

# Acute Respiratory Distress Syndrome: The Best Ventilator Techniques

Abdulrahman Theeb Mohammed Alqahtani<sup>1</sup>, Khaled Mohammed Nahil Alotaibi<sup>2</sup>, Amer Obelk Attya Alanazi<sup>3</sup>, Owaidh Faisal Bijad Albagami<sup>4</sup>

<sup>1-4</sup>Respiratory Therapy Technician.  
DOI: 10.47750/pnr.2019.10.01.17

## Abstract

As our knowledge of the pathophysiology of acute respiratory distress syndrome (ARDS) has grown, so too have the methods for mechanical ventilation in these patients. The provision of lung-protective mechanical ventilation, which gives priority to tidal volume and plateau pressure limitation, is essential to the treatment of afflicted patients. By tailoring target tidal volumes to each patient's unique respiratory mechanics, lung protection can likely be further improved. There is still uncertainty regarding the best process for choosing the ideal positive end-expiratory pressure (PEEP) in ARDS; lung recruitability is one of the important factors to take into account. In ARDS, noninvasive ventilation must be used carefully because an overly high respiratory drive can worsen lung damage; newer delivery methods show promise in treating hypoxemic respiratory failure. Although clinical trials have not shown that airway pressure release ventilation improves survival over traditional ventilation techniques, it does provide an alternate method to optimize lung recruitment and oxygenation. In cases of refractory hypoxemia, high-frequency oscillatory ventilation rescue therapy is a crucial alternative. High-frequency oscillation may help patients with extremely severe hypoxemia, despite its disappointing lack of benefit (and potential harm) in patients with moderate or severe ARDS, which may be caused by lung hyperdistention and right ventricular dysfunction.

**Keywords:** Acute Respiratory Distress Syndrome, Mechanical Ventilation, High-Frequency Oscillation, Noninvasive Ventilation, Oxygen.

## Introduction

The cornerstone of treatment for acute respiratory distress syndrome (ARDS) is mechanical ventilation; without it, ARDS probably would not have been identified. Optimal ventilator management in ARDS has been the focus of intense research efforts for the past few decades and will remain so for decades to come, given the significance of preventing lung injury and guaranteeing adequate gas exchange. This review offers a cutting-edge synopsis of various ARDS mechanical ventilator management topics (Amato, 1998).

### Tidal Volume Optimization:

Through a number of mechanisms, lower tidal volumes ( $V_T$ ) reduce biophysical lung injury. Cellular and extracellular matrix damage can be decreased by preventing frank overdistension (volutrauma/barotrauma), reducing cyclic opening and collapse of small airways/alveoli (atelectrauma), and lowering tidal shear strain in areas of mechanical heterogeneity. Targeting  $V_T$  of 6 mL/kg predicted body weight (PBW) as opposed to 12 mL/kg PBW has been shown in multicenter trials to improve survival in patients with ARDS, reduce systemic inflammation, and speed up the resolution of multiple organ failures (Dreyfuss, 1988).

Although it is better than 12 mL/kg PBW, it is unknown what the best  $V_T$  strategy is for ARDS. According to two mechanistic human studies, in certain patients with severe ARDS and poor respiratory system compliance, lowering  $V_T$  below 6 mL/kg PBW by using extracorporeal life support as necessary to maintain adequate gas exchange may reduce systemic inflammation. In order to answer this question, patients with severe acute respiratory distress syndrome (ARDS) were randomly assigned to either extracorporeal membrane oxygenation or conventional mechanical ventilation (CMV) in the recently released EOLIA trial (Cressoni, 2014).

**Lung recruitment and positive end-expiratory pressure:** PEEP has been essential to the treatment of hypoxemia in ARDS since Ashbaugh and colleagues first described the condition. Higher PEEP has been investigated more recently as a potential preventative measure against lung damage. Although a number of PEEP

optimization techniques have been tried, none of them have yet to reduce mortality, and one of them was even linked to negative outcomes in ARDS patients. As a result, PEEP titration is still a contentious aspect of ventilator management, and PEEP at the bedside requires extreme caution (Cressoni, 2016).

### The physiological foundation for PEEP titration:

PEEP may reduce the risk of ventilator-induced lung injury (VILI) when used appropriately. PEEP avoids the shear stresses brought on by the cyclic opening and closing of alveolar units, or "atelectrauma," by preventing the end-expiratory collapse of alveoli and small airways during tidal ventilation. PEEP opens collapsed lung units to participate in gas exchange, thereby improving oxygenation and effectively reducing intrapulmonary shunt. The dynamic tidal strain and stress placed on the lung are decreased by increasing the number of aerated lung units involved in ventilation. Lastly, PEEP-induced lung recruitment leads to more uniform lung inflation, which can greatly lessen mechanical stress brought on by the lung's local inhomogeneities, which serve as stress multipliers (Ghadiali, 2011).

### ARDS Noninvasive Ventilation:

Noninvasive ventilation (NIV) has a contentious and developing role in the treatment of acute respiratory distress syndrome. There is little evidence to support the use of NIV in ARDS, despite the fact that it has significant benefits for acute exacerbations of cardiogenic pulmonary edema and chronic obstructive pulmonary disease. Given the established risks associated with invasive mechanical ventilation, deep sedation, neuromuscular blockade, and immobility, the possibility of avoiding this procedure in ARDS patients is intriguing. NIV is frequently used as an initial supportive therapy for ARDS despite the lack of evidence, and it was officially recognized as a therapeutic approach in the Berlin Criteria that define ARDS (Muscadere, 1994).

### Oscillatory ventilation at high frequencies:

HFOV is a special kind of mechanical ventilation that delivers ventilation at very low tidal volumes and high frequencies by using unconventional gas exchange mechanisms.

Justification for ARDS Use: The recurrent recruitment and collapse of lung units (atelectrauma) as well as excessive tidal volume and pressure applied to the lung are thought to be the causes of VILI (Taskar, 1997).

### Principles of Operations and Physiology:

Through a number of hypothesized mechanisms, ventilation (CO<sub>2</sub> clearance) is accomplished during HFOV even though V<sub>T</sub> is delivered below physiological dead space.

One important mechanism of gas exchange is convective bulk flow. Usually, the proximal gas exchange units exhibit the most noticeable bulk flow. Here, gas exchange is even more noticeable at airway bifurcations, where opposing convection currents are created by the asymmetric velocity profiles of inspired and expired gases. Other gas exchange mechanisms during HFOV include cardiac oscillations and pendelluft. Gas exchange is facilitated by the movement of gas between lung units with different inflation and deflation time constants (pendelluft), as well as by the percussion-like movement of gas molecules brought on by cardiac contractions (Gajic, 2003).

### Recommendations:

- Even though HFOV continuously improves oxygenation, it is currently only advised as a rescue treatment because it is linked to worse outcomes in unselected ARDS patients. This technique's effectiveness is probably limited by cardiopulmonary effects.
- Applying HFOV carefully is necessary to prevent hemodynamic impairment. By increasing afterload and decreasing preload, HFOV can significantly affect the right ventricle (RV) through elevated mPaw.
- The potential harm of NIV from delays in endotracheal intubation or worsening of lung injury must be weighed against the risk of complications with invasive mechanical ventilation. More research is necessary to determine patient subgroups who might benefit from NIV, comprehend how the NIV interface affects the physiology and outcomes of lung injury, and compare NIV to other respiratory support devices like HFNC as the debate over controlled invasive mechanical ventilation versus spontaneous breathing in ARDS continues.

## Conclusion:

According to this review, there is still a lot of work to be done to optimize mechanical ventilation. To decide how best to ventilate the patient, the knowledgeable clinician will carefully take into account the patient's clinical characteristics, physiological state, and reaction to ventilatory support. Providing suitable gas exchange while attempting to reduce dynamic stress and strain on the injured lung are the main objectives of optimal ventilator management. This can be achieved by reducing tidal volume as much as is clinically allowed, raising PEEP if better mechanics and gas exchange indicate that it is beneficial, and using evidence-based techniques like prone positioning and neuromuscular blockade. It may also be appropriate to use NIV sparingly in carefully chosen patients with mild to moderate ARDS. Although extracorporeal therapies may become more significant in this situation, HFOV and APRV may be taken into consideration as options for rescue therapy in patients who are deteriorating.

## References:

1. Amato M B, Barbas C S, Medeiros D M et al. Effect of a protective-ventilation strategy on mortality in the acute respiratory distress syndrome. *N Engl J Med.* 1998;338(06):347–354.
2. Dreyfuss D, Soler P, Basset G, Saumon G. High inflation pressure pulmonary edema. Respective effects of high airway pressure, high tidal volume, and positive end-expiratory pressure. *Am Rev Respir Dis.* 1988;137(05):1159–1164.
3. Cressoni M, Cadringher P, Chiurazzi C et al. Lung inhomogeneity in patients with acute respiratory distress syndrome. *Am J Respir Crit Care Med.* 2014;189(02):149–158.
4. Cressoni M, Chiumello D, Chiurazzi C et al. Lung inhomogeneities, inflation and [18F]2-fluoro-2-deoxy-D-glucose uptake rate in acute respiratory distress syndrome. *Eur Respir J.* 2016;47(01):233–242.
5. Ghadiali S, Huang Y. Role of airway recruitment and derecruitment in lung injury. *Crit Rev Biomed Eng.* 2011;39(04):297–317.
6. Muscedere J G, Mullen J B, Gan K, Slutsky A S. Tidal ventilation at low airway pressures can augment lung injury. *Am J Respir Crit Care Med.* 1994;149(05):1327–1334.
7. Taskar V, John J, Evander E, Robertson B, Jonson B. Surfactant dysfunction makes lungs vulnerable to repetitive collapse and reexpansion. *Am J Respir Crit Care Med.* 1997;155(01):313–320.
8. Gajic O, Lee J, Doerr C H, Berrios J C, Myers J L, Hubmayr R D. Ventilator-induced cell wounding and repair in the intact lung. *Am J Respir Crit Care Med.* 2003;167(08):1057–1063.
9. Matthay M A, Bhattacharya S, Gaver D et al. Ventilator-induced lung injury: in vivo and in vitro mechanisms. *Am J Physiol Lung Cell Mol Physiol.* 2002;283(04):L678–L682.
10. Pelosi P, Rocco P R. Effects of mechanical ventilation on the extracellular matrix. *Intensive Care Med.* 2008;34(04):631–639.