Evaluation of Left Ventricular Function by Echocardiography

By Nicholas J. Fortuin, M.D., William P. Hood, Jr., M.D., and Ernest Craig, M.D.

SUMMARY

Ventricular minor-axis dimensions were measured by echocardiography at end-systole ($S_\text{s}$) and end-diastole ($S_\text{d}$) in five groups of patients: (I) normal; (II) mitral stenosis; (III) compensated volume overload; (IV) idiopathic hypertrophic subaortic stenosis; and (V) congestive heart failure. Cardiac pump function was evaluated by determination of left ventricular volumes and ejection fraction (EF) from the echographic dimensions using formulae previously reported. The mean velocity of circumferential fiber shortening ($V_{\text{CF}}$), a parameter of cardiac muscle performance previously obtained only by invasive methods, was determined from the echographic dimensions by the formula: \[
\frac{(S_\text{d} - S_\text{s})}{S_\text{d}} \times \frac{1}{dt}.
\]

The duration of minor-axis shortening (dt) was measured directly from the echocardiogram.

Dimension and volume measurements in these groups of patients were similar to those reported in similar patients determined by angiographic methods. Measurement of the relative changes in echographic dimensions with systole ($\% \Delta S$), EF, and $V_{\text{CF}}$ allowed separation of patients with clinical heart failure (group V) from normal subjects (group I). Patients in group II had reduced values for these parameters compared to group I. Those in III did not differ significantly, and those in IV had increased values. In general, $V_{\text{CF}}$, EF, and $\% \Delta S$ showed similar trends, but individual patients sometimes differed. The findings reported here demonstrate the feasibility of evaluating ventricular function by a noninvasive method in a large group of ambulatory patients.

Additional Indexing Words:

Left ventricular volumes Ejection fraction Circumferential shortening velocity
Ventricular minor-axis dimensions Noninvasive technics

Recent interest in assessing cardiac function has resulted from advances in understanding of the physiology of cardiac muscle contraction as well as from improved therapeutic technics, most notably surgical, for treating patients with valvular or congenital heart disease.1,2 The use of intravascular pressure recordings and left ventricular angiography or other methods to estimate chamber volume and dimensions has allowed characterization of cardiac muscle function in patients undergoing cardiac catheterization.3-6 The impracticality of doing these studies routinely, repeatedly, or in acutely ill patients has promoted a search for bedside or noninvasive methodology for quantitation of cardiac function. Recently, the noninvasive technic of ultrasonic echocardiography has been developed to provide quantitative information about ventricular performance by derivation

From the Bio-Medical Research Branch, Division of Effects Research, Environmental Protection Agency, Department of Medicine, School of Medicine, University of North Carolina, and the C. V. Richardson Laboratory, North Carolina Memorial Hospital, Chapel Hill, North Carolina.

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Address for reprints: Dr. Nicholas J. Fortuin, Department of Medicine, The Johns Hopkins Hospital, Baltimore, Maryland 21205.

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of ventricular volumes and ejection fraction from echo-measured ventricular minor-axis dimensions.\textsuperscript{7–10} The ejection fraction, which is a measure of the pump performance of the heart and thus only indirectly related to mechanical muscle function, has nonetheless been widely utilized as an indicator of myocardial performance.\textsuperscript{11, 12} In addition to this use of echo-measured dimensions, we describe in this report another parameter of ventricular function which can be determined by the echo technique, the mean velocity of circumferential fiber shortening ($V_{CF}$). This parameter, which has previously been determined by invasive methods, is related to cardiac muscle function directly and thus may provide information about ventricular performance which cannot be obtained from a consideration of ejection fraction alone. The purposes of this report are to describe a method for determining $V_{CF}$ by echocardiography and to document the utility with which $V_{CF}$ and echo-measured ventricular dimensions and derived volumes can aid in the clinical assessment of ventricular pump and muscle performance in ambulatory patients with cardiac lesions.

Methods

Patients

Subjects were patients attending the General Cardiac Clinic of the North Carolina Memorial Hospital. They were divided into five separate groups:

- **Group I**, normal subjects (10 subjects). These were all under the age of 30 years without evidence of cardiac disease or hypertension.

- **Group II**, mitral stenosis (13 patients). This was defined by the usual clinical criteria and confirmed by cardiac catheterization in 10. In all patients mitral stenosis was the predominant lesion, other valvular lesions being graded as trivial when present. Atrial fibrillation was present in seven of these patients.

- **Group III**, compensated volume overload (20 patients). There were 11 patients with mitral regurgitation, seven with aortic regurgitation, one with a ventricular septal defect, and one with a patent ductus arteriosus in this group. All patients had cardiomegaly on chest X-ray. They were generally young patients (mean age 26.3 years) who had not exhibited clinical signs or symptoms of congestive heart failure. In many the magnitude of the volume overload was felt to be great on clinical grounds, but surgery had not been performed because of the young age and absence of symptoms. The magnitude of volume overload in this group is reflected in the mean end-diastolic chamber diameter which was 39\% larger than that of group I (see below and Table 1).

- **Group IV**, idiopathic hypertrophic subaortic stenosis (five patients). These five patients exhibited typical physical findings of this disorder, and each had the diagnosis confirmed by cardiac catheterization. In one, a gradient across the left ventricular outflow tract was not recorded but a markedly hypertrophied left ventricular wall and hyperdynamic cardiac contraction were demonstrated by angiography which, when considered with the clinical findings, suggested the diagnosis of hypertrophic cardiomyopathy.

- **Group V**, congestive heart failure (17 patients). This group was defined by the presence of symptoms and signs of circulatory congestion with cardiac enlargement in the absence of evidence of obstruction or restriction to ventricular filling. Six patients had primary myocardial disease, five had hypertensive arteriosclerotic disease, and six patients had valvular heart disease.

Echocardiographic Technic

A Smith-Kline Ekoline 20 Ultrasonoscope with 2.25-MHz transducer of 1.9-cm (0.75-in) diameter was employed in these studies. The technic for obtaining ventricular minor-axis measurements by echocardiography has been described in detail elsewhere.\textsuperscript{7–9} In brief, the ultrasonic transducer is placed in the fourth or fifth intercostal space just to the left of the sternum. The ultrasonic beam is directed posteriorly and medially until the characteristic motion of the anterior leaflet of the mitral valve is detected. The beam is then directed slightly laterally and inferiorly away from the mitral valve until a plane is found in which motion of the interventricular septum and posterior left ventricular wall is noted. Motion of these structures as a function of time is displayed on an oscilloscope, and a time exposure on Polaroid film is taken of a full oscilloscopic screen sweep with distance represented on the vertical axis and time on the horizontal axis (Fig. 1). End-diastolic ($S_D$) diameter is measured at the peak of the R wave of the simultaneously displayed electrocardiogram, and end-systolic ($S_S$) diameter at the time of maximal anterior movement of the posterior left ventricular wall. In patients with atrial fibrillation, the mean of five determinations of $S_D$ and $S_S$ was used. The relative change in the minor axis with systole ($\% \Delta S$) is determined by

$$\frac{S_D - S_S}{S_D},$$
Derivation of Ventricular Volumes

We used equations previously reported to derive end-diastolic (EDV) and end-systolic (ESV) volumes from the echo dimensions:

\[ \text{EDV} = 59S_D - 153 \]
\[ \text{EDV} = 47S_S - 120 \]

Stroke volume (SV) was determined by subtracting ESV from EDV and ejection fraction (EF) by dividing SV by EDV.

Derivation of Mean Velocity of Circumferential Fiber Shortening (Mean \( V_{\text{CF}} \))

We assumed in these studies that the minor axis measured by echocardiography was equal to the true minor axis of an ellipsoid of revolution in which minor axes in all planes are equal. Therefore, the internal circumference at end-diastole \( (C_D) \) can be determined by:

\[ C_D = \pi S_D \]

and at end-systole \( (C_S) \):

\[ C_S = \pi S_S \]

The mean velocity of circumferential shortening \( (V_{\text{CF}}) \) in centimeters per second (cm/sec) is equal to the change in circumference during systole divided by the duration of shortening:

\[ V_{\text{CF}} = \frac{dC}{dt} = \frac{C_D - C_S}{dt} = \frac{\pi(S_D - S_S)}{dt} \]

The duration of shortening \( (dt) \) can be determined directly from the echocardiogram as illustrated in figure 1 by measuring the time from initial anterior movement of the posterior wall to the peak of anterior-wall motion. Since \( V_{\text{CF}} \) is affected by heart size, we normalized for this by dividing by \( C_D \) to obtain a value of mean velocity of shortening in circumferences per second (circ/sec).

\[ V_{\text{CF}} = \frac{(S_D - S_S)/dt)}{\pi S_D} = \frac{(S_D - S_S)/dt}{S_D} \]

Student's \( t \) test was used to compare the mean values obtained in each group with those of group I.

Figure 1

Echocardiogram obtained from a patient with mitral insufficiency to demonstrate technic for measuring minor-axis dimensions \((S_D \text{ and } S_S)\) and duration of minor-axis shortening \((dt)\).
Table 1

Mean Values of Echo-Measured Dimensions, Derived Volumes, Circumferences, and Shortening Velocities in Patient Groups

<table>
<thead>
<tr>
<th>Pt group*</th>
<th>Age (yr)</th>
<th>No.</th>
<th>SP (cm)</th>
<th>SQ (cm)</th>
<th>% Δ S</th>
<th>ESV (ml)</th>
<th>EDV (ml)</th>
<th>SV (ml)</th>
<th>EF (%)</th>
<th>CT (cm)</th>
<th>CS (cm)</th>
<th>dt (msec)</th>
<th>VCF (cm/sec)</th>
</tr>
</thead>
<tbody>
<tr>
<td>I. Normal</td>
<td>22.4</td>
<td>10</td>
<td>5.0</td>
<td>3.8</td>
<td>23.7</td>
<td>59</td>
<td>142</td>
<td>83</td>
<td>59</td>
<td>15.8</td>
<td>12.0</td>
<td>268</td>
<td>0.92</td>
</tr>
<tr>
<td></td>
<td>±0.41</td>
<td></td>
<td>±0.36</td>
<td>±4.0</td>
<td>±17</td>
<td>±24</td>
<td>±14</td>
<td>±7</td>
<td>±1.3</td>
<td>±1.1</td>
<td>±31</td>
<td>±0.15</td>
<td></td>
</tr>
<tr>
<td>II. Mitral stenosis</td>
<td>41.8</td>
<td>13</td>
<td>4.9</td>
<td>3.9</td>
<td>20</td>
<td>66</td>
<td>138</td>
<td>73</td>
<td>53</td>
<td>15.5</td>
<td>11.6</td>
<td>278</td>
<td>0.73</td>
</tr>
<tr>
<td></td>
<td>±0.36</td>
<td></td>
<td>±0.38</td>
<td>±4.4</td>
<td>±18</td>
<td>±21</td>
<td>±13</td>
<td>±8.4</td>
<td>±1.1</td>
<td>±2.9</td>
<td>±44</td>
<td>±0.18</td>
<td></td>
</tr>
<tr>
<td>P</td>
<td>NS</td>
<td></td>
<td>&lt;0.05</td>
<td>NS</td>
<td>&lt;0.05</td>
<td>NS</td>
<td>&lt;0.05</td>
<td>&lt;0.05</td>
<td>NS</td>
<td>NS</td>
<td>&lt;0.01</td>
<td></td>
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</tr>
<tr>
<td>III. Comp vol overload</td>
<td>26.3</td>
<td>20</td>
<td>6.06</td>
<td>5.2</td>
<td>26</td>
<td>122</td>
<td>258</td>
<td>135</td>
<td>54</td>
<td>21.8</td>
<td>16.2</td>
<td>298</td>
<td>0.87</td>
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<tr>
<td>P</td>
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<td>NS</td>
<td>&lt;0.001</td>
<td>NS</td>
<td>&lt;0.001</td>
<td>&lt;0.001</td>
<td>NS</td>
<td>&lt;0.001</td>
<td>&lt;0.025</td>
<td>NS</td>
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<tr>
<td>IV. IHSS</td>
<td>42.2</td>
<td>5</td>
<td>5.9</td>
<td>3.8</td>
<td>35</td>
<td>60</td>
<td>192</td>
<td>132</td>
<td>70</td>
<td>18.3</td>
<td>120</td>
<td>267</td>
<td>1.27</td>
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<tr>
<td></td>
<td>±0.74</td>
<td></td>
<td>±0.61</td>
<td>±6.5</td>
<td>±29</td>
<td>±43</td>
<td>±28</td>
<td>±9.9</td>
<td>±2.2</td>
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<tr>
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<td></td>
<td>NS</td>
<td>&lt;0.001</td>
<td>NS</td>
<td>&lt;0.005</td>
<td>&lt;0.001</td>
<td>&lt;0.025</td>
<td>&lt;0.005</td>
<td>NS</td>
<td>&lt;0.005</td>
<td></td>
<td></td>
</tr>
<tr>
<td>V. CHF</td>
<td>49.7</td>
<td>17</td>
<td>7.4</td>
<td>6.5</td>
<td>11.8</td>
<td>185</td>
<td>281</td>
<td>96</td>
<td>35</td>
<td>23.1</td>
<td>20.4</td>
<td>249</td>
<td>0.49</td>
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<tr>
<td></td>
<td>±1.0</td>
<td></td>
<td>±1.0</td>
<td>±3.5</td>
<td>±48</td>
<td>±61</td>
<td>±18</td>
<td>±4.8</td>
<td>±3.3</td>
<td>±3.2</td>
<td>±37</td>
<td>±0.17</td>
<td></td>
</tr>
<tr>
<td>P</td>
<td>&lt;0.001</td>
<td></td>
<td>&lt;0.001</td>
<td>&lt;0.001</td>
<td>&lt;0.001</td>
<td>&lt;0.001</td>
<td>&lt;0.001</td>
<td>&lt;0.001</td>
<td>&lt;0.001</td>
<td>&lt;0.001</td>
<td>&lt;0.001</td>
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</tbody>
</table>

Abbreviations: As in text.
*P values refer to differences from group I.
Results

The mean values (±sd) for minor-axis dimensions, circumferences, left ventricular volumes and ejection fraction, the duration of minor-axis shortening, and circumferential shortening velocities of each group are presented in table 1.

Minor-Axis Dimensions

Patients with mitral stenosis (group II) did not show significant differences in minor-axis dimensions in systole or diastole compared to normal subjects. All other groups had larger $S_D$ than normal subjects, and groups III and V had increased $S_S$. Values for $S_D$ in each patient are plotted in figure 2. The smallest chamber dimensions at end-diastole were found in groups I and II, while groups III and V had the largest values. Only one patient in group V and two in group III overlapped with values for group I.

Values of $\%\Delta S$ from individual patients with group means and standard deviations are shown in figure 3. Groups II and V had significantly smaller $\%\Delta S$ than group I, although the difference was much greater for group V. Group IV showed a markedly increased $\%\Delta S$, while group III did not differ significantly from group I. Two patients in
group III had very high values (43 and 46%); both had mitral regurgitation. No patient in this group had a value below the lowest value in the normal group of 19%. Only one patient in group V had a value this high, the remainder being well below this figure.

**Ventricular Volumes**

End-diastolic volume and ESV, which are derived from $S_D$ and $S_S$, showed differences between groups similar to those found for the minor-axis dimensions alone. Stroke volume was significantly less than normal in group II, but significantly greater than normal in the other groups. In figure 4, SV is plotted against EDV. The regression line that is plotted is determined from groups I and III, subjects with presumed normal ventricular function, and reveals a good correlation between these two variables ($P < 0.001$). All patients in group V fall below the regression line, indicating an inappropriately small SV for the degree of increase in EDV. Another expression of this relationship is the LVEF which is shown for each patient and group in figure 5.

EF was reduced in groups II and V, increased in group IV, and not different from normal in group III. The range of EF in group I was 51–72% and in group III, 43–91%. Eleven patients in group III had EFs below 51%, the lowest value found in group I. This decrease in EF from normal in subjects with presumed normal ventricular function and normal $\% S$ may be explained in some by a slight underestimation of SV by the echo technic in larger volume chambers. All subjects in group V had values for EF below 45%.

**Circumferential Shortening Velocities**

$C_D$ and $C_S$, which are derived from $S_D$ and $S_S$, showed differences among groups similar to those for minor-axis dimensions. The duration of left ventricular circumferential shortening ($dt$) was significantly longer than normal only in group III. Group V showed a diminished value for $dt$. Values for $V_{CF}$ (figure 6) in group III were not significantly different from normal, in group II were only slightly decreased from normal, and in group V were markedly diminished. A high $V_{CF}$ was seen in group IV. Two patients in group V overlapped with the normal group. Both had valvular heart disease, and in one the valvular

![Figure 5](image1.jpg)

*Figure 5*

Values for ejection fraction (EF) in patient groups.

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![Figure 6](image2.jpg)

*Figure 6*

Values for mean circumferential shortening velocity ($V_{CF}$) in patient groups.
lesion was relatively acute (aortic regurgitation secondary to infective endocarditis without known previous heart disease). While both these subjects had circulatory congestion as a result of overwhelming volume loads to the left ventricle, the normal value for Vcr suggests that their cardiac muscle function was quite adequate. One of these patients has had an excellent result from aortic valve replacement; the other has not yet had surgery. Several patients in group III fell below the range of normal subjects and thus, though asymptomatic, may have abnormal left ventricular function.

**Discussion**

In a previous investigation, we attempted to validate the use of echocardiographic-measured left ventricular minor-axis dimensions as correlates of left ventricular volumes.9 We found that echo dimensions measured at end-systole and end-diastole were good approximations of these dimensions determined from biplane angiograms, that the echo dimensions correlated well with left ventricular volumes, and that ventricular volumes and ejection fraction could be determined from echo-measured dimensions alone.9 In the present study, we have measured left ventricular dimensions by echocardiography in groups of patients with cardiovascular lesions to demonstrate how this technic allows characterization of ventricular performance. The information provided by the echo technic in this study falls into three areas, all of which are useful in evaluating left ventricular function: (1) an index of end-diastolic left ventricular chamber size, i.e. the Sd and parameters derived from this, Cd and EDV; (2) estimates of left ventricular pump performance, i.e. %ΔS and EF, and of pump output, SV; and (3) an index of left ventricular muscle performance, i.e. mean Vcr.

The data obtained for the five patient groups in each of these three areas are similar to data obtained from such groups by invasive technics. The values for Sd, Cd, and EDV found in our normal subjects compare favorably with values for these parameters obtained by angiographic technics. In the study of Gault et al.4 in which single-plane cineangiography was employed, the mean end-diastolic circumference in six normal subjects was 14.5 cm, compared with our value of 15.8 cm. The mean values for minor-axis dimensions obtained by biplane angiography in our laboratory in normal subjects are similar to those obtained by echocardiography. Similarly, the mean value for EDV in normal subjects by the echo technic, 142 ml, is the same as that previously obtained in six normal subjects in our laboratory by biplane angiography (140 ml),13 but slightly greater than the value of 125 ml reported by Kennedy et al. using a similar method.14 Comparison of groups with respect to the parameters of end-diastolic chamber size likewise reveals trends which are similar to those found with angiographic volume technics. Thus, patients with mitral stenosis did not differ significantly from normal, while all other groups showed an increase in end-diastolic size.13, 15

The relative change in minor-axis dimensions or circumference with systole (%ΔS) with the echo technic is somewhat less than that determined by angiographic methods. In our laboratory the mean change in normal subjects studied by biplane angiography was 33%,16 a value slightly less than that reported by Gault et al.4 in normal subjects, but higher than the mean value of 23.7% obtained in the present studies. The underestimation of %ΔS by the echo method probably results from a slight underestimation of Sd, and overestimation of Sb.9 In spite of this the %ΔS allowed excellent separation of patients with clinically normal ventricular function (groups I and III) from those with obvious heart failure (group V) and provided information about patient groups which is similar to that provided by the left ventricular EF. This similarity no doubt results from the close linear correlation between %ΔS and EF which has been observed in angiographic volume studies.16, 17

Since ventricular volumes were derived from minor-axis dimensions alone in this study, EF, like %ΔS, was slightly underestimated. Several groups have reported the mean

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value of EF for normal subjects to be 67%,13, 14, 18 while the mean value for normal subjects in this study was 59%. No subject in the normal group had a value below 51%, although several patients in group III had values below this figure. There is a tendency for a small underestimation of SV, particularly in larger volume chambers by the methods employed here,9 and this may be in part responsible for the lower values for EF noted in some subjects in group III. Some of the subjects may have had borderline ventricular function. It is noteworthy that the calculation of EF afforded no advantage in separation of individual patients or patient groups than that obtained by the use of %ΔS alone. Its main advantage results from the more general familiarity with the use of this fraction.

The accuracy with which the echo technic can compute left ventricular stroke volume has been discussed in previous reports.7-10 In this study, each patient group differed significantly from normal with respect to stroke volume. Subjects with mitral stenosis had a reduced value, and all other groups had an increase. In group V this was the result of inclusion of subjects with decompensated volume overload, in whom the stroke volume is increased but still inadequate for the demands on the circulation. Determination of SV by the echo technic allows description of the pump performance of the heart by the calculation of EF or by position on the classical ventricular function curve in which SV is related to EDV (fig. 4). It is also useful in estimating the amount of volume overload in patients with left-to-right shunts or valvular regurgitation if the forward SV is determined simultaneously or if a certain forward stroke volume is assumed.7

Studies in isolated cardiac muscle and the intact heart in experimental animals have established the utility of describing cardiac performance in terms of the mechanical properties of cardiac muscle exclusive of the pump performance.19 The inverse relationship between force and velocity observed in isolated cardiac muscle20 and confirmed in the intact heart of animals21, 22 and more recently humans4, 23, 24 provides a basis for characterization of cardiac muscle function. In this study only noninvasive methods were employed, and no measure of ventricular force determined. However, since wall forces or stress tend to be normalized in chronic heart disease by ventricular hypertrophy, ventricular performance can be evaluated by a consideration of velocity alone.13, 23 The velocity of contractile element shortening and its extrapolation to zero load, Vmax, have been used as indices of contractility, but the calculation of these velocities requires high-quality angiographic studies and high-fidelity intraventricular pressure tracings in addition to assumptions about the coefficient of elasticity of the series elastic element of heart muscle.3, 6, 19 The peak velocity of circumferential fiber shortening can be determined from high-speed angiocardiograms and has been shown to be a useful parameter of myocardial contractility.5 Mean Vcf, as computed in this study, would be expected to be a less sensitive measure of contractility than peak Vcf. However, earlier studies in which mean Vcf was computed by thermodilution technic showed that this parameter allowed good separation of normal from abnormal ventricular function,25 and in the study of Gault et al.4 mean Vcf of the internal circumference provided excellent discrimination between failing and nonfailing ventricles. Subsequent study in additional patients by this group has confirmed this finding.26

In this study we have described a method whereby mean Vcf can be determined by a noninvasive technic. The method must be further validated by comparing echo-determined Vcf with Vcf measured by angiography or thermodilution. To compute mean Vcf by the echo technic, we have assumed that the left ventricular diameter measured is equal to the true minor-axis diameter of the ventricle and further that the minor axis is equal in all planes. Our previous study in which echo dimensions were compared to angiographic dimensions established that the echo dimension is a good approximation of the angiographic dimension.9 Other workers have shown

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that minor-axis dimensions determined from angiocardiograms in AP and lateral planes do not differ by more than 10%.27

The value of mean VeF obtained by this technic in normal subjects is somewhat less than that reported by Gault et al.4 Since the C0, reported by these workers did not differ greatly from our value, the difference in VeF may be a reflection of overestimation of C0 by the angiographic technic. Our values are similar to those obtained by thermodilution.25 The determination of VeF allowed excellent separation of patients with clinically abnormal ventricular function (group V) from clinically normal subjects (group I). Reduction of VeF in patients with heart failure probably results from both diminished myocardial contractility and the increased systolic wall forces or stress which tends to occur in decompensated states.13 In general, VeF provided the same separation of patients and groups as $\% A S$ and EF. This is not unexpected since the basic echographic measurements, $S_D$ and $S_S$, are involved in all three determinations. Ejection fraction and $\% A S$ are indicators of the extent of cardiac muscle shortening. VeF adds to this the dimension of time and in individual patients provides different information than EF or $\% A S$. Thus, two patients who had signs of circulatory congestion and who had diminished EF had values of VeF in the lower range of normal. It is possible that muscle function in these patients is better than suggested by a consideration of EF alone. Conversely, several patients with compensated volume overload had values of mean VeF which were much reduced from normal and groups as VeF and EF. These patients, though asymptomatic, may have impaired myocardial function. The highest values for VeF were found in two young patients with mitral regurgitation. Excessive VeF in these patients may be due to reduced impedance to ejection as has been demonstrated in experimental animals.28

The increased VeF observed in patients with IHSS in this study can be explained by the inverse force-velocity relationship. In this group of patients ventricular hypertrophy does not occur in response to a chronic hemodynamic stress and thus is inappropriately large in relation to hemodynamic burden, and systolic wall stresses are reduced below normal.13 The reduced wall force or stress allows shortening of individual circumferential fibers against a reduced load and therefore increased velocity of fiber shortening. The presence of mitral regurgitation would also be expected to increase VeF by reducing impedance to ejection.28 The excessive EF and $\% A S$ which we have noted has also been described in angiographic volume studies.29,30 The increased VeF, EF, and $\% A S$ demonstrable by echocardiography provide additional information to aid in the sometimes difficult diagnosis of this condition.

The present investigation is an extension of previous work in which we validated the use of echo-measured dimensions as approximations of left ventricular minor-axis dimensions and correlates of left ventricular volumes.9 In this study we have shown how it is possible to estimate the velocity as well as the extent of fiber shortening with this method. The information about left ventricular function provided by the echo technic in this study has heretofore been obtained only by invasive methods. Most of the subjects included in this study were outpatients in whom the echo study could be accomplished painlessly, at low cost, repeatedly, and without danger to the patient. The sensitivity of this method in assessing left ventricular function is as yet undetermined. Further studies in which data obtained by the echo technic are compared with parameters of ventricular function obtained by invasive methodology are needed to answer this important question.

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