Correspondence

Table 1 Patients. NS, not significant

<table>
<thead>
<tr>
<th></th>
<th>3% Dextrose/1/3 NaCl (n=30)</th>
<th>5% Dextrose/1/3 NaCl (n=30)</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/1/3 NaCl (n=30)</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.

M. G. Coulthard*
L. S. Cheater
D. A. Long
Brisbane, Australia
*E-mail: Mark_Coulthard@health.qld.gov.au

References

1 Cunliffe M, Potter F. Four and a fifth and all that. Br J Anaesth 2006; 97: 274–7

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 ² ⁴ This would imply that it is not dependent on a normal or excessive contralateral thermal and emotional flushing response⁵ 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

A. Majumder*
P. Farquhar-Smith
London, UK
*E-mail: anjalina@mac.com

doi:10.1093/bja/ael297
Editor—We would like to thank Drs Majumder and Farquhar-Smith for contributing with a further case report to the available literature regarding the perioperative Harlequin syndrome, and also for their valuable comments regarding the mechanism underlying this syndrome. In regard to the prevalence of Harlequin syndrome in patients exposed to regional anaesthesia in the vicinity of thoracic and cervical sympathetic system, there are few reported encounters, indeed. At the date of our report publication, there was only one other published case of Harlequin appearance in adults after an internal jugular central venous cannulation. Other cases were recently described, all in patients where migration of local anaesthetic at the level of preganglionic sympathetic fibres originating from T2 to T4 spinal segments was highly likely. Ever since we published our first case report, we encountered a further similar postoperative Harlequin syndrome in a 48-yr-old ASA I female patient after high thoracic (T3) unilateral continuous paravertebral analgesia for breast reconstruction surgery. She presented contralateral hemifacial flushing and sweating well-demarcated in the midline without the distinctive ipsilateral Horner syndrome. We were also anecdotally told of at least two other similar presentations after central venous line insertion. In all cases the hemifacial flushing and sweating was transient and not complicated. The question that arises is whether the prevalence of Harlequin syndrome after high thoracic regional anaesthesia is low or the conditions that trigger the presentation, such as heat, emotion, gustatory stimuli or exercise, are not always met, especially in the immediate perioperative period.

We cannot exclude that some patients are responding with disproportionate vasodilatation and sweating of the contralateral hemiface in response to stimuli such as heat or emotion. Experimental studies in rabbit models have described a closer proximity of endothelial beta- than alpha-adrenoceptors in relation to the sympathetic nerve terminals to the face. In vitro research on human facial veins has demonstrated a large inter-individual variability in the relative density and sensitivity of alpha- and beta-adrenoceptors. It is possible therefore that, in some patients, the adrenergic receptors abundance and disposition may facilitate excessive beta-adrenergic vasodilatation in response to environmental stimuli.

As Richardson and Cheema recently affirmed, there is more fascinating matter to be learnt about the interaction between the paravertebral block and the afferent sympathetic supply to the face.

C. L. Burlacu*
D. J. Buggy
Dublin, Ireland
*E-mail: crina@ireland.com

References
1 Crawley SM. Coexisting Harlequin and Horner syndromes after high thoracic paravertebral block. Br J Anaesth 2006; 96: 537–8
6 Coleman P, Goddard JM. Harlequin syndrome following internal jugular vein catheterisation in an adult under general anaesthetic. Anaesthesiology 2002; 97: 1041
doi:10.1093/bja/ael301

Role of dantrolene in treatment of heat stroke associated with Ecstasy ingestion

Editor—In their recent review Hall and Hendry advocate the use of dantrolene in the management of hyperthermia in acute MDMA (3,4-methylene-dioxy-meth-amphetamine, ‘Ecstasy’) toxicity. MDMA and other amphetamine analogues may present with the syndrome identical to exercise induced heatstroke, a multisystem disorder which has been reviewed by Bouchama and Knochel. One of these authors also carried out a randomized controlled trial of the use of dantrolene (2 mg kg–1) in heat stroke and found no difference between the treatment and placebo group in terms of cooling time, complications, or length of stay. A recent meta analysis concluded that there is no place for the use of dantrolene in heat stroke. In addition, each 20 mg vial of dantrolene contains 3 g of mannitol and is reconstituted in solution of pH 9.5 which alkalizes the urine, thereby reducing the clearance of amphetamine like drugs such as MDMA.

We would recommend that heatstroke of whatever aetiology is treated as a multisystem disorder resembling severe sepsis with appropriate invasive monitoring and organ support including cooling, but not dantrolene. The danger of insidious late development of organ impairment or failure sometime after admission must not be overlooked and will not be prevented by early administration of dantrolene.

M. R. Duffy*
C. Ferguson
Plymouth, UK
*E-mail: mike_r_duffy@hotmail.com