SUMMARY The diastolic viscous properties of the intact heart were studied in seven conscious dogs and in four right heart bypass preparations. The passive elastic properties were first determined by fitting a linear relation between the natural logarithm (ln) of the left ventricular pressure and the diameter or between ln of the diastolic wall stress and the strain (r = 0.93-0.99). When the velocity of lengthening was less than 1 diam/sec, the pressure and stress data fell within the 95% confidence interval of the static elastic curve, both at low and high strain. At velocities of lengthening greater than 1 diam/sec, the pressure and stress data always deviated from the passive elastic relationship, but the magnitude of this deviation was not linearly related to the velocity of lengthening. When pressure and stress data at high velocities of lengthening were compared, the pressure or stress deviation from the passive elastic curve was always greater at high diameter than at low diameter. Moreover, the pressure and stress data at the time of minimum left ventricular pressure after systole showed greater deviation from the passive curve than expected from the diameter and the velocity of lengthening alone (+8 mm Hg in conscious dogs, P < 0.002; +2.4 mm Hg in open-chest dogs, P < 0.05), suggesting incomplete ventricular relaxation at that time. We conclude that the viscous properties of the left ventricle, like those of isolated cardiac muscle, are not linear but increase with length. These observations, together with the influence of incomplete ventricular relaxation in early diastole, preclude the determination of diastolic properties from only one diastolic cycle.
Methods

This study was performed on two different groups of dogs. In this section we will describe first the methods used in the group of open-chest dogs using a right heart bypass preparation and then the methods used in the conscious instrumented dogs.

Open-Chest Studies

Experimental data were obtained from four mongrel dogs (mean weight 27.7 kg, range 22-32 kg), anesthetized with sodium pentobarbital (25 mg/kg, iv). Ventilation was maintained by a Harvard respirator with room air enriched with oxygen. After a median sternotomy and bilateral thoracotomy, the heart was suspended in a pericardial cradle. A 3 mm i.d. stainless steel cannula was inserted into the left ventricle through a stab incision at the apex and connected to a Statham P23Db strain gauge manometer. The frequency response of the stainless steel cannula coupled to the manometer was flat until 100 Hz; the zero drift throughout the experiment (60 minutes) was less than 0.2 mm Hg, and gain was linear up to 200 mm Hg. A micromanometer (Millar SF catheter, model PC482) was introduced through the right femoral artery with its tip located just above the aortic valve. The micromanometer and its amplifying unit, after prewarming, produced through the right femoral artery with its tip located just above the aortic valve. The micromanometer and its amplifying unit, after prewarming, was recorded at necropsy. All data, together with a standard lead II electrocardiogram, were simultaneously recorded on paper and on FM magnetic tape for further computations.

Experimental Protocol

While a constant venous return was maintained, control data were recorded at a mean aortic pressure that ranged between 100 and 120 mm Hg. Thereafter, 3- to 4-minute steady states, at levels of left ventricular end-diastolic pressure ranging from 0 to 29 mm Hg, were produced by the following sequence of maneuvers: aortic clamping below the left subclavian artery (mean aortic pressure, 130-150 mm Hg); phenylephrine infusion (0.095-0.380 mg/min; mean aortic pressure, 130-150 mm Hg); nitroprusside infusion (0.195-0.760 mg/min; mean aortic pressure, 70-90 mm Hg). Between each of these hemodynamic interventions, the preparation was allowed to return to the control state for 1-2 minutes. At least one increase and one decrease in venous return during the basal state also were performed randomly before or after the above sequence. These changes in venous return produced steady states in left ventricular end-diastolic pressure ranging from 3.5 to 11 mm Hg (mean aortic pressure, 90-120 mm Hg).

After completion of this protocol, the heart was arrested with KCl given intravenously. The left ventricular long axis (aortic valve to apex), as well as left ventricular mass (excluding both atria and right ventricular free wall), were determined. Crystal positions also were checked at that time. Since in each dog the posterior crystal was positioned on the endocardium of the posterior papillary muscle which tended to bulge into the left ventricular cavity, the measured diameter as used for the computations was generally 3-5 mm less than the maximum left ventricular diameter at this level as recorded at necropy. All data, together with a standard lead II electrocardiogram, were simultaneously recorded on paper and on FM magnetic tape for further computations.

Measurements and Computations

All the hemodynamic interventions described above were performed at steady state in each dog at a constant heart rate [average, 111 ± 6 (SE) beats/min; range, 99-121 beats/min]. Representative beats were digitized every 5 msec. The constancy of inotropic state was defined by the linear relation \( r = 0.96-0.99 \) found between end-systolic wall stress and end-systolic diameter (Pouleur et al., 1978). The period of diastole was defined arbitrarily as beginning at the minimum left ventricular pressure after ventricular systole and ending at the minimum pressure after atrial systole (Rankin et al., 1977). The minimum left ventricular diameter frequently occurred 5-15 msec before the minimum left ventricular pressure.

Left ventricular wall stress was calculated from the digitized data by an EAI 590 hybrid computer, using the following formula for a thick walled ellipsoidal model:

\[
\text{Stress} = \frac{PR_i (1 - 2R_i^2/L^2)/(R_o - R_i)}{
\text{Where} \ P = \text{left ventricular pressure,} \ L = \text{left ventricular long axis,} \ R_i = \text{measured internal radius,} \ R_o = \text{external radius computed, assuming a constant left ventricular mass (Burns et al., 1971).}
\]

The midwall strain at the minor equator was normalized using a natural strain definition, \( \epsilon = \ln (l/l_0) \), where \( l \) is strain, \( l \) is the midwall circumference, calculated as \( \pi \) (diameter + wall thickness),

\[
\text{DIASTOLIC VISCOS PROPERTIES/Pouleur et al. 411}
\]
and $l_0$, the minimum end-diastolic circumference measured during either nitroprusside infusion or a decrease in venous return at a diastolic pressure of 0–0.5 mm Hg. Thus, a single value of $l_0$ was determined in each dog and used in all the computations of all the data for that animal. The velocity of chord shortening ($V$) in mm/sec was calculated as $\Delta$ diameter/$\Delta t$, where $\Delta t$ was taken as 10 msec and was normalized in diameter/sec by dividing by the diameter at $l_0$. The study of the left ventricular dimensions at the minor equator seemed suitable, since diastolic filling occurs principally in the circumferential dimension (Hawthorne, 1961).

Using linear least squares regression analysis, we obtained the data from each beat during each hemodynamic maneuver in the 6–8 steady states when $V$ was negligible ($0 + 1$ mm/sec) and fitted them to the following equations:

$$\ln \sigma = a + b \varepsilon$$

$$\ln P = a' + b' \text{diam.}$$

These relations and their respective 95% confidence intervals were assumed to reflect the control static elastic properties of the left ventricle. The minimum left ventricular pressure-diameter or stress-strain relationship also could be fitted using similar exponential relations:

$$\ln \sigma = a'' + b'' \varepsilon$$

$$\ln P = a''' + b''' \text{diam.}$$

The decay of left ventricular pressure from the aortic dicrotic notch to the minimum left ventricular pressure also was studied by plotting left ventricular pressure against time on semilogarithmic paper. This relation was best described by two sequential exponential relations, and the inverse slopes of these relations defined the time constant, $T'$, for left ventricular pressure fall (Weiss et al., 1976).

Conscious Dog Studies

Seven mongrel dogs, ranging in weight from 22 to 28 kg (average 25 kg), underwent a left 5th interspace thoracotomy under sterile conditions during sodium thiopental anesthesia (25 mg/kg). A high-fidelity micromanometer (Konigsberg P-20) and a Silastic rubber catheter (i.d., 1.1 mm) for pressure calibrations were inserted at the left ventricular apex through separate stab wounds. Pacing electrodes were sutured to the left atrial appendage. Two 4.5-mm ultrasonic piezoelectric crystals (Stegall et al., 1967; Mahler et al., 1974) were positioned at opposing sites on the anterior and posterior left ventricular endocardial surfaces, to measure continuously the internal diameter of a transverse left ventricular chord (Horwitz et al., 1968). The pacing wires, ultrasound crystal and micromanometer leads, and the left ventricular catheter were implanted subcutaneously in the neck where they could be exposed easily for subsequent study. The dogs were permitted to recover from the operation for an average of 2 weeks and were trained to lie quietly on the right side. At the time of each study, they were vigorous and healthy and had normal temperatures and hematocrits ranging from 34 to 42%.

Measurements were recorded on an eight-channel forced ink pen oscillograph (Hewlett-Packard, model 7868A). The output from the micromanometer was adjusted to the pressure measured through the fluid-filled catheter by means of a Statham P23Db transducer, which was calibrated with a mercury manometer; the zero reference point was at the level of the vertebral column. The tracings from the high-fidelity transducer were recorded at full scale and at high gain for accurate reading of diastolic pressure. The signals from the ultrasonic diameter gauges were calibrated as previously described (Mahler et al., 1974). This system permits continuous recording of left ventricular internal diameter. The error in left ventricular diameter measurement induced by angular distortion up to $30^\circ$ is less than 4% of the total measured distance (Kirkpatrick et al., 1973).

The experiments were performed 2 weeks to 4 months after operation while the unsedated dogs were lying quietly on the experimental table. For control studies, atrial pacing was performed at various rates so that heart rates could be matched with the experimental studies. The mean paced heart rate was 108 ± 3 beats/min. A constant current stimulator (Nuclear-Chicago, model 7150) was used for atrial pacing. Volume studies were performed by infusing 250–400 ml of Ringer's lactate over 3–5 minutes, through a catheter inserted into a peripheral vein. After volume infusion, left ventricular end-diastolic pressure averaged 17.1 ± 0.8 mm Hg (range, 15–20 mm Hg) and the mean heart rate was 117 ± 5 beats/min. To ensure sufficient venous return and to avoid wide variations in the values for left ventricular end-diastolic pressure in individual dogs during phenylephrine infusion (0.1–0.2 mg/min), volume infusion always was carried out before phenylephrine administration. With this method, it then was consistently possible to achieve left ventricular end-diastolic pressures during phenylephrine infusions, ranging from 20 to 33 mm Hg (average, 27.0 ± 1.9 mm Hg) at a mean heart rate of 109 ± 2 beats/min. During these interventions, pretreatment with atropine and atrial pacing prevented reflex alterations in heart rate.

In each dog, data were obtained from beats recorded during the expiratory phase of respiration at rapid paper speed (100–200 mm/sec). For each control and intervention (volume, phenylephrine) study, 10 left ventricular pressure and diameter data points were selected as follows: the point of minimum left ventricular pressure; 3 points during the rapid filling phase; 3 during the slow filling
FIGURE 1 Changes in left ventricular diameter and velocity of lengthening (dD/dt) observed in one open-chest dog at different levels of left ventricular end-diastolic pressure. The arrows indicate the time of minimum left ventricular pressure. It can be seen that peak dD/dt occurs after this point. (A = nitroprusside at 190 μg/min; B = control; C and D = phenylephrine at 0.095 and 0.110 mg/min, respectively.)

phase, and 3 at end-diastole. All points were at least 10 msec apart. Velocity of chord lengthening was calculated and normalized in the same manner as for the right heart bypass preparation with diameter taken as the smallest diameter obtained under control conditions.

From each of the selected beats, static elastic properties were determined by fitting the equation,

\[ V = c \cdot D \]

where \( V \) is the velocity of chord lengthening, \( D \) is the diameter, and \( c \) is a constant. The constants were determined by least-squares fitting the data points to the equation.

FIGURE 2 On panel A, the relation between left ventricular pressure and diameter data obtained in an open-chest dog when the velocity was 0 mm/sec (closed circles) is illustrated on a semilogarithmic scale. The regression line and its 95% confidence interval also are illustrated. The open circle represents data observed at identical velocity (64 mm/sec) during the rapid filling period of two different beats. It can be seen that the point of higher diameter deviates more from the regression line than does the point of lower diameter (7 mm Hg vs. 1.4 mm Hg). On panel B, similar relations are illustrated in a conscious dog.

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TABLE 1  Static Elastic Properties of the Left Ventricle

<table>
<thead>
<tr>
<th>No.</th>
<th>n</th>
<th>( r )</th>
<th>( \alpha )</th>
<th>( \beta )</th>
<th>( \alpha' )</th>
<th>( \beta' )</th>
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<tr>
<td>1</td>
<td>7</td>
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<td>0.998</td>
</tr>
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<td>0.000003</td>
<td>35.26</td>
<td>-3.97</td>
<td>0.954</td>
</tr>
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</table>

**Open-chest dogs**

<table>
<thead>
<tr>
<th>No.</th>
<th>n</th>
<th>( r )</th>
<th>( \alpha )</th>
<th>( \beta )</th>
<th>( \alpha' )</th>
<th>( \beta' )</th>
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<td>5</td>
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<td>0.0008</td>
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<tr>
<td>8</td>
<td>5</td>
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<td>0.009</td>
<td>0.912</td>
<td>-43.06</td>
<td></td>
</tr>
<tr>
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<td>0.003</td>
<td>0.254</td>
<td>-5.13</td>
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<tr>
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<td>0.924</td>
<td>0.001</td>
<td>0.438</td>
<td>-7.58</td>
<td></td>
</tr>
<tr>
<td>11</td>
<td>6</td>
<td>0.966</td>
<td>0.002</td>
<td>0.545</td>
<td>-8.55</td>
<td></td>
</tr>
</tbody>
</table>

**Conscious dogs**

In \( P = \alpha + \beta \) diam, where \( P \) and diameter were data obtained when \( V = 0 \pm 2 \text{ mm/sec} \). Since intrapleural pressure was not measured in these studies, the pressure data corresponded to intraluminal pressures referred to atmospheric pressure. Thus, some early diastolic pressure values in the control state were negative. Therefore, to allow computations of the logarithmic data, we analyzed only the data in which pressure values were positive.

**Results**

Figure 1 illustrates changes in left ventricular diameter observed in one open-chest dog at different levels of left ventricular end-diastolic pressure. Figures 2–4 illustrate typical pressure-diameter relations observed both in open-chest and conscious dogs.

**Static Elastic Properties of the Left Ventricle**

In the four open-chest dogs, the data obtained, when \( V \) was negligible and at pressure values ranging between 1.0 and 25–30 mm Hg, could be fitted accurately, as illustrated in Figure 2A, by an exponential relation between pressure and diameter or midwall stress and midwall strain (Table 1). The data at both ends of the relation were excluded from the exponential fittings and from subsequent analysis when either pressure or diameter changed by 10% and 1%, respectively, without any modification of the other parameter.

In the conscious dogs, since the errors made while neglecting transmural pressure are greater at low pressure, the analysis of the static properties was limited to the data in a higher range of intraluminal pressures (lowest values between 3 and 6 mm Hg; highest values between 20 and 30 mm Hg) and diameter. In this range, as illustrated in Figure 2B, the static data could be fitted very reasonably by a single exponential relation. Data at higher pressures
TABLE 2 Deviation from the Static Curve at Two Different Diameters and at Identical Velocity

<table>
<thead>
<tr>
<th></th>
<th>ΔP (mm Hg)</th>
<th>ΔStress (g/cm²)</th>
<th>Diameter (mm)</th>
<th>Strain</th>
<th>Velocity (mm/sec)</th>
<th>V normalized (diam/sec)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Low diameter</td>
<td>4.7 ± 1.2</td>
<td>28.7 ± 3.3</td>
<td>68 ± 16</td>
<td>NS</td>
<td>NS</td>
<td>NS</td>
</tr>
<tr>
<td>High diameter</td>
<td>9.2 ± 2.0</td>
<td>30.6 ± 3.6</td>
<td>68 ± 15</td>
<td>NS</td>
<td>NS</td>
<td>NS</td>
</tr>
</tbody>
</table>

Conscious dogs (n = 7)

<table>
<thead>
<tr>
<th></th>
<th>ΔP (mm Hg)</th>
<th>ΔStress (g/cm²)</th>
<th>Diameter (mm)</th>
<th>Strain</th>
<th>Velocity (mm/sec)</th>
<th>V normalized (diam/sec)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Low diameter</td>
<td>2.2 ± 0.5</td>
<td>13.2 ± 2.7</td>
<td>31 ± 3.6</td>
<td>0.107 ± 0.0260</td>
<td>68 ± 10</td>
<td>2.17 ± 0.48</td>
</tr>
<tr>
<td>High diameter</td>
<td>5.5 ± 0.5</td>
<td>14.2 ± 3.2</td>
<td>41.2 ± 3.2</td>
<td>0.168 ± 0.009</td>
<td>68 ± 10</td>
<td>2.17 ± 0.48</td>
</tr>
</tbody>
</table>

Open-chest dogs (n = 4)

All data are expressed as mean ± 1 SE. P = pressure; V = velocity; n = number of dogs; NS = not significant.

Length-Dependency of the Deviation from the Diastolic Curve during Rapid Filling

Figure 3 shows that most of the diastolic points in a single filling cycle deviated from the static relation obtained from points in several cycles when V was negligible. This deviation appears greater at larger diameters. To confirm this observation, data at identical levels of V were selected during the rapid filling phase of two beats with different diameters. As illustrated in Figure 2, A and B, these data were selected only in the portion of the diastolic relation in which a good fitting of the static data was obtained. The deviation in mm Hg or g/cm² from the passive curve then was calculated by deriving the expected left ventricular pressure, or stress from the equations for the static curve (In P = a + βdiam, Table 1), and subtracting the expected from the observed values, which then were compared by the paired t-test. The data in Table 2 demonstrate that, despite identical velocity of lengthening, the deviation in mm Hg or g/cm² from the static curve is always greater when the diameter is greater.

Lack of Deviation from the Static Curve during the Slow Filling Period

Theoretically, if the deviation from the static curve were related linearly to the strain rate, this deviation should be zero only when V is zero. Figure 4 illustrates, on a logarithmic scale, the relation and 95% confidence intervals between pressure and diameter, and Figure 5, between stress and strain when V = 0 in an open-chest dog. A complete diastolic cycle also is illustrated and values of V at several points are noted. Points with a high V (rapid filling and A wave) fell out of this confidence interval but, during slow filling, points with velocities between 0.45 and 1.0 diam/sec fell within the interval. This observation suggests that, below a critical velocity, the viscous deviation should be small and not different from the experimental error. This is confirmed by the data in Table 3 where, at high diameter and low V (0.64 ± 0.08 diam/sec in conscious dogs and 0.69 ± 0.13 diam/sec in open-chest dogs), no significant deviation from the static relation was demonstrated.
Deviation from the Passive Curve in Very Early Diastole

Figure 3 indicates that the minimum diastolic pressure (solid arrows) generally deviated more from the passive relation than did a pressure value taken at the same diameter, but occurring during the rapid filling phase of another beat (dotted arrows). Figure 1 indicates that such deviation cannot be explained by a greater V at that time since the peak V generally occurs 10–40 msec after the minimum diastolic pressure [see also first (Δ) and second (○) points in Figure 5, separated by 25 msec]. Table 4 confirms that, despite lower velocities of lenthening and identical strain or diameter, the greatest values for pressure and stress always are observed in very early diastole. When data points corresponding to the minimum diastolic pressure were obtained from five to eight different beats in each dog and fitted to an exponential relation, the slope always was greater than that derived from any other diastolic point. For wall stress vs. strain, the slopes in early and late diastole (velocity = 0) were 46.197 ± 8.470 vs. 32.269 ± 4.627, respectively (P < 0.05). For pressure vs. diameter, they were 0.509 ± 0.012 vs. 0.383 ± 0.070 (P < 0.025).

Ventricular Relaxation and Early Diastolic Pressure

The influence of ventricular relaxation on the deviation from the static relationship in early diastole was investigated in the open-chest dogs before and after phenylephrine infusion. Following phenylephrine infusion, end-systolic pressure was elevated (85 ± 13 mm Hg control and 126 ± 13 mm Hg phenylephrine), the rate of relaxation was significantly attenuated (28.5 msec control, 53.8 msec phenylephrine, first component; 23.8 msec control, 32.3 msec phenylephrine, second component). However, the duration of relaxation estimated as the time from the aortic diastolic notch to minimum diastolic pressure was not different following phenylephrine (133 msec ± 11 control, 132 msec ± 13 phenylephrine), indicating that the attenuation of relaxation rate and higher end-systolic pressures may have contributed to the higher minimum diastolic pressure following phenylephrine (4.8 ± 1 mm Hg control, 14.8 ± 1.2 mm Hg phenylephrine) and further deviation from the static curve.

Discussion

Our data indicate that the viscous properties of the left ventricle during the rapid filling phase of diastole are dependent on length and are negligible at low velocity. The critical level of velocity appears to be 1 diam/sec. These observations are in agreement with the report of Noble (1977) who demonstrated both length and velocity dependence for the viscous properties of cat papillary muscle; the crit-

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**Table 3: Lack of Deviation from the Static Curve at High Diameter and Low Velocity**

<table>
<thead>
<tr>
<th></th>
<th>Conscious dogs (n = 7)</th>
<th>Open-chest dogs (n = 4)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Observed pressure (mm Hg)</td>
<td>10.9 ± 2.7</td>
<td>13.1 ± 3.0</td>
</tr>
<tr>
<td>Predicted pressure at this diameter (mm Hg)</td>
<td>10.4 ± 2.5</td>
<td>NS</td>
</tr>
<tr>
<td>Diameter (mm)</td>
<td>30.8 ± 3.8</td>
<td>17.2 ± 2.6</td>
</tr>
<tr>
<td>Velocity (mm/sec)</td>
<td>16 ± 4</td>
<td>17.1 ± 2.6</td>
</tr>
<tr>
<td>Normalized velocity (diam/sec)</td>
<td>0.64 ± 0.08</td>
<td>NS</td>
</tr>
</tbody>
</table>

All data are expressed as mean ± 1 SE.
tical level of velocity he reported was 1 muscle length/sec. Parnley et al. (1976) also showed that, at low stretching rates in cat papillary muscle, no significant viscous properties were detected. Similarly, the data of Lundin (1944) and of Loeffler and Sagawa (1975) can be interpreted as showing that viscous properties are length dependent. Others have suggested that viscous properties could be important in determining left ventricular diastolic properties (Mursky, 1976; Alderman and Glantz, 1976; Brodie et al., 1977; Sonnenblick et al., 1966), but to date, the influence of viscosity during diastole has not been quantified in the intact ventricle. As one potential solution to this problem, Rankin et al. (1977) recently proposed that the viscous properties of the whole heart essentially were linear.

One possible reason for the difference between our observations and those of Rankin et al. is that different models were used for wall stress calculation and strain normalization procedures. However, our data suggest that, during acute interventions, such as changes in left ventricular volume and pressure with constant wall mass, conclusions drawn from both left ventricular wall stress calculations and from pressure-diameter data do not differ. This is so because, in our study, deviations in pressure or stress from the static curve always were calculated at identical levels of strain (Tables 2-4), and therefore, the wall thickness and diameter data used in stress calculations will not influence the results. We used a simple exponential equation to define the static properties of the left ventricle properties (in \( a = a + \beta e \), instead of the equation \( \sigma = a(e^\gamma - 1) \) employed by Rankin et al. (1977). However, use of the latter approach offers no advantage since it merely forces the intercept to the origin without altering the deviation from the passive curve that we observed and which also can be seen in Figure 3 of Rankin et al. (1977).

Another difference between our experimental approach and that of previous workers is the use of transmural, as opposed to intraluminal, pressures in our conscious dogs. Although the first approach is theoretically more suitable, careful selection of the respiratory phase during which data are obtained can minimize any potential error, as far as comparisons between the two studies are concerned. However, it should be recognized that in both studies the deviations from the static curve calculated at low pressures probably are underestimated. Indeed, one can show, in a ventricle subject to both cavity and external pressure, that the circumferential stress is of the form: Stress = [(luminal pressure − external pressure) × (geometrical factor − a function of the external pressure)]. Therefore, in both studies in conscious dogs, all data should be corrected by a constant factor which is a function of the pleural pressure; this correction is proportionally more important at low values of intraluminal pressures. However, even if we postulate a constant pleural pressure of —5 mm Hg, the differences observed in Table 2 remain significant, although decreased. This point is confirmed by our data in open-chest dogs in which the external pressure was the "0" reference pressure.

Another difference between our experimental approach and that of Rankin et al. (1977) is the position of the crystals, which could produce a difference in the timing of the events (minor and major equator in their study, minor equator only in our study). Nevertheless, strain and changes in dimension with time have been calculated at the minor equator in both studies, and the striking similarity between our data (both in open- and closed-chest dogs) and the stress-strain relations reported in a previous study (Rankin et al., 1977) suggests that the differences in the conclusions depend largely on the model used to interpret the data and on a more complete examination of the diastolic properties at high end-diastolic pressures in our experimental protocol, rather than on major differences in the basic experimental findings.

Indeed, if an algorithm using a linear viscous element is designed to fit the diastolic stress-strain
relation, the value of this viscous element will adapt automatically to represent the deviation from the static elastic curve, whether or not this deviation is due to a viscous factor. Consequently, if part of this deviation is due to incomplete ventricular relaxation or inertia, or if the viscous factor is not linear, the number found by the algorithm cannot be interpreted as an index of the viscous properties only.

Evidence that factors other than viscosity and inertia could play a role in early diastole has been furnished by Mitchell et al. (1960), who showed that inadequate time for ventricular relaxation could influence the pressure deviation of the passive pressure-length elastic curve. Subsequently, it has been well documented that ventricular relaxation may be influenced by various interventions, such as acute alterations in heart rate, blood pressure, and administration of inotropic agents such as isoproterenol and calcium (Karliner et al., 1977).

Under conditions of our experiments, an increase in left ventricular end-diastolic pressure was always accompanied by an increase in end-systolic pressure. Since the duration of isovolumic relaxation was unchanged and velocity of relaxation was reduced, it is clear that, during very early filling, residual levels of left ventricular pressure will be higher, thus contributing to deviation from the static curve.

Another factor that could play a role in early diastole is inertia of the left ventricular walls (Horwitz and Bishop, 1972; Mitchell et al., 1960). Inertial forces generally are neglected during systole because, according to Tallarida et al. (1970), they represent only 1 or 2% of the static force, i.e., about 1–3 g/cm². Nevertheless, during rapid filling, left ventricular wall acceleration may be of the same magnitude as during systole, particularly in conscious dogs, and consequently, the inertial forces could reach a level of 1–3 g/cm². In some cases this could represent 20–30% of the early diastolic wall stress. A reexamination of this factor, particularly when left ventricular mass is changing, such as during hypertrophy, therefore may be useful.

Further evidence that the deviation in early diastole cannot be explained by a viscous element only is that this deviation was already maximal when left ventricular pressure was minimal before peak V, and therefore before the peak strain rate had been reached (Fig. 1). This observation is consistent with the report of Horwitz and Bishop (1972) but not with the strain rate data reported by Rankin et al. (1977), which were maximal at the level of minimal left ventricular pressure. This would suggest that, in their experimental conditions, the left ventricular wall already was moving before this point, indicating either a shape change during true isovolumic relaxation or early filling before left ventricular relaxation is complete, as recently demonstrated (Tsakiris et al., 1978).

In conclusion, our data suggest that the viscous properties of the left ventricle are closely related to those of isolated cardiac muscle and are length dependent and negligible at low velocity. Moreover, pressure and stress values obtained in early diastole probably are affected by incomplete relaxation of the left ventricle under some experimental conditions, particularly when the end-systolic wall stress is allowed to vary, or if changes in contractility occur. The clinical implications of these data are 2-fold. First, the estimation of the diastolic properties of the whole ventricle, based on the fitting of only one diastolic cycle, could be affected by the position of this cycle relative to the static pressure-dimension curve. Second, our data suggest that incomplete ventricular relaxation and viscous properties by themselves could affect the level of mean left atrial pressure, particularly when end-systolic wall stress and diameter are elevated.

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