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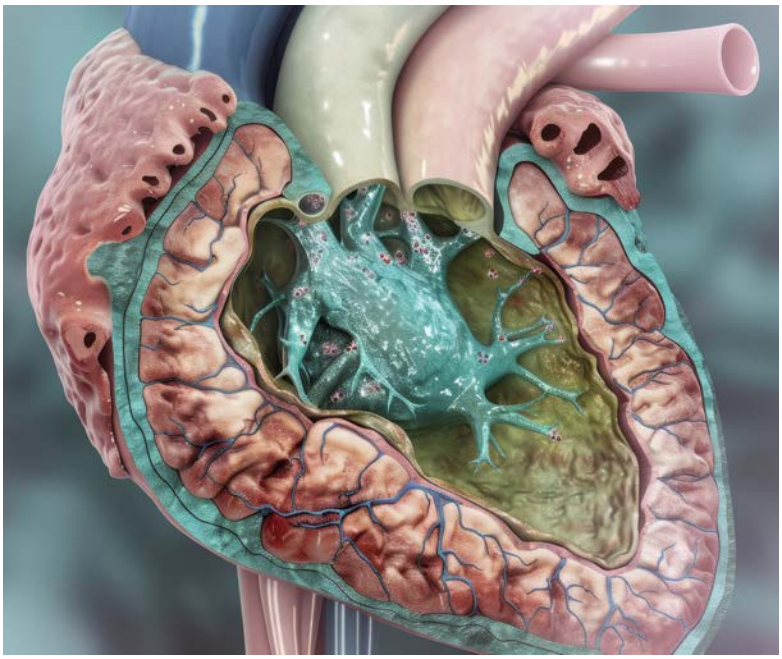

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Preventing acute and chronic endocarditis in dentistry

Looking beyond published antibiotic prophylaxis and protocols

A PEER-REVIEWED ARTICLE | by Eric S. Bornstein, DMD, BS

ABSTRACT

This course will examine the nature and progression of endocarditis along with peer-reviewed data from 1995 to 2024 concerning bacterial endocarditis and antibiotic prophylaxis in dentistry. The oral microbiology and the evolving antimicrobial treatments utilized to prevent bacterial endocarditis will be reviewed. Further, it will investigate the antibiotic safety profiles and comorbidities that may extend the subset of patients who should undergo prophylaxis beyond the published recommendations. Finally, this report will critically examine the potential need for new and expanded guidelines to increase patient safety and endocarditis prevention efficacy.

EDUCATIONAL OBJECTIVES

1. Describe the patient presentation of subacute bacterial endocarditis and outline the risk factors for subacute bacterial endocarditis associated with the provision of dental care
2. Describe the prophylactic antibiotics recommended to minimize the risk of subacute bacterial endocarditis during dental care delivery
3. Review the interprofessional team members' roles in preventing subacute bacterial endocarditis and prosthetic joint infection

Between 2007 and 2009, the American Heart Association (AHA) antibiotic prophylaxis (AP) guidelines to prevent bacterial endocarditis associated with dental care delivery underwent significant change, and similar changes occurred in Europe. The newly updated guidelines reduced the clinical conditions for which AP was recommended, focusing on only a select group of high-risk individuals—those with "cardiac conditions associated with the highest risk of adverse outcome from endocarditis for which prophylaxis with dental procedures is reasonable."[1] The new guidelines eliminated recommendations for AP prior to dental care for individuals with noncongenital, nonprosthetic heart valve disease. Further, the new guidance

reduced recommended AP for dental procedures to include approximately 90% fewer patients. Other regional, national, and international heart associations and numerous antibiotic stewardship programs praised these changes. However, following the changes in guidelines and their implementation, clinicians and researchers observed an increase in the incidence of bacterial endocarditis and subsequent patient deaths.[2,3]

By 2017, there was recognition from clinicians and policymakers regarding the increase in this deadly disease and stagnation of patient survival rates with current therapies for infective endocarditis (IE). Additionally, in the last 16 years, reports on oral microbiology have changed, with higher levels of pathogenic *Staphylococcus aureus* present than previously recorded.[4-7] Most recently, in 2021, the suggested antibiotic regimens for endocarditis prophylaxis for patients receiving dental care were amended by the AHA.[8] The recommendation for the use of the antibiotic clindamycin for individuals with a penicillin allergy was removed to prevent *Clostridium difficile* infections and was replaced with doxycycline.

The pathophysiology and disease progression of infective endocarditis

IE is a damaging and possibly fatal inflammation of the heart valves and chambers. Inflammation and infection occur after transient bacteremia. Transient bacteremia refers to the brief presence of bacteria in the bloodstream, typically following certain medical or dental procedures, that is swiftly cleared by the immune system without lasting impact. However, when a patient's immune system does not adequately clear microbial cells before the cells can attach to the lining of the heart valves, IE occurs.[9] The subsequent growth, vegetation, and inflammation resulting from bacterial adhesion and attachment can then cause endocarditis and cardiac damage. Bacterial vegetation can damage heart valves and lead to distal site infections, septicemia, and death.[10] Without prompt and adequate treatment, endocarditis is usually fatal.[11]

Bacteremia occurs when inflamed or infected oral tissues are abraded, and pathogenic bacteria enter the bloodstream. The bacteria are also associated with oral infections.[12] Particularly in the presence of oral inflammation, oral hygiene measures such as toothbrushing and interdental cleaning can lead to bacterial endocarditis if the mechanical action causes a transient bacteremia.[13] Periodontopathic bacteria can also gain entrance to the circulation through poor oral hygiene[14] and periodontal disease.[15] Other pathogenic bacteria gain access to the bloodstream through catheterization,[16] sexually transmitted diseases,[17] and contaminated needles in drug abuse.[18] Any abrasion or surgical procedure can cause the disease in a susceptible patient.[19] Bacteria travel through the bloodstream and attach to tissues (damaged or healthy) in the endocardium, quickly forming bacterial colonies that grow into larger vegetations.[20] These large bacterial vegetations then release toxins and enzymes that impact heart tissues, causing cell death and preventing healing. Risk factors for bacterial endocarditis include artificial heart valves, intravenous drug abuse, damaged heart valves, and several congenital heart issues.[21] Treatment usually consists of IV antibiotics and supportive care.[22]

Acute and subacute endocarditis

Acute endocarditis is the more deadly and aggressive form of the disease that develops suddenly and can become life-threatening within a few days.[23] Acute endocarditis presents rapidly with a high fever, tachycardia, fatigue, and swift and extensive heart valve damage.[24] Acute endocarditis is often caused by staphylococcal bacteria that enter the bloodstream, fail to be quickly cleared by the innate immune system, and attach to damaged areas in the heart, such as aberrant valves.[25] Acute endocarditis species of note are *Staphylococcus aureus*, coagulase-negative staphylococci, and enterococcal species.[26]

Subacute endocarditis is a more slowly developing type of IE. It develops gradually over several weeks to months.[27] Subacute endocarditis is usually associated with abnormal heart valves, including those that demonstrate insufficiency.[28] The bacteria involved generally are streptococcal species and frequently colonize the oropharyngeal cavity. These bacteria include *Streptococcus mutans*, *Streptococcus sanguis*,[29] and *Streptococcus mitis*.[30] The symptoms of subacute bacterial endocarditis typically develop slowly and may differ for each person.[27] Without treatment, subacute endocarditis is a fatal disease.[31]

Acute and subacute endocarditis often occur in individuals with common risk factors, including heart valve disease, congenital heart anomalies, hypertrophic cardiomyopathy, prosthetic heart valves, and intravenous drug misuse.

VALVULAR HEART DISEASE[32]

Valvular heart disease, which involves damage to one or more of the heart's valves, can increase the risk of endocarditis. Conditions such as valvular stenosis (narrowing of the valve) and valvular regurgitation (valve leakage) can disrupt the blood flow through the heart, making it

easier for bacteria to adhere to the valves and cause infection. As heart valves do not have a direct blood supply, it is difficult for the body's immune system to access and fight off the infection.

CONGENITAL HEART DEFECTS[33]

Congenital heart defects include malformed valves or fenestrations in the cardiac septum, which can allow blood to leak from one side of the heart to the other. Such defects can result in turbulent and abnormal blood flow, which can create an environment conducive for bacteria to attach and multiply, leading to endocarditis.

HYPERTROPHIC CARDIOMYOPATHY[34]

Hypertrophic cardiomyopathy (HCM), a thickening of the heart muscle, can reduce the amount of blood the ventricle can hold, potentially increasing the risk of endocarditis.

PROSTHETIC VALVES[35]

Individuals with prosthetic heart valves are at a tenfold greater risk of infection compared to those with native heart valves, making them more susceptible to endocarditis.

IV DRUG ABUSE[36]

Using unsterile needles can introduce bacteria into the bloodstream, which can then travel to the heart and cause infection.

Common clinical presentations of endocarditis

Fever is most often caused by the innate immune system reacting to the bacterial antigens from organisms that are not adequately cleared from the circulation.[37] Symptoms of endocarditis vary based on a person's health and age and may develop slowly or rapidly. Common indications of endocarditis include flulike symptoms, such as fever and chills. A fever's etiology should be immediately assessed in patients with suspected endocarditis with relevant cardiac or noncardiac risk factors.[38]

Fatigue is often associated with IE as well as fever, tachycardia, weight loss, sweating, a low red blood cell count (anemia), and headaches.[39]

Anemia (deficiency of healthy red blood cells or hemoglobin) is frequently observed in patients with IE. Medical professionals have generally not adequately emphasized anemia in the bacterial endocarditis diagnostic phase.[40]

An increase in pro-inflammatory markers in the blood is seen in endocarditis. Leukocytosis (elevated WBC count) or leukopenia (decreased WBC count), raised C-reactive protein, erythrocyte sedimentation rate, and procalcitonin, a marker of possible sepsis, are often seen.[41] Hemolysis may lead to anemia, and severe end-organ dysfunction can result in elevated lactate levels, thrombocytopenia, and derangements in renal and liver function tests.[42]

Heart murmurs are often detected during cardiac auscultation in patients with IE.[43] Heart murmurs are associated with valvular incompetence due to vegetations on the heart valves.

MITRAL VALVE MURMUR[44]

An abnormal sound heard during cardiac auscultation due to blood leaking in reverse through the mitral valve. It occurs when the valve between the left heart chambers doesn't close fully.

AORTIC VALVE MURMUR[45]

Occurs when the valve between the left ventricle and the aorta doesn't open fully. This murmur is also known as an aortic regurgitation murmur and occurs when blood leaks in reverse from the aorta into the left ventricle.

TRICUSPID VALVE MURMUR[46]

Occurs when the valve between the right heart chambers (ventricle and atrium) fails to close properly, resulting in blood leaking in reverse into the upper right chamber (right atrium).

A further significant complication of IE is **embolic phenomena**. Typical sequelae of right-sided cardiac lesions are septic pulmonary emboli. Pulmonary infarction, pneumonia, or empyema (pus within the pleural cavity) may occur as a result.[47] Typical sequelae of left-sided cardiac lesions are the embolization (blocked blood vessel) of somatic tissues. Such embolization is mainly seen in the kidneys, spleen, and central nervous system. These small clots can cause life-threatening complications, inhibiting the blood supply to vital organs.[48]

Endocardial vegetations arise when an adherent platelet-fibrin nidus becomes secondarily infected and produces vegetations, which in turn may directly damage the endocardial tissue and/or valves.[49] The microbes then form a biofilm, protecting themselves from immune cells or antibiotic drugs for clearance and eradication. The biofilm-protected vegetations can release toxins and enzymes that destroy healthy cardiac cells and valves, while simultaneously preventing healing.[50]

Petechiae are the most common cutaneous manifestation of IE, and they occur on the extremities and mucous membranes, such as the palate and conjunctivae.[51]

Finally, patients can suffer from **encephalopathy** in IE. Encephalopathy is a neurological manifestation of IE that occurs due to septic embolization from endocardial vegetation, leading to occlusion of cerebral arteries. This can result in ischemic or hemorrhagic stroke or a transient ischemic attack. The condition is often associated with ischemia and/or

suppuration, resulting in infarction, hemorrhage, meningoencephalitis, or abscess. The clinical findings vary depending on the portion of the nervous system affected.[52]

Neurologic complications of infective endocarditis

Transient ischemic attack (TIA) is a brief disruption in the blood circulation to the brain. As a major complication of IE, it can be caused by emboli that break off from the heart valve vegetations and travel to the brain.[53] The symptoms of a TIA are similar to those of an ischemic stroke but last for a shorter period of time and do not cause permanent deficit.[54] Current recommendations indicate that surgery or fibrinolytic therapy without any delay is indicated in patients after silent embolism or TIA.[55,56]

Stroke can affect up to 70% of patients with IE. Stroke in IE can be ischemic or hemorrhagic. Ischemic stroke is a likely neurological complication of IE and is frequently associated with high morbidity.[57] Hemorrhagic stroke occurs primarily in intravenous drug abusers and is associated with uncontrolled *S. aureus* infection with pyogenic arteritis.[58]

Sepsis occurs when the body's response to an infection is so great that it injures its own tissues and organs. In the context of IE, sepsis can occur when the bacteria causing the endocarditis enter the bloodstream in large numbers.[59] This widespread inflammation leads to disseminated intravascular coagulation (DIC—widespread blood clotting in the body), which can cause organ dysfunction and failure.[60] In severe cases, sepsis can progress to septic shock, which is a medical emergency that happens when the body has an extreme response to the infection.[61]

Peripheral manifestations of endocarditis

Splinter hemorrhages are small, dark, straight lines that often appear at the tips of fingernails. For diagnostic value, their indication has poor sensitivity but high specificity. If present, they should not be ignored.[62]

Osler nodes are red-purple, slightly raised, tender lumps, often with a pale center.[63] Pain will often be felt 24 hours before the development of a visible lesion. Osler nodes are typically found on the fingers and/or toes and can manifest at any time during the course of endocarditis development but are usually seen in subacute endocarditis. They last from hours to several days.[64] Osler nodes

TABLE 1: Staphylococcal species in the oral microbiome and oral infections

2013 – Colombo et al.[76]	*S. aureus* was significantly higher in patients with periodontal disease (among the total bacteria associated with oral epithelial cells) than in healthy subjects.
2014 – Mahalle et al.[77]	*S. aureus* was isolated in 13 instances (43.3%). The susceptibility and resistance of bacteria to 12 antibiotic panels most recommended for gram-positive organisms showed maximum resistance to amoxicillin (37%).
2016 – Shah et al.[78]	*S. aureus* was found in head and neck space infection of odontogenic origin (16%) with amoxicillin resistance of 31.3%.
2017 – Fritoli et al.[79]	A higher percentage of volunteers with periodontitis were colonized with *S. aureus* (86%) than periodontally healthy individuals (60%).
2017 – Cahill et al.[80]	"Despite optimal care, mortality approaches 30% at one year. The challenges posed by infective endocarditis are significant. It is heterogeneous in etiology, clinical manifestations, and course. "*Staphylococcus aureus*, which has become the predominant causative organism in the developed world, leads to an aggressive form of the disease, often in vulnerable or elderly patient populations. "There is a lack of research infrastructure and funding, with few randomized controlled trials to guide practice. Longstanding controversies such as the timing of surgery or the role of antibiotic prophylaxis have not been resolved."
2019 – Garbacz et al.[81]	Phagocytosis of staphylococcal biofilm cells from denture wearers is less effective, despite a stronger monocyte response than in nonwearers. This implies that despite triggering a stronger monocyte response, biofilm staphylococci from denture wearers are phagocytized less effectively than those from the nonwearers.
2020 – Al-Akwa et al.[82]	*S. aureus* was the predominant pathogen (43.1%). The prevalence of MRSA was 23.5%.
2021 – Chervinets et al.[83]	The microbiota of the oral cavity of patients with periodontitis was characterized by a decrease in the frequency of bacteria of the genera: streptococcus, peptostreptococcus, peptococcus, and an increase in *S. aureus*, *Veillonella spp.*, *Bacillus spp.*
2021 – Garbacz et al.[84]	110 oral *S. aureus* isolates were phage-typed, and their antibiotic resistance was determined using standard and molecular methods. The prevalence of MSSA and MRSA strains was 89.1% and 10.9%, respectively.
2021 – Uribe-García et al.[85]	"*S. aureus* was identified in 18.6% (50/268) of the samples. "All strains were resistant to methicillin, ampicillin, dicloxacillin, cefotaxime, and penicillin and were multidrug-resistant to 6-12 antibiotics. "Although *S. aureus* has been considered a transient member of the oral microbiota, our results indicate a high-level expression of virulence genes and multidrug resistance in the strains isolated from periodontal lesions."
2023 – Colombo et al.[86]	"The overall prevalence of subgingival staphylococci was 46%, especially in severe periodontitis (> 60%; $p < 0.01$). "Penicillin-resistant staphylococci is highly prevalent in the subgingival biofilm regardless of the periodontal status. Strains carrying virulence genes related to tissue adhesion/invasion, inflammation, and cytotoxicity support the pathogenic potential of these opportunists in the periodontal microenvironment."

result from a deposition of immune complexes, following the inflammatory response that leads to swelling, redness, and pain that characterize these lesions.[65]

Janeway lesions are red or purple painless spots that form on the palms, soles, or under the toes or fingers[66] and are also a common sign of IE. Janeway lesions differ from Osler nodes in that they are nontender and are thought to be due to embolic phenomenon in cutaneous palmar and plantar blood vessels.

Endocarditis diagnosis

The diagnostic work-up of IE begins with laboratory blood tests. A complete blood count (CBC) will determine if leukocytosis or anemia is present; both are signs of infection.[67] Other lab tests include testing the inflammatory condition with C-reactive protein and erythrocyte sedimentation rate assays. Also, liver and kidney function are generally tested for diagnostic criteria for endocarditis.[68]

Blood cultures are a crucial part of the diagnostic work-up for IE, as they will identify the causative organism of the infection. Two to three blood cultures should be obtained from separate venipuncture sites to confirm proper antibiotic therapy. Common blood culture isolates include *Staphylococcus aureus*, *Streptococcus viridans*, enterococci, and coagulase-negative staphylococci.[69]

Echocardiography plays a vital function in the diagnosis and management of IE. It can help determine the underlying anatomy of the valvular structures, as well as the presence, location, size, and number of vegetation-like masses.[70] Two types of echocardiograms are commonly used: transthoracic echocardiogram (TTE) and transesophageal echocardiogram (TEE). TTE is a noninvasive test that uses sound waves to create images of the heart. TEE provides a closer look at the heart's valves and chambers, making it a more sensitive and specific test in diagnosing IE.[71]

Current AHA guidelines and endocarditis prevalence

The current recommendations from the American Dental Association and the AHA for endocarditis prophylaxis are based on the belief that the causative organisms from the mouth are streptococcal species.[8] This has been the standard belief since the 2007 prophylaxis recommendations. In 2007, the AHA issued a warning, advising dentists to cease prescribing prophylactic antibiotics to a broad group of previously treated patients due to growing concerns about antibiotics and the risk of antibiotic-resistant organisms.[1,72]

However, not long after the new recommendations, researchers in the United Kingdom studied the impact of cutting back on prophylactic antibiotic prescriptions and found that as the number of prescriptions went down, the number of bacterial endocarditis cases increased by 35 more per month.[73] This data strongly suggested a correlation between reduced AP recommendations and an uptick in endocarditis cases.

A changing microbiome in the mouth: cause for concern

For many years, decisions have been made about AP for dental therapy based on the belief that dental professionals were protecting patients against streptococcal species such as *Streptococcus sanguinis*.[74] As discussed above, streptococcal species generally cause a more slowly developing chronic endocarditis[27] than staphylococcal species, which cause more acute and aggressive endocarditis.[23] In recent years, it has become apparent that both streptococci and staphylococci are present in oral microbiota, and these findings indicate that prophylactic protocols to prevent bacterial endocarditis related to dental care should also be reexamined.

Staphylococcus aureus found in oral infections increased in the decade from 1990 to 2000, with reports ranging from 1% to 15%.[4-7] By 2010, researchers were also culturing significant amounts of *Staphylococcus epidermidis* from refractory endodontic lesions.[75] In the last 15 years, the discovery of staphylococcal species in the oral microbiome and oral infections has significantly increased. See Table 1.

Many researchers have recently discussed the need for alterations to AP recommendations to address these emerging findings from a variety of perspectives.

In 2017, Pippi deliberated on the issue of increasing comorbidities in dental patients, stating, "Another issue in IE prevention is to establish whether and when the existence of any comorbidity conditions which may imply a reduction of host defenses, such as older age, diabetes mellitus, immunosuppressive conditions or therapies, and dialysis, indicates AP in cardiac conditions for which, although at risk for endocarditis, prophylaxis to date is not indicated."[87] Pippi further stated, "These conditions may complicate IE. Each of them independently increases the risk of an adverse outcome from IE, and they often occur in combination, which further increases morbidity and mortality rates ... Therefore, in absence of any specific indications by international guidelines, it seems reasonable to perform AP in all patients with compromised immune defenses having risk conditions for IE."[87]

In 2019, Garg et al. showed that *S. aureus* IE hospitalizations increased substantially after the new 2007 guidelines for adults 18 to 64 years old.[88]

In 2022, Dreyfuss et al. stated, "Should *S. pyogenes* or *S. aureus* infect beyond skin incision and get seeded in the blood, they have a strong chance of contributing to an infective endocarditis."[89] Also, in 2022, Østergaard et al. stated, "*S. aureus* was the most frequent microbiological cause of IE, followed by streptococcus species and enterococcus species. Patients with *S. aureus* IE had the highest in-hospital mortality."[90]

In 2023, Lean et al. conducted a systematic review and meta-analysis of prophylactic antibiotic use for IE. They concluded that "None of the high-risk patients developed IE across all studies where 100% of the patients were treated with prophylactic antibiotics, and IE patients are 12% more likely to have undergone recent dental manipulation compared with matched controls."[91]

While some clinicians and researchers have argued that AP can be dangerous, with increased morbidity and mortality, it has been definitively shown that this is not the case with amoxicillin and doxycycline.[92,93] Cloitre et al. stated, "Fatal or severe adverse drug reactions with amoxicillin or clindamycin is not a rational argument to stop IE AP before invasive dental procedures."[94] Further, Hussein et al. stated, "Three out of four clinical practice guidelines support that the benefits of prevention of infective endocarditis outweigh the risks of antibiotic resistance."[95] Some reports have also identified dental misuse of antibiotics as a large driver of antibiotic resistance, but these claims have been demonstrated to be overblown. Much of the peer-reviewed evidence in the last decade is contrary to this opinion, showing that the vast majority of antimicrobial use that causes resistance is in animal feed and other nontherapeutic uses, with smaller contributions from poor adherence to antibiotic prescriptions and poor antibiotic choice.[96-100]

Finally, in outright criticism of the current AP guidelines, van den Brink et al. recently stated, "We should, therefore be brave and admit that we might have been wrong. We should reinstate chemoprophylaxis for all patients with valvular anomalies to prevent IE from affecting these patients at risk. We should make all possible effort to prevent this disease from happening because we have great difficulty in treating it, and the toll that patients have to pay is high."[101]

Conclusion

The prevalence of *Staphylococcus aureus* found in oral microbiota has increased in the last 25 years. This finding combined with a growing older demographic of dental patients, who have more significant cardiovascular, immunological, and metabolic comorbidities and burdens, may highlight the need for revision of guidelines for AP. Given that the peer-reviewed evidence also shows (1) a more aggressive and deadly acute bacterial endocarditis due to highly pathogenic *Staphylococcus aureus* is on the rise, (2) demonstrated high levels of safety associated with AP when amoxicillin and doxycycline are used, and (3) a limited, if any, role of dental prescribing patterns in antimicrobial resistance, it is time that we reassess the current AP guidelines. Dental practitioners should remember that AP for dental patients remains the choice of the dentist and patient, based on the dentist's best clinical judgment, given the patient's current comorbidity and polypharmacy issues at the time of the procedure. If a dentist believes that a patient should receive prophylaxis beyond the current guidelines, that is what the dentist should do without fear of ramifications from any recommending group or association.

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1. Infective endocarditis is:
   - A. An infection of the endocardium of the heart
   - B. An infection that affects the pericardium
   - C. An infection of the cardiac blood vessels
   - D. A dental infection

2. The most commonly affected cardiac structures in infective endocarditis are:
   - A. Valves
   - B. Chordae
   - C. Chamber walls
   - D. Periprosthetic tissues

3. High-risk groups for infective endocarditis include:
   - A. Individuals with BMI of 20-30
   - B. 20-year-old athletes
   - C. IV drug abusers and congenital heart disease
   - D. Women

4. Which of the following groups is at the highest risk for developing bacterial endocarditis?
   - A. Children under the age of 10
   - B. Adults aged 30-40
   - C. Adults over the age of 60
   - D. Adolescents aged 15-20

5. Which of the following conditions increases the risk of bacterial endocarditis?
   - A. Pulmonary insufficiency
   - B. Artificial (prosthetic) heart valves
   - C. Nonalcoholic fatty liver disease
   - D. Damaged heart valves

6. The symptoms of endocarditis include:
   - A. Fever, chills, fast heart rate
   - B. Fatigue, night sweats, aching joints and muscles
   - C. Persistent cough or swelling in the feet, legs, or abdomen
   - D. All of the above

7. Which of the following symptoms is commonly associated with bacterial endocarditis?
   - A. Sudden weight gain
   - B. Persistent cough
   - C. Joint pain
   - D. Frequent headaches

8. Which of the following is not a common symptom of bacterial endocarditis?
   - A. Fever and chills
   - B. Shortness of breath
   - C. Blood in the urine
   - D. Painless red spots on the soles of the feet

9. Antibiotics are molecules that inhibit the growth of or kill:
   - A. Fungi
   - B. Viruses
   - C. Bacteria
   - D. Insects

10. Which of the following statements best differentiates Osler nodes from Janeway lesions in the context of bacterial endocarditis?
   - A. Osler nodes are painful, tender, and located on the fingertips and toes; Janeway lesions are painless, flat, and typically found on the palms and soles.
   - B. Osler nodes are painless and flat; Janeway lesions are painful, tender, and commonly located on the fingertips.
   - C. Osler nodes are associated with high fever and are usually located on the palms; Janeway lesions are found on the mucous membranes.
   - D. Osler nodes result from immune complex deposition, leading to necrosis; Janeway lesions are associated with direct bacterial embolization causing inflammation.

11. According to the American College of Cardiology, the bacteria most commonly associated with bacterial endocarditis is:
   - A. *Staphylococcus aureus*
   - B. *Streptococcaceae*
   - C. *Enterococci*
   - D. *Albicans*

12. Which of the following statements accurately reflects the prevalence of different organisms in infective endocarditis?
   - A. Fungal organisms are the most common cause of infective endocarditis, followed by enterococcal species.
   - B. Staphylococcal, streptococcal, and enterococcal species are more prevalent in infective endocarditis than fungal organisms.
   - C. Streptococcal species are the least prevalent cause of infective endocarditis compared to staphylococcal and fungal organisms.
   - D. Enterococcal species are less prevalent in infective endocarditis compared to fungal organisms.

13. Which bacteria are most commonly associated with subacute bacterial endocarditis?
   - A. *Streptococcus pneumoniae*
   - B. Streptococcus pyogenes
   - C. Streptococcus viridans
   - D. Streptococcus agalactiae

14. Dental professionals have been given guidelines on antibiotic prophylaxis for bacterial endocarditis by:
   - A. CDC
   - B. FDA
   - C. ADA and AHA
   - D. OSHA

15. Antibiotic resistance is:
   - A. Resistance on the part of the patient to take antibiotics
   - B. Resistance on the part of the dentist to prescribe antibiotics
   - C. Resistance on the part of the pharmacist to dispense antibiotics
   - D. The ability of the bacteria to resist the antibiotics

16. The most common reason that antibiotic resistance develops is due to inappropriate antibiotic use in:
   - A. Dental patients
   - B. Farmers and livestock
   - C. Cardiac patients
   - D. Orthopedic patients

17. Antibiotic prophylaxis for dental patients remains:
   - A. The choice of the dentist and patient
   - B. The mandate of the ADA
   - C. The mandate of the FDA
   - D. The mandate of the CDC

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18. What is the primary characteristic of hypertrophic cardiomyopathy?
- A. Enlarged heart chambers
- B. Thinning of the heart muscle
- C. Thickening of the heart muscle
- D. Irregular heartbeats

19. Why does endocarditis pose a threat to the heart valves?
- A. Bacteria can grow on the valves, leading to embolization.
- B. Bacteria cause thickening of the valve leaflets.
- C. Bacteria weaken the heart muscle.
- D. Bacteria cause calcification of the valve annulus.

20. For many years, decisions have been made about antibiotic prophylaxis for dental therapy based on the belief that dental professionals were protecting patients against:
- A. Vibrio species
- B. Staphylococcal species
- C. Streptococcal species
- D. Candidal species

21. Which imaging technique is used to detect damaged heart valves in IE?
- A. Echocardiogram
- B. Computerized tomography scan
- C. Magnetic resonance imaging
- D. Ultrasound

22. What does a blood culture test help identify in IE?
- A. White blood cell count
- B. Microorganisms in the bloodstream
- C. Valve calcification
- D. Heart murmur

23. Which patients are currently recommended for antibiotic prophylaxis before dental or surgical procedures to prevent IE?
- A. Patients with artificial heart valves
- B. Patients with hypertension
- C. Patients with a history of rheumatic heart disease
- D. Patients with mitral regurgitation

24. What is the primary cause of the changing epidemiology of IE in the elderly population?
- A. Increased incidence of rheumatic fever
- B. Rise in diabetes mellitus, immunosuppressive conditions or therapies, and dialysis
- C. Decreased use of valvular prostheses
- D. Higher prevalence of *Staphylococcus aureus*

25. Aggressive biofilm producing species of *S. aureus* are now found more frequently:
- A. In odontogenic infections
- B. In periodontal pockets
- C. On removable prostheses
- D. All of the above

26. What are painless hemorrhages seen on the palms and soles in patients with IE called?
- A. Petechiae
- B. Splinter hemorrhages
- C. Osler nodes
- D. Janeway lesions

27. What term describes hemorrhages in the nail beds (usually seen as dark streaks under the nails) associated with IE?
- A. Petechiae
- B. Splinter hemorrhages
- C. Osler nodes
- D. Janeway lesions

28. What is the most common neurologic complication associated with IE?
- A. Meningitis
- B. Transient ischemic attack or intracranial hemorrhage
- C. Parkinson's disease symptoms
- D. Mycotic aneurysm

29. Why should the AHA revisit its current recommendations for subacute bacterial endocarditis prophylaxis?
- A. The oral microbiome has been stable for the last 25 years.
- B. The oral microbiome has become less pathogenic in the last 25 years.
- C. The oral microbiome now has more pathogenic staphylococcal species.
- D. There are now more fungi in the mouth than in previous decades.

30. Why are evidence-based guidelines important in managing endocarditis?
- A. They ensure compliance with insurance policies.
- B. They help reduce health-care costs.
- C. They improve patient outcomes.
- D. They promote pharmaceutical companies' products.

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Preventing acute and chronic endocarditis in dentistry

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TELEPHONE (PRIMARY): TELEPHONE (OFFICE):

REQUIREMENTS FOR OBTAINING CE CREDITS BY MAIL/FAX: 1) Read entire course. 2) Complete info above. 3) Complete test by marking one answer per question. 4) Complete course evaluation. 5) Complete credit card info or write check payable to Endeavor Business Media. 6) Mail/fax this page to DACE.

If you have any questions, please contact dace@endeavorb2b.com or call (800) 633-1681. A score of 70% or higher is required for CE credit.

COURSE CAN ALSO BE COMPLETED ONLINE AT A LOWER COST. Scan the QR code or go to dentalacademyofce.com to take advantage of the lower rate.

EDUCATIONAL OBJECTIVES

1. Describe the patient presentation of subacute bacterial endocarditis and outline the risk factors for subacute bacterial endocarditis associated with the provision of dental care
2. Describe the prophylactic antibiotics recommended to minimize the risk of subacute bacterial endocarditis during dental care delivery
3. Review the interprofessional team members' roles in preventing subacute bacterial endocarditis and prosthetic joint infection

COURSE EVALUATION

1. Were the individual course objectives met?
   Objective #1: Yes No Objective #2: Yes No Objective #3: Yes No

Please evaluate this course by responding to the following statements, using a scale of Excellent = 5 to Poor = 0.

2. To what extent were the course objectives accomplished overall?	5	4	3	2	1	0
3. Please rate your personal mastery of the course objectives.	5	4	3	2	1	0
4. How would you rate the objectives and educational methods?	5	4	3	2	1	0
5. How do you rate the author's grasp of the topic?	5	4	3	2	1	0
6. Please rate the author's effectiveness.	5	4	3	2	1	0
7. Was the overall administration of the course effective?	5	4	3	2	1	0
8. Please rate the usefulness and clinical applicability of this course.	5	4	3	2	1	0
9. Please rate the usefulness of the references.	5	4	3	2	1	0
10. Do you feel that the references were adequate?	Yes	No				
11. Would you take a similar course on a different topic?	Yes	No				

12. If any of the continuing education questions were unclear or ambiguous, please list them.

13. Was there any subject matter you found confusing? Please describe.

14. How long did it take you to complete this course?

15. What additional dental continuing education topics would you like to see?

Mail/fax completed answer sheet to:
Endeavor Business Media
Attn: Dental Division; 7666 E. 61st St. Suite 230, Tulsa, OK 74133
Fax: (918) 831-9804

☐ Payment of $69 is enclosed (this course can be completed online for $39. Scan the QR code or go to dentalacademyofce.com to take advantage of the lower rate).

Make check payable to Endeavor Business Media

If paying by credit card, please complete the following:

☐ MC ☐ Visa ☐ AmEx ☐ Discover

Acct. number:_____

Exp. date: _____CVC #: _____

Billing address:_____

_____

Charges on your statement will show up as Endeavor.

1.	Ⓐ	Ⓑ	Ⓒ	Ⓓ	16.	Ⓐ	Ⓑ	Ⓒ	Ⓓ
2.	Ⓐ	Ⓑ	Ⓒ	Ⓓ	17.	Ⓐ	Ⓑ	Ⓒ	Ⓓ
3.	Ⓐ	Ⓑ	Ⓒ	Ⓓ	18.	Ⓐ	Ⓑ	Ⓒ	Ⓓ
4.	Ⓐ	Ⓑ	Ⓒ	Ⓓ	19.	Ⓐ	Ⓑ	Ⓒ	Ⓓ
5.	Ⓐ	Ⓑ	Ⓒ	Ⓓ	20.	Ⓐ	Ⓑ	Ⓒ	Ⓓ
6.	Ⓐ	Ⓑ	Ⓒ	Ⓓ	21.	Ⓐ	Ⓑ	Ⓒ	Ⓓ
7.	Ⓐ	Ⓑ	Ⓒ	Ⓓ	22.	Ⓐ	Ⓑ	Ⓒ	Ⓓ
8.	Ⓐ	Ⓑ	Ⓒ	Ⓓ	23.	Ⓐ	Ⓑ	Ⓒ	Ⓓ
9.	Ⓐ	Ⓑ	Ⓒ	Ⓓ	24.	Ⓐ	Ⓑ	Ⓒ	Ⓓ
10.	Ⓐ	Ⓑ	Ⓒ	Ⓓ	25.	Ⓐ	Ⓑ	Ⓒ	Ⓓ
11.	Ⓐ	Ⓑ	Ⓒ	Ⓓ	26.	Ⓐ	Ⓑ	Ⓒ	Ⓓ
12.	Ⓐ	Ⓑ	Ⓒ	Ⓓ	27.	Ⓐ	Ⓑ	Ⓒ	Ⓓ
13.	Ⓐ	Ⓑ	Ⓒ	Ⓓ	28.	Ⓐ	Ⓑ	Ⓒ	Ⓓ
14.	Ⓐ	Ⓑ	Ⓒ	Ⓓ	29.	Ⓐ	Ⓑ	Ⓒ	Ⓓ
15.	Ⓐ	Ⓑ	Ⓒ	Ⓓ	30.	Ⓐ	Ⓑ	Ⓒ	Ⓓ

CUSTOMER SERVICE: (800) 633-1681

EXAM INSTRUCTIONS. All questions have only one answer. If mailed or faxed, grading of this examination is done manually. Participants will receive confirmation of passing by receipt of a Verification of Participation form. The form will be mailed within two weeks after receipt of an examination.

COURSE EVALUATION AND FEEDBACK. We encourage participant feedback. Complete the evaluation above and e-mail additional feedback to Rachel McIntyre (rmcintyre@endeavorb2b.com) and Laura Winfield-Roy (lwinfield@endeavorb2b.com).

COURSE CREDITS AND COST. All participants scoring 70% or higher on the examination will receive a verification form for three (3) continuing education (CE) credits. Participants are urged to contact their state dental boards for CE requirements. The cost for courses ranges from $20 to $110.

CANCELLATION AND REFUND POLICY. Participants who are not 100% satisfied can request a refund by contacting Endeavor Business Media in writing.

RECORD KEEPING Endeavor Business Media maintains records of your successful completion of any exam for a minimum of six years. Please contact our offices for a copy of your CE credits report. This report, which will list all credits earned to date, will be generated and mailed to you within five business days of receipt.

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