Dynamic Geometry of the Left Atrium and Left Ventricle in Acute Mitral Regurgitation

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SUMMARY  The instantaneous transverse diameter of the left atrium, left ventricular free wall segment length (SEG), and the long axis of the anterior papillary muscle (APM) length were measured throughout the cardiac cycle, using ultrasonic dimension gauges together with left atrial and left ventricular pressures in 12 open-chest dogs. During atrial contraction, left atrial diameter decreased from 19.7 to 18.7 mm, while left ventricular dimensions increased simultaneously. During ventricular ejection, percent shortening was 26% in SEG and 10% in APM, while atrial diameter increased continuously to 20.5 mm, with a concomitant rise in the v wave of left atrial pressure. After normal mitral valve opening, left atrial diameter decreased rapidly simultaneously with the y descent of atrial pressure. Graded mitral regurgitation was then produced by sectioning the chordae tendinae. With moderate mitral regurgitation, end-diastolic length of the SEG increased by 27%, while extent of shortening (ΔL) was augmented by 96%. End-diastolic length of the APM increased by 7%, and ΔL was augmented by 60%. Left atrial pressure was sharply elevated, with a distinct a wave followed by the more prominent v wave. End-diastolic diameter of the left atrium was enlarged to 22.9 mm with increased atrial shortening and expansion. As mitral regurgitation was increased to a severe degree by additional chordal rupture, end-diastolic length and ΔL continued to increase both in SEG and APM. Left atrial pressure was further elevated (a wave 25 mm Hg and v wave 47 mm Hg). Left atrial end-diastolic diameter further increased to 24.9 mm. However, the amplitude of left atrial shortening and expansion decreased remarkably. In severe mitral regurgitation, isoproterenol and nitroprusside decreased left atrial pressure and diameter, restoring more forceful atrial shortening.

CARDIAC PERFORMANCE at rest or its alterations secondary to the acute imposition of mechanical load has been studied extensively in relation to the events in the left ventricle. The left atrium is also a muscular contractile chamber located in the inflow path to the ventricle, and functions as a pump as well as a blood reservoir; however, fewer studies have been directed to the role of the left atrium in ventricular filling during the normal cardiac cycle and in various pathologic circumstances. The reasons for this are the difficulty in making a precise assessment of the changes in size and geometry of the left atrium throughout the cardiac cycle and the paucity of knowledge of phasic changes in atrial diameter during filling and emptying in relation to ventricular ejection and its function under variable loading conditions.

We used an ultrasonic approach to measure cardiac dimension, which allowed simultaneous quantification of the phasic changes of left atrial diameter, left ventricular free wall segment length (SEG) and anterior papillary muscle (APM) length during each cardiac cycle. We then investigated changes induced by sectioning of the chordae tendinae, and also by pharmacological interventions.

Methods

Twelve mongrel dogs were anesthetized with sodium pentobarbital (25 mg/kg i.v.), and small supplemental doses were administered as required. Respiration was controlled by a Harvard pump delivering room air via an endotracheal tube; a thoracotomy was performed in the fifth left intercostal space, and the pericardium opened. A small silicone rubber catheter (1 mm o.d.) was positioned in the left ventricle for pressure measurements. In some dogs, a high-fidelity pressure micromanometer (Konigsberg P-22) was also inserted at the left ventricular apex through a stab incision. We used a fluid-filled catheter to obtain zero pressure reference and to calibrate the micromanometer. A high-fidelity, micromanometer-tipped catheter (Millar Instruments) was inserted into the left atrial cavity through an appendage.

We distinguished the anterior papillary muscle from the free wall of the left ventricle by tapping the depth of endocardium from the epicardial surface with an exploring needle, as described previously. A pair of ultrasonic crystals (6 MHz) was then implanted, each about 1.8 mm in diameter with a convex lens approximately 1 mm thick. The crystal, held at the end of a small Teflon tube containing its wires, was then advanced directly through the needle track into the base of the APM. The Teflon tube was withdrawn, leaving the crystal within the papillary muscle with the lens facing the muscle tip. We repeated this procedure to place the superior crystal in the APM, making needle punctures as necessary approximately 1 cm.
cephalad to the site of the first crystal. Reorientation of the lens direction by removing and reinserting a crystal through the same needle track was repeated until we obtained an optimal signal. An identical pair of crystals was applied near the endocardium of the anterior free wall of the left ventricle (between the septum and the APM) in the circumferential plane. A third pair of crystals (4 mm in diameter) was sutured externally on the anterior and posterior epicardial surfaces of the left atrium for the continuous measurement of transverse chamber diameter (fig. 1).

The details of ultrasonic technique used for the continuous measurement of dimensions have been described elsewhere. Briefly, the transit time of the sound pulse from the transmitter crystal (which is excited by a 0.2-μsec, 200-V pulse) to the opposing receiver crystal is proportional to the distance separating the crystals. The measured transit time was calibrated in 1-μsec increments against a standard signal of precisely known duration derived from a stable crystal-controlled oscillator (1000 MHz). The measured transit time was converted to the distance separating the crystals using a velocity of sound constant of 1.5 mm/μsec. The resolution capability of the instrument was a small fraction of the wavelength of the sonic signal (less than 0.05 mm).

We verified the location of the crystals at the end of each experiment. The transducer pairs were adequately oriented in the anterior wall segment in its circumferential plane close to the endocardium, and across the left atrial transverse diameter, in all 12 dogs; however, in the APM, the blind implantation failed to place the ultrasonic crystal pair exactly along the long axis and in its center in three of 12 dogs. The behavior of these three muscles appeared to be correct at control recordings, but their waveforms were markedly distorted after interventions. Therefore, the averaged data presented are from 12 dogs for free wall segment and left atrial diameter and from nine dogs for APM.

We measured left ventricular (LV) pressure by a fluid-filled catheter with a Statham P23Db transducer which was calibrated directly with a mercury manometer; the zero reference point was at the midpoint of the left ventricle. The micromanometer was calibrated by adjusting its output to the pressure measured by the open-tip catheter. Calibration of the transducer-tip catheter was accomplished electrically.

The left ventricular end-diastolic pressure (LVEDP) and systolic pressure were determined from the pressure tracing, but the rate of rise of LV pressure was not analyzed because not all pressure tracings had a sufficient fidelity for this determination.

End-diastolic dimensions of the LV free wall segment and APM were identified from the simultaneously recorded LV pressure tracing as the nadir of the pressure after atrial contraction. End-systolic dimensions were taken approximately at the nadir of the segment tracing. The measured values were normalized to a 10-mm initial dimension by dividing the observed length by the control end-diastolic length (EDL) and multiplying by 10. This procedure is necessary to compare data in different dogs, because the distance between pairs of crystals was variable and arbitrary and represents the actual values as a constant fraction of the EDL. The extent of shortening during systole was calculated as the difference between the correct end-diastolic and end-systolic lengths. The left atrial diameter immediately preceding atrial shortening subsequent to the p wave of the ECG was regarded as the atrial end-diastolic diameter (EDD).4 The excursion from the EDD to the nadir of the diameter tracing was regarded as the extent of atrial muscle shortening. The difference between the minimum and the maximum atrial diameter was considered the magnitude of the chamber expansion. Data were recorded during the experiment on an eight-channel, forced-ink oscillograph (San-Ei Instruments, Model 142-8) and were stored by use of a TEAC magnetic tape recorder (TEAC, Model R-260).

After control recordings, mitral regurgitation was produced by sectioning the chordae tendineae. A pursestring suture was placed in the small avascular area on the anterior wall of the left ventricle, 1 cm below the left circumflex coronary artery and about 2 cm to the left of anterior descending branch. A stab wound was placed in its center and a small hook was inserted to section the chordae tendineae directly, or to withdraw a chorda to cut it with small eye scissors. The wound was immediately closed by tying the pursestring suture. After stabilization, the recordings of the moderate mitral regurgitation were made. The
regurgitation was rendered severe by loosening the tie and sectioning an additional chorda.

The left atrial pressure (LAP) was fed into a storage oscilloscope on the y-axis and the left atrial diameter signal on the x-axis to produce left atrial pressure-diameter loops during individual cardiac cycle. The loops during the control period and after production of mitral regurgitation of graded severity were superimposed on one photograph. This procedure allowed us to delineate the passive pressure-length relation at each stage.

When mitral regurgitation became severe with deterioration of atrial shortening, isoproterenol (1 μg) was given i.v. by bolus injection into five dogs. In three of these five, and in four other dogs, we infused nitroprusside at a rate of 5 μg/kg/min; recordings were repeated when systolic pressure had decreased by 20% below control levels. An adequate recovery period was allowed between each of these interventions in cases when both the drugs had been given. The pharmacological study was not feasible in three of the dogs. All values were analyzed by a paired t test and expressed as mean ± SEM.

Results

Geometry of Left Atrium and Left Ventricle During the Control Period

Typical tracings from one dog during the resting control state and with graded mitral regurgitation are shown in figure 2, and all the data are summarized in table 1 and figure 3.

With atrial contraction, LAP increased from 4 to 6 mm Hg, forming a slight a wave. Left atrial diameter decreased by an average of 4.6% from the end-diastolic value of 19.7 mm. The beginning of atrial shortening followed by 20–50 msec the onset of the atrial pressure rise in the control state. This time interval shortened with the production of mitral regurgitation.

SEG and APM lengthened to a certain extent during atrial contraction and shortened during ventricular ejection, the total percent shortening averaging 26% in SEG and 10% in APM.

During ventricular contraction, left atrial diameter increased continuously to 20.5 mm until the mitral valve opened, coincident with peak v wave. The left atrial diameter changes after mitral valve opening to end-diastole were variable from plateau to rapid decrease, which was coincident with the y descent of the v wave of LAP (fig. 2, left panel).

Effects of Acute Mitral Regurgitation

With the production of a moderate mitral regurgitation after sectioning of the chordae tendineae, heart rate (HR) increased from 138 to 150 beats/min. The left ventricular end-diastolic pressure (LVEDP) increased from 4 to 19 mm Hg, while LV systolic pressure decreased from 119 to 107 mm Hg. The EDL was augmented by 27% in SEG and 8% in APM. Extent of shortening increased from 2.6 to 5.2 mm in SEG and from 1.0 to 1.7 mm in APM. Mean LAP was elevated from 6 to 17 mm Hg, with more prominent a and v waves (6 to 19 mm Hg and 8 to 29 mm Hg, respectively). Left atrial end-diastolic diameter (LAEDD) increased to 22.9 mm, while the extent of shortening increased from 0.9 to 1.9 mm. The amplitude of atrial expansion during ventricular systole also increased from 1.8 to 3.4 mm (fig. 2, middle panel).

As the mitral regurgitation was further increased by additional chordal rupture, LVEDP was elevated higher (24 mm Hg). The HR and LV systolic pressure was significantly the same as in the earlier stages. EDL continued to increase both in SEG (by 31% over control value) and in APM (by 12% over control value); extent of shortening was further augmented to 5.7 mm in SEG and 2.1 mm in APM. Mean LAP was progressively elevated to 26 mm Hg, accompanied by a more marked v wave (47 mm Hg). The atrial cavity was enlarged, with the LAEDD averaging 24.9 mm. However, the amplitude of atrial shortening and expansion diminished significantly (1.0 mm and 2.6 mm, respectively) (fig. 2, right panel).

Variations in the left atrial geometry during graded mitral regurgitation were analyzed by the superimposed atrial pressure-diameter (P-D) loops at different stages (fig. 4). In control, atrial pressure before the onset of atrial systole was low and diameter change was associated with minimal alteration in pressure, resulting in a relatively flat P-D loop (lower left). When left atrial diameter and pressure were moderately increased with the production of acute mitral regurgitation, enhanced booster function of the atrium was evident from the augmented initial counterclockwise loop. As the mitral regurgitation worsened, the left atrium operated on a higher and steeper portion of its pressure-length relationship, which was delineated by the ascending limb of each v wave and the concomitant diameter change. Here, there was little change in the diameter and a considerable alteration in pressure throughout the cardiac cycle (upper right loop).

Effects of Pharmacological Interventions on Acute Mitral Regurgitation

Isoproterenol

Bolus injection of 1 μg of isoproterenol caused an increase in average HR from 149 to 165 beats/min. The LVEDP decreased from 17 to 9 mm Hg. There was a significant increase in systolic ventricular pressure. The decrease in EDL was 9% in SEG (p < 0.005) and 2% in APM (NS). The extent of shortening of SEG and APM was unchanged. The reduction in mean atrial pressure after isoproterenol was 50%, with a 43% reduction in a wave and 57% decrease in v wave amplitudes. LAEDD was decreased from 26.1 to 23.8 mm. The amplitude of atrial shortening was augmented from 1.4 to 2.4 mm.

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Maximum atrial diameter at the peak of \( v \) wave was diminished from 28.1 to 25.4 mm (figs. 5A and 6A).

**Nitroprusside**

Nitroprusside caused a fall in LV systolic pressure of 16%, and was associated with the reduction of EDP from 23 to 10 mm Hg. The increase in HR was not significant. EDL was decreased by 11% in SEG and by 6% in APM. The extent of shortening in SEG and APM was not changed significantly. There was a significant fall in atrial pressure (mean LAP by 58%, \( a \) wave by 52% and \( v \) wave by 61%). LAEDD decreased from 24.5 to 22.6 mm with 50% restoration of atrial shortening. Maximum atrial diameter was also reduced from 26.4 to 24.8 mm (figs. 5B and 6B).

**Discussion**

The ultrasonic approach we used allowed a relatively atraumatic implantation procedure, with minimal strain on the area under study and provided sensitive and stable measurements of dynamic changes of dimension between the crystals.

It may be difficult to draw definite conclusions about the overall performance of the left atrium from the analysis of only one projection of dimension, nevertheless, identical excellent correlations were shown to exist between angiographically determined left atrial anteroposterior minor axes and left atrial volume in both normal and volume overloaded left atrium with a wide variety of lesions. Therefore, the present approach, using a single transverse diameter for evaluation of the atrial contractile function, appears valid. Payne et al. studied continuous changes in one atrial dimension in conscious dogs with this same ultrasonic method and documented an increase in atrial diameter during isovolumic ventricular systole, with decreasing dimension during ventricular ejection. They attributed the former change to the reversed mitral flow due to bulging of the mitral leaflets into the atrium and the latter to downward displacement of the mitral annulus.
### Table 1. Effects of Acute Mitral Regurgitation on the Dynamic Geometry of Left Heart and Modification by the Drug

<table>
<thead>
<tr>
<th></th>
<th>Response to acute MR</th>
<th>Modification of MR by isoproterenol</th>
<th>Modification of MR by nitroprusside</th>
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<td>Control</td>
<td>Moderate MR</td>
<td>Severe MR</td>
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<td>HR</td>
<td>138 ± 8</td>
<td>150 ± 9</td>
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<td>LVEDP</td>
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<td>LVSP</td>
<td>119 ± 5</td>
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<td>SEG EDL</td>
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<td>12.7 ± 0.5</td>
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<td>ESL</td>
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<td>p&lt;0.001</td>
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<td>ΔL</td>
<td>2.6 ± 0.4</td>
<td>5.2 ± 0.7</td>
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<td>p&lt;0.001</td>
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<td>APM EDL</td>
<td>10</td>
<td>10.8 ± 0.3</td>
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<td>ESL</td>
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<td>9.0 ± 0.1</td>
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<tr>
<td>ΔL</td>
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<td>1.7 ± 0.3</td>
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<td>LAP a</td>
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<td>x</td>
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<td>11 ± 2</td>
<td>20 ± 4</td>
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<td>v</td>
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<td>m</td>
<td>6 ± 1</td>
<td>17 ± 3</td>
<td>26 ± 4</td>
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<td>LAD EDD</td>
<td>19.7 ± 1.3</td>
<td>22.9 ± 2.0</td>
<td>24.9 ± 2.2</td>
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<tr>
<td>min</td>
<td>18.7 ± 1.2</td>
<td>21.0 ± 1.8</td>
<td>23.9 ± 2.2</td>
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<tr>
<td>max</td>
<td>20.5 ± 1.4</td>
<td>24.4 ± 2.1</td>
<td>26.7 ± 2.2</td>
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<td>Δa</td>
<td>0.9 ± 0.2</td>
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<tr>
<td>Δv</td>
<td>1.8 ± 0.3</td>
<td>3.4 ± 0.5</td>
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<td>p&lt;0.001</td>
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All values are mean ± SEM. All p values derived by the paired t test. In response to acute mitral regurgitation (MR), p = comparison of the values observed after the onset of MR to the control values; p' = comparison of moderate and severe stages of MR. In response to isoproterenol and nitroprusside, p = comparison of the data before and after the drugs.

Abbreviations: MR = mitral regurgitation; HR = heart rate; LVEDP = left ventricular end-diastolic pressure; LVSP = left ventricular peak systolic pressure; SEG = subendocardial segment length of the left ventricular anterior free wall; EDL = end-diastolic length; ESL = end-systolic length; ΔL = extent of shortening; APM = anterior papillary muscle; LAP = left atrial pressure; m = mean pressure; LAD = left atrial diameter; EDD = end-diastolic diameter; min = minimum diameter; max = maximum diameter; Δa = extent of atrial shortening; Δv = extent of atrial expansion during ventricular systole.
Contrary to their observations, we found three definite phasic changes during the cardiac cycle under control conditions: shortening of left atrial diameter with atrial contraction, continuous chamber expansion during ventricular ejection, and either a reduction in diameter coincident with the y descent of LAP or no further change after mitral valve opening until the onset of the next atrial contraction. These changes are more in accord with angiographic observations in the literature,\textsuperscript{12-14} in which atrial circumference was shown to decrease generally in symmetrical fashion with atrial contraction, although the anteroposterior dimension was always eccentric,\textsuperscript{12, 13} and was comparable to a cylinder with an almost fixed head and distensible walls attached to a piston;\textsuperscript{12} despite the lengthening of the atrial cavity due to annular displacement, the transverse diameter of the left atrium always increased during ventricular systole.

Atrial contraction was associated with concomitant expansion of the LV dimension.\textsuperscript{9} Subsequently, the APM and free wall segment began to shorten with ventricular ejection, but the extent of shortening was significantly less in the former than in the latter, supporting our previous observations.\textsuperscript{3} These differences may be explained on the structural basis that papillary muscles are the only myocardial fibers which exert a direct pull between their origin and site of attachment, and consequently they may support greater force during systole than any other parts of the myocardium, thereby rendering less shortening.\textsuperscript{3}

Mitral regurgitation was created acutely by sectioning a chorda tendinea. This model cannot be perfectly related to any of the mitral diseases in man; nevertheless, it produced a lesion similar to that which occurs with ruptured chordae tendineae of various origins. The present model also resembles the clinical syndrome of the papillary muscle dysfunction regardless of the anatomical or functional integrity, in which mitral regurgitation is due to alteration in the normal spatial relationships between papillary muscle, chordae tendineae and the atrioventricular orifice, imposing a similar mechanical load on the left heart chambers.

Acute production of mitral regurgitation resulted in two major alterations in the mechanical load on the left ventricle. One was an increase in preload as evidenced by the augmented EDL of free wall segment and APM. The other was a reduction in the afterload. Although peak wall stress could be elevated above control levels with chamber dilation, via the LaPlace relation, the more rapid reduction in ventricular size resulting from regurgitation into the left atrium may accelerate the decline of the ventricular wall stress during ejection, resulting in a further increase in the velocity and extent of shortening.\textsuperscript{16} Functional responses of the free wall segment and APM to acute mitral regurgitation were directionally similar with shortening increasing in both. Despite an enhanced total stroke excursion, effective forward flow can be reduced, depending on the amount of the regurgitation.\textsuperscript{16, 17}

As atrial diameter initially increased after the onset of acute mitral regurgitation, atrial shortening was remarkably enhanced and was associated with a prominent a wave. This may be a simple manifestation of the Frank-Starling mechanism in atrial muscle.\textsuperscript{16} When the force of atrial contraction was determined directly in vivo using a strain-gauge arch, the active tension developed by atrial myocardium was found to increase as a function of initial muscle length until a 50-60% increase above control was reached.\textsuperscript{16} In this regard, the atrial myocardium was regarded as analogous to the ventricular muscle.

When mitral regurgitation was progressively in-

\begin{figure}[h]
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\includegraphics{fig3.png}
\caption{Average data showing the effects of graded acute mitral regurgitation on the end-diastolic dimension (upper panel) and the extent of shortening (lower panel) of subendocardial segment length of left ventricular free wall (SEG), anterior papillary muscle (APM), and left atrial diameter (LAD). Twelve dogs are averaged for SEG and LAD and nine dogs for APM. C = control; M = moderate mitral regurgitation; S = severe mitral regurgitation. Numbers above the bars indicate $p$ values compared with control, and the values below the bars are a comparison of the moderate and severe stages. The brackets represent SEM.}
\end{figure}
increased by additional chordal rupture, it caused a further increase in atrial diameter, and was associated with decreased atrial shortening and expansion. The LAP was markedly elevated, with an abbreviation of the x descent and striking increase in v wave with early onset of its ascent. Although it is accepted that when a chamber is enlarged, a larger volume can be ejected with less diameter change, the diminished atrial stroke during volume overload does not appear to be effect-
tively compensated for by the geometrical advantage of a larger initial chamber size. This decline in atrial function has been attributed by other investigators to the atrial myocardium operating on a descending limb of function.\textsuperscript{5, 19} The decrease in atrial shortening may also be related to a decrease in contractile force resulting from distension of the atria above the peak of the length-active tension curve, as well as to an increased afterload to the atrial contraction due to an elevated LV diastolic pressure.

The passive P-D relation of the left atrium (ascending limb of v wave and increase of atrial diameter during ventricular systole in each stage, shown in figure 4) appears to fall on the same curve, and thus the left atrium in severe mitral regurgitation can be regarded as operating simply on a steeper portion of a single P-D relation, where the same volume increments result in larger increases in pressure than on the low, flat portion.\textsuperscript{20} Thus, atrial muscle was rendered less compliant in the face of extensive volume overload.

We previously observed in cases of experimental mitral regurgitation that inotropic intervention with isoproterenol resulted in a significant augmentation of effective stroke volume, despite the essentially unaltered total stroke volume, as measured by angiography.\textsuperscript{8} In the present experiments, isoproterenol was given to dogs with relatively low LV systolic pressure. The data obtained also confirmed the salutary effect of isoproterenol in suppressing the regurgitation, as evidenced by diminished atrial size and pressure during ventricular ejection. This effect might be related to 1) a diminished regurgitant orifice after a decreased preload,\textsuperscript{14} 2) a reduction in the time of deformation of the ventricular fluid mass and an enhancement of the rapidity and tightness of valve cusp apposition at the onset of systole in consequence of the increased speed of ventricular contraction,\textsuperscript{21} and 3) the increased contractility of atrial myocardium by isoproterenol leading to improvement of the pump function of the atrium, and to further diminution of the regurgitant area due to an enhanced contraction of the muscular portion of the annular margin.\textsuperscript{19} Although the contractile behavior of the papillary muscle and LV free wall segment in response to inotropic intervention was directionally the same, the magnitude of response was less in the former, which again may relate to the particular spatial relation of papillary muscle in the left ventricular cavity, providing for direct support of greater force acting upon the mitral valve.

Nitroprusside produces beneficial hemodynamic changes in patients with mitral regurgitation by improving forward flow.\textsuperscript{22-24} In the present study, stroke excursion of the LV free wall was somewhat diminished by nitroprusside. This response can be explained by the fact that the left ventricle with mitral regurgitation is already in an unloaded state during systole, due to regurgitation through the mitral valve into the low-pressure left atrium. Accordingly, the increase in forward flow reported in the literature\textsuperscript{22-24} does not appear to be the result of an increase in the extent of shortening of the LV free wall. The present experiments demonstrated a decrease in left atrial

\footnotesize{\textbf{Figure 4.} The superimposed atrial pressure-diameter loops at three stages of graded mitral regurgitation. Control loop at the left, loop at moderate regurgitation in the middle and at the severe stage at the right. The original photograph (lower panel) is schematized in the upper panel to indicate the end-diastolic point (thick arrow) and the direction of rotation (thin arrows). The loops rotate counterclockwise initially with active atrial contraction, and are then in a clockwise loop due to atrial expansion during ventricular systole, giving rise to a figure-eight inscription of the loops. The enhanced booster function of the atrium in the mild regurgitation is evident by the augmented initial counterclockwise loop. The left atrium with severe mitral regurgitation is operating on the upper portion of its pressure-length curve delineated by the ascending limb of v wave and the corresponding diameter change. Here the diameter changes little with a considerable change in pressure. LA = left atrial.}
**FIGURE 5.** Original tracings showing the effects of isoproterenol (A) and nitroprusside (B) in severe mitral regurgitation (MR). Drugs were administered when MR became severe with a distension of the atrial cavity and deterioration of the atrial shortening.
The decrease in regurgitation with nitroprusside was not entirely caused by a decrease in systemic impedance, but by a decrease in the size of the left-heart cavity, which brought closer together components of the mitral apparatus and increased its competence.

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