



## High-flow nasal cannula oxygen therapy: P-SILI or not P-SILI?

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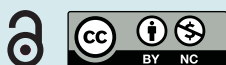
### From the authors:

We would like to thank A.S. Saini and co-workers for their interest and their valuable and constructive comments concerning our study [1]. We have shown that, assessed by pulmonary electrical impedance tomography (EIT), high-flow nasal cannula (HFNC) oxygen therapy and noninvasive ventilation (NIV) could generate comparable alveolar recruitment, but NIV generated larger lung volumes. This increase in lung volumes with NIV could be involved in alveolar lesions worsening, induced by the patient's spontaneous ventilation or "patient self-inflicted lung injury" (P-SILI) [2]. However, A.S. Saini and co-workers argue that HFNC could also generate P-SILI and describe the main pathophysiological determinants involved in this deleterious effect. If one can accept this potential risk with HFNC and share the arguments proposed by A.S. Saini and co-workers concerning its mechanisms, we nevertheless wish to make a few additional comments.

During hypoxaemic acute respiratory failure (ARF), compared to face mask at  $12 \text{ L}\cdot\text{min}^{-1}$ , HFNC set at  $40 \text{ L}\cdot\text{min}^{-1}$  has been shown not only to increase end-expiratory lung volumes (EELV) but also improve ventilation distribution and homogeneity, dynamic lung compliance and transpulmonary driving pressure [3]. A linear relationship has been shown between increased flow and EELV of dependent lung regions, whereas this relationship did not exist for non-dependent lung regions, without any evidence of increased tidal volume [4]. BASILE *et al.* [5], using HFNC at  $100 \text{ L}\cdot\text{min}^{-1}$ , have confirmed the correlation between flow and EELV as well as with the homogeneity of lung volume distribution. Although we did not perform an analysis at different flow rates of HFNC, our results are consistent with these studies and highlight an increase in EELV but not in tidal volume with HFNC [1, 3–5]. Flow with HFNC did not seem to be involved in the increase of tidal volume, directly implicated in P-SILI [6]. On the contrary, by increasing the EELV and the homogeneity of lung volume distribution, HFNC would be able to prevent P-SILI occurrence.

A.S. Saini and co-workers also focused on the importance of using high flow rates to reduce the inspiratory efforts (intrapleural pressure swings) during spontaneous ventilation and, therefore, the risk of P-SILI. HFNC was shown to be considered as a true ventilator "support" by reducing the inspiratory work of breathing (WOB) in patients with hypoxaemic ARF compared to face mask [3, 7]. Moreover, this beneficial effect on the variations of intrapleural pressure, respiratory rate and WOB seems to be correlated with the level of flow rate: maximum at  $60 \text{ L}\cdot\text{min}^{-1}$  but also observed from  $30 \text{ L}\cdot\text{min}^{-1}$  [4]. We chose a flow rate of  $50 \text{ L}\cdot\text{min}^{-1}$  as it is the maximum flow rate available on most intensive care unit ventilators, HFNC and NIV being delivered with the same ventilator in our study. Such a flow rate was also the one applied in the FLORALI trial, which first opened the debate on the potentially deleterious effects of NIV in hypoxaemic ARF [8]. HFNC using high flow rates such as  $50 \text{ L}\cdot\text{min}^{-1}$  could have, therefore, a protective effect against P-SILI by reducing intrapleural pressure variations.

We do agree with A.S. Saini and co-workers in considering that, during severe ARF, high flow rates with HFNC are necessary to overcome the patient's peak inspiratory flow and thus improve oxygenation by avoiding the phenomenon of inspiratory oxygen fraction ( $F_{\text{IO}_2}$ ) dilution. Better oxygenation with HFNC can also be explained by a lower metabolic cost of ventilation and an improvement in ventilation/perfusion ratios [7]. In our study, oxygenation was found to be lower with HFNC than with NIV but comparable to face mask. This could be partly explained by the flow rate used with HFNC but also by the fact that we used the pulse oxygen saturation ( $S_{\text{pO}_2}$ )/ $F_{\text{IO}_2}$  ratio and that  $F_{\text{IO}_2}$  was only estimated with a face mask. Nonetheless, the improvement in oxygenation with HFNC could contribute to reducing the risk of P-SILI by decreasing the stimulation of the respiratory drive.



### Shareable abstract (@ERSpublications)

The risk of P-SILI with HFNC is controversial and may be less than with NIV. Some physiological mechanisms of the protective effect of HFNC are hypoxaemia correction, reduction of inspiratory efforts and homogeneity of lung volume distribution. <https://bit.ly/3skOEKX>

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Regional variations of transpulmonary pressures leading to a heterogeneous distribution of tidal volumes, with a volume transfer from non-dependent to dependent regions, named the Pendelluft phenomenon, also occur in the pathophysiology of P-SILI [2, 9]. This phenomenon has been particularly described during invasive mechanical ventilation. To our knowledge, the Pendelluft phenomenon has been neither specifically evaluated during hypoxaemic ARF with HFNC nor compared with face masks or NIV. While Pendelluft detection and measurement methods are relatively complex, EIT could be a relatively simple and feasible bedside assessment technique for this phenomenon [9].

Finally, with comparable alveolar recruitment between HFNC and NIV for the settings used, our study highlighted with NIV, unlike with HFNC, a risk of pulmonary overdistension that could contribute to P-SILI [1]. However, our results cannot exclude that such a risk could also exist with HFNC. More studies are therefore necessary for a better knowledge about the risk of P-SILI with HFNC, and its determinants, but also to establish between NIV and HFNC the potentially most protective ventilation strategy during hypoxaemic ARF.

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