Antibiotic Prophylaxis for Dental Procedures
Are We Drilling in the Wrong Direction?

Peter B. Lockhart, DDS

The possibility that bacteremia from the mouth could cause infective endocarditis (IE) was first suggested >100 years ago, and it was later reinforced by others who targeted the viridans group streptococci from poor oral hygiene and dental extractions.1–3 These observations, along with the advent of antibiotics, eventually led to the first guidelines from the American Heart Association (AHA) in 1955. Antibiotic prophylaxis (AP) became the primary focus for prevention of IE and a standard of care for countries around the world. Controversy concerning efficacy and safety issues has existed for >30 years, and there has been a progressive reduction in the patient populations and the procedures suggested for AP since that time. Of concern, and in spite of a decreasing emphasis on AP for cardiac patients, upwards of 25 noncardiac patient populations are recommended for AP by some clinicians out of concern for systemic infections that might originate from dental procedures (eg, prosthetic joints).4

Significant differences in recommendations from experts in the United States, United Kingdom, and other countries over the years highlight the lack of convincing data to either support or reject this practice. The National Institute for Health and Clinical Excellence (NICE) in the United Kingdom issued new recommendations in 2006, which took the bold step of eliminating AP altogether.5 Current (2007) AHA guidelines narrow the focus to only 4 cardiac groups at higher risk for a bad outcome from IE but who represent ≈10% of all people at risk for IE.6 The AHA-defined moderate-risk groups represent ≈90% of people at risk for IE, all of whom were recommended for AP before 2007. There are, therefore, no preventive guidelines for the millions of people in the United States at risk for IE but who are not felt to benefit from AP.7

Although there is understandable concern on the part of some cardiologists and some patients about eliminating AP, a recent retrospective study by Thornhill et al8 suggests that AP has no significant impact on the incidence of, or death from, IE. The authors point out that these findings may not apply to patients in the higher risk groups because ≈20% of physicians in the United Kingdom may continue to use AP in spite of NICE guidelines that mandate a cessation for this practice, unless the patient requests it. This group did find a modest increase in the incidence of IE over their 10-year period as a result of both streptococci and staphylococci.

Support for the role of oral bacteria as a significant cause of IE comes largely from 2 sources. The best evidence for this association is the frequency with which specifically oral bacterial species are cultured from the blood of patients with community-associated IE (CA-IE). The literature suggests that 20% to 65% of cases of IE worldwide result from bacteria that can be found in, or are exclusive to, the mouth.7,9

This wide range of cases of IE attributed to oral bacteria stems, in part, from the use of standard biochemical rather than molecular methods of bacterial identification.10 For example, reports of viridans group streptococci are not specific enough to implicate the mouth as the source. Other supporting documentation comes from >75 years of bacteremia studies of dental procedures and other manipulations of gingival tissues.3 These studies suggest a wide range of incidence of bacteremia from species known to cause IE, and one must conclude that oral bacteremia is likely to occur with the majority of dental office visits. These studies use surrogate measures of risk for IE (ie, incidence, duration, nature, and magnitude of bacteremia), but they have driven the focus on AP for dental procedures since the 1950s.

Retrospective case-control studies provide a closer look at this relationship. Strom et al11 contacted patients who had been hospitalized (cases) for IE at 1 of 54 hospitals in the Philadelphia area and compared them with matched community residents (controls). His group found that recent dental treatment was no more frequent in cases than in controls, and they concluded that dental treatment did not seem to be a risk factor for CA-IE. The study by DeSimone12 et al in this issue of Circulation moves us 1 step closer to a better understanding of this question of efficacy for AP by conducting the first population-based study in the United States to determine the impact of the 2007 AHA guidelines. They focused on cases of IE from presumed oral streptococci over a 12-year period and found no increase in viridans group streptococcus IE in the 2+ years since 2007. Of interest, 2 of the 3 patients who developed IE from viridans group streptococcus after 2007 had not been to the dentist in the previous 6 months, and the third patient had AP before a recent dental office procedure.

Case-control, population-based, and other epidemiological investigations often have methodological weaknesses that soften their impact: (1) small sample size and power; (2) subject recall bias; (3) demographics that may not be representative of the general population; (4) incubation time...
frames that are too long for IE; (5) the imperfect nature of hospital and national databases on admission and discharge coding and nonspecific ICD-9 coding of bacterial species. Nevertheless, results from well-designed studies are important because they provide additional evidence that the well-intentioned focus on AP may be misdirected.

The AHA, Institute of Medicine, NICE, and other professional groups have called for studies that would resolve this longstanding question concerning the efficacy of AP and clarify the role of poor oral hygiene and resulting periodontal disease in the pathogenesis of IE.1,2,5,6,13 A prospective, randomized, double-blind study of AP in people at risk for CA-IE, however, has significant obstacles: (1) there are ethical and legal concerns about randomizing people in the AHA higher risk group to a placebo; (2) such a trial would only address the 10% of people currently recommended for AP in the United States; and (3) given the rarity of IE, it has been estimated that upwards of 30,000 people at risk would be needed to detect a clinically important AP treatment effect, and the cost of this trial would be prohibitive.

Transient bacteremia frequently occurs as a result of dental plaque accumulation, evolving to a dense mat of oral bacteria around the teeth which cross the inflamed periodontal pocket tissues to the circulation. Clearly, this must be the main source and portal of entry for the oral bacterial species that cause upwards of 25% of cases of CA-IE. Current science strongly suggests that poor oral hygiene and periodontal disease are far greater risk factors for the development of oral bacteria–related IE than invasive dental procedures. The largest study to date compared tooth extraction, a highly invasive dental procedure, with tooth brushing as a common, naturally-occurring source of bacteremia.14,15 This group found that the incidence of bacteremia from tooth brushing (32%) was high enough to strongly suggest that bacteremia from various activities of daily living (chewing food as well) may occur hundreds of times more often than bacteremia from dental office procedures. Indeed, it has been suggested that some individuals may generate bacteremia for 90 hours each month from such physiological causes,16 by comparison with dental office procedure–generated bacteremia of 1 to 2 times per year on average. This study provides unique documentation of a strong association between 3 oral hygiene, periodontal disease, and oral bacteria are associated with CA-IE. These data would improve our understanding of risk factors and refocus efforts on prevention of IE to improve oral hygiene and preventing periodontal disease. This information has the potential to reduce the overall incidence of CA-IE, it would be immediately transferable to everyday clinical practice, and it would inform future AHA and other international guidelines on preventive strategies for IE.

Disclosures

None.

References


Key Words: Editorials cardiovascular diseases echocardiography heart diseases
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Circulation. 2012;126:11-12; originally published online June 11, 2012;
doi: 10.1161/CIRCULATIONAHA.112.115204

Circulation is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
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Print ISSN: 0009-7322. Online ISSN: 1524-4539

The online version of this article, along with updated information and services, is located on the
World Wide Web at:
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