Estimation of the Mechanical Power of Ventilation at the Bedside to Lessen Ventilator-Induced Lung Injury

The conjunction of adverse pulmonary consequences of mechanical ventilation has been termed ventilator-induced lung injury (VILI), and its presence is associated with worse outcomes.[1,2] After decades of research and clinical observations, a lung-protective ventilation strategy is now proposed as a standard of care to decrease VILI and improve survival in patients with ARDS. This protective ventilation strategy includes the use of low tidal volumes (V_T) of 6 mL/kg predicted body weight and maintaining the inspiratory plateau pressure $<$ 28–30 cm H_2O.[3-5] More recently it has been proposed that even more important than V_T and plateau pressure is the distending pressure of the respiratory system (ie, driving pressure).[6] Others have proposed that the critical determinants of VILI may be lung stress and strain.[7]

As an attempt to reconcile these different perspectives, a unifying physical concept has been proposed—mechanical power (MP)—that would allow understanding the contribution of the different variables potentially involved in VILI in terms of their relative impact on energy transfer to the lungs over time. This energy dissipation within the lungs may lead to inflammation and harmful deformation of lung parenchyma.[8]

In physical terms, the energy transferred per breath corresponds to the product of the change in volume times the change in pressure, and MP corresponds to the product of the energy transferred per breath times the breathing frequency. As the direct measurement of MP is rather complex, an equation based on the equation motion of the respiratory system has been proposed to estimate it[9]:

$$\text{Power}_{rs} = \text{RR} \cdot \left\{ \Delta V^2 \cdot \left[\frac{1}{2} \cdot \text{EL}_{rs} + \text{RR} \cdot \frac{(1 + I:E)}{60 \cdot I:E} \cdot R_{aw} \right] + \Delta V \cdot \text{PEEP} \right\}$$

Although the equation looks complex, it reflects the 3 components of inspiratory airway pressure: flow resistance, respiratory system elastance, and the end-expiratory pressure, multiplied by the breathing frequency and by the tidal volume. Thus, the equation includes some variables that have been consistently associated to VILI, such as V_T and driving pressure, but others for which the relation with VILI is unclear, such as breathing frequency and PEEP.

SEE THE ORIGINAL STUDY ON PAGE 177

Clinical studies have shown that high MP of ventilation is independently associated with higher in-hospital mortality and several other negative outcomes.[10] In experimental studies, a lung MP threshold of about 12–13 J/min and respiratory MP of 25 J/min have been associated to increased lung damage.[8,11]

The concept of having a unified measurement to evaluate VILI risk appeared promising both from a clinical point of view, by allowing us to summarize the risk of VILI in a single variable, and from a conceptual point of view, by indicating that the impact of any respiratory variable on VILI ultimately depended on a single factor, energy. This notion suggested that as long as total energy remains constant variations in specific ventilatory variables should not modify the risk of VILI.

Five years after MP emerged as a novel concept to understand VILI, several criticisms have been raised: The size of the lung into which energy is dissipated is not considered; no injurious threshold has been identified for humans; the resistive energy dissipated into the airways or the elastic energy dissipated into the thoracic cage is included in the equation; the inclusion of PEEP and its linear relation with MP is disputed; and finally, the different components of MP are considered equivalent, which is not consistent with the evidence accumulated to date. To address some of these limitations, Marini and Rocco[12] recently proposed that MP may be separated into 3 major pressure components: flow resistive pressure, tidal elastic (driving) pressure, and the static elastic pressure baseline set by PEEP. In addition, they proposed that a pressure threshold should be included in the analysis, further dividing MP into 7 subcomponents to identify the more

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hazardous fraction of MP (ie, damaging power model). These subcomponents could be calculated through simple mathematical equations.[13]

In this issue, Tawfik et al[14] assessed the accuracy of these mathematical equations to estimate different types of ventilation power using input variables that could be obtained at the bedside. The authors used a simulated physical one-compartment model of the respiratory system to test and validate 7 equations derived from premeasured resistance and compliance (ie, "predicted" equations) and 7 other equations derived from observed output values for peak airway and plateau pressure at the end of insufflation (ie, "observed" equations) as previously described.[12] All these 14 equations were compared against planimetry as the accepted standard measurement to quantify energy (ie, area under the pressure-volume loop) under a variety of ventilatory combinations including constant and decelerating flow and different values of resistance, tidal volume, breathing frequency, and PEEP.[14]

In general, all equations (predicted and observed) demonstrated excellent accuracy and strong correlation with the planimetry calculations. Only a small proportion (0.5%) of total MP estimations was underestimated in $>$ 5 J/min as compared with planimetry, suggesting excellent reliability. These deviations from planimetry energy estimation occurred in "predicted" equations when high frequency rate was simulated and theoretical *set* PEEP rather that total PEEP observed was computed. Besides, as intuitively expected, estimation of ventilation power during decelerating flow presented greater variability than constant flow mode since equations were originally designed for the latter. Notwithstanding, decelerating flow equations showed an acceptable and sufficient accuracy.

This exhaustive simulation work invites a couple of comments. As a bench study, it does not necessarily reflect exactly some biological conditions observed in clinical practice such as tidal recruitment, bronchoconstriction, and presence of secretions in the airway, factors that could modify compliance and airway resistance and subsequently power estimation. In addition, the presence of partial or total airway closure specially in ARDS and obese patients would affect observed MP estimations and, consequently, predicted equations may not be accurate enough under these circumstances.[15] Thus, a cautious interpretation is advised, and future clinical validation of these equations is needed.

The study by Tawfik et al[15] used different equations to subdivide the total inflation energy into several components based on an assumed injurious pressure threshold. This proposed model of damaging power is appealing and assumes that not all inflation energy has the potential to inflict damage on the lungs. Thus, quantitative partitioning of insufflation energy could help to better understand VILI development; and therefore, lung-protective ventilation strategies might be implemented in the future. Interestingly, computation of different types of ventilation power could be estimated using ventilatory parameters easily obtained at the bedside.[12,13] Of note, the concept of an injurious threshold for damaging power remains hypothetical, and its interpretation is limited. Consequently, the estimation of different types of MP based on an arbitrary threshold constitutes a theoretical exercise of a potentially applicable concept that cannot be immediately transferred into clinical practice. Current data are insufficient to propose a rational threshold, and further research is needed to understand its utility. Several factors may influence the hypothetical damaging threshold including positioning and the severity of lung injury. It has been well recognized that lung inhomogeneities causing heterogeneous ventilation may facilitate further lung injury in patient with ARDS since local stress/strain are key determinants of regional VILI.[16,17]

Finally, there is still debate regarding the potential applicability of MP as a unifying concept to predict the risk of VILI when compared with single ventilatory measurements (ie, driving pressure and breathing frequency).[18] The present study may be a relevant contribution to improve the analysis of MP for research. However, decomposing MP into multiple variables runs the risk of losing the main appeal of MP, which is being a unifying variable, and at the same time adding even more complexity to its clinical applicability. It remains to be elucidated whether this proposal to decompose MP will constitute a step forward or a step backward.

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