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Clinical value of calibrated abdominal compression plus transthoracic echocardiography to predict fluid responsiveness in critically ill infants: a diagnostic accuracy study

Julien Gotchac[1,2*], Anouk Navion[3], Yaniss Belaroussi[4], Roman Klifa[3], Pascal Amedro[1,2], Julie Guichoux[3] and Olivier Brissaud[3]

Abstract

Background Predicting fluid responsiveness is challenging in infants. It is however crucial to avoid unnecessary volume expansion, which can lead to fluid overload. We tested the hypothesis that the stroke volume changes induced by a calibrated abdominal compression (ΔSV-AC) could predict fluid responsiveness in infants without cardiac disease.

Methods This prospective single center study of diagnostic test accuracy was conducted in a general pediatric intensive care unit (PICU). Children under the age of two with acute circulatory failure and requiring a 10 $mL.kg^{-1}$ crystalloid volume expansion over 20 min, ventilated or not ventilated, were eligible. Stroke volume was measured by transthoracic echocardiography at baseline, during a gentle calibrated abdominal compression (22 mmHg for 30 s), and after volume expansion. The area under the receiver operating characteristic curve (AUROC) of ΔSV-AC was measured to predict fluid responsiveness, defined as a 15% stroke volume increase after volume expansion.

Results Twenty-seven cases of volume expansion were analyzed, in 21 patients. Seventeen VE cases were administrated to spontaneously breathing children. Fluid responsiveness was observed in 12 cases. The AUROC of ΔSV-AC was 0.93 (95% confidence interval (95%CI) 0.82–1). The best threshold value for ΔSV-AC was 9.5%. At this threshold value, sensitivity was 92% (95%CI 62–100), specificity was 87% (95%CI 60–98), positive and negative predictive values were 85% (95%CI 60–95) and 93% (95%CI 66–99) respectively.

Conclusions Echocardiographic assessment of stroke volume changes induced by a calibrated abdominal compression is a promising method to predict fluid responsiveness in infants without cardiac disease hospitalized in PICU.

Trial registration ClinicalTrials.gov registration number NCT05919719, June 22, 2023, retrospectively registered, https://clinicaltrials.gov/study/NCT05919719.

Keywords Circulatory failure, Echocardiography, Fluid responsiveness, Goal-directed fluid management, Volume Expansion

*Correspondence:
Julien Gotchac
julien.gotchac@chu-bordeaux.fr
Full list of author information is available at the end of the article

Background

Although volume expansion (VE) remains the cornerstone of acute circulatory failure treatment [1–4], a significant increase in stroke volume (SV) only occurs in approximately 40% to 60% of VE [5–8]. Yet, the harmful impact of fluid overload is well documented in children [9, 10]. Therefore, identifying fluid responders is of paramount importance in pediatric intensive care units (PICUs) [6].

In children and neonates, the respiratory variability of the peak aortic velocity (ΔPeak) is considered the best-validated fluid responsiveness test [6, 7, 11], but requires specific clinical conditions that are not always met in critically ill children [6]. Notably, ΔPeak is only reliable in intubated children and in the absence of spontaneous breathing [5, 6, 8], as this test in based on cardiopulmonary interactions. Only dynamic tests based on another source of preload variation are reliable in spontaneously breathing patients. In adults, this applies to the classic passive leg-raising test, which relies on an endogenous, reversible, and ventilation-independent increase in preload through the mobilization of the venous reservoir of the lower limbs [12]. Passive leg-raising test is also applicable to children, although its diagnostic accuracy appears to be poorer than in adults [13–15]. This might be due to a lower blood volume in the lower limbs, as leg length is proportionately smaller in infants [16]. Conversely, the hepatosplanchnic venous reservoir is easily accessible in children. A gentle abdominal compression can rapidly mobilize the unstressed venous blood volume from the abdominal organs, transiently increasing venous return and cardiac preload by the same mechanism as passive leg-raising [17].

Yet, the calibrated abdominal compression maneuver has been scarcely evaluated to predict fluid responsiveness in children, whereas this technique could be very useful for spontaneously breathing children requiring VE. Previous reports have used this technique in postoperative pediatric cardiac surgery [17–19], however fluid responsiveness using calibrated abdominal compression has not been validated in general PICUs. We hypothesized that the SV changes induced by the calibrated abdominal compression maneuver (ΔSV-AC) could predict fluid responsiveness in a mixed population of ventilated and non-ventilated infants without cardiac disease.

Methods

The study was carried out in accordance with the Good Clinical Practices protocol and Declaration of Helsinki principles. It was approved by our Institutional Review Board (Comité de Protection des Personnes Ouest IV, number 2021-A02876-35, approval date January 11 th 2022) and retrospectively registered on Clinicaltrials.gov (NCT05919719, June 22, 2023). Informed consent was obtained from all parents or legal guardians for minors, before or within 24 h after study procedures.

This study of diagnostic test accuracy was prospectively conducted in a single general PICU of a tertiary care center (Bordeaux University Hospital, France), from February 2022 to January 2023. Infants aged < 2 years with acute circulatory failure and requiring VE (crystalloid, 10 $mL.kg^{-1}$ I.V., over 20 min maximum) were consecutively screened. Acute circulatory failure was defined by tachycardia (heart rate (HR) > 2 standard deviations (SD) for age) or arterial hypotension (systolic or mean arterial pressure (MAP) < 2 SD for age), associated with at least one of the following criteria: oliguria (diuresis < 1 $mL.kg^{-1}.h^{-1}$), blood lactate > 2 $mmol.L^{-1}$, capillary refill time (CRT) > 3 s, or mottling. Exclusion criteria were preterm newborn under 37 weeks of corrected gestational age, uncorrected or early postoperative congenital heart disease, cardiogenic pulmonary edema, abdominal pain (as subjectively perceived by the intensivist in charge of the patient during the routine clinical abdominal palpation), postoperative period of abdominal surgery, prone position, severe hemodynamic instability prohibiting any test, patient restlessness, and poor ultrasound window.

In this diagnostic accuracy study, fluid responsiveness was defined by an increase in echocardiography-estimated SV > 15% after VE, which is the most commonly reported gold-standard test in pediatrics [5, 8, 20, 21]. To measure the index test, e.g. ΔSV-AC, a calibrated abdominal compression maneuver was performed following a previously described standardized protocol [17]. A closed sphygmomanometer inflated with 50 mL of air was connected to a pressure-measuring device and interposed between the operator's hand and the patient's abdomen. The center of the sphygmomanometer was placed at the center of the patient's abdomen and covered a third of the patient's abdomen. The operator then performed a gentle manual compression in an anteroposterior direction, gradually reaching a pressure of 22 mmHg for 30 s, while verifying tolerance (eFigure 1, Additional File). Three echocardiographic measures were performed, at baseline (T0), during the calibrated abdominal compression maneuver, after 30 s of abdominal compression (T1), and 30 min after VE (T2). Transthoracic echocardiograms were performed by the intensivist in charge of the patient using the Vivid S60 ultrasound system and the 6S probe (GE Healthcare, Little Chalfont, United Kingdom). At each time point, 6 consecutive aortic velocity–time integrals (VTI) were acquired and averaged from an apical five-chambers view. Consecutive VTIs were selected irrespective of the respiratory cycle, but infants' high respiratory rate ensured that both expiratory and inspiratory phases were obtained. Measures were performed

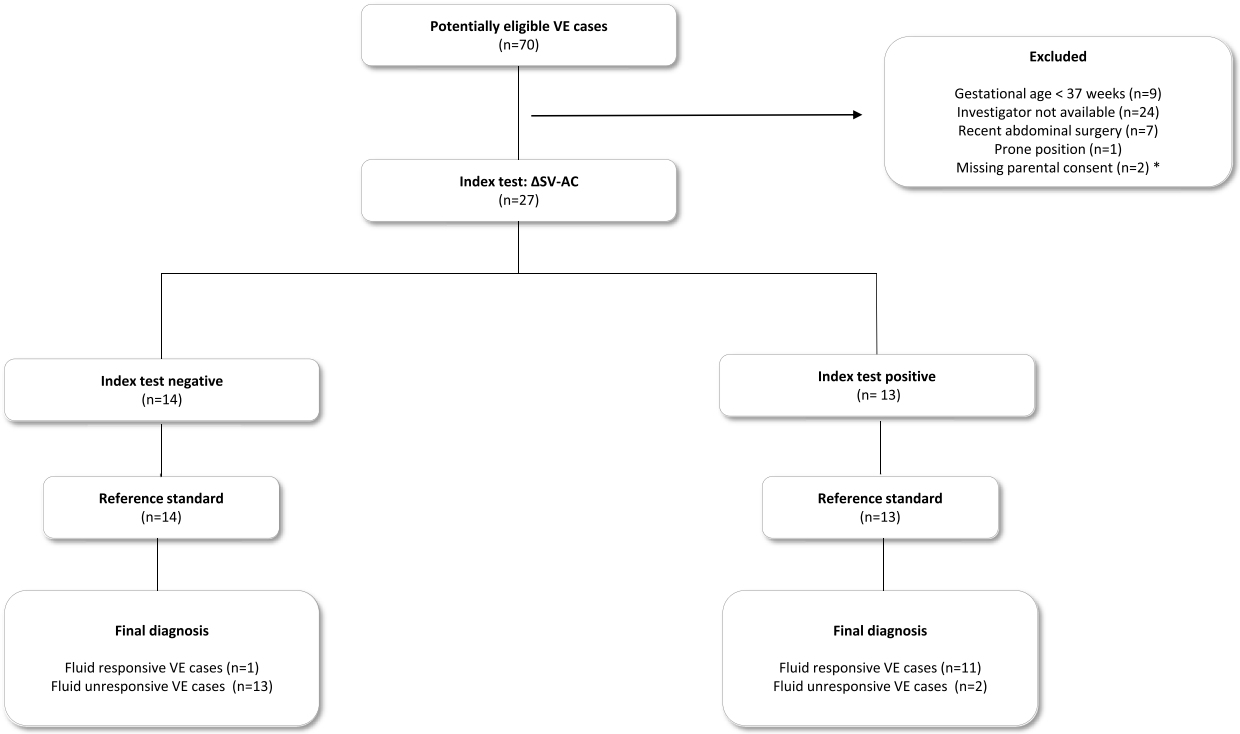

Fig. 1 Study flow chart. Legend: ΔSV-AC, percentage of stroke volume variation between baseline and during a calibrated abdominal compression, VE volume expansion.* For these two subjects, the study procedures were performed but we could not obtain parental informed consent to having their child's data retained and analysed because of language barrier

offline so that the results would not affect patients'care. For all VTI measures recorded, a second offline analysis was performed by the same investigator to assess intraobserver reproducibility and by a second investigator to assess interobserver reproducibility, with no access to the results of the first analysis. All offline measurements were performed blind to patient data and outcome of the fluid challenge. Left ventricular ejection fraction (LVEF) using the Teicholz method and left ventricular outflow tract diameter (LVOTd) were measured at baseline. SV was defined as $VTI \times \Pi \times \frac{LVOTd^2}{4}$. LVOT diameter was only measured at baseline, as it was considered stable over the study period. Cardiac output was the product of SV and HR. Cardiac output was indexed by body surface area to obtain cardiac index (CI). The change in SV during the calibrated abdominal compression, *e.g.*, ΔSV-AC (%), was measured by the difference between SV at T1 and SV at T0, divided by SV at T0 (index test). The change in SV after VE, *e.g.*, ΔSV-VE (%) was measured by the difference between SV at T2 and SV at T0, divided by SV at T0, a ΔSV-VE >15% defining fluid responsiveness. In addition, clinical and hemodynamic baseline parameters were collected, including patient characteristics, diagnosis at admission, ventilation mode, presence of spontaneous breathing, positive end-expiratory pressure, presence of a vasoactive or inotropic hemodynamic support, vasoactive-inotropic score, and previous VE for the current episode of circulatory failure. In addition, the intensivist in charge of the patient reported the presumed cause of hemodynamic failure using four categories: hypovolemia, vasoplegia, cardiac dysfunction, and mixed or unclassifiable. The following variables were also collected before and after VE: HR, MAP, mottling, CRT, urine output, peripheral oxygen saturation, fraction of inspired oxygen, and blood lactate if available. Due to the non-interventional design of this study, central venous pressure was not collected, as its measure was not a standard practice in our center. Exact duration of mechanical ventilation, PICU length of stay (discharge date minus admission date), and mortality were collected at discharge. Finally, the clinician's global perception regarding fluid responsiveness after VE was collected immediately before the post-VE echocardiographic assessment at T2.

The sample size was calculated with the Obuchowski method [22], prior to data collection. A total of 24 cases of VE were needed to detect an area under the receiver-operating characteristic (ROC) curve (AUROC) of 0.78 [18], with an alpha risk of 0.05, a statistical power of 80%, and a 1:1 ratio of fluid responsive to unresponsive cases.

Patient characteristics were presented in median form (first quartile, third quartile), or as frequencies and proportions for qualitative variables. ROC curves were drawn to determine the ability of ΔSV-AC to predict fluid responsiveness (primary objective). The AUROC, with its 95% confidence interval (95%CI) represented the overall diagnostic accuracy of ΔSV-AC. The best ΔSV-AC threshold value was determined using diagnostic accuracy. Sensitivity, specificity, positive and negative predictive values, and positive and negative likelihood ratios were identified at this threshold value, with their respective 95%CI. For categorical variables, group comparisons were performed with the $\chi 2$ test with correction for continuity or with an exact Fisher's test, as appropriate. Quantitative variables were compared using the Student t-test when their distribution followed a normal distribution, or the Mann–Whitney test otherwise. A Shapiro–Wilk test was used to test the normal distribution. The Wilcoxon test was used to compare hemodynamic parameters between before and after VE. To test linear correlations, Spearman's correlation coefficient was used. We evaluated the reproducibility of VTI measurements by calculating intraclass correlation coefficients to assess interobserver and intraobserver reliability. To explore the evolution of hemodynamic parameters after VE and to compare the effect of time and group, we used a repeated measure analysis of variance. To investigate variables independently associated with fluid responsiveness (secondary objective), we first used a univariate binomial logistic regression. Variables significantly associated with fluid responsiveness ($p < 0.05$) were then integrated into a multivariable binomial logistic regression model. The two groups, *e.g.*, fluid responsive *vs.* unresponsive cases, were analyzed according to VE cases and not to patients, as some patients underwent two VE. We performed a sensitivity analysis using only the first VE for each patient since repeated measurements obtained from the same patients might be correlated. Analysis were performed with RStudio version 4.0.3 (RStudio, PBC, Boston, MA, USA) and the Jamovi 1.2 graphical interface (The Jamovi project, Sydney, Australia). Differences with a *P-value* of less than 0.05 were considered statistically significant.

Results

From February 2022 to January 2023, 27 consecutive cases of VE were prospectively collected from a cohort of 21 children (6 children underwent two VE). Two VE were infused at a dosage of 20 instead of 10 $mL.kg^{-1}$. The study flow chart is reported in Fig. 1. Patient ages ranged from 0 to 13 months, and 14 were neonates. Only one patient had an invasive arterial blood pressure monitoring. Demographic and clinical data are reported in Table 1. Twenty-two VE cases (81%) were administrated to intubated patients. They were ventilated in a pressure-control mode in 20 VE cases with a median Mean Airway Pressure of 10 (9,12) cmH2O. As per local guidelines, all intubated patients were sedated with a combination of sufentanyl (0.1–0.9 $\mu g.kg^{-1}.h^{-1}$) and midazolam (15–240 $\mu g.kg^{-1}.h^{-1}$) or dexmedetomidine (0.35–1.5 $\mu g.kg^{-1}.h^{-1}$), and two of them were curarized. Among the 5 VE cases administrated to non-intubated patients, 2 infants received a 20 $\mu g.kg^{-1}.h^{-1}$ continuous morphine infusion, and 3 received no sedation.

Table 1 Population demographic and clinical characteristics

Variable	Patients ($N = 21$)
Age (days)	8 (1,35)
Weight (kg)	3.80 (3.4,4.5)
Male (N)	11
PELODS II	6.76 ± 4.35
Diagnosis at admission (N)	
Hypoxic-ischemic encephalopathy	8
Respiratory failure	6
Sepsis	2
Post-operative of neurosurgery	2
Other	2
Cardiac failure	1
PICU length of stay (days)	10.0 (6,14)
PICU mortality (N)	1
Duration of MV (days)	6.0 (3,9)

MV mechanical ventilation, *PELODS II* Pediatric Logistic Organ Dysfunction Score II, *PICU* Pediatric Intensive Care Unit. Data are reported as No., means ± standard deviation, or medians (first quartile, third quartile)

Twelve cases resulted in a SV increase of more than 15%, defining fluid responsiveness. Clinicians assumed fluid responsiveness in 20 cases. The clinical and echocardiographic data at baseline of both fluid-responsive and fluid-unresponsive cases are reported in Table 2. SV increase was higher in the fluid-responsive group than in the fluid-unresponsive group [31% (21%–38%) vs. 5% (0%–9%)]. After VE, the following clinically significant changes were observed: decrease in HR (140 [111–153] vs. 148 [122–167] bpm, $p = 0.015$), increase in MAP (50 [45–63] vs. 41 [38–57] mmHg, $p = 0.001$), resolution of mottling (11% vs. 26%, $p < 0.001$), decrease in CRT (2 [2, 3] vs. 3 [3, 4] sec, $p < 0.001$), and increase in urine output (3.5 [2.5–5.4] vs. 1.6 [0.8–2.5] $mL.kg^{-1}.h^{-1}$, $p = 0.005$). However, these changes were not significantly different between fluid responsive and unresponsive cases. Hemodynamic changes after VE, in both fluid-responsive and fluid-unresponsive groups, are reported in Table 3.

ΔSV-AC was able to assess fluid responsiveness with an AUROC of 0.93 (95%CI 0.82–1). The best threshold for ΔSV-AC was 9.5%. At this threshold value, Youden index

Table 2 Baseline group characteristics

Variable	All VE cases (*N* = 27)	Fluid-unresponsive cases (*N* = 15)	Fluid-responsive cases (*N* = 12)	*p* value
Intubated (N)	22	12	10	1
Spontaneous breathing (N)	17	12	5	0.057
PEEP (cmH2O)	6 (5,7)	5 (5,7)	6 (6,7)	0.332
FiO_2 (%)	30 (23,43)	35 (28,50)	25 (21,30)	0.038
SpO_2/FiO_2 ratio (%)	330 (215,427)	271 (197,347)	382 (322,462)	0.044
Previous VE (N)	10	3	7	0.057
Hemodynamic support (N)	10	5	5	0.706
VIS ($\mu g.kg^{-1}.min^{-1}$)	0.0 (0.0,5.2)	0. 0 (0.0,5.1)	0.0 (0.0,16.4)	0.727
Heart rate ($beats.min^{-1}$)	148 (122,167)	150 (122,170)	145 (122,160)	0.722
Mean arterial pressure (mmHg)	41 (38,57)	41 (39,66)	41 (37,47)	0.607
Mottling (N)	7	3	4	0.662
Capillary refill time (s)	3 (3,4)	4 (3,4)	3 (3,4)	0.602
Urine output ($mL.kg^{-1}.h^{-1}$)	1.6 (0.8,2.5)	1.4 (0.8,2.4)	1.6 (0.8,2.7)	1
Lactate ($mmol.L^{-1}$)	2.7 (1.5–4.3)	2.5 (1.3–4.6)	2.7 (1.7–3.1)	0.970
LVEF (%)	70 (65–75)	67 (65–74)	73 (70,77)	0.049
VTI (cm)	9.3 (7.8,11.5)	9.6 (8.8,11.6)	7.9 (7.2,10.6)	0.092
SVi ($mL.m^{-2}$)	14.5 (12.3,18.7)	14.5 (12.5,18.7)	14.6 (12.2,17.8)	0.943
Cardiac index ($L.min^{-1}.m^{-2}$)	2.0 (1.4,3.2)	2.0 (1.6,3.3)	2.0 (1.5,2.7)	0.719
Presumed cause of hemodynamic failure				0.826
Hypovolemia (N)	18	9	9	
Vasoplegia (N)	2	1	1	
Cardiac dysfunction (N)	2	2	0	
Mixed or unclassifiable (N)	5	3	2	

FiO_2, fraction of inspired oxygen, *LVEF* left ventricular ejection fraction, *PEEP* positive end-expiratory pressure, *SpO_2/FiO_2* ratio of peripheral oxygen saturation to fraction of inspired oxygen, *SVi* indexed stroke volume, *VE* volume expansion, *VIS* vasoactive inotropic score, *VTI* aortic velocity–time integral. Data are reported as No. or medians (first quartile, third quartile)

was 0.78 (95%CI 0.21–0.98), sensitivity was 92% (95%CI 62–100), specificity was 87% (95%CI 60–98), positive predictive value was 85% (95%CI 60–95), negative predictive value was 93% (95%CI 66–99), positive and negative likelihood ratio were 4.89 (95%CI 1.74–13.75) and 0.10 (0.02–0.68) respectively. ROC curve is shown in Fig. 2. Median ΔSV-AC was higher in fluid responders than in fluid non-responders [14% (12%–16%) vs. 0% (−10%–3%), $p < 0.001$]. ΔSV-AC was significantly associated with ΔSV-VE (Rho = 0.79, $p < 0.001$). After adjustment for ventilation status, ΔSV-AC remained significantly associated with fluid responsiveness (OR = 1.391 (95%CI 1.037–1.865), $p = 0.028$). Pre-planned sensitivity analysis including only the first VE for each patient; and post-hoc sensitivity analysis conducted exclusively with per-protocol 10 $mL.kg^{-1}$ VE both found similar results. Similar results were also observed in a post-hoc sensitivity analysis using other definitions of fluid responsiveness (eTables 1–2, Additional File).

The reproducibility of the VTI measurement was excellent: the intraclass correlation coefficients were 0.96 and 0.98 for intraobserver and interobserver reliability respectively. No indeterminate index test or reference standard were reported. No missing data were reported. The calibrated abdominal compression maneuver was well tolerated, even in non-sedated patients with a normal neurological status. No adverse events from performing the index test or the reference standard was reported, neither any discomfort nor pain, which is supported by the absence of HR variation during the maneuver (Table 3).

Clinician global perception was not associated with fluid responsiveness: clinicians assumed fluid responsiveness in 9 out of 12 fluid-responsive cases and in 11 out of 15 fluid-unresponsive cases. In univariate analysis, two baseline parameters were significantly associated with fluid responsiveness: previous VE for the current episode of acute circulatory failure (OR = 5.60 (95%CI 1.02–30.90), $p = 0.048$), and presence of spontaneous breathing (OR = 0.18 (95%CI 0.03–0.99), $p = 0.048$). However, in multivariate analysis, none of these parameters was significantly associated with fluid responsiveness. Neither clinical global perception nor any post-VE variation in clinical hemodynamic parameters (HR, MAP, urine

Table 3 Hemodynamic changes after volume expansion in both groups

Variable	Baseline (T0)	Calibrated abdominal compression (T1)	After VE (T2)	*p* value		
				Group effect	Time effect	Interaction
Heart rate (beats.min^{-1})				0.713	0.011	0.963
Fluid unresponsive cases (*N* = 15)	150 (122,170)	150 (118,167)	140 (112,158)			
Fluid responsive cases (*N* = 12)	145 (122,160)	145 (123,156)	138 (112,152)			
Mean arterial pressure (mmHg)				0.581	< 0.001	0.049
Fluid unresponsive cases (*N* = 15)	41 (39,66)	NA	48 (45,63)			
Fluid responsive cases (*N* = 12)	41 (37,47)	NA	53 (44,58)			
Mottling				0.392	0.046	0.817
Fluid unresponsive cases (*N* = 15)	3	NA	1			
Fluid responsive cases (*N* = 12)	4	NA	2			
Capillary refill time (s)				0.295	< 0.001	0.544
Fluid unresponsive cases (*N* = 15)	4 (3,4)	NA	2 (2,3.3)			
Fluid responsive cases (*N* = 12)	3 (3,4)	NA	2 (2,3)			
Urine output (mL.kg^{-1}.h^{-1})				0.662	0.007	0.180
Fluid unresponsive cases (*N* = 15)	1.4 (0.8,2.4)	NA	4.2 (3.0,5.4)			
Fluid responsive cases (*N* = 12)	1.6 (0.8,2.7)	NA	2.6 (1.0,4.6)			
Lactate (mmol.L^{-1})				0.841	0.940	0.559
Fluid unresponsive cases (*N* = 15)	2.5 (1.3,4.6)	NA	2.3 (1,3.4)			
Fluid responsive cases (*N* = 12)	2.7 (1.7,3.1)	NA	3.1 (1.9,3.7)			
VTI (cm)				0.717	< 0.001	< 0.001
Fluid unresponsive cases (*N* = 15)	9.6 (8.8,11.6)	9.4 (7.8,12.1)	10.2 (9.0,12.7)			
Fluid responsive cases (*N* = 12)	7.9 (7.2,10.6)	9.5 (8.8,11.9)	11.4 (9.8,13.9)			
SVi (mL.m^{-2})				0.453	< 0.001	< 0.001
Fluid unresponsive cases (*N* = 15)	14.5 (12.5,18.7)	14.0 (11.7,20.1)	15.3 (13.7,20.3)			
Fluid responsive cases (*N* = 12)	14.6 (12.2,17.8)	16.4 (15.2,20.4)	19.3 (17.4,23.9)			
Cardiac index (L.min^{-1}.m^{-2})				0.777	< 0.001	< 0.001
Fluid unresponsive cases (*N* = 15)	2.0 (1.6,3.3)	2.1 (1.6,3.3)	2.0 (1.6,3.0)			
Fluid responsive cases (*N* = 12)	2.0 (1.5,2.7)	2.2 (1.8,3.0)	2.7 (2.0,3.4)			

NA not available, *Svi* indexed stroke volume, *VE* volume expansion, *VTI* aortic velocity–time integral. Data are reported as No. or medians (first, third quartile)

output, CRT) were associated with fluid responsiveness in univariate analysis, although the association with post-VE MAP variation approached statistical significance. Binomial logistic regression analyses are reported in eTables 3–5 (Additional File).

Discussion

This study reported that SV changes induced by a calibrated abdominal compression maneuver could predict fluid responsiveness with a good diagnostic accuracy in PICU infants without underlying heart disease, regardless of their ventilation status. No other baseline parameter was independently associated with fluid responsiveness.

Our results are consistent with other studies on calibrated abdominal compression. In their study, Lee et al. found that the variation of diastolic arterial pressure during liver compression was predictive of fluid responsiveness, with an AUROC of 0.78 in children undergoing cardiac surgery [18]. They also investigated this test in children with single ventricle physiology and found the best diagnostic accuracy for systolic arterial pressure variation during liver compression (AUROC 0.93) [19]. Jacquet-Lagreze et al. used an SV-based approach and found an AUROC of ΔSV-AC of 0.94 to predict fluid responsiveness in 39 children, including 32 postoperative cases of congenital cardiac surgery [17]. However, the performance of fluid responsiveness tests might be significantly different in cardiac PICU [23], as the post-cardiotomy context affects the Frank-Starling curve morphology.

The diagnostic accuracy observed for ΔSV-AC in this study was similar to that of the ΔPeak, which is the most studied fluid responsiveness test in children [6, 7, 21]. The ΔSV-AC cut-off value of 9.5% measured in this study is consistent with previous measures reported in the literature, using various fluid responsiveness tests [5–7, 11, 17–19, 21, 24]. However, the ΔPeak is only validated in children without spontaneous breathing [7, 21]. This situation is rare in clinical practice, and the tests based on

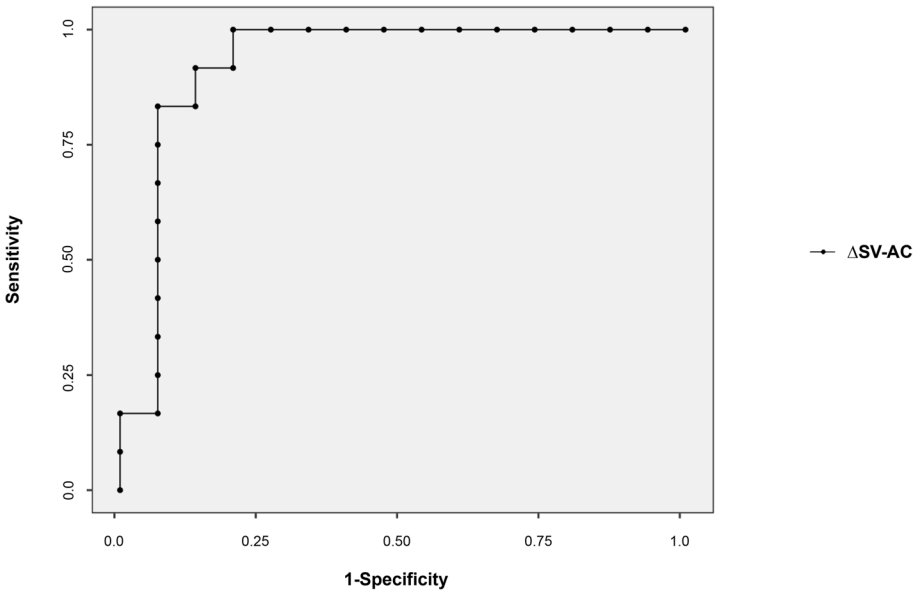

Fig. 2 Receiver operating characteristics (ROC) curve of ΔSV-AC to predict fluid responsiveness. Legend: ΔSV-AC, percentage of stroke volume variation between baseline and during a calibrated abdominal compression

cardiopulmonary interactions are often incorrectly used [25]. Likewise, respiratory variability of the inferior vena cava lacks reliability in spontaneously breathing children [26, 27]. Conversely, in our study, most patients were breathing spontaneously. In addition, fluid responsiveness prediction remains challenging in younger children. The ΔPeak might have a lower performance in children under 25 months of age [7], although a study in preterm neonates ventilated with low respiratory rate and without spontaneous breathing found an excellent diagnostic accuracy under these strict conditions (AUROC 0.91) [11]. Regarding passive leg raising, Luo et al. found an AUROC of 0.88 in 40 children, but the mean age was 3.7 years [15]. In our study, most of the patients were neonates. Calibrated abdominal compression may therefore be of particular interest in this younger population, when strict conditions for ΔPeak are not met.

Finally, no baseline parameter was independently associated with fluid responsiveness. Although this result may be due to a lack of power, it highlights the importance of using dynamic tests. Interestingly, no association was found between clinical assessment and fluid responsiveness. Likewise, no VE-induced change in hemodynamic parameters was associated with fluid responsiveness. However, MAP increase after VE was nearly significantly associated with fluid responsiveness. It is possible that with a slight increase in sample size this result would have been significant. Nevertheless, these results support the importance of monitoring cardiac output in PICU when assessing response to VE, especially as clinicians'ability to estimate cardiac output has been shown to be inadequate [28, 29].

Our study presents several limitations. First, the number of VE cases included was relatively small. However, it was enough to demonstrate our hypothesis in terms of statistical power. In addition, the population was very homogeneous in age compared with existing pediatric studies. Second, six patients were analyzed twice. Intrinsic patient factors could have influenced both ΔSV-AC and fluid responsiveness, but the sensitivity analysis does not support this hypothesis. Third, more advance invasive hemodynamic parameters would have been of interest. However, the non-interventional design of this study prohibited the collection of any data that was not already being measured as part of patient care. Fourth, fluid responsiveness was assessed using echocardiography, i.e., an operator-dependent examination. Nevertheless, reproducibility was excellent and echocardiography is widely used as a gold standard pediatric test [5, 8, 21], transpulmonary thermodilution being marginally used in children in clinical practice [29]. Fifth, few critically ill patients were included in the study, and most infants were on low doses of catecholamines and had low ventilatory settings. Our findings may therefore not be generalizable to more severely ill infants. Finally, this study included both non-intubated and intubated patients, with or without spontaneous breathing. Although inspiratory effort could have had a significant impact on the accuracy of the calibrated abdominal compression test, its diagnostic accuracy was excellent despite the inclusion

of these patients. Furthermore, ΔSV-AC remained associated with fluid responsiveness after adjustment for ventilation status, although the proportion of non-intubated patients was insufficient to analyze this subgroup specifically. Beyond these limits, as with any diagnostic test, predictive value is affected by prevalence. Clinicians should therefore interpret the test result with caution in patients with a high pre-test probability of being responders or non-responders.

Overall, these results suggest that calibrated abdominal compression might be one of the fluid responsiveness tests suitable for infants in non-cardiac PICU, whether ventilated or non-ventilated. A mini fluid challenge might also be of interest, but the calibrated abdominal compression test offers additional advantages: it does not require any volume administration, it is relatively simple and fast to perform, easy to learn, and it requires minimal equipment. Although this study includes a significant number of non-ventilated patients, further studies focusing on this specific population are warranted.

Conclusions

Echocardiographic assessment of SV changes induced by a calibrated abdominal compression is a promising method to predict fluid responsiveness in critically ill infants without cardiac disease. This maneuver can be performed regardless of the child's ventilation status.

Abbreviations

95%CI	95% Confidence Interval
ΔPeak	Respiratory variability of the peak aortic velocity
ΔSV-AC	Stroke Volume variations induced by the calibrated abdominal compression
ΔSV-VE	Stroke Volume variations induced by a volume expansion
AUROC	Area under the Receiver-Operating Characteristic curve
CI	Cardiac Index
CRT	Capillary Refill Time
HR	Heart Rate
LVEF	Left Ventricular Ejection Fraction
LVOTd	Left Ventricular Outflow Tract diameter
MAP	Mean Arterial Pressure
PICU	Pediatric Intensive Care Unit
ROC	Receiver-Operating Characteristic
SD	Standard Deviation
SV	Stroke Volume
VE	Volume Expansion
VTI	Velocity Time Integral

Supplementary Information

The online version contains supplementary material available at https://doi.org/10.1186/s12887-025-05728-z.

Supplementary Material 1.

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Authors' contributions

JGo: Conceptualization, Data curation, Formal analysis, Investigation, Methodology, Writing – original draft. AN: Conceptualization, Investigation, Writing – review & editing. YB: Data curation, Formal analysis, Methodology, Writing – review & editing. RK: Investigation, Writing – review & editing. PA: Supervision, Writing – review & editing JGu: Investigation, Methodology, Supervision, Writing – review & editing. OB: Project administration, Supervision, Writing – review & editing. All authors read and approved the final manuscript. All authors agree to be accountable for all aspects of the work.

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Data availability

The datasets used and/or analyzed during the current study are available from the corresponding author on reasonable request.

Declarations

Ethics approval and consent to participate

The study was carried out in accordance with the Good Clinical Practices protocol and Declaration of Helsinki principles. It was approved by our Institutional Review Board (Comité de Protection des Personnes Ouest IV, number 2021-A02876-35, approval date January 11 th 2022) and retrospectively registered on Clinicaltrials.gov (NCT05919719, June 22, 2023). Informed consent was obtained from all parents or legal guardians for minors, before or within 24 h after study procedures.

Consent for publication

Not applicable.

Competing interests

The authors declare no competing interests.

Author details

[1]Department of Pediatric and Congenital Cardiology, M3C National Reference Center, Bordeaux University Hospital, Bordeaux, France. [2]IHU Liryc, INSERM 1045, University of Bordeaux, Bordeaux, France. [3]Pediatric Intensive Care Unit, Children's Hospital, Bordeaux University Hospital, Bordeaux, France. [4]Department of Thoracic Surgery, Haut-Leveque Hospital, Bordeaux University Hospital, Pessac, France.

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