effects in different individuals. The 40–50 window is classically considered as a well-defined moderate hypnotic state, where synchronized–fast–slow activity of the EEG is the main determinant of BIS calculation. It is important to mention that, although using the model of Marsh, we were waiting for the equilibration between plasma and effect–site concentration of propofol before considering BIS and SE recording at steady state. This might not have appeared clearly enough in the method section of our manuscript. We can therefore reasonably consider our steady state as a real one. Noteworthy, when looking at the results of the Bland–Altman analysis presented in Table 2 of our paper, it appears that the mean difference between BIS and SE at steady state is not significantly different from 0 either in paralysed and non-paralysed patients, and that the limits of agreement are in the range of 20 U. This is in agreement with the results of Iannuzzi.2

Second, although the Xp version of the BIS monitor has been designed to better eliminate EMG artifacts, the possibility of EMG contamination during BIS calculation cannot be excluded. Vivien and colleagues4 have demonstrated that overestimation of BIS in sedated intensive care unit patients may be revealed by the administration of neuromuscular blocking agents. Although neuromuscular blocking agents may deepen anaesthetic depth through the limitation of muscle-emerged ascending inputs to the brain, EMG contamination is still possible.

Third, in our opinion, laryngoscopy can be considered as a relatively standardized nociceptive stimulus, provided it is performed during a predefined constant length of time and always by the same investigator. Laryngoscopy, algometry and tetanic stimulation are the three most frequently used standardized stimuli in pharmacodynamic studies. Of course, we do not recommend performing systematically a test laryngoscopy to assess the nociceptive–anti-nociceptive balance in patients. The aim in our study was to elicit a frank response of BIS and SE to a standardized nociceptive stimulation, and compare both responses. Tracheal intubation was not performed at that time because it would have introduced too much variability in terms of intensity and duration of the nociceptive stimulation. A 20 s duration seemed to be a good compromise between the need of a significant nociceptive stimulus and the risk of patients experiencing an unpleasant event. We found that, in non-paralysed patients, the limits of agreement between BIS and SE are large in those circumstances. This might not have been the case if opioids had been administered, but the response evoked by the nociceptive stimulation would probably have been truncated. Patients were systematically interviewed during the postoperative period and none of them reported any episode of unpleasantness. We acknowledge that this ethical issue is of importance, and Iannuzzi was right when mentioning it.

Finally, the definition of acceptable limits of agreement between two methods of measurement is empirical and debatable. It must be based on the magnitude of the variable scales and on the clinical relevance of the chosen interval. In our opinion, as BIS and SE scales are close to 100 U large limits of agreement of plus or minus 10 sound reasonable. We have seen that BIS and SE differ more than that in several circumstances.

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Perioperative fluid therapy in children

Editor—Cunliffe and Potter’s1 editorial raises important questions about the prescription of i.v. fluids to the perioperative paediatric population. The survey by Way and colleagues2 confirms the lack of guidelines for fluid prescribing, and the potential risk of hyponatraemia in this group of patients. We would like to share the results of our recent work on this topic.

The paediatric intensive care unit at the Royal Children’s Hospital in Brisbane, Australia admits 600 patients a year including approximately 30 children who undergo spinal instrumentation surgery. In 2003, a clinical pathway was introduced to standardize the care of postoperative paediatric patients undergoing spinal instrumentation. In July 2004 the standard i.v. fluid regimen was changed from dextrose 3.0% and sodium chloride 0.3% (Cohort 1) at two-thirds ‘maintenance’ rate to dextrose 5% and Hartmann’s solution (Cohort 2) at full ‘maintenance’ rate. The hourly full maintenance rate was defined as 4 ml kg–1 h–1 for the first 10 kg; 2 ml kg–1 for the next 5 kg, and 1 ml kg–1 for each kilogram thereafter.3 All other aspects of the postoperative clinical care remained the same as per the clinical pathway. The administration of postoperative fluid boluses was at the discretion of the treating doctor.

We conducted a retrospective study to compare the incidence of postoperative hyponatraemia in the two cohorts of children undergoing spinal instrumentation surgery who had...
Correspondence

Table 1  Patients. NS, not significant

<table>
<thead>
<tr>
<th></th>
<th>3% Dextrose/1/3 NaCl (n=30)</th>
<th>5% Dextrose/Hartmann’s (n=29)</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Number of males (%)</td>
<td>15.0 (50)</td>
<td>9 (31)</td>
<td>NS</td>
</tr>
<tr>
<td>Mean weight (kg)</td>
<td>33.3</td>
<td>39.6</td>
<td>NS</td>
</tr>
<tr>
<td>Mean age (yr)</td>
<td>11.4</td>
<td>11.8</td>
<td>NS</td>
</tr>
<tr>
<td>Mean no. of segments instrumented</td>
<td>8.8</td>
<td>9.3</td>
<td>NS</td>
</tr>
</tbody>
</table>

Table 2  Clinical and laboratory results. *Mean (sd). SD, standard deviation. NS, not significant

<table>
<thead>
<tr>
<th></th>
<th>3% Dextrose/1/3 NaCl (n=30)</th>
<th>5% Dextrose/Hartmann’s (n=29)</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Initial serum Na (mmol litre $^{-1}$)*</td>
<td>140.7 (2.4)</td>
<td>140.1 (2.5)</td>
<td>NS</td>
</tr>
<tr>
<td>Serum Na at 12–16 h (mmol litre $^{-1}$)*</td>
<td>135.5 (2.5)</td>
<td>137.6 (2.8)</td>
<td>NS</td>
</tr>
<tr>
<td>Fall in serum Na (mmol litre $^{-1}$)</td>
<td>5.2</td>
<td>2.2</td>
<td>0.00</td>
</tr>
<tr>
<td>Follow-up serum Na &lt;135 mmol litre $^{-1}$; n (%)</td>
<td>11 (37)</td>
<td>5 (17)</td>
<td>0.08</td>
</tr>
<tr>
<td>Patients receiving additional fluid boluses; n (%)</td>
<td>18 (60)</td>
<td>19 (66)</td>
<td>NS</td>
</tr>
<tr>
<td>Mean number of additional boluses given</td>
<td>5.2</td>
<td>4.0</td>
<td>NS</td>
</tr>
<tr>
<td>Mean total fluid bolus volume (ml kg $^{-1}$)</td>
<td>24</td>
<td>17</td>
<td>NS</td>
</tr>
</tbody>
</table>

received the two different i.v. fluid regimens. The two groups were equivalent for age, gender, underlying diagnosis, operative procedure and amount of bolus fluid received (Table 1). The main results are shown in Table 2.

We conclude that the change in postoperative fluid regimen from dextrose 3% and sodium chloride 0.3% at two-thirds maintenance rate to dextrose 5% and Hartmann’s at full maintenance rate reduced the proportion of patients with postoperative hyponatraemia and the fall in serum sodium at 12–16 h after operation. However, in our 2 yr study there were no patients in either cohort with clinically significant hyponatraemia. We are not aware of good quality clinical trials to guide the management of paediatric perioperative fluid therapy. We are currently enrolling patients in a randomized control trial to further investigate perioperative fluid management in children.

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Coexisting Harlequin and Horner’s syndromes

Editor—We would like to correspond further with regard to the article entitled ‘Coexisting Harlequin and Horner’s syndromes after high thoracic paravertebral anaesthesia’.1,2 A combined technique of general anaesthesia and a thoracic paravertebral block was performed. In this case a well-demarcated contralateral hemifacial flushing and ipsilateral pallor developed, without the distinctive ipsilateral Horner’s syndrome.

After induction of general anaesthesia, a left paravertebral block was performed at T3/T4 using a 20G spinal needle and loss of resistance to saline technique. A total of 40 ml of local anaesthetic, bupivacaine 0.25% with 1 in 200 000 adrenaline, was injected into the paravertebral space. The operation lasted approximately 4 h and entailed patient repositioning from right lateral to supine. Cardiovascular stability was maintained throughout and the operation was completed without incident.

In the recovery room the patient was noted to have a marked hemifacial flushing of the right side (contralateral to the block) and marked pallor on the left side, in the absence of Horner’s syndrome. The colour change persisted for approximately 5 h after operation and resolved without consequence.

Perioperative Harlequin syndrome is caused by sympathetic block of the thermal and emotional flushing response on the pale side, with normal or excessive flushing on the contralateral side.2 This case demonstrates that Harlequin syndrome can occur without Horner’s syndrome after high volume paravertebral block (denoting T2/T3 sympathetic interruption with sparing of T1 oculomotor branches). Contrary to a previous report of harlequin syndrome without oculomotor signs following a T10/11 thoracic epidural, intraoperative positioning was not a factor.

Given that thoracic paravertebral injection of bupivacaine 0.5%, 15 ml causes somatic and sympathetic block over 5–8 dermatomes,3 it is perhaps surprising that Harlequin syndrome, is not a common occurrence. Only three perioperative adult cases following local anaesthetic administration have been described.1,4 This would imply that it is not dependent on a normal or excessive contralateral thermal and emotional flushing response3 but that this preserved response must be excessive and rare. Perioperative, local anaesthetic-induced Harlequin syndrome therefore may be different in mechanism to others described in the literature.5

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