Effects of Breathing Retraining in Patients With Chronic Obstructive Pulmonary Disease*

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Subjects with severe chronic obstructive pulmonary disease (COPD) were studied to evaluate the effects of breathing retraining (BRT) on exercise tolerance. Twenty-two subjects exercised on a treadmill three times weekly for six weeks. Twelve of the subjects (controls) then exercised for three more weeks; the other ten subjects received three more weeks of exercise reconditioning plus BRT. Results of routine pulmonary function and exercise tests were similar in both groups at the beginning of the study and after six weeks of exercise. However, in the last three weeks of the study, increments in exercise performance were significantly greater in the BRT subjects than in controls ($P < .002$). Following BRT, respiratory rate during exercise decreased ($P < .005$) and tidal volume and PaO_2 during exercise increased ($P < .05$). Thus, these data suggest that BRT increases exercise performance in subjects with severe COPD.

Several investigators have shown significant improvement in exercise tolerance following pulmonary rehabilitation programs consisting of several treatment modalities including bronchodilator therapy, antibiotic therapy, oxygen, postural drainage, somatic (exercise) reconditioning and breathing retraining (BRT).[1-4] However, the relative contribution of each of these modalities to the overall improvement of the patients is not clear. Pierce and associates[5] have shown that, following exercise training, maximal oxygen consumption was higher, and, at any given level of exercise, minute ventilation, oxygen consumption, and heart rate were lower even though there was no improvement in ventilatory function or lung volumes. Others have confirmed the beneficial effects of exercise reconditioning without BRT.[6-10]

On the other hand, pursed-lip breathing and abdominal augmentation can effect a decrease in minute ventilation and respiratory rate and an increase in tidal volume, as well as an improvement in blood gas tensions.[11-13] Motley[14] found that slow deep breathing led to a reduction in the ratio of dead space to tidal volume and an increase in oxygen saturation in most patients with emphysema. Following a program of breathing retraining. Sinclair[15] reported a reduction of inefficient spinal and should girdle movements during respiration. Nevertheless, Levy[16] has suggested that breathing retraining "has no substantial effect on ventilatory capacity and blood gas tensions in groups of patients with obstructive pulmonary disease."

In view of these conflicting findings, we undertook a study to evaluate the relative contributions of somatic reconditioning and BRT in a pulmonary rehabilitation program.

Material and Methods

Subjects: Subjects with physician-diagnosed severe chronic obstructive pulmonary disease (COPD) were chosen for participation in this study. Group 1 consisted of ten male subjects, aged 58.6±8.5 years (mean±SD). Group 2 consisted of 12 subjects, eight male and four female. This group averaged 59.7±6.6 years of age. All subjects in both groups met the following criteria for entry into the study: (1) a forced expiratory volume in one second FEV_1 equal to or less than 1.2 L and 50 percent of predicted; (2) a ratio of FEV_1/forced vital capacity (FEV_1/FVC) less than 50 percent; (3) improvement of FEV_1 by less than 20 percent after administration of aerosolized metaproterenol sulfate (1.3 mg); (4) a single-breath-diffusing capacity for carbon monoxide ($DLco_{SB}$) less than 60 percent of predicted (after correction of blood hemoglobin concentration to 14.5 g percent); (5) steady-state regarding smoking status; (6) absence of significant sputum production; (7) no serious medical problems other than COPD and its consequences; (8) ability to stabilize and maximize medical management; (9) willingness to sign an informed consent statement concerning participation in the study.

Physiologic measurements: Spirometry, lung volumes, and

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airways resistance (R_{aw}) were determined by standard techniques using a 13.5-L, water-sealed spirometer and a constant volume body plethysmograph.[17-19] Distribution of ventilation was measured using a modified single-breath nitrogen dilution technique.[20,21] The $DLco_{SB}$ was determined by the method of Ogilvie and associates.[22]

Arterial blood gas analyses were done using standard electrode methods. Samples were drawn by percutaneous puncture or, during exercise studies, from indwelling arterial cannulae. Arterial oxygen tension (PaO_2), arterial carbon dioxide tension ($PaCO_2$), and arterial pH were measured; base excess or deficit were calculated using a standard formula.

Magnetometry of the anterior-posterior (AP) chest, lateral chest, and AP abdomen was performed as described by Ashutosh and Gilbert.[23,24] An oscilloscopic display was used to view abdominal and thoracic motion during biofeedback maneuvers. Electromyography was performed using surface electrodes. Electromyographic signals were amplified using a Nicolet HGA-100 preamplifier and an A-C amplifier (Electronics for medicine, ACV-21 channel for the VR-6 oscilloscope). Signals were recorded on the VR-6 oscilloscope; a visual display was used for biofeedback maneuvers. Respiratory rate, tidal volume, and minute ventilation at rest and during exercise were determined using impedance pneumography.[25] Heart rate at rest and during exercise was monitored with surface ECG chest leads and an oscilloscope.

Exercise stress testing was performed using a treadmill while monitoring ventilatory, blood gas, and circulatory variables as just described. The treadmill had no side rails; however, the patients were allowed to balance themselves (without bearing weight) on front handrails. Patients were exercised to maximum using a steady-state protocol. Maximal exercise tolerance was defined as the greatest level of exercise a patient could maintain for three minutes.

Work performance during treadmill exercise was estimated using the method of Workman and Armstrong.[26] With this method, work is a function of speed and grade of the treadmill, body height, and weight and can be quantitated in units of estimated oxygen consumption ($\dot{V}o_2$). Workman and Armstrong found an excellent correlation between estimated and actual $\dot{V}o_2$ ($r = 0.94$).[26] Nevertheless, the relationship between such estimates of external work performance and actual $\dot{V}o_2$ is approximate since subjects differ in the amount of energy expended in doing this type of mechanical work. Initial improvements in treadmill work performance may be due to increased efficiency in using the apparatus.[8] Yet, in the study of Paez et al,[8] more than half of the improvement due to lengthening of stride occurred by the eighth day of training, suggesting that much of the improvement in efficiency of treadmill use occurs early. Once acclimation to the apparatus has occurred, relative changes in the individual's treadmill work performance can be quantitated by serially estimating Vo_2. Such serial changes in estimated $\dot{V}o_2$ should parallel relative changes in actual $\dot{V}o_2$. In this regard, Bruce and associates[27] found excellent correlation ($r > 0.90$) between treadmill work performance and measured $\dot{V}o_2$.[27]

Experimental Design

Group 1: Medical management consisted of therapy, as indicated, with bronchodilators, antibiotics, diuretics, digoxin, and oxygen. When medical management was judged to have been optimized, the patients advanced into the somatic (exercise) reconditioning phase of the program. Baseline pulmonary function tests, arterial blood gas, and treadmill exercise tests were performed before initiation of exercise reconditioning. These studies (pulmonary function tests, blood gas, exercise tests) were repeated at the end of the period of exercise reconditioning and also after the subsequent period of BRT. Thus, physiologic data were available and comparable from baseline, postexercise reconditioning, and post-BRT.

Somatic reconditioning consisted of a program of treadmill exercise, performed as an outpatient three times weekly for six consecutive weeks. An exercise period would last one hour, approximately 30 minutes of which would be spent exercising and 30 minutes spent resting. Each exercise session consisted of a warm-up period at slow speeds; rest; at stress period during which the patient would be encouraged to exceed for three minutes his previous maximal exercise tolerance; rest; and then several longer exercise periods (longer than three minutes) at submaximal work loads. Maximal stress testing was performed only after the subjects had rested sufficiently to allow their heart rate to return to baseline levels.

Breathing retraining consisted of: (1) education about chronic obstructive lung disease, (2) pursed-lip breathing,[11,12] (3) expiratory abdominal augmentation,[13] (4) synchronization of movement of the abdomen and thorax using magnetometry and biofeedback, (5) relaxation techniques for the accessory respiratory muscles[28] using electromyography and biofeedback, and (6) pyschologic assurance. Attempts were made to teach the patients to integrate these BRT techniques into their activities of daily living. Breathing retraining was continued for three weeks in all group 1 subjects.

Group 2: The medical management protocol used in group 2 subjects was identical to the protocol used in group 1. The somatic reconditioning protocols were also identical, with the single exception that group 2 subjects received nine consecutive weeks of somatic reconditioning. Thus, during the three-week period when group 1 subjects were receiving BRT, group 2 subjects received only exercise reconditioning (ER).

Statistics

Mean, standard deviation, and standard error of the mean were calculated from standard equations. Mean data at different stages of the exercise and BRT programs were compared within and between groups using an analysis of variance.[29] A p value of 0.05 or less was considered statistically significant.

Results

There were no complications of exercise in this study. Several subjects in both groups required oxygen therapy during exercise. Oxygen was administered by nasal cannula from the beginning of the program and the flow rates were kept constant for the duration of the study. Results from male and female subjects in group 2 were compared. Although there were some minor differences in baseline data related mainly to body size, response to the program was not related to the gender of the patients. Therefore, results were combined for all male and female group 2 subjects and compared with the data from group 1 subjects.

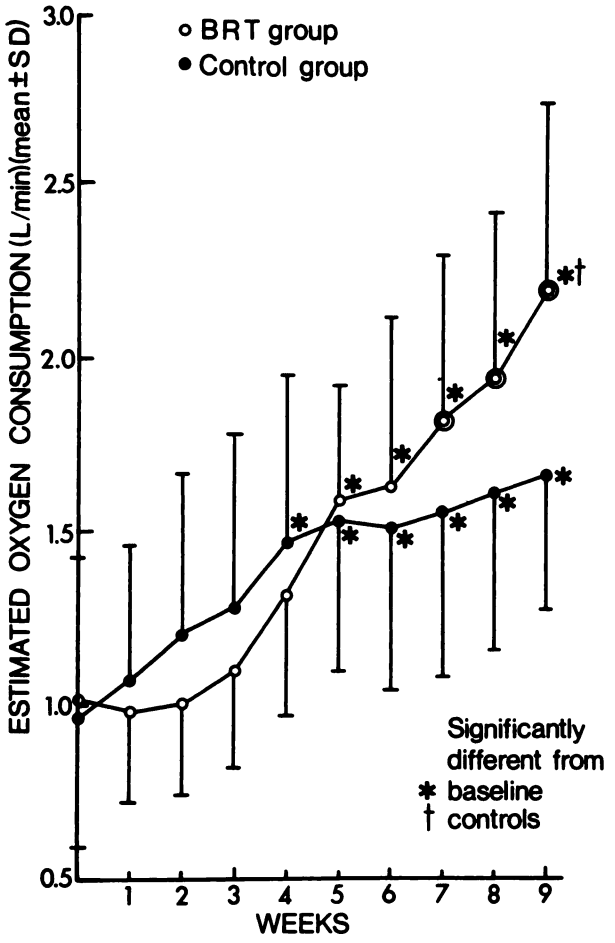

FIGURE 1. Estimated oxygen consumption in breathing retrained and control subjects at each stage of study. Estimated $\dot{V}O_2$ increased significantly in each group; however, the increment in estimated $\dot{V}O_2$ was greater in subjects receiving breathing retraining (weeks of BRT indicated by double circles).

Table 1—*Baseline Pulmonary Function Results* in Subjects With COPD†*

	Group 1	Group 2
FEV_1 (L)	0.66±0.31	0.68±0.25
$FEV_1/FVC\%$	27.4±7.4	29.6±9.9
R_{aw} (cm/L/sec)	6.0±2.3	6.1±2.9
MVV (L/min)	39.4±19.0	40.7±18.7
Vital Capacity (L)	2.6±0.8	2.4±0.9
Residual Volume (L)	5.1±1.1	4.7±1.1
$DLCO_{SB}$ (ml/min/mm Hg)	11.1±3.0	10.9±3.2
SBN_2 (%N_2/L)	11.2±5.3	11.3±4.6

*Results of resting pulmonary function tests after exercise reconditioning and BRT were similar and not significantly different from the data shown in this table. Therefore, for purposes of brevity, only the baseline results are presented.
†R_{aw} = airways resistance, MVV = maximal voluntary ventilation, SBN_2 = single-breath nitrogen washout. For other abbreviations, see text.

Resting Pulmonary Function Tests

Results of routine pulmonary function tests were very abnormal but similar between the two groups. There were no significant improvements in resting pulmonary function in either group of subjects during this study.

Exercise Performance

Increases in work performance were significant in both groups for the entire nine-week program. In group 1, estimated $\dot{V}O_2$ increased from 1.02 ± 0.40 L/min (baseline) to 2.20 ± 0.53 L/min after ER plus BRT ($F = 12.1$, $P < .001$). In group 2, estimated $\dot{V}O_2$ increased from 0.96 ± 0.37 to 1.67 ± L/min after nine weeks of ER ($F = 3.6$, $P < .005$). After completion of the entire nine-week program, estimated $\dot{V}O_2$ was higher in group 1 than in group 2 ($F = 6.5$, $P = 0.02$). In contrast, there were no significant differences between the two groups in estimated $\dot{V}O_2$ at baseline ($P > .7$) or after six weeks of ER ($P > 0.5$).

In both groups, six weeks of exercise was associated with significant improvements in estimated $\dot{V}O_2$ (Fig 1); however, the relative increments in estimated $\dot{V}O_2$ did not differ significantly between the two groups in the first six weeks of the program ($P > .4$). In the last three weeks of the program, the increment in estimated $\dot{V}O_2$ was significantly higher in the BRT subjects (group 1) than in the controls (group 2). ($F = 10.5$, $P < .002$).

Ventilatory Studies

In group 1, the resting respiratory rate decreased from 17.4 ± 5.9 breaths/min (mean ± SD) to 15.0 ±4.0 breaths/min after ER (P = NS) to 9.7±2.5 breaths/min after BRT ($F = 8.2$, $P < .01$). During maximal exercise, the respiratory rate decreased from 32.6 ± 7.5 breaths/min (baseline) to 30.3 ± 9.4 breaths/min after ER (P = NS) to 23.8 ± 5.7 breaths/min after BRT ($F = 3.5$, $P < .05$). Minute ventilation during maximal exercise was 28.1 ± 10.7 L/min at baseline, 27.5 ± 12.8 L/min after ER, and 31.4 ± 11.1 L/min after BRT (P = NS). Tidal volume (V_T) during exercise increased from 800 ml at baseline to 910 ml after ER (P = NS) to 1,320 ml after BRT ($F = 4.0$, $p < .05$). There were no significant changes in resting arterial blood gases during this study. During exercise, however, PaO_2 increased between ER and BRT ($F = 9.5$, $P < .01$) and base excess decreased between baseline and BRT ($F = 10.2$, $P < .01$). After nine weeks of the study, PaO_2 and base excess differed significantly in groups 1 and 2 (Table 2).

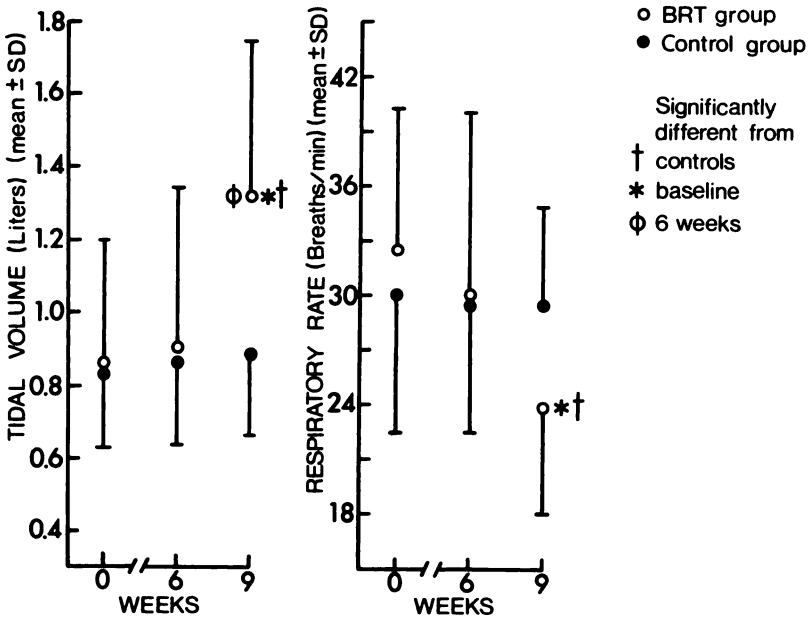

FIGURE 2. Changes in respiratory rate and tidal volume during maximal exercise at start of study and after six and nine weeks. Following breathing retraining, respiratory rate decreases significantly and tidal volume increases significantly.

In group 2 subjects, resting as well as exercising respiratory rates were virtually unchanged during this study. Similarly, changes in minute ventilation, V_T, and arterial blood gases during rest and exercise were not statistically significant (Fig 2, Table 2).

Heart Rate

Resting heart rate did not change significantly in either group during this study. In group 1, heart rate during maximum exercise was 120 ± 16 at baseline, 123 ± 19 after ER and 118 ± 17 after BRT. In group 2, heart rates during maximum exercise were 123 ± 11 at baseline, 125 ±15 after six weeks of ER, and 126 ± 14 after nine weeks of ER. In both groups, these exercising heart rates were not significantly different, even though the levels of work increased throughout the program.

DISCUSSION

This study demonstrated significant improvement in exercise tolerance following a program of somatic reconditioning and BRT. Based on earlier work, the improvement from exercise reconditioning was expected. It is generally accepted that this improvement is due to a combination of factors including: (1) improved neuromuscular coordination and acclimation to walking on the treadmill; (2) improved utilization and distribution of delivered oxygen; and (3) improved effort due to motivational factors.[1,5,6,8] On the other hand, this study also indicated that a program of BRT may significantly improve exercise performance in subjects with COPD. This observation has not been previously reported to our knowledge.

In this study, two groups of subjects with severe COPD were studied. The only difference in their pulmonary rehabilitation programs was that group 1 received BRT whereas group 2 did not. Inspection of the results of this study shows that both groups increased exercise tolerance significantly, although the magnitude of improvement was greater in group 1. Importantly, however, there were no significant differences between the groups in estimated $\dot{V}O_2$ after six weeks of exercise reconditioning. It was only after nine weeks that significant differences were evident (Fig 1). At this time, estimated $\dot{V}O_2$

Table 2—*Arterial Blood Gas Results During Maximum Exercise in Control and Breathing Retrained Subjects (Mean ± SD)*

	Baseline	6 Wk*	9 Wk*
		PaO_2 (mm Hg)	
BRT	69.3±9.0	63.9±11.0	77.5±8.5†‡
Controls	65.3±13.2	62.6±13.3	60.0±11.5
		$PaCO_2$ (mm Hg)	
BRT	39.2±5.3	36.9±6.6	35.2±8.2
Controls	39.8±5.0	39.5±8.0	40.4±7.6
		Base Excess (mEq/L)	
BRT	−1.2±1.9	−3.6±3.3	−5.5±3.8‡‖
Controls	−0.4±3.4	−1.8±3.5	−2.3±3.2
		pH	
BRT	7.39±0.03	7.35±0.04	7.36±0.06
Controls	7.40±0.04	7.38±0.06	7.37±0.08

*At six weeks, both groups had received exercise reconditioning only. At nine weeks, the controls (group 2) had received only exercise reconditioning, whereas the subjects in group 1 had also received breathing retraining.
†Significantly different from results at six weeks.
‡Significantly different from results in controls.
‖Significantly different from baseline results.

was higher ($P = .02$) in the group that received BRT. In addition, the increment in work performance during the final three weeks of the program was also significantly higher in the group that received BRT ($P < .002$). Thus, these data indicate that, compared with controls, exercise performance increased significantly in the group of COPD subjects receiving breathing retraining.

Improvement in exercise tolerance after BRT is difficult to explain. Most previous workers have not found any consistent improvements in resting pulmonary function following a program of pulmonary rehabilitation.[1,5,6,7] Similarly, we did not find any postexercise or post-BRT improvement in resting pulmonary function in our patients. On the other hand, there was some evidence of improved gas exchange after BRT, since PaO_2 during exercise improved significantly in our BRT patients. This improvement in exercise PaO_2 has also been noted by others.[14,26] However, it would seem unlikely that the increases observed in exercise PaO_2 could result in large enough changes in arterial oxygen content to account for the improved work performance after BRT.

Cherniack[29] has shown that there is decreased efficiency of the respiratory muscles and a high oxygen cost of ventilation in patients with emphysema, particularly during exercise. It has been suggested that breathing retraining may decrease the work of breathing by decreasing the respiratory rate and relaxing accessory muscles.[1,15] Our BRT program was designed to cause relaxation of the accessory muscles of respiration. Furthermore, respiratory rate during maximal exercise did decrease significantly ($P < .05$) in our patients following BRT. Thus, a very possible explanation for the improvement in this study after BRT is increased efficiency of the respiratory muscles. The presumed beneficial result of improvement in respiratory muscle efficiency would be a reduction in the oxygen cost of breathing.

Jones has reported in normal persons that increases in tidal volume (V_T) during exercise are associated with a reduction in the ratio of dead space to tidal volume (V_D/V_T).[31] In our COPD subjects, V_T during exercise increased substantially after BRT ($P < .05$). Thus, another possible beneficial effect of BRT might have been reduced ratios of V_D/V_T. Since we did not actually measure V_D/V_T, we cannot comment on this possibility. Certainly, determination of the effects of BRT on the V_D/V_T of exercise would be of interest.

In conclusion, a program of exercise reconditioning and BRT training was associated with a marked improvement in exercise performance in ten subjects with severe COPD. Comparison with data from 12 control subjects with severe COPD indicated that the improvement from this exercise-BRT program was significantly greater than would have been expected from exercise alone. Although the numbers of patients studied were relatively small, our results suggest that breathing retraining may be useful therapy in subjects with exercise limitation from severe COPD.

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