Neurally Adjusted Ventilatory Assist vs Pressure Support Ventilation During Noninvasive Mechanical Ventilation

Another Physiologic Evaluation to Consider?

To the Editor:

Neurally adjusted ventilatory assist (NAVA) is an original approach to noninvasive ventilation that can improve patient-ventilator interaction and may influence physiologic outcomes because the electrical activity of the diaphragm, a pneumatically independent signal, is used to control the timing and level of assist provided, regardless of the interface used. In a recent issue of CHEST (January 2013), Bertrand et al compared patient-ventilator interaction during pressure support ventilation (PSV) and NAVA applied noninvasively and showed reductions in all of the following with NAVA: ineffective efforts, trigger and cycling-off delays, and inspiratory times. This, thus, demonstrated a lower “severe asynchrony” defined by an asynchrony index (>10%), but despite these positive findings in patient-ventilator interaction, the clinical impact was not so clear and deserves comment.

First, there were no significant changes in the oxygenation index when patient-ventilator synchrony improved with NAVA. This finding is relevant because it is not in accordance with the findings of previous studies. It could be hypothesized that the restoration of diaphragmatic activity contributes to a recruitment of poorly ventilated areas. In this study, were the selection positive end-expiratory pressures too low and were the observation periods too short? Furthermore, in terms of practical implications, is the expected oxygenation improvement correlated to a reduced patient-ventilator asynchrony index or to independent factors?

Second, the ventilatory parameters selected (PSV and positive end-expiratory pressure) were restricted and were applied for short observation times, which may have limited the extension of the results outside this protocol. Third, the tidal volume during PSV was significantly higher than during NAVA (Table 3, tidal volume 515 mL [410-593] in the PSV group, 498 mL [421-663] in the NAVA group). Does this finding affect the PSV-increased asynchrony index in this group? Moreover, were the selection parameters of PSV high or did they not require pressure support but simply continuous positive airway pressure? We can hypothesize that the high level of tidal volume was responsible for the asynchrony. Furthermore, we may ask what are the criteria for tidal volume selection and its relationship with the uncomfortable respiratory pattern of the PSV trial?

One final interesting question is, how do physiologic studies influence the discomfort during PSV and NAVA and what is the degree of correlation? No information regarding the involvement of the respiratory center and the subsequent discomfort is reported. Does one need to know how the brain operates to understand the lungs? Or is this another physiologic evaluation to consider? This is an open question that may influence results and interpretation.

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Response

To the Editor:

We thank Dr Esquinas for his comments on and interest in our study. We agree that a physiologic evaluation cannot resolve all uncertainties and that further studies are mandatory to define the scope of the neurally adjusted ventilator assist (NAVA) mode. That being said, we would like to address his comments.

First, we are doubtful about the proposed contribution of diaphragmatic activity in the recruitment of poorly aerated lung areas, which refers to a dynamic process of reopening collapsed lung units through a transient increase in transpulmonary pressure. The same applies for the possible correlation between an improved patient-ventilator interaction with the NAVA mode and an increase in the oxygenation index, which is a complex...
parameter that depends on multiple factors, such as flow and cardiac index. Although no difference in oxygenation was evident between the two study groups, each of the two modes (pressure support ventilation and NAVA) significantly improved oxygenation during noninvasive ventilation (NIV) compared with baseline. This is, to some extent, good news in that NAVA can be reliably and safely delivered to patients receiving NIV.

Second, although we agree that some of the patients could have benefited from higher positive end-expiratory pressure levels, both positive end-expiratory pressure (between 5 and 10 cm H2O) and tidal volume setting (to achieve a tidal volume of 6–8 mL/kg of ideal body weight) were (1) standardized to reflect common clinical practice during NIV for acute respiratory failure3,4 and (2) close to that used by Terzi and colleagues5 in the physiologic evaluation of patients with ARDS. Third, we agree that excessive levels of assistance could affect the asynchrony index during NIV. Nevertheless, the difference in tidal volumes was not significant (pressure support ventilation group, 8 mL/kg [range, 6–8 mL/kg]; NAVA group, 8 mL/kg [7–8 mL/kg]; P = .08) and, in any event, was not clinically relevant. Finally, we are convinced that physiologic assessment is an indispensable prerequisite of clinical application and an add to our knowledge.

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Backup Respiratory Rate During Noninvasive Positive Pressure Ventilation in Obesity Hypoventilation Syndrome
Can This Difficult Puzzle Be Resolved?

To the Editor:

Obesity hypoventilation syndrome (OHS) refers to sleep-related hypoventilation with repetitive episodes of complete and partial obstructions of the upper airway.1 Some finesse is required to determine the appropriate ventilator settings to prevent such episodes, which can alter the efficacy of noninvasive positive pressure ventilation (NPPV).

In a recent article in CHEST (January 2013), Contal et al2 analyzed the effects of three strategies: a spontaneous (S) mode, a low backup respiratory rate (BURR), and high BURR. The S mode was worse than the S/T mode, and changing the BURR from an S/T mode with a high or low BURR to an S mode was associated with the occurrence of a highly significant increase in respiratory events and oxygenation desaturation index events.

It is worth highlighting some features of this study, which help place the findings in context. First, the population selected had some interesting characteristics. The prior use of NPPV for at least 42.7 months (duration of NPPV) and a baseline BURR of 14 may have influenced the results. Specifically, patients who had already been acclimated to NPPV might have adjusted more easily to a range of BURR. Second, a high BMI of 48.5 kg/m2 could also have reduced the efficacy of NPPV, making it difficult to generalize from these results to the entire spectrum of OHS.3 Third, the authors did not consider the potential effects of upper airway obstruction during sleep (obstructive apneas and hypopneas), which are common in severely obese patients and could further reduce the therapeutic efficacy,4 depending on the algorithm for setting the respiratory pressure. Fourth, it is difficult to determine the effect of the NPPV strategy on PaCO2 over the relatively short period of intervention in this study in the group that was not hypercapnic at baseline (pH 7.44, PaCO2 41.3, bicarbonate, 28.1). Other outcomes would be required to assess the acute effects of NPPV in this group (Table 1 in Contal et al).5 Fifth, the oxygenation desaturation index was higher during periods of S-mode NPPV as compared with the S/T mode with low BURR and the S/T mode with high BURR (Table 2 in Contal et al).4 It is possible that several factors could have accounted for these findings, including decreased lung volume and hypoventilation during sleep, especially in patients with a marked elevated BMI.3

Finally, this study did not characterize conventional parameters describing patient/ventilatory asynchrony.5 I believe that the BURR is an important tool for treating hypoventilation in OHS. Further studies are necessary to establish best practices to maintain adequate ventilatory support and to achieve long-term outcomes.

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