to assist physicians in optimizing acute pain management in young children.

Declaration of interest

None declared.

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Use of analgesia monitors to optimize the management of immediate postoperative pain

Reply from the authors

Editor—We read with great interest the letter by Ly-Liu and Reinoso-Barbero in response to our publication.[1]

Similar to what we previously observed in adult patients using Analgesia/Nociception Index (ANI), the authors observed a significant correlation between the Pupillary Pain Index (PPI) and a behavioural paediatric pain scale (LLANTO scale) in children undergoing general anaesthesia using sevoflurane and remifentanil. This relationship was not observed with the PPI and Verbal Analogue Scale (VAS) pain scores, mostly because the VAS may be influenced by many factors other than pain in awake children, but also because young children may not use the VAS adequately.

Monitoring analgesia is a new and very challenging concept and developing tools for the prediction of immediate postoperative pain may have an important impact in clinical practice. Indeed, it as been shown in a recent study that severe pain still occurs in 20–40% of patients, including patients undergoing so-called minor surgical procedures (appendectomy, tonsillectomy, etc.).[2] In this perspective, the use of analgesia monitors such as the ANI or PPI may provide useful information to physicians to optimize the management of immediate postoperative pain. It is postulated that ANI or PPI values immediately before extubation may be highly predictive of acute pain within the following minutes, thus the administration of a prophylactic dose of opioid may provide a reduction in pain scores at arrival in the post-anaesthetic care unit. This, however, remains to be demonstrated in prospective studies.

For us, use of the ANI presents an advantage over the PPI since it allows for continuous analgesia monitoring directly from the patient monitor, whereas the PPI requires measuring the changes in pupil dilation in response to increasing electric stimulations. Moreover, in cephalic procedures such as ear–nose–throat surgery, the use of a device placed over the face is not possible.

Nevertheless, the authors should be commended for performing this study in young children in an effort to optimize postoperative analgesia. Prospective studies comparing various monitors such as the ANI and PPI are urgently needed in both adults and children to determine the clinical benefit of analgesia monitoring in routine practice for acute postoperative pain management.

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Predictability of stroke volume variation

Editor—I read with great interest the study of Guinot and colleagues[1] regarding predictability of the respiratory variation of stroke volume, which showed that monitoring stroke volume may provide more reliable information than cardiac output concerning the effect of fluid infusion. Surely, this study would help us to guide fluid management, particularly in the subset of patients who have a significantly higher baseline heart rate, for example, patients in hypovolaemic shock? However, the authors have failed to mention whether they took into consideration the ventilatory and other confounding factors affecting stroke volume variation (SVV) in their study population.

Studies have shown that respiratory variations in stroke volume and its derivatives are affected by respiratory rate,

resulting in limited ability to predict the response to fluids at high respiratory rate.[2] It has also been shown that pulse pressure variation (PPV), and hence SVV, cannot predict fluid responsiveness reliably in patients who either trigger the ventilator or breathe spontaneously.[3] Indeed, it has been proposed that PPV (and other indices of ventilation-induced SVV) may not apply to patients breathing spontaneously.[3]

The SVV is also affected by both the depth and the pattern of respiratory effort during mechanical ventilation. Thus, low tidal volume or the imposition of variable respiratory effort often results in false-negative PPV and SVV values.[4] For accuracy, reproducibility, and consistency in SVV measurements, it is recommended that the tidal volume should be between 8 and 10 ml kg^{-1} ideal body weight before and after a fluid challenge.[5]

Lastly, all of these techniques for measuring SVV assume a fixed heart rate; therefore, in the setting of arrhythmias, such as atrial fibrillation or frequent premature ventricular contractions, SVV measurements become inaccurate.[4]

Unfortunately, no information is provided by Guinot and colleagues[1] on whether the aforementioned influences were taken into account when conducting their study on SVV for functional preload monitoring.

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Monitor stroke volume and heart rate

Reply from the authors

Editor—We thank Dr Rajesh K. Dudeja for his critical comment on our article.[1] We agree with its reflections.

Dynamic preload indices are affected by several factors that may limit their interpretations and bedside use. Of these factors, those influencing interactions between the respiratory and circulatory systems are essential. Thus studies evaluating preload indices must be interpreted in the light of their clinical conditions: study population (operating room, critically ill patients, lung compliance), inclusion criteria, exclusion criteria, and of course ventilation management (tidal volume, respiratory rate, end expiratory positive pressure).

All patients included in our study received neuromuscular blockade and were ventilated with a tidal volume set to 7–9 ml kg^{-1} of ideal body weight, an end expiratory pressure of 5 cm H_2O, and a respiratory rate adjusted to maintain end tidal CO_2 at 30–35 mm Hg. Patients with arrhythmia, known right ventricular dysfunction, frequent ectopic beats, or breathing spontaneously were excluded. The overall mean tidal volume was 8.3 ml kg^{-1} (SD 0.8) and did not differ between the three groups of patients [8.3 (SD 0.8) *vs* 8.4 (0.7) *vs* 8.2 (0.7), $P=0.62$]. The mean respiratory rate was 13 (SD 2) min^{-1} with a heart rate:respiratory rate ratio of 5.3 (SD 1.3). This ratio did not differ between the three groups of patients [5.2 (SD 1.4) *vs* 5.3 (1.2) *vs* 5.8 (1.4), $P=0.29$]. In summary, we can assume that the groups were comparable and that variation of the predictability was associated with changes in stroke volume (SV), heart rate (HR), and cardiac output (CO).

We would like to improve the understanding of the concept of fluid responders. Our study was more a thought on the evolution of SV and HR with fluid expansion (that may change the final evolution of CO) than a study on the respiratory-derived fluid responsiveness indicator itself. We believe that fluid expansion must be interpreted in regard to the evolution of SV and HR rather than CO, based the concept of fluid responsiveness from Starling's law of the heart, which is itself based on SV.[2] According to this principle, myocardial muscular contraction is dependent on myocardial preload.[3 4]

These experiments were made on an 'isolated heart beating at constant rhythm, and well supplied with blood',[2 3] which may not reflect *in vivo* conditions. CO is a complex controlled parameter dependent on several factors.[4] CO delivers blood flow to body tissues. Also, CO control cannot be separated from blood pressure control that maintains blood pressure at a sufficiently high level to ensure blood supply to all tissues. These two variables are interdependent and are regulated by nervous, local, and hormonal control.[5 6] Moreover, anaesthetic drugs can alter homeostasis of these controls.[7 8] Variations in preload can alter CO via effects on SV and/or HR. Nixon and colleagues[9] demonstrated that variations in preload produce effects on SV corresponding to the normal ascending part of the Franck–Starling curve. Moreover, variations in preload were associated with variations in HR: HR decreased, whereas SV increased during a head down tilt test. Vatner and colleagues,[10 11] studying dogs, demonstrated that an increase in CO in response to an elevation of preload depends on the state of consciousness and baseline HR values. In their experiments, these authors demonstrated an increase of CO via various mechanisms: CO could increase as a result of increased HR or increased SV.[10 11] Finally, these studies confirmed that CO optimization is complex and can be altered by consciousness, drugs, or surgical trauma.