Mechanical Ventilatory Support: What Every Anesthesia Provider Should Know

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Respiratory failure is the leading cause for admission to most intensive care units (ICU’s). Providing mechanical ventilation is a core competency for every anesthesia provider, whether in the operating room or ICU. Mortality from respiratory failure continues to fall, in part due to the recognition that the technique of mechanical ventilation may result in propagation of injury. A number of recent advances have identified superior techniques for the management of patients with acute respiratory failure and the acute respiratory distress syndrome (ALI/ARDS). In order for the clinician to develop an understanding of modern mechanical ventilation, this lecture will review new understanding of the pathophysiology of ventilator associated lung injury (VALI) and new techniques for the management of patients requiring mechanical ventilation. In addition, new data regarding support of postoperative respiratory failure is reviewed. New data regarding lung recruitment maneuvers is examined.

Ventilator Associated Lung Injury

Patients with ALI/ARDS usually require mechanical ventilation, yet the process of mechanical ventilation itself can induce or worsen lung injury.1,2 To understand the rationale behind the various modes of mechanical ventilation, we must first examine how the interactions between the ventilator and the lung. The mechanisms for VALI are multifactorial, and include:

1. **Volutrauma**: Direct injury to alveoli from over-distention of the lung.
2. **Barotrauma**: Injury resulting from high intra-pulmonary air pressures.
3. **Biotrauma**: Lung and distant organ injury resulting from the release of inflammatory mediators into the airspaces and into the systemic circulation.
4. **Atelectrauma**: Injury to alveoli resulting from the cyclic collapse and opening of atelectatic alveoli.

**Volutrauma**: Mechanical ventilation with large tidal volumes causes both physiologic and histologic injury to the lung. This phenomenon can be separated from barotrauma, since when thoracic expansion is prevented, high inspiratory pressures without lung expansion do not cause injury. In patients with ALI/ARDS, the lung parenchyma tends to be heterogeneous, with atelectasis in dependent areas of the lung, and relative over-expansion in the non-dependent areas. When a mechanical ventilatory breath is delivered, it will distribute according to regional compliance. Heterogeneity in compliance may result with large tidal volumes being delivered to normal or already overdistended lung regions. Limitation of tidal volume can prevent regional overdistention, and was the basis of the successful ARDSnet trial. It is likely that the most important determinant of volutrauma is end-inspiratory volume, rather than the tidal volume delivered. There is no convincing evidence that volutrauma occurs in normal lungs until tidal volumes are very large.

**Barotrauma**: It can be difficult to separate the effects of high inspiratory pressure from those of high tidal volume. In patients with ALI/ARDS, edema, infiltration of inflammatory cells, and formation of hyaline membranes all contribute to worsened pulmonary compliance. Regardless of the mode of mechanical ventilation used, inspiratory pressures are increased. Peak inspiratory pressures (PIP’s) are increased from a combination of decreased static and dynamic lung compliance, and large and small airway secretions. As opposed to PIP, it is likely that the most important determinant of barotrauma is the inspiratory plateau pressure. It is this pressure that most accurately reflects end-inspiratory volume, and therefore lung injury. Keeping plateau pressure below 35 cmH2O is a reasonable target to minimize barotraumas, and has been associated with improved outcomes.

**Biotrauma**: Mechanical ventilation, especially with large tidal volumes, results in inflammatory mediator release into the alveolar space and systemic circulation. When lung epithelial cells are stretched, they are stimulated to produce these inflammatory mediators. These mediators have local and systemic effects identical to those seen in...
severe sepsis. It is in this manner that patients presenting with primary pulmonary pathology, such as pneumonia, can progress to multiple organ dysfunction syndrome (MODS). A large number of mediators have been implicated, including TNFα, IL-1, IL-6, and metalloproteinases.\textsuperscript{10} There are no specific strategies to prevent biotrauma, other than targeting prevention of VALI.

Atelectrauma: The cyclic opening and closing of atelectatic alveoli results in the phenomenon of atelectrauma. ALI/ARDS results in pulmonary edema, and the inflammatory mediators and proteases released into the alveolar space can disrupt the functioning of surfactant.\textsuperscript{5} These patients often have pleural effusions, and the combination of these factors result in significant atelectasis, best appreciated on CT scan.\textsuperscript{11} When the atelectatic lung is inflated, there is thought to be transmission of high shear pressures to the delicate alveolar wall. At very low tidal volumes, lung compliance is low, up to what is termed Pflex, where atelectatic alveoli start to be recruited, and lung compliance improves. To avoid this region of low lung compliance, and avoid atelectrauma, positive end-expiratory pressure (PEEP) can be set to a level higher than Pflex. Alternatively, alveolar recruitment maneuvers can be employed. The efficacy of these maneuvers is discussed in the section on protective mechanical ventilation.

Protective Mechanical Ventilation

Protective mechanical ventilation refers to the practice of setting the mechanical ventilator with the goal of minimizing lung injury.\textsuperscript{12} There is no single, agreed upon approach to protective ventilation, yet most strategies share the basic components of low tidal volumes and permissive hypercapnea. This section will review the rationale for this practice, along with the major clinical trials demonstrating its efficacy.

Traditionally, patients with ALI/ARDS were mechanically ventilated with large tidal volumes. A 1996 survey by Carmichael et al found that tidal volumes greater than 10 ml/kg were routinely used.\textsuperscript{13} Clinicians used these larger tidal volumes because in general, when tidal volume and therefore mean airway pressure are increased, oxygenation improves. With recent understanding of the detrimental effects of mechanical ventilation, a number of studies have been carried out in order to identify the most efficacious (least injurious) method to support patients with ALI/ARDS. After identification of volutrauma as a probable mediator of VALI, the first study to prospectively test low tidal volumes was that by Hickling et al, who used low tidal volume and permissive hypercapnea in patients with ARDS.\textsuperscript{14} Following this landmark study, a number of prospective clinical trials were performed testing protective ventilation, culminating in the ARDSnet trial.\textsuperscript{4}

The ARDSnet trial was a randomized, prospective trial comparing traditional tidal volume (12 ml/kg ideal body weight) to low tidal volume (6 ml/kg IBW). All patients received assist-control ventilation, and were weaned by protocol. Specific targets were set for all ventilator parameters, including rate, PEEP, FiO2, and management of acidosis. A total of 861 patients were enrolled, and the trial was halted early by the data safety monitoring board because of efficacy in the low tidal volume group. The important outcomes in this study included a reduction in mortality from 40% to 31%, an increase in the number of ventilator-free days, and a decrease in non-pulmonary organ system failures. There was a considerable amount of controversy regarding this study, focused primarily on the use of 12 ml/kg in the control group.\textsuperscript{15} Subsequent investigation fully supported the results of the trial, and at this time, ventilation of patients with ALI/ARDS should be according to the ARDSnet protocol. This study contained an important surrogate outcome: patients in the high tidal volume group had significantly improved oxygenation, yet increased mortality. This finding suggests that biotrauma, with remote organ injury, is a critical component of the efficacy of protective mechanical ventilation.

An important component of the ARDSnet trial was the selection of PEEP and FiO2 parameters. As stated previously, levels of PEEP that exceed Pflex were thought to protect against atelectrauma, and possibly improve outcomes. Adverse effects of high levels of PEEP include increased risk of barotrauma and decreased venous return. The ARDSnet group examined the effect of higher levels of PEEP compared to traditional levels of PEEP, when used in conjunction with 6 ml/kg IBW tidal volume. They were unable to demonstrate additional benefit.
when higher levels of PEEP were used, and therefore the same PEEP/FiO2 parameters employed in the ARDSnet trial are recommended. Similarly, recruitment maneuvers, where the lung is subjected to prolonged periods of continuous positive airway pressure, have been attempted to try to recruit atelectatic lung. Recruitment maneuvers in patients with severe ALI/ARDS can be dangerous, as venous return may decrease significantly, causing hypotension, and alveolar ventilation is decreased, which may result in respiratory acidosis. In general, recruitment maneuvers have been found to only transiently increase oxygenation, without changing outcome. At this time, routine use of recruitment maneuvers cannot be recommended.

**Prone Mechanical Ventilation**

Patients with ALI/ARDS develop dependent atelectasis. Atelectatic areas of the lung cause intrapulmonary shunting and ventilation/perfusion mismatching, both of which lead to hypoxemia. In addition, dependent atelectasis results in regional compliance differences, and may contribute to volutrauma in normal lung segments. Computerized tomography of patients with ARDS clearly shows this relationship. If these changes are gravity dependent, then it is logical to assume that if the patient is turned from the supine to the prone position, that they would dissipate, resulting in improved gas exchange. Although there are other, theoretical advantages to prone positioning such as higher functional residual capacity and improved secretion drainage, it is improved oxygenation that has spurred the enthusiasm for this technique. There are substantial logistical problems to prone positioning, including accidental extubation, line or chest tube removal, and padding of the facial area.Gattinoni et al performed a multicenter, prospective, randomized trial in patients with ALI/ARDS comparing supine positioning to placing patients in the prone position for six or more hours per day for 10 days. They studied 304 patients, and did not identify a difference in mortality. They did find a significant improvement in oxygenation, but as the ARDSnet trial taught us, this is not a valid outcome parameter in patients with ALI/ARDS. Although there may be specific patients that may benefit from this technique, routine use of prone positioning is not supported by the data. An alternative to prone positioning is the use of beds that can provide significant side-to-side rotation. These beds have not been tested in a randomized, prospective trial.

**High Frequency Ventilation**

High frequency ventilation (HFV) employs small tidal volumes and a high respiratory rate in an effort to minimize inspiratory pressures in the injured lung. Whereas traditional mechanical ventilation uses respiratory rates from approximately 2-40 breaths/minute, HFV uses rates up to 100 breaths/minute, and high frequency oscillation (HFO) uses rates as high as 2,400 breaths/minute. In general, HFV results in increased mean airway pressure without increasing PIP, but also is less efficacious for CO2 removal than traditional ventilation. This technique is frequently used in neonatal respiratory failure, but remains controversial. The trials that have been conducted in adults, similar to those trials of prone positioning, have been able to demonstrate improved oxygenation, but not reduced mortality. HFV has recently been reviewed, including its role in lung protection during mechanical ventilation.

**Dual Control Modes of Mechanical Ventilation**

Traditional mechanical ventilation tends to be volume-cycled (synchronized intermittent mandatory ventilation and assist control ventilation) with the thought that critically ill patients should have guaranteed minute ventilation. Pressure limited mechanical ventilation may limit inspiratory pressures, but does so at the expense of guaranteed minute ventilation; a rapid decrease in lung compliance may result in significant alveolar hypoventilation. Dual control modes of mechanical ventilation try to exploit the advantages of both these methods: limiting inspiratory pressure while ensuring adequate minute ventilation. A theoretical advantage is improved patient comfort, as the ventilator can respond instantaneously to increased flow demand. There are a large number of proprietary dual control modes, depending on the ventilator manufacturer. This fact alone dampens the enthusiasm for this technique, as a clinician may not be familiar with the modes if multiple ventilators are used in an ICU.
Microprocessor technology now allows a ventilator to change pressure and flow variables both between breaths (Pressure Regulated Volume Control {PRVC}) and within a breath (Adaptive Support Ventilation).

For example, with PRVC (Siemens), the clinician chooses a minimum respiratory rate, target tidal volume, and upper pressure limit. The ventilator continuously monitors tidal volume and minute ventilation, and if minute ventilation falls behind, can increase inspiratory pressures up to the limit in order to “catch up”. None of the dual control modes have been demonstrated to improve outcomes, including mortality, length of ICU stay, or duration of mechanical ventilation.

Continuous Positive Airway Pressure for Postoperative Respiratory Failure

Hypoxemia is a common postoperative complication in patients undergoing abdominal surgery, and a portion of those patients will go on to requiring reintubation. Postoperative care of these patients should include lung re-expansion therapies such as incentive spirometry and intermittent positive pressure breathing (IPPB). A recent prospective trial examined the efficacy of continuous positive airway pressure (CPAP) in preventing postoperative hypoxemia and decreasing the incidence of reintubation in patients who had undergone abdominal surgery. Squadron et al compared 6 hours of oxygen therapy by facemask to CPAP at 7 cmH2O in 209 patients. They measured the need for intubation in the first seven days after surgery. Ten percent of the control group required intubation, whereas only 1% of the CPAP group required intubation. The event rates for pneumonia, infection, and sepsis were lower in the CPAP group as well. Criticisms of the study include the large number of patients excluded (1332 patients were enrolled, but only 209 were randomized), and the fact that the control group received only supplemental oxygen, without other postoperative respiratory therapy maneuvers. A recent meta-analysis of nine clinical trials has confirmed the benefit of CPAP in preventing postoperative pulmonary complications.

Recruitment Maneuvers in ARDS

ARDS is characterized by alveolar flooding and abnormalities in surfactant function. This results in alveolar collapse, ventilation-perfusion mismatching, and hypoxemia. Use of positive end-expiratory pressure (PEEP) will recruit collapsed lung units, allowing them to participate in oxygenation and ventilation. One solution to recruiting collapsed lung is to turn patients prone, whereas another technique is to use high levels of CPAP repeated at regular intervals. This results in opening of collapsed alveoli, improved oxygenation, and decreased airway pressures. There is little data, however, that these benefits are more than transient. Gattinoni et al studied the amount of potentially recruitable lung using CT scanning in patients with ARDS. Using airway pressures of 5, 15, and 45 cmH2O, they found wide variability in the amount of recruitable lung. They also found that the proportion of potentially recruitable lung was correlated with the physiologic response to PEEP. This suggests that PEEP should be chosen based on the potential recruitability of the lung parenchyma.

How Much PEEP Should We Use?

Although it has been demonstrated that PEEP can improve oxygenation, as can be seen from the literature on prone mechanical ventilation and recruitment maneuvers that transient improvement in oxygenation doesn’t mean that patients will show improved outcomes. A recent meta-analysis examined 2299 patients in 3 large trials comparing lower to higher PEEP levels in patients with ALI and ARDS. Overall, there was no improvement in outcome in the patients with ALI treated with higher PEEP. However, higher PEEP levels were associated with improved survival in the subset of patients with ARDS. There were no differences in vasopressor use or in the incidence of pneumothorax.
Performance of Newer Generation Anesthesia Ventilators

Earlier models of anesthesia ventilators suffered from poor performance when ventilation demands were high. The combination of poor lung compliance and high minute ventilation demand require that the ventilator deliver effective minute ventilation in order to avoid hypercapnea and respiratory acidosis. Maximum ventilatory capacity can be calculated for a specific ventilator by using the mean inspiratory flow versus airway pressure curves, and the maximum allowable inspiratory duty cycle (the ratio of inspiratory time to total breathing cycle duration). This approach permits prediction of ventilator performance for patients with high minute ventilation requirements. With low lung compliance, inspiratory flow rate falls as airway pressure increases. The clinical implication of this finding is that alveolar ventilation will fall. Recognition of the limitations of anesthesia ventilators is critical as patients that are acidic may not tolerate the respiratory acidosis associated with hypoventilation. In addition, the ventilator tubing used with ICU ventilators tends to be much less compliant than the tubing used with anesthesia ventilators. This means that in patients with high airway pressures, a larger fraction of the tidal volume will be “wasted” in tubing expansion when anesthesia ventilator tubing is used. When a patient with severe respiratory failure requires mechanical ventilation in the operating room, consider using an ICU ventilator for transport and in the OR if peak inspiratory pressures are greater than 50 cmH\textsubscript{2}O and minute ventilation is greater than 15 liters/minute.

References

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Disclosure

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