CRITICAL CARE

COMMENTARY

Why do pulse pressure variations fail to predict the response to fluids in acute respiratory distress syndrome patients ventilated with low tidal volume?

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

Abstract

Respiratory-associated variations in stroke volume and pulse pressure are frequently used to predict the response to fluid administration. However, it has been demonstrated that low tidal volume ventilation may limit their use in patients with acute respiratory distress syndrome (ARDS). In this issue, a trial investigates the value of pulse pressure variation to predict fluid responsiveness in a large series of patients with ARDS ventilated according to current guidelines.

Fluid management in patients with acute respiratory distress syndrome (ARDS) is particularly difficult [1]. In hemodynamically stable patients fluid restriction is warranted as it decreases the length of need for ventilatory support [2]. However, at the initial phases, patients with ARDS also often present hemodynamic instability and are at risk of tissue hypoperfusion and even tissue hypoxia, which may further contribute to exacerbation of ARDS by boosting activation of inflammation and coagulation [3,4]. Guidance of fluid administration is often complicated by the high pleural pressures, associated with high positive end-expiratory pressure (PEEP) levels, that affect measurements of intravascular pressures. Multiple studies have shown that static indices of preload, being pressures or volumes, often fail to predict the response to fluids. On the contrary, dynamic indices based on heart-lung interactions, such as pulse pressure variations (ΔPP), have repeatedly been found to reliably predict the response to fluids in mechanically ventilated patients.

In patients with ARDS, ventilation with low tidal volume is recommended [5]. In patients ventilated with low tidal volume, pulse pressure variations do not predict adequately the response to fluids [6-8]. In this issue of *Critical Care*, Lakhal and colleagues [1] confirm these findings. In 65 patients with ARDS, Lakhal and colleagues [1] observed that pulse pressure variations moderately predicted the response to fluids and that the predictive value was equivalent to that of pulmonary artery pressure.

What does the study by Lakhal and colleagues [1] add to the current literature? First, this trial confirms that pulse pressure variations fail to predict fluid responsiveness in a large series of patients with ARDS ventilated according to current guidelines. Second, this trial tried to evaluate several of the potential mechanisms implicated.

In particular, Lakhal and colleagues [1] evaluated the impact of driving pressure. Indeed, it has been advocated that changes in pleural pressure may be preserved, as lung compliance is also reduced in ARDS patients. The issue is that changes in pleural pressure cannot be reliably estimated from the difference between plateau and end-expiratory pressure [9,10] as the transmission of pressure from airway to pleura markedly varies among patients. Even selecting patients with large driving pressure failed to improve the predictive value of ΔPP. These results are in line with the observations of Vallée and colleagues [8], who found that correcting DPP by driving pressure failed to improve the predictive value for fluid responsiveness. Interestingly, in the few patients with a difference between inspiratory and expiratory pulmonary artery pressure higher than 4 mmHg, the prediction of ΔPP was excellent (area under the curve 1.0 (95% confidence interval 0.73 to 1.0)). This suggests that these indices can be used when changes in alveolar pressure are effectively transmitted to pleural pressure. Unfortunately, this

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requires invasive measurements of intravascular pressures by pulmonary artery catheter or of esophageal pressure.

Another important factor may be that respiratory rate is often high when ventilating with low tidal volume. We observed that ΔPP was negligible in fluid responders when the ratio of heart rate to respiratory rate was decreased below 3.6 by increasing respiratory rates [11]. Muller and colleagues [7] recently confirmed that ΔPP can be low in fluid responders when this ratio is low. Lakhal and colleagues [1] confirmed the combined influence of tidal volume and respiratory rate. Using a composite index computed as the product of tidal volume by heart rate divided by respiratory rate, they observed that ΔPP was significantly larger in responders than in non-responders only in patients with above median values of this composite index.

It is important to acknowledge the limitations of ΔPP and related indices [12]. When applied correctly, these indices can adequately predict the response to fluids. More importantly, resuscitation strategies based on these indices are associated with better hemodynamic stability and lower incidence of postoperative organ dysfunction [13]. In patients with ARDS, the use of these indices is unfortunately limited by several factors, including low tidal volume, high respiratory rate and right ventricular dysfunction.

Abbreviations

ARDS, acute respiratory distress syndrome.

Competing interests

The authors declare that they have no competing interests.

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