

and appears perfectly normal. This sequence of events is quite dissimilar from that of a total spinal, where apnea occurs in 2 min with dilation of the pupil and an unrecordable blood pressure; and where on recovery it takes 30 min from the first faint chin tug to the time when the jaw can be left unsupported."⁴ Leivers's patient regained consciousness 115 min following the last dose of lidocaine.

I agree with Leivers's suggestion that performance of a prophylactic epidural blood patch be postponed until the epidural anesthetic has resolved, for the patient is then able to report back pain if too much blood is injected too quickly. It is possible that the blood injection hastened or worsened the apparent subdural anesthetic. However, it is not clear that the blood injection caused the reported complication.

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In Reply:—I agree with Dr. Leighton that the possibility of subdural injection of local anesthetic in this case¹ had to be considered. The fact remains, however, that the sudden rapid increase in the level of a previously stable block occurred immediately following the blood injection. In addition, if the catheter had migrated into the subdural space, it is unlikely that the injection of 15 ml of blood would have been subsequently so benign. The respiration did not "slowly fail" but ceased abruptly. Massey Dawkins² gave only a general description of his "massive extradural," but it is possible that some of his 28 cases would now be considered to be examples of subdural injection. Sykes³ earlier had reported details of a case which coincide more with the typical subdural injection. In all instances the progress was more protracted than in my case.

Recently, another patient experienced a sudden increase in the level of a previously stable epidural block, when 10 ml of saline with 5 mg morphine was injected. This patient had also had an accidental dural puncture prior to the establishment of satisfactory analgesia *via* an epidural catheter. In similar circumstances, I suggest that the mecha-

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Value of Spinal Block in Central Pain

To the Editor:—The recent report by Crisologo *et al.*¹ concluded that the value of spinal block in making the diagnosis of central pain is questionable. This conclusion is not warranted, although the authors are correct to the extent that the interpretation of the effect of spinal blocks may be difficult. The use of spinal block to aid in the diagnosis of central of psychogenic pain is still useful in the absence of a positive response (relief of pain). However, a positive response including "cure" may still be entirely consistent with a central pain diagnosis. Whenever lidocaine (or any local anesthetic) is used, any conclusion made from a positive response must be made cautiously, since systemic absorption can cause pain relief in a wide variety of painful disorders.* In order

* Glazier S, Portenoy R: Systemic local anesthetics in pain control. *Journal of Pain and Symptom Management* 6:30–39, 1991

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nism postulated in my case report should be considered as a possible alternative explanation to subdural injection.

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to definitely conclude that the effects of the injection are due to the local effects of the block, one would need to compare the results to the results obtained after systemic injection (with equivalent serum lidocaine concentrations) without noticeable nerve block. Such a test is impossible to perform in a patient who remains improved after spinal block; in this instance the test is really a moot point.

In addition, the cases cited that did have a positive response could very well have had a peripherally mediated, sympathetic maintained pain syndrome since these have been described after strokes.²

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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 <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				Relief of spontaneous pain
Crisologo <i>et al.</i> ⁵	Central pain	Brain	Spinal cord	

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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 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