



FOUR FORGOTTEN FACTORS OF RESPIRATORY MECHANICS

by John Marini MD

Understanding how the lungs inflate is fundamental to the delivery of respiratory care to the intubated and ventilated patient. Acknowledging the wealth of experimental and clinical evidence that documents the potential for ill-advised settings to damage the lungs, few would argue that we can ignore the pressures associated with delivery of tidal airflow. But which pressures are important, what values are concerning, and what do the calculated values for resistance and compliance really imply about the lung?

We all learned the elements of lung mechanics from single compartment, "balloon-on-a-straw" models whose mechanical properties are summarized in the simplified "equation of motion" of the respiratory system: $P_{aw} = P_{res} + P_{el} + P_{EEPtot}$. The physical law of energy conservation requires us to account for the pressure applied across the airway opening as the sum of flow-resistive (dissipated) pressure (P_{res}), tidal elastic (conserved) pressure (P_{el}), and the value of end-expiratory pressure from which the inspiratory phase begins (total PEEP, $PEEP_{tot}$). Understanding this core concept of pressure balance is invaluable, especially

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because our standard ventilators measure and display only one pressure and one flow—that registered at the airway opening. Yet, as useful as the pressure readings (and the resistance and compliance calculations based upon them) may be for some purposes, they are downright misleading unless four often-forgotten factors—chest wall stiffness, patient effort, accessible lung capacity, and heterogeneity—are taken into account. Let's consider them one at a time.

Arranged in series, the lungs and chest wall theoretically occupy identical volumes. While the lung is inherently a passive structure, its enveloping container—the chest wall—is not. After flow is stopped by a pause, airway pressures that exceed the starting value expand both the lungs and the chest wall. How much of that displayed airway opening pressure was required to expand the lung and how much the chest wall are determined by their relative stiffness (elastance). Therefore, if the lung and chest wall elastance are similar, the pressure increment measured relative to atmosphere (plateau minus total PEEP) will be evenly split—pleural pressure will rise by the same amount as the pressure that expanded the lung. Trouble is, we seldom know what the lung and chest wall elastances actually are, and their ratio may not stay constant

throughout the tidal volume range. (I will come back to that point a little later.) Stiff chest wall conditions (e.g., extreme obesity or ascites) might make it prudent to accept a plateau pressure considerably higher than the upper recommended value of ~30 cmH₂O if the alternative is failing to meet pressing physiological demands—e.g., metabolic acidosis.

To further complicate matters, thus far I have been implying passivity of the chest wall. If inspiratory effort and muscle tone add to the positive pressure applied at the airway opening, the effective elastance of the chest wall may be less positive or even negative. It follows that any pressure displayed at the airway opening underestimates the pressure that would be recorded under passive conditions—at times seriously so. Unless the clinician has eliminated patient effort, (s)he cannot feel entirely comfortable about a plateau pressure in the 20's. Without the pre-requisite of passivity or of recording pleural (esophageal) pressure, it makes little sense to try to identify a "safe" upper pressure limit to target. Sadly, none of the major randomized clinical trials that have attempted to probe the best ventilatory strategy for ARDS have assured passive conditions or used an esophageal balloon catheter. Resistance calculations, likewise, cannot be relied upon if the alveolar pressure during inspiration is unknown.

Just as troubling as the influence of chest wall characteristics, the values we customarily record for mechanical properties do not take the dimensions of the aerated compartment into account. When characterizing the impedance characteristics of the lung, we usually calculate "resistance" and "compliance", expressing these in absolute measurable units—cmH₂O/L/sec and L/cmH₂O, respectively. But here, too, we are on very shaky ground, because in routine practice (if not research) we generally ignore the size of the subject (expressed as ideal body weight) as we interpret those impedance values. Were we to do so, we would find that even assuming perfect health, the uncorrected value for airway resistance is considerably higher (and uncorrected compliance lower) for a small person than for a large one. Their specific resistance and compliance values, referenced to the lung's capacity to accept gas volume, are theoretically identical. The "time constant" of the respiratory system obtained under passive inflation or deflation conditions (the product of resistance and compliance) is less susceptible to these anatomical size issues, as resistance and compliance move in opposite directions with dimensional variations.



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This "compartment sizing" issue assumes consummate importance in the setting of acute illness, where the capacity of the lung to receive gas routinely is reduced. In other words, the same principle of accounting for innate volume applies to lungs that have been made smaller not by anatomy, but by disease. The capacity of a patient with severe ARDS to accept gas is often reduced by more than two thirds, as fluids, cells, and inflammatory debris fill the remainder. The patient ventilated for acute asthma usually has many air channels blocked off by mucus. Our interpretations of resistance and compliance must take this smaller capacity into account. On the very near horizon, ventilators that determine the lung's resting size by wash-in and wash-out methods promise to compensate for such anatomy and pathological aberrations of lung size when computing mechanics.

There is one more very important principle to consider as we attempt to adjust the ventilator attached to a heterogeneous lung--observe mechanical behaviors and parameters over a testing range, not only at single point. For reasons already covered, airway opening pressure is only an average, and key variables that influence the recorded numbers may not be knowable. For example, even knowing the pleural pressure only helps in estimating global trans-lung pressure, not the local ones. To contend with these problems, a rational principle is to observe changes that occur with a known intervention and thereby infer from the response what the underlying mechanical properties must be. The pressure-volume curve or loop offers a good and familiar example. Looking for inflection and deflection zones alerts the clinician to changes in the underlying mechanics that may hold implications for selecting PEEP and tidal volume when attempting to avoid both widespread over-distention and widespread tidal collapse. A recent innovation along this line to simplify and standardize the process of data collection is to calculate the "stress index", which characterizes shape changes (or lack thereof) that occur during the course of tidal inflation with constant flow. Unfortunately, the absence of a shape change may hide simultaneous over-distention and recruitment within different sectors of a heterogeneous lung and therefore may be falsely re-assuring. Nonetheless, the detection of a radical shape change of the airway pressure tracing more certainly calls attention to the need for settings adjustment.

Advanced imaging techniques such as electrical impedance tomography, vibration and acoustic imaging may allow bedside calculation of heterogeneity indexes that provide insight to lung behaviors in response to pressure, volume and flow. These innovations are still in the hands of developers and investigators, however, and are not yet ready to be widely deployed. In my view, even advanced technologies will be hard pressed to replace the thoughtful practitioner who is armed with a solid understanding of mechanical principles and the dedication needed to determine the requirements and responses of the individual patient. The four forgotten factors must be remembered.

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