Failure of ST segment elevation to predict severity of acute myocardial infarction¹

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Praecordial ST segment elevation was measured at 35 electrode positions in each of 40 patients admitted to a coronary care unit after acute transmural anterior myocardial infarction. Serial praecordial electrocardiographic maps were recorded to determine (a) the time course as well as reproducibility of measurements of ST segment alterations, and (b) the degree of correlation between the magnitude of ST segment elevation and the severity of infarction, as assessed clinically or by sequential estimations of serum creatine kinase activity. Large variations in ST segment elevation were found in different patients with a comparable degree of myocardial damage, and at intervals of as little as four hours in the same patient. These variations were greater than could be explained by technical factors, and were not related to apparent changes in the patients' clinical status. The patterns of release of myocardial creatine kinase showed that the time course of ST segment elevation was longer than the period of myocardial necrosis. No correlation was found between the myocardial infarct size as determined by enzyme release and the highest levels of ST segment elevation recorded. The findings suggest that ST segment elevation as measured by praecordial electrocardiographic mapping does not constitute a reliable index of the size or severity of myocardial infarcts in man.

Though elevation of the ST segment of the electrocardiogram has been recognized for many years as one of the classical signs of acute myocardial infarction (Eppinger and Rothberger, 1909), the electrophysiological correlates of ischaemic injury were not established until intracellular potentials could be recorded from the intact heart. It is now known that ischaemia following coronary artery occlusion leads to an acceleration of repolarization, with loss of resting membrane potential, which results in ST segment elevation in the surface electrocardiogram (Samson and Scher, 1960; Prinzmetal *et al.*, 1961). Of particular interest recently has been the report of Maroko and his associates (1971) that there is a significant 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 histological changes 24 hours later. The possibility has thus arisen that quantification of praecordial ST segment elevation by a simple non-traumatic technique in patients might prove of value for the assessment of size and severity of the initial ischaemic damage (Maroko *et al.*, 1972a; Reid, Pelides, and Shillingford, 1971; Neilsen, 1973; Maroko, 1974), and that serial measurements might be used to evaluate subsequent progress (Reid *et al.*, 1974) or test the effectiveness of drugs in altering the extent of ischaemic injury (Pelides *et al.*, 1972; Maroko *et al.*, 1973).

The present study was undertaken to investigate further the value of ST segment elevation as a clinical guide to infarct size in patients in a coronary care unit. The main aims were to study the relation between the extent of ST elevation and clinical indices of severity of infarction, the time course of ST segment changes, and the relation between ST elevation and infarct size estimated by calculations of total CK release from four-hourly determinations of serum enzyme activity (Shell, Kjekshus, and

¹Supported by the National Heart Foundation and the Medical Research Council of New Zealand.

Received 12 August 1975.

Sobel, 1971; Sobel *et al.*, 1972; Norris *et al.*, 1975). However, initial results showed such wide variations in the extent of ST elevation from patient to patient, and at different times in the same patients, that it also became necessary to investigate the reproducibility of measurements.

Methods

Electrocardiographic mapping

A modified version of the electrode blanket described by Maroko and colleagues (1972a) was used. This was a plastic fabric template measuring 45×25 cm in which 35 square holes were cut, in 7 vertical and 5 horizontal rows. The holes measured 2×2 cm, the distance between centres of horizontal rows being 4 cm, and between successive centres of the second to seventh vertical rows 4.5 cm. Centres of the first and second vertical rows, which lay to the right and left of the sternum respectively, were 7 cm apart. The template was placed horizontally, with the top right hand hole in the second intercostal space, 2 cm lateral to the right sternal border. The first vertical row of holes lay to the right of the sternum and the last in the mid-axillary line; the blanket thus covered the praecordial area overlying the anterior and lateral surfaces of the left ventricle. Between four and six QRS complexes from each of the 35 sites were recorded with a metal suction electrode 1.5 cm in diameter attached to the V lead of a standard electrocardiograph and recorded at a sensitivity of $1 \text{ cm} = 1 \text{ mV}$, and at a paper speed of 25 mm per second. A representative complex with a stable baseline was selected from each of the 35 positions, cut out, and pasted on a card. The ST segment elevation from each position was recorded to the nearest 1 mm, excluding those with elevations of less than 2 mm. Where the ST segment was sloping, elevation at the mid-point was taken as the height of ST elevation for that complex. Each 'map' was analysed (in many cases, independently by two observers) for the total ST elevation from all 35 positions (ΣST), and the number of positions showing ST elevation of 2 mm or greater. The QRS duration was checked in all cases, since delayed intraventricular conduction caused by complete or partial bundle-branch block can obscure ischaemic ST elevation (Beckwith, 1970). Cases in which the QRS duration was greater than 0.08 s were therefore excluded from analysis; in the event, it became necessary to exclude only one case because of abnormal QRS prolongation. Since ST segment elevation caused by inferior infarction cannot be detected using praecordial electrode positions, studies were carried out only on patients with anteroseptal or anterolateral infarction, as shown on the standard electrocardiogram.

Measurement of total CK release

Samples of peripheral venous blood were analysed for CK activity four-hourly, and total CK release was calculated by our modification (Norris *et al.*, 1975) of the method of Sobel and colleagues (1972). In our

method, accuracy of the calculation of total CK release is improved by individual measurement of CK degradation rate for each patient.

Patients studied

Forty patients with transmural anterior infarction and five normal subjects were studied. The following investigations were made, most patients being included in more than one group:

i) Normal subjects A single map was recorded on each of 5 subjects aged between 40 and 70 years, who had no clinical evidence of heart disease.

ii) Reproducibility studies Reproducibility of recordings was investigated in 12 patients as follows:

- Two QRS complexes with stable baseline were selected from each praecordial electrode position and were mounted separately, so that ΣST could be measured using different complexes from the same record (beat-to-beat variation). Ten such comparisons were made on 5 patients.
- A possible effect of changes in skin resistance on ΣST was investigated by making 2 consecutive records from each position after removal and immediate replacement of the suction electrode (electrode placement variation). Ten such comparisons were made on a further 5 patients.
- Mapping was repeated in the same patient after one hour (hour-to-hour variation). This was done in 12 patients who were free from chest pain and arrhythmias, 1 to 3 days after the onset of infarction.

iii) Correlation of ST elevation with clinical indices of severity Clinical severity of infarction, assessed by the arterial blood pressure and pulse rate (recorded routinely by the nursing staff), and the presence or absence of evidence of pulmonary venous hypertension, interstitial oedema, or pulmonary oedema in a chest x-ray taken during the acute phase (Norris *et al.*, 1969), was compared with the maximum recorded ΣST in 29 patients. These patients had a mean of 6 (range 2 to 19) maps taken at intervals of 1 to 2 days, the first being recorded within 24 hours of onset of the most severe pain.

iv) Time course of ST elevation The time course of ST elevation during 1 week after the onset of infarction, and its relation to the duration of CK release from the infarct, was studied in 16 patients in whom 4-hourly measurements of serum CK activity had been made, and in whom maps were available for at least 4 of the following 5 periods after the onset of infarction: 0 to 12 hours, 1, 2, 3 to 4, and 5 to 7 days. The time course over the first 24 hours was examined further in 11 patients by comparing the ST elevations recorded within 6 hours of onset of infarction with the ST elevations at 7 to 24 hours after the onset.

v) Correlation of ST elevation with total enzyme release The highest recorded value for ΣST was compared with the total release of CK (IU/ml serum)

in 15 patients in whom the latter integral could be calculated accurately (Norris *et al.*, 1975).

vi) Effect of beta-adrenergic receptor blockade on ST elevation during recovery from infarction In order to determine whether Σ ST could be altered after completion of the infarct, at a time when infarct size could presumably not be altered, practolol 20 mg was given intravenously to 8 patients at a mean time of 9 days (range 5 to 13 days) after the onset of infarction. Maps were recorded immediately before, and 1 hour after the injection, and Σ ST and the heart rate were measured. Approximately 6 maps had already been recorded on these patients, who were all convalescing and had no clinical evidence of reinfarction at the time of the study.

Results

i) Normal subjects

No ST segment elevation was found in the 5 normal subjects.

ii) Reproducibility studies

Results of these measurements are shown in Table 1. The mean variation of Σ ST measured from different QRS complexes in 10 comparisons (beat to beat variation) was 4.7 per cent (range, 0 to 17%), while the mean variation in 10 comparisons in which the suction electrode was removed and then immediately reapplied (electrode placement varia-

tion) was 8.3 per cent (range, 2 to 17%). When recordings were made one hour apart in 12 patients, the variation was greater (mean variation, 14.3%; range, 0 to 47%). In 7 patients, the hour-to-hour variation was within the range which we found with beat-to-beat and electrode placement variation, but in 5 patients was greater (22 to 47%). These results suggested that the pronounced variations in Σ ST which occurred within hours or days in individual patients (see below) were in most cases the result of real changes, and were not caused by random beat-to-beat variations or by differences in technique of making the recordings.

iii) Clinical correlation

No relation was apparent between arterial blood pressure, pulse rate, and Σ ST when different patients were compared; nor were variations in Σ ST within the same patient related in any constant way to changes in blood pressure or heart rate. Furthermore, there was no relation between the clinical severity of infarction, judged by the degree of pulmonary venous hypertension present in chest x-rays taken during the acute phase, and the highest recorded value for Σ ST. In 11 patients without pulmonary congestion, the mean maximum Σ ST was 76 mm (range 19 to 130 mm), while in 9 patients with interstitial or frank pulmonary oedema, the mean maximum Σ ST was 55 mm

TABLE 1 Reproducibility of ST segment elevation measurements

Case No.	Beat-to-beat variation			Electrode placement variation			Hour-to-hour variation		
	Σ ST ₁	Σ ST ₂	% Variation	Σ ST ₁	Σ ST ₂	% Variation	Σ ST ₁	Σ ST ₂	% Variation
1	36	35	3				36	58	47
	57	58	2						
2	30	31	3				30	24	22
	47	47	0						
3	154	144	7				149	145	3
	142	148	4						
4	65	55	17				60	55	9
	55	55	0						
5	54	53	2				54	68	23
	71	65	9						
6				128	136	6	132	126	5
				123	128	4			
7				85	92	8	88	88	0
				85	91	7			
8				50	52	4	51	38	29
				41	36	13			
9				54	64	17	59	57	3
				55	58	5			
10				56	55	2	56	59	5
				54	64	17			
11							96	120	22
12							76	73	4
Mean $\pm$ 1 SD			4.7 $\pm$ 5.2			8.3 $\pm$ 5.5			14.3 $\pm$ 14.3

(range 9 to 112 mm). In 9 patients with pulmonary venous congestion but no oedema, the mean maximum Σ ST was 68 mm (range 12 to 118 mm). Substitution of the number of electrode positions showing ST elevation of 2 mm or greater for maximum Σ ST did not improve these correlations.

iv) Time course of ST elevation

The time course of ST elevation in 16 patients in whom serial records were available for the first week after onset of infarction is shown in Fig. 1. There was a very wide variation in Σ ST between individual patients, with no important difference in the range of values for Σ ST at different times within the first week. The protracted course of ST elevation, compared with the duration of myocardial necrosis, as judged by the time course of CK release into the circulation, is also shown in Fig. 1. ST elevation persisted, in general, for a considerably longer period than did CK release.

Changes in 11 patients in whom maps were recorded within 6 hours (mean 4.5 hours) of the onset and again at 7 to 24 hours (mean 16 hours) after the onset are shown in Table 2. Σ ST was less on the second occasion in 6 cases, greater in 3, and the same in 2. The mean Σ ST at 7 to 24 hours was 39 mm; because of the large variation, this was not

FIG. 1 Temporal changes in ST segment elevation are shown in 16 patients with transmural anterior infarction, in whom ST segment maps were available for at least 4 of the 5 periods indicated on the horizontal axis. Closed circles refer to observations made during the period of creatine kinase release, while open circles refer to those made after completion of enzyme release. There is no obvious tendency for ST elevation to decrease at 5 to 7 days after onset of infarction, nor after cessation of enzyme release.

TABLE 2 Changes in ST segment elevation during 24 hours after onset of transmural anterior infarction

Case No.	Σ ST (mv) Within 6 hours	7 to 24 hours
13	19 (6)	19 (14)
14	6 (4)	20 (23)
15	9 (4)	9 (24)
16	107 (4)	25 (24)
17	49 (6)	33 (9) 51 (15)
18	130 (3)	118 (7)
19	68 (4)	22 (10) 40 (15)
20	66 (5)	39 (9) 21 (16)
21	123 (4)	21 (8)
22	46 (4)	18 (10)
23	15 (6)	31 (10) 68 (14)

Figures in parentheses refer to time after the onset of chest pain in hours.

significantly lower than the mean Σ ST of 58 mm at 3 to 6 hours.

The striking differences between patients are shown by the four illustrative cases in Fig. 2 in which the course and extent of myocardial necrosis, as judged by the release of CK into the circulation, is compared on the same time-scale with the course of ST elevation. It should be noted that the closed circles relate to the total of CK release into the circulation which had occurred up to the time shown in the graph (Sobel *et al.*, 1972; Norris *et al.*, 1975); they do not represent the serum levels which are in fact declining in an exponential fashion when total release reaches a plateau.

Fig. 2 shows that in some patients a great reduction in ST elevation occurred during the acute phase of CK release (Fig. 2A), while in others a gradual increase in elevation occurred during the first few days even though the rate and extent of CK release was small (Fig. 2B). In other cases Σ ST fell and rose again (Fig. 2C), or remained constant and of slight degree, in patients with large infarcts as judged by CK release (Fig. 2D).

v) Correlation of ST elevation with total enzyme release

The relation between the maximal figures for Σ ST and myocardial infarct size assessed from four-hourly measurements of serum CK activity is shown in Fig. 3. No significant correlation is demonstrated, and the correlation was not improved by substitution of the figures for the number of electrode positions showing ST elevation for Σ ST.

vi) Effect of beta-adrenergic reception blockade after completion of infarction

When practolol 20 mg was given intravenously to 8 patients who were convalescent after myocardial infarction, and the epicardial map repeated after

FIG. 2 Four illustrative cases of transmural anterior infarction are shown, contrasting the sum of ST elevations from 35 precordial positions (Σ ST) (open circles) with the development of myocardial necrosis, as judged by the integral of creatine kinase (CK) activity released into the circulation (closed circles). This latter integral relates to total enzyme release, and not to the serum levels (see text). The plateau on the total enzyme release curve denotes cessation of enzyme release, while the slope of the curve before the plateau indicates the velocity of release; the height of the plateau is considered to be an index of myocardial infarct size. Case (A): Σ ST drops from 123 mm to 21 mm over 4 hours, despite continuing myocardial necrosis, evidenced by a steeply rising enzyme curve. Case (B): a considerable increase in ST segment elevation has occurred 3 to 4 days after the onset, despite a comparatively small extent and rate of enzyme release. Case (C): increasing ST segment elevation has occurred from 1 to 3 days, though myocardial necrosis, judged from the plateau of total CK release, has ceased. Case (D): ST segment elevation is relatively constant but minimal, though the infarct is large, judged from the enzyme curve. The development of pulmonary oedema in this patient, who did not have pre-existing heart disease, suggested that enzyme release was a better predictor of the severity of infarction than was ST segment elevation in his case.

one hour, there was a consistent reduction in Σ ST, accompanied by a 10 per cent reduction in heart rate (Fig. 4). The mean change in ST elevation was -23 per cent (range -10% to -47%), which was significantly different from the change which was found to occur in one hour in the patients shown in Table 1 ($t=3.17$; $P<0.01$).

FIG. 3 The highest recorded Σ ST is plotted against total release of creatine kinase (CK) activity in 15 patients. No correlation was apparent between these measurements.

Discussion

Our studies in patients with transmural anterior myocardial infarction have not revealed a simple relation between the magnitude of precordial ST segment elevation and clinical or biochemical indices of severity or size of the infarct. The time course of ST elevation was prolonged beyond the period of myocardial necrosis as judged by the activity of CK enzyme released into the circulation. Moreover, considerable variations in the degree of ST elevation, accompanied neither by changes in clinical status nor in the rate of enzyme release, occurred in individual patients. Our reproducibility studies showed that these alterations were too large to be explained solely by variability in recording techniques, since the temporal changes were, in most cases, outside the range of variation which occurred from beat to beat or with slight alterations in placement of the precordial electrode.

The results thus emphasize the limitations of the precordial ST segment mapping technique for the bedside estimation of ischaemic injury in patients with acute anterior myocardial infarction. Since this conclusion is seemingly at variance with results from previously reported experimental (Maroko *et al.*, 1971) and clinical studies (Maroko *et al.*, 1972a; Reid *et al.*, 1974), it is of importance to examine similarities and differences between these and our studies.

Maroko and his colleagues have clearly established that a direct correlation exists between

FIG. 4 The effect of practolol (20 mg intravenously) on ST segment elevation and heart rate is shown in 8 convalescent patients who were studied at a mean of 9 days after the onset of infarction. The fall in ST segment elevation (23%) was significantly different ($P < 0.01$) from changes occurring in 12 other patients in whom measurement of ST segment elevation was repeated after one hour without administration of practolol. Heart rate fell by 10 per cent (average of 7 cases).

the extent of ST segment elevation measured at 15 minutes after experimental coronary occlusion and infarct size measured either by myocardial CK depletion (Maroko *et al.*, 1971) or the severity of tissue necrosis determined both by light and electron microscopy (Maroko *et al.*, 1972b). Extrapolation of these results to the clinical setting may be hazardous for a number of reasons.

First, ST segment elevation is an immediate effect of myocardial ischaemia, but as cells undergo necrosis it is likely that elevation becomes less. Enzyme release, on the other hand, is a manifestation of cellular death, starting approximately 6 hours after the onset of ischaemia, and continuing for 24 to 48 hours (Sobel *et al.*, 1972). In an area of infarcting myocardium both processes may coexist for a time, some cells undergoing necrosis more quickly than others. It is conceivable that Σ ST measured at the very onset of infarction in man might correlate more closely with clinical or biochemical indices of severity than Σ ST measured after patients have been admitted to a coronary care

unit, when infarction is well established. Thus it might be argued that in patients such as those illustrated in our Fig. 2A and D, necrosis of all ischaemic cells had occurred quickly, so that ST elevation (though perhaps present early) declined quickly, followed by a rapid release of CK enzyme. This seems unlikely as a general explanation for our findings, because of the protracted course of ST elevation in many patients (Fig. 1 and 2B and C), and the lack of any clear evidence that Σ ST was higher when measured at 3 to 6 (mean 4.5) hours after the onset than at 7 to 24 (mean 16) hours after the onset in the same patients.

Second, the experimental results of Maroko *et al.* (1971) relate to epicardial mapping of a small well-defined infarct involving an accessible part of the free wall of the left ventricle, produced by occlusion of a branch of the left anterior descending coronary artery. In patients, myocardial infarction may be more extensive and diffuse, often involving parts of the heart (e.g. the interventricular septum) not readily accessible to surface electrocardiography. Also, praecordial mapping must be done through the intact chest wall, so that variations in body build or of anatomical positions of the heart may seriously distort comparisons among different patients. For these reasons it would be surprising if a strictly quantitative correlation between praecordial ST elevation and size or severity of infarct was apparent when different patients were compared with one another. However, data did not reveal even a rough correlation between ST segment elevation and the degree of ischaemic damage assessed by clinical and biochemical indices. It might nevertheless be argued that these findings do not negate the value of changes in Σ ST in predicting favourable or unfavourable trends in an individual patient. No such trends were, however, suggested by the results of the present study in which large hour-to-hour increases or decreases in Σ ST were noted without obvious alterations either in the clinical status or in the patterns of release of the enzyme CK.

One difficulty in evaluating methods for measuring infarct size in man during life is that there is at present no absolute method against which other methods may be compared. In particular, discrepancies between infarct size assessed on the one hand by ST elevation, and on the other by total CK release, might be the result of deficiencies in the enzyme method rather than in the electrocardiographic mapping technique. Clearly, as has been discussed previously, these two variables are reflecting different pathophysiological processes; moreover it is possible that release curves of enzymes or products of myocardial catabolism other than CK might indicate a different duration or

magnitude of myocardial necrosis. We have data on this point from an unpublished study in which the time course of release of CK was compared with that of α -hydroxybutyrate dehydrogenase (HBD) in a further 16 patients with transmural infarction. In 50 per cent of these patients the duration of release of the 2 enzymes was similar, while in 50 per cent HBD release continued for 11 to 48 hours after CK release had ended. Even if it is assumed that in these patients late HBD release represented continuing myocardial necrosis rather than hepatic damage from venous congestion, it is still likely that in many patients the duration of ST elevation was greater than that of myocardial necrosis as measured by HBD release.

From a practical viewpoint, a good correlation between total CK release and clinical indices of severity has been reported by ourselves (Norris *et al.*, 1975) and others (Sobel *et al.*, 1972; Mathey *et al.*, 1974). No comparable information is available from clinical studies of ST elevation, but a lack of correlation between clinical severity of infarct and praecordial ST elevation has already been suggested (Reese, Scheidt, and Killip, 1973). Thus, we believe that the measurement of CK release is an inherently more accurate method for the clinical assessment of myocardial infarct size than is the quantification of ST segment elevation.

A possible explanation for the present findings is that the extent of ST segment elevation overlying ischaemic myocardium is related only indirectly to the extent and degree of ischaemia, but more directly to mechanical or electrical imbalance occurring at boundaries between healthy and infarcting muscle. It is well known that paradoxical wall movement occurs at the site of an infarct, that this persists during the healing stage and even permanently if a ventricular aneurysm develops, and that this is associated with chronic ST segment elevation. Moreover, experimental evidence suggests that ST elevation is commonly greatest at boundaries between healthy and ischaemic tissue, and may diminish at the centre of large infarcts (Cohen and Kirk, 1974). Such an explanation for the causation of ST elevation would be consistent with the present finding that ST elevation often persists after myocardial necrosis has ceased, and declines gradually over 2 to 3 weeks as the infarct heals. It could also explain the effect of practolol, which caused a reduction in ST elevation at a time when necrosis was presumably complete. This effect could have occurred through reduction in paradoxical movement by beta-adrenergic receptor blockade at the boundary between infarcted and normal muscle. An alternative explanation for the reduction in ST elevation after practolol is that

the change was related to slowing of the heart rate by the drug. However, the reduction in heart rate was slight (10%), and there was no relation between the degree of slowing in individual patients and the reduction in ST elevation; this finding is in agreement with that of Pelides *et al.* (1972) who found a similar degree of reduction in ST segment elevation after practolol (-19%, compared with -23% in the present study) in patients during the acute phase of myocardial infarction. The possibility that practolol reduced ST elevation by relieving persistent ischaemia, possibly at the boundary of the infarct, should also be considered, but seems unlikely, as the patients were fully convalescent and had been free of chest pain for several days before injection of the drug.

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