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 failure and (2) close to that used by Terzi and colleagues 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. 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 al analyzed the effects of three strategies: a spontaneous (S) mode, a low backup respiratory rate (BURR), and a 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.

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 depending on the algorithm for setting the expiratory 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; Pco2, 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).

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). 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 markedly elevated BMI.

Finally, this study did not characterize conventional parameters describing patient/ventilatory asynchrony. 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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Response

To the Editor:

We would like to submit the following answers to the comments made by Dr Esquinas on our study on backup respiratory rate (BURR) in subjects with obesity hypoventilation syndrome (OHS).

1. The population under evaluation was indeed not naive to noninvasive positive pressure ventilation (NPPV). This was a deliberate choice, as we wished to analyze the impact of changes in BURR in subjects with OHS under noninvasive ventilation with as few other changes as possible. Thus, studying a population of patients familiar with NPPV certainly had a favorable impact on their acceptance of the protocol. This is a study limitation stated in the “Discussion” section. Performing the same study in subjects naive to NPPV may have been associated with more important changes in nocturnal PtcCO₂, although this remains to be documented.

2. Patients had a high BMI, but this had no effect on the efficacy of NPPV. Patients were normocapnic during daytime, with an appropriate correction of average nocturnal transcutaneous CO₂ monitoring (PtcCO₂) and oxygen saturation as measured by pulse oximetry under NPPV. Apnea-hypopnea indices obtained under NPPV were in fact quite acceptable for patients with such a high BMI. The majority of the studies in the field reported BMIs > 40 kg/m² both in the United States and in Europe. 1,2

3. It is widely accepted that 90% of patients with OHS have upper airway obstruction, and, thus, we cannot accept that the potential effects of upper airway obstruction were not considered in patients. Average expiratory positive airway pressure values were 9.2 ± 1.8 cm H₂O (ie, far above what is used in ventilated obstructive or restrictive patients without associated obstructive sleep apnea syndrome); this clearly shows that upper airway obstruction was taken into account. Pressure settings were in agreement with available publications on long-term NPPV in patients with OHS and similar BMI. 3–7 However, all patients in this study were put under noninvasive ventilation after an acute episode of hypercapnic respiratory failure and not electively. Polysomnograms performed during our study show a residual obstructive apnea-hypopnea index of 8.6/h under high BURR, vs higher values under low or no BURR, which shows to what extent BURR per se can be an important factor for stabilizing the upper airway.

4. We agree that these patients were stabilized, and, thus, daytime PaCO₂ was normalized, which is one of the main goals of NPPV. One can assume that to some extent ventilator drive and ventilator response to CO₂ were improved when compared with baseline. Therefore, the same study in subjects naive to NPPV may have been associated with more important changes in nocturnal PtcCO₂, although this remains to be documented.

5. There is no evidence to support this hypothesis: Neither total ventilation nor tidal volume significantly decreased under spontaneous (S) mode (in fact, they were slightly higher than under both S/T modes). PtcCO₂ clearly shows that there was no hypoventilation under S mode; in all modes, in spite of high BMI values, correction of alveolar hypventilation was satisfactory.

6. Patient/ventilator asynchrony has been an interest of our group for several years and was specifically looked for in this study. We did not find significant patient/ventilator asynchrony on polysomnogram tracings in this study, whatever mode was considered.

In conclusion, we have clearly shown that an S mode is associated with more frequent central and obstructive respiratory events when compared with S/T modes with low or high BURR in severe OHS and high BMI. This shows that BURR settings per se can have an important influence on the quality of NPPV. The impact of BURR must be further assessed in naive subjects with OHS and in other patient groups (ie, COPD, neuromuscular diseases) to better understand its importance in these clinical settings.

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