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Hypertrophic Cardiomyopathy: Two-Dimensional Echocardiographic Score Versus Clinical and Electrocardiographic Findings

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Summary: The severity and site of hypertrophy is important in determining the clinical picture and the natural history of hypertrophic cardiomyopathy (HCM). We evaluated left ventricular hypertrophy by means of two-dimensional echocardiographic score and score index, and correlated these findings with symptoms, electrovectorcardiographic data, and ventricular arrhythmias. A total of 42 patients with HCM were studied by clinical examination, ECG, VCG, M-mode and 2D echocardiography, and 24-h Holter monitoring. The extent and severity of the hypertrophic process were calculated by a score system. The left ventricle was divided into 11 segments and a hypertrophic score (HS) was given to each segment. A hypertrophy score index (HSI) was also calculated by dividing the number of hypertrophied segments by 13. No correlation was found between symptoms and HS and HSI, nor ECG-VCG abnormalities and HS and HSI. A statistically significant relationship between the severity of ventricular arrhythmias and HS and HSI was found ($p<0.01$). The mechanism responsible for ventricular tachyarrhythmias in severe and diffuse hypertrophy might reside in the high intraventricular pressures which produce or worsen areas of myocardial ischemia.

Key words: hypertrophic cardiomyopathy, ventricular arrhythmias

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Introduction

Hypertrophic cardiomyopathy (HCM) is a primary disease characterized by symmetric or asymmetric hypertrophy of the left and/or right ventricle;[1] the site and extent of this hypertrophic process determine the clinical picture and the natural history of the disease.[2]

The purpose of this study was to evaluate myocardial hypertrophy by means of a two-dimensional echocardiographic score and score index, and correlate these with symptoms, electrovectorcardiographic data, and ventricular arrhythmias.

Material and Methods

Between February 1984 and October 1987, 70 patients diagnosed or suspected clinically of having HCM were studied by two-dimensional (2D) echocardiography.

Adequate recordings were obtained in 42 patients, who form the basis of this study. This series consisted of 20 males and 22 females, ranging in age from 16 to 65 years (mean, 39 years). HCM diagnosis was based on M-mode[3] and two-dimensional echocardiograms showing a nondilated, hypertrophied left ventricle in the absence of other cardiac or systemic diseases capable of producing left ventricular hypertrophy. All the patients underwent physical examination, electrocardiogram (ECG), vectorcardiogram (VCG), and 24-h Holter monitoring; none were taking cardiac drugs at the time of this study, and all examinations were carried out within a short time period. Twenty-four patients underwent cardiac catheterization. Hemodynamic and angiographic studies included left and right ventricular angiography and coronary arteriography. Left ventricular outflow obstruction was considered present if the peak systolic outflow gradient under basal conditions was 30 mmHg or more. VCGs were obtained in the supine position with either a Fukuda or an ICR vectorcardiograph, utilizing the Frank lead system; dash time interval was 2 ms.

Echocardiography

M-mode echocardiograms were obtained with an Irex System III using a 2.5 MHz transducer. Chamber size, wall thickness, and fractional shortening were analyzed according to standard procedures.[4]

Two-dimensional echocardiograms were recorded on Irex System III and HP 77020 AV, with 2.5 MHz or 3.5 MHz phased-array transducers.

Standard parasternal, apical, and subcostal views were stored on Sony Betamax and Panasonic AG 6200 video tapes, which enabled frame by frame and real-time playback for detailed evaluation of structures and function. Single frozen still frames were printed directly in end-diastole. Two observers evaluated the 2D echocardiographic images in a double-blind study. Distribution of left ventricular hypertrophy was appraised by classifying the patients into four types, according to Maron *et al.*[5] The extent of hypertrophy was calculated by a score system, which was a modification of Edwards' method of segmental analysis.[6] The left ventricle was divided into 11 segments and each was assigned a hypertrophy score (HS); a final score was then calculated for each patient (Fig. 1). A segment with a thickness <12 was scored "0"; a thickness $\geq 12 \leq 17$ was scored "1"; >17 and ≤ 22 was scored "2" and if >22 mm a score "3" was given.[7] The hypertrophy score index (HSI) was then calculated by dividing the number of the hypertrophied segments by 13; the apical region included three segments.[7] In agreement with the guidelines proposed by Maron *et al.*,[5] segments were considered hypertrophied only if they were at least 17 mm thick to avoid overestimation due to the limited lateral resolution.

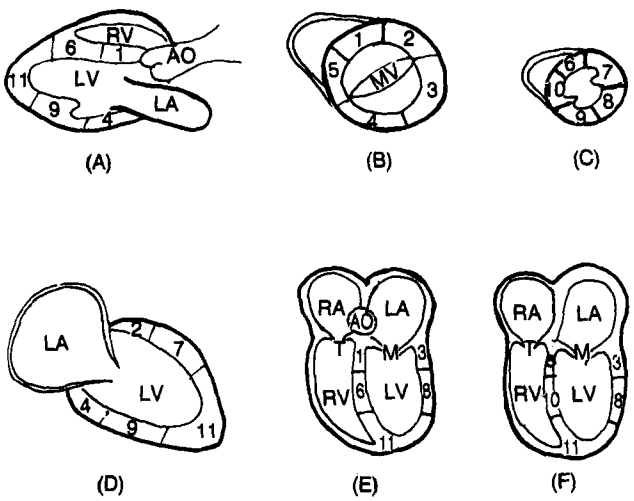

FIG. 1 Segmental subdivision used in the two-dimensional echocardiographic analysis of hypertrophic cardiomyopathy. (A) parasternal long-axis view; (B) parasternal short-axis view at the level of mitral valve; (C) parasternal short-axis view at the level of the papillary muscles; (D) apical two-chamber view; (E) apical five-chamber view; (F) apical four-chamber view. Ao=aorta, LA=left atrium, LV=left ventricle, MV=mitral valve, RA=right atrium, RV=right ventricle, T-tricuspid valve.

Electrocardiography-Vectorcardiography

Left ventricular hypertrophy was diagnosed on ECG using the point score system proposed by Romhilt and Estes (≥ 5 points)[8] and the Sokolow-Lyon index (SV1+RV5=35 mm).[9] Q or QS waves were considered abnormal if they were ≥ 0.004 s in duration or ≥ 3 mm deep and were present in two or more leads. T waves were considered giants if they were ≥ 10 mm in depth. Septal hypertrophy was diagnosed if R/S was >0.20. Diagnosis of left ventricular hypertrophy and left atrial enlargement was formulated on VCG according to the criteria of Piccolo.[10]

Statistical Analysis

Values are reported as the mean $\pm$ standard deviation. Student's *t*-test was used to analyze the differences between variables.

Results

Clinical Data

In our study group of 42 patients, 14 (33%) were asymptomatic. Among the others, chest discomfort was present in 12 (28%), dyspnea in 17 (40%), syncope in 11 (26%).

Electrovectorcardiographic Data

ECG-VCG abnormalities were recorded in all patients (Table I). Left atrial enlargement and repolarization changes were the most common findings (21 cases, 50%). Left ventricular hypertrophy was present in 18 patients (42%), pseudonecrosis in 18 (42%), septal hypertrophy in 7 (16%), and left anterior fascicular block in 6 (14%). More than one abnormality was frequently observed in the same patient.

24-H Holter Monitoring

Eight patients showed isolated or repetitive sporadic atrial premature beats. Two patients had chronic atrial flutter or fibrillation. Ventricular arrhythmias were observed in 26 patients (61%): 10 had isolated monomorphic PVCs (Lown grade 1); 10 had polymorphic PVCs (Lown grade 3), and 6 had repetitive ventricular arrhythmias which presented as couplets and triplets in 5, and sustained ventricular tachycardia in 1 (Lown grade 4a and b) (Fig. 2). One patient with polymorphic PVC presented rare premature beats with R/T phenomenon (Fig. 3). Asymptomatic episodes of ST depression with negative T waves were recorded in 2 patients, while a ST depression was associated with angina in 1 case (Fig. 4).

TABLE I Percentage of ECG-VCG abnormalities

	Number of patients	%
Left atrial enlargement	21	50
Repolarization changes	21	50
Left ventricular hypertrophy	18	42
Pseudonecrosis	18	42
Septal hypertrophy	7	16
Grade I AV block	6	14
Left anterior fascicular block	6	14
Right bundle-branch block	5	11
Right ventricular hypertrophy	2	4
Right atrial enlargement	1	2
Left posterior fascicular block	1	2

Echocardiography

M-Mode Echocardiography

Ventricular septal thickness ranged from 8 to 32 mm (mean 19.5±5 mm); posterior left ventricular free wall thickness ranged from 6 to 18 mm (mean 10±22). In 40 patients, the septal to free wall thickness ratio was ≥1.3 mm.

Midsystolic closure of the aortic valve was present in 14 patients (33%) and systolic anterior motion of the mitral valve was recorded in 26 patients (61%). Left atrial size ranged from 25 to 62 mm (mean 42±7). End-diastolic and end-systolic dimensions ranged from 32 to 54 mm (mean 44±5), and 18 to 36 mm (mean 26±5 mm), respectively. The mean shortening fraction (Δ%) was 42%±8.3.

Two-Dimensional Echocardiography

Ten patients (23%) had hypertrophy localized at the anterior portion only of the ventricular septum (type I) (Fig. 5), while in 3 patients (7%), hypertrophy involved most, or all of the ventricular septum (type II) (Fig. 6). Twenty-seven patients showed extensive hypertrophy involving substantial portions of both the ventricular septum and the anterolateral LV free wall (Fig. 7) (type III). Two patients presented apical hypertrophy (type IV) (Fig. 8). The HS and HSI means in type I were HS 7±1.9 and 0.18±0.4, respectively; in type II, 14±2, and 0.40±0.9, respectively, and in type III, 26±5, and 0.71±0.11, respectively. Two patients with apical hypertrophy had an HS of 3 and 7, respectively. The HS and HSI were significantly higher in patients with type III distribution ($p<0.001$) than in others.

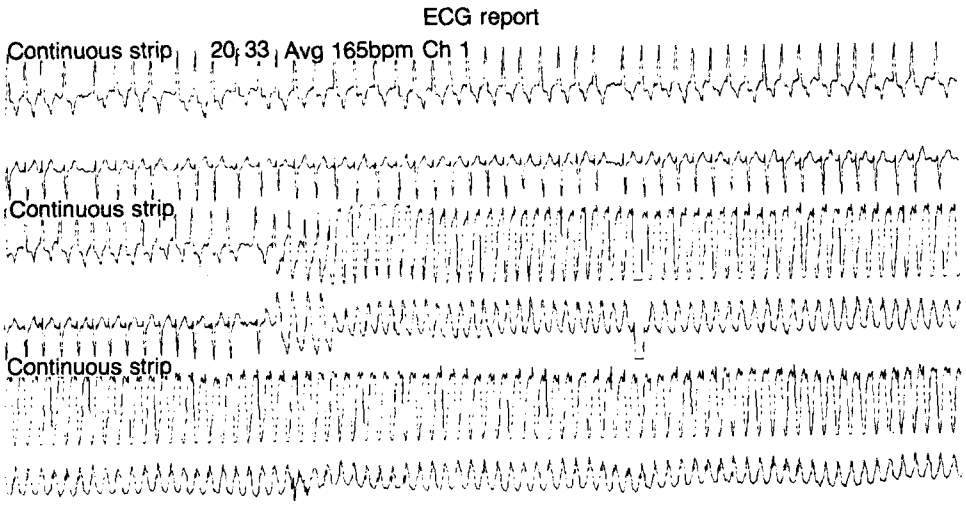

FIG. 2 Electrocardiographic recording during 24-h Holter monitoring in a patient with sustained ventricular tachycardia. The basal strip shows atrial fibrillation at a mean ventricular rate of 150 beats/min. At 20.33, a sustained monomorphic ventricular tachycardia with a cycle length of 300 ms appears.

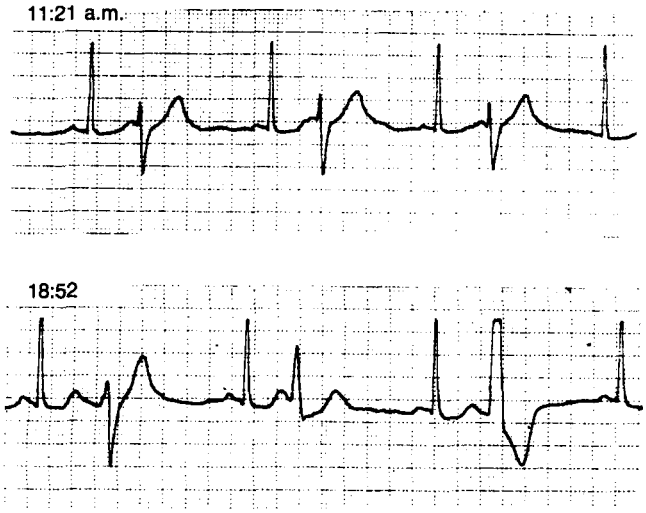

FIG. 3 Electrocardiographic strips in the patient with polymorphic premature ventricular contractions and R/T phenomenon. (Top) Premature monomorphic ventricular contractions with R/T phenomenon are present at 11:21 AM. (Bottom) The same patient showed polymorphic ventricular contractions at 18:52.

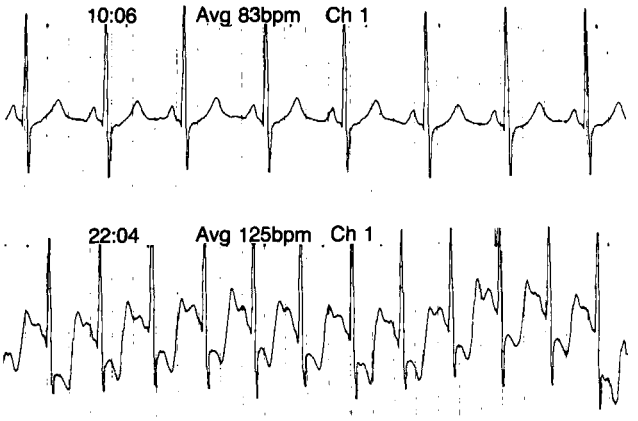

FIG. 4 Electrocardiographic strips of the patients with ST depression and angina. (Top) Basal ECG at 10:06 AM. (Bottom) ECG recorded at 22:04 during chest pain attack. Note the marked ST depression associated with a significant increase in heart rate.

Correlations Between Symptoms and Left Ventricular Hypertrophy

Fourteen asymptomatic patients showed a wide range of HS and HSI (respectively, 6–31 mean 19.1±9.9 and 0.15–0.84, mean 0.51±0.28). Five of these patients fullfilled criteria for type I, one for type II, and 8 for type III (Table II).

Seventeen patients who reported dyspnea had HS in the range of 6–33 (mean 23.1±8.6), and HSI 0.15–0.84 (mean 0.61±0.22), 13 of 17 had a score ≥22. Angina was present in 12 patients, and their HS range was 3–33 (mean 19.2±10.4) (Fig. 9).

Eleven patients were symptomatic for lipothymia or syncope; in these cases HS range was 10–35 (mean 23.5±7.8), and HSI was 0.15–0.84 (mean 0.63±0.21).

The correlation between symptoms and different patterns of hypertrophy is reported in Table II; no statistical significance was found between HS and HSI, and symptoms.

Correlation Between ECG-VCG Patterns and Left Ventricular Hypertrophy

In 21 patients with left atrial enlargement, HS and HSI covered a wide range (5–35, mean 19.8±9.7; 0.15–0.84, mean 0.5±0.25, respectively) (Fig. 10). In the patients with atrial enlargement on ECG, the mean left atrium dimension calculated by M-mode was not significantly different from the mean in patients without this ECG abnormality (43±7.8; 40±6).

Left ventricular hypertrophy on ECG-VCG was present in 18 patients, in which the HS range was 10–33 (mean 25±6.9), and HSI was 0.15–0.84 (mean 0.67±0.18). Moreover, this finding was more frequent in patients showing type III distribution (16 cases) (Table III).

TABLE II Relationship between symptoms, type of left ventricular hypertrophy, hypertrophy score, and hypertrophy score index

Type	Dyspnea	Angina	Syncope	Asymptomatic
I (n=10)	3	2	1	5
II (n=3)	1	—	2	1
III (n=27)	13	8	8	8
IV (n=2)	—	2	—	—
HS (mean±SD)	23.1±8.6	19.2±10.4	23.5±7.8	19.1±9.9
HSI (mean±SD)	0.61±0.22	0.59±0.23	0.63±0.21	0.51±0.28

Abbreviations: HS=hypertrophy score; HSI=hypertrophy score index.

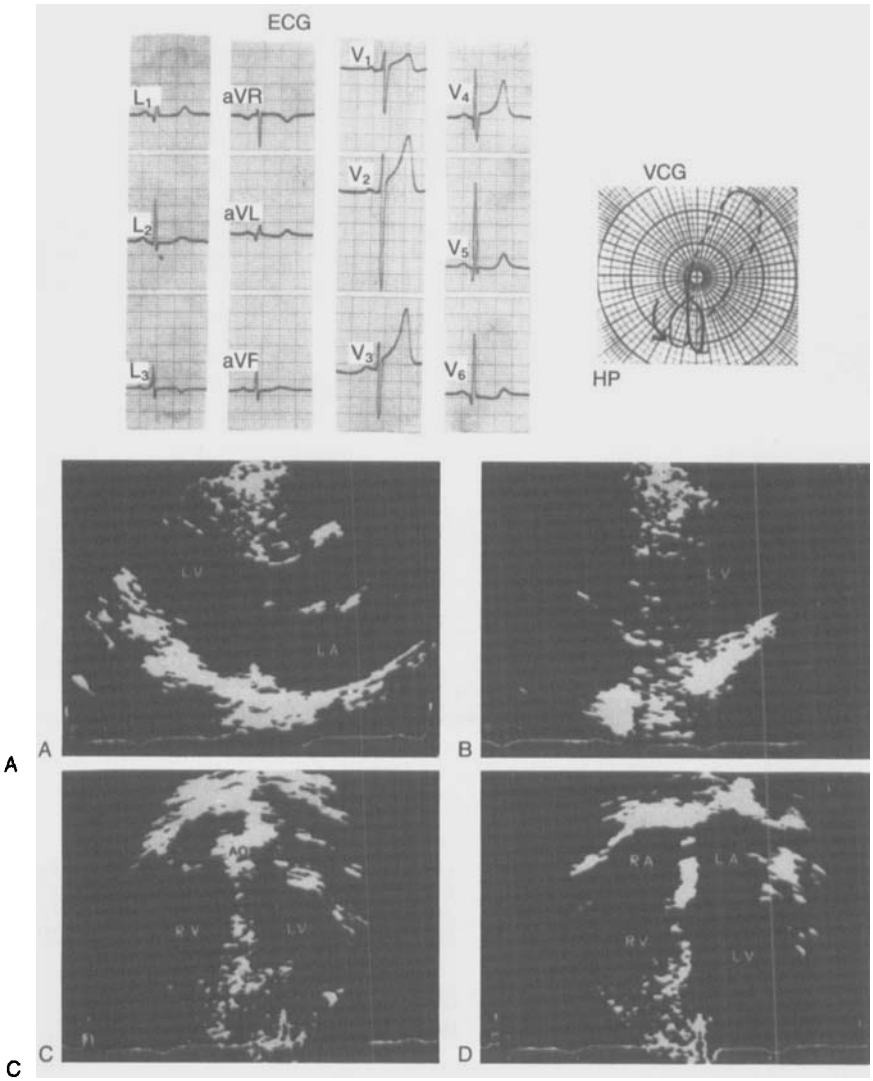

FIG. 5 ECG, horizontal plane VCG, and two-dimensional echocardiogram in a patient with type I distribution of hypertrophy. ECG-VCG: note marked amplitude of septal and paraseptal forces. Two-dimensional echocardiogram: presence of marked hypertrophy of anterior septum. (A) parasternal long-axis view; (B) parasternal short-axis view; (C) apical five-chamber view; (D) apical four-chamber view. Ao=aorta; IVS=interventricular septum; LA=left atrium; LV=left ventricle; RA=right atrium; RV=right ventricle.

TABLE III Relationship between ECG-VCG abnormalities, type of left ventricular hypertrophy, hypertrophy score, and hypertrophy score index

Type	LAE	RC	LVH	PN	SH	LAFB
I (n=10)	6	5	1	4	3	2
II (n=3)	3	2	1	2	—	—
III (n=27)	12	12	16	11	4	4
IV (n=2)	—	2	—	1	—	—
HS (mean±SD)	19.8±9.7	19.7±9.7	25±6.9	19±9.	18±10.9	18±9.4
HSI (mean±SD)	0.5±0.25	0.54±0.25	0.67±0.18	0.53±0.24	0.49±0.27	0.48±0.26

Abbreviations: LAE=left atrial enlargement, RC=repolarisation changes, LVH=left ventricular hypertrophy, PN=pseudonecrosis, SH=septal hypertrophy, LAFB=left anterior fascicular block.

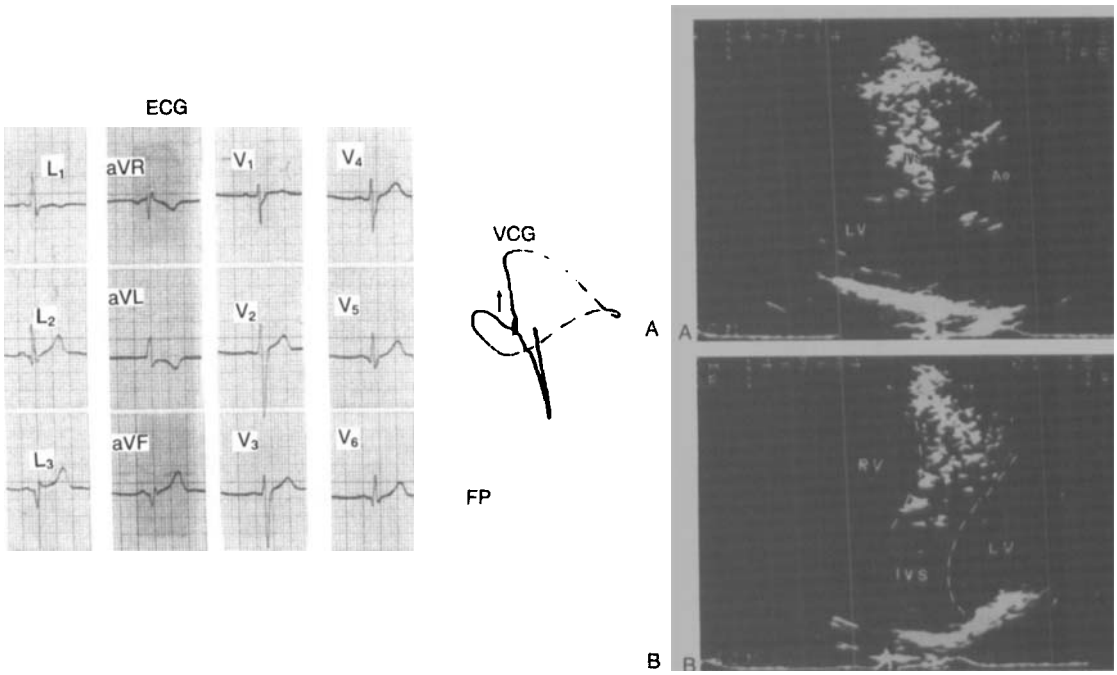

FIG. 6 ECG, frontal plane VCG, and two-dimensional echocardiogram in a patient with type II distribution of hypertrophy. ECG: note the pseudonecrosis pattern in L2-L3 and aVF leads. VCG: Clockwise rotation with upwards displacement of the initial forces. Two-dimensional echocardiogram showing marked hypertrophy of the interventricular septum. (A)=parasternal long-axis view. (B)=parasternal short-axis view. Ao=aorta; IVS=interventricular septum; LV=left ventricle, RV=right ventricle.

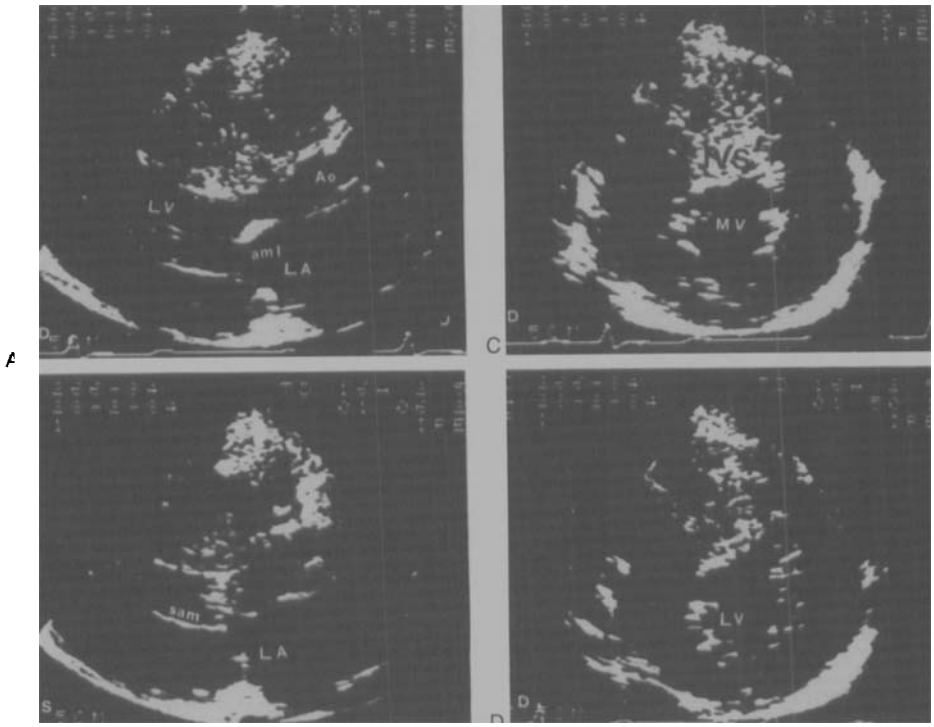

FIG. 7 Two-dimensional echocardiogram in patient with type III distribution of hypertrophy. (Left) parasternal long-axis view (diastole) (A), (systole) (B). (Right) parasternal short-axis views at the mitral valve (C) and at the papillary muscles (D). Stop-frames of two-dimensional echocardiograms showing marked hypertrophy of both the ventricular septum and anterolateral left ventricular free wall. Note the systolic anterior motion of the mitral valve. Ao=aorta, aml=anterior mitral leaflet, D=diastole, LA=left atrium, LV=left ventricle, MV=mitral valve, S=systole, SAM=systolic anterior motion.

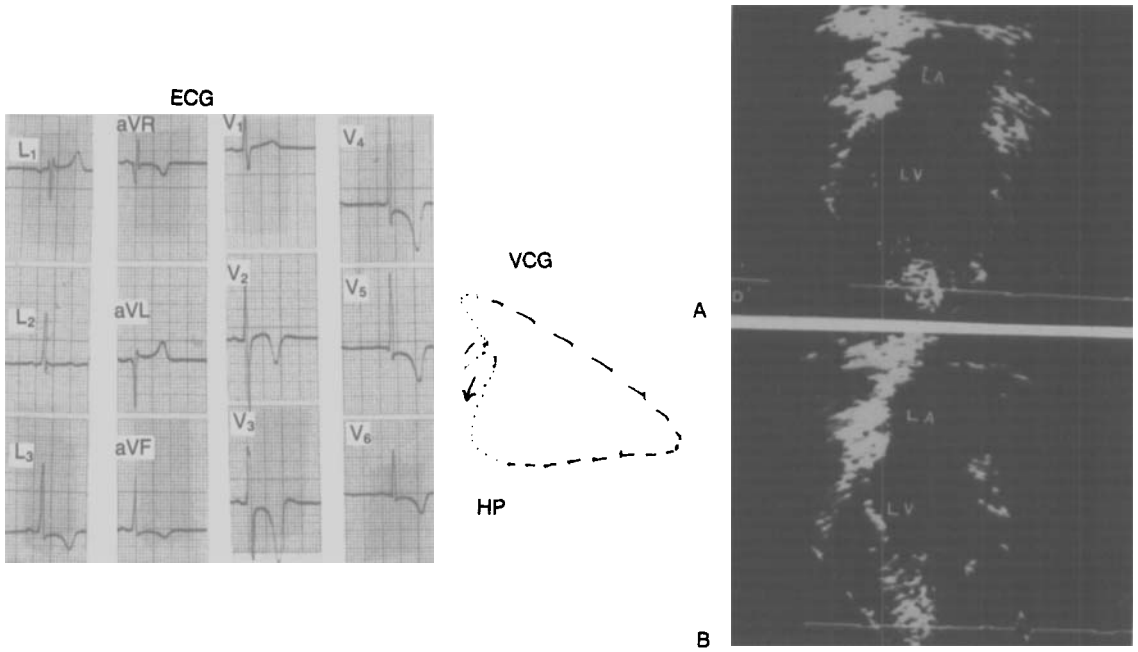

FIG. 8 ECG, horizontal plane VCG and two-dimensional echocardiogram in a patient with type IV distribution of hypertrophy. ECG: note marked amplitude of R wave in V1, V2, V3 leads and negative "giant" T waves. VCG: note marked predominance of anterior forces. Two-dimensional echocardiogram showing apical hypertrophy of the left ventricle. Apical two-chamber views. (A) diastole (D); (B) systole (S). La=left atrium, LV=left ventricle.

Abnormal Q waves in the inferior and lateral leads were demonstrated in 18 patients, with HS and HSI in the range 5-35 (mean 19±9.7), and 0.15-0.84 (mean 0.53±0.24), respectively. Electrocardiographic septal hypertrophy was found in 7 patients, with either low or high HS and HSI (Fig. 10). In 3 cases, ECG septal hypertrophy showed a type I pattern, and in 4 cases a type III pattern was shown. Left anterior fascicular block was observed in 6 patients (Fig. 10) (Table III). HS and HSI values are given in Table III.

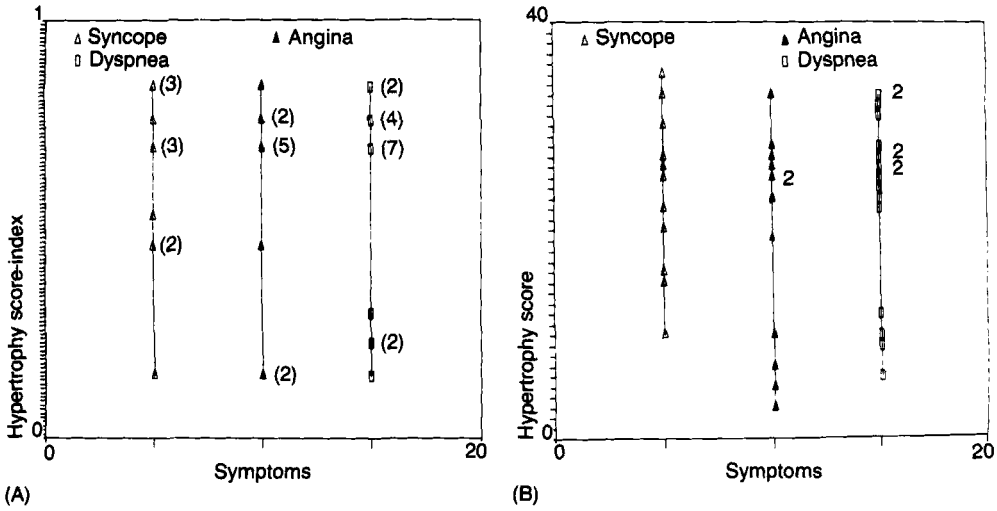

FIG 9 (A) Relationship between symptoms and hypertrophy score index. The number in parentheses represents the number of patients with the correspective index. The correlation is not significant. (B) Relationship between symptoms and hypertrophy score. No significance was found. The number represents the number of patients with the same score.

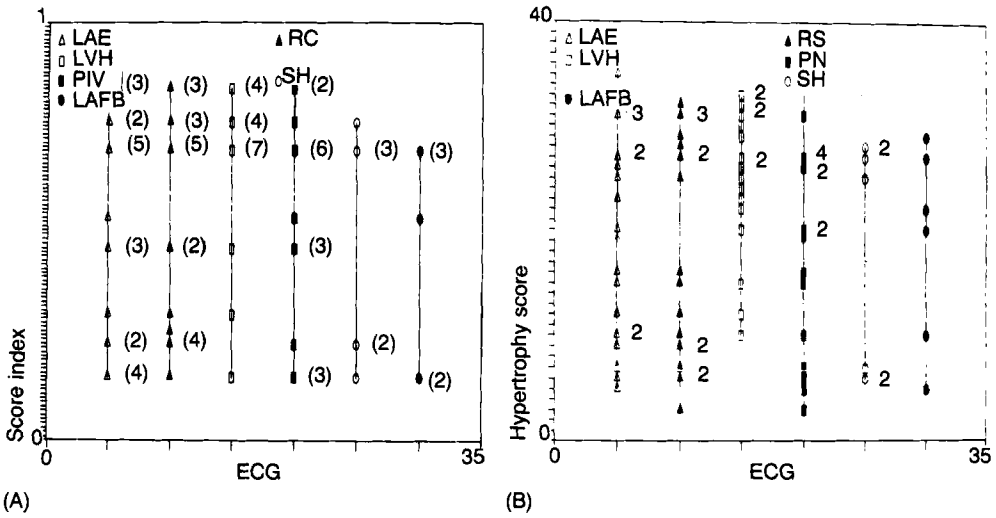

FIG. 10 (A) Relationship between ECG findings and hypertrophy score index. The number in parentheses represents the number of patients with the correspective index. No significance was found. (B) Relationship between ECG abnormalities and hypertrophy score. No significance was found. LAE=left atrial enlargement; LAFB=left anterior fascicular block; LVH=left ventricular hypertrophy; PN=pseudonecrosis; RC=repolarization changes; SH=septal hypertrophy.

ST-segment changes and T-wave inversion were evident in 21 cases, showing a wide range of HS and HSI (3–35, mean 19.7±10.7, and 0.15–0.84, mean 0.54±0.25, respectively). Negative giant T waves were present in 2 patients with type IV distribution of hypertrophy (apical hypertrophy) (Fig. 8). Different ECG-VCG patterns were present in the same patients. No significant correlation between ECG-VCG findings, and HS and HSI emerged even through patients with ECG evidence of LVH had the highest HS and HSI values.

Correlation of Ventricular Arrhythmias with Left Ventricular Hypertrophy

Ventricular arrhythmias Lown grade ≥3 were significantly more frequent in patients with a type III distribution pattern (Table IV). An analysis of HS and HSI disclosed a good correlation with the severity of ventricular arrhythmia (Fig. 11).

Hemodynamic Characterization

Twenty-four patients underwent cardiac catheterization, but only 7 demonstrated a left ventricular outflow tract pressure gradient of 30 mmHg or more (range 50–120, mean 72±27) under basal conditions. Among these 7 patients, 6 showed a type III pattern, and 1 a type I pattern with the hypertrophy localized in the basal portion of the anterior interventricular septum. The other 17 patients had either no basal gradient, or a small gradient (15–20 mmHg). Nine of the 24 patients had high left ventricular end-diastolic pressure (range 16–32; mean 18.8±5.3), and all 9 patients showed a type III pattern with a hypertrophy score ≥20.

Discussion

Maron*et al.*[5] identified four basic distribution patterns of left ventricular hypertrophy in patients with hypertroph-

TABLE IV Relationship between ventricular arrhythmias, type of hypertrophy, hypertrophy score, and hypertrophy score index

Type	Grade 1	Grade 3	Grade 4	Grade 5
I n=10	5	—	—	—
II n=3	1	1	—	—
III n=27	3	9	6	1
IV n=2	1	—	—	—
HS (mean±SD)	13.2±9.6	24.4±5	26.3±3.9	
HSI (mean±SD)	0.38±0.27	0.69±0.12	0.75±0.07	

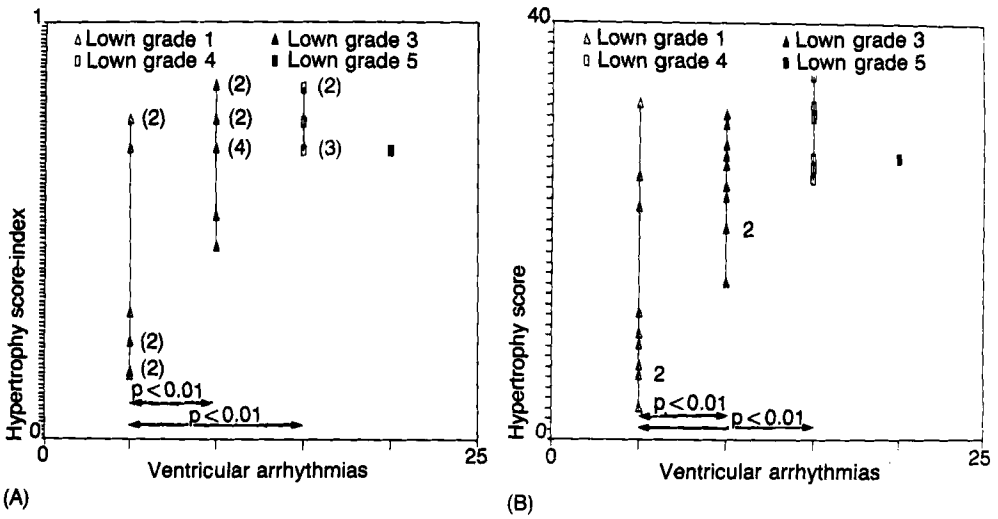

FIG. 11 (A) Relationship between severity of ventricular arrhythmias and hypertrophy score index. The number in parentheses represents the number of patients with the correscpective index. (B) Relationship between severity of ventricular arrhythmias and hypertrophy score. The number represents the number of patients with the same score.

ic cardiomyopathy, and many correlations with clinical and ECG findings were demonstrated.[7,11,12,24] Wigle *et al.* stressed the importance of the site and extent of hypertrophy in determining the disease manifestations.[2,13]

Patients with extensive hypertrophy are more likely to present abnormalities of systolic and diastolic function, as well as show a tendency for atrial and ventricular arrhythmias and sudden death.

To better quantify the extent of hypertrophy we used a two-dimensional echocardiographic score and score index[7] and we correlated these values with symptoms, electrovectorcardiographic data, and life-threatening arrhythmias. No correlation was found between symptoms and hypertrophy score and score index, even though dyspnea was more frequent in our cases with highest scores and score indexes; 76% of these dyspneic patients showed type III pattern of hypertrophic cardiomyopathy.[5]

These findings agreed with our hemodynamic data, as all the patients with high left ventricular end-diastolic pressure had a type III pattern.

According to Louie and Maron,[14] patients with elevated left ventricular hypertrophy and elevated score index may be asymptomatic. This contradictory phenomenon could be explained by complex interactions of different pathophysiological mechanisms in the production of symptoms.[15]

In correlating 2D echocardiograms with ECG-VCG findings, no significance was found between score and score index, and ECG-VCG data. However, ECG-VCG was a relatively sensitive marker (59%, 16/27) for extensive left ventricular hypertrophy even though our percentage is lower than that reported by Maron *et al.* (75%) in type III pattern of hypertrophy.[11] Abnormal Q waves were present in all the morphological types of HCM and for every score, but in patients with diffuse and severe hypertrophy, pseudonecrosis patterns were associated with ECG hypertrophy. Isolated conduction disturbances were more frequent in patients with localized hypertrophy, while left anterior fascicular block associated with left ventricular hypertrophy was present in diffuse hypertrophy.

According to Maron and others, except for the "giant" T waves in apical cardiomyopathy (where "giant" T wave inversion and "spadelike" morphology of the left ventricle are characteristic) no single ECG-VCG abnormality is typical of HCM.[11,12]

Therefore, it is extremely difficult to predict the site and extent of left ventricular hypertrophy on the basis of ECG-VCG findings. In our series more than one abnormality was often present in the same patient.

Previous studies indicate that ventricular tachycardia is the rhythm disturbance predictive of sudden death,[17,18] so we look for a correlation between life-threatening arrhythmias and the extent of the left ventricular hypertrophy. Ventricular arrhythmias were documented by Holter monitoring in 54 to 96% of patients[17-24] and ventricular tachycardia in 20 to 30% of patients.[24] In our series, ventricular arrhythmias were present in 61% of patients and in 14% complex, ventricular arrhythmias were recorded during Holter monitoring.

This incidence is lower than that found by others and could be due to a shorter monitoring time (24 h), compared to previous studies (48–72 h).[24-27] Independent of the incidence of ventricular arrhythmias, however, a good correlation was found between HS and HSI, and the severity of the ventricular arrhythmias. In fact, complex ventricular arrhythmias were more frequent in patients with high HS and HSI. The mechanism responsible for ventricular tachyarrhythmias in patients with severe and

diffuse hypertrophy could be the high intraventricular pressures which produce or worsen areas of myocardial ischemia.[28,29]

In conclusion, our study confirms the controversial literature reports of correlations between clinical data and left ventricular hypertrophy. Many pathophysiologic mechanisms, such as diastolic dysfunction, myocardial ischemia, and subaortic obstruction may play a different and unpredictable role in subjects with hypertrophic cardiomyopathy.

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