In 38 patients with acute anterior myocardial infarction or ischaemia, the extent and amplitude of ST segment elevation was measured on the chest wall with a praecordial mapping technique. There was a poor correlation with measurements of clinical severity and with the extent of necrosis as measured by the peak levels of creatine kinase (CK) and the estimated total amount of CK released. In two patients ST segment re-elevation occurred without re-elevation of serum CK and in two other patients reinfarction obvious on enzyme re-elevation occurred without changes in ST segments. Praecordial ST segment mapping appears to have a limited role in measuring or monitoring human infarct size and would be an unreliable tool for evaluation of methods to limit myocardial necrosis. The chest leads of the standard 12-lead electrocardiogram provided sufficient information for clinical evaluation of ST segment elevation.

It has been shown that the extent of myocardial necrosis after experimental coronary occlusion can be limited by appropriate interventions (Maroko et al., 1971; Braunwald and Maroko, 1974). Clinical application of these findings is possible only if accurate methods of measuring and following trends in infarct size in patients can be developed. Mapping of the ST segments of the electrocardiogram in the praecordium has been proposed (Maroko et al., 1972; Reid et al., 1974) for this purpose. Despite adequate documentation of the validity of ST segment elevation as an index of infarct size in dogs (Maroko et al., 1971), it is not known how accurately ST segment changes in the praecordial electrocardiogram reflect changes in human myocardial infarction. Theoretical considerations (Boineau, 1971) and preliminary clinical reports (Reese, Scheidt, and Killip, 1973) suggest that use of the technique in measuring human infarct size requires critical evaluation.

The present study was undertaken to evaluate the technique and compare it with clinical and enzymatic estimates of infarct size.

Patients and methods
Studies were made on 38 patients with suspected acute anterior or anterolateral myocardial infarction. Altogether 313 praecordial ST segment maps were recorded. The diagnosis was based on the clinical features and the results of routine daily serum enzymes and electrocardiograms. On WHO Criteria (World Health Organization, 1969) there were 35 patients with 'definite' and 3 patients with 'possible' myocardial infarction. In 17 patients praecordial maps were performed soon after admission to hospital, each morning for 10 consecutive days, and on the day of discharge from hospital. The day of admission to hospital is referred to as day 1.

Technique of praecordial ST segment mapping
All recordings were made with the patient in the supine position. The chest wall of male patients was shaved. Forty-two positions were marked on the anterior chest wall; indelible felt pencil was used and markings were retouched daily to ensure accurate placement of the recording electrode. A Philips class B electrocardiograph was used. The amplitude frequency response (without muscle filter) was linear from 0-3–150 ±10 per cent Hz with maximum 10 per cent at 150 Hz, time constant 2 seconds. All recordings were done on standard electrocardiograph paper running at 25 mm/s with standardization of 1 cm/mV. Approximately 15 minutes were required for recording each 42 lead electrocardiogram.
The maximum height of the ST segment was measured to the nearest 0.5 mm from the isoelectric line (TP segment). When the ST segment was upward sloping the maximum height was taken at a point immediately preceding the origin of the T wave (determined in a lead where the T wave was clearly demarcated). This point varied from 0.6 to 0.12 s from the R wave. The recordings and measurements were made throughout by the same observer (VK). The amplitude of the ST segments measured in each recording position were summed and expressed as the sum ST (ΣST). The number of cells showing ST segment elevation of >0.5 mm was expressed as NST and an ST segment map was constructed. On patients who had consecutive daily recordings the mean of the ΣST (ΣST) and the mean of the NST (NST) was calculated by dividing by the number of days on which recordings were made. For comparison with 12 lead tracings, standard electrocardiograph records were taken on the same machine and ST segment measurements performed by the same observer.

Serum enzyme estimations
Creatine kinase was estimated (a) each morning for 5 to 6 days and (b) every 2 hours from admission until the serum enzyme levels returned towards normal. Blood samples were taken from an indwelling catheter in a central vein, stored at 4°C, and separated within several hours of sampling. The enzyme was measured by a standard autoanalyser technique (Siegel and Cohen, 1967).

Estimation of cumulated CK release
From the results of the 2-hourly CK estimations, the total amount of CK released was calculated by a modification of the formula of Shell, Kjekshus, and Sobel (1971). This technique has been previously shown in experimental animals to reflect the total amount of CK released into the serum.

Results
The ΣST on day 1 was 102 ± 63.5 mm and progressively declined to 62 ± 27.4 mm by day 10 (Fig. 1). There was a wide variation between patients: the ΣST on day 1 ranged from 23.5 to 280.5 mm. The standard deviations from the mean ΣST was >50 per cent for each of days 1 to 10. The trends in ΣST varied, even for patients with approximately similar clinical and serum enzyme abnormalities (Fig. 2). Similar trends were observed in NST.

Comparison with clinical estimates of severity
The ΣST on day 1 or the ΣST did not correlate with clinical grades of severity of infarction as
TABLE 1  
Comparison of \( \Sigma ST \) day 1 and \( \Sigma ST \) with various subgroups: there were no significant differences in ST segment elevation between any subgroups

<table>
<thead>
<tr>
<th>Subgroup</th>
<th>( \Sigma ST ) day 1 Mean ( \pm SD )</th>
<th>( \Sigma ST ) Mean ( \pm SD )</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age &gt; 60</td>
<td>6 66 ±54</td>
<td>7 82 ±43</td>
</tr>
<tr>
<td>&lt; 60</td>
<td>9 117 ±64</td>
<td>10 97 ±40</td>
</tr>
<tr>
<td>Previous</td>
<td>Yes 5 79 ±42</td>
<td>6 86 ±41</td>
</tr>
<tr>
<td>infarction</td>
<td>No 10 135 ±79</td>
<td>11 86 ±43</td>
</tr>
<tr>
<td>Previous</td>
<td>Yes 5 87 ±43</td>
<td>6 69 ±27</td>
</tr>
<tr>
<td>angina</td>
<td>No 10 107 ±74</td>
<td>11 102 ±45</td>
</tr>
<tr>
<td>Pulmonary</td>
<td>Yes 7 129 ±76</td>
<td>8 95 ±44</td>
</tr>
<tr>
<td>vascular</td>
<td>No 8 78 ±41</td>
<td>9 85 ±46</td>
</tr>
</tbody>
</table>

Estimated by the Peel index or the Norris index (Fig. 3).

The \( \Sigma ST \) on day 1 failed to predict haemodynamic or arrhythmical complications during the subsequent hospital course (Table 1), or physical capacity (NYHA classification) among the survivors at 6 weeks after infarction.

Comparison with peak and cumulated serum CK

\( \Sigma ST \) on day 1 correlated approximately with the peak of the 2-hourly and the routine daily serum enzyme estimations (r=0.69, 0.68, respectively), as did \( \Sigma ST \) (r=0.61, 0.52, respectively) (see Table 2). These correlations were significant (P<0.05) but there was a wide individual scatter (Fig. 4). The cumulated CK did not correlate with \( \Sigma ST \) on day 1 or \( \Sigma ST \). The area of ST segment elevation (NST) did not correlate with any of the serum enzyme parameters.

Trends in \( \Sigma ST \) compared with trends in serum CK

Daily praeordial ST segment maps were compared with CK estimations in 38 patients. In 3 patients re-elevation of \( \Sigma ST \) occurred; on one occasion there was an associated re-elevation in serum enzyme levels indicating reinfarction, but in 2 patients no change in serum enzymes resulted (Fig. 5).

Conversely, changes in serum enzyme levels measured every 2 hours indicated significant reinfarction in 2 patients, but the praeordial ST segment map failed to show any change in the \( \Sigma ST \) or NST. A striking example is seen in Fig. 6.

Comparison of 42 lead and 12 lead electrocardiogram

\( \Sigma ST \) calculated from 42 praeordial leads correlated well (r=0.90) with \( \Sigma ST \) calculated from the 6 praeordial leads (V1–V6) of a standard electrocardiogram.

TABLE 2  Coefficient of correlation between ST segment elevation parameters and indices of clinical severity and serum enzyme estimations (NS=not significant)

<table>
<thead>
<tr>
<th>Parameter</th>
<th>( \Sigma ST ) day 1 (n=15)</th>
<th>( \Sigma ST ) (n=17)</th>
<th>NST day 1 (n=15)</th>
<th>NST (n=17)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Norris index</td>
<td>0.01 (NS)</td>
<td>0.22 (NS)</td>
<td>-0.18 (NS)</td>
<td>-0.52 (NS)</td>
</tr>
<tr>
<td>Peel index</td>
<td>0.41 (NS)</td>
<td>-0.01 (NS)</td>
<td>0.05 (NS)</td>
<td>-0.18 (NS)</td>
</tr>
<tr>
<td>Cumulated CK</td>
<td>0.49 (NS)</td>
<td>0.41 (NS)</td>
<td>0.357 (NS)</td>
<td>0.08 (NS)</td>
</tr>
<tr>
<td>Peak 2-hour CK</td>
<td>0.69 (P&lt;0.05)</td>
<td>0.61 (NS)</td>
<td>0.43 (NS)</td>
<td>0.304 (NS)</td>
</tr>
<tr>
<td>Peak daily CK</td>
<td>0.68 (P&lt;0.01)</td>
<td>0.52 (NS)</td>
<td>0.34 (NS)</td>
<td>0.14 (NS)</td>
</tr>
</tbody>
</table>
failed two the no pain a peak and the
lateral myocardial necrosis; and no
ST segment changes; subsequent serum enzyme estimations was not detected in the daily ST segment maps performed in this study. Though it is conceivable that transient ST segment elevation may have occurred between praecordial maps performed each morning, our findings (Fig. 1) and those of others (Madias, Venkataraman, and Hood, 1975; Mills et al., 1975) indicate that when ST segment elevation does accompany significant myocardial necrosis, it is sufficiently persistent to be detected within 24 hours after the event. Our findings showed an unacceptable disparity between ST segment elevation and serum levels of enzymes known to reflect myocardial necrosis. While serum creatine kinase elevation has been shown to reflect myocardial necrosis in patients dying of myocardial infarction (Mathey et al., 1975), no such evidence validating ST segment elevation as an index of human myocardial necrosis has been presented.

The extensive experimental work which has shown the relation of ST segment elevation to myocardial necrosis (Maroko et al., 1971, 1972) has been performed in dogs with normal myocardium and localized coronary artery obstruction. When applied to patients with atherosclerotic heart disease who have suffered acute myocardial infarction it appears to measure phenomena that do not necessarily reflect myocardial necrosis. It is evident in the light of current attempts to limit infarct size (Braunwald and Maroko, 1974) that interventions cannot be evaluated solely by changes in praecordial ST segment mapping and will need to be combined with other methods which provide a more accurate measurement of myocardial necrosis (Sobel et al., 1972; Norris et al., 1975).

The possible prognostic significance of persistent ST segment elevation in identifying high risk patients for late sudden death (Wilson and Partridge, 1973) or those unsuitable for early mobilization (Morris et al., 1974) may indicate a possible role for the measurement of ST segment elevation. However, our findings suggest that there is no additional benefit in constructing a 42 lead ST segment map over measurement of the ST segments in the chest leads of the conventional 12 lead tracing.

We thank Mr. Ed Fletcher and the State Health Laboratory Services for estimating the serum enzymes, and calculating the cumulated enzyme rise.

**Discussion**

This study shows that the extent and amplitude of ST segment elevation detected in the praecordium is a poor indicator of the extent of myocardial necrosis; there was only a poor correlation between ST segment changes and serum enzyme release, the peak of two hourly or daily serum enzyme levels, and no correlation with several indices of clinical severity or clinical state at six weeks.

More significantly, these findings cast doubt on the assumption (Maroko et al., 1972, 1973; Reid et al., 1973, 1974; Braunwald and Maroko, 1974) that changes in ST segments detected by praecordial mapping will reflect trends in the extent of human myocardial injury. There was a wide day-to-day variation in individual patients and secondary rises in ST segments were not always corroborated with other evidence of reinfarction, such as changes in serum enzyme levels. In several patients, extension of necrosis which was obvious on sequential serum enzyme estimations was not detected in the daily ST segment maps performed in this study.
References


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