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Extent of hypertrophy in hypertrophic cardiomyopathy: two-dimensional echocardiographic and angiographic correlation

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Introduction

Since the first anatomical and functional descriptions of hypertrophic cardiomyopathy (HCM)[1,2] there have been convincing attempts at better understanding and definition of the controversial aspects of this complex disease. Echocardiography has proven to be the non-invasive technique best suited to assessing its morphological and functional configuration, allowing also the recognition of many asymptomatic patients and increasing the understanding of its genetic transmission[3–5]. The development of two-dimensional echocardiography, providing dynamic images of the heart in different cross-sectional planes, has facilitated the identification of different patterns of distribution of the hypertrophy[6–8]. However, since this technique has failed up to now to ascertain the presence and the severity of haemodynamic obstruction, cardiac catheterization remains an important diagnostic tool in symptomatic patients. The anatomic peculiarities of the left ventricle in HCM have been well described with cineangiographic studies[9–11].

The aim of the present study was to characterize the patterns of myocardial hypertrophy in patients with hypertrophic cardiomyopathy using both two-dimensional echocardiography and biventricular cineangiography.

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Methods

Our study considered two groups of patients with symptomatic hypertrophic cardiomyopathy: Group I, 28 patients (19 males and nine females) ranging in age from 15 to 68 years (mean 37 years) in whom two-dimensional echocardiography was available. Three of these patients had also haemodynamic measurements and left ventricular cineangiography. Group II, 10 patients (eight males and two females) ranging in age from 16 to 68 years (mean 45 years) had both two-dimensional echocardiography and biventricular cineangiography during cardiac catheterization. Both these evaluations were carried out within 1 month of each other in all cases. Two dimensional echocardiograms were performed using a Toshiba SSH-10 phased array 86° sector scan with a 2·25 MHz transducer. The following views were recorded: parasternal long axis (PSLAX) and short axis (PSSAX) at mitral valve, papillary muscles and apical levels: apical four chamber (AP4Ch) and apical long axis (APLAX). In some patients not all views were obtained.

Biventricular cineangiograms were performed during cardiac catheterization in the caudocranial left anterior oblique projection using two pigtail F8 catheters with simultaneous injection of Renographin 76 in the left and right ventricles.

All electrocardiographic and cineangiographic images were analysed independently by two observers. For the echocardiographic identification

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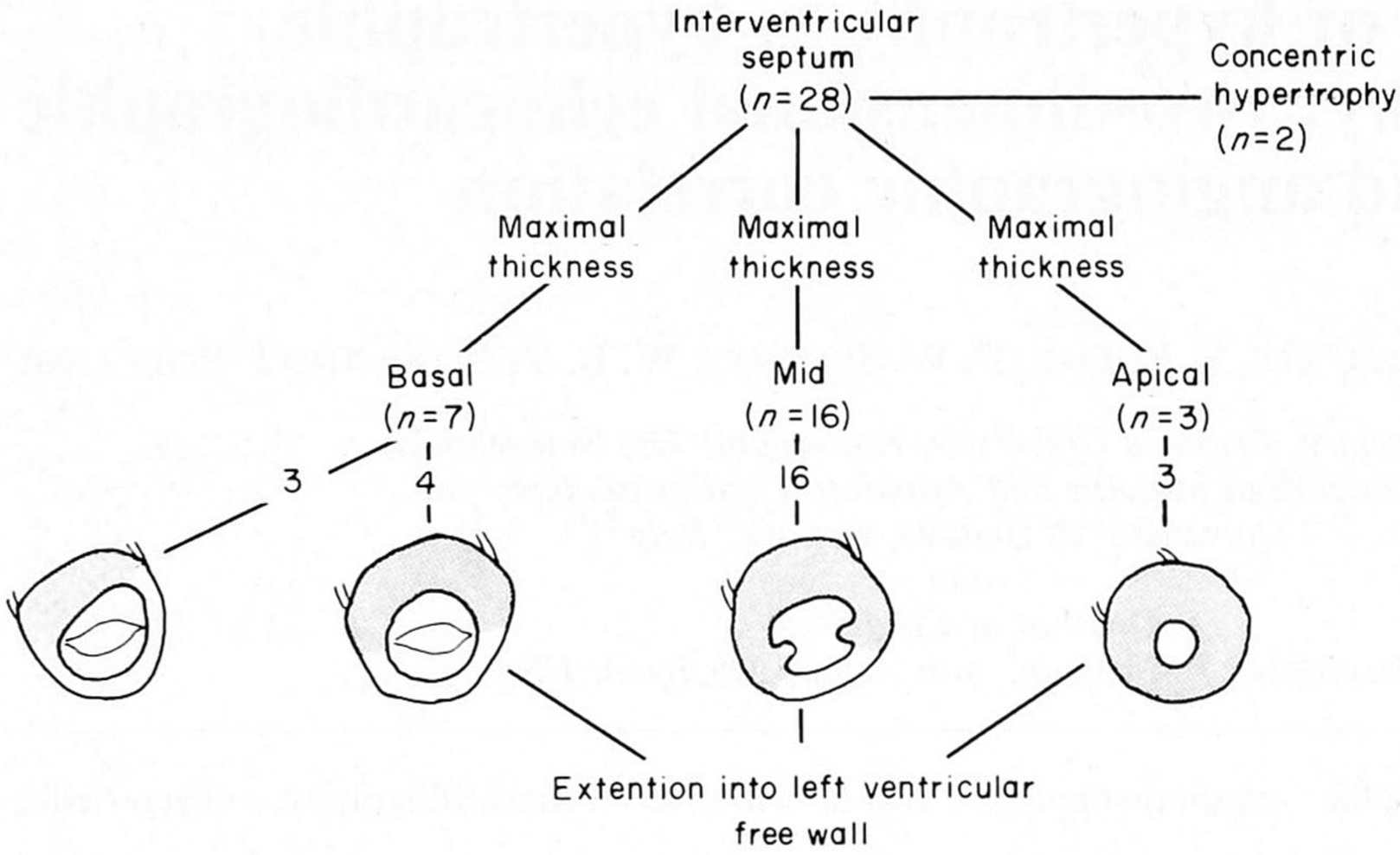

Figure 1 Distribution of left ventricular hypertrophy in patients with hypertrophic cardiomyopathy determined by two-dimensional echocardiography.

of the hypertrophic myocardial wall segments we followed the recommendations of the Committee of Nomenclature and Standards of the American Society of Echocardiography.

Results

GROUP I (TWO-DIMENSIONAL ECHOCARDIOGRAPHIC STUDY – 28 PATIENTS)

Echocardiographic study revealed the presence of septal hypertrophy in all 28 patients (Fig. 1). In two patients there was concentric hypertrophy. We divided the other 26 patients into three subgroups based on the location of maximal septal thickness in the long axis plane of the heart. In seven patients the maximal thickness was in the basal portion, in 16 in the mid portion and in three in its apical portion. Using the three different standard short axis views it was possible to evaluate the extension of the hypertrophy into the left ventricular free wall. Hypertrophy extended into the free wall in four of seven patients with maximal thickness in the basal septal region, whereas it was present in all 16 patients with maximal septal thickness in the mid portion. In the three patients in whom maximal septal thickness was detected in the apical region, the left ventricular free wall was also found to be hypertrophied.

GROUP II (TWO-DIMENSIONAL ECHOCARDIOGRAPHIC AND BIVENTRICULAR CINEANGIOGRAPHIC STUDY – 10 PATIENTS)

In the 10 patients studied by both two-dimensional echocardiography and biventricular cineangiography, close agreement was found between the two techniques for demonstrating the distribution of the hypertrophy along the interventricular septum. However, biventricular cineangiography failed to detect the presence of concentric hypertrophy. In two patients the extension of the hypertrophy into the anterolateral left ventricular free wall which was observed during echocardiographic examination was not detected (Fig. 2). This may be explained by the fact that cineangiography does not allow adequate visualization of the free wall of the left ventricle.

On the basis of cardiac catheterization data obtained in 13 patients (undergoing both two-dimensional echocardiograms and cardiac catheterization), seven of these patients had a resting pressure gradient and one only on provocation (Fig. 3). The gradient was noted in the outflow tract of the left ventricle in all except one patient, in whom it was located in the body of the left ventricle.

The data were analysed for the presence of echocardiographic features of cavity elimination and systolic anterior motion of the anterior mitral valve leaflet. Cavity elimination was defined as an

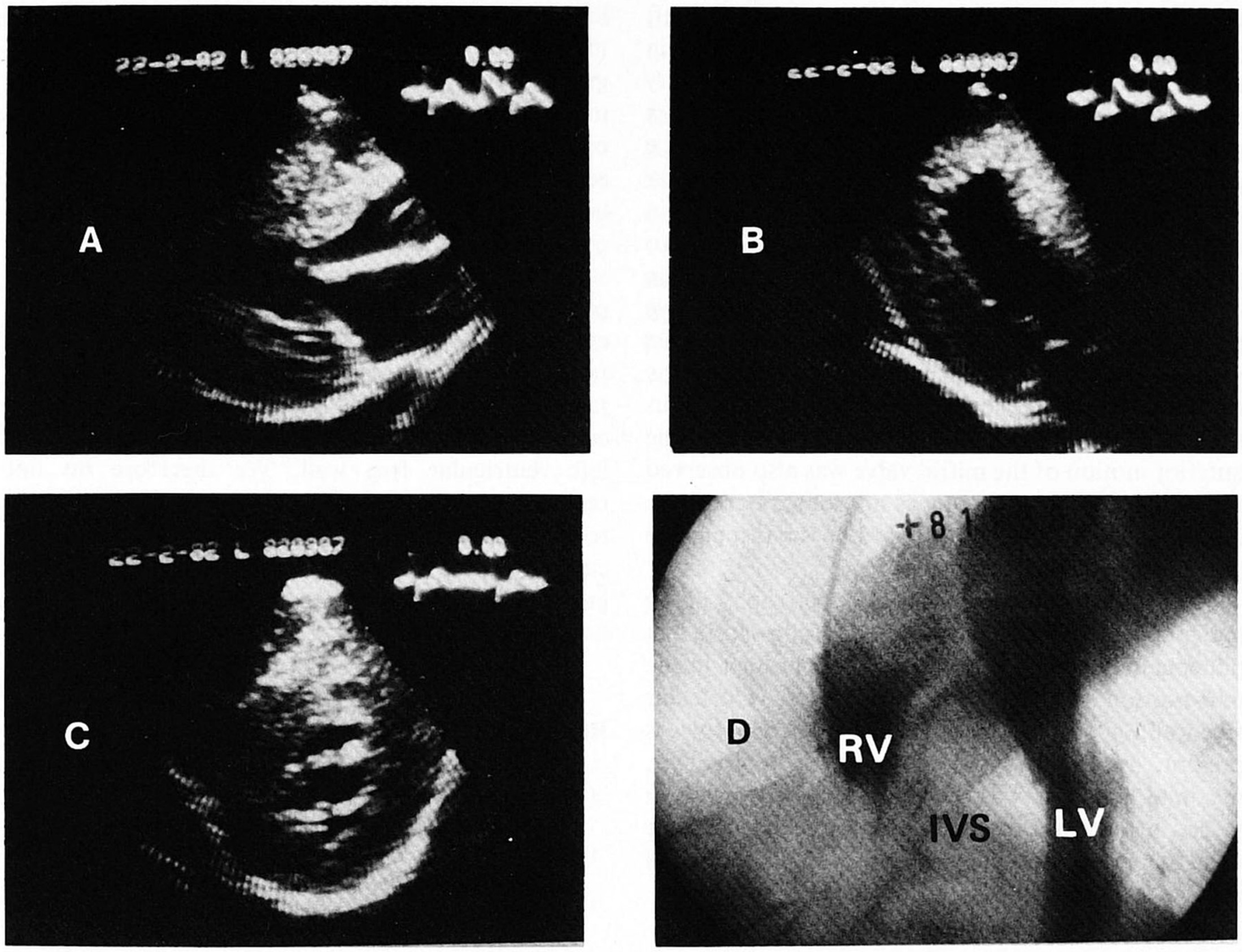

Figure 2 Echocardiographic and biventricular cineangiographic portrayal of the distribution of hypertrophy in a patient with hypertrophic cardiomyopathy. Echocardiographic views showing concentric hypertrophy (ABC) and biventricular cineangiogram showing marked septal hypertrophy (D).

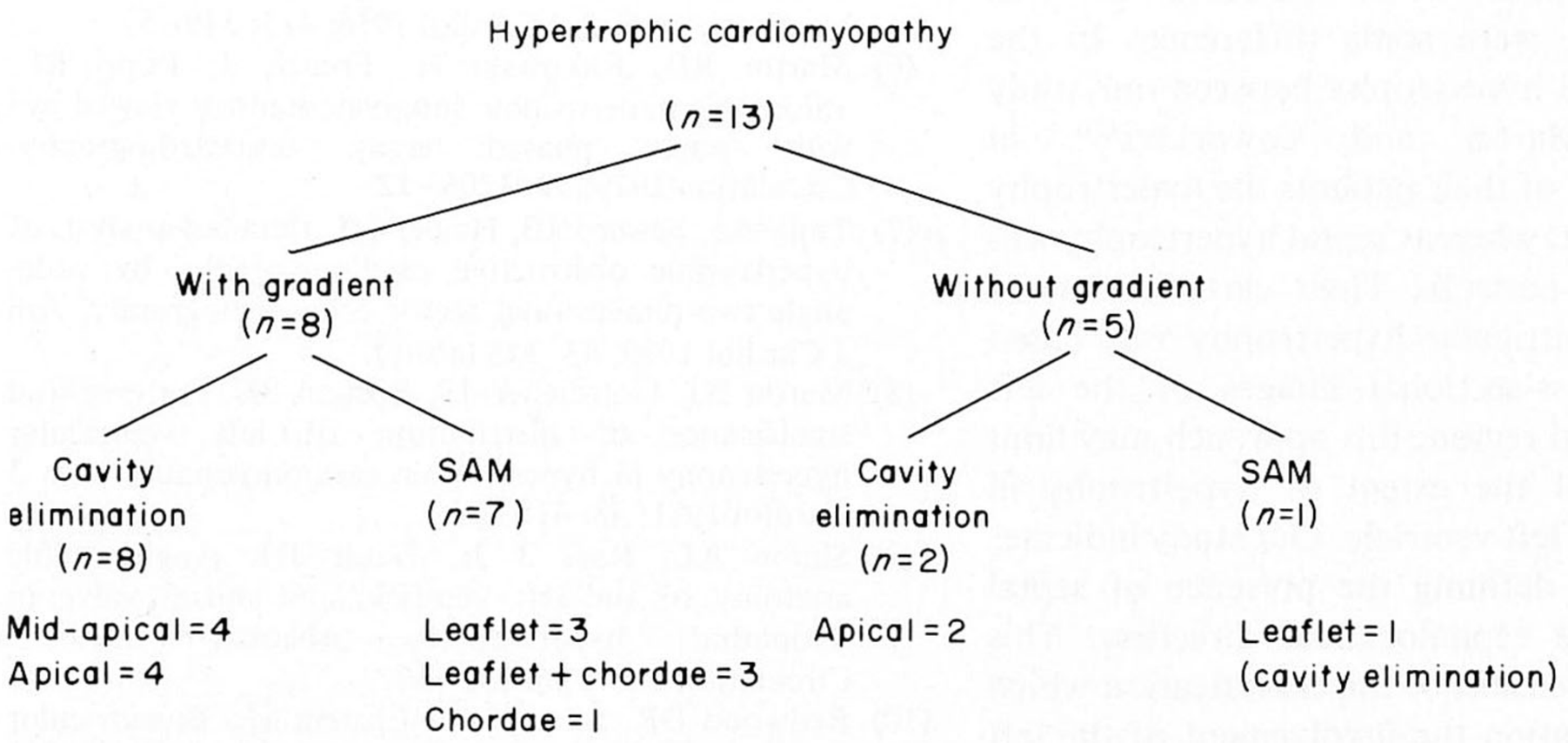

Figure 3 The prevalence of cavity elimination and systolic mitral leaflet motion in patients with and without left ventricular gradient in hypertrophic cardiomyopathy.

echocardiographic feature wherein a portion of left ventricular cavity is obliterated during systole, in more than one cross-sectional view. Cavity elimination was noted in all eight patients with a gradient and in two out of the five patients without a gradient. In the patients with a gradient, cavity elimination was confined to the mid-apical region in four and to the apical region in four. In the two patients without a gradient, it involved only the apical region. Systolic anterior motion of mitral valve leaflet was noted in seven out of the eight patients with a gradient: in three it involved the body of the anterior mitral valve leaflet, in three the leaflet plus the chordae, in one only the chordae. Systolic anterior motion of the mitral valve was also observed in one case in whom there was no pressure gradient: in this case the leaflet appeared to be the responsible structure involved.

The patterns of distribution of hypertrophy in the patients with a gradient were as follows: in six patients maximal septal thickness was present in the mid portion with extension into the left ventricular free wall; in two patients concentric hypertrophy was present. In the five patients without a gradient, maximal thickness was in the mid septal portion in three, in the basal septal portion in one and in the apical septal portion in one; left ventricular free wall involvement was noted in three of these patients.

Discussion and conclusion

Our study confirms that, although the extent of left ventricular hypertrophy in patients with hypertrophic cardiomyopathy is heterogeneous, it is possible to recognize basic patterns of this distribution. There were some differences in the prevalence of septal hypertrophy between our study and that of Maron and coworkers[7]: in approximately 90% of their patients the hypertrophy involved the septum, whereas septal hypertrophy was present in all our patients. Their classification of patterns of left ventricular hypertrophy was based principally on cross-sectional images of the left ventricle in the basal region; this approach may limit the appreciation of the extent of hypertrophy in other regions of the left ventricle. Our study indicates the importance of defining the presence of septal hypertrophy in the cephalocaudal direction. This allows a further extension of the classification which takes into consideration the involvement of the left ventricular free wall and its relation to the location of the maximal septal thickness. The application of such identification of myocardial wall segments recommended by the American Society of Echocardiography to patients with hypertrophic cardiomyopathy could be of particular help for a better comparison between the results of different echocardiographic studies. It may lead to a better understanding of the haemodynamic and anatomical correlates of this intriguing disease.

Biventricular cineangiography is useful in providing a more graphic appreciation and evaluation of the extent of septal hypertrophy in relation to the ventricular cavities. The method failed, however, to give precise information concerning presence or absence of involvement in the left ventricular free wall. We therefore do not recommend biventricular cineangiography as a routine procedure for the study of hypertrophic cardiomyopathy since two-dimensional echocardiograms will allow adequate morphological classification.

References

(1) Teare D. Asymmetrical hypertrophy of the heart in young adults. Br Heart J 1958; 20: 1–18.
(2) Brock RC. Functional obstruction of the left ventricle. Guys Hosp Rep 1957; 106: 221–38.
(3) Henry WL, Clark CE, Epstein SE. Asymmetrical septal hypertrophy (ASH). Echocardiographic identification of the pathognomonic anatomic abnormality of IHSS. Circulation 1973; 47: 225–33.
(4) Clark CE, Henry WL, Epstein SE. Familial prevalence and genetic transmission of idiopathic hypertrophic subaortic stenosis. N Engl J Med 1973; 289: 709–14.
(5) Van Dorp WG, ten Cate FJ, Vletter WB, Dohmen H, Roelandt J. Familial presence of asymmetric septal hypertrophy. Eur J Cardiol 1976; 4/3: 349–57.
(6) Martin RP, Rakowski H, French J, Popp RL. Idiopathic hypertrophic subaortic stenosis viewed by wide angle phased array echocardiography. Circulation 1979; 59: 1206–12.
(7) Tajik AJ, Seward JB, Hagler DJ. Detailed analysis of hypertrophic obstructive cardiomyopathy by wide-angle two-dimensional sector echocardiography. Am J Cardiol 1979; 43: 348 (abstr).
(8) Maron BJ, Gottdiener JS, Epstein SE. Patterns and significance of distribution of left ventricular hypertrophy in hypertrophic cardiomyopathy. Am J Cardiol 1981; 48: 418–28.
(9) Simon AL, Ross J Jr, Gault JH. Angiographic anatomy of the left ventricle and mitral valve in idiopathic hypertrophic subaortic stenosis. Circulation 1967; 36: 852–67.
(10) Redwood DR, Scherer JL, Epstein SE. Biventricular cineangiography in the evaluation of patients with asymmetrical septal hypertrophy. Circulation 1974; 49: 1116–21.

(11) Green CE, Elliott LP, Coghlan HC. Improved cineangiographic evaluation in hypertrophic cardiomyopathy by caudocranial left anterior oblique. Am Heart J 1981; 102: 1015–21.

Olsen Two questions: First, have you been able to compare the dimensions of the interventricular septal thickening with these two methods, biventricular angiography and two-dimensional echocardiographic studies? Secondly, have you been able to compare either of these methods with subsequent post-mortem studies and can you give me an indication of the margin of error in these various forms of investigations?

Das To answer the second question, none of these patients is dead, so I could not verify actual wall measurements. With respect to the first question, in terms of determining thickness, obviously these are two different methods and we did not attempt to measure or estimate the thickness because each method has its own limitation. The angiographic method is often misleading, particularly in estimating wall thickness.