Left Ventricular Preejection Period 
and Ejection Time in Patients 
with Acute Myocardial Infarction

By Morrison Hodges, M.D., Barry L. Halpern, M.D.,
Gottlieb C. Friesinger, M.D., and Gilles R. Dagenais, M.D.

SUMMARY
The phases of systole were measured in 51 patients with acute myocardial infarction and three control groups: (1) a group of 40 patients without heart disease, (2) a group of 23 patients admitted to a coronary care unit for chest pain, who did not have an acute myocardial infarction, and (3) a group of 16 patients with stable angina pectoris and arteriographically proven coronary atherosclerosis. In addition, serial measurements were made in the acute myocardial infarction group. Total electrical-mechanical systole (QS2), the preejection period (PEP), and left ventricular ejection time (LVET) were measured in each patient from simultaneous recordings of the ECG, phonocardiogram, and carotid pulse tracing. The systolic and diastolic blood pressures and QRS duration were also measured. Corrections were made for heart rate where appropriate.

The average PEP was elevated on the first day of myocardial infarction but was within normal limits thereafter. The LVET and QS2 were significantly shortened until the fourth week of hospitalization. There was considerable overlap in the PEP values among the four groups. Eight patients died of acute myocardial infarction; the PEP was abnormally short in three (two of whom had cardiogenic shock), normal in two, and abnormally long in three. The PEP/LVET ratio separated the acute MI group from the normal group but not from the other two patient groups. Clinical class, digitalis, and infarct location did not produce characteristic changes in the systolic time intervals.

A reduction in stroke volume is the most likely explanation for the reduction in LVET and QS2. The wide range in PEP values observed is best explained by alterations in the multiple determinants of PEP.

The systolic time intervals do not appear to be useful as a diagnostic or prognostic tool in acute myocardial infarction.

Additional Indexing Words:
Systolic time intervals Total electrical-mechanical systole PEP/LVET ratio
Ischemic heart disease

The measurement of systolic time intervals is a simple and noninvasive method for evaluating cardiac function. Changes have been noted in congestive heart failure and after several types of pharmacologic interventions in normal groups and in patients with heart disease. This noninvasive technic can be repeatedly utilized at

From the Myocardial Infarction Research Unit and Cardiovascular Division, Department of Medicine, Johns Hopkins University School of Medicine, Baltimore, Maryland.

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Address for reprints: Morrison Hodges, M.D., Cardiology Unit, Strong Memorial Hospital, Rochester, New York 14642.

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the bedside in seriously ill patients. Diamant and Killip found that patients with acute myocardial infarction (MI) had significant prolongation of the pre-ejection period (PEP) and significant shortening of the left ventricular ejection time (LVET). Habte-Michael et al. also found a shortening of the LVET but observed a normal PEP in 20 patients with acute MI. We have made serial measurements of the intervals of systole in 51 patients with acute MI, as well as in suitable control groups. Our purpose was to determine whether the measurement of PEP and LVET might be useful for diagnosis and prognosis in patients with acute MI. A preliminary report has been presented.

Methods

Patient Groups

Normal Group

Forty hospital patients were selected as a control group. The majority of patients were studied on the ophthalmologic and orthopedic wards. Each patient had a negative cardiac history, a normal cardiac examination, and a normal ECG. There were 23 males and 17 females with an average age of 48 years.

Acute MI Group

Fifty-one patients with definite acute MI were studied serially in a coronary care unit. Diagnosis of acute MI was based on a positive history accompanied by evolutionary ECG changes and a rise and fall of serum glutamic oxalacetic transaminase (SGOT). Twenty-three patients had an anterior MI as indicated by Q-wave formation in the precordial leads; 20 patients had a diaphragmatic MI as indicated by Q-wave formation in leads II, III, and aV_F; and eight patients had a subendocardial MI as indicated by ST-T wave changes without Q-wave formation. Eight of the 51 patients (16%) died during hospitalization. Patients were placed in one of four classes with respect to left ventricular (LV) function, as follows: class I—no signs of LV dysfunction (25 patients); class II—basilar pulmonary rales and a third heart sound gallop (16 patients); class III—pulmonary edema (six patients); class IV—shock (four patients).

Nonmyocardial Infarction Chest Pain Group

Twenty-three patients were studied who entered the coronary care unit with a history of chest pain suggestive of ischemic heart pain. Serial ECG and SGOT determinations failed to document an acute MI. Seventeen patients of this group had a history and ECG changes suggestive of nonacute ischemic heart disease. Six patients were felt to have no significant heart disease.

Stable Chronic Ischemic Heart Disease Group

Sixteen patients were studied who had been admitted to the hospital for coronary arteriography. Each patient had a history of typical angina pectoris, as previously defined, which had been present for at least six months. In each patient angina pectoris was stable without suggestion of an acute MI in the previous 6 months. Coronary arteriography was performed in all patients and revealed significant coronary atherosclerosis (greater than 50% narrowing in at least one vessel) in all.

Data Collection

A four-channel pressurized-ink, direct-writing recorder* was used to record simultaneously the ECG, phonocardiogram, and the carotid arterial pulse tracing at a paper speed of 100 mm/sec. Lead II was the electrocardiographic lead selected in all patients. A microphone† was applied just inside the apex, and the first high-frequency component of the second heart sound was used for the S_2 measurement. The carotid arterial pulse was recorded by means of a funnel-shaped pick-up, attached by polyethylene tubing to a Statham P23Db strain gauge. The funnel was placed directly over the right carotid artery with the gauge vented to air. The vent was then closed to record the carotid pulse. QRS was recorded as the interval from the QRS complex onset to the first high-frequency vibration of the second heart sound. The LVET was measured from the beginning upstroke to the trough of the incisura of the carotid arterial pulse tracing. The pre-ejection period was obtained by subtracting LVET from QRS. The QRS duration was measured directly. Heart rate was taken as the average (60/B-R) value for 10 consecutive beats.

For each recording an average figure was obtained from 10 to 15 consecutive beats. All patients were in sinus rhythm. All systolic time interval measurements were expressed in msec. The PEP/LVET ratio was obtained by dividing the PEP by LVET.

Recordings were usually made in the early morning, however, some patients had tracings made at other times during the day depending on when they were admitted to the coronary care unit. In this study all days ended at midnight.

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*Brush Mark 240, Clevite Corporation, Cleveland, Ohio.
†Electronics for Medicine, Inc., White Plains, New York.

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Hence, if a patient was admitted at 10 PM and a recording made at 8 AM on the following morning, this first record would be termed day 2. All recordings were made in the supine position. Blood pressure was recorded by cuff sphygmomanometer. In the normal group all the recordings were made after a 15-minute rest. No attempt was made to correlate the time of measurements with meals.

The Brush instrument has a frequency response of 40 Hz at full scale and 100 Hz at 20% full scale. Heart sound recordings were ordinarily done at approximately 20 to 30% full scale. Preliminary experiments were performed to test the suitability of this instrument for measurement of systolic time intervals. The phonocardiogram, carotid pulse, and electrocardiogram from subjects were recorded simultaneously on these three different instruments. The phonocardiogram, carotid pulse, and electrocardiogram from subjects were recorded simultaneously on four-channel direct-writer recorder used in these studies (Brush), an oscilloscopic recorder (model DR 8), and a galvanometer recorder.* Systolic time intervals on two patients with atrial fibrillation and three normal subjects were measured simultaneously on these three different recorders and analyzed independently by two observers. The four-channel direct-writer recorder (Brush) proved satisfactory for measurement of systolic time intervals. Agreement among these three instruments was within 1.5%. Agreement within 2.0% was noted between the two observers.

Data Analysis

All data were punched on cards, and analysis was performed on a digital computer (XDS Sigma 3). Standard statistical techniques were used for linear regression analysis, analysis of covariance, and unpaired t tests.10

Results

The data obtained from the four patient groups are shown in table 1 and figures 1 to 4.

Preejection Period

As shown in figure 1, there was no significant correlation between the PEP and heart rate in the normal group. Thus, no correction for heart rate was applied to the PEP values for the other three groups. As indicated in the table, the average of first PEP measurements obtained in the coronary care unit on MI patients (101.6 ± 3.1 msec) was not statistically significantly different from the mean PEP measurements in the control group (96.0 ± 2.3 msec). However, if the mean values for all PEP measurements on MI patients obtained on day 1 (as defined above) are utilized, the MI patient group has a mean value (108.8 ± 4.7 msec) which is statistically higher than the control group. The mean PEP value for the non-MI chest pain group was 111.5 ± 4.0 msec and for the stable ischemic

Table 1

<table>
<thead>
<tr>
<th>Patient Data</th>
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</thead>
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<table>
<thead>
<tr>
<th>Normal group</th>
<th>Acute MI group*</th>
<th>Non-MI chest pain group*</th>
<th>Stable IHD group</th>
</tr>
</thead>
<tbody>
<tr>
<td>Number</td>
<td>40</td>
<td>51</td>
<td>23</td>
</tr>
<tr>
<td>Age (years)</td>
<td>48.1 ± 2.2</td>
<td>59.9 ± 1.4†</td>
<td>57.0 ± 2.4†</td>
</tr>
<tr>
<td>Systolic blood pressure (mm Hg)</td>
<td>129.6 ± 2.7</td>
<td>117.4 ± 2.4†</td>
<td>131.3 ± 5.9</td>
</tr>
<tr>
<td>Diastolic blood pressure (mm Hg)</td>
<td>78.9 ± 1.3</td>
<td>75.7 ± 1.8</td>
<td>79.8 ± 3.5</td>
</tr>
<tr>
<td>QRS duration (msec)</td>
<td>75.0 ± 1.8</td>
<td>80.6 ± 1.5†</td>
<td>80.6 ± 2.6</td>
</tr>
<tr>
<td>Heart rate (msec)</td>
<td>73.9 ± 1.8</td>
<td>84.2 ± 2.3†</td>
<td>67.7 ± 2.9</td>
</tr>
<tr>
<td>PEP (msec)</td>
<td>96.0 ± 2.3</td>
<td>101.6 ± 3.1</td>
<td>111.5 ± 4.0†</td>
</tr>
<tr>
<td>LVETI (msec)</td>
<td>443.7 ± 2.4</td>
<td>424.7 ± 3.1†</td>
<td>408.2 ± 4.8†</td>
</tr>
<tr>
<td>Q81 (msec)</td>
<td>550.2 ± 3.3</td>
<td>538.3 ± 3.2†</td>
<td>529.3 ± 5.6</td>
</tr>
<tr>
<td>PEP/LVET</td>
<td>0.346 ± 0.010</td>
<td>0.442 ± 0.019†</td>
<td>0.436 ± 0.018†</td>
</tr>
</tbody>
</table>

Key to table: Values given as means ± ssm. * = first value obtained in coronary care unit; † = significantly different from normal group at the 5% level; MI = myocardial infarction; IHD = ischemic heart disease.

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heart disease group 111.8 ± 3.6 msec. Hence, both these groups also had significantly higher PEP values than the normal group. It is apparent from figure 1 that there is considerable overlap for PEP values in all groups, with the widest scatter in the acute MI group.

Despite these statistically significant differences between the acute MI group and the other three groups, consideration of individual values was of limited usefulness in diagnosis. Although 14 (28%) of the acute MI patients had a PEP value longer than 115 msec, 11 (48%) of the non-MI chest pain patients and nine (56%) of the stable ischemic heart disease patients also had PEP values longer than 115 msec. Short PEP values were common in the acute MI group: 13 (26%) had a PEP value less than 85 msec. In contrast, none of the stable ischemic heart disease group and only one (4%) of the non-MI chest pain group had values in this range.

Serial PEP measurements in MI patients indicated that the average value on each day was higher than the normal group, but the difference was statistically significant only on day 1. There was no significant trend in the daily average of the PEP (see fig. 3).

There was no characteristic pattern of the PEP in the eight patients who died. On admission three in this group had a PEP less than 85 msec (two died of shock and one of primary ventricular fibrillation), two had a normal PEP, and three had a PEP longer than 115 msec. There was no day-to-day trend to distinguish patients who died from those surviving.

Left Ventricular Ejection Time (Fig. 2)

There was a highly significant correlation between LVET and heart rate (HR) in the normal group \( r = -0.85; \ P < 0.001; \ LVET = -2.22 \cdot HR + 443.7 \). The LVET for each patient was thus corrected for heart rate and expressed as the left ventricular ejection time index (LVETI),\textsuperscript{11} calculated as follows:

\[
LVETI = 2.22 \cdot HR + LVET
\]

On admission to the coronary care unit, the LVETI was significantly shorter than normal in the acute MI and non-MI chest pain groups (table 1). In contrast, LVETI was normal in
the stable ischemic heart disease group. Serial measurements revealed that the LVETI was significantly shorter than normal on each day of MI during the first 3 weeks of hospitalization (fig. 3). The LVETI was normal in the six patients in whom measurements were made beyond 3 weeks of hospitalization. The LVETI reached its lowest value on day 6 and subsequently increased toward normal.

There was a significant relationship between heart rate and LVET in all three patient groups (fig. 2). Analysis of covariance revealed that there was no significant difference between the slopes of the four groups. However, the intercept in the acute MI and non-MI chest pain groups was significantly lower than the intercept in the normal and stable ischemic heart disease groups. The fact that the slopes of the regressions of LVET on heart rate are statistically the same in all four groups means that use of LVETI for comparison purposes is valid.

**Q-Second Sound Interval**

There was a significant correlation between the QS₂ and heart rate in all groups. The regression equation for the normal group (\( QS₂ = -2.33 \text{ HR} + 548.0; \ r = -0.78; \ P < 0.001 \)) was used to calculate a QS₂ index (\( QS₂\text{I} \)) for each patient, as follows:

\[
QS₂\text{I} = 2.33 \text{ HR} + QS₂
\]

The admission QS₂\text{I} for the two coronary care unit groups was significantly lower than the normal group (table 1). The stable ischemic heart disease group had a QS₂\text{I} which was significantly longer than the normal group and was due to a long PEP, since the LVETI was normal in this group.

Serial determinations of QS₂\text{I} indicated that the average value for the acute MI group paralleled the LVETI, being significantly low until the fourth hospital week (fig. 3). The lowest value was reached on day 6 with a gradual return to normal during the fourth hospital week.

**PEP/LVET Ratio**

This ratio has been suggested by Weissler et al.\(^1\) as a convenient measure of ventricular function since it is presumably independent of
heart rate. Values for all patient groups are shown in figure 4 and indicate a considerable overlap among all groups. However, the three patient groups have a statistically higher PEP/LVET ratio than the normal group. The three patient groups are not statistically different from one another, however.

Serial measurement of the PEP/LVET ratio revealed that it was significantly higher than that in normal subjects, including the fourth week of hospitalization. This was true despite the fact that PEP and LVETI were within normal limits when measured during the fourth week. Fourteen of the acute MI patients (28%) had a PEP/LVET ratio larger than the highest value for the normal group. Among all patient groups the eight highest PEP/LVET ratio values were obtained in the acute MI group (fig. 4).

**Blood Pressure**

Systolic blood pressure was significantly depressed on admission in the acute MI group (table 1). However, there were no other differences in systolic or diastolic blood pressure among the four patient groups. Serial measurements of systolic and diastolic pressures in the acute MI group indicated a significant drop in both over a 3-week period. Among surviving patients the blood pressure at discharge was 111.7 ± 5.1 mm Hg systolic and 66.8 ± 4.4 mm Hg diastolic.

**QRS Duration**

QRS duration was prolonged in all three patient groups (table 1). This prolongation was significant in the acute MI and stable ischemic heart disease groups and was of borderline significance in the non-MI chest pain group. No patient had a QRS duration greater than 100 msec. Serial measurement of

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**Figure 3**

Serial values for QS 
LVETI, PEP, and PEP/LVET ratio in patients with acute MI are shown. Mean values for each day are shown, with the 95% confidence limits around the mean. Open circles at left are the values from the normal group. * = significantly different from the normal group at 5% level.

**Figure 4**

The PEP/LVET ratio values are given for the four patient groups. The first value obtained in each acute MI patient is plotted. The solid bars are the mean values for each group. Open circles represent the patients who died.

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QRS duration indicated no significant lengthening during hospital stay in the acute MI group.

**Drug Effects**

The PEP and LVETI on admission were examined in the two coronary care unit groups with respect to a history of digitalis administration. No difference in either group was noted between those patients who had been taking digitalis and those who had not. Serial studies indicated that administration of intravenous lidocaine had no effect on the PEP or LVETI.

**Effect of Clinical Class**

In the acute MI patients there was no significant difference in PEP, LVETI, QS₂I, or PEP/LVET between any of the clinical classes. A stepwise multiple linear regression analysis was performed, using patient class as the dependent variable and PEP, LVETI, QS₂I, heart rate, age, and systolic and diastolic blood pressures as the independent variables. There was no significant correlation between clinical class and any of these variables. In addition, no combination of these variables could be found which could significantly correlate with patient class.

**Infarct Location**

In patients with acute myocardial infarction, there was no significant difference in PEP, LVETI, QS₂I, or PEP/LVET ratio among those patients with diaphragmatic, anterior, or subendocardial infarctions.

**Discussion**

The purpose of this study was to determine whether systolic time intervals were useful in diagnosing or predicting the course of acute myocardial infarction. The results demonstrate that systolic time intervals are usually abnormal in acute MI, but the abnormalities are not helpful diagnostically since a control group of patients admitted to the coronary care unit with non-MI chest pain had PEP prolongation similar to that seen in the patients with acute MI. In addition, another control group of 16 patients with stable chronic ischemic heart disease had a significantly prolonged PEP in nine instances. These data suggest that many patients with acute MI have "baseline" PEP lengthening prior to the onset of their acute episode. Pouget et al.¹² also found a long PEP (average 118 msec) at rest in 20 patients with ischemic heart disease. It is possible that the long PEP in our non-MI chest pain group is related to the presence of chronic ischemic heart disease since 17 of the 23 patients in this group were thought to have ischemic heart disease. The remaining six patients, considered to have no heart disease despite their chest pains, all had normal or short PEP values.

The systolic time intervals did not prove useful in predicting outcome. We observed the onset of clinical congestive heart failure in several patients who did not have the expected changes in systolic time intervals.² Furthermore, in the eight patients who died, three had short PEP values (two died in shock, one of primary ventricular fibrillation), two patients had a normal PEP, and three patients had prolonged values.

The PEP/LVET ratio was increased in patients with myocardial infarction, but this was also true in the patients with stable chronic ischemic heart disease and non-MI chest pain. In this particular study a PEP/LVET ratio of 0.600 or greater was associated exclusively with acute MI. However, in any patient in whom this ratio was extremely high, there were other criteria present which were also diagnostic for acute MI. It is of interest that the PEP/LVET ratio in the eight patients who died ranged from low to high values (fig. 4), and serial measurements of this ratio gave no indication of subsequent complications or death.

Since the PEP in our acute MI patients ranged from abnormally short to very long, it is appropriate to consider the determinants of the PEP and how they might influence the measurements in acute MI. Determinants of the PEP include QRS duration, rate of rise of pressure in the left ventricle, left ventricular end-diastolic pressure, and aortic diastolic pressure. QRS duration is usually 50 to 90% of the duration of the PEP. QRS duration did not
change significantly in individual patients and thus did not cause changes in the PEP of individual patients. Differences in QRS duration between patients will affect comparisons of the PEP between patients, however. Identification of the interval from the first to the second heart sound and subtraction of the LVET from this interval allow one to determine the isovolumic contraction time (ICT)\(^2\) and eliminate the QRS duration as a determinant. We were unable to identify the first heart sound confidently in all of our patients with acute MI, and we thus did not attempt to calculate the ICT in this manner. The QRS duration was longer in our patient groups than in our normal group, and this factor accounts in part for the PEP lengthening in the patient groups. Indeed, subtraction of the average QRS duration from the average PEP yields an identical answer (21 msec) for our normal and acute MI groups, while longer values were obtained for the non-MI chest pain (31 msec) and stable ischemic heart disease (28 msec) groups.

Animal investigations have shown that maximum LV dp/dt is reduced after coronary artery occlusion.\(^13\) Recently, direct measurements of the rate of rise of left ventricular pressure have been made in patients with acute MI\(^14\) and indicate that maximum LV dp/dt is depressed in many patients with acute MI, particularly those with shock. Depression of LV dp/dt would help to account for the PEP lengthening in acute MI patients. Normal values for maximum LV dp/dt have been difficult to establish because of varying recording technics, but values of 1,000 mm Hg/sec or less have been observed in ischemic heart disease patients at rest.\(^15, 16\) It is possible that such a depression was present in our non-MI chest pain and stable ischemic heart disease groups, helping to account for the long PEP in these patients.

Left ventricular end-diastolic pressure (LVEDP) is elevated in the majority of patients with acute MI.\(^14\) Since there is an inverse relationship between PEP and LVEDP\(^17\) and LV end-diastolic volume,\(^18\) even in patients with heart disease,\(^18\) a rise in LVEDP in our patients would tend to shorten the PEP.

There is a direct relationship between aortic diastolic pressure and the PEP.\(^17, 18\) When all the values in the acute MI group are pooled, there is a weak but significant positive correlation \((r = +0.28; P < 0.001; N = 201)\) between the aortic diastolic pressure and the PEP. Thus, in acute MI, hypertension will lengthen and hypotension shorten the PEP if other determinants remain constant.

Heart rate was not significantly correlated with the PEP in our normal and coronary care unit groups. Other workers have found significant correlations,\(^2, 6\) but \(r\) values have not been published. It can be inferred that the relationship is not a strong one since the regression coefficients have been relatively small. In an animal study\(^17\) no relationship was found between heart rate and PEP when other determinants of PEP were held constant.

Combinations of alterations in the determinants mentioned above probably account for the wide range of PEP values we found in acute MI patients. For example, a high LVEDP and a low aortic diastolic pressure (a combination present in cardiogenic shock\(^19\)) may result in a short PEP despite a low LV dp/dt. Two of our patients in shock had a PEP less than 90 msec. On the other hand, a patient with a normal or mildly elevated LVEDP and a normal or high aortic diastolic pressure could have a greatly prolonged PEP, particularly if LV dp/dt is low. Presumably, such interrelationships of determinants led to the apparently paradoxic findings of a short PEP in a patient with cardiogenic shock and a markedly prolonged PEP in a patient with subendocardial infarction.

The majority of our acute MI patients had a significantly shortened QS\(_3\)I during the first 3 weeks of hospitalization. This is in keeping with other reports.\(^7, 20-22\) Some investigators have found this shortening to be correlated with an increased level of urinary catecholamine excretion,\(^23\) but others have not found such a relationship.\(^21\) In our patients the shortened QS\(_3\)I was shortened primarily by
the shortened LVETI since PEP was within normal limits or elevated during hospitalization. A brief review of the determinants of LVET is in order since it is such an important feature of the abnormalities found in our studies and since a reduction of LVET in acute MI patients has been previously noted.6, 7, 20–22

Determinants of LVET include heart rate, stroke volume, arterial pressure (or afterload), and left ventricular contractility. We have presumably accounted for the effects of heart rate by converting LVET to LVETI. There was a small but significant decrease in systolic and diastolic blood pressures in the acute MI patients during hospitalization. There is a direct relationship between LVET and blood pressure,5 and it is likely the reduction in blood pressure in our patients contributed to the shortening of LVETI, even though the overall correlation between LVETI and systolic and diastolic blood pressure (r = + 0.07 and + 0.16, respectively) was not significant.

An inotropic intervention shortens the LVET if other determinants remain constant.3 Since almost all acute MI patients have evidence of reduced contractility,14, 24 it is unreasonable to assume that increased contractility played a role in the LVETI shortening in any of our patients.

Stroke volume is a major determinant of LVET. Harley et al.25 measured stroke volume on a beat-to-beat basis in patients with heart block or atrioventricular dissociation using the pressure gradient technic. Over a wide range of stroke volumes, they found linear relationships between LVET and stroke volume and between LVET and heart rate. However, stroke volume appeared to play a much larger role than heart rate in determining the length of LVET. Other studies have also found a close relationship between LVET and stroke volume, particularly after correction for the effects of heart rate.26, 27 A reduced stroke volume is a common accompaniment of acute MI.28–30 Although we do not have hemodynamic measurements for our patients, it is likely that a reduced stroke volume is the primary reason for a shortened LVETI in our patients.

Our serial measurements of LVETI would suggest that stroke volume is maximally reduced on the sixth hospital day, with a gradual return to normal in the fourth week after infarction. Hemodynamic studies are consistent with this suggestion. Hodges et al.24 found a significant reduction in stroke volume during the first 4 days after infarction in 20 patients with uncomplicated acute MI. Murphy et al.29 found a low stroke volume in the majority of acute MI patients studied, with a return toward normal after the second week of hospitalization in those patients who survived. Nager et al.31 also found a reduced stroke volume in most patients with acute MI and felt that the level of stroke volume was a useful prognostic sign.

Factors aside from disease state and stage of disease could influence the measurements. Bed rest, time of day, recently ingested food, and drug therapy are among such possible factors. These matters were considered, and it seems unlikely that any of these or similar factors would be of major importance except for the possibility of bed rest. The “deconditioning” which could result from bed rest undoubtedly could play a role in the shortening of the QS2 and LVET and lengthening of the PEP. Regardless of the potential role such factors might play, the wide range of values obtained in the acute MI group and the overlap with control groups make it impossible to use PEP as a guide to prognosis or management in any specific patient being evaluated in the usual clinical situation. It is possible that LVETI, because of its close relationship to stroke volume, could be more useful, particularly when serial measurements are made.

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MORRISON HODGES, BARRY L. HALPERN, GOTTLIEB C. FRIESINGER and GILLES R. DAGENAIS

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