Noninvasive Ventilation in Critically Ill Patients

Cesare Gregoretti, MD[a,*], Lara Pisani, MD[b], Andrea Cortegiani, MD[c], V. Marco Ranieri, MD[d]

KEYWORDS

• Noninvasive ventilation • Acute respiratory failure • Critically patients

KEY POINTS

- Noninvasive ventilation (NIV) is widely used in the critical care area and it is the first-line intervention for certain forms of acute respiratory failure.
- Explore the results of clinical studies on NPPV is very important to avoid drawbacks and to reduce the rate of failure during its application.
- Understanding principle of functioning of ventilator and modes will lead the operator to choose the best approach for his/her patients.

INTRODUCTION

Noninvasive ventilation (NIV) refers to the delivery of noninvasive intermittent positive pressure ventilation (NPPV) or noninvasive continuous positive airway pressure (CPAP) through the patient's mouth, nose, or both via an external interface. In contrast with conventional invasive mechanical ventilation (IMV) delivered via endotracheal tube or tracheostomy, NIV does not interfere with the patient's native upper airways and overall it does not impair glottis function. It may reduce the patient's effort and improve gas exchange while preserving the ability to swallow, cough, and speak. In addition, NIV may avert iatrogenic complications associated with invasive ventilation (ie, complications associated with endotracheal intubation)[1] and may reduce the risk of infections (ie, ventilator-associated pneumonia).[2]

[a] Department of Anesthesia and Intensive Care, Azienda Ospedaliero Universitaria "Città della Salute e della Scienza", Corso Dogliotti 14, Turin 10126, Italy; [b] Department of Specialistic, Diagnostic and Experimental Medicine (DIMES), Respiratory and Critical Care, Sant'Orsola Malpighi Hospital, Alma Mater Studiorum, University of Bologna, Via Massarenti 9, Bologna 40126, Italy; [c] Department of Biopathology, Medical and Forensic Biotechnologies (DIBIMEF), Section of Anesthesiology, Analgesia, Emergency and Intensive Care, Policlinico "P. Giaccone," University of Palermo, Palermo, Italy; [d] Department of Anesthesia and Intensive Care, Sapienza Università di Roma, Ospedale Policlinico Umberto I, Viale del Policlinico 155, Roma 00161, Italy
* Corresponding author. Department of Anesthesia and Intensive Care, Azienda Ospedaliero Universitaria "Città della Salute e della Scienza", Corso Dogliotti 14, Turin 10126, Italy.
E-mail address: C.gregoretti@gmail.com

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The rationale behind its use can be divided in 2 distinct categories:

1. Patients with lung failure caused by alveolar perfusion mismatching (ie, the ratio of arterial oxygen partial pressure to fractional inspired oxygen (PaO_2FiO_2) <300] and hypoxemia but without hypercapnia). In these patients the rationale of using NIV is to open derecruited alveoli, maintain them open, and decrease the patient's dyspnea through a reduction of the work of breathing (WOB) and respiratory rate.
2. Patients with ventilatory pump failure with hypercapnia and respiratory acidosis ($Paco_2$ >45 mm Hg and pH <7.35). This subgroup should be further divided into:
   a. Hypercapnia caused by an acute exacerbation of chronic obstructive pulmonary disease (AECOPD) in which carbon dioxide (CO_2) increase is not homogeneous in the lung. The reduction of CO_2 level is obtained with an amelioration of alveolar ventilation and an amelioration of ventilation-perfusion mismatching. In these patients WOB may be reduced by resting the respiratory muscles (patients are fatigued but not weak) and also counterbalancing intrinsic positive end-expiratory pressure (PEEPi).[3–5]
   b. Hypercapnia caused by genuine alveolar hypoventilation (ie, neuromuscular patients or other restrictive chest/lung disorders). In these patients, in contrast with patients with AECOPD, the reduction of WOB is achieved mainly by resting the respiratory muscles. Some patients with neuromuscular disorders have weak respiratory muscles and are thus incapable of regaining muscle strength[6] after resting on NPPV. Some patients in this subgroup need to counterbalance with PEEPi (ie, patients with severe obesity or quadriplegic patients in supine position).[7,8]
   c. Hypercapnia in end-stage interstitial lung diseases. These patients show impaired oxygen diffusion and Ventilation-Perfusion ratio (VA/Q) inequalities; the level of hypoxemia depends critically on the interplay between mixed venous Po_2 and the degree of VA/Q mismatching. Thus patients with a high cardiac output can have little hypoxemia if there are severe VA/Q mismatching, whereas patients with inadequate cardiac output can have moderately severe hypoxemia with little VA/Q mismatching; in contrast with patients with acute de novo hypoxemia, in whom pulmonary shunt is often present, the arterial Po_2 alone cannot indicate the severity of VA/Q mismatch in any given situation. However although these patients are often normocapnic/hypocapnic in the early stage of the disease, persistent hypercapnic respiratory failure is a feature of advancing end-stage interstitial pulmonary disease caused by both severe alteration in pulmonary diffusion and pump failure.[9,10]

As a consequence, although hypoxia and/or hypercapnia and muscle fatigue are always the basis of NIV treatment, the NIV settings and the clinical response may be different for different causes of acute respiratory failure (ARF).

This article provides physicians and respiratory therapists with a comprehensive, practical guideline for using NIV in critical care.

EPIDEMIOLOGY OF NONINVASIVE VENTILATION

Since its first application in the late 1980s,[11] NIV has become a first-line intervention for certain forms of ARF.[12–14] However, NIV is still underused in certain parts of the world because of lack of experience and inadequate training and economic resources.[15,16] In Europe, NIV use ranges from 35% of ventilated patients in intensive care units (ICUs) to about (60%) in respiratory ICUs (RICUs) or emergency departments (EDs).[16] In North America, where NIV is most often begun in the ED,