Ultrasound Localization of Left Ventricular Outflow Obstruction in Hypertrophic Obstructive Cardiomyopathy

By Pravin M. Shah, M.D., Raymond Gramiak, M.D., and David H. Kramer, M.D.

SUMMARY
Simultaneous recordings of reflected ultrasound from the anterior mitral leaflet and left ventricular outflow, the ECG, the phonocardiogram, and a recording of the carotid artery pulse were obtained in six patients with hypertrophic obstructive cardiomyopathy. Abnormal sharp systolic anterior movement (SAM) of the mitral leaflet was observed. This movement began with the onset of ventricular ejection and reached a peak with the initial peak in the arterial pulse. The leaflet was apposed to the interventricular septum up to 60% of the ejection period. In the latter part of systole as the mitral leaflet moved away from the interventricular septum, the arterial pulse showed a second systolic wave. Onset of SAM coincided with onset of the systolic murmur. Spontaneous variations in amplitude of SAM coincided with alterations in contour of the arterial pulse and in the intensity of the murmur. Administration of methoxamine to four patients resulted in disappearance of SAM. In one patient following surgery, the SAM of the mitral leaflet was noted only in the post-ectopic beats. This specific abnormality of mitral leaflet movement represents the localization of dynamic outflow obstruction in hypertrophic obstructive cardiomyopathy.

Additional Indexing Words:
Idiopathic hypertensive subaortic stenosis (IHSS)
Systolic anterior movement

The physiologic behavior of left ventricular (LV) outflow in hypertrophic obstructive cardiomyopathy (HOCM) has been studied extensively over the last 10 years. However, the anatomic site of obstruction has been elusive. It is generally difficult to appreciate the obstructive lesion in the exposed nonbeating heart. Earlier angiographic demonstration of a probable site of obstruction was questioned by Criley and associates. They introduced the concept of cavity obliteration as the cause of artifactual pressure gradients.

Based on observations at surgery, Bjork proposed that asymmetrical hypertrophy of the septum brings about an abnormal rotation of the anterior mitral leaflet. This leaflet balloons into the outflow tract and results in obstruction. This view of the mechanism of LV outflow obstruction did not find general support. More recently Dinsmore and co-workers have demonstrated angiographically an abnormal anterior movement of the anterior mitral leaflet during systole. These observations were extended by Simon and associates who em-
phasized the role of mitral valve in contributing to obstruction.

The present study was undertaken to evaluate the anterior mitral leaflet function in HOCM from reflected ultrasound recordings. Ultrasound cardiology has been established as a method to study movement of the anterior mitral leaflet in a consistent and reproducible manner. The method has been found reliable in the evaluation of mitral stenosis by a number of investigators. Among the several advantages of the method is the rapid pulse frequency between 200 and 2,000/sec. This is in sharp contrast with the 30 to 60 frames/sec rate of exposure in routine cineangiographic technics. Hence, ultrasound cardiology offers a distinct advantage in the accurate evaluation of rapidly moving structures such as valve cusps.

Methods

Six patients, two males and four females, ranging in age from 17 to 57 years were studied. The diagnosis was established in each case by hemodynamic and angiocardiographic studies. One patient had undergone surgical resection of LV outflow for relief of obstruction 34 months prior to the present study.

Ultrasound recordings of the anterior mitral valve leaflet were made using a Physionics echograph and a 2.0-megacycle transducer. The transducer was placed in the fourth or fifth left interspace and angled appropriately to obtain the typical free and snapping movement of the mitral leaflet. The ultrasonic “B” mode display was presented on the upper beam of Tektronic 565 dual-beam oscilloscope and continuous recordings were made on a 35-mm film by means of a Fairchild camera (type 321-A). The lower beam on the oscilloscope was used in multitrace operation by means of a four trace amplifier to record the electrocardiogram, phonocardiogram, and indirect carotid pulse simultaneously with the reflected ultrasound.

The phonocardiogram was obtained through a Maico contact microphone held at the apex. The output was passed through a Sanborn heart sound pre-amplifier (350-1700 B) and filtered through a high-pass filter over 100-cycles/sec. The carotid arterial pulse was obtained by a Sanborn pick-up placed over a carotid artery and a DC amplifier (Model 350-3200).

The observations were made with the subject resting in the supine position. In four patients the effects of intravenous methoxamine were studied. Ultrasound study was carried out during cardiac catheterization in one patient and the identification of the structures by ultrasound technic was obtained by injection of “contrast” substances (for example, indocyanine green and saline) into the specific cardiac chambers. The method of “contrast” ultrasound cardiology for the detection of cardiac chambers and structures developed in our laboratory has been used in a variety of cardiac disorders to validate the functional anatomy.16

Results

To demonstrate the specific nature of the ultrasound findings in HOCM, the recordings from the anterior mitral leaflet in a normal subject and in a patient with aortic valve stenosis were compared.

Normal Subjects

The following description is based on our experience with 50 normal subjects and is in agreement with the results published from several centers. A representative example is shown in figure 1 along with the electrocardiogram and the phonocardiogram. An upward deflection in this and subsequent tracings represents anterior movement and a downward deflection, posterior movement.

Point C, the most posterior position on the leaflet, occurs at the onset of ventricular systole and represents the point of mitral valve closure. A gradual anterior movement from C to D during ventricular systole results from the movement of the mitral ring, the valve cusps remaining firmly closed. In early diastole, a sharp anterior movement, DE, represents the opening of the mitral valve, which is immediately followed by a rapid filling phase. As filling advances, the valve leaflet moves posteriorly to the point F, which marks the end of rapid filling. The slope of EF movement is an index of the rate of rapid ventricular filling. During diastasis, the valve remains partially open, and in late diastole a further anterior opening movement to a peak A occurs from atrial systole. Ventricular filling resulting from atrial systole once again brings about posterior movement of the valve leaflet toward closure. Further posterior movement to complete closure at point C results from the onset.
of ventricular systole. This pattern of movement can be consistently recorded with well-defined limits for the normal amplitudes and the rates of movements.

**Aortic Valve Stenosis**

The pattern of mitral valve movement in patients with aortic stenosis is similar to that observed in normal subjects (fig. 2, upper panel). In those with gross left ventricular hypertrophy, a slight reduction in slope EF may be observed. Similar findings have been reported by us in patients with primary myocardial disease and left ventricular hypertrophy. It may represent a reduced rate of early filling as a result of diminished compliance of the left ventricle. The systolic component (C to D) of the closed mitral leaflet remains undistorted in patients with fixed aortic obstruction.

**Hypertrophic Obstructive Cardiomyopathy**

A representative ultrasound recording of the anterior mitral leaflet in a patient with LV outflow obstruction due to HOCM is shown in figure 2 (lower panel). The gradual anterior systolic movement (C to D) (fig. 2) in the normal has been replaced by a sharp systolic anterior movement (SAM) which does not begin until after the onset of ejection. The SAM reaches a maximum coinciding in time with the early peak of the arterial pulse, as shown in figure 3, and often results in apposition of the mitral leaflet which remains in this position for a period ranging up to 60% of the ejection time while the arterial pulse falls.
Toward the latter part of systole the mitral valve leaflet moves posteriorly to a position of closure. This movement away from the interventricular septum is associated with the late systolic wave in the arterial pulse. The onset of SAM of the anterior mitral leaflet was temporally related to a low frequency systolic click in one patient and with the onset of the murmur in others.

The prominent changes in the ultrasound recording of the mitral leaflet in diastole consist of apposition of the leaflet at point E to the interventricular septum (IVS) and a markedly reduced slope EF. Neither of the two findings can be considered specific, since they may be observed in the presence of massive left ventricular hypertrophy in absence of outflow obstruction. A reduced slope EF has been considered a pathognomonic feature of mitral stenosis by others. Its presence in patients with HOCM and other forms of left ventricular hypertrophy probably represents a reduced rate of ventricular filling resulting from reduced compliance.

In five of six patients with HOCM, the aforementioned characteristic abnormalities of mitral valve movement were observed. Beat-to-beat changes in the contour of the arterial pulse and amplitude of the murmur were associated with changes in amplitude of SAM (fig. 3). Large movements accompanied more pronounced notching of the carotid pulse wave as well as a systolic murmur of greater intensity, suggestive of greater obstruction. On the other hand, the mitral leaflet abnormality was small or absent when the carotid pulse contour was normal and the murmur was low in intensity. Similar changes in the degree of outflow obstruction in HOCM in relation to the phase of respiration have been previously reported.

The one remaining patient who had undergone surgery demonstrated the SAM of the anterior mitral leaflet only in the post-ectopic beats following spontaneous ventricular premature contractions (VPC) during continuous recording (fig. 4). During the beat before the VPC a normal carotid pulse contour and a normal CD movement of the mitral leaflet during ventricular systole were recorded. Furthermore, no significant cardiac murmur was audible. In early diastole the opening movement DE of the mitral valve was interrupted by a VPC which closed the valve back to point C. Following the long pause during the post-ectopic beat, the characteristically abnormal SAM of the mitral leaflet was associated with the abnormal carotid pulse and prominent systolic murmur.

This abnormal systolic anterior movement of the anterior mitral leaflet has not been noted in other conditions causing LV outflow obstruction nor in other forms of primary myocardial disease. On the basis of our experience, the abnormality seems to be specific for HOCM, although its absence does not exclude this entity. False negative results are believed to be related to the position and angulation of the transducer as shown in figure 5. The free edge of the mitral valve leaflet usually shows the maximum anterior excursion in systole while the area adjacent to its annular

Figure 2
Simultaneous recording of mitral valve movement (MV), PCG, and ECG in a patient with aortic valve stenosis (upper panel), and in a patient with HOCM (lower panel). IVS = interventricular septum; AM = anterior mitral leaflet; S1, S2, and S4 = fourth, first and second heart sounds, respectively. The arrow in the lower panel shows the onset of sharp anterior systolic movement (SAM).
attachment may show only minor movement. Thus, it is possible to miss the abnormality if the ultrasound beam is directed adjacent to the annulus. This is in keeping with the angiocardiographic finding of Simon and associates who demonstrated that the major

Figure 3

Two consecutive beats in a patient with HOCM with change in pattern of mitral valve movement, carotid pulse contour, and amplitude of systolic murmur. CP = carotid pulse. Temporal relationship of SAM to arterial pulse wave may be noted.

Figure 4

Changes in pattern of anterior mitral leaflet movement; carotid artery pulse and systolic murmur associated with the ventricular premature contraction and the post-ectopic beat (see text).
The effect of transducer position on ultrasound recording is illustrated in the lower right panel. The course of the ultrasound beam as shown by A (upper left quadrant) is reflected from the free edge of the cusp and shows pronounced abnormality in movement. The course of the beam as shown by B (upper right quadrant) shows less pronounced abnormality, while the course of the beam as in C near the annular attachment of the cusp shows the least abnormality (lower left quadrant). All the three recordings, A, B, and C were obtained in the same patient.

Figure 5

The intravenous administration of methoxamine resulted in characteristic changes in the contour of the arterial pulse, amplitude of the systolic murmur, and the abnormalities of mitral valve movement in HOCM. Figure 6 shows such changes after methoxamine injection in one patient. A prominent systolic murmur and bifid arterial pulse were associated with prominent SAM of the anterior mitral leaflet in the control state. Following systemic vasoconstriction produced by methoxamine, the arterial pulse appeared normal with a small notch near the peak, the systolic murmur was sharply reduced, and the abnormal SAM of the anterior mitral leaflet was small and short-lived. Following recovery from the effects of methoxamine the changes in arterial pulse, the murmur, and the mitral valve movement resembled those recorded during the control state.

Discussion

The ultrasound method has been used extensively in the study of mitral valve function. The anteromedial (aortic) leaflet of the mitral valve faces anteriorly throughout the cardiac cycle, and the pattern of its movement can be consistently recorded in normal subjects as well as in patients with heart disease. It can be recognized from its characteristic snapping movement in the “A” mode display. In a previous paper we reported the
Acute effects of methoxamine on mitral valve movement, carotid pulse, and the systolic murmur are shown. AM = anterior (medial) and PL = posterior (lateral) leaflets.

Functional anatomy in a patient with HOCM is studied by contrast ultrasound cardiology in the lower panel. Injection of saline through the catheters placed just below the aortic valve resulted in dense echo source in the LV outflow space formed by interventricular septum (IVS) in front and anterior (medial) leaflet (AM) of mitral valve (MV) posteriorly. The use of contrast ultrasound cardiology as a method of validating functional anatomy and of studying different conditions including HOCM. In brief, the method consists of injections of indocyanine green and saline as ultrasonic contrast agents into the cardiac chamber during cardiac catheterization. Dense echoes then appear in the ultrasound recording. These are first seen in the chamber of injection and subsequently downstream with blood flow. By this method, we have shown that, with the transducer in the mitral valve recording position, the path of the ultrasound beam goes through the body of the right ventricle, interventricular septum, left ventricular outflow tract, anterior mitral leaflet, and generally the left atrium posteriorly. On injection of the contrast material through the catheter in the left ventricular outflow tract, the space in front of the anterior mitral leaflet is filled with dense echoes. We have used this method to validate the role of mitral valve movement in the outflow obstruction.

onset of SAM is indicated by an arrow in the first beat to the left. Dense echoes appear to spill into left atrium during ventricular systole.
An example of such an injection study in a patient with HOCM is shown in figure 7. The injection was made with the catheter placed in the LV outflow tract, and the resulting contrast effect is shown in the lower panel. The anterior margin of the dense cloud of echoes in the LV outflow is formed by the interventricular septum. This shows little movement, if any, during the phases of cardiac cycle. The posterior margin of the outflow tract is formed by the anterior mitral leaflet which shows a normal anterior movement in diastole and an abnormally sharp anterior movement in systole compromising the lumen. In addition, the dense echoes appear to "spill" behind the mitral valve into the left atrium in systole during the SAM of the mitral leaflet. This probably represents mitral valve incompetence brought about by the abnormal movement of the anterior mitral leaflet.

The small width of the LV outflow in HOCM is primarily related to the asymmetric hypertrophy of the interventricular septum. The finding that the mitral leaflet abuts against the interventricular septum in early diastole (point E) in patients with HOCM has recently been reported. This is not an unexpected finding owing to small size of the left ventricular outflow space. This, however, is not a specific finding in our experience and is irrelevant to the dynamic outflow obstruction in systole, a characteristic feature of the disease. The abnormalities in ultrasound recordings suggest that the narrowing of the LV outflow space in systole results mainly from the abnormal movement of the mitral leaflet. This is further supported by the timing of abnormalities of mitral leaflet movement with other hemodynamic evidences of obstruction. The abnormal systolic movement of mitral leaflet begins sometime after the onset of ejection as registered in the carotid pulse and is maximum at the time of the early peak in the carotid pulse. This also heralds the onset of systolic murmur and negative descent of the pulse. In the latter part of ejection, as the mitral leaflet moves away from the septum widening the LV outflow tract, the late systolic wave in the carotid pulse is noted. The early peak of the typical arterial pulse in HOCM is related to unobstructed early ejection and the notch after the peak to the dynamic outflow obstruction. The second systolic wave seems to be related to the relief of obstruction as the mitral leaflet moves away from the outflow space. Whether the presence of mitral incompetence is a direct consequence of this abnormality is not established. It is an attractive possibility that the late onset of the systolic murmur of mitral incompetence in HOCM may be causally related to the abnormal mitral valve function.

The association of abnormal mitral valve function in the post-ectopic beat along with other evidence of increased dynamic obstruction would further lend support to the role of mitral valve in LV outflow obstruction in HOCM. The changes observed with administration of methoxamine may also be best explained on the basis of a causal relationship. Vasoconstriction results in amelioration of dynamic obstruction in HOCM with striking changes in arterial pulse contour and diminution of the systolic murmur. This is associated with either reduction or abolition of the abnormal mitral valve movement. On a beat by beat basis the abnormalities in the pulse contour, the systolic murmur, and the mitral valve movement reappear simultaneously as the effects of methoxamine are no longer present.

The mechanisms that result in the sharp anterior movement of the anterior leaflet are not yet clear. Geometric distortions in the anatomy and function of papillary muscle from asymmetric hypertrophy or excessive traction by the hypertrophied papillary muscle in a small cavity, or both, may be important factors. Increased intracavitary pressures in a large filled ventricle would tend to oppose this traction on the anterior leaflet. This would provide an explanation for many of the physiologic and pharmacologic factors known to influence the degree of obstruction, since change in ventricular volume has been implicated as the prime determinant of changes in severity of obstruction.

This abnormality of mitral valve function is, in our experience, specific for HOCM. We do not consider that the mitral valve plays a
primary role in the development of the disease. It is more likely the result of changes in papillary muscle function from the distortion associated with extreme asymmetric septal hypertrophy. The factor or factors which bring about this asymmetric hypertrophy of the interventricular septum remain to be elucidated.

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References

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