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Improvement in maximal isokinetic cycle ergometry with cardiac rehabilitation

NEIL B. OLDRIDGE, NEIL MCCARTNEY, AUDREY HICKS, and
NORMAN L. JONES

Departments of Physical Education and Medicine,
McMaster University,
Hamilton, Ontario, CANADA

ABSTRACT

OLDRIDGE, N. B., N. MCCARTNEY, A. HICKS, and N. L. JONES. Improvement in maximal isokinetic cycle ergometry with cardiac rehabilitation. *Med. Sci. Sports Exerc.*, Vol. 21, No. 3, pp. 308–312, 1989. It is unclear whether improvements in short-term (30 s) exercise capacity are associated with the increased aerobic exercise tolerance frequently observed in cardiac patients following training. Carefully selected patients with documented coronary artery disease were randomly allocated either to a control group ($N = 10$) or to 12 wk of endurance exercise training ($N = 12$); both progressive incremental cycle ergometer testing (maximal power output and peak $\dot{V}O_2$) and 30 s maximal isokinetic cycle ergometry (peak power, total work, and fatigue index) were measured on entry into the study and 12 wk later. Initial maximum performance measures in progressive incremental exercise and in maximal short-term isokinetic cycling were similar in both groups. Following the training program, maximum power output measured during progressive incremental exercise increased by 21% ($P < 0.005$) and peak $\dot{V}O_2$ increased by 18% ($P < 0.005$) in the exercise group, but they were unchanged in the control group. Isokinetic peak power and total work improved by 14% ($P < 0.001$) and 11%, respectively, in the exercise group, whereas there were corresponding reductions of 6 and 8% in the control subjects, with little change in fatigue index in either group. The similar relative increases in isokinetic peak power and peak $\dot{V}O_2$ suggest that improvement in short-term exercise capacity may be an important contributor to the improvement in aerobic exercise tolerance frequently observed in cardiac patients undergoing an endurance exercise program.

CORONARY ARTERY DISEASE, REHABILITATION, MAXIMAL EXERCISE TESTING, ISOKINETIC CYCLE ERGOMETRY, PROGRESSIVE INCREMENTAL CYCLE ERGOMETRY

Patients taking part in cardiac exercise rehabilitation programs often report a perception of improvement in activities of daily living that is more dramatic than the accompanying increases observed in maximal oxygen intake. Although the improvement in maximal oxygen intake observed following endurance training in cardiac patients is usually ascribed to an improved capacity of the oxygen delivery system (4), several systems contribute to an improved exercise tolerance, and it is often difficult to separate a primary cardiac improvement from a primary skeletal muscle improvement (10). The greater cardiac capacity following exercise training, therefore, may be accompanied by a greater capacity for muscle energy production as well as greater muscle power.

The relationship between greater aerobic exercise tolerance, associated with an increased maximal aerobic capacity, and increased muscle power, associated with maximal "anaerobic" or glycolytic capacity, is not clearly defined in patients with coronary artery disease who undergo endurance training. We have demonstrated clearly that both increasing age and low activity levels were associated with reductions in peak power and total work during 30 s of maximal isokinetic cycling exercise in healthy subjects (15). This may reflect an associated loss of Type II muscle fibers (1,14), perhaps secondary to motor neurone dysfunction (1,3), with a relative preservation of Type I, fatigue-resistant motor units. The functional consequences of these changes appear to be amenable to improvement with exercise training (2), and, if similar performance decrements were to occur in middle-aged patients who become relatively sedentary secondary to coronary artery disease, an improvement in muscle power capacity may be expected as one of the benefits of an exercise program. A recent study of supplementary circuit weight training in cardiac patients has reported a 24% increase in muscle strength associated with a 12% increase in treadmill time to exhaustion using a modified Bruce protocol (13).

Our recent experience with the use of short-term maximal isokinetic cycle ergometry (12,15–18) suggested that the method might be useful in assessing outcome in cardiac rehabilitation. We wished to identify the relationships between changes in short-term exercise capacity, specifically peak power, total work, and fatigue index, and changes in peak oxygen intake at maximum power output in a progressive incremental exercise test, in cardiac patients randomized either to a 12 wk exercise rehabilitation program or to usual care.

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METHODS

Subjects. Male patients with documented coronary artery disease, under the age of 65 yr, and referred to our exercise rehabilitation program, were considered for entry into the study. Only patients who completed a maximal progressive incremental exercise cycle ergometer test, without angina, marked ST segment depression (more than 0.2 mV), significant cardiac dysrhythmias, or evidence of myocardial dysfunction, were eligible to participate in the study. The study was approved by the institution's ethics committee; a full description of the experimental procedures was given, and informed consent was obtained.

Two procedures were used to assess exercise tolerance: 1) a progressive incremental exercise test on a cycle ergometer to determine maximum power output and peak oxygen intake and 2) a 30 s test of short-term exercise capacity on an isokinetic cycle ergometer. The maximal progressive exercise test and the 30 s isokinetic test were both carried out on two separate occasions 1 wk apart, prior to entry into the study, 1) for screening purposes on the first occasion and 2) to ensure reproducibility of these techniques (18). Following the initial assessments, the patients were randomized either to the rehabilitation program ($N = 12$) or to the control group ($N = 10$) and then reassessed 12 wk later.

The progressive incremental exercise test was carried out on an electrically braked cycle ergometer (Siemens Elema 370); the procedures and associated quality control measures have been described elsewhere in detail (9–11) and in the accompanying paper (18). Maximal exercise tolerance was defined as 1) the highest power output (W_{max}, $\text{kpm} \cdot \text{min}^{-1}$) and 2) the highest oxygen intake defined as peak $\dot{V}O_2$ ($1 \cdot \text{min}^{-1}$). The data obtained were compared with normal standards (11).

The short-term exercise capacity test was carried out on an isokinetic cycle ergometer and is described in detail elsewhere (16,17) and in the accompanying paper (18). Briefly, subjects pedaled for 30 s with maximum effort at a constant pedal velocity of 60 rpm; torque was measured by foil strain gauges bonded to the shafts of the pedal cranks; a laboratory computer derived measurements of torque, power, and work for each pedal revolution; from these measurements, peak power, the total work in 30 s, and the fatigue index (percentage decline in power during the test) were calculated. The data obtained were compared with normal standards (15).

The exercise rehabilitation program required the subjects to attend two 75- to 90-min sessions of training each week, with a request to exercise at home on three other occasions. A conventional approach to exercise prescription for the experimental group was taken; prescriptions were individually executed and calculated to produce a heart rate between 65 and 80% of the maximal heart rate attained during the progressive incremental exercise test. The aerobic stimulus phase of the exercise session lasted approximately 45–60 min; the warm-up and cool-down phases lasted for a total of about 30 min. During the aerobic stimulus phase, patients performed cycle ergometry, treadmill exercise, and track walking for approximately 15–20 min each. Subjects in the usual care or control group increased their daily activity under the advice of their personal physicians.

Standards and statistics. Predicted values for maximal power output (W_{max}) and peak $\dot{V}O_2$ ($1 \cdot \text{min}^{-1}$) in the progressive incremental test were calculated using the following equations (11):

$$W_{max} = (25.3 \times \text{height}) - (9.06 \times \text{age}) - 2{,}759\ \text{kpm} \cdot \text{min}^{-1}\ (\text{SEE } 245), \quad [1]$$

$$\text{peak } \dot{V}O_2 = (0.034 \times \text{height}) - (0.028 \times \text{age}) + (0.022 \times \text{weight}) - 3.76\ 1 \cdot \text{min}^{-1}\ (\text{SEE } 0.483). \quad [2]$$

Predicted values for peak power (PP) and total work (TW) in 30 s were calculated using the following equations (15):

$$PP = (8.2 \times \text{height}) - (5.2 \times \text{age}) - 310\ \text{W}\ (\text{SEE } 119), \quad [3]$$

$$TW = (0.125 \times \text{height}) - (0.097 \times \text{age}) - 3.14\ \text{kJ}\ (\text{SEE } 1.90), \quad [4]$$

where height is expressed in centimeters, weight in kilograms, and age in years. The normal value for fatigue index (decline in power over 30 s) was taken as 26 ± 10% (15).

Repeated-measures two-way ANOVA were used to compare the responses of the experimental and control groups for all data. These analyses allowed us to compare initial data, changes occurring over time, and the differences between exercise and control groups, with results reported as means ± SD. Statistical significance was accepted at $P < 0.05$.

RESULTS

The exercise and control groups were comparable with respect to mean (± SD) age (51.5 ± 2.3 vs 54.4 ± 2.4 yr), weight (82.4 ± 12.3 vs 84.5 ± 13.4 kg), and height (177.3 ± 2.3 vs 175.7 ± 2.1 cm). From a clinical standpoint, five patients in the control group had sustained a myocardial infarction, two had undergone bypass surgery after infarction, and three had bypass surgery with no infarction, while comparable numbers in the exercise group were eight, three, and one, respectively; five control patients and six rehabilitation patients were taking beta blockade medication. Initial maximal power output and peak $\dot{V}O_2$ (Table 1) in the progressive incremental exercise test, and the variables measured in the 30 s isokinetic exercise test (Table 2), were comparable between the control and exercise groups. All subjects randomized to each group com-

TABLE 1. Variables at maximum power output during incremental exercise testing before and after training.

		Control (N = 10)	Experimental (N = 12)
Power (kpm·min^{-1})	Pre	910 ± 152	925 ± 187
	Post	890 ± 142	1117 ± 197*
Power (% predicted)	Pre	78 ± 19	73 ± 10
	Post	76 ± 16	89 ± 14**
O_2 intake (l·min^{-1})	Pre	1.79 ± 0.35	1.85 ± 0.42
	Post	1.79 ± 0.35	2.19 ± 0.38*
O_2 intake (% predicted)	Pre	71 ± 19	71 ± 10
	Post	71 ± 13	85 ± 14**
Heart rate (bpm)	Pre	127 ± 22	135 ± 24
	Post	126 ± 19	140 ± 24
Systolic BP (mm Hg)	Pre	192 ± 25	184 ± 31
	Post	197 ± 32	193 ± 21

Values are mean ± 1 SD.
Pre- vs post-training. * $P < 0.005$; ** $P < 0.001$.

TABLE 2. Short-term (30 s) isokinetic cycle ergometry before and after training.

		Control (N = 10)	Experimental (N = 12)
Peak power (W)	Pre	857 ± 142	774 ± 111
	Post	805 ± 146	883 ± 142**
Peak power (% predicted)	Pre	101 ± 19	88 ± 10
	Post	95 ± 16	100 ± 17*
Total work (kJ)	Pre	13.5 ± 2.2	12.5 ± 2.1
	Post	12.6 ± 3.2	13.9 ± 3.1
Total work (% predicted)	Pre	100 ± 16	90 ± 14
	Post	93 ± 19	99 ± 17
Fatigue index (% decline)	Pre	38 ± 11	36 ± 11.4
	Post	43 ± 11	38 ± 7.6
Heart rate (bpm)	Pre	124 ± 19	136 ± 21
	Post	123 ± 16	136 ± 17

Values are mean ± 1 SD.
Pre- vs post-training. * $P < 0.01$; ** $P < 0.001$.

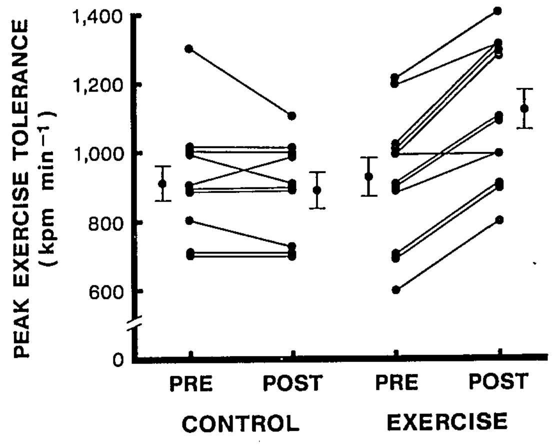

Figure 1—Changes in maximal exercise tolerance (kpm·min^{-1}) during progressive incremental exercise testing in patients assigned to control and exercise groups.

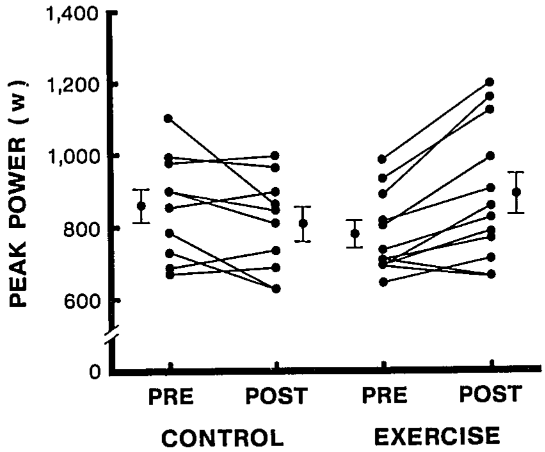

Figure 2—Changes in peak power (W) during maximal isokinetic cycle ergometry in patients assigned to control and exercise groups.

pleted the study, with none of the control or usual care group exercising regularly or at the intensity prescribed for the exercise group.

Following completion of the exercise training program, there was a 21% increase ($P < 0.005$) in maximal power output (kpm·min^{-1}) achieved in the progressive incremental exercise study in the exercise group, with an associated 18% increase ($P < 0.005$) in peak $\dot{V}O_2$ (Table 1). Expressed as a percentage of predicted values for healthy males (equation 1), mean maximal power output increased from 73 ± 10% (mean ± SD) before training to 89 ± 14% ($P < 0.001$) at the end of the training period in the exercise group; comparable increases in peak $\dot{V}O_2$ (equation 2) were from 71 ± 10% to 85 ± 14% ($P < 0.001$) (Table 1). In contrast to the exercise group, the control group showed no improvement in either the maximal power output or peak $\dot{V}O_2$ attained during progressive incremental exercise (Table 1). Individual data for maximal power output during progressive incremental exercise are presented in Figure 1.

During 30 s of maximum isokinetic cycle ergometry, peak power (PP) increased by 14% in the exercise group ($P < 0.05$), with a 6% decrease in the control group (Table 2) and individual data plotted in Figure 2; expressed in terms of predicted normal values (equation 3), PP increased from 88 ± 10% predicted to 100 ± 17% ($P < 0.01$) after training in the exercise group, compared to a decrease from 101 ± 6% to 95 ± 16% (NS) in the control group. While not statistically significant, the total work in 30 s increased in the exercise group and decreased in the control group, with the fatigue index increasing slightly in each group (Table 2). Individual data for percent change in predicted PP (W) and W_{max} (kpm·min^{-1}) are plotted in Figure 3.

DISCUSSION

In the present study we used an isokinetic cycle ergometer to examine the changes in a number of variables related to short-term exercise capacity—peak power, the total work carried out in 30 s, and the fall in power generated during the test or fatigue index—over the course of a 12-wk exercise training program in

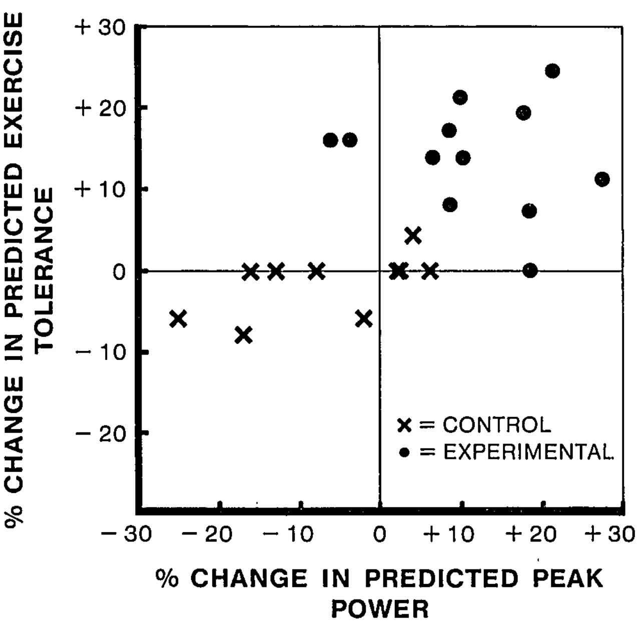

Figure 3—Changes in peak exercise tolerance (kpm·min^{-1}) and peak power (W), each expressed as percent change in predicted normal values (15) in control patients and exercise patients.

selected patients with coronary artery disease. Our hypothesis was that if, in addition to a reduced aerobic capacity, deconditioning secondary to coronary artery disease and the recovery from an acute event such as myocardial infarction or bypass surgery were associated with a decrease in short-term exercise capacity, then conventionally prescribed exercise training would improve not only aerobic exercise tolerance but also maximal short-term exercise capacity. We demonstrated that a conventional cardiac rehabilitation exercise program lasting 12 wk produced a significant 14% improvement in peak power output during isokinetic cycle ergometry; in conjunction with this, we also observed significant improvements of 21% in maximum power output and 18% in peak $\dot{V}O_2$ determined during progressive incremental exercise testing.

Recent studies have reported the natural history of change in exercise tolerance following myocardial infarction as well as the effects of an exercise training intervention (5,8,21). Hung et al. (8) demonstrated improvement in perfusion defects and left ventricular ejection fraction in both trained and control subjects during the first 3 months following infarction, but improvement in maximum aerobic exercise capacity was confined to the exercise trained group only. Thus, in cardiac patients, these authors (8) were unable to relate an improved aerobic exercise tolerance with training to improvements in "central" cardiac function, and they suggested that improvements in "peripheral" muscle were of greater importance. However, in cardiac patients undergoing prolonged (12 months) exercise training of progressively increased intensity, other investigators (6) have demonstrated an improved "central" cardiac function as reflected by an increased stroke volume and stroke work at a comparable, or higher, level of systemic vascular resistance. It is also well known that increases in $\dot{V}O_2$ are associated with increases in muscle oxidative enzymes; for example, Henriksson (7) trained one leg and studied the changes occurring in both control and trained legs and demonstrated that the respiratory quotient and lactate release were lower in the trained than in the untrained leg, suggesting a greater aerobic metabolism and fatty acid oxidation in the trained leg. Although many occupational and leisure activities require considerable muscle endurance and strength, strength training has not been widely used in cardiac rehabilitation programs. However, Kelemen et al. (13), using a circuit weight training technique in patients with coronary artery disease, have demonstrated a 24% increase in muscle strength in association with a 12% increase in peak treadmill exercise tolerance. Therefore, it is possible that rehabilitation activities which improve muscle power contribute to the improvement in peak $\dot{V}O_2$ and vice versa in cardiac patients.

In the 12 patients randomized to a conventional endurance exercise program, the mean improvement in aerobic exercise tolerance was similar to that reported in other studies (4,5,8,20). However, whereas there have been many studies of maximal oxygen intake with training in cardiac patients, little attention has been paid to short-term exercise capacity. In this study we demonstrated that peak power was increased significantly by 14% in the exercise group with a small decrement in the controls. Evidence from studies on the aging process suggests that with inactivity there is a relative reduction in the size and number of fast-twitch, Type II muscle fibers (1,3,14). It is possible that the reduction in daily activity levels associated with coronary artery disease may lead to a reduction in the functional capacity of fast Type II fibers and that, conversely, this loss may be prevented or even reversed by exercise training; in addition, we have recently reported that exercise training may also increase the excitability of descending motor neurons (19). The observation that short-term exercise performance in the 30 s test deteriorated in six of ten control patients, whereas it improved in ten of 12 exercise patients (Fig. 2), provided indirect substantiation for prevention of the loss of fast Type II fibers with exercise training. Further, in the exercise group, improvement in short-term exercise capacity was frequently accompanied in the same individual by an improvement in progressive incremental exercise tolerance. Both short-term peak power and maximal power output during progressive incremental exercise increased in nine of 12 of the exercise group patients, while only one of ten control group patients demonstrated an increase in both variables (Fig. 3). We propose that the improvement in aerobic exercise tolerance, observed in 92% of the exercise group (Fig. 1), and the improvement in short-term exercise capacity, observed in 83% of the exercise group (Fig. 2), were both related to aerobic exercise training, while the lack of improvement and actual decrement seen in both measures in the control group were due to their continued relative inactivity.

There is little doubt that the limitation to exercise performance following myocardial infarction is multifactorial and that exercise training may help to improve more than one of these factors at the same time (10). As it is well documented that patients with the lower initial exercise tolerance improve most with exercise training, it also seems likely that patients with reduced muscle power will benefit more from an aerobic exercise program when compared to patients with good residual muscle power. However, although the present study has shown that short-term exercise capacity increased in response to a conventional cardiac rehabilitation training regimen, it is difficult to quantify the part that this improvement played in the accompanying gains in aerobic exercise capacity. The relationship observed between the improved peak power in a 30 s isokinetic cycle test and the maximal power output and peak $\dot{V}O_2$ during progressive incremental exercise testing warrants further investigation in order to define more precisely the relationships between aerobic capacity and "anaerobic" or glycolytic capacity in patients with coronary artery disease. It may be that an increase in peak power, or the "anaerobic" capacity of the peripheral skeletal muscles, is at least as important in carrying out many activities of daily living such as climbing stairs and moving objects as is an improvement in aerobic capacity. Therefore, the results of this study may have implications for conventional approaches to exercise prescription for many low-risk patients with coronary artery disease. Further investigation of the relationships between improvements in aerobic exercise tolerance and short-term exercise capacity after conventional endurance exercise programs, high intensity cycle ergometer exercise, and other activities such as circuit weight training is warranted.

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Present address for Neil B. Oldridge, Ph.D. and address for correspondence: Department of Health Sciences, School of Allied Health Professions, University of Wisconsin, P.O. Box 413, Milwaukee, WI 53201.

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