Evaluation of praecordial ST segment mapping as an index of infarct size in patients with acute myocardial infarction

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SUMMARY We evaluated the usefulness and limitations of praecordial ST segment mapping as a clinical means of assessing the size of acute myocardial infarction in 14 patients with anterior myocardial infarction and 13 patients with inferior myocardial infarction. \( \Sigma ST \), the sum of ST segment elevations, and \( nST \), the number of leads showing ST segment elevation, were obtained from serial electrocardiograms recorded through 39 praecordial leads. The infarct size and period of the evolution of myocardial infarction were estimated respectively from the total creatine kinase (CK) released and the serial changes of the CK releasing rate. \( \Sigma ST \) and \( nST \) obtained at the time when the CK release had ceased correlated closely with the total CK released. Peak \( \Sigma ST \) and \( nST \), and values 48 hours after the onset of myocardial infarction, also correlated well with the total CK released; but those on admission or 12 hours after the onset correlated poorly.

These results suggest that \( \Sigma ST \) and \( nST \) at the end of evolution of myocardial infarction or 48 hours after the onset may be two useful indices for the assessment of infarct size in patients with either anterior or inferior myocardial infarction.

Many attempts (Maroko et al., 1972; Norris et al., 1975; Henning et al., 1978; Sharpe et al., 1978) have been made to assess infarct size in patients with acute myocardial infarction, because the extent of necrosis directly influences cardiac function and subsequent prognosis (Page et al., 1971; Sobel et al., 1972; Mathey et al., 1974; Hori et al., 1979). Maroko et al. (1971) reported a close correlation between the magnitude of epicardial ST segment elevation 15 minutes after experimental coronary occlusion in dogs, and infarct size as determined by loss of myocardial creatine kinase (CK) activity or as determined histologically 24 hours later. Their further study (Muller et al., 1975) also showed that in dogs the magnitude of epicardial ST segment elevation correlated well with that of praecordial ST segment elevation. Though it is now widely recognised that surface mapping of the electrocardiogram is useful in evaluating the effect of therapeutic interventions on the extent of infarction, the usefulness of this technique in measuring human infarct size remains controversial (Reese et al., 1973; Norris et al., 1976; Thompson and Katavitis, 1976) perhaps because serial changes of ST segment elevation after the onset of infarction have been studied inadequately. In this study we attempted to investigate the serial changes in the praecordial ST segments to evaluate the feasibility of the surface mapping technique as an index of infarct size both in patients with anterior infarction and with inferior infarction.

Subjects and methods

Of 34 consecutive patients with acute myocardial infarction who were admitted to the Coronary Care Unit of Sakurabashi Watanabe Hospital within 12 hours after the onset of infarction, 27 were studied. We excluded two patients with previous myocardial infarction, three patients whose infarct size could not be assessed because of early death, and two patients with intraventricular conduction delay. None of the 27 patients in our series had pericarditis or a cerebrovascular accident which might influence

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ST segment changes. Fourteen patients had anterior infarction (13 men and one woman, ages 33 to 77, with a mean of 53 years). This group included one patient with lateral infarction and another with dominantly subendocardial infarction and only partial transmural anterior infarction; these two were excluded from the statistical analysis. Thirty patients had inferior infarction (eight men and five women, ages 44 to 82 years, with a mean of 66 years). The diagnosis was made by a typical history, characteristic alterations of ST-T wave changes on the standard 12 lead electrocardiograms, and diagnostic rise and fall in serum enzymes (creatine kinase, α-hydroxybutyric dehydrogenase, aspartate aminotransferase, and lactic dehydrogenase).

(1) PREAERCIAL ST SEGMENT MAPPING TECHNIQUE

Electrocardiograms were recorded through unipolar leads ('V' lead) from 39 points, which were marked with an indelible pen so that all subsequent recordings were obtained from the same points. The 39 points comprised 21 points on the anterior chest and 18 points on the left lateral chest as shown in Fig. 1.

Electrocardiograms were recorded, using a thermal pen recorder on standard electrocardiographic paper running at 25 mm/s with standardisation of 1 mV/cm, on admission and 12, 24, 36, 48, 72 hours, and seven and 14 days after the onset of chest pain. Whenever possible, electrocardiograms were also obtained three and six hours after the onset of pain. The magnitude of ST segment elevation of recorded electrocardiograms was measured at the J point to the nearest 0.5 mm.

Fig. 1 The positions of 39 praecordial leads. Twenty-one points on the anterior chest were at the intersections of three imaginary vertical lines (1: right sternal border; 2: left sternal border; 3: left mid-clavicular line) and seven horizontal lines (A–E: second to sixth intercostal space near the sternum; F: 2.5 cm below E; G: 2.5 cm below F). Eighteen points on the left lateral chest were at the intersections of three vertical lines (4: anterior axillary line; 5: mid-axillary line; 6: posterior axillary line) and six horizontal lines (B to G as above).

The sum of ST segment elevations from all leads (ΣST) and the number of leads showing ST segment elevation of 0.5 mm or greater (nST) were calculated from each recording.

In order to evaluate the reproducibility of measured ST segment elevation, the praecordial ST segment mapping was recorded again one hour after we recorded the three-hour or the 48-hour electrocardiograms in 10 patients. Two cardiologists independently measured ST segment deviation in each electrocardiogram recorded from five patients to assess the variation among observers.

(2) ASSESSMENT OF INFARCT SIZE BY TOTAL CK RELEASED (ΣCK)

Venous blood samples for CK determination were obtained at four-hour intervals from the time of admission until serum enzyme levels returned to normal. The appearance function of CK released from the infarcted myocardium was calculated by the method of Sobel et al. (1972) as modified by Norris et al. (1975) and used as a guide in assessing the time course of evolution of the infarct (Inoue et al., 1977). Total CK released (ΣCK) was measured by integrating this appearance function and was used as an index of infarct size. Total CK released up to 24 and 48 hours after the onset, ΣCK_{24} or ΣCK_{48}, respectively, were also measured in order to compare the change in ΣST from 24 to 48 hours. Though the evolution of the infarct was considered to cease at the time when the appearance function of CK released became zero IU/ml per h (Inoue et al., 1977), the exact determination of this time was difficult because it could be influenced substantially by subtle alterations in the disappearance rate of serum CK activity. Therefore, in this study the time when the appearance function decreased to 5 IU/ml per h was regarded as the end point of evolution; this time is much less influenced by the alteration in the disappearance rate of serum CK activity. In practice, ΣST and nST at the end of evolution were obtained by linear interpolation of the two values which were measured at the closest points immediately before and after the time when the appearance function became 5 IU/ml per h or less.

Results

(A) EVALUATION OF ST SEGMENT MAPPING AS AN INDEX OF INFARCT SIZE

(1) Serial changes in ΣST and nST

ΣST and nST were obtained on admission and 12, 24, 36, 48, and 72 hours, and on the seventh and 14th day after the onset in all patients studied. ΣST and nST were also obtained three and six
hours after the onset in six and 17 patients, respectively. Fig. 2 and 3 show the serial changes in $\Sigma$ST and nST in patients with anterior and inferior infarction. The data three hours after the onset in patients with inferior infarction are not depicted in Fig. 3 since they were obtained in only two patients. Serial changes in $\Sigma$ST, nST, and the appearance function of CK in two representative cases are shown on the same time scale in Fig. 4 and 5.

In one case with anterior infarction shown in Fig. 4, $\Sigma$ST and nST increased to their peaks 48 hours and 24 hours after the onset, respectively, and then decreased gradually. In contrast, another case with anterior infarction showed a typical biphasic pattern of $\Sigma$ST and nST as shown in Fig. 5; both indices showed a transient decrease between six and 12 hours after the onset of infarction and then increased again without any important changes in blood pressure, heart rate, or symptoms. Such a biphasic pattern was observed in six out of 12 patients (50%) with anterior infarction and eight out of 13 patients (62%) with inferior infarction. It was of great interest, however, that in almost all patients both $\Sigma$ST and nST reached a somewhat stable state at the end of evolution of infarction judged from the appearance function of CK released (see Methods) and showed a gradual decrease thereafter, as shown in Fig. 4 and 5.

(2) $\Sigma$ST and nST at end of evolution of infarction

The mean values of $\Sigma$ST and nST at the end of
evaluation of infarction (henceforth designated as $\Sigma ST_{end}$ and $nST_{end}$, respectively) and the duration of evolution (the duration of CK release) are shown in Table 1. It was seen that $\Sigma ST_{end}$ and $nST_{end}$ correlated closely with the total CK released, which represents the infarct size in 12 patients with anterior infarction ($\Sigma ST$: $r=0.82$, $P<0.01$; $nST$: $r=0.85$, $P<0.01$, see Fig. 6). Values from two patients who were excluded from the statistical analysis (one with lateral infarction and another with subendocardial infarction and only partial transmural anterior infarction) were also plotted in Fig. 6; they were far below the regression line, indicating that these $\Sigma ST$ and $nST$ were disproportionately too small in comparison with the infarct size estimated enzymatically. In 13 patients with inferior infarction there was also a significant correlation between $\Sigma ST_{end}$ and $nST_{end}$ and infarct size ($\Sigma CK$) ($\Sigma ST_{end}$: $r=0.84$, $P<0.01$; $nST_{end}$: $r=0.72$, $P<0.01$, see Fig. 7). These results strongly suggest that $\Sigma ST$ and $nST$ at the end of the evolution of infarction directly reflect infarct size in both anterior infarction and inferior infarction, but not for lateral or subendocardial infarction.

(3) $\Sigma ST$ and $nST$ 48 hours after onset of infarction
Since in 80 per cent of all patients the evolution of infarction had ceased within 48 hours after the onset of chest pain, the relation between $\Sigma ST$ and $nST$ 48 hours after the onset (designated as $\Sigma ST_{48}$ and $nST_{48}$, respectively) and infarct size ($\Sigma CK$)

Table 1  Mean values of $\Sigma ST$ and $nST$ at end of evolution of infarct and duration of evolution in patients with anterior and inferior infarction

<table>
<thead>
<tr>
<th>Number of cases</th>
<th>$\Sigma ST_{end}$ (SEM) (mm)</th>
<th>$nST_{end}$ (SEM)</th>
<th>Duration of evolution (SEM) (h)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Anterior myocardial infarction</td>
<td>$n=14$</td>
<td>$35.3 \pm 7.3$</td>
<td>$21.5 \pm 2.2$</td>
</tr>
<tr>
<td>Inferior myocardial infarction</td>
<td>$n=13$</td>
<td>$10.2 \pm 2.0$</td>
<td>$11.0 \pm 1.6$</td>
</tr>
</tbody>
</table>

Fig. 6  Relations between $\Sigma ST$ (upper panel), $nST$ (lower panel), and the total CK released ($\Sigma CK$) at the end of evolution of anterior infarction. □: a case with lateral infarction; ○: a case with subendocardial infarction and partial transmural anterior infarction.

Fig. 7  Relations between $\Sigma ST$ (upper panel), $nST$ (lower panel), and the total CK released ($\Sigma CK$) at the end of evolution of inferior infarction. □: a case with inferoseptal infarction; △: case with inferoposterior infarction.
was also investigated. We found that $\Sigma ST_{48}$ and $nST_{48}$ also correlated significantly with $\Sigma CK$ in both anterior infarction and inferior infarction; $r=0.72$ ($P<0.01$) for $\Sigma ST_{48}$ and $r=0.77$ ($P<0.01$) for $nST_{48}$ in anterior infarction and $r=0.86$ ($P<0.01$) for $\Sigma ST_{48}$ and $r=0.66$ ($P<0.01$) for $nST_{48}$ in inferior infarction (see Table 2).

Table 2 Correlations between $\Sigma ST$ and/or $nST$ and total CK released ($\Sigma CK$) at various time intervals after onset of infarction

<table>
<thead>
<tr>
<th>Time Interval</th>
<th>$\Sigma ST$</th>
<th>$nST$</th>
<th>$\Sigma ST$</th>
<th>$nST$</th>
</tr>
</thead>
<tbody>
<tr>
<td>On admission</td>
<td>$\Sigma ST$</td>
<td>$nST$</td>
<td>$\Sigma ST$</td>
<td>$nST$</td>
</tr>
<tr>
<td>$\Sigma ST_{\text{initial}}$</td>
<td>$nST_{\text{initial}}$</td>
<td>0.43</td>
<td>0.67*</td>
<td>0.63*</td>
</tr>
<tr>
<td>At 12 h after onset</td>
<td>$\Sigma ST_{12}$, $nST_{12}$</td>
<td>0.58</td>
<td>0.58</td>
<td>0.74†</td>
</tr>
<tr>
<td>At 48 h after onset</td>
<td>$\Sigma ST_{48}$, $nST_{48}$</td>
<td>0.72‡</td>
<td>0.77‡</td>
<td>0.86†</td>
</tr>
<tr>
<td>At peak time</td>
<td>$\Sigma ST_{\text{max}}$, $nST_{\text{max}}$</td>
<td>0.76†</td>
<td>0.86†</td>
<td>0.79†</td>
</tr>
<tr>
<td>At end of evolution</td>
<td>$\Sigma ST_{\text{end}}$, $nST_{\text{end}}$</td>
<td>0.82‡</td>
<td>0.85†</td>
<td>0.84‡</td>
</tr>
</tbody>
</table>

* $P<0.05$. † $P<0.01$.

(4) $\Sigma ST$ and $nST$ at their peaks

In our series maximal $\Sigma ST$ and $nST$ were obtained at $38.3 \pm 7.0$ (SEM) hours and $33.3 \pm 5.8$ (SEM) hours, respectively, in patients with anterior infarction, and $31.9 \pm 6.3$ (SEM) hours and $25.9 \pm 4.3$ (SEM) hours, respectively, in patients with inferior infarction. The maximal $\Sigma ST$ and $nST$ (henceforth designated as $\Sigma ST_{\text{max}}$ and $nST_{\text{max}}$) also correlated significantly with the infarct size ($\Sigma CK$): $r=0.76$ ($P<0.01$) for $\Sigma ST_{\text{max}}$ and $r=0.86$ ($P<0.01$) for $nST_{\text{max}}$ in anterior infarction, and $r=0.79$ ($P<0.01$) for $\Sigma ST_{\text{max}}$ and $r=0.68$ ($P<0.01$) for $nST_{\text{max}}$ in inferior infarction.

(5) $\Sigma ST$ and $nST$ on admission and 12 hours after onset of infarction

$\Sigma ST$ and $nST$ on admission ($\Sigma ST_{\text{initial}}$ and $nST_{\text{initial}}$) and 12 hours after the onset ($\Sigma ST_{12}$ and $nST_{12}$) were found to be poorly correlated with total CK released (especially $\Sigma ST_{\text{initial}}$) in anterior infarction ($r=0.43$, $P>0.05$) and $nST_{\text{initial}}$ in inferior infarction ($r=0.53$, $P>0.05$, see Table 2). These results strongly suggest that $\Sigma ST$ and $nST$ during the early phase of the progression of infarction do not directly reflect the extent and severity of tissue necrosis of the myocardium.

(6) Detection of extension of infarction by serial change of $\Sigma ST$ or $nST$

To investigate whether the serial change of $\Sigma ST$ or $nST$ can directly reflect the instantaneous evolution of infarct, the change of $\Sigma ST$ or $nST$ between 24 and 48 hours after the onset was compared with the amount of increase in CK released during this interval ($\Sigma CK_{48}$ to $\Sigma CK_{24}$). In five of 11 patients (45.5%) with anterior infarction and nine of 13 patients (69%) with inferior infarction, $\Sigma ST$ increased with the increase in $\Sigma CK$, shown by arrows in the right upper part of Fig. 8. One patient with anterior infarction was excluded because the electrocardiogram recorded 24 hours after the onset showed transient right bundle-branch block. In this figure, however, some arrows point downwards, indicating that $\Sigma ST$ decreased from 24 to 48 hours after the onset of infarction. This suggests that $\Sigma ST$ decreases in some patients despite the evolution of infarct. This was also true of $nST$.

In summary, our results indicate that the infarct size in patients both with anterior infarction and with inferior infarction may be estimated by $\Sigma ST$ and $nST$ obtained from the precardial electrocardiographic mapping at the time when the
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evolution of infarct has ceased ($\Sigma ST_{\text{end}}$ and $nST_{\text{end}}$) or 48 hours after the onset, when the evolution is considered to have been completed in most of the cases ($\Sigma ST_{48}$ and $nST_{48}$). $\Sigma ST$ and $nST$ at these times after the onset closely correlated with infarct size ($\SigmaCK$), probably because ST segment elevation becomes stable after evolution of the infarct has ceased, and the ischaemic injury zone would be proportional to the extent of tissue necrosis at this stage of the disease. In contrast, however, at the early stage during the evolution of the infarct, præcordial ST segment elevation does not reflect the infarct size. Thus, $\Sigma ST$ and $nST$ 12 and 24 hours after the onset correlated poorly with $\Sigma CK$, probably because the surrounding ischaemic zone is labile and greatly influenced by small alterations in coronary blood supply and oxygen demand during evolution of the infarct.

(B) Reproducibility of ST segment mapping technique

The variation in measurement of ST segment displacement was $7.6 \pm 8.3$ per cent (mean $\pm$ SD) in 25 recordings obtained from five patients, indicating that errors from this source are small. Though the hour-to-hour variation about three hours after the onset was $26.7 \pm 8.9$ per cent (mean $\pm$ SD) in five patients, the variation about 48 hours after onset was $3.5 \pm 2.6$ per cent (mean $\pm$ SD) in five patients.

These results suggest that $\Sigma ST$ obtained about 48 hours after the onset is stable enough to be meaningful.

Discussion

In this study we evaluated the validity of præcordial ST segment mapping and also its limitation as an index of infarct size in patients both with anterior infarction and with inferior infarction. The sum of the magnitude of ST segment elevations ($\Sigma ST$) and the total number of leads showing ST segment elevation ($nST$) during the early phase of infarction, within 12 hours after the onset, did not accurately reflect the extent of infarction. However, $\Sigma ST$ and/or $nST$ measured at the time when the evolution of infarct had ceased or the maximal $\Sigma ST$ and/or $nST$ did closely correlate with infarct size. This method may be useful in patients with inferior as well as anterior infarction.

Since Maroko et al. (1971) established epicardial ST segment mapping as a method for the assessment of infarct size, it has been predicted that præcordial ST segment mapping would also be useful clinically to evaluate the effect of therapeutic interventions during the course of acute myocardial infarction and also to detect the extension of infarction. However, the validity of quantification of præcordial ST segment elevation for the assessment of infarct size has remained controversial. Norris et al. (1976) reported that there was no significant correlation between the peak value of $\Sigma ST$ and the infarct size estimated by the same method in our study. Thompson and Katavitis (1976) also showed the clinical limitation of this method; only a rough correlation ($r=0.48$, $n=12$) was found between ST segment elevation and infarct size ($\Sigma CK$), and in some cases the extension of infarction could not be detected with this technique.

In evaluating the quantification of ST segment elevation, however, the factors influencing the deviation of the ST segment on the præcordial leads must be taken into account. Though the origin of ST segment elevation, that is ischaemic injury current, is believed to be derived from the potential gradient across the boundary of ischaemia resulting from the alteration in action potential, electrical potential recorded on the chest surface can be influenced by other factors, for example the solid angle of the ischaemic zone subtended at a point of the electrode, and electrical conductivity of the tissues between the ischaemic zone and the electrode on the chest surface. Indeed, these factors which greatly influence underlying ischaemic potential gradient are of great importance in the interpretation of ST segment deviations in surface electrocardiograms, because the extent of infarction as well as the site of infarction directly influence the solid angle of the ischaemic area subtended at a point of the electrode. Even if individual variations in the potential gradient across the boundary of the ischaemic area and electrical conductivity of the thorax were both negligibly small, the difference in infarct site would greatly distort the distribution of ST segment elevation. In fact, in our study the regression lines indicating a significant correlation between $\Sigma ST$ or $nST$ and infarct size ($\Sigma CK$) in anterior infarction were quite different from those in inferior infarction. Moreover, in two patients who were excluded from the statistical analysis, one with lateral infarction and another with subendocardial infarction and only partial transmural anterior infarction, both $\Sigma ST$ and $nST$ deviated downward from the regression line (see Fig. 6), indicating that in these cases the solid angle, which is proportional to ST segment elevation, was disproportionately small for the infarct size.

Similarly, infarct site can influence ST segment elevation in patients with inferior infarction. Indeed, there were two patients with inferior infarction like those with anterior infarction whose $\Sigma ST$ and $nST$ substantially deviated from the
regression line (see Fig. 7). Of these patients, one had an infarct extending towards the septal wall showing that \( \Sigma ST \) and nST deviate upward from the regression line (Fig. 7). The other had an infarct extending toward the posterior wall, with downward deviation of \( \Sigma ST \) and nST from the regression line (Fig. 7). In the former, the ST segment elevation caused by septal infarction might be superimposed on that of inferior infarction; while, in the latter, the reciprocal ST segment depression caused by posterior infarction may offset the underlying ST segment elevation. These results strongly suggest that both infarct site and infarct size are major factors which should be taken into account when we evaluate the ST segment elevation. In this study, apart from the patients with infarction extending toward other regions than the anterior or inferior wall and also in subendocardial infarction, the \( \Sigma ST \) and nST correlated closely with the infarct size in patients both with anterior and with inferior infarction.

The present study showed that the time course of ST segment change was of great importance in assessing infarct size. Since the variation of measurement was so small as to be neglected (observation variation: \( 7.6 \pm 8.3\% \) and hour-to-hour variation 48 hours after the onset: \( 3.5 \pm 2.6\% \)), changes in ST segment elevation should reflect the change in extent and/or severity of ischaemia even though changes in electrical conductivity of the thorax such as that resulting from pulmonary oedema would also contribute to ST segment change to some extent. However, the extent of the ischaemic area may not be proportional to that of tissue necrosis, especially during the acute phase of infarction. Cox et al. (1968) showed that in experimental dogs the size of myocardial ischaemia reached its maximum 18 hours after ligation of a coronary artery, while that of myocardial necrosis reached its maximum several hours later than that of ischaemia. These findings agree with our present observation: ST segment elevation which should represent the extent and/or severity of ischaemia became maximal on average 35-2 hours after the onset of chest pain, while the duration of CK release, which represents the evolution of tissue necrosis, was on average about 40 hours.

The discrepancy of evolution between ischaemia and necrosis may be substantially large in the early phase of infarction because in this phase the regional oxygen balance is easily influenced by haemodynamic changes. Indeed, in more than 50 per cent of patients, \( \Sigma ST \) and nST decreased a day after the onset while CK release continued. This discrepancy between the extent of ischaemia and necrosis size would account for poor correlation between \( \Sigma ST \) or nST and infarct size (\( \Sigma CK \)) on admission and 12 hours after the onset of infarct. In contrast, ST segment elevation around the end of the evolution of infarct correlated highly with infarct size (Fig. 6 and 7). This finding indicates that at this stage the surrounding ischaemic area has already been established in proportion to infarct area.

Morris et al. (1974) also reported that the degree of ST segment displacement at 48 hours of infarction was related to the mean maximum serum AST (GOT) activity. This report agrees with our results. Poor or absent correlation between \( \Sigma ST \) and infarct size (\( \Sigma CK \)) observed by Norris et al. (1976), and Thompson and Katavitis (1976) might have resulted because they measured \( \Sigma ST \) long before the end of the evolution of infarction. The variation in infarct size in their studies might also account for poor correlation.

Although \( \Sigma ST \) or nST at the end of the progression of the infarct or the maximal \( \Sigma ST \) or nST can represent infarct size, the determination of these indices is difficult in patients who are not admitted immediately after the onset, and when enzymatic assessment of infarct size is not available. In such cases, praecordial ST segment mapping 48 hours after the onset could be of value in assessing infarct size.

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