Comparative Physiologic Effects of Noninvasive Assist-Control and Pressure Support Ventilation in Acute Hypercapnic Respiratory Failure*

Christophe Girault, MD; Jean-Christophe Richard, MD; Virginie Chevron, MD; Fabienne Tamion, MD; Pierre Pasquis, MD; Jacques Leroy, MD; and Guy Bonmarchand, MD

**Study objective:** To compare the effects of noninvasive assist-control ventilation (ACV) and pressure support ventilation (PSV) by nasal mask on respiratory physiologic parameters and comfort in acute hypercapnic respiratory failure (AHRF).

**Design:** A prospective randomized study.

**Setting:** A medical ICU.

**Patients and interventions:** Fifteen patients with COPD and AHRF were consecutively and randomly assigned to two noninvasive ventilation (NIV) sequences with ACV and PSV mode, spontaneous breathing (SB) via nasal mask being used as control. ACV and PSV settings were always subsequently adjusted according to patient’s tolerance and air leaks. Fraction of inspired oxygen did not change between the sequences.

**Measurements and results:** ACV and PSV mode strongly decreased the inspiratory effort in comparison with SB. The total inspiratory work of breathing (WOBinsp) expressed as WOBinsp/tidal volume (VT) and WOBinsp/respiratory rate (RR), the pressure time product (PTP), and esophageal pressure variations (ΔPes) were the most discriminant parameters (p<0.001). ACV most reduced WOBinsp/VT (p<0.05), ΔPes (p<0.05), and PTP (0.01) compared with PSV mode. The surface diaphragmatic electromyogram activity was also decreased >32% as compared with control values (p<0.01), with no difference between the two modes. Simultaneously, NIV significantly improved breathing pattern (p<0.01) with no difference between ACV and PSV for VT, RR, minute ventilation, and total cycle duration. As compared to SB, respiratory acidosis was similarly improved by both modes. The respiratory comfort assessed by visual analog scale was less with ACV (57.23±30.12 mm) than with SB (75.15±18.25 mm) (p<0.05) and PSV mode (81.62±25.2 mm) (p<0.01) in our patients.

**Conclusions:** During NIV for AHRF using settings adapted to patient’s clinical tolerance and mask air leaks, both ACV and PSV mode provide respiratory muscle rest and similarly improve breathing pattern and gas exchange. However, these physiologic effects are achieved with a lower inspiratory workload but at the expense of a higher respiratory discomfort with ACV than with PSV mode.

(CHEST 1997; 111:1639-48)

**Key words:** acute hypercapnic respiratory failure; assist-control ventilation; breathing pattern; chronic obstructive pulmonary disease; noninvasive ventilation; pressure support ventilation; respiratory comfort; work of breathing

**Abbreviations:** ABG=arterial blood gas; ACV=assist-control ventilation; AHRF=acute hypercapnic respiratory failure; Cw=chest wall compliance; EMGdi=surface diaphragmatic electromyogram; PetCO₂=end-tidal carbon dioxide fraction; FIO₂=fraction of inspired oxygen; NIV=noninvasive ventilation; Paw=airway pressure; PEEP=positive end-expiratory pressure; PEEPi=dynamic intrinsic PEEP; pEMGdi=maximal or peak amplitude of the integrated EMGdi signal; Pes=end-expiratory pressure; PetCO₂=end tidal carbon dioxide pressure; PSV=pressure support ventilation; PTP=pressure time product; RR=respiratory rate; SaO₂=arterial oxygen saturation; SB=spontaneous breathing; TE=expiratory time; TI=inspiratory time; Ttot=total cycle duration; VAS=visual analog scale; VT=minute ventilation; VT/VTI=inspiratory work; WOBinsp=total inspiratory work of breathing

---

*From the Medical Intensive Care Unit (Drs. Girault, Richard, Chevron, Tamion, Leroy, and Bonmarchand) and the Department of Respiratory Physiology (Dr. Pasquis), Charles Nicolle University Hospital, Rouen, France.

Manuscript received May 30, 1996; revision accepted December 30.

Reprint requests: Christophe Girault, MD, Service de Réanimation Médicale, Hôpital Charles Nicolle, Centre Hospitalier et Universitaire de Rouen, 76031 Rouen cedex, France

---

Four prospective and randomized controlled studies have now well demonstrated the usefulness and established the clinical efficacy of noninvasive ventilation (NIV) in the treatment of patients with acute respiratory failure. More accurately, selected patients with acute hypercapnic respiratory...
failure (AHRF)\(^5\) with\(^1,3,4\) or without COPD\(^2\) may potentially benefit from NIV. In such conditions, NIV could avoid the need for endotracheal intuba-

ion in 50% to 70% of cases and reduce the length of ICU stay and mortality rate.\(^2,3,6\) Performed via nasal or face mask, NIV may use volume assist-control ventilation (ACV)\(^1\) or pressure support ventilation (PSV).\(^2,6\) According to numerous studies, ACV\(^1,7,8\) and PSV\(^2,6,9,10\) may provide similar clinical results regarding success or failure in terms of the need for endotracheal intubation or not. Nevertheless, there are potentially important differences between these two NIV modes. For example, PSV may compensate for mask air leaks better than ACV but may deliver volumes less reliably in case of variable respiratory mechanics, especially in COPD patients. These findings, therefore, could have deleterious consequences on respiratory physiologic parameters and comfort and subsequently on clinical efficacy of NIV.

In the literature, to our knowledge, only one prospective randomized study has attempted to compare the clinical usefulness of both noninvasive ACV and PSV mode in 29 COPD patients with AHRF.\(^11\) The authors reported no significant clinical difference in terms of success rate between the two NIV modes. However, on a physiologic basis, the most appropriate ventilatory mode for NIV in AHRF is not yet known. To our knowledge, no study has been performed to compare the physiologic effects of these two NIV modalities during AHRF.

The objective of this study, therefore, was to compare the effects of noninvasive ACV and PSV mode via nasal mask on respiratory physiologic parameters and on respiratory comfort in COPD patients with AHRF.

**Materials and Methods**

The study was conducted in a medical ICU and approved by the Ethical Committee of the Charles Nicolle University Hospital. All patients or their families gave a written informed consent. Patients enrolled in the study had known COPD or a high probability of the disease on the basis of the clinical history, results of physical examination, chest radiograph, and/or previous pulmonary function tests data. Additional criteria for enrollment included AHRF requiring NIV according to the following criteria:\(^5,9\) polyphyes > 25/min or bradypnea ≤ 12/min; PaO\(_2\) ≤ 8 kPa (60 mm Hg) in ambient air; PaCO\(_2\) ≥ 6.5 kPa (49 mm Hg) in ambient air or worsening with a low nasal oxygen flow (≤ 3 L/min), respiratory acidosis (pH ≤ 7.35), a normal level of consciousness, or moderate signs of respiratory encephalopathy (drowsiness, confusion, flapping tremor). Patients were included if they showed at least three of these criteria and if they tolerated NIV during the first few hours after hospital admission. Patients with AHRF requiring immediate endotracheal intubation or any contraindication to the insertion of an esophageal tube were not included.

**Study Protocol**

The study was conducted during the 48 h following admission to the hospital for AHRF. Each patient was investigated in a fasting state, in a semirecumbent position in bed, and in quiet conditions. Throughout the study, patients were asked to close their mouth firmly to limit the deleterious effect of air leaks.\(^12\) All patients were consecutively and randomly assigned to two NIV sequences with ACV and PSV mode performed via the same ventilator and nasal mask for a minimal duration of 30 min. These two NIV sequences were preceded and separated by a run-in spontaneous breathing (SB) period of 30 min allowing us to define baseline parameters. The SB sequence was performed with additional inspired oxygen through the nasal mask and the ventilator circuit without pressure support level. Patients were informed of the change in sequence, but they were not aware of the type of ventilatory mode used (SB, ACV, or PSV).

Initial settings in ACV mode were those usually reported with NIV:\(^9,13\) insufflated tidal volume (VT) of 15 mL/kg; respiratory rate (RR) of 12 to 20 cycles/min; inspiratory time (TI)/expiratory time (TE) ratio of 1/2 to 1/3; inspired oxygen fraction (FiO\(_2\)) enabling arterial oxygen saturation (SaO\(_2\)) ≥ 90%; no positive end-expiratory pressure (PEEP); constant inspiratory flow rate of 60 L/min; and trigger sensitivity of ~0.5 cm H\(_2\)O. Ventilatory settings in PSV mode were as follows:\(^9,11\) maximal tolerated inspiratory pressure support level (15 to 20 cm H\(_2\)O) able to achieve an expiratory VT ≥ 10 mL/kg, FiO\(_2\) similar to ACV mode; no PEEP; constant inspiratory flow rate of 60 L/min; constant inspiratory flow time of 0.5 L/s, and trigger sensitivity of ~0.5 cm H\(_2\)O. In all cases, these initial settings (VT, RR, and PSV level) were always subsequently adjusted in relation to the patient’s clinical tolerance and the presence or lack of air leaks around the nasal mask before measurements were performed. FiO\(_2\) did not change between SB, ACV, and PSV sequences. All measurements were performed at the end of each ventilation sequence after at least 30 min of stable and appropriate ventilation.

Patients were observed clinically by a physician not involved in the procedure, as well as by continuous monitoring of heart rate, noninvasive BP, and transcutaneous oximetry (Biox 3700; Ohmeda Inc; Boulder, Colo).

**Measurements**

All patients were ventilated using the same pressure-triggered ventilator (Evita 2; Dräger Medical Inc; Lübeck, Germany). They were connected to the ventilator via a comfortable tightly fitted nasal mask (Respironics; Murrysville, Pa). We also disconnected the humidifier of the ventilator circuit during the trial to decrease the workload necessary to overcome the circuit resistances. The following general data were recorded for all patients: history; current diseases and treatment; arterial blood gas (ABG) and simplified acute physiologic score on hospital admission.\(^14\) Each patient was investigated using the same apparatus. Instantaneous flow rate was measured using a pneumotachograph (Fleisch No. 1; Zurich, Switzerland) connected to a differential pressure transducer (Validyne MP45; Validyne Corp; Northridge, Calif). The pneumotachograph was connected between the nasal mask and the Y-piece of the ventilator circuit. Corresponding volume variations (VT) were obtained by electrical integration of instantaneous flow rates. Minute ventilation (Ve) was defined as the product of VT and RR. Airway pressure (Paw) was measured at the nasal mask by a differential pressure transducer (Validyne...
MP45: Validyne Corp. Esophageal pressure (Pes), reflecting intrapleural pressure, was measured using the method described by Milic-Emili et al.\textsuperscript{15,16} via a latex balloon placed in the middle third of the esophagus and connected to a differential pressure transducer (Validyne MP45; Validyne Corp). The proper position of the balloon was confirmed by an occlusion test.\textsuperscript{17} Variations or swings in Pes (ΔPes), considered to be a parameter of inspiratory effort, were measured as the difference between minimum and maximum Pes.\textsuperscript{12} Dynamic intrinsic PEEP (PEEP dyn), reflecting the end of expiratory pressure gradient between the alveoli and the nasal mask, was measured on the Pes curve as the difference between point 0 and the Pes point corresponding to cancellation of inspiratory flow.\textsuperscript{18} It was averaged from 10 representative respiratory cycles. Total inspiratory work of breathing (WOBinsp) was determined using the diagram method of Campbell et al.\textsuperscript{19} by integration of the area plotted between the pressure-volume (Pes-Vr) and the chest wall compliance (Cw) curves\textsuperscript{20,21} (Fig 1). Since it is impossible to measure Cw without complete relaxation, we assumed as others have\textsuperscript{22,23} that it was equal to 4% of the theoretical vital capacity.\textsuperscript{24} The WOBinsp was calculated from the mean of five cycles and expressed in joules. The WOBinsp thus enabled calculation of WOBinsp in relation to Vr (WOBinsp/Vr in joules/L) and to RR (WOBinsp/RR in joules/min). The pressure time product (PTP), a better index of the energy expenditure of the respiratory muscles,\textsuperscript{25,26} was obtained from the product of TI and area under the Pes curve corresponding to TI. In addition to ΔPes, WOBinsp, and PTP, respiratory muscle activity was evaluated by the diaphragmatic electromyogram (EMGdi) activity amplitude with bipolar skin surface electrodes.\textsuperscript{27} The EMGdi signal obtained (raw EMGdi) was then amplified (Universal Amplifier 13-4615-58; Gould Electronics Inc; Cleveland) and filtered (30 to 300 Hz), then rectified and electronically integrated every 100 ms (integrated EMGdi) to obtain a moving time average, which was then analyzed using the method of Lopata et al.\textsuperscript{28} The diaphragmatic activity was evaluated by the maximum or peak amplitude of the integrated EMGdi signal (pEMGdi). This quantitative value, averaged from 10 cycles, was expressed as a percentage of the control value obtained in SB mode.

The end-tidal carbon dioxide fraction (FETCO\textsubscript{2}) and SaO\textsubscript{2} were measured continuously, respectively, at the mask using a rapidly responding CO\textsubscript{2} analyzer (Medical Gas Analyzer LB-2; Sensor Medics Corp; Anaheim, Calif) and by transcutaneous pulsed oximetry (Biox 3700; Ohmeda Inc). ABGs, sampled by radial catheter at the end of each ventilation period, were immediately analyzed (ABL3; Radiometer Inc; Copenhagen, Denmark). The end-tidal CO\textsubscript{2} pressure (PETCO\textsubscript{2}) was calculated from the cycles in which Vt, TI, TE, total cycle duration (Ttot), and PEF were measured.

All signals (flow, Vt, Paw, FETCO\textsubscript{2}, and EMGdi) were simultaneously displayed and recorded using a polygraphic recorder (ES 2000 V12, Gould Electronics Inc) (Fig 2). Polysgraphic recordings of 10 to 15 respiratory cycles with a paper speed of 10 and 50 mm/s were used to measure Vt, TI and TE, Ttot, RR, mean inspiratory flow (Vr/TI), relative inspiratory time (TI/Ttot), and PEF for each cycle. They were then averaged from the entire tracing. A digitization table (Hewlett Packard 9874 A) and a personal computer (Hewlett Packard 9835 A) were used for measurements and calculations on each entire recording.

The respiratory comfort (level of dyspnea, well-being) was assessed in the last minutes of each ventilation sequence on a 100-mm visual analog scale (VAS). The patient’s status was located between a value of 0 (“I don’t feel at all comfortable”) and 100 (“I feel very comfortable”). The VAS rating was then converted into a numeric value for statistical analysis. Preference for each of the three modes was also noted.

**Statistical Analysis**

The primary end point involved comparison of respiratory muscle function parameters, gas exchange, and breathing pattern between ACV and PSV, using SB mode as control. The secondary end point evaluated respiratory comfort with these two NIV modalities. Qualitative data assessed by VAS were subsequently

---

**Figure 1.** WOBinsp calculation using Campbell’s diagram method by integration of the area plotted between the pressure-volume (Pes-Vr) and the chest wall compliance (Cw) curves from three representative respiratory cycles during NIV with ACV and PSV mode in patient 4. Note the decrease in WOBinsp between SB and ACV or PSV mode and that WOBinsp is close to the relaxation curve of the chest wall with ACV mode.
The analysis showed ventilatory individual chial superinfection 15 <0.05 on hospital with two PSV and converted into numeric values in millimeters for statistical analysis. The two SB periods were compared using a Wilcoxon’s test for paired data. The lack of significant difference between the two SB periods parameters allowed us to use the first sequence in SB mode as the control period. Comparison of results among SB control period, ACV, and PSV sequences was performed using an analysis of variance for repeated measures. If significant (p<0.05), a Dunnet’s t test was performed to compare values obtained with ACV and PSV with those obtained in SB mode or on hospital admission. Results were expressed as mean±SD. A difference was considered as statistically significant for an alpha probability <0.05 (p<0.05).

RESULTS

The main clinical and respiratory characteristics of the 15 COPD patients are shown in Table 1. All patients showed evidence of severe AHRF requiring NIV. These AHRF episodes were related to bronchial infection in 14 cases and pneumonia in one case (patient 3). Table 1 also shows the main individual ventilatory settings finally used with ACV and PSV mode during the trial.

In comparison with SB mode, NIV strongly decreased the inspiratory effort. The WOBinsp parameters (WOBinsp/cycle, WOBinsp/Vt, WOBinsp/RR), PTP, ΔPes, and pEMGdi were significantly decreased with ACV and PSV modes (Table 2). The reduced inspiratory muscle effort was more reflected by WOBinsp/Vt, WOBinsp/RR, PTP, and ΔPes (p<0.001) and all patients demonstrated a decrease in their individual values (Fig 3). Noninvasive ACV reduced inspiratory effort more than PSV mode as illustrated by WOBinsp/Vt (p<0.05), ΔPes (p=0.02), and PTP (0.01) (Table 2 and Fig 3). The surface pEMGdi was also decreased >32% compared with control values (p<0.01) without any difference between the two modes (Table 2).

The effects of noninvasive ACV and PSV on breathing pattern are shown in Table 3. In comparison with SB mode, NIV significantly improved
breathing pattern (p<0.01) with no difference between ACV and PSV for Vt, RR, RB, and Ttot. The improvement in Vt was achieved by a significant increase in Vt (p=0.001), while a parallel decrease in RR was observed (p<0.01) with the two modalities. The decrease in RR was related to an increase in Ttot with both NIV modes. The increase in Ttot was found to be due to a significant decrease in TI with ACV mode only (p<0.01) and to a significant increase in TE (p<0.001), higher with ACV than with PSV mode (p<0.05). Simultaneously, Vt/RR also significantly improved with NIV as compared with SB mode (p<0.001), much more with ACV than with PSV mode (p<0.001).

The comparison of ABG values on hospital admission to those obtained with both NIV modes revealed a significant correction of PaO2 and SaO2 (p<0.001) and a parallel improvement in the initial hypercapnia and respiratory acidosis with no difference between ACV and PSV (Table 4). During the trial, oxygenation parameters did not change among the three sequences since FIO2 was maintained constant, except for PaO2/FIO2 with ACV mode. Among alveolar ventilation parameters, baseline pH, PaCO2, and PETCO2 were significantly and similarly improved with the two NIV modes (Table 4).

With regard to the respiratory comfort, ACV (57.23±30.12 mm) was found to be more uncom-

---

Table 1—Clinical and Respiratory Characteristics of the 15 COPD Patients on Hospital Admission and Settings Used With ACV and PSV Mode

<table>
<thead>
<tr>
<th>Patient No./Sex/Age</th>
<th>FEV1, % pred</th>
<th>VC, % pred</th>
<th>FEV1/VC, %</th>
<th>PaO2, mm Hg</th>
<th>PaCO2, mm Hg</th>
<th>pH</th>
<th>SAPS</th>
<th>Vt, mL</th>
<th>Set Rate, cycles/min</th>
<th>Assisted Breaths</th>
<th>FIO2, %</th>
<th>PSV Level, cm H2O</th>
</tr>
</thead>
<tbody>
<tr>
<td>I/M/54</td>
<td>57</td>
<td>24</td>
<td>60</td>
<td>33</td>
<td>52.35</td>
<td>67.05</td>
<td>7.33</td>
<td>8</td>
<td>840</td>
<td>12</td>
<td>19.9</td>
<td>100.3</td>
</tr>
<tr>
<td>2/F/71</td>
<td>50</td>
<td>—</td>
<td>—</td>
<td>72.07</td>
<td>63.45</td>
<td>11</td>
<td>750</td>
<td>14</td>
<td>14</td>
<td>0</td>
<td>0.3</td>
<td>16</td>
</tr>
<tr>
<td>3/F/65</td>
<td>72</td>
<td>—</td>
<td>—</td>
<td>35.17</td>
<td>53.55</td>
<td>7.35</td>
<td>750</td>
<td>12</td>
<td>16.7</td>
<td>91</td>
<td>0.4</td>
<td>15</td>
</tr>
<tr>
<td>4/F/68</td>
<td>40</td>
<td>32</td>
<td>42</td>
<td>59.79</td>
<td>66.45</td>
<td>10.47</td>
<td>7.27</td>
<td>9</td>
<td>750</td>
<td>18</td>
<td>18.5</td>
<td>50.9</td>
</tr>
<tr>
<td>5/M/52</td>
<td>66</td>
<td>16</td>
<td>37</td>
<td>34.42</td>
<td>49.50</td>
<td>7.34</td>
<td>10</td>
<td>600</td>
<td>18</td>
<td>17.6</td>
<td>64.4</td>
<td>0.14</td>
</tr>
<tr>
<td>6/M/74</td>
<td>69</td>
<td>—</td>
<td>—</td>
<td>4.53</td>
<td>67.50</td>
<td>7.32</td>
<td>14</td>
<td>600</td>
<td>12</td>
<td>15.6</td>
<td>21.5</td>
<td>0.10</td>
</tr>
<tr>
<td>7/M/60</td>
<td>51</td>
<td>23</td>
<td>62</td>
<td>28.95</td>
<td>35.37</td>
<td>3.97</td>
<td>8</td>
<td>700</td>
<td>17</td>
<td>16.2</td>
<td>0</td>
<td>0.10</td>
</tr>
<tr>
<td>8/M/69</td>
<td>106</td>
<td>53</td>
<td>63</td>
<td>62.50</td>
<td>42.15</td>
<td>56.62</td>
<td>7.32</td>
<td>10</td>
<td>800</td>
<td>13</td>
<td>13.8</td>
<td>56.4</td>
</tr>
<tr>
<td>9/M/60</td>
<td>59</td>
<td>20</td>
<td>53</td>
<td>29.84</td>
<td>38.87</td>
<td>66.62</td>
<td>7.32</td>
<td>10</td>
<td>800</td>
<td>12</td>
<td>12.7</td>
<td>0.04</td>
</tr>
<tr>
<td>10/M/68</td>
<td>83</td>
<td>34</td>
<td>59</td>
<td>45.12</td>
<td>61.35</td>
<td>74.47</td>
<td>7.35</td>
<td>12</td>
<td>600</td>
<td>15</td>
<td>18.9</td>
<td>100.3</td>
</tr>
<tr>
<td>11/M/71</td>
<td>79</td>
<td>—</td>
<td>—</td>
<td>82.20</td>
<td>85.72</td>
<td>7.24</td>
<td>10</td>
<td>500</td>
<td>23.3</td>
<td>100</td>
<td>0.15</td>
<td>0.46</td>
</tr>
<tr>
<td>12/M/58</td>
<td>58</td>
<td>32</td>
<td>72</td>
<td>35.37</td>
<td>84.52</td>
<td>72.22</td>
<td>7.33</td>
<td>10</td>
<td>700</td>
<td>15</td>
<td>17.2</td>
<td>55.4</td>
</tr>
<tr>
<td>13/M/70</td>
<td>61</td>
<td>34</td>
<td>48</td>
<td>55.09</td>
<td>83.25</td>
<td>78.15</td>
<td>7.27</td>
<td>11</td>
<td>700</td>
<td>15</td>
<td>15.3</td>
<td>40.0</td>
</tr>
<tr>
<td>14/M/67</td>
<td>50</td>
<td>—</td>
<td>—</td>
<td>74.32</td>
<td>63.00</td>
<td>7.32</td>
<td>9</td>
<td>800</td>
<td>13</td>
<td>19.6</td>
<td>100.0</td>
<td>0.40</td>
</tr>
<tr>
<td>15/M/61</td>
<td>73</td>
<td>24</td>
<td>39</td>
<td>48.05</td>
<td>83.25</td>
<td>97.42</td>
<td>7.30</td>
<td>7</td>
<td>600</td>
<td>20</td>
<td>22.1</td>
<td>15.0</td>
</tr>
<tr>
<td>Mean—64.53</td>
<td>64.27</td>
<td>29.20</td>
<td>53.50</td>
<td>43.21</td>
<td>63.37</td>
<td>71.40</td>
<td>7.32</td>
<td>9.73</td>
<td>682.67</td>
<td>15.0</td>
<td>17.43</td>
<td>54.80</td>
</tr>
<tr>
<td>SD—6.75</td>
<td>16.21</td>
<td>10.43</td>
<td>11.65</td>
<td>12.65</td>
<td>21.97</td>
<td>12.07</td>
<td>0.03</td>
<td>1.87</td>
<td>110.22</td>
<td>2.95</td>
<td>3.02</td>
<td>39.78</td>
</tr>
</tbody>
</table>

*aVC=vital capacity; SAPS=simplified acute physiologic score; FIO2 did not change between ACV and PSV mode.

Table 2—Effects of ACV and PSV Mode on Respiratory Mechanical Parameters and Diaphragmatic Function During NIV in 15 COPD Patients

<table>
<thead>
<tr>
<th>Parameter</th>
<th>SB</th>
<th>ACV</th>
<th>PSV</th>
<th>p Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Paw, cm H2O</td>
<td>0.43±1.05</td>
<td>6.94±2.12</td>
<td>4.56±1.11</td>
<td>0.001</td>
</tr>
<tr>
<td>PEEPi dyn, cm H2O</td>
<td>4.48±2.41</td>
<td>4.90±4.02</td>
<td>5.29±3.42</td>
<td>NS</td>
</tr>
<tr>
<td>∆Pes, cm H2O</td>
<td>21.39±7.09</td>
<td>6.03±4.77</td>
<td>9.8±5.32</td>
<td>0.02</td>
</tr>
<tr>
<td>pEMGdi, %</td>
<td>100</td>
<td>60.47±50.64</td>
<td>67.47±29.81</td>
<td>NS</td>
</tr>
<tr>
<td>WOBinsp, J</td>
<td>0.85±0.4</td>
<td>0.38±0.35</td>
<td>0.52±0.33</td>
<td>NS</td>
</tr>
<tr>
<td>WOBinsp/Vt, J/L</td>
<td>1.89±0.64</td>
<td>0.58±0.52</td>
<td>0.65±0.51</td>
<td>NS</td>
</tr>
<tr>
<td>WOBinsp/RB, J/min</td>
<td>17.06±8.18</td>
<td>6.95±7.05</td>
<td>9.38±6.78</td>
<td>NS</td>
</tr>
<tr>
<td>PTP, cm H2O/s</td>
<td>17.94±6.34</td>
<td>4.05±3.97</td>
<td>8.17±4.95</td>
<td>0.01</td>
</tr>
</tbody>
</table>

*Values are mean±SD. NS=not significant.

1 Dunnett’s t test, SB vs ACV or PSV, <0.001

2 Dunnett’s t test, SB vs ACV or PSV, <0.01

3 p=ACV vs PSV
Figure 3. Individual values of the WOBinsp in relation to VT (WOBinsp/VT) and RR (WOBinsp/RR), of the PTP and esophageal pressure variations (ΔPes) during NIV trial. Note the decrease for all respiratory muscle parameters from SB to ACV and PSV mode with a significant difference between ACV and PSV mode for WOBinsp/VT, PTP, and ΔPes. Statistical analysis (Dunnett’s t test): SB vs ACV or PSV: three asterisks <0.001; p: ACV versus PSV.

This prospective randomized study emphasizes the physiologic mechanisms underlying the clinical

Table 3—Effects of ACV and PSV Mode on Breathing Pattern During NIV in 15 COPD Patients*

<table>
<thead>
<tr>
<th></th>
<th>SB</th>
<th>ACV</th>
<th>PSV</th>
<th>p Value*</th>
</tr>
</thead>
<tbody>
<tr>
<td>VT, mL</td>
<td>439.8±125.28</td>
<td>617.6±116.41</td>
<td>590.4±120.32</td>
<td>NS</td>
</tr>
<tr>
<td>RR, cycles/min</td>
<td>20.27±3.14</td>
<td>17.43±3.02</td>
<td>17.58±4.49</td>
<td>NS</td>
</tr>
<tr>
<td>VE, L/min</td>
<td>8.73±2.06</td>
<td>10.65±1.86</td>
<td>10.32±3.07</td>
<td>NS</td>
</tr>
<tr>
<td>TI, s</td>
<td>1.12±0.18</td>
<td>0.86±0.26</td>
<td>1.28±0.26</td>
<td>0.001</td>
</tr>
<tr>
<td>TE, s</td>
<td>1.92±0.46</td>
<td>2.67±0.64</td>
<td>2.34±0.06</td>
<td>0.05</td>
</tr>
<tr>
<td>Ttot, s</td>
<td>3.04±0.59</td>
<td>3.54±0.61</td>
<td>3.62±0.88</td>
<td>NS</td>
</tr>
<tr>
<td>Tt/Ttot</td>
<td>0.37±0.04</td>
<td>0.25±0.08</td>
<td>0.36±0.05</td>
<td>0.001</td>
</tr>
<tr>
<td>VT/TL, L/s</td>
<td>0.4±0.1</td>
<td>0.77±0.22</td>
<td>0.49±0.13</td>
<td>0.001</td>
</tr>
</tbody>
</table>

*Values are mean±SD. NS=not significant
†Dunnett’s t test, SB vs ACV or PSV, <0.001
‡Dunnett’s t test, SB vs ACV or PSV, <0.01
§Dunnett’s t test, SB vs ACV or PSV, <0.05
‖p=ACV vs PSV

Downloaded From: http://journal.publications.chestnet.org/pdffaccess.ashx?url=/data/journals/chest/20383/ on 06/21/2017
benefit of noninvasive ACV and PSV mode in the management of AHRF in COPD patients. To our knowledge, our study is the first to compare the physiologic effects of ACV and PSV in these circumstances. It shows that, using settings adapted to patient's tolerance and mask air leaks, both ACV and PSV modes may decrease the inspiratory muscle effort and similarly improve breathing pattern and gas exchange. Above all, our study demonstrates that these physiologic effects are achieved with a lower inspiratory workload but at the expense of a higher respiratory discomfort with ACV than with PSV mode.

Despite the total workload under baseline conditions changed from a greater value than the diaphragmatic fatigue threshold to a workload within normal limits, complete muscle rest was not achieved during the trial with the two NIV modes. This was due to the fact that we used two assisted ventilation modes in which RR is determined by patient triggering, with a set back-up rate in ACV mode. During conventional mechanical ventilation, ACV can also allow the persistence of considerable muscle activity, often close to that required with SB. Furthermore, it is well demonstrated that conventional PSV can either totally or partially unload ventilatory muscles during spontaneous breaths according to the PSV level applied. Inhibition of spontaneous inspiratory efforts during ACV and total unloading during PSV could be achieved, respectively, only in a setting of controlled rate or increased alveolar ventilation relative to SB and when the only patient's effort is to trigger the breath as with a PSV level ≥30 cm H2O. These maximal settings could be applied during NIV but should be certainly deleterious on patient's tolerance and respiratory comfort by increasing Paw and thus mask air leaks. Our results demonstrate a significant reduction in all respiratory muscle function parameters (ΔPes, pEMGdi, WOBinsp indexes, PTP) during NIV with both modes in comparison to SB mode. The ΔPes, WOBinsp/Vt, WOBinsp/RR, and PTP were the most discriminant parameters. Comparison of our results to those of previous studies with conventional mechanical ventilation is difficult due to the variability of populations studied; ventilators, settings, and ventilatory modes applied; or WOBinsp calculation used. However, our data support that NIV, with either ACV or PSV mode, can achieve, like endotracheal ventilation, one of the goals of assisted ventilation, which is to provide muscle rest.

In this study, we have also observed that for a similar volume pattern, the inspiratory effort was lower during ACV than during PSV. That was demonstrated by the decrease of all the WOB parameters between ACV and PSV mode, in a significant way for WOBinsp/Vt, ΔPes, and PTP. The total WOBinsp normally includes physiologic (ie, elastic and flow-resistant work) and imposed work of breathing due to the ventilator circuit. During mechanical ventilation, these WOB components are dependent on the underlying disease, the ventilator used, and the settings applied. In our study, these factors did not change between ACV and PSV mode. Settings adjustment before measurements concerned only Vt, RR, and PSV level during ACV and PSV, respectively. Moreover, an additional imposed workload in COPD patients during noninvasive PSV could be due to an increase in PEEPi dyn but this parameter did not change between the two modes. Finally, we can assume that the difference in total WOBinsp between ACV and PSV mode results in lower patient WOB during controlled mechanical breaths in ACV mode. It is likely that a higher but probably more uncomfortable level of pressure support would have resulted in significantly less patient.

Table 4—Effects of ACV and PSV Mode on Gas Exchange vs Hospital Admission and During NIV Trial in 15 COPD Patients*

<table>
<thead>
<tr>
<th></th>
<th>Admission</th>
<th>SB</th>
<th>ACV</th>
<th>PSV</th>
<th>p Value*</th>
</tr>
</thead>
<tbody>
<tr>
<td>PaO2, mm Hg</td>
<td>65.47±21.22</td>
<td>89.10±11.55</td>
<td>100.72±16.13</td>
<td>95.77±20.17</td>
<td>NS</td>
</tr>
<tr>
<td>PaCO2/FiO2, mm Hg</td>
<td>—</td>
<td>233.17±40.94</td>
<td>267.3±76.8</td>
<td>253.52±66.81</td>
<td>NS</td>
</tr>
<tr>
<td>SaO2, %</td>
<td>79.24±23.31</td>
<td>95.95±1.38</td>
<td>97.5±0.76</td>
<td>96.53±1.32</td>
<td>NS</td>
</tr>
<tr>
<td>pH</td>
<td>7.32±0.03</td>
<td>7.35±0.05</td>
<td>7.42±0.06</td>
<td>7.39±0.03</td>
<td>NS</td>
</tr>
<tr>
<td>PaCO2, mm Hg</td>
<td>71.62±12.52</td>
<td>63.82±10.20</td>
<td>54.82±10.57</td>
<td>59.10±9.67</td>
<td>NS</td>
</tr>
<tr>
<td>HCO3, mmol/L</td>
<td>35.76±4.54</td>
<td>35.01±3.69</td>
<td>34.09±3.91</td>
<td>34.6±4.05</td>
<td>NS</td>
</tr>
<tr>
<td>PCO2, mm Hg</td>
<td>—</td>
<td>48.63±8.30</td>
<td>43.12±8.26</td>
<td>45.01±8.36</td>
<td>NS</td>
</tr>
</tbody>
</table>

*Admission=ABG value on hospital admission in ambient air or with additional O2≤3 L/min. Values are mean±SD; NS=not significant.
1Admission vs ACV or PSV, <0.001
2Dunnett's t test, SB vs ACV or PSV, <0.05
3Dunnett's t test, SB vs ACV or PSV, <0.01
4Dunnett's t test, SB vs ACV or PSV, <0.001
5p=ACV vs PSV

CHEST 111 / 6 / JUNE, 1997 1645
WOB during PSV. Previous studies have found similar patient WOB during conventional ACV and PSV mode.\textsuperscript{35,36} However, the authors studied heterogeneous populations of stable\textsuperscript{35} or acutely ill intubated patients,\textsuperscript{36} they calculated WOB only during assisted mechanical breaths, and they used match settings in volume\textsuperscript{35} or peak Paw\textsuperscript{36} between ACV and PSV mode.

Since the WOBinsp measurement may be difficult with NIV, we combined measurement of the surface EMGdi. It has been shown that during NIV, the surface EMGdi activity was well correlated with that of EMGdi assessed by esophageal electrodes, a more invasive technique.\textsuperscript{12} Our results were found in the same way as WOBinsp parameters, ie, a significant reduction in surface EMGdi amplitude with both ACV and PSV mode. While the interpretation of surface EMGdi activity may be debatable,\textsuperscript{27} its decrease during NIV appears herein to be supported by that of ΔPes and PTP. These parameters, easier to obtain and interpret in clinical practice, have been found to be good indexes of respiratory muscles' energy expenditure and oxygen consumption in response to the ventilatory drive of patients.\textsuperscript{12,25,36}

Our overall results regarding respiratory muscle function are in agreement with those of previous noncomparative physiologic studies on noninvasive ACV\textsuperscript{12} and PSV\textsuperscript{6,39} in patients with AHRF. Those authors did not calculate WOBinsp but also found a significant reduction in transdiaphragmatic pressure, PTP\textsuperscript{6,39} EMGdi activity,\textsuperscript{6,12,39} or ΔPes in patients with COPD or restrictive disease.\textsuperscript{12}

This study confirms the improvement in breathing pattern during NIV and PSV\textsuperscript{5,38} as well as with ACV mode. This was demonstrated by the significant increase in VE, VT, Ttot, and decrease in RR with both ACV and PSV modes. That improvement was also similar between the two modes despite the independent settings we have chosen.

The improvement in gas exchange found in comparison with hospital admission or SB values during the trial reflects ventilatory findings. These results are in agreement with those of previous clinical studies using NIV in acute respiratory failure.\textsuperscript{1-11} Our findings also indicate that we may expect an improvement in alveolar hypoventilation within 30 min after starting NIV with PSV as well as with ACV mode.\textsuperscript{5,8} Our results also support the hypothesis according to which NIV should be more beneficial in patients with AHRF.\textsuperscript{2,5}

Our study also demonstrates that for a similar breathing pattern and despite a lower respiratory workload, ACV was found to be more uncomfortable than both PSV and SB mode. In contrast, respiratory comfort during PSV was similar to that experienced by our patients during SB mode. The discrepancy between the respiratory muscle rest provided and the discomfort experienced by all patients with ACV mode could be related to a slight but significant increase in Paw providing a upper airway discomfort also related in part to the higher inspiratory flow rate observed during ACV compared with PSV mode.\textsuperscript{40} Finally, the same respiratory comfort experienced during both PSV and SB mode certainly speaks for a major role of the loss of control of breathing pattern during ACV mode. Our results are in agreement with those of Vitacca et al,\textsuperscript{11} who also found that PSV was better accepted and tolerated than ACV mode without any difference in clinical results.

The conditions of our study involved the usual emergency situation in which such patients are admitted to the hospital. In these circumstances and contrary to others,\textsuperscript{35} we deliberately have chosen independent settings between ACV and PSV modes. Despite this, our settings provided a similar breathing pattern with the two NIV modalities. We found that a mean VT of 9 mL/kg during NIV was achieved with a mean insufflated tolerated VT of 10.62 mL/kg and a mean maximal tolerated PSV level of 15 cm H\textsubscript{2}O, respectively, in ACV and PSV mode. These NIV settings, obviously lower than those previously reported, in particular with ACV mode,\textsuperscript{8,13} appeared sufficient to significantly decrease the WOBinsp and provide a relevant improvement in breathing pattern and gas exchange. These values were nevertheless subject to interindividual variations in our patients and this had to be taken into account when setting ventilators for NIV. The successful application of NIV through a nasal mask may be limited by air leaks around the nasal prosthesis or through the mouth,\textsuperscript{12} and patients were asked to close their mouths firmly throughout the trial. So, in routine clinical practice, our settings could be inappropriate if air leaks are not well controlled. Nevertheless, it has been suggested that NIV applied during sleep was able to accurately assist ventilation, even if some degree of leakage through the mouth occurred.\textsuperscript{41,42} For these reasons, some prefer to use PSV, which may compensate for mask air leaks better than ACV. However, an inspiratory pressure assist may persist if preset PSV level is not reached due to mask or circuit air leakage that can also cause automated triggering of the demand flow or auto-cycling.\textsuperscript{53} However, one can also increase VT with noninvasive ACV mode to compensate more for mask air leaks. In our NIV experience, auto-cycling in PSV mode or increasing VT in ACV mode both increase the degree of leakage and/or patient-ventilator asynchrony that constitute a cause of intolerance, inefficacy, and therefore failure of the technique. So, we consider
that ventilator settings for NIV had first to take into account patient's tolerance and control of air leaks before increasing PSV level or Vt in ACV mode, and then must be individualized for each patient. Thus, according to our results on inspiratory muscle activity, breathing pattern, gas exchange, and respiratory comfort, settings applied in this study could constitute a good compromise to initially set ventilators with noninvasive ACV or PSV mode.

In conclusion, our study shows that during NIV, using settings adapted to patient's clinical tolerance and nasal mask air leaks, both ACV and PSV modes provide respiratory muscle rest and similarly improve breathing pattern and gas exchange. These physiologic effects are achieved with a lower inspiratory workload, but at the expense of a higher respiratory discomfort with ACV than with PSV mode. The settings used with both modes in our study appear reliable to initially set ventilators for NIV and provide a significant improvement in the respiratory physiologic parameters. Since respiratory comfort appears essential for the acceptance of NIV, it might seem logical to prefer PSV in noninvasive management of AHRF. Nevertheless, at the present time, this choice does not seem more clinically relevant than that of ACV mode in terms of success rate and duration of NIV. These two NIV modalities are therefore probably both useful in management of AHRF. Noninvasive PSV mode should certainly be used with caution in patients with unstable ventilatory drive or respiratory mechanics, and ACV mode should apply more to patients with unreliable respiratory effort.

ACKNOWLEDGMENTS: The authors would like to thank Jean-François Gibon, Françoise Burel, and Yann Lacoume for their valuable technical assistance.

REFERENCES

7 Marino W. Intermittent volume cycled mechanical ventilation via nasal mask in patients with respiratory failure due to COPD. Chest 1991; 99:681-84
12 Carrey Z, Gottfried SB, Levy BD. Ventilatory muscle support in respiratory failure with nasal positive pressure ventilation. Chest 1990; 97:150-58
24 Quanjer P. Standardized lung function testing. Bull Eur Physiopathol Respir 1983; 19(suppl 5):1-95
27 Lansing R, Savelle J. Chest surface recording of diaphragm
Mechanical Ventilation

The First Annual Symposium and Workshop for Critical Care Providers

June 26-28, 1997
San Diego, California

FOR INFORMATION CALL:
1-800-343-ACCP or 847-498-1400