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 $1.078 \times$ ED Myo CSA $- 0.385 \text{ cm}^2$ ($r = 0.947$, SEE = 1.183 $\text{ cm}^2$) when assessed by one observer and was $1.042 \times$ ED Myo CSA $- 0.142 \text{ cm}^2$ ($r = 0.906$, SEE = 1.831 $\text{ cm}^2$) 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 carnæ 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 ($\Sigma$ 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 ellipsoidal model for calculating left ventricular myocardial volume and 10% left ventricular long-axis systolic shortening, end-systolic Myo CSA (ES Myo CSA)
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 videocassette recorder for subsequent analysis. M mode echocardiograms were recorded from the same site on the left ventricle in all 18 subjects by passing an 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 planimetered 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:

$$\text{ED Myo CSA} = \text{ED Epi CSA} - \text{ED Endo CSA}$$  
$$\text{ES Myo CSA} = \text{ES Epi CSA} - \text{ES Endo CSA}$$

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:

$$\text{SWT} = \frac{\text{Ws} - \text{Wd}}{\text{Wd}} \times 100\%$$

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

![FIGURE 1. Calculation of ED Myo CSA and ES Myo CSA in subject No. 5 by subtracting the planimetered Endo CSA from the Epi CSA at end-diastole and end-systole.](http://circ.ahajournals.org/doi/fig/10.1161/01.CIR.70.2.227)
TABLE 1
Data on left ventricular Myo CSA and wall thickness

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<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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<table>
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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 = 1.831 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 reported echocardiographic values but exceeded the values obtained by more direct methods in animal studies. The major contention of this study was that if echocardiographic 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 overestimation 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 five normal subjects.

Use of the ellipsoid formula to predict the relationship between ES Myo CSA and ED Myo CSA assumes, however, that this formula is equally representative 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 increasingly exceed ED Myo CSA toward the left ventricular apex.

Using the isolated canine heart preparation, Eaton et al. have measured two-dimensional echocardiographic short-axis Myo CSA at 0.3 cm intervals along the length of the left ventricle, calculating left ventricular myocardial volume as \( \Sigma \text{Myo CSA} \times 0.3 \text{ cm}^3 \). They found no significant differences in echocardiographic left ventricular myocardial volumes at different phases of the cardiac cycle from end-diastole to end-systole. These findings are not consistent with any significant overestimation of SWT by echocardiographic 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 echocardiographic 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 volume 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 demonstrated by two-dimensional echocardiography. SWT values were shown to increase progressively from the base to the apex of the left ventricle and to vary considerably in different segments of the left ventricular circumference, being greatest in posterior segments. Thus, the SWT data presented here, derived from posterior left ventricular wall thickness measurements, probably represent maximal values for the left ventricular cross-sections studied.

Posterior left ventricular wall thickness was determined in this study from the two-dimensional echocardiograms by direct measurement of the distance between 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 end-diastolic 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 unanesthetized 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 observed 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 discrepancy.
ancy 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\(^5,6\) are not supported by data from studies using different techniques.\(^1,7,8,24,25\) The spring-loaded caliper used by Feigel and Fry\(^3\) 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,\(^6\) 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\(^7\) and ultrasonic crystal\(^8\) 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.\(^7\) 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\(^16\) 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\(^6,8\) were not specified. In the study of Feigel and Fry,\(^3\) 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.\(^7\) 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\(^5-8\) 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.,\(^10\) 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

\[ \text{Myo V} = \pi/6 abc - \pi/6 xyz \]  

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

\[ \pi/6 (a_S b_S c_S - x_S y_S z_S) = \pi/6 (a_D b_D c_D - x_D y_D z_D) \]  

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

\[ a_S b_S c_S - x_S y_S z_S = a_D b_D c_D - x_D y_D z_D \]  

If \( k_S \) and \( k_D \) are the apical left ventricular wall thickness at end-systole and end-diastole, respectively, then \( a_S = x_S + k_S \) and \( a_D = x_D + k_D \). Thus,

\[ (x_D + k_D) b_D c_D - x_D y_D z_D = \frac{a_S b_S c_S - x_S y_S z_S}{a_D b_D c_D - x_D y_D z_D} \]  

Rearranging equation A.4

\[ x_S (b_S c_S - y_S z_S) = x_D (b_D c_D - y_D z_D) + k_D b_D c_D - k_S b_S c_S \]  

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

\[ 0.9 x_D (b_S c_S - y_S z_S) = x_D (b_D c_D - y_D z_D) + k_D b_D c_D - k_S b_S c_S \]  

Dividing by \( 0.9 x_D \)

\[ b_S c_S - y_S z_S = \frac{1.11 (b_D c_D - y_D z_D) + k_D b_D c_D - k_S b_S c_S}{x_D} \]  

Multiplying by \( \pi/4 \),

\[ \frac{\pi/4 (b_S c_S - y_S z_S)}{x_D} = \frac{1.11 [\pi/4 (b_D c_D - y_D z_D)] + 0.87 (k_D b_D c_D - k_S b_S c_S)}{x_D} \]  

That is,

\[ \text{ES Myo CSA} = 1.11 \times \text{ED Myo CSA} + V \]  

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 = fractional left ventricular shortening. Hence,

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

\[ = 0.435 \times [k_D - (1 - FS)^2 k_S] \times b_D \]

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,

\[ \text{ES Myo CSA} \approx 1.11 \times \text{ED Myo CSA} \]  

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

\[ \text{ES Myo CSA} \approx 1.05 \times \text{ED Myo CSA} \]

Appendix 2

Calculation of SWT data presented in figure 3. At end-diastolic radius of epicardium \( r_c = 3.5 \) cm; radius of endocardium \( r_e = 2.5 \) 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:

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

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

\[ \text{ES Epi CSA} - \text{ES Endo CSA} = \text{ES Myo CSA} = 6\pi \]

That is.

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

Solving for \( r_{endo} = 1.25 \) 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:

Apparent Myo CSA = Epi CSA - Apparent Endo CSA

Assuming, again for simplicity, constant Myo CSA, then

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

That is.

\[ \pi (2.75)^2 - \pi (x)^2 = \pi (3.5)^2 - \pi (3.0)^2 \]

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

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

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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