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Lung Injury Still Too Common in Ventilation, Measures for Protection Insufficient

Ventilator-induced injury in the lungs is responsible for a vast number of deaths in acute respiratory distress syndrome (ARDS). Even healthy surgical patients, who require temporary mechanical ventilation, are at risk of ventilator-induced lung injury. Although such injuries have been reduced tremendously over the last few decades, a new study suggests, they have much further to go.

"It is ironic, because for a large number of patients with ARDS, it is the treatment, rather the syndrome, which ends up killing them," says Luciano Gattinoni, M.D., lead researcher of the study, which was published in the second issue for August of the American Thoracic Society's American Journal of Respiratory and Critical Care Medicine.

Assessing patients' lung stress and strain appropriately could mean the difference between life and death. Overestimating stress may lead to carbon dioxide build-up in the blood and low ventilation with atelectasis— or lung tissue collapse. Underestimating stress may enhance the risk of ventilator-induced lung injury. The only bedside assessments currently available are to compute tidal volume and plateau pressure as surrogate measures for lung stress and strain in ARDS patients.

To determine whether those measurements are adequate, Dr. Gattinoni and colleagues analysed true stress—the internal counterforce that reacts to an external load—and strain, the structural change associated with stress—in a total 80 patients, including postsurgical patients, patients with ARDS, patients with Acute Lung Injury (ALI) and patients with a medical disease. They used a number of measurements of lung stress and strain, primarily oesophageal pressure and lung volume assessment with helium dilution technique. The investigators found that there was little correlation between plateau pressure and tidal volume and actual lung stress and strain in all four groups.

While plateau pressures and tidal volumes may be reflective of the chest wall elastance and lung volume of the population as a whole, in circumstances where patients require mechanical ventilation, those general guidelines are inadequate to assess the individual's lung stress and strain. For example, there are certain clear indicators that the chest wall elastance may be altered (e.g. severe obesity). In this case, the plateau pressure would overestimate the stress.

"The consequences are, of course, potentially more dangerous in patients in which the chest wall elastance is more compromised and the lung volume is more reduced," said Dr. Gattinoni. "The immediate clinical implications are that clinicians should not trust the conventional measurements."

Going forward, Dr. Gattinoni and colleagues would like to see improved measures of lung stress and strain, including routine assessment of oesophageal pressure and lung volumes to compute stress and strain in large populations of mechanically ventilated patients.

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