Exercise-induced ST segment elevation

Electrocardiographic, angiographic, and scintigraphic evaluation

A LAHIRI, V BALASUBRAMANIAN,* M W MILLAR CRAIG, J CRAWLEY, E B RAFTERY

From the Department of Cardiology and the Division of Radioisotopes, Northwick Park Hospital and Clinical Research Centre, Harrow, Middlesex

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,¹⁻⁵ and of abnormal left ventricular wall motion.⁶⁻¹¹ 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

Table 1 *Modified Balke's treadmill exercise procedure*

Stage	Time (min)	Speed (mph)	Gradient (%)
I	3	2	0
II	3	3	4
III	3	3	8
IV	3	3	12
V	3	3	16
VI	3	3	20
VII	3	4.5	20
VIII	3	6	20

* Supported by the British Heart Foundation.

Received for publication 31 December 1979

maximum "Q" wave depth were continuously monitored by an on-line digital computer during exercise. A symptom-related maximal exercise endpoint 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 sphygmomanometer.

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" configuration 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 similarly measured from the 12 lead electrocardiogram.

ANGIOGRAPHY

Selective coronary and left ventricular cine-angiography was performed in multiple views using the Judkins technique¹² 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 asynergy.¹³

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 continued for at least another 15 seconds. Electrocardiographic 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 scintigraphic 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 dyspnoea 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 movement; 32 had either dyskinesia or akinesia or a combination, and were, therefore, identified as having an "aneurysm"¹³; 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-sign¹⁴) (Fig. 5).

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

Case no.	LV angiography				Coronary arteriography			Reciprocal ST depression exercise	'Positive' ²⁰¹ Tl scintigraphy exercise
	Dys	Akin	Hypo	Asyn	LMS	LAD	Cx		
1	+	-	-	+	-	t	-	-	-
2	+	-	+	-	-	t	-	-	-
3	+	-	-	-	-	90%	-	-	+
4	+	+	+	-	-	-	90%	+	-
5	-	+	+	-	-	t	70%	90%	+
6	+	-	-	-	-	t	-	+	-
7	+	-	-	+	-	t	-	+	-
8	+	-	+	-	-	t	70%	-	+
9	+	-	+	+	-	t	70%	90%	+
10	-	+	-	-	-	t	-	70%	-
11	-	+	+	-	-	t	-	+	-
12	+	-	-	-	-	t	-	-	-
13	+	-	-	-	-	t	-	+	-
14	+	-	+	-	50%	t	-	70%	+
15	-	-	+	-	-	t	-	90%	+
16	+	-	-	+	-	t	70%	70%	+
17	+	-	-	-	-	90%	-	t	+
18	+	-	+	-	-	t	-	-	-
19	-	+	-	+	-	t	70%	70%	-
20	+	-	-	-	-	t	-	90%	+
21	+	-	-	-	-	t	-	-	+
22	+	-	+	-	-	t	-	-	-
23	+	-	-	-	-	t	-	70%	-
24	-	-	+	+	50%	t	70%	t	+
25	-	+	+	-	-	t	-	90%	+
26	+	-	-	-	-	t	-	-	+
27	-	+	-	-	50%	t	-	70%	-
28	+	-	+	-	-	t	-	+	-
29	+	-	-	-	-	t	-	-	-
30	-	-	+	-	50%	t	-	-	-
31	-	+	+	-	-	t	-	+	-
32	-	+	+	+	-	t	70%	70%	+
33*	-	+	-	-	-	-	70%	t	-
34*	+	-	+	-	-	70%	-	t	+
35*	+	+	-	-	-	70%	-	t	-
36*	-	-	+	-	-	-	-	t	+
37†	+	-	-	-	-	-	+	+	-

Dys, dyskinesia; 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 praecordial 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.

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.

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 movement 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. 2 ²⁰¹Tl 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}Tl 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).

Fig. 3 12 lead electrocardiogram at rest showing an anterolateral myocardial infarction (a) and significant ST segment elevation on exercise (b) in V3 to V5 but no ST segment depression is noted in this patient with triple vessel disease and "positive" scintigraphy.

Fig. 4 ^{201}Tl 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.

Fig. 5 ^{201}Tl 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.

There is some controversy regarding the specificity and sensitivity of ^{201}Tl 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.¹⁶⁻²⁰ 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 precordial 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.*⁸ have also suggested that inferoposterior ischaemia is indicated when exercise-induced ST segment elevation occurs specifically over the V1 precordial lead. This rare finding

Fig. 6 Precordial 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

- ¹Atterhög JH, Ekelund L-G, Kaijser L. Electrocardiographic abnormalities during exercise 3 weeks to 18 months after anterior myocardial infarction. *Br Heart J* 1971; **33**: 871-7.
- ²Kemp GL. Value of treadmill stress testing in variant angina pectoris. *Am J Cardiol* 1972; **30**: 781-3.
- ³MacAlpin RN, Kattus AA, Alvaro AB. Angina pectoris at rest with preservation of exercise capacity. *Circulation* 1973; **47**: 946-58.
- ⁴Hegge FN, Tuna N, Burchell HB. Coronary arteriographic findings in patients with axis shifts or ST-segment elevations on exercise stress testing. *Am Heart J* 1973; **86**: 603-15.
- ⁵Fortuin NJ, Friesinger GC. Exercise induced ST-segment elevation. Clinical, electrocardiographic and angiographic studies in twelve patients. *Am J Med* 1970; **49**: 459-64.
- ⁶Manvi KN, Ellestad MH. Elevated ST-segments with exercise in ventricular aneurysm. *J Electrocardiol* 1972; **5**: 317-23.
- ⁷Chahine RA, Raizner AE, Ishimori T. Clinical significance of exercise-induced ST-segment elevation. *Circulation* 1976; **54**: 209-13.
- ⁸Castellanet MJ, Greenberg PS, Ellestad MH. Comparison of ST-segment changes on exercise testing with angiographic finding in patients with prior myocardial infarction. *Am J Cardiol* 1978; **42**: 29-35.
- ⁹Paine TD, Dye LE, Roitman DI, et al. Relation of graded exercise test findings after myocardial infarction to the extent of coronary artery disease and left ventricular dysfunction. *Am J Cardiol* 1978; **42**: 716-23.
- ¹⁰Weiner DA, McCabe C, Klein MD, Ryan TJ. ST-segment changes post-infarction: predictive value of multi-vessel coronary disease and left ventricular aneurysm. *Circulation* 1978; **58**: 887-91.
- ¹¹Lahiri A, Wilkins R, Raftery EB. Correlation of coronary angiography, left ventricular angiography and graded treadmill exercise testing in the diagnosis of left ventricular aneurysms. *Ann Radiol* 1979; **22**: 261-3.
- ¹²Judkins MP. Percutaneous transfemoral selective coronary arteriography. *Radiol Clin North Am* 1968; **6**: 467-92.
- ¹³Herman MV, Heinle RA, Klein MD, Gorlin R. Localized disorders in myocardial contraction. Asynergy and its role in congestive heart failure. *N Engl J Med* 1967; **277**: 222-32.
- ¹⁴Rosenblatt A, Lowenstein JM, Kerth W, Handmaker H. Post-exercise thallium-201 myocardial scanning: a clinical appraisal. *Am Heart J* 1977; **94**: 463-70.
- ¹⁵Pohost GM, Zir LM, Moore HR, McKusick KA, Guiney TE, Beller GA. Differentiation of transiently ischemic from infarcted myocardium by serial imaging after single dose of thallium-201. *Circulation* 1977; **45**: 294-302.
- ¹⁶Hamilton GW, Ritchie JL. In Ritchie JL, Hamilton GW, Wackers FJT, eds. *Thallium-201 myocardial imaging*. New York: Raven Press, 1978: 55-68.
- ¹⁷Blood DK, McCarthy DM, Sciacca RR, Cannon PJ. Comparison of single-dose and double dose thallium-201 myocardial perfusion scintigraphy for the detection of coronary artery disease and prior myocardial infarction. *Circulation* 1978; **58**: 777-88.
- ¹⁸Sonnemaker RE, Floyd JL, Nusynowitz ML, Bode RF, Spicer MJ, Waliszewski JA. Single injection of thallium-201 stress and redistribution myocardial perfusion imaging: comparison with stress electrocardiography and coronary arteriography. *Radiology* 1979; **131**: 199-203.
- ¹⁹Corne RA, Gotsman MS, Atlan H. Radionuclide assessment of regional myocardial perfusion with thallium-201. *Am Heart J* 1979; **97**: 112-8.
- ²⁰McCarthy DM, Blood DK, Sciacca RR, Cannon PJ. Single dose myocardial perfusion imaging with thallium-201: application in patients with non-diagnostic electrocardiographic stress tests. *Am J Cardiol* 1979; **43**: 899-906.

Requests for reprints to Dr E B Raftery, Northwick Park Hospital, Watford Road, Harrow, Middlesex HA1 3UJ.