Resistive Breathing Training in Patients with Chronic Obstructive Pulmonary Disease*

Michael J. Belman, M.D., F.C.C.P.; Scott G. Thomas, Ph.D.; and Michael I. Lewis, M.D., F.C.C.P.

In order to investigate the effect of resistive breathing training on ventilatory muscular endurance, we examined the maximal sustained ventilatory capacity in ten patients with chronic obstructive pulmonary disease (COPD) before and after a six-week program of resistive breathing training. In addition, we investigated the effect of altered breathing strategy on resistive breathing performance. The patients performed two 15-minute sessions of resistive breathing daily for six weeks using an inspiratory resistive device (Pflex). Before and after the training, we found no significant change in spirometric data, pulmonary volumes, maximal inspiratory pressure, and maximal expiratory pressure. Of the ten patients, seven failed to show an improvement in their performance of resistive breathing. Furthermore, the maximal sustained ventilatory capacity was unchanged after the resistive breathing training. After the completion of the training program, seven of the patients participated in an additional experiment in which they were instructed to take long slow inspirations while breathing through the resistive device. With this change in breathing pattern, five of the seven were able to improve their performance of resistive breathing. Analysis of the breathing strategy showed that a reduction in the peak mouth pressure, breathing frequency, and external resistive work with a longer inspiratory time was beneficial. We conclude that (1) neither resistive breathing performance nor ventilatory muscular endurance, as measured by sustained hyperpnea, is improved by resistive breathing training performed according to the current instructions with the resistive device, and (2) alterations in breathing strategy have a profound effect on the performance of resistive breathing. The lack of details of breathing strategy in previous studies of resistive breathing makes it difficult to determine if previously demonstrated improvements were due to a real enhancement of ventilatory muscular performance or merely secondary to a different strategy.

Two methods of ventilatory muscular training have emerged.[1] The first is the hyperpneic method, in which patients rebreathe at high minute ventilations for prolonged periods.[2-5] This method has resulted in improved ventilatory muscular endurance as measured by the maximal sustained ventilatory capacity. The second method is the resistive method, in which patients breathe through inspiratory resistances of varying magnitude, usually at normal breathing frequencies.[6-14] Several reports have documented that after resistive training, there is an improved ability to breathe through smaller inspiratory orifices. It is assumed, but not proven, that in these studies the use of a smaller orifice implies a higher inspiratory resistance. Furthermore, based on one of these studies,[8] a resistive breathing device (Pflex) has been developed and is now used by patients with chronic obstructive pulmonary disease (COPD). Recent studies have shown that breathing strategy is an important determinant of resistive breathing endurance. The important variables include inspiratory mouth pressure (Pm), inspiratory time (Ti), duty cycle (Ti/Ttot), breathing frequency, and mean inspiratory flow (VT/Ti).[15-20] Whereas most patients with COPD have improved their ability to breathe through higher resistances after resistive training, it is not clear whether this is due to increased ventilatory muscular endurance or whether it results from a change in breathing strategy. The latter possibility was not evaluated in previous studies in patients with COPD.[6-13] As the instructions for the use of the inspiratory resistive device do not include measures to control for breathing strategy, it is possible that improved performance with the device may result from a change in breathing technique. In the previous studies, ventilatory endurance was measured by means of resistive breathing, and it is not known if current recommendations for resistive training would improve sustained unloaded hyperpnea. The purpose of this study was twofold: to examine in patients with COPD if (1) changes in breathing strategy affected performance during resistive breathing with the resistive device, and (2) if resistive breathing training improved the capacity for eucapnic sustained hyperpnea.

Materials and Methods

The study was approved by the Institutional Review Board of the City of Hope Medical Center, and informed consent was obtained

*From the Division of Respiratory Disease, City of Hope National Medical Center, Duarte, CA.
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Table 1—*Age and Physiologic Data of Patients**

Patient	Age, yr	FVC, L	FEV_1, L	FRC, L	PaO_2, mm Hg	$PaCO_2$, mm Hg
1	64	2.54	1.13	5.50	65	36
2	56	3.26	0.87	7.20	65	30
3	65	2.61	1.00	4.50	62	41
4	57	2.40	1.01	6.31	71	47
5	64	3.39	2.30	2.69	56	44
6	49	2.73	0.88	4.97	72	44
7	74	2.02	0.49	4.09	70	53
8	57	2.34	0.52	6.18	72	37
9	70	3.47	1.70	3.97	62	30
10	75	2.20	0.59	4.69	72	38
Mean	63	2.69	1.03	5.29	67	40
SD	9	0.51	0.57	1.49	6	6

*FVC, Forced vital capacity; and PaO_2, partial pressure of oxygen in arterial blood.

from each participant. The study was carried out in two parts: (1) a resistive training portion, in which the patients were free to choose their own breathing pattern and in which specifically no instructions regarding breathing pattern were given; and (2) after completion of training, the patients were coached in the technique of long, slow deep breathing while using the resistive device.

Patients

Ten patients with COPD were selected according to the following three criteria: (1) presence of COPD as defined by the American Thoracic Society;[21] (2) improvement in forced expiratory volume in one second (FEV_1) of less than 20 percent after inhaled isoproterenol; and (3) free from overt coronary arterial disease, cardiac arrhythmias, congestive heart failure, and orthopedic problems such as shoulder girdle and spinal abnormalities which would interfere with performance of the breathing maneuvers. The patients' characteristics are shown in Table 1.

Before and after the training program, patients underwent measurements of spirometric data, maximum voluntary ventilation (MVV), and pulmonary volumes by standard methods.[22] Pulmonary volumes were measured by the technique of helium dilution.[23] Measurements were also made of the maximal mouth inspiratory pressure (MIP) at functional residual capacity (FRC) and maximal mouth expiratory pressure (MEP) at total lung capacity (TLC).[24] During measurement of the MIP, an 18-gauge needle was placed in the mouthpiece in order to prevent the oral pressure artifacts. Ventilatory muscular endurance was measured as the maximal sustained ventilatory capacity in a rebreathing circuit.[2-4] This system allows continuous monitoring of the patient's ventilatory level and provides a visual target to encourage the patient to perform maximally. Concentrations of oxygen and carbon dioxide are maintained within physiologic limits. The initial target was set at the level of 90 percent of the MVV and adjusted to encourage maximal performance. Mean ventilatory levels were calculated each minute, and the mean of the latter eight minutes was defined as the maximal sustained ventilatory capacity. Each patient performed a practice maneuver for maximal sustained ventilatory capacity before the baseline test in order to overcome the small learning effect.[4]

Resistive breathing was performed through an inspiratory resistive device (Pflex). This is a plastic tube with six variable inspiratory resistances ranging in diameter from 0.54 cm (orifice 1) to 0.17 cm (orifice 6). The device is fitted with a one-way valve so that expiration is unimpeded. Our experimental setup is shown in Figure 1. The patients breathed through a one-way valve (Hans Rudolph 1400). Between this and the resistance, a pneumotachometer (Fleish No. 3) was placed to measure airflow. Volume was derived from flow using an integrator (Electronics for Medicine). Mouth pressure was recorded continuously via a differential transducer (Validyne; ±140 cm H_2O) connected to an orifice in the mouthpiece of the inspiratory resistive device. Flow, volume, and pressure signals were transmitted to a computer (DEC PDP 11/34) and used for the determination of tidal volume (V_T), frequency of breathing, inspired minute ventilation ($\dot{V}_I$), peak inspiratory flow rate, Pm, Ti, the ratio of inspiratory to total breath time (Ti/Ttot), and (V_T/Ti). In addition, the computer continuously integrated the product of Pm and volume during each minute to calculate the inspiratory external resistive work done per minute by the patient while breathing through the resistive device.

The critical orifice, defined as the smallest orifice through which each patient was able to breathe continuously for 15 minutes, was evaluated at entry into the study, after training, and after coaching. In determining the critical orifice, all patients wore nose clips and began resistive breathing through the largest orifice (orifice 1). If successful, they progressed to smaller orifices on successive days. During this testing and during training, patients were free to choose their own breathing pattern, and no instructions were given. The failure point was determined by the patient's inability to continue. After determination of the critical orifice, patients were instructed to perform two daily training sessions for 15 minutes. Training was done, while wearing nose clips, at the orifice just smaller than the critical orifice. If patients were unable to complete 15 minutes at that orifice, they were instructed to return to the critical orifice to complete the full training session. The total training time was six weeks. The patients were contacted weekly and evaluated after three weeks of training to monitor their progress. Patients who missed more than three successive training sessions or who did not maintain at least an overall 80 percent compliance with the training protocol were withdrawn from the study.

Coached Breathing

After completion of the tests after training, seven of the patients (patients 4 through 10) underwent additional testing at one orifice

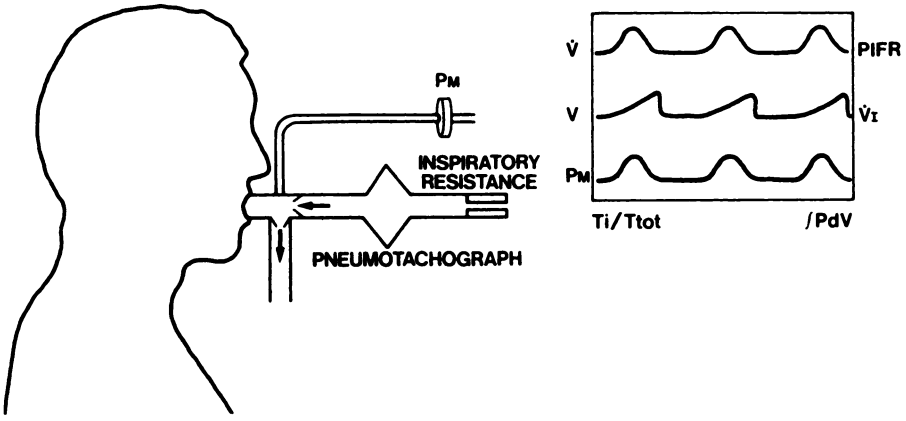

FIGURE 1. Experimental setup for measurement of breathing strategy during resistive breathing (see text for explanation).

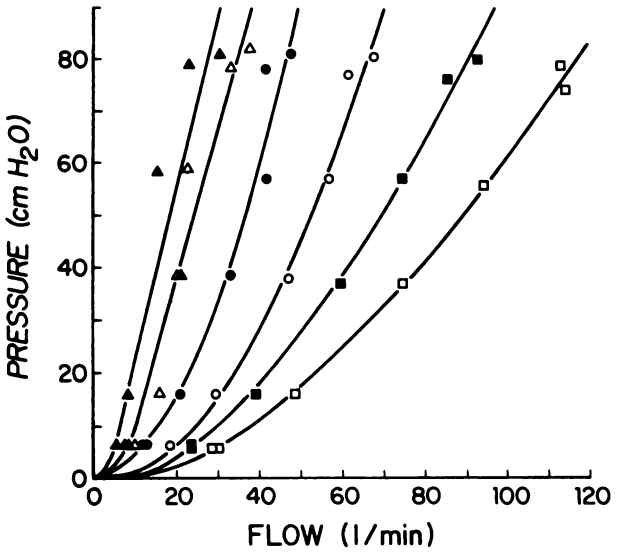

FIGURE 2. Flow characteristics of resistive training device. Each regression line represents pressure required to produce flow at each of six orifices. By decreasing flow, it is possible to maintain pressure relatively low, even though smaller orifice is used. Regression lines from left to right represent orifices 6 (diameter, 0.17 cm) through 1 (diameter, 0.54 cm), respectively.

smaller than the critical orifice. During these tests, they were continually coached to breathe using long slow inspirations.

Pressure-Flow Characteristics of Device

The flow-pressure curve for each orifice of the resistive device was determined. A negative pressure ventilator (Monaghan 170C) was used to pull air through the resistive device at a variety of measured flow rates, and pressure across the device was measured. From these measurements the resistance at different flows was calculated for each orifice.

Statistical analyses were performed by means of the paired *t*-test to compare the values before and after resistive training and the uncoached and coached indices of breathing strategy. The change in critical orifice before and after training was measured by the signed rank test.[25]

RESULTS

Flow Characteristics of the Device

With increasing flow, there is an increase in the pressure across the resistive device (Fig 2). Of importance is the fact that a lower flow reduced the pressure differential observed for a small orifice to equal or below the value attained with a larger orifice.

Table 2—*Pulmonary Function Before and After Resistive Training

Data	Before	After
FVC, L	2.69± 0.51	2.47± 0.66
FEV_1, L	1.03± 0.57	0.97± 0.63
MVV (4 min)	47 ±25	43 ±22
FRC, L	5.29± 1.49	5.45± 1.23
TLC, L	7.29± 1.27	6.96± 0.82

*Values are means ± SD (n = 10).

Table 3—*Critical Orifice Before and After Training*

Patient	Orifice No. Before	Orifice No. After
1	3	5
2	1	3
3	4	4
4	3	3
5	2	2
6	3	3
7	3	3
8	3	3
9	4	5
10	3	3

Resistive Training and Ventilatory Function

Ten of 15 recruited patients completed the study. Of the five patients who failed to complete the study, two dropped out because of intercurrent illness, and three were eliminated because of lack of compliance with the training protocol. The patients in the study had moderate to severe obstruction of the airways, with marked hyperinflation. Only two were hypercapnic (arterial carbon dioxide tension [$PaCO_2$]>45 mm Hg) (Table 1). The data on pulmonary function before and after training are shown in Table 2. Measures of airflow limitation and pulmonary volumes were similar before and after training. Table 3 shows the critical orifice before and after resistive training. Only three of the ten patients (patients 1, 2, and 9) showed an increase in the critical orifice, and overall this change was not significant. Figures 3 and 4 show the maximal sustained ventilatory capacity and respiratory pressures in the patients with COPD. These values were not significantly different before and after training.

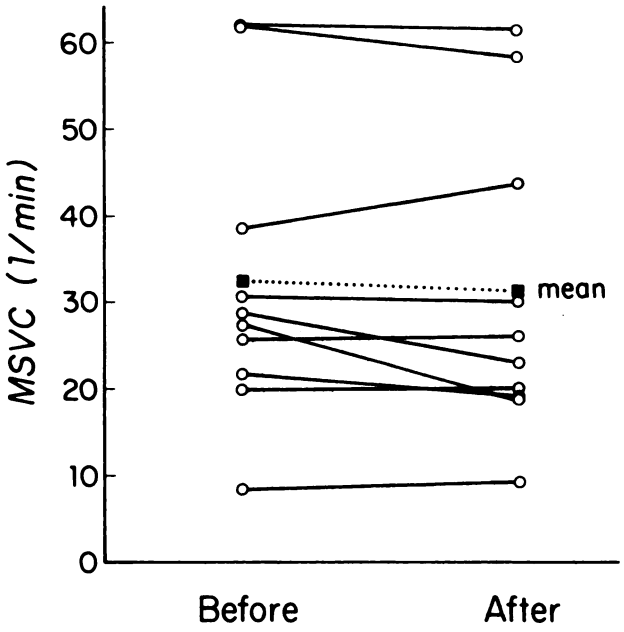

FIGURE 3. Maximal sustained ventilatory capacity (MSVC) before and after training. Values before and after are similar, and mean is not significantly different (32.5 ± 17.4 L/min to 30.4 ± 20.1 L/min).

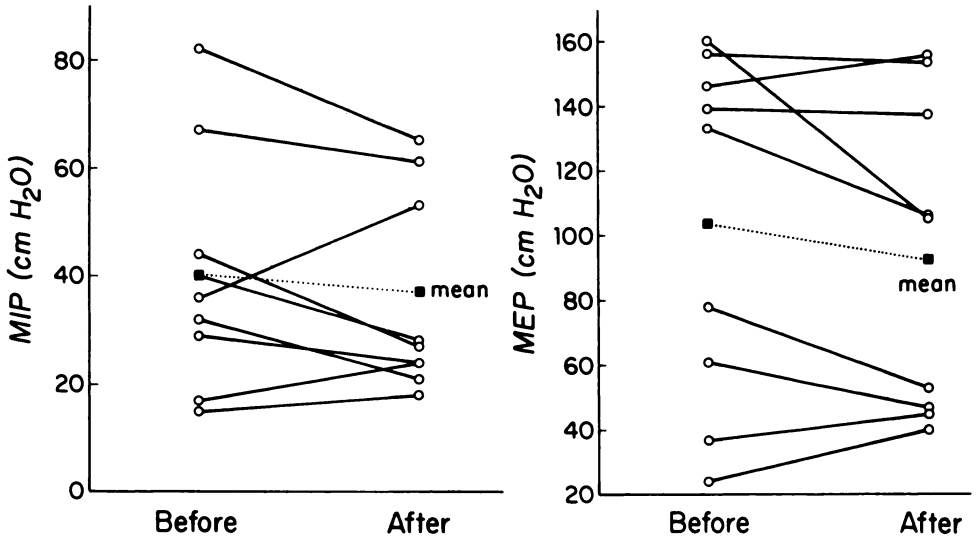

FIGURE 4. Maximal inspiratory pressure (MIP) (40.2 ± 23.0 cm H_2O to 35.6 ± 19.6 cm H_2O) and maximal expiratory pressure (MEP) (103.8 ± 56.6 cm H_2O to 93.4 ± 50.6 cm H_2O) before and after training (n = 9). These values were not significantly different.

Effect of Coached Breathing

The endurance time for resistive breathing increased significantly when the patients were coached (Fig 5). In Figure 5, we show the data of seven patients (patients 4 through 10) in whom complete analysis of the breathing pattern was possible. Five of the seven patients shown in Figure 5 increased their endurance for resistive breathing through one orifice smaller than the critical orifice to the full 15 minutes. Table 4 shows several indices of breathing strategy at the critical orifice and at one orifice smaller in these five patients. No significant difference was seen in the external resistive work, Pm, Ti, frequency of breathing, VT, and Ti/Ttot while breathing through the critical orifice before and after resistive training. While breathing uncoached through the orifice one smaller than the critical orifice, there was a significant increase in the external resistive work, Pm, and the Ti/Ttot in comparison to the critical orifice before training. In comparing the data between the orifice one smaller than the critical orifice uncoached and coached, there was in the coached a significant decrease in the external resistive work, Pm, and breathing frequency and a significant increase in the Ti. The VT was larger, but this change was not significant. The average external resistive work per breath in these five patients was unchanged (9.5 cm H_2O/L uncoached and 9.8 cm H_2O/L coached), but the work rate per minute when coached was lower due to a decrease in breathing frequency. A typical change in breathing strategy at the orifice one smaller than the critical orifice with coaching in one of the patients is shown in Figure 6. This figure shows a reduced Pm, breathing frequency, and peak inspiratory flow rate with a larger VT and Ti.

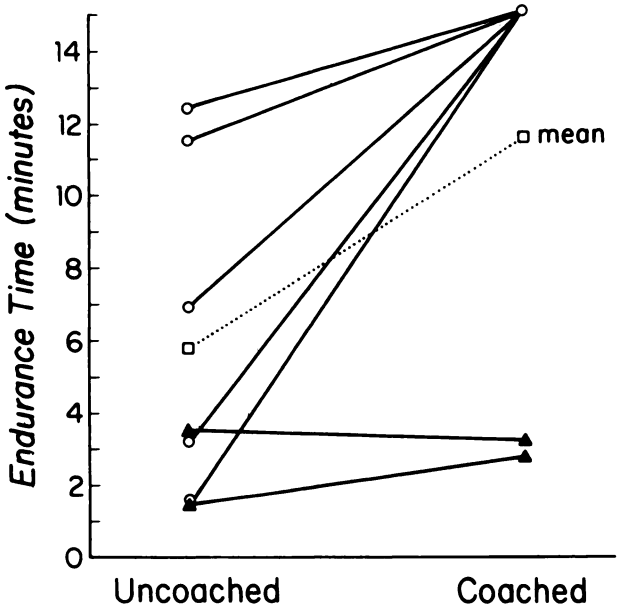

FIGURE 5. Endurance time for seven patients while breathing through orifice one smaller than critical orifice in uncoached and coached manner. There was significant increase in endurance time (5.78 ± 4.59 minutes to 11.56 ± 5.86 minutes) ($p<0.05$). Five patients who improved their endurance time to 15 minutes are shown as *open circles*. Two patients who did not improve are shown as *closed triangles*.

DISCUSSION

We have shown that resistive breathing training did not improve the ability to breathe through higher resistances. Furthermore, we found no improvement in ventilatory muscular endurance as measured by the maximal sustained ventilatory capacity and no change in maximal respiratory pressures; however, we did find that an altered pattern of breathing with a longer Ti but lower Pm, breathing frequency, and external resistive work was associated with an improved ability to inspire through smaller orifices.

Table 4—*Comparison of Breathing Strategy Between Critical Orifice (CO) Before and After Training**

Data	CO		CO+1	
	Before Training	After Training	Uncoached	Coached
Endurance, min	15 ± 0	15 ± 0	7.10± 5.73†	15 ± 0‖
ERW, cm H_2O/L/min	65.9 ±57.9	83.7 ±75.1	127.5 ±113.7†	93.1 ±78.9§
Pm, cm H_2O	11.03± 6.0	13.3 ± 7.8	19.4 ± 10.6‡	15.2 ± 8.2§
Ti, sec	2.21± 0.38	1.98± 0.36	2.60± 0.84	3.62± 0.50‖
Breathing frequency, breaths per min	12.6 ± 2.68	13.9 ± 1.6	13.4 ± 4.5	9.4 ± 0.8§
Vt, L	0.59± 0.09	0.56± 0.16	0.62± 0.08	0.80± 0.21
Ti/Ttot, percent	45 ± 4	46 ± 4	55 ± 5‡	57 ± 8

*N = 5. These five patients failed to improve with training but improved with coaching. Values are means ± SD. CO + 1, Orifice one smaller than critical orifice; and ERW, external resistive work. †$p<0.05$ comparison between CO pretraining and CO + 1 uncoached. ‡$p<0.01$ vs CO + 1 uncoached. §$p<0.05$, ‖$p<0.01$ comparison between CO + 1 uncoached and coached.

The majority of our patients failed to improve resistive breathing performance after training. This is in contrast to most previous investigators, who showed that overall resistive breathing training improves the ability to breathe through inspiratory resistances in patients with COPD.[6-13] In the previous studies of resistive training, the duration and frequency of the training was similar to our study, varying between four and six weeks with two to three 15-minute sessions per day, respectively; however, it should be noted that in one study a control group[7] which used a sham treatment also improved their resistive breathing performance, although in two other studies[8,9] the control group showed no change. Several factors may play a role in explaining the differing results, and these include (1) alteration in breathing strategy, (2) an inadequate training stimulus, and (3) an inadequate recovery from fatigue between tests.

Alteration in Breathing Strategy

The ability to perform resistive breathing is strongly related to breathing strategy. This has been expressed by Bellemare and Grassino[15] as the tension time index, which is the product of the transdiaphragmatic pressure (Pdi)/Pdi max ratio and the duty cycle (inspiratory time/total breathing time [Ti/Ttot]). When this ratio is less than 0.15, both normal subjects and patients were able to perform resistive breathing without the development of fatigue.[15,16] For tension time indices greater than 0.15, fatigue developed, and there was an inverse

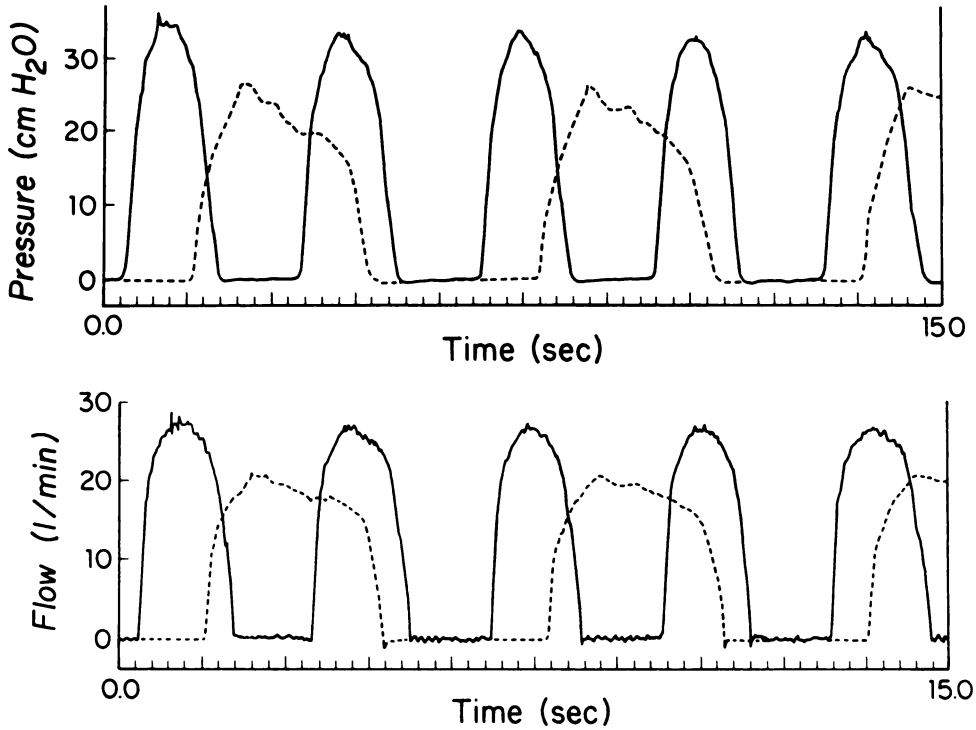

FIGURE 6. Fifteen-second records of pressure *(top)* and flow *(bottom)* patterns, respectively, in one patient while breathing through orifice one smaller than cricital orifice. Uncoached and coached tracings are superimposed, with *dotted line* representing coached breathing. From tracings, it can be seen that with coaching, there is reduced Pm, peak inspiratory flow rate, and breathing frequency, with increase in Ti and Vt.

relationship between the level of the tension time index and the endurance time. More recently, Collett and co-workers[17] have shown that there is a linear relationship between the oxygen cost of breathing ($\dot{V}O_2$ resp) and the work rate across external resistances and that the latter is a function of the tension time index and the mean inspiratory flow rate. The importance of this work is that it shows that the tension time index only describes the $\dot{V}O_2$ resp when inspiratory flow rates are constrained. For increasing inspiratory flow rates, there is an increase in the $\dot{V}O_2$ resp and the work rate even when the tension time index is constant.[17] More recently, endurance of the inspiratory muscles has been shown to vary inversely with the inspiratory flow rate even for the same tension time indices.[18,19] The work of Jones et al[20] confirmed the relationship between pressure time indices and the increase in the oxygen cost of external work. Furthermore, these investigations showed that the rating of perceived effort (RPE), as measured by a Borg scale during resistive breathing, was strongly related to the Pm, Ti, and breathing frequency (fb). This relationship was similar during both a freely adopted and constrained breathing pattern and was described by the equation, $RPE = Pm^{1.34} \times Ti^{0.52} \times fb^{0.26}$.

We did not specifically measure perceived effort in our study, but we noted that an improved endurance at the orifice one smaller than the critical orifice was associated with the breathing pattern which would reduce the RPE as defined by the previous relationship. Because the RPE is affected principally by the Pm, the fall in Pm would outweigh the increase in Ti observed when patients were coached (Fig 6). The five patients who increased their resistive breathing time showed a decrease in the calculated RPE, while the two patients who failed to increase their endurance also failed to reduce the calculated RPE (Fig 7); however, our study was not designed to investigate the relative importance of RPE, $\dot{V}O_2$ resp, and external resistive work in determining resistive breathing performance, and any of these variables singly or in combination could have played a role in improving resistive breathing performance with coaching.

Despite the well-known relationship between resistive breathing endurance and breathing strategy, there are very few data on these variables in the previous studies in COPD. The Pm and pattern of breathing was not monitored during training, and only in a few[6,8,9] was the Pm recorded during the testing of resistive breathing, although it is not clear if the signal was displayed to the patients. One group of investigators who noted that their patients tended to take longer, slower breaths actually suggested that the alteration in breathing pattern may have been responsible for the improvement but did not make measurements of the various patterns.[8] In another study[9] the investigators encouraged their patients to use a pattern of long slow breaths, but again no measurements of strategy were made. It was only in the study of Clanton and coworkers[14] that breathing strategy was recorded during both testing and training, but this study dealt with young normal women. Without this information, it is not possible to judge if the previously described improvements in resistive breathing in patients with COPD were due to real increases in ventilatory muscular function or were secondary to changes in breathing pattern.

Inadequate Training Stimulus

There is a vast body of information which deals with the appropriate intensity, duration, and frequency of training necessary to induce the classic training responses for whole-body exercise.[26] This information is as yet not available for ventilatory muscle training, and in fact the recommended methods of training differ greatly in their strategies. In the resistive form, breathing frequency is generally within the normal range, whereas the respiratory pressures are increased by breathing through the resistors.

Whereas most authors have used two to three 15-minute sessions per day for periods varying from four to six weeks, this does not appear to be essential to elicit a training response. In the study by Clanton et al[14] in normal subjects, the total training time was only 7½ minutes daily (25 percent of the total time performed by patients with COPD); however, the inten-

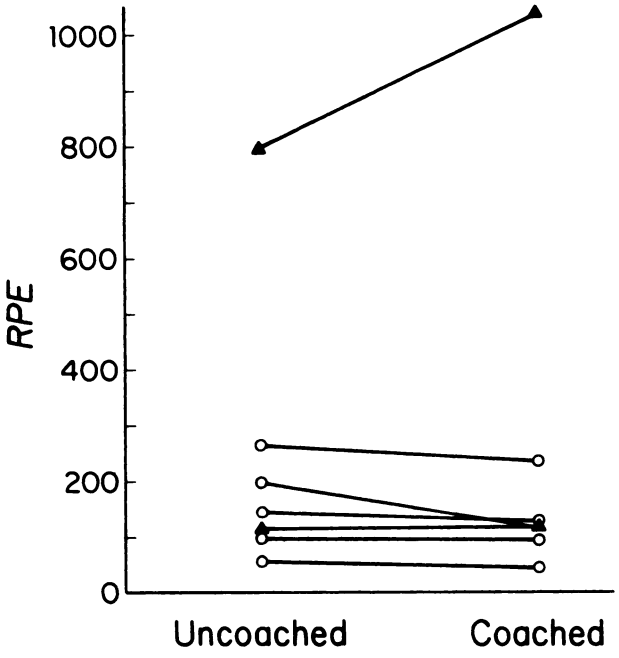

FIGURE 7. Rating of perceived effort (RPE) calculated from equation $(Pm^{1.3} \times Ti^{0.52} \times fb^{0.26})^{20}$ for five patients who increased with endurance time to 15 minutes; RPE before and after training was 152 ± 36 and 123 ± 13 ($p = 0.054$), respectively. Two patients who failed to increase their endurance time had the same or higher values for RPE *(solid triangles)*.

sity was considerably higher with the subjects aiming for maximal mouth pressure with each inspiration, and the duty cycle was controlled. In this study, there was improvement in both strength, as measured by the MIP, and endurance, as measured by the ability to breathe while following pressure and flow target. If training for such short duration is efficacious in patients with COPD, this would facilitate the use of resistive training, but the appropriate proportions of intensity and duration remain to be determined. Work from studies in animals suggests that alteration of these factors determines the pattern of oxidative enzyme enhancement.[27] High-intensity short-duration work predominantly affects the high-glycolytic low-oxidative fibers, whereas low-intensity longer-duration exercise predominantly affects high-oxidative low-glycolytic muscle fibers. Furthermore, in humans, it has been shown that a program of combined strength and endurance training will have the same effect as endurance training alone, but improvement in muscle strength is less than that achieved by a strength-only training program.[28]

Improvement in maximal strength of the respiratory muscle is important because the RPE during resistive breathing is a function of the ratio of the Pm developed to the MIP.[20] Previous studies in COPD have shown varying results with regard to MIP. Patients with COPD have generally not shown increases in this index,[8,13] although increases were found in young children with cystic fibrosis[9] and in the normal young women studied by Clanton and associates.[14] In previous studies and our study, the breathing strategy may have been inadequate to elicit a true training response. This may be the case *de novo*, or changes may develop when patients are presented with smaller orifices. By means of appropriate alterations in breathing strategy when confronted with a smaller orifice, the increase in Pm can be minimized. This change may be self-defeating, as it may negate the training stimulus.

Several of the studies which have used resistive breathing have also tested the response to overall exercise as the measure of the efficacy of inspiratory muscular training; however, only a minority of patients in these studies have shown improved exercise capacity, although they improved their inspiratory muscle endurance for resistive breathing.[6-13] Because the hyperpnea of exercise is associated with volume overload, increased breathing frequency, and a decreased Ti or increased velocity of contraction, it may be that hyperpneic training is the more suitable approach to improve exercise performance in patients with COPD.[4] In this study, we found that the maximal sustained ventilatory capacity, a measure of endurance for hyperpnea, did not increase after the resistive training; however, because the patients failed to improve the resistive breathing, we cannot rule out the possibility that the lack of change in hyperpneic endurance was due to an inadequate training stimulus, rather than an inappropriate form of training. This question would need to be examined in patients who showed a definite improvement in resistive breathing performance.

Inadequate Rest Time

Resistive breathing produces "low-frequency fatigue" of the diaphragm and sternomastoid muscles,[29] and recently it was shown[30] that there is a reduction in muscle endurance in the presence of low-frequency fatigue. As recovery from low-frequency fatigue may take several hours,[30] it is possible that the repetition of successive testing runs within a short time prevents optimal performance because of the cumulative effect of fatigue. In our study, there was at least a 24-hour rest between tests, a sufficient time to allow complete recovery of low-frequency fatigue.[30] In several of the previous studies,[6-13] the tests of resistive breathing performance were all done on the same day, with only short rests (20 to 30 minutes) between runs. As sufficient recovery time was not available, the baseline critical orifice may have been underestimated, and this would give an erroneous impression of improvement. In a study in which the pulmonary function of the patient was comparable to ours,[10] the critical orifice before training was larger (0.48 cm vs 0.40 cm).

In our study, determination of the critical orifice was done based on the patient's subjective response to the resistive breathing. The majority of other studies in COPD also used subjective end points for determination of the critical orifice.[7,9-11,13] While hypercapnea is a potential problem during resistive breathing, it only occurred in a minority of cases at the time of failure at the critical orifice in the study by Asher et al,[9] and it is probably not a major cause of endurance failure. Simple objective measures, such as abdominal paradox or respiratory alternans, have been used,[7,9] but these also are not invariably present. Power spectral analysis of the electromyogram recorded from the diaphragm and accessory muscle has been used.[4,6,7] In most studies, spectral changes have been examined by means of the H/L ratio. Whether or not this will prove to be an effective means of detecting fatigue remains controversial. Not all patients show evidence of electromyographic fatigue despite failure at a critical orifice, and in some patients, fatigue was only detectable from scalene or intercostal muscles and not from the diaphragm.[8,9] Furthermore, at the end of a fatiguing run, the H/L ratio rapidly returns to baseline values, even though low-frequency fatigue persists for several hours.[31] The initial studies of high/low ratio were done with strict control of breathing strategy.[15,16] Whether or not results from these studies are applicable to spontaneously breathing patients with COPD is as yet unclear, especially as the H/L ratio was used in the

resistive training studies without control of the breathing pattern.[8,9] Furthermore, there is also some doubt that the high/low ratio is the best index to follow power spectral changes, and it has been suggested that measurement of the centroid frequency is more specific.[32] In this study the investigators showed decreases in the centroid frequency without changes in the high/low ratio, even though patients were performing sustainable eucapnic hyperpnea. For future studies, therefore, it would seem appropriate to adopt the approach used by some investigators[14-16] who have controlled breathing strategy during the testing and have used objective measures, such as an inability to achieve target pressures or inspiratory flow rates, as a sign of failure.

Our patients with COPD performed resistive training as suggested by the manufacturers of the inspiratory resistive device and in a manner similar to that described in several of the previous studies. Despite this, our patients failed to improve ventilatory muscle endurance or strength. We believe that in order for resistive breathing to be successful, a feedback signal of the resistive load during training is essential. By this means, both the physician and the patient would be able to regulate the training intensity and ensure a satisfactory training stimulus. Furthermore, it would prevent the patient from adopting a breathing strategy which could improve resistive breathing performance without providing direct benefits to the respiratory muscles.

References

1 Belman MJ, Sieck GC. The ventilatory muscles: fatigue, endurance and training. Chest 1982; 82:761-66

2 Leith DE, Bradley M. Ventilatory muscle strength and endurance training. J Appl Physiol 1976; 41:508-16

3 Keens TG, Krastins IRB, Wannamaker EM, Levison H, Crozier H, Bryan AC. Ventilatory muscles endurance training in normal subjects and in patients with cystic fibrosis. Am Rev Respir Dis 1977; 116:853-60

4 Belman MJ, Mittman C. Ventilatory muscles training improves exercise capacity in chronic obstructive pulmonary disease. Am Rev Respir Dis 1980; 121:273-80

5 Levine S, Weiser P, Gillen J. Evaluation of a ventilatory muscle training program in the rehabilitation of patients with chronic obstructive pulmonary disease. Am Rev Respir Dis 1986; 133:400-06

6 Andersen JB, Dragsted L, Kann NT, Johansen SH. Resistive breathing training in severe chronic obstructive pulmonary disease. Eur J Respir Dis 1979; 61:151-56

7 Bjerre-Jepsen K, Secher NH, Kok-Jensen A. Inspiratory resistance training in severe chronic obstructive pulmonary disease. Eur J Respir Dis 1981; 62:405-11

8 Pardy RL, Rivington RN, Despas PJ, Macklem PT. The effect of inspiratory training on exercise performance in chronic airflow limitation. Am Rev Respir Dis 1981; 123:426-34

9 Asher MI, Pardy RL, Coates AL, Thomas E, Macklem PT. The effect of inspiratory muscle training in patients with cystic fibrosis. Am Rev Respir Dis 1982; 126:855-59

10 Sonne LJ, Davis JA. Increased exercise performance in patients with severe COPD following inspiratory resistive training. Chest 1982; 81:436-39

11 Chen H, Dukes R, Martin BJ. Inspiratory muscle training in patients with chronic obstructive pulmonary disease. Am Rev Respir Dis 1985; 131:251-55

12 Jones DT, Thomson RJ, Sears MR. Physical exercise and resistive breathing in severe chronic airways obstruction: are they effective? Eur J Respir Dis 1985; 67:159-66

13 Madsen F, Secher NH, Kay L, Kok-Jensen A, Rube N. Inspiratory resistance versus general physical training in patients with chronic obstructive pulmonary disease. Eur J Respir Dis 1985; 67:167-76

14 Clanton TL, Dixon G, Drake J, Gadek JE. Inspiratory muscle conditioning using a threshold device. Chest 1985; 87:62-6

15 Bellemare F, Grassino A. Effect of pressure and timing of contraction on human diaphragmatic fatigue. J Appl Physiol 1982; 53:1190-95

16 Bellemare F, Grassino A. Evaluation of human diaphragmatic fatigue. J Appl Physiol 1982; 53:1196-206

17 Collett W, Perry C, Engel LA. Pressure time product flow and oxygen cost of resistive breathing in humans. J Appl Physiol 1985; 58:1263-72

18 McCool FD, McCann DR, Leith DE, Hoppin FG, Jr. Pressure-flow effects on endurance of inspiratory muscles. J Appl Physiol 1986; 60:299-303

19 Clanton TL, Dixon GF, Drake J, Gadek JE. Effects of breathing pattern on inspiratory muscle endurance in humans. J Appl Physiol 1985; 59:1834-41

20 Jones JL, Killian KJ, Summers E, Jones NL. Inspiratory muscles forces and endurance in maximum resistive loading. J Appl Physiol 1985; 58:1608-21

21 ATS. Chronic bronchitis, asthma and pulmonary emphysema: a statement by the committee on diagnostic standards for nontuberculosis disease: respiratory disease definitions and classifications. Am Rev Respir Dis 1962; 85:762-63

22 ATS statements. SnowBird workshop and standardization of spirometry. Am Rev Respir Dis 1979; 119:831-38

23 Black LF, Hyatt RE. Maximal respiratory pressures: normal values and relationships to age and sex. Am Rev Respir Dis 1969; 99:696-702

24 Boren HG, Kory RC, Syner JC. The Veterans Administrations-Army cooperative study of pulmonary function: 2. the lung volume and its subdivisions in normal men. Am J Med 1966; 41:96-114

25 Colton T. Statistics in medicine. Boston: Little, Brown, 1974

26 American College of Sports Medicine. Recommended quantity and quality of exercise for developing and maintaining fitness in healthy adults. Med Sci Sports 1978; 10:7-10

27 Dudley GA, Abraham WM, Terjung RL. Influence of exercise intensity and duration: biochemical adaptations in skeletal muscle. J Appl Physiol 1982; 53:844-50

28 Dudley GA, Djamil R. Incompatibility of endurance and strength training modes of exercise. J Appl Physiol 1985; 59:1446-51

29 Moxham J, Wiles CM, Newham D, Edwards RHT. Sternomastoid muscle function and fatigue in man. Clin Sci 1980; 59:463-68

30 Efthimiou J, Belman MJ, Holman RAE, Edwards RHT, Spiro SG. The effect of low frequency fatigue on endurance exercise in the sternomastoid muscle of normal humans. Am Rev Respir Dis 1986; 133:667-71

31 Moxham J, Edwards RHT, Aubier M, De Troyer A, Farkas G, Macklem PT, et al. Changes in EMG power spectrum (high to low ratio) with force fatigue in humans. J Appl Physiol 1982; 53:1094-99

32 Sieck GC, Mazar A, Belman MJ. Changes in diaphragmatic EMG spectra during hyperpneic loads. Respir Physiol 1985; 61:137-52