

LETTER

Ability of respiratory pulse pressure variation to predict fluid responsiveness in ARDS: still an unanswered question?

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See related research by Lakhali *et al.*, <http://ccforum.com/content/15/2/R85>

We read with interest the study of Lakhali and colleagues [1] suggesting that respiratory pulse pressure variation (PPV) is not an accurate predictor of fluid responsiveness in patients with acute respiratory distress syndrome (ARDS) ventilated with low tidal volume (Vt). They stated that the poor performance of PPV is attributed to small changes in respiratory pleural pressure related to low Vt, such that PPV is low even in the case of marked cardiac preload dependency. These findings are not surprising since in this study [1] the mean static compliance was not very low (40 cmH₂O); therefore, the percentage of transmission of alveolar pressure to the pleural space was not really altered. Thus, the ventilation with low Vt induced small variations in pleural and transpulmonary pressures.

It must be stressed, however, that patients with ARDS usually exhibit a severe decrease in lung compliance and

high plateau pressure. Moreover, in ARDS patients ventilated with low Vt, application of relatively high levels of positive end expiratory pressure (between 10 and 15 cmH₂O) is now recommended [2]. Consequently, respiratory changes in transpulmonary pressure should remain greater than normal, and in spite of reduced lung compliance, cyclic changes in intrathoracic pressure may still be high enough for PPV to predict fluid responsiveness [3].

The findings of Lakhali and colleagues [1] perhaps provide insufficient support for the assertion that PPV is unable to predict volume responsiveness in ARDS patients ventilated with low Vt. Additional studies in severe ARDS patients (compliance <30 cmH₂O) are necessary to investigate whether or not PPV could be used in such cases.

Authors' response: In ARDS, stiff lungs keep the pressure inside

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We thank Dr Mallat and colleagues for giving us the opportunity to clarify some physiological points tackled in our study [1]. Their assertion that 'the mean static compliance was not very low(...); therefore, the percentage of transmission of alveolar pressure to the pleural space was not really altered' is in accordance with Jardin and colleagues findings [4] and their recalculated data [5]: the lower the lung compliance, the lower the alveolar-to-pleural space transmission of pressure. By contrast, in the case of lower respiratory system compliance [Crs], the alveolar-to-pleural pressure transmission may be altered in quite different ways, depending on the causal mechanism: stiffer chest wall with no change in lung

compliance leads to an increase in respiratory pleural pressure changes [$\Delta P_{\text{pleural}}$] [6]; and stiffer lungs lead to lower $\Delta P_{\text{pleural}}$ (and lower respiratory variations in pulse pressure ($\Delta_{\text{RESP}} \text{PP}$)) for similar changes in alveolar pressure ($\Delta P_{\text{alveolar}}$). Importantly, the latter is the predominant mechanism of low Crs in ARDS [7].

In ARDS, two phenomena work in opposite directions for a given Vt [4,5]: (i) high P_{alveolar} swings increase $\Delta P_{\text{pleural}}$ but (ii) high lung stiffness prevents P_{alveolar} from being transmitted to the pleural space, therefore lowering $\Delta P_{\text{pleural}}$. Confusion may arise when one takes the 'pressure transmission-reasoning shortcut' and thinks, as Dr Mallat and colleagues do, that the first phenomenon outweighs the second. Actually, basic physiological equations ($\Delta P_{\text{pleural}} = Vt / \text{Chest wall compliance}$) remind us that $\Delta P_{\text{pleural}}$ depends only on the Vt [5] and chest wall compliance [6] and not on lung compliance: the increase in P_{alveolar} (induced by decreased lung compliance) does not outweigh, but exactly compensates, the decrease in

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alveolar-to-pleural space transmission of pressure, as previously observed [4,5]. Further, other limitations for $\Delta_{\text{RESP}}^{\text{PP}}$ (heart-to-respiratory rate ratio or acute cor pulmonale [1]) would become even more prominent in a severe ARDS population, and further studies are likely to yield the same conclusion: $\Delta_{\text{RESP}}^{\text{PP}}$ fails to predict fluid responsiveness in ARDS.

Abbreviations

ARDS, acute respiratory distress syndrome; Crs, respiratory system compliance; P_{alveolar} , alveolar pressure; P_{pleural} , pleural pressure; PPV, respiratory pulse pressure variation; Vt, tidal volume.

Competing interests

The authors declare that they have no competing interests.

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