# Assessment of Left Ventricular Function by Meridional and Circumferential End-Systolic Stress/Minor-Axis Shortening Relations in Dilated Cardiomyopathy

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**Echocardiographic meridional wall stress–endocardial shortening relations provide estimates of left ventricular (LV) contractility that do not uniformly detect myocardial dysfunction despite severe symptoms in dilated cardiomyopathy. To improve detection of myocardial dysfunction in patients with congestive heart failure (CHF) due to dilated cardiomyopathy, echocardiographic meridional and circumferential end-systolic stress were related to endocardial and midwall shortening in 42 patients (95% dead within a mean of 22 months) with dilated cardiomyopathy and 140 normal subjects. A method to estimate LV long-axis dimension from M-mode minor-axis epicardial measurements was developed in a separate series of 115 subjects. Endocardial shortening to meridional wall stress relation identified 31 of 42 CHF patients falling below the 95% normal confidence interval of the reference population; use of midwall shortening decreased this number to 26 (p = NS). The use of circumferential wall stress identified 39 of 42 patients with subnormal endocardial LV shortening and 41 of 42 patients with depressed midwall performance (p <0.01 vs use of meridional stress). The circumferential/meridional wall stress ratio was 2.6 ± 0.5 in normal subjects and 1.3 ± 0.2 in CHF patients (p <0.0001). Thus, use of circumferential end-systolic stress as the measure of afterload improves the detection of myocardial dysfunction by stress/shortening relations in patients with CHF. The ratio between the 2 stresses decreases with more spherical LV shape. Midwall and endocardial shortening measurements are equivalent in the setting of thin LV walls as occurs in dilated cardiomyopathy.**

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Left ventricular (LV) performance has been traditionally assessed by stress-shortening relations,[1–5] which take afterload into account and thereby provide measures that predominately reflect intrinsic myocardial contractility.[6,7] Recently, measurement of minor-axis shortening at the LV midwall has been shown to identify LV dysfunction in asymptomatic hypertensive patients.[7] Indeed, shortening of LV midwall fibers reflects the anatomic location of the circumferential muscle bundles whose contraction contributes most to LV minor-axis shortening during systole. The difference between shortening of LV endocardial and midwall fibers is exaggerated in the presence of concentric LV geometry[7–9] and is conversely minimized in the setting of thin LV walls in anorexia nervosa.[10] An additional issue concerning use of stress/shortening relations to identify LV dysfunction is whether to use meridional or circumferential stress. In hypertensive patients in whom LV chamber size and shape are relatively normal, meridional and circumferential stress matched to midwall shortening identify a similar number of patients with LV dysfunction.[7] There is no information about the relative ability to detect LV dysfunction by endocardial or midwall LV minor-axis shortening matched with meridional or circumferential stress in the presence of marked LV dilation in congestive heart failure (CHF) patients. In the present study, wall stress/shortening relations have been reanalyzed in a series of patients with dilated cardiomyopathy[5] to compare the ability of LV endocardial and midwall shortening with different measures of wall stress to detect prognostically important depressed inotropic states.

## METHODS

**Subjects:** Two different populations were studied. The first group, used to develop methods, was formed by 50 patients with borderline to moderate systemic hypertension, 50 normotensive adults, and 15 patients with moderate to severe mitral regurgitation, who underwent 2-dimensional echocardiographic LV measurements and whose characteristics have been previously reported in part.[11] The second population (study population) consisted of 42 patients with CHF due to ischemic (n = 23) or idiopathic (n = 19) dilated cardiomyopathy (5 women and 37 men, 58 ± 11 years; arterial pressure 107 ± 17/71 ± 13 mm Hg) and 140 normal reference individuals from a population-based study (68 women and 72 men, 44 ± 13 years, arterial pressure 121 ± 10/75 ± 8 mm Hg) whose characteristics have been previously reported.[5,7] Among CHF patients, 26

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were in New York Heart Association functional class IV, 12 in class III, and 4 in class II. All patients had been referred to the Congestive Heart Failure Research Group of the New York Hospital–Cornell Medical Center, New York, New York, for invasive hemodynamic evaluation that documented depressed LV function (cardiac index = 1.68 ± 0.43 l/min/m$^2$ despite an elevated mean pulmonary artery wedge pressure of 24 ± 6 mm Hg). Detailed information on this group of patients has been previously reported.[5] Baseline echocardiography excluded primary valvular disease or diastolic dysfunction as potential causes of CHF. Forty patients died during follow-up at mean and median intervals of 22 and 16 months, respectively, after initial study (range 1 to 83 months). Two patients were lost to follow-up.

**Echocardiography:** In the method development population, 2-dimensional and M-mode echocardiograms were recorded on videotape and stripchart paper, and measurements of long-axis LV dimension were taken in apical 2- or 4-chamber view at end-diastole (Q-wave of the electrocardiogram) and end-systole (the smallest cavity size) in 4 to 6 stopframe images chosen after reviewing the videotape in slow motion and frame by frame.[12] The view that showed the largest LV long-axis dimension from apex to midmitral anular plane was selected. Two-dimensional measurements of LV short-axis diameter were taken at papillary muscle level in the parasternal short axis view.[12] In the study population, M-mode echocardiograms of the left ventricle and aortic valve were obtained with patients in the partial left decubitus position and recorded on stripcharts at 50 mm/s. Echocardiograms were coded and read blindly by 2 independent observers. Septal and posterior wall thickness and LV chamber dimensions were measured according to American Society of Echocardiography recommendations.[13] Systolic measurements of LV midwall diameter were obtained, taking into account the epicardial migration of the midwall during systole.[7]

Meridional end systolic stress (ESS) was calculated using the catheterization-validated formula of Reichek et al.[14] Circumferential (c) ESS was calculated at the midwall level using the model validated by Sandler and Dodge, McHale and Greenfield,[15,16] based on a moderately thick-walled ellipse and using a measurement of base-to-apex hemiaxis:

$$cESS = \frac{BPs * Rms}{hs} \frac{1 - Rms^3}{Ls^2(2Rms + hs)} \quad (1)$$

where BPs was systolic blood pressure, Rm the midwall hemi-minor axis, h the posterior wall thickness, and L the hemi-major axis. In the study population of CHF patients and normal subjects, LV major axis dimension was estimated as a function of LV minor axis by the following procedure. The method group formed by hypertensive, mitral regurgitation patients and normotensive subjects (n = 115) was sorted according to rank order of M-mode LV end-diastolic dimension and divided into 2 groups of 57 and 58 individuals each, with even (learning series, 25 normotensive subjects, 24 hypertensive, and 8 mitral regurgitation patients) and odd (test series, 25 normotensive subjects, 26 hypertensive, and 7 mitral regurgitation patients) sequence numbers, to obtain 2 subsets with similar distributions of minor-axis dimensions. Patients with mitral regurgitation had LV minor-axis dimensions ranging from 5.3 to 8.3 cm. In the learning series, the ratio (Z) between diastolic epicardial long axis (endocardial long axis + 45% of posterior wall thickness[17]) and short axis (LV diameter + posterior wall + septal thickness), which has been shown to be constant through the cardiac cycle,[18] was related to epicardial ($_{epi}$) minor axis (MA = 2(R + h)) by an exponential equation (by a best fitting procedure):

$$Z = \exp^{(0.98 - 0.1 * MA_{epi})} \quad (2)$$

This relation, which is equivalent to a linear regression calculating a logarithmic dependent variable (i.e., $Ln(Z) = 0.98 - 0.1 * MA_{epi}$) was close in the learning series ($r = 0.78$, *SEE* = 0.07, $p < 0.0001$) and predicted Z equally well in the test series (*SEE* = 0.08, $p < 0.0001$) despite a lower correlation coefficient ($r = 0.72$). Epicardial long axis (LA) was calculated as:

$$LA_{epi} = MA_{epi} * \text{predicted Z.} \quad (3)$$

Endocardial ($_{endo}$) LA was therefore estimated as:

$$LA_{endo} = LA_{epi} - 0.55\ PWT \quad (4)$$

where PWT is posterior wall thickness, and apical wall thickness is estimated as 0.45*PWT based on data of Streeter and Hanna.[17]

In the test series, circumferential wall stress was calculated using both direct 2-dimensional echocardiographic measurement and an indirect estimate of it, based on the assumption of a constant Z ratio through the cardiac cycle.[18] Thus:

$$LAs = MA^*_{epi_s} Z. \quad (5)$$

Because of the difficulty of imaging a perfect apical plane during systole due to the shift of the heart during its contraction, even in the best apical position, Zile and colleagues[18] suggested that using the Z ratio might, in fact, be more precise than direct measurement of the systolic left ventricular long axis. The values of circumferential wall stress derived using the measured long axis were compared, therefore, with the stresses calculated using the estimated systolic long axis derived from equations (3) through (5).

**Statistical analysis:** Statistical analysis was performed using SPSS 6.1 for Windows (SPSS Inc., Chicago, Illinois). Data are presented as mean values ± 1 SD. One-way analysis of variance was used to detect differences between patients and normal individuals. Chi-square statistics were used when appropriate. Univariate relations between variables were assessed by least-squares regression analysis. Log transformation of both circumferential and me-

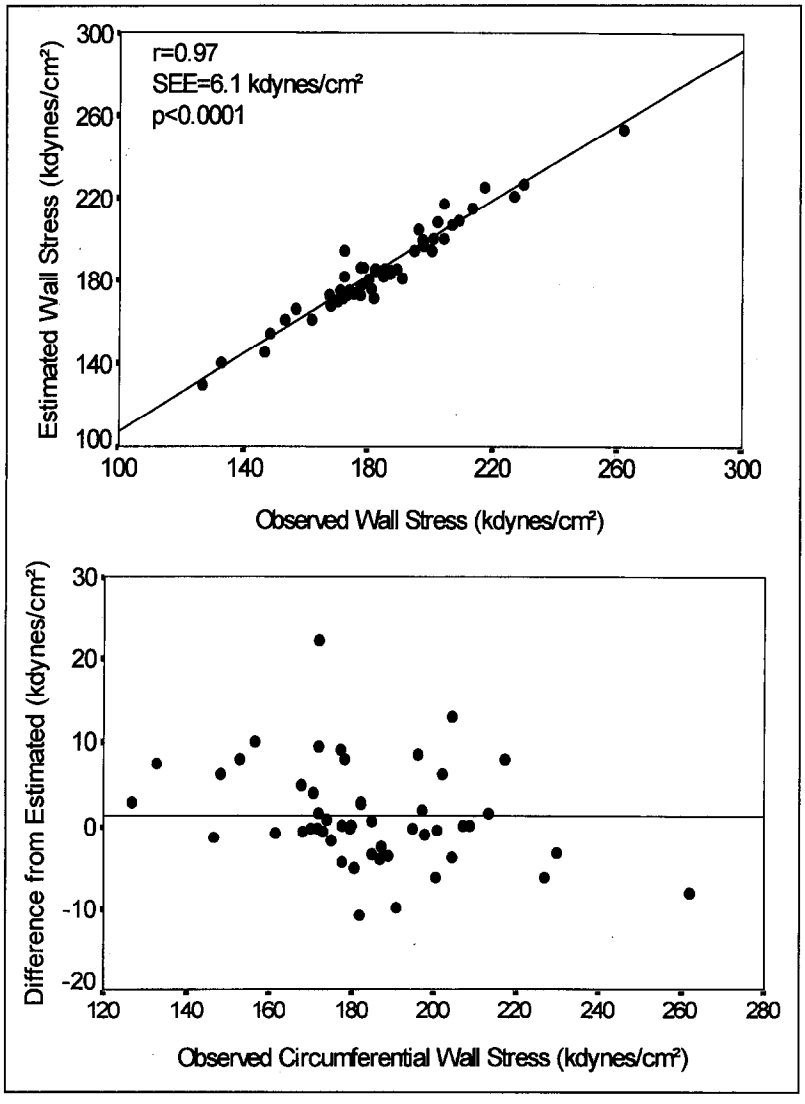


**FIGURE 1.** ***Top panel:*** **relation between observed circumferential wall stress** ***(horizontal axis)*** **and its estimate obtained using a prediction of long-axis length in systole** ***(vertical axis)*** **in the test series of normal subjects and hypertensive patients. The prediction of long-axis length is based on the assumption of a constant long-to-short axis ratio through the cardiac cycle. The regression line is close to the identity line in the test series (y = 1.01 × −4.65).** ***Bottom panel:*** **interval of agreement between values of circumferential end-systolic wall stress** ***(horizontal axis)*** **and difference between observed and estimated value in test series** ***(vertical axis).*** **The error ranged from −12 to 24 kdynes/cm².**

ridional ESS strengthened relations with endocardial fractional shortening and was used in the resulting regression equations, whereas log transformation did not improve the regressions with midwall shortening. The method of Bland and Altman[19] to calculate the "limit of agreement" was used to determine whether predicted circumferential wall stress was less accurately predicted at higher levels of observed wall stress.

## RESULTS

**Estimation of circumferential wall stress in the test series of the method population:** In the test series, circumferential wall stress calculated using estimated long-axis dimension in equation (2) (185 ± 23 kdynes/cm$^2$) related closely to circumferential stress computed from directly measured systolic long-axis dimension (176 ± 22 kdynes/cm$^2$, r = 0.90, SEE = 9.5 kdynes/cm$^2$, observed cESS = 15 + 0.87 predicted cESS). As shown in the top panel of Figure 1, predicted circumferential wall stress calculated from measured epicardial systolic minor axis and the diastolic Z ratio was even more closely related to observed circumferential wall stress; the error was not statistically related to the magnitude of observed values (Figure 1, bottom panel). Because the regression line was closely related to the identity line, estimated circumferential stress was used in the study population.

**Left ventricular systolic mechanics in the study population:** On average, endocardial shortening of CHF patients was 12% ± 4% and midwall shortening was 8% ± 3%. Table I shows the relations of measures of minor-axis systolic shortening with meridional and circumferential end-systolic wall stress in the normal subject population and in patients with CHF. In the reference population, endocardial and meridional LV minor-axis shortening were negatively related to both meridional and circumferential stress (all p <0.0001). Although the coefficients of regression were lower with midwall shortening, the standard errors of the estimate were also lower, reflecting the narrower range of LV midwall shortening.

**TABLE I** Relation Between Measures of Left Ventricular Minor-Axis Shortening and Wall Stress

| | | Normal Subjects (n = 140) | | | CHF Patients (n = 42) | | |
|---|---|---|---|---|---|---|---|
| | Model | r | SEE (%) | p < | r | SEE (%) | p < |
| Meridional stress–endocardial shortening | semilog | −0.76 | 3.6 | 0.0001 | −0.36 | 4.0 | 0.02 |
| Meridional stress–midwall shortening | linear | −0.33 | 2.2 | 0.0001 | −0.22 | 2.6 | 0.2 |
| Circumferential stress–endocardial shortening | semilog | −0.63 | 4.2 | 0.0001 | −0.11 | 4.2 | 0.5 |
| Circumferential stress–midwall shortening | linear | −0.33 | 2.2 | 0.0001 | −0.10 | 2.7 | 0.6 |

CHF = congestive heart failure.

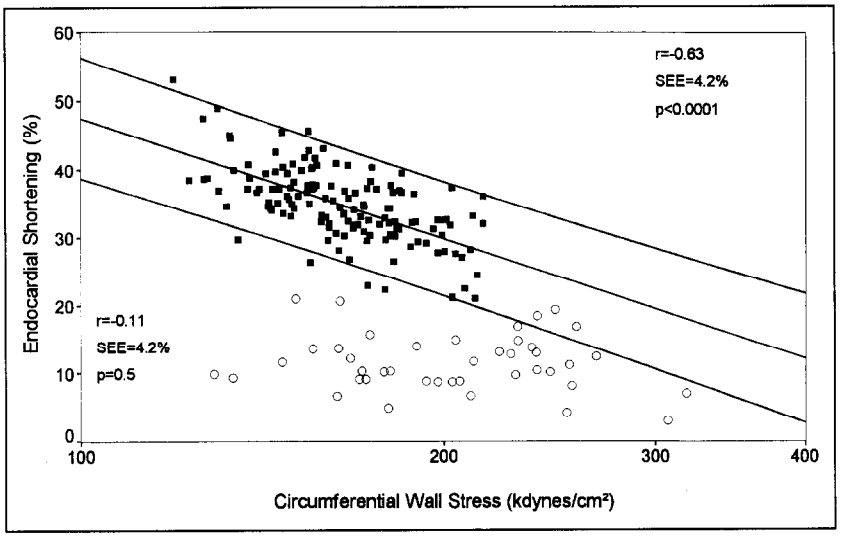


**FIGURE 2. Relation between left ventricular endocardial shortening *(vertical axis)* and circumferential end-systolic stress *(horizontal axis)*, in patients with congestive heart failure *(open circles)* and normal controls *(closed squares)*. The coefficient of correlation refers to the normal reference population. Three of 42 patients fell within the 95% normal confidence interval.**

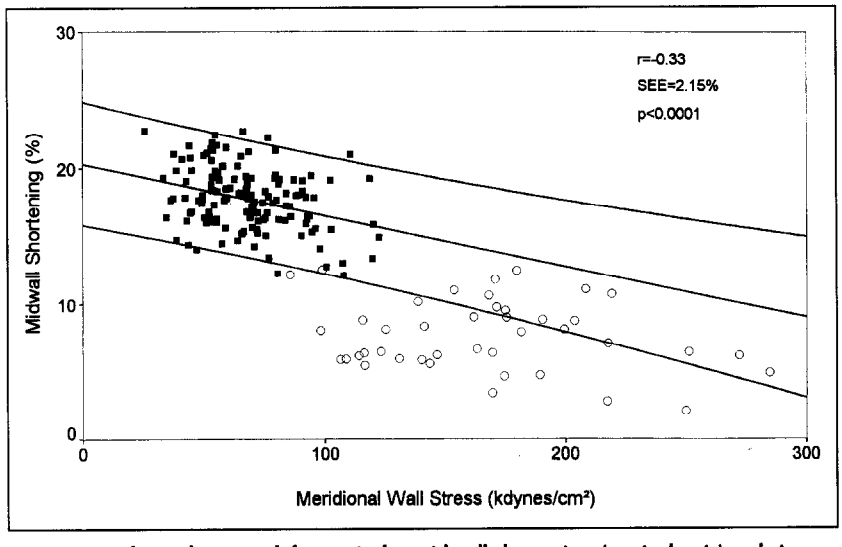


**FIGURE 3. Relation between left ventricular midwall shortening *(vertical axis)* and circumferential wall stress *(horizontal axis)* in patients with congestive heart failure *(open circles)* and normal controls *(closed squares)*. The coefficient of correlation refers to the normal reference population. One of 42 patients fell in the 95% normal confidence interval.**

LEFT VENTRICULAR ENDOCARDIAL MINOR-AXIS SHORTENING: The meridional stress–endocardial shortening relation was weak in CHF patients ($r = 0.36$, $p < 0.02$). As previously reported,[3] 11 of 42 CHF patients (26%) fell within or on the lower boundary of the 95% confidence interval of endocardial shortening in relation to meridional end-systolic wall stress. When circumferential wall stress was used as the measure of afterload, only 3 of 42, or 7%, of CHF patients fell within the 95% normal confidence interval or on its lower border (open circles, Figure 2, $p < 0.01$).

LEFT VENTRICULAR MINOR-AXIS SHORTENING AT MIDWALL LEVEL: When midwall shortening was substituted for endocardial shortening in relation to meridional ESS, the proportion of CHF patients who fell within or on the lower limit of the normal 95% confidence interval was statistically indistinguishable from the proportion with normal endocardial shortening–stress relations. When circumferential wall stress was substituted for meridional stress (Figure 3), only 1 of 42, or 2% of patients fell within or on the lower boundary of the 95% normal confidence interval ($p < 0.0001$ vs results for meridional wall stress). Midwall LV shortening was weakly related to echo meridional and circumferential wall stress in CHF patients (as expected) since LV function was reduced because of impaired contractility rather than elevated afterload (Table I).

DIFFERENCE BETWEEN MERIDIONAL AND CIRCUMFERENTIAL STRESS: Meridional wall stress in 32 of 42 CHF patients exceeded the highest level in any normal subject and fell in the upper quarter of the normal distribution in the remainder. In contrast, circumferential wall stress was normal ($<215$ kdynes/cm$^2$) in 24 of 42, or 57%, of CHF patients. At each level of circumferential wall stress, meridional wall stress was higher in CHF patients than in normal reference subjects ($p < 0.001$). As expected, the circumferential/meridional wall stress ratio was higher in normal subjects ($2.6 \pm 0.47$), than in CHF patients ($1.3 \pm 0.2$, $p < 0.0001$). The 24 CHF patients with normal circumferential stress had equally low circumferential/meridional wall stress ratios as did the 18 CHF patients with supranormal circumferential stresses ($1.3 \pm 0.3$ vs $1.3 \pm 0.2$). There were no differences between CHF patients with normal or increased circumferential wall stress in body size, gender, or LV chamber size or wall thickness, but those with higher wall stress had higher blood pressures ($117/78 \pm 20/10$ vs $100/66 \pm 8/12$ mm Hg in patients with normal wall stress, both $p < 0.002$).

## DISCUSSION

Myocardial systolic dysfunction can be identified using quantitative echocardiography by computing stress–shortening relations. The commonly used endocardial shortening–meridional wall stress relation, however, does not detect LV contractile dysfunction in all patients with even ad-

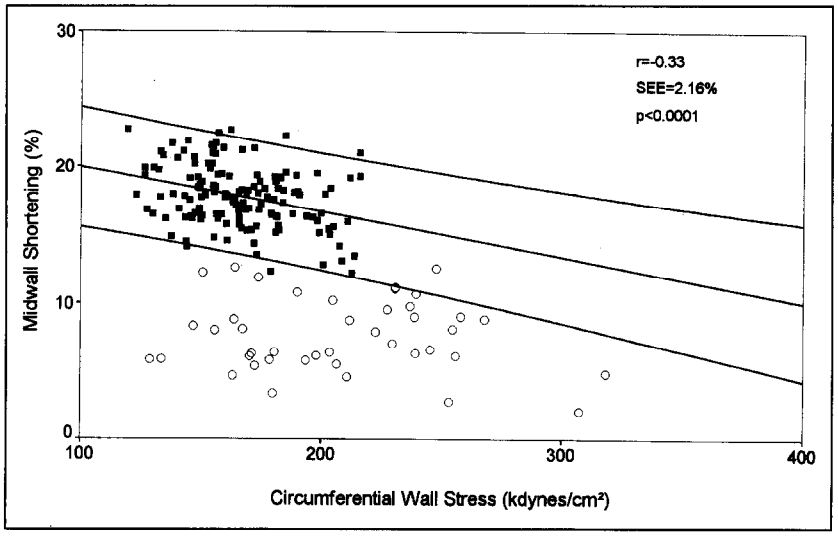


**FIGURE 4. Relation between endocardial to midwall shortening ratio *(vertical axis)* and relative wall thickness *(horizontal axis)* in patients with congestive heart failure *(open circles)* and normal controls *(closed squares)*. At each level of relative wall thickness, the ratio of endocardial to midwall left ventricular fractional shortening was lower in patients with congestive heart failure than in normal persons.**

vanced CHF. In the present study, an extensive analysis of different stress–shortening relations has been carried out, to define the best noninvasive identifier of LV dysfunction.

**Endocardial versus midwall left ventricular minor-axis shortening:** In the presence of thick LV walls, minor-axis midwall shortening is more appropriately related to wall stress, because it is measured at the same level at which wall stress is applied on average.[7,9] In the presence of thin LV walls, (as occurs in patients with anorexia nervosa[10]) the difference between endocardial and midwall fiber shortening is minimized because of the smaller distance between endocardial and midwall fibers. In our CHF patients with LV dilation, LV geometry is also characterized by a low relative wall thickness (0.25 ± 0.06 vs 0.33 ± 0.06 in our normal subjects, $p < 0.0001$). In addition, the ratio of endocardial to midwall LV fractional shortening was lower at any level of relative wall thickness in CHF patients than in normal subjects (Figure 4). Accordingly, LV midwall shortening did not separate normal subjects and CHF patients better than endocardial shortening.

**Meridional versus circumferential wall stress:** Comparing LV minor-axis shortening to circumferential wall stress improves differentiation between CHF patients and normal adults, compared with the traditional use of meridional wall stress as the measure of myocardial afterload. This difference may reflect the parallel orientations of force development by circumferential fibers during systole, located at the LV midwall, and LV circumferential wall stress. On the contrary, meridional end-systolic wall stress is oriented perpendicularly to midwall circumferential fibers. Of note, the only patient with normal midwall shortening in relation to circumferential wall stress in Figure 1 (1 of the 2 patients lost to follow-up and not known to be dead) and who had a normal, invasively determined cardiac index at baseline (3.5 L/min/m$^2$), exhibited a dilated left ventricle (7.2 cm LV diastolic dimension), suggesting a massive recruitment of Starling-forces reserve.

In this study, we calculated circumferential wall stress using an elliptical model instead of a cylindrical one previously used in studies with hypertensive patients.[7] When normal prolate ellipsoidal LV shape[20] is lost, as occurs in dilated cardiomyopathy, the more spherical LV shape exaggerates the increase in circumferential stress when it is calculated using the cylindrical model (from 166 ± 22 kdynes/cm$^2$ using the elliptical and 136 ± 34 kdynes/cm$^2$ for cylindrical model in normal patients, a mean difference of 30 kdynes/cm$^2$ or 19% ± 10%; in CHF patients: 208 ± 44 kdynes/cm$^2$ for elliptical, 289 ± 76 kdynes/cm$^2$ for the cylindrical model, a 26% ± 12% mean difference).

In addition, this study showed that at each level of circumferential wall stress, meridional stress was higher in CHF patients than in normal subjects, as previously reported[21]; thus, as the LV becomes more spherical in CHF, the circumferential/meridional wall stress ratio decreases.

**Methodologic considerations:** In this study, we used an estimate of systolic long axis to calculate elliptical circumferential wall stress, based on excellent correlation between estimated and directly measured values in our method development series (Figure 1). In addition to the good agreement with the observed circumferential stress in the "test series" of the present study, we also reexamined how circumferential stress calculated by the elliptical model with estimated long-axis dimension performed in a previously reported population of hypertensive patients.[7] Replacing the previously reported cylindrical by the elliptically calculated circumferential wall stress using the systolic long-axis dimension predicted from Z did not influence the proportion of patients identified as having normal, supranormal, or depressed midwall LV function, compared with the previously reported distribution[7] (79%, 7%, and 14%, with the elliptical, vs 79%, 5%, and 16.5% with the cylindrical model, p = NS).

To determine a 95% confidence limit of stress–shortening relations, we assumed that these relations are still linear even for the highest LV wall stresses. However, this simplification is not necessarily true, because stress–shortening relations are more likely not linear. It should be noted that fewer CHF patients had supranormal circumferential stresses than supranormal meridional stresses, thus reducing the impact of the extrapolation of normal stress–shortening relations.

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