Intensive Care Med (2007) 33:1114–1116
DOI 10.1007/s00134-007-0677-0

EDITORIAL

John J. Marini

The "open lung" compromise

Received: 2 April 2007
Accepted: 13 April 2007
Published online: 25 May 2007
© Springer-Verlag 2007

This editorial refers to the article available at:
http://dx.doi.org/10.1007/s00134-007-0629-8.

J. J. Marini (✉)
University of Minnesota, Department of Medicine,
Regions Hospital, Pulmonary and Critical Care Medicine,
640 Jackson St, Rm 3571, St Paul 55101-2595, MN, USA
e-mail: john.j.marini@healthpartners.com
Tel.: +1-651-2543411
Fax: +1-651-2543098

How best to select positive end-expiratory pressure (PEEP) in acute respiratory distress syndrome (ARDS) remains among the most actively debated questions of critical care practice. Controversy stems partially from imprecision of disease diagnosis, confusion about which objective to prioritize, and uncertainty regarding safe limits for airway pressure. Perhaps the root cause for such indecisiveness, however, involves the mechanical heterogeneity of the acutely diseased lung coupled with the need to set only one PEEP value. In fact, when lung protection is the issue, PEEP selection is always a tradeoff between improving recruitment and increasing tissue stress. The article by Di Rocco and colleagues in this issue [1] offers novel experimental data that address how that compromise is best struck.

How is the optimum PEEP to be recognized at the bedside? Not many years ago, clinicians facing the PEEP selection issue were focused primarily on physiological variables, such as tidal compliance and the efficiency of oxygen exchange. (Some caregivers—perhaps most—still are.) With minimizing shunt the primary objective, high PEEP levels were often selected after relatively long periods of ventilator adjustment. More recently, awareness of the roles of high alveolar pressures and tidal opening and closure cycles in producing ventilator-induced lung injury (VILI) have reshuffled our priorities to emphasize lung protection. Although all are agreed that adverse patterns of ventilation can damage lungs, as yet no firm consensus has emerged as to the hierarchy of factors causative in VILI generation. No doubt remains regarding the importance of maximum tidal stretching pressure—little ventilator-caused injury is likely to result if the plateau pressure *recorded under passive conditions* can be kept low. From that point of general agreement, however, the conceptual waters become murky.

In a fully recruited lung, using sufficient PEEP (and/or associated smaller tidal pressure excursions) dramatically reduces the risk of VILI from any specified plateau pressure. One explanation for PEEP's benefit in this setting is its ability to eliminate high-stress interfaces that exist at the junctions of aerated and atelectatic tissues, to lower shearing stresses, and to prevent repeated recruitment cycles within small airways. Such observations have given rise to the "open lung" approach to the ventilatory management of ARDS and to methods that can be used to accomplish it [2]. Doubters cogently argue that complete lung opening is unnecessary, impossible to accomplish, and requires excessive airway pressure to attain and sustain. This is not an easily settled issue, and although we are slowly making progress, there is still no agreement on the best way to find the "open lung" position.

DiRocco, Carney and Nieman use their novel technique of in vivo video-microscopy and a small animal model of airway instability to address the question of which feature of the static airway pressure–volume (PV) loop correlates best with the lowest "open lung" PEEP [1]. In reporting their experience with directly observed, surfactant-depleted lung tissue, these authors confirm both that alveolar recruitment in this rat model is not completed until the lungs reach total lung capacity (TLC) and that

the upper inflection point on the deflation limb of the PV curve (UIPd) better indicates the least pressure associated with full recruitment than does the traditionally used lower inflection point (LIP) obtained during inflation. To a point, this makes good sense and resonates with recent theory [3], experimental findings [4] and clinical trial evidence [5]. However, we need to be very careful not to confuse any such *global* measure with certainty regarding *regional* behavior.

Conceptually, there is a "disconnect" between flow, volume, and pressure observations made at the airway opening (which pool information from all lung units) with the events that occur in any specific lung region. The contours of the recordable PV curve—inflation or deflation limb—can only describe the volume or pressure at which tissue overdistension or recruitment *predominates*, not the point at which either no longer occurs. Furthermore, in real-world clinical settings the LIP and UIPd are usually indistinct *zones* that require mathematical curve fitting to estimate, not visually distinct "points" with unequivocal identity features.

We all learned lung physiology from useful but highly oversimplified "balloon on a stick" models that ignore the complexities of diseased anatomy, interdependence and regional behaviors. With that single-compartment healthy lung model in mind, and unintentionally disregarding the mechanical hysteresis of the ARDS lung, easily measured indices of recruitment and overinflation have been popularized that are based on the characteristics of the inspiratory PV curve. Somewhat surprisingly, the LIP, first advocated for the clinical setting by Matamis, Lemaire and colleagues [6], has served reasonably well in at least two "lung protective" clinical trials focused on securing an open lung [7, 8]. Moreover, the ease and speed with which the LIP can be determined at the bedside with automated technology adds to its appeal [9]. By serendipity, the inspiratory pressure associated with the LIP often—but not invariably—approximates the pressure associated with the UIPd. Those two inflections were rather widely separated in the surfactant-depletion model of DiRocco and colleagues, but in other models of injury they may be much closer.

Because a truly open lung—one whose every portion is stable during tidal breathing and without atelectatic units—cannot be achieved without applying tidal pressures that are intolerably elevated, the real questions are: How "open" do we need the lung to be? How high a pressure should we tolerate in pursuit of this elusive goal? If a block of atelectatic tissue has a higher opening pressure than ever experienced during tidal ventilation, is persistent collapse dangerous? Conversely, if it opens readily at low pressure, are the repeated shearing forces tolerable? "Full" recruitment is clearly desirable when this helps prevent VILI in a highly recruitable lung but loses value as the number of units in jeopardy declines. For this reason, open lung strategies should prove more useful earlier than later in the clinical course of ARDS.

Raising PEEP implies an increase in plateau pressure, a reduction in tidal volume (and ventilation), or both. Within already open lung units, raising plateau pressure produces greater tissue stretch, and the strains experienced at the junctions of open and persistently closed units rise disproportionately. Disturbingly, recent analyses can find no safe plateau pressure threshold, even within the ventilating range readily accepted in modern practice [10, 11]. Even though several reassuring explanations can be offered for the correlation between end-tidal pressure and mortality, such observations suggest that PEEP-driven increases of plateau pressure should not be undertaken without concern.

Apart from the very small number of animals investigated, the interesting and carefully collected data of DiRocco and colleagues [1] must be questioned on several methodological levels. As the authors point out, this work cannot be directly extrapolated to the clinical arena. The healthy rats used as the subjects of this study not only had anatomic dimensions an order of magnitude smaller than those of large mammals and humans, but were studied with a highly recruitable, saline lavage preparation—not acute injury that simulates usual clinical causes of lung injury. Hysteresis in this model is quite pronounced and not representative of most clinical conditions. Moreover, PV curves that aptly characterize the lungs of a small animal may not correspond well with those obtained in large animals with gravity-influenced regional mechanics and disproportionate collapse in dependent zones. Even if this "size" reservation is set aside, video-microscopy of alveoli at the pleural surface conducted in a non-dependent region of the open chest under gentle suction may not characterize events occurring deeper within the lungs of closed-chest animals.

One clear lesson emerges from this paper and from others attempting to address the optimal PEEP question: Careful intervention and closely observed feedback are essential when formulating a well-reasoned bedside approach to this common clinical problem. Individual differences in pathology, changeable and heterogeneous mechanics, and serious hazards associated with our therapeutic interventions demand firm grounding in the relevant patho-physiology and preclude easy, formula-driven answers to an innately complex challenge.

References

1. DiRocco J, Carney D, Nieman G (2007) Correlation of alveolar recruitment/derecruitment with inflection points on the pressure–volume curve. Intensive Care Med DOI 10.1007/s00134-007-0629-8 (this issue)
2. Lachmann B (1992) Open up the lung and keep the lung open. Intensive Care Med 18:319–321
3. Hickling KG (2001) Best compliance during a decremental, but not an incremental positive end expiratory pressure trial is related to open lung positive end expratory pressure: a mathematical model of acute respiratory distress syndrome lungs. Am J Respir Crit Care Med 163:69–78
4. Crotti S, Mascheroni D, Caironi P, Pelosi P, Ronzoni G, Mondino M, Marini J, Gattinoni L (2001) Recruitment and de-recruitment during acute respiratory failure: a clinical study. Am J Respir Crit Care Med 164:131–140
5. Girgis K, Hamed H, Khater Y, Kacmarek RM (2006) A decremental PEEP trial identifies the PEEP level that maintains oxygenation after lung recruitment. Respir Care 51:1132–1139
6. Matamis D, Lemaire F, Harf A, Brun-Buisson C, Ansquer JC, Atlan G (1984) Total respiratory pressure–volume curves in the adult respiratory distress syndrome. Chest 86:58–66
7. Amato M, Barbas C, Medeiros D, Magaldi R, Schettino G, Lorenzi-Filho G, Kairalla R, Deheinzelin D, Munoz C, Oliveira R, Takagaki T, Carvalho C (1998) Effect of a protective ventilation strategy on mortality in the acute respiratory distress syndrome. N Engl J Med 338:347–354
8. Villar J, Kacmarek R, Perez-Mendez L, Aguirre-Jaime A (2006) A high positive end-expiratory pressure, low tidal volume ventilatory strategy improves outcome in persistent acute respiratory distress syndrome: a randomized, controlled trial. Crit Care Med 34:1311–1318
9. Servillo G, Svantesson C, Beydon L, Roupie E, Brochard L, Lemaire F, Jonson B (1997) Pressure-volume curves in acute respiratory failure. Automated low flow inflation versus occlusion. Am J Respir Crit Care Med 155:1629–1636
10. Terragni PP, Rosboch G, Tealdi A, Corno E, Menaldo E, Davini O, Gandini G, Herrmann P, Mascia L, Quintel M, Slutsky AS, Gattinoni L, Ranieri V (2007) Tidal hyperinflation during low tidal volume ventilation in acute respiratory distress syndrome. Am J Respir Crit Care Med 175:160–166
11. Hager DN, Krishnan JA, Hayden DL, Brower RG (2005) ARDS Clinical Trials Network. Tidal volume reduction in patients with acute lung injury when plateau pressures are not high. Am J Respir Crit Care Med 172:1241–1245