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Antibiotic prophylaxis: problems in paradise

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Once upon a time it all seemed so simple: antibiotics cured patients and antibiotics prevented infections. Fifty years of therapy were based on the assumption that if antibiotics treated infections, surely they must prevent them.

As often occurs with assumptions, these ''truisms'' no longer seem true. Except for some immunocompromised patients and diseases requiring bactericidal antibiotic activity (ie, endocarditis), antibiotics are not curative but rather provide time for a host defense system temporarily overwhelmed by microbial pathogenicity to re-establish homeostasis. Surgical antibiotic prophylaxis has only been documented clinically effective with a reasonable risk-benefit ratio with perioperative use for the prevention of surgical wound infections in clean-clean or clean-contaminated operations (oral cavity surgery is contaminated-contaminated).

Antibiotic prophylaxis to prevent bacterial endocarditis has been advocated in risk-patients since 1955 based on animal studies and empirical reasoning by experts in cardiology and infectious disease. Over the years the American Heart Association guidelines have been modified with the advent of new clinical and laboratory evidence and experience. These guidelines have become more restrictive about antibiotic dosing and indications and are likely to become even more so as certain observations gain attention: the increasing resistance of viridans group streptococci (VGS) to amoxicillin and other beta-lactams, data from several studies that dental treatment procedures are rarely if ever a cause of bacterial endocarditis, that antibiotic prophylaxis as presently constituted does not appreciably reduce bacteremias (bringing into question just how prophylaxis works, if it does at all), and the need for careful assessment of cost-benefit and risk-benefit. Although no data presently exist, some estimation also is required of the

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effect of massive antibiotic prophylaxis, much of which is misguided, as a factor in the epidemic of global microbial resistance to antibiotics.

While these questions await evaluation, it is appropriate to examine their potential impact on antibiotic prophylaxis from the view of evidence-based medicine. It also is important to explore the rationale for various clinical situations in which antibiotic prophylaxis may be advocated but for which there are no official guidelines. This information will then allow dental practitioners to make informed empirical decisions in their best clinical judgment based upon the theory and reality of antibiotic prophylaxis.

Principles of antibiotic prophylaxis

Antibiotic prophylaxis is the use of antibiotics to prevent infections. In virtually all situations the infection likely will not occur, but prophylaxis may be justified if the infection to be prevented is common but not fatal or is rare but carries an unacceptably high morbidity or mortality rate [1]. The inattention to the basic principles of prophylaxis as formulated more than 40 years ago has led to massive antibiotic overuse.

The pioneering efforts of Polk, Burke, Stone, and Weinstein [1–5] provided for the rational use of antibiotic prophylaxis (Box 1): the health benefits must outweigh the risks, the cost-benefit ratio must be acceptable, the antibiotic must be in the blood or target tissue before the onset of the surgery or bacteremia, an antibiotic-loading dose should be used to attain high blood/tissue concentrations, the choice of the antibiotic should be based on the single most likely microorganism to cause an infection, and the antibiotic should be continued only as long as microbial contamination of or from the operative site continues. The contraindications to antibiotic prophylaxis include: the at-risk group to potentially benefit from prophylaxis cannot be narrowly defined to prevent overuse of antibiotic prophylaxis, prophylaxis is too random in efficacy to be reliable or proof of efficacy is too limited, the bacteremia to be prevented is too seldom a proximate cause of disease, and prophylaxis is directed against any or all potential pathogens rather than the colonization of a single microbial pathogen [2,3,5].

The indications for surgical prophylaxis are: clean-clean surgery where the risk of infection is remote but its potential consequences are grave or in clean-contaminated surgery where the likelihood of infection is great but is seldom fatal, to prevent contamination of a sterile area, where infection is unlikely but is associated with major morbidity, in surgical procedures with high infection rates, and during implantation of prosthetic material [6–8]. The adverse effects of antibiotic prophylaxis are: increased risk of antibiotic toxicity or allergy, increased risk of superinfection (development of a new infection while attempting to treat or prevent a primary infection), selection of antibiotic-resistant microorganisms, and induction of antibiotic resistance gene expression or transfer [9].

Box 1. Principles of antibiotic prophylaxis

Indications:
The infection to be prevented is common but not fatal or is rare but carries and unacceptably high mortality rate [1]

Criteria for use:
1) The health benefits must outweigh the antibiotic risks
2) The cost-benefit ratio must be acceptable
3) The antibiotic must be in the blood/target tissue before surgery or bacteremia
4) An antibiotic loading dose should be used
5) The choice of the antibiotic should be made on the single most likely microorganism to cause an infection
6) The antibiotic should be continued only as long as the microbial contamination of or from the operative site continues [1–4]

Contraindications:
1) The at-risk group to potentially benefit from prophylaxis cannot be narrowly defined so as to prevent overuse of prophylaxis
2) Prophylaxis is too random in efficacy to be reliable or proof of efficacy is too limited
3) The bacteremia to be prevented is too seldom a cause of disease
4) Prophylaxis is directed at any/all potential pathogens rather than the colonization of a single microbial pathogen [2,3,5]

Indications for surgical prophylaxis:
1) Clean-clean surgery where the risk of infection is remote but its potential consequences grave or in clean-contaminated surgery where the likelihood of infections is great but seldom fatal
2) To prevent contamination of a sterile area
3) Where infection is unlikely but is associated with major morbidity
4) In surgical procedures with high infection rates
5) During implantation of prosthetic material [6–8]

Adverse effects:
1) Increased risk of antibiotic toxicity or allergy
2) Increased risk of superinfections
3) Selection of antibiotic-resistant microorganisms
4) Induction of resistance gene expression or transfer [9]

Based on these principles, the use of antibiotics to "prevent" postoperative complications from dental treatment procedures by giving the antibiotic after treatment completion is inappropriate as the drug is not in the system before microbial contamination. Generally, it is done to "prevent" infection by any and all potential pathogens, which is another violation of principle, and often the drug is continued for many days after the procedure, which allows for selection of resistant bacteria or resistance gene expression or transfer. Orofacial infections after dental treatment procedures are uncommon, are rarely grave with high associated morbidity or mortality, and occur in a highly contaminated area. The only possible indication for surgical antibiotic prophylaxis in the oral cavity is implant placement. No clinical studies have adequately documented the efficacy of perioperative (begun before and stopped shortly after the surgery) antibiotic prophylaxis in the prevention of orofacial infections.

Bacteremias and the oral cavity

That bacteremias arise from the oral cavity on a daily basis is well established. The magnitude of the bacteremia after various oral manipulations (oral hygiene procedures, dental treatment) is of a "low-grade" or "transient" variety, usually consisting of 1 to 12 colony-forming units (cfus) per milliliter of blood (a single microbe that can be the progenitor of a colony of bacteria) [10]. Although the incidence of bacteremias is well documented, data on the precise microorganisms and their concentrations present in the blood sample are often lacking. It is important to know what percentage of these are viridans group streptococci (VGS) and which are pathogens associated with periodontal disease, because the former cause approximately 25% of all cases of bacterial endocarditis and the latter are associated with fewer than 150 cases of endocarditis in the entire medical literature. It is widely stated that the blood is rapidly cleared of the bacteremia within 15 to 50 minutes but this depends on whether the bacteremia was ongoing or simply due to a very short procedure such as dental flossing. Because the lungs, spleen, liver, and reticuloendothelial system are very efficient in removing microorganisms from blood, it is probable that many of these bacteremias are of a much shorter duration than 15 minutes.

The incidence (not magnitude) of bacteremias with various dental treatment procedures has been calculated to be: tooth extraction (40%–89%), periodontal surgery (36%–88%), scaling and root planing (8%–80%), simple prophylaxis (0%–40%), buccal local anesthetic injection (16%), intraligamentary injection (97%), rubber dam or matrix and wedge placement (9%–32%), and endodontic treatment (0%–15%) [11–13]. The incidence of bacteremias from oral hygiene procedures or chewing is: tooth brushing (0%–26%), dental flossing (20%–58%), wooden cleansing devices

(20%–40%), water irrigation devices (7%–50%), and mastication (17%–51%) [11,14–17]. Because the lymphatics and not the blood vessels may be the primary means of entry of oral bacteria into the blood, mastication may be the most important mechanism for oral bacteremia induction [14,18].

Guntheroth [14] and Roberts [19] have calculated that the likelihood of a bacteremia arising from normal daily living activities is 1000 to 8000 times greater than from a dental treatment procedure. Others have stated that the incidence and magnitude of oral metastatic bacteremias are directly proportional to the amount of gingival inflammation and periodontitis present in the individual [11,15,16]. However, substantial documentation has shown that the presence or absence of dental disease may have little to do with the advent of positive bacterial blood cultures [20]. Others have found no significant correlation between the magnitude of trauma and the incidence of oral bacteremias [21].

Given the data presented earlier, it becomes impossible to determine causality in any case of endocarditis purported to be the result of dental treatment unless the organism is genetically identified as the same in the infected cardiac valve as in the mouth. VGS are ubiquitous inhabitants of the GI, GU, and pharyngeal tracts as well as the skin and conjunctiva. Furthermore, it is equally impossible to determine if the bacteremia originated from the dental treatment or as a result of daily oral hygiene activities either before or after the dental treatment. Coupling this data with the recent epidemiologic evidence that dental treatment procedures are not associated with endocarditis should sound the death knell for the persecution of dental health professionals as a cause of bacterial endocarditis [22,23].

Oral microorganisms and endocarditis

The previous discussion is not meant to preclude the role of oral bacteria in causing bacterial endocarditis but simply to document that dental treatment is rarely the cause. Approximately 25% of all cases of endocarditis are caused by VGS, an incidence that has declined from 40% over the past few years [24]. It is highly likely that a vast majority of these cases arise from daily oral hygiene and other activities; however, the claim that periodontal disease, particularly periodontitis, is a significant factor in endocarditis causation is not substantiated by clinical data. The number of cases of bacterial endocarditis reported in the literature caused by periodontal pathogens is: 102 cases due to *Actinobacillus actinomycetemcomitans* [25], 2 cases due to *Prevotella oralis* [26,27] and 1 to *Prevotella bivia* [28], 1 due to *Bacteroides melaninogenicus* [28], 5 due to *Veillonella* (*dispar* and *alcalescens*) [29], with none reported due to *Porphyromonas* species. These obligate anaerobes are not likely to survive well in the highly oxygenated blood of the heart and also may not possess the surface

adhesion factors that are a major factor in the propensity of streptococci and staphylococci to stick to surfaces (cardiac valves).

Peptostreptococci are a very rare cause of endocarditis with *Peptostreptococcus micros* found in the oral cavity but *Peptostreptococcus magnus* only in the GU tract [30]. Only 10 cases of *Actinomyces* endocarditis of human origin have been reported and may be associated with intrauterine device use [31,32]. As of 1997, approximately 50 cases of *Lactobacillus* endocarditis were reported [33]. Nutritionally variant streptococci (NVS) that require pyridoxal (vitamin B6) for growth have been removed from streptococcal speciation and are now considered a separate species consisting of *Abiotrophia adiacens*, *A defective*, and *A elegans* [34]. *Abiotrophia* are commonly resistant to amoxicillin, difficult to culture, often associated with endocarditis relapse, and are a possible etiologic agent for culture-negative endocarditis [34].

Risk of endocarditis due to dental treatment procedures

It is unfortunate that published estimates regarding the percent of endocarditis cases caused by dental treatment procedures or poor oral hygiene have not been derived from sound clinical data. These estimates have ranged from 8% of bacterial endocarditis due to periodontal disease/dental disease without dental treatment having been performed [35], to a risk of 1/3000 to 1/5000 for each single dental procedure [36], to 19% to 30% of endocarditis resulting from dental treatment procedures [37]. Rarely do endocarditis epidemiologic studies assiduously attempt to determine the incubation period (from the onset of the bacteremia to the onset of symptoms) for VGS endocarditis: 50% within 7 days and 84% within 14 days [38]. From malpractice litigation involving the author as an expert witness, this incubation period has been alleged to be between 1 and 270 days. Also in the approximately 250 to 300 cases of bacterial endocarditis examined by the author only 2 to 3 have occurred within this 7- to 14-day incubation period but all have been ascribed to dental treatment by a physician or nurse during the patient's hospital stay, thereby initiating malpractice litigation.

In contrast to these allegations, several evidence-based, case-control studies strongly suggest that no association exists between dental treatment and endocarditis [22,23,39]. These studies do, however, document a strong association between cardiac valve pathology and endocarditis risk. In the classic study by Steckelberg and Wilson [40], the incidence of infective endocarditis in the general population is 1.7 to 4.9 cases per 100,000 person years but rises to 380 to 440 cases per 100,000 person years for rheumatic heart disease, 308 to 630 per 100,000 person years for cardiac valve prostheses, 300 to 740 per 100,000 person years for previous endocarditis, a mean 120 cases per 100,000 person years for congenital heart disease on

down to 52 cases per 100,000 person years for mitral valve prolapse with regurgitation.

If certain assumptions are made, then a reasonable estimate of the absolute risk for acquiring bacterial endocarditis can be made (Box 2). If 250 million people in the United States visit the dentist on average 1.6 times per year (400 million visits per year) and the incidence of infective endocarditis is 11,200 cases annually in the United States (population of 280 million with a risk rate of 4,100,000 per year) with 25% caused by VGS, then the absolute risk is 1/142,258 in the general population for VGS endocarditis if all are caused by dental treatment. If it is further assumed that only 1% of all VGS endocarditis is caused by dental treatment (112 cases annually), then the absolute risk rises to 1/14,258,714 in the general population with no known cardiac risk factors. The absolute risk for endocarditis due to a single dental treatment episode rises substantially in those with cardiac risk factors for endocarditis (worst case scenario): previous endocarditis (1/95,058), cardiac valve prostheses (1/114,069), rheumatic heart disease (1/142,258), congenital heart disease (1/475,290), and mitral valve prolapse with regurgitation (1/1,096,824). These values are only approximations but indicate that the absolute risk for acquiring bacterial endocarditis from a single dental treatment episode in at-risk patients is extremely low, ranging from 1/95,058 for a patient with a history of previous endocarditis to 1/1,096,824 in a patients with mitral valve prolapse with regurgitation. Future guidelines on antibiotic prophylaxis should consider these absolute risks carefully and, if generally valid, use them to produce cost-benefit and risk-benefit determinations particularly regarding penicillin allergy and the effect of prophylaxis on global microbial resistance to antibiotics.

Antibiotic prophylaxis and bacteremia reduction

Numerous studies have indicated that antibiotic prophylaxis reduces bacteremias after the onset of dental treatment [41], but as yet there is no explanation as to how drugs (antibiotics) that work so slowly (hours) eliminate bacteremias so quickly (seconds to minutes). The penicillins and cephalosporins require dividing bacteria to prevent the final transpeptidation reaction responsible for the formation of the bacterial cell wall. If the organism is not dividing, the beta-lactams have no activity. It is unreasonable to expect that all VGS will suddenly begin to divide as soon as the dentist places the forceps on the tooth to be extracted so that amoxicillin in the blood can do its work. Similarly bacteriostatic antibiotics (macrolides, clindamycin) take hours to produce their effects by inhibition of bacterial ribosomal protein synthesis.

Studies in Sweden indicate that neither penicillin V, amoxicillin, cefaclor, erythromycin, nor clindamycin given orally 1 to 1.5 hours before dental

Box 2. Absolute risk rate (ARR) for bacterial endocarditis from a single dental treatment appointment utilizing certain assumptions*

General population

1) Risk of 4/100,000 person years: 11,200 annual cases (280 million total population)
2) VGS causes 25% of all endocarditis (2800 cases annually)
3) If all VGS endocarditis caused by dental treatment: ARR = 1/142,578 (400,000,000 million visits per 2800 cases of VGS endocarditis)
4) If 1% of VGS endocarditis (28 cases) caused by dental treatment: ARR = 1/14,258,714 per dental visit

At-risk populations [40]: 1% caused by dental treatment

1) Previous endocarditis: 1/95,058 ARR
2) Cardiac valve prosthesis: 1/114,069 ARR
3) Rheumatic heart disease: 1/142,258 ARR
4) Congenital heart disease: 1/475,290 ARR
5) Mitral valve prolapse with regurgitation: 1/1,096,824 ARR

**Assumptions:*

1) 250 million population with 1.6 dental visits annually (400 million annual visits); 11,200 annual cases of infective endocarditis in the United States with 25% caused by viridans group streptococci (2800 cases); 1% of viridans group streptococcal endocarditis caused by dental treatment (28 cases annually)
2) Annual risk of endocarditis [40]:

General population (GP): 4/100,000 person years (range 1.7–4.9)
Previous endocarditis: 600/100,000 person years (150 times > GP)
Cardiac valve prosthesis: 500/100,000 person years (125 times > GP)
Rheumatic heart disease: 400/100,000 person years (100 times > GP)
Congenital heart disease: 120/100,000 person years (30 times > GP)
Mitral valve prolapse with regurgitation: 52/100,000 person years (13 times > GP)

extractions reduce bacteremias significantly, using the lysis filtration method under anaerobic conditions [42–44]. It has then been postulated that antibiotic prophylaxis may prevent endocarditis not by blood bactericidal activity but rather by preventing adherence of the microbes to the valvular

vegetation or by eliminating bacteria once attached to the damaged valves [45,46]. The reduced attachment may occur via beta-lactam alteration of the cell wall and adhesion expression, but such a mechanism would not likely apply to bacteriostatic agents. The likelihood of antibiotics killing or inhibiting microorganisms once they have attached to the nonbacterial thrombotic vegetation (endocarditis) on the valve and their entrapment in successive layers of platelets and fibrin seems remote.

Best case scenario for endocarditis prevention

Kaye [47] has calculated that antibiotic prophylaxis in a best case scenario may prevent 10% of all bacterial endocarditis cases. Studies in the Netherlands have indicated that antibiotic prophylaxis may prevent 5.7% of all native valve endocarditis and 3.8% of all prosthetic valve endocarditis cases, which would then prevent (if 49% efficacy is presumed) five endocarditis cases per year in the Netherlands with a population of 14.5 million [48,49]. If extrapolated to the United States, this would be 50 cases of endocarditis per year, assuming 145 million persons at risk. Optimal antibiotic prophylaxis might prevent 240 to 480 cases of endocarditis annually in the United States [45].

Viridans group streptococci antibiotic resistance

Another of the many assumptions underlying antibiotic prophylaxis particularly for the prevention of endocarditis is that VGS remain uniformly sensitive to the beta-lactams, macrolides, and clindamycin. In a cohort of Japanese children at high risk for bacterial endocarditis, 31.7% of the VGS exhibited resistance at minimal inhibitory concentrations (MICs) ranging from 4 to16 μg/mL [50]. In the United States, 40% to 50% of sampled VGS were resistant to penicillin at MICs $\geq$0.25 μg/mL [51] and in a survey of 43 US medical centers in 1993–1994, 352 VGS blood cultures exhibited a resistance rate of 13.4% at MICs of $\geq$4 μg/mL (high resistance) and 42.9% at MICs of 0.25 to 2.0 μg/mL (intermediate resistance) [52].

In 207 blood isolates, 35% of *Streptococcus oralis*, 16% of *Streptococcus mutans*, 6% of *Streptococcus salivarius*, and 3% of *Streptococcus sanguis* exhibited penicillin resistance at $\geq$4.0 μg/mL, whereas penicillin-resistant *Streptococcus oralis* displayed a 20% to 50% resistance to clindamycin [53]. In Spain, high-level resistance to penicillin occurred in 16% of *Streptococcus mitis*, 6% of *Streptococcus salivarius*, and 3% of *Streptococcus sanguis* [54]. In 139 cultures of VGS isolated from mixed orofacial infections, 23% were resistant to penicillin G, 45% to erythromycin, and 46% to clindamycin [55]. It seems that oral microbial resistance to common antibiotics may impair not only the management of acute orofacial infections but also the prevention of endocarditis.

Penicillin allergy

It was not until the mid-1980s that any attention was paid to the adverse effects of antibiotic prophylaxis particularly regarding serious penicillin allergy. A precise determination of the rate of major allergic reactions to penicillin (anaphylaxis, angioedema, exfoliative dermatitis, bronchospasm) is difficult to ascertain, but estimates have ranged from 0.04% to 0.11% [56] to 0.2% [57] to a fatality rate of 1 per 60,000 courses of penicillin (16 per million exposures) [58]. Some have suggested a serious reaction rate of 1 per 2000 to 2500 penicillin exposures [59]. It has been estimated conservatively that penicillin allergy is responsible for 400 to 800 annual deaths in the United States, and if 240 to 480 endocarditis cases could be prevented annually, then penicillin prophylaxis might conceivably result in a net loss of life [60,61]. This is particularly likely with VGS endocarditis, because the fatality rate is $<10\%$.

Assuming an endocarditis incidence rate of 11 to 50 per million persons per year, a 25% to 40% mortality rate from endocarditis, and a 16 per million population mortality rate from penicillin anaphylaxis, Pallasch [60] calculated that the death rate from endocarditis exceeded that from penicillin only in the highest incidence (50 per million) and highest mortality rate (40%). Tzukert et al [61] determined that 1.36 people per million population are likely to die from penicillin anaphylaxis during endocarditis prevention, whereas only 0.26 deaths per million population are the result of dental treatment–induced endocarditis [61].

Financial aspects of antibiotic prophylaxis

Some investigators have calculated that the use of antibiotic prophylaxis to prevent bacterial endocarditis is cost effective [41]. Others have calculated that on a strict economic basis, more than 3 to 5 million prophylaxis dose regimens annually in the United States to prevent endocarditis may not be cost effective (at the old nine amoxicillin dose of the 1990 AHA guidelines) as the cost of the antibiotics exceeds the cost of the treatment of endocarditis [62], Under the 1990 AHA dosing regimen with 200 million adults visiting the dentist 1.6 times per year and 5% requiring endocarditis prevention prophylaxis, the prevention of 32 fatal endocarditis cases (assuming all were caused by dental treatment) would require 16 million regimens of amoxicillin prophylaxis at \$6.00 per regimen or \$96,000,000, translating to \$3,000,000 per life saved (assuming antibiotic prophylaxis was 100% effective). The cost to prevent a single non-fatal case (assuming VGS endocarditis is 10% fatal) would then be \$300,000. If the cost to treat each endocarditis case is \$46,000 [62], then the cost of prevention of VGS endocarditis is approximately \$250,000 greater than treatment under the 1990 regimens. With the reduction of the dose to 2 g in the 1997 AHA guidelines, the costs of prevention have been substantially reduced but

would still likely be in the range of $800,000 to $1,000,000 per life saved and $80,000 to $100,000 per case prevented. One can argue about the precise financial costs of endocarditis prevention but not that such costs are substantial. The economic burden becomes much more significant when antibiotic prophylaxis is used inappropriately [41].

What to do during this time of reappraisal

It is not for the individual clinician to decide on the basis of these questions how established guidelines should now be modified. Although it may be true that antibiotic prophylaxis is more for the benefit of the doctor (prevention of malpractice litigation) than the patient (prevention of infections), the issue of guideline modification should be left to the experts. The recommendations of the American Heart Association regarding antibiotic prophylaxis for the prevention of bacterial endocarditis [63] and those of the American Dental Association/American Academy of Orthopedic Surgeons [64] regarding patients with prosthetic joints are still appropriate (with the provision that the best clinical judgment of the practitioner in any specific situation is paramount) [63].

Unfortunately the practitioner still commonly encounters requests from our medical colleagues for antibiotic prophylaxis for dental patients supposedly "at-risk" from oral metastatic infections. Such requests include patients with indwelling catheters, those with arterial stents or grafts or solid organ transplants, those that are immunocompromised, and increasingly those with breast or penile implants. The evidence for any significant hematogenous infections in such patients with oral microorganisms is extremely limited or nonexistent [41,65]. Equally there is no evidence that antibiotic prophylaxis is effective in the prevention of infections in these patients and any such use of antibiotic prophylaxis would very likely contradict the principles listed in Box 1. As for nonvalvular cardiovascular infections of pacemakers, implantable cardioverter defibrillators, cardiac or peripheral vascular stents, and prosthetic vascular grafts, there is no convincing evidence that microorganisms associated with dental or other invasive procedures cause infection of these nonvalvular devices at any time after implantation. Antibiotic prophylaxis is not routinely recommended for these patients who undergo dental or other invasive procedures [65].

Patients without spleens do not require antibiotic prophylaxis before dental treatment, but a consensus may exist that immunocompromised patients with a white cell count <500 to 1000 might benefit from antibiotic prophylaxis [41]. There is not a single documented case of a genetically identical micro-organism from the oral cavity causing an infection of a breast or penile implant. If the attending physician requests antibiotic prophylaxis for such patients before dental treatment, the dentist can state that there is no medical reason for such a practice and suggest that the physician provide the prophylaxis. The same would hold true for a patient

with a prosthetic joint where after more than 20 years of study, there is not yet a single case report of a prosthetic joint infection caused by a dental treatment bacteremia with a micro-organism genetically identical in the oral cavity and the joint. Such an association has been established with oral sepsis but not dental treatment–induced bacteremias [66].

The 1997 ADA/AAOS guidelines on antibiotic prophylaxis for prosthetic joint patients has been updated in 2003 and remains essentially the same with an added patient information handout [67].

Summary

The age of antibiotic prophylaxis may be receding into its twilight years because the assumption upon which it was based has not proved generally true. Although antibiotics treat infections, limited benefit has been demonstrated in preventing infections. These are two entirely different biologic entities, a distinction which appears to have gone unappreciated by many for more than 50 years. If the principles of antibiotic prophylaxis established more than 40 years ago had been assiduously followed, many of its abuses could have been avoided. This may not have stopped our legal colleagues, but it would have been worth an effort on behalf of our patients.

It is likely that the massive overuse of antibiotics as litigation prevention has contributed to the global epidemic of antibiotic-resistant micro-organisms and an unknown number of serious adverse effects to the antibiotics themselves. Even with this abuse, much money has still flowed from defendant to plaintiff. Substantial data exist that antibiotics do not prevent bacteremias. The absolute risk rate for bacterial endocarditis after dental treatment even in at-risk patients is very low. Antibiotic prophylaxis for surgical infections requires specific dosing schedules (perioperative surgical prophylaxis) to be successful. Hopefully the difficulties presented herein regarding antibiotic prophylaxis will lead to their more enlightened use in the future.

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