

Acute myocardial infarction

Evaluation of praecordial ST segment mapping¹

Peter L. Thompson and Vi Katavatis

From Coronary Care Unit, Perth Medical Centre, Western Australia

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.

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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 (Σ ST). 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 Σ ST ($\overline{\Sigma$ ST) and the mean of the NST (\overline{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 auto-analyser 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.

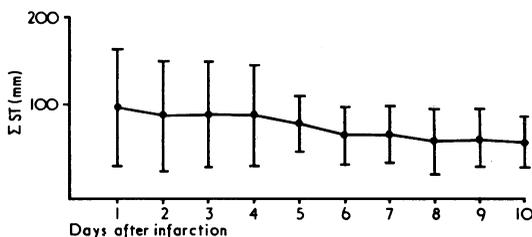


FIG. 1 ST on days 1 to 10 of hospital course. Mean \pm SD, n=17.

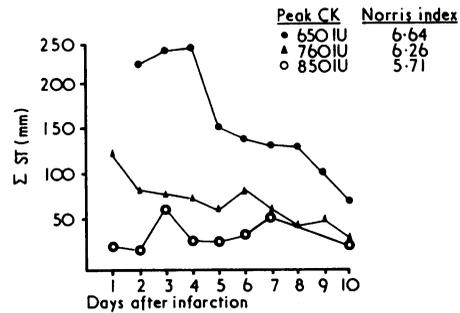


FIG. 2 ST in 3 patients of approximately equivalent enzyme rise (peak CK) and clinical severity (Norris index). Note the differences in patterns of ST segment elevation in 3 patients.

Clinical data recording

Each patient had past history, admission physical examination, electrocardiogram, and chest x-ray tabulated on admission; haemodynamic complications and arrhythmias were recorded daily. Clinical severity was calculated by the methods of Peel *et al.* (1962) and Norris *et al.* (1969). Survivors were reviewed at 6 weeks to assess physical capacity (NYHA classification).

Results

The Σ ST 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 Σ ST on day 1 ranged from 23.5 to 280.5 mm. The standard deviations from the mean Σ ST was >50 per cent for each of days 1 to 10. The trends in Σ ST 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 Σ ST on day 1 or the Σ ST did not correlate with clinical grades of severity of infarction as

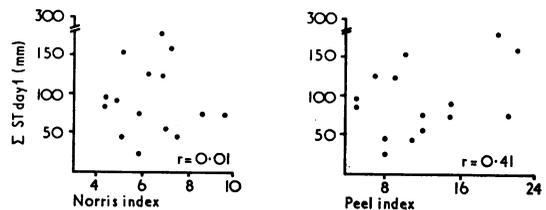


FIG. 3 Correlation between ST on day 1 with clinical indices of severity of infarction.

TABLE 1 Comparison of ΣST day 1 and $\Sigma \overline{ST}$ with various subgroups: there were no significant differences in ST segment elevation between any subgroups

		ΣST day 1		$\Sigma \overline{ST}$	
		n	Mean $\pm SD$	n	Mean $\pm SD$
Age	> 60	6	66 \pm 54	7	82 \pm 43
	< 60	9	117 \pm 64	10	97 \pm 40
Previous infarction	Yes	5	79 \pm 42	6	86 \pm 41
	No	10	135 \pm 79	11	86 \pm 43
Previous angina	Yes	5	87 \pm 43	6	69 \pm 27
	No	10	107 \pm 74	11	102 \pm 45
Pulmonary vascular congestion (x-ray)	Yes	7	129 \pm 76	8	95 \pm 44
	No	8	78 \pm 41	9	85 \pm 46
Basal crepitations and/or 3rd heart sound	Yes	10	114 \pm 68	11	83 \pm 35
	No	5	77 \pm 50	6	106 \pm 58
Ventricular ectopic beats	Yes	8	115 \pm 81	9	92 \pm 48
	No	7	86 \pm 34	8	89 \pm 37
Norris index	> 6.5	7	114 \pm 84	8	95 \pm 45
	< 6.5	8	88 \pm 42	9	87 \pm 42
Peel index	> 10	9	107 \pm 78	10	93 \pm 45
	< 10	6	95 \pm 47	7	89 \pm 41

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

The ΣST on day 1 failed to predict haemodynamic or arrhythmic 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

ΣST 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 $\Sigma \overline{ST}$ ($r=0.61$, 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 ΣST on day 1 or $\Sigma \overline{ST}$. The area of ST segment elevation (NST) did not correlate with any of the serum enzyme parameters.

Trends in ΣST compared with trends in serum CK

Daily praecordial ST segment maps were compared with CK estimations in 38 patients. In 3 patients re-elevation of ΣST 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

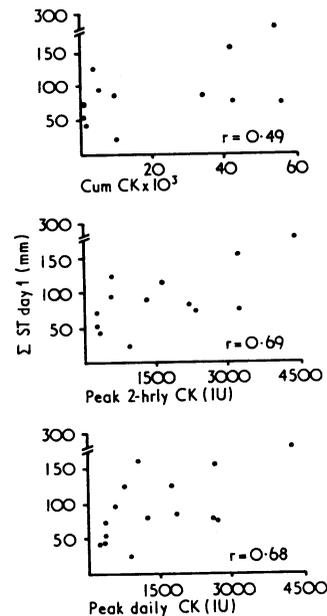


FIG. 4 Correlation between ST on day 1 with enzymatic indices of necrosis. (Cum. CK=cumulated CK derived from Shell et al., 1971).

measured every 2 hours indicated significant reinfarction in 2 patients, but the praecordial ST segment map failed to show any change in the ΣST or NST. A striking example is seen in Fig. 6.

Comparison of 42 lead and 12 lead electrocardiogram

ΣST calculated from 42 praecordial leads correlated well ($r=0.90$) with ΣST calculated from the 6 praecordial leads (V1-V6) of a standard electro-

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

	ΣST day 1 (n=15)	$\Sigma \overline{ST}$ (n=17)	NST day 1 (n=15)	\overline{NST} (n=17)
Norris index (n=17)	0.01 (NS)	0.22 (NS)	-0.18 (NS)	-0.52 (NS)
Peel index (n=17)	0.41 (NS)	-0.01 (NS)	0.05 (NS)	-0.18 (NS)
Cumulated CK (n=15)	0.49 (NS)	0.41 (NS)	0.357 (NS)	0.08 (NS)
Peak 2-hour CK (n=15)	0.69 ($P<0.05$)	0.61 ($P<0.05$)	0.43 (NS)	0.304 (NS)
Peak daily CK (n=17)	0.68 ($P<0.01$)	0.52 ($P<0.05$)	0.34 (NS)	0.14 (NS)

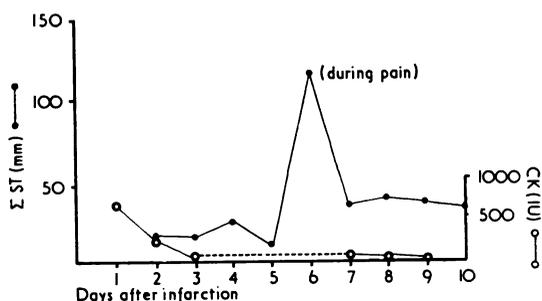


FIG. 5 A 62-year-old man with acute anterior myocardial infarction. On 6th hospital day, chest pain was associated with gross elevation of ST segments; subsequent serum enzyme estimations showed no rise.

cardiogram and the trends in Σ ST estimated by the two methods closely paralleled each other.

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

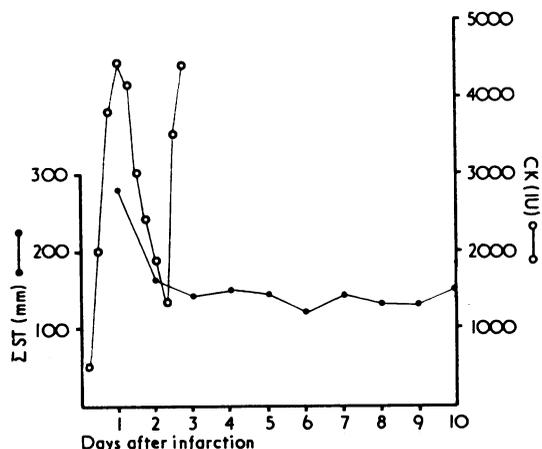


FIG. 6 A 52-year-old man with extensive anterolateral myocardial infarction. On 2nd hospital day, recurrence of chest pain was associated with gross re-elevation of serum CK; praecordial mapping failed to show re-elevation of ST segments.

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. 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 Pantridge, 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.

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Requests for reprints to Dr. Peter L. Thompson, Coronary Care Unit, Perth Medical Centre, Shenton Park, Western Australia 6008.