Left ventricular function in rheumatic mitral stenosis

Clinical echocardiographic study

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SUMMARY Echocardiography was used to examine the extent and significance of impairment in left ventricular function in 20 patients with rheumatic mitral stenosis. Indices of left ventricular performance—normalised mean rate of circumferential fibre shortening (Vcf), ejection fraction, normalised posterior wall velocity, and stroke volume were reduced. The impairment in left ventricular function was related to the degree of functional disability (NYHA), right ventricular dilatation, and left atrial enlargement. Vcf was inversely related to both the internal right ventricular diameter ($r = -0.767$, $P < 0.001$) and the degree of left atrial enlargement ($r = -0.554$; $P < 0.05$). The normalised velocity of the interventricular septum and the maximum systolic and diastolic endocardial velocities were also reduced.

These results suggest that abnormalities in contractility of left ventricular myocardium are responsible for the impaired myocardial function in patients with mitral stenosis and that such impairment is clinically significant.

Rheumatic mitral stenosis is one of the commonest valvular heart lesions in Egypt and other developing countries. The only effective treatment is surgical. However, the results of operation, morbidity, and mortality are governed not only by the mechanical defect but also by the state of the myocardium. The presence of such a myocardial factor in rheumatic mitral stenosis (Harvey *et al.*, 1955; Fleming and Wood, 1959) has remained speculative until recently (Feigenbaum *et al.*, 1966; Curry *et al.*, 1970; Heller and Carleton, 1970; Holzer *et al.*, 1973). Since rheumatic heart affection is more severe and the degree of valvular damage is greater in developing countries than in industrialised Western communities (El-Sherif, 1975), it seems appropriate to examine the incidence and extent of left ventricular dysfunction in Egyptian patients. Left ventricular dimensions and function were therefore investigated in a group of patients with rheumatic mitral stenosis who were referred for mitral commissurotomy. In addition, the clinical correlates and the changes in right ventricular and left atrial dimensions were examined. Echocardiography had been used in this study for measurements of cardiac chamber dimensions and for the assessment of left ventricular performance. Good correlation between angiographic and echocardiographic measurements

of left ventricular dimensions has already been shown (Pombo *et al.*, 1971; Feigenbaum *et al.*, 1972).

Methods

Twenty patients with isolated rheumatic mitral stenosis were investigated. All patients were evaluated clinically and mitral commissurotomy was found necessary. Symptoms, that is degree of dyspnoea and effort intolerance, were classified according to New York Heart Association (NYHA). Patients with hypertension (systolic blood pressure > 170 mmHg or diastolic > 100 mmHg), ischaemic heart disease, or other possible causes for left ventricular enlargement or impairment were excluded. The severity of mitral stenosis was determined from history, physical examination, chest radiograph to assess the size of the left atrium and pulmonary venous congestion, and the electrocardiogram to detect right ventricular hypertrophy and left atrial enlargement. The E-F slope of the mitral valve in the echocardiogram was also determined to assess the severity of mitral stenosis.

ECHOCARDIOGRAPHY

Recording technique

The echocardiograms were recorded with a Unirad ultrasonoscope model 849 Sono III-GZD using a 2.25 MHz transducer with an active diameter of

Left ventricular function in rheumatic mitral stenosis

13 mm and internal focus 4 to 7 cm, with a repetition rate of 10.0 impulses/s. The ultrasonoscope was coupled to a strip chart recorder. Recordings were obtained at a paper speed of 50 mm/s and when necessary 100 mm/s. Each patient had an electrocardiogram recorded simultaneously with the echocardiogram. All patients were examined in the supine position. The transducer was placed on the third, fourth, or fifth intercostal space, just to the left of the sternum, and the ultrasonic beam was directed posteriorly and slightly medially until the echogram of the anterior leaflet of the mitral valve was identified. The transducer was then rotated inferolaterally until the echoes from the endocardial surface of the interventricular septum and the posterior left ventricular wall were clearly visualised. Measurements of left ventricular dimensions were made at an angle in which fragmented echoes of the mitral valve together with the interventricular septal and posterior wall endocardial echoes could be recorded (Fig. 1). Particular care was taken to standardise the pathway of the ultrasonic beam through the left ventricle, to identify correctly the internal and external surfaces of the left ventricular walls. The transducer was then directed in a posterior cephalad and medial manner to record the aorta and left atrium. The transducer was then rocked inferolaterally to the point at which the aortic valve cusps could be visualised. Echocardiographic measurements were made only on recordings of good quality.

Measurements and calculations

The left ventricular echocardiographic dimensions were measured from the endocardial echo of the posterior wall to the endocardial echo of the left side of the interventricular septum. Measurements at end-diastole (EDD) were made at the peak of the R wave of the QRS, and measurements at end-systole (ESD) were made at the point of the smallest distance separating the septum from the posterior wall (Fig. 1). The left ventricular ejection time (LVET) was measured from the aortic valve echogram or from a simultaneously recorded carotid pulse. Septal (IVST), posterior left ventricular thickness (LVT), and right ventricular internal diameter (RVD) were measured at the peak of the R of the QRS (Fig. 1). Posterior left ventricular wall excursion (PWE) was measured from the most posterior point of the left ventricular endocardial tracing to the most anterior point in systole (Fig. 1). Similar points at the beginning and peak of the septal systolic excursion were selected to measure septal excursion (IVSE). The maximal systolic endocardial velocity (MSEV) was measured from the slope of the tangent to the steepest part of the

Fig. 1 Echocardiographic measurements. The transducer was directed towards the cavity of the left ventricle immediately below the mitral valve leaflets. Fragmentary echoes of the mitral valve (MV) are seen. EDD, end-diastolic diameter of left ventricle; ESD, end-systolic diameter of left ventricle; IVS, interventricular septum; LVPW, left ventricular posterior wall. The slope of the tangent to the steepest part of the endocardium of the left ventricular posterior wall was measured in systole (A) and diastole (B) to determine the maximal systolic endocardial velocity and the maximal diastolic endocardial velocity. (C) Interventricular septal excursion. (D) Posterior wall excursion. RVID, right ventricular internal diameter; En, endocardium; Ep, epicardium; CT, chordae tendineae; RV, right ventricle.

endocardium of the left ventricular posterior wall in systole as previously described (Kovick *et al.*, 1975) (Fig. 1). The maximal diastolic endocardial velocity (MDEV) was measured similarly in diastole.

Other measurements made from the echocardiogram were the left atrial (LAD) and aortic root (AoRD) dimensions at end-systole (Brown *et al.*, 1974) (Fig. 2). Mitral valve diastolic excursion was measured as the vertical line from the lowest point of the mitral echogram (C) to the E point. The mitral diastolic closure slope (E-F) was measured.

Fig. 2 Echocardiographic measurements. The transducer was directed towards the aortic root (AoR). A, aortic root diameter; B, left atrial diameter; LA, left atrial cavity.

Left ventricular volumes were derived by cubing EDD and ESD. The difference between end-diastolic volume (EDV) and end-systolic volume (ESV) was the left ventricular stroke volume (SV). These volumes were also used to calculate the ejection fraction (EF):

$$EF = \frac{EDV - ESV}{EDV}$$

The normalised mean rate of circumferential fibre shortening (Vcf) was calculated as:

$$Vcf \text{ (circ/s)} = \frac{EDD - ESD}{LVET \times EDD}$$

The normalised mean posterior wall velocity (Vpw) was calculated as:

$$Vpw \text{ (s}^{-1}\text{)} = \frac{PWE}{LVET \times EDD}$$

Similarly a normalised mean interventricular septal velocity (Vivs) was calculated as:

$$Vivs \text{ (s}^{-1}\text{)} = \frac{IVSE}{LVET \times EDD}$$

The ratio of left atrial to aortic root diameters

(LA/Ao) was calculated for improved detection of left atrial enlargement (Brown *et al.*, 1974).

All the calculations were made using the average obtained from five cardiac cycles, excluding four patients who were in atrial fibrillation. In these patients an average of 10 cycles was used.

Normal values for these echocardiographic measurements were those obtained from a group of 14 closely matched healthy subjects with no clinical, radiological, or electrocardiographic evidence of cardiovascular abnormality.

Results

CLINICAL CHARACTERISTICS

Eleven male subjects and nine female subjects were studied, their ages ranged from 16 to 54 years, with a mean age of 31 years. Five patients were in class II (HYHA), 11 were in class III, and four were in class IV. The electrocardiogram was normal in only three patients. Four patients had atrial fibrillation, seven had left atrial enlargement, and eight patients had electrocardiographic evidence of right ventricular hypertrophy. None of the patients had clinical or laboratory evidence of rheumatic activity. In all patients the degree of mitral stenosis was judged to be severe enough to warrant mitral commissurotomy.

ECHOCARDIOGRAPHIC FINDINGS

Mitral valve echogram

All patients showed the classic findings of mitral stenosis (Feigenbaum, 1976). The E-F slope was flat in six patients, 10 mm/s or less in eight, and between 11 and 18 mm/s in six. The mitral valve excursion (C-E) ranged from 13 to 30 mm. It was less than 20 mm in nine patients and the mean value for the whole group was 22 mm.

Measurements

Table 1 shows the measured internal diameters of the right and the left ventricles, aortic root, and left atrium. The thickness and excursion of the septum and left ventricular posterior wall are shown in Table 2. Though the right ventricular internal diameter was greater and end-diastole was less in patients with mitral stenosis than in normal subjects, the changes were not statistically significant. However, the thickness of the left ventricular posterior wall was significantly reduced ($P < 0.05$). In addition, the increase in left atrial dimension and decrease in aortic root dimension were significant ($P < 0.001$). The extent of excursion of the posterior wall and interventricular septum was not very different from that in normal subjects.

Table 1 Echocardiographic measurements

Case no.	RVD (cm)	EDD (cm)	AoRD (cm)	LAD (cm)
1	1.3	4.8	2.9	3.8
2	0.9	4.4	2.6	4.8
3	1.0	4.4	2.6	4.8
4	1.7	4.9	3.3	5.0
5	1.6	5.0	2.5	3.8
6	3.1	4.5	1.9	4.5
7	1.1	4.4	2.9	4.4
8	—	—	1.9	3.8
9	—	—	2.5	4.1
10	2.5	3.9	2.3	3.6
11	2.1	4.1	—	3.8
12	—	5.0	3.1	3.1
13	1.9	4.4	1.9	4.4
14	4.0	3.8	1.9	3.8
15	2.0	—	2.5	4.4
16	2.8	4.4	2.5	—
17	1.9	4.0	1.9	5.0
18	—	4.5	2.4	4.8
19	1.9	4.4	2.1	4.4
20	2.1	5.0	2.4	4.8
Mean	2.0	5.4	2.4	4.3
SE	0.19	0.09	0.098	0.125
Normals (mean)	1.5	4.7	3.27	3.23
SE	0.12	0.102	0.064	0.062
P <	0.1	NS	0.001	0.001

RVD, right ventricular internal diameter; EDD, end-diastolic diameter of left ventricle; AoRD, aortic root diameter; LAD, left atrial diameter.

Table 2 Echocardiographic measurements

Case no.	IVST (cm)	IVSE (cm)	LVT (cm)	PWE (cm)
1	0.8	—	0.6	0.8
2	0.8	0.6	0.6	1.1
3	1.0	0.5	0.6	0.9
4	0.7	—	0.6	1.1
5	1.1	0.6	0.5	0.9
6	0.6	0.4	0.6	0.8
7	1.0	0.8	0.6	1.2
8	1.3	0.6	0.5	0.8
9	—	—	0.5	0.9
10	0.6	0.5	0.6	0.8
11	1.3	0.5	0.8	0.9
12	—	0.4	0.5	0.6
13	1.3	0.6	0.9	0.9
14	0.9	0.4	0.8	0.7
15	—	—	0.5	1.1
16	0.8	—	1.0	0.6
17	1.0	0.6	0.8	1.2
18	0.9	1.0	0.9	0.8
19	0.6	0.4	0.8	0.8
20	1.1	0.8	0.7	0.6
Mean	0.9	0.6	0.7	0.9
SE	0.058	0.045	0.036	0.042
Normals (mean)	0.8	0.6	0.8	1.2
SE	0.040	0.10	0.040	0.10
P <	NS	NS	0.05	0.1

IVST, interventricular septal thickness; IVSE, interventricular septal excursion; LVT, left ventricular posterior wall thickness; PWE, posterior wall excursion.

Indices of left ventricular performance

The cardiac output, stroke volume, ejection fraction, and normalised mean rate of circumferential fibre shortening (Vcf) were all reduced in patients with mitral stenosis, and the changes were significant (Table 3). Vcf was reduced by more than two standard errors (less than 1.10 circ/s) in 14 out of 17 patients, while the ejection fraction was reduced to less than 0.55 in about 50 per cent of patients. The mean values for the normalised velocities of the posterior wall (Vpw), interventricular septum (Vivs), and the maximum systolic and diastolic endocardial velocities were significantly lower in patients than in normal subjects (Table 4).

The changes in myocardial contractility expressed as Vcf were inversely related to both the internal right ventricular diameter ($r = -0.767$, $P < 0.001$) (Fig. 3) and the degree of left atrial enlargement expressed as LA/Ao ratio ($r = -0.554$, $P < 0.05$) (Fig. 4). The latter relation did not include case 12. A positive correlation was present between Vcf and the extent of posterior wall excursion ($r = 0.635$, $P < 0.01$) (Fig. 5) and the ejection fraction ($r = 0.847$, $P < 0.001$).

Clinical echocardiographic correlates

Fig. 6 and 7 show the extent of impairment of left

Table 3 Indices of left ventricular function

Case no.	SV (ml)	CO (l/min)	EF	Vcf (circ/s)
1	54	5.18	0.51	0.99
2	57	5.70	0.68	1.40
3	53	4.24	0.63	1.06
4	83	5.83	0.71	1.40
5	67	5.09	0.54	1.00
6	33	2.71	0.36	0.52
7	53	3.71	0.63	1.22
8	—	—	—	—
9	—	—	—	—
10	37	2.15	0.63	0.77
11	27	2.38	0.39	0.50
12	56	4.48	0.45	0.58
13	50	3.10	0.60	0.93
14	22	2.33	0.42	0.61
15	—	—	—	—
16	36	2.88	0.43	0.83
17	37	4.07	0.58	0.83
18	60	3.48	0.66	0.98
19	47	3.76	0.56	0.83
20	61	3.90	0.49	0.72
Mean	49	3.82	0.55	0.89
SE	3.76	0.197	0.030	0.067
Normals (mean)	72	5.02	0.74	1.26
SE	6.2	0.23	0.013	0.08
P <	0.001	0.001	0.001	0.001

SV, stroke volume; CO, cardiac output; EF, ejection fraction; Vcf, normalised mean rate of circumferential fibre shortening.

Table 4 Indices of myocardial contractility

Case no.	V _{pw} (s ⁻¹)	V _{ivs} (s ⁻¹)	MSEV (cm/s)	MDEV (cm/s)
1	1.27	—	2.5	3.2
2	1.09	0.60	2.5	5.0
3	0.72	0.40	4.6	6.9
4	0.95	—	—	—
5	0.81	0.54	—	—
6	0.71	0.35	5.0	4.1
7	1.11	0.74	6.1	3.2
8	—	—	1.8	5.3
9	—	—	2.8	6.9
10	0.56	0.35	3.1	0.3
11	0.73	0.41	5.3	6.0
12	0.39	0.26	—	—
13	0.71	0.51	2.9	2.2
14	0.62	0.35	4.0	3.7
15	—	—	5.0	6.9
16	0.63	—	4.2	2.9
17	1.00	0.50	5.2	6.0
18	0.56	0.71	4.3	10.1
19	0.65	0.33	4.0	7.6
20	0.43	0.58	—	—
Mean	0.76	0.47	4.0	5.6
SE	0.056	0.039	0.308	0.579
Normals (mean)	0.95	0.61	6.2	18.0
SE	0.02	0.02	0.256	0.548
P <	0.005	0.005	0.001	0.001

V_{pw}, normalised velocity of the posterior wall; V_{ivs}, normalised velocity of the interventricular septum; MSEV, maximum systolic endocardial velocity; MDEV, maximum diastolic endocardial velocity.

ventricular function expressed as changes in V_{cf}, EF, and V_{pw} in relation to the clinical severity of mitral stenosis (NYHA classification). Deterioration in left ventricular performance was greater in patients of class IV than in those of class II. Increase in right ventricular internal diameter progressed from patients in class II to patients in class IV (Fig. 7). Patients in class IV had the largest right ventricles.

Discussion

The high incidence of impairment in left ventricular function in our patients with mitral stenosis was at variance with previous reported incidences in other studies. Harvey *et al.* (1955) found an incidence of 13 per cent in their patients, while Kennedy *et al.* (1970) found that 37 per cent of patients with mitral stenosis had evidence of impaired myocardial contraction. In a recent echocardiographic study, McDonald (1976) found an incidence of 30 per cent. The very high incidence in our patients was not completely unexpected. Two factors might have been responsible; first, patients in our study had mitral stenosis necessitating cardiac surgery. In such a selected group of patients the degree of mitral stenosis and functional disability would be greater than in a random selection of patients present in other studies. Secondly, the severe and

Fig. 3 Relation between V_{cf} and right ventricular internal diameter. $r = -0.767$; $P < 0.001$.

Fig. 4 Relation between V_{cf} and the degree of left atrial enlargement expressed as LA/Ao ratio. $r = -0.554$; $P < 0.05$. Case 12 is not included in the correlation.

Fig. 5 Relation between V_{cf} and the extent of posterior wall excursion. $r = 0.635$; $P < 0.01$.

Fig. 6 Clinical-echocardiographic correlates. Clinical severity of mitral stenosis (NYHA classification) and changes in Vcf and Vpw. The hatched area represents the mean values for the normals ± 2 standard errors.

Fig. 7 Clinical-echocardiographic correlates. Clinical severity of mitral stenosis (NYHA classification) and changes in ejection fraction (EF) and right ventricular internal diameter (RVD). The hatched area represents the normals ± 2 standard errors.

florid nature of rheumatic fever, its early age of onset, and the significant haemodynamic burden in Egyptian patients is different from that in western countries (El-Sherif, 1975). Botros and Sabaa (1976) in a haemodynamic study of 32 young Egyptian patients with mitral stenosis have shown that the pulmonary arterial pressure approached systemic level in 50 per cent of their patients. This figure was 13 per cent in Wood's series (Wood, 1954). The cardiac index was reduced in all patients in the Egyptian series, ranging between 2 and 2.5 l/min per m².

Indices of left ventricular performance, viz. Vcf, ejection fraction, Vpw, and stroke volume were reduced in patients with mitral stenosis. The impairment in left ventricular function was related to the degree of functional disability (NYHA), right ventricular dilatation, and left atrial enlargement.

In a disease like mitral stenosis where the haemodynamic burden of the mechanical obstruction is on the pulmonary circulation and right ventricle

it was unusual to find a relation between left ventricular dysfunction and clinical disability. This suggests that some of the symptoms experienced by patients with mitral stenosis may be the result of impairment in left ventricular function. The inverse relation between Vcf and right ventricular internal diameter ($r = -0.767$; $P < 0.001$) (Fig. 3) and the degree of left atrial enlargement (LA/Ao ratio) ($r = -0.554$, $P < 0.05$) favours the assumption that the impairment of left ventricular contractility was also related to the severity of mitral stenosis reflected in left atrial and right ventricular dilatation. On the other hand, hypokinesis of the left ventricular wall was found by Curry *et al.* (1970) not only in patients with mitral stenosis but also in patients with right ventricular enlargement not secondary to mitral valve disease.

Subtle changes in left ventricular contraction have been reported in mitral stenosis by Heller and Carleton (1970). Of their 25 patients, 20 showed distortion, immobility, and rigidity of the postero-basal area of the left ventricle. Similarly, localised abnormalities in the wall of the left ventricle have been described by Horwitz *et al.* (1973). Fibrosis in or near the papillary muscles was postulated as a possible cause. However, the reduction in our patients of the mean values for the normalised velocities of the posterior wall (Vpw), interventricular septum (Vivs), and the maximum systolic (MSEV) and diastolic (MDEV) endocardial velocities are consistent with the view that a generalised abnormality of myocardial mechanical performance is responsible for the occurrence of left ventricular dysfunction in some patients with mitral stenosis. Holzer *et al.* (1973) found abnormalities of ventricular contraction in 10 patients with rheumatic mitral stenosis.

The present study suggests that abnormalities in contractility of the left ventricular myocardium can be responsible for the impaired myocardial function in patients with mitral stenosis and that such impairment is clinically significant. It may be a contributing factor in some patients who have an unsatisfactory clinical response to mitral commissurotomy. Histopathological studies in patients with mitral stenosis showed that the failure of improvement after mitral commissurotomy was related to the extent of myocardial involvement by the rheumatic process (Selim, 1976).

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