Respiratory Muscle Rest Using Nasal BiPAP Ventilation in Patients With Stable Severe COPD*

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To more systematically evaluate the effect of respiratory muscle rest on indices of ventilatory function, nine outpatients with stable, severe COPD were treated with nasal pressure-support ventilation delivered via a nasal ventilatory support system (BiPAP, Respironics, Inc) for 2 h a day for 5 consecutive days. An additional eight control patients were treated with sham-BiPAP. Maximum inspiratory pressure (MIP), maximum expiratory pressure (MEP), maximum voluntary ventilation (MVV), arterial blood gas values, Borg dyspnea score, dyspnea-associated functional impairment scales, and distance walked in 6 min were measured in subjects prior to and following the week-long trial. Nasal BiPAP produced a 66.3±6 percent reduction in peak integrated diaphragmatic electromyographic (EMG) activity. There were no statistically significant changes in MIP, MEP, MVV, arterial pH, PaCO<sub>2</sub>, or PaO<sub>2</sub> or in objective measures of functional impairment from dyspnea in either group after ventilator or sham treatment. However, nasal BiPAP reduced the Borg category score during resting, spontaneous breathing from 2.0±0.4 to 0.7±0.3 (p<0.01) after 5 days of treatment. In contrast, sham BiPAP-treated patients had no change in their dyspnea score, which was 1.8±0.4 and 1.3±0.4 before and after sham treatment, respectively. Nasal BiPAP also increased distance walked in 6 min from 780±155 to 888±151 ft (p<0.01) (23,400±4,650 to 26,840±4,530 cm) (p<0.01), whereas sham-BiPAP had no effect (768±96 and 762±166 ft [23,040±2,880 and 22,860±3,160 cm]) before and after sham treatment, respectively. In conclusion, these results indicate that nasal pressure-support ventilation, delivered via nasal BiPAP, improves exercise capacity and reduces dyspnea over the short term in selected outpatients with stable severe COPD. Whether such short-term improvement can be sustained merits further study.

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**BiPAP=ventilatory support system; EMG=electromyogram; MEP=maximum expiratory pressure; MIP=maximum inspiratory pressure; MVV=maximum voluntary ventilation**

Chronic fatigue of the respiratory muscles has been postulated to contribute to the dyspnea, decreased ventilatory capacity and reduced exercise tolerance of individuals with severe COPD. If this hypothesis is correct, then resting the respiratory muscles of these patients would be expected to ameliorate these abnormalities.

In keeping with such a possibility, resting the respiratory muscles using noninvasive negative or positive pressure ventilation has resulted in improved ventilatory function, respiratory muscle strength, and sense of well-being in several studies of patients with moderate to severe COPD. For example, using negative pressure ventilation, including an iron lung, Cropp and DiMarco demonstrated significant improvement in duration of sustained ventilation, maximum inspiratory pressure (MIP), and reduction in degree of hypercapnia in patients with severe COPD. Several other studies have also demonstrated improvement in ventilatory parameters following ventilatory support in similar patients. In contrast, other investigations using similar protocols have failed to demonstrate improvement in ventilatory function. Several factors may have contributed to these observed discrepancies, including variation in the utility or composition of control groups, the use of subjective rather than objective measures of dyspnea and exercise tolerance, differences in the degree of respiratory muscle rest achieved, and patient selection. For example, while half of these studies used a control group, none of them employed sham or blinded controls. In addition, only two studies measured changes in sensation of dyspnea, exercise tolerance, or sense of well-being using objective scales to assess these parameters. As a result, the relationship between respiratory muscle rest and ventilatory function in patients with COPD remains controversial.

A number of ventilatory devices have been utilized to provide respiratory muscle rest. The nasal ventilatory support system (BiPAP, Respironics, Inc, Murrysville, Pa) employs nasal pressure-support...
as a means of noninvasively delivering positive airway pressure to patients with respiratory failure.\textsuperscript{22-24} This system is small, easy to use, and appears to be better tolerated than negative pressure devices, which are cumbersome and impractical for patient use. Consequently, the nasal BiPAP system may be more amenable for clinical use in the outpatient setting.

The purpose of the present study was twofold. First, using objective clinical measurements, we endeavored to more systematically evaluate the influence of respiratory muscle rest on indices of ventilatory function, including respiratory muscle strength, gas exchange and, in particular, on exercise tolerance, severity of dyspnea, and patient sense of well-being. Second, we sought to determine the utility of a less complex and more practical noninvasive ventilatory device—the nasal BiPAP ventilatory system—for resting the respiratory muscles. We report, herein, the results of a prospective, randomized, sham-controlled trial of respiratory muscle rest on ventilatory function, using nasal BiPAP, in a group of outpatients with stable, severe COPD.

\section*{Methods}

\subsection*{Patient Selection}

Seventeen outpatients with severe COPD were identified from the records of the pulmonary function laboratory at MetroHealth Medical Center. Severe COPD was defined as an FEV\textsubscript{1} less than 50 percent of the predicted value or a ratio of FEV\textsubscript{1} to forced vital capacity less than 55 percent of the predicted value. Patients were in stable condition at the time of study; all had had pulmonary function tests and arterial blood gas measurements performed within approximately the previous 12 months. Patients with congestive heart failure, asthma, lung cancer, thoracic cage abnormalities, prior thoracic surgery, restrictive pulmonary disease, obstructive sleep apnea, degenerative joint disease, or who had experienced an exacerbation of COPD or pulmonary infection within the preceding 6 weeks were excluded. Patients were alternately assigned to the two study groups.

Informed consent was obtained from all patients prior to participation. The protocol was approved by the Committee on Human Investigation at our institution.

\subsection*{Surface Diaphragmatic Electromyogram (EMG)}

To assess the effectiveness of nasal BiPAP in reducing the work of the inspiratory muscles, surface diaphragmatic EMG activity was monitored by placing gel electrodes over the sixth to seventh intercostal space near the right costal margin and recording EMG activity before and after initiation of nasal BiPAP. The EMG signal was amplified using a preamplifier (WPI), filtered (10 to 10,000 Hz), rectified, integrated using a filter (Paynter), and recorded on a four-channel recorder (Gould). Tidal volume and respiratory rate were measured using a pneumotachograph (Fleisch) (±2 cm H\textsubscript{2}O) placed in-line between the nasal mask and ventilator tubing.

\subsection*{Measurement of Physiologic Parameters}

Patients performed a 6-min walk test, which has been shown to be a simple and reproducible index of exercise tolerance.\textsuperscript{25} During the 6-min walk test, patients were asked to walk as far as possible within a 6-min period while breathing room air or their previously prescribed level of home oxygen. They were allowed to stop, rest, and resume walking at their own discretion. Patients were given no encouragement and eye contact was kept minimal. Maximum inspiratory pressure, maximum expiratory pressure (MEP), maximum voluntary ventilation (MVV), and an arterial blood gas determination were also performed. Measurements of MIP and MEP were performed at residual volume and total lung capacity, respectively, according to the method proposed by Black and Hyatt.\textsuperscript{26} using a pressure gauge calibrated between 0 and 150 cm H\textsubscript{2}O pressure (Magnehelic; Dwyer Instruments, Inc). The average value of three efforts was utilized for the MIP and MEP measurement. Maximum voluntary ventilation was measured over 15 s during maximal breathing effort. Arterial blood gas determinations were performed at rest, while patients were in the sitting position and breathing room air or their prescribed level of home oxygen.

\subsection*{Assessment of Dyspnea and Functional Impairment}

To assess severity of dyspnea, we administered the modified Borg category scale,\textsuperscript{27} an instrument that categorizes severity of dyspnea from "0=not at all" to "10=extremely." Patients were shown a reproduction of the modified Borg scale and asked to indicate the category that most closely described their current level of breathlessness at that moment.

To more objectively assess the impact of lung disease on ability to perform activities of daily living, functional impairment from breathlessness was evaluated using three different clinical scales designed for this purpose. First, the Modified Medical Research Council Dyspnea Scale\textsuperscript{28} rates functional impairment associated with breathlessness from "0=not troubled by breathlessness, except with strenuous exercise" to "4=too breathless to leave the house or breathe while dressing or undressing." A second scale, the Oxygen-cost diagram,\textsuperscript{29} is a visual analogue scale that rates breathlessness from "0=breathless while sleeping" to "40=breathless with brisk walking uphill." The third scale, termed the BiPAP "Functional Impairment Scale," is a brief questionnaire devised by us, which rates breathlessness as "none" (1 point), "moderate" (2 points), or "severe" (5 points) for each of several daily tasks, including combing hair, brushing teeth, making the bed, bathing, walking, and shopping, resulting in a total score between 12 (no breathlessness) and 36 (maximal breathlessness). If patients did not perform an activity, they were asked to estimate their anticipated degree of dyspnea. We devised this scale in an attempt to better discriminate breathlessness associated with various routine activities of daily living. Patients were presented facsimiles of these various scales and asked to respond by identifying their current functional level. All dyspnea and functional impairment scales were administered at rest, immediately before initiation of physiologic testing.

\subsection*{Protocol}

To reduce the possibility of a learning effect, all patients executed a practice 6-min walk test and MIP and MEP maneuvers prior to beginning the 5-day study.\textsuperscript{30} Patients were then randomized to receive either nasal BiPAP (nasal BiPAP, model S/T-D; Respironics, Inc, Murrysville, Pa) (nine patients) or sham-BiPAP (eight patients). Nasal BiPAP was initiated in the experimental group using the "spontaneous" mode by slowly increasing inspiratory positive airway pressure until diaphragm EMG activity was reduced to a minimum. This usually required between 15 and 20 cm H\textsubscript{2}O pressure. Expiratory positive airway pressure was maintained at 2 cm H\textsubscript{2}O pressure. Patients were
instructed to keep their mouths closed at all times to avoid air leaks. Patients receiving sham-BiPAP were connected to the BiPAP ventilator with a T piece interposed between the nasal mask and ventilator tubing, with the power turned on, and both inspiratory positive airway pressure and expiratory positive airway pressure adjusted to the minimum of 2 cm H2O pressure. Dead space between the nasal mask and T piece was minimal. Control patients received no special instructions. Patients had no knowledge of the purpose of the study or function of the device. All other conditions and patient interactions were identical in both groups.

Subjects received nasal BiPAP (or sham-BiPAP) for 2 h/d over 5 consecutive days, while seated in a comfortable chair. The optimal regimen for nasal BiPAP is unknown. This regimen was selected because patients indicated they would be willing to use the nasal BiPAP instrument as outpatients for about 2 h/d. We empirically chose 5 consecutive days because this represented a practical time frame for patient participation.

Physiologic parameters and degree of dyspnea and functional impairment were assessed at rest in all patients on the first day of the study prior to beginning the ventilator trial and again 15 min following termination of ventilation on the fifth study day.

All participants completed the entire study protocol without discomfort or adverse effects on respiration or sense of well-being.

Statistical Analysis

Patient demographic information was analyzed using unpaired Student's t tests. Nasal BiPAP vs sham-BiPAP control values obtained at baseline (day 1) were compared using unpaired Student's t tests for the following measurements: arterial pH, PaCO2, PaO2, MIP, MEP, MVV, Borg dyspnea score, distance walked in 6 min, and scales of functional impairment. Values at baseline (day 1) compared with postventilation (day 5) treatment within groups, for the above measurements, as well as for surface diaphragm EMG activity, respiratory rate, and tidal volume, were compared using paired Student's t tests. A Bonferroni correction was employed to correct for assessment of multiple variables in the study. Using the Bonferroni correction, comparisons were taken as statistically significant only if they had a p value less than 0.02.

All results are expressed as the mean ± SEM.

RESULTS

Demographics

The characteristics of the study group are shown in Table 1. There were six women and three men with a mean age of 62 years in the nasal BiPAP group, and five women and three men with a mean age of 68 years in the sham-BiPAP group. Mean FEV1 was 0.76 ± 0.08 L and 0.73 ± 0.06 L for the nasal BiPAP and sham-BiPAP treated groups, respectively.

Diaphragmatic EMG

The peak heights of the integrated EMG signals were averaged over five consecutive breaths before, and again, 1 min after initiation of BiPAP ventilation. For the group as a whole, nasal BiPAP produced a mean reduction in the amplitude of the surface diaphragm EMG activity of 66.3 ± 6 percent. The reduction in surface diaphragmatic EMG activity was maximal within 5 to 10 breaths after initiation of nasal BiPAP in all patients. There were no statistically significant differences in respiratory rate (15.9 ± 1.2 and 16.3 ± 1.8 breaths/min) or tidal volume (0.65 ± 0.10 and 0.92 ± 0.18 L) measured immediately before and after application of nasal BiPAP, respectively, for each parameter. We interpreted the reduction in surface diaphragmatic EMG activity to be an indication that diaphragm muscle activation was being reduced by nasal BiPAP administration.

Pulmonary Function

The results of arterial blood gas measurements at baseline (day 1) and following 5 days of ventilator treatment (day 5) are shown in Figure 1. There were no statistically significant differences in values at baseline between the two groups. No significant change in arterial blood gas measurements was observed in either study group following the week-long ventilator trial.

Values for MIP, MEP, and MVV at baseline and on day 5 of ventilator treatment, for the sham- and nasal BiPAP groups, are shown in Figure 2. There were no statistically significant differences in these measures at baseline or after ventilator treatment in either study group.

Dyspnea and Functional Impairment Scales

Figure 3 shows the severity of dyspnea, as assessed by the modified Borg category scale, in the

| Table 1—Characteristics of the Patients With COPD Who Participated in the Study |
|-----------------|--------|-----------------|-----------------|
| Group          | Age, yr/Sex | FEV1, L (% predicted) | FEV1/FVC, % |
| Nasal BiPAP, n=9 |       |                  |               |
| 1              | 62/F   | 0.95 (50)         | 42             |
| 2              | 59/F   | 0.58 (26)         | 28             |
| 3              | 61/F   | 0.91 (40)         | 34             |
| 4              | 68/F   | 1.01 (54)         | 40             |
| 5              | 62/F   | 0.45 (22)         | 23             |
| 6              | 60/F   | 0.49 (23)         | 32             |
| 7              | 61/M   | 0.71 (21)         | 34             |
| 8              | 57/M   | 0.73 (22)         | 24             |
| 9              | 64/M   | 1.05 (32)         | 36             |
| Mean           | 62     | 0.76 (32)         | 33             |
| Sham-BiPAP, n=8 |       |                  |               |
| 1              | 73/F   | 0.71 (38)         | 42             |
| 2              | 75/F   | 0.65 (27)         | 29             |
| 3              | 71/F   | 0.69 (45)         | 30             |
| 4              | 60/F   | 1.04 (58)         | 47             |
| 5              | 69/F   | 0.95 (53)         | 55             |
| 6              | 63/M   | 0.56 (26)         | 29             |
| 7              | 76/M   | 0.50 (22)         | 29             |
| 8              | 55/M   | 0.77 (23)         | 28             |
| Mean           | 68     | 0.73 (37)         | 36             |
nasal BiPAP and sham-BiPAP groups, before and following termination of ventilator treatment. There was no statistically significant difference between groups at baseline (day 1). Nasal BiPAP produced a reduction in category of dyspnea, as measured by the Borg score, whereas sham-BiPAP had no effect. During resting spontaneous breathing, the Borg category score was reduced by 67 percent by nasal BiPAP treatment (p<0.01; sham-BiPAP produced only a 27 percent reduction, which was not statistically significant.

Following the 5-day ventilator trial, values for the modified medical research council dyspnea scale and oxygen-cost scales in the sham-BiPAP group suggested impairment, whereas in the nasal BiPAP group, values for all functional impairment scales suggested improvement (Table 2). However, these changes were not statistically significant.

Distance Walked

The mean net change in distance walked in 6 min on day 5, compared with the baseline value, for the sham- and nasal BiPAP-treated groups, is shown in Figure 4. There was no statistically significant difference in baseline values between groups. However, all patients receiving nasal BiPAP walked

FIGURE 1. Arterial blood gas measurements at baseline (day 1) and following 5 days (day 5) of sham- and nasal BiPAP treatment. No statistically significant difference was observed in pH, PaCO₂, or PaO₂ in either study group at baseline or following the week-long ventilatory trial.

FIGURE 2. MIP, MEP, and MVV values at baseline (day 1) and following 5 days (day 5) of sham- and nasal BiPAP treatment. No statistically significant difference was observed in MIP, MEP, or MVV in either study group at baseline or following the week-long ventilatory trial.

FIGURE 3. Modified Borg category scale dyspnea score at baseline (day 1) and following 5 days (day 5) of sham- and nasal BiPAP treatment. There was a statistically significant reduction in category of dyspnea in the nasal BiPAP-treated group (p<0.01), but not the sham-BiPAP group, during resting spontaneous breathing (au=arbitrary units).
Further following the week-long ventilator trial, with distance walked in 6 min increasing from 780 ± 155 to 888 ± 151 ft (23,400 ± 4,650 to 26,640 ± 4,530 cm) (p < 0.01). In the control group, mean distance walked averaged 768 ± 96 and 762 ± 106 ft (23,040 ± 2,880 and 22,860 ± 3,180 cm) before and after sham treatment, respectively (p = NS).

**Discussion**

In this study, nasal pressure-support ventilation, delivered via the nasal BiPAP ventilatory system, administered for 2 h/d for 5 consecutive days, significantly reduced severity of dyspnea and improved distance walked in 6 min in outpatients with stable severe COPD.

**Methodologic Considerations**

Several points regarding methodology must be considered in the interpretation of our results.

First, we used surface diaphragmatic EMG activity to demonstrate the adequacy of respiratory muscle rest by the nasal BiPAP system. Measurement of surface diaphragmatic EMG activity may be prone to artifact due to motion of the chest and abdominal wall, and to electrical interference from nearby muscles. An alternative technique for measuring diaphragm muscle contractile activity includes use of transdiaphragmatic balloon pressure catheters. However, use of such catheters is more invasive and can potentially induce air leaks, thus compromising the function of the nasal mask. Our findings of marked reductions in surface diaphragmatic EMG activity suggest that the work of the diaphragm was reduced. Our results are in keeping with those of Ambrosino et al. who showed reductions of up to 53 percent in surface diaphragmatic EMG signals in patients with COPD who received noninvasive positive pressure ventilation.

Second, we employed the 6-min walk test to assess low-intensity exercise tolerance in our patients, which has been shown to objectively measure exercise tolerance in patients with chronic bronchitis. The 6-min walk test may be a better indicator of the level of dyspnea and functional impairment experienced by patients with COPD in their daily lives, compared with other tests, such as cycle ergometry or use of a treadmill, both of which measure high-intensity exercise tolerance.

Finally, we administered nasal BiPAP for 2 h/d for 5 consecutive days. This regimen was chosen to assure optimal patient comfort, compliance, and tolerance. Longer periods of nasal BiPAP utilization, reported in other studies, have met with limited patient acceptance and resulted in a significant patient dropout rate. It is possible that alternative regimens, such as longer sessions or duration of use, might have led to greater improvement in sensation of dyspnea and exercise tolerance. In our study, all patients completed the protocol, suggesting that this regimen was well accepted and potentially suitable for routine clinical use by outpatients with severe COPD.
Comparison With Previous Studies

Previous studies aimed at assessing the association between respiratory muscle rest and ventilatory function in patients with COPD have yielded conflicting results. Cropp and DiMarco,3 in a prospective controlled study, reported improved respiratory muscle strength, endurance, and reduced hypercapnia in outpatients with stable severe COPD who received negative pressure ventilation using either tank or cuirass ventilators for 3 to 6 h/d over 3 consecutive days. Scano et al17 showed a slight, but significant, increase in MIP and a reduction in PaCO2 in patients treated 4 h/d for 7 consecutive days in a tank ventilator. Compared with nasal BiPAP used in the present study, the tank ventilator may have achieved a greater degree of respiratory muscle rest, as evidenced by the reduction in hypercapnia seen in these patients. This may also explain the lack of improvement in respiratory muscle strength observed in our study, compared with the previous investigations. Gutierrez et al19 administered cuirass ventilation for 8 h, once a week, to patients with advanced chronic airflow limitation and demonstrated improvement in MIP and arterial blood gas measurements, as well as in distance walked in 12 min and in quality of life. Gutierrez et al attributed these changes to correction of chronic inspiratory muscle fatigue. These results were supported by Fernandez et al20 who treated patients with chronic respiratory failure for 6 to 8 h/d for 2 consecutive days with a ventilator (Pneumowrap) and demonstrated improvement in gas exchange and respiratory muscle strength. Despite the positive findings from these latter two investigations, a placebo effect could not be excluded, since both studies lacked control groups. Similar results have been shown in several studies that have employed noninvasive positive airway pressure ventilation in patients with severe COPD. Carrey et al11 demonstrated reductions in diaphragmatic EMG activity and improvement in oxygen saturation and PaCO2 in some patients with severe COPD given intermittent positive pressure ventilation for 1 to 2 h. These changes may have resulted from having set the ventilator at a slightly higher rate than the spontaneous respiratory rate of their study patients. This is in contrast to our study, in which patients using the nasal BiPAP system were allowed to breathe at their own spontaneous rate. Finally, Ambrosino et al5 using nasal BiPAP for 2 h/d for 2 consecutive days, found improvement in gas exchange in patients with stable severe COPD. However, the number of patients tested in their study was small, and sham control subjects were not utilized.

In contrast to these observed beneficial effects of noninvasive negative and positive pressure ventilation, Zibrik et al18 found no change in gas exchange, respiratory muscle strength, or exercise duration in patients with severe COPD, using a poncho wrap ventilator for 4 h/d for 4 to 6 months. These results may have been influenced by a significant patient dropout rate and by intolerance of the ventilatory device used in their study. Celli et al17 using a ventilator (Pulmowrap) for 3 to 11 h/d for 3 weeks, were unable to show any increased benefit of respiratory muscle rest therapy over that achieved by a comprehensive pulmonary rehabilitation program. In this study, however, most patients were normocapnic, compared with our patients who exhibited significant hypercapnia. Since Juan et al31 have previously shown that hypercapnia can have deleterious effects on respiratory muscle function, this may partially explain the negative results observed in their study. Lastly, Strumpf et al19 found no improvement in pulmonary function, respiratory muscle strength, gas exchange, exercise endurance, or dyspnea ratings in patients who were administered nasal BiPAP for 6 to 7 h per night for 3 months. Since patients performed the nasal BiPAP protocol at home, these negative results may have been influenced by the lack of supervision and/or by noncompliance with use of the nasal BiPAP instrument.

Several well-characterized scales were administered to our patients to assess the effect of nasal BiPAP treatment on severity of dyspnea and on the ability to perform activities of daily living. These scales provide a means of quantifying the subjective responses of patients with regard to the effects of lung disease on sense of well-being. Nasal BiPAP-treated patients, but not the sham group, showed a trend toward improvement in all functional scales following the week-long ventilator trial. In addition, statistically significant decreases in dyspnea were achieved as measured by the Borg category scale. Our results support the findings of Marino and Braun,5 who reported improvements in dyspnea and functional ability following long-term intermittent mechanical ventilation in patients with respiratory muscle fatigue, and Gutierrez et al5 who found significant improvements in quality-of-life score in patients treated with weekly cuirass ventilation.

Our study did not assess the degree to which the observed improvement in dyspnea and exercise tolerance could be sustained following nasal pressure-support ventilation. Such improvement may be rather short lived. Consequently, our results might reflect short-term improvement related to use of nasal BiPAP and cannot be taken as unequivocal evidence that long-term benefit can be achieved or expected.
from the application of nasal BiPAP, as was employed in our study protocol.

Mechanism of Action

There are several theoretic mechanisms by which nasal BiPAP may have influenced respiratory muscle function.

Nasal BiPAP treatment reduced surface diaphragmatic EMG activity, which has been taken as an indication of inspiratory muscle rest.\(^4\) Therefore, nasal BiPAP may have elicited a beneficial effect by resting chronically fatigued respiratory muscles. However, following nasal BiPAP administration, we observed no statistically significant improvement in respiratory muscle strength, a measurement that would be expected to improve with the resting of fatigued muscles. It is known that some types of muscle fatigue reduce force output primarily during submaximal muscle activation (ie, low-frequency fatigue), and that this type of dysfunction cannot be detected by simple assessment of muscle function during maximal levels of muscle activation, as was done in the present study.\(^1,3,22\) Recent work suggests that low-frequency fatigue may be a clinically important determinant of respiratory muscle function, since the respiratory muscles are normally driven at low excitation frequencies.\(^1,16\) It is possible that our study patients were limited primarily by low-frequency muscle fatigue, which was ameliorated by nasal BiPAP treatment, but was not detected by our methods used to assess respiratory muscle strength (ie, maximal static pressure production).

Previous studies have suggested that respiratory muscle function can be impaired by the presence of significant hypercapnia or hypoxemia.\(^31\) Since we observed no significant changes in arterial \(\text{PaCO}_2\) or \(\text{PaO}_2\) measurements following nasal BiPAP treatment, the observed improvement in dyspnea or exercise tolerance cannot be explained by this mechanism.

Lastly, respiratory muscle afferent neural pathways have been shown to play a role in several aspects of breathing.\(^33\) Furthermore, changes in respiratory muscle afferent neural input may result in delayed effects on respiratory muscle function.\(^34\) Nasal BiPAP may have affected respiratory motor outflow by altering respiratory muscle afferent activity in response to changes in respiratory muscle activation or lung mechanics. Theoretically, such effects could have contributed, at least in the short term, to the reduced Borg score and improved exercise tolerance seen following discontinuation of nasal BiPAP treatment. However, similar long-term effects by this mechanism are only speculative.

CONCLUSIONS

In the present study, a sham control group was employed to reduce placebo effects. Functional impairment, exercise tolerance, and severity of dyspnea were assessed using several different objective scales designed for this purpose in an attempt to lessen inaccuracies associated with subjective reports by patients. In addition, the regimen of nasal BiPAP treatment used was well tolerated by our patients. Our results suggest that, at least in the short term, nasal pressure-support ventilation, delivered via the nasal BiPAP ventilatory system, results in a reduction in severity of dyspnea and improvement in exercise tolerance as assessed by distance walked in 6 min, in selected outpatients with stable severe COPD. Whatever the mechanism, nasal BiPAP treatment appears to be effective and well tolerated, and may prove beneficial and cost-effective for use in selected outpatients with stable severe COPD.

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