Comparison of Calculations of Left Ventricular Wall Stress in Man from Thin-Walled and Thick-Walled Ellipsoidal Models

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ABSTRACT
Using angiocardiographic data from 50 human subjects, a comparison was made of calculations of circumferential wall stress in the left ventricle based on the thin-walled ellipsoidal model of Sandier and Dodge and the thick-walled ellipsoidal model of Wong and Rautaharju. The Sandier and Dodge formula consistently overestimated mean stress as determined from the Wong and Rautaharju model. The degree of overestimation in terms of percent error usually varied between 5% and 15% and overall averaged about 10% at end-systole as well as at end-diastole. Analysis of the various factors influencing the discrepancy between calculations indicated that the expected increase in error associated with an increase in wall thickness during systole tended to be mitigated by a concomitant change in chamber geometry, specifically, an increase in the ratio of major to minor semiaxis. This study, then, offers an estimate of the error introduced by employing the Sandier and Dodge or similar thin-walled ellipsoidal models for computation of mean circumferential stress.

ADDITIONAL KEY WORDS wall thickness circumferential stress stress distribution computers in medicine chamber geometry

Following the classic papers of Burch, Ray, and Cronvich (1) and Burton (2) on the importance of cardiac geometry, attention was focused on the forces acting within the wall of the left ventricle. In keeping with Woods' (3) earlier emphasis on the applicability of Laplace's law to the heart, later workers (4, 5) used a thin-walled spherical model to estimate wall force in terms of membrane tension, i.e., force per unit circumferential length. Subsequently it was appreciated that the left ventricle is more closely approximated by a thick-walled ellipsoid (6, 7) and that wall force is more suitably considered in terms of stress (force per unit cross-sectional wall area) (8). In 1963 Sandier and Dodge (9) described an angiographic technique for estimating mean wall stress at the equator of an ellipsoidal ventricle. Their analysis was founded on the basic Laplace relation modified to incorporate wall thickness, and they pointed out that such an application of thin-wall theory to a thick-walled structure could introduce appreciable error into the calculations. Recently Wong and Rautaharju (10) developed equations for defining the nonlinear stress distribution across a thick-walled ellipsoidal shell, making possible for the first time a comparison of mean stress calculations from thin- and thick-walled ellipsoidal models. Such a comparison forms the basis of the present report.

Methods
Biplane angiocardiograms obtained in 50
human subjects during diagnostic left heart catheterization for the purpose of computing left ventricular volume constituted the material of this study. On the basis of clinical, hemodynamic, and angiographic criteria, the subjects were divided into several physiologic categories. Five subjects with functional systolic murmurs and a sixth with mild pulmonic stenosis were classified as having normal left ventricles. Nine patients had mitral valve areas calculated as less than 1.5 cm² and were classified as mitral stenosis. There were 19 patients with varying degrees of aortic or mitral regurgitation but without clinical evidence of congestive heart failure. These subjects were classified as volume overload, compensated. Five patients with resting mean systolic pressure gradients across the aortic valve of greater than 45 mm Hg but without clinical evidence of failure were classified as pressure overload, compensated. Nine subjects had decompen-sated pressure or volume overload or primary myocardial disease with clinical evidence of marked failure. Two patients with markedly thickened left ventricular walls but normal-sized cavities were classified as having idiopathic myocardial hypertrophy. Dynamic subaortic obstruction was demonstrated in one of these.

The biplane angiograms (obtained at a filming speed of 6/sec for 5 sec) were used to derive chamber dimensions and wall thickness of the left ventricle throughout the cardiac cycle. Details of the technique employed in this laboratory have been reported previously (11). In brief, the major semiaxis, a, of the chamber was taken to be one-half the longest length measurable within the cavity silhouette on either the anteroposterior or lateral film. The planimetered silhouette area and measured longest length from each x-ray film were substituted into the equation for area of an ellipse in order to derive the minor axis (6). Minor axes from each anteroposterior and lateral film pair were averaged (geometric mean) and divided by two to give the minor semiaxis, b, of an idealized prolate ellipsoid. Wall thickness was derived as the average width of a 4-cm segment of free wall immediately below the equator on each anteroposterior film. After correction for x-ray magnification, cavity dimensions and measurements of wall thickness were related to simultaneous chamber pressure to calculate circumferential (latitudinal) wall stress by the methods outlined below. The stress values from several cardiac cycles were rearranged into a composite curve according to time after onset of the QRS complex.

Calculations
In the thin-walled ellipsoidal model of Sandler and Dodge (S&D), average circumferential stress, $\widetilde{\sigma}$, across the wall at the equator is approximated by the following expression:

$$\widetilde{\sigma}_{S&D} = \frac{Pb}{h} \left[ 1 - \frac{b^2}{a^2(2b + h)} \right], \quad (1)$$

where $a = \text{major semiaxis (cm)}$, $b = \text{minor semiaxis (cm)}$, $P = \text{pressure (dynes/cm²)}$, and $h = \text{wall thickness (cm)}$.

In addition to pressure, chamber dimensions, and wall thickness, the thick-walled model of Wong and Rautaharju takes into account Poisson's ratio, $\mu$, which is the ratio of lateral strain (decrease in width per unit width) to longitudinal strain (increase in length per unit length). If the ratio is 0.5, total volume of an elastic body is unchanged when it is stretched (12). In their report Wong and Rautaharju (10) used a value of 0.25 for Poisson's ratio. This implies that the total volume of muscle increases on stretching. The actual value for this constant in cardiac muscle has not been established, but studies in other biological tissues have suggested that Poisson's ratio is approximately 0.5 (13, 14).

Since the left ventricle consists of about 80% water by weight (15), it is logical to assume that ventricular myocardium would extend isovolumetrically (12). Hence, in the present study a value of 0.5 was used for Poisson's ratio.

Wong and Rautaharju developed a general equation permitting calculation of stress at any level from apex to base, whereas Sandler and Dodge solved for stress only at the equator. Consequently, for the purpose of comparison in the present study, circumferential stress calculations were made only at the equator. Application of thick-wall theory at the equator permits simplification of the general Wong and Rautaharju (W&R) equation into an expression using the same terms as that of equation 1. If a value of 0.5 is assigned to $\mu$, circumferential stress at any depth, $T$, in the wall in the plane of the equator is given by:

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\[
\sigma = \frac{PR_o}{(R_o + h)^n - R_o^n} \left[ 1 + \left( \frac{3b^4}{2(a^4 + a^2b^2 + b^4)} \right) \left( \frac{R_o + h}{R_o} \right)^n \right] (2)
\]

where \( R_o = \) longitudinal radius of curvature at the endocardium = \( a^2/b \), \( R = R_o + T \), \( n = \frac{2a^2 + b^2}{b^2} \), and \( a, b, P, \) and \( h \) are as previously defined.

In the present study, stress distribution across the ventricular wall was determined by using equation 2 to calculate stress at the endocardium, at the epicardium, and at nine evenly spaced points between. Mean stress could have been approximated by taking the average of these 11 values. For greater accuracy, however, mean circumferential stress was calculated from the following expression:

\[
\bar{\sigma}_{W\&n} = \frac{1}{h} \int_0^h \sigma(T) dT, \quad (3a)
\]

which, at the equator becomes:

\[
\bar{\sigma}_{W\&n} = \frac{2PR_o}{h[(R_o + h)^n - R_o^n]} \left[ \frac{(R_o + h)^n - R_o^n}{n - 1} \right] + \frac{(R_o + h)^n}{(R_o + h)^n - R_o^n} \left( \frac{2a^4 - b^4}{2(a^4 + a^2b^2 + b^4)} \right), \quad (3b)
\]

where all variables are as previously defined.

Mean stresses as calculated from the two models were then compared in terms of percent error, thick-wall stress being taken as the standard of reference:

\[
\text{percent error} = \frac{\sigma_{W\&n} - \sigma_{W\&S}}{\sigma_{W\&S}} \times 100.
\]

All calculations were performed by an IBM 1130 computer using a program similar to one previously reported (16) but modified to incorporate the thick-wall equations. The computer print-out included the values of the semiaxes, \( a \) and \( b \), and the ratio of \( h/b \) for each stress determination.

Results

1. MEAN CIRCUMFERENTIAL STRESS

Selected mean stress and dimensional data from all subjects are summarized in Table 1. Mean stress curves throughout the cardiac cycle from three representative subjects are plotted in Figure 1.

The time course of mean stress as calculated by the two models was nearly identical, peak stress occurring early in systole simultaneously with or just prior to peak ventricular pressure, and then declining relatively rapidly. Figure 1 illustrates, however, that the Sandler and Dodge formula consistently overestimated mean stress as determined from the Wong and Rautaharju model. The degree of overestimation in terms of percent error usually varied between 5% and 15% in individual patients and overall averaged about 10% (Table 1).

End-diastolic volume per se appeared to affect the percent error very little. Thus, the discrepancy between thin-wall and thick-wall calculations among patients with compensated volume overload differed only slightly from that in normal subjects despite an average end-diastolic volume in the former group of more than one and one-half times normal. Pressure exerted no influence on the percent error since it is incorporated into each mean stress equation in identical fashion and cancels out in the calculation of percent error.

Within each group, the percent error was surprisingly consistent throughout the cardiac cycle. This was so in some cases despite large increases in the ratio between wall thickness and minor chamber semiaxis \( h/b \) as systole progressed. Thus, in normal subjects there was no significant increase in percent error from end-diastole to end-systole although \( h/b \)
Thin-Wall versus Thick-Wall Stress Analysis

<table>
<thead>
<tr>
<th>Group</th>
<th>No.</th>
<th>h/b</th>
<th>a/b</th>
<th>$\sigma_{wR}$</th>
<th>$\sigma_{wD}$</th>
<th>% error</th>
<th>h/b</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal left ventricle</td>
<td>6</td>
<td>0.35 ± 0.03</td>
<td>2.1 ± 0.1</td>
<td>32 ± 6</td>
<td>29 ± 5</td>
<td>7.2 ± 0.8</td>
<td>0.44</td>
</tr>
<tr>
<td>(EDV = 140 ± 11)</td>
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<td></td>
<td></td>
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<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mitral stenosis</td>
<td>9</td>
<td>0.30 ± 0.02</td>
<td>1.9 ± 0.1</td>
<td>42 ± 7</td>
<td>39 ± 6</td>
<td>7.4 ± 0.4</td>
<td>0.38</td>
</tr>
<tr>
<td>(EDV = 121 ± 8)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Volume overload, compensated</td>
<td>19</td>
<td>0.31 ± 0.01</td>
<td>1.8 ± 0.03</td>
<td>53 ± 0.4</td>
<td>48 ± 0.5</td>
<td>9.3 ± 0.5</td>
<td>0.43</td>
</tr>
<tr>
<td>(EDV = 239 ± 22)</td>
<td></td>
<td></td>
<td></td>
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<td></td>
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</tr>
<tr>
<td>Pressure overload, compensated</td>
<td>5</td>
<td>0.43 ± 0.07</td>
<td>2.0 ± 0.1</td>
<td>33 ± 3</td>
<td>31 ± 3</td>
<td>8.0 ± 1.0</td>
<td>0.53</td>
</tr>
<tr>
<td>(EDV = 123 ± 20)</td>
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<tr>
<td>Pressure or volume</td>
<td></td>
<td></td>
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<td></td>
<td></td>
</tr>
<tr>
<td>overload, decompensated, and</td>
<td></td>
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<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>primary myocardial disease</td>
<td>9</td>
<td>0.35 ± 0.03</td>
<td>1.7 ± 0.04</td>
<td>74 ± 0.15</td>
<td>67 ± 0.14</td>
<td>11.2 ± 0.7</td>
<td>0.38</td>
</tr>
<tr>
<td>(EDV = 314 ± 51)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Idiopathic myocardial</td>
<td>2</td>
<td>0.82 ± 0.24</td>
<td>2.0 ± 0.2</td>
<td>43 ± 0.16</td>
<td>39 ± 0.17</td>
<td>14.0 ± 1.0</td>
<td>1.05</td>
</tr>
<tr>
<td>hypertrophy</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>(EDV = 161 ± 41)</td>
<td></td>
<td></td>
<td></td>
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<td></td>
<td></td>
</tr>
</tbody>
</table>

Group means ± 1 se are shown. For purposes of this comparison, End-diastole includes data at or near end-diastole, and Peak systole includes data at or near the time of peak stress. End-systole was chosen arbitrarily as the point in the cardiac cycle when h/b was maximal.

For a table giving complete data, order document NAPS-00368 from ASIS National Auxiliary Publications Service, c/o CCM Information Sciences, Inc., 22 West 34th Street, New York, New York 10001, remitting $1.00 for microfiche or $3.00 for photocopy.

approximately tripled. Similar results were seen in other groups. That an increase in percent error is to be expected on the basis of changes in h/b alone is shown in Figure 2 (circles). The computer was used to simulate effects of varying h while volume was held constant at 89 ml, and a/b was held constant at 2/1. It can be seen in Figure 2 that percent error varies almost linearly with h/b, i.e., the error increases as wall thickness increases provided geometry is constant.

Examination of the dimensional data in the table discloses, however, that constant geometry in the contracting ventricle is exceptional. Rather, the ventricle usually shortens in its major axis less than in its minor axis; hence, a/b increases. The results of changing geometry were simulated by computer and are also illustrated in Figure 2 (squares). In this analysis, h was held constant at 7.5 mm and a was held constant at 4.5 cm while b was varied. It can be seen that the effects on percent error of varying b alone were opposite to those of varying h alone. It appears, then, that the influence of changes in wall thickness during contraction on percent error is usually mitigated by concomitant changes in chamber geometry.

2. DISTRIBUTION OF CIRCUMFERENTIAL STRESS

In their report, Wong and Rautaharju (10) used computer simulation to illustrate stress distribution in "hypertrophied" and "dilated"
WALL STRESS CALCULATIONS

### Peak systole

<table>
<thead>
<tr>
<th>a/b</th>
<th>$\sigma_{h,k}$</th>
<th>$\sigma_{w,k}$</th>
<th>% error</th>
</tr>
</thead>
<tbody>
<tr>
<td>2.1</td>
<td>3.26</td>
<td>3.04</td>
<td>7.0</td>
</tr>
<tr>
<td>± 0.1</td>
<td>± 0.24</td>
<td>± 0.24</td>
<td>± 0.8</td>
</tr>
<tr>
<td>2.0</td>
<td>3.59</td>
<td>3.33</td>
<td>8.0</td>
</tr>
<tr>
<td>± 0.1</td>
<td>± 0.29</td>
<td>± 0.29</td>
<td>± 0.9</td>
</tr>
<tr>
<td>2.2</td>
<td>3.93</td>
<td>3.65</td>
<td>7.6</td>
</tr>
<tr>
<td>± 0.1</td>
<td>± 0.43</td>
<td>± 0.43</td>
<td>± 0.7</td>
</tr>
</tbody>
</table>

### End-systole

<table>
<thead>
<tr>
<th>h/b</th>
<th>a/b</th>
<th>$\sigma_{h,k}$</th>
<th>$\sigma_{w,k}$</th>
<th>% error</th>
</tr>
</thead>
<tbody>
<tr>
<td>1.7</td>
<td>3.86</td>
<td>3.47</td>
<td>11.6</td>
<td>9.5</td>
</tr>
<tr>
<td>± 0.04</td>
<td>± 0.15</td>
<td>± 0.15</td>
<td>± 0.8</td>
<td>± 0.5</td>
</tr>
<tr>
<td>2.5</td>
<td>1.71</td>
<td>1.55</td>
<td>11.5</td>
<td>9.0</td>
</tr>
<tr>
<td>± 0.6</td>
<td>± 0.26</td>
<td>± 0.26</td>
<td>± 7.0</td>
<td>± 0.39</td>
</tr>
</tbody>
</table>

hearts, whereas in the present investigation stress distribution was determined from actual data from patients. Figure 3 illustrates the distribution of circumferential stress across the ventricular wall at the equator in five representative patients. For ease in comparing patterns of stress distribution among different physiologic states, stress is plotted as

![Time course of circumferential wall stress in three representative subjects. Closed circles identify calculations using the thin-wall equation of Sandler and Dodge. Open circles identify calculations based on the thick-walled model of Wong and Rautaharju (see text). EDV = end-diastolic volume.](http://circres.ahajournals.org/)

*Figure 1*

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Discussion

As discussed by Wong and Rautaharju (10), the ventricular wall is subjected to three different stresses: radial stress, acting perpendicular to the endocardial surface, longitudinal stress, and circumferential stress, acting within the wall at right angles to each other. In the present study we were interested in comparing values for circumferential stress only, since it alone of the three has been applied clinically. Circumferential stress has been shown to be an important determinant of myocardial oxygen consumption (17). Peak systolic circumferential stress has been used as the force factor in applications of the Hill force-velocity concept in intact man (18). Additionally, peak systolic circumferential stress has been shown to reflect the “appropriateness” of wall hypertrophy in chronic heart disease (11).

All these studies employed thin-walled models in quantifying mean circumferential stress. The validity of applying thin-wall theory to the relatively thick-walled left ventricle was questioned (19), but the issue could not be resolved until the nonlinear distribution of stress across an ellipsoidal shell had been defined. Although the Wong and Rautaharju analysis is more sophisticated than that of Sandier and Dodge, it should be emphasized that the same basic assumptions have been made in both models. In both, stress is considered in its passive sense, and the assumption is made that heart muscle is an isotropic and homogeneous material. Consequently the factor of fiber orientation is disregarded. In both models, other wall forces such as wall shear and bending moments are neglected. Even had these factors of shear and bending movements been included in the models, the measurements necessary to compute them cannot be made in man.

On the basis of data from thick-walled cylinders and spheres, Sandier and Dodge (9) estimated that, for the range of $h/b$ encountered in their study at end-diastole, the maximal error introduced by using thin-wall theory would be between 15% and 20% and for most subjects closer to 10%. They anticipated a...
progressively greater error, however, as wall thickness increased during systole. In the present comparison of calculations at end-diastole, the thin-wall equation overestimated mean thick-wall stress by more than 15% in only one patient, and in most cases agreement was within 10%. Furthermore, even at end-systole, when $h/b$ had its maximum value, the percent error between calculations generally was also around 10%. Analysis of the role played by factors other than $h/b$ indicates that changes in geometry of the ventricle during contraction, namely, proportionately more narrowing than shortening of the ventricular cavity, tend to offset effects of increases in wall thickness.

The present investigation, then, offers an estimate of the error introduced by employing the Sandler and Dodge or similar thin-walled ellipsoidal models for the computation of mean circumferential stress. It suggests that the Sandler and Dodge derivation is more accurate than the authors themselves suspected. The Sandler and Dodge formula might still find usefulness in the event that computer facilities are not available, since it is less arduous to calculate than the Wong and Rautaharju equation. The Sandler and Dodge equation, however, permits calculation only of

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Distribution of circumferential wall stress as computed from the Wong and Rautaharju model in representative patients. For comparison, stress is plotted on the ordinate in relative units, i.e., as percent of endocardial (maximum) stress, against wall thickness on the abscissa expressed as percent thickness from endocardium to epicardium. Open triangles indicate mean stress calculated from the thick-walled model, $h/b = \text{ratio of wall thickness to minor chamber semiaxis}$. Note scale difference of ordinate in C.
mean stress and only at the equator. The Wong and Rautaharju analysis permits calculation of stress at any depth within the wall. It also theoretically allows quantitation of stress at any point on the ventricle, but difficulties in measuring wall thickness from angiograms in human subjects will, in most cases, limit stress determination to the region of the equator.

Acknowledgment

The authors are grateful to Mrs. Linda Cleveland for the computer programming.

References


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Circ Res. 1969;24:575-582
doi: 10.1161/01.RES.24.4.575

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

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