Validity of echocardiographic determination of left ventricular systolic wall thickening


ABSTRACT Previous direct measurements of left ventricular systolic wall thickening (SWT) in animal studies have yielded values approximately one-half those found echocardiographically in humans, suggesting a possible overestimation of SWT by echocardiographic techniques. To test the validity of echocardiographic SWT measurements, the relationship between echocardiographically determined end-diastolic and end-systolic left ventricular short-axis myocardial cross-sectional areas (ED Myo CSA and ES Myo CSA, respectively) was assessed in 18 normal subjects. Since Myo CSA is a function of wall thickness and wall circumference, overestimation of SWT by echocardiography would be expected to produce an overestimation of ES Myo CSA relative to ED Myo CSA. SWT, as determined by both M mode (52%) and two-dimensional echocardiography (48%), was consistent with previously reported echocardiographic values, but exceeded that reported in animal studies. By least squares linear regression analysis, ES Myo CSA was 1.078 × ED Myo CSA − 0.385 cm² (r = .947, SEE = 1.183 cm²) when assessed by one observer and was 1.042 × ED Myo CSA − 0.142 cm² (r = .906, SEE = 1.831 cm²) when assessed independently by another. The close relationship observed between echocardiographically determined ES Myo CSA and ED Myo CSA was consistent with constant left ventricular myocardial mass throughout the cardiac cycle and thus did not suggest an overestimation of SWT by echocardiographic techniques.


ECHOCARDIOGRAPHIC STUDIES of systolic wall thickening (SWT) of the free wall of the normal human left ventricle have generally yielded values ranging from 40% to 80%.1-4 In contrast, studies of SWT in animals in which various more direct techniques have been used have produced mean values ranging from 10% to 30%.5-8 The major explanation given for this discrepancy has been that echocardiographic measurements of SWT include the contribution of compressed trabeculae carnae to apparent end-systolic wall thickness, while the more direct measurement techniques used in animal studies do not.1,7

In as much as systolic trabecular compression displaces blood volume (albeit incompletely) and thus contributes to left ventricular stroke volume, it also contributes to effective SWT.2 SWT determinations are derived from wall thickness measurements. The accuracy of wall thickness measurements relies on the accuracy of epicardial and endocardial edge detection. The aim of our investigation was to determine whether echocardiographic techniques overestimate the systolic movement of the endocardial surface of the left ventricle relative to the epicardial surface, resulting in overestimation of SWT.

Assuming that myocardial density remains constant throughout the cardiac cycle, myocardial volume would be expected to remain constant in order to preserve constant myocardial mass. In fact, a negligible reduction of approximately 0.3% in left ventricular myocardial volume has been estimated to occur during systole due to a reduction in intramyocardial blood volume.9 Myocardial volume may be considered virtually constant, therefore, for the purposes of wall thickness determinations. Left ventricular myocardial volume may be calculated, with the use of Simpson’s rule, as the sum of serial myocardial cross-sectional areas (Myo CSAs) along the length of the left ventricle (Σ Myo CSAs) times the length of the interval separating consecutive cross-sections.4-10 In view of the relatively small change in left ventricular long-axis length during systole (5% to 10%),11-13 Simpson’s rule would predict only a small increase in Myo CSA from end-diastole to end-systole. For example, assuming an ellipsoid model for calculating left ventricular myocardial volume and 10% left ventricular long-axis systolic shortening, end-systolic Myo CSA (ES Myo CSA)

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would be approximately equal to $1.11 \times$ end-diastolic Myo CSA (ED Myo CSA). Assuming 5% long-axis shortening, ES Myo CSA would be approximately equal to $1.05 \times$ ED Myo CSA (Appendix 1).

Myo CSA is a function of wall thickness and wall circumference. An overestimation of wall thickness would thus result in an overestimation of Myo CSA. In particular, an overestimation of SWT (viz an overestimation of end-systolic wall thickness relative to end-diastolic wall thickness) would result in an overestimation of ES Myo CSA relative to ED Myo CSA.

In our investigation, the relationship between two-dimensional echocardiographic measurements of ED Myo CSA and ES Myo CSA was determined in human subjects as an indicator of the reliability of echocardiographic measurements of SWT.

**Methods**

**Subjects.** The study group consisted of 18 normal subjects. Eight subjects were volunteers and 10 subjects were referred for cardiac evaluation but were found to have no clinical, electrocardiographic, or echocardiographic evidence of heart disease. There were 13 male and five female subjects ranging in age from 14 to 54 (mean 28) years.

**Echocardiographic recordings.** Parasternal, short-axis two-dimensional echocardiograms of the left ventricle at the midpapillary muscle level were recorded in each of the 18 subjects with a Diasonics V-3400R Ultrasonograph equipped with a 2.25 MHz phased-array transducer. Recordings were obtained with the subjects in a semirecumbent, left lateral position. End-diastolic and end-systolic frames of the same cardiac cycle were automatically "frozen" at the peak of the R wave and terminus of the T wave, respectively, of the simultaneously recorded electrocardiogram, and recorded on a Sony ½ inch videotape recorder for subsequent analysis. M mode echocardiograms were recorded from the same site on the left ventricle in all 18 subjects by passing a M mode cursor through the two-dimensional echocardiographic image immediately before recording of the end-diastolic and end-systolic frames.

**Determination of Myo CSA.** The epicardial and endocardial cross-sectional areas of the left ventricle (Epi CSA and Endo CSA, respectively) were determined at end-diastole and end-systole by direct planimetry of the previously recorded frozen images on the ultrasonograph screen with the in-built planimetric function of the Diasonics V-3400R Ultrasonograph. The leading-edge method was used to trace the endocardial and epicardial outlines, as evaluated by Wyatt et al. The papillary muscles were excluded from planimetry of Endo CSA. Two independent observers planimetered each end-diastolic and end-systolic frame. The end-systolic frames were planimetrated separately from, and without knowledge of the planimetric results for, the end-diastolic frames. Further, the values obtained by planimetry of the epicardium or endocardium were not visible to the observer performing planimetry, but were recorded automatically on hard-copy prints of the selected frames for subsequent analysis. ED Myo CSA and ES Myo CSA were then calculated with the following formulas:

$$
ED\ Myo\ CSA = ED\ Epi\ CSA - ED\ Endo\ CSA \quad (1)
$$

$$
ES\ Myo\ CSA = ES\ Epi\ CSA - ES\ Endo\ CSA \quad (2)
$$

An example of one such computation is shown in figure 1.

**Measurement of SWT.** Posterior left ventricular wall thickness at end-diastole (Wd) and end-systole (Ws) was measured as the distance between the epicardial and endocardial outlines of the posterior wall traced for planimetry. Thus, for two-dimensional echocardiographic measurements of wall thickness the same endocardial edge detection was relied on as for determination of Myo CSA. Wd and Ws were also measured from the M mode echocardiographic recording of the posterior left ventricular wall at the same level of the ventricle to ensure that the two-dimensional echocardiographic measurements of SWT were comparable with M mode values (since most of the published data on echocardiographic SWT refer to M mode measurements).

SWT was then determined, for both the M mode and two-dimensional echocardiograms, with the formula:

$$
SWT = \frac{Ws - Wd}{Wd} \times 100% \quad (3)
$$

**Statistical analysis.** The relationship between ES Myo CSA and ED Myo CSA was assessed by two-variable least squares linear regression analysis of the data of both observers. In addition, the significance of differences between paired values of ES and ED Myo CSA was analyzed with the paired t test. Interobserver variability for the Myo CSA determinations was calculated by dividing the difference between paired observations by their mean and was expressed as a percentage. M mode and two-dimensional echocardiographic measurements of Wd and Ws were compared with the paired t test. All results are expressed as mean ± SD.

**Results**

The results in all 18 subjects studied are listed in table 1. The relationship between ES Myo CSA and ED Myo CSA, based on the data obtained by observer 1, is shown in figure 2. Not surprisingly, these two determinations showed a high linear correlation ($r = .947, p < .001$). Of more significance, however, was the fact that the slope of the equation for least squares linear regression of ES Myo CSA on ED Myo CSA.
TABLE 1
Data on left ventricular Myo CSA and wall thickness

<table>
<thead>
<tr>
<th>Subject No.</th>
<th>Sex</th>
<th>Age (yr)</th>
<th>Myo CSA per observer 1 (cm²)</th>
<th>Myo CSA per observer 2 (cm²)</th>
<th>Wd (mm)</th>
<th>Ws (mm)</th>
<th>SWT (%)</th>
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<td></td>
<td></td>
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<td>ES</td>
<td>ED</td>
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<td>F</td>
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<tr>
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<td>20.4</td>
<td>21.6</td>
<td>21.8</td>
<td>22.5</td>
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</table>

2D = two-dimensional echocardiogram; M = M mode echocardiogram.

approached unity and that the y intercept was close to zero, with ES Myo CSA = 1.078 × ED Myo CSA−0.385 cm² (SEE = 1.183 cm²). Nevertheless, ES Myo CSA (21.6 ± 3.7 cm²) exceeded ED Myo CSA (20.4 ± 3.2 cm²) in 14 of the 18 subjects and this discrepancy, although small, achieved statistical significance (p < .001).

Observer 2 obtained similar results (table 1). The interobserver variability for ED Myo CSA determinations was 8.8% and for ES Myo CSA, 6.1%. Linear regression analysis of observer 2’s data gave ES Myo CSA = 1.042 × ED Myo CSA−0.142 cm² (r = .906, p < .001, SEE = 1.83 cm²). The discrepancy found by observer 2 between ES Myo CSA (22.5 ± 4.3 cm²) and ED Myo CSA (21.8 ± 3.8 cm²) did not achieve statistical significance by paired t test.

M mode and two-dimensional echocardiographic measurements of posterior left ventricular wall thickness were in close agreement. No significant difference was noted between the mean M mode echocardiographic value for Wd (8.9 ± 1.1 mm) and the two-dimensional echocardiographic value (9.0 ± 1.1 mm). There were also no significant differences noted between the Ws measurements by M mode (13.4 ± 1.6 mm) and those by two-dimensional echocardiography (13.3 ± 1.5 mm). As a result, average SWT by M mode (52%) was similar to the two-dimensional echocardiographic value (48%).

Discussion
In our study M mode and two-dimensional echocardiographic measurements of SWT averaged approxi-
mately 50% and were comparable to previously report-
ed echocardiographic values but exceeded the values
obtained by more direct methods in animal studies. The
major contention of this study was that if echocar-
diographic techniques overestimate SWT to the extent
suggested by animal studies, then these techniques
should greatly overestimate ES Myo CSA relative to
ED Myo CSA. The close agreement shown, therefore,
between ES and ED Myo CSA values in this study
constitutes evidence against a significant overestima-
tion of SWT by echocardiographic techniques.

The relationship observed between ES and ED Myo
CSA closely approximated that predicted in Appendix
1, where 5% to 10% left ventricular long-axis shortening
was assumed. Determination of long-axis shortening
by several different methods has produced values
in this range. Long-axis shortening, determined
from the apical four-chamber or parasternal long-axis
view, in 13 of the 18 subjects in this study ranged from
4.8% to 13.0% (mean 7.5%), which is comparable to
the values assumed in Appendix 1. Only short-axis left
ventricular views were recorded for analysis in the firstive normal subjects.

Use of the ellipsoid formula to predict the relation-
ship between ES Myo CSA and ED Myo CSA assumes,
however, that this formula is equally representa-
tive of left ventricular geometry at end-systole and
end-diastole. This is an oversimplification. Anatomic
considerations and the recent study of Haendchen et
al. suggest that left ventricular long-axis shortening
is greater at the apex than at the base. If this is the case,
then ES Myo CSA would be expected to be in close
agreement with ED Myo CSA at the base, but to in-
creasingly exceed ED Myo CSA toward the left ven-
tricular apex.

Using the isolated canine heart preparation, Eaton et
al. have measured two-dimensional echocardiog-
graphic short-axis Myo CSA at 0.3 cm intervals along
the length of the left ventricle, calculating left ventric-
ular myocardial volume as \( \Sigma \text{Myo CSAs} \times 0.3 \text{ cm}^3 \).
They found no significant differences in echocardiog-
graphic left ventricular myocardial volumes at differ-
ent phases of the cardiac cycle from end-diastole to
end-systole. These findings are not consistent with any
significant overestimation of SWT by echocardiog-
graphic techniques.

It is not possible to accurately make such direct
measurements of myocardial volume by Simpson’s
rule in the intact human subject, in whom echocardiog-
diagnostic determinations of left ventricular volume
must be made relying on geometric assumptions. The
echocardiographic technique of measurement of Myo
CSA, as used in our study, has the advantage that no
geometric assumptions need be made. The results of
this study, allowing for a small discrepancy between
ES and ED Myo CSA values due to left ventricular
long-axis shortening, are consistent with the findings
of Eaton et al.

Critique of methods. It was assumed that myocardial
volume remains constant throughout the cardiac cycle.
Several investigators have documented a decrease in
skeletal muscle volume of the order of 0.002% during contraction. The myocardium is, however,
more vascular than skeletal muscle. Intramyocardial
blood may comprise 10% or more of left ventricular
myocardial volume. Despite constant myocardial
mass, therefore, left ventricular myocardial volume
could alter with changes in intramyocardial blood
volume throughout the cardiac cycle. Spaan et al. have
calculated, however, that the systolic reduction in
coronary blood flow can be accounted for by only a 3% 
reduction in intramyocardial blood volume, equivalent
to only a 0.3% reduction in left ventricular myocardial
volume. Such a minute alteration in myocardial vol-
ume would not significantly influence changes in left ventricular wall thickness or Myo CSA.

Recently, important regional differences in SWT of
the normal human left ventricle have been demonstra-
ted by two-dimensional echocardiography. SWT values
were shown to increase progressively from the base
to the apex of the left ventricle and to vary consid-
érably in different segments of the left ventricular cir-
cumference, being greatest in posterior segments.
Thus, the SWT data presented here, derived from pos-
terior left ventricular wall thickness measurements,
probably represent maximal values for the left ventric-
ular cross-sections studied.

Posterior left ventricular wall thickness was deter-
mined in this study from the two-dimensional echocar-
diograms by direct measurement of the distance be-
 tween the epicardial and endocardial outlines traced
for planimetry at the position transected by the M
mode cursor; this was done to allow comparison of M
mode and two-dimensional echocardiographic values
of wall thickness. SWT values were not derived from
the planimetric area data used to determine Myo CSA.
The close relationship demonstrated between ES Myo
CSA and ED Myo CSA does not, therefore, imply that
mean SWT was approximately 50% for all segments of
the left ventricular cross-sections studied. Rather the
Myo CSA data constitute evidence that endocardial
edge detection by echocardiography is reliable at end-
diastole and end-systole. Since the same endocardial
edge detection had to be relied upon for the SWT and

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the Myo CSA determinations, it is argued that the SWT data are also reliable. The SWT data, however, refer only to the posterior left ventricular wall.

Possible differences in the accuracy of end-diastolic and end-systolic determinations of Myo CSA due to the lateral resolution of echocardiographic instrumentation could conceivably account for the small systematic discrepancy observed between end-diastolic and end-systolic values. Since such an error would favor overestimation of ES Myo CSA, no attempt was made to correct for it.

**Constant myocardial mass and regional left ventricular function.** The recent echocardiographic study of Haendchen et al. demonstrated a progressive increase in SWT, endocardial fractional area change, and percent endocardial circumferential shortening from left ventricular base to apex, in agreement with results of previous studies. Percent epicardial circumferential shortening and epicardial fractional area change, however, remained constant from left ventricular base to apex. The authors were unable to explain these findings, although they discussed several possible explanations.

A close relationship has been demonstrated in our study between ES Myo CSA and ED Myo CSA, reflecting constant myocardial mass. Geometric considerations dictate that, to maintain this close relationship between ES and ED Myo CSA, a small left ventricular cross-section must thicken more than a large left ventricular cross-section if percent epicardial circumferential shortening is held constant. Successive left ventricular cross-sections diminish in circumference from the base to the apex. Given that percent epicardial circumferential shortening remains constant, therefore, our findings suggest that a progressive increase in SWT from left ventricular base to apex is inevitable if myocardial mass is to remain constant. This would be the case even if left ventricular long-axis shortening were uniform. If long-axis shortening is greater at the apex than at the base, as has been suggested, then this would further increase SWT at the apex.

**Direct measurements of SWT in animal studies.** Feigel and Fry, using a spring-loaded caliper wall-thickness transducer in anesthetized dogs, found that the left ventricular wall thickened 11% during the isovolumetric period and a further 10% during ejection. Hawthorne et al., using mercury resistance gauges, found SWT values of approximately 10% in anesthetized horses. They also found that approximately half of the SWT occurred during the isovolumetric period.

Using epicardial clips and beads positioned beneath the endocardium in dogs, Mitchell et al. determined SWT by cinefluorography, obtaining values of 25% to 45%. Sasayama et al., using implanted ultrasonic crystals in conscious dogs, recorded a mean SWT value of 31.3%. Wall thickening was not noted during the isovolumetric period in their study.

All of these animal studies have one feature in common: the exclusion of the contribution of the compressed trabeculae carnae from measurements of Ws, and hence, of SWT. McDonald et al. found M mode echocardiographic values for SWT to average approximately 65%. They suggested that the inclusion of the compressed trabeculae carnae in echocardiographic measurements of Ws was responsible for the discrep-

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**FIGURE 3.** Diagrammatic representation of a left ventricular cross-section at end-diastole (ED) and at end-systole (ES). As measured by the true endocardial marker (E), the wall thickens from 1.0 cm at ED (Wd) to 1.5 cm at ES (Ws), producing a true SWT of 50%. As measured by the marker M, apparent Wd = 0.5 cm and apparent Ws = 0.67 cm, producing an apparent SWT value of only 34% (see Appendix 2 for calculations).
anomaly between echocardiographic determinations of SWT and the values obtained in animal studies.

Studies in which it was found that approximately half of SWT occurred during isovolumetric systole, are not supported by data from studies using different techniques. The spring-loaded caliper used by Feigel and Fry may well have indented the myocardium during diastole, as the authors acknowledged, and resulted in localized wall thickening during the isovolumetric period. In any case, since wall thickening is the major cause of systolic volume displacement, it is difficult to conceive how 50% of left ventricular wall thickening could occur during a period when intraventricular volume is, by definition, constant.

The endocardial marker and ultrasonic crystal techniques of measuring SWT were subject to a further error: the ultrasonic crystals and markers used in these studies were located in a subendocardial position, at variable distances from the true endocardial surface. Mitchell et al. noted this factor in explaining the low absolute values they obtained for wall thickness, but added that percent wall thickening values should still be correct. This latter contention, however, is invalid, as is demonstrated in figure 3.

The top of figure 3 represents a left ventricular short-axis cross-section at end-diastole with a true Wd of 1.0 cm, as measured by the endocardial marker E, but an apparent Wd of 0.5 cm, as measured by the midwall marker M. The bottom of figure 3 depicts the same left ventricular cross-section at end-systole. Assuming, for simplicity, constant Myo CSA, the true endocardial marker E will move 1.25 cm toward the center of the left ventricle during systole, producing a true Ws value of 1.5 cm and a true SWT value of 50%, while the marker M will move only 0.92 cm during systole, producing an apparent Ws value of 0.67 cm and an apparent SWT value of only 34% (see Appendix 2 for calculations). Clearly, “endocardial markers” will underestimate SWT unless they are actually placed at the level of the endocardium.

Regional and segmental differences in SWT of the normal left ventricle may also have contributed to the discrepancy between SWT values obtained in these animal studies and the echocardiographic values. The precise locations from which SWT was measured in two of these studies were not specified. In the study of Feigel and Fry, SWT was determined at midventricular level on one or other side of the anterior papillary muscle. The SWT data in the study of Mitchell et al. were derived from both the anterior and posterior left ventricular wall at a level just above the papillary muscles. Differences in the sites from which SWT was determined do not, therefore, appear sufficient alone to explain the difference in values obtained in these animal studies and those obtained by echocardiography.

In conclusion, there are a number of reasons for the discrepancy between measurements of SWT in previous animal studies and the echocardiographically determined values. Techniques that accurately measure SWT should also reflect the constancy of left ventricular myocardial mass throughout the cardiac cycle. The study of Eaton et al., using their isolated canine heart preparation, and the present study in humans suggest that echocardiographic techniques more accurately measure SWT than previous “direct” techniques used in animal studies.

We thank Dr. Tom Gavaghan and Sally Kellaway for their assistance in the preparation of the manuscript.

References

Appendix 1

Theoretical relationship between ES Myo CSA and ED Myo CSA. Assuming ellipsoid left ventricular geometry, where a, b, and c are the major, anteroposterior minor, and septal–free wall minor diameters of the epicardial surface, respectively, and x, y, and z are the major, anteroposterior minor, and septal–free wall minor diameters of the endocardial surface, respectively, left ventricular wall volume (Myo V) is given by

\[ \text{Myo V} = \frac{\pi}{6} abc - \frac{\pi}{6} xyz \quad (A.1) \]

Assuming Myo V is constant at end-diastole and end-systole, then

\[ \pi (a_0 b_0 c_0 - a_0 y_0 z_0) = \pi (a b c - a y z) \quad (A.2) \]

where S = end-systole and D = end-diastole. Dividing by \( \pi / 6 \),

\[ a_0 b_0 c_0 - a_0 y_0 z_0 = a b c - a y z \quad (A.3) \]

If \( k_s \) and \( k_D \) are the apical left ventricular wall thickness at end-systole and end-diastole, respectively, then \( a_0 = a + k_s \) and \( a_D = a_0 + k_D \). Thus,

\[ (a_0 + k_s) b_0 c_0 - a_0 y_0 z_0 = (a + k_s) b c - a y z \quad (A.4) \]

Rearranging equation A.4

\[ \frac{x_0}{b c} = \frac{a_0}{y z} \]

Assuming 10% left ventricular systolic long-axis shortening, then \( x_0 = 0.9 x_D \). Substituting this value into equation A.5

\[ 0.9 x_D \left( \frac{b c}{y z} \right) = b c \left( \frac{a_0}{y z} \right) + k_s b c - k_D b c \]

Dividing by \( 0.9 x_D \)

\[ \frac{b c}{y z} = \frac{a_0}{y z} + \frac{0.9 x_D}{1.11} \quad (A.6) \]

Multiplying by \( \pi / 4 \),

\[ \pi / 4 \left( \frac{b c}{y z} \right) = \pi / 4 \left( \frac{a_0}{y z} \right) + 0.87 \frac{0.9 x_D}{1.11} \quad (A.7) \]

That is,

\[ \text{ES Myo CSA} = \frac{1.11 \times \text{ED Myo CSA}}{x_D} \quad (A.8) \]

where

\[ V = \frac{0.87 (k_D b_D c_D - k_S b_S c_S)}{x_D} \]

In a prolate ellipsoid model, where \( c_D = b_D, c_S = b_S, \) and \( x_D = 2b_D \), then

\[ V = \frac{0.87 (k_D b_D^2 - k_S b_S^2)}{2b_D} \]

But \( b_S = (1 - FS) b_D \), where \( FS \) is fractional left ventricular shortening. Hence,

\[ V = \frac{0.87 [k_D b_D^2 - k_S (1 - FS)^2 b_D^2]}{2b_D} \]

Since \( k_D < b_D \) and \( [k_D - (1 - FS)^2 k_S] < k_D \), \( V \) will be very small in relation to ES Myo CSA for all values of \( k_D, k_S, FS, \) and \( b_D \). Therefore,

\[ \text{ES Myo CSA} \approx 1.11 \times \text{ED Myo CSA} \quad (A.9) \]

Similarly, if left ventricular long-axis shortening is 5\%, then

\[ \text{ES Myo CSA} \approx 1.05 \times \text{ED Myo CSA} \quad (A.10) \]

Appendix 2

Calculation of SWT data presented in figure 3. At end-diastolic radius of epicardium \( r_{epi} \) = 3.5 cm; radius of endocardium \( r_{endo} \) = 2.5 cm; true \( W_d \) = \( r_{epi} - r_{endo} \) - 1.0 cm; apparent \( W_d \) (marker M) = 0.5 cm. From equation 1 of the text:

\[ \text{ED Myo CSA} = \pi (3.5)^2 \quad \text{ED Endo CSA} = \pi (2.5)^2 \]

At end-systole \( r_{epi} - r_{endo} = \) true \( W_s \) = 1.5 cm; \( r_{epi} = r_{endo} + 1.5 \) cm. Using equation 2 of the text and assuming, for simplicity, constant Myo CSA, then

\[ \text{ES Epi CSA} = \pi (3.5)^2 - \pi (2.5)^2 = 6\pi \]

That is,

\[ \pi (r_{endo} + 1.5)^2 - \pi (r_{endo})^2 = 6\pi \]

Solving for \( r_{endo} \), \( r_{endo} = 1.25 \) cm; \( r_{epi} = 1.25 + 1.50 = 2.75 \) cm. Similarly, the marker M defines a false endocardium with an apparent Endo CSA. Apparent Myo CSA may be calculated as follows:

\[ \text{Apparent Myo CSA} = \text{Epi CSA} - \text{Apparent Endo CSA} \]

Assuming, again for simplicity, constant Myo CSA, then

\[ \text{ES Epi CSA} - \text{Apparent Endo CSA} = \text{ED Epi CSA} - \text{Apparent ED Endo CSA} \]

That is,

\[ \pi (x^2) = \pi (3.5)^2 - \pi (2.0)^2 \]

where \( x = \) distance from marker M to center of left ventricle at end-systole. Solving for \( x \), \( x = 2.08 \) cm. Thus,

\[ \text{Apparent CSA} = 2.75 - 2.08 = 0.67 \text{ cm} \]

and

\[ \text{Apparent SWT} = \frac{0.67 - 0.5}{0.5} \times 100\% \]

\[ = 34\% \]

But,

\[ \text{True SWT} = \frac{1.5 - 1.0}{1.0} \times 100\% \]

\[ = 50\% \]
Validity of echocardiographic determination of left ventricular systolic wall thickening.
M P Feneley and J B Hickie

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