Effects of Exercise Training on Ventricular Function
in Patients with Recent Myocardial Infarction

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SUMMARY We evaluated the effects of 6 months of exercise training (bicycle ergometry, walking and jogging) on exercise performance and ventricular function in patients with recent myocardial infarction. Fifteen patients were selected on the basis of myocardial infarction at least 6 weeks but not more than 6 months before the study and age younger than 65 years. The patients were evaluated by maximal treadmill exercise testing and radionuclide angiography at rest and exercise before and after training.

Before exercise training, maximal treadmill exercise time ranged from 1.5 to 11 minutes, ejection fraction at rest from 18% to 67% and end-diastolic volume from 108 to 208 ml. The mean EF was 48 ± 5% (± SD) at rest and did not change at maximal exercise (48.5 ± 5%). All 11 patients who completed the exercise training program achieved a significant training effect, as defined by a reduction in heart rate at 50% maximal pretraining effort or an increase in maximal treadmill time. The mean ejection fraction and end-diastolic volume and wall motion abnormalities at rest and at comparable pretraining exercise work loads and heart rates were not significantly different after training.

Despite a wide range of rest and exercise ventricular function, patients with recent uncomplicated myocardial infarcts significantly increased their exercise performance. Because rest and exercise ventricular function were comparable before and after training, improvement in exercise performance probably resulted from training effects on the peripheral vasculature.

PATIENTS with coronary artery disease demonstrate a wide range of impaired cardiovascular adjustments during exercise.1 Although exercise performance in certain patients with coronary artery disease may not be different from that in age-matched normal subjects, most patients have lower maximal cardiac output and maximal oxygen consumption,1-4 a decreased heart rate response to exercise and a decreased stroke volume at submaximal work loads.1-5 During mild supine exercise, left ventricular filling pressures are often elevated6-10 and systolic ejection rate and the rate of rise of pressure at a given ventricular pressure are reduced.11-13 Noninvasive evaluation of ventricular function using radionuclide angiography has shown that the left ventricular ejection fraction frequently decreases and regional wall motion abnormalities appear during exercise in patients with coronary disease.14-17 These findings indicate that left ventricular dysfunction commonly occurs in patients with coronary artery disease during exercise stress.

Exercise training improves exercise tolerance in most patients with coronary disease.2, 5, 18-22 Clausen1 summarized the central and peripheral effects of exercise training and pointed out that the effects are qualitatively similar in subjects with and without coronary artery disease. In patients with coronary disease and no chest pain, training may improve maximal exercise capacity by increasing maximal oxygen consumption.1, 2, 4, 19, 20 In patients with exercise-induced angina, training may increase exercise tolerance by improving the balance between myocardial and total body oxygen consumption. At a given work load, exercise training reduces the product of heart rate and arterial pressure, which are major determinants of myocardial oxygen consumption.20, 22-24 In certain patients, exercise training increases the rate-pressure product at which pain develops.2, 20-22 Whether exercise training in patients with coronary artery disease can clinically affect ventricular performance by improving myocardial blood flow and oxygen delivery or decreasing variables other than heart rate or arterial pressure that influence myocardial oxygen consumption has not been defined.

We evaluated the effects of 6 months of exercise training on ventricular function at rest and during exercise in patients with recent myocardial infarction. We also determined the extent to which baseline ventricular function during rest and exercise predict the safety and efficacy of an exercise training program in increasing exercise work capacity.

Methods

Patient Selection

Fifteen patients, 13 males and two females, were selected because they had a myocardial infarction 6 weeks to 6 months before the study. Candidates were identified by reviewing the records of admission to the Coronary Care Units of Duke and the Veterans Administration Medical Centers and the County General Hospital, Durham, North Carolina. Myocardial infarction was documented by positive blood analyses for CK-MB isoenzymes or evolutionary QRS changes of acute myocardial infarction. Patients were excluded on the basis of age greater than 65 years, New York Heart Association (NYHA) class IV angina, or uncontrolled congestive heart failure. Before entry into the study

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each patient underwent a complete physical examination and a treadmill exercise test.

Eight patients were receiving propranolol for angina at the time of entry into the study and three patients not taking propranolol were receiving long-acting nitrates. Four patients were in NYHA class I, five in class II, and six in class III. No patient had previously participated in an exercise program.

Treadmill Testing

Graded treadmill exercise tests were performed using a modification of the Balke protocol.25 The patients exercised at a constant treadmill speed of 3.3 mph with progressive increments of treadmill grade (2% increase after the first minute and 1% per minute subsequently) on a treadmill (Quinton Instruments, model 18-54). The heart rate was measured with a Hewlett-Packard 4685-A digital cardiotachometer, and the ECG was measured continuously. The end points of exercise were exhaustion, moderate chest pain, or ST depression consistent with myocardial ischemia.

Each treadmill exercise test was performed between 6:30 and 8:00 a.m. after a 12-hour fast. A practice exercise test was not given. Beta-blocking agents were tapered beginning 3–5 days before testing and were withdrawn entirely for 48 hours. Oral, sublingual or topical nitrates were discontinued 12 hours before testing. No complications occurred as a result of stopping medications before the exercise test. Maximal oxygen uptakes were estimated from the duration of treadmill exercise using published conversion factors.26

Radionuclide Studies

Radionuclide angiograms (RNAs) were recorded at rest and exercise, with the patient in the upright sitting position on a bicycle ergometer, using a multiscrystal gamma camera with a 1-inch parallel-hole collimator (Baird Atomic Systems 77) positioned in an anterior projection. Exercise was begun immediately after the rest RNA. Each RNA was obtained by injecting 10-mCi bolus of technetium-99m pertechnetate through a 20-gauge Teflon cannula in the external jugular vein. The data processing procedures have been described.16, 17, 27 Left ventricular ejection fraction (EF) was calculated from the difference of end-diastolic and end-systolic counts corrected for background activity. End-diastolic and end-systolic left ventricular images were outlined using a computer program that identified the perimeter of the images as a 21% isocount contour of the end-diastolic counts. The aortic valve plane was identified from dynamic images and by isolation of the plane representing alternating increasing and decreasing counts during diastole and systole. The area of the end-diastolic image was planimetered and the length of the major axis was measured by ultrasonic digitizer (Graph pen) coupled to a PDP-11/45 computer. Left ventricular end-diastolic volume (EDV) was calculated by the area-length method of Sandler and Dodge28 measurements of EDV and EF permitted calculation of stroke volume and cardiac output (CO). Previous stud-

ies from our laboratory29 showed that the interobserver variability of EF and EDV in normal subjects was 2.0 ± 1.0% (± SD) and 7.5 ± 4.7 ml at rest and 2.1 ± 1.0% and 7.9 ± 5.7 ml during exercise, respectively. The variability for EF and EDV performed 3 days apart was 4.0 ± 3.8% and 9.9 ± 5.1 ml at rest and 3.2 ± 2.5% and 9.8 ± 6.2 ml during exercise, respectively. Regression analysis between radionuclide and contrast angiography showed correlation coefficients of 0.89 (see 6.1%) and 0.89 (see 23 ml) for EF and EDV, respectively.30

Regional motion of anterior, apical and inferior walls was analyzed from cineangiographic displays of average cardiac cycles and from static displays of superimposed end-diastolic and end-systolic perimeters.

Bicycle Ergometer Exercise Protocol

The same procedure for drug withdrawal before treadmill testing was used before bicycle testing. The patients exercised on an isokinetic bicycle ergometer (Fitron, Lumex, Inc.). Exercise was begun at a work load of 200–300 kilopond-meters (kpm)/min and 60 rpm. The work load was increased by 200 kpm every 2 minutes until the heart rate reached 85% of the maximal predicted rate for age, or moderate chest pain, ST-segment depression consistent with myocardial ischemia, or excessive fatigue occurred. The RNA was taken after at least 1 minute of exercise at a given work load. After 6 months of exercise training, RNAs were recorded at rest and at two levels of exercise. The pretraining bicycle exercise protocol was followed. The first exercise RNA was obtained at the maximal work load and duration of exercise recorded before training. The work load was then increased or exercise prolonged at the maximal work load until heart rate reached the maximal rate of the pretraining study and a final RNA was obtained.

Conditioning Program

The interval from infarction to initiation of the conditioning program ranged from 10 weeks to 6 months (average 3.9 months). Patients exercised three times weekly. The exercise sessions began at 6:30 a.m. and consisted of 10 minutes of bicycle ergometry, 10 minutes of warm-up calisthenics, 30–40 minutes of continuous walking, jogging or climbing stairs, and a 10-minute cool-down period of calisthenics. Intensity of exercise was prescribed on an individual basis so that heart rate during exercise was 75–85% of the maximal heart rate reached during the baseline treadmill test. Patients were checked at frequent intervals during each exercise session to assure achievement of the prescribed heart rate. Exercise prescriptions were adjusted every 2 weeks to ensure a gradual increase in exercise performance. The study period was for 6 months to allow a gradual and relatively long period of conditioning.

Patients were encouraged to reduce their intake of calories, salt, cholesterol and saturated fats. Cigarette smoking was discouraged. Beta-blocking agents, ni-
trates and antihypertensive medications were used during the conditioning period as clinically indicated.

**Statistical Analysis**

Paired measurements before and after training were compared by the Wilcoxon’s signed rank test. Group values are mean ± SD.

**Results**

The patients were 44–65 years old (mean 55.6 years). Eleven of the 15 patients completed 6 months of training. One of the four patients who did not complete the study had a myocardial infarction approximately 2½ months after entry into the study. The infarction was not preceded by a change in anginal pattern and was not related to physical activity. Another patient experienced increased angina and could not participate effectively in the program. Two patients did not complete the study because of personal reasons.

Five of the 11 patients who completed the study were taking propranolol, which was discontinued during the study in two patients. Three patients were receiving long-acting nitrates at entry; nitrates were discontinued in one of these subjects during the study. Two of the 11 patients who completed the study were in NYHA functional class I, five in class II, and four in class III at entry into the study. Three of the four class III patients improved one functional class, as did two of the five class II patients. The remaining subjects did not change functional class. Only patients 2 and 10 had had a previous myocardial infarction (5 and 10 years before the study, respectively).

**Effects of Training on Treadmill Performance**

Training resulted in a significant reduction in heart rate at 50% of the maximal treadmill effort achieved before training (123 ± 5 before and 106 ± 4 beats/min after training; p < 0.001) (table 1, fig. 1). Training resulted in a significant increase in exercise performance. The mean maximal treadmill exercise time increased from 4.8 to 11.0 minutes. Only patient 5 failed to increase maximal exercise time after training, but did have a lower heart rate at submaximal treadmill workload after conditioning. The latter patient had the lowest EF and largest EDV of the group (table 2). The mean heart rates at maximal exercise before and after training were not significantly different. The reduction in heart rate at 50% of the maximal exercise workload before training and increase in maximal exercise time indicate that all but one patient achieved a conditioning effect.

**Body Weight and Serum Cholesterol and Triglycerides After Training**

Training was associated with significant reductions in mean body weight (from 189 ± 3 to 179 ± 4 pounds, p < 0.001) and serum cholesterol (from 253 ± 10 to 192 ± 7 mg/dl, p < 0.001) (table 1). Several patients had a marked decrease in fasting triglyceride levels after training, but the group response was not statistically significant. The higher triglyceride value in patient 5 after training suggests that he was not fasting.

**Hemodynamic Measurements at Rest and Exercise Before Training**

There was a wide range of ventricular function at rest (table 2, fig. 2). EF ranged from 18% to 67% and EDV from 108 to 208 ml. Six patients had an EF below 55%, and six had regional wall motion abnormalities.

All patients achieved 85% of maximal heart rate for age or had moderate chest pain or electrocardiographic evidence of ischemia. Seven patients experienced chest pain consistent with angina pectoris and two patients had ST-segment changes consistent with myocardial ischemia during bicycle exercise (table 3). Using a 5% or greater increase in EF and increased wall motion as criteria for a normal response to exercise,14–17 only patients 1 and 9 had normal hemodynamic measurements during exercise before training (table 3, fig. 2). The EF increased 1–5% in four patients and decreased in five patients during exercise. Six patients had normal regional wall motion abnormalities during exercise. The mean EF was unchanged during exercise (mean 48 ± 5% at rest and 49 ± 5% during exercise). End-diastolic volume increased from 165 ± 13 ml at rest to 209 ± 13 ml during exercise (p < 0.01). CO increased from 5.6 l/min at rest to 12.9 l/min during exercise.

**Effects of Training on Daily Exercise Performance**

Nine of 11 patients increased their physical performance during the training program. The maximal level of exercise per 440 yards of jogging or walking achieved by each patient is listed in table 1. Two patients were jogging continuously, six were jogging 50–220 yards, and three were walking at 3–4 mph. Of the two patients who were jogging continuously, patient 1 had normal ventricular function at rest and exercise...
before training and patient 3 had normal ventricular function at rest, but a decrease in function during exercise. Both patients had normal ventricular function at rest and exercise at the end of the program. The three patients who had the poorest ventricular function at rest and exercise (patients 4, 5 and 10), were walking continuously at the end of the training program.

Effects of Training on Hemodynamic Measurements at Rest

Heart rate and systolic and diastolic arterial pressures, mean EF, EDV, CO and wall motion were also comparable before and after training (table 2). The maximal difference in EF at rest before and after training was 5%. The EF differed by 3% or less in eight patients.

Effects of Training on Hemodynamic Measurements at Comparable Exercise Work Loads and Durations

At comparable exercise work loads and durations, heart rate was significantly less after training, indicating a conditioning effect (mean 142 ± 3 beats/min before and 132 ± 4 beats/min after training, \( p = 0.01 \)) (table 4). Mean CO at comparable work loads was also slightly reduced (12.9 ± 1.2 l/min before and 10.9 ± 1.2 l/min after training, \( p < 0.001 \)). Arterial pressure, EF and EDV were comparable before and after training (table 4). The EF change from rest to comparable exercise work loads before and after training differed by more than 5% in three patients. The EF decreased 10% before training, but increased 10% after training in patient 3. The EF decreased 2% before training and 13% after training in patient 7. The EF decreased 2% before training and 9% after training in patient 8. Of the six patients who had new wall motion abnormalities during exercise before training, five had new wall motion abnormalities after training. Patient 8 had a new wall motion abnormality concomitant with the decrease in EF, after training.

Effects of Training on Hemodynamic Measurements at Comparable Heart Rates

Duration of exercise to achieve maximal heart rate before training was increased in all but patient 5 (table 3); the mean maximal exercise time was 6.44 minutes before training and 8.11 minutes after training (\( p < 0.001 \)). Of the seven patients who experienced chest pain before training, four had chest pain after training. The two patients who developed ST-segment depression before training also developed ST-segment depression after training.
The mean peak systolic pressure during exercise was higher after training (176 mm Hg vs 157 mm Hg, \( p = 0.01 \)). The mean CO was less after training (11.6 l/min vs 12.9 l/min, \( p = 0.001 \)). The other hemodynamic variables were not significantly different after training (table 3).

The change in EF from rest to exercise before and after training differed by more than 5% in two patients. The EF decreased 10% before training in patient 3, but increased 7% after training. The EF decreased 2% before training and 8% after training in patient 7. Of the six patients who had new wall motion abnormalities before training, four had new wall motion abnormalities after training.

**Discussion**

All patients who completed the 6-month exercise training program experienced a significant training effect (see Results). Our data are in agreement with other studies that showed that exercise training significantly improves exercise performance in most patients with coronary artery disease.1, 2, 4, 5, 18-22 In addition, training was associated with significant reduction in total body weight and serum cholesterol levels. A major objective of the present study was to determine whether exercise training affected left ventricular function at rest and during exercise. Ventricular function was assessed using the noninvasive radionuclide angiographic technique. The most common responses to supine and upright bicycle exercise in normal volunteers and in patients with normal coronary arteries are increased EF and wall motion.16-17, 29 Recent studies from our laboratory indicate that failure of the global EF to increase or a decrease in EF during exercise are not specific diagnostic indexes of coronary artery disease. Despite anatomically normal coronary arteries, the global EF may decrease during exercise, especially in women with atypical chest pain.31 We also observed a progressive decline in the exercise change in the EF with increasing age in asymptomatic volunteers with no apparent cardiovascular disease.32 In patients with coronary artery disease, the EF decreases or does not change and regional wall motion abnormalities appear during exercise.14-17, 33 The frequency of these abnormalities during exercise appears to be related to the extent of vessel disease, and to the ability to exercise to clinical end points of predicted maximal heart rate, pain or ST-segment changes.33
Radionuclide angiography demonstrated a wide range of resting ventricular function in patients in the present study. The EF at rest varied from 18% to 67% and EDV from 108 to 208 ml. All patients exercised either to 85% of the predicted rate for age or to chest pain or significant ST-segment depression. Only two patients increased their EF by more than 5% and had normal wall motion during exercise.\textsuperscript{16-17}

In the present study, resting heart rate, arterial pressure, EF, EDV and wall motion did not change after training. At comparable exercise work loads, there was a significant training effect as measured by a reduction in heart rate and a reduction in CO; EF, EDV and arterial pressure were not significantly different. Clausen\textsuperscript{1} and others\textsuperscript{2-5} reported that CO may decrease after training when patients with coronary artery disease exercise at a given submaximal work load. The decrease in CO may result from a decrease in heart rate and effects of training on the peripheral muscles, including an increased aerobic capacity and oxygen extraction.\textsuperscript{1-5}

To achieve the pretraining maximal heart rate after

![Figure 2](image_url)  
**Figure 2.** Ejection fraction at rest and exercise before and after training.
TABLE 4. Effects of Training on Hemodynamic Measurements at Comparable Work Loads and Exercise Duration

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Table 4. Effects of Training on Hemodynamic Measurements at Comparable Work Loads and Exercise Duration

Exercise measurements

- Pt: Patient
- kpm: Kilometers per minute
- Ex time: Exercise time
- HR: Heart rate
- SBP: Systolic blood pressure
- DBP: Diastolic blood pressure
- EF: Ejection fraction
- EDV: End-diastolic volume
- CO: Cardiac output
- Exercise change: Changes in EF and WMA

**Abbreviations:** See Table 3.

Exercise training, the duration of bicycle exercise was increased in all but one patient. At comparable exercise heart rates before and after training, there was no significant difference in EF or EDV. Six patients had a new wall motion abnormality before training, compared with four after training. There was a small but statistically significant decrease in CO after training. At comparable exercise heart rates, seven patients experienced chest pain before and five after training; the two patients who had significant ST-segment decreases before training had similar changes after training. These data indicate that during bicycle exercise at a comparable heart rate before and after training, left ventricular function did not change significantly; consequently, exercise capacity was improved primarily by peripheral effects rather than by an improvement in left ventricular function.

Jensen et al. measured the EF at rest and during submaximal and maximal exercise in 19 patients with coronary artery disease before and after a 6-month exercise training program. At matched submaximal work loads and comparable heart rates and systolic arterial pressures they observed a small but statistically significant increase in EF after training (0.55 ± 0.12 vs 0.59 ± 0.13). At higher maximal work loads after training, arterial pressure was slightly higher, heart rate was unchanged, and the mean EF was not different (0.56 ± 0.13 before and 0.56 ± 0.16 after training). There are several differences between the study by Jensen et al. and the present study. In their study, 13 of the 19 patients had had a myocardial infarction, but in nine patients, the infarction occurred more than 6 months before training; measurements were made during supine exercise. All of our patients had a myocardial infarction less than 6 months before the study and ventricular function was assessed during upright bicycle exercise. Measurements of ventricular function were more variable before and after training in the study by Jensen et al. than in the present study. After training, the EF at rest increased more than 0.05 in five patients (more than 0.10 in two patients) and decreased more than 0.05 in seven patients (greater than 0.10 in four patients). The maximal difference in rest EF before and after training in the present study was 0.05. In the study of Jensen et al., the EF at maximal exercise after training increased more than 0.05 in seven pa-
patients (more than 0.10 in four patients) and decreased more than 0.05 in five patients (more than 0.10 in four patients). In the present study, the EF at maximal exercise after training increased more than 0.05 in one patient and decreased more than 0.05 in one patient.

A second objective of the present study was to determine whether ventricular function measured during rest and bicycle exercise stress would help predict a patient’s ability to safely participate in an exercise rehabilitation program and to achieve a training effect. Despite the wide range in rest and exercise ventricular function, all patients actively participated in the exercise program. Two patients discontinued the program because of a clinical event or uncontrolled symptoms. The patient who experienced a myocardial infarction was in NYHA functional class I and had normal rest and exercise function before beginning the program; his EF was 54% at rest and 73% during exercise. One patient could not participate in the exercise program because of increasing chest pain; his baseline EF was 48% at rest and 41% with exercise. No adverse clinical events related to exercise occurred during the study. The three patients with the lowest EF were walking at a rate of 3–4 mph rather than jogging intermittently or continuously at the end of the exercise program. These patients increased their exercise capacity. Three patients were jogging less than 100 of 440 yards; their EFs were 51%, 67%, and 37% at rest and 42%, 68%, and 38% during exercise. The five patients who were jogging more than 100 of 440 yards had a rest EF greater than 54%, but three had a decrease in EF during exercise before training.

Despite a wide range of rest and exercise ventricular function, the patients in the present study safely and actively participated in the exercise rehabilitation program and significantly increased their exercise capacity and achieved a conditioning effect. Measurements of ventricular function at rest and during exercise at comparable heart rates did not change after 6 months of exercise training. Consequently, the increase in exercise performance in the present study probably resulted from training effects on the periphery rather than effects on left ventricular performance.

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References

The Effects of Airway Pressure on Cardiac Function in Intact Dogs and Man

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SUMMARY Ventilation with positive end-expiratory pressure (PEEP) is associated with reduced cardiac output, but the mechanisms involved are controversial. Possible explanations include increased intrathoracic pressure, reflex changes in myocardial inotropism, pulmonary vascular obstruction and abnormal ventricular interaction. Three types of conscious canine preparations were developed to examine simultaneously each of these factors during ventilation with PEEP. In addition, similar measurements were obtained in patients after cardiac surgical procedures and compared with the results of animal experiments. The primary cause of reduced cardiac output during PEEP appeared to be a diminished end-diastolic volume of the left ventricle, and this appeared to be the result of elevated intrathoracic pressure and increased impedance to blood flow through the lungs. Abnormal interventricular septal shifting and reflex autonomic alterations did not appear to be significant in the normal cardiovascular system. These data provide insight into the cardiac effects of PEEP and emphasize the importance of simultaneous quantification of biventricular performance when assessing cardiopulmonary function.

UNTIL recently, most studies of cardiac function have focused primarily on physiologic events within the left ventricle. Measurements of intracardiac pressures, dimensions and flows have increased the understanding of ventricular dynamics and have improved the care of patients with cardiac disease. However, factors external to the left ventricle significantly influence cardiac performance. For example, during mechanical ventilation, increased intrathoracic pressure may shift a portion of the circulating blood volume away from the chest, decreasing cardiac filling and reducing cardiac output.1,2 Other investigators, however, have observed increased right and left atrial filling pressures during ventilation with positive end-expiratory pressure (PEEP) and have hypothesized that depressed myocardial function may be significant.3 A circulating negative inotropic agent in the blood of dogs ventilated with PEEP has been reported.4 Alterations in ventricular geometry and septal shifting that could contribute to reduced cardiac output have also been noted.5,6 Because the pulmonary vasculature hemodynamically couples the right and left ventricles, alterations in the pulmonary circulation induced by airway pressure could change the functional characteristics of the ventricles in opposite directions and contribute to abnormal ventricular interaction.7,8

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