Assessment of Ventricular Function after Acute Myocardial Infarction by Plasma Volume Expansion

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SUMMARY
In order to see if changes in preload could be used to help assess left ventricular function, 24 patients with uncomplicated acute myocardial infarction (AMI) received plasma volume expansion (PVE). In eight patients (group A) cardiac index (CI) increased by 20% or more, and in 16 patients (group B) CI increased by less than 20% or decreased. By plotting left ventricular stroke-work index (LVSWI) against left ventricular end-diastolic pressure (LVEDP) before and after PVE, the ventricular function curves upon which the heart was operating could be assessed. In group A patients the values moved upward and to the right, while in group B the values during PVE moved horizontally or downward and to the right suggesting that at rest these hearts were operating at the peak of their function curves.

Patients in group B had a higher incidence of anterior infarction and a lower control mean arterial pressure than patients in group A, but other clinical and control hemodynamic values did not differ between the two groups. Follow-up data suggest that patients in group B may have had a higher mortality within the first 6 months following AMI.

Changing preload by PVE appears to be a safe and potentially useful means of assessing ventricular function following AMI and deserves further study.

Additional Indexing Words:
- Ventricular function curve
- Cardiac output
- Preload
- Left ventricular end-diastolic pressure
- Left ventricular peak dp/dt
- Systolic time intervals
- Maximal velocity of contractile element shortening
- Left ventricular stroke work

Since the reduction in death due to arrhythmias, shock and cardiac failure have become the most common cause of in-hospital mortality in patients with acute myocardial infarction (AMI). In addition, patients who survive the acute episode frequently show significant ventricular impairment several weeks later. Prevention of acute pump failure and late disability might be possible if during the acute phase of myocardial infarction the extent of myocardial damage can be limited using counterpulsation to reduce ventricular work and improve coronary blood flow. Before this can be tested in man, however, a more accurate method of defining left ventricular dysfunction early in AMI must be sought.

Hemodynamic studies of patients with AMI have been useful in separating patients into different prognostic groups. Patients with marked reduction in left ventricular work have been shown to have a higher mortality than patients in whom this parameter is normal or only slightly reduced following AMI.

In some patients, however, reduced cardiac output and work indices may be the result of inadequate ventricular filling rather than extensive ventricular damage. In addition, hemodynamic measurements in patients with severe myocardial damage may give values which overlap with values obtained in patients in whom ventricular damage is of lesser degree.

The purpose of this study has been to determine if hemodynamic measurements used to assess ventricular function after AMI could be made more sensitive by using plasma volume expansion to alter preload as to define the ventricular function curve on which the damaged heart is operating.

Methods
Patients admitted to the coronary care unit of the Cook County Hospital with a diagnosis of AMI were examined.
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evaluated. Informed consent was obtained from each patient prior to the study. Patients with shock, clinical congestive failure, or hemodynamically significant arrhythmias were excluded. Patients with control central venous pressures over 10 mm Hg or with left ventricular end-diastolic pressure above 20 mm Hg (except for one patient) were not given plasma volume expanders. The diagnosis of AMI was substantiated by history, serial ECG, and/or serial enzyme changes in all cases.

Studies were conducted in a specially equipped hemodynamic study area adjacent to the coronary care unit. Mean arterial pressure (MAP) was measured from an indwelling catheter or plastic cannula introduced percutaneously into the right brachial or radial artery. Central venous pressure (CVP) was measured through a standard intracatheter advanced from a superficial arm vein into the superior vena cava or right atrium. Pulmonary artery end-diastolic pressure (PAPD) measured just prior to the rapid systolic upstroke was obtained in most patients from a no. 5F Teflon end-hole catheter advanced under fluoroscopic guidance into the left ventricle following percutaneous insertion into the right brachial or femoral artery. All catheters were connected to Statham P23 Db transducers. Mean pressures were derived electronically.

Cardiac output (CO) was measured by averaging the values derived from two or more indicator-dilution curves obtained following injection of indocyanine green dye into the central circulation and sampling arterial blood by continuous withdrawal through a Waters densitometer using a Harvard constant-infusion withdrawal pump. Cardiac index (CI) was calculated by dividing the CO by the body surface area derived from the nomogram of DuBois. Stroke volume (SV) was calculated by dividing CO by the heart rate (HR) which was determined from a continuously monitored ECG lead. Systemic vascular resistance (SVR), in mm Hg/liter/min, was calculated as:

\[
SVR = \frac{MAP - CVP}{CO}
\]

Left ventricular stroke-work index (LVSWI), in g-m/beat/m², was obtained by the formula:

\[
LVSWI = CI (LVSP - LVEDP) \times 13.6
\]

where LVSP = mean left ventricular systolic pressure determined by planimetry.

Left ventricular peak rate of pressure rise (LV dp/dt) was recorded using a precalibrated differentiating circuit. An estimate of maximal velocity of contractile element shortening (Vmax) was determined by analysis of three or more left ventricular pressure and derivative complexes recorded at a paper speed of 200 mm/sec, using the two-component model and a series elastic constant of 32.7. Since left ventricular pressures were obtained from a fluid-filled catheter the values obtained for LV dp/dt and Vmax must be considered as approximations only. When assessing directional changes in the same patient following therapeutic interventions we have found that results obtained from a fluid-filled catheter are similar to results obtained using a catheter-tip transducer.8

Systolic time intervals (STI) were measured according to the method of Weissler and co-workers9 using a Hewlett-Packard no. 62-1500-C16 dynamic microphone for recording of heart sounds and a Hewlett-Packard no. 21051D pulse-wave pickup for recording the external carotid pulse. The QS2 is the time from the Q wave of the ECG to the first high-frequency component of the second heart sound. Left ventricular ejection time (LVET) was measured from the onset of the external carotid pulse to its incisura. In some patients the central aortic pressure pulse measured directly was substituted for the external carotid pulse. This substitution has been shown not to influence the results significantly.10 The pre-ejection period (PEP) was derived by subtracting the LVET from the QS2.

QS2 - LVET, and PEP were calculated from the STI and the HR using the regression equations of Weissler et al.9 Mean rate of left ventricular ejection (MRE) was calculated as the ratio of SV to LVET.

Following control hemodynamic measurements plasma volume expansion (PVE) with saline (four patients) or low molecular weight dextran (LMWD) (20 patients) was accomplished by infusion of 100-cc increments while monitoring the CVP or LVEDP. The infusion rate averaged 6.5 cc/min (range 2-12 cc/min). Repeat hemodynamic measurements were made after at least 400 cc of volume had been given, or after CVP, PAPD, or LVEDP had risen by at least 6 mm Hg.

Significance was determined by the Student t test for paired or unpaired data. A P value of greater than 0.05 was considered to be not significant (ns).

Results

The 24 patients studied have been grouped according to their CI response to PVE. Eight patients (group A) each increased CI by 20% or more for a mean change in CI of 46%. In each of 16 patients (group B) CI changed by less than +20%, and the average change for this group was +1%.

The average time from the onset of acute myocardial infarction to the onset of the study was not significantly different between the two groups, averaging 1.8 days in group A and 2.1 days for group B. Six of the eight patients in group A and 12 of the 16 group B patients were studied within 2 days of AMI, and the remaining patients were studied 3 and 5 days (group A) and 3, 3, 4, and 7 days (group B) after the onset of AMI.

Clinical Data

Certain clinical features were compared between these two groups and are listed in table 1. The groups were not different with regard to age, sex distribution, or the presence of previous hypertension, diabetes mellitus, myocardial infarction, or
angina pectoris. Vomiting was associated with the acute episode in three group A and in four group B patients. Two patients in group A and four in group B were considered to have nontransmural infarction. Inferior wall myocardial infarction was present in five patients from each group, while anterior infarction occurred in six patients from group B and one patient from group A. Patients in group B had higher peak values for CPK and SGOT than did patients in group A; however, differences between the mean values did not achieve statistical significance. Arterial blood pO₂ during room air breathing was measured in all but two patients from each group and was not significantly different, averaging 69 ± 4 mm Hg (mean ± SEM) for group A patients and 63 ± 3 mm Hg for group B patients. Arterial pO₂ measured in 13 patients (six in group A and seven in group B) following PVE was not significantly changed from control values. Group A patients received 550 cc of volume (range 100–1800 cc), which was not significantly different from the volume given to group B patients which averaged 350 cc (range 100–1000 cc). No patient in group A and one patient in group B died during the initial hospitalization. Follow-up data are available in six group A patients and all group B patients. All six group A patients were alive 6 months following hospital discharge, whereas six of the 15 group B patients who were discharged from the hospital died within 6 months. Subsequently, three group A patients and two group B patients have expired.

**Hemodynamic Studies**

The results of measurements obtained during the control period and following PVE in each patient are listed in table 2.

**Control Values**

Mean control values (table 3) were compared between the two groups. Patients in group A had a control MAP of 105 ± 3 mm Hg which was significantly higher than the control MAP for group B patients which averaged 93 ± 3 mm Hg (P < 0.05). SVR was 27 ± 4 mm Hg/liter/min in group A and 21 ± 2 mm Hg/liter/min in group B, but this difference was not significant. Patients in group A tended to have lower control values for CVP, PADP, LVEDP, and MRE; however, the differences between the mean values for the two groups were not statistically significant. Mean control values for CI, SV, HR, LVSWI, peak LV dp/dt, Vmax, and systolic time intervals were similar in both groups.

**Hemodynamic Changes following PVE**

HR was not significantly changed in either group. Therefore, changes in SV were similar to changes in CI and averaged +38 ± 7% for group A patients and –3 ± 3% for group B patients. Increases in MAP occurred in both groups averaging +15 ± 4% for group A (P < 0.02) and +6 ± 3% for group B (P < 0.05). SVR fell by 21 ± 4% in group A (P < 0.01) but increased by 2 ± 5% in group B (NS).

**Left Ventricular Response to PVE**

Left ventricular pressure was measured directly in 13 patients, and in 12 patients (five group A and seven group B) this measurement was made both prior to and following PVE. Figure 1 shows a plot of the LVEDP-LVSWI relationship for each group prior to and following PVE. During the control period, the five patients in group A had an LVEDP of 9 ± 2 mm Hg and an LVSWI of 36 ± 4 g-m/beat/m². Following PVE, the LVEDP-LVSWI plot had shifted upward and to the right, and the resultant mean values were LVEDP 24 ± 4 mm Hg and LVSWI 54 ± 3 g-m/beat/m². Prior to PVE the seven patients in group B had an LVEDP of 15 ± 2 mm Hg and an LVSWI of 33 ± 5 g-m/beat/m². Following PVE the mean LVEDP-LVSWI plot had shifted downward and to the right with the LVEDP

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**Table 1**

Clinical and Laboratory Data for Group A and Group B Patients

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Group A</th>
<th>Group B</th>
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<tr>
<td>Age (yr)</td>
<td>58 (41–89)</td>
<td>56 (32–74)</td>
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<tr>
<td>Sex, M</td>
<td>3 (37%)</td>
<td>8 (50%)</td>
</tr>
<tr>
<td>F</td>
<td>5 (63%)</td>
<td>8 (50%)</td>
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<td>History of:</td>
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<td>Hypertension</td>
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<td>11 (69%)</td>
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<td>Diabetes mellitus</td>
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</tr>
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<td>Prior infarction</td>
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<td>3 (19%)</td>
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<tr>
<td>Angina pectoris</td>
<td>4 (50%)</td>
<td>4 (25%)</td>
</tr>
<tr>
<td>Vomiting</td>
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<td>4 (25%)</td>
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<td>Site of Infarction (ECG):</td>
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<tr>
<td>Nontransmural</td>
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<td>4 (25%)</td>
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<tr>
<td>Inferior</td>
<td>5 (62%)</td>
<td>5 (32%)</td>
</tr>
<tr>
<td>Anterior</td>
<td>1 (13%)</td>
<td>6 (37%)</td>
</tr>
<tr>
<td>Lateral (only)</td>
<td>0 (0%)</td>
<td>1 (6%)</td>
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<td>Peak CPK (units)*</td>
<td>181 ± 37</td>
<td>308 ± 94</td>
</tr>
<tr>
<td>Peak SGOT (units)f</td>
<td>76 ± 11</td>
<td>165 ± 47</td>
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</table>

*Wrightman-Franklin.

†International.
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in: 50
Three patients
mean LVSWI-LVEDP (av-
ations) following PVE. In this and subsequent figures values for group A patients are represented by solid circles and for group B patients by open circles. Note that the plot of mean LVSWI-LVEDP for group A patients moved upward and to the right, and for group B patients this relationship moved downward and to the right.

averaging 22 ± 2 mm Hg and LVSWI of 30 ± 4 g.m/beat/m².

Changes in peak LV dp/dt following PVE were measured in five group A and seven group B patients. Three patients in group A and four patients in group B increased peak LV dp/dt; however, mean changes were not significant for either group.

Changes in V_max after PVE were recorded in three group A and six group B patients. V_max increased in one patient from each group and decreased in the remainder; however, mean changes were not significant.

Systolic Time Intervals

Systolic time intervals before and following PVE were measured in six group A and 11 group B patients. Mean values are shown in Figure 2. Following PVE, QSv increased 16 ± 6 msec in group A (NS) and 17 ± 6 msec in group B (P < 0.02). LVETc increased 26 ± 10 msec in group A (P < 0.05) and 22 ± 5 msec in group B (P < 0.01). PEP, decreased 13 ± 5 msec in group A (NS) and 5 ± 5 msec in group B (NS). The PEP/LVET ratio decreased from 0.46 to 0.36 in group A (P < 0.05) and from 0.45 to 0.39 in group B (P < 0.05). Figure 3 shows values for MRE which increased by 50 ± 12 cc/sec in group A (P < 0.02) and decreased by 34 ± 14 cc/sec in group B (P < 0.02).

Ventricular Filling Pressures

A total of 10 patients had simultaneous measurement of CVP and LVEDP during PVE. Changes for each patient are shown in figure 4. During PVE, CVP increased by 5.2 ± 0.6 mm Hg (P < 0.01) while LVEDP increased by 11.4 ± 1.9 mm Hg (P < 0.01).

Figure 1

Mean changes in LVSWI and LVEDP (see text for abbreviations) following PVE. In this and subsequent figures values for group A patients are represented by solid circles and for group B patients by open circles. Note that the plot of mean LVSWI-LVEDP for group A patients moved upward and to the right, and for group B patients this relationship moved downward and to the right.

Figure 2

Systolic time intervals before and after PVE. Note that similar increases in LVETc and decreases in PEP/LVET occurred in both groups in spite of the fact that only group A patients had large increases in SV following PVE.

Figure 3

Mean rate of left ventricular ejection (MRE) before and after PVE. Note that patients in group A increased MRE whereas patients in group B decreased MRE. Although average control MRE was higher in group B than in group A, the difference was not statistically significant.
### Table 2

**Hemodynamic Responses to Plasma Volume Expansion**

<table>
<thead>
<tr>
<th>Pt</th>
<th>Vol (cc)</th>
<th>CI (liters/min/m²)</th>
<th>SV (cc)</th>
<th>MAP (mm Hg)</th>
<th>CVP (mm Hg)</th>
<th>HR (beats/min)</th>
<th>SVET (ml/liter/min)</th>
<th>PADP (mm Hg)</th>
<th>LWVET (beats/m²)</th>
<th>LV (mm Hg)</th>
<th>V max (ml/sec)</th>
<th>QS (msec)</th>
<th>LVET (msec)</th>
<th>PEP (msec)</th>
<th>PEP/LVET (msec)</th>
<th>MRE (cc/sec)</th>
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<td>E.J.</td>
<td>1800*</td>
<td>C</td>
<td>1.5</td>
<td>26</td>
<td>120</td>
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<td>31</td>
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</tbody>
</table>
In nine patients (fig. 5) simultaneous measurements of PADP and LVEDP were obtained during PVE. Following PVE, PADP had increased by $9.1 \pm 0.9$ mm Hg and LVEDP had increased by $11.4 \pm 2.2$ mm Hg ($P < 0.01$).

**Adverse Effects**

No patient developed signs of acute pulmonary edema or congestion during the study. One patient (W.L.) with extensive inferior and lateral infarction suddenly developed hypotension terminating in asystole shortly after completion of the study. Resuscitation attempts were not successful, and he was the only patient among the 24 studied who expired during the initial hospitalization. Another patient (J.H.) developed chest pain after receiving 100 cc of LMWD. The pain responded promptly to analgesics and did not return. No further dextran was given.

**Discussion**

A basic hypothesis in dividing our patients into two groups is that, as a group, the 16 patients in group B whose CI failed to increase by 20% or more following PVE had worse ventricular function and, therefore, more ventricular damage than the eight patients in group A. Although the percentage of patients dead after 1 year was similar in both groups, each of the six group A patients for whom follow-up data were available lived for at least 6 months, whereas seven group B patients expired within 6 months of the study.

Of the clinical parameters listed, only the presence of anterior infarction was of help in predicting how the patient would respond to PVE.

---

**Figure 4**

Changes in CVP and LVEDP following PVE in ten patients. Although increases in both CVP and LVEDP occurred in each patient, large increases in LVEDP were accompanied by relatively small increases in CVP in six patients.
Worse ventricular function and a poorer prognosis have been reported recently for patients with ECG evidence of anterior infarction when compared to inferior or other infarction patterns.\textsuperscript{11, 12}

Our group A patients did have a significantly higher control MAP than our group B patients, and this difference was even greater following PVE. Hypotension per se was infrequent among our patients, however, and the wide range of "normal" blood pressures expected in this age group limits the value of a single blood pressure measurement as a means of assessing ventricular function or prognosis. None of the other control measurements was significantly different between the two groups, although it is possible that the differences in PADP or LVEDP would have become significant if more patients had been studied.

From our data it appears that, in the absence of clinical signs of pump failure, static baseline hemodynamic measurements are of little value as a means of estimating ventricular performance following acute myocardial infarction. Parameters influenced by ventricular contractility such as LV dp/dt and $V_{max}$ are often normal after AMI\textsuperscript{13, 14} presumably due to increased contractility in noninvolved muscle counteracting decreased contractility in ischemic or infarcted muscle.

Prakash et al.\textsuperscript{8} found a 94% mortality when LVSWI was less than 25 g-m/m² following AMI. Since patients with such a poor prognosis may be considered for aggressive therapeutic intervention, it is important to remember that reduced CI or LVSWI may be the result of inadequate ventricular filling, as was demonstrated by patient O.G. who increased LVSWI from 22 to 43 g-m/m² following PVE.

Although knowing LVSWI and LVEDP allowed us to locate a point on the ventricular function curve, we were unable from these data to predict the slope of the curve, and thus could not use these static measurements to assess ventricular function. Since none of these patients had a control LVEDP over 22 mm Hg, we do not know how patients with

---

**Table 3**

*Control Values (Mean ± SEM)*

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Group A</th>
<th>Group B</th>
<th>$P$ value*</th>
</tr>
</thead>
<tbody>
<tr>
<td>CI (liters/min/m²)</td>
<td>2.3 ± 0.2</td>
<td>2.6 ± 0.3</td>
<td>NS</td>
</tr>
<tr>
<td>SV (cc)</td>
<td>52 ± 5</td>
<td>54 ± 5</td>
<td>NS</td>
</tr>
<tr>
<td>MAP (mm Hg)</td>
<td>105 ± 3</td>
<td>93 ± 3</td>
<td>&lt;0.05</td>
</tr>
<tr>
<td>CVP (mm Hg)</td>
<td>2.9 ± 0.8</td>
<td>3.8 ± 0.8</td>
<td>NS</td>
</tr>
<tr>
<td>HR (beats/min)</td>
<td>82 ± 5</td>
<td>88 ± 4</td>
<td>NS</td>
</tr>
<tr>
<td>SVR (mm Hg/liter/min)</td>
<td>27 ± 4</td>
<td>21 ± 2</td>
<td>NS</td>
</tr>
<tr>
<td>PADP (mm Hg)</td>
<td>9.8 ± 2.1</td>
<td>13.9 ± 2.0</td>
<td>NS</td>
</tr>
<tr>
<td>LVEDP (mm Hg)</td>
<td>8.9 ± 2.1</td>
<td>15.0 ± 2.1</td>
<td>NS</td>
</tr>
<tr>
<td>LVSWI (g-m/beat/m²)</td>
<td>35 ± 4</td>
<td>33 ± 5</td>
<td>NS</td>
</tr>
<tr>
<td>LV dp/dt (mm Hg/sec)</td>
<td>1679 ± 146</td>
<td>1654 ± 200</td>
<td>NS</td>
</tr>
<tr>
<td>$V_{max}$ (ml/sec)</td>
<td>1.26 ± 0.12</td>
<td>1.22 ± 0.08</td>
<td>NS</td>
</tr>
<tr>
<td>QS (msec)</td>
<td>526 ± 9</td>
<td>518 ± 9</td>
<td>NS</td>
</tr>
<tr>
<td>LVET (msec)</td>
<td>379 ± 11</td>
<td>306 ± 6</td>
<td>NS</td>
</tr>
<tr>
<td>PEP (msec)</td>
<td>143 ± 6</td>
<td>136 ± 4</td>
<td>NS</td>
</tr>
<tr>
<td>PEP/LVET (msec)</td>
<td>0.46 ± 0.04</td>
<td>0.46 ± 0.02</td>
<td>NS</td>
</tr>
<tr>
<td>MRE (cc/sec)</td>
<td>223 ± 20</td>
<td>264 ± 28</td>
<td>NS</td>
</tr>
</tbody>
</table>

*Obtained by Students t test for unpaired data. NS = $P > 0.05$. Abbreviations: see text.

---

**Figure 5**

*Changes in PADP and LVEDP after PVE in nine patients. In most patients increases in PADP and LVEDP were similar in magnitude.*

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AMl and LVEDP above this level might have responded to PVE.

Russell et al.10 studied the response to PVE in 19 patients with AMI and found that when PADP exceeded 20–24 mm Hg, the ventricular function curve became flat. However, LVEDP was measured in only a few of these patients, and, since PADP may be significantly less than LVEDP due to a marked increase in LVEDP coincident with atrial contraction,10 the actual LVEDP at which patients with AMI will show a flat ventricular function curve has yet to be determined. The highest control LVEDP among our group A patients was 18 mm Hg, and thus our data do not answer the above question; however, LVEDP was increased to 30 mm Hg or above in three group A patients and one group B patient without ill effects. It should also be stressed that the presence of a normal, or only moderately elevated, control LVEDP was not always associated with improved CI following PVE. This is illustrated by the control LVEDP under 15 mm Hg in five of the seven group B patients in whom it was measured. The LVEDP for any given patient is determined by multiple factors including acute or preexisting alterations in ventricular compliance, geometry, contractility, the central blood volume, and probably the force and timing of left atrial contraction. Since some or all of these factors may be affected by AMI, it is not surprising that the control LVEDP did not correlate well with the subsequent response to PVE. Since we did not measure blood volume, the presence of hypovolemia in some of our group A patients cannot be excluded. However, the incidence of vomiting was similar in both groups, and in no patient was there an obvious cause for significant hypovolemia.

An additional aspect of our study which deserves comment relates to the interpretation of data obtained from frequent measurements of CVP, PADP, and systolic time intervals in patients with AMI. Hamosh and Cohn17 have found a poor relationship between CVP and LVEDP in patients with uncomplicated AMI, and we have made a similar observation in patients convalescing from AMI.1 In the present study, changes in both CVP and LVEDP following PVE were recorded in 10 patients, and, although directional changes were the same in every instance, the increase in LVEDP was more than triple the increase in CVP in half of the patients. Although PADP monitoring is more accurate than CVP monitoring as a means of estimating LVEDP during fluid administration, CVP should be measured in patients with shock following AMI if for some reason PADP or LVEDP cannot be measured. In such patients a normal or low CVP is very suggestive of hypovolemia, and fluid administration during careful CVP monitoring is indicated.8 Measurement of pulmonary artery pressure has been facilitated with the development of the Swan-Ganz catheter,6 and mean left atrial and ventricular diastolic pressures agree closely with PADP and wedge pressures obtained through this catheter. However, when assessing left ventricular function it is important to note that, in patients with AMI, LVEDP is often considerably higher than PADP or wedge pressure.18

The measurement of systolic time intervals as a noninvasive means of evaluating cardiac function has received much attention, but when applied to patients with AMI a poor correlation between STI and direct hemodynamic measurements has been observed.18,19 Our data further suggest that, even when serial changes in STI are considered, interpretation of such changes is far from simple. Although an increase in LVET following PVE occurred in five of six group A patients, all of whom increased SV, a similar increase in LVET occurred in 10 of 11 group B patients of whom eight had no change or a fall in SV. The increase in LVET, in our group A patients could be explained by the large increase in SV which exceeded the increase in the rate of left ventricular ejection. In group B patients, however, the increase in LVET appears to have been due entirely to a decrease in the rate of left ventricular ejection. Although interpretation of changes in LVET might be aided by considering associated changes in PEP (i.e. a decreased MRE associated with an increased PEP as evidence of diminished contractility) we did not find this to be the case, because PEP was decreased slightly following PVE in both groups of patients. Thus, since an increase in LVET, and a decrease in the PEP/LVET ratio can result from either improved SV or a decreased rate of left ventricular ejection the value of these measurements as a means of following individual patients with AMI remains in doubt.

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