

Praecordial ST segment elevation

New technique for continuous recording and analysis

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The measurement of praecordial ST segment elevation after myocardial infarction is of value in assessing the natural history of ischaemic injury and the effectiveness of intervention. Hand analysis is, however, time consuming and inaccurate. A technique for continuous recording from 35 praecordial leads and subsequent computer analysis is presented, together with illustrative case studies. Changes in body posture and in heart rate are of importance in subsequent data interpretation.

Interest in attempts to reduce the extent of ischaemic myocardial damage in patients after myocardial infarction and thus to decrease the late incidence of shock and cardiac failure and improve long-term prognosis has led us to reappraise the use of praecordial ST segment mapping as a method of assessing the degree of myocardial ischaemic injury. The technique of praecordial ST segment mapping in man was introduced by Reid *et al.* (1971) and Maroko *et al.* (1972), and is based on experimental work in animals showing a positive correlation between epicardial ST segment elevation early after ligation of a coronary artery and subsequent myocardial damage (Rakita *et al.*, 1954; Maroko *et al.*, 1971; Kjekshus *et al.*, 1972). Though absolute infarct size may not be accurately predicted by this technique (Norris *et al.*, 1976), it is likely that changes in praecordial ST segment elevation, at least in the early hours after infarction, do reflect acute changes in underlying myocardial ischaemic injury. A close correlation has been shown in the dog between the magnitude of praecordial and of epicardial ST segment changes (Muller *et al.*, 1975), and further between epicardial ST segment elevation and changes in local myocardial oxygen tension (Sayen *et al.*, 1961) and blood flow (Kjekshus *et al.*, 1972). The serial measurement of praecordial ST segment elevation should then be of value in assessing both the natural history of myocardial ischaemic injury after infarction and in testing the effectiveness of any pharmacological or metabolic intervention.

The method of praecordial ST segment mapping in common use (Reid *et al.*, 1971; Maroko *et al.*, 1972) is laborious, involving measurements by hand from multiple electrocardiographic recordings. The intermittency of these recordings and the potential subjectivity of the assessment do not allow accurate documentation of spontaneous variations in ST segment elevation, particularly over short periods of time. This knowledge is, however, essential for the accurate interpretation of the results of clinical studies involving therapeutic intervention. For these reasons, we have developed a new technique for the continuous recording of average ST segment elevation and its subsequent measurement by computer.

Technique

LEADS AND ELECTRODES

Thirty-five praecordial leads were used arranged in 5 horizontal rows of 7 according to the method described by Maroko *et al.* (1972) (Fig. 1). The skin was prepared with Cambridge electrode jelly, care being taken not to smear the jelly. Self-adherent electrodes (Devices) were applied, the plastic body of each electrode remaining attached to the permanent lead system; only the adhesive disc was changed between studies.

SYSTEM OF RECORDING

A block diagram of the recording system is shown in Fig. 2a. The lead selector unit allowed signals from individual leads to be examined and recorded separately, and by a matrix of 35 switches corres-

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Received for publication 23 June 1976

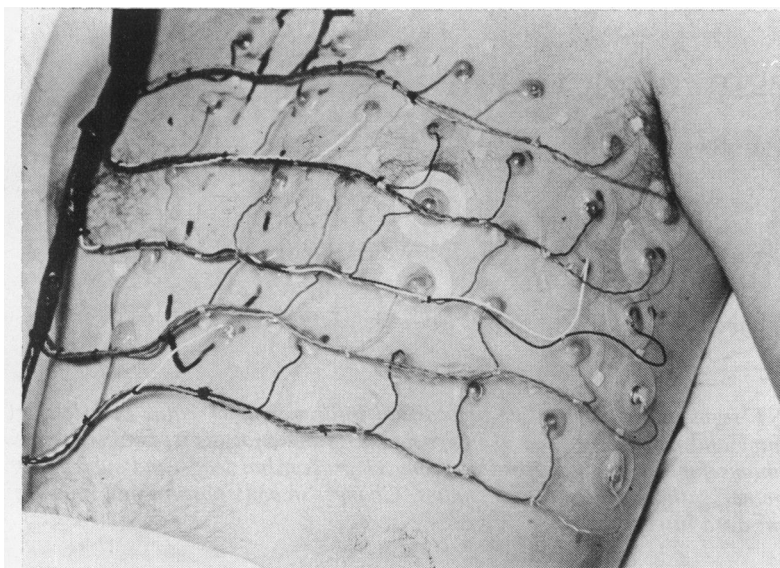


Fig. 1 The position of the 35 praecordial leads and the self-retaining electrode system in situ.

ponding to the chest electrode pattern, combined in any combination to produce a 'mean lead' signal. This mean lead signal was formed by summing the selected electrode potentials in a 100 K resistor summing network at the input of a single high input impedance instrumentation amplifier, and then subtracting the reference potential similarly

derived from the limb leads. The switching was arranged so that the output thus obtained was the arithmetic mean of the selected electrode potentials with respect to the Wilson central terminal potential. The mean lead signal was then recorded on one of the 3 channels of an FM magnetic tape recorder. The other 2 channels were used to

Fig. 2a A schematic representation of the system for continuous recording of average ST elevation.

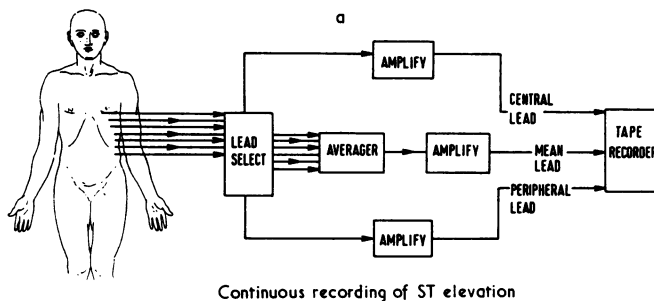
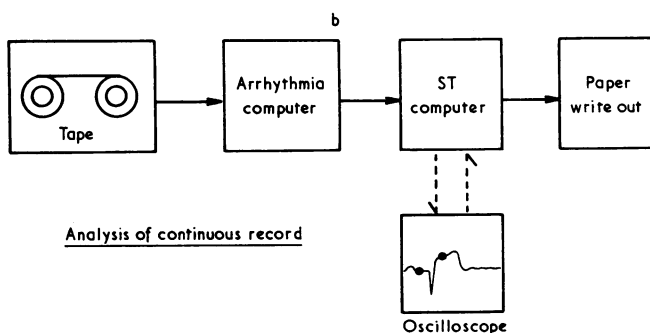


Fig. 2b A schematic representation of the system of analysis. The ST computer measures the potential difference between the points illustrated on the oscilloscope electrocardiogram.



record separately any 2 single leads from the 35-lead matrix individually selected at the lead selector unit. The whole system was mounted on a compact trolley which could be wheeled to the bedside (Fig. 3).

Signals from each of the 35 leads were recorded on paper immediately before monitoring and this was repeated at the end of the tape recording period so that changes in the pattern of ST elevation could be recognised as well as changes in magnitude. Leads with more than 1 mm ST elevation were selected for averaging and the 'mean lead' was then continuously recorded on tape together with one central lead (generally the lead showing maximum ST elevation) and one lead from the periphery of the area showing ST elevation. A 1 millivolt calibration signal was included on the mean lead recording for each study.

A typical distribution of ST elevation over the 35 leads from a patient with anteroseptal myocardial infarction is shown in Fig. 4. Representative peripheral and central leads are illustrated and at the bottom of the figure is the mean lead derived from those leads enclosed by the interrupted line.

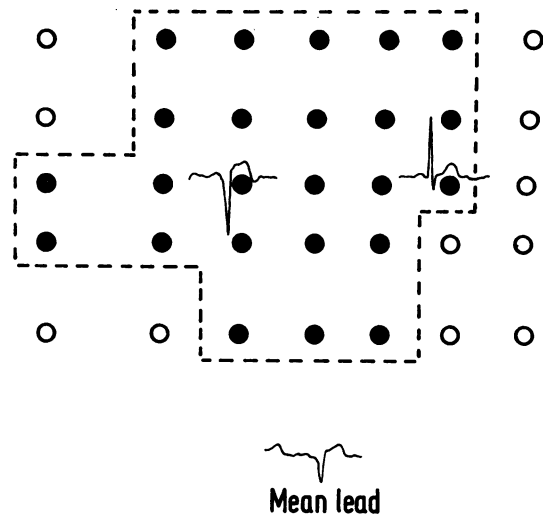


Fig. 4 A typical distribution of leads showing more than 1 mm ST elevation in a patient with anteroseptal infarction. Representative central and peripheral leads are shown. The lead at the bottom is the mean of all those enclosed by the interrupted line.

System of analysis

Subsequent analysis of the tape recorded signals was carried out using the system illustrated in Fig. 2b. After calibration of the system, the mean lead signal was replayed from tape at 60 times the original recording speed and analysed using a

special purpose arrhythmia computer (Neilson, 1974), which identified each of the normally conducted sinus complexes and rejected those with conducted defects, ventricular extrasystoles, and artefacts. Trigger pulses identifying the normal beats were fed from the arrhythmia computer to

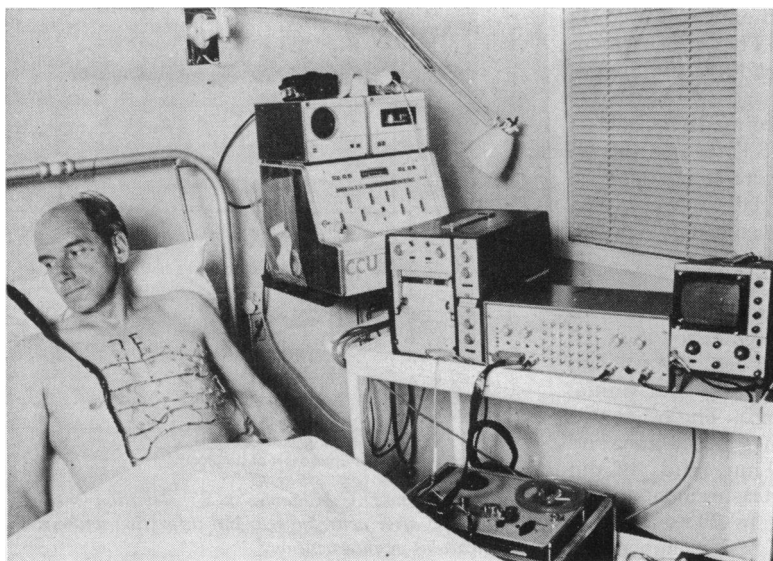


Fig. 3 The Edinburgh system for continuous recording of ST elevation. The electrodes are in place on the patient's chest. The 'ST averager' is on the top of the trolley with an oscilloscope to its right and a paper recorder to the left. The tape recorder is on the lower shelf.

the ST computer along with a delayed version of the meaned electrocardiographic signal.

In the ST computer (Neilson *et al.*, 1968) the trigger pulses associated with each electrocardiographic complex control the timing of two samples of the amplitude of the signal voltage at selected instants, each of which is fixed in relation to QRS. Operator controls allow positioning of these sample points. The reference point is positioned in the centre of the isoelectric PR segment and the measurement point on the ST segment between the J point and the upstroke of the T wave. For each complex the ST computer subtracts the voltage at the reference point from that at the measurement point to obtain a measurement of ST elevation for that beat independent of baseline deviations. Each measurement is held in the computer until replaced by that for the next beat, generating a beat-by-beat record of ST elevation, this waveform is smoothed in a low pass filter and the resultant smoothed signal representing the time course of ST elevation is recorded on paper.

The overall system noise in the final record is equivalent to less than 0.004 mV peak-to-peak ST deviation which when processing from a real electrocardiogram is negligible in comparison with natural variation.

Results

COMPARISON WITH HAND MEASUREMENTS

In a preliminary study, 35-lead maps for each of 3 patients were recorded on paper less than 10 minutes before monitoring and again after a period of at least 2 hours of monitoring. The ST elevations greater than 1 mm were selected and measured on both maps for all 3 patients. The measurement point used was 0.06 s after the nadir of the S wave (or the peak of the R wave in leads without an S wave). These measurements were made by each of 5 observers and the summated ST segment elevations obtained for each patient were compared with the corresponding initial and final values derived from the product of the computer mean, which was continuous over the monitoring period, and the number of leads.

The first patient showed a computer value of 89 mm for the initial map and 70 mm for the final map, by comparison with values from hand measurement ranging from 85 to 109 mm (initial map) and 66 to 79 mm (final map). The other two patients showed computer values of 68 mm and 30 mm (initial map), and 49 mm and 45 mm (final map), whereas hand measurements gave values of 51 to 80 mm and 21 to 29 mm (initial map), and 32 to 51 mm and 36 to 42 mm (final map),

respectively. The values obtained by the 5 observers differed greatly showing the considerable interobserver variation introduced by the hand method of analysis. In addition, the change in ST elevation between the initial and final maps showed considerable variation between observers: from -19 mm to -30 mm (computer -19 mm) in the first patient; from -19 to -36 mm (computer -19 mm) in the second patient; and from +12 mm to +18 mm (computer +15 mm) in the third patient.

ILLUSTRATIVE CASE STUDIES

Continuous recording and analysis of praecordial ST segment change has been carried out in patients with anterior myocardial infarction within 6 hours of onset of symptoms. Three representative case studies are reported to illustrate the potential application of this technique.

(a) Patient D.G. (Fig. 5)

This 72-year-old woman was studied from 5½ hours after the onset of chest pain. The standard 12-lead electrocardiogram showed an acute anteroseptal myocardial infarct and the diagnosis of infarction was subsequently confirmed by serum enzyme levels. At the time of study she was free of pain, having received 10 mg morphine intramuscularly 3 hours after the onset of pain. The blood pressure was 170/100 mmHg and the heart rate 80 per min; there were no signs of cardiac failure. She remained free of pain and cardiac failure throughout the 2 hours of study, and received no further

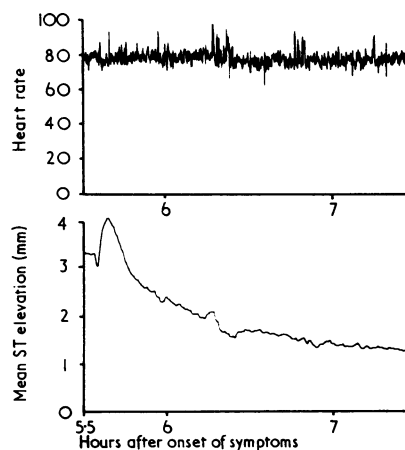


Fig. 5 Patient D.G. Changes in ST elevation (mean of 13 leads) after acute myocardial infarction without complications or intervention.

medication; throughout this time she lay flat and slept. The blood pressure on completion of the study was 155/95 mmHg and the heart rate was unchanged.

More than 1 mm ST elevation was found in 13 of the 35 leads at the onset of the study, but in only 5 leads 2 hours later. The average ST elevation from the 13 leads was 3.3 mm initially and had fallen to 1.2 mm by 2 hours. Thus a patient without a major change in clinical state or treatment had a 60 per cent fall in ST elevation over 2 hours.

(b) *Patient C.R.* (Fig. 6)

This 56-year-old man was studied from 3 hours after the onset of symptoms. The standard electrocardiogram showed anteroseptal infarction, confirmed by enzyme studies. He had mild chest pain throughout the study but no other complications. One hour before the study he received 10 mg morphine intramuscularly, but no other drugs were administered. When the recording was started he had no signs of cardiac failure and the blood pressure was 120/80 mmHg, with a heart rate of 60 per minute which remained unchanged over the following 2½ hours. More than 1 mm ST elevation was found in 21 leads at the onset of recording. Over the 2½-hour period the mean ST elevation fell from 2.5 mm to 1 mm; thus, in this man a spontaneous fall in ST elevation of 40 per cent took place during this time.

(c) *Patient G.C.* (Fig. 7)

This 53-year-old man was monitored from 4 hours after the onset of symptoms. One hour after the

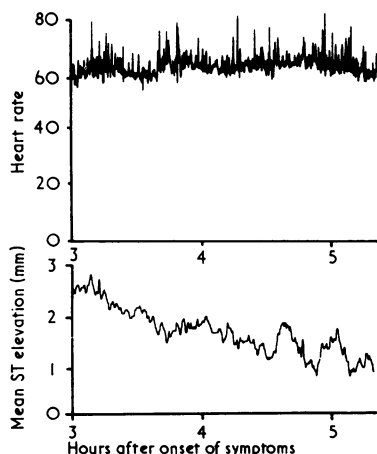


Fig. 6 *Patient C.R.* Changes in ST elevation (mean of 21 leads) after myocardial infarction without complications or intervention.

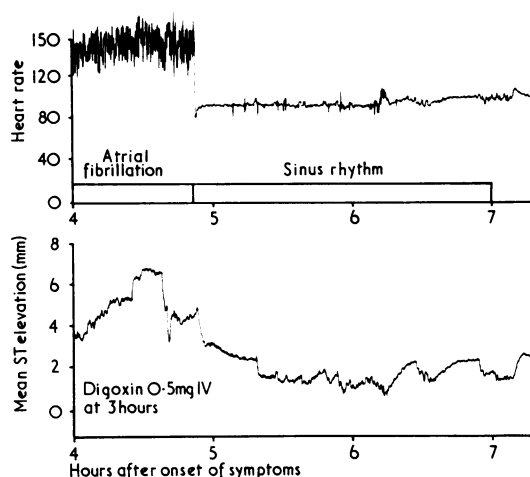


Fig. 7 *Patient G.C.* Changes in ST elevation from 4 hours after onset of symptoms of myocardial infarction, during atrial fibrillation and after reversion to sinus rhythm.

chest pain began he required DC conversion from ventricular fibrillation and was started on a lignocaine infusion. The electrocardiogram showed anteroseptal myocardial infarction. Two hours before ST monitoring was started he developed atrial fibrillation and was given digoxin 0.5 mg intravenously one hour later. At the onset of monitoring his blood pressure was 100/70 mmHg and heart rate 130 per min (atrial fibrillation), but there were no signs of cardiac failure. ST elevation of more than 1 mm was found in 24 of the 35 leads. During the period of ST recording he reverted to sinus rhythm, but apart from nausea and vomiting had no further complications. The average ST elevation rose progressively during atrial fibrillation from 3.5 mm at 4 hours to a maximum of 6.7 mm 30 minutes later. There was a short period (at 4 hours 40 minutes) of slowing of the ventricular rate during atrial fibrillation which was accompanied by an abrupt fall in ST elevation. A further abrupt fall occurred at the time of reversion to sinus rhythm, and was followed by a more gradual decline over the ensuing ¾ hour.

EFFECTS OF POSITIONAL CHANGE

Conspicuous variations in recorded ST elevation have been noted as a result of positional change alone, related to a change in direction of the electrical axis of the heart with respect to the chest wall in different body postures. Three illustrative studies are reported.

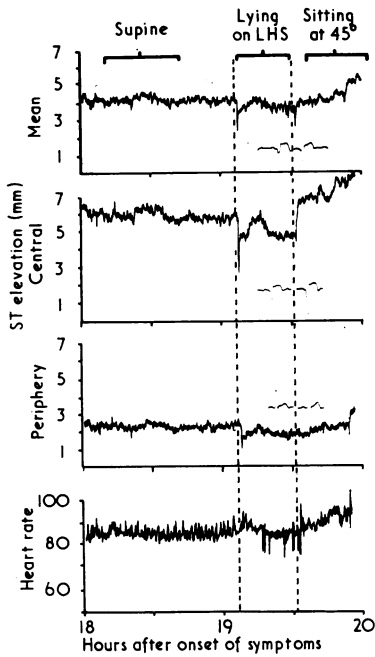


Fig. 8 Patient G.S. The effect of posture on ST elevation measured from (1) the average of 26 leads, (2) a selected central lead, and (3) a selected peripheral lead. LHS = left-hand side.

(a) Patient G.S. (Fig. 8)

This 44-year-old man was studied from 18 hours after the onset of symptoms. There was electrocardiographic and enzyme evidence of anteroseptal myocardial infarction. At the time of the study he was free of pain and clinical signs of cardiac failure. His blood pressure was stable throughout the study at 130/80 mmHg and no drugs were given. For the first hour of recording he remained still and supine, he then turned onto his left side for 25 minutes, and then sat up for a further 30 minutes. A small reduction in recorded ST elevation was seen in the mean lead while lying on his left side. This positional effect was more obvious in the recording from the central lead.

(b) Patients A.H. and A.W. (Fig. 9a, 9b)

These men, aged 52 and 61 years, respectively, were asymptomatic but showed persistent stable ST elevation from old anterior myocardial infarction. Mapping was performed to show the effect of positional change on the spatial distribution as well as magnitude of ST elevation. Fig. 9a shows the isopotential maps of these 2 patients recorded in a variety of body positions. Fig. 9b shows the summed ST elevations of all 35 leads. Distinct changes occurred, both in the surface distribution and in the sum of ST elevation. Maximum ST

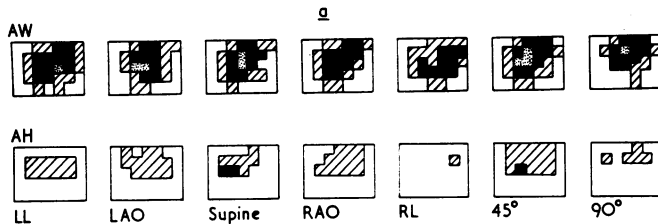
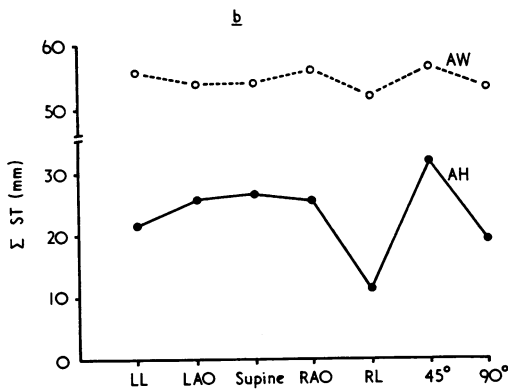


Fig. 9 (a) Isopotential maps in 2 patients with chronic ST segment elevation plotted in 7 different body postures viz left lateral, left anterior oblique, supine, right anterior oblique, right lateral, and sitting at 45° and 90°. Hatched area: over 0.1 mV ST elevation. Solid area: over 0.2 mV ST elevation. Dotted area: over 0.3 mV ST elevation. (b) Sum of ST elevations in mV in patients shown in Fig. 9a in 7 different body postures.



elevation was seen in the sitting position at 45°. The shift in electrical axis of the heart was confirmed by concomitant changes in the Q wave vector in both cases.

EFFECT OF CHANGES IN HEART RATE

Striking changes in ST elevation may occur independently of heart rate as in patients D.G. and C.R. (Fig. 5 and 6), but in patient G.C. (Fig. 7) a considerable change in heart rate, as a result of reversion from atrial fibrillation to sinus rhythm, was associated with a similar change in ST elevation. Minor fluctuations in heart rate are at times related to similar minor fluctuations in ST elevation. A transient fall in heart rate in patient D.G. (Fig. 5) at 6 hours was associated with a greater reduction in ST elevation. A transient rise in heart rate in patient C.R. (Fig. 6) at 3 hours was followed by a transient increase in ST elevation. Other fluctuations in heart rate in patient G.C. (Fig. 7) between 6 and 7 hours after onset of symptoms were associated with similar changes in magnitude of ST elevation.

MEAN AND CENTRAL LEADS

In each case computer analysis of the ST elevation has been performed in a meaned complex ('mean lead') and in a 'central' lead (showing maximum ST elevation) and a 'peripheral' lead (showing only 1 mm ST elevation).

A striking similarity was seen between ST recordings from mean and central leads, but the effect of positional change was greater in the recordings from the central lead (Fig. 8). A similarity between mean and peripheral recordings was seen in only some of the cases studied.

Discussion

We have described a new technique whereby accurate continuous analysis of praecordial ST segment change may be made. By contrast, standard methods of praecordial ST segment mapping involve hand measurements from electrocardiograms of 35, 49, or 72 leads (Reid *et al.*, 1971; Maroko *et al.*, 1972; Madias *et al.*, 1975). This is not only laborious, each individual mapping taking 10 or 15 minutes to complete, but also subject to considerable observer variation. In our 3 cases in whom a comparison was performed between hand measurement and computer analysis there were large differences between the results obtained by 5 observers, both for the absolute level of ST elevation and for the change in ST elevation over a period of 2 hours. Under these conditions the significance of sequential changes in ST elevation

must be questioned. A further major disadvantage of the hand mapping technique is the necessity for intermittent data sampling. Our system of computer analysis which can measure mean ST elevation on a beat-by-beat basis has shown that major fluctuations in ST segment change may occur over short periods of time. Patient G.C. (Fig. 9), for example, showed a reduction in ST elevation of over 70 per cent in under 1 hour. In this case 15- or 30-minute sampling would have failed to reveal major changes in ST segment shift. It is known that rapid changes in coronary perfusion and myocardial oxygen tension may occur in the early hours after infarction, yet evidence from conventional ST mapping has been contradictory; thus, it has been claimed that the rate of ST segment change is small (Madias 1975) or very variable (Reese *et al.*, 1973). Continuous analysis also reveals the occurrence of minor fluctuations in ST elevation, related to transient changes in heart rate, so providing an extremely sensitive index of underlying electrophysiological changes of myocardial ischaemia.

Conventional hand mapping, however, does make it possible to analyse sequential changes in distribution of ST segment shifts with delineation of areas of ST segment elevation and of ST segment depression. The latter are excluded from our system of computer analysis by the initial lead selection procedure. These spatial analyses can be performed at any selected time during monitoring by direct recording on paper. In the absence of complex modifications of the recording and computing system such information can only be obtained intermittently and is limited by the number of recording electrodes. Our measurements of mean ST segment elevation from a fixed number of praecordial leads may result in a certain loss of information as a result of spatial change in ST segment shifts. Voltage change itself, however, is partly dependent upon underlying spatial changes in distribution of ischaemic injury, and it is not clear what additional information may be obtained from a separate continuous spatial analysis.

Computer analysis of continuously recorded data provides highly accurate and easily reproducible data. The noise level of our system allows for a measurement of ST elevation to an accuracy of 0.004 mV. The usefulness of this information is, however, limited by the inherent limitations of praecordial mapping itself. Excellent correlation has been shown in the dog between praecordial and epicardial ST elevation (Muller *et al.*, 1975), and changes in epicardial ST elevation after acute coronary occlusion have been correlated with subsequent creatine kinase depletion (Maroko *et al.*,

1971), changes in local myocardial oxygen tension (Sayen *et al.*, 1961), blood flow (Kjekshus *et al.*, 1972), and potential gradients (Prinzmetal *et al.*, 1962). The situation in man is more complex. Infarction may not be the result of a single discrete coronary occlusion and patients are not studied until some hours after onset of symptoms. Praecordial mapping is limited to cases of anterior or anteroseptal infarction who may well have additional inferior, posterior, or lateral injury, not contributing to praecordial ST segment change. In addition the 35 chosen lead positions may not subtend the whole of the injured and potentially injured area. Other factors than ischaemia are well known to affect ST segment elevation, in particular pericarditis, conduction defects, sympathetic stimulation, temperature change, and drugs such as quinidine and digitalis.

It is likely that at least in the early hours after infarction praecordial ST elevation does reflect the electrophysiological properties of the underlying epicardium, in turn related to the degree of ischaemia. The spontaneous changes in mean ST elevation shown in the 3 illustrated cases all studied within 6 hours of the onset of chest pain probably reflect changes in severity of ischaemic injury. Our initial studies, however, have emphasised some difficulties which arise in interpreting these data. It is well known that the electrical axis of the heart with respect to the chest wall varies with body posture and indeed with respiration. It is apparent that the same is true for the ST segment. Patients A.H. and A.W. (Fig. 5) show the enormous variations in both spatial distribution of elevated ST segments and in mean ST elevation that may occur in a variety of body postures. Any interpretation of ST segment change must then be dependent upon an exact knowledge of body position. Merely sitting forward to sip a cup of tea is associated with a minor transient change in recorded mean ST elevation.

Similarly increases in heart rate are at times associated with increased ST segment elevation. This may result partly from the effect of changes in heart rate on the QT interval, affecting the upward slope of an ST segment. It is more likely, in view of the haemodynamic effect of changes in heart rate on coronary blood flow, that these directly influence the imbalance between oxygen supply and demand in the ischaemic myocardium.

The observation that changes in ST elevation in the mean lead are similar to those in the single central lead showing maximum ST elevation is of practical interest in that, if as much useful information is contained in a recording from a single lead as from 35, the computer analysis technique

may have an application in other than specialist centres. The single lead is more affected by changes in body posture (Fig. 8) than is the mean lead; the significance of measurements of ST elevation obtained from it requires further evaluation.

Interest has grown in recent years in the possibility that pharmacological or metabolic intervention after myocardial infarction may reduce the degree of myocardial ischaemic injury, and it has been suggested that measurement of praecordial ST elevation may be of use in testing the efficacy of such therapy (Pelides *et al.*, 1972). It is apparent, however, that before any such study is possible knowledge of the natural course of ST segment change is necessary. Our technique of continuous recording and analysis is a significant advance on existing techniques and is ideally suited for study of the natural history of ST segment change. It is presented as an objective research tool which is likely to be of particular value when assessing the effects of intervention after myocardial infarction.

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