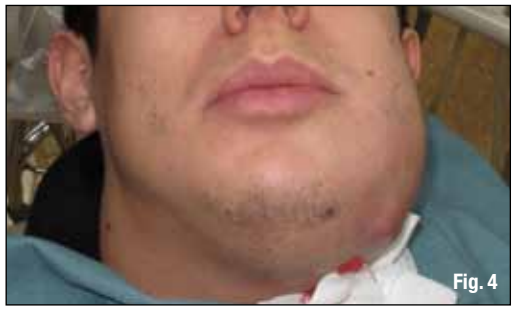

Fig. 4_Acute apical abscess with extraoral diffuse facial cellulitis.

One method of education is to teach from errors rather than principles. Psychologists from the University of Exeter have identified an "early warning signal" in the brain that helps us avoid repeating previous mistakes. Published in the Journal of Cognitive Neuroscience,[10] their research identifies for the first time, a mechanism in the brain that reacts, in just one-tenth of a second, to things that have resulted in us making errors in the past. Evaluating the following eight misconceptions or "myths" may help to establish general guidelines to aid us in making clinical decisions regarding the use of antibiotic therapy, thereby leading to optimum use and therapeutic success.[11]

Myth No. 1: Antibiotics cure patients. Except in patients with a compromised immune system, antibiotics are not curative, but instead function to assist in the re-establishment of the proper balance between the host's defenses (immune and inflammatory) and the invasive agent(s). Antibiotics do not cure patients; patients cure themselves.

Myth No. 2: Antibiotics are substitutes for surgical intervention. Very seldom are antibiotics an appropriate substitute for removal of the source of the infection (extraction, endodontic treatment, incision and drainage, periodontal scaling and root planing). Occasionally, when the infection is too diffuse or disseminated to identify a nidus for incision, or the clinical situation does not allow for immediate curative treatment, the prudent dentist will choose to place the patient on appropriate antibacterial therapy until such time as curative treatment can be implemented. It is imperative to remove the cause of the infection prior to, or concomitant with, antibiotic therapy,

(Tables/Provided by American Association of Endodontists)

Primary Reasons for Revision of Infective Endocarditis Guidelines

1. IE is much more likely to result from frequent exposure to random bacteremias associated with daily activities than from bacteremias caused by a dental, GI tract or GU tract procedure.
2. Prophylaxis may prevent an exceedingly small number of cases of IE, if any, in individuals who undergo a dental, GI tract or GU tract procedure.
3. The risk of antibiotic-associated adverse events exceeds the benefit, if any, from prophylactic antibiotic therapy.
4. Maintenance of optimal oral health and hygiene may reduce the incidence of bacteremia from daily activities and is more important than prophylactic antibiotics for a dental procedure to reduce the risk of IE.

Table 1

when the cause is readily identifiable. Whenever antibiotic therapy is used, the risk of bacterial selection for antibiotic resistance is present.

Myth No. 3: The most important decision is which antibiotic to use. To avoid the deleterious effects of needless antibiotics on patients and the environment, the most important initial decision is not which antibiotic to prescribe but whether to use one at all. It has been estimated that up to 60 percent of human infections resolve by host defenses alone following removal of the cause of the infection without antibiotic intervention.

Endodontic disease is infectious. Microorganisms cause virtually all pathoses of the pulp and periapical tissues. There is ample evidence to support that opportunistic normal oral microbiata colonize in a symbiotic relationship with the host, resulting in endodontic infections.[12] The majority of endodontic infections do not require systemic antibiotic therapy when the cause of the infection has been properly managed (complete debridement of the pulp space and proper obturation and sealing of the pulp space from the oral environment).

Apical periodontitis lesions of pulpal origin are generated by the immune system and are the result of intraradicular infections (Fig. 1). In most situations, this inflammatory process successfully eliminates the bacteria emerging from the apical foramen and prevents their spread to the periapical tissues. This process is primarily facilitated by the polymorphonuclear leukocytes that eventually phagocytize and kill the bacteria.[13] Asymptomatic apical periodontitis of pulpal origin does not routinely require systemic antibiotic therapy for satisfactory resolution and healing. Endodontic therapy alone is usually sufficient.

When the intraradicular infection is able to overwhelm the host's immune response, viable bacteria are able to gain access to the periapical tissues and colonize, forming an active infection. This results in the formation of an apical abscess. A chronic apical abscess usually presents with gradual onset, no to mild symptoms and the presence of a sinus tract or parulis (Fig. 2). The majority of chronic apical abscesses of endodontic origin do not require systemic antibiotic therapy for satisfactory resolution and healing.

An acute apical abscess usually presents with rapid onset, spontaneous pain and swelling, both localized and intraoral, sometimes with exudate present, or with diffuse facial cellulitis. When the abscess is intraoral and localized (Fig. 3), debridement of the pulp space and placement of calcium hydroxide and surgical incision for drainage is usually sufficient to resolve the problem. Systemic antibiotic therapy is not routinely indicated, depending on the patient's

Medical Conditions for Which Endocarditis Prophylaxis <u>is Recommended</u>:

Premedication is recommended <u>ONLY</u> for patients with the following conditions associated with the highest risk of adverse outcomes from endocarditis:

1. Prosthetic cardiac/heart valve.
2. History of IE.
3. Cardiac transplant recipients who develop valve pathology.
4. One of the following congenital heart diseases:
   - Unrepaired cyanotic CHD, including palliative shunts and conduits.
   - Completely repaired congenital heart defects with prosthetic material or device, whether placed by surgery or by catheter intervention, **during the first six months after placement of the material or device (because endothelialization of prosthetic material occurs within six months after the procedure).**
   - Repaired CHD with residual defects at, or adjacent to, the site of a prosthetic patch or prosthetic device (which inhibits endothelialization).
5. Special situations and circumstances:
   - **Patients already receiving antibiotics**—Occasionally, a patient may be taking an antibiotic when coming for a dental appointment. If the patient is taking an antibiotic normally used for endocarditis prophylaxis, it is prudent to select a drug from a different class rather that increase the dose of the current antibiotic. If possible, you should delay the dental procedure until at least 10 days after completion of the antibiotic. This will allow for the usual oral flora to be re-established. If an individual receiving long-term parenteral antibiotic therapy for IE requires dental treatment, the treatment should be timed to occur 30 to 60 minutes after the parenteral antibiotic therapy has been delivered.
   - **Failure to administer pretreatment antibiotic dose**—If the dosage of an antibiotic is inadvertently not administered before the procedure, the dosage may be administered up to two hours after the procedure.
     However, administration of the dosage after the procedure should be considered only when the patient did not receive the preprocedure dose.
   - **Individuals with kidney dialysis shunts**—Individuals with permanent kidney dialysis shunts should be placed on prophylactic antibiotics using the same protocol as for IE.

Table 2

general medical status. However, when the patient presents with diffuse facial swelling (cellulitis) resulting from an acute apical abscess or an infection with systemic involvement (fever or malaise) (Fig. 4), debridement of the pulp space with placement of calcium hydroxide, surgical incision for drainage, when possible, and an appropriate regimen of systemic antibiotics (oral or IV) are the treatments of choice.

Understanding the enemy is an important factor in winning any battle. The rational choice and use of antimicrobial agents begins with the knowledge of the microorganisms most likely responsible for common dental infections of pulpal origin. The bacterial flora found in endodontic infections is indigenous, mixed (Gram-positive and Gram-negative) and predominately anaerobic. Several species have been implicated with acute apical abscesses. These species include dark-pigmented bacteria (Prevotella and Porphyromonas), eubacteria, fusobacteria and Actinomyces.[12]

Baumgartner and Xia published a report of the susceptibility of bacteria recovered from acute apical abscesses to five commonly used antibiotics in dentistry. Antibiotic susceptibility data from 98 species of bacteria recovered from 12 acute apical abscesses led to the following conclusions:

1. Pen-V-K is the antibiotic of choice for endodontic infections due to its effectiveness in polymicrobial infections, its relative narrow spectrum of activity against bacteria most commonly found in endodontic infections, its low toxicity and low cost.

2. Clindamycin is the antibiotic of choice for patients allergic to penicillins.

3. While amoxicillin and augmentin (amoxicillin plus clavulanate) demonstrated a higher antibacterial effectiveness than Pen-V-K, due to the broader antibacterial spectrum of amoxicillin and the increased cost of augmentin, the authors recommended that amoxicillin/augmentin be reserved for unresolved infections and patients who are immunocompromised.

4. Metronidazol demonstrated the greatest amount of bacterial resistance and is only effective against anaerobes. Therefore, it should not be used alone for the treatment of endodontic infections.[14]

Myth No. 4: Antibiotics increase the host's defense to infection. The increased prevalence in organ and tissue transplants, resulting in patients with compromised immune systems, has heightened the interest in the potential effects of antimicrobial drugs on the host's resistance to infection.[15] In vivo and in vitro studies are highly variable and sometimes contradictory. However, the following considerations appear valid: 1) Antibiotics that can penetrate into the mammalian cell (erythromycin, tetracycline, clindamycin and metronidazole) are more likely to affect the host defenses than those that cannot (beta-lactams); 2) Tetracyclines may suppress white cell chemotaxis; 3) Most antibiotics (except tetracycline) do not depress phagocytosis; and 4) T- and B-lymphocyte transformation may be depressed by tetracyclines. The greatest potential harm to the host defenses may result from antibiotics that easily penetrate into the mammalian cell and the least harm is observed with bactericidal, nonpenetrating agents (penicillins and cephalosporins).

Myth No. 5: Multiple antibiotics are superior to a single antibiotic. It is often assumed that a combination of antibiotics is superior to a single carefully chosen antibacterial agent. When the purported benefits of antibiotic combinations are weighed against the possible consequences to the host as well as to the bacterial environment, this assumption is not always reality. The usual sequela to combined antibiotic therapy results in a greater selective pressure on the microbial population to develop drug resistance. The greater the antibacterial spectrum of the antimicrobials used, the greater the number of drug-resistant microorganisms that develop, and the more difficult it is to treat a resulting superinfection. The primary clinical indication for combined antimicrobial therapy is a severe infection in which the offending organism(s) is unknown and major consequences may ensue if antibiotic therapy is not instituted immediately before culture and sensitivity tests are available.[3]

Myth No. 6: Bactericidal agents are always superior to bacteriostatic agents. Bactericidal agents are required for patients with impaired host defenses.[3] However, bacteriostatic agents are usually satisfactory when the host's defenses against infections are unimpaired. Postantibiotic effects (PAEs – persistent suppression of bacterial growth after previous exposure to antibiotics) are more persistent and reliable with bacteriostatic agents (erythromycin, clindamycin) than with bactericidal agents (beta-lacatamase) because the clinical effects of bacteriostatic agents are less dose-dependent.

Myth No. 7: Antibiotic dosages, dosing intervals and duration of therapy are established for most infections. After more than 80 years of antibiotic usage, the proper

Dental Procedures for Which Antibiotic Prophylaxis is Reasonable

- Dental extractions
- Periodontal procedures, including surgery, subgingival placement of antibiotic fibers/strips, scaling and root planing, proving, recall maintenance
- Dental implant placement
- Replantation of avulsed teeth
- Endodontic (root canal) instrumentation only if beyond the root apex and endodontic surgery
- Initial placement of orthodontic bands (not brackets)
- Intraligamentary and intraosseous local anesthetic injections
- Postoperative suture removal (in selected circumstances that may create significant bleeding)
- Prophylactic cleaning of teeth or implants where bleeding is anticipated

Table 3