Determinants of symptoms and exercise capacity in aortic stenosis: a comparison of resting haemodynamics and valve compliance during dobutamine stress

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Aims Valve compliance might determine the onset of symptoms better than resting measures of aortic stenosis. This study compared valve compliance measured by dobutamine stress echocardiography with resting haemodynamic variables against the endpoint of symptoms at low workload during exercise testing.

Methods and results Echocardiography was performed at rest and during each stage of a dobutamine stress test in 65 asymptomatic patients with moderate or severe aortic stenosis. Each patient also completed a modified Bruce treadmill exercise test. During dobutamine stress, peak transaortic velocity increased by 1.0 (0.4) m/s and effective orifice area by 0.25 (0.22) cm². Valve compliance was 0.23 (0.10) cm²/100 ml.s⁻¹, and was independent of baseline effective orifice area. In the 19 patients limited by symptoms on exercise testing, valve compliance was significantly lower (0.19 (0.09) cm²/100 ml.s⁻¹, p=0.03) than in those who remained asymptomatic (0.25 (0.10) cm²/100 ml.s⁻¹, p=0.03). Effective orifice area at peak stress was also lower (1.0 (0.3) vs 1.2 (0.4) cm², p=0.03), but there were no significant differences in resting measures of effective orifice area, transaortic velocity, or mean pressure drop.

Conclusions Effective orifice area is flow-dependent in patients with moderate and severe aortic stenosis with preserved left ventricular function. Exertional symptoms are better predicted by compliance than resting effective orifice area, mean pressure drop or peak transaortic velocity.

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KEYWORDS
Aortic stenosis; Valve compliance; Stress echocardiography

Introduction
For measures of aortic stenosis to be clinically useful, they should correlate with independent clinical variables, for example mortality, surgical rate, symptoms, or exercise tolerance.1–3 Symptoms occur during exercise and, furthermore, both geometric and effective orifice area may be affected by changes in flow.4–8 It is therefore possible that measures assessed during stress studies will be more useful than measures made at rest. The flow-dependency of effective orifice area can be expressed as valve compliance, defined as the slope of the regression line when effective orifice area is plotted against flow. A valve with a high compliance has a greater effective orifice area.
with increased flow than does a valve with a low compliance. Prospective data suggest that patients with higher valve compliance are less likely to require valve replacement over a follow-up period of 30 months. However, valve replacement is not always an accurate clinical end point. Whilst surgery is not usually recommended in asymptomatic aortic stenosis, it may be performed for patients with non-specific symptoms and severe stenosis. Exercise testing reveals symptoms in a significant proportion of patients with aortic stenosis who claim to be asymptomatic and these symptoms can predict outcome. Exercise testing may provide a more reliable end-point than selection for valve replacement.

We therefore compared valve compliance, measured by dobutamine stress echocardiography, and resting haemodynamic variables against the end-point of symptoms at low workload during exercise testing.

**Methods**

**Patients**

A total of 86 consecutive asymptomatic patients with aortic valve leaflet thickening and moderate or severe aortic stenosis defined as an effective orifice area of 1.2 cm² or less attended a cardiac tertiary referral centre between June 1998 and July 2000 for transthoracic echocardiography. All were asymptomatic and had normal left ventricular systolic function (defined as a fractional shortening of >28% and no regional wall motion abnormality), no more than grade 1/4 aortic regurgitation and no other significant valve disease. Of these, 16 patients declined and five were unable to exercise. This left a study group of 65 subjects, median age 69 years (range 27 to 81) of whom 48 were male and 17 female.

Aortic stenosis was graded using the continuity equation calculated at rest as moderate (effective orifice area 0.8 to 1.2 cm²) or less or severe (effective orifice area <0.8 cm²). No patient had commissural fusion or associated rheumatic disease of the mitral valve. None had more than mild aortic or mitral regurgitation. Coronary angiography was performed in 20 patients, of whom four had significant coronary disease defined as a stenosis of at least 50% in one or more arteries.

The study received approval from the local research ethics committee and all subjects gave written informed consent.

**Echocardiography**

Subjects stopped beta-blocker medication 48 h before and fasted for at least 3 h before the procedure. An ATL HDI 3000 (Seattle, Washington, USA) was used with a 3-2 20 mm duplex probe and 1.9 MHz continuous wave stand-alone probe. M-mode recordings were made at a level immediately apical to the tips of the mitral valve leaflets and end-diastolic measurements were made using the American Society of Echocardiography convention. Fractional shortening was calculated as the systolic decrease in left ventricular short axis divided by the end-diastolic diameter. The sub aortic diameter was measured from inner edge to inner edge at the level of the base of the aortic cusps in a parasternal long axis frame frozen in systole, and the average of three estimates taken. Where possible, the number of aortic valve cusps was counted in a parasternal short axis view and calcification of each leaflet scored from 0 (no thickening) to 3 (dense calcification). Pulsed Doppler recordings were made in the apical 5-chamber view with the sample volume moved axially from the level of the aortic annulus until a clear non-aliased signal was obtained, usually 0.5 to 1 cm below the valve. The signal was traced to obtain peak velocity, velocity time integral and mean pressure difference using the online software. Continuous wave recordings were made from the apex and right intercostal positions and the optimal signal was traced to obtain peak velocity, velocity time integral, systolic ejection time and mean pressure difference using the on-line software. The average of three pulsed or continuous wave signals was taken.

Dobutamine was infused intravenously from 5 µg/kg/min with increments of 5 µg/kg/min in 5-min stages to a maximum of 40 µg/kg/min. The peak dobutamine dose was 10 µg/kg/min in two patients, 15 µg/kg/min in 21 patients, 20 µg/kg/min in 28 patients, 30 µg/kg/min in 11 patients and 40 µg/kg/min in three patients. The dobutamine infusion was stopped before 40 µg/kg/min because of a sustained fall in heart rate (n=35) aliasing of the outflow tract Doppler signal (n=10), symptoms alone (n=7), a fall in peak transaortic pressure difference (n=5), sustained hypotension (n=3) and cardiac dysrhythmias (n=2) including asymptomatic sustained atrial fibrillation requiring pharmacological cardioversion in one patient. There were no other complications.

A 12-lead electrocardiogram was monitored continuously throughout the procedure and blood pressure was recorded at the end of each stage and
if a subject reported any symptom. Pulsed and continuous wave recordings were repeated after 3 min of each stage. Measurement of left ventricular outflow tract diameter was not repeated at each stage, but this has been shown not to change significantly during dobutamine stress.6

Analysis
Pulsed and continuous wave Doppler traces were analysed off-line. Effective orifice area (EOA in cm²) was calculated by the classical continuity equation at rest and for each stage:

\[
EOA = \frac{CSA_{LVOT} \times VTI_{LVOT}}{VTI_{Ao}}
\]

Where \( CSA_{LVOT} \) is the cross sectional area of the left ventricular outflow tract (in cm²), calculated assuming circular geometry:

\[
CSA_{LVOT} = \frac{\pi D_{LVOT}^2}{4}
\]

Stroke volume (SV in ml) and transaortic flow (in ml/s) were calculated as follows where SET is systolic ejection time (in ms):

\[
SV = CSA_{LVOT} \times VTI_{LVOT}
\]

Flow = \( \frac{SV}{SET} \) 1000

Cardiac output (CO in l/min) was calculated as the product of stroke volume and heart rate.

Mean pressure drop was calculated in mmHg by the Bernoulli equation as the difference between aortic and outflow tract mean pressure drop.

A mean valve calcification score was calculated by dividing the total calcification score by the number of cusps.

Valve compliance
Effective orifice areas were plotted against flow at each stage of dobutamine stress for each case and a regression line was fitted to the plot where possible. Valve compliance in cm²/100 ml.s⁻¹ was calculated directly from the slopes of the regression lines and effective orifice area at zero flow was calculated from the intercept of the regression line.

Exercise testing
Exercise testing was performed using a Quinton Q55xt treadmill and Q5000 monitor (Washington, USA) according to American College of Cardiology/American Heart Association practice guidelines.14 All subjects undertook a treadmill exercise test using a Bruce protocol modified by two warm-up stages,15 supervised by a technician who was unaware of the echocardiographic data and in the presence of a physician. Maximum workload was predicted from age and sex. Subjects were questioned for symptoms every 2 min and heart rate, blood pressure and a 12-lead electrocardiogram were recorded at baseline, peak and the end of each stage. The test was stopped prematurely upon significant limiting breathlessness/chest discomfort or dizziness. Only clear symptoms preventing continuation of exercise at less than 80% of predicted maximum workload were considered significant.16

Other predetermined criteria for cessation were ST segment depression of >5 mm measured 80 ms after the J point, more than three consecutive premature ventricular beats, and hypotension defined as a fall in systolic blood pressure of more than 20 mmHg from baseline,9 although in practice no test was terminated for any of these reasons. Otherwise the test continued until the patient was fatigued. Total exercise time in seconds and maximum ST depression in mm in a single lead at 80 ms after the J point during the test were recorded. ST depression of 1 mm or greater was considered significant. Maximal exercise capacity was estimated by calculating MET level from peak treadmill speed and gradient using on-line software.

Statistical analysis
Results were expressed as mean (standard deviation) for normally distributed data or median [lower quartile, upper quartile] for skewed data. Echocardiographic variables at rest and peak dobutamine dose were compared using the paired t test or Wilcoxon signed-rank test for skewed data. The unpaired t test or the nonparametric Mann–Whitney U-test for skewed data were used to compare patients with and without limiting symptoms and patients with moderate or severe aortic stenosis. Categorical variables were compared using the chi-squared test or Fisher’s exact test. Associations of continuous variables were assessed by Pearson correlation. Interobserver and intraobserver measurement variability of off-line Doppler analysis was assessed by Pearson correlation and mean differences between two independent observers and repeated measurements by a single observer.

Statistical analysis was performed using commercial software (GB-STAT V8.0: Dynamic Micro-
systems Inc, Maryland, USA and Stata Statistical Software Release 7.0: Stata Corp, Texas, USA).

**Results**

**Baseline and dobutamine stress echocardiography**

At rest, mean effective orifice area was 0.84 (0.21) cm$^2$ (Table 1) and the aortic stenosis was moderate in 36 and severe in 29 patients. Median transaortic flow and mean cardiac output were significantly higher in patients with moderate than with severe aortic stenosis (Table 2). Calcification scores could be calculated in 36 cases and were 2.00 (range 0.67 to 3.00).

At peak dobutamine dose, transaortic flow was higher than at rest in all patients. Heart rate and stroke volume were significantly higher, whilst ejection time was shorter and the resulting cardiac output was higher. Systolic and diastolic blood pressures were lower (Table 1).

Peak transaortic velocity and mean pressure drop increased in all patients. Effective orifice area increased at peak stress in 59 cases, decreased in five and was unchanged in one. The absolute change in orifice area in the whole population was 0.25 (0.22) cm$^2$ and was greater in moderate than severe stenosis although there was substantial overlap between the two groups (Table 2).

**Valve compliance**

Effective orifice area was plotted against transaortic flow for each individual patient. A linear, but not curvilinear, regression line fitted in 63 cases (Fig. 1 and Fig. 2). The points were too dispersed to plot a regression line for the remaining two

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**Table 1** Clinical, haemodynamic and echocardiographic variables at rest and peak dobutamine stress ($n=65$)

<table>
<thead>
<tr>
<th>Variable</th>
<th>Rest</th>
<th>Peak stress</th>
<th>$P$-value (paired $t$ test)*</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Clinical</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Heart rate (beats/min)</td>
<td>77 (16)</td>
<td>113 (21)</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Systolic blood pressure (mmHg)</td>
<td>145 (28)</td>
<td>129 (25)</td>
<td>0.0001</td>
</tr>
<tr>
<td>Diastolic blood pressure (mmHg)</td>
<td>84 (14)</td>
<td>63 (15)</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td><strong>Haemodynamic</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Stroke volume (ml)</td>
<td>68.6 (14.6)</td>
<td>73.9 (18.2)</td>
<td>0.001</td>
</tr>
<tr>
<td>Ejection time (msec)</td>
<td>308 (41)</td>
<td>220 (35)</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Transaortic flow (ml/s)</td>
<td>237 (76)</td>
<td>355 (106)</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Cardiac output (l/min)</td>
<td>5.4 (1.8)</td>
<td>8.4 (2.9)</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td><strong>Echocardiographic</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Peak aortic velocity (m/s)</td>
<td>3.7 (0.8)/2.5–6.1</td>
<td>4.7 (0.8)/3.3–6.5</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Mean pressure drop (mmHg)</td>
<td>33.2 (16.9)/12.7–100.6</td>
<td>46.7 (21.5)/17.6–125.8</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Effective orifice area (cm$^2$)</td>
<td>0.84 (0.21)/0.46–1.20</td>
<td>1.10 (0.35)/0.51–2.01</td>
<td>&lt;0.0001</td>
</tr>
</tbody>
</table>

*Mean (standard deviation)/range, paired $t$ test.

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**Table 2** Breakdown at rest and peak dobutamine stress by resting grade of stenosis

<table>
<thead>
<tr>
<th>Variable</th>
<th>Moderate $n=36$</th>
<th>Severe $n=29$</th>
<th>$P$-value ($t$ test)*</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Rest</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Flow (ml/s)</td>
<td>248 (43)</td>
<td>195 (37)</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Cardiac output (l/min)</td>
<td>5.7 (1.3)</td>
<td>4.6 (1.4)</td>
<td>0.003</td>
</tr>
<tr>
<td>Effective orifice area (cm$^2$)</td>
<td>1.01 (0.12)</td>
<td>0.65 (0.11)</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td><strong>Peak stress</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Flow (ml/s)</td>
<td>388 (87)</td>
<td>282 (58)</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Cardiac output (l/min)</td>
<td>9.0 (2.2)</td>
<td>7.3 (1.9)</td>
<td>0.002</td>
</tr>
<tr>
<td>Effective orifice area (cm$^2$)</td>
<td>1.31 (0.30)</td>
<td>0.83 (0.18)</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td><strong>Derived</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Change in area (cm$^2$)</td>
<td>0.31 (0.26)</td>
<td>0.18 (0.14)</td>
<td>0.013</td>
</tr>
<tr>
<td>% change in area</td>
<td>31 (28)</td>
<td>28 (21)</td>
<td>0.6</td>
</tr>
<tr>
<td>Valve compliance (cm$^2$/100 ml.s$^{-1}$)</td>
<td>0.24 (0.11)</td>
<td>0.22 (0.09)</td>
<td>0.41</td>
</tr>
</tbody>
</table>

*Mean (standard deviation), $t$ test.
patients. Mean valve compliance was 0.23 (0.10) cm$^2$/100 ml.s$^{-1}$ with no significant difference between patients with moderate or severe stenosis (Table 2). However, the regression line at zero flow was offset from zero by 0.40 cm$^2$ in moderate stenosis and 0.20 cm$^2$ in severe stenosis ($p<0.0001$).

Valve compliance was not related to resting effective orifice area, but there were weak inverse relations with resting peak transaortic velocity ($r=-0.33$, $p=0.007$) and mean aortic pressure drop ($r=0.32$, $p=0.01$). Valve compliance was not related to mean valve calcification score. The mean valve compliance of the seven patients who experienced symptoms during dobutamine stress was not significantly different from the remainder of the group (0.28 (0.12) vs 0.23 (0.10) cm$^2$/100 ml.s$^{-1}$, $p=0.25$).

For valve compliance, the interobserver correlation coefficient was $r=0.97$, and the mean difference was 0.036 cm$^2$/100 ml.s$^{-1}$. The intraobserver correlation coefficient was $r=0.83$, and the mean difference was 0.028 cm$^2$/100 ml.s$^{-1}$.
Exercise testing

All 65 patients performed the exercise test satisfactorily. Despite claiming to be asymptomatic, 19 patients (29%) stopped because of limiting symptoms: breathlessness or chest discomfort in 16 and dizziness in three. Four patients had minor symptoms at high workload and the remaining 42 experienced no symptoms and stopped because of fatigue. No exercise test was terminated for another reason and there were no complications. There were no significant differences in resting measures of aortic stenosis between patients with limiting symptoms and those without. Of those with symptoms, 11 (58%) had severe stenosis and 8 (42%) had moderate stenosis (p=0.17).

However, valve compliance was significantly lower in patients with limiting symptoms, at 0.19 (0.09) cm²/100 ml.s⁻¹, than in those without, at 0.25 (0.10) cm²/100 ml.s⁻¹ (p=0.03, Fig. 3 and Fig. 4). Among the 29 patients with severe stenosis, valve compliance was 0.20 (0.09) cm²/100 ml.s⁻¹ in those with symptoms and 0.24 (0.08) cm²/100 ml.s⁻¹ in those without, but this difference was
not statistically significant. For the 36 patients with moderate stenosis, valve compliance was 0.18 (0.08) and 0.26 (0.12) cm²/100 ml.s⁻¹ in respectively those with and without symptoms (p=0.047). Peak effective orifice area and the absolute increase in area from rest to peak were also slightly lower in patients with symptoms (Table 3). Patients with coronary artery disease were not more likely to have limiting symptoms.

Total exercise time was inversely related to age and directly related to effective orifice area but did not correlate significantly to other measures of stenosis including valve compliance. The overall MET level achieved was also related to resting and peak measures of area but not to valve compliance.

In 18 cases, systolic blood pressure either did not increase or fell during exercise. There were no differences in resting measures, valve compliance or peak effective orifice area between these patients and those showing a positive blood pressure response.

### Discussion

This is the first study to compare valve compliance with symptoms and exercise capacity in patients with isolated aortic stenosis and normal left ventricular systolic function.

### Valve compliance in aortic stenosis

Effective orifice area increased with transaortic flow in 59 of 65 patients with moderate or severe aortic stenosis. The mean increase was slightly greater in moderate (0.31 cm²) than severe stenosis (0.18 cm²) although the relative increases were similar. This is consistent with previous studies that demonstrated flow dependency of effective orifice area in patients with preserved left ventricular function as well as those with impaired left ventricular function provided that contractile reserve was maintained.

Like Bermejo et al., we found that valve compliance was not related to resting effective orifice area. This means that two valves could have the same effective area at rest, but potentially significantly different effective areas with increased flow induced by dobutamine or exercise.

### Mechanism of valve compliance

Greater valve compliance might be explained by a progressive increase in maximal geometric orifice area with flow or by a more rapid opening despite a constant maximal orifice area. There is evidence for both mechanisms and it is likely that both can occur together.

Increases in geometric orifice area were demonstrated by video recording of stenotic native aortic valves mounted in a pulse simulator. In contrast, a small study of 11 patients using transoesophageal echocardiography failed to show an increase in geometric orifice area with dobutamine stress. This may reflect technical limitations of two-dimensional planimetry of the calcific stenotic valve, which forms a relatively complex three-dimensional structure.
Using frame-by-frame planimetry, Arsenault measured aortic valve area at different points during the ejection cycle, and found that more severely stenotic valves opened and closed more slowly.\(^{23}\) Effective orifice area by the continuity equation correlated more closely with maximum than mean area measured by planimetry. The present study examined the relation between continuity area and mean systolic flow rate. A linear relation between area and flow was identified over a physiological range of flow in most patients regardless of the severity of stenosis. However, the linear area/flow regression lines were offset from zero by 0.4 cm\(^2\) in moderate and 0.2 cm\(^2\) in severe stenosis, suggesting that the relation is in fact non-linear at very low flow. This finding would be consistent with more rapid opening of the valve in less severe stenosis. The relationship between geometric valve area and effective orifice area may also be affected by non-valvar factors. Using a canine in vivo model of aortic stenosis, Bermejo also showed that pressure reversal occurred late in the ejection period, which could alter effective functional orifice area despite the maintenance of geometric area.\(^{20}\)

It is possible that the effect of flow is mediated by valve morphology. Shively showed that valves that were bicuspid or had significant commissural fusion on transoesophageal echocardiography were less compliant than calcific degenerative valves.\(^{6}\) The pattern of valvar calcification could influence compliance, with diffuse calcification of the cusp bodies associated with poorer compliance. In the present study, the degree of calcification did not relate to compliance, although the assessment of calcification by transthoracic echocardiography must be regarded as no more than semi-quantitative.

Clinical implications: the relationship of valve compliance to symptoms and exercise capacity

The effect of flow on effective orifice area is well documented in patients with impaired left ventricular function. However, even in those with a preserved left ventricle, the presence of high transaortic flow, for example as a result of anaemia, pain or fever may modify effective orifice area. The mean increase in effective orifice area during dobutamine stress was 0.25 cm\(^2\) in this study, sufficient to change the classification in several patients from severe to moderate or from moderate to mild if peak rather than resting calculations were used. It may, therefore, be inaccurate to grade aortic stenosis by resting haemodynamic measures alone.

It has been proposed that patients with limited valve compliance might develop exertional symptoms earlier in the progression of the condition.\(^{1,2}\) The results of this study confirm an association between aortic valve compliance and exercise-limiting symptoms. In models of aortic stenosis, left ventricular workload and energy loss are inversely related to valve area and directly related to flow.\(^{21,24}\) A failure of effective orifice area to increase during exercise would be expected to lead to a greater rise in left ventricular workload and poorer exercise tolerance than in a more compliant valve.

There can be discrepancies in the frequency of symptoms between moderate and severe aortic stenosis when defined in the conventional way by resting measures.\(^{25,26}\) In this study, valve compliance was lower in patients with symptoms on exercise testing. It was also lower in some patients with moderate rather than severe stenosis as calculated by the continuity equation at rest. This may partly explain these apparent discrepancies in symptoms between moderate and severe stenosis. However, valve compliance may be of less significance in the patients with the most severe stenosis at rest. Effective orifice area at peak stress also related more closely to exertional symptoms than did resting orifice area, although peak stress did not always correspond to peak transaortic flow.

Study limitations

Doppler measurements of pressure drop and effective orifice area in moderate and severe aortic stenosis are recognized as accurate and reproducible. However, technical difficulties in performing and analysing these measurements at higher flow rates may reduce this accuracy. This is likely to be the reason for an apparent drop in effective orifice area in spite of increased flow in eight patients. Furthermore, it was not possible to establish the relationship of effective orifice area to flow in four patients because of lack of data points and excessive scatter. This might be improved in subsequent studies by increasing the frequency of Doppler readings taken.

The assessment of symptoms during exercise testing can be subjective. To minimize this, only clearly limiting symptoms at less than 80% maximum predicted workload were accepted.\(^{15}\) Minor, non-limiting symptoms at high workload were not considered significant.
Safety of dobutamine stress in aortic stenosis

Dobutamine stress has been used safely at doses of up to 40 µg/kg/min to study flow-dependent haemodynamics in patients with moderate and severe aortic stenosis. However, dobutamine cannot exactly replicate the physiological effects of physical activity. In addition to chronotropic and inotropic effects, it can cause a reduction in peripheral vascular resistance theoretically leading to a fall in transaortic pressure drop at constant flow. This can cause a reduction in physical activity. In addition to chronotropic and inotropic effects, it cannot exactly replicate the physiological effects of physical activity.

Seven patients experienced significant symptoms at peak dobutamine stress but there were no significant complications. These symptoms were not predicted by resting measures of stenosis or related to valve compliance, but symptomatic patients tended to have greater falls in systolic and diastolic blood pressure, suggesting that the symptoms were caused in part by peripheral effects of dobutamine.

Satisfactory stress echocardiography was achieved at a dose of 20 µg/kg/min dobutamine or less in 51 cases. Above this level, aliasing of the outflow tract pulsed Doppler signal and a significant decrease in heart rate became more frequent. We would therefore recommend 20 µg/kg/min as the maximum dose for a stress study in asymptomatic aortic stenosis, with continuous heart rate monitoring and blood pressure recording at a frequency of at least once during each stage. However, dobutamine infusion should be stopped prior to this if significant symptoms or sustained falls in heart rate or systolic blood pressure occur.

Conclusions

This study confirms that effective orifice area is flow-dependent in patients with moderate and severe aortic stenosis and preserved left ventricular systolic function. Valve compliance is not predicted accurately by resting measures. Patients with and without symptoms on exercise testing were better distinguished by valve compliance than by resting effective orifice area, transaortic velocity or mean pressure drop. However, the significance of valve compliance requires prospective study against clinical outcome measures such as new symptoms during follow-up before stress echocardiography can be recommended in patients with aortic stenosis and normal left ventricular function.

References


