Inspiratory Muscle Training in Patients with Bronchial Asthma*

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In patients with asthma, the respiratory muscles have to overcome the increased resistance while they become progressively disadvantaged by hyperinflation. We hypothesized that increasing respiratory muscle strength and endurance with specific inspiratory muscle training (SIMT) would result in improvement in asthma symptoms in patients with asthma. Thirty patients with moderate to severe asthma were recruited into 2 groups; 15 patients received SIMT (group A) and 15 patients were assigned to the control group (group B) and got sham training in a double-blind group-comparative trial. The training was performed using a threshold inspiratory muscle trainer. Subjects of both groups trained five times a week, each session consisted of ½-h training, for six months. Inspiratory muscle strength, as expressed by the PImax at RV, increased significantly, from 84.0±4.3 to 107.0±4.8 cm H_2O ($p<0.0001$) and the respiratory muscle endurance, as expressed by the relationship between P_{mu}peak and PImax from 67.5±3.1 percent to 93.1±1.2 percent ($p<0.0001$), in patients of group A, but not in patients of group B. This improvement was associated with significant improvements compared with baseline for asthma symptoms (nighttime asthma, $p<0.05$; morning tightness, $p<0.05$; daytime asthma, $p<0.01$; cough, $p<0.005$), inhaled B_2 usage ($p<0.05$), and the number of hospital ($p<0.05$) and sick-leave ($p<0.05$) days due to asthma. Five patients were able to stop taking oral/IM corticosteroids while on training and one in the placebo group. We conclude that SIMT, for six months, improves the inspiratory muscle strength and endurance, and results in improvement in asthma symptoms, hospitalizations for asthma, emergency department contact, absence from school or work, and medication consumption in patients with asthma.

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P_{mu}peak = peak pressure; SIMT = specific inspiratory muscle training; TIMT = threshold inspiratory muscle trainer

The mechanism underlying dyspnea suggests that dyspnea, at least in part, results from a perception of respiratory muscle effort.[1,2] Patients with asthma are exposed to increased resistance of their airways and to hyperinflation. The hyperinflation of the lung flattens the diaphragm, shortens the inspiratory muscles, and places them at a mechanical disadvantage.[3] In addition to the reduced efficiency of the inspiratory muscles, large amounts of pressure work are required to overcome the high airway resistance.[4] In a previous study performed by us,[5] it was suggested that hyperinflation adversely affects the performance of the inspiratory muscles in patients with asthma.

It has been shown previously that the inspiratory muscles can be trained for both strength and endurance in normal subjects,[6] quadriplegics,[7] patients with cystic fibrosis, as well as in patients with COPD.[8]

We hypothesized that increasing respiratory muscle strength and endurance with specific inspiratory muscle training (SIMT) will result in improvement in asthma symptoms in patients with asthma.

This study was designed to compare the effects of SIMT with sham training on inspiratory muscle strength, and endurance, asthma symptoms, hospitalizations for asthma, emergency department contact, absence from school or work, and inhaled β_2-agonist consumption in a population of adult bronchial asthmatic patients.

Methods

Thirty patients, 12 men and 18 women, with moderate to severe asthma, who satisfied the criteria of the American Thoracic Society for asthma,[9] were recruited into 2 groups; 15 patients received SIMT (group A) and 15 patients were assigned to the control group (group B) and got sham training in a double-blind group-comparative trial (Table 1).

Patients used daily diary cards during the three months before entering the training program and throughout the last three months of the training to record hospitalizations for asthma, emergency department contact, absence from school or work, and inhaled β_2-agonist consumption, and during the last two weeks of each time period to record the severity of asthma symptoms, as follows: (a) nighttime asthma, recorded each morning, on a scale of 0, no asthma; 1, slightly wheezy; 2, awoke once because of asthma; 3, awoke several times because of asthma; 4, awake most of night because of asthma; (2) daytime asthma, recorded each evening on a scale of 0, no asthma; 1, occasional wheezing or breathlessness; 2, frequent wheezing or breathlessness; 3, wheezing or breathlessness for most of the day that interfered with normal activities; 4, breathlessness so bad that it prevented the patient attending work or school; and (3) cough recorded each evening on a scale of 0, no cough; 1, occasional cough; 2, frequent coughing but with no interference with normal activities; 3, frequent coughing that interfered with normal activities; 4, cough so bad preventing normal activities.

Tests

All tests were performed before, every two months during the training period, and after six months of training.

Spirometry: The forced vital capacity (FVC) and the forced

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expiratory volume in 1 s (FEV_1) were measured three times on a computerized spirometer (Compact, Vitalograph, Buckingham, England) and the best trial is reported. Lung functions were measured before and following the training period.

Respiratory Muscle Strength: Respiratory muscle strength was assessed by measuring the maximal inspiratory mouth pressure (PImax) and expiratory pressure (PEmax), at residual volume (RV) and total lung capacity (TLC), respectively, as previously described by Black and Hyatt.[10] The value obtained from the best of at least three efforts was used.

Respiratory Muscle Endurance: To determine inspiratory muscle endurance, a device similar to that proposed by Nickerson and Keens[11] was used. Subjects inspired through a two-way valve (Hans-Rudolph) whose inspiratory port was connected to a chamber and plunger to which weights could be added externally. Inspiratory work was then increased by the progressive addition of 25- to 100-g weights at 2-min intervals, as was previously described by Martyn and coworkers,[12] 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 ($P_m$peak).

The technicians who performed the tests were totally blinded to the mode of training the patients received.

Training Protocol: Subjects of both groups trained five times a week; each session consisted of ½-h training for six months. The training was performed under the supervision of a physiotherapist, and once a week had an interview with the physician. Both groups received the same attention and adjustment in medications and were treated equally during the training period.

In the SIMT group, subjects started to train with a resistance equal to 15 percent of their PImax and the resistance was then increased incrementally to 60 percent of their PImax, through the first month. SIMT was then continued at 60 percent of the PImax. The level of load has been adjusted every two months according to the new measurements of the PImax achieved by the patients. For the last two months of the study, the patients trained in a level of resistance equal to 80 percent of their PImax. Patients in group B breathed through the same trainer with no resistance. The subjects received either SIMT or a sham training with a threshold inspiratory muscle trainer (TIMT) (Threshold Inspiratory Muscle Trainer, Healthscan, New Jersey).

Patients in both groups were highly motivated and highly compliant with the training. Even the control patients continued using the sham training to the end of the study. However, most of the patients in the control group became gradually aware of the fact that they were using a sham device, but there was no interaction among the subjects in each group.

Statistical Analysis

Comparisons of lung function and respiratory muscle performance values between the training group and the control group and the effect of training on these parameters were carried out using the two-way repeated measures analysis of variance (ANOVA). When the overall ANOVA was significant, *post hoc* comparisons have been made. The χ^2 (degree of freedom, 1) statistics were used to compare changes in asthma symptoms, emergency department contact, absence from school or work, and inhaled β_2-agonist consumption.

Results

There were no differences between the two groups in age, duration of asthma, lung functions, or medication, before training (Table 1). However, there was a small but significant increase, from 57.3 ± 3.2 to 65.2 ± 3.2 (mean $\pm$ SEM, $p<0.005$) in FEV_1 and from 76.8 ± 3.1 to 86.6 ± 2.5 ($p<0.005$) in FVC (percentage of predicted normal values) after six months in the training group but not in the control group (Table 2).

Respiratory muscle strength and endurance were unchanged in the control group after the six months of training (Table 2). In contrast, there was a significant increase in respiratory muscle strength as expressed by the PImax at RV (from 84.0 ± 4.3 cm H_2O to 107.0 ± 4.8 cm H_2O; $p<0.0001$), and in respiratory muscle endurance, as expressed by the relationship between $P_m$peak and PImax (from 67.5 ± 3.1 percent to 93.1 ± 1.2 percent, $p<0.0001$), in patients of group A.

There was also significant improvement as compared with baseline for asthma symptoms (nighttime asthma, $p<0.05$; morning tightness, $p<0.05$; daytime asthma, $p<0.01$; cough, $p<0.005$) in patients of group A but not in group B (Fig 1), following the training period.

Similar results were evident in the diary cards scores for inhaled β_2 usage ($p<0.05$) and the number of hospital ($p<0.05$) and sick-leave ($p<0.05$) days due to asthma (Fig 2).

Five patients were able to stop oral/IM corticosteroid therapy while on training and one in the placebo group was able to stop.

Discussion

In our study, we found that specific inspiratory threshold loading training, five times a week, for ½ h each session, for six months, markedly improved inspiratory muscle strength and endurance, as well as reduced asthma symptoms, hospitalizations for asthma, emergency department contact, absence from school or work, and medication consumption in patients with asthma.

Asthmatic patients are exposed to airway obstruction and hyperinflation. Airway resistance is increased up to 15 times normal[13] but it is probably the concomitant hyperinflation that impaires the capacity of the respiratory muscles to handle this load.

The main mechanism whereby hyperinflation adversely affects the inspiratory muscle is by forcing them to operate in an inefficient part of their force-length relationship. Hyperinflation shortens the inspiratory muscles and diminishes their ability to

Table 1—*Characteristics of Patients*

	Group A	Group B
No. of patients, M/F	15 (9/6)	15 (9/6)
Mean age, yr	42.3 ± 7.6	38.7 ± 6.2
Mean duration of asthma, yr	14.7 ± 4.3	15.4 ± 4.8
FEV_1, % of predicted	57.3 ± 6.2	60.6 ± 5.8
FVC, % of predicted	76.8 ± 7.1	73.6 ± 6.6
Current medication		
Oral steroids	6	7
Inhaled steroids	8	6
Sodium cromoglycate	2	3
Inhaled β_2-agonist	15	15

Table 2—*Lung Function Values, Inspiratory Muscle Strength, and Endurance in 30 Patients with Bronchial Asthma**

	FEV_1		FVC		PImax		Pm Peak/PImax, %	
Patient	Pre	Post†	Pre	Post†	Pre	Post†	Pre	Post†
Group A								
1	63	70	78	95	76	96	82	99
2	55	55	74	89	102	118	65	91
3	68	64	78	80	87	117	48	88
4	35	47	65	77	66	84	84	95
5	57	63	62	82	84	108	60	89
6	64	62	76	72	113	130	54	96
7	56	74	71	84	69	92	70	97
8	34	46	64	86	81	111	83	93
9	67	62	82	75	95	123	66	89
10	75	84	94	106	66	82	68	98
11	66	85	104	98	78	100	78	94
12	46	55	80	96	57	73	83	98
13	70	82	85	83	112	132	57	88
14	47	56	59	81	96	127	55	84
15	56	73	80	95	78	112	60	97
Mean ± SEM	57.3 ± 3.2	65.2‡ ± 3.2	76.8 ± 3.1	86.6‡ ± 2.5	84.0 ± 4.3	107.0§ ± 4.8	67.5 ± 3.1	93.1§ ± 1.2
Group B								
1	66	60	79	80	80	78	54	50
2	45	52	62	59	70	75	80	80
3	54	67	66	70	81	83	72	80
4	74	58	86	92	94	96	55	68
5	76	70	75	71	86	80	64	76
6	63	67	90	78	72	74	60	57
7	65	80	68	77	70	75	72	78
8	54	50	75	75	92	88	76	80
9	48	54	68	78	77	74	63	65
10	67	61	66	64	69	65	67	70
11	75	75	80	73	81	76	72	62
12	63	55	64	64	83	80	61	70
13	70	58	96	63	96	95	75	70
14	50	42	65	59	78	75	68	75
15	68	63	59	62	82	80	61	60
Mean ± SEM	62.5 ± 2.6	60.8 ± 2.6	73.3 ± 2.8	71.0 ± 2.4	80.7 ± 2.2	79.6 ± 2.1	66.7 ± 2.0	69.4 ± 2.4

*All lung function data are expressed as percentage of predicted normal values. PImax values in cm H_2O.
†Posttraining values.
‡$p<0.005$ (post vs pre values).
§$p<0.0001$ (post vs pre values).

generate negative pressure while inspiring.[14] It causes the flattening of the diaphragm, which in turn places it in a serious mechanical disadvantage, because it has to be curved upwards (according to Laplace's law) in order to be effective.[15] The axial direction of the diaphragmatic fibers is also lost by hyperinflation. They are directed medially or inward and have mainly expiratory action.[16] The area of apposition between the costal fibers of the diaphragm and the inner rib cage becomes smaller,[17] resulting in less effective rib cage expansion during inspiration. The thoracic elastic recoil that is normally directed outwardly, in resting lung volume, becomes directed inwardly with hyperinflation causing an added elastic load to the inspiratory muscles.[18] Hyperinflation also places the ribs in a more horizontal position, causing the external intercostal muscles to act as an expiratory muscle instead of the normal inspiratory action.[19] Finally, as the contractile forces increase in order to develop the inspiratory pressure necessary to inflate the hyperinflated lung, the respiratory muscle blood supply may be altered.[20]

A number of studies have been carried out to correlate dyspnea and respiratory muscle performance. It was well documented that the intensity of breathlessness is related to the activity and the strength of the inspiratory muscles.[2,21,22] Although the patients studied had only slightly reduced inspiratory muscle strength and endurance (mean PImax, 76 percent of predicted values), their increased work combined with impaired function might account for the patients' sense of dyspnea. Therefore, there appears to be a rational therapeutic place for SIMT in patients with asthma, as an alternative to common acceptable medical therapy. If dyspnea is related to the increased work, combined with impaired function of the inspiratory muscles, then improved strength and endurance of those muscles must be followed by

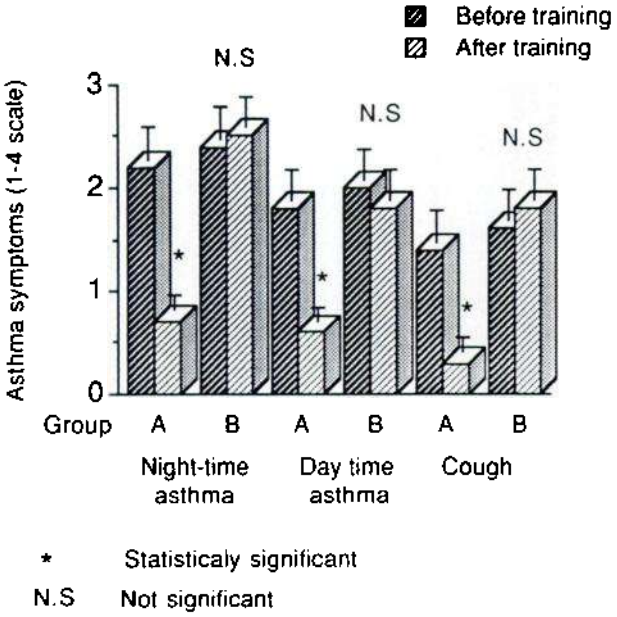

FIGURE 1. Diary card data for asthma symptoms judged by the patients on a scale 0, no symptoms, 4, very severe before and during the last two weeks of training. Values are mean (± SEM).

improving this symptom. In addition, circumstantial evidence exists to suggest that the inspiratory muscles may suffer damage during an acute asthmatic attack that may lead to acute respiratory failure.[23] Improved strength and endurance of the inspiratory muscles may delay the onset of respiratory muscle fatigue and respiratory failure in those patients. However, there is no good explanation for the improvement in cough observed in our patients following the training period.

It is well established that respiratory muscles can be trained like other skeletal muscles, and several reviews have been published dealing with ventilatory muscle training.[24,25] The new threshold inspiratory muscle trainers are designed to provide a specific, constant workload that is independent of variations in inspiratory flow rate. In a recent double-blind study, Larson and associates[26] demonstrated that patients who trained with threshold trainer at 30 percent of their PImax for two months were able to increase their respiratory muscle strength and endurance. Therefore, it is not surprising that all our patients who trained with the threshold pressure breathing device improved their inspiratory muscle strength and endurance. The improved performance of the inspiratory muscles was associated with improvement in all of the clinical parameters recorded by us. In addition, when assessing the results of the present study, it is important to take in account that five of six patients in the training group who were receiving systemic corticosteroids when entering the study stopped the treatment during the training period without any clinical

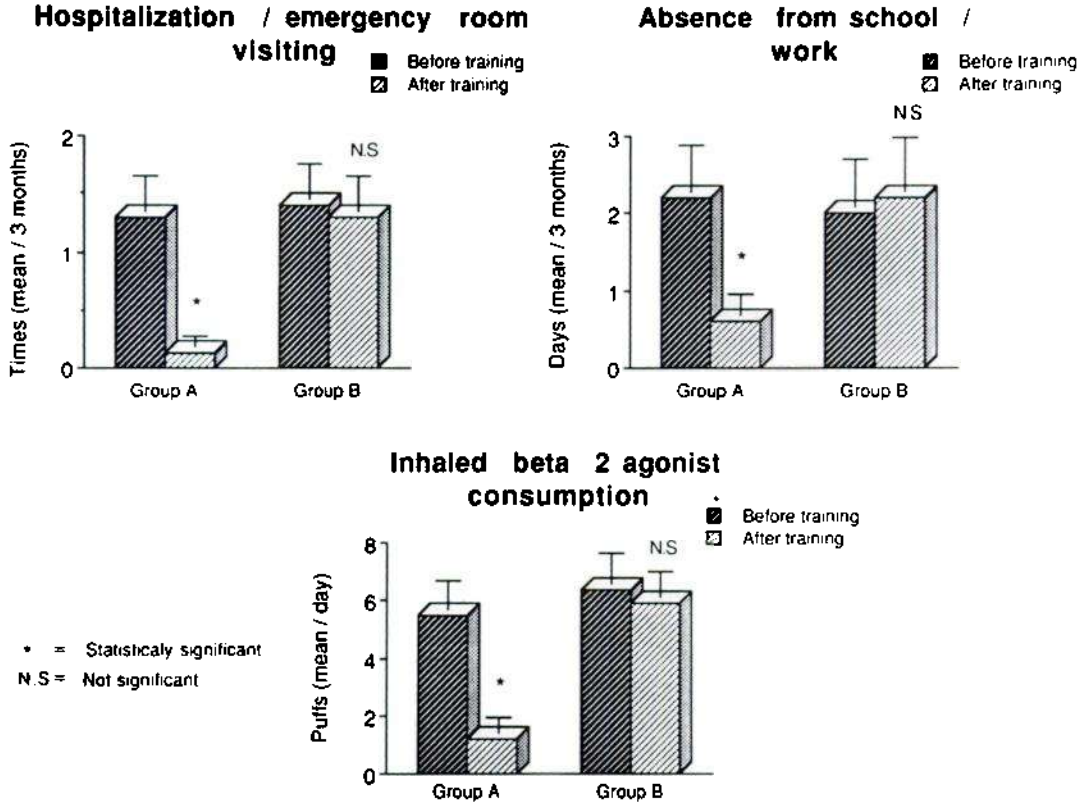

FIGURE 2. Change in number of hospitalizations or emergency department visitings for asthma, days of absence from school or work, and inhaled β_2-agonist consumption before and during the last three months of training. Values are mean (± SEM).

deterioration. This, by itself, might improve respiratory muscle performance, as it is known that systemic corticosteroids may have an adverse affect on those muscles. However, the improvement in inspiratory muscle performance, in our study, was seen much before the corticosteroids therapy was stopped. An alternative explanation for the improvement in asthma symptoms and the reduced usage of bronchodilators observed in our patients could be that subjects became desensitized to the sensation of dyspneas experienced with increased airway resistance and because of desensitization they were less bothered by dyspnea.

The mechanism underlying the improvement in lung functions, observed in our patients, is not clearly understood. The absolute volume of the total lung capacity and its subdivisions is determined by the balance between the elastic forces and the inspiratory muscles.[27] Thus, the increase in FVC observed in our patients might be related to the enhanced strength of the inspiratory muscles following training. The resistance to airflow varies with lung volume, and it is less at higher lung volumes. Thus, the increase in flow rates is probably secondary to the change in lung volumes rather than a real change in airway resistance. It is still possible that the increase in FVC and FEV_t, presumably with reductions in the degree of hyperinflation, had an advantageous affect on the respiratory muscles. However, such degree of improvement in inspiratory muscle performance had not been observed by us, in a previous study,[5] just by decreasing the degree of hyperinflation in patients with bronchial asthma. Lung volume measurements would obviously be of interest in these patients. However, lung volumes were not measured in this study.

In conclusion, we believe that SIMT may prove to be a complementary or alternative and more physiologic therapy with the aim of reducing systemic corticosteroids requirement and inhaled β_2-agonist consumption and improving the control of asthma symptoms in patients with asthma.

References

1 Killian KG, Campbell EJM. Dyspnea and exercise. Ann Rev Physiol 1983; 45:465-79

2 Killian KG, Jones NL. The use of exercise testing and other methods in the investigation of dyspnea. Clin Chest Med 1984; 5:99-108

3 Tobin MJ. Respiratory muscles in disease. Clin Chest Med 1988; 9:263-86

4 Permutt S. Physiologic changes in the acute asthmatic attack. In: Austen KF, Lichtenstein L, eds. Asthma, physiology, immunopharmacology and treatment. New York: Academic Press, 1973:15

5 Weiner P, Suo J, Fernandez E, Cherniack RM. The effect of hyperinflation on respiratory muscle strength and efficiency in healthy subjects and patients with asthma. Am Rev Respir Dis 1990; 141:1501-05

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

7 Gross D, Ladd HW, Riley E, Macklem P, Grassino A. The effect of training on strength and endurance of the diaphragm in quadriplegia. Am J Med 1980; 68:27-35

8 Pardy RL, Rivington RN, Despas PJ, Macklem PT. The effects of inspiratory muscle training on exercise performance in chronic airflow limitation. Am Rev Respir Dis 1981; 123:426-33

9 Committee on Diagnostic Standards for Nontuberculous Respiratory Disease. Chronic bronchitis, asthma and pulmonary emphysema. Am Rev Respir Dis 1962; 85:762

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

11 Nickerson BG, Keens TG. Measuring ventilatory muscle endurance in humans as sustainable inspiratory pressure. J Appl Physiol 1982; 52:768-72

12 Martyn JB, Moreno RH, Pare PD, Pardy RL. Measurement of inspiratory muscle performance with incremental threshold loading. Am Rev Respir Dis 1987; 135:919-23

13 Rochester DF, Arora NS. The respiratory muscles in asthma. In: Lavietes MH, Reichman L, eds. Symposium on bronchial asthma: diagnostic aspects and management of asthma. New York: Purdue Frederick Co, 1982:27-38

14 Rahn H, Otis AB, Chadwick LE. The pressure-volume diagram of the thorax and lung. Am J Physiol 1946; 146:161-78

15 Sharp JT. The respiratory muscles in chronic obstructive pulmonary disease. Am Rev Respir Dis 1986; 134:1089-91

16 Minh VD, Dolan GF, Konopka RF, Moser KM. Effect of hyperinflation on inspiratory function of the diaphragm. J Appl Physiol 1976; 40:67-73

17 Mead J. Functional significance of the area of apposition of diaphragm to rib cage. Am Rev Respir Dis 1979; 119:31-2

18 Sharp JT. The respiratory muscles in emphysema. Clin Chest Med 1983; 4:421-32

19 De Troyer A, Kelly S, Macklem PT, Zin WA. Mechanics of intercostal space and actions of external and internal intercostal muscles. J Clin Invest 1985; 75:850-57

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

21 Jones GL, Killian KJ, Summers E. The sense of effort, oxygen cost, and pattern of breathing associated with progressive elastic loading to fatigue. Fed Proc 1984; 42:1420

22 Killian KJ, Gandevia SC, Summers E. Effect of increased lung volume on perception of breathlessness, effort, and tension. J Appl Physiol 1984; 57:686-91

23 Pardy RL, Leith DE. Ventilatory muscle training. In: Rousos C, Mackelm PT, eds. The thorax. New York: Marcel Dekker, Inc, 1985:1353-69

24 Appel D, Rubenstein R, Schrager K. Lactic acidosis in severe asthma. Am J Med 1983; 75:580-84

25 Burki NK, Diamond L. Serum creatinine phosphokinase activity in asthma. Am Rev Respir Dis 1977; 116:327-31

26 Larson JL, Kim MJ, Sharp JT, Larson DA. Inspiratory muscle training with a pressure threshold breathing device in patients with chronic obstructive pulmonary disease. Am Rev Respir Dis 1988; 138:689-96

27 Cherniack RM. Pulmonary function testing. Philadelphia: WB Saunders Co, 1977:39, 195