The Role of Left Atrial Transport in Aortic and Mitral Stenosis

By Donald K. Stott, M.D., Derek G. F. Marpole, M.D., J. David Bristow, M.D., Frank E. Kloster, M.D., and Herbert E. Griswold, M.D.

SUMMARY
The relationship of left atrial contraction to ventricular filling was studied in 24 patients. Eight patients had aortic stenosis, eight had mitral stenosis, and eight others served as a control group. All had normal sinus rhythm. Cineangiocardiographic volumetric determinations of the left ventricle were done throughout the cardiac cycle, and the rate of left ventricular filling before and during left atrial contraction was calculated.

In the group with aortic stenosis 39% of the left ventricular stroke volume entered the ventricle during left atrial contraction; in the group with mitral stenosis 24% was contributed during left atrial contraction, and in the control patients, 26%.

The rate of left ventricular filling more than doubled during left atrial contraction in aortic stenosis, while no consistent change in the rate of filling occurred during left atrial contraction in mitral stenosis and in the control group.

The character of the resistance to left ventricular filling in aortic stenosis and mitral stenosis is discussed. An important contribution by left atrial contraction to left ventricular performance in aortic stenosis is suggested.

Additional Indexing Words:
Left ventricular cineangiocardiography
Left ventricular filling
Left ventricular volume
Ventricular compliance

The transport functions of the left atrium and their hemodynamic importance for cardiac performance in various disease states continue to evoke interest and controversy. It is generally recognized that the left atrium may serve as (1) a conduit for the passage of blood from the pulmonary veins to the left ventricle during diastole; (2) a reservoir for the storage of blood during ventricular systole; and (3) an actively contracting chamber, capable of providing additional energy for left ventricular filling beyond that supplied by right ventricular systole. The relative contributions of each of these functions toward left ventricular filling and, consequently, cardiac performance may vary with the specific type of cardiac dysfunction in question.

Cineangiography presently provides a method for the estimation of the cyclic changes in ventricular dimensions and volume in the intact human. Our attention was directed toward this method when cineangiograms showed the marked effect of left atrial contraction upon left ventricular filling in certain lesions. This communication reports an analysis of the influence of left atrial contraction upon left ventricular filling during the late phase of diastole in patients with aortic or mitral stenosis, based on angiographic observations.

Methods
Twenty-four patients were divided into three groups before analysis of the data. Their diagnoses and hemodynamic findings appear in...
Results from All Patients

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Mitral stenosis

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Controls

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*Postoperative study; preoperative aortic valve pressure gradient = 55 mm Hg.
†Mitral stenosis and insufficiency.
‡Small ventricular septal defect.
§Pure mild mitral insufficiency.

Abbreviations: SE = standard error of the mean; LV = left ventricular; LA = left atrial.

table 1. Eight had aortic stenosis without clinical or cineangiographic evidence of important regurgitation; selection of these patients was based upon the finding of a systolic gradient across the aortic valve. The average peak systolic gradient for these eight was 73 mm Hg (range, 14 to 110) and the average aortic valve area was 0.54 cm²/m² body surface area (range, 0.27 to 1.25). The one patient with a mild aortic valve gradient of 14 mm Hg was studied postoperatively. He had definite left ventricular hypertrophy and a preoperative aortic valve gradient of 55 mm Hg. Eight patients had mitral stenosis or combined mitral stenosis and insufficiency with an average mean mitral diastolic gradient of 14 mm Hg (range, 6 to 23), and an average mitral valve area of 0.88 cm²/m² (range, 0.48 to 1.23). Eight patients, selected for the control group, had undergone cardiac catheterization for various clinical indications and were found to be either hemodynamically normal or to have mild valvular disease. None had mitral or aortic valve gradients. The patients in this latter group had no clinical, electrocardiographic, or roentgenologic evidence of left ventricular hypertrophy.

All 24 patients had normal sinus rhythm, with an average P-R interval of 0.16 sec (range, 0.14 to 0.20).

All patients were studied by cardiac catheterization and left ventricular cineangiography.
Filming was with a 16-mm single-plane camera at 60 frames/sec using the right anterior oblique projection. Thirty to 40 ml of methylglucamine diatrizoate (Renograin 76) were injected rapidly (20 ml/sec) into either the pulmonary artery or the left atrium to opacify the left ventricular chamber throughout several cardiac cycles. The left ventricular outline was traced on each cine frame throughout two cardiac cycles. Each tracing was planimetered three times by hand or twice by electroplanimeter* to determine the circumscribed area. The major axis of the ventricle was measured directly from each tracing. The area-length measurements were corrected for magnification and distortion by use of a reference grid arbitrarily set 10 cm above the table top. 18 The fluoroscope was set at the same height that existed during the filming of the patient to approach as closely as possible the conditions present during the cineangiogram. The left ventricular volume was calculated by computer from the single plane length-area values using the methods of Sandler, 14 Dodge, 16 and Greene, 18 and their associates. Instantaneous values for length, frontal area, midplane circumference, and volume were determined by computer for each 1/60th of a second throughout two cardiac cycles. Changes in the dimensions of the cardiac chamber were plotted by computer in terms of both volume and circumference at the level of the minor axes (fig. 1). The rate of change in cardiac dimension was determined from the slope of the curve. From these plots, it was possible to calculate the rate of volume change within the ventricle as well as the rate of circumferential change of the myocardial fibers at the endocardial surface.


Circulation, Volume XLI, June 1970
In figure 1, period I represents the phase of left ventricular filling from the inception of the filling slope to the onset of left atrial contraction. This period is designated as the "passive" phase of ventricular filling. Period II denotes that portion of left ventricular filling which occurs during left atrial systole and has been designated as the "active" phase of left ventricular filling. The last 0.15 sec of diastole, as measured to the origin of the down slope of the ejection curve, was chosen as the period encompassing left atrial systole when it could not otherwise be determined directly from the plot. These distinct phases of ventricular filling have been noted previously by the use of cineangiographic technics.17

Results

The complete results are presented in tables 1 and 2.

Left Atrial Systolic Contribution to Left Ventricular End-Diastolic and Stroke Volumes

Aortic Stenosis

The volume entering the left ventricle during left atrial systole was greater in the aortic stenosis group than in the mitral stenosis (P < 0.01) or the control patients (P < 0.05). This volume averaged 40 ± 3 ml (mean ± standard error) for the aortic stenosis group and was equal to 29% of the left ventricular end-diastolic volume. Considered in terms of the total left ventricular stroke volume, left atrial forward stroke volume contributed 39% of the volume ejected during ventricular systole.

Mitral Stenosis

The volume entering the left ventricle during left atrial systole in mitral stenosis

Table 2

<table>
<thead>
<tr>
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<th>Aortic stenosis vs mitral stenosis</th>
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<td>Passive LV filling rate</td>
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<td>Active LV filling rate</td>
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<tr>
<td>Passive LV filling volume</td>
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<td>NS</td>
<td>NS</td>
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<tr>
<td>Active LV filling volume</td>
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<td>&lt;0.05</td>
<td>NS</td>
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*P < 0.10 was considered of borderline significance and < 0.05, of significance.

Abbreviations: NS = not significant; LA = left atrial; LV = left ventricular.
averaged $26 \pm 3$ ml. This amount represented 16% of the left ventricular end-diastolic volume and 24% of the left ventricular stroke volume. Due to some mitral regurgitation in three patients, the average total left ventricular stroke volume in the mitral stenosis patients was somewhat greater than in the patients with aortic stenosis or the controls. Despite this, the average left atrial forward stroke volume was one and one-half times greater in aortic stenosis than in the other two groups.

Control Patients

The volume entering the left ventricle during left atrial contraction in the controls did not differ importantly from that found in mitral stenosis, averaging $29 \pm 5$ ml. This fraction represented 20% of the left ventricular end-diastolic volume and 26% of the stroke volume.

Left atrial contraction provided a greater contribution to left ventricular filling and subsequent stroke volume in our patients with aortic stenosis than those with mitral stenosis or patients without valvular obstruction. The finding that left atrial contraction did not substantially augment left ventricular filling in the patients with mitral stenosis was unexpected and was not explicable by a low total stroke volume in the mitral stenosis patients.

Left Atrial Systolic Contribution to the Rate of Left Ventricular Filling

The average rates of left ventricular filling before and during left atrial contraction were calculated from the volume curve for each patient. An increase in the slope of the volume curve from period I to period II represents a change in the rate of left ventricular filling due to left atrial contraction.

Aortic Stenosis

Volume and circumference plots of the left ventricle in aortic stenosis demonstrated marked alteration in the shape of the curves during period II, indicating a substantial augmentation to left ventricular filling during left atrial contraction (figs. 1 and 2). The filling rate during period I averaged $156 \pm 20$ ml/sec in the eight patients with aortic

![Aortic Stenosis](image-url)
stenosis. During period II there was a consistent increase in filling rate, with an average of $353 \pm 34$ ml/sec. The left ventricular filling rate during atrial systole was nearly twice as rapid as in mitral stenosis ($P < 0.01$) and exceeded that in the control patients without aortic or significant mitral stenosis, although with borderline statistical significance ($P < 0.10$). The average filling rate during atrial systole more than doubled in the patients with aortic stenosis, while no consistent augmentation was seen in mitral stenosis or the control patients.

Left ventricular filling prior to left atrial contraction was lowest in patients with aortic stenosis, but this was not statistically different from the other groups.

**Mitral Stenosis**

Figure 3 is from a patient with mitral stenosis and is representative of that group as a whole. No great augmentation to the rate of left ventricular filling from atrial contraction was seen in patients with mitral stenosis. The rate of passive left ventricular filling (period I) averaged $199 \pm 25$ ml/sec, and this increased to only $206 \pm 26$ ml/sec during left atrial contraction (period II). Only a trivial increase in filling rate occurred despite high a-wave peak pressure and elevated mean pulmonary capillary or left atrial pressure.

**Control Patients**

Both the volume and circumference plots showed only slight alterations to the left ventricular filling curve during left atrial systole in those patients without valvular obstruction. The left ventricular filling rate for the patients in this group averaged $216 \pm 30$ ml/sec during period I, increasing to an average of $243 \pm 50$ ml/sec during period II.

**Left Atrial and Left Ventricular Pressure**

Table 1 compares left ventricular end-diastolic pressure, a-wave peak pressure, and mean left atrial or pulmonary wedge pressures in the three groups of patients. Mean left atrial pressure was higher in mitral stenosis.
than in the other two classes of patients ($P < 0.01$). The left ventricular end-diastolic pressure was somewhat higher in the patients with aortic stenosis and exceeded the mean left atrial pressure.$^1$

The peak a-wave pressure was higher in mitral stenosis than in aortic stenosis ($P < 0.05$) or the control patients ($P < 0.002$). Peak a-wave pressure was higher in aortic stenosis than in the control patients ($P < 0.05$).

**Discussion**

Not unreasonably, questions have been raised challenging the validity of angiocardiographically determined left ventricular volume data.$^{14, 15, 18, 20-24}$ These volume calculations are usually based upon an assumed ellipsoidal configuration for the left ventricle throughout the cardiac cycle and do not compensate for distortions from this ideal geometric model that are apt to occur. The deviations in the geometric shape of the ventricle and spatial orientation of the long cardiac axes are greatest during systole and could operate toward an underestimation of left ventricular end-systolic volume and thus toward overestimation of the angiocardiographically determined left ventricular stroke volume.$^{14}$ These measurement errors are of less importance in the present study, since primary attention is here directed toward the events that occur during the latter portion of left ventricular diastole. During that period, the shape and orientation of the ventricle most closely conform to the ideal ellipsoidal model. Errors which produce consistent underestimation of left ventricular end-systolic volume, with the resultant overestimation of left ventricular stroke volume, would also tend to operate toward a devaluation of the influence of left atrial contraction upon left ventricular filling rate and stroke volume.

As an alternative to volume calculations, assessment of the influence of left atrial contraction upon left ventricular filling can be obtained by considering its influence upon left ventricular end-diastolic circumference. Figure 1 depicts changes in the left ventricular midplane circumference throughout the cardiac cycle, in turn related to alterations in muscle segment length. Here, the influence of left atrial contraction upon myocardial dimension, and end-diastolic fiber length is estimated without resorting to volumetric determinations. The same phenomena are evident, as described before.

Our results are in reasonable agreement with estimation of the relative contribution of atrial systole to ventricular filling and stroke volume obtained by other methods.$^{4, 5, 7, 8, 13, 25-29}$ No clear separation has been made, however, between the conduit, reservoir, and contractile functions of the atrium. All three may operate with varying degrees of influence upon ventricular filling, depending upon the heart rate, rhythm, ventricular compliance, the contractile state of the atrium, and the functional state of the mitral valve. Where diastasis has not occurred, ventricular filling would be expected to continue to some extent during the period

**Figure 4**

The relationship between left atrial stroke volume and left atrial ejection time is shown for the aortic and mitral stenosis patients. The line of identity represents no augmentation to left ventricular filling during left atrial systole assuming that passive filling were to continue at the same rate. All patients with aortic stenosis show augmentation to left ventricular filling during left atrial contraction, while patients with mitral stenosis demonstrate less uniformity in this respect.
occupied by left atrial systole, even if effective atrial contraction did not occur. The conduit function of the left atrium would remain operative despite the absence of a coordinated atrial contraction. The true contribution of left atrial contraction to left ventricular filling, therefore, represents the augmentation to the total left ventricular filling volume over that supplied by the reservoir and conduit functions of the chamber, assuming that the passive filling would continue at the same rate. In reality, the passive filling rate might well be expected to decline with further diastolic time.

Figure 4 attempts to separate the left atrial reservoir plus conduit functions from contractile function in augmentation of left ventricular filling. The percentage of the total left ventricular filling time taken up by the left atrial contraction is represented on the abscissa, and the percentage of the total left ventricular filling volume contributed during left atrial contraction is represented on the ordinate. Values falling on the line of identity represent conditions in which left atrial contraction provides no augmentation to left ventricular filling rate, although it may be sustaining flow at this point. Values falling above the line of identity indicate positive augmentation to left ventricular filling by left atrial contraction. The distance these values fall above the line reflects the extent of

Figure 5

The relationship between passive left ventricular filling and mean left atrial pressure is shown for aortic and mitral stenosis. Patients with mitral stenosis achieve filling rates equivalent to those of patients with aortic stenosis but only at the cost of a high mean left atrial pressure.
augmentation by this active process over the preceding level of flow. Values falling below the line not only depict conditions whereby left atrial contraction fails to provide augmentation but also suggest that the passive left atrial influences upon left ventricular filling are diminishing, as would be seen when the energy provided by the elastic recoil of the atrium is dissipated. The patients with aortic stenosis all fall above the line of identity, indicating some degree of augmentation to left ventricular filling by left atrial contraction. At higher heart rates, when left atrial systolic ejection time occupies a greater proportion of the total left ventricular filling time, the total volume entering the left ventricle during left atrial contraction is highest, as would be expected. The distribution in the mitral stenosis patients appears to be more random, with the majority showing no augmentation to left ventricular filling during left atrial contraction. This does not exclude the possibility, however, that left atrial contraction operates to sustain flow across the mitral valve during the later phases of ventricular diastole when the left atrial-to-left ventricular pressure gradient has otherwise waned, and the compliance curve of the distending ventricle is steep.

The contribution of left atrial contraction to left ventricular filling is influenced by the character of the impediment to left atrial emptying.1, 4, 7, 25, 26, 30-33 Prior to left atrial systole, the mechanics of left ventricular filling depend upon energy provided by right ventricular systole, the compliance characteristics of the left atrium and ventricle, and the impedance to flow at the mitral orifice. In the absence of mitral valve obstruction, the rate of ventricular filling will depend largely upon the pressure-volume characteristics of the left ventricle. Early in diastole, when the left ventricle is most distensible and the pressure-volume curve is relatively flat, the energy needed for ventricular filling will be least and high filling rates will be produced.

In mitral stenosis a "fixed" resistance to left ventricular inflow exists, linked in series with a "variable" resistance determined by the compliance characteristics of the ventricle. The magnitude of the pressure gradient between left atrium and left ventricle largely reflects the magnitude of kinetic energy loss in overcoming this impediment to left ventricular filling. In mitral stenosis the early diastolic filling rates were not significantly lower than the passive filling rates for the other groups, but the cost for achieving these rates is a markedly elevated mean left atrial pressure (fig. 5). The a-wave pressure, produced during left atrial contraction, reflects energy transmitted to the left ventricle which should, in the absence of mitral valve obstruction, shift the ventricle's position upward on the pressure-volume curve. In mitral stenosis, however, the a-wave pressure largely reflects kinetic energy dissipated in overcoming the resistance across the valve. Despite good left atrial contractility, as reflected in the peak and pulse pressures, little augmentation to left ventricular filling was encountered in these patients (fig. 6). In addition, some kinetic energy may also be lost to left ventricular

![Figure 6](image-url)

**Figure 6**

The relationship between active left ventricular filling and peak a-wave pressure is shown for aortic and mitral stenosis. The magnitude of the a-wave pressure reflects the severity of impedance to LV filling. Patients with mitral stenosis do not show augmentation to the rate of ventricular filling despite high a-wave pressure.
filling by pulmonary vein regurgitation during atrial systole. The failure of left atrial systole to greatly augment left ventricular stroke volume in mitral stenosis, even in the presence of good a-wave pressure, has been noted previously by Carleton and Graettinger. Because of the impendiment produced by valvular narrowing, left atrial contraction may be of hemodynamic importance in mitral stenosis by serving to sustain flow across the valve at a time the pressure gradient and flow rate would otherwise fall. Recent work by Heidenreich’s group has, in fact, demonstrated the value of a properly timed atrial systole on the left ventricular peak systolic pressure and ejection time in patients with mitral stenosis.

In the hypertrophied left ventricle, as demonstrated by the patients with aortic stenosis, the filling rate before atrial systole tended to be lower than in the nonhypertrophied heart, as depicted by the control patients. This difference should reflect alterations in left ventricular compliance, with a generally steeper pressure-volume curve for the hypertrophied ventricle. In aortic stenosis, the pressure difference between the left atrium and left ventricle is small and diastasis has frequently occurred before the onset of left atrial contraction. Despite a steep slope to the pressure-volume curve in aortic stenosis, our results show that left atrial contraction contributes an unusually large flow and volume to the left ventricle. This puts it higher on the pressure-volume and length-tension curves and so augments left ventricular performance and stroke work. As has been noted previously, this effect is accomplished without unduly elevating the mean left atrial pressure. This pronounced influence of left atrial contraction upon left ventricular filling in aortic stenosis is not encountered in mitral stenosis where left ventricular filling seems largely dependent upon the existence of a high mean left atrial pressure (figs. 5 and 6).

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