Noninvasive Ventilation in Hypoxemic Patients: an Ongoing Soccer Game or a Lost One?

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Noninvasive ventilation (NIV) is nowadays a medical intervention used worldwide in daily practice in many clinical settings (1). Almost 35 years have been passed by since the first clinical studies on the use of NIV in the critical area (2, 3). NIV has been increasingly used to avoid or to serve as an alternative to invasive mechanical ventilation (IMV) (4). Compared with standard medical therapy (STM), and in some cases with IMV, NIV has been found to improve survival and reduce complications in selected patients with acute respiratory failure (ARF). The main indications are exacerbation of chronic obstructive pulmonary disease (COPD), cardiogenic pulmonary oedema, pulmonary infiltrates in immunocompromised patients, weaning of previously intubated stable patients with chronic obstructive pulmonary disease, postoperative patients, terminally ill patients, or as ventilatory assistance during invasive procedure as bronchoscopy (4). NIV has also been used in the so labelled “de novo” acute hypoxemic respiratory failure and acute respiratory distress syndrome (ARDS). Nevertheless, after many years from NIV institution in ICU (3) its use in hypoxemic patient not only need to be better “tuned” but it also seems to be decreasing compared to the last ten years (5, 6). In this patient population despite a satisfactory initial response (7), late failure may occur leading to increases in mortality rate (5). The possible causes of immediate failure may be due to excessive secretions, interface intolerance and agitation, and severe patient-ventilator asynchrony. However beyond these risk factors there are possibly some differences between failing in a hypercapnic or in hypoxemic ARF. In patients with hypoxemic ARF, delayed intubation may lead to an increase mortality while it does not occur in COPD patients (8). In addition, in hypoxemic patients keep using NIV and delaying intubation may expose the deleterious effects on an increased transpulmonary pressure. The major determinant of lung stress, the transpulmonary pressure, is the result from the sum of the pressure applied to the airway by the ventilator and the pleural pressure generated by the patient’s spontaneous effort (9). The pressure generated by the respiratory muscle (respiratory effort) added to the level of patient-synchronized pressure support level (10) may generate high...

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Non-Invasive Ventilation in Hypoxemic Respiratory Failure/ARDS

DEBATE

First Round

GREGORETTI, CORTEGIANI, RAINERI, GIARRJATANO VS. HILL, GARPESTAD, SCHUMAKER, SPOLETINI

tidal volumes that are far from the suggested “safe” level of tidal volume suggested to protect the lung (11). In addition the level of PEEP may be insufficient to recruit consolidated lung areas (12, 13). This in turn may cause a “self-induced ventilation lung injury” (14). Last but not least, it has been recently found that to improve the outcome in ARDS patients, one needs to identify potentially modifiable factors associated with mortality. Higher positive end expiratory pressure (PEEP), lower peak, plateau, and driving pressures, and lower respiratory rate seem to be associated with improved survival from ARDS (15). Carteaux et al. (16) found that expired tidal volume was one of the factors determining NIV failure. In their study they found that patients with a tidal volume above 9.5 mL kg⁻¹ of predicted bodyweight had increased risk of NIV failure. Interestingly, the relationship between NIV success or failure and expired tidal volume was observed only among patients with moderate-to-severe hypoxaemia (PaO₂/FiO₂ ≤ 200), and was not found in patients with milder (PaO₂/FiO₂ > 200) degrees of hypoxaemia (17). Frat et al. (18) found that the use high-flow nasal cannula (HFNC) reduced ICU and 90-day mortality as compared to standard oxygen and NIV. The authors speculated that the greater mortality with NIV might have been related to the use of tidal volumes greater than 9 mL kg⁻¹, predisposing to ventilator-induced lung injury (VILI). However, NIV was used intermittently and not continuously. The level of noninvasive pressure-support was only of 8±3 cmH₂O of water, a PEEP only of 5±1 cmH₂O. The level of noninvasive pressure-support was only of 8±3 cmH₂O of water, a PEEP only of 5±1 cmH₂O. In addition it can be hypothesized that the use of other interfaces as helmets could have increase patient tolerability and time on NIV (19). Patel et al. (19) found that among patients with ARDS, treatment with helmet NIV resulted in a significant reduction of intubation rates. There was also a statistically significant reduction in 90-day mortality with helmet NIV. Nevertheless, although NIV failure seemed not to longer associated with higher mortality rate suggesting improved patient selection (13). Bellani et al. (20) found that NIV failure occurred in 22.2% of mild, 42.3% of moderate and 47.1% of patients with severe ARDS. Hospital mortality in patients with NIV success and failure was 16.1% and 45.4%, respectively. NIV use was independently associated with increased ICU (HR 1.446-95% CI 1.159-1.805), but not hospital mortality. In a propensity-matched analysis, ICU mortality was higher in NIV than invasively ventilated patients with a PaO₂/FiO₂ lower than 150 mmHg. Among immunocompromised patients admitted to the ICU with hypoxemic ARF, early NIV compared with oxygen therapy alone did not reduce 28-day mortality (21). However the median durations of NIV were 8 hours (interquartile range [IQR], 4-11) in the first 24 hours, 6 hours (IQR, 4-8) on day 2, and 5 hours (IQR, 3-7) on day 3. Overall, in the first 72 hours the patients received NIV for a median time of 19 hours only, potentially too few for NIV to be effective in reducing the intubation rate (9). The results of the post-hoc analysis study of Frat et al. (22) indicates that oxygen delivery through HFNC was associated with lower mortality and a lower risk of IMV compared with NIV in immunocompromised patients. However, although the patients were ventilated with 7-10 mL kg⁻¹ of expired tidal volume, the amount of tidal volume that reached the lungs was probably not more than 5-8 mL kg⁻¹ of bodyweight because of dead space thus making unlikely to lead to substantial likelihood of developing VILI (17). In conclusion, NIV in hypoxemic patients still remain a hot topic. Although clinicians should be aware of the possible harmful effects of using NIV in hypoxemic patients there are still many issues to discuss. Among them the experience and familiarity to NIV use, which hypoxemic patient do we have to prefer to undergo NIV (i.e. ARDS vs non ARDS), which patients should need immediate IMV, inspiratory synchronization especially when high VT is undesirable. Maybe, we can reason in another way starting a new soccer game: late vs early extubation (using NIV as tool to early extubate our patients) in hypoxemic patients to prevent IMV complications (23-25).

References


