Distribution of Left Ventricular Hypertrophy in Hypertrophic Cardiomyopathy: a Two-Dimensional Echocardiographic Study

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The distribution of left ventricular hypertrophy was assessed by M-mode and two-dimensional echocardiography in 89 patients with hypertrophic cardiomyopathy. Myocardial thickness was measured in the septum and the free and posterior wall in both the proximal and distal left ventricle. All patients had at least one myocardial region that was hypertrophied. The predominant pattern of hypertrophy was defined as symmetric (31%), asymmetric septal (55%) and distal ventricular (14%). The spectrum of wall thickness measurements between patients with symmetric hypertrophy was wide (1.5 to 4.5 cm) and was not related to age. In patients with asymmetric septal hypertrophy, the distribution of hypertrophy conformed to previously described patterns; hypertrophy was localized to the anterior septum (14%) or the anterior and posterior septum (35%) or involved both the septum and the left ventricular free wall (51%). The patients with distal ventricular hypertrophy had marked papillary muscle thickening, and only 1 of 12 patients could be correctly diagnosed using M-mode echocardiography.

The proportion of patients with symmetric and distal ventricular hypertrophy was greater than that reported when patients are selected on the basis of M-mode diagnostic criteria. This reflects the limitations of the M-mode technique in the assessment of left ventricular hypertrophy and suggests that the recognition and understanding of hypertrophic cardiomyopathy have been biased by patients with asymmetric septal hypertrophy who previously were most readily identified.

Hypertrophic cardiomyopathy is defined as a heart muscle disorder of unknown origin that is characterized by unexplained hypertrophy of a nondilated left ventricle (1). During the past decade, the most widely applied diagnostic criteria were derived from the M-mode echocardiogram. These criteria emphasized the demonstration of asymmetric hypertrophy between the upper anterior septum and the left ventricular posterior wall as well as features associated with left ventricular pressure gradients, such as systolic anterior motion of the mitral valve and mid-systolic closure of the aortic valve. Recent studies, however, have shown that myocardial regions that are not visualized by the M-mode beam may be hypertrophied in the absence of asymmetric septal hypertrophy (2,3). Thus, using the M-mode technique, the diagnosis and pattern of myocardial hypertrophy may be undetected. Two-dimensional echocardiography permits evaluation of the entire left ventricle (4–6). The purpose of this study was to use two-dimensional echocardiography to assess the regional distribution of left ventricular hypertrophy in patients with hypertrophic cardiomyopathy.

Methods

Study Patients

One hundred five patients with hypertrophic cardiomyopathy were studied by two-dimensional echocardiography between November 1981 and July 1982. Of these 105 patients, 89 had adequate recordings. Of the 89, 46 were male and 43 were female with an age range of 9 to 70 years (mean 39) at the time of study. Seventy-nine of those studied were consecutive patients who had been followed up at the Hammersmith Hospital for 1 to 20 years (mean 6). The diagnosis of hypertrophic cardiomyopathy was made in the remaining 10 patients during the period of study. In all patients, this was based on clinical (7) and angiographic (8) or echocardiographic (9) demonstration of unexplained left ventricular hypertrophy. None of these patients had documented high blood pressure, abnormal renal function or ocular changes of systemic hypertension.

Cardiac catheterization was performed in 71 patients and 50 had a left ventricular pressure gradient of 20 mm Hg or greater measured at rest or on provocation (first with iso-prenaline infusion and later with amyl nitrate inhalation and the Valsalva maneuver). Of the patients who did not undergo catheterization, 12 had clinical and M-mode echocardio-graphic features of hypertrophy and a left ventricular pres-sure gradient (9) and 6 had unexplained electrocardiographic and M-mode echocardiographic evidence of left ventricular hypertrophy and a family history of hypertrophic cardio-myopathy.

Control subjects. Twenty-five normal volunteers (mean age 43 years, range 18 to 61; 16 male and 9 female) under-went similar echocardiographic study.

Echocardiography

Apparatus. Each patient was studied with a combina-tion of M-mode and wide angle (90°) two-dimensional echo-cardiography at 8, 12, 16 and 24 cm depths using a 2.5 or 3.5 MHz transducer with a Hewlett-Packard phased array (model 77020A) or an ATL mechanical sector (MI) ultra-sound scanner. Images were stored on a Sony Betamax video recorder (model 323). Single frozen still frames were pho-tographed directly.

Techniques. A complete M-mode and two-dimensional study was performed and recorded on video tape and in-cluded parasternal long-axis and multiple short-axis views of the left ventricle with the transducer positioned at the fourth intercostal space. Great care was taken to make an accurate short-axis left ventricular sweep from aortic root to apex ensuring an approximately circular cavity through-out; this occasionally required inferolateral movement of the transducer. M-mode recordings of the septum, posterior wall, cavity size and mitral and aortic valves were obtained by identifying the relevant structure in a short-axis para-sternal view with an M-mode cursor displaying this portion of the sector. Two- and four-chamber views were obtained with the transducer at the apex.

Echocardiographic interpretation. The M-mode re-cordings were studied for the presence of mid-systolic clo-sure of the aortic valve, systolic anterior motion of the mitral valve (graded as severe if there was systolic apposition of the mitral valve and septum, mild if there was no apposition, or absent) and asymmetric septal hypertrophy (upper an-terior septal to posterior wall ratio of 1.5:1 or greater). Asymmetric septal hypertrophy with a ratio of 1.3:1 or greater was also considered. Of the classic echocardio-graphic features of hypertrophic cardiomyopathy (9), these, as well as wall thickness measurements, were assessed. Systolic and diastolic cavity dimensions (cm) were mea-sured at minimal cavity size and at the onset of the R wave of the electrocardiogram, respectively.

From the two-dimensional echocardiogram, two short-axis scans were used for analysis, one in the upper left

ventricle at the level of the mitral valve tips (the portion usually sampled by the M-mode echocardiographic beam) and the second toward the apex at a level below the papillary muscles (Fig. 1). Measurements were made at end-diastole (maximal cavity size) after careful localization of epicar-dium and endocardium using normal and slow forward play-back. Wall thickness was measured at the quadrants (Fig. 1) and the thickness of the apical myocardium was measured in the four chamber apical view. The papillary muscles were assessed by studying all views and then grading their hy-pertrophy as absent, mild (larger than normal but not elim-inating the lower cavity) or severe (producing lower cavity elimination).

For the purpose of analysis, this sample was divided into three groups: 1) patients with asymmetric septal hypertro-phy, that is those with a ratio of anterior or posterior septum to left ventricular posterior wall of 1.5:1 or greater in the upper or lower ventricle; 2) patients with predominantly distal ventricular hypertrophy, defined as a ratio of 1.5:1 or greater in at least two of four measurements comparing lower and upper ventricular wall thickness; and 3) patients with symmetric hypertrophy, that is, those with neither of the above, in whom the coefficient of variation for the mea-surements of left ventricular myocardial thickness was less than 20%.

Reliability and reproducibility of the measurements. Reliability was assessed by reanalyzing the original echo-cardiographic recordings of 20 patients without knowledge of the original measurements or the frames from which they

Figure 1. Diagrammatic representation of two echographic left ventricular cross sections. Quadrant measurements are shown by **arrows.** Ao = aorta; AVS and PVS = anterior and posterior ventricular septum, respectively; LA = left atrium; LV = left ventricle; PW and FW = posterior and free wall, respectively.
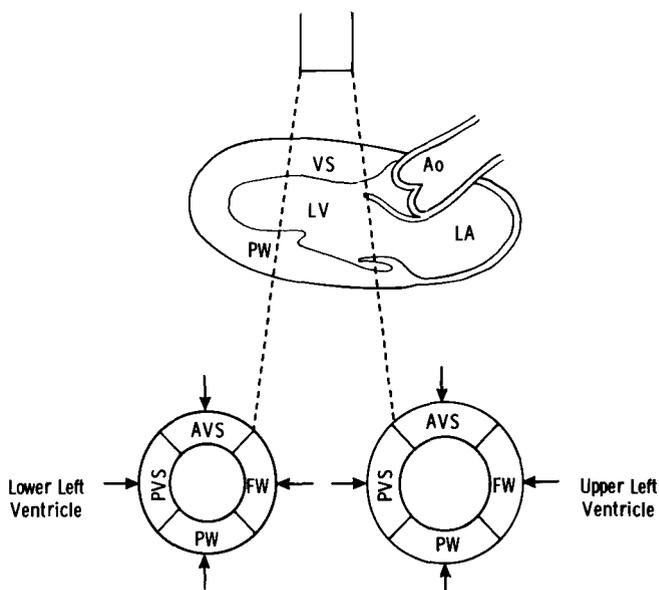
Table 1. Reliability and Reproducibility of Echocardiographic Measurements: Retest Reliability Coefficients

Echocardiographic Measurement	Echocardiogram	
	Same	Repeat
M-mode		
Septum	0 96	0.95
Posterior wall	0.96	0.94
Diastolic dimension	0.95	0.94
Systolic dimension	0.95	0.93
SAM	0.99	0 92
Mid-systolic closure of aortic valve	0.98	0.91
Two-dimensional		
Upper left ventricle		
Anterior ventricular septum	0.95	0.93
Posterior ventricular septum	0.92	0.90
Free wall	0.92	0 90
Posterior wall	0 94	0.91
Lower left ventricle		
Anterior ventricular septum	0.93	0.92
Posterior ventricular septum	0.86	0.81
Free wall	0.90	0 89
Posterior wall	0.92	0.90
Apical myocardium	0.73	0 67

SAM = systolic anterior motion of the mitral valve.

were made. Reproducibility was determined in 20 unselected patients by comparing the original measurements with those made from a repeat echocardiographic study that was performed 1 to 6 months later.

Statistical analysis. Values are quoted as the mean ± 1 standard deviation. The scatter of values was expressed as the coefficient of variation, that is (standard deviation − mean) × 100. The Student's t or chi-square test was used

for analyzing the differences between variables. For the assessment of reliability and reproducibility, a retest reliability coefficient was calculated; this assumed an ideal value of unity.

Results

Reliability and reproducibility (Table 1). The reliability and reproducibility of the M-mode and two-dimensional measurements made perpendicular to the ultrasound beam were excellent. Two-dimensional measurements made lateral to the ultrasound beam were less so; apical thickness measurements were, therefore, excluded from the subsequent analysis.

Distribution of Left Ventricular Hypertrophy (Tables 2 and 3)

All patients (Fig. 2 to 5). In all patients, at least one wall thickness measurement exceeded 2 standard deviations from the normal (greater than 1.4 cm). Such minimal left ventricular hypertrophy was present in four patients and exceeded this value in the others. The mean ratio of upper anterior septum to posterior wall thickness was 1.5 ± 0.5. In comparison with normal subjects, the mean wall thicknesses for all left ventricular regions were greater (probability [p] < 0.001) and left ventricular cavity dimensions were decreased (p < 0.001).

The thickest region was the upper anterior septum in 32 patients (36%), the lower anterior septum in 18 (20%), the upper posterior septum in 12 (14%), the lower posterior septum in 8 (9%) and the upper or lower free wall in 19 (21%). In 35 patients (40%), the lower anterior septum was

Table 2. Left Ventricular Dimension and Wall Thickness Measurements

	Patients With Hypertrophic Cardiomyopathy				
	Total Group (n = 89)	Asymmetric Septal Hypertrophy (n = 49)	Distal Ventricular Hypertrophy (n = 12)	Symmetric Hypertrophy (n = 28)	Normal Subjects (n = 25)
Upper left ventricle					
Ratio VS/posterior wall	1.5 ± 0.5*	1 9 ± 0.4*	1.2 ± 0 2	1.2 ± 0 2	1.1 ± 0.2
Anterior VS	1.8 ± 0.5*	1.9 ± 0.5*	1 4 ± 0.4†	1.6 ± 0.6*	0.9 ± 0.2
Posterior VS	1.7 ± 0.5*	1.7 ± 0 4	1.5 ± 0.5*	1.6 ± 0.5*	0.8 ± 0 3
Free wall	1.5 ± 0.4*	1.5 ± 0.4*	1.3 ± 0.5†	1.6 ± 0.5*	0.8 ± 0.3
Posterior wall	1.2 ± 0.5*	1.1 ± 0.2	1.2 ± 0 4†	1 5 ± 0.7*	0.8 ± 0.2
Lower left ventricle					
Anterior VS	1.7 ± 0 6*	1 7 ± 0.6*	2.3 ± 0.8*	1 6 ± 0.6*	0.9 ± 0.2
Posterior VS	1.6 ± 0.5*	1.5 ± 0.3*	2.2 ± 0.8*	1.5 ± 0 5*	1.0 ± 0 2
Free wall	1 5 ± 0.6*	1.3 ± 0.3*	2 2 ± 0.7*	1.5 ± 0.6*	1 0 ± 0.1
Posterior wall	1.5 ± 0.6*	1.3 ± 0.5*	1.9 ± 0.6*	1 6 ± 0.6*	1.0 ± 0.2
LV diastolic dimension	4.1 ± 0.8*	4.1 ± 0.7†	4.0 ± 0.8‡	4.1 ± 0 9§	4.5 ± 0.3
LV systolic dimension	2.6 ± 0.7‡	2.6 ± 0.6‡	2.7 ± 0.9	2.5 ± 0.8§	2.9 ± 0 5

*probability (p) < 0.001; †p < 0.005; ‡p < 0.01; §p < 0.05 (differences from normal). All measurements are mean ± 1 standard deviation (cm). LV = left ventricular; VS = ventricular septum

Table 3. Number of Patients With Systolic Anterior Motion of the Mitral Valve, Mid-Systolic Closure of the Aortic Valve and Papillary Muscle Hypertrophy in Relation to the Pattern of Left Ventricular Hypertrophy

	Patients With Hypertrophic Cardiomyopathy			
	Total Group (n = 89)	Asymmetric Septal Hypertrophy (n = 49)	Distal Ventricular Hypertrophy (n = 12)	Symmetric Hypertrophy (n = 28)
Systolic anterior motion of the mitral valve (n = 42)				
Severe/mild/absent	27/15/47	16/12/21	2/1/9	9/2/17
Mid-systolic closure of the aortic valve (n = 41 of 69)				
Present/absent	41/28	23/14	4/7	14/7
Papillary muscle hypertrophy (n = 85)				
Severe/mild/absent	58/27/4	31/16/2	9/2/1	18/9/1

thicker than the upper anterior septum. The lower free wall was thicker than the upper in 42 patients (47%) and the thickness of the upper or lower free wall exceeded that in the upper anterior septum in 28 patients (31%).

Systolic anterior motion of the mitral valve was detected in 42 patients (47%) and mid-systolic closure of the aortic valve was observed in 41 (59%) of 69 patients. Of the classic M-mode features that were assessed, 21 patients (24%) had all three features, 42 patients (47%) had two and 69 patients (78%) had only one feature.

Asymmetric septal hypertrophy (Fig. 2). Forty-nine patients who had a ratio of septum to posterior wall thickness of 1.5:1 or greater in the upper or lower left ventricle were designated as having asymmetric septal hypertrophy. Within this group, asymmetric septal hypertrophy was present in all septal regions in 2 patients, three septal regions in 6, the upper anterior and posterior septum in 17, the upper and

lower anterior septum in 5 patients and in a single septal region in 19 patients. Forty (82%) of the 49 patients had asymmetric septal hypertrophy of the upper anterior septum; in 22 patients (45%) this was the thickest myocardial region. This region was thicker than the upper posterior septum in 31 patients (63%) and the lower anterior and posterior septum in 25 patients (51%). Hypertrophy was confined to the anterior septum in seven patients (14%) and to the upper anterior septum alone in five patients. In 17 patients (35%), hypertrophy was present only in the anterior and posterior septum (three or four septal regions in 8 patients and two septal regions in 9).

Twenty-five patients (51%) had hypertrophy of both the septum and the free wall. In these 25 patients, the thickest region was the septum in 16 patients and the free wall in 4; in 5 patients, the septum and free wall were of approximately equal thickness. Hypertrophy was present in three or four septal regions in 17 patients, in two regions in 6 patients and in one region in 2 patients.

Left ventricular cavity dimensions were similar to those of the complete group, but significantly different from those of the normal group. Systolic anterior motion of the mitral valve was present in 28 patients (57%) and mid-systolic closure of the aortic valve was noted in 23 (62%) of 37 patients.

Distal ventricular hypertrophy (Fig. 3). A ratio of 1.5:1 or greater comparing myocardial thickness in the lower and upper left ventricle was present in the anterior septum in 13 patients (15%), the posterior septum in 4 (5%), the left ventricular posterior wall in 22 (25%) and the free wall in 7 patients (8%). This ratio was present in two of these areas in 12 patients and they were designated as having distal ventricular hypertrophy. If a ratio of 1.3:1 or greater had been used, seven additional patients would have been included within this group. Hypertrophy was absent or mild in the upper left ventricle; however, measurements of wall thickness in the lower cavity were grossly increased and 11

Figure 2. Parasternal long-axis view of a patient with asymmetric septal hypertrophy, showing disproportionate thickening of the upper septum.
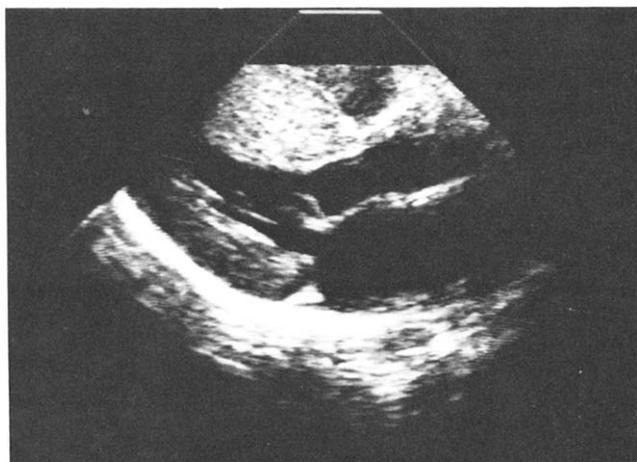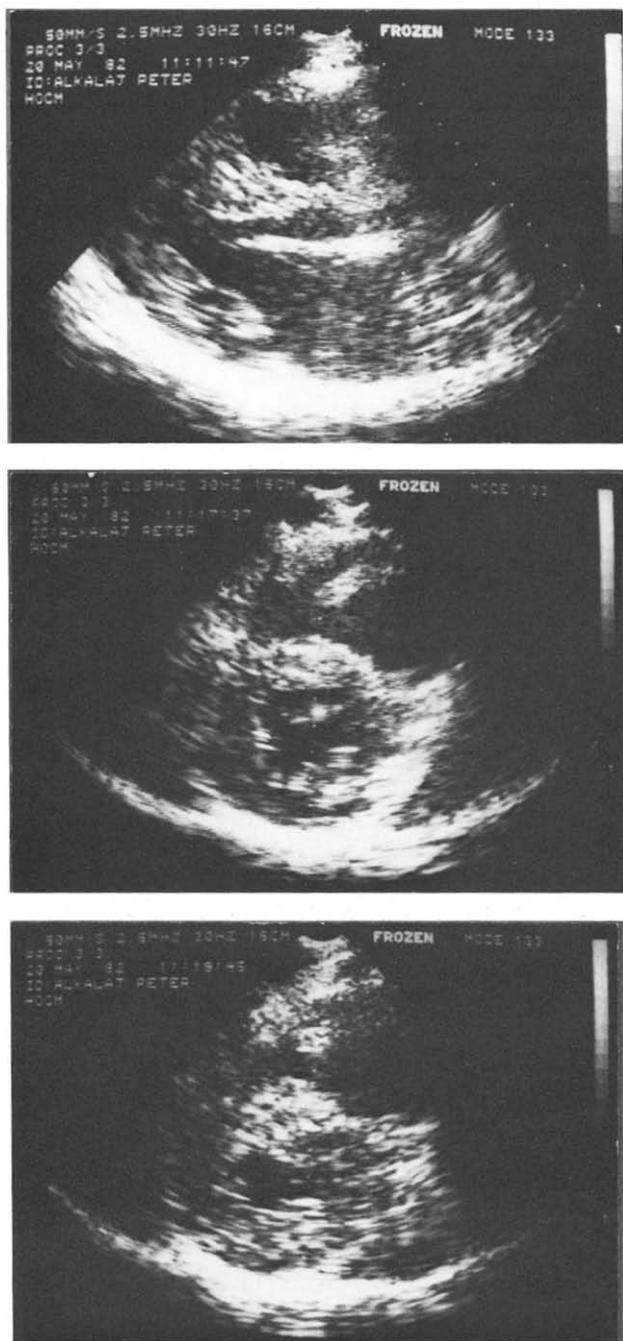
Figure 3. Parasternal long-axis (**top**) and short-axis (**middle and bottom**) view of a patient with distal ventricular hypertrophy. The upper ventricular wall thickness in the portion sampled by the M-mode beams is relatively normal (**top and middle**), but there is gross distal ventricular hypertrophy (**bottom**).

of the 12 patients had hypertrophy of the papillary muscles that resulted in lower cavity elimination.

Six of the patients with distal ventricular hypertrophy also had asymmetric septal hypertrophy, which was confined to the lower anterior or posterior septum in three pa-

tients and to the upper posterior septum in two patients. In the remaining patient, asymmetric septal hypertrophy was present in all four septal regions, but the predominant pattern of hypertrophy was distal ventricular because the ratio of lower to upper wall thickness was 1.5:1 for both anterior and posterior septum and 1.7:1 for the left ventricular posterior wall and free wall. Systolic anterior motion of the mitral valve was seen in 3 patients (35%) and mid-systolic closure of the aortic valve in 4 (36%) of 11 patients; all of these patients also had asymmetric septal hypertrophy.

Symmetric hypertrophy (Fig. 4 and 5). Twenty-eight patients who did not have asymmetric septal or distal ventricular hypertrophy and who had a coefficient of variation for the measurements of myocardial thickness of less than 20% fulfilled the criteria for symmetric hypertrophy. If the criteria of asymmetry had been a ratio of 1.3:1 rather than 1.5:1, 11 of these patients would not have been included in this group. In patients with symmetric hypertrophy, measurements of wall thickness were significantly thicker (p < 0.001) over a wide range (1.4 to 4.5 cm). The degree of hypertrophy was not related to age. The variation in the measurements of wall thickness within individual patients, was similar in those with symmetric hypertrophy and in the normal subjects, but was significantly less than in patients with asymmetric septal and distal ventricular hypertrophy (p < 0.001) (Fig. 6).

Left ventricular cavity dimensions were similar to those of the other groups. Systolic anterior motion of the mitral valve was present in 11 (39%) of the 28 patients and mid-systolic closure of the aortic valve in 14 (66%) of 21 patients. Nine (32%) of the 28 patients had two of the classic M-mode features and 16 (57%) had one of the features.

Discussion

Asymmetric septal hypertrophy. All of the 89 patients studied had at least one myocardial region that was 1.5 cm or more in thickness. The original descriptions of hypertrophic cardiomyopathy highlighted the feature of hypertrophy as predominantly affecting the upper septum (10). Whereas earlier M-mode studies (11,12) had confirmed asymmetric septal hypertrophy, more recent work (2,3,6) has shown that the upper septum may not, in fact, be disproportionately thickened or even hypertrophied. In 49 (55%) of our 89 patients, the predominant pattern of hypertrophy was asymmetric septal hypertrophy. The distribution of hypertrophy among these patients was similar to that reported by Maron et al. (6). Hypertrophy was confined to the anterior and posterior septum in 35%, to the anterior septum in 14% and involved both septum and free wall in 51%. Although these 49 patients were identified as having the "classic" form of the disease, 27 (55%) had a region of the left ventricle that was thicker than the upper anterior
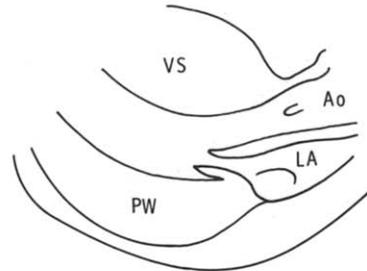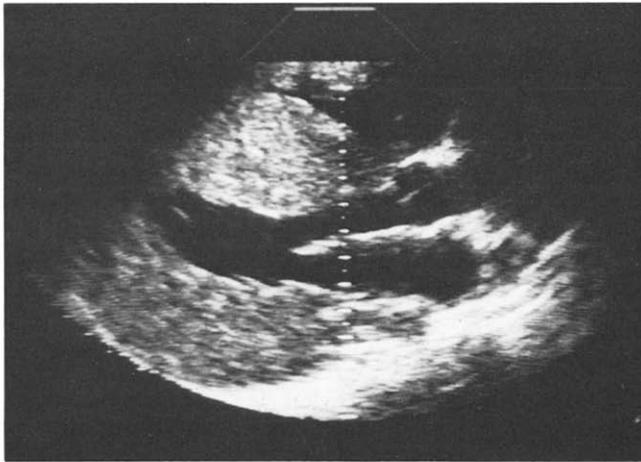
Figure 4. Parasternal long-axis echocardiographic (**left**) and diagrammatic representation (**right**) of a patient with symmetric ventricular hypertrophy, showing a small cavity and similarly increased wall thickness in the septum and posterior wall. Abbreviations as in Figure 1.

septum and 9 (18%) did not have asymmetric hypertrophy of the upper anterior septum.

Distal ventricular hypertrophy. Apical hypertrophic cardiomyopathy is thought to be rare in Western populations (3,13). In our study, measurements of myocardial thickness at the apex of the left ventricle were not reliable or reproducible. Twelve (14%) of 89 patients, however, had left ventricular hypertrophy that was predominantly in the distal ventricle. From a two-dimensional echocardiographic evaluation, "distal ventricular" is a more appropriate term for the findings in these patients. They had minimal hypertrophy in the proximal ventricle, but in the distal ventricle hyper-

Figure 5. Short-axis mitral (**left**) and subpapillary (**right**) echocardiograms in the same patient as in Figure 4, demonstrating marked symmetric hypertrophy of the septum and posterior wall.

trophy was massive and the cavity was eliminated by huge papillary muscles. M-mode echocardiography would not have identified these patients, and the apparent rarity of this form may reflect patient selection in Western population studies (3,13).

Symmetric ventricular hypertrophy. In 28 patients (31%), the left ventricular hypertrophy was symmetric. The coefficient of variation for the measurements of wall thickness was similar among the normal subjects and those with symmetric hypertrophy. These patients exhibited a wide spectrum of wall thickening, from those with 3.5 to 4.5 cm thickness in all regions with severe cavity elimination to those with thickness of only 1.5 cm and near normal cavity dimensions. We do not know whether this different expression of the condition reflects variable sensitivity to otherwise minor stimuli for the development of hypertrophy (for example, physiologic increases in afterload) or whether it is due to some other factor.

Definition of asymmetric septal hypertrophy. In a previous analysis (6) of hypertrophy in hypertrophic cardiomyopathy, 78% of patients had asymmetric septal hypertrophy with a septal to posterior wall ratio of 1.3:1 or greater.
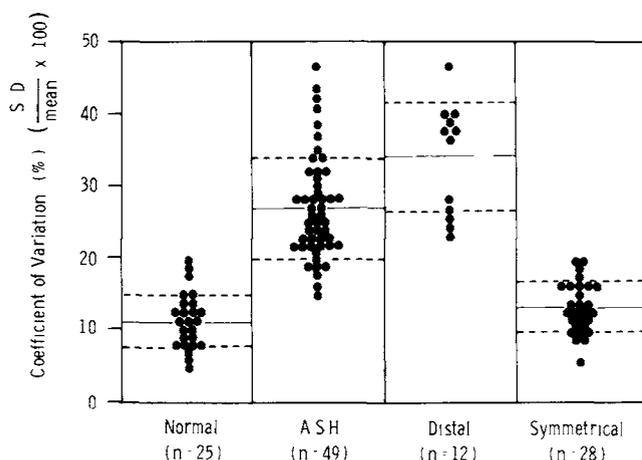
Figure 6. Coefficient of variation for measurements of left ventricular wall thickness in 89 patients with hypertrophic cardiomyopathy and 25 normal subjects. In patients with symmetric hypertrophy and in the normal subjects, the coefficient of variation was less than 20%. In those with asymmetric septal hypertrophy (ASH), the coefficient of variation was significantly greater (p < 0.001) and was less than 20% in only 6 (12%) of 49 patients. Of these, five had mild to moderate hypertrophy (15 to 18 mm) localized to the upper anterior septum. The patients with distal ventricular hypertrophy had minimal or no hypertrophy in the upper ventricle. The pattern of hypertrophy in the lower ventricle was asymmetric septal in six and symmetric in six; this distribution is accurately reflected by the separation of the coefficient of variation in these patients.

This degree of asymmetry, however, has been reported in up to 44% of "normal" control subjects (9), in 42% of professional athletes (14) and in 30 to 70% of patients with secondary left ventricular hypertrophy (15–18). A ratio of 1.5:1 or greater is more likely to identify disproportionate myocardial thickening. The discrepancy between the observations of Maron et al. (6) and our data in the proportion of patients with asymmetric septal hypertrophy and symmetric hypertrophy is, in part (10 to 15%), due to a different definition of asymmetry. The remaining difference (approximately 15%) may be due to patient selection.

At our institution before two-dimensional echocardiography was available, the diagnosis of hypertrophic cardiomyopathy was based on the characteristic clinical and angiographic features (7,8,19); M-mode echocardiography was the primary diagnostic tool for only 4 of 289 patients. Although Maron et al. (6) utilized both M-mode and two-dimensional echocardiographic criteria, it seems probable that their patient selection was influenced by their previous M-mode experience, and their findings, therefore, favor upper septal hypertrophy because of the limitations of the M-mode technique when assessing the free wall, the posterior septum and distal regions of the left ventricle.

Papillary muscle hypertrophy. Although it is well recognized that papillary muscle hypertrophy is common in hypertrophic cardiomyopathy, there is no standardized method

of quantitation. Acknowledging the limitations of the method used, which will overestimate papillary muscle size when the end-systolic cavity is small, only 3 patients were normal and in 58 patients (65%) there was sufficient hypertrophy to give the appearance of distal cavity elimination. Papillary muscle hypertrophy was present in all the subgroups and it seems that this is a consistent finding in hypertrophic cardiomyopathy.

Reliability of two-dimensional echocardiography. At present, two-dimensional echocardiography is the best method for studying regional hypertrophy. Although most of the heart may be examined using the different views, problems remain with image resolution and the tendency to overestimate wall thickness and underestimate cavity size. Measurements made at right angles to the ultrasound beam have excellent reproducibility, but those requiring lateral resolution (even if centered in the middle of the image) are less reliable. It may also be difficult to identify endocardium and epicardium, as is indicated by the unacceptable reliability of measurements at the apex.

Clinical implications. Left ventricular hypertrophy in hypertrophic cardiomyopathy can thus broadly be characterized as having one of three distinct patterns. The criteria to define these patterns are arbitrary and do not represent a discontinuity of myocardial wall thickness ratios between groups. It is uncertain whether these patterns identify "subgroups" of patients with hypertrophic cardiomyopathy of different origin. Our data show marked differences in the degree and the distribution of hypertrophy within the groups, as well as a continuity of myocardial wall thickness ratios between the groups. It is important for the diagnosis of hypertrophic cardiomyopathy to recognize that the degree and distribution of hypertrophy are variable and that symmetric and distal ventricular hypertrophy are common and not always amenable to M-mode echocardiographic diagnosis. It remains to be determined if identifiable patterns of hypertrophy are of clinical or prognostic importance.

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