We thank Dr Moretti for his interest in our study. First of all, we would like to clarify the goal of our study. The main purpose of the study was to test the clinical applicability of respirophasic variations of the inferior vena cava diameter, termed IVCCI, for noninvasive detection of elevated central venous pressure (CVP). The study was not designed to show that CVP or IVCCI is a good static or dynamic “preload” indicator. With this in mind, discussing the issue raised by Dr Moretti should help improve our understanding of the ventilator–vascular–pulmonary interactions and their impact on “preload” indicators, including IVCCI, CVP and variations in aortic pressure or flow.

Regarding the first comment by Dr Moretti, we agree that both the ventilatory setting during the measurements and the lung compliance of the studied subjects affect the IVCCI. All the mechanically ventilated patients in our study had a positive end-expiratory pressure (PEEP) of 3–5 mmHg and peak inspiratory pressure (PIP) of 13–18 mmHg (pressure-controlled ventilation is much more common than volume-controlled ventilation in pediatric patients). We did not standardize the ventilatory setting during the measurements, and this could have, to some extent, contributed to the variations in the data of mechanically ventilated patients. On the other hand, we had found in our preliminary study that increasing the PIP over 20 mmHg, to maximize cardiopulmonary interaction, minimally affected IVCCI regardless of CVP level. This is also consistent with data reported by Dr Moretti and co-workers, suggesting a significant overlap in the CVP–IVCCI relationship even under the standardized ventilatory setting (data in Table 1 of their paper); the mean IVCCI under the standardized ventilatory setting with PEEP of 0 mmHg and tidal volume of 8 ml/kg was very low and a difference of 6% in IVCCI corresponds to a difference in only 2 mmHg of CVP. Therefore, it appears that under mechanical ventilation, low IVCCI at baseline hinders the use of IVCCI to discriminate patients with elevated CVP from those without. However, because we did not test the impact of PEEP of 0 mmHg (although it was tested in Dr Moretti’s study in adults), and because children have higher lung compliance than adults, as suggested by Dr Moretti, it may be worth studying whether measurements of IVCCI at 0 mmHg of PEEP could improve the predictive power of IVCCI for elevated CVP in mechanically ventilated children.

With regard to the second comment, it must be noted that selection of parameters that can be used to assess preload condition depends on the definition of preload, and the definition of preload can be diverse depending on what researchers and/or clinicians want to know. For a precise assessment of hemodynamics, it is also important to understand that CVP, like other hemodynamic parameters such as stroke volume and arterial pressures, denotes an integrated hemodynamic state determined by interactions between ventricular systolic- and diastolic-properties and loading properties (ventricular–vascular interaction). The pulmonary system also contributes to such an interaction. Therefore, we should interpret these parameters as such. In this sense, CVP can be termed a “preload” index, but one cannot simply say that high CVP indicates high volume status. Similarly, variations in arterial pressure or IVCCI during controlled ventilation can be defined as a dynamic “preload” index, but it does not necessarily indicate volume responsiveness, because volume responsiveness is primarily determined by the ventricular–vascular interaction. For example, patients with ventricular systolic- and diastolic-stiffening should exhibit large pressure variations upon changes in preload and afterload with mechanical ventilation, but volume-loading should result in marked increases in systolic and diastolic pressures with minimal increase in stroke volume. They could even develop venous congestion with the volume-loading. These phenomena can be amplified also by arterial stiffening. Fluid challenge based on large variations in IVC diameter in such patients can also lead to similar hemodynamic derangement.

To conclude, our study demonstrated that IVCCI under spontaneous breathing accurately predicts elevated CVP in pediatric patients with heart disease. Whether the information of elevated CVP determined by noninvasive measurement of IVCCI in a given patient is useful clinically depends really on how well one understands the underlying pathophysiology of elevated CVP as an index of “preload”. Likewise, if one obtains variations in arterial pressure or IVC diameter in a mechanically ventilated patient, one should understand the underlying ventricular–vascular pathophysiology, and interpret the data accordingly. Otherwise, the condition of some patients with large variations in pressure or IVC diameter as an indicator of “dynamic preload” can worsen by fluid infusion.

References

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