# Transcatheter aortic valve replacement in failed surgical valves

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## ABSTRACT

Aortic valve-in-valve is a less invasive alternative to surgical redo in the treatment of failed bioprosthetic valves. While only inoperable patients underwent the procedure before, operators currently offer it to those at lower risk and worldwide experience is in the thousands. Early mortality has diminished in recent analyses and improvements in symptoms and quality of life have been documented. Main considerations with aortic valve-in-valve include elevated postprocedural gradients, coronary obstruction and leaflet thrombosis. Risk factors for each of these adverse events have been described at length. Aortic valve-in-valve offers a safe and effective option in the management of failed bioprosthetic valves.

## INTRODUCTION

The last two decades have seen a dramatic increase in the use of bioprosthetic, rather than mechanical, valves for patients requiring aortic valve replacement[1] (figure 1). While these devices obviate the need for long-term anticoagulation, the key disadvantage to their use is structural valve degeneration.[2] Some devices have reported rates of freedom from reintervention over 95% at 10 years, but this number substantially drops to about 60% by 20 years follow-up.[3 4] Not all devices are this successful, with some demonstrating greater rates of failure earlier after implantation.[5] The definitions of valve degeneration have been variable, with several studies using reintervention as the only marker of failure and others, more recent, considering echocardiographic parameters.[2] International groups have attempted to standardise the field by publishing definitions featuring a stepwise process including echocardiographic changes and also the need for reoperation.[2 6]

Patients with bioprosthetic valve degeneration are usually old and highly comorbid, which often makes them unsuitable candidates for reoperation.[7] As a less invasive approach is needed to treat these patients, transcatheter aortic valve replacement (TAVR) has offered an excellent alternative. Since the first valve-in-valve (ViV) procedures performed in 2007,[8] a substantial worldwide experience has developed. Our objective in this review is to provide a balanced assessment of the published data and also to discuss the key complications of the procedure, while demonstrating some of the solutions that have been developed with the maturation of the technique.

### ViV patient population and procedural outcomes

The ViV patient population was initially composed of high surgical risk patients. The mean age of patients treated with ViV was often in the late 70s.[7 9 10] In the Valve-in-Valve International Data (VIVID) Registry landmark study, almost half had chronic renal failure and the rate of other comorbidities was high.[7] In a subanalysis of the PARTNER II trial, 26% were considered to be frail.[10] Most major studies on the topic have reported mean Society of Thoracic Surgeons (STS) scores greater than 9%,[7 10 11] with the exception of the TVT Registry, which still reported a relatively high mean of 6.9%.[9]

Reported ViV outcomes have been variable, but appear to have progressively improved as operator experience and technique have developed. In an early 202 patient study, 30-day mortality was 8.4%, slightly lower than the predicted surgical mortality of 11.8% by STS score.[12] Comparatively, in the PARTNER II paper published 5 years later with a similar comorbidity profile, 30-day mortality was only 2.7%.[10] Similar results were found in other recent analyses,[9 11] with particularly the TVT Registry reporting lower 30-day mortality, 1-year mortality and less heart failure hospitalisations in ViV patients versus native TAVR in adjusted analyses.[9] Finally, patients with small surgical valves and stenosis as the mechanism of failure, both of which are risk factors for elevated postprocedural gradients, have been found to have worse survival at 1-year follow-up (figure 2).[7]

Symptom improvement is an important benefit of ViV. One study reports that, after ViV, 93% of patients had improved to NYHA class I or II,[11] which translated to a significant gain in quality of life as measured by the validated Kansas City Cardiomyopathy Questionnaire. This gain persisted at both 6 months and 1-year postprocedure.[11]

### Surgical and transcatheter heart valves

The type of surgical valve treated with ViV is associated with patient outcomes. Two main groups of bioprosthetic surgical valves exist: stented and stentless surgical devices (figure 1). Stentless valves represent around 20% of ViV patients[7] and tend to present more commonly with regurgitation as the mode of degeneration, with correspondingly lower postprocedural gradients that are observed with failing stented bioprostheses.[7 13 14] However, stentless valves may be associated with higher rates of malpositioning, given their lack of visible fluoroscopic markers, as well as higher rates of coronary obstruction.

The type of transcatheter heart valve (THV) selected is also relevant. Some devices are supra-annular (ie, functional leaflets positioned above the aortic annulus), such as the CoreValve/Evolut (Medtronic, Minneapolis, Minnesota, USA), while others are intra-annular, such as the SAPIEN family



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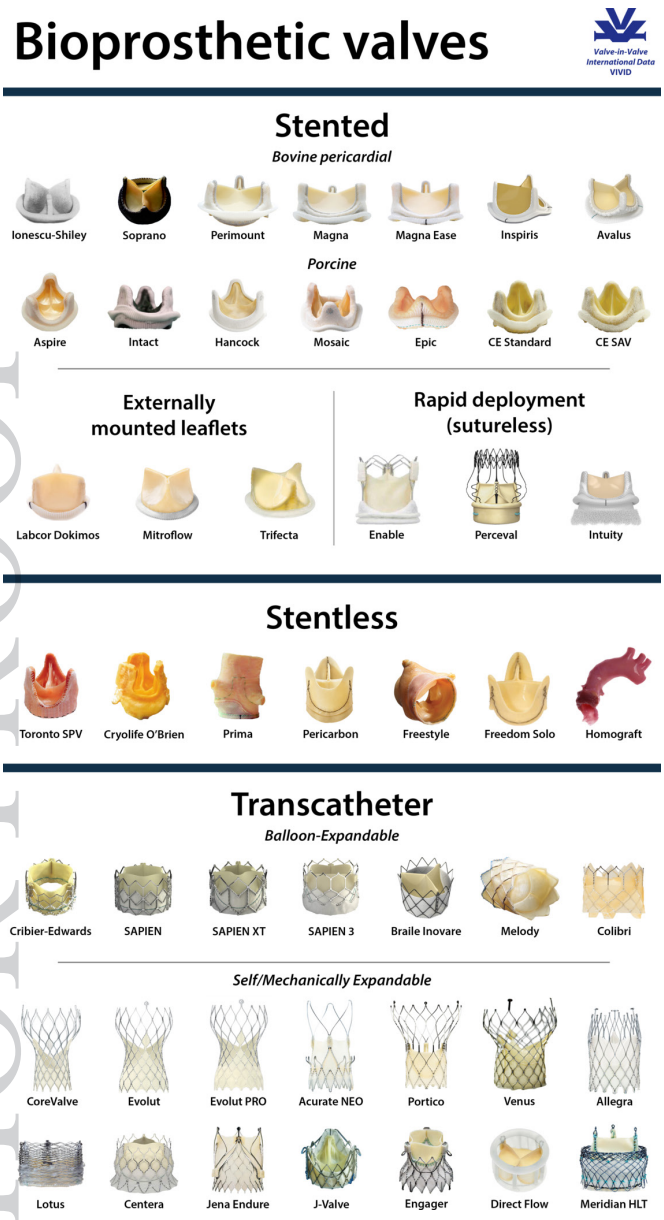


**Figure 1** Diagram demonstrates the several different types of bioprosthetic valves that can be found in ViV patients. VIV, valve-in-valve.

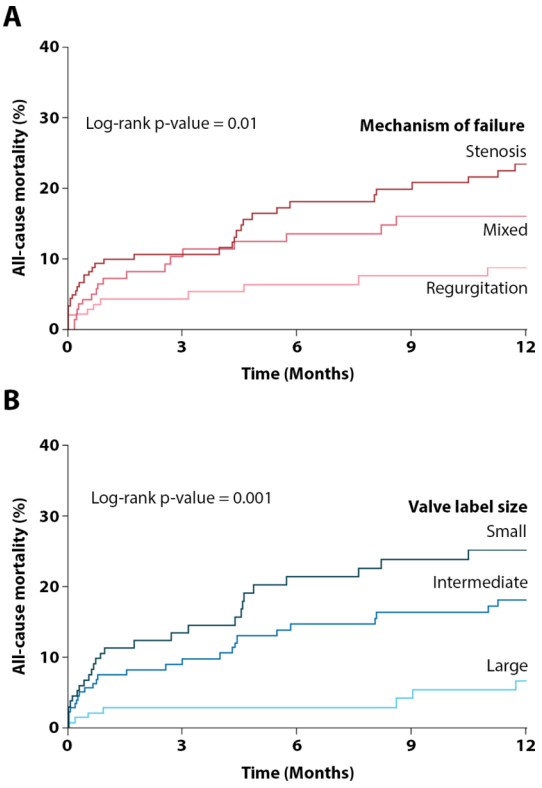


**Figure 2** Kaplan-Meier curves demonstrating mortality difference between patients with stenosis as the baseline mechanism of failure (A) and small surgical valves (B), both statistically significant. Many factors play a role in this phenomenon, such as pre-procedural prosthesis-patient mismatch and device selection. Adapted from Dvir *et al*.[7]

(Edwards Lifesciences, Irvine, California, USA) and the Portico (Abbott, Lake Bluff, Illinois, USA).

There are no randomised comparisons between transcatheter aortic ViV and surgical redo procedures or between different transcatheter devices in the context of ViV. Nevertheless, two propensity-matched comparisons between transcatheter valves have been published. One of them, with a centralised core laboratory for echocardiographic data, showed higher effective orifice area (EOA), lower mean gradients, lower incidence of moderate to severe aortic regurgitation and lower mortality in the CoreValve group when compared with the Portico valve.[15]

A second analysis, comparing 514 SAPIEN XT and SAPIEN 3 patients, was able to identify a trend towards lower 30-day mortality in SAPIEN 3 cases (0.6% vs 3.5%).[16] These may reflect increased experience with balloon-expandable devices. No difference in elevated postprocedural gradients (ie, mean gradient ≥20 mm Hg) was found between the devices. SAPIEN 3 patients required more pacemakers after ViV (6% vs 2.5%, p=0.07), likely due to the longer profile of this device in comparison to the SAPIEN XT.

## Haemodynamics: challenges and solutions

Suboptimal haemodynamics is a limitation of aortic ViV. Early in vitro analyses identified the challenge, and even inability in rare cases, of relieving pressure gradients with transcatheter devices in small and stenotic surgical valves.[17] This issue was confirmed in an analysis showing a rate of 28.4% of elevated gradients, with a significant difference favouring CoreValve cases versus SAPIEN XT (21.3% vs 40%).[12] A similar overall rate of elevated postprocedural gradients was found in a larger cohort.[7] In this group, elevated mean gradients and lower EOA were disproportionally more common in patients who had stenosis as the baseline mechanism of failure.[7] Finally, the authors found the use of balloon-expandable devices to be an independent predictor for elevated mean gradients in the same cohort.[7]

Many factors play a role in residual stenosis after ViV. We may divide them into three categories: factors related to the surgical valve patient dyad, factors related to the selection of the transcatheter device and its relation to the patient, and factors associated with device degeneration and thrombosis. As mentioned previously, prior analyses identified the associations between small surgical valves, baseline stenosis and poor haemodynamic results. At least part of this issue may stem from baseline prosthesis–patient mismatch (PPM) (ie, a device that is inadequately sized for a given patient). One major meta-analysis including 27 186 patients and 13 141 patient-years after surgical aortic valve replacement identified a statistically significant increase

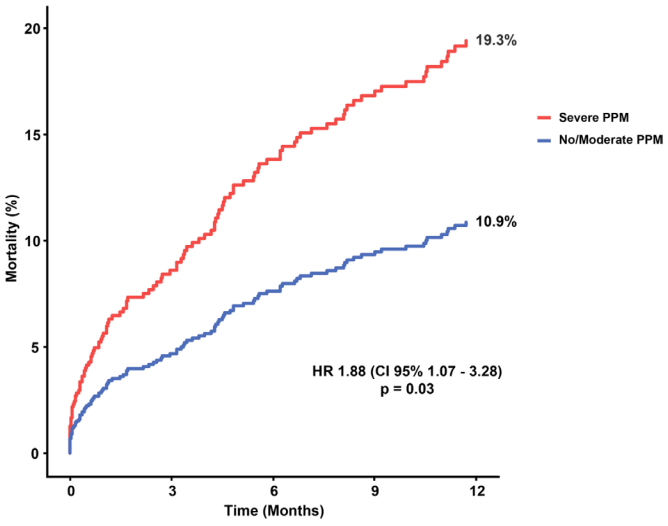


**Figure 3** Severe preprocedural prosthesis–patient mismatch (PPM) is associated with increased mortality after valve-in-valve (ViV) in a regression model controlled for multiple comorbidities and risk factors. This increased risk may be derived from long-standing increased afterload and cardiac remodelling caused by an excessively small surgical valve that is not resolved by ViV. *Extracted from* Pibarot *et al*.[19]

in all-cause and cardiac mortality in patients with moderate and severe PPM.[18] This effect was particularly pronounced in patients with severe PPM, who demonstrated an over sixfold increase in cardiovascular death.[18]

One analysis had originally identified an association between preprocedural PPM and elevated postprocedural gradients, especially in cases of severe PPM.[11] However, this did not appear to confer a mortality difference related to PPM. A more recent 1168 patient analysis specific to ViV confirmed the association of severe preprocedural PPM and elevated mean gradients.[19] All patients with severe PPM had previously received stented surgical valves. This paper also identified an independent increase in 1-year mortality in a regression controlled for label size, STS score, renal failure, diabetes and type of surgical valve (stentless vs stented) (figure 3).[19] There are several potential explanations for this mortality effect. One hypothesis is that the exposure to many years (ie, time to failure of the surgical valve) of PPM led to irreversible changes in cardiac physiology (eg, remodelling), which precipitated ventricular dysfunction and earlier death. This major long-term issue cannot be corrected by the ViV procedure, particularly given that the small rigid annulus of the surgical valve would not be expanded by the transcatheter device without additional procedures. The VIVID Registry has developed a ViV calculator, which allows operators to estimate the degree of projected PPM in their workup for ViV, giving advice on conditions with severe PPM and residual stenosis. It is available at http://valveinvalve.org/calc/.

Design differences in THVs play an important role in postprocedural gradients. Intra-annular devices have been found in several analyses to have higher gradients after ViV.[7 12] The mechanism has been elucidated through in vitro studies,[20] which demonstrate impaired expansion and leaflet movement of deeply implanted devices in simulated stenotic conditions (figure 4). Poor expansion may lead to decreased EOA and residual stenosis. One paper on postprocedural PPM demonstrated that while moderate or severe PPM was quite common after ViV (61% moderate or severe, 24.6% severe), there was no association identified with 1-year mortality or other short-term

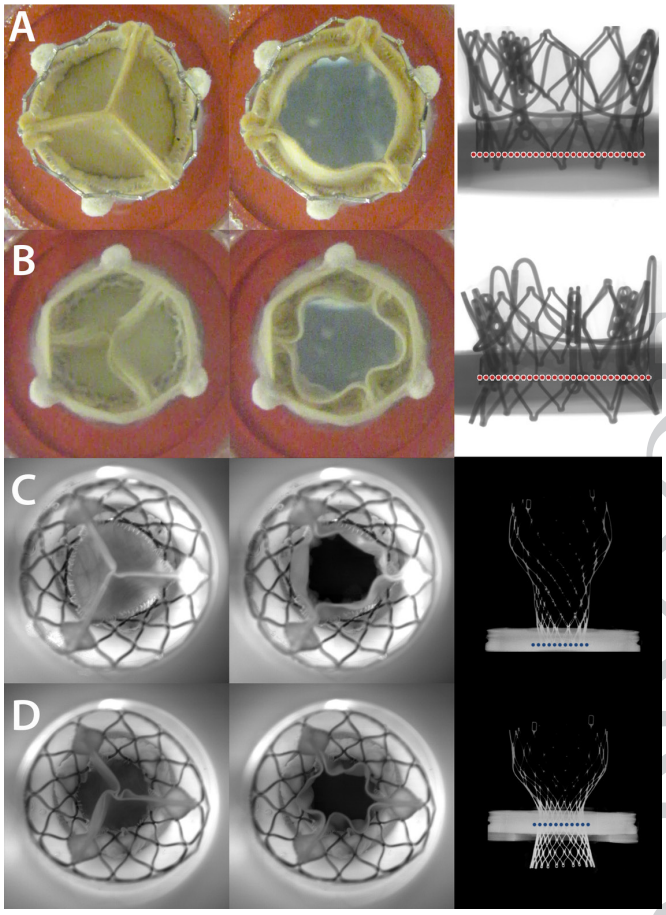


**Figure 4** In vitro testing demonstrating differences in device expansion and leaflet coaptation for the SAPIEN XT and the Evolut valves. Devices implanted high (A, C) have optimal orifice area, while those implanted deep (B, D) have incomplete opening and pin-wheeling phenomenon on leaflet closure. *Adapted from* Simonato *et al*.[20]

complications.[21] This phenomenon was more common in patients with stented surgical valves and those who received balloon-expandable devices.[21] The longer term effects on valve durability or mortality are unknown.

Finally, other issues that may be involved in the late onset of elevated gradients are transcatheter device failure and thrombosis. While transcatheter degeneration in ViV has been described,[22] it is not common at this point in time and the solution would essentially be a valvular replacement.

All of the factors mentioned above combine to create the final haemodynamic result (box 1). One example is the interaction between severe preprocedural PPM and balloon-expandable devices, with a rate of 78.3% of elevated mean gradients compared with only 33.9% in patients with similar severity of preprocedural PPM treated with self-expandable prostheses.[19]

Except for THV selection, the risk for elevated postprocedural gradients can be reduced by other operator approaches, first, valve positioning. Previous papers have determined that optimal positioning varies with different devices.[20 23 24] The reasoning behind this is that one should attempt to avoid constriction at the level of valve function. In a valve designed to be supra-annular, such as the CoreValve/Evolut, positioning too deep may lead to the device effectively functioning as an intra-annular one, with suboptimal haemodynamics. For an intra-annular device, high positioning attempts to 'supra-annular' it as much as possible. The recommended cut-offs for high positioning for

**Box 1 Correlates for elevated postprocedural gradients. All of these factors operate in conjunction to determine the final results**

**Correlates for elevated postprocedural gradients**

Before valve-in-valve
- Stenosis as the baseline mechanism of failure.
- Preprocedural severe prosthesis–patient mismatch.
- Stented surgical valve.
- Small surgical valve (internal diameter ≤20 mm).

During valve-in-valve
- Intra-annular transcatheter heart valve.
- Deep transcatheter heart valve position.
- Lack of bioprosthetic valve ring fracture.

After valve-in-valve
- Leaflet thrombosis.
- Postprocedural prosthesis–patient mismatch.
- Structural valve degeneration.

the CoreValve/Evolut and SAPIEN 3 are 5 mm and 20%, respectively. As an added note, high positioning in the SAPIEN 3 may also be associated with a decreased need for pacemakers.[24]

The second approach is the use of bioprosthetic valve fracture (BVF). While the technique was described earlier,[25 26] only recently a large case series has been published.[27] The authors reported on 20 consecutive ViV cases that were accompanied by BVF (figure 5). Five cases were performed before ViV and 15 afterwards. They reported that, in the cases of BVF performed after ViV, mean gradients were reduced (20.5±7.4 mm Hg to 6.7±3.7 mm Hg, p<0.001) with mean EOA increased (1.0±0.4 $cm^2$ to 1.8±0.6 $cm^2$, p<0.001).[27] No complications were identified in this series apart from a single transient ischaemic attack, although several theoretical risks were described as well: coronary obstruction, annular rupture, severe damage to the new transcatheter device, leaflet tearing, accelerated degeneration, dislodging of debris, among others. The technique employs non-compliant balloons such as the Atlas Gold (BARD Peripheral Vascular, Tempe, Arizona, USA) and the TRUE Balloon (BARD Peripheral Vascular) that are commonly sized 1 mm above the label size.[27] Not all surgical valve rings are amenable to cracking, but in vitro analyses have identified which devices can be cracked and what pressure in atmospheres is required.[28] The risks and benefits of BVF will be further studied and in the future clearer recommendations on how and when to use this technique will be provided.

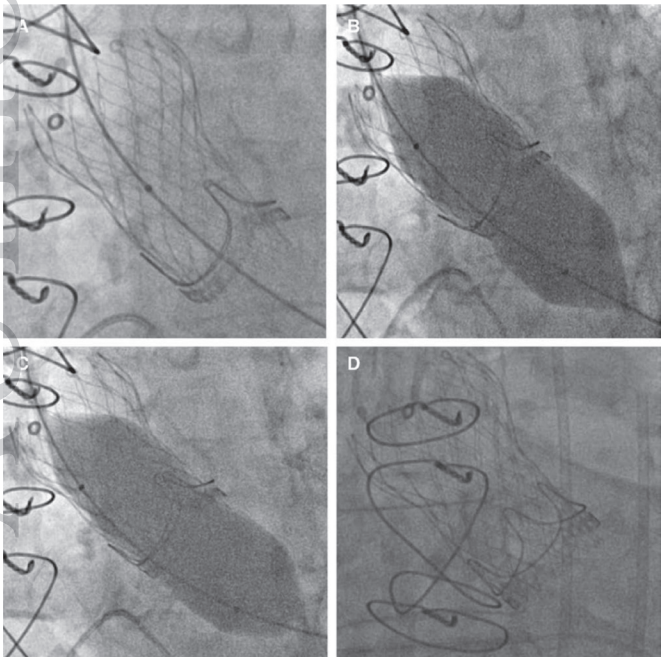


**Figure 5** Case example of bioprosthetic valve fracture. Figure 5A demonstrates a poor expansion of the self-expandable valve. Figure 5B,C shows the non-compliant balloon expanded and subsequent release of the waist. Figure 5D displays the final result, with a proper expansion of the self-expandable valve. Extracted from Chhatriwalla *et al*.[27]

Considering the role of ViV in small surgical valves, which represent almost one-third of cases,[7] operators must integrate all available data and use appropriate clinical judgement. ViV techniques offer several methods to potentially improve outcomes (eg, high positioning, BVF), however, some patients may be better served by redo surgery with annular enlargement.[29] The main factor that should be taken into consideration is patient life expectancy.

## Coronary obstruction: clinical presentation, treatment and prevention

Coronary obstruction is probably the most worrisome complication of ViV. The rate of coronary obstruction in ViV is 1%–3.5%.[9–12] The largest analysis published on the topic is a 1612 patient cohort with 37 coronary obstructions, with a total rate of 2.3%.[30] Thirty-day mortality in patients who had coronary obstruction was 48.6% vs 3.7% in unaffected patients.[30]

Most patients with coronary obstruction presented with severe hypotension and electrocardiographic changes, such as ST segment elevation.[30] Percutaneous coronary intervention (PCI) was attempted in 77.8% of patients and was successful in only 64.3% of attempts. Eighty per cent of patients with unsuccessful PCI died.[30] Thirty-six per cent of obstructions were delayed and most of these (77%) took place in patients with self-expandable devices, as these tend to continue expanding after the procedure is complete.[30] A similar phenomenon has been described in native TAVR interventions, however, the risk for this complication in non-ViV conditions is significantly lower.[31]

The most important factor identified to have an association with coronary obstruction is the failed surgical valve model. Stentless valves and stented ones with externally mounted leaflets, such as the Mitroflow (Livanova PLC, London, UK) and the Trifecta (Abbott), were the only factor independently associated with coronary obstruction in a model controlling for coronary artery bypass grafting to the left system, STS score and postdilation (OR 7.67; 95% CI 3.14 to 18.7; p<0.001).[30] It is possible that when the surgical ring is not present or when leaflets are mounted outside of the posts, the interaction between the surgical valve leaflets and the coronary ostium is more pronounced and the risk is higher.

Operators have attempted to pre-emptively position stents in the coronaries in high-risk cases and deploy them back towards the inflow of the coronary ostium. There are complications to this 'chimney technique', such as stent entrapment, and the durability of a stent deployed in such conditions seems to be suboptimal, with an associated severe challenge in reattempting coronary interventions.[32] The Vancouver group defined a measure called virtual transcatheter valve-to-coronary distance (VTC).[33] It is a computed tomography-based parameter that is calculated using the horizontal distance between the coronary

**Box 2 Correlates for coronary obstruction. Presence of risk factors should prompt operators to consider leaflet laceration. Virtual transcatheter-to-coronary distance (VTC) should be included in the workup of all valve-in-valve patients and VTCs ≤4 mm are associated with severely increased risk of obstruction**

**Correlates for coronary obstruction**

Anatomic factors
- Low lying coronary ostia.
- Narrow sinotubular junction/low sinus height.
- Narrow sinuses of Valsalva.
- Prior root repair (eg, root graft, coronary reimplantation).

Bioprosthetic valve factors
- Supra-annular position.
- High leaflet profile.
- Internal stent frame (eg, Mitroflow, Trifecta).
- No stent frame (homograft, stentless valves).
- Bulky leaflets.

Transcatheter valve factors
- Extended sealing cuff.
- High implantation.

ostium and the border of a virtual transcatheter device, simulating to where the surgical valve leaflet will be deflected. It is clear that coronary height alone is not an accurate predictor of obstruction (unless the ostium is completely above the deflected surgical valve leaflet, as in such cases there is virtually no risk for obstruction). VTC was validated in a 110 patient (20 obstruction cases, 90 controls) core laboratory adjudicated study.[30] VTC below 4 mm is a very accurate cut-off with high sensitivity and specificity identified by receiver operating characteristic analysis with an area under the curve of 0.943 ($p<0.001$).[30] All factors associated to coronary obstruction act together to determine the final risk in a given patient (box 2).

A novel transcatheter technique that may prevent coronary obstruction is the Bioprosthetic Aortic Scallop Intentional Laceration to prevent Iatrogenic Coronary Artery obstruction (BASILICA) procedure.[32] This technique employs an electrified wire to perforate surgical valve leaflets, which can be subsequently electrically lacerated (figure 6). As it is known that the surgical valve leaflets are responsible for the obstruction in most cases, the goal is that the laceration would allow blood flow to still take place.[32] Since the first procedure in July 2017, there have been many dozens of BASILICAs performed worldwide and it seems to be an effective approach.

### Leaflet thrombosis after VIV

Valve thrombosis was identified as a potential concern when reduced leaflet motion was observed by four-dimensional CT in 40% of patients in a clinical trial.[34] A more modest rate of 12%, with an association to increased risk of transient ischaemic attacks, was identified in a larger analysis.[35] However, true clinical thrombosis with poor haemodynamics is not common in native valve TAVR. This complication had not been thoroughly described in ViV patients until a more recent analysis[36] which identified 23 cases of valve thrombosis among a cohort of 300 patients (7.3%). Sixty-five per cent of patients affected presented with symptoms of heart failure and 91% had increased mean gradients. No patient had evidence of a stroke. Median mean gradient at diagnosis was 39 mm Hg, which subsequently reduced to 17.5 mm Hg ($p<0.001$) after oral anticoagulation.

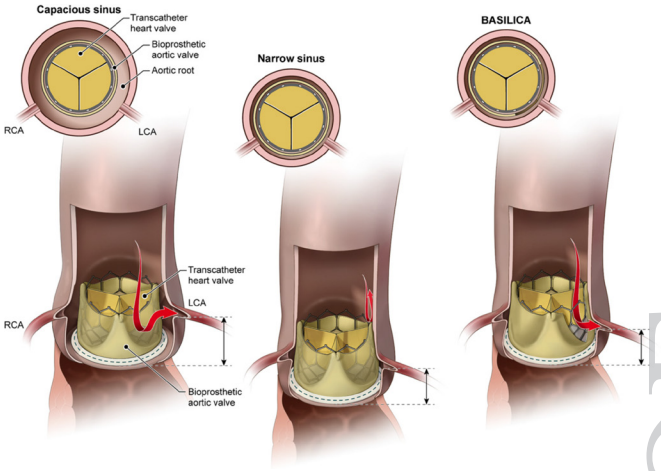


**Figure 6** Diagram demonstrating the closer relationship between the surgical valve and the coronary ostium in narrow sinuses. In such situations, the risk of coronary obstruction is severely increased. The Bioprosthetic Aortic Scallop Intentional Laceration to prevent Iatrogenic Coronary Artery obstruction procedure can be used to tear surgical valve leaflets and ensure proper blood flow to coronary vessels. *Extracted from* Khan *et al*.[32]

Of note, thrombosis was identified much more commonly in patients with Mosaic (Medtronic) and Hancock II (Medtronic) compared with other devices: 20.3% of patients with these valves discharged on no anticoagulation (all on antiplatelet therapy) developed this complication, compared with 7.2% of similarly treated patients with other valves.[36] This issue may stem from design factors.[37] These results may support a more aggressive anticoagulation strategy after VIV, particularly in patients at low risk for bleeding and high risk of events.

Currently, there is no solid randomised data on antiplatelet versus anticoagulation after ViV. The American Heart Association and the American College of Cardiology offered a focused guideline update with a class IIb recommendation for anticoagulation in TAVR for up to 3 months, given thrombosis reports.[38] There are no specific recommendations for ViV at this point. Several randomised clinical trials on this topic are ongoing. Several randomised clinical trials on this topic are ongoing. Some, such as the GALILEO trial (NCT02556203), include ViV patients and are expected to be concluded shortly. Future data will hopefully provide a better ground for decision-making in post-ViV patient population.

## CONCLUSIONS

Transcatheter ViV in the aortic position is a safe, effective and well-established procedure backed by a large body of evidence. Procedural complications, apart from those inherent to TAVR itself, are elevated postprocedural gradients, coronary obstruction and thrombosis. Risk factors for these adverse events have been described and commonly can be prevented with appropriate procedural planning, increased operator experience and novel transcatheter techniques.

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## REFERENCES


1 Brown JM, O'Brien SM, Wu C, *et al*. Isolated aortic valve replacement in north america comprising 108,687 patients in 10 years: changes in risks, valve types, and outcomes in the society of thoracic surgeons national database. *J Thorac Cardiovasc Surg* 2009;137:82–90.
2 Dvir D, Bourguignon T, Otto CM, *et al*. Standardized definition of structural valve degeneration for surgical and transcatheter bioprosthetic aortic valves. *Circulation* 2018;137:388–99.
3 David TE, Armstrong S, Maganti M. Hancock II bioprosthesis for aortic valve replacement: the gold standard of bioprosthetic valves durability? *Ann Thorac Surg* 2010;90:775–81.
4 Forcillo J, Pellerin M, Perrault LP, *et al*. Carpentier-Edwards pericardial valve in the aortic position: 25-years experience. *Ann Thorac Surg* 2013;96:486–93.
5 David TE, Feindel CM, Bos J, *et al*. Aortic valve replacement with Toronto SPV bioprosthesis: optimal patient survival but suboptimal valve durability. *J Thorac Cardiovasc Surg* 2008;135:19–24.
6 Capodanno D, Petronio AS, Prendergast B, *et al*. Standardized definitions of structural deterioration and valve failure in assessing long-term durability of transcatheter and surgical aortic bioprosthetic valves: a consensus statement from the European Association of Percutaneous Cardiovascular Interventions (EAPCI) endorsed by the European Society of Cardiology (ESC) and the European Association for Cardio-Thoracic Surgery (EACTS). *Eur J Cardiothorac Surg* 2017;52:408–17.
7 Dvir D, Webb JG, Bleiziffer S, *et al*. Transcatheter aortic valve implantation in failed bioprosthetic surgical valves. *JAMA* 2014;312:162–70.
8 Wenaweser P, Buellesfeld L, Gerckens U, *et al*. Percutaneous aortic valve replacement for severe aortic regurgitation in degenerated bioprosthesis: the first valve in valve procedure using the Corevalve Revalving system. *Catheter Cardiovasc Interv* 2007;70:760–4.
9 Tuzcu EM, Kapadia SR, Vemulapalli S, *et al*. Transcatheter aortic valve replacement of failed surgically implanted bioprostheses: The STS/ACC Registry. *J Am Coll Cardiol* 2018;72:370–82.
10 Webb JG, Mack MJ, White JM, *et al*. Transcatheter Aortic Valve Implantation Within Degenerated Aortic Surgical Bioprostheses: PARTNER 2 Valve-in-Valve Registry. *J Am Coll Cardiol* 2017;69:2253–62.
11 Deeb GM, Chetcuti SJ, Reardon MJ, *et al*. 1-Year Results in Patients Undergoing Transcatheter Aortic Valve Replacement With Failed Surgical Bioprostheses. *JACC Cardiovasc Interv* 2017;10:1034–44.
12 Dvir D, Webb J, Brecker S, *et al*. Transcatheter aortic valve replacement for degenerative bioprosthetic surgical valves: results from the global valve-in-valve registry. *Circulation* 2012;126:2335–44.
13 Cohen G, Zagorski B, Christakis GT, *et al*. Are stentless valves hemodynamically superior to stented valves? Long-term follow-up of a randomized trial comparing Carpentier-Edwards pericardial valve with the Toronto Stentless Porcine Valve. *J Thorac Cardiovasc Surg* 2010;139:848–59.
14 Borger MA, Carson SM, Ivanov J, *et al*. Stentless aortic valves are hemodynamically superior to stented valves during mid-term follow-up: a large retrospective study. *Ann Thorac Surg* 2005;80:2180–5.
15 Alnasser S, Cheema AN, Simonato M, *et al*. Matched Comparison of Self-Expanding Transcatheter Heart Valves for the Treatment of Failed Aortic Surgical Bioprosthesis. *Circulation* 2017;10.
16 Seiffert M, Treede H, Schofer J, *et al*. Matched comparison of next- and early-generation balloon-expandable transcatheter heart valve implantations in failed surgical aortic bioprostheses. *EuroIntervention* 2018;14:e397–e404.
17 Azadani AN, Jaussaud N, Matthews PB, *et al*. Transcatheter aortic valves inadequately relieve stenosis in small degenerated bioprostheses. *Interact Cardiovasc Thorac Surg* 2010;11:70–7.
18 Head SJ, Mokhles MM, Osnabrugge RL, *et al*. The impact of prosthesis-patient mismatch on long-term survival after aortic valve replacement: a systematic review and meta-analysis of 34 observational studies comprising 27 186 patients with 133 141 patient-years. *Eur Heart J* 2012;33:1518–29.
19 Pibarot P, Simonato M, Barbanti M, *et al*. Impact of pre-existing prosthesis-patient mismatch on survival following aortic valve-in-valve procedures. *JACC Cardiovasc Interv* 2018;11:133–41.
20 Simonato M, Azadani AN, Webb J, *et al*. In vitro evaluation of implantation depth in valve-in-valve using different transcatheter heart valves. *EuroIntervention* 2016;12:909–17.
21 Bleiziffer S, Erlebach M, Simonato M, *et al*. Incidence, predictors and clinical outcomes of residual stenosis after aortic valve-in-valve. *Heart* 2018;104:828–34.
22 Schaefer A, Deuschl F, Conradi L, *et al*. Valve-in-valve-in-valve: balloon expandable transcatheter heart valve in failing self-expandable transcatheter heart valve in deteriorated surgical bioprosthesis. *Catheter Cardiovasc Interv* 2018;10.
23 Simonato M, Webb J, Kornowski R, *et al*. Transcatheter replacement of failed bioprosthetic valves: large multicenter assessment of the effect of implantation depth on hemodynamics after aortic valve-in-valve. *Circ Cardiovasc Interv* 2016;9.
24 Simonato M, Webb J, Seiffert M, *et al*. TCT-375 Current generation balloon-expandable transcatheter valve positioning during aortic valve-in-valve procedures and clinical outcomes. *J Am Coll Cardiol* 2018;72:B152.
25 Tanase D, Grohmann J, Schubert S, *et al*. Cracking the ring of Edwards Perimount bioprosthesis with ultrahigh pressure balloons prior to transcatheter valve in valve implantation. *Int J Cardiol* 2014;176:1048–9.
26 Nielsen-Kudsk JE, Christiansen EH, Terkelsen CJ, *et al*. Fracturing the Ring of Small Mitroflow Bioprostheses by High-Pressure Balloon Predilatation in Transcatheter Aortic Valve-in-Valve Implantation. *Circ Cardiovasc Interv* 2015;8:e002667.
27 Chhatriwalla AK, Allen KB, Saxon JT, *et al*. Bioprosthetic valve fracture improves the hemodynamic results of valve-in-valve transcatheter aortic valve replacement. *Circ Cardiovasc Interv* 2017;10:e005216.
28 Allen KB, Chhatriwalla AK, Cohen DJ, *et al*. Bioprosthetic Valve Fracture to Facilitate Transcatheter Valve-in-Valve Implantation. *Ann Thorac Surg* 2017;104:1501–8.
29 Okamoto Y, Yamamoto K, Sugimoto T, *et al*. Early and late outcomes of AVR with aortic annular enlargement in octogenarian. *Gen Thorac Cardiovasc Surg* 2015;63:453–8.
30 Ribeiro HB, Rodés-Cabau J, Blanke P, *et al*. Incidence, predictors, and clinical outcomes of coronary obstruction following transcatheter aortic valve replacement for degenerative bioprosthetic surgical valves: insights from the VIVID registry. *Eur Heart J* 2018;39:687–95.
31 Jabbour RJ, Tanaka A, Finkelstein A, *et al*. Delayed Coronary Obstruction After Transcatheter Aortic Valve Replacement. *J Am Coll Cardiol* 2018;71:1513–24.
32 Khan JM, Dvir D, Greenbaum AB, *et al*. Transcatheter laceration of aortic leaflets to prevent coronary obstruction during transcatheter aortic valve replacement: concept to first-in-human. *JACC Cardiovasc Interv* 2018;11:677–89.
33 Blanke P, Soon J, Dvir D, *et al*. Computed tomography assessment for transcatheter aortic valve in valve implantation: the vancouver approach to predict anatomical risk for coronary obstruction and other considerations. *J Cardiovasc Comput Tomogr* 2016;10:491–9.
34 Makkar RR, Fontana G, Jilaihawi H, *et al*. Possible subclinical leaflet thrombosis in bioprosthetic aortic valves. *N Engl J Med* 2015;373:2015–24.
35 Chakravarty T, Søndergaard L, Friedman J, *et al*. Subclinical leaflet thrombosis in surgical and transcatheter bioprosthetic aortic valves: an observational study. *The Lancet* 2017;389:2383–92.
36 Abdel-Wahab M, Simonato M, Latib A, *et al*. Clinical valve thrombosis after transcatheter aortic valve-in-valve implantation. *Circ Cardiovasc Interv* 2018.
37 Brown ML, Park SJ, Sundt TM, *et al*. Early thrombosis risk in patients with biologic valves in the aortic position. *J Thorac Cardiovasc Surg* 2012;144:108–11.
38 Nishimura RA, Otto CM, Bonow RO, *et al*. 2017 AHA/ACC Focused Update of the 2014 AHA/ACC Guideline for the management of patients with valvular heart disease. *J Am Coll Cardiol* 2017;70:252–89.