Is there a place for esophageal manometry in the care of patients with injured lungs?

Rolf D. Hubmayr
Mayo Clinic College of Medicine, Rochester, Minnesota

As someone who trained in an era in which respiratory physiology occupied center stage in the education of budding pulmonologists, I recall, not so fondly I might add, being locked in a body plethysmograph, chewing on a mouth piece, and, with every swallow, feeling the tuck of the esophageal balloon catheter protruding from my nostril. After all, who wouldn’t want to construct his very own isovolume-pressure-flow curve, even though most of us struggled to really understand its physical basis. Luckily, as we migrated from the physiology research laboratory to the bedside, we soon appreciated that esophageal pressure (Pes)-derived measurements of lung recoil rarely influenced our clinical judgment and, thus, had little impact on our management decisions. The emergence of sleep medicine transiently rekindled interest in esophageal manometry as a component of a “Cadillac sleep montage,” but because there are alternative less invasive means for distinguishing between central and obstructive apneas, the technique never achieved mainstream status. Case in point: a Wikipedia search for esophageal manometry refers to its utility in the diagnosis of esophageal motility disorders, but it is silent on the technique’s role in the management of patients with lung disease.

In this issue of the Journal of Applied Physiology, Loring and colleagues (6) report on the relationships between esophageal, gastric, and bladder pressure in recumbent mechanically ventilated patients with acute lung injury (ALI) and conclude that Pes measurements provide meaningful information, otherwise unavailable, in critically ill patients. It would be easy to dismiss this report as biased by many untested and, indeed, untestable assumptions, were it not for the fact that the same group of investigators had previously reported that, compared with the current standard of care, a ventilator strategy using Pes to estimate transpulmonary pressure (Pl) significantly improves oxygenation and respiratory system compliance of patients with ALI (10). More importantly, the improvement in lung mechanics and gas exchange was paralleled by a strong trend toward favorable clinical outcomes, such as shorter durations of mechanical ventilation and increased survival. These findings raise a question that is central to the debate on optimal ventilator management and the pathogenesis of ventilator-associated lung injury: Is a strategy that embraces the open lung concept at the risk of “overstressing” aerated non-dependent lung parenchyma superior to a strategy that seeks to minimize end-inspiratory airway plateau pressure, while accepting less than full recruitment of consolidated, flooded, and/or collapsed dependent lung?

Skeptics of the validity of Pes-derived estimates of the average lung surface (pleural) pressure (Ppl) will note that, even at end-expiratory airway pressures (PEEP) as high as 20 cmH2O, most patients had a negative apparent transpulmonary pressure (Pl,EES, i.e., Pes,EES exceeded PEEP, (see Fig. 3 of Ref. 6). Since the lung parenchyma is a prestressed network structure that is distorted by surface tension and offers little resistance to tissue folding, it is unreasonable to think that aerated lung units, which communicate freely with the central airways, would ever be exposed to a compressive stress in situ, i.e., would be subjected to a negative transalveolar pressure. Therefore, Pl,EES is likely biased by units that are closed and extrinsically compressed by pleural fluid and/or the weight of abdominal contents. As pointed out by the authors, the high prevalence of obesity, bowel distension, and ascites in critically ill patients with ALI offers an attractive explanation for the relatively close correlations between pressures measured in abdominal hollow organs (stomach and bladder) and the distal esophagus. Moreover, the effects of abdominal weight on Pes explain the lack of correlation between Pes,EES, an estimate of end-expiratory chest wall recoil pressure, and chest wall elasticity (Ecw). Mass loading of thorax and abdomen produces a rightward shift of the chest wall pressure-volume curve, as reflected in the very high end-expiratory Ppl estimates, but need not alter Ecw per se (9). In aggregate then, the report by Loring and colleagues (6) suggests that the mechanical properties of the chest wall of many patients with ALI are abnormal and that changes in chest wall mechanics have bearing on PEEP management and lung function but may not be inferred from measurements of end-inspiratory airway plateau pressure or respiratory system compliance alone. To a clinical audience, the latter point is critical, insofar as most PEEP management trials of ALI have assumed otherwise (2, 7, 8).

The debate as to how to appropriately balance the risk of mechanical ventilation-associated lung injury from “overdistension” against that from “underrecruitment” continues to occupy the critical care literature (1, 3, 5, 11). Esophageal manometry has a chance of informing this debate, provided Loring and colleagues (6) are correct in their assumptions that 1) the bias between Pes and the true average Ppl, the so-called medistinal artifact,” approximates 5 cmH2O, 2) the bias is insensitive to PEEP and lung volume, 3) the bias varies little between patients, and 4) gradients in local Ppl may be inferred from vertical distance measurements. To the purist, these assumptions will forever remain suspect. The topographical distribution of lung surface pressure results from two gravitationally deformed elastic solids (lungs and thorax) having to conform to the same shape (12). Even if one preserved their respective volumes, removal of the lungs from the chest would result in an isovolumic deformation of both structures. If one wanted to restore lung shape, one would have to apply a topographically nonuniform surface pressure, which would mirror the Ppl distribution in situ. There would be a gravitational component proportional to height and weight, but the heterogeneity in pressure would also depend on the weight of...
the heart, the position/shape of the diaphragm/abdomen, and, most importantly, the lungs’ resistance to a shape change (as reflected in its shear modulus). As long as the lung is healthy and its volume (and prestress) is low, its behavior is similar to that of a fluid; i.e., very little energy is required to effect an isovolumic shape change. Under these conditions, lung density becomes more or less the sole determinant of the gravitational pressure gradient. At high volumes (and high prestress), however, the lung becomes stiff, and even small deviations from the shape that would have been observed in a uniformly inflated state (e.g., ex vivo) produce large local stress gradients. The stress distribution in the injured lung is even more heterogeneous, because altered barrier properties promote alveolar flooding, occlusion of small airways by liquid plugs, small-scale variability in surface tension, and varying degrees of gas absorption atelectasis (4). These mechanisms conspire to increase the apparent shear modulus, as well as density and weight of the lungs. Given the greatly increased topographical variability in surface (pleural) pressure of injured lungs, what then is the chance that the pressure anywhere along the lumen of the esophagus informs about the average lung stress?

Although the purists may puzzle over the complexity of the problem and agonize over the lack of a “gold standard” for characterizing the topographical distribution of lung stress in health, let alone disease, we must not forget that an exploratory trial of Pes-guided ventilator management already exists and that it suggests clinical benefit (10).

If this result withstands the test of time, we will have to conclude that 1) assumptions of Loring and colleagues (6) about the validity of Pes-based Ppl estimates are correct in most instances or, more importantly, 2) assumptions concerned with the maintenance of low airway plateau pressures, irrespective of associated PEEP and tidal volume settings, may have overestimated the risk of lung injury from overdistension. In either case, we will have learned an important lesson: data are always more informative than mind experiments.

DISCUSSIONS

No conflicts of interest are declared by the authors.

REFERENCES


