The Effect of Comprehensive Outpatient Pulmonary Rehabilitation on Dyspnea*

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To evaluate the effect of outpatient pulmonary rehabilitation (OPR) on dyspnea, we measured this symptom using a visual analogue scale during graded treadmill exercise testing and with baseline and transitional dyspnea indices (TDI). The latter measure overall dyspnea in three spheres: functional impairment, magnitude of task, and magnitude of effort. Twenty patients with COPD referred for OPR were randomly assigned to either a treatment group (T, n=10), with dyspnea evaluated at baseline then shortly following a 6-week OPR program, or a control group (C, n=10), with dyspnea evaluated at baseline then following a 6-week waiting period. No significant change in maximal exercise performance from baseline to repeated testing was observed in either group. Dyspnea at maximum treadmill workload (Dmax), which did not significantly change in C, decreased from 74.4 ± 18.9 percent at baseline to 50.5 ± 23.2 percent post-OPR in T (p=0.006). The Dmax related to minute ventilation (Dmax/VEmax) and oxygen consumption (Dmax/Vo2max) also significantly decreased following OPR. The reduction in exertional dyspnea was apparent by the second minute of exercise. Additionally, TDI focal scores were significantly higher in T than C (2.3 ± 1.06 vs 0.2 ± 1.75 units, p=0.006), indicating decreased overall dyspnea following OPR. These results point to significant improvements in both exertional and clinically assessed dyspnea following OPR.

(Chest 1994; 105:1040-52)

BDI=baseline dyspnea index; C=control group; Dmax=dyspnea at maximum treadmill exercise workload; HRmax=heart rate at maximum workload; OPR=outpatient pulmonary rehabilitation; SaO2=oxygen saturation; SaO2max=oxygen consumption at maximum workload; T=treatment group; TDI=transition dyspnea index; VE=minute ventilation; VEmax=minute ventilation at maximum workload; Vo2=carbon dioxide production; Vo2max=carbon dioxide production at maximum workload; VR=ventilatory reserve; VRmax=ventilatory reserve at maximum workload

Substantial improvement in exercise endurance and quality of life are usual outcomes when patients with COPD undergo comprehensive outpatient pulmonary rehabilitation (OPR).1-3 Since pulmonary function does not improve and traditional aerobic exercise training levels are infrequently reached, the basis for these favorable outcomes remains obscure. One suggestion is that OPR may lead to a decrease in the patient's perception of dyspnea,4 although this has not received much scientific investigation. We evaluated the effect of OPR on dyspnea by measuring this symptom in two ways: with a visual analogue scale during graded treadmill exercise testing to look at exertional dyspnea, and with the baseline and transitional dyspnea indices developed by Mahler et al5 to examine overall dyspnea. One group of patients with COPD was tested before and after OPR while the other group was tested before and after a waiting period.

METHODS

Patients

Patients referred to our OPR program were considered for the study. Inclusion criteria included the following: (1) a clinical diagnosis of moderately severe to severe COPD; (2) a significant exertional dyspnea despite conventional medical therapy; (3) an FEV1 equal to or less than 1.4 L; and (4) the absence of significant, associated medical problems that might interfere with the patient's ability to undergo OPR. Because our oxygen analyzer for treadmill exercise testing was not accurate at oxygen concentrations other than room air, patients requiring continuous, low-flow oxygen therapy prior to the study had to be excluded.

As part of the initial evaluation, all patients had measurements of prebronchodilator and postbronchodilator FEV1, the 12-min walking distance, and the baseline dyspnea index (BDI).5 For the latter, open-ended questions were used to quantify three components of the patient's dyspnea: functional impairment, magnitude of task, and magnitude of effort. The score of each of the three components was summed to give a baseline focal score, which could range from zero to 12, with lower scores indicating greater impairment from dyspnea.

OPR

Between four and eight patients met for 12, 3-h sessions

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1046

Comprehensive Outpatient Pulmonary Rehabilitation (Reardon et al)
over a 6-week period for OPR. The first half of each session included educational activity such as breathing retraining, energy conservation and work simplification, nutritional and medication education, relaxation techniques, panic control, stress management, and symptom control measures. The second half of each session was devoted to physical conditioning, such as upper extremity training with therabands and light weight lifting, stair climbing, treadmill and stationary bicycle exercise, and inspiratory resistive training.

Dyspnea for each strenuous activity was measured using a ten-point Borg category scale. Workloads for the treadmill and stationary bicycle were originally set based on initial performance on exercise testing, with a goal to produce a moderate amount of dyspnea or a maximum heart rate (HR) between 70 and 85 percent of that found on maximal exercise testing. At subsequent exercise sessions, the duration or intensity (or both) was increased by small degrees, when possible, based on subjective and objective data.

**Study Design**

After patients gave informed consent, they were randomly assigned to either a treatment (T) or a control (C) group. Initial evaluation for both groups included a session with the OPR nurse clinician, where optimization of pulmonary therapy was provided and specific instructions were given for unsupervised exercise at home. The type and intensity of this home exercise was determined by the interview and, if available at the time, results from baseline exercise testing. Patients assigned to T then began OPR at the next available 6-week block, while patients assigned to C waited approximately 6 weeks for a subsequent 6-week block. The T patients were studied before and after OPR while C patients were studied before and after the waiting period. Following the initial evaluation with the nurse clinician, maintenance medical and pulmonary therapy, including bronchodilators and steroids, was not changed.

Although patients could not be blinded to study group, they were not specifically informed that the purpose of the investigation was to test the effect of OPR on dyspnea. They were told simply that this was a study evaluating the effectiveness of OPR. The exercise physiologist performing the treadmill exercise testing and the investigators measuring the baseline dyspnea index (BDI) and transition dyspnea index (TDI) were blinded to the study group randomization.

**Exercise Testing With Dyspnea Measurements**

Incremental multistage exercise testing on a treadmill (Quinton-65) was done in all patients initially (baseline study) and again following either OPR in T patients or the waiting period in C patients (repeat study). Prebronchodilator and postbronchodilator spirometry was obtained approximately 30 min before each exercise test. Patients breathed room air during testing. Speed and grade settings were set by the exercise physiologist based on the patient’s estimated physical impairment at baseline evaluation. Two-minute stages were used. Patients were encouraged not to lean on the hand bar during exercise. Testing was terminated when the patient could exercise no longer because of severe shortness of breath or fatigue. Repeated testing speed and grade settings were the same as those used at baseline testing.

Systolic blood pressure was measured at each stage, while 12-lead electrocardiography, HR, and oxygen saturation (SaO₂) using a pulse oximeter were measured continuously. Heart rate in beats per minute was converted to percent of maximum predicted HR using the following formula: maximum HR = 210 – (0.65) X (age in years). Respiratory frequency, tidal volume, and minute ventilation (Ve) were measured with a dry gas meter; oxygen and carbon dioxide in expired air were determined using analyzers (Ametek). Oxygen consumption (Vo₂) and carbon dioxide production (VCO₂) were averaged and recorded every 30 s. Maximum voluntary ventilation (MVV) was estimated by multiplying the postbronchodilator FEV₁ from pre-exercise spirometry by 35; this value was used to calculate the ventilatory reserve (VR) using the following formula: VR = (1 – Ve/MVV) x 100.

Each patient indicated his/her level of dyspnea at rest with the mouthpiece on and at 1-min intervals during the exercise testing by pointing to a vertical, 300-mm visual analogue scale that had both ends anchored. Immediately above the visual analogue scale line was written, “Shortness of breath—As bad as it can be,” while immediately below the line was written, “No shortness of breath.” At repeat testing, patients were not shown their dyspnea ratings from their first study.

Dyspnea scores were converted to percent of line length, with higher percents indicating greater dyspnea. Dyspnea then, could vary from zero (no dyspnea) to 100 percent (greatest possible degree of dyspnea). In addition, dyspnea was related to corresponding Ve (dyspnea/Ve, in percent/L) and Vo₂ (dyspnea/Vo₂, in percent/ml).

**The Transition Dyspnea Index (TDI)**

Following either OPR or the waiting period, changes in the three BDI components of dyspnea, functional impairment, magnitude of task, and magnitude of effort, were quantified using the TDI of Mahler et al. This rate changes in each of these three components using seven-point scales, with −3 representing major deterioration, zero no change, and +3 major improvement. The three components were then summed to give a focal transition score. Thus, a score of −9 indicates greatest deterioration, zero no change, and +9 greatest improvement in overall dyspnea.

**Statistical Analysis**

Unpaired Student’s t tests were used to compare the two groups with respect to baseline patient characteristics, including FEV₁ and BDI, and changes in dyspnea measured by the TDI. Since individual patient time on the treadmill ranged from 2 to 11 min, exercise variables and dyspnea measures could be compared in the two groups without missing value bias only at rest, 1 min, 2 min, and at maximum workload. Analysis of variance for repeated measures (SAS) was used for these analyses. The relationship between changes in dyspnea at maximum treadmill exercise workload (Dmax) to changes in TDI scores was evaluated using simple linear regression using all 20 patients in the analysis. A p value < 0.05 was considered significant.

**Results**

**Patients**

Age, sex, FEV₁, 12-min walking distance, and BDI focal scores of the 20 study patients are given in Table 1. As indicated, all had moderately severe to severe airways obstruction. Although baseline FEV₁ tended to be somewhat lower in C than T patients (0.79 ± 0.18 L vs 0.94 ± 0.32 L), this difference was not statistically significant. Both groups were similar with respect to exercise endurance, as measured by the 12-min walk distance, and overall dyspnea, as measured by the BDI focal score.
Table 1—Patient Characteristics*

<table>
<thead>
<tr>
<th>Patient/Age, yr/Sex</th>
<th>FVC, L (% pred)</th>
<th>FEV₁, L (% pred)</th>
<th>12 MD, cm</th>
<th>BDI Focal</th>
<th>Baseline Dyspnea</th>
</tr>
</thead>
<tbody>
<tr>
<td>Control patients</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1/57/M</td>
<td>2.05 (56)</td>
<td>0.94 (32)</td>
<td>86,910</td>
<td>4</td>
<td></td>
</tr>
<tr>
<td>2/69/F</td>
<td>1.30 (52)</td>
<td>0.59 (30)</td>
<td>102,330</td>
<td>6</td>
<td></td>
</tr>
<tr>
<td>3/6/5/M</td>
<td>2.39 (56)</td>
<td>0.65 (19)</td>
<td>54,120</td>
<td>5</td>
<td></td>
</tr>
<tr>
<td>4/56/M</td>
<td>2.19 (48)</td>
<td>0.48 (13)</td>
<td>48,000</td>
<td>2</td>
<td></td>
</tr>
<tr>
<td>5/66/M</td>
<td>2.16 (55)</td>
<td>0.83 (27)</td>
<td>83,640</td>
<td>5</td>
<td></td>
</tr>
<tr>
<td>6/71/M</td>
<td>3.39 (79)</td>
<td>0.99 (27)</td>
<td>88,560</td>
<td>7</td>
<td></td>
</tr>
<tr>
<td>7/75/F</td>
<td>1.78 (75)</td>
<td>0.78 (43)</td>
<td>78,720</td>
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</tr>
<tr>
<td>8/62/F</td>
<td>2.24 (84)</td>
<td>0.95 (45)</td>
<td>66,420</td>
<td>9</td>
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<tr>
<td>9/60/F</td>
<td>2.70 (85)</td>
<td>0.73 (32)</td>
<td>60,000</td>
<td>6</td>
<td></td>
</tr>
<tr>
<td>10/75/F</td>
<td>1.41 (77)</td>
<td>0.95 (66)</td>
<td>41,820</td>
<td>9</td>
<td></td>
</tr>
<tr>
<td>Group: 66.1 5M/5F</td>
<td>2.16 (67)</td>
<td>0.79 (33)</td>
<td>76,060</td>
<td>5.5</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Treatment patients</th>
<th></th>
<th></th>
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<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>1/55/M</td>
<td>2.33 (68)</td>
<td>1.26 (45)</td>
<td>41,820</td>
<td>2</td>
<td></td>
</tr>
<tr>
<td>2/62/M</td>
<td>2.47 (53)</td>
<td>0.76 (21)</td>
<td>68,880</td>
<td>6</td>
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</tr>
<tr>
<td>3/72/M</td>
<td>3.37 (77)</td>
<td>1.24 (37)</td>
<td>95,940</td>
<td>5</td>
<td></td>
</tr>
<tr>
<td>4/75/F</td>
<td>3.01 (97)</td>
<td>1.15 (50)</td>
<td>63,960</td>
<td>7</td>
<td></td>
</tr>
<tr>
<td>5/66/M</td>
<td>2.57 (59)</td>
<td>1.05 (31)</td>
<td>77,490</td>
<td>6</td>
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</tr>
<tr>
<td>6/66/F</td>
<td>2.25 (64)</td>
<td>1.35 (48)</td>
<td>73,800</td>
<td>5</td>
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</tr>
<tr>
<td>7/70/M</td>
<td>2.13 (37)</td>
<td>0.87 (30)</td>
<td>54,120</td>
<td>6</td>
<td></td>
</tr>
<tr>
<td>8/70/F</td>
<td>1.43 (57)</td>
<td>0.66 (34)</td>
<td>85,560</td>
<td>7</td>
<td></td>
</tr>
<tr>
<td>9/62/F</td>
<td>2.25 (80)</td>
<td>0.57 (39)</td>
<td>54,120</td>
<td>6</td>
<td></td>
</tr>
<tr>
<td>10/75/F</td>
<td>1.53 (63)</td>
<td>0.44 (24)</td>
<td>72,570</td>
<td>5</td>
<td></td>
</tr>
<tr>
<td>Group: 66.3 5M/5F</td>
<td>2.33 (68)</td>
<td>0.94 (35)</td>
<td>102,120</td>
<td>5.5</td>
<td></td>
</tr>
</tbody>
</table>

*Numbers in parentheses are percent of predicted values. 12 MD = 12-min walk distance; BDI Focal = baseline dyspnea index focal score, which could range from 0 to 12, with greater scores indicating greater impairment from dyspnea.

Baseline Exercise Testing—Pre-exercise and Maximal Workload Values

Pre-exercise values for HR, VE, V̇O₂, VR, SaO₂, and dyspnea for the two groups were not significantly different at baseline testing. Similarly, as Table 2 indicates, maximum workload values for the exercise variables (HRmax, V̇Emax, V̇O₂max, VRmax, SaO₂max), the dyspnea measures (Dmax, Dmax/ V̇Emax, and Dmax/V̇O₂max), as well as time spent on the treadmill, were not significantly different at baseline testing.

Repeated Exercise Testing—Pre-exercise and Maximal Workload Values and Changes From Baseline

Similar to baseline testing, pre-exercise HR, VE, V̇O₂, VR, SaO₂, and dyspnea were not significantly different in T and C at repeated testing. Maximum workload values for the exercise variables, dyspnea measures, and time on the treadmill at repeated testing are given in Table 2. Heart rate at maximum workload (HRmax), V̇Emax, V̇O₂max, VRmax, and SaO₂max at maximum workload were not significantly different in the two groups. In C, the decrease in HRmax from 83.1 ± 13.7 to 75.5 ± 10.7 percent of maximum was the only significant change from baseline. In T, none of the exercise variables was significantly different from baseline. The increase in treadmill time of 2.0 ± 2.1 min from baseline to repeated testing in T was significantly greater than 0.3 ± 1.3 min increase in C (p<0.03).

Maximal workload values for dyspnea (Dmax) in T decreased from 74.4 ± 18.9 percent at baseline to 50.5 ± 23.2 percent at repeated exercise testing (p<0.015). In contrast, the Dmax did not change significantly in C. The baseline to repeat change in Dmax was significantly different between the two groups (−23.9 ± 25.3 percent in T vs 7.4 ± 19.4 percent in C, p<0.006). The Dmax related to corresponding maximum workload values for V̇E

Table 2—Exercise and Dyspnea Variables at Maximum Workload*

<table>
<thead>
<tr>
<th>Time, min</th>
<th>Baseline</th>
<th>Change</th>
<th>Baseline</th>
<th>Change</th>
</tr>
</thead>
<tbody>
<tr>
<td>HRmax, %</td>
<td>83.1 ± 13.7</td>
<td>−7.6 ± 9.9</td>
<td>82.4 ± 9.3</td>
<td>−15.5 ± 7.7</td>
</tr>
<tr>
<td>V̇Emax, L/min</td>
<td>24.2 ± 6.7</td>
<td>2.7 ± 7.0</td>
<td>30.5 ± 6.5</td>
<td>2.0 ± 5.0</td>
</tr>
<tr>
<td>V̇Rmax, %</td>
<td>17.5 ± 19.3</td>
<td>−8.7 ± 16.4</td>
<td>22.6 ± 14.3</td>
<td>−1.1 ± 5.7</td>
</tr>
<tr>
<td>V̇O₂max, ml/min</td>
<td>791 ± 302</td>
<td>74 ± 275</td>
<td>865 ± 194</td>
<td>−1.1 ± 5.7</td>
</tr>
<tr>
<td>SaO₂max, %</td>
<td>83.0 ± 7.9</td>
<td>7.4 ± 19.4</td>
<td>74.4 ± 18.9</td>
<td>−1.1 ± 5.7</td>
</tr>
<tr>
<td>Dmax</td>
<td>72.0 ± 18.4</td>
<td>7.4 ± 19.4</td>
<td>74.4 ± 18.9</td>
<td>−1.1 ± 5.7</td>
</tr>
<tr>
<td>Dmax/V̇Emax, %/L</td>
<td>3.11 ± 0.95</td>
<td>0.02 ± 0.73</td>
<td>2.61 ± 1.05</td>
<td>0.06 ± 0.03</td>
</tr>
<tr>
<td>Dmax/V̇O₂max, %/ml</td>
<td>0.10 ± 0.04</td>
<td>0.02 ± 0.03</td>
<td>0.09 ± 0.04</td>
<td>−0.04 ± 0.03</td>
</tr>
</tbody>
</table>

*Time=time on treadmill to maximum workload; HRmax=heart rate at maximum workload; V̇Emax=minute ventilation at maximum workload; V̇Rmax=ventilatory reserve at maximum workload; V̇O₂max=oxygen consumption at maximum workload; SaO₂max=oxygen saturation at maximum workload; Dmax=dyspnea at maximum workload, expressed as the percent of visual analogue scale line length, with higher scores indicating greater dyspnea; and Post-OPR=post-outpatient pulmonary rehabilitation. In the treatment group, time on the treadmill increased, while Dmax, Dmax/V̇Emax and Dmax/V̇O₂max all decreased following OPR. Decreases in the latter 3 variables were significant when compared to the control group.

†p<0.05.
‡Less than control group, p<0.01.
§p<0.01.
(Dmax/Vemax) and VO2 (Dmax/VO2max) were also reduced following OPR, with Dmax/Vemax decreasing from 2.61 ± 1.05 to 1.64 ± 0.80 percent/L and Dmax/VO2max decreasing from 0.09 ± 0.04 to 0.06 ± 0.03 percent/ml at these times (p=0.012 and 0.002, respectively).

**Exercise Variables and Dyspnea Measures During Exercise Testing**

Baseline and repeated dyspnea measured at each minute of treadmill exercise is shown in Figure 1 for T patients. Inspection of the individual patient dyspnea-time relationships in C reveals no consistent changes from baseline to postwait. Additionally, there were no significant differences in exercise variables or dyspnea measures in the first 2 min of exercise in this group. Substantial improvement in dyspnea, however, is apparent in seven of the ten T patients; in these individuals, baseline and post-OPR dyspnea curves separate early in exercise.

Table 3 outlines baseline and post-OPR exercise variables and dyspnea measures for the 10 T patients at rest, and at 1 and 2 min. Significant reductions in post-OPR VE and VO2 at 1 and 2 min were found despite the fact that treadmill speed and grade settings were identical at baseline and repeated testing. Post-OPR dyspnea was significantly reduced by the first minute, while all three post-OPR dyspnea measures were significantly decreased by 2 min.

**TDI and Its Relationship to Changes in Dmax**

The change in each patient’s overall level of dyspnea was determined with the TDI. The focal score of 2.3 ± 1.06 U in T, which was significantly greater than the 0.20 ± 1.75 U in C (p=0.006),
Table 3—Exercise Variables and Dyspnea Measurements for 7 Patients at Rest, and at 1 and 2 Minutes During Baseline and Post-OPR

<table>
<thead>
<tr>
<th>Time (Min)</th>
<th>Baseline</th>
<th>Post-OPR</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>HR, % of max</td>
<td>55 ± 29</td>
<td>51 ± 9</td>
<td>ns</td>
</tr>
<tr>
<td>VE, L</td>
<td>13.7 ± 3.2</td>
<td>12.0 ± 4.5</td>
<td>ns</td>
</tr>
<tr>
<td>V̇O₂, ml</td>
<td>357 ± 77</td>
<td>286 ± 95</td>
<td>ns</td>
</tr>
<tr>
<td>Dyspnea, %</td>
<td>17.7 ± 19.1</td>
<td>15.6 ± 15.9</td>
<td>ns</td>
</tr>
<tr>
<td>Dyspnea/VE, %/L</td>
<td>1.21 ± 1.36</td>
<td>1.33 ± 1.53</td>
<td>ns</td>
</tr>
<tr>
<td>Dyspnea/V̇O₂, %/ml</td>
<td>0.05 ± 0.06</td>
<td>0.05 ± 0.06</td>
<td>ns</td>
</tr>
</tbody>
</table>

*HR=heart rate; VE=minute ventilation; V̇O₂=oxygen consumption; and post-OPR=post-outpatient rehabilitation. Dyspnea is the percent of line length from a visual analog scale, with higher scores indicating greater dyspnea. p refers to the difference between baseline and post-OPR means.

indicates decreased dyspnea following OPR. The change in Dmax from baseline to repeated testing was inversely related to the change in TDI score over this period, given by the linear regression formula: change in Dmax=3.6-9.5 • (focal TDI score) (r²=0.35, p=0.004). Thus, improvement in exertional dyspnea from baseline to repeated testing was in part related to improvement in overall, clinically assessed dyspnea over this period.

DISCUSSION

The purpose of this investigation was to evaluate the effect of OPR on dyspnea in advanced COPD. One group of patients was studied before and after OPR while a control group was studied before and after a waiting period. Exertional dyspnea during graded treadmill exercise testing was measured with a visual analogue scale, while overall impairment from dyspnea was measured with the baseline and transitional dyspnea indices. A decrease in dyspnea at maximal workload was observed only in the group completing OPR, with Dmax decreasing from 74 percent at baseline to 50 percent at repeated testing. Similar improvement was also seen in Dmax/V̇Emax and Dmax/V̇O₂max.

The effect of pulmonary rehabilitation on exertional dyspnea has been evaluated previously by Strijbos and colleagues, who used a Borg category scale to measure this symptom during progressive cycle ergometry. Thirty patients with COPD were studied before and following 12 weeks of pulmonary rehabilitation. Their results were compared with 15 patients with COPD who served as untreated control subjects. Dyspnea at maximum workload, which was 6.5 ± 1.3 before rehabilitation, decreased significantly to 4.0 ± 1.6 at similar workloads following rehabilitation, while no change in this measurement was seen in the control patients. This reduction in dyspnea, therefore, was roughly similar to that observed in our study.

Interestingly, the reduction in post-OPR Dmax in our patients occurred without a significant improvement in V̇O₂max: although it increased by 89 ml, or 10 percent over baseline, this was not statistically significant. While this may merely reflect the small number of patients studied, the increase in V̇O₂max for the control group, which was 9.5 percent, was almost identical. This underscores the need for a control group in studies such as this. Previous studies have demonstrated that increases in maximum exercise performance with pulmonary rehabilitation are relatively small, and usually less in degree than concomitant increases in exercise endurance.

Not only was dyspnea at maximum workload reduced with OPR, improvement in this symptom became apparent within 1 to 2 min of treadmill exercise. This undoubtedly would have more clinical and functional relevance in patients with exercise capacity severely limited by advanced airways disease. Unlike the situation at maximum workload, significant decreases in post-OPR VE and V̇O₂ were found at 1 and 2 min of treadmill exercise. Since external workloads for each patient were unchanged from baseline to repeated testing, this suggests that OPR improved exercise efficiency early in graded treadmill exercise.

Although mechanical workload during treadmill testing is difficult to quantitate, the increased time on the treadmill following OPR suggests that maximum workload increased in this group. This improvement, which occurred without a concomitant increase in V̇O₂max, probably also reflects the increased exercise efficiency mentioned above. Since a considerable portion of OPR is dedicated to activities that might improve exercise efficiency, such as breathing retraining, energy conservation, work simplification, inspiratory resistive training, and physical conditioning, improvement in exercise efficiency is not unexpected.

In all likelihood, the improvement in exercise efficiency contributed to the decrease in dyspnea in the OPR patients. However, the decrease in dyspnea
was out of proportion to the decrease in Ve or Vo2, since Dmax/Vemax and Dmax/Vo2max were also reduced. This suggests that the improvement in this symptom reflected more than increased efficiency, and may have been related to changes in such factors as motivation or the perception of dyspnea.

It is certainly possible that increased motivation gained during OPR may have caused patients to underreport their dyspnea in an effort to please the investigator. However, although complete blinding of the study patients could obviously not be accomplished, they were not told the specific purpose of the investigation.

In patients with COPD, dyspnea correlates better with general health status than with the degree of airways obstruction, suggesting that this symptom is complex and probably modulated by nonphysiologic as well as physiologic factors. It has been shown that anxiety, depression, hysteria, degree of social support, grief, anger, frustration, fear, and past-life experiences may all affect its perception. Comprehensive OPR, by emphasizing strategies such as problem solving, support, stress management, relaxation techniques, and panic control, may thereby improve dyspnea by its effect on these psychosocial variables.

Desensitization to dyspnea may occur when patients with COPD are repeatedly exercised in the presence of supportive medical personnel. By repeatedly exercising patients to a moderately-severe level of dyspnea during the supervised, bi-weekly exercise conditioning sessions, we may have partially desensitized them to this emotionally charged, often feared sensation. In support of this concept, Belman and colleagues found a progressive reduction in exercise dyspnea on four near-maximal exercise tests given over a 10-day period. This reduction in dyspnea occurred without concomitant changes in Ve or Vo2, and may have resulted from desensitization to this symptom or to the exercise testing milieu. The latter, by itself, may be an anxiety-raising experience. Since treadmill exercise training was an important component of our OPR program, this desensitizing effect may have been of considerable importance.

In addition to decreased exertional dyspnea on treadmill testing, OPR also led to significant improvement in overall dyspnea as measured by the TDI. This instrument, which has been shown to be responsive to changes in dyspnea resulting from pulmonary rehabilitation and inspiratory muscle training, is far more general in scope than the visual analogue scale that was used to measure exertional dyspnea. Again, the comprehensive nature of OPR might be expected to have a substantial impact on this more global measure of dyspnea.

Interestingly, despite the obvious difference in methodology, changes in Dmax were significantly correlated with changes in overall dyspnea measured by the TDI.

In summary, OPR led to a significant reduction in dyspnea measured during graded treadmill exercise testing and with the TDI. The improvement in Dmax occurred without a concomitant increase in maximal exercise performance. The decrease in exertional dyspnea became apparent early in exercise, and may, in part, have been due to improvement in exercise efficiency at low workloads. Outpatient pulmonary rehabilitation also led to a significant decrease in overall dyspnea, as determined by the TDI. A significant relationship was present between changes in these two widely different measures of dyspnea.

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