Tidal volumes and lung-protective strategy in non-ARDS patients

18.02.2019

Author: Munir Karjahagli, Reviewer: David Grooms

Mechanical ventilation is a lifesaving technique, but one that potentially has important complications. According to several experimental and clinical studies, mechanical ventilation has great potential to augment and initiate injury to the lungs and respiratory muscles (1, 2).

Takeaway messages:

The recommended target tidal volume for most mechanically ventilated patients in the ICU is 6–8 ml/kg PBW.
Tidal volume targets should be individualized according to lung mechanics and the functional size of the lung, not only the body weight.
Tidal volume is only one of several variables that make up the mechanical power applied to the lung and therefore just one of several aspects to be considered for the prevention of ventilator-induced lung injury.
To limit lung stress and strain, driving pressure, plateau pressure, and tidal volume all have to be considered.
Airway driving pressure represents the cyclic strain placed on the lung parenchyma and setting ventilation parameters to decrease it may help improve outcomes.

In the year 2000, a landmark study comparing lower tidal volume (VT) values (6 ml/kg PBW) with higher VT values (12 ml/kg PBW) in ARDS patients was undertaken by the ARDS Net group (3). It was a large, multi-center prospective randomized trial with over 800 subjects enrolled. The study showed a survival advantage of 22% in the lower VT group. Currently, lung-protective ventilation is considered the standard of care for patients with moderate or severe acute respiratory distress syndrome (ARDS) (4–6), and since publication of this study, a growing body of evidence has suggested that the use of lower VT values may also improve clinical outcomes in patients without ARDS.

Lower tidal volumes in patients without ARDS

A meta-analysis of 20 publications (7) and a multi-center trial using lung-protective ventilation in 400 patients undergoing abdominal surgery (8) found that a low tidal volume strategy in non-ARDS patients was associated with significant survival benefits. These findings have led some editorialists to suggest that low tidal volumes (6–8 ml/kg PBW) should be used in most patients receiving mechanical ventilation (9).

The PReVENT trial was conducted with the specific objective of determining whether a ventilation strategy using low tidal volumes is superior to one using intermediate tidal volumes in critically ill patients without ARDS (10). The primary outcome was the number of ventilator-free days and alive at day 28. Results show that in ICU patients without ARDS, who were not expected to be extubated within 24 hours, a low tidal volume strategy (4–6...
ml/kg PBW) did not result in a greater number of ventilator-free days compared with an intermediate tidal volume strategy (8–10 ml/kg PBW). However, there was inadequate separation between the two strategies and the majority of the patients in the low tidal volume group did not achieve the trial targets; on average they received tidal volumes of around 7.8 ml/kg PBW (11).

**Concerns about using low VT**

There are several concerns about the potential consequences of a low tidal volume strategy, such as an increase in sedation needs and the incidence of ICU delirium (12). It may also increase ICU-acquired weakness (13) and patient-ventilator asynchrony (14), and promote the collapse of lung tissue (15). All these possible effects could offset the benefits of lower tidal volumes. Therefore, uncertainty remains as to whether ventilation with lower tidal volumes (≤ 6ml/kg) should be used routinely in all ICU patients and it is not yet recommended in guidelines for ventilation of patients without ARDS.

**Physical and biological triggers of ventilator-induced lung injury (VILI)**

Ventilator-induced lung injury (VILI) is commonly attributed to the application of excessive tidal volume (volutrauma) or airway pressure (barotrauma) (16). However, volutrauma and barotrauma are primarily caused by unphysiological lung distortion or strain - the ratio between VT and functional residual capacity (FRC) - and stress (the transpulmonary pressure). VILI is therefore the global/regional excessive stress and strain applied to the lungs. The rough equivalent of the stress in the whole lung is the transpulmonary pressure (PL), while the equivalent of the strain is the change in the size of the lung from its resting position, i.e., the ratio of VT to the size of the lung at end-expiration. To prevent VILI by applying stress and strain within physiological limits, we must take the VT/FRC ratio, not the VT/kg ratio. In 2016, Chiumello et al. showed that similar amounts of volume can produce different stress with similar body weights, and the tidal volume based on ideal body weight is not related to the amount of aerated lung volume (17). One possible solution for limiting lung stress may be to titrate VT according to respiratory compliance, which reflects the functional size of the lung and volume of aerated lung tissue that is available for tidal ventilation. In the normal lung, doubling of the resting volume occurs at approximately 80% of total lung capacity, and at this level of strain (VT/end-expiratory lung volume=1) PL equals the specific lung elastance, normally 12 cmH2O. In 2011, Protti et al. showed that in healthy humans, the critical threshold for the development of lung edema may correspond to a strain interval of between 1.5 and 2 (18).

**Tidal volume is not the only parameter to consider**

A recent study by Amato et al. (19) used data from nine randomized trials evaluating mechanical ventilation in ARDS to assess whether VT normalized to respiratory system compliance (strain) was a better predictor of injury. He found that the respiratory system
driving pressure ($\Delta P$; plateau pressure - positive end-expiratory pressure) was the ventilation variable that best stratified risk. Decreases in $\Delta P$ (< 15 cmH2O) owing to changes in ventilator settings were strongly associated with increased survival. A recent analysis by Kassis reviewed data from the original EPVent 1 study and revealed survivors at day 28 were most commonly ventilated with a transpulmonary driving pressure of < 10 cmH2O (20). Airway driving pressure represents the cyclic strain to which the lung parenchyma is subjected during each ventilatory cycle. It is a physiological way of adjusting VT to the residual lung size (respiratory system compliance) of the patient and correlates directly with transpulmonary pressure. Therefore, setting ventilation parameters to decrease driving pressure may play a role in improving outcomes in patients requiring mechanical ventilation.

**Lung injury components**

VILI originates from the interaction between the lung parenchyma and the mechanical power or energy applied to them by the ventilator. Tidal volume is only one of several variables that make up this mechanical power, namely pressures, volume, flow, and respiratory rate (21). We should therefore take into account all these variables - and the combination of them - when mechanically ventilating patients. To limit lung stress and strain, for example, it is $\Delta P$, plateau pressure (Pplat), and VT that all have to be considered. We must keep in mind that mechanical ventilation is a “global strategy” for diagnosis, management and prevention, so it is very important to focus on the underlying lung pathophysiology, individualized ventilator settings and VT targets during the management of mechanically ventilated patients.

On Hamilton Medical ventilators, Adaptive Support Ventilation (ASV®) selects the tidal volume, respiratory rate and inspiratory time according to respiratory mechanics. If respiratory system compliance is decreased, the automatically selected tidal volume will be lower. In addition, ASV has been shown in a prospective observational study to ventilate 95% of patients with different lung conditions with driving pressures of less than 14 cmH2O (22).

**References**


Date of Printing: 23.08.2019

Disclaimer: The content of this newsletter is for informational purposes only and is not intended to be a substitute for professional training or for standard treatment guidelines in your facility. Any recommendations made in this newsletter with respect to clinical practice or the use of specific products, technology or therapies represent the personal opinion of the author only, and may not be considered as official recommendations made by Hamilton Medical AG. Hamilton Medical AG provides no warranty with respect to the information contained in this newsletter and reliance on any part of this information is solely at your own risk.