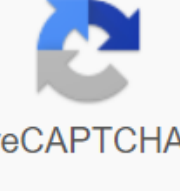


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The most common cause of endocarditis for dental, oral, respiratory or esophageal procedures is *S. viridans* (alpha-hemolytic streptococcus). Antibiotic regimens for the prevention of endocarditis are aimed at *S. viridans*, and the recommended standard prophylactic regimen is a single dose of oral amoxicillin. Amoxicillin, ampicillin and penicillin V are equally effective *in vitro* against alpha-hemolytic streptococcus; however, amoxicillin is preferred due to superior gastrointestinal absorption, which provides a higher and more resistant serum level. All doses shown below are administered once as a single dose 30-60 minutes before the procedure. Amoxicillin Adult dose: 2 g PO Pediatric dose: 50 mg/kg PO; Do not exceed 2 g/dose of Ampicillin Adult dose: 2 g IV/IM Pediatric dose: 50 mg/kg IV/IM; Do not exceed 2 g/dose of clindamycin Adult dose: 600 mg PO Pediatric dose: 20 mg/kg PO; do not exceed 600 mg/dose of cephalexine or other oral cephalosporin of the first or second generation at an equivalent dose (do not use cephalosporins in patients with a history of hypersensitive penicillin allergies of immediate type such as hives, angioedema, anaphylaxis) Adult dose: 2 g PO pediatric dose: 50 mg/kg PO; Do not exceed 2 g/dose of azithromycin or claritromycin Adult dose: 500 mg PO Pediatric dose: 15 mg/kg PO; Do not exceed 500 mg/dose of clindamycin Adult dose: 600 mg IV Pediatric dose: 20 mg/kg IV; Do not exceed 600 mg/dose of cephalosolin or ceftioxone (do not use cephalosporins in patients with a history of immediate hypersensitivity penicillin allergies such as hives, angioedema, anaphylaxis) Adult dose: 1 g IV/IM Pediatric dose: 50 mg/kg IV/IM; Do not exceed 1 g/dose article, see p 170

Infection endocarditis (IE) is a rare disease, but has devastating consequences; A significant proportion of patients develop heart failure or stroke or require valve surgery, with high hospital and 6-month mortality approaching 20% and 30%, respectively.¹ Due to significant morbidity and mortality, recommendations on preventive strategies were developed as far back as the 1950s. The recommendations were based on animal IE models along with the susceptibility of *in vitro* microorganisms, which are known to cause endocarditis. It has been shown that amoxicillin has had a significant impact on the incidence and duration of bacteriology after dental procedures.² However, clinical evidence for the prevention of endocarditis does not exist, and there has never been a randomized controlled trial to determine whether the use of antibiotics prevents IE. Thus, these recommendations were based not only on evidence, but also on an expert consensus on that prevention in at-risk patients is a better strategy than treating a disease with high morbidity and mortality. The guidelines of the American Heart Association on IE Prevention have evolved since the first iteration, published in 1955, including expanding the expansion To include patients with moderate risk and, subsequently, shorter durations of prevention.³ The latest version in 2007 represented a serious departure from previous guidelines because it no longer recommended the antibiotic prophylaxis system (AP) simply based on life risk for IE development and instead focused on those most at risk of adverse outcomes from IE. Reasons for the change in recommendations include the lack of randomized controlled trial data demonstrating the benefit of AP and the lack of observational data demonstrating a consistent link between IE procedures and development. In addition, the lack of evidence to support the profitability of apes, as well as recognition of the importance of antibiotic management in an era of increased antibiotic resistance, have contributed to a more conservative stance on AP. Finally, the cumulative risk of life developing IE from tooth brushing and mastication is estimated to be higher than from single tooth extraction.⁴ The expectation was that AP prescriptions would decrease after guiding changes, unchanged in the incidence of the viridans group streptococcus endocarditis. The European Society of Cardiology followed up in 2009 with similar recommendations from the American Heart Association,⁵ while in the United Kingdom, the National Institute for Health and Care Excellence approved a complete cap on AP regardless of risk in 2008.⁶ Studying the impact of these revised guidelines on changing AP prescribing patterns and IE prevalence is challenging. Most studies, as necessary, use environmental analyses to examine public health in relation to public health intervention or policy change. These studies are useful for generating hypotheses, but have a number of important limitations, including incomplete control over the confusion of variables and problems in determining whether temporary changes are caused by intervention of interests or other factors that change over time. Moreover, because they relate to public health, they cannot distinguish causality at the individual level. Previous studies have assessed the impact of these guidance changes on AP prescribing practices. For example, researchers in North America found a 64% decrease in AP prescriptions in moderate-risk patients and a significant decrease of 20% even in high-risk patients.⁷ Studies of the impact of these revised guidelines on IE incidence showed conflicting results. Several studies have shown no increase in EE incidence shortly after revising the guidelines.^{8,9} In contrast, a study looking at the U.S. Nationwide stationary sample found an age-adjusted annual increase in IE at 2.4% between 1998 and 2009, but no change in IE incidence caused by streptococcus when comparing 1998 in 2006 and 2008 to 2009.¹⁰ However, studies with a longer follow showed trend trends For example, a study using a nationwide stationary sample with a longer follow-up noted a significant increase in streptococcal (but not staphylococcal) IE from 2000 to 2007 to 2008 to 2011, although the official analysis of the point of change has not been implemented.¹¹ Similarly, in Germany, there was a relatively 26% increase in the annual level of IE after the 2009 European Society of Cardiology Guidelines , with a greater trend in the increase in annual prevalence in 2011-2014 compared to 2006 to 2010.¹² These last 2 studies were limited by the base data that did not capture the use of antibiotics and failed to distinguish between oral and other streptococcus.^{9,11,12} including enterococcus in some cases. As a result, these studies could not discern whether the infected organisms were likely to have been affected by the AP. In this issue of Circulation, Garg et al¹³ add to a growing set of epidemiological data by performing a population-based, cross-sectional time analysis in adults with moderate to high risk for IE between 2002 and 2014 in Ontario, Canada, to assess differences in the practice of prescribing AP and IE incidence. In adults ≥65 years, they found that AP significantly decreased in the moderate risk group for IE after changes in guidelines, with no significant changes in AP prescriptions in the high-risk group. Garg et al¹³ then assessed the incidence of IE in all adults in Ontario and found a significant increase for all at-risk groups over time. Analysis of the tone changes showed that significant changes in new episodes in both moderate and high-risk groups occurred in 2010, three years after the revision of the guidelines. Among the elderly, there has been no significant change in the proportion of cases caused by streptococcus vs. staphylococcus over time. In contrast, among the 18-64-year-old group, staphylococcus's share increased, while cases related to streptococcus decreased over time. Garg et al¹³ concluded that the increase in the number of hospitalizations due to IE, which occurred in both groups three years after the revision of the guidelines, is unlikely to be related to a change in the recommendation for the guidelines, since (1) the increase occurred only 3 years after the revision of the guidelines; (2) This increase occurred in both high- and moderate-risk groups, not just the moderate-risk group for which AP recommendations were amended; and (3) the relative proportion of streptococcal IE compared to staphylococcus IE has decreased over time, at least in young adults. Authors should be applauded for providing important information and contributing to a better understanding of the impact of changes in AP guidelines. Strong included a large study population, granular AP prescribing data for those patients ≥65 years, the inclusion of patients based on moderate to high risk for IE, the presence of some microbiological data, data, use tone analysis to determine the timing of changes in IE incidence. While the findings appear to be encouraging with regard to the current guidelines on IE prevention, they should be interpreted with caution. It is important to note that the finding that the ratio of streptococcus to staphylococcus remains unchanged over time among older adults is a concern, especially because the authors found that comorbidities that increase contact with health and the risk of staphylococcus infection increases over time. The data suggest that the proportion of IE cases caused by staphylococcus is increasing, with a decrease in cases caused by oral streptococcus in developed countries.¹⁴ Given the overall increase in IE in older adults without proportional changes in streptococcus vs. staphylococcus, it remains possible that the absolute growth of IE caused by streptococcus species is associated with a reduction in AP. The last point is relevant, given the recent findings that patients with bicuspid aortic valves and mitral valve prolapse (more than half with moderate or more mitral regurgitation) were found to have higher rates of viridococ group streptococcus and IE with suspected odontological origin than other IE.¹⁵ patients with a clinical profile similar to those at high risk. The authors propose to review the case of AP in patients with bicuspid aortic valves and mitral valve prolapse. In addition, there are a number of important limitations in the Garg et al¹³ study. First, there is no international classification of disease code characteristic of the viridans group streptococcus, the body is likely to be affected by the AP. Secondly, microbiological data were not available in 26% of cases. Thirdly, as in all environmental studies, cause and effect cannot be established. Future studies should ideally include granular data on underlying cardiac conditions and devices, invasive dental exposures over a predetermined period of time, whether patients have received AP, and microbial pathogenesis cases of IE incident, focusing on microorganisms specifically targeted at AP. Electronic medical records can provide an opportunity for large pragmatic clinical trials to evaluate these important factors. Preventing IE remains an empirical practice with uncertainty and contradictions. The cost of diagnosing and treating CV is high, and the disease comes at a high cost to the patient in terms of morbidity and mortality. In the absence of definitive data from randomized controlled trials, epidemiological studies provide important insights. Most of the data to date, including from Garg et al.¹³, support current data The AP's conduct in those at moderate risk, though whether there may be some subgroups¹⁵ that still benefit from the AP, has yet to be definitively determined. FootnoteReference1. Park LP, Chu VH, Peterson G, Skoutelis A, A, T, Bouza E, Tattevin P, Habib G, Tan R, Gonzalez J, Altclas J, Edathodu J, Fortes Si, Siciliano RF, Pachirat O, Kanj S, Wang A; and the international cooperation of endocarditis investigators (ICE). A proven risk score for predicting 6-month mortality in infectious endocarditis. J Am Heart Assoc. 2016; 5:e003016. doi: 10.1161/JAHA.115.003016LinkGoogle Scholar2. Lockhart PB, Brennan MT, Kent ML, Norton HJ, Weinrib DA. The effect of amoxicillin prevention on the morbidity, nature and duration of bacteriology in children after innumera and dental procedures. Circulation. 2004; 109:2878-2884. doi: 10.1161/01.CIR.0000129303.90488.29LinkGoogle Scholar3. Wilson W, Taubert SA, Gewitz M, Lockhart PB, Baddour LM, Leveson M, Bolger A, Cabell CH, Takahashi M, Baltimore RS, Newburger JW, Strom BL, Thani LY, Gerber M, Bonow RO, Pallasch T, Shulman ST, Rowley, Burns JC, Ferry P, Gardner T, and the American Heart Association Rheumatic Fever, and Council of the American Heart Association on Cardiovascular Diseases among Youth; Council of the American Heart Association for Clinical Cardiology; Council of the American Heart Association on Cardiovascular Surgery and Anesthesia; Interdisciplinary Working Group on the Study of the quality of care and outcomes. Prevention of Infectious Endocarditis: Guidelines of the American Heart Association: Guidelines of the American Heart Association rheumatic fever, endocarditis and kawasaki disease committee, Council on Cardiovascular Diseases in Young, and Council for Clinical Cardiology, Council on Cardiovascular Surgery and Anesthesia, as well as the quality of care and research results of the Interdisciplinary Working Group (published correction appears in circulation. 2007; 116:1736-1754. doi: 10.1161/CIRCULATIONAHA.106.183095LinkGoogle Scholar4. Brennan MT, Sussner HC, Fox PC, Pasteur BJ, Bahrani-Mougeot FC. 117:3118-3125. doi: 10.1161/CIRCULATIONAHA.107.758524LinkGoogle Scholar5. Habib G, Hoen B, Toreros P, Thuny F, Prendergast B, Vilacosta I, Moreillon P, De Jesus Antunes M, Thilen U, Lekakis J, Lengyel M, Muller L, Nabler CK, Nihoyannopoulos P, Moritz A, Zamorano JL, Guidelines of the ESC Committee on Practice, , diagnosis and treatment of infectious endocarditis (new version 2009). Task Force on the Prevention, Diagnosis and Treatment of Infectious Endocarditis of the European Society of Cardiology (ESC). Eur Heart J. 2009; 30:2369-2413. doi: 10.1093/eurheartj/ehp285CrossrefMedlineGoogle Scholar6. Ritchie R, Wray D, Stokes T; The guidelines group. Prevention against Infectious Endocarditis: a summary of the NICE guide. BMJ. 2008; 336:770-771. Doi: Scholar7. Thornhill MH, Gibson TB, TB, E, Dayer MJ, Chu VH, Lockhart PB, O'Gara PT, Baddour LM. Antibiotic prophylaxis prevention and the incidence of endocarditis before and after the 2007 AHA recommendations. J Am Coll Cardiol. 2018; 72:2443-2454. doi: 10.1016/j.jacc.2018.08.2178CrossrefMedlineGoogle Scholar8. Duval X, Delahaye F, Alla F, Tattevin P, Obadia JF, Le Moing V, Doco-Lecompte T, Celard M, Poyart C, Strady C, Chirouze C, Bes M, Cambau E, lung B, Selson-Suty C, Hoen B; and the AEPPI training group. Temporary trends in infectious endocarditis in the context of changes in prevention guidelines: three consecutive demographic surveys. J Am Coll Cardiol. 2012; 59:1968-1976. doi: 10.1016/j.jacc.2012.02.029CrossrefMedlineGoogle Scholar9. Bickedel B, Wang Y, Kim N, Desai MM, Cugliarello V, Krumholz HM. Trends in hospitalization and endocarditis outcomes among Medicare beneficiaries. J Am Coll Cardiol. 2013; 62:2217-2226. doi: 10.1016/j.jacc.2013.07.071CrossrefMedlineGoogle Scholar10. Bor DH, Woolhandler S, Nardin R, Bruschi J, Himmelstein DU. Infectious endocarditis in the United States, 1998-2009: a nationwide study. PLoS one. 2013; 8:e60033. doi: 10.1371/journal.pone.0060033CrossrefMedlineGoogle Scholar11. Pant S, Patel NJ, Deshmukh A, Gowlala H, Patel N, Badheka A, Hirsch GA, Mehta JL. Trends in the incidence of infectious endocarditis, microbiology and valve replacement in the U.S. from 2000 to 2011. 2015; 65:2070-2076. doi: 10.1016/j.jacc.2015.03.518CrossrefMedlineGoogle Scholar12. Keller K, von Bardeleben RS, Ostad MA, Hobohn L, Munzel T, Konstantinides S, Lankeit M. Temporary trends in the prevalence of infectious endocarditis in Germany between 2005 and 2014. Am J Cardiol. 2017; 119:317-322. doi: 10.1016/j.amjcard.2016.09.035CrossrefMedlineGoogle Scholar13. Garg P, Co DT, Bray Jenkin KM, Lee L, Shariff SH. Infectious endocarditis hospitalization and antibiotic prophylaxis rates before and after the 2007 American Heart Association Review Guidelines. Circulation. 2019; 140:170-180. doi: 10.1161/CIRCULATIONAHA.118.037657LinkGoogle Scholar14. Murdoch DR, Corey GR, Hoen B, Miro JM, Fowler VG, Bayer AS, Karchmer AW, Olaison L, Pappas PA, Morelyon P, Chambers ST, Chu VH, Falco V, Holland DJ, Jones P, Klein JL, Raymond NJ, Read KM, Tripodi MF, Ullri R, Wang A, Woods, CW International Collaboration on Endocarditis-Promising Cohort Research (ICE-PCS) Investigators. Clinical presentation, etiology, and the result of infectious endocarditis in the 21st century: International Collaboration on Endocarditis-Promising Cohort Study. Arch Intern Med. 2009; 169:463-473. doi: 10.1001/archinternmed.2008.603CrossrefMedlineGoogle Scholar15. Segri-Reiris I, de Alarcón A, Muñoz., Martínez Selles M, González-Ramallo V, Miro JM, Falches C, González Rico C, Cortaharena Urcola X, Lepe JA, Rodríguez Alvarez R, Reguera Iglesias JM, Navas E, Dominguez Collaboration on endocarditis-grupo de Apoyo al-Manejo de la Endocardite infecciosa en Spain (GAMES). Infectious endocarditis in patients with bicuspid aortic valve or mitral valve prolapse. J Am Coll Cardiol. 2018; 71:2731-2740. doi: 10.1016/j.jacc.2018.03.534CrossrefMedgle Scientist

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