Use and abuse of antibiotics

By Steven G. Morrow, USA

For the past 90 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 sulfamethazine in 1935 by Domagk, the entire world has benefitted from the antibiotics that greatest medical advancements in history. The discovery of safe, effective, and potent 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 are expected to live 78 years. 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 2005. The major reason for these increases was the development of control of infectious diseases.1

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 or excessive use 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 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 infections, especially nosocomial infections.2

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 $45 billion per year.

Nosocomial infections also contribute to 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.3

The British Society for Antimicrobial Chemotherapy published a review in the Journal of Antimicrobial Chemotherapy that reviews 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.4 The review concluded that inappropriate antibiotic drug prescribing by dental practitioners 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.5 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.6 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 Tyle- nod to feel better," said Dr. John Haug, infectious disease specialist at Aker University Hospital.7 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.8 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 employed only for the management of active infectious disease or the prevention of metastatic infection, such as infective endocarditis, in medically high-risk patients.10 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,11 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 failed 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.12

Myth No. 1: Antibiotics cure pain. 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, incisions 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 cutaneous treatment, the prudent dentist will choose to place the patient on appropriate antibacterial therapy until such time as cutaneous treatment can be implemented. It is imperative to remove the cause of the infection prior to, or concomitant with, antibiotic therapy, 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 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 nidus.13 Once the nidus is gone, the antibacterial agent is unnecessary.

Endodontic disease is infectious. Microorganisms cause virtually all pathologies of the pulp and periradicular tissues. There is ample evidence to support that opportunistic normal oral microflora colonize in a symbiotic infectious relationship with the host, resulting in endodontic infections.14 The majority of endodontic infections are systemic antibiotic therapy when the cause of the infection is unknown.

Primary Reasons for Revision of Infective Endocarditis Guidelines

1. IE is much more likely to result from frequent exposure to random bacteriaes associated with daily activities than from bacteremias caused by dental, GI tract or GU tract procedures.

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.

Fig. 1. Symptomatic aperi- stentitis. Image courtesy of American Association of Endodontists.

Fig. 2. Chronic apical abscess. Image courtesy of American Association of Endodontists.

Fig. 3. Acute apical abscess with introral localized swelling.

Table I. (Tables Provided by American Association of Endodontists)

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has been properly managed (complete debridement of the pulp space and proper obtu-
ration and sealing of the pulp space from the oral environ-
ment).

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 suc-
scessfully eliminates the bacteria emerging from the apical foru-
men and prevents their spread to the periapical tissues. This process is primarily facilitated by the polymorphonuclear leuko-
cytes that eventually phago-
ditize and kill the bacteria.13

Asymptomatic apical periodon-
titis of pulpal origin does not routinely require systemic anti-
biotic therapy for satisfactory resolution and healing. Endo-
donital therapy alone is usually sufficient.

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

An acute apical abscess usually presents with rapid onset, spon-
taneous pain and swelling, both
local and intraoral, and sometimes
with exudate present, or with diffuse facial cellulitis. When the abscess is intraoral and localized (Fig. 5), debride-
ment of the pulp space and placement of calcium hydro-
oxide and surgical incision for drainage is usually sufficient to resolve the problem. Systemic antibiotic therapy is not routine-
ly indicated, depending on the patient’s general medical status. However, when the patient pres-
ents with diffuse facial swell-
ning (cellulitis) resulting from an acute apical abscess or an
infection with systemic involv-
ment (fever or malaise) (Fig. 4), debridement of the pulp space with placement of calcium hy-
droxide, surgical incision for
bacterial drainage, and an appropriate regimen of sys-
temic 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 be-
gins with the knowledge of the microorganisms most likely responsible for common dental infections of pulpal origin. The bacterial flora found in en-
do-
ontic infections is indigenous, mixed (Gram-positive and Gram-negative) and predomi-
nately anaerobic. Several spe-
cies have been implicated with acute apical abscesses. These species include dark-pigment-
ed bacteria (Prevotella and Por-
phyromonas), eubacteria, fuso-
bacteria and Actinomyces.6

Baumgartner and Xia published a report of the susceptibility of bacteria recovered from acute
apical abscesses to five com-
nonly used antibiotics in den-
tistry. 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 infec-
tions 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 aug-
mentin (amoxicillin plus clavu-
lanate) demonstrated a higher antibacterial effectiveness than
Pen-V-K, due to the broader an-
tibacterial spectrum of amoxi-
cillin and the increased cost of augmentin, the authors rec-
ommended that amoxicillin/ augmentin be reserved for un-
resolved 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 infec-
tions.11

Myth No. 4: Antibiotics increase
the host’s defense to infection. The increased prevalence in organ and tissue transplants, resulting in patients with com-
promised immune systems, has heightened the interest in the potential effects of antimicro-
bial drugs on the host’s resist-
ance to infection.12 In addi-
tion to the above, and in vitro studies are highly vari-
able and sometimes contradic-
tory. However, the following considerations appear valid: 1) Antibiotics that can penetrate into the mammalian cell (eryth-
rocytes, tetracycline, clinda-
mycin and metronidazol) are more likely to affect the host defenses than those that can-
not (beta-lactams); 2) Tetracy-
clines may suppress white cell chemo\systoxis; 3) Most antibiotic-
except tetracyclines) do not depress phagocytosis, and 4) T-
and B-lymphocyte transforma-
tion may be depressed by teta-

cyclines. The greatest potential harm to the host defenses may result from antibiotics that eas-
ily penetrate into the mammalian

cell and the least harm is observed with bactericidal, non

The potential effects of antimicro-
bial therapy on the host’s defenses are not yet fully understood, and the assumption is not always real-
ly. The usual sequela to com-
pletion of mCME

Myth No. 5: Multiple antibiotics are superior to a single antibiot-
istically. It is often assumed that a combination of antibiotics is su-
prior to a single carefully cho-

en antibacterial agent. When the purported benefits of antibi-

otically combination therapy are weighed against the possible conse-
quences to the host as well as to the bacterial environment, this assumption is not always real-
the program.

The answers and critiques published herein have been checked

and represent authoritative opinions about the questions
concerned. Articles are available on www.cappmea.com after the publication. For more information please contact events@cappmea.com or +971 4 3616174

Table 2 (Tables Provided by American Association of Endodontists)

<table>
<thead>
<tr>
<th>Medical Conditions for Which Endocarditis Prophylaxis is Recommended</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Prosthetic cardiac/heart valve.</td>
</tr>
<tr>
<td>2. History of IE.</td>
</tr>
<tr>
<td>3. Prosthetic cardiac/heart valve.</td>
</tr>
<tr>
<td>4. Medical Conditions for Which Endocarditis Prophylaxis is Recommended:</td>
</tr>
<tr>
<td>5. Special situations and circumstances:</td>
</tr>
<tr>
<td>6. Cardiac transplant recipients who develop valve pathology.</td>
</tr>
<tr>
<td>7. History of IE.</td>
</tr>
<tr>
<td>8. 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.</td>
</tr>
<tr>
<td>9. 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.</td>
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<tr>
<td>10. The risk of antibiotic-associated adverse events exceeds the benefit, if any, from prophylactic antibiotic therapy.</td>
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FOR INTERACTION WITH THE AUTHORS FIND THE CONTACT DETAILS AT THE END OF EACH ARTICLE.
bined antibiotic therapy results in a greater selective pressure on the microbial population to develop resistance. The greater the antibiotic spectrum of the antimicrobials used, the greater the likelihood of developing 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 no other treatments may suffice if antibiotic therapy is not instituted immediately before culture and sensitivity tests are available.3

Myth No. 6: Bacterioidal agents are always superior to bacteriostatic agents. Bacterioidal agents are required for patients with impaired host defense.1 However, bacteriostatic agents are usually satisfactory when the host’s defenses against infections are unimpaired. Postoperative suture removal is a persistent bacterial growth after previous exposure to antibiotic therapy with vaccine-induced serum levels of an antibiotic does not necessarily reflect those in tissues. Blood concentrations of the antibiotic should exceed the MIC by a factor of two to eight times in order to restrict the tissue barriers to the infected site. 

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 dosages, dosing intervals and duration of therapy are essentially unknown for most specific infections.4 Infectious diseases are associated with a high number of variables that affect treatment outcome (microbial characteristics and drug sensitivity, diverse resistance mechanisms, tissue barriers to antibiotic diffusion, and the integrity and activity of the host’s defense mechanisms). However, basic principles are available to guide the dental health care provider in establishing proper doses, dosing intervals and duration of therapy (e.g., the microbiological pathogen(s) is identified and a rational choice of antimicrobial agent is made. The following principles of antibiotic dosing are adapted from Dr. Thomas J. Palaszynski.1

1. The current recommendation is to employ an antimicrobial on an intermittent basis with vitamin-induced serum levels of this drug. A loading dose for as short a period of time as the clinical situation permits is possible. The major factors in the clinical success of most antimicrobial agents is the height of the serum concentration of the drug and the resulting amount in the infected tissue(s). Also important to the host is the antimicrobial agent for as short a duration of therapy as possible. The initial dose of antibiotic should be a loading dose of two times the maintenance dose. The goal of antibiotic dosing is to achieve drug levels in the infected tissue equal to or exceeding the minimal inhibitory concentration of the target organism. Serum levels of antibiotics do not necessarily reflect those in tissues. Blood concentrations of the antibiotic should exceed the MIC by a factor of two to eight times in order to restrict the tissue barriers to the drug the infected site. 

3. It is advisable to initiate antibiotic therapy with a loading dose (an initial dose higher than the maintenance dose). An antibiotic loading dose should be used whenever the half-life of the drug is longer than three hours or whenever a delay of 12 hours or longer to achieve a therapeutic blood level is expected. Most antibiotics used in the treatment of oral infections have a half-life shorter than three hours but, due to their acute nature, most orofacial infections require therapeutic drug blood levels sooner than 12 hours. Steady-state levels of any drug are usually achieved in a time equal to three to five times the drug’s half-life. Amoxicillin has a half-life of one to one-and-a-half hours. A steady-state blood level would then be achieved in three to seven-and-a-half hours, thereby leading to a substantial steady-state concentration that remains during therapeutic antibiotic blood levels. A loading dose of two times the maintenance dose is recommended for acute orofacial infections, which better achieve the goal of rapid, high blood levels rather than initiating therapy with the maintenance dose. 

4. All patients during first two years following joint replacement

5. Myocarditis due to the pathogen is beyond the root apex and endodontic surgery

6. Initial placement of orthodontic bands (not brackets)

7. Intraligamental and intraosseous local anesthetic injections

8. Postoperative suture removal (in selected circumstances that may cause significant bleeding)

9. Prophylactic cleaning of teeth or implants where bleeding is anticipated

10. Patients allergic to penicillin and unable to take oral medication

11. Dental patients presenting for treatment with impaired host defenses (chemotherapy, orificial infections, insulin-dependent diabetes, alcoholics) or patients with impaired host defenses (immunosuppression) may benefit from antibiotic prophylaxis if their risk of IE is below 0.5% and if the infecting microorganisms are beta-lactamase producers.17 IE prophylaxis may be effective, such therapy should be restricted to those patients with the highest

**Table 1. (Tables/Provided by American Association of Endodontists)**

<table>
<thead>
<tr>
<th>Patient Type</th>
<th>Drug Regimen*</th>
<th>Drug</th>
<th>Regimen*</th>
</tr>
</thead>
<tbody>
<tr>
<td>Patients not allergic to penicillin</td>
<td>Cephalothin, cefoxitin or amoxicillin</td>
<td>2g every 1 hour prior to dental procedure</td>
<td></td>
</tr>
<tr>
<td>Patients allergic to penicillin</td>
<td>Cefazolin or ampicillin</td>
<td>Cefazolin 1g or ampicillin 2g IM or IV 1 hour prior to dental procedure</td>
<td></td>
</tr>
<tr>
<td>Patients allergic to penicillin and unable to take oral medication</td>
<td>Cindamycin</td>
<td>Cindamycin 600mg orally 1 hour prior to dental procedure</td>
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<td>Cindamycin</td>
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**Table 3. (Tables/Provided by American Association of Endodontists)**

<table>
<thead>
<tr>
<th>Suggested Patient Type</th>
<th>Drug Regimen for Antibiotic Prophylaxis for Total Prosthetic Joint Infection</th>
<th>Patient Type</th>
<th>Drug Regimen*</th>
</tr>
</thead>
<tbody>
<tr>
<td>Patients not allergic to penicillin</td>
<td>Cephalothin, cefoxitin or amoxicillin</td>
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**Table 4. (Tables/Provided by American Association of Endodontists)**

<table>
<thead>
<tr>
<th>Patients at Potential Risk of Experiencing Hematogenous Total Joint Infection</th>
<th>Patient Type</th>
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<tr>
<td>Patients with comorbidities</td>
<td>Cefazolin or ampicillin</td>
<td>Cefazolin 1g or ampicillin 2g IM or IV 1 hour prior to dental procedure</td>
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<td>Patients with comorbidities</td>
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**Antibiotic prophylaxis for prevention of infective endocarditis**

The American Heart Association (AHA) published guidelines for the prevention of IE in medically at-risk patients for more than 50 years. The most recent guidelines, published in April 2007, represent a significant change from previous guidelines.17 One of the stated goals of the guidelines is that prolonged (after clinical resolution of the infection) antibiotic therapy is necessary to prevent “rebound” infections from occurring. Oralofacial infections do not persist for seven days or longer. Therefore, the recommended antibiotic dose is 0.75 hours. Higher continuous blood levels of this antibiotic are more likely to be obtained with four-hour rather than six-hour dosing intervals. The shorter the half-life of the drug, the shorter the dosing interval will need to be in order to maintain continuous therapeutic blood levels of the drug. When determining the appropriate dosing interval, it is also important to consider the following: 1) the postantibiotic effects of the drug; and 2) the relative merits of continuous or pulse dosing. PAEs are more persistent (two to seven hours) with antibiotics that act intra-

**Patients at Potential Risk of Experiencing Hematogenous Total Joint Infection**

**Patient Type** | **Drug Regimen** |
<table>
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<td>All patients during the two years following joint replacement</td>
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**Criteria for Placing Patient at Risk**

<table>
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<th>Inflammatory arthropathies such as rheumatoid arthritis, systemic lupus erythematosus</th>
<th>Oral or injectable radiodinated immunoglobulin</th>
</tr>
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<tr>
<td>Hematologic disorders</td>
<td>Malnourishment</td>
</tr>
<tr>
<td>Hemophilia</td>
<td>Drug or radiation-induced immunosuppression</td>
</tr>
</tbody>
</table>

**Prophylactic cleaning of teeth or implants where bleeding is anticipated**

**Myth No. 8:** Bacterial infections require a “complete course” of antibiotic therapy. There is no such thing as a “complete course” of antibiotic therapy. For dental patients presenting for treatment with impaired host defenses (chemotherapy, orificial infections, insulin-dependent diabetes, alcoholics) or patients with impaired host defenses (immunosuppression) may benefit from antibiotic prophylaxis if their risk of IE is below 0.5% and if the infecting microorganisms are beta-lactamase producers.17 IE prophylaxis may be effective, such therapy should be restricted to those patients with the highest...
risk of adverse outcomes from IE, and who would derive the greatest benefit from prevention. The dental literature indicates that certain cardiovascular conditions associated with the highest risk of adverse outcomes from IE are a periodontal status, suggesting that for some dental procedures is reasonable, even though we acknowledge that its effectiveness is unknown.\textsuperscript{16}\textsuperscript{17} Therefore, the 2007 AHA guideline statement on antibiotic prophylaxis should be considered for patients presenting for dental treatment with these conditions identified in Table 2, and who are undergoing any dental procedure that involves gingival tissue or periapical root of a tooth and for that oral bacteria that can be introduced to the oral mucosa. This would include procedures such as biopsies, suture removal, placement of orthodontic bands, and intra- ligamentary and intraosseous local anesthetic injections, but it does not include routine local anesthetic injections through noninfected tissue (Table 5).

### Antibiotic prophylaxis for patients with prosthetic joint infection

In 1997, the ADA and the American Academy of Orthopedic Surgeons convened an expert panel of dentists, orthopedic surgeons, infectious disease specialists and published an Advisory Statement on Antibiotic Prophylaxis for Patients with prosthetic joints.\textsuperscript{18} A 2003 advisory statement included some recommendations for stratification of patients at potential risk and the stratification of bacteremia.\textsuperscript{19,20} This statement identified patients prior to any invasive procedure that may cause bacteremia.\textsuperscript{20} In response to this statement, the American Academy of Oral Medicine published a position statement in June 2010 edition of the Journal of the American Dental Association.\textsuperscript{21}

The authors of the AAMOS position statement reviewed the available literature on the subject as it relates to the AAOS 2003 advisory statement and concluded: “The risk of patients experiencing drug reactions, infection, or bacterial infections and the cost of antibiotic medications alone do not justify the practice of antibiotic prophylaxis in (all) patients with prosthetic joints.”

The authors called for future multidisciplinary, systematic review of the literature relating to antibiotic prophylaxis in patients with prosthetic joints. In the meantime, they concluded that the new AAOS 2009 information statement\textsuperscript{19} should not replace the 2005 joint consensus statement.\textsuperscript{20}

In December 2012, a panel of experts representing the American Academy of Orthopedic Surgeons and the American Academy of Oral Medicine reviewed a systematic review and clinical practice guideline, titled “Prevention of Orthopaedic Implant Infection in Patients Undergoing Dental Procedures: Evidence-based Guideline and Evidence Report.” This report contained the following three recommendations:

1. “The practice might consider discontinuing the practice of routinely prescribing prophylactic antibiotics for patients with hip and knee prosthetic joint implants undergoing dental procedures.”
2. “We are unable to recommend for or against the use of topical or systemic antimicrobial prophylaxis with prosthetic joint implants or other orthopedic implants undergoing dental procedures.”
3. “In the absence of reliable evidence linking poor oral health to prosthetic joint infection, it is the opinion of the work group that patients with prosthetic joints are no different from other orthopedic implants maintain appropriate oral hygiene.”

The panel also stated that the above recommendations are not intended to stand alone. Treatment decisions should be made in light of all circumstances presented by the patient. Treatment and procedures applicable to the initial consultation or evaluation, including medical history and examination, treatment, diagnosis and treatment, and the patient’s preferences. This includes the patient’s recommendation or, if in his or her professional judgment, the dentist’s recommendation is indicated, decide to proceed with the needed dental or orthopedic care, and the treatment of the underlying dental or orthopedic condition. The dentist is ultimately responsible for making treatment decisions for his or her patient based on the dentist’s professional judgment.

In February 2000, the AAO published an information statement in which it recommends “that clinicians consider antibiotic prophylaxis for patients undergoing dental procedures” and stated: “We recommend patients prior to any invasive procedure that may cause bacteremia.”

In response to this statement, the American Academy of Oral Medicine published a position statement in June 2010 edition of the Journal of the American Dental Association.\textsuperscript{21}

“General, for patients with prosthetic joint implants, prophylactic antibiotics are not recommended prior to dental procedures in patients with prosthetic joint infection. The practitioner and patient should consider possible risks and benefits of procedures that may suggest the presence of a significant medical risk to the patient due to infection with an orthopedic prosthesis, as well as the known risks of frequent antibiotic use.”

As part of the evidence-based approach to care, this clinical practice guideline should be integrated with the practitioner’s professional judgment and the patient’s needs and preferences.

### Summary

Since their discovery decades ago, safe systemic antibiotics have transformed the treatment of infections, turning once deadly diseases into manageable health problems. However, the growing phenomenon of bacterial resistance, caused by the use of antibiotics and the simultaneous decline in research and development of new antibiotics, is now threatening to take us back to the pre-antibiotic era. Without effective treatment and prevention of bacterial infections, we also risk rolling back important achievements in medical care such as major surgery, organ transplantation and cancer chemotherapy.\textsuperscript{22}

A fundamentally changed view of antibiotic resistance is needed. They must be looked on as a common good, where individuals must be aware that their choice to use an antibiotic will affect the possibility of effectively treating infections in other people. All antibiotic use, appropriate or not, “uses up” the antibiotic potential in nature and may render it useless to us in the future. For current and future generations, it is crucial that we have access to effective prevention and treatment of bacterial infection. We must also care for their right to health, all of us need to act now. The windows of opportunity is rapidly closing.\textsuperscript{22}

### References


### About the Author

Having taught oral healthcare professionals at Loma Linda University School of Dentistry since 1965, Steven Morrow, DDS, MS, FICD, is a past president of the American Academy of Endodontists and as president of the California State Association of Endodontists from 1987 to 1999. He maintains responsibilities accepted in 2000 as director of patient care services and clinical quality assurance. He was director, District VI, of the American Association of Endodontists from 1993 to 1995, and has also served as president of the Southern California Academy of Endodontics and as president of the California State Association of Endodontists from 1993 to 1995.

In 1987, he earned diploma status from the American Board of Endodontics. Since 1989, he has been a Fellow of the American College of Dentists; and since 1990, he serves on the editorial review board of the Journal of Endodontics. He was a member of the American Dental Association Council on Scientific Affairs, the American Academy of Endodontists and the California State Association of Endodontists. Among his accomplishments was his second term as a member of the Dental Board of California.