

## COMMENTARY

# Respiratory variation in inferior vena cava diameter: surrogate of central venous pressure or parameter of fluid responsiveness? Let the physiology reply

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See related research by Muller *et al.*, <http://ccforum.com/content/16/5/R188>

### Abstract

In the previous issue of *Critical Care*, Muller and colleagues investigated whether respiratory variation in inferior vena cava diameter ( $\Delta$ IVC) could be a useful predictor of fluid responsiveness in spontaneously breathing patients. The study concludes that accuracy was not very good and therefore that this parameter should be used with caution in these patients. There is still confusion about the meaning of IVC respiratory variations, whether the patient is spontaneously breathing or mechanically ventilated. In this brief commentary, we try to summarize as clearly as possible the significance of IVC variation in different clinical settings.

In the previous issue of *Critical Care*, Muller and colleagues [1] investigated whether the respiratory variations of inferior vena cava diameter ( $\Delta$ IVC) could be an accurate predictor of fluid responsiveness in spontaneously breathing patients with acute circulatory failure. The main result of this study is that  $\Delta$ IVC predicted fluid responsiveness moderately well: a  $\Delta$ IVC value above 40% was associated slightly with fluid responsiveness, whereas values under 40% were inconclusive. The main conclusion is that  $\Delta$ IVC should be interpreted with caution in spontaneously breathing patients.

Fluid responsiveness is crucial in the management of a patient in shock. Hypovolemia is associated with a worse

outcome, but fluid overload is also associated with increased mortality [2], rendering somewhat hazardous the management of fluids in the most critically ill patients by using fluid challenges alone. In the last 10 years, many clinical and experimental studies on this subject have been carried out, leading to the validation in mechanically ventilated patients of a few 'dynamic' parameters based on heart-lung interactions. Notably, a significant dilation of the IVC during tidal ventilation accurately predicts fluid responsiveness [3,4].

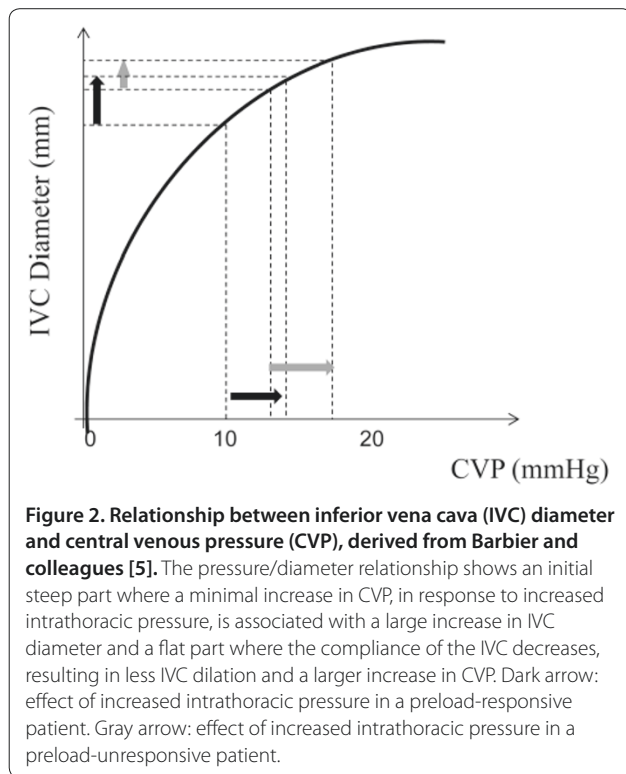
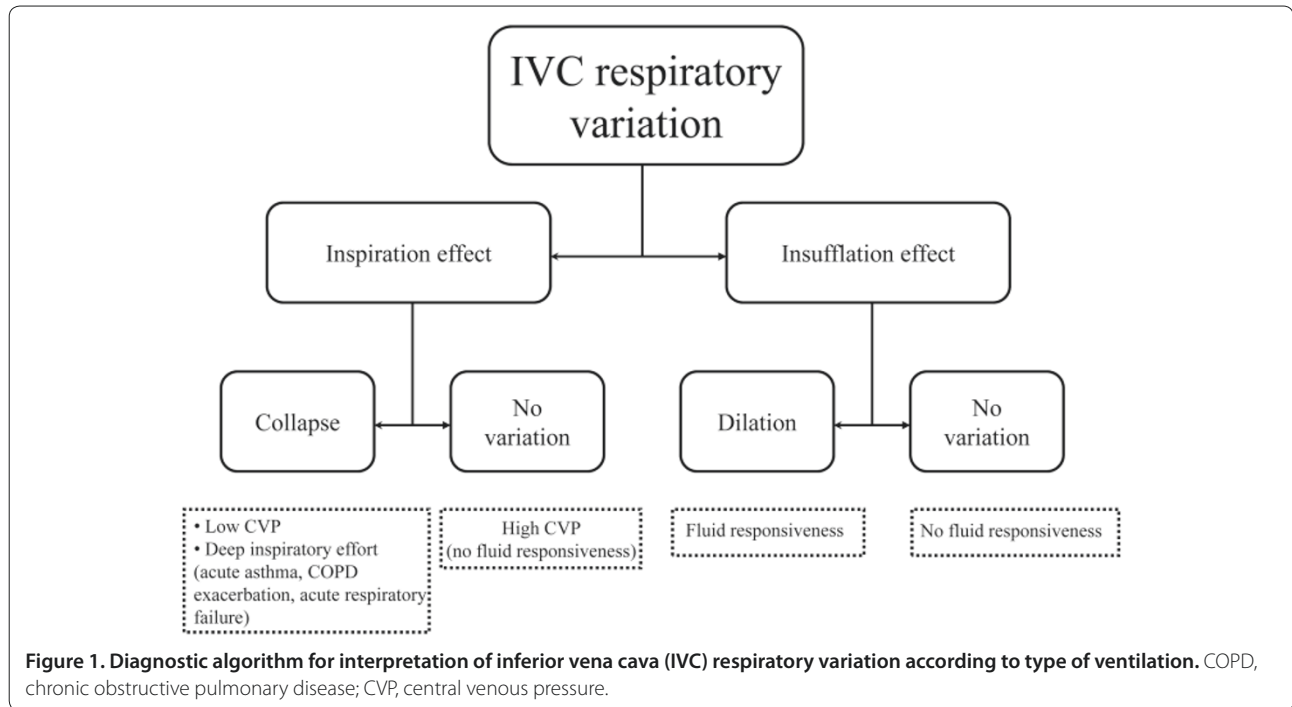
However, there is still some confusion regarding the study of the IVC, and of its respiratory changes, in the intensive care unit. The paper by Muller and colleagues [1] offers an opportunity to try to re-emphasize the basic physiology of the IVC (Figure 1).  $\Delta$ IVC depends on few factors: the intrathoracic and abdominal pressures, the central venous pressure (CVP), and the compliance of the vessel. In mechanically ventilated patients, the objective of studying the IVC is to assess its ability to dilate during tidal ventilation, when intrathoracic pressure is increasing more than abdominal pressure. This dilation actually reflects the ability of the IVC to receive more volume (preload reserve), like a preserved compliance. The IVC is then on the steep part of the relationship between IVC diameter and CVP (Figure 2). As reported by Barbier and colleagues [5], such a relationship is curvilinear. In contrast, the absence of significant dilation reflects the inability of the IVC to receive more fluid (no preload reserve), owing to low compliance. The IVC is then on the flat part of its relationship with CVP (Figure 2).

In spontaneously breathing patients, the situation is completely different. Now, the objective of studying the IVC is not to evaluate its ability to dilate but its ability to collapse in response to a decrease in intrathoracic pressure and an increase in abdominal pressure. In such a situation, changes in IVC diameter reflect simply the interaction between CVP and the range of gradient

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between intrathoracic and abdominal pressures. In other words, the vein may collapse either because the CVP is very low or because the intrathoracic pressure becomes

markedly negative. The latter situation occurs in severe acute asthma [6], exacerbation of acute chronic obstructive pulmonary disease, or any marked respiratory failure. In 1981, Mintz and colleagues [7] described a positive relationship between IVC diameter during expiration and right atrial pressure (RAP). In 1990, Kircher and colleagues [8] reported the value of IVC collapsibility in predicting RAP in patients who breathed quietly and hence did not develop any important variation of intrathoracic pressure. A collapsibility of greater than 50% indicated an RAP of below 10 mm Hg, and a collapsibility of less than 50% indicated an RAP of above 10 mm Hg [8]. In 2007, Osman and colleagues [9] found that a CVP of less than 10 mm Hg could not reliably distinguish responders from non-responders to fluids but that a CVP of greater than 15 mm Hg correctly identified non-responders. Therefore, one can hypothesize that the absence of respiratory changes in IVC diameter, reflecting elevated CVP, should be associated with fluid unresponsiveness but that respiratory changes in IVC diameter, reflecting a normal or low CVP, are inconclusive. Surprisingly, the findings of Muller and colleagues [1] indicate the opposite: a significant IVC diameter variation was associated with fluid responsiveness, whereas the absence of variation was inconclusive. This discrepancy may be explained by the study population. As reported in Table 2 of the article [1], 40% of the patients had hemorrhagic or hypovolemic shock, with a  $\Delta$ IVC expected to be high (many patients actually had a complete collapse).

In conclusion, as re-emphasized by Muller and colleagues [1], it seems hazardous to manage fluids in a spontaneously breathing patient by using IVC respiratory variations only, until further data are published. Alternative methods, such as passive leg raising [10] and a mini-fluid challenge [11], could be used.

#### Abbreviations

CVP, central venous pressure; IVC, inferior vena cava; RAP, right atrial pressure.

#### Competing interests

The authors declare that they have no competing interests.

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