Ultrasonic Measurement of Left Ventricular Wall Motion in Acute Myocardial Infarction

By KIYOSHI INOUE, M.D., HAROLD SMULYAN, M.D., SAKTIPADA MOOKHERJEE, M.D., AND ROBERT H. EICH, M.D.

SUMMARY
The time-motion representation of the echogram was used to evaluate the left ventricular posterior wall motion in myocardial infarction. The left ventricular posterior wall echo was obtained in 10 normal subjects and 26 patients with chest pain admitted to a coronary care unit, including 11 with acute myocardial infarction (group 1), nine with old myocardial infarction (group 2), and six with chest pain of miscellaneous origin (group 3). The total amplitude of posterior wall excursion, left ventricular isometric contraction time, left ventricular systolic ejection time, and mean posterior wall velocity (ratio of posterior wall excursion to ejection time) were measured. By using the measurements of posterior wall excursion, mean posterior wall velocity, and isometric contraction time, group 1 patients were differentiated from those of groups 2 and 3 and from the normal subjects (all \( P < 0.01 \)). The data from patients in groups 2 and 3 were not statistically different from those of normal subjects. The measurements obtained by the time-motion curve of the left ventricular posterior wall echo appear to be of value in the bedside evaluation of acute myocardial infarction.

Additional Indexing Words:
Apexcardiogram Echocardiography Left ventricular contraction velocity
Myocardial asynergy Systolic and diastolic bulges

ONINVASIVE techniques for the evaluation of cardiac function have been of importance in patients with acute myocardial infarction because of difficulty in performing elaborate diagnostic procedures in patients with a major illness. Recent studies from this laboratory\(^1\) and others\(^2-8\) have demonstrated the accuracy of the apexcardiogram in assessing the severity of cardiac dysfunction in patients with coronary artery disease. However, an abnormal apical impulse on the chest wall, previously demonstrated in this disease,\(^1-4\) could not be detected in all patients with myocardial infarction. Suh and Eddelman,\(^5\) for example, studied the kinetocardiograms of 42 patients with myocardial infarction and noted that abnormal precordial bulges in 10 patients were either transient or absent. The occasional inability to record a satisfactory apical impulse in patients with acute myocardial infarction similarly prevents the detection of precordial bulges in these patients from the apexcardiogram.

Recent advances in techniques for the recording of cardiac motion by the ultrasonic reflectoscope have made available a safe and simple method for the assessment of cardiac function.\(^9-10\) The ability to obtain a discrete echo from the left ventricular posterior wall has been well accepted\(^9,10\) and used for the ultrasonic detection of pericardial effusion.\(^11\) Since the left ventricular posterior wall echo is
The normal ultrasonic pattern of posterior wall motion shown with the electrocardiogram (ECG) superimposed. The upward movement in the tracing represents a motion toward the anterior chest wall. The vertical scale indicates 1.0 cm of wall excursion, and the horizontal scale shows 200 msec of sweep speed. For abbreviations PWV, PWE, A, B, C, D, E, and F, see text.

Methods

A total of 36 subjects, including 10 normal men and 26 patients with chest pain, were studied. The normal men, aged 22 to 38 years, had no clinical evidence of heart disease. Twenty-six patients with acute chest pain were admitted to a coronary care unit. Their ages varied from 38 to 86 years, and there were 18 men and eight women. Patients were classified into three groups. Group 1 consisted of 11 patients with acute myocardial infarction proved by diagnostic changes in the ECG and serial blood enzyme studies; group 2, of nine with historical and ECG evidence of old myocardial infarction but no laboratory data to suggest acute involvement, and group 3, of six with acute chest pain, who subsequently proved not to have myocardial infarction. Two of these six had coronary insufficiency, three had small pulmonary emboli, and one had chest pain of undetermined origin. All patients had echocardiograms with 72 hr of admission to the coronary care unit.

A commercially available ultrasonoscope (phys-ionic-Tektronix-VT 500 CA-M) was used to record the left ventricular posterior wall echo with a transducer (0.75 inch in diameter, 2.25 MHz) and a repetition rate of 1,000 impulses/sec. A water-soluble gel was used to produce airless contact between the transducer and the patient's chest. With the patient in the recumbent position, the transducer was placed in the fourth intercostal space at the left sternal border and directed posteriorly, laterally, and inferiorly to obtain a group of strong echoes from the posterior left ventricular wall. These echoes were recognized by their characteristic motion—anteri- orly during ventricular systole and posteriorly during diastole. The sensitivity, rejecting, and damping modalities were adjusted to obtain the clearest posterior wall echo signals, as free as possible from low intensity noise, as visualized on the A-scan mode on the oscilloscope. When clear posterior wall echoes were visualized on the A-mode, the continuous echogram during several cardiac cycles was displayed by using a B-scan mode (time-motion representation), and permanent Polaroid photographs were taken of this B-scan presentation from the oscilloscope. An electrocardiogram was taken simultaneously on a standard strip-chart recorder at a paper speed of 50 mm/sec. The transducer-oscillator-oscilloscope system was calibrated by placing the transducer on a water-filled glass container of known dimensions. By means of an immersed movable rod, calibration was obtained by relating the transducer-rod distance to the amplitude of the echo pips.

Figure 1 is a Polaroid photograph taken of the B-scan presentation which illustrates the methods of measurement of the posterior wall echogram. The simultaneously recorded ECG has been photographically superimposed by use of the known relationships between the two curves. The letters on figure 1 follow those introduced by Krauza and Kennedy to describe the time-motion events of the posterior wall echo. The
posterior wall moves anteriorly (upstroke) during systole and posteriorly (downstroke) during diastole. During systole a reduction of ventricular diameter occurs and, therefore, the total amplitude of wall motion from end-diastole to end-systole has been related to the stroke volume. This total amplitude or excursion of posterior wall motion (PWE) was measured as the vertical distance between the C and D points and is expressed in centimeters. The mean velocity of posterior wall excursion (PWV) was measured as the slope of the line extending from the onset of anterior displacement (onset of left ventricular systolic ejection, C point) to the peak posterior wall echogram (end of left ventricular systolic ejection, the D point) and computed as the ratio of posterior wall excursion to the interval from C to D (C-D interval). This mean posterior wall velocity has been used as an index of mean systolic ejection rate. Point B, the wall position at end diastole, has been shown to appear nearly synchronously with the R wave of the ECG, and during isometric contraction (B-C interval), the wall moves posteriorly before its major motion anteriorly during left ventricular systolic ejection. In our study, the B point was easy to detect and, therefore, was used to mark the onset of left ventricular isometric contraction. Left ventricular isometric contraction time was measured as the interval between points B and C, and the interval between points C and D was used to measure the left ventricular systolic ejection time (ET). During early diastole (interval between E and F points), the posterior wall echo returns to its base-line position. The onset of sharp posterior displacement (E point) was taken as the end of isometric relaxation, and the interval between D and E points was used as a measurement of isometric relaxation time. Point A corresponds to ventricular filling as a result of atrial contraction.

To evaluate the time intervals obtained from the echocardiogram, recordings of the apexcardiogram, phonocardiogram, external carotid pulse tracing, and electrocardiogram were carried out on seven normal subjects and on 18 patients who had a recordable apical impulse. The apexcardiogram was obtained by using a pulse transducer microphone (model PS-2*), placed at the point of maximum left ventricular apical impulse. The phonocardiogram was obtained by use of a microphone (model PS-1B*) placed in the third

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**Table 1**

Echocardiographic Measurement in 10 Normal Subjects

<table>
<thead>
<tr>
<th>PWE (cm)</th>
<th>Mean PWV (cm/sec)</th>
<th>B-C</th>
<th>C-D</th>
<th>D-E</th>
</tr>
</thead>
<tbody>
<tr>
<td>Range</td>
<td>0.98-0.66</td>
<td>4.6-3.3</td>
<td>88-50</td>
<td>280-240</td>
</tr>
<tr>
<td>Mean</td>
<td>0.79</td>
<td>3.6</td>
<td>66.1</td>
<td>255.6</td>
</tr>
<tr>
<td>SD</td>
<td>0.11</td>
<td>0.4</td>
<td>14.8</td>
<td>16.7</td>
</tr>
<tr>
<td>SE</td>
<td>0.03</td>
<td>0.1</td>
<td>4.5</td>
<td>5.0</td>
</tr>
</tbody>
</table>

Abbreviations: PWE = posterior wall excursion; mean PWV = mean posterior wall velocity; for B-C, C-D, and D-E, see text and figure 1.

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*Electronics for Medicine, White Plains, N. Y.
intercostal space at the left sternal border with simultaneous recording of low and high frequency ranges. The carotid pulse tracing was obtained by using a pulse transducer microphone (model PS-1A*) held manually over the right common carotid artery. These tracings plus a routine ECG were simultaneously recorded at a paper speed of 100 mm/sec on a multichannel oscilloscopic photographic recorder (model DR-8*). Details of the techniques used for the recording and the measurement of these intervals have been described previously from this laboratory.1

Results

Normal Subjects

A characteristic posterior wall motion was recorded in all 10 normal subjects. All

*Electronics for Medicine, White Plains, N. Y.

patients, landmarks were sufficiently obvious to warrant measurement. All measurements from the total of 26 patients are presented in table 2.

Table 2

Echocardiographic and Hemodynamic Measurements

<table>
<thead>
<tr>
<th>Subject No.</th>
<th>Sex &amp; age (yr)</th>
<th>Transducer-PW distance (cm)</th>
<th>PWE (cm/sec)</th>
<th>Mean PWV (cm/sec)</th>
<th>Systolic time interval (msec)</th>
<th>Heart rate (beats/min)</th>
<th>Blood pressure (mm Hg)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>M60</td>
<td>12.8</td>
<td>0.70</td>
<td>2.5</td>
<td>120</td>
<td>78</td>
<td>110/85</td>
</tr>
<tr>
<td>2</td>
<td>M59</td>
<td>15.6</td>
<td>0.63</td>
<td>3.7</td>
<td>90</td>
<td>100</td>
<td>130/80</td>
</tr>
<tr>
<td>3</td>
<td>M86</td>
<td>11.6</td>
<td>0.56</td>
<td>1.9</td>
<td>125</td>
<td>60</td>
<td>120/75</td>
</tr>
<tr>
<td>4</td>
<td>F 42</td>
<td>9.8</td>
<td>0.63</td>
<td>2.4</td>
<td>90</td>
<td>81</td>
<td>110/76</td>
</tr>
<tr>
<td>5</td>
<td>M58</td>
<td>10.5</td>
<td>0.74</td>
<td>3.1</td>
<td>100</td>
<td>95</td>
<td>107/70</td>
</tr>
<tr>
<td>6</td>
<td>F 71</td>
<td>14.8</td>
<td>0.71</td>
<td>3.0</td>
<td>135</td>
<td>79</td>
<td>100/67</td>
</tr>
<tr>
<td>7</td>
<td>M43</td>
<td>9.9</td>
<td>0.31</td>
<td>1.2</td>
<td>130</td>
<td>76</td>
<td>108/70</td>
</tr>
<tr>
<td>8</td>
<td>F 65</td>
<td>10.4</td>
<td>0.26</td>
<td>1.4</td>
<td>90</td>
<td>100</td>
<td>110/80</td>
</tr>
<tr>
<td>9</td>
<td>F 38</td>
<td>11.4</td>
<td>0.18</td>
<td>0.8</td>
<td>90</td>
<td>88</td>
<td>98/70</td>
</tr>
<tr>
<td>10</td>
<td>M58</td>
<td>11.7</td>
<td>0.58</td>
<td>2.6</td>
<td>110</td>
<td>98</td>
<td>110/60</td>
</tr>
<tr>
<td>11</td>
<td>M57</td>
<td>11.3</td>
<td>0.65</td>
<td>2.7</td>
<td>90</td>
<td>71</td>
<td>115/75</td>
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</tbody>
</table>

Acute Myocardial Infarction (Group 1: 11 Cases)

<table>
<thead>
<tr>
<th>Subject No.</th>
<th>Sex &amp; age (yr)</th>
<th>Transducer-PW distance (cm)</th>
<th>PWE (cm/sec)</th>
<th>Mean PWV (cm/sec)</th>
<th>Systolic time interval (msec)</th>
<th>Heart rate (beats/min)</th>
<th>Blood pressure (mm Hg)</th>
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</thead>
<tbody>
<tr>
<td>12</td>
<td>M65</td>
<td>10.6</td>
<td>0.68</td>
<td>2.4</td>
<td>100</td>
<td>71</td>
<td>115/75</td>
</tr>
<tr>
<td>13</td>
<td>F 60</td>
<td>11.1</td>
<td>0.69</td>
<td>2.1</td>
<td>90</td>
<td>64</td>
<td>120/70</td>
</tr>
<tr>
<td>14</td>
<td>M84</td>
<td>12.1</td>
<td>0.90</td>
<td>3.3</td>
<td>70</td>
<td>76</td>
<td>140/90</td>
</tr>
<tr>
<td>15</td>
<td>M80</td>
<td>11.6</td>
<td>0.90</td>
<td>3.8</td>
<td>70</td>
<td>83</td>
<td>130/92</td>
</tr>
<tr>
<td>16</td>
<td>M57</td>
<td>10.3</td>
<td>0.98</td>
<td>4.5</td>
<td>80</td>
<td>90</td>
<td>145/85</td>
</tr>
<tr>
<td>17</td>
<td>M64</td>
<td>9.8</td>
<td>0.85</td>
<td>3.3</td>
<td>65</td>
<td>81</td>
<td>130/75</td>
</tr>
<tr>
<td>18</td>
<td>M60</td>
<td>13.6</td>
<td>0.80</td>
<td>3.8</td>
<td>55</td>
<td>93</td>
<td>128/70</td>
</tr>
<tr>
<td>19</td>
<td>F 69</td>
<td>14.0</td>
<td>0.75</td>
<td>3.8</td>
<td>50</td>
<td>84</td>
<td>150/88</td>
</tr>
<tr>
<td>20</td>
<td>F 54</td>
<td>12.4</td>
<td>0.85</td>
<td>3.4</td>
<td>90</td>
<td>75</td>
<td>117/80</td>
</tr>
</tbody>
</table>

Old Myocardial Infarction (Group 2: Nine Cases)

<table>
<thead>
<tr>
<th>Subject No.</th>
<th>Sex &amp; age (yr)</th>
<th>Transducer-PW distance (cm)</th>
<th>PWE (cm/sec)</th>
<th>Mean PWV (cm/sec)</th>
<th>Systolic time interval (msec)</th>
<th>Heart rate (beats/min)</th>
<th>Blood pressure (mm Hg)</th>
</tr>
</thead>
<tbody>
<tr>
<td>21</td>
<td>M50</td>
<td>10.7</td>
<td>0.89</td>
<td>3.4</td>
<td>70</td>
<td>75</td>
<td>130/80</td>
</tr>
<tr>
<td>22</td>
<td>M61</td>
<td>11.5</td>
<td>0.97</td>
<td>4.2</td>
<td>50</td>
<td>90</td>
<td>135/76</td>
</tr>
<tr>
<td>23</td>
<td>M64</td>
<td>12.1</td>
<td>0.78</td>
<td>3.6</td>
<td>85</td>
<td>87</td>
<td>110/70</td>
</tr>
<tr>
<td>24</td>
<td>M68</td>
<td>12.8</td>
<td>0.76</td>
<td>3.2</td>
<td>90</td>
<td>80</td>
<td>140/90</td>
</tr>
<tr>
<td>25</td>
<td>M44</td>
<td>9.6</td>
<td>0.69</td>
<td>3.0</td>
<td>55</td>
<td>78</td>
<td>150/85</td>
</tr>
<tr>
<td>26</td>
<td>F 61</td>
<td>10.1</td>
<td>0.81</td>
<td>3.2</td>
<td>60</td>
<td>70</td>
<td>130/75</td>
</tr>
</tbody>
</table>

Chest Pain of Miscellaneous Origin (Group 3: Six Cases)

Abbreviations: See table 1 and text.
Acute Myocardial Infarction (11 Cases)

There were seven cases with single wall damage: In three the anterior wall was damaged (cases 1, 2, and 4), in two, the lateral wall (cases 5 and 6), in one, the posterior wall (case 10), and in one the inferior wall (case 11). In four cases there was combined wall damage (cases 3 and 7 to 9). Two of these four patients (cases 7 and 9) died. Five patients had posterior wall excursions less than 0.6 cm, including the four with combined wall damage. These four patients also had mean posterior wall velocities of less than 2.0 cm/sec. Six patients had B-C intervals of more than 100 msec, and three had D-E intervals of more than 100 msec. The configuration of the posterior wall curve was altered in 10 individuals. There were anterior bulges during systole in four patients and prominent bulges during diastole (F and A wave) in six including the four with combined wall dam-

age. Figure 3 shows an altered configuration of the posterior wall echogram. Figure 4 shows the temporal relationship between the posterior wall echogram and the simultaneous tracings of the apexcardiogram, phonocardiogram, external carotid pulse, and electrocardiogram in a patient with an acute myocardial infarction and detectable apical impulse.

Old Myocardial Infarction (Nine Cases)

None of these patients had posterior wall excursions of less than 0.6 cm and mean posterior wall velocities of less than 2.0 cm/sec. Two (cases 12 and 13) had excursions measuring less than 0.7 cm, mean velocities of less than 3.0 cm/sec, and B-C and D-E time intervals of 80 msec or more. Four patients (cases 12, 13, 18, and 19) had radiologic evidence of left ventricular aneurysm and an altered configuration of the posterior wall echogram; two of these four had both systolic
and diastolic bulges, and two had prominent A waves.

No Myocardial Infarction (Six Cases)

Two patients (cases 23 and 24) had transient ST-T changes in the ECG, but normal results of serial cardiac enzyme studies. Three other patients (cases 21, 22, and 25) were suspected of having small pulmonary emboli and in the remaining patient (case 26), the genesis of the transient chest pain was unclear. No patient had posterior wall excursions of less than 0.7 cm and mean posterior wall velocities of less than 3.0 cm/sec, and the configuration of the posterior wall echogram of five individuals was similar to that of normal subjects.

Comparison of Measurement Data Between Normals and the Three Groups of Patients

The ranges and the mean values of each measurement obtained in the three groups of patients with chest pain are presented in table 3. The variation of the measurements in these groups of patients could not be explained by variations in age, duration of the cardiac cycle, or the level of arterial blood pressure. In comparing the results shown in table 3 with those in table 1, no measurements on groups 2 and 3 patients were statistically different from those of normal subjects (P > 0.05). However, the differences between group 1 patients and the normal subjects were significant (P < 0.01) for all measurements except C-D time intervals which were similar in these two groups. As seen in table 3, group 1 patients could be statistically separated from patients in groups 2 and 3, by using the posterior wall excursion, mean posterior wall velocity, and the B-C time interval. There were no differences in C-D time intervals among the groups. The D-E time interval in group 1 patients was similar to that of group 2 patients, but different from that of group 3 patients or normal subjects.

Discussion

One dimensional view of ventricular wall motion provided by the ultrasonic echogram of the posterior wall offers a noninvasive means for assessing ventricular volume and velocity of contraction, and thus for indirectly
evaluating left ventricular function.\textsuperscript{17-19} This echogram is easy to record and provides a safe and simple procedure which can be performed at the bedside. It has been stated that the posterior wall echogram has a strong resemblance to the standard ventricular volume curve.\textsuperscript{12} The posterior wall motion during a cardiac cycle, therefore, may be a function of ventricular volume and diameter, since the amplitude of the total wall excision, from end diastole to end systole, has been related to left ventricular stroke volume.\textsuperscript{18, 19} The velocity of anterior displacement of the posterior wall during systole has been taken as an index of the rate of decrease in ventricular diameter and of the velocity of circumferential fiber shortening.\textsuperscript{12, 14} The ratio of the posterior wall excursion to the left ventricular systolic ejection time, therefore, may be an indirect expression of left ventricular mean systolic ejection rate. A slow sweep time-motion representation of the echoes obtained in this study also offers a noninvasive measurement of the left ventricular systolic time intervals, namely, the isometric contraction and relaxation periods, and ejection time.

Normal values of the posterior wall excursion and mean posterior wall velocity have been previously measured by Kraunz and Kennedy\textsuperscript{19} using similar techniques but in larger numbers of subjects. They reported a wide range of normal posterior wall excursions from 1.03 to 0.36 cm and of normal mean posterior wall velocities from 6.7 to 2.2 cm/sec. Our study, however, demonstrated a relatively narrow range of these normal values, although the mean values in both studies were in close agreement. This narrow normal range made it possible for us to compare these data with those obtained in patients with coronary artery disease.

Observations on patients with acute myocardial infarction in this study demonstrated a significant reduction in the amplitude of the posterior wall motion when compared with that obtained in subjects without acute myocardial infarction or in the normal subjects. Even more striking was a similar decrease in the velocity of wall motion during systole. These findings are suggestive of a diminution of left ventricular stroke volume and a reduction in myocardial contractility. An alteration in the configuration of the posterior wall echogram was also noted in 10 of 11 patients with acute myocardial infarction. This consisted of early and mid-systolic bulges and abnormal diastolic waves (F and A waves). The mechanism of production of these bulges probably relates to the distortion of ventricular motion and to myocardial asynergy which has been demonstrated by cineventriculography in patients with coronary artery disease.\textsuperscript{6, 20, 21} Similar findings have been shown in experimental myocardial ischemia which produces local akinesis and an abnormality in force, speed, and sequence of ventricular contraction.\textsuperscript{22} An increase in left ventricular end-diastolic pressure and diminished ventricular distensibility might produce abnormal diastolic curves, since similar abnormalities have previously been detected by the apexcardiogram.\textsuperscript{1, 6-8, 23} In this study some patients with a detactable apical impulse were also examined by means of the apexcardiogram. The abnormal waves in these patients were consistently depicted in both apexcardiogram and echogram. The similarity of the wave-form abnormalities detected by these two methods lends support to the echocardiographic detection of abnormal ventricular motion in patients with severe myocardial ischemia.

The systolic time intervals, as measured by noninvasive polygraphic techniques, have been widely accepted as a sensitive means for the assessment of the severity of cardiac dysfunction.\textsuperscript{1, 24-27} Although the normal values for systolic time intervals using echographic techniques are not available from previous publications, the values obtained in this study were in the previously described normal range obtained by using the apexcardiogram, phonocardiogram, and carotid pulse tracing.\textsuperscript{1, 24, 26, 28} The isometric contraction period obtained in this study by the time-motion curve of the posterior wall echogram demonstrated significant differences between patients with acute myocardial infarction and
those without. This finding further supports a reduction in myocardial contractility in patients with acute myocardial infarction as predicted by measurements of posterior wall excursion and mean velocity and provides another bedside means for assessing myocardial contractility in patients without a detectable apical impulse.

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