An Investigation in Patients with Previous Myocardial Infarction Who Present with Chest Pain

ANDREW PETER SELWYN, M.B.CH.B., KIM FOX, M.B.CH., GERALD FORSE, TIMOTHY PRATT, M.SC., AND ROBERT STEINER, M.D.

SUMMARY Thirty-five patients who presented with chest pain underwent mapping of the ECG with exercise and angiocardiography. Krypton-81m was used to assess regional myocardial perfusion before, during and after atrial pacing.

Twelve of the 35 patients had negative exercise tests. Eight of these 12 had normal coronary arteries and four had \( \leq 50\% \) stenosis of at least one major coronary artery. All 12 patients had uniform increases in regional myocardial perfusion \( (98 \pm 14.0\%) \) during atrial pacing. Thirteen of the 35 patients had a history of myocardial infarction and precordial areas of Q waves. During exercise, all 13 patients complained of chest pain and showed precordial areas of both ST-segment elevation and depression. These 13 patients had \( \geq 70\% \) stenosis of at least one major coronary artery. Myocardial blood flow studies showed fixed defects of perfusion corresponding to the Q waves and ST-segment elevation. In addition, there were separate transient decreases of regional myocardial perfusion \( (70 \pm 9.0\%) \) during atrial pacing corresponding to ST-segment depression and chest pain. Ten of the 35 patients had a history of myocardial infarction and precordial areas of Q waves. During exercise, only two of these 10 complained of chest discomfort and all showed precordial areas of significant ST-segment elevation alone. All these patients had \( \geq 70\% \) stenosis of one or more major coronary arteries. Myocardial blood flow studies showed fixed defects of perfusion that corresponded to Q waves. These areas showed no changes during atrial pacing. All the patients showed at least one remote region of myocardium that increased perfusion \( (74 \pm 170\%) \) throughout pacing.

Patients with a history of myocardial infarction may present with chest pain. In this study, ST-segment elevation during an exercise ECG was not associated with chest pain or detectable myocardial ischemia. Regional perfusion in infarcted segments of myocardium did not change with atrial pacing. However, separate precordial areas of ST-segment depression during exercise were associated with angina during exercise and pacing. ST-segment depression was also associated with the presence of a separate region of myocardium showing reversible disturbances of perfusion during pacing.

PATIENTS with a history of myocardial infarction may present with chest pain and the clinician needs objective evidence as to whether there is new myocardial ischemia. The complaint of chest pain may not be specific, and the ECG at rest may show only pathologic Q waves. The ECG during exercise may show ST-segment elevation or depression. The clinician must know what disturbances of regional myocardial perfusion occur in patients who have a history of myocardial infarction and who present with chest pain. In addition, diagnosis and management may be aided if the clinician knows what the ST-segment changes mean during stress in this situation.

We describe the exercise ECG and disturbances of regional myocardial perfusion during stress in a group of patients with a history of myocardial infarction who present with chest pain. Permanent and reversible disturbances of regional myocardial perfusion have been recorded in these patients and have been related to abnormal electrocardiographic signs. We examine the disturbances of myocardial perfusion in coronary artery disease (CAD) and its relation to the widely available and noninvasive use of the ECG.

METHODS

Between January 1978 and December 1979, 23 patients (22 male and one female, ages 31–68 years,
mean 51 years) were admitted to Hammersmith Hospital with a history of daily anginal chest pain and an abnormal ECG at rest showing pathologic Q waves. Thirteen patients were in functional class IIa, seven were in class III and three were in class IV of the New York Heart Association classification (assessment of severity). Past myocardial infarction was documented in each patient by characteristic ECG and enzyme changes which occurred 8 months to 5 years before this study. During the same period 12 patients presented with angina. These 12 patients had no history of myocardial infarction and had normal ECGs at rest.

Each patient underwent a symptom-limited exercise test (with electrocardiography), left ventricular (LV) and coronary angiography and an assessment of regional myocardial perfusion using krypton-81m and atrial pacing. Catheterization was performed either because a firm diagnosis of angina pectoris and myocardial ischemia were in doubt or because the symptoms were not controlled by medical treatment. The exercise mapping, the angiography and the scintigraphy are described below.

Precordial Mapping of the ECG

Sixteen precordial electrocardiographic leads (unipolar V leads connected to a central Wilson terminal) were positioned on the chest in order to cover the left hemithorax. Each patient performed an increasing work load on a bicycle ergometer using a standardized procedure. The exercise tests were limited by chest pain, dyspnea, fatigue or multiple ventricular ectopic beats.

Five measurements were calculated from each exercise test: total work load; onset and duration of chest pain; number of precordial positions showing significant ST-segment depression, elevation or pathologic Q waves; the sum of all the ST-segment depression (mm); and the precordial distribution of ECG signs, identified as anterior or inferior from the leads involved and their position within the precordial map. The method, reproducibility, sensitivity and specificity of this technique have been published.

Left Ventricular and Coronary Angiography

After local anesthesia, LV angiography and selective coronary arteriography were performed using the Judkins technique. The surface ECG (lead II and aVF) was monitored throughout the procedure. Afterwards, all the patients were free of chest pain and the ECG had returned to the control pattern.

A #5F pacing wire was inserted into the right femoral vein and advanced to the right atrium. The pacing threshold was tested until it was less than 1 V.

Each angiogram was reported by inspection by a radiologist and cardiologist who were unaware of the other investigations and routinely report more than 200 angiograms per year. A second cardiologist reported independently. Disagreement was settled by an independent cardiac radiologist. Each investigator was asked to report whether the LV angiogram was normal, showed dyskinesia or diffuse failure of contraction. The borders of the LV angiogram were divided into two anterior, one apical and two inferior segments for analysis of contraction. Akinesis and dyskinesia were grouped together. Dyskinesia was identified if all observers reported akinesia or dyskinesia in at least two segments on the borders of the LV angiogram.

Each investigator was asked to report whether the coronary arteries were normal or showed stenosis ≤ 50% or ≥ 70%.

Krypton-81m Scintigraphy of Myocardial Perfusion

After the coronary angiogram, a specially designed catheter was introduced to deliver krypton-81m in 5% dextrose by continuous infusion into the right and left aortic sinuses.

Krypton-81m was continuously eluted in sterile 5% dextrose from a portable pyrogen-free store of rubidium-81 (20–35 mCi). This solution was passed through a millipore filter (Milllex, Millipore SA) and then delivered to the cardiac catheter at 10 ml/min by a roller pump (Watson-Marlow MRHE 88). The activity delivered at the aortic sinuses was calculated to be 5–7 mCi/min during the continuous infusion.

The patient was then positioned with the chest in the field of a GEC maxi gamma camera (Type 400 T). This was linked to a Delton-Nova 1220 digital computer. The energy detection was set at 190 keV ± 15% and images of the myocardial distribution of krypton-81m were recorded by collecting 200,000 counts on 35-mm film. Electronic gating and a visual display unit were used to construct up to seven areas of interest and record regional myocardial counts per minute.

Krypton-81m scintigrams of the heart were recorded with each patient in the anterior, right anterior oblique and left anterior oblique (LAO) positions. The LAO position was then chosen for continuous imaging and regional myocardial measurement of counts per minute during transvenous pacing. The heart rate was increased by 10 beats/min at 2-minute intervals until the patient complained of chest pain, shortness of breath, discomfort or had a heart rate of 140 beats/min. Images were recorded throughout this procedure and for 10 minutes thereafter. Scintigraphy took no longer than 40 minutes in any patient.

At the end of each study the serial images were independently assessed by a radiologist and cardiologist.

Data were recorded every 30 seconds as digitized images throughout the study. At the end of each study a cardiologist, radiologist and technician were asked to analyze the data independently. An electronic light pen and visual display unit were used to enclose up to seven areas of interest. The areas included background, the catheter with krypton-81m, the aortic sinuses, the total myocardial image, any area of myocardium showing abnormal changes or defects during pacing and finally the rest of the myocardium remote from the defects. Time-activity graphs with tables of the counts per minute in each area of interest were
recorded with the serial images of regional myocardial perfusion from each study.3-9

The nature, intention and potential dangers of the procedure were explained to each patient before the study. Signed consent was obtained as required by the hospital ethics committee. Isotope panel clearance was obtained before the studies commenced. Analysis of variance was used to assess changes in regional myocardial activity of krypton-81m. The parameters were analyzed by a radiologist and technician who were unaware of the clinical details. A cardiologist also analyzed the data and all disagreements were settled by an independent cardiologist. Results are expressed as mean ±SD unless otherwise stated.

Results

Twenty-three of the 35 patients had a history of myocardial infarction. Eighteen of the 23 patients had pathologic Q waves in anterior leads and five had inferior Q waves at the time of this study.

Exercise Electrocardiography

The 12-lead ECG showed all the patients to be in sinus rhythm, with no significant widening of the QRS or ST-segment changes at rest. Three of the 35 patients had significant ST-segment elevation in the resting ECG.4

The heart rate increased from 83 ± 17 beats/min to 154 ± 23 beats/min. The systolic blood pressure changed from 140 ± 15 mm Hg to a peak of 179 ± 24 mm Hg.

The results of the exercise electrocardiography and the regional myocardial perfusion are expressed in three groups.

Group 1

Twelve of the 35 patients with no history of myocardial infarction had no significant ST-segment changes or chest pain during the exercise test. These patients achieved a work load of 40,000-58,000 J (mean 45,000 J).

Group 2

Thirteen of the 35 patients had a history of myocardial infarction and an area of pathologic Q waves in the precordial ECG. The same precordial area showed significant ST-segment elevation in the exercise test. In addition, all 13 patients showed a separate precordial area of ST-segment depression (5.0 ± 2.0 positions affected; 10 ± 4.0 mm of ST depression at peak). The 13 patients complained of chest pain during the test and achieved 15,000-37,000 J (mean 28,000 J).

Group 3

Ten of the 35 patients with a history of myocardial infarction exhibited pathologic Q waves in the precordial ECG at rest. During and after exercise all 10 patients showed significant ST-segment elevation in

<table>
<thead>
<tr>
<th>Coronary arteriogram</th>
<th>Left ventriculogram: pattern of contraction</th>
<th>No. of pts</th>
</tr>
</thead>
<tbody>
<tr>
<td>Reported as normal</td>
<td>Normal</td>
<td>8</td>
</tr>
<tr>
<td>≤ 50% stenosis</td>
<td>Normal</td>
<td>2</td>
</tr>
<tr>
<td>LAD alone</td>
<td>Normal</td>
<td>2</td>
</tr>
<tr>
<td>LAD, RCA + LCX</td>
<td>Normal</td>
<td>2</td>
</tr>
<tr>
<td>≥ 70% stenosis</td>
<td>Anterior dyskinesia in 7</td>
<td>7</td>
</tr>
<tr>
<td>LAD alone</td>
<td>Anterior dyskinesia in 2</td>
<td>2</td>
</tr>
<tr>
<td>LAD + RCA</td>
<td>Anterior dyskinesia in 8, inferior in 4</td>
<td>12</td>
</tr>
<tr>
<td>RCA + ≤ 50% of LAD</td>
<td>Inferior dyskinesia</td>
<td>2</td>
</tr>
<tr>
<td>Total</td>
<td></td>
<td>35</td>
</tr>
</tbody>
</table>

Abbreviations: LAD = left anterior descending coronary artery; RCA = right main coronary artery; LCX = left circumflex coronary artery.

leads with Q waves. None of the patients complained of chest pain. All of these patients achieved a work load of 8000-36,000 J (mean 20,000 J). Unlike the other groups, the 10 members of group 3 stopped exercising because of dyspnea.

Left Ventricular and Coronary Angiograms

The 16 patients with anterior Q waves all had anterior or apical dyskinesia reported in the LV angiograms. Minor degrees of wall motion abnormality (i.e., hypokinesia) were not identified and not included in this study. Similarly, the seven patients with inferior Q waves had inferior dyskinesia reported in the LV angiograms. None of the patients had stenosis of the main stem of the left coronary artery. The distribution of stenoses identified in the coronary arteriogram is summarized in tables 1 and 2.

Regional Myocardial Perfusion

The background activity measured in each study was always ≤ 5% of the total activity in the myocardial images. The counts per minute in the aortic sinuses varied by ≤ 5% throughout each study.

Group 1

Twelve of the 35 patients with negative ECG exercise tests all had uniform increases in regional myocardial activity of krypton-81m (98.0 ± 11.5%; p < 0.01) and no chest pain during atrial pacing. An example of serial images from one patient recorded before, during and after atrial pacing is shown in figure 1. The areas of interest include the interventricular septum and apex and the free wall of the left ventricle. An analysis of variance showed that there were no significant differences between the changes in activity in the two areas.

Group 2

The changes in the regional myocardial activity of krypton-81m were calculated by the three inter-
TABLE 2. The Findings at Angiography in Relation to the Exercise ECG

<table>
<thead>
<tr>
<th>Angiograms</th>
<th>Coronary arteriogram</th>
<th>Left ventriculogram</th>
<th>Exercise ECG</th>
<th>ST depression</th>
<th>Angina</th>
<th>No. of pts</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Normal</td>
<td>Normal</td>
<td></td>
<td>Q waves + ST elevation</td>
<td>None</td>
<td>None</td>
<td>8</td>
</tr>
<tr>
<td>≤ 50% stenosis</td>
<td>None</td>
<td></td>
<td>ST depression</td>
<td>None</td>
<td>None</td>
<td>2</td>
</tr>
<tr>
<td>2. LAD alone</td>
<td>None</td>
<td></td>
<td>Angina pts</td>
<td>None</td>
<td>None</td>
<td>2</td>
</tr>
<tr>
<td>≥ 70% stenosis</td>
<td>None</td>
<td></td>
<td>Anterior Q + ST elevation (7)</td>
<td>None in 1</td>
<td>Angina in 1</td>
<td>7</td>
</tr>
<tr>
<td>3. LAD alone</td>
<td>LAD + RCA</td>
<td>Anterior dyskinesia (7)</td>
<td>Anterior Q + ST elevation (7)</td>
<td>None in 1</td>
<td>Angina in 1</td>
<td>2</td>
</tr>
<tr>
<td>LAD + RCA</td>
<td>Anterior dyskinesia (2)</td>
<td></td>
<td>Anterior Q + ST elevation (2)</td>
<td>None in 1</td>
<td>Angina in 1</td>
<td>12</td>
</tr>
<tr>
<td>LAD + RCA + LCX</td>
<td>Inferior dyskinesia (5)</td>
<td></td>
<td>Inferior Q + ST elevation (5)</td>
<td>Angular in 5</td>
<td>None</td>
<td>2</td>
</tr>
<tr>
<td>RCA + ≤ 50% of LAD</td>
<td>Inferior dyskinesia (2)</td>
<td></td>
<td>Inferior Q + ST elevation (2)</td>
<td>None in 1</td>
<td>Angina in 1</td>
<td>35</td>
</tr>
<tr>
<td>Total</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>35</td>
</tr>
</tbody>
</table>

Abbreviations: LCX = left circumflex coronary artery; RCA = right coronary artery; LAD = left anterior descending coronary artery.

The 13 patients in this group complained of angina during the exercise ECG and showed precordial areas of Q waves with ST elevation and a separate area of ST depression in the exercise ECG. Figure 2 is an example of the changes in regional myocardial perfusion during pacing in a patient from this group.

Group 3

Figure 4 shows the scintigrams from one of 10 patients who had a history of myocardial infarction.

Figure 1. Myocardial scintigrams recorded from a patient with a coronary arteriogram and an exercise ECG reported as normal. The distribution of perfusion is shown in the left anterior oblique (LAO) projection while the patient's heart rate was (A) 70 beats/min, (B) 110 beats/min, (C) 140 beats/min and (D) 75 beats/min. No maldistribution of regional myocardial perfusion is shown during atrial pacing.
These 10 patients had precordial Q waves with transient ST-segment elevation but no chest pain during exercise.

The reviewers agreed that all 10 showed a fixed defect of regional myocardial perfusion before, during and after atrial pacing. During pacing these patients showed no significant change in the regional myocardial activity of krypton-81m in the affected area. However, the remote myocardium showed significant (82.0 ± 17.0%; p < 0.001) increases in perfusion during the pacing. None of the patients complained of chest pain during exercise or pacing.

The relationships between precordial areas of Q waves, ST elevation, ST depression and underlying disturbances of regional myocardial perfusion are summarized in table 3.

All of the changes in regional myocardial perfusion seen during the atrial pacing returned to the control state when pacing was discontinued.

**Discussion**

Patients who have suffered myocardial infarction may complain of chest pain after the event. The pain may or may not be characteristic of angina pectoris and the diagnosis is important because the recurrence of ischemia affects the patient's treatment and prognosis.10 The ECG is frequently difficult to interpret in these patients because of the abnormalities related to the past infarction. Nevertheless, the clinician must know whether the chest pain represents new regional myocardial ischemia and how to interpret the ST-segment and T-wave changes that occur in these patients during an exercise test.10-12

ST-segment depression during exercise in patients with CAD is a well-known ECG sign that is widely used in clinical practice. Experimentally, this is related to patchy ischemia of ventricular myocardium with a poor boundary between affected muscle and surrounding normal tissue. This ECG sign correlates with angina pectoris, CAD and transient disturbance of regional myocardial perfusion. ST-segment elevation has been described in normal subjects and during the course of myocardial infarction. Transient ST elevation is also associated with variant angina pectoris and experimentally, this ECG sign correlates with severe homogeneous regional myocardial ischemia with a more clearly defined border zone between normal and ischemic myocardium.13-17
Longhurst and Kraus described a group of patients presenting with chest pain who showed ST-segment elevation during exercise. These patients had no history of myocardial infarction and this ECG sign was related to significant CAD. This clinical finding is infrequent in relation to the more frequent finding of ST-segment elevation during exercise in ECG leads showing pathologic Q waves.

A more common clinical picture is the patient who presents with chest pain, has a history of myocardial infarction and shows ST elevation during exercise in leads with Q waves. These patients may or may not experience chest pain during the stress test; they frequently have wall motion abnormalities in the left ventriculogram and significant CAD. The ST elevation may or may not be accompanied by ST depression during exercise in the same patient. A continuous infusion of krypton-81m provides an assessment of changes in regional myocardial perfusion. The technique allows each patient to act as his or her own control and provides detailed information about directional changes in regional myocardial perfusion from the resting state in response to stress.

Nevertheless, the spatial resolution of changes in regional myocardial perfusion is limited using radionuclides and planar imaging; therefore, the absence of new, detectable, transient disturbances of regional myocardial perfusion must be interpreted with caution.

We have investigated only one aspect of ST elevation in patients with past myocardial infarction. The question arises as to whether ST depression can represent new transient regional myocardial ischemia and not simply reciprocal changes. This study suggests that separate precordial areas of ST depression can be associated with angina pectoris during exercise and reversible disturbances of regional myocardial perfusion during pacing.

The findings in this study also showed that ST elevation during exercise in leads showing Q waves was not associated with angina pectoris or transient disturbances of regional myocardial perfusion during stress. This supports the past research that suggests that ST elevation in these circumstances is related to the wall motion abnormality and to the electrophysiologic differences between normal and infarcted myocardium.

This study has shown that patients with a history of myocardial infarction who present with angina pectoris may show precordial areas of Q waves associated with ST-segment elevation and separate precordial areas of ST-segment depression during exercise. Q waves and ST elevation were associated with angiographic evidence of infarction and fixed defects

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**Figure 4.** This series of images was recorded before (A), during (B) and after (C) atrial pacing in one of 10 patients who had Q waves with ST elevation alone in the ECG during exercise and no angina during exercise or pacing. Remote myocardium increased activity during pacing. Infarcted segments did not change significantly.

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**Table 3. Regional Myocardial Perfusion and the Exercise ECG**

<table>
<thead>
<tr>
<th>Regional myocardial perfusion</th>
<th>No. of pts</th>
<th>Anterior Q + ST elevation</th>
<th>Anterior Q + ST depression</th>
<th>Inferior Q + ST elevation</th>
<th>Inferior Q + ST depression</th>
<th>No. of pts</th>
</tr>
</thead>
<tbody>
<tr>
<td>Uniform perfusion</td>
<td>12</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Anteroseptal fixed defects</td>
<td>8</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Inferior fixed defect</td>
<td>2</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Anteroseptal fixed defects and reversible inferior defect</td>
<td>8</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Inferior fixed defect and reversible anterior defect</td>
<td>5</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Total</td>
<td>35</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Abbreviations: ST + = ST-segment elevation; ST − = ST-segment depression.
of regional myocardial perfusion unaffected by stress. Separate precordial areas of ST-segment depression are not necessarily reciprocal changes and may be associated with angina pectoris, CAD and reversible disturbances of regional myocardial perfusion during atrial pacing. These findings aid the understanding of the ECG.

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An investigation in patients with previous myocardial infarction who present with chest pain.
A P Selwyn, K Fox, G Forse, T Pratt and R Steiner

Circulation. 1981;64:1156-1162
doi: 10.1161/01.CIR.64.6.1156

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