ORIGINAL ARTICLE

Six-month Nocturnal Nasal Positive Pressure Ventilation Improves Respiratory Muscle Capacity and Exercise Endurance in Patients with Chronic Hypercapnic Respiratory Failure

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Background/Purpose: This study was designed to investigate the effects of 6 months of nocturnal nasal positive pressure ventilation (NNPPV) on respiratory muscle function and exercise capacity in patients with chronic respiratory failure.

Methods: A prospective, randomized, controlled design was used. Twenty-nine patients with chronic respiratory failure were enrolled and allocated to either the NNPPV (n = 14) or control group (n = 15). Patients in the NNPPV group received bi-level positive pressure ventilation via nasal mask for 6 consecutive months. Arterial blood gas, respiratory muscle assessment and 6-minute walk test (6MWT) were performed before and after the 6-month NNPPV intervention. Respiratory muscle function was assessed using the variables of maximal inspiratory pressure (Pimax), maximal expiratory pressure (Pemax), and maximum voluntary ventilation (MVV).

Results: Subjects in the NNPPV group showed a significant improvement in blood gas exchange and increased 6-minute walk distance (6MWD) compared to baseline and the control group. The 6MWD was significantly increased from 257.1 ± 114.1 to 345.2 ± 109.9 m (34.3%) in the NNPPV group. NNPPV also significantly improved MVV and Pimax relative to baseline. MVV was significantly increased from 19.2 ± 6.5 to 22.3 ± 7.1 L/min (16.1%) in the NNPPV group ($p < 0.05$). Furthermore, there was a significant correlation between the magnitude of MVV improvement and 6MWD change.

Conclusion: The 6-month NNPPV treatment significantly decreased the partial pressure of carbon dioxide and improved daytime respiratory muscle function, thus contributing to exercise-capacity increase in patients with chronic respiratory failure. [*J Formos Med Assoc* 2006;105(6):459–467]

Key Words: chronic respiratory failure, nocturnal nasal positive pressure ventilation, respiratory muscle function, 6-minute walk test

Noninvasive positive pressure ventilation (NPPV) is effective for the treatment of chronic respiratory failure in patients with restrictive ventilatory disorders, particularly those with neuromuscular disease and kyphoscoliosis.[1,2] In most reports, long-term intermittent NPPV was implemented during sleep to avoid interruption of daytime activities, as well as to attenuate sleep-related breathing disturbances in patients with significant underlying respiratory disorders.[3,4] There is limited quantitative data on the physiologic effects of NPPV in patients with severe hypercapnic respiratory failure. In addition, conflicting results have been reported with respect to its efficacy for improving gas exchange, respiratory mechanics and exercise capability.

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It has been demonstrated that short-term NPPV markedly improves sleep efficiency and total sleep time in patients with hypercapnic chronic obstructive pulmonary disease (COPD) without significantly improving gas exchange.[5] The long-term nightly use of noninvasive mechanical ventilation (NIMV) to treat chronic respiratory failure in COPD patients, however, is not widely recommended, partly because of the lack of clear clinical results, and partly because the physiologic mechanisms by which the daily application of NIMV would be helpful in these patients have not yet been clarified.[6]

The exercise capacity of patients with chronic ventilatory failure is markedly reduced.[7] It appears reasonable to suggest that daytime respiratory muscle dysfunction may contribute to exercise impairment. Accessory muscle and diaphragmatic electromyographic activity may be reduced by both negative[8] and positive[9] ventilation in patients with chronic respiratory failure. It could be hypothesized, therefore, that the reduction in energy expenditure at night could result in improved respiratory muscle function during the day. Although many studies have explored the effects of nocturnal nasal positive pressure ventilation (NNPPV) on respiratory muscle function, the benefits proposed for long-term NNPPV, in terms of exercise performance, remain controversial.[10–12]

We investigated the effects of a 6-month course of NNPPV treatment on arterial blood gas tension, respiratory muscle function, and exercise capacity in a prospective, randomized controlled trial in patients with chronic respiratory failure.

Methods

Subjects

Patients with chronic hypercapnic respiratory failure were recruited from the outpatient clinic of Chung Gung Memorial Hospital. The inclusion criteria were: (1) daytime $PaCO_2$ > 45 mmHg, pH 7.30–7.45;[13] (2) pulse oxygen saturation (SpO_2) < 88% for more than 5 consecutive minutes on room air during sleep according to polysomnography study;[8] (3) medically stable; and (4) well motivated. Exclusion criteria were: (1) diagnosis of moderate to severe obstructive sleep apnea, i.e. apnea hypopnea index (AHI) > 20 events/hour;[14] (2) unable to complete the 6-minute walk test (6MWT); (3) unable to tolerate NNPPV or failure to cooperate; and (4) participation in an exercise rehabilitation program. All enrolled patients were assigned a computer-generated random number.

Subjects in the NNPPV group were asked to use bi-level positive pressure (BiPAP) ventilation via nasal mask for at least 6 hours during sleep for 6 consecutive months. The study protocol was approved by the Ethical Review Committee at Chung Gung Memorial Hospital, and informed consent was obtained from all patients prior to study enrollment.

Interventions

Nocturnal ventilation was delivered using the BiPAP system (Respironics Inc, Murrysville, PA, USA) via nasal mask (Respironics Inc). All patients were admitted to the thoracic ward to adjust the inspiratory positive airway pressure (IPAP) and the expiratory positive airway pressure (EPAP). Initially, IPAP was set at 2 cmH_2O and EPAP was set at 2 cmH_2O. IPAP was then increased to 8, 12, 16 and 20 cmH_2O and EPAP was then increased to 4, 6 and 8 cmH_2O. Inspiratory and expiratory pressures were set to achieve daytime $PaCO_2$ < 45mmHg and PaO_2 > 60 mmHg or oxygen saturation (O_2SAT) > 88%. Supplemental oxygen was added to maintain $O_2SAT \geq 88\%$ during NNPPV. Subjects in the NNPPV group were instructed to use nasal ventilation at night during sleep. Compliance with the NNPPV treatment regimen was evaluated by reviewing the subjects' log records and the machine meter. Subjects were followed up monthly at our clinic. Five patients in the NNPPV group withdrew from the study due to intolerance of BiPAP.

Measurements

Patients in the NNPPV group were asked to stop using bronchodilators for 12 hours and then

breathed room air for 4 hours before tests. Baseline data were measured before subjects began the study treatment. These included arterial blood gas tension, respiratory muscle function, and 6-minute walk distance (6MWD). All measurements were then repeated at the end of the 6-month treatment. Arterial blood samples were collected on room air. A Corning 278 blood gas analyzer (Ciba-Corning Diagnostics Co, Walpole, MA, USA) was used to perform arterial blood gas analysis. Maximum voluntary ventilation (MVV) was measured by a portable spirometer (Spiro Analyzer ST 250; Fukuda, Sangyo, Japan) in accordance with the recommendations of the American Thoracic Society.[15] The 12-second MVV was also measured. Mouth occlusion pressures at maximum inspiratory pressure (Pimax) and maximum expiratory pressure (Pemax) were measured using a plethysmograph pulmonary function test system (Erich Jaeger, Hoechberg, Germany). Pimax was measured at residual volume (RV) and Pemax was measured at total lung capacity (TLC) with the subject seated in a hard-backed chair. As soon as the patient commenced inhalation, the shutter closed and the pressure was measured automatically. These procedures were repeated until three measurements with less than 5% variability were recorded, with the highest of these utilized for analysis.

Exercise capacity was measured by 6MWT[16] before and after 6 months of treatment. In order to exclude the learning effect, all patients were asked to complete a practice session of 6MWT 1 week before enrollment in the study. Subjects were encouraged every 30 seconds using one of the two phrases, "You are doing well" and "Keep up the good work". Subjects were allowed to stop and rest during the test, but were instructed to resume walking as soon as they felt able to do so. The longest of the two walk distances was used in the analysis. The test was performed without oxygen supplementation. Modified Borg scales were evaluated at rest and immediately after exercise, and expressed as RBorg and ExBorg, respectively.[17] Subjects were connected to a pulse oximeter (3301; BCI International Co, Waukesha, WI, USA) for monitoring of O_2SAT. O_2SAT was recorded and printed out every 6 seconds at rest and during exercise. Both resting oxygen saturation (RO_2SAT) and exercise oxygen saturation (ExO_2SAT) were determined by the pulse oximeter.

Statistical analysis

SPSS version 10.0 software (SPSS Inc, Chicago, IL, USA) was used for statistical analysis. The results are presented as mean ± SD. The Wilcoxon signed rank test was used to determine within-group differences before and after treatment. The Mann-Whitney rank sum test was used to assess the difference between groups, with the Spearman rank correlation coefficient used to examine the relationship between the changes in walking distance and respiratory muscle function. A p value of less than 0.05 was considered to be statistically significant for all tests.

Results

A total of 37 patients were enrolled and allocated randomly to either the control (standard treatment; n = 18) or the NNPPV groups (standard treatment plus NNPPV; n = 19) during the period from June 2001 through July 2003, inclusively. Four subjects in the NNPPV group were unable to tolerate the treatment within 3 months of commencement and were excluded from the data analysis. One patient in the NNPPV group and three controls died during the 6-month intervention period, leaving respective patient completion totals of 14 and 15. No significant between-group differences were found on comparing age, gender distribution, body mass and AHI, disease character, and home oxygen usage (Table 1).

For the NNPPV group, peak inspiratory pressure was 15.8 ± 0.6 cmH_2O, and positive expiratory pressure was 4.5 ± 0.4 cmH_2O. Treatment compliance was confirmed by duration of use as recorded by the ventilator. The mean nightly duration of use was 6.8 ± 0.6 hours. There was a strong correlation between patient and machine records (r = 0.88), with the former exceeding the latter by about 12%.

Table 1. Demographic, clinical and respiratory related characteristics of patients in the control and nocturnal nasal positive pressure ventilation (NNPPV) groups

	Control (n = 15)	NNPPV (n = 14)	p
Age (yr)	65.6 ± 10.0	58.9 ± 12.7	0.1904
Gender (M:F)	11:4	8:6	0.3590
Weight (kg)	54.5 ± 12.8	50.1 ± 14.1	0.3049
Height (cm)	155.2 ± 9.7	151.4 ± 12.4	0.4712
BMI (kg/m^2)	22.6 ± 5.5	21.2 ± 4.3	0.6625
Parameters during sleep study			
Desaturation, minimum O_2SAT (%)	71.1 ± 10.9	71.1 ± 11.3	0.9478
ODI (%)	23.3 ± 28.0	17.7 ± 20.3	0.8613
AHI (%)	9.6 ± 7.0	9.0 ± 9.2	0.4321
Patients using oxygen at home, n (%)	8 (53.3)	7 (50.0)	0.5730
Diagnosis related to respiratory function, n (%)			
COPD	11 (73.3)	10 (71.4)	
Pneumoconiosis	2 (13.3)	1 (7.1)	0.7610
Scoliosis	2 (13.3)	3 (21.4)	
Baseline ABG analysis			
pH	7.41 ± 0.07	7.40 ± 0.05	0.7328
PaO_2 (mmHg)	57.85 ± 9.40	54.31 ± 9.01	0.3114
$PaCO_2$ (mmHg)	52.65 ± 4.02	56.26 ± 7.78	0.1237
HCO_3^- (mmol/L)	33.41 ± 4.01	34.95 ± 5.58	0.3974
Borg scale			
RBorg	2.7 ± 0.9	3.2 ± 1.5	0.2928
ExBorg	5.8 ± 1.2	5.8 ± 1.5	0.9774
Respiratory parameters			
Pimax (Kpa)	4.68 ± 1.79	4.00 ± 1.44	0.2738
Pemax (Kpa)	7.40 ± 2.27	6.11 ± 1.48	0.0846
MVV (L/min)	18.07 ± 5.68	19.16 ± 6.45	0.6370

BMI = body mass index; O_2SAT = oxygen saturation; ODI = oxygen desaturation index (frequency of 4% < baseline per hour); AHI = apnea hypopnea index (events per hour); COPD = chronic obstructive pulmonary disease; ABG = arterial blood gas; PaO_2 = partial pressure of arterial oxygen; $PaCO_2$ = partial pressure of arterial carbon dioxide; HCO_3^- = arterial bicarbonate; RBorg = resting Borg score; ExBorg = Borg score at the end of the 6-minute walk test; Pimax = maximal inspiratory pressure; Pemax = maximal expiratory pressure; MVV = maximum voluntary ventilation.

Arterial blood gas tension at baseline and after completion of the 6-month study are shown in Table 2. There were no significant between-group differences comparing the baseline data for pH, PaO_2, $PaCO_2$, and HCO_3^-. NNPPV therapy showed a significant improvement in daytime blood gas parameters, with a significant increase in PaO_2 (p = 0.013) and a significant decrease in $PaCO_2$, HCO_3^- and BE (p = 0.001, p = 0.001 and p = 0.002, respectively). Further, the magnitudes of the $PaCO_2$ and HCO_3^- decreases were more significant for the NNPPV group than for the controls.

At the end of the 6-month treatment period, MVV was significantly improved in the NNPPV group (from 19.16 ± 6.45 to 22.33 ± 7.11 L/min; $p < 0.05$, n = 14), but not in the control group (from 18.07 ± 5.68 to 17.04 ± 6.17 L/min; $p > 0.05$, n = 15) (Figure 1). Further, Pimax was significantly increased in the NNPPV group (from 4.00 ± 1.44 to 4.83 ± 1.88 Kpa; $p < 0.05$, n = 14) but not in the controls (from 4.68 ± 1.79 to 4.47 ± 2.03 Kpa; $p > 0.05$, n = 15) (Figure 2). In this study, 6-month NNPPV therapy resulted in mean increases of 16.7% and 20.8% from baseline for

Table 2. Arterial blood gases at baseline and after 6 months in the control and nocturnal nasal positive pressure ventilation (NNPPV) groups

	Control (n = 15)		NNPPV (n = 14)	
	Baseline	After treatment	Baseline	After treatment
pH	7.41 ± 0.07 (7.32–7.48)	7.39 ± 0.06 (7.30–7.49)	7.40 ± 0.05 (7.33–7.50)	7.40 ± 0.03 (7.34–7.44)
PaO_2 (mmHg)	57.85 ± 9.40 (42.8–72.50)	61.69 ± 9.29 (50.10–75.90)	54.31 ± 9.01 (39.60–68.00)	58.79 ± 11.61* (39.00–80.60)
$PaCO_2$ (mmHg)	52.65 ± 4.02 (46.80–60.30)	53.18 ± 13.10 (39.70–93.00)	56.26 ± 7.78 (46.90–69.30)	44.59 ± 6.02†‡ (35.00–55.70)
HCO_3^- (mmol/L)	33.41 ± 4.01 (26.30–41.60)	32.01 ± 5.78 (22.60–46.00)	34.95 ± 5.58 (28.00–49.20)	28.59 ± 5.53†‡ (22.40–45.10)
BE (mmol/L)	7.75 ± 4.48 (–3.00–16.90)	6.16 ± 4.41 (–1–15.90)	8.72 ± 5.24 (2.90–22.00)	3.73 ± 4.54§ (–0.9–17.00)
O_2SAT (%)	88.38 ± 5.38 (79.5–95.3)	89.95 ± 4.66 (78.3–95.3)	85.82 ± 7.23 (70.40–93.40)	88.35 ± 7.14* (71.40–96.10)

**p < 0.05 vs. baseline; †p < 0.001 vs. baseline; ‡p < 0.05 vs. control group; §p < 0.01 vs. baseline. PaO_2 = partial pressure of arterial oxygen; $PaCO_2$ = partial pressure of arterial carbon dioxide; HCO_3^- = arterial bicarbonate; BE = base excess; O_2SAT = oxygen saturation.*

MVV and Pimax, respectively. As shown in Figure 3, however, there were no significant differences in Pemax for either group (from 6.11 ± 1.48 to 6.87 ± 1.92 Kpa and from 7.40 ± 2.27 to 6.76 ± 2.17 Kpa for the NNPPV and control groups, respectively; $p > 0.05$). As shown in Figure 4, a significant correlation was found between improved Pimax and increased MVV ($r_s = 0.56$, $p < 0.05$; n = 14). No significant differences were found between baseline data for MVV, Pimax and Pemax between the NNPPV and control groups.

As shown in Figure 5, NNPPV treatment also resulted in a significant improvement in 6MWD compared to baseline (from 257.1 ± 114.1 to 345.2 ± 109.9 m; $p < 0.001$), whereas this value was significantly decreased in the control group (from 291.7 ± 96.0 to 258.8 ± 97.7; $p < 0.001$). The mean increase from baseline was 88.1 m (34.3%) for the NNPPV group, and the mean reduction was 32.9 m (–11.3%) for the control group ($p < 0.05$; Figure 5). As shown in Figure 6, the change in 6MWD in the NNPPV group was significantly correlated with increased MVV ($r_s = 0.77$, $p < 0.01$; n = 14) for the NNPPV group.

Compared to baseline, NNPPV patients also achieved significant reductions in resting dyspnea sensation (RBorg) and improved RO_2SAT; however, no such significance was achieved for the control group (Table 3).

Discussion

This study demonstrated that 6 months of NNPPV treatment was effective in improving arterial blood gas tension and 6MWD in patients with chronic respiratory failure. NNPPV was also associated with

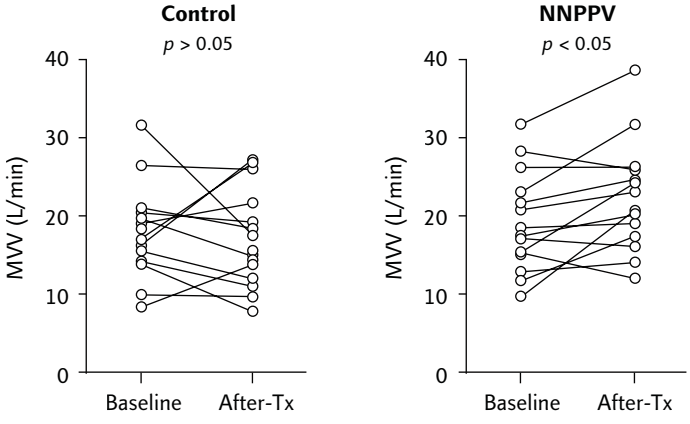

Figure 1. Maximum voluntary ventilation (MVV) was significantly improved for the nocturnal nasal positive pressure ventilation (NNPPV) group (n = 14), but not for the control group (n = 15).

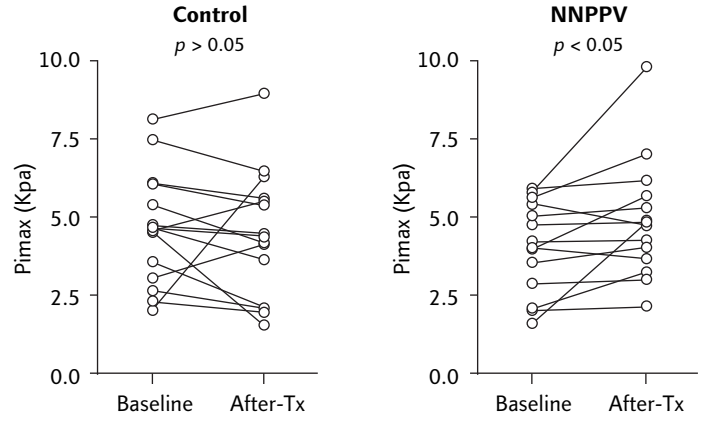

Figure 2. Maximal inspiratory pressure (Pimax) was significantly improved for the nocturnal nasal positive pressure ventilation (NNPPV) group (n = 14), but not for the control group (n = 15).

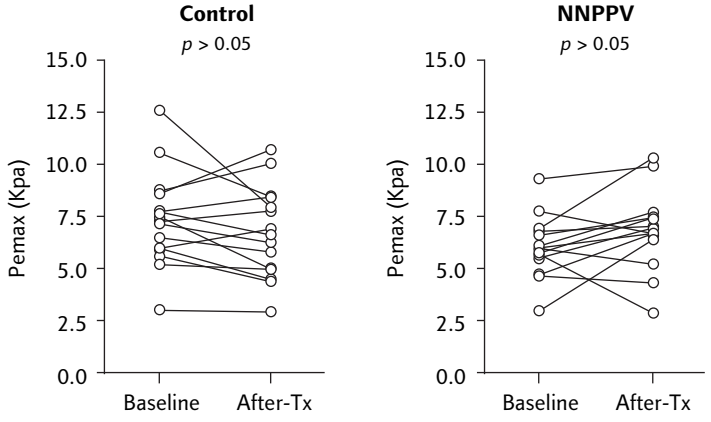

Figure 3. There were no significant differences in maximal expiratory pressure (Pemax) in either group.

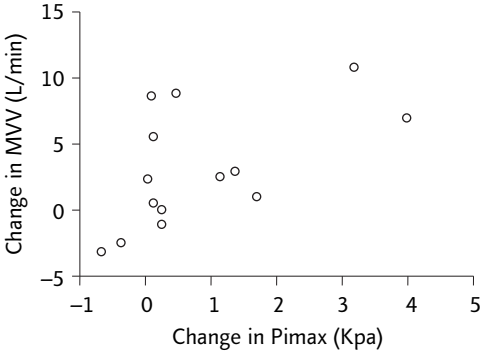

Figure 4. Correlation between changes in maximum voluntary ventilation (MVV) and maximal inspiratory pressure (Pimax) (r_s = 0.56, p < 0.05; n = 14).

improvements in respiratory muscle capacity (including Pimax and MVV), which may have also contributed to the increase in daytime exercise endurance.

While several previous studies suggested that NNPPV may improve arterial blood gas tension in chronic respiratory failure patients, these studies lacked a randomized controlled design and the treatment period ranged from 3 to 6 months.[3,4,18,19] Conversely, Krachman et al concluded that, despite improved sleep efficiency and total sleep time, short-term NPPV does not alter gas exchange in patients with hypercapnic COPD.[5] Our study demonstrated that 6-month NNPPV is effective for improving alveolar ventilation in chronic respiratory failure patients. The increased level of ventilation may result in a resetting of the central respiratory chemoreceptors that lead to a reduction in daytime $PaCO_2$.[20,21]

The benefits of NNPPV with respect to respiratory muscle function remain controversial. Some studies of respiratory muscle function have failed to confirm improvement in Pimax and Pemax after NNPPV treatment in chronic respiratory failure patients,[3,17] especially for specific diagnostic subgroups such as severe COPD[22] and severe cystic fibrosis.[23] However, Piper et al found that NNPPV improved Pimax in patients with cystic fibrosis and hypercapnic respiratory failure.[24] Moreover, Schonhofer et al[11] and Goldstein et al[12] reported beneficial effects of NNPPV on inspiratory threshold-loading test in patients with restrictive lung diseases. Similarly, Cropp and Dimarco found that nocturnal ventilation increased daytime respiratory muscle endurance in patients with severe COPD based on the measurements of the maximum duration of 12-second MVV.[10] It could be speculated, therefore, that the major benefits of NNPPV are enhancement of rest in chronically fatigued inspiratory muscles and improved alveolar ventilation and exercise tolerance. This study demonstrated that NNPPV treatment is not only associated with a significant increase in Pimax, but that it also improves MVV, which influences muscle strength and endurance. We also found that the Pimax increase was associated with improvement in MVV, suggesting that the major effects of NNPPV on maximum voluntary alveolar ventilation may be through influence on respiratory muscle capacity.

Patients with severe COPD often have intrinsic positive end-expiratory pressure (PEEPi), which requires that inspiratory muscles overcome an opposing positive-recoil pressure before inspiratory airflow begins.[25] The expiratory positive airway pressure settings used in NNPPV treatment in our patients may have partially compensated for the PEEPi, thus reducing the inspiratory threshold load needed to trigger the IPAP boost. This may decrease inspiratory muscle work and central drive. A decrease in inspiratory threshold load also allows a triggered IPAP-related increase in tidal volume (VT). This may partially explain the observed NNPPV-related improvements in Pimax and MVV in our chronic respiratory failure patients. However, the lack of use of an endoesophageal balloon to measure the effects of NPPV on inspiratory muscle effort, which would have also enabled us to measure PEEPi and the effects of NPPV on EELV, is an important study limitation.

In recent years, there has been increasing interest in the use of NPPV to increase exercise capacity in chronic respiratory failure patients. Pathophysiologic factors known to contribute to exercise limitations in chronic respiratory failure patients include increased intrinsic mechanical loading of inspiratory muscles (i.e. PEEPi), inspiratory threshold load,[26] increased mechanical restriction of the thorax, inspiratory muscle weakness, increased ventilatory demand relative to capacity, gas-exchange abnormalities, dynamic airway compression, cardiovascular factors, or any combination of the above.[27] Gains in muscle mass and strength have been associated with improved exercise tolerance and survival.[28] In this study, we found that NNPPV therapy for 6 months was associated with significantly increased 6MWD. We also demonstrated that NNPPV improved gas exchange, Pimax and MVV, which may have also contributed to improvements in 6MWD. These findings suggest that the mechanisms responsible for the effects of NNPPV on exercise endurance are complex and their delineation will require further investigation.

A sensation of breathlessness is the most common symptom limiting exercise, with alleviation of breathlessness seemingly important for improving exercise endurance.[29,30] This study showed that NNPPV not only significantly decreased the sensation of breathlessness (RBorg; Table 3), but it also increased RO_2Sat (Table 3) and Pimax (Figure 2). Thus, the reduction in breathlessness in our study may have been due to an improvement in gas exchange.

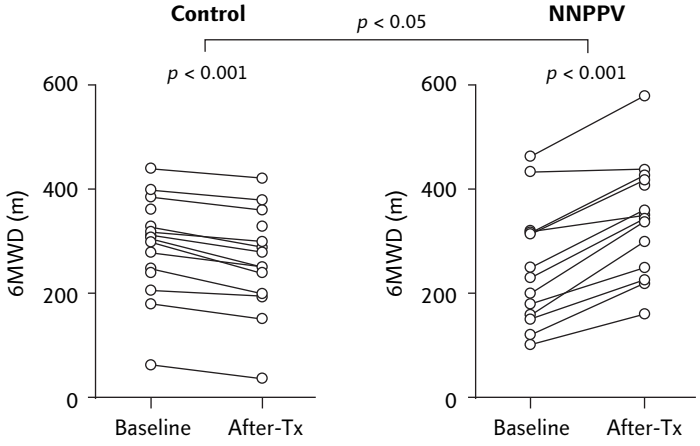

Figure 5. Relative to baseline, nocturnal nasal positive pressure ventilation (NNPPV) resulted in a significant improvement in 6-minute walk distance (6MWD), whereas this value was significantly decreased in the control group.

The changes in exercise endurance may also be associated with the various support modalities and/or treatment periods. In a recent study, it was found that 2-month NNPPV significantly improved 6MWD in patients with chronic respiratory failure (by approximately 18%), but did not improve quadriceps strength.[31] In our investigation, 6 months of NNPPV therapy was associated with a significant increase in 6MWD (88.1 m, 34.3%), a finding superior to that reported by Schonhofer et al.[31] This suggests that long-term treatment may

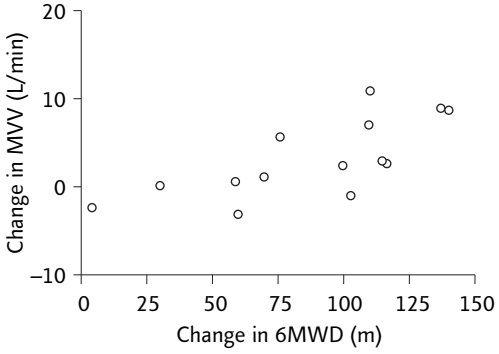

Figure 6. Correlation between changes in maximum voluntary ventilation (MVV) and 6-minute walking distance (6MWD) (r_s = 0.77, $p < 0.01$; $n = 14$).

Table 3. Parameters of dyspnea sensation and oxygen saturation at baseline and after 6 months in the control and nocturnal nasal positive pressure ventilation (NNPPV) groups

	Control (n = 15)		NNPPV (n = 14)	
	Baseline	After treatment	Baseline	After treatment
RBorg	2.7 ± 0.9 (1.0–4.0)	2.7 ± 0.7 (2.0–4.0)	3.2 ± 1.5 (0.0–5.0)	2.4 ± 1.2* (0.0–4.0)
ExBorg	5.8 ± 1.2 (4.0–7.0)	5.9 ± 1.6 (4.0–9.0)	5.8 ± 1.5 (4.0–8.0)	5.7 ± 1.1 (4.0–7.0)
RO_2SAT (%)	90.2 ± 3.1 (84.0–95.0)	91.3 ± 2.7 (88.0–97.0)	87.4 ± 8.2 (66.0–95.0)	89.4 ± 7.6* (71.0–96.0)
ExO_2SAT (%)	78.4 ± 7.4 (66.0–92.0)	79.4 ± 7.4 (69.0–93.0)	75.6 ± 11.2 (54.0–88.0)	74.9 ± 10.6 (51.0–91.0)

*$p < 0.05$ *vs.* baseline. RBorg = resting Borg score; ExBorg = Borg score at the end of the 6-minute walk test; RO_2SAT = resting oxygen saturation; ExO_2SAT = oxygen saturation at the end of the 6-minute walk test.*

be important for improved exercise endurance. The effects of the 6-month therapy on quadriceps strength remain unclear. Further, the mean level of improvement in 6MWD in our study was comparable to that achieved after a 6-week outpatient exercise training program for COPD patients,[32] and superior to an inpatient rehabilitation program for severe but stable COPD patients,[33] suggesting that improvements in exercise tolerance and quality of life can be achieved and sustained for 6 months in patients undergoing NNPPV compared with those receiving conventional care.

Four of the NNPPV patients withdrew from the study within 3 months because they could not tolerate BiPAP during sleep. The dropout rate in our study (21.1%; 4/19) was similar to that reported by Gay et al (19%),[3] but less than that described by Criner et al (28%).[18] The major causes for dropout in our study included mask leak, discomfort arising from the headwear, nasal bridge pressure and eye irritation. The comfort and compliance of patients may have been improved through recruitment of a home care company to apply technological readjustment, personal reassurance and education.

Other limitations of our study also need to be mentioned. Ideally, the effects of NNPPV on respiratory muscle endurance performance should be examined using the pressure threshold-loading test. As clinical facilities for such testing were not available, however, the MVV was measured as an alternative. Further, patients with neuromuscular disorders were excluded from this investigation because they were not able to complete the 6MWT.

We conclude that, for our sample of chronic respiratory failure patients, 6-month NNPPV therapy improved arterial blood gas and exercise capacity compared to the control group. Further, NNPPV therapy was also associated with increased Pimax and MVV, which may have contributed to the increased exercise capacity in these patients.

Acknowledgments

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