Transcatheter Aortic Valve Implantation (TAVI) for Aortic Stenosis

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Background

Aortic stenosis remains the commonest form of valve disease in modern cardiology. With fifty years' experience, surgical valve replacement remains the gold standard treatment for survival benefit, durability and symptomatic relief. Percutaneous transcatheter aortic valve replacement has recently gained a credible momentum for inoperable and very high risk patients with severe aortic stenosis. Early and medium term results have demonstrated a proven survival benefit over conservative management, with documented complication rates for stroke, vascular complications and pacemaker implantation. The evidence base for cost effectiveness and long-term results are eagerly anticipated.

Mechanisms and Natural History

Degenerative aortic valve disease, characterised by progressive thickening, narrowing and obstruction of the valve leaflets, is a common finding in the elderly population. This triggers initially a chronic compensatory mechanism of elevated inotropic state and ventricular hypertrophy to minimise wall stress. The physiological aorto-ventricular coupling is disturbed with increasing pressure drop and hypertrophy, and the prolonged initially asymptomatic course of the disease is followed by a more rapid symptomatic phase. Progressive cavity dilatation leads to decompensation due to falling cardiac output and anginal symptoms.

The prevalence of non-rheumatic, non-congenital aortic valve sclerosis has been reported as 20% in populations aged 65 to 74, rising to 48% by age 85 and beyond. The prevalence of calcific aortic stenosis (AS) rises with age -- 1.3% and between 2.8-4%, respectively in the same population.1-2 The pathogenesis of calcific aortic stenosis has evolved beyond the hypothesis of age-related ‘wear and tear’ to one of a complex interplay of conventional atherosclerotic risks, mechanical leaflet stress, endothelial dysfunction, and calcium deposition.2 Clinically symptomatic aortic stenosis is heralded by chest pain (often in the absence of obstructive coronary artery disease), syncope, and exercise intolerance.4 Left untreated, symptomatic disease results in repeated hospitalisations and confers a mortality risk of 50% at two years5-10 -- thus the natural history of aortic stenosis is strikingly similar to many forms of cancer.11-13

Assessment of aortic stenosis

Aortic stenosis may be objectively assessed by valve area or transaortic pressure drop. Non-invasive assessment of aortic stenosis by valve area, regardless of imaging modality, is based on principles of conservation of mass and the continuity equation. Invasive LV catheter pressure measurements of pressure drop are taken at peak-to-peak intervals and may thus differ slightly from Doppler measurements; invasive catheter aortic valve area is calculated using the Gorlin equation.15 The typical definition of ‘severe’ aortic stenosis varies according to assessment method as detailed in Table 1.

All calculated measures of aortic valve area are subject to measurement error15, 22, 23 of the LVOT diameter and Doppler envelope. Finegold et al.22 reported better reproducibility ofAVA by ratio of peak Doppler velocity compared to Doppler VTI, but with wide confidence limits of +/- 25%.

Predictors of prognosis

Several studies have reported that in asymptomatic severe AS, change in peak velocity and calcification predicted survival, along with ischaemic heart disease, lung and renal comorbidities,24 whereas aortic valve area was not similarly predictive of prognosis.25-27 There is no standardised definition of low flow, “low gradient” aortic stenosis. Typical criteria have included impaired LV with EF ≤ 35 - 40%, mean gradient ≤ 30 -40 mm Hg, and AVA ≤ 1 - 1.2 cm2. In low-flow “low-gradient” AS where the apparent transaortic pressure drop falls due to LV impairment, prognosis is dictated by a combination of functional impairment, atrial fibrillation, stress response, and corrected valve area.25,26 Low baseline ejection fraction, restrictive LV filling and raised pulmonary artery pressure have been found as predictors of survival after AVR.27

Paradoxical low-flow low-gradient severe AS

Patients with preserved EF (>50%), low indexed stroke volume (< 35ml/m2), low ‘gradient’ (<40mm Hg), A VA < 0.7 cm2/m2 [AVA < 1cm2 unindexed] appear to comprise a clinically distinct subset of AS patients. They have a poor prognosis as the severity of disease is underestimated and hence conventional surgical AVR referral happens late in the course of the disease. The conventional hypothesis is that these patients have severe AS that is masked by a combination of decreased arterial compliance, increased valvulo-arterial impedance (Za), hypertrophied ventricles with smaller cavity volume, and lower LV mid-wall radial shortening, longitudinal shortening and ['strain' rate]28. The constellation above implies the pathological triad of: increased systemic vascular resistance, obstructive valve disease, and intrinsic myocardial disease. The decreased arterial compliance may be due to hypertensive disease and can be represented by \[Z_{av} = \text{systolic pressure} + \text{mean pressure gradient} / \text{indexed stroke volume}\]. The obstructive valve disease is manifest as calcific aortic stenosis. Intrinsic myocardial disease may manifest as systolic and

<table>
<thead>
<tr>
<th>Peak aortic velocity19</th>
<th>Aortic Valve Area20, 21</th>
<th>Dimensionless Index22</th>
<th>Mean gradient21</th>
</tr>
</thead>
<tbody>
<tr>
<td>&gt;4 m/s</td>
<td>&lt;1 cm²24</td>
<td>&lt; 0.25</td>
<td>&gt;40 mm Hg</td>
</tr>
<tr>
<td></td>
<td>&lt;0.75 cm²23 (indexed to BSA)</td>
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Table 1: Equivalent definitions of typical severe aortic stenosis.
diastolic ‘dysfunction’== typical EF value in the lower-end of normal despite LVH and small cavity volume; this belies a low stroke volume due to pronounced LV fibrosis, fibre architecture disarray and thus deranged remodelling compared to high gradient AS. Flett et al. found in severe AS patients, increased diffuse myocardial fibrosis correlated with severity of diastolic dysfunction and predicted exercise tolerance by 6-minute walk test. Thus underlying fibrosis may also manifest as a restrictive LV physiology due to high end-diastolic pressures and decreased total LV filling time. Ultimately, the patient experiences anginal symptoms and effort intolerance disproportionate to conventional markers of aortic valve disease due to underlying pathological disturbance in VA impedance, decreased coronary diastolic flow reserve, and raised filling pressures. Thus, a unifying theory of arterio-valvulo-ventricular coupling is required to explain the pathophysiology in paradoxical low-flow low-gradient severe AS.

Management of aortic stenosis

Surgical aortic valve replacement (AVR) remains the gold standard treatment for the majority of symptomatic patients. Fifty years’ experience has resulted in improved survival due to developments in cardiopulmonary bypass techniques, valve technology, and myocardial preservation. In developed Western economies, the annual volume of AVR ranges from 12,000 in Germany to more than 60,000 in the USA. Operative survival after elective AVR in 1960 was initially 50%, but now routinely exceeds 97.5% in low-risk patient groups.

Surgical AVR techniques differ mainly in terms of valve prosthesis; minor variations occur due to operator choice of bypass or cardioplegia. Options for prosthetic valves include mechanical or tissue (allograft, homograft or autograft) in stented or stentless montagings. Mechanical valves offer the advantage of long term durability and reliability balanced against risk of thromboembolism and haemorrhage from requisite anticoagulation. To date, there are no satisfactory medical treatments for aortic stenosis that alter progression or mortality. Recent trials (SEAS, SALTIRE) predicated on pleiomorphic principles of statin and fibrate inhibition of calcium deposition have shown no benefit to alter survival or progression in AS.

Although surgical AVR remains the definitive treatment for AS, a significant proportion of patients are denied surgical valve replacement due to high surgical risk. Somewhat surprisingly from the EuroHeart survey, up to one-third of symptomatic severe AS patients were denied surgery primarily on the basis of age and LV systolic impairment over and above validated cardiovascular comorbidities. Thus, in the absence of viable medical management or open surgical AVR as options, symptomatic patients with severe AS may be managed palliatively or offered percutaneous transcatheter aortic valve intervention (TAVI).

Transcatheter Aortic Valve Implantation (TAVI)

Transcatheter aortic valve implantation (TAVI) was first reported in man by Cribier et al. in 2002 and has since gained popularity and credibility as a viable treatment option for patients with inoperable and high-risk aortic stenosis. Percutaneous valve replacement has a proven evidence base from pulmonary valve replacement in the right ventricular outflow tract (RVOT) and TAVI procedures offer similar advantages by avoiding cardiopulmonary bypass and open chest access. Two competing systems with MHRA, CE- and FDA approval from Edwards Lifesciences (Sapien series) and Medtronic (CoreValve) have been evaluated in high-quality trials. The two devices differ in terms of leaflet and stent material, annulus sizing, deployment method, and can be delivered via transapical, transfemoral, subclavian or axillary access. Evolving experience suggests that TAVI offers improved survival compared to medical or valvuloplasty treatment for inoperable patients, and comparable results to high-risk surgical AVR patients. Ongoing trial data have also highlighted the impact of TAVI-associated complications of stroke, vascular complications, pacemaker implantation and paravalvular aortic regurgitation (AR) on morbidity and mortality.

Clinical Outcomes after TAVI and the importance of aortic regurgitation

Even in isolated aortic stenosis, aortic regurgitation remains an important prognostic finding after both surgery or percutaneous intervention. Post-procedural aortic regurgitation is a mediator of morbidity and prognosis. The incidence of significant post-operative prosthetic paravalvular AR after surgical AVR is rare at less than 1%, but is more common (pooled estimate 7.4%) and important after transcatheter AVR. Major TAVI trials consistently confirm that post-procedural aortic regurgitation predicts survival.

There is no standardised grading for paravalvular AR, and thus Valve Academic Research Consortium (VARC) definitions have been proposed to standardise definitions for clinical endpoints for TAVI. Central jet AR is graded on criteria for jet density, jet width, Doppler deceleration time. Paravalvular AR may be assessed on the percentage of circumferential paraprosthetic leak, but this has not yet been validated and remains controversial.

Genereux et al., Jilaihawi et al., and Moat et al. reported separate meta-analyses of TAVI studies across more than 3,500 patients, respectively. Pooled TAVI outcomes were broadly similar as illustrated in Table 3. TAVI 30-day mortality was similar at 7.8 – 8.5%; 1-year mortality of 21-22%, and 2-year mortality of 26%. Multivariate predictors of mortality were LV dysfunction (EF<30%), moderate to severe AR, and COPD. Overall incidence of stroke was 2.6 – 4.1%, moderate AR 13.6%, major vascular complication 6.3%. Pooled pacemaker implantation incidence was 16.3% (CoreValve v Sapien, 24 – 28.9% v 4.9 – 7.4%, p<0.001), with higher rate of occurrence in the CoreValve groups. This may be due to the self-expansible
properties of the nitinol mesh and taller implant profile in the LV outflow tract.50

The PARTNER trials
The US partner multi-centre randomised trials recruited patients into two separate arms – high-risk surgical aVR and inoperable surgical AS patients – with the Edwards Sapien percutaneous aortic valve prosthesis.

Partner 1A (High-risk operable AS patients -- Non-inferiority of TAVI v Surgical AVR)
The Partner 1A46,47 arm recruited 699 patients across 25 centres for randomisation to either conventional surgical aVR or transcatheter aortic valve intervention. The primary end point was non-inferiority of all-cause death in TaVI compared to surgical aVR, predefined as a percentage-difference of 7.5%. All-cause mortality was reported at 3.4% v 6.5% at 30 days (TAVI v surgical AVR, p=0.07), and 24% v 26.8% (p=0.45) at 1 year, (47) respectively. Two-year mortality was also not significantly different between the TAVI and surgical AVR treatment groups – 26.5% v 23.3% (p=0.54), (47) respectively. Stroke or TIA was more frequent after TaVI at 2 years – 6.5% v 11.2% (p=0.05), (47) At two years, increase in aortic valve area was similar in the two groups. Paravalvular aortic regurgitation was more frequent after TAVI (p<0.001), and notably more than mild paravalvular regurgitation was associated with increased late mortality; hazard ratio 2.11 (95% CI 1.43 – 3.1, p<0.001, p<0.001), see Figure 4(D). The Partner 1A authors recommended work should be directed toward (1) reducing paravalvular AR (2) techniques for precise valve sizing and positioning and (3) judicious use of post TAVI dilatation.

PARTNER 1B (TAVI v conservative management in inoperable severe AS patients)
The Partner 1B44, 45 trial arm recruited from 21 centres, 358 patients with inoperable severe as randomised to receive either conservative (medical management and/or balloon angioplasty) or TAVI. The primary end point for all-cause mortality at 1 year was reached in 30.7% v 50.7% (TaVI v medical, hazard ratio 0.55 [CI 0.4 to 0.74], p<0.001), Figure 5.44. a composite end point of death from any cause or repeat hospitalisation similarly favoured TaVI over conventional management, 42.5% v 71.6%, p<0.001). Major vascular complications were more frequent in TaVI patients (16.2% v 1.1%, p<0.001), and there was a trend toward more frequent major strokes following TaVI at 30 days (5.0% v 1.1%, p=0.06). At two years, the survival advantage for TaVI patients remained significant – all-cause mortality was lower in the TaVI group (43.4% v 68%, p<0.001). Major strokes were more common in the TAVI group (13.8% v 5.5% p=0.01) due to more ischaemic strokes in the TAVI group at 30 days (p=0.02), and a non-significant trend for more haemorrhagic strokes in the TAVI group beyond 30 days (p=0.16). Echocardiographic indicators of paravalvular AR and aortic valve area/gradient after TAVI did not significantly worsen at two years.

Table 3: Comparison of TAVI outcome data across pooled studies.

<table>
<thead>
<tr>
<th>Outcome</th>
<th>Genereux (50) N=3519</th>
<th>Jilaihawi (51) N=8536</th>
<th>Partner 1 (46) N=699</th>
<th>Moat (52) N=870</th>
</tr>
</thead>
<tbody>
<tr>
<td>30-day mortality</td>
<td>7.8%</td>
<td>8.5%</td>
<td>3.4%</td>
<td>7.1%</td>
</tr>
<tr>
<td>1-year mortality</td>
<td>22.1%</td>
<td>22.8% (p=0.85)</td>
<td>24%</td>
<td>26.8% (p=0.45)</td>
</tr>
<tr>
<td>2-year mortality</td>
<td>26.5%</td>
<td>23.3% (p=0.54)</td>
<td>33.9%</td>
<td>35% (p=0.78)</td>
</tr>
<tr>
<td>Paravalvular AR (moderate)</td>
<td>7.4%</td>
<td>11.6%</td>
<td>12.7% (moderate/severe)</td>
<td>13.6%</td>
</tr>
<tr>
<td>Stroke</td>
<td>3.2%</td>
<td>2.6% (p=0.72)</td>
<td>4.6% (30 d, p=0.1)</td>
<td>6%</td>
</tr>
<tr>
<td>Vascular complications</td>
<td>11.9%</td>
<td>6.6%</td>
<td>11%</td>
<td>3.2% (p=0.01)</td>
</tr>
<tr>
<td>Pacemaker insertion</td>
<td>13.9%</td>
<td>5.9% (p=0.05)</td>
<td>6.4%</td>
<td>5% (p=0.44)</td>
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</tbody>
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At two years, increase in aortic valve area was similar in the two groups. Paravalvular aortic regurgitation was more frequent after TAVI (p<0.001), and notably more than mild paravalvular regurgitation was associated with increased late mortality; hazard ratio 2.11 (95% CI 1.43 – 3.1, p<0.001, p<0.001), see Figure 4(D). The Partner 1A authors recommended work should be directed toward (1) reducing paravalvular AR (2) techniques for precise valve sizing and positioning and (3) judicious use of post TAVI dilatation.

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Notably, the data suggested that survival benefit from TAVI may diminish with higher surgical risk (STS) scores. Predictors of survival for TAVI patients were body mass index (hazard ratio 0.95 per unit increase [95% CI 0.91 to 0.98, p=0.005), prior stroke (hazard ratio 2.99, 95% CI 1.19 to 7.51, p=0.01) and COPD requiring supplemental oxygen (hazard ratio 1.69, 95% CI 1.05 to 2.73, p=0.03).\(^{48}\)

### Vascular complications in TAVI

Genereux et al.\(^{46}\) analysed TAVI recipients from both Partner 1A and 1B groups for predictors of major vascular complications. Major vascular complication was defined as any (1) thoracic aortic dissection or (2) any access site vascular injury requiring intervention, surgery, >4u blood transfusion leading to end-organ damage or death. Major vascular complications occurred in 15% of all TAVI patients within 30-days of the procedure, with significantly higher 30-day and 1-year mortality. Female gender was the only identifiable independent predictor of such vascular complications, even after accounting for small femoral artery diameters in women.

### Microcirculation and differential function improvement after TAVI

Both arms of the Partner trials demonstrated sustained improvements in functional class, aortic valve area and mean gradient following TAVI. Jin et al.\(^{51,54}\) documented early favourable changes in incoordination (asynchrony), myocardial wall stress and power following surgical AVR for AS with or without coronary artery disease. Davies et al.\(^{55}\) studied coronary microcirculation using invasive arterial wave intensity analysis in 11 patients undergoing TAVI (Sapien n=10). Their findings support improved coronary reserve after TAVI as documented by increasing coronary diastolic suction wave with rising heart rate, consistent with relief of angina symptoms with unobstructed coronary arteries.

Differential changes in longitudinal, circumferential and radial function are known to occur with progression of aortic stenosis even with preserved global ejection fraction. These changes are successive compensatory mechanisms to wall strain and pressure rise, and normalise following both surgical and transcatheter aortic valve replacement. In contrast, RV function remains preserved after TAVI compared to surgical AVR; Zhao et al.\(^{56}\) postulated RV myocardial preservation, aortic cross clamp, or right atrial cannulation during open chest cardiopulmonary bypass as putative mechanisms.

### Modelling for TAVI and the Heart Team approach

Successful outcomes for TAVI procedures depend on careful patient selection, optimal device implantation, and post-procedural high dependency care. The multidisciplinary team approach is critical for each of these stages. Multi-modality imaging\(^{47,57-59}\) employing catheter angiogram, echocardiographic, multi-detector CT and cardiac MRI techniques is becoming the standard approach for anatomical modelling prior to TAVI. Transthoracic and transoesophageal echocardiograms underestimated aortic annulus dimensions due to the limitation of imaging in a single plane thereby contributing to annulus-prosthesis mismatch and hence paravalvular AR.\(^{51,57,58}\) Thus, cardiac CT has become thefavoured standard for annular sizing. In native TAVI patients, cardiac MRI was reported by Jabbour et al.\(^{17}\) to be reliable and equally reproducible to CT and superior to TTE. Furthermore, Quail et al.\(^{59}\) showed that cardiac MRI was comparable to CT for aortic dimensions and coronary ostia height for valve-in-valve TAVI procedures. Table 4 illustrates agreement between CT, MRI and TTE measurements.

### Table 4: Precision of aortic annulus measurements across modalities.

<table>
<thead>
<tr>
<th>Maximum Ao diameter</th>
<th>CT v CMRI</th>
<th>CT v CMRI</th>
<th>CT v TTE</th>
</tr>
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<tbody>
<tr>
<td>Mean bias ± sd (mm)</td>
<td>0.39 ± 2.54</td>
<td>4.52 ± 3.29</td>
<td>4.10 ± 3.2</td>
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</table>

Willson et al.\(^{62}\) and Jilaihawi et al.\(^{64}\) have shown the importance of aortic annulus dimensions and calcium burden on predicting TAVI outcomes for paravalvar AR. Willson showed that the absolute difference between TAVI prosthesis size and pre-procedural annulus measurements by CT and transoesophageal echocardiograms (TOE) predicted paravalvar AR at discharge. By receiver operating characteristic (ROC) the area under the curve (AUC) for CT was 0.81, 95% CI 0.68–0.88 and for TOE was 0.70, 95% CI 0.51 – 0.88, making CT a better predictor of paravalvular AR outcome than TOE. Furthermore, the incidence of paravalvular AR was strikingly higher for undersized TAVI prostheses – paravalvular AR incidence 2.2% vs 21.4% for oversized >1mm vs. undersized 1mm (odds ratio 9.4, 95% CI 2.15 – 88.8, p<0.05). Jilaihawi used retrospectively gated CT cross-sectional and multi-planar reconstructions in 40 patients compared to TOE measurements in 96 TAVI patients, to predict per-procedural paravalvar AR as an outcome. CT was superior to TOE for predicting paravalvular AR by maximum annulus diameter, ROC AUC of 0.82 (95% CI 0.7 – 0.94), p<0.001. Applying the CT sizing rule prospectively successfully predicted paravalvular AR after TAVI (mild+ paravalvar AR 7.5% vs 21.9%, p=0.045). Furthermore, a simplified LVOT calcium score was a powerful risk predictor for paravalvular AR exceeding that of annulus mismatch (Odds ratio for LVOT calcium= 9.1, 95% CI 1.6 – 50, p=0.02, v OR for annulus mismatch =1.6, 95% CI 1.3 – 2, p<0.001).

### Indications for TAVI

Although there has been concern of ‘indication creep’ for TAVI procedures, this is unlikely to be the case at present for several reasons. Total TAVI procedure volumes tend to increase but surgical AVR procedures remained unchanged, owing to increased pickup and constant rate of refusal for surgical AVR in new AS patients. This compares favourably to the German Aortic Replacement GARY registry. The GARY registry reported at the recent ESC congress 2012 that of 13,590 total AVRs, there were 3875 TAVI procedures, thus TAVI volume accounted for 28.5% of all AVR procedures.

### Cost-effectiveness of TAVI

Cost-effectiveness data from the Partner 1A series\(^{59}\) documented similar outcomes for cost and QALY (Quality-Adjusted Life Years) between TAVI and high-risk surgical AVR patients. However, sub-group analysis comparing transfemoral and transapical approaches favoured transfemoral-TAVI over surgical AVR for cost-effectiveness and QALYs; whereas transapical-TAVI was economically advantageous in only 7.1% of cases\(^{60}\). However, given the higher rate of pacemaker implantation and vascular complications with TAVI, overall cost and hospital stay calculations may prove less favourable in TAVI patients experiencing complications.
Endocarditis and TAVI

Endocarditis after TAVI remains low, reported incidence varying from 2.3 to 3.4%61, 62. This compares favourably with that reported after percutaneous pulmonary valve replacement63. However, the increased incidence of paravalvular AR complicates assessment of regurgitation attributable to infection, and isolated case reports document involvement of valves other than the original neo-aortic prosthesis.

Conclusion

Transcatheter aortic valve implantation has improved survival and symptom relief for inoperable and high risk aortic stenosis patients, but there remain important limitations. One-year mortality after TAVI compared favourably to conservative management of AS (21 – 30% vs. 51%), equating to a number needed-to-treat (NNT) of 5; with one- and two-year TAVI mortality outcomes that are not inferior to high-risk surgical AVR. TAVI outcomes demonstrate sustained relief of valvular obstruction, but significant paravalvular regurgitation remains an important weakness due to limited device sizes. Vascular complications and permanent pacemaker implantations are substantially increased after TAVI, with a trend towards higher stroke rates. Future developments in device choice and pre-implantation modelling may increase the efficacy and benefit from TAVI.

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References


