

PEEP of 14 cm H₂O) in the setting of requiring three inotropic-vasopressor medications (MAP of 72 mm Hg and heart rate of 94 bpm). During the recruitment maneuver, \dot{Q} increased by 2.7 L/min, stroke volume increased by 8 ml/beat (both measured by transpulmonary thermodilution), and MAP increased by 20 mm Hg (Figure 1 [case 2]). We also observed a decreased stroke volume variation (from 16% to 6%) and no changes in central venous pressure (13–14 cm H₂O). His heart rate was 103 bpm. PEEP was set at 20 cm H₂O. The improved blood pressure allowed the epinephrine dose to be decreased rapidly during and in the hours after lung recruitment (no fluid challenges were administered).

Taken together, both the data presented in our manuscript and these two cases make us hypothesize that *high pleural pressure acts like a shield for the cardiovascular system against high ventilator pressures*. Prior investigations of the effects of positive-pressure ventilation on heart–lung interactions did not examine the role of high baseline pleural pressure (3–5).

As shown in the patients represented in Figure 1, the counterbalance of high pleural pressure due to obesity permitted the use of high airway pressures that not only improved respiratory system compliance (reduced driving pressure) and lung volumes but also raised \dot{Q} and systemic blood pressure. The increased pleural pressure prevents high-airway-pressure lung injury (6) and hemodynamic collapse (7) that might otherwise occur during high-pressure ventilation in patients with low baseline pleural pressures. Future studies are needed to determine the validity of our hypothesis. ■

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Bedside Evaluation of Pulmonary Embolism by Saline Contrast-enhanced Electrical Impedance Tomography: Considerations for Future Research



To the Editor:

We read with great interest the article by He and colleagues (1) entitled “Bedside Evaluation of Pulmonary Embolism by Saline Contrast Electrical Impedance Tomography Method: A Prospective Observational Study.” The authors found that pulmonary embolism (PE)-invoked regional perfusion deflection could be detected with saline-contrasted electrical impedance tomography (EIT) and claimed that the method showed high sensitivity and specificity for diagnosis of PE. However, several factors potentially affecting the reported findings should be discussed.

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For measurement of pulmonary perfusion, a short apnea is needed during bolus injection of 10 ml 10% NaCl to eliminate the interruption from cyclic breath. The conscious patients were required to hold their breath at the end of expiration for 8 seconds or longer. Although the shorter the apnea, the more feasible for conscious patients to hold their breath, it needs imperative time to allow blood mixed with saline to travel through the whole pulmonary circulation. Slutsky and colleagues (2) found that mean pulmonary transit time ranged from 4.3 to 12.6 seconds (mean 7.7 ± 1.5 s) in humans. Moreover, acute PE can lead to an increase of pulmonary vascular resistance, which would remarkably prolong pulmonary transit time (3). In this context, it is questionable that a period with a lower level of 8 seconds is enough for saline to pass through the lung in patients with PE.

On the other hand, for those intubated, holding breath for even 8 seconds might be challenging as dyspnea is common among patients with PE; manual expiratory hold is likely to trigger spontaneous breath, which would dramatically impact the intrathoracic electric impedance. To avoid spontaneous breath, sometimes neuromuscular relaxant is needed, which was not detailed in this article.

Recently, Mauri and colleagues published a study exploring the \dot{V}/\dot{Q} ratio in patients with coronavirus disease (COVID-19), in which a lower concentration (5%) of saline and end-inspiration occlusion for 20 seconds were implemented for determination of pulmonary perfusion (4). Compared with breath hold at the end of expiration, inspiratory hold might be more tolerable for patients with dyspnea and seems more practicable owing to the Hering-Breuer deflation reflex. A maximal inflation of lung during inspiratory breath hold can suppress respiratory drive through activation of the pulmonary stretch receptors. Vice versa while holding breath after an expiration. In addition, CO₂ accumulation contributes more to the urge to breathe than O₂ through chemoreceptors; therefore, a larger lung volume is conducive to dilute the increase in metabolically derived CO₂ levels (5).

Finally, because of the low spatial resolutions, EIT is prone to detect large emboli. On the condition that embolism occurs in segmental branches of pulmonary arteries or lower, EIT might be insensitive to such redistribution of local blood flow. To improve the sensitivity of EIT on PE, Nguyen and colleagues (6) assessed the right lung to left lung perfusion ratio of peak value, maximum uptake, maximum washout, and area under the curve of the averaged contrast dilution curve in each lung. It was concluded that the right lung to left lung perfusion ratios of area under the curve and peak value of the averaged contrast dilution curve are the most promising and reliable in assessing PE, suggesting that EIT might detect the difference between normal and embolized lungs with a unilateral perfusion defect as small as 8% of the lung with a bolus of hypertonic saline.

In general, contrast-enhanced EIT is potentially a promising bedside approach in PE diagnosis. However, numerous issues in regard to feasibility, efficacy, and safety need to be addressed before its clinical application. ■

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Reply to Wang and Zhong



From the Authors:

We read the comments on our recently published paper (1) with interest and are delighted with the inspiring discussion.

In their letter to the editor, Drs. Wang and Zhong expressed their concern about breath-holding time being too short, given that a mean pulmonary transit time (PTT) could be as long as 12 seconds. However, PTT is defined as the time requested for blood flow transfers from the right ventricle to the left atrium. To evaluate lung perfusion, apnea time does not need to be as long as PTT. Animal studies suggested that the time from saline injection to saline entering and concentrating in the lungs was about 3–5 seconds (2, 3). For the patients with pulmonary embolism (PE) evaluated in our study (1), the trough value of global impedance was observed at 2–5 seconds after saline bolus injection. Because we evaluate the slope of regional impedance decrease as lung perfusion, even a shorter breath-holding time is theoretically sufficient. In extreme cases with very low- and high- \dot{Q} patients,

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