The Value of Left Parasternal Impulse Recordings in the Assessment of Mitral Regurgitation

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SUMMARY

Left parasternal cardiograms were recorded at the fifth intercostal space in 30 patients with mitral regurgitation. Early systolic outward movements (E), late systolic outward movements (LOM), and the ratio of the LOM area to the total area occupied by the left parasternal cardiogram (LPC area) were examined. Thirteen patients had pure mitral regurgitation due to ruptured chordae tendineae (ten patients) or "floppy mitral valve" (three patients). In these patients LOM/E correlated significantly with the regurgitation volume determined by the difference between angiographic and Fick stroke volumes, \( r = 0.93, P < 0.01 \), and with the height of the "v" wave in the pulmonary artery wedge pressure tracings \( r = 0.79; P < 0.01 \). Simultaneous recordings of left parasternal movements and pulmonary wedge pressures in eight patients in this group showed a close relationship between LOM and "v" waves.

Fourteen patients had rheumatic heart disease and mitral regurgitation associated with mitral stenosis of varying degrees of severity. In this group there was no significant correlation between LOM/E or LOM area/LPC area and the regurgitation volume or the height of the "v" wave. Of three patients who had congenital atrial septal defect in addition to mitral regurgitation, none had prominent LOM in the left parasternal cardiogram.

This study suggests that if mitral stenosis and atrial septal defect can be excluded on clinical evidence, the left parasternal cardiogram may be used to obtain a noninvasive assessment of the severity of mitral regurgitation, particularly in patients with recent onset of the disease.

Additional Indexing Words:
Mitral stenosis  Atrial septal defect  Apex cardiogram  Kinetocardiogram  Left atrium  Pulmonary hypertension  Ruptured chordae tendineae

IN NORMAL ADULTS, tracings of left parasternal precordial movement show in systole an early outward movement (E) which begins before the first heart sound, peaks in early systole, and is followed by a major negative wave that is maximum at or near the aortic component of the second heart sound.\(^1-4\) In ventricular diastole the tracing is characterized by a positive filling wave and a small presystolic positive deflection corresponding to atrial contraction (fig. 1A).

In patients with mitral regurgitation, a left parasternal systolic lift can be palpated and recorded which peaks at or near the time of aortic valve closure. This late systolic outward movement (LOM) is believed to be caused by anterior displacement of the heart by the enlarging left atrium; the systolic volume of left atrium is augmented by the volume of blood regurgitating through the incompetent mitral valve. Although this important sign was first noted in 1937,\(^5\) it is only recently that it has received due emphasis.\(^6-8\)

In the present study, we have examined the hypothesis that the magnitude of the late systolic outward movement in the left parasternal cardiogram quantitatively reflects the severity of mitral regurgitation.

Material and Methods

This study is based on data from 30 patients, 13 women and 17 men, ranging in age from 17 to 77 years, who were hospitalized at the University of Iowa and Veterans Hospitals. In addition to standard clinical, electrocardiographic, and radiologic studies, all patients had external noninvasive studies, including simul-
The left parasternal cardiogram (LPC) in the normal individual (A) and in patients B.H., F.R., and H.D. (B,C,D, respectively) with pure mitral regurgitation of different grades of severity. The horizontal line drawn at the base of “E” wave is the reference line for measurement of early (E) and late (LOM) systolic outward movements. The shaded area is LOM area and the quadrangle is the total systolic area of the left parasternal cardiogram (LPC). The amplitude of the o wave (mm Hg) is given beneath each set of tracings. RV = regurgitant volume; CAR = carotid pulse tracing; MF = medium frequency; LSB = left sternal border.

Simultaneous recordings of lead II of the electrocardiogram, phonocardiograms, carotid tracings, and apex cardiograms or left parasternal cardiograms (LPC). Left parasternal cardiograms were recorded during expiration, at the fifth intercostal space with patients in the supine position. Tracings were obtained using a Schwartz transducer (Z 101/37) with a time constant of 800 msec which gives a flat response of from 0.1–20 Hz. The transducer was strapped firmly to the chest wall and recordings were made on a direct-writing four-channel Schwartz polygraph. All tracings were obtained prior to cardiac catheterization and thus without prior knowledge of the hemodynamic findings.

Patients in the study had right and left cardiac catheterization using standard techniques. Simultaneous recordings of pulmonary artery wedge pressure pulse, left parasternal cardiogram, electrocardiographic lead II and phonocardiogram were repeated in the catheterization laboratory on 18 patients. Left parasternal cardiograms were obtained in the same fashion using an Electronics for Medicine crystal microphone (time constant 650 msec) with filters for the selective recording of low frequencies (0.1–20 Hz). Catheter pressures were obtained using a Statham strain gauge (p23Db) with reference to the midchest point. Pressure recordings were obtained on a multichannel Electronics for Medicine photographic recorder (D16EFM). Cardiac outputs were measured by the Fick principle. Left ventricular volume measurements were obtained from high speed cineangiograms filmed in the right anterior oblique position using a Phillips 9-inch image intensifier, Renografin-76 in a dose of 1–1.5 ml/kg body weight was injected into the left ventricle through #7 or #8 angiographic catheters at a rate of 20 ml/sec by a Cordis-II injector. Ventricular extrasystoles and beats immediately following premature contractions were excluded. Correction for image magnification was made according to the method of Greene et al. by filming a metal grid of 1 cm squares placed at the midchest level of the patient.

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End-systolic and end-diastolic volumes were obtained from the planimetered area and longest measurable length according to the method of Dodge, Hay, and Sandler using an ellipsoidal reference figure. Volumes were corrected (c) by the regression equations
\[ EDV_c = EDV \times 0.92 - 32.5; \quad ESV_c = ESV \times 1.02 - 4.4; \quad \text{and} \quad SV_c = SV \times 0.79 - 14.8, \]
where EDV is the end-diastolic volume, ESV is the end-systolic volume and SV is the stroke volume. Regurgitation volumes were measured as the difference between the angiographic and the Fick stroke volumes. In patients with atrial fibrillation, angiographic volumes were represented by the average of few acceptable beats.

In patients with mitral stenosis, the mitral valve area was estimated using the formula of Gorlin and Gorlin and utilizing angiographic cardiac output to represent flow through the mitral valve.

Left parasternal cardiograms were examined for the early (E) and late systolic outward movements (LOM). The ratio LOM/E was obtained by measuring the amplitude of these waves at their farthest vertical distance from a horizontal line drawn at the point of onset of the E wave (fig. 1). In patients with atrial fibrillation, the average of several representative beats was taken.

The area occupied by the late outward movement in systole was measured by planimeter and was related to the total systolic area of LPC. This LPC area was measured from a rectangle extending from the onset of the q wave in the electrocardiogram to the aortic component of the second sound and from the highest point of the left parasternal cardiogram to its lowest point (fig. 1A, B, D). The area under LOM in systole (LOM area) was measured from the nadir of the "E" wave to the aortic closure sound (shaded area in fig. 1B, D).

Measurements were made independently by two observers, one of whom had no knowledge of the patients or the type of their disease, and the data were averaged. Measurements obtained by the two observers were practically identical.

Six patients were excluded from the study because discrete left parasternal movements could not be recorded, and/or the volume of mitral regurgitation was too small to be measured angiographically, or technical problems precluded accurate estimation of left ventricular volumes from cineangiograms. Also excluded from the study were patients with mitral regurgitation associated with dyskinesia of the left ventricle because the latter condition is known to produce changes in the left parasternal cardiograms indistinguishable from those caused by mitral regurgitation.

**Results**

Patients were divided into three groups.

**Group I: Pure Mitral Regurgitation**

This group was composed of 13 patients. Their ages, sex, external graphic, and hemodynamic findings are shown in table 1. Eight of the ten patients with ruptured chordae tendineae, and one of three patients with floppy mitral valve underwent surgery and the diagnosis was confirmed in each. In this group, the ratio of late outward movement/early systolic movement (LOM/E) correlated significantly with the angiographically determined regurgitation volume (r = 0.93, P < 0.01) (fig. 2A), and with the amplitude of the pulmonary artery wedge v wave (r = 0.79, P < 0.01) (fig. 3A). The LOM area/LPC area also correlated significantly with the regurgitation volume (r = 0.89, P < 0.01)

<table>
<thead>
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<th>Table 1</th>
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**Findings in Patients with Isolated Mitral Regurgitation Due to Ruptured Chordae Tendineae (RC) or "Floppy Mitral Valve" (FM)**

| Patient | Age | Sex | Diagnosis | Rhythm | LOM/E | LOM area LPC area | "v" wave mm Hg | Regurgitation vol. ml/beat | Pulmonary resistance units | Left atrial size |
|---|---|---|---|---|---|---|---|---|---|---|---|
| *IJ | 54 | F | RC | Sinus | 2.4 | 0.30 | 43 | 67 | 1.6 | + |
| FR | 38 | M | RC | Sinus | 1.0 | 0.14 | 12 | 32 | 1.8 | + |
| *EH | 60 | M | RC | Sinus | 1.3 | 0.31 | 13 | 45 | 1.5 | + |
| *NG | 38 | M | RC | Sinus | 4.0 | 0.43 | 29 | 159 | 1.5 | + |
| *KE | 62 | M | RC | Sinus | 6.0 | 0.48 | 38 | 164 | 7.0 | + |
| *VI | 58 | M | RC | AF | 3.0 | 0.50 | 35 | 150 | 5.5 | + |
|KF | 61 | M | RC | AF | 3.3 | 0.38 | 39 | 123 | 3.5 | + |
| *LR | 38 | M | RC | Sinus | 1.3 | 0.26 | 14 | 34 | 1.5 | + |
| *RD | 39 | M | RC | Sinus | 2.0 | 0.51 | 20 | 64 | 1.3 | + |
| *HD | 52 | M | RC | Sinus | 4.0 | 0.58 | 32 | 130 | 2.7 | + |
| *TI | 47 | M | FM | AF | 2.1 | 0.29 | 14 | 45 | 1.5 | + |
| BH | 17 | M | FM | Sinus | 0.2 | 0.05 | 8 | 12 | 1.0 | + |
| WF | 20 | M | FM | Sinus | 0.5 | 0.13 | 9 | 15 | 1.2 | + |

*Diagnosis confirmed at surgery.

†Pulmonary resistance in units = \( \frac{\text{Mean pulmonary artery pressure} - \text{mean pulmonary wedge pressure (mm Hg)}}{\text{Minute cardiac output (liters/min)}} \)

‡Left atrial size was estimated from cineangiography (+ slight enlargement, +++++ huge enlargement).
The correlation between the angiographically determined regurgitant volume in ml/beat and LOM/E (late/early systolic outward movement) in patients with pure mitral regurgitation (M.R.) (A) and in those with mixed mitral valve lesions (M.R. & M.S.) (B). The solid line is the regression line and the stippled lines represent ± two standard deviations.

Figure 2

The correlation between the height of the v wave in the pulmonary wedge pressure and the LOM/E (late/early systolic outward movements) in patients with pure mitral regurgitation (A) and in those with rheumatic mitral disease (B). Stippled lines represent ± two standard deviations from the regression (solid) line.

Figure 3

1. Regurgitation volume (R.V.) using LOM/E
   \[ R.V., \text{ml} = 32(\text{LOM/E}) + 3.4 \text{ ml} \]

2. Regurgitation volume using LOM area/LPC area
   \[ R.V., \text{ml} = 11.7 (e)^{4.5} \frac{\text{LOM area}}{\text{LPC area}} \]

*Exponential function \( e = 2.718. \)
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3. v wave using LOM/E
   \[ v, \text{mm Hg} = 7.2 \times (\text{LOM/E}) + 7.9 \]

4. v wave using LOM area/LPC area
   \[ v \text{ wave, mm Hg} = 7.1 \times (e^{2.2 \times \left( \frac{\text{LOM area}}{\text{LPC area}} \right)}) \]

Simultaneous left parasternal and wedge pressure recordings were obtained in eight patients including six patients with ruptured chordae tendineae (fig. 6). The onset and peak of LOM in the external recording preceded the same events related to the pulmonary wedge v waves consistently by 70-90 msec, which is equal to the time lag between left atrial events and the wedge pressure recording in our laboratory. There was striking parallelism between the two waves in all but one patient (T.I.) with a markedly dilated left atrium and longstanding mitral regurgitation (fig. 6D).

Group II: Mitral Regurgitation and Stenosis

This group was composed of 14 patients who had rheumatic mitral regurgitation associated with variable degrees of mitral stenosis, and either minimal or no aortic valve disease. Table 2 shows the age, sex, data obtained from the left parasternal cardiogram, and the hemodynamic findings in this group of patients. As shown in the table and figures 2-5, neither LOM/E nor LOM area/LPC area correlated with either the regurgitation volume or the height of the v wave observed in wedge pressure pulse tracings.

Simultaneous left parasternal and wedge pressure pulse recordings were obtained in ten patients. In four of these, (N.P., E.J., G.J., F.Mi.) there was great similarity between the LOM wave in left

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**Figure 4**

The correlation between the regurgitation volume in ml/beat and the LOM (late systolic outward movement) area/LPC (left parasternal cardiogram) area in patients with pure mitral regurgitation (M.R.) (A) and those with mixed mitral valve lesions (M.R. + M.S.) (B).

**Figure 5**

The correlation between the height of the v wave in the pulmonary wedge pressure and the LOM area/LPC area (ratio of areas of late systolic outward movement to left parasternal cardiograms) in patients with pure mitral regurgitation (M.R.) (A) and in those with rheumatic mitral disease (M.R. + M.S.) (B).

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Table 2
Findings in Patients with Rheumatic Mitral Valve Disease with Both Stenosis and Regurgitation

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<th>Patient</th>
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<th>Rhythm</th>
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<th>LOM area LPC area</th>
<th>v wave mm Hg</th>
<th>Regurgitation vol. ml/beat</th>
<th>Mitral valve resistance cm/m²</th>
<th>Pulmonary resistance units</th>
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See table 1 for abbreviations.

parasternal cardiomogram and v wave in pulmonary wedge tracing (fig. 7A). In four patients (F.M., S.K., V.I., K.K.) there was an exaggerated E wave which affected the pattern in left parasternal

Figure 6
Simultaneous tracings of wedge pressure waves (redrawn 0.08 second earlier) and left parasternal cardiograms (LPC) in patients L.R., R.D., H.J., and T.I. (A,B,C,D) with pure mitral regurgitation. RV = regurgitant volume. The amplitude of the v wave (mm Hg) is given beneath each set of tracings. E = early systolic outward movement; LOM = late systolic outward movement.

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Figure 7
Simultaneous recordings of the left parasternal cardiogram (LPC) and wedge pressure in patients with rheumatic mitral valve disease with both stenosis and regurgitation. (A) from patient E.J. with mitral valve area 1.4 cm²/m², pulmonary systolic pressure of 40 mm Hg and pulmonary vascular resistance of 2.8 units. (B) from patient S.K. with mitral valve area 0.52 cm²/m² and pulmonary systolic pressure of 72 mm Hg (pulmonary resistance = 11 units). E = early systolic outward movement; LOM = late systolic outward movement. The amplitude of the v wave (in mm Hg) is given beneath each tracing.

cardiogram (fig. 7B); these included two patients with pulmonary vascular resistance in excess of eight units accompanied by tricuspid regurgitation. In two patients (C.J., R.W.) the late outward movement was distinctly more prominent in comparison with pulmonary wedge v wave; one of these patients had a giant left atrium (fig. 8B).

Group III: Mitral Regurgitation with Atrial Septal Defect

This group included three female patients. The septal defect was of secundum type in two patients and of primum type in the other. All three patients had prominent E waves but no positive late systolic outward movement in left parasternal cardiograms.

Discussion

The present study adds further evidence to the hypothesis that the late systolic outward movement observed in left parasternal cardiograms of patients with mitral regurgitation reflects left atrial events characteristic of this disease. In patients with pure mitral regurgitation, particularly of recent origin, the good positive correlation between the regurgitation volume and findings in the left parasternal cardiogram allows a quantitative assessment of the severity of mitral regurgitation by a simple, noninvasive, and reproducible technique.

Although the amplitude of discrete waves in tracings of precordial movements has been studied and the quantitated apical impulse has been utilized to differentiate patients with mitral regurgitation from normal people, there is a wide range of normality in these measurements with considerable overlap between findings in normal people and in patients with mitral regurgitation. A major reason for this difficulty is that wave amplitude in impulse cardiograms is influenced not only by the cardiac movements and volume changes, but also by the proximity of the heart to the anterior wall of the chest and by chest wall thickness. It was thus necessary to measure the late systolic outward movement (LOM) in relation to other measurable events in the left parasternal cardiogram of which the most constant are the early systolic E wave and the total area occupied by the left parasternal
The height of the "LOM" wave was measured in relation to the height of the E wave and the area contained under the LOM wave in systole (LOM area) was related to the systolic LPC area.

In our group of patients with pure mitral regurgitation when LOM/E was 3 or more and/or LOM area/LPC area 0.4 or more, the regurgitation volume was in excess of 100 ml/beat and the pulmonary wedge v wave usually exceeded 30 mm Hg. Also, when LOM/E was less than 2 and LOM area/LPC area less than 0.3 the regurgitation volume was less than 50 ml/beat and the pulmonary wedge v wave was less than 20 mm Hg. It should be emphasized, however, that this group was composed of patients with ruptured chordae tendineae of less than one year's duration (except K.F.) and patients with floppy mitral valve of whom only one (T.I.) had longstanding severe mitral regurgitation while the findings in the others were not impressive. In this group the degree of right ventricular activity represented by the E wave17-19 correlated with the severity of mitral regurgitation, which is responsible for the LOM wave in the left parasternal cardiogram.

The 18 patients in whom left parasternal cardiograms were taken twice, once before and once during cardiac catheterization, showed a mean value for LOM/E of 2.42 ± 0.33 in the initial tracings and 2.73 ± 0.28 in the second tracings. This indicates that when care is exercised in the proper placement of the microphones the left parasternal cardiograms are highly reproducible. The fact that the two tracings were obtained by two different pick-ups (Schwartzter and Electronics for Medicine) did not seem to significantly affect the reproducibility of the data. The transducers used had comparable time constants (800 msec and 650 msec, respectively) and have been shown by previous observers to give practically identical tracings.20

Findings in left parasternal cardiograms of our patients with mitral regurgitation were correlated with angiographically determined regurgitation volumes and with pulmonary wedge pressures. There are theoretical limitations of the technique of angiographic estimation of volumes in man21-24 and the degree of regurgitation may be affected by temporary depression of myocardial contractility20 and increase in left ventricular volume caused by
dye injection. Angiography, nevertheless, provides "the most accurate means presently available for determining left ventricular volumes in man." Furthermore, it has been shown that angiographic estimates of mitral regurgitation correlate well with the severity of the lesion encountered at surgery.

Similarly, pulmonary wedge pressure tracings may occasionally not reflect adequately the left atrial events, particularly in patients with long-standing mitral disease and pulmonary hypertension. Equally important is that while left atrial pressures usually reflect volume changes in patients with recent mitral regurgitation, such pressures are also influenced by the size and compliance of the left atrium. These factors may explain at least partly the lack of correlation between findings in the left parasternal cardiogram, of which LOM probably represents volume displacement, and the pulmonary wedge v wave in our patient (fig. 6D) with long-standing pure mitral regurgitation, and other patients with mixed mitral lesions.

In patients with rheumatic mitral disease, neither the LOM/E nor LOM area/LPC area correlated significantly with the angiographically determined regurgitation volume or the pulmonary wedge v wave. The majority of these patients were in atrial fibrillation which caused marked beat-to-beat variation in the left parasternal cardiogram, pulmonary wedge pressure pulse tracing, and probably regurgitant volumes consequent to changes in cycle length and ventricular filling. Atrial fibrillation can thus adversely affect measurement obtained from these recordings.

Also, the great variability in the size of the left atrium may influence the proximity of the anterior heart surface to the chest wall, and the ease with which further systolic increase in left atrial volume can lead to late systolic left parasternal lift. Armstrong, Meeran, and Grotsman did not feel that left atrial size significantly influenced the left parasternal cardiogram in their patients with severe mitral regurgitation. Our findings agree with those of Ewy, Gomez, and Marcus who demonstrated that patients with markedly dilated left atria have the largest LOM in left parasternal cardiograms. The largest LOM/E we observed was in a patient with a hugely dilated left atrium and only moderately severe mitral regurgitation. His tracing (fig. 8B) is similar to that recorded by Bedford from the right axillary region in a patient with extreme dilatation of left atrium. Conversely, longstanding pulmonary hypertension and tricuspid regurgitation tend to produce a more prominent and sustained early outward movement so that left atrial regurgitation volume may be underestimated by the LOM/E.

The coexistence of mitral stenosis seems to be another major factor which invalidates the predictive value of left parasternal cardiograms in patients with mitral regurgitation. It has been shown that in patients with both mitral stenosis and regurgitation, the severity of mitral regurgitation cannot be predicted from analysis of pressure pulse waves obtained directly from the left atrium, from pulmonary wedge position, or indirectly by esophageal balloons. Furthermore, it has been our experience, as well as of others, that some patients with isolated mitral stenosis may have left parasternal cardiograms characterized by prominent LOM and therefore be indistinguishable from those obtained from patients with mitral regurgitation (fig. 8A). It is notable that among our group with rheumatic mitral valve disease (table 2) there were patients with almost pure or greatly predominant mitral regurgitation (valve area 1.4 cm²/m² or more), and who did not show the good correlation between the hemodynamic findings and left parasternal recordings seen in patients with pure mitral regurgitation due to ruptured chordae tendineae. This suggests that even minor degrees of mitral stenosis may influence the pattern of left parasternal cardiogram in patients with rheumatic mitral regurgitation.

In addition, left parasternal cardiograms in patients with congenital mitral regurgitation associated with atrial septal defect demonstrated features of right ventricular volume overload but not mitral regurgitation. It is likely that in these patients a major portion of regurgitation volume goes preferentially into the right atrium.

In over 150 consecutive left parasternal cardiograms, the authors did not observe a late systolic outward movement in healthy adults or in cardiac patients without mitral disease or ventricular dyskinesia. A characteristic tracing can therefore substantiate the clinical diagnosis of mitral regurgitation when the auscultatory phenomena may be confused with ventricular septal defect or left ventricular outflow obstruction as is often the case with ruptured chordae tendineae. Furthermore, the left parasternal cardiogram may be valuable in the clinical assessment of severity of disease in cases of acute mitral regurgitation in whom other clinical investigations, including the roentgenograms and electrocardiograms, may not reflect the severity of regurgitation.
experience with a larger number of patients is required not only to substantiate the value of left parasternal cardiogram in the quantitative assessment of mitral regurgitation in patients with acute disease and those with floppy mitral valve but also to identify whether the data are also reliable in patients with long-standing pure mitral regurgitation of rheumatic and nonrheumatic origin.

Addendum

Since submitting this manuscript for publication we have examined the predictive value of the left parasternal cardiograms in four additional cases with severe pure mitral regurgitation, three of whom had suffered recent rupture of chordae tendineae. Utilizing LPC, the regurgitation volumes were estimated to be 76, 104, 65, and 192 ml/beat. The corresponding angiographically measured volumes were 82, 68, 67, and 169, respectively ($r = 0.94$).

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