Acknowledgment

We gratefully acknowledge the assistance of Anne D. Sevin, Ph.D., for the statistical analyses of the experimental data.

References
2. Parmley WW, Chuck L, Sonnenblick EH: Relation of $V_{\text{max}}$ to different models of cardiac muscle. Circ Res 30: 34, 1972

A Mathematical Model of the Dynamic Geometry of the Intact Left Ventricle and Its Application to Clinical Data

JEAN G. DUMESNIL, M.D., F.R.C.P. (C), RACHAD M. SHOUCRI, PH.D., JEAN-Louis LAURENCEAU, M.D., PH.D., AND JULES TURCOT, B.Sc.

SUMMARY In this paper we examine the relations that may exist between the geometric variables most frequently used to describe left ventricular contraction. The left ventricle is represented by a thick-walled cylinder contracting both radially and longitudinally. For this model, wall thickening, mid-wall radius shortening and longitudinal axis shortening can be shown to be uniquely related during contraction, whereas it can be demonstrated that internal radius shortening is not uniquely related to these variables, but is also determined by the specific geometry of the cylinder, expressed in terms of the mid-wall radius-to-wall thickness (R/h) ratio of the cylinder. Detailed analysis of the same variables in 44 normal subjects, 32 patients with aortic stenosis and 54 patients with valvular regurgitation (33 aortic and 21 mitral), strongly suggests that the same relations are also clinically applicable. For instance, ventricular longitudinal axis shortening can be estimated with some accuracy from the standard M-mode echocardiogram. Also, wall thickening can be viewed as the direct reflection of the shortening that occurs in the circumferential and longitudinal directions, whereas internal radius shortening is significantly influenced by the R/h ratio of the ventricle, a consideration which becomes important when analyzing results in patients with left ventricular hypertrophy.

MYOCARDIAL PERFORMANCE in the intact left ventricle has been evaluated in terms of extent and velocity of shortening with respect to the meridional and equatorial dimensions of the ventricle. More recently, the extent and rate of left ventricular wall thickening during systole have also been shown to be sensitive parameters of changes in ventricular function.1-8 Gould et al.7 particularly underlined the importance of wall thickening as a component of ventricular contraction, and attempted to describe the separate contributions of longitudinal shortening, circumferential shortening and wall thickening in terms of left ventricular power. There remain, however, many questions about the precise relationship between the directional components of contraction and myocardial fiber shortening. Recent investigators have, for instance, reported that myocardial wall thickening in aortic stenosis could be decreased despite normal circumferential shortening.4

In this paper, we analyze in detail the geometrical changes occurring during left ventricular contraction in patients with pressure or volume overload of the left ventricle. We used a geometrical model of the ventricle to express the relations that may exist between the various variables used to describe the dimensional changes occurring within the ventricle during contraction.

Methods

Theoretical Considerations

These considerations are aimed at deriving the geometrical relations between wall thickening, internal radius shortening, mid-wall radius shortening and...
longitudinal axis shortening during systole in the left ventricle. For this purpose, a geometrical model of the ventricle is given in appendices A, B and C. This model basically shows that in a thick-walled, three-dimensional cylinder contracting both radially and longitudinally (fig. 1) there is a unique relation between wall thickening, mid-wall radius shortening and longitudinal axis shortening (appendix A); internal radius shortening, however, is not uniquely determined by these variables, but is also influenced by the specific geometry of the cylinder (appendix B). The relation between the relative changes in wall thickness \( h \), mid-wall radius \( R \) and length \( L \) during contraction in a thick-walled cylinder can be expressed as follows:

\[
\left( 1 + \frac{\Delta R}{R} \right) \left( 1 + \frac{\Delta h}{h} \right) \left( 1 + \frac{\Delta L}{L} \right) = 1
\]  

(1)

Since \( \frac{\Delta R}{R} \) is negative during contraction, \( \frac{\Delta L}{L} \) negative and \( \frac{\Delta h}{h} \) positive, equation (1) can be written in the following form:

\[
- \left| \frac{\Delta L}{L} \right| = \frac{1}{\left( 1 - \frac{\Delta R}{R} \right) \left( 1 + \frac{\Delta h}{h} \right)} - 1
\]  

(2)

This last equation allows for the calculation of the variation in the length of the cylinder from the knowledge of mid-wall radius shortening and wall thickening and is theoretically applicable to data obtained by echocardiography.

Figures 2 and 3, derived from appendix B, illustrate the relationship between relative internal radius shortening \( \Delta a/a \), relative mid-wall radius shortening \( \Delta R/R \) and relative longitudinal axis shortening \( \Delta L/L \) in the cylinder during contraction. This relationship is influenced by the mid-wall radius-to-
excluded patients with clinical or angiographic evidence suggestive of coronary artery disease, except for a few patients with aortic stenosis and clinical evidence of angina who had normal coronary arteriograms. Normal values for echocardiographic measurements were obtained in a group of 44 healthy adult subjects with no evidence of heart disease on clinical history, physical examinations or ECG.

ECGs

We recorded standard M-mode echocardiograms with an SKI Ekoline 20A echocardiograph interfaced with a Honeywell 1856 stripchart recorder and used a 2.25 MHz transducer, 1.25 cm in diameter, focused at 7.5 or 10 cm. We performed all examinations with the patient in the supine position, with the transducer held in the third or fourth intercostal space. After performing repeated longitudinal and transversal scans of the left ventricle, high speed (≥50 mm/sec) recordings were performed just below the mitral valve, where the interventricular septum and left ventricular posterior wall are best seen with some parts of both mitral leaflets or chordae. Such recordings are routinely used for echocardiographic studies of left ventricular function, and left ventricular dimension in this position is thought to approximate the short axis of the left ventricle. During each recording, we took particular care to vary gain controls in order to obtain tracings which allowed accurate measurements of left ventricular internal dimensions as well as thicknesses of the interventricular septum and the posterior wall.

The dimension of the left ventricle was measured both in diastole and systole as the distance between the left side of the interventricular septum and the anterior border of the posterior free wall. The left ventricular internal dimension in diastole (LVIDd) was measured at the peak of the R wave on the simultaneously recorded ECG, and the left ventricular internal dimension in systole (LVIDs) was measured at the point where the posterior wall endocardium achieves its most anterior direction (fig. 4). Since echocardiographic left ventricular dimensions theoretically represent left ventricular internal diameters in the short axis, the value for fractional shortening in left ventricular diameter can also be considered to approximate the relative change of internal radius (Δa/a) of the ventricle in the short axis:

$$|\Delta a/a| = \frac{LVIDd - LVIDs}{2}$$

We calculated the endocardial mean velocity of circumferential fiber shortening (Vcf endo) using the method of Cooper et al.10

$$Vcf\ endo = \frac{LVIDd - LVIDs}{LVIDd \times (\Delta t - 0.05)}$$

where Δt is the time elapsed between the peak of the R
wave on the ECG and the point where LVIDs is measured.

From the same recordings, we measured the thickness of the interventricular septum so as to include both the right and left sides of the septum, and posterior wall thickness as the distance between the posterior border of the epicardial echo to the top of the endocardial echo (fig. 4). We measured diastolic thickness (TwD) at the peak of the R wave on the ECG and systolic thickness (TwS) at the point where the thickness appears to be maximal during systole. We used the values for diastolic thickness to calculate the septal-to-posterior wall thickness ratio and estimated left ventricular mass index using the method of Troy et al. \textsuperscript{11} Fractional thickening (Δh/h) of the posterior wall was computed using the formula

$$Δh/h = \frac{TwS - TwD}{TwD}$$

The normalized mean velocity of thickening (VTWC) of the posterior wall was calculated as follows:

$$VTWC = \frac{TwS - TwD}{TwD \times Δt}$$

where Δt is the thickening interval or the time elapsed between the peak of the R wave on the ECG and the point where systolic thickness is measured.

Using the values for left ventricular dimension and posterior wall thickness, we calculated the fractional change in mid-wall radius (ΔR/R) and the mean velocity of circumferential shortening at mid-wall (Vcf mid-wall) using the following formulas:

$$|ΔR/R| = \frac{|LVIDd + TwD|}{2}$$

$$Vcf \text { mid-wall} = \frac{|ΔR/R|}{R} \times \frac{1}{(Δt - 0.05)}$$

where Δt is the time having elapsed between the points where LVIDd and LVIDs are measured. From the concepts derived in appendix A, we also calculated values for Δh/(h + Δh) and ΔL/L. We computed Δh/(h + Δh) as the change in thickness as related to systolic thickness

$$\frac{Δh}{h + Δh} = \frac{TwS - TwD}{TwS}$$

whereas values for ventricular longitudinal axis shortening (|ΔL/L|) were calculated from the values for |ΔR/R| and Δh/h, using equation (2) of the theoretical considerations.

**Angiograms**

In 24 patients with good quality angiograms, we compared the echocardiographic estimates of ventricular longitudinal axis shortening (|ΔL/L|) with direct measurements performed on the angiograms. The echocardiograms were performed within a week before or after the angiographic procedure; no important change in clinical status occurred. Of the original group of patients studied by echocardiography, 11 patients with aortic stenosis, five patients with aortic regurgitation and two patients with mitral regurgitation had angiograms suitable for analysis. In order to obtain normal values, we also included six subjects who had undergone cardiac catheterization for the evaluation of chest pain, and whose left ventricular angiogram and coronary arteriogram were normal. All ventricular angiograms were performed in the 30° right anterior oblique projection and the measurements were done on regular non-postextrasystolic sinus beats by two observers unaware of the echocardiographic results. We used a simple linear regression equation to make statistical comparisons.

**Results**

**Left Ventricular Measurements**

The mean ± SD of the echocardiographic data for left ventricular measurements are given in table 1. Heart rates and body surface areas were comparable in all groups of subjects. Patients with aortic stenosis had normal left ventricular dimensions, but marked
increases in thickness both in the interventricular septum and the posterior wall, resulting in significant decreases of R/h ratio values. In patients with aortic or mitral regurgitation, increases in wall thicknesses were more moderate, but left ventricular internal dimensions were significantly increased. The net result was a moderate increase of the R/h ratio. Despite these differences in patterns of hypertrophy, the increases in left ventricular mass index were nevertheless comparable in the three groups of patients with valvular disease. Finally, we observed no significant differences in septal-to-posterior wall thickness ratios.

**Left Ventricular Function**

Table 2 summarizes the results for the various parameters of ventricular function calculated from the echocardiograms. Compared with normals, the patients with aortic stenosis had increased internal radius fractional shortening and endocardial mean Vcf, normal mid-wall shortening and mid-wall mean Vcf and decreased values for posterior wall thickening. Referring to the mathematical model, the increase in internal radius fractional shortening is most readily explained by the marked decrease in R/h ratio in these patients. However, the decrease in wall thickening associated with normal values for mid-wall radius shortening would be consistent with a decrease in ventricular longitudinal axis shortening (see equation (2)). The mean value for echocardiographic estimates of ventricular longitudinal axis shortening in these patients was 0.09 ± 0.08 vs 0.21 ± 0.09 in normals (p < 0.01).

Most results in patients with aortic or mitral regurgitation were comparable to results in normal subjects. Patients with aortic regurgitation merely had a moderate increase in mid-wall mean Vcf and a moderate decrease in estimated longitudinal axis shortening. This last result can be understood best by considering the slight decrease in wall thickening as opposed to a slight increase in mid-wall radius shortening. Patients with mitral regurgitation had moderate increases in posterior wall thickening values. This finding can be interpreted as the result of slight increases both in mid-wall radius and longitudinal axis shortening. The mathematical model can also be used to explain the small variations in values for internal radius shortening. (See figs. 2 and 3 and equation (B4) in appendix B for the complete relation between internal radius shortening, mid-wall radius shortening, longitudinal axis shortening and R/h ratio.)

**Angiographic Correlation**

The comparison between echocardiographic estimates of ventricular longitudinal axis shortening and direct angiographic measurements is provided in figure 5. There is a good correlation (r = 0.84), with most values around the identity line. Individual variations may be because the echogram and the angiogram were not performed at the same time. For the 24 subjects, ventricular longitudinal axis shortening averaged 0.17 ± 0.08 using echocardiography vs 0.17 ± 0.07 using angiography. The mean echocardiographic value for the 11 patients with aortic stenosis was 0.12 ± 0.08, and the angiographic value was 0.13 ± 0.08. In the normal subjects, ecocar-
TABLE 2. Echocardiographic Indices of Left Ventricular Function

<table>
<thead>
<tr>
<th></th>
<th>Normals (n = 44)</th>
<th>Aortic stenosis (n = 32)</th>
<th>Aortic regurgitation (n = 34)</th>
<th>Mitral regurgitation (n = 21)</th>
</tr>
</thead>
<tbody>
<tr>
<td>LV internal radius</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>fractional shortening</td>
<td>0.36 ± 0.05</td>
<td>0.42 ± 0.10</td>
<td>0.35 ± 0.08</td>
<td>0.39 ± 0.08</td>
</tr>
<tr>
<td></td>
<td>p &lt; 0.01</td>
<td>NS</td>
<td>NS</td>
<td>NS</td>
</tr>
<tr>
<td>Endocardial mean Vcf</td>
<td>1.18 ± 0.24</td>
<td>1.37 ± 0.45</td>
<td>1.28 ± 0.53</td>
<td>1.28 ± 0.37</td>
</tr>
<tr>
<td>(circ/sec)</td>
<td></td>
<td>p &lt; 0.05</td>
<td>NS</td>
<td>NS</td>
</tr>
<tr>
<td>LV mid-wall radius</td>
<td>0.21 ± 0.04</td>
<td>0.23 ± 0.06</td>
<td>0.23 ± 0.06</td>
<td>0.23 ± 0.05</td>
</tr>
<tr>
<td>fractional shortening</td>
<td>NS</td>
<td>NS</td>
<td>NS</td>
<td>NS</td>
</tr>
<tr>
<td>Mid-wall mean Vcf</td>
<td>0.69 ± 0.19</td>
<td>0.75 ± 0.24</td>
<td>0.83 ± 0.31</td>
<td>0.77 ± 0.21</td>
</tr>
<tr>
<td>(circ/sec)</td>
<td></td>
<td>NS</td>
<td>p &lt; 0.05</td>
<td>NS</td>
</tr>
<tr>
<td>PW fractional thickening</td>
<td>0.62 ± 0.16</td>
<td>0.45 ± 0.13</td>
<td>0.57 ± 0.19</td>
<td>0.75 ± 0.22</td>
</tr>
<tr>
<td></td>
<td>p &lt; 0.01</td>
<td>NS</td>
<td>NS</td>
<td>p &lt; 0.05</td>
</tr>
<tr>
<td>PW thickening as a fraction of systolic thickness</td>
<td>0.38 ± 0.05</td>
<td>0.30 ± 0.06</td>
<td>0.36 ± 0.08</td>
<td>0.42 ± 0.07</td>
</tr>
<tr>
<td></td>
<td>p &lt; 0.01</td>
<td>NS</td>
<td>NS</td>
<td>p &lt; 0.05</td>
</tr>
<tr>
<td>PW corrected mean thickening velocity (uTw/sec)</td>
<td>1.75 ± 0.44</td>
<td>1.25 ± 0.44</td>
<td>1.73 ± 0.78</td>
<td>2.13 ± 0.74</td>
</tr>
<tr>
<td></td>
<td>p &lt; 0.01</td>
<td>NS</td>
<td>NS</td>
<td>p &lt; 0.05</td>
</tr>
<tr>
<td>LV longitudinal axis</td>
<td>0.21 ± 0.09</td>
<td>0.09 ± 0.08</td>
<td>0.16 ± 0.10</td>
<td>0.24 ± 0.08</td>
</tr>
<tr>
<td>shortening</td>
<td>p &lt; 0.01</td>
<td>p &lt; 0.05</td>
<td>NS</td>
<td>NS</td>
</tr>
</tbody>
</table>

Values are mean ± sd. Level of significance is given for each group compared with normal subjects by the t test.
Abbreviations: LV = left ventricle; PW = posterior wall; uTw/sec = units of wall thickening per second; Vcf = velocity of circumferential fiber shortening.

![Comparison of echocardiographic estimates and direct angiographic measurements of relative ventricular longitudinal axis shortening (%ΔL/L) in 24 patients.](image)

**FIGURE 5.** Comparison of echocardiographic estimates and direct angiographic measurements of relative ventricular longitudinal axis shortening (%ΔL/L) in 24 patients. • = aortic stenosis; ○ = aortic regurgitation; □ = mitral regurgitation; x = normal ventriculograms and coronary arteriograms. Regression equation is given in inset. Dotted line represents identity line. We obtained echocardiographic estimates using equation 2.

diographic values averaged 0.20 ± 0.05 vs a mean of 0.18 ± 0.02 for the angiographic measurements.

**Discussion**

The primary purpose of this study is to establish more precisely the relations that may exist between the geometrical variables most frequently used to describe ventricular contraction. We based the theoretical considerations on a cylindrical model of the ventricle, chosen primarily because of its mathematical simplicity. The basic analogies between this model and the contracting ventricle are as follows: 1) The volume and mass of the ventricular wall are assumed constant during contraction; 2) during ventricular contraction, both the left ventricular cavity and the left ventricular wall assume a new shape; 3) the dimensional changes during ventricular contraction can be described in terms of similar changes in a cylinder contracting radially and longitudinally. Although the cylindrical model may appear to oversimplify the geometrical changes occurring during contraction, it can be most helpful in validating certain basic concepts, as has been done in previous studies.7,9 As shown in the appendices, the basic concept of a unique relation between the geometric variables used to describe ventricular contraction (appendix A,
equation (A5) can also be discussed for a spherical or an ellipsoidal model (appendix C, equation (C9)), but the formulation for the cylindrical model, in addition to being simpler, seems to apply to all the situations studied (appendix C). We used equation (2) to predict ventricular longitudinal axis shortening from the echocardiographic measurements of mid-wall radius shortening and posterior wall thickening, and there was a good correlation between these results and the direct measurements performed on angiograms. In a recent study (see table 2 of Gould et al.), the same variables as in equation (A9) were directly measured from angiograms in nine different groups of subjects, but no assumption about a specific relationship between the variables was made. In each of the nine groups studied, the average value for the normalized peak thickening velocity could be predicted within an error of 1–7% (mean 4.8%) simply by adding the average value for normalized peak mid-wall radius shortening velocity to the average value for normalized peak longitudinal axis shortening velocity as in equation (A9). Although the individual data of the latter study are not available, the results of both studies strongly suggest that the relations given by the cylindrical model are in fact clinically applicable.

The relation between wall thickening, mid-wall radius shortening and longitudinal axis shortening derived from the mathematical model may also give some insight into the interrelation between these variables and myocardial shortening. As mentioned by Gould et al., wall thickening, circumferential shortening and longitudinal shortening can all be viewed as contributing directly to the ejection of blood from the ventricle. The findings of this study, however, suggest that the wall thickening component can be viewed as the direct result as well as the reflection of the shortening in the circumferential and longitudinal directions. Although there are three mechanical components for the ejection of blood from the ventricle, myocardial shortening per se can be viewed as giving rise to active shortening mainly in the circumferential and longitudinal directions (fig. 6). Previously, wall thickening had often been considered only in terms of the circumferential shortening. Physiologically, this concept agrees with the fact that fiber orientation within the ventricular wall is either circumferential, longitudinal or oblique, but always perpendicular to the left ventricular cavity. During contraction, the oblique fibers probably contribute directly to the circumferential and longitudinal shortening of the ventricle, whereas wall thickening is merely a consequence of the shortening in these directions. The relevance of these considerations with regard to the changes in wall stress is not considered in the present study.

Another basic concept which can be derived from our results is that internal radius shortening cannot be uniquely described in terms of circumferential and longitudinal shortening, but is also related to the specific R/h ratio of the ventricle (appendix B). Although this finding may be less important when comparing ventricles with similar geometry, it may become relevant when studying ventricles with variable R/h ratio values, as may occur in patients with left ventricular hypertrophy. In our patients with aortic stenosis, the average value for internal radius shortening was 16% higher than in the normals, a statistically significant difference, whereas values for mid-wall radius shortening in the two groups did not differ significantly. In fact, if the values for mid-wall radius shortening and longitudinal axis shortening had been identical to normal in aortic stenosis, the difference in values between the two groups for internal radius shortening could theoretically (see equation B4) have been as much as 22%. The significance of this observation is shown in figure 3, where for the same values of circumferential shortening ($\Delta R/R$) and longitudinal shortening ($\Delta L/L$), the value of internal radius shortening ($\Delta a/a$) depends on the geometry of the ventricle expressed by the ratio R/h. Thus, significant variations in internal radius shortening may occur solely on the basis of a difference in ventricular geometry (different R/h ratios). Since the internal radius shortening ($\Delta a/a$) is often used to characterize ventricular function, this observation may have important clinical implications.

The echocardiographic measurements in the normal subjects are consistent with those previously reported by other investigators, and when a standardized recording technique is used, such measurements have been shown to be both reliable and reproducible. Since patients with coronary artery disease frequently have regional ventricular dysfunctions and the problems involved may be different, we excluded such patients from the study. However, three groups of patients with coronary artery disease are included in the work of Gould et al., and their average values also seem to be consistent with the relation given by the mathematical model. In our patients, angiographic

Myocardial Fiber Shortening

Circumferential

Longitudinal

Mechanical Power

Circumferential

Thickening

Longitudinal

Ejection of Blood

Figure 6. Conceptual representation of myocardial fiber shortening and mechanical power in the intact left ventricle during contraction.
measurements were performed whenever possible and they appeared to validate both the echocardiographic measurements and the basic concepts given by the mathematical model.

Our range of values for ventricular longitudinal axis shortening is also consistent with that observed in man by other investigators. In the study by Gault et al.,1,8 this variable averaged 18.6% in the subjects without left ventricular disease compared with 7.4% in the patients with left ventricular disease; Lewis and Sandler16 reported values ranging from 7–31% (mean 13%) in 24 patients with various forms of heart disease. To our knowledge, however, the fact that ventricular longitudinal axis shortening may be selectively decreased in patients with aortic stenosis, as we found in this study, has never been previously observed and there is no clear explanation for this phenomenon. Since subendocardial myocardial fibers are mostly oriented longitudinally,18 one might hypothesize that the decrease in longitudinal axis shortening is a consequence of the subendocardial abnormalities that have been observed in patients with aortic disease.17 Whether measurements of ventricular longitudinal axis shortening in these patients may become useful in estimating the extent of such abnormalities as well as the severity of the disease remains to be determined. Nevertheless, the findings demonstrate that physiological factors influencing the dynamic change of the ventricle do not necessarily exert their influence in an orderly way among longitudinal shortening, circumferential shortening and wall thickening. In this sense the noninvasive evaluation of longitudinal shortening from the M-mode echocardiogram may reveal interesting new clinical information.

Our findings require further confirmation. Nevertheless, the mathematical model we present provides an integrated approach for viewing the relations between some geometric variables routinely used to describe ventricular contraction and our results as well as those of others1 strongly suggest that the concepts derived from this model are clinically applicable. For example, a noninvasive evaluation of ventricular longitudinal axis shortening can be obtained with some accuracy from the standard M-mode echocardiogram. Moreover, the results demonstrate the influence of ventricular shape on the relations between left ventricular wall dynamics and the changes within the left ventricular cavity, which has important implications, particularly when evaluating left ventricular performance in patients with left ventricular hypertrophy.

References


Appendix A

Relation Between the Variables Describing the Relative Change of Ventricular Dimensions

The left ventricle is represented by a cylinder which is assumed to contract both radially and longitudinally. The length (L) is thus allowed to change but the volume of the wall of the cylinder is assumed to remain constant (fig. 1).

The volume \( V_w \) of the wall of the cylinder is given by

\[
V_w = \pi (b^2 - a^2) \cdot L \quad (A1)
\]

or

\[
V_w = 2 \pi Rh \cdot L \quad (A2)
\]

where \( b - a = h \) represents the thickness, and \( b + a = 2R \) is equal to twice the mid-wall radius R. Since the volume \( V_w \) is assumed constant during ventricular motion, equation (A2) gives
In reference to figure 1, internal radius (a) may be written in the form:

$$a = R - \frac{h}{2}$$  \hspace{1cm} (B1)

and the change in internal radius can be expressed as:

$$\Delta a = \Delta R - \frac{\Delta h}{2}$$  \hspace{1cm} (B2)

Dividing (B2) by (B1) we get

$$\frac{\Delta a}{a} = \frac{\Delta R}{R - h/2} - \left(\frac{\Delta h}{2}\right) \left(\frac{1}{R - h/2}\right)$$  \hspace{1cm} (B3)

Substituting for $\Delta h/h$ from equation (A4), we obtain

$$\frac{\Delta a}{a} = \left(\frac{R/h}{R/h - \frac{1}{2}}\right) \left(\frac{\Delta R}{R}\right) - \left(\frac{1}{2}\right)$$

$$= \left(\frac{1}{R/h - \frac{1}{2}}\right) \left(\frac{\Delta R}{R}\right) - \left(\frac{1}{2}\right)$$  \hspace{1cm} (B4)

In this case, it can be seen that the values for $R/h$ ratio, $\Delta R/R$ and $\Delta L/L$ must all be considered in determining $\Delta a/a$. Graphical representation of the relation between $\Delta a/a$, $\Delta R/R$ and $\Delta L/L$ for a $R/h$ ratio value of 3.0 is given in figure 2. Three-dimensional representation of the relation between $\Delta a/a$, $\Delta R/R$ and $\Delta L/L$ for different $R/h$ values is given in figure 3. As demonstrated by figures 2 and 3 and equation (B4), this relation will be altered by a change in $R/h$ ratio value.

### Appendix C

#### Ellipsoidal and Spherical Models

In this section, we derive the expression for the relative variation of the longitudinal axis for an ellipsoidal model and a spherical model of uniform thickness, and show that the equation (A5) derived for a cylindrical model is, for all practical purposes, valid for the three cases.

The volume of an ellipsoid, with semi-axes, $R_c$, $R_b$, $R_a$ is given by

$$V = \frac{4}{3} \pi R_c R_b R_a$$  \hspace{1cm} (C1)

The volume of the wall of the cylinder is given by

$$V_w = \frac{4}{3} \pi R_{ce} R_{be} R_{ae} - \frac{4}{3} \pi R_{ci} R_{bi} R_{ai}$$  \hspace{1cm} (C2)

The suffix $e$ stands for the external radius and the suffix $i$ for the internal radius. We write:

$$R_{ce} = R_c + \frac{h}{2} \quad \quad R_{ci} = R_c - \frac{h}{2}$$
$$R_{be} = R_b + \frac{h}{2} \quad \quad R_{bi} = R_b - \frac{h}{2}$$
$$R_{ae} = R_a + \frac{h}{2} \quad \quad R_{ai} = R_a - \frac{h}{2}$$

where $h$ is the thickness of the ellipsoidal wall. Substitution of equation (C3) into equation (C2) gives easily
\[ V_\omega = \frac{4\pi}{3} R_c R_b h \left( 1 + \frac{R_a}{R_c} + \frac{R_b}{R_c} + \frac{h^2}{4 R_c R_b} \right) \]  
(C4)

If the two minor axes are equal, as is usually assumed in ventriculographic studies, one obtains

\[ V_\omega = \frac{4\pi}{3} R_c R_a h \left( 2 + \frac{R_a}{R_c} + \frac{h^2}{4 R_c R_a} \right) \]  
(C5)

In the case of a sphere \( R_c = R_a = R_b \), and equation (C4) becomes

\[ V_\omega = \frac{4\pi}{3} R^2 \left( 3 + \frac{h^2}{4 R^2} \right) \]  
(C6)

For the case of a sphere, it is easily seen that \( \frac{h^2}{4 R^2} \) can be neglected with respect to unity. By writing \( D = 2R \), equation (C6) becomes

\[ V_\omega \approx 2\pi R h D \]  
(C7)

which is similar to equation (A2).

For an ellipsoid, equation (C5) gives

\[ V_\omega \approx \frac{4\pi}{3} R_c R_a h \left( 2 + \frac{R_a}{R_c} \right) \]

\[ = \frac{4\pi}{3} R_a h (2 R_c + R_a) \]  
(C8)

where the quantity \( \frac{h^2}{4 R_c R_a} \) has been neglected. We write \( K = 2 R_c + R_a \). It is then easily shown, as in equation (A5), that for constant \( V_\omega \)

\[ \frac{\Delta K}{K} = \frac{1}{\left( 1 + \frac{\Delta R_a}{R_a} \right) \left( 1 + \Delta h / h \right)} - 1 \]  
(C9)

\[ \frac{\Delta (2 R_c)}{2 R_c + R_a} = \frac{1}{\left( 1 + \frac{\Delta R_a}{R_a} \right) \left( 1 + \Delta h / h \right)} - 1 - \frac{\Delta R_a}{2 R_c + R_a} \]  
(C10)

**Table C1. Comparison of Different Values of Longitudinal Axis Shortening for Different Geometrical Models in 24 Patients with Angiograms**

<table>
<thead>
<tr>
<th>( \Delta R_a / R_a ) from echograms</th>
<th>( \Delta L / L ) from echograms</th>
<th>( \Delta L / L ) from end-diastole</th>
<th>( \Delta L / L ) from angiograms at end-diastole</th>
<th>( \Delta L / L ) from angiograms at end-diastole</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal subjects</td>
<td>0.22</td>
<td>0.25</td>
<td>0.27</td>
<td>0.26</td>
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<td>Patients with mitral regurgitation</td>
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*Indicates lengthening rather than shortening during systole.

Abbreviations: \( \Delta R_a / R_a \) = mid-wall radius shortening; \( \Delta L / L \) = longitudinal axis shortening; \( L:D \) = ventricular length-to-diameter ratio.
If we start measuring our variation from end-diastole, then we can substitute in (C10) \( R_a = R_e = \frac{L}{2} \) if we suppose a 1:1 length/diameter (L:D) ratio. If we suppose L:D = 2:1 at end-diastole, then \( R_a = L/4 = R_e/2 \). For L:D = 1:1 at end-diastole, equation (C10) becomes

\[
\frac{2}{3} \frac{\Delta R_e}{R_e} = \frac{1}{1 + \frac{\Delta R_a}{R_a}} \left( 1 + \frac{\Delta h}{h} \right)
- 1 - \frac{1}{3} \frac{\Delta R_a}{R_a}
\]

\( (C11) \)

or

\[
\left( \frac{\Delta L}{L} \right)_\text{ell} = \frac{3}{2} \left( \frac{\Delta L}{L} \right)_\text{cyl} - \frac{1}{2} \frac{\Delta R_a}{R_a}
\]

\( (C12) \)

where \( \left( \frac{\Delta L}{L} \right)_\text{ell} \) is the change in length expected for an ellipsoidal model, and \( \left( \frac{\Delta L}{L} \right)_\text{cyl} \) is the change in length expected for a cylindrical model.

Similarly, for L:D = 2:1 at end-diastole, equation (C10) becomes

\[
\left( \frac{\Delta L}{L} \right)_\text{ell} = \frac{5}{4} \left( \frac{\Delta L}{L} \right)_\text{cyl} - \frac{1}{4} \frac{\Delta R_a}{R_a}
\]

\( (C13) \)

We reproduced in table (C1) the results of longitudinal relative variations as derived for a cylindrical model by using equation (A5), and for an ellipsoidal model by using equations (C12) and (C13). We note generally that the results obtained by using an ellipsoid with L:D = 2:1 are nearer to the cylindrical model than the results obtained using L:D = 1:1. For normal subjects, subjects with aortic regurgitation and subjects with mitral regurgitation, the difference between the three results is insignificant. In the case of aortic stenosis, we observed that the results obtained from angiography are closer to those obtained using a cylindrical model. It may well be that when there is concentric hypertrophy, the ventricle becomes less globular and assumes a shape closer to that of a cylinder. Finally, the equation (B4) showing the dependence of \( \Delta a/a \) on the R/h ratio is still valid for an ellipsoidal model by replacing \( \Delta L/L \) by \( \Delta K/K \) (see equations (A5) and (C9)).
A mathematical model of the dynamic geometry of the intact left ventricle and its application to clinical data.
J G Dumesnil, R M Shoucri, J L Laurenceau and J Turcot

Circulation. 1979;59:1024-1034
doi: 10.1161/01.CIR.59.5.1024

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