Threshold Pressure Training, Breathing Pattern, and Exercise Performance in Chronic Airflow Obstruction


The effects of six weeks of threshold pressure inspiratory muscle training (IMT) on inspiratory muscle performance, breathing pattern and exercise performance were studied in eight patients with severe airflow obstruction. The results indicated that IMT improved inspiratory muscle performance but did not affect exercise performance or breathing pattern during maximal exercise. (Chest 1989; 95:535-40)

Im = inspiratory muscle training; 15MWD = 15-minute walking distance; MSBC = maximum sustained breathing capacity; MIP = maximum inspiratory pressure; SIP = maximum pressure sustainable for 15 minutes

In recent years, a number of studies have examined the role of IMT in patients with CAO. Some of these studies have suggested that IMT has a beneficial effect on exercise tolerance, whereas other studies have failed to demonstrate such an effect. One factor influencing the outcome of these various studies has been identified by Belman et al., who showed that patients can be coached to minimize the work of breathing through resistive orifices by reducing inspiratory flow and frequency.

The lack of detailed information regarding breathing pattern in some previous reports makes it difficult to ascertain whether improvement in resistive breathing performance was due to a real enhancement of ventilatory muscle performance or merely secondary to the adoption of a different breathing strategy. Use of a threshold pressure valve in which there is little change of inspiratory pressures over a wide range of flow would be one means of avoiding the possibility of apparent improvement following inspiratory muscle training being simply due to the adoption of a markedly altered breathing pattern.

With a threshold pressure device, the work done across the external resistance (external work) is given by the product of an adjustable threshold pressure and the minute ventilation, and it is largely independent of inspiratory flow. Thus, patients are unable to avoid the work of breathing, and any observed training effect of an increased threshold pressure achieved is likely to be a real increase in work capacity.

Part of our hypothesis was that such a valve would encourage patients to maximize inspiratory flow and minimize inspiratory time during threshold pressure breathing. We designed a pocket-size threshold pressure breathing valve, and used it in a supervised training program designed to increase inspiratory muscle strength and endurance, and to compare these changes with the effects on exercise tolerance.

METHODS

Patients with CAO attending the Department of Thoracic Medicine were invited to take part in an inspiratory muscle training program if they fulfilled the following criteria:

1. Poor exercise tolerance that had led to curtailment of usual activities.
2. The ratio of FEV1 to VC was less than 40 percent, and the FEV1 was less than 50 percent of predicted normal.
3. The baseline FEV1, altered by less than 0.2 L after the administration of 200 μg of aerosol salbutamol.
4. The CAO was demonstrated to be stable as shown by clinical assessment, FEV1/VC measurement and blood gas analysis on at least two occasions four weeks apart.
5. There was no evidence of heart failure clinically on two occasions, and by chest roentgenography on one occasion.
6. Drug treatment had been stable for at least two months and was likely to remain so for at least two months. The intake of corticosteroid drugs, either systemic or topical, did not exclude patients. However, the need for supplemental oxygen therapy and digitalis or diuretic therapy did lead to exclusion.
7. The patient was able to attend the hospital three times weekly for six weeks.
8. The patient was able to cycle on an ergometer.

Informed consent was obtained from each subject, and the project was approved by the Hospital Ethics Committee.

Training Device

The breathing valve design was based on the principles described by Nickerson and Keens but adapted for portable use. It consisted of a plastic plunger which contained lead weights seated over a circular inspiratory port (air intake) in the base of a plastic cylinder (Fig 1). As the patient inspired from the mouthpiece, negative pressure caused the plunger to rise clear of the air intake. Basic physical principles suggested that the threshold pressure would be related to the mass (m) of the plunger and the area (A) of its base by the equation: \[ F = mg/A \] where g is the acceleration due to gravity.

The valve characteristics were studied by connecting it to an electric blower and measuring flow at the air intake by means of a...
pneumotachograph, pressure at the mouthpiece, and plunger mass on electronic scales.

For any given mass there is a threshold opening pressure. At pressures less negative than threshold, the valve is completely closed (this occurs at flows less than 0.05 L/s). Once the inspiratory pressure reaches threshold, the valve opens and thereafter pressure changes by less than 1 cm H2O/L/s for flows up to 3.0 L/s.

Flow volume curves, single breath carbon monoxide transfer, and arterial blood gas tensions were determined for each patient entering the study.

**Training Group**

Eight patients underwent IMT. Spirometry (Godart water-filled spirometer), cycle exercise performance, 12 minute walking distance (MWD), maximum sustained breathing capacity (MSBC), and MIP were determined before and after the six week training period.

Exercise performance was assessed using 8W increments each minute on an electrically-braked cycle ergometer. During the last 30 seconds of each minute of exercise, expiratory flow (pneumotachograph) was electrically integrated to provide minute ventila-

tion. Mixed expired gas was analyzed to determine oxygen uptake and carbon dioxide output. All signals were recorded on a chart recorder.

Duty cycle (T/Ttot) which is the ratio of inspiratory muscle contraction time to total respiratory cycle time, was determined from a flow-time trace at rest and during maximal cycle exercise.

Twelve minute walking distance was measured in an air-conditioned hospital corridor of 60 meters length. The patient was asked to walk as far as he could, resting if he so desired, and was given standardized verbal encouragement.

The MSBC was adapted from the four-minute test described by Freedman (1970). It was determined by asking the patients to breathe humidified air as rapidly and as deeply as possible for three minutes via a low resistance two-way valve and minute ventilation was recorded. Eucapnea was maintained by adding carbon dioxide to the inspired gas at a rate adjusted to maintain the end-tidal level at 5 percent. The patient was informed of the passing of each 30-second interval but was given no other encouragement.

For both the 12 MWD and the MSBC, the patient was allowed a practice trial during the four weeks prior to baseline testing. He then performed the test three times on one day with a minimum of 30 minutes between tests. The best performance was taken as the pretraining value provided there was a matching value within 5 percent; if not, the test was repeated until this situation obtained. The posttraining measurement was made in a similar way but without a practice trial. The mean values and standard deviations for the coefficients of variability of the triplicate baseline values for 12 MWD and MSBC were 2.17 ± 1.77 percent and 3.03 ± 1.83 percent, respectively.

Maximum inspiratory pressure was determined in a body plethysmograph at functional residual capacity (MIP-FRC) and residual volume (MIP-RV). Serial inspiratory efforts (minimum number six) were made at each lung volume until performance had reached a plateau and then began to decline. The highest pressure sustainable for one second was taken as MIP.

Lung volumes (FRC, TLC and RV) were measured in a body plethysmograph in four of the eight patients before and after training.

The patient was then introduced to the training valve. Threshold pressure was initially set at 70 percent of MIP-RV and then decreased by 5 percent intervals until the maximum pressure sustainable for 15 minutes (SIP) was determined. Twenty minutes of rest was allowed between trials.

During the first and last threshold pressure trials of the training period, we continuously measured inspiratory flow and pressure (Pm) at the mouth. Breath by breath flow-time and pressure-time signals and inspiratory flow-volume and pressure-volume curves were recorded on a chart (Fig 2). This allowed measurement of respiratory frequency (f) and inspiratory minute ventilation (Vi). The Ti/Ttot was determined from the pressure-time trace. (Flow-time

**Figure 2.** Pressure-volume and flow-volume curves (left panel) with pressure-time and flow-time curves (right panel) obtained from a patient during threshold pressure training. Ti is inspiratory time; Te, expiratory time.
Table 1 — Baseline Respiratory Function*  

<table>
<thead>
<tr>
<th></th>
<th>Control</th>
<th>Subject</th>
</tr>
</thead>
<tbody>
<tr>
<td>No. patients</td>
<td>8</td>
<td>7</td>
</tr>
<tr>
<td>Age, yr</td>
<td>70 ± 6</td>
<td>67 ± 8</td>
</tr>
<tr>
<td>FEV₁, L</td>
<td>.9 ± .2</td>
<td>.9 ± .3</td>
</tr>
<tr>
<td>VC, L</td>
<td>3.7 ± 1</td>
<td>3.2 ± .8</td>
</tr>
<tr>
<td>TₐCO₂, % pred*</td>
<td>54 ± 19</td>
<td>53 ± 26</td>
</tr>
<tr>
<td>PaO₂, mm Hg</td>
<td>73 ± 8</td>
<td>70 ± 8</td>
</tr>
<tr>
<td>PaCO₂, mm Hg</td>
<td>38 ± 4</td>
<td>38 ± 3</td>
</tr>
</tbody>
</table>

*Mean ± 1 SD. No significant difference between groups (Student's unpaired t-test). FEV₁, forced expiratory volume in 1 second; VC, vital capacity; TₐCO₂, single breath transfer factor for carbon monoxide; % pred, percent of predicted normal; PaO₂, arterial oxygen partial pressure; PaCO₂, arterial carbon dioxide partial pressure.

Measurements underestimate the period of muscle contraction because they do not include the time spent attempting to inspire against a closed valve. Tidal volume (Vt) was calculated from Vt and f. By dividing Vt by Tt mean inspiratory flow was determined. Planimetry of the pressure-volume and pressure-time recordings yielded the external work/minute f Pm/dt and pressure-time integrals Pmdt, respectively. Mean mouth pressure (Pm) during the period of inspiratory flow was calculated from f Pm/dt divided by Vt (use of f Pm/dt divided by Ti would underestimate Pm required to open the valve as it would include the period of inspiratory pressure less than threshold when the valve was closed). All measurements were made from one minute segments of the record in the third, eighth and thirteenth minute of each 15-minute trial.

Trainees attended the hospital three times weekly for six weeks and commenced threshold pressure training at the SIP determined as described above. At each visit up to three patients performed two, 15-minute trials under the close supervision of the same physiotherapist (ICN). Training pressure was increased when the patient had successfully completed two 15-minute sessions. Pressure could be adjusted in 0.8 cm H₂O intervals. A mouth valve and nose peg were used to ensure that all air was inspired at threshold pressure. The patients were given their own valves and asked to train in the same way at home on the other four days of the week.

Control Group

We considered the possibility that increases in MIP observed in our training group were produced by familiarization with the test rather than as a result of IMT. Therefore, we recruited a further seven patients who satisfied the entry criteria and measured MIP on two occasions six weeks apart. They had no IMT and no other intervention.

Table 2 — Maximal Inspiratory Pressure  

<table>
<thead>
<tr>
<th></th>
<th>Week 1</th>
<th>Week 6</th>
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<tbody>
<tr>
<td>IMT (n = 8)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>FEV₁, L</td>
<td>.9 ± .2</td>
<td>.8 ± .3</td>
</tr>
<tr>
<td>VC, L</td>
<td>3.7 ± 1</td>
<td>3.6 ± .9</td>
</tr>
<tr>
<td>MIP-FRC, cm H₂O</td>
<td>40 ± 19</td>
<td>66 ± 27*</td>
</tr>
<tr>
<td>MIP-RV, cm H₂O</td>
<td>59 ± 27</td>
<td>79 ± 34†</td>
</tr>
<tr>
<td>Control (n = 7)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>FEV₁, L</td>
<td>.9 ± .3</td>
<td>.9 ± .3</td>
</tr>
<tr>
<td>VC, L</td>
<td>3.2 ± .8</td>
<td>3.2 ± 1</td>
</tr>
<tr>
<td>MIP-FRC, cm H₂O</td>
<td>34 ± 16</td>
<td>37 ± 17</td>
</tr>
<tr>
<td>MIP-RV, cm H₂O</td>
<td>45 ± 16</td>
<td>45 ± 14</td>
</tr>
</tbody>
</table>

* p < 0.005; †p < 0.025.

Table 3 — Maximal Ventilation and Exercise*  

<table>
<thead>
<tr>
<th></th>
<th>Before IMT</th>
<th>After IMT</th>
</tr>
</thead>
<tbody>
<tr>
<td>Vₑmax, L/min</td>
<td>33 ± 9</td>
<td>34 ± 10</td>
</tr>
<tr>
<td>Vₒmax, L/min</td>
<td>.8 ± .3</td>
<td>.8 ± .2</td>
</tr>
<tr>
<td>Wₑmax, W</td>
<td>45 ± 16</td>
<td>49 ± 12</td>
</tr>
<tr>
<td>12 MWD, M</td>
<td>906 ± 257</td>
<td>905 ± 268</td>
</tr>
<tr>
<td>MSBC, L/min</td>
<td>33 ± 10</td>
<td>32 ± 9</td>
</tr>
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</table>

*No significant difference before and after training in any variable (Student's paired t-test).

RESULTS

Compliance with Training Program

Nine patients enrolled in the training program. One patient attended training on an infrequent basis (less than two out of three visits) and was withdrawn from the program. The remaining eight patients completed the full number of visits. Patients were not asked to keep records of their training at home, but our impression from the supervised visits was that motivation was high and domestic compliance good.

Baseline Data

All results are analyzed by Student's t-test and presented as the mean ± SD. Baseline pulmonary function data on eight trainees and seven control patients are presented in Table 1. There was no significant difference between the two groups.

The FEV₁ and VC did not significantly alter over the training period in either the IMT or control group (Table 2).

Exercise Performance

There was no significant change in maximum ventilation (Vₑ max), oxygen uptake (Vₒ max) or workload (Wₑ max) during incremental cycle exercise and no significant change in 12 MWD or MSBC (Table 3). No patient reached 100 percent of predicted maximum heart rate; two reached 90 percent, and the other six attained less than 80 percent of predicted maximum rate. They did not appear limited by cardiac factors.

Measurements during Threshold Breathing

Figure 3 shows the relationship of mean inspiratory mouth pressure Pm (cm H₂O) to plunger mass (g). The regression line for all data before and after training is given by \( P_m = 0.31 m + 0.6 \) (r = .997, standard error of estimate SEE = 1.3) and is very close to that obtained under steady state flow conditions using a blower motor (p = .31 m + 0.8, r = .999, SEE = 1.2) and to that predicted by physics, \( p = 0.32 m \).

We used \( P = 0.31 m \) to estimate pressure and found it to be 99.3 ± 5.7 percent of measured Pm determined by planimetry. The SIP before training was significantly correlated with MIP-FRC (r = .667, p < .05) and with MIP-RV (Fig 4, r = .89, p < .01).
trated in Figure 5. Duty cycle during threshold breathing was initially quite long (0.54 ± 0.10) but shortened after six weeks of training (0.42 ± 0.11, p < 0.005). Mean inspiratory flow (VT/Ti) during threshold pressure breathing increased from 0.40 ± 0.15 L/s before training to 0.53 ± 0.21 L/s after training (p < 0.05). Resting Ti/Tot was shorter after training (0.36 ± 0.06) than before (0.44 ± 0.06, p < 0.005) but resting VT/Ti was not significantly altered. None of these breathing pattern indices measured during incremental cycle exercise was significantly different after six weeks IMT than before it.

Static Inspiratory Pressure

The IMT produced substantial increases in MIP-FRC (p < 0.005) and MIP-RV (p < 0.025). In the control group, no significant change in MIP was observed over six weeks (Table 2). Patients who underwent IMT had significantly higher MIP-FRC and MIP-RV after training than those who did not (p < 0.05).

Lung Volumes

The FRC, TLC and RV were measured in four of the eight patients, and no significant change after training was detected. In two patients, all three lung volumes were higher after training, while in one, all three volumes were lower.

DISCUSSION

Sonne and Davis\(^5\) found that resistive inspiratory training led to improved maximal exercise ventilation, oxygen uptake and work rate on a cycle ergometer, with no change in a sham training group. Jones et al.\(^6\) using a very similar program, with the addition of a third group who undertook simple physical exercises, found that all three groups improved equally.

Many of these studies\(^7\) used inspiratory muscle training methods which left it open to the subjects to adopt an altered breathing strategy, and thereby avoid doing increased respiratory muscle work. The predictable performance of our threshold pressure valve allowed the prescription of a known inspiratory pres-

**Table 4—Breathing Pattern During IMT**

<table>
<thead>
<tr>
<th></th>
<th>Week 1</th>
<th>Week 6</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pm, cm H(_2)O</td>
<td>23 ± 14</td>
<td>37 ± 15(^\dagger)</td>
</tr>
<tr>
<td>fPm.dV Joule/min</td>
<td>28 ± 20</td>
<td>47 ± 21(^\dagger)</td>
</tr>
<tr>
<td>fPm.dt, cm H(_2)O s/min</td>
<td>544 ± 296</td>
<td>627 ± 293(^\ddagger)</td>
</tr>
<tr>
<td>f, breaths/min</td>
<td>18 ± 7</td>
<td>19 ± 6</td>
</tr>
<tr>
<td>Vt, L/min</td>
<td>13 ± 3</td>
<td>14 ± 3</td>
</tr>
</tbody>
</table>

\(^*\) Pm, mean mouth pressure; fPm.dV, integral of mouth pressure against inspired volume (external work); fPm.dt, integral of mouth pressure against time; f, frequency of breathing; Vt, inspired minute ventilation.

\(^\dagger\) p < 0.001.

\(^\ddagger\) p < 0.05.
The work done in decompressing inspired air across the valve (external work) could be altered only by the patient changing his minute ventilation. Minute ventilation during IMT in week 6 was no different from that in week 1. Pardy and Leith, reviewing the principles of skeletal muscle training as applied to the inspiratory muscles, noted that isocapnic hyperpnea is the form of training which most closely resembles exercise ventilation. It can be described as endurance training for high speed muscle changes at short length. However, this type of training is cumbersome and was found to be no more effective than intermittent positive pressure breathing. Our subjects trained with threshold pressure breathing which is a form of combined strength and endurance training, and which can be practiced at home.

The training led to decreased Ti/Tot during resting breathing and during threshold pressure breathing. Reduction in Ti/Tot may be beneficial in terms of diaphragmatic blood flow, leading to increase in oxygen supply to these important muscles of inspiration. If pressure-time integral is a better predictor of \( \dot{V}O_2 \) resp than external work being done, then a shortening of Ti/Tot might reduce \( \dot{V}O_2 \) resp. Dyspnea has been found to correlate with mouth pressure and inspiratory time during breathing against both resistive and elastic loads, and so a shorter Ti/Tot at a given Pm might reduce dyspnea.

Not only did Ti/Tot decrease during the course of threshold breathing training, but this training led to substantial improvements in maximum inspiratory pressure, training pressure, and the work and pressure-time integral of threshold breathing. These improvements leave little doubt that IMT did train the inspiratory muscles of our patients.

We cannot exclude the possibility of improvement in inspiratory muscle performance being due to placebo effects of the training device or frequent contact with a physiotherapist. Our control patients were not submitted to sham training or to training on a very low threshold load, and only restricted measurements were made in this group. Furthermore, they were recruited after increases were observed in the training group, and we may have been biased against detecting improvement in the control subjects. However, the trainees showed no improvement in 12 MWD, which is subject to motivational factors, nor in cycle exercise performance, which may also improve due to placebo factors.

Furthermore, it is unlikely that a decrease in lung volume accounted for the increase in inspiratory pressures in the training group. No consistent trend was observed in the four patients in whom lung volumes were measured.

Despite marked improvement in inspiratory performance, we observed no change in MSBC, maximum ventilation, or oxygen uptake during exercise, or in overall exercise performance as assessed by 12 MWD. The failure of the training-induced reduction of Ti/Tot to occur during exercise may well be relevant to the absence of improved exercise tolerance. The rationale for supposing that IMT might lead to an improved exercise performance is based on the assumption that minute ventilation and breathing pattern during exercise are going to be appropriately altered by inspiratory muscle training. A reduced inspiratory time, achieved by increased inspiratory flow, would allow greater time for expiration, and therefore allow an increased exercise maximum ventilation to be achieved without any change in lung mechanics.

Dillard et al. have shown that maximal exercise ventilation in patients with CAO correlates with peak inspiratory flow rate as well as the FEV. It is not clear why the training induced decrease of Ti/Tot in our study was not translated into improved exercise performance. Possibly threshold pressure breathing is just not specific enough with regard to exercise ventilation.

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**Figure 5.** Left column: average respiratory cycle with Vt/Ti represented by the slope of the ascending limb of the schematic spirogram and end-expiratory lung volume taken as zero. Continuous lines; before training; dashed lines; after training; Bars, one SD from the mean. Right column: Ti/Tot before (open circles) and after (closed circles) six weeks IMT. Top row; during threshold breathing; middle row; at rest; Bottom row; during maximal cycle exercise. Bars indicate one SD either side of the mean.
to induce a useful training effect.

It may be that inspiratory muscle fatigue was not a significant limitation in our patients. Pardy et al. concluded that specific training of the inspiratory muscles is usually associated with improved exercise performance only in those who demonstrate electromyographic changes heralding inspiratory muscle fatigue during exercise. The interrelationships between the presence or absence of fatigue, and the degree to which Ti/Ttot might be reduced after training, is unknown. Furthermore we did not conduct nutritional studies in our patients and it is conceivable that this may have influenced our results.

In our study, SIP was a slightly smaller proportion of MIP-FRC than that found in normal subjects by Nickerson and Keens, though our results are closer to the values predicted by Clanton et al. who reported an increase in MIP-FRC and endurance time at 65 percent of MIP-FRC after ten weeks threshold IMT in normal subjects. In our subjects, there was, in fact, a training effect in the appropriate direction, as shown by the reduction of Ti/Ttot. Therefore, it is unlikely that the SIPs were just too small.

We did not measure blood gas values during our threshold pressure breathing, and therefore, we cannot say whether patients allowed arterial PCO₂ to rise in the face of increased CO₂ production, in an effort to attenuate the increased external work. This has been a matter of concern to previous authors. Although there was no overall change in mean ventilation for the group, there were individual increases or decreases in ventilation, possibly reflecting variable tolerance of hypercapnoea.

In conclusion, this study clearly demonstrates that a threshold pressure device can be used to improve inspiratory muscle performance. However, no improvement in any index of exercise performance has been observed in this group of patients with severe CAO.

REFERENCES
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