calculation’. As presented in our paper, we used the mean of CO values displayed by the Mostcare monitor within three 1 min periods around the thermodilution measurements. Additionally, the measurements were performed in stable patients. Thus, it is rather unlikely that we might have missed relevant drifts in CO.

We thank the authors for detecting two mistakes in our paper. The first one relates to the fact that we were unprecise in writing ‘aortic replacement’ instead of the procedure that was really performed: aortic valve replacement. The second one relates to the indicated range of 2.6–9.2 litre min⁻¹ for PA-ThD and 2.4–12 litre min⁻¹ for PRAM-CO that was a mistake due to restructuring of the paper before patients with intra-aortic balloon pump were excluded. The correct range—as presented in Figures 1–4—is 3.55–7.0 litre min⁻¹ for PA-ThD and 2.4–12.5 litre min⁻¹ for PRAM-CO.

We agree with the authors that the pitfalls and limitations of PCMs should be clearly addressed in any studies on this technology. However, from a clinician’s point of view, it is questionable that such a delicate technology may be useful in daily practice. The clinical user of a non-invasive haemodynamic monitor does not have information about aortic pathologies, aortic valve diseases, or both: he simply takes the monitor to a patient to obtain information about haemodynamics for optimizing therapy. And it is highly doubtful if he will truly succeed in doing this, if the information derived from the monitor needs to be interpreted with respect to unknown variables and is of unknown reliability.

**Conflict of interest**

M.H. receives honoraria for lectures from Edwards Lifesciences, Germany.

M. Heringlake*
H. Paarmann
H. V. Groesdonk
B. Sedemund-Adib
T. Hanke
H. Heinze
J. Schoen
Lübeck, Germany

*E-mail: heringlake@t-online.de


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**Predicting fluid responsiveness in children: are the studied indicators of value in the setting of loss and replacement?**

Editor—We read with interest the recent article which aimed to validate, in a paediatric population, various dynamic variables derived from invasive and non-invasive monitors that are of demonstrated utility in adult patients.1 Interestingly, none of the dynamic variables derived from the arterial and plethysmographic waveforms were shown to reliably predict fluid responsiveness in children.

The authors point out that ‘static indicators such as central venous pressure, pulmonary capillary wedge pressure, or left ventricular end-diastolic area have been shown to be poor indicators of fluid responsiveness’, and ‘preload variables, such as central venous pressure or left ventricular end-diastolic area, are invasive or operator-dependent and have been shown to be poor predictors of fluid responsiveness’. Nearly all of the data upon which these statements are based come from studies in adults, most of which were conducted in the clinical setting of critical illness or after aortic or cardiac surgery.2 3 The current study presents left ventricular end-diastolic area data that support these statements in a paediatric population.1 Two other studies in children reached similar conclusions. One involved 12 subjects, primarily adolescents in the prone position.4 In the other, nearly all of the children had sepsis/multiorgan failure or were post-cardiac surgery.5

Clinically significant hypovolaemia was unlikely to have been present in any of the subjects in this study.1 To the practicing clinician, predicting fluid responsiveness in patients without haemodynamic instability may be interesting but not necessarily clinically relevant. Would the performance of the variables evaluated in this study be different in the presence of actual hypovolaemia in the setting of haemorrhage and replacement?

Given the different contexts from which the available data on this subject have been derived, we wonder how to apply the results of these studies to our care of elective paediatric surgical patients with the potential for significant blood loss and fluid shifts. While it may be the case that many static and dynamic indicators are of little value in children, a similarly carefully conducted study in the setting of clinically significant hypovolaemia and fluid resuscitation is needed to convince clinicians of the value (or lack thereof) of the various monitoring modalities in question.

**Conflict of interest**

None declared.

P. A. Stricker*
J. E. Fiajdie
D. R. Jobes
Philadelphia, USA

*E-mail: strickerp@email.chop.edu
Reply from the authors

Editor—We would like to thank Dr Stricker and colleagues for their constructive feedback and comments regarding our study.1 We concur that further studies in the setting of clinically significant hypovolaemia are needed in order to assess the accuracy of dynamic parameters of fluid responsiveness in children. While dynamic parameters have been demonstrated to be strong predictors of fluid responsiveness in adults,2 their accuracy in children is still debated and recently published papers, including animal ones, have found conflicting results.3–6 In our study, we have shown that dynamic variables such as respiratory variation in arterial pulse pressure (DeltaPP and PPV) or in the plethysmographic waveform amplitude (DeltaPOP and PVI) were not able to predict fluid responsiveness in children undergoing general surgery.3

Dr Stricker points out that these variables may not be reliable in the setting we studied (neurosurgical patients immediately after induction of anaesthesia) but that they may be accurate during severe hypovolaemia. We personally believe that these indices may detect hypovolaemia and may be predictive of fluid responsiveness during severe haemorrhage. However, the interest of these indices would be limited if they could only detect severe hypovolaemia. Originally, the guiding principle of these variables was to detect hypovolaemia very early.7–8 If DeltaPP/DeltaPOP can only detect a more than 20% decrease in estimated blood volume in children, then their usefulness would be similar to that of central venous pressure or clinical signs such as oliguria, tachycardia, or hypotension. We strongly feel that clinicians definitely need earlier indicators of hypovolaemia. The main point of our study is that in standardized conditions, dynamic parameters of fluid responsiveness based on arterial pressure and on plethysmographic waveform analysis fail to predict fluid responsiveness in children. However, respiratory variation in stroke volume (assessed using respiratory variations in the peak aortic flow velocity obtained with transthoracic echocardiography) is a strong predictor of fluid responsiveness in this setting. If the concepts of haemodynamic optimization based on these variables can be applied to the paediatric population, we believe that variables derived from stroke volume variation analysis would be more appropriate than pulse pressure or plethysmographic waveform variations. Recently, data obtained with PVI in mechanically ventilated children undergoing cardiac surgery found surprisingly good results regarding the ability of PVI to predict fluid responsiveness in this very challenging setting.9

Most patients are neonates, some with very complicated physiology, where cardiopulmonary interactions are extremely specific and may not be reflective of preload dependence. This clearly emphasizes that when it comes to the accuracy of dynamic predictors of fluid responsiveness in children, the only thing that we know is that we know nothing. Consequently, as suggested by Stricker and colleagues, further studies are required in this specific and challenging setting.

Conflict of interest

M.C. is a consultant for Masimo Corp. and Edwards Lifesciences.

E. P. Souza Neto1,2,*
M. Cannesson3
1São Paulo, Brazil
2Lyon, France
3Irvine, USA
*E-mail: edmundo.pereira-de-souza@hotmail.fr

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