Study of left ventricular wall thickness and dimension changes using echocardiography¹

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SUMMARY The relation between transverse left ventricular cavity dimension and posterior wall thickness has been studied using digitised echocardiograms from 10 normal subjects and 63 patients with heart disease. In normal subjects, the peak systolic rate of wall thickening (4.6 ± 1.2 cm/s) is less than the peak diastolic rate of wall thinning (10.7 ± 1.7 cm/s). Maximum wall thickness, minimum dimension, and mitral valve opening are synchronous, and there follows a distinct early period of rapid thinning of the wall (100 ± 20 ms) which corresponds to the rapid filling period. In ischaemic heart disease the rate and duration of rapid thinning are normal, but the onset precedes mitral valve opening (by 50 ± 30 ms). In hypertrophic cardiomyopathy the rate (7.4 ± 4.6 cm/s) and pattern of wall thinning are more variable than normal, and closely predict the pattern of change in cavity dimension. Inflow obstruction, associated with slow and protracted increase in cavity dimension, causes the thinning period to be prolonged, but the peak rate of thinning is often not reduced to the same extent as the rate of increase in dimension. This entails reversal of septal movement. We conclude that rapid wall thinning is an intrinsic property of the left ventricular myocardium, normally associated with rapid filling, which may, however, be dissociated from filling by asynchronous relaxation or inflow obstruction, or modified by myocardial disease.

Use of echocardiography combined with simple digitising techniques has made it possible to study continuous changes in left ventricular cavity dimension in man (Gibson and Brown, 1973). Though the pattern of change in a single diameter resembles the left ventricular volume curve (Henderson *et al.*, 1906), the two may be dissociated from one another, especially in ischaemic heart disease. Here, the rapid increase in transverse dimension at the start of ventricular relaxation, which normally corresponds to the rapid filling period, commonly occurs before opening of the mitral valve, indicating an isovolumic change in cavity shape and incoordinate relaxation (Upton *et al.*, 1976). A similar study has shown evidence of incoordinate relaxation in patients with hypertrophic cardiomyopathy (Sanderson *et al.*, 1978).

Since the position of the endocardium may depend not only on movement of the whole ventricular wall but also on changes in its thickness, we

have studied continuous changes of wall thickness with relation to cavity dimension and mitral valve movement. An angiographic study (Gibson *et al.*, 1977) comparing patients with normal left ventricular function and those with ischaemic heart disease suggests that epicardial movement is of relatively minor importance in determining either the rapid filling period or rapid endocardial movement during isovolumic shape changes. The present study takes advantage of the superior resolution of time and position attainable with echocardiography in order to study the peak rate and timing of wall thickness changes in a single region, particularly in relation to normal and abnormal relaxation.

Methods

(1) PATIENTS

The following 5 groups of patients were studied:

- (a) *Normal*: There were 10 normal subjects, 4 of them women, whose ages ranged from 17 to 42 (mean 29) years.
- (b) *Ischaemic heart disease*: 20 patients were studied with angina caused by coronary artery disease confirmed at catheterisation.

¹The computing equipment used in this study was provided by the D.H.S.S. as part of their experimental programme.

All had regional abnormalities of wall movement evident on left ventriculography. Three had aneurysms which were confirmed at surgery. Two were women and all were in sinus rhythm.

- (c) *Hypertrophic cardiomyopathy*: 17 patients were studied with hypertrophic cardiomyopathy. In 10 the diagnosis was confirmed by angiography, and in the remainder echocardiography showed asymmetrical hypertrophy of the septum and systolic anterior movement of the mitral valve. Five patients were women and 1 was in atrial fibrillation.
- (d) *Mitral stenosis*: 19 patients were studied of whom 12 were women; 16 were in atrial fibrillation and the others were in sinus rhythm.
- (e) *Starr-Edwards mitral valve replacement*: 7 patients were studied with Starr-Edwards mitral valve prostheses. Their operations had been between 1 month and 3 years before study. All were in atrial fibrillation and 3 were women.

(2) ECHOCARDIOGRAPHY

The patients were studied semisupine and partly rotated to the left, using a 2.25 MHz transducer and a Smith-Kline 20 Echocardiograph, modified by addition of a 'switched-gain' system with which, by selectively attenuating every 5th pulse, echoes from strongly reflecting surfaces such as the posterior epicardium may be appreciated without loss of sensitivity at other levels. Tracings were made photographically with a Cambridge Instruments strip chart recorder, at a paper speed of 100 mm/s, showing continuous echoes from the epicardium, posterior wall endocardium, left side of the interventricular septum, and anterior mitral valve cusp (Fig. 1). Superimposed on the records were the electrocardiogram, a phonocardiogram adjusted to show the aortic component of the second heart sound, and an apex cardiogram, the last using a Cambridge transducer with time constant of 4 s and lower frequency limit of 0.04 Hz.

(3) ANALYSIS OF RESULTS

The records were digitised as previously described (Gibson and Brown, 1973) using a Summagraphics digitising table and Prime 300 computer system. From the strings of co-ordinates generated by the digitiser were obtained continuous plots over one cardiac cycle showing the transverse cavity dimension, the posterior wall thickness, their respective rates of change, and the velocity of the anterior mitral valve cusp.

Fig. 1 Echocardiogram showing continuous echoes from either side of the interventricular septum, the mitral valve, and the endocardial (endo) and epicardial (epi) surfaces of the posterior left ventricular wall. The abscissae are 40 ms and the superimposed depth markers are 1 cm apart. Recorded simultaneously are the apex cardiogram (ACG), electrocardiogram (ECG), and phonocardiogram (Phono). Isovolumic contraction (ivc) is taken as the period between the onset of inscription of the QRS complex and the 'E' point of the apex cardiogram, and isovolumic relaxation (ivr) from the aortic closure sound (A_2) to the onset of mitral valve opening (MVO).

The following events were identified in order to delimit the periods of isovolumic contraction and relaxation (Fig. 1): (a) the onset of inscription of the 'q' wave; (b) the E point of the apex cardiogram, which corresponds to the onset of aortic valve opening; (c) the aortic valve closure sound (A_2); and (d) the onset of forward movement of the anterior cusp of the mitral valve.

During diastole, the pattern of change in wall thickness usually included a discrete early phase which may be described as a 'rapid thinning period' (Fig. 2). Its end point was arbitrarily defined as the time at which the rate of wall thinning had fallen to 20 per cent of its peak value, for this satisfactorily identified the point of discontinuity on the plot of its rate of change. An analogous point was defined on the record of the first derivative of cavity dimension.

Thus, from the computer plots could be measured the values of wall thickness and its peak rates of change, and the time intervals listed in Table 1.

Fig. 2 Normal subject. Set of computer plots from the digitised echocardiogram reproduced in the lowest panel. On the ordinates are plotted cavity dimension; its rate of change; posterior wall thickness; its rate of change. The two vertical lines correspond to the times of aortic valve closure and mitral valve opening. The two open triangles at the top of the figure define the period of rapid posterior wall thinning.

Results

(1) NORMAL SUBJECTS (Fig. 2)

The normal end-diastolic wall thickness was 0.8 ± 0.2 cm (mean $\pm$ 1 standard deviation). During isovolumic contraction (before the E point of the apex cardiogram) there was slight decrease in thickness of between 0 and 2 mm. Wall thickness then increased by 30 to 100 per cent (mean 60 per cent) to the end-systolic value 1.3 ± 0.2 cm. During isovolumic relaxation, between aortic closure and mitral valve opening, the wall thickened further, by a mean of 1.5 mm, to a maximum thickness of 1.4 ± 0.2 cm.

The peak rate of thickening was 4.6 ± 1.2 cm/s, significantly less than that of reduction in wall thickness during diastole—'thinning'—which was

10.7 ± 1.7 cm/s ($P < 0.001$). There were close and consistent time relations between the changes in wall thickness, cavity dimension, and mitral valve movement. Maximum wall thickness was within 10 ms of minimum cavity dimension in all subjects and occurred close to the time of onset of mitral valve opening (mean separation 2 ± 20 ms). The peak rate of thinning was synchronous with the peak rate of increase in cavity dimension in all but one subject in whom they were separated by 40 ms. The rapid thinning period was 100 ± 20 ms, not significantly different from the period of rapid increase in cavity dimension (115 ± 20 ms). The rapid thinning period accounted for a mean of 90 per cent of the total change in wall thickness, which in several subjects remained constant throughout the subsequent period of diastasis.

(2) ISCHAEMIC HEART DISEASE (Fig. 3)

Three patients had values of maximum wall thickness 1.0 cm or less and in these the peak rate of thickening was also reduced to 3 cm/s or less. In

Fig. 3 Ischaemic heart disease. Set of plots laid out as in Figure 2. Note that change in dimension and wall thickness precede mitral valve opening.

Table 1 Results

	Wall thickness			Rate of change of wall thickness		Peak diastolic rate of change of dimension	Intervals		Peak thinning to peak rate of change of dimension	Duration of rapid inc. in dimension	
	End-diast.	End-syst.	Max	Peak syst.	Peak diast.		Max. thickness to min MVO dimension			rapid thinning	rapid inc. in dimension
	(cm)	(cm)	(cm)	(cm/s)	(cm/s)	(cm/s)	(ms)	(ms)	(ms)	(ms)	(ms)
Normal subjects (10)	0.8 ±0.2	1.3 ±0.2	1.4 ±0.2	4.6 ±1.2	10.7 ±1.7	16 ±3	0 ±15	2 ±20	4 ±20	100 ±20	115 ±20
Ischaemic heart disease (20)	0.9 ±0.2	1.5 ±0.4	1.5 ±0.4	4.8 ±1.5	8.4 ±2.6	12 ±4	10 ±40	50* ±30	5 ±30	120 ±40	140 ±35
Hypertrophic cardiomyopathy (17)	1.3* ±0.5	2.2* ±0.4	2.2* ±0.4	6.2 ±3.0	7.4† ±4.6	14† ±7	20 ±45	50* ±50	5 ±15	200 ±75	240 ±70
Mitral stenosis (19)	0.8 ±0.3	1.4 ±0.2	1.5 ±0.3	4.6 ±1.7	5.3* ±1.1	7* ±2	-40 ±30	-30 ±50	-5 ±40	150* -400	250* -500
Mitral Starr valves (7)	1.1 ±0.2		1.7 ±0.2	4.4 ±1.4	6.8* ±1.8	8* ±2	-40 ±50		20 ±30	120* -400	200* -550

The symbol ± refers to the standard deviations of normal distributions. Values clearly not so distributed are given as ranges.

*Indicates significant differences from normal subjects (Student's t test or Mann Whitney U test).

†Indicates abnormal scatter.

Fig. 4 Hypertrophic cardiomyopathy. Set of plots laid out as in previous figures from a patient with particularly slow change in both wall thickness and dimension.

the other 17 patients, end-diastolic and maximum wall thickness were normal as were the values for the peak rates of change.

The mean interval between minimum cavity dimension and maximum wall thickness was 10 ± 40 ms, again not different from normal, and the time of peak thinning was similarly synchronous with the time of peak rate of increase in cavity dimension. However, mitral valve opening was delayed by up to 100 ms (mean 50 ms) with respect to minimum dimension and maximum wall thickness, so that significant thinning and dimension increase occurred during a period of constant cavity volume.

(3) HYPERTROPHIC CARDIOMYOPATHY (Fig. 4) Both end-diastolic and maximum wall thickness were increased relative to normal in this group being 1.3 ± 0.5 cm and 2.2 ± 0.4 cm, respectively. Peak rate of thickening during systole was 6.2 ± 3 cm and peak thinning rate was 7.4 ± 4.6 cm/s. The means are not significantly different from normal, but in the case of diastolic thinning, the scatter is great, with 2 patients falling above the normal range, thinning abnormally fast, and 10 falling below. There was a significant correlation in this group between the peak rate of wall thinning and the peak rate of increase in cavity dimension ($r = 0.90$, $P < 0.001$) the regression equation being:

$$\frac{dW}{dt} \text{ max} = 0.56 \frac{dD}{dt} \text{ max} - 0.5 \text{ cm/s.}$$

(Fig. 5)

Fig. 5 Relation between peak diastolic rate of wall thinning and peak rate of increase in cavity dimension in normal subjects, hypertrophic cardiomyopathy, and left ventricular inflow obstruction.

In those patients in whom it could be assessed, the mean interval between maximum thickness and minimum cavity dimension was 20 ms, but the scatter was wide. More consistent was the delay (50 ± 50 ms) similar in duration to that of ischaemic heart disease, between peak wall thickness and the onset of mitral valve opening.

(4) MITRAL STENOSIS (Fig. 6)

In 2 patients with mitral stenosis the posterior wall thickness was reduced. In the others the systolic and diastolic wall thickness were normal, as was the peak rate of systolic thickening. The pattern of endocardial movement during filling was typical of patients with mitral stenosis for the peak rate of increase in dimension was reduced to 7 ± 2 cm/s and the rate of dimension increase fell to 20 per cent of the peak only after 250 to 500 ms. The pattern of diastolic change in wall thickness was similar in that the mean peak rate of thinning was reduced to 5.3 ± 1.1 cm/s and the time to 20 per cent of peak thinning was prolonged to 150 to 400 ms. In contrast to hypertrophic cardiomyopathy there was, however, no clear correlation between the peak rate of change of cavity dimension and the peak rate of wall thinning, the tendency being for peak thinning rate to exceed that expected from the reduction in rate of dimension increase (Fig. 5).

(5) STARR-EDWARDS PROSTHESIS

End-diastolic and maximum wall thickness were normal in this group. In 1 patient the peak rate of systolic thickening was reduced (to 2 cm/s) but in the others it was normal. The pattern of wall movement during thinning was similar to that of mitral stenosis with prolongation of the rapid phase and reduction in the peak rate of increase in dimension. This was reflected by the pattern of change in wall thickness, for the period of thinning was greatly prolonged (120 to 400 ms) and the peak thinning

rate was usually reduced (mean = 6.8 ± 1.8 cm/s). Again, however, the peak rate of thinning was often greater than would be predicted from the rate of change of cavity dimension, and in 2 patients, though peak rate of increase in dimension was reduced, the peak thinning rate was within the normal range (Fig. 5).

Discussion

Echocardiography may be used to make unambiguous continuous recordings of the position of the left ventricular endocardium and posterior wall

Fig. 6 Mitral stenosis. Plots similar to those in previous figures, showing slow and protracted diastolic change in cavity dimension and wall thickness.

Table 2 Values of wall thickness in normal subjects

Author	Method	Wall thickness (cm)	
		End-diastolic	End-systolic
Feigenbaum <i>et al.</i> (1968)	Echo	0.8-1.3	
McDonald <i>et al.</i> (1972)	Echo	0.9 ± 0.14	1.48 ± 0.3
Corya <i>et al.</i> (1977)	Echo	0.7-1.2	
Eber <i>et al.</i> (1969)	Angio	0.82-1.0	1.43-2.0
Dumesnil <i>et al.</i> (1974)	Angio	1.16 ± 0.43	
Dodge <i>et al.</i> (1974)	Angio	1.19 ± 0.16 (males)	
		0.89 ± 0.13 (females)	

epicardium throughout the cardiac cycle. The origin of the epicardial echo has been confirmed by finding an echo-free space posterior to it in patients with pericardial effusion (Feigenbaum *et al.*, 1968). The technique provides resolution of depth to 1 mm and with a repetition frequency of 1000/s, though limited to examination of a small region, for this it is nevertheless superior to cineangiography. It has been pointed out that estimates of end-systolic wall thickness from angiograms are subject to error introduced by exclusion of opacified blood from between trabeculae (Hugenholtz *et al.*, 1969; Guntheroth, 1974) and that the estimate of wall thickness obtained at necropsy is closest to the end-systolic value obtained *in vivo* (Maron *et al.*, 1977). Absolute values of end-systolic and end-diastolic posterior wall thickness in the normal subjects of previous studies are listed in Table 2, and are similar to those reported herein. Other authors who did not describe normal comparison series obtained values in patients with heart disease which are consistent with the remainder (Sjögren *et al.*, 1970; Troy *et al.*, 1972; Maron *et al.*, 1977).

Asymmetry between systole and diastole in the records of cavity dimension and wall thickness has previously been shown in animal experiments and by cineangiography in man (Feigl and Fry, 1964; Hawthorne *et al.*, 1966; Dumesnil *et al.*, 1974; Rankin *et al.*, 1976; Sasayama *et al.*, 1976; Gibson *et al.*, 1977). It resembles the asymmetry of the ventricular volume curve. During systole in normal subjects the wall thickens as cavity diameter lessens. After aortic valve closure there is further outward movement of the endocardium and increase in wall thickness before the end of isovolumic relaxation. The onset of mitral valve opening is, therefore, at the time of maximum wall thickness and minimum cavity dimension. From then transverse dimension increases first rapidly and then more slowly and there is a reciprocal rapid then slow decrease in wall thickness, so that the onset, peak, and termination of rapid filling and rapid increase in dimension are synchronous. In the presence of normal left ventricular function, endocardial movement is uniform so

that changes in transverse dimension reflect the filling pattern, and it might be concluded, therefore, that the shape of the normal volume curve, characterised by the rapid filling period, is linked to the pattern of wall thickness change, with its 'rapid thinning' period.

In order to explore further the relation between the pattern of change in wall thickness and endocardial position, we studied patients in whom the timing and co-ordination of endocardial movement are known to be abnormal. Upton *et al.* (1976) showed that characteristic features in patients with ischaemic heart disease and abnormal ventriculograms are delay in mitral valve opening relative to minimum dimension and abnormal movement of the endocardium during this extended isovolumic period. The patients with ischaemic heart disease in the present study behaved similarly, and in addition showed that abnormal changes in posterior wall thickness were responsible for the abnormal endocardial movement, thereby indicating that thinning of the ventricular wall can precede the start of filling of the cavity. The normal reciprocal relation between wall thickness and endocardial dimension was maintained, in that the onset of thinning was synchronous with minimum cavity dimension, peak rate of thinning occurred at the same time as the peak rate of outward wall movement, and the rapid thinning period was as long as the period of rapid increase of dimension. However, mitral valve opening was delayed with respect to minimum dimension and maximum wall thickness by up to 100 ms, so that much of the wall thinning in the region studied had already occurred before the onset of ventricular filling. Thus, the abnormal shift of endocardial position, already interpreted as indicating isovolumic change in cavity shape, is mediated by 'premature' thinning of the wall, and the latter, by analogous argument, indicates redistribution of myocardium around the constant volume of blood within the cavity. As one region of wall thins another must be becoming thicker.

In patients with left ventricular inflow obstruction, the result of mitral stenosis or the presence of a mechanical prosthesis, the behaviour of the free wall was affected in two ways. Frequently, the pattern of thinning reflected abnormal filling in that peak thinning rate was reduced as was the peak rate of increase in dimension and the thinning period was prolonged. However, in a number of patients, particularly those with Starr-Edwards prostheses, the peak thinning rate was more than would be predicted from the rate of change of dimension and was sometimes even normal. In these patients, the dissociation between posterior wall behaviour on the one hand and dimension increase and filling on the

Fig. 7 Mitral stenosis. Echocardiogram showing the septum and posterior left ventricular wall. Though the pattern of posterior wall thinning is nearly normal, septal movement in early diastole is almost parallel to the posterior endocardium, so that increase in cavity dimension is slow.

other hand occurred by virtue of the change in the pattern of septal movement. Frequently the septum moved rapidly in a posterior direction at the onset of posterior wall thinning (Fig. 7) and sometimes, in particular in the cases of Starr-Edwards valve replacement, its pattern was totally reversed.

In hypertrophic cardiomyopathy, isovolumic relaxation is frequently prolonged, as in ischaemic heart disease, but a previous study showed that there was a significant difference between the two in that in the former the extent of outward wall movement before mitral valve opening was much less. In the present study, early diastolic changes in wall thickness predicted the timing and extent of abnormal change in dimension, so that there was a correlation between the peak rate of thinning and the peak rate of wall movement, whether normal, abnormally slow, or abnormally fast and howsoever timed with relation to mitral valve opening. A feature of this condition is akinesis of the septum, so that a close relation between posterior wall behaviour and change in cavity size is to be expected, and the degree of abnormality detected indicates the extent to which myocardial disease is generalised.

We conclude that changes in left ventricular transverse dimension are largely mediated by changes in wall thickness. Normal diastole is characterised by a period of rapid wall thinning which is closely associated with rapid filling. However, decrease in wall thickness may be dissociated from filling and frequently precedes it in ischaemic heart disease, which we interpret as suggesting that

rapid thinning in early diastole is an intrinsic property of the wall. When relaxation is co-ordinated, then rapid thinning is accompanied by rapid filling, but when there is delayed relaxation of an ischaemic segment then rapid thinning can occur elsewhere without change in cavity volume, the result being delay in mitral valve opening and an isovolumic change in cavity shape. There is angiographic evidence to support the view that the ischaemic regions are those with delayed—'post-systolic'—thickening of the wall and that it is those areas with normal local function which manifest premature thinning (Gibson *et al.*, 1977). The existence of forces which cause rapid thinning of the posterior wall independent of filling would help to explain the finding in some patients with inflow obstruction that the peak thinning rate is normal despite the slow increase in cavity dimension, which entails rapid early diastolic posterior movement of the septum. If rapid thinning in early relaxation is indeed an intrinsic property of the left ventricular myocardium, its mechanical basis is unlikely to be explained by the pattern of relaxation of individual muscle fibres, but is more likely to lie in the forces generated between fibres as a result of the way in which they are arranged in space (Rushmer *et al.*, 1953). Thus, in hypertrophic cardiomyopathy, a disease characterised by fibre disarray, it is not unexpected that wide variations are encountered in the pattern of early diastolic behaviour. Early diastolic events may prove to be a fruitful field for study of the relation of left ventricular structure to function.

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