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Diastolic disease in left ventricular hypertrophy: comparison of M mode and Doppler echocardiography for the assessment of rapid ventricular filling

C H Lee, J C Hogan, D G Gibson

Abstract

***Objective*—To investigate possible discrepancies between M mode and Doppler echocardiography in assessing early diastolic filling.**

***Design*—Forty seven patients with left ventricular hypertrophy due to aortic stenosis and 26 healthy controls with a similar age range were studied by M mode, Doppler, apexcardiography, and phonocardiography. The patients also underwent cardiac catheterisation. M mode echograms were digitised by a computer. Early diastolic filling in both groups as assessed by the two techniques was compared.**

***Setting*—A tertiary cardiac referral centre with facilities for non-invasive and invasive investigations.**

***Subjects*—Patients referred for assessment of aortic stenosis who had left ventricular hypertrophy.**

***Main outcome measures*—Filling velocities on Doppler and rates of wall thinning and dimension increase on M mode.**

***Results*—Digitised M mode indices of diastolic filling (peak wall thinning rate 6·4 (3·0) *v* 10·0 (3·0) cm/s and peak rate of dimension increase 9·3 (3·3) *v* 16 (4·5) cm/s) in the patients and controls were consistently different. In contrast, the Doppler A/E ratio and peak E wave velocity were not; they varied widely among patients with left ventricular hypertrophy. In part, this variability was because the Doppler A/E ratio, but not the digitised M mode indices, was very sensitive to the abnormalities of isovolumic relaxation frequently present in left ventricular hypertrophy. The Doppler A/E ratio varied similarly with age in both normal and hypertrophied hearts; in the patients with ventricular hypertrophy the peak rate of dimension increase depended on age only, whereas the thinning rate was independent of age in both the patients and controls. Neither the A/E ratio nor the M mode indices could be related to the left ventricular end diastolic pressure or the peak aortic pressure difference.**

***Conclusions*—When Doppler and M mode techniques are used to assess rapid filling in patients with left ventricular hypertrophy the M mode indices are more consistently abnormal. The two methods measure different aspects of left ventricular diastolic function and should be regarded as complementary rather than interchangeable.**

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All phases of diastole can be abnormal in left ventricular hypertrophy. Particularly prominent is a reduced rate of rapid early diastolic filling, which has been assessed both by Doppler and digitised M mode techniques.[1 2] Because the physical basis of these techniques is different the information they give may not be the same, particularly as a recent comparison during isovolumic relaxation found major discrepancies between the two techniques.[3] We therefore examined possible differences in their performance in assessing rapid filling in a homogeneous group of patients with left ventricular hypertrophy due to aortic stenosis and compared the results with those from healthy controls of a similar age range.

Patients and methods

STUDY POPULATION

We studied 47 patients (24 men, 23 women, age 14–82) with left ventricular hypertrophy (end diastolic posterior wall thickness greater than 1·1 cm). All had clinical and investigative evidence of important aortic stenosis and were being considered for aortic valve replacement. Cardiac catheterisation and coronary angiography were undertaken as part of the preoperative assessment. All patients were in sinus rhythm and none had a dilated left ventricle (end diastolic dimension > 6 cm). Twenty six healthy controls (18 men, eight women, age 20–82) underwent similar non-invasive examinations.

M MODE ECHOCARDIOGRAPHY

M mode echograms of the mitral valve and left ventricle were recorded with a 3·0 MHz transducer and ATL Imager Mk 300I equipment. The left parasternal long axis view was used with the patient in the left lateral position. Phonocardiograms and electrocardiograms

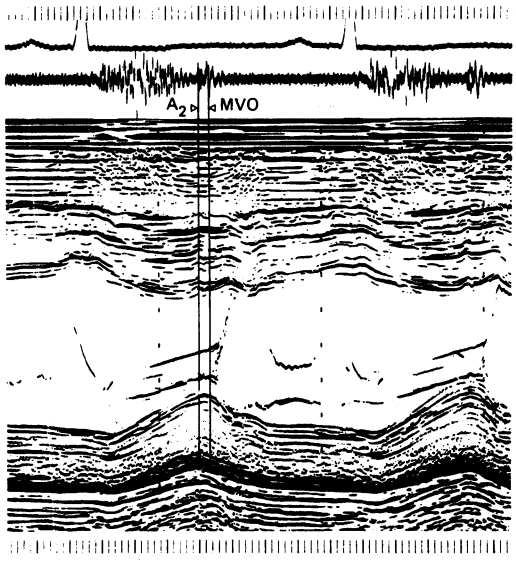

Figure 1 M mode echogram of the left ventricle from a patient with left ventricular hypertrophy secondary to aortic stenosis, showing aortic valve closure (A_2) on the phonocardiogram and mitral cusp separation (MVO) on the M mode.

were recorded simultaneously by a Honeywell recorder at a paper speed of 10 cm/s. Only records showing clearly the point of mitral cusp separation and continuous echoes from both the septum and posterior wall were used for analysis (fig 1). Aortic valve closure (A_2) was taken as the onset of the first high frequency vibration of the second heart sound recorded on the phonocardiogram and was checked with the aortic echograms and the aortic closure artefact on Doppler recordings. The isovolumic relaxation time was measured as the interval from A_2 to the initial separation of the mitral cusps. The thickness of the septum and posterior wall and the left ventricular internal dimension were measured at end diastole. The radius to thickness ratio was obtained by dividing half the left ventricular dimension by the average wall thickness.[4]

Echocardiograms were digitised[5] and the peak rates of posterior wall thinning during left ventricular relaxation (dW/dt) and the increase in left ventricular dimension (dD/dt) determined. The intervals from A_2 to peak dD/dt and from minimum cavity dimension to mitral valve opening were measured and the increase in transverse cavity dimension during that period expressed as a percentage of the total dimension change during the cardiac cycle (fig 2).

An approximation to left ventricular mass was calculated from the equation[6]:

$$\text{LV mass} = 1{\cdot}05\ ([\text{LVEDD} + \text{PW} + \text{IVS}]^3 - \text{LVEDD}^3) - 14 \text{ grams}$$

where LVEDD is the left ventricular end diastolic dimension (cm) and PW and IVS are the posterior wall and septal thickness in end diastole (cm), respectively. We used the leading edge for all measurements.

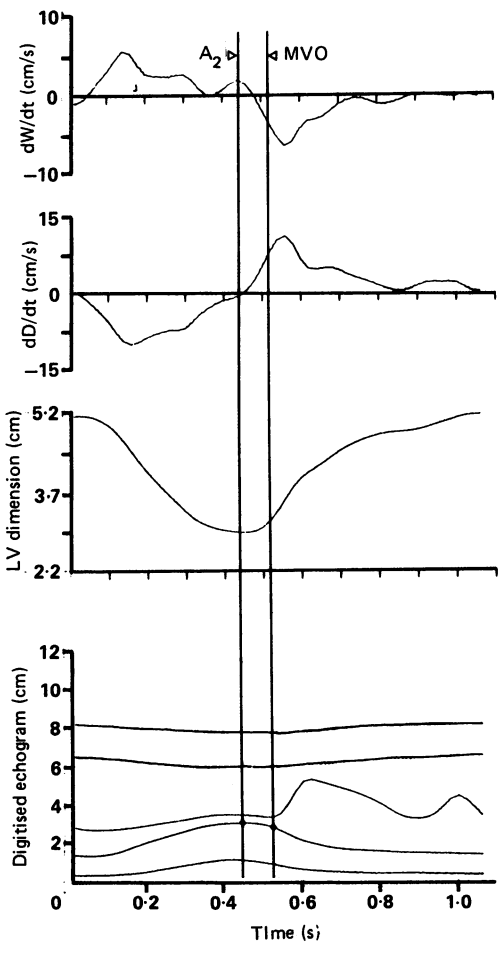

Figure 2 Digitised M mode echocardiogram showing the original digitised echogram, and plots of left ventricular dimension, rate of change in dimension (dD/dt), and rate of change in wall thickness (dW/dt). The two vertical lines correspond to the time of aortic valve closure (A_2) and mitral cusp separation (MVO).

DOPPLER ECHOCARDIOGRAPHY

Doppler recordings were made with Doptek equipment and a 2·0 MHz transducer. Forward transmitral flow was recorded in all patients. Peak mitral flow velocity was initially identified by continuous wave Doppler and then recorded with pulsed mode and a 3 mm gate. A wall filter of 250 Hz was used in all cases. Records were taken at a paper speed of 10 cm/s, again with simultaneous phonocardiograms and electrocardiograms. The peak velocities of the atrial (A) wave and the early (E) wave were recorded and their ratio (A/E ratio) derived. The intervals from A_2 to peak transmitral flow velocity and A_2 to peak dD/dt were measured and the phase lag between the two determined.[7] The peak instantaneous pressure difference across the aortic valve was determined in all patients by continuous wave Doppler from the apical position. Mean values from three cardiac cycles were used in the analysis.

APEXCARDIOGRAMS

The apexcardiogram was recorded from the point of maximum cardiac impulse with the patient in the left lateral position by a Cambridge transducer with a time constant of 4 seconds. The ratio of the height of the A wave to the total deflection (A/H) was calculated and the mean of three cardiac cycles recorded.

CARDIAC CATHETERISATION

The patients studied required coronary arteriography to assess their disease and the aortic valve had been crossed in all of them. The procedure was performed via either the brachial or femoral artery. Pressure was recorded before left ventricular angiography by a fluid filled 8F pigtail catheter attached to a manifold micromanometer transducer (Medex Medical). The left ventricular end diastolic

Table 1 Mean (SD) values of Doppler and M mode measurements in patients with left ventricular hypertrophy

	Controls	Patients
Age (yr)	50 (17)	58 (17)
RR intervals (ms)	930 (155)	850 (150)
A wave (m/s)	0·54 (0·12)	0·68 (0·29)*
E wave (m/s)	0·70 (0·18)	0·83 (0·29)
A/E ratio	0·79 (0·28)	0·91 (0·50)
Isovolumic relaxation time (ms)	60 (9)	65 (25)
Minimum dimension to mitral opening (ms)	36 (22)	72 (43)*
Percentage dimension change before mitral opening (ms)	5 (4)	17 (10)*
Peak dW/dt (cm/s)	10 (3)	6·4 (3)*
Peak dD/dt (cm/s)	16 (4·5)	9·3 (3·3)*
LV end diastolic dimension (cm)	5 (0·6)	4·8 (1·0)
Posterior wall thickness (cm)	0·86 (0·11)	1·2 (0·3)*
Septal thickness (cm)	1·0 (0·1)	1·6 (0·4)*
A/H ratio	0·18 (0·05)	0·27 (0·14)*
LV mass (g)	200 (50)	325 (145)*
Phase lag (ms)	68 (14)	66 (30)
Radius/thickness ratio	2·7 (0·4)	1·78 (0·6)*

*p < 0·01.

pressure was taken as the point on the diastolic waveform at the onset of the QRS complex on the simultaneously recorded electrocardiogram. The mean of 10 cardiac cycles was recorded. All studies were done within 48 hours of the echocardiographic examination.

STATISTICAL ANALYSIS

Values are quoted as mean (SD). Relations between variables were analysed by least squares linear regression. Patients with left ventricular hypertrophy were, in addition, divided into three groups according to the A/E ratio (> 0·9, 0·6–0·9, < 0·6). The range 0·6–0·9 was used as an approximation of the 99% confidence limit of our normal range (0·63–0·94). Student's unpaired *t* test was used to compare group mean values.

Results

Tables 1 and 2 show mean values of the measurements.

M MODE MEASUREMENTS AND GENERAL DATA

Left ventricular mass (325 (145) *v* 200 (50) g) and wall thickness were, by definition, increased in patients with left ventricular hypertrophy. Relaxation was very incoordinate in hypertrophied hearts compared with normal, as shown by the long interval from minimum cavity dimension to mitral valve opening (70 (45) *v* 35 (20) ms) and a large increase in transverse dimension before the mitral valve was opened (17 (10) *v* 5 (4) %). The A/H ratio on the apexcardiogram was higher in the hypertrophy group (0·27 (0·14) *v* 0·18 (0·05). There were no significant differences in mean age, RR interval, isovolumic relaxation time, and phase lag between the patients and the controls.

Coronary arteriography was normal or showed minor irregularities only in 35 patients but in 12 the disease was severe enough to warrant coronary artery grafting. Patients with coronary artery disease were older (65 (8) *v* 56 (19) years) than those without and their isovolumic relaxation time was longer (80 (25) *v* 60 (20) ms, p < 0·05). There were no other significant differences. Specifically, neither the interval from minimum cavity dimension to mitral valve opening (75 (40) *v* 70 (45) ms) nor the percentage change in transverse dimension before mitral valve opening (18 (12) *v* 16 (10) %) was different in the patients with coronary artery disease and those without.

SPECIFIC DIGITISED M MODE INDICES

Peak dD/dt (16 (4·5) *v* 9·3 (3·3) cm/s) and peak dW/dt (10·0 (3·0) *v* 6·4 (3·0) cm/s) were both significantly lower in patients with aortic stenosis than in the controls. They were consistently abnormal in hypertrophied hearts, even when the A/E ratio was apparently normal.

DOPPLER MEASUREMENTS

As a group, patients with left ventricular hypertrophy had a higher mean A wave velocity (0·68 (0·29) *v* 0·54 (0·12) m/s). However, the mean values for the A/E ratio and peak E wave velocity were not significantly

Table 2 Subgroup analysis of patients with left ventricular hypertrophy

	Left ventricular hypertrophy		
Variable	*Dominant E (A/E < 0·6) (n = 11)*	*Dominant A (A/E > 0·9) (n = 19)*	*Normal (0·6 < A/E < 0·9) (n = 17)*
Age (yr)	48 (19)*†	68 (8)	56 (18)
RR interval (ms)	810 (90)	880 (160)	830 (170)
A wave (m/s)	0·39 (0·24)*†	0·84 (0·22)	0·70 (0·24)
E wave (m/s)	1·0 (0·24)*†	0·62 (0·13)	0·95 (0·30)
A/E ratio	0·37 (0·22)*†	1·38 (0·39)	0·73 (0·09)
Isovolumic relaxation time (ms)	39 (23)*†	80 (15)	64 (22)
Minimum dimension to mitral opening (ms)	49 (21)*	96 (43)	51 (33)
Percentage dimension change before mitral opening (ms)	12 (10)*	21 (10)	15 (8)
Peak dW/dt (cm/s)	5·6 (2·9)	5·9 (2·4)	7·5 (4·0)
Peak dD/dt (cm/s)	10·3 (3·1)*	7·4 (2·7)	10·6 (3·4)
Left ventricular end diastolic dimension (cm)	5·3 (1·0)*	4·4 (0·8)	4·8 (1·1)
Posterior wall thickness (cm)	1·2 (0·4)*	1·1 (0·3)	1·3 (0·4)
Septal thickness (cm)	1·3 (0·4)*	1·8 (0·2)	1·5 (0·5)
A/H ratio	0·31 (0·16)	0·21 (0·1)	0·30 (0·14)
Left ventricular end diastolic pressure (mm Hg)	25 (10)	20 (9)	22 (6)
Peak aortic pressure difference (mm Hg)	70 (29)	60 (30)	66 (25)
Left ventricular mass (g)	320 (95)	320 (60)	330 (75)
Phase lag (ms)	58 (30)*	92 (30)	48 (34)

*p < 0·05 *v* group with dominant A wave;
†p < 0·05 *v* group with normal A/E ratio.

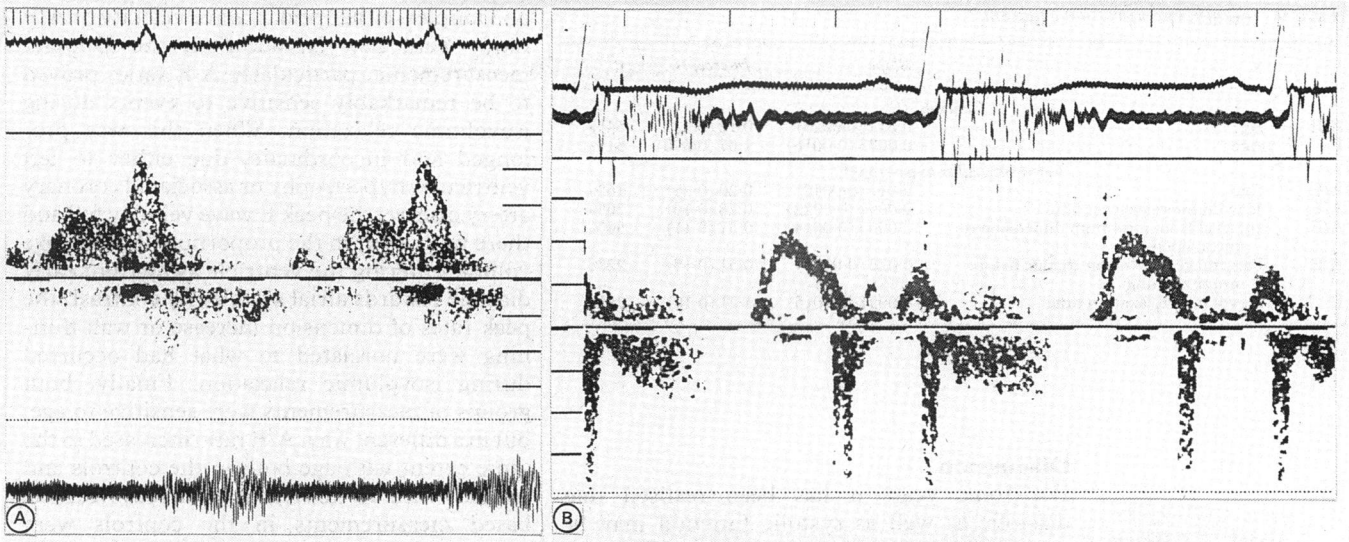

Figure 3 Pulsed Doppler recordings of transmitral flow velocity from two patients with left ventricular hypertrophy owing to aortic stenosis, showing the wide variation in A/E ratio in this condition. (A) A dominant A wave and (B) a dominant E wave. Full scale deflection is 4 kHz.

different in the controls and patients with left ventricular hypertrophy.

The Doppler A/E ratio varied widely in patients with left ventricular hypertrophy, ranging from a dominant E wave to a dominant A wave (fig 3). To investigate possible underlying factors, we divided the patients into three groups according to the A/E ratio, depending on whether it is greater, less than, or within the 99% confidence interval of normal. The group with a dominant E wave were younger (48 (19) *v* 68 (8) years), had a shorter isovolumic relaxation time (39 (23) *v* 80 (15) ms), a shorter interval from minimum cavity dimension to mitral valve opening (49 (21) *v* 96 (43) ms), a higher peak rate of dimension increase in early diastole (10·3 (3·1) *v* 7·4 (2·7) cm/s), a larger cavity size at end diastole (5·3 (1·0) *v* 4·4 (0·8) cm), a thinner septum (1·3 (0·4) *v* 1·8 (0·2) cm), and a smaller phase lag between dimension increase and transmitral flow (58 (30) *v* 92 (30) ms) than those with a dominant A wave (all p values < 0.05). The peak rates of posterior wall thinning and dimension increase were consistently abnormal across the three groups and there were no significant differences between them. The same applied to left ventricular end diastolic pressure, A/H ratio on the apexcardiogram, and the peak instantaneous aortic pressure difference.

Table 3 Regression analysis for patients with left ventricular hypertrophy

Variable	*A/E ratio*	*E wave velocity*	*Peak dD/dt*	*Peak dW/dt*
Dimensions				
Left ventricular end diastolic dimension	NS	NS	NS	NS
Radius to thickness ratio	NS	NS	NS	NS
Left ventricular mass	NS	NS	NS	
Isovolumic relaxation events				
Isovolumic relaxation time	$p < 0.01$ (30%)	$p < 0.01$ (27%)	NS	NS
Interval from minimum cavity dimension to mitral valve opening	$p < 0.01$ (50%)	NS	NS	NS
Percentage dimension change before mitral opening	$p < 0.01$ (22%)	NS	NS	NS
Age	$p < 0.01$ (16%)	NS	$p < 0.01$ (35%)	NS
Loading conditions				
Left ventricular end diastolic pressure	NS	NS	NS	NS
Peak left ventricle − aorta pressure difference	NS	NS	NS	NS

*Figures in brackets are the values for r^2.

REGRESSION ANALYSIS BETWEEN VARIABLES (TABLES 3 AND 4)

Age

The A/E ratio depended on age in both the controls and the patients with hypertrophied hearts; the regression equations for the two groups were indistinguishable. The A/E ratio thus varied with age in the same way in the normal and hypertrophied hearts.

In contrast, peak dD/dt was unrelated to age in the controls, but was sensitive to age in those with left ventricular hypertrophy. Peak dW/dt, though, was independent of age in both groups.

Isovolumic relaxation events

In patients with left ventricular hypertrophy the A/E ratio also correlated with events during isovolumic relaxation (isovolumic relaxation time, interval from minimum dimension to mitral opening, and percentage dimension change before mitral opening) and the E wave velocity correlated with the isovolumic relaxation time.

The peak rates of dimension increase (dD/dt) and wall thinning (dW/dt) by contrast were independent of events during isovolumic relaxation in both the normal and hypertrophied hearts.

Loading conditions

The Doppler A/E ratio, peak E wave velocity, and the digitised M mode indices (peak dD/dt, dW/dt) were independent of the left ventricular end diastolic pressure and the peak instantaneous aortic pressure difference.

Geometrical dimensions

Left ventricular end diastolic dimension, left ventricular mass, and radius to thickness ratio did not affect Doppler A/E ratio, E wave velocity, peak dD/dt, or peak dW/dt.

*Table 4 Significant regression equations**

y	x	Slope	Intercepts	r^2
		Controls		
A/E	Age	0·012 (0·0023)	0·18 (0·12)	54%
E	Age	−0·0073 (0·0016)	1·07 (0·090)	51%
		Left ventricular hypertrophy		
A/E	Age	0·012 (0·0042)	0·20 (0·25)	16%
A/E	Isovolumic relaxation time	0·0092 (0·0022)	0·28 (0·16)	30%
A/E	Interval from minimum dimension to mitral opening	0·0083 (0·0014)	0·37 (0·11)	50%
A/E	Percentage dimension change before mitral opening	0·022 (0·0076)	0·51 (0·15)	22%
E	Isovolumic relaxation time	−0·0057 (0·0015)	1·21 (0·10)	28%

*$p < 0·01$.

Discussion

For some years it has been realised that diastolic as well as systolic function may be abnormal in left ventricular disease and that it is possible to quantify such disturbances non-invasively. An early criterion was the diastolic closure rate of the normal mitral valve, as detected by M mode echocardiography.[8] While this was undoubtedly reduced in patients with various ventricular diseases, as a measurement it proved to be multifactorial and difficult to determine unambiguously, so its use in this way has been effectively abandoned. A second approach was to observe the rates of increase of cavity dimension and wall thinning by digitising M mode echocardiograms. This proved effective in delineating disturbed early diastolic function in several conditions, including left ventricular hypertrophy,[9] where the rate was reduced, and coronary artery disease, where wall motion appeared incoordinate.[10] These findings were validated by contrast angiography,[11] and confirmed by radionuclide[12] angiography. As cross sectional echocardiography has developed, however, and in particular as colour flow Doppler has appeared, M mode echocardiograms are recorded less and less frequently. The method is held to be time consuming and some find it difficult to obtain records of adequate quality for digitisation.[13]

During the same period, Doppler methods have been used more widely. They also allow early diastolic function to be inferred from the velocity of inflow into the ventricle, either in absolute terms, or, more commonly, as the ratio of velocity during atrial systole to that in early diastole (A/E ratio). In view of the M mode and other findings, it is not surprising that these Doppler measurements are also abnormal in patients with left ventricular hypertrophy. Peak early diastolic filling velocity was reduced and the A/E ratio increased. It seems that the two approaches measure the same disturbance, and so could be used interchangeably, with Doppler displacing M mode altogether. We designed the present study to examine whether this was so.

Despite their superficial similarity, our results show clear differences between methods based on M mode and Doppler when they are used to assess events during early diastole. Digitised M mode echocardiographic indices were consistently abnormal in patients with left ventricular hypertrophy, whereas those based on Doppler, either peak E wave velocity or A/E ratio, were not. In addition, the Doppler measurements, particularly A/E ratio, proved to be remarkably sensitive to events during isovolumic relaxation. When this was prolonged and incoordinate, due either to left ventricular hypertrophy or associated coronary artery disease, the peak E wave velocity fell and there was a shift in the proportion of the stroke volume entering the ventricle away from early diastole towards atrial systole. By contrast, the peak rates of dimension increase or wall thinning were unrelated to what had occurred during isovolumic relaxation. Finally, both groups of measurements were sensitive to age, but in a different way. A/E ratio increased to the same extent with age both in the controls and patients with aortic stenosis, whereas M mode based measurements in the controls were apparently insensitive to age. Age did, however, affect the M mode measurements in patients with aortic stenosis. It is clear, therefore, that the two types of measurement perform quite differently in this group of patients with left ventricular hypertrophy caused by a single, clearly defined entity (aortic stenosis), and therefore that they cannot be taken to be interchangeable.

If Doppler and M mode did indeed both measure early diastolic left ventricular filling rate, these differences would be hard to explain. However, both assess it only indirectly. Doppler echocardiography measures a peak velocity rather than a filling rate, and M mode measures a dimension rather than a volume. It is thus likely that in these indirect relations lies the explanation of our apparently conflicting results. Filling rate is related to blood velocity by cross sectional area; there is though no reason to suppose that this is consistently abnormal in patients with left ventricular hypertrophy unless the cavity shape is very distorted. It has previously been shown, however, that discrepancies between changes in cavity dimension and cavity volume are particularly obvious during early diastole. They are caused by a change in cavity shape with part, or even all the diastolic increase in cavity dimension occurring before mitral valve cusp separation and thus in the absence of volume change.[14] In these circumstances, the rapid early diastolic dimension increase and wall thinning measured by M mode echocardiography may still occur normally, and should thus be regarded as manifestations of an autonomous process normally coupled to filling. Rapid dimension increase and particularly rapid wall thinning may thus be a direct cause of rapid filling; they certainly do not depend on it. Even in healthy controls, the cavity dimension consistently leads flow velocity as measured by Doppler, again showing that it can potentially be separated from filling rate.[7] It was thus of interest that peak dimension increase and wall thinning rate proved so much more sensitive than Doppler in detecting disturbed rapid filling in patients with left ventricular hypertrophy.

The lack of sensitivity of the most commonly used Doppler measurement of ventricular dia-

stolic function, the A/E ratio, seemed to have several causes. Though it was related to age, this relation was less strong than for the M mode measurements, and so could not have been the only cause of the very wide scatter seen in the patients with left ventricular hypertrophy. In patients with a dominant E wave, cavity size tended to be greater and wall thickness less, while isovolumic relaxation was shorter. This was not due to a high left ventricular end diastolic pressure. In approximately one third of patients the A wave was abnormally small or absent. This might suggest "left atrial failure", possibly because of chronically raised left atrial pressure. However, the relative height of the A wave on the apexcardiogram was normal or even increased in these patients, showing that left atrial mechanical activity was intact. The combination of a normal increase in atrial pressure as reflected in the apexcardiogram[15] with a lower than normal increase in volume thus strongly suggest that end diastolic left ventricular compliance was reduced in some patients with left ventricular hypertrophy. No such effect could be identified as influencing the M mode measurements.

At first sight, it is surprising that neither of the two sets of measurements were sensitive to loading conditions, as assessed by left ventricular end diastolic pressure. Not only is there much experimental evidence to suggest that such a relation should exist,[16 17] but, more importantly, it should follow directly from the physics of flow. Blood flow velocity, however, is not directly proportional to end diastolic pressure, but depends on Newton's Laws of motion. Blood velocity accelerates in response to a force, expressed in a liquid as a pressure gradient with the physical dimensions of mm Hg per cm. During early diastole, the pressure gradient is changing very rapidly as atrial and ventricular pressure approach one another. It is also affected by ventricular restoring forces, which effectively lower left ventricular pressure and are asymmetrically distributed throughout the ventricle. Rather than a simple pressure difference, therefore, pressures are likely to vary not only with time, but also in space, across the ventricular inflow. Finally, the pressure gradient relates to blood acceleration not blood velocity; velocity depends also on the time over which the acceleration has operated as well as on acceleration itself. It would thus be simplistic to suppose that these complex physical determinants of early diastolic flow could possibly be described by a single value of end diastolic pressure. It is thus not surprising that our results show no relation between the two. The absence of such a relation does not, of course, prove that blood flow is independent of loading conditions, but it does show that the appropriate loading conditions in humans are difficult to measure. If the early diastolic increase in transverse dimension and fall in wall thickness are autonomous processes, as suggested above, then one would not expect them to be related even to appropriate measures of loading conditions. Indeed, if they are related to restoring forces, they will contribute to the overall atrioventricular pressure gradient. For these quite different reasons, we were not surprised at finding no relation between left ventricular end diastolic pressure and either of our two sets of measurements.

Our results suggest that the relation between age and diastolic function in patients with left ventricular hypertrophy may be more complex than has previously been supposed.[18 19] In healthy controls M mode measurements did not depend on age; the relation seen in the patients with left ventricular hypertrophy, therefore, might be because in older individuals the hypertrophy has been more long standing and its functional disturbances more severe. This would imply that the natural course of left ventricular outflow tract obstruction is longer in the elderly, an assumption for which there is no evidence. Alternatively, the response to hypertrophy when the heart is young may differ from that when it is old. The Doppler results also give some support to this idea. Patients with lower A/E ratios were younger and though their ventricular mass was identical their cavity size was larger, and, in particular, late diastolic filling was often restricted. It is thus possible that the slowly filling ventricle with a prolonged isovolumic relaxation time and an accentuated A wave on Doppler is a feature not of hypertrophy alone, but of hypertrophy in the elderly. Younger patients seem to respond to hypertrophy with a more restrictive filling pattern. The peak early inflow velocity is maintained but that in late diastole falls, so the A/E ratio is low.

Our results thus show that M mode and Doppler measurements of early diastolic function are not identical in patients with left ventricular hypertrophy secondary to aortic stenosis. M mode measurements seemed more specific, in being unaffected by events during isovolumic relaxation and in discriminating more completely between normal and abnormal. They are less directly related to early diastolic transmitral flow rate, but may well reflect processes within the ventricular wall which underlie the restoring forces. Since ventricular filling is closely related to the effects of restoring forces, the two approaches to assessing early diastolic function can therefore be regarded as complementary rather than exclusive or interchangeable. Their combined use, especially with other non-invasive methods, is likely to be much more productive than that of either alone.

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