Title

Personalizing mechanical ventilation for acute respiratory distress syndrome

Journal

Journal of Thoracic Disease, 8(3)

ISSN

2072-1439

Authors

Berngard, S Clark
Beitler, Jeremy R
Malhotra, Atul

Publication Date

2016-03-01

DOI

10.21037/jtd.2016.02.57

Peer reviewed

Review Article

Personalizing mechanical ventilation for acute respiratory distress syndrome

S. Clark Berngard, Jeremy R. Beitler, Atul Malhotra

Division of Pulmonary and Critical Care Medicine, University of California, San Diego, La Jolla, CA, USA

Contributions: (I) Conception and design: All authors; (II) Administrative support: None; (III) Provision of study materials or patients: None; (IV) Collection and assembly of data: None; (V) Data analysis and interpretation: None; (VI) Manuscript writing: All authors; (VII) Final approval of manuscript: All authors.

Correspondence to: S. Clark Berngard. Division of Pulmonary and Critical Care Medicine, University of California, San Diego, 9300 Campus Point Drive, 7381 La Jolla, CA 92037-7381, USA. Email: sberngard@ucsd.edu.

Abstract: Lung-protective ventilation with low tidal volumes remains the cornerstone for treating patient with acute respiratory distress syndrome (ARDS). Personalizing such an approach to each patient's unique physiology may improve outcomes further. Many factors should be considered when mechanically ventilating a critically ill patient with ARDS. Estimations of transpulmonary pressures as well as individual's hemodynamics and respiratory mechanics should influence PEEP decisions as well as response to therapy (recruitability). This summary will emphasize the potential role of personalized therapy in mechanical ventilation.

Keywords: Acute respiratory distress syndrome (ARDS); acute lung injury; ventilator-induced lung injury (VILI); positive-pressure respiration; respiratory mechanics

Submitted Jan 20, 2016. Accepted for publication Jan 21, 2016.
doi: 10.21037/jtd.2016.02.57
View this article at: http://dx.doi.org/10.21037/jtd.2016.02.57

Introduction

The optimal ventilatory support strategy for patients with acute respiratory distress syndrome (ARDS) remains to be defined. Several interrelated cardiopulmonary physiological factors—transpulmonary pressures, lung and chest wall mechanics, hemodynamics, and lung recruitability—can influence the risks and benefits of various ventilator strategies and thus the optimal ventilatory support approach. Here we review physiological concepts to consider in developing a personalized approach to mechanical ventilation, namely one tailored to the individual physiology of a patient to maximize lung protection in ARDS.

Balancing mechanics and gas exchange

To date, the majority of interventions suggested in major clinical studies to improve survival in ARDS (low tidal volumes, prone positioning, high PEEP, neuromuscular blockade) also appear to prevent mechanical lung injury. Indeed, the landmark NHLBI ARDS Net trial demonstrated that low tidal volume ventilation may worsen PaO_2:FiO_2 ratio for the first several days and yet improves survival compared to high tidal volume approaches (1). Classically, four mechanical mechanisms of ventilator-induced lung injury (VILI) have been described: volutrauma (overdistension), barotrauma (high distending pressures), atelectrauma (local interfacial stress from cyclic opening/collapse during tidal ventilation), and high shear strain from regional inhomogeneity (inflation of normal alveolus adjacent to collapsed or fluid-filled alveolus) (2-4). Resulting mechanical injury may cause a systemic cytokine storm, termed biotrauma, which exacerbates lung injury and contributes to multiorgan failure (5). Maintaining adequate oxygenation to preserve end-organ oxygen delivery and CO_2 excretion to preserve physiological pH unquestionably are important aspects of mechanical ventilation. The preponderance of literature indicates the optimal ventilatory

strategy should also prioritize prevention of mechanical lung injury while maintaining adequate gas exchange.

Transpulmonary pressure

Current standard of care for ARDS includes limiting tidal volume to ~6 mL/kg predicted body weight (1,6). The most widely used strategy also recommends maintaining plateau pressures of no more than 30 cm H_2O. However, plateau airway pressure alone is an unreliable estimate of lung distension. Transpulmonary pressure (airway opening minus pleural pressure) is the pertinent distending pressure of the lung (7). Pleural pressure, estimated via esophageal manometry, has been shown to differ considerably among patients with acute respiratory failure, indicating that chest wall mechanics contribute substantially and unpredictably to respiratory system mechanics and airway pressures measured by the ventilator (8). In a small pilot clinical trial of 61 patients with ARDS, esophageal pressure-guided ventilation was associated with improved oxygenation and, after adjusting for illness severity, improved survival (8,9). A multicenter validation trial powered for patient-centered outcomes is ongoing (10).

Recruitability

ARDS is characterized by heterogeneous parenchymal involvement, with well-aerated regions adjacent to collapsed or fluid-filled regions as evident with chest computed tomography. High shear forces can occur at junctions of normal and abnormal lung. There is no applied airway pressure that is clearly safe (7,11). Axial imaging studies have provided insight into the ARDS lung. In the classic CT for ARDS, some lung anteriorly appears radiographically to be relatively normal, whereas some portion of the lung is partially collapsed i.e. recruitable, and the most posterior portion of the lung is collapsed/flooded and unable to participate in gas exchange. The concept of the 'baby lung' has been put forward to define the portion of the lung able to participate in gas exchange (7) and a rationale for low tidal volume ventilation. However, this baby lung is highly variable in ARDS patients and unlikely to be predicted by ideal body weight or other demographic factors. Thus, strategies to define the size of the baby lung may have value in guiding the optimal ventilator settings (12). In some patients, elevation in airway pressures can lead to increased alveolar volume (i.e., recruitment). However, in other patients a similar elevation in airway pressure may lead to overdistension and hemodynamic sequelae. Thus, an assessment of the recruitability of the lung can be helpful in guiding individualized ventilator settings. In theory, patients with large 'baby lung' could tolerate higher tidal volumes than those with smaller 'baby lungs' (13). Strategies to promote lung homogeneity, such as higher PEEP, recruitment maneuvers (i.e., sustained high pressure inflation), and probing, may promote lung protection by reducing parenchymal stress in areas of heterogeneity.

Pleural pressure and body habitus effect on hemodynamics

Depending on the patient's intravascular volume status, cardiac function, and pulmonary physiology, mechanical ventilation can have beneficial or deleterious effects on hemodynamics. Elevated intrathoracic pressures in a hypovolemic patient with normal lungs can compress pulmonary vasculature, leading to increased pulmonary resistance and decreased preload, and cause a fall in cardiac output. However, in diseased lung states such as ARDS, low lung volumes and atelectasis also contribute to increased pulmonary vascular resistance. Optimizing PEEP may recruit more lung and actually improve cardiac output (14). Further, elevated intrathoracic pressure can also lead to reduced ventricular afterload due to lowering of ventricular transmural pressure and wall stress.

Clearly, the effect of various manipulations in mechanical ventilator settings is highly variable on hemodynamics depending on volume status and ventricular function of a particular patient. Focusing ventilator adjustments purely on blood gases and/or lung mechanics is likely to overlook potentially important effects on heart-lung interactions (15,16).

Conclusions

In summary, many factors should be included when mechanically ventilating a critically ill patient. Individual's hemodynamics and respiratory system mechanics should influence PEEP decisions as well as response to therapy (recruitability). Although further data are needed, small, randomized trials have shown promise in titrating ventilator settings based on lung and chest wall mechanics.

Acknowledgements

None.

Footnote

Conflicts of Interest: The authors have no conflicts of interest to declare.

References

1. Ventilation with lower tidal volumes as compared with traditional tidal volumes for acute lung injury and the acute respiratory distress syndrome. The Acute Respiratory Distress Syndrome Network. N Engl J Med 2000;342:1301-8.
2. Fu Z, Costello ML, Tsukimoto K, et al. High lung volume increases stress failure in pulmonary capillaries. J Appl Physiol (1985) 1992;73:123-33.
3. Oeckler RA, Lee WY, Park MG, et al. Determinants of plasma membrane wounding by deforming stress. Am J Physiol Lung Cell Mol Physiol 2010;299:L826-33.
4. Perlman CE, Lederer DJ, Bhattacharya J. Micromechanics of alveolar edema. Am J Respir Cell Mol Biol 2011;44:34-9.
5. Imai Y, Parodo J, Kajikawa O, et al. Injurious mechanical ventilation and end-organ epithelial cell apoptosis and organ dysfunction in an experimental model of acute respiratory distress syndrome. JAMA 2003;289:2104-12.
6. Malhotra A. Low-tidal-volume ventilation in the acute respiratory distress syndrome. N Engl J Med 2007;357:1113-20.
7. Mead J, Takishima T, Leith D. Stress distribution in lungs: a model of pulmonary elasticity. J Appl Physiol 1970;28:596-608.
8. Talmor D, Sarge T, O'Donnell CR, et al. Esophageal and transpulmonary pressures in acute respiratory failure. Crit Care Med 2006;34:1389-94.
9. Talmor D, Sarge T, Malhotra A, et al. Mechanical ventilation guided by esophageal pressure in acute lung injury. N Engl J Med 2008;359:2095-104.
10. Talmor DS. EPVent 2- A Phase II Study of Mechanical Ventilation Directed by Transpulmonary Pressures (EPVent2). ClinicalTrials.gov 2012;NCT01681225. Available online: https://clinicaltrials.gov/ct2/show/NCT01681225
11. Terragni PP, Rosboch G, Tealdi A, et al. Tidal hyperinflation during low tidal volume ventilation in acute respiratory distress syndrome. Am J Respir Crit Care Med 2007;175:160-6.
12. Beitler JR, Majumdar R, Hubmayr RD, et al. Volume Delivered During Recruitment Maneuver Predicts Lung Stress in Acute Respiratory Distress Syndrome. Crit Care Med 2016;44:91-9.
13. Gattinoni L, Pesenti A. The concept of "baby lung". In: Pinsky MR, Brochard L, Hedenstierna G, et al., editors. Applied Physiology in Intensive Care Medicine 1. Berlin: Springer Berlin Heidelberg, 2012:289-97.
14. Sarge T, Loring SH, Yitsak-Sade M, et al. Raising positive end-expiratory pressures in ARDS to achieve a positive transpulmonary pressure does not cause hemodynamic compromise. Intensive Care Med 2014;40:126-8.
15. Malhotra A, Muse VV, Mark EJ. Case records of the Massachusetts General Hospital. Weekly clinicopathological exercises. Case 12-2003. An 82-year-old man with dyspnea and pulmonary abnormalities. N Engl J Med 2003;348:1574-85.
16. Kato T, Suda S, Kasai T. Positive airway pressure therapy for heart failure. World J Cardiol 2014;6:1175-91.

Cite this article as: Berngard SC, Beitler JR, Malhotra A. Personalizing mechanical ventilation for acute respiratory distress syndrome. J Thorac Dis 2016;8(3):E172-E174. doi: 10.21037/jtd.2016.02.57