CLINICAL STUDIES

DOES HYPERTENSION OR AGING MODULATE DIRECTIONAL SHORTENING OF THE LEFT VENTRICULAR MIDWALL?

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It is unclear whether aging or hypertension modulates directional contractile function of the left ventricular (LV) wall. We investigated LV midwall shortening and thickening in 35 normal subjects and in 15 patients with mild-to-moderate systemic hypertension (HT) using 2-dimensional echocardiography. The normal subjects were divided into 3 subgroups according to age: 13 subjects below 30 years, 12 subjects over 31 and below 59 years, and 10 subjects over 60 years. In normal subjects of all ages, no significant difference was observed between meridional shortening (%Lm = 16.3 ± 2.4) and circumferential shortening (%Lc = 17.1 ± 4.0), and a significant increase in the short-axis cross-sectional area (CSA) of the LV wall at end-systole was observed (p<0.001). No significant differences with age were found in the measurements, except that %Lc/%Lm was reduced (p<0.05) in the elderly subgroup. In HT, all measured parameters, i.e., %Lm, %Lc, %Lc/%Lm, wall thickening, and the change in CSA, showed no difference from those of normal subjects.

We conclude that the shortening of the normal LV midwall is similar in both meridional and circumferential directions, and that aging and mild-to-moderate HT do not significantly affect this characteristic, when echocardiographic measurements are taken at rest at end-diastole and end-systole. This observation may be applied to simulation analyses of basic LV mechanics, such as the finite element method.

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ANATOMICAL studies have demonstrated that while subepicardial and subendocardial fiber architecture is complex, deeper in the myocardium, the fiber arrangement is simpler. Most of the fibers in the left ventricular wall are located in the midwall and oriented circumferentially. Striking regional architectural differences can be seen in the proportion of longitudinal and circumferential fibers! 3 Based on these anatomical findings, various studies have been performed to characterize left ventricular wall contraction and to evaluate myocardial functions4—7 However, it is not yet clear whether myocardial fiber orientation affects contraction of the left ventricular wall in the circumferential and meridional directions (i.e., direction-dependent contraction). It is also uncertain whether or not aging or hypertension affects directional contractile function of the left ventricular wall.

The finite element method, which was originally developed in the field of mechanical engineering, has been applied to myocardial dynamics to investigate left ventricular...
wall function in detail. This method enables the distribution of wall stress or left ventricular regional work to be derived from left ventricular pressure and geometric changes in the left ventricular wall during the cardiac cycle. The accuracy of this method depends upon precise knowledge of changes in 3 dimensions of the left ventricular wall: meridional shortening, equatorial shortening, and radial thickening.

However, little information is available regarding these directional components of left ventricular wall contraction, which are essential to such an analyses. The present study was designed to investigate left ventricular wall contraction, and in particular, the precise relationship between meridional and circumferential (equatorial) shortening. We also assessed whether aging or hypertension influenced the dimensional changes within the left ventricular wall during contraction.

SUBJECTS AND METHODS

The study groups consisted of 35 normal subjects (18 men and 17 women, aged 16 to 83 years, mean 43.9 years) and 15 patients with systemic hypertension (>160/95 mmHg, 9 men and 6 women, aged 44 to 76 years, mean 57.5 years). The duration of the existing hypertension had ranged from about 2 to 20 years (mean 5.7 years). To examine the influence of aging, the normal subjects were divided into 3 subgroups according to age: 13 young (Y) subjects below 30 years of age (4 men and 9 women, mean 22.8 years), 12 middle-aged (M) subjects over 31 and
below 59 years old (8 men and 4 women, mean 47.8 years), and 10 elderly (E) subjects over 60 years (6 men and 4 women, mean 66.5 years). Among the normal subjects, 18 were volunteers and 17 had been referred for cardiac evaluation, however, no clinical, electrocardiographic, or echocardiographic evidence of heart disease was found.

In patients with hypertension, those with valvular or ischemic heart disease, and those who had received medication which would affect the cardiovascular system, were excluded from the study. We selected patients with hypertension whose resting 12-lead ECGs were normal or had minimal, nonspecific ST-T wave abnormalities.

Parasternal, short-axis 2-dimensional (2-D) echocardiograms of the left ventricle at the level of the chordae tendineae, as well as apical 2-chamber views, were recorded in each patient with a Toshiba SSH65A ultrasonograph equipped with a 3.75 MHz or 2.5 MHz phased-array sector transducer. Recordings were obtained with the subjects in the semireclining, left lateral position. End-diatolic 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 of the electrocardiogram, and recorded on a Toshiba LSR100A Line Scan Recorder. The 2-D echocardiograms during an entire cardiac cycle were also recorded on a videocassette recorder (Sony V0581Z) so that the images could be reviewed. Two-dimensional echocardiographic images were obtained by a single operator to obtain consistent recordings. During the examination, gain settings were continuously adjusted until the best available recordings could be made. Blood pressures were measured in the supine position with standard sphygmomanometric methods at the time of the echocardiographic study.

The left ventricular midwall line was determined manually as a series of the midpoints between the endocardium and epicardium. Meridional perimeter (Lm), the length along the long-axis left ventricular midwall from the base of the anterior aortic valve leaflet to the base of the posterior mitral valve leaflet, and circumference (Lc), the length along the short-axis left ventricular midwall, were measured with a Ushikata X-plan 360d Area-curvimeter (Fig. 1). When a small portion of the cardiac outline was missing from the image, echocardiograms recorded on videocassette were reviewed in real-time and slow motion, and thus used as a guide for accurate recognition of the epicardial and endocardial edges. Several mid-points, which were sufficient to allow reliable delineation of the midwall, were determined from detectable parts or segments of the epicardial and endocardial edges. When delineating the long-axis midwall lines, the outlines of the papillary muscles themselves were not included in the measurements. The short-axis cross-sectional areas of the left ventricular wall (CSA) were determined at end-diastole and end-systole by direct planimetry of the images recorded on the hard-copy prints. The leading-edge method was used to trace the endocardial and epicardial outlines in all of the short-axis images. To obtain mean values, 4 or 5 pairs of frames at both end-diastole and end-systole were measured for each subject. Circumferential shortening (%Lc), meridional shortening (%Lm), and mean wall thickness (mT) were calculated using the following formulas:

\[ \%Lc = \frac{\|Lcd - Lcs\|}{Lcd} \times 100 \]

\[ \%Lm = \frac{\|Lmd - Lms\|}{Lmd} \times 100 \]

\[ mT = CSA/Lc \]

\[ \%T = \frac{\|mTs - mTd\|}{mTd} \times 100 \]

where subscript d=end-diastolic and s=end-systolic.

To assess the interobserver variability of the measurements, 2 independent observers planimetrized the same frame, and then 20 pairs of determinations for Lm, Lc, and CSA were analyzed by paired t-test. The coefficient of variation for each determination was calculated by dividing the standard error by its mean, and was expressed as a percentage.

The significances of the differences between %Lc and %Lm, and between CSA1 and CSA2 were analyzed by Student's paired t-test. To compare the 3 age subgroups, a one-way analysis of variance was used. The unpaired t-test was used to compare the differences between 2 groups. Differences with P values less than 0.05 were considered statistically significant. All results were expressed as mean±SD (standard deviation).
Fig. 2. Systolic changes in circumferential and meridional perimeters in 35 normal subjects of all ages. 
A: Relationship between end-diastolic and end-systolic circumference of the left ventricular midwall (Lcd and Lcs, respectively). The equation for least squares linear regression of Lcs on Lcd is shown. 
B: Relationship between end-diastolic and end-systolic perimeter (Lmd and Lms, respectively) along the left ventricular midwall obtained from an apical 2-chamber view.

Fig. 3. Relationship between end-diastolic and end-systolic values of mean wall thickness (mTd and mTs, respectively) of the left ventricular wall in 35 normal subjects of all ages (A) and 15 patients with hypertension (B). Mean wall thickness (mT) was defined as CSA/Lc. Abbreviations are as in Fig. 1.

RESULTS

Normal Subjects of all Ages

The relationship between Lcd and Lcs was expressed by the equation for least squares linear regression as Lcs = 0.80 × Lcd + 0.53 cm (r = 0.90, p < 0.001), and that between Lmd and Lms as Lms = 0.83 × Lmd + 0.11 cm (r = 0.93, p < 0.001) (Fig. 2). For each equation, the intercept of the ordinate axis was close to 0. There were no significant differences between the circumferential and meridional percent shortening (%Lc = 17.1 ± 4.0, %Lm = 16.3 ± 2.4), and the value for %Lc/%Lm was 1.06 ± 0.26. The relation between mTd and mTs was expressed as mTs = 1.12 × mTd + 1.7 mm (r = 0.89, p < 0.001) (Fig. 3). Mean %T was 30.5 ± 8.1%. Linear regression analysis of end-systolic CSA on end-diastolic CSA gave CSAs = 1.07 × CSAd + 0.11 cm² (r = 0.95, p < 0.001) (Fig. 4). CSAs/CSAd was 1.08 ± 0.06. End-systolic CSA exceeded end-diastolic CSA in 32 of the 35 subjects, and this difference, while small was statistically significant (p < 0.001).

Effects of Aging

The 3 age subgroups were compared by a one-way analysis of variance and by

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unpaired t-test, in order to evaluate the effects of aging on left ventricular wall contraction. The magnitude of change in each measurement with age is summarized in Table I. There was no significant difference in body surface area (BSA) between any of the 3 subgroups. Heart rate also did not vary with age. Although the blood pressure of all subjects was within normal limits the aged subgroup had higher systolic pressures than the youngest subgroup (p<0.001). Left ventricular diastolic mean wall thickness (mTd) also increased with advancing age: the average mTd was 9.0±0.8 mm for subgroup Y, 10.2±1.3 mm for subgroup M, and 10.9±1.0 mm for subgroup E. Although %Lm, %Lc, CSAs/CSAd, and %T showed no significant changes with age, the mean ratio of %Lc/%Lm was lower (p<0.05) in the elderly subgroup than in the youngest subgroup (Fig. 5).

**Effects of Hypertension**

Clinical characteristics of the hypertensive
shortening ($\%Lm=15.5 \pm 3.9$), and the mean ratio of $\%Lc/\%Lm$ was $1.16 \pm 0.84$. Left ventricular diastolic mean wall thickness (mTd) in the hypertensive subjects was significantly greater than that of normal subjects ($p<0.001$). With respect to the change in mean wall thickness, $mTs=0.95 \times mTd+4.0 \text{ mm} (r=0.78, \ p<0.001)$ (Fig. 3) and $\%T=31.7 \pm 10.9\%$. Linear regression analysis of end-systolic CSA on end-diastolic CSA (Fig. 4) gave $CSAs=1.15 \times CSAd-0.74 \text{ cm}^2 (r=0.95, \ p<0.001)$. End-systolic CSA exceeded end-diastolic CSA in 13 of the 15 subjects with hypertension (CSAs/CSAd=1.11 \pm 0.08), and a significant increase in short-axis cross-sectional area of the left ventricular wall at end-systole was also observed ($p<0.001$). Although there was a difference in age ranges between normal and hypertensive subjects, none of the measured parameters in hypertensive subjects, i.e., $\%Lm$, $\%Lc$, $\%T$, $\%Lc/\%Lm$, and CSAs/CSA differed from those in any subgroup of normal subjects (Table II).

**Accuracy of Measurements**

No significant interobserver differences were noted in any of the measured parameters. The maximum values for each coefficient of variation determined by 2 independent observers for Lc, Lm, and CSA at end-diastole and end-systole were 2.7%, 2.2%, and 4.6%, respectively, which were sufficiently small for evaluation of the dimensional changes in the left ventricular wall.

**DISCUSSION**

The primary purposes of this study were to determine whether or not direction-dependent shortening exists in the normal left ventricular wall, and, if so, to examine the effects of aging and hypertension on directional shortening. Left ventricular internal dimensions, such as short-axis diameter and apex-to-base length, are often used to characterize ventricular contraction. However, the degree of myocardial wall shortening is reflected in wall thickening during systole and changes in left ventricular internal dimensions are largely mediated by changes in wall thickness. The shortening rates of intracavitary diameters and of midwall pe-
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Abbreviations: CSA=cross-sectional area of the left ventricular wall; d=end-diastole; s=end-systole; mTd=end-diastolic left ventricular mean wall thickness; %Lc=percent circumferential shortening; %Lm=percent longitudinal shortening; %T=left ventricular percent wall thickening.

*p < 0.001 as compared with normal group.

Values are means ± standard deviation (range).

Fig. 6. A: relationship between end-diastolic and end-systolic circumference of the left ventricular midwall (Lcd and Lcs, respectively) in 15 patients with hypertension. B: Relationship between end-diastolic and end-systolic perimeter (Lmd and Lms, respectively) along the left ventricular midwall obtained from apical 2-chamber views in 15 patients with hypertension.

Perimeters are not always identical. For example, when the left ventricle changes from an ellipsoid shape to a conoid shape while retaining identical internal short-axis and long-axis diameters, the meridional perimeter of the left ventricular midwall decreases, whereas internal diameters do not decrease. Thus, meridional and circumferential shortening at the left ventricular midwall was considered to be more appropriate for analyzing myocardial wall contraction. Furthermore, anatomical findings that most myocardial fibers in the left ventricular wall are located in the midwall and oriented circumferentially suggest that midwall dynamics best represent overall ventricular myocardial dynamics. Therefore, left ventricular midwall shortening was evaluated in this study.

As suggested by Gallagher et al., the midwall at end-diastole does not remain at the midpoint during the entire cardiac cycle, but shifts toward the epicardium during systole. If this is the case, underestimation of Lcs and Lms by our methods would be expected.
to produce an overestimation of the true midwall shortening. However, when relationship between the circumferential and meridional shortening is evaluated by the ratio of \(\%Lc/\%Lm\), the effects of the midwall shift are considered to be negated, and were not believed to have had a large influence in the present study.

The accuracy of our measurements relies on the accuracy of epicardial and endocardial edge detection. The validity of 2-D echocardiographic determinations in this study was supported by previous reports\(^{15,16}\) that the interobserver variability of measurements for CSA and wall thickness was within acceptable limits. However, torsional deformation of the left ventricular wall\(^{17}\) and intrathoracic cardiac motion\(^{4,18}\) also influence echocardiographic determinations. When measuring wall thickness at the lower left ventricular level, intrathoracic cardiac motion toward the base must be taken into account. Because the shape of the left ventricular apex is more conical than cylindrical, intrathoracic motion will significantly affect echocardiographic findings near the apex. Thus, in the present study we did not examine systolic changes at the lower left ventricular level. Regarding echocardiographic equipment, lateral resolution problems due to an inability to completely focus ultrasonic waves over the entire depth range of the beam\(^{19}\) may distort echocardiographic images. However, relative geometric, rather than absolute dimensional, changes during systole were the focus of the present study. Therefore, we did not believe that the errors caused by lateral resolution had a large influence on the present results.

Little information is available on the relationship between meridional and circumferential shortening.\(^{17,20-22}\) Ishikawa et al\(^{21}\) measured the coordinates of the ramifying points of the left coronary artery using biplanar coronary cineangiograms in patients who were later diagnosed as normal, and investigated regional shortening in the left ventricular wall. They reported that there were no significant differences between the amount of regional shortening in the meridional and circumferential directions. However, their results cannot be directly compared to the present results, because they measured regional changes in epicardial perimeters, whereas the present study looked at overall shortening in the midwall. The present study showed that, in normal subjects, meridional and circumferential overall shortening rates are similar. The complex structure of muscle fibers in the left ventricular wall may contribute to this observation.

In the elderly subgroup, the ratio of \(\%Lc/\%Lm\) was reduced. Possibly, the elderly subgroup had a relatively higher systolic blood pressure than the youngest subgroup, and left ventricular wall thickness increased with age, as reported earlier\(^{23-26}\). In hearts with myocardial damage, the left ventricle gradually assumes a more spherical shape because of a greater increase in short-axial diameter than in long-axial diameter\(^{27-29}\) and circumferential shortening is reduced\(^{27}\). This change decreases the ratio of \(\%Lc/\%Lm\). A small decrease in myocardial function which is undetectable by conventional indexes in clinical echocardiography may have been present in the elderly group. This is supported by the finding that age-related changes in the amount of fibrous tissue in the myocardium are induced by relatively higher afterload\(^{24,30}\).

Assuming that the volume of ventricular muscle is constant throughout the cardiac cycle\(^{31}\) and that radial contraction without longitudinal contraction occurs in the thick-walled cylindrical ventricular model\(^{13,17}\), systolic changes should not increase the short-axis cross-sectional area. However, cross-sectional area will increase with longitudinal contraction. Feneley et al\(^{15}\) reported the regression equation of \(CSAs=1.078 \times CSAd - 0.385 \text{ cm}^2 (r=0.947)\) in 18 normal subjects. Their results and the findings of the present study were in close agreement. With a constant left ventricular wall volume, if we assume a 16% systolic meridional shortening, as was observed in this study then systolic changes in cross-sectional area calculated using the thick-walled prolate ellipsoid model for the left ventricle may be expressed as \(CSAs=1.19 \times CSAd\). This relationship is consistent with the finding that the slopes of the equations for linear regression of CSAs on CSAd in this study were greater than 1.0. Thus, an increase of several percent in the short-axis cross-sectional area during systole was expected to occur with longitudinal contraction. The increase
in cross-sectional area calculated using the ellipsoid model was greater than the values obtained echocardiographically, because the effects of a non-uniform left ventricular wall thickness and asymmetrical deformation of left ventricular shape were not taken into account in the model.

It has been widely accepted that circumferential contraction reduces the diameters of the chamber and accounts for most of the power and volume of ejection, since the volume decreases in proportion to the square of the radius of a cylinder. Furthermore, shortening of the longitudinal axis is considered less prominent and less effective in ejecting blood because the volume displacement in this case in only directly proportional to the change in length. However, as we have discussed, meridional shortening increases the short-axis cross-sectional area and indirectly contributes to reduction of the short-axis diameter of the left ventricle. In other words, the wall thickening can be viewed as the direct result of shortening in both the circumferential and meridional directions. Furthermore, contractions in these directions are interdependent. Therefore, the claim that circumferential shortening ejects blood more effectively than meridional shortening is an oversimplification.

Previous echocardiographic studies have shown that left ventricular short-axis diameters, ejection fraction, percent fractional shortening, and mean velocity of circumferential fiber shortening (mVcf) in subjects with mild-to-moderate hypertension were not significantly different from values in normal subjects and that aging did not influence left ventricular myocardial contractility. The present result that hypertension and aging did not significantly affect directional shortening does not appear to be due to the inaccuracy of the measurements, since significant differences in directional shortening rates were previously observed in hypertrophic cardiomyopathy using the same methods.

In summary, the present results show that, in normal subjects, the overall shortening of the left ventricular midwall is similar in both meridional and circumferential directions. Secondly, mild-to-moderate hypertension and aging did not significantly affect this characteristic. This characteristic may be applied to simulation analyses of basic left ventricular mechanics, including the finite element method. However, the present study was limited to the difference between end-diastolic to end-diastolic dimensions in the resting state. Additional studies are necessary to determine how the left ventricular wall behaves throughout the entire cardiac cycle, and under various hemodynamic conditions.

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