



LETTER TO THE EDITOR

Reply to Pressure-controlled ventilation and sleep in COPD patients in the intensive care unit: The role of tidal volume?


To the Editor:

We thank Dr Esquinas Rodriguez and Pr BaHammam for their comments on our study recently published in *Respiratory Medicine* [1]. They raise three issues and first hypothesize that the poor sleep quality we observed with low levels (6 cm H₂O) of pressure support ventilation (PSV) could be attributed to central apneas. Indeed, Parthasarathy and Tobin found that sleep fragmentation related to central apneas was significantly greater during PSV when compared to assist-controlled ventilation (ACV) [2]. All our 26 patients were monitored with standard polysomnography (including recording of rib cage and abdominal motion through inductance plethysmography) and no apneas were noted during low-PSV as well as during PCV (see “polysomnographic results”, page 536). With PSV, higher pressure levels (leading to higher tidal volumes) than those used in our study are needed to decrease the patients’ CO₂ level under the apneic threshold [3] and to induce central apneas (i.e. mean pressure-support level of 16.8 ± 1.5 cm H₂O in the Parthasarathy and Tobin’s study [2]). Moreover, central apneas could occur with PSV because the respiratory frequency is determined in this mode by patients themselves. By contrast, because the respirator-frequency was fixed at ≥12/min, absence of recorded patient’s tidal volume longer than 10 s in duration (i.e. definition of apneas) could not occur during PCV. For all these reasons, we are confident that the differences we observed in sleep architecture between PCV and low-PSV are not related to such events.

Secondly, the authors emphasize the potential role of CO₂ on sleep quality. We agree that end-tidal carbon dioxide (EtCO₂) or transcutaneous carbon dioxide tension (T_{cp}CO₂) monitoring are lacking in our study. However, the respiratory parameters collected in our COPD patients give us some relevant information. Resting respiratory

muscles with PCV was associated with both a better breathing pattern and a better sleep quality and quantity when compared to low-PSV which was associated by contrast to some rapid, shallow breathing. Indeed, four hours of low-PSV resulted in lower tidal volume, higher spontaneous respiratory frequency but similar minute ventilation than with PCV (respectively: 11.8 ± 2.6 L/min vs 11.1 ± 2.6 L/min, $p = 0.4$) suggesting an increase in dead-space ventilation with low-PSV. This pattern of breathing which is frequently encountered in COPD patients during the weaning period is known to lead to CO₂ retention [5]. This is the key result of our study showing that the disrupted sleep observed with PSV was not related to hypocapnic central apneas (as in the well known Parthasarathy and Tobin’s study) [2] but to respiratory muscles fatigue (with probable hypercapnia). To provide rest without inducing central apneas, breathing must be captured by using not only a sufficient tidal volume but also a sufficient respirator-frequency. Thus, controlled modes are imperative and, in our study using PCV, a 20 cm H₂O inspiratory pressure support with a ≥12/min respirator-frequency allowed complete disappearance of spontaneous inspiratory efforts. Such high pressure level explains the high tidal volumes we observed in our COPD patients who were sometimes nearly obese.

Finally, the aim of our studies was not to favor PCV [1] or ACV [4] versus PSV during the weaning in COPD patients hospitalized for acute on chronic respiratory failure but to draw intensive care teams’ attention to their patients’ sleep quality. According to our results, resting the respiratory muscles during night and encouraging weaning during the day seems to be an appropriate compromise but, to coin a phrase, large prospective multi-center studies are needed to clarify this important area...

Authors have not any potential conflict of interest.

References

- [1] Andr jak C, Monconduit J, Rose D, Toubanc B, Mayeux I, Rodenstein D, Jounieaux V. Does using pressure-controlled ventilation to rest respiratory muscles improve sleep in ICU patients? *Respir Med* 2013;107:534–41.

- [2] Parthasarathy S, Tobin MJ. Effect of ventilator mode on sleep quality in critically ill patients. *Am J Respir Crit Care Med* 2002;166:1423–9.
- [3] Jounieaux V, Parreira VF, Aubert G, Dury M, Delguste P, Rodenstein DO. Effects of hypocapnic hyperventilation on the response to hypoxia in normal subjects receiving intermittent positive-pressure ventilation. *Chest* 2002;121:1141–8.
- [4] Toubanc B, Rose D, Glérant JC, François G, Mayeux I, Rodenstein D, Jounieaux V. Assist-control ventilation vs. low levels of pressure support ventilation on sleep quality in intubated ICU patients. *Intensive Care Med* 2007;33:1148–54.
- [5] Jabour ER, Rabil DM, Truitt JD, Rochester DF. Evaluation of a new weaning index based on ventilatory endurance and the efficiency of gas exchange. *Am Rev Respir Dis* 1991;144: 531–7.

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