Systolic anterior motion of the posterior mitral leaflet: a previously unrecognized cause of dynamic subaortic obstruction in patients with hypertrophic cardiomyopathy

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ABSTRACT Dynamic obstruction to left ventricular outflow in patients with hypertrophic cardiomyopathy usually occurs when the anterior mitral leaflet moves forward in systole and approaches or contacts the ventricular septum. However, we have recently identified, by M mode and two-dimensional echocardiography, 21 patients with hypertrophic cardiomyopathy who had a unique pattern of mitral valve motion characterized by abnormal mitral valve coaptation and systolic anterior motion of the posterior mitral leaflet. This abnormality of mitral valve motion was most reliably identified with two-dimensional echocardiography in views of the left ventricle obtained from the apex. At end-diastole the anterior and posterior mitral leaflets did not appear to coapt at their distal free margins. Rather, at mitral valve closure, the anterior mitral leaflet contacted the basal portion of posterior mitral leaflet. Subsequently, during systole the "residual" distal portion of posterior mitral leaflet approached or contacted the ventricular septum. Morphologic observations in nine other patients with hypertrophic cardiomyopathy suggested that systolic anterior motion of the posterior mitral leaflet is due to elongation of the middle scallop of the posterior leaflet, which probably comes into apposition with the ventricular septum during systole by passing through the space created by the normal pattern of chordal attachments onto the anterior mitral leaflet. Of the 16 patients who underwent cardiac catheterization, nine had basal subaortic gradients of 20 to 85 mm Hg, which were apparently due to moderate or marked systolic anterior motion of the posterior mitral leaflet. Ventricular septal myotomy-myectomies were performed in two patients and resulted in markedly diminished systolic anterior motion of the posterior mitral leaflet in each and abolition of subaortic gradient in the one patient who underwent postoperative cardiac catheterization. Hence, in patients with hypertrophic cardiomyopathy, systolic anterior motion of the posterior mitral leaflet (1) is not uncommon (identifiable in about 10% of a consecutively studied series of patients), (2) constitutes a previously undescribed mechanism for dynamic subaortic obstruction, and (3) is due to a malformation of the posterior mitral leaflet.


A SUBSTANTIAL PROPORTION of patients with hypertrophic cardiomyopathy demonstrate obstruction to left ventricular outflow. Most available angiographic and echocardiographic data appear to indicate that this dynamic subaortic obstruction occurs when the anterior mitral leaflet (AML) (with or without its attached chordae tendineae) approaches or comes into contact with the ventricular septal endocardium. Others have suggested that the chordae tendineae or papillary muscles may primarily produce left ventricular outflow obstruction. However, the role played by the posterior mitral leaflet (PML) in this obstructive process has not been defined. In this regard, we have identified a subgroup of patients with hypertrophic cardiomyopathy in whom the PML showed systolic anterior motion (SAM) that appeared to be responsible for subaortic obstruction.

Methods

Selection of patients. Between July 1979 and April 1980, 150 consecutively studied patients with a technically satisfactory two-dimensional echocardiogram were diagnosed at the National Heart, Lung and Blood Institute as having hypertrophic cardiomyopathy. In each patient, the diagnosis of hypertrophic cardiomyopathy was established by the characteristic clinical findings and the echocardiographic demonstration of a hypertrophied and nondilated left ventricle in the absence of...
another cardiac or systemic disease that could produce left ventricular hypertrophy. 29-31

Of the 150 patients with hypertrophic cardiomyopathy, 77 had SAM of the mitral valve that was evident on their M mode echocardiograms. In nine (12%) of these 77 patients, two-dimensional echocardiography demonstrated that this SAM was produced by the PML.

In addition, between May 1980 and May 1982, another 12 patients with hypertrophic cardiomyopathy were identified as having SAM of the PML by two-dimensional echocardiography and were added to the patient group. Therefore, the overall study group comprised 21 patients with hypertrophic cardiomyopathy and SAM of the PML.

Patient characteristics. The 21 study patients ranged in age from 9 to 50 years (mean 28); 13 (62%) were male patients. Nine patients were asymptomatic, the activities of five were mildly limited by their symptoms (New York Heart Association class II), and the activities of seven were moderately to severely limited (classes III and IV). The symptoms most commonly experienced were dyspnea on exertion and fatigue (seven patients), lightheadedness (six patients), and palpitations (five patients). Fifteen patients were taking cardiac medications at the time of the echocardiographic studies (propranolol in nine and verapamil in six).

The two other groups of patients were studied by echocardiography and compared with the patients with hypertrophic cardiomyopathy and SAM of the PML. One control group consisted of 48 subjects in whom there was no evidence of heart disease; their ages ranged from 20 to 50 years (mean 24) and 15 (60%) were male patients. The second group consisted of 137 patients with cardiac diseases other than hypertrophic cardiomyopathy, including coronary heart disease (51 patients), aortic valve disease (48 patients), congenital heart malformations (18 patients), mitral valve disease other than prolapse (13 patients), and dilated cardiomyopathy (seven patients). Ages ranged from 2 to 73 years (mean 45); 102 (75%) were male patients.

Hemodynamic studies. Cardiac catheterization was performed in 16 of the 21 patients with hypertrophic cardiomyopathy and SAM of the PML after cardioactive medications were discontinued. Left ventricular outflow tract gradients of 20 to 85 mm Hg were present under basal conditions in nine patients, including five with gradients of 40 mm Hg or more. In six of the nine patients with basal subaortic obstruction, higher gradients of 40 to 110 mm Hg were produced by provocative intervention (i.e., isoproterenol infusion, Valsalva maneuver, or amyl nitrite inhalation).

Seven other patients had small (5 mm Hg) or no left ventricular outflow gradient under basal conditions. Five of these seven patients had provoked gradients of 30 to 60 mm Hg, one had no gradient with provocation, and the remaining patient underwent no provocative maneuvers.

M mode echocardiography. M mode echocardiograms were performed with an IREX System II Ultrasound unit with a 2.25 MHz unfocused transducer 1.3 cm in diameter. Maximal thickness of the ventricular septum was measured just before atrial systole, with the ultrasound beam directed either through or slightly below the mitral leaflets. 29, 31 Thickness of the posterior left ventricular free wall was measured during the same phase of the cardiac cycle, with the ultrasound beam passing through the mitral leaflets.

Position of the mitral valve in the left ventricular cavity was assessed at the point of closure of the valve leaflets. Mitral valve position index was calculated by dividing the distance between mitral valve and posterior free wall endocardium by the distance between mitral valve and ventricular septal endocardium. 18

Other M mode echocardiographic parameters were measured in accordance with the recommendations of the American Society of Echocardiography. 32 When appropriate, echocardiographic dimensions were adjusted for body surface area and compared with normal standards. 33

SAM of the mitral valve was evaluated in a semiquantitative fashion. SAM was defined as mild if the minimal mitral-septal distance was greater than 10 mm; moderate, if this distance was 10 mm or less but there was no mitral-septal contact; and marked if there was either brief or prolonged (greater than 30% of echocardiographic systole) mitral-septal contact.

Two-dimensional echocardiography. A Varian (V-3400) real-time, phased-array, ultrasonic sector scanner with a handheld 2.25 MHz transducer was used to perform the two-dimensional echocardiographic studies. The field of view is 80 degrees in an azimuthal direction and 15 or 21 cm deep. Azimuthal resolution is 2 to 5 mm within the field of view and range resolution is 1.5 to 2 mm. Images were produced at 32 frames/sec and recorded on 1 inch (Sony) reel-to-reel videotape or an Ampex DR-10 rhodium-plated chromium disc for subsequent review in real-time, slow-motion, or stop-action modes. Single still-frame photographs were made from a television monitor with the use of a 35 mm camera.

Two-dimensional echocardiographic examination included the imaging of a number of cross-sectional planes through the heart. 34 Serial short-axis views of the left ventricle were obtained by initially orienting the sector plane perpendicular to the long axis of the left ventricle from a standard parasternal transducer location and slowly angling the image plane from aorta (cephalad) to apex (caudad). 35 The long-axis view was obtained from the same transducer placement by orienting the sector plane parallel to the longitudinal axis of the left ventricle, with care being taken to avoid improper angulation of the scan plane tangentially through the ventricle.

The apical four-chamber view was obtained with the transducer at the cardiac apex and the tomographic plane directed perpendicular to the ventricular and atrial septa and through the plane of the mitral and tricuspid valve orifices. This permitted simultaneous display of atria and ventricles and atrioventricular valves and cardiac septae. 36 The right anterior oblique equivalent (two-chamber) view of the left ventricle was obtained by rotating the transducer clockwise 60 to 90 degrees so that the ultrasound beam was in a plane approximately parallel to the ventricular septum, permitting simultaneous visualization of the aorta (and aortic valve), left ventricular outflow tract, and mitral valve leaflets. 34

Valvular motion is best appreciated by analysis of real-time (motion) video recordings. The freeze-frame photographs we use for illustration do not permit the visual integration that is possible with the real-time movie, and the quality of such static images is therefore unavoidably degraded optically compared with the dynamic real-time display. We also recognize the difficulty in reliably establishing the precise point of deceleration between the distal portions of mitral valve leaflet and the attached chordae tendineae. To aid in making this distinction (as well as that between the anterior and posterior leaflets), M mode recordings were made while the cardiac anatomy was being directly observed with two-dimensional echocardiography in the long-axis view. In addition, imaging the left ventricle in the right anterior oblique view from the apex was often helpful in discerning the proximal extent of the chordae from the distal portion of the mitral leaflets. Nevertheless, for the purposes of simplicity and clarity, we have referred to the adjacent portions of mitral valve leaflet and attached chordae tendineae as either the AML or PML.

The cross-sectional orifice area of the left ventricular outflow tract was assessed by a method described in detail elsewhere. 37 In brief, the outflow tract was outlined in the short-axis plane at the level of the mitral valve during diastole, and the cross-
sectional area was quantitated with a video planimetry system.

Four basic patterns of distribution of left ventricular hypertrophy were identified by two-dimensional echocardiography in the study patients. This classification has been reported in detail previously.31

Morphologic studies. The case records of the Pathology Branch and those of the Echocardiography Laboratory were reviewed and nine patients were identified in whom (1) the gross heart specimen (seven patients) or operatively excised mitral valve (two patients) were in suitable condition to permit examination and measurement of the mitral valve leaflets30 and (2) a technically adequate two-dimensional echocardiogram had been obtained while they were alive. None of these patients are included among the 21 patients who comprise the primary study group.

Results

Mitral valve motion

Two-dimensional echocardiography. An abnormality of mitral valve motion and coaptation was identified in each of the 21 study patients by two-dimensional echocardiography (figures 1 through 6).

In the apical view of the left ventricle (figures 1 through 3) it was evident that the distal margins of the AML and PML did not come into apposition at end-diastole in the normal fashion. Rather, the distal portion of the PML passed caudal to the anterior leaflet while the anterior leaflet coapted at or near the base of the posterior leaflet (figures 1 through 3). Hence, the distal portion of the PML did not participate in mitral valve coaptation. This “residual” posterior leaflet distal to the site of coaptation varied in length depending on precisely where the anterior leaflet made contact with the posterior leaflet. For example, when the AML coapted at the base of the posterior leaflet the residual portion of posterior leaflet was relatively long (figures 1 through 3). If the point of coaptation was closer to the
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FIGURE 2. Schematic illustrations of stop-frame images shown in figure 1. Abbreviations are as in figure 1.

FIGURE 3. Sequential stop-frame images obtained in the apical two-chamber view with the transducer rotated laterally to permit visualization of the aorta (Ao), aortic valve, and left ventricular outflow tract (LVOT). From the same patient shown in figure 1. A and B, during diastole; C, during the subsequent systole. A, The AML and PML approach each other. B, The mitral leaflets fail to coapt normally at their distal margins; at mitral closure, the anterior leaflet contacts the base of the posterior leaflet. C, The portion of posterior leaflet distal to the site of coaptation protrudes into the left ventricular outflow tract and approaches the ventricular septum (VS). LA = left atrium; LV = left ventricle; RV = right ventricle.
FIGURE 4. Sequential two-dimensional echocardiographic stop-frame images obtained during the same cardiac cycle in the parasternal long-axis view. A through C, during diastole; D, during the subsequent systole. Mitral valve motion corresponds to that shown in the apical views in figure 1. A, Mitral valve is wide open; the AML is in apposition to ventricular septum (VS) and the PML apposes the posterior free wall. B and C, The mitral leaflets approach each other. D, The anterior leaflet coaps at the base of the posterior leaflet and the free residual portion of the posterior leaflet comes into apposition with the ventricular septum.

Ao = aorta; LV = left ventricle; RV = right ventricle; PW = posterior free wall.

tip of the posterior leaflet, the residual portion of that leaflet was relatively short.

In early systole, the residual posterior leaflet continued its anterior movement and approached or contacted the ventricular septal endocardium during midsystole (figures 1 through 3). When imaged in the right anterior oblique equivalent view, it was evident that the PML projected into the left ventricular outflow tract during systole (figure 3). Recognition of SAM of the PML could usually be achieved in the conventional four-chamber view; however, it was often necessary to tilt or rotate the transducer slightly clockwise or counterclockwise in order to identify that segment of the PML that showed SAM.

In each study patient, the PML appeared to be elongated (in the base to apex plane); the length of the posterior leaflet was approximately equal to or greater than that of the anterior leaflet in each patient. In five of the 21 patients, the site of attachment of the PML to the posterobasal free wall appeared to be displaced toward the apex relative to the AML; in the other 16 patients the mitral leaflets were normally positioned with respect to each other.

Abnormal mitral valve coaptation and SAM of the PML similar to that observed in the apical views was also evident in the parasternal long-axis view (figures 4 and 6). The distal margins of posterior leaflet appeared to pass caudal to the tip of anterior leaflet and approach the ventricular septum in systole.

Analysis of the short-axis views confirmed and sup-
plicated findings obtained in the other tomographic planes. In the short-axis planes through the most basal portions of the left ventricle, both mitral leaflets could be imaged together and showed normal anatomic relationships and valvular motion. However, when the echocardiographic beam was directed more distally (to a level where the chordae tendineae were visualized) it was often possible to image the PML alone (figure 5).

At this level, the posterior leaflet was observed to move anteriorly during systole between the chordal bundles; the middle one-third of the leaflet appeared to most closely approach or contact the septum (figure 5).

SAM involving the PML (or AML) was absent in 177 of the 185 control subjects studied by echocardiography. The remaining eight subjects (two normal subjects and six with other cardiac diseases) showed SAM of the PML that was particularly trivial in magnitude — i.e., did not come within 18 mm of the ventricular septum during systole.

**M mode echocardiography.** It was also possible to identify SAM of the PML by M mode echocardiography in each study patient (figures 7 through 9). In diastole, the PML occupied a relatively posterior position in the left ventricular cavity and showed the normal peaks of anterior and posterior motion (i.e., E', F', and A') at appropriate times in the cardiac cycle. Diastolic excursion of the posterior leaflet was, however, often exaggerated.

At the onset of ventricular systole (just after the peak of the R wave on electrocardiogram) the PML moved abruptly anteriorly toward the ventricular septum, reaching its maximum forward excursion in midsystole, and then moved sharply posteriorly, returning to baseline position just before the end of systole (figure 8). Usually one or two notches, produced by rapid anterior and posterior systolic leaflet motion, were evident in the upstroke of the PML contour (figure 8).

The contour of the posterior leaflet on the echograms varied among the study patients. In nine patients the leaflet showed a rapid, relatively smooth upstroke and descent (figure 8); in the other 12 patients the initial rapid ascent was directly anterior, but as the

**FIGURE 5.** Two-dimensional echocardiographic stop-frame images obtained during late diastole (A) and systole (B) in the parasternal short-axis view. From the same patient shown in figure 4. A. At this cross-sectional level, the PML is imaged alone since the entire anterior leaflet is positioned more cephalad. B. At the onset of systole, the middle one-third of the posterior leaflet has moved anteriorly and appears to pass between bundles of chordae tendineae (dotted structures on schematic drawings). In subsequent systolic frames (not shown here) the posterior leaflet came into contact with the ventricular septum (VS). FW = free wall of left ventricle.

**FIGURE 6.** Two-dimensional echocardiographic stop-frame images from a 41-year-old man with hypertrophic cardiomyopathy in whom hypertrophy was primarily confined to the apical one-half of the left ventricle. A. In the long-axis view, mitral valve motion is shown during early systole (A). Magnitude of SAM of the PML is mild and left ventricular outflow tract dimension is normal; hence, the PML does not closely approach the ventricular septum (VS), accounting for the absence of subaortic obstruction under basal conditions. Solid lines B1, B2, and B3 denote sampling sites for the M mode echocardiograms shown in figure 7. B. Short-axis view at papillary muscle level shows hypertrophy of anterolateral free wall and posterior portion of septum, which can not be identified by M mode echocardiography or seen in the long-axis view. FW = free wall of left ventricle; LA = left atrium; RV = right ventricle.
leaflet approached the septum its velocity diminished and it assumed a “squared-off” appearance (figure 7). The magnitude and duration of SAM of the PML also varied greatly among individual patients and was mild in three (figure 7), moderate in seven, and severe in 11 (figure 8). Since the PML occupies a posterior position in the cavity during diastole, moderate-to-marked SAM of the PML represents an example of particularly exaggerated leaflet excursion.

In six study patients the residual portion of PML was particularly long and extended far caudal to the anterior leaflet. Therefore, it was possible to pass the M mode beam preferentially through the posterior leaflet and record its motion without imaging the anterior leaflet (figures 6 through 8). Verification that this isolated valve structure represented the posterior leaflet was obtained by scanning the M mode beam (either conventional or derived from the two-dimensional echocardiogram) until it was possible to identify continuity between the distal portion of posterior leaflet that was imaged alone and its more proximal segment (visualized with the anterior leaflet).

In 15 other patients the residual portion of the posterior leaflet was shorter and the distal margins of the two mitral leaflets were relatively close together. Hence, the posterior leaflet could not be selectively imaged without the anterior leaflet by M mode echocardiography. In such patients, the images of the mitral leaflets were superimposed and appeared to criss-cross each other at onset of systole (figure 9).

In seven patients M mode and two-dimensional echocardiograms were obtained during provocative maneuvers (i.e., Valsalva maneuver or amyl nitrite inhalation). A distinct increase in the magnitude and duration of SAM of the PML was evident with provocation in four of these seven patients, including two in whom the subaortic gradient had been shown at catheterization to be greater with provocation than under basal conditions. In the other three patients little or no change in SAM of the PML was observed with provocation.

Correlation of echocardiographic and hemodynamic findings. A relationship was apparent between the severity of the basal left ventricular outflow tract gradient and the magnitude and duration of SAM of the PML under basal conditions. In eight of nine patients with a gradient of 20 mm Hg or more at catheterization, SAM of the PML was marked — i.e., the PML came into contact with the ventricular septum during systole; in three of these patients mitral-septal contact was particularly prolonged. The other patient (with a basal subaortic gradient of 20 mm Hg) showed moder-ate SAM of the PML in which the posterior leaflet approached but did not contact the septum.

In seven other patients the basal subaortic gradient was 0 or 5 mm Hg. SAM of the PML was mild in four of these seven patients, moderate in two, and marked in one.

Postoperative echocardiographic findings. Two patients underwent ventricular septal myotomy-myectomy. Before surgery these patients had obstruction to left ventricular outflow (gradients of 40 and 50 mm Hg)

![Figure 7](image-url)
and their echocardiograms showed that they had marked SAM of the PML (figure 4). After surgery only a trivial degree of SAM of the PML was evident in each patient (figure 10). Cardiac catheterization performed 6 months after surgery in one of these patients showed no subaortic gradient under basal conditions and only a 5 mm Hg gradient with provocation; the other patient refused to undergo a postoperative catheterization.

Ventricular wall thicknesses and distribution of left ventricular hypertrophy. On M mode echocardiograms ventricular septal thicknesses ranged from 9 to 32 mm (mean 22) and posterior free wall thicknesses ranged from 9 to 21 mm (mean 13). Ventricular septal to free wall thickness ratios were 0.9 to 2.7 (mean 1.7).

Two-dimensional echocardiography demonstrated a wide variety of patterns of left ventricular hypertrophy. Most patients had hypertrophy of both the anterior and posterior portions of ventricular septum, but not of the left ventricular free wall (eight patients), or of both the septum and anterolateral free wall (seven patients). Three other patients had hypertrophy confined to only the anterior segment of septum and the remaining three patients showed involvement of portions of the left ventricle other than the basal anterior ventricular septum of the latter three patients, two had hypertrophy of the posterior septum and/or anterolateral free wall and one had septal and free wall hypertrophy confined to the apical one-half of the left ventricle (i.e., apical hypertrophic cardiomyopathy) (figure 6, B).

Other echocardiographic findings. In each of the 21 patients, the left ventricular internal dimension at end-diastole was either normal or reduced (range 30 to 49 mm, mean 38). Left atrial dimension was increased in 12 patients (45 to 60 mm) and normal in nine (30 to 38 mm). Premature systolic closure of the aortic valve was identified in 11 patients, absent in five, and could not be reliably assessed in five. No consistent relationship was evident between the magnitude and degree of SAM of the PML (or basal subaortic gradient) and either left atrial dimension or the presence of premature aortic valve closure.

Left ventricular outflow tract cross-sectional areas ranged from 1.7 to 8.8 cm² (mean 4.3) and each was reduced compared with normal. The small outflow tract area appeared to be largely due to the abnormal anterior position of the mitral valve in the left ventricular cavity (mitral valve position index of 0.3 to 1.1, mean 0.8; normal < 0.3).

Angiographic findings. Left ventricular angiograms were available for analysis in five patients, in-
cluding four with marked and one with moderate SAM of the PML on the two-dimensional echocardiogram. SAM of the mitral valve was identified in each of the three patients in whom an angiogram was obtained in the left anterior oblique projection. Mitral regurgitation (of a mild degree) was present in only one patient.

Morphologic observations. Of the nine patients studied morphologically, three had SAM of the PML that was evident on their two-dimensional echocardiograms. In each of these three patients the middle segment of the PML appeared elongated (in the cephalad-caudal axis) compared with the anterolateral and posteromedial segments (figure 11). The elongated segment measured 19, 21, and 24 mm from anulus attachment to leaflet tip (mean for normal valves, 13 mm), was approximately the same length or slightly longer than the anterior leaflet, and comprised about one-third the total width of the posterior leaflet (figure 11, a and b). In addition, when the mitral valve leaflets were positioned in their correct anatomic relationships, the elongated segment of the posterior leaflet extended distal to the caudal margins of the anterior leaflet and also was capable of passing through the V-shaped gap created by the normal pattern of chordal insertions onto the anterior leaflet (figure 11, c and d).

In each of the three patients, the elongated middle segment was thickened and had an indentation located parallel to the mitral anulus about two-thirds the distance from the anulus to the leaflet tip (figure 11, b); this groove appeared to correspond to the point of impact of the anterior leaflet tip on the posterior leaflet body during systole. Each of the remaining six patients had neither SAM of the PML nor an elongated segment of PML extending distal to the anterior leaflet.

Discussion

In this study we describe a newly recognized echocardiographic pattern of mitral valve motion in patients with hypertrophic cardiomyopathy in which the PML shows SAM. Furthermore, this SAM of the PML appears to occur not infrequently in patients with hypertrophic cardiomyopathy. In our consecutively studied series of patients with this disease and mitral SAM, about 10% showed SAM that was due to the posterior leaflet alone. The magnitude and duration of SAM of the PML varied considerably among our patients; in the majority, however, SAM was marked and was associated with left ventricular outflow tract systolic pressure gradients.

Some divergence of opinion exists among investigators regarding the precise mechanism by which subaortic gradients are produced in patients with obstructive hypertrophic cardiomyopathy. Most observers have concluded that the free edges of the mitral leaflets are primarily involved in this process and that obstruction to left ventricular outflow occurs at the site at which the AML contacts the ventricular septal endocardium. The PML also may appear to move anteriorly in synchrony with the AML, but does not itself contact the septum because of its posterior position.

In contrast, the type of mitral valve SAM described in this report differs distinctly from that previously described in hypertrophic cardiomyopathy (figure 12). In our patients, it was the PML that moved anteriorly in systole. Furthermore, each patient had a reduction in the cross-sectional area of the left ventricular outflow tract. Hence, it appears that the mechanism by which SAM of the PML occurs may be similar to that previously postulated for other patterns of mitral SAM. For example, the residual portion of the posterior leaflet may be pulled anteriorly toward the ventricular septum by the high-velocity jet of blood ejected through an anatomically narrow left ventricular outflow tract (i.e., a Venturi effect). In our experience, SAM of the PML is most reliably demonstrated with two-dimensional echocardiogra-

FIGURE 10. Two-dimensional echocardiographic stop-frame images obtained during late diastole (A) and early systole (B) in the parasternal long-axis view. From the same patient shown in figure 4, but 6 months after ventricular septal myotomy-myectomy. Site of operative excision of muscle from the anterior basal septum is evident (arrow) and the transverse dimension of the left ventricular outflow tract has been greatly increased by the operation. Magnitude of SAM of the PML shown here is trivial and much reduced when compared with the preoperative echocardiogram (compare with figure 4). Ao = aorta; C = chordae tendineae; LA = left atrium; LV = left ventricle; PW = posterior wall; RV = right ventricular wall; RVC = right ventricular cavity.
phy, particularly in the tomographic planes obtained from the left ventricular apex. However, it was also possible to identify SAM of the PML with M mode echocardiography. This was accomplished most convincingly in those patients in whom the posterior leaflet appeared displaced toward the apex relative to the anterior leaflet. In such patients this abnormality in mitral valve anatomy permitted the M mode beam to be directed preferentially through the posterior leaflet. SAM of the PML in such patients may be missed by conventional M mode echocardiography if care is not taken to scan the entire cephalad-caudal extent of the mitral valve apparatus. In other patients the distal margins of the two mitral leaflets were closer together, and an M mode echocardiographic image was created in which the posterior leaflet (showing SAM) and the anterior leaflet (showing no SAM) appeared to be superimposed (figures 9 and 12).

Our gross anatomic data, obtained in a separate group of patients with hypertrophic cardiomyopathy in whom the mitral valve could be studied morphologically, offer a probable structural basis for the occurrence of SAM of the PML. Each of these patients, who had SAM of the PML on their two-dimensional echocardiograms, showed a marked segmental elongation that was confined to the middle scallop of the PML; in addition, the free caudal margins of this abnormal portion of the posterior leaflet appeared to extend distal to the caudal margins of the AML. These findings have led us to postulate that it is the elongated scallop of the posterior leaflet that contacts the septum during systole by passing beyond the free edge of the anterior leaflet into the space created by the divergent sites of attachment of the chordae tendineae onto the anterior leaflet. Hence, our patients with SAM of the PML probably represent a subset of the overall population with hypertrophic cardiomyopathy in whom this particular malformation of the mitral valve occurs.

FIGURE 11. Mitral valve removed at operation from a 60-year-old woman with hypertrophic cardiomyopathy and mitral regurgitation. SAM of the PML was evident on two-dimensional echocardiogram. a, Valve is displayed so that AML (A) is between the broken lines and portions of PML (P) are seen to either side of the anterior leaflet. Elongated middle scallop (ES) of the PML is situated to the right of the anterior leaflet and between the two other normally appearing segments of posterior leaflet. b, Higher magnification view of the elongated middle scallop (ES) of posterior leaflet. The indentation or groove (arrows) located two-thirds of the distance from mitral anulus attachment to leaflet tip and parallel to the plane of the anulus was probably produced by impact of the distal margins of anterior leaflet on the middle scallop during systole. Four nontickened chordae tendineae insert onto the elongated scallop of posterior leaflet. c, Intact valve demonstrating anatomic relationships that permit the elongated scallop of the posterior leaflet to contact the septum during systole; the posterior leaflet is able to pass distal to the anterior leaflet through the V-shaped space created by the normal pattern and location of chordal insertions onto the anterior leaflet. d, Intact valve viewed from below, showing relation of elongated scallop of posterior leaflet (ES) to the anterior leaflet.
Segmental posterior leaflet elongation similar to that observed in our patients with hypertrophic cardiomyopathy appears to be uncommon in normal subjects or in patients with other heart diseases. In each of the 50 mitral valves examined by Ranganathan et al., the length of the AML exceeded that of all three scallops of the posterior leaflet. Furthermore, a gross anatomic analysis of PML morphology in 29 patients with coronary heart disease and mitral regurgitation identified no instance of a mitral leaflet abnormality similar to that which occurred in our patients with hypertrophic cardiomyopathy. In 60 patients with mitral valve prolapse who also demonstrated mitral regurgitation and one or more elongated scallops of the PML, either the AML was also elongated or an elongated middle scallop of the PML was present but buckled cephalad, which produced a relatively short mitral anulus-to-leaflet tip distance. Furthermore, the transverse groove in the middle scallop of the PML produced by the impact of the anterior leaflet that we observed in some study patients with hypertrophic cardiomyopathy was not present in patients with mitral valve prolapse.

Recognition of SAM of the PML may alter the operative approach in selected patients with hypertrophic cardiomyopathy and subaortic obstruction. This is a particularly pertinent consideration for those patients in whom the caudal margins of the posterior leaflet contact the ventricular septum at a point far distal to the anterior leaflet. In such patients, the longitudinal extent of the myotomy-myectomy should incorporate that region of septal muscle adjacent to the caudal margins of PML in order to include the site of subaortic obstruction.

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FIGURE 12. Schematic drawings of M mode echocardiograms showing normal pattern of mitral valve motion, compared with patterns of SAM identified in patients with hypertrophic cardiomyopathy. In each panel, the AML is depicted as a broken line and PML as a solid line. A. Normal subject without SAM. B. SAM of the type commonly observed in patients with hypertrophic cardiomyopathy. Both mitral leaflets move anteriorly in systole, but the anterior leaflet actually contacts the septum. C. SAM of the PML in a patient with hypertrophic cardiomyopathy, with the ultrasound beam directed preferentially through the posterior leaflet. D. SAM of PML in a patient with cardiomyopathy with ultrasound beam directed through both the AML (showing no SAM) and PML (showing SAM).

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