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Larger groups might have yielded significant intergroup differences that were not evident in our study. For example, the differences in average intracranial pressure might have been significant with groups of 80 patients each; or differences in mannitol use might have appeared with groups of 120 patients each (which is highly unlikely, given the noted pattern of mannitol administration); or groups of 200 patients each might have revealed differences in the incidence of severe swelling. They go so far as to argue that "any negative result derived from fewer than 200 patients per group should not be considered for publication." Unfortunately, these authors overlook the possibility that a larger trial might also fail to show any differences, and they ignore the enormous financial and human resources that would be needed. It is a trivial matter to state that someone needs to do a bigger trial; it is not a trivial matter to undertake one. This raises the issue of whether it is worth engaging in the multimillion dollar, multicenter trial (funded by whom?) to answer their challenges. In one way, our study can be viewed as a pilot. At the conclusion of any pilot trial, the investigators must decide whether it would be worthwhile to continue with a larger study. We had many discussion on this issue, and we firmly decided that the data so far collected did not warrant the effort and cost that would be needed. If Harrung and Cotrell would like to undertake this large study, we would gladly participate and provide them with any insights we might have gained. In the meantime, we have chosen to pursue questions that we believe are far more important.

We carried out what we believe to be the largest prospective comparative trial yet dealing with the issue of anesthetic selection in neurosurgery. We presented the results in an exhaustive fashion (perhaps excessively so) to allow readers to draw their own conclusions, which may be different than our own. Because we realize the limitations of such a trial (including those engendered by its relatively small size), we were extremely cautious about interpretation. In the absence of any other evidence, we believe that our goal should be to disprove the null hypothesis. We could not do this in most cases and therefore concluded that there were no important differences among the three anesthetics. We also realize that our clinical impressions, as well as our data, probably played some role in these conclusions, but we stand by those conclusions. More importantly, we believe that the burden of proof now resides with those who suggest that one of the commonly used anesthetic agents is indeed "better or worse" for the brain than another.

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Anterior Spinal Artery Syndrome?

To the Editor—Parris and Kirchner recently reported an unfortunate case of long-lasting motor paralysis of the lower extremities after lumbar sympathetic block.1 In this case, lumbar sympathetic block at L2 was abandoned because of arterial blood flow from the spinal needle. It was reattempted at L3, but motor and sensory disturbances of both lower extremities occurred, preceded by lower back pain radiating down both legs immediately after the injection of local anesthetic. The authors speculated that arteriopasm resulting from inadvertently puncturing the anterior spinal artery or the artery of Adamkiewicz may have produced progressive ischemia to the anterior segment of the spinal cord, presenting as anterior spinal artery syndrome.

However, there are a few problems with that speculation. First, motor and sensory disturbances followed a series of events after the injection of local anesthetic, and not the puncture of the artery. It is more reasonable to speculate that motor and sensory disturbances resulted from the injection of local anesthetic. Second, in most reported cases of anterior spinal artery syndrome, pain in the region fed by these arteries did not precede the motor and sensory disturbances, as in this case.2,4

Parris and Kirchner did not describe how the position of the needle tip was ascertained. If radiologic aid had not been used, the needle tip might have been malpositioned. We think that it is necessary to consider another possibility: local anesthetic may have been injected into the spinal cord directly or through a peripheral nerve, which would have caused severe pain and spinal cord damage,5,6 resulting in the intraspinal lesion observed in this patient.

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In Reply:—As we indicated in our original presentation, our impressions were purely speculative with regard to the possible etiology of sustained motor paralysis after the lumbar sympathetic block in our patient. Nagaro and Ari suggest that direct spinal cord injury was a possible etiology. We believe that this was unlikely because our technique used a paramedian approach (at least 2 inches from the midline) and the needle position on injection of the local anesthetic was L3; this was well below the termination of the spinal cord. Our technique was performed with meticulous and sequential aspiration of the syringe before injection of 2-ml increments of 0.25% bupivacaine. This technique is specifically designed to monitor inadvertent intravascular injections. All aspiration tests were negative for blood, cerebrospinal fluid, or other body fluids. Further, none of the usual signs of local anesthetic toxicity (e.g., tinnitus, seizure activity, hypotension) was observed after 6 h of postblock monitoring.

We do not routinely use fluoroscopic techniques to confirm needle placement when performing lumbar sympathetic blocks except when neurolytic agents are being used. In our experience, we find this practice to be expensive, unnecessary, and time-consuming. Further, verification of the position of the needle tip using radiologic techniques does not guarantee that subsequent needle tip migration during the performance of a nerve block would not occur and result in neural damage. Thus, we reject the speculation of direct spinal cord trauma.

Nevertheless, we are intrigued by the other speculation of Nagaro and Ari, that the needle tip may have been entered a spinal nerve, from where the local anesthetic may have migrated to the spinal cord, producing the patient’s neurologic lesion. This suggestion is a novel one and does warrant some consideration. However, the progressive improvement of the neurologic lesions with time and therapy tends to suggest that spinal cord injury, direct or indirect, was unlikely. We would also like to point out that neurologic lesions secondary to spinal artery vasospasm and subsequent spinal ischemia are not infrequently preceded by pain.

Thus, although our impressions regarding the etiology of this lesion are still speculative, we do not believe that intravascular injection or spinal cord trauma were likely factors, producing the motor paralysis in our patient.

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