Noninvasive Ventilation for Acute Hypercapnic Respiratory Failure: Is It the Same as in Hypercapnic Coma?

To the Editor:

I have read with interest the original article entitled, "Noninvasive ventilation for acute hypercapnic respiratory failure: intubation rate in an experienced unit."¹ In this paper, the authors prospectively evaluated 242 patients who received noninvasive ventilation (NIV) for acute hypercapnic respiratory failure in the presence of COPD or other causes not associated with COPD and for acute hypercapnic respiratory failure in the absence of chronic obstructive diseases. The authors found severe hypoxemia as an independent factor of failure in hypercapnic patients from any source. Alterations at the sensory level have been reported, and ventilatory settings do not influence the results.

I have some remarks on this study. The authors reported 31 (12.8% of all patients studied) hypercapnic coma patients either on admission (15 patients) or during the first 24 h (16 patients). The management of hypercapnic coma patients, which can be measured by the Glasgow coma scale² and the Kelly-Matthay scale,³ differs from that of patients with an altered level of consciousness who have not reached hypercapnic coma, especially regarding the levels of pressure support used during the first hours or target volume.

The authors found no significant differences in the levels of pressure used between the two groups, with a support pressure of 9.2 ± 2.6 cm H₂O (NIV success) versus 9.4 ± 2.8 (NIV failure). Díaz et al⁴ used BiPAP Vision or BiPAP S/T-D 30 (Philips Respironics, Murrysville, Pennsylvania), and inspiratory positive airway pressure (IPAP) was initially programmed at 12 cm H₂O and increased every 4 h, with an IPAP in the first hour of 17 ± 2 cm H₂O. Briones Claudett et al⁵ reported an IPAP baseline of 19.82 in the bi-level positive airway pressure spontaneous/timed (BPAP S/T) group with average volume-assured pressure support. Therefore, the use of pressure levels in this study in hypercapnic coma patients must be considered independently of the pressure levels used in patients with impaired sensory level that are without hypercapnic coma because levels may be below those routinely used in daily practice.^{6,7} In contrast, an underestimation of pressure

support or IPAP levels in this subgroup of patients may affect early clearance of P_{CO₂} in the blood and especially in the cerebrospinal fluid, prolonging coma and maintaining intubation risk for these patients. Furthermore, the authors found no significant differences in the tidal volume (V_T): 475 ± 140 (NIV success) versus 415 ± 166.06 (NIV failure).

We found a significant improvement in quick minute volume in patients with hypercapnic coma⁶ with rapid recovery of sensory level comparing the BPAP S/T-only group versus the BPAP S/T with average volume-assured pressure support group (BPAP S/T-only, 304 ± 60.6 vs 531.1 ± 63.6 ; BPAP S/T with average volume-assured pressure support, 298.6 ± 54.3 vs 617.6 ± 77.4 ; $P = .01$).

The rapid recovery of sensory level in these patients is also linked to an improvement in the exhaled V_T, which quickly reaches the levels required to maintain an appropriate V_T and correct hypoventilation, improving alveolar ventilation. The presence of secretions, which are essential in evaluating the failure prevention technique and endotracheal intubation, has not been evaluated. We believe that these assessments should be taken into account when analyzing these results.

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The author has disclosed no conflicts of interest.

DOI: 10.4187/respcare.03128

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Noninvasive Ventilation for Acute Hypercapnic Respiratory Failure: Is It the Same as in Hypercapnic Coma?—Reply

In Reply:

We read with a great interest the comments made by Dr Killen H Briones Claudett concerning adjustments of ventilatory settings during noninvasive ventilation (NIV) to treat subjects with hypercapnic coma. In a recent original article published in the December 2013 issue of *RESPIRATORY CARE*,¹ we reported an overall intubation rate of 15% in a cohort of 242 subjects receiving NIV for acute hypercapnic respiratory failure of all origins. After adjustment, acidosis and severe hypoxemia after 1 h of NIV initiation were independently associated with NIV failure, whereas altered consciousness on admission and ventilatory settings had no influence on outcome. Altered consciousness was defined using the Richmond Agitation-Sedation Scale (RASS),² and in all of the subjects who had encephalopathy at admission (defined as RASS < 0), the rate

of intubation was only 23% (14/60). However, this rate reached 52% (16/31) in those who were comatose during the first 24 h (defined as Glasgow coma scale ≤ 8). It has already been found that NIV can be successful in subjects with hypercapnic coma, and NIV failure rates of only 20% have been reported.³ It is important to note that patients who succeed with NIV have a faster improvement of consciousness compared with those who need intubation.³ Dr Briones Claudett emphasizes that subjects with altered consciousness may require higher levels of pressure support than those with normal consciousness. To support this, Briones Claudett et al⁴ recently found a faster recovery from hypercapnic encephalopathy using ventilatory mode with average volume-assured pressure support compared with pressure support ventilation during NIV due to higher levels of ventilatory assistance and larger tidal volumes (V_T).

In our cohort, of the 31 subjects who developed hypercapnic coma, 15 were comatose upon admission, and 16 developed coma during the first 24 h while receiving NIV.¹ The rate of intubation was only 20% (3/15) in subjects who were comatose at admission, in line with the results found by Díaz et al.³ By contrast, the rate of intubation was 81% (13/16) in those who developed delayed coma during NIV ($P = .001$). Subjects with hypercapnic coma upon admission had similar V_T values compared with other subjects (459 ± 175 vs 469 ± 142 mL, $P = .82$). However, they received higher pressure support levels (11.9 ± 2.7 vs 9.1 ± 2.5 cm H₂O, $P = .008$), meaning that the level of ventilatory assistance had been correctly adjusted according to our protocol that aims to target predetermined V_T . As we used exclusively ICU ventilators, inspiratory positive airway pressure, including pressure support and PEEP, reached 16.5 ± 2.8 cm H₂O in subjects who were comatose at admission, a value close to that reported in the abovementioned studies.^{3,4}

We agree that adjustment of an adequate pressure support level is the key setting to reverse hypercapnic coma, and we believe that our good results are due in part to our protocol of adjusting the pressure support level to target a minimal V_T , as would average volume-assured pressure support. Altered consciousness at admission does not seem to increase the risk of NIV failure. By contrast, al-

most all subjects who developed delayed coma needed intubation, whereas their V_T values and their pressure support levels adjusted at admission were similar to those of other patients (417 ± 142 vs 471 ± 142 mL, $P = .27$; 9.1 ± 7.4 vs 9.2 ± 2.6 cm H₂O, $P = .88$). Therefore, NIV failure was probably due to a patient's worsening and/or failure of this treatment and not to inadequate adjustment of ventilatory settings.

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The authors have disclosed no conflicts of interest.

DOI: 10.4187/respcare.03247

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Simulation Studies for Device Evaluation

To the Editor:

According to the Society for Simulation in Healthcare, "Simulation is the imitation or representation of one act or system by another. Healthcare simulations can be said to have 4 main purposes – education, assessment, research, and health system integration in facilitating patient safety."¹ Implicit in the concept of simulation is the understanding that the parameters of the simulation reflect realistic values of the system under study. If the model does not accurately represent the system being simulated, then any conclusions about the how the real system behaves based on how the model behaves are suspect.

Simulation for Ventilator Performance Studies

Simulation studies are often used to examine ventilator performance because models of the respiratory system are much easier to understand and experiment with than real respiratory systems (either human or animal). More importantly, models do not vary with time, so the differences observed in measurements are presumed to be related only to performance differences among the ventilators in the study. The simplest model of the respiratory system used for ventilator studies is the single-compartment model, composed of a single-flow resistance and a single-elastic compartment, represented by the equation of motion for the respiratory system (Equation 1).

$$P_{TR}(t) + P_{mus}(t) = EV(t) + R\dot{V}(t) + \text{auto-PEEP} [1]$$

where $P_{TR}(t)$ = the change in transrespiratory pressure difference (ie, airway opening pressure minus body surface pressure) as a function of time (t), measured relative to end-expiratory airway pressure. This is the pressure generated by a ventilator during an assisted breath; $P_{mus}(t)$ = ventilatory muscle pressure difference as a function of time (t); the theoretical chest wall transmural pressure difference that would produce movements identical to those produced by the