Exercise-induced ST segment elevation

Electrocardiographic, angiographic, and scintigraphic evaluation

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SUMMARY Two hundred and fifteen patients with previous myocardial infarction were investigated between four and six months after the acute episode by computer assisted 12 lead exercise electrocardiography. Thirty-six (17%) out of this group showed ST segment elevation over the infarct zone, reflected by leads presenting with “QS” configuration. They were further investigated by serial thallium-201 scintigraphy, coronary arteriography, and left ventricular angiography. All showed left ventricular wall motion abnormalities and 89 per cent were diagnosed to have left ventricular “aneurysm” (dyskinesia and akinesia). In a further patient with a posterior aneurysm, the exercise-induced ST elevation could only be detected by using an oesophageal lead.

We suggest that these changes reflect severe underlying left ventricular wall motion abnormalities in the presence or absence of reversible myocardial ischaemia. The mechanism of ST segment elevation in this situation, occurring in leads with a “QS” configuration, may be mechanical in the majority of the patients rather than due to reversible myocardial ischaemia.

ST segment depression induced by exercise is recognised as the prime criterion for ischaemia but ST segment elevation during exercise is rarely seen and its exact mechanism and clinical significance are not clearly understood. Exercise-induced ST segment elevation has been reported as indicative of severe myocardial ischaemia and of abnormal left ventricular wall motion. We investigated 37 patients who showed such changes with the object of elucidating the mechanisms and defining the clinical significance of exercise-induced ST segment elevation.

Patients and methods

Of a total of 215 patients with previous transmural infarction undergoing routine assessment (188 anterior, 26 inferior, and one true posterior), 36 patients showed ST segment elevation in one or more leads of the 12 lead electrocardiogram on exercise. The patient with the true posterior infarction showed ST elevation only in an oesophageal lead.

EXERCISE TESTING

Treadmill exercise testing was performed with a modified Balke’s procedure (Table 1) using a computer-assisted system (CASE, Marquette Electronics) four to six months after the acute episode. A standard 12 lead electrocardiogram was recorded in the supine and standing postures before exercise, every minute during exercise, and in the post-exercise period. Three unipolar chest leads with

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<th>Table 1</th>
<th>Modified Balke’s treadmill exercise procedure</th>
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maximum "Q" wave depth were continuously
monitored by an on-line digital computer during
exercise. A symptom-related maximal exercise end-
point was used unless exercise had to be terminated
because of dangerous arrhythmias. Systolic blood
pressure was measured every three minutes during
exercise using a standard mercury sphygmomano-
meter.

Significant ST segment deviations were defined
as 1 mm of ST segment depression or elevation
from the baseline occurring either during exercise
or recovery or both.

To standardise the degree of ST segment
elevation between patients the displacement of the
ST segment was measured in each lead with a "QS" con
figuration and a mean ST segment displacement
per lead was calculated at rest and at the maximal
change during exercise. A mean ST segment at
rest (STR) and a mean maximal exercise-induced
ST segment elevation (STE) were calculated.

Concomitant ST segment depression was simi-
larly measured from the 12 lead electrocardiogram.

ANGIOGRAPHY
Selective coronary and left ventricular cine-
angiography was performed in multiple views using
the Judkins technique in all patients. Significant
coronary artery narrowing was defined as 70 per
cent or more luminal narrowing of major vessels
and 50 per cent or more luminal narrowing of the
left main stem coronary artery (LMS). Left
ventricular angiograms were classified as normal,
dyskinetic, akinetic, and hypokinetic with or without
asynery.

THALLIUM-201 MYOCARDIAL IMAGING
A scalp-vein needle was placed in a forearm vein
and flushed with heparinised saline to facilitate the
rapid injection of thallium-201 during exercise. The
patient was exercised till the previously determined
maximal end point limit was reached, 1-5 mCi of
thallium-201 was injected, and exercise was con-
tinued for at least another 15 seconds. Electrocadi-
ographic monitoring was carried out throughout
the exercise and until ST segment changes reverted
to pre-exercise baseline. Before each series of studies
a phantom that gave an image similar to a heart was
recorded on 70 mm film (Kodak PF) using a high
resolution, low energy parallel hole collimator
(Searle Radiographic Pho/Gamma 111 HP). The
20 per cent window was centred on the 75 keV peak
and 200 000 counts were collected at a series of
intensity settings on the cathode ray tube. The
image with the highest contrast was selected and
the entire series of studies was carried out using
that intensity setting.

Scintigrams were taken immediately after exercise,
and approximately one-and-a-half hours and five
hours after exercise. Anterior, 45° left anterior
oblique, and left lateral views were obtained on
each occasion.

A closed circuit television system was used to
give an enlarged view of the exercise images with
background subtraction and contrast enhancement
preset using the television monitor controls, and
this setting was constant for reading all the scinti-
graphic data in identical fashion.

A focal defect present at rest was interpreted as
suggesting a previous myocardial infarction "scar".
If the focal defect increased or appeared with the
stress scintigram this was interpreted as indicative
of myocardial ischaemia and was termed a "positive"
scintigram.

Two observers independently interpreted and
reported the scintigrams and angiograms with no
knowledge as to the patient's identity or clinical
history.

Results

There were two women and 35 men in the study
with ages ranging from 34 to 67 years. Thirty-two
had anterior myocardial infarction, four had had
inferior myocardial infarction, and one had a true
posterior infarction. The latter has been considered
separately from the main group of 36 in the
analysis because the exercise-induced ST elevation
could only be detected by using an oesophageal
lead. The time of investigation was at an interval of
four to six months after infarction (mean 5·3
months).

EXERCISE DATA
Only 56 per cent of patients had elevated ST
segments on the resting electrocardiogram. The
mean resting ST level (STR) was 0·4 ±0·1 mm SE.
The mean ST segment level on peak exercise (STE)
was 2·9 ±0·2 mm SE. The mean resting heart rate
was 79 ±2 SE beats/min and the mean maximum
heart rate achieved during exercise was 135 ±3 SE
beats/min. The mean resting systolic blood pressure
was 127 ±2 SE mmHg and the mean of the peak
exercise systolic blood pressure was 135 ±4 SE
mmHg. Seventeen had a normal blood pressure
response to exercise. Those with a fall in blood
pressure (nine patients) or no change in systolic
blood pressure on exercise (10 patients) were
described as having an "abnormal" blood pressure
response. Exercise-limiting symptoms were dys-
pnoea in 17, fatigue in eight, and chest pain in
seven. In four the exercise was stopped because of
significant arrhythmias. Ventricular ectopic beats
were present in six at rest, but 16 developed ventricular arrhythmias on exercise and one patient had a run of ventricular tachycardia which spontaneously reverted to sinus rhythm.

**ANGIOGRAPHIC DATA**

All 36 patients had abnormalities of left ventricular wall motion; 32 had either dyskinesia or akinesia or a combination, and were, therefore, identified as having an “aneurysm”\(^1\); four had either hypokinesia or asynergy or both and the appearance was not diagnostic of an aneurysm. Insignificant mitral regurgitation was present in four patients.

Coronary arteriography revealed single vessel disease in 16 (45%) (15 anterior, one inferior myocardial infarction (AMI, IMI)), two vessel disease in 12 (33%) (9 AMI, 3 IMI), and triple vessel disease in eight (22%) (all 8 AMI). Out of 32 patients with anterior myocardial infarction two had incomplete occlusion of the left anterior descending artery, and 28 (88%) had total occlusion of this artery. Of the four patients with inferior myocardial infarction, two had 70 per cent occlusion of the left anterior descending artery, one had 70 per cent occlusion of the circumflex artery (Cx), and all four had total obstruction of the right coronary artery (RCA). Significant disease of the left main stem was present in four (all had AMI). Out of 15 patients with anterior myocardial infarction and single vessel disease, 13 had total obstruction of the left anterior descending artery (LAD), and one had 90 per cent luminal narrowing of the left anterior descending artery.

**THALLIUM-201 SCINTIGRAPHY**

A resting myocardial perfusion abnormality was disclosed in all 36 patients studied. A “positive” exercise scintigram was reported in 13 patients. Only one out of 13 with a “positive” scintigram had single vessel disease and the rest had multiple vessel disease (Table 2).

Twenty-three (64%) had “negative” exercise scintigrams out of which 15 patients (14 AMI and one IMI) had single vessel obstruction, thus excluding the possibility of reversible myocardial ischaemia causing ST segment elevation (Fig. 1 and 2).

Out of 12 (11 AMI and one IMI) with multiple vessel disease and a “positive” scintigram, six showed no evidence of ST segment depression in reciprocal leads (Fig. 3 and 4).

A prominent right ventricular image on the scintigram was obtained in three patients immediately after exercise and all three had a resting pulmonary capillary wedge pressure in excess of 19 mmHg (omega-sign\(^1\)) (Fig. 5).

### Table 2. Summary of angiographic, exercise, and scintigraphic changes in all 37 patients

<table>
<thead>
<tr>
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<th>Coronary arteriography</th>
<th>Reciprocal ST depression</th>
<th>'Positive' scintigraphy</th>
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Dys, dysskinesia; Akin, akinesia; Hypo, hypokinesia; Asyn, asynergy; LMS, left main stem coronary artery; LAD, left anterior descending coronary artery; Cx, left circumflex coronary artery; RCA, right coronary artery; t, total occlusion; +, present; −, negative; %, percentage occlusion of coronary artery; 1–32, anterior myocardial infarction; *, inferior myocardial infarction; †, true posterior myocardial infarction.

**CASE REPORT**

The patient who had a true posterior infarction had a posterior left ventricular aneurysm and the circumflex artery was totally occluded at its origin; the rest of the coronary tree appeared free of atheroma. An exercise test with an oesophageal lead showed significant ST segment elevation in the oesophageal lead and ST segment depression in V2–V4 praeordial leads (Fig. 6). This patient had no cardiac pain but stopped exercise because of dyspnoea. There was a significant reduction in systolic blood pressure during exercise. Scintigraphy showed a perfusion defect in the posterior wall (best seen in the left lateral view) and no evidence of myocardial ischaemia was noted after exercise.
Exercise-induced ST segment elevation

Discussion

Fortuin et al.\(^5\) reported five cases with previous myocardial infarction and exercise-induced ST segment elevation occurring only in the leads with a “QS” complex. They ascribed this finding to myocardial ischaemia. Hegge et al.\(^4\) and Atterhög et al.\(^1\) suggested that exercise-induced ST segment elevation was the result of myocardial ischaemia without excluding left ventricular wall motion abnormalities. On the other hand, Chahine et al.\(^7\) reported the presence of left ventricular aneurysm (86%) and previous anterior myocardial infarction and left anterior descending occlusion (85%) in patients with exercise-induced ST segment elevation.

Paine et al.\(^9\) reported a “larger angiographic scar size” and lowered ejection fraction in 14 patients with ST segment elevation on exercise. Castellanet et al.\(^8\), Lahiri et al.\(^11\), Paine et al.\(^9\), and Weiner et al.\(^10\) have produced evidence of a positive relation between left ventricular wall motion abnormalities and exercise-induced ST segment elevation.

Our study was designed to bring out the clinical significance of ST segment elevation in patients with ischaemic heart disease and to clarify the existing doubts as to its mechanism. The characteristic features were exercise-induced ST segment elevation associated with dyspnoea or fatigue (70%), abnormal or “poor” systolic blood pressure response to exercise, previous myocardial infarction, and abnormal left ventricular wall motion even at rest.

Fig. 1 12 lead electrocardiogram at rest (a) and during maximal exercise (b) showing significant ST segment elevation over leads V2 to V4. Reciprocal ST segment depression is present in II, III, aVF, and V6.

Fig. 2 \(^{201}\)Tl scintigrams of same patient as in Fig. 1, anteroposterior view (AP) and left anterior oblique (LAO) scintigraphic views at rest; (a) showing a large perfusion defect in the anterior (AP) and septal walls (LAO) representing the infarction “scar”. There is no relative alteration of regional myocardial perfusion during exercise (b). rv, right ventricular image.
We did not attempt exercise left ventriculograms as the resting films showed that 89 per cent of the patients had left ventricular "aneurysms". We considered that a powered injection into the ventricle during exercise could be dangerous when such significant left ventricular abnormality was already present at rest.

Thallium-201 scintigraphy was used as an indicator of myocardial ischaemia to determine whether ischaemia occurred during exercise-induced ST segment elevation. In 15 out of 16 patients with single vessel disease a "negative" $^{201}$TI scintigram was obtained during exercise-induced ST segment elevation, but an infarction "scar" was visualised at rest in all patients.

Even though it is difficult to exclude the absence of ischaemia in the infarction zone and its surrounding area, the development of reversible ischaemia as a cause of ST segment elevation in these areas in response to dynamic exercise was excluded by thallium-201 scintigraphy. However, exercise-induced reversible regional myocardial ischaemia was observed in 13 (36%). Reversible ischaemia was noted to occur in the zone surrounding the infarct "scar" (four cases), or in a different region from the site of infarction (nine cases).

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Fig. 4 $^{201}$TI myocardial scintigrams of the same patient as in Fig. 3, in the resting (a) anteroposterior (AP) and left anterior oblique (LAO) views showing perfusion changes in the anteroapical and inferior walls (small arrows) and a reduction of relative perfusion during exercise (b) in the apical and inferior walls (large arrows) in a patient with no exercise-induced ST segment depression.
There is some controversy regarding the specificity and sensitivity of $^{201}$TI scintigraphy in detecting myocardial ischaemia when exercise images are compared with late redistribution images (five to six hours) as opposed to imaging performed at rest. However, the majority of authors feel that redistribution imaging is as sensitive as rest scintigraphy. Two-thirds of these patients did not show any evidence of reversible perfusion abnormalities in the late redistribution images. However, in 12 patients with a “positive” redistribution image there was evidence of severe multiple vessel pathology.

The use of an oesophageal lead indicated exercise-induced ST segment elevation with ST segment depression in the praecordial leads (V1 to V4) in one patient. In this case a large area of dyskinesia on the posterior surface of the ventricle was detected and the anterior ST segment depression, which was associated with a “negative” exercise scan, was a reciprocal change from the ST segment elevation documented by the oesophageal lead. Castellanet et al. have also suggested that inferoposterior ischaemia is indicated when exercise-induced ST segment elevation occurs specifically over the V1 praecordial lead. This rare finding

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**Fig. 5** $^{201}$TI scintigram at rest and exercise in the left anterior oblique (LAO) and left lateral (LL) views showing exercise-induced ischaemia in the left lateral and left anterior oblique views and a prominent right ventricle (“omega sign”) indicating severe left ventricular dysfunction during exercise.

**Fig. 6** Praecordial electrocardiogram leads at rest (a) in the patient with a posterior aneurysm after myocardial infarction showing ST-T depression mainly in V3. During exercise (b) ST segment depression is noted in V1 to V5. Simultaneous oesophageal lead electrocardiogram at rest shows a “QS” pattern and “T” wave inversion, and significant ST segment elevation is noted during exercise and in the post-exercise periods.
supports the contention that local wall motion abnormalities are responsible for ST segment elevation in this context.

In conclusion, we believe that exercise-induced ST segment elevation occurring during exercise in the leads presenting with a “QS” configuration (over the infarction “scar”) of a 12 lead electrocardiogram is likely to be the result of abnormal left ventricular wall motion in the presence or absence of myocardial ischaemia.

References


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