Editorial

Mechanical ventilation in acute respiratory distress syndrome at ATS 2016: the search for a patient-specific strategy

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Acute respiratory distress syndrome (ARDS) was first defined by Ashbaugh *et al.* in 1967 (1). They described 12 patients who developed the acute onset of hypoxemic respiratory failure, diffuse bilateral alveolar infiltrates, and low respiratory system compliance brought on by a variety of different insults. Decades of dedicated research have followed this initial description, yet ARDS remains a common critical illness with an exceptionally high mortality rate of 35–46% (2). At the 2016 American Thoracic Society (ATS) International Meeting, Dr. Brian Kavanagh, a Professor of Anesthesia from the University of Toronto, delivered a highly popular keynote speech addressing the role professional societies play in promoting universal management guidelines. He made several important points about the challenges and possible downsides to this strategy. This article reviews the potential for a more patient-specific approach to ARDS care based on presentations at the ATS meeting and the recent literature.

Recent breakthroughs in ARDS management have been attained by minimizing lung injury due to stress and strain from mechanical ventilation. Ventilator-induced lung injury (VILI) contributes to a systemic inflammatory response, termed "biotrauma", which propagates not only further lung injury but also extra-pulmonary organ injury, contributing to multiorgan failure and risk of death (3). In 2000, the National Heart Lung and Blood Institute (NHLBI) ARDS Network published a milestone study showing that a lung-protective strategy targeting plateau pressures <30 cmH_2O and low tidal volumes of 6 mL/kg, compared to 12 mL/kg, decreased mortality and duration of both respiratory and extrapulmonary organ failure in patients with ARDS (4). Based on this publication, multiple professional societies have endorsed 6 mL/kg ideal body weight as the optimal tidal volume for all patients with ARDS, and this practice guideline has been widely disseminated. However, many believe we can do better than a "one size fits all" approach (5-7).

The initial enthusiasm for low tidal volume ventilation began in the 1980s, largely via work by Gattinoni *et al.* (8). They used computed tomography images to show that patients with ARDS have a reduced aerated lung volume due to the development of dense regions of dependent atelectasis. This concept, referred to as the "baby lung", continues to provide the basis for low tidal volume ventilation: lower tidal volumes are necessary to prevent overdistension in ARDS because the aerated "baby lung" volume itself is reduced from normal, non-injured lung (9). Therefore, scaling tidal volumes to "baby lung" size may provide the optimal balance of adequate ventilation and lung protection. Using imaging techniques or changes in lung mechanics, such as driving pressures, as a means to determine a patient's "baby lung" size is an active area of research (5,7), but widely available techniques remain to be developed. Alternatively, Beitler *et al.* found that the volume delivered during a simple bedside recruitment maneuver, analogous to the inspiratory capacity of the "baby lung", was inversely related to lung stress (6). They propose scaling tidal volume to "baby lung" inspiratory capacity as a means to individualize lung-protective ventilation. Validation through larger and prospective studies is required before any new strategy can be universally recommended, but the ability to use a bedside tool to set a patient-specific tidal volume is a promising concept.

Positive end expiratory pressure (PEEP) titration is

another important consideration in ARDS management. Over 40 years ago, Ashbaugh *et al*. observed that a higher PEEP seemed to be an effective therapy in the ARDS patients they initially identified (1), and a high PEEP strategy is still common practice today (10). This makes physiological sense as higher PEEP adequate to sustain recruitment may prevent damage from the repetitive opening and closing of atelectatic but recruitable alveolar units (atelectrauma). Adequate PEEP also may decrease stress concentration by promoting more homogeneous mechanical behavior of aerated lung zones (11), and prevent overdistension lung injury (volutrauma) by increasing "baby lung" volume.

Despite these physiological effects, large multicenter randomized trials investigating high PEEP strategies have failed to show a survival benefit (12,13). Lack of benefit in these trials may be due in part to inclusion of patients with less severe lung injury, who may benefit less if at all from higher PEEP. In addition PEEP has many physiological effects including hemodynamic changes which need to be considered in determining optimal ventilator settings. Prior trials may also have failed due to lack of accounting for patient-specific differences in the contribution of the lung versus chest wall to respiratory system mechanics (14). For example, patients with higher pleural pressures would be more prone to atelectasis and may benefit from higher PEEP, while patients with lower pleural pressures may be more prone to injury by overdistention. Talmor *et al*. investigated this idea in a small single-center ARDS trial, using esophageal manometry to determine each patient's pleural pressure and titrating PEEP to maintain positive transpulmonary pressure (airway minus pleural) (15). They observed an improvement in oxygenation and respiratory system compliance and, after adjusting for overall illness severity, increased survival with esophageal pressure-guided PEEP titration. A larger multicenter trial on esophageal pressure-guided PEEP titration in ARDS is ongoing, but again it appears tailoring mechanical ventilation to patient-specific respiratory mechanics may be the key to preventing VILI and improving outcomes.

In conclusion, current evidence suggests a role for personalizing lung-protective ventilation in ARDS, although the ideal tidal volume and PEEP titration strategies remain to be defined. In the interim, intensivists should strive to understand how each individual's respiratory mechanics, pathophysiology, and comorbidities translate to likely VILI risk. Promising studies aimed at providing tools for this patient-specific lung protection are ongoing, and may very well lead to the next major breakthrough in ARDS management. A thorough physiological understanding of lung and chest wall mechanics may thus provide benefits to patients beyond current guidelines which generally provide a 'one-size-fits-all' approach.

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Footnote

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