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

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 cmH\textsubscript{2}O), with an initial fractional inspired oxygen of 93.0–16% and a positive end-expiratory pressure of 3.5–2.3 cmH\textsubscript{2}O. 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, respectively. There were 5 (21%) failures and 21 (79%) successes. In both the success and 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 (Sp\textsubscript{O}\textsubscript{2}) 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 (PaCO\textsubscript{2}) 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: Sp\textsubscript{O}\textsubscript{2}, RR, heart rate, mean arterial pressure. The only differences between the success and failure groups were in the PaCO\textsubscript{2} (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.  Key words Noninvasive ventilation · Pressure support ventilation · Cardiogenic pulmonary edema · Acute respiratory failure · Face mask
Introduction

Noninvasive pressure support ventilation (NIPSV) has been proposed as an efficient alternative to conventional mechanical ventilation during acute exacerbations of chronic respiratory insufficiency [1–5]. The results are not as good for patients suffering from acute respiratory failure unrelated to chronic obstructive pulmonary disease (COPD) [6]. Some studies on ACPE have mostly used continuous positive airway pressure (CPAP) [7–9]. Otherwise, mechanical ventilation has been proposed as an added therapy to medical treatment because of its effects on intrathoracic pressure. Complications can result during intubation, during ventilation or after removal of the tube [10, 11]. The aim of this study was (1) to assess the short-term hemodynamic, respiratory and arterial blood gas effects of NIPSV in patients with ACPE who were likely to require endotracheal intubation, (2) to detect the initial causes of failure, and (3) to analyze the side effects and the difficulties of this technique.

Materials and methods

Patients

Over 6 months, 26 patients (17 men and 9 women) with ACPE and severe acute respiratory failure (ARDS), admitted to the intensive care unit (ICU) for treatment with an indication for mechanical ventilation, were prospectively and consecutively included. All patients had been transported to the ICU by ambulance and had been treated conventionally with nasal O2 (6 to 10 l/min) and diuretics without improvement.

Patients selected for NIPSV were those who required endotracheal intubation according to at least one of the following criteria: a respiratory rate (RR) greater than 30 breaths per minute, a pulse oximetry saturation (SpO2) below 90 % despite 6–10 l/min oxygen supplementation via a nasal catheter, severe dyspnea with use of accessory respiratory muscles or paradoxical abdominal motion. The level of consciousness was assessed by the Glasgow Coma Score (GCS). Cardiogenic pulmonary edema was defined by the association of the following criteria: a past history of cardiovascular disease, predisposing factors, cardiomegaly, bilateral alveolar and interstitial opacities and presence of crepitations on auscultation. The patients who required immediate intubation for cardiac arrest, bradypnea (RR < 8/min), shock or multiple organ failure were not included, nor were patients who refused the face mask or patients with a GCS under 10. This clinical protocol was conducted in accordance with the principles established in Helsinki.

Study design

NIPSV was administered to the patients via a face mask (Ambu, Bordeaux, France). The mask was adjusted to avoid air leaks and connected to a ventilator (CPU1, Ohmeda) set in the inspiratory pressure support (IPS) mode. The initial settings were as follow: the IPS level was set at 20 cmH2O and then adjusted for each patient in order to achieve a tidal volume between 7 and 10 ml/kg. We used a low level of positive end-expiratory pressure (PEEP) between 2 and 5 cmH2O to increase the arterial oxygen tension/fractional inspired oxygen (PaO2/FIO2) ratio and prevent atelectasis [6]. An FIO2 of 100 % was initially used and then gradually decreased after 30 min if the SpO2 was higher than 95 %. In the patients who developed hypoventilation under NIPSV, the ventilator mode was transiently changed to the assist-control volume mode.

NIPSV was considered a failure when intubation within the first 48 h was needed. Major criteria for intubation were standardized according to those published by Brochard et al. [2]: respiratory arrest, respiratory pauses with loss of consciousness or gasping for air, psychomotor agitation making nursing care impossible and requiring sedation, heart rate (HR) below 50 b/min with loss of alertness, and hemodynamic instability (systolic pressure < 70 mmHg). Other criteria for intubation were: the deterioration of clinical parameters (RR above the admission value, SpO2 < 90 % despite NIPSV or under 6–10 l/min of oxygen) associated with a refusal of the face mask; the necessity of keeping the face mask on for more than 12 h continuously; or the clinical judgment of the physician in charge. When the patient’s clinical status improved, ventilation support was used discontinuously or was stopped if clinical improvement remained stable. NIPSV weaning was started when the patients fulfilled the following conditions: FIO2 of 50 % with an RR < 25/min and an SpO2 > 90 %; NIPSV was resumed if the following conditions appeared within 60 min of spontaneous ventilation: RR > 25/min and SpO2 < 90 %.

Physiological measurements

The baseline assessment (before the start of NIPSV) of the patients included RR, SpO2, HR, mean arterial pressure (MAP), GCS, clinical signs of increase in respiratory muscle workload, electrocardiogram (ECG) chest X-ray, blood gases [PaO2, arterial carbon dioxide tension (PaCO2), pH], electrolytes, creatine phosphokinase (CPK), liver function tests and blood count. The severity of illness was assessed by the Simplified Acute Physiology II Score. The same physician performed transthoracic echocardiography on all of the patients.

The diagnosis, the etiology of ACPE, and the underlying heart disease were assessed by two physicians, one from the ICU and a cardiologist. The diagnosis of myocardial infarction (AMI) was established retrospectively on two of three of following: chest pain, increase in CPK and signs of myocardial necrosis on the ECG. Myocardial ischemia (MI) was diagnosed when there was a history of typical chest pain in combination with transient ST-segment depression or T-wave inversion. Congestive heart failure (CHF) was diagnosed if AMI or MI was absent and the patient was already receiving medication for CHF.

Clinical and biomedical parameters were monitored according to the following timing and regularly as long as necessary: clinical status at 15 and 30 min, 1 and 2 h; diuresis at 1 and 2 h; RR and SpO2 were also measured between 1 and 3 h after NIPSV was stopped.

All the side effects, mask intolerance and reasons for changes in the ventilator settings were recorded.

Statistical analysis

All values are reported as mean ± standard deviation (SD). The statistical analysis was made by a nonparametric test (Mann-Whitney U test) and a chi-square test with Yates’ correction in order to compare clinical and biological parameters at baseline between the success group (S) and the failure group (F). A nonparametric paired Wilcoxon test was next used to compare the earliest para-