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Volutrauma, atelectrauma and mechanical power: What really matters is intensity!

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To the Editor

We thank Dr. Tonetti and colleagues for their interest (1) in our recently published study in *Critical Care Medicine*, where we showed that the specific uptake rate of 18F-fluorodeoxyglucose, a marker of lung inflammation, was higher during volutrauma than atelectrauma in pigs with experimental acute respiratory distress syndrome (ARDS) (2).

In recent years, the importance of dynamic stress as a major mechanism of ventilator-induced lung injury (VILI) has gained much attention. During mechanical ventilation, dynamic stress in the lungs, approximated as the driving pressure of the respiratory system, is associated with increased mortality in ARDS patients (3) and with development of postoperative pulmonary complications in surgical patients (4). Although this knowledge

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about driving pressure represented an important advance in our understanding of VILI, we believe that the importance of static stress is currently underestimated. Our study (2) was intended to call attention to static stress as an additional mechanism of VILI.

The concept that transfer of mechanical power from the ventilator to the respiratory system might be a determinant of VILI is exciting. To our knowledge, this concept was first proposed in 2010 by Guttmann (5), although a possible association between mechanical power during spontaneous breathing and the development of bronchopulmonary dysplasia was addressed as early as 1988 (6). In 2016, Cressoni et al. (7) and Gattinoni et al. (8) contributed with formal calculations of mechanical power and association with VILI. In addition, our group introduced the term “intensity”, which, in this context, represents the normalization of mechanical power to the lung surface area or tissue mass (9). Certainly, for a given mechanical power, intensity is higher in smaller surface areas, as well as at the interface between lung zones with different mechanical properties, as proposed in 1970 (10).

We agree with Dr. Tonetti and colleagues that mechanical power differed between groups in our study (2). However, when calculating the intensity, i.e., normalizing the mechanical power (estimated by the authors) to lung tissue, differences between volutrauma and atelectrauma were negligible (0.064 vs. 0.066 J/min/g, respectively). Thus, we disagree that keeping the mechanical power comparable among groups by tailoring its components would have been helpful. Furthermore, in non-homogeneous lungs, volutrauma is more likely to occur in regions where static pressures are higher, whereas atelectrauma occurs preferentially in regions with lower static pressures. Since our study was designed as a proof-of-concept of the role of static stress in VILI, the model chosen had to reproduce the conditions under which these phenomena occur.

In our opinion, it is too early to make recommendations in terms of safety thresholds for mechanical power and/or intensity. Nevertheless, it is conceivable that, pending on confirmatory research, mechanical ventilators might display the mechanical power transferred to the lungs in the future. Even better, they might also normalize the mechanical power to surrogates of lung surface or tissue mass to display what really matters: intensity.

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