ORIGINAL PAPER 5

Coronary rehabilitation in the community

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SUMMARY. Over a five-year period, 162 patients with coronary disease joined a physical rehabilitation course at a community sports centre gymnasium under the supervision of a general practitioner. One hundred and forty-seven patients had suffered a recent or old myocardial infarction and 15 suffered from angina pectoris. One hundred and thirty-eight patients (85 per cent) completed a three-month course of exercises, 16 (10 per cent) defaulted and eight (5 per cent) were withdrawn. One patient died at home during the three-month course. There were no changes in weight, blood pressure or blood fat measurements during the course but predicted maximum oxygen uptake increased by 26.9 per cent and the double product after effort (which is proportional to myocardial oxygen uptake) decreased by 13.6 per cent.

We believe that the rehabilitation of patients in community sports centres is safe and effective and should be more widely practised.

Introduction

THE management of acute myocardial infarction has evolved over the past 20 years from weeks of bed rest and inpatient care to rapid mobilization and early discharge from hospital. Most hospitals are concerned only with the control of the acute electrical and mechanical problems produced by the infarct, and they send the patient home as soon as the risk to his life is minimal.

When he leaves the safe womb of the coronary care unit, however, the coronary patient faces many problems including depression, fear, insecurity and uncertainty about how much he can do. Most British hospitals have little to offer him at this stage (Groden *et al.*, 1971). In many parts of the world, medically supervised physical training is used to increase his activities after discharge from hospital and to guide him back to mental and physical fitness. Although such courses are becoming more common in this country, they are still provided by very few centres. This paper describes one solution to this lack of amenities; the use of a community sports centre and its equipment under the supervision of a general practitioner.

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The rehabilitation course

The course was started in January 1976 by a general practitioner (H.J.N.B.) and a sports centre sports officer (A.L.) giving physical training to patients with a variety of problems from coronary disease—angina pectoris, recent infarction or debility from longstanding coronary disease (Bethell, 1976). After two years the course was offered to Basingstoke District Hospital (BDH) for rehabilitation of patients recovering from acute myocardial infarction, and since then most patients have come from the BDH coronary care unit.

A secretary/physiotherapist (S.C.T.) has joined the team to help with a controlled trial which is now in progress.

The course is held in Alton Sports Centre, about 15 miles from BDH. During the first four years it evolved as procedures were modified and new equipment obtained.

Before and after the course each patient is examined for weight, blood pressure and cardiovascular signs. He has an electrocardiogram recorded and fasting blood lipids measured. At the start and the end of the course he has a submaximal exercise test on a bicycle ergometer. This is a continuous, multistage test starting at 25 to 100 W (cycling at 50 rpm against a load of 0.5 to 2.0 kg) and increasing by 25 W every 5 minutes until either the pulse rate reaches 85 per cent of the predicted maximum for the patient or he is stopped by angina pectoris, breathlessness or exhaustion. When the patient can complete at least one stage, his pulse rate response is used to calculate his predicted maximum oxygen uptake ($\dot{V}O_2max$) according to the nomogram of Åstrand and Ryhming (1954). The double product (heartrate × systolic BP) at the end of the test is also calculated.

During the course the pulse is measured before and after each session using a Murray pulsometer, and once weekly the blood pressure is taken before and after the session using a Lumitronic electronic sphygmomanometer. (The noise made by the other patients exercising makes auscultation of the BP difficult.)

The exercises are performed as eight stages on a circuit: bicycling on an ergometer; stepping up and down two steps; an overhead pull of 20 kg; a squat lift against 40 kg; trunk curls; a quadriceps exercise against 20 kg; a bench press against 10–20 kg; and sitting leg press against 50 kg. These

weight training exercises are performed on a Nissen polygym and involve frequent rapid repetitions with small loads; they are dynamic, not like weight lifting, which is nearly isometric. At the start of the course the patient cycles on the ergometer at 50 W for 15 s and repeats each exercise five times. The number of repetitions and the number of circuits are built up gradually depending on his pulse rate response at the previous session. The aim is to exercise him to between 70 and 85 per cent of his predicted maximum heart rate. (The patient on a beta-blocker needs more careful exercising since his pulse rate does not reflect the effect that exercise has on him. We exercise him to an equivalent degree of breathlessness as those patients not on beta-blockers, and he is carefully watched for signs of exhaustion or hypotension. He is warned to stop exercising if he feels faint or unduly breathless.) When he is performing three circuits of 15 to 20 repetitions, his electrocardiogram is monitored during exercise using a radiotelemeter to assess his pulse rate response to each exercise and to detect arrhythmias and S-T segment changes. By the end of the course, most patients can cycle for 60 s at 100 W and perform each exercise 25 times, repeating this circuit three times. The whole session lasts between 20 and 30 minutes.

The course lasts between three and four months and at the end of this time the patient is encouraged to continue to take regular exercise unsupervised, either at the Sports Centre or elsewhere, by cycling, jogging or swimming. Since Alton Sports Centre is 15 miles from Basingstoke District Hospital most patients find it too far to continue to attend regularly, though some do continue to exercise on a similar circuit in Basingstoke Sports Centre.

The equipment includes a Murray pulsometer, a Hewlett Packard defibrillator, an ECG oscilloscope, a pen recorder and cardiotachometer and a radiotelemetry transmitter and receiver.

Results

From January 1976 to December 1980, 162 patients entered the rehabilitation course. All were male, average age 54 years (range 32 to 73 years). The diagnoses were: myocardial infarction (147 cases); angina pectoris without recent infarction (15 cases). Forty-five patients had been referred by their general practitioners and 117 came from the coronary care unit at Basingstoke District Hospital. Of 162 starters, 138 (85 per cent) completed the course, 16 (10 per cent) withdrew and eight (5 per cent) were withdrawn for the following reasons:

1. Two patients had episodes of ventricular fibrillation. Both were successfully defibrillated and showed no evidence of recurrent myocardial infarction. One patient returned to the course but had a second attack of ventricular fibrillation without infarction and was again resuscitated; it was thought best not to let him rejoin.
2. One patient died suddenly, about 12 hours after an exercise session.
3. Three patients had worsening angina.
4. One patient had worsening diabetic control.
5. One had mounting anxiety.

Clinical and biochemical findings

One hundred and thirty-eight patients completed the course, but two were not examined at the end of the course. The weight, blood pressure, cholesterol and triglycerides before and after the course are shown in Table 1. No significant change in any of these measurements was found.

Table 1. Changes in weight, blood pressure, blood cholesterol and triglycerides during the course.

	Number of tests	Before exercise	After exercise
Weight (kg)	136	75.3 ± 8.0	75.6 ± 8.0
Systolic blood pressure	119	132.3 ±21.3	132.6 ±20.6
Diastolic blood pressure	119	82.7 ±12.6	79.6 ±13.3
Cholesterol	126	66.60± 1.20	66.55± 1.17
Triglycerides	126	1.89± 0.85	1.85± 1.01

The blood pressure was not recorded in 17 cases because of change of medication which might have affected the result. The blood fats were not measured in 10; in one case because of change of clofibrate dosage and in nine for organizational reasons.

Table 2. Changes in double product and predicted $\dot{V}O_2$ max during the course.

	Number of tests	Before exercise	After exercise	Percentage change
Double product	63	26,221 ± 4,533	22,651 ± 4,134	*−13.6**
Double product (beta-blockers)	8	20,228 ± 5,594	18,895 ± 4,040	− 6.6
$\dot{V}O_2$ max	66	23.8 ± 5.5	30.2 ± 6.6	*+26.9**

*$P<0.001$.

The electrocardiograms improved in 79 patients, remained unchanged in 47 and worsened in seven, one of whom showed a full thickness myocardial infarct which had not been recognized clinically during the course.

Exercise testing

From January 1979 to December 1980, 100 patients entered the course, 84 of whom completed it. Seventy-eight patients had exercise tests before and after the course. The tests were missed in two cases and not done in four because of change in beta-blocker dosage. The results are shown in Table 2.

The double product was measured in 63 patients who did not take beta-blockers. After the course, this had reduced by 13.6 per cent at the end of a measured workload ($P<0.001$).* For the patients on beta-blockers, the reduction was only 6.6 per cent (not significant).

*Significance testing was performed by carrying out a two-tailed t test on paired samples.

The predicted maximum oxygen uptake was calculated in 65 patients. (Of the rest, nine were on beta-blockers and four failed to complete the first stage of the exercise test.) The $\dot{V}O_2$ max increased by 26.9 per cent by the end of the course ($P<0.001$).

There was no correlation between age and increase in $\dot{V}O_2$ max. There was a weak negative correlation between the starting $\dot{V}O_2$ max and the percentage increase in $\dot{V}O_2$ max ($R=-0.28$).

Discussion

Physical training for the post-infarction patient has become more common over the past 25 years, and numerous reports of its benefits have been produced. Different ways to help patients include:

1. *Improved wellbeing.* Nearly all reports of coronary rehabilitation programmes emphasize this effect (Clausen *et al.*, 1969). The psychological advantages of physical fitness are particularly prominent in coronary patients, who are usually demoralized and fearful of exertion after the attack—the exercise course is an antidote to the inevitable depression of the post-infarction phase (Hackett and Cassem (1978)). The help given by physical training is extended by the group therapy provided by the patients and their wives meeting regularly at the gymnasium and discussing mutual problems.

2. *Increased physical fitness.* Exercise training in normal subjects produces improvements in cardiac performance as well as more efficient oxygen utilization by skeletal muscle (De Maria *et al.*, 1978; Ehsana *et al.*, 1978). Most studies of coronary patients after training show no change in myocardial performance (Bruce *et al.*, 1977; Letac *et al.*, 1977; Lee *et al.*, 1979), though almost all show increased exercise tolerance (Clausen *et al.*, 1969; Naughton *et al.*, 1969; Sanne, 1973; Ferguson *et al.*, 1974; Fletcher and Cantwell, 1975; Nixon *et al.*, 1976; Latac *et al.*, 1977; Stein, 1977; De Busk *et al.*, 1979; Lee *et al*, 1979). This is mediated by the effects of training on skeletal muscle alone, with more efficient distribution of blood and greater oxygen extraction (Clausen *et al.*, 1969; Detry *et al.*, 1971; Bruce *et al.*, 1977). For a given amount of exercise the pulse rate and cardiac output (and hence the myocardial oxygen need) are lowered by training; and for a given pulse rate and cardiac output more muscular work can be performed.

The pressure rate product (double product) which is the product of pulse rate and systolic pressure during exercise correlates well with myocardial oxygen uptake (Sarnoff *et al.*, 1958). A fall in the double product at a given workload after physical training is, therefore, good evidence of lessened demand on the heart at that workload. Our patients showed a significant fall of 13.6 per cent in the double product at the end of a submaximal exercise test, and this is similar to the falls produced by other exercise programmes (Letac *et al.*, 1977; Ben Ari *et al.*, 1978; Ferguson *et al.*, 1978).

The best measure of physical fitness is the maximum oxygen uptake ($\dot{V}O_2$ max), which may be expressed as litres per minute or ml per min per kg bodyweight. Since we did not have the facilities for measuring this directly, we used the Åstrand-Ryhming nomogram (1954) to predict the $\dot{V}O_2$ max of our patients from their pulse rate response to a submaximal exercise test. The increase of 27 per cent from start to finish of the course was highly significant, and within the range (10–40 per cent) produced by other courses (Ferguson *et al.*, 1974; Stein, 1977; Haskell and de Busk, 1979). It is equivalent to the improvement achieved by an unfit athlete from the start to the peak of his training. We cannot prove that the improvement in our patients' physical performance was due to the exercise programme rather than to natural improvement. We are now conducting a controlled trial of exercise rehabilitation after myocardial infarction to clarify this point.

Spin-off benefits of this increased physical fitness and improved well-being include earlier return to work (Fletcher and Cantwell, 1975; Schiller and Baker, 1976) and quicker return to full sexual function (Stein, 1977; Kavanagh and Shepherd, 1977). Physical training should also help to counteract the cardiac neurosis which is the culprit in about 50 per cent of those who have not returned to work by six months after the acute attack (Nagle *et al.*, 1971).

3. *Reduced angina pectoris.* The increased physical fitness produced by physical training allows the patient to do more exercise for a given rise in pulse rate and he can therefore do more before suffering chest pain. Exercise tolerance may increase by 100 per cent in angina patients (Sanne, 1973) and many lose their symptoms altogether (Ferguson *et al.*, 1974; Ferguson *et al.*, 1978; De Busk *et al.*, 1979; Lee *et al.*, 1979). We did not measure the angina threshold in our patients but, of those who had a formal exercise test, all but one had a greatly increased exercise tolerance.

4. *Reduced risk factors.* Weight loss after exercise has been reported (Mann *et al.*, 1969) but loss of excess fat may be masked by increased muscle bulk. Our patients did not lose weight, perhaps for this reason. Blood pressure may fall, particularly in hypertensive patients (Clausen *et al.*, 1969; Gottheiner, 1969; Naughton, 1969; Nixon *et al.*, 1976; Letac *et al.*, 1977). Our patients were nearly all normotensive and showed no fall in blood pressure. However, some rise might have been expected during this recovery phase and this could have been prevented by the exercise course.

Blood cholesterol after physical training has been found to fall (Naughton and Balke, 1964; Hoffman *et al.*, 1967; Clausen *et al.*, 1969; Mann *et al.*, 1969; Nixon *et al.*, 1976), rise (Ferguson *et al.*, 1974) or remain unchanged (Holloszy *et al.*, 1964; Fitzgerald *et al.*, 1965; Fletcher and Cantwell, 1975). Our patients showed no change. Triglycerides, however, have been found to be reduced by physical training in most studies

(Holloszy *et al.*, 1964; Hoffman *et al.*, 1967; Ferguson *et al.*, 1974; Fletcher and Cantwell, 1975; Huttunen *et al.*, 1979), and this effect seems to be an acute result of exercise lasting from two hours for two days after the exercise. Regular exercise is required to maintain it. The fall in triglycerides in our patients was small and not significant. Serum high density lipoproteins are increased by training (Huttunen *et al.*, 1979). We were unable to measure this effect in our patients through lack of laboratory facilities.

5. *Other benefits.* Ventricular irritability is lessened by physical fitness so that ventricular ectopic beats at rest and with exercise are less frequent in patients who have had physical training after myocardial infarction than in those who have not (Schiller and Baker, 1976; De Busk *et al.*, 1979), and this may reduce the risk of sudden death. No controlled trial of exercise therapy after infarction has shown a statistically significant improvement in survival rate in the trained group, though several studies have suggested that there may be (Gottheiner, 1969; Sanne, 1973; Wilhelmsen *et al.*, 1975; Kavanagh *et al.*, 1979). Certainly there is good evidence that vigorous exercise is important in the primary prevention of coronary heart disease (Paffenbarger *et al.*, 1977; Morris *et al.*, 1980).

Exercise after myocardial infarction is safe provided that it is dynamic, not isometric, that it is increased gradually and that its intensity is not too great. The most serious complication is ventricular fibrillation, which was found by Haskell (1978), in a survey of 30 cardiac rehabilitation courses, to occur once every 32,593 patient-hours of exercise. The survey included several programmes with continuous patient monitoring, which considerably reduces the risk of ventricular fibrillation. Our three attacks of ventricular fibrillation occurred in approximately 10,000 patient-hours, about 10 times the expected frequency, but the numbers are too small to draw conclusions. No episode of ventricular fibrillation was associated with myocardial infarction, which is an even rarer complication of exercise training (Haskell, 1978). Another potential hazard is musculoskeletal strain. This developed in 49 per cent of exercisers in one study (Mann *et al.*, 1969) and over 20 per cent of these dropped out because of it. This was not a significant problem in any of our patients and we attribute this to the wide variety of exercises performed so that no one muscle group was worked excessively, and to the gradual way in which exercise was increased. Several of our patients on beta-blockers developed quite severe hypotension after exercise, most likely to occur if they also took glyceryl trinitrate prophylactically. These patients all recovered within 30 minutes if laid flat. This problem might become more significant if prophylactic beta-blockade after myocardial infarction becomes standard treatment.

The drop-out rate for exercise courses varies from 10 per cent (De Busk *et al.*, 1979) or less to over 40 per cent, (Oldridge *et al.*, 1978) and most who default do so within six weeks. Those who drop out are those at greatest risk for recurrent infarction (Oldridge *et al.*, 1978; Kavanagh *et al.*, 1979). Our drop-out rate of 10 per cent was low considering that the centre of the population we were treating was 15 miles from Alton Sports Centre. We attribute this to the variety of exercises, to the out-of-hospital environment which made the exercise more like fun than treatment, and to the benefit which the patients derived from the course.

There is great variation in the design of coronary rehabilitation programmes. Some start rehabilitation two days after infarction (Tirlapur *et al.*, 1979) while others wait as long as five months (Rechnitzer *et al.*, 1972). Most courses involve walking, jogging, cycling and calisthenics (Ferguson *et al.*, 1978; Haskell and de Busk, 1979), but some use games (Gottheiner, 1969; Ferguson *et al.*, 1974). Programmes confined to only one or two activities train only a few muscle groups which may not be those most useful to the patient on his return to work and to his leisure activities. Our course has been planned to exercise as many different muscle groups as possible so that patients who work and play with their hands and arms receive as much benefit as those whose legs perform their chief muscular efforts. The number of sessions per week varies from one to five (Haskell and de Busk, 1979) and three seems about right since more frequent exercise does not produce greater fitness (Mann *et al.*, 1969) but does increase the risk of musculoskeletal complications (Hellerstein and Franklin, 1978). Exercise sessions usually last from 20 minutes to one hour; longer sessions also increase musculoskeletal complications without improving fitness (Hellerstein and Franklin, 1978). Optimal exercise intensity pushes the patient's pulse rate to between 70 and 85 per cent of his predicted maximum heart rate. Less vigorous exercise is inadequate to provide maximum fitness while more vigorous exercise carries a higher risk of cardiac complications without improving fitness further (Hellerstein and Franklin, 1978).

Nearly all coronary rehabilitation courses are run by hospitals using their own facilities, though some are run outside hospitals (Fletcher and Cartwell, 1975) and a few by general practitioners (Rucker, 1980). In this country very few hospitals provide this facility and it may be appropriate for general practitioners to set up more courses throughout the country, based on community sports centres. The rough cost today of the important pieces of equipment for the running of a coronary rehabilitation programme are: portable defibrillator, £960; portable oscilloscope, £600; bicycle ergometer, £200; hand-held pulsometer, £100. Such courses are cheap (ours costs £20 per patient); they employ the expensive equipment needed for physical training which is found in sports centres but not in hospitals; and being away from hospital they should seem more like fun than treatment and thus encourage patient compliance and long-term exercising.

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