Relationship Between Changes in Left Ventricular Dimensions and the Ejection Fraction in Man

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SUMMARY

Left ventricular length and calculated diameter were derived from angiocardiograms in 24 subjects with various heart diseases. Chamber diameter was noted to decrease an average of 27.5% from end-diastole to end-systole, and chamber length decreased 13%. Only subjects with calculations of left ventricular mass differing less than 25% from end-diastole to end-systole were accepted in order to avoid effects of spurious increases in end-systolic wall thickness on calculated diameter. Patients with localized disorders of contraction were also excluded. An extremely close linear relationship between the percentage change in diameter and the ejection fraction was demonstrated. Analysis of the radii of curvature showed that the lateral wall usually straightens during systole. However, if extreme ventricular dilatation is present, the lateral-wall curvature increases with systole. The possible implications of this alteration in contraction pattern and its effect upon the ejection fraction were discussed.

Additional Indexing Words:

Left ventricular volumes Left ventricular diameter Left ventricular length
Left ventricular radii of curvature Left ventricular mass

Numerous studies have confirmed the usefulness of the left ventricular ejection fraction (stroke volume/end-diastolic volume) in describing cardiac performance in chronic heart disease.1-9 Since the ejection fraction is essentially an index of the extent of fiber shortening, it would be of interest to know the relationship between the ejection fraction and changes in specific left ventricular dimensions. Studies of the normal canine left ventricle indicate that the canine ventricle empties primarily by inward motion of the lateral wall with little change in the base-to-apex dimensions.10-15 Previous studies in man have suggested a similar contraction pattern.16-19 This report will relate the ejection fraction to specific dimensional changes in the left ventricle in patients with various cardiac diseases. These studies also provide insight into alterations in the pattern of left ventricular contraction that occur when left ventricular disease is present.

Methods

Left ventricular angiocardiograms from 24 subjects with various cardiac diseases were analyzed by the area-length method.20-22 Most patients were studied with a Schonander biplane apparatus at a filming rate of 6-12 films/sec, but in five patients a single-plane RAO cine was used.21, 23, 24 Beats immediately following premature ventricular contractions were excluded. The volume calculations were adjusted by appropriate correction factors to adjust for errors caused by

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image magnification, distortion due to X-ray equipment, the use of an ellipsoid model to represent the left ventricular chamber, and inclusion of papillary muscles and trabeculae carneae in the volume estimation. Total left ventricular stroke volume was determined by subtracting end-systolic volume from end-diastolic volume. The ejection fraction was calculated as a ratio of total left ventricular stroke volume to end-diastolic volume. The calculation of left ventricular mass was used as a means for substantiating the accuracy of the volume determinations. Left ventricular mass was determined from measurements of end-diastolic and end-systolic free-wall thickness on AP or RAO angiographic films by methods previously described. The potential for greatest error in determining chamber dimensions and wall thickness occurs at end-systole when the area of the opacified ventricular image is the smallest and most irregular. The left ventricular wall may be gauged to be overly thick due to poor chamber-margin opacification. This results in falsely low end-systolic dimensions and, therefore, falsely elevated values for end-systolic left ventricular mass. It is assumed that left ventricular mass should be the same at end-diastole and end-systole. On this basis, patients in whom end-systolic mass exceeded calculated end-diastolic mass by more than 25% were excluded from this study. Such deviations must reflect errors in determination of chamber margins. The error limit for comparison of mass calculations was chosen on the basis of variations in chamber dimensions and volumes when such determinations are made for casts of postmortem left ventricles. Patients with striking localized disorders of contraction were also excluded.

Left ventricular length (L) was measured directly from the angiocardiogram as the distance from the apex to the midpoint of the aortic valve plane. Left ventricular diameter (D) was derived from the assumption that the ventricular chamber represents a prolate ellipse, where \( A \) represents the planimetered area of the ventricular image:

\[
D = \frac{4A}{\pi L}
\]  

(1)

Previous studies as well as our own measurements have indicated that D calculated in this manner correlates well with directly measured D. However, due to the irregular shape of the ventricular cavity, direct measurement of D involves arbitrary assumptions. The calculated D represents a mathematical approximation of the mean D.

Changes in ventricular shape were determined from calculations of the principal radii of curvature for a prolate ellipse used to represent the left ventricular chamber. These principal radii of curvature are mathematically defined as:

\[
r_1 = \frac{L^2}{2D}; r_2 = \frac{D}{2}
\]

(2)

where \( r_1 \) defines the curvature of the long axis of the ellipse (lateral-wall curvature), and \( r_2 \) defines the curvature along the short axis (circumferential curvature). These are illustrated in figure 1. Values for \( r_1 \) and \( r_2 \) were calculated at both end-systole and end-diastole, and the maximal difference during the cardiac cycle being expressed as \( \Delta r_1 \) and \( \Delta r_2 \). The shape of the left ventricle was also described by the ratio of L/D and calculated at both end-systole and end-diastole. In order to normalize for variations in initial ventricular volume from patient to patient, L and D were calculated as percentage changes from end-diastole (ED) and end-systole (ES):

\[
\% \Delta D = \frac{D_{ED} - D_{ES}}{D_{ED}}; \% \Delta L = \frac{L_{ED} - L_{ES}}{L_{ED}}
\]

(3)

Values for percentage change in diameter (\( \% \Delta D \)) are identical with percentage change of circumference since conversion of diameter to circumference (multiplication by \( \pi \)) is canceled when it is divided by its respective end-diastolic value.

All statistical analyses were carried out with a Hewlett-Packard model #9100-A calculator and the OMNITAB program developed at the Statistical Laboratory, Iowa State University, by R. L. Chamberlain, in conjunction with an IBM 360 model 75 computer.
### Table 1

Analysis of Angiocardiographic Data in Twenty-Four Subjects with Cardiac Disease

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Mean = 55 ± 2.9 SEM = 13 ± 1.2 EF = 27.5 ± 2.0 LVEDV = 53 ± 6.3 EF = 93 ± 20 ± 0.05 MS = 93 ± 20 ± 0.07

Abbreviations: ED = end-diastolic; ES = end-systolic; LVEDV = left ventricular end-diastolic volume; EF = ejection fraction; H = wall thickness; LVM = left ventricular mass; SEM = standard error of the mean; MS = mitral stenosis; MI = mitral insufficiency; AS = aortic stenosis; AI = aortic insufficiency; ILVH = idiopathic left ventricular hypertrophy; ASHD = arteriosclerotic heart disease; PMD = primary myocardial disease; POMVR = postoperative mitral valve replacement; LVP = left ventricular pressure; L = length; D = diameter.

*See text for explanation.
The relationship between end-diastolic length and diameter with end-diastolic volume. There is a nearly identical increase in both dimensions. This represents a relatively greater increase in diameter (see text).

Results

The individual data are presented in table 1, and the patients are ranked in order of increasing end-diastolic volume. There was a wide range of end-diastolic volumes and ejection fractions. The calculated end-systolic left ventricular mass changed by less than 25% of the end-diastolic value in all subjects and averaged 9.3% ± 2.0 (SEM). The change in left ventricular wall thickness from end-diastole to end-systole varied from 15 to 175% with a mean change of 53% ± 6.3.

Changes in Left Ventricular Diastolic Shape

Figure 2 illustrates the relationship between left ventricular length and calculated diameter as end-diastolic volume increased. The absolute increase in both dimensions with increasing left ventricular end-diastolic volume was virtually identical. Since the length is normally twice the diameter, the percentage increase in length was relatively less than the percentage increase in diameter. Thus, as the ventricle dilates the diameter becomes relatively larger, and the ventricle assumes a more spherical shape.

Changes in Left Ventricular Shape during Systole

Changes in ventricular shape during systole were analyzed by two methods: measurement of radii of curvature; and measurement of changes in the ratio of L/D. Measurement of changes in the radii of curvature from end-diastole to end-systole showed that the circumferential curvature increased in all subjects. However, analysis of changes in lateral-wall curvature revealed some interesting differences. In 17 patients (group I) the lateral wall straightened during systole as indicated by an increase in the radius of curvature L²/2D. In seven patients (group II), indicated by an asterisk in table 1, the lateral-wall curvature increased during systole as indicated by a decrease in L²/2D. All but one of these patients had an end-diastolic volume in excess of 250 ml. There were significant differences in the end-diastolic volume and ejection fraction between these two groups (fig. 3). Group I averaged 171 ml ± 12.1 versus 331 ml ± 37.4 for group II (P < 0.001), and the ejection fraction for group I was 60% ± 2.5 versus 41% ± 4.4 for group II (P < 0.001). When ΔL²/2D was correlated with end-diastolic volume, a correlation of r = 0.658 was obtained.

It was discovered that the simpler measurement of the end-systolic L/D ratio provided equally good discrimination between the two groups of patients.

Figure 3

The ratio of length/diameter (L/D) at end-diastole (ED) and end-systole (ES) in 17 subjects in whom the lateral wall straightened during systole (group I) and seven in whom the curvature increased (group II). The mean value is indicated by a heavy line.
The linear relationship between the ejection fraction and %Δ D. A highly significant correlation is present. The equation for the regression is indicated.

groups (fig. 3). Whereas the L/D ratio was not significantly different between the two groups at end-diastole (1.73 ± 0.06 and 1.59 ± 0.1; P > 0.1), there was a significant difference at end-systole (2.21 ± 0.06 versus 1.72 ± 0.12; P < 0.001). The absolute change in L/D ratio from ED to ES was less than 0.3 in all seven subjects in group II, whereas this figure ranged from 0.4 to 1.7 for the subjects of group I. It was concluded from these findings that as end-diastolic volume becomes increased due to disease the contraction pattern of the ventricle changes, and it becomes more spherical during systole. This is most marked when the ejection fraction is low.

Relationship of Dimensional Changes during Systole to the Ejection Fraction

The relationship of the ejection fraction to changes in length and diameter, given an ellipse of revolution as a model, is as follows:

\[
EF(\%) = \%ΔL + 2(\%ΔD) - 2 \times 10^{-2}(\%ΔD)^2 - 10^{-4}(\%ΔL)(\%ΔD)^2 - 2 \times 10^{-5}(\%ΔL) - 2 \times 10^{-2}(\%ΔD)^2
\]

(4)

By employing a factorial search technique of regression, the %ΔL terms were found to contribute very little to this relationship when the data from this study were analyzed. In part this is due to the relatively good correlation between %ΔD and %ΔL (r = 0.69; P < 0.001). Hence the linear and quadratic relationships of the ejection fraction to diameter change were analyzed, and the error as estimated by the standard deviation of the residuals (σ̂) was compared to the full model (equation 4). For the full model:

\[
EF(\%) = 1.52(\%ΔL) + 1.75(\%ΔD) - 4 \times 10^{-2}(\%ΔD)(\%ΔL) - 4.73 \times 10^{-5}(\%ΔL) - 3.30 \times 10^{-2}(\%ΔD)^2
\]

\[σ̂ = 1.76\]

(5)

For the quadratic model:

\[
EF(\%) = 7.05 + 2.08(\%ΔD) - 1.15 \times 10^{-2}(\%ΔD)^2
\]

\[σ̂ = 2.01\]

(6)

For the linear model:

\[
EF(\%) = 15.09 + 1.43(\%ΔD)
\]

\[σ̂ = 2.41\]

The standard deviation of the residuals is small for each model, and little reduction in error results from employing the more complex models. The linear relationship is illustrated in figure 4. The regression coefficient is r = 0.985 (P < 0.001).

Discussion

The configuration of the human left ventricular chamber was studied at end-diastole and end-systole. The changes in diameter were obtained from a calculated dimension using equation 1 of the text, rather than a direct measurement from the films. This was done because direct measurement of diameter is often technically difficult. Recent studies in this laboratory and by others have demonstrated a high correlation between such calculated chamber dimensions and directly measured values, although use of the direct measurement results in overestimation of volume.34,35 Additionally such calculations have shown that the human left ventricle has a nearly circular cross-sectional configuration at both end-diastole and end-systole. Finally there appears to be minimal rotation of the long and short axis of the left ventricle during
systole so that study of these axes from a single plane, as was the case in five of our subjects, should introduce no error.36

The mechanisms by which the ventricle changes its length during systole deserve comment. Recent studies have demonstrated that the base of the heart descends toward the apex during systole.36 This probably accounts for the change in length occurring in most of our patients. Layered dissections of healthy human hearts have demonstrated that the middle circumferentially oriented fiber layer covers only the upper 60% of the ventricular chamber.37 The apical or lower 40% of the chamber is covered almost entirely by spirally directed fibers. Contraction of these fibers should tend to approximate the papillary muscles and produce obliteration of the apex. The phenomenon of apical obliteration has been observed angiographically for many years. This phenomenon could produce a spurious shortening of ventricular length during systole. It is commonly seen in exaggerated form in idiopathic hypertrophic subaortic stenosis though it is occasionally seen in other disorders or in normal subjects.38–40 Our criteria for inclusion of patients in this study tended to exclude those who might show apical obliteration, because of difficulties in measuring end-systolic volume and mass in such patients. However, one patient with muscular subaortic stenosis was studied, and this individual had the highest %ΔL (31%), which was due to apical obliteration by the papillary muscles.

Layered dissections in a few hypertrophied and dilated hearts have demonstrated that the circular muscle bundles which cover only the basal 60% of normal-sized hearts may extend farther down toward the ventricular apex.37 As noted by Deliyannis, this fiber distribution inhibits apical obliteration. That the apex appears to empty poorly in many dilated hearts is a common observation during angiography, and was noted in several subjects in this series. Thus, left ventricular dilatation produces an "apical dead space" which could contribute to the reduced ejection fraction often associated with cardiac dilatation.

It would appear from the data derived from this study that the heart changes its mode of contraction when diastolic volume increases above 265 ml. This conclusion is based on calculations of lateral-wall curvature and/or L/D ratios at end-diastole compared to end-systolic values. In all subjects with end-diastolic volumes greater than 265 ml, the lateral wall became more curved with ejection. These dilated ventricles are contracting more nearly as a sphere.

Whether the more spherical contraction pattern exhibited by large ventricles is of consequence cannot be directly answered by this study. However, Sallin has suggested that the helical arrangement of the inner and outer myocardial fiber layers of the ventricular shell allows the ventricle to empty much more completely than would be the case if all the fibers were arranged in a circumferential fashion.41 If the ventricle were a sphere, then helical arrangement of fibers would confer no advantage over circumferential arrangement, and the extent of emptying would depend upon the extent the sarcomeres can shorten. Sarcomeres appear to have a finite extent of shortening.42 Left ventricular dilatation could diminish the advantage of a helical fiber arrangement and reduce the ventricle's ability to empty (and hence reduce the ejection fraction) even in the presence of relatively intact sarcomere function.

Studies of ventricular dynamic geometry in experimental animals have demonstrated that ejection is accomplished primarily by diameter changes without significant shortening of the apex-to-base length.10–15 Our studies confirm a similar pattern for the diseased human left ventricle, so long as the ejection fraction is normal. In such patients the percent change in diameter was nearly always two to four times greater than the percent change in length. As the ejection fraction fell below normal (usually in grossly dilated ventricles) the percent change in diameter approached the value of the percent change in length.

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It is of great practical interest that the percent change in diameter faithfully predicted the ejection fraction regardless of the size or contraction pattern of the ventricle. Analysis of the various models relating the ejection fraction to dimensional changes showed little reduction of error (\(\hat{\sigma}\)) by using models more complex than the simple linear model relating diameter change to ejection fraction. Thus for practical purposes measurement of change in length is unnecessary to predict the ejection fraction if knowledge of the change in diameter is available.

This relationship has important implications for studies of left ventricular function by nonangiocardiographic methods. Because measurements of percent diameter changes are virtually interchangeable with the ejection fraction, the good correlation found between the ejection fraction measured by the angiographic technique and by the echocardiographic technique are now readily explained.\(^{43, 44}\) Echocardiography measures wall motion at a plane just below the level of the mitral valve which approximates the true diameter of the left ventricle. It is acknowledged that the D measured by echocardiography may differ slightly from the D derived in this study. However, studies in which \(\%\Delta D\) measured from echocardiography have been correlated with EF have nonetheless yielded a high correlation with EF.\(^{45}\) This is of obvious clinical significance for it should allow an evaluation of the ejection fraction in critically ill patients who cannot be studied by angiocardiography, as well as providing a technique for serial long-term study of ventricular function.

It is important to emphasize that there were certain constraints placed upon patient selection in this study. First, all patients in whom calculated left ventricular mass increased by more than 25% from end-diastole to end-systole were excluded. This represents an effort to reduce spuriously high \(\%\Delta D\) values due to inaccurate end-systolic cavity estimation. We feel this method should be applied to all quantitative angiographic studies as a check on the reliability of the calculated ejection fraction, since the ejection fraction depends upon the subtraction of end-systolic volume from end-diastolic volume. In spite of using the criteria of a change in the value of a left ventricular mass of less than 25%, the value of \(\%\Delta H\) in this series is slightly higher than that found by Hugenholtz who used a more elaborate method to correct end-systolic wall-thickness measurements.\(^{30}\) By doing this we biased our sample to exclude subjects with marked concentric hypertrophy. Secondly, we excluded patients with striking localized disorders of contraction. This eliminated some patients with coronary artery disease and old myocardial infarction. In many of these subjects a meaningful \(\%\Delta D\) at the midwall cannot be obtained. Thus, the close correlation of \(\%\Delta D\) and ejection fraction may not necessarily be present in some patients with coronary artery disease.

Addendum

Since this work was performed, similar studies have been performed in 28 additional patients in whom left ventricular volumes and dimensions were measured from RAO cine projection at 60 frames/sec. Employing the correction factors of Kasser, a relationship virtually identical to that of the present study has been obtained (\(y = 1.31x + 15.0; r = 4.66; r = 0.963; P < 0.001\)).\(^{24, 28}\) Thus, the RAO cine method appears as useful as the biplane method for defining this relationship, but as might be expected the error is slightly larger (\(\hat{\sigma}\) of 4.66 compared to 2.41).

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