Alteration of the Left Ventricular Diastolic Pressure-Segment Length Relation Produced by the Pericardium

Kunio Shirato, M.D., Ralph Shabetai, M.D., Valmik Bhargava, Ph.D., Dean Franklin, and John Ross, Jr., M.D.

SUMMARY Left ventricular pressure and segment length were measured in seven conscious chronically instrumented dogs with the pericardium intact and 3–9 days after pericardiectomy. Diastolic pressure-length plots were obtained under control conditions and after acute volume loading followed by sodium nitroprusside infusion. In all dogs with intact pericardium, volume loading displaced the entire diastolic pressure-length curve upwards and sodium nitroprusside shifted it toward control. After pericardiectomy the pressure-segment length data during control, volume loading and sodium nitroprusside fell on a single curve (intercepts and slopes not statistically different). After dextran infusion, intrapericardial pressure rose from control 1.5 ± 0.7 mm Hg to 8.2 ± 0.5 mm Hg, and it fell to 4.8 ± 0.1 mm Hg after nitroprusside. Therefore, in acute cardiac dilatation the pericardium contributed significantly to the increased left ventricular diastolic pressure and to the fall during sodium nitroprusside infusion and appeared responsible for shifts in the diastolic pressure-segment length relation.

IT HAS BEEN POSTULATED since 1915 that one of the important functions of the normal pericardium is to protect the heart from overdistension, but experiments conducted in anesthetized open-chest animals using acute volume or pressure overloading have yielded conflicting results. It is of considerable physiologic and clinical significance whether or not the pericardium contributes to the elevations of left ventricular diastolic pressures that occur during transfusion and acute elevations of systemic arterial pressure, or to the striking reductions of diastolic pressures that accompany afterload reduction for acute cardiac failure, a phenomenon usually attributed to a reduction in preload.

We postulated that when acute cardiac dilatation is produced by overtransfusion or administration of a pressor agent, pericardial restraint contributes to the elevation of intracardiac diastolic pressures, and that when acute distension is reduced by vasodilator therapy a significant portion of the fall in ventricular diastolic pressure, and the displacement of the diastolic pressure-volume curve, are due to reduction of this pericardial restraint as cardiac volume diminishes. In order to avoid artifacts caused by experiments in the open-chest anesthetized animal, we undertook to study these questions in conscious dogs by measuring the relations throughout diastole between left ventricular diastolic pressure and the length of a segment of the left ventricular wall determined by means of implanted ultrasonic crystals. This diastolic relationship was examined during acute volume loading followed by afterload reduction with nitroprusside, before and several days after pericardiectomy.

Methods

Mongrel dogs weighing from 26.6 to 33.0 kg (average 29.4 kg) were studied. Of the seven dogs in which experiments were successful, five were studied with the pericardium intact and four were studied again after pericardiectomy; the other two dogs were studied only after pericardiectomy. For initial implantation of instruments, the dogs were anesthetized with pentobarbital (30 mg/kg) intravenously. Respiration was maintained through an endotracheal tube using a Harvard respirator, and a left thoracotomy was performed through the fifth intercostal space under sterile conditions. In the five dogs studied with pericardiectomy intact, two small incisions separated by approximately 1.5 cm were made in the anterior pericardium through which a pair of 5 MHz piezoelectric crystals 2 mm in diameter were inserted into the subendocardium of the anterior left ventricular free wall for the continuous measurement of a myocardial segment length. The position of the crystal pair was in the approximate plane of the minor equator. Another small incision (4–5 mm) was made in the pericardium 1.5 cm cephalad and anterior to the crystals through which a Tygon tube (ID 1.3 mm) was inserted for measurement of intrapericardial pressure. All of the incisions were loosely closed by suture to avoid tension on the pericardium and the electrical leads and tube were brought subcutaneously to the back of the animal’s neck. Seven to 15 days (average 9.4 days) after recovery from the operation the dogs were studied. One to three days following this study, a right thoracotomy was performed under sterile conditions and the pericardium was widely resected. After recovery from pericardiectomy (3–9 days, average 6.7 days) the study was repeated in four of the five dogs (dogs #1–4); in dog #5 a second study was not possible because of inability to train the animal to lie quietly for the experiment. In the two dogs studied only after pericardiectomy, a left thoracotomy was performed and the pericardium was opened widely at the first operation. In these two dogs, a pair of ultrasonic crystals was inserted in the anterior wall of the left ventricle...
as described above, and a Konigsberg P-22 micromanometer as well as a fluid-filled catheter (Tygon ID 1.3 mm) for pressure calibration were inserted into the left ventricular chamber through an apical stab incision. A pacing electrode was placed on the right ventricular outflow tract, and the crystal and micromanometer leads were brought subcutaneously to the back of the neck. These animals were studied seven days following recovery from operation.

**Experimental Protocol**

The dogs were premedicated with morphine sulphate (45 to 75 mg, average 60 mg) subcutaneously. Under local anesthesia a catheter tip micromanometer with fluid-filled lumen (Millar) was inserted into the left ventricle from a femoral artery and a pacing catheter was placed in the right ventricle from a jugular vein, except in the two dogs in which pacing lead and a pressure gauge had been implanted. During the studies, the dogs lay quietly on their right side and were conscious and responsive.

The same protocol was followed in the experiments performed before and after pericardiectomy. After recording the pressures in the control state, 6% dextran (37°C) was rapidly infused (56.1 ± 12.0 ml/min, mean ± SEM), the volumes infused ranging from 930 ± 70 ml in the dogs with intact pericardium to 742 ± 110 ml in the dogs studied after pericardiectomy; the difference was not significant. When the left ventricular end-diastolic pressure approached 25–30 mm Hg the drip was slowed and maintained at 30 drops per minute, and recordings during a steady-state of volume loading were made. Sodium nitroprusside infusion was then begun using a Harvard pump to achieve a fall in the left ventricular systolic pressure of 15–20 mm Hg; the infusion rates ranged from 41 to 586 μg/min (average 300 μg/min). Observations were again recorded three or more minutes after beginning the nitroprusside infusion, when the pressures, segment lengths and heart rate had stabilized. The hemodynamic measurements reported were obtained under these steady conditions, without use of cardiac pacing.

Tachycardia invariably accompanied the volume overload, and in order to achieve long diastolic cycle lengths for analysis, at each steady-state point the heart was paced at a rate slightly faster than the sinus rate; this resulted in the intermittent occurrence of early capture beats followed by pauses with cycle lengths longer than the sinus interval. The heart was paced for 15 to 30 seconds to obtain several such beats which were used for the determination of diastolic pressure-length curves. Left ventricular segment length was recorded with the left ventricular pressure both from the high fidelity device and from the fluid-filled catheter which was connected to a Statham P23Db pressure transducer. Pericardial pressure was measured in four experiments using a Statham P23Db pressure transducer calibrated against a mercury manometer. Pressures were recorded at full scales of 200 mm Hg and 40 mm Hg (left ventricular diastolic pressure) and from +10 to –10 mm Hg full scale for pericardial pressure. Points were taken at 10 msec intervals from the end of the rapid filling phase to the onset of the A wave of the high fidelity left ventricular diastolic pressure tracing simultaneously with points from the left ventricular segment length tracing to construct left ventricular diastolic pressure-segment length curves. The curves were constructed from at least two beats and the duration of diastasis exceeded 130 msec in all beats analyzed.

The catheter tip micromanometer system was calibrated externally against a mercury manometer before and after the experiment and its output was continuously compared throughout the experiment with the pressure measured through the catheter. The implanted Konigsberg gauge (two experiments) was calibrated against a simultaneously recorded pressure from the fluid-filled catheter. Zero reference for the catheters was at the mid-chest level and was checked before and after each intervention during experimental runs. The respiratory cycle was recorded by means of a thermistor placed at the dog’s nostril, and all pressure measurements were made during the resting expiratory phase of respiration. Pericardial pressure was measured as the mean of the diastolic value at that portion of the tracing used to construct the pressure-segment length curve. Data were recorded and stored on magnetic tape (Honeywell 5600) for subsequent replay and analysis on a Brush forced ink recorder at a paper speed of 100 mm/sec.

Left ventricular diastolic pressure-segment length data were fit to exponential curves29

\[ P = b e^{kL} \]

where P is the left ventricular diastolic pressure, e is the base of the natural logarithm (ln), L is left ventricular segment length, and k and b are constant. This equation may also be written:

\[ \ln P = kL + \ln b \]

Left ventricular segment stiffness (S) was calculated as the tangent to the curve at (or extrapolated to) the initial length \( L_e \) of the control curve.

After the experiment, each heart was examined grossly and sections were taken for histologic examination. On this basis, two of the nine dogs originally instrumented were rejected; one had malposition of the ultrasonic crystals and in one a dense scar had formed on the epicardial surface between the crystals. In all dogs, slight adhesions were seen around the crystal lead wires and the Tygon tube in the pericardium, but the epicardial surface of the left ventricle between the crystals was normal. Also, when pericardiectomy was performed, the pericardium removed at the time of surgery appeared normal.

Statistical analysis was performed using paired t-tests and the level of significance was taken as \( P < 0.05 \).

**Results**

The general hemodynamic and segment dimension responses to volume overload and to nitroprusside with and without the pericardium are summarized in figure 1. Left ventricular end-diastolic pressure (LVEDP) and left ventricular end-diastolic segment length (LVEDL) in the control state with the pericardium intact were not significantly different from those after pericardiectomy. Before pericardiectomy during acute volume overloading the heart rate (HR), left ventricular systolic pressure (LVSP), LVEDP, LVEDL, and pericardial pressure increased significantly (fig. 1). During nitroprusside administration all of these...
measures except the heart rate declined significantly. After pericardiectomy, acute volume loading increased the HR, LVSP, LVEDP, and LVEDL. During nitroprusside administration HR increased whereas LVSP, LVEDP and LVEDL decreased significantly (fig. 1). When LVEDL after dextran was compared only in the four dogs studied before and after pericardiectomy it increased (14.93 ± 1.18 to 15.26 ± 1.34 mm) but the change was not significant.

Diastolic Pressure-Segment Length Relations

In all of five dogs studied with the pericardium intact, the left ventricular diastolic pressure-length curve following acute volume loading shifted upward, and nitroprusside administration lowered the curve toward, but not to, the control position (figs. 2 and 3). In all six dogs studied after pericardiectomy, data points obtained during control, volume loading, and nitroprusside infusion fit a single pressure-segment length curve (figs. 2 and 3) with a strong correlation coefficient in each dog (table 1).

In the four dogs having studies both before and after pericardiectomy, the diastolic pressure-segment length curves in the control state with the pericardium intact fell almost on the curve obtained after pericardiectomy; however, the curves obtained during acute volume loading and nitroprusside infusion with the pericardium intact were shifted to the left of the combined curve obtained after pericardiectomy (fig. 2).

Before pericardiectomy, decreased slopes and increased pressure intercepts of the diastolic pressure-segment length curves were found during acute volume loading, but after pericardiectomy volume loading did not change the slopes or
the intercepts compared to control (figs. 2 and 3, table 1). When slopes and pressure intercepts were compared statistically before and after pericardectomy in the same dogs, the slopes with the pericardium intact were significantly lower and the intercepts significantly higher during both acute volume loading and nitroprusside infusion than those after removal of the pericardium. In the control state, however, slopes and pressure intercepts did not differ with the pericardium intact or removed (table 1).

With the pericardium intact left ventricular segment stiffness (S) was increased during dextran infusion ($P < 0.0025$) and slightly decreased toward control during nitroprusside infusion ($P < 0.005$). In contrast to these changes dextran infusion and nitroprusside administration
did not produce significant changes in left ventricular segmental stiffness after the pericardium had been removed (table 1).

Intrapericardial pressures

Intrapericardial pressure was close to zero, 1.5 ± 0.7 mm Hg under control conditions. It rose to 8.2 ± 0.5 mm Hg during dextran infusion and fell following nitroprusside infusion to 4.8 ± 0.1 mm Hg (fig. 1).

Discussion

Critique of Methods

We used a pair of miniature ultrasonic crystals for the continuous measurement of left ventricular segment length, an approach selected because of the simplicity and minimally traumatic nature of the implantation through the pericardium. In our experience, proper placement of the large crystals necessary for measuring chamber diameter is difficult under these circumstances and likely to result in hemopericardium. The ultrasonic crystals employed have proved reliable for measuring segment lengths or wall thickness continuously during both acute and chronic interventions in conscious animals.6-17 We have found nearly identical changes in dimensions in different regions of the left ventricle studied simultaneously.18 To validate the use of segment length to reflect overall dimensional changes, we analyzed data from four dogs previously studied in our laboratory. After pericardial opening, the animals were instrumented with crystal pairs positioned to measure internal diameter and a segment length in the same ventricular hoop. When large changes in ventricular volume were induced, the regression of segment length change on minor axis change ranged between 0.87 and 0.97. Levken et al.21 have also demonstrated that changes in segment length accurately mirror changes in left ventricular volume measured by thermodilution.

Both crystals were placed in the inner third of the myocardium, and the extent of scar formation was limited to at most 1 mm around the crystals in all dogs reported. A potential problem was adhesion formation between the myocardium and pericardium; the data used for analysis were obtained only from dogs with minor adhesions limited to the lead wires and pericardial catheter, and in which there were no adhesions or scar formation in the myocardium or epicardium subtended by the crystal pair.

Pressures recorded from the micromanometer were continuously referenced to an external transducer, and zero reference was obtained before and after each observation. The influence of respiratory variations of intrathoracic pressure on pulmonary venous return and left ventricular diastolic pressure was minimized by limiting analysis of diastolic events to data obtained during the resting phase of respiration, following active expiration. Intrapericardial pressure differs within the regions of the pericardial sac6-5 and pressure variations related to the cardiac cycle become more pronounced when intrapericardial pressure increases.12 Our intrapericardial pressure measurements were therefore approximations, but their accuracy was increased by making the measurement during the resting phase of expiration when intrathoracic respiratory pressure changes were minimal and by limiting them to the period of ventricular diastasis, when intrapericardial pressure fluctuations associated with cardiac pressure and dimension changes were minimal. Inertial and viscous properties may influence compliance during the rapid phase of ventricular filling23 and viscous forces may have been modified in post pacing beats. Diastolic pressure-length relations can also be affected by incomplete ventricular relaxation when the heart rate is very rapid,24 although tachycardia per se is not thought to alter compliance.26 We minimized rate dependent effects by confining our analyses after volume overload to the longest available periods of diastasis; also, the pacing method used to procure long diastolic pauses was employed in the same
### Table 1. Changes in Pressure-Segment Length Curves Induced by Volume Loading Followed by Afterload Reduction before and after Pericardiectomy

<table>
<thead>
<tr>
<th>Condition</th>
<th>N</th>
<th>k</th>
<th>ln b</th>
<th>R</th>
<th>S</th>
<th>N</th>
<th>k</th>
<th>ln b</th>
<th>R</th>
<th>S</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pericardium intact</td>
<td>5</td>
<td>1.979 ± 0.682*</td>
<td>-28.35 ± 12.03</td>
<td>0.88 ± 0.97</td>
<td>2.33 ± 0.78</td>
<td>6</td>
<td>0.921 ± 0.141</td>
<td>-11.02 ± 2.32</td>
<td>0.76 ± 0.98</td>
<td>2.73 ± 0.95</td>
<td>NS</td>
</tr>
<tr>
<td>Dextran</td>
<td>4</td>
<td>1.426 ± 0.356</td>
<td>-16.67 ± 3.51</td>
<td>0.88 ± 0.97</td>
<td>2.57 ± 0.96</td>
<td>4</td>
<td>0.956 ± 0.183</td>
<td>-11.71 ± 3.32</td>
<td>0.76 ± 0.98</td>
<td>1.96 ± 0.79</td>
<td>NS</td>
</tr>
<tr>
<td>Nitroprusside</td>
<td>4</td>
<td>0.376 ± 0.054</td>
<td>-2.52 ± 0.75</td>
<td>0.88 ± 0.99</td>
<td>4.16 ± 0.75</td>
<td>6</td>
<td>1.068 ± 0.209</td>
<td>-12.72 ± 2.71</td>
<td>0.83 ± 0.96</td>
<td>2.57 ± 1.04</td>
<td>NS</td>
</tr>
<tr>
<td>Combined</td>
<td>4</td>
<td>0.374 ± 0.069</td>
<td>-0.26 ± 0.75</td>
<td>0.88 ± 0.99</td>
<td>4.43 ± 0.90</td>
<td>4</td>
<td>1.197 ± 0.295</td>
<td>-14.01 ± 3.75</td>
<td>0.89 ± 0.96</td>
<td>1.67 ± 0.97</td>
<td>NS</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Pericardium removed</th>
<th>N</th>
<th>k</th>
<th>ln b</th>
<th>R</th>
<th>S</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Control vs Dextran</td>
<td>5</td>
<td>0.05</td>
<td>0.05</td>
<td>—</td>
<td>0.0025</td>
<td>NS</td>
</tr>
<tr>
<td>Control vs Nitroprusside</td>
<td>5</td>
<td>0.05</td>
<td>NS</td>
<td>—</td>
<td>0.005</td>
<td>NS</td>
</tr>
<tr>
<td>Dextran vs Nitroprusside</td>
<td>5</td>
<td>NS</td>
<td>0.05</td>
<td>NS</td>
<td>—</td>
<td>NS</td>
</tr>
</tbody>
</table>

*Mean ± SEM.

"Control" refers to the single curve that fit the data obtained in the 3 experimental conditions after pericardiectomy. Slopes and intercepts and correlation coefficients are based on curves fitted to two bas. Stiffness is calculated from k, ln b, and ln s of one beat. R: correlation coefficient; other abbreviations in text.

### Functions of the Pericardium

Several of our observations favor the hypothesis that the pericardium restrains the heart from overstretch in the face of acute circulatory stress. With the pericardium intact, diastolic length of the left ventricle and ventricular pressure-volume relations are conserved. After pericardiectomy, filling pressures in the left ventricle increased. These changes were due to the left ventricular pressure gradient and the increased filling pressures in the atria and atrioventricular chamber. The increase in filling pressures resulted in an increased diastolic length of the left ventricle. The increase in diastolic length of the left ventricle was caused by the increase in left ventricular filling pressures.

### Notes

1. The choice of the reference point for the pericardial pressure-volume curve is important. The reference point is chosen based on the location of the pericardial fluid. The pericardial fluid is located in the pericardial sac, which surrounds the heart. The pericardial fluid pressure is used as the reference point for the pericardial pressure-volume curve.

2. The pericardial pressure-volume curve is used to determine the effect of the pericardium on the heart. The pericardial pressure-volume curve is a plot of the pericardial pressure against the pericardial volume. The pericardial pressure-volume curve is used to determine the effect of the pericardium on the heart.

3. The pericardial pressure-volume curve is used to determine the effect of the pericardium on the heart. The pericardial pressure-volume curve is a plot of the pericardial pressure against the pericardial volume. The pericardial pressure-volume curve is used to determine the effect of the pericardium on the heart.
increase during hypervolemia such that the transmural ventricular diastolic pressure remained at 2–3 mm Hg until the left ventricular end-diastolic pressure exceeded 10 mm Hg and the limit of pericardial distensibility was reached. In studies of the distensibility of the canine left ventricle in open-chest dogs, resuture of the pericardium altered the end-diastolic pressure-circumference curve of the left ventricle: from 0 to 10 mm Hg, the curves before and after resuture were superimposed, but above 10 mm Hg the curve inscribed with the pericardium resutured became steeper than the curve obtained with the pericardium open. It was concluded that the pericardium was of little importance at lower ventricular diastolic pressures but assumed increasing importance in determining the pressure-circumference curve at higher diastolic pressures. When dogs previously subjected to pericardiectomy or to sham operation were volume overloaded, left ventriculography yielded larger left ventricular end-diastolic and end-systolic volumes in the animals which had undergone pericardiectomy. The same investigators studied patients five days following the appearance of acute mitral regurgitation; they found that the abnormalities of left and right ventricular end-diastolic pressure contours resembled those observed in constrictive pericarditis and ascribed the phenomenon to the restrictive effect of the unstretched pericardium. The pressure-volume relation of the nonbeating canine left ventricle was studied before and after removal of the pericardium by means of a balloon inserted through the mitral valve into the left ventricle immediately after the animal had been killed. It was observed that in the presence of an intact pericardium, pressures were elevated and volumes reduced over the entire range of pressures studied when compared to results after removal of the pericardium; the greatest difference was noted at high left ventricular volumes.

From our observation that curves obtained in the control resting state in conscious dogs were not altered by pericardiectomy, we conclude that under normal circumstances the pericardium exerts minimal, if any, restrictive influence on the left ventricle. The data points which show increased ventricular pressure at short segment lengths after dextran infusion are attributed to increased volume of the venae cavae, the atria and right ventricle which are more distensible than the left ventricle. When dilated, these intrapericardial structures therefore undoubtedly bring the pericardial influence into play.

The differences in left ventricular pressure-segment length curves with volume loading before and after pericardiectomy indicate that increased pericardial pressure contributes significantly to the increased left ventricular diastolic pressure accompanying acute cardiac dilatation. These findings lead us to speculate that the pericardium may play a role in the displacement of diastolic pressure-volume curves and the lowering of filling pressures observed in the clinical setting. However, in a study relating left ventricular end-diastolic volume to end-diastolic pressure in patients with various types of heart disease, a wide range of volumes was found for given levels of filling pressure: in patients with chronic heart disease, volumes three to four times normal were found with diastolic pressures that were normal. Moreover, studies in patients with chronic cardiac dilatation have shown that intrapericardial pressures may be nearly normal. Finally, species differences may exist, since man, unlike the dog, frequently alters cardiac volume by alterations in posture. Thus our results in acutely volume overloaded dogs cannot be directly extrapolated to patients with chronic heart failure.

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References

Cardiac Function in the Normal Newborn

Additional Information by Computer Analysis of the M-Mode Echocardiogram

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SUMMARY Computer analysis of the M-mode echocardiogram in 50 normal newborns provided measurements of wall thicknesses and chamber size and, in addition, assessment of right and left ventricular wall, septal, and cavity dynamics throughout the cardiac cycle. Data obtained with this new technique indicated that (1) right and left ventricular cavity functions are similar in the normal newborn, (2) right and left ventricular cavity filling and emptying vary directly with peak rates of septal and ventricular wall thinning and thickening, respec-

tively, and (3) there is a close time relationship among maximum left atrial dimension, minimum left ventricular dimension, and mitral valve opening. This analysis, which is the first complete analysis of the echocardiogram in the newborn, provides a normal range of septal and ventricular wall dynamics as well as right and left ventricular and left atrial function and has clinical implications in that it may allow early recognition of both congenital and perinatal myocardial disease.

M-MODE ECHOCARDIOGRAPHY lends itself well to the study of normal infants; it is an invaluable tool in the comprehension of cardiac anatomy and in elucidating the relations between the atrioventricular valves and the ventricles and between the ventricles and the great vessels in congenital heart disease.6 7 Hitherto, attention has been confined mainly to the anatomic disposition of cardiac structures and estimation of chamber size.6 8 10 There is relatively little information regarding right or left ventricular function except for estimates of atrioventricular valve closing velocities,6 10 13 systolic time intervals,15 16 and mean circumferential fiber shortening.17

In order to investigate right and left ventricular function in the newborn in greater depth and to examine left atrial function and its contribution to left ventricular filling, we analyzed M-mode echocardiograms by computer. This technique provides measurements of instantaneous right and left ventricular and left atrial cavity dimensions and also instantaneous thickness of the septum and ventricular walls throughout the cardiac cycle. From these measurements, which are obtainable manually only with difficulty, peak rates of systolic thickening and diastolic thinning of the right and left ventricular walls and septum were obtained so that their respective contributions to right and left ventricular filling and emptying could be assessed.

Methods

Echocardiograms and simultaneous electrocardiograms were obtained from 50 clinically normal newborns ranging in age from 1/2 to 64 hours, with a mean age of 19 hours. Twenty-six were female and 24 were male; they ranged in weight and body surface area from 2.6 to 4.5 kg and 0.18 to 0.28 m², respectively.

Echocardiograms were obtained with an Ekoline 20 ultrasonoscope using a 0.5-inch diameter, unfocused, 5 MHz transducer with a repetition frequency of 1,000 cycles/second. Recordings were made on a Cambridge Scientific Instruments multichannel strip-chart recorder at paper speeds of 100 mm/sec with simultaneous electrocardiograms while the patients were recumbent. Echoes of the right and left sides of the septum, endocardium, and epicardium of the right and left ventricular walls were obtained at the level of the mitral valve (fig. 1). Aortic root and left atrial wall echoes were recorded at the level of the aortic valve (fig. 2). Measurements were made only when these echoes were clear and continuous throughout the cardiac cycle. Echocardiograms were digitized as previously described18 with the use of a Science Accessories Corporation Graf pen and digitizing table, and the data were processed by a CDC 3500 computing system operating the master time-sharing mode. Echoes were calibrated with

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