Airway Pressures and Volutrauma: Is Measuring Tracheal Pressure Worth the Hassle?

Monitoring airway pressures during mechanical ventilation is a standard of care.[1] Sequential recording of airway pressures not only provides information regarding changes in pulmonary impedance but also allows safety parameters to be set. Safety parameters include high- and low-pressure alarms during positive pressure breaths and disconnect alarms. These standards are, of course, based on our experience with volume control ventilation in adults. During pressure control ventilation, monitoring airway pressures remains important, but volume monitoring and alarms are also required.

Airway pressures and work of breathing are also important components of derived variables, including airway resistance, static compliance, dynamic compliance, and intrinsic positive end-expiratory pressure (auto-PEEP), measured at the bedside.[2] The requisite pressures for these variables include peak inspiratory pressure, inspiratory plateau pressure, expiratory plateau pressure, and change in airway pressure within a breath. Plateau pressures should be measured at periods of zero flow during both volume control and pressure control ventilation. Change in airway pressure should be measured relative to change in volume delivery to the lung (pressure-volume loop) to elucidate work of breathing.

See the **related study** on Page 1179.

Evidence that mechanical ventilation can cause and exacerbate acute lung injury has been steadily mounting.[3-5] While most of this evidence has originated from laboratory animal studies, recent clinical reports appear to support this concept.[6,7] Traditionally, ventilator-induced lung injury brings to mind the clinical picture of tension pneumothorax. Barotrauma (from the root word *baro*, which means pressure) is typically associated with excessive airway pressures. Current thinking regarding ventilator-induced lung injury includes barotrauma, but the real emphasis has shifted to *volutrauma*.

For distinction, barotrauma is extra-alveolar air and includes pneumothorax, pneumomediastinum, pneumoperitoneum, and subcutaneous emphysema. Volutrauma, on the other hand, is lung injury secondary to overdistention of alveoli. Volutrauma is manifested by disruption of the alveolar capillary membrane and alterations in gas exchange that mimic acute lung injury. These distinctions are important because it is not peak airway pressure that is deleterious to the pulmonary architecture. For instance, in a classic example, we suppose that the lung is placed in a rigid container; under this condition, the airway pressures could rise to astronomical values with little to no effect on the pulmonary parenchyma thereby protecting the lung from injury. This protection occurs because the container limits lung inflation. The term volutrauma, then, may be a more representative term for the iatrogenic lung injury seen during mechanical ventilation.

Large tidal volumes are not inherently dangerous; in fact, volutrauma can occur at relatively modest tidal volumes. Volutrauma occurs when individual alveolar units are overstretched. The propensity for development of volutrauma is, therefore, related to the volume of lung available and to regional differences in compliance. In acute respiratory distress syndrome (ARDS) where lung volume is significantly diminished, compliance is reduced, and heterogeneous lung injury is the rule, the risk of volutrauma is magnified. In a postoperative patient with reduced lung volumes and relatively normal compliance, volutrauma is less likely, even at relatively large tidal volumes. This is a quandary that makes setting safe limits on tidal volume for a wide variety of patients extremely difficult.

So where does this leave us? Several groups have espoused the use of small tidal volumes (4-8 mL/kg), pressure-limited

ventilation, and permissive hypercarbia.[8,9] Guidelines for maximum airway pressure are based on laboratory data, and typically a plateau pressure < 35 cm H2O is thought to limit volutrauma.[8,9] In this issue of the Journal, Jager and Tweeddale[10] suggest that monitoring tracheal pressure is a better estimate of volutrauma and barotrauma risk. Their study demonstrates that the presence of the endotracheal tube causes the ventilator to overestimate true peak inspiratory pressure. The pressure drop across endotracheal tubes has been well described, and previous authors have made similar conclusions.[11,12] Jager and Tweeddale suggest, if one is to insist on following peak airway pressure, tracheal airway pressure is the more appropriate measurement.

In several respects, I believe Jager and Tweeddale are correct. Proximal and ventilator airway pressures overestimate the actual airway pressure applied to the lung. Tracheal pressure allows a more accurate estimate of airway resistance because it eliminates the endotracheal tube as a confounding factor. They also suggest that predicting tracheal pressure from proximal pressure is unreliable. Other authors have agreed and disagreed with this conclusion,[13,14] but this finding has implications for manufacturers who might use estimated tracheal airway pressure to automatically compensate for tracheal tube resistance. Jager and Tweeddale's[10] finding that the ventilator pressure is much higher than a separate measurement of proximal airway pressure is interesting. It is unfortunate that the type of ventilator(s) used in this study10 is not mentioned. The question then becomes, Is measuring tracheal pressure worth the hassle?

One limitation of this paper[10] is the tracheal pressure measurement technique. When a catheter is placed through the length of the endotracheal tube, the resistance characteristics of that tube are changed. The catheter effectively reduces the diameter of the tube and promotes turbulence. The fact that an endotracheal tube in vivo with a catheter running its length results in poor estimation of tracheal pressure based on in vitro constants measured without the catheter in place is not surprising.

With regard to the utility of proximal airway pressure as currently measured or tracheal airway pressure as suggested by Jager and Tweeddale[10] in establishing risk for volutrauma, I believe the answer is that neither is particularly helpful. The best estimate of alveolar distending pressure is plateau pressure.[2] When airway flow is zero at end-inspiration or end-expiration the plateau pressure is equivalent throughout the respiratory tract. In fact, the use of static pressure-volume curves has been shown to be helpful in identifying both overdistention (the upper inflection point) and optimal PEEP (the lower inflection point) during ventilatory support of patients with acute lung injury.[7,8] Therefore, I believe it is not only practical but also prudent to continue to measure peak inspiratory pressure during routine ventilator care. This pressure is readily available, has been used successfully for decades, and provides the kind of information required for routine monitoring. Jager and Tweeddale[10] are correct, peak inspiratory pressure is a poor predictor of alveolar pressure and can be misleading during the comparison of ventilatory modalities. But the measurement of tracheal pressure only adds complexity. In the face of rising airway pressure and concern for the development of ventilator-induced lung injury, plateau pressures should be used to estimate alveolar pressure, and static pressure-volume curves should be used to evaluate tidal volume and PEEP settings.

Another important point should be made regarding the example of the lung in a rigid container. The lung is normally in a semirigid container--the chest wall. Under this condition, the transalveolar pressure changes with changes in chest-wall compliance. This has clinical significance. The elderly patient with pneumonia and chronic lung disease and with mechanical ventilation for acute lung injury has an entirely different tolerance for airway pressures than the young trauma patient with acute lung injury following massive blood and fluid resuscitation. In the first case, the patient with relatively normal chest-wall compliance may only tolerate 30 cm H_2O of plateau pressure before regional alveolar overdistention occurs. In the second case, higher airway pressures may be tolerated due to the relatively stiff chest wall. The risk of volutrauma then, is best judged by transalveolar pressure (alveolar pressure-pleural pressure). This value does not easily lend itself to routine measurement, but the use of static pressure-volume curves does appear to offer some assistance in this area.

Jager and Tweeddale[10] have provided us with further evidence that plateau pressure is the key to monitoring alveolar

pressure. At present, static pressure-volume curves require time and expertise to accomplish correctly. However, a recent report has demonstrated that under appropriate conditions, upper and lower inflection points can be obtained during a single, slow passive inflation.[15] This technology may become extremely useful as we continue to care for patients with ARDS into the year 2000.

Richard D Branson RRT
Assistant Professor of Clinical Surgery
Department of Surgery
Division of Trauma and Critical Care
University of Cincinnati Medical Center
Cincinnati, Ohio

References

1. AARC Clinical Practice Guideline. Patient-ventilator system checks. Respir Care 1992;37(8):882-886.
2. Tobin MJ. Respiratory monitoring in the intensive care unit. Am Rev Respir Dis 1988;138(6):1625-1642.
3. Dreyfuss D, Basset G, Soler PS, Saumon G. Intermittent positive-pressure hyperventilation with high inflation pressures produces pulmonary microvascular injury in rats. Am Rev Respir Dis 1985;132 (4):880-884.
4. Kolobow T, Moretti MP, Fumagalli R, Mascheroni D, Prato P, Chen V, Joris M. Severe impairment in lung function induced by high peak airway pressure during mechanical ventilation: an experimental study. Am Rev Respir Dis 1987;135(3):312-315.
5. Webb HH, Tierney DF. Experimental pulmonary edema due to intermittent positive pressure ventilation with high inflation pressures. Protection by positive end-expiratory pressure. Am Rev Respir Dis 1974;110(5):556-565.
6. Hickling KG. Low-volume ventilation with permissive hypercapnia in the adult respiratory distress syndrome. Clin Intensive Care 1992;3:67-78.
7. Amato MPB, Barbas CSV, Medeiros DM, Schettino GdeP, Lorenzi Filho G, Kairalla RA, et al. Beneficial effects of the "open lung approach" with low distending pressures in acute respiratory distress syndrome: a prospective randomized study on mechanical ventilation. Am J Respir Crit Care Med 1995;152(6):1835-1846.
8. Pesenti A. Target blood gases during ARDS ventilatory management (editorial). Intensive Care Med 1990;16(6):3449-351.
9. Marini JJ, Kelsen SG. Re-targeting ventilatory objectives in adult respiratory distress syndrome. New treatment prospects-persistent questions (editorial). Am Rev Respir Dis 1992;146(1):2-3.
10. Jager K, Tweeddale M. In-vivo comparison of measured tracheal pressure and tracheal prssure determined by a simple bedside equation. Respir Care 1997;42(12):1179-1183.
11. Sullivan M, Paliotta J, Sakland M. Endotracheal tube as a factor in measurement of respiratory mechanics. J Appl Physiol 1976;41(4): 590-592.
12. Warters RD, Allen SJ, Tonnesen AS. Intratracheal pressure monitoring during synchronized intermittent mandatory ventilation and press controlled-inverse ratio ventilation. Crit Care Med 1997;25(2): 227-230.
13. Fabry B, Haberthur C, Zappe D, Guttmann J, Kuhlen R. Breathing pattern and additional work of breathing in spontaneously breathing patients with different ventilatory demands during inspiratory pressure support and automatic tube compensation. Intensive Care Med 1997;23(5):545-552.
14. Messinger G, Banner MJ, Blanch PB, Layon AJ. Using tracheal pressure to trigger the ventilator and control airway pressure during continuous positive airway pressure decreases work of breathing. Chest 1995;108(2):509-514.
15. Servillo G, Svantesson C, Beydon L, Roupie E, Brochard L, Lemaire F, Jonson B. Pressure-volume curves in acute respiratory failure: automated low flow inflation versus occlusion Am J Respir Crit Care Med 1997;155(5):1629-1639.

Correspondance: Richard D Branson RRT, Division Trauma/Critical Care, Department of Surgery, University of Cincinnati College of Medicine, 231 Bethesda Ave, ML 0558, Cincinnati OH 45267-0558.

Back Top