The Appropriate Setting of Noninvasive Pressure Support Ventilation in Stable COPD Patients*

Michele Vitacca, MD; Stefano Nava, MD; Marco Confalonieri, MD; Luca Bianchi, MD; Roberto Porta, MD; Enrico Clini, MD, FCCP; and Nicolino Ambrosino, MD, FCCP

**Study objective:** To evaluate the short-term physiologic effects of two settings of nasal pressure-support ventilation (NPSV) in stable COPD patients with chronic hypercapnia.

**Design:** Randomized controlled physiologic study.

**Setting:** Lung function units and outpatient clinic of two affiliated pulmonary rehabilitation centers.

**Patients:** Twenty-three patients receiving domiciliary nocturnal NPSV for a mean (± SD) duration of 31 ± 20 months.

**Methods:** Evaluation of arterial blood gases, breathing pattern, respiratory muscles, and dynamic intrinsic positive end-expiratory pressure (PEEPd, dyn) during both unassisted and assisted ventilation. Two settings of NPSV were randomly applied for 30 min each: (1) usual setting (U), the setting of NPSV actually used by the individual patient at home; and (2) physiologic setting (PHY), the level of inspiratory pressure support (IPS) and external positive end-expiratory pressure (PEEPe) tailored to patient according to invasive evaluation of respiratory muscular function and mechanics.

**Results:** All patients tolerated NPSV well throughout the procedure. Mean U was IPS, 16 ± 3 cm H2O and PEEPe, 3.6 ± 1.4 cm H2O; mean PHY was IPS, 15 ± 3 cm H2O and PEEPe, 3.1 ± 1.6 cm H2O. NPSV was able to significantly (p < 0.01) improve arterial blood gases independent of the setting applied. When compared with spontaneous breathing, both settings induced a significant increase in minute ventilation (p < 0.01). Both settings were able to reduce the diaphragmatic pressure-time product, but the reduction was significantly greater with PHY (by 64%; p < 0.01) than with U (56%; p < 0.05). Eleven of 23 patients (48%) with U and 7 of 23 patients (30%) with PHY showed ineffective efforts (IE); the prevalence of IE (20 ± 39% vs 6 ± 11% of their respiratory rate with U and PHY, respectively) was statistically different (p < 0.05).

**Conclusion:** In COPD patients with chronic hypercapnia, NPSV is effective in improving arterial blood gases and in unloading inspiratory muscles independent of whether it is set on the basis of patient comfort and improvement in arterial blood gases or tailored to a patient’s respiratory muscle effort and mechanics. However, setting of inspiratory assistance and PEEPe by the invasive evaluation of lung mechanics and respiratory muscle function may result in reduction in ineffective inspiratory efforts. These short-term results must be confirmed in the long-term clinical setting.

(CHEST 2000; 118:1286–1293)

**Key words:** breathing pattern; hypercapnia; noninvasive mechanical ventilation; respiratory failure; respiratory muscles

**Abbreviations:** ANOVA = analysis of variance; f = respiratory frequency; Fio2 = fraction of inspired oxygen; IE = ineffective efforts; IPS = inspiratory pressure support; NPPV = noninvasive positive-pressure ventilation; NPSV = nasal pressure-support ventilation; Pao2 = pressure at the airway opening; Pdi = transdiaphragmatic pressure; PEEPe = external positive end-expiratory pressure; PEEPd, dyn = dynamic intrinsic positive end-expiratory pressure; Pes = esophageal pressure; PHY = physiologic setting; PTPdi = pressure-time product for the diaphragm; PTPdi,b = PTPdi per breath; PTPdi,min = PTPdi calculated over a period of 1 min; PTPdi/Vt = PTPdi corrected per liter of ventilation; SB = spontaneous breathing; Ti = inspiratory time; Ttot = total cycle duration; U = usual setting; Vt = minute ventilation; Vr = tidal volume

*From the Pulmonary Department (Drs. Vitacca, Bianchi, Porta, Clini, and Ambrosino) Scientific Institute of Gussago, Fondazione Salvatore Maugeri IRCCS, Gussago, Italy; and Pulmonary Department (Dr. Nava), Scientific Institute of Monteskanco, Fondazione Salvatore Maugeri IRCCS, Monteskanco, Italy; and Bergamo General Hospital (Dr. Confalonieri), Bergamo, Italy. Manuscript received December 16, 1999; revision accepted May 30, 2000.

Correspondence to: Nicolino Ambrosino, MD, FCCP, Fondazione S. Maugeri, Lung Function Unit, Istituto Scientifico di Gussago, I-25064 Gussago (BS), Italy; e-mail: nambrsnino@fsm.it

C ontrolled trials of noninvasive positive-pressure ventilation (NPPV) in patients with stable COPD reported conflicting results on short-term clinical and functional outcome.1–4 Even fewer data are reported on the long-term effects.5 Furthermore, in a recent descriptive, prospective analysis, Crierer et al6 found that only 50% of patients with COPD continued to use NPPV during prolonged follow-up of approximately 6 months, despite enroll-
ment in a comprehensive inpatient and outpatient program.

Selection of patients, modalities of ventilation, and types of ventilators and their settings have been claimed to account for these conflicting results. Three possible physiologic mechanisms of the effects of NPPV in COPD patients have been claimed, namely the nighttime improvement in arterial blood gases, a direct effect on respiratory mechanics, and respiratory muscle unloading, which may lead to a clinical improvement of a supposed but unproved chronic respiratory fatigue. An effect of NPPV on nocturnal hypoventilation may be assessed by invasive evaluation of the respiratory muscle pressures or by electromyography. A 40 to 50% reduction of spontaneous diaphragmatic electrical activity has been claimed to be the minimal target to achieve a satisfactory unloading of these muscles. On the other side, overassistance, as assessed by a positive deflection of esophageal swing for most of inspiration, during assisted modes of ventilation should be avoided, as it may lead, at least theoretically, to the risk of respiratory muscle atrophy.

Most of the so-called domiciliary pressure ventilators have the possibility to set also an external positive end-expiratory pressure (PEEP). The application of PEEP has been shown to better unload the diaphragm, provided an adequate titration (ie, 80 to 90% of intrinsic dynamic PEEP [PEEPi,dyn]), while an incorrect setting of PEEP may lead, for example, to overassistance and may further increase hyperventilation.

In clinical practice, home NPPV for COPD patients is prescribed as nasal pressure-support ventilation (NPSV) and is set to achieve a decrease in PaO2 and an optimal patient’s compliance. However, with this setting, no information is available on the actual respiratory muscle unloading. The aim of this study was therefore to compare the effects on arterial blood gases, breathing pattern, respiratory muscle function, and patient–ventilator interaction of two settings of NPSV aimed to different targets: (1) a usual setting (U) at patient’s comfort, actually used in a population who are prescribed home NPSV; and (2) a physiologic setting (PHY), with the target being to properly unload the respiratory muscles and to tailor the PEEPs as suggested by the physiologic studies.

**Materials and Methods**

The investigative protocol was approved by the Institutional Ethics Committee (Salvatore Maugeri Foundation) and was conducted according to the Declaration of Helsinki. Informed consent was obtained from all patients before enrollment into the study.

**Patients**

From January 1, 1998, to January 1, 1999, 23 COPD patients with chronic hypercapnic respiratory insufficiency receiving long-term oxygen therapy and domiciliary nocturnal NPSV (see “Home Mechanical Ventilation” section below) were studied in the lung function units of the Rehabilitation Centers of Gussago (9 patients) and Montesano (14 patients). Diagnosis of COPD was made according to the American Thoracic Society guidelines. In fact, all the patients of this study were well known to the institutions to which they were referred as outpatients for periodic visits, arterial blood gas control tests, and treatment adjustment. At the time when they were recruited for this study, all patients were in stable condition, as assessed by arterial blood gas values and pH (> 7.35), and had been free from exacerbation for at least 1 month. Patients suffering from other organ failure, cancer, or inability to cooperate were excluded from the study. All patients were receiving regular drug therapy with inhaled bronchodilators; two patients were taking low doses of systemic steroids long term, and five were treated with inhaled steroids. No change in the routine medical and oxygen therapy was made in the week preceding the study. Demographic, anthropometric, and functional characteristics of patients in the study are shown in Table 1.

**Home Mechanical Ventilation**

All patients were receiving long-term oxygen therapy and had been on a home nocturnal NPSV program prescribed by the centers of Gussago and Montesano for 31 ± 20 months (range, 7 to 72 months). Home NPSV was delivered through a nasal mask by means of a portable ventilator. Criteria to enroll patients for home NPSV were as follows: long-term oxygen therapy > 15 h/d for ≥ 6 months; FEV1 < 50% predicted FEV1/vital capacity < 60%; PaO2 < 60 mm Hg; PaCO2 > 50 mm Hg; and pH > 7.34 breathing room air. The last mean (SD) arterial blood gas measurements made during spontaneous breathing of room air at discharge from the hospital at the beginning of home NPSV were the following: pH, 7.38 ± 0.2; PaCO2, 56.2 ± 3.0 mm Hg; and PaO2, 51.7 ± 5.3 mm Hg. In this clinical setting, NPSV had been set by a physician expert in the field (M.V., S.N., M.C.) at the

---

**Table 1—Demographic, Anthropometric, and Functional Characteristics of Patients in Study**

<table>
<thead>
<tr>
<th>Characteristics</th>
<th>Results</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age, yr</td>
<td>68 ± 5</td>
</tr>
<tr>
<td>Sex, No.</td>
<td></td>
</tr>
<tr>
<td>Female</td>
<td>2</td>
</tr>
<tr>
<td>Male</td>
<td>21</td>
</tr>
<tr>
<td>Weight, kg</td>
<td>71 ± 4</td>
</tr>
<tr>
<td>BMI</td>
<td>25 ± 4.6</td>
</tr>
<tr>
<td>FEV1, % pred</td>
<td>23 ± 7</td>
</tr>
<tr>
<td>FVC, % pred</td>
<td>40 ± 8</td>
</tr>
<tr>
<td>FEV1/FVC, %</td>
<td>40 ± 10</td>
</tr>
<tr>
<td>RV, % pred</td>
<td>189 ± 42</td>
</tr>
<tr>
<td>TLC, % pred</td>
<td>127 ± 27</td>
</tr>
<tr>
<td>Home NPPV, mo</td>
<td>31 ± 20</td>
</tr>
</tbody>
</table>

*Values given as mean ± SD, unless otherwise indicated. BMI = body mass index; RV = residual volume; TLC = total lung capacity.
maximal tolerated inspiratory pressure support (IPS) able to reduce awake PaCO₂ by ≥ 5% of the spontaneous breathing (SB) values. A preset level of PEEPi was added. Specifically, in each patient after setting IPS, the level of PEEPi was progressively increased by 1 cm H₂O at patient’s comfort. In all cases, the level of PEEPi had to be less than or equal to an arbitrary upper limit of 6 cm H₂O. This value was chosen with the purpose of not overcoming the level of PEEPi, dyn commonly reported in these stable COPD patients.\(^{16,17}\)

The effectiveness of NPSV was evaluated by assessing awake arterial blood gases during 2 h of continuous ventilation: nocturnal arterial oxygen saturation under ventilation (> 90%); and subjective tolerance to NPSV during a preliminary in-hospital 2-week acclimatization period. After daily practice trials in which the effects of NPSV were tested, patients were instructed to perform NPSV for at least five consecutive hours at night. Patients were discharged with home NPSV with a mean level of IPS of 16 ± 3 cm H₂O (range, 10 to 22 cm H₂O) with the addition of 3.1 ± 1.6 cm H₂O (range 0 to 6 cm H₂O) of PEEPi. The patients were periodically followed up in our institutions, so that changes in the ventilatory settings were performed if needed. Actually, only two of the patients enrolled had their variables modified before the current study. In one patient, the level of IPS was decreased from 16 to 13 cm H₂O because of lack of tolerance to the former inspiratory pressure. In the second patient, the IPS was increased from 10 to 14 cm H₂O because, at a control evaluation, the former level was unable to significantly decrease acutely the PaCO₂.

**Measurements**

Lung volumes and FVC were measured by means of constant volume body plethysmographs (mode 1085; Medical Graphics Corp; St. Paul, MN; or MasterLabs; Jaeger; Wurtzburg, Germany) and spirometric values (FEV₁ and FVC) using a water spirometer (Baires; Biomedin; Padova, Italy). The predicted values of Quanjet\(^{19}\) were used. PaO₂, PaCO₂, and pH were measured on blood sample from the radial artery by means of automated analyzers (840 Ciba Corning; Medfield, MA; and ABL 500: Radiometer; Copenhagen, Denmark).

For the experimental procedure of this study, flow was measured by means of a heated pneumotachograph connected to a differential pressure transducer. The pneumotachograph was inserted between the nasal mask and the plate valve of the ventilator circuit.\(^{19}\) Volume was obtained by numerical integration of the flow signal. The pressure at the airway opening (Pao) was measured at a side port placed between the mask and the pneumotachograph by means of 70-cm-long rigid tubing connected to a pressure transducer. Changes in pleural and abdominal pressures were estimated from changes in esophageal (Pes) and gastric pressures, respectively, by means of the balloon catheter technique. Transpulmonary and transdiaphragmatic (Pdi) pressures were obtained by subtraction of Pes from Pao and gastric pressure, respectively.\(^{14}\)

**Data Analysis**

All signals were digitized by an analog-to-digital converter with 12-bit resolution, connected to a Pentium 100 personal computer, at a sampling frequency of 100 Hz for the analysis of the breathing pattern and of respiratory mechanics. The subsequent analysis was performed using a software package (ANADAT 5.1; RHT-Infodat; Montreal, Quebec, Canada).

Tidal volume (VT), respiratory frequency (f), and minute ventilation (Ve) were computed from the volume signal. Total breathing cycle time (Ttot), inspiratory time (T₁), expiratory time, and T₁/Ttot were calculated from the flow signal as average values from 5 min of continuous recording of flow and volume. From the Pao signal, peak Pao was computed. PEEPi, dyn was measured as the negative deflection in Pdi from the onset of inspiratory effort to the onset of inspiratory flow. In the presence of expiratory muscle activity, that value of PEEPi, dyn was reduced by the decrease in gastric pressure to enable subtraction of the decrease in Pes preceding flow caused by expiratory muscle relaxation rather than inspiratory muscle contraction to counterbalance PEEPi, dyn.\(^{14}\) Changes in the magnitude of the inspiratory effort of the diaphragm were estimated from changes in Pdi, as previously described.\(^{14}\) We measured Pdi tidal swings as well as the pressure-time product for the diaphragm per breath (PTPdi/b) calculated for a period of 1 min (PTPdi/min) and also corrected per liter of ventilation (PTPdi/Ve). We used the pressure-time product as an index of inspiratory muscle energy expenditure as it has been shown to be correlated with the oxygen consumption of the respiratory muscles.\(^{20}\)

Patients’ inspiratory efforts unable to trigger a new ventilator cycle despite a negative deflection in Pes were defined as ineffective efforts (IE).\(^{21}\) The mean number per minute of IE recorded for 3 to 6 min was expressed as percent of patient respiratory rate (IE/min/T × 100).

**Experimental Procedure**

The patients were free to choose the most comfortable position so as to minimize their breathlessness. All patients adopted a semirecumbent position. In all patients, after application of a topical anesthesia (lidocaine [Xylocaine] spray, 10%), two balloon-tipped catheters were consecutively inserted through the nose into the stomach. The occlusion test was performed to verify the correct positioning of the esophageal balloon, and it was satisfactory in every instance.\(^{22}\) After instrumentation of the patients, the commercial nose mask used at home by each patient was applied and connected to the pneumotachograph. The commercial mask had adequate size to fit to each patient’s nose. Special care was devoted to ensure mouth closure throughout the procedure. A nurse not involved in the procedure was always present for patient care. The ventilator circuit was equipped with a non-rebreathing valve (Sanders NRV-2; Respiration; Merryville, PA) to prevent CO₂ rebreathing.\(^{19}\) All measurements were recorded at a fraction of inspired oxygen (FiO₂) of 0.21.

For the experimental setting, NPSV was delivered by means of a portable ventilator (BiPAP; Respironics). At the beginning of the study (baseline) and in the intervals between different settings, each subject breathed spontaneously through the pneumotachograph for about 20 to 25 min, once removed from the ventilator tubing. This period of time was long enough to bring all the physiologic variables measured back to baseline values. A set of data was collected under this control condition, that is, while the patient was breathing spontaneously. At the end of this baseline recording, each patient performed NPSV from a semirecumbent position, according to two settings:

1. **Usual setting (U):** the setting actually used by individual patients at home (see “Home Mechanical Ventilation” section, above).

2. **Physiologic setting (PHY):** the level of peak IPS able to achieve > 40% and < 90% decrease in Pdi in comparison to SB, avoiding a positive deflection of Pdi. A PEEPi level able to reduce PEEPi, dyn by ≥ 50% was added. Because of the characteristics of the portable ventilator used, a default 2 cm H₂O PEEPi was always added.

U and PHY were applied in random order, each mode of support lasting 30 to 45 min, and the trials were separated by returning to SB for 20 to 25 min. All physiologic signals were recorded in
the last 3 min of each unassisted or assisted breathing period. Arterial blood was sampled from the radial artery at the end of the initial unassisted breathing period and at the end of each setting of NPSV.

Statistical Analysis

Results are expressed as mean ± SD. Differences between treatments and within treatment were evaluated by analysis of variance (ANOVA) for repeated measures. Differences between paired groups of data were evaluated with post hoc paired Student’s t test with Bonferroni adjustment and were applied as requested by ANOVA interaction. A χ² analysis was performed to evaluate differences between the two groups in number of patients showing IE and of those reaching the target Pdi and pressure-time product. A p value showing IE and of those reaching the target Pdi and pressure-time product. A p value < 0.05 was considered significant.

RESULTS

All patients tolerated NPSV well throughout the procedure. No patients reported side effects or refused the procedure. The mean U for IPS was 16 ± 3 cm H₂O, and for PEEPe, 3.6 ± 1.4 cm H₂O; whereas the mean PHY for IPS was 15 ± 3 cm H₂O; and for PEEPe, 3.1 ± 1.6 cm H₂O without any significant difference between the two settings.

In comparison with U, PHY resulted in a greater level of IPS in 9 patients (39%) and of PEEPe in 7 of 23 patients (30%), whereas it resulted in a lower level of IPS in 12 patients (52%) and of PEEPe in 13 patients (56%). IPS and PEEPe remained unchanged in two and three patients, respectively.

NPSV was able to significantly (p < 0.01) improve both Pao₂ (from 49.7 ± 5.5 to 55.1 ± 7.7 and to 54.6 ± 7.5 mm Hg with U and PHY, respectively) and PaCO₂ (from 58.3 ± 7.0 to 53.0 ± 6.1 and to 53.0 ± 6.1 mm Hg with U and PHY, respectively) independent of the applied setting. Arterial pH increased, but not significantly, with both settings of NPSV (from 7.39 ± 0.02 to 7.40 ± 0.03 and to 7.40 ± 0.02 with U and PHY, respectively).

Mean changes in breathing pattern and Ve induced by the two settings of NPSV are shown in Table 2. When compared with SB, both settings induced a significant increase in Vt and a significant reduction in f, resulting in a significant increase in Ve, again without any significant difference between the two settings.

Table 3 illustrates the mean values of diaphragm function and PEEPI,dyn recorded in the three experimental conditions. As shown in Table 3, compared with SB, both ventilator settings induced significant reductions in all the variables of diaphragm effort, as assessed by PTPdi,min, PTPdi/b, and PTPdi/Ve. This reduction was significantly different between U and PHY. Figure 1 compares the individual Pdi changes from the values of SB with the two settings. The target 40% reduction in Pdi was obtained in significantly (p < 0.01) fewer patients during U (15 [65%]) than during PHY (22 [96%]). In none of the patients under PHY was a Pdi reduction greater than the target 90% found, whereas it was observed in three patients (13%) under U (p < 0.01; Fig 1).

Figure 2 is the identity plot comparing PEEP,dyn during SB and the PEEPe applied with U. In 13 of 23 patients, the PEEPe was applied to levels higher than PEEP,dyn. As shown in Table 3, the mean reduction in PEEP,dyn was significantly (p < 0.01) higher with PHY than with U. Figure 3 is the identity plot comparing the individual PEEPI,dyn changes from the SB values with the two settings. A PEEPI,dyn reduction less than the target 50% of SB was observed in six patients (26%) with U and in three patients (13%) with PHY; however, two patients under U and one patient under PHY showed a PEEPI,dyn increase. These differences were not statistically significant (Fig 3).

Eleven of 23 (48%) patients with U and 7 of 23 (30%) patients with PHY showed IE: this difference was not significant. The prevalence of IE (20 ± 39%) of their f vs 6 ± 11% with U and PHY, respectively) was statistically different (p < 0.05).

Table 2—Ventilatory Pattern During SB and Different Settings of NPSV*

<table>
<thead>
<tr>
<th>Variable</th>
<th>SB</th>
<th>U</th>
<th>PHY</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ve, L/min</td>
<td>9.2 ± 2.3</td>
<td>11.4 ± 4.2†</td>
<td>11.7 ± 3.9†</td>
</tr>
<tr>
<td>Vt, mL</td>
<td>484 ± 184</td>
<td>747 ± 217†</td>
<td>751 ± 187†</td>
</tr>
<tr>
<td>f, bpm</td>
<td>19.8 ± 3.5</td>
<td>15.3 ± 4.4†</td>
<td>15.3 ± 2.8†</td>
</tr>
<tr>
<td>Ttot/Ttot</td>
<td>0.36 ± 0.06</td>
<td>0.35 ± 0.07</td>
<td>0.36 ± 0.07</td>
</tr>
</tbody>
</table>

*Values given as mean ± SD.
†p < 0.01 vs baseline (ANOVA test); differences between U and PHY were not statistically significant.

Table 3—I E and PEEPI,dyn During SB at Different Settings of NPSV*

<table>
<thead>
<tr>
<th>Variable</th>
<th>SB</th>
<th>U</th>
<th>PHY</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pes, cm H₂O</td>
<td>13 ± 5.2</td>
<td>5.5 ± 3.7†</td>
<td>5.5 ± 2.5†</td>
</tr>
<tr>
<td>Pdi, cm H₂O</td>
<td>15 ± 5</td>
<td>7.0 ± 4.4†</td>
<td>6.6 ± 2.8†</td>
</tr>
<tr>
<td>PEEPI,dyn cm H₂O</td>
<td>3.22 ± 2.30</td>
<td>1.41 ± 1.51†</td>
<td>0.68 ± 1.04†</td>
</tr>
<tr>
<td>PTPdi/h, cm H₂O</td>
<td>17.97 ± 7.25</td>
<td>9.81 ± 7.11†</td>
<td>8.53 ± 4.88†</td>
</tr>
<tr>
<td>PTPdi/min, cm H₂O</td>
<td>347 ± 136</td>
<td>152 ± 116†</td>
<td>126 ± 83†</td>
</tr>
<tr>
<td>PTPdi/Ve, cm H₂O/L</td>
<td>38.5 ± 18.2</td>
<td>13.6 ± 11.0†</td>
<td>10.77 ± 7.11†</td>
</tr>
</tbody>
</table>

*Values given as mean ± SD. Differences between U and PHY for Pes and Pdi were not statistically significant.
†p < 0.01 vs baseline (ANOVA).
‡p < 0.01 vs U (ANOVA).
§p < 0.05 vs U (ANOVA).

CHEST / 118 / 5 / NOVEMBER, 2000 1289
In stable COPD patients with chronic hypercapnia, NPSV is effective in improving arterial blood gases and in unloading inspiratory muscles independent of whether it is set on the basis of patient comfort or tailored to a patient’s respiratory muscle effort and mechanics. However, a tailored setting of NPSV may result in reduction in ineffective inspiratory efforts.

The large majority of the so-called home-care ventilators perform as well as traditional and more expensive ICU ventilators.9,23 This introduces the importance of correctly setting the ventilator factors once a mode has been decided on. The operator has to adjust several settings on the ventilator: the inspiratory24 and expiratory25 triggers, the initial flow rate,26 and the minimum target volume to reach, but their best setting and clinical utility have been seldom demonstrated. Reviewing articles from 1990 to 1999 on long-term NPPV (source, MEDLINE; key words, noninvasive mechanical ventilation, chronic respiratory insufficiency), shows that 74% of the authors describe in details the setting adopted for their studies: among these, 19% used only clinical variables such as the maximal tolerated IPS or the patient’s comfort as in our U setting, and 36% used only changes in physiologic variables such as target arterial blood gases and breathing pattern, whereas 45% used both modalities to set ventilators. Apart from physiologic studies,16,27,28 in no clinical study was invasive evaluation of respiratory mechanics and muscle used.

Different levels of IPS may be needed in different patients to achieve the same target VT and a 40 to 50% reduction in inspiratory muscle activity. In patients similar to those of this study (ie, stable patients with hypercapnic COPD) and using similar techniques (ie, esophageal and gastric balloons), Nava and colleagues16 found that NPSV at 10 and 20 cm H2O improved arterial blood gases and unloaded the diaphragm. On average, the changes in PaCO2 and PaO2 were in the same range of the present study, ie, a few millimeters of mercury. In that study,16 with a preset level of assistance of 20 cm H2O of IPS and 5 cm H2O of PEEP, the mean reduction in PTPdi was 60% of SB with a wide individual variation. This value was similar to those found in our study with both U and PHY, ie, with lower mean levels of mask pressure.

The combined use of inspiratory and expiratory aids during pressure-assisted ventilation has been
shown both in chronic and acute settings to provide better unloading of the diaphragm and in some instance to improve arterial blood gas levels, as compared with inspiratory assistance alone. In the stable COPD patients of the study by Nava et al., addition of 5 cm H₂O PEEPe to PSV of 10 cm H₂O resulted in a further 42% reduction of PTPdi. The addition of a PEEPe reduces the amount of the inspiratory load by decreasing the magnitude of PEEPi,dyn. Baseline levels of PEEPi,dyn of our patients were in line with the low levels reported in stable patients with COPD. It has been suggested to avoid setting PEEPe above the level of PEEPi,dyn as this may determine a significant rise in end-expiratory lung volume, and therefore a greater hyperinflation. In clinical practice, when a direct measurement is lacking, it is usually recommended not to overcome 4 to 6 cm H₂O unless clinical evidence suggests the presence of marked dynamic hyperinflation. In our study, both settings induced a mean reduction in PEEPi,dyn (by 63% and 79% with U and PHY, respectively). Nevertheless, with U in 13 of 23 patients, the applied PEEPe was higher than PEEPi,dyn (Fig 3). Although excessive PEEPe might be likely to result in lung hyperinflation, we actually were unable to demonstrate it in our patients as we did not measure the changes in end-expiratory lung volume. An insufficient PEEPi,dyn compensation may decrease the trigger sensitivity to significantly reduce the threshold needed to initiate the inspiratory flow, which has been shown to account for up to 40% of the whole inspiratory burden.

In ventilator-dependent patients, the phenomenon of missing efforts or IE (ie, Pes swings unable to trigger the ventilator) has been shown. High resistance to airflow, PEEPi,dyn, low elastic recoil, high ventilatory demands, and short expiratory time on the ventilator have been advocated to explain this phenomenon in mechanically ventilated patients. To our knowledge, our study is the first one to describe the presence of IE also in stable COPD patients undergoing long-term home NPSV. With U, 48% of patients showed IE in 20% of their respiratory rate, whereas with PHY, IE decreased to 6% in 25% of patients. This result suggests that the setting of the ventilator may be crucial: in fact, by resetting the ventilator on the basis of the invasive evaluation (either increasing or decreasing pressure support or PEEPe), it was possible to reduce the incidence of IE by 70% in 36% of patients.

Considering the invasiveness required by setting PHY, its real usefulness may be questionable. Nevertheless, some points deserve to be addressed. In this study, the U setting had been prescribed for
home use by physicians expert in the field. Therefore, we are confident that differences between U and PHY were related to the settings per se. It may be conceivable that, if applied by less expert caregivers, these discrepancies might be enhanced. Furthermore, although also setting U was able to induce reductions in mean values of Pdi, PTPdi, and PEEPi,dyn, individual changes were not as satisfactory (Figs 1, 3), and IE were more frequent.

Limitations of Study

Our study was performed in awake patients whereas home NPPV is usually (and actually in our patients it was) prescribed at night. Therefore, the correct ventilator setting should theoretically be tested during a formal sleep study. Nevertheless, we reasoned that the lack of information on the physiologic effect of this setting of NPSV in those patients would warrant a daytime investigation, in particular when one takes into account the techniques needed to measure a patient’s respiratory muscle function, for example, with the esophageal balloon. Furthermore, daytime mechanical ventilation in awake patients was reported to be equally effective in reversing chronic hypercapnia as nocturnal mechanical ventilation.31

The U setting was applied according to the previous physician’s decision (months, if not years, before) and was the one usually used at home by patients. This might be considered as a bias. Nevertheless, this hypothetical bias should be to the advantage of the U setting, eg, for a better compliance (not assessed) or even for respiratory muscle unloading. This was not actually the case.

Although 23 patients in this study is a small number for a clinical follow-up study, a preliminary sample size had shown that it was enough for a physiologic study such as this. Nevertheless, before generalization, these short-term results must be confirmed in the long-term clinical setting. The present study must be considered a pilot study with physiologic information rather than a real clinical study owing to the lack of a follow-up.

In conclusion, in COPD patients with chronic hypercapnia, NPSV is effective in improving arterial blood gases and in unloading inspiratory muscles independent of whether it is set on the basis of patient comfort and improvement in arterial blood gases or tailored to the patient’s respiratory muscle effort and mechanics. However, setting NPSV by invasive evaluation of lung mechanics and respiratory muscle function may result in reduction in ineffective inspiratory efforts. Finally, despite the expertise of the teams, the U setting resulted in almost 50% of
discrepancies with the PHY setting, most of them resulting in IE. Therefore, an empirical setting may be not appropriate for a generalized use. However, these short-term results must be confirmed in the long-term clinical setting.

REFERENCES


4. Lin CC. Comparison between nocturnal nasal positive pressure ventilation combined with oxygen therapy and oxygen monotherapy in patients with severe COPD. Am J Respir Crit Care Med 1996; 154:353–358


学霸图书馆

www.xuebalib.com

本文献由“学霸图书馆-文献云下载”收集自网络，仅供学习交流使用。

学霸图书馆（www.xuebalib.com）是一个“整合众多图书馆数据库资源，提供一站式文献检索和下载服务”的24小时在线不限IP图书馆。

图书馆致力于便利、促进学习与科研，提供最强文献下载服务。

图书馆导航：
图书馆首页    文献云下载    图书馆入口    外文数据库大全    疑难文献辅助工具