

# A controlled trial of community based coronary rehabilitation

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## Abstract

**Two hundred patients who had suffered an acute myocardial infarction 4-6 weeks before entered a randomised controlled trial of exercise treatment at a community sports centre supervised by a general practitioner. Eighty one per cent of the treatment group continued to exercise until they returned to work and 73% completed three months' exercise. There were no serious complications of the exercise course. The prevalence of angina pectoris fell by 10% in the treatment group but rose by 60% in the control group. The perceived energy level rose by significantly more in the treatment group than in the controls. The rise in predicted maximum oxygen uptake was significantly greater in the treatment group than in the control group as was the reduction in the double product (a reflection of myocardial workload) at peak exercise.**

**Coronary rehabilitation in the community can be both safe and effective.**

The benefits of exercise training for patients with coronary heart disease are widely recognised. Numerous studies of this treatment have shown these effects and the mechanism of their production in selected groups of patients. There have, however, been very few large scale randomised controlled trials in unselected patients recovering from acute myocardial infarction<sup>1-9</sup> and only one in the United Kingdom—and that was hospital based.<sup>6</sup>

The Alton Coronary Rehabilitation Unit (which operates from a community centre and is supervised by a general practitioner, a sports officer, and a physiotherapist) opened in 1976. The purpose of the trial in this unit was to assess the results of managing patients after myocardial infarction.

## Patients and methods

All male patients aged 65 or less admitted to Basingstoke District Hospital from 1 December 1979 with a provisional diagnosis of acute myocardial infarction were recruited to the study. Fasting blood samples taken on the first morning after admission were sent for measurement of cholesterol and triglyceride concentrations. Serial measurements of serum aspartate transaminase activity and electrocardiograms were performed on the first three

days of admission. A chest x ray was taken within 48 hours of admission. By the fifth day of admission a positive diagnosis of acute myocardial infarction was made only if all of the following criteria were met: a history of chest pain typical of myocardial infarction, progressive electrocardiogram changes, and a rise and fall in aspartate transaminase concentrations with at least one reading above 40 units/ml. Patients were excluded if they lived more than 25 miles from Alton, if they had medical or orthopaedic problems that precluded their taking part in the exercise course, if they had insulin dependent diabetes mellitus or were in atrial fibrillation, if they had previously been through the course, or if they were on the investigator's personal general practice list.

The qualifying patients were randomised by order of admission into treatment and control groups by means of a random letter sequence. The nature of the trial was then explained and the patient was invited to join. At 4-5 weeks after admission all patients were interviewed and examined by one of two research assistants (one a state registered nurse, the other a state registered physiotherapist) who measured height and weight and, with the patient sitting down, recorded resting pulse rate and blood pressure. The patients were asked about symptoms and, if not mentioned specifically, chest pain. The occurrence of tight central chest pain or throat pain brought on by exercise and relieved within a few minutes by rest was recorded as angina. The patients were also asked to express their current well-being and energy level as a percentage of their normal level. One to two weeks later they underwent a submaximal bicycle ergometer exercise test at Alton Sports Centre. Entry to the trial was stopped when 200 patients had been tested. All exercise tests were performed on a mechanically braked Cardionics ergometer with a continuous multistage test with five minutes at each exercise load. The starting load was estimated to allow each patient to complete at least one stage but not more than three stages; the load was increased by 25 W at each level before the end point was reached. The test was terminated when the patient reached 85% of his predicted maximum heart rate, unless it was interrupted by angina pectoris, multifocal ventricular extrasystoles, exhaustion, or excessive breathlessness. Heart rates at the end of the test were calculated from six consecutive complexes on the electrocardiographic write-out (Hewlett-Packard) and for systolic pressure we used a Philips electronic analogue sphyg-

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momanometer calibrated from time to time against a mercury sphygmomanometer. Patients on short acting  $\beta$  blocking drugs stopped taking these drugs 48 hours before the test unless this was contraindicated. We calculated the predicted maximum oxygen uptake ( $\dot{V}O_2$  max) from the exercise test using the Astrand-Ryhming nomogram.<sup>10</sup> This could be done only for those who cycled for at least three minutes at their lowest load and for those who were not on  $\beta$  blocking drugs at the time of the test. The double product (heart rate  $\times$  systolic pressure) at the end of the test was also calculated.

Control patients were given a short talk on the sort of exercise that they might safely take unsupervised. Treatment patients entered a three month course of three times a week circuit training at Alton Sports Centre. The course has been described previously.<sup>11</sup>

Three months from the initial interview the patient was seen again by the research assistant who repeated the initial interview and examination. A second exercise test was performed within the next week, supervised by HJNB, on the same day of the week and at the same time of the day as the first test and with the same protocol.

Mann-Whitney U tests and two-sample  $t$  tests were used to test for differences between the two groups (controls and treated) and Wilcoxon signed-rank tests or paired  $t$  tests were used to test for differences within the groups. Chi-squared test with Yates's correction factor and McNemar's test were also used when appropriate. Ninety five per cent confidence intervals for  $\chi^2$  tests were calculated according to the method described by Armitage and Berry.<sup>12</sup>

Most of the statistical analyses were computed with the statistical packages SPSS-X<sup>13</sup> and Minitab.<sup>14</sup>

**Results**

The figure shows the outcome in the 311 male patients aged  $\leq 65$  who were admitted to the Basingstoke District Hospital Coronary Care Unit between 1 December 1979 and 1 March 1984 with a confirmed diagnosis of acute myocardial infarction. Twenty eight died before randomisation and 54 were excluded for one of the following reasons: geographical (36), medical/orthopaedic contraindications (seven), insulin dependent diabetes mellitus (three), atrial fibrillation (two), previous graduates of the course (three), and on the author's general practice list (three). The remaining 229 were randomised—113 to the treatment group and 116 to the control group. Of the 113 treatment patients, 99 attended Alton Sports Centre for the first exercise test (five died, four refused, and five developed other problems). Of the 116 control patients, 101 attended Alton Sports Centre (seven died, three refused, and five developed other problems). Thus of the 200 patients in the study, 99 were in the treatment group and 101 in the control group, and these patients comprised 85% of the infarct patients who had survived to the time of the first test and lived within 25 miles of Alton.

COMPARABILITY OF GROUPS (TABLES 1 AND 2)

The two groups were comparable in terms of age, presence of Q waves on the electrocardiogram, aspartate transaminase concentration, presence of pulmonary oedema, presence of complications, initial  $\dot{V}O_2$  max, and time to return to work.

FOLLOW UP

Of the 99 treatment patients two died and four could not be followed up. One of those patients who died did so between the final interview and the final exercise test and his interview and examination findings are included. Of the re-

Outcome in 311 male patients aged 65 or less who were admitted to the Basingstoke District Hospital coronary unit between 1 December 1979 and 1 March 1984 with a confirmed diagnosis of acute myocardial infarction.

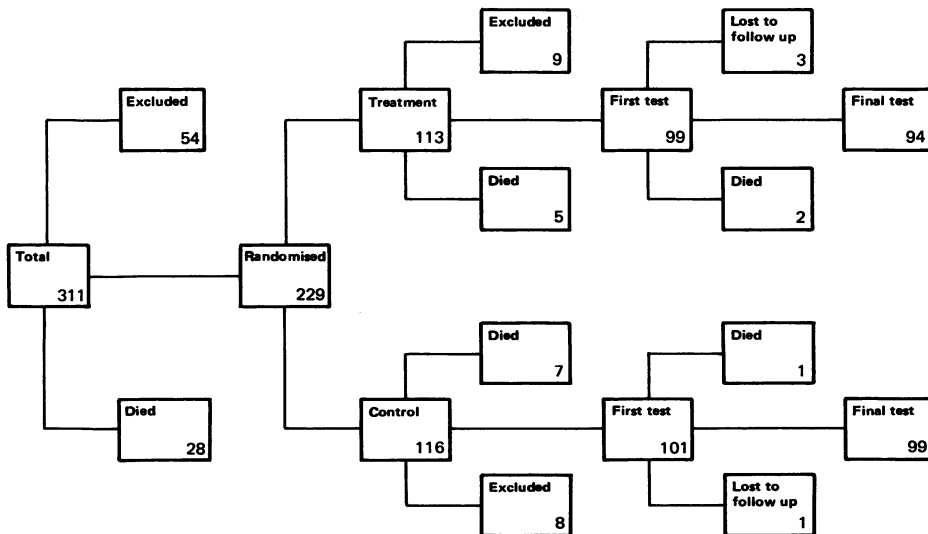


Table 1 Comparability of the two groups

	Mean age (yr) (SD)	Median peak AST (IU/l) (interquartile range)	Pulmonary oedema on chest x ray	Complications*	Mean initial $\dot{V}O_2$ max (ml/min/kg) (SD)	Mean time to return to work (wk) (SD)
Treatment group:						
n	99	99	99	99	78	63
	54.2 (7.2)	202 (122, 327)	11%	27%	22.2 (5.0)	13.7 (3.7)
Control group:						
n	101	101	100	101	80	66
	53.2 (7.7)	207 (114, 302)	11%	27%	23.4 (4.8)	13.3 (3.6)

\*Clinical left ventricular failure, ventricular fibrillation, ventricular tachycardia, atrial fibrillation, heart block, and pericarditis.

maining 93 patients, 73 completed the 12 week exercise course, eight defaulted on return to work, four defaulted at other times, and six were withdrawn for medical reasons (one re-infarct, one left ventricular failure, one increasing angina and three orthopaedic problems). Thus 73 completed the course and 81 continued to exercise at least until they returned to work.

Of the 101 control patients, one died and one refused follow up. By the end of the 12 weeks of the trial, 27 of these were taking unsupervised, apparently vigorous exercise at least twice a week. These exercises included bicycling, using a bicycle ergometer, press ups and sit ups, swimming, jogging, and weight training.

#### CHANGES IN OBSERVATIONS (TABLE 3)

There were no significant differences between the two groups for change in frequency of sexual intercourse (Mann-Whitney U,  $p = 0.40$ ), resting heart rate ( $t = 0.64$ ,  $df = 173$ ,  $p = 0.52$ ), blood cholesterol ( $t = 0.69$ ,  $df = 161$ ,  $p = 0.49$ ), or triglyceride concentration (Mann-Whitney U,  $p = 0.88$ ). The mean increase in weight of the treatment patients was significantly greater than in the control patients (difference = 1.2 kg) (95% CI for difference = 0.06 to 1.98;  $t = 2.09$ ,  $df = 191$ ,  $p = 0.04$ ).

Angina pectoris was present at the first follow up in 21 patients in the treatment group and in 20 control patients. By the second follow up angina was present in 19 (-10%) of the treatment patients but in 32 (+60%) of the controls. The increase in the occurrence of angina in the control group was significant (difference in proportions = 0.12) (McNemar's  $\chi^2 = 7.56$ , 95% CI = 0.04 to 0.20;  $df = 1$ ,  $p = 0.01$ ).

The median energy level for the treatment group rose from 50% before the exercise course to 95% after the exercise course with a median rise of 41%. The median energy level for the controls rose from 50% to 90% with a median rise of 25%. The median energy level was significantly greater in the treatment group than in the controls (difference = 10.00%) (Mann-Whitney U 95% CI = 0.00 to 21.92;  $p = 0.03$ ).

Fifty five of the treatment group and 60 of

the controls had been cigarette smokers before their infarcts. By the end of the period of observation, 43 (78%) of the smokers in the treatment group and 52 (87%) in the control group said that they no longer smoked.

Among patients in the treatment group, of the 42 smokers who finished the exercise course, six (14%) continued to smoke, while of the 13 smokers who failed to finish the course six (46%) continued to smoke (difference in proportions = 0.32) ( $\chi^2 = 4.19$ , 95% CI = 0.08 to 0.66;  $df = 1$ ,  $p = 0.04$ ).

#### CHANGES IN EXERCISE TEST RESULTS

In the treatment group, the mean predicted maximum oxygen uptake rose significantly from 22.16 to 27.32 ml/min/kg (paired  $t = 9.69$ ,  $df = 77$ ,  $p < 0.001$ ). In the control group, the mean predicted maximum oxygen uptake also rose significantly from 23.36 to 26.16 ml/min/kg (paired  $t = 5.71$ ,  $df = 79$ ,  $p < 0.001$ ). The mean difference in the increase of predicted maximum oxygen uptake was 2.35 ml/min/kg, which was significantly greater (95% CI for the difference = 0.92 to 3.78;  $t = 3.24$ ,  $df = 156$ ,  $p = 0.001$ ) in the treatment group than in the control group.

In the treatment group the mean double product at peak exercise decreased significantly from 246 to 212 (paired  $t = -8.71$ ,  $df = 84$ ,  $p < 0.001$ ). In the control group the mean double product decreased from 230 to 228 (paired  $t = -0.54$ ,  $df = 84$ ,  $p = 0.59$ ). The decrease in the mean double product was significantly greater in the treatment group than in the control group, difference = -32.58 (95% CI = -42.19 to -22.97;  $t = -6.70$ ,  $df = 168$ ,  $p < 0.001$ ).

#### Discussion

In the United Kingdom, coronary rehabilitation courses are available to few patients recovering from cardiac surgery or myocardial infarction. The number has grown considerably since the British Cardiac Society review of 1970,<sup>15</sup> but many existing courses were established or initiated by nurses and physiotherapists with little support, and sometimes opposition, from doctors.<sup>16</sup> Such courses may, therefore, be underused and their effectiveness is seldom measured. This trial attempted to assess the effects of a community based exercise programme for patients immediately after infarction; it showed that most patients can participate (85% of patients who lived within 25 miles of the sports centre took part). Nearly all the treatment group embarked upon the

Table 2 Comparability of electrocardiographic changes in the two groups

	Anterior	Inferior	Lateral	Q waves
Treatment group (99)	47	45	7	73
Control group (101)	47	50	4	73

Table 3 Changes in important observations of the two groups

	Median frequency of sexual intercourse per month (interquartile range)	Mean resting heart rate (beats/min) (SD)	Mean cholesterol (mmol/l) (SD)	Median triglyceride (mmol/l) (interquartile range)	Mean weight (kg) (SD)
<b>Treatment group:</b>					
Number	94	88	81	81	94
Before	2.0 (0.0, 4.0)	79.5 (13.5)	6.2 (1.3) (0.9, 1.8)	1.3	75.2 (11.4)
After	1.0 (0.0, 4.0)	76.5 (10.5)	6.3 (1.0)	1.4 (1.0, 1.9)	76.4 (10.9)
Difference (after - before)	0.0 (-1.0, 0.0)	-3.0 (11.7)	1.3 (10.6)	-0.2 (-0.3, 0.5)	1.2 (3.0)
<b>Control group:</b>					
Number	99	87	82	82	99
Before	2.0 (1.0, 6.0)	79.0 (12.0)	6.1 (1.1)	1.3 (0.9, 1.8)	76.9 (9.7)
After	1.0 (0.0, 4.0)	74.9 (12.4)	6.1 (1.0)	1.4 (1.0, 2.1)	77.1 (9.3)
Difference (after - before)	0.0 (-2.0, 0.0)	-4.1 (9.5)	0.2 (10.3)	0.2 (-0.4, 0.6)	0.2 (3.8)

exercise programme: 81% continued to exercise until they returned to work and 73% completed the course, despite the fact that Alton Sports Centre is 15 miles from the centre of population. This figure is similar to that of Carson *et al* who found that 70% of 151 exercising patients completed a hospital based exercise programme.<sup>5</sup> Higher compliance rates (85-100%) have been achieved in selected patients referred for exercise.<sup>11 17-20</sup> One problem in assessing the results of exercise training in randomised trials is the tendency for control patients to take up exercise—the so-called drop-ins.<sup>21</sup> In our case 27% of the controls were taking apparently vigorous exercise by the end of the trial, though this was probably much less intensive and effective than the supervised exercise in the treatment group.

As with most other controlled trials of exercise for coronary patients, there was no difference between the groups for blood pressure,<sup>7 22 23</sup> cigarette smoking,<sup>5 7</sup> blood cholesterol,<sup>4 5 24</sup> and time to return to work.<sup>14 5</sup> It was surprising that there was no difference between the groups for change in triglyceride concentrations, which are reduced for up to 48 hours by vigorous exercise.<sup>25</sup> We made no attempt to relate the time of blood sampling to a previous exercise session.

Unfortunately the exercise testing of patients could not be blind because HJNB performed all the tests and supervised all the exercise sessions. Heart rate measurements were taken from an electrocardiographic write-out and should not have been subject to bias. However, it is possible that peak systolic blood pressures, read from an analogue electronic sphygmomanometer, were subject to observer bias.

The improvement in physical fitness of the control patients as measured by the predicted maximum oxygen uptake, was expected. The recovery of the heart from the immediate effects of the infarct produces a spontaneous improvement over the first three months<sup>26</sup> associated with an increase in stroke volume.<sup>22</sup> The large number of drop-ins, perhaps a result of the Hawthorne effect,<sup>27</sup> probably had little effect on the increased fitness of the controls (see below). The greater improvement in fitness among the treatment group testified to the effectiveness of the exercise regimen, and was of a similar order to that achieved in hospital based rehabilitation programmes.<sup>23 5-9</sup> This

increase in physical work capacity produced by a short course of physical training is caused by peripheral adaptations rather than improved cardiac function.<sup>28-30</sup> There is an alteration in the distribution of blood within the working muscles and an increase in their capacity to extract oxygen, associated with an increase in the number and size of mitochondria, mitochondrial enzyme content, and capacity to metabolise various energy substrates.<sup>31 32</sup> The result of these changes is an increase in the arteriovenous oxygen difference of the blood flow to and from the working muscles.<sup>28 33-35</sup> Any given exercise load requires less blood to fuel it, with a reduction of demand on the heart and a lower heart rate.<sup>35 36</sup>

The reduction of exercise induced heart rate and blood pressure produced by exercise training of cardiac patients is a very important effect, because the heart rate  $\times$  systolic pressure product (the double product) is a direct reflection of myocardial workload and oxygen demand.<sup>37-39</sup> The double product at submaximal workloads does not decrease spontaneously after infarction because the fall in heart rate response is matched by an increase in blood pressure response.<sup>40 41</sup> Because the control patients in this study showed no change in double product at submaximal workload it is likely that their improved fitness was largely due to spontaneous cardiac recovery and that the unprompted exercising of the drop-ins was relatively ineffective. The appreciable fall (by 14%) in the submaximal double product among the treatment group was reflected in their reduction in clinical angina compared with the controls, an effect that has been demonstrated in other controlled trials.<sup>2 3 42</sup> However, in this trial the symptom of angina was determined from the patient's history given to a research assistant who also helped at exercise sessions. A standardised questionnaire was not used and the findings of this symptom, which showed a larger than expected difference between the two groups at the end of the course, may have been biased.

Nearly all reports on the results of coronary rehabilitation have emphasised the great improvement in well-being and in psychological health it engendered. Few, however, have quantified this improvement, and those who have measured the psychological impact of rehabilitation have shown either no change<sup>43</sup> or

rather minor benefit.<sup>44-47</sup> This study, too, found that the great improvement in subjective well-being among the treatment patients was nearly matched by the controls, implying that the overall effect of the physical training on this factor was modest. However, the combination of increased attention received by the controls<sup>48</sup> and the effect of the initial exercise test<sup>49,50</sup> may have given them a psychological advantage not shared by untreated patients who were not involved in controlled trials.

Most coronary rehabilitation programmes are based in hospitals, a few use home exercise,<sup>48</sup> and some are based in the community.<sup>2,51,52</sup> There are no published trials that compare the effectiveness or cost-effectiveness of hospital and community based programmes,<sup>53</sup> but in terms of adherence and measurable outcome the findings of our study are similar to those reported from hospitals. There are definite advantages in using community sports centres<sup>54</sup>—not only do they have more space than most hospitals but they also have the appropriate equipment for circuit training and have staff experienced in using it. An added benefit of exercising in the community is that it encourages a return to normal. The patient going to the sports centre is having fun rather than treatment and this should encourage compliance and, in the long term, lead to a habit of regular exercise.

This study showed the feasibility, the safety, and the benefits of a community based exercise programme supervised by a general practitioner for patients recovering from acute myocardial infarction. Every district general hospital should be able to offer this treatment to patients after myocardial infarction and those after coronary bypass graft but most do not. The community programme is one of the ways in which the gaps in the present provision could be filled.

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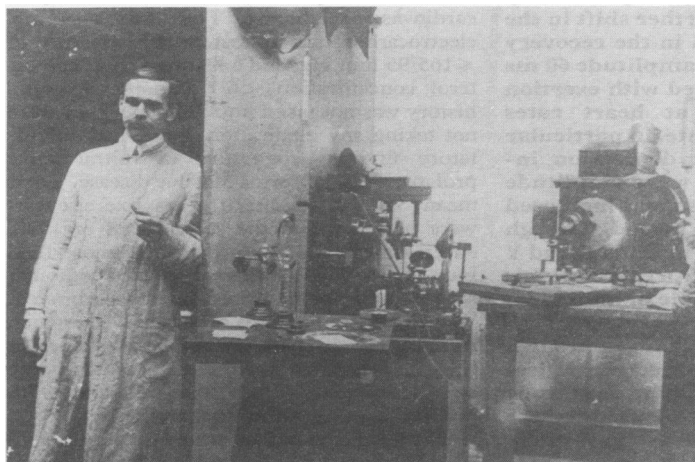
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## VIEWS FROM THE PAST *W A Jolly*

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W A Jolly studied electrocardiography with Einthoven in 1908. On his return to Edinburgh in 1909 he and W T Ritchie recorded the first electrocardiogram showing atrial flutter. When he took up the chair of physiology in Cape Town in 1911 he brought with him the first electrocardiograph to be installed in South Africa; it is seen with him in this photograph. Source: Department of Physiology, University of Cape Town.

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