Monitoring During Mechanical Ventilation

Craig R Rackley

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Mechanical ventilation is an indispensable form of life support for patients undergoing general anesthesia or experiencing respiratory failure in the setting of critical illness. These patients are at risk for a number of complications related to both their underlying disease states and the mechanical ventilation itself. Intensive monitoring is required to identify early signs of clinical worsening and to minimize the risk of iatrogenic harm. Pulse oximetry and capnography are used to ensure that appropriate oxygenation and ventilation are achieved and maintained. Assessments of driving pressure, transpulmonary pressure, and the pressure-volume loop are performed to ensure that adequate PEEP is applied and excess distending pressure is minimized. Finally, monitoring and frequent adjustment of airway cuff pressures is performed to minimize the risk of airway injury and ventilator-associated pneumonia. We will discuss monitoring during mechanical ventilation with a focus on the accuracy, ease of use, and effectiveness in preventing harm for each of these monitoring modalities. *Key words: mechanical ventilation; pulse oximetry; capnography; driving pressure; transpulmonary pressure; pressure-volume curve; airway cuff pressure.* [Respir Care 2020;65(6):832–846. © 2020 Daedalus Enterprises]

Introduction

Mechanical ventilation is a commonly required mode of support during general anesthesia or in the ICU. It is estimated that > 300 million surgeries are performed each year, with a large percentage of these happening while the patient is receiving mechanical ventilation.[1] In the United States alone, > 4 million patients are admitted to an ICU each year, and at any given time, approximately 40% of those patients are receiving invasive mechanical ventilation.[2,3] With the enormous global volume of mechanically ventilated patients, it is important to consider what monitoring should be used during mechanical ventilation to minimize harm to patients.

When considering monitoring in the mechanically ventilated patient, 3 questions must be considered: Why are we monitoring, how good are the tools we use to monitor, and does monitoring lead to a change in management that impacts outcomes? The general goal of any patient-monitoring system or parameter is to identify abnormalities or early warning

Dr Rackely is affiliated with the Department of Medicine, Duke University Medical Center, Durham, North Carolina.

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Correspondence: Craig R Rackley MD, Division of Pulmonary, Allergy, and Critical Care Medicine, Box 102355, Duke University Medical Center, Durham, NC 27710. E-mail: craig.rackley@duke.edu.

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signs and to mitigate patient harm. This is especially important when it comes to mechanical ventilation because almost every aspect of mechanical ventilation has the potential to cause patient harm. In fact, in the largest clinical trials that indicate a benefit in mechanically ventilated patients, the benefit is due to reduced harm from mechanical ventilation.[4,5] This review will discuss the utility of monitoring pulse oximetry, capnography, driving pressure, transpulmonary pressure, pressure-volume curves, and airway cuff pressures.

Should Pulse Oximetry Be Used With Every Mechanically Ventilated Patient?

Pulse oximetry is an undisputed standard in clinical monitoring of mechanically ventilated patients, and few providers could imagine caring for a critically ill patient requiring mechanical ventilation without it. Pulse oximetry combines a spectrometer to detect hypoxemia with a plethysmograph, which may be utilized for the diagnosis, monitoring, and follow-up of cardiovascular diseases.[6] The use of pulse oximetry can reduce the need for invasive monitoring and frequent arterial blood gas measurements to assess oxygenation status.[7]

The spectrometer is the key function of pulse oximetry that is universally utilized for continuously and noninvasively monitoring a patient's arterial oxygen saturation (S_{pO_2}) as opposed to oxygen saturation measured on an arterial blood sample (S_{aO_2}). In the absence of more severe hypoxemia (ie, $S_{pO_2} < 90\%$) and states of poor perfusion, S_{pO_2} generally approximates S_{aO_2} with good accuracy.[8] Pulse oximetry is used widely to assess S_{pO_2} in the out-patient clinic, cardiopulmonary rehab, throughout the in-patient setting, and even at home with small personal-use devices. In the largest study available, which included > 20,000 non-intubated, postoperative subjects, the routine use of pulse oximetry reduced the incidence of hypoxemia through early detection of desaturation.[9] Interestingly, despite reducing hypoxemic events, pulse oximetry had no impact on transfer to the ICU, mortality, or any other meaningful outcome.[9]

In patients with respiratory failure, the general goal of pulse oximetry monitoring is to ensure that adequate supplemental oxygen is provided to prevent hypoxia and its negative sequelae. In chronically hypoxemic patients, providing supplemental oxygen improves overall survival.[10,11] In the acute setting, mechanically ventilated patients with ARDS are at risk for long-term neuropsychological impairment, which may be worsened by hypoxemia. In a cohort of ARDS survivors, those with lower P_{aO_2} while receiving mechanical ventilation had increased risk of cognitive and psychiatric impairment 12 months after discharge, even though their average P_{aO_2} was 71 mm Hg.[12]

Conversely, patients receiving supplemental oxygen with high S_{pO_2} levels can have significant hyperoxia that may go unrecognized without arterial sampling of P_{aO_2}. This is important to recognize because providing supplemental oxygen to patients to maintain S_{pO_2} 98–100% also has the potential to worsen outcomes, even when only maintained for short periods of time. This was exemplified in a large multi-center observational study that explored the effect of postresuscitation hyperoxia (defined as $P_{aO_2} \geq$ 300 mm Hg) in cardiac arrest subjects. The investigators reported that hyperoxia was independently associated with a 24% increase in risk of death for each 100 mm Hg increase in P_{aO_2}.[13] Similarly, a randomized trial reported significantly less mortality among critically ill subjects assigned to a conservative strategy of oxygen therapy (P_{aO_2} 70–100 mm Hg or S_{pO_2} 94–98%) compared to those assigned to a liberal oxygen therapy strategy (P_{aO_2} up to 150 mm Hg or S_{pO_2} 97–100%).[14]

In patients with ARDS, another use of S_{pO_2} is to calculate the S_{pO_2}/F_{IO_2}, which has been proposed as a noninvasive alternative or surrogate for the standard P_{aO_2}/F_{IO_2}. In a large validated cohort of subjects with ARDS, the relationship between S_{pO_2}/F_{IO_2} and P_{aO_2}/F_{IO_2} was described as $S_{pO_2}/F_{IO_2} = 64 + 0.84 \times (P_{aO_2}/F_{IO_2})$ (Figure 1).

The S_{pO_2}/F_{IO_2} threshold value of 235 had an 85% sensitivity with 85% specificity for detecting $P_{aO_2}/F_{IO_2} \leq 200$. The S_{pO_2}/F_{IO_2} threshold value of 315 had a 91% sensitivity with 56% specificity for detecting $P_{aO_2}/F_{IO_2} \leq 300$.[15] It is important to note that S_{pO_2} had to be $\leq 97\%$ for the measurement of S_{pO_2}/F_{IO_2}. At S_{pO_2} levels > 97%, it is not possible to estimate P_{aO_2} accurately, as outlined above.

Whereas most respiratory therapists and physicians utilize pulse oximetry for its spectrometer function, information may also be gleaned from the plethysmography waveforms. Each wave on the tracing represents the volume of blood versus time curve measured during one cardiac cycle.[6] Using a finger pulse oximeter, changes in the waveform amplitude and notch position can help when assessing volume status and vascular tone.[16] Although this is potentially a promising adjunct in the assessment of a patient's cardiovascular status, assessment of plethysmography waveforms can be highly subjective, and data that support improvement in clinically relevant outcomes are lacking.

In summary, pulse oximetry combines a spectrometer with a plethysmograph. The main reasons to use the spectrometer function are to identify early warning signs of changes in respiratory status and to ensure that appropriate supplemental oxygen is provided to hypoxic patients. Although it is less clear whether continuous pulse oximetry truly improves outcomes in the postoperative period, it provides an accurate and noninvasive means to monitor patients who may have rapidly changing clinical conditions due to respiratory failure. Targeting S_{pO_2} 94–98% in most patients requiring mechanical ventilation best balances the risks of hypoxemia and hyperoxia. Plethysmography analysis may provide some insight into cardiovascular status,

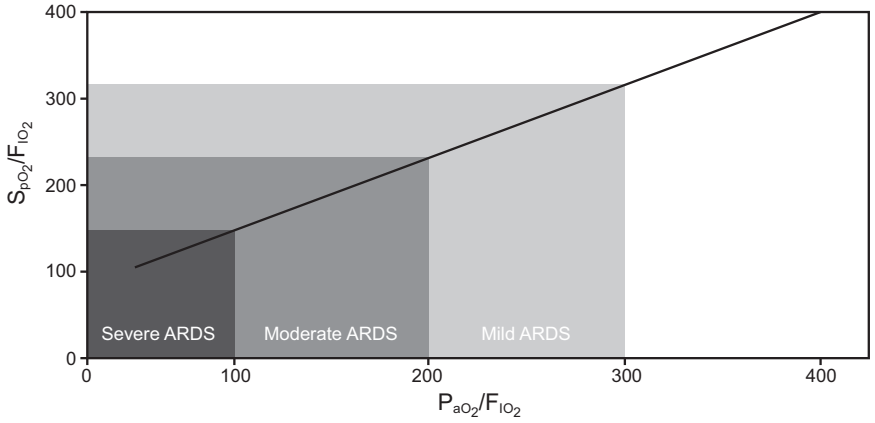

Fig. 1. S_{pO_2}/F_{IO_2} can be used to approximate P_{aO_2}/F_{IO_2}, providing a noninvasive means of identifying ARDS and quantifying its severity.

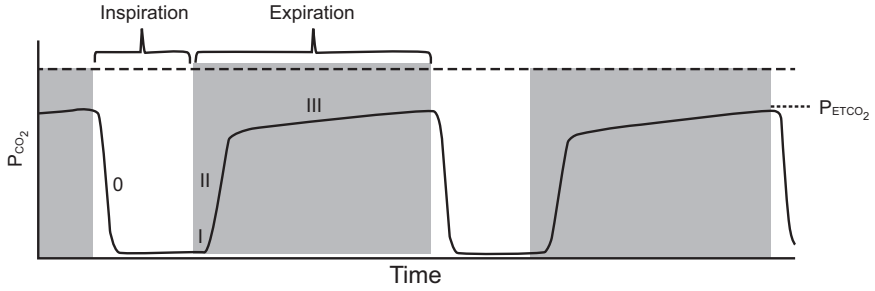

Fig. 2. Time capnography represents the concentration of CO_2 in the air passing the end of the endotracheal tube during ventilation cycles. The phases of ventilation include 0 (representing inspired P_{CO_2}), I (representing airway P_{CO_2}), II (representing airway-alveolar interface P_{CO_2}), and III (representing alveolar P_{CO_2}). The end-tidal carbon dioxide pressure (P_{ETCO_2}) represents P_{CO_2} at the end of expiration.

and it may be considered as an additional data point when managing acutely critically ill patients.

Should Capnography Be Used With Every Mechanically Ventilated Patient?

Unfortunately, S_{pO_2} does not provide necessary information about ventilation and CO_2 removal. This is important because poor ventilation and hypercarbia can lead to respiratory acidosis and cardiovascular collapse that may not be detected with pulse oximetry until very late in supplemental oxygen therapy. Capnography provides a measurement and graphical display of CO_2 concentration in exhaled gas in relation to time (ie, time capnography) or volume (ie, volume capnography). The most commonly used measurement is end-tidal CO_2 pressure (P_{ETCO_2}), which is frequently used to confirm proper endotracheal tube (ETT) placement and to monitor adequacy of ventilation. Use of capnography has been described to gauge the degree of ventilation/perfusion mismatch, to measure dead space (V_D), to quantify air-flow obstruction in asthma and COPD, to diagnose pulmonary embolism and distinguish it from exacerbations of COPD, to judge the adequacy of chest compressions in cardiac arrest and detect return of spontaneous circulation, to estimate changes in cardiac output, to predict fluid responsiveness, and to assist in metabolic assessment and nutritional needs.[17]

Capnometers most commonly utilize infrared light absorption or mass spectrometry to measure the partial pressure of CO_2 (P_{CO_2}) in a mixed gas.[9] Both methods are reliable and relatively accurate. Capnometers that are used in clinical practice use 2 different sampling techniques: sidestream or mainstream. A mainstream capnometer has an in-line airway adapter cuvette that is positioned close to the ETT. The cuvette incorporates an infrared light source and sensor that senses CO_2 absorption to measure mixed exhaled pressure of CO_2 (P_{ECO_2}). A sidestream capnometer uses a sampling line that is attached to a T-piece adapter at the airway opening, through which the instrument continuously aspirates tidal airway gas for analysis of CO_2.[18] A time capnogram is the method most commonly used in clinical practice and shows P_{CO_2} levels throughout the phases of respiration (Figure 2).

Volume-based capnography provides similar information as time capnography, but it plots P_{ECO_2} against exhaled tidal volume (V_T), which allows for more accurate calculation of V_D and CO_2 production. Volume-based capnography is

available in some commercial ventilators and as standalone monitors, but its higher cost and complexity currently preclude its widespread use in routine ICU monitoring.[17]

The limited evidence that is available to support the use of capnography comes from the periprocedural setting. Continuous time capnography is more effective than pulse oximetry at detecting postoperative respiratory depression, and the odds of recognizing postoperative respiratory depression were almost 6 times higher with capnography than with pulse oximetry.[19] Whether this early recognition of respiratory depression led to reduced rescue team activation, ICU transfers, or mortality was not examined.[19] Another meta-analysis of subjects undergoing procedural sedation reported that the addition of capnography to visual assessment and pulse oximetry was associated with a significant reduction in mild and severe desaturation, as well as in the need for assisted ventilation.[20]

The use of capnography can provide accurate measurements of P_{ECO_2} that are acceptable estimates of alveolar P_{CO_2} (P_{aCO_2}) in normal subjects; however, the difference between the P_{ECO_2} and P_{aCO_2} can be quite large in patients with diseased lungs. This difference is a reflection of the V_D fraction of ventilation: $V_D/V_T = (P_{aCO_2} - P_{ECO_2})/P_{aCO_2}$.

Physiologic pulmonary V_D is the fraction of the V_T that never comes in contact with a functioning alveolus. It is made up of the anatomic V_D, representing the conducting airways, and the alveolar V_D. Diseases that affect either the pulmonary parenchyma or the pulmonary vasculature can significantly increase the amount of alveolar V_D, either by reducing the number of alveoli receiving capillary blood flow, by decreasing the surface area of alveoli, or by some combination of these mechanisms. As the V_D fraction increases, the patient must concomitantly increase their minute ventilation to compensate for the reduced alveolar ventilation. In this scenario, exhaled alveolar CO_2 gets diluted when mixed with the very low concentration of V_D CO_2. Higher V_D fractions lead to a greater difference between P_{aCO_2} and P_{ECO_2}. Figure 3 shows a representation of the magnitude of impact from the V_D fraction (low P_{CO_2}) in patients with normal versus diseased lungs.

The use of time capnography may appear to be an attractive alternative to arterial blood gas analysis for adjusting mechanical ventilation settings because it is continuous and noninvasive. However, significant differences can be seen between P_{aCO_2} and P_{ETCO_2} in diseased lungs, as outlined above.[21] In fact, when ventilator settings are adjusted or patients are followed over time, changes in P_{ETCO2} and P_{aCO_2} are poorly correlated and may even go in opposite directions.[22,23] Therefore, P_{ETCO_2} should be used with caution as a surrogate for P_{aCO_2}.

In summary, time capnography has been used effectively to detect respiratory insufficiency during procedural sedation and in the postoperative period. In mechanically ventilated patients, it can ensure adequate ETT placement and provide an approximation of V_D (ie, V_D/V_T). Volumetric capnography is a promising tool that is based on physiological concepts, but further research is needed to define its diagnostic value and its potential utility for guiding therapeutic interventions.[24] When caring for patients with diseased lungs requiring mechanical ventilation, the inherent inaccuracies in P_{ETCO_2} as a surrogate for P_{aCO_2} must be taken into account when utilizing capnography as a monitoring tool.

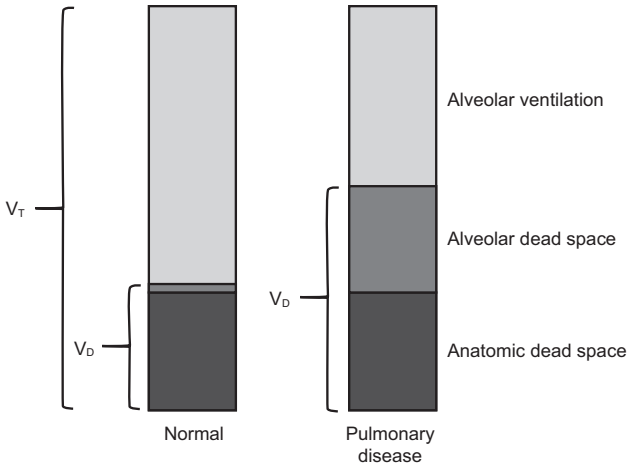

Fig. 3. In a normal patient, the dead space volume (V_D) makes up approximately 25% of normal tidal volume (V_T). In a patient with diseased lungs where the alveolar capillary interface is compromised (eg, emphysema, pulmonary embolism, ARDS, or pneumonia), the V_D fraction can be in excess of 50%.

Is Monitoring Driving Pressure Useful?

Driving pressure can be thought of as the pressure required to drive a given V_T into a patient's lungs. In fact, the relationship of V_T to static compliance of the respiratory system (C_{RS}) is the driving pressure: $\Delta P = V_T/C_{RS}$, which is also the difference between plateau pressure (P_{plat}) and PEEP (Figure 4).

Low V_T is generally believed to reduce ventilator-induced lung injury in both normal and diseased lungs.[25] How low V_T should be for any given patient is not known. In patients with ARDS, the damage to the lungs is quite heterogeneous, with some areas having dense consolidation and others appearing to have normal aeration. In adult patients with severe ARDS, the aerated portion of lung can be similar in volume to that of a 5–6-y-old child, giving rise to the concept of the baby lung.[26] This in turn has led to the notion that ventilator-induced lung injury is a regional phenomenon in which the delivered positive-pressure breath preferentially goes to healthier regions. A normal global V_T could thus produce excessive regional dynamic and static strain.[27] To

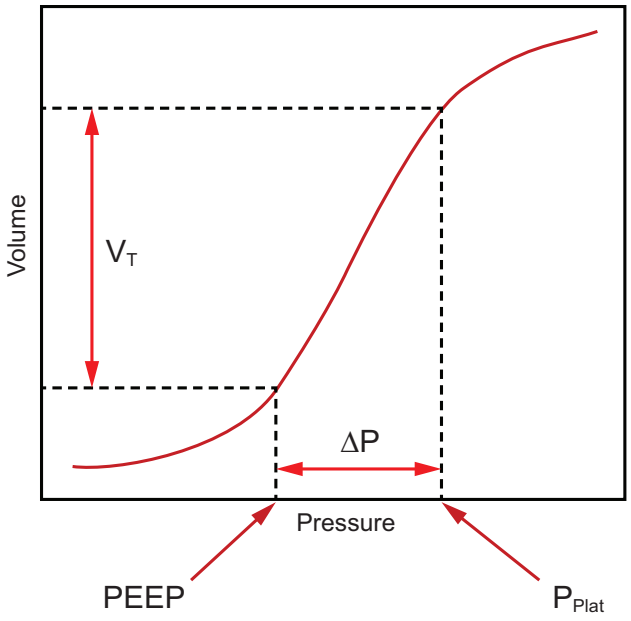

Fig. 4. The inspiratory limb of a pressure-volume curve. The driving pressure (ΔP) represents the difference between the inspiratory plateau pressure (P_{plat}) and PEEP. The static compliance of the lungs is represented by the tidal volume (V_T) achieved by a given ΔP.

account for this, it would make sense that targeting a V_T that matches the functioning aerated lung rather than an ideal normal lung may be the best approach.

One way to do this would be to visually or physiologically measure the functional lung size and set V_T as a fraction of functional lung.[28] A simpler strategy is to assume that lung compliance correlates with functional lung size and use it to scale the V_T. In a retrospective re-analysis of 3,562 subjects enrolled in several clinical trials evaluating mechanical ventilation management strategies, driving pressure was more strongly associated with survival than V_T, P_{plat}, or PEEP.[29] On the basis of this analysis, barotrauma and mortality both drastically increased at driving pressures exceeding 15 cm H_2O.[29] Another retrospective cohort of 778 subjects was analyzed and reported an incremental increased risk of death in subjects at driving pressures > 19 cm H_2O.[30] It would then follow that even a normal V_T of 6–8 mL/kg predicted body weight may be excessive if the driving pressure is excessive (eg, > 15–19 cm H_2O).[29-31]

In summary, driving pressure represents the stress applied to the lungs, and limiting driving pressure may allow for better matching of V_T to functional lung, especially in those with severely injured lungs. Although no prospective clinical trials have been performed to better solidify the relationship between driving pressure and mortality, the current body of evidence supports the routine monitoring of driving pressure to ensure patients are not being ventilated with excessive driving pressures.

Should Transpulmonary Pressure Be Monitored for All Mechanically Ventilated Patients?

When monitoring a patient on a mechanical ventilator, several pressure measurements are readily available to the clinician with each breath or by performing simple inspiratory or expiratory occlusion maneuvers. These pressures (ie, peak pressure, mean airway pressure, P_{plat}, and PEEP) are all measurements of airway pressure (P_{aw}). The alveolar distending pressure determines alveolar hyperinflation and alveolar collapse, and it is the most important pressure contributing to ventilator-induced lung injury. Esophageal pressure (P_{es}) must be measured to calculate the alveolar distending pressure. The question to be explored in this section is whether P_{aw} is an adequate surrogate for alveolar distending pressure, or should more direct measurements be employed in certain patients.

The distending pressure across the lung during tidal inhalation is the transpulmonary pressure (P_L). P_L incorporates the pressure difference across the lungs (both airways and alveoli) in the presence of flow. In the absence of flow (eg, during an end-inspiratory or end-expiratory occlusion maneuver), the P_{aw} measured by the ventilator is equal to the pressure inside the alveoli. P_L is calculated as the difference between P_{aw} and pleural pressure. P_L allows us to distinguish the pressure delivered to the lung from the one acting to simply move the chest wall and abdomen. Airway P_{plat}, measured during end-inspiratory or end-expiratory occlusion maneuvers, is the amount of pressure required to achieve a given lung inflation and to lift the chest wall and abdomen. Differences between airway P_{plat} and transpulmonary P_{plat} are most pronounced in obese patients and those with restrictive chest wall or pleural space disorders.[32] P_{es} is a proxy for pleural pressure and P_{es} measurements allow for the approximation of changes in pleural pressure ($P_L = P_{aw}$ − pleural pressure, or $P_L = P_{aw} - P_{es}$).[33]

Pleural pressure is approximated by measuring P_{es} using a pressure-transducing esophageal catheter with an air-filled balloon near the tip inserted either orally or nasally. In the supine position, P_{es} is thought to approximate the pressure at a mid-level of the pressure gradient from the nondependent to the dependent zones of the lung. The magnitude of P_{es}, either positive or negative, determines the difference between the P_{aw} and P_L. This concept can be illustrated by considering an obese patient with ARDS. An actively breathing patient may have significant respiratory drive and effort, thus generating high V_T with a low P_{aw}. Once this patient has been sedated and paralyzed, thus taking away the respiratory effort, the V_T plummets without any change in ventilator support. In the active breathing state, the patient is lifting the chest and abdomen and is also generating

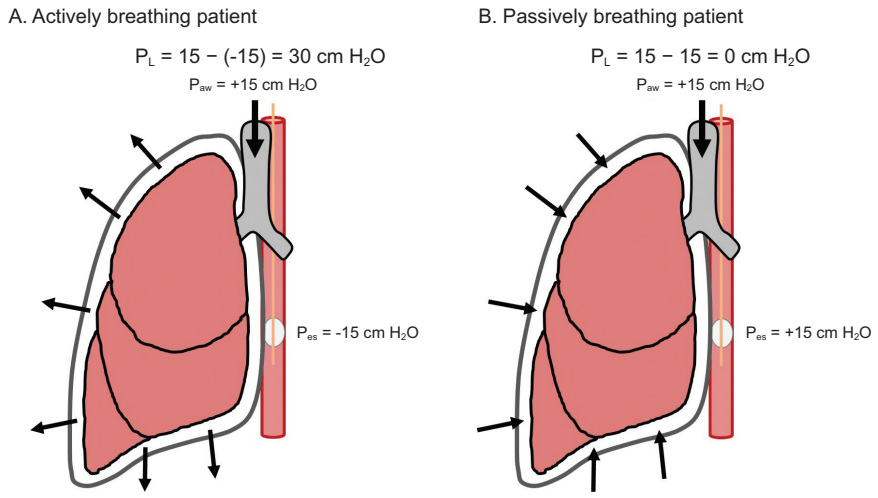

Fig. 5. Transpulmonary pressure (P_L) = airway pressure (P_{aw}) – esophageal pressure (P_{es}). (A) Example of an actively breathing patient generating $P_{es} = -15$ cm H_2O on top of $P_{aw} = 15$ cm H_2O. This leads to $P_L = 30$ cm H_2O. (B) Example of a passively breathing patient: $P_{es} = 15$ cm H_2O and effectively cancels out P_{aw}, leading to $P_L = 0$ cm H_2O.

negative pleural pressure. Once the patient has been paralyzed and is now passively breathing, the same P_{aw} generates a much smaller V_T (Figure 5).

The use of a pressure-transducing esophageal catheter to measure P_{es} has been most extensively studied in ARDS to set PEEP levels with the goal of achieving optimum alveolar recruitment while preventing collapse or overdistention. Because of reduced chest wall compliance, edema, or abdominal distention, P_{es} is often elevated in patients with ARDS. P_L can be negative at end-expiration at lower levels of PEEP, indicating that the pressure across the alveoli is negative and leading to alveolar collapse. For this reason, it has been proposed to set the end-expiratory pressure to a positive value of P_L. In addition, limiting end-inspiratory P_L to 20–25 cm H_2O appears to minimize alveolar overdistention.[34]

The usefulness of P_{es}-guided ventilator management in ARDS was evaluated in the Esophageal Pressure–Directed Ventilation (EPVent) study.[35] In this single-center, randomized controlled trial, the investigators compared mechanical ventilation guided by P_{es} measurements (ie, the experimental arm) with ventilation based on the ARDSNetwork protocol (ie, the control arm).[4] Subjects in the control arm had PEEP adjusted based on the ARDSNetwork lower PEEP table guided by the subjects P_{aO_2} and inspired F_{IO_2}.[36] In the experimental arm, PEEP levels were set to achieve an end-expiration P_L between 0 and 10 cm H_2O, according to a sliding scale based on P_{aO_2}/F_{IO_2}. At 72 h, PEEP was significantly higher in the experimental arm than in the control arm. The study was terminated early, after enrolling 61 subjects, due to an overwhelming improvement in oxygenation with the P_{es} strategy compared to the control arm, which was the primary end point of the study. C_{RS} was also significantly improved in the P_{es} group, probably as a consequence of improved recruitment. The trial was not sufficiently powered to detect a difference in mortality, but there was a trend toward reduced 28-d mortality (17% vs 35%, $P = .055$).

Subsequently, a meta-analysis comparing higher versus lower PEEP strategies demonstrated a survival benefit to higher levels of PEEP in subjects with moderate to severe ARDS having $P_{aO_2}/F_{IO_2} \leq 200$.[37] The baseline P_{aO_2}/F_{IO_2} in both arms of the EPVent study was approximately 145.[35] This would indicate that the best comparison to P_{es}-directed PEEP adjustment in patients with moderate to severe ARDS would be the ARDSNetwork higher PEEP table.[36] Therefore, the follow-up study EPVent-2 study was performed. This study was designed similarly to the EPVent study, but it aimed to compare a P_{es}-guided PEEP-titration strategy with one that followed the ARDSNetwork higher PEEP table. This was a larger, multi-center trial, and it revealed no difference in PEEP level following enrollment, survival, or days free from mechanical ventilation. Interestingly, 12% of the subjects in the P_{es}-guided group had PEEP $>$ 24 cm H_2O after enrollment compared to none in the control arm, and the P_{es} group was less likely to receive rescue therapies (eg, inhaled pulmonary vasodilators, prone positioning, or extracorporeal membrane oxygenation) compared to the control arm.[38] Although the results of EPVent-2 do not support the routine use of P_{es}-guided PEEP titration, there may be some benefit to this strategy in patients who are persistently hypoxic despite PEEP levels of 24 cm H_2O.

Another area where P_{es} measurement can be used is in the assessment of respiratory muscle effort during spontaneous breathing to improve identification of a asynchrony.

Patient–ventilator asynchrony is a frequent problem encountered in mechanically ventilated patients and can lead to excess patient work of breathing. Patients with asynchrony have more prolonged duration of mechanical ventilation and may receive excessive levels of ventilator support.[39] Furthermore, clinicians are poor at identifying patient–ventilator asynchrony with visual inspection of ventilator waveforms alone.[40] Excess spontaneous effort during positive-pressure ventilation can lead to significantly negative pleural pressure, and it is becoming clearer that this increases the risk of self-induced lung injury.[41,42] Vigorous inspiratory efforts can have other negative effects as well.[43] The accompanying increased diaphragm stimulation with vigorous efforts, especially in the presence of patient-ventilator asynchrony, can lead to respiratory muscle fatigue and injury.

In summary, estimating pleural pressure by measuring P_{es} can allow for calculation of P_L. Understanding P_L can potentially enable a clinician to titrate PEEP appropriately in patients with ARDS to prevent alveolar collapse and also to assist in identification of patient–ventilator asynchrony and excess patient work of breathing. Although these potential benefits are theoretically attractive, available evidence does not support routine use of P_{es} measurement to guide clinical care.

Is the Respiratory System Pressure-Volume Curve Useful?

The pressure-volume (P-V) curve describes the mechanical behavior of the respiratory system (lungs and chest wall) throughout inflation and deflation. Using a P-V curve may allow a clinician to identify the pressure below which the alveoli begin to collapse, as well as pressure and volume above which the alveoli begin to overdistend.[44] The P-V curve has been applied most extensively to patients with ARDS in hopes that it might allow clinicians to customize ventilator settings according to a patient's individual respiratory mechanics and thus protect the patient from ventilator-induced lung injury.

The P-V curve is measured in a quasi-static state during short periods of apnea or during very slow flow to allow for equilibration of pressure and volume. Accurate measurements require that the patient has no spontaneous effort during these maneuvers. Three main techniques for acquiring quasi-static P-V curves have been developed: the supersyringe method, the constant-flow method, and the multiple-occlusion method.[45] Each technique requires a degree of expertise, and it is important to note that many factors, such as oxygen consumption, temperature, and humidity, can impact P-V curve measurements.[46]

The point on the P-V curve of the respiratory system at which lung compliance begins to increase is known as the lower inflection point (Figure 6). It is thought that targeting a PEEP at or slightly above the lower inflection point will prevent cyclic alveolar collapse and reopening.[47] In fact, patients with respiratory failure tend to have a level of PEEP that achieves maximum static lung compliance, and this point also correlates with maximum oxygen transport and minimum V_D fraction.[48]

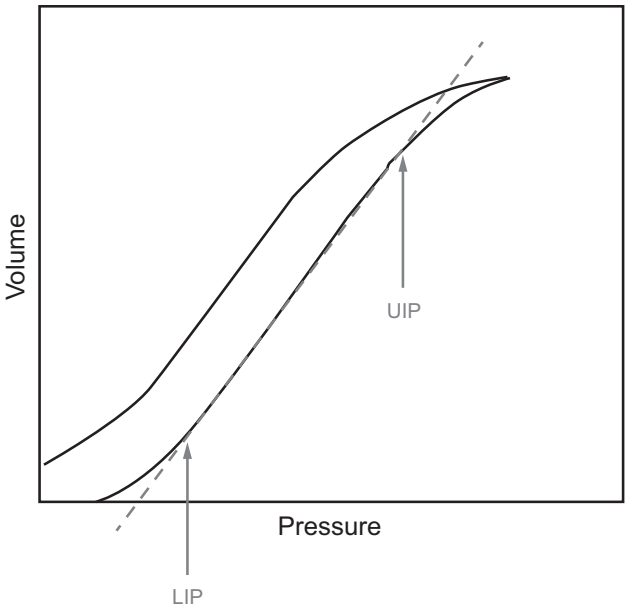

Fig. 6. The pressure-volume curve of the respiratory system. The lower inflection point (LIP) represents the point on the pressure-volume curve at which lung compliance begins to increase. The upper inflection point (UIP) represents the point during lung inflation at which lung compliance begins to diminish. The dashed line is the segment of the pressure-volume curve that represents the maximum compliance of the respiratory system.

Although it may seem appealing to use the lower inflection point as a target for adjusting PEEP in patients with ARDS, the interpretation of the P-V curve must be reliable and reproducible. Significant inter- and intra-observer variability in identifying the lower inflection point in P-V curves obtained from patients with ARDS has been noted, with maximum differences up to 17 cm H_2O between observers for the same patient's curve.[49] This may partly be explained by the fact that alveolar recruitment does not happen uniformly and instantaneously, especially in a heterogeneous disease such as ARDS. In a uniformly recruited lung, the lower inflection point should be well defined and sharp; however, a very sharp lower inflection point may represent the airway opening pressure rather than lung recruitment, which can further complicate interpretation of the lower inflection point of the P-V curve.[50] In nonuniform lung recruitment, alveoli are recruited across a broader range of pressures leading to an absent or unclear lower inflection point.[51]

The point of lung inflation at which lung compliance begins to diminish is known as the upper inflection point. The upper inflection point can be used to determine the

lung volume and corresponding static P_{aw} that indicates overdistention of lung. In fact, an upper inflection point can be identified in most patients with ARDS, and using the upper inflection point to guide ventilator settings led to a reduction in V_T and P_{plat} for subjects in a small study where all patients started with $V_T > 6$ mL/kg.[52] Notably, this study was performed prior to the 2000 ARDSNet study, which demonstrated the benefit of targeting a low V_T (eg, 6 mL/kg).[4]

Another method to identify the optimum PEEP or to determine lung recruitability is assessing the hysteresis of the P-V curve.[53] Hysteresis is represented as the area within the P-V curve and is influenced by the surface tension of the lung. This is graphically represented by a larger degree of separation between the inspiratory and expiratory limbs of the P-V curve.[54]

In summary, the quasi-static P-V curve can provide significant information about the mechanics of the lung and chest wall at different inflation pressures and volumes. Ideally, the P-V curve would allow for identification of the PEEP needed for optimum lung recruitment and the lung volume above which lung overdistention occurs, leading to safer ventilation. However, lack of standardized acquisition of P-V curves, difficulties in measuring absolute lung volume, poor agreement across interpreters, and a paucity of data showing a benefit in morbidity and mortality with the use of P-V curves have limited the clinical usefulness of the quasi-static P-V curve.

Why Is Monitoring Airway Cuff Pressure Important?

The airway cuff on an ETT or a tracheostomy tube serves 2 main purposes: to maintain P_{aw} during mechanical ventilation, and to prevent leakage of secretions into the lungs. Early ETT cuffs were smaller in volume and required very high pressures to maintain a seal. Consequently, there was potential for significant focal pressure injury to the airway. As an example, a cohort of subjects who underwent tracheostomy and ultimately died had autopsies to evaluate the extent of tracheal injury at the cuff site.[55] Some damage, most of it severe, was present in essentially every trachea through which mechanical ventilation was provided with a cuffed tracheostomy tube for > 48 h. In subjects who had a tracheostomy tube > 3 d, the damage ranged from superficial erosions over the cartilaginous rings to extensive baring of cartilage and perforation through the posterior tracheal wall.[55]

As the potential for severe tracheal damage was recognized, manufacturers began to transition to the low-pressure, high-volume ETT cuff that has become the standard in modern practice. The larger volume cuff is able to distribute the pressure more evenly across the airway mucosa and achieves a seal with a much lower pressure applied to any portion of the airway. Although using the newer cuffs reduces airway injury, it does not eliminate it.[56]

It is thought that a major determinant of airway injury is the focal pressure exerted on the tracheal mucosa, which can impede capillary blood flow and lead to ischemia of the tissue. In an elegant study performed in the early 1980s, tracheal mucosal blood flow was assessed using an endoscopic photographic technique while the cuff inflation pressure was varied in a series of 40 subjects intubated for surgery. The investigators reported that capillary blood flow was impaired at cuff pressures > 30 cm H_2O and that capillary blood flow over the tracheal rings was absent at a cuff pressure of 50 cm H_2O.[57]

Another cohort of subjects exhibited high rates of airway injury at the site of the cuff, despite attempting to maintain lower ETT cuff pressures. Cuff pressures were set at minimum occluding pressures (usually < 27 cm H_2O) every 8 h for subjects on controlled ventilation and at 27 cm H_2O for subjects breathing spontaneously. Less than 20% of subjects required a minimum occluding pressure of > 34 cm H_2O. On autopsy, 82% of tracheostomy subjects had airway injury at the cuff site ranging from moderate mucosal inflammation or edema to mucosal ulceration. With the ETT, 54% had similar injuries at the cuff site. Prolonged cuff pressure > 27 cm H_2O correlated with increased risk of tracheal injury on autopsy.[58]

It appears that limiting the upper range of airway cuff pressures to < 27–30 cm H_2O may help reduce pressure-related ischemia and injury to the trachea. Air leaks around the ETT cuff can typically be eliminated with cuff pressures at or slightly above P_{aw}. Therefore, at $P_{aw} \leq 25$ cm H_2O, the cuff pressures can typically be maintained below the capillary perfusion pressures of approximately 30 cm H_2O. Higher cuff pressures may be required to eliminate air leaks at $P_{aw} > 25$ cm H_2O.[59]

Preventing secretion drainage is not achieved solely by increasing airway cuff pressures. In fact, in a laboratory model, leakage of fluid around the ETT cuff occurred with all tubes studied and at all inflation pressures, even at cuff pressures of 60 cm H_2O.[60] Drainage of secretions was only prevented when the tracheal pressure was greater than the height of the fluid column above the cuff.[60,61] Part of the reason for the leakage of secretions is that modern high-volume, low-pressure cuffs have a diameter greater than that of the trachea. When the polyvinyl chloride cuff is inflated, it forms longitudinal folds that can form tracks for drainage. ETT cuffs made of different materials, such as polyurethane, have been tested as well, but whereas leakage of the fluid may be delayed, it was not eliminated.[61]

Although higher cuff pressures do not eliminate leakage of fluid around the ETT cuff, there does appear to be a lower threshold for pressure where leakage is significantly increased. As an example, in a laboratory model, leakage of fluid around the cuff occurred independently of CPAP or

PEEP at a cuff pressure of 15 cm H_2O.[60] This appears to be true in the clinical setting as well, where rates of microaspiration and ventilator-associated pneumonia (VAP) are higher in patients with ETT cuff pressures < 20 cm H_2O.[62]

In summary, airway cuff pressures > 30 cm H_2O appear to significantly impair capillary blood flow in the airway mucosa, and elimination of significant air leak can typically be achieved with pressures < 30 cm H_2O. Leakage of secretions around the airway cuff occurs regardless of the cuff material and is not eliminated with dangerously high cuff pressures. However, it is significantly increased when cuff pressures drop to < 20 cm H_2O. For these reasons, it is important to monitor airway cuff pressures to reduce the risk of both P_{aw} injury and leakage of secretions into the lungs. Based on the data available, an airway cuff pressure of 20–30 cm of H_2O should be targeted.

What Methods Are Available to Measure Airway Cuff Pressure?

Cuff pressures are assessed with various methods and at varying frequencies in different settings and institutions. Common methods include the pilot balloon palpation technique, minimum occlusive pressure/minimum occlusive volume test, intermittent manometry, and continuous manometry. The previous section outlined the rationale for measuring airway cuff pressures and the safest range of pressures to minimize airway injury, air leak, and leakage of secretions. Given that multiple methods for assessing airway cuff pressure are available, the reliability of each technique must be considered.

The pilot balloon palpation technique and the minimum occlusive pressure test are common and simple to perform at the bedside. The pilot balloon palpation technique is achieved by placing the pilot balloon of the airway cuff between the thumb and forefinger and squeezing to assess the tension within the balloon. The minimum occlusive pressure test is performed by inflating the cuff until audible leak can no longer be heard with a stethoscope. The pressure measured when no audible leak can be heard is the minimum occlusive pressure, and the volume required to achieve no audible leak is the minimum occlusive volume.

These 2 methods are highly subjective, however, and the following studies highlight the inaccuracy of these techniques. In a cohort of 101 cardiac surgery subjects admitted to the ICU, trained nurses palpated the cuff and 73% were felt to be in the appropriate range (ie, 20–30 cm H_2O). In fact, 90% of subjects had cuff pressures > 30 cm H_2O (average was 54 cm H_2O). When using the minimum occlusive volume test, 78% of subjects had pressures > 30 cm H_2O (average was 44 cm H_2O).[63] In a cohort of trained anesthesiologists using the minimum occlusive pressure and pilot balloon palpation techniques, the average cuff pressure was 50 cm H_2O.[64] Another study involving anesthesia providers who inflated the ETT cuff using their usual inflation technique reported that the average P_{aw} was 45 cm H_2O, and less than one third of providers inflated the cuff within an ideal range.[65]

To ensure the ETT cuff pressure is within an appropriate range, it can be monitored periodically using a hand-held manometer by the nursing or respiratory staff, or continuously with a continuous bedside pressure monitor. Intermittent monitoring with a manometer will reduce the risk of high airway cuff pressures, but this may not be adequate to prevent low airway cuff pressures. Cuff pressures < 20 cm H_2O were identified 45% of the time with routine intermittent monitoring but only 0.7% of the time with continuous regulation of the cuff pressure while using an automatic device.[66] During mechanical ventilation, the airway cuff pressure tends to decrease over time when not adjusted, but an automatic pressure control device can prevent this occurrence.[67] In fact, even frequent manual adjustments may not be adequate. In another study, cuff pressure decreased to < 20 cm H_2O in 45% of measurement occasions only 2 h after adjusting it to 24 cm H_2O.[68]

Even short durations of higher ETT cuff pressure can lead to symptomatic complications. This was illustrated in 100 subjects who underwent neurosurgery where postoperative airway complications (eg, sore throat, cough, hoarseness) were more than twice as common in the monitored group and adjusted with minimum occlusive pressure and pilot balloon palpation technique versus an automatic cuff pressure controller to maintain a ETT cuff pressure of 25 cm H_2O throughout the surgeries.[64]

Studies evaluating risk of aspiration and VAP have reported mixed results. Given the data presented above, it may make sense that there was no difference in the incidence of VAP when comparing infrequent ETT cuff pressure monitoring (ie, immediately after intubation and when clinically indicated for an observed air leak or due to tube migration) with frequent ETT cuff-pressure monitoring (eg, immediately after intubation, every 8 h, and when clinically indicated).[69] Because airway cuff pressures can fall out of range quickly, every 8 h may not be frequent enough. Comparing continuous versus intermittent ETT cuff pressure control indicates a lower incidence of VAP with continuous pressure control compared with intermittent pressure control.[62,70]

In summary, even skilled providers are poor at selecting the appropriate ETT cuff pressures using standard methods of pilot balloon palpation technique and the minimum occlusive pressure test, and the average pressures achieved using these methods are well above what is typically considered safe. Additionally, ETT cuff pressures are not static, and they change significantly over short time intervals. This is present under steady state conditions, and it is likely worse with movement and change in patient condition. Continuous monitoring and adjustment of airway cuff

pressure appears to be far superior to other techniques in maintaining an airway cuff pressure within the target range. It also appears to improve clinically meaningful outcomes of postoperative airway complications and VAP.

Conclusion

Mechanical ventilation is an extremely common form of life support used throughout the world to support patients undergoing general anesthesia or patients with respiratory failure in the setting of critical illness. While potentially life-saving, mechanical ventilation carries with it the potential for significant harm, even when provided for only short periods of time. Therefore, it is imperative that patients receiving mechanical ventilation be monitored closely to reduce the risk of injury. Pulse oximetry allows for the monitoring of adequate oxygenation and can identify early signs of worsening lung injury and desaturation. Capnography can allow for assessment of V_D fraction and changes in ventilation. Monitoring of driving pressure and P_L, as well as assessment of P-V loops, can help optimize PEEP and minimize overdistention and barotrauma to the lungs. Finally, continuous monitoring and adjustment of ETT cuff pressure can reduce the risk of airway injury and VAP. Although all of these monitoring modalities have potential benefits, their limitations must be understood to maximize utility in monitoring a patient receiving mechanical ventilation.

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Discussion

Dexter: Brady [Scott] and I just submitted a manuscript to RESPIRATORY CARE discussing cuff pressure management. We found some literature stating that researchers compared continuous pressure monitoring of the cuff to intermittent monitoring and results actually showed that there were decreased rates of VAP in subjects who were continuously monitored.[1] What are you thoughts on that?

Rackley: If the cuff pressure is lower then you have increased secretion drainage and increased risk, and if you continue to monitor the cuff pressure it goes down with time. You're looking at balancing two things. At a slightly higher cuff pressure there is increased risk of airway injury and at a slightly lower pressure there is increased risk for VAP. The risk for airway injury is probably higher than the risk for VAP, as long as you're in the range of accepted cuff pressures.

Scott: I thought another interesting paper that came out of the literature search we did was a study that showed whenever you do an intermittent check of the cuff you can actually drop the cuff pressure. You have to be thoughtful about the connection and disconnection of the syringe or manometer when checking cuff pressures.

***Hess:** Brady, don't you think that's a function of the dead volume in the system? If you hook the manometer directly to the pilot balloon, I think there's very little loss of air from the balloon, but if you have a big extension tube then for sure you're going to deflate the cuff by making the measurement.

Scott: Yes, you are right.

***Hess:** What you're saying is true but I think it's dependent on the type of system you use.

Scott: I agree.

Rackley: What is your practice for cuff pressure monitoring?

Scott: We do intermittent cuff pressure checks along with ventilator assessment throughout the day. At least once a shift we do a cuff pressure check with a manometer. We don't do minimum leak or minimum occlusion, we target an actual pressure. The way I understand it, that's the more recommended way to do it, the minimal leak technique is no longer recommended in terms of standardized practice.

***Hess:** I agree with that. I have referred to the minimum leak technique as the minimum aspiration technique.

MacIntyre: I'm not a therapist, so I don't do this, but if you go to 30 or 35 cm H_2O, if that's what you target Dean [Hess], Brady, and Amanda [Dexter]? If you hear a leak or see on the ventilator that there clearly is volume being lost, what do you do then?

***Hess:** I think that raises a whole other set of questions, for example maybe the tube size is too small. If you put a number 6 endotracheal tube in a large trachea, if there's still a leak there may be high pressure with no injury, because the cuff may not be touching the tracheal wall.

MacIntyre: But you're not going to maintain PEEP and you may increase your aspiration risk.

***Hess:** Right.

MacIntyre: So from a practical point of view, would you reintubate that patient?

***Hess:** I probably would not reintubate because that's also a risk factor for VAP. Where I have seen this more is with tracheostomy tubes. And I have often changed out a tracheostomy tube for a larger size for just that reason.

Scott: That actually happened with me not that long ago when we were troubleshooting a significant leak on a mechanical ventilator. To stop the leak, the pressure in the tracheostomy tube cuff had to be very high. When I demonstrated that to the team, and despite our attempts to mitigate the leak any other way, we consulted with otolaryngology and they replaced the smaller tracheostomy tube with a larger one.

MacIntyre: Again, I'm probably showing my lack of respiratory care expertise here, but if you go to 30 cm H_2O, is there a reason you might want to drop it to 25 or 20 cm H_2O and see if a leak develops? Is that a reasonable thing to do?

***Hess:** Sure. What I've taught is to maintain the cuff pressure at 20–30 cm H_2O. So if it's 30 and you reduce it to 25 I would not have a problem with that.

Scott: That's exactly what we teach, 20–30.

Dexter: Yes, we teach the same, 20–30 cm H_2O.

MacIntyre: Do you make an attempt to go to 15?

***Hess:** That will increase the risk of aspiration.

MacIntyre: I mean in assessing for a leak?

Scott: No, we don't do that. I don't know if it's the perfect way, but the way that we teach cuff pressure management is to attempt to keep pressures between 20–30 cm H_2O, with the idea that if the cuff pressure is too low there is an increased risk for aspiration. If it is too high, tracheal damage may occur.

Walsh: I'll add a comment about 35 cm H_2O, I've seen it herniating out.

***Hess:** So it's not positioned correctly.

Walsh: It's not positioned correctly and so people are having pressure that is potentially very high in the airway and you need to push the tube back down not keep inflating the cuff.

***Hess:** Brian makes an excellent point, for an endotracheal tube it's often because the tube is too proximal and the cuff is inflating in the larynx and pharynx.

Rackley: I think that occurs more often than is documented. I have seen that many times in my career – a leaky cuff because it's actually sitting at the level of the vocal cords. Importantly, that scenario causes high risk for subglottic stenosis, because the cuff is highly inflated right near the vocal cords.

Scott: Am I correct that some mechanical ventilator companies have continuous cuff pressure monitoring integrated? I'm not familiar with it, I've heard this technology now exists.

Branson: There are standalone devices and at least one company has built-in automatic cuff pressure monitoring.

Goligher: Craig [Rackley], thank you for a great talk. This came up earlier, do you think we should include monitoring tidal volume in the list of items to monitor during routine noninvasive and invasive ventilation? I know it was mentioned earlier, but if you're in a volume control mode maybe it's not so necessary. I don't use volume control modes. We always use pressure-targeted modes in our ICU but a colleague in New York, Jeremy Beitler, says that he always monitors exhaled tidal volume even in volume control because often the numbers can be surprisingly different, particularly if the patients are making substantial efforts. Do you have a comment or any experience with that?

Rackley: Absolutely, I think we should measure P_{plat}, tidal volume, and driving pressure in all mechanically ventilated patients, and I think tidal volume should be measured or recorded in an absolute value and also in mL/kg of predicted body weight. It is probably more important to record in mL/kg, as it provides more accountability for the therapists. They are more likely to consider changing the settings if they are recording a tidal volume of 10 mL/kg than if they are recording a tidal volume of 550 mL.

MacIntyre: I'll underscore that point. People think if you're in a volume control mode, that's it. In fact, with some ventilators if a patient sucks hard enough and pulls the pressure below 0 it will deliver more volume. A very aggressive effort can make the tidal volumes bigger, even in volume control.

Pham: And also when you have asynchrony in patients leading to double breaths where the ventilator will not recognizer that there were two breaths one on top of the other, it will display that it delivered 6 mL/kg two times but actually there was no expiration between the breaths so the patient received 10 or 12 mL/kg.

Rackley: That's why we have to overcome our gross incompetence and recognize it.

Blanch: Not only that. Some ventilators allow extra inspiratory flow and tidal volume will go to 14 or 15 mL/kg.

MacIntyre: This is also a phenomenon I think people forget in APRV. The ventilator delivers a tidal volume and then the spontaneous breathing adds to that tidal volume. If I may emphasize Craig's point, we should monitor tidal volume regardless of ventilator mode.

Goligher: There are some pretty impressive data that tidal volume predicts failing NIV, with patients with higher tidal volumes being worse. That's certainly made me pay much more attention to tidal volume in that group of patients, particularly in hypoxemic respiratory failure.

Piraino: I don't mean to divert away from tidal volume but to add a comment. I agree the pressure-volume curve is not something we should routinely do. However, in our more complex patients – this is not based on a lot of evidence, but a nice research letter[2] submitted by one of our lab members. Dr Lu Chen looked at the issue of airway closure that the pressure-time curve or pressure-volume curve can identify a level below which your patients are not being ventilated. We think this is an overestimation of an inflection point in the literature and one of the reasons why if you did it on

a number of ARDS patients many patients don't have an inflection point. When the inflection is very clear in someone with airway closure if you measure the volume and pressure (compliance) at the level of inflection, it's similar to the compliance of a ventilator circuit, which assumes you have complete airway closure and then it opens. In our more complex patients, rather than doing a slow flow pressure-volume curve (our ventilators don't have the automated feature), we use volume-control and do a pressure-time curve so we have the slow flow inflation of 5 L/min flow from PEEP of 5 and when you look at the pressure-time curve when you have airway closures it's a very clear inflection. The pressure rises very quickly and then completely changes direction and setting the PEEP below that makes no sense and makes your driving pressure appear arbitrarily high when some of the pressure is just required to overcome the closing pressure. In our more complex patients, like ARDS patients for example, we'll include this in their registry form that Tai [Pham] discussed earlier. The first step is to rule out that these patients don't have airway closure which, if somebody was routinely doing pressure-volume curves, they would capture. Again, it's not necessary for all patients, but I still think it has some utility despite some papers showing the inter-observer reliability is way off for assessing PV curves. Probably because, when patients don't have airway closure, it's not clear at all.

Rackley: How is that different than the stress index?

Piraino: This is slow flow inflation, so the stress index would certainly be represented by the section after the infection. However, a stress index should be done at your clinically set PEEP level to give valuable information.

MacIntyre: You can catch it at both top and bottom can't you? The stress index really is a constant flow, pressure-volume during the tidal breath.

Piraino: The pressure curve goes literally almost straight up on a slight angle and then inflects that way rather than going this way or around this way. It's very sharp.

Pham: Somewhere I have an example.

Piraino: In Lu's letter,[2] he had the pressure-time curve, or did he have the pressure-volume curve?

MacIntyre: But at constant flow breath it's essentially a pressure-volume curve.

Piraino: I just don't know that it's capturing the flow at which stress index would be done. We use 5 L/min so it's very slow flow inflation. Pressure-volume curve for slow flow inflation is usually <9 L/min flow. Here at the red line is an occluded circuit. He just overlapped the pressure-volume curve so you can see at which point and until you reach a pressure here, this is traditionally called the inflection point but essentially – again, you would manage it similarly. But what's interesting is that it's not recruitment, it's complete opening. That was his point, that if you take a ventilator and completely block it and overlap it you get this you're basically ventilating the circuit until that point at which it opens. Now, this part here is what you'd see on the stress indes typically, especially at a high flow. You may not see this at a very slow flow. But if this was curved upwards or downwards, like this one that's a bit rounded here, that would be similar to the SI.

MacIntyre: Greg [Schmidt] and I are sitting here nodding. That's a stress index with a PEEP that's too low.

Piraino: You think this is curved downwards?

MacIntyre: No, that's the typical stress index you would see in someone with inadequate PEEP. I accept your airway closure phenomenon, I think that would easily explain this and I don't disagree with your physiology. But I think you would see it with the stress index if you had a PEEP of zero. If the phenomenon was occurring.

***Hess:** You would see a downward concavity on the stress index.

Piraino: I agree you would see a downward concavity, but I've not seen such a drastic curve in any of the papers. It's literally a hockey stick.

MacIntyre: Tom, don't misunderstand me, I'm not saying it wouldn't look different.

Piraino: I agree, I see what you're saying. I'm so used to seeing stress index where you really have to think, 'is that curving outwards?' when you do this low flow inflation, it looks like a hockey stick.

Goligher: And if you have a higher flow you pressurize very rapidly, you don't think you would miss it?

Schmidt: But you should use a low flow and Neil does for the stress index.

MacIntyre: Yeah, you get rid of the flow-related stuff.

Piraino: If you use a flow slow enough, I know on the Servo-i for example, you can get a stress index with 40 L/min. I'm not sure if you would capture the airway closure. You have to go slow enough to be sure you capture it.

Schmidt: To go back to the square wave flow vs decelerating flow

profiles, if you use square-wave flow profile you'll see that there's a pressure increment at the beginning of the breath and a pressure decrement at the end of the breath that are equal – but they'll be unequal in this airway closure instance. There will be a bigger increment and then a smaller decrement.

Piraino: To bring it back to the clinical setting, we started using it more routinely in more complex patients with higher Pplat pressure, hypoxemia, etc, and doing the slow flow inflation to determine if this is somebody who needs PEEP at a certain level in order to ventilate them properly (above airway closure). Most of these patients, once we achieve the right PEEP, we see drop in Pplat pressure based on the fact we're overcoming airway closure.

Blanch: Recruitment or over-distension could be assessed in total pressure-volume curves performed in static conditions. Combined with driving pressure or ventilator efficiency measurements can offer more information.

Rackley: In this scenario did you measure the esophageal pressure?

Piraino: We've asked Lu Chen many times. In some patients it actually correlates quite well with things like esophageal pressure, but in many patients it did not. It wasn't such a phenomenon that just putting a balloon in tells us if we have airway closure.

Rackley: They needed more PEEP.

Piraino: We will often increase PEEP and try to achieve a transpulmonary pressure of 0 cm H_2O, but we ultimately look at the lung elastance ration (E_L/E_{RS}) based on the fact that it may reflect more healthy regions of the lung and will limit PEEP for lung protection. We often end up in patients with high elastance ratios with PEEP that is less than 0 transpulmonary pressure but it doesn't seem to result in issues keeping PEEP above airway closure in patients that have it.

Pham: Lu's first step in setting the PEEP is looking for this airway closure and if a patient has airway closure to set the minimum PEEP at the point of airway closure and then measure all your mechanics.

Piraino: This was the phenomenon Tai brought up when I did some work with Thiel cadavers doing a modified Baydur maneuver, obviously they're passive, but I performed the modified Baydur maneuver during an expiratory pause by pushing on the chest or the abdomen. Below airway closure the pressure is not transmitted to the ventilator screen so you appear to have improper placement of the catheter, if you did the same maneuver right after, but during an inspiratory pause, the maneuver is performed above the airway closure and you were able to get an accurate measurement of the ratio of airway pressure to esophageal pressure without needing to reposition the catheter.

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*Dean R Hess PhD RRT FAARC is Managing Editor of RESPIRATORY CARE.