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Echocardiographic assessment of left ventricular filling after mitral valve surgery

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In order to investigate the functional effects of mitral valve surgery, echocardiograms showing left ventricular dimension were recorded and digitised in 14 normal subjects and 129 patients after mitral valve surgery. Measurements were made of peak rate of increase of dimension (dD/dt) and duration of rapid filling, studies on left ventriculograms in 36 patients having shown close correlation between these values and changes in cavity volume. In 14 patients with mitral stenosis, peak dD/dt was reduced to 7·2±1·5 cm/s, and filling period prolonged to 330±65 ms, compared with normal (16·0±3·2 cm/s, and 160±50 ms, respectively), and after mitral valvotomy, these values improved significantly (10·4±2·7 cm/s and 245±55 ms). Characteristic abnormalities were found in 67 patients with mitral prostheses. Values for the Björk-Shiley (10·5±4·2 cm/s and 180±80 ms) and Hancock (10·3±3·7 cm/s, 245±80 ms) valves were similar, and both superior to the Starr-Edwards (7·4±3·0 cm/s, 295±105 ms). Results after mitral valve repair in 30 cases were not significantly different from normal (14·4±5·0 cm/s, 170±50 ms). Values outside the 95 per cent confidence limits for the valve in question allowed diagnosis of valve malfunction in 18 cases. The method is of value in comparing different operative procedures and in following up patients after mitral valve surgery.

The success of mitral valve repair and replacement is usually judged on clinical grounds and on the results of cardiac catheterisation performed at variable times after operation (Goodale *et al.*, 1955; Morrow *et al.*, 1967). Relief of symptoms and increased exercise tolerance correlate with reduction in pulmonary arterial and left atrial pressures, and with increased cardiac output (Braunwald *et al.*, 1965; Feigenbaum *et al.*, 1968; Hultgren *et al.*, 1968). More recently, echocardiography has made qualitative assessment of the mitral valve possible in terms of cusp thickness and mobility, and characteristic changes have been recorded after mitral valvotomy (Edler, 1967; Effert, 1967; Gustafson, 1967). Mitral prostheses have also been studied using this technique, and the appearance of abnormal echoes or of reduced disc or poppet mobility has confirmed the diagnosis of malfunction (Johnson *et al.*, 1970; Popp and Carmichael, 1971). In the present study, we have attempted to use echocardiography to assess patients postoperatively, not by direct observation of the mitral valve or prosthesis, but by analysis of left ventricular wall movement during diastole. This has allowed us to define specific patterns of wall movement for each of the procedures to the mitral valve, thus establishing a basis for recognition of valve obstruction or regurgitation. In addition, by concentrating on the physiology of ventricular filling, the results can be expressed in a way which allows functional comparison between these different surgical procedures.

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Subjects

ECHOCARDIOGRAPHY

Studies were made on 143 patients who fell into 6 groups.

Group 1

Fourteen patients with mitral stenosis underwent closed mitral valvotomy. Eight were women. Their ages ranged from 17 to 45 years. Eight were in sinus rhythm and 6 in atrial fibrillation. Echocardiograms were recorded preoperatively and 3 to 7 days postoperatively.

Group 2
Thirty patients underwent mitral valve repair. This operation has been described elsewhere (Burr *et al.*, 1976), and involves placing two double semicircular sutures around the mitral valve ring; at the same time, an open commissurotomy may be performed, ruptured chordae corrected, redundant cusp tissue excised, or adherent chordae and papillary muscles separated down the ventricular wall. It was applied, where practicable, to patients with degenerative or rheumatic mitral valve disease, the latter stenotic or regurgitant. Of the 30 patients, 20 were female, their ages ranged from 12 to 73 years. Of the 30 patients 17 had rheumatic mitral valve disease, and 13 had ruptured chordae tendineae or systolic prolapse causing severe mitral regurgitation. Observations were made within 2 years of operation.

Group 3
Thirty patients had normally functioning mitral Starr-Edwards prostheses, and of these 20 were women. Their ages ranged from 18 to 71 years. In 26 patients, mitral valve replacement had been performed for rheumatic mitral valve disease and in 4 for ruptured chordae. Valves of size 3 or 4 composite seat (series 6310 or 6320) or old type (series 6120) were used, and patients were studied between 3 months and 10 years of operation.

Group 4
Thirty patients had normally functioning Björk-Shiley prostheses and of these 16 were female. Their ages ranged from 10 to 68 years. Of the 30 patients, 17 had rheumatic heart disease, 10 had ruptured chordae, and 3 had previous Starr-Edwards prosthetic mitral valve replacement. A size 29 or 31 mm prosthesis was used and patients were studied 3 months to $3\frac{1}{2}$ years after operation.

Group 5
Seven patients had normally functioning Hancock prostheses, and of these 5 were female. Their ages ranged from 15 to 52 years. Four had had rheumatic mitral valve disease, and 3 ruptured chordae tendineae. Patients were studied between 3 months and 2 years of operation.

Group 6
Eighteen patients had evidence of abnormal postoperative valve function. Of these, 14 had a severe paraprosthetic leak associated with a Starr-Edwards prosthesis, 2 had a clotted Björk-Shiley prosthesis, and 2 had abnormal mitral valve function after repair, caused by stenosis in one and by regurgitation in the other. These findings were all confirmed at a second operation.

Normal subjects
Observations were also made in 14 subjects (10 of whom were male) with ages ranging between 13 and 56 with no clinical evidence of heart disease.

ANGIOGRAPHY
Left ventricular angiograms of 36 patients were studied. These fall into three groups.

Group A
Ten patients who complained of chest pain had normal haemodynamics, normal left ventricular function, and normal coronary angiograms at cardiac catheterisation and were referred to as a 'normal' group.

Group B
Ten patients had rheumatic mitral valve disease.

Group C
Sixteen patients had a mitral Starr-Edwards prosthesis, and were reinvestigated because of suspected malfunction. Significant paraprosthetic regurgitation was present in 5 patients.

Methods

ECHOCARDIOGRAPHY
All echocardiograms were made using the Ekoline 20 ultrasonoscope. Recordings were made photographically from a Cambridge multichannel or Honeywell 1856 strip-chart recorder at a paper speed of 100 mm/s, with a simultaneous electrocardiogram. All patients were studied recumbent, and rotated slightly to the left side. Echocardiograms of the left side of the septum and posterior left ventricular wall were obtained at the level of the mitral valve, or just below the level of the prosthesis. Measurements were made only on patients in whom clear continuous echoes were obtained simultaneously from the left side of the septum and posterior wall. Echocardiograms were digitised as described by Gibson and Brown (1973), on a Summagraphics digitising table, and were processed by a Prime 300 computing system. Instantaneous left ventricular dimension (D) and its peak rate of change (dD/dt) (Gibson and Brown, 1973) were derived from the echocardiograms. In the group of patients undergoing mitral valve repair, the echocardiogram of the anterior cusp of the mitral valve during diastole was also recorded and digitised, as previously described by Upton *et al.* (1976). Left ventricular filling pattern was assessed from the value of the peak rate of increase of left ventricular dimension and also from the time interval from minimum dimension until the filling rate

had fallen to 20 per cent of its peak value at the end of the rapid filling period.

ANGIOCARDIOGRAPHY

Angiocardiograms were digitised as previously described (Gibson and Brown, 1975). The superimposed outlines of the left ventricular cavity in successive cine frames were displayed on a Tektronix 4010 video display unit, and a cursor was positioned across the cavity, approximately perpendicular to the long axis, at its mid-point, to define a minor dimension. Plots were then made of this minor dimension (D) and its rate of change with time (dD/dt). Cavity volume (V) was derived from projected area by the area length method (Sandler and Dodge, 1968) and its rate of change (dV/dt) also plotted. From these graphs were derived the following: (1) End-diastolic and end-systolic dimensions and volumes; (2) peak diastolic dD/dt and dV/dt; and (3) the time interval between the onset of diastolic increases in dimension and in volume, peak rates of increase, and the time of reduction to 20 per cent peak value (Fig. 1).

The statistical significance of differences between means was assessed by Student's t test.

Results

ANGIOGRAPHY

There was no significant difference between the timing of minimum dimension and of cavity area in any of the patient groups studied. In normal subjects, the peak rate of increase of dimension occurred 170 ±25 ms after minimum dimension, and the corresponding value for cavity area was 190 ±15 ms; the difference (20 ±20 ms) was significant ($P < 0.01$). In patients with mitral stenosis, peak rate of increase of dimension occurred at 120 ± 40 ms, and of area 105 ±40 ms after the respective minimum values, and for patients with mitral prostheses the corresponding figures were 140 ± 40 ms and 120 ±50 ms; the latter two differences were not statistically significant.

In the group as a whole, there was very close correlation between the time to 20 per cent filling rate as measured from the dimension (TD) and from the area traces (TA) given by the regression equation

TA = 0·99TD + 8 ms, r = 0·96, SEE = 15 ms (Fig. 2).

Peak filling rate was derived from the area trace as a rate of increase of volume (dV/dt) and was related to peak rate of increase of dimension (dD/dt) in the whole group by the regression equation

Peak dV/dt = 22 peak dD/dt + 340 ml/s, r = 0·48, SEE = 210 ml/s.

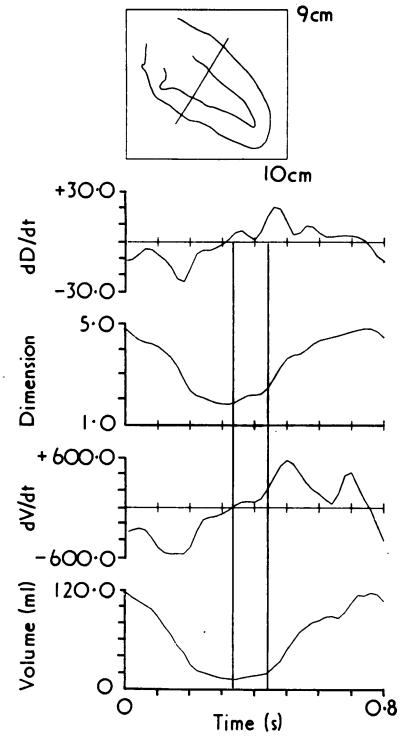

Fig. 1 *Relation between changes in left ventricular volume and transverse dimension in a normal subject, determined from a left ventricular angiogram. From below are shown cavity volume, rate of change of volume, transverse dimension, rate of change of dimension, and (top) end-diastolic and end-systolic cavity outlines, with the position of the dimension indicated. The vertical lines represent minimum cavity area and mitral valve opening.*

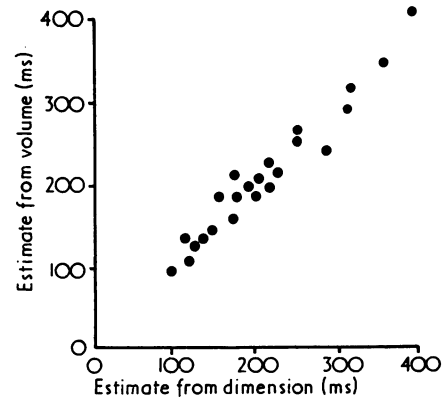

Fig. 2 *Comparison of estimates of time to 20 per cent peak filling rate (left ventricular filling period) from transverse dimension and cavity volume, derived from left ventricular angiograms. There is close correlation between the two.*

When only those patients in whom the left ventricular end-diastolic dimension was less than 6 cm were considered, the equation was

Peak dV/dt=43 peak dD/dt–50 ml/s, r=0·81, SEE=70 ml/s (Fig. 3).

ECHOCARDIOGRAPHY

These results are given in detail in the Table.

Normal subjects

The peak rate of increase of dimension measured from the echocardiogram was 16·0 ±3·2 cm/s (mean ± 1 standard deviation). This occurred 50 ±16 ms after the time of minimum dimension, and the rate of increase of dimension had declined to 20 per cent of its peak value by 160 ±50 ms (Fig. 4). These values, from strip-chart recordings, are similar to but rather higher than those measured from echocardiograms taken on polaroid film at a lower sweep speed (Gibson and Brown, 1973).

Mitral stenosis

Preoperatively the peak rate of increase of dimension was reduced compared with the normal group to 7·2 ±1·5 cm/s (P < 0·001), and the filling period significantly prolonged, so that the rate of increase of dimension had not dropped to 20 per cent of its peak value before 330 ±65 ms (P < 0·001). After operation, the peak rate of increase of dimension had increased to 10·4 ±2·7 cm/s, which was significantly greater than before operation (P < 0·01), though less than normal (P < 0·01). Similarly, the time to 20 per cent peak filling rate fell to 245 ±55 ms, significantly less than preoperatively (P < 0·01), though greater than normal (P < 0·01) (Fig. 5). Peak mitral diastolic closing rate was 20 mm/s preoperatively, increasing to 37 mm/s after operation (P < 0·01).

Starr-Edwards prosthesis

The peak rate of increase of dimension was 7·4 ± 3·0 cm/s, significantly less than normal (P < 0·001),

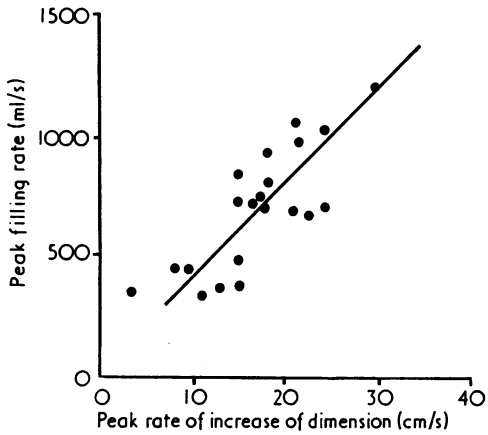

Fig. 3 *Comparison between estimates of peak filling rate and peak rate of increase of transverse dimension in normal patients and those with mitral valve disease or mitral prostheses, in whom end-diastolic transverse dimension was less than 6 cm.*

Table

Valve	*No. of patients*	*Peak dD/dt** (cm/s)	*Time to 20% peak dD/dt (ms)**
Normal	14	16·0 ±3·2	160 ±50
Mitral stenosis	14	7·2 ±1·5	330 ±65
Mitral valvotomy	14	10·4 ±2·7	245 ±55
Mitral repair	30	14·4 ±5·0	170 ±50
Starr-Edwards	30	7·4 ±3·0	295 ±110
Björk-Shiley	30	10·5 ±4·2	180 ±80
Hancock xenograft	7	10·3 ±3·7	245 ±80
Leaking Starr-Edwards	14	17·0 ±2·0	240 ±60

*Mean values ± standard deviation.

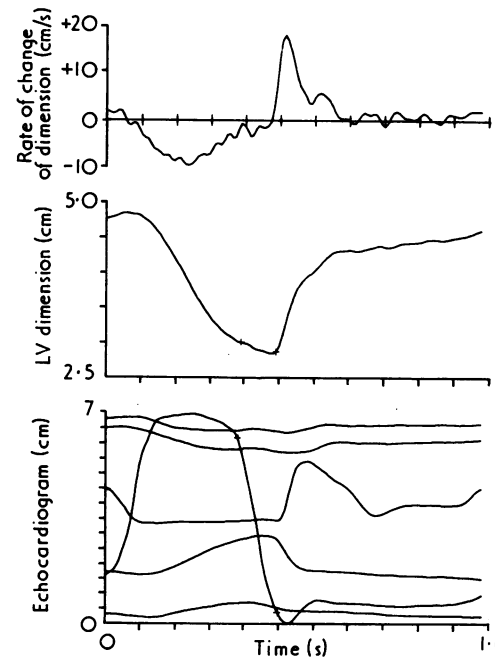

Fig. 4 *Digitised left ventricular echocardiogram from a normal subject, showing (from below) digitised data with superimposed apex cardiogram, left ventricular dimension, and (top) rate of change of dimension. The crosses represent the timing of aortic valve closure and mitral valve opening.*

and the filling period was prolonged to 295 ±110 ms, significantly greater than normal ($P < 0.01$) (Fig. 6). It was not possible to differentiate between the three valve models present in the patients from the pattern of left ventricular wall movement.

Björk-Shiley prosthesis

The peak rate of increase of dimension was 10·5 ± 4·2 cm/s, significantly less than normal ($P < 0.01$) though greater than in the patients with a Starr-Edwards prosthesis ($P < 0.001$). The time to 20 per cent peak rate of increase was 180 ±80 ms, which was not different from normal, though significantly less than in patients with a Starr-Edwards prosthesis ($P < 0.001$) (Fig. 7).

Hancock xenografts

The peak rate of increase of dimension was 10·3 ± 3·7 cm/s, similar to that in patients with a Bjørk-Shiley prosthesis though significantly less than normal ($P < 0.01$) and greater than in patients with a Starr-Edwards prosthesis ($P < 0.05$). In contrast to patients with a Björk-Shiley prosthesis, however, the time to 20 per cent peak rate was significantly prolonged to 245 ±80 ms ($P < 0.01$ with respect to normal).

Mitral valve repair

In this group of patients, peak rate of increase of dimension and the time to 20 per cent peak rate were both normal (14·4 ±5·0 cm/s and 170 ±50 ms, respectively). In 20 of the 30 patients satisfactory measurements of peak diastolic closure rate were made; in them, this ranged from 50 to 240 m/s (mean 95 ±60 mm/s), significantly lower than normal (mean 250 ±60 mm/s) ($P < 0.01$). There was no relation in this group of patients between peak diastolic closure rate and the pattern of left ventricular wall movement and, in particular, a low mitral valve diastolic closure rate did not imply an abnormal pattern of increase of dimension during diastole (Fig. 8).

Postoperative mitral valve malfunction

In the 14 patients with a Starr-Edwards prosthesis and a paraprosthetic leak, peak rate of increase of

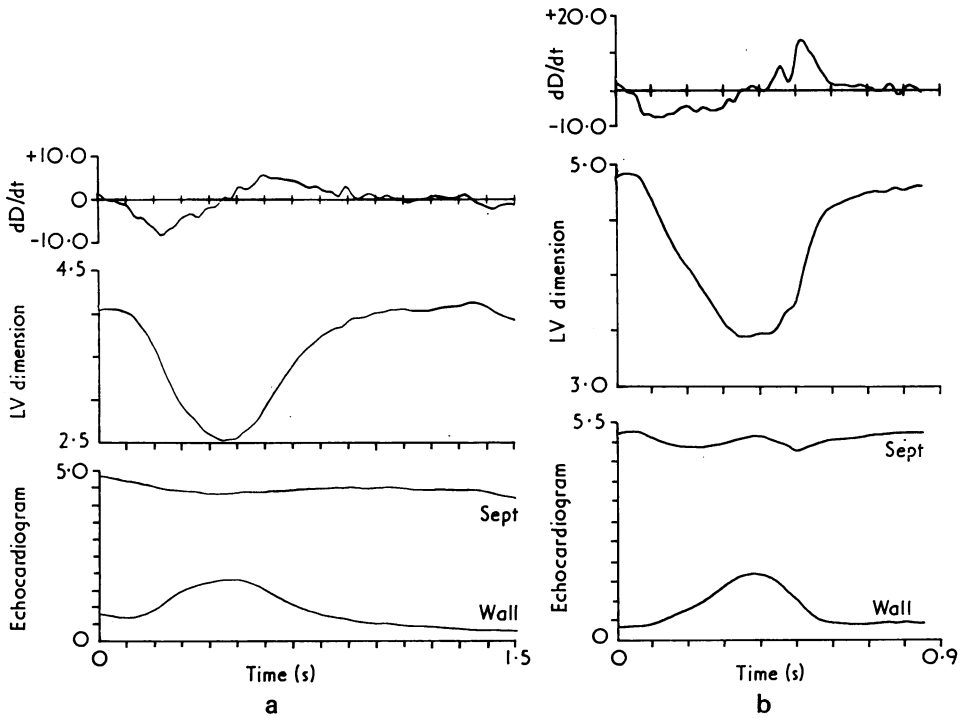

Fig. 5 (a) *Digitised left ventricular echocardiogram from a patient with mitral stenosis, showing (from below) septal and posterior wall echoes, transverse dimension, and (top) rate of change of dimension. Peak rate of change of dimension is reduced and the time to 20 per cent peak filling is prolonged.* (b) *Digitised echocardiogram from the same patient after closed mitral valvotomy.*

dimension was increased to 17·0 ±2·0 cm/s, significantly higher than in patients with a normally functioning Starr-Edwards prosthesis ($P < 0.01$), with almost no overlap between the two groups of patients. The time to 20 per cent peak filling rate, however, remained prolonged (240 ±60 mm/s), presumably reflecting the increased stroke volume. In the 2 patients with a thrombosed Björk-Shiley prosthesis, the peak rate of increase of dimension was 4 and 5 cm/s and the filling time to 20 per cent was 230 ms. All these values are outside the 95 per cent confidence limits for the normally functioning valve.

Discussion

The present study has shown that, after successful mitral valve surgery, the pattern of left ventricular wall movement as assessed by M-mode echocardiography may be very abnormal. Peak rate of increase of left ventricular dimension is frequently reduced, the duration of filling prolonged, and septal movement reversed. The magnitude of these changes can be related to the surgical procedure undergone by the patient; the most abnormal values were recorded after Starr-Edwards replacement, whereas after mitral valve repair, the measurements were not significantly different from normal.

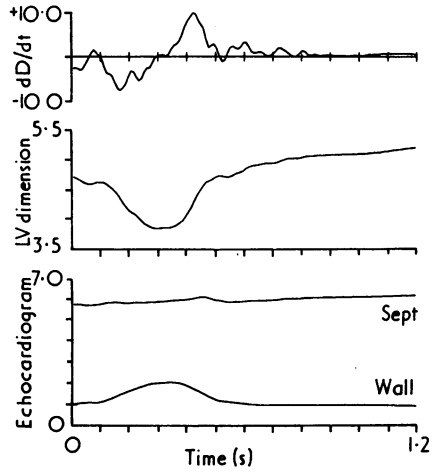

Fig. 7 *Digitised left ventricular echocardiogram from a patient with a mitral Bjork-Shiley prosthesis. Layout as Fig. 5.*

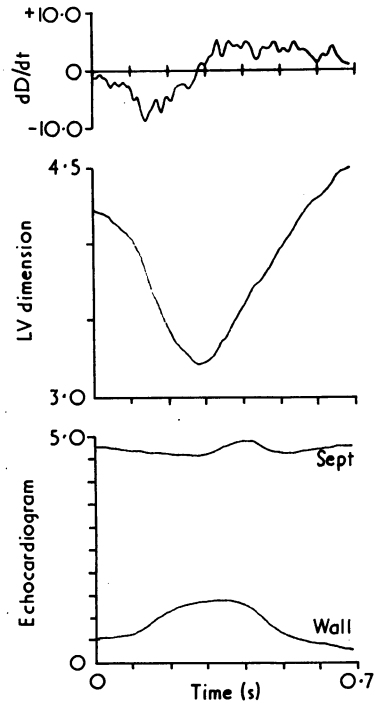

Fig. 6 *Digitised left ventricular echocardiogram from a patient with a mitral Starr-Edwards prosthesis. Layout as Fig. 5.*

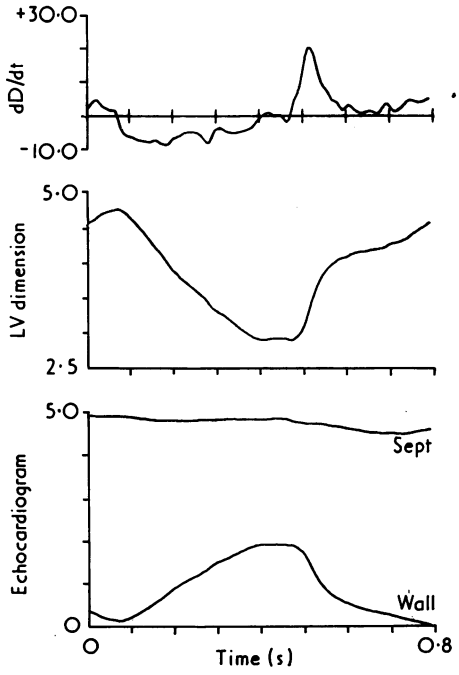

Fig. 8 *Left ventricular echocardiogram from a patient after mitral valve repair. Layout as Fig 5.*

In order to interpret these results, it was necessary to establish the relation between changes in a single ventricular dimension as measured by echocardiography and those in the cavity as a whole. This was most satisfactorily done using an angiocardiographic method in which the minor diameter was identified and compared with changes in cavity volume, estimated by the area length method (Sandler and Dodge, 1968). In this way, relatively close correlation between peak values of rate of change of dimension and those of volume was shown, a similar relation holding for normal subjects and patients with rheumatic mitral valve disease or mitral prosthesis, provided that the end diastolic dimension was less than 6 cm. This relation results from exclusion of patients in whom cavity size is very large, an unusual finding after mitral valve replacement, and reflecting the fact that changes in ventricular volume are mediated predominantly by alteration in minor axis (Lewis and Sandler, 1971). Comparison of the time relations between dimension and volume indicates that both the onset of increase and the time to 20 per cent peak value are the same in all patients studied, so that this method of assessing the filling pattern is independent of cavity size. Peak rate of increase of dimension was simultaneous with that of volume in patients with mitral stenosis or a mitral prosthesis, but preceded it by 20 ms in normal subjects, these results extending those previously reported (Prewitt *et al.*, 1975). There are, however, significant differences between echocardiographic and angiocardiographic measurements of left ventricular dimension. In particular, minimum angiographic dimension corresponds with end-ejection, and minimum echocardiographic dimension with the onset of mitral valve opening approximately 90 ms later. The exact basis of this discrepancy is not clear, but the difference almost exactly accounts for the apparent delay in the timing of peak rate of increase and 20 per cent peak rate of increase made by the two methods.

It has previously been shown that the diastolic pattern of left ventricular wall movement is abnormal in patients with mitral stenosis (Gibson and Brown, 1973). Not only is the peak rate of increase of dimension reduced, but there is also an increase in the interval from the onset of outward wall movement to the time when the rate of increase of dimension has fallen to 20 per cent of its peak value. The present angiographic results indicate that these changes reflect a corresponding reduction in peak left ventricular filling rate and an increase in filling time, both of which are characteristic of obstruction to the mitral valve orifice. This conclusion is strengthened by the increase in peak rate of increase of dimension and reduction in filling time occurring after successful mitral valvotomy.

In the present study, the lowest peak rates of increase of dimension were recorded after Starr-Edwards mitral replacement when filling time was prolonged to almost 300 ms, with a range of values overlapping that in patients with moderately severe mitral stenosis. In those patients with Björk-Shiley prostheses, peak rates of increase of dimension fell just on the lower limit of normal, and the filling time was within normal limits, this may result from the fact that the prosthesis has no central occluder and therefore a more laminar flow pattern. In patients with Hancock xenografts, peak rates of increase of dimension were virtually identical to those in patients with Björk-Shiley prostheses, though filling period was significantly longer. The only group of patients in whom both peak rate of wall movement and filling time were normal were those who had undergone mitral valve repair. The post-operative values of these two measurements in this last group were unrelated to whether the original mitral lesion was stenotic or regurgitant, or the underlying process rheumatic or degenerative, and in all the patients the peak rate of mid-diastolic mitral valve closure was strikingly reduced from normal values bearing no relation to peak rates of wall movement or filling time. This result is consistent with previous studies in patients with rheumatic mitral valve disease, showing that measurements of mitral diastolic closure rate are of little value in assessing the pattern of left ventricular filling (Gustafson, 1967; Winters *et al.*, 1969; Feigenbaum, 1972).

The present results are also consistent with haemodynamic assessments of mitral prostheses previously published. In general, mitral diastolic gradients at rest in patients with the models of Starr-Edwards prosthesis studied have been in the range 5 to 7 mmHg (Mason *et al.*, 1967; Morrow *et al.*, 1967; Glancy *et al.*, 1969; Kloster *et al.*, 1969; Russell *et al.*, 1972; Winter *et al.*, 1972; Rostad *et al.*, 1976). The present series did not contain any patients with size 2M prostheses, or model 6300, in whom mitral gradients are significantly higher (Glancy *et al.*, 1969; Kloster *et al.*, 1969; Reis *et al.*, 1970; Winter *et al.*, 1972). Diastolic gradients of 4 to 5 mmHg have been reported in patients with the mitral Björk-Shiley prosthesis 4 to 5 mmHg (Lepley *et al.*, 1973; Book, 1974; Rostad *et al.* 1976), and similar gradients have been observed in patients with Hancock valves (Horowitz *et al.*, 1974; Johnson *et al.*, 1975; McIntosh *et al.*, 1975). Though it is difficult to compare results in many different series, it appears

that these haemodynamic studies are not incompatible with our findings.

Although differences in peak rates of wall movement and filling periods were clear cut, a number of factors must be considered before direct comparisons are made, either between any one type of prosthesis and rheumatic mitral valve disease, or between one prosthesis and another. Septal movement was reversed in all patients with a normally functioning Starr-Edwards prosthesis, and to a lesser extent, in those with Björk-Shiley prostheses. Though a previous angiographic study (Gibson and Brown, 1975) has shown that satisfactory estimates of peak rates of wall movement can be made in these circumstances, the presence of reversed septal movement strongly suggests that the pattern of left ventricular filling was abnormal. It is possible, therefore, that the relation between changes in dimension and in volume was altered in these patients in a manner which our studies did not define. Comparison with rheumatic valve disease on the basis of peak rates of wall movement alone may also be unsatisfactory, since this assumes that obstruction at the mitral valve orifice alone is responsible for the haemodynamic disturbance in rheumatic mitral stenosis. Recent studies have identified a group of patients in whom mitral gradients are low or absent and left ventricular end-diastolic pressure high in spite of severe symptoms, but in whom symptoms were relieved by mitral surgery, suggesting additional impairment of left ventricular filling by the subvalve apparatus (unpublished). It appears, therefore, that though these abnormalities of wall movement are related to the presence of a rigid mitral valve ring, an obstructed mitral orifice, and an abnormal flow pathway into the left ventricle, this relation is likely to be complex and indirect. At present, we prefer to regard them as consistent patterns rather than to link them directly to mitral diastolic gradients or flows.

Nevertheless, the range of peak rates of wall movement and filling times for the six groups of patients studied were so characteristic that departures from them could readily be recognised. This has obvious clinical value. In 14 patients with Starr-Edwards prostheses and silent para-prosthetic regurgitation, the peak rate of increase of left ventricular dimension was considerably increased, along with the reappearance of normal septal movement (Miller *et al.*, 1973; Burggraf and Craige, 1975). Two patients developed obstructed Bjørk-Shiley prostheses, resulting in a major reduction in peak rate of wall movement and prolongation of filling period, both of which returned to normal after successful reoperation. Two patients with mitral valve repair had low rates of wall movement postoperatively as a result of significant left ventricular inflow tract obstruction, and two had abnormally high peak filling rates suggesting that the repairs had been inadequate and had resulted in mitral reflux. These findings were also confirmed at a second operation.

The technique we have described thus seems to offer a simple, non-invasive means of assessing mitral valve function by examining the characteristics of left ventricular wall movement during filling. It can be used in the immediate postoperative period when cardiac catheterisation would be impracticable and hazardous. It makes possible early detection of valve malfunction, can be used to characterise available mechanical and biological prostheses, and gives information about left ventricular systolic function, particularly if combined with measurements of left ventricular pressure or apex cardiogram (Venco *et al.*, 1977). It may prove to be useful after conservative operations on the mitral valve when the haemodynamic characteristics of the result cannot be exactly predicted, so that a baseline for the assessment of progress can be established postoperatively in individual patients. Preliminary results suggest that it can provide the basis of physiological follow-up of the increasingly large population of patients who have undergone successful mitral valve surgery.

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