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

Authors	Diagnosis	Site of Injury	Site of Local Anesthetic Block	Result
Kibler and Nathan[1]	Central or radicular pain	Spinal roots or spinal cord	Peripheral nerves	Relief of spontaneous pain and paresthesia
Xavier *et al.*[2] Kissin *et al.*[3]	Sciatica	Lumbar roots	Sciatic nerve or its branches	Relief of spontaneous pain
Xavier *et al.*[4]	Sciatica	Lumbar roots	Sciatic nerve	Prevention of pain caused by nerve-root tension test
Crisologo *et al.*[5]	Central pain	Brain	Spinal cord	Relief of spontaneous pain

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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 $ml \cdot kg^{-1} \cdot min^{-1}$), equivalent to 9.9 mEq/min of potassium (9.1 $mEq \cdot kg^{-1} \cdot h^{-1}$), just prior to cardiac arrest." But, since potassium values are those of plasma[2] 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 $mEq \cdot kg^{-1} \cdot h^{-1}$) for this 5-min interval. Moreover, the patient is noted to have "received more than 2.0 $mEq \cdot kg^{-1} \cdot h^{-1}$ during the previous 5 h." Based on the 36 units of packed cells administered during this period (each with a plasma

volume of 70 ml and a potassium level of 31.8 mEq/l—the higher of two measured prearrest values), the calculated potassium infusion rate is 0.25 $mEq \cdot kg^{-1} \cdot h^{-1}$.

In the studies of animals receiving potassium infusions to which the authors refer, ponies tolerated 1.78–1.99 $mEq \cdot kg^{-1} \cdot h^{-1}$ for slightly over 1 h on the average before developing terminal dysrhythmias,[4] and dogs received 2.0 $mEq \cdot kg^{-1} \cdot h^{-1}$ for 3 h prior to exhibiting "impending" ventricular fibrillation.[5,6] Also, Hiatt and Hiatt[5] found that 0.3 mM/kg rapid potassium injection produced ventricular fibrillation in dogs, and not 0.1 mM/kg, as noted in the case report.[1] Hence, we conclude that this patient's hyperkalemia resulted from a potassium load that was significantly less than stated in the case report and that was not given "in rates that far exceeded rates shown to cause cardiac toxicity in humans and animals."

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In Reply:—Hyperkalemic cardiac arrest occurs when the potassium (K^+) concentration gradient between interstitial fluid and the intercellular fluid of the myocardial cell decreases until cell membrane repolarization cannot occur. *In vivo* measurements of interstitial K^+ concentration during hyperkalemic arrest are not available. Thus, we used whole blood sample K^+ concentrations to reflect this gradient. Other studies of hyperkalemic arrest due to blood transfusions used blood infusion rates to reflect K^+ load.[1–4] The K^+ determined expresses milliequivalents per liter of fluid tested. University of Wisconsin Hospital uses ADSOL®-preserved packed red blood cells. The absolute volume of plasma in each unit is unknown. The absolute milliequivalent infusion can only be estimated. The K^+ in the packed red blood cell infusion significantly elevated the serum K^+ (measured). This resulted in increased interstitial fluid K^+ and cardiac arrest. The important point of the case report is that transfusion rates can exceed the ability of the body to equilibrate and normalize serum K^+. The result in this case was cardiac arrest and death. In addition, the higher the K^+ in these units, the greater the risk may be. Since I know of no other means to estimate the K^+ load during transfusion than transfusion rate and unit K^+ concentration, I continue to advocate vigilance when transfusion rates exceed 120 ml/min.[4]

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Antagonism of Vecuronium-induced Neuromuscular Block

To the Editor:—The study by Magorian *et al.*[1] "addressed the clinical problem of antagonizing a profound neuromuscular blockade" induced by vecuronium. The authors showed statistically that recovery time was the same regardless of whether neostigmine was given during deep