

OPTIMIZING PEEP - NEGOTIATING TOUGH CHOICES

by John Marini MD



In every ventilated patient with ARDS, certain choices are inescapable, and not all are easily made—selecting PEEP, for example. Controversy regarding optimal selection of PEEP stems in large part from imprecision of disease diagnosis, confusion about which objective to prioritize, and uncertainty regarding safe limits for airway pressure. The underlying cause for such confusion, however, involves the mechanical heterogeneity of the acutely diseased lung, coupled with the requirement to set only one PEEP value. In fact, when lung protection is the issue, PEEP selection is always a tradeoff between encouraging recruitment and increasing tissue stress elsewhere in the patchwork lung. How that compromise is best struck at the bedside? Recent awareness of the roles of high alveolar pressures and tidal opening and closure cycles in producing ventilator associated lung injury (VILI) have reshuffled our priorities in selecting PEEP and tidal volume 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.

Using sufficient PEEP dramatically reduces the risk of VILI arising 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. (Another contributor is the associated reduction of driving pressure.) 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. Doubters cogently argue that complete lung opening is unnecessary, impossible to accomplish, and requires excessive airway pressure. In other words, we can never achieve and sustain a fully open lung at acceptable levels of airway pressure. This debate is not an easily settled issue, and although we are slowly making progress, there is still no agreement on the best way to find an 'acceptably open' compromise.

Alveolar recruitment is not completed until the lungs reach total lung capacity (TLC). The upper inflection point on the deflation limb of the P-V curve (UIPd) better indicates the least pressure associated with full recruitment than does the traditionally used lower inflection point (LIP) obtained during inflation. This makes sense and resonates with the suggestions of recent theoretical, experimental and clinical trial evidence. We need to be careful not to confuse any global measure of lung mechanics with certainty regarding events at multiple regional levels. Even assuming that a safe "open lung" point exists, we may not be able to find it at the bedside without more specialized technology than is currently offered by displays of airway pressure and flow. We must acknowledge their limitations accept a settlement.

An inherent 'disconnect' exists 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. At best, the contours of the recordable PV curve—*inflation or deflation limb*—can only describe the volume or pressure at which tissue over distention 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 lower inflection point (LIP), has served reasonably well in at least two "lung protective" clinical trials focused on securing an open lung. Moreover, the ease and speed with which the LIP can be determined at the bedside with automated technology adds to its appeal. By serendipity, the inspiratory pressure associated with the LIP often—but not invariably—approximates the pressure associated with the UIPd.

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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 sector 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 associated 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 high risk lung units 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 lower than which there is no further correlation with mortality. This is true even within the ventilating pressure range readily accepted in modern practice. Even PEEP-driven increases of plateau pressure must not be undertaken without concern.

Returning to the "optimal PEEP" question, careful intervention and closely observed feedback are essential when formulating a well-reasoned approach to this common clinical problem. The recordable airway pressure merges information from various pools of units with very different properties. Unfortunately, any but the most extreme distortions of the PV curve have ambiguous

interpretations. 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.

Dr. Marini, MD, Professor of Medicine at the Univ of Minnesota, is a clinician-scientist whose investigative work has concentrated in the cardiopulmonary physiology and management of acute respiratory failure. In the majority of his research, he has been positioned at the interface between basic physiology and clinical medicine so as to develop insights into advancing clinical practice.



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but how do we pay for the gas?"**