Management of Failed Bioprosthetic Aortic Valves: Mitigating Complications and Optimizing Outcomes

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The use of bioprosthetic prostheses during surgical aortic valve replacements has increased dramatically over the last two decades, accounting for over 85% of surgical implantations. Given limited long-term durability, there has been an increase in aortic valve reoperations and reinterventions. With the advent of new technologies, multiple treatment strategies are available to treat bioprosthetic valve failure, including valve-in-valve (ViV) transcatheter aortic valve replacement (TAVR). However, ViV TAVR has an increased risk of higher gradients and patient-prosthesis mismatch (PPM) secondary to placing the new valve within the rigid frame of the prior valve, especially in patients with a small surgical bioprosthesis in situ. Bioprosthetic valve fracture allows for placement of a larger transcatheter valve, as well as a fully expanded transcatheter valve, decreasing postoperative gradients and the risk of PPM.

1. Introduction

Treatment of aortic valve pathology has evolved over the past decade with the advent of transcatheter aortic valve replacement (TAVR). In Europe, TAVR first received Conformité Européenne (CE) Mark approval in 2007, and the number of patients undergoing TAVR grew exponentially. In the United States (US), clinical trials began in 2007 and TAVR gained Food and Drug Administration (FDA) approval in 2011 for inoperable patients with severe aortic stenosis. Since then, surgical aortic valve replacements (SAVR) have decreased slightly as TAVR approval expanded to patients of all surgical risk profiles in 2019 [1]. However, overall aortic valve replacements, including TAVR and SAVR, have increased [2].

More than 85% of SAVRs are with bioprosthetic valves [3], but one of the major limitations is durability. Bioprosthetic valve dysfunction (BVD) can be categorized as either nonstructural valve deterioration (NSVD)--paravalvular regurgitation, patient-prosthetic mismatch (PPM), malposition, valve embolization, valve thrombosis, or endocarditis, or structural valve deterioration (SVD)--permanent intrinsic changes to the valve [4]. Valve durability is dependent on the valve manufacturer and type of prosthesis. SVD is an irreversible process resulting in hemodynamic and clinical changes similar to native valve aortic stenosis and regurgitation, eventually resulting in the need for reoperation. SVD definitions differ in the literature, leading to varying rates of reported valve failure. In most SAVR series, valve failure has been defined as a need for reintervention, but this is not a true “incidence of failure.” Patients can experience significant SVD without undergoing reoperation due to the underdiagnosis of SVD, minimalization of SVD severity, or patients not being considered surgical candidates [5].

The 2021 Valve Academic Research Consortium 3 (VARC-3) guidelines define bioprosthetic valve failure in three stages: (1) any bioprosthetic valve dysfunction with clinically expressive criteria (new-onset or worsening symptoms, left ventricular dilation/hypertrophy/
dysfunction, pulmonary hypertension, or irreversible stage three hemodynamic valve deterioration), (2) aortic valve intervention, and (3) valve-related death [6] (Figure 1). With the increased use of bioprosthesis valves, an increase in reoperations or reinterventions for BVD is predicted. Management strategies continue to evolve and range from traditional redo-sternotomy SAVR, minimally invasive redo-SAVR, and placement of a TAVR valve in a failed SAVR, also known as valve-in-valve (ViV).

2. The Problem

2.1. Risk Factors for Bioprosthetic Valve Failure. Given the increase in bioprosthetic AVR utilization, the identification of predictors of valve failure and the recognition of opportunities to reduce the incidence of SVD are imperative. A variety of factors contribute to valve failure, including patient characteristics and comorbidities, type of implanted valve, and size of implanted valve. In a recent systematic review and meta-analysis, Ochi et al. identified multiple risk factors for BVD including younger age, sex, prosthesis brand, prosthesis size (<19 mm, <21 mm, <23 mm), PPM, absence of anticalcification preparation, concomitant coronary artery bypass graft surgery, subcoronary implantation technique, postoperative pressure gradient, dyslipidemia, smoking, metabolic syndrome, use of lipid lowering medication, elevated body mass index and body surface area, and renal disease. Meta-analysis identified younger age (per 1-year increase in age, HR = 0.91, p < 0.0001), increased body surface area (HR = 1.77, p = 0.03), smoking (HR = 2.28, p = 0.0015), and PPM (HR = 1.95, p < 0.0001) as the four significant determinants for SVD [7] (Table 1).

2.2. Patient-Prosthesis Mismatch. Patient-prosthesis mismatch occurs when the effective orifice area (EOA) of the implanted prosthetic valve is too small for the patient’s body size [8]. PPM is defined by indexed EOA/body surface area (BSA) and is stratified by severity as follows: none (>0.85 cm²/m²), moderate (0.85 to 0.65 cm²/m²), and severe (≤0.65 cm²/m²). Fallon et al. reported that 65% of patients ≥65 years old with severe aortic stenosis who underwent SAVR had moderate or severe PPM [9]. Those patients with moderate or severe PPM had a significantly increased risk of readmission for heart failure (moderate, HR = 1.15, [95% CI: 1.09, 1.21]; severe, HR = 1.37, [95% CI: 1.26, 1.48]) and redo AVR (moderate, HR = 1.41, [95% CI: 1.13, 1.77]; severe, HR = 2.68, [95% CI: 2.01, 3.56]). Any degree of PPM has been associated with significantly lower survival [9, 10]. Older age, female sex, hypertension, diabetes, renal failure, larger BSA, and larger BMI have been identified as risk factors for PPM [11, 12]. TAVR has been associated with a decreased risk of PPM compared to SAVR, especially in patients with small aortic annuli. Alaeei-Andabili et al. found the incidence of PPM was almost double following SAVR compared to TAVR (54% vs. 29%, p < 0.001), especially among patients receiving a valve size ≤23 mm (SAVR, 65% vs. TAVR, 48%, p = 0.048) [13]. The average aortic valve size implanted in the US is 22 mm [3], leaving many patients with the risk of PPM, early valve failure, and increased mortality. PPM can be mitigated at the time of initial AVR by implanting an appropriately sized valve.

2.3. Valve Sizing. When selecting a valve, the internal orifice diameter (ID) of the proposed implant should be identified, as the ID differs amongst valve models and manufacturers for the same labeled valve size. The largest valve that can be safely implanted is recommended, but strategies for selecting valve size differ in SAVR vs. TAVR. For SAVR, the valve size is selected by the surgeon at the time of implant based on manufacturer-specific annular valve sizes, while TAVR sizing relies entirely on preoperative computed tomography angiography (CTA). This difference in measurement results in valves with smaller ID being implanted during SAVR [14]. Preoperative CTA analysis defines the aortic annulus and root anatomy, allowing for an appropriately sized implant, SAVR or TAVR, to be selected. If a small aortic prosthesis is predicted, a root enlargement or root replacement can be performed at the time of SAVR. Alternatively, the initial valve that provides the largest EOA and best hemodynamics is often utilized at the time of TAVR. Many structural heart teams will assess every patient with CTA to ensure the most appropriately sized implant is utilized.

Especially in young patients with small annuli, an aortic root enlargement or replacement should be performed when the EOA index is ≤0.65 cm²/m² and may be considered when the EOA index is ≤0.85 cm²/m² [15]. Aortic root enlargement has not been widely adopted and is performed in <10% of SAVRs [3, 16]. Techniques for root enlargement include Nicks [17], Manouguian and Seybold-Epting [18], Konno et al. [19], and the Y-incision [20]. Both the Nicks and Manouguian procedures enlarge the aortic annulus via posterior extension of the aortic root—the Nicks through the noncoronary sinus and the Manouguian through the left/ noncoronary commissure, extending onto the anterior mitral leaflet, then closure with patch augmentation [17, 18]. The annular patch enables the implantation of a valve size 1–2 sizes larger than the native annulus [21]. A Konno, rarely done in adults, is an anterior annular augmentation extending onto the right ventricle [19]. The Y-incision, also a posterior enlargement, undermines the left and noncoronary cusps and enables implantation of a valve 2–3 sizes larger, with reports of up to 5 sizes larger [20, 22, 23]. A posterior aortic root enlargement is not associated with increased risk of mortality or adverse events at expert centers and can facilitate future ViV TAVR but absolutely precludes balloon fracture as the native annulus is unsupported [16, 24].

3. Solutions

Once clinically significant BVD occurs, intervention is indicated. Redo-SAVR may not be appropriate for all patients and a full imaging assessment with CTA and heart team discussion is necessary to determine the best strategy. In Europe, ViV TAVR was first CE Mark approved in 2013 and FDA approved for inoperable and high-risk patients (30-day
Bioprosthetic Valve Failure
(as defined by VARC-3; Eur Heart J, 2021)

Stage 1
Any bioprosthetic valve dysfunction with clinically evident criteria: new-onset/worsening symptoms, left ventricular dilatation/hypertrophy/dysfunction, pulmonary hypertension, or irreversible stage three hemodynamic valve deterioration

Stage 2
Aortic valve intervention

Stage 3
Valve-related death

Figure 1: definition of Bioprosthetic Valve Failure. Adapted from VARC-3*. *Varc-3 Writing C, Genereux P, Piazza N, et al. Valve Academic Research Consortium 3: Updated Endpoint definitions for Aortic Valve Clinical Research. J Am Coll Cardiol. 2021; 77: 2717–2746.

### Table 1: Risk /protective factors for SVD.

| Risk factors                        | Hazard ratio (95% confidence interval) | p value   |
|-------------------------------------|----------------------------------------|-----------|
| **Risk factors**                    |                                        |           |
| Younger age                         |                                        |           |
| Age per 1 year decrease             | 1.10 (1.06, 1.12)                      | <0.0001   |
| Increasing BSA                      | 1.77 (1.04, 3.01)                      | 0.034     |
| PPM                                 | 1.95 (1.56, 2.43)                      | <0.001    |
| Smoking                             | 2.28 (1.37, 3.79)                      | 0.0015    |
| **Protective factor**               |                                        |           |
| Anticalcification preparation       | 0.41 (0.19, 0.89)                      | 0.025     |
| **Older age**                       |                                        |           |
| Age >60 years                       | 0.12 (0.06, 0.23]                      | <0.0001   |
| Age >65 years                       | 0.06 (0.02, 0.21)                      | <0.0001   |
| Age >70 years                       | 0.06 (0.01, 0.28)                      | 0.0004    |

BSA = body surface area; PPM = patient prosthesis mismatch. Adapted from Ochi A, Cheng K, Zhao B, Hardikar AA, Negishi K. Patient Risk Factors for Bioprosthetic Aortic Valve Degeneration: A Systematic Review and Meta-Analysis. Heart Lung Circ. 2020; 29:668–678.

surgical mortality >8% by STS PROM) with the balloon expandable valves [25] and subsequently with self-expandable valves in 2015 [26]. One of the limitations of ViV TAVR is the risk of severe PPM, since the transcatheter valve sits within the surgical valve’s true ID. This is especially true in smaller surgical valves and has been associated with higher one-year mortality in the Valve-in-Valve International Data Registry; surgical valves ≤21 mm had significantly higher one-year mortality (25%) compared to valves ≥23 and ≤25 mm (18%), and valves ≥27 mm (7%) (p = 0.001) [27]. Surgical prosthesis ≤21 mm (HR = 2.04, [95% CI: 1.14, 3.67], p = 0.02) and stenosis as the primary mechanism of failure (vs. regurgitation; HR = 3.07, [95% CI: 1.33, 7.08], p = 0.008) were significant risk factors for one-year mortality [27]. To improve the hemodynamic results of ViV TAVR, different techniques can be employed, including implanting the transcatheter valve high within the surgical valve, utilizing a supra-annular transcatheter valve, and bioprosthetic valve fracture (BVF) and bioprosthetic valve remodeling (BVR).

3.1. Bioprosthetic Valve Fracture and Bioprosthetic Valve Remodeling. The bioprosthetic valve fracture was first described by Nielsen-Kudsk et al. in 2015. In small mitroflow bioprostheses, a high-pressure balloon predilatation with an ATLAS Gold balloon fractured the annular stent ring of the SAVR valves and a 20 mm SAPIEN XT was placed in the 19 mm Mitroflow and a SAPIEN 3 23 mm TAVR valve was placed in a 21 mm mitroflow without any complications [28]. The BVF allows for greater expansion of the transcatheter valve and the implantation of a larger, more fully

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expanded, transcatheter valve. However, BVF is not an option for all patients. Bioprosthetic valve remodeling (BVR) is similar in concept with the intention of fully expanding the TAVR without fracturing the surgical valve annulus. Although BVR can improve the gradient across the valve and leaflet coaptation of the more fully expanded annulus. Although BVF is similar in concept with the intention of fully expanding the TAVR without fracturing the surgical valve (BVR) is used to predict the risk of coronary occlusion as the SAVR leaflets are pushed toward the coronary ostia during ViV TAVR and create a complete tube of leaflet tissue that can reach the level of the sinotubular junction (STJ). Coronary height, the distance from the coronary ostium to the aortic valve annulus, is one of the important factors to consider when evaluating risk for coronary obstruction [34]. Lower coronary heights are more often seen in patients with in-situ surgical valves compared to those with native valves. Therefore, in ViV TAVR planning, the coronary height should be measured from the sewing ring of the basal plane of the prosthesis and not the true native annular plane [35]. On preoperative CTA analysis, crucial factors include identification of the failed leaflets, bioprosthesis angulation in relation to the aortic annulus, coronary ostia height, sinus of Valsalva diameter, STJ height, and SAVR leaflet length. The distance from the surgical valve leaflet to the coronary ostia, the valve to coronary (VTC) distance, predicts the feasibility of ViV TAVR and the risk of coronary obstruction. A VTC of 4 mm or greater is necessary for ViV TAVR (Figure 2). Stentless bioprosthetic valves and stented bioprosthetic valves with externally mounted leaflets have an increased risk of coronary obstruction. Those at highest risk for coronary obstruction are female patients, coronary ostial height <10 mm, sinus of Valsalva (SOV) diameter <30 mm, VTC distance <4 mm, and previous aortic bioprostheses, particularly those with stented valves with externally mounted leaflets or stentless surgical valves (OR 7.67, [95% CI: 3.14, 18.7], p < 0.0001) [36]. When BVF is performed, the gain in annular dimension is 3-4 mm; therefore, the VTC distance should be at least 5 mm in order to accommodate valve expansion [37]. Additionally, when evaluating for BVF, the SOV diameter and STJ height must be measured to ensure the sinus is large enough to accommodate the increased valve size without root rupture or sinus sequestration and the STJ height is adequate to accommodate full leaflet excursion without the leaflet reaching the level of the STJ (minimum valve to ST) distance of 2 mm is suggested) [38].

BVF results in an increase in the ID of the surgical valve of 3-4 mm and the selection of transcatheter valve size should be based on this anticipated increased ID. BVF can be performed before or after ViV TAVR. When performed before ViV TAVR, it effectively fractures the surgical valve but does not ensure adequate expansion of the subsequent TAVR. If BVF is performed after ViV TAVR, it fractures the.
surgical valve and fully expands the transcatheter valve. BVF has been shown as a beneficial strategy to prevent PPM, in particular for small surgical prostheses. Despite patients with larger surgical valves having a lower risk of PPM and high gradients, BVF can still be utilized to promote full transcatheter valve expansion. However, BVF remains understudied in patients with larger surgical valve sizes [39]. BVF results in reduced transvalvular gradients and increased EOA; for optimal results, it is suggested that BVF be performed after ViV TAVR and with a non-compliant balloon at least 3 mm larger than the true ID of the surgical valve being fractured [30] but with a balloon no larger than the waste of the self-expanding valve to avoid damage to the valve leaflets.

### 3.1.4 Adjunctive Techniques

During SAVR, commissure-to-commissure alignment is maintained, while in TAVR the orientation of the commissures is often random and not consistently achievable. Tang et al. found that the Evolut "hat"
### Table 3: Surgical prosthesis amenable to valve remodeling.

| Make                  | Stent/Stentless | Leaflets | Valve sizes | ID (mm) | Profile height (mm) |
|-----------------------|-----------------|----------|-------------|---------|--------------------|
| **Trifecta**          |                 |          |             |         |                    |
| Abbott                | Stented         | External | 19          | 17      | 15                 |
|                       |                 |          | 21          | 19      | 16                 |
|                       |                 |          | 23          | 21      | 17                 |
|                       |                 |          | 25          | 23      | 18                 |
|                       |                 |          | 27          | 25      | 19                 |
| **CE standard porcine** |               |         |             |         |                    |
| Edwards lifesciences  | Stented         | Internal | 19          | 17      | 15                 |
|                       |                 |          | 21          | 19      | 16                 |
|                       |                 |          | 23          | 21      | 16                 |
|                       |                 |          | 25          | 23      | 18                 |
|                       |                 |          | 27          | 25      | 18                 |
|                       |                 |          | 29          | 27      | 19                 |
|                       |                 |          | 31          | 29      | 19                 |
| **CE supra-annular**  |                 |         |             |         |                    |
| Edwards lifesciences  | Stented         | Internal | 21          | 19      | 15                 |
|                       |                 |          | 23          | 21      | 16                 |
|                       |                 |          | 25          | 23      | 17                 |
|                       |                 |          | 27          | 25      | 17                 |
| **Inspiris resilia**  |                 |         |             |         |                    |
| Edwards lifesciences  | Stented         | Internal | 19          | 18      | 13                 |
|                       |                 |          | 21          | 20      | 14                 |
|                       |                 |          | 23          | 22      | 15                 |
|                       |                 |          | 25          | 24      | 16                 |
|                       |                 |          | 27          | 26      | 17                 |
|                       |                 |          | 29          | 28      | 19                 |
| **Perimount 2700**    |                 |         |             |         |                    |
| Edwards lifesciences  | Stented         | Internal | 19          | 18      | 13                 |
|                       |                 |          | 21          | 20      | 14                 |
|                       |                 |          | 23          | 22      | 15                 |
|                       |                 |          | 25          | 24      | 16                 |
|                       |                 |          | 27          | 26      | 17                 |
|                       |                 |          | 29          | 28      | 18                 |

*Figure 2: Procedural preplanning with 3D Reconstruction and virtual valve in a failed 21 mm Magna surgical valve. With the smallest sized balloon expandable valve, 20 mm, the valve to coronary (VTC) distance to the left main coronary ostium (2.5 mm) and valve to sinotubular junction (VTSTJ) (1.4 mm) are not adequate.*
marker and the ACURATE-neo commissural post facilitated improved commissural alignment and reduced coronary artery overlap [40]. The crimping of the Sapien 3 valve had no impact on commissural alignment predictability in the study. Commissural alignment facilitates coronary access and future options for transcatheter management if the ViV TAVR fails. This is an area of active investigation as younger and lower-risk patients, with a long life expectancy, receive ViV TAVR.

Bioprosthetic or native Aortic Scallop Intentional Laceration to prevent Iatrogenic Coronary Artery Obstruction (BASILICA) is an electrosurgical leaflet modification technique which is effective in preventing coronary obstruction in native and bioprosthetic valves. In patients at high risk of coronary obstruction due to a VTC distance <4 mm, or at risk of sinus sequestration due to a narrow SOV, short STJ height, and/or long bioprosthetic valve leaflets, using an electrified wire, the nadir of the bioprosthetic leaflet is crossed and leaflet lacerated to create a V shaped opening (leaflet splay) to increase blood flow and access to the coronary artery at risk. In patients in whom BASILICA is predicted to result in inadequate “splay” (particularly problematic for failed TAVR valves and the feasibility of TAVR-in-TAVR), balloon-assisted BASILICA can be utilized to expand the traversal point outward by balloon inflation prior to laceration [41].

3.2. Surgical Techniques. In addition to ViV TAVR, redo-SAVR is another option. Although redo-SAVR has traditionally been considered a higher-risk operation compared to primary AVR, the mortality of redo SAVR is 1–5% with appropriate patient selection [43,44]. Comparing ViV TAVR and redo-SAVR in a single center series, those undergoing ViV TAVR were older and had more comorbidities including peripheral arterial disease, congestive heart failure with NYHA class III or IV symptoms, hypertension, prior myocardial infarction (MI), and history of atrial fibrillation; however, postoperative outcomes were similar. The ViV-TAVR group had shorter lengths of stay while the redo-SAVR group had improved hemodynamics [44]. In a meta-analysis comparing ViV TAVR and redo-SAVR with degenerated bioprosthetic valves, all cause 30-day mortality was higher in the redo-SAVR group and there was no significant difference in stroke, MI, or permanent pacemaker at mid-term follow-up of up to 5 years. However, ViV TAVR was associated with a higher risk of PPM and greater transvalvular pressure gradients postimplantation [45]. Both ViV TAVR and redo-SAVR are viable options and patient selection is key to success; higher-risk patients and patients with larger valves benefit more from ViV TAVR while younger patients and patients with smaller valves benefit more from redo-SAVR.

4. Conclusion

Due to the increase in bioprosthetic valve utilization for the treatment of aortic valve disease and patients with longer life expectancy, bioprosthetic valve failure is becoming a significant problem requiring innovative treatment strategies. Redo-SAVR has traditionally been the only treatment modality for failed biologic valves, but many elderly patients are not candidates for a second operation or do not wish to undergo a redo-sternotomy. Valve fracture provides one strategy to achieve optimal hemodynamics by increasing the size of the annulus for ViV TAVR. BVF is especially useful in patients with small surgical valves to decrease the risk of PPM by removing the constraints of placing a transcatheter valve within a rigid surgical bioprostheses and when performed after ViV-TAVR facilitates expansion of the transcatheter valve. Although in the US ViV TAVR is reserved for high-risk patients, risk drift is expected with this technology. Not only do we need to provide a solution to the initial failed surgical valve, but planning for a third valve when the ViV TAVR fails must be considered in the lifetime management of aortic valve disease. It may be that all patients, not just those with small annuli, benefit long-term from valve fracture and additional study is needed.

Abbreviations

AVR: Aortic valve replacement  
BASILICA: Bioprosthetic or native aortic scallop intentional laceration to prevent iatrogenic coronary artery obstruction  
BSA: Body surface area  
BVD: Bioprosthetic valve dysfunction  
BVF: Bioprosthetic valve fracture  
BVR: Bioprosthetic valve remodeling  
CE: Conformité européenne  
CTA: Computed tomography angiography  
EOA: Effective orifice area  
FDA: Food and drug administration  
HR: Hazard ratio  
ID: Internal diameter  
LVOT: Left ventricular outflow tract  
MDCT: Multidetector computed tomography  
MI: Myocardial infarction  
NSVD: Nonstructural valve deterioration  
NYHA: New York heart association  
PPM: Patient-prosthesis mismatch  
SAVR: Surgical aortic valve replacement  
SVD: Structural valve deterioration  
TAVR: Transcatheter aortic valve replacement  
US: United states  
VARC-3: Valve academic research consortium 3  
ViV: Valve-in-valve.

Data Availability

This is a review article; data can be found on Pubmed as per the list of references.

Conflicts of Interest

The authors declare that there are no conflicts of interest. AG is a proctor for Edwards Lifesciences and Abbott Vascular;
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