Effects of Nasal Pressure Support on Ventilation and Inspiratory Work in Normocapnic and Hypercapnic Patients With Stable COPD*

Dominique Vanpee, MD; Charbel El Khawand, MD; Laurent Rousseau; Jacques Jamart, MD; Luc Delaunois, MD, PhD, FCCP

Objectives: To assess and compare the effect of nasal continuous positive airway pressure (nCPAP), inspiratory pressure support (PSV), and bilevel positive airway pressure (biPAP) on ventilatory parameters and inspiratory work (WOB) in normocapnic and hypercapnic patients with stable COPD.

Methods: While administering nasal pressure support to 10 normocapnic and 10 hypercapnic patients with COPD, we measured airflow and volume with a pneumotachograph as well as esophageal and gastric pressures under nCPAP, PSV, and biPAP conditions.

Results: nCPAP had no influence on ventilatory parameters but decreased WOB and transdiaphragmatic work (Wdi) at 10 cm H2O of pressure in both groups. With PSV and biPAP, ventilatory parameters increased proportionally to the inspiratory applied pressure. WOB and Wdi decreased significantly in both groups while increasing the pressure support. A similar decrease was observed during biPAP proportionally to the level of pressure support. The diaphragmatic pressure-time product decreased similarly in both groups during PSV and biPAP.

Conclusion: The ventilatory response under nCPAP, PSV, and biPAP conditions is similar in hypercapnic and normocapnic patients with stable COPD; PSV and biPAP increase ventilatory parameters and improve Wdi. On the contrary, nCPAP improves WOB but does not increase ventilatory parameters.

Key words: COPD; noninvasive ventilation; pressure support; respiratory work

Abbreviations: biPAP = bilevel positive airway pressure; f = respiratory frequency; nCPAP = nasal continuous positive airway pressure; NIV = noninvasive ventilation; Pdi = transdiaphragmatic pressure; PEEP = positive end-expiratory pressure; PEEPcorr = intrinsic positive end-expiratory pressure after Appendini correction; PEEPi = dynamic intrinsic positive end-expiratory pressure; Pga = gastric pressure; Poes = esophageal pressure; Ppl = pleural pressure; PSV = inspiratory pressure support; PTPdi/min = pressure-time product for the diaphragm by minute; SB = spontaneous breathing; Ti/Ttot = duty cycle; Ttot = total respiratory cycle duration; Vt = minute ventilation; VT = tidal volume; VT/Ti = mean inspiratory flow; Wdi = transdiaphragmatic work; Wdi/L = Wdi by liter; Wdi/min = Wdi by minute; WOB = inspiratory work; WOB/L = inspiratory work by liter; WOB/min = inspiratory work of breathing by minute.

Noninvasive ventilation (NIV) intends to correct hypoventilation and unload the inspiratory muscles. This decrease in muscle effort is due to modifications of the breathing pattern, increase in tidal volume (VT), and reduction in respiratory frequency (f). Inspiratory pressure support (PSV) via nasal or facial mask has been investigated in clinical trials1–6 with encouraging results. Generally, in these studies, only one mode of pressure support and one level of pressure were used; moreover, most studies only concern hypercapnic patients while severe acute dyspnea can occur in patients with COPD without hypercapnia.

There are only a few studies comparing different modes of NIV in the same patients7,8; moreover, lung ventilation measured at different levels of pressure support and its relationship with the work of breathing were rarely investigated. At present, in the literature, no consensus has determined the best mode of pressure support (nasal continuous positive airway pressure [nCPAP], PSV, or bilevel positive airway pressure [biPAP]) for patients with COPD.

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Since hypercapnic patients with COPD present with alveolar hypoventilation and are then unable to sustain additional inspiratory work (WOB), it should be logical that an external NIV improves both ventilatory parameters and WOB at a higher degree in hypercapnic patients than in normocapnic patients, but no study has proven that theory. In this study, we were interested to know whether PSV, nCPAP, and biPAP influence ventilation and inspiratory mechanics in the same way in normocapnic and hypercapnic patients.

**Materials and Methods**

This study protocol was approved by the Institutional Ethics Committee. Patients gave their written informed consent to be included in the study.

**Subjects**

We studied 10 normocapnic and 10 hypercapnic patients with COPD without any other pathologic conditions. When the patients were selected for the study, all were in a stable state for several days, as assessed by their arterial blood gas measurements and pH values (≥7.35), and they were free from exacerbations for at least 10 days. Hypercapnic patients were defined according to an arterial PaCO₂ ≥ 48 mm Hg, but with no acute or uncompensated respiratory acidosis. The diagnosis of COPD was confirmed by clinical history and previous pulmonary function test findings according to European Respiratory Society guidelines. These functional results are shown in Table 1. Results are expressed as a percentage of normal values according to predicted values for lung function variables proposed by the European Community for Coal and Steel. The same way in normocapnic and hypercapnic patients.

**Measurements**

Static and dynamic lung volumes were measured using a body plethysmograph (5300 Part Nair; Medisoft; Dinant, Belgium).

### Table 1—Patient Characteristics, Pulmonary Function Test Results, and Blood Gas Analyses*

<table>
<thead>
<tr>
<th>Characteristics</th>
<th>Hypercapnic Patients (n = 10)</th>
<th>Normocapnic Patients (n = 10)</th>
<th>p Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age, yr</td>
<td>60.4 ± 12</td>
<td>60.5 ± 13</td>
<td>ns</td>
</tr>
<tr>
<td>Height, m</td>
<td>1.65 ± 0.09</td>
<td>1.71 ± 0.01</td>
<td>ns</td>
</tr>
<tr>
<td>Weight, kg</td>
<td>85 ± 18</td>
<td>73 ± 16</td>
<td>ns</td>
</tr>
<tr>
<td>FVC, % predicted</td>
<td>60 ± 12</td>
<td>77 ± 21</td>
<td>ns</td>
</tr>
<tr>
<td>FEV₁, % predicted</td>
<td>32 ± 17</td>
<td>40 ± 26</td>
<td>ns</td>
</tr>
<tr>
<td>FRC, %</td>
<td>135 ± 45</td>
<td>191 ± 54</td>
<td>0.035</td>
</tr>
<tr>
<td>DLCO, %</td>
<td>48 ± 19</td>
<td>46 ± 26</td>
<td>ns</td>
</tr>
<tr>
<td>KCO, %</td>
<td>75 ± 9</td>
<td>47 ± 22</td>
<td>0.025</td>
</tr>
<tr>
<td>pH</td>
<td>7.38 ± 0.07</td>
<td>7.42 ± 0.04</td>
<td>0.007</td>
</tr>
<tr>
<td>PaO₂, mm Hg</td>
<td>50 ± 2.1</td>
<td>64 ± 14.6</td>
<td>&lt; 0.009</td>
</tr>
<tr>
<td>PaCO₂, mm Hg</td>
<td>57 ± 2.3</td>
<td>42 ± 6.03</td>
<td>&lt; 0.001</td>
</tr>
</tbody>
</table>

*Data are presented as mean ± SD. FRC = functional residual capacity; DLCO = diffusing capacity of the lung for carbon monoxide; KCO = diffusing capacity according to measured alveolar volume; ns = not significant.

Single-breath diffusion capacity of the lung for carbon monoxide was measured by using a 5200 Part Nair (Medisoft). Arterial blood gas tensions were also measured (IL BG3; Instrumentation Laboratory, Barcelona, Spain). Airflow was measured at the opening of the nasal mask with a Fleisch pneumotachograph connected to a differential pressure transducer (Validyne DP 45 ± 5 cm H₂O; Validyne Engineering, Northridge, CA). Volume was obtained from numerical integration of the flow signal. Minute ventilation (Ve), Vt, f, inspiratory time, expiratory time, total respiratory cycle duration (Ttot), mean inspiratory flow (Vr/Tt) and duty cycle (Tr/Ttot) were calculated as average values from 1-min continuous recordings of flow and volume. Changes in pleural pressure (Ppl) and abdominal pressure were respectively measured from esophageal pressure (Poes) and gastric pressure (Pga). Both Poes and Pga were measured using a catheter (length, 110 cm; external diameter, 2.1 mm) equipped with two pressure transducer sensors (Gaetee; Dunvegan, Isle of Skye, Scotland) and connected to an amplifier (Medatech; Brussels, Belgium). This system has recently been confirmed to be reliable for rapid changes in respiratory pressures and studies of respiratory muscle strength. Transdiaphragmatic pressure (Pdi) was obtained by subtracting Poes from Pga. Data obtained from these measurements were registered and processed with commercial respiratory software (Anadat 5.2; D. Bates; Montreal, PQ, Canada), which calculates the work of breathing by Campbell’s method. WOB was computed from Poes and volume [WOB = fVdPoes], and Wdi was calculated from Pdi and volume. It was calculated according to ventilation (WOB by liter [WOB/L], transdiaphragmatic work [Wdi] by liter [Wdi/L]), and time (WOB by minute [WOB/min], Wdi by minute [Wdi/min]).

Dynamic intrinsic positive end-expiratory pressure (PEEPi dyn) was measured as the amount of negative deflection in Ppl preceding the start of the inspiratory flow. When present, the concomitant decrease of Pga was subtracted from PEEPi dyn; indeed, if the expiratory muscles contract during expiration, part of the decrease in Ppl at the beginning of inspiration can be produced not only by the contraction of the inspiratory muscles but also by the relaxation of the expiratory muscles. The pressure-time product for the diaphragm by minute (PTPdi/min) was obtained by measuring the area under the Pdi-vs-time relationship. We used PTPdi/min as an index of inspiratory muscle activity since it has been shown to be correlated with the oxygen uptake of the inspiratory muscles.

**Experimental Procedure**

All subjects were studied in a sitting position while breathing room air through a nasal mask. They were asked to relax completely, and instructions were given to keep their mouths closed.

After topical anesthesia (xylocaine spray 10%) was administered, the catheter was inserted through the nose to the stomach, and the position of the sensors and the signal were checked according to the method of Baydur et al. Physiologic measurements during spontaneous breathing (SB) were performed when the patients appeared to be relaxed (mean time ≥ 5 min), and then a customized nose mask was applied for NIV. The three modes were applied via a portable ventilator (BiPAP S/T-D; Respironics; Pittsburgh, PA) during SB.

Measurements were performed in order without rest periods: (1) during the control condition, in which the patients breathed spontaneously through the nose mask and the measuring equipment; (2) during increasing levels of PSV of 5/0, 10/0, 15/0, and 20/0 cm H₂O (inspiratory pressure/expiratory pressure); (3) during nCPAP at 5 cm H₂O; (4) during increasing biPAP at...
10/5, 15/5, and 20/5 cm H₂O; (5) during nCPAP at 10 cm H₂O; and (6) during increasing biPAP at 15/10 cm H₂O and 20/10 cm H₂O.

Two periods of measurements were performed in control conditions to attest the stable state, and the measurements were repeated at increasing levels of pressure. The ventilation periods were not randomized, but the inspiratory pressure was progressively increased in order to avoid problems of intolerance to a sudden change of pressure. All physiologic signals were recorded during a 1-min period after at least 3 min of stabilization of the respiratory parameters at each breathing period, or longer if needed (between 3 min and 5 min); the measurements were repeated if artifacts or events had disturbed the recording. Transcutaneous pulse oximetry was performed during the whole procedure for safety.

**Table 2—Control Conditions**

<table>
<thead>
<tr>
<th>Variables</th>
<th>Normocapnic Patients</th>
<th>Hypercapnic Patients</th>
<th>p Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Vₑ, L/min</td>
<td>10.8 ± 5.8</td>
<td>5.8 ± 1</td>
<td>0.023</td>
</tr>
<tr>
<td>Vₜ, L</td>
<td>0.57 ± 0.25</td>
<td>0.30 ± 0.06</td>
<td>0.009</td>
</tr>
<tr>
<td>f</td>
<td>19.45 ± 8.75</td>
<td>19.8 ± 5.7</td>
<td>ns</td>
</tr>
<tr>
<td>Vt/Ti</td>
<td>0.47 ± 0.20</td>
<td>0.30 ± 0.05</td>
<td>&lt; 0.022</td>
</tr>
<tr>
<td>WOB/L, J/L</td>
<td>0.73 ± 0.35</td>
<td>1.03 ± 0.05</td>
<td>ns</td>
</tr>
<tr>
<td>WOB/min, J/min</td>
<td>7.6 ± 5.1</td>
<td>6.3 ± 5.2</td>
<td>ns</td>
</tr>
<tr>
<td>WdI/L, J/L</td>
<td>1.95 ± 1.51</td>
<td>1.05 ± 0.49</td>
<td>ns</td>
</tr>
<tr>
<td>WdI/min, J/min</td>
<td>17.8 ± 9.87</td>
<td>6.25 ± 3.62</td>
<td>0.005</td>
</tr>
<tr>
<td>PTPdi/min, cm H₂O/min</td>
<td>616.2 ± 469.8</td>
<td>233.8 ± 115.8</td>
<td>0.024</td>
</tr>
<tr>
<td>PEEP dyn, cm H₂O</td>
<td>4.8 ± 3.2</td>
<td>3.6 ± 2.41</td>
<td>ns</td>
</tr>
</tbody>
</table>

*Data are presented as mean ± SD. See Table 1 for expansion of abbreviation not used in text.

**Data Analysis**

Unless otherwise indicated, numerical variables are expressed as mean ± SD. The various parameters were compared between the control condition and nCPAP at 5 cm H₂O and 10 cm H₂O by two-way analysis of variance (group of patients, condition) for repeated measurements. A regression analysis with generalized estimating equations was used to study the influence of pressure in PSV conditions, and was performed by a repeated measures for generalized estimating equations program.

**Results**

**Populations**

The functional characteristics of the normocapnic and hypercapnic patients are listed in Table 1. Functional parameters were significantly different, with a higher functional residual capacity (p = 0.035) and a lower diffusion capacity of the lung according to measured alveolar volume (p = 0.025) in normocapnic patients when compared to hypercapnic patients. By definition, PaCO₂ was significantly higher in hypercapnic patients, while PaO₂ was lower.

**Ventilatory Parameters**

**Control Conditions:** Mean Vₑ was significantly higher in the normocapnic group (10.8 ± 5.8 L/min) than in the hypercapnic group (5.8 ± 1.0 L/min) (p = 0.023). This is due to a higher Vₜ with no significant difference of f and Ti/TTOT but with a
higher Vt/Ti (0.47 ± 0.20 vs 0.30 ± 0.05; p < 0.022) [Table 2].

**CPAP Conditions:** As shown in Figure 1, no significant changes of the ventilatory parameters were observed in both groups during nCPAP.

**PSV Conditions:** PSV caused a significant increase in Vt from 10.83 ± 5.80 to 14.99 ± 5.58 L/min (p < 0.001) and from 5.80 ± 1.00 to 9.17 ± 2.50 L/min (p < 0.001) [PSV of 20/0 cm H$_2$O in comparison to SB] in normocapnic and hypercapnic groups, respectively. This change in Vt was due to an increase of Vt/Ti (0.59 ± 0.18; p = 0.003), and Ti/TTtot (0.42 ± 0.05; p < 0.001) with regard to control conditions, without concomitant decrease of f in normocapnic patients. In hypercapnic patients, the increase of Vt was also due to an increase of Vt/Ti (0.36 ± 0.12; p = 0.046), and Ti/TTtot (0.42 ± 0.05; p < 0.001) with regard to control conditions; but in this group, a decrease in f (p < 0.001) at high PSV was observed (Fig 2, top, A). The slopes of the increase of Vt are similar in both groups, as shown in Figure 2, top, A.

**biPAP Conditions:** As shown in Figure 2 (middle, B, and bottom, C), changes similar to those observed during PSV were shown under ventilation with biPAP both in normocapnic and hypercapnic patients.

**Work of Breathing**

**Control Conditions:** Mean WOB/L and mean WOB/min were similar in normocapnic and hypercapnic patients. While Wdi/L was not statistically different between both groups, mean Wdi/min and PTPdi/min were significantly higher in normocapnic patients (Table 2). PEEPi dyn was similar in both groups.

**CPAP Conditions:** With nCPAP, we observed a decrease of WOB/L and Wdi/L, but only when the level of nCPAP was > 5 cm H$_2$O (Fig 3). The evolution of curves was also similar in both groups.

**PSV Conditions:** During PSV, WOB/L decreased from 0.73 ± 0.35 J/L in normocapnic patients and from 1.03 ± 0.65 J/L in hypercapnic patients in control conditions to a value of approximately zero during PSV at 20/0 cm H$_2$O (p < 0.001; Fig 4, top, A). Passive inflation by the respirator was observed in some patients at a high PSV level. The Poes-volume loop moved in a counterclockwise direction, indicating quasi-total respiratory muscle unloading.\(^{20,21}\) The minimal amount of work necessary to trigger the ventilator was too small to be recorded by our method. Wdi/L decreased also significantly (p < 0.0001) for normocapnic and hypercapnic groups.
The initiation of PSV led to a remarkably significant decrease in the principal indexes of diaphragmatic effort. The PTPdi/min decreased proportionally to the levels of PSV in both groups (Table 3). The decreasing slopes of WOB and Wdi were similar in both groups. During PSV for normocapnic patients, there was a significant increase of PEEPi dyn (p < 0.001) before and after Appendini correction (PEEPicorr) [p = 0.007], but there was no increase for hypercapnic patients (Table 3).

**biPAP Conditions:** biPAP did not cause further significant reductions in WOB, Wdi (Fig 4, middle, B, and bottom, C, and Table 3) or PTPdi/min (Table 3) in both patient groups. There was no increase of PEEPi dyn or PEEPicorr during biPAP in both patient groups (Table 3). The slopes were not significantly different.

**DISCUSSION**

The aim of this study was to assess and compare the effects of several modes of NIV on the ventilatory parameters and WOB of normocapnic patients (n = 10) and hypercapnic patients (n = 10) with stable COPD. Our results showed that ventilatory behavior during nCPAP, PSV, and biPAP was similar in both patient groups. In both groups, PSV and biPAP increased the ventilatory parameters and also relieved WOB significantly. However, in both groups, nCPAP improved WOB but did not increase ventilatory parameters.

In our study, WOB and Wdi were higher during control conditions for normocapnic patients, probably due to the effort needed to maintain a normal PaCO2. In both patient groups, nCPAP had no influence on ventilatory parameters (VE, VT, and f) but decreased WOB/min and Wdi/min or Wdi/L when the level of pressure was > 5 cm H2O (Fig 3), despite the fact that nCPAP was applied during short periods. A former study with a short-term (15 min) application of nCPAP (5 cmH2O) in hypercapnic patients showed that it did not affect f, VT, VE, or gas exchange. These results are similar to ours despite the fact they used a different protocol, with rest periods between the unrandomized different conditions. Conversely, another study with a nCPAP of 5 cm H2O applied for 4 h showed a significant reduction in f, dyspnea score, and PaCO2. Therefore, it seems that despite the fact that nCPAP quickly unloads the inspiratory muscles, there is no increase in ventilatory param-

![Graph showing Wdi/L and WOB/L in normocapnic and hypercapnic groups during control conditions, nCPAP at 5 cm H2O, and nCPAP at 10 cm H2O. For normocapnic patients, between the control condition and nCPAP at 10 cm H2O, Wdi/L and WOB/L decreased significantly (p = 0.006 and p = 0.05, respectively). Only Wdi/L decreased significantly (p = 0.001) for hypercapnic patients. Data are expressed as mean ± SEM. The slopes between both populations are similar.](http://publications.chestnet.org/pdfaccess.ashx?url=/data/journals/chest/21980/ on 06/17/2017)
eters at least in the initial period of treatment, and thus a long application is needed for a ventilatory improvement in hypercapnic patients.

Controlled trials of PSV in patients with stable COPD reported conflicting results on short-term clinical and functional outcomes. In patients with stable COPD and chronic hypercapnia, Vita et al evaluated the short-term physiologic effects of two settings of nasal PSV (empirical setting vs physiologic setting where the level of inspiratory pressure support and intrinsic positive end-expiratory pressure [PEEPi] was tailored to the patient according to an invasive evaluation). Their study showed that PSV was efficient in improving arterial blood gas measurements and in unloading inspiratory muscles independently, whether it was set taking into account the patient’s comfort and improvement in arterial blood gas measurements (empirical setting), or tailored to the patient’s respiratory muscle efforts and mechanics. However, there was a very important decrease of inefficient inspiratory efforts with the physiologic setting. In our study, we found that PSV increased V̇e, resulting in increased Vt, inspiratory flow, and Ti/Ttot; however, a significant decrease of f was observed in the hypercapnic group, at high PSV and biPAP pressures only. This change in the pattern of breathing should improve alveolar ventilation. Concerning inspiratory mechanics parameters during PSV, WOB and Wdi decreased largely proportionately to the level of pressure. Similar results had been shown by Brochard et al., who evaluated the physiologic effects of inspiratory positive airway pressure applied via a face mask in 11 patients with acute exacerbations of COPD. Treatment was associated with increased V̇r, decreased f, improved gas exchange, and significant decrease in diaphragmatic activity (as measured by Pdi, the pressure-time product for the diaphragm, and integrated surface diaphragmatic electromyographic activity). Best results were obtained with higher inspiratory positive airway pressures (20 cm H2O vs 12 cm H2O), as it was performed in our study in both normocapnic and hypercapnic states. This also confirms the results of other clinical studies performed in patients with acute exacerbations of COPD, which demonstrated that as the level of the inspiratory pressure was increased, the consequent V̇r increased and the f decreased with improved gas exchange; these studies also demonstrated that PSV without continuous positive airway pressure could unload ventilatory muscles during SB, either totally or partially.

When adequate pressure was applied, the improvement in gas exchange was rapid and proportional to the increments in V̇e; increasing the applied inspiratory pressure from 12 to 20 cm H2O resulted in a significant decrease of PaCO2 in hypercapnic patients despite a slower f.

In summary, it seems that all of these studies demonstrate that this mode of ventilation usually
results in a decrease of WOB and in an increase of alveolar ventilation in hypercapnic patients. Our results show that a similar improvement of an abnormally high WOB can also be obtained in normocapnic patients.

The results we obtained during biPAP showed that it increased \( V_{E} \) with a significant increase of \( V_{t} \). A decrease of \( f \) was observed in hypercapnic patients but at low expiratory pressure level (5 cm H\(_2\)O) only (Fig 2, middle, B). There was no decrease at a higher expiratory pressure (10 cm H\(_2\)O; Fig 2, bottom, C). WOB, Wdi, and PTPdi/min decreased also significantly in both groups.

Appendini et al.\(^7\) studied clinical and physiologic measurements during SB and during application of nCPAP alone, PSV alone, and PSV plus nCPAP. Inspiratory muscle effort was reduced by PSV as evidenced by decreases in Pdi measurements. Additionally, inspiratory muscle work was reduced further by the addition of external positive end-expiratory pressure (PEEP) at a level of 80 to 90% of PEEPi despite the fact that \( V_{E} \) and arterial blood gas measurements were improved by PSV only, with no further improvement with the addition of PEEP. Nava et al.\(^3\) showed that in patients with severe, stable COPD, 5 cm H\(_2\)O of PEEP plus 10 cm H\(_2\)O of PSV (reaching an overall inspiratory pressure of 15 cm H\(_2\)O) unloaded the inspiratory muscles in the same way as 20 cm H\(_2\)O of PSV alone. This study demonstrated that additional external PEEP can improve ventilatory parameters and reduce respiratory muscle work in patients with COPD at lower levels of PSV. Our study shows that these ventilatory and mechanical effects of biPAP can be observed in normocapnic patients as well as in hypercapnic patients. The consequences of these NIVs on PEEPi (increase during PSV, no change during CPAP and biPAP) are also similar in both populations.

The concomitant increase of ventilatory parameters and decrease of WOB during PSV and biPAP are certainly useful in hypercapnic patients. It is nevertheless probable that the use of PSV or biPAP in normocapnic patients could induce hyperventilation, and that the expected benefit of decreasing the WOB could be counterbalanced by the harmful effect of hypocapnia. The consequence of PSV and biPAP on PaCO\(_2\) should then always be checked before using these NIV systems in normocapnic patients. Conversely, since nCPAP improves WOB without modifying the ventilatory parameters, nCPAP could be safely used to decrease WOB in these normocapnic patients where no effect on ventilatory parameters is needed. It is then possible that nCPAP could be the preferable NIV to avoid

<table>
<thead>
<tr>
<th>Condition</th>
<th>WOB/min, J/min</th>
<th>PT Pdi/min, cm H(_2)O</th>
<th>PEPicorr, cm H(_2)O</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normocapnic</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hypercapnic</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>PSV, inspiratory pressure(expiratory pressure) (cm H(_2)O)</td>
<td>5.4 6.3</td>
<td>5.2 4.8</td>
<td>5.2 4.8</td>
</tr>
<tr>
<td>BiPAP, inspiratory pressure/expiratory pressure (cm H(_2)O)</td>
<td>5.2 6.2</td>
<td>4.8 5.3</td>
<td>4.8 5.3</td>
</tr>
</tbody>
</table>

*p value < 0.001

Table 3—Inspiratory Mechanical Parameters

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respiratory muscle fatigue in normocapnic patients, even though PSV and biPAP are better in hypercapnic patients.

CONCLUSION

We conclude that during nCPAP, PSV, and biPAP, ventilatory parameters are similar in hypercapnic and normocapnic patients with stable COPD. In both groups, PSV and biPAP increase ventilatory parameters with improvement of the WOB and Wdi, whereas nCPAP improves the WOB (> 5 cm H2O) but does not increase ventilatory parameters.

So, the application of a change in pressure to the airway (either as PSV or biPAP) improves ventilatory parameters and decreases WOB. This improvement in ventilation is proportional to the size of the change in pressure applied. Furthermore, significant further diminution in the WOB can be obtained by the addition of PEEP, with maximal effect being obtained when this is approximately the equivalent of PEEPi.

Patients with an acute exacerbation of COPD present with dyspnea, tachypnea, use of accessory muscles, and increased WOB. With increased WOB, muscle production of carbon dioxide increases and alveolar ventilation is often unable to increase enough to prevent carbon dioxide retention and respiratory acidosis. Using of NIV in these hypercapnic patients is recognized for its efficacy. In the case of COPD exacerbation without hypercapnia, the patients have to increase their ventilatory efforts, which results in an increase of dyspnea and induces respiratory muscle fatigue. Our results show that NIV can be useful in isocapnic patients with stable COPD, since nCPAP, PSV, and biPAP improve their WOB. NIV could possibly help these isocapnic patients during acute events by relieving their dyspnea and avoiding respiratory muscle fatigue, but the PaCO2 level must be controlled if PSV or biPAP are used, in order to avoid hypocapnia, and nCPAP is probably better in these patients.

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