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STATE OF THE ART REVIEW
Valvular heart disease

Which patients with aortic stenosis should be referred to surgery rather than transcatheter aortic valve implantation?

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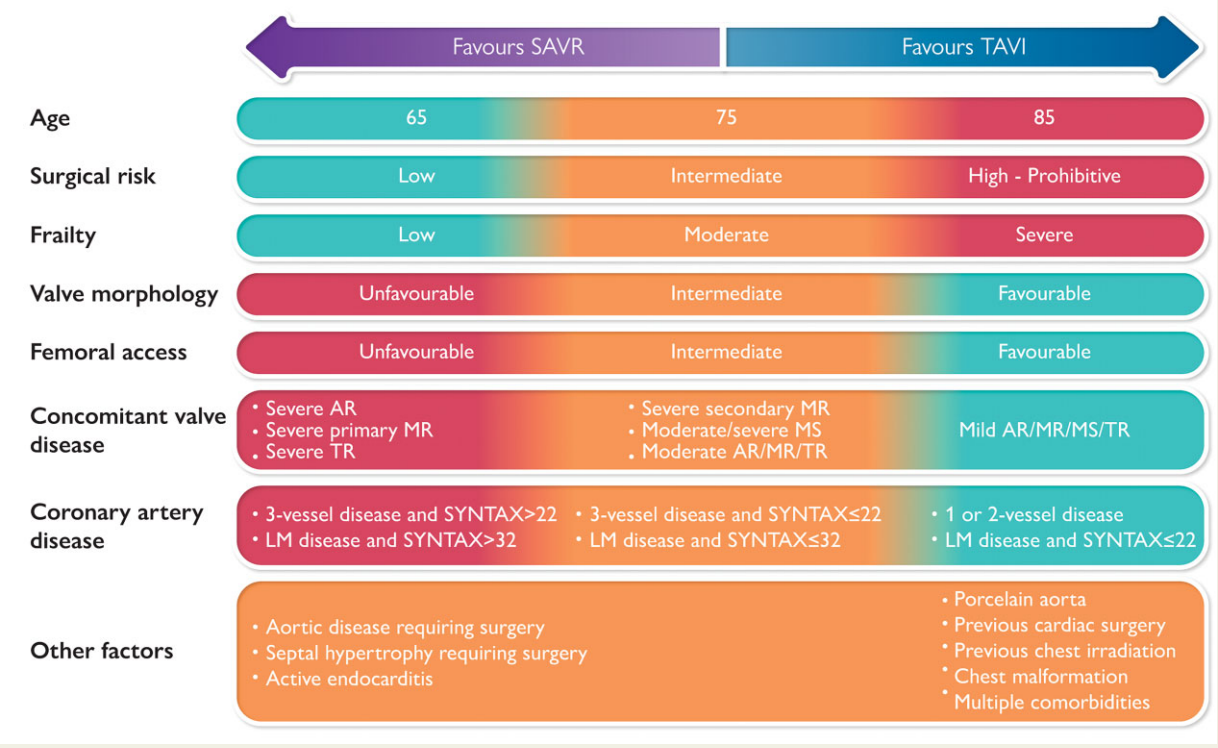

Graphical Abstract Decision-making process between TAVI and SAVR. Refer to *Figures 2*, *4*, and *6* for details of the valve morphology category. Refer to *Figure 3* for details on the femoral access category. Refer to *Figure 7* for more details on concomitant valve disease. Refer to *Figure 8* for more details on coronary artery disease. AR, aortic regurgitation; MR, mitral regurgitation; TR, tricuspid regurgitation; MS, mitral stenosis; LM, left main; SAVR, surgical aortic valve replacement; TAVI, transcatheter aortic valve implantation.

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Abstract

Transcatheter aortic valve implantation (TAVI) has matured into a standard treatment option for patients with severe symptomatic aortic valve stenosis (AS) across the whole spectrum of risk. The advances in the interventional treatment of AS raise the question of which patients with severe AS should be referred to surgery. The myriad of clinical permutations does not allow providing a single, uniform treatment strategy. Rather, the advent of TAVI along with established surgical aortic valve replacement (SAVR) fundamentally enforces the role of the multidisciplinary heart team for decision-making recommending the best individual choice of the two options based on a thorough review of clinical and anatomical factors as well as lifetime management considerations. Involvement of the informed patient expressing treatment preferences is a key for a shared decision-making process. Herein, we provide an in-depth review of evidence informing the decision-making process between TAVI and SAVR and key elements for treatment selection. Special attention is given to the populations that have been excluded from randomized clinical trials, and also lifetime management strategies of patients with severe AS are proposed.

Keywords Transcatheter aortic valve implantation • Surgical aortic valve replacement • Lifetime management

Introduction

Transcatheter aortic valve implantation (TAVI) has been directly compared with surgical aortic valve replacement (SAVR) in a series of randomized clinical trials across the entire spectrum of surgical risk.[1–7] Across these trials, TAVI has consistently been associated with clinical outcomes better or comparable to SAVR in terms of all-cause death and stroke throughout longest available follow-up (*Figure 1*, see Supplementary material online, *Table S1*).[8–11] In a meta-analysis including seven landmark trials, TAVI was associated with a modest reduction in all-cause death and stroke throughout 2 years irrespective of surgical risk and type of transcatheter heart valve (THV) system, a difference that was apparent in patients allocated to transfemoral TAVI.[12] These excellent outcomes, albeit still mid-term, have led to a paradigm shift in the management of patients with severe aortic valve stenosis (AS) by establishing a less-invasive treatment that allows for more rapid recovery while providing similar clinical benefits as the previous gold standard SAVR.

As a result, current European and US guidelines for the management of valvular heart disease consider transfemoral TAVI and SAVR both *Class I* recommendations for the majority of patients with severe, symptomatic AS (*Table 1*).[13,14] The decision is usually made by local heart teams taking into consideration multiple and complex clinical and anatomical factors (*Graphical Abstract*).

The available body of evidence supporting TAVI compared with SAVR was obtained in carefully selected patient populations, whereas the relative safety and efficacy remain less well defined in populations excluded from the landmark trials (*Table 2*).[1–7] Moreover, there remain uncertainties related to long-term durability with both TAVI and SAVR and the optimal lifetime management of patients with severe AS. In this article, we will discuss which patients with severe AS should be referred to surgery following the recent update of guideline recommendations. We focus on the factors that increase the risk or lead to suboptimal outcomes with TAVI as well as patient populations that have been excluded from the randomized trials, and we propose potential strategies for the lifetime management of patients with severe AS. As outlined in the current guidelines on valvular heart disease, these patients require a comprehensive patient-centred evaluation based on the sound clinical assessment as well as careful integrated imaging with transthoracic echocardiography providing the most important baseline qualitative and quantitative information supplemented by transesophageal echocardiography, cardiac computed tomography (CT), cardiac magnetic resonance, and stress testing as needed and final synthesis in terms of treatment allocation by the heart team.[13,14]

Anatomical risk stratification

Anatomical risk stratification based on the device implantation zone as well as vascular access is one of the central considerations in patient selection for either procedure (*Figures 2* and *3*).[13,14] Under circumstances where the aortic valve anatomy is favourable for TAVI and transfemoral access is feasible, successful implantation of a THV will result in clinical outcomes comparable to SAVR as evidenced in the randomized trials.[1–7] Conversely, in patients with unfavourable anatomy of the device implantation zone for TAVI or inadequate femoral access, procedural and device success of TAVI is diminished, and SAVR remains the treatment of choice. Notwithstanding, there remains a grey zone of intermediate-risk scenarios that require careful individual decision-making.

Severely calcified aortic valve/left ventricular outflow tract calcification

An excessive amount of asymmetrically distributed calcium or extension into the left ventricular outflow tract (LVOT) increases the risk for adverse procedural events including significant paravalvular regurgitation (PVR), annular rupture, conduction disturbances, coronary obstruction, and stroke (*Figure 2*).[15–19] Patients with bulky eccentrically calcified aortic valve leaflets or extensive LVOT calcification were excluded from the randomized trials.[1–7] Although the impact of aortic valve leaflet calcification on PVR has been largely mitigated owing to improvement in THV designs, such as a circumferential outer sealing skirt and repositionable features in some self-expanding devices, the adverse impact of LVOT calcification on PVR and annular rupture remains considerable even with newer-generation devices.[19] Accordingly, SAVR should be preferred in patients with excessive calcification in the device implantation zone, as the calcified leaflets can be safely resected and any calcium extension into the annulus and LVOT can be completely debrided.

Figure 1 Short- and long-term outcomes of major randomized clinical trials. The results of PARTNER 1A, PARTNER 2A, PARTNER 2B, PARTNER 3, SURTAVI, and PARTNER 3 are provided from intention-to-treat analyses. The results of U.S. CoreValve High risk, NOTION, and Evolut Low Risk are provided from as-treated analyses. #1: Results are provided with differences (transcatheter aortic valve implantation–surgical aortic valve replacement) and 95% Bayesian credible interval. #2: Results are provided with hazard ratios and 95% confidence intervals. #3: Any stoke.

Risk of conduction disturbances

Anatomical risk stratification for new conduction disturbances is an emerging field based on electrocardiographic and pre-procedural CT evaluation; calcium deposition adjacent to the conduction system, the length of membranous septum, the implantation depth, and computer-simulated contact pressure by a THV have been identified as risk modifiers for new conduction disturbances following TAVI (*Figure 2*).[20–23] Although the risk of new conduction disturbances remains an important limitation related to TAVI, especially with some self-expanding devices, new implantation techniques aiming at a higher device implantation appear effective to reduce this risk.[20,24–26] It remains uncertain whether the choice of SAVR may further reduce the risk of new conduction disturbances leading to permanent pacemaker implantation compared with TAVI, especially with high implantation techniques. Notwithstanding, the risk of new conduction disturbances is an important consideration for treatment selection in patients at high risk for conduction disturbances, and SAVR emerges as the preferred option particularly in young patients with long life expectancy.

Extreme annulus dimensions

Appropriate THV sizing is essential to achieve optimal device performance without adverse events.[27] Therefore, extreme aortic annulus dimensions (both large and small), that do not allow for optimal THV sizing, should be preferably treated by SAVR that offers more treatment options including aortic root enlargement/replacement and stentless valve implantation (*Figure 2*). Although haemodynamic performance of THVs is similar or superior to SAVR bioprostheses,[1–7] data in patients with annulus dimensions smaller than the recommended range are lacking. Transcatheter heart valve selection along with surgical options is particularly important in patients with small annulus; supra-annular self-expanding vs. balloon-expandable THV design may result in differential outcomes,[28] a concept which is currently evaluated in the randomized SMART trial (NCT04722250). For patients with annulus dimensions beyond the recommended range, a recent multicentre observational study suggested that TAVI implantation using a 29-mm SAPIEN 3 THV (Edwards Lifesciences, USA) using overexpansion was safe and effective up to 1 year with acceptable rates of PVR and new permanent pacemaker implantation.[29,30] A newer-generation balloon-expandable valve (Myval, Meril Life Sciences, India)[31] offers two additional sizes (30.5 and 32.5 mm), covering larger areas up to 840 mm^2.

Non-calcified aortic valve

Non-calcified aortic valve morphology, which is commonly seen in younger patients with rheumatic AS or pure native aortic valve regurgitation, has been considered a risk for valve embolization or dislocation following TAVI due to lack of calcification anchoring the THV (*Figure 2*). Although some dedicated devices for the anatomy are in pre-clinical and clinical evaluation,[32,33] the data on TAVI in

Table 1 Guideline recommendations: choice of surgical aortic valve replacement vs. transcatheter aortic valve implantation for whom a bioprosthesis is appropriate

Recommendations	TAVI		SAVR	
	Classa	Levelb	Classa	Levelb
2020 ACC/AHA Guideline for the Management of Valvular Heart Disease				
Symptomatic and asymptomatic patients with severe AS and any indication for AVR who are <65 years of age or have a life expectancy over 20 years			I	A
Symptomatic patients with severe AS who are 65–80 years of age and have no anatomical contraindication to transfemoral TAVI	I	A	I	A
Symptomatic patients with severe AS who are >80 years of age or younger patients with a life expectancy <10 years and no anatomic contraindication to transfemoral TAVI	I	A	IIa	A
Asymptomatic patients with severe AS and an LVEF <50% who are 65–80 years of age and have no anatomic contraindication to transfemoral TAVI	I	B-NR	I	B-NR
Asymptomatic patients with severe AS and an abnormal exercise test, very severe AS, rapid progression, or an elevated BNP			I	B-NR
Patients with an indication for AVR but valve or vascular anatomy or other factors are not suitable for transfemoral TAVI			I	A
Symptomatic patients of any age with severe AS and a high or prohibitive surgical risk (estimated life expectancy >12 months)	I	A		
2021 ESC/EACTS Guidelines for the Management of Valvular Heart Disease				
Younger (<75 years) patients who are low risk for surgery (STS-PROM/EuroSCORE II <4%), or patients who are operable and unsuitable for transfemoral TAVI			I	B
Older (≥75 years) patients, or in those who are high risk (STS-PROM/EuroSCORE II >8%) or unsuitable for surgery	I	A		
Remaining patients according to individual clinical, anatomical, and procedural characteristics	I	B	I	B

SAVR, surgical aortic valve replacement; TAVI, transcatheter aortic valve implantation; ACC, American College of Cardiology; AHA, American Heart Association; AVR, aortic valve replacement; AS, aortic valve stenosis; LVEF, left ventricular ejection fraction; BNP, brain natriuretic peptide; ESC, European Society of Cardiology; EACTS, European Association for Cardio-thoracic Surgery; STS-PROM, Society of Thoracic Surgeons Predicted Risk of Mortality.
[a]Class of recommendation.
[b]Level of evidence.

this population remain limited to small observational studies.[34–36] Therefore, unless the patient is at prohibitive surgical risk, SAVR with or without concomitant aortic root replacement should remain the preferred approach.

Low take-off of coronary ostium with shallow sinus of Valsalva

Low take-off of coronary ostia in association with shallow sinus of Valsalva confers an increased risk of coronary obstruction following TAVI, a rare but life-threatening complication (*Figure 2*).[37,38] Although preventive strategies such as coronary protection[39] and the bioprosthetic or native aortic scallop intentional laceration to prevent iatrogenic coronary artery obstruction (BASILICA) procedure[40] may mitigate these risks, they have not been established in routine clinical practice to date, and SAVR should be preferred when the risk of coronary obstruction is high.

Horizontal aorta

Aortic angulation, defined as the angle between the horizontal plane and the plane of the aortic annulus in a coronal projection is an important anatomical consideration for TAVI, particularly with some self-expanding devices without steerable delivery systems (*Figure 2*). Thus, patients with aortic angulation >70° were excluded from randomized trials of self-expanding devices.[2,4,7] Although the impact of the horizontal aorta on procedural success has been studied in observational studies, none of the studies adequately included patients with severe aortic angulation.[41,42] Aortic angulation >70°, albeit rare, remains a limitation for TAVI, and SAVR should be considered for these patients.

Poor femoral/peripheral access

Vascular access is another important anatomical consideration for the choice between TAVI and SAVR (*Figure 3*). The recent low-risk trials enrolled patients with good femoral access who were candidates for transfemoral TAVI,[6,7] and available evidence for non-transfemoral TAVI has been limited to observational studies and subgroup analyses in early randomized trials.[1,3,8,11,43] Although some preparation techniques such as percutaneous peripheral angioplasty (for stenosis), intravascular lithotripsy (severe calcific stenosis), use of parallel stiff wire (for tortuosity), and

Table 2 Key exclusion criteria in major randomized clinical trials (transcatheter aortic valve implantation vs. surgical aortic valve replacement)

Anatomical criteria	Clinical criteria
• Aortic annulus dimension unsuitable for TAVI devices • Unicuspid or bicuspid aortic valve anatomy • Bulky calcified aortic valve leaflets • Prohibitive left ventricular outflow tract calcification • Non-calcified aortic valve (balloon-expandable TAVI) • Small sinus of Valsalva (self-expanding TAVI) • Aortic root angulation >70° (self-expanding TAVI) • Pre-existing mechanical or bioprosthetic valve in any position • Porcelain aorta • Unfavourable femoral access	• Mixed valve disease (aortic regurgitation, mitral regurgitation, mitral stenosis, or tricuspid regurgitation) • Complex coronary artery disease (multivessel disease or left main disease) • Left ventricular dysfunction (LVEF <20%) • Intracardiac mass, thrombus, or vegetation • Hypertrophic obstructive cardiomyopathy • Significant aortopathy requiring ascending aortic replacement • Blood dyscrasias • Haemodynamic instability • Known hypersensitivity or contraindication to antithrombotic therapies • Active gastrointestinal bleeding • Recent acute myocardial infarction • Recent cerebrovascular accident • Severe comorbidities (renal insufficiency, lung disease, liver disease) • Severe pulmonary hypertension • Short estimated life expectancy (<12–24 months)

TAVI, transcatheter aortic valve implantation; LVEF, left ventricular ejection fraction.

surgical cut-down (for severely diseased puncture site) may facilitate transfemoral TAVI in selected patients with unfavourable iliofemoral access, the data are limited to small case series or case reports.[44–46] Accordingly, SAVR should remain the treatment of choice for these patients unless they are considered at increased surgical risk.

Valve-in-valve transcatheter aortic valve implantation feasibility

Patients with a failed surgical bioprosthesis were formally excluded from randomized clinical trials (*Table 1*).[1–7] However, due to the high risk of redo surgery in elderly patients, valve-in-valve TAVI is now widely performed and has been extensively investigated in terms of safety and effectiveness in several dedicated registries.[47–51] Although observational studies attest favourable short- and mid-term outcomes up to 3 years for valve-in-valve TAVI, these studies also revealed some important limitations related to the procedure (*Figure 4*).[52,53]

Valve-in-valve TAVI has been associated with higher risks of coronary obstruction, higher residual gradients, and more device malposition/dislocation compared with native aortic valve TAVI depending on the type of the implanted surgical bioprosthesis. In general, the risk of coronary obstruction with valve-in-valve TAVI is higher in externally mounted stented, stentless, and internally mounted stented surgical bioprostheses in this order.[54] In patients with a stented bioprosthesis, it is recommended to measure the virtual THV to coronary distance (VTC) if the surgical valve stent posts extend to or above the level of the coronary ostia. If VTC distances are <3–4 mm, the risk of coronary obstruction is deemed high.[55,56] In patients with a stentless surgical bioprosthesis, the risk is determined by the coronary artery height and sinus of Valsalva width as in native aortic valve TAVI, but it is also crucial to know the surgical implantation technique of the bioprosthesis. The use of a subcoronary surgical approach for stentless valves, as opposed to full root replacement, during initial SAVR has been associated with an increased risk of coronary obstruction (*Figure 5*).[57] In such cases and when redo SAVR is not a feasible alternative, TAVI with preventive measures such as BASILICA[40] should be considered. Surgical aortic valve replacement can also be complex in patients with calcified homograft where valve-in-valve TAVI if feasible may be preferred; implanting a THV under direct vision during a surgical procedure is an alternative option.

The risk of high residual gradients and prosthesis–patient mismatch is particularly increased when performing valve-in-valve TAVI for small surgical bioprostheses. Use of supra-annular devices and bioprosthetic valve fracture are means to achieve an appropriate low gradient[28,58]; however, redo SAVR with dedicated techniques for aortic root enlargement or replacement (Bentall procedure) should be considered as alternative.[59]

In general, the presence of significant PVR should not be primarily treated with valve-in-valve TAVI. In the case of valvular regurgitation as determined by transthoracic echocardiography, it is critical to assess whether the jet is transvalvular, paravalvular, or both, which mandates a careful assessment by transoesophageal echocardiography. Although valve-in-valve TAVI may effectively treat PVR in some selected cases of surgical bioprosthetic valve degeneration with the sealing cuff of a THV expanded below the bioprosthesis, the data are currently limited to few cases.[60,61] Current guidelines allocate a *Class I* recommendation for redo SAVR among operable patients with clinically relevant PVR and a *Class IIa* recommendation for transcatheter paravalvular leak closure among inoperable or high surgical risk patients.[13,14] We consider paravalvular leak closure as treatment of choice for PVR as long as technically feasible in terms of size, shape, and number of defect(s) and in the absence of infective endocarditis as it can resolve regurgitation with high success rate obviating the need and risks associated with redo SAVR.[62]

Finally, data on valve-in-valve TAVI for certain bioprostheses such as sutureless bioprostheses and degenerated THVs are scarce. Although observational studies suggest comparable short- and mid-term outcomes of valve-in-valve TAVI for failed THVs and surgical bioprostheses,[63,64] the investigation needs to be extended to a larger number of patients and longer follow-up to evaluate the safety and effectiveness of the procedure thoroughly. Efforts at conducting randomized trials of valve-in-valve TAVI compared with redo SAVR

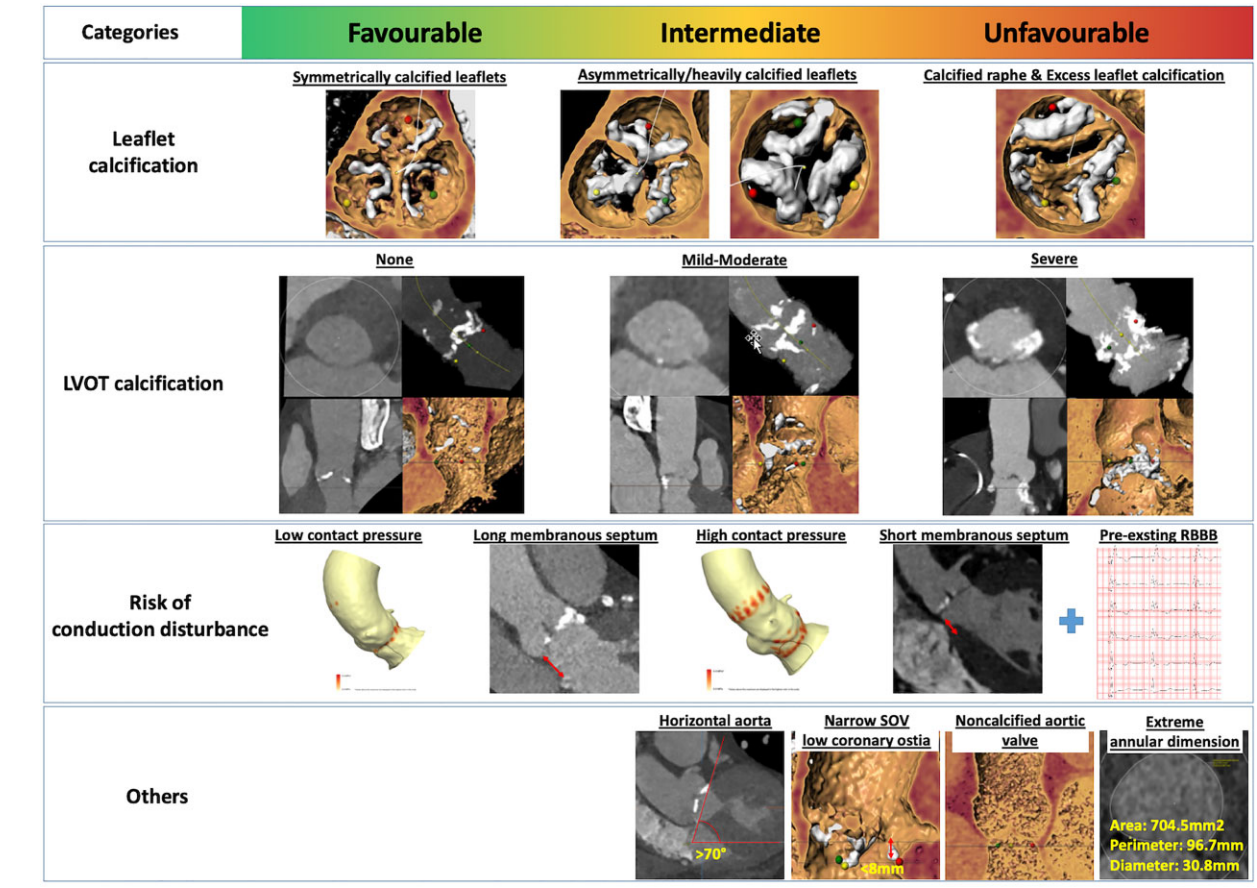

Figure 2 Anatomical risk stratification of native aortic valve morphology. The category (favourable, intermediate, unfavourable) indicates the suitability for transcatheter aortic valve implantation. RBBB, right bundle branch block; LVOT, left ventricular outflow tract; SOV, sinus of Valsalva.

have not been successful over the years but may become necessary in the future if valve-in-valve TAVI durability becomes an issue.

Bicuspid aortic stenosis

Bicuspid aortic valve accounts for ~5–10% of elderly patients currently treated by TAVI,[65–68] which has been systematically excluded from the landmark randomized trials.[1–7] The major concerns related to bicuspid AS include typical morphological features with the presence of a calcified raphe and extent/location of bulky, eccentric calcification as well as associated aortopathy. Aortic enlargement at the level of the sinuses and/or proximal ascending aorta is present in 20–80% of adult patients with the bicuspid aortic valve, and some develop greater degrees of aneurysmal dilation (≥45 mm) with an increased risk of aortic dissection or rupture, necessitating prophylactic replacement of the ascending aorta.[68,69] Surgical aortic valve replacement with/without concomitant aortic root replacement in patients with bicuspid AS has been associated with low in-hospital mortality ranging from 0.7 to 2.4% and a high 10-year survival rate of over 80%.[70–74]

Although the safety and efficacy of TAVI compared with SAVR for bicuspid AS have not been established in randomized trials,[1–7] a number of observational studies with a large number of highly selected patients with bicuspid AS undergoing TAVI have reported favourable outcomes comparable to tricuspid AS treated with TAVI or bicuspid AS treated with SAVR (*Table 3*).[65–67,75,76] In analyses in the STS/ACC TVT registry, TAVI in bicuspid AS was associated with comparable haemodynamics but with a slightly higher rate of moderate or severe PVR compared with TAVI in tricuspid AS even with the use of contemporary devices.[65–67,75] In the most recent propensity score-matched analysis from the STS/ACC TVT registry that included only low-risk patients treated with the SAPIEN 3/Ultra THV, there was no significant difference in the rate of 30-day stroke or procedural complications, as well as 30-day and 1-year mortality.[75] Finally, in a propensity score-matched comparison between TAVI and SAVR in patients with bicuspid AS from the National Inpatient Sample database, there were no significant differences in in-hospital outcomes including mortality, stroke, and other complications.[76]

A categorization of bicuspid morphology according to the presence of calcified raphe and/or excess leaflet calcification may have important clinical implications for decision-making between TAVI and SAVR[84,85] (*Figure 6*). In a recent multicentre study, patients with both calcified raphe and excess leaflet calcification undergoing

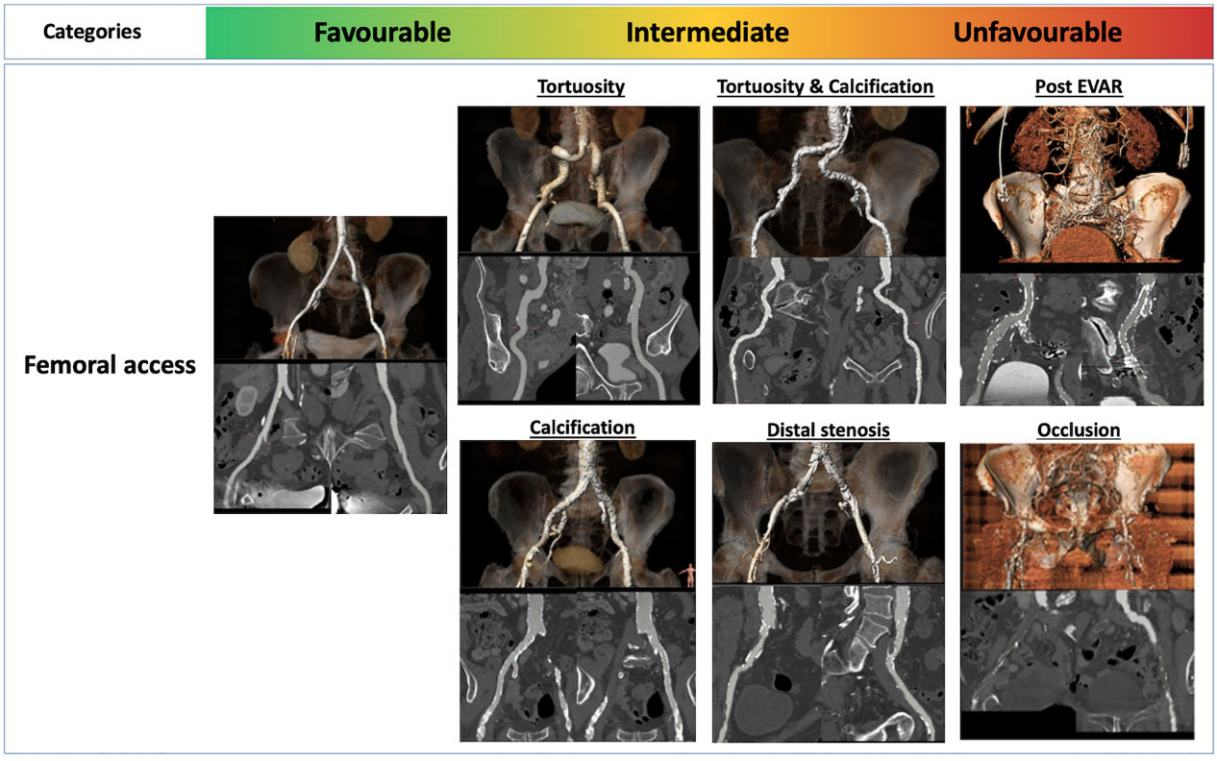

Figure 3 Anatomical risk stratification of femoral access. The category (favourable, intermediate, unfavourable) indicates the suitability for transfemoral transcatheter aortic valve implantation. EVAR, endovascular aortic repair.

TAVI had an excess risk of aortic root injury, moderate or severe PVR, 30-day and 2-year mortality compared with patients that had only one or none of these morphological features.[85] It should be noted, however, that Sievers type 0 bicuspid aortic valve is uncommon (<10%) and underrepresented in the above-mentioned studies.[65–67,76,85] Transcatheter aortic valve implantation with contemporary devices appears to be safe and effective for elderly patients with bicuspid AS; however, SAVR should remain the primary treatment option for bicuspid AS in young patients and independent of age when the bicuspid aortic valve morphology is unfavourable or significant aortopathy coexists.

Mixed valve disease

The presence of moderate-to-severe mitral regurgitation (MR) or stenosis (MS) and moderate-to-severe tricuspid regurgitation (TR), is observed in up to 30% of patients evaluated for the treatment of severe symptomatic AS (*Table 4*). Patients with mixed valve disease present a particular diagnostic challenge in assessing the true severity of the different valvular lesions and with regard to the optimal timing and sequence of the intervention. Transoesophageal echocardiography plays a crucial role both to assess the severity of individual valvular lesions as well as to determine the optimal strategy based on morphologic features of the valves. Patients with mixed valve disease should therefore be referred to a comprehensive valve centre (*Figure 7*).[119]

Mitral regurgitation

Relevant MR is the most common coexisting valvular lesion in patients with severe AS.[119] Mitral regurgitation severity is often more severe due to increased left ventricular (LV) pressure in patients with AS, and secondary MR may improve in up to 50% of cases after aortic valve intervention. The persistence of moderate or severe MR confers an increased risk of mortality in patients undergoing TAVI or SAVR (*Table 4*).[95,120]

The treatment strategy differs between patients with severe AS and primary MR and those with secondary MR. The first-line therapy for primary MR is surgical mitral valve repair while transcatheter edge-to-edge mitral valve repair may be considered for patients who are at high or prohibitive surgical risk. Concomitant surgery of the aortic and mitral valve should therefore be performed for patients with severe AS and severe primary MR whenever possible[13,14] (*Figure 7*). The decision is more challenging in patients with secondary MR, which is usually a disease of the ventricle rather than the mitral valve and may regress after resolution of AS.[121] According to the current guidelines, TAVI with staged transcatheter edge-to-edge repair is reasonable for patients with severe AS and persistent secondary MR after TAVI. Patients undergoing SAVR should undergo concomitant repair/replacement of the mitral valve in case of severe secondary MR[13,14] (*Figure 7*). In the case of moderate concomitant MR, the decision becomes further complicated. Transcatheter aortic valve implantation followed by re-evaluation of MR and transcatheter edge-to-edge repair if needed as well as SAVR combined

Categories	Favourable	Intermediate	Unfavourable
Stented Valve			
Coronary Obstruction/access	✓ Level of coronary ostia > stent post ✓ Level of coronary ostia < stent post and VTC >6 mm	✓ Internally mounted ✓ Intra-annular design ✓ Level of coronary ostia < stent post and VTC: 3 to 6 mm	✓ Externally mounted ✓ Supra-annular design ✓ Level of coronary ostia <stent post and VTC <3mm
Residual Gradients		✓ Externally mounted ✓ Small Valve (<23mm)	✓ Internally mounted ✓ Extremely small valve (<21mm) ✓ BVF not possible (Trifecta and Hancock II)
Other Considerations:	Paravalvular regurgitation between the sewing ring and the aortic annulus may remain after valve-in-valve TAVI.		
Stentless Valve			
Coronary Obstruction/access	✓ High coronary ostia and large Sinus of Valsalva	✓ Full root replacement ✓ Low coronary ostia or Small Sinus of Valsalva	✓ Subcoronary implant technique ✓ Low coronary ostia and Small Sinus of Valsalva
Residual Gradients		✓ Small Valve (<23mm)	✓ Extremely small valve (<21mm)
Other Considerations:	Risk of valve embolization/dislocation is high due to 1) difficulty of implantation due to radiolucent Dacron ring and 2) the absence of adequate calcification.		
Sutureless/TAVI Valve			
Coronary Obstruction/access	✓ Data/experience is limited; however, coronary access may frequently be impaired due to 1) overlay of two stent frames and 2) tissue tunnel created by the pushed-up degenerated leaflets.		
Residual Gradients			✓ Limited experience/evidence
Other Considerations:	Data/experience are limited to small numbers.		

Figure 4 Anatomical risk stratification of valve-in-valve transcatheter aortic valve implantation. The category (favourable, intermediate, unfavourable) indicates the suitability for transcatheter aortic valve implantation. Refer to the Clinical Atlas of Transcatheter Aortic Valve Therapies (Valve in Valve Aortic app: https://apps.apple.com/us/app/valve-in-valve/id655683780) for more information needed in planning of and performing valve-in-valve transcatheter aortic valve implantation. VTC, virtual transcatheter heart valve to coronary distance.

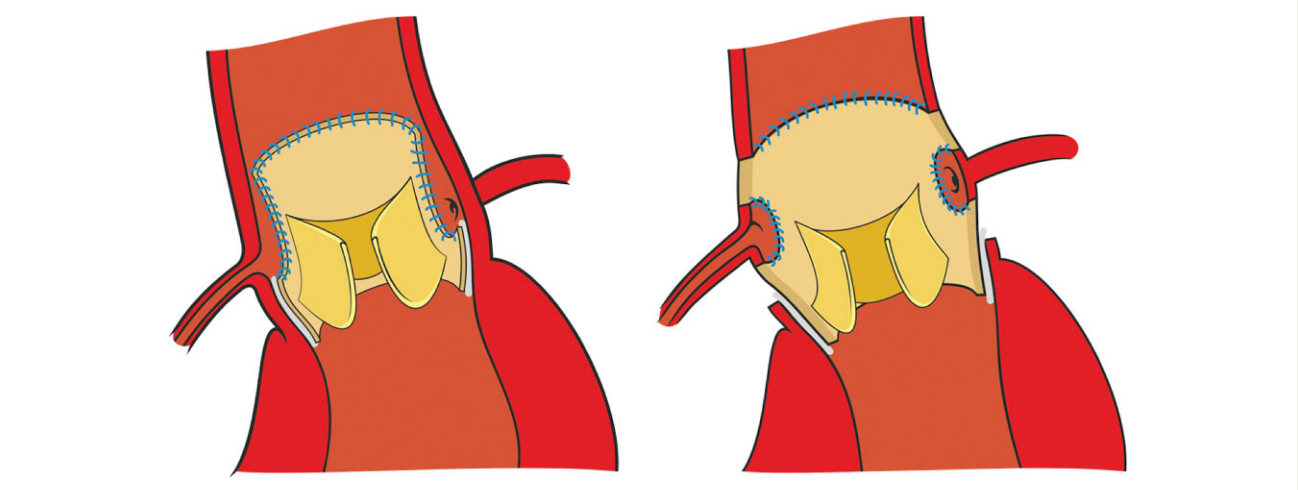

Figure 5 Implantation techniques for a stentless bioprosthesis. The subcoronary stentless valve (left) and the full root stentless valve (right) are shown.

with repair/replacement of the mitral valve are reasonable options as persistent MR may worsen the prognosis after aortic valve intervention.[120] Finally, isolated mitral valve repair/replacement especially by a minimally invasive approach is an option after successful TAVI.

Mitral stenosis

Concomitant MS requiring intervention is less common (<5%) in patients with severe AS, and may be due to rheumatic or degenerative aetiology.[108] Concomitant MS was associated with an increased risk

Table 3 Evidence summary: transcatheter aortic valve implantation (vs. surgical aortic valve replacement) in bicuspid aortic valve stenosis

Bicuspid vs. tricuspid	Valve type	*N* (bicuspid)	Echocardiographic outcome						30-day outcome					
			EOA (cm^2)		mPG (mmHg)		PVR (≥moderate)		Mortality		Stroke		Permanent pacemaker	
			Bicuspid	Tricuspid	Bicuspid	Tricuspid	Bicuspid	Tricuspid	Bicuspid	Tricuspid	Bicuspid	Tricuspid	Bicuspid	Tricuspid
STS/ACC TVT registry PS-matched study Makkar *et al.*[75]	SAPIEN 3/Ultra	3243	1.9 ± 0.6	1.8 ± 0.6	12.1 ± 5.5	12.6 ± 5.2	0.9%	0.3%	0.9%	0.8%	1.4%	1.2%		
			0.063 [0.032,0.095][a]		−0.4 [−0.7,−0.1][a]		0.6% [0.2,1.1][a]		*P* = 0.55		*P* = 0.55			
STS/ACC TVT registry Halim *et al.*[65]	SAPIEN XT/SAPIEN3/ CoreValve/Evolut R	5412	1.8 (1.4–2.2)	1.8 (1.5–2.2)	10 (7–14)	9 (7–12)	4.4%	3.2%	2.0%	2.2%	2.2%	1.9%		
			P = 0.473		*P* < 0.001		*P* < 0.001		*P* = 0.484		*P* = 0.151			
STS/ACC TVT registry PS-matched study Forrest *et al.*[67]	Evolut R/PRO	929	1.7 (1.4–2.1)	1.8 (1.4–2.1)	11 (8–14)	11 (7–13)	2.6%	2.0%	2.6%	1.7%	3.4%	2.7%	15.4%	13.7%
			P = 0.938		*P* < 0.001		*P* = 0.007		*P* = 0.63		*P* = 0.93		*P* = 0.52	
STS/ACC TVT registry PS-matched study Makkar *et al.*[66]	SAPIEN 3	2691	1.8 ± 0.6	1.8 ± 0.5	11.6 ± 5.7	11.8 ± 5.3	1.5%	0.8%	1.7%[b]	1.6%[b]	2.1%	1.2%	7.3%	5.9%
			P = 0.34		*P* = 0.15		*P* = 0.04		*P* = 0.75		*P* = 0.01		*P* = 0.05	
PS-matched study Elbadawi *et al.*[76]	NA	1035							2.9%[b]	3.4%[b]	1.9%[c]	1.9%[c]	14.0%	12.1%
									P = 0.762		*P* > 0.999		*P* = 0.506	
PS-matched study Nagaraja *et al.*[77]	NA	359							5.57%[b]	1.39%[b]	2.79%[d]	5.57%[d]	1.14%	4.18%
									P = 0.21		*P* = 0.42		*P* = 0.13	
Single centre Liao *et al.*[78]	CoreValve/Venus-A	87							9.2%	4.3%	1.1%	0%	24.1%	28.6%
									P = 0.35		*P* = 1.0		*P* = 0.53	
Two centres Sannino *et al.*[79]	BEV/SEV	88	2.15 ± 0.55	1.90 ± 0.54	7.96 ± 4.15	8.5 ± 4.2	5.3%	5.0%	3.4%	3.1%	2.3%	3.7%	22.7%	18.2%
			P = 0.007		*P* = 0.268		*P* = 0.903		*P* = 0.887		*P* = 0.499		*P* = 0.303	
PS-matched study Kochman *et al.*[80]	SAPIEN/CoreValve	28	1.54 ± 0.3	1.61 ± 0.2	11.5 ± 6.4	10.4 ± 4.5	32%[e]	23%[e]	4%	7%	0%cve	4%	29%	33%
			P = 0.22		*P* = 0.33		*P* = 0.45		*P* = 0.68		*P* = 0.57		*P* = 0.82	
Two centres Costopoulos *et al.*[81]	SAPIEN/CoreValve	21			10.3 ± 5.7	10.5 ± 4.7	0%	3%	14%	4%	0%[c]	1%[c]	14%	15%
					P = 0.28		*P* = 0.47		*P* = 0.02		*P* = 0.63		*P* = 0.93	
German Registry Bauer *et al.*[82]	SAPIEN/CoreValve	38			5.5 ± 7.1	5.9 ± 6.8	25%[e]	15%[e]	11%	11%	0%	3%	17%	35%
					NA		*P* = 0.05		NA		NA		*P* = 0.02	
Single centre Hayashida *et al.*[83]	SAPIEN/CoreValve	21			10.0 ± 3.4	9.7 ± 4.1	19.0%[e]	14.9%[e]	4.8%	8.2%	0%	0.5% ≤72 h	14.3%	7.2%

Continued

Table 3 *Continued*

Bicuspid vs. tricuspid	Valve type	N (bicuspid)	Echocardiographic outcome						30-day outcome					
			EOA (cm^2)		mPG (mmHg)		PVR (≥moderate)		Mortality		Stroke		Permanent pacemaker	
			Bicuspid	Tricuspid	Bicuspid	Tricuspid	Bicuspid	Tricuspid	Bicuspid	Tricuspid	Bicuspid	Tricuspid	Bicuspid	Tricuspid
					$P = 0.58$		$P = 0.54$		$P = 1.00$		$P = 1.00$		$P = 0.22$	
TAVI vs. SAVR	Valve type	N (TAVI)	TAVI	SAVR	TAVI	SAVR	TAVI	SAVR	TAVI	SAVR	TAVI	SAVR	TAVI	SAVR
PS-matched study Elbadawi *et al.*[76]	NA	975							3.1%	3.1%	2.1%[c]	2.6%[c]	13.8%	4.6%
									$P > 0.999$		$P = 0.547$		$P < 0.001$	

TAVI, transcatheter aortic valve implantation; SAVR, surgical aortic valve replacement; AS, aortic valve stenosis; EOA, effective orifice area; mPG, mean pressure gradient; PVR, paravalvular regurgitation; PS, propensity score.
[a]Difference and 95% confidence interval.
[b]In-hospital mortality.
[c]Acute stroke.
[d]Cerebrovascular accident including transient ischaemic attack.
[e]Aortic insufficiency ≥2 (0–4).

of mortality after TAVI in multiple observational studies[104,108] (*Table 4*). In this entity, the treatment strategy is largely dependent on mitral valve morphology and the presence of concomitant MR. As percutaneous mitral commissurotomy is recommended (*Class I*) for patients with isolated MS and favourable mitral valve morphology, TAVI followed by staged percutaneous mitral commissurotomy is a reasonable strategy for such patients. However, most patients with severe AS and MS have an unfavourable anatomy with heavily calcified annulus and leaflets,[122,123] thus a concomitant surgical replacement of the aortic and mitral valve is indicated.[13,14] In cases of high or prohibitive surgical risk and unfavourable valve morphology such as severe mitral annular calcification, TAVI followed by transcatheter mitral valve replacement is an option in experienced heart valve centres[124,125] (*Figure 7*).

Tricuspid regurgitation

Tricuspid regurgitation is predominantly a consequence of left-sided heart disease. While TR may improve in some patients after aortic valve intervention, a considerable proportion of patients have persistent TR or experience progression of TR resulting in an impaired prognosis (*Table 4*).[110,119] Given the difficulty to predict the response of TR to aortic valve intervention and in view of the high perioperative mortality of reoperation for severe TR after left-sided valve surgery, current guidelines support the addition of tricuspid valve surgery when performing SAVR among patients with severe TR (Class I) or among patients with moderate TR in the presence of a dilated annulus (≥40 mm) (Class IIa).[13,14] Surgical aortic valve replacement with concomitant tricuspid valve surgery should be the primary treatment option in patients with severe AS who are at low surgical risk. If patients are deemed surgical high risk or inoperable, staged transcatheter tricuspid valve intervention for persistent or worsening TR after TAVI can be considered[126,127] (*Figure 7*). Isolated tricuspid valve surgery by a minimally invasive approach after successful TAVI may be an alternative option in selected patients.

Coronary artery disease

Concomitant coronary artery disease (CAD) is present in 30–70%, depending on the definitions (*Table 5*). Although the prognostic importance of concomitant CAD and the need for revascularization in these patients remain to be determined, combined coronary artery bypass graft (CABG) or percutaneous coronary intervention (PCI) are recommended or considered reasonable for significant CAD.[13,14]

Patients with complex CAD, defined as (i) unprotected left main disease and/or SYNTAX score >32 or (ii) unprotected left main disease and/or multivessel disease with SYNTAX score of >22, were excluded from low and intermediate surgical risk trials.[3,4,6,7] These exclusion criteria are reflected in current guidelines on myocardial revascularization allocating a *Class III* recommendation for PCI in this setting.[151,152] For these patients, SAVR combined with CABG should be the primary treatment option unless the surgical risk is high or prohibitive. Since CABG is the preferred option (*Class I*) compared with PCI (*Classes IIa–IIb*) in patients with the left main disease with intermediate SYNTAX score (23–32) and in patients with diabetes and three-vessel disease and low SYNTAX score (0–22), a concomitant surgical strategy (SAVR + CABG) should be favoured if the surgical risk is

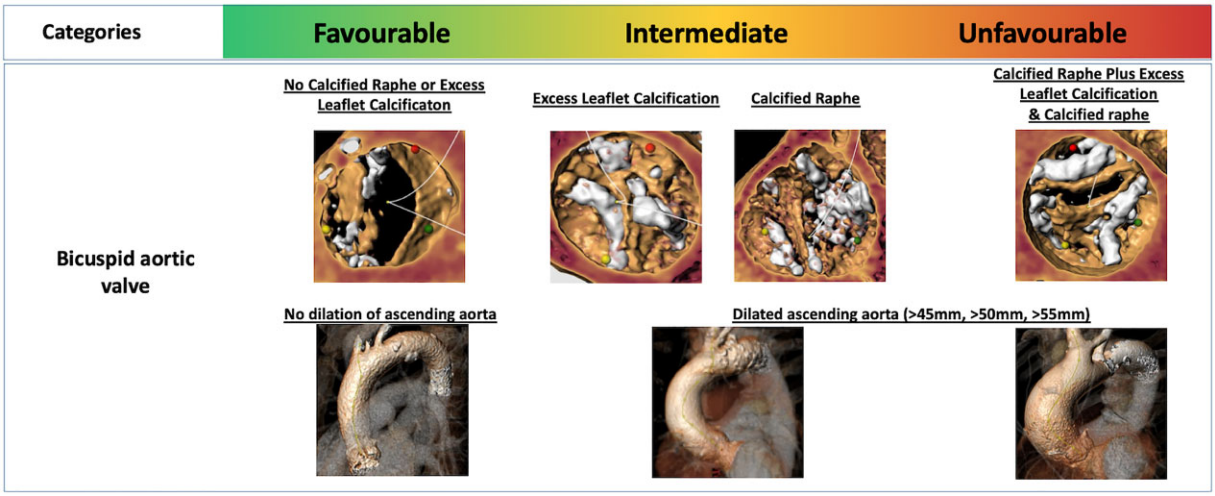

Figure 6 Anatomical risk stratification of bicuspid aortic valve. The category (favourable, intermediate, unfavourable) indicates the suitability for transfemoral transcatheter aortic valve implantation.

deemed low.[151,152] A percutaneous strategy (TAVI + PCI) is a reasonable treatment option in patients with complex CAD at high/prohibitive surgical risk (*Figure 8*). In a recent multicentre study including 800 patients with severe AS and complex CAD, TAVI + PCI was associated with a comparable rate of mid-term major adverse cardiac and cerebrovascular events but a higher rate of repeat revascularization than SAVR + CABG after propensity-score matching.[153]

Coronary access after TAVI is another important issue, which significantly affects decision-making between TAVI and SAVR in patients with or at risk of CAD. Unlike SAVR, where appropriate commissural alignment is easily accomplished and the native valve leaflets are resected, coronary access after TAVI may be complicated by lack of commissural alignment, displaced native calcified aortic valve leaflets, and the stent frame of a THV particularly with the high stent frame of self-expanding prostheses.[154,155] Coronary access may even be more challenging after valve-in-valve procedures due to the tissue tunnel created by the displaced degenerated leaflets and the overlay of two stent frames, particularly in cases of TAVI-in-TAVI.[156] Recently, a patient- and valve-specific TAVI implantation technique aiming to obtain commissural alignment has been developed to facilitate coronary access and potentiate performing BASILICA at the time of valve-in-valve TAVI later in life.[157–159] However, the technique remains immature and limited to self-expanding devices.

Lifetime management

As TAVI is expanding to younger and low-risk patients with longer life expectancy, it becomes increasingly important to anticipate lifetime management looking beyond the first 10–15 years after the index procedure and prospectively considering subsequent aortic valve replacement strategies. As comparative data between TAVI and SAVR are limited to 5–8 years,[8–11,160] there remain uncertainties as it relates to the long-term consequences of limitations such as mild PVR, conduction disturbances, CAD and coronary access, and subclinical leaflet thrombosis. In the PARTNER 2 trial, landmark analysis beyond 2 years and up to 5 years showed a higher rate of all-cause death and disabling stroke among patients allocated to TAVI as compared with SAVR.[11] Notwithstanding that the analysis comprised all patients including those undergoing transapical TAVI and refers to the SAPIEN XT prosthetic valve prosthesis, the possible adverse long-term impact of higher rates of PVR, prosthetic valve dysfunction, and more frequent valve re-intervention among patients allocated to TAVI deserves careful long-term analysis. Various treatment strategies can be considered depending on the patient's life expectancy (*Figure 9*); however, as there is no robust evidence supporting any of the strategies, it is important to regularly update available evidence and recognize the uncertainties that exist for both TAVI and SAVR.[161]

Remaining uncertainties

Iterative improvement of THV systems and sophisticated sizing algorithms based on routine CT angiography of the aortic annulus have significantly mitigated the risk of moderate or severe PVR after TAVI, which now approaches that of SAVR. However, even in the recent low-risk trials with the use of contemporary THV systems, rates of mild PVR after TAVI remain substantially higher than after SAVR.[6,7] The clinical relevance of mild PVR remains a matter of concern, and there is a lack of data especially in younger and low-risk patients.[162–164]

Similarly, new conduction disturbances, including left bundle branch block (LBBB) and high degree atrioventricular block, occur more frequently after TAVI than SAVR, particularly with some self-expanding devices.[1–7] Although there is conflicting evidence on the clinical impact of new conduction disturbances, a recent meta-analysis reported an increased risk of all-cause death and heart failure rehospitalization at 1 year in patients who had new LBBB or permanent pacemaker implantation after TAVI.[165]

Table 4 Evidence summary: prevalence and importance of concomitant valvular heart disease in patients undergoing transcatheter aortic valve implantation

Study	*N*	VHD	Prevalence	Change[a]	Importance of concomitant valvular heart disease
Mitral regurgitation (MR)					
Witberg *et al.*[86]	7303	≥Moderate MR	27%	Regression: 44%	Four-year mortality was higher for those with MR persistence, but not for those with MR regression after TAVI, compared with non-significant baseline MR (43.8 vs. 35.1% vs. 32.4%; adjusted HR 1.38; $P = 0.008$, and adjusted HR 1.02; $P = 0.383$, respectively).
Winter *et al.*[87]	429	Severe secondary MR	13%	Regression: 59%	Persistence of severe secondary MR was associated with an increased risk of mortality after TAVI (adjusted HR 2.44, 95% CI 1.15–5.20, $P = 0.021$).
Miura *et al.*[88]	1587	≥Moderate MR	9%	Regression: 77% (at 6-month)	Baseline ≥moderate MR was associated with an increased risk of mortality at 2 years (adjusted HR 1.64; 95% CI 1.15–2.34; $P = 0.007$).
Mauri *et al.*[88]	677	≥3+ MR	15%	Regression: 50%	Baseline MR ≥3+ was associated with an increased risk of mortality at 2 years (74.4 vs. 57.7%; $P < 0.001$).
Ben-Assa *et al.*[89]	486	≥Moderate MR	21%	Regression: 25%	Post-TAVI ≥moderate MR was associated with an increased risk of mortality and combined cardia events (mortality, heart failure rehospitalization, and new-onset atrial fibrillation) at median follow-up of 4 years ($P = 0.013$, $P < 0.001$, respectively).
Feldt *et al.*[90]	1712	≥Moderate MR	18%	Regression: 51%	Baseline ≥moderate MR was associated with an increased risk of 5-year mortality (adjusted HR 1.29; 95% CI 1.01–1.65; $P = 0.04$). This risk is offset if MR improved to ≤mild, whereas worsening of MR after TAVI was associated with a two-fold increased risk of mortality.
Abdelghani *et al.*[91]	970	≥Moderate MR	29%	Regression: 60%	Post-TAVI severe MR was associated with an increased risk of 5-year composite endpoint (cardiovascular mortality or heart failure rehospitalization) (adjusted HR 4.84; 95% CI 2.49–9.38).
Kindya *et al.*[92]	270	≥Moderate degenerative MR	20%	Regression: 42%	Baseline ≥moderate degenerative MR but not secondary MR was associated with an increased risk of composite endpoint of mortality and HF rehospitalization ($P = 0.011$).
		≥Moderate secondary MR	20%		
Vollenbroich *et al.*[93]	603	≥Moderate degenerative MR	16%	Regression: 59% (at 1 year)	Baseline ≥moderate degenerative MR but not secondary MR was associated with an increased risk of 2-year mortality (adjusted HR 2.21; 95% CI 1.4–3.49; $P = 0.001$, and adjusted HR 1.13; 95% CI 0.59–2.18; $P = 0.707$, respectively).
		≥Moderate secondary MR	9%	Regression: 56% (at 1 year)	
Mavromatis *et al.*[94]	11 104	Moderate MR	31%	Regression: 66%	Baseline moderate and severe MR were associated with an increased risk of 1-year mortality or HF rehospitalization (adjusted HR 1.16; 95% CI 0.99–1.35, and adjusted HR 1.21; 95% CI 0.97–1.50, respectively). Patients with MR regression had lower mortality ($P = 0.022$) and HF rehospitalization ($P < 0.001$) than those with persistent MR.
		Severe MR	6%	Regression: 79%	
Cortés *et al.*[95]	1110	≥3 (significant moderate) MR	16%	Regression: 58%	Baseline ≥3 MR was associated with an increased risk of 6-month mortality after TAVI (35.0 vs. 10.2%; $P < 0.001$)
Kirammijyan *et al.*[96]	589	≥Moderate MR	12%	Regression: 63%	Baseline ≥moderate MR was not associated with 1-year mortality (22.1 vs. 20.0%; $P = 0.331$).
Khawaja *et al.*[97]	316	≥3 MR	19%	Regression: 53%	Baseline ≥3 MR was associated with an increased risk of 1-year mortality (28.3 vs. 20.2%; $P = 0.024$).
Hutter *et al.*[98]	268	≥Moderate MR	22%	Regression: 68% (at 1 year)	Baseline ≥moderate MR was not associated with 1-year mortality (30.2 vs. 21.2%; $P = 0.068$).
Barbanti *et al.*[99]	331	≥Moderate MR	20%	Regression: 58%	Baseline ≥moderate MR was not associated with 2-year mortality (37.0 vs. 32.7% $P = 0.58$).
Bedogni *et al.*[100]	1007	Moderate MR	24%	Regression: 35% (at 1 year)	Baseline moderate and severe MR were associated with an increased risk of 1-year mortality (HR 1.4; 95% CI 1.2–2.2; $P = 0.03$, HR 1.7; 95% CI 1.2–3.4; $P < 0.001$, respectively).

Continued

Table 4 *Continued*

Study	N	VHD	Prevalence	Change[a]	Importance of concomitant valvular heart disease
		Severe MR	9%	Regression: 47% (at 1 year)	
Toggweiler *et al.*[101]	451	Moderate MR	20%	Regression: 62%	Baseline ≥moderate MR was associated with an increased risk of 30-day mortality (adjusted HR 2.10; 95% CI 1.12–3.94; $P = 0.02$) but not with 1-year mortality (adjusted HR 0.82; 95% CI 0.50–1.34; $P = 0.42$).
		Severe MR	10%	Regression: 60%	
D'Onofrio *et al.*[102]	176	≥2+ (moderate) MR	24%	Regression: 19%	Baseline MR was not identified as a risk factor for mortality.
Mitral stenosis (MS)					
Okai *et al.*[103]	156	≥Moderate MS	10%		Baseline MS was associated with acute decompensated heart failure within 72 h after TAVI (adjusted OR 14.3; 95% CI 2.7–86.7; $P = 0.002$).
Kato *et al.*[104b]	546	MS (mean gradient ≥4 mmHg)		MVA increased in 55%	MVA remained ≤2.0 cm^2 after AVR in 51%, which was associated with an increased risk of mortality during the median follow-up of 3 years (adjusted HR 1.9; 95% CI 1.2–2.9; $P < 0.01$).
Fischer *et al.*[105]	2113	≥Moderate MS	7%		Baseline MS was not associated with mortality at mean follow-up of 3 ± 2 years (35 vs. 36.2%; adjusted HR 1.16; 95% CI 0.81–1.67).
Al-Khadra *et al.*[106]	62 110	MS (ICD-9 codes)	1%		Baseline MS was associated with an increased risk of in-hospital mortality (5.1 vs. 3.5%; adjusted OR 1.46; 95% CI 1.06–2.00; $P = 0.021$) and complications after TAVI.
Sannino *et al.*[107]	928	Moderate MS	16%		Baseline severe MS (but not moderate MS) was associated with an increased risk of 5-year mortality (adjusted HR 2.91; 95% CI 1.17–7.20; $P = 0.02$).
		Severe MS	2%		
Asami *et al.*[108]	971	<Moderate MS	15%		Baseline MS (any) was associated with an increased risk of cardiovascular mortality (21.4 vs. 8.7%; adjusted HR 3.64, 95% CI 2.38–5.56).
		≥Moderate MS	3%		
Joseph *et al.*[109]	44 755	Non-severe MS	9%		Baseline severe MS (but not non-severe MS) was associated with an increased risk of 1-year mortality (adjusted HR 1.2; 95% CI 1.0–1.4) and HF rehospitalization (adjusted HR 1.3; 95% CI 1.1–1.5).
		Severe MS	3%		
Tricuspid regurgitation (TR)					
Tomii *et al.*[110]	2008	Moderate TR	12%	Regression: 55%	Post-TAVI, but not baseline, severe and massive TR were associated with an increased risk of 1-year mortality (vs. ≤mild TR: adjusted HR 1.90; 95% CI 1.03–3.49; $P = 0.039$, adjusted HR 2.17; 95% CI 1.10–4.30; $P = 0.026$, respectively).
		Severe TR	3%	Regression: 74%	
		Massive TR	3%	Regression: 72%	
Granot *et al.*[111]	3733	≥Moderate TR	13%		Baseline ≥moderate TR was not associated with mortality during mean follow-up of 3 years when adjusted to multiple echocardiographic parameters (adjusted HR 1.18; 95% CI 0.20–7.12; $P = 0.857$).
Winter *et al.*[87]	429	Severe secondary TR	17%	Regression: 43%	Persistence of severe secondary TR was associated with an increased risk of mortality after TAVI (adjusted HR 2.09, 95% CI 1.20–3.66, $P = 0.01$).
Shamekhi *et al.*[112]	1412	≥Moderate TR	33%		Baseline TR severity was an independent predictor of 1-year mortality (adjusted HR 1.37; 95% CI 1.0–1.8; $P = 0.031$).
Yoshida *et al.*[113]	1085	≥Moderate secondary TR	9%	Regression: 47%	Persistent TR (vs. improved TR) was associated with an increased risk of a composite endpoint of mortality and HF rehospitalization (HR 2.85; 95% CI 1.19–6.86; $P = 0.019$).
Worku *et al.*[114]	369	≥Moderate TR	16%	Regression: 36%	Persistent ≥moderate TR, but not baseline TR, was associated with an increased risk of mortality ($P = 0.02$).
	34 576	Mild TR	56%		

Continued

Table 4 *Continued*

Study	N	VHD	Prevalence	Change[a]	Importance of concomitant valvular heart disease
McCarthy *et al.*[115]		Moderate TR	19%		Baseline severe TR was associated with an increased risk of 1-year mortality in patients with LVEF >30% (adjusted HR 1.29; 95% CI 1.11–1.50).
		Severe TR	5%		
Schwartz *et al.*[116]	519	≥Moderate TR	11%	Regression: 59% (at 6 months)	Baseline ≥moderate TR was not independently associated with an increased risk of 1-year mortality (adjusted HR 0.75; 95% CI 0.28–1.8; $P = 0.50$).
Lindman *et al.*[117]	542	Moderate TR	23%	Regression: 31%	Baseline ≥moderate TR was associated with an increased risk of 1-year mortality (adjusted HR 1.76; 95% CI 1.14–2.70; $P = 0.01$).
		Severe TR	4%		
Barbanti *et al.*[118]	518	≥Moderate TR	15%	Regression: 15%	Baseline ≥moderate TR was not associated with 2-year mortality after TAVI (adjusted HR 1.55; 95% CI 0.91–2.64; $P = 0.105$).
Hutter *et al.*[98]	268	≥Moderate TR	22%	Regression: 50% (at 6 months)	Baseline ≥moderate TR was not associated with 1-year mortality in a multivariable analysis (33.9 vs. 20.9%; $P =$ NA).

HR, hazard ratio; CI, confidence interval; HF, heart failure; MVA, mitral valve area; AVR, aortic valve replacement; NA, not available.
[a]Change in severity of concomitant valvular heart disease after TAVI.
[b]The study includes both TAVI and SAVR populations.

Conversely, the occurrence of new-onset post-operative atrial fibrillation (POAF) has been consistently higher after SAVR than TAVI in the randomized clinical trials as well as temporary impairment of renal function and bleeding complications.[1–7] The natural course (development and resolution) and long-term impact of POAF remains to be determined. In the PARTNER 3 trial, in-hospital POAF was more frequent following SAVR compared with TAVI, but not independently associated with outcomes at 2 years, irrespective of treatment modality.[166]

Durability data

Durability data for THV are mainly limited to elderly populations with a mean age of over 80 years (*Table 6*). Although the randomized trials and observational studies reported durability data of THVs comparable to surgical bioprostheses up to 8 years,[9,160,167–176] the structural valve deterioration (SVD) has mainly occurred more than 10 years after surgical bioprosthesis implantation and the competing risk of mortality due to limited life expectancy may camouflage detection of SVD.[177] At the same time, it should be emphasized that, despite the long history of over half a century of implantation of surgical bioprostheses, there have been no systematic surveillance studies, and thus, there are insufficient data on the long-term durability of surgical bioprostheses. There are variable definitions of SVD (see Supplementary material online, *Table 2*), the absence of core laboratory analyses, the lack of systematic echocardiographic evaluation during longitudinal follow-up, incomplete clinical follow-up, and the iterative advent of new surgical bioprostheses that have led to uncertainty. The actuarial freedom from SVD was reported as almost 100% at 5 years, over 90% at 10 years, and over 80% at 15 years across surgical bioprostheses except for some studies[177] (*Figure 10*) with the general finding of accelerated structural deterioration in younger patient populations. Often surgical studies have equalled SVD with the need for reoperation, a poorly defined and easily biased endpoint that cannot be compared with the recent TAVI (vs. SAVR) studies using more rigorous SVD definitions, and core-lab adjudicated follow-up echocardiography (see Supplementary material online, *Table S2*). Thus, valve durability data are currently not conclusive for the decision-making between TAVI and SAVR, but need to be considered for the decision-making between bioprostheses and mechanical prostheses (or the Ross procedure) in younger patients <65 years of age.

Mechanical vs. bioprosthetic valves or the autograft

Several randomized clinical trials and observational studies have compared surgical mechanical prostheses vs. bioprostheses in patients of various ages ranging from their 40s to 70s[178–189] (see Supplementary material online, *Table S3*). Although the risk of reoperation was consistently lower for mechanical prostheses than bioprostheses, the overall benefit in terms of survival with mechanical valves was only evident in a few studies, predominantly including younger patients into their mid-60s.[180,181,184,189] According to current guidelines, patients >65 years of age should be treated with bioprostheses, whereas patients younger than 50 (AHA/ACC) or 60 (ESC/EACTS) years of age should be strongly considered for treatment with mechanical prostheses.[13,14]

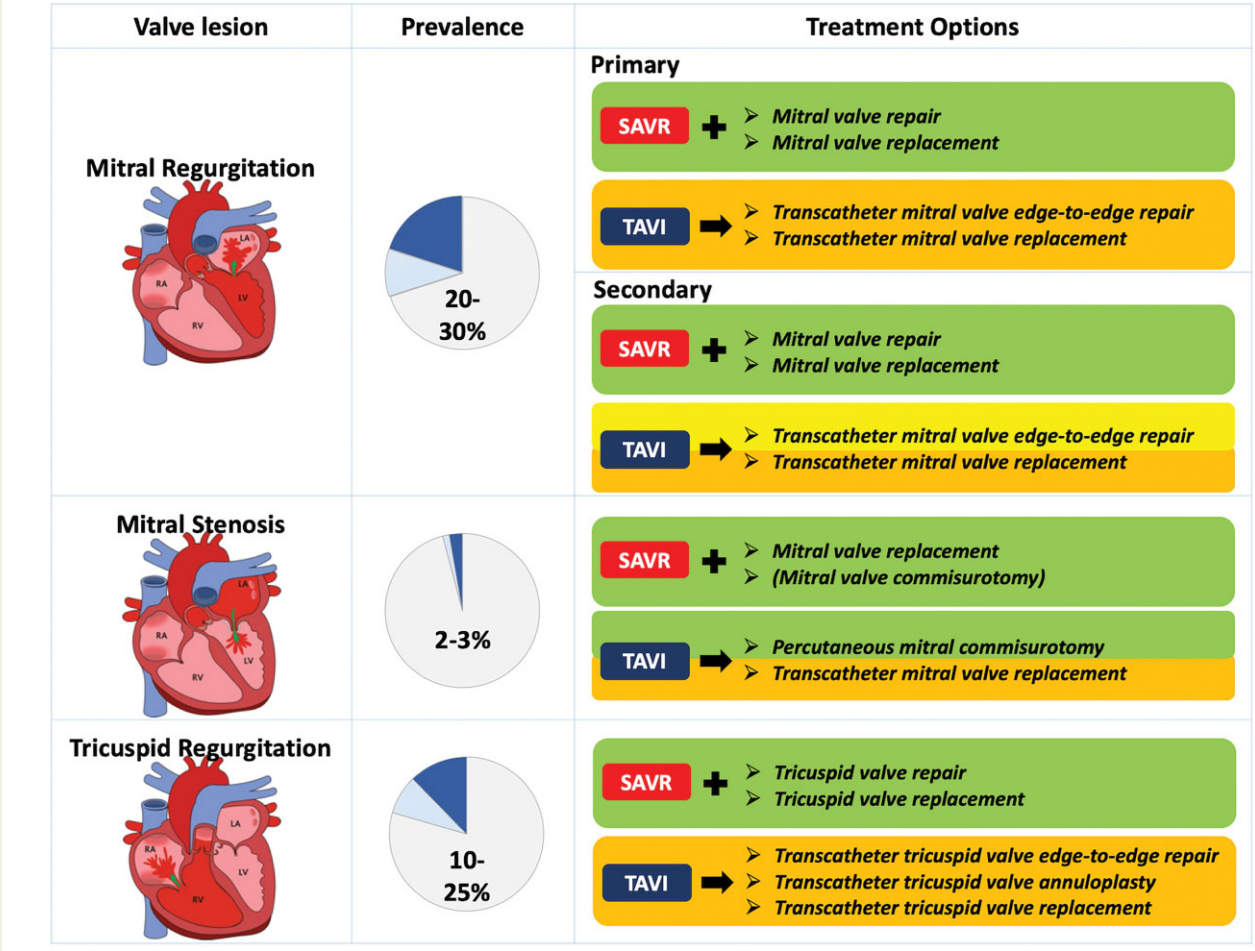

Figure 7 Potential strategies for mixed valve disease. The prevalence presented in the figure is for moderate or severe valvular disease derived from the transcatheter aortic valve implantation population in Western countries.

The Ross procedure, replacement of the aortic valve with a pulmonary autograft and placement of a homograft in the pulmonary position, is another attractive option for young and middle-aged adults[190–193] (see Supplementary material online, *Table S3*). It eliminates the need for life-long anticoagulation and provides a viable aortic valve substitute with adaptive remodelling and haemodynamics similar to that of the native aortic valve. In a recent meta-analysis including 3516 patients derived from 18 studies, the Ross procedure was associated with lower all-cause mortality but higher risk of reoperation (including reoperation on the pulmonary auto/homograft) compared with mechanical SAVR during a median follow-up of 5.8 years.[194]

Potential lifetime strategies

Although the Ross procedure and SAVR with mechanical prostheses remain attractive for younger patients as a longer-term solution, each procedure has its own inherent limitations (risk of reoperation both for aortic autograft and pulmonary homograft for the Ross procedure; bleeding risk due to life-long anticoagulation for mechanical prostheses). If these patients would undergo treatment with bioprostheses, three or more interventions may be required during their lifetimes.[161] While multiple open-heart surgeries may not be desirable for most patients, incorporating TAVI in the sequence of required interventions makes this strategy more realistic (*Figure 9*).

As redo SAVR and valve-in-valve TAVI are both considered reasonable treatment options,[13,14] redo SAVR in patients in their 60s followed by valve-in-valve TAVI as a third intervention in their 70s to 80s may be a plausible treatment strategy (SAVR–SAVR–TAVI).

A less-invasive strategy, such as TAVI–SAVR–TAVI and SAVR–TAVI–TAVI, is another potential strategy with the need for only one open-heart surgery during lifetime, which will be intuitively more attractive to patients. Transcatheter aortic valve implantation as a first intervention in young patients, who are working, exercising, or wishing to become pregnant, may be beneficial in terms of rapid recovery and no need for long-term anticoagulation. Surgical aortic valve replacement as a second intervention can be performed on a virgin chest at a reasonable age in their 60s to early 70s, and valve-in-valve TAVI remains a viable option for a third intervention. However, there is no data on the durability of THVs in such a young population. Depending on the type of implant at the index TAVI procedure, explantation of the valve may require additional manoeuvres and more extensive surgery such as root replacement and or replacement

Table 5 Evidence summary: prevalence and importance of concomitant coronary artery disease in patients undergoing transcatheter aortic valve implantation

Study	N	CAD definition	Prevalence	Importance of concomitant CAD
Matta et al.[128]	1030	Stenosis ≥50% in a major coronary vessel by pre-TAVI work-up	36.1%	No significant differences in in-hospital outcomes (death, bleeding, vascular complications, stroke, AKI, and pacemaker implantation).
Saia et al.[129]	540	Stenosis >70% (>50% for LMCA) in epicardial vessel with diameter ≥2 mm or previous coronary revascularization	53.9%	Neither CAD nor incomplete revascularization was associated with 5-year survival free from CV death (79.6 vs. 77.9%; $P = 0.98$, 84.3 vs. 74.3%; $P = 0.25$).
Elbaz et al.[130]	888	Stenosis >70% in any coronary artery (>50% for LMCA)	<3VD and non-LM: 62.2% 3VD or LM: 12.3%	Severity of CAD was not significantly associated with 1-year mortality (3 or LMCA vs. 1–2 vs. no CAD: 22.0 vs. 16.5 vs. 17.2%, $P = 0.38$).
Hollriegel et al.[131]	2624	Previous CABG	9.9%	Previous CABG was not associated with an increase in peri-procedural complications or 1-year mortality when adjusted for other comorbidities.
Guedeney et al.[132]	787	Prior MI or coronary revascularization, or diseased coronary vessels at angiography	CAD without recent PCI: 31.4% Recent PCI within 30 days: 10.3%	Both CAD groups were associated with VARC-2 efficacy endpoint at 1 year compared with no CAD (CAD without PCI vs. no CAD: adjusted HR 1.56; 95% CI 1.03–2.39; $P = 0.038$, CAD with recent PCI vs. no CAD: adjusted HR 1.96; 95% CI 1.1–3.5; $P = 0.021$).
Ryan et al.[133]	402	Stenosis ≥50% severity in vessel ≥1.5 mm	SS-II <37.4: 33.3% SS-II 37.4–44.0: 33.3% SS-II >44.0: 33.3%	The highest SS-II tertile was independently associated with an increased risk of mortality ($P = 0.046$) and MACE ($P = 0.001$). (MACE: a composite of mortality, cerebrovascular event, or myocardial infarction)
Huczek et al.[134]	896	Stenosis >70% severity in vessel >1.5 mm (50% for the left main)	51.6%	CAD was independently associated with an increased risk of mortality (adjusted HR 1.74; 95% CI 1.03–2.94; $P = 0.037$).
Millan-Iturbe et al.[135]	924	Stenosis >70% severity (>50% for LMCA)	1VD: 150 (16.2%) 2VD: 51 (5.5%) 3VD: 23 (2.5%)	There was no difference in survival and need for revascularization post-TAVI between those patients with or without CAD ± revascularization.
Shamekhi et al.[136]	666	Stenosis ≥50% in vessel ≥1.5 mm	Low SS (<24): 313 (47.0%) High SS (≥24): 124 (18.6%)	Baseline and residual CAD severity was not independently associated with mortality.
Puymirat et al.[137]	3444	Stenosis of >50% diameter in major epicardial coronary vessel	1252 (36.4%)	Neither the presence nor the extent of CAD was associated with 3-year mortality. However, a significant lesion of the LAD was associated with higher 3-year mortality.
Witberg et al.[138]	1270	History of PCI, CABG or MI, or stenosis >50% severity in major epicardial coronary artery	Low SS (≤22): 26.1% High SS (>22): 9.6%	Severe CAD and incomplete revascularization were associated with an increased risk of mortality after multivariable adjustment.
Franzone et al.[139]	744	History of PCI, CABG or MI, or stenosis ≥50% severity of a major native coronary vessel or bypass graft	33.3%	The presence of CAD was associated with an increased risk of MACCE (16.8 vs. 9.8%, HR 1.75, 95% CI, 1.06–2.89, $P = 0.030$). (MACCE: a composite of CV mortality, MI, or cerebrovascular events)
Paradis et al.[140]	377	≥50% stenosis by QCA in vessels ≥1.5 mm	Low SS (≤2): 34.2% Intermediate SS (23–32): 12.7% High SS (>32): 31.3%	Neither the severity of CAD nor completeness of revascularization after percutaneous coronary intervention or CABG was associated with clinical outcomes after TAVI, at 30 days and 1 year.

Continued

Table 5 ***Continued***

Study	N	CAD definition	Prevalence	Importance of concomitant CAD
Khawaja *et al.*[141]	271	Stenosis ≥70% severity (≥50% for left main), using QCA	34.3%	CAD was not associated with mortality after TAVI (23.7 vs. 21.5%; $P = 0.805$). However, patients with a SS >9 had an increased risk of mortality than those with a SS ≤9 (34.3 vs. 20.7%; $P = 0.005$).
Mancio *et al.*[142]	91	Prior PCI or CABG, or ≥50% stenosis	50.5%	CAD was associated with an increased risk of 2-year mortality after TAVI (50 vs. 24%; adjusted HR 2.6; 95% CI 1.1–6.0; $P = 0.03$).
Snow *et al.*[143]	2588	Stenosis >50% severity in major epicardial coronary vessel	45.2%	CAD was not associated with both 30-day ($P = 0.36$) or 4-year survival ($P = 0.10$).
Stefanini *et al.*[144]	445	Stenosis ≥50% severity in vessel ≥1.5 mm in diameter	Low SS (≤22): 46.5% High SS (>22): 18.0%	CAD severity was associated with an increased risk of a composite endpoint of cardiovascular death, stroke, or MI at 1 year (no CAD: 12.5%, low SS: 16.1%, high SS: 29.6%; $P = 0.016$).
Gasparetto *et al.*[145]	191	Prior revascularization or any coronary stenosis ≥50% severity	59.2%	CAD was not associated with combined safety endpoint at 30-days ($P = 0.57$) or efficacy endpoint at 1 year ($P = 0.25$). (Safety endpoint: all-cause mortality, major stroke, life-threatening or disabling bleeding, acute kidney injury Stage 3, peri-procedural MI, major vascular complication, or repeat procedure for valve-related dysfunction.) (Efficacy endpoint: all-cause mortality, hospitalization for symptoms of valve-related or cardiac decompensation, prosthetic heart valve dysfunction.)
Ussia *et al.*[146]	659	Previous CABG or PCI	38.1%	CAD was not independently associated with a composite endpoint of all-cause death, MI, major stroke, or conversion to open-heart surgery at 1 year (5.7 vs. 18.3%; $HR_{adjusted}$ 0.76; 95% CI 0.42–1.36; $P = 0.353$).
Abdel-Wahab *et al.*[147]	1382	Previous CABG or PCI and/or ≥50% stenosis	62.2%	CAD was not independently associated with in-hospital mortality (10.0 vs. 5.5%; adjusted OR 1.41; 95% CI 0.85–2.33).
Gautier *et al.*[148]	145	History of MI or coronary revascularization or ≥70% (≥50% for left main) stenosis by QCA	57.2%	CAD was not associated with 30-day (90 vs. 85%; $P = 0.37$) or 1-year survival (76.4 vs. 70.6%; $P = 0.28$).
Masson *et al.*[149]	136	Prior revascularization or any coronary stenosis ≥50% severity	75.7%	The presence of CAD or non-revascularized myocardium was not associated with an increased risk of mortality up to 1 year.
Dewey *et al.*[150]	171	Previous CABG or PCI	49.1%	Overall mortality was higher among the CAD group than the non-CAD group (35.7 vs. 18.4%; $P = 0.01$).

CAD, coronary artery disease; AKI, acute kidney injury; LMCA, left main coronary artery; CV, cardiovascular; VD, vessel disease; LM, left main; CABG, coronary artery bypass graft; MI, myocardial infarction; PCI, percutaneous coronary intervention; VARC, Valve Academic Research Consortium; HR, hazard ratio; CI, confidence interval; SS, syntax score; MACE, major adverse cardiac events; MACCE, major adverse cardiac and cerebrovascular events; QCA, quantitative coronary angiography.

of the ascending aorta.[195–198] In recent observational studies, more than half of patients undergoing TAVI explantation required concomitant procedures, the most common of which was aortic root repair/replacement, followed by mitral valve repair/replacement.[197,198]

Surgical aortic valve replacement as a first procedure may be followed by two valve-in-valve TAVI interventions (SAVR–TAVI–TAVI). However, the more frequently valve-in-valve TAVI is repeated, the more attention needs to be directed to careful procedural planning to minimize the risk of coronary obstruction and prosthesis–patient mismatch. Furthermore, coronary access may be challenging depending on the implanted THV and patient anatomy after a second procedure, and even more challenging after a third procedure.[156,199] In order to follow this strategy, it is of paramount importance to implant the largest possible surgical bioprosthesis to maximize effective orifice area at the time of first valve intervention and to avoid sutureless or stentless bioprostheses. In this scenario, however, there may be few options for a fourth procedure if the patient outlives the third THV.

In patients in their 60s to 70s with life expectancy of over 15–20 years, SAVR–SAVR, SAVR–TAVI, and TAVI–TAVI are potential strategies. Given the available evidence and current guideline

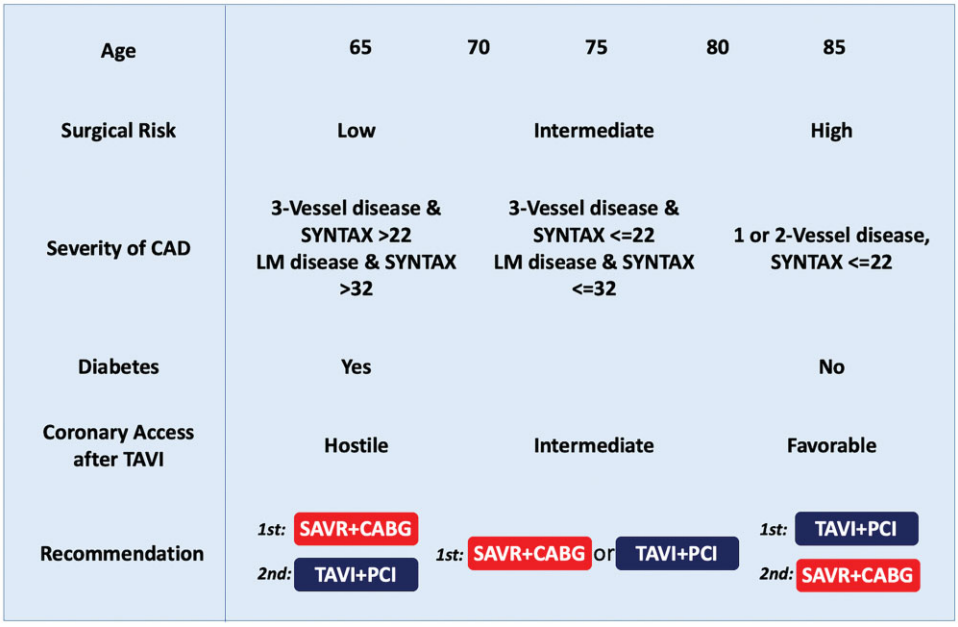

Figure 8 Recommendation for the management of severe aortic valve stenosis and concomitant clinically relevant coronary artery disease requiring intervention. CAD, coronary artery disease; LM, left main; CABG, coronary artery disease; PCI, percutaneous coronary intervention.

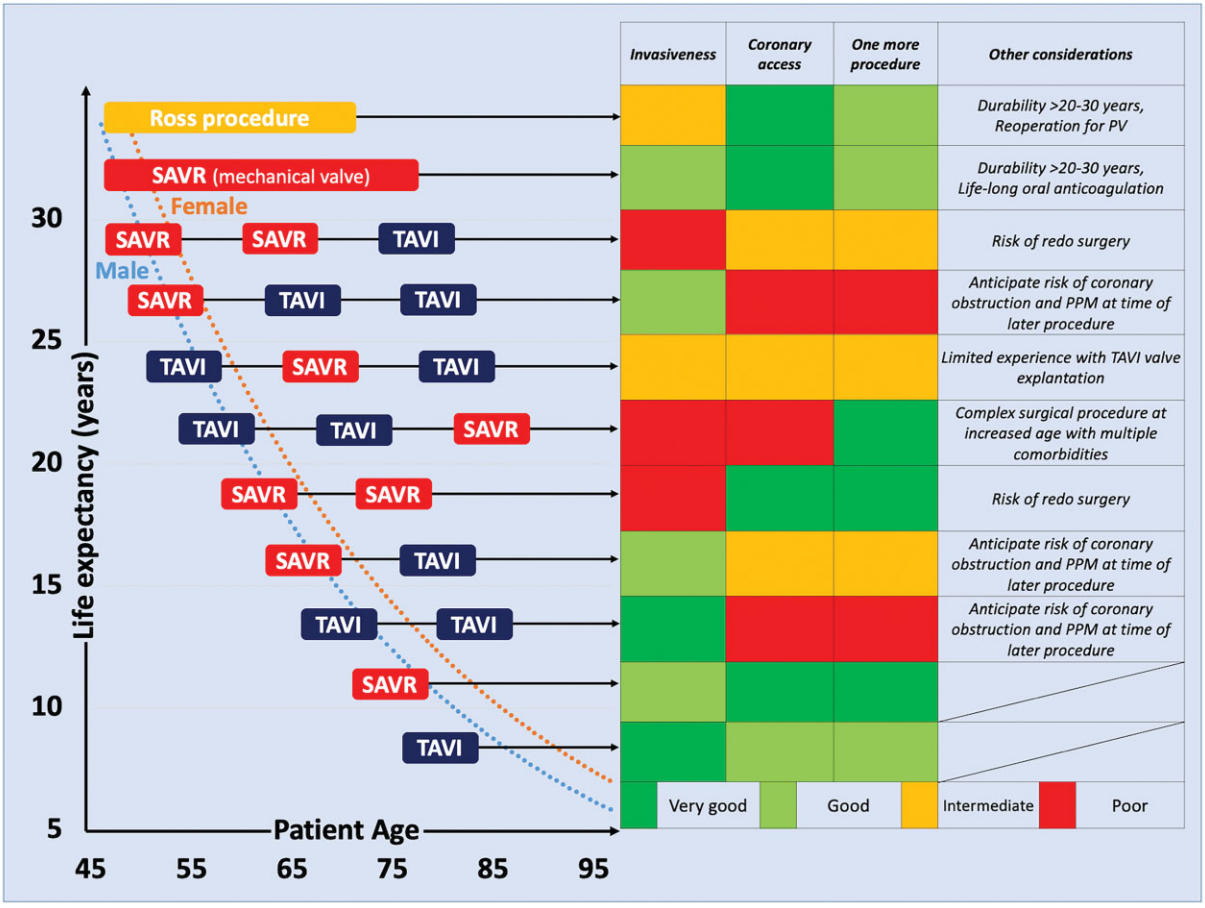

Figure 9 Strategies for lifetime management according to patient's life expectancy. Exponential lifetime curves derived from life expectancy data in Switzerland in 2019 are provided. PV, pulmonary valve; PPM, prosthesis–patient mismatch.

Table 6 Durability data of transcatheter aortic bioprostheses (vs. surgical aortic bioprostheses)

Study	Age	Follow-up	TAVI			SAVR		
			Valve type (*N*)	SVD	BVF	Valve type (*N*)	SVD	BVF
Randomized clinical trials								
NOTION Jorgensen *et al.*[160]	79	8 years	CoreValve (139)	13.9% Moderate: 13.2% Severe: 2.2%	8.7% Re-intervention: 3.6%	Multiple (135)	28.6% Moderate: 27.5% Severe: 6.8%	10.5% Re-intervention: 2.3%
PARTNER 2[a] Pibarot *et al.*[167]	82	4 years	SAPIEN 3 (891) SAPIEN XT (774)	3.9% 9.5%	2.6% 4.7%	Multiple (664)	3.5%	1.3%
CoreValve US High Risk Gleason *et al.*[9]	83	4 years	CoreValve (390)	9.5% Moderate: 9.2% Severe: 0.8%	NA	Multiple (354)	26.6% Moderate: 26.6% Severe: 1.7%	NA
Observational studies								
PS-matched study Tzamalis *et al.*[168]	78	7 years	SAPIEN/SAPIEN XT/CoreValve/ACURATE (209)	Moderate: 9.3% Severe: 10.5%	4.8% Re-intervention: 4.3%	Multiple (198)	Moderate: 2.3% Severe: 4.5%	2.0% Re-intervention: 2.0%
Italian multicentre registry Testa *et al.*[169]	82	8 years	CoreValve (990)	Moderate: 3.0% Severe: 1.6%	2.5%			
UK-TAVR Registry Blackman *et al.*[170]	79	5–6 years	SAPIEN/SAPIEN XT/CoreValve/Portico (241)	Moderate: 8.7% Severe: 0.4%				
French multicentre registry Durand *et al.*[171]	83	7 years		10.8% Moderate: 7.0% Severe: 4.2%	1.9% Re-intervention: 1.0%			
Single centre registry Panico *et al.*[172]	82	8 years	CoreValve (278)	3.6%	2.5%			
FRANCE-2 Registry Didier *et al.*[173]	83	5 years	SAPIEN/SAPIEN XT/CoreValve (4201)	13.3% Moderate: 10.8% Severe: 2.5%				
Single centre registry Deutsch *et al.*[174]	81	7 years	SAPIEN XT/CoreValve (300)	14.9%				
Single centre registry Eltchaninoff *et al.*[175]	83	8 years	SAPIEN/SAPIEN XT (378)	3.2%	0.58%			
Single centre registry Barbanti *et al.*[176]	81	8 years	SAPIEN XT/CoreValve (288)	Severe: 2.4%	4.5%			

SVD and BVF were defined according to the consensus statement by EAPCI/EACTS (Capodanno *et al.*[211]) except for the study below. TAVI, transcatheter aortic valve implantation; SAVR, surgical aortic valve replacement; SVD, structural valve deterioration; BVF, bioprosthetic valve failure.

[a]SVD and BVF according to the VARC-3 criteria.

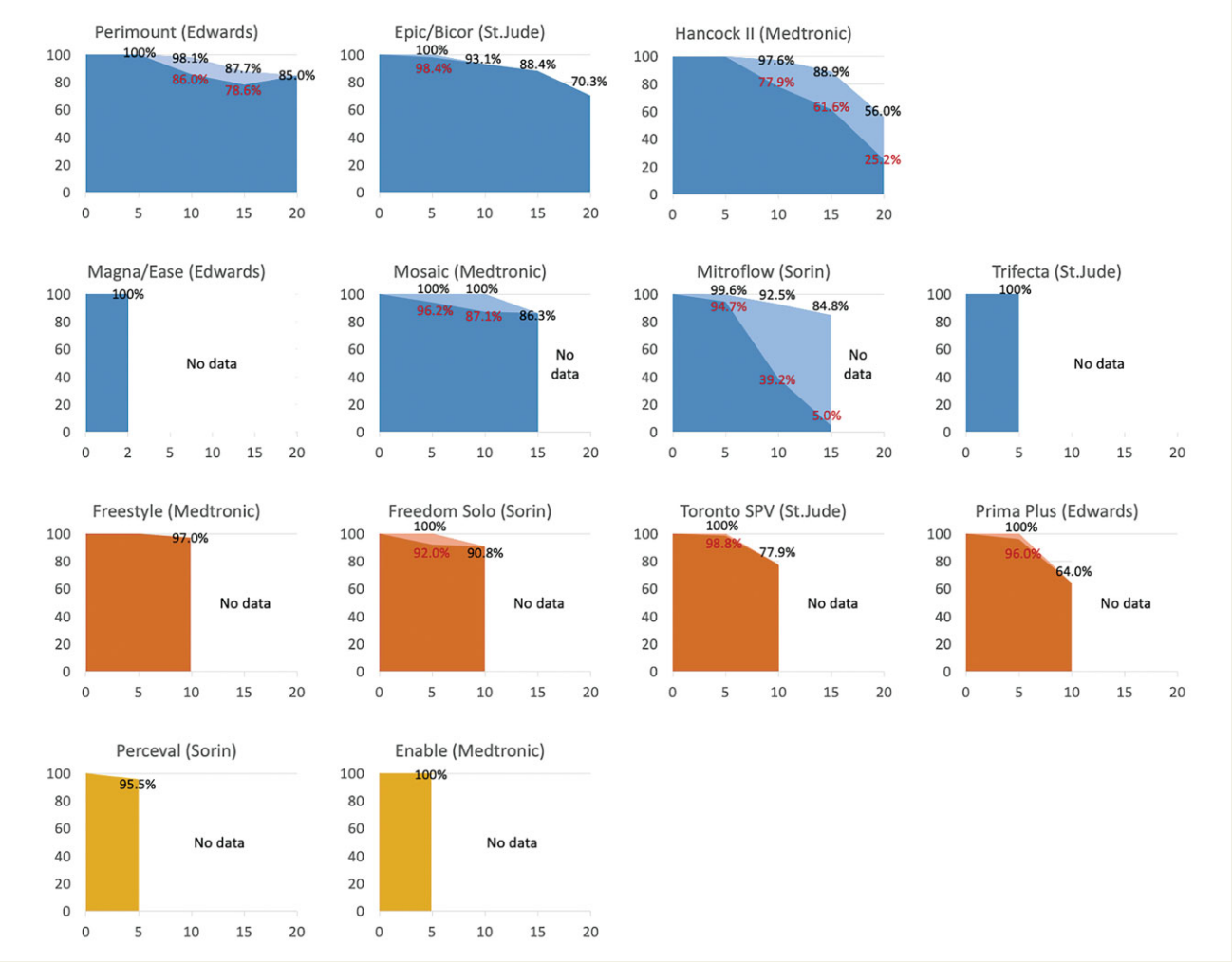

Figure 10 Durability data of surgical aortic bioprostheses. A range of actuarial freedom from SVD for each surgical bioprosthesis, obtained from studies in which these data were available for the whole cohort, is provided.[177]

recommendations, SAVR as a first intervention followed by valve-in-valve TAVI appears a reasonable strategy today.[13,14] However, TAVI followed by valve-in-valve TAVI may become the preferred option for selected patients at high surgical risk and optimal anatomy.[63,64] Although all of these scenarios are thoughtful hypothetical considerations, the one scenario that should most likely be avoided is TAVI–TAVI–SAVR in that the schema would require a complex surgical procedure when the patient is elderly with more comorbidities.

Improvements in surgical aortic valve replacement and transcatheter aortic valve implantation device technologies and techniques

Technologies and techniques continue to evolve both with SAVR and TAVI. Minimally invasive cardiac surgery can reduce blood loss, preserve pulmonary function, and enable more rapid recovery.[200] Although the technique was introduced already in the 90s, it has now become the access of choice in many centres.[201,202] New bioprostheses continue to be introduced to improve patient outcome by slowing the process of structural valve degeneration and with an intent to facilitate future valve-in-valve implantation.[203,204]

Similarly, new techniques continue to be advanced in the field of TAVI such as high THV deployment,[25,26] commissural alignment,[157,158] and electrosurgical procedures (BASILICA and transcaval access for patients with severe peripheral vascular disease) facilitating TAVI.[40,205] Existing devices are continuously improved, and new devices are developed to overcome current TAVI limitations.[31–33] The ongoing technological and technical innovations will continue to improve procedural outcomes, impact long-term durability, and adjunct medical therapy and will affect the lifetime management in patients with severe AS.

Centres of excellence

The establishment of multidisciplinary heart teams consisting of various professionals with extensive experience and familiarity with all

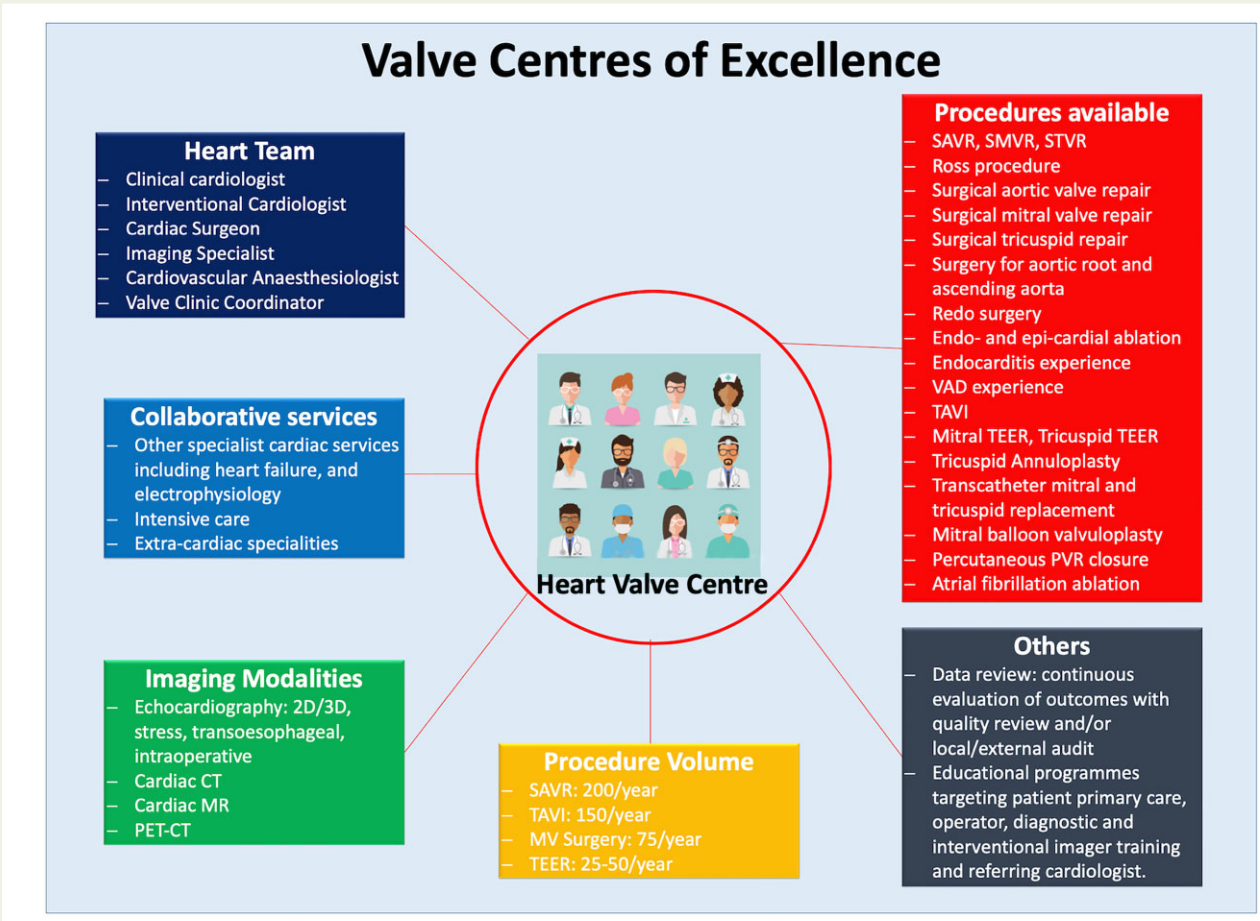

Figure 11 Requirements for comprehensive heart valve centres. Procedural volume for each procedure is based on previous literature.[206–210] The illustration at the centre of this figure was designed by Sapann-Design / Freepik. CT, computed tomography; PET, positron emission tomography; SAVR, surgical aortic valve replacement; SMVR, surgical mitral valve replacement; STVR, surgical tricuspid valve replacement; TAVI, transcatheter aortic valve implantation; TEER, transcatheter edge-to-edge repair.

aspects in the diagnosis and treatment of patients with valvular heart disease is a basic requirement to provide the highest level of care. To maintain the human resources, the facilities and necessary equipment required, and a sufficient volume for each procedure, it is increasingly important to centralize these resources and patients in large comprehensive heart valve centres. The heart valve centre must collect and report its results, continuously monitor its data, implement quality assurance systems with audits, and should establish a training programme to help ensure education and continuous improvement in quality[13,14] (*Figure 11*).

Conclusions

'Which patients with severe AS should be referred to surgery?' is an evolving clinical question in the management of patients with severe AS that has arisen with the advent of TAVI and its reproducible, excellent outcomes. Anatomical and clinical factors, remaining uncertainties related to TAVI and SAVR, and lifetime management strategies now take centre stage in the decision-making process and the proposed strategies in this review will require update based on forthcoming data (see Supplementary material online, *Table S4*).

The multidisciplinary heart team plays a pivotal role to provide an optimal treatment recommendation in a shared decision-making process for individual patients and to define the lifetime sequence of interventions (*Graphical Abstract*).

Supplementary material

Supplementary material is available at *European Heart Journal* online.

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payments by pharmaceutical companies or device manufacturers. He is also a member of the steering/executive committee group of several investigator-initiated trials that receive funding by industry without impact on his personal remuneration. T.O. reports speaker fees from Abbott. V.F. has relevant (institutional) financial activities outside the submitted work with following commercial entities: Medtronic GmbH, Biotronik SE & Co., Abbott GmbH & Co. KG, Boston Scientific, Edwards Lifesciences, Berlin Heart, Novartis Pharma GmbH, JOTEC/CryoLife GmbH, LivaNova, Zurich Heart in relation to Educational Grants (including travel support), fees for lectures and speeches, fees for professional consultation, Research and study funds. A.U. serves as a physician proctor to Boston Scientific, Edwards Lifesciences, and Medtronic. M.M. was the Co-Principal Investigator for the PARTNER 3 Trial of Edwards Lifesciences, the COAPT Trial of Abbott and is Study Chair of the Apollo Trial of Medtronic. Travel expenses for trial-related meetings were paid by the sponsors. All other authors have no relationships relevant to the contents of this article to disclose.

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