REVIEW

Low-gradient aortic valve stenosis: value and limitations of dobutamine stress testing

J Bermejo, R Yotti

Aortic valve stenosis has already reached endemic proportions in Western countries. As the prognosis of low-flow aortic valve stenosis under medical treatment is dismal, surgery is recommended in most patients. Preoperative dobutamine stress testing may help to assess surgical risk, but there is no strong scientific evidence to deny surgery based exclusively on the results of this test. The problems associated with clinical decision making in this condition are reviewed.

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A number of non-exclusive factors may account for such a high perioperative mortality in patients with a low pressure gradient. Firstly, for a similar degree of valvular obstruction, a lower pressure gradient may reflect more extensive myocardial damage and therefore worse tolerance to cardiac arrest surgery. Secondly, a lower pressure gradient before surgery blunts the afterload relief provided by the prosthetic valve, diminishing the benefit of surgery in terms of left ventricular end-systolic wall stress. Thirdly, despite the fact that their valve area may be $<1 \text{ cm}^2$, patients with a low pressure gradient may not have a major valvular obstruction to begin with.

Aortic valve stenosis has already reached endemic proportions in Western countries. Although population-based studies are scarce, its prevalence at the beginning of the last decade was estimated to be 2–5% in people older than 65.¹ Extrapolating this to European demographic data yields a prevalence of 3–7.5 cases per 1000 inhabitants. In addition, the aging of the populations of Western countries, the increase in population screening, and the ubiquity of echo scanning devices will turn aortic valve stenosis into a major health problem during the next decade. Degenerative aortic valve stenosis has already become the most common valvular disease treated surgically in Europe.² Noticeably, 19% of patients operated on for aortic valve stenosis in Europe show some degree of left ventricular systolic dysfunction at the time of surgery, and left ventricular ejection fraction is lower than 0.30 in 2.9% of patients who undergo surgery.

The Euro Heart Survey showed an overall surgical mortality of 2.7% for stand-alone aortic valve replacement and 4.3% for combined replacement and coronary revascularisation.² Recent single-institution series report a perioperative mortality in the range 8–18% if ejection fraction is below 0.30 or 0.35.^{3–5} Patients with severe aortic valve stenosis and a low ejection fraction often show a high preoperative transvalvular pressure gradient, with mean systolic values above 35 mm Hg. In these patients, left ventricular systolic function generally recovers after valve replacement, and surgery is clearly advocated.

Particularly challenging is the population showing a small valve area ($<1 \text{ cm}^2$), impaired systolic function, and a low mean pressure gradient ($<30 \text{ mm Hg}$). Contemporary series show that perioperative mortality may be as high as 21% in this population,⁶ and the pressure gradient independently correlates with surgical outcome in patients with aortic valve stenosis and systolic dysfunction undergoing aortic valve replacement.⁵

FLOW DEPENDENCE OF AORTIC VALVE STENOSIS: FACT OR FICTION?

This mechanism responsible for increased mortality is definitely possible because measurements of the severity of aortic valve stenosis are known to be modified by flow rate. This phenomenon, named “flow dependence of aortic valve stenosis”, is crucial in this context, as it may be responsible for an overestimation of the severity of the disease in the presence of a low transvalvular flow rate.

Although a number of different haemodynamic indices of aortic valve stenosis are available, aortic valve area is believed to be the most robust index in terms of true haemodynamic significance. However, in terms of clinical efficacy, non-flow-corrected indices such as the pressure gradient and the left ventricular stroke work perform better than valve area in predicting outcome even in the presence of impaired left ventricular systolic function.^{5–7} The improved predictive value of non-flow-corrected indices may be due to the fact that they integrate information not only on valvular obstruction, but also on left ventricular function and peripheral adaptation to aortic valve stenosis. However, preoperative pressure gradients correlate better with the weights of the excised stenotic valves⁸ than do the calculated valve areas. It is arguable whether valve weight is a reliable measure of aortic valve stenosis, but this finding undoubtedly questions the accuracy of valve area as an index of severity in unselected patients.

Nevertheless, according to flow-dynamic principles, the haemodynamic impact of the outflow obstruction is best assessed in terms of aortic valve area, and valve area is well known to be influenced by flow rate. Flow dependence of aortic valve stenosis is usually explained by incomplete

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Abbreviations: EOA, effective orifice area; GOA, geometric orifice area

anatomical valve opening because of a very low flow-mediated opening force. To illustrate this phenomenon, the valve is often exemplified as a door that never reaches its full aperture because the “pushing force” exerted by the ventricle is too weak. This situation, in which valve area is small but anatomical valvular damage is slight or even absent, is named “relative” or “pseudo-severe” aortic valve stenosis. To identify relative aortic valve stenosis, low-dose dobutamine stress testing is usually advocated. If inotropic stimulation causes transvalvular flow to rise, and measurements of aortic valve area increase in parallel, relative aortic valve stenosis is diagnosed and surgery is not recommended. Readers are referred to two excellent recent reviews^{9,10} and practice guidelines¹¹ covering the technical aspects and the interpretation of dobutamine testing in the context of low-flow aortic valve stenosis.

Valve area is a generic term that accounts for different haemodynamic concepts. Anatomic or geometric orifice area (GOA) accounts for the size of the cross-section left by the opened valve leaflets, as measured by planimetry using transoesophageal echocardiography or magnetic resonance. Remarkably, GOA is relatively unmodified by flow interventions.¹² Effective orifice area (EOA) accounts for the smallest cross-sectional area of the aortic jet (the area of the vena contracta), which typically develops a few millimetres distal to the valvular plane. The ratio between EOA and GOA defines the contraction coefficient, which is typically in the range 75–90% in aortic valve stenosis. In terms of valvular performance, it is the EOA and not the GOA that defines the burden related to aortic valve stenosis, because a large GOA is useless if the anatomic orifice cannot be fully occupied by the ejection flow. In terms of the total haemodynamic burden imposed on the left ventricle and its impact on the cardiovascular system, aortic valve stenosis is best defined in terms of the “energy loss coefficient”.¹³ This index corrects EOA for the fact that pressure recovery takes place in the ascending aorta, therefore causing the final pressure gradient to be lower than the net pressure decrease measured at the level of the vena contracta. However, because the relation between the EOA and the energy loss coefficient is relatively fixed in a given patient, flow dependence of aortic valve stenosis is assessed by analysing, either in vivo or in vitro, flow-mediated changes in EOA.

The fact that EOA increases parallel to flow rate in a particular patient is a well-established issue. Flow-mediated changes in EOA take place not only when flow rate is depressed, but also when output is in the normal or even the high range.⁷ Classically, flow dependence of aortic valve stenosis is believed to be almost exclusive of calcific-degenerative aortic valve stenosis,¹⁴ but there is only limited evidence supporting this.¹⁵

Flow-mediated changes in aortic valve area can be explained by two mechanisms: (1) true changes in orifice area (ie maximal leaflet excursion); (2) artefacts in the methods used to estimate valve area. The two mechanisms probably coexist to some degree in a particular patient.

During the last decade, a number of potential sources of error (artefacts) have been recognised to be responsible, at least to some extent, for the flow-mediated changes observed in EOA (table 1). The most extensively studied is the flow dependence of the discharge coefficient present in the Gorlin formula.¹⁶ Recently, errors in the measurement of the systolic ejection period and mean pressure gradient from pressure tracings have also been recognised as responsible for some previously reported flow-mediated changes in catheterisation-derived measurements of valve area.¹⁷ Consequently, cardiac catheterisation can no longer be considered the “gold standard” for measuring EOA.

EOA measured by Doppler echocardiography and the continuity equation is also flow-dependent.^{15,18} Although often explained as a demonstration of flow-mediated changes in GOA,¹⁸ the finding of flow-mediated modifications of EOA of rigid metal valve simulators¹⁹ clearly shows that EOA can change without any modification of leaflet excursion. Initially, flow-mediated changes in the non-flat profile of the aortic valve stenosis jet were identified in simulation studies.²⁰ However, a very recent in vitro study has provided further insight into flow dependence of aortic valve stenosis. Using an original method based on particle image velocimetry, Kadem *et al*.²¹ have shown that, in the presence of low transvalvular flow rates, reduced kinetic energy is insufficient to break the vortices that develop at the sides of the aorta. The ejection jet therefore becomes “squeezed” by the vortices, and the vena contracta is narrowed. When flow rate is increased, the high kinetic energy is able to break the vortex structures, and EOA rises in parallel.

If confirmed in the clinical setting, these observations have two important implications. Firstly, there would definitely be no role for anatomic measurements of GOA because the contraction coefficient is modified by flow rate. Secondly, and most importantly, there would not be much of a role for dobutamine testing for assessing valve dynamics. Augmentation of the EOA during inotropic stimulation would not necessarily reflect a truly flexible valve but a change in the size of the EOA through the formation of a large and well-established flow jet. The EOA measured at rest would be the value that accounts for the true haemodynamic burden imposed by aortic valve stenosis on the LV. Obviously, these important findings of this in vitro study require further assessment in the clinical setting. Whether this type of behaviour can also be found in only mildly stenotic valve should be ascertained. Furthermore, the potential impact of valve replacement in modifying this new type of “functional” aortic valve stenosis needs to be investigated.

Older studies postulated that aortic valve resistance may be more stable under changing haemodynamic conditions than EOA in patients with low-flow aortic valve stenosis, and it was proposed that valve resistance should guide clinical decision-making in patients with aortic valve stenosis.²² However, careful in vitro and in vivo experimental studies of the flow dynamics of aortic valve stenosis have shown that the pressure–flow relationship in aortic valve stenosis is squared (as implicit in the EOA formula) instead of linear (as implicit in the valve resistance formula) and that the model of flow resistance across a tubular conduit does not reflect valvular aortic valve stenosis.^{19,23} The greater stability observed for valve resistance under changing flow rates¹⁵ simply means that EOA is actually being modified by flow rate.

DECISION-MAKING BASED ON DOBUTAMINE STRESS

Before further considerations, it is important to clarify at what risk we are placing a particular patient if the surgical option is dismissed. The best information on this issue comes from a retrospective propensity-matched study by Pereira *et al*.²⁴ In a well-selected patient group with valve area ≤ 0.75 cm² and a mean pressure gradient ≤ 30 mm Hg, 4-year survival of patients not operated on was less than 15%, whereas survival of patients undergoing valve replacement was 78%. Despite the two patient groups being well matched for similar propensity scores for undergoing valve replacement, it is likely that these populations are not truly comparable. However, additional observational studies confirm that the 4-year survival rate of unselected patients with low-gradient aortic valve stenosis who do not undergo surgery is less than 20%.²⁵

Five non-randomised observational studies have related mid-term patient outcome to the results of dobutamine stress tests

Table 1 Some of the reported mechanisms of flow dependence of effective aortic orifice area (EOA) measured by either Doppler echocardiography or catheterisation

Mechanism	Reference
Doppler echocardiography	
Absence of a flat flow profile at the LVOT	20
Variable coefficient contraction of the vena contracta	20
Catheterisation	
Variable discharge coefficient	16
Inaccurate measurement of the ejection period	17
Both	
Variable timing of valve opening	35
Changes in EOA due to vorticity at low flow	21

LVOT, left ventricular outflow tract.

used to guide the indication of valve replacement in patients with a small EOA and a low pressure gradient.^{25–29} Importantly, exact information about how the dobutamine test data were integrated for clinical decision-making is lacking in most of these studies. Criteria on how relative aortic valve stenosis was defined depending on the dobutamine response are also not uniform. In general, cutoff values used to establish the diagnosis of relative aortic valve stenosis are an increase in EOA greater than 0.3 cm², and/or an EOA above 1.0 or 1.2 cm² at peak dobutamine dose. Recently, more sophisticated criteria to identify relative aortic valve stenosis have been proposed.³⁰

The first series on the use of dobutamine testing in low-flow aortic valve stenosis reported an incidence of relative aortic valve stenosis of 5/18 (28%).²⁶ None of these patients were operated on, and only one of them died during 1 year follow-up. According to these findings, and based on the belief that a true flexible valve is behind the finding of a relative aortic valve stenosis response, it is mostly accepted that patients who show this behaviour are unlikely to benefit from valve replacement.¹¹ However, when outcome of patients with relative aortic valve stenosis is carefully analysed, prognosis is much worse than originally believed. Zuppiroli *et al*²⁹ identified relative aortic valve stenosis in 10 of 48 patients with low-gradient aortic valve stenosis according to their response to dobutamine. Surgery was not performed on any of these patients, and after a mean follow-up of 2 years, seven of them (70%) had died (A Zuppiroli, personal communication). Slightly lower mortality has been reported in other series, but pooled data show that 2-year mortality of patients with relative aortic valve stenosis is close to 50% (table 2; fig 1). This figure doubles the expected mortality of non-selected patients with similarly advanced heart failure and severe systolic dysfunction undergoing standard medical treatment, as typically found in the control group of multicentre trials in this type of patient.³¹ Patient populations are, obviously, not comparable, and more advanced comorbidity (particularly coronary artery disease) may be present in patients who also have moderate aortic valve stenosis. However, according to the fluid-dynamic concepts discussed above, the assumption that the pseudo-severe stenotic aortic valve acts as an “innocent bystander” in patients with relative aortic valve stenosis may need to be revised. Only one patient with relative aortic valve stenosis undergoing simultaneous valve replacement and coronary revascularisation has been reported in the literature, and the patient was in functional class I after 14 months of follow-up.²⁵

It is possible that valve replacement of a theoretically “moderate” aortic valve stenosis can be beneficial in patients with severe left ventricular dysfunction, because these ventricles are particularly sensitive to increased afterload. Although there are no direct data on this, it is known that moderate patient–prosthesis mismatch (a postoperative equivalent to

moderate native aortic valve stenosis in terms of afterload) has no effect on perioperative mortality in patients with normal left ventricular systolic function, but a major effect on mortality in patients with poor ventricular function.³² On the basis of this evidence, we believe that identifying relative aortic valve stenosis as a result of a dobutamine test is not an absolute contraindication to valve replacement. Obviously, another consequence of this observation is that even moderate patient–prosthesis mismatch (prosthesis EOA ≤ 0.85 cm²) must be avoided if patients with aortic valve stenosis and severe left ventricular dysfunction eventually undergo valve replacement.

However, identifying patients with relative aortic valve stenosis is not the major aim of the dobutamine test in patients with low-flow aortic valve stenosis. The most universally accepted indication for dobutamine testing in such patients is to identify the existence of a contractile reserve to assess perioperative risk. For this objective, the criteria for defining contractile reserve is best established as a 20% increase in stroke volume from baseline to peak dobutamine dose. In the largest study published to date, Monin *et al*²⁵ analysed mid-term follow-up data from 136 patients with EOA ≤ 1 cm² and a mean pressure gradient ≤ 40 mm Hg enrolled at six institutions. When left ventricular contractile reserve was present, 6-year survival after valve replacement was above 75%. When contractile reserve was absent, prognosis was much worse. Kaplan–Meier analysis of patients without a left ventricular contractile reserve showed that the 2-year survival rate was above 35% in patients undergoing surgery, more than double the 15% survival observed for patients who were not operated on ($p = 0.07$). More importantly, both contractile reserve and the indication of valve surgery were independent predictors of mid-term survival in the Cox multivariate proportional hazards analysis.²⁵ In fact, the hazard ratio of valve replacement (0.30; 95% confidence interval 0.17 to 0.53) was lower than the hazard ratio of left ventricular contractile reserve (0.40; 95% confidence interval 0.23 to 0.69). Similarly in the study of Zuppiroli *et al*²⁹, valve replacement, and not the presence of contractile reserve, was an independent predictor of long-term survival. Consequently, the absence of contractile reserve should not be taken per se as a contraindication to surgery.³³

ADDITIONAL THERAPEUTIC CONSIDERATIONS

Although it may be better than medical treatment, it is true that a 35% 2-year survival rate of patients without left ventricular contractile reserve who undergo surgery is not good. Thus, alternative strategies should be considered. Cardiac transplantation may be a better option in these patients, if there is no contraindication because of comorbidity and the technique is available. If dobutamine stress testing shows contractility reserve, valve replacement should be preferred, because the survival rate of surgery in this group surpasses the actuarial survival rate of cardiac transplantation; if contractility reserve is absent, transplantation is a better option. Importantly, patients with a wide QRS complex may benefit from cardiac resynchronisation therapy, whatever surgical decision is taken.

CONCLUSIONS AND FUTURE RESEARCH

Valvular aortic stenosis associated with severe left ventricular systolic dysfunction and a low pressure gradient is a rare but particularly challenging situation. As the prognosis of low-flow aortic valve stenosis under medical treatment is dismal, surgery is recommended in most patients. Associated comorbidity, patient's preferences, and local institution surgical results should definitely be taken as strong determinants of the final decision. If cardiac surgery is being performed for associated valvular or ischaemic heart disease, the patient has severe left

Table 2 Incidence and outcome of patients with relative or pseudo-severe aortic valve stenosis, reported in studies of patients with low-flow, low-gradient aortic valve stenosis undergoing a dobutamine stress test

Reference	Inclusion criteria	Stress methodology	Criteria for relative aortic valve stenosis	Incidence	Follow-up (months)	Mortality
deFilippi <i>et al</i> ⁶⁶	EF ≤ 0.45 , mean G ≤ 30 mm Hg, EOA ≤ 0.5 cm ² /m ²	Echo, DBT up to 20 μ g/kg/min	Δ EOA ≥ 0.3 cm ² ; Δ WMSI $>20\%$	5/18 (28%)	12	20%
Schwammenthal <i>et al</i> ⁶⁸	EF ≤ 0.40 , mean G ≤ 40 mm Hg, EOA ≤ 1 cm ²	Echo, DBT up to 20 μ g/kg/min	Δ EOA ≥ 0.3 cm ² ; final EOA >1 cm ²	8/24 (30%)	11	25%
Nishimura <i>et al</i> ⁶⁷	EF ≤ 0.40 , mean G ≤ 40 mm Hg, EOA ≤ 1 cm ²	Cath, DBT up to 40 μ g/kg/min	Δ EOA >1.2 cm ² ; final mean G <30 mm Hg	7/32 (22%)	32	57–100%*
Monin <i>et al</i> ⁶⁵	CI ≤ 3.0 L/min/m ² , EOA ≤ 1 cm ² , mean G ≤ 40 mm Hg	Echo, DBT up to 20 μ g/kg/min	Δ EOA ≥ 0.3 cm ² ; final EOA >1 cm ²	7/136 (5%)	19	50%†
Zuppiroli <i>et al</i> ⁶⁹	EF ≤ 0.40 , mean G ≤ 30 mm Hg, EOA ≤ 1 cm ²	Echo, DBT up to 20 μ g/kg/min	Δ EOA ≥ 0.25 cm ²	10/48 (21%)	24	70%
Pooled				38/258 (15%)	20	48–57%

EF, left ventricular ejection fraction; CI, cardiac index; mean G, mean systolic transvalvular pressure gradient; EOA, effective orifice area; DBT, dobutamine; WMSI, wall motion score index; Δ , change from baseline to peak dose.

*Mortality is in this range, but the exact number is unavailable from the reported data.

†Mortality calculated excluding one patient finally undergoing valve replacement.

ventricular dysfunction, and aortic valve area is ≤ 1 cm², aortic replacement should always be performed.

Preoperative dobutamine stress testing may help to assess surgical risk, but there is no strong scientific evidence to deny surgery based exclusively on the results of this test. If left ventricular contractile reserve is identified, valve replacement must be encouraged. However, if contractile reserve is absent, there is still a potential benefit of surgery. Although perioperative mortality is very high in patients without contractile reserve, valve replacement improves prognosis in these patients if cardiac transplantation is not an option. According to recent evidence on the fluid dynamics of low-flow aortic valve stenosis, the relative or pseudo-severe aortic valve stenosis response to inotropic stimulation is not necessarily synonymous with a flexible valve. The best alternative for patients showing this behaviour is not clear, but the prognosis of patients on medical treatment is far worse than originally believed. Further multicentre studies involving a much greater number of patients are definitely warranted.

Imaging modalities for accurately assessing irreversible myocardial dysfunction are evolving rapidly, and preliminary data suggest that tissue characterisation techniques could be helpful in the context of low-flow aortic valve stenosis.³⁴ More importantly, greater efforts must be directed to prevent this end-stage situation of low-flow aortic valve stenosis. Careful echocardiographic screening in patients with a systolic

murmur, and close follow-up of patients with known asymptomatic aortic valve stenosis are probably the best available strategies for this purpose.

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Figure 1 Pooled data on mortality of patients with low-flow, low-gradient aortic valve stenosis undergoing a dobutamine stress test for clinical decision-making.^{25–29} Outcome is shown for patients referred for medical and surgical treatment. Numbers represent the number of patients in each group.

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IMAGES IN CARDIOLOGY

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Visualisation of a St Jude prosthetic mitral valve using electron beam tomography

A 58-year-old male patient, with a history of mitral valve replacement and coronary artery bypass surgery 10 years earlier, was treated with a cardiac resynchronisation therapy and defibrillator (CRT-D) device because of poor left ventricular function, left bundle branch block and first-degree atrioventricular block. However, during this procedure a dissection of the coronary sinus occurred. A second attempt to re-implant a ventricular lead of his CRT-D was planned. Two days before this intervention, a contrast-enhanced dual slice electron beam tomographic scan (e-Speed, Imatron, San Francisco, California, USA) was performed to visualise the coronary sinus. The coronary sinus showed a normal calibre and probably a dissection flap proximally.

Moreover, the scan showed the stainless St Jude 31 prosthesis between the left ventricle and the left atrium (panels A, B). A high concentration of contrast is visible in the right atrium. A three-dimensional image of the valve in systole was made using volume rendering (panel C). The orifice area was planimetrically calculated to be 2.4 cm² (mean of three measurements: 2.4, 2.4 and 2.5 cm²; panel D). Echocardiography routinely performed 5 months earlier measured a mitral valve area of 2.4 cm², in accordance with our findings. In conclusion, it is possible to visualise St Jude prosthetic valves with electron beam tomography. Electron beam tomography may be a suitable technique to evaluate some functional aspects of mechanical prosthetic valves.

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