sion rates were increased by 10 mL/h and fluid was finally noted at the second hole with infusion at 800 mL/h and at the third hole at 900 mL/h. When we repeated this experiment using softer nonradiopaque catheters, the results were better but still problematic. Appearance at the second hole occurred when infusion rates were 200 mL/h and at the third hole when they reached 250 mL/h.

Based on the fact that at the conventional rate of epidural infusion (10 mL/h), infusate only reaches the proximal epidural catheter orifice, perhaps these multiorificed catheters should not be used for continuous infusion techniques. If top-ups continue to be a necessity even during continuous-infusion labor analgesia, why initiate an infusion at all?

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What Causes Different Pain Sensations on Intradermal Injection of Local Anesthetics?

To the Editor:

With great interest we read the article by Farley et al. (1) about differing pain sensations when infiltrating local anesthetics intradermally. The findings basically confirm our observations that were published in a similar controlled study (2). Since Farley and colleagues were obviously not aware of our results, we would like to add to their conclusions about the possible causes of this finding.

We became aware of this phenomenon when utilizing mepivacaine as an anesthetic from two competing producers. The finding caused us to request the data about the complete chemical compositions of the two solutions which were not shown on the package inserts. We noticed that the solution that created less pain on injection contained calcium chloride besides saline. Reviewing the pharmacology of calcium, we found that calcium by itself has local anesthetic effects (3–5). In regard to the active substance or the hidden chemicals of the tested drug solutions by Farley et al., it is evident that the ones producing the lesser pain sensations contain calcium also. Since the range of the pH of our own investigated solutions was fairly narrow, we think that the main factor for this phenomenon is more related to the calcium content that to pH differences.

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Spinal Vasculature Thrombosis as the Etiology of Spinal Cord Injury Following Epidural Phenol

To the Editor:

Katz et al. (1) in their recent article describing the histopathologic changes in primate spinal cord following epidural phenol injection concluded that phenol had a direct neurodestructive action on spinal cord and nerve roots. They failed to consider the probability that the neuronal damage resulted from vascular compromise by the phenol.

The blood supply to the spinal cord of the rhesus monkey (like that of most primates, including humans) is composed of the anterior and posterior spinal arteries which are derived from the radicular arteries and the artery of Adamkiewicz (2). These vessels pass through the intervertebral foramina and traverse the epidural space before reaching the spinal cord. Therefore, they can come in direct contact with the phenol following epidural injection. The review by Wood (3) indicated that phenol has a high affinity for blood vessels and may injure these arteries. This form of injury may be an important pathogenic factor contributing to the observed neuropathology. Patchy neurodestruction and Wallerian degeneration observed by Katz et al. can occur following

ischemia (4) and is not unique to direct neurotoxicity of phenol. Indeed, thrombosis of the spinal cord blood supply has been demonstrated as the etiology of neuraxial deterioration following subarachnoid phenol injection in humans (5), and narrowing or obliteration of arterioles and arteries has been documented following subarachnoid phenol in cats (6). Katz et al. did not report the histology of the vasculature in their study nor consider the effects of phenol on vasculature.

We also take issue with the statement that subarachnoid injection is more controllable than epidural. Smith (7) found that "the distribution of the degenerated fibers support the view that the phenol solution has not only passed rostrally and caudally from the site of the injection, but has run along the long axis of the rootlets, both centrally and peripherally." Katz et al. must supply documentation for their conclusion regarding the spread of subarachnoid and epidural phenol.

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Postdural Puncture Headache

To the Editor:

The recent article by Dr. Hatfalvi (1) and the editor's comments were of special interest to me. Since I began my residency in 1950, I believe I have administered 8,000 to 10,000 spinal anesthetics, so there have been many occasions for me to consider postdural puncture headache (PDPH) and its cause and treatments. In my residency at the Cleveland Clinic, we did many laminectomies in the sitting position, and I repeatedly saw the dura exposed, stripped away from the arachnoid, with the cerebrospinal fluid held intact until the arachnoid was incised. As a result of that experience, the arachnoid has been an important part of my thinking during a lumbar dural puncture, both as a factor in "failed" or "spotty" spinal anesthetics despite a so-called free flow of cerebrospinal fluid (was it the fluid from my syringe in a pocket between the dura and the arachnoid?) Dr. Hatfalvi has not mentioned the arachnoid mater nor has he included it in the discussion or illustrations. I believe it may participate in some way in the production of PDPH.

My experience has led me to believe that PDPH is more common with failed or spotty spinal anesthetics (a subdural epiarachnoid injection is the only way I can explain spotty spinals, anyway). Conversely, I believe that if we could study large numbers of PDPH cases, we would find a higher incidence of failed, inadequate, or reduced-duration spinal anesthetics. The radiology literature refers often to subdural epi- or extra arachnoid injections, whereas it is seldom mentioned in our own literature.

A recent case illustrates the importance of the arachnoid mater. A patient developed the classical signs of PDPH (which was relieved with a blood patch) after an epidural steroid injection. The epidurally injected steroid with a local anesthetic had produced no motor or sensory changes, leading me to believe that the dura was punctured, but the arachnoid was not.

Finally, it is with a certain sense of satisfaction that I point out that in 1983 I called attention to the inclination or bending of the needle during its insertion (2), as Drummond and Scott (3) and Dr. Hatvalvi, have also done.

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Postdural Puncture Headache: The Betadine Factor

To the Editor:

In 1988, we suggested that in addition to the leakage of cerebrospinal fluid (CSF), unintentional betadine contamination of the central nervous system (CNS) during induction of spinal anesthesia played an important role in the etiology of postdural puncture headache (PDPH)