B-scan ultrasonography in idiopathic hypertrophic subaortic stenosis¹

Study of left ventricular outflow tract and mechanism of obstruction

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Studies were made with standard time motion and B-scan echocardiography on 48 patients including 5 with idiopathic hypertrophic subaortic stenosis (hypertrophic obstructive cardiomyopathy), undergoing diagnostic cardiac catheterization. The dimensions of the left ventricular outflow (O) and inflow (I) tracts were measured on the B-scan images. The outflow tract was significantly narrowed in idiopathic hypertrophic subaortic stenosis at both end-systole (1.1 ± 0.1 cm) and end-diastole (1.3 ± 0.1 cm) when compared with the average width in other patients (2.6 ± 0.1 and 3.0 ± 0.1 cm, at end-systole and end-diastole, respectively) ($P < 0.001$) or normal subjects (2.4 ± 0.3 and 2.9 ± 0.2 cm) ($P < 0.01$). Furthermore, the O/I ratio differed significantly in idiopathic hypertrophic subaortic stenosis (0.5 ± 0.1 at end-systole and 0.6 ± 0.1 at end-diastole) from that in all other groups (1.4 ± 0.1 at both end-systole and end-diastole) ($P < 0.005$). There was no appreciable change in the width of the outflow tract from mid- to end-systole in the two patients in whom this was examined. The data support the contention that the anterior leaflet of the mitral valve assumes an abnormally anterior position in idiopathic hypertrophic subaortic stenosis. Though the systolic anterior movement of the tip of the anterior leaflet of the mitral valve shown by M-mode echocardiography could not readily be confirmed with B-scans, we believe that the narrowed outflow tract found in the present investigation contributes to the obstruction that occurs in this disease. We suggest that this outflow tract narrowing is probably caused by hypertrophy of the ventricular septum which in itself contributes to the narrowing, but which also displaces the papillary muscles and thus produces abnormal traction on the mitral valve and striking anterior displacement of the valve apparatus.

Idiopathic hypertrophic subaortic stenosis (hypertrophic obstructive cardiomyopathy) is a familial condition (Clark, Henry, and Epstein, 1973) which results in varying degrees of obstruction of the left ventricular outflow tract. Recent haemodynamic (Brockenbrough, Braunwald, and Morrow, 1961; Braunwald and Ebert, 1962; Krasnow *et al.*, 1963; Pierce, Morrow, and Braunwald, 1964) and angiographic (Dinsmore, Sanders, and Harthorne, 1966; Simon, Ross, and Gault, 1967; Adelman *et al.*, 1969; Simon, 1972) studies have defined the dynamic nature of this obstruction. Throughout the

cardiac cycle the asymmetrically hypertrophied interventricular septum significantly narrows the outflow tract. Abnormal angulation of papillary muscles prevents the anterior mitral leaflet from normally swinging posteriorly at end-diastole, thus further compromising the outflow tract at the onset of systole (Simon *et al.*, 1967). Alteration of the outflow tract size and resulting pressure gradient by numerous pharmacological agents and physiological interventions attests to the dynamic nature of this obstruction (Brockenbrough *et al.*, 1961; Braunwald and Ebert, 1962; Krasnow *et al.*, 1963). The introduction of ultrasound techniques has greatly simplified the diagnosis of this disorder and has helped explain its pathophysiology. Asymmetric

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septal hypertrophy (Pridie, Behnam, and Wild, 1972; Henry, Clark, and Epstein, 1973a; Epstein *et al.*, 1974) and early systolic anterior movement of the anterior mitral leaflet (Popp and Harrison, 1969; Shah, Gramiak, and Kramer, 1969; Shah *et al.*, 1971; Pridie *et al.*, 1972; Henry *et al.*, 1973b; King *et al.*, 1973a; Epstein *et al.*, 1974) are the echocardiographic abnormalities which have been demonstrated in idiopathic hypertrophic subaortic stenosis. Not well appreciated is the evidence from echocardiography of narrowing of the outflow tract in both systole and diastole caused by the relatively fixed abnormal anterior displacement of the mitral valve apparatus (Gramiak, Shah, and Kramer, 1969; Moreyra *et al.*, 1969; Popp and Harrison, 1969; Shah *et al.*, 1969; Gramiak and Shah, 1971; Shah *et al.*, 1971; Abbasi *et al.*, 1972; Feigenbaum, 1972; Pridie *et al.*, 1972; Shah *et al.*, 1972; Henry *et al.*, 1973b; King *et al.*, 1973a; Williams, Ellison, and Nadas, 1973; Epstein *et al.*, 1974; Henry *et al.*, 1975). This narrowing can best be defined by an ultrasound technique which scans the entire length of the outflow tract. The use of this technique forms the basis of the present report.

Subjects and methods

Five patients (2 males, 3 females) with idiopathic hypertrophic subaortic stenosis were evaluated with both standard M-mode and B-scanning echocardiographic techniques. Examinations were performed less than 24 hours before diagnostic cardiac catheterization in all but one patient whose catheterization was 13 months before the ultrasound study. The diagnosis of idiopathic hypertrophic subaortic stenosis was confirmed by the finding of characteristic haemodynamic abnormalities either at rest or after inotropic stimulation (Braunwald *et al.*, 1964). Of the additional 43 patients (36 men, 7 women) studied in a similar fashion, 26 had coronary artery disease and 3 of these had co-existent mitral regurgitation. Six patients had primary valvular disease (aortic stenosis 2, aortic regurgitation 1, mitral stenosis 2, mitral regurgitation 1). An additional 4 patients had left-to-right shunts (atrial septal defect 3, ventricular septal defect 1), and 7 were normal.

The technique for performing the ultrasound studies has previously been described (Teichholz *et al.*, 1974). Briefly, standard echocardiographic recordings as well as B-scans were made in all patients using a Unirad Sonograf and a 2.25 MHz transducer placed in either the third, fourth, or fifth intercostal space. The ultrasonic 'B' mode display was presented on a Tektronix storage oscilloscope

and photographed with a Polaroid camera. A gate triggered by the patient's electrocardiogram was used to produce stop-action, two-dimensional cross-sectional images of the heart at end-systole and end-diastole. As previously explained (Teichholz *et al.*, 1974), the peak of the R wave was chosen as end-diastole and the D point of the mitral valve echogram as end-systole. The plane of the scanning arm was adjusted to pass through the aortic root, anterior leaflet of the mitral valve, interventricular septum, left ventricular posterior wall, and cardiac apex. The transducer was slowly moved along the chest wall, and portions of the left ventricular silhouette were developed at each point by sector scanning. In this manner a complete silhouette could be formed on a storage oscilloscope and subsequently photographed. Spatial orientation of all structures recorded in the silhouettes was possible by simultaneously observing an A-mode display and photographing a time-motion echogram on a strip-chart recorder while the scans were being formed. 1 cm interval markers were recorded in each photograph to allow measurement of absolute dimensions.

Only left ventricular silhouettes clearly showing the anterior leaflet of the mitral valve were analysed. Because of the rapid movement of the tip of this leaflet, it was not always possible to record its motion in the scan, and therefore measurements have been made to the mid-portion of the visualized leaflet, at least 1 cm below the mitral ring. The distance between the anterior leaflet and the base of the posterior left ventricular wall immediately distal to the atrioventricular junction was taken to represent the inflow tract (I) (Fig. 1). The left ventricular outflow tract (O) was measured as the distance between the leaflet and the base of the septum immediately caudad to the aortic root or bulging superior portion of the interventricular septum in those patients where this was apparent. These measurements were made directly from the scan photographs in both end-systole and end-diastole. The ratio O/I was calculated in both phases of the cardiac cycle.

Results are presented as mean values $\pm$ standard error of the mean.

Results

A B-scan from a patient with coronary artery disease is presented in Fig. 1. This scan shows the normal position of the anterior leaflet of the mitral valve at end-systole. In this patient $O=2.6$ cm, $I=2.5$ cm, and O/I is 1.04. By contrast, Fig. 2 represents an end-systolic left ventricular silhouette

FIG. 1 Photograph (left) and artist's sketch (right) of an end-systolic B-scan from a patient with two-vessel coronary artery disease. The anterior leaflet of the mitral valve (ALMV) is normally positioned. The outflow tract (O) measures 2.6 cm and the inflow tract (I) 2.5 cm, while the O/I ratio is 1.04. The distance between the interval markers on the photograph represents 1 cm.

Abbreviations: PM=papillary muscle, Ao=aorta, LA=left atrium, LV=left ventricle.

FIG. 2 Photograph (left) and artist's sketch (right) of an end-systolic B-scan from a patient (Case 3) with idiopathic hypertrophic subaortic stenosis and a 60 mmHg (8.0 kPa) gradient between left ventricle and aorta at rest. The anterior leaflet of the mitral valve (ALMV) is anteriorly displaced and is separated from the prominent septal bulge by only 0.9 cm, thus significantly narrowing the outflow tract (O). The width of the inflow tract (I) is 3.7 cm and the O/I ratio is 0.24. The distance between the interval markers on the photograph represents 1 cm.

Abbreviations: see Fig. 1.

in a patient (Case 3) with idiopathic hypertrophic subaortic stenosis. In this patient the resting outflow tract gradient was 60 mmHg (8.0 kPa). The anterior leaflet of the mitral valve is displaced anteriorly, producing significant narrowing of the left ventricular outflow tract during systole: O=0.9 cm, I=3.7 cm, and O/I=0.24. The data for individual patients with idiopathic hypertrophic

subaortic stenosis are presented in Table 1, and the results for all patients are tabulated in Table 2 and presented graphically in Fig. 3. There was obvious difference in the individual O/I ratios in all patient groups compared with those in the 4 patients with idiopathic hypertrophic subaortic stenosis with gradients either at rest or after spontaneous premature ventricular contractions. In these latter

patients O averaged 1.1 cm (range 0.9 to 1.3) at end-systole and 1.2 cm (range 0.9 to 1.5) at end-diastole with average O/I ratios of 0.38 (range 0.24 to 0.48) in systole and 0.44 (range 0.24 to 0.75) in diastole. By contrast, no patient with a cardiac diagnosis other than idiopathic hypertrophic subaortic stenosis had an outflow tract smaller than 1.5 cm at either end-systole or end-diastole. Only one subject without idiopathic hypertrophic subaortic stenosis had an O/I ratio less than 0.75 in either the end-systolic or end-diastolic scan. One patient with idiopathic hypertrophic subaortic stenosis had an outflow tract gradient seen only after isoprenaline infusion. This patient had a narrow outflow tract (1.3 cm in systole and 1.4 cm in diastole), though the O/I ratios were in the normal range (0.93 in systole and 1.00 in diastole).

study has used left ventricular B-scans to document the abnormal anterior position of the anterior mitral leaflet in idiopathic hypertrophic subaortic stenosis which may contribute to the outflow tract obstruction observed in this disorder.

Left ventricular angiographic studies have been invaluable for the understanding of the pathophysiology of idiopathic hypertrophic subaortic stenosis by showing an anatomical basis for the functional obstruction of the outflow tract (Dinsmore *et al.*, 1966; Simon *et al.*, 1967; Adelman *et al.*, 1969; Simon, 1972). The outflow tract is normally bounded by the interventricular septum anteriorly and laterally and the anterior mitral leaflet posteriorly. With asymmetrical hypertrophy of the muscular septum, there is direct encroachment of this muscle mass on the anterior part of the

TABLE 1 *Echocardiographic, angiographic, and haemodynamic data in patients with idiopathic hypertrophic subaortic stenosis*

Case No.	Date of cath.	Age	Echocardiography		B-scan			Mitral regurgitation	Left ventriculo-aortic pressure gradient (mmHg)		
			TAM-mode	MV anterior position	O (cm)	I (cm)	O/I		Rest	Post-ectopic	Isoprenaline
1	20/4/72	30	Yes	Yes	S 1.3 D 1.5	3.5 3.5	0.37 0.42	0	77	—	—
2	17/1/73	22	No	No	S 1.3 D 1.4	1.4 1.4	0.93 1.00	0	0	0	40–60
3	18/1/73	31	Yes	Yes	S 0.9 D 0.9	3.7 3.7	0.24 0.24	++	60	100	—
4	24/1/73	53	Yes	Yes	S 1.1 D 1.5	2.5 2.0	0.44 0.75	0	0	60	35
5	17/10/73	53	Yes	Yes	S 1.1 D 1.1	2.3 3.0	0.48 0.37	0	20	100	—

Abbreviations: Cath.=catheterization procedure; MV=mitral valve; O=width of left ventricular outflow tract; I=width of left ventricular inflow tract; Post-ectopic=beat following premature ventricular contraction; S=end-systole; D=end-diastole; SAM=systolic anterior movement.

In 2 patients with documented idiopathic hypertrophic subaortic stenosis and resting gradients a B-scan was also recorded at a time in mid-systole when the systolic anterior movement of the anterior mitral leaflet as seen in the standard echocardiogram was maximal. O, I, and O/I measurements at this time were identical to those measured at end-systole.

Discussion

B-scan ultrasonography uses echocardiographic techniques to generate conventional two-dimensional silhouettes of the left ventricle. These silhouettes illustrate geometrical relations between cardiac structures better than the standard time-motion display, though the latter provides better resolution of rapidly moving structures. The present

left ventricular outlet. The mid-superior portion of this hypertrophied septum projects into the ventricular cavity thus displacing the anterior papillary muscle anteriorly, superiorly, and medially away from the major longitudinal axis of the left ventricle (King *et al.*, 1973b; Reis *et al.*, 1974). This unusual angulation of the papillary muscle coupled with the lack of apex-to-base shortening, because of the massive septal hypertrophy (Klein, Lane, and Gorlin, 1965), produces abnormal traction on the chordae tendineae to the mitral valve. Therefore, during systole, when the septum bulges further posteriorly into the outflow tract, the anterior mitral valve leaflet is held in the outflow tract forming the posterior aspect of a dynamic obstruction. Direct observation at the time of surgery to relieve the obstruction has confirmed the role of the asym-

FIG. 3 Graph depicting the individual and mean inflow (I) and outflow (O) tract widths in patients with idiopathic hypertrophic subaortic stenosis (IHSS) and in all other patient groups. The difference between the mean O widths for IHSS and the other patients is highly significant ($P < 0.001$). The I widths are not statistically different. Abbreviations: NL=normal; CAD=coronary artery disease; RHD=rheumatic heart disease; Shunt (L→R)=left-to-right shunt.

metrical septal hypertrophy in the anterior displacement of the anterior mitral leaflet causing ballooning of the valve into the outflow tract and systolic obstruction (Bjork, 1964). As shown in the present study, the position of the mitral valve is abnormal during the entire cardiac cycle, and appreciable outflow tract narrowing exists even at the beginning of systole. During systole motion of the septum and mitral valve results in slight further narrowing of the outflow tract.

Echocardiography has confirmed the participation of the anterior mitral leaflet in the systolic narrowing of the left ventricular outflow tract in

idiopathic hypertrophic subaortic stenosis (Gramiak *et al.*, 1969; Moreyra *et al.*, 1969; Popp and Harrison, 1969; Shah *et al.*, 1969; Gramiak and Shah, 1971; Shah *et al.*, 1971; Abbasi *et al.*, 1972; Feigenbaum, 1972; Pridie *et al.*, 1972; Shah *et al.*, 1972; Henry *et al.*, 1973b; King *et al.*, 1973a; Williams *et al.*, 1973; Epstein *et al.*, 1974; Henry *et al.*, 1975), and three studies have successfully correlated the systolic anterior movement of the anterior mitral leaflet with the pressure gradient from left ventricle to aorta (Popp and Harrison, 1969; Shah *et al.*, 1971; Henry *et al.*, 1973b). However, few (Gramiak and Shah, 1971; Epstein

FIG. 4 End-diastolic B-scan from a patient with idiopathic hypertrophic subaortic stenosis. The outflow tract continues to be narrowed despite left ventricular relaxation. The outflow tract width is 0.9 cm, and the ratio of outflow to inflow tracts is 0.32. The distance between the interval markers represents 1 cm.

et al., 1974; Henry *et al.*, 1975) have commented on the degree of narrowing of the outflow tract, though many of the published echocardiograms show striking anterior displacement of the entire mitral valve echo in the left ventricular cavity. Using B-scan ultrasonography it is simple to measure accurately the width of the outflow tract. This study has shown that the average width of the outflow tract at end-systole in idiopathic hypertrophic subaortic stenosis in the plane of the scan is 1.1 cm, in contrast to the significantly wider outlet of 2.6 cm

in all other patients studied ($P < 0.001$) or 2.4 cm in the normal group ($P < 0.01$). Persistence of the narrow outflow tract at end-diastole is well shown in Fig. 4 from a patient with typical echocardiographic and phonocardiographic features of idiopathic hypertrophic subaortic stenosis who did not undergo cardiac catheterization. Significant differences exist between the average end-diastolic width of 1.3 cm in these patients and 3.0 cm in all other subjects or 2.9 cm in patients with normal hearts ($P < 0.001$). These measurements are similar to those of Henry *et al.* (1975) made with a real-time two-dimensional sector scanner. Echocardiographic measurements of the outflow tract width in idiopathic hypertrophic subaortic stenosis are similar to those made by standard angiographic techniques (Fix *et al.*, 1964). In addition, as noted in Fig. 2 and 4, the outflow tract is not narrowed at a single point, but over a distance of several centimetres, the entire length of the visualized anterior leaflet of the mitral valve.

The outflow tract narrowing probably results from a combination of anterior displacement of the anterior mitral leaflet and inward bulging of the interventricular septum. The relative importance of each factor cannot be established from this study. The average normal left ventricular inflow tract width of 2.1 cm is considerably less than that found in ventricles of patients with idiopathic hypertrophic subaortic stenosis (2.7 cm). Though one patient (Case 2) with a very narrow I prevents this difference from becoming statistically significant, the observation suggests that anterior displacement of the mitral valve is indeed plausible and may account for the wider I and narrower O in patients with idiopathic hypertrophic subaortic stenosis.

TABLE 2 Echocardiographic measurement of left ventricular inflow and outflow tracts

Condition	No. of patients	End-systole			End-diastole		
		O (cm)	I (cm)	O/I	O (cm)	I (cm)	O/I
Normal	7	$2.4 \pm 0.3^*$	2.1 ± 0.1	1.1 ± 0.1	2.9 ± 0.2	2.1 ± 0.2	1.5 ± 0.1
IHSS	5	$1.1 \pm 0.1^\S$	$2.7 \pm 0.4^\dagger$	$0.5 \pm 0.1^\S$	$1.3 \pm 0.1^\P$	$2.7 \pm 0.5^\dagger$	$0.6 \pm 0.1^\P$
Other cardiac disorders							
CAD	26	$2.7 \pm 0.1^\dagger$	$2.1 \pm 0.1^\dagger$	$1.4 \pm 0.3^\dagger$	$3.1 \pm 0.1^\dagger$	$2.5 \pm 0.1^\dagger$	$1.5 \pm 0.4^\dagger$
L→R shunt	4	$2.3 \pm 0.0^\dagger$	$2.0 \pm 0.5^\dagger$	$1.4 \pm 0.1^\dagger$	$2.6 \pm 0.3^\dagger$	$2.0 \pm 0.6^\dagger$	$1.3 \pm 0.1^\dagger$
Valvular disease							
Aortic	3	$2.9 \pm 0.5^\dagger$	$1.8 \pm 0.4^\dagger$	$1.8 \pm 0.5^\dagger$	$3.1 \pm 0.8^\dagger$	$2.0 \pm 0.5^\dagger$	$1.7 \pm 0.6^\dagger$
Mitral	3	$2.5 \pm 0.4^\dagger$	$1.6 \pm 0.1^\ddagger$	$1.5 \pm 0.2^\dagger$	$2.7 \pm 0.6^\dagger$	$2.3 \pm 0.4^\dagger$	$1.2 \pm 0.1^\dagger$

*Mean $\pm$ standard error of the mean.

Significance of difference between measurements in normal and pathological conditions:

$^\dagger P > 0.1$

$^\ddagger P < 0.05$

$^\S P < 0.01$

$^\P P < 0.001$.

Abbreviations: O=width of left ventricular outflow tract; I=width of left ventricular inflow tract; IHSS=idiopathic hypertrophic subaortic stenosis; CAD=coronary artery disease; L→R shunt=left-to-right shunt.

FIG. 5 A strip chart recording of an echocardiographic cardiac sweep in a patient with idiopathic hypertrophic subaortic stenosis. Note the anterior position of the mitral valve echo. As the transducer was moved in a cephalad direction (left to right on the strip chart recording), the aortic (Ao) root echoes became apparent. That the mitral valve is indeed anterior is conclusively shown by comparing the relative positions of the anterior leaflet of the mitral valve (ALMV) and the Ao root. In this recording one also notes a much thickened interventricular septum (IVS) and a prominent systolic anterior movement (SAM) of the mitral valve echo. Interestingly, as the transducer was moved in a cephalad direction, the size of the SAM diminished, until it almost disappeared in the last recorded mitral valve echo in the strip. Abbreviations: LA=left atrial; PLMV=posterior mitral valve leaflet.

FIG. 6 An ultrasonic cardiac sweep from a patient with a normal heart. The mitral valve is in a normal position relative to the aortic root and the left ventricular cavity. This position of the leaflet should be compared to the abnormal position of the mitral valve in a patient with idiopathic hypertrophic subaortic stenosis (Fig. 5).

FIG. 7 A strip chart recording of an echocardiogram in a patient with idiopathic hypertrophic subaortic stenosis.* The anterior position of the mitral valve echo is again noted. The systolic anterior movement (SAM) ('a') is also readily apparent. However, during systole there are multiple linear echoes ('b') immediately behind echo 'a'. It appears that different portions of the anterior mitral leaflet (AML) are drawn varying distances into the outflow tract, possibly because of a Venturi effect. Thus the SAM may represent the motion of only a small part of the mitral valve leaflet, perhaps the free edge or redundant valve tissue, while the major portion of the leaflet may move relatively little.

Abbreviations: PML=posterior mitral leaflet; post LV wall=posterior left ventricular wall.

*Recorded with Hoffrel Series 101 Ultrasonoscope interfaced with a Cambridge Multichannel recorder and strip chart recorder.

The above observation is probably not a result of the intrinsically small ventricle of these patients. Subjects with mitral stenosis and equally small left ventricular volumes had normal outflow tract dimensions and spatial orientation of the mitral valve. Therefore, the size of the outlet was not directly dependent on the size of the heart. We have studied only 3 patients with aortic valve disease and 2 patients with significant mitral stenosis, though King (1973) has published a B-scan from one further patient with mitral stenosis. In all these 6 patients the outflow tract measurement and the O/I ratio are in the expected normal range.

The narrowed outflow tract and the anterior position of the mitral valve can be detected as readily by careful time motion techniques as by B-scanning. The standard echocardiographic cardiac sweep (Fig. 5) shows this anterior position and confirms the close approximation of the mitral valve to the thickened septum. Comparison with a

cardiac sweep of a normal mitral valve (Fig. 6) emphasizes the abnormal mitral valve position in the left ventricle of a patient with idiopathic hypertrophic subaortic stenosis. Detection of an abnormally anterior position of the mitral valve can be helpful when other echocardiographic signs of idiopathic hypertrophic subaortic stenosis are equivocal.

The present B-scan study suggests that the left ventricular outflow tract is significantly narrowed throughout the cardiac cycle with only small increases in width during diastole. Examination of a standard mitral valve echogram from a patient with idiopathic hypertrophic subaortic stenosis (Fig. 7) confirms the minimal difference in end-systolic and end-diastolic positions of the anterior leaflet. The marked outflow tract narrowing during mid-systole was not recorded with B-scanning techniques in the 2 patients studied at this time in the cardiac cycle. In contrast, real time sector scans

have shown systolic anterior movement of the tip of the anterior leaflet (Henry *et al.*, 1975). The B-scan can measure the outflow tract in only 1 plane. It is possible that a scan in a perpendicular plane might have shown more significant outflow tract narrowing during systole. B-scanning records reflection of ultrasound from the main body of the anterior mitral leaflet, and perhaps may not record the motion of that part of the leaflet showing systolic anterior movement on standard echocardiograms; better resolution may make this apparent in B-scans.

Recent observations (King *et al.*, 1974; Rossen *et al.*, 1974) have questioned the central importance of the systolic anterior movement in the generation of the obstruction. In several patients simultaneous recording of the echocardiogram and left ventricular and aortic pressures has shown no gradient at a time when an obvious systolic anterior movement was present (King *et al.*, 1974; Rossen *et al.*, 1974). Furthermore, the systolic anterior movement may disappear before end-systole at a time when haemodynamic evidence of obstruction persists (Pierce *et al.*, 1964). In only one study has it been shown to persist to end-systole (Pridie *et al.*, 1972). Angiography has usually shown the systolic movement of the mitral valve leaflets anteriorly into the left ventricular outflow tract and the maintenance of this position until the onset of diastole (Simon *et al.*, 1967; Adelman *et al.*, 1969; Simon, 1972), though one study reported radiolucent structures appearing only transiently in the outflow tract in mid-systole in some patients (Simon *et al.*, 1967). Thus, the precise cause of the outflow tract obstruction is not apparent. It is possible that the anterior anatomical position of the mitral leaflet now shown by both B-scans and real time sector scans (Henry *et al.*, 1975) contributes to the critical narrowing of the outflow tract and is in part responsible for the pressure gradient. However, this would not explain the dynamic nature of the obstruction. Multiple systolic echoes as seen in Fig. 7 may indicate that different portions of the leaflet are drawn varying distances into the outflow tract, perhaps resulting from a Venturi effect (Wigle, Adelman, and Silver, 1971). Perhaps obstruction is determined by simultaneous narrowing of the outflow tract at several points.

B-scan ultrasonography has enabled us to examine the complex geometrical and anatomical relations in idiopathic hypertrophic subaortic stenosis and thus gain further insight into the pathophysiology of this disorder. The main body of the anterior mitral leaflet appears to be displaced anteriorly; this observation has made possible the definition of additional diagnostic criteria applicable to standard

time motion echocardiography. The anterior position probably contributes to the outflow tract obstruction. The precise relation of the abnormal leaflet position to the previously defined systolic anterior movement and the relative haemodynamic significance of each remain to be determined.

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