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Articles

Benefit of Selective Respiratory Muscle Training on Exercise Capacity in Patients With Chronic Congestive Heart Failure

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Abstract

Background Diminished respiratory muscle strength and endurance have been demonstrated in patients with heart failure. This may contribute to exertional dyspnea and reduced exercise capacity in these patients. The purpose of this study was to investigate whether selective respiratory muscle training could alleviate dyspnea and improve exercise performance in patients with chronic congestive heart failure.

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Methods and Results Fourteen patients with chronic heart failure (left ventricular ejection fraction, 22±9%) were enrolled in a supervised respiratory muscle training program. This consisted of three weekly sessions of isocapnic hyperpnea at maximal sustainable ventilatory capacity, resistive breathing, and strength training. Maximum sustainable ventilatory capacity, maximum voluntary ventilation, maximal inspiratory and expiratory pressures, peak $\dot{V}O_2$, and the 6-minute walk test were measured before (pre) and after (post) 3 months of training. Eight patients completed the training program. Respiratory muscle endurance was improved with training, as evidenced by increases in maximal sustainable ventilatory capacity (pre, 48.6±10.7 versus post, 76.9±14.5 L/min; $P<.05$) and in maximal voluntary ventilation (pre, 100±36 versus post, 115±39 L/min; $P<.05$). Respiratory muscle strength was also increased with training as maximal inspiratory (pre, 64±31 versus post, 78±33 cm H_2O; $P<.01$) and expiratory (pre, 94±30 versus post, 133±53 cm H_2O; $P<.001$) pressures rose. Submaximal and maximal exercise capacity were significantly improved with selective respiratory muscle training as the 6-minute walk (pre, 1101±351 versus post, 1421±328 ft; $P<.001$) and peak exercise $\dot{V}O_2$ (pre, 11.4±3.3 versus post, 13.3±2.7 mL $\cdot$ kg^{-1} $\cdot$ min^{-1}; $P<.05$) both significantly increased. Dyspnea during activities of daily living was subjectively improved in the majority of trained patients. Dyspnea quantified by the Borg scale was significantly reduced during progressive isocapnic hyperpnea but not during bicycle exercise. No statistically significant improvement in maximal sustainable ventilatory capacity, maximum voluntary ventilation, maximal inspiratory or expiratory mouth pressures, 6-minute walk, or peak $\dot{V}O_2$ was observed in the 6 patients who did not complete the training program.

Conclusions Selective respiratory muscle training improves respiratory muscle endurance and strength, with an enhancement of submaximal and maximal exercise capacity in patients with heart failure. Dyspnea during activities of daily living was subjectively improved in the majority of trained patients.

Key Words: exercise • heart failure

Introduction

Histochemical, metabolic, and vascular abnormalities have been described in the limb muscles of patients with heart failure.[1 2 3 4 5 6 7 8 9 10 11 12 13] Histochemical abnormalities include fiber atrophy, an increased percentage of glycolytic easily fatiguable type IIb fibers, and reduced oxidative and lipolytic enzymatic activity.[6 7 8 9] Metabolic abnormalities during exercise demonstrated by ^{31}P magnetic resonance spectroscopy include a reduced rate of oxidative metabolism with an earlier shift to glycolytic metabolism.[1 2 3 4 5] Decreased leg muscle perfusion during exercise has been observed in these patients.[11 12 13] Limb skeletal muscle endurance is also diminished.[14] It is likely that these intrinsic skeletal muscle changes are not limited to the limb skeletal musculature but that generalized skeletal muscle changes occur.[10] Abnormalities in respiratory muscle function have been described in these patients, including increased deoxygenation during exercise,[15 16] reduced endurance,[17] reduced strength,[18 19 20] and histochemical abnormalities.[21]

The symptoms of heart failure, ie, exertional fatigue and dyspnea, may result in part from these intrinsic

skeletal muscle abnormalities. Exertional dyspnea may occur when the activity of the respiratory muscles is increased and/or the respiratory muscles are weak.[22] Dyspnea may be most closely related to a corollary discharge that is proportional to the neural drive to the respiratory muscles. Increased work of breathing or respiratory muscle weakness is associated with an increased neural drive; therefore, they are associated with dyspnea.[23]

Aerobic training has been shown to partially reverse the skeletal muscle metabolic abnormalities,[24] [25] increase maximal exercise performance,[26] [27] [28] and reduce the excessive ventilatory response to exercise in patients with heart failure.[27] [28] Selective respiratory muscle training has yet to be investigated in patients with heart failure. In patients with chronic obstructive lung disease, selective respiratory muscle training regimens have been variably reported to be successful in alleviating dyspnea and improving exercise capacity.[29] [30] [31] [32] [33] Selective muscle training may be particularly advantageous for patients with heart failure, since previous animal and human studies suggest that aerobic training may adversely affect ventricular remodeling after large myocardial infarctions.[34] [35]

The purpose of the present study was to investigate the effect of selective respiratory muscle training on exertional dyspnea and exercise capacity in patients with heart failure. We postulated that if respiratory muscles or neural drive to the respiratory muscles were a key modulator of the sensation of dyspnea, then an increase in muscle strength and endurance should attenuate exertional dyspnea. We also postulated that selective respiratory muscle training, by diminishing dyspnea, would enhance submaximal and maximal exercise performance, at least for those patients whose exercise capacity is limited by dyspnea.

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Patient Population

The patient population consisted of 14 patients with congestive heart failure. The cause of heart failure was coronary artery disease in 6 patients and dilated cardiomyopathy in 8 patients. The average age was 55±14 years. Ejection fraction averaged 22±9%. Peak $\dot{V}O_2$ was 13.5±4.8 mL · kg^{-1} · min^{-1}. Two patients were in New York Heart Association functional class I, 2 in class II, 6 in class III, and 4 in class IV. All patients were receiving digoxin, diuretics, and angiotensin-converting enzyme inhibitors. Average height was 69±3 in.; average weight was 191±31 lb. All subjects were currently nonsmokers without a prior history of pulmonary disease. Nine patients had a past history of smoking (25±11 pack-years). Pulmonary function tests were obtained in all subjects.

The protocol was approved by the Human Studies Subcommittee at the Philadelphia Veterans Administration Medical Center and the Committee on Studies Involving Human Beings at the Hospital of the University of Pennsylvania. Written informed consent was obtained from all subjects.

Baseline Studies

Before participation in the training protocol, each subject underwent a battery of tests within a 2-week period (pre). These tests consisted of pulmonary function tests, maximal bicycle exercise with measurement of respiratory gases, 6-minute walk test, and measurement of maximal sustainable ventilatory capacity. All tests were repeated at the completion of training (post). After completion of the study period, the patients were asked whether they felt less short of breath during activities of daily living.

Pulmonary function tests. Spirometry, plethysmographic lung volumes (total lung capacity, tidal volume, vital capacity, and forced vital capacity), and maximum voluntary ventilation were measured in all subjects by standard techniques.[36]

Respiratory muscle strength was assessed by measurement of maximum mouth pressures. Maximum inspiratory pressure was measured at residual volume, whereas maximum expiratory pressure was assessed at total lung capacity. Maximum inspiratory and expiratory pressures were recorded in triplicate or until a stable value was achieved.[37]

Exercise testing with respiratory gases. Maximal upright bicycle exercise with measurement of respiratory gases was performed in the fasting state. Arterial oxygenation was monitored with an Ohmeda ear oximeter. Exercise was begun at 0 W and increased in 25-W increments every 3 minutes until exhaustion. The level of perceived dyspnea and fatigue at each workload was recorded according to the Borg scale. Heart rate was monitored continuously. Arterial blood pressure was measured by cuff sphygmomanometry. Measurements of mixed expired carbon dioxide, mixed expired oxygen, and expired volume were determined at rest and every 30 seconds throughout exercise with a metabolic cart (SensorMedics).

Anaerobic threshold was defined as (1) the point at which the ventilatory equivalent for O_2 ($\dot{V}E/\dot{V}O_2$) was minimal, followed by a progressive increase; (2) the point after which the respiratory exchange ratio consistently exceeded the resting respiratory gas exchange ratio; and (3) the $\dot{V}O_2$ after which a nonlinear increase in minute ventilation occurred relative to $\dot{V}O_2$.[38]

Ventilation was assessed by correlation of minute ventilation ($\dot{V}E$) with minute CO_2 production ($\dot{V}CO_2$). In humans, previous investigators have demonstrated that a close correlation exists between $\dot{V}E$ and $\dot{V}CO_2$ below the anaerobic threshold. A linear increase in $\dot{V}E$ with $\dot{V}CO_2$ occurs, so arterial PCO_2 remains constant. In patients with heart failure, $\dot{V}E$ at any given $\dot{V}CO_2$ is higher than normal. To contrast $\dot{V}E$ between subjects, it was correlated with $\dot{V}CO_2$. From the linear regression analysis, $\dot{V}E$ at $\dot{V}CO_2$ of 1 L/min ($\dot{V}E$-$\dot{V}CO_{2\ 1L}$) was derived in each subject and used as a quantitative index of ventilatory drive.[39]

Six-minute walk test. The unencouraged 6-minute walk test was performed in a 130-ft unobstructed corridor. Rest points were provided at the beginning, middle, and end of the concourse. Patients were told the time remaining at 3 and 5 minutes. Dyspnea at the end of the test was assessed by the Borg scale.[40]

Maximal sustainable ventilatory capacity. In attempts to measure the endurance of the respiratory muscles, previous investigators have measured the level of isocapnic hyperpnea that subjects can maintain for 12 minutes. They have used the term maximum sustainable ventilatory capacity (MSVC) to describe this measurement and have used it to evaluate the effects of interventions thought to affect respiratory muscle endurance. This test is carried out by providing subjects with visual biofeedback of their level of ventilation and asking subjects to maintain a given level of ventilation for 12 minutes. During these periods of volitional hyperpnea, various amounts of carbon dioxide are added to the inspired air to maintain isocapnia (in arterial blood) as dictated by continuous measurements of end-tidal PCO_2. These measurements are made by sequentially incrementing the target flow rate that the subject attempts to maintain until the subject can no longer keep up. The highest target flow rate that the subject can

maintain is called the MSVC.[29] [41]

To avoid the possibility of carrying out subsequent measurements in subjects who may have already developed respiratory muscle fatigue during a previous trial, subjects are generally tested at only one target flow rate per day. Consequently, measurements of MSVC usually require 5 to 8 days of testing. As part of our continuing studies of respiratory muscle endurance in patients with heart failure, we have developed a protocol to determine MSVC in a single testing session. This involved an incremental increase in the target flow rate every 3 minutes. To investigate whether this incremental measurement as described in detail below would equal the traditional approach, six normal subjects had MSVC measured randomly by both approaches. The measurements of MSVC by the incremental test and traditional approach were comparable (incremental MSVC, 80±14; traditional MSVC, 80±12 L/min; *P*=NS). The mean of the difference for the two approaches for each patient was 5±2 L/min, with a coefficient of variation of 17.5% for the incremental MSVC versus 15% for the traditional approach, indicating a similar spread of the data for both measurements.

In the present study, we used this novel technique to measure MSVC before and after the training period. Since our technique involved only 1 day of testing, we were able to carry out two measurements of MSVC in both pretherapy and posttherapy phases. The exact protocol was as follows.

Upon the subject's arrival in the exercise laboratory, maximal voluntary ventilation was measured in duplicate. The seated subject breathed into an MSVC apparatus with monitoring of end-tidal CO_2. Inspiratory and expiratory ventilation were monitored via pneumotachographs. Pulse oximetry, heart rate, blood pressure, and Borg scale ratings of dyspnea[40] were monitored throughout the test.

We have used the MSVC rebreathing circuit previously.[29] A graduated flow meter (Fisher and Porter Corp) was adjusted to a set rate. Airflow entered a 5-L anesthesia bag (model 5-035-106, Puritan-Bennett Corp), then passed into a 6-L mixing chamber, and then went to the patient, who inspired this air through a two-way nonrebreathing Hans Rudolph valve. The subject was instructed to regulate his breathing such that the folds in the 5-L target bag were always prominently displayed. No further instructions were provided. Exhaled air was then recirculated via a variable-flow CO_2 scrubber circuit. The 6-L dead-space mixing chamber served to humidify the inspired air. Inspiratory and expiratory flow rates were monitored with pneumotachographs interfaced in the airflow circuit (model 3800, Hans Rudolph). The pneumotachographs were connected to a variable-reluctance pressure transducer (model MP 45-871, Validyne Engineering). Ventilatory frequency, minute ventilation, inspiratory time per breath, and the inspiratory duty cycle, ie, fraction of breathing cycle spent in inspiration, were derived from these signals.

Isocapnia was maintained throughout the test. End-tidal CO_2 was monitored with a mass spectrometer (Perkin-Elmer) by continuous sampling of air from a mouth port. CO_2 levels were displayed on an oscilloscope, with CO_2 concentration controlled by varying flow through the CO_2 scrubber circuit. This circuit consisted of a canister containing CO_2 absorbent granules (barium hydroxide lime; Warren E. Collins) that was connected to both ends of the dead-space portion of the apparatus. Excess CO_2 was removed from and supplemental CO_2 was added to the inspired air to maintain isocapnia in arterial blood as dictated by continuous measurements of end-tidal CO_2. Oxygen concentration was also adjusted according to pulse oximetry readings. Supplemental oxygen was added if the O_2 saturation fell below

90%.

Each subject began isolated respiratory muscle exercise at a workload of 20% of his or her maximal voluntary ventilation. Workloads were of 3-minute duration and increased by 10% increments to a maximal tolerated level. MSVC was defined as the highest workload completed for the full 3 minutes. It was presumed that all subjects achieved steady state by the third minute of each workload; therefore, ventilatory measurements, ie, minute ventilation, time in inspiration, breathing frequency, and time per breath were derived from an average of 10 breaths during the last minute of each workload.

Specific Respiratory Muscle Training

After completion of the baseline studies, the patients were enrolled in a 3-month supervised selective respiratory muscle training program. Supervised training sessions were held 3 times per week. Each session lasted approximately 90 minutes. Components of the training session were as follows.

1. Isocapnic hyperpnea, which was carried out for 20 minutes of each training session. We used a training circuit similar to that described by Leith and Bradley.[42] Initially, the target flow rate was set at MSVC and progressively decreased until the subject was able to maintain the target flow rate for 20 minutes. Once the target flow rate was established, the subjects trained at this level for 2 weeks, ie, six training sessions. Subsequently, the target flow rate was increased by 5 L/min every 2 weeks.

2. Resistive breathing, which was performed for 20 minutes using the THRESHOLD inspiratory muscle trainer (Health Scan) set at 30% of maximal inspiratory pressure. The device is a clear plastic cylinder (1.5 in. in diameter) that contains an airflow valve at one end, an internal pressure regulator controlled by spring tension, and a mouthpiece at the other end. With the nares occluded, the patient inhaled through the mouthpiece, generating enough negative pressure to force the airflow valve open. As long as the subject's mouth pressure was more negative than the threshold pressure, inspired air would continue to flow through the device to the subject. The patients were instructed to take one breath every 4 seconds and to inspire for 2 seconds. A timer with a flashing light that stayed illuminated for 2 seconds was used to regulate the exercise.

Each patient received a threshold device, a timer, and a diary. The exercise was performed 3 times per week under supervision. The patients were instructed to use the device twice daily for 15 minutes on off days and to record their use in the diary. Every 2 weeks, the resistance on the device was increased by 5 cm H_2O.

3. Strength training, which was performed using a two-way valve connected to two pressure gauges. Maximal inspiratory efforts were performed at residual volume using the two-way valve turned to obstruct airflow except for a small air leak provided by an 18-gauge needle. Ten repetitions of maximal inspiratory pressure maintained for 10 seconds followed by 15-second rest periods were performed. This was followed by 10 repetitions of maximal expiratory pressure at total lung capacity held for 10 seconds and with 15-second rest periods between each contraction. A lighted timer facilitated completion of this exercise.

4. Rehabilitation medicine exercises, performed as a series of breathing calisthenics. Eight repetitions of each exercise were completed. These exercises strengthened the abdominal muscles. In the supine position, each leg was raised alternately as the patient exhaled. In a separate exercise, the head and

shoulders were raised from the bed as the patient exhaled. In a third exercise, a book was placed on the patient's abdomen. With inhalation, the patient pushed out his abdomen as far as possible. With exhalation, the abdomen was then pulled in as close as possible to the spinal column. In the final exercise, the patient was seated. The patient was instructed to inhale deeply, allowing the abdomen to expand against a small book. The book was then pressed in firmly as the patient exhaled forward.

Training Workloads

Over the 3-month training period, the training workloads increased as follows: the target flow rate rose from 52±12 to 77±14 L/min, resistive breathing increased from 19±9 to 39±5 cm H_2O, and strength training increased from 28±10 to 47±17 kPa for inspiratory muscles and from 44±20 to 83±18 kPa for the expiratory muscles (all $P<.01$).

Statistical Analysis

Data from patients in the trained and comparison groups were compared with Student's paired or nonpaired *t* tests or one-way ANOVA as appropriate. The relations between variables were examined by linear regression analysis. Three-way ANOVA with blocking by the patient of three factors (time: before and after training; workload: percent maximum voluntary ventilation or watts; and Borg scale recordings) was also performed to investigate perceived dyspnea before and after training. χ^2 analysis was used to examine nonnumerical differences between groups. A value of $P<.05$ was considered significant. Data are expressed as mean±SD.

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Patient Population

Six patients dropped out of the training program after an average of only three training sessions because of the large time commitment, travel problems, or work conflicts. These patients formed a comparison group. The clinical characteristics of the two groups were similar, although the trained group tended to have a lower peak $\dot{V}O_2$ (Table 1).

View this table: **Table 1.** Clinical Characteristics of the Comparison and Trained Groups
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Patients in the trained group required a significant increase in diuretic therapy approximately 1 month into training (Lasix dose: pre, 150±90 versus post, 235±146 mg; $P<.05$). Despite the increase in diuretic dosage, their weight remained unchanged (pre, 190±37 versus post, 188±36 lb; NS) during the study period. Diuretic therapy remained unchanged in the comparison group (Lasix dose: pre, 47±21 versus post, 63±51 mg; NS). No significant alteration in digoxin or vasodilator therapy occurred in the trained or the comparison groups.

Pulmonary Function Tests

After 3 months, significant increases occurred in maximal inspiratory (pre, 64±31 versus post, 88±34 cm H_2O; $P<.01$) and maximal expiratory (pre, 94±30 versus post, 152±40 cm H_2O; $P<.001$) mouth pressures

and maximal voluntary ventilation (pre, 100±36 versus post, 115±39 L/min; $P<.05$) in the trained group. No significant changes were observed in the comparison group. The significant improvement in these parameters demonstrates the efficacy of selective respiratory muscle training.

Additional results of the baseline conventional pulmonary function tests for the trained (T) and comparison (C) groups, respectively, were as follows: vital capacity (T, 3.4±0.9 versus C, 4.4±1.6 L), forced expired volume in 1 second (T, 2.3±0.7 versus C, 3.1±1.4 L/s), FEV_1/FVC (T, 72±8% versus C, 73±10%), and functional residual capacity (T, 3.0±0.9 versus C, 3.2±0.6 L); none of these differences were significant at the $P=.05$ level. Additionally, for both the trained and comparison groups, no significant differences were noted between baseline measurements and those obtained at the completion of the study period, although total lung capacity tended to increase in the trained group (pre, 5.4±1 versus post, 6.1±1.4 L; NS).

Maximal Sustainable Ventilatory Capacity

Individual data points for all subjects before and after training are shown in Fig 1. All of the trained patients increased MSVC over the course of the study period, with a mean increase of 28±6 L/min ($P<.001$). In contrast, 4 of the 6 comparison subjects exhibited decreases in MSVC over the duration of the study period, with only 2 subjects demonstrating small increases. The mean change in MSVC for the comparison group was a decrease in MSVC of 4±6 L/min (NS).

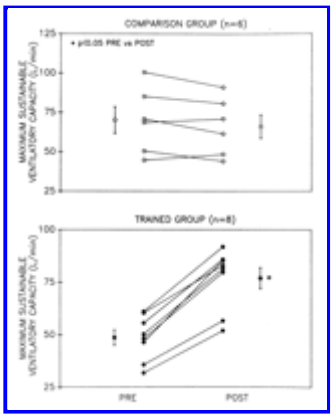

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Figure 1. Graphs showing maximum sustainable ventilatory capacity (mean±SEM) for the individual patients in the comparison and trained groups before and after the study period.

Table 2 summarizes the cardiovascular and ventilatory parameters measured at maximal sustainable ventilatory capacity before and at the end of the study period for the comparison and trained groups. The comparison group had a higher MSVC than the trained group during the prestudy measurements. Increases in both tidal volume and breathing frequency accounted for the statistically significant increase in MSVC exhibited by the trained group over the course of the study. Despite this increase in breathing frequency shown by the trained group, no changes were noted in the inspiratory duty cycle. This indicates that training did not alter the number of seconds per minute spent in inspiration by the trained group. During the study period, no changes in heart rate or mean arterial blood pressure during the MSVC measurements were observed for either the trained or the comparison group.

View this table: **Table 2.** Ventilatory Muscle Endurance Measurements
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Submaximal Exercise Performance

Submaximal exercise performance was estimated from the 6-minute walk test. Each of the subjects in the training group increased the distance covered in their 6-minute walk (pre, 1101±351 versus post, 1420±328 ft; $P<.001$) (Fig 2). The mean increase for the trained group was 320±105 ft, or 33±15% of the distance covered during their prestudy 6-minute walk. In contrast, 1 of the comparison subjects decreased the distance covered during the 6-minute walk, 2 exhibited no change, and 3 showed small increases over the study period, with no overall significant change for the group (pre, 1212±541 versus post, 1243±565 ft; NS).

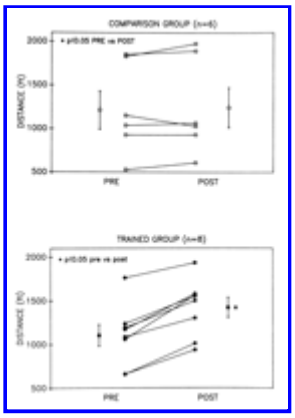

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Figure 2. Graphs showing results (mean±SEM) of the 6-minute walk test before and after training in the comparison and trained groups.

Borg scale rating of perceived dyspnea at the completion of the 6-minute walk test was unchanged in both the comparison (pre, 10.3±2.9 versus post, 10.7±2.3; NS) and the trained (pre, 11±4 versus post, 10±2; NS) groups. Thus, the subjects in the trained group were able to do more work at the same level of perceived dyspnea.

Maximal exercise performance was assessed by measurement of peak $\dot{V}O_2$. No change in peak $\dot{V}O_2$ or exercise duration was noted in the comparison group (pre, 16±6 versus post, 15±6 mL · kg^{-1} · min^{-1}; NS). However, 7 of the 8 patients in the training group increased peak $\dot{V}O_2$ over the course of the study (pre, 11.4±3.3 versus post, 13.3± 2.7 mL · kg^{-1} · min^{-1}; $P<.05$), with a mean increase of 1.8±1.7 mL · kg^{-1} · min^{-1} (Fig 3).

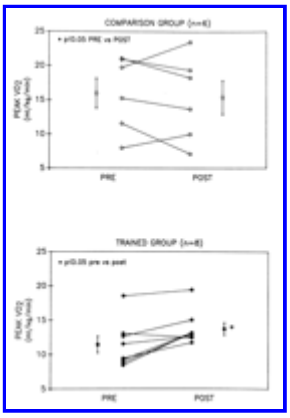

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Figure 3. Graphs showing results (mean±SEM) of peak exercise oxygen consumption (Peak VO_2) in the comparison and trained groups.

Table 3 summarizes rest and peak exercise measurements for the training group before and after the study period. No differences were noted in the rest measurements in the prestudy and poststudy tests. Exercise duration increased over the study period. Maximum respiratory rate, tidal volume, heart rate, and mean arterial blood pressure did not change. Respiratory quotient at end exercise was similar before and after training, suggesting that maximal effort was comparable. The lack of change in resting heart rate and $\dot{V}O_2$ at the anaerobic threshold suggests that the improvement in exercise capacity in the trained group was independent of a systemic training effect.

View this table: **Table 3.** Results of Exercise Testing in the Trained Group
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Peak exercise ventilation increased significantly in the trained group, consistent with their increased workloads. However, minute ventilation normalized for CO_2 production and expressed as minute ventilation at a CO_2 production of 1 L was unchanged (pre, 38.2±5.9 versus post, 38.3±5.3; NS). This suggests that the ventilatory response to exercise was unchanged by selective respiratory muscle training.

Effect of Selective Respiratory Muscle Training on the Sensation of Dyspnea

Subjective improvement in dyspnea was noted in 6 of the 8 trained subjects but only 1 of the comparison group subjects. None of the patients in the trained group had worsening dyspnea. None of the trained patients were hospitalized for heart failure, although two hospitalizations for chest pain occurred, suggesting that the patients were more active (Table 4).

View this table: **Table 4.** Subjective Changes to Training
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Perceived dyspnea during measurement of maximal sustainable ventilatory capacity was significantly improved in the trained ($P<.001$; Fig 4) but not the comparison (P=NS) group. However, perceived dyspnea at each bicycle workload was unchanged before and after training in both groups (P=NS for both). Dyspnea index derived as maximum voluntary ventilation minus peak minute ventilation divided by maximum voluntary ventilation was unchanged at peak exercise before and after training (pre, 53±18 versus post, 50±11%; NS).

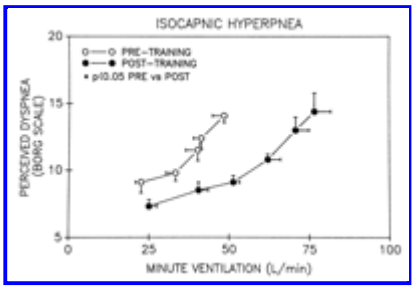

Figure 4. Graph showing perceived dyspnea during isocapnic hyperpnea in the trained group before and after training.

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The major findings of the present study are that selective respiratory muscle training in a cohort of patients with chronic congestive heart failure was associated with (1) increases in ventilatory muscle endurance, (2) increases in strength of both the inspiratory and expiratory muscles, (3) increases in submaximal exercise capacity as assessed by the 6-minute walk test, (4) increases in maximal exercise capacity as assessed by measurement of peak $\dot{V}O_2$ during bicycle ergometry, (5) increases in peak exercise ventilation, and (6) decreases in rating of perceived dyspnea during volitional isocapnic hyperpnea. More importantly, the majority of our cohort who received this type of training reported a subjective decrease in dyspnea during activities of daily living. This represents the first report of improvements in both clinical and physiological measurements in patients with chronic heart failure elicited by selective respiratory muscle training.

Several previous reports in patients with chronic obstructive lung disease have yielded contradictory findings on the ability of selective respiratory muscle training to alleviate dyspnea and improve exercise performance in that patient population.[29] [30] [31] [32] [33] These studies have varied by the type and duration of training. Few have been controlled studies. In our study, we investigated the impact of both strength and endurance training of the respiratory muscles in patients with heart failure. Repeated maximal sustained contractions during inspiration and expiration combined with resistive breathing provided strength training. Isocapnic hyperpnea and resistive breathing were used for endurance training. Both inspiratory and expiratory muscles were trained. The classic breathing calisthenics performed in this study target primarily the expiratory muscles. Thus, the program we designed was unique in that it was more comprehensive and intense than regimens used in other respiratory muscle training studies. Our study also

included a comparison group. The patients who composed the comparison group were not ideal control subjects, since the motivation of this group was clearly reduced compared with the subjects who completed the rigorous training program. Nevertheless, this comparison group provided a cohort of stable heart failure patients in which to assess the reproducibility of respiratory and exercise measurements.

Efficacy of Training

The efficacy of our respiratory muscle strength and endurance training program was demonstrated by significant increases in maximal inspiratory and expiratory pressures, maximal voluntary ventilation, and maximal sustainable ventilatory capacity measured during progressive isocapnic hyperpnea in the trained but not the comparison group. Moreover, the improvement in maximal expiratory pressure demonstrates that expiratory as well as inspiratory muscles were trained. Since the percentage increase in maximal expiratory pressure was greater than inspiratory muscle pressure, the training effect may actually have impacted more on the expiratory muscles. The percent change in maximal mouth pressures, maximum voluntary ventilation, and maximal sustainable ventilatory capacity were similar to prior reports in patients with chronic obstructive pulmonary disease (COPD).[33] As in other selective respiratory muscle training programs, the impact on standard pulmonary function tests was minimal, with only a trend toward increased total lung capacity in the trained group probably related to the increase in respiratory muscle strength.

Recent studies have demonstrated a variety of histochemical abnormalities in the diaphragm of patients who have undergone cardiac transplantation,[20] as well as changes in the diaphragm in patients with COPD.[43] [44] These intrinsic changes may result in part from deconditioning. Previous animal studies demonstrated that overloading the ventilatory muscles can produce training effects in the diaphragm and other inspiratory muscles.[45] [46] The improvement in respiratory muscle function in our training study suggests that these intrinsic skeletal muscle changes may be partially reversible with training.

Ventilatory Response to Exercise

A consistent finding in patients with heart failure is an excessive ventilatory response to exercise without evidence of arterial hypoxemia or altered carbon dioxide tension.[47] In our study, peak exercise ventilation increased significantly after training, consistent with the higher workloads achieved during bicycle exercise. However, the ventilatory response to aerobic exercise was totally unaffected by selective respiratory muscle training. Our findings are in contrast to those of Sullivan et al[27] and Coats et al,[28] who described a decrease in the ventilatory response to incremental exercise testing before and after aerobic training. Aerobic training will result in the nonspecific training of the respiratory muscles. Since an improvement in the ventilatory response to exercise is specific for whole-body but not selective respiratory muscle training, this would indicate that the improvement in the ventilatory response is probably derived from changes in the leg skeletal muscles. Aerobic training has been shown to improve skeletal muscle metabolism in patients with chronic heart failure.[24] [25] Alternatively, this beneficial effect on ventilation with aerobic training may result from increased ß-endorphin levels, an improvement in matching of ventilation to lung perfusion, or reductions in pulmonary capillary wedge pressures.

Submaximal and Maximal Exercise

Submaximal exercise performance increased significantly with training. The dyspnea scale at the completion of the 6-minute walk test was not significantly lower after training, but the distance covered during the testing period was greater. This suggests that perceived dyspnea was actually attenuated.

Desensitization to the symptom of exertional dyspnea may have occurred and may have resulted in the improved submaximal exercise performance.[48] Previous selective respiratory muscle training trials in patients with COPD report similar increases in the 6-minute walk test.[33]

Peak exercise oxygen consumption significantly increased with training. The improvement in peak $\dot{V}O_2$ is more difficult to explain. Possible mechanisms include the higher oxygen consumption by the respiratory muscles as a result of training. However, this only partially explains the increase in $\dot{V}O_2$, since exercise duration was significantly greater with training. A generalized training effect may have occurred. However, resting and exercise heart rate responses and $\dot{V}O_2$ at the anaerobic threshold were not affected by the selective muscle training. Thus, a systemic training effect appears unlikely. Motivational factors may have contributed to improved exercise performance with training. However, peak heart rates and respiratory quotients after training were not significantly different. Selective respiratory muscle training may have improved right ventricular function. Increased respiratory muscle strength may have resulted in enhanced right ventricular filling and ejection fraction. Prior studies have suggested that right ventricular ejection fraction is strongly related to peak $\dot{V}O_2$.[49] Unfortunately, we obtained no central hemodynamic measurements or noninvasive cardiac studies to assess right ventricular function and test this hypothesis. Finally, if there is a respiratory limitation to peak $\dot{V}O_2$, then selective respiratory muscle training could improve peak exercise performance. In normal subjects, selective respiratory muscle training can improve maximal exercise performance.[32] [42] [50]

Effect on Perceived Dyspnea

The majority of trained patients reported a subjective improvement in dyspnea during the activities of daily living. Improved respiratory muscle strength and/or decreased work of breathing during submaximal exercise may have produced the effect. Ventilatory reserve was enhanced with training. Desensitization to dyspnea may also have occurred.[48]

Perceived dyspnea during progressive isocapnic hyperventilation was also significantly improved with selective respiratory muscle training in the trained but not in the comparison group. This may have resulted from the improved strength and endurance of the respiratory muscles. Alternatively, desensitization to dyspnea may have occurred.

Surprisingly, during maximal bicycle exercise no alteration in perceived dyspnea occurred even at submaximal workloads. It is likely that the work of breathing and/or the tension time index of the diaphragm was lessened with training. Previously, we had described a significant correlation between Borg scale ratings of perceived dyspnea during submaximal bicycle exercise and maximal mouth pressures. We therefore had anticipated that if this was a cause-and-effect relation, then an improvement in respiratory muscle strength would have resulted in a diminution of exertional dyspnea during whole-body exercise. Our inability to demonstrate this suggests that during maximal exercise, other receptors originating in the leg (ie, ergoreceptors), chemoreceptors, or central nervous system may be dominant and that respiratory muscle input constitutes a secondary signal. Alternatively, the Borg scale may provide too crude an index by which to assess dyspnea. Breathlessness, being a complex physiological phenomenon, results from a variety of sensory inputs.[51] [52] Other investigators have emphasized that several other respiratory parameters during exercise, such as pleural pressure, peak inspired flow, tidal volume, forced vital capacity measured at rest, respiratory rate, and the duty cycle, may contribute to exertional dyspnea. What combination of variables should be used to assess dyspnea

during exercise remains unclear.

Limitations

The present study has several limitations. It was an unblinded and uncontrolled study. A comparison group that consisted of study dropouts was included. The motivation of this group was clearly less than that of the active participants. Although the baseline characteristics of the patients in the comparison group were not significantly different from those of the trained group, several parameters tended to be slightly higher in the comparison group, such as peak $\dot{V}O_2$, ejection fraction, total lung capacity, etc. These trends suggest that these comparison group patients tended to be less ill than the trained group. Nevertheless, this does not minimize our findings, since the major role of the comparison group was to demonstrate the reproducibility of the respiratory measurements in a stable heart failure population and not intergroup comparisons.

The patients who composed the comparison group underwent the same number of 6-minute walk tests and bicycle ergometer tests as the active training group in both the prestudy and poststudy phases. Therefore, a practice or learning effect did not account for the improvements noted by the trained group over the course of the study. However, the improvement shown by the trained group in ventilatory muscle endurance and ventilatory strength is more difficult to interpret. Since the active training group carried out exercises on a daily or thrice-weekly regimen, we cannot exclude the possibility that the improvements in ventilatory endurance and strength were merely a consequence of a practice effect and not derived from changes in the intrinsic properties of the respiratory muscles.

The clinical benefit to the trained group derived from repeated visits with their medical team and the psychosocial support gained from engaging in a group activity with others with similar functional impairments cannot be determined and may have impacted significantly on the findings. In a prior study investigating the impact of pulmonary rehabilitation in patients with COPD, we demonstrated that a group who received hospital-based sham therapy showed marked improvements in the ability to carry out activities of daily living compared with a control group who received placebo tablets at home.[29] These observations demonstrate that coming to the hospital several times a week to obtain outpatient rehabilitation therapy is associated with functional and psychological improvements.

The patients who completed the training program did require a small but significant increase in diuretic dosage approximately 4 weeks into the training regimen. This increased need for diuretics may have resulted from a greater activity level, which would lead to decreased renal perfusion and sodium retention. Despite the increased diuretic dosage, the weight of the trained patients remained unchanged from pretraining values. Thus, it is unlikely that the clinical benefit derived from the training program was a consequence of higher diuretic dosages.

The training regimen used was comprehensive. Which element of the strength or endurance training afforded most clinical benefit cannot be determined. Also, whether the different components of training were complementary or actually offset each other cannot be discerned, although the impressive changes in respiratory muscle function in the trained group suggest that this combination training was highly beneficial. Future studies that focus on single aspects of the training program are needed. Indeed, the training program could be greatly simplified. Home administration using the commercially available THRESHOLD device may be found to have clinical benefit and would be easily affordable and widely

applicable.

Clinical Implications

This study has a variety of clinical implications. It demonstrates that selective muscle training can provide safe and effective treatment for patients with severe congestive heart failure. Patients who participated in the training program had severely reduced exercise capacity, as evidenced by the peak $\dot{V}O_2$, which was lower than the $\dot{V}O_2$ of patients in prior training studies. Despite their severe limitation, they were able to successfully complete this rigorous program. No hospitalizations for worsening heart failure occurred, although there were two hospitalizations for angina, suggesting that the clinical benefit derived from the training program enabled the patients to be more active.

Most prior studies have focused on whole-body aerobic training in patients with heart failure.[26] [27] [28] The benefits derived from endurance training result primarily from adaptations that occur in the skeletal muscles and not from central hemodynamic changes.[26] [53] Recent studies in animals and humans suggest that aerobic training may actually be deleterious in heart failure by leading to left ventricular dilatation. A study by Jugdutt et al[34] suggests that a 12-week exercise training program in patients after extensive anterior infarctions can further distort left ventricular shape, increase infarct expansion, reduce scar thickness, and reduce ejection fraction. Endurance training in rats after large myocardial infarctions resulted in reduced survival and left ventricular dilatation.[35] Our study, which focuses on small muscle mass training that does not stress the cardiovascular system, may have the added advantage of inducing peripheral skeletal muscle changes without adverse cardiac effects. Future studies investigating the clinical benefit of low-intensity exercise training and/or regional muscle group training are warranted.

Selective respiratory muscle training was shown to improve submaximal and maximal exercise capacity. The improvements in the 6-minute walk test, exercise duration, and oxygen consumption were greater than changes previously reported with other commonly used therapeutic interventions such as digoxin or angiotensin-converting enzyme inhibitors. Simplification of the present study design may be possible and could provide a simple and useful adjunct to medical therapy.

Selective respiratory muscle training alleviated dyspnea during activities of daily living and progressive isocapnic hyperpnea but not during bicycle exercise. This suggests that the respiratory muscles are not the key modulators of this sensation during aerobic exercise. However, the symptoms of heart failure, ie, exertional fatigue and dyspnea, may still result from abnormalities in skeletal muscle function, although paradoxically, the primary stimulus for dyspnea may originate in the leg muscles.

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Footnotes

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Table 3. Results of Exercise Testing in the Trained Group

	Rest		Peak Exercise	
	Pre	Post	Pre	Post
HR, bpm	83 ±13	81±11	141±12	140±18
BP, mm Hg	91±12	83 ±12	105±16	102±16
$\dot{V}O_2$, mL · kg^{-1} · min^{-1}	3.4±0.7	3.6 ±0.6	11.4±3.3	13.3±2.7[1]
VE, L/min	11.8 ±3.1	12.6±3.5	44±15	55±12[1]
F, breaths/min	16 ±8	17±4	32±7	35±7
Respiratory quotient	0.81 ±0.06	0.9±0.09	1.07±.12	1.12±0.07
Exercise time, s		597±315		785±230[1]
$\dot{V}O_2$AT, mL · kg^{-1} · min^{-1}		7.6 ±2.2		7.8±1.3

Pre indicates prestudy; post, poststudy; HR, heart rate; bpm, beats per minute; VE, minute ventilation; F, respiratory rate; and $\dot{V}O_2$AT, oxygen consumption at the anaerobic threshold.

[1] $P<.05$ pre vs post.

Table 2. Ventilatory Muscle Endurance Measurements

	Comparison Group		Trained Group	
	Pre	Post	Pre	Post
Heart rate, bpm	101±18	104±13	93±14	94 ±15
Mean BP, mm Hg	107±20	108±20	95±14	91±12
MSVC, L/min	70±21	66±34	49±11[2]	77±15[1]
F, breaths/min	47±15	46±10	45±13	52±17[1]
Ti/TTOT	0.38±0.07	0.44±0.10	0.37±0.04	0.36±0.05
Tidal volume, L/min	1.6±0.7	1.5±0.6	1.1±0.3	1.6 ±0.6[1]

Pre indicates prestudy; post, poststudy; bpm, beats per minute; BP, blood pressure; MSVC, maximal sustainable ventilatory capacity; F, respiratory rate; and Ti/TTOT, duty cycle.

[1] $P<.05$ pre vs post;

[2] $P<.05$ comparison vs trained group.

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Table 1. Clinical Characteristics of the Comparison and Trained Groups

	Comparison Group (n=6)	Trained Group (n=8)
Training sessions, n	3±3	28±5[1]
Age, y	56 ±15	55±15
Height, in	68.2±2.2	69.8±3.9
Weight, lb	193±40	190±38
NYHA class	2.3±1.2	2.8±1.0
Ejection fraction, %	24±10	20±8
$\dot{V}O_2$, mL · kg^{-1} · min^{-1}	16.1 ±5.5	11.4±3.3
Lasix dose, mg	47±21	150±90[1]

[1] $P<.05$ comparison vs trained group.

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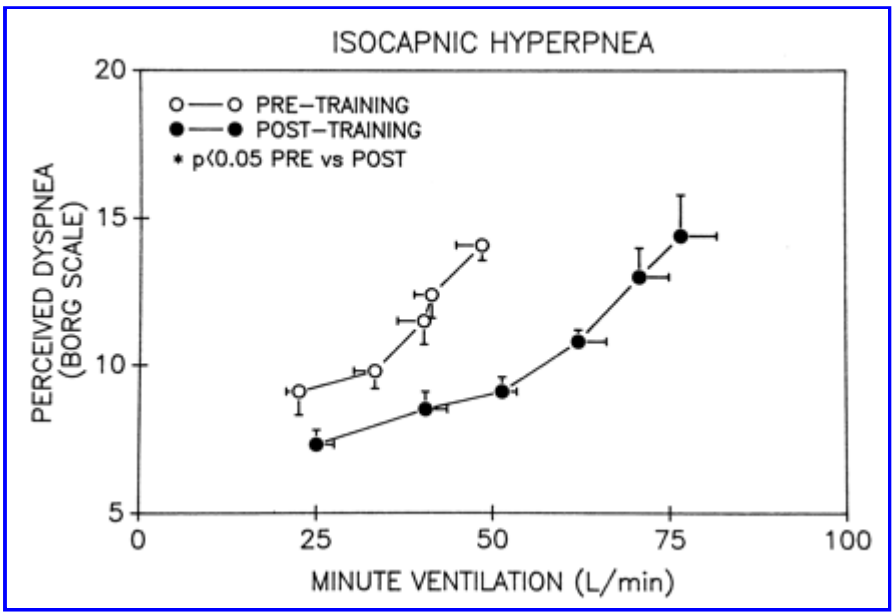

Figure 4. Graph showing perceived dyspnea during isocapnic hyperpnea in the trained group before and after training.

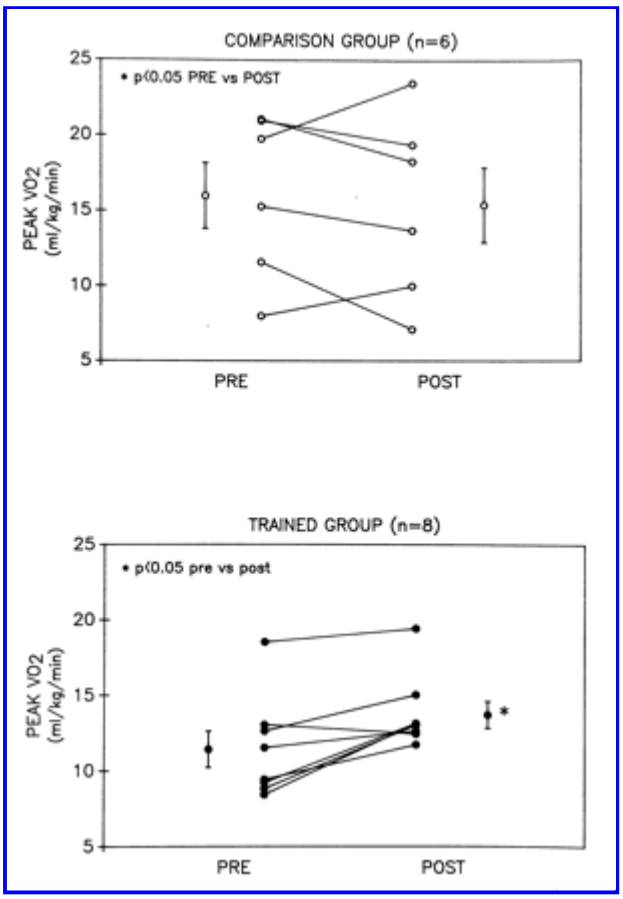

Figure 3. Graphs showing results (mean±SEM) of peak exercise oxygen consumption (Peak VO_2) in the comparison and trained groups.

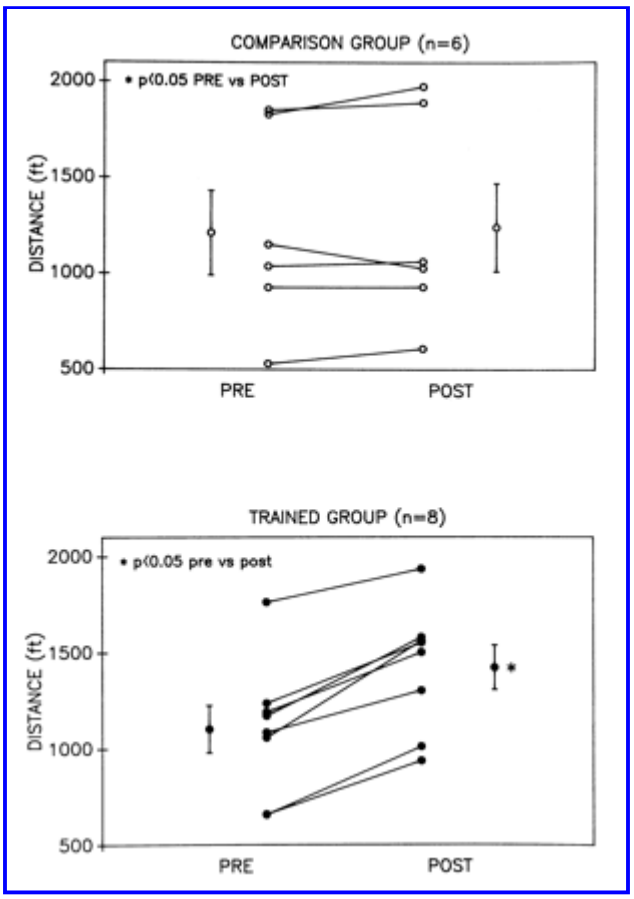

Figure 2. Graphs showing results (mean±SEM) of the 6-minute walk test before and after training in the comparison and trained groups.

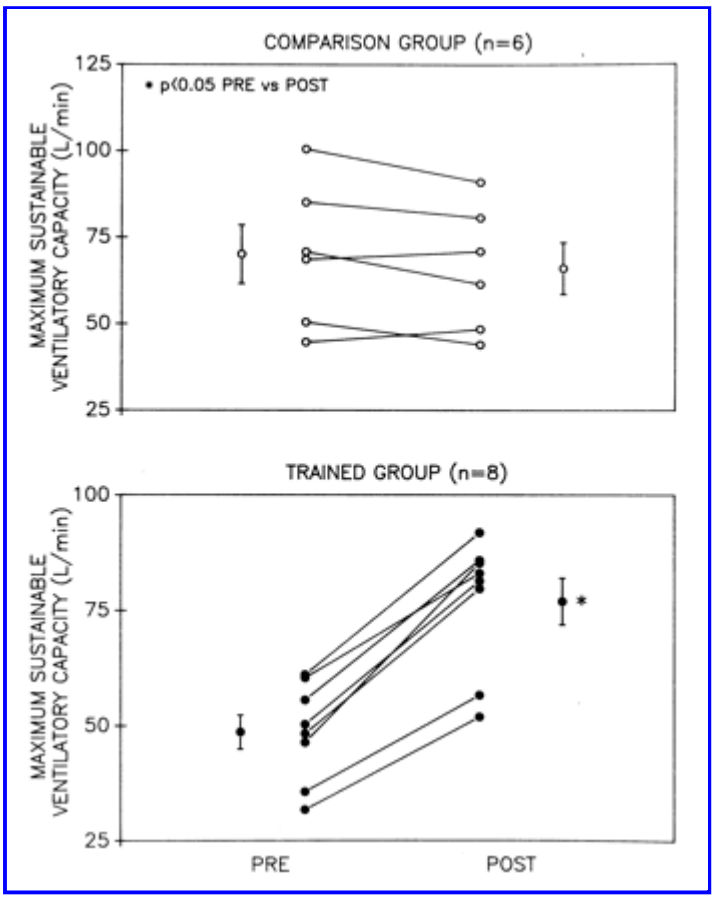

Figure 1. Graphs showing maximum sustainable ventilatory capacity (mean±SEM) for the individual patients in the comparison and trained groups before and after the study period.

Table 4. Subjective Changes to Training

	Comparison Group	Trained Group
Dyspnea, n		
Improved	1	6[1]
Unchanged	3	2
Worse	2	0
Emergency room visits, n	5	2
Hospitalizations, n		
CHF	1	0
Angina	0	2
Infection	1	0
Other	1	0

CHF indicates congestive heart failure.

[1] $P<.05$ comparison vs trained group.