BJA

British Journal of Anaesthesia 115 (1): 38–44 (2015)

doi: 10.1093/bja/aev109
Advance Access Publication Date 29 April 2015
Cardiovascular

CARDIOVASCULAR

Prediction of fluid responsiveness using a non-invasive cardiac output monitor in children undergoing cardiac surgery

J.-H. Lee, H.-J. No, I.-K. Song, H.-S. Kim, C.-S. Kim, and J.-T. Kim*

Department of Anaesthesiology and Pain Medicine, Seoul National University Hospital, # 101 Daehakno, Jongnogu, Seoul 110-744, Republic of Korea

*Corresponding author. E-mail: kimjintae73@dreamwiz.com

Abstract

Background: This study evaluated the ability of a non-invasive cardiac output monitoring device (NICOM) to predict fluid responsiveness in paediatric patients undergoing cardiac surgery.

Methods: Children aged <5 yr undergoing congenital heart surgery were included. Once the sternum had been closed after repair of the congenital heart defect, 10 ml kg^{-1} colloid solution was administered for volume expansion. Transoesophageal echocardiography (TOE) was performed to measure stroke volume (SV) and respiratory variation in aortic blood flow peak velocity (ΔV_{peak}) before and after volume expansion. Haemodynamic and NICOM variables, including SV_{NICOM}, stroke volume variance (SVV_{NICOM}), cardiac index (CI_{NICOM}), and percentage change in thoracic fluid content compared with baseline (TFCd0%), were also recorded. Patients in whom the stroke volume index (SVI), measured using TOE, increased by >15% were defined as fluid responders.

Results: Twenty-nine patients were included (13 responders and 16 non-responders). Before volume expansion, only ΔV_{peak} differed between groups (P=0.036). The SVV_{NICOM}, HR, and central venous pressure did not predict fluid responsiveness, but ΔV_{peak} did. The CI_{NICOM} was not correlated with CI_{TOE} (r=0.107, P=0.43). Using Bland–Altman analysis, the mean bias between CI_{TOE} and CI_{NICOM} was 0.89 litre min^{-1} m^{-2}, with a precision of 1.14 litre min^{-1} m^{-2}. Trending ability of NICOM for SVI and CI was poor when TOE was a reference method.

Conclusions: The SVV_{NICOM} did not predict fluid responsiveness in paediatric patients during cardiac surgery. In addition, there was no correlation between CI_{TOE} and CI_{NICOM}. Fluid management guided by NICOM should be performed carefully.

Clinical trial registration: ClinicalTrials.gov NCT01996956.

Key words: cardiac output; cardiac surgical procedure; fluid therapy; paediatrics; stroke volume

In paediatric patients undergoing cardiac surgery, adequate fluid management is important. Hypovolaemia can impair organ perfusion and cause metabolic acidosis; however, excessive fluid overload can cause acute ventricular dysfunction and pulmonary oedema.

It is more difficult to predict the fluid responsiveness in children compared with adults. In adults, pulse pressure variation and stroke volume variation (SVV) reliably predict the fluid loading response and are superior to traditional central venous pressure (CVP).[1–4] However, evidence that these dynamic variables predict fluid responsiveness in children is limited, with conflicting results.[5–8] Except for respiratory variation in aortic blood flow peak velocity (ΔV_{peak}), no variables reliably predicted fluid responsiveness in paediatric patients.[9] Furthermore, the need for non-invasive fluid management methods in children continues to increase.

Accepted: February 10, 2015

Editor's key points

- Fluid responsiveness in paediatric patients is difficult to predict, and there are few data on the validity of non-invasive cardiac output monitoring in children.
- This study found that stroke volume variation estimated using the NICOM device did not predict fluid responsiveness.
- There was also poor correlation between cardiac index assessed using NICOM or transoesophageal echocardiography.
- Clinicians should interpret variables derived using NICOM with caution in children undergoing cardiac surgery.

The non-invasive cardiac output monitoring device (NICOM; Cheetah Medical, Wilmington, DE, USA) uses a non-invasive technique, based on transthoracic bioreactance, which applies a high-frequency electrical current of known amplitude and frequency across the thorax and detects the relative phase shift between the injected current and recorded voltage. Phase shifts occur as a result of pulsatile changes in blood volume in the aorta. Several studies have demonstrated the clinical usefulness and feasibility of the NICOM in adults,[10–14] but its effectiveness in children remains controversial.[15 16] However, in one study, NICOM values predicted fluid responsiveness non-invasively in children in the intensive care unit.[17] During operations, the NICOM may facilitate volume administration in paediatric patients.

In the present study, we assessed whether SVV values measured using NICOM (SVV_{NICOM}) can predict fluid responsiveness in children after repair of congenital heart defects and compared NICOM-derived values with other possible predictors of fluid responsiveness. We also evaluated the accuracy of cardiac index (CI) measured using NICOM (CI_{NICOM}) *vs* transoesophageal echocardiography (TOE; CI_{TOE}).

Methods

The study was approved by the Institutional Review Board of Seoul National University Hospital and was registered at ClinicalTrials.gov (NCT01996956). Written informed consent was provided by all parents. Children <5 yr of age undergoing congenital heart surgery were enrolled. Patients with a single ventricle, right heart failure, renal failure, respiratory disease, any cardiac arrhythmia, or moderate or severe valvular stenosis and regurgitation were excluded.

No premedications were administered. Anaesthesia was induced with midazolam (0.1 mg kg^{-1}), thiopental (5 mg kg^{-1}) and atropine (0.02 mg kg^{-1}). Rocuronium (0.6 mg kg^{-1}) and fentanyl (5–10 μg kg^{-1}) were used for endotracheal intubation. Anaesthesia was maintained using midazolam (0.1–0.2 mg kg^{-1} h^{-1}), sufentanil (2.5–5 μg kg^{-1} h^{-1}), and vecuronium (0.05–0.1 mg kg^{-1} h^{-1}). Mechanical ventilation was controlled to obtain an arterial partial pressure of CO_2 of 35–40 mm Hg, with a constant tidal volume of 10 ml kg^{-1} maintained during surgery.

After induction of anaesthesia, a peripheral arterial catheter was placed at the left or right radial artery; a central venous catheter was inserted into the right internal jugular vein. Monitoring using the NICOM device commenced with four NICOM dual electrodes attached to the patient's back in accordance with the manufacturer's instructions. A TOE probe (either s8–3t or s7–3t; iE33 Echocardiography System; Philips, Andover, MA, USA) was then inserted.

Following weaning from cardiopulmonary bypass, lung recruitment was performed to prevent atelectasis. Ventilation was commenced using a tidal volume of 10 ml kg^{-1}; the respiratory rate was controlled to maintain normocarbia.

Experimental protocol

The NICOM was recalibrated when the patients were fully weaned from cardiopulmonary bypass. After closure of the sternum, baseline NICOM-derived variables [stroke volume (SV_{NICOM}), SVV_{NICOM}, CI_{NICOM}, and percentage change in thoracic fluid content compared with baseline (TFCd0%)] were recorded. Volulyte® (6% hydroxyethyl starch 130/0.4) at 10 ml kg^{-1} was administered for 10 min; after fluid loading, NICOM variables were recorded again. The mean of three serial NICOM values was used in the analysis. Peak inspiratory pressure, CVP, arterial blood pressure, body temperature, and vasoactive and inotropic agent doses were also recorded during the experimental period. Inotropic agent use (vasoactive–inotropic score)[18] was then calculated as follows: dopamine dose (in μg kg^{-1} min^{-1}) + dobutamine dose (in μg kg^{-1} min^{-1}) + [epinephrine dose × 100 (in μg kg^{-1} min^{-1})] + [milrinone dose × 100 (in μg kg^{-1} min^{-1})] + [vasopressin dose × 10 000 (in U kg^{-1} min^{-1})] + [norepinephrine dose × 100 (in μg kg^{-1} min^{-1})].

Echocardiographic measurement

All echocardiography data were measured by a single expert, who was blinded to all values measured by NICOM.

Stroke volume and cardiac index

To measure SV_{TOE}, the aortic annulus diameter (D) was measured during the systolic phase mid-oesophageal aortic valve long-axis view. A TOE probe was then advanced to obtain a deep transgastric long-axis view. Using pulsed wave Doppler, the aortic blood flow waveform at the level of the aortic annulus was recorded. The mean velocity time integral (VTI) was calculated from three consecutive waves at the end of the expiratory period.

Variables were calculated as follows:

$$SV = 3.14^{*}(D/2)^{2},$$

$$CO = SV \times HR,$$

$$CI = CO \times BSA^{-1},$$

where CO is the cardiac output, HR the heart rate, and BSA the body surface area.

Respiratory variation in aortic blood flow peak velocity

Using pulsed wave Doppler, the maximal and minimal blood flow velocity (V_{peak}) during one respiratory cycle was measured, at the aortic valve level in the deep transgastric long-axis view. The respiratory variation in V_{peak} (ΔV_{peak}) was calculated as follows:

$$\Delta V_{peak}(\%) = (V_{peak,max} - V_{peak,min}) \times \left[(V_{peak,max} + V_{peak,min})2^{-1}\right]^{-1} \times 100.$$

The mean ΔV_{peak} was calculated using three consecutive respiratory cycles.

Statistics

The required number of patients was determined according to data from previous studies, in which the area under the receiver operating characteristic (ROC) curve for variable predictors

ranged from 0.77 to 0.86, with a responder to non-responder ratio of approximately 1:1.[5 7] We assumed that the SVV_{NICOM} would predict fluid responsiveness with an area under the ROC curve of >0.8. We calculated a required sample size of 29 using the PASS software 2008 (version 8.0.16; NCSS Statistical Software, Kaysville, UT, USA), with an α error of 0.05, a power of 0.8, and an estimated 10% attrition rate.

Table 1 Patient characteristics and intraoperative variables expressed as mean (SD or range) or number. ASD, atrial septal defect; AVSD, atrioventricular septal defect; TOF, tetralogy of Fallot; VSD, ventricular septal defect; ACC, aorta cross clamping

Characteristic	Responders (*n*=13)	Non-responders (*n*=16)
Sex (male/female)	6/7	9/7
Age (months)	16.9 (2–36)	11.1 (1–36)
Weight (kg)	9.4 (2.3)	8.7 (5.3)
Height (cm)	77.6 (11.1)	71.7 (16.5)
Operation		
VSD	3	2
VSD+ASD	2	5
VSD+infundibular muscle resection	2	1
ASD	6	6
AVSD	0	1
TOF	0	1
Peak inspiratory pressure (cm H_2O)	12.8 (2.0)	13.3 (2.9)
Temperature (°C)	35.7 (0.7)	35.8 (0.6)
Cardiopulmonary bypass time (min)	105.2 (37.5)	104.8 (36.1)
ACC time (min)	57.6 (27.3)	59.9 (26.3)
Vasoactive–inotropic score	8.8 (2.7)	9.9 (3.7)

Following fluid challenge, we defined two study groups: the fluid responder group, comprising patients in whom fluid administration increased SVI_{TOE} by >15%; and the non-responder group, comprising the remaining patients.[7 17]

Statistical analyses were performed using SPSS (version 20; SPSS Inc., Chicago, IL, USA) and MedCalc software (version 12.7.7; MedCalc, Ostend, Belgium). Student's unpaired *t*-test or the Mann–Whitney *U*-test was used to evaluate group differences. To determine the ability of all variables to predict fluid responsiveness, ROC curves were generated, and the area under the ROC curve was calculated. To assess correlation between TOE and NICOM in SV, SVI, and CI, linear regression analysis was performed. The concordance between CI_{NICOM} and CI_{TOE} was evaluated using the Bland–Altman method. The mean difference (bias) and precision (SD) between CI_{NICOM} and CI_{TOE} was also calculated. The 95% limits of agreement were calculated using the interval defined by the observed bias (1.96 SD). Polar plot was used to evaluate the trending ability of NICOM compared with TOE. A value of $P<0.05$ was taken to indicate statistical significance.

Results

The study included a total of 29 paediatric patients (15 males and 14 females) who had undergone either ventricular septal defect (*n*=5), ventricular septal defect with atrial septal defect (*n*=7), ventricular septal defect with infundibular muscle resection (*n*=3), atrial septal defect (*n*=12), atrioventricular septal defect (*n*=1), or tetralogy of Fallot operations (*n*=1). After surgery, no significant valvular regurgitation or ventricular dysfunction occurred in any patient. Table 1 lists the patient characteristics and intraoperative variables.

In total, 13 patients were volume responders and 16 non-responders (Fig. 1). No significant group difference was observed in clinical characteristics or intraoperative variables. Before volume expansion, only the ΔV_{peak} differed between the two groups

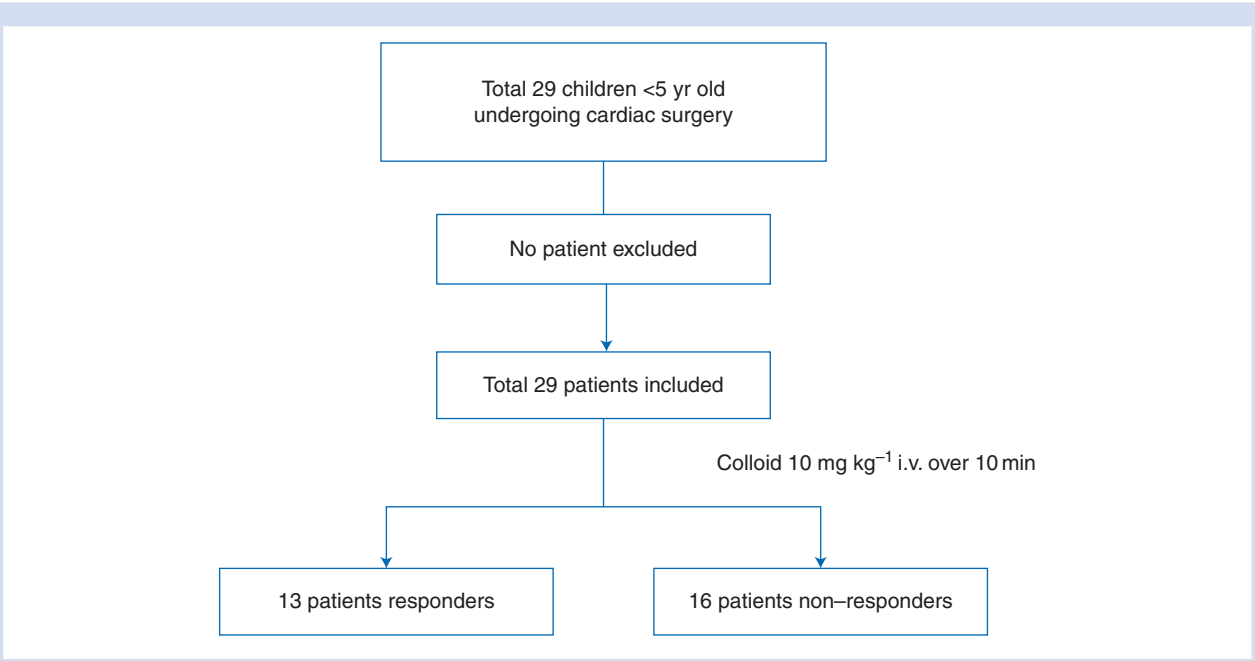

Fig 1 Consolidated Standards of Reporting Trials (CONSORT) diagram.

Table 2 Haemodynamic variables before and after fluid loading. CI, cardiac index; FL, fluid loading; NICOM, non-invasive cardiac output monitoring device; SVI, stroke volume index; SVV, stroke volume variation; TFCd0%, percentage change in thoracic fluid content compared with baseline; TOE, transoesophageal echocardiography. *$P < 0.05$ before FL *vs* after FL. †$P < 0.05$ between responders and non-responders in both before FL. *P*-value was adjusted for multiple comparisons. Variables are expressed as mean (SD) or number

Variable	Responders (n=13)		Non-responders (n=16)	
	Before FL	After FL	Before FL	After FL
Heart rate (beats min^{-1})	135 (22)	127 (22)*	136 (22)	135 (21)
Systolic arterial pressure (mm Hg)	97 (12)	116 (12)*	94 (17)	108 (14)*
Diastolic arterial pressure (mm Hg)	47 (4)	63 (14)*	48 (9)	54 (6)*
Mean arterial pressure (mm Hg)	61 (6)	74 (12)*	64 (12)	74 (9)*
Central venous pressure (mm Hg)	5 (3)	8 (3)*	6 (3)	9 (3)*
SVI_{TOE} (ml m^{-2})	20.5 (6.2)	27.4 (8.2)*	22.9 (8.6)	25.7 (8.9)*
CI_{TOE} (litre min^{-1} m^{-2})	2.7 (0.6)	3.2 (0.8)*	3.2 (1.4)	3.2 (1.3)
SVI_{NICOM} (ml m^{-2})	16.0 (4.8)	19.1 (7.4)*	14.4 (4.9)	16.2 (5.7)*
CI_{NICOM} (litre min^{-1} m^{-2})	2.1 (0.4)	2.1 (0.9)	2.0 (0.5)	2.2 (0.6)
SVV_{NICOM} (%)	13.6 (4.4)	11.4 (4.6)*	13.8 (4.3)	12.8 (4.1)
TFCd0%	3.4 (7.6)	6.1 (8.1)*	1.5 (3.9)	4.2 (4.5)*
ΔV_{peak} (%)†	15.5 (6.3)	8.7 (2.6)*	10.9 (4.4)	12.6 (4.6)*

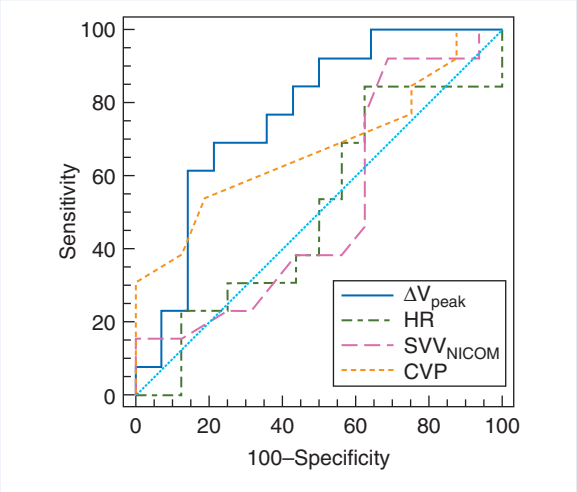

Fig 2 Comparison of areas under receiver operating characteristic curves before volume expansion. The areas under the curve for SVV_{NICOM}, HR, CVP, and ΔV_{peak} were 0.51 (95% confidence interval 0.32–0.70), 0.507 (95% confidence interval 0.32–0.70), 0.68 (95% confidence interval 0.48–0.84), and 0.77 (95% confidence interval 0.57–0.91), respectively. A ΔV_{peak} of >13.5% alone predicted fluid responsiveness, with a sensitivity of 69.2% and specificity of 78.6%. CVP, central venous pressure; HR, heart rate; SVV_{NICOM}, stroke volume variation measured by non-invasive cardiac output monitoring device (NICOM); ΔV_{peak}, respiratory variation in aortic blood flow peak velocity.

(*P*=0.036). Volume expansion significantly changed blood pressure, CVP, SVI_{TOE}, SVI_{NICOM}, TFCd0%, and ΔV_{peak} in both groups, but HR, CI_{TOE}, and SVV_{NICOM} were changed only in the responders (Table 2).

Figure 2 illustrates the ROC curve analysis. Fluid responsiveness was not predicted by SVV_{NICOM}, HR, and CVP, but ΔV_{peak} predicted a 15% increase in SVI (*P*=0.005). The optimal cut-off value of ΔV_{peak} was 13.5%, with a specificity of 69.2% and sensitivity of 78.6%. There was positive relationship between SVV_{NICOM} and ΔV_{peak} (r=0.416, *P*=0.031). However, SVV_{NICOM} did not reflect the changes in SVI_{TOE} induced by volume expansion (r=0.08, *P*=0.7).

Values of SV_{NICOM} and SVI_{NICOM} were correlated with SV_{TOE} and SVI_{TOE} (r=0.751 and 0.409, *P*<0.001 and *P*=0.001, respectively). However, CI_{NICOM} was not correlated with CI_{TOE} (r=0.107, *P*=0.43; Fig. 3). The CI_{NICOM} values were 2.12 (0.56) litre min^{-1} m^{-2}, which was significantly lower than the CI_{TOE} value of 3.01 (1.07) litre min^{-1} m^{-2} (*P*<0.01). Using Bland–Altman analysis, the mean bias and precision of CI_{NICOM} and CI_{TOE} were 0.89 and 1.14 litre min^{-1} m^{-2}, respectively (Fig. 4). The limits of agreement were −1.3 and 3.1 litre min^{-1} m^{-2}.

Polar plots were created to assess the trending ability of NICOM for SVI and CI when TOE was a reference method; however, it showed poor trending ability (Fig. 5).

Discussion

This study demonstrated that the NICOM device cannot be used to predict fluid responsiveness in paediatric patients <5 yr of age undergoing cardiac surgery. The correlation between NICOM- and TOE-derived CI following volume expansion was weak. In addition, the trending ability of NICOM was poor.

The NICOM device uses bioreactance to measure cardiac output, with four dual electrodes attached to the thorax; this has been considered as an innovative method that is potentially applicable in children.[15–17] A low-amplitude, high-frequency current is delivered and picked up by the electrodes, with the frequency of relative phase shifts through the thorax analysed to calculate SV; this method appears to be less affected by interference than other noninvasive technique such as bioimpedance.

Regarding cardiac output measurement in adult patients, NICOM exhibited clinically acceptable accuracy, precision, and responsiveness to haemodynamic changes in various clinical settings.[10 11 13 19] However, data pertaining to paediatric populations are controversial.[15–17 20] Some studies report a strong correlation between NICOM and echocardiographic measurements of CI in both neonates and children.[16 17] However, in another report using a paediatric haemorrhagic shock model, CI values measured by bioreactance were not correlated with values recorded using the pulmonary artery thermodilution technique and did not

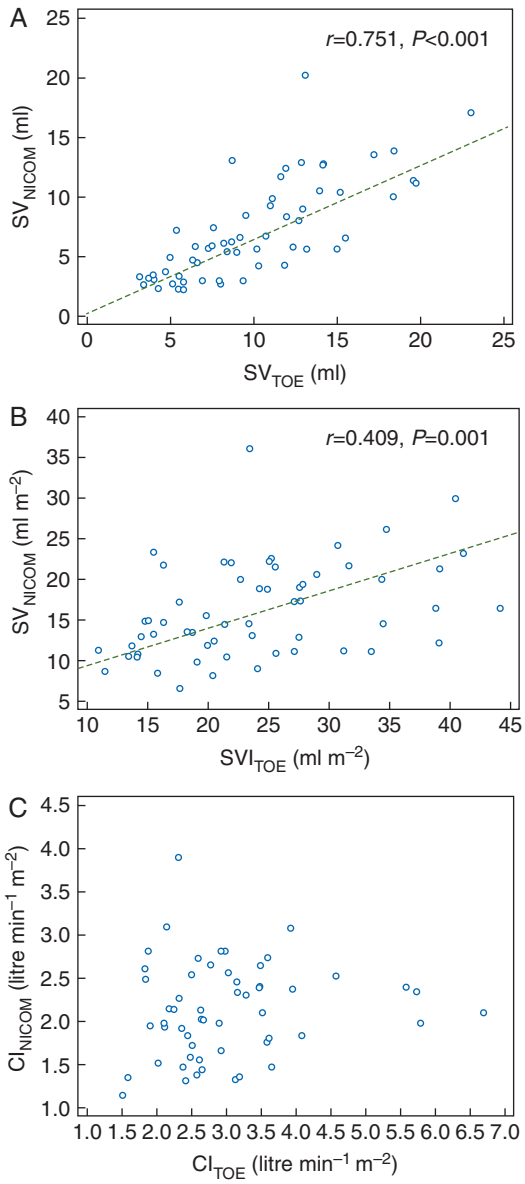

Fig 3 Correlation between the transoesophageal echocardiography (TOE) and non-invasive cardiac output monitoring device (NICOM) variables. (A) Stroke volume (SV). (B) Stroke volume index (SVI). (C) Cardiac index (CI). Dotted lines represent the estimate statistical formulae derived from linear regression analysis.

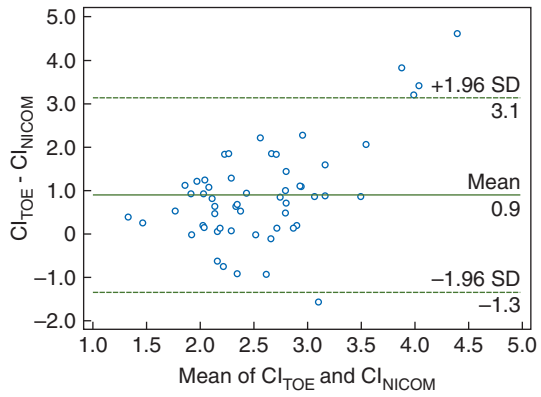

Fig 4 Bland–Altman plot of cardiac indices measured by NICOM and TOE. The bias and precision of the two methods were 0.89 and 1.14 litre min^{-1} m^{-2}, respectively.

change significantly during hypovolaemia and volume expansion.[20] In addition, CI values measured by bioreactance differed according to the weight of the patient and were lower than normal-range values, particularly in smaller children.[15] Similar results were obtained in another neonate study,[16] in which NICOM underestimated CI values when compared with echocardiographic data; in the present study, NICOM also underestimated CI values by ~30% compared with TOE. We observed no correlation between CI_{NICOM} and CI_{TOE} values. Our data suggest that bioreactance may not be a suitable measure of CI in small children.

Predicting fluid responsiveness in children is important in instances of low cardiac output. The predictive ability of several haemodynamic variables of fluid responsiveness has been evaluated. Static variables, such as HR and CVP, were assessed during major paediatric operations, with area under the ROC curve values of 0.53–0.55 in HR and 0.47–0.61 in CVP.[5 7 21] In our study, the areas under the ROC curves of HR and CVP were 0.51 (95% confidence interval 0.32–0.70) and 0.68 (95% confidence interval 0.48–0.84), respectively. Although typically used to estimate intravascular volume status, HR and CVP are poor predictors of fluid responsiveness.

The ΔV_{peak} has been known to be the only variable shown to predict fluid responsiveness in children.[9] The ΔV_{peak} is considered an accurate method of evaluating preload in ventilated patients with septic shock.[22] Suggested cut-off values are 7–20%.[5 6 21 23] Variations in study population, such as surgical patients without concomitant disease or critically ill patients, explain the wide range of cut-off values.[23] In the present study, ΔV_{peak} was also a good predictor of fluid responsiveness, with cut-off values of ~13.5%, consistent with previous studies.[5 23]

Although evidence that SVV can be a predictor of fluid responsiveness is still insufficient,[9] there was still a possibility that SVV would be a target parameter for volume management in children.[8] Recently, two studies showed that the dynamic parameters of NICOM can guide fluid management in paediatric patients after major surgery.[24 25] According to these reports, SVV_{NICOM} reliably predicts fluid responsiveness with an area under the ROC curve of 0.8–0.9 and cut-off value of 10%. Therefore, we also anticipated that intraoperative SVV_{NICOM} would be a useful indicator of volume response in paediatric patients immediately after cardiopulmonary bypass. However, in our study there was no group difference in SVV_{NICOM} values before fluid challenge, in contrast with a previous study;[24] furthermore, these values did not predict fluid responsiveness during paediatric cardiac surgery. There are several possible reasons for this. First, the accuracy of NICOM may be reduced in small children because of initial calibration difficulties.[15] Second, colloid infusion of 10 ml kg^{-1} can induce haemodilution, and reduced haemoglobin could affect bioreactance readings by altering the iron content of the thorax,[26] thus affecting NICOM accuracy.[19] Finally, SVV_{NICOM} itself may not be a good predictor in younger paediatric patients because of reduced thoracic, lung, and vascular compliance.[9] In a recent study,[25] SVV_{NICOM} reliably predicted fluid responsiveness only in children aged >3 yr, while an area under

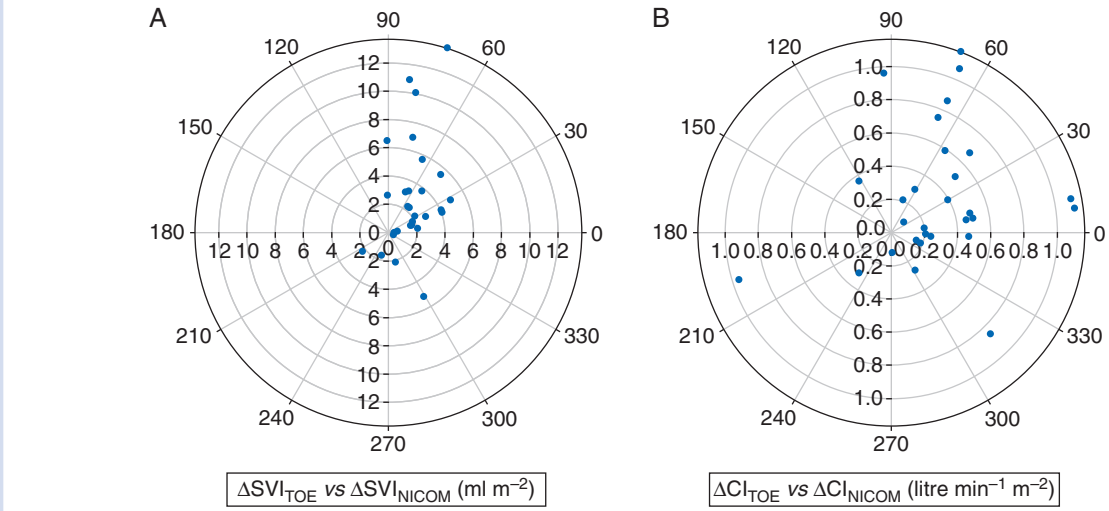

Fig 5 Polar plots between TOE and NICOM. The trending ability of NICOM for SVI and CI, when compared with TOE, was analysed. Many dots are outside (30°) radial limits in both (A) and (B), which means that NICOM showed poor trending ability for monitoring changes in cardiac output.

the ROC curve was 0.57 in younger children. This result coincides with our findings, because the range of age of our patients was between 1 and 36 months.

The present study had several limitations. First, echocardiographic measurement errors may have occurred, even though the same expert obtained all of the echocardiographic data. Second, electrodes were sometimes folded or applied to different sites because of limited space in smaller patients, which may have influenced the NICOM variables. Finally, the reason for inaccuracy of NICOM has not been clearly determined yet, as commented in a previous report.[26]

In conclusion, SVV measured using the NICOM system did not predict fluid responsiveness in paediatric patients during cardiac surgery. In addition, there was no correlation between the CI obtained using NICOM and echocardiography. Careful fluid management is needed when using NICOM during paediatric cardiac surgery.

Authors' contributions

L-J.H.: patient recruitment, data collection, data analysis, and writing up of the first draft of the paper. N-H.J.: patient recruitment, data collection, and analysis. S-I.K: data analysis and revision of the article. K-H.S: correction and discussion of all results and approval of final version. K-C.S: revision of the article and approval of final version. K-J.T: study design, data analysis, revision of the manuscript, and approval of final version.

Declaration of interest

None declared.

References

1. Cannesson M, Attof Y, Rosamel P, *et al*. Respiratory variations in pulse oximetry plethysmographic waveform amplitude to predict fluid responsiveness in the operating room. *Anesthesiology* 2007; **106**: 1105–11
2. Hofer CK, Müller SM, Furrer L, Klaghofer R, Genoni M, Zollinger A. Stroke volume and pulse pressure variation for prediction of fluid responsiveness in patients undergoing off-pump coronary artery bypass grafting. *Chest* 2005; **128**: 848–54
3. Michard F. Changes in arterial pressure during mechanical ventilation. *Anesthesiology* 2005; **103**: 419–28; quiz 449–5
4. Marik PE, Cavallazzi R, Vasu T, Hirani A. Dynamic changes in arterial waveform derived variables and fluid responsiveness in mechanically ventilated patients: a systematic review of the literature. *Crit Care Med* 2009; **37**: 2642–7
5. Byon HJ, Lim CW, Lee JH, *et al*. Prediction of fluid responsiveness in mechanically ventilated children undergoing neurosurgery. *Br J Anaesth* 2013; **110**: 586–91
6. Pereira de Souza Neto E, Grousson S, Duflo F, *et al*. Predicting fluid responsiveness in mechanically ventilated children under general anaesthesia using dynamic parameters and transthoracic echocardiography. *Br J Anaesth* 2011; **106**: 856–64
7. Renner J, Broch O, Duetschke P, *et al*. Prediction of fluid responsiveness in infants and neonates undergoing congenital heart surgery. *Br J Anaesth* 2012; **108**: 108–15
8. Tran H, Froese N, Dumont G, Lim J, Ansermino JM. Variation in blood pressure as a guide to volume loading in children following cardiopulmonary bypass. *J Clin Monit Comput* 2007; **21**: 1–6
9. Gan H, Cannesson M, Chandler JR, Ansermino JM. Predicting fluid responsiveness in children: a systematic review. *Anesth Analg* 2013; **117**: 1380–92
10. Keren H, Burkhoff D, Squara P. Evaluation of a noninvasive continuous cardiac output monitoring system based on thoracic bioreactance. *Am J Physiol Heart Circ Physiol* 2007; **293**: H583–9
11. Raval NY, Squara P, Cleman M, Yalamanchili K, Winklmaier M, Burkhoff D. Multicenter evaluation of noninvasive cardiac output measurement by bioreactance technique. *J Clin Monit Comput* 2008; **22**: 113–9
12. Lee MF, Chen WS, Fu TC, *et al*. Non-invasive cardiac index monitoring during cardiopulmonary functional testing provides additional prognostic value in patients after acute heart failure. *Int Heart J* 2012; **53**: 364–9

13. Squara P, Denjean D, Estagnasie P, Brusset A, Dib JC, Dubois C. Noninvasive cardiac output monitoring (NICOM): a clinical validation. *Intensive Care Med* 2007; **33**: 1191–4
14. Marik PE, Levitov A, Young A, Andrews L. The use of bioreactance and carotid Doppler to determine volume responsiveness and blood flow redistribution following passive leg raising in hemodynamically unstable patients. *Chest* 2013; **143**: 364–70
15. Ballestero Y, López-Herce J, Urbano J, *et al*. Measurement of cardiac output in children by bioreactance. *Pediatr Cardiol* 2011; **32**: 469–72
16. Weisz DE, Jain A, McNamara PJ, El-Khuffash A. Non-invasive cardiac output monitoring in neonates using bioreactance: a comparison with echocardiography. *Neonatology* 2012; **102**: 61–7
17. Lee JY, Kim JY, Choi CH, Kim HS, Lee KC, Kwak HJ. The ability of stroke volume variation measured by a noninvasive cardiac output monitor to predict fluid responsiveness in mechanically ventilated children. *Pediatr Cardiol* 2014; **35**: 289–94
18. Gaies MG, Gurney JG, Yen AH, *et al*. Vasoactive–inotropic score as a predictor of morbidity and mortality in infants after cardiopulmonary bypass. *Pediatr Crit Care Med* 2010; **11**: 234–8
19. Marque S, Cariou A, Chiche JD, Squara P. Comparison between Flotrac-Vigileo and Bioreactance, a totally noninvasive method for cardiac output monitoring. *Crit Care* 2009; **13**: R73
20. Ballestero Y, Urbano J, López-Herce J, *et al*. Pulmonary arterial thermodilution, femoral arterial thermodilution and bioreactance cardiac output monitoring in a pediatric hemorrhagic hypovolemic shock model. *Resuscitation* 2012; **83**: 125–9
21. Renner J, Broch O, Gruenewald M, *et al*. Non-invasive prediction of fluid responsiveness in infants using pleth variability index. *Anaesthesia* 2011; **66**: 582–9
22. Feissel M, Michard F, Mangin I, Ruyer O, Faller JP, Teboul JL. Respiratory changes in aortic blood velocity as an indicator of fluid responsiveness in ventilated patients with septic shock. *Chest* 2001; **119**: 867–73
23. Choi DY, Kwak HJ, Park HY, Kim YB, Choi CH, Lee JY. Respiratory variation in aortic blood flow velocity as a predictor of fluid responsiveness in children after repair of ventricular septal defect. *Pediatr Cardiol* 2010; **31**: 1166–70
24. Lee JY, Kim JY, Choi CH, Kim HS, Lee KC, Kwak HJ. The ability of stroke volume variation measured by a noninvasive cardiac output monitor to predict fluid responsiveness in mechanically ventilated children. *Pediatr Cardiol* 2014; **35**: 289–94
25. Vergnaud E, Vidal C, Verchère J, *et al*. Stroke volume variation and indexed stroke volume measured using bioreactance predict fluid responsiveness in postoperative childrendagger. *Br J Anaesth* 2015; **114**: 103–9
26. Kupersztych-Hagege E, Teboul JL, Artigas A, *et al*. Bioreactance is not reliable for estimating cardiac output and the effects of passive leg raising in critically ill patients. *Br J Anaesth* 2013; **111**: 961–6

Handling editor: J. P. Thompson