Echocardiographic Assessment of Left Ventricular Function in Aortic Valve Disease

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SUMMARY Echocardiography was used to study left ventricular size and contraction in 128 patients with isolated aortic valve disease — 45 patients with aortic stenosis, 25 with mixed aortic valve disease and 58 with aortic regurgitation. Left ventricular measurements included the end-diastolic internal dimension (LVIDd), mural thickness (PWTD); an index of circumferential myocardial contraction — fractional shortening (FS = ([LVIDd – LVIDs]/LVIDd) x 100) — and stroke volume (LVSV).

In the absence of left ventricular failure, measurements in aortic stenosis were characteristic of pressure overload with normal LVIDd and FS and an increase in PWTD related to the severity of stenosis; in aortic regurgitation, there was volume overload with increases in LVIDd and PWTD which were related to the severity of regurgitation, while FS was slightly reduced. In mixed aortic valve disease there was evidence of both pressure and volume overload. When left ventricular failure was associated with aortic stenosis, mixed aortic valve disease and chronic aortic regurgitation, FS was usually reduced. By contrast, in a recent patient with acute severe aortic regurgitation, FS was normal despite left ventricular failure, suggesting pump rather than myocardial failure.

ECHOCARDIOGRAPHY is proving to be a valuable addition to routine noninvasive clinical evaluation of patients with heart disease. The technique has been applied to estimation of the severity of left ventricular hypertrophy1 and of regurgitant lesions,2 3 and to the evaluation of left ventricular function.6 7

Previous reports have included small numbers of patients with aortic valve disease.2 8 This study is an analysis of our experience with the echocardiographic assessment of left ventricular function in aortic valve disease with particular reference to the manifestations of left ventricular failure and to the effects of acute aortic regurgitation.

Patients and Methods

All patients with isolated aortic valve disease who had been routinely studied by echocardiography over a three year period were considered for inclusion in this study, provided that the echocardiogram was technically adequate for measurement of left ventricular dimensions. Specifically excluded were patients with malfunction of a prosthetic aortic valve and those with other possible causes for left ventricular enlargement or impairment, such as ischemic heart disease or hypertension (systolic blood pressure > 180 mm Hg or diastolic > 100 mm Hg). Also excluded were two patients in whom aortic regurgitation was thought to be coincidentally associated with cardiomyopathy; the impairment of left ventricular function was severe and disproportionate to the severity of aortic regurgitation which was clinically trivial in one patient, and shown by cardiac catheterization and aortography to be only moderate in the other.

Of the 128 patients included in the study, 45 had aortic stenosis, 58 aortic regurgitation and 25 mixed aortic stenosis and regurgitation. Included were seven patients with severe acute aortic regurgitation and left ventricular failure.

Investigations

The correlation between angiographic and echocardiographic measurements of left ventricular dimensions has already been studied and found to be good.5 Furthermore, an important advantage of echocardiography as a noninvasive technique is that patients with mild and moderate heart disease can be studied as well as those with severe disease. Hence our aim was to document the effects on left ventricular size and contraction of aortic valve disease of any severity rather than to present data only for those studied by cardiac catheterization and angiography. Cardiac catheterization and angiography were clinically indicated in 56 patients and the remainder were assessed by noninvasive tests.

Cardiac catheterization, including aortic root angiography, was performed as discussed for patients with mitral valve disease6 with the exception that selective coronary angiography was undertaken in all patients with aortic valve disease.

The severity of aortic stenosis was graded as mild, moderate or severe according to the aortic valve area (>1.0 cm², 0.5 to 1.0 cm², <0.5 cm²). The calculation was made from the mean systolic pressure gradient across the aortic valve, left ventricular ejection time and cardiac output.10 If there was trivial aortic regurgitation, indicated by a faint regurgitant jet which rapidly disappeared, the classification was still aortic stenosis but the severity was graded according to the pressure gradient (<20 mm Hg, 20–70 mm Hg and >70 mm Hg).

The cineangiograms had not been calibrated for measurement of left ventricular volume, hence aortic regurgitation was graded as mild, moderate or severe by inspecting the angiogram and applying the following criteria of severity: the size and amplitude of contraction of the left ventricular cavity, the appearance of the regurgitant jet, opacification of the left ventricle and the rate of clearing of contrast from it.11 12

Mixed aortic stenosis and regurgitation was not graded for severity since the combined effects of stenosis and regurgitation could not be adequately assessed.

For those patients in whom cardiac catheterization was not clinically indicated, assessment of the severity of the
valve lesion was made from the clinical data and from non-invasive investigations. Thus the severity of aortic stenosis was determined from the rate of upstroke of the carotid pulse and from the length and shape of the systolic murmur, confirmed by recording the indirect carotid pulse tracing and phonocardiogram, respectively. The severity of aortic regurgitation was determined from the blood pressure and from clinical, phonocardiographic and apicescardiographic evidence of increased diastolic left ventricular filling; the size of the left ventricle and left atrium were assessed from the chest radiograph and electrocardiogram. If any doubt remained, cardiac catheterization was always performed.

Left Ventricular Failure

Patients with severe aortic stenosis, aortic regurgitation and mixed aortic valve disease were classified according to the presence or absence of clinical left ventricular failure, defined as pulmonary venous congestion manifest as a reduction in effort tolerance due to dyspnea, characteristic radiographic changes and often progressive cardiomegaly. Patients with mitral regurgitation have previously been classified in a similar manner and the rationale has been discussed. For patients with aortic stenosis, aortic regurgitation, and mixed aortic valve disease without left ventricular failure, studied by cardiac catheterization, the left ventricular end-diastolic pressure and cardiac output were 19 mm Hg and 4.9 L/min, 13 mm Hg and 4.6 L/min, 14 mm Hg and 5.3 L/min, respectively; the corresponding values for patients with left ventricular failure were 23 mm Hg and 4.1 L/min, 19 mm Hg and 4.4 L/min, 21 mm Hg and 4.3 L/min, respectively.

Echocardiography

This investigation was performed using a commercial echocardiograph, transducer and multi-channel oscillographic recorder. The technique of examination and precautions required to standardize measurements have been described in detail previously.

Measurements of the left ventricular internal dimension at end-diastole (LVIDd) and end-systole (LVIDs) were used to calculate an index of circumferential myocardial contraction, fractional shortening (FS),

\[
FS = \frac{LVIDd - LVIDs}{LVIDd} \times 100\%
\]

The end-diastolic and end-systolic dimensions were also used to calculate corresponding left ventricular end-diastolic volume (LVEDV), end-systolic volume, stroke volume (LVSV) and ejection fraction (EF).

Other measurements made from the echocardiogram were the end-diastolic thickness of the posterior left ventricular wall (PWTd), the right ventricular internal dimension (RV) and the left atrial dimension at end-systole (LA).

Normal values for these echocardiographic measurements were those obtained from 23 healthy subjects with no clinical evidence of cardiovascular abnormality and with a normal chest radiograph.

Results

Aortic Stenosis

The results are summarized in table 1 and figure 1. When stenosis was mild, the mean values of all dimensions were normal. In moderate stenosis, there was a 20% increase in the thickness of the left ventricular posterior wall (P < 0.05) and a significant increase in left atrial size (P < 0.02).

In severe aortic stenosis without left ventricular failure, the mean values for the left ventricular internal dimension, end-diastolic volume and stroke volume were all normal. However, there was a 44% increase in thickness of the posterior left ventricular wall (P < 0.01). The left atrium and aortic root were also enlarged (P < 0.01, P < 0.05). When aortic stenosis was severe and associated with left ventricular failure (fig. 2 left), the left ventricular end-diastolic

![Figure 1. Echocardiographic measurements in aortic stenosis: left) left ventricular end-diastolic dimension (LVIDd), right) fractional shortening (FS).](http://circ.ahajournals.org/)

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http://circ.ahajournals.org/ Downloaded from
Table 1. Echocardiographic Measurements in Aortic Valve Disease

<table>
<thead>
<tr>
<th>Group</th>
<th>N</th>
<th>LVIDd (cm)</th>
<th>FS (%)</th>
<th>PWTd (cm)</th>
<th>LVEDV (ml)</th>
<th>EF (%)</th>
<th>LVSV (ml)</th>
<th>LA (cm)</th>
<th>Ao (cm)</th>
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<td><strong>Aortic Stenosis</strong></td>
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<td></td>
<td></td>
<td></td>
<td></td>
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<td></td>
<td></td>
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<tr>
<td>Mild</td>
<td>7</td>
<td>4.6*</td>
<td>34.8</td>
<td>1.0</td>
<td>97.6</td>
<td>63.8</td>
<td>63.1</td>
<td>3.0</td>
<td>2.9</td>
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<tr>
<td></td>
<td></td>
<td>(0.5)</td>
<td>(5.3)</td>
<td>(0.1)</td>
<td>(27.0)</td>
<td>(7.1)</td>
<td>(21.9)</td>
<td>(0.8)</td>
<td>(0.2)</td>
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<tr>
<td>Moderate</td>
<td>9</td>
<td>4.5</td>
<td>33.4</td>
<td>1.1</td>
<td>94.6</td>
<td>61.7</td>
<td>58.6</td>
<td>3.2</td>
<td>2.9</td>
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<tr>
<td></td>
<td></td>
<td>(0.5)</td>
<td>(6.9)</td>
<td>(0.2)</td>
<td>(26.8)</td>
<td>(9.9)</td>
<td>(21.9)</td>
<td>(0.3)</td>
<td>(0.6)</td>
</tr>
<tr>
<td>Severe — No LVF</td>
<td>15</td>
<td>4.6</td>
<td>34.8</td>
<td>1.4</td>
<td>101.0</td>
<td>63.4</td>
<td>61.6</td>
<td>3.3</td>
<td>2.9</td>
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<tr>
<td>— LVF</td>
<td>14</td>
<td>5.7</td>
<td>18.5</td>
<td>1.4</td>
<td>165.0</td>
<td>37.3</td>
<td>56.4</td>
<td>3.7</td>
<td>3.1</td>
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<tr>
<td></td>
<td></td>
<td>(0.9)</td>
<td>(6.5)</td>
<td>(0.3)</td>
<td>(56.8)</td>
<td>(11.9)</td>
<td>(11.7)</td>
<td>(0.7)</td>
<td>(0.6)</td>
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<td><strong>Aortic Regurgitation</strong></td>
<td></td>
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<td></td>
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<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mild</td>
<td>7</td>
<td>4.4</td>
<td>33.1</td>
<td>1.1</td>
<td>86.8</td>
<td>61.9</td>
<td>53.1</td>
<td>2.9</td>
<td>3.1</td>
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<td>(0.2)</td>
<td>(5.6)</td>
<td>(0.2)</td>
<td>(11.3)</td>
<td>(7.6)</td>
<td>(3.3)</td>
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<td>(0.2)</td>
</tr>
<tr>
<td>Moderate</td>
<td>7</td>
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<td>32.2</td>
<td>1.1</td>
<td>147.6</td>
<td>59.3</td>
<td>87.6</td>
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<td>3.1</td>
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<td></td>
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<td>(0.2)</td>
<td>(7.0)</td>
<td>(0.2)</td>
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<td>(9.8)</td>
<td>(16.2)</td>
<td>(0.6)</td>
<td>(0.5)</td>
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<tr>
<td>Severe — No LVF</td>
<td>28</td>
<td>6.7</td>
<td>29.9</td>
<td>1.3</td>
<td>233.8</td>
<td>55.6</td>
<td>128.5</td>
<td>3.2</td>
<td>3.7</td>
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<tr>
<td>— Chronic LVF</td>
<td>9</td>
<td>7.0</td>
<td>20.5</td>
<td>1.3</td>
<td>257.0</td>
<td>40.2</td>
<td>103.1</td>
<td>4.1</td>
<td>3.5</td>
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<td></td>
<td></td>
<td>(1.0)</td>
<td>(6.8)</td>
<td>(0.2)</td>
<td>(83.4)</td>
<td>(10.9)</td>
<td>(48.4)</td>
<td>(1.1)</td>
<td>(0.4)</td>
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<tr>
<td>— Acute LVF</td>
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<td>6.5</td>
<td>30.1</td>
<td>1.1</td>
<td>218.8</td>
<td>62.4</td>
<td>122.6</td>
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<td></td>
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<td>(0.2)</td>
<td>(4.5)</td>
<td>(0.2)</td>
<td>(17.6)</td>
<td>(6.2)</td>
<td>(19.1)</td>
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<td><strong>Aortic Stenosis and Regurgitation</strong></td>
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<td></td>
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<td></td>
<td></td>
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<tr>
<td>No LVF</td>
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<td>34.0</td>
<td>1.3</td>
<td>168.0</td>
<td>61.8</td>
<td>100.6</td>
<td>3.3</td>
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<tr>
<td></td>
<td></td>
<td>(0.8)</td>
<td>(6.1)</td>
<td>(0.1)</td>
<td>(56.5)</td>
<td>(8.6)</td>
<td>(23.9)</td>
<td>(0.6)</td>
<td>(0.4)</td>
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<tr>
<td>LVF</td>
<td>13</td>
<td>6.1</td>
<td>22.0</td>
<td>1.4</td>
<td>195.5</td>
<td>42.9</td>
<td>76.1</td>
<td>4.2</td>
<td>2.9</td>
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<tr>
<td></td>
<td></td>
<td>(1.0)</td>
<td>(8.4)</td>
<td>(0.3)</td>
<td>(78.0)</td>
<td>(14.4)</td>
<td>(15.3)</td>
<td>(0.6)</td>
<td>(0.6)</td>
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<td>Normal</td>
<td>23</td>
<td>4.5</td>
<td>33.5</td>
<td>0.9</td>
<td>92.6</td>
<td>62.2</td>
<td>57.5</td>
<td>2.6</td>
<td>2.6</td>
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<tr>
<td></td>
<td></td>
<td>(0.4)</td>
<td>(4.4)</td>
<td>(0.2)</td>
<td>(18.7)</td>
<td>(6.0)</td>
<td>(12.9)</td>
<td>(0.6)</td>
<td>(0.3)</td>
</tr>
</tbody>
</table>

*Mean values with one standard deviation in parentheses.

dimension was larger ($P < 0.01$), but the stroke volume, left ventricular posterior wall thickness and left atrial size were not significantly different.

Myocardial contraction (mean value of fractional shortening) was normal in severe aortic stenosis without left ventricular failure but markedly depressed for the group with left ventricular failure ($P < 0.01$).

**Aortic Regurgitation**

The results are summarized in table 1 and figure 3.

When regurgitation was mild, the mean values for all measurements were normal. In moderate regurgitation, there were significant increases (22%) in the mean values of the left ventricular end-diastolic dimension and posterior wall thickness ($P < 0.01$, $P < 0.05$), and the left ventricular end-diastolic and stroke volumes which were increased by 59% and 52%, respectively (each $P < 0.01$).

In severe aortic regurgitation without left ventricular failure, the mean values of the left ventricular end-diastolic dimension and posterior wall thickness were increased to a similar extent — 37% and 44%, respectively (each $P < 0.01$). The end-diastolic and stroke volumes were increased by 152% and 123%, respectively (each $P < 0.01$). The aortic root was enlarged ($P < 0.01$) but the increase in left atrial size was not significant.

There were important differences between echocardiographic measurements depending on whether left ventricular failure was associated with chronic or acute aortic regurgitation. In chronic aortic regurgitation (fig. 2 middle), the left ventricular end-diastolic internal dimension, end-diastolic volume and posterior wall thickness were slightly larger and the stroke volume slightly less, but none of these differences was statistically significant. The left atrial dimension was larger in patients with severe aortic regurgitation when there was left ventricular failure ($P < 0.01$).

Myocardial contraction (mean value of fractional shortening) was slightly but significantly reduced in severe chronic aortic regurgitation without left ventricular failure ($P < 0.05$) but more severely reduced with failure ($P < 0.01$). However, when aortic regurgitation was of acute onset (fig. 2 right), the fractional shortening fell within the normal range in all patients and the reduction in mean value was not statistically significant.

**Mixed Aortic Stenosis and Regurgitation**

The results are summarized in table 1 and figure 4.

In the absence of left ventricular failure, the left ventricular internal end-diastolic dimension, end-diastolic volume and stroke volume were all moderately increased (each $P < 0.01$). The left ventricular posterior wall thickness, left atrial and aortic root dimensions were also increased above normal (each $P < 0.01$). In the presence of left ventricular failure, fractional shortening was significantly lower ($P < 0.01$) and the left atrium larger ($P < 0.01$).
Discussion

Left Ventricular Overload

The echocardiographic measurements did reflect the anticipated response of the left ventricle to increased load. Thus pressure overload due to aortic stenosis was manifest as concentric hypertrophy — the cavity was of normal size and the increase in mural thickness was related to the severity of stenosis. Volume overload due to aortic regurgitation caused magnification hypertrophy — there was an increase in left ventricular end-diastolic internal dimension with a proportionate increase in mural thickness and an increase in stroke volume which was related to the severity of regurgitation.

In chronic severe aortic valve disease without left ventricular failure, myocardial contraction (mean value of fractional shortening) was normal. However, reduced values of fractional shortening were recorded in 13% of patients (figs. 1, 3 and 4). This finding is consistent with earlier reports of impairment of left ventricular function in some patients with chronic left ventricular overload and hypertrophy even in the absence of failure.

Left Ventricular Failure

In chronic severe aortic valve disease with left ventricular failure, reduced myocardial contraction was manifest as a substantial reduction in fractional shortening. Such a reduction in the extent of shortening is a characteristic feature of myocardial failure and has been previously detected in aortic valve disease as a reduced angiographic ejection fraction. In aortic stenosis, there was also a substantial increase in the size of the left ventricular cavity; this increase is believed to be a compensatory mechanism which opposes the tendency...
of impaired myocardial contraction to reduce the stroke volume and cardiac output. When left ventricular failure was associated with aortic regurgitation and mixed aortic valve disease, the left ventricle was only slightly larger and these differences were not statistically significant.

In a minority of patients with chronic aortic valve disease and clinical left ventricular failure, the value of fractional shortening which was recorded was within the normal range (figs. 1, 3 and 4). Since we have already indicated that some asymptomatic patients with severe aortic valve disease have a reduced value of fractional shortening, the implication is that the values of this index in patients with and without left ventricular failure did overlap. This does limit the value of an isolated estimation of the index in the detection of left ventricular failure. Nevertheless, if other clinical evidence of failure is equivocal, the lower the value of fractional shortening, the more likely is it that decompensation has begun. Furthermore, it is likely that serial estimations of fractional shortening will prove more useful in detecting a progressive decline in myocardial contraction. This remains to be proved in chronic disease, but we have been able to detect a decline in fractional shortening over several months in some patients with acute severe aortic regurgitation when aortic valve disease had been deferred (unpublished observations).

When severe aortic regurgitation was of acute onset, the left ventricle responded quite differently. Myocardial contraction remained normal despite the presence of pulmonary venous congestion; this suggests that sudden severe left ventricular overload had caused pump failure rather than myocardial failure with impaired contraction. A similar situation has been described when heart failure has been produced in animals by an arteriovenous fistula or by acute damage to the aortic valve.

Conclusions

Echocardiographic measurements derived from the left ventricular echocardiogram reflect both the severity and the type of aortic valve disease. Reduced left ventricular myocardial contraction is characteristically found in chronic severe aortic valve disease with clinical left ventricular failure but myocardial contraction is initially normal in patients with acute severe aortic regurgitation.

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References

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I G McDonald

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