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The Effect of Specific Inspiratory Muscle Training on the Sensation of Dyspnea and Exercise Tolerance in Patients with Congestive Heart Failure

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Summary

Background: It has been previously shown that the inspiratory muscles of patients with congestive heart failure (CHF) are weaker than those of normal persons. This weakness may contribute to the dyspnea and limit exercise capacity in these patients. The respiratory muscles can be trained for both strength and endurance.

Hypothesis: The present study was designed to evaluate the effect of specific inspiratory muscle training (SIMT) on inspiratory muscle performance, lung function, dyspnea, and exercise capacity in patients with moderate heart failure.

Methods: Twenty patients with CHF (NYHA functional class II–III) were recruited for the study. The subjects were randomized into two groups: 10 patients were included in the study group and received SIMT and 10 patients were assigned to the control group and received sham training. Subjects in both groups trained daily, 6 times/week, for one-half h, for 3 months. The subjects started breathing at a resistance equal to 15% of their PImax for 1 week and the resistance was then increased incrementally to 60%. Spirometry, inspiratory muscle strength (assessed by measuring the PImax at residual volume), and endurance (expressed by the relationship between PmPeak and PImax), the 12-min walk test, and peak VO_2 were performed before the beginning and at the end of the training period.

Results: All patients in the training group showed an increase in the inspiratory muscle strength [mean (± standard error of the mean) PImax increased from 46.5 ± 4.7 to 63.6 ± 4.0 cm H_2O, $p < 0.005$], and endurance (mean PmPeak/PImax from 47.8 ± 3.6 to 67.7 ± 1.7%, $p < 0.05$), while they remained unchanged in the control group. This was associated in the training group with a small but significant increase in forced vital capacity, a significant increase in the distance walked (458 ± 29 to 562 ± 32 m, $p < 0.01$), and an improvement in the dyspnea index score. No statistically significant change in the mean peak VO_2 was noted in either group.

Conclusions: Specific inspiratory muscle training resulted in increased inspiratory muscle strength and endurance. This increase was associated with decreased dyspnea, increase in submaximal exercise capacity, and no change in maximal exercise capacity. This training may prove to be a complementary therapy in patients with congestive heart failure.

Key words: specific inspiratory muscle training, dyspnea, exercise tolerance, congestive heart failure

Introduction

The mechanism of dyspnea in chronic heart failure is not well understood,[1] and it correlates poorly with the severity of heart failure.[2–4] Dyspnea in acute heart failure is a consequence of raised left atrial pressure;[5] however, it does not appear to be due to changes in pulmonary vascular pressures in chronic heart failure.[6,7]

Studies investigating dyspnea suggest that dyspnea, at least in part, is perceived as respiratory muscle effort.[8,9] In addition, several studies have been carried out in order to correlate dyspnea and respiratory muscle performance. It is well documented that the degree of breathlessness, subjectively reported by the patients, is related to the activity and the strength of the inspiratory muscles.[10,11] It has been previously shown that the respiratory muscles of patients with congestive heart failure (CHF) are weaker than those of normal persons.[12,13] Moreover, when Hammond *et al.*[14] attempted to measure inspiratory muscle endurance, most patients with CHF could not breathe on an inspiratory threshold loading device for more than 1 or 2 min, even at 40–50% of their PImax. The symptoms of heart failure, that is, exertional fatigue and dyspnea, may result in part from this impaired respiratory muscle performance.

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It is well established that respiratory muscles can be trained like other skeletal muscles.[15–17] Respiratory muscle training may be particularly advantageous for patients with heart failure, since an increase in inspiratory muscle strength and endurance may attenuate dyspnea and enhance exercise performance. In one previous study, the benefit of respiratory muscle training in patients with CHF was studied.[18] It was concluded that respiratory muscle training improved exercise capacity and dyspnea during daily activities in patients with CHF.

We hypothesized that if respiratory muscle weakness plays a role in the sensation of dyspnea, then respiratory muscle training that improves respiratory muscle performance will result in attenuation of dyspnea and endurance exercise performance in patients with CHF.

Patients and Methods

Twenty patients with CHF (18 men and 2 women) with an age from 55 to 74 years [mean ± standard error of the mean (SEM) 68 ± 6.2] were recruited for the study. Inclusion criteria included the following: (1) New York Heart Association (NYHA) functional class II–III (defined by two cardiologists who were blinded to the study design), (2) left ventricular ejection fraction (LVEF) < 30%, (3) no evidence of lung disease, (4) clinical stability, and (5) PImax < 70% of predicted normal values. All patients were receiving digoxin, diuretics, and angiotensin-converting enzyme (ACE) inhibitors while entering the study, and the medication was not changed during the study. The study was approved by the institutional committee on human research and informed consent was obtained from all patients.

Study Design

The subjects were randomized into two groups: 10 patients comprised the study group and received specific inspiratory muscle training (SIMT) (Group 1), and 10 patients were assigned to the control group and received sham training (Group 2).

Tests

All tests were performed before and at the end of the training period. Several training tests before the baseline value were performed in all patients to correct possible training and learning effect. List of abbreviations and meanings are shown in Table I.

Spirometry: Pulmonary functions were assessed by spirometry, performed before and at the end of the training period. The forced vital capacity (FVC) and the forced expiratory volume in one second (FEV1) were measured three times on a computerized spirometer (Compact, Vitalograph, Buckingham, England) and the best trial is reported.

Respiratory muscle strength: Respiratory muscle strength was assessed by measuring the maximal inspiratory mouth pressure (PImax) at residual volume (RV) and the maximal expiratory mouth pressure (PEmax) at total lung capacity (TLC) as previously described by Black and Hyatt.[19] The value obtained from the best of at least three efforts was used.

TABLE I List of abbreviations

Abbreviation	Meaning
NYHA	New York Heart Association
LVEF	Left ventricular ejection fraction
VO_2max	Maximal O_2 consumption during maximal exercise
PImax	Maximal inspiratory pressure—assesses the inspiratory muscle strength
PEmax	Maximal expiratory pressure—assesses the expiratory muscle strength
P_mPeak	Maximal negative pressure produced by the patient handling incremental resistance during the endurance test
FVC	Forced vital capacity
FEV_1	Forced expiratory volume during the first second of maximal expiration
RV	Residual volume
TLC	Total lung capacity

Inspiratory muscle endurance: To determine inspiratory muscle endurance, a device similar to that proposed by Nickerson and Keens[20] was used. Subjects inspired through a two-way Hans-Rudolph valve whose inspiratory port was connected to a chamber and plunger to which weights could be added externally. Inspiratory elastic work was then increased by the progressive addition of 25 to 100 g weights at 2 min intervals, as was previously described by Martyn *et al.*,[21] until the subjects were exhausted and could no longer inspire. The pressure achieved with the heaviest load (tolerated for at least 60 s) was defined as the peak pressure (PmPeak).

Twelve-minute walk: The distance the patient was able to walk in 12 min was determined in a measured corridor as described by McGavin *et al.*[22]

Exercise tolerance test: Exercise testing was performed using the multi-stage testing technique on a treadmill. The patient began at the lowest possible speed, and the speed was then increased at the conclusion of each min of exercise until exercise could not be continued. Throughout the procedure, oxygen consumption, CO_2 production, tidal volume, and minute ventilation, as well as arterial oxygen saturation were measured.

Training protocol: Subjects in both groups trained daily, six times a week, each session consisting of one-half hour training, for 3 months. The training was performed under the supervision of a physiotherapist. The subjects received SIMT with a threshold inspiratory muscle trainer (Threshold™ Inspiratory Muscle Trainer, Healthscan, N.J.). The subjects started breathing at a resistance equal to 15% of their PImax for 1 week. The resistance was then increased incrementally, 5% each session, to reach 60% of their PImax at the end of the first month. The SIMT was then continued for the next 2 months at 60% of the subjects' PImax, adjusted every week to the new

PImax achieved. Patients in Group 2 received sham training with the same device, but with no resistance.

Dyspnea index: The clinical rating of dyspnea was graded by a physician blinded to the kind of treatment the patients had received, using the dyspnea index described by Mahler and Harver.[23] This index consists of three components evoking dyspnea: magnitude of task, magnitude of effort, and the functional impairment occurring as a consequence of dyspnea. The physician selected from one of five grades of dyspnea (0–4 scale) for each of the three components, and the mean of the three numbers is reported.

Data Analysis

The results are expressed as means ± SEM. Comparisons of lung function, respiratory muscle strength, inspiratory muscle endurance, 12-min walk, exercise tolerance test, and rating of dyspnea, within and between the two groups, were carried out using the ANOVA two-way repeated measures analysis of variance.

Results

The characteristics of 20 patients with CHF are summarized in Table II. Four patients of Group 2 dropped out of the study because they became aware that they were undergoing sham training. However, all completed all the tests and their results were included in Group 2.

Respiratory muscle strength: The effect of the training on the inspiratory muscle strength is shown in Figure 1. All patients in Group 1 showed an increase in the inspiratory muscle strength, as was assessed by measuring the PImax at RV. Mean PImax increased significantly from 46.5 ± 4.7 to 63.6 ± 4.0 cm H_2O ($p < 0.005$). Mean PImax remained unchanged in Group 2 patients. The mean PEmax also remained unchanged in the patients of both groups.

TABLE II Characteristics of patients with congestive heart failure[a]

	Training group	Control group
Age (years)	66.2 ± 4.6	63.8 ± 4.0
Height (cm)	170.6 ± 8.3	171.2 ± 7.9
Weight (kg)	75.3 ± 3.8	77.3 ± 4.5
NYHA class	2.3 ± 0.2	2.4 ± 0.2
LVEF (%)	24.7 ± 1.6	22.9 ± 2.4
$\dot{V}O_2$max (ml·Kg^{-1}·min^{-1})	13.1 ± 0.8	13.5 ± 0.9
PI_{max} (cm H_2O)	46.5 ± 4.7	50.7 ± 4.2
(% of Pred.)	(44)	(47)
PE_{max} (cm H_2O)	82.1 ± 6.1	80.8 ± 5.7
(% of Pred.)	(41)	(40)
P_mPeak/PI_{max} (%)	47.8 ± 3.6	45.6 ± 3.5
(% of Pred.)	(64)	(62)
12-min walk (m)	458 ± 29	428 ± 31
Dyspnea index	1.70 ± 0.2	1.75 ± 0.2
FVC (L)	3.14 ± 0.2	3.02 ± 0.6
(% of Pred.)	(86)	(83)
FEV_1 (L)	2.46 ± 0.2	2.33 ± 0.2
(% of Pred.)	(86)	(85)

[a] Values are expressed as mean ± standard error of the mean.
Abbreviation: Pred. = predicted normal values. Other abbreviations as in Table I.

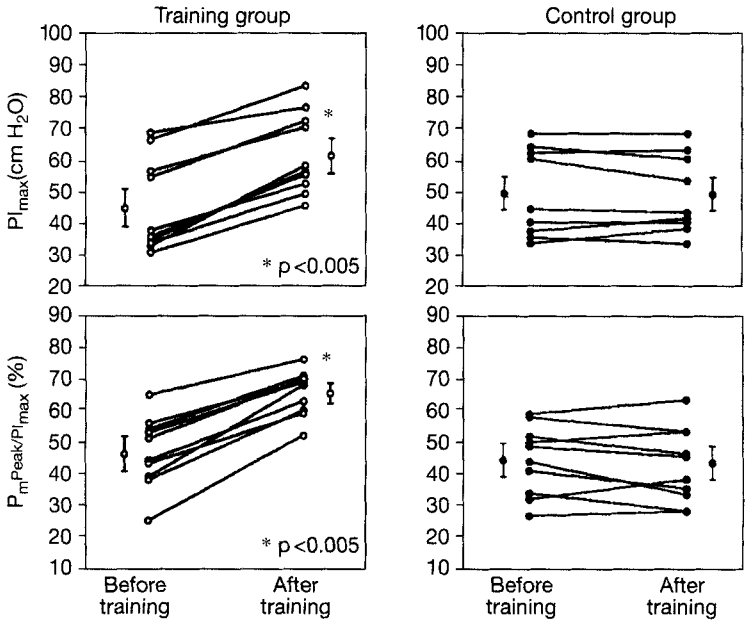

FIG. 1 Inspiratory muscle strength, as expressed by the PImax at residual value, and inspiratory muscle endurance as expressed by the relationship between PmPeak and the PImax, before and following 3 months of training, in the control group (Group 2) and in the training group (Group 1).

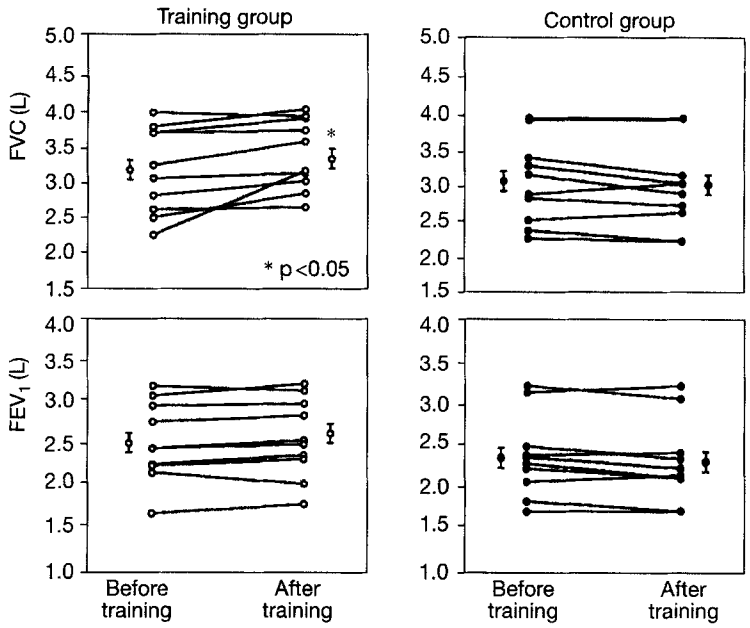

FIG. 2 Spirometry data before and following 3 months of training in the control group (Group 2) and in the training group (Group 1).

Respiratory muscle endurance: The respiratory muscle endurance, as expressed by the relationship between PmPeak and PImax, also increased significantly, from 47.8 ± 3.6 to 67.7 ± 1.7%, $p < 0.05$, in Group 1 but not in Group 2 patients (Fig. 1).

Spirometry: The improved respiratory muscle performance was associated with a small but significant increase, from 3.14 ± 0.5 to 3.37 ± 0.2 (mean ± SEM, $p < 0.05$), in FVC and remained almost unchanged in Group 2 (Fig. 2).

Twelve-minute walk: The results of the 12-min walk test are shown in Figure 3. There was no difference in the mean 12-min distance walk between the groups before training. However, the patients in Group 1 showed a significant increase in the distance walked after the training: from 458 ± 29 to 562 ± 32 m ($p < 0.01$). In Group 2 there was a small, not significant decrease in the 12-min walk test after 3 months (from 428 ± 31 to 419 ± 25 m).

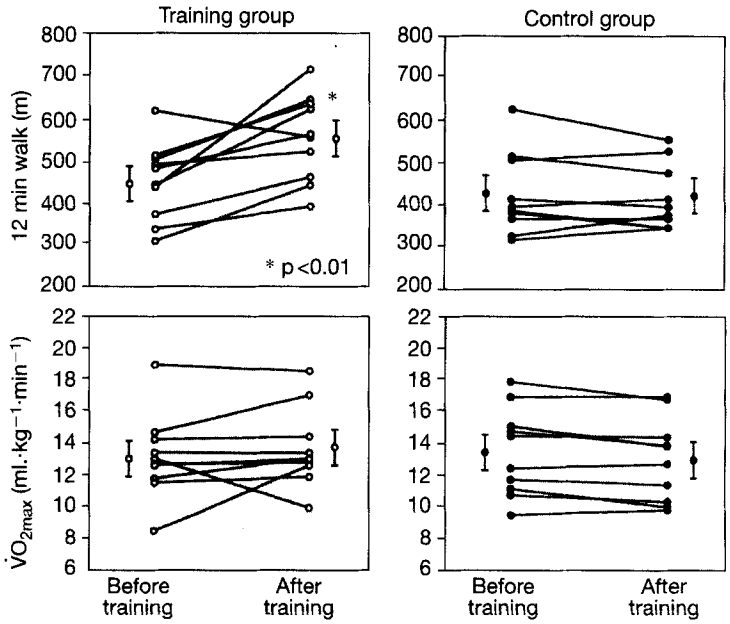

FIG. 3 Results of the 12-min walk test and the peak exercise oxygen consumption (VO_2max) before and following 3 months of training in the control group (Group 2) and in the training group (Group 1).

TABLE III Respiratory rate during rest and during peak exercise

	Training group		Control group	
	Pre	Post	Pre	Post
RR, tpm (rest)	18±4	17±4	17±5	17±5
RR, tpm (peak exercise)	34±6	33±6	37±7	37±6

Abbreviations: RR = respiratory rate, tpm = times per minute, Pre = prestudy, Post = poststudy.

Exercise tolerance test: No statistically significant change in the mean peak VO_2 was noted in both groups (Fig. 3). However, 3 of the 10 patients in Group 1, but none in Group 2, increased peak VO_2 over the time of the study (pre: 8.5, 11.8, 14.7, vs. post: 12.6, 13.1, 17.0 ml·kg-1·min-1, respectively). The respiratory rates during rest and during peak exercise are shown in Table III.

Dyspnea index: The clinical rating of dyspnea was assessed before and at the end of the training period. Mean dyspnea index score also increased significantly from 1.70±0.2 to 2.70±0.2 ($p < 0.005$) in Group 1 and remained unchanged in Group 2. The results of the individual dyspnea index score are shown in Figure 4. All Group 1 patients but one improved their dyspnea index score significantly.

Discussion

Our study showed that in patients with CHF the inspiratory muscle performance was significantly decreased before training. What are the possible reasons for the weakened inspiratory muscles? Since patients with severe CHF generally have low cardiac output, we believe that they probably have relatively reduced blood flow to the respiratory muscles[14] despite increased sympathetic drive and respiratory rate, and that this may play a role in this weakness.

The efficacy of the SIMT program was demonstrated in our study by the significant increase in respiratory muscle strength and endurance in all patients in the training group. The magnitude of the improvement of the inspiratory muscle performance was similar to prior reports in patients with chronic obstructive pulmonary disease.[24,25]

The impact of the SIMT program on FVC was small but significant. This increase in vital capacity is probably related to the increase in inspiratory muscle strength.

The majority of trained patients reported a subjective improvement in dyspnea during daily activities following the training period. Dyspnea is a complex physiologic phenomenon and results from many sensory inputs.[23] Recent studies in patients with respiratory disease and in normal individuals suggest that the magnitude of dyspnea experienced is at least partly determined by the perception of the work of muscles of inspiration and by their strength.[1,9,26] Therefore, it is not surprising that we observed improvement in reported dyspnea following the training period in our training group.

Submaximal exercise performance, assessed by the 12-min walk test, also increased significantly with training. As exertional dyspnea might have limited submaximal exercise performance, it is suggested that the increased submaximal exercise performance in the trained patients is associated with decreased dyspnea, observed with training. Similar results were reported in the study performed by Mancini *et al.*[18]

Peak exercise oxygen consumption remained unchanged following training. As only selective respiratory muscle training was performed in our study, it is logical to assume that a generalized training effect is unlikely. It is more difficult to explain how three subjects in Group 1 improved their VO_2 max significantly. Possible mechanisms include (1) a higher oxygen consumption by the trained respiratory muscles; (2) a generalized training effect, although it seems unlikely to occur. It was shown previously[27] that neurohumoral hormones and inflammatory cytokines may have an adverse effect on the heart in CHF and may themselves contribute to the development of heart failure. Endurance exercise training exerts its benefit in large part through neurohumoral modulation; (3) motivational factors; (4) improved right heart function following training. Increased inspiratory muscle performance may have resulted in improved right ventricular ejection fraction. It has been previously shown that right ventricular ejection fraction is related to VO_2 max[28]—however, no hemodynamic

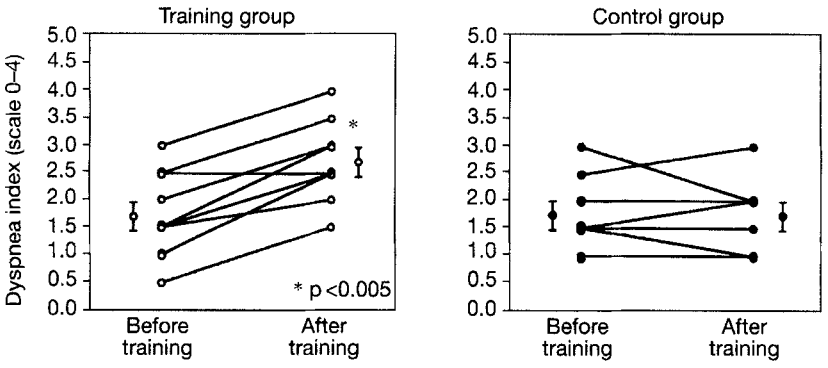

FIG. 4 Subjective perceived dyspnea, during daily activities, before and following 3 months of training in the control group (Group 2) and in the training group (Group 1).

measurements were performed in our study; and (5) improved respiratory muscle function that can enhance maximal exercise performance[29] if respiratory limitation occurred during exercise tolerance test.

A limitation of our study is that patients in the control group became aware that they were performing only sham training, and therefore the motivation of this group of patients was clearly decreased compared with the training group. A motivational factor for the improvement in the training group therefore cannot be denied. However, all the patients in the control group underwent the same number of tests, pre- and post-training, with no improvement in any of the parameters studied, and therefore practice or a learning effect did not account for the improvement noted in the training group.

This study demonstrates that the respiratory muscles can be safely trained in patients with CHF. The SIMT improved the respiratory muscle performance, and this improvement was associated with increased submaximal exercise capacity and alleviated dyspnea during daily activities. Most previous studies in patients with CHF have shown that exercise training can further decrease cardiac ejection fraction[30, 31] and, if benefit occurs, it derives primarily from adaptation of skeletal muscles rather than from hemodynamic changes.[32, 33] Only in one previous research[18] was the benefit of SIMT in patients with CHF studied. Although in that study only eight patients completed the training program and no control group was included, our study is in agreement with most of the parameters studied. Respiratory muscle performance, submaximal exercise test, and dyspnea improved significantly in both studies. However, in contrast to their results that showed a small but significant improvement in maximal exercise performance (VO_{2max} increased from 11.4 ± 3.3 to 13.3 ± 2.7 ml/kg/min), only three patients in our group had similar improvement.

We believe that SIMT may prove to be a complementary therapy, with the aim of improving capacity of handling daily activities with less dyspnea in patients with CHF.

References

1. Cokcroft A, Guz A: Breathlessness. *Postgrad Med J* 1987;63:637–641
2. Sullivan MJ, Higginbotham MB, Cobb FR: Increased exercise ventilation in patients with chronic heart failure: Intact ventilatory control despite hemodynamic and pulmonary abnormalities. *Circulation* 1988;77:552–559
3. Franciosa JA, Leddy CL, Wilen M, Schwartz DE: Relation between hemodynamic and ventilatory responses in determining exercise capacity in severe congestive heart failure. *Am J Cardiol* 1984;53:127–134
4. Packer M: How should we judge the efficacy of drug therapy in patients with chronic congestive heart failure? The insights of six blind men. *J Am Coll Cardiol* 1987;9:433–438
5. Lipkin DP, Poole-Wilson PA: Symptoms limiting exercise in chronic heart failure. *Br Med J* 1986;292:1030–1031
6. Gibbs JSR, Keegan J, Wright C, Fox KM, Poole-Wilson PA: Pulmonary artery pressure changes during exercise and daily activities in chronic heart failure. *J Am Coll Cardiol* 1990;15:52–61
7. Lipkin DP, Canepa-Anson R, Stephens MR, Poole-Wilson PA: Factors determining symptoms in heart failure: Comparison of fast and slow exercise tests. *Br Heart J* 1986;55:439–445
8. Killian KG, Campbell EJM: Dyspnea and exercise. *Ann Rev Physiol* 1983;445:465–479
9. Killian KG, Jones NL: The use of exercise testing and other methods in the investigation of dyspnea. *Clin Chest Med* 1984;5:99–108
10. Jones GL, Killian KJ, Summers E: The sense of effort, oxygen cost, and pattern of breathing associated with progressive elastic loading to fatigue (abstr). *Fed Proc* 1984;42:1420
11. Killian KJ, Gandevia SC, Summers E: Effect of increased lung volume on perception of breathlessness, effort, and tension. *J Appl Physiol* 1984;57:686–691
12. McParland C, Krishnan B, Wang Y, Gallagher CG: Inspiratory muscle weakness and dyspnea in chronic heart failure. *Am Rev Respir Dis* 1992;146:467–472
13. Mancini DM, Henson D, LaManca J, Levine S: Respiratory muscle function and dyspnea in patients with chronic congestive heart failure. *Circulation* 1992;86:909–918
14. Hammond MD, Bauer KA, Sharp JT, Rocha RD: Respiratory muscle strength in congestive heart failure. *Chest* 1990;98:1091–1094
15. Pardy RL, Leith DE: Ventilatory muscle training. *Respir Care* 1984;29:278–284
16. Shaffer TH, Wolfson MR, Bhutani VK: Respiratory muscle function, assessment, and training. *Phys Ther* 1981;61:1711–1723
17. Pardy RL, Leith DE: Ventilatory muscle training. In *The Thorax* (Eds. Roussos C, Macklem PT), p. 1353–1369. New York: Marcel Dekker Inc., 1986
18. Mancini DM, Henson D, LaManca J, Donchez L, Levine S: Benefit of selective respiratory muscle training on exercise capacity in patients with chronic congestive heart failure. *Circulation* 1995;91:320–329
19. Black LF, Hyatt RE: Maximal respiratory pressures: Normal values and relationship to age and sex. *Am Rev Respir Dis* 1969;99:696–702
20. Nickerson BG, Keens TG: Measuring ventilatory muscle endurance in humans as sustainable inspiratory pressure. *J Appl Physiol* 1982;52:768–772
21. Martyn JB, Moreno RH, Pare PD, Pardy RL: Measurement of inspiratory muscle performance with incremental threshold loading. *Am Rev Respir Dis* 1987;135:919–923
22. McGavin CR, Gupta SP, McHardy GJR: Twelve-minute walking test for assessing disability in chronic bronchitis. *Br Med J* 1976;1:822–823
23. Mahler D, Harver A: Clinical measurement of dyspnea. In *Dyspnea* (Ed. Mahler D), p. 75–100. Mt Kisco, N.Y.: Futura Publishing, 1990
24. Beiman M: Respiratory training and unloading. In *Principles and Practice of Pulmonary Rehabilitation* (Eds. Casaburi R, Petty T), p. 225–240. Philadelphia, Pa.: WB Saunders Co, 1993
25. Weiner P, Azgad Y, Ganam R: Inspiratory muscle training combined with general exercise reconditioning in patient with COPD. *Chest* 1992;102:1351–1356
26. Altose M: Assessment and management of breathlessness. *Chest* 1985;88:77S–83S
27. Middlekauff HR, Mark AL: The treatment of heart failure: The role of neurohumoral activation. *Intern Med* 1993;37(2):112–122
28. Polak J, Holman B, Wynne J, Colucci WS: Right ventricular ejection fraction: An indicator of increased mortality in patients with congestive heart failure associates with coronary artery disease. *J Am Coll Cardiol* 1983;2:217–224
29. Leith D, Bradley M: Ventilatory strength and endurance training. *J Appl Physiol* 1976;41:508–516
30. Jugdutt B, Michorowski B, Kappagoda C: Exercise training after anterior Q wave myocardial infarction: Importance of regional left ventricular function and topography. *J Am Coll Cardiol* 1988;12:362–372
31. Gaudron P, Hu K, Schamberger R, Budin M Walter B, Ertl G: Effect of endurance training early or late after coronary artery occlusion on left ventricular remodeling, hemodynamics, and survival in rats with chronic transmural myocardial infarction. *Circulation* 1994;89:402–412
32. Sullivan M, Higginbotham M, Coob F: Exercise training in patients with severe left ventricular dysfunction: Hemodynamic and metabolic effects. *Circulation* 1988;778:506–515
33. Musch T, Moore R, Smaldone P, Riedy M, Zelis R: Cardiac adaptations to endurance training in rats with a chronic myocardial infarction. *J Appl Physiol* 1989;66:712–719