Left Ventricular Size after Acute Myocardial Infarction
Serial Changes and their Prognostic Significance

By William J. Kostuk, M.D., Thomas M. Kazamias, M.D., Martin P. Gander, M.D., Allan L. Simon, M.D., and John Ross, Jr., M.D.

SUMMARY
The distance from the midline to the left heart border measured in a standardized manner on calibrated chest roentgenograms was determined serially in 125 patients after acute myocardial infarction. This external left heart dimension (LHD) was compared to the normal value, and its prognostic value was assessed during early and late follow-up periods (average 10%, range 2–31 months). Within 96 hours after acute myocardial infarction the initial LHD was enlarged (>52 mm/m² BSA [body surface area]) in 42% of the patients. The LHD became normal, or remained persistently normal in 60% of the 115 patients without shock, and it became enlarged or remained persistently enlarged in 40%. If the 10 patients with cardiogenic shock are excluded, early death (<4 weeks) occurred in three of 68 patients (4%) with initially normal LHD, and in 12 of 47 patients (26%) with initially enlarged LHD (P < 0.001). The LHD remained normal or became normal in 62 of 96 patients followed who survived more than 1 month. During the follow-up period, 8% of patients with a normal LHD have died and the remainder (with one exception) are in New York Heart Association (NYHA) class I or II. Only 5% of this group died of acute cardiac causes. In 34 patients the LHD became or remained persistently enlarged; of these patients 24% have expired (P < 0.05) and 42% are in NYHA class III (P < 0.001). This study validates a method for the early detection and serial assessment of left ventricular enlargement and indicates that the LHD is a useful predictor of survival and morbidity after acute myocardial infarction.

Additional Indexing Words:
Left heart dimension Heart size Prognosis after MI

A NUMBER of risk factors have been evaluated as predictors of mortality following acute myocardial infarction. These have included such variables as age, sex, obesity, previous history of myocardial infarction, presence and severity of cardiac failure, shock, rhythm disorders, and complicating illnesses.1–9 A variety of prognostic indices based on these and other factors have been derived.10–14

Several studies have suggested that overall heart size is of prognostic importance following acute myocardial infarction,5, 4, 12–14 although some earlier reports were at variance with this conclusion.15, 16 We considered the possibility that changes in overall heart size could be relatively insensitive to alterations in the size of the left ventricle, the chamber predominantly involved by acute myocardial infarction.17 We further postulated that heart size might prove to be of greater significance provided selective chamber enlargement could be identified, and if serial studies could be performed accurately during early and late follow-up periods. In a previous communication18 we have described a method which allows estimation of left ventricular size from a standardized, plain chest roentgenogram. The method was adapted for use in acutely ill patients, and it appeared to be sufficiently accurate and reproducible for reliable serial measurements.18 Accordingly, the present report describes the alterations in left ventricular size which occur following acute myocardial infarction and analyzes the significance of an enlarged left ventricle during early recovery and late follow-up periods.

From the Department of Medicine, School of Medicine, University of California at San Diego, La Jolla, California.

Supported by U. S. Public Health Service, National Heart and Lung Institute Myocardial Infarction Research Unit Contract PH-43-1352. Dr. Kostuk is the recipient of the Ontario Training Fellowship, Ontario Heart Foundation and Royal College H. K. Detweiler Travel Fellowship.

Address for reprints: John Ross, Jr., M.D., University of California, San Diego, School of Medicine, Department of Medicine, La Jolla, California 92037.

Received October 12, 1972; revision accepted for publication February 6, 1973.
Methods

Studies were performed on 125 patients studied sequentially in the Myocardial Infarction Research Unit of the National Heart and Lung Institute. Their ages ranged from 36 to 85 (mean 58.6) years; 104 patients were males and 21 were females. Each patient had experienced recent acute myocardial infarction, established by the presence of at least two of the following: (1) a history of typical prolonged chest pain; (2) electrocardiographic changes indicative of acute myocardial injury with subsequent evolution of a typical infarction pattern; (3) characteristic elevations of serum enzymes (SGOT, CPK, and/or LHD).

In each patient, a calibrated chest roentgenogram was taken on admission to the unit, the times varying from 1 to 96 hours following the onset of chest pain. As described elsewhere, the roentgenograms were taken with the patient in a specially modified, radiolucent bed with the head of the bed placed horizontally, or at a 45° angle; however, all recent studies have been carried out in the supine position. Films were triggered to be exposed at end-diastole and during inspiration to 1 liter above functional residual capacity. On the developed X-ray film, a vertical line was drawn from the jugular notch to the xyphoid process (small lead markers were placed on the chest to mark these sites) and a second parallel line was drawn tangentially to the widest point of the left ventricular silhouette. The horizontal line joining these two lines, corrected for X-ray magnification and normalized for body surface area (BSA), represented the left heart dimension (LHD). A C-arm mounted X-ray tube was employed which had a fixed tube-to-film distance of 9.15 cm. One half of the patient's anteroposterior chest diameter (x) was measured and the distance from X-ray film to the heart then represented 91.5 - x. Since the film cassette was positioned against the anterior chest, the correction factor = (91.5 - x)/91.5. The mean normal value for the LHD was 44 mm/m² and the upper limit of normal for LHD was considered to be greater than 2 SD from the mean of the normal group (> 52 mm/m²). Most of the patients studied were first admissions to the University Hospital and previous chest films were not available; therefore, the incidence of left ventricular enlargement prior to myocardial infarction is unknown. A history of previous myocardial infarction was the same (37%) in patients with and without initial enlargement of the LHD. There was a slightly greater incidence of a history of hypertension when LHD was increased (43 vs 30%); and a prior history of congestive heart failure was obtained in 27% of patients with an enlarged LHD and 15% of those in whom the initial LHD was normal.

Serial measurements of the left heart dimension were made on admission and every 12-24 hours during the first 2-3 hospital days; it was again measured prior to hospital discharge and at outpatient follow-up visits every 2-4 months. The longest follow-up period was 31 months, the shortest 2 months, and the average 10% months following discharge from the hospital.

Results

On the initial film, 53 patients (42%) demonstrated an enlarged LHD, and among all 125 patients 54% at some time during their course exhibited an augmented LHD.

If patients in cardiogenic shock are excluded (in whom six of six had an enlarged LHD), 47 patients (41%) exhibited an initially enlarged LHD. Of these, the LHD remained enlarged in 36 (77%) during the subsequent days to weeks (in two it became transiently normal and then persistently abnormal) while in 11 patients (23%) it became normal.

In 68 of the patients without cardiogenic shock, the LHD was initially normal. In four it became transiently enlarged but returned to normal, whereas in 10 patients (15%) it became enlarged and remained so.

Therefore, in 46 of the 115 patients without shock (40%) the LHD became or remained persistently enlarged, and in 69 of the patients without shock (60%) the LHD became normal or remained persistently normal. Representative serial studies of the LHD are shown in figure 1.

Mortality

Thirty-eight of the 125 patients (30%) expired during the entire study period. The early mortality (< 4 wks) was 20%.

Of the early deaths, all 10 patients in cardiogenic shock died, and 15 other patients expired during their in-hospital phase of up to 1 month. The average age of patients who died early was 58.8 years (mean of entire group 58.6 years). The remaining 13 deaths occurred outside the hospital 4% weeks to 18 months after the onset of myocardial infarction. The early and late deaths (with two exceptions) were of acute cardiac origin, or they were sudden and unexplained (table 1).

LHD and Acute Mortality

Among the 72 patients with an initially normal LHD (including patients with cardiogenic shock), 10% expired within 4 weeks, whereas among the 53 patients with an enlarged LHD, 18 patients (34%) expired acutely.

If the 10 patients with cardiogenic shock are omitted, the early mortality rates were three of 68 (4%) in the initially normal LHD group and 12 of 47 (26%) in the initially enlarged LHD group (P < 0.001).

Although shock and sudden death occurred in patients with or without an increased LHD, heart failure and other complicating causes of death occurred only in those with an enlarged LHD (table 1).
Representative changes in left heart dimension (LHD) of three patients studied following acute myocardial infarction and during recovery. (A) Patient exhibited a persistently normal LHD. (B) Illustrates an initially enlarged LHD that becomes normal within a few days; clinically both of these patients were well 2 years after myocardial infarction. (C) The patient showed an initially enlarged LHD which became transiently normal and then enlarged again. The patient had severe angina pectoris and congestive heart failure prior to his sudden death.

LHD and Late Mortality

Ninety-six of the 100 survivors of acute infarction have undergone follow-up for 2–31 months (table 2). In 62 patients, the LHD has become normal or remained normal; five patients (8%) have expired. Three died of acute cardiac causes, one suddenly, and two of recurrent myocardial infarction (table 1).

In 34 patients the LHD has become enlarged or remained abnormal; eight patients (24%) have expired, six suddenly and two of other cardiac causes (tables 1 and 2).

The differences in these two mortality rates are significant at the \( P < 0.05 \) level, and if two of the deaths (one surgical, one due to cerebrovascular accident) in the group with normal LHD are excluded to yield a 5% mortality, the rates are significantly different at the \( P < 0.01 \) level.

Morbidity

Of the patients who survived acute infarction and are alive at present with normal LHD, only one patient (who has severe angina pectoris) is in New York Heart Association classification III (table 2). In contrast are the 26 surviving patients with an enlarged LHD, 11 of whom (42%) are in class III with symptoms of severe congestive heart failure and/or angina pectoris. The difference in the incidence of class III symptoms in the normal enlarged LHD groups is significant at the \( P < 0.001 \) level.

Discussion

The incidence of cardiomegaly and its prognostic importance in acute myocardial infarction have been the subject of study for many years. Parkinson in 1936, using the cardiothoracic ratio, reported that...
Late (>4 wk) congestive failure; those that when left ventricular enlargement develops or persists, the mortality rates over the relatively short period studied are higher than those previously estimated using the overall heart size, whereas those with normal LHD are at low risk over this period. The method employed in the present study is considered to be more selective as well as simpler to apply than the methods discussed above, and it was shown previously to be more sensitive for detecting left ventricular enlargement than the cardiothoracic ratio.18

Our findings lend support to the value of analyzing the left ventricular response to acute myocardial infarction. Hemodynamic and other measures of left ventricular function, as well as clinical status, appear to correlate with the severity of the myocardial infarction,17 and the survival and clinical status of patients recovering from acute myocardial infarction depend to a large degree on the extent of myocardial necrosis.17, 27 Experimental, clinical, and pathologic studies support the view

### Table 1

**Causes of Death**

<table>
<thead>
<tr>
<th></th>
<th>Enlarged LHD</th>
<th>Normal LHD</th>
</tr>
</thead>
<tbody>
<tr>
<td>Early (&lt;4 wk)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Total number</td>
<td>53</td>
<td>72</td>
</tr>
<tr>
<td>Deaths, total</td>
<td>18</td>
<td>7</td>
</tr>
<tr>
<td>10 Shock</td>
<td>6</td>
<td>4</td>
</tr>
<tr>
<td>6 Sudden</td>
<td>3</td>
<td>3</td>
</tr>
<tr>
<td>6 CHF</td>
<td>6</td>
<td>0</td>
</tr>
<tr>
<td>2 Myocardial rupture</td>
<td>2</td>
<td>0</td>
</tr>
<tr>
<td>1 Septicemia</td>
<td>1</td>
<td>0</td>
</tr>
<tr>
<td>% Mortality:</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Including shock pts (%)</td>
<td>34</td>
<td>10</td>
</tr>
<tr>
<td>Excluding shock pts (%)</td>
<td>26</td>
<td>4</td>
</tr>
<tr>
<td>Late (&gt;4 wk)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Total number</td>
<td>34</td>
<td>62</td>
</tr>
<tr>
<td>Deaths, total</td>
<td>8</td>
<td>5</td>
</tr>
<tr>
<td>3 Acute MI</td>
<td>1</td>
<td>2</td>
</tr>
<tr>
<td>7 Sudden</td>
<td>6</td>
<td>1</td>
</tr>
<tr>
<td>1 CHF</td>
<td>1</td>
<td>0</td>
</tr>
<tr>
<td>1 CVA</td>
<td>0</td>
<td>1</td>
</tr>
<tr>
<td>1 Coronary artery surgery</td>
<td>0</td>
<td>1</td>
</tr>
<tr>
<td>% Mortality:</td>
<td>24</td>
<td>8</td>
</tr>
</tbody>
</table>

Abbreviations: CVA = cerebrovascular accident; CHF = congestive heart failure; pts = patients.

64% of 200 patients with acute myocardial infarction had enlargement of the heart but concluded that heart size after infarction bore no relation to the ultimate prognosis.15 Palmer employed the same method for assessing cardiac size and found that patients with large hearts lived almost as long as those with hearts of normal size.16 However, Bjerkelund19 and later Amundsen,20 using the Rohrer-Kahlstorf technic (Rohrer21 and later Kahlstorf22 calculated the volume of the heart from the frontal and sagittal orthodiagrams using an arealength method) concluded that a significant correlation existed between cardiac size and mortality in patients with acute myocardial infarction. In more recent studies both Norris and co-workers12, 13 and Waris et al.14 have affirmed that overall heart size is an important prognostic factor. The latter workers applied a modified Rohrer-Kahlstorf technic23 with frontal and lateral roentgenograms, and over a 5-year follow-up period they found that 53% of the patients in whom cardiomegaly was noted expired compared with 32% of those with normal heart size.14 A higher death rate was also noted if patients developed cardiac enlargement during the follow-up period.14

The present study analyzed patients serially throughout their early and late course and attempted to assess the size of the left ventricle alone. We found that in many patients the left ventricle becomes transiently or persistently enlarged after acute myocardial infarction. It was further found that when left ventricular enlargement develops or persists, the mortality rates over the relatively short period studied are higher than those previously estimated using the overall heart size, whereas those with normal LHD are at low risk over this period. The method employed in the present study is considered to be more selective as well as simpler to apply than the methods discussed above, and it was shown previously to be more sensitive for detecting left ventricular enlargement than the cardiothoracic ratio.18

Our findings lend support to the value of analyzing the left ventricular response to acute myocardial infarction. Hemodynamic and other measures of left ventricular function, as well as clinical status, appear to correlate with the severity of the myocardial infarction,17 and the survival and clinical status of patients recovering from acute myocardial infarction depend to a large degree on the extent of myocardial necrosis.17, 27 Experimental, clinical, and pathologic studies support the view

### Table 2

**Late Mortality and Morbidity (Survivors > 1 mo)**

<table>
<thead>
<tr>
<th>Type</th>
<th>Pts (no.)</th>
<th>Avg age (yrs)</th>
<th>Follow-up (mo.)</th>
<th>Avg LHD (mm/m²)</th>
<th>Deaths</th>
<th>NYHC</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal (≤ 52 mm/m²)</td>
<td>62</td>
<td>58.4</td>
<td>11.7 (2-31)</td>
<td>46</td>
<td>5</td>
<td>17</td>
</tr>
<tr>
<td>Enlarged (&gt; 52 mm/m²)</td>
<td>34</td>
<td>59.2</td>
<td>8.6 (2-28)</td>
<td>57</td>
<td>8</td>
<td>24</td>
</tr>
<tr>
<td>Total</td>
<td>96</td>
<td>59.2</td>
<td>8.6 (2-28)</td>
<td>57</td>
<td>13</td>
<td>14</td>
</tr>
</tbody>
</table>

Abbreviations: Avg LHD = average of the most recent left heart dimensions; NYHC = New York Heart Classification.
that an increased heart size is associated with more muscle damage and extensive coronary vascular disease. 24-26 Therefore, it seems logical that the left ventricular size should prove to be a relatively sensitive indicator of the magnitude of myocardial damage and thereby help to predict morbidity and mortality.

The factors responsible for in-hospital and late death following discharge from the coronary care unit have received some study, but the mechanisms remain largely unknown. A number of risk factors, such as major arrhythmias while under surveillance, 28 have been considered. 9,28-30 Some studies suggest that in the general population ventricular premature contractions and electrocardiographic evidence of left ventricular hypertrophy carry a substantially increased risk of sudden death. 31 In the patients in the present study who died suddenly during late follow-up, it seems possible that the increased left ventricular size, with or without associated left ventricular hypertrophy, 31 could have predisposed to the development of arrhythmias or to recurrent myocardial infarction.

The serial measurements of the LHD as described herein provide a practical means for assessment of the responses to acute therapy, and for objective follow-up after acute myocardial infarction. Although additional confirmation of the long-term prognostic significance of an enlarged LHD will be necessary, should the present trend continue, early aggressive management of such high risk patients undoubtedly will be considered.

Acknowledgment

The technical assistance of Mr. Joseph Byrne, Mr. Michael Mohl, and Miss Ardelle Troy is gratefully acknowledged.

References

1. Rosenbaum FF, Levine FA: Prognostic value of various clinical and electrocardiographic features of acute myocardial infarction. Arch Intern Med (Chicago) 68: 913, 1941
LV SIZE AFTER MI


26. EYSTER JAE: Experimental and clinical studies in cardiac hypertrophy. JAMA 91: 1881, 1928


Left Ventricular Size after Acute Myocardial Infarction: Serial Changes and their Prognostic Significance

WILLIAM J. KOSTUK, THOMAS M. KAZAMIAS, MARTIN P. GANDER, ALLAN L. SIMON and JOHN ROSS, JR.

Circulation. 1973;47:1174-1179
doi: 10.1161/01.CIR.47.6.1174

Circulation is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 1973 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/47/6/1174

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/