Left Ventricular Function in Acute Myocardial Infarction Evaluated by Gated Scintiphotography


SUMMARY
Ten normal volunteers and 38 patients with acute myocardial infarction were evaluated by biplane gated blood pool scanning. The mean left ventricular end-diastolic volume in those with infarction was 125 ± 41 ml/m² compared to 82 ± 10 ml/m² in the normals. The left ventricular end-systolic volume was 82 ± 35 ml/m² compared to 35 ± 4 ml/m², and the left ventricular ejection fraction 36 ± 8% compared to 56 ± 3% in the normals. Thirty-six of the 38 patients with infarction had an area of akinesis which ranged from 15 to 59% of the left ventricular wall. Patients with acute myocardial infarction were found to have a significant increase in left ventricular end-systolic volume and decrease in ejection fraction compared to normals. The end-diastolic volume was, however, increased only in those with an elevated left ventricular filling pressure or decreased cardiac index.

Follow-up studies obtained in 20 patients between one week and three months following infarction showed that in the 14 who improved clinically, left ventricular ejection fraction significantly increased from 38 to 45% (P < 0.001) while in six who failed to show clinical improvement or worsened, left ventricular ejection fraction remained at 30%.

Left ventricular ejection fraction was significantly greater and the extent of akinesis significantly less in the patients who survived compared to those who died.

Additional Indexing Words:
- Hemodynamics
- Ejection fraction
- End-diastolic volume
- Nuclear medicine
- Nuclear medicine

HEMODYNAMIC MONITORING in patients with acute myocardial infarction has increased our understanding of the pathophysiology of myocardial infarction and allowed an objective assessment of therapy.1-7 The hemodynamic parameters commonly measured in patients with acute myocardial infarction include left ventricular filling pressure, cardiac output, and aortic pressure. Indices of left ventricular function derived from these values are, however, incomplete and on occasion misleading due to variations in left ventricular compliance.8 The development of gated cardiac blood pool scanning permits a non-invasive determination of left ventricular volumes, ejection fraction, and regional wall motion.9, 10

Methods

Thirty-eight patients, age 39 to 77 (mean 59) years with acute myocardial infarction were studied within 48 hours of the onset of symptoms. All patients had acute myocardial infarction as determined by serial enzyme changes, (creatine phosphokinase greater than 50 International Units or lactic dehydrogenase greater than 300 International Units, or both) as well as serial electrocardiographic changes of infarction.

Hemodynamic studies were performed in the Coronary Care Unit. A number 5 or 7 Swan Ganz catheter was inserted percutaneously or by cutdown into an antecubital vein and floated into the pulmonary artery under fluoroscopic guidance. A two and one-half inch teflon needle was placed percutaneously or by cutdown into the radial artery. All pressures were measured, using the midsternal position as zero reference point and recorded employing a pressure transducer. Cardiac output was determined in duplicate by the indicator dilution technique using either indocyanine green or room temperature normal saline. Indocyanine green was injected into the pulmonary artery with sampling from the radial artery through a densitometer at a rate of 38.2 ml/min and the blood reinfused. Dynamic on-line calibration was used.12 Room temperature saline was injected into the proximal lumen of the #7 Swan-Ganz catheter and the temperature recorded with a distal ther-

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Supported by Contract No. PH 43 NHLI 67-1444 with the National Institutes of Health, Department of Health, Education and Welfare.

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Received April 29, 1974; revision accepted for publication June 24, 1974.

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mistor. The thermodilution curves were analyzed using an analog computer. Stroke work index was calculated as:

\[ (\text{MSP} - \text{LVFP}) \times \text{SI} \times 13.6 \]

where MSP = mean systolic pressure (mm Hg); SI = stroke volume (ml/m²); and LVFP = left ventricular filling pressure (mm Hg).

On the basis of these hemodynamic studies the patients were divided into three groups.

Group I — normal hemodynamics. Left ventricular filling pressure less than or equal to 15 mm Hg, cardiac index greater than or equal to 2.5 L/min/m², and a systolic arterial pressure greater than 90 mm Hg.

Group II — abnormal hemodynamics. An increase in left ventricular filling pressure or a decrease in cardiac index, or both, associated with a systolic arterial pressure of 90 mm Hg or greater.

Group III — cardiogenic shock. A systolic pressure less than 90 mm Hg or a fall in systolic arterial pressure of 50 mm Hg or more from previous hypertensive levels associated with evidence of circulatory insufficiency.

Ten normal volunteers with a mean age of 30 (range 20 to 38) were also evaluated by the gated scintigraphic technique.

Gated cardiac blood pool scans were obtained within two hours of the hemodynamic study by positioning the patient in the right anterior oblique position under the detector of a scintillation camera with a high resolution parallel hole collimator. Twenty milliliter units of technetium 99m labeled human serum albumin was administered intravenously as a bolus and the first passage of the tracer through the heart and great vessels recorded on 35 mm film at one frame/second for 30 seconds. After initial equilibration of the isotope with blood was complete, a blood pool scan was recorded to outline the chambers of the heart and great vessels for use in defining the limits of the chambers in subsequent studies. The scintillation camera was then gated so that signals were only recorded on film during 40 to 60 msec portions of each cardiac cycle. Since each heart beat contributes only 4 to 600 counts, the process is repeated until 200,000 counts were recorded during the preselected portion of the cardiac cycle. End systole was recorded as the portion of the cardiac cycle that corresponded to the downslope of the T wave; end diastole, 60 msec before the next R wave. After recording the end-systolic and end-diastolic scintiphotographs in the right anterior oblique view, the patient was then positioned in the left anterior oblique position and the gated portions of the examination repeated.

The scintiphotographs were projected life-size and the left ventricle outlined in each view at both systole and diastole. The life-sized traced outlines are then treated in a manner similar to contrast angiograms. Patients in whom the scintiphotographs were inadequate due to right ventricular dilatation or other technical problems were not analyzed. In two patients analysis was not performed due to right ventricular dilatation. Scans were inadequate in two patients because of blurring due to patient motion, in one because of incorrect positioning, and in another because of the presence of free pertechnetate which increased background activity. End-diastolic volume and end-systolic volumes were calculated as suggested by Dodge et al. from the formula:

\[ V = \pi \frac{L \times M_1 \times M_2}{6} \]

where L = long axis of the left ventricle in the right anterior oblique projection and M1 and M2 = the average short axis in the right anterior oblique and left anterior oblique projections, respectively, calculated from the formula M = \( \frac{4 \text{ Area}}{\pi L} \)

Ejection fraction was calculated by the formula:

\[ \text{EF} = \frac{\text{EDV} - \text{ESV}}{\text{EDV}} \]

where EF = percent left ventricular ejection fraction; EDV = left ventricular end-diastolic volume (ml) and ESV = left ventricular end-systolic volume (ml).

Regional myocardial wall motion was determined by superimposition of the end-diastolic and end-systolic images in both the right and left anterior oblique projections (fig. 1). The perimeter of the left ventricular chamber was measured in each projection excluding the area of the valve planes. The portion of this perimeter which overlapped with the end-systolic perimeter was measured. The extent of akinesis was expressed as a percentage of the left ventricular wall, averaged from both the right and left anterior oblique projections. Since these measurements were performed in only two planes, they can only be an estimate of the true surface area of the left ventricle which is akinetic.

Results

Normal Volunteers

The mean left ventricular end-diastolic volume was 82 ± 10 ml/m², end-systolic volume, 35 ± 4 ml/m², and ejection fraction, 56 ± 3% for the ten normal volunteers, respectively. No areas of akinesis were found in these volunteers.

Patients with Acute Myocardial Infarction

The results in the 38 patients with acute myocardial infarction are shown in table 1 according to hemodynamic group. The mean left ventricular end-diastolic volume for all of the 38 patients with myocardial infarction was 125 ± 41 ml/m² and end-systolic volume 82 ± 35 ml/m². measures which were significantly different from the values obtained in the normal volunteers (P < 0.01, P < 0.001 respectively). The mean left ventricular ejection fraction was 35.7 ± 8.4%. There was only one patient in the entire group who had an ejection fraction within normal range.

Patients in hemodynamic Group I (normal hemodynamics) (table 1) had a significant increase in left ventricular end-systolic volume and a decrease in left ventricular ejection fraction compared to the normal patients (fig. 2). Left ventricular end-diastolic volume, although not significantly increased for Group I patients as a whole, was elevated in approximately one-half of the patients. Patients in Groups II (abnormal hemodynamics) and III (cardiogenic shock) had a significant increase in both left ventricular end-systolic volume and end-diastolic volume as well as a decrease in left ventricular ejection fraction compared to the normal control patients.
Left ventricular end-systolic volume was significantly greater and ejection fraction significantly less in Group II than Group I and in Group III than in Group II. Left ventricular end-diastolic volume was significantly greater in Group III than in Group II but Group II was not significantly greater than Group I.

The relationship between left ventricular filling pressure and left ventricular end-diastolic volume is shown in figure 3 for the entire group of 38 patients. A poor correlation ($r = 0.48$) was observed.

Thirty-six of the 38 patients had an area of akinesis detected on their scintiphotographs which ranged from 15 to 59% of the left ventricular wall. An example of a patient with an antero-septal akinesis is shown in figure 1. The location of the akinesis was consistent with the location of the acute infarction identified by the electrocardiogram.

Left ventricular ejection fraction and extent of akinesis were compared for the entire group (fig. 4) and were found to have a negative correlation ($r = -0.75$).

Follow-up studies were obtained in 20 patients between one week and three months following their acute infarction. Of these 20 patients, 14 had a favorable course in that they were uncomplicated on admission and remained so, or showed improvement prior to discharge. In these patients, left ventricular ejection fraction increased significantly from 38 to 45% ($P < 0.001$, paired t-test). The two patients who did not show improvement of their ejection fraction in that group had recurrent chest pain, and in one an extension was documented. This is in contrast to six patients in failure who failed to improve, or whose condition worsened. In these patients, left ventricular ejection fraction was 30% both on the original and follow-up studies (fig. 5). The one patient who had an increase in ejection fraction had papillary muscle dysfunction and episodes of paroxysmal left ventricular failure with pulmonary edema which persisted.

### Table 1

<table>
<thead>
<tr>
<th>Patients with acute myocardial infarction</th>
<th>Normal control</th>
<th>Group I (15 pts)</th>
<th>Group II (22 pts)</th>
<th>Group III (3 pts)</th>
</tr>
</thead>
<tbody>
<tr>
<td>EF (%)</td>
<td>56.3 ± 3</td>
<td>41.8 ± 5.4†</td>
<td>34.7 ± 6.1†</td>
<td>18.7 ± 4.5†</td>
</tr>
<tr>
<td>ESV (ml/m²)</td>
<td>35.3 ± 3.6</td>
<td>61.3 ± 23.7†</td>
<td>85.5 ± 29.8†</td>
<td>146.7 ± 29.3†</td>
</tr>
<tr>
<td>EDV (ml/m²)</td>
<td>82 ± 10.2</td>
<td>104.1 ± 34.7 (NS)</td>
<td>129.3 ± 38.6</td>
<td>178.7 ± 36.1†</td>
</tr>
<tr>
<td>HR (beats/min)</td>
<td>70.3 ± 10.3</td>
<td>83.8 ± 19.9*</td>
<td>96.7 ± 18.5 (NS)</td>
<td></td>
</tr>
<tr>
<td>LVFP (mm Hg)</td>
<td>10.2 ± 2.7</td>
<td>20.8 ± 7.5†</td>
<td>26 ± 6.6 (NS)</td>
<td></td>
</tr>
<tr>
<td>CI (L/min/m²)</td>
<td>3.3 ± 0.55</td>
<td>2.6 ± 0.67†</td>
<td>2.2 ± 0.26 (NS)</td>
<td></td>
</tr>
<tr>
<td>MAP (mm Hg)</td>
<td>103.5 ± 24.2</td>
<td>103.1 ± 20.9 (NS)</td>
<td>77.3 ± 26.1*</td>
<td></td>
</tr>
<tr>
<td>SWI (g-m/m²)</td>
<td>75.4 ± 29.5</td>
<td>43.8 ± 18.9*</td>
<td>17.9 ± 2.75 ‡</td>
<td></td>
</tr>
</tbody>
</table>

The results are expressed as the mean ± 1 standard deviation.

* = a significant difference from the preceding group using the Student's t-test.
† = a significant difference from the normal group.
‡ = difference is the result of the criteria used to classify the patient.

Abbreviations: EF = left ventricular ejection fraction; ESV = end-systolic volume; EDV = end-diastolic volume; HR = heart rate; LVFP = left ventricular filling pressure; CI = cardiac index; MAP = mean aortic pressure; SWI = stroke work index; Pts = patients; NS = not significant.
Six patients died, one in Group I (normal hemodynamics), three in Group II, (abnormal hemodynamics), and two in cardiogenic shock, Group III. The ejection fraction was significantly greater and the extent of akinesia significantly less in the patients who survived than in those who died (table 2).

In figure 6 the left ventricular ejection fraction of those who died is compared to those who survived in relation to the initial clinical findings. Two patients with uncomplicated courses died suddenly of arrhythmias. One of those who died with left ventricular failure had the sudden onset of electromechanical dissociation. The other three patients had signs of progressive left ventricular failure prior to death.

Discussion

Left ventricular ejection fraction, volume, and abnormalities of regional myocardial wall motion obtained by gated cardiac blood pool scanning have been shown to correlate with contrast angiography both in our own and other laboratories. The determination of left ventricular ejection fraction may, however, be more reliable than individual
Comparison of initial ejection fraction (EF) obtained less than 48 hours from onset of symptoms of infarction to that obtained after one week. The large cross is the mean of the 14 patients who clinically improved and the large dot the mean of the six who failed to show clinical improvement. The line of identity is shown. The increase in left ventricular ejection fraction in those who clinically improved was significant \( P < 0.01 \), using the paired Student's t-test. There was no significant increase in ejection fraction in those who did not improve.

Patients with clinically uncomplicated myocardial infarction and normal hemodynamic studies including left ventricular filling pressure and cardiac index (Group I) in the present study have been found to have a significant increase in left ventricular end-systolic volume and a decrease in left ventricular ejection fraction. Left ventricular ejection fraction appears to be a sensitive indicator of left ventricular dysfunction in these clinically uncomplicated patients. These findings suggest that diminished mean systolic fiber shortening is the primary alteration of left ventricular function in patients with acute myocardial infarction. The results of this clinical study are consistent with previous animal studies.\(^{16, 17, 18}\) The increase in left ventricular end-diastolic volume, seen in some of the patients with a normal left ventricular filling pressure and cardiac index, suggests that the cardiac index is, in part, maintained by the Starling mechanism.\(^{16, 17}\) While the increase in left ventricular end-diastolic volume maintains cardiac index in these patients, it also increases left ventricular wall tension, myocardial oxygen demands, and therefore, possibly infarct size. Dependence on the Starling mechanism is even more evident in Group II and III patients with signs of manifest left ventricular dysfunction in whom left ventricular end-diastolic volume is significantly increased compared to normal patients.

The increase in left ventricular filling pressure in Group II and III patients and occasionally in Group I (normal hemodynamics) patients has been attributed to a decrease in left ventricular compliance.\(^{19}\) The results of the present study suggest that the increase in left ventricular filling pressure may also be due to a decrease in left ventricular function. Indeed, the poor correlation between left ventricular filling pressure and end-diastolic volume for the group as a whole implies a large variability in left ventricular diastolic compliance. The finding of an increase in left ventricular end-diastolic volume in some of the patients with a normal left ventricular filling pressure suggests that in at least some of the patients with myocardial

**Table 2**

<table>
<thead>
<tr>
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<th>% Ejection fraction</th>
<th>% Akinesis</th>
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<tbody>
<tr>
<td>Survivors (32)</td>
<td>37.5 ± 7</td>
<td>27.2 ± 10.3</td>
</tr>
<tr>
<td>Nonsurvivors (6)</td>
<td>27.2 ± 9.3</td>
<td>40.8 ± 16</td>
</tr>
<tr>
<td></td>
<td>( P &lt; 0.01 )</td>
<td>( P &lt; 0.02 )</td>
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</table>
infarction left ventricular compliance may actually be increased, as shown early in the course of experimental myocardial infarction.\textsuperscript{36, 31} Multiple points on the pressure volume curve must, however, be obtained in each individual patient before reaching a conclusion about changes in left ventricular compliance during infarction. The variable relationship between left ventricular filling pressure and left ventricular end-diastolic volume suggests caution in the use of left ventricular filling pressure alone in patients with an acute myocardial infarction to plot left ventricular function curves. This has been recently pointed out by Swan et al.\textsuperscript{8}

In addition to providing measurements of left ventricular volume, the scintigraphic technique also reveals the site and extent of myocardial infarction as judged by the site and extent of left ventricular akinesis. The site of the infarct determined by the scintigraphic technique corresponded to that suspected from the patients’ electrocardiogram in all of the patients with a single myocardial infarction. The failure of Kostuk et al.\textsuperscript{11} to find a higher percentage of akinesis in their study of patients with acute myocardial infarction may in part be due to their failure to obtain biplane images and in part to differences in technique. The extent of akinesis in the present study correlated with left ventricular ejection fraction ($r = -0.75$). The failure to find an even higher correlation between extent of infarction and left ventricular ejection fraction may reflect the dependence of ejection fraction on the state of the uninfarcted muscle or other factors such as variation in impedance to ejection. Two patients with similar degrees of myocardial muscle loss may have different degrees of left ventricular dysfunction dependent upon the presence of previous fibrosis and the blood supply to the noninfarcted myocardium. The slightly better correlation between the extent of akinesis and left ventricular ejection fraction as found by Feild et al.\textsuperscript{22} using contrast angiography in the convalescent phase of myocardial infarction could be due to better sensitivity of contrast angiography in defining the extent of akinesis and/or the presence of a variable degree of reversible left ventricular dysfunction in border zones surrounding the acutely infarcted area in the present study.

The determination of left ventricular ejection fraction appears to be of prognostic value (table 2, fig. 6). Left ventricular ejection fraction is a more sensitive index of left ventricular dysfunction than left ventricular filling pressure or cardiac index. It is, however, unlikely that it will identify those uncomplicated patients who die, since their deaths are often unrelated to left ventricular failure. Although those who died in left ventricular failure tended to have relatively low ejection fractions, our experience is as yet too limited to determine whether this measurement will be superior to other measurements in predicting mortality.

The extent of akinesia, a reflection of infarct size, may also be of prognostic value since the size of the akinetic site was larger in patients who died than in those patients who survived. Sobel et al., using serial CPK analysis to estimate infarct size, also found infarct size to be an important prognostic indicator.\textsuperscript{23}

The follow-up scintigraphic studies have been of interest in revealing that those patients who have an uncomplicated clinical course or who improve their clinical status following the initial stages of myocardial infarction have an increase in left ventricular ejection fraction, while those who fail to show clinical improvement also failed to significantly increase their left ventricular ejection fraction. Serial changes in left ventricular ejection fraction following infarction may, therefore, allow objective assessment of prognosis and serve as a guide to therapy.\textsuperscript{11}

While the number of patients studies with the scintigraphic technique is relatively small, the safety with which the information about left ventricular volume, ejection fraction, and regional wall motion can be obtained in these acutely ill patients suggests that the role this technique in the evaluation of patients with acute myocardial infarction will increase.

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Circulation. 1974;50:678-684
doi: 10.1161/01.CIR.50.4.678

Circulation is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
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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:
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