Clinical significance of myocardial echo intensity in hypertrophic cardiomyopathy and other forms of left ventricular hypertrophy

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Summary: An approach by quantifying the echoes arising from hypertrophied myocardium, is described in patients with left ventricular hypertrophy of various causes, including hypertrophic cardiomyopathy. Amplitude of echoes judged by pixel count was significantly increased in patients with hypertrophic cardiomyopathy, hypertension and aortic stenosis as compared with normals and athletes with a comparable increase in left ventricular mass.

It is suggested that the increase in echo intensity reflects an increase in myocardial collagen content.

The echocardiographic features of classical hypertrophic cardiomyopathy (HCM) are so characteristic that their apparent specificity has raised the possibility of identifying pathognomonic features for use in doubtful cases. This approach has proved remarkably unfruitful, in spite of repeated attempts based on a number of features of the disease. Initially, attention was directed towards valve motion, either to systolic anterior motion of the anterior cusp of the mitral valve, or to mid-systolic closure of the aortic valve (Doi et al., 1980; Wong et al., 1980). Both these findings have since been shown to be non-specific; not only are they absent from otherwise typical cases, but they may appear in a variety of unrelated conditions. Systolic anterior motion, for example, is seen with hypovolaemia or after the Mustard operation for transposition of the great arteries, while mid-systolic aortic closure has been documented with dissecting aneurysm, congestive cardiomyopathy, mitral regurgitation and Fallot's tetralogy (Wong et al., 1980). Attempts to identify specific patterns of myocardial hypertrophy have proved no more successful.

Although asymmetric septal hypertrophy was proposed as a 'pathognomonic' abnormality (Henry et al., 1973), allowing hypertrophic cardiomyopathy to be separated from secondary left ventricular hypertrophy, this suggestion has not been confirmed. Marked asymmetric rather than symmetric hypertrophy is frequently seen in patients with left ventricular hypertrophy, not only in that due to hypertension or left ventricular outflow tract obstruction (Gibson et al., 1979), but even in the physiological hypertrophy of athletes (Menapace et al., 1977). Selective apical hypertrophy alone has so far proved specific for hypertrophic cardiomyopathy, but this occurs in only a small minority of cases (Shapiro & McKenna, 1983). The same considerations apply to abnormalities of diastole; prolongation of isovolumic relaxation, delay in mitral valve opening, and reduced rate of dimension increase or posterior wall thinning (Sanderson et al., 1977). Although these abnormalities are frequently seen in hypertrophic cardiomyopathy, even in this condition, their presence is not universal, while they occur with even greater frequency in patients with severe secondary left ventricular hypertrophy (Gibson et al., 1979).

Methods

In the present study, an attempt is made to examine another echocardiographic approach to the study of patients with hypertrophic cardiomyopathy, by quantifying the echoes arising from the hypertrophied myocardium itself. A number of findings suggested that this might be a fruitful approach. There is much evidence to suggest that myocardial structure, particularly in the septum, might be abnormal, showing the appearances of fibre disarray (Maron & Roberts, 1979). In addition, it has previously been reported that the echo texture of the septum is abnormal (Martin et al., 1979), showing a 'ground glass' appearance. We thus studied 16 patients with hypertrophic cardiomyopathy using two dimensional echocardiography, but, in view of the findings outlined above, we extended our observations to those with secondary hypertrophy of a variety of causes. These included 20 with aortic stenosis, 7 with subaortic stenosis, 8 with severe essential hypertension, 7 with coarctation of the aorta, and 15 athletes with left ventricular hypertroph-

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phy. These results were compared with those from 12 normal subjects.

The methods used have been described in detail elsewhere (Logan-Sinclair et al., 1981). Two dimensional echocardiograms were recorded with a mechanical (ATL) sector scanner. The images were processed in real time through a colour encoder. The gain of the instrument was set to the lowest level at which the echo from the parietal pericardium behind the posterior left ventricular wall appeared as a continuous line at the highest grey scale level. Near gain compensation was not used. Images were recorded on 3/4 inch U-matic video tape, and end-diastolic frames analysed retrospectively (Shaw et al., 1984). On an appropriate stop frame, areas of interest were identified on the basal and mid-regions of the septum and posterior walls. All the pixels in the area were counted automatically, and their relative amplitudes displayed as a histogram, with an additional numerical read-out. From this information, the median value of pixel intensity in each area was derived. Simultaneous M-mode echocardiograms and phonocardiograms were also recorded from each patient, and these records were digitized (Sanderson et al., 1977) allowing diastolic events to be analysed. Finally, the standard 12 lead electrocardiogram (ECG) was examined for evidence of ST-T wave abnormalities constituting the 'strain' pattern. Interrelations between these observations were investigated (Shapiro et al., 1984).

**Results**

The results are summarized in Tables I and II. Normal myocardium reflects ultrasound with low intensity, the median pixel intensity in the posterior wall being 0.7 unit at the base and 0.62 in the mid-portion of the cavity. Values from the septum are characteristically higher, being 1.1 in both regions. Normal values were also seen in the athletes, in spite of a considerable increase in left ventricular mass. However, increased values were seen in all the groups of patients with secondary left ventricular hypertrophy, whatever the cause. It was of interest that almost identical values were recorded from patients with hypertrophic cardiomyopathy, results from the septum in these patients being virtually identical to those seen in patients with aortic stenosis. Thus any abnormality of echo texture seen in these patients is not accompanied by any objective evidence of a specific increase in local pixel intensity.

The clinical correlates of an increase in pixel intensity are given in Table II. Since no significant difference was demonstrated between the patients with hypertrophic cardiomyopathy and those with secondary left ventricular hypertrophy, all the patients have been considered as a single group. Here, it is apparent that patients with an increased pixel intensity in any region of the myocardium show significant differences from those without, even though the increase in left ventricular muscle mass is the same. The former patients show a greatly increased tendency to have abnormalities of diastolic function, as shown by prolongation of isovolumic relaxation time, delay in mitral valve opening with respect to minimum cavity dimension, and also the peak rates of dimension increase and wall thinning during filling. In addition, these patients show a greatly increased incidence of the ECG pattern of 'strain'. Virtually identical results were obtained if patient selection was made on the basis of any other pattern of pixel intensity.

**Discussion**

A number of conclusions can be drawn from these results. First, an increase in left ventricular muscle mass on its own is not associated with diastolic abnormalities, a strain pattern on the ECG, or increase in echo intensity. Secondly, as with valve motion and the ventricular morphology, no difference can be demonstrated by the myocardial echo amplitude in hypertrophic cardiomyopathy and that in severe secondary left ventricular hypertrophy. The reason for this increase in each amplitude is not clear. The possibility obviously arises that it might be due to fibre

**Table I** Regional echo intensity in left ventricular hypertrophy (median pixel count)

<table>
<thead>
<tr>
<th></th>
<th>n</th>
<th>Posterior wall</th>
<th>Septum</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>Med Basal</td>
<td>Mid Basal</td>
</tr>
<tr>
<td>Normal</td>
<td>12</td>
<td>0.7±0.2</td>
<td>0.6±0.1</td>
</tr>
<tr>
<td>Athletes</td>
<td>15</td>
<td>0.6±0.1</td>
<td>0.6±0.1</td>
</tr>
<tr>
<td>Septum</td>
<td>16</td>
<td>1.1±0.4*</td>
<td>1.2±0.4*</td>
</tr>
<tr>
<td>Hypertension</td>
<td>15</td>
<td>0.9±0.3</td>
<td>0.9±0.4*</td>
</tr>
<tr>
<td>Aortic stenosis</td>
<td>27</td>
<td>1.3±0.6**</td>
<td>1.3±0.6**</td>
</tr>
</tbody>
</table>

HCM = hypertrophic cardiomyopathy. Significance of differences between pairs of values: *P < 0.05, **P < 0.001.
Table II  Association between increased echo amplitude in any region and left ventricular function

<table>
<thead>
<tr>
<th></th>
<th>Normal</th>
<th>Increased</th>
</tr>
</thead>
<tbody>
<tr>
<td>End-diastolic dimension (cm)</td>
<td>4.9±0.9</td>
<td>4.4±0.9*</td>
</tr>
<tr>
<td>Left ventricular mass (g)</td>
<td>320±130</td>
<td>340±170</td>
</tr>
<tr>
<td>Peak rate of dimension increase (cm/s)</td>
<td>12.9±5</td>
<td>7.7±3***</td>
</tr>
<tr>
<td>Peak rate of wall thinning (cm/s)</td>
<td>7.6±2.7</td>
<td>4.7±1.8***</td>
</tr>
<tr>
<td>Isovolumic relaxation time (ms)</td>
<td>65±16</td>
<td>85±28***</td>
</tr>
<tr>
<td>Minimum dimension to MVO (ms)</td>
<td>26±31</td>
<td>54±80***</td>
</tr>
<tr>
<td>Number with ECG 'strain'</td>
<td>6</td>
<td>34***</td>
</tr>
</tbody>
</table>

MVO = mitral valve opening. Differences between pairs of values: *P < 0.05, ***P < 0.001.

disarray, but if this were the case, particularly high values might be expected to arise from the septum in the patients with hypertrophic cardiomyopathy (Maron & Roberts, 1979). There is some experimental evidence to suggest that myocardial oedema or inflammation may be associated with an increase in integrated backscatter (Mimbs et al., 1981), but there seems no reason to suppose that either of these processes are consistently present in patients with left ventricular hypertrophy. The likeliest cause, therefore, is that increased echo amplitude is due to an increase in myocardial collagen content. This relation has previously been demonstrated in man, using identical echocardiographic methods (Shaw et al., 1984). If this is the case, it is tempting to speculate that this increase in fibrosis is also responsible for the disturbed diastolic function and ECG abnormalities. The present results are based on a very simple approach to the analysis of returning echoes, that of measurement of amplitude only. However, work is now proceeding in several centres investigating ways in which a more comprehensive examination can be made. It seems likely that a study of the hypertrophied myocardium will provide a fruitful clinical field for these new methods as they become available.

Acknowledgement

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References


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