Editorial

Lung and chest wall mechanics in COVID-19 acute respiratory distress syndrome

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Submitted Sep 29, 2021. Accepted for publication Oct 20, 2021.
doi: 10.21037/jtd-21-1567
View this article at: https://dx.doi.org/10.21037/jtd-21-1567

The acute respiratory distress syndrome (ARDS) was first described by Ashbaugh *et al.* in *Lancet* 1967 (1). Although rarely acknowledged as such it is a leading contributor to death in the US, since many patients who succumb to respiratory infections meet criteria for ARDS before death. The coronavirus disease 2019 (COVID-19) pandemic has created a focus on ARDS, although there has been some controversy regarding whether COVID-19 causes typical ARDS or some related condition (2,3). Initially in the pandemic some sources were labeling these patients as having various other diseases including primarily vascular disease, congestive heart failure, high altitude pulmonary edema, and mucus plugging (4). Based on the ARDS Berlin definition, COVID-19 patients clearly meet ARDS criteria despite assertions to the contrary (5). Some authors pointed to relatively high compliance measurements as evidence against ARDS; however, compliance is not included as a criterion in the Berlin definition. Moreover, respiratory system compliance values in early COVID-19 are similar to reported values from other ARDS cohorts (6). We have observed low compliance values later in COVID-19 ARDS similar to what was previously observed with fibroproliferative ARDS (7). Given the similarities between ARDS in COVID-19 and non-COVID-19 ARDS, we recommend the same guiding principles of lung protective ventilation in the treatment of COVID-19 ARDS. We will highlight several aspects of pulmonary mechanics and lung protective ventilation relevant to COVID-19 and other ARDS patients.

One risk factor that has been commonly observed in COVID-19 is obesity (8,9). When considering lung and chest wall mechanics in morbid obesity, an important consideration is that of transpulmonary pressure (P_L) (10,11). P_L is defined as the pressure at the airway opening minus the pressure in the pleural space, which can be estimated using esophageal manometry (12). In obesity pleural pressures are often elevated and can contribute to risk of collapse of the lung at end-exhalation yielding atelectasis and reduced lung compliance. Elevated transpulmonary pressure at end-inflation is a marker of overdistension whereas low transpulmonary pressures at end-exhalation may contribute to atelectasis (13). Strategies using measurement of esophageal pressure to set positive end expiratory pressure (PEEP) have not consistently led to improved outcomes in ARDS (14,15); however, we do believe that the decisions regarding PEEP should be based on multiple factors. A tailored PEEP strategy should take into account gas exchange, hemodynamic issues, recruitability of the lung, minimizing lung stress and strain, and transpulmonary pressure to name a few (16). Thus, PEEP decisions should likely be personalized based on an individual's physiological characteristics (e.g., body fat distribution and its effects on pleural pressure) rather than a "one size fits all" approach. We have recently observed reasonably good outcomes in obese COVID-19 patients compared to non-obese when open lung protective ventilation strategies were appropriately provided (9,17).

One strategy that has received considerable attention is that of prone positioning (18). Prone positioning has shown consistent mortality benefits in ARDS and recent data are supportive of benefits to proning in non-intubated (PINI) patients (19,20). The mechanisms of improvement with prone positioning are debated, but we believe they are more complex than simply improvement in gas exchange (21).

With proning, there is recruitment of dorsal lung units, a reduction in the vertical pleural pressure gradient, and more homogeneous distribution of ventilation, which may serve to reduce local stresses on the lung (12). Mead *et al.* estimated markedly elevated local stresses at the junctions of normal and abnormal lung speaking to the potential benefits of lung homogeneity (12). We have had good results with proning of non-intubated patients even with morbid obesity, in many cases obviating the need for intubation and mechanical ventilation (22). At the beginning of the pandemic there was a notion that early intubation would be protective. However, more recent data suggest that standard criteria for intubation may be prudent (23). Thus, the strategy to prevent or delay intubation by reducing mechanical stresses on the lung may well be advisable.

In summary, we are highly supportive of further research to understand lung and chest wall mechanics in ARDS in general and in COVID-19 specifically. The association between obesity and severe COVID-19 highlights the important principles of transpulmonary pressure and prone positioning in the approach to lung protective ventilation. There are many promising strategies on the horizon including using electrical impedance tomography, esophageal manometry, predictive analytics, and novel biomarkers to help optimize outcomes in afflicted patients (24). A strong physiological understanding remains critical to a personalized approach to ARDS care (25). Mechanical ventilation continues to be a pivotal component in ARDS management and additional research is needed to further our understanding of pulmonary mechanics and clinical bedside application.

Acknowledgments

Funding: None.

Footnote

Provenance and Peer Review: Provenance and Peer Review: This article was commissioned by the editorial office, *Journal of Thoracic Disease*. The article did not undergo external peer review.

Conflicts of Interest: All authors have completed the ICMJE uniform disclosure form (available at https://dx.doi.org/10.21037/jtd-21-1567). AM receives research funding from the NIH. University of California, San Diego has received philanthropic donations from ResMed. The other authors have no conflicts of interest to declare.

Ethical Statement: The authors are accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved.

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Cite this article as: Pearce AK, McGuire WC, Malhotra A. Lung and chest wall mechanics in COVID-19 acute respiratory distress syndrome. J Thorac Dis 2021;13(11):6214-6216. doi: 10.21037/jtd-21-1567

