Commentary

Pulse pressure variation: beyond the fluid management of patients with shock

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Abstract

In anesthetized patients without cardiac arrhythmia the arterial pulse pressure variation (PPV) induced by mechanical ventilation has been shown the most accurate predictor of fluid responsiveness. In this respect, PPV has so far been used mainly in the decision-making process regarding volume expansion in patients with shock. As an indicator of the position on the Frank–Starling curve, PPV may actually be useful in many other clinical situations. In patients with acute lung injury or with acute respiratory distress syndrome, PPV can predict hemodynamic instability induced by positive end-expiratory pressure and recruitment maneuvers. PPV may also be useful to prevent excessive fluid restriction/depletion in patients with pulmonary edema, and to prevent excessive ultrafiltration in critically ill patients undergoing hemodialysis or hemofiltration. In the operating room, a goal-directed fluid therapy based on PPV monitoring has the potential to improve the outcome of patients undergoing high-risk surgery.

In the previous issue of Critical Care, Keyl and colleagues [1] have investigated the effects of cardiac resynchronization therapy on arterial pulse pressure variation (PPV). Many studies [2] have shown that PPV is much more accurate than cardiac filling pressures and volumetric markers of preload to predict fluid responsiveness (that is, the hemodynamic effects of volume loading). PPV is also more reliable than other dynamic parameters such as systolic pressure variation [3,4] or pulse contour stroke volume variation [4]. In this respect, PPV is used increasingly in the decision-making process regarding volume expansion in patients with hemodynamic instability [2]. Limitations to the use of PPV do exist (mainly active breathing, cardiac arrhythmia, and low tidal volume) and have been described in detail elsewhere [2,5].

It is very important to point out that PPV is not an indicator of volume status, nor a marker of cardiac preload, but is an indicator of the position on the Frank–Starling curve [2]. Briefly, patients operating on the flat portion of the Frank–Starling curve are insensitive to cyclic changes in preload induced by mechanical inspiration, such that PPV is low (Figure 1). Conversely, PPV is high in patients operating on the steep portion of the preload/stroke volume relationship (and hence sensitive to cyclic changes in preload induced by mechanical inspiration) (Figure 1). This information has so far been used mainly to predict fluid responsiveness in patients with shock, but actually could be useful in many other clinical situations.

PPV and fluid depletion/restriction

As an indicator of the position on the Frank–Starling curve, PPV is as useful to predict the deleterious hemodynamic effects of fluid depletion as it is to predict the beneficial effects of fluid loading [6]. In critically ill patients undergoing hemodialysis or hemofiltration the volume of ultrafiltration is often determined roughly on the basis of body weight gain or fluid balance, and is further adjusted in case of hemodynamic instability. In patients with acute respiratory distress syndrome, a therapeutic strategy based on fluid restriction/depletion has been shown to shorten the duration of mechanical ventilation and intensive care [7]. In such clinical situations, fluid management could be refined by PPV monitoring: a large PPV or an increase in PPV indicates that the patient is operating on the steep portion of the Frank–Starling curve, and hence indicates that further ultrafiltration or further fluid restriction/depletion will induce hemodynamic instability.

PPV and respiratory settings

The first description of PPV [8] was a study showing that the parameter can be used to predict the deleterious hemodynamic effects of positive end-expiratory pressure. We must

PPV = pulse pressure variation.
Determinants of pulse pressure variation. Pulse pressure variation (PPV) is a marker of the position on the Frank–Starling curve, not an indicator of blood volume or a marker of cardiac preload. Increasing preload induces a decrease in PPV (from \( \Theta \) to \( \Phi \)). PPV is minimal when the heart is operating on the plateau of the Frank–Starling curve (\( \Theta \) and \( \Phi \)). Decreasing preload induces an increase in PPV (from \( \Phi \) to \( \Theta \)), also increasing contractility (from \( \Theta \) to \( \Phi \)).

PPV and perioperative fluid optimization

Another potential field of application for PPV is the intraoperative fluid optimization of patients undergoing high-risk surgery. Several studies [11-13] have shown that monitoring and maximizing stroke volume by fluid loading (until the stroke volume reaches a plateau, actually the plateau of the Frank–Starling curve) during high-risk surgery is associated with improved postoperative outcome. The benefit in using such a peroperative fluid strategy was first established in patients undergoing cardiac surgery or hip surgery, and has been extended more recently to patients undergoing major bowel surgery or general surgery [11-13]. This strategy has so far required the measurement of the stroke volume by a cardiac output monitor. By increasing cardiac preload, volume loading induces a rightward shift on the preload/stroke volume relationship and hence a decrease in PPV (Figure 1).

Patients who have reached the plateau of the Frank–Starling relationship can be identified as patients in whom PPV is low. The clinical and intraoperative goal of ‘maximizing stroke volume by volume loading’ can therefore be achieved simply by minimizing PPV. A large multicenter trial is currently ongoing to investigate whether minimizing PPV by volume loading may improve the postoperative outcome of patients undergoing high-risk surgery.

PPV as a tool to track changes in contractility?

In the previous issue of Critical Care, Keyl and colleagues [1] reported a slight but significant increase in PPV (from 5.3% to 6.9%) during resynchronization therapy. Although the noninvasive method used by the authors to monitor blood pressure lacks validation, their finding makes sense since increasing left ventricular contractility means increasing the slope of the Frank–Starling curve, and hence increasing PPV (Figure 1). This result also suggests that PPV may be used to track changes in contractility in situations where changes in preload are unlikely. Keyl and colleagues did not, however, assess left ventricular contractility (for example, by measuring the maximum left ventricular pressure derivative, \( dP/dt_{\text{max}} \)). Moreover, biventricular pacing may induce a decrease in left ventricular volumes [14], which may also explain the increase in PPV. The relationship between changes in PPV and changes in contractility during cardiac resynchronization therefore remains to be proven.

Competing interests

The authors declare that they have no competing interest.

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