The Deformational Characteristics of the Left Ventricle in the Conscious Dog

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SUMMARY We studied left ventricular minor and major axis diameters and equatorial wall thickness in eleven conscious dogs with chronically implanted pulse-transit ultrasonic dimension transducers. Left ventricular transmural pressure was measured with micromanometers. Left ventricular volume was varied by inflation of implanted vena caval or aortic occluders. The geometry of the left ventricle was represented as a three-dimensional ellipsoidal shell. Left ventricular eccentricity was found to be a linear function of ventricular volume during both diastole and ejection. However, the relationship was not the same for diastole and ejection, and during diastole the left ventricle was more spherical at large volumes and more elliptical at small volumes than during ejection. The rearrangements in geometry observed during isovolumic contraction appeared to be transitional stages from the diastolic to the ejection-phase relationship. Thus, during isovolumic contraction, the left ventricle became more elliptical at large volumes and more spherical at small volumes. These relationships were not altered significantly by increased afterload or inotropic interventions. We also observed that the diastolic deformation of the ventricular chamber occurred in a set and predictable manner that seemed to be determined by the three-dimensional mechanical properties of the myocardium. The geometric interrelationships of the ventricular wall determined the relationship between diastolic transmural pressure and mural stress. These findings probably reflect basic structural characteristics of the myocardium and provide a convenient method for quantitatively representing the dynamic geometry of the left ventricle. Circ Res 49: 843-855, 1981

ANALYSIS of myocardial function in the intact heart requires a thorough understanding of the dynamic geometry of the left ventricle. As first hypothesized by Woods (1892) and recently verified by several investigators (Hefner et al., 1962; Burns et al., 1971; McHale and Greenfield, 1973), the geometry of the ventricular chamber is a major determinant of the force within the ventricular wall. This principle is an embodiment of the Laplace relationship (Laplace, 1839) which predicts that the tensile stress within any shell is a function of the distending pressure, the radii of curvature, and the mural thickness. Because diastolic and systolic stresses are important determinants of myocardial function, it is possible that primary alterations in left ventricular geometry could affect overall cardiac performance adversely, independent of changes in the intrinsic functional capabilities of the myocardium. Thus, a better understanding of the geometric characteristics of the left ventricle could improve our ability to differentiate between normal and abnormal myocardial function in the intact heart.

Advances in pulse-transit sonomicrometry have allowed precise measurements of left ventricular geometry in the experimental animal (Theroux et al., 1974; Rankin et al., 1976). It has been shown that the dynamic contraction pattern of the left ventricle is not constant but changes with variations in the physiological state of the preparation (Rusher, 1954; Leshin et al., 1972; Rankin et al., 1976). The present report develops this qualitative observation into a quantitative description of the dynamic geometry of the left ventricle in the conscious dog.

Methods

The preparation and techniques of data collection used in these studies have been described in previous communications (Rankin et al., 1976, 1977). Briefly, 11 mongrel dogs were anesthetized with intravenous sodium pentobarbital (25 mg/kg) and underwent implantation of pulse-transit ultrasonic dimension transducers on the left ventricle to permit measurement of minor axis diameter, major axis diameter, and equatorial wall thickness. Silicone rubber pneumatic occluders were positioned around both venae cavae, and a silicone rubber tube was left in the pleural space to allow the measurement of intrapleural pressure. Another silicone rub-
The geometry of the left ventricle was presented as a three-dimensional prolate spheroidal shell. The validation of this geometric model has been presented previously. The left ventricle through the implanted left atrial tube was studied in the conscious state, resting quietly on its right side. The dimension transducer connectors were exteriorized under local anesthesia (5 ml of 1% lidocaine) and coupled directly to a sonomicrometer. The flow probe leads were connected to a Statham TTFQ series electromagnetic flow probe implanted on the ascending aorta. The azygous vein was ligated, and the pericardium was left widely open. After the connectors had been tunnelled to a subcutaneous pouch, the thoracotomy was repaired.

Seven to 10 days after implantation, each dog was studied in the conscious state, resting quietly on its right side. The dimension transducer connectors were exteriorized under local anesthesia (5 ml of 1% lidocaine) and coupled directly to a sonomicrometer. The flow probe leads were connected to a Statham M4001 electromagnetic flowmeter. A Millar PC-350 micromanometer was passed into the left ventricle through the implanted left atrial tube and positioned fluoroscopically between the minor axis diameter transducers. An identical manometer was passed into the pleural cavity to the surface of the mid-left ventricle. Measurements of minor axis diameter, major axis diameter, equatorial wall thickness, aortic blood flow, left ventricular intracavitary pressure, and intrapleural pressure were recorded on magnetic tape with a Hewlett-Packard 3520-B FM recorder. Data were obtained during a control period and then during several transient venae cavae occlusions which decreased left ventricular diastolic transmural pressure to 0 mm Hg. In four of the dogs, data also were collected during the steady state infusion of isoproterenol (0.03 μg/kg per min). In four other dogs, measurements were obtained during inflation of the aortic occluders to a stable peak-systolic left ventricular pressure of approximately 200 mm Hg. The performance characteristics of the equipment used in this study have been presented in detail elsewhere (Rankin et al., 1976, 1977).

The analog data were digitized at 5-msec intervals by an IBM-1130/System 7 computer and analyzed as previously described (Rankin et al., 1976). The geometry of the left ventricle was presented as a three-dimensional prolate spheroidal shell. The dynamic internal volume of the defined shell (V) was calculated by means of the formula:

\[ V = \frac{\pi}{6} (b - 2h)^2 (a - 1.1h) \]  

where b is the external minor axis diameter, h is the equatorial wall thickness, and a is the external minor axis diameter. The validation of this geometric model has been presented previously. The left ventricular minor axis diameter vs. volume relationship was calculated for the diastatic and ejection phases of the cardiac cycle. The equatorial wall thickness vs. volume relation was calculated similarly.

The calculated intracavitary volume was normalized according to the Lagrangian equation:

\[ e = \frac{V - V_0}{V_0} \]  

where \( e \) is the normalized volume, V is the instantaneous volume, and \( V_0 \) is the volume obtained at 0 mm Hg transmural pressure. The dynamic eccentricity (e) at the midwall of the shell was determined from the equation:

\[ e = \frac{\sqrt{(a - 0.55h)^2 - (b - h)^2}}{(a - 0.55h)} \]  

Eccentricity, as defined here, refers to the degree of deviation of a geometric figure from a circle. A circle has an eccentricity of 0; a straight line has an eccentricity of 1.

The relationship between eccentricity (e) and normalized volume (\( e_v \)) was determined for each ventricle during the diastatic phase of diastole and the ejection phase of systole. Diastatic data were defined by the computer program. Data from multiple cardiac cycles during a vena caval occlusion were selected, and the periods of diastasis and ejection were defined over the entire physiological range of ventricular volumes. The diastatic and ejection phase relationships between e and \( e_v \) were fitted with linear regression analyses and the regression constants calculated. Similarly, in four dogs, e-\( e_v \) data along with the corresponding linear regression constants were obtained during the infusion of isoproterenol (0.03 μg/kg per min). Within each study, isoproterenol data were compared to control data using a t-test of the linear regression constants. In four other experiments, e-\( e_v \) relationships were determined during acute increases in peak-systolic pressure by inflation of aortic occluders and were compared to control data in a similar fashion.

Diastolic midwall minor axis circumference (\( \xi \)) of the assumed ellipsoid was calculated using the equation:

\[ \xi = \pi (b - h) \]  

Major axis midwall circumference (\( \xi_m \)) was computed using the formula:

\[ \xi_m = 4(a - 0.55h) E(K) \]  

where \( K^2 = 1 - (b - h)^2/(a - 0.55h)^2 \) and was evaluated as an elliptical function \( E(K) \) between 0 and \( \pi/2 \) according to \( E(K) = \frac{\pi}{2} \sqrt{1 - K^2} \sin^{-1} x \) dx, x being the function of integration. The minor and major axis circumferences and the wall thickness measurements were normalized to a natural strain definition:

\[ e = \ln \left( \frac{\xi}{\xi} \right) \]  

where e is the strain, \( \xi \) is the instantaneous dimen-

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sion measurement, and \( \delta_0 \) is the value of the dimension at 0 mm Hg transmural pressure determined during a maximal vena caval occlusion. The normalized midwall minor axis circumference was termed the latitudinal strain, the normalized midwall major axis circumference the longitudinal strain, and the normalized wall thickness was called the wall thickness strain. Positive strains in the wall thickness direction were used to denote wall thinning.

The diastolic deformational characteristics of each left ventricle were determined from vena caval occlusion data. Diastolic latitudinal strain (\( \varepsilon_L \)) was plotted vs. wall thickness strain (\( \varepsilon_h \)) and also vs. longitudinal strain (\( \varepsilon_L \)) over the entire physiological range of ventricular volumes, and the data were fitted to the equations:

\[
\varepsilon_L = K_{\delta h} \times \varepsilon_h \quad (7a)
\]

and

\[
\varepsilon_L = K_{\delta \phi} \times \varepsilon_\phi \quad (7b)
\]

using linear regression analyses. The constants \( K_{\delta h} \) and \( K_{\delta \phi} \) defined the relative deformational properties of each ventricle.

The minor axis midwall radius-to-wall thickness ratio (\( r/h \)) was calculated for each ventricle during diastasis and ejection, and the \( r/h \) relationship was determined over the entire range of physiological volumes during caval occlusions. The diastatic and ejection phase relationships were compared using a \( t \)-test of the linear regression constants.

The transmural pressure of the left ventricle was computed from the digitized data as the difference between the intracavitary pressure and the intrapleural pressure. The mean tensile stress in the minor axis circumference (latitudinal stress) was calculated using the formula:

\[
\sigma_L = \frac{P_{rb}}{h} \left[ 1 - \frac{r_b^3}{r_a^2(2r_b + h)} \right] \quad (8)
\]

where \( \sigma_L \) is the latitudinal stress, \( P \) is the transmural pressure, \( r_b \) is the minor axis midwall radius, \( r_a \) is the major axis midwall radius, and \( h \) is the equatorial wall thickness. This equation has been validated previously in our laboratory (McHale and Greenfield, 1973). The mean tensile stress in the major axis circumference (longitudinal stress) was computed using the equation:

\[
\sigma_L = \frac{P_{rb}^2}{h(2r_b + h)} \quad (9)
\]

where \( \sigma_L \) is the longitudinal stress (Sandler and Dodge, 1963). Average stress in the wall thickness direction (wall thickness stress), \( \sigma_h \), was calculated as one-half of the transmural pressure.

The diastatic relationship between transmural pressure and the calculated latitudinal tensile stress was determined by fitting data from multiple diastoles during a vena caval occlusion to the formula:

\[
\sigma_L = P \times K_{\sigma \phi} \quad (10a)
\]

where \( K_{\sigma \phi} \) is the linear regression constant. Similarly, the relationship between diastolic pressure and the calculated longitudinal stress was derived from the equation:

\[
\sigma_L = P \times K_{\sigma \phi} \quad (10b)
\]

where \( K_{\sigma \phi} \) is the linear regression constant. The ejection phase relationships between \( \sigma_L \) and \( \sigma_L \) vs. transmural pressure were calculated in a similar manner and the regression constant \( K_{\sigma \phi} \) and \( K_{\sigma \phi} \) were determined. The ejection phase and diastatic data were compared with a \( t \)-test of the linear regression constants.

The static elastic characteristics of the myocardium in the latitudinal, longitudinal, and wall thickness directions were determined from vena caval occlusion data. In each occlusion, the periods of diastasis (\( dc/dt = 0 \sec^{-1} \pm 5\% \sec^{-1} \)) were selected automatically by the computer program from successive diastoles as left ventricular volume progressively decreased. Using a nonlinear regression computational algorithm based on a Gauss-Newton iterative technique that employed a Taylor Series linearization (Draper and Smith, 1966), the static stress-strain data in each of the three directions were fitted to the equation:

\[
\sigma = a(\varepsilon_L^b - 1) \quad (11)
\]

where \( a \) and \( b \) are nonlinear static elastic coefficients. Latitudinal, longitudinal, and wall thickness static elastic coefficients were calculated for each ventricle. The static elastic coefficients from all the dogs for each of the three directions were compared statistically with the Hotelling's \( T^2 \) test employing the pooled within-groups covariance matrix analysis of the logarithmically transformed coefficients.

In the statistical analysis, linear relationships were fitted with a standard least squares linear regression analysis, and the slope (m), intercept (b), and correlation coefficient (r) were calculated. Data were compared with the use of Student’s two-tailed \( t \)-test for paired data.

Results

Representative dimension, flow, and pressure data are shown in digital form in Figure 1. Data at two different end-diastolic volumes are presented to illustrate the volume-dependence of left ventricular dynamic geometry. At normal end-diastolic volumes, isovolumic contraction (indicated by segment IC in Fig. 1A) was accompanied by a decrease in minor axis diameter and an increase in major axis diameter; left ventricular shape became more elliptical. During isovolumic relaxation (segment
IR), the opposite shape change occurred and the left ventricle became more spherical. However, as ventricular volume progressively decreased during vena caval occlusion, the isovolumic contraction pattern gradually changed, so that at small end-diastolic volumes, a spherical shape change was observed (Fig. 1B). The dynamic geometric patterns observed in the conscious dog and the validation of this experimental model have been presented previously (Rankin et al., 1976).

Mathematically, these findings were expressed in Figure 2A by relating left ventricular cavitary volume, shown on the abscissa, to midwall chamber eccentricity, shown on the ordinate. An eccentricity of 0.750 is more elliptical than that of 0.625. The wide range of data was obtained from multiple cardiac cycles during the course of a transient vena caval occlusion. During the diastatic phase of filling, a linear relationship was observed between eccentricity and volume (filled circles, \( r = -0.975 \)) with the chamber becoming considerably more spherical as volume increased. During ejection, however, a different linear relationship was obtained (open circles, \( r = -0.982 \)), and the ventricle became only slightly more elliptical as volume decreased. Thus, the dimensional changes in the major and minor axis diameters during ejection were more concentric than during filling. As shown in Figure 2A, the diastatic and ejection phase relationships consistently intersected. Because of the intersection of curves, changes from the diastatic to the ejection phase during isovolumic contraction produced an elliptical shape change of the chamber at large volumes and a spherical shape change when the volume was small. This is graphically depicted in Figure 2B.

In the dog illustrated in Figure 2A, the point at
which the diastatic and ejection phase curves intersected (the 'transitional volume') occurred at the lower range of the physiological ventricular volumes. From dog to dog, there was considerable variation in the observed transitional volume (Table 1). In some dogs the two curves intersected at larger volumes (Fig. 3A) and, in others, at smaller volumes (Fig. 3B). However, the same basic type of relationship was noted in every study. The regression data for the eccentricity-volume relationships determined for each dog are given in Table 2. The normalized volume ($e$) was used in the regression analysis to eliminate any dog-to-dog variability due to differences in ventricular size.

During infusion of isoproterenol (Fig. 3A), end-diastolic volume was decreased and stroke shortening was augmented, but the diastatic and ejection phase eccentricity-volume relationships were unchanged from control in every dog ($P > 0.20$). During inflation of the aortic occluders, the peak systolic left ventricular pressure increased from 126 ± 6 mm Hg to 195 ± 13 mm Hg ($P < 0.01$). The end-diastolic volume uniformly increased and stroke volume decreased. The control eccentricity-volume relationships was not altered ($P > 0.10$) in any study by this degree of increased afterload (Fig. 5).

A graphic representation of the dimensional interrelationships of the left ventricle is shown in Figure 4. An inverse linear relationship was consistently observed between midwall minor axis circumference and wall thickness over the entire physiological range of ventricular volumes (Fig. 4A). Conversely, a direct linear relationship existed between midwall minor axis circumference and midwall major axis circumference (Fig. 4B). As was observed with the eccentricity-volume data, different relationships were present during diastasis (filled circles) and ejection (unfilled circles). Therefore, the three-dimensional systolic and diastolic deformation of the left ventricle appeared to occur in a set and predictable manner.

When the diastatic dimension data were normalized as an extension from the unstressed length (Fig. 5), linear relationships were observed between the diastolic deformation in each of the three directions. At any given ventricular volume, wall thickness deformation was slightly greater than lateral deformation in most dogs, and deformation in the longitudinal direction was relatively small. The regression constants for the three-dimensional geometric interrelationships of each ventricle are presented in Table 2.

Shown in Figure 6A is a typical epicardial minor axis diameter to left ventricular volume relationship. For the 11 studies, a direct linear correlation was observed over the entire physiological range of volumes during diastasis ($r \geq 0.978$) and ejection ($r \geq 0.974$). The slopes of the diastatic and ejection relationships differed due to alterations in the dynamic chamber geometry between these two phases; diastatic slope was 0.029 ± 0.002 cm/ml, whereas ejection slope was 0.025 ± 0.001 cm/ml ($P < 0.001$). An inverse relationship was observed between wall thickness and left ventricular volume (Fig. 6B). Correlation coefficients for these relationships were similar to those obtained for minor axis diameter vs. volume (diastasis, $r \geq 0.912$; ejection, $r \geq 0.969$) whereas the slopes were −0.005 ± 0.0006 cm/ml for diastasis and −0.009 ± 0.0010 cm/ml for ejection ($P < 0.01$).

Radius was also inversely related to wall thickness. Individual and mean regression data for these relationships are listed in Table 3. The $r/h$-ventricular volume relationship for one study is graphically presented in Figure 7 and showed a direct linear relationship.

**Table 1 Three-Dimensional Diastolic Elastic Properties and $e$ Values Obtained in Conscious Dogs**

<table>
<thead>
<tr>
<th>Dog</th>
<th>$e_{0}$ (cm)</th>
<th>$e_{10}$ (cm)</th>
<th>$e_{20}$ (cm)</th>
<th>$V_{0}$ (ml)</th>
<th>$V_{1}$ (ml)</th>
<th>$e_{0}$ $e_{10}$</th>
<th>$e_{20}$</th>
<th>$a$</th>
<th>$b$</th>
<th>$R_{ms}$</th>
<th>$MS$</th>
<th>$a$</th>
<th>$b$</th>
<th>$R_{ms}$</th>
<th>$MS$</th>
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<tbody>
<tr>
<td>1</td>
<td>11.96</td>
<td>1.27</td>
<td>16.94</td>
<td>20.64</td>
<td>22.80</td>
<td>0.828</td>
<td>0.794</td>
<td>1.43</td>
<td>9.79</td>
<td>1.87</td>
<td>0.85</td>
<td>5.41</td>
<td>0.12</td>
<td>1.53</td>
<td>15.46</td>
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<td>2</td>
<td>10.05</td>
<td>1.19</td>
<td>15.99</td>
<td>14.90</td>
<td>10.48</td>
<td>0.874</td>
<td>0.804</td>
<td>0.64</td>
<td>9.84</td>
<td>0.30</td>
<td>1.11</td>
<td>5.90</td>
<td>0.43</td>
<td>2.29</td>
<td>11.80</td>
</tr>
<tr>
<td>3</td>
<td>12.90</td>
<td>1.43</td>
<td>16.83</td>
<td>21.39</td>
<td>34.40</td>
<td>0.760</td>
<td>0.730</td>
<td>2.06</td>
<td>9.35</td>
<td>0.56</td>
<td>0.97</td>
<td>5.80</td>
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<td>17.69</td>
<td>24.73</td>
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<td>0.772</td>
<td>4.96</td>
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<td>17.32</td>
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<td>0.850</td>
<td>0.708</td>
<td>0.36</td>
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<td>1.99</td>
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<td>16.69</td>
<td>16.11</td>
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<td>0.788</td>
<td>1.27</td>
<td>7.89</td>
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<td>6.01</td>
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<td>Mean</td>
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<td>17.26</td>
<td>22.05</td>
<td>48.34</td>
<td>0.833</td>
<td>0.767</td>
<td>1.02</td>
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<td>1.15</td>
<td>0.51</td>
<td>8.38</td>
<td>1.76</td>
<td>0.76</td>
<td>21.73</td>
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</table>

$e_{0}$ = length at zero load, $V_{0}$ = volume at zero load, $V_{1}$ = transitional volume, $e_{0}$ = eccentricity at zero load, $e_{10}$ = eccentricity at transitional volume, $e_{20}$ = eccentricity at 20 mm Hg, $a$ = longitudinal, $h$ = wall thickness, $\phi$ = longitudinal, and Res MS = residual mean square. $a$ and $b$ are the non-linear static elastic coefficients determined by regression analysis of the diastolic stress-strain relationships.

*Expressed as dynes x 100 cm$^{-2}$.

†$P < 10^{-4}$ Holtinger T test of $a$ and $b$ vs. $\theta$ vs. $h$, $h$ vs. $a$, and $h$ vs. $\phi$. 
relation during diastasis and ejection; however, the slopes were not significantly different for this as well as the other experiments \((P > 0.07)\).

The relationships that were typically observed between diastatic transmural pressure and the calculated three-dimensional stresses are shown in Figure 8. The pressure-stress relationships were linear in all three directions \((r \geq 0.985)\). At any given pressure, the calculated stress was greatest in the latitudinal direction and least in the radial direction. During ejection, similar linear relationships were observed between calculated stress and transmural pressure \((r \geq 0.704)\). The linear regression constants \(K_{pf}\) and \(K_{ps}\) were reduced by \(17 \pm 2.7\%\) and \(23.8 \pm 3.6\%\), respectively, from the corre-
Discussion

The development of pulse-transit sonomicrometry has provided an excellent method for measuring cardiac dimensions in either acute or chronic experimental preparations. This technique provides accurate, high-fidelity analog dimension signals that are easily calibrated and are not subject to significant temperature sensitivity or drift. The three-dimensional transducer configuration used in the present study has been validated experimentally and has been shown to estimate left ventricular mass, volume, and stroke volume with a reasonable degree of accuracy in the normal dog (Rankin et al., 1976).

Although the ultrasonic method of measuring wall thickness is probably the most precise yet devised, several technical considerations are essential to obtain satisfactory data. It is important to use an endocardial crystal of 2 mm or less in diameter and to tunnel the crystal obliquely across the wall so that the myocardium in the area of measurement is not traumatized. The connecting wires should be very fine and flexible to minimize tethering of the endocardial crystal. It is also important to align the crystals as directly as possible across the ventricular wall. This is accomplished by securing the epicardial crystal at the point of minimum diastolic thickness and maximum systolic excursion. In most dogs, this method results in a measurement that is very close to, but probably not exactly, that of a direct wall thickness. This inaccuracy may be responsible for the slight underestimation of volume change that has been reported with this technique (Rankin et al., 1976). In addition, there appears to be some regional variability in wall thickness measurements (Rankin et al., 1977).

**TABLE 2 Dynamic Eccentricity-Volume Strain and Geometric Relationships Obtained in Conscious Dogs**

| Dog | b   | m   | SEE | r   | h   | m   | SEE | r   | K_m | r   | K_s | r   |
|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|-----|
| 1   | 0.815| -0.049| 0.004| -0.984| 0.811| -0.027| 0.002| -0.974| 0.90  | 0.973| 2.06| 0.964|
| 2   | 0.871| -0.039| 0.002| -0.966| 0.836| -0.021| 0.001| -0.941| 0.95  | 0.964| 2.26| 0.990|
| 3   | 0.763| -0.055| 0.007| -0.966| 0.751| -0.037| 0.003| -0.983| 0.91  | 0.977| 1.73| 0.968|
| 4   | 0.859| -0.056| 0.008| -0.956| 0.783| -0.013| 0.006| -0.792| 1.01  | 0.952| 2.40| 0.966|
| 5   | 0.805| -0.045| 0.003| -0.990| 0.791| -0.026| 0.002| -0.988| 0.96  | 0.960| 1.65| 0.994|
| 6   | 0.811| -0.076| 0.002| -0.993| 0.786| -0.032| 0.002| -0.946| 0.91  | 0.963| 2.17| 0.993|
| 7   | 0.848| -0.049| 0.003| -0.996| 0.823| -0.026| 0.002| -0.988| 0.99  | 0.976| 2.19| 0.988|
| 8   | 0.816| -0.057| 0.005| -0.975| 0.788| -0.034| 0.002| -0.882| 1.22  | 0.984| 1.86| 0.994|
| 9   | 0.839| -0.044| 0.004| -0.977| 0.803| -0.021| 0.001| -0.887| 1.18  | 0.954| 1.99| 0.988|
| 10  | 0.848| -0.068| 0.002| -0.998| 0.763| -0.020| 0.004| -0.864| 1.06  | 0.992| 2.42| 0.998|
| 11  | 0.870| -0.033| 0.002| -0.993| 0.826| -0.016| 0.001| -0.882| 1.03  | 0.993| 2.07| 0.967|

The slope constants (m), intercepts (b), correlation coefficients (r), and sum of error of the estimates (SEE) for linear regression analysis of diastatic (D) and ejection phase (E) eccentricity (e)-normalized volume (v) relationships are given. K_m and K_s are the linear slope constants of equations 7A and 7B respectively.

* P < 0.001 for regression coefficients, D vs. E.

† P < 10^-4 for linear slope constants, K_m vs. K_s.
FIGURE 6  Panel A: Comparison of external minor axis diameter (b) and intracavitary volume (V) during diastasis (filled circles) and ejection (unfilled circles) from data obtained during a transient vena caval occlusion in dog 5. Linear regression for D is \( b = 0.002V + 4.865 \) \((r = 0.993)\) and E is \( b = 0.016V + 5.144 \) \((r = 0.996)\). Panel B: Comparison of equatorial wall thickness (h) and V from data obtained during same vena caval occlusion as in A above. Linear regression for D is \( h = -0.004V + 1.355 \) \((r = -0.951)\) and E is \( h = -0.007V + 1.522 \) \((r = -0.987)\). D and E are the same as Figure 2.

Recently, Osakada et al. (1980) developed an ultrasonic triangulation technique which employed one subendocardial transmitting crystal with three overlying epicardial receiving crystals. This method theoretically measures wall thickness more accurately by accounting for potential shear of the ventricular wall (Streeter et al., 1969). In the control state, the average end-diastolic wall thickness by this method was 9.6 ± 2.6 mm and wall thickening during systole was 20.3 ± 10.5%. This compared favorably with 9.1 ± 1.0 mm and 30.0 ± 19.0% observed, respectively, from the current experiments. The degree of shear in their study seemed to be small, confirming the observations of Feigl and Fry (1964). In addition, the degree of systolic wall thickening obtained with the ultrasonic technique correlates well with previous observations (Mitchell et al., 1969; Bove, 1971), and any underestimation of wall thickening or non-uniformity of mural deformation must be minimal.

It has been proposed that the intact pericardium limits diastolic filling of the left ventricle to some extent (Misbach et al., 1979) and accentuates influences of the right ventricle on septal shifting and distortion of left ventricular geometry (Tyberg et al., 1978). In most of these studies, however, the pericardium had been surgically manipulated. The classic work of Kenner and Wood (1966) indicated that surgical closure of the pericardium may be the primary factor affecting ventricular dynamics in this setting. Because of the unsettled nature of this controversy, the pericardium was left widely open in the present study. Other factors, such as septal shifting and non-uniformity of minor axis geometry caused by high right ventricular diastolic pressures, could invalidate the prolate spheroidal model. The transient vena caval occlusions as employed in the present experiments routinely result in low right ventricular pressure throughout the time when left ventricular pressure and dimension data are obtained for analysis. Shortening and filling of the left ventricular minor axis circumference under these conditions seem to be relatively uniform and concentric in the plane of the minor axis circumference (Olsen et al., 1981). Thus, the external influences of the pericardium and right ventricle during the physiological conditions examined in the current study are felt to have affected the dynamic geometry of the left ventricle minimally (Mirskey and Rankin, 1979). Further investigation of the external determinants of left ventricular geometry should be performed.

Previous studies both in dogs and humans have shown significant differences in regional myocardial shortening throughout the ventricle. LeWinter et al. (1975) used segmental ultrasonic techniques and demonstrated a greater percentage of shortening from the end-diastolic dimension in the apical region of the ventricle as compared to the mid-ventricular or basilar regions. Kong et al. (1971) noted similar findings in humans, using coronary artery bifurcations as markers. There are, however, several problems with these data. The observed dimensional changes in these studies were normalized as a percentage shortening from the end-diastolic value. From an engineering standpoint, this type of normalization is not valid and may have introduced certain errors into the results. Furthermore, small variations in the three-dimensional spatial relationships of either the segmental crystals or the coronary markers could have caused considerable variability in the observed regional geometry. In a study of regional diameters measured with ultrasonic...
transducers, Ling et al. (1979) demonstrated no significant differences in shortening during ejection between the apical and midventricular regions when each dimension was properly normalized as a ratio of curvature to wall thickness relationship and \( V \), left ventricular internal volume. D and E are same as for Table 2.

The slopes (m), intercepts (b), correlation coefficients (r), and sum error of the estimates (SEE) of the regression analysis are given.

The relation between the \( r/h \) ratio and intracavitary volume (V) for diastasis (filled circles) and ejection (unfilled circles) in same vena caval occlusion as in Figure 7 for dog 5. Linear regression for D is \( r/h = 0.092V + 1.105 \) (r = 0.985) and E is \( r/h = 0.024V + 0.922 \) (r = 0.999). There was no significant difference between the linear slope constants (P > 0.07). D and E are same as Figure 2.

During early rapid ventricular filling, midventricular transmural pressure was substantially higher than apical pressure, whereas the apical pressure increased more rapidly and reached diastasis earlier once the diastolic minimum had been reached. Concurrently, the apical diameter increased more rapidly and reached diastasis earlier than the midventricular diameter. The average end-diastolic strains at each level, however, were not significantly different. These regional differences in diastolic geometry and pressure during rapid ventricular filling possibly were due to the velocity and acceleration of blood flow across the ventricle and
emphasize the need to measure pressures and dimensions at the same ventricular level when assessing dynamic geometry. Thus, it is clear that any geometric reference figure that assumes uniform mural deformation must be regarded, at best, as an approximation of the actual three-dimensional geometry. The question that should be answered about any given model is how close an approximation does it provide? Based on previous experiments (Rankin et al., 1976), we conclude that the prolate spheroidal model used in this study is a satisfactory approximation of left ventricular geometry in the normal canine heart under the experimental conditions of the present study, especially when diastolic data are considered.

There are several engineering methods available for normalization of left ventricular dimensions (Mirsky and Parmley, 1974). A natural strain normalization was selected in the present study because it does not assume uniform deformation or stress distribution along the ventricular wall. Since wall thickness stress is a compressive stress, positive strains in the wall thickness direction denoted wall thinning. Positive strains in the latitudinal and longitudinal directions represented lengthening because latitudinal and longitudinal stresses are tensile stresses. Only diastolic dimensions were normalized to 4 because the diastolic and ejection-phase geometries were clearly different, and no mechanically valid reference length could be defined for ejection. Despite the relatively low stresses in the wall thickness direction, wall thickness deformation was the greatest in most dogs. Whenever the functional characteristics of the ventricle were

![Figure 9](http://circres.ahajournals.org/)

**FIGURE 9** The diastatic stress-strain relationships typically observed for each of the three dimensions are shown for dog 1. The nonlinear elastic coefficients, α and β, are shown in Table 1.
altered, the percentage change observed in the wall thickness usually was the greatest. In this context, wall thickness, latitudinal, and longitudinal stresses and strains correspond, respectively, to radial, circumferential, and meridional stresses and strains (Mirsky, 1969).

In the initial phases of the present study, \( \varepsilon_p \) was derived as a fractional change of \( \zeta \) from \( \varepsilon_{\phi_0} \), calculating \( \zeta \) with an elliptical function (Eq. 5). Because strain in the longitudinal direction of a prolate spheroid varies with distance from the equator, this may not be a totally valid approach. Attempts were made to calculate \( \varepsilon_p \) from \( \varepsilon_{\phi} \) and \( \varepsilon_l \) data, using the formula for incompressibility of mass:

\[
\varepsilon_p = \frac{1}{(1 + \varepsilon_h)(1 - \varepsilon_l)^{-1}} \tag{12}
\]

where \( \varepsilon_h \) denotes wall thinning. The resulting data were not physiologically suitable, with highly variable strain values noted from dog to dog. This finding may reflect dimensional inconsistencies or problems with the applicability of this equation to intact heart muscle. Eventually, we reverted to the original technique of calculating \( \varepsilon_p \) as an average fractional change of the major axis circumference. These theoretical limitations with this method, but it yields results that are most consistent, physiologically. Hopefully, the error introduced with this method is small and the data are proportional to the actual equatorial major axis strain.

Theoretical limitations have been raised to the application of Laplace's relationship to model ventricular mechanics (Moriarty, 1980). When the left ventricle is modeled as a sphere, the law of Laplace markedly underestimates wall stresses and geometric deformations of the left ventricle for a given transmural pressure. The results of the present study depend integrally upon the validity of the ellipsoidal shell theory used to derive the three-dimensional stresses and deformations. This theory has been tested experimentally, and the latitudinal stress calculated from transmural pressure and ventricular dimensions correlated well with the directly measured latitudinal stress (McHale and Greenfield, 1973). In addition, the end-diastolic latitudinal stress calculated previously with this model in the conscious dog (Rankin et al., 1977) was similar to that directly measured by Burns et al. (1971). Longitudinal stress calculated in the present study was approximately 55% of the calculated latitudinal stress, a figure that is comparable to the 43% value directly measured in Burns' experiments. The calculation of wall thickness stress as 50% of transmural pressure may not be entirely valid. There is some evidence that the wall thickness stress distribution across the wall is non-linear, and its calculation as 50% of transmural pressure would result in a certain amount of error. However, even with thick-walled stress theories (Mirsky, 1969), the degree of non-linearity is small, and the error introduced by the above method would be less than 10%. This amount of error would not have affected the results of the present study significantly.

Two previous studies (Suga and Sagawa, 1974; Rankin et al., 1980) have demonstrated essentially linear relationships between minor axis diameter of the left ventricle and measured internal volume over the physiological range of volumes. In both of these experiments, differences in the diameter-volume relationships were observed from diastole to systole, and probably were related to changes in myocardial geometry between these two phases. Furthermore, equatorial wall thickness and internal ventricular volume were found to be related in a similar linear fashion, although the relationship was inverse (Fig. 7B). The totally independent nature of the measurements of Suga and Sagawa (1974), and unpublished observations made in our laboratory with an intracavitary balloon technique (Rankin et al., 1976) reinforce the findings illustrated in Figure 6.

Because ventricular volumes were calculated from three-dimensional geometry data, problems of mathematical coupling of data arise. An introduced measurement error of 5% in the minor or major axis diameters would produce a corresponding 5–12% error in calculated chamber volume with an error of less than 5% in the derived chamber eccentricity, whereas an introduced error of 10% in wall thickness would produce a similar error in chamber volume and eccentricity of 12% and 1%. The resulting eccentricity-volume relationships would be shifted either up or down, depending on the dimension changed, but the slopes would remain relatively constant throughout the diastasis and ejection phases, indicating that this magnitude of introduced error would have minimal effect on the basic result of the study. The magnitude of these errors introduced in each direction of measurement would be well beyond the minimum resolution of measurement of the pulse-transit ultrasonic technique, which is approximately 0.08 mm.

Most currently accepted stress theories are modifications of the Laplace relationship:

\[
\sigma \propto P \times r/h \tag{12}
\]

where \( \sigma \) is the tensile stress, \( P \) is the transmural pressure, \( r \) is the radius of curvature, and \( h \) is the wall thickness. In the current study, the latitudinal radius of curvature was observed to be related to wall thickness in an inverse manner (Table 3). Similar findings have been published by Sasayma et al. (1976). The relationship for diastasis was not the same as for ejection, again pointing out differences in diastatic and ejection phase geometry. Since radius and wall thickness were related inversely (Table 3), the \( r/h \) ratio increased in a linear fashion with increasing diastolic left ventricular volume (Fig. 7). The \( r/h \) ratio vs. volume relationship followed a similar pattern during the ejection phase.
This constant geometric relationship determines wall stress at any given transmural pressure (Fig. 8) and constitutes the major influence of ventricular geometry on cardiac function. The differences in the diastatic and ejection phase \( y/h \) vs. volume relationships (Table 3) probably account for the discrepancy in the diastatic and ejection phase pressure-stress regression constants, \( K_{pr} \) and \( K_{pr} \), (Table 4). In summary, the relationship between intracavitary pressure and mural stress in any given ventricle is linear during diastasis and ejection and is determined by the three-dimensional geometric interrelationships of the ventricular wall.

During the initial phase of this study, it became evident that the dynamic geometry of the left ventricle was more complex than had been appreciated previously. Not only were there significant rearrangements in ventricular geometry during the isovolumic phases of the cardiac cycle, but the direction and magnitude of these rearrangements varied with ventricular volume. This finding led to the observation (Fig. 2A) that the eccentricity of the left ventricle during diastasis and during ejection was related linearly to intracavitary volume. Although this method of defining ventricular geometry is undoubtedly a simplification of more complex geometric rearrangements, it provides a means of representing the dynamic geometry of the left ventricle over the entire physiological range of ventricular volumes. The geometric rearrangements observed during the isovolumic phases of contraction and relaxation seemed to represent transitional states between the diastatic and ejection phase curves. It must be emphasized that Figure 2A illustrates diastatic data. When dynamic e-\( v \) data from single cardiac cycles were examined (Fig. 2B), the rapid filling phases did not fall exactly on the diastatic curve. This observation may be due to phase differences in the rate of deformation in the latitudinal and longitudinal dimensions during early rapid filling and atrial systole and again reflects differences in regional ventricular geometry during dynamic diastolic filling.

Even though there was moderate variability in the geometric characteristics observed from dog to dog (Table 1), the eccentricity-volume relationship in any given ventricle seemed to be constant. This relationship did not change with isotropic interventions or with alterations in afterload (Fig. 3). Changes in myocardial function indirectly altered the dynamic geometry by changing the end-diastolic position on the curve or the degree of ejection-phase shortening, but did not alter the basic linear relationships. Therefore, we hypothesize that the findings in Figure 2 are independent of intrinsic myocardial performance.

The relationships between ventricular volume and shape probably reflect structural properties of the left ventricular wall. It is unclear, however, why the diastatic and ejection-phase curves shown in Figure 2 should be different. During diastatic filling, the dimensional changes of the left ventricle were asymmetrical, with a disproportionate increase occurring in the minor axis diameter as compared to the major axis diameter. As a result, the ventricle became considerably more spherical as diastolic filling progressed. These diastolic shape changes probably are determined by the three-dimensional stress-strain properties of the diastolic myocardium and have been reviewed elsewhere (Rankin and Olsen, 1980). As illustrated in Figure 9, the left ventricular wall seemed to be anisotropic, and the longitudinal dimension was significantly stiffer than the latitudinal dimension. Thus, with a given change in diastolic transmural pressure, deformation in the minor axis diameter was greater.

The dimensional changes during ejection were more symmetrical, and very little alteration in shape occurred as ventricular volume decreased. Perhaps the ejection-phase relationship was different from the diastatic relationship because it was determined primarily by intramural fiber orientation rather than the more anisotropic stress-strain properties. The distribution of fiber angles has been shown to be fairly uniform across the ventricular wall (Streeter et al., 1969; Streeter and Hanna, 1973), with longitudinally oriented fibers predominating in the subendocardial and subepicardial layers and latitudinal oriented fibers predominating at the midwall level. Such an even distribution could account for the more concentric volume changes that were observed during ejection.

The method of analysis presented in this communication represents a convenient way to quantify dynamic left ventricular geometry. The data presented characterize the dynamic geometry of the normal canine left ventricle. These findings may be useful in improving the assessment of myocardial function in the intact heart and serve as a basis for further study of dynamic geometry of the left ventricle under the abnormal conditions of myocardial ischemia, chamber dilation, or hypertrophy.

Acknowledgments

We gratefully acknowledge the valuable assistance of Ruth Dixon in typing the manuscript and Robert L. Margulies in preparing the figures and illustrations.

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doi: 10.1161/01.RES.49.4.843

_Circulation Research_ is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
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Print ISSN: 0009-7330. Online ISSN: 1524-4571

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