Electrocardiographic Precordial Mapping in Anterior Myocardial Infarction

The Critical Period for Interventions as Exemplified by Methylprednisolone

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SUMMARY Serial 72-point precordial mapping of the ECG has been recorded to describe the natural history of changes in the precordial areas of ST segment elevation and the development of Q waves in 51 patients with acute uncomplicated anterior myocardial infarction. Eight patients have been studied in the same way but received 25 mg/kg of methylprednisolone sodium succinate as a single intravenous injection within 6 hours from the onset of chest pain.

There was a linear relationship between the stable precordial area of Q waves at 24 hours and the rapidly changing precordial areas of ST segment elevation at 2–3 hours, 5–6 hours and 12 hours after the onset of pain in the untreated patients. When methylprednisolone was given, the treated patients developed a smaller precordial area of Q waves at 24 hours than was predicted from the precordial area of ST elevation recorded before the drug was given.

This study has introduced a technique that can provide a qualitative assessment of the relationship between ECG evidence of ischemia and infarction in each patient.

A NONINVASIVE TECHNIQUE that can assess the salvage of active myocardium during acute infarction of the heart is needed.1 Serial recordings of the ECG as 72-point precordial maps can describe the relationship between the early signs of myocardial ischemia (ST segment elevation) and later signs of cell death (pathological Q waves) in dogs and patients suffering from anterior myocardial infarction.2 4 When a drug is given after recording the precordial area of ST segment elevation, this technique might be used to investigate the progress from myocardial ischemia to cell death following the onset of chest pain.2

Experimental work has shown that methylprednisolone sodium succinate can delay and diminish the extent of tissue damage during acute myocardial ischemia.5 6 However, clinical studies have produced contradictory results.7 8

The purpose of this study is to examine progressive changes of ECG signs in 72-point precordial maps in a group of patients with anterior myocardial infarction and to assess a similar group who received methylprednisolone within 6 hours from the onset of chest pain.

Methods

Eighty-five consecutive patients (71 males and 14 females, mean age 63 years) were admitted to the coronary care unit at the Hammersmith Hospital with a clinical diagnosis of acute anterior myocardial infarction. All of these patients had ECG evidence of acute anterior infarction in the standard 12 lead ECG and all had a diagnostic rise in plasma enzyme activity.9 The diagnosis of anterior myocardial infarction in the 12 lead ECG depended on the appearance of pathological Q waves in one or more of the precordial unipolar chest leads V1–V6. Fifty-one of these patients (42 males and nine females, age range 42–77 years, mean 57 years) did not manifest any of the following important features:

1) recurrent chest pain lasting more than 10 minutes that was separate from the initial episode;
2) clinical or radiological evidence of congestive heart failure or pulmonary edema;
3) cardiac rhythm disturbances other than unifocal isolated ventricular ectopic beats;
4) changes in daily serum electrolyte estimations that were outside the normal range; and
5) a past history or past ECG evidence of myocardial infarction.

None of these patients were on any treatment before admission to the hospital.

An additional eight consecutive patients were admitted to the coronary care unit with a diagnosis of anterior myocardial infarction (seven males and one female, age range 47–59 years). These patients experienced an uncomplicated course, as was described for the control group. Three patients were admitted during the second hour, three during the fourth hour and two during the sixth hour following the onset of chest pain. These eight patients received a single intravenous injection of methylprednisolone sodium succinate (25 mg/kg) over 30 minutes after recording a 72-point precordial ECG map. This was carried out within 30 minutes of admission. All these patients developed ECG evidence of anterior myocardial infarction in the 12 lead ECG and all had a diagnostic
rise in plasma enzyme activity. No other therapeutic interventions were given in either group other than diamorphine.

Precordial ECG maps were recorded on admission, again at 1 hour, then four hourly for 12 hours and daily thereafter. At least three precordial maps were recorded during the first hour in those patients admitted within 60 minutes from the onset of chest pain. The ECG calibration throughout was 10 mm for 1 mV and the paper speed was 25 mm/sec using a direct writing three channel ink jet Mingograf (Elema-Schonander). At each of the 72 positions three unipolar complexes were analyzed and a mean value for ST segment elevation was used. Each position was recorded before and during a 5-second period with respiration held at end expiration. When patients were unable to cooperate, the mean ST segment elevation throughout a complete respiratory cycle was calculated in millimeters. The ST segment elevation was measured using the TP segment as the isoelectric line, or the PQ segment when the TP segment was difficult to locate. The ST segment elevation was measured in millimeters to the nearest 0.5 mm at 0.06 seconds after the end of the QRS complex. Pathological Q waves were identified using the criteria of the Minnesota Code. A pathological Q wave was identified when a negative deflection in the QRS complex preceded an R wave and was ≥ 25% of that R wave and/or was ≥ 0.04 seconds in duration. A totally negative QRS deflection was also identified as a pathological Q wave. Precordial ECG maps were recorded from 72 positions using Welsh suction electrodes with a contact diameter of 1 cm with each patient at rest and reclining at 45°. Patients were excluded if on admission or at any time during the study the ECG showed changes in the mean frontal axis beyond −30° and +120° or QRS widening to or beyond 110 msec. This was done in order to identify the loss of R and development of Q waves that were directly associated with the infarction.

Precordial ST segment elevation was recorded as pathological when ≥ 2 mm. The number of precordial positions showing significant ST segment elevation in each precordial map was recorded as such in the figures and calculations and referred to in the text as precordial area ST. The number of precordial positions showing pathological Q waves were treated in the same way and referred to as precordial area Q.

The progressive changes in the precordial areas of ST and Q were examined by analysis of variance. The relationship between these ECG signs was examined using linear regression analysis. A slope and 90% confidence limits were calculated for the observations in the relationship between precordial area ST at the three different times (fig. 1) and precordial area Q at 24 hours after the onset of chest pain.

Results

Figure 2 shows the natural history of precordial ST segment elevation and development of Q waves in a typical control patient. The development of pathological ST segment elevation in both groups is shown in figure 3A. The precordial area of this ECG sign reached a maximum within 1 hour after the onset of chest pain. This was followed by a consistent decrease in precordial area ST during the next two days. The changes in precordial area ST in both groups during the first 24 hours following the onset of chest pain were highly significant (P < 0.001 in both groups for the changes from 1–6 hours and from 6–24 hours).

Figure 3B shows the development of the precordial area of Q waves during anterior infarction in both groups. Q waves were detected in the second hour and the precordial areas developed rapidly up to 12 hours after the onset of chest pain. The analysis of variance showed that the changes in area Q after 12 hours were not significant.

There was a linear relationship between: 1) the precordial area of ST segment elevation at 2–3 hours and the precordial area of Q at 24 hours (Y = 914X + 3.26, r = 0.91, n = 33), 2) the precordial area of ST at 4–6 hours and precordial area Q at 24 hours (Y = 1.03X + 10, r = 0.92, n = 51), and 3) the precordial area of ST at 12 hours and precordial area Q at 24 hours (Y = 1.06X + 19.4, r = 0.92, n = 51). Figures 3A, B and C show these linear relationships between precordial area ST at the different times and the final precordial area of Q waves at 24 hours after the onset of chest pain. The 90% confidence limits for the observations were calculated. Those patients admitted within 2 hours after the onset of chest pain and who received methylprednisolone showed that for any value of precordial area ST at 2–3 hours, they developed a smaller precordial area of Q waves at 24 hours than the untreated group. Those patients admitted during the fourth to sixth hours also showed results outside the 90% confidence limits for the untreated group with smaller precordial areas of Q waves at 24 hours (fig. 1).

Discussion

This study used serial 72-point precordial mapping of the ECG in order to record the natural history of two well-known ECG signs during uncomplicated anterior myocardial infarction.

The natural history of precordial ST segment elevation in these patients showed maximal changes in the first hour and consistent decreases thereafter. In contrast, the precordial area of Q waves appeared in the second hour and showed that the loss of electrically active myocardium progressed rapidly up to 12 hours after the onset of pain. There is considerable controversy over the interpretation of ST segment elevation during the course of acute myocardial infarction. This ECG sign originates from complex electrophysiological events that involve the ischemic and surrounding normal myocardium. The projection onto the chest is influenced by the highly variable relationships between the precordial ECG positions, the heart, the infarcting myocardium and intervening tissues.

The regional epicardial and precordial development
Figure 1. There was a linear relationship between the precordial area of ST segment elevation at three different times and the precordial area of Q waves at 24 hours after the onset of chest pain in the untreated group (A, B, and C). The relationship between early myocardial ischemia (precordial area ST) and later cell death (precordial area Q) was altered beyond the 90% confidence limits of the untreated group in eight patients who received intravenous methylprednisolone within 6 hours. This suggests smaller than predicted precordial areas of Q waves and myocardial salvage.
of Q waves in acute anterior myocardial infarction almost certainly represents the progressive loss of electrically active myocardium. Experimental and clinicopathological studies have shown evidence to substantiate the relationship between the development of Q waves and the death of subjacent viable myocardium. An analysis of the individual natural history of these ECG signs showed that the decreasing precordial areas of ST segment elevation at 2–3 hours were approximately equal to the stable precordial areas of Q waves at 24 hours after the onset of chest pain in the untreated patients. In the first 2 hours, there were precordial positions that showed pathological ST segment elevation that did not subsequently develop Q waves. The complete natural history of these precordial ECG signs must be followed in order to describe the relationship between early signs of ischemia and later signs of cell death. Possibly, the factors that influence the projection of ST segment elevation and Q waves are the same in each patient. If this is so, the early precordial manifestations of ischemia, i.e., precordial area of ST segment elevation, can be used to predict the later precordial signs of cell death, i.e., precordial area of Q waves at 24 hours. The patients selected for this study all had anterior infarction diagnosed in the routine 12 lead ECG in the conventional way. This was done because previous research has shown that there is a consistent and more direct association between precordial ECG signs of anterior infarction and the underlying pathology. The same assumptions cannot be made for diaphragmatic and posterior wall infarction. This study has shown a linear relationship between these ECG signs at the stated times and supports the above hypothesis. Any interventions given soon after recording the precordial area ST might be assessed by measuring their effects on the relationship between early ischemia and later cell death (precordial area of Q at 24 hours) in each patient.

This study investigated eight patients who received methylprednisolone at different times after the onset of chest pain. There are encouraging but contradictory experimental clinical studies that suggest that this drug might be beneficial during acute myocardial infarction. This study and others have shown that the regional precordial loss of R and development of Q waves progress rapidly and mostly in the first 6–12
Figure 3.  

A) The sequential changes in the precordial area of ST segment elevation in the treated and untreated groups. The three treated patients (●) studied in the first hour are shown and the number of patients in each group is indicated above each standard deviation bar. Eight treated patients had no significant ST elevation at 72 hours. B) shows the rapid precordial development of Q waves that occurred in the 12 hours after the onset of chest pain in the treated and untreated patient groups. The 10 untreated and three treated patients studied at 1 hour showed no pathological Q waves. The six treated patients studied at 2 hours also showed no Q waves.
hours following the onset of chest pain. Those patients who received the drug within 6 hours showed smaller than predicted precordial areas of Q waves, suggesting myocardial salvage. Larger numbers of treated patients in a randomized trial are essential before this drug can be recommended for use in man during acute myocardial infarction.

In addition, there is clinical evidence to suggest that the more prolonged use of methylprednisolone may alter the inflammatory response and delay healing. This study has tried to avoid these unproven risks and associations by giving a single dose within 6 hours from the onset of chest pain and during the loss of electrically active myocardium.

In conclusion, serial precordial mapping of the ECG in patients with uncomplicated acute anterior myocardial infarction can demonstrate the individual natural history of precordial areas of ST segment elevation and the development of precordial areas of Q waves that follow the onset of chest pain. These ECG signs have shown that in each patient there was a useful relationship between the early ECG signs of myocardial ischemia and later ECG signs of cell death. The technique and findings in this study are probably only applicable to patients with anterior myocardial infarction. However, the time course for the development of precordial Q waves might suggest that active myocardium is lost rapidly, within 6–12 hours from the onset of chest pain.

References

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