Echocardiographic Assessment of Left Ventricular Function in Mitral Valve Disease

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SUMMARY Echocardiography was used to study left ventricular size and contraction in 90 patients with isolated mitral valve disease (47 patients with mitral stenosis, 26 with mixed mitral valve disease and 89 with mitral regurgitation). Left ventricular measurements included end-diastolic internal dimension (LVIDd), mural thickness (PWTd), an index of circumferential myocardial contraction — fractional shortening,

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

— and stroke volume (LVSV).

The left ventricle was abnormally small only when mitral stenosis was severe. Reduced myocardial contraction was common in patients with rheumatic valvular heart disease but was rarely severe. In mitral regurgitation without left ventricular failure, measurements were characteristic of volume overload with increases in LVIDd, LVSV and PWTd which were related to the severity of regurgitation. In other diseases, left ventricular failure is usually associated with reduced myocardial contraction (FS) but in mitral regurgitation with failure, myocardial contraction (FS 32%) did not differ significantly from normal (34%). The reduction in afterload caused by mitral regurgitation probably increases myocardial contraction and may lead to underestimation of the severity of myocardial impairment. Also potentially misleading was severe mitral regurgitation with normal values for LVIDd and LVSV (three patients). When the distinction between cardiomyopathy with secondary mitral regurgitation and primary mitral regurgitation was difficult clinically, echocardiography could usually make the distinction by demonstrating severe reduction of myocardial contraction with a slight or moderate increase in LVSV.

ASSESSMENT OF MITRAL STENOSIS was the first application of diagnostic ultrasound to clinical cardiology. More recently, the scope of the technique has been extended to include the measurements of cardiac chamber dimensions and to the assessment of left ventricular myocardial function. This study represents an analysis of our experience with echocardiographic assessment of the left ventricle in patients with mitral valve disease. The incidence of impairment of left ventricular function in mitral stenosis and problems in the detection of impaired myocardial function in patients with severe mitral regurgitation have been emphasized.

Patients and Methods

All patients with isolated mitral valve disease who had been routinely studied by echocardiography over a three year period were considered for inclusion in this study, provided their echocardiograms were technically adequate for measurement of left ventricular dimensions. Specifically excluded from the analysis were patients with prosthetic valves or with other possible causes for left ventricular enlargement or impairment such as ischemic heart disease, hypertension (systolic blood pressure > 180 mm Hg or diastolic > 100 mm Hg). Two patients with mitral regurgitation studied while in hospital with acute rheumatic fever were also excluded. Four others with less definite evidence for rheumatic activity, who were clinically well at the time of investigation, were included. All four had an elevated erythrocyte sedimentation rate, two had an elevated antistreptolysin O titer, one had joint pains, and evidence of recent acute pericarditis was found at surgery in one patient.

Of the 153 patients who were suitable for analysis, 47 had mitral stenosis, 26 mixed mitral stenosis and regurgitation and 80 had pure mitral regurgitation — mitral valve prolapse 53, rheumatic valvulitis 17, endocarditis 1 (and a contributory factor in 4) and of unknown cause in 9 patients. All patients were over the age of 16 years (42 years).

For comparison with the patients with primary mitral regurgitation, nine patients with mitral regurgitation considered to be secondary to cardiomyopathy were included as a separate group. In these patients, it was doubtful clinically whether mitral regurgitation was the cause of left ventricular impairment or whether mitral regurgitation was secondary to left ventricular enlargement. Since left ventricular angiography revealed that mitral regurgitation was only moderate and excluded ischemic heart disease as the cause, the final diagnosis was cardiomyopathy with secondary (or associated) mitral regurgitation. During the period of this study, there were two patients in whom left ventricular angiography could not distinguish between primary mitral regurgitation and cardiomyopathy with secondary regurgitation; these patients were excluded from further analysis.

Investigations

Good correlation between angiocardiographic and echocardiographic measurements of left ventricular dimensions has already been demonstrated. An important advantage of echocardiography as a noninvasive investigation is that all patients, including those with mild and moderate as well as with severe heart disease, can be studied; hence our aim was to document the effects on left ventricular size and contraction of mitral valve disease of any severity, rather than to present data only for those studied by cardiac catheterization and angiography. Patients were studied by cardiac catheterization to assess them for cardiac surgery or to clarify the nature and severity of their disease as an aid to optimal medical management. Using these selection criteria, 102 of 153 patients were studied by cardiac catheterization and angiography and the remainder were assessed by noninvasive tests.

Right heart catheterization was performed via an antecu-
bital vein and left heart catheterization via a right brachial arteriotomy. A left ventricular cineangiogram in the right anterior oblique projection was performed routinely; selective coronary angiography was performed if there was any clinical evidence to suggest ischemic heart disease. The accuracy of pressure measurements was checked regularly with a mercury manometer; duplicate measurements of cardiac output in our laboratory have been shown to be reproducible within 6.9% (standard error of the mean).

The severity of mitral stenosis was graded I–IV according to the mitral valve area (> 2.5 cm², 1.6 to 2.5 cm², 1.0 to 1.5 cm² and < 1.0 cm²). The calculation was made from the mean diastolic pressure gradient (pulmonary arterial wedge to left ventricular diastolic pressure), cardiac output and diastolic filling time. If there was trivial mitral regurgitation, indicated by a faint regurgitant jet which rapidly disappeared, the classification was still mitral stenosis but the severity was graded according to the pressure gradient (< 5 mm Hg, 5–10 mm Hg, 11–15 mm Hg, > 15 mm Hg).

The cineangiograms had not been calibrated for measurement of left ventricular volume, hence mitral 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 atrium and ventricle, pulsation of the left atrium and reflux into the pulmonary veins.

Mixed mitral 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. The severity of mitral stenosis was determined from the EF slope of the mitral valve echocardiogram,² from the phonocardiogram,¹¹ and using the echocardiogram, electrocardiogram and chest radiograph to assess the size of the left atrium and the chest radiograph to detect pulmonary venous congestion. If any doubt remained, cardiac catheterization was always performed. The severity of mitral regurgitation was determined by seeking evidence of increased diastolic blood flow across the mitral valve from the clinical examination, echocardiogram,² phonocardiogram and apexcardiogram,¹¹ and evidence of enlargement of the left atrium and left ventricle and of pulmonary venous congestion from the chest radiograph.

Left Ventricular Failure

Patients with severe MR 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. This classification was chosen because clinical left ventricular failure is the usual indication for surgery in our unit and its early detection is a major aim of serial echocardiographic studies. Hemodynamic measurements were always used to support the diagnosis of left ventricular failure and, in cases of doubt, to exclude its presence but were not sufficiently specific to be used to define failure;¹³ furthermore, abnormal hemodynamics in MR have not been validated as an adequate guide to prognosis when considering mitral valve replacement. The left ventricular end-diastolic pressure was 15 mm Hg and cardiac output 3.3 L/min in 21 patients with clinical left ventricular failure and 9 mm Hg and 4.1 L/min in four without failure.

Echocardiography

This investigation was performed using an Ekoline 20 echocardiograph, a 1.27 cm, 2.25 MHz transducer and a Honeywell 1856 Visicord multi-channel oscilloscopic recorder. The technique of the examination has been described in detail previously.³ Four particular care was taken to standardize the pathway of the ultrasonic beam through the left ventricle, to correctly identify the internal and external surfaces of the left ventricular walls and to obtain echoes from these surfaces which were as complete as possible throughout the cardiac cycle.

Echocardiographic measurements were made only on recordings of good quality which allowed accurate measurement of the internal left ventricular dimension at end-diastole (LVIDd) and end-systole (LVIDs). These dimensions were also used to calculate an index of extent of myocardial contraction, fractional shortening (FS):

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

This index is the percentage shortening of the left ventricular internal dimension or minor axis or diameter during systolic contraction. Provided that the left ventricle is approximately circular in cross-section, fractional shortening is the same as shortening of the internal circumference of the chamber — circumferential shortening.³

Left ventricular volumes were derived by cubing LVIDd and LVIDs, then correcting for the error introduced by the more spherical shape of the larger left ventricle.¹⁵ The difference between the end-diastolic volume (LVEDV) and end-systolic volume (LVESV) was the left ventricular stroke volume (LVSV). These volumes were also used to calculate the ejection fraction:

\[
EF = \frac{LVEDV - LVESV}{LVEDV} \times 100\% 
\]

Note that the same echocardiographic measurements were used to calculate both FS and EF. The calculation of FS is simpler and involves no assumptions about the axis ratio of the left ventricular model, while calculation of EF has the advantage of familiarity to those accustomed to calculations of left ventricular volume by angiography.

Other measurements made from the echocardiogram were the end-diastolic thickness of the posterior left ventricular wall (PWTd),¹⁷ the right ventricular internal dimension (RV)¹⁸ and the left atrial dimension at end-systole (LA).¹⁸

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**

**Mitral Stenosis**

The results are summarized in Table 1 and Figure 1. The left ventricular end-diastolic internal dimension was abnormally small only when mitral stenosis was severe (grade IV) (fig. 2 left). The mean posterior wall thickness was normal for all groups.

Myocardial contraction (mean fractional shortening) was reduced in the group which included all patients with mitral stenosis ($P < 0.01$). In fact, the reduction in fractional shortening was similar for all grades of severity and was not related to the presence of atrial fibrillation nor to elevation of pulmonary vascular resistance ($> 3$ Wood Units). In individual patients, this reduction in myocardial contraction was often moderate but rarely severe (fig. 2 right, 3).

The left ventricular stroke volume was subnormal in mitral stenosis ($P < 0.01$) and was significantly lower for grade IV than for grade I stenosis ($P < 0.02$).

The mean left atrial dimension was abnormally increased even in grade I stenosis ($P < 0.01$) but was only slightly

**Table 1.** Echocardiographic Measurements in Mitral Valve Disease

<table>
<thead>
<tr>
<th>Grade</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>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>11</td>
<td>4.3*</td>
<td>30.5</td>
<td>0.9</td>
<td>83.8</td>
<td>57.4</td>
<td>47.4</td>
<td>3.5</td>
</tr>
<tr>
<td></td>
<td></td>
<td>(0.4)</td>
<td>(0.7)</td>
<td>(0.1)</td>
<td>(21.1)</td>
<td>(9.0)</td>
<td>(9.2)</td>
<td>(1.0)</td>
</tr>
<tr>
<td>II</td>
<td>14</td>
<td>4.3</td>
<td>27.1</td>
<td>1.0</td>
<td>88.0</td>
<td>51.9</td>
<td>43.7</td>
<td>4.0</td>
</tr>
<tr>
<td></td>
<td></td>
<td>(0.7)</td>
<td>(6.7)</td>
<td>(0.2)</td>
<td>(34.3)</td>
<td>(11.5)</td>
<td>(16.1)</td>
<td>(0.9)</td>
</tr>
<tr>
<td>III</td>
<td>13</td>
<td>4.3</td>
<td>27.1</td>
<td>1.0</td>
<td>84.8</td>
<td>52.5</td>
<td>42.9</td>
<td>4.4</td>
</tr>
<tr>
<td></td>
<td></td>
<td>(0.5)</td>
<td>(7.3)</td>
<td>(0.2)</td>
<td>(21.2)</td>
<td>(12.4)</td>
<td>(10.8)</td>
<td>(0.7)</td>
</tr>
<tr>
<td>IV</td>
<td>9</td>
<td>3.8</td>
<td>30.3</td>
<td>0.9</td>
<td>62.2</td>
<td>57.1</td>
<td>35.1</td>
<td>4.2</td>
</tr>
<tr>
<td></td>
<td></td>
<td>(0.5)</td>
<td>(5.9)</td>
<td>(0.2)</td>
<td>(19.7)</td>
<td>(5.7)</td>
<td>(9.5)</td>
<td>(0.6)</td>
</tr>
<tr>
<td>All</td>
<td>47</td>
<td>4.2</td>
<td>28.5</td>
<td>0.9</td>
<td>81.2</td>
<td>54.4</td>
<td>54.4</td>
<td>4.0</td>
</tr>
<tr>
<td></td>
<td></td>
<td>(0.6)</td>
<td>(6.7)</td>
<td>(0.2)</td>
<td>(26.5)</td>
<td>(10.3)</td>
<td>(10.3)</td>
<td>(0.9)</td>
</tr>
</tbody>
</table>

*Multiply values with one standard deviation in parentheses.

**Mitral Regurgitation**

<table>
<thead>
<tr>
<th>Grade</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>
</tr>
</thead>
<tbody>
<tr>
<td>Mild</td>
<td>19</td>
<td>4.5</td>
<td>33.6</td>
<td>1.0</td>
<td>92.0</td>
<td>62.2</td>
<td>57.0</td>
<td>2.9</td>
</tr>
<tr>
<td></td>
<td></td>
<td>(0.5)</td>
<td>(5.1)</td>
<td>(0.2)</td>
<td>(25)</td>
<td>(7.0)</td>
<td>(15.9)</td>
<td>(0.6)</td>
</tr>
<tr>
<td>Moderate</td>
<td>24</td>
<td>5.1</td>
<td>34.6</td>
<td>1.0</td>
<td>123.8</td>
<td>64.4</td>
<td>79.9</td>
<td>3.1</td>
</tr>
<tr>
<td></td>
<td></td>
<td>(0.6)</td>
<td>(6.0)</td>
<td>(0.2)</td>
<td>(29)</td>
<td>(8.2)</td>
<td>(20.2)</td>
<td>(0.6)</td>
</tr>
<tr>
<td>Severe — No LVF</td>
<td>16</td>
<td>6.1</td>
<td>38.7</td>
<td>1.2</td>
<td>185.4</td>
<td>67.6</td>
<td>125.5</td>
<td>4.7</td>
</tr>
<tr>
<td></td>
<td></td>
<td>(0.6)</td>
<td>(6.7)</td>
<td>(0.2)</td>
<td>(37.7)</td>
<td>(8.1)</td>
<td>(28.2)</td>
<td>(0.7)</td>
</tr>
<tr>
<td>Severe — LVF</td>
<td>21</td>
<td>6.2</td>
<td>32.4</td>
<td>1.1</td>
<td>201.1</td>
<td>58.9</td>
<td>115.0</td>
<td>4.9</td>
</tr>
<tr>
<td></td>
<td></td>
<td>(0.9)</td>
<td>(8.1)</td>
<td>(0.2)</td>
<td>(63.3)</td>
<td>(11.6)</td>
<td>(34.0)</td>
<td>(1.0)</td>
</tr>
<tr>
<td>Cardiomyopathy</td>
<td>9</td>
<td>6.5</td>
<td>14.8</td>
<td>1.0</td>
<td>217.4</td>
<td>30.6</td>
<td>66.8</td>
<td>4.4</td>
</tr>
<tr>
<td></td>
<td></td>
<td>(0.8)</td>
<td>(2.0)</td>
<td>(0.1)</td>
<td>(57.3)</td>
<td>(3.7)</td>
<td>(21.1)</td>
<td>(1.1)</td>
</tr>
<tr>
<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>
</tr>
<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>
</tr>
</tbody>
</table>

**Figure 1.** Echocardiographic measurements in mitral stenosis: left) left ventricular end-diastolic dimension (LVIDd), right) fractional shortening (FS).
larger in grades II, III and IV which were not significantly different from each other.

Mitral Stenosis and Regurgitation

The results are summarized in table 1. Left ventricular volume overload due to mitral regurgitation was reflected in significant increases above normal in left ventricular end-diastolic internal dimension ($P < 0.01$), end-diastolic volume ($P < 0.01$) and stroke volume ($P < 0.01$). The incidence of impaired myocardial contraction (reduced fractional shortening) was similar to that found in patients with mitral stenosis (fig. 3).

Mitral Regurgitation

The results are summarized in table 1 and figure 4. When regurgitation was mild, mean values for all measurements were normal. When regurgitation was moderate, there were significant increases in left ventricular end-diastolic dimension ($P < 0.05$), end-diastolic volume ($P < 0.05$), stroke volume ($P < 0.05$) and left atrial dimension ($P < 0.05$).

In severe mitral regurgitation without left ventricular failure (fig. 5), the mean values of the left ventricular end-diastolic dimension and posterior wall thickness were both increased by approximately one-third (each $P < 0.01$), while the end-diastolic volume and stroke volume were approximately double the corresponding normal mean values (each $P < 0.01$).

When severe mitral regurgitation was associated with left ventricular failure, the mean values of the left ventricular end-diastolic internal dimension and end-diastolic volume were slightly higher, and for the stroke volume slightly lower, but these differences were not statistically significant. Three patients with severe mitral regurgitation due to rupture of chordae tendineae had a left ventricle of normal size and a stroke volume which was within the normal range.

Figure 2. Left) left ventricular echocardiogram in severe mitral stenosis: incomplete echo contours from chordae tendineae (C) and anterior leaflet of the mitral valve (AMV) indicate that the beam direction has been correctly standardized for measurement of dimensions of the left ventricular cavity (LV), bounded by echoes from the left side of the septum (LS) and endocardial surface of the posterior wall (EN). The chamber is small (LVIDd 3.8 cm), myocardial contraction is at the lower limit of normal (FS 26%) and the stroke volume is reduced (32 ml). Right) Mitral stomasis with impaired myocardial contraction. The left ventricle is of normal size (LVIDd 4.5 cm) but FS is severely reduced (11%). No clinical evidence of rheumatic activity. Left bundle branch block is the cause of paradoxical septal motion.

Figure 3. Reduced myocardial contraction in rheumatic mitral valve disease. The frequency (%) of normal, mild and moderate reduction of fractional shortening (FS) is shown for the control group and for patients with mitral stenosis (MS), mixed mitral valve disease (MS/MR) and mitral regurgitation (MR).
Myocardial contraction (mean value of fractional shortening) was abnormally increased in severe mitral regurgitation without left ventricular failure ($P < 0.05$) but was normal with failure.

**Discussion**

**Left Ventricle in Rheumatic Mitral Valve Disease**

The traditional view is that the left ventricle is small in mitral stenosis because it is chronically underfilled.$^{26}$ Previous studies of left ventricular size by angiography and thermomodulation have yielded conflicting results on this point — some have found such a reduction in left ventricular end-diastolic volume$^{21-23}$ while others have not.$^{24-26}$ However, the numbers of patients in these studies were small and the left ventricular size was not related to the severity of mitral stenosis. Our echocardiographic data indicate that the left ventricle is significantly reduced in size when mitral stenosis is severe.

The association of impaired myocardial function with chronic rheumatic mitral stenosis has long been suspected.$^{27,28}$ The reported incidences of impairment seem to have depended on the sensitivity of the method of detection and have ranged from 3.2% for clinical evidence,$^{29}$ 13% for cardiac catheterization data$^{30}$ and a higher incidence still if cardiac catheterization with exercise stress testing$^{31}$ or left ventricular angiography$^{31,32}$ have been used. Thus Kennedy et al.$^{31}$ found that 37% of patients with mitral stenosis had evidence of impaired myocardial contraction (subnormal angiographic left ventricular ejection fraction); in our echocardiographic data, 30% of patients with mitral stenosis had impaired myocardial contraction (reduced fractional shortening). Furthermore, the incidences of reduced myocardial contraction in mixed mitral valve disease and in mitral regurgitation was similar for both angiographic and echocardiographic investigations. In short, both angiography and echocardiography agree that myocardial contraction is reduced in about one-third of patients with mitral stenosis, in a similar proportion of patients with mitral stenosis and regurgitation, and in a smaller proportion with mitral regurgitation. The reduction is usually moderate but rarely severe (fig. 3).

The cause of impaired myocardial function is obscure$^{33,34}$ and it is not known whether or not it is clinically significant. Nevertheless, it is our impression that it is a contributing factor in some patients who have an unsatisfactory clinical response to mitral valve replacement; it could also contribute to the low cardiac output which sometimes complicates the immediate postoperative management of patients who have had mitral valve replacement for mitral stenosis.

**Left Ventricle in Mitral Regurgitation**

Our echocardiographic estimates of left ventricular size and stroke volume in severe mitral regurgitation are in good agreement with the angiographic measurements of Kennedy et al.$^{21}$ This is not surprising since good correlation has been demonstrated between left ventricular volume measured by echocardiography and by angiography; this correlation also explains the increase in echocardiographic size and stroke volume measured by echocardiography coincident with the increase in severity of mitral regurgitation.

The potentially misleading association of a normal left ventricular size in severe mitral regurgitation of acute onset has been previously demonstrated by angiography.$^{35}$ It is important to note that circumferential myocardial contraction (fractional shortening) was, on average, greater than normal in patients with severe mitral regurgitation who were not in left ventricular failure but normal in the presence of failure. These findings are in striking contrast to other forms of chronic heart disease with left ventricular failure in which we have observed marked reduction in myocardial contraction. Thus reduced values of fractional shortening are associated with left ventricular failure due to cardiomyopathy (11%), aortic stenosis (19%), chronic aortic regurgitation (21%).$^{7}$ A likely explanation for this difference is that severe mitral regurgitation represents a low impedance systolic leak into the left atrium which tends to reduce left ventricular afterload, increase myocardial contraction and reduce the end-systolic volume.$^{36-39}$ Thus in severe mitral regurgitation with left ventricular failure, myocardial contraction would be affected by two opposing influences — the tendency of severe regurgitation to increase myocardial shortening and of impaired myocardial function due to prolonged overload to reduce shortening. The important implication of this observation is that a normal value of

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**Figure 4.** Echocardiographic measurements in mitral regurgitation: left) left ventricular end-diastolic dimension (LVIDd), right) fractional shortening (FS).
fractional shortening or ejection fraction in a patient with severe mitral regurgitation could represent impaired myocardial function and a reduced value could represent more severe myocardial impairment than would the same value recorded in a patient with cardiomyopathy, aortic stenosis or chronic aortic regurgitation. In this way, severe mitral regurgitation tends to mask impaired myocardial contraction.

If the clinical distinction between cardiomyopathy with moderate secondary (or associated) mitral regurgitation and severe primary mitral regurgitation was difficult but could be made by left ventricular angiography, the distinction could also be made by echocardiography. Thus the group with mitral regurgitation secondary to cardiomyopathy had severe reduction of fractional shortening and only a slight increase in the stroke volume (fig. 5). Conversely, in our experience, if the distinction between primary and secondary mitral regurgitation remained equivocal after a technically satisfactory echocardiogram, left ventricular angiography was not able to solve the problem.
Conclusions

The main conclusions drawn from this study which could help in the interpretation of echocardiographic data are: (a) impairment of myocardial function is common in patients with rheumatic mitral valve disease but is rarely severe; (b) the left ventricle is abnormally small in mitral stenosis only when stenosis is severe; (c) left ventricular failure can occur in mitral regurgitation with normal myocardial contraction; (d) the size and contraction of the left ventricle may be normal in mitral regurgitation of acute onset.

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I G McDonald

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