Noninvasive Ventilation in Cardiogenic Pulmonary Edema
A Multicenter Randomized Trial

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Studies employing noninvasive pressure support ventilation in cardiogenic pulmonary edema have been performed in the intensive care unit when overt respiratory failure is already present and in small groups of patients. In this multicenter study, performed in emergency departments, 130 patients with acute respiratory failure were randomized to receive medical therapy plus O2 (65 patients) or noninvasive pressure support ventilation (65 patients). The primary end point was the need for intubation; secondary end points were in-hospital mortality and changes in some physiological variables. Noninvasive pressure support ventilation improved PaO2/FIO2, respi-rin-hospital mortality and changes in some physiological variables. Noninvasive pressure support ventilation improved PaO2/FIO2, respiratory rate, and dyspnea significantly faster. Intubation rate, hospital mortality, and duration of hospital stay were similar in the two groups. In the subgroup of hypercapnic patients noninvasive pressure support ventilation improved PaCO2 significantly faster and reduced the intubation rate compared with medical therapy (2 of 33 versus 9 of 31; p = 0.015). Adverse events, including myocardial infarction, were evenly distributed in the two groups. We conclude that during acute respiratory failure due to cardiogenic pulmonary edema the early use of noninvasive pressure support ventilation accelerates the improvement in PaO2/FIO2, PaCO2, dyspnea, and respiratory rate, but does not affect the overall clinical outcome. Noninvasive pressure support ventilation does, however, reduce the intubation rate in the subgroup of hypercapnic patients.

Keywords: acute respiratory failure; cardiogenic pulmonary edema; chronic obstructive pulmonary disease; noninvasive pressure support ventilation

The rationale for using continuous positive airway pressure (CPAP) in acute pulmonary edema is based on the fact that it may limit the decrease in functional residual capacity, improve respiratory mechanics and oxygenation, and decrease left ventricular afterload (1, 2). The best therapy for treating an episode of acute respiratory failure due to cardiogenic pulmonary edema is, however, controversial. A systematic review on the effect of CPAP on mortality and need for intubation of patients with cardiogenic pulmonary edema (3) concluded that experimental evidence exists to support its use in these patients, although the potential for harm has not been excluded; the widespread use of this ventilatory technique is, however, still not recommended by major clinical guidelines (4–6). Indeed, all randomized controlled trials using CPAP (3) excluded a priori the patients with preexisting hypercapnic chronic obstructive pulmonary disease (COPD), whereas one study included patients with a PaCO2 greater than 45 mm Hg, but without chronic airflow obstruction (7).

A physiologic study demonstrated that noninvasive pressure support ventilation (NPSV) was more effective at unloading the respiratory muscles than CPAP alone in patients with acute cardiogenic pulmonary edema (8).

In patients affected by COPD and hypercapnia recovering from an acute exacerbation of their disease, the addition of an inspiratory aid (NPSV) to CPAP has been shown to further reduce inspiratory muscle effort (9), so that the application of NPSV may be particularly useful in patients with cardiogenic pulmonary edema and signs of pump failure (i.e., hypercapnia).

In one uncontrolled study using NPSV it was noted that patients who responded to NPSV had a higher baseline carbon dioxide pressure than those who did not respond, suggesting that this strategy is of potential benefit only in patients affected by chronic pulmonary diseases or by disorders in which respiratory muscles are likely to be fatigued (10). In a similar population not balanced for subgroups according to the value of PaCO2, Masip and coworkers (11) reported that NPSV was superior to conventional oxygen therapy in reducing the intubation rate and more rapidly improving oxygenation. As a matter of fact, four of six patients (66%) requiring intubation in the conventional therapy group were hypercapnic, whereas no hypercapnic patients in the NPSV-treated group required intubation. Unfortunately, the small sample size did not allow a subgroup analysis of the impact of the different degrees of hypercapnia (PaCO2 greater than or less than 45 mm Hg) on the main outcomes. Indeed, most of the previous investigations, employing either CPAP or NPSV (7, 12–14), were performed in single, specialized centers (usually in an intensive care unit [ICU]), whereas first-line interventions are often carried out in emergency departments (15). The use of noninvasive ventilation directly in this environment could theoretically allow earlier use of this ventilatory technique and at the same time widen its use, because a consistent portion of patients are already intubated when they are admitted to the ICU. We designed a large, multicenter, randomized, prospective study in the setting of emergency rooms, comparing NPSV with conventional oxygen therapy in the treatment of acute cardiogenic pulmonary edema. The aim was to assess the feasibility of NPSV outside the ICU and to detect any differences in mortality, intubation rate, and some physiological variables such as dyspnea and respiratory rate. We also analyzed separately the subgroups of patients with and without hypercapnia, because this latter group is more likely to receive a greater benefit from the application of NPSV (11).

Some of the results of these studies have been previously reported in the form of an abstract (16).
METHODS

Patients
One hundred and thirty consecutive patients with acute cardiogenic pulmonary edema were prospectively recruited in five emergency departments. The study protocol was approved by the local research ethics committees, and oral consent was obtained from the patient or next-of-kin. inclusion criteria were the following: severe acute respiratory failure (P\(_{aCO2}\)/F\(_{iO2}\) less than 250), breathing oxygen at less than 10 L/minute for at least 15 minutes (time needed to stabilize the patients and to make a diagnosis), dyspnea of sudden onset with respiratory rate exceeding 30 breaths/minute, and typical physical signs of pulmonary edema. Exclusion criteria were as follows: (see online supplement for details): immediate need for endotracheal intubation, severe sensorial impairment (Kelly score greater than 3) (17), shock, ventricular arrhythmias, life-threatening hypoxia (\(Sp_{O2}\) [oxygen saturation as indicated by pulse oximetry] less than 80% with oxygen), acute myocardial infarction necessitating thrombolysis, severe chronic renal failure, and pneumothorax. Echocardiography was performed in 86 patients once the clinical condition allowed. Patients were randomly assigned to receive standard medical treatment plus O\(_2\) or standard treatment plus NPSV though a full face mask.

Standard Treatment
The patients had continuous \(Sp_{O2}\) and electrocardiographic monitoring. Oxygen therapy was delivered through a face mask with an inspired oxygen fraction aimed to maintain an \(Sp_{O2}\) greater than 90%. Medical treatment besides O\(_2\) therapy was also standardized.

NPSV
A portable ventilator, furnished with an oxygen analyzer and specifically designed for noninvasive ventilation (PV 102; Breas Medical, M"olndycke, Sweden), was connected to a full face mask (see online supplement for details). A common standardized protocol was used. The positive end-expiratory pressure was initially set at 5 cm H\(_2\)O and could be increased by 1 cm H\(_2\)O until a brisk increase in \(Sp_{O2}\) was observed, whereas the inspiratory pressure support was initially set at 10 cm H\(_2\)O and then increased in increments of 2 cm H\(_2\)O to the maximum tolerated.

Primary Outcomes
The primary end point was the need for endotracheal intubation according to standardized criteria defined in the online supplement.

Secondary Outcomes
Arterial blood gases, respiratory rate, systolic and diastolic blood pressure, heart rate, and dyspnea were recorded at fixed intervals. The duration of hospital stay was also recorded. Cardiac enzymes (creatine phosphokinase and its MB isoenzyme and troponins) were analyzed in all patients at study entry, and 4 and 10 hours after; additional analyses were performed in patients with myocardial infarction (18).

Statistical Analysis
The scheduled sample size of 130 patients would allow us to detect, at \(p = 0.05\), a difference between a postulated 35% rate of intubation in the conventionally treated group (2), and 10% in the NPSV group (5–7), with a power of 90%. The randomization was also balanced to distribute hypercapnic (P\(_{aCO2}\) \(\approx\) 45 mm Hg) patients and nonhypercapnic (P\(_{aCO2}\) \(<\) 45 mm Hg) patients evenly within each treatment group. Differences in baseline characteristics between standard treatment and NPSV groups (whole groups and subgroups according to a P\(_{aCO2}\) threshold of 45 mm Hg) were tested by means of an unpaired t-test and \(\chi^2\) test for continuous and categorical variables, respectively. Tables (2 \(\times\) 2) with expected counts less than five were analyzed by Fisher exact test.

Repeated measures two-ways analysis of variance was used to evaluate trends over time. Kaplan–Meier curves were generated for time data and compared by log-rank and Wilcoxon tests.

A logistic regression analysis was performed using intubation (yes/no) with input being the P\(_{aCO2}\) threshold greater than or less than 45 mm Hg, to verify the hypothesis that hypercapnia was a determinant of intubation.

All tests and \(p\) values are two tailed and analyses were performed on an intention-to-treat basis, using the SAS package (19). Results are given as means (plus the SD or SE, as specified in the figure legends).

RESULTS

For details see tables in the online supplement.

Patient Characteristics
Sixty-five patients were randomly assigned to standard treatment and 65 to NPSV (Figure 1) (see Table E1 in the online supplement for the distribution by center). The two groups had similar characteristics on admission (see Table E2 in the online supplement). Patients with a P\(_{aCO2}\) greater than or less than 45 mm Hg were equally distributed between the two treatment groups (Table 1). Preexisting cardiac or other disease, New York Heart Association class, possible precipitating causes of cardiogenic pulmonary edema, and echocardiographic findings were also similar in the two subgroups (see Table E3 in the online supplement).

Hyperthermia (i.e., body temperature \(>37.0^\circ\text{C}\)) was present in a consistent subgroup of patients despite their not showing any signs of pulmonary infection. Ten patients had a urinary tract infection, 12 had a suspected viral infection not related to the respiratory system (i.e., enteritis, sinusitis), 4 had a positive sputum culture (without signs of exacerbation), 2 had purulent skin infections, 1 had a dental abscess, whereas in the remaining patients no focus of infection was found. After the initial adjustments, the ventilator settings were set at 14.5 \(\pm\) 21.1 cm H\(_2\)O for the inspiratory support and at 6.1 \(\pm\) 3.2 for positive end-expiratory pressure. These settings were kept constant throughout the study, except in five patients who needed a small reduction of 2 cm H\(_2\)O in both inspiratory and expiratory levels.

Doses and frequencies of medical therapy are shown in Table E4 (see the online supplement). No significant differences in medical therapy were observed in the two groups of patients.

Primary Outcome and Hospital Mortality
Table 2 shows that overall there were no significant differences between the two treatment groups in the need for endotracheal intubation or hospital mortality, but when the statistical analysis was performed by dividing each treatment group into subsets of hypercapnic and nonhypercapnic patients, the percentage of patients needing intubation was significantly lower in those with a \(P_{aCO2}\) greater than 45 mm Hg.

Figure 1. Trial profile. NPSV = noninvasive pressure support ventilation.
The logistic regression analysis, based on the need (or the lack of need) for intubation and the level of $P_{aCO_2}$ (less than or greater than 45 mm Hg) did not, however, show any statistically significant correlation.

The mean duration of NPSV was 11.4 ± 3.6 hours. The reasons for and timing of intubation are shown in Table 3.

**Secondary Outcomes**

After 30 minutes of treatment patients receiving NPSV had a significantly higher $P_{aCO_2}/FiO_2$ ratio and this was still the case after 3 hours (Figure 2). Figure 3 illustrates the changes in $P_{aCO_2}$ recorded in the subset of hypercapnic patients in the two treatment groups; a significant decrease from baseline was observed in the NPSV group in the first hour of treatment. Most of the intubations in the subset of patients treated with medical therapy occurred in the first 3 hours (seven of nine patients).

In comparison with baseline values, respiratory rate, dyspnea score, blood pressure, and heart rate showed significant improvement earlier in the NPSV group than in the control group (Table 4).

**Other Clinical Outcomes**

As illustrated in Table E5 (see in the online supplement), there were no differences between the two groups in total hospital stay, occurrence of a “new” acute myocardial infarction, or infectious and noninfectious complications. Skin lesions due to the presence of the mask were assessed according to Gregoretti and coworkers (20): area of redness was recorded in 14 patients, initial ulcer without involvement of the muscle and/or bone in 9 patients, and area of necrosis in 4 patients. One patient complained of claustrophobia and three tolerated ventilation poorly during the night hours.

**DISCUSSION**

The present multicenter randomized study shows that early use of NPSV in emergency departments to treat severe cardiogenic pulmonary edema is feasible and effective in providing a more rapid improvement in oxygenation and dyspnea compared with standard medical therapy alone. NPSV in this context did not decrease the overall endotracheal intubation except in the subgroup of patients with baseline hypercapnia. Mortality and adverse events were equally distributed in the two treatment groups.

Our study of NPSV versus standard medical therapy in patients with acute pulmonary edema is the first to balance the enrollment of hypercapnic and normocapnic patients, in each treatment group, so that a subgroup statistical analysis was feasible. The study was also designed to avoid the occurrence of some confounding variables. For example, echocardiography was performed in our study in more than half of the patients and the two groups were extremely well balanced with respect to cardiac dysfunction, so that we are confident that this potential bias in the recruitment of patients was avoided. The same can be stated for the causes of the acute pulmonary edema and baseline characteristics. Some of the previous studies (10, 12) did not record the $P_{aCO_2}/FiO_2$ ratio, but only the $SpO_2$, which clearly depends on the fraction of oxygen delivered. For this reason enrollment criteria were not based on the ratio, which remains the major score of severity in these patients.

The overall intubation rate in our NPSV group was relatively higher than that in the study by Masip and coworkers (11), and this was particularly true for the nonhypercapnic patients, in whom the intubation rate was even higher than in the medically treated group. However, as illustrated in Table 3, only a small number of nonhypercapnic patients needed intubation for respiratory reasons (i.e., refractory hypoxia), because most of them were promptly intubated for cardiovascular problems. It is possible that another potential reason for the lack of greater benefit from NPSV may also be related to the limited experience that from NPSV may also be related to the limited experience that most of the centers participating in the study had in the administration of the technique, but this should also be true for the hypercapnic subgroup in which the intubation rate, in contrast, was fairly low.
The best therapy for treating an episode of acute respiratory failure due to cardiogenic pulmonary edema is still a controversial matter. For example, the use of CPAP is not yet judged standard in the Guidelines of the American Heart Association (4), the European Society of Cardiology (5), the International Liaison Committee on Resuscitation (6), and in most textbooks of medicine. Furthermore, definitive data are not available concerning either the use of NPSV versus medical therapy (two small studies) (11, 21) and NPSV versus CPAP (one small study) (22), so that larger randomized and controlled studies are needed.

NPSV has been the subject of some criticism, and its widespread use has not been recommended, so that the present study was designed to assess this controversial issue. We found that NPSV in hypoxemic patients is not superior to medical treatment in avoiding intubation, although it may produce faster improvement of some physiological variables. A rapid improvement in PaO₂/FiO₂ ratio was demonstrated in several other studies, using CPAP and NPSV, and indeed a decreased need for intubation was observed in these latter investigations (7, 11, 13, 14). Unfortunately, the design of the studies did not allow the authors to discriminate whether a specific subset of patients was responsible for the overall outcome.

In fact, different effects of NPSV in hypoxemic and hypercapnic acute respiratory failure have already been shown in a randomized, controlled study enrolling patients affected by pathologies other than cardiogenic pulmonary edema (23), whereas a trial in patients with cardiogenic pulmonary edema was underpowered to detect any possible difference (11).

Our study design allowed us to perform a subgroup analysis to eventually detect a possible difference between the outcomes of the patients with a PaCO₂ greater than 45 mm Hg and those with a PaCO₂ less than 45 mm Hg, because a power analysis of the sample size could not be determined a priori, simply because no data in the literature allowed us to build it. Our randomization was, however, balanced to obtain similar numbers of patients with and without hypercapnia in the two treatment groups, so that a subgroup analysis was performed. Unfortunately, despite a significantly different pattern of intubation being found between the two subgroups of patients, the logistic regression analysis did not confirm that a PaCO₂ level greater than 45 mm Hg was, per se, a determinant of intubation. This is likely to be

### Table 3. Reasons for and timing of intubation

<table>
<thead>
<tr>
<th>Reason for Intubation</th>
<th>Standard Treatment</th>
<th>NPSV</th>
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</thead>
<tbody>
<tr>
<td></td>
<td>PaCO₂ &lt; 45 mm Hg</td>
<td>PaCO₂ &lt; 45 mm Hg</td>
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<tr>
<td></td>
<td>No. of patients</td>
<td>No. of patients</td>
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<tr>
<td></td>
<td>Time from Admission (h)</td>
<td>Time from Admission (h)</td>
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<tr>
<td>MI necessitating thrombolysis</td>
<td>1</td>
<td>2</td>
</tr>
<tr>
<td>Hemodynamic instability Stroke</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>Cardiac arrest</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>Refractory seizure</td>
<td>1</td>
<td>5</td>
</tr>
<tr>
<td>GI bleeding</td>
<td>3</td>
<td>0.5</td>
</tr>
<tr>
<td>Refractory hypoxia</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>PaCO₂ &gt; 5 mm Hg from baseline after 1 h</td>
<td>2</td>
<td>1</td>
</tr>
<tr>
<td>Intolerance to NPSV</td>
<td>2</td>
<td>0.5</td>
</tr>
</tbody>
</table>

**Definition of abbreviations:** GI = gastrointestinal; MI = myocardial infarction; NPSV = noninvasive pressure support ventilation.
due to the relatively small sample size of the two subgroups of patients, so that further larger, stratified, randomized controlled trials are needed to eventually confirm the hypothesis that the hypercapnic patients are more likely to benefit from the application of NPSV.

Some studies have reported a high incidence of myocardial infarction when using NPSV. Mehta and coworkers (22) had to stop their trial because a high proportion of patients with myocardial infarction was detected in the patients randomized to NPSV. The authors suggested that the prolonged increase in intrathoracic pressure during inspiration may explain their results. It is of interest to note that they delivered NPSV using a spontaneous timed mode with a ventilator that at the time of the study was not equipped with the now available sophisticated expiratory triggering system, so that air leaks, when present, may have unduly prolonged the inspiratory time (phenomenon described as a failure to cycle off). This problem has now been solved by the new-generation ventilators. Furthermore, most of the patients (10 of 14) reported chest pain at admission, so that it is likely that acute ischemia preceded rather than followed the application of noninvasive ventilation. A more recent study by Sharon and coworkers (21) also described a higher rate of myocardial infarction using NPSV, but the low inspiratory and expiratory pressures used suggest that in this group of patients the ventilatory assistance may have been inadequate. In the present study we found the same incidence of myocardial infarction as that reported by Masip and coworkers (11) and by Takeda and coworkers (24), who described a satisfactory outcome in a group of patients with acute pulmonary edema secondary to myocardial infarction.

Overall, the number of adverse events occurring in our patients during their hospital stay was similar in the two groups. The most common adverse events during NPSV were skin lesions; the rate of these was comparable to that in some other published reports, but higher than in some others. This may be a reflection of the relative inexperience of the staff and could have contributed to the lack of tolerance in a small subset of patients.

In conclusion, we have shown that, compared with standard medical therapy, early use of NPSV in emergency departments for treatment of acute respiratory failure due to cardiogenic pulmonary edema produces faster gas exchange, and dyspnea score and respiratory rate improvements, but does not affect the overall clinical outcome. The subgroup analysis showed, however, that the need to intubate hypercapnic patients may be reduced by the use of NPSV. Considering that CPAP has been shown to reduce the intubation rate, but not mortality, and to improve physiological variables more rapidly compared with standard medical therapy (3), we can reasonably say that both NPSV and CPAP may be used in the treatment of cardiogenic pulmonary edema. Larger multicenter randomized studies are needed to compare the efficacy of CPAP versus NPSV, especially regarding the rate of intubation, so that we may determine which modality should then be tested versus the medical therapy (i.e., actually the “gold standard” for the major clinical guidelines) to assess whether mortality may be improved.

**Conflict of Interest Statement:** S.N. has no declared conflict of interest; G.C. has no declared conflict of interest; N.D. has no declared conflict of interest; A.B. has declared conflict of interest; M.M. has no declared conflict of interest; F.G. has no declared conflict of interest; N.D. has no declared conflict of interest; A.B. has declared conflict of interest.

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**References**


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**TABLE 4. PHYSIOLOGIC MEASUREMENT DURING THE FIRST 24 HOURS OF STUDY**

<table>
<thead>
<tr>
<th></th>
<th>Baseline</th>
<th>30 min</th>
<th>1 h</th>
<th>3 h</th>
<th>6 h</th>
<th>12 h</th>
<th>24 h</th>
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<tbody>
<tr>
<td>pH</td>
<td></td>
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<tr>
<td>ST</td>
<td>7.26 ± 0.09</td>
<td>7.28 ± 0.10</td>
<td>7.31 ± 0.09</td>
<td>7.36 ± 0.07</td>
<td>7.39 ± 0.06</td>
<td>7.41 ± 0.04</td>
<td>7.42 ± 0.03</td>
</tr>
<tr>
<td>NPSV</td>
<td>7.25 ± 0.11</td>
<td>7.28 ± 0.10</td>
<td>7.32 ± 0.07</td>
<td>7.36 ± 0.07</td>
<td>7.40 ± 0.05</td>
<td>7.42 ± 0.04</td>
<td>7.42 ± 0.03</td>
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<tr>
<td><strong>Respiratory rate</strong></td>
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<tr>
<td>ST</td>
<td>38.1 ± 6.9</td>
<td>36.4 ± 9.6</td>
<td>32.3 ± 7.1</td>
<td>29.8 ± 8.2</td>
<td>26.1 ± 8.6</td>
<td>24.0 ± 11.1</td>
<td>18.9 ± 4.2</td>
</tr>
<tr>
<td>NPSV</td>
<td>40.1 ± 7.7</td>
<td>33.8 ± 7.3*</td>
<td>29.3 ± 7.1*</td>
<td>24.9 ± 6.5*</td>
<td>20.6 ± 5.4*</td>
<td>18.2 ± 5.9*</td>
<td>16.1 ± 2.9</td>
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<tr>
<td><strong>Borg Scale</strong></td>
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<tr>
<td>ST</td>
<td>7.5 ± 1.2</td>
<td>7.3 ± 1.5</td>
<td>7.1 ± 1.3</td>
<td>4.3 ± 1.4</td>
<td>2.8 ± 1.5</td>
<td>1.5 ± 1.3</td>
<td>0.7 ± 1.1</td>
</tr>
<tr>
<td>NPSV</td>
<td>7.9 ± 1.2</td>
<td>5.7 ± 1.7*</td>
<td>3.8 ± 1.6*</td>
<td>2.0 ± 1.5*</td>
<td>1.2 ± 1.3*</td>
<td>0.8 ± 1.4</td>
<td>0.7 ± 0.8</td>
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<tr>
<td><strong>Heart rate</strong></td>
<td></td>
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<tr>
<td>ST</td>
<td>118 ± 17</td>
<td>114 ± 16</td>
<td>109 ± 18</td>
<td>102 ± 19</td>
<td>98 ± 20</td>
<td>90 ± 17</td>
<td>86 ± 11</td>
</tr>
<tr>
<td>NPSV</td>
<td>123 ± 17</td>
<td>111 ± 18</td>
<td>104 ± 18*</td>
<td>98 ± 14</td>
<td>89 ± 14</td>
<td>83 ± 15</td>
<td>82 ± 12</td>
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<tr>
<td><strong>Mean BP</strong></td>
<td></td>
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<tr>
<td>ST</td>
<td>117 ± 22</td>
<td>116 ± 24</td>
<td>111 ± 23</td>
<td>101 ± 12</td>
<td>92 ± 27</td>
<td>88 ± 11</td>
<td>85 ± 8</td>
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<tr>
<td>NPSV</td>
<td>119 ± 18</td>
<td>111 ± 25</td>
<td>106 ± 17*</td>
<td>89 ± 20*</td>
<td>86 ± 14</td>
<td>83 ± 11</td>
<td>82 ± 9</td>
</tr>
</tbody>
</table>

**Definition of abbreviations:** BP = blood pressure; NPSV = noninvasive pressure support ventilation; ST = standard therapy.

* p < 0.05, NPSV vs. ST.

† p < 0.01, NPSV vs. ST.


