Reelevation of ST Segment on Precordial Mapping in Natural Time Course Following Acute Anterior Myocardial Infarction

OSAMU NAKAGAKI, M.D., HIROSHI YANO, M.D.
ARAHITO MITSUTAKE, M.D., YUTAKA KIRUCHI, M.D.
AKIRA TAKESHITA, M.D., Hideo Kanaide, M.D.
and MOTOOMI NAKAMURA, M.D.

Serial recordings of ST segment mapping were performed in 15 patients with acute anterior myocardial infarction. Reelevation of ST segment was observed in all cases between 12 hours and one week after infarction without clinical evidence of reinfarction. There was significant correlation between the magnitude of the sum of ST reelevation (ΣST) and time to the peak of ST reelevation. The higher was ΣST at the peak, the later was the appearance of the peak of ST reelevation. ΣST recorded between 24 and 72 hours after infarction correlated with the extent of abnormal Q (NQ) in the precordial mapping as well as the percent of abnormally contracting segment (%ACS) on left ventricular angiography examined after 4 weeks following infarction. However, there was no correlation between ΣST recorded at 12 hours after infarction and NQ studied at 4 weeks after infarction. The results indicate that the magnitude of ΣST changes in time during the course after infarction and that the time of recording is important when ΣST is used for the estimation of infarct size. Reelevation of ST segment should be considered in the study of therapeutic intervention in acute myocardial infarction.

The electrocardiography is one of the most useful technique for the diagnosis of myocardial infarction. The electrocardiographic mapping with multi-lead system is said to be useful in the determination of the infarct size and evaluation of the efficacy of the therapeutic intervention. Experimentally, it has been reported that ΣST on the epicardial or precordial leads in acute coronary occlusion correlated with the enzymatically and histologically estimated infarct size. In clinical studies, precordial ST mapping has been used in evaluation of therapeutic intervention on the extent of infarction in the patient with acute myocardial infarction. However, there are a few studies which have shown that ΣST does not correlate with the infarct size estimated by serum CK activity.

In this study, we examined the serial recordings of precordial electrocardiographic mapping in patient with acute anterior myocardial infarction to clarify the time course of ST elevation,
and to examine correlation between $\Sigma$ST and indices of infarct size, namely the extent of abnormal Q and of asynergy of the left ventricle.

METHODS

Seventeen patients, including 15 males and 2 females, with acute anterior infarction were studied with serial recordings of 36 precordial electrocardiogram. Their ages ranged from 30 to 68 years old (an average 54 years old). The diagnosis of acute myocardial infarction was made by the symptom of severe chest pain sustained over 60 minutes, evolutional changes of serum enzyme activity, and electrocardiographic criteria from AHA committee report (1974). The first recording of precordial mapping was made within 12 hours after the onset of chest pain in 9 patients, within 24 hours in 6 patients and after one week in 2 patients. The cases combined with inferior infarction or complicated by reinfarction or bundle branch block were removed from the study.

Precordial mapping were recorded using 36 (6 x 6) leads on the anterior chest wall. Analysis of precordial ECGs were done with the computer (IBM S/7, S/370). The ST elevation and the width of Q wave were measured by the IBM-Bonner program. The ST map and Q map were printed on the line printer (Fig. 1), and $\Sigma$ST and

Fig. 1. Schematic illustration of the positions of 36 precordial leads, and the ST and Q map of a typical case with acute anterior myocardial infarction. The ST map shows the distribution of each level of ST elevation which is graded every 0.1 mV, and the Q map shows the extent of abnormal Q wave with more than 40 msec duration.

Fig. 2. A typical course of serum CK activity and the magnitude of $\Sigma$ST in a patient with acute anterior myocardial infarction. ST reelevation was noted without the re-elevation of serum CK activity.
Fig. 3. Changes of $\sum$ST in the serial precordial mappings in 15 patients with acute myocardial infarction. ST reelevation was noted between 12 hours and one week after the onset of myocardial infarction. The peak of $\sum$ST reelevation in each patient is shown by a star mark.

Fig. 4. Relationship between the magnitude of $\sum$ST at the peak of ST reelevation and the time at the peak of $\sum$ST reelevation after the onset of infarction. There was significant correlation between these parameters.

NQ were calculated. $\sum$ST is the sum of ST segment elevation in 36 precordial leads, and NQ is number of precordial leads where abnormal Q wave with more than 40 msec duration was observed.

Left ventricular cineangiography was performed in 11 patients, one month after acute myocardial infarction. Utilizing the modified Leighton's method, the end-diastolic and end-systolic silhouettes of the left ventricle in the $30^\circ$ right anterior oblique plane were traced and superimposed in order to correct the rotation. Instead of dividing the longitudinal line into 5 segments as done by Leighton et al., we divided it into 10 equal segments and 9 minor axis were drawn perpendicular to the longitudinal axis. The segment of myocardium in which systolic shortening of minor axis was less than 5 percent was considered as abnormally contracting segment (ACS). And %ACS was obtained by dividing numbers of ACS by total (18) myocardial segments.

Serial determination of serum creatine kinase (CK) activity were done at every 4 to 12 hours. CK activity was measured according to the Rosalki's method.

RESULTS
A typical course of $\sum$ST and serum CK activity is presented in Fig. 2. The patient who was
TABLE I  CORRELATION COEFFICIENTS BETWEEN $\Sigma$ST RECORDED AT VARIOUS TIMES AFTER THE ONSET OF INFARCTION AND NQ RECORDED AT 4 WEEKS AFTER INFARCTION

<table>
<thead>
<tr>
<th>Time after onset of anterior infarction</th>
<th>Number of cases</th>
<th>Correlation coefficient</th>
</tr>
</thead>
<tbody>
<tr>
<td>12 Hours</td>
<td>9</td>
<td>0.380</td>
</tr>
<tr>
<td>24</td>
<td>14</td>
<td>0.730***</td>
</tr>
<tr>
<td>48</td>
<td>13</td>
<td>0.757***</td>
</tr>
<tr>
<td>72</td>
<td>13</td>
<td>0.682**</td>
</tr>
<tr>
<td>1 Week</td>
<td>15</td>
<td>0.751***</td>
</tr>
<tr>
<td>2</td>
<td>15</td>
<td>0.686**</td>
</tr>
<tr>
<td>3</td>
<td>15</td>
<td>0.614*</td>
</tr>
<tr>
<td>4</td>
<td>15</td>
<td>0.766***</td>
</tr>
</tbody>
</table>

*P < 0.05, **P < 0.02, ***P < 0.01

TABLE II  CORRELATION COEFFICIENTS BETWEEN $\Sigma$ST RECORDED AT VARIOUS TIMES AFTER THE ONSET OF INFARCTION AND %ACS ESTIMATED BY LEFT VENTRICULAR CINEANGIOGRAM (LVG)

<table>
<thead>
<tr>
<th>Time of LVG after onset</th>
<th>Number of cases</th>
<th>Correlation coefficient</th>
</tr>
</thead>
<tbody>
<tr>
<td>24 Hours</td>
<td>9</td>
<td>0.754**</td>
</tr>
<tr>
<td>48</td>
<td>8</td>
<td>0.813**</td>
</tr>
<tr>
<td>72</td>
<td>8</td>
<td>0.815**</td>
</tr>
<tr>
<td>1 Week</td>
<td>11</td>
<td>0.685*</td>
</tr>
<tr>
<td>2</td>
<td>11</td>
<td>0.598</td>
</tr>
<tr>
<td>3</td>
<td>11</td>
<td>0.585</td>
</tr>
<tr>
<td>4</td>
<td>11</td>
<td>0.765***</td>
</tr>
</tbody>
</table>

11                       | 0.764***

*P < 0.05, **P < 0.02, ***P < 0.01

41 years old male was admitted with sustained chest pain, not relieved by sublingual administration of nitroglycerin. He had been treated for one year because of variant type of angina pectoris. Serial blood sampling for CK activity and precordial ECG mapping were started 3 hours after the onset of chest pain. In this case, serum CK activity was increased at 12 hours after the onset of chest pain and attained the peak within 24 hours. Thereafter it gradually decreased and returned to the normal level by 72 hours. The ST segment was initially elevated but this initial elevation was gradually increased over the first 16 hours. The reelevation of ST segment occurred after 22 hours and reached the peak at 60 hours, but CK did not show reelevation at this time.

Serial ST mapping were performed beginning at the early phase of infarction in 15 patients with acute anterior myocardial infarction. The time course of $\Sigma$ST in these patients were presented in Fig. 3.

$\Sigma$ST reelevation was defined as follows: (1) The reelevation of $\Sigma$ST after initial drop. (2) The gradual increase of $\Sigma$ST from the first recording (Fig. 3). The reelevation of $\Sigma$ST was recognized between 12 hours and one week in all 15 patients. Pericardial friction rub was ausculted in only 5 patients among them. Strong correlation was noted between the magnitude of $\Sigma$ST at the peak of reelevation and the time to the peak of $\Sigma$ST reelevation ($r = +0.754$, p < 0.002) (Fig. 4).

In order to examine the relationship between $\Sigma$ST and the infarct size, we examined correlation between $\Sigma$ST at various time after infarction...
and NQ in precordial mapping recorded at 4 weeks after infarction, and between \( \Sigma ST \) and \%ACS evaluated by left ventricular cineangiography. Correlation coefficients between \( \Sigma ST \) and NQ are shown in Table I. There was statistically significant correlation between NQ and \( \Sigma ST \) recorded between 24 hours and 4 weeks, but not at 12 hours.

Correlation coefficients between \( \Sigma ST \) and \%ACS are shown in Table II. Significant correlation were noted between \( \Sigma ST \) recorded at 24, 48 and 72 hours as well as 4 weeks after infarction and \%ACS evaluated after 4 weeks following infarction (Table II). Correlation between \( \Sigma ST \) recorded at 48 hours and NQ recorded at 4 weeks, and between \( \Sigma ST \) and \%ACS by left ventricular angiography are shown in Figs. 5 and 6, respectively.

**DISCUSSION**

Our results indicate that reelevation of ST segment was recognized in all patients with acute anterior myocardial infarction without reeeleva-

\[ r = 0.757 \]
\[ P \leq 0.01 \]
\[ n = 13 \]

Fig. 5. Relationship between \( \Sigma ST \) recorded at 48 hours after the onset of myocardial infarction (\( \Sigma ST \) (48 h) and NQ recorded at 4 weeks after infarction (NQ (4 w))). Significant correlation was noted between these values of precordial mapping.

\[ r = 0.813 \]
\[ P \leq 0.02 \]
\[ n = 8 \]

Fig. 6. Comparison between \( \Sigma ST \) (48 h) and \%ACS estimated by left ventricular cineangiogram in 8 patients with anterior myocardial infarction. There was significant correlation between these values.

The recent investigations on ST segment mapping in acute myocardial infarction have been mainly focused on following two aspects: (1) The evaluation of the infarct size by \( \Sigma ST \). (2) The natural time course of \( \Sigma ST \) and its alteration by therapeutic interventions. Experimentally, epicardial ST segment elevation caused by coronary artery occlusion was reported first in 1949. The magnitude of ST elevation recorded from the epicardial leads at 15 minutes after coronary occlusion highly correlated with the reduction of CK activities in the heart muscle examined 24 hours later and similar results were obtained in the histological studies. It is shown that there is a good correlation between epicardial and precordial \( \Sigma ST \). Clinical and experimental studies have shown that various factors, such as O2 inhalation, hyaluronidase, nitroglycerin, nitroprusside, \( \beta \)-blocking agent calcium antagonist and intraaortic balloon pumping may alter the ST segment in acute myocardial
infarction.

However, it is not conclusive whether $\Sigma ST$ of precordial mapping in patients with acute myocardial infarction correlates with indices of the infarct size. $\Sigma ST$ of the precordial mapping correlated with the infarct size estimated by serum CK activities in some reports$^{20,21}$ but not in others$^{5,6}$. Multiple factors can influence ST segments. The magnitude of ST elevation is determined not only by the infarct size but also by electrolyte, various drugs and the distance between electrodes and the myocardium$^{22}$. Therefore, the improvement of ST elevation by therapeutic interventions may not always reflect the reduction of infarct size. As correlation between $\Sigma ST$ and infarct size is examined, the time of recording of $\Sigma ST$ in respect to the onset of myocardial infarction should be considered. The present study indicated that the magnitude of ST elevation changed in time following myocardial infarction and showed reerelevation. The causes of ST elevation may not be same at the different time following the myocardial infarction.

The time course after myocardial infarction may be divided into three phases as regard to ST elevation. In the first phase, nearly 15 minutes after the onset, $\Sigma ST$ correlated with the infarct size in experimental studies$^3$. But, clinically, the recording of precordial mapping at such extremely early phase after the onset of chest pain may be possible only in rare cases. The second phase is when reerelevation of ST segment occurs. Previous studies reported ST reerelevation in the time course of acute myocardial infarction$^{23,24}$ and $\Sigma ST$ recorded in this phase correlated with infarct size. Inoue et al. reported the biphasic ST changes during 6 to 12 hours after infarction in clinical study. They found a significant correlation between infarct size estimated by serum CK activity and $\Sigma ST$ recorded at 48 hours after the onset of infarction but not at 12 hours$^{21}$. We obtained similar results in correlation between $\Sigma ST$ and NQ, and between $\Sigma ST$ and $\%ACS$ of left ventricular angiography. In other studies, the cases which were complicated with extension of infarction or pericarditis were found to show greater reerelevation of $\Sigma ST$ as compared to $\Sigma ST$ observed in patients without such complications.$^{23,24}$ Various therapeutic interventions is often given at this second period. The interpretation of change of $\Sigma ST$ by therapeutic interventions should be carefully done in light of naturally changing ST elevation.

The third phase is later than 2 weeks after infarction. The persistent ST elevation after 2 weeks following infarction has been considered as the findings of ventricular aneurysm$^{25,26}$. Our result showed the good correlation between $\Sigma ST$ and NQ recorded at 4 weeks after the onset of infarction or between $\Sigma ST$ and $\%ACS$ of left ventricular angiography.

Generally, it has been believed that the reerelevation of ST segment in time course of acute myocardial infarction is caused by the reattack of infarction, coronary spasm, pericarditis, ventricular aneurysm, enlargement of left ventricle or various states increasing the work of left ventricle. The cause and the clinical significance of ST segment reerelevation in this study was not clear. The ST reerelevation in our cases cannot be explained by pericarditis or extension of myocardial infarction.

REFERENCES


7. AHA Committee Report: A reporting system on patients evaluated for coronary artery disease. Circulation 51: No. 4, 1975


10. ROSALKI SB: An improved procedure for serum


17. GOLD HK, LEINBACH RC, MAROKO PR: Propranolol induced reduction of signs of ischemic injury during acute myocardial infarction. *Am J Cardiol* 38: 689. 1976


*Japanese Circulation Journal Vol. 45, May 1981*