Since January 2020 Elsevier has created a COVID-19 resource centre with free information in English and Mandarin on the novel coronavirus COVID-19. The COVID-19 resource centre is hosted on Elsevier Connect, the company's public news and information website.

Even patients undergoing short-term surgical procedures have benefited from low-tidal-volume ventilation. A small prospective trial (n=46) of lung-protective ventilation (6 mL/kg; positive end-expiratory pressure 10) versus conventional operative ventilation (12 mL/kg; positive end-expiratory pressure 0) in elective surgeries lasting longer than 5 hours showed increased bronchoalveolar coagulation and inflammatory mediators in bronchoalveolar lavage fluid,[8,9] despite no difference in clinical outcomes. This scenario suggests that lung injury may develop quickly with an injurious ventilator strategy and implies that even patients who are boarding in the ED should be protected from the injury that high-tidal-volume ventilation seems to promote.

For an intervention that has no cost, has limited risk, and saves lives of patients with diagnosed lung injury,[4] there are few reasons not to use a lung-protective ventilation strategy for all patients. The critical hours patients spend in the ED can set the course for the remainder of their hospital stay, and routinely providing protective ventilation is one important intervention for patients receiving mechanical ventilation in the ED. Acute lung injury and acute respiratory distress syndrome are iatrogenic diseases, and limiting tidal volume is the first step to improving outcomes in the critically ill.

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LOW TIDAL VOLUME SHOULD NOT ROUTINELY BE USED FOR EMERGENCY DEPARTMENT PATIENTS REQUIRING MECHANICAL VENTILATION

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Low tidal volume ventilation, convincingly demonstrated by the Acute Respiratory Distress Syndrome Network (ARDSNet) group in 2000,[1] is currently one of the most important treatment strategies in critically ill patients with acute lung injury or acute respiratory distress syndrome. Rather than adjust mechanical ventilation settings to normalize blood gas values, low tidal volume ventilation focuses on using smaller tidal volume (4 to 6 mL/kg of predicted body weight) and plateau pressure limits ($\leq$30 cm H_2O). A relatively higher $PaCO_2$ can be permitted—permissive hypercapnia—provided that the pH is in a "safe" range. The ARDSNet trial had a goal pH of 7.30 to 7.45 but tolerated pH greater than 7.15 in certain clinical scenarios.[1] Using low tidal volume ventilation, the ARDSNet trial showed a 22% reduction in mortality in acute lung injury/acute respiratory distress syndrome patients.[1] It can be tempting to apply these results "routinely" to patients without acute lung injury/acute respiratory distress syndrome, but the emergency physician should take caution because there are some clinical scenarios in which low tidal volume ventilation may be harmful.

In conditions of hypoxemic respiratory failure, low tidal volume ventilation may lead to atelectasis and continued hypoxemia, which will require the use of higher levels of positive end-expiratory pressure (PEEP) and oxygen. This was observed in the ARDSNet trial because patients in the low tidal volume ventilation group had worse pulmonary performance—as evidenced by a worse PaO_2/FiO_2 ratio and higher PEEP—in the first 3 days before improving.[1] If a low tidal volume ventilation strategy is to be used, PEEP must be titrated to avoid atelectrauma from the repeated opening and closing of lung units.[2] In sepsis or in multitrauma patients, using higher levels of PEEP to compensate for hypoxemia can lead to hypotension by decreasing venous return. Preload has to be monitored carefully, and excessive PEEP can lead to increased fluid and vasopressor requirements, potentially prolonging length of mechanical ventilation use.[3]

In conditions of hypercarbic respiratory failure, low tidal volume ventilation can increase dead space ventilation and lead to alveolar hypoventilation. To compensate, the respiratory rate must be increased. In the original ARDSNet study, the low tidal volume ventilation group had mean respiratory rates of approximately 30 breaths/min.[1] Higher respiratory rates will shorten the expiratory time, potentially leading to air trapping and auto-PEEP in certain patient populations with increased airway resistance. Acute respiratory distress syndrome is often characterized by a decrease in lung compliance, with a "stiffer" lung that can more easily exhale without gas trapping.[4] Patients with obstructive lung disease may

not tolerate such high respiratory rates without developing auto-PEEP. Auto-PEEP, especially in preload dependent states, can decrease venous return and lead to hemodynamic instability, increased vasopressor and fluid needs, and even cardiovascular collapse in severe cases. A recent trial examining the use of low tidal volume ventilation in patients without acute lung injury/acute respiratory distress syndrome patients specifically excluded patients with chronic obstructive pulmonary disease.[5]

Similarly, in severe brain injury patients, a low tidal volume ventilation strategy can lead to hypercarbia and increased intracranial pressure if the alveolar minute ventilation is too low and not monitored carefully. This can be dangerous and may require the constant monitoring of end tidal CO_2 ($ETCO_2$) once a baseline $PACO_2$-$ETCO_2$ gradient is established. Low tidal volume ventilation studies excluded patients with increased intracranial pressure.[1,5]

Recent work by Determann et al[5] suggests that low tidal volume ventilation results in a lower incidence of acute lung injury compared with "conventional" tidal volumes. In addition to being a small study that excluded many diagnoses for which emergency physicians typically prescribe mechanical ventilation, the study has a critical design flaw in that there is no true "control" group. Prerandomization tidal volume was approximately 8 mL/kg predicted body weight, whereas the authors compared 6 versus 10 mL/kg predicted body weight. A tidal volume of 10 mL/kg would therefore seem to be highly unconventional. It is difficult to determine whether these positive results reflect benefit from low tidal volume ventilation or harm from excessive "conventional" ventilation. A similar controversy surrounded the original ARDSNet trial, and the reader is encouraged to examine the work by Eichacker et al[6] and the ARDSNet response.[7]

Unfortunately, a "one size fits all" approach to mechanical ventilation does not exist. Given the available evidence, it is safe to say that 10 mL/kg is probably excessive and might be harmful in some emergency department (ED) patients who require mechanical ventilation.[1,5] Low tidal volume ventilation (4 to 6 mL/kg predicted body weight, depending on plateau pressure) is appropriate for patients with acute lung injury/acute respiratory distress syndrome.[1] However, pending further data, it is premature to advocate for the "routine" use of low tidal volume ventilation for all ED patients requiring mechanical ventilation. The authors' preference is to start at a modest tidal volume of 8 mL/kg predicted body weight, provided that the plateau pressure is acceptable ($\leq$30 cm H_2O) or to judiciously use pressure-based modes. The wisdom of this routine begs to be proven in a well-designed ED-based study.

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