M-mode echogram as a means of distinguishing between mild and severe mitral stenosis

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SUMMARY Fifty-two patients with pure mitral stenosis (27 with severe stenosis and 25 with mild stenosis) were studied to assess the ability of different M-mode echocardiographic measurements to separate mild and severe disease. Variables related to valve motion, for example diastolic closure rate, the mitral valve closure index, and the amplitude of valve motion, accurately divided patients with mitral stenosis from normal subjects but did not distinguish usefully between mild and severe disease. In contrast, variables dependent on left ventricular dimension change in diastole, for example the rapid filling period and the peak rate of left ventricular diastolic dimension change, accurately separated mild and severe disease. No patient with severe mitral stenosis had a rapid filling period, whereas 21 of the 25 patients with mild disease did have one. The peak rate of left ventricular diastolic dimension change was <10 cm/s or <2.4 cm/s per cm when normalised for left ventricular dimension in all patients with severe disease and in only six of the 25 patients with mild disease.

One of the first uses of M-mode echocardiography was the evaluation of mitral stenosis.¹ Despite this the accurate assessment of the severity of mitral stenosis has proved difficult and several different techniques have been described. These techniques depend either on measurements of mitral valve motion in diastole, such as the diastolic closure rate of the valve¹ or the more complicated mitral valve closure index,² or on the assessment of left ventricular diastolic dimension changes or wall motion either directly^{3,4} or indirectly.^{5,6} Conflicting evidence has been produced for the usefulness of these variables.⁵⁻⁷

Many studies involve the measurement of the relative degree of the severity of stenosis among groups of patients all of whom have severe disease whereas often the important clinical question is whether the stenosis is mild or severe. The present study was designed to reassess M-mode echocardiography as a means of answering this important clinical question.

Patients and methods

The study was prospective. Fifty-eight patients were originally selected for the study, before an echocardiogram was performed, and were later allocated to a

mild or severe group depending on catheter, operative, and clinical findings.

Thirty-six consecutive patients with severe symptoms (New York Heart Association class III or IV) and signs of mitral stenosis were studied. Four of these patients were excluded because the subsequent echocardiogram was technically unsatisfactory. Of the remaining 32 patients, 27 made up the severe group and five were assigned to the mild group (see below).

GROUP 1: SEVERE MITRAL STENOSIS

(a) In 24 patients severity was assessed at operation. In all the mitral valve area was less than 1.5 cm^2 and all but three had valve areas of less than 1 cm^2 .

(b) A further three patients had severe mitral stenosis confirmed by cardiac catheterisation; all had an end-diastolic gradient of more than 5 mmHg at rest at a heart rate of 70/min, a mitral valve mean gradient of greater than 10 mmHg, and a pulmonary artery wedge mean pressure of greater than 15 mmHg. An accurate valve area could not be calculated from these data since reliable cardiac output measurements were not available, but the haemodynamic data were similar to those gathered in the 16 patients who had been catheterised and in whom the valve area was measured at operation. These three patients did not have surgery: one died after catheterisation while awaiting operation, one refused operation, and the

third had poor but co-ordinate left ventricular function with normal coronary arteries. In this last patient poor left ventricular function had been identified by echocardiography before catheterisation.

(c) No patient had clinical, operative, or catheter evidence of organic tricuspid valve disease, mitral regurgitation, or aortic valve disease.

GROUP 2: MILD MITRAL STENOSIS

The patients in this group were selected in one of two ways.

(a) Five patients with severe symptoms were shown to have mild disease at catheterisation. In all there was, at most, a trivial mitral valve gradient and a minor increase in the pulmonary artery wedge pressure at rest and on exercise. In one case symptoms were the result of poor left ventricular function and in the other four of chronic obstructive airways disease. Two of these patients had had previous mitral valvotomies.

(b) Twenty-two patients were selected on clinical grounds from an outpatient follow-up clinic using the criteria below. Two subsequently had technically unsatisfactory echocardiograms and were excluded from the study. Of the remaining 20 patients, 13 had had previous mitral valvotomies. For inclusion in this group patients had to be asymptomatic, leading active lives, taking no treatment other than digitalis and oral anticoagulants, and had to have physical signs of mild mitral stenosis with a late opening snap, and a short mid-diastolic and/or presystolic murmur. In addition, none had evidence of right axis deviation or right ventricular hypertrophy on the electrocardiogram or abnormal lung fields on the chest x-ray film. Finally, none had gross obesity or chronic lung disease that might have obscured the auscultatory findings.

Patients with symptoms and signs suggesting moderate stenosis were not included in this study since such a group would have been impossible to define using clinical findings alone and all 21 patients catheterised had either severe or very mild stenosis.

Echocardiograms

M-mode echocardiograms were recorded at a paper speed of 75 or 100 mm/s using either a Cambridge or Picker echocardiograph. These echocardiograms were recorded to provide clear echoes from the anterior and posterior cusps of the mitral valve throughout diastole and clear continuous echoes from the left side of the interventricular septum and the left ventricular posterior endocardium. On occasions both mitral valve and left ventricular echoes were obtained on the same recording but generally the echo beam had to be directed slightly more towards the aortic root when recording the mitral valve than when recording the left ventricle.

Echocardiographic measurement

(A) VALVE-RELATED VARIABLES

(1) Diastolic closure rate

This was measured in the standard manner by drawing a tangent to the earliest part of the mitral valve E-F slope.¹

(2) Amplitude of valve motion

The forward excursion of the anterior cusp of the mitral valve during early diastole from its fully closed to fully open position was measured, as was the maximum separation of the anterior and posterior cusps of the valve.

(3) Mitral valve closure index

The mitral valve closure index was measured by the method of Shiu.² This is a measurement of the movement of the anterior and posterior mitral valve cusps relative to each other during diastole, and is said to correlate closely with the severity of mitral stenosis.

(B) VENTRICLE-RELATED VARIABLES

The left ventricular echocardiogram was digitised using a Graf/pen digitiser and this information was processed using a PDP 8 computer to derive a plot of left ventricular dimension against time, the rate of change of left ventricular dimension, and this rate of change normalised for instantaneous left ventricular dimension. This is similar to the method described by Gibson and Brown.³ The following measurements were made from these traces:

(1) Maximum and minimum left ventricular dimension

(2) Presence or absence of a definite rapid filling period during early diastole

This could be detected by eye as an alteration of the slope of the left ventricular dimension trace and confirmed by examination of the tracing of rate of change of left ventricular dimension (Fig. 1). A definite rapid filling period was considered to be present when the rate of change of left ventricular dimension in the first third of diastole was at least three times the rate of change of dimension either immediately before the onset of the next ventricular systole in atrial fibrillation or at the onset of atrial contraction in sinus rhythm in those patients in whom atrial systole produced an increase in the rate of left ventricular dimension change.

(3) Presence or absence of early diastolic motion of the septum in posterior direction

All measurements are expressed as the mean $\pm$ one standard deviation. Statistical comparison of groups of variables were made using Student's *t* test for unpaired data, and the relative incidence of a particular finding in the two groups was compared using the χ^2 test.

Fig. 1 Digitised echocardiograms from a normal subject (left) and a patient with severe mitral stenosis. From below upwards the panels represent, (1) tracing of original echocardiogram; (2) left ventricular dimension; (3) rate of change of left ventricular dimension; (4) rate of change of left ventricular dimension normalised for instantaneous left ventricular dimension.

ANALYSIS OF ECHOCARDIOGRAMS

All echocardiographic measurements were made by AA who had no knowledge of the clinical state of the patients and played no part in the clinical decisions involving any of them.

Results

VALVE-RELATED VARIABLES

The values of these variables in the two groups are shown in Table 1, and the diastolic closure rate is also shown in Fig. 2. Though both the total amplitude of

Table 1 Valve-related variables

	Severe n=27	Mild n=25	
Maximum cusp separation (mm)	14.5±3.5	18.0±3.6	p<0.001
Anterior cusp motion (mm)	13.5±4.2	15.9±3.7	p<0.05
Diastolic closure rate (mm/s)	14.0±7.0	27.0±13.0	p<0.001
Mitral valve closure index (%)	63±38	78±50	NS

NS, not significant (p>0.05).

anterior mitral valve cusp diastolic motion and the maximum separation of the anterior and posterior valve cusps were significantly less in the severe than the mild groups, there was so much overlap between the two groups that these variables are not useful in their differentiation. Similarly, the diastolic closure rate was significantly less (p<0.001) in the severe group than in the mild group but overlap was considerable. Despite this overlap the diastolic closure rate gave some separation between the groups. A diastolic closure rate of <10 mm/s identified 12 of the 27 patients with severe disease and only two patients with mild disease. At the other end of the range only one patient with severe disease had a diastolic closure rate of >24 mm/s, while 13 of the 25 patients with mild disease had values of >24 mm/s. If absolute separation is required then only seven

Fig. 2 Mitral valve diastolic closure rate (DCR) in patients with mild and severe mitral stenosis. The vertical bars equal mean $\pm$ 1 SD.

patients with mild disease and three with severe disease could be identified with certainty (Fig. 2).

VENTRICLE-RELATED VARIABLES

Ventricular dimensions and peak rates of systolic dimension change were the same in both groups. When the presence or absence of a rapid filling period and the peak rates of diastolic dimension change are compared, however, there are striking differences between the groups (Table 2).

Table 2 Ventricle-related variables

	Severe n=27	Mild n=25	
<i>Peak LV diastolic dimension change</i>			
A cm/s	6.9±1.7	12.0±4.0	p<0.001
B cm/s per cm	1.8±0.3	2.9±0.9	p<0.001

LV, left ventricular.

Fig. 3 Peak diastolic rate of change of left ventricular dimension (D) in patients with mild and severe mitral stenosis. The horizontal dotted lines represent the normal limits and the vertical bars equal the mean ± 1 SD.

Fig. 4 Peak diastolic rate of change of left ventricular dimension (D) normalised for instantaneous left ventricular dimension in patients with mild and severe mitral stenosis. The horizontal dotted lines represent the normal limits and the vertical bars equal the mean ± 1 SD.

(1) Rapid filling period

None of the patients with severe disease had a rapid filling period, whereas 21 of the 25 patients with mild disease showed a definite rapid filling period (χ^2 test $p<0.001$).

(2) Septal motion in early diastole

A posterior movement of the interventricular septum in early diastole is common in severe mitral stenosis. This movement was seen in 25 of the 27 patients with severe disease but in only nine of the 25 patients with mild disease (χ^2 test $p<0.001$).

(3) Peak rate of diastolic dimension change

Both the peak rate of diastolic dimension change and this variable normalised for left ventricular dimension produced good separation of the two groups (Fig. 3 and 4). The lower limit of these variables was previously determined in 50 normal subjects as 10 cm/s and 2.4 cm/s per/cm, respectively. Of the 27 patients with severe disease, 26 fell below the lower limit of normal for each variable and the patient falling just above the lower limit of normal was different for the two groups. Therefore, a combina-

tion of the two variables correctly identified all the patients with severe disease. Only six of the 25 patients with mild disease had values for either of these variables that fell below the normal range (Fig. 3 and 4).

Discussion

Valve-related variables, though significantly different in patients with mild and severe stenosis, did not discriminate well enough between the two groups to be clinically useful. One factor that may have determined this result was the large number of patients in the mild group who had had previous valvotomies. We examined separately, however, the findings for patients who had and had not had valvotomies and did not find that valve-related variables were any more reliable in patients who had not had a previous valvotomy. Moreover, the inclusion of patients who have had mitral valvotomies is important since restenosis is now a prominent and common diagnostic problem. In contrast the ventricle-related variables, peak rate of left ventricular diastolic dimension change, and the presence or absence of a rapid filling period, allowed identification of all patients with severe disease. Use of a combination of both types of variable did not enhance the distinction between the groups because of the considerable overlap between the two groups seen with the valve-related variables.

The conclusions of this study depend on the validity of the way in which the patients were divided into the mild and severe groups using a variety of non-echocardiographic criteria. Patients were entered into the study before the echocardiograms were performed, though the results of these were available to clinicians making clinical decisions about surgery. This is important since otherwise a serious bias could have been introduced into the study if only patients with certain predetermined echocardiographic findings were submitted to investigation and/or surgery. Though there is no doubt about the classification of patients in the severe group, the composition of the mild group must be examined in more detail. Ideally we would have preferred to study a large group of patients with mild disease confirmed by catheterisation. Few patients with mild mitral stenosis, however, are catheterised since clinical assessment is usually correct. Five patients in this group were in fact catheterised because of severe symptoms (resulting from chronic lung disease in the majority) but the remaining patients had to be selected on clinical grounds. Using such a method of selection some misclassification is inevitable. The selection criteria were designed to avoid patients who had any symptoms and in whom mitral stenosis of moderate

severity might be present. Patients with severe mitral stenosis, however, may be asymptomatic, and occasionally the severity of mitral stenosis may be underestimated from the physical signs. Nevertheless, the criteria used for the 20 uncatheterised patients implied that a patient with severe stenosis would have to have had no symptoms, normal lung fields on the chest x-ray film, and misleading physical signs. Though any one of these features is not uncommon by itself, the combination of all three is exceedingly so. Patients with severe stenosis but no symptoms usually have a well maintained cardiac output and obvious physical signs, whereas the commonest cause of "occult" mitral stenosis in patients of normal build is a low cardiac output with reduced flow across the valve, and such patients are usually symptomatic. Therefore, while accepting that a very small number of the patients in our mild group may have had the severity of their disease underestimated there cannot have been many and their possible presence does not detract from the finding that ventricle-related variables were a better predictor of severity than valve-related variables.

Our findings must be considered in their clinical context. Echocardiographic findings are never the only factor on which clinical decisions are based. In the straightforward, typical case of severe mitral stenosis they may have little importance other than ruling out an alternative diagnosis such as left atrial myxoma, and the decision to operate can be made purely on the physical signs, chest x-ray film, and the electrocardiogram. In a more difficult case where the patient is severely symptomatic but there is doubt about the severity of stenosis, not only echocardiographic evidence but also the results of other non-invasive tests are used to determine the correct course of action. No single non-invasive investigation is an infallible guide to severity and even cardiac catheterisation data⁸ can on occasion be misleading. If a severely symptomatic patient has an echocardiogram suggesting mild mitral stenosis, other explanations should be sought for the symptoms, such as disordered pulmonary or ventricular function. If no other obvious cause is found, the echocardiographic findings should be disregarded and catheterisation undertaken if appropriate. Similarly, if the echocardiogram suggests severe disease but there are incongruous features such as unimpressive physical signs or an unremarkable chest x-ray, further investigations, usually including catheterisation, are necessary before undertaking operation. The patients in this study were selected to include only those with mild or severe disease since we did not have enough patients with mitral stenosis of moderate severity confirmed by catheterisation to constitute a group and could not define such a group clinically. Such patients

may be severely symptomatic particularly if they have additional cardiorespiratory disease. Though such a group was not studied, clinical mistakes will not be made, regardless of whether their echocardiographic findings are in the mild or severe range, if all available clinical and non-invasive data are considered and catheterisation used if necessary.

If the ventricle-related variables described are to be used, it is important that extremely high quality echocardiograms are obtained and that any tracings falling short of this standard are rejected. It is particularly important that the septum is accurately recorded in early diastole since at this time the septum moves posteriorly almost parallel with the posterior wall in most patients with severe mitral stenosis. If this posterior motion is not recorded accurately then a falsely high peak rate of left ventricular diastolic dimension change is measured and a false rapid filling period may be created. Recently a simple measurement of posterior wall motion alone has been suggested as a method of assessing the severity of mitral stenosis⁹ but further studies are required to assess its value.

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