Echocardiographic left ventricular dimensions in pressure and volume overload

Their use in assessing aortic stenosis

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Left ventricular 'relative wall thickness', determined from the ratio between echocardiographic measurements of end-systolic wall thickness and cavity transverse dimension, was related to peak systolic intraventricular pressure in 15 normal subjects, in 15 patients with left ventricular volume or pressure overload without aortic stenosis, and in 23 patients with aortic stenosis. All these patients had a mean rate of circumferential fibre shortening greater than 1.0 circumference per second and were regarded as having good ventricular function. Relative wall thickness was found to be normal in cases of volume overload and to be increased in pressure overload, being proportional to the systolic intraventricular pressure. Values for the ratio of systolic intraventricular pressure to relative wall thickness in the normal subjects and patients without aortic stenosis were similar (mean 30 ± 2.5). Based on this relation, estimates of peak systolic intraventricular pressure were made in the cases of aortic stenosis using the formula: systolic intraventricular pressure (kPa) = $30 \times \text{wall thickness} \div \text{transverse dimension}$. Peak systolic aortic valve gradients derived by subtracting brachial artery systolic pressure, measured by sphygmomanometer, from the echocardiographic estimates of intraventricular pressure compared favourably with the gradients measured at left heart catheterization ($r=0.87$, $P<0.001$).

Aortic valve orifice areas, derived from echocardiographic estimates of stroke volume, ejection time, and valve gradient, ranged from 0.21 to 3.16 cm² and appeared to correlate with the severity of aortic stenosis. All patients with aortic stenosis, with or without coexistent mild aortic regurgitation, who were recommended for aortic valve surgery, had estimated valve orifice areas of less than 0.8 cm².

A further 10 patients with pressure or volume overload had mean rates of circumferential fibre shortening of less than 1.0 circumference per second and were regarded as having poor ventricular function. In these cases values for relative wall thickness were lower than in those with good ventricular function and were not proportional to systolic intraventricular pressure.

In patients with good left ventricular function systolic intraventricular pressure is proportional to, and can be estimated from, echocardiographic measurement of relative wall thickness.

The relation between left ventricular dimensions and load has been demonstrated by angiographic studies. Left ventricular wall thickness increases as a result of, and generally in proportion to, both pressure and volume overload (Levine, Rockoff, and Braunwald, 1963; Grant, Greene, and Bunnell, 1965). Left ventricular cavity size increases in proportion to a volume overload (Kennedy *et al.*, 1968, 1970); no increase occurs in pure pressure overload (Linzbach, 1960; Levine *et al.*, 1963).

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'Relative wall thickness', the ratio of wall thickness to cavity minor axis, remains normal in pure volume overload but is increased in pressure overload (Levine *et al.*, 1963; Grant *et al.*, 1965). In cases of poor left ventricular function, cavity dilatation may occur even in the absence of a volume overload and under this circumstance relative wall thickness may be reduced in both volume and pressure overload (Grant *et al.*, 1965). Wall stress studies suggest that systolic ventricular dimensions are more closely related than diastolic dimensions to left ventricular

load (Sandler and Dodge, 1963; Hood, Rackley, and Rolett, 1968).

Echocardiography can be used to measure left ventricular wall thickness (Feigenbaum *et al.*, 1968; Sjögren, Hytönen, and Frick, 1970; Troy, Pombo, and Rackley, 1972). The echocardiographic left ventricular cavity transverse dimension has been shown to approximate to the minor axis of the left ventricular cavity measured angiographically (Fortuin *et al.*, 1971; Pombo, Troy, and Russell, 1971; Feigenbaum *et al.*, 1972; Gibson, 1973).

In this study relative wall thickness, determined from the ratio between echocardiographic measurements of end-systolic wall thickness and cavity transverse dimension, was related to peak systolic brachial artery pressure (assuming close approximation to peak systolic intraventricular pressure) in normal subjects and in patients with left ventricular hypertrophy not caused by aortic stenosis. In accordance with the angiocardiographic findings, it was found that relative wall thickness was increased in pressure overload and that, in the absence of impaired ventricular function, relative wall thickness was proportional to systolic intraventricular pressure. Based on this relation, estimates of peak systolic intraventricular pressure were made from

echocardiographic ventricular dimensions in the patients with aortic stenosis. In these cases systolic brachial artery pressure was subtracted from the calculated intraventricular pressure in order to derive an estimate of the peak systolic pressure gradient across the aortic valve. The estimated gradients were compared with those measured at left heart catheterization.

Using a modification of Gorlin and Gorlin's (1951) formula, involving peak rather than mean systolic pressure gradients (Bache, Jorgensen, and Wang, 1972), aortic valve orifice areas were calculated in the patients with aortic stenosis using echocardiographic estimates of stroke volume, ejection time (Fortuin, Hood, and Craige, 1972), and valve gradient.

Subjects and methods

Data were obtained from 15 normal subjects, 22 patients with left ventricular hypertrophy caused by volume or pressure overload, but not by aortic stenosis, and from 26 patients with aortic stenosis who had undergone left heart catheterization. Most of the patients without aortic stenosis, including all those with an aortic systolic murmur, had also undergone left heart catheterization for clinical purposes, at which the absence of a pressure

FIG. Left ventricular echocardiogram showing left ventricular posterior wall (PLVW), interventricular septum (IVS), end-diastolic and end-systolic transverse dimensions (D_d and D_s , respectively), end-systolic wall thickness (W), and ejection time (T).

gradient across the aortic valve had been confirmed. None of the patients studied had evidence of coronary artery disease.

Left ventricular echocardiograms were recorded either on Polaroid film using an Eskoline 20 ultrasonoscope or on light sensitive paper using a Honeywell 1856 strip chart recorder and Ekoline 20A ultrasonoscope. The patients rested for at least 15 minutes before the records were taken. The ultrasound transducer, either a 2.25 MHz unfocused or 10 cm focused transducer, was placed parasternally on the left fourth or fifth intercostal space and directed posteriorly and slightly laterally and inferiorly until the interventricular septum, mitral valve apparatus, and posterior left ventricular wall were located. Brachial artery systolic pressure was measured, using a sphygmomanometer, immediately after the echocardiograms were recorded.

Measurement of end-systolic cavity dimension (D_s), end-diastolic cavity dimension (D_d), end-systolic wall thickness (W_s), and ejection time (T) were made from the echocardiograms with the aid of the simultaneously recorded calibration scale (Fig.). Cavity dimension was measured as the distance between the endocardium of the septum and that of the posterior left ventricular wall. Wall thickness was measured as the distance between the inner surface of the endocardium and the outer surface of the epicardium of the posterior wall. Ejection time was taken as the time between the initial and the maximum anterior movement of the posterior wall. End-systole was defined as the point of maximum anterior movement of the posterior wall and end-diastole as the time of onset of the QRS complex of the simultaneously recorded electrocardiogram.

Apart from the normal subjects, all cases had an increased left ventricular mass as estimated by echocardiography (Bennett and Evans, 1974). The mean velocity of circumferential fibre shortening (V_{cf}), calculated according to the method of Fortuin *et al.* (1972), was used as an index of ventricular performance:

$$V_{cf} = \frac{D_d - D_s}{T} \text{ circumferences per second.}$$

Those patients whose V_{cf} was less than the lowest value found in the normal subjects were regarded as having poor left ventricular function.

The ratio (c) of peak systolic pressure (P) to relative wall thickness (W_s/D_s) was calculated, thus:

$$c = P \times D_s / W_s.$$

Peak systolic intraventricular pressure was calculated in the cases of aortic stenosis using the echocardiographic measurements of their ventricular dimensions and the mean value for c derived from the normal subjects and the patients without aortic stenosis, who were regarded as having good left ventricular function, thus:

$$P = c \times W_s / D_s.$$

Aortic valve systolic gradients were obtained by subtracting the brachial artery systolic pressure from the calculated peak systolic intraventricular pressure. The estimated gradients were compared with those measured at left heart catheterization. In 6 cases the echocardiograms were recorded at the time of catheterization.

Estimates of stroke volume, derived by subtracting the cube of the end-systolic cavity dimension from the cube of the end-diastolic cavity dimension (Feigenbaum *et al.*, 1969), were used, together with the echocardiographic estimates of gradient and ejection time to calculate the areas of the aortic valve orifices using the formulae of Bache *et al.* (1972):

$$\text{Valve orifice area} = \frac{(D_d^3 - D_s^3)/T}{37.8 \sqrt{\text{PPSG} + 10}}$$

where PPSG = peak to peak systolic gradient in mmHg, or gradient $\times 7.5$ in SI units.

The catheterization data were measured originally to the nearest 1 mmHg and sphygmomanometric data to the nearest 5 mmHg. The pressures were then converted to SI units by dividing by 7.5. The SI units so derived are expressed to 2 decimal places but it is not intended to imply that the pressure measurements or echocardiographic estimates of intraventricular pressure are accurate to this degree.

Results

Data from the normal subjects are given in Table 1. Values for c were similar, the mean being 29.1 (SD ± 1.8). Values for V_{cf} ranged from 1.03 to 1.62 (mean 1.34, SD ± 0.16) circumferences per second. Patients whose V_{cf} was less than 1.0 circumference per second were regarded as having poor left ventricular function.

Data from the patients with left ventricular hypertrophy not caused by aortic stenosis, with good left ventricular function, are given in Table 2. Relative wall thickness was greater in the patients with high systolic pressures. In spite of a considerable range of values for W_s (1.6–2.7 cm), D_s (2.8–5.0 cm), and P (14–24 kPa), values for c varied little

TABLE 1 Data from normal subjects

Subject	Age (yr)	BP (kPa)	V_{cf} (cf/s)	W_s (cm)	D_s (cm)	W_s/D_s	c
1	29	14.67	1.40	1.6	3.3	0.48	30.2
2	32	14.00	1.62	1.3	2.5	0.52	26.9
3	38	15.33	1.30	1.4	2.8	0.50	30.7
4	25	14.67	1.48	1.5	3.1	0.48	30.3
5	27	17.33	1.41	1.5	2.5	0.60	28.9
6	27	16.00	1.31	1.6	3.0	0.53	30.0
7	27	13.33	1.10	1.5	3.1	0.48	27.5
8	29	13.33	1.20	1.4	3.1	0.45	29.5
9	28	14.67	1.48	1.4	2.7	0.52	28.3
10	27	19.33	1.20	1.9	3.0	0.63	30.5
11	19	14.67	1.36	1.6	2.9	0.55	26.6
12	29	13.33	1.34	1.5	3.0	0.50	26.7
13	25	15.33	1.35	1.5	2.7	0.55	27.6
14	25	16.00	1.52	1.5	3.1	0.48	33.1
15	25	14.00	1.03	1.5	3.1	0.48	28.9

Conversion factor from SI units to Traditional units: 1 kPa $\approx$ 7.5 mmHg.

TABLE 2 Data from patients with left ventricular hypertrophy (not caused by aortic stenosis) and normal ventricular function

Patient	Age (yr)	Diagnosis	BP (kPa)	Vcf (cf/s)	W _s (cm)	D _s (cm)	W _s /D _s	c
1	17	Coarctation	22.67	1.71	2.3	3.0	0.77	29.6
2	30	"	21.33	1.39	2.0	2.8	0.71	29.9
3	31	"	22.00	1.75	2.0	3.0	0.67	33.0
4	24	"	24.00	1.37	2.4	2.8	0.86	28.0
5	44	AR	16.67	1.52	1.9	3.3	0.58	28.9
6	46	"	18.67	1.33	2.6	4.2	0.62	30.2
7	27	"	17.33	1.30	1.9	3.4	0.56	31.0
8	37	"	14.00	1.37	1.6	3.6	0.44	31.5
9	42	"	18.00	1.54	2.7	5.0	0.54	33.3
10	17	AR, VSD	16.00	1.15	2.0	4.2	0.48	33.6
11	26	" "	18.67	1.23	1.9	3.6	0.53	35.4
12	51	MR	14.67	1.34	2.0	3.4	0.59	24.9
13	26	"	15.33	1.31	2.3	5.0	0.46	33.3
14	8	"	14.67	1.54	1.6	3.1	0.52	28.4
15	39	AR, MR	21.33	1.70	2.5	3.8	0.66	32.4

AR – aortic regurgitation; MR – mitral regurgitation.

VSD – ventricular septal defect.

Conversion factor from SI units to Traditional units: 1 kPa $\approx$ 7.5 mmHg.

TABLE 3 Data from patients with aortic stenosis and good ventricular function

Patient	Age (yr)	Additional lesions	Echocardiographic data						Catheterization data				
			Vcf (cf/s)	T (s)	W _s (cm)	D _s (cm)	D _a (cm)	W _s /D _s	P (kPa)	Gradient (kPa)	Valve area (cm ²)	P (kPa)	Gradient (kPa)
1*	18	SD ar	1.83	0.26	2.0	2.1	4.0	0.95	28.57	15.23	0.51	25.33	12.00
2*	11	" "	1.70	0.32	2.0	2.2	4.8	0.91	27.27	13.94	0.77	26.67	13.33
3*	17	" "	2.10	0.25	2.6	1.9	4.0	1.37	41.05	30.40	0.39	32.00	21.33
4*	52	"	1.14	0.30	2.5	2.5	3.8	1.00	30.00	18.67	0.28	32.00	20.67
5*	56	AR	1.53	0.30	2.5	2.7	5.0	0.93	27.78	5.07	1.34	26.67	5.33
6	15	ar	1.20	0.29	1.8	2.6	4.0	0.69	20.77	7.47	0.52	21.33	6.67
7*	52	"	1.36	0.33	2.0	2.2	4.0	0.91	27.27	8.67	0.49	30.40	10.67
8*	52	"	1.24	0.30	2.2	2.7	4.3	0.81	24.44	5.73	0.73	26.67	8.00
9	11	"	1.42	0.29	1.8	2.7	4.6	0.67	20.00	9.33	0.79	18.67	7.33
10*	59	"	1.60	0.28	2.5	2.2	4.0	1.14	34.09	21.47	0.38	34.67	20.67
11*	70	"	1.12	0.32	2.5	2.5	3.9	1.00	30.00	12.67	0.35	25.33	13.33
12	39	ar	1.50	0.30	2.2	2.3	4.0	0.96	28.70	12.67	0.41	26.00	8.00
13	42	"	2.10	0.26	1.9	1.8	4.0	1.05	31.67	17.60	0.50	24.00	13.33
14	20	"	1.40	0.28	2.0	3.1	5.1	0.65	19.35	2.00	1.95	20.67	4.00
15*	58	"	1.50	0.27	2.5	2.3	3.7	1.09	32.60	13.33	0.36	30.67	14.00
16	33	"	2.00	0.25	2.1	2.6	5.3	0.81	24.23	8.27	1.53	25.33	9.33
17*	47	AR	1.50	0.29	2.6	3.1	5.6	0.84	24.89	1.60	2.84	30.40	5.87
18*	52	"	1.43	0.25	2.6	3.4	5.3	0.76	22.94	7.60	1.42	26.67	6.67
19	19	"	1.28	0.26	2.2	4.0	6.0	0.55	16.50	1.87	3.16	21.33	4.00
20	18	"	1.40	0.30	1.9	3.3	5.7	0.58	17.27	2.67	2.40	22.40	4.27
21*	57	ar, MS	1.64	0.26	2.1	2.4	4.2	0.88	26.25	6.93	0.78	24.67	5.33
22*	35	"	1.27	0.36	2.2	1.9	3.5	1.16	34.74	20.80	0.21	27.33	8.67
23*	35	AR, MVD	1.16	0.30	1.9	2.8	4.3	0.68	20.36	3.73	0.83	20.67	4.67

* Recommended for aortic valve surgery. Cases 1 to 6 echocardiography simultaneous with catheterization. ar, mild aortic regurgitation; AR, moderate aortic regurgitation; SD, subvalvar diaphragm; MS, mitral stenosis; MVD, mixed mitral valve disease.

Conversion factor from SI units to Traditional units: 1 kPa $\approx$ 7.5 mmHg.

TABLE 4 Patients with poor left ventricular function

Patient	Age (yr)	Diagnosis	P (kPa)	V _{cf} (cf/s)	W _s (cm)	D _s (cm)	W _s /D _s	c
1	42	AR	20.00	0.92	2.0	5.0	0.40	50.0
2	54	"	21.33	0.84	2.3	4.6	0.50	42.7
3	51	"	16.67	0.98	2.5	5.3	0.47	35.3
4	42	"	16.67	0.68	2.2	7.3	0.30	55.3
5	53	"	20.00	0.52	2.1	6.8	0.31	64.8
6	72	"	20.00	0.62	2.2	5.2	0.42	47.3
7	58	AS	23.73	0.46	1.9	7.1	0.27	88.7
8	59	"	26.67	0.73	2.2	4.6	0.48	55.8
9	57	"	37.33	0.66	1.5	4.3	0.35	107.0
10	23	AR, VSD	17.33	0.93	2.5	5.9	0.42	40.9

AR = aortic regurgitation, AS = aortic stenosis, VSD = ventricular septal defect.
Conversion factor from SI units to Traditional units: 1 kPa $\approx$ 7.5 mmHg.

and the mean (30.9 ± 2.7) was similar to that found in the normal subjects.

The mean value for c derived from the normal subjects together with the patients without aortic stenosis having good left ventricular function was 30.

Data from the cases of aortic stenosis with good ventricular function are given in Table 3. There was good correlation between echocardiographic estimates and direct measurements of aortic valve gradient ($r = 0.87$, $P < 0.001$).

The calculated values for aortic valve orifice areas ranged from 0.21 to 3.16 cm². Those patients with severe stenosis with or without mild regurgitation who, on the basis of symptoms and catheterization data, were recommended for aortic valve surgery all had aortic valve areas less than 0.8 cm².

Data from the cases with poor left ventricular function are given in Table 4. Values for c were higher than those with good left ventricular function (mean 59.8, SD $\pm$ 22.7). There was poor correlation between relative wall thickness and peak systolic intraventricular pressure ($r = -0.11$, $P > 0.5$).

There was close correlation between relative wall thickness and peak systolic intraventricular pressure in all 53 cases with good left ventricular function ($r = 0.91$, $P < 0.001$).

Discussion

The results of this study confirm the angiocardigraphic findings that, in the presence of good ventricular function, both wall thickness and cavity size are increased in volume overload so that relative wall thickness remains normal, whereas wall thickness is increased without cavity enlargement in pressure overload and hence relative wall thickness is increased. In this study it was found possible to obtain useful estimates of intraventricular pressure

from relative wall thickness measurements and thus to estimate aortic valve gradients. Though in a few cases the gradient was substantially overestimated, this method offers a noninvasive means of estimating valve gradients, which appears to be sufficiently accurate to be of value for clinical purposes. This method is suitable for cases with coexistent mitral valve disease or aortic regurgitation. The method is not suitable for cases with poor ventricular function or hypertrophic cardiomyopathy where the degree of ventricular hypertrophy is inappropriate to the load. Angiocardigraphic measurements of V_{cf} have been used to assess ventricular performance and have shown good correlation with other indices of ventricular function (Karliner *et al.*, 1971). Close correlation between angiocardigraphic and echocardiographic measurements of V_{cf} has been demonstrated in several studies, and the range of normal values for V_{cf} found in this study is very similar to that found in those studies (Cooper *et al.*, 1972; Ludbrook *et al.*, 1973; Quiriones, Gaasch, and Alexander, 1974). Measurements of V_{cf} were used in this study to identify those cases where, due to poor ventricular performance, cavity dilatation was likely to be out of proportion to the volume load. It is not always possible, particularly in the older patient, to obtain echocardiograms of sufficient quality for unequivocal identification of the endocardium and epicardium necessary for relative wall thickness measurement. When working in mmHg rather than SI units the appropriate value of c to be substituted in the formula is 225.

Bache *et al.* (1972) demonstrated a very close correlation ($r = 0.98$) in 32 cases of aortic stenosis between mean and peak systolic aortic valve gradients and thus were able to modify Gorlin and Gorlin's formula so that aortic valve orifice areas could be calculated from peak rather than mean gradients.

Calculation of aortic valve areas using echocardiographic estimates of peak systolic gradient, stroke volume, and ejection time, in spite of the limitations of these estimates, gave meaningful results. All patients with pure stenosis and those with additional mild aortic regurgitation who were recommended for aortic valve surgery had calculated valve areas in the 'critical range' (Wood, 1950; Gorlin *et al.*, 1955). Unfortunately, accurate measurements of total stroke volume were not made at catheterization and so it was not possible to compare the echocardiographic values for valve areas with those measured at catheterization.

Wall stress, a measure of left ventricular wall tension per unit cross-sectional area of ventricular wall, is proportional to intraventricular pressure and cavity radius and inversely proportional to wall thickness. It has been suggested that the degree of ventricular hypertrophy is determined by wall stress, wall thickness increasing in proportion to left ventricular load until systolic wall stress returns to normal (Linzbach, 1960; Hood *et al.*, 1968). The simplest formula for wall stress is:

$$\text{mean circumferential wall stress} = \frac{\text{pressure} \times \text{cavity radius}}{\text{wall thickness}}$$

Hermann, Singh, and Dammann (1969) have shown that the use of this simple formula correlates closely with the more complex formula, based on a thin walled ellipsoidal model, of Sandler and Dodge (1963), which in turn has been shown to correlate closely with Wong and Rautarharju's (1968) highly complex, theoretically more correct, formula based on a thick walled ellipsoidal model (Hood *et al.*, 1969). Though in this study measurements of ventricular dimensions and pressure were not made at the same point in the cardiac cycle, the formula used to calculate *c* is similar to the simple formula for wall stress and *c* could be regarded as an index of systolic wall stress; both *c* and systolic wall stress remain normal in hypertrophied ventricles with good ventricular function and are raised in those with poor function (Bennett, Evans, and Raj, 1975).

The severity of aortic stenosis is not always apparent from clinical examination, particularly when there is coexistent systemic hypertension or other valvular defects. Echocardiography offers a non-invasive method of assessment of the severity. Where the severity is clinically obvious echocardiography can be used to document the severity of the lesion and to assess ventricular function. At the same time the characteristic features of hypertrophic cardiomyopathy (Popp and Harrison, 1969; Henry, Clarke, and Epstein, 1973) which can mimic aortic stenosis, and mitral stenosis (Edler, 1956) will, if present, be detected.

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