Myocardial infarction and thrombolysis

Electrocardiographic short term and long term results using precordial mapping

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SUMMARY In a consecutive series of 56 patients with acute myocardial infarction, ST segment depression and elevation in the electrocardiographic limb leads I, II, and III were summated for each patient before and immediately after intracoronary streptokinase infusion and the results compared with the angiographic findings. Forty three patients had angiographically confirmed reperfusion of an initially occluded vessel and showed a significant decrease in summated ST shift. The ST segment changes in the limb leads virtually returned to normal in all 43 patients, and in most, inverted T waves developed. Thrombolysis was unsuccessful in 10 patients, and the infarct related coronary artery was already patent in three. When these two groups are combined, all 13 patients without reperfusion showed no significant change in summated ST segment shift. During percutaneous transluminal angioplasty inflation of the balloon in the vessel that was previously occluded simulated reocclusion and was followed by new ST elevation if the artery supplied viable myocardium.

In a further consecutive study of 54 patients with anterior myocardial infarction, the precordial R waves and Q waves were studied over the four to six months following infarction using a standardised 48 electrode mapping system. All patients underwent a repeat angiogram after four to six months. In 36 patients the infarct related vessel was patent. They showed a significant mean increase in summated precordial R wave amplitude and a reduction in the mean number of precordial leads without R waves. In 18 patients with unsuccessful thrombolysis or reocclusion there was a further reduction in mean summated R wave amplitude and an increased number of precordial leads not showing R waves.

Precordial R wave mapping seems to be a valuable non-invasive method of assessing the salvage of myocardium after reperfusion and the damage caused by reocclusion. Loss of R waves in the acute phase of myocardial infarction does not necessarily mean an irreversibly damaged myocardium.

Electrocardiography is the most important technique for diagnosing acute myocardial infarction. ST segment elevation occurs within a few seconds of complete occlusion of a coronary artery. During the next six to eight hours R waves are lost or their amplitude reduced and pathological Q waves develop.^{1 2}

A rapid decrease in ST elevation during intravenous streptokinase infusion in patients with acute myocardial infarction was described in the 1960s,³

and changes in the standard electrocardiographic leads after intracoronary streptokinase infusion have recently been reported.^{4 5} There are, however, no detailed studies that have used electrocardiographic mapping to study the effectiveness of reperfusion. We therefore studied the ST segment changes during angiographically documented reperfusion and the Q and R wave changes in the subacute and chronic stages of myocardial infarction.

Patients and methods

The patients in the present study were all admitted to our hospital within eight hours of the onset of chest pain, had electrocardiographic changes consistent

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with acute myocardial infarction (ST elevation of ≥ 0.2 mV in the limb leads or of ≥ 0.3 mV in the chest leads), and had persistent chest pain. They were treated with an intracoronary infusion of streptokinase at a dose of 3000 units per minute for 60 minutes. Coronary angiography was performed before, every 20 minutes during, and at the end of the infusion.

The protocol was approved by the local ethical committee, and all patients gave informed consent for the study.

ST SEGMENT CHANGES IN THE ACUTE STAGE (Group 1)

In a consecutive series of 56 patients (group 1) (47 male, nine female; age range 41–81 years) 21 had anterior and 35 inferior infarcts. We summated the ST segment elevation and depression (ST shift) in the limb leads I, II, and III before and after streptokinase infusion and compared the results with the angiographic findings. In 34 patients online monitoring of the ST segment using up to eight precordial leads was performed during the 48 hours after streptokinase infusion in order to detect recurrent ischaemia.⁶ In six patients we used a precordial online mapping system to study the ST segment changes during balloon inflation while performing percutaneous transluminal angioplasty of the remaining stenosis of an infarct related vessel after successful reperfusion.

CHANGES IN Q AND R WAVES AT SIX MONTHS (Group 2)

In a second series of 54 consecutive patients (group 2) with anterior myocardial infarction (41 male, 13 female; age range 27–79 years), which did not include the patients in group 1, standardised 48 electrode precordial mapping was performed immediately after streptokinase infusion, at 72 hours, at three weeks, and at four to six months, when repeat coronary angiography was also performed.

The 54 patients in group 2 were divided into four groups according to the angiographic findings at follow up coronary angiography four to six months after

infarction. Group A comprised 11 patients in whom reperfusion occurred spontaneously before streptokinase infusion and the infarct related vessel was patent four to six months later; group B comprised 25 patients achieving successful thrombolysis in whom the infarct related vessel was still patent four to six months later; group C comprised eight patients with reoccluded vessels at four to six months; and group D comprised 10 patients in whom thrombolysis was unsuccessful.

STATISTICAL ANALYSIS

Statistical analyses were performed using Student's *t* test. Values are expressed as mean (standard deviation).

Results

GROUP 1

In 43 of the 56 patients the infarct related vessel was occluded at the start of the streptokinase infusion, thrombolysis was successful, and reperfusion was achieved. In these patients there was a significant decrease in mean summated ST shift from 0.68(0.32) mV to 0.09(0.12) mV ($p < 0.001$). Figure 1 shows an example of an electrocardiogram that had virtually returned to normal a few minutes after reperfusion.

In 10 patients either the occluded vessel could not be reperfused or inadequate reperfusion was achieved. In three patients the vessel was already patent before the start of the streptokinase infusion. In this combined group of 13 patients there was no significant change in mean ST shift during streptokinase infusion (mean ST shift before infusion 0.44(0.29) mV and after infusion 0.44(0.36) mV) (Fig. 2). All patients achieving successful reperfusion showed a reduction in mean ST shift of $> 55\%$ within one hour.

During percutaneous transluminal coronary angioplasty performed three days after successful thrombolysis in four of these six patients inflation of the

Fig. 1 Electrocardiograms showing rapid "improvement" of ST-T changes after reperfusion in a 45 year old man in whom a streptokinase infusion into the occluded right coronary artery was started four and a half hours after the onset of chest pain.

Fig. 2 ST segment shift in leads I, II, and III before and one hour after streptokinase infusion in (a) 43 patients with successful thrombolysis and reperfusion and (b) 10 patients with unsuccessful thrombolysis (● inferior, ▲ anterior infarction) and three with the infarct related vessel already patent at the start of infusion (○). Bars represent mean (SD).

balloon in the remaining stenosis of the infarct related vessel simulated reocclusion and was accompanied by new elevation of the ST segment in the precordial

leads around the centre of the initial ischaemia (Fig. 3). In two patients no ST segment changes were seen during angioplasty. Although initial reperfusion had been successful and both patients had shown a reduction in mean ST shift during the first hour, they had both had subsequent episodes of recurrent chest pain and ST elevation, repeated estimations of plasma creatine kinase MB activity had shown a delayed release pattern, and left ventricular angiography showed severely depressed regional wall motion.

GROUP 2

Groups A and B showed a moderate but significant increase in precordial R wave amplitude between the initial admission and repeat angiography, whereas patients in groups C and D showed a further loss of amplitude (Table). A similar trend could be seen with respect to the number of precordial leads showing a complete loss of R waves: in groups A and B the number of leads decreased, but increased or remained the same in groups C and D. Four to six months after infarction group A had the fewest precordial leads with complete loss of R waves (mean 12.3) and group C the most (mean 31) (Table). The increase in R wave amplitude in groups A and B could

Fig. 3 Electrocardiograms and angiograms showing simulation of reocclusion during inflation of the balloon causing new ST elevation in leads II and III in a patient with inferior myocardial infarction and successful thrombolysis two days previously.

Table Mean (SD) of summated R wave amplitude (mV) and mean (SD) number of leads showing no R waves in a 48 electrode precordial map recorded immediately after intracoronary streptokinase infusion and 4–6 months later in group 2 patients with an initially patent infarct related vessel (group A), successful thrombolysis with vessel remaining patent (group B), unsuccessful thrombolysis (group D), and successful thrombolysis with subsequent reocclusion (group C)

Subgroup	No of patients	Summated R wave amplitude (mV)				Mean (SD) No of leads showing no R waves			
		After infusion	After 4–6 mnth	Change	p value	After infusion	After 4–6 mnth	Change	p value
A	11	18.1 (5.4)	23.2 (7.0)	+5.1 (7.5)	<0.04	14.7 (10.8)	12.3 (7.5)	–2.4 (4.8)	NS
B	25	12.4 (10.9)	16.2 (11.2)	+3.9 (5.0)	<0.006	27.9 (13.1)	21.2 (13.7)	–6.6 (8.9)	<0.001
C	8	14.0 (13.0)	9.8 (11.0)	–4.3 (2.8)	<0.003	26.5 (12.2)	31.0 (12.2)	+4.3 (3.2)	<0.007
D	10	11.8 (12.8)	10.7 (12.7)	–1.1 (4.8)	NS	30.4 (12.2)	30.7 (12.6)	+0.3 (10.7)	NS

Fig. 4 Diagrams showing (a) increase in R wave amplitude (related to position of electrode and mean value in each of the four groups) during the 4–6 months after infarction in patients with initially patent infarct related vessel (group A) and those with successful thrombolysis in whom the vessel remained patent (group B) and (b) decrease in R wave amplitude in patients with unsuccessful thrombolysis (group D) and those with reocclusion of the infarct related vessel (group C).

be seen in those electrodes having the highest ST segment elevation at the time of admission, whereas in patients with reocclusion (group C) the further loss of R wave amplitude mainly affected the border zone of initial precordial ischaemia (Fig. 4b).

Discussion

In this study a decrease in ST elevation and ST depression in the electrocardiographic limb leads occurred within one hour after reperfusion of the infarct related vessel in patients with anterior or inferior myocardial infarction treated with intracoronary streptokinase. In patients with unsuccessful thrombolysis no change in summated ST shift occurred during streptokinase infusion. Similar results have been reported in patients with anterior myocardial infarction using leads V1–V6.^{4,5,7} This rapid reduction in summated ST depression coincides with the relief of pain. Although it might be explained by rapid death of the infarct area, a reduction in ischaemia is more likely since reocclusion or simulated reocclusion by balloon inflation at the same site induced a new ST elevation (Fig. 3).

This simple electrocardiographic criterion should be useful for judging the likelihood and timing of reperfusion in patients treated with intravenous streptokinase.

It is generally accepted that the loss of R waves and the appearance of new Q waves after acute myocardial infarction represent loss of myocardium.^{8–11} The time course of these changes has been investigated in detail during the first 48 hours after the onset of chest pain.^{2,10} During this period a recovery of R waves and a regression of Q waves was found in <10% of conservatively treated patients.² Although transient Q waves have been reported in patients with Prinzmetal's angina, during myocardial ischaemia or coronary arteriography, or after coronary artery bypass surgery,^{12–16} they are rare after myocardial infarction and are confined to patients with small infarcts.^{17,18} The mechanism is unknown; some authors believe that shrinking of the scar and hypertrophy of the myocytes in the border zones is the most likely reason.^{18,19}

In this study patients with unsuccessful throm-

bolysis (group D) had no significant change in precordial Q waves or R waves when the acute and chronic stages are compared, whereas an extension of the electrocardiographic infarct area was seen in those with angiographically documented reocclusion (group C). Although the numbers of Q waves and the precordial R wave amplitudes immediately after streptokinase infusion were nearly the same in groups B and C, patients in whom reperfusion was maintained showed a gradual but significant reduction in the number of Q waves and an increase in R wave amplitude in the centre of the infarct during the weeks after reperfusion. Patients with spontaneous reperfusion (group A) had the smallest infarct areas and no significant change in Q waves during long term follow up. Thus shrinking of the scar and hypertrophy of the healthy myocardium cannot be the only explanation for the increase in R wave amplitude. The significant increase in R wave amplitude and the reduction in the number of pathological Q waves after reperfusion probably means salvage of jeopardised myocardium. Obviously, the electrical recovery of the ischaemic cell takes more than a few days and coincides with the recovery of function.²⁰

Since there is a significant difference in these electrocardiographic findings between patients in whom the infarct related vessel could be reperfused within the first four to six hours and those in whom reperfusion was unsuccessful, precordial mapping should be a useful non-invasive method for judging the long term effects of reperfusion. These results will need to be confirmed in a prospective randomised study.

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