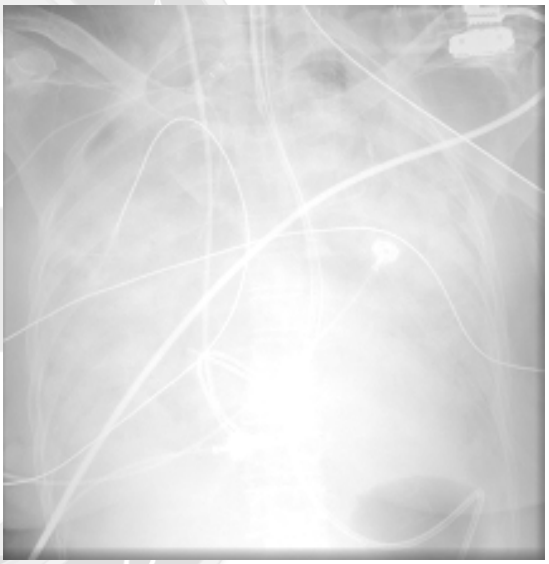
**Is there something special in taking the critically injured patient back to the operating room?**

Carl W. Peters, MD
Associate Professor of Anesthesiology
Joint Associate Professor of Surgery
Department of Anesthesiology
University of Florida, Gainesville, FL 32611

Date for publication: May 14, 2015

Respiratory issues can be among the most challenging to overcome when providing an anesthetic to a critically ill patient. In this piece, the following will be addressed:

- Transporting the critically ill patient to the operating room
- Maintenance of functional residual capacity [FRC]
- Optimal patient position and its impact on respiratory volumes
- Prevention of ventilator-induced lung injury [VILI]
- Monitoring end-tidal $CO_2$ and $PaCO_2$
- Role of recruitment maneuvers [RMs] in improving gas exchange; in particular, in improving intraoperative oxygenation
- Determining optimal ventilator settings in the patient with "stiff lungs" using the time constant ("tau")
- Employment of "low tidal volume ventilation"



The ordeal of anesthetizing a critically ill patient with respiratory compromise may begin with the very first step: deciding whether to remove the patient from the ICU ventilator for the trip to the operating room. For a patient who requires an inordinate level of positive end expiratory pressure [PEEP] to maintain oxygenation (such as the in the radiograph above), it may be advisable to keep the patient connected to the ICU ventilator for the entire event and to use a total IV anesthetic during the case. Any disconnection of the circuit for machine switchover, however brief, invites collapse of the stiffer alveoli, and such "de-recruitment" inevitably compromises gas exchange.[1] In such instances, one may briefly occlude the patient's endotracheal tube during switchover to avoid de-recruitment of dependent alveoli that may be quite problematic to re-expand. A large surgical Kelly clamp with rubber boots applied to the clamp jaws is an ideal device for such a purpose. The integrity of endotracheal tube placement during the switchover must be assured.

Maintenance of PEEP and lower tidal volume lung-protective ventilation throughout the transport and perioperative periods minimizes, on the one hand, *atelectrauma* (injury from the continuous cycle of alveoli collapsing and reopening), and on the other,

*volutrauma* and *barotrauma* (injury from the excessive over-distention of more compliant alveoli). These damaging processes are fundamental to the development of *VILI*, a major instigator of the inflammatory cytokine-generating processes leading to acute respiratory distress syndrome [ARDS] and multiple organ dysfunction syndrome.[2,3] The supine position further compromises the FRC, resulting in worsening ventilation-perfusion matching.[4] Positioning the patient supine causes the weight of edematous viscera to push the diaphragm toward the head and promotes dependent alveolar collapse. This results in maldistribution of ventilation to the more compliant, non-dependent segments of the lung, while the dependent compressed alveoli remain collapsed and do not participate in gas exchange. This effect may be accentuated in the patient with elevated intra-abdominal pressure. The overall result is a substantial decrease in FRC by as much as 1 liter in volume.[5,6] Slight reverse Trendelenburg positioning can somewhat ameliorate this effect. Higher PEEP and low tidal volumes more optimally distribute gas exchange throughout the lung while minimizing extremes of pressure and volume.[7]

Intermittent RMs, applied with sufficient pressure and duration and combined with PEEP, will often restore aeration of dependent alveoli and maintain their expanded state at a level of pressure lower than that required to "open" them in the first place ("hysteresis effect").[8,9] The use of the RM benefits alveolar anatomy by transiently elevating airway pressure to restore volume to a collapsed alveolus. This is the application of the Law of LaPlace (see **Figure 1**), which defines wall tension to be a function of pressure and radius, thus allowing alveoli with smaller radii and higher surface tension to be recruited. The use of and even the applicability of RM to improve gas exchange is controversial,[10-13] but in the clinical scenario of a desaturating critically ill patient, the judicious use of RMs appears to be appropriate. The hysteresis effect is attributed to several etiologies: (1) the viscous resistance of pulmonary surfactant, (2) the increased resistance associated with turbulent airflow during early inspiration (when airways are narrower), and (3) the fact that because the lungs act as an elastic body during positive pressure ventilation, the force required to stretch the lung by a given distance is proportional to that distance.[14] Thus, alveoli that are "open" from a recent RM and maintained thus with adequate PEEP achieve adequate volume changes (and gas exchange) over a lower range of pressures than those that receive the same airway pressure on expiration that, during a previous respiratory cycle, dropped below the pressure at which they collapse (termed the "lower inflection point" on the alveolar pressure volume curve).[15] The combination of intermittent RMs and maintenance of PEEP above the lower inflection point is most effective in optimizing oxygenation in the patient with diffuse lung injury.[16]

$$2 \times \Upsilon = P \times r$$

**Figure 1. Law of LaPlace (Υ = tension, P = pressure, r = radius)**

Oxygenation is likely to improve with PEEP. This benefit, however, may be acquired in exchange for an increase in dead space from the loss of cardiac output associated with elevated intrathoracic pressure (in the *extreme* case actually leading to cardiac arrest) and from the increase in intrathoracic volume within which alveolar pressure exceeds capillary pressure. Both effects result in greater difficulty offloading carbon dioxide.[17] One crucial proviso to remember: while the acceptance of elevated $PaC0_2$ that may accompany minimization of PEEP and tidal volumes to avoid VILI, termed "permissive hypercapnia,"[18] may not be a critical issue in many patients, patients with barely controlled intracranial pressure are at increased risk for cerebral compromise from increased cerebral blood flow

if the arterial $CO_2$ content climbs excessively. Recall also that the difference between displayed $PetCO_2$ and $PaCO_2$ widens as dead space increases. Arterial blood gas measurements are warranted early during procedures to determine the baseline gradient. Ignorance of the gradient may lead to unintentional hypercarbia if an apparently "normal" end-tidal $CO_2$ level is maintained in the face of a large gradient that has not been identified. Chemical paralysis further worsens VQ matching by decreasing FRC 15% to 20%.[19,20] The obvious positive trade-off with paralysis is less chest- and abdominal wall-associated resistance to inspiratory airflow. Pressure-control ventilation offers the advantages of limiting inspiratory pressure and variability of inspiratory time [$T_i$] for patients with restrictive respiratory physiology by virtue of intrinsic lung disease (such as pulmonary fibrosis), intra-abdominal hypertension, non-cardiogenic pulmonary edema, or a variety of other restrictive pathologies. Lengthening of the $T_i$ to a duration equal to *three* times the $T_i$ allows filling of ~95% of alveoli.[21] The duration of one time constant ("*tau*") is the product of compliance and resistance, both measurable by modern ICU and OR ventilators with sophisticated software. Manipulation of the ratio between inspiratory and expiratory durations using the calculated value of *tau* offers the best likelihood of more uniform alveolar gas exchange while avoiding the problems of over-distension, insufficient/excessive tidal volumes, and potentially problematic auto-PEEP leading to "breath stacking." Breath stacking (the repeated initiation of an inspiration prior to full exhalation with eventual detrimental intra-thoracic pressure buildup) may occur with rapid respiratory rates and is identified by the presence of expiratory airflow below the "zero-flow" line on the flow-time ventilator waveform at the initiation of the next inspiration, as seen in **Figure 2**. In this clinical scenario, which is usually characterized by *restrictive* physiology, it is unlikely that breath stacking will significantly impact hemodynamic stability[22], but one must, nonetheless, be vigilant to detect its insidious deleterious onset. As compliance improves, the clinician must adjust the "delta-P," likely reducing the "high-pressure" setting, to maintain the lowest tidal volumes consistent with sufficient gas exchange. In most scenarios, it is prudent to maintain ventilation parameters within ARDSNet guidelines, which have been demonstrated to decrease mortality in ARDS patients.[23,24]

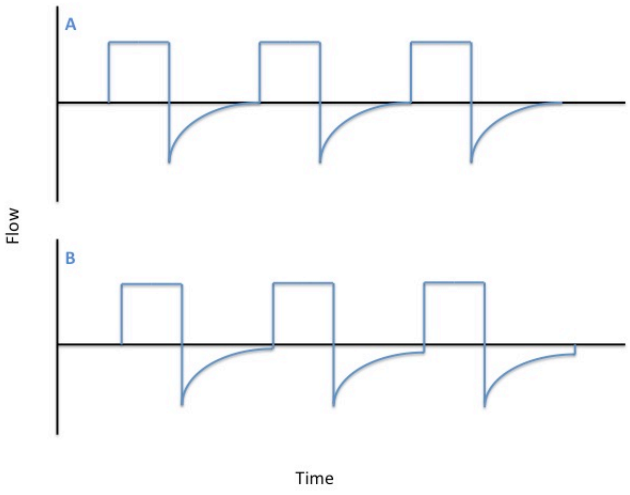



**Figure 2. Flow Versus Time Ventilator Waveforms. Tracing A shows the flow in a normal patient on a volume-controlled mechanical ventilation. Tracing B shows breath stacking with each subsequent breath during the same volume-controlled mechanical ventilation.**

In summary, anesthetizing a critically ill surgical patient can be quite problematic because of challenging respiratory physiology. The astute anesthesiologist with awareness of the parameters of compliance, resistance, and maintenance of sufficient mean airway pressure to achieve and maintain parenchymal aeration should be able to maintain the critically ill patient safely under general anesthesia for a complex surgical procedure.

**References**


1. Maggiore SM, Lellouche F, Pigeot J, et al. Prevention of Endotracheal Suctioning-induced Alveolar Derecruitment in Acute Lung Injury, American Journal of Respiratory and Critical Care Medicine, Vol. 167, No. 9(2003), pp. 1215-1224,
2. Belperio JA, Keane MP, Lynch JP 3rd, Strieter RM. The role of cytokines during the pathogenesis of ventilator-associated and ventilator-induced lung injury. Semin Respir Crit Care Med. 2006 Aug;27(4):350-64.
3. Slutsky AS. Lung injury caused by mechanical ventilation. Chest 1999; 116 (1 Suppl):9S-15S.
4. Harris R.S. Pressure-Volume Curves of the Respiratory System. Respir Care, January 1, 2005 50:1 78-99.
5. Wilson W, Benumof J. Physiology of the airway. In Benumof's Airway Management, 2nd Edition. Edited by Hagberg C.A. Philadelphia; Mosby Elsevier; 2007: 147;
6. Wahba RW. Perioperative functional residual capacity. Can J Anaesth. 1991 Apr;38(3):384-400.
7. Gattinoni L, Pelosi P, Crotti S, Valenza F. Effects of positive end-expiratory pressure on regional distribution of tidal volume and recruitment in adult respiratory distress syndrome; Am J Respir Crit Care Med 1995;151: 1807-14.
8. Pelosi P1, Goldner M, McKibben A, et al. Recruitment and derecruitment during acute respiratory failure: an experimental study. Am J Respir Crit Care Med. 2001 Jul 1;164(1):122-30.
9. Escolar JD, Escolar A. Lung hysteresis: a morphological view. Histol Histopathol. 2004 Jan;19(1):159-66.
10. Guerin C, Debord S, Leray V, et al. Efficacy and safety of recruitment maneuvers in acute respiratory distress syndrome. Ann Intensive Care. 2011 Apr 19;1(1):9. doi: 10.1186/2110-5820-1-9.
11. Villagrá A, Ochagavía A, Vatua S, et al. Recruitment maneuvers during lung protective ventilation in acute respiratory distress syndrome. Am J Respir Crit Care Med. 2002 Jan 15;165(2):165-70.
12. Fan E, Wilcox ME, Brower RG, et al. Recruitment maneuvers for acute lung injury: a systematic review. Am J Respir Crit Care Med. 2008 Dec 1;178(11):1156-63
13. Prange HD. Laplace's law and the alveolus: a misconception of anatomy and a misapplication of physics. Adv Physiol Educ. 2003 Dec;27(1-4):34-40.
14. Beachey W. Chapter 3: Mechanics of Ventilation; in Respiratory Care Anatomy and Physiology – Foundations for Clinical Practice. St. Louis, Missouri; Elsevier Mosby, pgs. 52-53.
15. Chambers D, Huang C, Matthews G. section 2, chapter 19, Lung Compliance; in Basic Physiology for Anaesthetists, Cambridge, United Kingdom; Cambridge University Press, 2015, page 85.
16. Lim CM1, Jung H, Koh Y, et al. Effect of alveolar recruitment maneuver in early acute respiratory distress syndrome according to anti-derecruitment strategy, etiological

category of diffuse lung injury, and body position of the patient. Crit Care Med. 2003 Feb;31(2):411-8.

17. Nieman GF, Paskanik AM, Bredenberg CE. Effect of positive end-expiratory pressure on alveolar capillary perfusion; J Thorac Cardiovasc Surg. 1988 Apr; 95(4):712-6.
18. Tuxen DV: Permissive Hypercapneic Ventilation; AmJRespirCritCareMed Vol 150.pp870-874,1994.
19. D' Angelo E, Milic-Emili J. Statics of the respiratory system. In Physiologic basis of respiratory disease; Hamid Q, Shannon J, Martin J. Hamilton, Ontario; BC Decker, Inc, 2005: 20.
20. Hedenstierna G, Strandberg A, Brismar B, et al. Functional residual capacity, thoracoabdominal dimensions, and central blood volume during general anesthesia with muscle paralysis and mechanical ventilation; Anesthesiology. 1985 Mar;62(3):247-54.
21. Lumb AB. Appendix F: Mathematical functions relevant to respiratory physiology; in Nunn's Applied Respiratory Physiology, 5th edition; Woburn, Mass; Butterworth-Heinemann 2000 pgs. 657-671.
22. Blanch L, Bernabe´ F, Lucangelo U: Measurement of Air Trapping, Intrinsic Positive End-Expiratory Pressure, and Dynamic Hyperinflation in Mechanically Ventilated Patients Respiratory Care, January2005 Vol 50(1), 110-124.
23. The Acute Respiratory Distress Syndrome Network. Ventilation with lower tidal volumes as compared with traditional tidal volumes for acute lung injury and the acute respiratory distress syndrome. The Acute Respiratory Distress Syndrome Network. N Engl J Med. 2000 May 4;342(18):1301-8.
24. Lellouche F, Lipes J. Prophylactic protective ventilation: lower tidal volumes for all critically ill patients? Intensive Care Med. 2013 Jan;39(1):6-15.