

# Submaximal exercise testing early after myocardial infarction

## *Difficulty of predicting coronary anatomy and left ventricular performance*

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**SUMMARY** Impaired left ventricular function and extensive coronary artery disease are important determinants of prognosis after acute myocardial infarction. The ability of clinical and predischARGE submaximal exercise test variables to predict multivessel coronary artery disease and impaired left ventricular function was assessed in 62 survivors of acute myocardial infarction. Abnormal exercise blood pressure response and short exercise performance were predictors of multivessel disease, but exercise induced ST segment changes and clinical variables were not. Q wave infarction, high grade Killip classification, and exercise induced ST segment elevation predicted statistically significant impairment of resting left ventricular function, whereas other clinical and exercise test variables did not.

Exercise induced ST segment changes were therefore of little value in detecting extensive coronary disease, although exercise induced ST elevation was an indicator of poor resting left ventricular function. Although abnormal exercise haemodynamics may detect extensive coronary artery disease, other physiological markers of reversible myocardial ischaemia are probably necessary to plan optimal management in these patients.

The single most important predictor of adverse prognosis after myocardial infarction is a reduced left ventricular ejection fraction.<sup>1-3</sup> The presence of extensive coronary artery disease may be an important independent predictor of cardiac complications in those patients with moderate rather than severe impairment of left ventricular performance.<sup>2-4</sup>

We therefore analysed the ability of exercise testing early after infarction, together with simple clinical factors of known adverse prognostic significance, to predict left ventricular performance and coronary anatomy. The exercise test variables studied were those previously reported to define groups of patients at risk of subsequent complications: exercise induced ST depression,<sup>5-7</sup> inability to complete a submaximal exercise protocol,<sup>8</sup> an abnormal blood pressure

response to submaximal exercise,<sup>9</sup> and exercise induced ST elevation.<sup>10 11</sup>

### Patients and methods

#### STUDY GROUP

The study group consisted of 62 patients (58 men, four women; age range 29-64 (mean 53.8) years) who had performed submaximal exercise tests between 7 and 23 (mean 10.8) days after acute myocardial infarction and in whom coronary anatomy was known. In 60 patients coronary anatomy had been established by coronary arteriography and in two at necropsy. The study group was derived from a total of 131 patients aged  $\leq 65$  years admitted to hospital between May 1981 and August 1982 who fulfilled at least two of the following criteria: (a) typical chest pain lasting longer than 30 minutes; (b) increase in serum creatinine kinase and lactate dehydrogenase activity to more than twice the upper limit of the hospital normal ranges, with a typical evolutionary pattern on serial estima-

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tions; and (c) evolutionary electrocardiographic changes. Q wave infarction occurred if the electrocardiographic changes included the development of new Q waves of at least 0.04 s duration and >25% of the amplitude of the subsequent R wave and was classified as anterior if new Q waves occurred in any of leads I, aVL, V1 to V6 or as inferior if new Q waves occurred in at least two of leads II, III, or aVF.

During the time this investigation was in progress 43 patients with acute infarction did not undergo early exercise testing because of death in hospital (16), persistent heart failure (3), previous coronary artery bypass surgery (5), intractable angina requiring early surgery (2), various non-cardiac diseases (9), or patient inability or physician refusal to undertake exercise testing at this time (8). A further 26 patients with infarction performed the early exercise test but were not studied by cardiac catheterisation because they did not fulfil any of the indications for this procedure—namely: exercise induced ST depression, angina during follow up, or young age in asymptomatic patients.

All the patients with exercise induced ST depression underwent left ventricular angiography and coronary arteriography six weeks after infarction except for two who died before their planned readmissions. One of these, who had sustained inferior myocardial infarction, had severe widespread disease in all the major coronary arteries at postmortem examination and was included in the data analysis. In the other, who had both ST depression and elevation induced by submaximal exercise after anterior infarction, necropsy was not performed. Twenty seven patients without exercise induced ST depression underwent left ventricular angiography and coronary arteriography (median time 6 (range 3–56) weeks) after acute myocardial infarction. Fourteen were investigated because of symptoms, and 13 asymptomatic patients (mean age 45.6 years) were investigated to aid prognostic assessment. A man who died suddenly six months after inferior infarction had severe multivessel coronary disease at necropsy and was included in the data analysis. No patient had recurrent myocardial infarction before angiography.

#### EXERCISE TESTING

Exercise testing was performed according to a modified Naughton protocol<sup>12</sup> using a constant treadmill speed of 2 mph, with the initial treadmill grade of zero increasing by 3.5% at the end of each two minute stage. This protocol is designed so that the oxygen consumption required increases by approximately one metabolic equivalent (about 3.5 ml/kg/min) with each two minute stage, starting with a workload of two metabolic equivalents. Twelve lead electrocardiograms were recorded at rest, at the end of each two

minute stage, immediately after exercise, and at one minute intervals thereafter for a minimum period of five minutes or until the resting electrocardiographic pattern had been regained. A three lead rhythm strip was recorded continuously throughout exercise and recovery periods. Cuff blood pressure was recorded at rest, at the end of each two minute stage during exercise, and at one and four minutes after termination of exercise.

ST segment depression or elevation induced by exercise was defined as at least 0.1 mV depression or elevation of the ST segment respectively compared with the resting tracing, measured 0.06 s after the QRS complex in three consecutive complexes with a stable baseline.

End points for the submaximal exercise tests were: eight minutes exercise, the workload of the final two minute stage being five metabolic equivalents; exceeding 70% of the maximum predicted heart rate for age; induction of angina; undue fatigue or dyspnoea; induced ST segment depression of more than 0.5 mV; serious ventricular arrhythmias, or a fall in systolic blood pressure of  $\geq 20$  mm Hg from peak values during exercise. An inadequate blood pressure response to exercise was defined as an increase in systolic blood pressure of  $\leq 10$  mm Hg during exercise, with peak systolic blood pressure being  $< 140$  mm Hg, or a decrease in systolic blood pressure of  $\geq 20$  mm Hg during exercise.<sup>9</sup>

At the time of exercise testing all the patients were ambulant without chest pain and free of clinical and radiological signs of heart failure. Heart failure during the acute infarction period that had subsequently resolved was not a contraindication to exercise testing.

Normal drug treatment was continued on the day of the submaximal exercise tests. Ten of the 62 patients were receiving beta blocking agents: three were taking antihypertensive drugs before hospital admission and seven had beta blockade instituted in the periinfarction period. An attempt was made to minimise any effect of this by performing exercise tests as long as possible after the last dose whether the frequency of dosage was once or twice daily. No patient was taking digitalis.

#### CARDIAC CATHETERISATION

Coronary arteriography and left ventriculography were performed using standard Sones techniques. Multiple coronary angiograms in planes of varying obliquity, including cranial caudal views in both right and left anterior oblique planes, were obtained. Coronary anatomy was assessed by agreement between two observers, at least one of whom did not know the outcome of the preceding exercise test. Stenotic lesions of  $> 70\%$  luminal diameter in one projection, or  $> 50\%$  in two or more projections, were considered

significant as these criteria are commonly used to assess the suitability of a vessel for coronary artery bypass grafting.<sup>13</sup> Stenoses in major diagonal or obtuse marginal vessels were considered to constitute left anterior descending or left circumflex coronary artery disease respectively. Patients were classified according to the presence or absence of multivessel coronary disease—that is, significant stenoses in at least two of the three major coronary arteries—thereby avoiding some of the problems of assessing the relative importance of two vessel and three vessel disease in patients with major individual variations in coronary anatomy. Left ventricular angiography was obtained in the 30° right anterior oblique plane and left ventricular ejection fraction determined by the area-length method<sup>14</sup> using a Vanguard XR 80 LV analyser.

#### DATA ANALYSIS

The relations between the presence of multivessel coronary disease and resting left ventricular ejection fraction and clinical and exercise test variables were determined. The clinical variables assessed were a history of previous myocardial infarction, Killip classification<sup>15</sup> of the index infarction, and cardiomegaly, which required a cardiothoracic ratio of >50% on the predischARGE chest x ray film. Differ-

Table 1 *Clinical and exercise test variables related to the presence or absence of multivessel coronary disease. Figures are numbers of patients (n=62)*

	Present	Absent
Site of infarction:		
Anterior	14	6
Inferior	22	7
Non-Q wave	7	6
Previous infarction	8	1
Killip classification		
I or II	37	15
III or IV	6	4
ST depression:		
Total		
Present	25	9
Absent	18	10
After anterior infarction:		
Present	4	2
Absent	10	4
After inferior infarction:		
Present	17	4
Absent	5	3
After non-Q wave infarction:		
Present	4	3
Absent	3	3
ST elevation:		
Present	15	5
Absent	28	14
Inadequate blood pressure response:		
Present	18	1
Absent	25	18**
Inability to complete 5 metabolic equivalents:		
Yes	27	6
No	16	13*

\*p<0.05; \*\*p<0.01.

Table 2 *Left ventricular ejection fraction (LVEF), according to clinical and exercise test variables. Values are mean (SD) (n=60)*

	LVEF(%)
Site of infarction:	
Anterior (n=20)	47.9 (15.3)
Inferior (n=27)	49.3 (12.0)
Non-Q wave (n=13)	59.5 (10.9)*
Previous infarction:	
Yes (n=8)	53.0 (17.2)
No (n=52)	50.7 (13.1)
Cardiomegaly:	
Present (n=13)	44.7 (15.7)
Absent (n=47)	52.5 (12.8)
Killip classification:	
I or II (n=51)	52.4 (13.4)
III or IV (n=9)	43.8 (11.8)*
Multivessel coronary disease:	
Yes (n=41)	50.5 (14.0)
No (n=19)	52.2 (12.9)
ST depression:	
Present (n=33)	50.8 (14.0)
Absent (n=27)	50.6 (13.1)
ST elevation:	
Present (n=20)	43.2 (13.3)**
Absent (n=40)	54.9 (11.8)
Inadequate blood pressure response:	
Present (n=18)	48.4 (15.5)
Absent (n=42)	52.2 (12.1)
Inability to complete 5 metabolic equivalents:	
Yes (n=31)	48.7 (15.0)
No (n=29)	53.4 (11.2)

\*p<0.05; \*\*p<0.01.

ences between the means of independent observations were assessed by Student's *t* test and the differences between proportions by the  $\chi^2$  test with Yates's correction.

#### Results

The only statistically significant predictors of multivessel coronary disease among the clinical and exercise test variables were an abnormal blood pressure response to submaximal exercise or limited effort capacity (Table 1). There was a trend towards multivessel disease in patients with previous myocardial infarction and in those with exercise induced ST depression after inferior myocardial infarction, which did not reach statistical significance. Exercise induced ST depression was a poor predictor of multivessel disease overall, as was exercise induced ST elevation.

Q wave infarction and high grade Killip classification were the clinical variables which were significantly associated with reduced left ventricular ejection fraction (Table 2). Of the exercise test variables, only exercise induced ST elevation predicted poor resting left ventricular function (Table 2). Patients with multivessel coronary disease did not have worse left ventricular function as a group than those who did not.

Twenty nine of the 34 patients who had exercise

induced ST depression had this change confined to leads other than those which showed ischaemic changes during the evolution of the acute infarction if transient ST depression in remote leads is considered reciprocal and not primarily ischaemic in origin. Conversely, all 20 patients with exercise induced ST elevation had this change induced in leads with acute infarction changes. Of these 20 patients with exercise induced ST elevation, 16 had anterior and four inferior infarction, and in 18 of the 20 exercise induced ST elevation was confined to leads bearing Q waves.

None of the 10 patients receiving beta blockers had exercise limited by excessive heart rate response. Nevertheless, the proportions of patients who had other exercise test variables were similar to those of patients not receiving beta blockers: limited by angina (10% vs 8%) or fatigue (20% vs 10%), exercise induced ST depression (40% vs 58%) or elevation (20% vs 35%), and inadequate blood pressure response (20% vs 33%). None of these differences was significant.

## Discussion

There is a high prevalence of extensive coronary artery disease in survivors of acute myocardial infarction. We have shown that ST depression induced by a submaximal exercise test before hospital discharge after acute myocardial infarction was a poorer indicator of the presence of multivessel coronary disease than previously reported.<sup>10-19</sup> Clinical variables were similarly poor predictors of multivessel coronary disease. An abnormal haemodynamic response to submaximal exercise in the form of limited effort capacity or an abnormal exercise blood pressure response predicted multivessel disease. Importantly, however, the absence of an abnormal haemodynamic exercise response was not a good indicator of the absence of multivessel disease. When resting left ventricular ejection fraction was considered, Q wave infarction and high grade Killip classification were clinical factors which were statistically significant predictors of impaired left ventricular function. A reduced resting left ventricular ejection fraction was also associated with exercise induced ST elevation but not exercise induced ST depression nor other exercise variables.

Patients in this series were consecutive survivors of acute myocardial infarction who had ST depression induced by early submaximal exercise testing or whom were investigated because of symptoms or young age. Ideally, the coronary anatomy would be investigated in every patient who survived myocardial infarction, but this is clearly difficult on many grounds, and studies of the type we report here are confined to selected series.<sup>10-19</sup> The prevalence of

multivessel disease among our patients was very similar to that in other reported series,<sup>4,20,21</sup> and it is unlikely that differences between our findings and those of other workers can be attributed to different proportions of patients with multivessel disease.

It is also of interest that the incidence of angina, ST depression, fatigue, and abnormal blood pressure changes with exercise was similar in a small group of patients receiving beta blockers and in the remaining patients. We therefore consider it is unlikely that the results can be explained by differences in medication, either within our own series or by comparison with other published results.

## CORONARY ANATOMY

Exercise testing early after myocardial infarction has been used to predict the presence of multivessel coronary disease.<sup>10-19</sup> Each of these studies has reported results in selected populations: survivors of uncomplicated infarction,<sup>16,17,19</sup> consecutive patients fulfilling specified criteria but an undefined proportion of the total number of patients with acute myocardial infarction,<sup>10</sup> and non-consecutive patients who were investigated by coronary arteriography to assess postinfarction angina or "to assess the extent of coronary disease and left ventricular performance."<sup>18</sup> Criteria for a positive exercise test, and hence a predictor of multivessel disease, have included ST segment depression alone,<sup>19</sup> ST depression or exercise induced angina or both,<sup>10,17,18</sup> or inability to complete a very low workload exercise test protocol because of the occurrence of angina, ST segment changes, or ventricular arrhythmias.<sup>16</sup> In this series, if exercise induced ST depression early after myocardial infarction was considered a positive exercise test result for the detection of multivessel coronary disease predictive values of positive and negative tests were lower than previously reported (Table 3). Several factors might explain this difference. Dillahunt and Miller excluded patients unable to complete their low workload treadmill protocol for "non-cardiac reasons."<sup>16</sup> Survivors of inferior myocardial infarction, the site in which exercise induced ST depression may be most

Table 3 Comparison of predictive values of positive and negative exercise tests for the prediction of multiple vessel coronary disease

	No of patients	Positive* (%)	Negative (%)
Dillahunt and Miller 1979 <sup>16</sup>	28	76	73
Schwartz <i>et al</i> 1981 <sup>10</sup>	48	90	42
Fuller <i>et al</i> 1981 <sup>17</sup>	40	87	72
Starling <i>et al</i> 1981 <sup>18</sup>	57	88	69
Akhras <i>et al</i> 1982 <sup>19</sup>	61	98	88
Present study	62	74	36

\*See text for definition.

sensitive for detecting multivessel disease, comprised more than half the total number of patients (21/40) reported by Fuller *et al.*<sup>17</sup> Nearly a third (15/48) of the patients described by Schwartz *et al* had sustained non-Q wave infarction and nearly a quarter (5/21) of their positive exercise tests were so described because of exercise induced angina, without induced ST depression.<sup>10</sup> The importance of exercise induced angina for detecting multivessel coronary disease was stressed by Starling *et al.*<sup>18</sup> Their predictive accuracy for submaximal exercise detection of multivessel disease was 82% if exercise induced ST depression or angina was considered positive but fell to 60% if ST depression alone was analysed. Nevertheless, this difference in the definition of a positive exercise test would not have altered our results because only five of our 62 patients had angina induced by submaximal exercise, all of whom also had exercise induced ST depression. The most impressive correlation between a positive exercise test early after myocardial infarction and multivessel coronary disease has been that of Akhras *et al.*<sup>19</sup> Their young survivors of uncomplicated infarction performed a symptom limited, rather than submaximal, exercise test a mean time of two weeks after the acute infarction. A positive test required exercise induced ST depression of at least 0.15 mV. The use of a symptom limited exercise test protocol may have contributed to the very high accuracy of their exercise tests in predicting multivessel disease, although heart rate limited and symptom limited exercise tests conducted early after acute myocardial infarction have a similar yield of exercise induced ST depression.<sup>22</sup> As these authors themselves point out<sup>19</sup> it may not be appropriate to extrapolate their results to all survivors of acute myocardial infarction.

There are usually major persisting electrocardiographic abnormalities after acute myocardial infarction. In our patients exercise induced ST depression occurred predominantly in leads other than those which showed evolutionary changes of acute myocardial infarction. As the anterior wall of the heart is the area which is best mapped by the standard 12 lead electrocardiogram this may explain why exercise induced ST depression occurred more often after inferior infarction than after anterior infarction.

Advocates of the non-invasive prediction of multivessel coronary disease early after myocardial infarction suggest that this affords a method of selecting patients for early coronary arteriography,<sup>10 17-19</sup> presumably as a prelude to coronary artery bypass surgery in those with suitable anatomy. Akhras *et al* suggest that the same criteria for selecting patients for surgical revascularisation should be used after infarction as in patients with angina,<sup>19</sup> in whom surgery may offer improved prognosis in the presence of three

vessel coronary artery disease.<sup>13</sup> Nevertheless, this last multicentre study included patients who were not only symptomatic but had left ventricular ejection fractions >50%, thereby excluding any patient with significant left ventricular damage and many with preceding myocardial infarction. Therefore, the benefits of surgical treatment in survivors of myocardial infarction with multivessel disease who have greater impairment of left ventricular function remain uncertain.<sup>1</sup>

#### LEFT VENTRICULAR PERFORMANCE

Exercise induced ST elevation was a poor predictor of multivessel coronary disease. Mean resting left ventricular ejection fraction was, however, significantly lower in the group of patients with ST elevation induced by submaximal exercise but not in the group with exercise induced ST depression. Similar results have been reported.<sup>10</sup> Nevertheless, a reduced resting left ventricular ejection fraction has also been found in patients with exercise induced ST depression.<sup>17</sup> The importance of ST elevation induced by exercise after myocardial infarction is controversial,<sup>23 24</sup> but it may be an indicator of an increased probability for future complications.<sup>10 11</sup> The lower left ventricular ejection fractions in our patients with exercise induced ST elevation suggest that this group of patients has more extensive myocardial damage and this may result in a worse prognosis. As left ventricular ejection fraction was similar in each of the two groups who had sustained either anterior or inferior infarction, it seems unlikely that exercise induced ST elevation was merely a marker of anterior infarction, although it occurred predominantly in this group.

An abnormal blood pressure response to submaximal exercise early after myocardial infarction is reported to predict multivessel disease in association with reduced left ventricular ejection fraction.<sup>18</sup> Using the same definition of inadequate blood pressure response to exercise and a similar exercise protocol, we found that this variable predicted multivessel coronary disease but could not confirm a lower resting left ventricular ejection fraction of statistical significance in this group, although there was a trend in this direction. Inability to complete the submaximal exercise protocol predicted multivessel coronary disease in our patients, together with a trend towards greater impairment of resting left ventricular function. We did not measure left ventricular ejection fraction during exercise. This is more likely to correlate with poor exercise performance than variables measured at rest. The association of extensive coronary artery disease with abnormal exercise haemodynamic response is consistent with reports that poor exercise duration and abnormal blood pressure response to exercise early after infarction may be

of adverse prognostic significance.<sup>8 25</sup>

#### CLINICAL IMPLICATIONS

It has been suggested that a suitable screening procedure to select those survivors of acute myocardial infarction who should undergo coronary arteriography might be the performance of an exercise test early after infarction, followed by gated blood pool imaging in those who have ST depression induced by exercise, to exclude those with severe left ventricular damage from invasive investigation.<sup>26</sup> Our data suggest that exercise testing based on ST segment changes is a poor predictor of multivessel coronary disease unless haemodynamic factors are considered. Even then a large proportion of survivors of acute myocardial infarction with multivessel disease will not be detected. It is unlikely, however, that survivors of myocardial infarction with multivessel disease and similar degrees of resting left ventricular functional impairment have a uniform prognosis. It may be more appropriate to use other physiological measurements, such as thallium scintigraphy,<sup>27</sup> which are more specific detectors of reversible regional myocardial ischaemia to determine prognosis and select appropriate surgical or medical management or both.

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