The Posterior Left Atrial Echocardiogram of Mitral Regurgitation

R. Patton, M.D., L. Dragatakis, M.D., D. Marpole, M.D., and A. Sniderman, M.D.

SUMMARY The motion of the posterior wall of the normal left atrium has not been studied systematically. The superoposterior portion of the left atrium is adynamic throughout the cardiac cycle, whereas the inferoposterior portion is displaced posteriorly with left atrial filling during ventricular systole. In the present study, the left atrial diameter (LAD), the left atrial systolic motion (LASM) and the left atrial systolic velocity (LASV), were determined in the following groups of patients: 34 normals; eight patients with either coronary artery disease or aortic stenosis; six patients with aortic insufficiency; and three patients with ventricular septal defect. The results obtained were compared to 15 patients with angiographically documented mitral regurgitation. In the last group, the LAD (4.2 ± 1.9 cm) and LASM (12.3 ± 1.23 cm) and LASV (1.2 ± 0.04 cm) were significantly greater reflecting the early accentuated filling of the left atrium induced by mitral regurgitation. As well, the product of these three parameters was greater in the mitral regurgitation group (63.2 ± 7.34 cm²/sec) than in the other groups and patients with mild to moderate regurgitation had a significantly lower value than those with moderate to severe regurgitation (45.7 ± 4.1 vs 78.5 ± 10.9, P < 0.02). The left atrial echocardiogram, therefore, is an aid in the diagnosis of mitral regurgitation and provides a rough index of the severity of the lesion.

IN CONTRAST TO MITRAL STENOSIS, the echocardiogram at present is considered to be of limited value in the diagnosis of mitral regurgitation. This is unfortunate, not only because this lesion is a common chronic abnormality, but also because even severe mitral regurgitation may be difficult to recognize in the presence of acute left ventricular failure. The problems posed in recognizing and determining the clinical significance of mitral regurgitation differ in the two situations. In the one, there may be doubt as to the cause of a systolic murmur in an otherwise stable patient, whereas in the other there may be doubt as to presence of a correctible cause of sudden severe pulmonary edema. At both extremes, although for different reasons, the patient may undergo echocardiographic examination; it is the purpose of this study to extend the information that may be derived from this procedure.

To date, most attention in echocardiography has been paid to the abnormalities in left ventricular performance and thus ejection of blood through the aortic valve which can occur in this lesion. Consequently, signs of left ventricular volume overload and premature closure of the aortic valve suggest the presence of mitral regurgitation in an individual patient. However, little attention has been paid to abnormalities in left atrial movement. Enlargement of the left atrium has, of course, been recognized as a consequence of insufficiency of the mitral valve, but this abnormality as well as signs of left ventricular volume overload, are far from diagnostic. Because the principal hemodynamic abnormality in mitral regurgitation is systolic flow from the left atrium to left atrium, the present study examines changes in systolic velocity of left atrial posterior motion in mitral regurgitation which can be recognized by the echocardiogram.

Habitually, the left atrium is visualized echocardiographically with the sonar beam passing through the aortic valve. The posterosuperior portion of the left atrium is thus seen and is essentially adynamic during the cardiac cycle. However, Winsberg and Goldman in 1969 pointed out that in the normal heart, the postero-inferior portion of the left atrium did move in systole. Furthermore, this motion was characteristic and could easily be distinguished from that of the posterior left ventricle. During systole, the latter moves anteriorly with ventricular emptying, but the former moves posteriorly as the left atrium is filled with blood from the pulmonary veins. The mitral valve provides an initial guide to the topography. The inferoposterior left atrium is usually seen in a plane including only the anterior leaflet of the mitral valve; when the posterior left ventricular wall is seen, the posterior mitral leaflet is present as well. This sequence can be recognized in a sweep beginning from the aortic valve and gradually sweeping inferiorly to reach the left ventricle.

Since systolic filling of the left atrium is increased in mitral regurgitation by the regurgitant volume, we reasoned that the pattern of normal inferoposterior left atrial posterior motion would be altered. That is, this region would be displaced a greater distance posteriorly at a more rapid rate than the normal. Although this concept has been mentioned briefly in the literature, no systematic study of the changes in posterior left atrial wall motion has been made.

Methods

Sixty-six individuals were studied and divided into five groups. Group A consisted of thirty-four normals with a mean age of 36.4 years (range 20–51). Group B was made up of eight patients with either coronary artery disease or aortic stenosis with a mean age of 54.6 years (range 26–69). Group C consisted of six patients with left ventricular volume overload due to aortic regurgitation with a mean age of 53 years (range 32–72). In Group D, there were three patients with left atrial volume overload due to ventricular septal defect. In two of these patients, the lesion was congenital, whereas in one it appeared after a myocardial infarction. Group E consisted of fifteen patients with mitral regurgitation. The etiology of mitral regurgitation varied, but included rheumatic valvular disease (10), ruptured chordae tendineae (2) and papillary muscle dysfunction (3). None had mitral valve prolapse syndrome. In both patients with ruptured
chordae tendineae and in two with papillary muscle dysfunction, the lesion was acute. The mean age of these patients was 47.9 with a range from 25–72 years.

Diagnostic cardiac catheterization was performed for all patients except group A, by standard methods previously described from our laboratory. The angiographic degree of mitral regurgitation was quantitated as described by Honey et al.

The echocardiograms were obtained using a Picker Echoview-10 ultrasonoscope utilizing a 3.5 MHz transducer with a focal zone of 7.5 cm. The recordings with simultaneous electrocardiograms were made on a strip-chart recorder (Honeywell Fiberoptic 1856 Visacorder), with paper speeds between 25–50 mm/sec. The patients were studied in the supine or left decubitus position with the transducer placed in the third or fourth left intercostal space adjacent to the sternal border.

The left atrium was examined at two positions. When the ultrasound beam passed through the aortic valve, the usual pattern of left atrial motion was obtained. The superoposterior left atrial wall is relatively adynamic and left atrial diameter (LAD) was measured in this view at end systole. The inferoposterior section of the left atrium was recognized by a characteristic posterior excursion which begins after the R wave of the electrocardiogram. In this position, the anterior, but not posterior, mitral leaflet was visualized. To quantitate this pattern of left atrial motion we measured: 1) the total systolic posterior motion of the left atrium (LASM); and 2) the initial velocity of the posterior motion of the left atrial wall (LASV) (fig. 1).

All echocardiograms were analyzed without knowledge of the clinical or angiographic data. Interobserver variation was less than 10%.

The data were analyzed statistically by Student's unpaired t-test with each group compared in turn to the patients with mitral regurgitation.

Results

The normal motion of the posterior segments of the left atrium, A-V groove and left ventricle during the cardiac cycle can be seen in figure 1. The four areas encountered sequentially during an echocardiographic sweep are superoposterior left atrium (a), inferoposterior left atrium (b), A-V groove (c), and left ventricle (d). In panel a, there is very little motion of the left atrium. This view is obtained when the echocardiographic beam passes through the aortic valve; in contrast, in panel d, the characteristic systolic anterior motion of the posterior left ventricular wall is apparent. This pattern differs from that of the inferoposterior left atrial wall shown in panel b. Here, following the R wave there is a holosystolic (but gradual) posterior displacement of the left atrial wall. This continues until the opening of the mitral valve. If the transducer is angled slightly toward the left ventricle from position b, an area intermediate between the inferoposterior left atrial wall and left ventricle is seen. Here, the motion of the posterior wall follows no simple pattern, but rather is complex. We have designated this as the region of the A-V groove. In the 34 normal patients (group A, table 1), the left atrial diameter was 2.9 ± 0.1 cm (mean ± SEM). The total posterior displacement of the left atrium during ventricular systole averaged 0.9 ± .01 cm. The velocity of the posterior left atrium in systole (LASV) was 4.3 ± 0.2 cm/sec. Following ventricular systole, the mitral valve opens and the left atrial wall abruptly moves anteriorly during the initial rapid filling phase of ventricular diastole.

The motion of the left atrium is markedly changed with mitral regurgitation. Figure 2 demonstrates the typical findings in this lesion. In panel a, the superior portion of the left atrium is seen with the sonar beam passing through the aortic valve. The posterosuperior portion of the left atrium remains, as in normals, adynamic throughout the cardiac cycle. However, mitral regurgitation causes three characteristic changes in the inferoposterior accelerated wall motion during ventricular systole. First, the absolute systolic posterior motion increases; second, LASV increases presumably due to accelerated as well as greater left atrial filling; and third, posterior excursion of the left atrial wall ends in early systole. All these occur in addition to left atrial enlargement.

The echocardiographic data supporting these conclusions

Figure 1. Normal atrial wall motion. Four areas of the normal heart are shown sequentially: a) superoposterior left atrium; b) inferoposterior left atrium; c) AV groove; d) left ventricle.
### Table 1. Echocardiographic and Angiographic Data

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#### Group A – Normal

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### Table 2. (continued)

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#### Group D – (Ventricular Septal Defect)

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Abbreviations: AMR = angiographic mitral regurgitation ranging from 0 (absent) to IV; N = normal; CAD = coronary artery disease; AS = aortic stenosis; NS = not significant; AR = aortic regurgitation; VSD = ventricular septal defect; CMR = chronic mitral regurgitation; AMR = acute mitral regurgitation.

Rates are given in table 1. It can be seen, except for group D (ventricular septal defect) that the left atrium is significantly larger (4.2 ± 1.9 cm) in patients with mitral regurgitation than in any of the other groups. Similarly, the left atrial systolic motion in mitral regurgitation (1.2 ± 0.4 cm) is significantly greater (table 1) than the mean of patients in the other four groups. The most striking difference for an individual parameter is obtained for LASV (table 1). Here, the patients with mitral regurgitation had a mean value of 12.3 ± 1.23 cm/sec whereas the mean values for the other groups were: A) 5.5 ± 0.4; B) 5.8 ± 0.7; C) 5.1 ± 1.44; D) 6.8 ± 1.8.

Left atrial systolic wall motion was quantitated as the simple product of these parameters. As would be anticipated, the mean value of such a product is significantly...
greater in the patients with mitral regurgitation (63.2 ± 7.34) than in the other groups: A) 10.6 ± 0.7; B) 18.0 ± 3.8; C) 13.7 ± .66; D) 24.3 ± 2.6.

To examine whether these indices indicated the degree of mitral regurgitation, the product of the three parameters was compared in normals and patients with mild (grades I-II) or moderate to severe (grades III-IV) regurgitation. The findings are shown in figure 3. This product was significantly greater in patients with grade III-IV regurgitation than in those with grade I-II lesions (78.5 ± 10.9 vs 45.7 ± 4.1, \( P < 0.02 \)).

It is clear, however, that although a statistically significant difference exists between the groups, there is individual patient overlap (table 1).

**Discussion**

The systolic posterior wall motion of the normal left atrium likely reflects systolic filling through the pulmonary veins. In dogs, Gribbe et al. showed that filling of the left atrium begins immediately after the R wave and is more rapid at the beginning rather than the end of systole. Furthermore, atrial expansion is eccentric with the posteroinferior area moving most while the superior portion appears immobile, apparently being fixed by the pulmonary veins. With mitral regurgitation, the systolic increase in volume in the left atrium is greater than normal, consisting of the regurgitant volume from the left ventricle as well as normal filling from the pulmonary veins. The major portion of the regurgitant volume is delivered to the left atrium early in systole. Thus, the findings of the present study confirm the initial hypothesis. The increased initial rate of systolic motion of the left atrium, the increased total atrial displacement and increased left atrial size are all consistent with the increased rate and extent of atrial filling in mitral regurgitation.

In general, a correlation between the severity of the mitral regurgitation and the degree of alteration of left atrial filling has been shown, agreeing with the previous work of Kennedy et al. Nevertheless, exceptions should be expected.

**FIGURE 2.** Left atrial motion in mitral regurgitation. Panel a depicts the superoposterior motion of the left atrium in a patient with mitral regurgitation. It remains as in the normal adynamic. Panel b — the inferoposterior portion of the left atrium — demonstrates the characteristic alterations. The initial slope, i.e., the left atrial systolic velocity (taken between single headed arrows) is not only increased, but terminates early in systole. The left atrial posterior motion (distance of double headed arrow) is shown to the right.

**FIGURE 3.** The product of LASV (a), LAPM (b), and LAD (c), is shown for normals, for patients with grade I-II mitral regurgitation and for those with grade III-IV regurgitation judged by the angiogram.
since, depending on the compliance of the left atrium, a variable amount of blood may regurgitate into the pulmonary veins. The amount of regurgitation is also influenced by the contractile state of the left ventricle and the afterload the ventricle faces. There is also imprecision in the quantitative determination of the severity of regurgitation by angiography.

In this study, the inferoposterior portion of the left atrium has been identified because it moves as anticipated. That is, the motion in an anteroposterior direction is appropriate since it expands during ventricular systole and diminishes in diastole. Although the chance remains that different regions have been observed in systole and diastole, this would not impair the empirical value of the observations. Thus, in figure 1, the posterior wall of the atrium appears thicker in diastole than systole. These additional echoes might be reflecting from structures posterior to the left atrium or could indicate that we have seen the left atrium in systole but the A-V groove in diastole. Evidence in patients with pericardial effusion extending behind the left atrium suggests the former explanation is correct. Thus, in figure 4, we see normal movement while the true left atrial wall thickness is unchanged throughout systole and diastole.

Reasonable care — both technical and clinical — must be used for this approach. In patients with a giant left atrium, the posteroinferior wall may be parallel to the transducer beam. The ultrasound beam from this area is thus reflected away from the transducer and the area is incompletely visualized. Therefore, in such patients (who in any case are easily recognized by left atrial size), the diagnosis should not be excluded by a negative examination. From a technical viewpoint, transducer frequency is an important consideration. As can be seen in figure 5, a frequency of 2.25 MHz is inadequate to visualize the posteroinferior left atrial wall. Resolution of an apparently inchoate motion improves markedly with the 3.5 MHz transducer although both were focused at 7.5 cm. Both panels were obtained from the same patient at the same examination. The right hand panel clearly delineates the exaggerated motion of the posteroinferior left atrial wall in mitral regurgitation; the left hand panel is of no diagnostic value.

An acute ventricular septal defect could be a difficult distinction to make, since in this lesion, as in mitral insufficiency, there is left atrial volume overload. Left atrial systolic excision can be increased as well as left atrial size. The systolic velocity rate will also be brisk. It has been our experience, however, that the normal curvilinear posterior atrial excursion is retained with the left atrium continuing to fill throughout systole. In contrast, in mitral regurgitation, left atrial posterior wall excursion terminates in mid systole.

In considering the application of this method, the case of mitral valve prolapse with late systolic murmur must be mentioned. This syndrome provides an example of how the concepts in the present study must be applied hemodynamically to an apparent exception. Figure 6 demonstrates the findings in such a case. From the right hand panel, it is obvious that a prolapse of the mitral valve is present with a marked late systolic accentuation of the prolapse. The
left hand panel is obtained from the same patient, but with a slight change in angulation so that left atrial motion could be visualized. Note that the early systolic filling of the left atrium is biphasic. Filling occurs at a normal rate early in systole (5.5 cm/sec), but the posterior left atrial wall is rapidly displaced posteriorly in mid-systole (10.8 cm/sec). Thus, mitral valve prolapse with non-holosystolic regurgitation does not behave as an exception to the general concept, but rather confirms the hypothesis that the rapid posterior motion of the atrial wall is directly related to the mitral regurgitation itself.

In many, if not most patients, the diagnosis of mitral regurgitation is obvious by physical examination, chest X-ray and electrocardiogram. These, taken together, provide a reasonable clinical base to evaluate the severity of the lesion. However, in the very ill patient with acute mitral regurgitation, whether produced by endocarditis, a torn chorda or myocardial infarction, these criteria may fail. It is precisely these patients in whom the physical signs are known to be least reliable. For these people, advances in noninvasive diagnosis of lesions which may demand major and potentially hazardous changes in therapy are most important. At present, our confidence in the quantitative determination of mitral regurgitation by echocardiogram remains limited since substantial overlap persists between moderate and severe regurgitation. This examination is complimentary to, but does not replace, bedside catheterization by the Swan-Ganz technique.

In the present study, the normal motion of the posterior left atrium has been described and quantitated. Recognition of these features which have previously not been studied systematically allows the diagnosis of mitral regurgitation.

The motion of the left atrium is a sufficiently faithful mimic of hemodynamic abnormalities that typical mitral regurgitation can be distinguished from other causes of left ventricular volume overload, delayed onset mitral regurgitation and ventricular septal defect. Studies of posterior left atrial motion should, however, always be considered in conjunction with the other data derived from the echocardiogram and the clinical examination.

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