Infective endocarditis (IE) is a microbial infection of the endothelial lining of the heart. The disease is well-known for its ability to destroy the structural integrity of the heart and its valves. It is also a complex systemic illness affecting many body organs and physiologic processes. Once known as bacterial endocarditis, the change in nomenclature recognizes that organisms other than bacteria (e.g., fungi, rickettsia, chlamydia) are also to blame (Millar & Moore, 2004).

Enigma, conundrum, dilemma, puzzling, baffling, and perplexing are all words used in the literature and clinical practice to describe this centuries-old disease. Jean François Fernal gave a first account of IE in 1554. Nearly a century later, Lazare Riviere (1646) described the autopsy findings that were consistent with this disease. In 1885, Sir William Osler described fever, heart murmur, and hemiplegia as a triad of symptoms associated with IE (Millar & Moore, 2004).

Since then, two major advances in the management of IE have occurred. The first was antibiotics in the 1940s. This dramatically reduced the mortality rate from overwhelming infection. Controlling the infection, however, unmasked congestive heart failure (CHF) that is still a prominent and fatal feature of IE (Hasbun, Vikram, Barakat, Buenconsejo, & Quagliarello, 2003). The second advance was cardiac surgery and the realization that a prosthetic valve could be implanted into a patient with active IE. Since that time, proceeding with surgery remains a difficult and, at times, controversial decision (Baddour, Wilson, Bayer, Fowler, Bolger, Levison, et al., 2005; Bayer, Bolger, Taubert, Wilson, Steckelberg, Karchmer, et al., 1998; Delahaye, Célard, Roth, & De Gevigney, 2004).

Infective endocarditis, despite the use of antibiotics and cardiac surgery, remains a life-threatening disease. This harsh reality is attributed to congestive heart failure that comes from incompetence of a normal, defective, or prosthetic valve, or from destruction of the heart itself. Unfortunately, diagnosis and treatment may be delayed. This is due, in part, to the vague and, at times, baffling clinical picture of this disease and to the frequency with which antibiotics are administered prior to obtaining a microbiological diagnosis. Nurses play a pivotal role in the prevention, early recognition, diagnostic work-up, and prompt, effective treatment of this devastating disease and its complications. A review and update of this striking, yet elusive disease is provided in this article. By transferring current knowledge into practice, nurses will be better able to plan, implement, and evaluate the care needed by this unique and challenging patient population.

**Key words:** infective endocarditis, bacteremia, embolization, vegetation, antimicrobial therapy, antibiotic prophylaxis, nosocomial infection, blood cultures, echocardiogram, altered bio-psycho-social-spiritual status

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**Pathogenesis**

The pathogenesis of this disease is based on speculation and animal studies. IE is believed to begin with a transient bacteremia. Short-lived bacteremic
episodes are a frequent, unnoticed everyday occurrence. IE is not a consequence of every episode, as the immune system rapidly deals with the invasion. For this sinister disease to take hold, there needs to be an interaction between a receptive heart and the invading pathogen (Wilson et al., 2007).

The infecting microorganism from the transient bacteremia needs to adhere to the endothelial lining of the heart. Normal endothelium is non-adherent and most organisms, save Streptococcus, are unable to self-adhere. Blood will naturally clot on traumatized endothelium or an aberrant heart valve. This provides a medium for the circulating pathogen to attach and thus organize into a vegetation (Beynon, Bahl, & Prendergast, 2006; Ferrieri, Gewitz, Gerber, Newburger, Dajani, Shulman, et al., 2002; Mylonakis & Calderwood, 2001; Wilson, Taubert, Gewitz, Lockhart, Baddour, Levison, et al., 2007).

Vegetations are a meshwork of fibrin and platelets with deeply embedded microorganisms that form into irregular, friable masses of varying sizes. They form just beyond the narrowing of a stenotic valve and on the low-pressure side of a regurgitant valve. Platelets and fibrin cover the infecting microorganism making it extremely difficult for host defences and antimicrobial agents to penetrate the vegetation and eradicate the infection. Immature vegetations are metabolically very active and each gram may contain as many as $10^8$ to $10^{11}$ colony-forming units of bacteria. Mature vegetations are metabolically inactive and contain dormant bacteria (Ferrieri et al., 2002; Mylonakis & Calderwood, 2001; Wilson et al., 2007).

### Causative organisms

Many infecting microorganisms are indigenous to the body and Streptococcus viridans and Staphylococcus aureus have historically topped the list as the pathogens most likely to cause IE. Recently, Staphylococcus aureus has taken the lead position. This shift is concerning as Staphylococcus aureus IE tends to be caused by nosocomial or iatrogenic inoculation and is associated with serious complications and a high mortality rate (Baddour et al., 2005; Chu, Cabell, Benjamin, Kuniholm, Fowler, Engemann, et al., 2004; Martin-Dávila, Fortún, Navas, Javier, Jiménez-Mena, Moya, et al., 2005; Wang, Athan, Pappas, Fowler, Olaison, Paré, et al., 2007).

Streptococcus viridans normally inhabits the oral cavity, respiratory tract, skin, and gastrointestinal (GI) tract. Although several streptococcus species cause IE, Streptococcus bovis has been associated with colon cancer (Baddour et al., 2005; Mylonakis & Calderwood, 2001; Pound & Townsend, 2007; Wilson et al., 2007). Staphylococcus is either coagulase positive (Staphylococcus aureus), which live on skin and in nasal passages; or coagulase negative (Staphylococcus epidermidis), which also live on skin and mucus membranes. Where once Staphylococcus aureus tended to cause native valve endocarditis (NVE) and Staphylococcus epidermidis prosthetic valve endocarditis (PVE), there is now considerable overlap (Baddour et al., 2005; Bayer et al., 1998; Wang et al., 2007).

Enterococcus (e.g., Enterococcus faecalis and Enterococcus faecium) live in the genitourinary (GU) and lower GI tracts and are often the cause of IE. Gram negative bacilli account for much fewer cases with Pseudomonas aeruginosa occurring most frequently in patients with indwelling lines and intravenous drug users (IDU) (Baddour et al., 2005; Bayer et al., 1998).

HACEK (Hemophilus parainfluenzae, H aphrophilus, H paraprophilus, Actinobacillus [Haemophilus] actinomycetemcomitans, Cardiobacterium hominis, Eikenella species, and Kingella kingae) are a group of fastidious gram negative bacilli that inhabit the oropharyngeal tract. These microorganisms are difficult to isolate by standard blood culture (BC) processing techniques (Baddour et al., 2005; Bayer et al., 1998).

IE from fungus (e.g., candida, aspergillus), although rare, tends to occur in hospitalized and immunocompromised patients. Patients with this form of IE will frequently relapse even years after treatment (Baddour et al., 2005; Bayer et al., 1998).

Culture-negative IE is on the rise and may be present in as many as 20% of cases. The culprit may well be the administration of antimicrobial agents before BCs have been drawn. Without a microbiological diagnosis, antimicrobial therapy is given to cover the most common causes of IE. Such an empirical approach exposes the patient to toxic therapy that may not kill the causative organism or alter the clinical course or outcome (Baddour et al., 2005).

### Epidemiology

Unfortunately, an inverse relationship between improvements in diagnosis and treatment, and the incidence of IE has not occurred. In fact, the incidence is on the rise and delay in recognizing and treating this disease persists. The rise is attributable to several major health care trends, such as an increased lifespan both pre- and post-surgery for patients with defective valves and congenital heart defects (CHD); improved cardiac surgical techniques and the ability to do complex procedures; and increased use of implantable devices, prosthetic material, and...
prosthetic valves (Di Filippo, Delahaye, Semiond, Célard, Henaine, Ninet, et al., 2006; Ferrieri et al., 2002; Martín-Dávila et al., 2005).

The yearly incidence of IE is approximately 15,000 to 20,000 new cases. This rate, despite the many advances in health care, has not appreciably changed in 50 years. What have changed are the pathogen and patient profiles. Pathogens are increasingly multiple, fastidious, and resistant to antimicrobial therapy. Patients increasingly present with nosocomial (health care-associated) or surgical site infection(s); and comorbid conditions such as chronic HIV/AIDS, renal failure on hemodialysis, diabetes mellitus, and respiratory, GI, or GU tract illnesses. There is also a subset of patients who are involved in high-risk activities such as IDU, excessive alcohol consumption, and body piercing or tattooing (Baddour et al., 2005; Bayer et al., 1998; Beynon et al., 2006; Chu et al., 2004; Martín-Dávila et al., 2005; Millar & Moore, 2004; Mylonakis & Calderwood, 2001; Wang et al., 2007; Wilson et al., 2007).

The male-to-female ratio is 1.7:1. Although IE tends to attack in mid- to later-life (median age 47 to 69 years), it is no respecter of age and is no easier to diagnose or treat in neonatal, pediatric, or geriatric patients (Beynon et al., 2006; Ferrieri et al., 2002; Mylonakis & Calderwood, 2001; Vahanian, 2003).

In developed countries, mitral valve prolapse has surpassed rheumatic heart disease as the heart disorder most likely to cause IE. Many patients (47%) who develop IE have no knowledge of a pre-existing cardiac condition. Rates of mechanical and bioprosthetic PVE are equal at 0.3% to 1% per patient year and account for 1% to 5% of all cases (Beynon et al., 2006; Mylonakis & Calderwood, 2001). The latter may be an underestimation as Wang and colleagues (2007) found PVE accounted for more than 20% of all IE cases in their prospective, multicentre, international study.

IE is the fourth leading cause of infection-related death. The overall mortality rate, despite antibiotic therapy, remains high (20% to 50%) for both PVE and NVE. The mortality rate is less (10%) for patients with right-sided IE. Mortality from this disease is closely linked to CHF, the result of worsening valvular insufficiency, ventricular dysfunction, abscesses, and intra-cardiac fistulas (Baddour et al., 2005; Bayer et al., 1998; Millar & Moore, 2004; Mylonakis & Calderwood, 2001). Efforts targeting prevention, recognition, diagnosis, and treatment are needed to curb the high morbidity and mortality associated with this devastating disease.

**Preventing IE**

Failure to prevent IE is worrisome, yet, the majority of cases are not preventable and it is impossible to predict which patients will develop this disease. Preventing IE is not an exact science. Despite this, the American Heart Association (AHA) has been writing and updating guidelines on the topic since 1955. The latest guideline moves away from exclusive reliance on antibiotic prophylaxis as a prevention strategy (Wilson et al., 2007). Admittedly, in situations where the infecting organism enters through self-inoculating activities such as poor dental hygiene/caries, brushing/flossing teeth, defecating, or chewing a hard candy, prophylaxis would be impractical and unwarranted. Prevention is also difficult when less than 50% of IE patients have a pre-existing valve lesion that would qualify for antibiotic prophylaxis (Wilson et al., 2007).

An over-reliance on antibiotic prophylaxis and an over-estimation of its success may have detracted from a focus on fundamental prevention strategies. For the individual, this would involve maintenance of oral health and an understanding of self-inoculation. For health care organizations, this would involve maintaining high infection control standards, reducing nosocomial infection rates and eliminating health care-associated infections as a cause of iatrogenic IE. These issues could be addressed through participation in “Safer Healthcare Now” initiatives that focus on preventing harm from antibiotic-resistant organisms; and preventing infections from central lines, surgical sites, and ventilator-associated pneumonia (Safer Healthcare Now, 2007).

The presence of multiple resistant organisms (e.g., methicillin-resistant *Staphylococcus aureus* [MRSA], vancomycin-resistant *Enterococcus* [VRE]) is a current health care reality. Widespread use of antibiotic prophylaxis has been shown to give little to no gain and has unintentionally contributed to the creation of multiple resistant organisms. Narrowing the prophylaxis criteria is one strategy to address this problem.

The AHA now recommends that only patients with a prosthetic valve, previous IE, CHD, and cardiac transplant recipients with valvulopathy receive antibiotic prophylaxis for dental procedures that involve manipulation of gingival tissue or the periapical region of the teeth; or perforation of the oral mucosa. Prophylaxis is also recommended in these patients for procedures involving the respiratory tract, infected skin, skin structures, or musculoskeletal tissue. It is no longer recommended, however, for procedures
Recognizing IE

It takes approximately two weeks for symptoms of IE to develop after an inoculating event. However, even when symptoms appear, treatment delay often continues (Wilson et al., 2007). In looking for ways in which to improve diagnosis and avoid delays in treatment, Kjerulf and colleagues (1998) found that on the average it took three to four weeks after the onset of symptoms and a full two weeks after admission to hospital before a diagnosis was established. Such delays allow this disease to continue its destructive course unchecked.

Failure or delay in recognizing IE may be, in part, due to the way in which it presents. The onset may be an acute catastrophic illness or a prolonged systemic non-specific flu-like illness. The latter gives rise to several distinct peripheral immune mediated features such as Osler’s nodes (painful lesions on the finger tips and toe pads); Janeway lesions (nontender macules on the palms of the hand or the soles of the feet); Roth spots (hemorrhagic retinal lesions); splinter hemorrrhages (dark streaks beneath finger and toe nails), petechiae of the conjunctivae, buccal mucosa, or extremities; and clubbing (Baddour et al., 2005; Millar & Moore, 2004).

Septic features relate to the ongoing bacteremia. Although a hallmark of IE is fever, it may be intermittent or absent in patients who are immunocompromised or elderly. Bacteremia also produces a number of symptoms such as rigors, chills, night sweats, anorexia, weight loss, nausea, vomiting, malaise, and arthalgias (Beynon et al., 2006; Habib, 2006; Millar & Moore, 2004; Mylonakis & Calderwood, 2001).

Cardiac features, especially the development of CHF, relate to the extent to which IE has destroyed the heart’s infrastructure or valves. Whether CHF develops abruptly or insidiously, it has the greatest impact on prognosis. As IE makes a valve incompetent, a new or worsening heart murmur will develop (Baddour et al., 2005; Habib, 2006). A diastolic murmur heard along the left sternal border is indicative of aortic regurgitation whereas a systolic murmur heard at the apex and radiating towards the axilla is indicative of mitral regurgitation. Auscultation skills and the identification of murmurs take concentrated listening and practice. Audio tools to help refine this skill can be found at websites such as www.wilkes.med.ucla.edu.

Abscesses usually occur at the weakest part of the annulus. For the aortic valve this is very near the atrioventricular node. Heart block may develop as the pathogen impinges on the conduction system. Destruction of the mitral-aortic trigone and the disruption of the ventricular-aortic continuity spell disaster and mandate urgent surgical correction (Baddour et al., 2005; Habib, 2006).

Embolic features occur in 22% to 50% of IE patients and relate to fragmentation and embolization of the vegetation. The death rate after sustaining an embolus is reported at 24% to 50%. Although most emboli occur within the first few weeks of treatment, they may also occur before treatment has begun or after treatment has finished. Large (i.e., >10 mm in size), highly mobile vegetations are most likely to cause an embolus. Right heart vegetations cause pulmonary emboli or infarct, and pneumonia or empyema. Left heart vegetations embolize throughout the arterial tree causing end organ damage and infarction. The prime target organ is the brain (65% to 70%) and the stroke, which occurs primarily in the territory of the middle cerebral artery (> 90%), carries a high mortality rate (Homma & Grahame-Clarke, 2003).

Although rare, vegetative emboli can cause an acute myocardial infarction. The spleen is also vulnerable to emboli that can lead to infarction, abscess and, although rare, splenic rupture. If a splenectomy is warranted it should be done before valve surgery to eliminate the risk of seeding the new valve prosthesis from the infected spleen (Baddour et al., 2005; Bayer, et al., 1998; Ferrieri et al., 2002; Habib, 2006; Millar & Moore, 2004; Mylonakis & Calderwood, 2001).

Mycotic aneurysms (MA) occur in 15% to 25% of patients with IE; are often multiple; may involve any vessel; are difficult to treat; and, although rare, are extremely dangerous, especially if they rupture. These intracranial or extracranial MAs tend to develop at arterial branching points and are believed to form after the infecting microorganism invades the small arteries that are distributed to the adventitia and medial layers (i.e., vasa vasorum) of the arterial wall (Baddour et al., 2005; Bayer et al., 1998; Mylonakis & Calderwood, 2001).

Diagnosing IE

Hastening the time to diagnosis is pivotal to patient outcome. Diagnosis primarily rests on obtaining BCs to identify the infecting microorganism and an echocardiogram to assess the status of the heart (e.g., vegetation(s), valves, structural damage, abscesses, ventricular function). The results of other laboratory tests, with the exception of BCs, are seldom helpful.
Non-specific findings can include anemia, leukocytosis, elevated erythrocyte sedimentation rate, and elevated C-reactive protein level (Mylonakis & Calderwood, 2001).

It has been known for almost 50 years that a vegetation releases bacteria at a relatively constant rate. This explains the continuous bacteremia and the ability to culture bacteria from the blood even in the absence of fever. BCs are considered positive and significant even if fewer than 50 colony-forming units/mL of the microorganism are grown. Indwelling catheters are a common source for iatrogenic and nosocomial IE and they should also be cultured. Identifying the causative organism establishes the microbiological diagnosis so that specific antimicrobial treatment can be tailored accordingly. There is cool comfort in receiving a false-negative BC report as “culture-negative” IE is on the rise and a poor outcome is projected for the estimated 20% of patients who fall into this category (Baddour et al., 2005; Bayer et al., 1998; Beynon et al., 2006; Ferrieri et al., 2002; Mylonakis & Calderwood, 2001).

To reduce the possibility of a false-negative or “culture-negative” report: collect BCs prior to beginning antibiotic therapy (if this is not possible, withhold the antibiotic[s] [if the patient’s condition allows] for two to four days and repeat the BCs); obtain three sets of BCs (one aerobic and one anaerobic bottle) one hour apart; use a blood-broth dilution ratio of 1:5 for adults and less for infants and children (if it is not possible to obtain large volumes of blood, the emphasis, especially in children, is on the aerobic inoculation); and advise the laboratory that IE is suspected (if the BCs are negative after 48 to 72 hours, the laboratory should continue to incubate an additional two to three weeks to facilitate recovery of fastidious bacteria) (Bayer et al., 1998; Beynon et al., 2006; Ferrieri et al., 2002, Mylonakis & Calderwood, 2001).

An echocardiogram is very useful in confirming the diagnosis and is recommended in all patients with suspected IE. Echo-dense vegetations as well as any valve abscesses, prosthetic valve dehiscence, and valve regurgitation are usually well-visualized, especially via transesophageal echocardiography (TEE). TEEs, however, may give a false-negative result and should be repeated in seven to 10 days if the clinical suspicion of IE remains (Baddour et al., 2005; Bayer et al., 1998; Beynon et al., 2006; Mylonakis & Calderwood, 2001).

**Treating IE**

Lengthy treatment with bactericidal antimicrobial therapy is required in order to kill the infecting microorganism that lives sequestered and possibly dormant within the vegetation. Most NVE is treated for four to six weeks with double antimicrobial therapy. Some NVE patients (e.g., right-sided IE) and some pathogens (e.g., *Streptococcus viridans*) may respond well to a shorter course of treatment. All PVE patients, however, require six weeks of therapy, regardless of pathogen. When an NVE patient receives a prosthetic valve, the postoperative treatment needs to follow the recommendations for treating PVE. If the tissue specimen taken in the operating room is culture-positive, then the clock is reset and the entire six-week course is administered. If there is no growth on this tissue, then the clock continues and the duration of treatment is lessened by the number of days of therapy that has already been administered before surgery (Baddour et. al., 2005; Pound & Townsend, 2007).

Treatment is also dependent on the infecting microorganism and whether the patient has NVE or PVE. *Streptococcus* NVE or PVE is treated with β-lactam antibiotics (e.g., penicillin and ceftriaxone), which are often combined with an aminoglycoside (e.g., gentamycin) for synergy. *Staphylococcus* NVE is treated with nafcillin or oxacillin, or vancomycin if the patient is oxacillin-resistant. *Staphylococcus* PVE is treated the same with the addition of gentamicin or...
rifampin for synergy. *Enterococcus* NVE or PVE are both treated with β-lactam antibiotics or vancomycin combined with an aminoglycoside (gentamicin or streptomycin) for synergy. To maximize the synergistic (i.e., killing) effect, combination antimicrobial therapy should be administered as close together as possible. Serum peak and trough concentrations of vancomycin and gentamicin need to be regularly measured (Baddour et al., 2005; Pound & Townsend, 2007).

Medical treatment alone carries an extremely high (56% to 86%) mortality rate. Rates improve significantly (11% to 35%) when surgery is combined with medical therapy. Approximately 50% of IE patients will undergo surgery as part of their treatment plan. The decision to proceed with surgery is guided by the patient’s clinical status and findings known to be unresponsive to medical therapy (e.g., abscesses, fistulas, prosthetic dehiscence, obstructive vegetations, flail leaflets, conduction disturbances, recurrent emboli, drug toxicity, uncontrolled sepsis). Infection of the newly implanted valve is estimated at 2% to 3% and is a definite concern. Although not ideal, this risk is outweighed by the high mortality rate of delaying surgery and continuing with medical therapy alone. Surgery in active IE is high-risk and often requires extensive work to replace or repair the valves, drain abscesses, and close fistulas. The overall surgical mortality in this setting has been reported at 8% to 16%. The actuarial survival is reported as 75% at five years and 61% at 10 years. Although it seems contrary to surgical principles to implant foreign material into an infected area, once intractable CHF begins, a rapid downhill course can be expected and in spite of adequate antibiotics, surgery is the only viable option (Baddour et al., 2005; Bayer, et al., 1998; Beynon et al., 2006; Delahaye et al., 2004; Ferrieri et al., 2002; Iung Rousseau-Paziaud, Cormier, Garbarz, Fondard, Brochet, et al., 2004; Mylonakis & Calderwood, 2001).

All IE patients have a life-long risk of recurrent infection. Relapse usually occurs within the first two months after antimicrobial therapy is complete and is dependent on factors such as treating “culture-negative” IE; failing to treat according to the type of microorganism and/or type of IE (i.e., NVE, PVE); ongoing self-inoculation; and developing a nosocomial infection or iatrogenic IE. At the completion of therapy all IE patients should have repeat BCs and echocardiogram to establish their new baseline (Baddour et al., 2005; Mylonakis & Calderwood, 2001).

A multidisciplinary and specialist team approach is essential in treating IE. The team should include a microbiologist, infectious disease specialist, cardiologist, cardiac surgeon, clinical pharmacist, and nurses with the knowledge and skill to care for these vulnerable and highly complex patients. The services of a neurologist, neurosurgeon, intensivist, general surgeon, nephrologist, psychiatrist, drug rehabilitation specialist, social worker, dietitian, physiotherapist, occupational therapist, clinical psychologist, dentist, dental hygienist, and chaplain or spiritual care advisor may also be needed (Kothari & Hargrave, 2006; Mylonakis & Calderwood, 2001).

Nurses diagnose and treat the actual or potential problems patients may encounter during this illness. A dynamic care plan should be in place to address the evolving bio-psycho-social-spiritual needs of the patient and family. Nurses know which services need to be consulted and how to work their recommendations into the plan of care. Nurses are also ideally positioned to address issues related to prevention, recognition, diagnosis, and treatment of IE.

Preventing IE involves practising according to infection control standards and implementing the safety bundles recommended by “Safer Healthcare Now” (2007). A prevention strategy that all can participate in is hand hygiene, as it is the hands of health care providers that inadvertently inoculate some patients leading to a health care-associated infection. Patients and the entire health care team need to know about the revised AHA antibiotic prophylaxis recommendations. They also need to understand that revisions were called for as the previous recommendations were considered high-risk (e.g., increase in multiple resistant organisms) for poor return (e.g., unable to prevent most transient bacteremia).

Recognizing IE involves maintaining a high index of suspicion, monitoring the patient closely for complications, and conducting thorough cardiac assessments that include listening for a new or changing heart murmur. Diagnosing IE involves collecting BCs and communicating with the laboratory that IE is suspected so that the specimens will be kept an additional two to three weeks.

Treating IE involves managing venous access devices, giving the antimicrobial therapy on time and as close together as possible, understanding the limitations and complications of the antimicrobial therapy (e.g., ototoxicity, nephrotoxicity, *Clostridium difficile* colitis), monitoring for relapse and treatment effectiveness, ensuring that NVE patients who are stable and at low risk for emboli and CHF receive outpatient antibiotic therapy, and teaching patients about this multifaceted disease (Baddour et al., 2005).
IE touches all human dimensions. Nurses need to ensure that the focus is not exclusively on the physical, as the patient’s psycho-social-spiritual status is definitely altered by this life-threatening illness. Facing one’s mortality can be difficult and very stressful (Bayer, et al., 1998). As patients deal with this aspect they must also continue to cope with life and deal with issues such as family strain caused by separation during a lengthy hospital stay, financial life and deal with issues such as family strain caused by separation during a lengthy hospital stay, financial stress, concern, anxiety, fear, anger, resentment, disbelief. Yet, throughout the patient’s journey and regardless of prognosis or actual outcome, nurses are a constant, ready to provide compassionate, empathic, and stellar care.

**Conclusion**

This review has highlighted how IE, a centuries-old disease, remains a current-day challenge. The updated information shows how pathogen and patient profiles have evolved and parallel current health care issues and trends. The shift, and with it the concern over the prevalence of *Staphylococcus aureus* as the leading cause of this disease, along with the increase in MRSA and VRE are troubling and warrants our attention and action. The fact that the AHA has refined and limited its recommendations for antibiotic prophylaxis to include only high-risk cardiac patients and not all cardiac patients deemed to be at risk is a significant change. Put into practice, this recommendation may also serve to curb the antibiotic resistance that is on the rise. To unravel this clinical enigma requires that nurses understand how to prevent, recognize, diagnose, and treat this devastating disease.

**About the Author**

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**References**


Clinical practice questions

1. The statement that BEST defines infective endocarditis is:
   a) a bacterial infection of the endocardium, myocardium, and epicardium
   b) a microbial infection of the endothelial lining of the heart that can destroy the structural integrity of the heart and its valves and also cause a complex systemic illness
   c) a viral infection of the heart and great vessels
   d) a bacterial infection of the endothelial lining of the heart that destroys diseased or prosthetic valves

2. The leading cause of death for patients with infective endocarditis is:
   a) overwhelming infection
   b) embolic stroke
   c) congestive heart failure
   d) ruptured spleen

3. The microorganism most likely to cause infective endocarditis is:
   a) Staphylococcus aureus
   b) Enterococcus faecalis
   c) fungus
   d) Streptococcus viridans

4. A negative blood culture report will rule out infective endocarditis as a diagnosis?
   a) true
   b) false

5. The rise in the incidence of infective endocarditis is related to:
   a) increased lifespan both pre- and post-surgery for patients with defective valves and congenital heart defects
   b) improved cardiac surgical techniques and the ability to do complex surgical procedures
   c) increased use of implantable devices, prosthetic material, and prosthetic valves
   d) all of the above

6. Infective endocarditis is easier to diagnose and treat in which of the following patient populations?
   a) neonatal
   b) pediatric
   c) geriatric
   d) none of the above

7. Health care associated infection as a cause of infective endocarditis can be prevented by:
   a) proper hand hygiene
   b) implementing the Safer Healthcare Now bundle for preventing central lines
   c) implementing the Safer Healthcare Now bundle for preventing surgical sites
   d) all of the above

8. A patient with infective endocarditis may initially present in which of the following ways:
   a) stroke, afebrile, “flu-like” symptoms
   b) acute myocardial infarction, clubbing, normal white blood cell count
   c) left-sided abdominal pain, petechiae, new regurgitation murmurs
   d) all of the above

9. All patients with a known cardiac history should receive antibiotic prophylaxis prior to visiting the dentist?
   a) true
   b) false

10. After treatment of infective endocarditis is finished, a relapse is the result of:
    a) ongoing self-inoculation (e.g., poor oral health)
    b) causative organism was never identified
    c) treatment did not match with that recommended for the type of microorganism or type of IE (i.e. NVE, PVE)
    d) patient contracted a new health care associated infection during treatment
    e) all of the above

11. Surgery is recommended when which of the following occur:
    a) abscess and fistulae develop
    b) prosthetic valve dehiscence
    c) vegetations obstruct the valve orifice
    d) antimicrobial therapy is not controlling the infection
    e) all of the above