Variability, Reproducibility, and Applications of Precordial ST-segment Mapping Following Acute Myocardial Infarction

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SUMMARY In 58 patients with uncomplicated acute anterior myocardial infarction, a mean decline in the sum of ST segments (ΣST) of 34% was observed when comparing ΣST values recorded at 3–6 hours with those recorded at 6–9 hours after the onset of symptoms (P < 0.05). The mean absolute difference between 19 paired readings 1–2 hours apart was 2.8 ± 3.0 mm and between 29 readings 2–4 hours apart 3.0 ± 3.0 mm. However, the mean absolute difference between 38 paired readings 4–8 hours apart was 12.2 ± 11.8 mm with a wide range of differences. Left ventricular failure and pericarditis were also associated with significantly higher ΣST values. We conclude that there is a complex relationship between ST-segment elevation and a number of clinical factors during the first 48 hours after infarction. Nevertheless, precordial mapping remains a useful method for the evaluation of short-term (< 4 hours) therapeutic interventions, if other relevant variables are unaltered and if carefully matched control groups are employed.

THERAPEUTIC METHODS for limiting the size of an evolving myocardial infarction have been under study, but reliable methods are needed for early characterization of alterations in the zone of ischemic injury before the efficacy of such medical or surgical interventions can be established in man. An approach of potential value is the recording of a number of unipolar epicardial or precordial electrocardiographic (ECG) leads for analysis of height and distribution of ST-segment elevations.

Most animal studies have shown a general correlation between the extent and degree of epicardial ST-segment elevations after coronary occlusion and the severity of ischemia in the tissue beneath the ECG lead, as estimated from measurements of coronary blood flow, myocardial oxygen tension, metabolic indices and the extent of myocardial necrosis on postmortem examination. In man, although such correlations are not available, a similar relationship has been postulated to exist between ischemic injury and the precordial ECG leads. Several investigators have shown that acute pharmacological interventions in patients can lead to a decrease in the sum of ST-segment elevations (ΣST) measured from multiple precordial leads and hence to a possible reduction of myocardial ischemia.

Interpretation of induced ST-segment changes requires knowledge of their evolution over time from the onset of ischemia and of the directional alterations which can be associated with physiologic and pathologic changes. Accordingly, we have reviewed serial ST-segment maps in 58 selected patients in order to provide data on: 1) the natural time course of ST elevations during a 48 hour period following admission for acute myocardial infarction, the period when interventions are most likely to be contemplated; 2) reproducibility of ST maps at intervals varying from one to eight hours; 3) correlations between variations in ΣST in individual patients and the development of pericarditis and/or chest pain; and 4) other factors in the patients’ course including clinical class, heart rate and blood pressure changes, and heart size.

Patients and Methods

The following criteria were set for the review and acceptance of multiple precordial ECG recordings (maps).

1) All patients included in the study had an acute transmural myocardial infarction involving the anteroseptal or anterolateral wall of the left ventricle, as determined by a history of prolonged chest pain, characteristic elevations in serum enzymes (creatine kinase, glutamic-oxaloacetic transaminase and lactic dehydrogenase) and serial 12-lead electrocardiograms.

2) No patient had had a previous infarction involving the anterior or lateral walls of the left ventricle.

3) All patients were in sinus rhythm at the time of the study. ST maps were excluded when the QRS duration exceeded 0.09 sec or when a definite change in the QRS frontal plane axis occurred between two recordings. None of the patients had evidence of left or right ventricular hypertrophy by ECG criteria.

4) In all patients, at least two ST maps were available during the first 48 hours following the onset of acute symptoms; in a number of patients, maps were also available beyond that time period.

5) None of the patients had historical or clinical evidence of other systemic diseases.

Among 318 patients admitted and studied over a 5-year period in the Research Unit at the University of California, San Diego, 141 patients had anterior infarction and underwent ECG mapping. Fifty-eight patients satisfied criteria for inclusion in this study. These 58 patients had 354 maps that were acceptable for analysis, with an average of 6.1 maps per patient. Eleven patients had 10 or more maps. Forty patients were men and 18 were women. Their average age

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was 59.5 years (range 36 to 82 years). The mean time from onset of acute symptoms to admission to the unit was 9 ± 4.5 (sd) hours. All the studies were referenced to the time of the onset of acute symptoms. Twelve patients had experienced previous infarcts which were inferior (nine patients), posterior (one patient) or subendocardial (two patients) by ECG criteria.

The patients were placed in classes I–IV, according to the presence and severity of left ventricular failure.¹⁸ On the first hospital day, 17 patients were in class I, 36 in class II, four in class III and one in class IV.

Sixteen patients (28%) developed symptoms and signs suggestive of pericarditis during their stay in the research unit. In five patients, pain consistent with myocardial ischemia recurred during the second day after the onset of their initial symptoms but serial serum enzymes (CK and GOT) revealed no evidence of infarct extension. Other major complications were absent during the period of study.

The systemic arterial pressure by cuff sphygmomanometer and the heart rate were measured simultaneously with the ST mapping. The serum electrolytes were measured daily in all patients. In three patients, correction of hypokalemia by the oral administration of KCl was associated with an insignificant average SST increase from 41.5 mm on the day of admission to 44.5 mm (individual increases: 2, 3 and 4 mm) on the following day, but the serum electrolytes were normal in all the other patients. The left heart dimension was assessed in 42 patients using our previously described radiographic method.¹⁹

Seven patients received digoxin during the first 48 hours and seven other patients were treated with intravenous trimethaphan. Two patients received intravenous practolol and two intravenous propranolol. ECG maps obtained during or following drug administration were excluded from the analyses.

For the ST-segment mapping, a blanket containing 35 unipolar electrodes arranged in seven horizontal and five vertical columns was connected to a switch which in turn was connected to the unipolar V lead of an electrocardiograph.¹ The right upper corner of the blanket was located over the second right intercostal space, adjacent to the sternum, and the site was marked to assure identical locations in the same patient at each study. The left border of the blanket was close to the midaxillary line. Any ST-segment elevation above the baseline was measured to the nearest 0.5 mm, 20 msec following the J-joint and at least three ECG complexes averaged. All the ST elevations were summed for each study and expressed as SST. The extent of ST elevations was also evaluated by counting the number of sites having ST-segment elevations equaling 1 mm or more (nST). Although changes in nST paralleled changes in SST, they did not reach statistical significance. The subsequent discussion is therefore limited to SST.

For study of the natural evolution of ST-segment elevations the period from three to 48 hours after the onset of symptoms was divided into seven time windows: 3–6, 6–9, 9–12, 12–18, 18–24, 24–36 and 36–48 hours after the onset of symptoms. The mean SST values were calculated for the ST maps recorded within each time interval; the earliest SST value for each patient within that time interval is reported if more than one was available. Six patients had complete data for all seven time windows (table 1). In fourteen other patients SST values were interpolated from the SST of adjacent time windows, thereby substituting a total

<table>
<thead>
<tr>
<th>Table 1. SST Measurements from Twenty Uncomplicated Patients</th>
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<tr>
<td>SST (3–6 hours)</td>
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<tr>
<td>-----------------</td>
</tr>
<tr>
<td>11</td>
</tr>
<tr>
<td>12</td>
</tr>
<tr>
<td>13</td>
</tr>
<tr>
<td>20</td>
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*Values interpolated from SST from the two adjacent time intervals.
Numbers in brackets indicate the time of the ST-segment recording in reference to the time of acute symptoms.
The natural evolution of $\Sigma ST$-changes with time from the onset of acute symptoms. Figure 1a presents the $\Sigma ST$ time course for ten patients with complete data for all time intervals. A significant decline in $\Sigma ST$ values occurred between three to six and six to nine hours, but no significant changes were found subsequently. The insert (top) shows the percentage changes in $\Sigma ST$, with the 3–6 hour mean value as 100%. Figure 1b shows the evolution of $ST$ over time for twenty patients (see table 1 except for the first time window which depicts 10 patients) utilizing in addition interpolated $\Sigma ST$ values from the adjacent time intervals. Figure 1c shows the $\Sigma ST$ time course from the data from all 58 patients.

The individual $\Sigma ST$-maps were constructed from the serial $\Sigma ST$ of these 20 patients (table 1, fig. 1b). None of the 20 patients had received drug therapy during the 48 hour period. The $\Sigma ST$ time course was also constructed separately from $\Sigma ST$ values for the ten patients who had $\Sigma ST$ measurements in the early time period of three to six hours after acute symptoms and subsequent time intervals (table 1, fig. 1a), and also from all $\Sigma ST$ values of the 58 patients (fig. 1c). For the latter analysis, the precordial maps were excluded if a patient received digoxin, trimethaphan, beta-adrenergic blocking agents, developed a pericardial rub, or recurrence of chest pain.

In the study of $\Sigma ST$ reproducibility, the variations between paired $\Sigma ST$ measurements in the same patient spaced 1–2 hours, 2–4 hours and 4–8 hours apart were examined. Only recordings taken 12 to 48 hours after the onset of symptoms were included, and ST changes were examined only when the clinical condition of the patient was unaltered and unaffected by change in treatment. Thus, recordings associated with an unstable clinical condition, such as recurrent chest pain, increasing heart failure, the development of pericarditis, or changes in blood pressure or heart rate of over 10% were disregarded, as were recordings shortly preceded by a new treatment or a change in treatment. Seventeen patients had ST-segment recordings for all three time intervals (1–2 hours, 2–4 hours and 4–8 hours), 25 patients had ST-segment recordings for two time intervals and 16 patients for one of the time intervals. The variations between paired $\Sigma ST$ measurements were examined for the group of seventeen patients with complete data for the three time intervals and for the total population.

Standard statistical methods were used and included Student's $t$-test for paired data, correlation studies and repeated measures of analysis of variance. All deviation symbols ($\pm$) in the text indicate the standard deviation.

### Results

#### Natural Evolution of ST Changes

In the patients who had an uncomplicated early course, without therapeutic interventions, the first precordial maps were recorded between three and six hours after the onset of symptoms. The individual data from the 10 patients whose initial ST-maps were recorded three to six hours after the onset of symptoms are shown in table 1. The mean $\Sigma ST$ value at 3 to 6 hours was $66.5 \pm 54$ mm, at 6–9 hours $43.8 \pm 28$ mm and at 9–12 hours $41.3 \pm 23$ mm (fig. 1a). Between the first two time windows the mean $\Sigma ST$ declined by 34% (insert, fig. 1a). Analysis of variance comparing the paired $\Sigma ST$ values from the first two time windows reveals that the fall in $\Sigma ST$ values was statistically significant ($P < 0.05$).

Figure 1b shows the serial mean $\Sigma ST$ measurements for the 20 patients utilizing all data presented in table 1. The mean $\Sigma ST$ in the 10 patients with maps at 3–6 hours was $66.5 \pm 54$ mm. At 6–9 hours, the average value in all 20 patients was $32.2 \pm 26.6$ mm, and remained stable at 9–12 hours ($30.0 \pm 24.7$ mm). Minor and insignificant mean
changes in ΣST between 12 and 48 hours after the onset of symptoms were observed, although the individual variation was considerable. The mean ΣST values were similar when all data from all 58 patients for the seven time intervals were averaged (fig. 1c).

Reproducibility of ST Maps

In the study of ΣST reproducibility, a number of paired precordial ST-segment maps spaced 1–8 hours apart were compared in individual patients. The results are summarized in table 2. In the 17 patients who had ST-segment record-ings for all three time intervals, paired ΣST measurements (ΣST₁ and ΣST₂) one to two hours apart showed small mean absolute differences between ΣST₁ and ΣST₂, but coupled measurements four to eight hours apart showed a larger mean absolute difference with a wide range of differences and large standard deviations (table 2A). Table 2B shows the paired ΣST measurements for the three time intervals for the total population which showed similar trends. The paired ΣST measurements for the three time intervals are summarized in figure 2. There was minimal variation between paired readings which were obtained 1 to 2 hours and 2 to 4 hours apart, and correlations between the first and

![Table 2. Reproducibility of ΣST](image)

**TABLE 2. Reproducibility of ΣST**

<table>
<thead>
<tr>
<th>A. Paired ΣST Measurements from 17 Patients</th>
<th>1-2 Hours</th>
<th>2-4 Hours</th>
<th>4-8 Hours</th>
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<tbody>
<tr>
<td>Number of patients (number of intervals)</td>
<td>17</td>
<td>17</td>
<td>17</td>
</tr>
<tr>
<td>Mean difference of ΣST (mm)</td>
<td>−0.6 ± 4.2</td>
<td>1.1 ± 2.3</td>
<td>−3.8 ± 11.3</td>
</tr>
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<td>Range of differences (mm)</td>
<td>−5.9 to 12.0</td>
<td>6.0 to 4.5</td>
<td>−18.9 to 16.7</td>
</tr>
<tr>
<td>Mean absolute difference of ΣST (mm)</td>
<td>2.8 ± 3.1</td>
<td>1.8 ± 1.8</td>
<td>9.7 ± 6.6</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>B. Paired ΣST Measurements from All 58 Patients</th>
<th>19</th>
<th>29</th>
<th>38</th>
</tr>
</thead>
<tbody>
<tr>
<td>Number of patients (number of intervals)</td>
<td>19</td>
<td>29</td>
<td>38</td>
</tr>
<tr>
<td>Mean difference of ΣST (mm)</td>
<td>−0.3 ± 4.2</td>
<td>0.2 ± 4.3</td>
<td>−3.7 ± 16.5</td>
</tr>
<tr>
<td>Range of differences (mm)</td>
<td>−5.9 to 12.0</td>
<td>−11.5 to 10.5</td>
<td>−45.6 to 50.0</td>
</tr>
<tr>
<td>Mean absolute difference of ΣST (mm)</td>
<td>2.9 ± 3.0</td>
<td>3.0 ± 3.0</td>
<td>12.2 ± 11.8</td>
</tr>
</tbody>
</table>

**Figure 2.** The correlation between the first (ΣST₁) and second (ΣST₂) of paired precordial ST-segment recordings, separated by 1–2 hours, 2–4 hours and 4–8 hours. The diagonal lines represent the lines of identity. A considerably increased scatter was present over 4–8 hours in comparison with the shorter time intervals (1–4 hours). The increased variability is particularly evident at the higher ΣST values. Figure 2a shows the ΣST measurements for the three time intervals in the same 17 patients. Figure 2b depicts the paired ΣST measurements from 19 patients for the 1–2 hour recording interval, from 29 patients for the 2–4 hour interval and from 38 patients for the 4–8 hour interval.
second ΣST measurements were good \((r = 0.98\) and \(r = 0.99\), respectively [figs. 2a and 2b]). There was more variability in the ΣST measurements when obtained in 4 to 8 hour intervals with a less favorable correlation between the first and second ΣST reading \((r = 0.91\), and \(r = 0.87\), respectively).

**Pericarditis and Chest Pain**

Fifteen patients (26%) in the group studied had or developed pericarditis. In two patients, a pericardial rub was present on admission, and two patients developed pericarditis later than 48 hours after the onset of acute symptoms. In the remaining eleven patients (fig. 3), the appearance of a pericardial rub was associated with an increase in ΣST values from 46.1 ± 30 to 64.7 ± 30.9 mm \((P < 0.001)\). The time between the paired recordings was 8.2 ± 2.7 hours and the mean time of recording for the first of each pair of maps was 17.5 hours (range 9 to 46 hours) after the onset of symptoms. The development of pericarditis was not associated with significant changes in heart rate or arterial blood pressure, and no secondary rises in CK were observed. In five patients, moderate to severe ischemic chest pain unassociated with a friction rub recurred later than 10 hours after the initial symptoms. This was associated with an average increase of 19.6 mm (range 7.3 to 51.7 mm) in ΣST in four patients, whereas no change in ΣST occurred in the other patient. No secondary rises in serum CK values were noted in these patients but the heart rate-blood pressure product increased by an average of \(1.9 \times 10^{10}\) \((0.5, 1.7, 3.5 \times 10^{10}, \) respectively) in three patients, decreased in one patient, and remained unchanged in the patient who had stable ΣST with the occurrence of ischemic chest pain.

**Other Clinical Correlations**

There were weak positive associations between the highest ΣST of the first 24 hours for each patient and the simultaneously measured systolic blood pressure \((r = 0.40)\), diastolic blood pressure \((r = 0.44)\), and heart rate \((r = 0.30)\). The product of heart rate and systolic blood pressure showed a somewhat stronger correlation \((r = 0.51)\). Changes in the double product correlated weakly with simultaneous changes in the ΣST \((r = 0.32)\). This correlation was not improved by examining only short-term \((< 6\) hours) changes \((r = 0.34)\).

No correlation was found between ΣST and the radiological measurement of the initial left heart dimension. However, serial changes in the ΣST and simultaneously measured changes in the left heart dimension showed a weak positive correlation \((r = 0.40)\).

The highest ΣST values recorded on the first day were related to the initial clinical class. The average highest ΣST in patients in clinical class II was 57.9 ± 45.8 mm, a value significantly higher than the average ΣST value in clinical class I patients \((24.6 \pm 24.0\) mm, \(P < 0.02)\). In addition, the highest ΣST measured between 24 and 48 hours after onset of symptoms when related to the simultaneously determined clinical class was also different: the average ΣST value was 46.7 ± 36.7 mm in patients in clinical class II and 20.6 ± 19.6 mm \((P < 0.01)\) in patients in clinical class I.

The number of patients in class III (four) and in class IV (one) were too few to analyze.

**Discussion**

Early indicators of the extent of eventual ischemic injury during acute myocardial infarction ideally should reflect accurately the amount of ischemic myocardium at a given time and allow a quantitative determination of reversal or progression of the ischemic process. An ideal indicator also should make possible an early prediction of the extent of the completed infarction. Several methods for estimating or predicting infarct size have been proposed, but have so far found limited application in man.\(^4\) The measurement of the precordial ST-segment elevations has the advantage of being rapidly responsive to acute therapeutic interventions.\(^4, 13-16\)

Several reports have been published concerning the effects of acute pharmacological interventions on the precordial ECG, but quantitative data on the temporal evolution of precordial ECG changes in the early hours following infarction have been lacking and reproducibility of ST changes has been uncertain. In analyzing the natural evolution of ST changes, an attempt was made to carefully select only those patients whose course was uneventful and free of therapeutic interventions. This approach may, of course, have resulted in exclusion of many patients, some of whom had severe symptoms, and thereby have led to some bias in the series of patients reported. Thus the conclusions of this investigation apply only to the highly selected subset of patients we studied. To illustrate this problem, we examined the records of 141 consecutive patients with acute anterior transmural infarction. Thirty-four patients \((24\%)\) had either pericarditis or infarct extension during the first 48 hours; twenty-three patients \((16\%)\) had either complete heart block or bundle branch block; eighty-nine patients \((63\%)\) received one or more of the following medications: digoxin, isoproterenol, norepinephrine, beta-adrenergic blocking agents, or anti-hypertensive agents. Ten other patients were in clinical class
I and had an uneventful clinical course. It was therefore difficult to identify a sizable, representative group of patients with acute anterior myocardial infarction who pursued an uneventful and stable course during their first two days in the hospital. In studying short-term reproducibility, we were nevertheless able to choose short time intervals for analysis in 44 patients which were associated with no major clinical or therapeutic changes.

In this study, a significant downward trend in mean 3ST occurred between three and 12 hours. From 12 to 48 hours there were no significant mean changes in 3ST, although considerable individual variation was present. An intervention study would have to take into account this early trend, a particularly important consideration if the view is adopted that interventions must be utilized early in order to be effective in salvaging a significant amount of ischemic myocardium. However, the relative stability of mean values at 12 to 48 hours lends support to the use of ST mapping during this period for the evaluation of acute interventions. Studies in dogs show that following acute coronary occlusion, the ST segments tend to rise within one minute and reach a maximum after about 5-7 minutes. They then remain stable for at least one-half hour, but a decline is observed by three hours. Studies in monkeys have shown a maximal rise of ST segments at two hours after coronary occlusion which is followed by a decline for at least six hours, a finding in good agreement with the present study. The results of the reproducibility studies 12 hours or more after the onset of symptoms show that over periods ranging from one to four hours, the 3ST remains relatively stable provided that no major therapeutic or clinical changes occur. Over these time ranges, using estimates of variance derived from the mean difference values for 3ST in table 2A, and employing standard methods for calculating sample sizes, a group of seven to eight patients undergoing a therapeutic intervention compared with an equal size control group would suffice to reveal a mean 3ST difference of 10 mm at the 5% probability level. For periods of 4-8 hours, however, reproducibility was markedly reduced, and groups of 35-40 patients in each group would be required to evaluate the results of medical or surgical treatment based on the same criteria. The reproducibility of 3ST values at 3-12 hours was not analyzed separately in view of the substantial downward trend. Thus, 19 of the 20 patients (95%) who had serial ST recordings in the first 12 hours after onset of symptoms showed a decline of 3ST values and in one patient 3ST values remained unchanged during this period.

The significant increase in 3ST in eleven patients associated with the development of pericarditis shows that complications of infarction unrelated to the ischemic process may markedly modify the course of ST changes. It is also possible that unrecognized infarct extensions may have caused secondary ST-segment elevations in some of our patients, as suggested by Reid et al., although this was not supported by associated late rises in cardiac enzymes.

A secondary rise in 3ST within the first 48 hours was observed in 15 patients who had no symptoms or clinical signs of pericarditis or secondary rises in CK. However, 15 other patients who had serial 3ST measurements over the first 48 hours after the onset of symptoms exhibited a decrease of ST-segment elevation over this time period. The secondary rise or persistence of ST elevations occurred in patients with large myocardial infarctions as determined from completed CK curves. Thus, the average CK infarct size of 51.0 ± 14.9 I.U./1-h was significantly larger in patients with a secondary 3ST rise or persistence of ST-segment elevation than in patients without 3ST rise or persistent ST-segment elevation (21.3 ± 12.6 I.U./1-h, P < 0.01). This finding is similar to the observations of Mills et al. who found persistent ST-segment elevation in patients with anterior infarction who had high levels of maximal serum CK and severe left ventricular failure. Similarly, Kronenberg et al. observed persistently elevated ST segments in patients with acute myocardial infarction associated with congestive heart failure and a progressive decrease in clinically uncomplicated patients. It is uncertain whether pericarditis not detected clinically contributed to the observed secondary ST-segment elevations.

The amplitude and extent of ST-segment elevations in the early phase of acute myocardial infarction has been related to the size of infarction. The variable distribution of infarct size may therefore largely account for the variable 3ST at a given time after onset of acute infarction in a large group of patients with acute myocardial infarction, and the characteristic time evolution of 3ST may be modified according to infarct sizes in a study group. This is further supported by our findings in 13 patients who had an estimate of infarct size by CK curve analysis and ST-segment recordings obtained an average of 10 hours after onset of symptoms with a difference of less than 1.5 hours in the time of recording between patients. In eight patients with large infarct sizes over 35 I.U./1-h (average time of 3ST recording 11.1 hours after onset of symptoms), the mean 3ST of 50.5 ± 29.0 mm was significantly higher than the mean value of 13.4 ± 10.8 mm (P < 0.02) in the five patients with smaller infarcts (average time of 3ST recordings 10.9 hours after acute symptoms). Similarly, Thompson et al. have observed a correlation between the early 3ST measurements and maximal CK in patients with acute anterior myocardial infarction.

The present study indicates that other factors, such as recurrence of ischemic chest pain without enzymatic evidence of infarct extension, also can cause shifts of the ST segments. Furthermore, fluctuations in serum potassium levels may cause ST-segment alterations. In our series only three patients had hypokalemia and showed small increases in 3ST with correction of the low potassium levels. However, animal experiments show that coronary perfusion with a solution high in potassium ion concentration results in ST-segment elevation on the epicardial ECG lead, accompanied by TQ-segment depression on the intracellular electrogram. The systolic blood pressure-heart rate product was positively related to 3ST. These and other determinants of myocardial oxygen consumption also need careful consideration when interpreting apparently spontaneous short-term ST changes. The early instability of 3ST values underlines the importance of a carefully matched control group for any therapeutic intervention performed during the first 12 hours. In addition, if any individual patient is used as his own control, the fall in 3ST after drug administration should substantially exceed the expected natural decline in ST-segment elevations found between three and 12 hours, to be considered significant. Moreover as shown by our reproducibility studies, interventions examined after 12
hours should have a sufficiently rapid effect to produce \( \Delta S T \) changes within four hours. Although a recent study indicates little change in \( \Delta S T \) over one hour in stable patients during the acute phase of myocardial infarction earlier than 12 hours after onset of acute symptoms,\textsuperscript{83} the majority of our patients showed a progressive decline in \( \Delta S T \) during the first 12 hours. Thus, in 10 patients with stable clinical course and no change in heart rate and blood pressure there was a statistically significant \( (P < 0.02) \) mean decline of 9.3 mm between \( \Delta S T \) recordings 50 minutes to 1.2 hours apart obtained between 2 and 11 hours after onset of symptoms; in this group, \( \Delta S T \) decreased by more than 5% in all but one patient. In the previously mentioned study, individual \( \Delta S T \) changes of over 30% were observed between short-term recordings in the first 12 hours after infarction.\textsuperscript{83}

Despite these individual variations in the evolution of \( \Delta S T \) the technique of ST-segment mapping has been employed as a useful means to document the effects of therapeutic interventions. Studies in animals by Maroko et al.,\textsuperscript{1} and in man by Pelides et al.\textsuperscript{16} and Maroko et al.\textsuperscript{18} using precordial ST maps have shown that beta-adrenergic blockade is associated with a decrease in \( \Delta S T \). The reduction of afterload by sublingual or intravenous nitroglycerin has also been reported to diminish ST-segment elevations in patients during acute myocardial infarction.\textsuperscript{84, 85} Other pharmacological studies have shown that the administration of hyaluronidase\textsuperscript{45, 47} and oxygen inhalation\textsuperscript{48} are associated with a decline in ST-segment elevation following acute myocardial infarction and a similar decline was seen in dogs after corticosteroid administration\textsuperscript{89} using epicardial leads.

Using epicardial leads experimentally, \( \Delta S T \) values have shown a relationship to a number of functional and metabolic markers of ischemic myocardial damage,\textsuperscript{8} such as local lactic acid accumulation,\textsuperscript{9} potassium efflux,\textsuperscript{20, 40} ATP and CP depletion,\textsuperscript{7} decrease in local oxygen tension and diminution of coronary flow.\textsuperscript{8-12} A linear relation to log myocardial CK-activity 24 hours later has also been reported.\textsuperscript{17} In animal experiments and human studies, directional alterations in myocardial oxygen requirements and delivery are accompanied by corresponding \( \Delta S T \) variations.\textsuperscript{1, 3, 38, 41}

It is not easy to compare the results of well-controlled animal experiments with those of human studies in which a number of additional factors must be taken into account, particularly those concerning therapy. The results of our study illustrate the complex, interdependent relationship which exists between temporal (time since infarction), pathologic (pericarditis) and physiologic (heart rate, blood pressure) factors, as well as infarct size, which may complicate interpretations of ST changes in individual patients undergoing routine hospital treatment. Alternative techniques, such as R-wave mapping\textsuperscript{15, 26, 27} and ST-vectorcardiography,\textsuperscript{28, 42, 43} or combined analysis of ST segment and R wave changes in precordial maps\textsuperscript{44} may offer some advantages. Nevertheless, this study demonstrates that later than 12 hours from the onset of symptoms, and over relatively short periods of time, ST-segment elevations remain quite stable if standard variables known to affect them remain unaltered. Provided that such variability of the ST segments as well as the basic time trends are recognized, precordial mapping remains a potentially useful method for the evaluation of rapidly-acting interventions in groups of patients during acute myocardial infarction.

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**Conditions for Vasodilator-induced Coronary Steal in Experimental Myocardial Ischemia**

**Lewis C. Becker, M.D.**

**SUMMARY** The conditions for coronary steal were determined using the two vasodilators — dipyridamole and nitroglycerin — in anesthetized dogs after ligation of the left anterior descending coronary artery (LAD). Previous studies have shown that when the non-ligated coronary arteries are normal, collateral flow increases after dipyridamole. This study utilized a model in which the distal LAD was ligated and the proximal LAD and left circumflex (LC) arteries were stenosed. Heart rate and blood pressure were kept constant. In 20 dogs, 1–1.5 mg/kg dipyridamole caused a decrease in flow to the ischemic region as measured by radioactive microspheres (0.19 to 0.14 ml/min/g, P = 0.05) while flow increased four-fold to surrounding nonischemic myocardium. The decrease in collateral flow was confined to the epicardial half of the ischemic region (0.26 to 0.14 ml/min/g, P < 0.001) and was associated with an increase in ST from 30.9 to 44.7mV (P < 0.01). In five dogs nitroglycerin, 5 µg/kg/min, produced no significant changes in collateral flow or flow to other parts of the LV, and ST was unchanged. Vasodilator-induced coronary steal therefore appears to require 1) an arteriolar-type dilator like dipyridamole and 2) stenoses of the arteries supplying collateral flow to the ischemic region. The steal phenomenon is probably caused by a decrease in pressure distal to the stenoses in these vessels, resulting in reduced driving pressure for collateral flow.

**VASODILATOR DRUGS** are gaining increasing use in the treatment of acute myocardial infarction. Vasodilators have been found to improve left ventricular function in patients with congestive heart failure or low cardiac output states resulting from myocardial infarction.1 In addition, there is evidence to suggest that vasodilators may limit myocardial ischemia in the early stages of acute myocardial infarction.2,4 At least part of the beneficial effect on ischemia has been attributed to the decreases in myocardial oxygen demands which result from reductions in blood pressure and left ventricular filling.1,3 However, experimental studies in animals support the concept that vasodilators may also reduce ischemia by increasing collateral blood flow to ischemic myocardium.5–7 Both direct and indirect effects on the heart could theoretically account for improved collateral flow. Direct dilatation of collateral channels has been demonstrated in several studies,5–8 but reduction in left ventricular filling could also improve collateral flow indirectly by decreasing compressive forces on collateral vessels.9

Under certain conditions, however, vasodilators may have adverse effects on collateral flow. Vasodilator-induced hypotension is generally beneficial because of the associated decrease in left ventricular wall tension, but an excessive reduction in perfusion pressure may be harmful.10 When the decrease in pressure is disproportionately more than the reduction in collateral vessel resistance, collateral flow diminishes.5–8, 11 Furthermore, vasodilators may decrease flow to an ischemic region by markedly increasing flow to surrounding nonischemic areas.10–12 This diversion of flow from ischemic to nonischemic tissue has been termed a myocardial steal or coronary steal.12, 14 The steal is believed to be related to a selective dilatation of resistance vessels in nonischemic areas; since vessels within ischemic regions are
Variability, reproducibility, and applications of precordial ST-segment mapping following acute myocardial infarction.

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