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EDITORIAL

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Can we rely on "calibrated" central venous pressure to measure pleural pressure at the bedside?

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Measuring the transpulmonary (alveolar–pleural) pressure may be important. However, estimating pleural pressure requires esophageal manometry, which is only available at selected centers. Previous studies have suggested that ventilation-induced changes in central venous pressure (ΔCVP) may reflect those in pleural pressure (ΔPpl) [1], but results have been conflicting.

In this issue of the Journal, Kyogoku et al. describe a method for predicting ΔPpl from ΔCVP after calibrating the two during an occlusion test [2]. The study involved ten mechanically ventilated pigs with acute lung injury and an intrathoracic catheter to measure ΔPpl directly (reference technique), an esophageal balloon to measure changes in esophageal pressure (ΔPes), and an intrathoracic central venous catheter to measure ΔCVP. The method involves four steps. First, compress the animal's chest during an end-expiratory airway occlusion maneuver and record the change in airway pressure (ΔPaw) and ΔCVP. The lung volume remains constant with an occluded airway, so ΔPaw should reflect ΔPpl. Second, calculate the calibration coefficient "k" as the ratio of ΔPaw (≈ΔPpl) to ΔCVP. It represents ΔPpl for each 1-cmH_2O change in CVP. Third, resume ventilation and record ΔPaw and ΔCVP during an end-expiratory and end-inspiratory occlusion maneuver. Fourth, multiply this ventilation-induced ΔCVP by k to estimate the corresponding (ΔCVP-derived) ΔPpl. This method was also used in animals with low or high intravascular volume or intrabdominal hypertension.

During the occlusion tests, k was 2.2 ± 1.3, suggesting reduced venous return or partial transmission of ΔPpl to the right heart. During ventilation, ΔPpl was 7.6 ± 4.5 cmH_2O, ΔPes 7.2 ± 3.6 cmH_2O, and ΔCVP-derived ΔPpl 8.0 ± 4.8 cmH_2O. In the Bland–Altman analysis, the bias between ΔPpl and ΔCVP-derived ΔPpl was −0.3 cmH_2O, and between ΔPpl and ΔPes, 0.5 cmH_2O. The 95% limits of agreement (LOA) ranged from −4.1 to 3.4 cmH_2O and from −2.8 to 3.9 cmH_2O respectively. These results were consistent across all experimental conditions and indicate that calibrated CVP was accurate (small bias) but not precise enough to be deemed clinically acceptable (wide 95%-LOA and an average percentage error of around 50%) [3]. Of note, during the occlusion test, the positive end-expiratory pressure (PEEP) was 0 cmH_2O and the lung volume and transpulmonary pressure were constant. During ventilation, PEEP was 6 cmH_2O, and lung volume and transpulmonary pressure increased. It is possible that lung inflation also affected the ΔCVP and the precision of the estimates, which (instead) assumed a constant k.

ΔCVP-derived ΔPpl was as good or bad as ΔPes in estimating ΔPpl, which is surprising. Esophageal manometry was reliable in other studies where ΔPpl was measured with flexible flat, wafer-type, air-filled balloons [4, 5]. Here it was measured with an intrathoracic method at risk of compression or distortion. In 7/60 occlusion tests, ΔPpl unexpectedly differed by more than 20% from ΔPaw.

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To sum up, using an occlusion test to calibrate the ΔCVP against ΔPaw is a welcome step in measuring transpulmonary pressure. However, the estimates are still too imprecise. Further studies are needed to understand better the intricate impact of changes in intrathoracic pressure on the cardiopulmonary system.

Author contributions
AP conceived the manuscript. MC revised the manuscript for important intellectual content. Both authors read and approved this final version of the manuscript.

Declarations

Conflict of interest
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