Evolution of Abnormalities in Left Ventricular Function after Acute Myocardial Infarction

By Martin I. Broder, M.D., and Jay N. Cohn, M.D.

SUMMARY

Twelve patients with acute myocardial infarction (AMI) were studied by left ventricular (LV) catheterization, echocardiography, and volume manipulation (tourniquets, phlebotomy, and dextran infusion) within 24 hours of infarction (study 1) and 3 weeks later (study 2). Three patients also were studied 6–10 months later (study 3). Cardiac index rose (from 2.50 to 3.15 liters/min/m²) and right atrial pressure fell (from 9.4 to 5.3 mm Hg) from study 1 to study 2. LV end-diastolic pressure (LVEDP) remained elevated (21.8 mm Hg at study 1 and 20.9 mm Hg at study 2) and was actually higher at study 2 in six patients who were otherwise improved. At study 1, end-diastolic ventricular diameter was within normal limits in five patients with clinical LV failure, four of whom had grossly elevated LVEDP. At study 2, ventricular diameter remained normal in three patients with elevated LVEDP but no LV failure. At study 3, LVEDP was still elevated in two patients and chamber diameter normal in two and slightly increased in one. Ventricular function curves (LVEDP vs stroke index) constructed from results during volume manipulation showed no consistent improvement during convalescence.

These data indicate that clinical improvement after AMI is not usually associated with a fall in LVEDP or a normalization of the impaired LV function curve. Normal ventricular diameter indicates normal ventricular volume which, in the presence of an elevated LVEDP, suggests a persistent decrease in LV compliance.

Additional Indexing Words:
Ventricular compliance  Ventricular volume  Ventricular function curves
Echocardiography

The abnormalities in systemic hemodynamics during the acute phase of myocardial infarction have been well described during the past two decades. Changes in cardiac output and systemic vascular resistance during the first week after infarction also have been reported. Recent studies in this laboratory using a bedside technic for left ventricular catheterization have revealed that left ventricular function is almost invariably impaired after acute myocardial infarction even in the absence of clinical signs of left ventricular failure. However, it is not known whether this abnormality is transient and reversible or persists during convalescence.

In the present study left ventricular catheterization was performed in the acute phase and again 3 weeks later in 12 patients with acute myocardial infarction. In a few patients,
studies were repeated again 6–10 months after the acute episode. Echocardiography was used in an attempt to relate left ventricular end-diastolic pressure to a left ventricular chamber dimension.

Material and Methods

Twelve male patients admitted to the Veterans Administration Hospital or Providence Hospital in Washington, D. C., were studied within 24 hours of onset of chest pain (study 1). The same 12 patients were restudied 3 weeks later (study 2). All had a typical history of ischemic cardiac pain accompanied by electrocardiographic evidence of acute transmural myocardial infarction and a typical pattern of change in serum enzymes. Patients with a known history of previous cardiomyopathy or left ventricular failure were excluded. Eight gave a history of previous hypertension without electrocardiographic evidence of left ventricular hypertrophy. Two patients had had previous myocardial infarction, but the other 10 had no clinical or electrocardiographic evidence of old myocardial infarction, although several had previous symptoms of angina pectoris (table 1). At study 1, all but one patient (no. 10) showed the clinical syndrome of mild-to-moderate left ventricular failure, with basilar rales, ventricular diastolic gallop rhythm, and varying degrees of breathlessness and orthopnea. No patients were in frank pulmonary edema at the time of study. Heart size could not be accurately evaluated because the admission chest X-rays were portable, but only two patients showed obvious pulmonary venous congestion.

Prior to the study most patients had received narcotics or sedatives as indicated for pain, and a few had received a diuretic, but none had been treated with digitalis. No patients were in shock either before or during study 1, although one had a transient episode of severe hypotension that cleared spontaneously. All patients gave written informed consent after the purpose of the study and its possible complications were described in detail.

During the 3 weeks between study 1 and study 2 most patients were given diuretics for the first several days but none thereafter. None received digitalis or propranolol during this period. At study 2, all patients were free of signs and symptoms of left ventricular failure and were much improved clinically. The heart was not enlarged by X-ray at this time in any of the patients. Hemodynamic studies produced no complications, and all patients were eventually discharged. Three patients were restudied 6–10 months later (study 3), not having received digitalis or propranolol during this time. A summary of the pertinent clinical characteristics of these patients is given in table 1.

Methods

All studies were performed either at the bedside in the medical intensive care unit or in an adjoining special procedure room. The right atrium was catheterized with a PE 160 catheter from an antecubital or femoral vein. Using a specially curved red Kifa catheter,8 the left ventricle was entered from the femoral artery using the Seldinger technic. Pressures were recorded using Statham P23Db and P23Bb strain gauge transducers positioned at the midchest and

Table 1

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*By history.

Abbreviations: AL = anterolateral; I = inferior; A = anterior; P = posterior; Ang pect = angina pectoris; AMI = acute myocardial infarction; Clin HF = clinical heart failure.
amplified and recorded on a direct-writing multichannel Hewlett-Packard or Waters recording system. Mean pressures were obtained electronically. Cardiac output (CO) was measured by the dye-dilution technic using an injection of 5 mg indocyanine green dye into the right atrium while sampling at a constant rate from the left ventricle or ascending aorta through a Gilford or Waters cuvette. Calculations of CO were made using either the standard Stewart-Hamilton method or a specially programmed Olivetti Programma 101 computer. Dye curves were obtained in duplicate or triplicate in most patients and were reproducible ±15%.

Left ventricular end-diastolic pressure (LVEDP) was measured either at the onset of the rapid ventricular pressure increase on a high-gain tracing or 0.04–0.05 sec after the QRS onset on a 100-mm/sec tracing. Mean ventricular pressure during diastole (LVMDP) was measured visually. Left ventricular dP/dt, the maximum rate of the rise of left ventricular pressure prior to aortic valve opening, was measured from pressure tracings recorded at a paper speed of 100 mm/sec after careful flushing of the arterial catheter. The catheter was withdrawn into the aorta periodically for measurement of aortic pressure and then was readvanced into the left ventricle. The frequency response of the catheter and recording system under ideal laboratory conditions is ±5% to 15 Hz. Since waveforms recorded with such a non-high-fidelity system may be distorted, dP/dt data may be unreliable.

Systemic vascular resistance (SVR) in dynes-sec-cm⁻⁵ was calculated from the formula:

\[ \frac{\text{MAP} - \text{RAP}}{\text{CO}} \times 1.332 \times 60, \]

where MAP is mean aortic pressure and RAP is mean right atrial pressure, both in mm Hg. Mean transit time (MTT) from right atrium to left ventricle or ascending aorta was calculated as the sum of the appearance time and the mean transit time of the dye-dilution curve, with correction for the delay time of the catheter-densitometer system. The central blood-volume index (CBVI) in ml/m² was calculated from the formula:

\[ \text{CBVI} = \frac{\text{MTT} \times \text{CI}}{60}, \]

where CI is the cardiac index in liters/min/m². Left ventricular stroke work index (LVSWI) in gm-m/m² was calculated from the formula:

\[ \text{LVSWI} = \frac{\text{MAP} - \text{LVEDP}}{1000} \times \text{SI} \times 13.6, \]

where SI = the stroke index (stroke volume corrected for body surface area).

Echocardiography was performed with a Smith-Kline Ekoline 20 ultrasonoscope using a transducer placed in the fourth interspace just to the left of the sternum. Measurements of left ventricular internal diameter were made on Polaroid photographs using calibration markers internally generated by the ultrasonoscope. An end-diastolic left ventricular internal diameter (LVIDd) from the left side of the interventricular septum to the endocardial surface of the posterior left ventricular wall was measured just before the QRS complex of a simultaneously recorded electrocardiogram. Although ventricular volume calculated from LVID is reliable, the validity of echocardiographic left ventricular volume measurements could be made in five patients. Venous occlusive tourniquets were then applied to both upper thighs and one upper arm, and in six patients measurements were repeated after mean RAP had fallen. In five others tourniquets produced an inadequate change in mean RAP, and a 500-cc phlebotomy was performed over a 15-min period, after which measurements were repeated. Five patients were allowed to return to a control state after release of the tourniquets and were then infused with low molecular-weight dextran in 100-cc increments, with continuous monitoring of intracardiac pressures. Measurements were repeated when pressures had appreciably risen, and the study was terminated. Two patients did not undergo volume manipulation during study 1.

In study 2 all 12 patients again had baseline measurements of aortic and right atrial pressures and CO, with left ventricular pressures successfully obtained in 10 subjects. Satisfactory left ventricular diameter measurements could be made in four patients. Six patients then underwent the volume manipulation sequence using tourniquets followed by dextran infusions, while in a seventh only tourniquets were applied. Five patients did not undergo volume alteration during study 2.
Table 2

Hemodynamic Data during Recovery from Acute Myocardial Infarction

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Mean 1  | 99.7   | 21.8    | 14.2         | 9.4          | 2.5         | 79.8             | 32.1            | 31.8      | 2039.7        | 579.8               | 1626.7      |

Mean 2  | 88.9   | 20.9    | 12.9         | 5.3          | 1.3         | 83.8             | 37.6            | 34.9      | 1816.5        | 573.6               | 1196.7      |

Mean 2  | 90.1   | 21.2    | 13.3         | 5.6          | 1.3         | 83.8             | 37.6            | 34.9      | 1816.5        | 573.6               | 1196.7      |

P value  | NS     | NS      | NS           | <0.002       | <0.01       | NS               | NS             | NS        | NS            | NS                  | <0.02       |
ABNORMALITIES AFTER AMI

There patients were restudied 6–10 months after acute myocardial infarction. In two of the tourniquet dextran sequence was followed, while the third only had tourniquets applied. Left ventricular diameter was measured in all three.

No complications attributable to the investigative procedures were noted. As previously reported, fluoroscopy was only rarely necessary to cross the aortic valve. Several ectopic ventricular beats usually signaled the entrance of the catheter into the left ventricle. The ectopic beats always disappeared promptly, and no other arrhythmias appeared during the course of the study. No antiarrhythmic drugs were administered prior to left ventricular catheterization.

Results

Studies 1 and 2

Hemodynamic data from all three studies are shown in table 2. All values represent control measurements before volume manipulation. The mean and standard error of the mean are indicated, and P (probability) values were calculated using Student’s t-test for paired samples.

Cardiac index (CI) ranged from 1.52 to 3.81 liters/min/m² during the first study with a mean of 2.50 ± 0.19 liters/min/m². Three weeks later, it had risen to an average of 3.15 ± 0.20 (range 2.42–4.70 liters/min/m²) (P < 0.02).

LVEDP for all 12 patients averaged 21.8 ± 2.7 mm Hg during study 1 (range 9–36 mm Hg) and was elevated (over 12 mm Hg) in 10 of the 12. At study 2, mean LVEDP averaged 20.9 ± 2.4 mm Hg in the 10 patients in whom follow-up left ventricular pressures could be obtained. LVEDP during study 1 in these same 10 patients averaged 20.7 ± 3.1 mm Hg. Indeed, in six of these 10 patients LVEDP was found to have risen between study 1 and 2, despite obvious clinical improvement associated with a higher CI and lower RAP in each case.

Insignificant negative correlations (r = -0.38 in study 1 and -0.45 in study 2) were noted between LVEDP and CI. In seven patients changes in LVEDP and CI from study 1 to study 2 were concordant, i.e., CI rose if LVEDP was higher and CI fell if LVEDP was lower. In two patients directional changes in CI were opposite to changes in LVEDP, and in one CI remained the same despite a considerably lower LVEDP at study 2.

The relationship between stroke volume and LVEDP usually did not change from study 1 to 2 (fig. 1). In nine of the 10 patients in whom data were available, directional changes in stroke volume and LVEDP were similar, making it impossible to document a change in LV function by this method during the first 3 weeks after infarction. In one patient (no. 11) left ventricular function appeared to be improved because of a higher stroke volume and lower LVEDP at study 2.

Mean arterial pressure (MAP) at study 1 ranged from 50 to 132 mm Hg with a mean of 99.7 ± 6.8 mm Hg. The lowest value was a transient finding in patient 6, whose arterial pressure spontaneously returned to normal levels. At study 2, MAP had fallen to a mean of 88.9 ± 3.2 mm Hg (range 70–112 mm Hg). The decrease was not significant. A positive correlation (r = 0.60; P < 0.05) was noted between LVEDP and MAP at study 1 but was not significant (r = 0.31) at study 2 (fig. 2).

Patients with a history of hypertension were scattered through the entire range of values of

![Figure 1](http://circ.ahajournals.org/)

Relationship in 10 patients between left ventricular end-diastolic pressure (LVEDP) and stroke index during the acute phase of myocardial infarction (study 1) and 3 weeks later (study 2).
MAP and LVEDP and did not spuriously bias the correlation.

Mean right atrial pressure (RAP) decreased significantly from study 1 to study 2, from an initial average of $9.4 \pm 1.2$ mm Hg (range 3-16 mm Hg) to $5.3 \pm 0.7$ mm Hg (range 1-9 mm Hg) ($P < 0.002$). The one patient (no. 10) with no clinical evidence of heart failure when first studied had the lowest RAP at that time, with no change 3 weeks later. Despite the absence of clinical evidence of either left or right ventricular failure at study 2, three patients with inferior wall myocardial infarctions still had modest elevations of RAP at that time. During study 1, RAP correlated poorly with LVEDP ($r = 0.39$), but a stronger correlation was seen in study 2 ($r = 0.70; P < 0.01$) (fig. 3). However, for any given patient there was no consistent relationship between directional changes in RAP and LVEDP from study 1 to study 2.

No significant changes between studies 1 and 2 occurred in LVMSP, heart rate (HR), stroke index (SI), or LVSWI. Left ventricular $dP/dt$ fell slightly from $2040 \pm 196$ mm Hg/sec in study 1 to $1817 \pm 115$ mm Hg/sec in study 2, but the change was not significant. CBVI remained virtually constant ($579 \pm 40$ ml/m² in study 1 and $573 \pm 44$ ml/m² in study 2). SVR fell significantly ($P < 0.02$) from $1627 \pm 223$ to $1197 \pm 83$ dyne-sec-cm⁻² although much of the decrease was contributed by the fall of the extremely high SVR noted in one patient in study 1. MTT from the right atrium to either the left ventricle or ascending aorta showed a barely significant decrease ($P < 0.05$), falling from $14.7 \pm 1.2$ sec to $12.1 \pm 1.1$ sec.

Twelve technically satisfactory measurements of LVID were made in eight patients (table 3). In three patients chamber dimension was measured in both studies 1 and 2. In two, satisfactory photographs could be obtained only in study 1, and in the other three only in studies 2 and/or 3. In study 1 all five patients studied exhibited a diastolic left ventricular internal diameter within the normal range, despite a clinical picture of heart failure in all five and grossly elevated LVEDP (22-36 mm Hg) in four (fig. 4). Three weeks later LVID again was normal despite elevated
LVEDP. At this time no patients had clinical signs of congestive heart failure. In the subjects in whom sequential data are available no consistent change was noted in the relationship between LVEDP and LVID to allow conclusions regarding shifts of compliance in the first 3 weeks after infarction.

Study 3

Patients 1, 4, and 5 (table 2) were restudied 10, 9, and 6 months, respectively, after infarction (in each case their first). All had returned to their previous occupations without significant cardiorespiratory symptoms or disability. At study 3 all hemodynamic parameters were normal except for MAP, which was modestly elevated in all three patients, and LVEDP, which was elevated in two. Patient 1 had an LVEDP of 24 mm Hg as compared to a study 1 value of 34 mm Hg, and his LVIDd was slightly increased (table 3). The MAPs at both studies were virtually identical. Patient 4, whose LVEDP previously had been recorded at 16 and 18 mm Hg had an LVEDP of 12 mm Hg with a normal LVIDd. Patient 5, however, whose LVEDP had fallen during hospitalization from 34 to 16 mm Hg as his MAP had fallen from 120 to 84 mm Hg, had an LVEDP of 36 mm Hg and an MAP of 112 mm Hg. His LVIDd was normal. This latter patient died suddenly several months after study 3, the only death in this series.

Response to Alterations in Preload

Acute volume depletion and/or expansion was utilized in 10 patients during study 1 in order to analyze in more detail their left ventricular function curves (fig. 5). In most patients stroke volume rose as LVEDP increased and fell as LVEDP was reduced. The ventricular function curves were rather flat, since large changes in LVEDP were associated with only small changes in stroke volume. In three patients (9, 11, and 12), however, the ventricle responded to reduction in LVEDP with a rise in stroke volume. In six patients (4, 5, 7, 8, 10, and 11), similar volume manipulations were carried out in the follow-up studies. When cardiac function curves from different studies in the same patient were compared, no consistent pattern emerged. In four of six patients in whom complete data were available (4, 7, 8, and 11) the curves from studies 1 and 2 were almost identical and indicated no significant change in ventricular function between the two studies.

The response to change in preload was particularly revealing in patient 11, whose resting data suggested improvement in left ventricular function between study 1 and 2. At both studies, however, this patient appeared to be operating on a paradoxic function curve in which an increase in LVEDP was associated with a fall in stroke volume. The improvement in ventricular performance at the time of study 2 may therefore have been more apparent than real, since the upper end of the function curve during study 2 could be superimposed on the function curve from study 1. In patient 5 ventricular function apparently worsened by the time of study 2, as shown by a decreased stroke volume at virtually the same LVEDP; yet the isolated finding of a lower baseline LVEDP at study 2 might have been interpreted as reflecting improved ventricular function. Conversely, the resting LVEDP in patient 10 was considerably higher during study 2 than in study 1, an observation which might have been interpreted as a worsening in LV function; but function curves described during volume manipulation indicated that left ventricular function actually had improved.

At study 3 the function curve in patient 5

<table>
<thead>
<tr>
<th>Study 1</th>
<th>Study 2</th>
<th>Study 3</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pt</td>
<td>LVIDd (cm)</td>
<td>LVEDP (mm Hg)</td>
</tr>
<tr>
<td>1</td>
<td>4.4</td>
<td>15</td>
</tr>
<tr>
<td>6</td>
<td>4.7</td>
<td>22</td>
</tr>
<tr>
<td>8</td>
<td>4.9</td>
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<tr>
<td>9</td>
<td>5.4</td>
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<tr>
<td>11</td>
<td>5.4</td>
<td>36</td>
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<tr>
<td>12</td>
<td>5.0</td>
<td>24</td>
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</table>
documented persistence of the severe abnormality in ventricular performance. Sudden death occurred in this patient 3 months after study 3. In contrast, ventricular function in patient 4 was markedly improved at the time of study 3.

Ventricular function curves for all three studies were also plotted using left ventricular stroke work rather than stroke index. No significant differences were noted in the shape or position of the curves.

Discussion

The 12 patients evaluated in this study all exhibited considerable impairment of left ventricular function at the time of their initial catheterization within 24 hours of their acute myocardial infarction. Eleven presented with clinical signs usually associated with left ventricular failure, and in 10 the left ventricular end-diastolic pressure was elevated above the normal range. In the two patients whose initial LVEDP was normal (9 and 10 mm

Figure 4

Echocardiogram demonstrating normal left ventricular diameter in patient 6 at a time when his left ventricular end-diastolic pressure was 22 mm Hg.
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Hg), modest volume loading increased LVEDP to abnormal levels (21 and 25 mm Hg), and both had elevated resting LVEDP when restudied 3 weeks later. Since none of the subjects had a previous history of heart failure or evidence of preexisting cardiomegaly it is likely that the left ventricular dysfunction was the direct result of the acute myocardial infarction.

Elevated left ventricular filling pressures have previously been documented in most patients with acute myocardial infarction even in the absence of clinical signs of left ventricular failure. The data from the present study demonstrating an average cardiac index of 2.50 liters/min/m² during the acute phase are also in agreement with results from other series in which the cardiac output in patients with myocardial infarction without shock has usually been found to be at the lower limits of normal.1–5 As previously shown,7 RAP was only modestly correlated with LVEDP at the onset of infarction, and the absolute value of the RAP did not serve as a useful guide to the magnitude of elevation of LVEDP. Others have shown a poor correlation between RAP and simultaneously measured pulmonary capillary wedge pressure immediately following acute myocardial infarction.12

An elevation of LVEDP in patients with acute myocardial infarction is often thought to indicate an accompanying increase in LVEDV as the ventricle dilates in an attempt to maintain stroke output by the Frank-Starling mechanism. Measurements of LVEDV in other cardiac conditions in man by angiography,13 thermodilution,14 or dye dilution,15 or in normal dogs using tantalum screws,16 however, have failed to show a consistent relationship between LVEDV and LVEDP.

Figure 5
Ventricular function curves described from data obtained during volume depletion and volume expansion in 12 patients during the three study periods.
Indeed, angina produced by exercise or atrial pacing has been shown to be accompanied by an increase in LVEDP and a decrease in LVEDV. Experimental acute myocardial infarction in the dog is characterized by a rise in LVEDP with no change in LVEDV. In these cases the rise in LVEDP is thought to reflect a decrease in diastolic compliance of the left ventricle. This altered distensibility could be due either to localized or generalized myocardial anoxia or to a stiffening of the infarcted zone after the period of aneurysmal bulging of the infarct subsides.

In the present study left ventricular internal diameter was measured by an ultrasonic technic. Technically satisfactory photographs demonstrating both the posterior wall of the left ventricle and the left side of the interventricular septum could not be obtained in all cases. This failure could have been related in part to dyskinetic or akinetic areas in the infarcted myocardium or in surrounding ischemic zones making it difficult to detect the typical motion of the septum or posterior left ventricular wall. Nonetheless, in those patients in whom satisfactory tracings could be obtained, the left ventricular internal diameter during diastole was not enlarged despite considerable elevations in LVEDP. Volume calculated from this measurement by formulae previously validated in other clinical situations was within the normal range of 50–90 ml/m². These data strongly suggest that the elevated LVEDP observed so frequently in acute myocardial infarction is due more to a decrease in left ventricular compliance than to enlargement of the chamber. However, in the absence of comparable data on compliance characteristics of normal ventricles this conclusion must be tentative.

The subsidence of symptoms of heart failure and the rise in cardiac output and fall in right atrial pressure during the first 3 weeks of convalescence of these patients, each of whom had a relatively uncomplicated hospital course, is consistent with previous sequential studies in acute myocardial infarction. Despite this clinical and hemodynamic improvement, however, left ventricular end-diastolic pressure did not fall and actually was higher 3 weeks after infarction in six of the 10 patients in whom data were obtained. In three of these patients a diuretic administered prior to the initial study might have falsely reduced LVEDP, but in none of the other acute studies nor in any of the patients studied 3 weeks after infarction was therapy which could have altered resting LVEDP administered. Therefore, the elevated LVEDP and normal ventricular diameter noted at the time of the second study must be viewed as evidence for continued impairment of diastolic distensibility. Reduced ventricular compliance has also been demonstrated to persist 6–8 weeks after experimental myocardial infarction in the dog.

The consistent fall in RAP without an accompanying fall in LVEDP further attests to the unreliability of RAP as an index of LVEDP during the 3 weeks following acute myocardial infarction. However, the RAP seemed to serve as a better guide to clinical improvement than either the LVEDP or LV mean diastolic pressure. The disappearance of the clinical and radiographic signs of pulmonary congestion without a change in LVEDP or central blood volume suggests that factors other than pulmonary blood volume and pulmonary venous pressure may be important in the genesis of pulmonary edema.

The correlation noted between arterial pressure and LVEDP, particularly during study 1, suggests that aortic pressure may be a significant determinant of LVEDP after acute myocardial infarction. Indeed, the only three patients whose LVEDP fell significantly from study 1 to study 2 exhibited a concomitant fall in MAP between the two studies. This relationship between LVEDP and MAP is not surprising, since afterload is an important determinant of left ventricular performance. An increase in arterial pressure during sustained handgrip is associated with a rise in LVEDP in patients with heart disease, and pharmacologic reduction in arterial pressure is accompanied by a fall in LVEDP in patients with acute myocardial infarction.
An understanding of the evolution of left ventricular dysfunction after acute myocardial infarction is of vital importance in establishing the rationale for drug therapy, mechanical cardiac assistance, and direct surgical intervention. Function curves relating left ventricular filling pressure to stroke volume or stroke work have been widely used as a guide to myocardial performance but a single measurement of left ventricular pressure and cardiac output provides limited information about the shape and position of the function curve at that moment. Therefore, preload was acutely altered both by volume depletion and volume expansion in the patients in this series in order to obtain data adequate to construct left ventricular function curves in the acute as well as convalescent periods.

In the first 24 hours after infarction most patients exhibited relatively flat function curves, but in seven of 10 patients stroke volume varied directly with LVEDP, even at LVEDPs over 25 mm Hg in five patients. In the other three, however, an apparent descending limb of the function curve was observed when LVEDP was at 25, 30, and 36 mm Hg, respectively. These results are somewhat similar to those reported by Russell and associates, but the latter group concluded that 25 mm Hg usually represented the LVEDP at which cardiac output peaked. A higher LVEDP could support a higher output in half the subjects in our series. Kumar and associates have produced function curves with descending limbs in dogs with experimental acute myocardial infarction and LVEDPs greater than 35 mm Hg. Since a descending limb of a ventricular function curve probably is not physiologically possible, the decrease in output observed at higher filling pressures in some subjects should probably be viewed as a shift to a less favorable function curve as a result of the increase in ventricular wall tension induced by a larger ventricular volume.

Left ventricular function assessed by such function curves was remarkably unchanged during convalescence from acute myocardial infarction in most of these patients. Significant improvement by 3 weeks could be demonstrated in only one or at most two patients, and by 6 months in only one of three patients. Indeed, one subject with a “descending limb” curve during the acute phase exhibited the same response to volume manipulation at the 3-week study, although he was clinically much improved. One patient whose function showed deterioration from the first to second study demonstrated no improvement 8 months after his infarct and died suddenly 11 months after the first study.

These data therefore must be interpreted to mean either that left ventricular function usually does not improve during convalescence from an acute myocardial infarction or that the relationship between left ventricular end-diastolic pressure and stroke volume or stroke work does not provide a reliable index of left ventricular function in this setting. Increased sympathoadrenal discharge during the acute phase could have masked subsequent improvement by causing increased contractility of noninvolved myocardium and shifting the initial function curve to the left. Since collagen deposition has not yet produced a dense scar by the third postinfarction week, dyskinesia of the infarcted area could have contributed to continued impairment of left ventricular function even if some previously ischemic zones had recovered function. Furthermore, the patients had undergone a 3-week period of limited physical activity between study 1 and study 2, and this cardiovascular deconditioning could have depressed the left ventricle response to volume manipulation.

Several factors may render the left ventricular end-diastolic pressure a poor guide to overall left ventricular function. Nonhomogeneity of myocardial structural changes could certainly alter the relationship in different areas of the ventricle between myocardial fiber length and intraventricular pressure at the end of diastole. In the acute phase alterations in compliance may represent a response to hypoxia, whereas at the time of the second study scar formation may be an added factor in reducing distensibility.
addition, however, considerable left ventricular hypertrophy may already have developed within the 3-week convalescence, and this increase in myocardial mass could account for an overall improvement in cardiac performance not reflected in changes in left ventricular end-diastolic pressure or volume.

Acknowledgment

The valuable assistance of Misses Eleanor Garlisi and Helen Firmin is gratefully acknowledged.

References

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Evolution of Abnormalities in Left Ventricular Function after Acute Myocardial Infarction

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Circulation. 1972;46:731-743
doi: 10.1161/01.CIR.46.4.731

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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