Monitoring Spontaneous Effort During Mechanical Ventilation: Are Our Tools Good Enough?

After decades of experimental and clinical research on lung injury during mechanical ventilation, cyclic overdistention caused by high transpulmonary pressures imposed onto the lungs stands out as the primary pathophysiological mechanisms of ventilator-induced lung injury.[1-3] Whether the excessive distending pressure is positive or negative, alveolar damage seems to follow, nonetheless.[4]

Physiological consequences of inspiratory negative swings in pleural pressure during strong spontaneous efforts have been studied in depth experimentally. In those studies, lung injury was consistently demonstrated to occur due to vigorous inspiratory efforts,[5-7] especially when applied to inflamed lungs. In this scenario, in which lungs are more prone to further damage, there can be an uneven distribution of negative pleural pressures leading to regional overdistention.[6,7] Recently, this lung injury caused by strong spontaneous efforts has been termed patient self-inflicted lung injury (P-SILI).[8]

However, the benefit to risk of a strategy to minimize P-SILI has not yet been demonstrated in prospective clinical trials. Since most available interventions to prevent P-SILI encompass deep sedation and eventually neuromuscular blockade, concerns have emerged that such a strategy could elicit the occurrence of prolonged mechanical ventilation with its unfavorable consequences such as ventilator-associated pneumonia and ventilator-induced diaphragmatic dysfunction,[9,10] which have been associated with worse clinical outcomes.[11,12]

To better understand the impact of vigorous spontaneous efforts during mechanical ventilation and even to better design feasible clinical trials regarding this matter, noninvasive methods to accurately monitor spontaneous breathing are needed. Recently, some maneuvers have been proposed as surrogates of inspiratory muscle effort or pressure (P_{mus}). They include (1) $P_{0.1}$, an estimate of respiratory drive measured as the airway pressure drop during a 100-ms end-expiratory pause; (2) increase in airway pressure during a prolonged end-inspiratory occlusion maneuver; or (3) decrease in airway pressure during a prolonged end-expiratory pause (ΔP_{occ}).[13-15]

The authors have disclosed no conflicts of interest.

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DOI: 10.4187/respcare.09567

In this issue of the Journal, Kallet et al[16] describe a relatively brief (1-s) end-expiratory pause maneuver (EPM) to estimate P_{mus} simulated in bench tests. They found that the airway pressure drop (ΔP_{aw}) during the EPM had reasonable accuracy and reproducibility across multiple operators

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to estimate the different P_{mus} generated with the ASL 5000 simulator (IngMar, Pittsburgh, Pennsylvania), with almost 95% of the 2,412 P_{mus} estimations within the predefined agreement criteria of 2 cm H_2O. Bias between ΔP_{aw} and P_{mus} pointed toward underestimation across all tested modes and ventilators from different manufacturers, with a larger error when simulated P_{mus} was more extreme.

Different from the previously described end-expiratory occlusion maneuver (ΔP_{occ}), defined as the maximal deflection

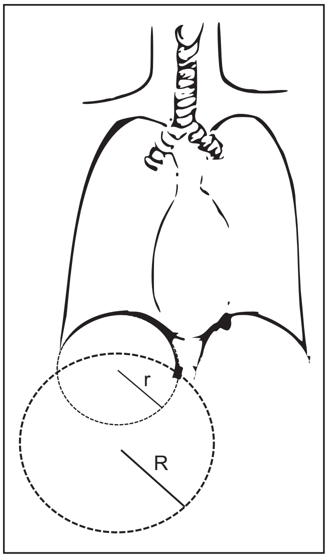

Fig. 1. Theoretical model of the respiratory system. With the diaphragm descent during inspiration, there is an increase in its curvature radius. Assuming (1) that each hemidiaphragm is roughly a hemisphere with radius r at the functional residual capacity and (2) that there is a fixed partitioning of the tidal volume between the rib cage and the diaphragm, it is possible to compute the curvature of the diaphragm (with radius R) for each lung volume using simple laws of geometry. One can then apply Laplace law to draw conclusions about the relative efficiency of the diaphragmatic contraction at different lung volumes.

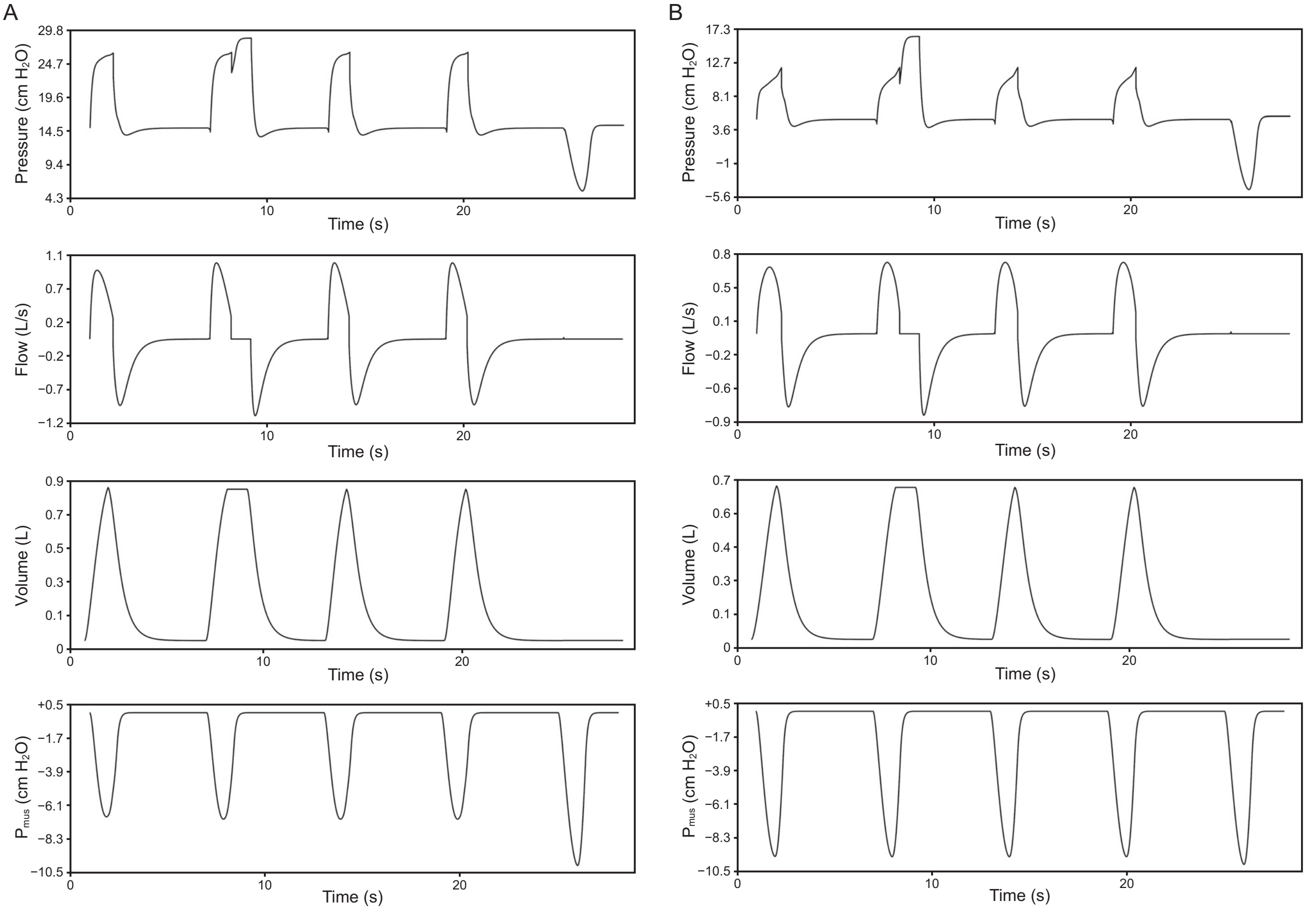

Fig. 2. Waveforms of pressure-support ventilation showing maneuvers to estimate muscle pressure (P_{mus}). Plateau pressure after an end-inspiratory occlusion (second cycle) and airway pressure drop during an end-expiratory pause (ΔP_{occ}, fifth cycle) are shown. Panel A shows these maneuvers with 15 cm H_2O of PEEP and panel B with 5 cm H_2O of PEEP. Note that plateau pressure underestimates and ΔP_{occ} overestimates the breadth of P_{mus} of an unobstructed breath. According to the predictions, the bias between ΔP_{occ} and P_{mus} is greater with higher PEEP levels. Waveforms were generated from a mechanical ventilator numerical simulator developed by the authors using R statistical software. Parameters used for the simulations were compliance of 60 mL/cm H_2O, airway resistance of 10 cm $H_2O \times$ s/L, inspiratory effort of 10 cm H_2O, and diaphragm radius at the functional residual capacity of 7.5 cm.

in airway pressure during a *complete* occluded breath, Kallet et al[16] proposed a brief end-expiratory occlusion lasting 1 s. This shorter pause has the theoretical advantage of being less prone to the influence of conscious reactions against the occluded breath, similar to the rationale underlying $P_{0.1}$ measurements. It is important to consider, however, that in their original description the prolonged ΔP_{occ} maneuvers did not significantly alter respiratory drive, as measured by the electrical activity of the diaphragm.[13] Notwithstanding, this finding pertains to the final analysis after the exclusion of 36% of airway recordings and requires confirmation. The briefer method proposed by Kallet et al,[16] which limited end-expiratory occlusions to 1 s, could possibly still capture maximal inspiratory effort in a fraction of patients, with the potential aforementioned advantage of a lesser impact on respiratory drive, especially in those patients with lighter sedation. The sensitivity of the maneuver to different durations of inspiratory effort, however, was not assessed given only one duration was simulated (0.85 s). P_{mus} could be further underestimated in more prolonged efforts.

One characteristic of EPM is systematic overestimation of P_{mus} due to greater diaphragm-contracting force during an occluded (isovolumetric) breath when compared to an unoccluded breath with abdominal wall displacement.[17,18] This overestimation was found in the publication from Bertoni et al[15] to be by approximately a third on average. As a result, the authors proposed that predicted P_{mus} be calculated as $0.75 \times \Delta P_{occ}$ to take into account the average bias. Unfortunately, this impact of the lung inflation on the neuromechanical coupling is very difficult to simulate in bench studies.

We illustrated the concept of neuromechanical coupling using a simple theoretical model of the respiratory system (Fig. 1). Based on a few reasonable assumptions, ΔP_{occ}

measurement bias should depend on end-expiratory lung volume according to Laplace law ($P = 2T/r$), where T is the tension generated by the diaphragmatic contraction and r is the diaphragm curvature radius. Neuromechanical efficiency is highest at lung volumes closer to the functional residual capacity and diminishes progressively as lungs inflate toward their total lung capacity (eg, with higher PEEP). This loss of neuromechanical efficiency throughout inspiration would only take place during unoccluded breaths during which there is diaphragmatic displacement and a change in its radius, leading to a bias between ΔP_{occ} and P_{mus} measurements. ΔP_{occ} could, therefore, point toward overestimation of P_{mus} especially with higher levels of PEEP, which, as compared to lower PEEPs, are associated with larger changes in the diaphragm radius for a given tidal volume (Fig. 2).

Another previously described surrogate for P_{mus} is plateau pressure after a brief end-inspiratory pause maneuver. During this pause, inspiratory muscles relax against closed inspiratory and expiratory valves, thus producing an increase in airway pressure, which reflects P_{mus}. There are important differences between P_{mus} estimates with end-inspiratory and end-expiratory pauses. First, a portion of the inspiratory effort is spent against resistive forces (Fig. 2), which, at end-inspiration, are usually much smaller.[19] Second, the end-inspiratory pause is insensitive to expiratory muscle activity at the end of expiration, whereas the EPM will capture both, expiratory (relaxing) and inspiratory (contracting) muscle pressure. As a result, the actual inspiratory muscle pressure must be somewhere in between these 2 estimates, with end-inspiratory occlusion maneuvers providing a lower boundary and ΔP_{occ} an upper boundary.

Kallet and colleagues[16] have provided us with a new method to monitor spontaneous effort during mechanical ventilation, especially during the challenging period of transition from fully-controlled mechanical ventilation to partial support. Under the simulated conditions studied, their method of measurement had satisfactory accuracy to estimate P_{mus}. Validity of this briefer occlusion maneuver should now be tested in vivo with naturally varying durations and intensities of inspiratory efforts and under the influence of diaphragmatic neuromechanical coupling.

As Immanuel Kant[20] stated in his *Critique of Pure Reason,* "as travelers in the pursuit of truth, surrounded by a broad and stormy ocean, it is prudent to first cast another glance at the map of the land we are yet to explore." Similarly, before venturing into clinical trials to test interventions to possibly reduce P-SILI, we should first decide whether our tools and spectacles to search for its presence are satisfactory.

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