

Critical Care Medicine Section (CCMS)

# Mechanical Ventilation in the Difficult Patient and Ventilator Cycling

Joseph Levin, MD

Ashika Jain, MD RDMS FAAEM FACEP, President-Elect CCMS



Mechanical ventilation, conventionally known as invasive positive pressure ventilation, is employed to partially or fully replace spontaneous breathing in patients with impaired ability to adequately oxygenate or ventilate, or both. In severe pulmonary disease or neurologic processes affecting spontaneous respiration, invasive ventilation via the insertion of an endotracheal tube is used to deliver this

respiratory support. Modification of the ventilator's delivery of air volume, pressure, partial pressure of oxygen, flow, and rate of delivery should be tailored to the specific pathology afflicting the patient. These variables are sequenced to optimize gas exchange in the lungs while minimizing pulmonary trauma the ventilator itself may cause. Striking a balance between these two tenets of therapy is often challenging due to pathology that alter lung mechanics.

There is a growing body of evidence that protective lung ventilation can be beneficial in non-diseased lungs.<sup>1</sup> This paradigm is defined by low lung volumes (often 4-8ml/kg ideal body weight) and permissive arterial hypercapnea. Prior to this strategy, earlier models of ventilation often employed tidal volumes of 10 to 15ml/kg of body mass, which were required to yield comparable partial pressures of arterial carbon dioxide and physiologic pH values when compared to spontaneously breathing individuals, but subjected patients' lungs to substantial volutrauma and barotrauma known as ventilator-associated lung injury (VALI).<sup>2,3</sup> Lung protective strategy can render a mechanically ventilated patient with respiratory acidosis and decreased arterial oxygenation, yet is associated with lower risk of developing acute respiratory distress syndrome (ARDS), pulmonary infection, and atelectasis in previously uninjured lungs in surgical and ICU patients.<sup>1,4</sup>

In patients with ARDS, protective lung ventilation has become the standard of care, however investigators have challenged this notion.<sup>1,5</sup> ARDS is a constellation of lung pathologies that arise from an intense inflammatory state and increased vascular permeability. Due to decreased lung compliance arising from atelectasis and proteinaceous deposits, recruitment of alveoli to allow oxygenation is impaired. Assist control (pressure control or volume control) with low tidal volumes (6ml/cc of ideal body weight), minimum FiO<sub>2</sub>, and permissive hypercapnea, can recruit vulnerable alveoli and reduce distending volutrauma. The use of formal alveolar recruitment strategies including high peak end expiratory pressure (PEEP) and open lung ventilation is not routinely recommended due to lack of standardization and unclear benefits, in addition to potential harm associated with prolonged distending pressures.<sup>6,7</sup> Studies have shown, however, that employing a low tidal volume ventilation strategy is associated with lower mortality in patients with ARDS. Since the landmark ARDSNET ARMA trial showed lower mortality associated with low tidal volumes, subsequent investigations have corroborated the benefit of lung protective strategy in ARDS.<sup>2,8,9</sup> Airway pressure release

ventilation (APRV) has challenged the ARDSnet protocol in its methodology. Nevertheless, both support open lung ventilation.

Low tidal volume is only one part of the equation. In fact it is a compensatory parameter in order to achieve open lung ventilation, which is mostly achieved by higher PEEPs. When thinking about ventilation pressures, and mechanical ventilator support, it is crucial to understand the physiology first, i.e., peak inspiratory pressure (PIP), plateau pressure and PEEP. PIP is the maximum pressure in the lungs during inhalation. Increased airway resistance will increase the PIP. Plateau pressure is the pressure in the alveoli. It is measured when there is no airflow in the system, when inspiration is complete. Lung compliance will greatly affect the plateau pressure. In order to open the alveoli, ventilator pressure must be higher than the plateau pressure. PEEP is the airway pressure above atmospheric pressure at the end of exhalation by means of mechanical impedance. It can be created intrinsically, (pursing lips during exhalation) or extrinsically (dialed in by ventilator). PEEP mitigates alveolar collapse.

Whether you believe in ARDSNET or APRV, open lung ventilation is the key to prevent atelectrauma and barotrauma during this low compliance state. The ventilator will deliver the programmed amount of volume and pressure without regard for successful ventilation. Whether or not it was delivered to the alveoli is up to the parameters set. Without adequate pressure, the ventilator will continue to deliver inadequate breaths causing a spiral incomplete ventilation.

Other disease states affecting the lung at the level of the alveoli may benefit from low tidal volumes, as well. Acute pulmonary edema, pneumonia, sepsis, trauma, shock are proven risk factors for development of acute lung injury (ALI) and ARDS.<sup>10,11</sup> While data is limited to cohort studies and animal models, traditional ventilation strategies in critically ill subjects without previously injured lungs are associated with increased inflammatory markers in bronchial washings and are at higher risk for the development of ALI/ARDS, where utilization of lung protective measures demonstrates reduced mortality.<sup>10,12,13,14</sup>

During acute exacerbations of obstructive lung disease such as chronic obstructive pulmonary disease (COPD) or asthma, patients may require invasive mechanical ventilation as a result of respiratory distress refractory to medical therapy and noninvasive positive pressure ventilation. The goal of mechanical ventilation in these patients is to rest fatigued respiratory muscles, provide adequate oxygenation, and prevent dynamic hyperinflation from "air trapping." Due to increased airway resistance at the level of the large and medium airways, heightened peak airway pressures are expected. Even with non-elevated plateau pressures, however, there is speculation that due to heterogeneous obstruction patterns, high peak pressures may pose the risk for barotrauma to alveoli distal to less obstructed regions.<sup>15</sup>

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Mechanical ventilation strategies in patients with obstructive lung disease require a balance between airway pressures and inspiration-expiration ratio to maximize gas exchange. In patients with severe obstruction, presumed intrinsic PEEP) may lead to deterrence in applying extrinsic PEEP due to concern of worsening already increased lung volumes. In contrast, the application of external peep will allow for acceleration of expiratory phase of breathing and improved CO<sub>2</sub> unloading.<sup>15,16</sup> The key to successful ventilation in this case is a prolongation of the expiratory phase, often 1:3 to 1:5. This often comes at a sacrifice of minute ventilation required to normalize the pACO<sub>2</sub>. As with ARDS, a strategy of permissive hypercapnea can be employed which has been shown to be well tolerated by patients with obstructive lung disease and reduce ventilator associated lung injury.<sup>14,17</sup>

Assisted and supported mechanical ventilation is intended to share the work of breathing with the patient, serving to unload fatigued respiratory muscles, facilitate ventilation and oxygenation, and coincide with the patient's own efforts. Despite employing optimized ventilator strategies, however, undesired interactions between the patient and ventilator called dyssynchrony can lead to "imposed" respiratory muscle loads and impair gas exchange. These can present during any phase of the ventilator cycle: initiation, breath delivery, or the inflection point of inspiration/expiration. Most dyssynchrony occurs when the ventilator does not sense patient efforts leading to missed breaths or there is a process driving excessive triggering.<sup>18</sup> Absent triggers are largely due to inappropriately set negative pressure (or flow) trigger thresholds that exceed a patient's ability to generate necessary inspiratory force as a result of fatigue and illness. Elevated lung volumes at end-expiration, intrinsic PEEP, can also be prohibitive by increasing the amount of negative pressure required by the patient to overcome for ventilator triggering. Inappropriately high sensitivity thresholds can induce unintended triggering (autotriggering) of the ventilator, which can in turn result in patient tachypnea, barotrauma, worsening intrinsic PEEP, and increase sedation requirements. Noxious stimuli including pain, cardiac ischemia, foreign body sensation from the endotracheal tube, among others will also stimulate the respiratory center of the CNS to cause excessive triggering.<sup>19</sup> Perhaps most interestingly, there is suggestion that a controlled mechanical breath itself can stimulate a subsequent spontaneous breath, a process known as entrainment.<sup>20</sup>

Synchrony of the patient with the ventilator relies on the interplay between respiratory mechanics (loading patterns) and the central neural drive of respiration (controller).<sup>21</sup> Acute illness producing acidosis, hypoxemia, and increased metabolic demands all stimulate the controller to increase minute ventilation. An inability of the ventilator to match controller demand promotes ventilator dyssynchrony with ensuing imposed respiratory loads.<sup>16</sup>

Dyssynchrony during cycling phase arises from a mismatch between anticipated breath termination by the controller and the end of the delivered mechanical breath. A mechanical breath that ends after the neural inspiratory time will promote discomfort via initiation of expiratory muscle contraction. In contrast, an inspiratory time delivered by the ventilator that terminates before the controller inspiratory time prolongs muscle contraction of the diaphragm and accessory muscles driving imposed loading. These factors can both result in dynamic hyperinflation.<sup>19</sup>

Patient-ventilator interactions are complex and can be difficult to manage in situations where patients have significantly altered lung mechanics and capacity for gas exchange, as in diffuse lung disease such as ARDS or obstructive processes. Ventilator strategies must be employed that optimize gas exchange while minimizing iatrogenic harm from volutrauma, atelectrauma, and barotrauma. Understanding pulmonary pressures as they relate to the ventilator is crucial in achieving synchrony and adequate ventilation. Without the basic understanding of the relationship of physiology and ventilator mechanics, management of complex pulmonary processes prove to be intangible. Novel ventilation strategies that respond automatically to patient-ventilator feedback such as proportional assist ventilation (PAV) and neutrally adjusted ventilator assist (NAVA) may be the next step in addressing these complex interactions, but more research is needed.<sup>19,22</sup> ●

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