Comparison of Directly Measured Left Ventricular Wall Stress and Stress Calculated from Geometric Reference Figures

By John W. Burns, James W. Covell, Richard Myers, and John Ross, Jr.

ABSTRACT

Mean left ventricular wall force was determined with a calibrated transmural auxotonic strain gauge in the left ventricle of six anesthetized, open-chest dogs with intact circulation. The gauge was oriented in the plane of the minor left ventricular equator, midway between the papillary muscles. Left ventricular internal volume was derived from the passive pressure-volume curve of the arrested heart and calculated mean wall stress was derived both from spherical and ellipsoidal reference figures for the left ventricle and compared with measured forces. Control left ventricular end-diastolic pressure averaged 3.0 ± 0.6 mm Hg (xx). At this level of end-diastolic pressure, measured peak wall stress averaged 97.2 ± 14.4 g/cm², whereas calculated peak wall stress averaged 79.3 ± 9.9 and 118.6 ± 12.9 g/cm² for the spherical and ellipsoidal models, respectively. Measured end-diastolic wall force values averaged 9.4 ± 4.5 and 29.2 ± 8.1 g/cm² at an end-diastolic pressure of 3.0 and 12.3 mm Hg, respectively. In all cases, stress values calculated from spherical reference figures for the left ventricle were significantly lower than those measured directly. In four other experiments, using right heart bypass, the ventricular septum was exposed and active wall force was determined at two or more sites on the left ventricular minor equator. Wall stress at these sites differed by an average of 15.3%, indicating that stresses around the minor equator are relatively uniform. These studies lend validity to the application of geometric models in the calculation of mean wall stress and favor the application of an ellipsoid for the geometric reference figure.

KEY WORDS auxotonic force gauge average left ventricular wall force Laplace relationship major axis stress ellipsoid of revolution

The recent interest in assessing cardiac performance in terms of muscle function has provided a stimulus for attempting to evaluate stress and strain within the left ventricular wall. Various techniques have been used to calculate left ventricular wall stress with geometric reference figures. These approaches have varied from the use of a thin-walled spheroid (1, 2) or an ellipsoid of revolution (3, 4) to treatment of the ventricle as a thick-walled, passive elastic body (5-7). More recently, such analyses have been extended to include consideration of fiber angle distribution within the left ventricular wall (8, 9). There are indications that some reference figures may be more appropriate than others (3, 10-12). However, all of this evidence is indirect and does not involve direct measurement of force. Although Hefner et al. (13) reported measurements of auxotonic force in the heart, little is known about the relation...
between wall forces measured in the beating left ventricle and those determined from various geometric reference figures in the same heart. Therefore, in the present study, wall stress was directly measured in the intact left ventricle with an auxotonic strain gauge (14), and the results were compared with values calculated from commonly used geometric reference figures.

**Methods**

Mongrel dogs of both sexes were anesthetized with 30 mg/kg of sodium pentobarbital. Two preparations were used. In both, the animal was studied with the heart exposed and the auxotonic force gauges directly implanted in the left ventricle. In one, right heart bypass was used (15) to provide access to the ventricular septum; in the other preparation, the circulation was intact. Left ventricular volumes and dimensions were obtained from the passive pressure-volume curve of the left ventricle (2) or by Phillips (model Medio 50) direct cineradiographic measurements. The surgical preparation and the implantation of gauges were essentially the same in both preparations. The heart was exposed through a midsternal incision and supported in a pericardial cradle. Aortic flow was measured with an electromagnetic flowmeter (Biotronex Laboratories model 610) and left ventricular pressure was determined with a Statham (P23Db) strain gauge attached directly to a wide-bore steel cannula sutured into the apex of the left ventricle. Major and minor axis force, aortic flow rate, the integral of aortic flow rate (stroke volume) and left ventricular pressure (LVP) were recorded using an oscillographic recorder (Clevite Brush Instruments model Mark 200) at paper speeds of 200 mm/sec. The sinus node was crushed and the heart rate was controlled by pacing the right atrium with an electronic stimulator (American Electronics Laboratory model 410).

In each experiment, two to three different levels of left ventricular end-diastolic pressure (LVEDP) were produced by the transfusion of donor blood or, in the four right heart bypass experiments, by increasing pump output. The relationship of wall force to left ventricular pressure and volume was determined at each of these different levels of LVEDP.

**MEASUREMENT OF WALL FORCE IN THE MAJOR AND MINOR AXES OF THE LEFT VENTRICLE**

Mean left ventricular wall force was measured by a transmural auxotonic strain gauge (Fig. 1), which has been described in detail elsewhere (14). To facilitate adjustment of distance between the pins, a small screw was added between the top support and the knurled set screw. When the set screw was tightened, this additional adjusting device was effectively removed from the system. Since the terms force, stress, and tension have been used in varying ways in the medical literature, for the purposes of this study the term force will be defined as gram weight, or the force equal to one gram mass in a constant gravitational field, and by convention it is expressed in units of g/cm². For the measurement of force in the minor axis of the left ventricle, the gauge was positioned on the free wall at the point of the greatest radius of curvature, perpendicular to the plane of the apex to base axis and midway between the anterolateral and posterolateral papillary muscles. In five of the six experiments, a second gauge was placed slightly caudally in the plane of the apex-to-base axis, i.e., on the major axis of the left ventricle.

To measure auxotonic forces in the left ventricle accurately, it was essential that the muscle between the pins of the gauge (Fig. 1) did not influence the active force recorded by the gauge. Accordingly, following implantation of the gauge, the pins were gradually approximated to relax the muscle between the pins. Figure 2

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**FIGURE 1**

Preparation used for the measurement of wall stress in the major and minor axes of the left ventricle. Inset in upper right shows the relationship between the gauge and the myocardium. Aortic flow was measured with an electromagnetic flow transducer at the root of the aorta. The sinoatrial node was crushed and the heart was paced from the right atrium at a constant rate. Left ventricular pressure was measured through a wide-bore steel cannula sutured into the apex of the left ventricle (L.V.). R.V. = right ventricle; L.A.D. = left anterior descending coronary artery; A.L.P.M. = anterolateral papillary muscle.
VENTRICULAR WALL STRESS

Example of the technique used to determine optimum pin positioning. The points designated by the arrow denote the range over which additional pin approximation does not influence either resting or peak force (see text).

shows an example of the effect of this procedure on the resting and peak forces recorded by the gauge. It is apparent that there was a range where further approximation of the pins produced little or no change in either active or resting force (arrow, Fig. 2). Within this range, it was considered that compression of the segment of muscle between the pins did not contribute to resting force and that this segment of muscle was relaxed and therefore did not affect measured systolic force.

ANALYSIS OF LOCAL WALL FORCES AT MORE THAN ONE MINOR AXIS SITE

In four experiments, right heart bypass (15) was used to allow measurement of wall force at more than one site in the minor circumferential plane. The free wall of the right ventricle was cut away to expose the intraventricular septum, and a second auxotonic strain gauge was implanted on the septum in the same plane on the minor axis, 120° to 180° from the gauge placed at the usual minor axis site.

DETERMINATION OF THE COUPLING OF THE GAUGE TO THE MYOCARDIUM

To precisely evaluate the effectiveness of coupling between the muscle and the transmural pins, experiments were carried out in seven dogs in which the hearts were rapidly excised to obtain a circumferential strip of left ventricular muscle (average dimensions = 13.7 by 1.3 by 1.0 cm).

The strip was suspended and the gauge was placed into the muscle with the pins perpendicular to the long axis of the muscle strip. Various loads were then suspended from the muscle strip. From the weight of the muscle strip and the measured variations in muscle length due to changing loads, the geometry of the muscle was determined and actual stress in the rectangular strip of excised muscle could be calculated. These stresses were compared with those directly measured by the gauge assuming that all the cross-sectional area of muscle subtended by the pins was directly coupled to the gauge. Feigl et al. (14) have clearly shown that the dynamic response of the gauge itself is more than adequate. An assessment of the dynamic coupling of the gauge to the myocardium is more difficult. However, the fact that there was no apparent phase difference between left ventricular pressure and wall force indicates that the dynamic coupling of the muscle to the pins was also adequate (Fig. 3).

Wall force recordings were corrected for variations in myocardial wall thickness by two methods. The first (M0, Table 1) was to divide the myocardial wall force recording throughout systole and for all ventricular end-diastolic volumes by a constant estimate of the cross-sectional area subtended by the pins. The wall thickness subtended by the pins at the end of the experiment was measured and it was assumed that the full cross-sectional area bounded by the pins was coupled to the muscle. The second method (M1) combined correction of myocardial wall force recordings for (1) alterations in wall thickness resulting from the large changes in end-diastolic volume produced in the study (2), dynamic variations in wall thickness, and (3) the effective coupling of the gauge to the myocardium. For the first, left ventricular wall thickness for each contraction was calculated as follows. End-diastolic wall thickness was calculated from internal volume and left ventricular weight, assuming that the left ventricle could be represented by a thick-walled sphere with constant muscle mass (2). This value for end-diastolic wall thickness at the end-diastolic pressure for a given contraction was used as the starting point for further dynamic corrections. For the second correction, the dynamic changes in wall thickness reported by Feigl and Fry (17) were mathematically approximated to permit the calculation of wall thickness at any time during the cardiac cycle following end-diastole. To fit the dynamic changes in wall thickness to an individual systolic contraction, systole was divided into four parts: two during isovolumic
The run number represents the level of LVEDP for that run (low, medium, or high); LV wt = left ventricular weight; length = base to apex length; HR = heart rate; LVEDP = left ventricular end-diastolic pressure; EDV = end-diastolic volume; Meas. WT = measured wall thickness; Calc. EDWT = calculated end-diastolic wall thickness; SV = stroke volume.

The third correction applied to the force determinations corrected for force-dependent alterations in the coupling of the gauge to the myocardium. The linear relationship between force applied to a muscle strip (see above) and the force measured was used to correct all dynamic force measurements.

### TABLE 1

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<th>EDV (ml)</th>
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Mean of all runs: 119.8 g, 8.3 cm, 123.5 mm Hg, 66.6 ml, 45.9 cm, 1.2 cm, 4.8 cm.

The force determinations were corrected for force-dependent alterations in the coupling of the gauge to the myocardium. The linear relationship between force applied to a muscle strip (see above) and the force measured was used to correct all dynamic force measurements.

**Calculation of Left Ventricular Myocardial Wall Stress**

In each study, left ventricular volume was determined directly at the end of the experiment.
with the passive pressure-volume curve technique, which has been described in detail previously (6). However, in the present study, to improve the correlation with ventricular dimensions, a plug 2.8 cm in diameter was tied into the internal cavity adjacent to the minor axis of an ellipsoid of revolution (22), again assuming that the total force at the internal cavity is distributed evenly about the internal cavity. Wall stress was also calculated using a thick-walled ellipsoidal reference figure (22), again assuming that the total force at the internal cavity adjacent to the minor axis of an ellipsoid of revolution is distributed evenly across the wall. Thus, PS = Pr^2 / (r_e^2 + r_i^2) where L = base to apex length and h = wall thickness.

In all experiments for the calculation of stress, a thick-walled spherical reference figure (20) was used, assuming that the total force at the endocardial surface may be represented by the Laplace relationship and that force is evenly distributed over the total left ventricular wall thickness (21). Thus, force equals P \pi r_i^2 and WS = Pr_{v}^2 / (r_e^2 + r_i^2), where P is left ventricular intracavitary pressure in g/cm², r_e is internal left ventricular radius (from V = 4/3 \pi r_e^3), r_i is external left ventricular radius, assuming an even distribution of left ventricular mass about the internal cavity. Wall stress was also calculated using a thick-walled ellipsoidal reference figure (22), again assuming that the total force at the internal cavity adjacent to the minor axis of an ellipsoid of revolution is distributed evenly across the wall. Thus, WS = Pr_{v}^2 / (r_e^2 + r_i^2), where L = base to apex length and h = wall thickness.

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The effect of an increase in end-diastolic pressure produced by the rapid infusion of donor blood upon directly measured minor and major axes left ventricular wall force.

A: Control. B: After transfusion.

**Ao. flow** = aortic root blood flow; **L.V.P.** = left ventricular pressure; **S** = stimulus artifact.

All data obtained in this study were recorded on an FM tape recorder (Honeywell model 7600) for subsequent data analysis. Following the experiment, appropriate experimental runs were selected, A/D converted, and calculations were made with a hybrid computer (Electronics Associates, model 580 and 640). Statistical comparisons were made by paired t-test (24).

**Results**

**FORCE MEASUREMENTS IN THE MAJOR AND MINOR AXES OF THE LEFT VENTRICLE**

Forces recorded from the major and minor axes of the left ventricle are illustrated in Figure 3. The relationship between force in the minor axis and the left ventricular pressure was similar to that observed previously (13, 14). It can also be noted that major axis force differs considerably from that in the minor axis both in magnitude and time course. In all studies, the peak left ventricular major axis force averaged 43.0 ± 3.3% (se) of minor axis force (Table 1). Moreover, the initial rise in major axis force was usually slower and occasionally somewhat delayed relative to minor axis force.

Minor axis force was significantly increased at the higher levels of left ventricular end-diastolic and peak pressure averaging 63.0 ± 6.7 g at an end-diastolic pressure of 3.0 ± 0.6 mm Hg (PF, Table 1), and at the highest levels of LVEDP examined (average 12.5 ± 2.7 mm Hg) minor axis force was 127.3 ± 15.9 g (P < 0.01). Moreover, peak minor axis force could be augmented by increasing LVEDP without a substantial change in LVP (Table 1, expt. 11, 14, 18).

**ANALYSIS OF LOCAL WALL FORCES**

The four experiments in which force was determined at more than one site in the same circumferential plane are summarized in Table 2. Measurements were made at left ventricular end-diastolic pressures ranging from 2.6 to 15.3 mm Hg. Figure 4 shows representative tracings obtained in one such experiment.

Active wall force was measured simultaneously at a site on the left ventricular septum (force 1) and on the free wall (force 2) along the minor axis of the left ventricle. Abbreviations as in Figure 3.
VENTRICULAR WALL STRESS

TABLE 2
Simultaneous Circumferential Stress Measurements

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WTi = wall thickness at gauge on ventricular septum; WT2 = wall thickness at the gauge on the left ventricular free wall; PAsi = peak active stress from the septal gauge; PAso = peak active stress from the free-wall gauge. Other abbreviations as in Table 1.

The time course of active wall force is similar in both gauges and not influenced by changes in left ventricular end-diastolic pressure (Fig. 4B). At all volumes, peak active wall stress from the septal gauges averaged slightly but significantly (P < 0.005) higher than those on the free wall, with the average differences being 15.3 ± 2.8% (Table 2). Occasional differences in end-diastolic wall stress at the two sites were not statistically significant.

DETERMINATION OF THE COUPLING OF THE GAUGES TO THE MYOCARDIUM

In the seven excised muscle strips, stress measured with the gauge averaged 101.3 ± 8.5% of true stress applied by a 30-g weight on the muscle, 97.7 ± 7.2% of true stress at 50 g, 88.5 ± 6.3% at 100 g, and 78.4 ± 6.0% at 150 g. These data indicate that at higher stress levels the gauge measured relatively less of the total stress applied to the muscle. However, in all experiments the relationship between measured stress and actual stress on the muscle was linear and the average relationship as determined by least squares linear approximation of the individual curves was: corrected stress = 1.4 times measured stress - 11.2 g/cm². Recorded wall forces corrected for dynamic changes in wall thickness, as well as for this force-dependent variation in gauge coupling, are referred to as Mo•.

CORRECTION OF DIRECT MYOCARDIAL WALL FORCE DETERMINATIONS

Correction of the wall force recordings by the measured wall thickness (Mo•) provided the most direct measure of ventricular wall stress (Fig. 5, C and D). The average value calculated at low LVEDP was 84.0 ± 8.9 g/cm² and for higher LVEDP was 167.6 ± 28.4 g/cm² (Table 1).

Figure 5 (C and D, open squares) shows the influence of correcting the wall force measurements for dynamic changes in wall thickness and for coupling of the gauge to the myocardium (Mo•). It can be seen that these corrections produce an increase in peak wall stress in addition to a steeper decrease in stress during ejection. The latter finding is
Figure 5 illustrates the relationship between left ventricular pressure (L.V.P.), aortic flow, directly measured minor axis wall stress, and calculated ellipsoidal and spherical stress. In 12 of the 16 measurements, peak left ventricular wall stress calculated from both a spherical and ellipsoidal model (PSo- PEo-) was greater than that measured assuming a constant wall thickness (PMo-, Table 1). In all experiments PMo- averaged 124.1 ± 12.8 g/cm² and was significantly less than both PSo- (141.7 ± 15.8 g/cm², P < 0.05) and PEo- (200.1 ± 19.7 g/cm², P < 0.001). Following correction of the directly measured wall forces for dynamic wall thickness changes and coupling of the gauge to the myocardium, average measured wall stress (PMo-) increased to 192.3 ± 28.8 g/cm². PSo- was lower than PMo- in 11 of 16 determinations (avg. 26% at low levels of end-diastolic pressure and 56% at higher levels). PEo- showed a similar failure to increase proportionally to PMo- with increases in volume. PEo- averaged 18% higher than PMo- at the low levels of end-diastolic pressure and 15% lower than PMo- at the higher levels (Table 1). Moreover, there was a consistent and significant difference between the spherical and ellipsoidal reference figures, PSo- averaging 20% less than PEo- at all levels (P < 0.001). PEo- was a significantly better approximation to PMo-2. Thus, there was no significant difference between PSo- and PEo- while PSo- was significantly lower (P < 0.01) than PMo- (141.7 ± 15.8 g/cm², PSo- and 192.3 ± 28.8 g/cm², PMo-). This lower PSo- was accompanied by significantly lower rates of change of stress during contraction. Thus, the average rate of rise of stress for PMo- from end-diastolic to peak stress was 1773 ± 214 g/cm²/sec and 1414 ±
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180 g/cm²/sec and 2019 ± 224 g/cm²/sec for Scr and Ecr, respectively. The rate of change in Mcr² from peak stress to the end of ejection was 676 ± 82 g/cm²/sec and was 746 ± 57 g/cm²/sec for Ecr and 545 ± 44 g/cm²/sec for Scr.

To examine the relationship between calculated wall stress (Ecr, Scr) and that derived from the direct determination of dimensions (3, 5, 22), an additional experiment was performed in which single plane cineangiograms at 300 frames/sec were obtained. Beads were implanted at the endocardium (25) in the minor axis of the left ventricle, and wall stress was calculated using an ellipsoidal reference figure (22). At an end-diastolic pressure of 3.5 mm Hg, PMcr² was 171.6 g/cm², whereas stress calculated from the ellipsoidal reference figure was 170.9 g/cm².

Discussion

Since the studies of Woods (26) and later Burton (1) and others (27, 28) on the applicability of the law of Laplace to the intact heart, the importance of myocardial wall forces in the study of cardiac physiology has been appreciated. Myocardial wall forces have been shown to be a major determinant of the heart’s energy utilization (29, 30), and the evaluation of myocardial wall stress has been shown to be an important determinant of the intrinsic function of the myocardium in the intact heart (2, 4, 31). Moreover, variations in stress and in the time course of myocardial wall stress development have been shown to be of significance in clinical disease states (22, 28, 32). However, the evaluation of myocardial wall stress in the intact ventricle is at best difficult. The left ventricle is an irregularly shaped thick-walled structure composed of a continuum of fibers oriented at angles to the main axis of the ventricle (9, 33). In generating myocardial wall tension, these fibers undergo marked rearrangements and shear during the cardiac cycle (34). Nevertheless, investigators have estimated myocardial wall stress by a variety of techniques. Most of these techniques assume that the ventricle is a thick-walled ellipsoid of revolution and that it is composed of an isotropic, homogenous layer of muscle (3, 4, 11, 22). It was recognized, however, that these applications of the basic Laplace relationship to thick-walled structures might involve an appreciable error (3). More recently, techniques have been developed to evaluate myocardial wall stress across a thick-walled ellipsoidal shell with nonlinear stress distribution (5-8).

The major finding of the present study is the close agreement between measured wall stress and wall stress calculated from geometric reference figures. Perhaps of even more significance for the studies in which force-velocity parameters are calculated from the rate of change of wall stress (2, 4, 30) is the close correspondence of the rate of change of force during isovolumic and auxotonic systole determined by the three techniques. On the other hand, both Scr and Ecr did not accurately reflect the increase in Mcr² with increases in volume. However, it is possible that at least a portion of this error resulted from underestimating the increase in wall thickening during contraction that occurs with increasing ventricular volume (25). It should be noted that the values obtained in this study averaged about 10% to 20% lower than that estimated in normal unanesthetized man (22). However, when comparisons are made at the same level of end-diastolic pressure, wall stress in this study averaged about 12.5 mm Hg averaged 328.7 g/cm² and compared quite favorably with those calculated in the intact human heart (11, 22, 32). For example, in a recent study by Hood et al. (32) in the normal human left ventricle, the ventricular end-diastolic pressure averaged 12 mm Hg and peak left ventricular wall stress averaged 328 g/cm². Moreover, the average ratio of measured major to minor axis wall stress of 43.0 ± 3.8% observed in this study compared quite favorably with that calculated from the ratio of the numbers of fibers distributed in these two axes and their radii of curvature (8) or with calculated major axis stress (11). The measured wall stresses obtained by this technique compared quite favorably with those obtained in isolated cat papillary muscle.
Assuming an average ratio of auxotonic to isovolumic stress of 30% (36) and an average isometric force in isolated papillary muscle of 320 g/cm² (35), peak force during ejection would be 188 g/cm² and would compare favorably with the average value of PM0-2 (192.3 g/cm²).

The applicability of the many formulations for mean wall stress in the left ventricle as descriptors of ventricular myocardial function depends on the assumption that there are not large local variations in wall force (21). If this were the case, more complex systems for the calculation of myocardial wall force would become necessary (6, 7). In the current study, at least in the minor equatorial plane, only small variations in wall stress were observed (Table 2), thus supporting the use of mean wall stress as a descriptor of myocardial function in the circumferential plane.

Although the linearity and dynamic response of the gauges used in this current study have been described in detail previously (14) and are quite adequate for the measurements obtained in this study, the direct measurements of myocardial wall stress described in these studies are not without potential error. The major problem with gauges of this type is the coupling of the myocardial wall to the pins. In the present study, an attempt was made to assess the adequacy of this coupling in full-thickness left ventricular strips. The highly significant linear relationship obtained between measured stress and applied stress across the gauge reveals that this gauge can indeed be used to provide an adequate description of the force subtended by the entire surface area bounded by the pins. Another drawback associated with these stress measurements is the fact that instantaneous wall thickness at the gauge could not be directly measured. However, even the assumption that wall thickness was fixed at the value measured at the end of the experiments (Table 1, PM0-1), or that it varied throughout systole as described by other authors (22, 23), did not lead to large quantitative or qualitative alterations in measured wall stresses.

Despite wide variations in techniques and assumptions, it is interesting that very little quantitative difference appears to exist between myocardial wall stresses calculated from either thick-walled or thin-walled ellipsoidal reference figures (7, 10, 11, 30, 32). The results of the present study reinforce these conclusions and, in addition, have shown that there are relatively minor differences between directly measured myocardial wall stresses and wall stress calculated assuming relatively simple geometric reference figures, a sphere or ellipsoid, and thin-wall theory for the basis of the calculation. However, stresses calculated from both geometric reference figures failed to increase proportionally to the increase in measured stress with increasing ventricular volume. Although both the sphere and ellipse gave good approximation of measured wall stress, the results of the current study indicate that an ellipsoidal reference figure provides a better overall estimate of wall stress.

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References

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Comparison of Directly Measured Left Ventricular Wall Stress and Stress Calculated from Geometric Reference Figures
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