Structural Valve Deterioration and Bioprosthetic Valve Failure

Definitions, mechanisms, and treatment options.

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Treatment strategies for severe symptomatic aortic stenosis have evolved rapidly during the last few decades. Historically, the field has been dominated by surgical replacement of the native diseased valve with a new mechanical prosthesis. This strategy has been challenged by the need for long-term anticoagulation and its inherent risk of bleeding, particularly in elderly patients. Subsequently, biological prostheses, typically made of porcine and bovine pericardium, entered the scene—first in the surgical field and then in the emerging field of transcatheter interventions. The increased biocompatibility of these prosthetic valves addressed the shortcoming of long-term anticoagulation required by mechanical valves, albeit at the price of implanting a biological structure that is more prone to degeneration. Hence, the quest for the "ideal valve" continues.[1]

Since the first transcatheter aortic valve replacement (TAVR) procedure in 2002, devices, techniques, and experience have significantly improved, with indications expanding from symptomatic inoperable patients (or those deemed to be at high surgical risk)[2] to patients at intermediate surgical risk[3-5] and, more recently, to patients at low surgical risk.[6-9] TAVR is usually offered to elderly individuals across the spectrum of risk, but younger patients (including those in age categories where a surgical bioprosthesis is currently offered) could be potentially targeted in the future.

However, the long-term durability of transcatheter bioprostheses is less characterized compared with their surgical counterparts, and the actual lifespan of TAVR bioprostheses is unknown. As such, the idea of expanding TAVR indications to younger patients, who are expected to survive longer, requires long-term follow-up data.[10-13] Importantly, to meaningfully collect these outcomes, definitions of valve durability should be standardized and easily applicable to allow comparability among procedures and valve iterations.

DEFINITIONS OF STRUCTURAL VALVE DETERIORATION

Comparing the results of studies reporting structural valve deterioration (SVD) of surgical or transcatheter bioprosthetic valves is challenging because of their heterogeneous definitions.[14] Initially, most studies exclusively referred to SVD as the need for reoperation, resulting in underreporting of other meaningful valve-related outcomes.[14] Other definitions based on symptoms[15] and echocardiographic criteria[16,17] have been introduced over time, but substantial heterogeneity remained. The Valve Academic Research Consortium (VARC) document was the first authoritative attempt to standardize the reporting of TAVR outcomes.[18] It was followed by an update published in 2012 (VARC-2)[2] and will be followed by a second update (VARC-3). More recently, the European Association of Percutaneous Cardiovascular Interventions (EAPCI) published a consensus definition of valve durability endorsed by the European Society of Cardiology (ESC) and the European Association for Cardio-Thoracic Surgery (EACTS), followed by a position paper from the VIVID registry.[1,14]

VARC and VARC-2

Only minor changes occurred between the two versions of the document concerning definitions of valve durability. Echocardiography is considered the gold standard for assessing prosthetic valve dysfunction in patients with recurrent symptoms, with CT suggested as a second-line investigation. Stenosis and regurgitation

(and associated hemodynamic dysfunction) are identified as the main mechanisms of failure, with specific thresholds for normal, mild, moderate, and severe disease. Echocardiographic assessment is suggested before discharge (ideally, 24–48 hours after the index procedure), at 1 month, at 1 year, and annually thereafter.[18] In the VARC-2 document, 1-month assessment is only suggested for more invasive approaches (ie, transapical or transaortic), with a first evaluation 6 months after hospital discharge for other patients.[2] Prosthetic valve thrombosis and endocarditis have also been included as potential mechanisms for bioprosthetic valve failure (BVF), with accompanying specific treatment algorithms.[18]

EAPCI/ESC/EACTS Definition

SVD is defined in the EAPCI/ESC/EACTS document as a "permanent intrinsic change of the valve (ie, leaflet tear, calcification, pannus deposition, flail, or fibrotic leaflet) leading to degeneration and/or dysfunction." In this context, the presence of symptoms is not required to define SVD. However, the concept of BVF emerges when SVD is accompanied by clinical manifestations.[1] In light of this, potentially reversible causes of bioprosthetic valve dysfunction, such as thrombosis or endocarditis, are not included in the SVD definition but are considered separate entities. At the same time, events that are not strictly related to the valve itself (eg, prosthesis malposition resulting in paravalvular regurgitation, patient-prosthesis mismatch, late embolization) are categorized separately (Figure 1).[1]

Two specific types of SVD can be considered: hemodynamic and morphologic. Hemodynamic SVD (ie, isolated hemodynamic dysfunction) with or without evidence of morphologic abnormalities is characterized by permanent hemodynamic changes assessed by echocardiography. Moderate hemodynamic SVD is defined as a mean transprosthetic gradient ≥ 20 mm Hg and < 40 mm Hg and/or a mean transprosthetic gradient change ≥ 10 mm Hg and < 20 mm Hg from baseline and/or new or worsening moderate (> 1+/4+) intraprosthetic aortic regurgitation. Severe SVD is defined as a mean transprosthetic gradient ≥ 40 mm Hg and/or a mean transprosthetic gradient change ≥ 20 mm Hg from baseline and/or new or worsening severe (> 2+/4+) intraprosthetic aortic regurgitation.[1]

Morphologic SVD requires abnormalities of at least one of the following, even in the absence of reintervention or significant hemodynamic changes: leaflet integrity (ie, torn or flail causing intraframe regurgitation), leaflet structure (ie, pathologic thickening and/or calcification causing valvular stenosis or central regurgitation), leaflet function (ie, impaired mobility resulting in stenosis and/or central regurgitation), or strut/frame (ie, fracture).[1]

VIVID Definition

The definition of SVD in the VIVID consensus document[14] substantially resembles the main elements of the EAPCI/ESC/EACTS definitions but introduces the concept of progressive SVD stages over time, with specific management recommendations according to the degree of dysfunction and the underlying pathophysiology (Figure 2).

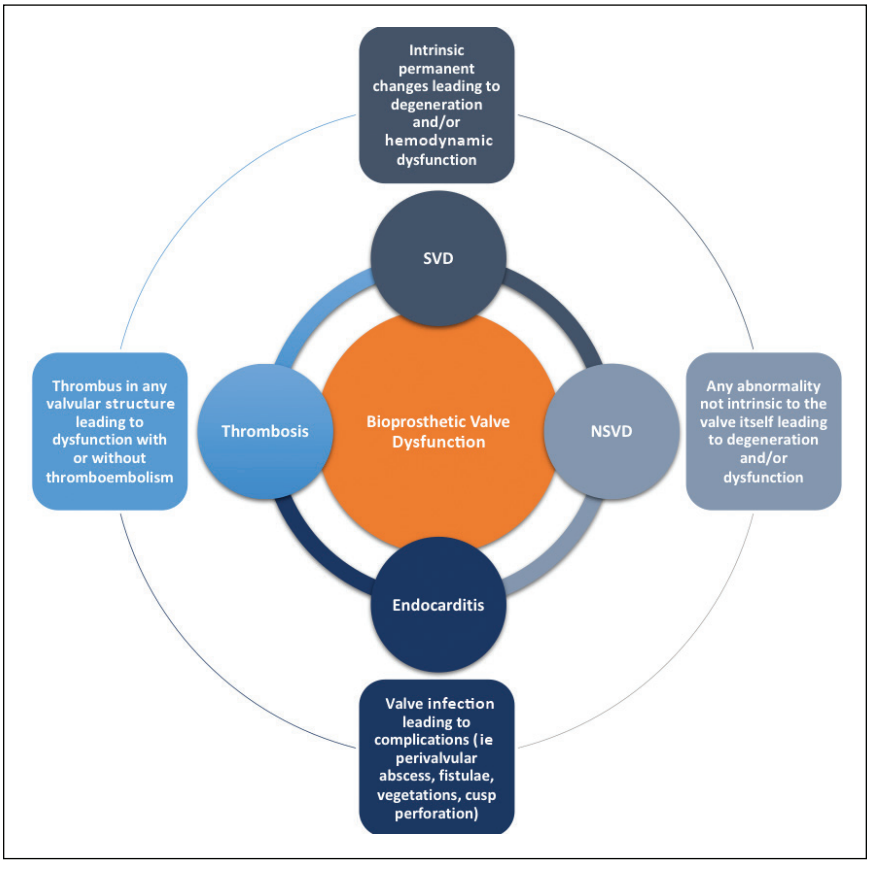

Figure 1. EAPCI/ESC/EACTS definition for bioprosthetic valve dysfunction. NSVD, nonstructural valve deterioration.

PATHOPHYSIOLOGY

The main difference between native and bioprosthetic heart valves is represented by the intense tissue activity caused by continuous functional remodeling and repair of injuries secondary to repetitive deformations.[19] Fabrication and fixation of bioprosthetic heart valves are key features associated with postimplantation modifications (if any) and subsequent SVD. Specifically, nonvital tissue, lack of endothelium at the blood-cusp interface, and use of glutaraldehyde make the valve itself more prone to blood cell and fluid penetration ("cuspal hematoma" and "fluid insudation," respectively), impairing its in vivo dynamics and tissue endothelization.[19,20] Historically, glutaraldehyde has been widely accepted as one of the leading factors eventually associated with SVD. Although glutaraldehyde reduces allograft immunogenicity and promotes collagen linkage to establish a solid tissue structure, it also impairs the rearrangement of the extracellular matrix and irreversibly modifies cellular permeability to calcium and phosphorus, favoring the appearance of calcification deposits.[21]

SVD may affect the valve leaflets through two different mechanisms: cellular calcification and noncalcific extracellular matrix degradation.[22,23] The "intrinsic mineralization" process usually involves the commissural zones near the basal attachment of the cusp margins. Thrombi or infective vegetations may also promote calcification ("extrinsic mineralization").[22,24,25] In this context, loss of tissue vitality inhibits energy-dependent pumps that maintain a physiologically low intracellular concentration of calcium. Reaction with phosphorus promotes intracellular deposits and calcification of collagen and elastin.[26,27] On the contrary, areas of maximal tissue deformation and shear stress, such as the free leaflet extremities ending in the nodulus of Arantius, are predisposed to developing noncalcific valvular deterioration because of loss of physiologic flexural stiffness.[28,29] Calcific and noncalcific deterioration are strictly related because an initial extracellular matrix damage may promote the formation and growth of fluid insudation and nucleation sites.[19]

DIAGNOSIS

Echocardiography

Transthoracic echocardiography is the gold standard for early patient evaluation and follow-up assessment,[1,14] and it allows for both morphologic and hemodynamic valvular assessment according to current definitions. In some cases, transesophageal imaging or three-dimensional reconstruction may be useful for more enhanced assessment of prosthetic valve dysfunction.

Multidetector CT

Higher-resolution anatomic analysis is provided by multidetector CT (MDCT), especially when subclinical valve thrombosis or pannus formation are suspected.[14] Eccentricity index, hypoattenuated leaflet thickening, motion reduction, and calcification are key elements in determining the causes of SVD.[28,30] However, hemodynamic assessment is not feasible with MDCT, hence limiting its role and diagnostic value.[1]

TAVR DURABILITY

Although the first TAVR procedure was performed in 2002, the technique was only introduced in daily clinical practice in 2007. In contrast, surgical aortic valve replacement (SAVR) was introduced in 1960, allowing a much longer follow-up and more extensive literature regarding valve durability. Durability data relevant to the TAVR population are limited and, at best, report

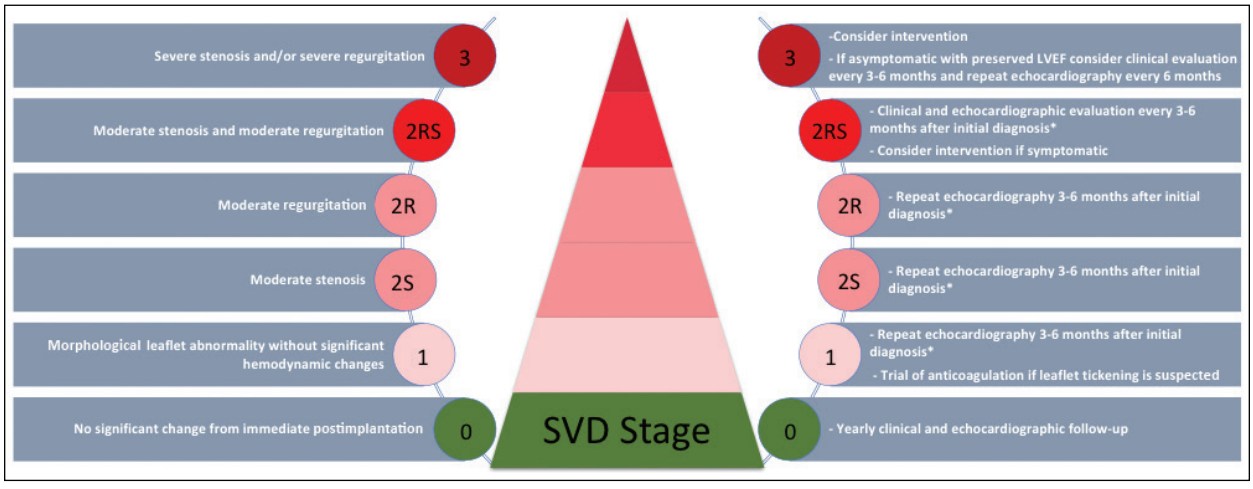

Figure 2. VIVID definitions of and recommended management of SVD (* = then, if stable, every 6-12 months). LVEF, left ventricular ejection fraction.

"mid-term follow-up" (5–8 years). The reason is attributable not only to the temporal gap between the birth of the two procedures but also because of the competitive risk of death in such an elderly cohort.[1,14,31,32] A recently published study from Aldalati et al reported mid-term SVD outcomes of TAVR (mainly using balloon-expandable valves) versus SAVR.[33] Based on the VIVID and VARC-2 definitions, respectively, the SVD rates for TAVR were 11.5% and 28.3% at a median follow-up of 33.4 months compared with 19% and 31% at 54 months for SAVR.[33] The varying performance of different generations of balloon-expandable bioprostheses compared with SAVR bioprostheses comes to light in intermediate-risk patients from the PARTNER 2A trial and S3 registry.[34,35] These results displayed better long-term outcomes for the new-generation Sapien 3 valve (Edwards Lifesciences) compared with the second-generation Sapien XT valve (Edwards Lifesciences). Severe SVD according to a modified EAPCI/ESC/EACTS definition was documented in 8.8% of Sapien XT patients and 3.5% of SAVR patients at 5 years (P = .002). On the other hand, the 4-year rate of severe SVD for TAVR with the Sapien 3 valve and SAVR were 2.6% and 2.5% (P = .86), respectively.[35] Other comparisons of high-risk TAVR and SAVR patients at 5 years using the EAPCI/ESC/EACTS definition come from the CoreValve United States pivotal high-risk trial, in which the rates of severe SVD were 0.8% for the self-expandable CoreValve (Medtronic) TAVR cohort and 1.7% for the SAVR cohort (P = .32).[36] Furthermore, in low-risk patients from the NOTION trial, the rates of severe SVD at 6 years for TAVR with the CoreValve self-expandable bioprosthesis versus SAVR were 0.7% and 3%, respectively.[37] Testa et al recently reported the 8-year outcomes of almost 1,000 patients treated with a CoreValve self-expandable TAVR device according to the EAPCI/ESC/EACTS definition (22% alive at 8 years). The cumulative incidences of moderate SVD, severe SVD, and BVF were 3%, 1.6%, and 2.5%, respectively.[32] These data are consistent with other studies reporting ≥ 5-year outcomes of TAVR with self-expandable and/or balloon-expandable valves using the EAPCI/ESC/EACTS definition, with an overall incidence of 3.6%–10.8%, 0%–2.5%, and 0.6%–7.5% for moderate SVD, severe SVD, and BVF, respectively.[32,36-44]

Overall, the durability data from the available registries seem acceptably similar to those from surgical series at up to 5 to 8 years. Nevertheless, even though the scientific literature on longer follow-up in the TAVR population is rapidly expanding,[33] it is still too early to know whether TAVR bioprostheses will behave the same way as their surgical counterparts at 10 to 20 years.

DURABILITY EXPECTATIONS OF TAVR AND SAVR BIOPROSTHESES

First-generation transcatheter devices differed substantially from their surgical counterparts, mainly due to the lack of anticalcification treatment and different leaflet tissues employed (eg, equine for the Cribier-Edwards valve; Edwards Lifesciences).[14] Nowadays, gaps between different bioprosthesis in tissue biology and subsequent treatment have been progressively addressed, thereby limiting the considerations regarding different SVD outcomes between TAVR and SAVR to mostly procedure-related features. First, remaining native valve leaflets and the lack of a complete surgical toilette could negatively affect stent frame expansion and leaflet distension.[14] Second, in-lab studies have suggested that crimping of the leaflets allows for transcatheter valve delivery and could provoke microscopic tissue damage.[45,46] Third, both underexpansion and overexpansion, especially when balloon postdilatation is performed, may result in different mechanical stresses.[14] Fourth, progressive reduction of sheath diameters over time, paralleled by the engineering of thinner valve materials and use of greater compression forces, allow the bioprosthesis to enter the delivery system.[14,31] These features may disadvantage TAVR compared to SAVR in the long run. Conversely, transcatheter bioprostheses might have certain advantages, including (1) a reduced rate of patient-prosthesis mismatch, (2) larger mean areas after the procedure, and (3) a greater margin of improvement resulting from the continuous iterations of devices and techniques.[44]

RISK FACTORS FOR SVD

The onset of SVD seems to be influenced by several patient- and prosthesis-related risk factors. Younger age, mitral location, hypertension, and pathologies involving calcium and phosphorus metabolism (eg, end-stage renal disease or hyperparathyroidism) are among the most reported patient-related risk factors.[24,47-49] Diabetes mellitus, metabolic syndrome, and an excess of low-density lipoprotein could also favor SVD through lipid-mediated inflammation.[21,47] Among prosthesis-related factors, the implantation of smaller devices and use of valve-in-valve procedures may increase the mechanical leaflet stress, potentially facilitating SVD (Figure 3).[21,50]

The putative influence of different tissue biologies (ie, bovine or porcine) remains controversial; some studies affirm that porcine valves behave worse in the long term, whereas others found no difference between these two types of tissue.[51,52] Nevertheless, most studies agree that new-generation valves are more durable than older models.[24,47,49] Among the new-generation devices, it has been suggested that the supra-annular

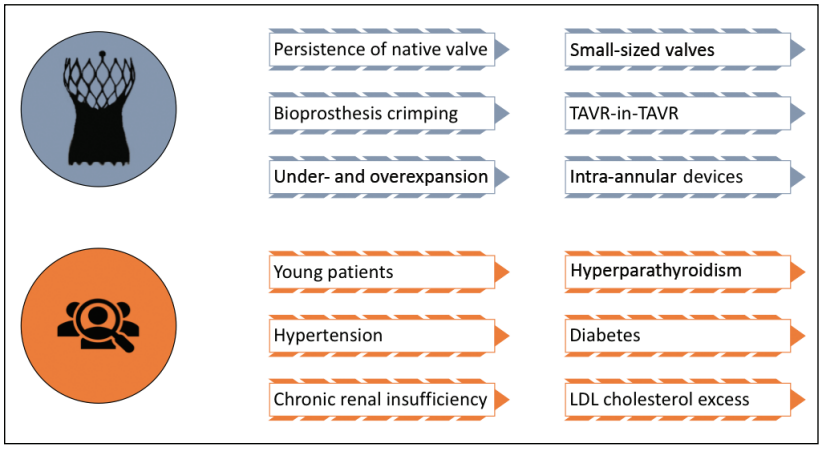

Figure 3. Procedural (blue) and patient-related (orange) risk factors for SVD. LDL, low-density lipoprotein.

design could hypothetically reduce SVD when compared to the annular design. The supra-annular design promotes sealing in noncircular annuli, maintaining circularity at the level of coaptation, forming larger effective orifice areas, and decreasing the likelihood of a neosinus, which could represent a source of thrombus formation.[53] An unpublished substudy of the CHOICE trial[54], a head-to-head comparison between balloon-expandable (annular) and self-expandable (supra-annular) prostheses, reported a statistically significant difference in moderate SVD in favor of the self-expandable–treated cohort at 5 years (0% vs 5.6%; $P = .047$)[44]; however, the number of patients available for the analysis was small, and more evidence is warranted.

TREATMENT OPTIONS

Anticoagulant and antibiotic therapy followed by strict echocardiographic follow-up should be considered the first therapeutic option for nonhemodynamically relevant leaflet thrombosis or infective endocarditis.[55] However, reintervention is generally necessary when BVF is linked with nonreversible symptoms or hemodynamic consequences. Open heart surgery allows for complete valvular toilette and de novo implantation of another bioprosthetic valve. Nevertheless, given the high risk of a second surgery, many patients are referred for a valve-in-valve procedure, which consists of delivery of a new bioprosthesis inside the previously implanted dysfunctional one. The current literature mostly agrees that patients undergoing TAVR-in-TAVR are more prone to SVD due to mechanical and hemodynamic factors.[50]

CONCLUSIONS

Expanding indications to lower-risk and, possibly, younger patients is one of the main points of debate in the current TAVR era. Standardized definitions and grading of SVD are now available and allow for meaningful comparisons between procedures. Different mechanisms and risk factors are involved in the onset of SVD. Although continuous improvements in tools, techniques, pharmacotherapy, and operator experience are apparent, all bioprostheses are expected to fail at some point. In light of this, robust long-term data are essential to better investigate the incidence and mechanisms of SVD before offering TAVR to a broader cohort of younger patients. ■

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