Utility of Total Face-mask in a COPD patient
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Citation

Abstract
Noninvasive positive pressure ventilation (NPPV) has been shown to be an effective modality for the treatment of acute or chronic respiratory failure in patients with chronic obstructive pulmonary disease (COPD). Interest in NPPV has grown in recent years with the development of comfortable and effective masks, but the selection of an appropriate patient/ventilator interface may play a key role. Currently a variety of disposable NPPV masks are available, classified broadly as either nasal mask or face mask. Here, a case with COPD whose respiratory acidosis improved by total-face mask (TFM) was presented, and therefore we discussed the efficacy of TFM in acute respiratory failure.

INTRODUCTION
Noninvasive positive pressure ventilation (NPPV) has been shown to be an effective modality for the treatment of acute or chronic respiratory failure in patients with chronic obstructive pulmonary disease (COPD) (1,2). Interest in NPPV has grown in recent years with the development of comfortable and effective masks, but the selection of an appropriate patient/ventilator interface may play a key role (3). Currently a variety of disposable NPPV masks are available, classified broadly as either nasal mask or face mask. Both seem to be effective and each has advantages and disadvantages (4). The full-face mask may be more effective, because it eliminates mouth leaks, but because of greater surface contact, leaks can occur around the contact between the mask and the face, especially in edentulous patients. The selection of an appropriate mask has remained open to debate, as the 2 types have been directly compared in only one clinical trial that specifically analysed the efficacy and patient tolerance of nasal versus full-face mask (5).

Here, a case with COPD whose respiratory acidosis improved by only total face mask (TFM) was presented, and therefore we discussed the efficacy of TFM in acute respiratory failure.

CASE REPORT
A 69 year-old man admitted with severe dyspnea, cough, expectoration of purulent sputum, cyanosis and tendency to sleep. He had diagnosis of COPD and using NPPV and long term oxygen therapy together with his medical treatment since last 3 years. He had a history of 40 packs-year smoking, and he was ex-smoker for 20 years. His medical history revealed systemic hypertension and usage of antihypertensive medication.

Initial physical examination revealed that, he was semi-conscious, obese with barrel chest, dyspneic and using accessory respiratory muscles with supraclavicular, suprasternal and intercostal retractions, heaves and had flapping tremor, chemosis in his conjuctiva and cyanosis in his nails and mucosa. His physical examination was as follows: body temperature 36.8 oC, arterial blood pressure 154/72 mmHg, heart rate 96 beats/min, respiratory rate 40 breaths/min. Pulmonary auscultation revealed silent chest in both hemithoraces and inspiratory crackles in both basal areas. There were no heart murmurs, no peripheral oedema, no jugular vein distention. The liver and spleen were not palpable. The abdomen was soft and flat without tenderness or rigidity. His hemogram, blood chemistries and hemostasis tests were within normal limits. Arterial blood gas (ABG) analysis in room air was as the following pH:7.23, P$\text{CO}_2$: 79 mmHg, P$\text{O}_2$: 39 mmHg, O$_{\text{sat}}$%: 73 % and HCO$_3$-: 34 mEq/L (Table 1). Chest radiography revealed bilateral hyperlucency, increased bronchovascular images of the lung fields and enlargement of right descending pulmonary artery. Pulmonary function tests (PFT) revealed very severe bronchoconstriction; FVC: 560 ml(14%), FEV1: 450 ml (15%), FEV1/FVC: 80%, PEF: 990 ml (13%), FEF 25-75: 420 ml (14%) (Table 1). His ECG revealed sinus rhythm with incomplete right bundle branch block and left anterior hemiblock. His echocardiography revealed very severe
pulmonary hypertension with 60 mmHg mean and 75 mmHg systolic pulmonary arterial pressures by Doppler (Table 1). He had right atrial (40 mm) and right ventricular (44 mm) enlargement and right ventricular hypertrophy with 10 mm of right ventricular free wall diameter. However, right ventricular ejection fraction of the patient was within normal limits (65%).

**Figure 1**

Table 1: Arterial blood gases (ABG) with different NPPV pressures and masks in the patient with COPD acute attack.

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**DISCUSSION**

NPPV is safe and well tolerated in most properly selected patients, and in acute attacks it allows time for other conventional therapy to work, thus reversing the progression of respiratory failure, and reducing morbidity and mortality and also improvements occur in minute ventilation, respiratory rate and transdiaphragmatic activity. Intubation rates and complications associated with the use of mechanical ventilation were also reduced with noninvasive ventilation (1). It is well known that invasive mechanic ventilation (IMV) could cause several serious complications such as pneumonia and tracheal stenosis. However, a few rare complications of NPPV has been reported as single case reports (10, 11). In our patient, although respiratory acidosis insisted for 5 days after starting NPPV, he never lost his consciousness. Therefore, we changed the nasal mask with TFM since we did not want to intubate and apply IMV. Fortunately, he was very compatible to TFM and his ABGs were dramatically improved at one hour after instituting ventilation with that mask. Moreover, he had been protected from the potential complications of intubation and IMV.

Bronchodilator therapy, systemic corticosteroid, intravenous antibiotic treatment and NPPV were initiated with a fraction of inspired oxygen of 2 L, a positive end-expiratory pressure of 4 cm H₂O, and a pressure-support level of 8 cm H₂O with bilevel positive airway pressure (BIPAP) (Respironics Inc.; Murrysville, PA). One hour after this treatment, a little improvement seen in his clinical appearance and ABG analysis (pH: 7.24, PaCO₂: 66 mmHg, PaO₂: 51 mmHg, O₂sat: 77.9%, and HCO₃⁻: 28.4 mEq/L). So, BIPAP treatment with nasal and oro-nasal masks has been continued with the increased pressure supports (Table 1). Afterwards, his mental status improved and tendency to sleep gradually diminished, but his ABG results still insisted in respiratory acidosis and hypercapnia for an approximately 120 hours (5 days). Then, we changed his nasal mask with TFM, and his ABG analysis, PFT and mean and systolic PAPs were improved at the 11th day of NPPV treatment. ABG with different NPPV pressures and masks in the patient with COPD was shown in Table 1. He was discharged from the hospital with good ABG results.
lightweight plastic faceplate adds to the comfort and does not obstruct vision to help minimize claustrophobia in some patients. Some potential complications specific to the TFM such as gastric distention and aspiration of vomitus should be watched.

The outcome in a patient on NPPV depends on many factors. So, we should choose appropriate masks depending on the conditions of each patient to overcome respiratory failure.

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References
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