Integration of Pulmonary Mechanics in a Personalized Approach to Mechanical Ventilation

The concept of safe ventilation continues to evolve. Initially, a low tidal volume (V_T) strategy,[1] then an open lung strategy,[2,3] and now as mortality improvements have stagnated and promising targets such as PEEP and plateau pressure yield unsatisfactory and conflicting results,[4,5] a search for better, more integrative therapeutic targets has ensued. Whether in a patient without lung injury who is undergoing surgery or in the patient with severe ARDS and refractory hypoxemia, a successful mechanical ventilation strategy likely must use a personalized approach to impart the least amount of energy on the respiratory system to meet an individual's oxygenation and ventilation requirements-measuring respiratory mechanics at the bedside is required to accomplish this goal and to limit the likelihood of lung injury. A single time assessment of respiratory mechanics at the bedside provides a snapshot of the patient's condition while serial measurements trend clinical progress. Moreover, such temporal resolution serves to provide feedback to the practitioner as he or she makes each turn of the ventilator knob, assisting in the selection of better PEEP, V_T, or another parameter.[6]

Although an individual's respiratory system mechanics can be estimated through direct measurements at the bedside, assessing the elastic properties of the lung is more invasive in nature and requires specialized equipment and expertise to provide reliable data, and must be used frequently to maintain proficiency.[7,8] Because it is not usually feasible to place an esophageal balloon in every patient, interest has turned to surrogate markers to estimate lung distending pressure, stress, and strain, and to guide safe ventilation without the need for more-invasive and complicated testing. One such integrative parameter is airway driving pressure estimated as plateau pressure minus PEEP.[9,10] Another parameter, the stress index, uses the ventilator airway pressure (P_{aw})–time waveform readout under specific conditions to estimate respiratory system compliance.[11,12] Both parameters may help guide the setting of safe ventilation at the bedside, yet may also succumb to the same pitfalls related to their dependence on surrogates (plateau pressure for alveolar pressure, respiratory system vs lung mechanical properties measured by the stress index) and an understanding of appropriate patient selection or exclusion.[10,13]

SEE THE ORIGINAL STUDY ON PAGE 1094

In this issue of RESPIRATORY CARE, Sun et al[14] describe a method to assess the stress index at the bedside by visually inspecting the P_{aw}-time curve of patients without ARDS and who are on mechanical ventilation while in a neurosurgical ICU. They concluded that this rapid visualization of the stress index at the bedside "will facilitate its implementation in clinical practice to personalize mechanical ventilation,"[14] but did not comment on several key limitations of the stress index also applicable to airway driving pressure and related to the inability to separate the compliance of the respiratory system and the lung. We discussed the challenging clinical implications of the stress index, which go beyond the simplification of its measure and incorporates several factors that are essential when rendering an interpretation of this parameter in the clinical setting, especially in patients with ARDS who are critically ill.

The stress index and its technique evolved in the 1990s after first being described by Ranieri et al[11] and gained widespread acceptance in the literature in the early 2000s.[12] Initial efforts were directed at predicting the effects of PEEP on static (airway occlusion) and dynamic (constant flow) pressure–volume curves,[11] which showed a good correlation between these coefficients at several clinically relevant levels of PEEP. In addition, these investigators demonstrated that the shape of the P_{aw}-time curve during constant flow inflation corresponds to radiologic evidence of tidal recruitment or tidal hyperinflation in an experimental model of acute lung injury.[12] As such, a downward concavity shape with a stress index of <0.9, a linear shape with a stress index of 0.9–1.1, and an upward concavity shape with a stress index of >1.1 correlated with a position near or below the lower inflection point, between the upper and lower inflection points (ie, the sweet spot), or

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near or above the upper inflection point of the pressure-volume curves, respectively. From these inferences, ventilator changes, such as increasing PEEP for a negative stress index (underrecruited) or reducing V_T for a positive stress index (overdistended), might be indicated.[11,12]

The importance of proper technique (volume control with a slow and constant flow, in the absence of respiratory efforts) to reliably obtain and interpret the stress index cannot be overemphasized.[11,12] Sun et al[14] showed that each subject's stress index maneuver seemed to have been performed at a different PEEP level and V_T (5–10 mL/kg of predicted body weight), and a relatively high flow. This is concerning in that the increased flow will contribute to the total P_{aw} through the flow resistance portion of the equation of motion. Because the stress index is a measure of total P_{aw} (which represents the total pressure applied across the respiratory system) over time, this elevated and varying flow from patient to patient likely will alter the P_{aw}-time curve and impair the interpretation and clinical applicability of the stress index. Moreover, regardless of the method used to estimate the stress index, a personalized approach to mechanical ventilation implies integrating all the potential factors that contribute to ventilator-induced lung injury (VILI), which is different than simply interpreting the upper inflection region of the P_{aw}-time curve (the stress index) or its surrogate (30 cm H_2O airway plateau pressure) as suggested by the authors.[14]

As the debate continues regarding the relative importance among the mechanical determinants of VILI, the concept of a personalized approach to mechanical ventilation continues to evolve. Recent interest has arisen in airway driving pressure (DP_{AW}, the quotient of V_T, and respiratory system compliance) which could serve as a direct and easily measured marker for VILI risk[9,10]; however, the correspondence between DP_{AW} and transpulmonary driving pressure (DP_{TP}) also known as "true driving pressure", which is the quotient of V_T and lung compliance, may be influenced by changes in chest wall compliance and by the lung inhomogeneities in patients with ARDS.[10] When compared with the stress index, both DP_{AW} and DP_{TP} provide a more comprehensive approach by integrating PEEP, V_T, respiratory system compliance and lung compliance.[9,10] Many other factors may also influence clinical decisions at the bedside, for example, body positioning affects the configuration and dynamic properties of the chest wall, and, therefore, may influence decisions made to increase or decrease ventilating pressures and V_T based on parameter such as the stress index, airway driving pressure, or transpulmonary driving pressure.[15,16] Moreover, the vascular side has been de-emphasized as a potential contributor to VILI, despite revealing experimental data that demonstrate that raising precapillary vascular pressure intensifies VILI,[17] and that large vascular pressure gradients promote West zone 2 conditions in which microvascular waterfalls (vascular pressure gradients) predispose the vascular endothelium to be injured by poorly tolerated shear stress and applied energy,[17,18,] all factors well beyond the evaluative scope of the stress index.

Most recently, Gattinoni et al[19] postulated the mechanical power as the unifying variable that integrates all the machine-derived factors that contribute to VILI, in which V_T, pressures, flow, and breathing frequency are considered components of the energy load applied to the respiratory system per units of time, the proximate mechanical stimulus for VILI.[19] The power theory of VILI is that the energy component of primary relevance is the driving pressure (airway and transpulmonary), while also including the energy components related to dissipation in overcoming resistance and the static element represented by PEEP.[19,20]

In our institution, we have incorporated real-time stress index visualization at the bedside in challenging ARDS cases in which patients are already heavily sedated and/or paralyzed, with no spontaneous effort. We usually use a flow of <10 L/min and alter the zoom or axes on the ventilator pressure-time waveform to visualize one breath per screen. The slower flow (and longer waveform) provides an easier analysis, accentuates the shape, and also minimizes or eliminates the flow resistance component of the equation of motion. Several mechanical ventilator vendors incorporate a tool to automate measurement of the stress index on the screen, which provides not only a visual waveform but also a numeric readout to simplify what the investigators of the current study propose.[14]

Mechanical ventilation is one of the cornerstones, and a defining intervention, in critical care medicine. A personalized approach to mechanical ventilation demands a deep understanding of cardiac and respiratory physiology as well as mastery of the pathophysiologic principles that inform and guide bedside decisions. The results of the present study by Sun et al[14] do not address the fundamental principle of the internal distributions of gas volume, collapse, ventilation, and tidal recruitment, even when commonly measured global indicators of transpulmonary pressure and functional residual capacity provide little indication of these important changes.[21]

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