

# Failure of non-invasive mechanical ventilation in acute hypercapnic respiratory failure: Still, there are more things to learn

Sir,

Gursel *et al.* provide great and original contributions to understand non-invasive mechanical ventilation (NIV) response in a broad population of patients with acute hypercapnic respiratory failure (AHRF).<sup>[1]</sup> In this study, there are two major findings relevant, such as prior home mechanical ventilation (HMV) and high levels of pressure support (PS) which are predictors of NIV failure in AHRF. We would add that these issues are highlight relevant and extend information to acute strategy in NIV failure prevention and treatment. However, there are some limitations of the study to improve the discussion that may achieve overall interpretation of results and should be taken into account.

First, previous HMV is a risk factor for failure, as few studies have highlighted this as a factor in the NIV failure.<sup>[2]</sup> However, this aspect still remains controversial since some previous studies published results in the opposite direction.<sup>[3]</sup> We consider that previous HMV has potential influence for NIV failure through other mechanisms, such as: (1) a worse baseline functional status, muscular respiratory fatigue, and severe air trapping auto-positive end-expiratory pressure (auto-PEEP); (2) adaptive changes among nasal to facial interface is a well-known intolerance factor; and (3) incompatibility of NIV equipment and parameters such as trigger (ventilator-patient asynchrony).<sup>[3]</sup>

Second, the point of view of design and analysis methodology in some aspects needs considerations: (a) heterogeneous mixture of patients with the need for a better definition of AHRF (chronic obstructive pulmonary disease, Obesity hypoventilation syndrome, Overlap syndrome) where pathophysiology and interaction with NIV in acute setting are critical factors to understand failure. (b) Criteria to select NIV parameters and setting used. In this sense, there is no clear explanation for efficiency of PS mode and tidal volume target (VT) in decreasing the PaCO<sub>2</sub> levels and pH. Did the authors target decrease of PaCO<sub>2</sub> who had normal pH? The authors considered the implementation strategy that PS-VT may be more appropriate for a volume of 6-8 ml/kg instead of a 450-500 ml for every patient. Nevertheless, this strategy

cannot be extrapolated to all forms of etiologies in AHRF. Other factor that this study was not adequately assessed with a potential contribution could be the level of hypoxemia response and NIV-failure, e.g., fraction of inspired oxygen (FiO<sub>2</sub>) levels applied is unknown. (c) The criteria established to the indications to endotracheal intubation are not explained in detail. (d) Sedation drugs used reduces the rate of pCO<sub>2</sub> control.

Third, readmission rate, as NIV response, is not supported by authors in their conclusions. This is a great relevant aspect to consider in NIV failure as it was not evaluated as determined in previous studies. In this regard, the relationship with prognosis in acute stage is a well-established factor.<sup>[4,5]</sup>

We believe that this study provides a substantial baseline and profile information for the planning of decision-making strategies and appropriate treatment in the first day of therapy. Further prospective studies are required to validate these observations.

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