Reciprocal ST Change in Acute Myocardial Infarction: Assessment by Electrocardiography and Echocardiography

EDMUNDO J. N. CAMARA, MD, NISHA CHANDRA, MD, PAMELA OUYANG, MD, SHELDON H. GOTTLIEB, MD, EDWARD P. SHAPIRO, MD, FACC

Baltimore, Maryland

To evaluate the incidence, time course and significance of reciprocal change, 25 consecutive patients admitted with their first acute transmural myocardial infarction were studied with serial electrocardiography and two-dimensional echocardiography. Reciprocal change was noted in all patients with inferior infarction (mean maximal ST segment depression 3.53 ± 1.97 mm) and 70% of patients with anterior infarction (mean maximal ST depression 1.45 ± 0.8 mm, p = 0.001). When initially present, reciprocal change had resolved within 24 hours in 59% of patients. The sum of reciprocal ST depression correlated with the sum of ST elevation in anterior (r = 0.92, p < 0.001) and inferior (r = 0.55, p = 0.035) infarction, and this relation persisted when maximal ST depression and elevation were considered. Echocardiographic evidence of contraction abnormalities in areas of the left ventricle remote from the infarction was seen in 45% of patients. However, its presence did not correlate with the presence of reciprocal change. Although reciprocal change progressively diminished on serial electrocardiograms (maximal ST depression 2.73 ± 1.77 mm at 19 hours after onset of symptoms; 1.0 ± 0.92 mm at 2 to 3 days; and 0.22 ± 0.26 mm at 7 to 10 days; p < 0.05), the corresponding serial echocardiograms showed no change in the function of the remote wall (remote wall motion index 1.87 ± 0.65, 1.81 ± 0.62, 1.86 ± 0.47, respectively, p = NS)

These data, therefore, do not support the hypothesis that reciprocal ST depressions during early acute transmural myocardial infarction reflect remote ischemia. Rather, these changes are influenced by factors determining the degree of acute ST elevation, previously shown to include infarct size, shape, location, transmurality and duration.

Electrocardiographic ST segment depression is frequently observed in patients with acute anterior or inferior myocardial infarction and is called reciprocal change when it occurs simultaneously with ST elevation. There is recent evidence that this finding at presentation may define a population at risk for later complications (1,2). However, the mechanism and actual significance of reciprocal change have been debated. Some authors (2,3) have presented evidence that these ST depressions may represent true remote ischemia, while others (4–6) have shown reciprocal change to be associated with extensive myocardial damage. Because the presence of ischemic yet viable myocardium during infarction constitutes a powerful risk factor treatable by aggressive medical or surgical management, the clarification of this issue has clinical significance.

Experimental and clinical studies (7,8) have shown that left ventricular wall motion is impaired during ischemia. Two-dimensional echocardiography provides a means of assessing wall motion remote from the region of acute infarction. In the present investigation, we assessed the incidence and time course of ST segment depression in acute transmural myocardial infarction and its correlation with remote wall motion abnormalities as evaluated by serial two-dimensional echocardiograms.

Methods

Study patients. Twenty-five consecutive patients admitted to Baltimore City Hospitals with acute transmural myocardial infarction were studied prospectively. There were 21 men and 4 women, aged 38 to 79 years, admitted 3.97 ± 4.8 hours (mean ± standard deviation) after the onset of chest pain. The diagnosis of acute transmural myocardial infarction was made if all of the following criteria were present: 1) chest pain or symptoms compatible with myo-
cardiac ischemia lasting longer than 30 minutes; 2) ST segment elevation of 0.1 mV and new abnormal Q waves (0.04 second’s duration) developing in at least two leads within 48 hours; and 3) abnormal elevation of serum creatine kinase with positive MB fraction. The electrocardiographic location of the myocardial infarction was considered either anterior (leads I, aVL, V₁ through V₃) or inferior (leads II, III, aVF).

Patients with subendocardial myocardial infarction and one patient with a true posterior myocardial infarction who had no changes in leads II, III and aVF were excluded. Patients with previous myocardial infarction by history or electrocardiogram were excluded, as were those with chronic electrocardiographic abnormalities associated with ST segment depressions, including left ventricular hypertrophy with strain, left bundle branch block and marked digitalis effect.

**Electrocardiograms.** Standard 12 lead electrocardiograms were obtained in the emergency room, on admission to the coronary care unit and at least once daily thereafter. The electrocardiographic criterion for the diagnosis of reciprocal ST changes was a depression of the ST segment of at least 0.1 mV in at least two leads in the wall opposite the infarction (leads I, aVL, V₁ through V₃ or inferior leads II, III, aVF). The ST segment elevation or depression was measured 0.06 second after the nadir of the S wave, taking the TP segment as the baseline. The maximal ST depression for any individual patient was defined as the greatest ST depression reached in any lead on any electrocardiogram. The same electrocardiogram was then used to measure the maximal ST elevation and to calculate the sum of ST elevation and of ST depression. The electrocardiograms were interpreted without previous knowledge of the echocardiograms.

**Echocardiograms.** Serial two-dimensional echocardiograms were performed using a phased array ultrasonograph (Diasonic V3400R) and recorded on videotape for later analysis. The first study was done within 1 day of admission (19.0 ± 13 hours after chest pain), the second on day 2 or 3 (2.7 ± 1.0 days after chest pain) and the third between days 7 and 10 (7.8 ± 1.6 days after chest pain). Adequate echocardiograms could not be obtained in three patients, two with and one without reciprocal change. Echocardiograms of sufficient quality for interpretation were obtained in 22 patients. In 13 patients (Group I), reciprocal change was still present on the electrocardiogram at the time of the first echocardiogram (20.6 ± 17.9 hours after the onset of chest pain). In nine patients (Group II), reciprocal change had resolved at the time of the first echocardiogram (18.2 ± 6.1 hours after the onset of chest pain [n = 6]) or had never been present [n = 3]).

The standard views were performed in the parasternal long- and short-axis positions and the apical four and two chamber positions. Studies were interpreted independently by two experienced echocardiographers without knowledge of electrocardiographic findings, and differences were later resolved by conference. For purposes of analysis, the left ventricle was divided into 18 segments as defined in Figure 1. Each segment was evaluated for asynergy on the basis of visual assessment of systolic thickening and endocardial movement of the wall, in a fashion similar to that of Gibson et al. (9). A numerical score of 2 was given to segments considered normal in thickening and inward endocardial wall movement. A score of 3 was used for segments that were hyperkinetic in comparison with normal. A score of 1 was assigned to hypokinetic segments that did not thicken, even if slight passive endocardial wall motion remained. Dysskinetic motion was graded −1.

**Echocardiograms.** Standard 12 lead electrocardiograms were obtained in the emergency room, on admission to the coronary care unit and at least once daily thereafter. The electrocardiographic criterion for the diagnosis of reciprocal ST changes was a depression of the ST segment of at least 0.1 mV in at least two leads in the wall opposite the infarction (leads I, aVL, V₁ through V₃ or inferior leads II, III, aVF). The ST segment elevation or depression was measured 0.06 second after the nadir of the S wave, taking the TP segment as the baseline. The maximal ST depression for any individual patient was defined as the greatest ST depression reached in any lead on any electrocardiogram. The same electrocardiogram was then used to measure the maximal ST elevation and to calculate the sum of ST elevation and of ST depression. The electrocardiograms were interpreted without previous knowledge of the echocardiograms.

**Echocardiograms.** Serial two-dimensional echocardiograms were performed using a phased array ultrasonograph (Diasonic V3400R) and recorded on videotape for later analysis. The first study was done within 1 day of admission (19.0 ± 13 hours after chest pain), the second on day 2 or 3 (2.7 ± 1.0 days after chest pain) and the third between days 7 and 10 (7.8 ± 1.6 days after chest pain). Adequate echocardiograms could not be obtained in three patients, two with and one without reciprocal change. Echocardiograms of sufficient quality for interpretation were obtained in 22 patients. In 13 patients (Group I), reciprocal change was still present on the electrocardiogram at the time of the first echocardiogram (20.6 ± 17.9 hours after the onset of chest pain). In nine patients (Group II), reciprocal change had resolved at the time of the first echocardiogram (18.2 ± 6.1 hours after the onset of chest pain [n = 6]) or had never been present [n = 3]).

The standard views were performed in the parasternal long- and short-axis positions and the apical four and two chamber positions. Studies were interpreted independently by two experienced echocardiographers without knowledge of electrocardiographic findings, and differences were later resolved by conference. For purposes of analysis, the left ventricle was divided into 18 segments as defined in Figure 1. Each segment was evaluated for asynergy on the basis of visual assessment of systolic thickening and endocardial movement of the wall, in a fashion similar to that of Gibson et al. (9). A numerical score of 2 was given to segments considered normal in thickening and inward endocardial wall movement. A score of 3 was used for segments that were hyperkinetic in comparison with normal. A score of 1 was assigned to hypokinetic segments that did not thicken, even if slight passive endocardial wall motion remained. Dysskinetic motion was graded −1.

**Echocardiograms.** Serial two-dimensional echocardiograms were performed using a phased array ultrasonograph (Diasonic V3400R) and recorded on videotape for later analysis. The first study was done within 1 day of admission (19.0 ± 13 hours after chest pain), the second on day 2 or 3 (2.7 ± 1.0 days after chest pain) and the third between days 7 and 10 (7.8 ± 1.6 days after chest pain). Adequate echocardiograms could not be obtained in three patients, two with and one without reciprocal change. Echocardiograms of sufficient quality for interpretation were obtained in 22 patients. In 13 patients (Group I), reciprocal change was still present on the electrocardiogram at the time of the first echocardiogram (20.6 ± 17.9 hours after the onset of chest pain). In nine patients (Group II), reciprocal change had resolved at the time of the first echocardiogram (18.2 ± 6.1 hours after the onset of chest pain [n = 6]) or had never been present [n = 3]).

The standard views were performed in the parasternal long- and short-axis positions and the apical four and two chamber positions. Studies were interpreted independently by two experienced echocardiographers without knowledge of electrocardiographic findings, and differences were later resolved by conference. For purposes of analysis, the left ventricle was divided into 18 segments as defined in Figure 1. Each segment was evaluated for asynergy on the basis of visual assessment of systolic thickening and endocardial movement of the wall, in a fashion similar to that of Gibson et al. (9). A numerical score of 2 was given to segments considered normal in thickening and inward endocardial wall movement. A score of 3 was used for segments that were hyperkinetic in comparison with normal. A score of 1 was assigned to hypokinetic segments that did not thicken, even if slight passive endocardial wall motion remained. Dysskinetic motion was graded −1.

The total wall motion index was then calculated by adding the scores and dividing the number of segments analyzed. The opposite (remote) wall of the infarcted area was defined in our study as follows (Fig. 1): segments 3, 4, 7, 9, 10, 17 and 18 (anteroseptal and anterolateral walls) for inferior myocardial infarction; segments 1, 2, 5, 13, 14 and 15 (inferior and posterobasal walls) for anterior myocardial infarction. Segments 6, 8, 11, 12 and 16 (posterior septum, apex and portions of lateral wall) were not included for evaluation of remote asynergy because these areas may be included in the infarct zone in both anterior and inferior myocardial infarction. We defined remote contraction abnormality as a wall motion index of the opposite wall of less than 2.0.

**Statistical analysis.** Data were recorded as mean ± 1 standard deviation. Differences between groups were compared using the unpaired two-tailed t test. The paired t test was used to evaluate mean differences within the same group. Correlations between ST segment elevation and depression were made by linear regression analysis.

**Results**

The 25 patients included 15 with inferior and 10 with anterior myocardial infarction.

**Analysis of ST segment elevation and correlation with ST depression.** The mean maximal ST elevation in any lead in the 25 patients was 3.26 ± 1.25 mm (range 1.5 to 7). The mean maximal ST depression in opposite leads was 2.7 ± 1.89 mm (range 0.5 to 8.0). The maximal ST elevation in inferior myocardial infarction (3.43 ± 1.43 mm) was similar to that in anterior myocardial infarction (3.0 ± 0.91 mm). However, reciprocal ST depression was substantially greater in inferior infarction (3.53 ± 1.97 mm) than in anterior infarction (1.45 ± 0.80 mm) (p = 0.001) (Fig. 2). Reciprocal ST depression was seen in 22 (88%) of the 25 patients in this study. All patients with inferior myocardial infarction and 7 (70%) of 10 patients with anterior infarction had reciprocal changes. Furthermore, there...
was a correlation \( r = 0.57; p = 0.003 \) between the maximal ST depression and maximal ST elevation. When the data were analyzed separately, this relation was maintained for both inferior \( r = 0.57; p = 0.027 \) and anterior myocardial infarction \( r = 0.69; p = 0.028 \) (Fig. 3). Three patients who did not have reciprocal changes had anterior myocardial infarction with a mean maximal ST segment elevation of only \( 1.83 \pm 0.3 \text{ mm} \), a value considerably lower than the \( 3.5 \pm 0.5 \text{ mm} \) ST elevation seen in the seven patients who had reciprocal change with anterior myocardial infarction.

*When the sum of ST segment elevation in all leads was compared with the sum of ST depression in all leads, correlation was present in both anterior \( r = 0.92; p < 0.001 \) and inferior \( r = 0.55; p = 0.035 \) infarction.*

*Reciprocal change was transient in most patients.* Only eight patients (32%) continued to demonstrate reciprocal change 24 hours after admission, and ST elevation was still present in all eight. Five of these eight patients had persistent ST elevation at the time of hospital discharge. In Group 1, maximal ST depression decreased from \( 2.73 \pm 1.8 \text{ mm} \) at the time of the first echocardiogram to \( 1.0 \pm 0.9 \text{ mm} \) at the time of the second echocardiogram on day 2 or 3, and to \( 0.22 \pm 0.26 \text{ mm} \) at the time of the third echocardiogram between days 7 and 10 \( p < 0.01 \) (Fig. 4, bottom).

*Wall motion and reciprocal change.* Two patients with reciprocal changes and one patient without were excluded from evaluation of wall motion because adequate echocardiograms could not be obtained. Of the remaining 22 patients, 45% had some degree of asynergy in a region of the left ventricle remote from the infarction. This was most marked in anterior myocardial infarction, where remote involvement was seen in 67% of patients as opposed to 31% of patients with inferior infarction; this difference, however, did not reach statistical significance.

*Figure 2. Maximal (MAX) ST elevation and ST depression in anterior (ANT) and inferior (INF) myocardial infarction (MI). N.S. = not significant; \( p = \) probability.*

![Figure 1. Standard echocardiographic views including parasternal long-axis, parasternal short-axis (at the level of the papillary muscles), apical four chamber and apical two chamber views. Wall segments are numbered for analysis.](image-url)
The initial remote wall motion index was not different in the 20 patients with, versus the 2 without reciprocal change (1.89 ± 0.51 versus 1.74 ± 0.58). In four patients whose ST depression was greater than expected for the degree of ST segment elevation (those patients in Figure 3 whose values are far above the line), the initial remote wall motion index was not different from that of the other patients with reciprocal change (1.89 ± 0.68 versus 1.87 ± 0.49). When all patients were considered, there was no significant change in the mean remote wall motion index on serial echocardiograms (1.87 ± 0.54, 1.89 ± 0.53, 1.85 ± 0.35, respectively, p = not significant).

The serial mean remote wall motion indexes are plotted in Figure 4 for Group I patients only, those in whom the first echocardiogram was recorded when reciprocal changes were still present. Importantly, although reciprocal change progressively diminished on serial electrocardiograms, the corresponding serial echocardiograms showed no change in the mean remote wall motion index (1.87 ± 0.65, 1.81 ± 0.62, 1.86 ± 0.47, respectively, p = not significant). This is shown for individual patients in Figure 5. In most patients, there was little change during the hospital course. In Group I, only 3 of 11 patients demonstrated a change of greater than 0.30 in the remote wall motion index; in 2 patients the index worsened and in 1 patient it improved. However, these changes were not necessarily related in timing to changes in reciprocal ST depressions. Of the nine patients in Group II, seven showed no change in remote wall motion index over time; in one patient the index improved and in one it deteriorated (Fig. 5).

There was no significant change in the wall motion index of the infarcted area during the hospital course, which was 0.28, 0.36 and 0.36 at the time of the first, second and third echocardiogram, respectively. Infarcted area wall motion index did not correlate with remote wall motion index (r = 0.017).

**Discussion**

The concept that the relative polarity of ST segment deviation depends on the position of the exploring electrode originated in the 1940s (10). Since that time, it has been accepted by most investigators that current flow across an ischemic border zone in acute myocardial infarction may manifest as ST segment elevation in some of the standard and augmented leads, and as ST segment depression in others. ST depression in this situation is known as reciprocal change and has classically not been considered to represent distant myocardial ischemia. It has recently been noted that the presence of residual ischemic myocardium after acute myocardial infarction, as manifested by the development of postinfarction angina (11) or a positive early exercise test (12), identifies a population at high risk for reinfarction and death. Because ischemia can be treated successfully by medical or surgical management, much attention has recently focused on the question of whether reciprocal change early in myocardial infarction represents an electrical phenomenon or true distant myocardial ischemia. In particular, the significance of precordial ST depression when there is inferior wall myocardial infarction has been debated.
**Precordial ST depression in inferior infarction.** Shah et al. (1) demonstrated that patients with precordial ST depressions during inferior myocardial infarction were at higher risk of death, congestive heart failure and postinfarction angina and suggested either additional anterior or posterior ischemia as a cause. Bush et al. (13) confirmed a high incidence of remote thallium perfusion defects in patients with inferior infarction and reciprocal change. Salcedo et al. (3) detected a high incidence of significant left anterior descending coronary obstruction in the same group of patients and assumed that precordial ST depression represented acute anterior ischemia. However, Goldberg et al. (5) found more extensive infarction involving the distribution of the obstructed artery rather than anterior ischemia by angiographic and scintigraphic methods, and Croft et al. (6) found no major differences between the groups. Our study was designed to include both anterior and inferior infarction, to explore the relation between ST elevation and ST depression early in myocardial infarction and to correlate the presence and time course of reciprocal change with serial echocardiographic assessment of remote wall function as an indicator of ischemia.

**Time course of reciprocal ST changes.** Reciprocal change is most marked in the early hours of infarction, often at a time when ST segment elevation is most striking. Our data confirmed the transient nature of these changes. In 59% of patients who had reciprocal changes on presentation, changes were absent on the routine electrocardiogram taken the next morning, less than 24 hours later. In 91%, the degree of ST segment depression had decreased by that time. Rapid resolution of such changes early in the hospital course was also documented by Croft et al. (6).

**Incidence of reciprocal ST changes.** In this study, all patients with inferior transmural myocardial infarction and 70% of patients with anterior transmural infarction had reciprocal changes, suggesting that this phenomenon is the rule rather than the exception. These incidences are higher than those reported by most authors. Both Salcedo et al. (3) and Croft et al. (6) reported an incidence rate of 82% in inferior infarction; however, the time between the onset of chest pain and presentation to the emergency room was not mentioned. Shah et al. (1) reported reciprocal change in 45% of patients with inferior myocardial infarction, but the time between onset of symptoms and admission was 7 hours for patients with reciprocal changes, and 8 hours for those without. Goldberg et al. (5) reported a 44% incidence rate in inferior infarction, but reciprocal change was defined in terms of the sum of ST depression across the precardial leads rather than maximal ST depression. The fact that we found so few patients without reciprocal changes may be related to the relatively early presentation of patients in our population; the interval between onset of chest pain and admission was 3.7 hours and 71% of patients were admitted 3 hours or less after the onset of pain. Therefore, the variability in the reported incidence of reciprocal change may be explained by differences in its definition and in the time to admission. However, the incidence is clearly high and this must severely limit the usefulness of reciprocal change in identifying a poor prognosis subgroup of patients with myocardial infarction.

**Correlation of magnitude of ST elevation and ST depression.** Significant correlation between the sum of ST elevation and the sum of ST depression was found in both anterior ($r = 0.92; p < 0.001$) and inferior infarction ($r = 0.55; p = 0.035$). This relation persisted when maximal ST elevation and maximal ST depression were correlated ($r = 0.69; p = 0.028$ and $r = 0.57; p = 0.027$ for anterior and inferior infarction, respectively). The three patients with anterior infarction who did not show reciprocal change had a mean maximal anterior ST elevation of only $1.83 \pm 0.29$ mm as compared with $3.5 \pm 0.5$ mm for patients who did have reciprocal change ($p < 0.001$). These data suggest that the factors that determine the magnitude of ST elevation also influence the amount (only) of ST depression. These factors are complex and include the position of the recording electrodes, the exact location of the infarct, the shape of
the infarct, the duration of ischemia (14), as well as infarct size (15). The relation of reciprocal change to ST elevation may explain the poorer prognosis in patients with ST depression reported by several investigators (1-4); these are likely to be patients with marked and persistent ST segment elevation and a concomitant large infarct.

Although maximal ST elevation was the same for inferior and anterior infarction (3.4 ± 1.4 mm and 3.0 ± 0.9 mm, respectively, \( p = \text{not significant} \)), maximal reciprocal ST depression was significantly greater for inferior myocardial infarction (3.5 ± 1.9 mm and 1.45 ± 0.8 mm, respectively, \( p = 0.001 \)) (Fig. 2). This is a well known clinical observation also supported by the data of Croft et al. (6).

**Role of echocardiography.** Two-dimensional echocardiography is a convenient and accurate method for evaluation of segmental wall function in acute myocardial infarction (16). In most patients, all wall segments can be visualized and studies can be repeated frequently without risk or radiation exposure. Various methods for image analysis have been introduced. Frame by frame outlining of endocardial surfaces provides quantitative data, although significant image degradation occurs when video recordings are stopped. These methods often rely on the short-axis plane alone. Analysis by visual assessment of inward movement and wall thickening provides only semiquantitative information and relies on subjective distinction between hypokinetic and normal motion, but allows for integration of various views and the use of echoes of less than perfect quality. This type of analysis has been used in contrast angiography (17), echocardiography (9) and nuclear angiography (18), and its validity and accuracy have been well established (9).

**Contraction abnormalities in noninfarcted segments.** Abnormalities in contraction of wall segments remote from the site of myocardial infarction have been noted by several investigators (9,16). Our study confirms that observation, demonstrating remote abnormalities in 45% of patients. This phenomenon may be due to various factors including infarction involving an artery with an extensive vascular distribution, unrelated congestive cardiomyopathy, mechanical tethering of adjacent wall segments without reduced perfusion (19), or ischemia. Because 74% of myocardial infarcts occur in patients with multivessel disease (20), remote ischemia may well be a common cause of remote wall motion abnormalities. The ischemia may antedate the infarction or may be of sudden onset, related to loss of collateral flow from the infarct vessel to distant vascular beds (11), a change in vessel tone after infarction (21), frank coronary spasm (22) or excessive demand placed on noninfarcted wall segments.

**Correlation of electrocardiographic and echocardiographic findings.** In this study, we assessed whether the presence and severity of reciprocal electrocardiographic changes correlated with remote wall motion abnormalities during acute myocardial infarction. Our data show that remote contractile abnormalities do not significantly change...
during the 10 days after infarction (Fig. 4) and therefore do not correlate with ST depression, which predictably resolves early. In Group I (reciprocal change present at time of first echocardiogram), wall motion did not change as reciprocal changes resolved in 9 (82%) of 11 patients (Fig. 5). Improvement or deterioration in function was each seen in one patient. Of note is that 69% of our patients with inferior myocardial infarction and reciprocal change did not have remote dysfunction, making distant ischemia an improbable cause of the electrocardiographic findings in those patients. In particular, four patients with striking reciprocal changes disproportionate to ST segment elevation (Fig. 3) did not have remote contractile dysfunction, while three patients with anterior infarction and no reciprocal change did indeed show remote hypokinesia. Failure to detect wall motion abnormalities in many patients with resolving ST depression is unlikely to be due to insensitivity of the technique, because contraction abnormalities are clearly seen during exercise-induced ischemia that is severe enough to cause ST depressions (8). Serial thallium-201 scintograms to directly assess the presence of and changes in remote perfusion defects might prove useful in confirming these findings.

It can be postulated that compensatory hyperdynamic remote wall motion might itself precipitate some degree of ischemia, resulting in ST depression that would not be associated with hypokinesia. If that were the case, return of hyperkinetic segments to normal would be expected to accompany resolution of ST depression. That pattern was not seen in our patients (Fig. 5). Therefore, although remote wall motion abnormalities are common and are probably due to ischemia in some cases, their presence is not related to the presence of reciprocal change.

Implications. Our data do not support the hypothesis that reciprocal ST segment depressions during early acute transmural myocardial infarction reflect remote ischemia. Rather, these changes are influenced by factors determining the degree of ST elevation, which include size, shape, location, transmurality and duration of infarction. The poorer prognosis previously reported in patients with reciprocal change is probably related to these factors. Reciprocal change is very common in early infarction and, therefore, its presence alone cannot identify a high risk group of patients.

We thank Joyce Rathell for her expert assistance in the preparation of this manuscript, and Diane Davis and June Sacktor for their technical help.

References
10. Bayley RH. An interpretation of the injury and the ischemic effects of myocardial infarction in accordance with the laws which determine the flow of electric currents in homogeneous volume conductors and in accordance with relevant pathologic changes. Am Heart J 1942;24:514–28.