Evaluation of Several Geometric Models for Estimation of Left Ventricular Circumferential Wall Stress

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ABSTRACT

Phasic left ventricular wall force and wall thickness were monitored with appropriate transducers to provide a direct measurement of circumferential wall stress in open-chest dogs. Left ventricular pressure and measurements of chamber geometry were used to estimate the wall stress using several geometric models. During the initial control period, peak and end-ejection measured wall stresses were 207 ± 19 and 104 ± 13 g/cm², respectively. The best estimates of these values were 198 ± 18 and 117 ± 11 g/cm² obtained from a modified thin-wall ellipse formula in which the midwall rather than the endocardial radius was used. Wide variations in hemodynamic conditions were produced with intravenous infusions of nitroglycerin, phenylephrine, and isoproterenol. Comparison of directly measured and estimated values during all control periods and during the response to these interventions showed that both the modified thin-wall ellipse and a thick-wall ellipse were generally accurate predictors of the measured wall stress. All other models tended to underestimate measured stress. The sensitivity of the estimated wall stress computed by the models to geometric measurement errors was also evaluated. A thick-wall sphere was the most sensitive to both circumferential length and wall thickness measurement errors, and a thick-wall ellipse was the least sensitive. All models examined were relatively insensitive to base-to-apex length measurement errors.

KEY WORDS  
dogs  
thick-wall elliptical ventricular model  
fiber-oriented elliptical ventricular model  
thin-wall elliptical ventricular model  
spherical ventricular model  
ventricular geometric models

An important aspect of left ventricular function concerns the condition of equilibrium which must exist between the stresses in the wall of the chamber and the blood pressure developed within the cavity. The ability of the heart to vary its geometry is responsible for maintaining this equilibrium and forms the basis for Laplace's law. The desire to estimate the wall stress from measurements of left ventricular pressure and shape has led to the development of numerous geometric models of the left ventricle (1-13). To establish the validity of a particular model it is necessary to estimate the wall stress by an independent technique and compare this value with the estimate computed from left ventricular pressure and the geometric parameters contained in the model. A valid model which estimates wall stress accurately would have important physiological uses, especially if only a measurement of left ventricular pressure and an estimate of cardiac dimensions were required since these parameters can be obtained readily in patients.

Several studies have attempted directly to measure the tension or stress in the left ventricular wall. Hefner et al. (14) developed a force gauge which was inserted in series with the myocardial fibers and measured the tension generated during contraction. No direct measurement of ventricular wall thickness was obtained, and these workers did not attempt to calculate wall stress. Burns et al. (15) used a transmural auxotonic strain gauge to measure the circumferential left ventricular wall force. The internal volume of the ventricle was estimated either from a postmortem pressure-volume curve of the left ventricle or by cineradiography rather than measured directly, and changes...
in wall thickness were approximated from previously published data. Comparison of the measured values with those predicted by a thick-wall sphere and a thick-wall ellipse showed that, in all cases, stress values obtained from the spherical model were significantly lower than the measured values. The results obtained by Burns et al. (15) favored the application of an ellipsoidal model to estimate left ventricular wall stress. Lewartowski et al. (16) directly measured the tension within the left ventricular wall. Stress was computed by dividing the tension by an estimate of wall thickness obtained by inserting a calibrated wire across the wall. The measured stress was compared with estimates obtained from a thick-wall ellipse (12) and a thin-wall ellipse (3). Both models overestimated the measured stress, although the differences were small for the thick-wall ellipse (18).

The experiments described in this report were designed to compute the time course of stress in the free wall of the left ventricle during the cardiac cycle from directly measured values of left ventricular wall force and wall thickness. The data obtained in this manner will be termed "measured" left ventricular wall stress throughout this report. An auxotonic transducer (17) was used to measure the wall force, and a mutual inductance transducer (18) specifically designed for this study, provided phasic measurements of wall thickness. An electrical recording caliper (19) was used to measure the length changes of a circumferential segment of the left ventricle. Left ventricular pressure and these geometric data were used to compute the estimated wall stress by employing several geometric models. The individual models chosen for evaluation are representative of the various classes of proposed models.

The measured and estimated values of wall stress were compared to determine the ability of the various models to predict left ventricular circumferential wall stress. In addition, the models were examined to determine the sensitivity of the predicted wall stress to errors made in measuring the geometric parameters. The results of this analysis have important implications concerning the clinical application of these models in evaluating left ventricular function.

**INSTRUMENTATION**

**Methods**

It is essential that the measurements of wall force and cardiac dimensions be as accurate as possible. The static and dynamic response characteristics of the force transducer, wall thickness gauge, and displacement caliper were evaluated in some detail before this series of animal experiments was initiated (20).

An auxotonic force transducer (17) was used for this investigation. Static calibration of this instrument was performed by suspending the gauge in the vertical position and hanging known weights on it. The calibration curve, consisting of points obtained during both loading and unloading, was linear over the range of 0 to 300 g. The dynamic response was evaluated by adding a 100-g weight to one set of coupling pins as rapidly as possible while recording the transducer output at a paper speed of 100 mm/sec. The undamped natural frequency was 30 Hz. To further establish the validity of the force recorded with the auxotonic instrument as employed in this study, the following experiment was carried out. Under general anesthesia, the semimembranosus muscle of a dog hind limb was dissected free, leaving the blood supply and the nerve intact. The tibial insertion of this muscle was detached and directly coupled to a Biocom model 1030 cantilever type of gauge to record the total muscle force. An auxotonic cardiac force transducer was implanted near the midpoint of the muscle with the coupling pins squeezed completely together to inactivate the muscle between them. Simultaneous recordings of the total muscle force and the force measured by the myocardial gauge were made during muscle stimulation. When the steady-state data were converted to stress measurements by dividing them by the appropriate cross-sectional areas, the values were essentially identical. In addition, a time constant, defined as the time required to reach 63% of the steady-state value, was calculated for the loading phase of each curve. This value for the total muscle force was 70 msec and that for the cardiac force transducer was 120 msec. During the unloading phase of the cycle the time constant for both curves was 40 msec. These results indicate that the output of the cardiac force transducer tends to lag the actual developed force as the muscle is stimulated but follows the relaxation phase accurately. When the transducer is used on the left ventricle, this lag will result in an understimation of the peak force developed in early systole, but the decrease in force which occurs as systole progresses will be indicated accurately. It should be pointed out that some of the lag in the skeletal muscle experiment probably was due to inadequate mechanical coupling caused by disruption of the linear array of the skeletal muscle fibers by the coupling pins. The coupling of the gauge to cardiac muscle, with its interlocking network of fibers, should be better than that described for skeletal muscle. The results of these studies tend to confirm the validity of the absolute values of left ventricular wall force measured with this instrument.

The mutual inductance wall thickness transducer has been described in detail previously (18). Static calibration was performed by attaching the transducer to a micrometer vise which provided a precisely adjustable jaw separation. The calibration curve was linear throughout the range of ventricular wall thickness values encountered in the animal studies. A
variable-frequency displacement generator was used to determine the dynamic amplitude response of this measuring system, and it was flat to 15 Hz. To evaluate the coupling of this gauge to the myocardium, a Hyecam high-speed motion picture camera was used to record the motion of one of these instruments on a beating heart at 1000 frames/sec. The epicardial induction coil appeared to be well coupled to the heart wall throughout the cardiac cycle and no surface "denting" was visible when these films were viewed at 16 frames/sec.

The displacement caliper described by Mallos (19) was modified by adding four pins to the feet of the instrument to improve the mechanical coupling to the heart. These pins were long enough to protrude completely through the ventricular wall. Static calibrations, obtained with a micrometer vise, were linear over the range of interest. The frequency response of this type of transducer was flat to 20 Hz (19).

ANIMAL STUDIES

Complete studies were obtained in seven adult mongrel dogs weighing 23-32 kg and were performed 7-10 days after implantation of an electromagnetic flowmeter probe around the ascending aorta. This prior preparation allowed the flowmeter probe to become firmly seated and provided a noise-free signal with a stable baseline. The dogs were sedated with an injection of morphine sulfate (30 mg, im) prior to induction of anesthesia with alpha-chloralose (80 mg/kg, iv). Positive-pressure respiration was instituted, a left thoracotomy was performed, and the heart was suspended in a pericardial cradle. A mutual inductance wall thickness transducer was mounted on the free wall of the left ventricle by the technique described previously (18). An auxotonic force transducer was installed on the free wall as near the equator as possible and oriented to measure circumferential wall force. The coupling pins of this instrument, which extended through the wall, were squeezed together and locked to completely inactivate the piece of ventricular muscle between the pins. A displacement caliper was also positioned as close to the equator as possible to measure length changes of this circumferential segment. Figure 1 illustrates the various transducers on the left ventricular free wall. A stiff nylon cannula (10 cm long, 2 mm, i.d.) was inserted through the apex of the heart. The cannula used to record left ventricular pressure is inserted in the apex. A = auxotonic wall force transducer, B = wall thickness transducer, and C = electrical recording caliper. The cannula used to record left ventricular pressure is inserted in the apex. The electromagnetic flowmeter probe on the ascending aorta is not illustrated.
determined by weighing. A silicone casting material was used to passively distend the ventricle, taking care not to distort the shape of this chamber. The distance between the holes left by the displacement caliper coupling pins was measured, and the circumference of the ventricle was determined at the level where the caliper had been installed. This measurement was obtained at a segment length within the range of values which had been recorded during the study. The base-to-apex length was also measured. When the casting material had hardened, the ventricle was slit open and the location of the force gauge coupling pin holes was determined to ensure that these pins had protruded through the ventricular free wall and not into a papillary muscle. The nominal wall thickness was also measured to check the dynamic thickness recordings.

**DATA ANALYSIS**

All data analysis was carried out using an IBM model 1130 digital computer system. A Redcor model 663 analog-to-digital converter was used to digitize the previously recorded data at a sampling rate of 200/sec. Five data channels representing wall force, left ventricular pressure, circumferential segment length, wall thickness, and ascending aortic blood flow were digitized in this manner and entered into the computer. The time course of wall force and aortic blood flow were graphically displayed. The onset and the end of mechanical systole were manually identified from the force data, and the onset and the end of ejection were similarly defined from the aortic blood flow data.

The following hemodynamic parameters were calculated for each beat. The duration of the cardiac cycle, measured between the end of ejection of two successive beats, was used to compute the heart rate. Stroke volume was determined as the integrated area under the phasic aortic blood flow curve during ejection. Cardiac output was computed as the product of heart rate and stroke volume. Peak systolic pressure was the maximum left ventricular pressure attained during systole. The onset and the end of mechanical systole were used to define the end-diastolic and the endsystolic point, respectively.

The circumferential segment length data were converted to values representing the external radius of the left ventricle at the equator. It was assumed that the recording caliper measured a constant fraction of the circumference of the ventricle at the level at which it was installed. As noted above, a calibration constant was determined at the end of the study as the ratio of the total circumference to the distance between the caliper coupling pin holes as measured on the arrested heart. It was assumed that this calibration value would be the same if the caliper had been positioned exactly on the equator. External circumference was calculated by multiplying the segment length data by this calibration value. The external radius was then calculated as the external circumference divided by 2π. The values of external radius at end-diastole and endsystole were identified along with the corresponding values of left ventricular wall thickness. Internal ventricular radius was determined by subtracting the wall thickness from the external radius. The base-to-apex length, as measured on the arrested heart, was assumed to be constant during ejection.

Measured left ventricular circumferential wall stress (g/cm²) was obtained at each 5-msec data point as the measured force divided by the product of the distance across the coupling pins of the force gauge (0.5 cm) and the measured wall thickness. It was assumed throughout this study that the diastolic wall stress was essentially zero. The value at end-ejection was measured and a mid-ejection value was determined at a point midway in time between the peak and end-ejection. The percent decrease in wall stress to the mid-and end-ejection points was computed using the peak value as the reference.

Estimated left ventricular wall stress was computed at each 5-msec data point using the following geometric models.

**Ellipse 1:** \( \sigma_e = \frac{P}{h} \left[ 1 - \frac{r_e^2}{a^2(3r_e + h)} \right] \),

where \( P = \) left ventricular pressure, \( r_e = \) internal ventricular radius, \( h = \) wall thickness, \( a = \) base-to-apex semiaxis, and \( \sigma_e = \) circumferential wall stress. This model is the thin-wall ellipse proposed by Sandler and Dodge (3). They calculated the mean wall stress using the endocardial radius and assumed that this stress was uniformly distributed across the wall.

**Ellipse 2:** \( \sigma_e = \frac{P}{h} \left[ 1 - \frac{r_m^2}{a^2(2r_m + h)} \right] \).

This model is the same as ellipse 1 except that the midwall radius, \( r_m \), has been used in place of the internal ventricular radius, \( r_e \).

**Ellipse 3:** \( \sigma_e = \frac{P}{h} \left( 2a^2 - r_e^2 \right) \).

This thick-wall ellipsoid model was proposed by Falsetti et al. (12) in an attempt to account for the relatively large wall thickness encountered in the left ventricle.

**Ellipse 4:** \( \sigma_e = \frac{P g_i}{\Delta h \sum_{i=1}^{n} k g_i} \),

where \( k_i = \) estimated myocardial fiber curvature, \( g_i = \) normalized surface area, and \( g_i = \) normalized endocardial surface area Streeter et al. (13) developed this thick-wall model which takes the myocardial fiber orientation into consideration. The details of this model are too lengthy to be presented completely in this report, and the original paper of Streeter et al. (13) should be consulted.

**Sphere:** \( \sigma_e = \frac{P r_e^2}{r_e^2 - r_i^2} \),

where \( r_e = \) outer ventricular radius. This formulation for a thick-wall sphere has been used by several investigators (5, 8).

The peak, mid-ejection, and end-ejection values for these estimated wall stress data were determined in the same manner as were the directly measured values. All
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Statistical comparisons of control and response data were carried out using paired t-tests (21). The effect of circumferential segment length, base-to-apex length, and wall thickness measurement errors on the directly measured and the estimated peak wall stress values was evaluated using the data for one dog. A relative error was introduced into these parameters independently, and the measured and estimated peak wall stress values were recomputed using the various geometric models. These values then were compared with those obtained when no relative error was introduced into the calculation.

Results

A typical recording obtained during an initial control period is shown in Figure 2; the characteristic time course of ventricular wall force can be seen. Wall force rose rapidly to a peak which occurred at the onset of ventricular ejection and then declined as systole progressed, reaching a distinct shoulder at the end of ejection. Notice that, although the measured force decreased to approximately 50% of its peak value, the left ventricular pressure was maintained practically constant during ejection. Notice also the time course of changes in left ventricular wall thickness. An initial increase of 7-8% was recorded during the isovolumic phase of contraction, and an additional increase of similar magnitude occurred during ejection. The hemodynamic and geometric data for the three interventions used in this study are shown in Table 1. Nitroglycerin reduced peak systolic blood pressure by 16% (Table 1). The hemodynamic data for the NTG and PHEN interventions are presented in Figure 2. The hemodynamic and geometric data for the ISOP interventions are presented in Table 1. Nitroglycerin reduced peak systolic blood pressure by 16% (Table 1).

Table 1: Hemodynamic and Left Ventricular Dimension Data

<table>
<thead>
<tr>
<th>Intervention</th>
<th>Heart rate (beats/min)</th>
<th>Stroke volume (ml)</th>
<th>Cardiac output (ml/min)</th>
<th>Peak systolic blood pressure (mm Hg)</th>
<th>End-diastolic radius (cm)</th>
<th>End-systolic radius (cm)</th>
<th>Ventricular wall thickness (cm)</th>
</tr>
</thead>
<tbody>
<tr>
<td>NTG Control</td>
<td>144 ± 7</td>
<td>12 ± 1</td>
<td>1700 ± 200</td>
<td>118 ± 7</td>
<td>2.60 ± 0.11</td>
<td>2.30 ± 0.08</td>
<td>1.16 ± 0.05</td>
</tr>
<tr>
<td>Response</td>
<td>166 ± 8</td>
<td>12 ± 1</td>
<td>2000 ± 200</td>
<td>99 ± 6</td>
<td>2.57 ± 0.11</td>
<td>2.27 ± 0.10</td>
<td>1.16 ± 0.05</td>
</tr>
<tr>
<td>P</td>
<td>&lt;0.005</td>
<td>NS</td>
<td>NS</td>
<td>&lt;0.01</td>
<td>&lt;0.005</td>
<td>NS</td>
<td>NS</td>
</tr>
<tr>
<td>PHEN Control</td>
<td>150 ± 7</td>
<td>12 ± 1</td>
<td>1800 ± 200</td>
<td>116 ± 9</td>
<td>2.50 ± 0.12</td>
<td>2.31 ± 0.10</td>
<td>1.12 ± 0.06</td>
</tr>
<tr>
<td>Response</td>
<td>106 ± 11</td>
<td>17 ± 2</td>
<td>1800 ± 200</td>
<td>152 ± 8</td>
<td>2.75 ± 0.15</td>
<td>2.45 ± 0.09</td>
<td>1.14 ± 0.06</td>
</tr>
<tr>
<td>P</td>
<td>&lt;0.01</td>
<td>&lt;0.05</td>
<td>NS</td>
<td>&lt;0.01</td>
<td>&lt;0.005</td>
<td>NS</td>
<td>NS</td>
</tr>
<tr>
<td>ISOP Control</td>
<td>133 ± 7</td>
<td>14 ± 1</td>
<td>1900 ± 200</td>
<td>122 ± 11</td>
<td>2.65 ± 0.12</td>
<td>2.34 ± 0.09</td>
<td>1.14 ± 0.06</td>
</tr>
<tr>
<td>Response</td>
<td>181 ± 6</td>
<td>14 ± 1</td>
<td>2500 ± 300</td>
<td>140 ± 12</td>
<td>2.69 ± 0.13</td>
<td>2.25 ± 0.11</td>
<td>1.18 ± 0.07</td>
</tr>
<tr>
<td>P</td>
<td>&lt;0.01</td>
<td>NS</td>
<td>&lt;0.01</td>
<td>&lt;0.01</td>
<td>&lt;0.01</td>
<td>NS</td>
<td>&lt;0.01</td>
</tr>
</tbody>
</table>

Values represent control and maximum response observed after administration of nitroglycerin (NTG), phenylephrine (PHEN), and isoproterenol (ISOP). Results are given as means ± se. P values compare the intervention response to its appropriate control. NS = not significant.
pressure by 16% \((P < 0.01)\); left ventricular end-diastolic radius decreased slightly \((P < 0.005)\), and wall thickness was not significantly changed \((P > 0.6)\). Phenylephrine increased peak systolic blood pressure by 32% \((P < 0.001)\), but the wall thickness was unchanged \((P > 0.5)\). Isoproterenol slightly increased peak systolic blood pressure \((P < 0.05)\). The end-diastolic ventricular radius was slightly decreased \((P < 0.01)\), and the end-diastolic wall thickness was increased by 4% \((P < 0.01)\). The left ventricular masses in these seven dogs ranged from 97 to 150 g with a mean of 129 g.

Measured left ventricular wall stress data during the control periods and in response to the three interventions are shown in Figure 3, and the characteristic time course of the wall stress has been sketched. Notice that the phasic configuration of measured wall stress is similar to that of the recorded wall force shown in Figure 2.

A comparison of directly measured and estimated wall stress values before and during the nitroglycerin infusion is shown in Figure 4. The peak, mid-ejection, and end-ejection values are plotted to describe the time course of wall stress during ejection, and the directly measured data are repeated at the far left to facilitate comparison. Ellipse 2 correctly estimated the wall stress during both control and response; ellipse 3 underestimated the peak during the control period but did well otherwise. All other models underestimated the measured wall stress throughout ejection. Figure 5 shows the model comparison results before and during phenylephrine infusion. Ellipse 2 again correctly estimated the wall stress during the control period but did not predict the large peak value measured during the response. Ellipse 3 underestimated the peak stress during both the control and the response period. All other models underestimated the measured wall stress. The estimated wall stress values obtained during isoproterenol infusion are shown compared with the
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directly measured values in Figure 6. Ellipse 2 correctly estimated the wall stress during control but did not predict the time course during the response. Ellipse 3 again did not predict the peak stress during control, but this model did follow the time course during the response. All other models underestimated the measured wall stress.

The comparison of the estimated stress values with the directly measured data can be summarized as follows: ellipse 2 and ellipse 3 were good predictors of wall stress under most conditions and all other models underestimated the wall stress. Ellipse 2 did not predict the enhanced systolic decrease measured during isoproterenol infusion, and ellipse 3 tended to underestimate the peak stress.

Although ellipse 4 and the spherical model produced poor estimates of wall stress, it is possible that they might still predict the percent decrease which occurs during ejection with reasonable accuracy. This possibility was tested by normalizing the data for these two models in the following manner. The estimated peak stress was set equal to the measured peak stress, and the mid-ejection and end-ejection points for each model were computed from the percent change which occurred in the original estimated values. This analysis showed that these models were good predictors of the percent decrease in wall stress during ejection. This finding indicates that a scaling factor added to the original equations would make these models good predictors of left ventricular wall stress. In the case of the spherical model, such an addition would be convenient because the basic simplicity of the equation lends itself to ease of calculation.

Figure 7 demonstrates the effect that errors in the measurement of circumferential segment length will have on the directly measured and the estimated peak wall stress. These errors correspond to errors made in measuring the minor semiaxis of the elliptical models or the radius of the spherical model. The thick-wall sphere was the most sensitive model in this respect, and the thick-wall ellipse (ellipse 3) was the least sensitive, with the other models falling between these two extremes. Segment length does not enter into the calculation of directly measured wall stress. A similar analysis for base-to-apex length measurement errors is shown in Figure 8. It is of particular interest that the elliptical models examined in this study were relatively insensitive to this geometric parameter. The same analysis for errors in wall thickness
measurements is shown in Figure 9. Again, the spherical model was the most sensitive, and the fiber-oriented model (ellipse 4) was the least sensitive.

**Discussion**

The present study is the first in which dynamic measurements of both ventricular wall force and thickness were made simultaneously to provide a direct estimate of wall stress. It is of interest to compare the results of this study with those measured previously. Burns et al. (15), using a similar instrument, found that the measured peak force was $101 \pm 14$ g at a left ventricular end-diastolic pressure of 6 mm Hg. At the same end-diastolic pressure, the peak force measured in the present study was $126 \pm 10$ g. This discrepancy could be due to the myocardial depressant effect of sodium pentobarbital anesthesia in the animals studied by Burns et al. (15). However, when these investigators converted their force data to stress measurements and corrected for what they felt to be inadequate coupling of the force gauge to the myocardium, they found that the peak left ventricular circumferential wall stress was $196 \pm 27$ g/cm². This value compares quite favorably with the value of $207 \pm 19$ g/cm² found in the present study at the same left ventricular end-diastolic pressure. Based on the results of the experiment with the dog semimembranosus muscle described above, it was not felt that any correction for inadequate coupling was necessary in the studies reported in the present paper. The decrease in measured wall stress as systole progressed was also noted by Burns et al. (15). Lewartowski et al. (16) reported a control peak wall stress of $96 \pm 12$ g/cm² but did not indicate the left ventricular end-diastolic pressure at which this value was measured. Thus it is not possible to compare the measured peak stress found by Lewartowski et al. (16) with the results of the present study. These authors (16) and Burns et al. (15) observed that the measured stress increased considerably as left ventricular end-diastolic pressure was raised.

Comparison of the directly measured and the estimated wall stress values revealed that ellipse 2 provided reasonable estimates of peak stress except during the maximum stress achieved with phenylephrine infusion and failed to follow the systolic time course during the response to isoproterenol.
Ellipse 3 tended to underestimate the peak stress during all control periods and during the response to phenylephrine infusion; however, this model did predict the time course of the isoproterenol response. Thus, neither of these models estimated the wall stress correctly in all situations, but ellipse 2 was an accurate predictor during the control periods and ellipse 3 was useful in predicting the isoproterenol response. If estimates of peak wall stress alone were of interest, ellipse 2 would be the model of choice.

Burns et al. (15) compared the measured wall stress with that predicted from a thick-wall sphere and a thick-wall ellipse. Using the same formulation for the spherical model as was used in the present study, they found that this model consistently underestimated the measured wall stress (15). This result was confirmed by the studies reported here. The thick-wall ellipse model evaluated by Burns et al. (15) was not the same as either ellipse 3 or ellipse 4 and thus no direct comparison of results is possible. Due to the lack of information concerning the level of left ventricular end-diastolic pressure at which measurements were obtained, it is not possible to compare the results of the model evaluation of Lewartowski et al. (16) with those of the present study.

Ellipse 4, which takes myocardial fiber orientation into consideration in a thick-wall model, did not estimate the stress as well as might be expected from a model of this complexity. The reason for this underestimation was not immediately apparent. However, this model did predict the percent decrease from peak wall stress which occurred as ejection progressed. This finding implies that scaling the predicted values by some constant would make this model a good estimator of the absolute stress values, but there is nothing inherent in the model which would provide a rational basis for this scaling procedure. Thus, the inclusion of fiber orientation has been helpful in predicting the time course of wall stress, but the model underestimates the absolute stress values.

Ellipse 1, which is the same geometric model as ellipse 2 except that the endocardial radius rather than the midwall radius is used, estimated quite different values for the wall stress. Since the difference between the endocardial and midwall radii is on the order of 5 mm, a small measurement error will lead to widely varying estimates of wall stress.

The sensitivity of estimated wall stress values to measurement error will be a serious limitation when these models are used in a clinical situation. This fact is demonstrated in Figures 7-9 for the models examined in this report. The thick-wall sphere was particularly sensitive to measurement error and thus has a serious disadvantage when it is used to predict absolute values of wall stress. Ellipse 2 and ellipse 3 were the least sensitive to segment length and wall thickness measurement errors. As noted previously, all ellipse models examined were relatively insensitive to base-to-apex length measurement errors.

In summary, the comparison of measured wall stress with estimates obtained from various geometric models of the left ventricle has shown that reasonably accurate estimates of this parameter can be computed using a slight modification of the thin-wall ellipse of Sandler and Dodge (3). This modification consists only of employing the midwall radius rather than the endocardial radius as the minor axis of the ellipse. This model will underestimate the peak stress if the left ventricular pressure is high and will not accurately describe the systolic time course during inotropic stimulation. Except in these situations, this thin-wall ellipse model will serve as an adequate predictor of left ventricular circumferential wall stress in the open-chest dog, provided sufficient care is taken when measuring the geometric parameters.

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