

Inspiratory Muscle Training in Patients With COPD*

Effect on Dyspnea, Exercise Performance, and Quality of Life

Hildegard Sánchez Riera, MD; Teodoro Montemayor Rubio, MD; Francisco Ortega Ruiz, MD; Pilar Cejudo Ramos, MD; Daniel Del Castillo Otero, MD; Teresa Elias Hernandez, MD; and Jose Castillo Gomez, MD

Objective: The aim of the study was to assess the effect of target-flow inspiratory muscle training (IMT) on respiratory muscle function, exercise performance, dyspnea, and health-related quality of life (HRQL) in patients with COPD.

Patients and methods: Twenty patients with severe COPD were randomly assigned to a training group (group T) or to a control group (group C) following a double-blind procedure. Patients in group T (n = 10) trained with 60 to 70% maximal sustained inspiratory pressure (SIPmax) as a training load, and those in group C (n = 10) received no training. Group T trained at home for 30 min daily, 6 days a week for 6 months.

Measurements: The measurements performed included spirometry, SIPmax, inspiratory muscle strength, and exercise capacity, which included maximal oxygen uptake ($\dot{V}O_2$), and minute ventilation ($\dot{V}E$). Exercise performance was evaluated by the distance walked in the shuttle walking test (SWT). Changes in dyspnea and HRQL also were measured.

Results: Results showed significant increases in SIPmax, maximal inspiratory pressure, and SWT only in group T ($p < 0.003$, $p < 0.003$, and $p < 0.001$, respectively), with significant differences after 6 months between the two groups ($p < 0.003$, $p < 0.003$, and $p < 0.05$, respectively). The levels of $\dot{V}O_2$ and $\dot{V}E$ did not change in either group. The values for transitional dyspnea index and HRQL improved in group T at 6 months in comparison with group C ($p < 0.003$ and $p < 0.003$, respectively).

Conclusions: We conclude that targeted IMT relieves dyspnea, increases the capacity to walk, and improves HRQL in COPD patients. (CHEST 2001; 120:748–756)

Key words: COPD; exercise performance; inspiratory muscle training; quality of life

Abbreviations: BDI = baseline dyspnea index; CI = confidence interval; CRQ = chronic respiratory questionnaire; HRQL = health-related quality of life; IMT = inspiratory muscle training; MCID = minimum clinically important difference; Pbreath/Pimax = balance between the magnitude of the breathing task and the strength to carry out the task; Pimax = maximal static inspiratory pressure; SIPmax = maximal sustained inspiratory pressure; SWT = shuttle walking test; TDI = transitional dyspnea index; $\dot{V}E$ = minute ventilation; $\dot{V}O_2$ = oxygen uptake; Wmax = maximal workload

Patients with COPD experience increased resistance to airflow, air-trapping, and hyperinflation of the lung. Hyperinflation places the inspiratory muscles at a mechanical disadvantage. Inspiratory muscle weakness, resulting from mechanical disadvantage or from any other cause, contributes to the

increase in the balance between the magnitude of the breathing task and the strength to carry out the task (Pbreath/Pimax) and to the sensation of respiratory effort that appear to mediate the reduction in tidal volume. Breathing is diminished, and respiratory rate becomes more rapid. The precise neural mechanisms responsible for changing the respiratory pattern are unknown but are thought to involve a response to the perception of a large increase in Pbreath/Pimax.¹ According to some authors,^{2,3} inspiratory muscle training (IMT) may improve inspiratory muscle strength and lower Pbreath/Pimax, and probably will diminish the sensation of respiratory effort.

*From the Pneumology Service, Virgen Del Rocio University Hospital, Sevilla, Spain.

Supported by "Junta de Andalucía" grant No. 94/535–119. Manuscript received December 9, 1999; revision accepted March 16, 2001.

Correspondence to: Hildegard Sánchez Riera, MD, Urb. "La Motilla," C/Rayo 4, 41700 Dos Hermanas, Sevilla, Spain; e-mail: ablucil@mx2.redestb.es

Several authors have shown^{2,4} that targeted IMT may enhance respiratory muscle function and reduce dyspnea in patients with moderate-to-severe COPD. Of the 13 studies included in a meta-analysis of IMT in COPD patients,⁵ the authors reported nonsignificant changes in maximal static inspiratory pressure (P_{imax}) in 11 studies and in respiratory muscle endurance in 9 studies in which these factors were evaluated. The findings demonstrate that the training stimulus in these studies was inadequate to induce the expected physiologic training responses. Of the 11 studies that addressed the effects of resistance training, the flow rates (and thus the resistance) generated during training were controlled by the investigators in only 4 studies.^{2,4,6,7} Additional sensitivity analysis by Smith et al⁵ suggested that resistance training might result in appreciable improvements in strength and endurance if the breathing pattern was controlled. The results of this analysis further suggest that when the breathing pattern is controlled, the increase in respiratory muscle strength and endurance may translate into clinically important improvement in functional status. There have been numerous studies carried out concerning IMT in COPD patients, but there have been very few using a controlled breathing pattern. Our IMT protocol controlled the target flow and respiratory cycle using both an incentive target-flow device to facilitate control through visual feedback and another simple feedback device of our own design. In addition, we evaluated the clinical effect of IMT with a validated questionnaire for COPD patients.⁸

Finally, according to the theory of Rochester³ on therapeutic measures, we hypothesized that the combination of a training program that attempts to obtain better inspiratory muscle shortening with biofeedback techniques would reduce the sensation of inspiratory effort.

The goal of our study was to assess the effect of IMT on dyspnea, exercise performance, and health-related quality of life (HRQL) in COPD patients.

MATERIALS AND METHODS

Patients

Twenty patients with COPD were selected from the outpatient clinic. Patients were randomly assigned to the training group (group T) or to a control group (group C) on a double-blind basis. Table 1 shows the baseline characteristics of the patients. All patients had severe airflow obstruction (*ie*, FEV₁ < 50%) and were included with the presence of COPD as defined by the American Thoracic Society.⁹ Patients were in stable condition and were free of any clinical evidence of cardiovascular, musculoskeletal, or neuromuscular disease or of any other disease that might interfere with exercise. Group T was trained with a load of

Table 1—General Characteristics and Pulmonary Function of the Patients*

Characteristics	Group T (n = 10)	Group C (n = 10)	p Value
Sex			NS
M	9	9	
F	1	1	
Age, yr	67 ± 4	67.6 ± 5	NS
FVC, % predicted	63.6 ± 16	64.2 ± 15	NS
FEV ₁ , % predicted	38.3 ± 13	41.3 ± 11	NS
P _{imax} , cm H ₂ O	44.5 ± 14	50.3 ± 13	NS

*Values given as mean ± SD. M = male; F = female; NS = not significant.

60 to 70% of the maximal sustained inspiratory pressure (SIP_{max}; *ie*, approximately 30% of the P_{imax}), and the control group was trained at zero load (the flowmeter air leak was closed). Patients were unaware of the magnitude of the load, and the investigators performing the measurements were also unaware of the patient's training load. All subjects completed a battery of pulmonary function tests and were instructed to take their prescribed medications as usual.

Patients were informed of the purpose of the study and had agreed to participate. The protocol was approved by the Ethics Committee of the Hospital Universitario Virgen del Rocío of Seville, Spain.

Target-Flow IMT

Training was performed at home using an incentive flowmeter device with visual feedback (INSPIRx; Intertech Resources Inc; Ft. Myers, FL), which was used for target-flow IMT. Patients were instructed to generate an inspiratory flow rate at which the ball in the flowmeter reached the top of the device (target-flow). The adjustable flowmeter escape was set so that the patient had to generate 60 to 70% of his/her SIP_{max}. The duration of inspiration was 1.5 to 2 s, and the duration of expiration was 6 s. The respiratory rate, therefore, was approximately 8 breaths/min. The target-flow device was used both for testing in the laboratory as well as for training at home. Measurements were taken in group T every 6 weeks, and the training load was modified, adjusting the target-flow training device to the new SIP_{max} to maintain it at 60 to 70% during the 6 months of the training period. Group C patients also were evaluated every 6 weeks, but the flowmeter air leak was kept closed during the study period so that there would be no load.

Group T patients started with relaxation exercises for 5 min and continued on to IMT for 15 min bid, 6 days a week for 6 months. Group C patients started with relaxation exercises for 5 min and continued breathing with the flowmeter with no load for 15 min bid, 6 days a week for the 6 months of the study.

Respiratory Cycle Feedback Device

The respiratory cycle was controlled by means of a simple feedback device designed by our study group. This device consists of two sequence timers that were adjusted in such a way that the first paced the inspiratory timing using a light signal that remained lit for 2 s, and the second paced the expiratory timing, which was regulated so that the light signal was turned off for 6 s. Since there was a time lag of 0.5 s between the onset of inspiration and the achievement of the target pressure, the inspiratory time lasted approximately 1.5 to 2 s. Patients were

instructed to inhale when this light went on, and they maintained the target-flow until the light went out. The respiratory cycle feedback device directed the patient to maintain a constant preset respiratory cycle. This respiratory cycle was well-tolerated by COPD patients. The respiratory cycle device was used in the first part of the study (pretraining phase) as well as in successive endurance tests.

Methods

Experimental Setup (Laboratory Study): A two-way nonrebreathing valve (HansRudolph; Kansas City, MO) and mouthpiece were placed proximal to the flowmeter (target-flow-IMT). A pneumotachometer (Fleisch No. 3) was placed between the valve and the flowmeter to measure airflow. Mouth pressure was recorded via a differential pressure transducer (model MP-45; Validyne; Northridge, CA) connected to an opening in the flowmeter mouthpiece. The respiratory pattern was controlled by means of the respiratory cycle feedback device (previously described). Patients breathed through the flowmeter, inspiratory flow and pressure signals were transmitted to a microcomputer (IBM-AT; IBM; White Plains, NY), and both measures were recorded. In this way, there was confirmation of the pressures at each flow level of the incentive flowmeter and of the correspondence of the flows registered in the pneumotachograph with those displayed in the incentive flowmeter. There were no significant differences between the systems.

Protocol: The study was carried out in two parts. In the first part, a 4-week learning phase (pretraining), patients in group T were familiarized with the target-flow device and were taught the relaxation techniques by a physiotherapist. They also were shown how to acquire the desired respiratory pattern by learning respiratory times and flow. At the end of each week, each patient was checked to see whether he or she had been able to assimilate the preset respiratory cycle without using the respiratory cycle feedback device, although they were aided by means of a metronome. Before performing each endurance test, to verify that the patients had followed the preset respiratory pattern at home, each followed the respiratory cycle with the metronome, and mouth pressure was recorded via a differential pressure transducer connected to an opening in the mouthpiece of the flowmeter (target-flow) device. Patients adhered to the preset respiratory rhythm and pressure without having to make corrections. Since they regularly visited the laboratory and did not demonstrate significant changes in the respiratory pattern, we assumed they were compliant. We also tested group C patients, who learned the relaxation techniques for a period of 4 weeks, in a manner similar to the training group. They were instructed to breathe without discomfort, raising the flowmeter ball but not maintaining it at the top of the device. We checked that breathing was at zero load (with the flowmeter air leak closed). Patients in each group received a similar level of attention. Baseline parameters were measured during the 4 apprenticeship weeks, and the number of tests applied to each group was the same. In the second part of the study (training phase), the patients visited the laboratory twice (every 15 days) only in the first month to verify that the ventilatory pattern was unchanged.

All tests were carried out at the beginning and end of training, except for the P_{imax}, which was recorded three times (at the beginning of training, after 3 months, and at the end of training), and the SIP_{max}, which was measured four times (every 6 weeks). The effects of training were assessed by measuring changes in pulmonary function, SIP_{max}, P_{imax}, dyspnea, HRQL, exercise capacity on the cycle ergometer, and the distance walked in the shuttle walking test (SWT). For the latter two tests, respiratory effort was evaluated using the Borg psychophysical scale.¹⁰

Measurements

Pulmonary Function: Spirometry was performed with the patient in the seated position using a 9-L bell-type spiograph (Stead Wells Volumograph; Minjhardt; Cologne, Germany). Pulmonary function values were based on the effort (the best of three) having the greatest sum of FEV₁, FVC, FEV₁ as a percentage of the FVC, and midexpiratory flow. Predicted normal values were determined using the equations of Morris and coworkers.¹¹

Inspiratory Muscle Endurance Test: To evaluate changes in the SIP_{max}, the progressive inspiratory muscle endurance test of Martyn et al,¹² which determines the SIP_{max}, was used. The respiratory pattern was controlled using increments of the inspiratory load target every 2 min. The patient breathed slowly (through the training device) with no load for 2 min. The target load began at -6 cm H₂O pressure, and it was increased every 2 min by -2 cm H₂O until it reached a flow level such that, if it was sustainable for at least 60 s, the pressure achieved was considered to be the maximal pressure and was defined as the SIP_{max}. This was repeated every 6 weeks.

P_{max}: The P_{max} was measured under static conditions, at functional residual capacity, by means of a flanged mouthpiece that was occluded with a small leak and was connected to a manometer (model 163; Sibelmec; Barcelona, Spain), according to the method proposed by Black and Hyatt.¹³ The P_{max} was recorded three times (at the beginning of the study period, after 3 months, and at the end of the study period), and three measurements were made in each testing session. Before obtaining these measurements, patients were given a minimum of three practice attempts to reduce any learning effect. The best effort was recorded as the P_{max} for each test session.

Maximal Exercise Capacity: Exercise capacity was evaluated by a maximal incremental exercise test using a commercially available cardiopulmonary exercise system (CPX/PLUS System; Collins Medical; Boston, MA). Exercise testing was performed on a cycle ergometer. After 1 min of unloaded pedaling, the work rate was increased 10 W/min at a time. The test was stopped when patients were unable to continue because of dyspnea or leg fatigue. The following parameters were determined during this test: the maximal workload (W_{max}), minute ventilation (\dot{V}_E), and maximal oxygen uptake ($\dot{V}O_2$). Heart rate, arterial oxygen saturation, and BP were monitored. The respiratory effort was evaluated using the Borg psychophysical scale.⁸ Maximal exercise capacity was evaluated through changes in the \dot{V}_E and $\dot{V}O_2$ that were developed at the W_{max}.

Exercise Performance: Functional capacity during exercise was assessed using the SWT, which is a maximal symptom-limited test with 12 progressive levels.¹⁴ Patients were required to walk 10 m back and forth. The walking speed was paced by an audio signal from a cassette that emitted beeps at regular intervals. The speed was increased each minute by 0.17 m/s until the next level was attained. The end of the test was determined by patients, when they were too breathless to maintain the required speed, or by operators, if patients failed to complete a shuttle in the time allowed. The level reached in the SWT, the distance traveled in meters, and the respiratory effort sensation determined by the Borg scale were measured at the end of each effort. Only one test was necessary in each patient since our group had verified its reproducibility in a previous study.¹⁵

Dyspnea: Dyspnea is defined as the perception of difficult breathing provoked by an activity not expected to produce it. Dyspnea was measured by the following two instruments: the baseline dyspnea index (BDI) and the transitional dyspnea index (TDI), which were scored using the procedures of Mahler et al.¹⁶ The BDI measures the following three components that are influenced by dyspnea: functional impairment (*ie*, the degree to

which the activities of daily life are impaired); magnitude of effort (*ie*, the overall effort exerted to perform activities); and the magnitude of the task that provokes breathing difficulty. The BDI measures dyspnea at one point in time, with scores ranging from 0 (dyspnea with no task, dyspnea with no effort, very severe impairment) to 4 (dyspnea with extraordinary activity, dyspnea with extraordinary effort, no impairment). The effect of training was assessed using the TDI, which is a clinical questionnaire designed to evaluate and quantify dyspnea and to measure changes from baseline values, with scores ranging from -3 (*ie*, major deterioration from baseline) to $+3$ (*ie*, major improvement from baseline). The influence of dyspnea on functional impairment, magnitude of effort, and magnitude of task were assessed. Improvement was reflected by positive scores, and worsening was reflected by negative scores.

Quality of Life: Quality of life in relation to health was assessed by the chronic respiratory questionnaire (CRQ) developed by Guyatt et al.⁸ The CRQ has been translated and validated for use in Spanish¹⁷ and is useful for quantifying the quality of life of COPD patients. The CRQ is divided into four categories (dyspnea, fatigue, emotion, and mastery) and measures both physical and emotional function. Physical function assessment includes questioning patients to quantify their dyspnea during five activities that are frequently performed and are important in day-to-day life. Each patient is asked to choose 5 activities from a list of 25, or the patient may mention other activities that are not on the list. This means that the dyspnea category is strictly individualized. Physical function also was investigated with four items related to fatigue and energy levels. The assessment of emotional function, which is related to the categories of emotion (seven items) and mastery (four items), included questions about frustration, depression, anxiety, panic, and fear of dyspnea. Patients were asked to rate their physical and emotional function on a 7-point scale, a higher score representing better function (1, extremely tired; 7, not tired at all). The clinical consequence of the changes obtained in the quality of life after training was evaluated by a comparison with the minimum clinically important difference (MCID), which is defined as the smallest difference perceived to be important by the average patient.¹⁸ We considered an increase of at least 0.5 points as being an MCID.¹⁹ A change of 1 indicated a moderate change, and a change of >1 indicated a large difference.

Statistical Analysis

The Student's *t* test for paired samples was used in the comparison of means between groups before and after training, as well as for comparing independent samples between the trained group and the control group. Nonparametric tests were used in the evaluation of clinical data for paired samples (Mc-

Nemar test) or independent samples (Mann-Whitney *U* test). A *p* value < 0.05 was taken as the level of statistical significance. We calculated the 95% confidence interval (CI) around the differences in means between the baseline and posttraining values in the trained group.

RESULTS

All selected patients completed the study. There were no significant differences between the two groups at the start of the study. The general characteristics and pulmonary function data are presented in Table 1.

Pulmonary Function

After the study period, there were no significant differences in spirometry results in either group.

Inspiratory Muscle Endurance Test

The SIPmax increased after training in group T ($p < 0.003$) with a significant difference at 6 months compared to that in group C ($p < 0.003$). There was no statistically significant difference in group C at the end of the study period. The means are shown in Table 2.

P_{imax}

In group T, the peak P_{imax} increased from 44.5 ± 14.1 to 66.1 ± 15.8 cm H₂O after training ($p < 0.003$), with no change in group C. Differences between the two groups after training were significant ($p < 0.003$). Table 2 shows the mean values at the end of the study period.

Maximal Exercise Capacity

There were no significant changes in the maximal $\dot{V}O_2$, maximal exercise ventilation, and W_{max} in either group at the sixth month of the study period. There were no changes in Borg scale scores in either

Table 2—Ventilatory Muscle Function and Maximal Exercise in Ergometric Bicycle Test*

Variable	Group T		Group C	
	Baseline	After 6 mo	Baseline	After 6 mo
$\dot{V}O_2$ max, L/min	1.3 ± 0.2	1.1 ± 0.2	1.17 ± 0.3	1.21 ± 0.3
W _{max} , w	49.0 ± 16	52.0 ± 15.4	66.0 ± 17	58.5 ± 18
HR _{max} , beats/min	116.0 ± 11	108.0 ± 11	116.0 ± 9.8	115.0 ± 14.4
$\dot{V}E$ max, L/min	35.9 ± 11.3	35.3 ± 7.1	34.0 ± 7.9	37.0 ± 9.1
P _{imax} , cm H ₂ O	44.5 ± 14.1	$66.1 \pm 15.8^\dagger$	50.3 ± 13.6	48.5 ± 17.3
SIPmax, cm H ₂ O	20.2 ± 5.9	$27.5 \pm 4.2^\dagger$	21.3 ± 3.2	20.0 ± 2.4

*Values given as mean \pm SD. $\dot{V}O_2$ max = maximal oxygen uptake; HR_{max} = maximal heart frequency; $\dot{V}E$ max = maximal minute ventilation. $^\dagger p < 0.003$, significant in relation to its baseline value and at 6 months compared to group C.

group. Table 2 shows the mean \pm SD of the maximal exercise test for both groups.

Exercise Performance

The patients in group T significantly increased the distance walked in the SWT in relation to the baseline value ($p < 0.001$) and at 6 months with respect to group C ($p < 0.05$), while group C showed no changes. The perceived sensation of exercise (Borg scale score) did not change in either group. Table 3 shows the statistical significance of the means of both groups, at baseline and at the end of 6 months. The results of the SWT adjusted to the 95% CI for the mean differences between the baseline values and those measured after training in group T are shown in Table 4.

Dyspnea

Both groups had similar baseline dyspnea values. After 6 months of IMT, the TDI was 4.7 ± 0.6 points in group T and 0.2 ± 0.11 points in group C. Statistical significance was seen at 6 months in group T in comparison with the values in group C ($p < 0.003$). Figure 1 shows the global change of the TDI at the end of the 6-month period for both groups, and its statistical significance. The results adjusted to the 95% CI for the differences in means in group T in relation to its baseline value are shown in Table 4.

Quality of Life

For each category, the overall treatment effect was greater in the trained group than the MCID (0.5 points). The score at the end of 6 months is shown in Figure 2 in terms of the mean values for both groups, as is the statistical significance for group T with regard to group C. The 95% CI values, adjusted for differences in mean values in group T in relation to baseline values, are shown in Table 4.

DISCUSSION

This study shows that in COPD patients, IMT at home using an incentive flowmeter device (target-flow)

Table 3—Distance Walked in the SWT and the Respiratory Effort Measured by the Borg Score at Baseline and After 6 Months in Both Groups*

Variable	Group T		Group C	
	Baseline	After 6 mo	Baseline	After 6 mo
SWT, m	448 \pm 121	541 \pm 112†‡	551 \pm 174	493 \pm 140
Borg score	8.1 \pm 1.04	8.3 \pm 0.78	7.6 \pm 1.43	8.3 \pm 1.41

*Values expressed as mean \pm SD.

† $p < 0.001$, for group T in relation to its baseline value.

‡ $p < 0.05$, after 6 mo in group T with respect to group C.

Table 4—Outcome Measures and Mean Differences Between Values at Baseline and After Training in Group T

Outcome Measures	Mean Difference in Group T* (95% CI)
Dyspnea (TDI)	4.7 (4.2–5.2)
Dyspnea (CRQ)	1.6 (0.41–1.78)
Fatigue (CRQ)	1.55 (0.99–2.01)
Emotion (CRQ)	1.28 (0.90–1.66)
Mastery (CRQ)	1.48 (0.73–2.24)
SWT	93 (58.1–127.9)

*At baseline and after training in group T.

diminishes dyspnea and improves respiratory muscle function, exercise performance, and HRQL.

The baseline characteristics of the two groups were similar. Although the trained patients were not supervised while performing the training at home, we assume from the tests performed in the laboratory throughout the study period that they followed the same regimen at home. In addition, the study shows a load-dependent training effect on P_{imax}. In the trained group, good results were obtained in P_{imax} and other tests, which we think should not be attributed to a learning or coaching effect since the number of tests applied to each group was the same and since group C showed no changes in the results at the end of the study period. In both groups, we reviewed the routine medical therapy and relaxation exercises, which were without load. The training was planned this way since in other studies,^{20,21} loads of 10%²⁰ and 15%²¹ of the P_{imax} had a training effect

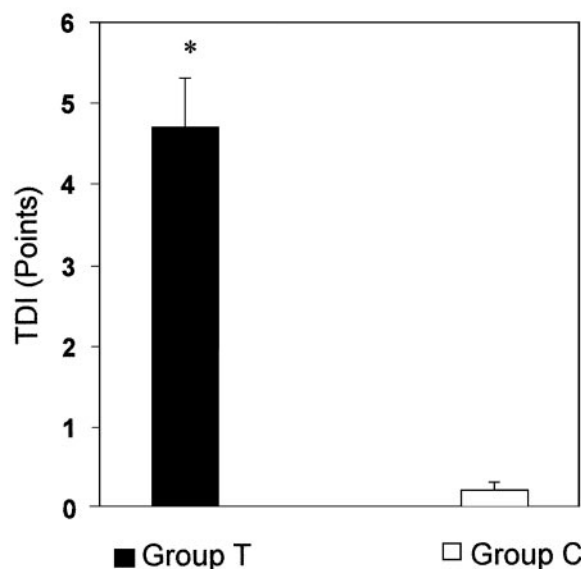


FIGURE 1. Mean values of TDI after 6 months in group T and group C. * = $p < 0.003$.

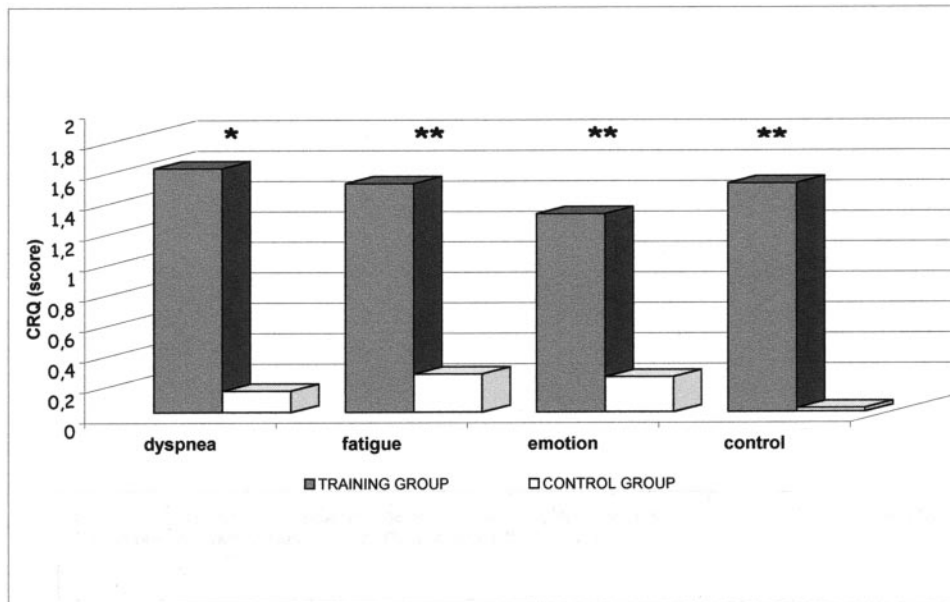


FIGURE 2. Mean score for each category of the HRQL after 6 months in group T and group C. * = $p < 0.003$; ** = $p < 0.001$.

on the P_{imax}. Patients in the control group in our study breathed through the flowmeter at essentially zero load so that it would be considered double-blind for both the patients and the investigators. As has been shown, there was no training effect on P_{imax} values in this group during the study period.

No changes were observed for the pulmonary function parameters, as has been the case in similar studies.^{2,4} Training intensity was based on a percentage of the SIP_{max}. Clanton et al²² have shown that the ability to sustain a given pressure when the muscles can shorten appreciably is not a fraction of the P_{imax}. In this case, the pressure was defined by the point at which muscle contraction occurred in relation to the maximal pressure-flow-volume relationship. Hence, the fatigue threshold considered in our study depended on the type of contraction employed during ventilation. The respiratory pattern was controlled during the progressive endurance test, and approximately every 3- to 4-cm H₂O increment in SIP_{max} values corresponded to a 10-cm H₂O rise in P_{max} values. The load was similar to that observed by Larson et al.²³ However, the increments in the workload in group T were smaller (2 cm H₂O to maintain the training load at 60 to 70% of the SIP_{max}) and were adjusted every 6 weeks. P_{imax} was measured to assess the influence of strength on this type of breathing. In the training group, the increment observed in the P_{imax} between the first and second determination was 13 cm H₂O, which is similar to that observed by Larson et al²³ (14 cm H₂O). However, this elevation occurred after 1

month of training. Although smaller, there was also an increment of approximately 9 cm H₂O between the second and third determinations. In our case, the longer training period (6 months), together with the slow and deep breathing pattern, may have permitted greater increments in the P_{imax}. The plateau effect in the rise of P_{imax} after 1 month of IMT observed in the study by Larson et al,²³ as compared to a meta-analysis commentary,⁵ can probably be explained by the fact that the training intensity could have been less than that achieved had breathing frequency been controlled. For this reason, breathing frequency should be controlled when a pressure threshold device is used. In our study, the training intensity corresponded approximately to 30% of the P_{imax}, which is a good overload with which to achieve a training effect.^{20,23,24} According to other authors,^{25,26} the use of moderate loads to train inspiratory muscles achieves improvements in both strength and shortening velocity, thus in power output. The training protocol in the present study was performed with a target-flow device and an intermediate load. The SIP_{max} and inspiratory flow (target load) increased posttraining. For this reason, we assume that the shortening velocity of inspiratory muscles was augmented, and, thus, power output was improved. These issues were not addressed by the present study, but they need to be studied further.

Three basic principles of skeletal muscle training are overload, specificity, and reversibility.²⁷ The training used in our study rested on a training incentive of sufficient intensity to produce a training

effect (overload), and the training and testing devices used the same training modality (specificity). The reversibility principle states that the effects of conditioning decline after training ceases. Therefore, when patients finish the training period, we recommend that they continue performing breathing exercises regularly in order to maintain the obtained improvements.

No significant changes were documented in the progressive cycle ergometer test.²¹ The $\dot{V}O_2$ peak did not increase, and there were no changes in the $\dot{V}E$ after training. Flynn et al²⁸ did not observe changes in $\dot{V}O_2$ during exercise after 6 weeks of training. Although our study lasted longer (6 months), there were no changes in the maximal exercise capacity, and the patients did not tolerate this exercise (cycle) better, since the perception of dyspnea, as measured by the Borg scale, did not change and there were no changes in the maximal workload.

Exercise performance was assessed with a maximum and progressive test, the SWT, in which the patients in group T showed a significant improvement in the distance walked. We did not carry out the 6-min walking test since, according to our experience,¹⁵ the SWT has been demonstrated to yield more information concerning the functional assessment of the patient. The 6-min walking test has higher variability and is less reproducible. To evaluate different forms of effort (*ie*, pedaling and walking), we have used the following two tests: cycle ergometer and SWT. Group T patients walked 93 m further at the end of training. Another study showed an increment of 88 m²⁹; however, patients in that study were grouped according to the severity of dyspnea (Medical Research Council scale), as opposed to our patients who were grouped according to the severity of obstruction (FEV_1). We agree with the results of other studies^{20,29} that attribute the greater distance covered by trained patients to desensitization to dyspnea as a benefit of IMT. No patient had followed any exercise program specific to the lower limbs. Patients were accustomed to inactivity, and when the sensation of dyspnea decreased they showed greater mobility, resuming activities that they had abandoned and experiencing emotional improvement along with better control of their disease. Consequently, patients have been able to make greater efforts, demonstrating better functional status during exercise. We think that improvement in dyspnea has bettered the physical and mental status of patients, even if there has been no true stimulus for training the lower limbs. There were no changes, however, in Borg scale scores after the bicycle or walking exercises. We infer that trained patients tolerated the walking exercise better

since, as indicated in the study by Lisboa et al,²⁰ there were no changes in dyspnea intensity for more substantial effort.

The total dyspnea score was significantly greater in the trained group, indicating a decrease in dyspnea. This result agrees with that of the study of Lisboa et al,²⁰ since the trained patients were able to make greater efforts and perform harder tasks than they were before IMT and were able to carry out activities faster without dyspnea. There are significant correlations in this study between changes in the P_{max} and the components of the TDI.^{2,30} This supports the concept that an increase in the strength of inspiratory muscles can ameliorate dyspnea. Harver et al² have shown that targeted IMT results in significant increases in respiratory muscle function and significant reductions in dyspnea.

After training, patients experienced an important improvement in their HRQL. Each category increased more than 1 point, and the total quality-of-life score was 5.9 points. Guyatt et al³¹ thought that an improvement of at least 4 points in the total quality-of-life score, consisting of four categories, was necessary for subjective improvement in quality of life. Only two studies^{23,32} have assessed the clinical consequences of IMT with a quality-of-life test, although both yielded negative results. One of the studies²³ documented no response to training when a generic quality-of-life measure relating to overall health status was employed. The other study³² used a COPD-specific measure of quality of life (the CRQ) in the clinical assessment of an IMT protocol but did not control the ventilatory pattern. In our study, the CI value in group T suggested that the smallest treatment effect exceeded the MCID (0.5 points) for the categories of fatigue, emotion, and mastery. For dyspnea, the CI value was close to, but did not achieve, clinical significance (MCID), although statistical significance was noted. The treatment effects on fatigue, emotion, and mastery were statistically and clinically significant. We have not found studies that evaluate the clinical effects of IMT with a specific quality-of-life test for COPD patients. However, there are two published studies^{33,34} that include IMT as a component of a pulmonary rehabilitation program, evaluating the clinical response with a specific quality-of-life test. The first study³³ introduced relaxation exercises and pursed-lip breathing into their program, achieving clinically significant differences between the treatment and control groups in regard to dyspnea and mastery. The other was a randomized study³⁴ that lasted 18 months and showed improvements in quality of life by combining exercises for the extremities and IMT. This study first showed that home rehabilitation improved quality of life, as assessed by

a validated questionnaire. This group had previously demonstrated³⁵ that the questions related to the categories of fatigue, emotion, and mastery from the CRQ are reproducible and valid for the patient with severe COPD. However, the dyspnea category showed low and unreliable internal consistency. For this reason, the authors³⁵ suggested that the items of the dyspnea category were less reliable and should not be included in the overall CRQ score in comparative research. Nevertheless, scoring the items of dyspnea separately may be useful for the evaluation of the effects of intervention in a specific patient. We have assessed the four categories separately.

The American Association for Cardiovascular and Pulmonary Rehabilitation,³⁶ the American Thoracic Society,⁹ and other general reviews on rehabilitation in COPD³³ have recognized that there is scarce scientific evidence to support the use of IMT as a routine component in treatment programs for patients with COPD, and that such treatment should be administered on an individual basis. Nevertheless, our own data, along with those of other studies,^{20,24} have shown improvements in dyspnea and exercise tolerance with the use of IMT. In addition, we have demonstrated improvement in quality of life.

Since the training lasted for 6 months, it was decided to use an incentive flowmeter (target-flow) training device rather than a pressure threshold training device. The flowmeter is useful both to control the depth of inspirations (target-flow) and to encourage the patient (incentive) to perform them by means of visual feedback. Moreover, the incentive flowmeter is disposable material and is less expensive than a manometer in which respiratory frequency also must be controlled.

According to the recommendation of the American Association for Cardiovascular and Pulmonary Rehabilitation committee,³⁶ we have included the following three basic outcome measures in the evaluation of IMT in the COPD patient: dyspnea rating, functional status, and HRQL. Our results indicate that IMT can be a useful part of pulmonary rehabilitation for COPD patients. Further study is required to determine which patients are most likely to benefit from this training modality in regard to HRQL.

REFERENCES

- 1 Killian KJ, Jones NL. Respiratory muscles and dyspnea. *Clin Chest Med* 1988; 9:237-248
- 2 Harver A, Mahler DA, Daubenspeck JA. Targeted inspiratory muscle training improves respiratory muscle function and reduces dyspnea in patients with chronic obstructive pulmonary disease. *Ann Intern Med* 1989; 111:117-124
- 3 Rochester DF. The diaphragm in COPD: better than expected, but not good enough. *N Engl J Med* 1991; 325:961-962
- 4 Dekhuijzen R, Folgering H, van Herwaarden CLA, et al. Target-flow inspiratory muscle training during pulmonary rehabilitation in patients with COPD. *Chest* 1991; 99:128-133
- 5 Smith K, Cook D, Guyatt GH, et al. Respiratory muscle training in chronic airflow limitation: a meta-analysis. *Am Rev Respir Dis* 1992; 145:533-539
- 6 Belman MJ, Shadmehr R. Targeted resistive ventilatory muscle training in chronic obstructive pulmonary disease. *J Appl Physiol* 1988; 65:2726-2735
- 7 Goldstein R, DeRosie J, Long S, et al. Applicability of a threshold loading device for inspiratory muscle testing and training in patients with COPD. *Chest* 1989; 96:564-571
- 8 Guyatt GH, Berman LB, Townsend M, et al. A measure of quality of life for clinical trials in chronic lung disease. *Thorax* 1987; 42:773-778
- 9 American Thoracic Society. Standards for the diagnosis and care of patients with chronic obstructive pulmonary disease (COPD) and asthma. *Am J Respir Crit Care Med* 1995; 152:S77-S121
- 10 Borg GAV. Physiological basis of perceived exertion. *Med Sci Sports Exerc* 1982; 14:377-378
- 11 Morris JF, Koski A, Johnson LC. Spirometric standards for healthy nonsmoking adults. *Am Rev Respir Dis* 1971; 103:57-67
- 12 Martyn J, Moreno R, Pare P, et al. Measurement of inspiratory muscle performance with incremental threshold loading. *Am Rev Respir Dis* 1987; 135:919-923
- 13 Black LF, Hyatt RE. Maximal respiratory pressures: normal values and relationship to age and sex. *Am Rev Respir Dis* 1969; 99:696-702
- 14 Singh SJ, Morgan MDL, Scott S, et al. Development of a shuttle walking test of disability in patients with chronic airways obstruction. *Thorax* 1992; 47:1019-1024
- 15 Elias MT, Fernandez J, Toral J, et al. Reproducibility of the shuttle walking test in patients with chronic obstructive pulmonary disease. *Arch Bronconeumol* 1997; 33:64-68
- 16 Mahler DA, Weinberg DH, Wells CK, et al. The measurement of dyspnea: contents, interobserver agreement and physiologic correlates of two new clinical indexes. *Chest* 1984; 85:751-758
- 17 Güell R, Casan P, Sangenis M, et al. Quality of life in patients with chronic respiratory disease: the Spanish version of the Chronic Respiratory Questionnaire (CRQ). *Eur Respir J* 1998; 11:55-60
- 18 Jaeschke R, Singer J, Guyatt GH. Measurement of health status: ascertaining the minimum clinically important difference. *Control Clin Trials* 1989; 10:407-415
- 19 Lacasse Y, Wong E, Guyatt GH, et al. Meta-analysis of respiratory rehabilitation in chronic obstructive pulmonary disease. *Lancet* 1996; 348:1115-1119
- 20 Lisboa C, Villafranca C, Leiva A, et al. Inspiratory muscle training in chronic airflow limitation: effect on exercise performance. *Eur Respir J* 1997; 10:537-542
- 21 Berry MJ, Adair EN, Sevensky KS, et al. Inspiratory muscle training and whole-body reconditioning in chronic obstructive pulmonary disease: a controlled randomized trial. *Am J Respir Crit Care Med* 1996; 153:1812-1816
- 22 Clanton TL, Ameredes BT, Thomson DB, et al. Sustainable inspiratory pressures over varying flows, volume, and duty cycles. *J Appl Physiol* 1990; 69:1875-1882
- 23 Larson JL, Kim MJ, Sharp JT, et al. Inspiratory muscle training with a pressure threshold breathing device in patients

- with chronic obstructive pulmonary disease. *Am Rev Respir Dis* 1988; 138:689–696
- 24 Lucas P, Rodriguez JM, Garcia J, et al. Inspiratory muscle training in chronic obstructive pulmonary disease: impact lung functional and exercise tolerance. *Arch Bronconeumol* 1998; 34:64–70
- 25 Tzelepis GE, Vega DL, Cohen ME. Pressure-flow specificity of inspiratory muscle training. *J Appl Physiol* 1994; 77: 795–801
- 26 Villafranca C, Borzone G, Leiva A, et al. Effect of inspiratory muscle training with an intermediate load on inspiratory power output in COPD. *Eur Respir J* 1998; 11:28–33
- 27 Pardy RJ, Reed WD, Belman MJ. Respiratory muscle training. *Clin Chest Med* 1988; 9:287–296
- 28 Flynn MG, Barter CE, Nosworthy JC, et al. Threshold pressure training, breathing pattern, and exercise performance in chronic airflow obstruction. *Chest* 1989; 95:535–540
- 29 Wedzicha JA, Bestall JC, Garrod R, et al. Randomized controlled trial of pulmonary rehabilitation in severe chronic obstructive pulmonary disease patients, stratified with the MRC dyspnea scale. *Eur Respir J* 1998; 12:363–369
- 30 Mahler DA, Harver A. A factor analysis of dyspnea ratings, respiratory muscle strength, and lung function in patients with chronic obstructive pulmonary disease. *Am Rev Respir Dis* 1992; 145:467–470
- 31 Guyatt GH, Berman LB, Townsend BA. Long-term outcome after respiratory rehabilitation. *Can Med Assoc J* 1987; 137:1089–1095
- 32 Guyatt G, Keller J, Singer J, et al. Controlled trial of respiratory muscle training in chronic airflow limitation. *Thorax* 1992; 47:598–602
- 33 Goldstein RS, Gort EH, Stubbing D, et al. Randomised controlled trial of respiratory rehabilitation. *Lancet* 1994; 344:1394–1397
- 34 Wijkstra PJ, Ten Vergert EM, Van Altena R, et al. Long term effects of rehabilitation at home on quality of life and exercise tolerance in patients with chronic obstructive pulmonary disease. *Thorax* 1995; 50:824–828
- 35 Wijkstra PJ, Ten Vergert EM, Van Altena R, et al. Reliability and validity of the chronic respiratory questionnaire (CRQ). *Thorax* 1994; 49:465–467
- 36 American College of Chest Physicians/American Association of Cardiovascular and Pulmonary Rehabilitation. Pulmonary rehabilitation: joint ACCP/AACVPR evidence-based guidelines; ACCP/AACVPR Pulmonary Rehabilitation Guidelines Panel. *Chest* 1997; 112:1363–1396