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Alveolar Collapse Is a Threat in Injured Lungs, but What About the Airway Opening Pressure?

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To the Editor:

We read with interest the study by Sousa and colleagues that was published recently in the *Journal* (1). In a two-hit porcine homogenous lung injury model (surfactant depletion and high stretch ventilation), ventilation with positive end-expiratory pressure (PEEP) set to minimize overdistension (<3% of overdistension) resulted in a high amount of lung collapse and a higher risk of mortality attributed to right heart failure followed by cardiopulmonary collapse.

These findings, if confirmed in clinical studies, counter the view that preventing overdistension is more important than avoiding collapse. All three groups in this study were ventilated with PEEP levels that were within the vicinity of current clinical practice (median values = 7, 11, and 15 cm H_2O, respectively), albeit the vertical lung height and associated gravitational collapse pressure are not comparable between the porcine model and humans. We suggest that airway closure, rather than alveolar collapse, may provide an alternative explanation for the study findings. This is relevant, as the deterioration in respiratory mechanics, gas exchange, and hemodynamics (epinephrine dose) in animals randomized to the low-over-distension group was evident almost immediately after randomization (at Time 0), leading to early mortality.

Airway opening pressure is important in all types of lung injury, but it is most important in a model of increased surface tension (2). Surfactant depletion can cause central and peripheral airway closure, leading to injury, distal inflated alveoli at end expiration, and absorption atelectasis (3). Airway opening pressure can be observed on pressure–volume curves in excised dog lungs independent of chest wall or pleural mechanics (2). Airway opening pressure is observed in air-filled lungs but not in liquid-filled lungs, where surface tension that develops at the liquid–gas interface has been eliminated (2). In the canine caudal lobe, similar findings have been demonstrated because of the liquid–gas interface (4). Airway closure at end expiration has been reported in one third of patients with moderate or severe acute respiratory distress syndrome, with an airway opening pressure anywhere between 6 and 20 cm H_2O (5). This phenomenon of airway closure is particularly relevant, as the surfactant depletion model of lung injury, used in the study by Sousa and colleagues, aims to increase surface tension. As global and regional airway closure may be measured with electrical impedance tomography (6), it would be of interest to know these data.

It may be postulated that animals in the low overdistension group (1) had PEEP set below the airway opening pressure, resulting in repeated opening and closing of the distal airways, causing absorption atelectasis (3); alveolar collapse; and resultant ventilation perfusion mismatch, shunt, and right ventricular failure. Finally, although the authors measured intrinsic PEEP, failure to account for airway closure will lead to erroneous estimates of mean alveolar pressure and driving pressure.

Reporting airway opening pressure in the article and studies that explore PEEP in lung injury would help resolve these concerns. ■

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

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