The Effects of Body Composition Changes to Observed Improvements in Cardiopulmonary Parameters After Exercise Training With Cardiac Rehabilitation*

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Study objective: To discriminate the effects of body fat reduction on improvements in peak aerobic capacity made following exercise training during cardiac rehabilitation.

Design: Observational, prospective study.

Setting: Outpatient cardiovascular health center at regional academic center.

Patient interventions: Peak oxygen uptake (pkVo2), percent body fat, lean body mass (LBM), and other anthropometric measures were assessed before and after a 3-month program of cardiac rehabilitation and exercise training in 500 consecutive cardiac patients following a major coronary event. Baseline pkVo2 was corrected for LBM (pkVo2 lean) and compared with posttraining values.

Results: Following exercise training, percent body fat decreased 5% from 26.2±8.0 to 24.8±7.5 (p<0.0001), and LBM increased 1% from 61.3±12.5 to 61.7±11.8 kg (p=0.02). pkVo2 increased 16% from 16.0±4.1 to 18.5±4.8 mL/kg/min (p<0.0001), and pkVo2 lean increased 13% from 21.7±5.3 to 24.6±6.0 mL/kg/min (p<0.0001). Isolating the effects of reduction in body fat, we discern that these changes contributed to 0.3 of the 2.5 mL/kg/min increase in pkVo2 or 12% of the increase in pkVo2 observed.

Conclusions: Changes in body composition, as a consequence of dietary and exercise modification, contribute to 12% of the “observed” improvement noted in weight-adjusted peak aerobic capacity following cardiac rehabilitation and exercise training. Changes in pkVo2 lean should be used by investigators to assess the singular effects of exercise conditioning alone.

(CHEST 1998; 113:599-601)

Key words: body fat; cardiac rehabilitation; exercise capacity

Abbreviations: LBM=lean body mass; pkVo2=peak oxygen uptake; pkVo2 lean=peak oxygen uptake per kilogram of lean body mass

Cardiac rehabilitation and exercise training are known to result in improvements in multiple risk factors, including reductions in total cholesterol level, body fat, systolic BP, and increases in peak aerobic capacity.1,2 Augmentation of peak aerobic capacity is known to reduce total and cardiovascular mortality and is thought to be a result of exercise conditioning that takes place during the rehabilitation process, the so-called “training effect.”3-6 Peak aerobic capacity, measured as peak oxygen consumption (pkVo2), is traditionally corrected for total body weight and is reported in milliliters per kilogram of body weight per minute.7 Body fat, however, can represent a significant portion of total body weight, and it consumes essentially no oxygen. Moreover, considerable variability in body composition is present across populations8-11 and especially after exercise conditioning programs such as cardiac rehabilitation1,2 thereby potentially contributing to the changes observed in peak aerobic capacity.

We therefore sought to evaluate the independent roles of body fat reduction and exercise conditioning in assessing improvements observed in peak aerobic capacity following cardiac rehabilitation.

Materials and Methods

We prospectively evaluated 500 consecutive patients 4 to 6 weeks following a major cardiac event (coronary bypass surgery,
myocardial infarction, percutaneous coronary angioplasty) who were referred for cardiac rehabilitation at the Cardiovascular Health Center of Ochsner Medical Institutions. Patients participated in outpatient cardiac rehabilitation and exercise training that lasted 12 weeks and included 36 educational and exercise sessions. Each session consisted of approximately 10 min of warm-up exercises, including stretching and calisthenics, followed by 30 to 40 min of continuous upright aerobic and dynamic exercise (various combinations of walking, bicycling, and arm ergometry) and light isometric exercise (hand weights), and approximately 10 min of cool-down stretching and calisthenics. Exercise intensity was prescribed individually so that the patient’s heart rate was at the level of anaerobic threshold measured at entry testing. In addition to the supervised exercise sessions, exercise approximately one to three times per week outside of the formal program was encouraged. Each patient’s exercise prescription was adjusted periodically to encourage a gradual increase in overall exercise performance. Each patient was evaluated by a dietitian who prescribed a phase I diet of the American Heart Association. Furthermore, if the patient was found to be obese, further caloric restriction was recommended. Patients with significant hypercholesterolemia and obesity were periodically followed up by the dietitian during the 3-month program to ensure compliance with and understanding of the prescribed therapy.

Height, weight, body mass index, percent body fat, age, gender, and pkVo2 were assessed at baseline and again 1 week after completing the cardiac rehabilitation and exercise program. Body fat was determined by the skinfold technique by the average of three skinfolds (thigh, chest, and abdomen in men; thigh, triceps, and supra-illium in women). Skinfolds were measured in the fasting state in the morning prior to exercise by a single observer who was blinded to the exercise and previous anthropometric data.

Before entering the program, patients underwent symptom-limited maximal exercise testing assessed via a ramping treadmill protocol. Breath-to-breath on-line gas analysis was performed using a metabolic cart (MedGraphics CPX/D; Medical Graphics Corporation; St. Paul, Minn) with incremental data (tidal volume, respiratory rate, oxygen uptake, carbon dioxide output, minute ventilation) collected every 15 s. pkVo2 was determined by standard methods. To determine the oxygen uptake of primarily oxygen-consuming tissues, we assessed the oxygen consumption corrected for lean body mass (milliliters of oxygen consumed per kilogram of lean body mass per minute), defined as pkVo2 lean. After the outpatient cardiac rehabilitation exercise training program, each patient underwent a protocol similar to the preprogram exercise assessment.

Statistics

All results are expressed as mean±SD. Paired t tests were used to evaluate effects of cardiac rehabilitation. p values ≤0.05 are considered significant.

RESULTS

The mean age of the cohort was 63±11 years, and 80% of the study population was male. The percent body fat was 26.2±5.0 at baseline and decreased to 24.8±7.5 (-5%; p<0.0001) following cardiac rehabilitation (Table 1). Although there was a modest 1% reduction in total weight (83.0±17.5 to 82.0±16.4 kg; p<0.0001) following the rehabilitation program, lean body mass (LBM) increased a modest 1% (61.3±12.5 to 61.7±11.8 kg; p=0.02). Weight-adjusted pkVo2 increased by 16% (16.0±4.1 to 18.5±4.8 mL/kg/min; p<0.0001), and absolute pkVo2 increased by 14% (1,328±438 to 1,517±494 mL/kg/min; p<0.0001).

Table 2 summarizes the hypothetical changes from cardiac rehabilitation and exercise training on the identical cohort of patients if no exercise conditioning was present and only anthropometric changes occurred. Although the amount of LBM may change by anthropometric effects, thus producing a change in the nonweight-adjusted pkVo2, the abstention of the exercise training effect would force the variable pkVo2 lean to be held constant. Therefore, traditional weight-adjusted pkVo2 would be calculated to increase by 3 mL/kg/min (16.0 to 16.3 mL/kg/min) or 2%.

Since the true increase in pkVo2 observed was 2.5 mL/kg/min (16.0 to 18.5 mL/kg/min) (Table 1), we can conclude that 0.3 of the 2.5 mL/kg/min (12%) increase of oxygen consumed was secondary to reduction in body fat and that 2.2 of the 2.5 mL/kg/min (88%) was due to the effect of exercise conditioning.

Table 1—Effects of Cardiac Rehabilitation and Exercise Training on Body Composition and Gas Exchange Variables in 500 Patients With Coronary Heart Disease

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Before</th>
<th>After</th>
<th>% Change</th>
<th>p Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Body fat, %</td>
<td>26.2±5.0</td>
<td>24.8±7.5</td>
<td>-5</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Total weight, kg</td>
<td>83.0±17.5</td>
<td>82.0±16.4</td>
<td>-1</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>LBM, kg</td>
<td>61.3±12.5</td>
<td>61.7±11.8</td>
<td>+1</td>
<td>0.02</td>
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<tr>
<td>Body fat, kg</td>
<td>21.7±4.5</td>
<td>20.3±4.2</td>
<td>-6</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>pkVo2 lean, mL/kg/min</td>
<td>21.7±5.3</td>
<td>24.6±6.0</td>
<td>+13</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>pkVo2, mL/kg/min</td>
<td>16.0±4.1</td>
<td>18.5±4.8</td>
<td>+16</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>pkVo2, mL/min</td>
<td>1,328±438</td>
<td>1,517±494</td>
<td>+14</td>
<td>&lt;0.0001</td>
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</tbody>
</table>

Table 2—Hypothetical Effects* of Changes in Body Composition Following Cardiac Rehabilitation and Exercise Training on Gas Exchange Variables in 500 Patients With Coronary Heart Disease

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Before</th>
<th>After</th>
<th>% Change</th>
<th>p Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Body fat, %</td>
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<tr>
<td>Total weight, kg</td>
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<td>82.0</td>
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<tr>
<td>LBM, kg</td>
<td>61.3</td>
<td>61.7</td>
<td>+1</td>
<td></td>
</tr>
<tr>
<td>Body fat, kg</td>
<td>21.7</td>
<td>20.3</td>
<td>-6</td>
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<tr>
<td>pkVo2 lean, mL/kg/min</td>
<td>21.7</td>
<td>21.7</td>
<td>0</td>
<td></td>
</tr>
<tr>
<td>pkVo2, mL/kg/min</td>
<td>16.0</td>
<td>16.3</td>
<td>+2</td>
<td></td>
</tr>
<tr>
<td>pkVo2, mL/min</td>
<td>1,328</td>
<td>1,336</td>
<td>+1</td>
<td></td>
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</table>

*Assuming no effect from exercise conditioning.
1Actual changes.
Hypothetical and calculated changes.
DISCUSSION

This investigation demonstrates that reduction in body fat contributes to the "observed" improvement in weight-adjusted pkVo₂ following exercise conditioning programs, such as cardiac rehabilitation, thereby overestimating the true improvement present. Furthermore, although a training effect was clearly present, exercise conditioning alone was responsible for 88% of the improvement noted in weight-adjusted peak aerobic capacity.

Dietary modification of both fat, and when appropriate, total calories, is an integral component of risk factor modification currently used in the treatment of patients with coronary heart disease. When applied to individuals without concomitant exercise therapy, there is generally a loss of body weight consisting of reductions in both body fat and LBM. One of the benefits of cardiac rehabilitation is that dietary changes are made in concert with exercise therapy such that most of the resulting weight loss is in the form of body fat.

Peak aerobic capacity is an important determinant of morbidity and mortality in coronary heart disease patients, especially following major cardiac events. Cardiopulmonary exercise stress testing reliably measures several parameters of gas exchange, including peak aerobic capacity (measured as pkVo₂ in milliliters per minute). However, because of substantial variation in body weight across populations and between gender, pkVo₂ is corrected for body weight and is reported in milliliters per kilogram per minute. However, there still remains considerable variability in percent body fat across populations and between gender, and this variability becomes magnified when patients undergo therapies designed to reduce body fat, such as cardiac rehabilitation. Since body fat does not consume any significant oxygen, a reduction in fat weight could lead to an observed increase in the traditional weight-adjusted pkVo₂ without a true increase in peak aerobic capacity having occurred.

One potential limitation to this study is our method for assessment of body fat. Although we did not use the gold standard of hydrostatic weighing for assessing body fat, the three-site skinfold method used has been validated against hydrostatic weighing in various populations as both accurate and reproducible.

Our data demonstrate that changes in body composition as a consequence of dietary and exercise modification contribute to 12% of the observed improvement noted in weight-adjusted peak aerobic capacity following cardiac rehabilitation and exercise training. In the future, changes in pkVo₂ corrected for LBM should be used by investigators to assess the singular effects of exercise conditioning alone.

REFERENCES

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