Intrathecal drug spread

Editor—I read with interest the review by Hocking and Wildsmith\(^1\) on intrathecal drug spread. I feel they omitted to mention an important aspect of drug spread that could have a bearing for all blocks.

They quite rightly stated that vasoconstrictors added to intrathecal local anaesthetic could prolong the duration of a block, although not affect the height of block. They failed to mention the effect of i.v. vasopressors on spinal block height. An article by Cooper and Mowbray\(^2\) in 2003 first mentioned the effect of choice of i.v. vasopressor on the rostral spread of spinal anaesthetic. He conducted a formal study that was published in 2004,\(^3\) which showed that when using phenylephrine as the hypotensive rescue drug, the block height, when assessed to cold and light touch sensation, was on average two dermatomes lower compared with ephedrine.

Given the increasing use of phenylephrine; particularly in obstetric anaesthesia, to treat sympathetic blockade related hypotension, it should certainly have been mentioned.

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Editor—In their review of intrathecal drug spread, Hocking and Wildsmith state that pulse and blood pressure are related to block height.\(^4\) This relationship however is not precise, as there are other more significant influences.\(^4\)

Although later, the authors stress the importance of venous return for the maintenance of blood pressure, they omitted the clinically highly significant relationship between a dropping venous return to the right heart and bradycardia.\(^4\)

I accept that the cardiovascular responses to spinal blockade formed only a small incidental part of this review. Unfortunately, however, this short sentence relating pulse and blood pressure solely to cephalad spread of spinal anaesthesia could encourage the anaesthetist erroneously to attribute a slowing heart exclusively to the height of the spinal block, so inducing a false sense of security.

In 1988, Caplan and colleagues\(^5\) demonstrated the serious consequences of failing adequately to appreciate the significance of bradycardia during spinal anaesthesia. This warning sign must alert the anaesthetist to the possibility and hence the consequences of a dropping venous return, and cannot be passed off as a side-effect of cephalad spread of spinal anaesthesia.

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Editor—We thank Drs Akerman and Hutter for their interest in our review, and are grateful for the opportunity to respond.

Dr Akerman raises an interesting point, but should recognize the various dynamics involved. These are both relevant and important. First, the definitive paper by Cooper and colleagues\(^1\) did not appear until our review was well advanced in the publication process. Second, Cooper and colleagues\(^1\) only studied their patients for 20 min after intrathecal injection, and it is well recognized that intrathecal spread will continue for at least 30 min. Thus, the third issue, that this was the first description of a previously unreported association, was made in a dynamic clinical situation. Before an influence of systemic vasopressor on intrathecal drug disposition can be accepted as definitive it will need confirmation, and preferably confirmation in patients whose blocks are followed for at least 30 min, if not longer. Type 2 error is not just a theoretical possibility; it happens.

Dr Hutter is correct in stating that extent of block is not the only factor that may contribute to perioperative bradycardia, but it is a major factor and we stand by our wording, particularly given the subject matter of this paper. The other issues have been discussed elsewhere,\(^6\) including some thoughts on the series from Caplan and colleagues.\(^5\)

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2 Cooper DW, Mowbray P. Can choice of vasopressor therapy affect the rostral spread of spinal anaesthetic? Anesthesiology 2003; 98: 1524
3 Cooper DW, Jeyeraj L, Hynd R, et al. Evidence that intravenous vasopressors can affect rostral spread of spinal anaesthesia in pregnancy. Anesthesiology 2004; 101: 28–33
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Leucocyte depleted whole blood in elective surgery programs

Editor—The preparation of autologous blood components is regulated by rules that are derived from the production of homologous blood components. Whole blood units (WB) are stored and transfused as leucocyte- and plasma-depleted red blood cell concentrates (PRBCs) in order to minimize adverse reactions in the transfused patient. Adverse reactions caused by the plasma fraction are mainly a problem in the homologous setting. We found that leucocyte-depleted whole blood units (LD WB) stored for 35 days meet the quality requirements for transfusion of the European Council\(^1\) and is a feasible option, in addition to PRBCs, for autologous transfusion, especially in hospitals without a blood bank.

Standard units (450 ml) of WB were collected from 12 healthy male donors (age range 28–59 yr) using bags with an integrated

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Table 1 Metabolic parameters, increase of haemolysis, and ATP content in CPDA-1 preserved whole blood. Values are expressed as mean (SD).

<table>
<thead>
<tr>
<th>Days</th>
<th>0</th>
<th>21</th>
<th>35</th>
</tr>
</thead>
<tbody>
<tr>
<td>K⁺ (mmol litre⁻¹)</td>
<td>3.8 (1.2)</td>
<td>19.9 (2.5)</td>
<td>24.3 (2.6)</td>
</tr>
<tr>
<td>LDH (U litre⁻¹)</td>
<td>166 (60)</td>
<td>175 (56)</td>
<td>182 (67)</td>
</tr>
<tr>
<td>fHb (mg dl⁻¹)</td>
<td>15.7 (19.2)</td>
<td>27.8 (22.4)</td>
<td>30.3 (22.5)</td>
</tr>
<tr>
<td>Hemolysis (%)</td>
<td>0.07 (0.09)</td>
<td>0.13 (0.1)</td>
<td>0.15 (0.11)</td>
</tr>
<tr>
<td>ATP (pmol 10⁶ erythrocytes⁻¹)</td>
<td>112 (27)</td>
<td>114 (28)</td>
<td>73 (17)</td>
</tr>
</tbody>
</table>

filter system (MacoPharma, Toucoing, France), containing 70 ml CPDA-1 as anticoagulant/preservative solution. All units were kept at room temperature for at least 4 h to ensure bacterial phagocytosis and were then leucocyte depleted by inline filtration (Lucile, MacoPharma, Toucoing, France). Storage erosion was evaluated by the increase of K⁺, LDH, free haemoglobin (fHb), haemolysis rate, and the course of intracellular ATP content over the storage period of 35 days as described previously.²

As required by the European Council, leucocyte contamination was less than 1×10⁶ cells per unit, determined by flow cytometry (FACScan, Becton-Dickinson, San Jose, CA, USA) using the Leuco-Count kit (Becton-Dickinson) and the median Hct was 38.8% (range 34–46.9%). Haemolysis, reflected by the increase of K⁺, LDH and fHb is limited to 0.8% at the end of shelf life.¹

In LD WB units a haemolysis of 0.15%±0.11 was measured on day 35 that is clearly below the threshold. Values for K⁺, LDH and fHb were within the ranges observed in PRBCs (Table 1).² The mean (SD) intracellular content of ATP rose within 14 days from 112 (27) pmol 10⁶ erythrocytes⁻¹ to 146 (44) pmol 10⁶ erythrocytes⁻¹ and decreased thereafter. At the end of shelf life, 67 (20)% of the initial value was found. As described previously, the 24 h recovery of transfused PRBCs correlates strongly with red cell quality³ and is significantly affected when ATP concentrations fall below 10% of initial values.⁴

Erythrocytes stored as LD WB fulfil the requirements for transfusion warranted by the European Guidelines and the in vitro assessed adequate quality of erythrocytes allows the assumption that the 24 h post-transfusion recovery will not be impaired by the storage of erythrocytes as LD WB. Thus storage and transfusion of LD WB is an option in the autologous setting.

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Epiduroscopy for lumbar spinal stenosis

Editor—Igarashi and colleagues have presented their series of epiduroscopies in a most interesting way,¹ and Richardson has written a highly supportive editorial.² They have focused on the visual findings as well as outcome, and have not made comparison with other treatments, apart from epidural steroid injection. Their paper raises a number of questions.

Patients with radicular (monosegmental) pain had a greater amount of fatty tissue and vascularity within the epidural space than those with more widespread (multisegmental) pain. We are told that both groups received epidural blocks 2–14 weeks before epiduroscopy with local anaesthetic, either with or without steroid. The number of epidural blocks was not specified. Diagnosis of spinal stenosis was made either by computed tomography with myelography or by magnetic resonance imaging. Can the authors ascertain that a previous epidural or epidurals at various time points, whether with or without steroid, would not affect the amount of vascularity? Epidural steroid injection is often reserved for those primarily with radicular pain and this group may have had a greater number of epidurals, thus affecting the epiduroscopy findings.

Using failure of response to epidural steroid injection as a ratification of efficacy of epiduroscopically applied steroid can only be made with a full account of the approach used for epidural steroid injection before epiduroscopy. It is known that up to 36% of caudal injections are inaccurately positioned when checked with fluoroscopy, and 6–9% of lumbar injections are similarly inaccurately positioned.³ ⁴

It has already been documented that ‘targeted’ steroid injection for sciatic radicular pain can produce a good response. Lutz⁵ showed that transforaminal epidural steroid injection produced greater than 50% relief of pain in 75% of patients for an average of 80 (28–144) weeks, and 78% were satisfied with the final outcome. These results appear to compare very favourably with those of the authors.¹ Kraemer has shown similar results with perineural and epidural steroids.⁶ My own experience with epiduroscopy suggests that those with problematic fibrosis around the nerve root were unlikely to respond to targeted steroid infiltration. Problematic fibrotic ‘scar’ tissue is of such density that little can at present be offered through the epiduroscope, and this paper does not convince me that epiduroscopy as a means of applying steroid to nerve roots has advantages over simpler methods. It may have advantages once further epiduroscopic interventions are possible, and as a diagnostic aid, but first we need to understand what we are seeing. The authors clearly are trying to advance this knowledge.

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Editor—We would like to thank Dr Nash for his letter regarding our article. Dr Nash’s comments raise several questions: (i) influence of the previous epidural block on the epidural structure; (ii) therapeutic outcome compared with other treatments; and (iii) possibility of incorrect placement of epidural injectates owing to heavy fibrosis around the involved nerve roots.

First, as it was pointed out by Dr Nash, we also recognize that previous epidural blocks might affect epiduroscopic findings, as previously published literature suggests that previous epidural blocks may increase the amount of fibrosis or the degree of adhesion of epidural space.⁷ ⁸ In this study, the mean numbers of epidural blocks before epiduroscopy were 4.9 and