Xue et al (1) highlight that when comparing laryngoscopy devices, accounting for the experience of the operator performing intubation with each laryngoscopy device is critical. In the Facilitating EndotracheaL intubation by Laryngoscopy technique and apneic Oxygenation Within the intensive care unit (FELLOW) trial (2), we collected and adjusted for each operator's previous intubating experience overall and previous intubating experience with the specific laryngoscopy device at the time of each intubation. This dynamic variable (overall and device-specific experience evolving over the course of the trial) is unique to the FELLOW trial and provides a granular account of operator's device-specific experience than any previous trial of endotracheal intubation.

Xue et al (1) also raise the relationship between operators' intubating experience and "proficiency." Although intubating proficiency (defined variably) may occur after around 50 intubations in the operating room, the experience required to achieve proficiency during intubation of critically ill adults is unknown. Over the wide range of prior experience in our trial, we did not find that increasing device and procedure experience modified the effect of VL on first pass success. Most importantly for a trial comparing VL to direct laryngoscopy (DL), there was no signal in favor of VL among those with more experience, with the point estimate for first pass success actually favoring DL among those with greater than 50 prior intubations.

Xue et al (1) raise the point of the current trial not mandating the shape of the intubating stylet. In the ICU where the trial occurred, usual care is a straight-to-cuff stylet shape; however, since there are no definitive studies on how to shape a stylet we chose not to protocolize this technique.

Finally, Xue et al (1) point out that the reason for failure to intubate on the first laryngoscopy attempt was not systematically collected in the FELLOW trial. We would refer the reader to recent data from another trial of VL (3) suggesting that failed first attempts with VL are most commonly due to poor camera image and inability to pass an endotracheal tube into the trachea.

We performed a pragmatic trial to test the effectiveness of VL in an ICU with a heterogeneous patient and operator population while not mandating any other procedural aspects so that we can learn how VL performs in real world rather than highly controlled scenarios. Our data only indirectly speak to how VL would perform if all operators were very experienced with each device, if certain neuromuscular blocking drugs were used, and the shape of stylets were protocolized. Fortunately, we have other recently published randomized trials of VL in critically ill adults showing that our result was not likely a result of our study design. The findings are the same across the board: routine use of VL during endotracheal intubation of critically ill adults improves laryngeal visualization but does not increase procedural success or decrease complications (3–5).

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Volutrauma, Atelectrauma, and Mechanical Power

To the Editor:

We read with great interest the article in a recent issue of *Critical Care Medicine* by Güldner et al (1), which showed a different inflammation extent and distribution (measured as [^{18}F]fluorodeoxyglucose uptake rate) in two experimental models of ventilator induced lung injury (VILI): volutrauma and atelectrauma.

The authors found that volutrauma yielded higher inflammation than atelectrauma. In addition, the control lung (no VILI) and the atelectrauma lung (VILI) showed a similar degree of inflammation. The authors suggest that static stress and strain, because of positive end-expiratory pressure

(PEEP), are major determinants of VILI because tidal volumes were equally low in both groups.

We recently proposed a new way of looking at the ventilator side of VILI: the mechanical power (2). According to this approach, every component of the mechanical ventilation already known to be causative of VILI (tidal volume, driving pressure, respiratory rate, and flow) plus the PEEP (which has been so far considered mostly protective) contribute, each one at a different extent, to the mechanical power delivered to the respiratory system (and to the lung). Starting from the classical equation of motion, we developed an equation that enables to calculate the mechanical power by some easily obtainable ventilator variables (2).

Using this equation and the average values provided by Güldner et al (1), we computed the mechanical power delivered to the respiratory system in the two groups of pigs. In the volutrauma group, the average mechanical power to the respiratory system was 17.12 J/min, a value more than double than the one computed in the atelectrauma group (7.13 J/min). These are obviously rough estimates as the mechanical power should be better expressed using the transpulmonary pressure, normalizing it to the lung volume (only one lung was used in the experiments by Güldner et al (1)) and taking into account its possible maldistribution (stress raisers) (3).

Despite these limitations, it is still impressive to see the difference in terms of power between the two groups. In a previous study, in pigs slightly smaller than the ones used by Güldner et al (1), we found a transpulmonary power threshold for VILI of 12.1 J/min (4) (corresponding to 16.7 J/min to the respiratory system). Therefore, we can hypothesize that the pigs in the atelectrauma group were under the VILI threshold, whereas the ones in the volutrauma group were above it. The role of PEEP (averaging 32.4 cm H_2O in the volutrauma group and just 0.8 cm H_2O in the atelectrauma) in determining VILI seems to emerge very clearly in the data provided by Güldner et al (1), being the most important determinant of the differences in mechanical power between the two groups (same tidal volume and respiratory rate).

Accordingly, in our opinion, possible differences between VILI induced by cyclic opening and closing of lung tissue (if any) or by tidal overstretching should be investigated under the same "ergotrauma" conditions (5), that is, keeping the mechanical power equal in the two experimental groups by appropriate tailoring of its components (tidal volume, respiratory rate, driving pressure, flow, and PEEP).

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The authors reply:

We thank Tonetti et al (1) for their interest 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 during 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 about dynamic stress 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 its 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 as early as 1970 (10).

We agree with Tonetti et al (1) that mechanical power differed between groups in our study (2). However, when calculating the intensity, that is, 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