

[\[Print Version\]](#)

TABLE OF CONTENTS

[\[REFERENCES\]](#)

The Angle Orthodontist: Vol. 70, No. 1, pp. 15-15.

Investigation of Bacteremia Following Orthodontic Debanding

Mark T. Drangsholt, DDS, MPH^a

This article is important because little scientific work has been done to investigate the effect of common orthodontic procedures on the presence of measurable bacteria in the bloodstream. After approximately 1000 case reports and case series, we now know that the consequence of displaced oral bacteria from dental manipulations can cause death or serious illness from bacterial endocarditis.¹ Bacteremia is a necessary intermediate in the causal pathway of endocarditis, but it must act in conjunction with other risk factors, such as the presence of a damaged heart valve. Many other factors may determine whether bacteremia will lead to endocarditis in a person with cardiac pathology. These factors, as extrapolated from animal models, include the size of the bacteremia, the ability of the bacteria to attach, whether other infections are present, and the immunocompetence of the person.²

Studies of bacteremia after procedures have demonstrated their potential to cause bacterial endocarditis, but it is a very rough approximation of the risk. What we really should be interested in is whether orthodontic procedures increase the risk of endocarditis or other systemic diseases. For example, suture removal, periodontal probing, intraligamental injections, and other minor dental procedures have been shown to cause bacteremia. However, few if any cases of endocarditis associated with these procedures have been reported in the literature over the past 70 years. High frequency of bacteremia does not mean high endocarditis risk. However, procedures that create no measurable bacteremia are generally disregarded as potential causes of endocarditis. More significantly, several controlled epidemiological studies have shown an increased risk of endocarditis after some types of dental procedures that generally cause bacteremia, such as dental extractions, root canal therapy, and periodontal scaling. Performing these analytic epidemiological studies is difficult, costly, and complex, hence, despite their "proxy" nature, studies of the incidence of bacteremia are still needed.

Four case reports of endocarditis temporally associated with orthodontic treatment have been published: Hobson and Clark,³ Dajani,⁴ and Biancianiello and Romero⁵ (2 cases). Each of these cases was associated with minor adjustments, not with molar banding or debanding. Although these cases may be causally unrelated to orthodontic treatment, they are important because there are many minor dental procedures that have never been reported to be associated with bacterial endocarditis, such as intraoral local anesthesia and dental impressions. There have now been at least 4 controlled studies of the risk of bacterial endocarditis associated with dental treatment: Strom et al⁶, Drangsholt,¹ Lacassin et al⁷ and van der Meer et al.⁸ Each of the studies showed a subset of dental procedures that put patients at a greater risk for endocarditis, although Strom et al⁶ conservatively interpreted their data as not increasing the risk. None of these studies reported minor dental procedures, such as orthodontic banding or debanding, as a procedure associated with the development of endocarditis.

The methodology of this study appears to be standard and sound. I believe this paper is a valuable contribution to the orthodontic literature because it illustrates that orthodontic debanding is associated with a low rate of measurable bacteremia, and a concomitant low risk of endocarditis in an 'at risk' person. It may remain prudent, however, to place patients 'at risk' for endocarditis on antibiotics before orthodontic banding and debanding until we have clear evidence that endocarditis is not caused by these orthodontic procedures.⁹

1. Drangsholt MT. A new causal model of dental diseases associated with endocarditis. *Ann Periodontol.* 1998; 3:184–196. [[PubMed Citation](#)]
2. Freedman LR. To prevent or not to prevent bacterial endocarditis—that is the question!. *Clin Infect Dis.* 1993; 17:195–197. [[PubMed Citation](#)]
3. Hobson , Clark JD. Infective endocarditis associated with orthodontic treatment: a case report. *Br J Orthod.* 1993; 20:241–244. [[PubMed Citation](#)]
4. Dajani AS. Bacterial endocarditis after minor orthodontic procedures. *J Pediatr.* 1991; 119:339–340. [[PubMed Citation](#)]
5. Biancaniello TM, Romero JR. Bacterial endocarditis after adjustment of orthodontic appliances. *J Pediatr.* 1991; 118:248–249. [[PubMed Citation](#)]
6. Strom BL, Abrutyn E, Berlin JA. et al. Dental and cardiac risk factors for infective endocarditis. A population-based, case-control study. *Ann Intern Med.* 1998; 129:761–769. [[PubMed Citation](#)]
7. Lacassin F, Hoen B, Lepout C, Selton-Suty C, Delahaye F, Goulet V, Etienne J, Briancon S. Procedures associated with infective endocarditis. A case control study. *Eur Heart J.* 1995; 16:1968–1974. [[PubMed Citation](#)]
8. van der Meer JT, Thompson J, Valkenburg HA, Michel MF. Epidemiology of bacterial endocarditis in the Netherlands. II. Antecedent procedures and use of prophylaxis. *Arch Intern Med.* 1992; 152:1869–1873.
9. Dajani AS, Taubert KA, Wilson W. et al. Prevention of bacterial endocarditis. Recommendations by the American Heart Association. *Circulation.* 1997; 96:358–366.

^aActing Instructor, Department of Oral Medicine, School of Dentistry, University of Washington, PO Box 357446, Seattle, WA 98195 (E-mail: drangs@u.washington.edu)