

Natural history and evaluation of Q waves during acute myocardial infarction¹

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SUMMARY Serial 72 point praecordial electrocardiographic maps were recorded in 45 patients with uncomplicated acute anterior myocardial infarction. These were analysed and the serial changes in Q waves and ST segments were recorded. Cardiospecific enzyme release curves were constructed from repeated measurements of the plasma activity of the myocardial isoenzyme of creatine kinase (MB CK) during 4 days after the onset of chest pain. The praecordial maps showed that Q waves appeared in the second hour after the onset of chest pain and the development of this electrocardiographic sign was completed within 12 hours. There were complex relations between the development of Q waves, the natural history of ST segment elevation, and the release of MB CK activity. The praecordial area of ST segment elevation at 2 hours was directly related to the fully developed Q wave area at 24 hours after the onset of chest pain. The electrical activity of affected myocardium was lost before peak plasma MB CK activity.

Methods for the therapeutic salvage of active myocardium during infarction of the heart will be influenced by a clear understanding of the pattern and time course of the death of tissue during the acute phase. It is a challenge to clinical research to provide techniques that can indicate the natural history of the loss of electrically active myocardium and can assess effects of interventions during acute infarction. By praecordial mapping it is possible to make repeatable noninvasive measurements of changes in a number of well-known electrocardiographic signs. The purpose of this paper is to show the natural history of the development of Q waves in praecordial maps from a group of patients with uncomplicated acute anterior myocardial infarction. The temporal relations between this electrocardiographic sign, the sum of all the praecordial ST segment elevations (Σ ST), and plasma activity of the myocardial isoenzyme of creatine kinase (MB CK) after the onset of chest pain are described; the significance of these findings in relation to the progressive regional loss of electrically active myocardium is also discussed.

Methods

Studies were made on 45 patients (37 men and 8 women, aged between 45 and 77 years, mean 59 years) admitted to the Coronary Care Unit (CCU) at the Hammersmith Hospital with a clinical diagnosis of acute anterior myocardial infarction. All developed electrocardiographic evidence of acute myocardial infarction and all had a diagnostic rise in serum levels of cardiac enzymes while in the coronary care unit. None of these patients developed the following important complications while in the CCU: (a) Recurrent chest pain lasting more than 10 minutes that was separate from the initial episode, (b) clinical or chest x-ray evidence of congestive heart failure or pulmonary oedema, or (c) cardiac rhythm disturbances other than isolated unifocal ventricular ectopic beats.

Eight patients were admitted in the first hour, 10 in the second hour, 19 between 2 and 6 hours, and 8 between 6 and 12 hours after the onset of chest pain. In each patient praecordial electrocardiographic maps were recorded on admission, 1 hour later, 4-hourly for 12 hours, and daily thereafter, and additionally if the clinical condition changed. Praecordial mapping at these time intervals made it possible to observe and measure changes in the electrocardiograms in the groups of patients avail-

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able for study in the first hour, at 2, 6, 12, and 24 hours, and at 2 and 4 days after the onset of chest pain.

Electrocardiograms were recorded using a 3-channel direct writing ink jet Mingograf (Eliam-Schonander). The gain employed was 10 mm for 1 mV and the paper speed was 25 mm per second. The electrodes were the Welsh suction type with a contact diameter of 1 cm. Electrocardiograms were recorded from 72 points distributed evenly over the praecordium as described by Reid *et al.* (1971). Praecordial maps were always recorded with the patients at rest, reclining at 45°, and all values recorded were the mean of measurements made from 4 complexes at end-expiration; it has been shown that measurements of ST segment elevation, and of R, S, and Q waves are reproducible with a ± 5 per cent variation if the effects of posture and phase of respiration are taken into consideration (Selwyn and Shillingford, 1977).

The appearance of QRS widening, bundle-branch block, or pathological axis changes can alter the interpretation of electrocardiographic signs. Patients whose electrocardiogram showed QRS widening beyond 110 ms or QRS axis beyond -30° or $+130^\circ$ in the frontal plane were therefore excluded from this study.

The TP segment was taken as the isoelectric line but when this was difficult to locate because of tachycardia the PQ segment was used. ST segment elevation was measured in millimetres to the nearest 0.5 mm at 0.06 s after the nadir of the S wave. Pathological Q waves were identified using the Minnesota code (Blackburn *et al.*, 1960; Rose and Blackburn, 1968) and measured in mm. The surface maps produced in this way from patients after acute myocardial infarction showed areas of Q waves and of QR or Qr waves.

Venous blood samples were taken from 27 of the patients, at 3-hourly intervals for the first 25 hours after the onset of chest pain, then 6 hourly for 4 days, placed in lithium heparin tubes, and centrifuged at 2000 g for 10 minutes. Total plasma CK activity was measured spectrophotometrically (using Cecil 272 system spectrophotometer, Cecil Instruments, Cambridge) by the method of Oliver (1955) as modified by Hearse *et al.* (1973). Using this method the upper limit of plasma CK for healthy subjects is 50 mU/ml (Ogunro *et al.*, 1976). Samples with activity more than 250 mU/ml were diluted before repeat determination of total CK. In order to minimise the dilution-activation effect observed with plasma CK, heat inactivated plasma was employed as diluent (Graig *et al.*, 1967). Isoenzymes were separated by electrophoresis on agarose gels and quantified in aqueous solution by fluorimetry

(Ogunro *et al.*, 1976). MB CK isoenzyme release curves were constructed by measuring the plasma MB CK activity (mU/ml) at the stated time intervals after the onset of chest pain.

Praecordial maps from each patient at each time interval were used to calculate the sum of all the Q waves measured in mm (ΣQ) and the number of positions showing Q waves in the 72 point map (Q wave area). Each map was also used to calculate the sum of all the ST segment elevations in mm (ΣST) and the number of praecordial positions showing ST elevation of more than 2 mm (area of praecordial ST segment elevation). The significance of changes in ST segments and Q waves was examined by analysis of variance during 3 time intervals after the onset of chest pain (2 to 6 hours, 6 to 12 hours, and 12 to 24 hours).

Results

The 18 patients admitted within two hours of the onset of symptoms showed abnormal areas of R wave loss and Q waves in the first 72 point praecordial electrocardiogram. The areas showing R wave loss and appearance of Q waves increased to

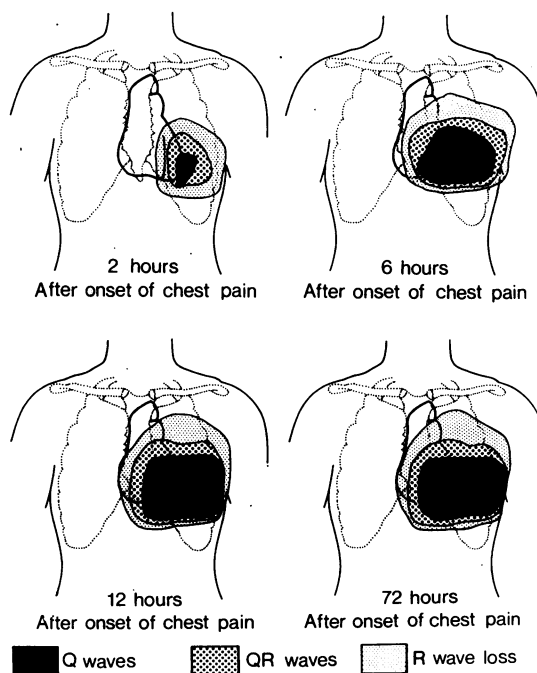


Fig. 1 Praecordial mapping of Q, QR, and R wave loss in uncomplicated anterior myocardial infarction. This typical example shows Q waves within 2 hours and complete development of the area of Q and QR waves within 12 hours from the onset of chest pain.

full development up to 12 hours after the onset of chest pain. The changes in these electrocardiographic signs between 12 and 24 hours were not significant (Selwyn and Shillingford, 1977). A typical example is shown in Fig. 1.

Q waves were detected in the praecordial maps during the second hour after the onset of symptoms. The praecordial sum of Q waves in mm (ΣQ) and the number of positions showing Q waves increased to full development within 12 hours. The analysis of variance showed that the changes between 2 and 6 hours, and 6 and 12 hours were highly significant ($P < 0.001$). However the changes between 12 and 24 hours were not significant. This is shown in Fig. 2a and 3.

Praecordial ST segment elevation (ΣST) reached a peak within the first hour after the onset of

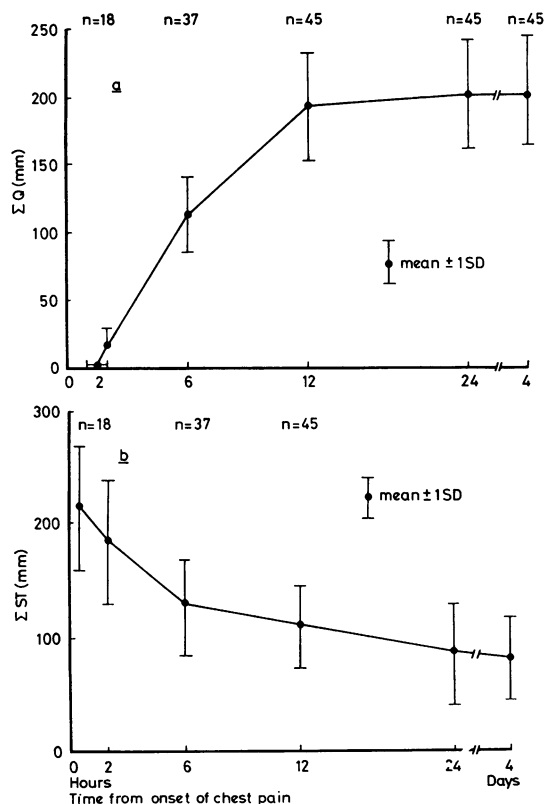


Fig. 2 (a) The sum of the praecordial Q waves in mm (ΣQ) during uncomplicated anterior myocardial infarction. Q waves appear in the second hour and ΣQ rapidly increases to completion within 12 hours of the onset of pain. (b) The sum of praecordial ST elevation (ΣST) during uncomplicated anterior myocardial infarction.

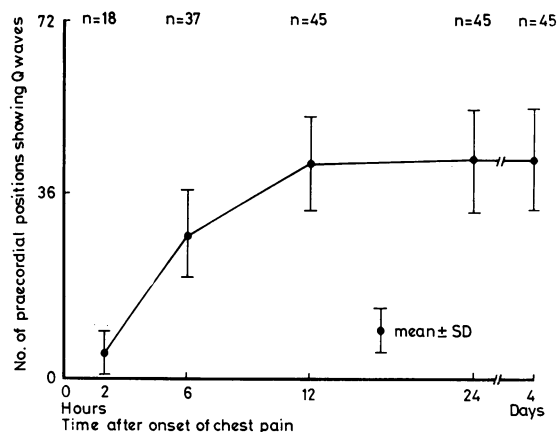


Fig. 3 Q wave area in uncomplicated anterior myocardial infarction develops rapidly to completion within 12 hours of the onset of pain.

symptoms (Fig. 2b), and the analysis of variance showed that the decrease in ΣST that followed over 12 hours was highly significant ($P < 0.001$).

Plasma MB CK activity in mU/ml was first detected between 6 and 10 hours after the onset of chest pain in 27 patients. The peak plasma MB CK activity was detected at 19.5 ± 1.7 hours (mean ± 1 SD). In these 27 patients the praecordial electrocardiographic changes of R wave loss and full development of Q waves were complete before this enzyme activity had reached a peak. There was a significant relation between the praecordial sum of Q waves (ΣQ) at 24 hours and the praecordial sum of ST segment elevation (ΣST) at 45 minutes after the onset of chest pain. ΣQ at 24 hours was not related to ΣST at any other time (Fig. 4).

The number of praecordial positions showing ST segment elevation greater than 2 mm at 45 minutes was greater than the number of positions showing Q waves at 24 hours in the 8 patients studied. There were 33 patients available for study in the third hour after the onset of pain. In this group the number of praecordial positions showing ST segment elevation was more directly related to the number of positions showing Q waves at 24 hours (Fig. 5). The decrease in ST segment elevation during the natural history of this electrocardiographic sign resulted in the praecordial area of ST elevation at 12 hours being smaller than the Q wave area at 24 hours.

Discussion

It has long been known that the loss of R waves and the development of Q waves over the ischaemic area

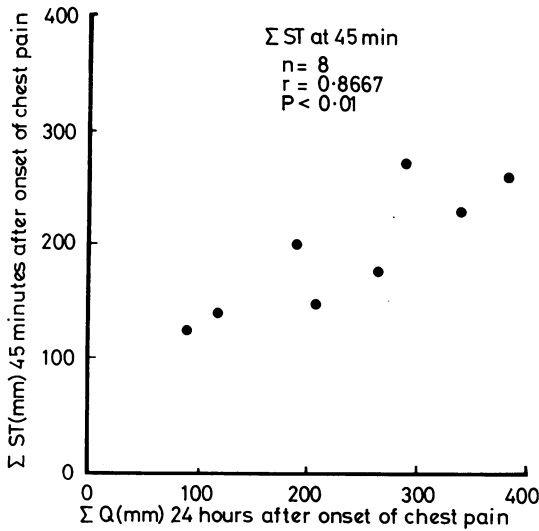


Fig. 4 There was a weak relation between praecordial Σ ST segment elevation at 45 minutes and Σ Q at 24 hours after the onset of pain ($n = 8$). (There was no relation between praecordial Σ ST segment elevation at 6 hours ($n = 37$) or 24 hours ($n = 45$) Σ Q at 24 hours.)

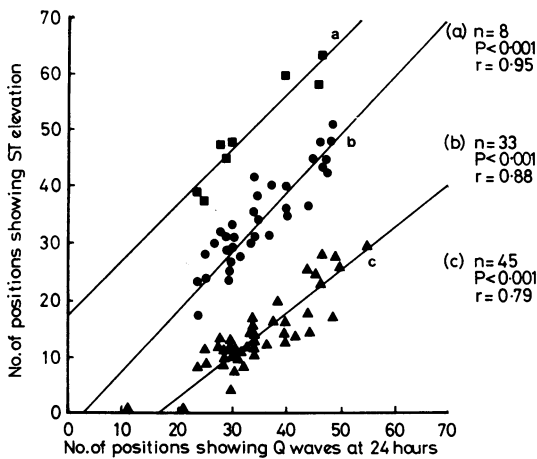


Fig. 5 Area of praecordial ST segment elevation at (a) 45 minutes, (b) 2 hours, and (c) 12 hours, plotted against Q wave area at 24 hours. The area of praecordial ST segment elevation at 2 hours was equal in size and related to the final stable Q wave area at 24 hours after the onset of chest pain.

of the myocardium represent death of the muscle cells (Johnston *et al.*, 1935; Wilson *et al.*, 1935; Myers *et al.*, 1948; Shaw *et al.*, 1954). Previous studies have suggested that it is possible to outline

on praecordial surface maps the temporal changes in R/S ratio as well as R and Q wave amplitude in acute myocardial infarction, if posture and phase of respiration during recording remain constant (Selwyn and Shillingford, 1977; Selwyn *et al.*, 1977). These maps show a different time course of development of Q waves from that of the ST segment changes. In the present group of patients with uncomplicated anterior infarction, Q waves could be found in the praecordial maps during the second hour after the onset of chest pain and the development of Σ Q and Q wave area proceeded rapidly to a maximum within 12 hours. There was a weak but significant relation between Σ ST at 45 minutes and Σ Q at 24 hours. When the praecordial Q wave area at 24 hours after the onset of chest pain was compared with the area of praecordial ST segment elevation at different times, it was found that only at 45 minutes was the area of ST elevation greater than the Q wave area; 2 hours after the onset of pain the praecordial areas of these two electrocardiographic signs were equal in size and directly related.

Plasma MB CK activity was detected at a time when most of the praecordial R wave loss and Q waves development had already occurred. The peak release of MB CK activity occurred long after the complete development of Q waves in the praecordial maps. These time intervals suggest a delay between the regional loss of electrically active myocardium and the release of MB CK activity into the plasma, and might suggest that interventions designed to limit infarct size and given during the release of MB CK activity will take effect at a time when most of the loss of electrically active myocardium has happened (Shell *et al.*, 1971; Sobel *et al.*, 1972).

Muller *et al.* (1975) showed a significant temporal relation between changes in electrocardiographic signs observed on the epicardium and over the praecordium. However, the extent of these changes must depend on the orientation of the infarct in relation to the electrode, the resistance of the intervening tissues, and the effects of surrounding normal myocardium. The geometric factors influencing the projection of ST segment and Q wave changes from the heart onto the praecordial map will be the same for each patient.

In conclusion, this study has shown that (a) in a group of patients with uncomplicated anterior myocardial infarction serial praecordial electrocardiographic maps provided a noninvasive and repeatable assessment of the onset and development of Q waves, (b) the pattern and time course of these electrocardiographic changes in this group suggested that the regional loss of electrically active myocardium started in the second hour and was

completed between 6 and 12 hours after the onset of chest pain, (c) the praecordial area of ST segment elevation at 2 hours was related to the stable Q wave area at 24 hours so that the effects of interventions given at 2 hours on these electrocardiographic signs may provide useful information about the relation between acute regional myocardial ischaemia and cell death, and (d) the time course for the regional loss of electrically active myocardium suggests that if interventions aimed at salvage are to be most effective they should be given before MB CK activity is detected in the plasma.

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