Determination of Left Ventricular Volumes by Ultrasound

By Nicholas J. Fortuin, M.D., William P. Hood, Jr., M.D., M. Eugene Sherman, M.D., and Ernest Craigie, M.D.

SUMMARY
We compared dimensions of the left ventricular minor axis (S) measured at end-diastole \( (S_D) \) and end-systole \( (S_S) \) by echocardiography with dimensions and left ventricular volumes measured by biplane angiocardiography in 27 patients with diverse cardiac abnormalities. There were high correlations between echographic and angiographic ventricular minor-axis dimensions \((r = 0.85 \text{ for } S_D \text{ and } 0.87 \text{ for } S_S)\), between echographic dimensions and ventricular volumes \((r = 0.84 \text{ for } \text{end-systolic volume [ESV]} \text{ and } S_S, 0.83 \text{ for } \text{end-diastolic volume [EDV]} \text{ and } S_D)\), and between the relative change in the echographic minor axis with systole \((\% \Delta S)\) and ejection fraction \((r = 0.79)\). Regression formulae were derived from these relationships which allowed calculation of ventricular volumes from echo dimensions alone: ESV = 47 \( S_D - 120 \), EDV = 59 \( S_D - 153 \). These equations allowed relatively accurate prediction of volumes over a wide range of ventricular sizes. The use of a cube function of the echographic minor axis was an accurate predictor of volumes only in smaller ventricular chambers, but overestimated volumes in larger hearts.

Additional Indexing Words:
Angiocardiography  Minor-axis dimensions  Noninvasive techniques
Left ventricular geometry  Ejection fraction  Echocardiography

The ability to measure left ventricular volumes during the cardiac cycle allows characterization of the mechanical or pump function of the heart in a variety of normal and abnormal clinical states.\(^1,\)\(^2,\)\(^5,\)\(^6\) Such determinations permit measurement of left ventricular stroke volume,\(^3\)\(^7,\) quantitation of regurgitant or shunt flow,\(^4,\)\(^5\) characterization of ventricular wall stresses,\(^6,\)\(^7\) estimation of ventricular mass,\(^8\) and evaluation of velocity of circumferential fiber shortening.\(^9\) Further, several groups have stressed the clinical utility of the left ventricular ejection fraction, which is derived from volume measurements, as an index of myocardial pump function.\(^1,\)\(^2,\)\(^10\) Unfortunately, measurement of left ventricular volumes and ejection fraction has heretofore been possible only by techniques which require cardiac catheterization with the risks, inconvenience, patient discomfort, and expense attendant on this procedure. Because of this, routine or repeated study of ventricular volumes has not been generally applicable to the longitudinal follow-up of patients with cardiac disorders. It is likely that the ability to estimate left ventricular volumes accurately and repeatedly would result in improved management of patients with many types of heart disease, particularly those in whom cardiac surgical interventions may favorably alter the natural history, and better under-
### Table 1

**Left Ventricular Volumes and Dimensions Obtained by Angiocardiography, and Ventricular Dimensions Obtained by Echocardiography**

<table>
<thead>
<tr>
<th>Patient no.</th>
<th>Diagnosis</th>
<th>Age (years)</th>
<th>Ventricular Dimensions Obtained by Echocardiography</th>
<th>Ventricular Dimensions Obtained by Angiocardiography</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>MS, MR</td>
<td>52</td>
<td>133</td>
<td>EDV = end-diastolic volume; ESV = end-systolic volume; SV = stroke volume; EF = ejection fraction; S0 = minor axis at end-diastole; Sa = minor axis at end-systole; %ΔS = relative change in minor axis with systole; MS = mitral stenosis; MR = mitral regurgitation; AR = aortic regurgitation; IHSS = idiopathic hypertrophic subaortic stenosis; VSD = ventricular septal defect; AS = aortic stenosis; MV = mitral valve; PDA = patent ductus arteriosus; ASD = atrial septal defect; OMI = old myocardial infarction.</td>
</tr>
<tr>
<td>2</td>
<td>AR</td>
<td>56</td>
<td>265</td>
<td>154</td>
</tr>
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<td>MS</td>
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<td>148</td>
<td>51</td>
</tr>
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<td>4</td>
<td>IHSS</td>
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<td>138</td>
<td>51</td>
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<tr>
<td>5</td>
<td>VSD</td>
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<td>132</td>
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<tr>
<td>6</td>
<td>VSD</td>
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</tbody>
</table>

Abbreviations: EDV = end-diastolic volume; ESV = end-systolic volume; SV = stroke volume; EF = ejection fraction; S0 = minor axis at end-diastole; Sa = minor axis at end-systole; %ΔS = relative change in minor axis with systole; MS = mitral stenosis; MR = mitral regurgitation; AR = aortic regurgitation; IHSS = idiopathic hypertrophic subaortic stenosis; VSD = ventricular septal defect; AS = aortic stenosis; MV = mitral valve; PDA = patent ductus arteriosus; ASD = atrial septal defect; OMI = old myocardial infarction.

Understanding of the effects of other therapeutic interventions.

Recent studies in our laboratory on the geometry of contraction of the human left ventricle have indicated that in both normal and abnormal hearts changes in volume with ventricular systole occur mainly as a result of shortening of the minor ventricular axis. In addition, the relative amount of change in the minor axis with systole has been found to correlate closely with left ventricular ejection fraction. Others have recently reported that a minor axis of the left ventricle can be measured noninvasively by the use of ultrasonic echocardiography and have shown a significant correlation between the minor axis determined by this technique and left ventricular volumes measured by angiographic methods.

We undertook the present study to validate further the relationship between left ventricular minor-axis dimensions measured by echocardiography and minor-axis dimensions and volumes measured by angiocardiography. We have also characterized this relationship in mathematical terms so that accurate estimates of left ventricular volume can be obtained from the echo measurements alone.
Cross section through the heart at the level of the mitral valve to show paths traversed by echo beams. Sternum and echo transducer are to the left, spine to the right. The short dashed line represents the path of the echo beam to the anterior mitral valve leaflet. Lateral and inferior movement of the transducer results in an ultrasonic path which passes through chest wall, right ventricular wall, interventricular septum, and posterior left ventricular wall successively, as indicated by the line with longer dashes. RA = right atrium; RV = right ventricle; LA = left atrium; LV = left ventricle.

Method

The 27 patients included in this study were those referred for routine diagnostic catheterization at our hospital in whom high-quality echocardiograms and angiograms for measurement of left ventricular dimensions and volumes respectively were obtained. The diagnoses and ages of these patients are shown in table 1.

Cardiac catheterization was performed under local anesthesia without premedication. Angiocardiograms were recorded in the anteroposterior and lateral projections at 6 or 8 per second by means of an Elema-Schonander roll film changer following injection of contrast material into the pulmonary artery, left atrium, left ventricle, or aortic root depending upon the cardiac lesion present. Left ventricular volumes were calculated by the area-length method of Dodge et al.17 employing regression data and a computer program previously reported from this laboratory.18 End-diastolic volume (EDV) and end-systolic volume (ESV) were obtained from the angiograms and stroke volume (SV) was calculated by subtracting ESV from EDV. Left ventricular ejection fraction (EF) was obtained by dividing SV by EDV.

The left ventricular minor axis (S) was derived from the angiocardiograms assuming an elliptical reference figure and utilizing the formula:

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

where A = planimetered chamber area and L = the longest measured length on each film. The value obtained from both anteroposterior and lateral films was corrected for X-ray magnification and the values for S derived from paired anteroposterior and lateral films were averaged to obtain the short axis of an idealized prolate ellipsoid at end-diastole (Sn) and end-systole (Ss).

Echocardiograms were recorded for measurement of the left ventricular minor axis within 24 hours of cardiac catheterization employing a Smith-Kline Ekoline 20 machine utilizing a 2.25-megaHertz transducer of 0.75-inch diameter. The minor axis was measured by the technique described by Popp and associates.13, 14 In brief, the ultrasonic transducer was placed in the fourth or fifth intercostal space just to the left of the sternum. The ultrasonic beam was directed posteriorly and slightly medially through the chest wall until the characteristic pattern of mitral valve motion was encountered. The direction of this beam is shown in figure 1, a diagrammatic horizontal section through the thorax at the level of the mitral valve. The ultrasonic beam was then directed slightly laterally and inferiorly away from the mitral valve until a plane was found in which motion of the posterior left ventricular wall and interventricular septum was observed (fig. 1). Echoes from these structures produce highly characteristic patterns of motion (fig. 2), the posterior wall moving anteriorly or toward the transducer with ventricular systole, the septum moving away from the transducer or posteriorly during systole. These movements were recorded on film by displaying the echoes in the time-motion mode and taking a time exposure of a single oscilloscopic sweep (fig. 2).

Standardization of the technique from subject to subject was attempted by using the easily recognized mitral valve echo as a reference point from which to direct the echo beam to a plane where septum and posterior wall are seen. Minor-axis end-diastolic dimension (Sn) was measured at the time of the R wave of the simultaneously recorded electrocardiogram, and end-systolic dimension (Ss) at the point where posterior wall and septum approach each other maximally (fig. 2). The echo ejection
fraction or relative change in minor axis with systole \( \Delta S_{\text{echo}} \) was computed by

\[
\frac{S_d - S_S}{S_d}.
\]

In patients with atrial fibrillation, measurements of the minor-axis dimensions for at least five cardiac cycles were made and the mean of these determinations was used as the final value. All measurements on echocardiograms were made by one of us (N.J.F.). The reproducibility of the measurements among observers and at different times in the same patient has recently been documented by Pombo et al.\(^{19}\)

**Results**

Minor-axis dimensions and ventricular volumes and ejection fraction determined by angiocardiography and minor-axis dimensions determined by echocardiography for each patient are given in table 1.

**Minor-Axis Measurements by Angiocardiography and Echocardiography**

A comparison of minor-axis dimensions determined by the two techniques at end-systole and end-diastole is presented in figure 3. In both systole and diastole the correlation coefficients are highly significant \( (P < 0.001) \). There is a slight overestimation of \( S_S \) by echocardiography and a small underestimation of \( S_d \) in hearts of less than 7.0 cm end-diastolic diameter. This results in an underestimation of \( \Delta S \) by the echo method, although the correlation of \( \Delta S \) determined by the two techniques remains highly significant \( (r = 0.80, P < 0.001) \).

**Angiographic Volume Measurements and Echographic Minor-Axis Dimensions**

Figure 4 presents the relationship between angiographic ventricular volumes and echo dimensions in systole and diastole. There are highly significant correlations between the variables \( (P < 0.001) \). From these data we derived regression formulae for derivation of ventricular volumes from the echo dimensions alone:

\[
\text{ESV} = 47 S_{\text{echo}} - 120 \quad (1)
\]

\[
\text{EDV} = 59 S_{\text{echo}} - 153 \quad (2)
\]

There is also a significant relationship between \( \%\Delta S_{\text{echo}} \) and EF \( (\text{fig. 5}, \ r = 0.79, P < 0.001) \). The regression equation relating the two variables derived from these data is:

\[
\text{EF} = 1.3 \%\Delta S_{\text{echo}} + 25 \quad (3)
\]

We excluded one patient, no. 27, from the data analysis shown in figures 3, 4, and 5. This
LV VOLUMES BY ULTRASOUND

VOLUME XLIV, October 1971

Comparison of echo-measured minor-axis dimensions (Sp, at end-diastole; Sb, at end-systole) with angiographic end-systolic (ESV—x’s) and end-diastolic (EDV—solid circles) volumes. Solid and dashed lines are least-squares regression lines for variables. R = correlation coefficient; SEE = standard error of the estimate.

The patient deviated markedly from the statistical trends shown by the others in the %ΔS echo-EF comparison. This probably occurred because he had a localized disorder of myocardial contraction. We felt justified in excluding this patient because the echo technique, which samples only a single point on the septum and posterior left ventricular wall, may not accurately reflect ejection fraction in this situation.

Derivation of Ventricular Volumes from Echo Dimensions

We used two techniques to derive ventricular volumes from the echo data. Equations 1 and 2 were employed to compute ESV’ and EDV’, and from these values SV’ and EF’ were calculated.* Volumes were also derived from the formula:

\[
V = 1.047 \, S_{\text{echo}}^3
\]  

(4)

as described recently by others.\textsuperscript{14} Using equation 4, ESV”, EDV”, SV”, and EF” were computed.

In figures 6, 7, and 8 end-systolic volume, end-diastolic volume, and stroke volume derived from the echo dimensions by equations 1 and 2 and those derived by equation 4 are compared with volumes determined by angiography. The correlation coefficients are high and similar for both methods; however, the volumes obtained by the use of equations 1 and 2 are closer to the line of identity and the standard error of the estimate is lower. Examination of figures 6 to 8 reveals that volumes calculated by equation 4 are relatively accurate in smaller-volume chambers but are overestimated with increasing chamber size.

Figure 9 depicts the relationship between ejection fraction derived by the echocardiographic methods and that determined by angiography. Ejection fraction is accurately predicted by both methods, although the correlation coefficient is slightly better for

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*We used the methods of Wherry\textsuperscript{20} and Olkin and Pratt\textsuperscript{21} to determine whether a reduction or shrinkage in the correlation between predicted and observed values would occur when equations 1, 2, and 3 were applied to new data. The r\textsuperscript{2} estimated by these methods did not significantly differ from the observed r\textsuperscript{2} for each of the three equations, indicating that little shrinkage would occur.

Circulation, Volume XLIV, October 1971
Figure 6

Angiographic end-systolic volume (ESV) plotted against end-systolic volume determined by equation 1 (ESV′—solid circles) and end-systolic volume determined by equation 4 (ESV″—x’s). Solid line is the line of identity. R = correlation coefficient; SEE = standard error of the estimate.

Figure 7

Angiographic end-diastolic volume (EDV) plotted against end-diastolic volume determined by equation 2 (EDV′—solid circles) and end-diastolic volume determined by equation 4 (EDV″—x’s). Solid line is the line of identity. R = correlation coefficient; SEE = standard error of the estimate.

Figure 8

Angiographic stroke volume (SV) plotted against stroke volume determined from equations 1 and 2 (SV′—solid circles) and stroke volume determined from equation 4 (SV″—x’s). Solid line is the line of identity. R = correlation coefficient; SEE = standard error of the estimate.

Figure 9

Left ventricular ejection fraction determined by angiography (EF) plotted against ejection fraction determined from equations 1 and 2 (EF′—solid circles) and from equation 4 (EF″—x’s). Solid line is the line of identity. R = correlation coefficient; SEE = standard error of the estimate.
EF". The standard errors of the two methods are, however, the same.

Discussion

A study recently completed in our laboratory of the geometry of left ventricular contraction in the normal and pathologic human heart by analysis of biplane angiocardiograms provided the theoretic background for the present investigations.11-12 This study demonstrated that in the normal heart 80% of left ventricular stroke volume could be accounted for by changes in the minor ventricular axis during systole; shortening of the long or major ventricular axis was responsible for the remaining 20%. In pathologic conditions such as mitral stenosis or chronic left ventricular volume overload, there was a slight increase in the contribution of minor-axis shortening to stroke volume (86%), whereas in ventricles with compensated pressure overload or decompensated states the minor-axis contribution resembled that of the normal situation. In each of these conditions the relative amount of change in the minor ventricular axis with systole exceeded the relative change in the major axis. Other investigators have made similar observations in human subjects utilizing radiologic methods.9,22 An additional observation relevant to the present investigation was that the percentage change in left ventricular minor-axis dimensions with systole correlated closely and in a linear manner with the percentage change in ventricular volume with systole, that is, the left ventricular ejection fraction. These findings in the human heart are in accord with the earlier work of Rushmer and others,23-26 who showed by a variety of techniques that in the canine left ventricle ejection is accomplished primarily by shortening in the minor ventricular axis. Recently, other investigators employing different techniques have confirmed these findings in the dog.27-30 These observations suggested that a great deal of information about left ventricular volume and its change during the cardiac cycle could be derived from a consideration of the minor axis of the chamber alone.

Popp and co-workers13,14 have developed the technique that was employed in the present studies of noninvasively measuring the minor ventricular axis by the use of reflected ultrasound. The method is based on the principle that an ultrasonic signal when passed through the body is reflected backward or echoed when tissues of differing acoustic impedance are encountered, such as heart muscle and blood. A single transducer which is applied to the chest wall both transmits the ultrasonic signal and receives the resultant reflected echoes. By proper manipulation of the transducer, the ultrasonic beam can be directed to a plane in which the beam passes through and echoes from both the interventricular septum and the posterior left ventricular wall. Since the speed of sound in tissue is known, the distance of echoed structures from the transducer can be calculated and the distance between two echoed structures, such as the septum and the posterior left ventricular wall, determined.

The left ventricular dimension measured by this technique may not be a true minor axis of the chamber because of the relationship of the interventricular septum and left ventricular cavity to the chest wall. This relationship varies with differing pathologic states. In many instances the echo method will probably measure a somewhat oblique axis of the left ventricular chamber, similar to that which is illustrated schematically in figure 1. In spite of this, we have found in the present investigations good correlations between the minor axes determined by the echo technique in systole and diastole and those derived from left ventricular angiocardiograms. Further, the regression equations derived from these relationships are close to the line of identity, indicating that the echo-measured axes closely resemble the angiographic axes over the wide range of ventricular dimensions found in our study. The relative change in the minor axis with systole also showed a significant correlation between the two methods, although there was a regular underestimation of this value by the echo method. This occurred because of a small overestimation of end-systolic diameter.
and underestimation of end-diastolic diameter by the echo method. Alternatively, it is possible that the end-systolic dimension derived from angiograms may be an underestimation of the true minor axis because of difficulties in determining end-systolic area from angiograms.

We also found highly significant correlations between the end-diastolic and end-systolic echo dimensions and end-diastolic and end-systolic angiographic volumes. Our results are similar to those reported by Murray and co-workers. The high correlations between dimensions and volumes occur in spite of a great diversity of patient types, chamber sizes, and ejection fractions in the patient population studied.

As predicted from the angiographic analysis of the geometry of ventricular contraction, there was a significant correlation between the relative change in the minor axis (ΔΔS revision) and the ejection fraction. In only a single patient studied (no. 27) was there a marked disparity between these two variables. This patient had been in congestive heart failure following a myocardial infarction and had mitral regurgitation thought to be secondary to papillary muscle dysfunction. The ΔΔS echo was normal, but the EF was reduced. The explanation for this may be that the echo beam was directed through septal and left ventricular wall muscle which had not been involved with the infarction and thus was contracting normally or perhaps supranormally to counteract the hemodynamic burden of noncontractile scar tissue. It is possible that the echo technique, which samples motion in only a single portion of the septum and posterior left ventricular wall, may not accurately reflect ejection fraction where localized disorders of myocardial contractility are present, but further studies on this question are needed.

The excellent linear correlation between echo dimensions and ventricular volumes found here allows prediction of ventricular volumes from the echo dimensions alone by the use of regression equations derived from these echo-angio correlations. As can be seen in figures 6, 7, and 8, volumes determined by these equations resemble angiographic volumes over the entire range of volumes encountered. We found that equation 4, which utilizes a cube function of the minor axis to compute volume, allowed accurate prediction of volumes in smaller hearts but resulted in a progressive overestimation of volume with increasing chamber size. In spite of this, the correlation coefficient remained high for this method when compared with the angiographic method, as noted by others. Popp and Harrison validated the use of equation 4 in patients with relatively normal-sized ventricles and low to normal stroke volumes. The assumptions required to use a cube function of the minor axis to determine volume in this manner are: (1) that the echo-measured minor axis is a true minor axis; (2) that the minor axes in two planes are equal; and (3) that the long axis of the chamber equals twice the minor axis. The first assumption is validated by the present studies, the second by the previous work of Sandler and Dodge.

The last assumption applies well at end-diastole in conditions in which the ventricular volume is normal (normal subjects, mitral stenosis, compensated pressure overload), but does not hold true in large-volume chambers (compensated volume overload, decompensated hearts) in which the ventricle becomes more spherical with a decrease in long axis/short axis ratio to less than 2. Failure to account for this change in chamber geometry with increasing heart volume would therefore theoretically result in a progressive overestimation of volume in larger-volume hearts as has been shown in the present study. Such a problem does not occur with the use of regression equations 1 and 2, which permit accurate prediction of volumes even in very large chambers.

The prediction of ejection fraction, which is based on the relationship of ΔΔS echo and EF, is accurate by either method. These investigations further validate the use of echocardiographic minor-axis dimensions as correlates of left ventricular volumes. The
LV VOLUMES BY ULTRASOUND

583

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