Editorial

Acute respiratory distress syndrome and mechanical ventilation: ups and downs of an ongoing relationship trap

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During the early 20th century, patients suffering from acute severe pulmonary diseases died because medicine lacked the possibility to sufficiently treat lung failure (i.e., to assure ventilation with adequate oxygenation and exhalation of carbon dioxide) until lung healing occurred. The polio epidemic of the 1950s led to the evolution of mechanical ventilation (i.e., inspiratory positive pressure ventilation). This treatment increased the survival of patients with respiratory failure. However, about ten years later initial evidence revealed that mechanical ventilation—using ventilator settings may be adequate for healthy lungs—in itself bears the potential for further lung damage and in worst-case scenarios kills patients. Since then, almost thirty years have passed until the concept of "lung-protective ventilation" entered into clinical practice. There are two cornerstones of lung-protective ventilation. The first is low tidal volume (i.e., 6 mL/kg of predicted body weight) ventilation combined with limited inspiratory plateau pressures (i.e., <30 cmH_2O) to prevent lung over-distension ("barotrauma"/"volutrauma") (1). The second cornerstone is the application of high levels of positive end-expiratory airway pressure (PEEP) to prevent repetitive opening and closing of terminal lung units ("atelectrauma") (2,3). If present, "volutrauma" and/or "atelectrauma" can promote local and systemic inflammatory processes. These processes may lead to further injury of the lungs and distal organs, a phenomenon described as "biotrauma" by Tremblay and Slutsky (4).

In this issue of CHEST, Curley *et al*. present a comprehensive review of the potential side effects of mechanical ventilation (5). They focus on ventilation-induced inflammatory processes (i.e., "biotrauma"), on the promising role of biomarkers for risk stratification and ventilator management, and on potential genetic factors that might play a pivotal role in ventilation-induced inflammatory responses. Furthermore, their review provides an overview of measures proven to reduce mortality in acute respiratory distress syndrome (ARDS) patients (i.e., lung-protective ventilation, prone positioning, and neuromuscular blockade). Finally, it addresses the current significance of extracorporeal strategies [i.e., extracorporeal membrane oxygenation (ECMO) and extracorporeal carbon dioxide removal ($ECCO_2R$)]. Medical treatments (e.g., steroids, inhaled nitric oxide, surfactant, etc.) and other ventilatory possibilities (e.g., high-frequency oscillatory ventilation, etc.) that have failed to be effective in reducing mortality in adult ARDS patients are not the subject of this review.

Lung-protective ventilation with low tidal volumes, low inspiratory plateau pressures, and high levels of PEEP might depict a new chapter in the already-mentioned "relationship trap of ARDS and mechanical ventilation". Considering inter-individual and intra-individual inhomogeneity of ARDS lungs, the recommended tidal volume of 6 mL/kg of predicted body weight will only occasionally fit the individual accessible lung size (i.e., the "baby lung"). Likewise, uniform application of high levels of PEEP will concurrently result in the recruitment of atelectatic lung regions on the one hand and over-distension of less affected lung regions on the other, depending on the degree of lung inhomogeneity (6). To better deal with the inter-individual inhomogeneity at the very least, tidal volumes and PEEP should be applied according to the current individual respiratory system mechanics. In doing so for PEEP,

several methods have been proposed. The most promising method, as suggested by the authors of this review, is based on a PEEP level sufficiently high to maintain the transpulmonary pressure (i.e., the difference of airway opening pressure and esophageal pressure as a surrogate for pleural pressure) above zero at end-expiration (7). Similarly for the setting of tidal volumes, applied tidal volumes should be normalized to current dynamic respiratory system compliance according to the ratio $V_T/C_{rs} = P_{plat} - PEEP$. In this formula, the pressure difference $P_{plat} - PEEP$, termed "driving pressure", is proposed to be set as low as possible but to a maximum of 15 cmH_2O (8). From a conceptual point of view, such individualized setting of PEEP and tidal volume is highly promising although survival benefits have not yet been proven in prospective clinical trials.

Since the first mention of ARDS by Ashbaugh *et al.* in 1967 (9), things have progressed. Advancements include a better knowledge of the benefits and harms of mechanical ventilation as well as of treatment options in line with a consistent improvement in morbidity and mortality of these patients. While evidence on the superiority of lung-protective ventilation is most compelling in patients requiring prolonged mechanical ventilation in the intensive care unit, several studies have also shown its superiority in patients undergoing major surgery (10,11). Unfortunately, this knowledge still is far from being consequently implemented into clinical practice (12) although the targets and benefits of lung-protective ventilation have been known for more than a decade. The most burning problem at present is the gap between published knowledge and the actions of intensivists and anesthesiologists in charge of these patients. Vital knowledge is already present but needs to be consistently implemented into clinical practice.

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Footnote

Conflicts of Interest: The authors have no conflicts of interest to declare.

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