I read with interest Dr. Weingart's primer on the initial management of the ventilated patient in the emergency department.[1] I appreciated the simplicity of his approach regarding ventilator modes and gas exchange. However, I am concerned his approach to alveolar protection sacrifices new data and nuances of thoracic compartment physiology for the simplicity of using plateau pressures (P_{pl}) and PEEP-FiO_2 tables to guide management.

Since the ARMA trial,[2] emphasis has been placed on P_{pl} generated from the interaction of tidal volume and lung compliance to minimize dynamic strain on the lungs. However, this approach largely ignores the potential for altering lung compliance, which is related to the size of the lung and the degree of atelectasis, and the contribution of the chest wall, which limits alveolar distention and can result in high P_{pl} without alveolar overdistention. From the perspective of the Emergency Department, abnormalities in gas exchange are met with empiric changes in PEEP without knowledge of whether alveoli are being recruited or overdistended, or if there is contribution from a noncompliant chest or abdominal wall. How then can we monitor for alveolar overdistention, but also determine whether increases in lung volume from recruitment might improve lung compliance and thus reduce ventilator induced lung injury (VILI)?

In 2015, Amato et al published compelling observational data identifying driving pressure as the best predictor of VILI, with a driving pressure of 14cmH_2O associated with an increase in mortality.[3] Driving pressure is defined as the difference between the P_{pl} and PEEP, and functionally normalizes tidal volume to lung size. In the study, increasing P_{pl} predicted increased mortality only when associated with an increase in driving pressure. Likewise, they found that increased PEEP was associated with worsening mortality if driving pressure also increased, and that mortality decreased if driving pressure decreased because of alveolar

recruitment. Because the concept of driving pressure encompasses more aspects of pulmonary physiology than P_{pl}, and because it is simple to measure, titrating the ventilator based on this parameter rather than P_{pl} would be a more precise way of managing ventilated patients.

Dr. Weingart also recommends 8mL/kg of tidal volume for newly intubated patients. Although the ARDSnet study studied a 6-8mL/kg tidal volume, lower tidal volumes produce lower dynamic strain. Indeed, some endorse using ultra-protective tidal volumes of 4mL/kg or less in patients with ARDS. One study demonstrated an 18% increase in mortality for every 1mL/kg over 6mL/kg of tidal volume,[4] while another study suggested that using lower tidal volumes (6 vs. 10ml/kg) can actually prevent the progression to ARDS in high risk patients.[5] The data for lower tidal volume in non-ARDS patients is less clear, particularly between 6 and 8mL/kg, but physiologically, 6mL/kg is an adequate, physiologic tidal volume with intrinsically less risk for VILI than 8mL/kg.

Finally, it should be emphasized that visual inspection of flow-time curves to detect auto-PEEP, although often helpful, have not been definitively evaluated and anecdotally, false positives and negatives regularly occur, particularly in severe obstructive lung disease or in tachypnic patients.

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