Analysis of Left Ventricular Wall Motion by Reflected Ultrasound

Application to Assessment of Myocardial Function

By Ian G. McDonald, M.D., Harvey Fegenbaum, M.D.,
and Sonia Chang, B.S.

SUMMARY
Ultrasound echocardiograms from the septal and posterior left ventricular walls were displayed with a simultaneously recorded electrocardiogram, phonocardiogram, and indirect carotid pulse. These echoes differed in both amplitude and waveform. The contour of the posterior wall echo resembled an inverted ventricular volume curve, while the septal echo was of smaller amplitude and had a characteristic notched appearance. Most of the movement of the left ventricular walls relative to the ultrasound transducer was attributable to systolic contraction and diastolic expansion of the cavity. However, superimposed on this motion due to change in cavity size was movement of the left ventricle as a whole, first anteriorly toward the ultrasound transducer during late systole then posteriorly away from it at the beginning of left ventricular relaxation. These movements added to the amplitude of posterior wall motion but subtracted from the motion of the septum and were responsible for the notch in the waveform of this echo. Attachment superiorly to the aortic root might also have limited septal motion which was less near the base than nearer the apex of the left ventricle.

The internal left ventricular dimension measured by ultrasound was standardized by using the mitral valve as a landmark and by recording the motion of the left side of the interventricular septum and endocardial surface of the posterior left ventricular wall simultaneously. This measurement was reproducible. In normal subjects, the ultrasonic dimension measured 4.40 ± 0.28 cm at the beginning of systole and shortened by 35.5 ± 3.9% at a rate of 1.22 ± 0.31 lengths/sec. By contrast, the average figures for six patients with primary myocardial disease were 6.96 ± 0.43 cm, 14.9 ± 4.2%, and 0.64 ± 0.11 lengths/sec. Calculation of such indices of left ventricular size and of rate and extent of myocardial shortening should be useful in the detection of impaired myocardial function and in following its progress.

Additional Indexing Words: Left ventricular geometry Left ventricular movements Myocardial function

The CLINICAL application of external ultrasound to examination of the heart began with the discovery of the echo from the anterior leaflet of the mitral valve by Edler and Hertz and with the use of the technic to assess the severity of mitral stenosis. Edler subsequently described the echoes arising from the tricuspid valve, left ventricular outflow tract, aortic valve, and pulmonary artery; he also recognized the abnormal

From St. Vincent’s Hospital and University of Melbourne, Melbourne, Australia, and Indiana University Medical Center, Indianapolis, Indiana.

Supported in part by a Grant-in-Aid from the National Heart Foundation of Australia and by the Department of Medicine, University of Melbourne and St. Vincent’s Hospital.

Address for reprints: Dr. Ian G. McDonald, Cardiovascular Unit, St. Vincent’s Hospital, Victoria Parade, Fitzroy, 3065, Melbourne, Australia.

Received July 14, 1971; revision accepted for publication March 10, 1972.
appearances of right ventricular enlargement in patients with atrial septal defect, of left atrial myxoma, and of pericardial effusion. A more recent development has been the clear description of echoes arising from the left ventricular walls, which has stemmed from the studies of pericardial effusion by Feigenbaum and associates. The ability to record the motion of the walls of the left ventricle has led to the development of methods of measuring its volume and mural thickness. Despite these applications, no attempt has previously been made to explain the waveform of the echoes from the left ventricular walls in terms of the geometry and movements of the left ventricle during the cardiac cycle. Hence, this was the principal aim of the present study. The second aim was to use the ultrasonic left ventricular measurements to derive indices of myocardial function suitable for clinical assessment of the left ventricle.

Methods

Subjects Studied

Ultrasound studies were performed on 20 subjects with normal hearts, comprising 16 volunteers and four patients referred for cardiac evaluation but found to have no clinical evidence of heart disease. There were 11 males and nine females, with an average age of 27.0 years (range 13–50). Three additional patients were studied but excluded from analysis because it was not possible to obtain technically satisfactory simultaneous recordings of echoes from the left septum and posterior left ventricular wall endocardial surfaces. Two of these three subjects were healthy but one had a past history of bronchial asthma.

Six patients with primary myocardial disease were also studied for comparison with the normal subjects. The diagnosis previously had been confirmed by cardiac catheterization in two of them. In the remaining four patients, cardiac catheterization was not indicated since the diagnosis had been made clinically by the presence of left ventricular hypertrophy and failure in the absence of evidence of ischemic heart disease or of a cause of left ventricular overload. The myocardial disease was considered to be due to excessive alcohol consumption in two patients; there was a familial incidence of unexplained heart failure in two patients; and in the remaining two patients there were no clinical clues to the cause of myocardial disease. At the time of the study, the symptoms and signs indicated that four of these patients (patients 2, 3, 4, and 6) were in clinical class II (New York Heart Association), and the remaining two were in clinical class III. All but one of these patients (case 3) were receiving digitalis and diuretic therapy at the time of the ultrasound examination. The exception had been in nodal rhythm for several years, and despite the presence of severe myocardial disease his symptoms were not progressive.

Recording Equipment

The principle of medical ultrasound recording and of its application to cardiac investigation had been reviewed recently. The methods of study previously reported were slightly modified for the present investigation. Two 1.27-cm (0.5-in) 2.25-MHz transducers were used; one* was not focused and the other focused at 5 cm. No difference was noted in the waveform of the echoes obtained by these transducers. The output of the ultrasonoscope was displayed and recorded on a multichannel oscilloscopic recorder⁸ This method of recording had advantages over the conventional method of Polaroid photography of the screen of the ultrasonoscope, since echoes could be continuously recorded during systematic scanning of the heart by the ultrasound beam, allowing easier and more certain identification of the cardiac echoes; in addition, the electrocardiogram (lead II), the phonocardiogram (second left intercostal space), and the indirect carotid pulse could be recorded with the echogram.

Recording Technic

The subjects were studied initially in the semi-reclining posture with the trunk elevated to an angle of approximately 45°. The echoes from the interventricular septum were more easily obtained when the subject was rotated into the right oblique position. This maneuver usually increased the distance of left ventricular echoes from the transducer but there was no perceptible change in echo contour nor in the distance between the left ventricular walls.

The scanning procedure commenced with the transducer in the third or fourth left intercostal space near the left sternal edge. Figure 1 illustrates the waveform of echoes obtained from the heart when the ultrasound beam is passed along directions 1–4 in figure 2, which is a

---

*Smith Kline Instruments, Inc., Palo Alto, California.
†Aerotech Laboratories, Philadelphia, Pennsylvania.
‡Ekoline 20, Smith Kline Instruments, Inc., Palo Alto, California.
§Model DR8, Electronics for Medicine, White Plains, New York.
Ultrasound recordings illustrating the waveform of the echoes obtained when the ultrasound beam passes along directions 1 to 4 in figure 2. Direction 2 is used for the measurement of left ventricular dimension. Note the fragmentary mitral valve echoes. T is the echo from the face of the transducer. The electrocardiogram (EKG), indirect carotid pulse (CAR), and phonocardiogram (PHON) were recorded simultaneously. D is the ultrasound dimension, the distance between LS and PLV, measured at intervals of 25 msec. Time lines are at intervals of 100 msec. Original record (rapid writer) retouched for illustrative purposes.

Diagrammatic transverse section of the chest wall and left heart. The echo from the anterior leaflet of the mitral valve was first identified (fig. 1) usually by directing the ultrasound beam from the third or fourth left intercostal space in a dorsal, medial, and slight cephalic direction (fig. 2, direction 3). Rotation of the beam in a lateral and caudal direction (fig. 2, direction 2) displayed the echoes from the septum and posterior left ventricular wall (fig. 1), although slight modification of the beam direction and transducer position were generally necessary to obtain echoes of good quality from the endocardial surfaces of the left side of the interventricular septum and of the posterior left ventricular wall simultaneously. In fact, such a recording could only be obtained from a small area of precordium with the ultrasound beam directed in a specific direction which had to be found by trial and error during systematic scanning. A characteristic feature was the presence, immediately anterior to the posterior left ventricular wall endocardial echo, of an echo which changed into that of the posterior leaflet of the mitral valve during scanning. This echo was recorded in 18 of 20 normal subjects and in four of the six patients with cardiomyopathy. In the remaining two patients, the typical echo from the posterior leaflet of the mitral valve was immediately proximal to that of the endocardial surface of the posterior left ventricular wall. Inadvertent rotation of the ultrasound beam further in a caudal and lateral direction toward the left ventricular apex (fig. 2, direction 1) caused a reduction in left ventricular dimension, disappearance of the fragments of the mitral valve echoes, and an increase in the amplitude of septal wall motion relative to that of the posterior left ventricular wall (fig. 1). Even slight movement of the ultrasound beam in other directions resulted in loss of resolution of one or both of the left ventricular endocardial echoes. Echoes from the aortic root (fig. 1) were obtained by aiming the ultrasound beam in a dorsal, medial, and cephalic direction (fig. 2, direction 4) either from the same position on the chest wall from which the anterior mitral valve leaflet had been recorded, or from one intercostal space higher.
posterior left ventricular wall endocardial surfaces was facilitated by tracing these echoes separately and bringing the tracings close together (fig. 4).

The ultrasonic left ventricular dimension was measured only in recordings showing simultaneous resolution of echoes from the endocardial surfaces of both left ventricular walls as previously described (fig. 1, direction 2). The distance between left septal and posterior left ventricular wall endocardial surfaces was measured to the nearest 0.5 mm at 25-msec intervals and manually superimposed as a graph onto the photographic recordings of the ultrasound echoes and physiologic reference tracings (fig. 3). Measurements by two observers at the beginning and end of the study varied, on the average, by only 0.22 cm.

Analysis of Recordings

The phases of the cardiac cycle were marked on the photographic record as follows (fig. 3): The beginning of electrical systole was indicated by the initial deflection of the electrocardiogram. The first high-frequency vibration of the aortic component of the second sound was taken to be the end of ejection. The time interval between the aortic component of the second sound and the dichrotic notch of the indirect carotid pulse represented the sum of the delay in transmission of the arterial pulse wave from the aortic root to the carotid artery and time lag in the pulse recording device. Hence, correction of the systolic upstroke of the indirect carotid pulse tracing for these delays identified the onset of ejection. The “E-point” of the echo from the anterior mitral valve leaflet was also correlated with the other reference tracings in order to indicate the time of maximal opening of the mitral valve toward the ultrasound transducer. Comparison of the waveform of the echoes from the left septal and

Figure 2

Diagrammatic transverse section of the precordial chest wall (W) and sternum (S) showing the position of the ultrasound transducer (T), direction of the beam (1 to 4) and structures identified by characteristic ultrasound echoes: the left side of the interventricular septum (LS), endocardial surface of the posterior left ventricular wall (PLV), anterior mitral valve leaflet (AMV), posterior mitral valve leaflet (PMV), aortic root (AO), and left atrium (LA). Note that 4 is directed cephalically as well as medially and 1 and 2 caudally as well as laterally.

Figure 3

Left ventricular echoes recorded along direction 2 (fig. 2) in a normal subject. Abbreviations same as for figure 2. The typical contours of LS and PLV are shown. Ds is the dimension at the beginning of systole, De at the onset of ejection, and Ds at the end of ejection. ΔLS and ΔPLV are the contributions of the left septal and posterior left ventricular wall endocardial echoes, respectively, to the total shortening during systole ΔDs. Original record (rapid writer) retouched for illustrative purposes.
grams obtained is of thickness $D_d$, respectively.

Mean $(W_d)$ and systole dimension at the mean per shortening. Carotid pulse tracing10 during Shortening ultrasound transducer toward surface motion of the left ventricular wall endocardial echo resembled an inverted ventricular volume curve and had an average amplitude of motion of 1.15 cm between the beginning of systole and end of ejection. During preejection systole, there was either no movement relative to the ultrasound transducer or a slight movement either toward it or away from it. During ejection, this surface moved toward the ultrasound transducer and anterior chest wall at a rate which was initially rapid but which slowed progressively toward the end of ejection, reaching a position closest to the ultrasound transducer at the end of ejection. This position was usually maintained as a brief plateau, following which there was a rapid movement posteriorly and away from the transducer during opening of the mitral valve and early left ventricular filling, then a slow movement in the same direction during the remainder of diastole.

The waveform of the echo from the left side of the interventricular septum was of smaller amplitude (0.42 cm) and had a curious notched appearance. The cause of this notch became clearer when the waveform of the left septal and posterior wall endocardial echoes were compared by placing them close together (figs. 4, 5). During approximately the initial two thirds of ejection, as the left ventricle narrowed, the walls moved toward each other but in opposite directions with respect to the ultrasound transducer. However, during late systole both walls were moving toward the ultrasound transducer, a movement which was reversed as both walls moved together away from the transducer during early ventricular relaxation. Simultaneous movement of the septal and posterior left ventricular walls in the same direction was thought to represent movement of the left ventricle as a whole.

Figure 4

Tracings of the left septal (LS) and posterior left ventricular wall endocardial (PLV) echoes juxtaposed and compared with the standard left ventricular ultrasonic dimension D for the first six normal subjects studied. $d$ indicates the beginning of systole, $e$ the onset of ejection, $s$ the end of ejection, and $m$ the point of maximum anterior motion of the mitral valve echo during early diastole. The shaded areas represent intervals during which both left ventricular walls are moving in the same direction.

(5.0%) for the dimension at the beginning of systole and 0.13 cm (4.7%) for the dimension at the end of ejection. The extent of shortening of this dimension from its length at the beginning of systole ($D_{s1}$) to its length at the end of ejection ($D_{e1}$) was termed the total systolic shortening ($\Delta D_{s1}$). The individual contributions to $\Delta D_{s1}$ of the motion of the left septal wall away from the ultrasound transducer ($\Delta LS$) and of the motion of the posterior left ventricular endocardial surface toward it ($\Delta PLV$) were also measured. Shortening during ejection ($\Delta D_{e}$) was divided by the ejection time obtained from the indirect carotid pulse tracing10 to derive the mean rate of shortening. Since myocardial shortening is best expressed per unit length, total shortening and the mean rate of shortening were divided by the dimension at the beginning of systole to calculate the fractional shortening, $\Delta D_{s1}/D_{s1}$, and the fractional mean rate of shortening, $(\Delta D_{e}/ET)/D_{e}$, respectively. The left ventricular wall thickness7 was measured at the beginning of systole ($W_d$) and end of ejection ($W_e$). The duration of the preceding cardiac cycle was also measured.

Results

Waveform of Left Ventricular Echoes

Representative examples of echocardiograms obtained in normal subjects are shown in figures 1 and 3 and tracings of these in figure 4. Unless otherwise specified, the following description applies only to echoes recorded in the standard manner described for the measurement of left ventricular ultrasonic dimension (fig. 2, direction 2). The waveform of the left ventricular posterior wall endocardial echo resembled an inverted ventricular volume curve and had an average amplitude of motion of 1.15 cm between the beginning of systole and end of ejection.
The upper diagrams marked I, II, and III are analogous to figure 1. The black arrows indicate the motion of the septal and posterior left ventricular walls relative to the ultrasound transducer along direction 2. The solid arrows represent movements of the left ventricle described in an earlier study.\textsuperscript{15} The lower diagrams are tracings of the left septal and posterior wall endocardial echoes as in figure 4. See text for detailed analysis.

The amplitude of movement and the contour of the septal echo changed with alteration of the ultrasound beam. When the beam was directed in a dorsal, medial, and cephalic direction to record the maximum movement of the anterior mitral valve leaflet (fig. 2, direction 3) the amplitude of septal motion during systole was small, and the notch was more prominent (fig. 1). However, when the beam was directed further caudally and laterally from the direction used for the measurement of left ventricular dimension (fig. 2, direction 1), the amplitude of septal motion became greater and approximately equal to that of the posterior left ventricular wall (fig. 1).

Motion of the outer walls of the left ventricle was represented by the right septal and posterior wall epicardial echoes, which were of similar waveform but smaller amplitude than the inner walls.

**Dimension during the Cardiac Cycle**

During pre-ejection systole, changes in the left ventricular ultrasonic dimension were inconstant and small (figs. 3, 4). Shortening began abruptly with the onset of ejection, and the minimum length of the dimension was reached at the time of the aortic component of the second heart sound. During isovolumic relaxation, lengthening began either gradually or with a small notch, becoming more rapid as the mitral valve opened and rapid left ventricular filling began. During diastasis there was usually a slow increase in the left ventricular ultrasonic dimension, and a small expansion was sometimes observed during atrial systole.

**Quantitative Data**

In normal subjects, the standard ultrasonic left ventricular dimension at the beginning of systole measured $4.40 \pm 0.28$ cm and shortened by $1.57 \pm 0.22$ cm ($35.5 \pm 3.9\%$) during systole, at a mean rate during ejection of $5.35 \pm 0.64$ cm/sec ($1.22 \pm 0.31$ lengths/sec) (table 1). The anterior movement of the endocardial surface of the posterior left ventricular wall contributed $1.15 \pm 0.17$ cm toward the shortening of the left ventricular ultrasonic dimension, and posterior movement of the septal wall contributed $0.42 \pm 0.22$ cm. The thickness of the posterior left ventricular wall at the end of diastole averaged $0.90 \pm 0.14$ cm and increased by 64.4\% to its maximum thickness of $1.48 \pm 0.30$ cm at the end of ejection.

In patients with primary myocardial disease (table 2), the standard ultrasonic left ventricular dimension at the beginning of systole was $6.96 \pm 0.43$ cm and shortened by $1.03 \pm 0.27$ cm ($14.9 \pm 4.2\%$) during systole at a mean rate of $4.43 \pm 0.81$ cm/sec ($0.64 \pm 0.11$ lengths/sec). The left ventricular wall thickness at the end of diastole was $1.14 \pm 0.13$ cm, and this increased to $1.48 \pm 0.82$ cm (26.7\%) at the end of ejection. The characteristic increase in left ventricular dimension and reduction of fractional shortening can be seen by comparison with a normal subject in figure 6.

**Discussion**

Since proof of the identity of the echoes from the left ventricular walls is crucial to the validity of the present study, evidence will first be presented that the echoes described do arise from the left ventricular walls and that it
Table 1

Quantitative Data—Normal Subjects*

<table>
<thead>
<tr>
<th>No.</th>
<th>Age (yr)</th>
<th>Sex</th>
<th>Surface area (m²)</th>
<th>D₁ (cm)</th>
<th>D₂ (cm)</th>
<th>ΔD₁ (cm)</th>
<th>ΔD₁/D₁ (%)</th>
<th>ET (msec)</th>
<th>ΔD₂/ET (cm/sec)</th>
<th>W₁ (cm)</th>
<th>W₂ (cm)</th>
<th>R-R (msec)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>37</td>
<td>M</td>
<td>1.77</td>
<td>1.59</td>
<td>1.45</td>
<td>31.5</td>
<td>1.25</td>
<td>0.75</td>
<td>1.20</td>
<td>742</td>
<td></td>
<td></td>
</tr>
<tr>
<td>2</td>
<td>13</td>
<td>M</td>
<td>1.85</td>
<td>1.95</td>
<td>1.75</td>
<td>34.5</td>
<td>1.19</td>
<td>1.05</td>
<td>1.00</td>
<td>1013</td>
<td></td>
<td></td>
</tr>
<tr>
<td>3</td>
<td>27</td>
<td>M</td>
<td>1.95</td>
<td>2.05</td>
<td>1.85</td>
<td>31.0</td>
<td>0.91</td>
<td>0.70</td>
<td>1.40</td>
<td>1056</td>
<td></td>
<td></td>
</tr>
<tr>
<td>4</td>
<td>10</td>
<td>F</td>
<td>1.60</td>
<td>1.75</td>
<td>1.60</td>
<td>40.0</td>
<td>1.19</td>
<td>0.90</td>
<td>1.60</td>
<td>685</td>
<td></td>
<td></td>
</tr>
<tr>
<td>5</td>
<td>41</td>
<td>F</td>
<td>1.94</td>
<td>2.05</td>
<td>1.95</td>
<td>33.7</td>
<td>1.25</td>
<td>0.90</td>
<td>1.55</td>
<td>934</td>
<td></td>
<td></td>
</tr>
<tr>
<td>6</td>
<td>16</td>
<td>M</td>
<td>1.66</td>
<td>1.85</td>
<td>1.75</td>
<td>33.3</td>
<td>1.17</td>
<td>0.70</td>
<td>1.00</td>
<td>1011</td>
<td></td>
<td></td>
</tr>
<tr>
<td>7</td>
<td>24</td>
<td>M</td>
<td>1.96</td>
<td>2.10</td>
<td>2.05</td>
<td>36.5</td>
<td>1.27</td>
<td>1.10</td>
<td>1.90</td>
<td>1005</td>
<td></td>
<td></td>
</tr>
<tr>
<td>8</td>
<td>11</td>
<td>M</td>
<td>1.40</td>
<td>1.55</td>
<td>1.40</td>
<td>30.4</td>
<td>1.09</td>
<td>0.90</td>
<td>1.55</td>
<td>590</td>
<td></td>
<td></td>
</tr>
<tr>
<td>9</td>
<td>10</td>
<td>M</td>
<td>2.10</td>
<td>2.25</td>
<td>2.10</td>
<td>31.0</td>
<td>1.06</td>
<td>1.00</td>
<td>1.95</td>
<td>1031</td>
<td></td>
<td></td>
</tr>
<tr>
<td>10</td>
<td>15</td>
<td>M</td>
<td>1.20</td>
<td>1.35</td>
<td>1.20</td>
<td>38.2</td>
<td>1.21</td>
<td>0.65</td>
<td>0.95</td>
<td>702</td>
<td></td>
<td></td>
</tr>
<tr>
<td>11</td>
<td>20</td>
<td>M</td>
<td>1.85</td>
<td>2.00</td>
<td>1.85</td>
<td>33.7</td>
<td>1.26</td>
<td>1.00</td>
<td>1.60</td>
<td>927</td>
<td></td>
<td></td>
</tr>
<tr>
<td>12</td>
<td>13</td>
<td>M</td>
<td>1.50</td>
<td>1.65</td>
<td>1.50</td>
<td>30.7</td>
<td>1.28</td>
<td>0.85</td>
<td>1.25</td>
<td>647</td>
<td></td>
<td></td>
</tr>
<tr>
<td>13</td>
<td>15</td>
<td>M</td>
<td>1.45</td>
<td>1.60</td>
<td>1.45</td>
<td>33.0</td>
<td>1.20</td>
<td>0.90</td>
<td>1.60</td>
<td>699</td>
<td></td>
<td></td>
</tr>
<tr>
<td>14</td>
<td>20</td>
<td>M</td>
<td>1.30</td>
<td>1.45</td>
<td>1.30</td>
<td>40.2</td>
<td>1.50</td>
<td>0.80</td>
<td>1.35</td>
<td>794</td>
<td></td>
<td></td>
</tr>
<tr>
<td>15</td>
<td>21</td>
<td>F</td>
<td>1.50</td>
<td>1.65</td>
<td>1.50</td>
<td>39.0</td>
<td>1.20</td>
<td>1.00</td>
<td>1.60</td>
<td>1053</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mean</td>
<td>27.0</td>
<td></td>
<td>1.76</td>
<td>2.23</td>
<td>1.76</td>
<td>35.5</td>
<td>1.22</td>
<td>0.90</td>
<td>1.48</td>
<td>881</td>
<td></td>
<td></td>
</tr>
<tr>
<td>1 sd</td>
<td>9.5</td>
<td></td>
<td>0.21</td>
<td>0.28</td>
<td>0.21</td>
<td>3.9</td>
<td>0.31</td>
<td>0.14</td>
<td>0.30</td>
<td>140</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

*See text for definition of abbreviations.

Table 2

Quantitative Data—Myocardial Disease

<table>
<thead>
<tr>
<th>Pt</th>
<th>Age (yr)</th>
<th>Sex</th>
<th>Surface area (m²)</th>
<th>D₁ (cm)</th>
<th>D₂ (cm)</th>
<th>ΔD₁ (cm)</th>
<th>ΔD₁/D₁ (%)</th>
<th>ET (msec)</th>
<th>ΔD₂/ET (cm/sec)</th>
<th>W₁ (cm)</th>
<th>W₂ (cm)</th>
<th>R-R (msec)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>45</td>
<td>M</td>
<td>1.77</td>
<td>1.59</td>
<td>1.45</td>
<td>31.5</td>
<td>1.25</td>
<td>0.75</td>
<td>1.20</td>
<td>607</td>
<td></td>
<td></td>
</tr>
<tr>
<td>2</td>
<td>30</td>
<td>M</td>
<td>1.95</td>
<td>2.05</td>
<td>1.75</td>
<td>34.5</td>
<td>1.19</td>
<td>1.05</td>
<td>1.00</td>
<td>1154</td>
<td></td>
<td></td>
</tr>
<tr>
<td>3</td>
<td>35</td>
<td>M</td>
<td>1.88</td>
<td>1.95</td>
<td>1.75</td>
<td>31.0</td>
<td>0.91</td>
<td>0.70</td>
<td>1.40</td>
<td>1056</td>
<td></td>
<td></td>
</tr>
<tr>
<td>4</td>
<td>50</td>
<td>M</td>
<td>1.85</td>
<td>2.00</td>
<td>1.85</td>
<td>40.0</td>
<td>1.19</td>
<td>0.90</td>
<td>1.60</td>
<td>685</td>
<td></td>
<td></td>
</tr>
<tr>
<td>5</td>
<td>55</td>
<td>M</td>
<td>1.90</td>
<td>2.05</td>
<td>1.90</td>
<td>33.7</td>
<td>1.25</td>
<td>0.90</td>
<td>1.55</td>
<td>934</td>
<td></td>
<td></td>
</tr>
<tr>
<td>6</td>
<td>40</td>
<td>M</td>
<td>1.60</td>
<td>1.75</td>
<td>1.60</td>
<td>33.3</td>
<td>1.17</td>
<td>0.70</td>
<td>1.00</td>
<td>1011</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mean</td>
<td>43.8</td>
<td></td>
<td>1.86</td>
<td>2.05</td>
<td>1.86</td>
<td>35.5</td>
<td>1.22</td>
<td>0.90</td>
<td>1.48</td>
<td>744</td>
<td></td>
<td></td>
</tr>
<tr>
<td>1 sd</td>
<td>9.6</td>
<td></td>
<td>0.21</td>
<td>0.28</td>
<td>0.21</td>
<td>3.9</td>
<td>0.31</td>
<td>0.14</td>
<td>0.30</td>
<td>140</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

*Nodal rhythm.

is feasible to standardize the method of measurement of the distance between them.

Validity of Echo Interpretation

In vitro experiments performed by Edler proved that the interfaces between the ventricular walls and blood produced strong ultrasonic echoes.11 The same author passed a needle into the chest of a cadaver from the fourth left intercostal space and found that the needle had traversed the right ventricle, interventricular septum, and the posterior left ventricular wall below the posterior leaflet of the mitral valve.11 Similar results have been obtained in the dog.12 The echoes from the epicardium of the posterior left ventricular wall and pericardium were identified in vivo by Feigenbaum and associates during studies of pericardial effusion3, 4 and confirmed by the

Circulation, Volume XLVI, July 1972
injection of saline into the pericardial sac of dogs during ultrasonic examination.\(^3\) The echoes arising from the endocardial surface of the posterior left ventricular wall and from both sides of the interventricular septum were subsequently identified.\(^5\) Important confirmation of the origin of the cardiac echoes followed the discovery that injection of fluid by catheter into the cardiac chambers or great vessels during an ultrasound examination greatly increased reflection of ultrasound by the contained blood and hence transiently opacified the lumen.\(^13\) In this way the walls of the chamber could be identified with certainty. Feigenbaum et al. used this technic to prove the identity of the echoes postulated to arise from the endocardial surfaces of the septal and posterior walls of the left ventricle.\(^9\) Finally, the technic of continuous recording of echoes during scanning of the heart by the ultrasound beam has added further strong evidence of the identity of echoes. By gradually altering the direction of the ultrasound beam in the appropriate directions, the waveform of the echo from the interventricular septum can be observed to change into that of the anterior wall of the aortic root, the echo from the anterior leaflet of the mitral valve into that of the posterior wall of the aortic root, and the echo from the posterior left ventricular wall into that of the left atrial posterior wall (fig. 1). Such observations are consistent with the known anatomic relationships between these structures (fig. 2).

**Standardization of Measurement**

It might be imagined that a variety of left ventricular dimensions could be measured in a single subject by simply altering the direction of the ultrasound beam. However, provided that the left ventricular echoes were recorded in the standard manner described, the measurement of left ventricular dimension was reproducible when measured successively by independent observers, and this has also been the experience of other workers.\(^14\) This reproducibility can be attributed to the use of the mitral valve as an anatomic landmark to identify the portion of the left ventricular cavity traversed by the ultrasound beam, and to the fact that the echoes from the endocardial surfaces of the left side of the interventricular septum and posterior left ventricular

---

**Figure 6**

_Echocardiograms obtained from a normal subject (left) and from a patient with myocardial disease (right). The large divisions on the scale between the left ventricular echoes represent 1 cm. Note the larger size and diminished contraction of the diseased left ventricle._

_(Polaroid photograph, reversal print.)_
wall could be recorded simultaneously from only a limited area of the precordium and with the ultrasound beam aimed in this specific direction. The failure of an experienced operator to obtain such a recording seems to depend on many factors. For example, technical failure is more common in older subjects, in patients with chronic lung disease, and in those with severe mitral stenosis; failure is much less common in left ventricular volume overload due to aortic or mitral regurgitation. The failure rate of 13% in the present study can be considered a representative figure for normal subjects.

**Motion of the Left Ventricular Walls**

The waveform of the posterior left ventricular wall echo resembled an inverted ventricular volume curve. If both septal and posterior walls moved toward and away from the left ventricular long axis in a symmetric fashion during the cardiac cycle, and if the chamber as a whole did not move relative to the chest wall, it would have been expected that the contour of the septal echo would have been a mirror image of that of the posterior left ventricular wall. However, the smaller amplitude of the septal echo and its characteristic notched appearance can be explained if the changing distance between the left ventricular wall and the ultrasound transducer during the cardiac cycle is considered to be a composite of two movements. The main movements of the ventricular walls were inward toward the left ventricular long axis due to concentric narrowing of the chamber during systole with a corresponding outward movement occurring during diastole. However, superimposed on this motion was a movement of both septal and posterior walls, first toward the anterior chest wall during late systole then away from it in early diastole (fig. 5). Corresponding movements of the left ventricle were also observed during a cineradiographic study in man. Epicardial markers on the left ventricle indicated that the chamber moved slightly toward the anterior chest wall with a counterclockwise twisting motion during approximately the last one third of left ventricular ejection, a movement which was abruptly reversed as the left ventricle began to relax. This phenomenon was tentatively attributed to the late persistence of contraction near the epicardial surface of the left ventricle where the fibers spiral from apex to base in a counterclockwise direction. It is of interest that Wiggers described a similar movement of the exposed heart: . . . the ventricles rotate to the right giving a more frontal exposure to the left ventricle. On palpation, one experiences not only a sensation of great stiffening but also one of twisting.”

Finally, it should be noted that these movements would tend to add to the amplitude of posterior wall movement but subtract from that of the septum, as well as producing the characteristic notch in the waveform of its echo. This may be the sole explanation of the smaller amplitude of septal motion, but the observation that septal movement was greater nearer the apex than at the base of the left ventricle (fig. 2) suggested the additional possibility that the septal motion might have been limited also by its attachment superiorly to the aortic root (fig. 7).

![Figure 7](http://circ.ahajournals.org/)

---

Circulation, Volume XLVI, July 1972
Dimension during the Cardiac Cycle

The left ventricular dimension measured by ultrasound has been found to approximate closely the left ventricular minor axis in the anteroposterior projection calculated by the area-length method\(^1\) both at end-diastole and end-systole.\(^2\) When this evidence is supplemented with the anatomic data obtained by passing a needle through the thorax and heart to simulate the ultrasound beam,\(^3\) by plotting the approximate direction of the ultrasound beam on a left ventricular angiogram,\(^4\) and by observing the relationship of the mitral valve to the left ventricular echoes during ultrasound scanning, it seems most likely that the direction of the ultrasound beam which records the left ventricular dimension is slightly oblique to the left ventricular minor axis but possibly below the maximum width of the chamber in normal subjects—two errors which would tend to cancel each other. During preejection systole, the small and inconstant changes in left ventricular ultrasonic dimension were not in accord with the theory of “initial systolic expansion” which has been observed in the dog,\(^5\) a finding which was in agreement with the previous radiographic study in man.\(^6\) The changes in ultrasonic dimension during the remainder of the cardiac cycle corresponded with changes in left ventricular volume and were in agreement with previous studies.\(^7\) ,\(^8\)

Wall Thickness

Good agreement has been reported previously between the end-diastolic left ventricular wall thickness measured by ultrasound and measurements made at operation or autopsy.\(^9\) -\(^12\) The increase in left ventricular wall thickness during ejection was approximately 65% in the present study. A similar figure of 60–70% was obtained by an angiographic method.\(^13\) However, direct methods of measuring the systolic increase in left ventricular wall thickness used in animals have shown a systolic increase of approximately 20–30%.\(^15\) -\(^24\) The difference between the estimates obtained by these methods and by angiography has been attributed to the squeezing of contrast material from the spaces between trabeculae toward the end of ejection.\(^15\) ,\(^21\) ,\(^24\) ,\(^25\) Hence, it seems likely that the end-systolic surface outlined by contrast and that reflects ultrasound is represented by the tips of trabeculae which are compressed together.

Comparison with Angiography

In the present study, the left ventricular dimension at the beginning of systole had an average length of 4.40 cm and shortened to 2.83 cm during ejection. Representing the normal left ventricle as an ellipsoid with an axis ratio of 2:1, these dimensions correspond to left ventricular end-diastolic and end-systolic volumes of 89.2 and 23.7 ml, respectively, with an ejection fraction of 73.4. Both volumes are substantially smaller than those found in normal subjects using angiography.\(^26\) While it is possible that the ultrasonic measurement underestimates the left ventricular minor axis in normal subjects, the difference between angiographic and ultrasonic estimates could be explained by other factors such as the elevation of the thorax during the ultrasound examination and the transient expansion of the left ventricle caused by contrast injection.\(^27\) Although the use of the ultrasonic left ventricular dimension to estimate left ventricular volume has practical value in the measurement of cardiac output, the calculation of the ejection fraction as an index of myocardial shortening\(^14\) ,\(^28\) involves assumptions of left ventricular geometry which are unnecessary and which may not be valid when the chamber is enlarged.\(^29\) ,\(^30\)

Myocardial Function

Since the left ventricular circumference has been found to contract about the long axis of the chamber in an approximately symmetric manner,\(^31\) ,\(^32\) changes in the left ventricular ultrasonic dimension should reflect changes in the extent and rate of shortening of the left ventricular internal circumference. Thus, it should be possible to calculate indices of extent and rate of myocardial shortening in a manner analogous to those derived from estimates of left ventricular volume by thermodilution\(^33\) or from the left ventricular minor
axis measured by angiography. Using the latter method, Gault et al. found reductions in extent and rate of myocardial shortening in patients with myocardial disease which were similar to those found in the present study. Furthermore, a simple index of rate of myocardial shortening—the fractional shortening of the calculated left ventricular circumference—seemed to distinguish normal subjects from those with myocardial disease as effectively as did the more stringent calculation of rate of shortening of contractile elements at the left ventricular midwall. Thus, information on myocardial shortening similar to that obtainable by angiography can be elicited atraumatically using ultrasound.

Acknowledgments

The technical assistance of Mrs. Sue Livengood and the typing of the text by Mrs. Sue Williams are gratefully acknowledged.

References

17. Dodge HT, Sandler H, Ballew DW, Lord JD: The use of biplane angiography for the measurement of left ventricular volume in man. Amer Heart J 60: 762, 1960


27. DAVILLA JC, SAN MARCO ME, PHILLIPS CM: Continuous measurement of left ventricular volume in the dog: I. Description and validation of a method employing direct external dimensions. Amer J Cardiol 18: 574, 1966


33. PALEY HW, MCDONALD IG, WEISSLER AM: The relationship of left ventricular circumferential contraction to left ventricular ejection time as an inotropic index. (Abstr) Clin Res 12: 191, 1964
Analysis of Left Ventricular Wall Motion by Reflected Ultrasound: Application to Assessment of Myocardial Function
IAN G. MCDONALD, HARVEY FEIGENBAUM and SONIA CHANG

Circulation. 1972;46:14-25
doi: 10.1161/01.CIR.46.1.14
Circulation is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 1972 American Heart Association, Inc. All rights reserved.
Print ISSN: 0009-7322. Online ISSN: 1524-4539

The online version of this article, along with updated information and services, is located on the World Wide Web at:
http://circ.ahajournals.org/content/46/1/14

Permissions: Requests for permissions to reproduce figures, tables, or portions of articles originally published in Circulation can be obtained via RightsLink, a service of the Copyright Clearance Center, not the Editorial Office. Once the online version of the published article for which permission is being requested is located, click Request Permissions in the middle column of the Web page under Services. Further information about this process is available in the Permissions and Rights Question and Answer document.

Reprints: Information about reprints can be found online at:
http://www.lww.com/reprints

Subscriptions: Information about subscribing to Circulation is online at:
http://circ.ahajournals.org//subscriptions/