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Prosthetic Valve Endocarditis

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Abstract

The management of infections of the cardiac structures—specifically native heart valves—remains a difficult clinical challenge. Patients often present with a systemic infection that is made worse by embolic complications, such as strokes, along with pathophysiologic sequelae of acute valvular dysfunction. The timing of interventions has a significant impact on short- and long-term outcomes. The challenges and management decisions are even more complex when the infection involves a prosthetic valve—as risks of reoperative cardiac surgery can be substantial. The goal of this chapter is to discuss the history of prosthetic valve endocarditis, review the current literature on the management of specific valvular involvement (i.e., aortic and/or mitral), and illustrate the challenging problems and outcomes that drive clinical decision making. While many of the indications for surgery are similar to those associated with native valve infections, there is increased risk with reoperative surgery, often difficulties in clearing infection due to prosthetic material being in place. Unfortunately, antibiotics alone are not always effective, and frequent communications between the cardiac surgeon and infectious disease physicians are often necessary to find the “sweet spot” to perform the surgery.

Keywords: endocarditis, valve disease, aortic valve, mitral valve, cardiac surgery, prosthetic heart valves, infections

1. Introduction

Prosthetic valve endocarditis (PVE) is a rare, but serious complication of cardiac valve replacement surgery. As the prevalence of prosthetic valves increases, the incidence of PVE also rises. PVE constitutes approximately 20% of all cases of endocarditis, now greater than previous estimates of 1–5% [1]. The incidence of PVE is estimated at 0.3–1% per patient-year, with a

cumulative risk of 3% at 5 years and 5% at 10 years [1, 2]. The incidence of PVE in the aortic position is significantly higher than in the mitral position [3]. In comparison, the mitral valve is more commonly affected than the aortic valve in native valve endocarditis [4]. Patients undergoing simultaneous aortic and mitral valve replacement have an even greater risk of PVE than with a single valve replacement [5, 6].

2. Historical note

In 1885, Osler observed an association between perioperative bacteremia and endocarditis [7]. In 1935, Okell and Elliott noted that 11% of patients with poor oral hygiene had positive blood cultures for *Streptococcus viridans*, and that 60% of patients had bacteremia associated with dental extraction [8]. Not long after, initial reports of valve replacements by Starr and Harken, the first reports of PVE appeared in the literature [9, 10]. Before the routine use of prophylactic antibiotics, Geraci and Stein reported incidences of early PVE of 10 and 12%, respectively [11, 12]. The use of routine prophylactic antibiotics was noted to reduce the incidence of early PVE to 0.2% [11]. From the outset, the surgical management of PVE has been a formidable challenge. In the 1960s and 1970s, surgery for PVE was associated with an extremely high mortality rate. Discouraged by early operative experience, cardiac surgeons avoided intervention in cases of PVE despite recognition that antibiotic therapy alone was ineffective and often fatal. Surgery for PVE was reserved for high risk cases, and the surgical outcomes were predictably poor. Hence, a vicious cycle developed in which surgery was avoided for fear of poor surgical outcomes, and poor surgical outcomes achieved in high risk cases reinforced this fear.

In 1972, Ross successfully performed an aortic root replacement for PVE using an aortic homograft [13]. His report stressed surgical principles still true today: complete surgical debridement of all infected tissue, the use of homograft for reconstruction, and the minimal use of foreign material in the infected area [13]. In 1977, Olinger and Maloney reported replacement of an infected aortic prosthesis and external felt buttressing for correction of aortoventricular discontinuity [14]. In 1980, Frantz reported successful repair of an aortoventricular discontinuity from endocarditis and abscess formation by aortic root replacement using a synthetic valved conduit [15]. In 1981, Reitz successfully applied this technique to the treatment of prosthetic aortic valve endocarditis [16]. In 1982, Symbas combined aortic valve replacement with patch repair of a periannular abscess cavity [17]. In 1987, David and Feindel described techniques to reconstruct the mitral annulus with pericardium after debridement for PVE [18].

Surgical treatment of PVE remains a significant challenge, but outcomes improved in the 1990s. Factors that contributed to improved outcomes included:

1. widespread use of transthoracic and trans-esophageal echocardiography in making an early, accurate diagnosis,
2. an appreciation that, like surgical infections elsewhere, surgery for PVE requires radical debridement of infected and devitalized tissue,

3. improvements in myocardial protection, including routine use of retrograde cardioplegia, permitted longer and safer cardiac operations, and
4. cryopreserved homograft availability. Combined with resistance to reinfection, homografts provided flexibility in cardiac reconstruction after debridement. Currently, homograft aortic root replacement is considered the procedure of choice in the treatment of complex aortic PVE.

3. Risk

The risk of PVE to the patient is lifelong. However, as assessed by hazard function analysis, the risk of infection is greatest during the first 3 weeks after valve implantation [19]. Most deaths occur within 3 months of PVE development [18]. By clinical convention, PVE is classified as early or late [20]. Early PVE is acquired perioperatively and accounts for approximately one-third of all cases [20, 21]. Although traditionally defined as occurring within 60 days of initial valve replacement, the contemporary literature variably defines early PVE as occurring within 2, 6, or 12 months of initial valve replacement [20–22]. Late PVE results from infection unrelated to the initial valve operation and accounts for the remaining two-thirds [20]. The prognosis for early PVE is significantly worse than that of late PVE and often requires surgical intervention [20].

The distinction between early and late PVE provides insight into the acquisition of infection, expected clinical course, and appropriate management. Early PVE arises from the contamination of the valve during the perioperative period of valve implantation [20]. However, a patient with a prosthetic valve placed more than 12 months prior remains at risk for PVE commonly related to a healthcare-associated infection [20]. In a prospective, multicenter study of 171 patients with prosthetic heart valves by Fang et al., 43% developed endocarditis [23]. At the time, bacteremia was discovered, 33% had prosthetic valve endocarditis. These cases were described as having endocarditis at the outset. In comparison, 11% developed endocarditis at a mean of 45 days after the bacteremia was discovered. These cases were described as having new endocarditis. All cases of new endocarditis were health care associated with 33% developing bacteremia from intravascular devices [23].

Patients with central venous catheters are at particular risk for bacteremia. In the United States, approximately 80,000 central venous catheter-related cases of bacteremia are reported annually [24]. Urinary catheters are another source of bacteremia [25]. Catheter-associated bacteriuria develops at a rate of 8% per day in the first week of catheterization [25]. After the tenth day of catheterization, over 50% of patients are bacteriuric. Bacteremia develops in 0.4–4% of patients with catheter-associated bacteriuria [25]. Bacteremia per se does not invariably cause PVE. In a 10-year review of 890 patients by Parker et al., 3.6% undergoing cardiac valve replacement developed bacteremia in the early postoperative period. Only 6% of bacteremic patients developed PVE, though uniformly fatal [26]. Other authors have suggested that the risk of PVE may be significantly higher in cases of bacteremia; Murray reported an infective endocarditis rate of up to 25% in cases of *Staphylococcus aureus* bacteremia [27].

Although PVE secondary to candidemia is rare, accounting for 5–10% of all cases, it carries a high mortality rate [28]. In a retrospective study of 44 cases of nosocomial fungemia in patients with prosthetic heart valves by Nasser et al., 9% developed fungal endocarditis at a mean of 232 days after documented candidemia [29]. Hence, patients with candidemia must be treated aggressively in the acute setting and be provided close long-term follow-up.

Implantation of a prosthetic valve in the setting of native valve contamination without known active infection may increase the risk of PVE. For this reason, many surgeons routinely culture excised valve leaflets to ensure that the new valve is not contaminated at the time of implantation. In a study of 222 patients by Campbell et al., 14.4% who underwent elective valve replacement had positive valve cultures [30]. Coagulase-negative *Staphylococcus* was the most common bacterial isolate [30]. None of these patients had clinical evidence of infection. Only 3% of patients with positive valve cultures developed PVE. Most positive native valve cultures were thought to be false positives. Campbell concluded that positive cultures did not predict PVE and recommended against routinely obtaining native valve cultures [30].

Nonetheless, the potential morbidity and mortality of PVE may justify the practice of culturing excised valve tissue and treating patients with positive cultures. Intraoperative contamination at the time of valve implantation may occur from a variety of sources. Cardiac surgical procedures are complex and entail numerous intravascular monitoring devices as well as the circuit of the cardiopulmonary bypass machine. This complexity may contribute to the incidence of positive intraoperative blood cultures. In 1969, Ankeney and Parker reported positive intraoperative blood cultures in 19% of patients undergoing open cardiac surgery [31]. In a 1974 study of 66 patients undergoing open cardiac surgery, Kluge et al. reported a 71% incidence of positive intraoperative cultures from at least one site and a 20% incidence from two or more sites [32]. Several decades later, the issue remains unresolved.

In a 2004 study of 64 patients who underwent cardiovascular surgery, Shindo et al. reported positive intraoperative blood cultures in 16% of patients who underwent cardiopulmonary bypass [33]. Intraoperative blood salvage is routinely used in cardiac surgical procedures to avoid homologous blood transfusion. Autotransfusion is associated with lower risk of hypersensitivity reactions and infections compared to transfusion of homologous blood [33]. However, intraoperative blood salvage is associated with a high incidence of positive cultures. Shindo et al. reported positive blood cultures in 67% of cases using intraoperative blood salvage, excluding cardiopulmonary bypass [33]. In a 1992 study of 31 patients, Bland et al. reported positive cultures in 97% of cases using intraoperative blood salvage [34]. In a 1999 study of 10 patients by Reents et al., 90% of cases using a cell-saving device had bacterial contamination [35].

Hemodialysis has also been associated with endocarditis, particularly with the increasing prevalence of dialysis dependence. In a study of 329 patients with endocarditis by Cabell et al., 20.4% were hemodialysis dependent [36]. Hemodialysis was independently associated with the development of *Staphylococcus aureus* endocarditis. The frequency of hemodialysis dependence also significantly increased during the 7-year study period, from 6.7 to 21% [36]. There was a corresponding significant increase in *Staphylococcus aureus* endocarditis during

the study period, from 10 to 68.4% [36]. The prognosis of endocarditis in hemodialysis patients is poor, with in-hospital death rates of 25–45% and 1-year death rates of 46–75% [37].

Healthcare-associated infections are a significant source of PVE, accounting for 10–34% of all cases [38]. The majority of cases of healthcare-associated PVE develop more than 72 h following hospital admission [38]. The source of healthcare-associated PVE is frequently an intravascular device, such as a pacemaker or implantable cardioverter defibrillator. PVE is classified as healthcare-associated if it occurs within 1 year of device insertion [38].

4. Type of prosthesis

The incidence of PVE in mechanical and bioprosthetic valves is comparable [39]. Patients with mechanical prostheses have a higher risk of PVE in the first 3 months following valve replacement than those with bioprostheses [19]. The reason for higher risk of PVE in the early postoperative period with mechanical prostheses is unclear. Allografts lack prosthetic material and have a very low incidence of PVE in the early postoperative period. This suggests that mechanical prostheses have a tendency to develop early PVE, attributed to surface contamination at the time of surgery [19].

PVE in mechanical and bioprosthetic valves differs in anatomic involvement [40]. Infection of mechanical valves involves the junction between the sewing ring and annulus. This leads to the development of perivalvular abscesses, valve dehiscence, pseudoaneurysms, and fistulas. In comparison, infection of bioprosthetic valves is localized to the leaflets, leading to vegetations, cusp rupture, and perforation [40]. Endocarditis after mitral valve repair is rare. In a study of 30 patients, Gillinov et al. reported only 3% of cases of failed mitral valve repair as being caused by endocarditis [41]. In a study of 1275 mitral valve repairs over a 9-year period, Karavas et al. reported a 0.7% incidence of mitral valve endocarditis requiring surgical intervention [42]. The reason for this low incidence is likely related to less prosthetic material for potential infection with mitral valve repair than replacement.

4.1. Aortic valve prosthetic valve endocarditis

Aortic PVE is associated with substantial early morbidity and mortality. Regardless of the type of infected valve, mechanical or bioprosthetic, extensive tissue destruction may complicate aortic PVE. In a 20-year study of surgical treatment of aortic PVE by Perrotta et al., perivalvular abscess was reported in 83% of patients [43]. Comparably, Sabik et al. reported a 78% abscess rate in 103 patients with aortic PVE [44]. Abscess formation may be complicated by pseudoaneurysm and fistulisation [40]. Complete aortoventricular discontinuity has been reported in 40% of patients with aortic PVE [44]. Medical therapy alone has been associated with mortality rates as high as 70%, improved to 4–20% with surgical intervention. Significant risk factors for mortality include older age, higher preoperative creatinine, shorter interval from initial valve operation to reoperation for PVE, and fistula development. Mortality results from sepsis and multiple organ failure [44].

Aortic PVE is characterized by varying degrees of annular involvement. Extension of infection into the annular and periannular structures is a major determinant of both early and late surgical outcomes. The extent of valvular destruction relates to the virulence of the infecting organism and the duration of infection [45]. The inflammatory process of aortic PVE begins at the prosthetic sewing ring and extends through the aortic annulus, commonly in the region of aortomitral continuity [46]. The spectrum of periannular infection ranges from simple localized abscess to larger subannular aneurysm, with or without perforation into adjacent cardiac chambers. Progressive periannular infection may disrupt aortoventricular continuity or the aortomitral trigone, leading to intracardiac fistulae [44].

The goals of surgical intervention for aortic PVE include [44]:

1. complete debridement of infected and nonviable tissue,
2. repair of associated cardiac defects,
3. reconstruction of the aortic root, and
4. placement of a competent valve.

Reconstruction is complicated by severe destruction of the aortic root seen in PVE, characterized by development of abscesses, fistulas, aortoventricular discontinuity, and ventricular septal defects [47]. Achievement of the goals of surgical intervention for aortic PVE may require radical cardiac debridement. Failure to adhere to these principles poses significant risk for recurrent infection and valve dehiscence.

Following complete debridement, appropriate surgical reconstruction is guided by specific circumstances. In the majority of cases, an aortic root replacement is indicated [48]. A tension-free repair, excluding attenuated areas from high pressures, is essential [48]. If necessary, transmural sutures may be used to secure the conduit to the interventricular crest. Surgical principles dictate minimal use of synthetic material in the infected area. Aortic homograft is considered the replacement valve-conduit of choice in the treatment of aortic PVE [49]. Homograft vascular tissue is significantly more resistant to infection than prosthetic material. Aortic root replacement with homograft minimizes prosthetic material in the area of infection, thereby reducing risk of recurrent infection. The incidence of reinfection is low, ranging from 0 to 6.8% [49].

The use of allograft provides greater flexibility in the reconstruction of debrided areas [50]. Implantation may exclude abscess cavities from circulation by sewing the proximal anastomosis of the allograft to the inferior border of the abscess cavity [50]. Use of an aortic homograft with its attached mitral leaflet is particularly valuable in this regard [51].

The Ross operation, using pulmonary allograft, has been proposed as an alternative surgical option for the treatment of complex aortic PVE [51]. An initial study in 1994 by Joyce et al. of pulmonary allograft replacement reported success in six patients between 10 and 32 years of age with aortic valve endocarditis, with no mortality or reinfection [52]. In 2002, a retrospective study of 343 patients who underwent the Ross procedure by Takkenberg et al. reported

low operative mortality, but limited durability due to progressive dilation of the autograft root causing severe aortic valve regurgitation [53]. The Ross procedure is typically performed in critically ill patients and is used very selectively in PVE.

Morbidity and mortality associated with allograft aortic root replacement in the setting of PVE with involvement of the periannular region is significant [54]. A retrospective study of 32 patients with complicated aortic PVE who underwent allograft aortic root replacement by Dossche et al. reported annular abscess in 81%, aortomitral discontinuity in 43%, and aortoventricular discontinuity in 34%. There was a 9.4% operative mortality rate in this study, attributed to multiple organ failure and low cardiac output. The reported 5-year survival rate was 97.3%, and 5-year freedom from recurrent endocarditis was 96.5% [54]. As described, Sabik et al. reported similar rates of periannular abscess and aortoventricular discontinuity at 78 and 40%, respectively [44]. Reconstruction with cryopreserved allograft was associated with an in-hospital mortality rate of 3.9% in this study. Long-term survival rates at 1, 2, 5, and 10 years were 90, 86, 73, and 56%, respectively. Only 3.9% of patients required reoperation for recurrent PVE; 95% were free of recurrent PVE at 2 years [44].

Despite the advantages provided by allografts in the treatment of aortic PVE, their availability is limited. This has led to the use of mechanical valve-conduits for aortic root reconstruction with excellent results in the treatment of aortic PVE. Hagl et al. reported favorable results in a retrospective study of 28 patients who underwent aortic root replacement for PVE using prosthetic material rather than homograft [55]. Reported in-hospital mortality was 11%, and the incidence of recurrent endocarditis requiring reoperation was only 4% [55].

A study of 127 patients by Avierinos et al. compared the treatment of aortic endocarditis with aortic homograft in 43% and with conventional prosthesis in 57% [56]. In-hospital mortality was comparable between homograft and prosthesis at 11 and 8%, respectively. Prosthetic valve endocarditis was the only variable independently associated with in-hospital mortality. This mortality rate was not influenced by the type of valvular substitute, even in cases of annular abscess. There was no significant difference in endocarditis recurrence, prosthesis dysfunction, or cardiovascular mortality between aortic homograft and prosthesis at 10 years [56].

Aortic root replacement with stentless porcine xenografts has been developed as a surgical alternative in aortic PVE [57]. The stentless valve provides flexibility in reconstruction of the debrided myocardium. However, it places prosthetic material in the infected area, risking infection of the prosthetic valve-conduit. A study of 132 patients who underwent aortic root replacement with stentless porcine xenografts by LeMaire et al. reported a 7.6% mortality rate. There was a 6.8% incidence of late valve-related complications, including prosthetic endocarditis and annular pseudoaneurysm [57]. Reconstruction with cryopreserved allograft remains the preferred surgical strategy.

In addition to the difficulty associated with extensive resection of the prosthetic valve-conduit and surrounding tissue, two particular challenges must be overcome to replace the infected valve-conduit. The first challenge is reimplantation of the coronary artery ostia into the allograft. Scarring from the initial procedure may make it difficult to effectively mobilize

the left and right main coronary ostia for anastomosis to the allograft without undue tension. Raanani et al. described surgical reconstruction of the left main coronary artery using an autologous pericardial or saphenous vein patch [58]. The second challenge is achieving adequate resection and debridement of the distal graft-to-aorta anastomosis, which may require deep hypothermia and circulatory arrest. Furthermore, an allograft may not have sufficient length to reach the distal aortic anastomosis. Sabik et al. described the use of a second allograft to bridge the distance between the first allograft and the aorta [44].

High operative mortality rates have been reported for the replacement of infected valve-conduits, attributed to the degree of surgical difficulty. In a study of 11 patients with infected ascending aortic grafts who underwent composite valve graft placement by LeMaire and Coselli in 2007, a 30-day mortality rate of 46% was reported [59]. In comparison, a study of 12 patients who underwent composite replacement of the aortic valve and ascending aorta for infective endocarditis by Ralph-Edwards et al. reported an operative survival rate of 91.7% [60]. In this series, extensive debridement was performed, often requiring resection of the infected portion of the left ventricular outflow tract with circumferential reconstruction using bovine pericardium. It was often necessary to extend the length of the coronary arteries with saphenous vein or expanded polytetrafluoroethylene grafts to facilitate reimplantation as well [60]. As described, in a study of 23 patients who underwent ascending aorta and aortic valve replacement with the prosthetic material for acute PVE, Hagl et al. reported an 11% in-hospital mortality rate and a 4% incidence of recurrent endocarditis requiring reoperation at 4 months [55].

4.2. Mitral prosthetic valve endocarditis

Endocarditis is rare after mitral valve repair. The rate of freedom from endocarditis at 10 years following mitral valve repair is estimated at 95–99% [61]. Although native valve endocarditis can often be managed medically, PVE typically requires early operation. In a study of 22 patients with endocarditis after mitral valve repair by Gillinov et al., 68.1% underwent repeat mitral valve operations. Mitral valve replacement was required in 73.3%, and rerepair was performed in 26.7%. Following reoperation, 30-day, 1-year, and 5-year rates of freedom from reoperation were 65, 41, and 26%, respectively [61]. The principles of surgical management include the removal of all infected and devitalized tissue as well as the removal of the annuloplasty ring. If rerepair is not possible, replacement is necessary. Destruction of the mitral annular region is less common than periaortic annular destruction. Surgical debridement and resection of abscess formation in the posterior mitral annulus or in the region of aortomitral continuity is a significant surgical challenge, associated with a high operative mortality.

The mitral annulus may be reconstructed with autologous pericardium after debridement, as described by David and Feindel [62]. If the posterior mitral annular region requires reconstruction, this may be done with pericardium as well [15]. If necessary, the new mitral prosthesis may be translocated onto either the atrial or ventricular side of the annulus. If technically feasible, ventricular translocation may prevent exposure of the attenuated area to high pressure [15]. Aortomitral discontinuity is uncommon and particularly difficult to reconstruct. This trigonal region may be reconstructed using a modification of the technique described by Rastan et al. [63].

5. Operations with recent stroke

Neurologic sequelae occur in 25–70% of cases of infective endocarditis and portend increased mortality [64]. The mechanisms of neurologic injury include ischemic infarction secondary to embolization, hemorrhagic transformation of ischemic infarction, pyogenic arteritis, and rupture of intracranial mycotic aneurysm [65]. Systemic embolization occurs in 12.9% of patients with left-sided endocarditis after initiation of antibiotic therapy [66]. Of those with embolic events, 52% affect the central nervous system, and 65% occur within 2 weeks of initiation of antibiotic therapy [66]. Risk factors for embolization include vegetation size and mobility [66, 67]. There is no significant difference in incidence of embolization between native and prosthetic valve endocarditis. The risk of embolization is higher in mitral endocarditis than in aortic endocarditis [66].

The most common neurologic complication is ischemic stroke [65]. From a surgical perspective, the primary concern is hemorrhagic transformation of an ischemic infarct as a consequence of anticoagulation required during cardiopulmonary bypass [65]. Asymptomatic cerebral infarctions may occur in 30–40% of patients with endocarditis [64]. For this reason, it may be advisable to exclude an ischemic stroke with preoperative computed tomography. Clinically, silent or small infarcts should not delay cardiac surgery, since the risk of progression is low [64]. However, with the evidence of larger infarcts or intracerebral hemorrhage, surgical intervention should be delayed up to 4 weeks due to the associated risk of a significant neurologic event during cardiopulmonary bypass [64]. In such patients, the need for valve replacement should be balanced with high perioperative neurologic risk.

6. Indications for surgery

While there are a variety of resources available to assist in the decision making regarding interventions for prosthetic valve endocarditis, the key principles of therapy have been advocated by both American [68, 69] and European societies [70].

1. *Indications for surgery.*¹

- Valve dysfunction resulting in symptoms of heart failure (Class I).
- Left-sided infectious endocarditis caused by *S. aureus*, fungal, or other highly resistant microorganisms (Class I).
- Relapsing infection (Class IIa).
- Recurrent emboli and persistent vegetations despite appropriate antibiotic therapy (Class IIa).

¹Adapted from The American Association of Thoracic Surgeons consensus statement on the management of infectious endocarditis [68].

2. *Timing of surgery.*

- Once an indication for surgery is established, the patient should be operated on within days (Class I). Earlier surgery (emergency or within 48 hours) is reasonable for patients with large, mobile vegetations (Class IIa).
- Patients should be on appropriate antibiotic therapy at the time of surgery (Class I). Once a patient is on an appropriate antibiotic regimen, further delay of surgery is unlikely to be beneficial (Class IIa).

3. *Neurologic complications and surgery for PVE.*

- An operative delay of 3 weeks or more is reasonable among patients with recent intracranial hemorrhage (Class IIa).
- Patients with PVE and neurologic symptoms should undergo brain imaging (Class I); it is reasonable to screen patients with left-sided IE for possible stroke or intracranial bleeding prior to operation (Class IIa).

4. *Technical considerations.*

- Aortic PVE. If the root and the annulus are preserved after radical debridement in prosthetic aortic valve IE, implantation of a new prosthetic valve (tissue or mechanical) is reasonable (Class IIa). If there is annular destruction and invasion outside the aortic root, then the root reconstruction and use of an allograft or a biologic tissue root are preferable to a prosthetic valved conduit (Class IIa).
- Mitral PVE. When there are annular destruction and invasion, the annulus is reconstructed and the new prosthetic valve anchored to the ventricular muscle or to the reconstruction patch in a way to prevent leakage and pseudoaneurysm development (Class IIa).
- Among patients on dialysis, normal indications for surgery are reasonable, but additional comorbidities must be factored into assessments of risks and outcomes (Class IIa). Shorter durability of bioprostheses and allografts may be considered in the choice of valve prostheses used (Class IIa).

7. **Conclusions**

Without a doubt, the incidence of native valve endocarditis is growing-the reasons for this are multifactorial and, in general, reflect a greater access to advanced cardiac surgical therapies. Sicker patients, older patients, and more patients are undergoing valve replacement surgery for an ever-expanding list of indications. Increased use of vascular access, be it for chronic electrical system therapies (i.e., pacemakers and defibrillators), medical therapies (i.e., chemotherapy, dialysis), or as an extension of intravenous substance abuse, all have contributed to a growing incidence of both native and prosthetic valve infections. Regardless, any prosthetic valve replacement leads to a life-time risk that these patients face for the development of prosthetic

valve infections-either as a result of their initial operation, their ongoing (and potentially worsening) comorbidities, or simply as a function of patients living longer and with a cumulative annual risk. The development of prosthetic valve endocarditis is often, and appropriately so, viewed as a catastrophic event due to its association with devastating complications (i.e., strokes), substantial risk for operative morbidity and/or mortality, and baseline comorbidities and functional status at the time of presentation. More than most other medical and surgical therapies, a timely engagement by a multidisciplinary team is crucial to the establishment of a short- and long-term treatment plan. Clearly, much like native valve endocarditis, patients with prosthetic valve infections have shown benefit from early and aggressive surgical therapies-once established indications for surgery have been met or it has been demonstrated that optimized medical therapies have failed. Such therapies, despite substantial perioperative risks, must be focused on with aggressive debridement and elimination of all prosthetic and infected material. While prolonged courses of antibiotics and nonoperative management may have a role in select patients with limited disease burden, or for those in whom surgical reintervention is deemed to be a prohibitive, it must be recognized that the risk of treatment failure in such patients often results in worse complications or premature death. In conclusion, the medical and specific surgical decisions when dealing with a prosthetic valve infection must be individualized to provide the patient with the best opportunity for a cure.

Conflict of interest

None of the authors of this chapter have any disclosures or conflicts of interest to report in the context of the material presented.

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