Diastolic movement of mitral valve in hypertrophic cardiomyopathy

*An echocardiographic study**

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SUMMARY Diastolic movement of the mitral valve and its relation to various aspects of left ventricular anatomy and function has been investigated in 15 patients with hypertrophic cardiomyopathy. Echocardiograms showing left ventricular cavity and mitral valve simultaneously were recorded and subsequently digitised in order to obtain continuous plots of left ventricular dimension, its rate of change, and peak diastolic closure rate of the anterior leaflet of the mitral valve. Septal and posterior wall thicknesses, together with left ventricular cavity and outflow tract dimension in mid-systole, were also measured directly on the echocardiograms. All the measurements were compared with those in 14 normal subjects. Patients with hypertrophic cardiomyopathy had smaller ventricles, with a conspicuous increase in septal thickness compared with those of normal subjects. Posterior wall thickness was only slightly increased, rapid filling period was normal, whereas both the peak rate of increase of left ventricular dimension and the peak mitral diastolic closure rate were low. However, no relation between these two indices was found. Instead, mitral valve closing velocity was strongly correlated with left ventricular outflow tract dimension. This relation was further validated by the infusion of isoprenaline, resulting in a simultaneous reduction of left ventricular outflow tract dimension and peak mitral closing velocity.

In patients with hypertrophic cardiomyopathy it is concluded that anatomical changes of the left ventricular outflow tract are responsible for a disruption of the normal pattern of flow behind the anterior leaflet of the mitral valve, which results in a diminished closure rate of the valve in diastole.

Slow diastolic closure rate of the anterior leaflet of the mitral valve is a common finding in patients with hypertrophic cardiomyopathy. It has been related to a reduction in left ventricular diastolic compliance^{1 2} which usually results in diminished transmitral flow.³

However, angiographic studies in hypertrophic cardiomyopathy^{4 5} have shown that filling rates of the left ventricle can be within normal limits, whereas the filling pattern may be abnormal.⁶ Subsequently, abnormal relaxation of the left ventricle was confirmed by echocardiography.⁷

An echocardiographic study was undertaken in a group of patients with hypertrophic cardiomyopathy in an attempt to gain further information about the relation between the diastolic closure rate

of the mitral valve and left ventricular filling. In addition, left ventricular anatomical changes were investigated to assess their connection with mitral valve motion in diastole.

Subjects and methods

Fifteen patients (12 men and three women, age range 18 to 54 years, mean 36 years) with hypertrophic cardiomyopathy were studied. In all the patients the diagnosis was based on angiographic criteria.⁸ All the patients were in sinus rhythm at the time of the study; nine of them were taking beta-blocking drugs.

For comparison, 14 normal subjects of similar ages were also studied.

Echocardiograms were obtained by means of an Ekoline 20A ultrasonoscope with a 2.25 MHz non-focused transducer, and a simultaneous electro-

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cardiogram was recorded in all cases. Studies were made with the patients either supine or in a slightly left lateral position. The transducer was angled in order to obtain simultaneous recordings of the mitral valve, interventricular septum, and posterior left ventricular wall (Fig. 1). Measurements were made only when these echoes were clear and continuous throughout the cardiac cycle. In six patients, after informed consent, the tracings were repeated after an intravenous infusion of isoprenaline. The drug was injected as a single bolus of 3 to 5 μg over 15 seconds.

DIGITISING METHODS

Echocardiograms were digitised, as described by Gibson and Brown⁹ using a Summagraphics digitising table, and subsequently processed by a Prime 300 computing system. Data points were generated for both sides of the interventricular septum, endocardium, and epicardium of the posterior wall and mitral valve leaflets, together with calibration signals representing 0.5 s and 1 cm, and the RR interval of the beat to be studied.

From the digitised data, plots were made of the position of the wall and mitral valve echoes, left ventricular dimension and its rate of change, and

Fig. 1 Echocardiogram of a patient with hypertrophic cardiomyopathy, showing septum, posterior wall, and mitral valve cusps. An apex cardiogram (ACG) is superimposed. AMV, anterior mitral valve leaflet; PMV, posterior mitral valve leaflet; EN and EP, endocardial and epicardial surfaces of the posterior left ventricular wall.

Fig. 2 Computer output of the left ventricular cavity of a patient with hypertrophic cardiomyopathy, showing digitised echoes from the right (R Sep) and left (L Sep) side of the septum, endo- and epicardial (Epic) surfaces of the posterior wall, and mitral valve cusps. The left ventricular dimension curve (above), its rate of change ($D \text{ Dim} / DT$), its normalised rate of change (lengthening rate), and mitral leaflet velocity (top).

rate of change of mitral valve position. From these tracings (Fig. 2), the following measurements were made:

- (1) Left ventricular dimension, measured directly at end-diastole, along with its peak rate of increase during diastole.
- (2) Posterior wall and septal thicknesses, measured directly at end-diastole.
- (3) Peak diastolic closure rate of the anterior leaflet of the mitral valve, taken from the curve of first differential of position.
- (4) Rapid filling period, measured as the interval between the onset of forward movement of the

anterior mitral leaflet and the point at which the rate of increase of dimension had fallen to 20 per cent of its peak value⁹.

(5) Dimension of the left ventricular outflow tract in mid-systole, taken as the minimum distance between the mitral valve apparatus and the left side of the septum. If systolic anterior movement of the mitral valve was present, the most anterior echo originating from the valve was used.

The comparison with the normal group was carried out using Student's *t* test.

Results

NORMAL SUBJECTS

Mean left ventricular cavity dimension at end-diastole was 4.7 ± 0.5 cm, while septal and posterior wall thicknesses at the same time were 0.86 ± 0.11 and 0.86 ± 0.10 cm, respectively (Table). The peak rate of increase of dimension in diastole was 15.1 ± 4.4 cm/s, the duration of the rapid filling period was 148 ± 29 ms, and the peak diastolic closure rate of the anterior mitral leaflet was 240 ± 65 mm/s.

Table Results of echocardiographic measurements in 14 normal subjects and 15 patients with hypertrophic cardiomyopathy

	Normals	Hypertrophic cardiomyopathy	P
Diastolic dimension (cm)	4.7 ± 0.5	3.8 ± 0.6	<0.01
Septal thickness (cm)	0.86 ± 0.11	1.92 ± 0.39	<0.01
Post. wall thickness (cm)	0.86 ± 0.10	1.17 ± 0.17	<0.05
DDim/DT (cm/s)	15.1 ± 4.4	7.6 ± 3.2	<0.01
Rapid filling period (m/s)	148 ± 29	157 ± 42	n.s.
Peak closing rate of anterior mitral valve	240 ± 65	96 ± 51	<0.01

PATIENTS WITH HYPERTROPHIC CARDIOMYOPATHY

Mean cavity dimension at end-diastole was 3.8 ± 0.6 cm, significantly smaller than normal ($P < 0.01$). Posterior wall thickness was only slightly increased (1.17 ± 0.17 cm; $P < 0.05$), whereas septal thickness was greatly increased (1.92 ± 0.39 ; $P < 0.01$).^{*} Mean value of the rapid filling period was normal, with a wide scatter of values (157 ± 42 ms). Both peak rates of dimension increase in diastole and diastolic closure of the mitral valve were low: 7.6 ± 3.2 cm/s and 96 ± 5 mm/s, respectively ($P < 0.01$). However, no relation was found between these two indices (Fig. 3). In addition, no relation was found between the peak diastolic closure rate of the mitral valve and the rapid filling period, the diastolic dimension

^{*} Significant systolic anterior movement of the mitral valve was present in seven patients.

Fig. 3 Relation between peak diastolic closure rate (DCR) of the anterior mitral leaflet and peak rate of increase in left ventricular dimension (DDim/DT).

of the left ventricle, or septal and posterior wall thicknesses. Instead, a strong correlation ($r = 0.86$) was found between peak mid-diastolic closure velocity of the mitral valve and left ventricular outflow tract dimension in mid-systole (Fig. 4).

In the six patients in whom isoprenaline was infused, the decrease in peak diastolic closure rate of the anterior leaflet of the mitral valve corresponded with the decrease in left ventricular outflow tract dimension, providing further validation of the relation between the two (Fig. 5). Peak diastolic closure rate of the posterior leaflet of the mitral valve remained unaffected by the drug infusion (Fig. 5). Isoprenaline was also injected in five

Fig. 4 Relation between peak DCR of the anterior mitral leaflet and left ventricular outflow tract (LVOT) dimension at mid-systole.

Fig. 5 Echocardiogram of a patient with hypertrophic cardiomyopathy after the infusion of isoprenaline. The recording was taken when the effect of the drug was wearing off (from left to right), as shown by the decrease in heart rate. Together with the increase in left ventricular outflow tract (LVOT) dimension, a result of an almost complete disappearance of systolic anterior motion (SAM) of the anterior mitral cusp (AM), an increase of diastolic closing velocity of the latter is apparent. The movement of the posterior mitral cusp (PM) is unaffected by the drug.

patients without echocardiographic evidence of hypertrophic cardiomyopathy and in none of them could we reproduce these findings.

Discussion

The significance of slow diastolic closure rate of the anterior leaflet of the mitral valve in patients with hypertrophic cardiomyopathy is still controversial. Reduction in left ventricular diastolic compliance,^{1,2} diminished transmitral flow,³ and slow ventricular filling¹⁰ have been suggested as mechanisms of this reduction. However, the observation of normal movement of the posterior leaflet of the mitral valve with abnormal movement of the anterior one (Fig. 6) suggests that a local factor is interfering with mitral valve motion. This explains the asymmetrical behaviour of the valve.

In patients with hypertrophic cardiomyopathy, abnormal anatomical insertion of the anterior mitral valve leaflet¹¹ and abnormal angulation of the papillary muscles¹² could affect the diastolic movement of the valve in addition to contributing to the obstruction of the left ventricular outflow tract in systole.

In our study we have shown that a linear relation exists between left ventricular outflow tract

dimension and peak diastolic closure rate of the mitral valve, whereas the latter is not related to any other anatomical or functional aspect of the left ventricle. Furthermore, when the dimension of the left ventricular outflow tract was acutely changed, using an inotropic drug such as isoprenaline, a concomitant decrease in peak diastolic closure rate of the anterior mitral valve was obtained. This is a further validation of the relation between the two indices.

It is difficult to explain how the anatomical changes of the left ventricular outflow tract interfere with the pattern of mitral valve motion in diastole. Bellhouse,¹³ using a model mitral valve and left ventricle, has shown that when the ventricle is small a strong ring vortex is formed within the ventricle; this tends to close the valve in diastole. The vortex is asymmetrical, with its main strength concentrated behind the anterior cusp. This force which tends to close the valve can presumably be affected by the altered anatomy of the left ventricular outflow tract. If so the usual pressure would not be exerted on the anterior leaflet during mid-diastolic closure.

For this reason, in patients with hypertrophic cardiomyopathy we suggest that anatomical and functional abnormalities of both septum and mitral valve, which ultimately result in gross narrowing

Fig. 6 Echocardiogram of a patient with hypertrophic cardiomyopathy, showing left ventricular cavity and mitral valve leaflets. Slow mid-diastolic closure rate of the anterior cusp (AMV) is evident, whereas the posterior one (PMV) moves normally.

of the left ventricular outflow tract, also interfere with the normal pattern of transmitral flow during diastole. This results in disruption of the ring vortex behind the anterior mitral leaflet and consequently in reduced mid-diastolic closure of the valve.

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