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(Accepted for publication March 13, 1991.)

Anesthesiology
74:1168, 1991

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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(Accepted for publication March 13, 1991.)

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 <i>et al.</i> ²	Sciatica	Lumbar roots	Sciatic nerve or its branches	Relief of spontaneous pain
Kissin <i>et al.</i> ³	Sciatica	Lumbar roots	Sciatic nerve	Prevention of pain caused by nerve-root tension test
Xavier <i>et al.</i> ⁴	Sciatica	Lumbar roots	Sciatic nerve	Relief of spontaneous pain
Crisologo <i>et al.</i> ⁵	Central pain	Brain	Spinal cord	Relief of spontaneous pain

Anesthesiology
74:1168-1169, 1991

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 po-

tassium 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