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Early View

Correspondence

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

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

The authors reply:

We would like to thank Saini *et al*. [1] for their interest and their valuable and constructive comments concerning our study [2]. We have showed that, assessed by pulmonary electrical impedance tomography (EIT), high flow nasal cannula oxygen therapy (HFNC) and non invasive ventilation (NIV) could generate comparable alveolar recruitment but larger lung volumes with NIV. 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) [3]. However, Saini *et al.* [1] 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 Saini *et al.* [1] concerning its mechanisms, we nevertheless wish to make a few additional comments.

During hypoxemic acute respiratory failure (ARF), compared to facemask at 12L/min, HFNC set at 40L/min 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 [4]. A linear relationship has been shown between increase 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 [5]. Basile et al. [6], using HFNC at 100L/min, 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 [2,4-6]. Flow with HFNC did not seem to be involved in the increase of tidal volume, directly implicated in P-SILI [7]. On the contrary, by increasing the EELV and the homogeneity of lung volume distribution, HFNC would be able to prevent P-SILI occurrence.

Saini et al. [1] 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 hypoxemic ARF compared to facemask [4,8]. 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 L/min but also observed from 30 L/min [5]. We chose a flow rate of 50L/min as the maximum flow rate available on most ICU 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 hypoxemic ARF [9]. HFNC using high flow rates such as 50L/min could have, therefore, a protective effect against P-SILI by reducing intrapleural pressure variations.

We do agree with Saini et al. [1] 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 FiO_2 dilution. Better oxygenation with HFNC can also be explained by a lower metabolic cost of ventilation and an improvement in ventilation/perfusion ratios [8]. In our study, oxygenation was found to be lower with HFNC than with NIV but comparable to facemask. This could be partly explained by the flow rate used with HFNC but also by the fact that we used the SpO_2/FiO_2 ratio and that FiO_2 was only estimated with a facemask. Nonetheless, the improvement in oxygenation with HFNC could contribute to reduce the risk of P-SILI by decreasing the stimulation of the respiratory drive.

Regional variations of transpulmonary pressures lead to a heterogeneous distribution of tidal volumes, with a volume transfer from non-dependent to dependent regions, named Pendelluft phenomenon, also occur in the pathophysiology of P-SILI [3,10]. This phenomenon has been particularly described during invasive mechanical ventilation. To our knowledge, the Pendelluft phenomenon has been neither specifically evaluated during hypoxemic ARF with HFNC nor compared with facemasks or NIV. While Pendelluft detection and measurement methods are relatively complex, electrical impedance tomography could be a relatively simple and feasible bedside assessment technique for this phenomenon [10].

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 [2]. However, our results cannot exclude that such a risk could not 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 non-invasive ventilation strategy during hypoxemic ARF.

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