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LETTER TO THE EDITORS

Deventilation syndrome in severe COPD patients during long-term noninvasive mechanical ventilation: poor sleep pattern, hyperinflation, or silent chronic muscular fatigue?

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Abbreviations

COPD	Chronic obstructive pulmonary disease
NIV	Noninvasive mechanical ventilation
PSG	Polysomnography
$TcPCO_2$	Transcutaneous CO_2 tension

Dear Editors:

Currently, nocturnal noninvasive mechanical ventilation (NIV) in chronic obstructive pulmonary disease (COPD) patients with severe impaired lung function may develop alterations in early morning period, commonly named deventilation syndrome. Still, this is an open new controversial problem in polysomnography studies. However, there are some complex pathways to understand all factors that could influence this phenomenon. The diagnosis and possible ways of monitoring and treatment of deventilation syndrome are complex, involving not only the parameters related to ventilation but also patient lung function parameters that are relevant in the stage and severity of the disease and basal hypercapnia levels. The suppression periods preceding the positive pressure syndrome "deventilation" may explain some mild symptoms and not well explained deterioration in some patients, which, in the beginning, are subtle symptoms. In part, these changes are related to changes in sleep pattern and positive pressure levels applied [1, 2].

In this issue, Adler et al. described how polysomnography studies may optimize noninvasive mechanical ventilation for optimizing positive pressure parameters and avoid "deventilation dyspnea" [3]. This is an interesting analysis of how to optimize the performance and comfort and to avoid patient-ventilator asynchrony in severe COPD.

However, there are some points that deserve to be taken into account for all practical implications in this study.

1. Although authors consider that the concept of deventilation dyspnea is complex, the physiological basis and whether these symptoms are associated with poor sleep pattern or chronic muscular fatigue after rapid cessation of positive pressure in ventilator-dependent patients are still unknown. We believe that this is an interesting aspect that could be assessed, and in this regard, the assessment muscle strength would be relevant to discriminate both pathways.
2. The authors determined that the alveolar hypoventilation is insufficient, and there is a residual hypercapnia. However, there is no clear explanation why levels of partial pressure of carbon dioxide (pCO_2) remain higher after night time. Additional, there is no completed interrelation regarding the interpretation of the absence of significant decline among transcutaneous CO_2 tension ($TcPCO_2$) levels and $PaCO_2$. This is important, especially in severe hypercapnic patients if long-term application, complications, and quality of life related with hypercapnia are to be considered.
3. It is relevant to try to decrease the pCO_2 to normal level with high pressure despite poor sleep quality. The authors reported that, in their center, they aim to obtain the best compromise between $PaCO_2$/$TcPCO_2$, induced leaks, and

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patient tolerance. They did not give the pH values of patients and take into consideration the pressure adjustment. They also declared that for all patients, initial settings of pressure support were as high as tolerated without polysomnography (PSG) study. Especially, the patients with severe COPD and residual hypercapnia may need a high pressure support at the beginning, but without PSG study, it may be difficult to find the right pressure for these patients with hyperinflation which may progress with high pressures. The pressures should be confirmed with PSG study at least with the latter. This problem in this extent may be special to their institution because of their strategy of beginning with high pressure support.

4. The authors focus their analysis and benefits in patients with hyperinflation treated with NIV but did not determine how they identified these patients and the condition. We consider that in these mechanisms, other mechanisms could be relevant: (a) pulmonary emphysema, (b) specific pulmonary functions test [4], or (c) mechanism associated with a high positive pressure during night periods [5]. In this study, there is lack of information regarding pulmonary function tests and how it differs from other deventilation symptoms in these patients. It is not clear if all patients analyzed are in the GOLD severe stage of COPD.
5. This is a short observation pilot study which avoided exploring more potential factors in this syndrome that influence, in a long period of time, changes in sleep breathing patterns, more complex change and instability, and higher level of patient-ventilator asynchrony index. In this sense, early polysomnography studies could be limited for long-term NIV implementation.
6. Addionally, there are some other key aspects to be taken into account: (a) Adler's report addresses issues of post-NIV-related dyspnea. In fact, these patients had a phenotype that was typified by a severe elevation of RV. The possible role of NIV in worsening air trapping and hyperinflation is a significant possible mechanism of dyspnea in these patients. Given that the intervention was successful when pulling back on ventilatory support, it is more likely that hyperinflation and not weakness is a significant cause of the post-NIV-related dyspnea. (b) Adler's report sheds light on some possible techniques. The need for surface EMG of the diaphragm, scoring asynchrony, technologist interventions to improve synchrony, etc., and (c) Adler's report highlights that the goals of NIV in COPD are unclear at best. I wish that they would have taken this opportunity to emphasize that Adler's report used settings other than PS to effect change. This is why CO_2 did not change. (d) Adler's report does not well characterize the COPD patients in their cohort.

Finally, what is the aim for long-term NIV for stable COPD patients to improve the blood gas or to improve the muscle strength and how should we monitor the pressure adjustment? In this study, we consider a more definition of high-risk population and prevalent mechanism, sleep breathing, positive pressure effect, or severe respiratory muscular fatigue-associated conditions in severe COPD patients. Further studies are welcome to answer all these questions and are encouraged to prepare the best strategy for the last mechanism.

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