Understanding the Mysteries of Mechanical Power

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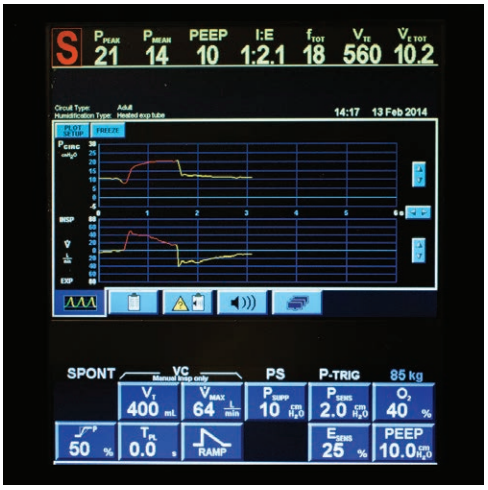
In the current issue, Vassali *et al.* compared the individual effects of high tidal volume (V_T), respiratory rate (RR), and positive end-expiratory pressure (PEEP), each delivered at two levels of mechanical power (15 and 30 J/min), on lung mechanics, hemodynamics, gas exchange, and pulmonary morphology in healthy piglets under prone positioning.[1] The study showed that different ventilatory strategies delivered at iso-mechanical power led to similar lung injury. After their theoretical study in which the contribution of different components of power was evaluated,[2] the authors also provided preclinical data on the contribution of V_T, RR, and PEEP to lung damage.



"Both static parameters … and dynamic ones … have been implicated in the pathophysiology of ventilator-induced lung injury."

Both static parameters—V_T, PEEP, respiratory system plateau pressure, and driving pressure—and dynamic ones—RR, inspiratory and expiratory airflow—have been implicated in the pathophysiology of ventilator-induced lung injury.[3] The knowledge that both static and dynamic respiratory variables may be injurious has led to the concept of mechanical power, defined as the product of the total inflation energy and respiratory rate. Several points should be discussed based on the authors' findings. In healthy lungs, V_T, RR, and PEEP must undergo substantial change to induce lung damage, contrasting with several preclinical and clinical studies in acute respiratory distress syndrome—a context in which even minor changes in V_T, RR, or PEEP are known to enhance lung injury.[4]

Interestingly, when considering two groups at similar low power (15 J/min), the first with high V_T (~33 ml/kg), respiratory system plateau pressure = 34 cm H_2O and driving pressure = 29 cm H_2O and the second with high RR (40 bpm), but low static parameters (respiratory system plateau pressure = 17 cm H_2O and driving pressure = 9 cm H_2O), the same degree of lung lesion was observed after 48 h of mechanical ventilation. Based on the current literature,[5] high respiratory system plateau pressure and driving pressure lead to more deleterious effects when compared to low respiratory system plateau pressure and driving pressure. Therefore, we may hypothesize that, even though increased RR results in lower respiratory system plateau pressure and driving pressure, the repetitive stimuli of higher RR in a specific damaged area can further injure the extracellular matrix, thus leading to proliferation of lung lesions over time during mechanical ventilation. In short, we must highlight that not only the level of a certain variable but also the duration of parenchymal exposure to it may cause further pulmonary injury. In a separate study, the same amount of mechanical power imparted to a severely injured lung resulted in more biologic impact than in healthy lungs, suggesting that rather than only power, we must also compute and consider the *intensity* (power per unit of lung area).[6]

When comparing low *versus* high mechanical power (15 *vs.* 30 J/min, respectively), careful attention should be paid to lung morphology, since iso-mechanical power was shown to result in a similar degree of lung damage regardless of whether the cause was high V_T, RR, or PEEP. This could be attributed to two factors: first, the study was performed in healthy animals; second, 15 J/min may already be high enough to cause lung damage, after which no significant changes were observed. According to the authors, low and high mechanical power levels were used to confirm the results of a previous study, in which 25 J/min during 48 h discriminated between lower and higher degrees of lung damage. However, values below 25 J/min are already associated with severe lung damage, as observed in a previous study in which ventilator-induced lung injury developed when mechanical power exceeded 12 J/min.[7] Therefore, further experiments should be performed at lower levels

Image: J. P. Rathmell.

This editorial accompanies the article on p. 1126.

Accepted for publication January 22, 2020. Published online first on March 2, 2020. From the Laboratory of Pulmonary Investigation, Carlos Chagas Filho Institute of Biophysics, Federal University of Rio de Janeiro, Brazil (P.L.S., P.R.M.R.); the Department of Surgical Sciences and Integrated Diagnostics, University of Genoa, Genoa, Italy (P.P.); and the San Martino Policlinico Hospital, Scientific Institute for Research, Hospitalization and Healthcare for Oncology and Neurosciences, Genoa, Italy (P.P.).

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<zdoi;. DOI: 10.1097/ALN.0000000000003222>

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of mechanical power (less than 15 J/min) to better clarify whether iso-mechanical power with different V_T, RR, and PEEP levels indeed leads to similar lung damage.

Another issue under debate is the presentation of lung histologic data. When presenting lung-mechanics data, the authors reported low and high mechanical power groups separated by high V_T, RR, and PEEP within each mechanical power level. In contrast, the results of lung histologic analyses were presented stratified by low *versus* high mechanical power (with pooling of histologic data at high V_T, RR, and PEEP) and by ventilatory strategy (with pooling of histologic data at low *vs.* high mechanical power). Therefore, we are unable to clearly differentiate the effect of each ventilatory strategy at a given mechanical power, since the impact of these variables on lung extracellular matrix could result in different types of lung injury.[8] One main example is the high PEEP groups, as acknowledged by the authors.

To achieve equivalent high mechanical power with comparable V_T and RR, PEEP was increased to very high levels. Comparable values have been used in obese patients with acute respiratory distress syndrome,[9] but these were protectively ventilated. The high PEEP group, regardless of mechanical power, received greater fluid volumes and higher vasopressor doses than the other groups, leading to a high vascular pressure gradient and vascular flow, which may have skewed the hemodynamic balance toward ventilator-induced lung injury. Unfortunately, the authors did not analyze specific markers of alveolar-capillary membrane permeability. Higher PEEP recruits the lungs but derecruits the capillaries, increasing the pulmonary artery pressure. To minimize the increase in pulmonary artery pressure, PEEP should be kept to the minimum necessary to maintain satisfactory oxygenation while avoiding excessive atelectasis or increases in volemia.

The findings of Vassali *et al.* are an important step toward comparing key ventilatory variables at equivalent mechanical power levels. Mechanical power may be computed and displayed by future mechanical ventilators, and the finding of Vassali *et al.* suggests that, at very high mechanical power, the difference between static and dynamic parameters is of relatively little consequence. Additional preclinical and clinical studies should be performed in healthy and diseased animals at much lower mechanical power before considering this parameter as a reference variable for use at bedside rather than other already standard parameters, such as V_T, RR, and PEEP. Indeed, special attention should be paid to PEEP levels and hemodynamic effects. Compared to V_T and RR, PEEP at iso-mechanical power was associated with profound hemodynamic changes that may tilt the balance in favor of pulmonary vascular damage.

Competing Interests

The authors are not supported by, nor maintain any financial interest in, any commercial activity that may be associated with the topic of this article.

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