ECHOCARDIOGRAPHIC ASSESSMENT OF CONGENITAL MITRAL STENOSIS

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Echocardiographic assessment of congenital mitral stenosis

To assess the severity and precise anatomy of congenital mitral stenosis (MS), 17 patients with congenital left ventricular inflow obstruction were studied by M-mode and two-dimensional echocardiography (2DE) and by cardiac catheterization. In six patients MS was an isolated lesion and in 11 it was combined with other cardiovascular malformations. The diagnosis was confirmed at operation or autopsy in 15 patients. Twenty normal subjects of the same age and sex were selected as controls. M-mode amplitude and speed of diastolic closure (E-F slope) of the anterior mitral valve leaflet were determined in all patients. Mitral valve areas were traced after careful short-axis 2DE scans in 15 patients. Supravalvar, valvar, or subvalvar obstruction was evaluated in patients with surgical or autopsy documentation. Analysis of M-mode echocardiograms showed a reduction of E-F slope in all patients compared to normal control subjects but a poor correlation between E-F slope and hemodynamic data (mitral valve areas or pressure gradients). Diastolic fluttering of either or both mitral valve leaflets was found in 12 patients. It is concluded that M-mode echocardiography may be useful for qualitative assessment of congenital MS, even in the presence of associated heart defects, but less useful in evaluating its severity. Analysis of 2DE revealed good correlation between mitral valve areas as calculated with 2DE and with the Gorlin formula at cardiac catheterization, despite the complexity of the congenital mitral lesion. Anatomic varieties of congenital left ventricular inflow obstruction, such as stenosing supravalvar mitral ring or parachute deformity of the mitral valve, were recognized at 2DE. It is concluded that 2DE may be useful in estimating the severity of congenital MS and determining the site of the inflow obstruction. (Am Heart J 108:523, 1984.)

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Congenital mitral stenosis (MS) is a cardiac malformation that occurs in about 0.8% of patients with congenital heart disease.[1] It is often unrecognized but its diagnosis and evaluation are important for both the cardiologist and surgeon, especially in the presence of associated heart defects.[2-4] Previous studies[5-8] suggested the possibility of diagnosing congenital MS even when it is combined with other cardiac malformations, but some difficulty was shown in delineating anatomic patterns of the mitral lesion or estimating its severity by means of M-mode echocardiography.

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Table I. Echocardiographic and hemodynamic data in patients with congenital MS and 20 normal subjects of the same age

		Echocardiographic data)					
Patient No.	*Age/Sex*	*CE (mm)*	*DE (mm)*	*EF (mm/sec)*	*LA (mm)*	*(mm/m²)*	*MVA (cm²)*
1	2/12/M	12	9	20	20	45	
2	3/12/F	15	11	16	22	63	0.8
3	6/12/F	21	10	29	18	34	1.8
4	7/12/M	17	12	35	17	25	2.7
5	8/12/M	9	7	18	20	41	1.5
6	1/F	10	7	15	23	39	0.9
7	2/M	14	10	14	26	49	1.4
8	2/F	16	12	17	28	50	1.8
9	5/M	20	15	40	23	25	3.2
10	6/M	14	11	25	25	29	
11	9/M	20	14	18	42	44	1.9
12	12/M	17	14	20	50	41	1.7
13	13/F	15	10	16	45	36	1.6
14	16/F	24	19	30	45	32	2.5
15	19/F	21	16	25	55	37	1.9
16	20/F	18	15	25	52	45	2.0
17	23/M	16	13	20	56	44	1.7
Control group (20 subjects)		16-30 (mean 22.3)	13-25 (mean 17.2)	60-180 (mean 114.8)	15-42 (mean (27.8)	16-31 (mean 23.4)	2.9-6.4 (mean 4.3)

Abbreviations: ASD II = atrial septal defect (ostium secundum type); CE = total amplitude of anterior mitral leaflet; CoA = coarctation of aorta; DE = early diastolic amplitude of anterior mitral leaflet; DORV = double-outlet right ventricle; ΔP = diastolic mitral valvar pressure gradient; EF = speed of diastolic closure of anterior mitral leaflet; LA (echo) = left atrial dimension; LA (cath) = mean left atrial pressure; LV = left ventricular peak systolic pressure; MVA = mitral valve area; PA = main pulmonary artery pressure; PM = papillary muscles; Qp = pulmonary blood flow; Qs = systemic blood flow; TGA = transposition of the great arteries; wedge = pulmonary artery wedge pressure.
*No autopsy or surgery documentation.

The present investigation was planned to establish: (1) if the echocardiographic criteria for assessing acquired MS apply for congenital MS; (2) if some echocardiographically detectable pathologic aspects allow the ability to distinguish between congenital and acquired types; and (3) if two-dimensional echocardiography (2DE) has the advantage of more detailed assessment of both the severity and anatomy of congenital stenosis compared to the M-mode technique.

METHODS

Patient population. The study population consisted of 17 patients, ranging in age between 2 months and 23 years, observed during the period 1978 to 1982. All of them underwent cardiac catheterization and angiocardiography. In six of them MS was an isolated lesion; in 11 patients the following cardiac defects were present: coarctation of the aorta (six patients), double-outlet right ventricle (two patients), d-transposition of the great arteries (one patient), single ventricle (one patient), and atrial septal defect (one patient).

Patients with cor triatriatum or forms of hypoplastic left heart were excluded from the study. Ten patients underwent mitral valve surgery (valvotomy or replacement). Autopsy was performed on five patients who did not have surgical repair. None of the patients had a history of rheumatic heart disease. Congenital etiology was suggested on the basis of an early diagnosis of heart disease in the first years of life, even in cases of isolated MS. Twenty normal subjects, of the same age and sex, were selected as controls.

Equipment. All echocardiograms were performed within 4 weeks before or after cardiac catheterization. These studies were done with commercially available Ekoline or ATL instruments. A variety of transducers from 2.5 to 5 MHz were used for M-mode recording. 2DEs were obtained with the use of a mechanical sector scanner with oscillating or rotating elements at a scan rate of 30 or 80 frames/sec. The field of view was 80 degrees in an azimuthal direction and 15 or 21 cm deep. The range resolution was 1.5 to 2 mm. Cross-sectional images could be recorded on 1-inch reel-to-reel videotape and were available for analysis on an oscilloscope in real-time, slow motion, or single-frame format.

Analysis of M-mode echogram. The following measurements were obtained: left atrial size, measured at end systole from the leading edge of the posterior aortic wall to the leading edge of the left atrial posterior wall (mm/m²); total amplitude (C-E), early diastolic amplitude (D-E), and speed of diastolic closure (E-F slope) of the anterior mitral leaflet. The last measurement was determined only if the early diastolic passive closure of the valve was

Hemodynamic data								
Qp/Qs	*PA (mm Hg)*	*Wedge (mm Hg)*	*LA (mm Hg)*	*LV (mm Hg)*	*ΔP max*	*MVA (cm^2)*	*Mitral anomaly*	*Additional defects*
1:1	56/26 $\overline{35}$			120			Supravalvar ring, valvar thickening	
3:1	79/21 $\overline{44}$					1.2	Valvar thickening	Preductal CoA + VSD
1:1	57/27 $\overline{38}$	25	$\overline{28}$	135	19	2.3	Anomalous PM and chordae	
1:1	37/17 $\overline{26}$	19	$\overline{16}$	130	20	3.1	Anomalous PM and chordae	d-TGA
2:1	80/52 $\overline{64}$				32	1.7	Valvar thickening	Single ventricle
1:1	60/27 $\overline{38}$	26			45	0.7	Anomalous PM, valvar thickening	
1:1	77/49 $\overline{61}$		$\overline{32}$	148	40	2.2	Valvar thickening	Postductal CoA
1.7:1	46/28 $\overline{32}$	20	$\overline{30}$	150	35	1.6	Anomalous PM and chordae	DORV
1:1	37/16 $\overline{26}$	16	$\overline{18}$	118	9	3.6	Supravalvar ring, anomalous PM	
1:1	28/15 $\overline{19}$		$\overline{35}$	140	30		Anomalous PM* and chordae	
1.5:1	44/18 $\overline{27}$	17	$\overline{24}$	135	36	2.1	Valvar thickening	DORV
1:1	51/21 $\overline{31}$	21	$\overline{45}$	160	31	1.9	Supravalvar ring, valvar thickening	Postductal CoA
1:1	55/32 $\overline{40}$					1.8	Anomalous PM, * valvar thickening	
1:1	39/7 $\overline{18}$		$\overline{25}$	125	17	2.9	Anomalous PM and chordae	Postductal CoA
1:1	54/32 $\overline{37}$	23	$\overline{37}$	130	27	2.4	Anomalous PM and chordae	Postductal CoA
1.4:1	50/31 $\overline{35}$	20	$\overline{25}$	135	22	2.3	Anomalous PM and chordae	ASD II
1:1	55/30 $\overline{39}$	25	$\overline{35}$	130	30	1.6	Anomalous PM and chordae	Postductal CoA

clearly separated from the active closure due to ventricular systole. Since the A wave is not discernible in normal subjects with a fast heart rate, the E-F slope measurement was made only in subjects with a heart rate less than 120/min. Finally, the pattern of motion of posterior mitral leaflet was evaluated.

Analysis of 2DE. The present study was achieved by placing the transducer on the patient's chest in the third, fourth, or fifth intercostal space in a plane of scan perpendicular to the left ventricular long axis. With the sector scanner oriented in this way, after the transmitting power was varied for an optimized receiver gain setting, multiple short-axis scans were performed from the base of the heart toward the apex and various cross-sectional images were determined at different scanning levels. By performing this maneuver, serial images of the whole mitral apparatus were obtained including the number of papillary muscles underneath the valve. Echocardiograms of a mitral orifice were often visualized at a lower level than is normally seen in sequential cuts from the level of the upper part of the mitral valve leaflets to the region just above the papillary muscles. These scans were recorded on magnetic tape and the smallest orifice was selected for measurement. The single frame was stopped in early diastole, during the maximum valve opening (Fig. 1). The innermost margin of the echoes from the mitral orifice was traced on transparent film. A Hewlett-Packard light-pen computer system was used for planimetry on the basis of the recording calibration. In all cases measurements were obtained from five cycles average.

Cardiac catheterization. Right and left catheterization was performed (Seldinger technique) and different angiocardiographic projections were used according to different associated cardiac defects. The cardiac output was determined by the Fick oxygen method and the severity of MS was assessed (whenever possible) by the Gorlin formula.[9] When the mitral valve area was not reliable, as in complex congenital heart disease, only the mitral gradient was obtained by simultaneous measurements of left ventricular end-diastolic pressure and pulmonary wedge or left atrial pressure. The individual hemodynamic data were unknown to us at the time of the echocardiographic studies and their analyses.

Statistics. One-way analysis of variance and *t*-test were used to compare the mean values between groups. Linear regression analyses were performed by means of the least-squares method. The level of statistical significance for all analyses was $p < 0.05$.

RESULTS

Table I summarizes echocardiographic and hemodynamic data in patients and echocardiographic data in the control group.

M-mode data. Reduction of E-F slope was found in all patients compared to normal control subjects ($p < 0.001$). There was a poor correlation between the E-F slope of the anterior mitral leaflet and the mitral valve area obtained at cardiac catheterization by the Gorlin formula ($r = 0.42$; $p < 0.05$). Also mitral gradient and E-F slope were poorly correlated ($r = 0.44$; $p < 0.05$). No correlation was shown between E-F slope and pulmonary wedge pressure. A significant reduction of C-E and D-E amplitude

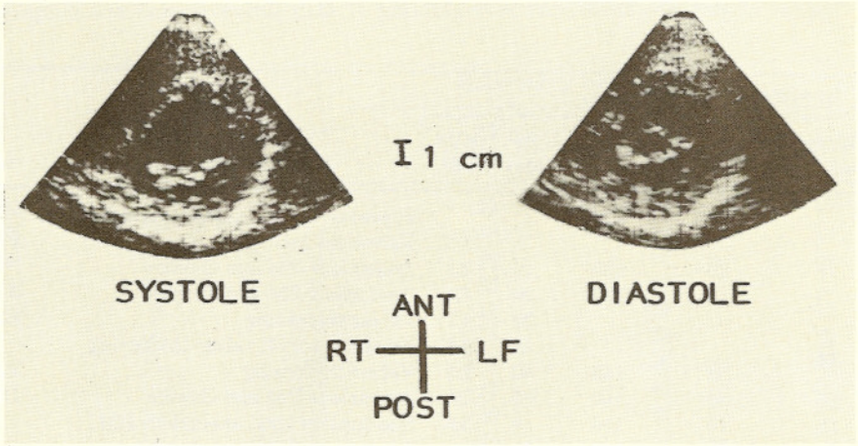

Fig. 1. Two-dimensional short-axis systolic and early diastolic images from a patient with congenital MS caused by accessory valvar tissue (autopsy documentation). Thickening of both mitral leaflets is shown. On the right frame, the mitral valve area can be drawn (see text).

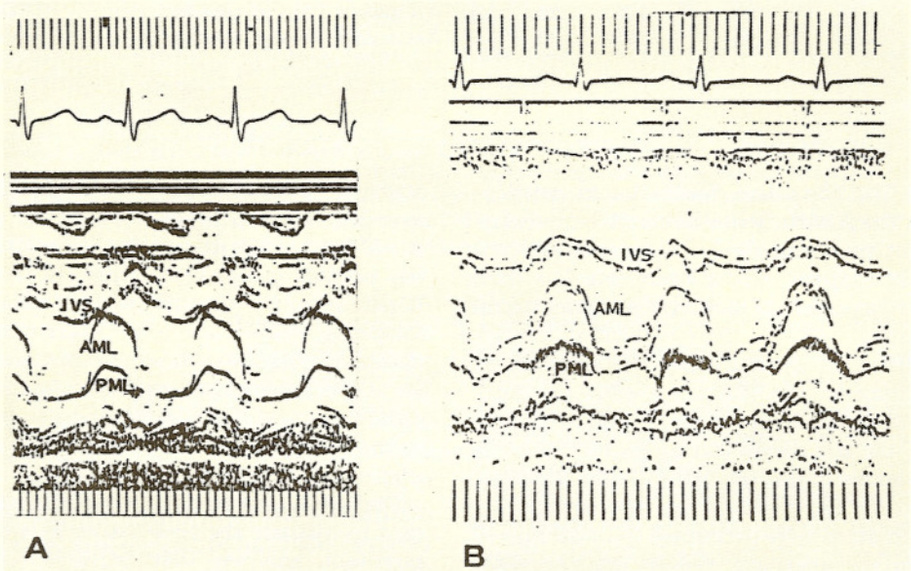

Fig. 2. M-mode mitral echograms from two patients with congenital left ventricular inflow obstruction. E-F slope is reduced and the posterior leaflet moves anteriorly in early diastole. *A*, Fine diastolic fluttering of the anterior mitral leaflet in a patient with valvar stenosis. *B*, Fine diastolic fluttering of the posterior mitral leaflet in a patient with verified supravalvar obstructing mitral ring. *AML* = anterior mitral leaflet; *IVS* = interventricular septum; *PML* = posterior mitral leaflet.

was present compared to normal control subjects ($p < 0.01$), but this reduction did not correlate with the transvalvar pressure gradient. Although left atrial size was larger in patients compared to control subjects ($p < 0.01$), no significant correlation was found between left atrial dimension and, respectively, mitral gradient and mitral area by Gorlin.

In 12 patients the posterior leaflet moved anteriorly in diastole and followed the anterior leaflet motion (paradoxic motion); in 11 patients diastolic high-frequency fluttering of either leaflet (Fig. 2) was present (in the absence of aortic regurgitation). Similar diastolic vibrations of leaflets, although of low amplitude, were also seen in 2 of the 20 normal control subjects.

2DE data. In 15 examined patients (Fig. 3) there was good correlation between the 2DE valve area and the Gorlin area ($r = 0.89$; $p < 0.001$; $s = 0.25$ cm^2). In two patients (Nos. 1 and 10, Table I) it was not possible to calculate the valve area because of unclear visualization of the mitral orifice in one of them and a not well-defined supravalvar obstruction

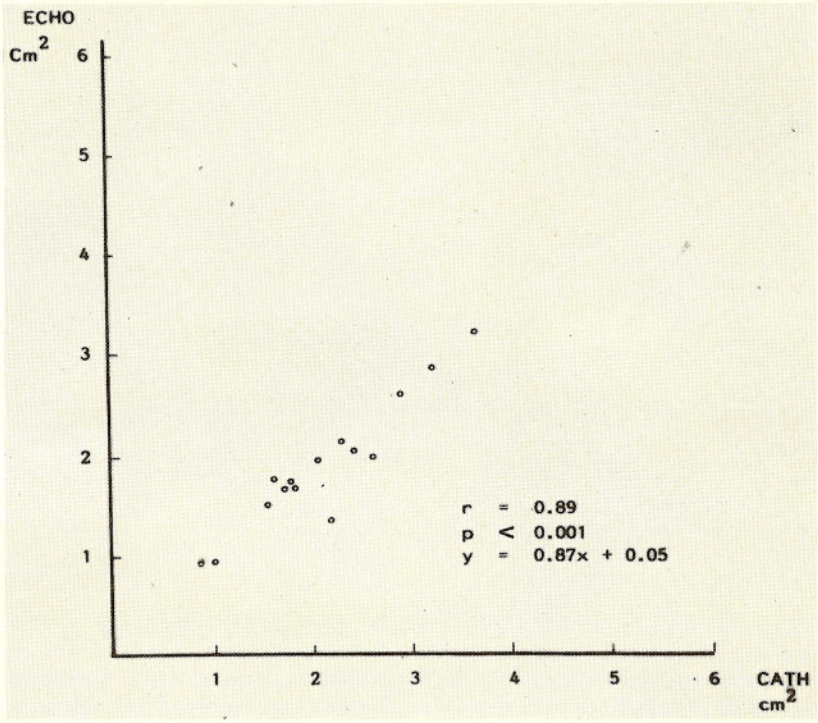

Fig. 3. Graph depicting the relation between mitral valve areas obtained by 2DE and cardiac catheterization in 15 patients with congenital MS.

in the latter. Mitral area was reduced in patients compared to normal control subjects ($p < 0.01$). A significant correlation was found between the 2DE mitral valve area and the transvalvar pressure gradient ($r = 0.86$; $p < 0.01$).

Review of echocardiograms of 15 patients with MS and surgical or autopsy documentation showed how 2DE can better define the anatomic obstruction at supravalvar and subvalvar levels. An anomalous echo stretching across the left atrium, presumably representing a left atrial membrane, was imaged in four-chamber apical views (Fig. 4) in three patients. By parasternal or subcostal views (Figs. 5 and 6), nine patients were assessed as having two papillary muscles and six as having one. M-mode echocardiography often did not allow the detection of supravalvar membrane or recognize papillary muscle anomalies.

DISCUSSION

The most widely accepted echocardiographic criterion for the diagnosis of MS, a reduction of diastolic E-F slope, was introduced by Edler,[10] in 1955. This sign has been critically reassessed[11, 12] and the clinical importance of recording an anterior diastolic motion of the posterior mitral leaflet in the evaluation of MS, in addition to the criterion of leaflet thickening, was pointed out. As far as congenital MS is concerned, after the first description by Lundström,[5, 6] it has been shown[7] how M-mode echocardiography allows qualitative judgment of MS, even in the presence of associated malformations, although it does not permit quantification of its severity.

2DE has played an important role in the assessment of acquired MS. The measurement of the stenotic mitral valve orifice has been determined by Henry et al.[13] and others,[14-16] who found good correlation between data obtained by this technique and perioperative measurements. In congenital MS there is little information regarding 2DE features.[17, 18] In the present study, we attempted to verify if 2DE enabled the ability to establish quantitative evaluation and define different varieties of congenital MS, anatomically more complex compared to the acquired type.

Anatomic varieties of congenital MS. Congenital MS may occur at different levels and involve single structures of mitral valve apparatus or a combination of them. Determination of the level of left ventricular inflow tract obstruction by M-mode echocardiography has been controversial[7] or denied.[8] As far as supravalvar obstruction is concerned, while we could differentiate patients with valvar forms of MS from patients with supravalvar obstructions, it was difficult to distinguish patients with supravalvar mitral ring from patients with cor triatriatum.[19-21] Pathologically,[20] with supravalvar ring the

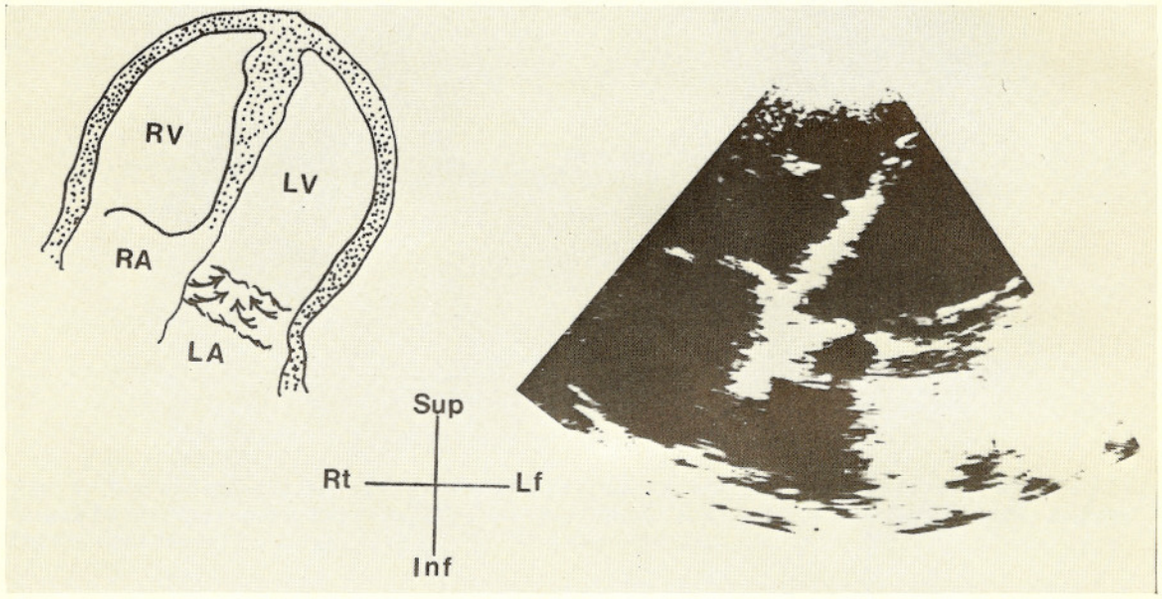

Fig. 4. Apical four-chamber echocardiogram from the same patient as in Fig. 2, *B*, with obstructing supravalvar mitral ring. A dividing membrane in the left atrium is shown. Arrows in the accessory chamber *(left)* indicate the turbulent blood flow that can be assumed to cause diastolic fluttering of the posterior mitral leaflet shown in Fig. 2, *B*. *LA* = left atrium; *LV* = left ventricle; *RA* = right atrium; *RV* = right ventricle.

left appendage and foramen ovale are located over the atrial membrane, while in cor triatriatum they are located underneath the membrane in the distal chamber. The pulmonary veins (all or part of them according to the anatomic varieties) come into the proximal accessory chamber.

In real-time 2DE, the echoes from the supravalvar mitral ring appeared moving toward the mitral valve leaflets in diastole and away from the mitral ring in systole. The attachments of the membrane were difficult to image; however, in the apical four-chamber view (Fig. 4) the membrane was positioned in a nearly horizontal plane adjacent to the mitral valve ring, extending to the right in the region of the atrial septum primum and to the left to the lateral left atrial wall. In cor triatriatum, the position and phasic motion of the atrial membrane are similar. It was suggested[7] that cor triatriatum might be distinguished from the supravalvar mitral ring by the absence of MS and the presence of a normal mitral valve echogram. "Pseudo" MS may appear to be present on echocardiographic findings if the transvalvar flow is reduced from the presence of supravalvar obstruction. In these cases we should image the exact attachment of the membrane relative to the pulmonary veins and left atrial appendage, but in our experience the left appendage and fossa ovalis were difficult to identify even by subcostal approach.

In one case of supravalvar ring, together with a reduction of mitral E-F slope, we observed coarse diastolic fluttering of the posterior mitral leaflet (Fig. 2, *B*) that could be explained by blood flow turbulence created in the accessory chamber. This was a suggestive but not specific finding, as it can also be present in normal subjects.

A pure valvar type of MS was not frequent in our series (Table I). It may be produced[2] either by fusion of commissures (with a similar appearance to the rheumatic form) or by accessory valvar tissue, adherent to the anterior or posterior mitral valve leaflet and creating an orifice obstruction. In two patients we could appreciate gross thickening of the valve leaflets, presumably due to accessory tissue. A thickened, stiffly moving mitral valve was imaged in all cardiac planes. On a scan from the aortic root to the apex in the short-axis plane (Fig. 1), the mitral leaflets were grossly thickened and the mitral orifice was small.

In six patients the anomaly of the mitral valve apparatus consisted of a single papillary muscle over which the chordae tendineae of both mitral leaflets had a convergent insertion, giving the appearance of a "parachute" mitral valve. The short-axis (Fig. 5) and subcostal four-chamber views (Fig. 6) were the most reliable for determining the number of papillary muscles, as both anterolateral and posteromedial muscles could be displayed. A single papillary muscle was often seen, the anterolateral one being absent in many patients, but a variable array is possible. In a few patients two papillary muscles were present, the chordae from the mitral leaflets

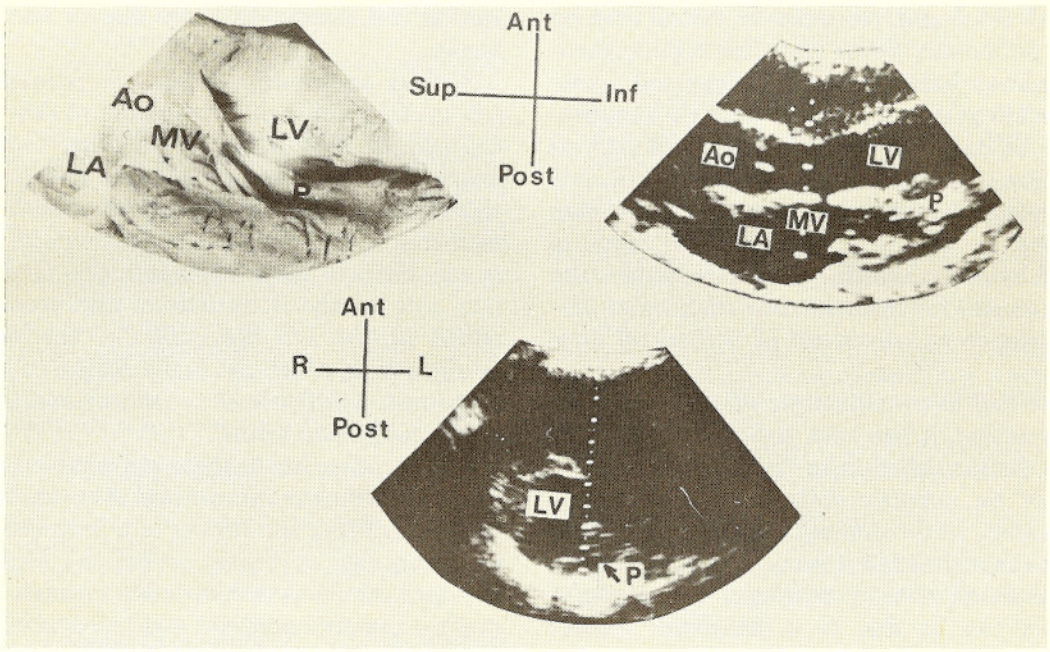

Fig. 5. Two-dimensional short-axis and long-axis views of the left ventricle *(middle and right panels)* and pathologic specimen *(left panel)* in a patient with a parachute deformity of the mitral valve caused by one large papillary muscle, centrally placed in the left ventricle. On the long-axis view mitral valve and subvalvar regions appear dense, with reduced movement observed during diastole. *Ao* = aorta; *LA* = left atrium; *LV* = left ventricle; *MV* = mitral valve; *P* = papillary muscle.

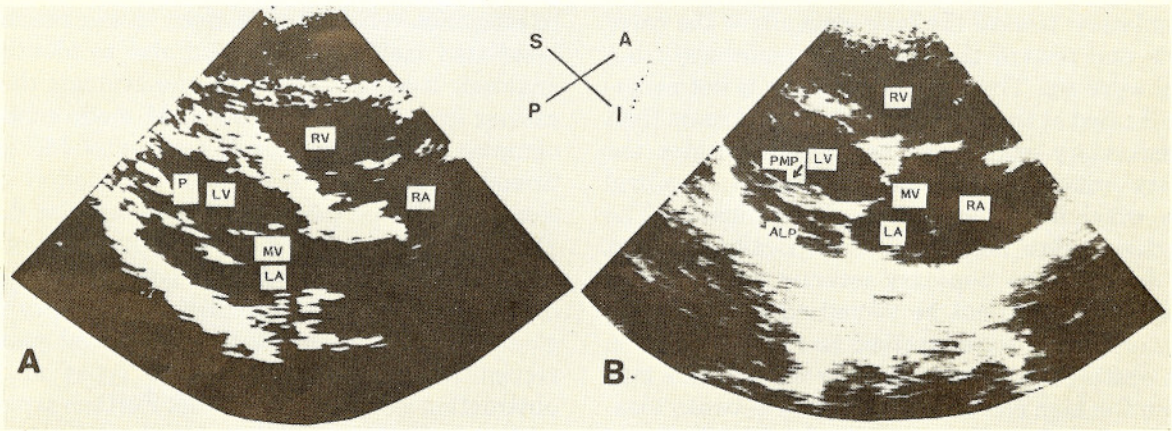

Fig. 6. Subcostal four-chamber view in a patient with *A*, A single papillary muscle and parachute mitral valve, and *B*, A patient with congenital MS and two papillary muscles. *ALP* = anterolateral papillary muscle; *LA* = left atrium; *LV* = left ventricle; *MV* = mitral valve; *P* = papillary muscle; *PMP* = posteromedial papillary muscle; *RA* = right atrium; *RV* = right ventricle.

inserting into only one. In all these cases the thickened and shortened chordae tendineae not only restricted the left ventricular inflow tract but also limited mitral leaflet motion, aggravating MS by a double mechanism.

In one of our patients parachute mitral valve was associated with complete transposition of the great arteries. Rosenquist et al.[22] recently described a spectrum of anomalies of the mitral valve in complete transposition, including some "formae frustae" of parachute mitral valve, characterized by a reduced space between papillary muscles. We have not observed such forms by echocardiography.

Value and limits of E-F slope. In all of our patients we found a reduction of the diastolic E-F slope compared to normal control subjects, either in patients with isolated MS or in those with associated cardiac malformations. Thus M-mode echocardiography can reveal the presence of congenital MS even in patients with a combination of anomalies, as described by Driscoll et al.[8] On the contrary, we found a poor correlation between the reduction of

diastolic E-F slope and, respectively, mitral area by Gorlin and pulmonary wedge pressure. A poor correlation was shown between the E-F slope and transmitral pressure gradient. Thus E-F slope measurements may be useful in serial follow-up of a given patient or for a qualitative assessment of MS, but they give a rough prediction of its severity. This may be explained by the fact that the E-F slope is determined by a combination of factors besides the pressure gradient, for instance, left ventricular diastolic compliance or movement of the mitral anulus.[12]

When the E-F slope reduction offers an ambiguous interpretation, it has been asserted that "true" MS can be distinguished from "pseudostenosis" on the basis of an anterior diastolic motion of the posterior leaflet. Unfortunately, this criterion is not valuable in congenital MS if we consider that in five patients we found normal motion of the posterior leaflet. This can be due to a somewhat greater mobility of the posterior mitral leaflet in congenital stenosis.

Value and limits of mitral area. Due to the complexity of congenital MS compared to the acquired form, and the possibility of a supravalvar, subvalvar, or mixed obstruction, a careful scan at different levels is mandatory: toward the base even above the valve leaflets and toward the apex even underneath the mitral valve tip. The main obstruction is not necessarily located at the valve leaflet level and more than one secondary orifice can be present besides the primary orifice, as a result of a tunnel of mitral tissue, which inserts into the papillary muscles. The secondary orifice may be asymmetrically situated, lying between the papillary muscles and chordae, and sometimes cannot be visualized by 2DE. The valvar orifice is always involved and restricted, even if the obstruction is not originally valvar. This may explain the high correlation we found between measurements of the mitral area obtained by 2DE and the Gorlin formula. The mitral orifice was imaged successfully in early diastole in 15 of 17 (88%) patients. Previous studies have shown up to 17% failure to obtain adequate echocardiographic images for quantitation of the mitral orifice in rheumatic valve stenosis.[15] Thus we believe the method can be reliable also in congenital mitral disease. Inadequate 2DE images for evaluation can be due to poor sound transmission qualities induced by abnormal chest wall configuration, patient age, and the presence of interposed lung. In individual cases where the mitral orifice cannot be adequately imaged, some other parameter, such as mitral anulus size,[18] might be evaluated, but no quantitative relation seems to exist between anulus size and the severity of congenital MS, except in cases due to annular hypoplasia.

Besides anatomic reasons, the reliability of the echocardiographic measurements of the mitral orifice can be affected by some other factors. False images can be caused by changes in the transducer angulation, producing distortions of both the orifice diameter and its rim. The resolution and line density capability of the instruments also play an important role in the definition of the contour of the mitral orifice. On the contrary, the presence of calcifications, which can make difficult a clear image of the mitral orifice in acquired stenosis, is quite rare in the congenital form.

On the other hand, there are some advantages of the echocardiographic method over the catheterization data. First, the former is a harmless, noninvasive, and easy to repeat technique. Moreover, the Gorlin formula is a hydraulic formula (it always needs a pressure gradient and transvalvar flow) and does not measure the valve orifice, per se. Finally, this formula does not give an evaluation of the mitral area in the presence of valvular regurgitation[9,13] when only forward output is known.

Implications. Clinically, it is important to distinguish congenital MS from the acquired form in order to recognize valve stenosis, even in the presence of associated malformations, and also to identify the anatomic level of obstruction and evaluate the indications for surgery. The role of M-mode echocardiography in the diagnosis of congenital MS is confirmed by our data, that show a reduction of E-F slope even in the presence of other cardiac defects. 2DE, despite some limitations due to both the methodology and the complex nature of the mitral disease, enables us to quantify the severity of the stenosis by the calculation of the mitral area. The recognition of various anatomic types of inflow obstruction can also be achieved. Further experience with these lesions and future refinement in image processing will allow more accurate diagnosis of these abnormalities.

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