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## Understanding COVID-19 Acute Respiratory Distress Syndrome

*To the Editor*:

We read the article by Bain and colleagues with great interest and strongly believe that such work should be the focus of research (1). The authors share some interesting observations, such as lower minute ventilation seen in patients with coronavirus disease acute respiratory distress syndrome (COVID-19 ARDS). As the authors have rightly pointed out, it would mean that lower minute ventilation would be required to clear $CO_2$ owing to a lower fraction of dead space ventilation. This is a novel finding not described in patients with COVID-19 ARDS, even though the authors discuss a study by Liu and colleagues with similar findings (2). Liu and colleagues, on the contrary, observed high ventilatory ratios, which would correspond to higher dead space ventilation. The work of Liu and colleagues, published early in 2020—at a time when little was known about COVID-19 ARDS—offered no plausible explanation for their observation. Grieco and colleagues observed higher ventilatory ratios in individuals with COVID-19 ARDS (3); however, other small sample studies, such as one by Brault and colleagues, could not find any significant difference between the ventilatory ratio of patients with COVID-19 ARDS and that of patients with non–COVID-19 ARDS (4)—which brought us to examine the power of the study by Bain and colleagues. Small sample size aside, the study used historical control subjects, notably using different exclusion criteria in COVID-19 ARDS than in non–COVID-19 ARDS groups, which would make results less than comparable. There have been multiple attempts to understand the pathophysiology of COVID-19 ARDS (5), with one of the earliest attempts by Gattinoni and colleagues (6), who described different phenotypes of COVID-19 ARDS; however, it remains an enigma. Autopsy studies have revealed increased thrombus burden in individuals with COVID-19 ARDS and a possible pathogenetic role of microangiopathic vasculopathy in individuals with COVID-19 ARDS. Thrombosis and the relative failure of a hypoxic respiratory vasoconstriction mechanism in patients with COVID-19 ARDS are hypothesized as reasons for increased dead space ventilation in individuals with COVID-19 (7). We hope that a better understanding of the respiratory dynamics of COVID-19 ARDS could help save lives. ■

**Author disclosures** are available with the text of this letter at www.atsjournals.org.

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