REFERENCES


In Reply—We disagree with Dr. Day. He puts forward two suggestions that could explain a positive response to the spinal block in central pain: systemic absorption of lidocaine and the role of the sympathetic component in the pain syndrome. Systemic absorption of lidocaine could not have been a mechanism of the pain relief in our cases because despite complete pain relief in the leg, there was no change in pain intensity in the arm after the lidocaine injection at the L2–L3 level. A sympathetic component in the mechanisms of pain also could not be a factor in our cases because we started the block procedure with an injection of 0.5% lidocaine, which caused an increase in skin temperature but no change in the pain intensity. A negative response (no pain relief) to the block is not a very useful sign in the central pain either, because even 2 ml 2% lidocaine may not block all sensory functions in the area, and those not blocked can be responsible for pain maintenance. The most important point regarding the role of the blocks in chronic pain diagnosis is that it is based on an assumption that the block distal to a lesion causing the pain cannot provide pain relief. Table 1 indicates that this may be an incorrect assumption.

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REFERENCES


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Table 1. Pain Relief Following Local Anesthetic Block Distal to a Lesion

<table>
<thead>
<tr>
<th>Authors</th>
<th>Diagnosis</th>
<th>Site of Injury</th>
<th>Site of Local Anesthetic Block</th>
<th>Result</th>
</tr>
</thead>
<tbody>
<tr>
<td>Kibler and Nathan</td>
<td>Central or radicular pain</td>
<td>Spinal roots or spinal cord</td>
<td>Peripheral nerves</td>
<td>Relief of spontaneous pain and paresthesia</td>
</tr>
<tr>
<td>Xavier et al.</td>
<td>Sciatica</td>
<td>Lumbar roots</td>
<td>Sciatic nerve or its branches</td>
<td>Relief of spontaneous pain</td>
</tr>
<tr>
<td>Kissin et al.</td>
<td>Sciatica</td>
<td>Lumbar roots</td>
<td>Sciatic nerve</td>
<td>Prevention of pain caused by nerve-root tension test</td>
</tr>
<tr>
<td>Xavier et al.</td>
<td>Sciatica</td>
<td>Lumbar roots</td>
<td>Sciatic nerve</td>
<td></td>
</tr>
<tr>
<td>Crisologo et al.</td>
<td>Central pain</td>
<td>Brain</td>
<td>Spinal cord</td>
<td>Relief of spontaneous pain</td>
</tr>
</tbody>
</table>

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Transfusion-induced Hyperkalemia

To the Editor:—Jameson et al.1 recently reported a case of fatal hyperkalemia secondary to massive transfusion. Though the patient undoubtedly received a large load of potassium, we question the calculated rates of infusion. Specifically, the "patient received up to 420 ml/min of blood (6.43 ml·kg⁻¹·min⁻¹), equivalent to 9.9 mEq/min of potassium (9.1 mEq·kg⁻¹·h⁻¹), just prior to cardiac arrest." But, since potassium values are those of plasma2 and since each unit of packed red blood cells contains about 70 ml of plasma,3 the calculated infusion rate of potassium is 2.3 mEq/min (2.1 mEq·kg⁻¹·h⁻¹) for this 5-min interval. Moreover, the patient is noted to have "received more than 2.0 mEq·kg⁻¹·h⁻¹ during the previous 5 h." Based on the 36 units of packed cells administered during this period (each with a plasma