Evaluation of posterior aortic wall echogram in diagnosis of mitral valve disease

R. J. C. HALL1, S. E. CLARKE2, AND D. BROWN

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SUMMARY  The clinical usefulness of detecting abnormal movement of the posterior wall of the aortic root by M-mode echocardiography was studied in 93 patients with common cardiac diseases (mitral and aortic valve disease, atrial septal defect, hypertrophic and congestive cardiomyopathy) and in 17 normal subjects. Though abnormally slow (< 3 cm/s) or prolonged (> 240 ms) diastolic movement was confirmed to be common in mitral stenosis, since it occurred in 35 of 36 patients it was non-specific. Similar abnormalities frequently occurred in other patients with, for example, mitral regurgitation, aortic valve disease, after aortic valve replacement, and in both hypertrophic and congestive cardiomyopathy. The severity of mitral stenosis, assessed at operation, could not be accurately predicted from abnormalities of aortic root movement. Information derived from aortic movement was not diagnostic and did not predict severity in isolated mitral regurgitation though both the peak rate of systolic aortic motion and total aortic excursion were significantly greater than normal. We conclude that abnormalities of posterior aortic wall movement are frequent and their specificity and clinical usefulness are limited.

Attention was drawn to the movement of the aortic root as long ago as 1907 by Sir Arthur Keith, who realised that the most mobile part of the left atrium is its anterior wall, bordered by the aortic root. He argued from purely anatomical evidence that during left ventricular systole the aortic root would move forwards as the left atrium filled. Recent echocardiographic studies (Strunk et al., 1976b; Akgün and Layton, 1977) have confirmed this prediction, that normal aortic root movement occurs mainly in response to left atrial volume changes, though the contributions of left ventricular ejection and elastic recoil of the aorta have yet to be defined (Pratt et al., 1976).

More recently it has been postulated that aortic root movement is so closely related to left atrial volume changes that it can be used to predict the pattern of left ventricular filling in patients and hence the severity of mitral stenosis (Strunk et al., 1977), the performance of mitral prostheses (Strunk et al., 1977), the severity of mitral regurgitation (Strunk et al., 1976a; Akgün and Layton, 1977), and the behaviour of the left ventricle itself in other unspecified conditions (Strunk et al., 1976b).

The present study was designed to test these assertions by investigation of the types of abnormal posterior aortic wall movement occurring in several common forms of heart disease and to assess the specificity of such abnormalities.

Subjects and methods

Studies were made on 110 patients. There were 17 normal subjects ranging in age from 15 to 65 years. The diagnoses of the 93 patients with heart disease are shown in Table 1. These were confirmed in all patients with valve disease or atrial septal defect.

<table>
<thead>
<tr>
<th>Diagnosis</th>
<th>No. of patients</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal</td>
<td>17</td>
</tr>
<tr>
<td>Mitral stenosis</td>
<td>18</td>
</tr>
<tr>
<td>Severe non-rheumatic mitral regurgitation</td>
<td>15</td>
</tr>
<tr>
<td>Mixed mitral stenosis and regurgitation</td>
<td>12</td>
</tr>
<tr>
<td>Mixed aortic and mitral valve disease</td>
<td>6</td>
</tr>
<tr>
<td>Aortic regurgitation</td>
<td>10</td>
</tr>
<tr>
<td>Aortic stenosis</td>
<td>7</td>
</tr>
<tr>
<td>Previous aortic valve replacement</td>
<td>6</td>
</tr>
<tr>
<td>Atrial septal defect</td>
<td>6</td>
</tr>
<tr>
<td>Hypertrophic cardiomyopathy</td>
<td>6</td>
</tr>
<tr>
<td>Congestive cardiomyopathy</td>
<td>7</td>
</tr>
</tbody>
</table>

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at cardiac catheterisation or at operation. Of the 6 patients with hypertrophic cardiomyopathy, 4 were catheterised and the remaining 2 patients had typical clinical and echocardiographic findings with no evidence of aortic valve disease or hypertension. Three of the patients with congestive cardiomyopathy were not catheterised but had gross cardiomegaly, dilated poorly contracting left ventricles on echocardiography, and no evidence of ischaemic heart disease. No patients with ischaemic heart disease were included in the study.

ECHOCARDIOGRAMS

Echocardiograms were recorded at 75 or 100 mm/s paper speed using a Smith Kline Ekoline 20 ultrasonoscope connected to a Cambridge strip chart recorder, using a 2:25 MHz 1:25 cm transducer.

Data processing

Echocardiographic traces of both aorta and left ventricle were digitised as previously described (Gibson and Brown, 1973), using a Summagraphics digitiser (20 by 20 in, with 0.01 in resolution) and a Prime 300 computer system. Left ventricular and aortic traces were compared from beats of the same duration ± 75 ms and were recorded during the same session.

(a) Aortic root echocardiograms. The leading edges of the anterior and posterior walls of the aortic root, and the posterior wall of the left atrium were digitised (Fig. 1). Traces of the aortic root were always taken so that some element of the aortic valve apparatus was included.

From these digitised traces the following data were calculated and plotted:

(i) Overall excursion of the posterior aortic wall during the cardiac cycle.

(ii) The amplitude of passive backward movement of the aorta; that is that occurring in early and mid-diastole, preceding the onset of atrial or ventricular systole.

(iii) The peak rates of early diastolic backward movement and systolic forward movement.

(iv) The duration of passive diastolic backward movement.

(v) The atrial emptying index (Strunk et al., 1977). This measurement is the proportion of passive diastolic backward movement of the posterior aortic wall that occurs during the first third of diastole. Diastole, for this purpose, is taken as the period from the onset of backward aortic movement until the posterior aortic wall is deflected by atrial systole or ventricular systole; if neither deflect the aortic root, the end of the QRS marks the end of diastole. The left atrial dimension throughout the cardiac cycle was also plotted. Changes in this variable always followed posterior aortic wall excursion and therefore have not been tabulated.

(b) Left ventricular echocardiograms. Left ventricular echocardiograms showed clear continuous echoes of the left side of the septum and the endocardium of posterior wall. These traces were digitised and the instantaneous dimension and rate of change of dimension were plotted (Gibson and Brown, 1973). The peak rate of change of left ventricular dimension during systole and diastole was measured.

STATISTICS

Differences between groups were examined using unpaired t tests, and correlations between variables were examined using least squares linear regression. Values which differed from the mean of the normal group by more than 2 standard deviations were taken as being abnormal.

Results

PATTERN AND RATE OF AORTIC ROOT MOVEMENT

All normal subjects showed a similar pattern of aortic root movement. The total excursion was
9·2 ± 2·0 mm (mean ± SD) of which 6·3 ± 1·6 mm (mean ± 1 SD) occurred passively during diastole and the remainder in atrial or ventricular systole (Table 2). Diastolic movement was in a backward direction and occurred rapidly at first with a peak rate of 6·3 ± 1·6 cm/s, and then more slowly until the onset of atrial systole. The duration of this initial rapid phase was 160 ± 41 ms. On the basis of these normal results, termed type I (Fig. 1), 2 further patterns of movement were defined (Fig. 2). In type II (Fig. 3), the peak rate of early diastolic movement was reduced to less than 3 cm/s and its duration prolonged to more than 240 ms. In type III, duration was also prolonged but the peak rate was normal.

In 17 of the 18 patients with pure mitral stenosis the duration of the period of early diastolic backward movement was prolonged. In 13 of these the peak velocity of backward movement was reduced to below 3 cm/s. In 1 patient with significant mitral stenosis confirmed at catheterisation and operation, aortic root movement was normal. Similar abnormalities were also seen in mixed mitral valve disease whether accompanied by aortic valve disease or not. In 35 of the 36 patients

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**Table 2** Results from digitised aortic and left ventricular echocardiograms of 110 patients

<table>
<thead>
<tr>
<th></th>
<th>Normal</th>
<th>Mitral stenosis</th>
<th>Severe mitral regurgitation</th>
<th>Mixed mitral stenosis and regurgitation</th>
<th>Mitral and aortic valve disease</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total aortic excursion (mm)</td>
<td>9·2 ± 2·0</td>
<td>9·1 ± 3·4</td>
<td>12 ± 3·5*</td>
<td>8·0 ± 1·7</td>
<td>10·6 ± 3·1</td>
</tr>
<tr>
<td>Passive aortic excursion (mm)</td>
<td>6·3 ± 1·6</td>
<td>6·1 ± 2·5</td>
<td>8·3 ± 4·6</td>
<td>6·3 ± 2·4</td>
<td>3·3 ± 3·1*</td>
</tr>
<tr>
<td>Peak rate of aortic forward motion (cm/s)</td>
<td>4·9 ± 1·6</td>
<td>4·1 ± 1·7</td>
<td>6·5 ± 2·7*</td>
<td>3·9 ± 1·7</td>
<td>4·6 ± 1·9</td>
</tr>
<tr>
<td>Peak rate early diastolic backward movement of Ao (cm/s)</td>
<td>6·3 ± 1·6</td>
<td>2·9 ± 1·7***</td>
<td>5·6 ± 3·8</td>
<td>3·3 ± 2·0**</td>
<td>1·5 ± 3·8***</td>
</tr>
<tr>
<td>Peak rate LV diastolic dimension change (cm/s)</td>
<td>15·7 ± 2·3</td>
<td>6·3 ± 1·2*</td>
<td>33 ± 10*</td>
<td>16·1 ± 5·1</td>
<td>9·2 ± 2·9*</td>
</tr>
<tr>
<td>No.</td>
<td>17</td>
<td>18</td>
<td>15</td>
<td>12</td>
<td>6</td>
</tr>
</tbody>
</table>

All results = mean ± SD.  *P < 0·05, ***P < 0·001.

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**Fig. 2** Echocardiographic data showing type II (derived from a patient with mitral stenosis) and type III aortic movement (from a patient after aortic valve replacement). The cross on the type II trace represents the onset of the QRS.

**Fig. 3** The echocardiogram of the aortic root in a patient with pure mitral stenosis. This is type II motion (peak rate of backward aortic root motion = 2·9 cm/s). The valve area at operation was 0·5 cm².
in whom there was any significant mitral stenosis there was an abnormal pattern of an aortic root movement. In patients with severe non-rheumatic mitral regurgitation the pattern of diastolic aortic root movement was normal in 7 and indistinguishable from that occurring in patients with significant mitral stenosis in the remaining 8 (Table 3). Abnormalities of diastolic aortic root movement were not confined to patients with mitral valve disease, but were also seen in pure aortic stenosis or regurgitation, and after aortic valve replacement. They were also present in other types of heart disease, particularly in congestive and hypertrophic cardiomyopathy (Tables 2 and 3), or after aortic valve replacement.

Patients with atrial septal defect, and those with severe mitral regurgitation both showed a significant increase in the total excursion of the posterior aortic wall (Table 2), and in severe, pure mitral regurgitation the peak rate of systolic anterior motion of the aorta was also significantly increased (P < 0.01).

### Table 3  Type of aortic movement occurring in all 110 patients

<table>
<thead>
<tr>
<th>Normal Mitral stenosis</th>
<th>Severe mitral regurgitation</th>
<th>Mixed mitral stenosis and aortic valve disease</th>
<th>Aortic regurgitation</th>
<th>Aortic stenosis</th>
<th>Aortic valve replacement</th>
<th>Atrial septal defect</th>
<th>Hypertrophic cardiomyopathy</th>
<th>Congestive cardiomyopathy</th>
</tr>
</thead>
<tbody>
<tr>
<td>Number</td>
<td>17</td>
<td>18</td>
<td>15</td>
<td>12</td>
<td>6</td>
<td>10</td>
<td>7</td>
<td>6</td>
</tr>
<tr>
<td>Atrial emptying index</td>
<td>&lt; 0.5</td>
<td>1</td>
<td>11</td>
<td>6</td>
<td>7</td>
<td>6</td>
<td>3</td>
<td>5</td>
</tr>
<tr>
<td>Type I</td>
<td>17</td>
<td>1*</td>
<td>7</td>
<td>—</td>
<td>5</td>
<td>4</td>
<td>1</td>
<td>5</td>
</tr>
<tr>
<td>Type II</td>
<td>—</td>
<td>13</td>
<td>2</td>
<td>8</td>
<td>2</td>
<td>2</td>
<td>2</td>
<td>—</td>
</tr>
<tr>
<td>Type III</td>
<td>—</td>
<td>4</td>
<td>6</td>
<td>4</td>
<td>4</td>
<td>3</td>
<td>1</td>
<td>3</td>
</tr>
</tbody>
</table>

*Only patient with mitral stenosis and a normal pattern of aortic movement.
INCREASE
DIMENSION
with
PEAK RATE
Values for peak rate of early diastolic aortic root movement (Fig. 5), total aortic root excursion, or atrial emptying index. Attempts to normalise the values of any of these aortic variables for left atrial size did not improve agreement. There was no linear correlation between peak rates of change of left ventricular dimension in diastole and the rate of diastolic aortic root movement, either when all patients were considered together or in separate diagnostic groups. In general, if left ventricular dimension change, measured echocardiographically, was slow (<10 cm/s), then aortic root movement in diastole (Fig. 6) was also slow or prolonged (34 of 41 patients), but aortic root movement was often abnormal when the left ventricular echocardiogram was normal (31 of 52 patients).

Fig. 4 The variation of the atrial emptying index with RR interval in 2 patients with atrial fibrillation and moderately severe mitral stenosis.

PEAK RATE OF LEFT VENTRICULAR DIMENSION INCREASE
Values for peak diastolic rate of left ventricular dimension increase are given in Table 2 and shown diagrammatically in Fig. 5. It can be seen that most patients with mitral stenosis or mitral regurgitation could be separated both from normal and from each other on the basis of measurements of peak rate of increase in their left ventricular dimension, but not by the peak rate of early diastolic aortic root movement (Fig. 5), total aortic root excursion, or atrial emptying index. Attempts to normalise the values of any of these aortic variables for left atrial size did not improve agreement. There was no linear correlation between peak rates of change of left ventricular dimension in diastole and the rate of diastolic aortic root movement, either when all patients were considered together or in separate diagnostic groups. In general, if left ventricular dimension change, measured echocardiographically, was slow (<10 cm/s), then aortic root movement in diastole (Fig. 6) was also slow or prolonged (34 of 41 patients), but aortic root movement was often abnormal when the left ventricular echocardiogram was normal (31 of 52 patients).

Fig. 5 Comparison of (A) peak rate of left ventricular dimension change and (B) peak rate of diastolic aortic root motion in patients with pure mitral stenosis (MS) or mitral regurgitation (MR) and normal subjects.
Discussion

The present study has confirmed previous ones in showing that the posterior wall of the aortic root moves in a way that might be predicted from the left atrial volume curve, and that abnormal patterns may occur in patients with heart disease. We were, however, unable to confirm that these abnormalities were specific to particular conditions or physiological disturbances, or that they were of value in the assessment of individual patients.

Two separate approaches to the quantification of aortic root movement have been used. The first, the left atrial emptying index, was described by Strunk et al. (1977), and found by them to correlate well with the mitral valve area, corrected for body surface area, in 25 patients with mitral stenosis. These authors also suggested that the atrial emptying index might provide useful information about prosthetic mitral valve dysfunction. We were not able to confirm these results. Though in the majority of patients with mitral stenosis, atrial emptying index was reduced, it was not possible to use it to predict the mitral valve area found at operation with any accuracy, and in 4 patients with severe narrowing this index was only slightly reduced or even normal. Its dependence on heart rate must introduce uncertainty into any attempt to use it in assessing patients with mitral stenosis and atrial fibrillation. Its value in detecting possible prosthetic valve dysfunction was not formally investigated in the present study, but the frequent appearance of abnormal aortic root movement after cardiac operations not involving the mitral valve did not encourage us to pursue its use in this direction.

A second approach was defined by Akgün and Layton (1977) who suggested that echocardiographic measurement of left atrial dimension change might help in assessing the severity of mitral regurgitation. We were able to confirm that left atrial dimension change assessed by measuring total excursion of the posterior aortic wall, which is the only mobile wall of the left atrium, is increased in mitral regurgitation and that forward aortic movement in systole is abnormally rapid.

These changes could not be used, however, to diagnose mitral regurgitation since aortic root excursion was also increased in patients with atrial septal defect, nor could they be used to define the severity of mitral regurgitation since they were not present in 8 of the 15 patients. The inability of early diastolic aortic root movement accurately to predict left ventricular filling rates is highlighted by the findings in mitral regurgitation. In 14 of 15 patients with severe mitral regurgitation the left ventricular filling rate measured as the peak rate of diastolic left ventricular dimension change was abnormally rapid (more than 2 standard deviations greater than normal, that is > 21.3 cm/s) but in 12 of these 14 patients the peak rate of early diastolic aortic root motion was normal (Fig. 7) or less than normal (< 9.5 cm/s).

A reduction in the peak rate of early diastolic aortic root motion (type II motion) was a sensitive (present in 13 of 18 patients) but non-specific indicator of pure mitral stenosis, since though it occurred most commonly in pure mitral stenosis it was also seen in 2 patients with pure mitral regurgitation, as well as in patients with aortic valve disease, after aortic valve replacement, and with congestive or hypertrophic cardiomyopathy. The ability to detect mitral stenosis from aortic root motion was increased further by considering type III motion (prolonged diastolic backward motion occurring at a normal rate), since 17 of the
18 patients with mitral stenosis had either type II or III motion. Though considering both types of motion increased sensitivity it also reduced specificity further since many other patients without mitral valve disease showed type III motion.

Measurements of peak rates of diastolic aortic root movement also performed much less well than those of transverse left ventricular dimension in separating patients with mitral stenosis or regurgitation from normal (Fig. 5). The theoretical possibility that measurement of posterior aortic root movement might be of value in excluding mitral stenosis in circumstances when other echocardiographic measurements could not be obtained for technical reasons was not found to apply, since a recognisable mitral valve echogram was recorded in all, and, though we failed to obtain satisfactory left ventricular echograms in 7 patients, aortic root measurements were not possible in 5 other patients.

The pathophysiological basis of this lack of specificity of aortic root movement seems clear. The amplitude and rate of this movement during the cardiac cycle must depend on the relative rates of left atrial inflow and outflow of blood, the size and distensibility of the left atrium, and the mobility of the aortic root. The left atrium has 5 large openings in its walls, only 1 of which has a valve; it is frequently enlarged in heart disease and the properties of its walls may be altered by many factors including rheumatic disease, fibrosis, mural thrombus, and scarring from previous operations. The mobility of the aortic root must also be affected by fibrosis, scarring, and calcification both in it and in the adjacent mitral valve and left atrium. Therefore, it is not surprising that movement of the posterior aortic wall should be altered in such a non-specific and unpredictable way in heart disease.

References


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