# Precordial Mapping of ST—T Segment in Acute Myocardial Infarction

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*A quantitative estimation of ST-T segment deviation from multiple precordial ECG leads is a fairly sensitive index of the geographic extent of myocardial ischaemia.*

*Precordial ST-T segment mapping was done with 72 electrode positions in 20 patients admitted with acute myocardial infarction to a coronary care unit. Mapping was also done on 5 normal subjects as controls. The results showed a good correlation with simultaneous clinical and enzymatic evaluation of the severity (extent) of myocardial ischaemia, and its duration in acute anterior myocardial infarction. It failed to reveal inferior wall as well as subendocardial infarction.*

*ST-T mapping is a good bedside guide for a clinician and retains its popularity because of its low-cost, non-invasive approach and standard rules of interpretation.*

THE elevation of the ST-T segment of the electrocardiogram (ECG) has been recognised for many years as one of the classical and earliest signs of acute myocardial ischaemia. Electrophysiological studies have proved that ischaemia following coronary artery occlusion leads to an acceleration of repolarisation with loss of resting membrane potential. This results in ST segment elevation in the surface ECG [8, 11]. Maroko and his associates [15] have shown that there is a significant epicardial ST segment elevation 15 minutes after experimental coronary artery occlusion in dogs. Attempts are being made by various workers all over the world for quantification of precordial ST segment elevation by a simple non-traumatic technique in patients which might prove of value in the assessment of size and severity of the initial ischaemic myocardial damage [5,6,9]. Serial measurements are being used to evaluate subsequent progress [13] or to test the effectiveness of drugs in altering the extent of ischaemic injury [3].

The present study was undertaken to investigate the value of ST-T segment elevation as a clinical guide to the size of infarct in patients in a coronary care unit. The main aim was to study :

a) the correlation between the extent of ST-T segment elevation and the clinical indices of severity of myocardial infarction,

b) the time course of ST-T segment changes, and

c) the relation between ST-T segment elevation and infarct size estimated by serum enzyme levels.

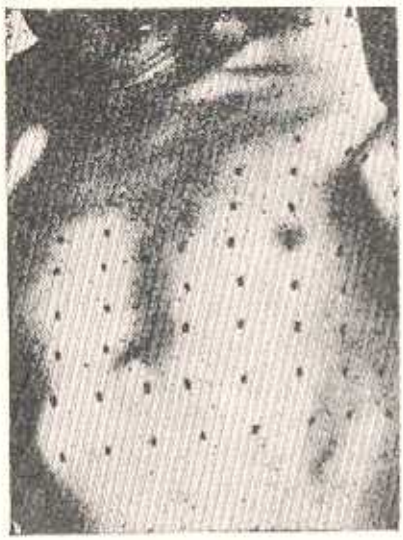

Fig. 1—Position and distribution of the recording points on the surface of the chest.

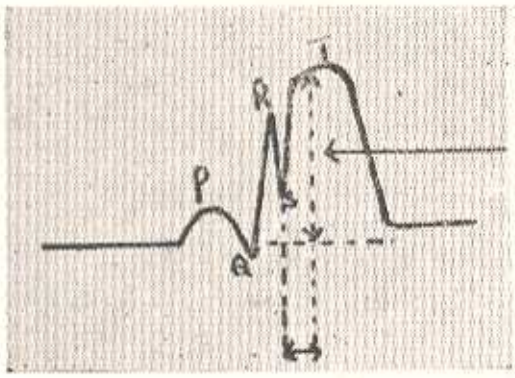


Fig. 2—Criteria used for ST segment elevation.

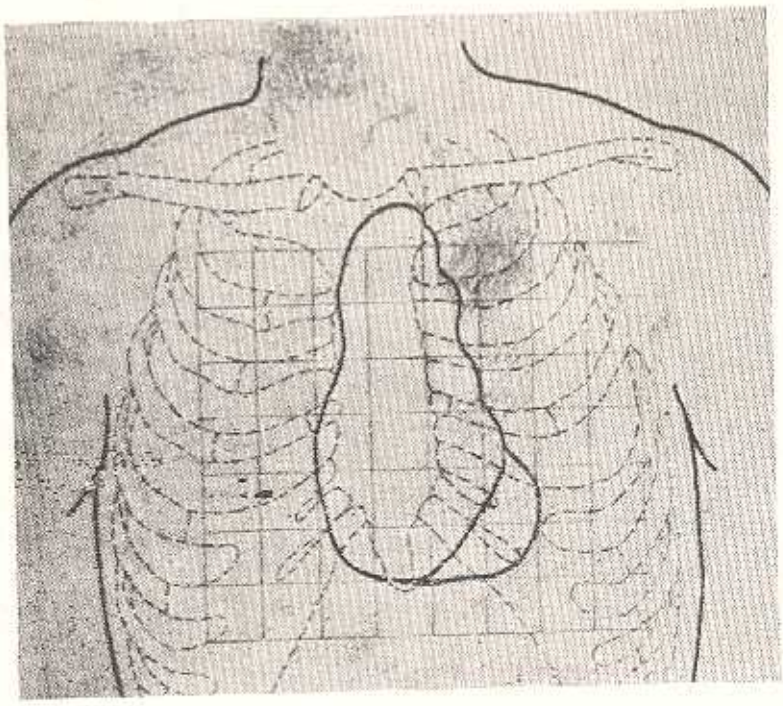

Fig 3.—72 points grid for ST mapping.

## Material and Methods

Studies were made on 20 patients (19 male and 1 female) aged between 31 and 57 years admitted with fresh myocardial infarction to the coronary care unit of Command Hospital, Air Force, Bangalore, between January 1978 and July 1979. Altogether, 98 precordial ST-T maps, each with 72 lead positions, were recorded. The diagnosis was based on the clinical features, the results of daily serum aspartate aminotransferase level and standard 12 lead ECG. Patients with right and left bundle branch block and infarction other than anterior or anterolateral were excluded from this study. Thus of the 20 patients, 2 cases who developed inferior wall infarction soon after and another 2 cases who developed evidence of bundle branch block were excluded. Therefore, the actual study group consisted of 16 patients only.

The precordial maps were recorded within 4—6 hours after admission, subsequently each day (i. e., at 24 hourly intervals) for the next 4 days, then on 10th day and subsequently, at weekly intervals as long as the patient was in the hospital. We were able to take records in 3 cases after 8 weeks sick leave. For computing the age of infarction, the day of admission to the hospital was taken as "Day 1".

ST-T maps were also recorded in 5 normal subjects who did not have any relevant past or present history of myocardial ischaemia or electrocardiographic evidence of any heart disease. These subjects were used as controls to find out the normal variability of ST-T segment. In this control group 72 point mapping was done on 3 consecutive days only.

*Technique of precordial ST-T segment mapping*

Single channel direct writing ECG machines, Cardioline and BPL Cardiart (108T), were used for all the records. It was ensured that all the records were taken with the same machine for the individual patient. The chest electrode used was metal suction type, with a diameter of 2.0 cm. In order to avoid spread of potentials, the electrode jelly was applied over a small area only and the electrode was carefully placed. All recordings were done on a standard ECG paper running at 25 mm per sec, and with standardisation of 10mm = 1 mv. The electrode was attached to the chest lead outlet. Recordings were made with the patients in supine position. Seventy two electrode positions were marked on the anterior chest wall, after shaving off the hair (Fig. 1). An indelible felt-pen was used for marking these points which were retouched daily to ensure accurate placement of the recording electrode for subsequent mapping. Once the chest points were marked, approximately 40-45 minutes were required for recording each 72 lead ST-T mapping electrocardiogram. For comparison, standard 12 lead ECGs were also taken simultaneously during each mapping. The results of various maps were analysed by the same observer, thereby eliminating observer error.

*Criteria used for ST-T segment measurements*

These have been described by Reid *et al* [30]. The ST-T segment deviations were measured using the TP segment as the isoelectric line, or the PQ segment when the TP segment was difficult to locate. The ST-T segment deviation was measured in mm to the nearest 0.5 mm at 0.06 sec, after the nadir of the S wave (Fig. 2) but excluding those with elevation of less than 2 mm.

The ST-T segment deviations so obtained were then recorded on a standard diagram on which 72 points were represented at equal intervals (Fig.3). Isopotential lines were drawn on the diagram to obtain a "Contour" map by joining points of equal magnitude and by interpolation when this was necessary. The surface maps produced in this way showed the area of maximum ST-T segment elevation and the area over which there was ST-T segment elevation or depression.

The QRS duration was checked in all the cases, since delayed intraventricular conduction caused by complete or partial bundle branch block can obscure ischaemic ST elevation [1]. In this study of 16 cases, no patient showed abnormal prolongation of QRS.

The daily sum of ST segment, i.e., $\Sigma$ ST was worked out for each patient by analysing all the 72 points recorded daily.

*Serum enzyme studies*

Daily evaluations of serum aspartate aminotransferase levels were done for first 4 days of admission and subsequently these enzyme levels were measured as and when ECG mapping for ST segment was done. The serum enzyme estimation was done by the standard technique used in our hospital laboratory.

*Clinical data recording*

Each patient's past history, presenting features, physical examination findings, ECG and chest X-Ray were recorded on admission. Haemodynamic complications and arrhythmias, if any, were recorded daily.

**Results**

*Control group*

The average age of these volunteers was 39.4 years, the range being 20-70 years.

No significant ST segment elevation was observed in these individuals. The maximum ST segment change was 1.5 mm which is not considered to be of any significance by any of the authorities with experience in this field.

The sum of ST segment, i.e., $\Sigma$ ST in these subjects was 26.1 $\pm$ 3.1 mm, and the maximum variation in the daily $\Sigma$ ST over the course of 3 days was 5.1 $\pm$ 2.5 mm within the same subject.

*Patients with acute myocardial infarction*

Table I shows the total number of male and female patients with their average ages. These patients had either transmural (9 cases) or non-transmural (7 cases) anterior myocardial infarction. None of these patients had any past history of ischaemic disease.

TABLE — I

*Sex and average age of patients with fresh anterior myocardial infarction* (n = 16)

| | Transmural Infarction | | Non-transmural infarcti | |
|---|---|---|---|---|
| Sex | No. of patients | Average age (yrs) | No. of patients | Averag age (y |
| Male | 8 | 46.2 | 7 | 42.6 |
| Female | 1 | 52.0 | — | — |
| Total | 9 | 49.1 | 7 | 42.6 |

Table II shows the time taken by the patients get admitted to intensive coronary care unit (ICC after the onset of symptoms which varied from che pain to breathing difficulty with profuse sweati and sinking sensation in the epigastrium.

TABLE — II

*Time of admission to hospital after the onset symptoms* (n = 16)

| Time (hours) | Number of patients |
|---|---|
| 0 - 4 | 4 |
| 4 - 8 | 7 |
| 12 - 24 | 5 |

Distinctive ST maps were obtained in all th cases of anterior myocardial infarction. In all cases there was an area of maximum ST segment eleva tion, surrounded by an area of lesser ST segmen elevation. The maximum height of the ST segmen elevation varied from 2mm to 10mm in differen patients on different days. Daily subsequent map showed either an area of resolution or extension o infarct size.

The maps of 2 cases have been shown in Figs 4 to 8 to show an extension of infarction (case No.2) and resolution of infarction (case No.9) on different days.

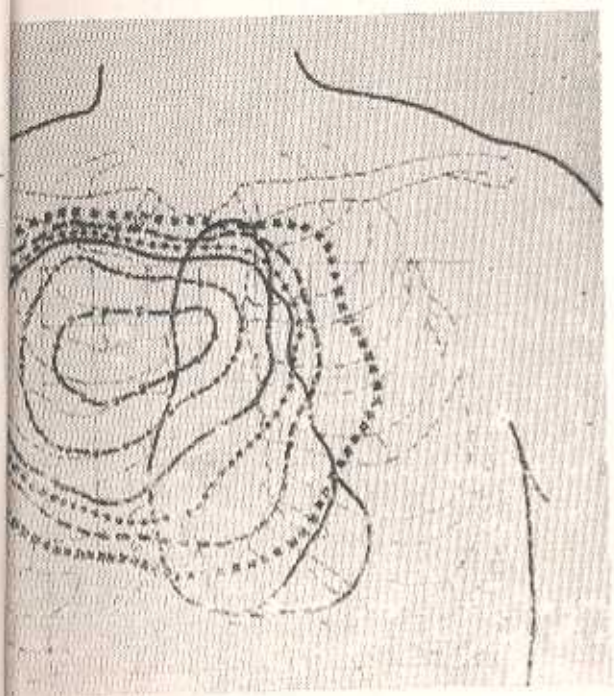

Fig. 4 — Case No 2 : ST elevation on 'day 1'

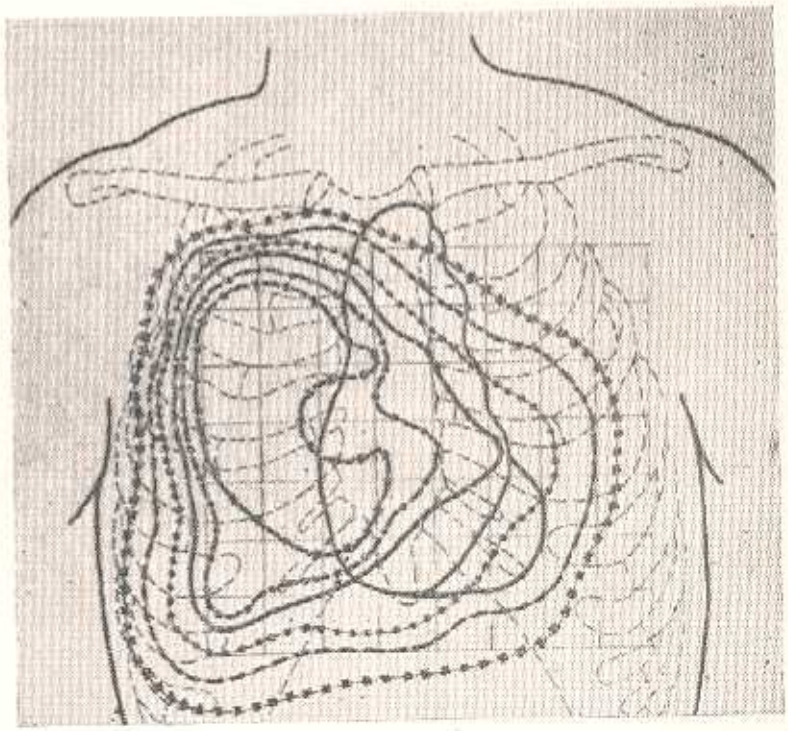

Fig. 5 — Case No 2 : Showing extension of infarction on 'day 2'.

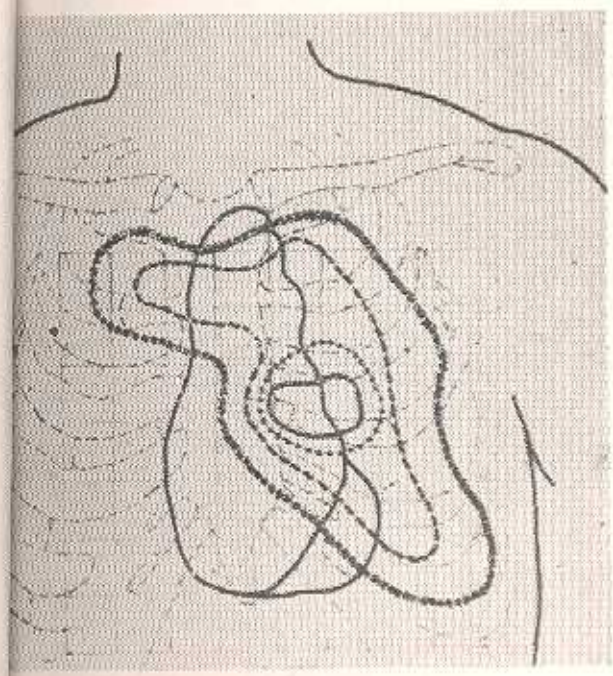

Fig. 6 — Case No 9 : ST elevation on 'day 1'.

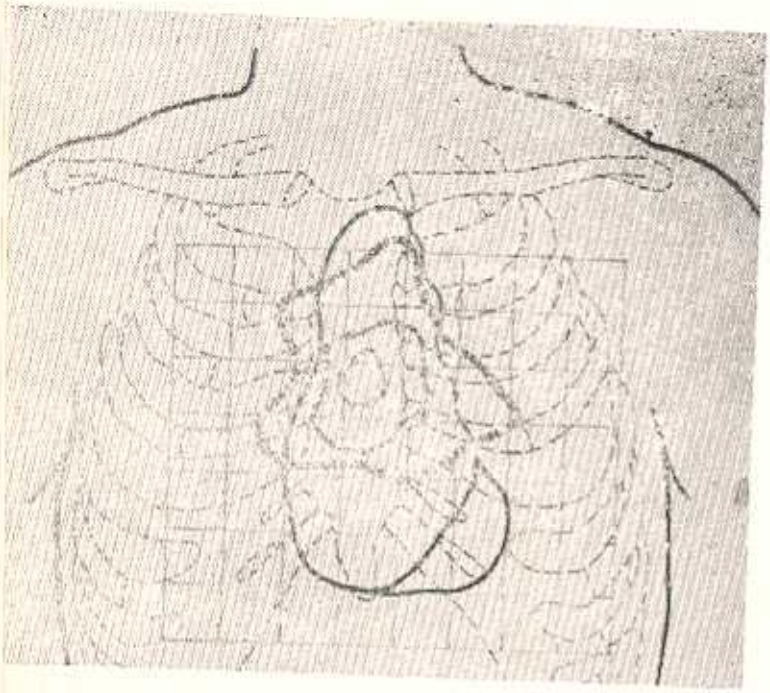

Fig. 7 Case No 9 : Showing regression of infarct on 'day 4'

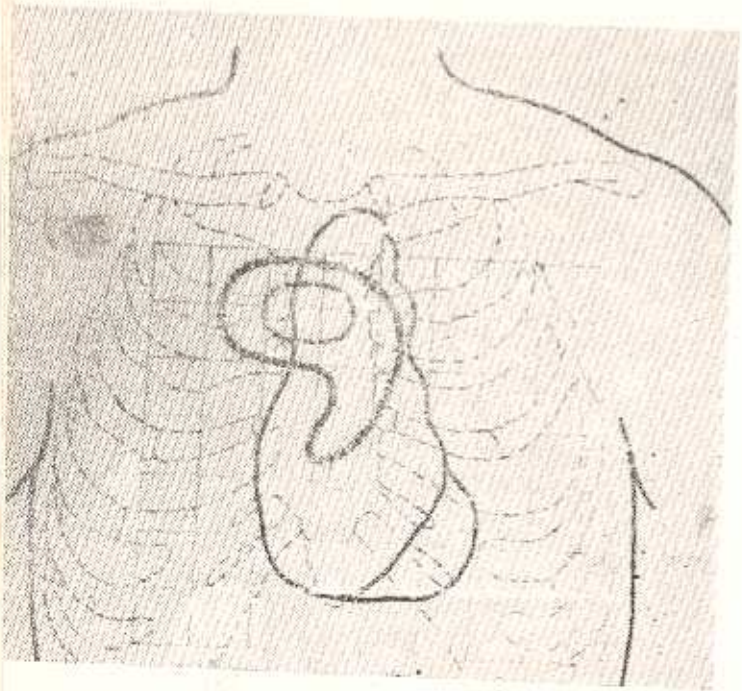

Fig. 8 —Case No 9 : Showing further regression of the infarct on 'day 24'

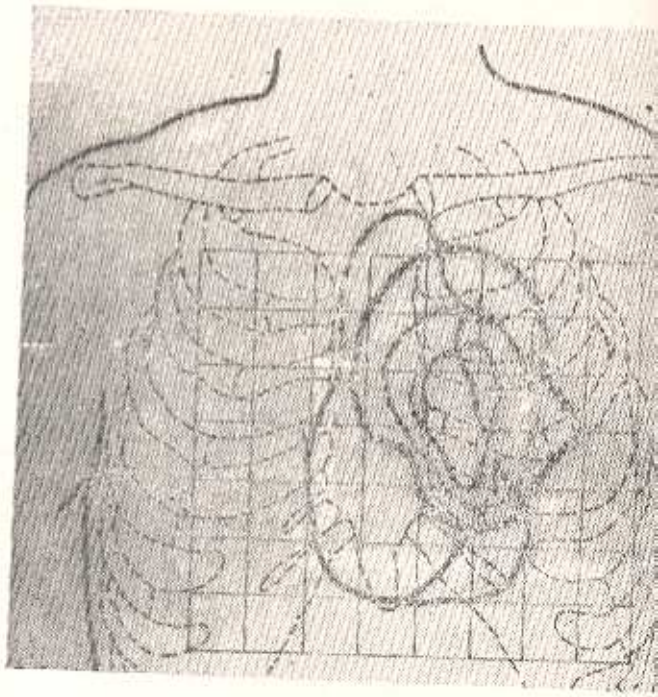

Fig. 9 —Case No 7 : ST mapping on 'day 1'

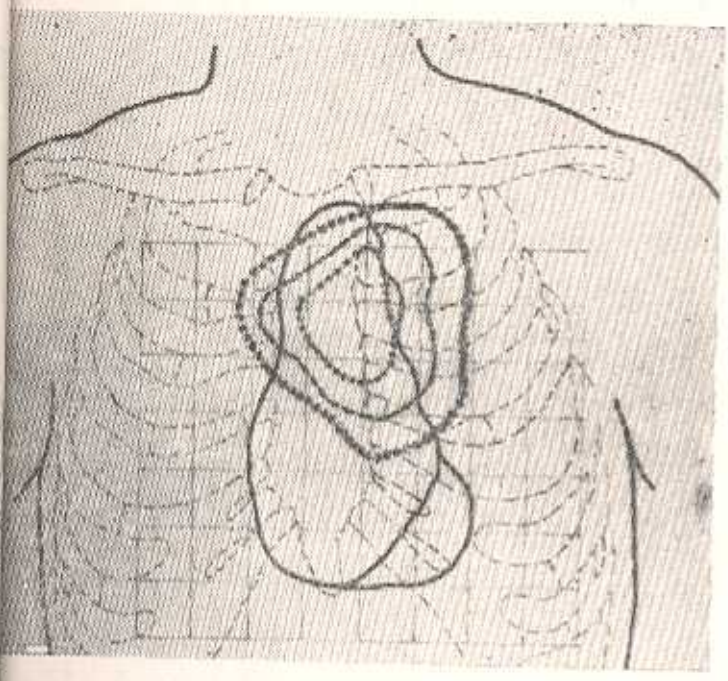

Fig. 10 — Case No. 7 : Showing regression of the infarct on 'day 4'

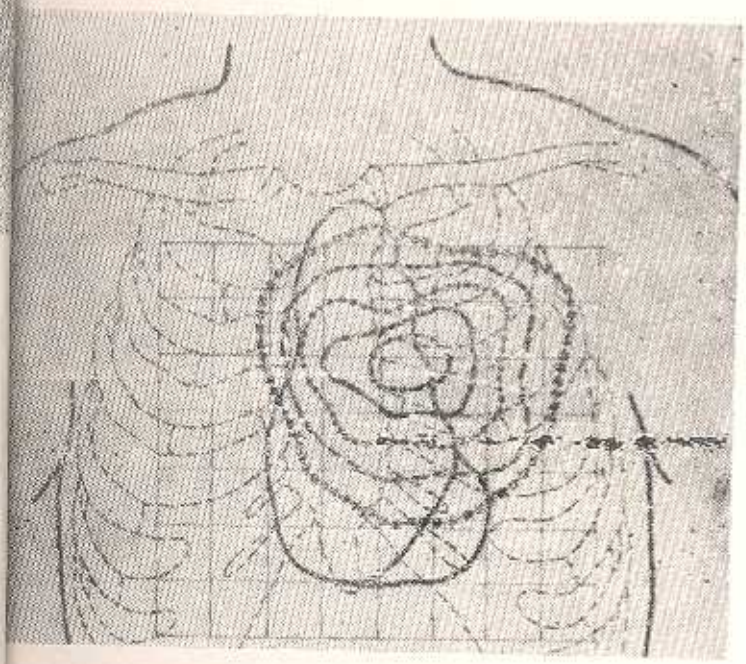

Fig. 11—Case No. 7 : Showing fresh extension of the infarct on 'day 10'

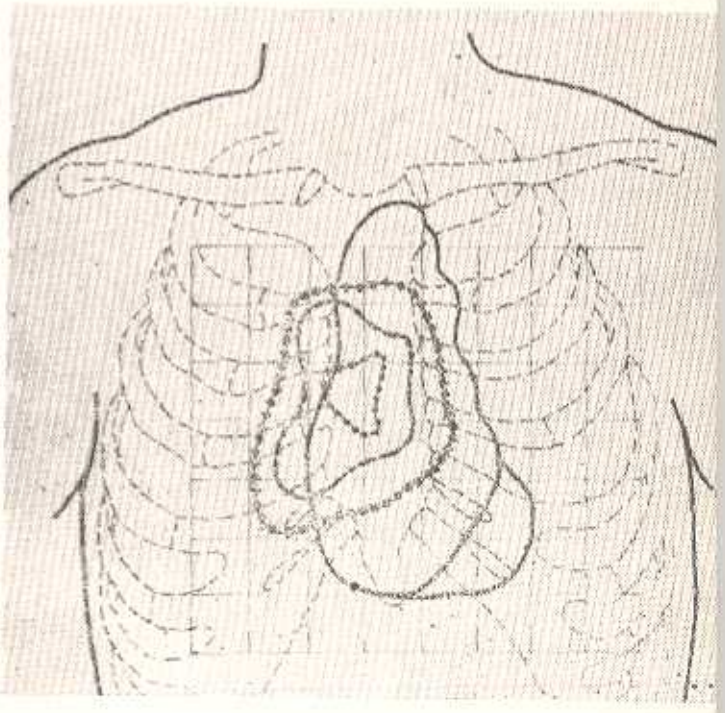

Fig. 12 — Case No. 2: Showing persistence of ST elevation even after 12 weeks. (compare with Figs. 4 and 5)

In 2 cases, initial maps showed resolution of infarction but subsequently, in both these cases patients developed fresh chest pain on the 10th day (case No.7) and 9th day (case No.14), and ST maps showed distinctly an extension of infarction by fresh ST segment elevation at various points (Figs. 9 to 11).

Except for 3 cases (case No. 2, 7 and 9), elevated ST segments came down to normal limits within 3 weeks. In case No.2, ST segment remained elevated even after 8 weeks of convalescence, i.e., after a total of 12 weeks (4 weeks hospitalisation plus 8 weeks sick leave) (Fig. 12).

Table III shows the sum of ST segment ($\Sigma$ST) in mm for each individual mapped day for all the 16 patients of myocardial infarction.

TABLE - III

*Showing daily $\Sigma$ ST (in mm) of each patient*

| Case No. | Day 1 | Day 2 | Day 3 | Day 4 | Day 10 | Day 17 | Day 24 |
|---|---|---|---|---|---|---|---|
| | | | Transmural Infarction | | | | |
| 1. | 66.5 | 86.5 | 52.5 | 35.0 | 33.5 | 37.5 | 28.5 |
| 2. | 116.5 | 156.0 | 150.5 | 100.5 | 98.0 | 84.5 | 82.5 |
| 3.* | 95.5 | 58.0 | 63.0 | 69.0 | 53.5 | 40.5 | — |
| 4. | 74.5 | 68.0 | 64.5 | 60.0 | 58.0 | 45.0 | 40.5 |
| 5. | 87.0 | 52.5 | 39.0 | 29.0 | 22.5 | 20.5 | — |
| 6. | 100.5 | 64.5 | 66.5 | 52.5 | 40.0 | 30.5 | 30.5 |
| 7. | 89.0 | 125.0 | 96.0 | 57.0 | 100.5 | 86.5 | 40.0 |
| 8.* | 32.5 | 31.5 | 46.5 | 40.0 | — | — | — |
| 9. | 126.0 | 92.0 | 80.5 | 80.5 | 60.5 | 68.5 | 60.5 |
| | | | Non-Transmural Infarction | | | | |
| 10. | 49.5 | 35.0 | 36.5 | 23.0 | 26.0 | 16.5 | 16.0 |
| 11. | 40.5 | 30.0 | 28.5 | 28.0 | 29.5 | 18.0 | 18.5 |
| 12.* | 23.0 | 32.5 | 31.5 | 35.0 | — | — | — |
| 13. | 56.5 | 50.0 | 30.5 | 26.5 | 20.5 | 20.0 | 20.0 |
| 14. | 30.5 | 21.0 | 20.5 | 19.0 | 49.5 | 42.0 | 30.0 |
| 15. | 36.5 | 36.0 | 28.5 | 21.5 | 18.0 | 23.0 | 18.5 |
| 16. | 42.5 | 36.0 | 30.5 | 28.0 | 25.5 | 23.0 | 25.0 |

* Patients discharged against medical advice.

On admission, the $\Sigma$ ST in myocardial infarction was greater than $\Sigma$ ST for normal (control) group. Similarly, Table III also shows that $\Sigma$ ST was more in transmural infarction than in non-transmural infarction ($P < 0.01$).

Table IV shows the fall in level of $\Sigma$ST of patients with transmural infarction on each mapped day. The cases showed a gradually decreasing $\Sigma$ST on subsequent days which signified resolution of the infarct size. In those cases where $\Sigma$ ST showed a progressive increase (case No.2) or re-elevation (case No.7), the patients had extension of infarction. This was corroborated also in standard 12 lead ECG. In other cases, although $\Sigma$ST showed a decline signifying resolution of infarct size, the 12 lead ECG did not necessarily show any evidence of resolution in the form of ST or T wave changes.

TABLE-IV

*Showing fall in levels of $\Sigma$ ST as compared to Day 1 $\Sigma$ ST in patients with transmural infarction on each mapped day* (n=9)

| Days | Levels 75% or above | Levels 50-75% | Levels raised but not normal | Normal levels |
|---|---|---|---|---|
| 1 | | | | |
| 2 | 7 | 2 | 4 | - |
| 3 | 5 | 3 | 1 | - |
| 4 | 3 | 4 | 1 | 1 |
| 10 | 1 | 5 | 2 | 1 |
| 17 | - | 2 | 6 | 1 |
| 24 | - | 1 | 4 | 4 |

Table V shows the daily serum aspartate aminotransferase levels of each patient on the mapped days. The highest enzyme level was 98 IU/litre in patient No. 2 on 2nd day of his admission to the hospital. For case No. 7, the enzyme showed a fresh rise to 80 IU/litre on 10th day when patient had fresh chest pain and ST map as well as $\Sigma$ST also showed an extension of the infarction.

TABLE—V

*Daily serum aspartate amino transferase levels of each patient in IU/litre* (n—16)

| Case No. | Day 1 | Day 2 | Day 3 | Day 4 | Day 10 | Day 17 | Day 24 |
|---|---|---|---|---|---|---|---|
| | | | Transmural Infarction | | | | |
| 1. | 30 | 75 | 50 | 38 | 30 | 12 | 10 |
| 2. | 58 | 98 | 78 | 56 | 46 | 25 | 20 |
| 3.* | 28 | 30 | 22 | 20 | 12 | 10 | — |
| 4. | 18 | 28 | 20 | 16 | 12 | 12 | 10 |
| 5. | 26 | 30 | 20 | 18 | 14 | 14 | 10 |
| 6. | 30 | 36 | 24 | 18 | 8 | 10 | 8 |
| 7. | 10 | 64 | 50 | 20 | 80 | 42 | 12 |
| 8.* | 16 | 18 | 10 | 8 | — | — | — |
| 9. | 12 | 12 | 10 | 10 | 10 | 10 | 10 |
| | | | Non-Transmural Infarction | | | | |
| 10. | 10 | 18 | 12 | 10 | 10 | 8 | 8 |
| 11. | 14 | 24 | 18 | 10 | 10 | 8 | 10 |
| 12.* | 8 | 20 | 12 | 8 | — | — | — |
| 13. | 10 | 16 | 8 | 10 | 10 | 10 | 10 |
| 14. | 8 | 22 | 12 | 8 | 12 | 10 | 10 |
| 15. | 12 | 10 | 16 | 10 | 12 | 8 | 8 |
| 16. | 12 | 12 | 14 | 10 | 8 | 10 | 8 |

*Patients discharged against medical advice.

It can be seen from Table V that rise in the enzyme level was more significant for transmural infarction cases. Amongst some of the transmural infarction cases also, although standard 12 lead ECG as well as ΣST and ST mapping showed a severe infarction, the enzyme level rise was not very significant. This is quite clear, for example in case No. 4 and 9 in Tables III and V.

Fig. 13 shows the relationship of the enzyme level and ΣST of a male patient (No. 7). The graph shows that, in keeping with the general trend, serum enzyme levels started rising on day 2, which corroborated with ΣST on the same day. It also shows that re-elevation of ΣST on day 10 was accompanied by a rise in the enzyme levels indicating the possibility of an extension of the infarct.

**Discussion**

*Advantages of ST mapping over standard 12 lead electrocardiogram.*

ST elevation in the routine 12 lead ECG strongly suggests myocardial infarction. The subsequent evolution confirms the diagnosis. According to Maroko *et al*[15], the degree of ST elevation may reflect

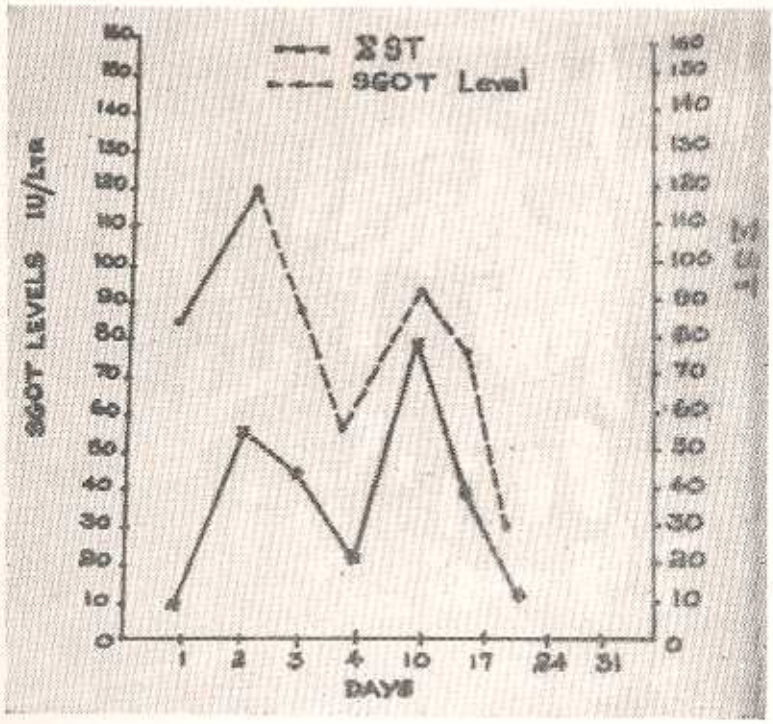


Fig. 13 — Σ ST and serum aspartate aminotransferase (SGOT) levels in Case No 7.

the size of the damage produced by myocardial ischaemia. Various studies of precordial ST segment mapping in myocardial infarction have used 16 to 72 electrode positions [3, 12, 13, 19, 20].

In this study, 72 exploratory electrode positions were used as described by Reid *et al* [20]. The ΣST from 72 lead position ECG is much more magnified and reflects changes that may not otherwise be discernible. It is more likely to show a re-elevation indicating infarct extension than the conventional 6 lead precordial ECG. In the present series, out of 2 patients with re-elevation of ST segment in 72 lead position ECG (case No.7 & 14), one (case No. 7) did not show any change in the standard 12 lead ECG. In a similar study, Reid *et al* [21] found that 4 of their 12 patients with re-elevation on the 48 lead, did not show any change in the conventional 6 lead precordial ECG.

*Measurement of ST segment deviation*

The magnitude of the ST segment deviation was measured 0.06 sec after the nadir of S wave[20]. Bruce *et al*[3] have shown that this is the most reliable point that is least affected by the S and T waves as well as the random artefacts that are obtained on the exercise ECG. Besides, this point is beyond the site of atrial repolarisation and, as such, any effect that this might have on the ST segment deviation is also avoided.

*Σ ST in myocardial infarction : initial level and course of changes*

In our study, the Σ ST in normal subjects (volunteers) was 26.1 ± 3.1 mm, which was significantly less than that of the patients with fresh myocardial infarction. It is also observed that Σ ST which was recorded in this study was highest on the first 2 days of admission and then fell progressively towards normal. Our findings are similar to those recorded by Reid *et al*, [20,21] in their own study.

*Correlation of ST segment deviation and serum enzyme levels in acute myocardial infarction*

Maroko and colleagues[13] have clearly established that a direct correlation exists between the extent of ST segment elevation measured in experimental coronary occlusion and infarct size measured by cardial creatine kinase depletion. However, M *et al*[14] found no correlation of maximum ΣST with enzyme values. They found a 32% drop of Σ uncomplicated infarcts in the first 24 hours. bility of resolution was noted in their pat According to them high Σ ST may be a function only of degree of ischaemic damage but als chest wall morphology and thickness.

The present study shows a significant co tion between Σ ST values and serum asparate am transferase peak values (Fig. 13), taking into co deration the fact that the infarction will first pro itself on the ECG as an elevation of ST segment later on in the serum, as elevated enzyme values.

Since investigations in laboratory ani have shown that the size of myocardial infarc after coronary artery occlusion can be changed pharmacologic interventions, it has been of g interest to measure extensions and reductions in size of the infarctions in patients. Two common n invasive techniques, which are presently in use are

a) serum enzyme studies, and

b) precordial mapping of ST segment.

The serum enzyme studies are primarily he ful in assessing the prognosis. A re-elevation serum level of enzymes can indicate an extension the size of the infarct. The drawbacks in the enz matic studies are that many tissues other than myoca dium are also rich in these enzymes, e.g., brain, lung skeletal muscles, liver and gastrointestinal tract. A of them can release these enzymes, thereby increa ing the serum levels. There is also a host of cond tions other than myocardial infarction which ma result in elevated serum enzyme level; some of thes are administration of salicylates, opiates, or coumari type anticoagulants; primary muscle disease, acut pancreatitis, extensive CNS damage, crush injury burns, infarction of kidney, spleen or intestine and hypothyroidism. Also minor trauma to limbs including intramuscular injections and tourniquet application to withdraw blood may increase these serum enzyme levels.

Another draw back with the enzyme studies is that their concentration in blood rises several hours after the onset of the clinical event and takes many hours to reach its peak. It may vary from 10 hours to 2—3 days or even more before the results of these serum enzymes are available from laboratory to estimate the infarct size. This time-lag between the onset of infarction and the time that the infarct size can be estimated limits the usefulness of this method in monitoring reduction in infarct size, since at that period the efficacy of any treatment is controversial.

The precordial mapping technique is a very simple electrocardiographic method that can be used by any physician with an ECG machine for bedside estimation of extent of ischaemic injury in patients with acute myocardial infarction. By evaluating the changes in precordial ST segment at specified times after the onset of the clinical events one can immediately assess the size and progression or regression of the ischaemic injury. The method has the advantage of showing practically instant changes in reversible tissue damage and makes it possible to monitor increase or reduction in size of the myocardium injured. This fact, together with the simplicity and rapidity of the method, makes it very suitable to evaluate without delay the effects of treatment on ischaemic myocardial injury.

ST segment mapping, however, does not provide an absolute value regarding the size of the infarct. One should remember that there can be many other causes of ST segment elevation other than myocardial ischaemia such as serum electrolyte concentration, bundle branch block, pericarditis and administration of certain drugs. In such patients, recording of precordial maps are invalid because of the non-specific effects of these on ST segment elevation. Therefore, the cause of the ST segment elevation will have to be determined by clinical and other means and patients with ST elevation from causes other than that of myocardial damage should not be taken up for study.

*Re-elevation of ST segment and extension of infarction.*

In our study, 2 (case No. 7 & 14) out of the 16 patients with anterior wall myocardial infarction, had a re-elevation of ST segments. Out of these 2 patients, one also had associated abnormal serum enzyme levels. Both these patients had recurrence of chest pain. The findings of re-elevation in $\Sigma$ ST, serum alterations in enzymes and associated recurrence of chest pain, strongly suggest the possibility of infarct extension.[21,22] This event occurs 5.8 days on an average after initial infarction. Other factors such as pericarditis or aneurysm formation must be considered in the interpretation of the re-elevation of ST segment (or $\Sigma$ ST) in patients with acute anterior wall myocardial infarction. Niarchos and Mckendriks[18] have recently reported incidence of pericarditis to be 11.3% following acute myocardial infarction. In their series, a friction rub appeared in most patients between the 2nd and 4th day after infarction.

Precordial maps are no longer useful for following up patients with myocardial infarction when pericarditis appears; they then maintain high $\Sigma$ ST for a number of days despite clinical and biochemical evidence of recovery. Since pericarditis cannot be differentiated from extension of injury by ECG, serial enzyme estimation and frequent auscultation are helpful in differential diagnosis.

In our group of 9 patients with transmural anterior myocardial infarction, re-elevation of $\Sigma$ ST occurred in one patient (case No.7) on 10th day and was not associated with appearance of friction rub and his chest pain was not pleuritic. Although pericarditis may have been present subclinically, there was still enzymatic evidence of myocardial necrosis.

The effects of left ventricular aneurysm formation on the ST segments are more difficult to evaluate since it is common for patients with anterior myocardial infarction to have dyskinetic areas of the anterior or apical myocardium [22]. The transient nature of the rise in ST segment (or $\Sigma$ ST), the associated clinical manifestations such as further ischaemic pain and the secondary rise in serum enzymes make it unlikely that aneurysm formation alone could have accounted for the findings in the present study.

*Relationship of high ST and prognosis*

Although death has been linked to high ST segment elevation of the standard ECG, in a study by Madias and co-workers [11] mortality did not correlate well with maximum $\Sigma$ ST.

In our study of 16 patients of fresh anterior myocardial infarction, there was no mortality. Since the number of patients in this study is small no conclusion can be drawn regarding relationship of high $\Sigma$ ST with prognosis.

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