Left Ventricular Wall Stress Calculated from One-Plane Cineangiography

AN APPROACH TO FORCE VELOCITY ANALYSIS IN MAN

By Herman L. Falsetti, Robert E. Mates, Colin Grant, David G. Greene, and Ivan L. Bunnell

ABSTRACT

Left ventricular dimensions from routine clinical one-plane cineangiograms were combined with left ventricular pressure measurements to permit calculation of left ventricular wall stresses. The 25 patients included 12 with normal left ventricular dynamics, 6 with volume overload, 3 with outflow obstruction, and 4 with cardiomyopathy. Average stresses calculated on the basis of an ellipsoid model agreed with average values obtained from the exact solution of a thick-walled elastic ellipsoidal shell. Peak values were 150 to 625 g/cm² in the circular direction and 75 to 365 g/cm² in the longitudinal direction. A fiber-corrected stress was defined which represents a force per muscle fiber. The variation in fiber-corrected stress during the cardiac cycle may be considerably different from the variation in simple stress.

The force-velocity characteristics of circular fibers for the 25 patients are presented. The data on peak wall stress overlap in the four groups of patients. Peak velocity of circumferential fiber shortening varied from 0.44 to 0.63 lengths/sec in patients with myocardial weakness and varied from 0.74 to 2.56 lengths/sec in the other patients. Contractile element velocity was determined during ventricular ejection when the rate of force change equaled zero. Contractile element velocity of shortening was 0.22 to 0.32 lengths/sec in the cardiomyopathy group and 0.50 to 1.32 lengths/sec in the other patients.

ADDITIONAL KEY WORDS

contractile element velocity of shortening left ventricular wall thickness cardiac muscle mechanics

The force developed by a contracting muscle is a function of the velocity of contraction. The force-velocity relation was first determined for skeletal muscle (1, 2) and more recently has also been determined in isolated cardiac muscle (3-5).

The mechanical behavior of the intact heart is a complicated function of its geometry as well as of the contractile behavior of individual muscle fibers. The performance of the heart may be determined by cineangiography to determine the geometry of the heart during contraction and simultaneous measurements of ventricular pressure (6-8). The velocity of contraction of the muscle fibers may be estimated from the cineangiograms. Velocity of contraction of ventricular segments has also been measured directly using silver-tantalum markers (9).

Determination of the force-velocity characteristics of the cardiac muscle from these gross measurements requires a model of the mechanical behavior of the intact heart. Early models were based on the Laplace's law (10), which yields the tension developed in a thin-walled shell. The simplest such model is the thin-walled sphere used by Levine and Britman (11), but the actual ventricular geometry may be better approximated as an ellipsoid of revolution (prolate spheroid). Thin-walled ellipsoid models were employed...
by Fry et al. (12) and by Gault et al. (7). All of these models neglect the finite thickness of the wall and permit one to calculate only the tension, or force developed per unit length of wall.

Sandier and Dodge (6) calculated average tensile stresses for an ellipsoidal model with finite wall thickness, but, as discussed below, their formulation predicts stresses higher than the actual average values. Recently, Wong and Rautaharju (13) calculated the stress distribution in a thick-walled ellipsoid of revolution, assuming linear elastic behavior. Their solution neglected shear and bending moments in the wall and followed the earlier solution of Lamé (14) for thick-walled spheres and cylinders. Mirsky (15) has extended the Wong and Rautaharju solution to include the effects of shear and bending moments. His results indicate that for typical ventricular geometry, these effects are negligible except near the apex of the ventricle. Hood et al. (16) have compared Sandier and Dodge’s results with average stresses obtained from Wong and Rautaharju and confirm the overestimation of average stress (approximately 10%) by the Sandier and Dodge formula.

The ventricular wall is a nonhomogeneous, anisotropic material composed of layers of fibers running largely parallel to the wall and separated by connective tissue. The elastic elements of the fiber are nonlinear (5). The force developed during contraction of the muscle is a function of velocity as well as length, and there exists no shell theory adequate to express this behavior. Detailed stress distributions, such as those obtained by Wong and Rautaharju (13) and Mirsky (15) assume homogeneous, isotropic, linear elastic behavior for the muscle fiber, but the actual stress distribution in the myocardial wall may be quite different.

No experiment has yet been designed to measure local stresses, hence it is not possible to verify a detailed stress distribution. It is, however, possible to measure approximately the total force per unit length of muscle wall exerted by the myocardium, that is, the resultant force generated by a distribution of stresses through the wall. This force cannot be related in any fundamental way to the contraction of an identifiable muscle element. The experiments of Hefner et al. (17) measured such tensile forces in one direction only, roughly the circumferential.

The present study utilizes a thick-walled ellipsoidal model. Because of the difficulties mentioned above in evaluating detailed stress distributions, only average stresses have been calculated. The model used is similar to that employed by Sandier and Dodge (6), modified to account correctly for the finite wall thickness. The calculated stresses agree with those obtained by averaging the result obtained by Wong and Rautaharju (13).

The myocardium is composed of large numbers of muscle fibers which contribute to the total force generated by the wall. The quantity of physiological significance is force per muscle fiber, but the stresses calculated above represent forces per unit area. Since the cross-sectional area of the wall changes during the cardiac cycle while the number of muscle fibers remains constant, the time history of fiber force may be different from that of the stress. In particular, peak stress may occur at a different time than peak fiber force.

Since the number of muscle fibers is unknown, the fiber force cannot be determined directly. A quantity proportional to fiber force can be obtained by multiplying the calculated stress by the ratio of the cross-sectional area to some reference area, here taken at end-diastole. This quantity, designated the fiber-corrected stress, is defined for the equatorial direction by

\[ (\sigma_{fe}) = \left(\frac{\sigma_{ee}}{A_{ea}}\right) \]

stress distribution in the myocardial wall may be quite different.

\[ A_{ea} \]

The conventional double subscript is used for specifying the stresses. The first subscript identifies the face of the element on which the stress acts, the second the direction of the stress. Thus \( \sigma_{ee} \) is the stress on the face perpendicular to the equatorial direction, acting in the equatorial direction.
The reference area chosen affects only the magnitude of the fiber corrected stress and not the time variation. The end-diastolic area was chosen because it can be accurately measured angiographically.

Comparison of stresses calculated for different patients must be interpreted cautiously since the number of muscle fibers per unit cross-sectional area probably varies from case to case even when there is a healthy myocardium. Thus two hearts composed of fibers with identical force-velocity characteristics may exhibit different stresses (force per unit area). The number of fibers per unit area is probably more nearly equal at end-diastole than at any other time during the cardiac cycle. This is true because the wall thickness during systole is influenced not only by anatomical structure but by the muscle contraction. Since the muscle volume remains approximately constant, a healthy muscle will experience a greater increase in wall thickness as it shortens than will a muscle which contracts weakly. Comparison of fiber-corrected stress between two patients is thus probably more meaningful than comparison of ordinary stress.

Stresses can be calculated from these formulas in man by one-plane cineangiography (18). This technique allows the measurement 60 times per second of length and breadth of the left ventricular cavity and thickness of the wall.

Calculation of the stresses during the cardiac cycle together with the time history of ventricular dimensions provided by the cineangiograms provides the necessary data for a force-velocity analysis of the cardiac muscle.

The important velocity physiologically is the velocity of shortening of the contractile element (19, 20). There is no simple relationship between the velocity of shortening of the muscle fiber and that of the contractile element (21, 22), since in general the series elastic component will also be changing in length. At the point of peak fiber force, however, the rate of change of force with time is zero, and hence the series elastic velocity is also zero, so the contractile element velocity can be inferred directly from the measurements. The peak fiber force does not necessarily occur at the same time as the maximum stress, as mentioned above. If the series elasticity of the human cardiac muscle were known, series elastic velocity, and hence contractile element velocity, could be estimated for other points in the cardiac cycle.

The data available on left ventricular dimensions can also be used to measure velocity of shortening of the circumferential muscle fiber. As discussed in the next section, myocardial force-velocity analysis is now feasible from data obtained during a routine clinical procedure (left ventricular catheterization and one-plane cineangiography). This technique further allows comparison of the contractile behavior of the left ventricle in patients with different types of heart disease.

Materials and Methods

Twenty-five patients, 10 male and 15 female, aged 17 to 58, were studied. The patients (see Table 1) included 12 with hemodynamically normal left ventricles, 7 of whom had mitral stenosis, 2 an atrial septal defect and 3 functional heart murmurs. Six patients had a volume overload with regurgitation at the mitral or aortic valve or both. Three patients had outflow obstruction (pressure overload), two from aortic stenosis and one from coarctation of the aorta. Four patients had cardiomyopathy (left ventricular muscle weakness), with high end-diastolic volumes in the left ventricle and low ejection fractions. Cardiac catheterization and angiography were carried out under mild sedation (sodium pentobarbital, meperidine and promethazine hydrochloride) and local anesthesia; the patients were in the fasting state. Cineangiograms in the right anterior oblique projection were made at 60 frames/sec with a Picker 9-inch image intensifier as 25 to 50 ml of 76% meglumine diatrizoate (Renografin) was injected into the left atrium through a transseptal catheter. Left ventricular pressure was recorded simultaneously with the cineangiographic frame signal by a catheter in the left ventricle (except in cases 20 and 21, in which sequential pressures were fitted in relation to the volumes). All pressures were referred to zero level 10 cm above the table top. The pressures were recorded by fluid-filled catheter systems which had a frequency response of 15 to 20 cps. Complete dynamic characteristics of the catheter system were not available; the phase lag was partly compensated by shifting the
Cineangiograms of left ventricular cavity in right anterior oblique projection showing long axis (L), short axis (M), and wall thickness (W).

Pressure one cine frame forward (17 msec). Left ventricular volume was determined by a one-plane cineangiographic method previously described (18). In this method, L, the long axis, and M, the short axis at right angles to the mid-point of L, are both measured directly in the right anterior oblique projection (Fig. 1). The assumption is made that the short axis, which is not visible, N, perpendicular to both L and M, is equal to M. Magnification and distortion of each axis are corrected by a factor, f (18). The formula for volume, V, is then 

\[ V = KLM^2 \]  

where \( K = \frac{\pi}{6f^2} \). One-plane cineangiographic measurements are known to overestimate left ventricular volumes; preliminary studies in this laboratory indicate a consistent overestimation of about 15%. Such an error could affect the magnitude of reported results but not the comparison between patients.

Stresses were calculated at the equator of the ventricle using the model described in the appendix. This model assumes an ellipsoidal geometry with finite wall thickness. Shear forces and bending moments are neglected. The model is similar to that used by Sandler and Dodge (6) but has been corrected to account properly for wall thickness. The average stress in the equatorial direction is given by

\[ \sigma_{eq} = \frac{PM(2L^2 - M^2)}{4W(L^2 + MW)} \]  

(A9)

and the longitudinal stress by

\[ \sigma_{\|} = \frac{PM^2}{4W(M + W)} \]  

(A10)

Peak velocity of shortening was calculated for a circular muscle fiber at the equator, lying in the middle of the wall halfway between lumen and external surface. This was done by plotting circular fiber length (\( \pi \times \text{diameter} \), or \( \pi [M + W] \)) against time as the fiber shortened during ejection. The steepest tangent in this curve gave peak velocity of shortening of the midwall circumferential muscle fiber in centimeters per second. The important value physiologically is the velocity of shortening of the contractile element (19, 20). As previously discussed, contractile element velocity of shortening can be calculated at the point of peak fiber force.

Velocity was normalized by dividing by the initial length, giving velocity of shortening in lengths per second. The objective in normalizing the velocity data was twofold: (1) to enable comparison of data between patients with different sized hearts; (2) to provide some way of relating the performance of the intact heart to the behavior of individual muscle fibers. These criteria governed our choice of normalization. The precise scaling of force-velocity data requires knowledge of two factors: first, how the force-velocity characteristics of an individual sarcomere are affected by its physical size (length, cross-sectional area); secondly, the relative size of sarcomeres in different intact hearts. Because this information is not available, we made the plausible, though probably not exact, assumption that sarcomeres were roughly the same size in each of the hearts in the present study. This enabled us to choose appropriate scaling for both force and velocity.

If a number of identical sarcomeres in parallel were stimulated, the developed force would be proportional to the number of sarcomeres, and the velocity would be that of a single sarcomere. Therefore, force per muscle fiber would be the appropriate variable for comparison. (We used a fiber-corrected stress for this purpose.) If the same number of identical sarcomeres were placed in series and stimulated, the developed force would be that for the individual sarcomere, and the velocity would be proportional to the number of fibers. Thus the appropriate variable is velocity developed per sarcomere, or, with our assumption of identical sarcomeres, velocity per unit length. Some support for this normalization can be drawn from isolated muscle experiments of Parmley and Sonnenblick (5). Their measurements of series elasticity were normalized in terms of initial length and showed reasonable agreement among muscle fibers of different lengths.

It might be thought more appropriate to normalize using the instantaneous length as did...
Gault et al. (7). This would increase the calculated velocities for all groups with the smallest increase occurring in the myopathy group and would thus provide greater separation of the myopathy data. However, the velocity resulting from such a normalization, \((1/\text{length}) \frac{d(\text{length})}{d(\text{time})}\), is a logarithmic derivative of length and not a true velocity. Thus, the shape of the force-velocity curve will differ from the unnormalized curve.

The instantaneous fiber-corrected stress \((\sigma_{00})_F\) may be calculated by multiplying the calculated equatorial stress by the ratio of the instantaneous cross-sectional area of the wall to the end-diastolic area. The appropriate wall cross-sectional area is that normal to the stress being considered. For the longitudinal stress \(\sigma_{00}\), this is the equatorial area shown in Figure 2. For the equatorial stress \(\sigma_{00}\) an area parallel to the long axis \(L\) is appropriate. However, the local area change at the equator is not readily available from the cineangiograms. We have assumed that the equatorial area

\[
\pi (r_2^2 - r_1^2) = \pi W (2r_1 + W) = \pi W (M + W)
\]

can be used for the equatorial stress as well. This is equivalent to assuming that the number of muscle fibers per unit area remains the same in both directions. The equatorial fiber-corrected stress is then given by

\[
(\sigma_{00})_F = \sigma_{00} \frac{A}{A_D} = \sigma_{00} \frac{W/(M + W)}{W_D/(M_D + W_D)},
\]

where \(W_D, M_D\) and \(A_D\) are the wall thickness, minor axis length and wall area at end-diastole. Substitution for \(\sigma_{00}\) from equation A9 gives

\[
(\sigma_{00})_F = \frac{PM}{W_D (L^2 - M^2)} (M + W) (M_D + W_D).
\]

Typical time histories of equatorial stress \(\sigma_{00}\), fiber-corrected stress \((\sigma_{00})_F\) and ventricular pressure \(p\) are shown in Figure 3. Peak fiber-corrected stress usually occurs between peak stress and peak pressure. Although all the equations given above are simple enough to be calculated on a desk calculator, we have processed these data on a Control Data Corporation 6400 Computer and a California Computer Products X-Y plotter.

In 13 additional patients, wall thickness \(W\) was compared in two planes by measuring the lateral ventricular wall in the right anterior oblique (RAO) and frontal cineangiograms. The lateral ventricular wall was planimetered over a 3-cm segment and compared with a single wall measurement in end-diastole. The planimetered frontal wall measurement correlates well with ventricular mass measured at autopsy (23, 24). Because of the possible error that might be introduced by right ventricular hypertrophy in the measurement of left ventricular wall thickness, no case with evidence of advanced right ventricular hypertrophy (right ventricular pressure greater than 40 mm Hg, ECG or physical findings of right ventricular hypertrophy) was included among patients reported in this paper.

### Results

Figure 4 compares the thickness of the left ventricular wall at end-diastole in right anterior oblique and frontal planes in 13 patients. The average wall thickness in the frontal plane determined by planimetry over a 3-cm segment is plotted against a single measurement of wall thickness at the short axis in the right anterior oblique projection. The largest deviation was 15%. From these data it appears that a single measurement of wall thickness at the short axis in the right anterior oblique projection gives just as precise a measurement of angiographic wall thickness as that determined by planimetry in the frontal plane.
FIGURE 3
Time course of fiber-corrected stress, equatorial stress and cavity pressure in cases 3 (left) and 12 (right). Peak stresses precede peak cavity pressure. Note that peak fiber-corrected stress need not be simultaneous with peak equatorial stress.

FIGURE 4
Comparison of left ventricular wall thickness at end-diastole in right anterior oblique and frontal angiogram.

Table 1 includes the diagnosis, the long axis (L), short axis (M), and wall thickness (W) at end-diastole, ejection fraction (EF), and peak wall stress in two directions in 25 patients. The wall thickness was measured through the complete cycle (60 times/sec) in all 25 patients. Figure 5 illustrates the time course of wall thickness in two subjects over a complete cardiac cycle. Circular and longitudinal wall stress were then calculated at 17-msec intervals for all 25 patients. Peak wall stress always occurred before peak pressure and was 40 to 100% greater in the equatorial direction (Table 1). Peak equatorial stress $\sigma_{eq}$ ranged from 150 to 625 g/cm$^2$ as compared with a range of 75 to 365 g/cm$^2$ in the longitudinal stress $\sigma_{o0}$ at the same point.

In Table 2, measurements of pressure, long axis length, short axis length, and wall thickness necessary for the calculation of equatorial stress and velocity of fiber shortening are presented in one patient (case 8). The time reference is the angiographic frame which occurs every 17 msec. These measurements were taken from successive points in a cardiac cycle; end-diastole, peak equatorial stress, maximum fiber-corrected stress, peak...
successive lengths throughout the cardiac cycle. Also included in the table are equatorial stresses calculated by the method of Gault et al. (7) (equation A12) and the method of

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TABLE 2
Representative Data from One Cardiac Cycle of a Normal Left Ventricle in a Patient with Mitral Stenosis (Case 8)

<table>
<thead>
<tr>
<th>Frame number</th>
<th>End-diastole</th>
<th>e&lt;sub&gt;98&lt;/sub&gt; Peak</th>
<th>(e&lt;sub&gt;98&lt;/sub&gt;)&lt;sub&gt;f&lt;/sub&gt; Peak</th>
<th>Peak systolic pressure</th>
<th>End-systole</th>
</tr>
</thead>
<tbody>
<tr>
<td>P (mm Hg)</td>
<td>14</td>
<td>106</td>
<td>136</td>
<td>147</td>
<td>143</td>
</tr>
<tr>
<td>L (cm)</td>
<td>10.2</td>
<td>9.3</td>
<td>8.8</td>
<td>8.7</td>
<td>8.7</td>
</tr>
<tr>
<td>M (cm)</td>
<td>6.4</td>
<td>5.6</td>
<td>4.9</td>
<td>4.6</td>
<td>4.5</td>
</tr>
<tr>
<td>W (cm)</td>
<td>0.73</td>
<td>0.93</td>
<td>1.06</td>
<td>1.14</td>
<td>1.14</td>
</tr>
<tr>
<td>σ&lt;sub&gt;98&lt;/sub&gt; (g/cm²)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Present study (Eq. A10)</td>
<td>64</td>
<td>330</td>
<td>338</td>
<td>320</td>
<td>309</td>
</tr>
<tr>
<td>Sandler (6) (Eq. A11)</td>
<td>68</td>
<td>370</td>
<td>372</td>
<td>354</td>
<td>341</td>
</tr>
<tr>
<td>Gault (7) (Eq. A12)</td>
<td>66</td>
<td>350</td>
<td>361</td>
<td>343</td>
<td>331</td>
</tr>
<tr>
<td>Wong (13) (Eq. A13)</td>
<td>62</td>
<td>333</td>
<td>336</td>
<td>320</td>
<td>310</td>
</tr>
<tr>
<td>Wong (13) (Eq. A14)</td>
<td>62</td>
<td>335</td>
<td>335</td>
<td>319</td>
<td>309</td>
</tr>
<tr>
<td>(σ&lt;sub&gt;98&lt;/sub&gt;)&lt;sub&gt;f&lt;/sub&gt; (g/cm²)</td>
<td>64</td>
<td>388</td>
<td>414</td>
<td>400</td>
<td>386</td>
</tr>
<tr>
<td>V&lt;sub&gt;CF&lt;/sub&gt; (length/sec)</td>
<td>0.0</td>
<td>0.77</td>
<td>0.87</td>
<td>0.61</td>
<td>0.0</td>
</tr>
</tbody>
</table>

Equatorial stress (σ<sub>98</sub>) has been calculated by the method presented in this paper as well as several other methods.

(σ<sub>98</sub>)<sub>f</sub> = Fiber-corrected stress; V<sub>CF</sub> = velocity of fiber shortening; other abbreviations as in Table 1.

Sandler and Dodge (6) (equation A11). These formulas give values which vary similarly in time, with somewhat higher stress values than the formula presented in this paper. Equation A11, which attempts to account for the finite wall thickness of the ventricle, yields slightly higher equatorial stress values than the thin-wall result (equation A12). Thus it actually corrects for wall thickness in the wrong direction.

Average equatorial stresses calculated from Wong and Rautaharju's (13) results, both from equation A13 and equation A14, are also presented in Table 2. Stresses calculated throughout the cardiac cycle from both equations agree very well with the present results (equation A10). The maximum difference between the stress values was 3% for the patients in this study.

No simple relation between end-diastolic volume and peak equatorial stress or peak fiber-corrected stress was evident in the 25 patients in this study. In the larger cavities, peak stress was not higher. These results indicate that the tendency toward an increase in wall stress due to increased volume is compensated by muscular hypertrophy (25).

In Figure 6, contractile element velocity of shortening is plotted against the peak fiber-corrected stress from the data presented in Table 3. Patients have been classified according to normal left ventricle, volume overload (regurgitation), pressure overload (obstruction), and cardiomyopathy. The important conclusion from this figure is that wall stress is not clearly different in the four groups. This
Velocity of Contractile Element ($V_{CE}$) at the Time of Peak Fiber-Corrected Stress ($\sigma_{CP}$) with Simultaneous Equatorial Wall Stress ($\sigma_{EE}$)

<table>
<thead>
<tr>
<th>Case</th>
<th>Length/sec</th>
<th>($\sigma_{CP}$)</th>
<th>($\sigma_{EE}$)</th>
<th>$V_{CF}$</th>
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</thead>
<tbody>
<tr>
<td>1</td>
<td>0.53</td>
<td>380</td>
<td>260</td>
<td>1.93</td>
</tr>
<tr>
<td>2</td>
<td>1.06</td>
<td>440</td>
<td>365</td>
<td>1.35</td>
</tr>
<tr>
<td>3</td>
<td>0.83</td>
<td>710</td>
<td>625</td>
<td>1.20</td>
</tr>
<tr>
<td>4</td>
<td>0.75</td>
<td>370</td>
<td>380</td>
<td>1.16</td>
</tr>
<tr>
<td>5</td>
<td>0.75</td>
<td>310</td>
<td>290</td>
<td>1.21</td>
</tr>
<tr>
<td>6</td>
<td>0.87</td>
<td>385</td>
<td>335</td>
<td>1.15</td>
</tr>
<tr>
<td>7</td>
<td>0.59</td>
<td>380</td>
<td>340</td>
<td>0.74</td>
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<td>8</td>
<td>0.71</td>
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<td>340</td>
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</tr>
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<td>9</td>
<td>0.90</td>
<td>235</td>
<td>245</td>
<td>2.56</td>
</tr>
<tr>
<td>10</td>
<td>1.04</td>
<td>260</td>
<td>230</td>
<td>1.38</td>
</tr>
<tr>
<td>11</td>
<td>0.83</td>
<td>290</td>
<td>310</td>
<td>1.20</td>
</tr>
<tr>
<td>12</td>
<td>0.71</td>
<td>175</td>
<td>140</td>
<td>1.58</td>
</tr>
<tr>
<td>13</td>
<td>1.08</td>
<td>365</td>
<td>355</td>
<td>1.38</td>
</tr>
<tr>
<td>14</td>
<td>0.50</td>
<td>340</td>
<td>265</td>
<td>1.15</td>
</tr>
<tr>
<td>15</td>
<td>1.32</td>
<td>530</td>
<td>555</td>
<td>2.34</td>
</tr>
<tr>
<td>16</td>
<td>0.81</td>
<td>530</td>
<td>435</td>
<td>1.22</td>
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<td>17</td>
<td>1.16</td>
<td>545</td>
<td>285</td>
<td>2.41</td>
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<tr>
<td>18</td>
<td>0.98</td>
<td>455</td>
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<td>1.28</td>
</tr>
<tr>
<td>19</td>
<td>0.50</td>
<td>435</td>
<td>325</td>
<td>1.36</td>
</tr>
<tr>
<td>20</td>
<td>0.79</td>
<td>385</td>
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<td>1.22</td>
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<tr>
<td>21</td>
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<td>435</td>
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<td>1.36</td>
</tr>
<tr>
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<td>280</td>
<td>375</td>
<td>0.50</td>
</tr>
<tr>
<td>23</td>
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<td>335</td>
<td>285</td>
<td>0.51</td>
</tr>
<tr>
<td>24</td>
<td>0.36</td>
<td>220</td>
<td>180</td>
<td>0.44</td>
</tr>
<tr>
<td>25</td>
<td>0.32</td>
<td>545</td>
<td>385</td>
<td>0.63</td>
</tr>
</tbody>
</table>

Peak circumferential fiber velocity of shortening ($V_{CF}$) is taken at different times in the cardiac cycle than the stress data.

has also been noted by other investigators (8). Of interest, however, is that contractile element velocity of shortening is very different in the group with myocardial disease (0.22 to 0.32 lengths/sec in the cardiomyopathy group; 0.50 to 1.32 lengths/sec in the three groups with apparently healthy myocardium). A similar separation of the myopathy group was apparent using velocity of circumferential fibers. Peak velocity of circumferential fiber shortening varied from 0.44 to 0.63 lengths/sec in patients with myopathy and 0.74 to 2.56 lengths/sec in the other three groups of patients.

As velocity of shortening falls with increasing myocardial disease, the ventricular ejection fraction will fall. Figure 7 demonstrates this relation between contractile element velocity of shortening and ejection fraction. Note that the four patients with myopathy (decreased ejection fraction, decreased velocity of shortening) are clearly separated from the other three patient groups. Also there appears to be a direct relation between velocity of shortening and ejection fraction.

Similar results may be obtained by plotting circumferential fiber velocity of shortening versus ejection fraction, as shown in Figure 8. These may be obtained from angiographic techniques alone and do not require simul-
Discussion

Stresses have been calculated in two directions at the equator of the ventricle. The larger of these two, the equatorial stress, has been taken as representative of the stresses in the ventricular wall. In view of the assumptions in the model, stress calculations at other locations do not appear to be worthwhile. While these results provide information only about average stresses, they are probably more useful than detailed stress distributions based on unrealistic assumptions about muscle properties. The stresses obtained agree with average values from more detailed models. The average stresses are also directly related to tensile forces which can be measured in an open chest preparation (17).

The determination of wall thickness in the right anterior oblique projection is a critical element of the one-plane cine method of measuring stress. We have tested this assumption by measuring the thickness of the left ventricle in right anterior oblique and frontal projection in 13 patients. This is evidence that a single measurement in the one-plane right anterior oblique angiogram can be used to measure angiographic wall thickness in the absence of right ventricular hypertrophy.

As shown in Figure 5, wall thickness increases during systole as the ventricular cavity becomes smaller. This change is expected, since contracting muscle alters very little in volume (26). As systole progresses, the shape of the ventricular cavity changes as well, from a round, relatively smooth circle in cross-section to an infolded, complex cross section. In late systole the wall thickness measured on angiograms may be in error. Peak stress values occur in early systole, before this possible error becomes important. Improvements in the quality of wall thickness measurement will directly improve the calculation of wall stress, and this complex topic is the subject of a separate study (27).

The significant quantity physiologically is the force per muscle fiber, not the force per unit area. A fiber-corrected stress has been defined which is proportional to this force per muscle fiber. The fiber-corrected stress is defined as the instantaneous total force on the equator of the ventricle divided by the end-diastolic cross-sectional area. This stress is useful in calculating contractile element velocity, which can be estimated directly from the measurements at the point of maximum force per muscle fiber.

The observations in this study suggest that, in spite of increased cavity pressure or volume load, neither the peak force per unit area nor the peak fiber-corrected stress vary greatly. One may speculate that the heart hypertrophies as a result of a chronic pressure or volume load in such a way as to maintain wall stress within certain limits. Peak stress measurements then may hold the key to hypertrophy but contribute little diagnostically in a given patient.

Velocity measurements, on the other hand, are clearly different in patients with myocardial weakness from those in other groups of patients and correlate well with ejection fraction. Both velocity of contraction and ejection fraction may be estimated by inspection of a cineangiogram of the left ventricle and have been used clinically in many laboratories. The quantitative evaluation of these parameters permits more precision and comparison from one laboratory to another. While the contractile element velocity has more theoretical interest to muscle physiologists, the simpler measurement of velocity of fiber shortening is probably as useful clinically.

Appendix

Calculation of the total force (tension) and stress on the equator of a thick ellipsoid of revolution

In analogy with Laplace's law, the total force per unit length exerted in any direction within a thick, curved wall can be calculated. An element cut from such a wall is shown in Figure 9. Note that the element is infinitesimal in extent in two directions but has finite thickness in the radial direction. Shear forces and bending moments are ne-
Shell element cut from a thick-walled shell as shown in the upper left. The element has the same thickness \( W \) as the wall. The pressure force \( \rho d^3 R d\phi \) acts on the inner radial face. Forces \( dF_{o\theta} \) and \( dF_{e\phi} \) act on the other faces.

Neglected. A force balance in the radial direction on this element yields

\[
pr_{1}d\theta R_{1}d\phi = 2dF_{o\theta} \sin \frac{d\theta}{2} + 2dF_{e\phi} \sin \frac{d\phi}{2}
\]

(A1)

where \( r_{1} \) and \( R_{1} \) are the radii of curvature. The forces \( dF_{o\theta} \) and \( dF_{e\phi} \) are the total forces exerted on the side of the element. In analogy with the formulation of Laplace’s law, we define the tension \( T_{o\theta} \) and \( T_{e\phi} \) as the force per unit length of wall:

\[
T_{o\theta} = \frac{dF_{o\theta}}{R_{1}d\phi}, \quad T_{e\phi} = \frac{dF_{e\phi}}{r_{1}d\theta}.
\]

Since the length of wall is different at the inner and outer surfaces, the inner wall is chosen arbitrarily in this definition. While the forces \( dF_{o\theta} \) and \( dF_{e\phi} \) are infinitesimal, the corresponding tensions are finite quantities. Substituting these definitions into Equation A1 yields the analog of Laplace’s law for a thick-walled shell:

\[
\frac{P}{r_{1}} = \frac{T_{o\theta}}{R_{1}} + \frac{T_{e\phi}}{r_{1}}.
\]

(A2)

Laplace’s law may also be written in terms of average force per unit area, or stress, using the identity

\[
total \ force = stress \times area = tension \times length.
\]

Then

\[
\sigma_{o\theta} \left( \frac{\pi(r_{2}^{2} - r_{1}^{2})}{2\pi} \right) = T_{o\theta}(r_{1}d\theta)
\]

and

\[
T_{o\theta} = \frac{r_{2}^{2} - r_{1}^{2}}{2r_{1}} \sigma_{o\theta}.
\]

Since \( r_{2} = r_{1} + W \),

\[
T_{o\theta} = \frac{W(2r_{1} + W)}{2r_{1}} \sigma_{o\theta}.
\]

(A3)

Similarly,

\[
T_{e\phi} = \frac{W(2R_{1} + W)}{2R_{1}} \sigma_{e\phi}.
\]

(A4)

Equation A2 may then be rewritten

\[
\frac{P}{W} = \frac{2R_{1} + W}{2R_{1}r_{1}} \sigma_{o\theta} + \frac{2r_{1} + W}{2R_{1}r_{1}} \sigma_{e\phi}.
\]

(A5)

If the wall is thin, \( W \) may be neglected compared to \( 2R_{1} \) and \( 2r_{1} \), and equation A5 reduces to the familiar form of Laplace’s law used by Sandler and Dodge (6) and Gault et al. (7):

\[
\frac{P}{W} = \frac{\sigma_{o\theta}}{r_{1}} + \frac{\sigma_{e\phi}}{R_{1}}.
\]

(A6)

Stresses can be calculated at the equator of the ellipsoidal ventricle from this uniform stress model. An overall force balance on the equatorial plane, shown in Figure 2, yields

\[
p\pi r_{2}^{2} = \sigma_{o\phi} \pi(r_{2}^{2} - r_{1}^{2})
\]

or, since \( r_{2} = r_{1} + W \), where \( W \) is the wall thickness

\[
\sigma_{o\phi} = \frac{pr_{1}^{2}}{W} \frac{1}{2r_{1} + W}.
\]

(A7)

Substituting back into equation A5 for \( \sigma_{o\phi} \) gives

\[
\sigma_{o\phi} = \frac{pr_{1}^{2}}{W} \frac{(2R_{1} - r_{1})}{(2R_{1} + W)}.
\]

(A8)

These results are more conveniently expressed in terms of the major and minor axes of the ellipse \( L \) and \( M \). Since

\[
R_{1} = \frac{L^{2}}{2M}, \quad r_{1} = \frac{M}{2},
\]

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Equation A9 is in agreement with the result of Sandler and Dodge (6). Equation A10 differs from theirs, which was obtained from equation A6. The Sandler and Dodge result is valid only for very thin walls, for which $W \ll M$. For the special case of a sphere, where $L = M$, equations A9 and A11 predict unequal values of $\sigma_{\theta \theta}$ and $\sigma_{\phi \phi}$. This is clearly not correct. If $W$ is neglected in the factor $(M + W)$, equation A11 reduces to the correct thin-wall result which was used by Gault et al. (7):

$$\sigma_{\theta \theta} = \frac{pM}{2W} \left( 1 - \frac{M^2}{2L^2(M+W)} \right) \quad (A12)$$

Equations A9 and A10 represent the average principal stresses at the equator. For the special case of a sphere, $L = M$,

$$\sigma_{\theta \theta} = \sigma_{\phi \phi} = \frac{pM^2}{4W(M + W)} .$$

For a very thin-walled ellipsoid, $W$ may be neglected compared to $M$ and $L$. Equation A9 then reduces to

$$\sigma_{\phi \phi} = \frac{pM}{4W} ,$$

and equation A10 reduces to equation A12.

An average stress can also be calculated from the detailed stress distribution obtained by Wong and Rautaharju (13), as shown by Hood et al. (16):

$$\bar{\sigma}_{\theta \theta} = \frac{1}{W} \int_{R_1}^{R_1 + W} \sigma_{\theta \theta} dR$$

$$= \frac{2A}{W} \left[ \frac{(R_1 + W)^{n+1} - (R_1)^{n+1}}{n+1} - \frac{B(R_1 + W)^n}{n+1} \left\{ \left( \frac{R_1 + W}{2} \right)^{\frac{n+1}{2}} - \frac{R_1^{\frac{n+1}{2}}}{2} \right\} \right] , \quad (A13)$$

where

$$A = \frac{p(R_1)^{n+2}}{(R_1 + W)^n - R_1^n} ,$$

$$n = 1 + 2 \frac{L^2}{M^2} ,$$

$$B = \frac{2L^4 + 2L^2M^2 - M^4}{2(L^4 + L^2M^2 + M^4)} .$$

An average stress can also be obtained from Wong and Rautaharju's results by determining the average force per unit area of wall

$$\bar{\sigma}_{\theta \theta} = \left[ \int_{R_1}^{R_1 + W} \sigma_{\theta \theta}(Rd\phi) dR \right] / \left[ \int_{R_1}^{R_1 + W} (Rd\phi) dR \right]$$

$$= \frac{4A}{(R_1 + W)^2 - R_1^2} \left[ \left( \frac{R_1 + W}{2} \right)^{\frac{(n+1)}{2}} - \frac{R_1^{\frac{(n+1)}{2}}}{2} \right]$$

$$- \frac{B(R_1 + W)^n}{n-1} \left\{ \left( \frac{R_1 + W}{2} \right)^{\frac{(n-1)}{2}} - \frac{R_1^{\frac{(n-1)}{2}}}{2} \right\} . \quad (A14)$$
This definition is consistent with the average stress defined by equation A4.

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References

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