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Valvular Heart Disease (J Dal-Bianco, Section Editor)

Tissue Valve Degeneration and Mechanical Valve Failure

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Abstract

Purpose of review The management of valvular heart disease has been dramatically influenced by recent evolutions in biomedical technology and surgical practice. With an aging population worldwide and accompanying increase in the prevalence of surgical valve disease, an understanding of prosthetic valve behavior and durability is essential for proper patient selection and management. This report offers an overview of the definitions, mechanisms, management, and clinical impact of structural valve degeneration and failure.

Recent findings Published literature has employed variable definitions and outcome measures, complicating our understanding of bioprosthetic valve behavior and function. The pathophysiology leading to structural valve degeneration is multifactorial and involves mechanical, hematologic, and immunologic elements. Technological advancements have resulted in improved valve performance and new strategies to mitigate the risks of degeneration.

Summary While mechanical valves have demonstrated negligible durability concerns, the benefits of bioprosthetic valves must be weighed against their potential for structural degeneration and subsequent reintervention. Valve selection should involve patient-specific deliberation, and guidelines have been established to help guide risk reduction strategies. Surgical valve replacement remains the standard of care for prosthetic valve failure, but emerging technology offers the potential to slow the development of structural degeneration and transcatheter valve-in-valve options are being increasingly explored.

Introduction

Valvular heart disease (VHD) impacts millions of people worldwide, necessitating 100,000 valve replacements annually in the USA and nearly 4 times as many across the globe each year. An association between advanced age and VHD has been previously demonstrated, and with epidemiological studies suggesting a dramatic rise in the elderly population in the coming decade, the prevalence of VHD can be expected to escalate in a corresponding manner [1, 2].

Valve replacement is known to improve both survival and quality of life for patients with severe disease [3]. Available prostheses are differentiated primarily by the composition of the valve leaflets—either mechanical or bioprosthetic—and the choice of prosthesis is a complex algorithm that must balance unique patient- and device-specific factors. While a thorough discussion of the advantages and disadvantages of each valve type is beyond the scope of this review, intrinsic differences in form, function, and outcomes can help to guide the clinical decision-making process.

Mechanical valves are known to offer superior structural durability but require lifelong anticoagulation and carry an associated increased incidence of bleeding events and stroke. By comparison, bioprosthetic valves mitigate thrombogenic risks through the elimination of obligatory anticoagulation but are associated with an increased risk of future reoperation. Largely due to their favorable hematologic profile and the intriguing prospect of future transcatheter valve-in-valve replacement, bioprosthetic valves have been used with increasing frequency in recent years [4]. However, the inevitable deterioration of bioprosthetic valves is a known—if poorly understood—phenomenon that must be carefully considered when evaluating a patient's candidacy for valve replacement.

The purpose of this review is to provide an understanding of structural valve degeneration (SVD) and prosthetic valve failure, including a discussion of the relevant intrinsic differences between mechanical and bioprosthetic valve construction, proposed mechanisms for valve degeneration, and definitions for standardization of diagnosis. In addition, we will discuss new directions for prevention and management of valve failure and discuss the impact of valve durability on preoperative patient selection.

Classification and definition

A significant hurdle in the evaluation of prosthetic valve durability is the lack of standardized terminology for the definition of SVD and valve failure. Published data involves a disparate set of outcome measures, definitions, and substitutes for valve degeneration that complicates the assessment of valve performance. The majority of studies rely upon *freedom from reoperation* as a primary surrogate for SVD or mechanical valve failure (MVF). While reoperation may provide diagnostic confirmation of valve deterioration, this approach underestimates the true incidence of SVD by failing to include patients considered too high risk for reintervention. In addition, a lack of consensus postoperative follow-up and imaging guidelines prevent consistent and reproducible comparisons between reported findings. Despite these challenges, several fundamental concepts pertaining to MVF and SVD can be extrapolated from the available data.

Structural vs. nonstructural valve dysfunction

In an effort to improve reporting and evaluation of reported clinical data, a joint commission from the American Association for Thoracic Surgery (AATS), the Society of Thoracic Surgeons (STS), and the European Association for Cardiothoracic Surgery (EACTS) released basic guidelines for reporting morbidity and mortality following valve surgery [5]. Nonstructural valve dysfunction (NVD) refers to any postoperative valvular abnormality or physiology not caused by the intrinsic valve components. Examples of NVD are largely iatrogenic and include: paravalvular leak, patient-prosthesis mismatch, leaflet entrapment by suture or pannus, thrombosis, and hemolysis. By contrast, structural valve deterioration refers to degenerative changes to the valve prosthesis itself, including fracture, leaflet tear, mechanical wear, and calcification. This basic distinction is essential for the interpretation of valve-related complications, separating intrinsic causes of valve dysfunction from a wide array of related physiologic and surgical phenomenon. This review will cover only structural causes of valve deterioration.

Mechanical valve failure

Mechanical valves have been in use longer than any other type of cardiac prosthesis, beginning with the Starr-Edwards caged ball valve in 1960 [6]. As a result of this decade-long experience, a considerable amount of data has been published regarding their clinical

durability. The landmark Veterans Affairs Hospital randomized control study was the first to demonstrate this advantage by reporting long-term outcomes in nearly 600 patients younger than 65 undergoing either aortic or mitral valve replacement [7]. The study revealed a significant reduction in primary valve failure—defined as postoperative obstruction or regurgitation—in patients with mechanical prostheses when compared with those receiving bioprosthetic valves. In fact, no patients experienced mechanical aortic valve failure in the study, and the four reported mitral valve failures were designated as technical complications—a distinction not eliminated by the study design. The superiority of mechanical valve durability has been duplicated in subsequent studies, including reports of more than 30-year survival, and a nearly 1000 patient experience from the Medical University of South Carolina without a single occurrence of structural valve failure requiring intervention or diagnosed on autopsy [8]. This advantage in mechanical valve design is associated with a decreased risk of subsequent reoperation. Structural MVF is thus an exceedingly rare event that confers a significant advantage in durability over bioprosthetic options.

Bioprosthetic structural valve degeneration

Bioprosthetic valves include a range of prostheses fashioned from chemically treated animal tissue—typically porcine valve leaflets or bovine pericardium—and constructed in a trileaflet configuration that is most often stent-mounted to facilitate implantation [9]. The principal advantage of bioprosthetic valves is the lack of need for lifelong anticoagulation, thus avoiding the thrombotic risk of a metallic prosthesis and obviating the associated bleeding risks. However, this physiologic advantage is counterbalanced by a reduction in durability afforded by the biologic tissue construction. While bioprosthetic valves have been proven to have a shorter lifespan than their mechanical counterparts, the lack of a universal definition for SVD complicates our understanding of this phenomenon.

The 2008 AATS/STS/EACTS guidelines define SVD as "dysfunction or deterioration involving the operated valve (exclusive of infection and thrombosis), as determined by reoperation, autopsy, or clinical investigation." [1] Other consortiums have proposed more specific definitions that incorporate clinical data such as an associated change in New York Heart Association functional class or echocardiographic parameters [10, 11, 12••]. However, these broad attempts at standardization have failed to impact widespread reporting measures or implementation of follow-up guidelines, making direct comparison of valve performance challenging. Highlighting this inconsistent reporting trend, published series within the last 15 years have employed more than 20 different criteria for defining SVD [13••].

SVD is considered to be an irreversible deterioration of bioprosthetic valvular function that is intrinsic to the valve components and can be demonstrated by objective clinical data and imaging. Multiple proposed echocardiographic grading systems have been proposed, with the EACTS Task Force for Prosthetic Valves suggesting a combination of hemodynamic and morphological criteria for characterization of SVD severity [12••]. For both the mitral and aortic position, hemodynamic parameters for *moderate* SVD using European metrics include a mean transprosthetic gradient between 20 and 40 mmHg, a 10–20 mmHg change in gradient from baseline, or the detection of new or worsening regurgitation. *Severe* SVD involves a mean transprosthetic gradient > 40 mmHg, a change in baseline > 20 mmHg, or severe regurgitation. In addition, morphologic changes permitting the diagnosis of SVD include leaflet tear, leaflet thickening, impaired mobility, or valve strut abnormalities.

Multiple large series have reported long-term outcomes for bioprosthetic valves with variable results. Typically evaluating a specific valve model, the majority of studies report outcomes at 5-year intervals—the largest of which extends to 20 years. The majority of these studies provide similar rates for survival without reintervention (the predominant surrogate for SVD in reported outcomes literature) after 5 and 10 years, with increasing variability during extended follow-up periods. In a large-scale series evaluating the Carpentier-Edwards bioprosthesis, Forcillo et al. reported survival without reintervention at 98, 96, and 67% at 5, 10, and 15 years, respectively [14]. The French have more recently published a similar experience with nearly 3000 patients that included rigorous echocardiographic criteria and demonstrated freedom from SVD at 15 and 20 years measuring 79 and 49%, respectively, and freedom from associated reintervention at 84 and 54% over the same timeframe, respectively [15]. Despite the variability in definitions, the available data suggests that clinically significant SVD is unusual within the first 10 years, but progressively develops thereafter (Fig. 1).

While most studies have suggested a trend toward improved durability with successive valve generations, this pattern is not without exception. The Sorin Mitroflow bioprosthetic valve was first introduced in

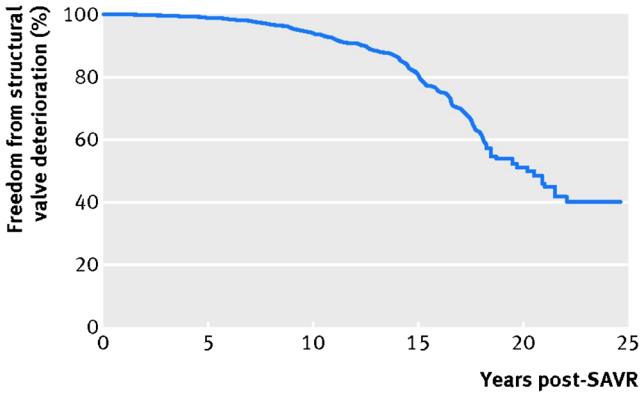

Fig. 1. Freedom from structural valve degeneration following bioprosthetic aortic valve replacement. Reproduced from Foroutan F, Guyatt GH, O'Brien K, et al. Prognosis After Surgical Replacement with a Bioprosthetic Aortic Valve in Patients with Severe Symptomatic Aortic Stenosis: Systematic Review of Observational Studies. BMJ. 2016 Sep 28;354, with permission from BMJ publishing Group Ltd.

the early 1980s and incorporated a unique design using a single sheet of bovine pericardium mounted upon a stent frame that results in a larger effective orifice area. Despite favorable hemodynamics well-suited for small aortic annuli, early generations demonstrated a predisposition for abrasive damage to the pericardial leaflets over time. A series of modifications introduced over subsequent decades ultimately led to the release of the Mitroflow LX pericardial valve, which eliminated the design elements responsible for leaflet tears but failed to incorporate anti-calcification measures thought to slow tissue degeneration. Early results with the Mitroflow LX demonstrated accelerated rates of valve degeneration and decreased survival when compared with the Carpentier-Edwards device [16]. These results were consistent with a simultaneous report from Boston Children's Hospital detailing their experience with universally rapid and life-threatening degeneration of Mitroflow prostheses within 1 year [17]. Many centers—including ours—have experienced similar episodes of early failure with the Mitroflow device in patients spanning all age groups, leading to the elimination of these valves from our inventory [18••].

Mechanisms and pathophysiology of valve failure

Understanding prosthetic valve dysfunction and failure requires recognition of the causative mechanisms resulting in their degradation. While in some cases, the underlying pathophysiology is known; the most common causative scenario for valve degeneration is likely multifactorial and represents an area of active study and investigation.

Structural failure of mechanical valves

First-generation mechanical valves—such as the Starr-Edwards caged-ball valve and Björk-Shiley tilting disc design—were known to have a risk of mechanical failure despite excellent long-term outcomes overall. Ball and disc fracture were reported along with dislodgement of the Björk-Shiley disc itself, but long-term follow-up nonetheless demonstrated a 91% freedom from reoperation rate at 30 years with survivorship reported as long as 51 years after implantation [19, 20]. These early-generation valves have since been replaced by modern bileaflet designs that confer improved physiologic performance and increased durability. Long-term outcomes data suggest that currently-available mechanical valves demonstrate no predisposition for structural failure [21]. While disadvantages can be highlighted in mechanical valve design and function, durability is not a significant concern.

Pathophysiology of bioprosthetic valve deterioration

Several mechanisms have been identified as playing a role in the pathogenesis of bioprosthetic SVD. The common endpoint of these degenerative pathways is an interactive combination of *calcification* and *degradation* that ultimately leads to failure of the leaflet apparatus. (Fig. 2a, b) Research efforts have identified a series of both active and passive processes by which SVD can occur. The interplay between these biologic mechanisms is likely complex and, as yet, incompletely understood.

Mechanical stress applied to bioprosthetic valve leaflets is a significant factor in the development of SVD. Animal studies have shown increased calcification on

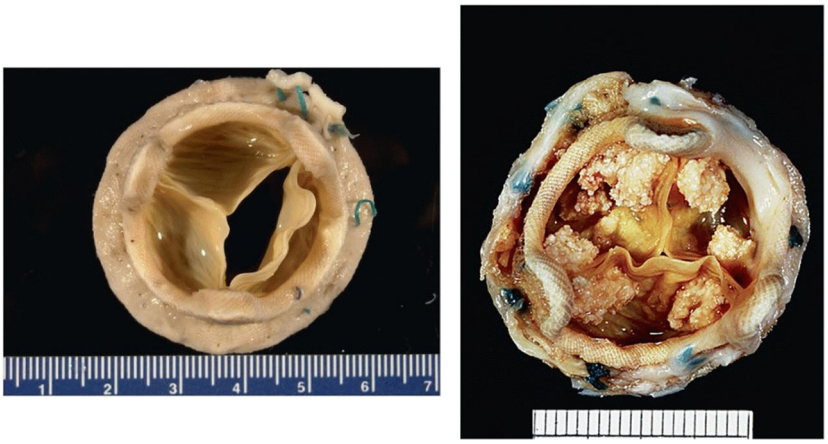

Fig. 2. a Porcine bioprosthetic valve without evidence of degeneration. Courtesy of Thoracic Key. b Porcine bioprosthetic valve with calcific degenerative changes. Courtesy of Thoracic Key.

leaflet components in proportion to the amount of shear stress applied to the apparatus [22]. In addition, histologic evaluation of explanted valves has demonstrated that leaflet tears and disrupted collagen fiber bundles are found in areas of high strain even in the absence of associated calcification. This suggests that mechanical stress plays an independent role in the development of SVD [23].

The manner in which bioprosthetic valves are manufactured may also contribute to their eventual degradation. Tissue valves are chemically treated with 0.6% glutaraldehyde solution in an effort to promote collagen cross-linking necessary for tissue preservation and eliminate cellular components within the xenograft that trigger immunogenicity. The altered extracellular membrane has been shown to contribute to a passive mode of calcification as a result of increased permeability to circulating calcium ions and the eradication of functional transmembrane ion pumps [24]. In addition, the glutaraldehyde is thought to promote phosphorylation of intracellular structures, thus predisposing to mineralization when exposed to high intracellular calcium levels [25].

While glutaraldehyde does mask xenoantigens to prevent rejection of the xenografted valvular components, the treatment does not completely eliminate the capability for residual antigenicity. Animal studies have demonstrated that residual immunologically active proteins result in the recruitment of inflammatory cells, activating humoral and cellular responses [26]. The inflammatory milieu created by the presence of macrophages, T cells, and transdifferentiated osteoblasts thus helps to establish an underlying process of active calcification that contributes to SVD.

Risk factors for bioprosthetic valve degeneration

Published data supports the association of several risk factors with the development of SVD. Patient age has been shown to be one of the most important determinants of SVD. Multiple studies have demonstrated that *younger* age at the time of implant is associated not only with increased rate of reoperation, but also with accelerated rates of SVD [27]. Available comparisons of a reported 10-year follow-up, for example, suggest evidence of SVD in just 10% of patients implanted above age 65, with rates as high as 30% in patients before age 40 [9, 28]. While these numbers may reflect some degree of variability in study design and management strategies, this discrepancy may also reflect a predisposition to robust immunologic response to residual xenoantigens in younger patients [29••].

Statistical analysis reveals several additional risk factors for the development of SVD that include patient comorbidities and valve-specific metrics. Common cardiovascular risk factors, such as diabetes, hyperlipidemia, hypertension, and smoking, all increase the risk of SVD, supporting an association between the lipid-mediated calcification seen in atherosclerotic disease and the degeneration of bioprosthetic valves [30]. Higher body mass index has also been associated with SVD, again raising comparisons with atherosclerotic risk factors while also contributing to an increased amount of mechanical strain on the valve leaflets. Similarly, valve size and reported prosthesis-patient mismatch has also been found to accelerate the development of

SVD, again due to an increased transvalvular gradient and excessive mechanical stress [31]. Patients with end-stage renal disease have been shown to be at increased risk for both native valve calcific disease and SVD, though outcomes studies have yet to demonstrate a survival disadvantage for bioprosthetic valves in this population [32].

Management strategies and emerging therapies

There is no therapeutic intervention known to reverse or prevent the development of SVD. However, new technologies and treatment strategies are being developed to mitigate the risk of both SVD and subsequent valve reintervention.

Prevention of valve dysfunction

Manufacturers are continually searching for design solutions to improve bioprosthetic valve durability. Newer generation valves boast improved hemodynamic performance and a relative reduction in SVD through the implementation of design elements that increase structural flexibility and expand the effective orifice area. In addition, chemical treatment of the biologic treatment has been modified in an effort to reduce free aldehyde formation and resultant calcification. A large-scale experience from Boston with the second-generation Carpentier-Edwards pericardial valve system demonstrated an 82% freedom from valve degeneration at 15 years [33]. These results were confirmed by a subsequent meta-analysis by Foroutan et al. that reported a 94 and 81% freedom from SVD at 10 and 15 years, respectively [34••].Performance of biologic prostheses is expected to continue to improve with evolving technology. Edwards' recent release of the Inspiris Resilia valve platform, for instance, boasts improved preservation strategies to reduce calcification, as well as an expandable stent design to allow for future valve-in-valve (ViV) replacements.

Management and valve replacement

The gold standard therapy for clinically significant prosthetic valve dysfunction remains surgical replacement. Clinical scenarios that dictate redo sternotomy are often complex and involve a wide range of comorbidities and indications, making determinations of operative risk challenging and patient specific. While efforts to refine patient selection are crucial, risks for reoperative surgery are nonetheless significant. Reported outcomes for isolated prosthetic aortic valve replacements suggest an operative risk between 5.8 and 9%, with 30-day mortality in redo mitral valve surgery slightly higher at 11.1% [35–37, 38••].The challenge of redo surgery highlights the clinical impact of valve degeneration.

With the advent of transcatheter aortic valve replacement (TAVR), much interest lies in the potential for valve-in-valve (ViV) therapies to mitigate the risks associated with redo sternotomy in those patients suffering from SVD. Interpretation of outcomes for this patient population is limited by the small sample size but is poised to be a topic of considerable interest as transcatheter technology continues to evolve. Early observational studies comparing redo sternotomy with transcatheter ViV replacement demonstrates similar 30-day mortality rates with increased risk of bleeding and kidney injury found in the surgical group while the percutaneous cohort were more likely to suffer from high transvalvular gradients and coronary obstruction [39]. AHA guidelines currently suggest consideration of transcatheter aortic ViV replacement for patients to be at high or prohibitive risk of reoperation [40]. The Edwards Sapien S3 TAVR valve is FDA approved for use in failing tissue valves in the mitral and aortic position for patients at high or greater risk of death or serious complications from traditional open-heart surgery. With the anticipated expansion of transcatheter therapies and technological advancements in the coming years, ViV approaches are likely to become more common as an alternative to reduce the periprocedural risks associated with reoperative sternotomy. ViV intervention for tissue valves in the pulmonic and tricuspid positions is currently under investigation.

Impact of durability on valve selection

The choice of valve prosthesis is a complex one that must balance clinical variables with patient preferences. (Fig. 3) While the full scope of advantages and disadvantages between valve types is beyond the scope of this review, the potential for SVD is a factor that must influence preoperative decision-making.

Patient age remains one of the most important considerations in the determination of valve candidacy. AHA guidelines recommend the use of bioprosthetic mitral and aortic valves for patients more than 70 years of age, and mechanical prostheses for patients younger than 50 (Class IIA) [30]. Recent publications have supported the implementation of age criteria. A retrospective population-level study from California recently reported a mortality benefit with the use of mechanical valves in the mitral position for patients up to 70 years of age, and for mechanical aortic valves until age 55

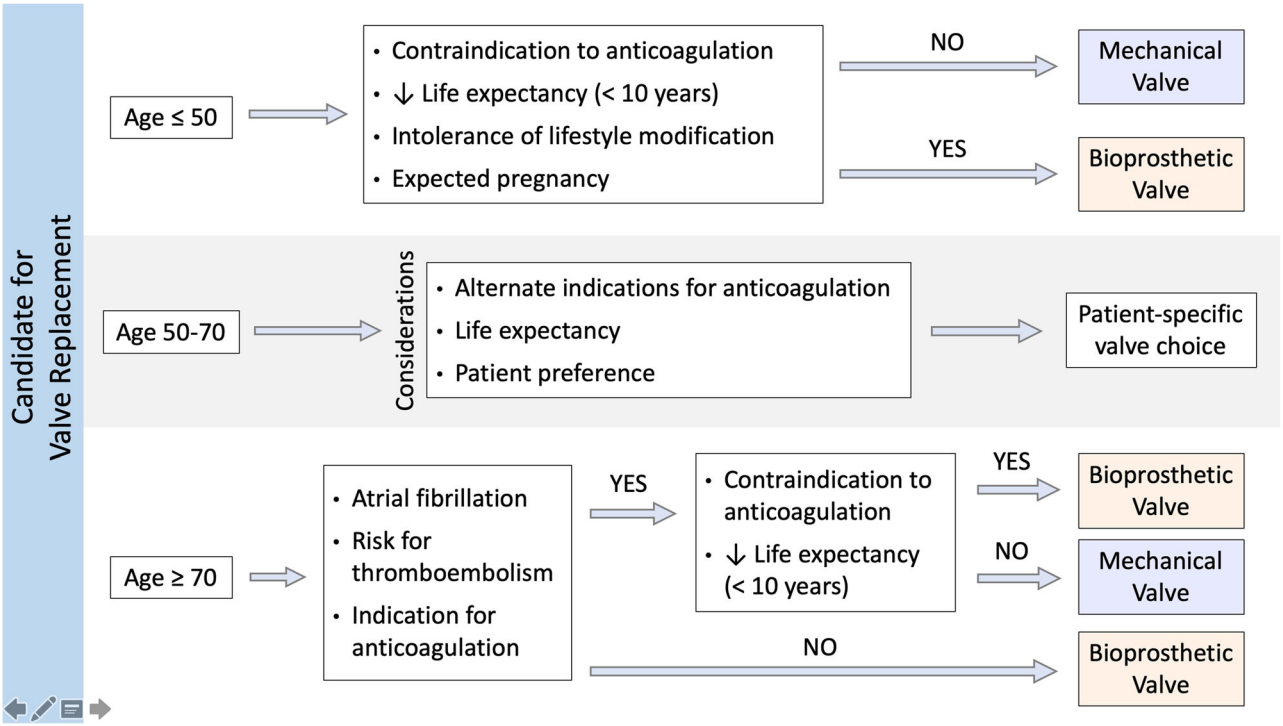

Fig. 3. Age-based decision-making algorithm for the appropriate selection of prosthetic valve options.

[41••].No consensus guidelines have been established for patients between the age of 50 and 70 due to the amount of conflicting data surrounding optimum valve choice. Patients within this age range must be counseled regarding the full scope of benefits and disadvantages associated with each valve.

Guidelines, however, do not supersede medical judgment, and a number of clinical exceptions should be considered. Female patients of childbearing age may be reluctant to commit to the lifelong anticoagulation required for mechanical valve therapy and the subsequent risks to potential fetal survival. Patients with known intolerance to anticoagulation should also avoid mechanical prostheses. Younger patients with comorbid conditions may not benefit from the survival advantages afforded by mechanical valves, while other patients may be unable to tolerate the lifestyle modifications required for long-term anticoagulation use [42]. It is the clinician's responsibility to accurately convey the risks and benefits of each valve type and allow patients to make an informed decision.

Conclusion

The choice of valve prosthesis is complex and increasingly influenced by evolving patient populations, emerging technology, and improved understanding of valve performance. While mechanical valves have demonstrated negligible durability concerns, the benefits of bioprosthetic valves must be weighed against their potential for SVD. Published literature has employed variable definitions and outcome measures, and standardization of reporting will improve our understanding of bioprosthetic valve behavior. The pathophysiology leading to SVD is multifactorial and involves mechanical, hematologic, and

immunologic elements. While the choice of valve should involve patient-specific deliberation, mechanical valves are recommended for patients younger than 50 due to a heightened risk of SVD. Surgical valve replacement remains the standard of care for prosthetic valve failure, but emerging technology offers the potential to slow the development of SVD and avoid the risks of revision sternotomy.

Compliance with Ethical Standards

Conflict of Interest
The authors declare that they have no conflicts of interest.

Human and Animal Rights and Informed Consent
This article does not contain any studies with human or animal subjects performed by any of the authors.

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