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ORIGINAL PAPER

Accuracy of Respiratory Variation in Inferior Vena Cava Diameter to Predict Fluid Responsiveness in Children Under Mechanical Ventilation

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Abstract

Proper assessment of fluid responsiveness using accurate predictors is crucial to guide fluid therapy and avoid the serious adverse effects of fluid overload. The main objective of this study was to investigate the accuracy of respiratory variations in inferior vena cava diameter (ΔIVC) to predict fluid responsiveness in mechanically ventilated children. This prospective single-center study included 32 children (median age and weight of 17 months and 10 kg, respectively) who received a fluid infusion of 10 ml kg^{-1} of crystalloid solutions over 10 min. ΔIVC and respiratory variation in aortic blood flow peak velocity (ΔVpeak) were determined over one controlled respiratory cycle before and after fluid loading. Thirteen (41%) participants were fluid-responders. ΔIVC, ΔVpeak, stroke volume index, and cardiac index were found to be predictors of fluid responsiveness. However, the area under the ROC curve of ΔIVC was smaller when compared to ΔVpeak (0.709 vs. 0.935, $p < 0.012$). The best cut-off values were 7.7% for ΔIVC (sensitivity, 69.2%; specificity 78.9%, positive predictive value, 69.2%; and negative predictive value, 78.9%) and 18.2% for ΔVpeak (sensitivity, 84.6%; specificity, 89.5%; positive predictive value, 84.6%; negative predictive value, 89.5%). Changes in stroke volume were positively correlated with ΔIVC ($\rho = 0.566$, $p < 0.001$) and ΔVpeak ($\rho = 0.603$, $p < 0.001$). A significant correlation was also found between changes in MAP and ΔVpeak ($\rho = 0.382$; $p = 0.031$), but the same was not observed with ΔIVC ($\rho = 0.011$; $p = 0.951$). In conclusion, ΔIVC was found to have a moderate accuracy in predicting fluid responsiveness in mechanically ventilated children and is an inferior predictor when compared to ΔVpeak.

Keywords Hemodynamic monitoring · Point-of-care ultrasound · Fluid responsiveness · Inferior vena cava ultrasound · Child · Infant

Abbreviations

ΔIVC	Respiratory variation of inferior vena cava diameters
ΔVpeak	Respiratory variation in aortic blood flow peak velocity
AUROC	Area under the ROC curve
Ci	Cardiac index
Da	Aortic diameter
IQR	Interquartile range
IVC	Inferior vena cava
MAP	Mean arterial pressure
NPV	Negative predictive value
PICU	Pediatric intensive care unit
POCUS	Point-of-care ultrasound
PPV	Positive predictive value
ROC curve	Receiver operating characteristic curve
SV	Stroke volume
SVi	Stroke volume index
TTE	Transthoracic echocardiography
VTI	Velocity–time integral

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Introduction

Fluid resuscitation is the mainstay of therapy in children with hypovolemic or septic shock. However, fluid bolus administration is not without risk or harm. Misguided fluid therapy can lead to fluid overload, which has been independently associated with poor in-hospital outcomes, such as impaired renal function, acute respiratory distress syndrome, prolonged mechanical ventilation, longer PICU stay, and increased mortality [1]. The main objective of fluid loading is to increase cardiac output. Unfortunately, only about 50% of children who receive a fluid loading have a significant increase in their stroke volume (SV) [2]. This low rate highlights the limited ability of attending physicians to predict the hemodynamic effects of fluid loading and the need for more accurate methods for hemodynamic assessment. Commonly used static variables, such as heart rate, blood pressure, or central venous pressure, have been shown to be poor predictors of fluid responsiveness in children [2]. Although dynamic variables based on heart–lung interactions are more accurate predictors than static variables, they are not widely available. In addition, the use of dynamic variables is limited in children, as the most studied methods require arterial lines and/or central venous catheterization [2–4].

Point-of-care ultrasound (POCUS) has been proposed as a valuable method for hemodynamic assessment in critical care patients. The respiratory variation of inferior vena cava diameters (ΔIVC) is among the most popular POCUS techniques used to predict fluid responsiveness. The IVC is a thin-walled vessel with very high compliance which diameter is affected by central venous pressure and cyclic changes variable for fluid responsiveness prediction. High values of ΔIVC suggest that the cardiovascular system is operating in the ascending portion of the Frank-Starling curve and will benefit from fluid loading [5, 6]. Conversely, patients whose IVC has low respiratory variability are unlikely to increase their SV with fluid infusion.

The first studies investigating the ΔIVC were published in 2004 and since then there has been an intense debate about the reliability of its measurements [5–7]. Although initial data showed good accuracy, a recent meta-analysis found extremely discordant results and concluded that ΔIVC does not seem to be an accurate method [7]. It was also pointed out that few studies have evaluated this technique in pediatric populations. Due to their reduced anatomic dimensions, accurately measuring IVC diameters can be challenging in children. Furthermore, we demonstrated in our previous study that IVC respiratory displacements can substantially impact measurements performed by M-mode ultrasound [8]. Therefore, the purpose of the present study was to investigate whether ΔIVC obtained by two-dimensional ultrasound (B-mode) can accurately predict fluid responsiveness in children under invasive mechanical ventilation.

Methods

Study Design, Subjects, and Setting

This prospective cohort study was conducted in the PICU of the Clinics Hospital of the State University of Campinas (UNICAMP), Sao Paulo, Brazil, between May and October 2021. The study was approved by the local institutional review board (UNICAMP's Research and Ethics Committee, approval #12894719.8.0000.5404), and written informed consent was obtained from the participants' legal guardians.

Children under invasive mechanical ventilation who required fluid loading at discretion of the attending physician were consecutively assessed for eligibility. The decision to give fluid was taken based on signs of inadequate tissue perfusion such as tachycardia, delayed capillary refilling, hypotension, oliguria, or hemodynamic instability despite vasoactive agents. Patients were included if they met the following criteria: (1) tidal volume of 8–10 ml kg^{-1}; (2) positive end-expiratory pressure of 5–6 cmH_2O; (3) absence of spontaneous breathing; (4) sinus rhythm. Exclusion criteria were as follows: (1) congenital heart diseases; (2) left ventricular systolic dysfunction (ejection fraction < 50%); (3) skin lesions or bandages at the sites of ultrasound or echocardiography exams; (4) poor echocardiographic windows; (5) operator or ultrasound machine unavailability.

Study Protocol

Participants received a fluid infusion of 10 ml $kg^{-1}$of crystalloid solutions (normal saline or Ringer's lactate solution) over 10 min. Data collection was performed before and immediately after fluid loading, and included: (1) heart rate, (2) blood pressure, (3) SV, (4) respiratory variation in aortic blood flow peak velocity (ΔVpeak), and (5) ΔIVC. Infusions of sedatives, analgesics and vasoactive drugs remained unchanged throughout the study period. After fluid infusion, participants were considered fluid responsive when their SV increased by more than 15% from baseline and were classified as "responders", otherwise they were classified as "non-responders". Likewise, increases in mean arterial pressure (MAP) greater than or equal to 10% were considered significant [9, 10].

Echocardiography Measurements

Transthoracic echocardiography (TTE) was performed using an ultrasound machine (Vivid Q; GE Healthcare, Tirat

Carmel, Israel) equipped with a phased array transducer (3.5–8 MHz). Although both exams were performed at the same time, the calculations of ΔIVC, ΔVpeak, and SV were performed later. Thus, the operator was unaware of these variables during the data collection period. All echocardiographic examinations were performed by an experienced pediatric ultrasound instructor from the Brazilian Society of Intensive Care, with 7 years of experience in pediatric point-of-care ultrasound.

IVC diameters were measured in the longitudinal plane using B-mode, 1 cm distal to the hepatic vein-IVC confluence, during both inspiration and expiration. The decision to use B-mode instead of M-mode was made to avoid the influence of IVC craniocaudal respiratory displacements on the measurements [8]. Thus, the IVC diameters were measured at the same point along its length during the respiratory cycle. ΔIVC was calculated as follows: $\Delta IVC = \frac{\frac{(Diameter\ máx - Diameter\ min)}{(Diameter\ máx + Diameter\ min)}}{2} \times 100$.

SV was determined by measuring the aortic diameter (Da) and the aortic velocity–time integral (VTI). The Da was measured at the level of the aortic annulus by the parasternal long-axis view, while the VTI was measured by the apical five-chamber view using pulsed wave Doppler, with the sample volume positioned at the level of the left ventricular outflow tract. The highest and the lowest VTI obtained during a single respiratory cycle were determined through automatically traced envelopes, and the mean value was registered. The VTI was measured in triplicate and the mean value was considered for SV calculation. Both diameter and VTI were determined in centimeters. Finally, the SV was calculated using the following standard formula: $SV(ml) = \left(\pi \times Da^2/4\right) \times VTI$.

Maximal and minimal values of aortic blood flow peak velocities were determined beat-to-beat over a single respiratory cycle. ΔVpeak was calculated as follows: $\Delta Vpeak = \frac{\frac{(Vpeak\ máx - Vpeak\ min)}{(Vpeak\ máx + Vpeak\ min)}}{2} \times 100$. The average of three ΔVpeak measurements was considered for analysis purposes.

Statistical Analysis

The normality of the data distribution was assessed using the Kolmogorov–Smirnov and Shapiro–Wilk tests. Continuous variables were expressed as medians and interquartile ranges (IQR), while categorical variables were expressed as absolute numbers (%). After fluid loading, participants were divided into two groups: responders and non-responders. The groups were compared using the Mann–Whitney test (continuous variables) and the chi-square or Fisher's exact test (categorical variables). Variables collected before and after fluid loading were compared using Wilcoxon rank-sum test. Receiver Operating Characteristic (ROC) curves were constructed to assess the accuracy of predictors and their areas under the curve were compared using the non-parametric technique proposed by DeLong et al. [11]. The optimal cut-off points were defined according to the optimal Youden's J statistic using univariable analysis. Spearman's correlation coefficient (ρ) was used to estimate and test the relationship between continuous variables. p-values < 0.05 were considered significant.

Assuming a fluid responsiveness rate of 33%, we determined that 30 measurements would be needed to detect a difference of 0.30 between the area under the ROC curve (AUROC) of ΔVpeak and the null hypothesis (AUROC = 0.5; i.e., no discriminating power), with an 80% power and type I error of 5%. Statistical analysis and sample size calculation were performed using MedCalc Statistical Software version 19.8 (MedCalc Software bvba, Ostend, Belgium).

Results

Thirty-two patients were enrolled and included in the final analysis. Median age and weight were 17 months (5.5–61) and 10 kg (5.4–19), respectively. Demographic characteristics of participants are presented in Table 1.

Thirteen subjects (41%) were responders to fluid loading. At baseline, responders and non-responders were similar in age, blood pressure, heart rate, vasoactive drugs use, and mechanical ventilation settings. However, the "responder" group had higher weight (13.5 kg vs 9.0 kg, $p = 0.048$), higher ΔVpeak (22.2% vs 7.3%, $p = 0.001$), higher ΔIVC (9.0% vs 2.4%, $p = 0.046$), lower Ci (2.27 L min^{-1} m^{-2} vs 3.47 L min^{-1} m^{-2}, $p = 0.009$), and lower SVi (15.82 mL m^{-2} vs 22.87 mL m^{-2}, $p = 0.003$).

The variables ΔIVC, ΔVpeak, SVi and Ci were predictors of fluid responsiveness (See Fig. 1). However, ΔVpeak was found to be more accurate than ΔIVC in the ROC curve analysis (AUROC of 0.935 vs 0.709, respectively, $p = 0.012$). No other significant differences were observed in the pairwise analysis of the AUROC of the predictors. The best cut-off value of ΔIVC found was 7.7%, which had a sensitivity of 69.2%, specificity of 78.9%, positive predictive value (PPV) of 69.2% and negative predictive value (NPV) of 78.9%. A ΔVpeak of > 18.2% was able to predict fluid responsiveness with a sensitivity of 84.6%, specificity of 89.5%, PPV of 84.6%, and NPV of 89.5%. The complete analysis of the ROC curves is presented in Table 2.

Fluid loading significantly changed heart rate and MAP in both groups. However, diastolic blood pressure, SVi, Ci, ΔVpeak were changed only in the "responder" group, while systolic blood pressure changed only in the "non-responder" group. ΔIVC did not change significantly in either group. A 10% increase in MAP was observed in

Table 1 Baseline clinical and demographic characteristics of participants at inclusion according to the response to fluid loading

Variable	All patients ($n=32$)	Responders ($n=13$)	Non-responders ($n=19$)	p-value
Age (months)	17 (5.5–61)	25 (8.5–121)	13 (4–35)	0.198
Weight (kg)	10 (5.4–19)	13.5 (9.2–40)	9 (4.6–12.7)	**0.048**
Male sex, *n* (%)	13 (41%)	7 (54%)	6 (32%)	0.215
Primary diagnosis group at PICU admission, *n* (%)				
Clinical	17 (53%)	6 (46%)	11 (58%)	0.520
Sepsis	9 (28%)	3 (23%)	6 (32%)	0.605
Respiratory failure	7 (22%)	3 (23%)	4 (21%)	0.893
Surgical	15 (47%)	7 (54%)	8 (42%)	0.520
Heart rate (bpm)	153 (130–170.5)	156 (137.5–179)	150 (122.5–166.5)	0.454
SVi (ml m^{-2})	20.61 (15.92–25.75)	15.82 (11.39–20.20)	22.87 (19.81–28.95)	**0.003**
Ci (L min^{-1} m^{-2})	3.11 (2.37–3.80)	2.27 (1.85–3.06)	3.47 (2.99–4.09)	**0.009**
Systolic blood pressure (mmHg)	91.5 (78–101)	94 (80.25–104.25)	88 (78–97)	0.337
Diastolic blood pressure (mmHg)	53 (40–58)	53 (43.5–58)	53 (40–58.25)	0.863
Mean blood pressure (mmHg)	64 (52–74)	69 (55.5–75.75)	64 (51.5–70.5)	0.357
ΔVpeak (%)	12.7 (5.1–21.6)	22.2 (19.8–26.0)	7.3 (3.2–11.7)	**< 0.001**
ΔIVC (%)	5.4 (0–11.4)	9.0 (5.8–16.0)	2.4 (0.0–7.5)	**0.046**
Vasoactive drugs use, *n* (%)	11 (34%)	7 (54%)	4 (21%)	0.059
Peak inspiratory pressure (cmH_2O)	20 (18–23.5)	18 (16.7–27.2)	20 (18.5–22)	0.522
Mean airway pressure (cmH_2O)	10 (9.75–10.5)	10 (9.6–10)	10 (9.9–11.25)	0.623
PEEP level (cmH_2O)	5 (5–5)	5 (5–5)	5 (5–5)	0.688
Tidal volume (ml kg^{-1} PBW)	8 (7–8)	8 (7–8)	7.5 (7–8)	0.602

Values are expressed as median (25th–75th percentiles) or number of subjects (%)

SVi stroke volume index; *Ci* cardiac index; *PEEP* positive end-expiratory pressure; *PBW* predicted body weight; Δ*IVC* respiratory variation in inferior vena cava diameter; Δ*Vpeak* respiratory peak velocity variation of aortic blood flow

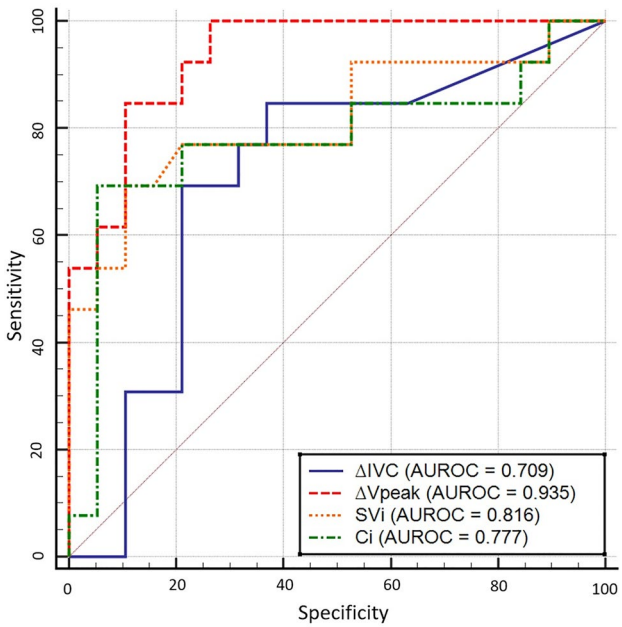

Fig. 1 Areas under the ROC curves of the evaluated predictors of fluid responsiveness. ΔVpeak was found to be more accurate than ΔIVC in the ROC curve analysis (AUROC of 0.935 vs 0.709, respectively, $p=0.012$). No other significant differences were observed in the pairwise analysis of the AUROC of the predictors

61% (8/13) of participants in the "responder" group as well as in 37% (7/19) of participants in the "non-responder" group (See Fig. 2). There was no association between the proportion of participants who had an increase in MAP (61%) and those who were fluid responders (41%) ($p=0.233$), and no significant correlation was found between changes in MAP and SVi ($\rho=0.191$, $p=0.294$). Hemodynamic variables before and after fluid loading in both groups are shown in Table 3.

SVi changes were positively correlated with ΔIVC ($\rho=0.566$, $p<0.001$) and ΔVpeak ($\rho=0.603$, $p<0.001$). A significant positive linear correlation was also found between changes in MAP and ΔVpeak ($\rho=0.382$; $p=0.031$), but the same was not observed with ΔIVC ($\rho=0.011$; $p=0.951$).

Discussion

In the present study, the ΔIVC presented an acceptable accuracy for predicting fluid responsiveness in mechanically ventilated children. However, its accuracy was significantly lower than ΔVpeak. Also, Ci and SVi had higher AUROC values than ΔIVC, but no statistical difference was observed. The best ΔIVC cutoff value found was

7.7%, which has a sensitivity of 69.2% and a specificity of 78.9%. This threshold is much lower than previously found in similar studies. Some methodological particularities may be responsible for the discrepancies between the results herein reported and those previously published [12–21].

The main differentiating feature of our study is the method used to determine the IVC diameters. While the other pediatric studies used M-mode, we determined the IVC diameters using two-dimensional ultrasound. This approach was chosen to mitigate the impact of IVC respiratory displacements on diameter measurements. Only two-dimensional ultrasonography allows the maximum and minimum IVC diameters to be measured at the same distance from the hepatic vein. When using M-mode, the IVC diameters are registered at different locations along its length, since the ultrasound beam is static and the vessel has a significant respiratory displacement in the craniocaudal direction [8]. Thus, the M-mode can register structural changes in IVC diameters rather than only the respiratory variability [22]. As a matter of fact, there is an interesting debate about which is the best method and location for measuring IVC diameter in adults, and this discussion also needs to take place in pediatrics [23–25].

In our previous study, we observed a large discrepancy between the ΔIVC values obtained by M-mode and two-dimensional ultrasound when evaluating the same respiratory cycle [8]. The median values of the ΔIVC obtained by two-dimensional ultrasound were about half of those obtained by M-mode (11.45% vs 21.82%, $p < 0.001$) [8]. Therefore, it is not surprising that we found a cut-off point much lower than those previously published. While the best IVC cutoff value in our study was only 7.7%, other authors reported values ranging from 12.3% to 28.5% [12–21]. The IVC respiratory displacements may be a major contributor to the disagreement between both methods for ΔIVC

Table 2 Areas under the ROC curve for assessed predictors of fluid responsiveness

Performance	ΔIVC	ΔVpeak	SVi	Ci
AUROC	0.709 (0.522–0.855)	0.935 (0.789–0.992)	0.816 (0.63–0.93)	0.777 (0.596–0.905)
Optimal cut-off	7.7%	18.2%	18.2 mL m^{-2}	2.5 L min^{-1} m^{-2}
Sensitivity (%)	69.2%	84.6%	69.2%	69.23%
Specificity (%)	78.9%	89.5%	89.5%	94.74%
PPV	69.2%	84.6%	81.8%	90.0%
NPV	78.9%	89.5%	81.0%	81.8%
PLR	3.29	8.04	6.58	13.15
NLR	0.39	0.17	0.34	0.32

Cut point calculated using Youden's index. Values are expressed as median (25th–75th percentiles)

Δ*IVC* respiratory variation in inferior vena cava diameter; Δ*Vpeak* respiratory peak velocity variation of aortic blood flow; *SVi* stroke volume index; *Ci* cardiac index; *AUROC* area under the receiver operating characteristic curve; *PPV* positive predictive value; *NPV* negative predictive value; *PLR* positive likelihood ratio; *NLR* negative likelihood ratio

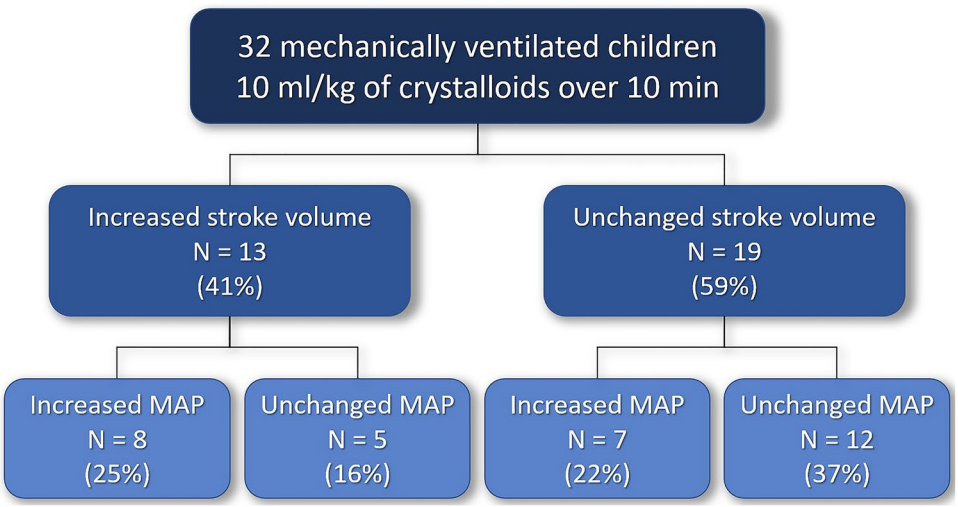

Fig. 2 Distribution of fluid bolus response according to the CI and MAP response. There was no association between the proportion of participants who had an increase in MAP (61%) and those who were fluid responders (41%) ($p = 0.233$)

Table 3 Hemodynamic variables before and after fluid loading

Variables	Responders ($n=13$)			Non-responders ($n=19$)		
	Baseline	Fluid loading	*p*-value	Baseline	Fluid loading	*p*-value
HR (bpm)	156 (137.5–179)	137 (121.5–177.0)	**0.010**	150 (122.5–166.5)	137 (120.75–162.25)	**0.001**
SBP (mmHg)	94 (80.25–104.25)	101.0 (84.0–126.5)	0.069	88 (78–97)	100 (84.25–105.50)	**0.009**
DBP (mmHg)	53 (43.5–58)	63 (52.5–68.0)	**0.006**	53 (40–58.25)	52 (43.5–61.75)	0.050
MAP (mmHg)	69 (55.5–75.75)	80.0 (63.0–83.5)	**0.009**	64 (51.5–70.5)	68 (55.25–74.25)	**0.006**
SVi (mL m^{-2})	15.82 (11.39–20.20)	22.47 (14.59–29.19)	**0.001**	22.87 (19.80–28.95)	25.22 (19.68–29.69)	0.126
Ci (L min^{-1} m^{-2})	2.27 (1.85–3.06)	3.15 (2.08–4.30)	**0.006**	3.47 (2.99–4.09)	3.45 (2.65–3.96)	0.076
ΔVpeak (%)	22.2 (19.8–26.0)	10.17 (5.93–15.33)	**0.002**	7.3 (3.2–11.7)	4.4 (2.2–7.9)	0.327
ΔIVC (%)	9.0 (5.8–16.0)	4.6 (0.0–10.1)	0.136	2.4 (0–7.5)	3.6 (1.8–5.3)	0.955

Values are expressed as medians and interquartile ranges (25th–75th percentiles)

HR heart rate; *SBP* systolic blood pressure; *DBP* diastolic blood pressure; *MAP* mean arterial pressure; *Svi* stroke volume index; *Ci* cardiac index; Δ*Vpeak* respiratory peak velocity variation of aortic blood flow; Δ*IVC* respiratory variation in inferior vena cava diameter

measurements. Accurately measuring IVC diameters can be challenging in children, especially younger ones, so future studies should perform measurements using two-dimensional ultrasonography to reduce inaccuracies.

The use of ΔIVC as a predictor of fluid responsiveness gained popularity in critical care medicine after early studies demonstrated its high accuracy in adults [5, 6]. However, the same exciting results were not found in similar later studies. A meta-analysis including adults and children found a pooled sensitivity and specificity of 63% and 73%, respectively (pooled AUROC of 0.79) [26]. In addition, the test performance appears to be better when patients are ventilated with a tidal volume ≥ 8 ml kg^{-1} and positive end-expiratory pressure ≤ 5 cmH2O. This aspect is especially important for mechanically ventilated patients in whom lung protective strategies are applied. Thus, a transient increase in tidal volume when performing the test may increase its accuracy, which has been called "tidal volume challenge". In children the accuracy of ΔIVC varies widely across studies, with sensitivity ranging from 47 to 100% and specificity ranging from 33 to 100% [12–21, 27].

In our study, ΔVpeak was shown to be a more reliable predictor of fluid responsiveness when compared to ΔIVC, with sensitivity and specificity of 84.6% and 89.5%, respectively. Similar results were found by other authors [12, 13, 27]. A recent meta-analysis of pediatric studies evaluating ΔVpeak found a pooled sensitivity of 84% and pooled specificity of 82% [27]. However, despite its good accuracy, the use of ΔVpeak is often limited in intensive care patients due to suboptimal acoustic windows caused by mechanical ventilation, dressings, interfering incisions, and positioning difficulties [28]. In addition, obtaining ΔVpeak can be challenging for inexperienced operators as it requires specific skills and training. Even so, its use should be encouraged due to the good results presented in several studies.

Blood pressure is perhaps the most determining hemodynamic variable in making decisions about fluid infusions, as well as in assessing the response to such an intervention. In clinical practice, patients are often considered fluid responders when they experience increased blood pressure after fluid loading [29]. However, in our study the percentage of patients who increased their MAP after volume expansion was similar between fluid responders and non-responders. The absence of an association between fluid bolus-induced changes in MAP and Ci (or SV) has also been reported in other studies involving children [9, 10, 30]. Furthermore, the adverse effects of volume expansion are not limited to fluid overload. Ranjit et al. found that some children experienced hemodynamic deterioration soon after the end of fluid infusion, with reduced MAP, reduced pulse pressure, and increased use of vasoactive drugs [9, 10]. Although blood pressure is an important hemodynamic variable for organ perfusion, it should not be used as a surrogate of SV when assessing response to fluid loading.

The present study has some limitations that need to be pointed out. First, participants were not receiving neuromuscular blocking agents. However, they were under sedation and the absence of spontaneous breathing was verified clinically and on ventilator monitoring. Second, fluid responsiveness was not assessed using gold standard techniques such as thermodilution or the direct Fick method. Nevertheless, the use of TTE for Ci measurements has been widely studied in children, proving to be accurate, precise and reproducible [31]. Third, both fluid responsiveness and ΔVpeak were determined using the same echocardiographic method (TTE). With this, the index test and the standard reference criteria were not independent, which may have led to an overestimation of diagnostic accuracy. Fourth, obtaining ΔVpeak by TTE is subject to technical artifacts caused by chest wall movements during breathing. This limitation can

be overcome by transesophageal echocardiography; however, this method is not available in most PICUs. Fifth, our study included a specific population of children under invasive mechanical ventilation. Therefore, the results reported here should not be extrapolated to other populations, such as spontaneously breathing children.

Conclusion

The ΔIVC has limited accuracy to identify children who will increase their SV after fluid loading. The use of ΔVpeak should be preferred over ΔIVC whenever possible, as it seems to be a better predictor of fluid responsiveness in mechanically ventilated children.

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Data Availability The data that support the findings of this study are available from the corresponding author, upon reasonable request.

Code Availability Not applicable.

Declarations

Conflict of interest The authors have no conflicts of interest relevant to this study to disclose.

Ethics Approval The study was approved by the local institutional review board (UNICAMP's Research and Ethics Committee, approval number 12894719.8.0000.5404).

Informed Consent Written informed consent was obtained from the participants' legal guardian.

Consent for Publication Not applicable.

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