Cardiac and respiratory effects of continuous positive airway pressure and noninvasive ventilation in acute cardiac pulmonary edema

Karim Chadda, MD; Djillali Annane, MD, PhD; Nicholas Hart, MD; Philippe Gajdos, MD; Jean Claude Raphaël, MD; Frédéric Lofaso, MD, PhD

Objective: Continuous positive airway pressure (CPAP) is considered an effective nonpharmacologic method of treating patients with severe acute cardiogenic pulmonary edema. However, we hypothesized that bilevel noninvasive positive-pressure ventilation (NPPV), which combines both inspiratory pressure support and positive expiratory pressure, would unload the respiratory muscles and improve cardiac and hemodynamic function more effectively than CPAP.

Design: Randomized crossover study.

Setting: Critical care unit, Raymond Poincaré Hospital.

Patients: Six consecutive patients with acute cardiogenic pulmonary edema.

Interventions: Patients were sequentially treated with 5 cm H₂O CPAP, 10 cm H₂O CPAP, and NPPV in a random order.

Measurements and Main Results: Cardiac and hemodynamic function and indexes of respiratory mechanics were measured at each treatment sequence. NPPV reduced the esophageal pressure swing and esophageal pressure-time product compared with baseline (p < .05). There was no reduction in esophageal pressure swing or esophageal pressure-time product with CPAP. NPPV and 10 cm H₂O CPAP reduced the mean transmural right and left atrial filling pressures without a change in cardiac index.

Conclusions: This study demonstrates that NPPV was more effective at unloading the respiratory muscles than CPAP in acute cardiogenic pulmonary edema. In addition, NPPV and 10 cm H₂O CPAP produced a reduction in right and left ventricular preload, which suggests an improvement in cardiac performance. (Crit Care Med 2002; 30:2457–2461)

Key Words: heart failure; hemodynamics; noninvasive ventilation; acute cardiogenic pulmonary edema; continuous positive airway pressure; noninvasive positive-pressure ventilation.
METHODS

We performed a single-blinded, randomized crossover study in patients with ACPE admitted to Raymond Poincaré Hospital intensive care unit. The study protocol was approved by the comité consultatif de protection des personnes pour la recherche biomédicale de l’hôpital Henri-Mondor. Written informed consent was obtained from all patients before randomization.

Selection of Patients

The study was subdivided into three phases. The first phase (6–12 hrs) was for assessment and treatment of ACPE with standard medical therapy. The second phase was patient recruitment, and the third phase was the study protocol itself. Patients were only included in the study if they met the following criteria: 1) orthopnea, 2) an elevated jugular venous pressure, 3) a third heart sound on auscultation, 4) a pulmonary arterial occlusion pressure of 18 mm Hg or more, and 5) a cardiac index below 2.80 L/min/m². Patients were excluded from the study if they had evidence of sepsis, pneumonia, altered mental status, acute myocardial infarction, or arrhythmias. Patients underwent the CPAP and NPPV trial at least 6 hrs after the last dose of diuretic and at least 1 hr after discontinuation of vasodilator and inotropic drugs.

Study Protocol

Patients were intensively monitored during five separate study periods. Every period lasted 20 mins. The first and fifth periods were periods of spontaneous breathing. CPAP of 5 cm H₂O (CPAP5), CPAP of 10 cm H₂O (CPAP10), and NPPV were administered between the two spontaneous breathing periods in a random order. A resting steady state period of 10 mins was performed before the first period. All patients were studied at the bedside in the recumbent position. We used a full face mask (Respironics, Murrysville, PA) to measure minute ventilation and to apply CPAP and NPPV. During spontaneous breathing, airway pressure was the ambient pressure (17). Patients were administered oxygen to maintain oxygen saturation at >90%. CPAP was administered with a REM + device (Nellcor Puritan Bennett, Nancy, France). This device incorporates a servo-controlled system that enables minimization of pressure variation and work of breathing (17). NPPV was administered with an Onyx device (Nellcor Puritan Bennett, Nancy, France). Positive end-expiratory pressure was 5 cm H₂O and pressure support was 5 cm H₂O; inspiratory pressure was therefore 10 cm H₂O.

Respiratory Measurements

Breathing Pattern. Flow was measured using a Fleisch 2 pneumotachograph (Fleisch, Lausanne, Switzerland) connected to a differential pressure transducer (Validyne MP-45, ±5 cm H₂O, Northridge, CA). The flow signal was electronically integrated to calculate tidal volume and minute ventilation.

Pressure Measurements. Esophageal pressure was recorded after the insertion of a catheter-mounted transducer system (Gaeltec, Dunvegan, Isle of Skye, UK). Appropriate placement of the esophageal transducer was verified with an occlusion test (18). A pressure transducer (Validyne MP-45, ±14 cm H₂O) was connected between the pneumotachograph and the patient to measure the pressure at upper airways. All signals were sampled at 128 Hz and passed to a computer using an analogic-numeric system (MP100, Biopac System, Goleta, CA). Data were analyzed after the completion of the study.

Dynamic Compliance. Dynamic pulmonary compliance (CLdyn, in L/cm H₂O) was calculated as the ratio of tidal volume to the difference in transpulmonary pressure at the start and end of inspiration. Esophageal pressure values at instants of zero flow were considered as the start and end of the inspiratory cycle. Onset of the sharp negative deflection of the esophageal pressure curve was taken as the start of inspiratory effort.

Esophageal Pressure-Time Product. Average esophageal pressure-time product (in cm H₂O·sec·min⁻¹) was measured from 40 consecutive breaths as the area subtended by esophageal pressure and chest-wall static recoil pressure-time curve, taking account of dynamic intrinsic positive end-expiratory pressure over inspiratory time (19). The chest-wall static recoil pressure-time curve was extrapolated from normal subjects’ chest-wall static recoil pressure-volume curve, which corresponded to 4% of the predicted vital capacity per centimeter H₂O (20). Using the theoretical chest-wall compliance may lead to an error, but since this error would be constant throughout the study, the results would still be valid.

Cardiac and Hemodynamic Measurements

Heart Rate and Blood Pressure. Systolic and diastolic blood pressures (in mm Hg) and heart rate were measured using an automatic sphygmomanometer (Dinamap, Critikon, Tampa Bay, FL). Mean arterial pressure was calculated as follows: (systolic pulmonary arterial pressure + [2 × diastolic pulmonary arterial pressure])/3. All hemodynamic measurements were taken at end-expiration. Mean transmural right atrial pressure (mm Hg) and mean transmural pulmonary arterial occlusion pressure (mm Hg) were calculated (21). In brief, the mean esophageal pressure calculated over whole breath is subtracted from intrathoracic vascular measurements at each level of airway pressure (21).

Cardiac Output and Derived Hemodynamic Variables. Cardiac output (L/min) was calculated with an Edwards Model 9250 (Edwards Laboratories) as a mean of five measurements obtained by injecting 10 mL of dextrose solution randomly during the respiratory cycle, with exclusion of highest and lowest values. Arterial and mixed venous blood gas samples were obtained from the radial artery and pulmonary artery, respectively. These samples were immediately measured (Radiometer ABL 720, Tacussel, Copenhagen, Denmark). Derived hemodynamic and blood oxygen variables, stroke volume index, mixed venous oxygen saturation, oxygen delivery, and oxygen uptake were calculated using standard formulas (20).

Statistical Analysis

Data are expressed as mean ± sd. Differences between spontaneous breathing, CPAP5, CPAP10, and NPPV were tested using the nonparametric Friedman test. The 5% level was chosen as significant. When a significant difference was observed, pairwise comparisons were performed using the Bonferroni test.

RESULTS

Population Description

Over a 2-yr period, six patients with ACPE were recruited into the study. Three patients had hypertensive cardiomyopathy, two had idiopathic cardiomyopathy, and one had ischemic cardiomyopathy. All the patients had New York Heart Association class III or IV heart failure. The anthrrometric and treatment data of the patients are shown in Table 1.

Respiratory Function

Breathing Pattern and Gas Exchange. The effects of CPAP and NPPV on breathing pattern, gas exchange, respiratory load, and respiratory muscle unloading are shown in Table 2. Although there was an increase in tidal volume with CPAP5 (15%), CPAP10 (8%), and NPPV (27%) compared with spontaneous breathing, this only reached statistical significance with NPPV (p = .018). There was a mean increase in minute...
SD spontaneous breathing were not statisti-
cally different compared with spontaneous
breathing. In contrast, with CPAP5 or CPAP10, the decreases in esophageal pres-
sure-time product (19% and 10%, respectively) compared with spontaneous
breathing. In other words, with NPPV and CPAP10, the decreases in esophageal
pressure-time product (48% and 48%, respectively) and in mean transmural pulmo-
nary arterial occlusion pressure (48% and 48%, respectively) when compared with sponta-
naneous breathing. There were no significant changes in cardiac index, stroke
volume index, mixed venous oxygen saturation, oxygen delivery, and oxygen up-
take with CPAP5, CPAP10, or NPPV compared with spontaneous breathing.

**DISCUSSION**

The main finding of this small study is that short-term use of NPPV compared with
CPAP 10 in patients with ACPE causes a greater reduction in respiratory load but with similar improvements in cardiac performance.

**Critique of Method**

**Patient Recruitment.** One of the major limitations of this study was patient re-
cruitment. However, this was an invasive physiologic study that required each pa-
tient to have an esophageal pressure monitoring catheter and a pulmonary ar-
tery catheter inserted to measure changes in respiratory mechanics, cardiac
function, and hemodynamic function during five separate study periods. To our knowledge, the only other study that has attempted to study patients with
ACPE so extensively in a similar setting, albeit with CPAP alone, was by Lenique et al. (10), and even in this study, in which the protocol was simpli-
ed, the investigators only managed to recruit and study eight patients. It is also noteworthy that only one patient (patient 3) had an isch-
emic cardiomyopathy and a left ventricular ejection fraction of 0.47 ± 0.64; the re-
mainder of the patients had idiopathic dilated cardiomyopathy or hypertrophic
cardiomyopathy. The behavior under NPPV and CPAP of this patient was sim-
ilar to the others as we observed both a decrease of esophageal pressure-time
product under NPPV and a slight decrease of <10% of cardiac index under
NPPV and CPAP10 compared with basal conditions. Thus, we do not believe this
patient would change the main findings of this study.

**Comparison with Other Studies.** Lenique et al. (10) reported a reduction in
work of breathing in patients with ACPE associated with a decrease in CLdyn
and lung resistance using CPAP10. Al-

Table 1. Patients’ characteristics

<table>
<thead>
<tr>
<th>Pt. No.</th>
<th>Age/Sex</th>
<th>Height, m</th>
<th>Weight, kg</th>
<th>Cardiac Disease</th>
<th>LVEF, %</th>
<th>Class, NYHA</th>
<th>Medications</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>58/M</td>
<td>1.72</td>
<td>66</td>
<td>IDCM</td>
<td>43</td>
<td>IV</td>
<td>VD-DR-AD</td>
</tr>
<tr>
<td>2</td>
<td>53/M</td>
<td>1.81</td>
<td>80</td>
<td>IDCM</td>
<td>43</td>
<td>IV</td>
<td>VD-BB-NI-DR</td>
</tr>
<tr>
<td>3</td>
<td>62/M</td>
<td>1.72</td>
<td>47</td>
<td>ISCM</td>
<td>28</td>
<td>IV</td>
<td>VD-DR-CA-AD</td>
</tr>
<tr>
<td>4</td>
<td>60/F</td>
<td>1.60</td>
<td>80</td>
<td>HCM</td>
<td>40</td>
<td>III</td>
<td>VD-NI-DR</td>
</tr>
<tr>
<td>5</td>
<td>70/M</td>
<td>1.70</td>
<td>81</td>
<td>HCM</td>
<td>40</td>
<td>III</td>
<td>CA-NI-DR-AD</td>
</tr>
<tr>
<td>6</td>
<td>80/F</td>
<td>1.68</td>
<td>80</td>
<td>HCM</td>
<td>45</td>
<td>IV</td>
<td>AD-BB-CA</td>
</tr>
</tbody>
</table>

**Ventilatory Mode**

<table>
<thead>
<tr>
<th>V̇E, mL/min</th>
<th>RR, breaths/min</th>
<th>V̇E, L/min</th>
<th>V₅₁/V₅₂</th>
<th>ΔPes, cm H₂O</th>
<th>PTPes, cm H₂Osec/min</th>
</tr>
</thead>
<tbody>
<tr>
<td>SB</td>
<td>CPAP5</td>
<td>CPAP10</td>
<td>NPPV</td>
<td>RSB</td>
<td></td>
</tr>
<tr>
<td>518 ± 44</td>
<td>20 ± 2</td>
<td>10.5 ± 1.2</td>
<td>0.47 ± 0.05</td>
<td>0.191 ± 0.049</td>
<td>212 ± 65</td>
</tr>
<tr>
<td>597 ± 70</td>
<td>19 ± 2</td>
<td>11.3 ± 1.7</td>
<td>0.50 ± 0.07</td>
<td>0.127 ± 0.065</td>
<td>172 ± 77</td>
</tr>
<tr>
<td>562 ± 48</td>
<td>19 ± 3</td>
<td>10.7 ± 1.9</td>
<td>0.48 ± 0.05</td>
<td>0.152 ± 0.073</td>
<td>191 ± 55</td>
</tr>
<tr>
<td>656 ± 123²</td>
<td>18 ± 2</td>
<td>13.1 ± 1.8</td>
<td>0.54 ± 0.13</td>
<td>0.153 ± 0.047</td>
<td>146 ± 76²</td>
</tr>
<tr>
<td>530 ± 65</td>
<td>20 ± 2</td>
<td>10.1 ± 1.3</td>
<td>0.48 ± 0.05</td>
<td>0.118 ± 0.047</td>
<td>203 ± 68</td>
</tr>
<tr>
<td>p</td>
<td>.018</td>
<td>.340</td>
<td>.225</td>
<td>.475</td>
<td>.001</td>
</tr>
</tbody>
</table>

¹p < .05; Bonferroni test vs. SB and RSB.

**Table 2.** Respiratory and mechanics variables during spontaneous breathing (SB), 5 and 10 cm H₂O continuous positive airway pressure (CPAP5 and CPAP10), noninvasive positive pressure ventilation (NPPV), and return to spontaneous breathing (RSB)

**Cardiac and Hemodynamic Function**

There were no changes in heart rate and blood pressure during CPAP 5, CPAP10, and NPPV (Table 3). CPAP10 and NPPV produced significant decreases in both mean transmural right atrial pressure (61% and 57%, respectively) and in mean transmural pulmonary arterial occlusion pressure (48% and 48%, respectively) when compared with spontaneous breathing. There were no signifi-
cant changes in cardiac index, stroke volume index, mixed venous oxygen saturation, oxygen delivery, and oxygen uptake with CPAP5, CPAP10, or NPPV compared with spontaneous breathing.

**Lung Compliance and Respiratory Muscle Unloading.** Although there was a mean increase in CLdyn of 33 mL/cm H₂O and 34 mL/cm H₂O with NPPV and CPAP10 compared with baseline, these rises were not statistically significant. Esophageal pressure swing and esophageal pressure-time product decreased significantly with NPPV (37%, p = .02, and 31%, p = .001, respectively) compared with spontaneous breathing. In contrast, with CPAP5 or CPAP10, the decreases in esophageal pressure swing (19% and 17%, respectively) or esophageal pressure-time product (19% and 10%, respectively) compared with spontaneous breathing were not statistically significant (Table 2).
though in our study the respiratory muscles were unloaded using NPPV, as evidenced by the decrease in esophageal pressure swing and esophageal pressure-time product, we found no statistical increase in Cl dyn with NPPV or CPAP10, although there was a trend for the Cl dyn to increase. In fact, the mean rise in Cl dyn was greater with CPAP10 and NPPV to increase. In fact, the mean rise in Cl dyn was greater with CPAP10 and NPPV compared with the study by Lenique et al. (10), but probably due to the small patient numbers in the current study, these differences were not significant.

### Significance of the Findings

**Respiratory Effects.** In the present study, neither CPAP or NPPV improved the respiratory mechanics. However baseline values of elastic loading, Cl dyn, in our patients were higher than that in previous reported studies (10), and this discrepancy in the load could have contributed to the reduced improvements in work of breathing in our study during CPAP5 and CPAP10. The observed unloading of the respiratory muscles and reduction in esophageal pressure swing and esophageal pressure-time product is probably attributable to inspiratory assistance during NPPV; thus, we hypothesize that NPPV unloads the respiratory muscles and increases tidal volume immediately at the initiation of ventilation, before any significant alteration in respiratory mechanics. This is therefore in contrast to CPAP that unloads the respiratory muscles as a result of an improvement in pulmonary mechanics.

**Cardiac Effects.** The effects of positive intrathoracic pressure on cardiac output are variable and dependant on the ventricular filling pressures. In contrast to the normal heart, the cardiac output of the failing heart is predominantly dependent on afterload changes (9). Studies using CPAP in patients with stable chronic heart failure have shown that the greatest increase in cardiac output is found in those patients with higher filling pressures (e.g., pulmonary arterial occlusion pressure of >12 mm Hg) (22). Although we only included in this study patients who had a pulmonary arterial occlusion pressure of >18 mm Hg, to maximize the chances of observing an increase in cardiac output, we did not observe any increase in cardiac output during either the CPAP or NPPV mode. In fact, there was a tendency for cardiac output to decrease during CPAP10 and NPPV in comparison with baseline conditions (p = .08). This difference could be statistically significant with a greater number of patients included. However, the differences between NPPV or CPAP10 and basal conditions were <10%, which is low considering that a >12% change in cardiac output, using the thermodilution method, is required to be of clinical relevance (23).

**Clinical Implications of Findings.** Until recently, NPPV was not considered effective treatment for ACPE (24). However, after two recent clinical trials in patients with ACPE, interest in the use of NPPV in the management of patients with acute cardiac decompensation has increased (14, 15). Although most patients with acute heart failure respond to standard medical therapy without the need for ventilatory assistance, NPPV has been shown to be better than CPAP (15) and conventional therapy with oxygen (12). NPPV causes a greater improvement in respiratory rate (15), blood pressure (15), arterial blood gases (15), and time to recovery (14), with a reduction in intubation rate (14). The unloading of the respiratory muscles and increase in tidal volume reported in this study is supportive evidence for the clinical benefits demonstrated in previous studies (14, 15).

However, there are still concerns about managing patients with acute heart failure with NPPV. Although Mehta et al. (15) reported the beneficial effects of NPPV in ACPE patients, this trial had to be terminated prematurely because of the high rate of acute myocardial infarction in the NPPV group. Despite a trend toward more chest pain in the NPPV group, which could be due to inadequate randomization, this observation raises the possibility that biventricular pressure-support ventilation stresses the myocardium greater than CPAP. However, we found no differences between CPAP and NPPV in terms of the cardiac or hemodynamic effects. CPAP10 and NPPV produced similar reductions in right and left ventricular preload, as evidenced by the fall in right and left atrial transmural pressures, without any change in cardiac output, which has previously been suggested as an improvement in cardiac performance (10). In addition, in this study, none of the patients experienced any ischemic problems using either NPPV or CPAP. Therefore, the advantages and pitfalls of using NPPV in patients with ACPE need to be appreciated, but in this study, as with the study by Masip et al. (14) we

### Table 3

<table>
<thead>
<tr>
<th>Ventilatory Mode</th>
<th>SB</th>
<th>CPAP5</th>
<th>CPAP10</th>
<th>NPPV</th>
<th>RSB</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>HR, bpm</td>
<td>85 ± 12</td>
<td>82 ± 13</td>
<td>86 ± 13</td>
<td>83 ± 13</td>
<td>88 ± 13</td>
<td>.588</td>
</tr>
<tr>
<td>BP, mean, mm Hg</td>
<td>86 ± 0.5</td>
<td>94 ± 0.3</td>
<td>93 ± 0.3</td>
<td>91 ± 0.3</td>
<td>85 ± 0.3</td>
<td>.675</td>
</tr>
<tr>
<td>MRAPTM, mm Hg</td>
<td>13.0 ± 3.0</td>
<td>8.5 ± 2.4</td>
<td>5.7 ± 2.4</td>
<td>5.5 ± 1.7</td>
<td>14.0 ± 3.3</td>
<td>.048</td>
</tr>
<tr>
<td>PAO2, torr</td>
<td>29 ± 0.4</td>
<td>19 ± 0.3</td>
<td>15 ± 0.9</td>
<td>15 ± 0.9</td>
<td>27 ± 0.9</td>
<td>.019</td>
</tr>
<tr>
<td>CI, L/min/m²</td>
<td>2.3 ± 0.18</td>
<td>2.37 ± 0.18</td>
<td>2.19 ± 0.17</td>
<td>2.11 ± 0.17</td>
<td>2.22 ± 0.22</td>
<td>.080</td>
</tr>
<tr>
<td>SVI, mL/m²</td>
<td>29.5 ± 4.1</td>
<td>31.0 ± 4.3</td>
<td>32.4 ± 3.6</td>
<td>31.4 ± 4.2</td>
<td>27.4 ± 4.2</td>
<td>.655</td>
</tr>
<tr>
<td>Svo₂%</td>
<td>60 ± 0.05</td>
<td>60 ± 0.05</td>
<td>60 ± 0.05</td>
<td>61 ± 0.05</td>
<td>60 ± 0.05</td>
<td>.998</td>
</tr>
<tr>
<td>pH</td>
<td>7.39 ± 0.05</td>
<td>7.41 ± 0.04</td>
<td>7.41 ± 0.03</td>
<td>7.41 ± 0.04</td>
<td>NA</td>
<td>.778</td>
</tr>
<tr>
<td>Pao₂, torr</td>
<td>71 ± 0.5</td>
<td>78 ± 0.5</td>
<td>78 ± 0.5</td>
<td>92 ± 0.7</td>
<td>NA</td>
<td>.245</td>
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<tr>
<td>Paco₂, torr</td>
<td>41.2 ± 5.0</td>
<td>39.6 ± 3.9</td>
<td>38.8 ± 2.5</td>
<td>38.8 ± 4.3</td>
<td>NA</td>
<td>.997</td>
</tr>
<tr>
<td>Do₂, mL/min</td>
<td>348 ± 37</td>
<td>366 ± 25</td>
<td>342 ± 16</td>
<td>330 ± 19</td>
<td>NA</td>
<td>.325</td>
</tr>
<tr>
<td>Vco₂, mL/min</td>
<td>135 ± 13</td>
<td>130 ± 16</td>
<td>124 ± 14</td>
<td>129 ± 11</td>
<td>NA</td>
<td>.287</td>
</tr>
</tbody>
</table>

HR, heart rate; BP, blood pressure; MRAP TM, transmural mean right atrial pressure; PAO2 TM, transmural pulmonary arterial occlusion pressure; CI, cardiac index; SVI, stroke volume index; Svo₂, mixed venous oxygen saturation; Do₂, oxygen delivery; Vco₂, oxygen consumption.

*p < .05, Bonferroni test vs. SB and RSB.
Short-term use of noninvasive positive-pressure ventilation has similar cardiac and hemodynamic benefits as 10 cm H₂O of continuous positive airway pressure in patients with acute cardiogenic pulmonary edema.

CONCLUSION

Short-term use of NPPV has similar cardiac and hemodynamic benefits as CPAP in patients with ACPE. In addition, NPPV unloads the respiratory muscles, reduces respiratory effort, and increases tidal volume before any alterations in pulmonary mechanics. This is in contrast to CPAP, which requires the pulmonary mechanics to change before any benefits of respiratory muscle unloading are observed. The results of this study favor the use of NPPV in selected patients with ACPE, and clinical trials are now warranted to compare the clinical and physiologic effects of standard medical therapy with NPPV and CPAP.

ACKNOWLEDGMENTS

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REFERENCES

Short-term Noninvasive Pressure Support Ventilation Prevents ICU Admittance in Patients With Acute Cardiogenic Pulmonary Edema*

Matteo Giacomini, MD; Gaetano Iapichino, MD; Marco Cigada, MD; Aldo Minuto, MD; Rebecca Facchini, MD; Andrea Noto, MD; and Elena Assi, MD

Study objectives: Noninvasive ventilation, although effective as treatment for patients with acute cardiogenic pulmonary edema when prolonged for hours, is of limited use in the emergency department (ED). The aim of the study was to determine whether a short attempt at noninvasive pressure support ventilation avoids ICU admittance and to identify lack of response prediction variables.

Design: Prospective inception cohort study.

Setting: ED of a university hospital.

Patients: Fifty-eight consecutive patients with cardiogenic pulmonary edema who had been unresponsive to medical treatment and were admitted between January 1999 and December 2000.

Interventions: Pressure support ventilation was instituted through a full-face mask until the resolution of respiratory failure. A 15-min “weaning test” was performed to evaluate clinical stability. Responder patients were transferred to a medical ward. Nonresponding patients were intubated and were admitted to the ICU.

Main outcome measures: The included optimal length of intervention, the avoidance of ICU admittance, the incidence of myocardial infarction, and predictive lack of response criteria.

Results: Patients completed the trial (mean [± SD] duration, 96 ± 40 min). None of the responders (43 patients; 74%) was subsequently ventilated or was admitted to the ICU. Two new episodes of myocardial infarction were observed. Thirteen of 58 patients died. A mean arterial pressure of < 95 mm Hg (odds ratio [OR], 10.6; 95% confidence interval [CI], 1.8 to 60.8; p < 0.01) and COPD (OR, 9.4; 95% CI, 1.6 to 54.0; p < 0.05) at baseline predicted the lack of response to noninvasive ventilation.

Conclusions: A short attempt at noninvasive ventilation is effective in preventing invasive assistance. A 15-min weaning test can identify patients who will not need further invasive ventilatory support. COPD and hypotension at baseline are negative predictive criteria.

Key words: acute cardiogenic pulmonary edema; acute myocardial infarction; endotracheal intubation; length of ventilatory treatment; predictive failure criteria; noninvasive pressure support ventilation

Abbreviations: ACPE = acute cardiogenic pulmonary edema; AMI = acute myocardial infarction; ED = emergency department; NIPSV = noninvasive pressure support ventilation; OR = odds ratio; PEEP = positive end expiratory pressure; SpO₂ = peripheral saturation of oxygen.

Acute cardiogenic pulmonary edema (ACPE) may be a rapidly reversible illness once its pathogenic factors are controlled and the vicious circle of hypoxia/heart failure/hypoperfusion has been interrupted. The beneficial effects of positive intrathoracic pressure are well-established, and its use through a facemask has been addressed by several authors.1–4 Pressure support ventilation adds to the effects of positive end-expiratory pressure (PEEP) the possibility of decreasing respiratory workload and oxygen consumption, thus resulting in a faster restoration of vital signs.5–9 The result can be the avoidance of endotracheal intubation.

If the duration of treatment were short enough, noninvasive pressure support ventilation (NIPSV) could be applied in the emergency department (ED), thus avoiding admittance to the ICU. However, no indications are available as to how long NIPSV has to be continued before judging it suc-
cessful or not. Noninvasive respiratory assistance is usually applied for hours. As a result, ICU facilities are required and possibly unavoidable invasive ventilation can be delayed.

Of note, predictive criteria for lack of response to NIPSV for ACPE are lacking and higher incidences of acute myocardial infarction (AMI) and mortality were reported in patients who had been treated with NIPSV in contrast to those receiving continuous positive airway pressure or conventional treatment.

This uncontrolled prospective trial was performed in the ED on patients with pure ACPE who were unresponsive to full medical treatment. The primary objectives were as follows: (1) to determine the optimal duration of NIPSV (ie, whether it can be short enough to be performed in the ED, yet effective in avoiding intubation and ICU admittance); (2) to identify specific criteria that are predictive of lack of response; and (3) to evaluate the effect of NIPSV on patients with AMI and its role in the presence of AMI.

Secondary objectives were hospital length of stay and mortality.

**Materials and Methods**

The setting was the ED of a university hospital. This study was performed in accordance with the Declaration of Helsinki. Informed consent was given by the patients in the study or by their next of kin. All consecutive patients affected by ACPE who required respiratory assistance after the institution of conventional medical treatment (defined as therapy with morphine oxygen via face mask, diuretics, and vasoactive drugs) had proven to be ineffective were eligible for the study.

The inclusion criteria were as follows: (1) pulmonary edema confirmed by rales over both lungs and signs of pulmonary congestion on chest radiographs within the first hour after presentation to the ED; (2) a pulse oximetric saturation (SpO2) of < 95% despite oxygen administration at 10 L/min via a reservoir mask; and (3) severe respiratory distress with dyspnea and use of accessory muscles, severe cyanosis, oligosanuria, and signs of peripheral hypoperfusion.

The exclusion criteria were life-threatening conditions (eg, bradycardia or malignant tachyarrhythmias with severe hemodynamic impairment), end-stage renal or liver disease, severe neurologic impairment (ie, Glasgow coma scale, < 7), and concomitant pneumonia. Demographic and anamnestic data were collected. A gastric tube was placed to avoid stomach distension.

NIPSV was applied (Respicare SC ventilator; Dräger Medi- zintechnik GmbH; Lübeck, Germany) through a full-face mask. PEEP and pressure support were initially set at 5 and 10 cm H2O (over PEEP), respectively. This setting then was modified in the attempt to obtain a tidal volume between 5 and 7 mL/kg. The fraction of inspired oxygen ranged between 0.8 and 1.

Noninvasive BP, SpO2, heart rate, and respiratory rate were monitored continuously. Arterial blood gas levels and ECG were recorded at baseline (on a reservoir oxygen mask before the onset of NIPSV) and just before the termination of NIPSV.

NIPSV was considered to be effective if dyspnea disappeared and if respiratory and hemodynamic parameters improved together with peripheral perfusion (ie, skin temperature and diuresis). The reporting of a subjective impression of “getting better” by the patient was also mandatory.

In the first 10 months of the study, the decision to stop NIPSV treatment and to perform a weaning test was left to the clinical judgment of the intensivist in charge, once the NIPSV efficacy criteria were met. After an interim analysis, which was intended to further reduce the duration of treatment, we decided to perform a weaning test within 90 min of the initiation of NIPSV.

The weaning test was conducted as follows. NIPSV was discontinued, and the patient was allowed to breath spontaneously on a reservoir oxygen mask for 15 min. If the patient remained clinically stable (ie, SpO2 of > 95%, absence of dyspnea, and stable hemodynamic parameters), the patient was discharged to the ward (defined as the responder group). The wards were defined medical wards with cardiologic expertise in which at least some beds were equipped with ECG and SpO2 monitoring equipment. If the patient did not respond to weaning, we proceeded to invasive ventilation, and the patient was transferred to the ICU (defined as the failure group).

The need for invasive ventilation, new episodes of AMI, hospital length of stay, and mortality were analyzed. AMI was diagnosed when two of the following three criteria were met: chest pain; increase in creatine phosphokinase concentration; and ECG signs of myocardial necrosis.

**Statistical Analysis**

The data were reported as the mean ± SD and interquartile range. The Student t test was used for statistical comparison. A p value of < 0.05 was considered to be significant. A logistic regression model, built using a backward stepwise approach, was carried out to identify the independent variables at hospital admission that could predict failure (the dependent variable). Age, the presence of COPD on hospital admission, AMI, heart rate, and respiratory rate, mean arterial pressure of < 95 mm Hg, PaO2, pH, and PaCO2 were considered to be independent variables and were introduced into the model only if they were associated with the dependent variable in the bivariate analysis at a permissive significance level (ie, p < 0.1 [χ2 test]) or if the odds ratio (OR) was > 1.5 or < 0.67. Variables that did not meet at least one of these conditions were not included in the final logistic model.

**Results**

Between January 1999 and December 2000, 58 consecutive patients with ACPE were enrolled in the study. The underlying diseases were as follows: ischemic heart disease (34 patients); COPD (16 patients); hypertension (15 patients); diabetes (7 patients); chronic renal failure (8 patients); and patent ductus (1 patient). Seven patients (12%) had signs of AMI at the time of hospital admission. Baseline hemodynamic and respiratory parameters, the data for which were collected before the onset of NIPSV, are reported in Table 1. Four patients could not be treated with NIPSV because of mask intolerance or refusal, five patients progressively worsened despite receiving NIPSV, and six patients failed the weaning test. All of these 15 patients were invasively ventilated and transferred to the ICU. NIPSV was
applied with a mean pressure support level of 14 ± 3 cm H_2O^* (responder group, 14.1 ± 3.2 cm H_2O; failure group, 14.1 ± 4.1 cm H_2O) with a PEEP of 8 ± 2 cm H_2O^8–10 (responder group, 8.4 ± 1.8 cm H_2O; failure group, 8.4 ± 1.8 cm H_2O). NIPSV significantly improved hemodynamic and respiratory parameters in the 43 patients in the responder group who were discharged from the ED. A positive but nonsignificant trend was found also in the failure group (Table 2).

Invasive mechanical ventilation was avoided in 76% of patients in the responder group (16 of 21 patients) and 73% of the patients in the failure group (27 of 37 patients). Excluding the four patients in whom NIPSV could not be applied, the mean duration of respiratory support was 118 ± 57 min (range, 105 to 143 min) in the first study period (19 patients) and 77 ± 22 min (range, 60 to 90 min) in the second study period (35 patients). In the responder group, 6 patients had AMIs on hospital admission and 37 did not. Only one patient who was admitted to the hospital with a diagnosis of AMI was not successfully treated. The effects of NIPSV were similar in AMI and non-AMI patients.

Throughout the hospital stay, after referral to the ward, none of the responder patients received ventilation or were admitted to the ICU.

Logistic regression analysis in 54 patients identified two risk factors for lack of response to NIPSV. Patients with a mean BP of < 95 mm Hg at hospital admission had a 10-fold increased risk of failing the NIPSV trial (OR, 10.6; 95% confidence interval, 1.8 to 60.8; p < 0.01). The presence of COPD was also significantly associated with the need for invasive ventilation (OR, 9.4; 95% confidence interval, 1.6 to 54.0; p < 0.05). Twenty-two percent of the patients died (13 of 58 patients), 26.7% of those (4 of 15 patients) in the failure group and 20.9% of those (9 of 43 patients) in the responder group. Of the 13 patients who died, 3 had been admitted to the hospital with a diagnosis of AMI. Two of the patients who died were in the responder group (one died of ventricular fibrillation on the ward 20 h after undergoing NIPSV, and the second patient died on day 19), and one patient in the failure group died on day 9. Two patients developed AMIs during their hospital stay (1 patient in the failure group on day 4, and 1 of 43 patients in the responder group on day 5).

The latter patient died on the 25th day of the hospital stay. The mean hospital length of stay did not differ between the patients in the responder group and those in the failure group (mean length of hospital stay, 17 ± 12 days [range, 9.5 to 19.5 days] vs 19 ± 10 days [range, 9.5 to 28 days], respectively; p = 0.4).

### Discussion

The great majority of patients with ACPE are initially managed in the ED. When patients do not respond to conventional medical treatment, ventilator assistance is needed. We tested the hypothesis that a short NIPSV run in the ED may avoid the use of invasive ventilation and admittance to the ICU. In the present study, critically ill patients were selected

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**Table 1—Demographic Characteristics and Baseline Clinical Parameters (During Oxygen Therapy) in the 58 Enrolled Patients**

<table>
<thead>
<tr>
<th>Variables</th>
<th>Values</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age, yr</td>
<td>74.1 ± 13.0 (69–84)</td>
</tr>
<tr>
<td>Male gender, %</td>
<td>60.0</td>
</tr>
<tr>
<td>Respiratory rate, breaths/min</td>
<td>36.8 ± 4.2 (35–40)</td>
</tr>
<tr>
<td>Arterial blood pH</td>
<td>7.20 ± 0.11 (7.14–7.28)</td>
</tr>
<tr>
<td>PaO_2, mm Hg</td>
<td>65.0 ± 34.8 (48–70)</td>
</tr>
<tr>
<td>PaCO_2, mm Hg</td>
<td>63.7 ± 20.7 (47–76)</td>
</tr>
<tr>
<td>Spo_2, %</td>
<td>80.6 ± 13.7 (75–90)</td>
</tr>
<tr>
<td>Heart rate, beats/min</td>
<td>112.8 ± 24.4 (100–130)</td>
</tr>
<tr>
<td>Mean arterial BP, mm Hg</td>
<td>112.6 ± 20.6 (88–136)</td>
</tr>
</tbody>
</table>

*Values given as mean ± SD (interquartile range).

---

**Table 2—Effects of NIPSV on Hemodynamic and Respiratory Parameters**

<table>
<thead>
<tr>
<th>Variables</th>
<th>Responder Group (n = 43)</th>
<th>Failure Group (n = 11)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Baseline</td>
<td>End-NIPSV</td>
</tr>
<tr>
<td>Respiratory rate, breaths/min</td>
<td>36.2 ± 6.0 (35–40)</td>
<td>24.5 ± 5.7 (20–28)†</td>
</tr>
<tr>
<td>Arterial blood pH</td>
<td>7.21 ± 0.10 (7.16–7.28)</td>
<td>7.38 ± 0.10 (7.3–7.4)†</td>
</tr>
<tr>
<td>PaO_2, mm Hg</td>
<td>67.7 ± 30.1 (51–70)</td>
<td>114.4 ± 52.0 (82–119)†</td>
</tr>
<tr>
<td>PaCO_2, mm Hg</td>
<td>62.3 ± 20.0 (48–75)</td>
<td>43.7 ± 10.5 (36–47)†</td>
</tr>
<tr>
<td>Spo_2, %</td>
<td>82.0 ± 13.7 (70–91)</td>
<td>97.5 ± 2.0 (96–99)</td>
</tr>
<tr>
<td>Heart rate, beats/min</td>
<td>114.6 ± 24.1 (100–131)</td>
<td>92.3 ± 16.6 (80–104)†</td>
</tr>
<tr>
<td>Mean arterial BP, mm Hg</td>
<td>118.6 ± 27.2 (102–138)</td>
<td>95.1 ± 14.1 (83–106)†</td>
</tr>
</tbody>
</table>

*Values given as mean ± SD (interquartile range).

†p < 0.05 compared to baseline.

‡p < 0.05 compared to responder patients at baseline.
by their need for ventilatory support after undergoing ineffective conventional medical therapy for ACPE (eg, morphine, oxygen mask, diuretics, and nitrates) [Table 1]. Patients with severe concomitant illnesses were excluded. Pneumonia was an exclusion criterion because in patients with pneumonia NIPSV already had proven to be a less effective therapy,12,13,16 and the patients probably needed a longer period of assistance. We did not perform a randomized trial since NIPSV has already proven to be effective in the treatment of ACPE6,7,11,12,14–16 and because our primary end point was the time needed to treat respiratory failure and to avoid ICU admission.

The duration of treatment is a critical issue when treating patients with ACPE outside the ICU. Prolonged ventilatory assistance is impractical in an environment like the ED, which is frequently understaffed, has limited space, and has a high turnover of patients. Moreover, NIPSV may dangerously delay, in some patients, unavoidable tracheal intubation and invasive mechanical ventilation.7,10,17,18

Despite the fact that most authors have reported a significant improvement of clinical parameters after 15 to 60 min,6,11–16 NIPSV is usually administered for a considerable length of time, ranging from 2 to > 24 h in patients who already have been admitted to the ICU or are transferred there soon after the beginning of NIPSV.6,10–16 As described by other authors,6,11–16 invasive respiratory support is avoided in a large percentage of patients, but we have shown that adequate clinical stability can be obtained in a much shorter time. A 90-min NIPSV trial applied in the ED with patients who had ACPE resulted in a rapid assignment to the best treatment, medical or invasive support, without inappropriate delay and use of ED resources. The weaning test identified patients who, although their condition improved during NIPSV, did not reach a sufficient clinical stability to be assigned to pure medical treatment. Patients in the failure group were invasively treated and transferred to the ICU, while patients in the responder group were discharged in a short time from the ED to the ward. The improvement was persistent in time, and none of the responder patients needed further ventilatory assistance throughout their hospital stays.

We do not confirm the reported high incidence of AMI that has been associated with NIPSV.6 During their hospital stays, only two patients developed new episodes of AMI, days after undergoing NIPSV and too late to be attributed to it. Moreover, at variance with another report,11 six of seven patients with AMI at baseline were responders. In accordance with the results of other trials,12,14,16 NIPSV thus may be used with reasonable safety in patients with AMI. The overall mortality rate was in the range that has been reported by other authors (ie, 7 to 25%),6,10–12,14,16 even if many studies6,10,14 have included patients before medical treatment was defined to be ineffective, thus enrolling a less critical population.

Moreover, it is reasonable to affirm that deaths were related to the pathology itself rather than to the type of respiratory treatment. This finding is supported by the fact that the only death potentially related to treatment (which occurred during the first day in a responder patient who had AMI at baseline) was actually due to a sudden and unexpected malignant arrhythmia while the patient was on the ward. All the other deaths occurred days after NIPSV was performed in those who were not candidates for intensive treatment and probably were the result of concomitant terminal disease.

Finally, only two baseline conditions, mean arterial pressure < 95 mm Hg and a history of COPD, significantly predicted the failure of NIPSV. The latter condition could be at least partially explained by the sum of long-term and short-term increases in the work of breathing, resulting in an excessive respiratory workload that could not be rapidly managed by NIPSV alone. However, other concomitant factors, such as chronic tracheobronchitis, malnutrition, or obesity, cannot be excluded. The absence of arterial hypertension at baseline is probably consistent with a decreased left ventricular function19 with decreased cardiac reserves, selecting a group of patients with more severe conditions. The use of a NIPSV in ACPE patients with a mean arterial pressure of < 95 mm Hg at hospital admission cannot thus be encouraged. In COPD patients with ACPE, NIPSV may be effective, but the predictable need for prolonged respiratory assistance suggests caution in using it in the ED.

ACKNOWLEDGMENTS: We thank Luca Bigatello for discussing our results, and Bruno Simini for his precious advice and suggestions in preparing and editing the manuscript.

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17 Sottiaux TM. Noninvasive positive pressure ventilation in the emergency department. Chest 1999; 115:301–302
We assessed cardiogenic pulmonary edema (CPE) patient response to full mask pressure support noninvasive positive pressure ventilation (NPPV). Adult patients presenting to the emergency department (ED) in acute respiratory failure who clinically required endotracheal intubation (ETI) were studied. In addition to routine therapy consisting of oxygen, nitrates, and diuretics, patients were started on full mask NPPV using a Puritan Bennett 7200 ventilator delivering pressure support 10 cm H2O, PEEP 5 cm H2O, FiO2 100%. Pressure support was titrated to achieve tidal volumes of 5 to 7 mL/kg, and PEEP titrated to achieve oxygen saturation (SaO2) > 90%. Outcome measures included arterial blood gas (ABG), Borg dyspnea score, vital signs, and need for ETI. Twenty patients mean age 74.7 ± 14.3 years were entered on the study. Initial mean values on FiO2 100% by nonbreather mask: pH 7.17 ± .13, paCO2 65.5 ± 19.4 mmHg, paO2 73.3 ± 27.3 mm Hg, SaO2 89.7 ± 10.0%, Borg score 8.1 ± 1.4, and respiratory rate (RR) 38 ± 6.3. At 60 minutes of NPPV, improvement was statistically significant: pH 7.28 (difference .11; 95% CI .04-.19), paCO2 45 (difference 20.5; 95% CI 8-33), Borg score 4.1 (difference 4.0; 95% CI 3-5), and RR 28.2 (difference 9.8; 95% CI 5-14). NPPV duration ranged from 30 minutes to 36 hours (median 2 hours, 45 minutes). Eighteen patients (90%) improved allowing cessation of NPPV. Two patients with concomitant severe chronic obstructive pulmonary disease (COPD) required ETI. There were no complications of NPPV. NPPV using full face mask and pressure support provided by a conventional volume ventilator is an effective treatment for CPE and may help prevent ETI. (Am J Emerg Med 2001;19:179-181. Copyright © 2001 by W.B. Saunders Company)

Acute pulmonary edema is a life-threatening emergency that can require mechanical ventilation to treat. Pressure support ventilation is commonly accomplished by endotracheal intubation (ETI). Emergency intubation in acute respiratory failure is an invasive procedure with the potential for serious complications.

In contrast, NPPV provides ventilation without using an endotracheal tube. During the last 10 years, increasing attention has focused on using noninvasive ventilatory support to treat patients in acute respiratory failure (ARF). This management is as effective as conventional ventilation in improving gas exchange and avoids the complications of an invasive intubation with an endotracheal tube. Meduri et al reported 158 ARF inpatients treated with face mask noninvasive positive pressure ventilation (NPPV). In this study, causes of ARF included COPD (chronic obstructive pulmonary disease), status asthmaticus, acute upper airway obstruction, pneumonia and cardiogenic pulmonary edema. In this series, 5 of 9 cardiogenic pulmonary edema (CPE) patients in the intensive care unit avoided ETI because of NPPV. No emergency department (ED) patients were studied. Rasanen et al and Bersten et al described mask CPAP (continuous positive airway pressure) treatment of CPE. Rusterholtz et al successfully treated 8 intensive care unit CPE patients with NPPV. Sachetti et al and Pollack et al studied nasal BiPAP (bilevel positive airway pressure) support for acute respiratory distress (ARD) in the ED and found it effective treatment of acute pulmonary edema. Recently, the efficacy of CPAP and nasal BiPAP for acute pulmonary edema has been questioned. Mask CPAP is not well tolerated because patients must exhale against continuous pressure. In addition, mask CPAP does not provide inspiratory pressure support. Nasal BiPAP also has disadvantages compared to a conventional volume ventilator. FiO2 is not controllable. Exhaled tidal volumes cannot be monitored to assess minute ventilation.

Pressure support ventilation is a different mode of ventilation than either CPAP or nasal BiPAP. Pressure support is a ventilatory mode that augments spontaneous tidal volume. The pressure is terminated when the inspiratory flow rate drops below a preset value (eg, 25% of the initial inspiratory flow rate) and is off during exhalation. In contrast, CPAP provides positive pressure throughout both the inspiratory and expiratory cycles of breathing, and at higher levels (ie, greater than 5 cm H2O) may be extremely uncomfortable. Although nasal BiPAP provides alternating

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**Key Words:** NPPV, pressure support ventilation, BiPAP, CPAP, cardiogenic pulmonary edema.

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From the *Department of Emergency Medicine, Lutheran General Hospital, Park Ridge, IL; Section of Emergency Medicine, Department of Medicine, University of Chicago, Chicago, IL; †Respiratory Care Department, Lutheran General Hospital, Park Ridge, IL; ‡Pulmonary Medicine, Lutheran General Hospital, Park Ridge, IL; †Pulmonary Medicine, the Chicago Medical School, North Chicago, IL; §Emergency Medicine Residency Program, University of Chicago, Chicago, IL.

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pressures for inspiration and expiration, it does not allow accurate monitoring of expired tidal volumes and FiO₂. A volume ventilator pressure support of 10 cm H₂O and 5 cm H₂O end expiratory pressure is equivalent to nasal BiPAP using settings of 15 cm IPAP and 5 cm EPAP. Using a volume ventilator, pressure support can be increased if exhaled tidal volumes and minute ventilation are low. Also, the volume ventilator can control FiO₂ as well as sensitivity adjustments for leakage within the system.

Despite several advantages, there are few reported cases of pressure support NPPV treatment for acute cardiogenic pulmonary edema using full mask and pressure support provided by a conventional volume ventilator. No studies have reported use in ED patients. The purpose of this pilot study was to determine the feasibility of pressure support NPPV treatment of acute cardiogenic pulmonary edema in ED patients using a conventional volume ventilator and full face mask.

MATERIALS AND METHODS

All patients more than 18 years of age who presented with ARF caused by CPE and clinically appeared to require ETI were candidates for study inclusion. Exclusion criteria included pregnancy, apnea, and shock. All patients were immediately treated with routine therapy including FiO₂ 100% oxygen by nonrebreather mask, diuretics, and sublingual nitroglycerin tablets 0.4 mg every 5 to 10 minutes. The Respiratory Care Department was notified on the patient’s arrival to bring a Puritan Bennett 7200 ventilator (Carlsbad, CA) and other appropriate equipment for NPPV to the ED. NPPV was initiated according to a standard protocol (initial settings pressure support 10 cm H₂O, CPAP 5 cm H₂O, FiO₂ 100%). Pressure support was titrated to achieve tidal volumes of 5 to 7 mL/kg, and PEEP titrated to achieve oxygen saturation (SaO₂) > 90%.

Outcome measures included ETI, SaO₂, arterial blood gas (ABG), Borg dyspnea score (10 = severest respiratory distress), and vital signs. Patients who clinically deteriorated despite NPPV underwent ETI and ventilation as per standard ED procedures.

Data were collected by the Respiratory Care Department, physicians, and nurses in the ED on a standard data sheet. Data included patient name; age; arrival vital signs, oxygen saturation, and arterial blood gas; time of treatment with NPPV; duration of NPPV; repeat measurements of vital signs and oxygen saturation.

Statistical analysis was performed using the paired t-test and the Wilcoxon Matched-Pairs Signed-Ranks Test. Patient confidentiality was protected and individual patient data were never disclosed. The study was approved by the Institutional Review Board at Lutheran General Hospital. Patients able to give informed consent were asked to participate on the study. However, in most cases, family members provided consent. Consecutive patients who met criteria were asked to enroll on the study. There were no refusals to requests for study enrollment.

RESULTS

Twenty patients mean age 74.7 ± 14.3 years were entered on the study. Initial mean values on FiO₂ 100% by nonrebreather mask and response to treatment with NPPV are summarized in Table 1.

NPPV duration ranged from 30 minutes to 36 hours (median 2 hours, 45 minutes). Mean pressure support was 10 cm H₂O and mean PEEP was 5 cm H₂O. Eighteen patients (90%) improved allowing cessation of NPPV. Two patients with concomitant severe COPD required ETI. There were no complications of NPPV. No patients showed electrocardiographic (ECG) evidence of acute myocardial infarction.

DISCUSSION

ED patients in ARF caused by acute CPE can be successfully treated with full mask pressure support NPPV. This technique does not have the complications of invasive ETI. However, just like ETI, ventilatory support using a full face mask and volume ventilator allows titration of pressure support to achieve desired tidal volumes.

One reason for this study was our experience with patients not being able to tolerate CPAP or nasal BiPAP. Full mask NPPV may prove to be more comfortable than mask CPAP and nasal BiPAP in acute pulmonary edema patients although this needs to be studied further.

The 2 treatment failures in this study requiring ETI had concomitant severe COPD. Patient 1 was a 66-year-old woman with a history of severe COPD and congestive heart failure (CHF). She presented with a Borg score of 9 (almost maximal breathing exertion). Initial ABG on 100% oxygen showed pH 7.12 and paCO₂ 77. Vital signs were blood

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**TABLE 1. Response to NPPV**

<table>
<thead>
<tr>
<th></th>
<th>Initial</th>
<th>60 Minutes</th>
<th>Difference (95% CI)</th>
</tr>
</thead>
<tbody>
<tr>
<td>pH</td>
<td>7.17 ± .13</td>
<td>pH 7.28 ± .09</td>
<td>0.11 (0.04-.19)*</td>
</tr>
<tr>
<td>pacoCO₂</td>
<td>65.5 ± 19.4 mmHg</td>
<td>45.0 ± 16.1 mmHg</td>
<td>20.5 (8-33)†</td>
</tr>
<tr>
<td>Borg score</td>
<td>8.1 ± 1.4</td>
<td>4.1 ± 2.3</td>
<td>4.0 (3-5)‡</td>
</tr>
<tr>
<td>Respiratory rate</td>
<td>38 ± 6.5</td>
<td>28.2 ± 8.7</td>
<td>9.8 (4-14)§</td>
</tr>
<tr>
<td>Systolic blood pressure</td>
<td>172 ± 45</td>
<td>121 ± 24</td>
<td>51 (31-71)#</td>
</tr>
<tr>
<td>Diastolic blood pressure</td>
<td>97 ± 25</td>
<td>68 ± 11</td>
<td>29 (19-40)∥</td>
</tr>
<tr>
<td>Heart rate</td>
<td>117 ± 21</td>
<td>97 ± 26</td>
<td>20 (11-29)**</td>
</tr>
</tbody>
</table>

*P = .007 by paired t-test.
†P = .004 by paired t-test.
‡P = .002 by Wilcoxon signed ranks test.
§P = .004 by paired t-test.
∥P = .001 by paired t-test.
∥∥P = .001 by paired t-test.
pressure 120/70, heart rate 130 beats/min, and respiratory rate 32 breaths/min. Despite intensive medical treatment and full mask pressure support ventilation, she continued to deteriorate. After 1 hour of treatment, a clinical decision was made to endotracheally intubate her. ABG drawn before intubation (but not available at the time of intubation) showed pH 7.07, pCO2 77, and pO2 145. Patient 2 was a 77-year-old man with a history of COPD and CHF. He presented with a Borg score of 10 (maximal breathing exertion). Vital signs were blood pressure 172/89, heart rate 103 beats/min, and respiratory rate 28 breaths/min. Initial ABG showed pH 7.11, pCO2 81, pO2 42, SaO2 59% on FiO2 100%. After 40 minutes of intensive medical treatment including 30 minutes of full mask pressure support ventilation, the patient became unresponsive and bradycardic necessitating ETI.

The effectiveness of NPPV may differ significantly between emergency patients presenting with acute CPE and those presenting with COPD. Therefore, patient selection for NPPV may be critical to its success. In our series, the only 2 treatment failures were patients with concomitant severe COPD. These results differ from Meduri et al who intubated 4 of 9 (44%) cardiogenic pulmonary edema patients, but only 9 of 51 (18%) COPD with acute exacerbation patients. However, in Meduri's series, 11 of 27 (41%) COPD with pneumonia patients required intubation as did 6 of 12 (50%) COPD with congestive heart failure patients.3

In addition to oxygenation and ventilation, patients can be treated with medication nebulizers while on NPPV. Although theoretically possible, pneumothorax was not reported as a complication in the study by Meduri et al.

Our study is a pilot study limited by the number of patients enrolled. However, this series is the largest number of CPE patients reported using pressure support supplied by a conventional volume ventilator and full face mask, and the only such series of ED patients. A larger comparative trial would provide additional information about the efficacy of NPPV in ED pulmonary edema patients. Future research is needed to compare pressure support ventilation against CPAP and BiPAP for treatment of CPE in ED patients.

**SUMMARY**

Ventilatory support without ETI using full face mask and volume ventilator is an effective treatment for CPE. Pressure support NPPV in the ED is feasible and may help prevent ETI.

The authors are indebted to Nancy Cipparone and Mary Dahman of the Lutheran General Hospital Research Institute for statistical analysis and to Jane Hynes for manuscript preparation.

**REFERENCES**

Noninvasive pressure support ventilation (NIPSV) with face mask in patients with acute cardiogenic pulmonary edema (ACPE)


A1 Service de Réanimation Médicale et Centre Anti Poisons, Hôpitaux Universitaires de Strasbourg, Strasbourg, France

Abstract

Objectives: To assess (1) the short-term hemodynamic, respiratory and arterial blood gas effects of NIPSV in patients with ACPE who were likely to require endotracheal intubation, (2) the initial causes of failure and (3) the side effects and the difficulties of this technique. Design: Uncontrolled, prospective clinical study. Setting: Teaching hospital intensive care unit. Patients: 26 consecutive patients with severe ACPE. Interventions: Noninvasive ventilation via a face mask, using a pressure support mode (20.5 - 4.7 cmH2O), with an initial fractional inspired oxygen of 93.0 - 16 % and a positive end-expiratory pressure of 3.5 - 2.3 cmH2O. The need to intubate the patients within 48 h was considered as a criterion of failure of the procedure. Measurements and results: Clinical and biological parameters were measured at 15 and 30 minutes, 1 h and 2 h and at 1 h and 2 h, respectively. There were 5 (21 %) failures and 21 (79 %) successes. In both the success and the failure groups, clinical and blood gas parameters improved at the first measure. In the success group, within 15 min of the start of NIPSV, pulse oximetry saturation (SpO2) had increased from 84 - 12 to 96 - 4 % (p < 0.001), the respiratory rate (RR) had decreased from 36 - 5.3 to 22.4 - 4.9 breaths/min (p < 0.0001) and within 1 h the arterial oxygen tension and pH, respectively, had increased from 61 - 14 to 270 - 126 mmHg (p < 0.0001) and from 7.25 - 0.11 to 7.34 - 0.07 (p < 0.01) and the arterial carbon dioxide tension (PaCO2) had decreased from 54.2 - 15 to 43.4 - 6.4 mmHg (p < 0.01). There were no statistical differences between the success and failure groups for the initial clinical parameters: SpO2, RR, heart rate, mean arterial pressure. The only differences between the success and failure groups were in the PaCO2 (54.2 - 15 vs 32 - 2.1 mmHg, p < 0.001) and the creatine kinase (CPK) (176 - 149 vs 1282 - 2080 IU/l, p < 0.05); this difference in CPK activity was related to the number of patients who had an acute myocardial infarction (AMI) (4/5 in the failure group vs 2/21 in the success group, p < 0.05). All patients with AMI in the failure group died. Conclusion: Among patients in acute respiratory failure, those with severe ACPE could benefit from NIPSV if they are hypercapnic, but NIPSV should be avoided in those with AMI.

L'utilisation de la VNI dans l'asthme aigu est rapportée par Meduri et al. 1998 dans une étude ouverte, non contrôlée. Nous rapportons 2 observations d'asthme aigu grave dont l'état clinique, gazométrique, neurologique et hémodynamique se sont améliorés lors de l'utilisation de la VNI. Les résultats observés sauraient être exploités dans l'avenir.


Introduction : Cette étude prospective portant sur 956 dossiers retrouvés 43 % d'hommes, âge moyen 41 ± 18 ans ; 32 % des patients ont été hospitalisés au cours de l'année précédente. Un traitement de fond était pris dans 70 % des cas (corticostéroïdes inhalés 48 %, xérimédicaments 21 %, xérogénotiques de longue durée 19 %, théophylline 14 %, anticholinergiques 5 %). Avant la prise en charge aux urgences, une intervention médicale a été réalisée dans 42 % des cas et 7 % des patients ont bénéficié d'un transport médicalisé. Le site était de souverain bruit (≥ 24 h) dans 420 cas (45 %) et subaigu (< 24 h) dans 530 cas (55 %). Un facteur déclenchant a été retrouvé dans 78 % des cas. Le traitement préhospitalier a comporté des 82 agonistes dans 263 cas (30 %) et des corticostéroïdes systémiques dans 204 cas (21 %). Le DEP à l'arrivée était de 225 ± 107 L/min, la PaCO₂ de 38,6 ± 7,9 mmHg, témoignant de la relative sévérité des crises.

Le traitement initié aux urgences a été : nébulisation de 82 agonistes 89 %, d'anticholinergiques 38 %, de corticostéroïdes systémiques 58 %, théophylline 0,8 %.

Après une période d'observation d'environ deux heures, 512 patients (53 %) ont été hospitalisés dont 11 % en Réanimation. La durée moyenne d'hospitalisation a été de 6 ± 5 jours. Ces données soulignent la fréquence de l'asthme aigu de l'adulte aux urgences et devraient permettre de mieux adapter la prise en charge et les mesures de prévention.

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Ventilatory and hemodynamic effects of continuous positive airway pressure in left heart failure

F Lenique, M Habis, F Lofaso, JL Dubois-Rande, A Harf and L Brochard
Physiology Departement, INSERM U296, Henri-Mondor Hospital, Creteil, France.

Abstract

The ventilatory and hemodynamic effects of continuous positive airway pressure (CPAP) delivered via a face mask (at 0, 5, and 10 cm H2O, and after a return to 0 cm H2O) were studied in nine patients with acute left heart failure (pulmonary artery occlusion pressure [PAOP] > or = 18 mm Hg, and cardiac index [CI] < or = 2.8 L/min/m2). CPAP at 10 cm H2O induced an improvement in lung compliance (60 +/- 10 ml/cm H2O to 87 +/- 20 ml/cm H2O, p < 0.05) and in lung and airway resistance (5.7 +/- 1.0 cm H2O/L/s to 3.4 +/- 1.0 cm H2O/L/s, p < 0.05), a reduction in work of breathing (18 +/- 3 J/min to 12 +/- 2 J/min, p < 0.05), and in the pressure-time index of the respiratory muscles (279 +/- 22 cm H2O/s/min to 174 +/- 25 cm H2O/s/min, p < 0.05), without significant changes in breathing pattern. Despite a significant reduction in the negative swings in intrathoracic pressure (15.2 +/- 1.9 cm H2O to 10.8 +/- 1.8 cm H2O, p < 0.001), no significant change was observed in CI or stroke volume during CPAP. However, mean transmural filling pressures decreased significantly with CPAP, suggesting a better cardiac performance. Neither the level of stroke volume nor of PAOP, was predictive of changes in CI or in stroke volume. In patients with respiratory insufficiency caused by congestive heart failure (CHF), CPAP reduces respiratory muscle effort without altering cardiac output. The slight decrease in mean transmural left and right atrial pressures suggests an improvement in cardiac performance.
Effect of nasal continuous positive airway pressure on cardiac output and oxygen delivery in patients with congestive heart failure

DM Baratz, PR Westbrook, PK Shah and Z Mohsenifar
Department of Medicine, Cedars-Sinai Medical Center, University of California, Los Angeles.

Abstract

We studied the acute hemodynamic effects of increasing nasal continuous positive airway pressure (CPAP) in 13 patients with acute decompensation of congestive heart failure. Heart rate, respiratory rate, pulmonary capillary wedge pressure, right atrial pressure, systemic blood pressure, and thermodilution cardiac outputs were measured at baseline, during, and after application of nasal CPAP at increasing pressures of 5, 10, and 15 cm H2O. Cardiac index, stroke volume, and oxygen delivery were calculated. Based on a significant change in cardiac output greater than or equal to 400 ml, seven patients were classified as responders, whereas six patients were considered to be nonresponders. In responders, significant increases were noted in cardiac index (2.5 +/- 0.7 to 2.9 +/- 0.9 L/min/m2), stroke volume (49 +/- 15 to 57 +/- 16 ml), and oxygen delivery (10.3 +/- 5.1 to 12.3 +/- 6.0 ml/min/kg) without a change in pulmonary capillary wedge pressure. In contrast, the nonresponders showed no significant change in any of the hemodynamic parameters. Improvement in cardiac output could not be predicted by any of the baseline hemodynamic or clinical variables, nor was it related to random variations since all variables returned to baseline after cessation of CPAP. Increase in stroke volume without a change in pulmonary capillary wedge pressure (preload) suggests either improved inotropic function of the left ventricle or reduced left ventricular afterload with CPAP. Thus, CPAP may offer a new noninvasive adjunct to improving left ventricular function and augmenting cardiac performance in a subset of patients with congestive heart failure.
Cas clinique

Traitement d’un œdème pulmonaire cardiogénique par ventilation en pression positive continue par l’intermédiaire d’une canule Copa™

C. Avril-Chaize, J.P. Estèbe*, C. Écoffey

Service d’anesthésie-réanimation chirurgicale II, CHRU, hôtel-Dieu, 2, rue de l’Hôtel-Dieu, 35000 Rennes, France

RÉSUMÉ
Le Copa™ ou cuffed oropharyngeal airway a des indications se situant entre celles du masque facial et celles de la sonde endotrachéale. Nous l’avons utilisé pour la ventilation chez une patiente atteinte d’un œdème pulmonaire cardiogénique grave. Il a permis une suppléance ventilatoire efficace en pression positive continue, évitant ainsi l’intubation. Sa facilité de mise en place et l’absence de stimulation laryngée le rendent intéressant pour la prise en charge ventilatoire de patients en décompensation cardiaque aiguë. Il existe deux limites à cette technique : l’absence de protection des voies aériennes profondes contre le reflux du contenu gastrique, la contre-indiquée dans la ventilation prolongée, et l’impossibilité de déglutir, incompatible avec un état de conscience normal. Cette nouvelle canule pourrait constituer une première étape, avant l’intubation trachéale, pour débuter la réanimation des insuffisances respiratoires aiguës, dont l’étiologie laisse supposer une amélioration rapide. © 1999 Elsevier, Paris

canule oropharyngée / Copa™ / ventilation mécanique / œdème pulmonaire

ABSTRACT
Treatment of severe cardiogenic pulmonary oedema with continuous positive airway pressure via a cuffed oropharyngeal airway (Copa™).

We report the use of a cuffed oropharyngeal airway (Copa™), in a patient with an acute respiratory failure from a cardiogenic pulmonary oedema, for continuous positive pressure ventilation. Considering the ease of use and the lack of laryngeal stimulation, this device can be considered for mechanical ventilation in selected cases with acute cardiac failure. There are two contra-indications: prolonged mechanical ventilation, because of the lack of airway protection from gastro-oesophageal reflux, and normal consciousness, as the patient cannot swallow. This device can be considered when starting intensive therapy including mechanical ventilation in patients with acute respiratory failure of foreseen short duration. © 1999 Elsevier, Paris

cuffed oropharyngeal airway / Copa™ / mechanical ventilation / pulmonary oedema

Le Copa™ (cuffed oropharyngeal airway), mis au point par Greenberg en 1995 [1], se présente comme une canule de Guedel munie d’un ballonnet gonflable de grand volume (25 à 40 mL) qui assure l’étanchéité du système dans l’oropharynx. Elle est munie d’un raccord standard de 15 mm qui permet le raccordement à un circuit de ventilation mécanique. Pour les adultes, quatre tailles, de 8 à 11 cm de longueur, sont actuellement disponibles. Ce nouveau dispositif a récemment fait son apparition en anesthésie [2]. Ses indications sont comparables à celles du masque laryngé. Nous l’avons utilisé pour la première fois chez une patiente en décompensation cardiogène avec œdème aigu pulmonaire (OAP), nécessitant quelques heures de ventilation artificielle.

OBSERVATION
Une femme de 82 ans était hospitalisée pour un bilan d’anévrisme de l’aorte abdominale comprenant entre autres une scanographie et une artériographie.
Ses antécédents médicaux étaient une miliaire tuberculeuse et une myocardopathie ischémique compliquée, l'année précédente, d'un infarctus du myocarde antéro-septo-apical et, quelques mois plus tard, d'une décompensation cardiaque gauche consécutive à un arrêt brutal du traitement diurétique. Le bilan objectivait une insuffisance rénale modérée (clairance de la créatinine : 0.56 mL·min⁻¹) associée à une altération de la fonction systolique du ventricule gauche (fraction d’expansion du ventricule gauche déterminée en échocardiographie à 40 %), stable sous un traitement associant diurétique, inhibiteur de l’enzyme de conversion et dérivé nitré.

Au cours de l’artériographie est apparu un OAP sévère résistant au traitement par furosémide (60 puis 80 mg), saignée (150 mL), dobutamine (5 μg·kg⁻¹·min⁻¹) et dérivés nitrés (1 puis 2 mg·h⁻¹). Deux heures après l’artériographie, l’ECG ne montrait pas de modification du tracé initial et les enzymes cardiaques s’avéraient normales. La décompensation cardiaque par surcharge osmotique provoquée par l’injection de produit de contraste (bolus de 290 mL de produit de contraste hexa-iodé-hydrodissoluble, contenant 320 mg·mL⁻¹ d’iode) était évoquée. Des troubles de la conscience (somnolence, absence de réponse verbale et hypotension) en rapport avec une hypercapnie majeure (PaCO₂ à 95 mmHg pour une PaO₂ à 74 mmHg) imposaient une assistance ventilatoire. Un Copa™ (longueur 9 cm) a été mis en place. La facilité de la manœuvre a permis le maintien de la patiente en position assise. Après gonflage du ballonnet avec 30 mL d’air environ, la ventilation assistée en pression positive continue a pu s’effectuer sans contrainte (pression de crête inférieure à 25 cmH₂O) ni fuite, vérifiée par auscultation cervicale, ni insufflation gastrique vérifiée par auscultation épigastrique (ventilateur de type SERVO 900 D™ en mode de pression assistée à +20 cmH₂O, associé à une PEP à +10 cmH₂O et à une FiO₂ = 100 %). Initialement la SpO₂ était à 70 % et la PETCO₂, à 80 mmHg. La ventilation a permis une normalisation des constantes en 45 min et une baisse de la FiO₂ à 0.4. L’hypotension artérielle (passage de la pression systolo-diastolique de 150/70 à 70/30 mmHg) contemporaine de la chute de l’hypercapnie a été corrigée par la dobutamine (10 μg·kg⁻¹·min⁻¹). Le maintien de la PaO₂ et de la PaCO₂ s’est accompagnée de la récupération d’une conscience normale. Une heure plus tard, la malade retirait sa canule. L’amélioration s’est poursuivie vers la guérison quasi complète en trois heures (PaO₂ : 90 mmHg pour un apport d’oxygène nasal de 3 L·min⁻¹ et PaCO₂ : 45 mmHg). L’administration de dobutamine a été interrompue six heures plus tard. Il n’y a pas eu de récidive. L’échocardiographie, pratiquée deux jours plus tard, était superposable aux précédentes. La patiente a repris son traitement habituel et a quitté le service de cardiologie pour son domicile au cinquième jour.

**DISCUSSION**

Au cours des OAP graves, l’assistance ventilatoire est assurée habituellement par l’intermédiaire d’une intubation endotrachéale. Le masque facial a aussi été employé à la phase précocce de l’OAP [3]. L’originalité de notre observation tient à l’utilisation du Copa™ pour la prise en charge ventilatoire d’une patiente inconsciente en hypercapnie grave, sans recourir à l’intubation. Celle-ci impose une mise en décubitus dorsal et l’exposition des cordes vocales, qui est source de désaturation, de modifications hémodynamiques, voire de troubles du rythme [4]. Ces conséquences néfastes sont dues à la stimulation laryngée. Elles ne surviennent pas lors de l’insertion d’un masque laryngé [5]. La tolérance du Copa™, dans le cas rapporté, a semblé comparable à celle du masque laryngé. Son utilisation a permis la prise en charge efficace de la ventilation, tout en respectant la position assise, sans interruption des apports d’oxygène et sans stimulation laryngée. Ces avantages semblent particulièrement intéressants dans un contexte de défaillance cardiaque. L’utilisation du Copa™ est limitée par le risque d’inhalation du contenu gastrique qui, dès qu’il est suspecté, fera précéder l’intubation endotrachéale. Par ailleurs, l’impossibilité de déglutition rend son utilisation incompatible avec un état de conscience normal. En l’absence de trouble de conscience, l’aide ventilatoire par le intermédiaire d’un masque facial est indiquée. Initialement utilisée en réanimation pour la prise en charge des maladies neuromusculaires, la ventilation au masque facial a été étendue à des atteintes telles que les décompensations de bronchopathies chroniques obstructives, des OAP, des échecs d’extubation ou extubation trop précoces [6-8]. Il en ressort un bénéfice immédiat évident en termes de raccourcissement de la phase aiguë de la dé-
compensation et d’une diminution du nombre d’intubations, surtout en présence d’une hypercapnie initiale. Le masque facial requiert un niveau de conscience normal, car il nécessite l’acceptation et la coopération du patient.

Le masque laryngé et le Copa™ sont des solutions intermédiaires entre le masque facial et l’intubation endotrachéale. Ils deviennent particulièrement intéressants dans les détresses respiratoires aiguës avec troubles de la conscience rapidement réversibles, telles que l’OAP ou l’extubation prématurée [9]. Une autre indication en urgence pourrait être la suppléance à une intubation difficile. Ils pourraient permettre d’attendre dans de meilleures conditions ventilatoires, une aide technique ou humaine. Ces indications semblent parfaitement adaptées au Copa™, dont la technique de mise en place s’apparente à celle de la canule de Guedel [2], alors que la mise en place d’un masque laryngé requiert une certaine expérience.

**CONCLUSION**

En situation d’urgence, l’utilisation du Copa™ pour le traitement de certaines détresses respiratoires aiguës, en particulier l’OAP paraît intéressante. Il pourrait, dans certains cas qu’il reste encore à évaluer, constituer un nouveau palier thérapeutique avant l’intubation. Comme le masque laryngé, il séduit par sa facilité de mise en place et l’absence de traumatisme en comparaison de l’intubation endotrachéale. Le Copa™ pourrait faire partie du matériel d’urgence de réanimation de premier secours dans les services de médecine, notamment en l’absence d’une personne en mesure de pratiquer une intubation. Ils ne permettent ni aspirations bronchiques efficaces, ni protection contre l’inhalation gastrique. Leur emploi se limite, aux situations de détresse respiratoire aiguë dont l’évolution est supposée rapidement favorable ou en attente d’une intubation endotrachéale, lorsque celle-ci s’avère difficile.

**RÉFÉRENCES**

Effectiveness of CPAP by mask for pulmonary edema associated with hypercarbia.

Perel A, Williamson DC, Modell JH.

Abstract

We describe continuous positive airway pressure (CPAP) by mask to reduce hypercarbia in two patients who had pulmonary edema due to congestive heart failure. In such patients, beside reducing venous return and filling pressures, CPAP improves compliance and decreases the work of breathing, thereby improving effective ventilation. Hence, CPAP may be useful to combat not only hypoxemia but also hypercarbia that is associated with pulmonary edema.