CORRESPONDENCE

In Reply.—I have no quarrel with Dr. Saidman's suggestions, particularly his offer of knighthood. And I would be pleased to collaborate with Dr. Ebert or others with a close, prolonged, and repeated connection with Abbott Laboratories.

However, as indicated in my response to Dr. Saidman's previous editorial, I believe that bias is not (or should not be) the issue. Regardless of our sources of support (commercial, National Institutes of Health, or other), we all come to the scientific enterprise with biases, with theories or points we would prove. We come as champions of a hypothesis (often a tiny hypothesis). We come with a passion that animates us. Pity the poor, independent analytical laboratory that cared little about the data other than as accurate results (and, of course, there is the question of whether an analytical laboratory has feelings). What a dull existence!

The problem I see with implementation of Dr. Saidman's suggestion is that it removes those who are most knowledgeable and interested from the tournament. The question of sevoflurane's potential to adversely affect the kidney would not likely have been addressed without the concern and effort of investigators such as Dr. Ebert and myself—and the support of Abbott Laboratories and Baxter Pharmaceutical Products. If there is a downside to the tournament, it is that it may diminish collegiality.

Each of us must strive to sustain the independence necessary for truly valid, important, and clinically relevant studies. Each of us has a responsibility to contain the potentially destructive influence of bias, while harnessing the energy and expertise of interested and committed investigators. In part, that responsibility lies in the mind of the reader who has been warned that I am a paid consultant to Baxter Pharmaceutical Products. In part, it lies in the hands of the investigator. In part, it becomes the responsibility of reviewers and editors of this and similar journals.

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References

1. Eger EI II. Motivation, bias, and scientific integrity. Anesthesiology 1994; 81:270–1
2. Saidman LJ. Unresolved issues relating to peer review, industry support of research, and conflict of interest. Anesthesiology 1994; 80:491–2

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Rate of Injection and Neurotoxicity of Spinal Lidocaine

To the Editor—I read with great interest the three recent articles relating to neurotoxicity after spinal lidocaine administration.1,2,4 The mechanisms proposed by the authors are plausible. I would, however, like to propose an additional factor predisposing to neurotoxicity: slow rate of injection.

Rate of injection was elegantly demonstrated by Rigler and Drasner4 to be a critical factor in the maldistribution of local anesthetic that was proposed to occur with 28-gauge microcatheters: the faster the injection, the more turbulent the flow of hyperbaric solution out of the catheter, and the more thorough the mixing. They also demonstrated that physicians voluntarily choose to inject slowly through a 25-gauge spinal needle, taking approximately 10 s to inject 1 ml (presumably out of fear that rapid injection will lead to a high block, although the reason is not stated). At this slow rate, the hyperbaric fluid was seen to layer out in the dependent portion of their spinal model.

All three of the recent articles about transient neurologic symptoms reported using either 25- or 27-gauge pencil-point needles. Liguori et al.4 reported injecting the 3-ml test solution over approximately 30 s, which is equal to the rate reported by Rigler and Drasner4; the other authors do not report speed of injection. I would like to ask Martinez-Bourio et al.2 and Hampl et al.3 whether comparable rates of injection were used in their respective studies.

I think we should reassess whether we need to inject slowly through the newer 25- and 27-gauge pencil-point needles. Is the risk of a high spinal lower with smaller needles? Does injection in the sitting position (vs. lateral) protect against a high spinal? Does slower injection lead to areas of highly concentrated local anesthetic? Are we seeing so many articles about neurotoxicity lately because we have made the switch to smaller needles?

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References


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In Reply.—We would like to thank Dr. Youngs for her interest in our article. Her comments highlight some important points regarding the etiology of transient neurologic symptoms (TNS). Intrathecal lidocaine has a long history of use without reports of TNS. Potential systematic changes that have occurred in the early 1990s may be responsible for the reports that appeared at this time. Several possibilities include the following: heightened awareness on the part of anesthesiologists to subtle clinical symptoms in their patients; early ambulation of patients who have been administered a spinal anesthetic; and, finally, as Dr. Youngs points out, the use of small-gauge, pencil-point needles with side ports.

We agree with Dr. Youngs comments that a slow injection of a hyperbaric solution may lead to pooling of the anesthetic and a maldistribution of local anesthetic concentration within the cerebrospinal fluid. However, in our study, we used isobaric solutions of meptivacaine and lidocaine. It is unclear whether the aforementioned mechanism for maldistribution applies to isobaric preparations. It is clear from the many studies looking at the incidence of TNS after spinal anesthesia with lidocaine that this syndrome occurs comparably when isobaric and hyperbaric solutions are used.

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In Reply.—We appreciate the interest in our article expressed by Dr. Youngs. Unfortunately we did not measure in the study the speed of injection.
The cause of transient neurologic symptoms is still unknown. The idea that the maldistribution of local anesthetic can be affected by the injection speed with the use of pencil-point needles cannot be ignored. However, Holman et al., in a recent study in a spinal cord model, concluded that, at clinically relevant rates of injection, needle characteristics minimally affect solution distribution.

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Secondly, Dr. Youngs implies that an injection of 3 ml of local anesthetic over 30 s is a “slow rate.” We would submit that it is difficult to inject much faster, given the high resistance of a 27-gauge spinal needle. Her point, however, is important, and we believe future studies should control for speed of injection as a potential variable in the development of TNS.

Finally, we would caution against equating TNS with “neurotoxicity.” Although TNS may be a mild form of local anesthetic toxicity, the etiology of these symptoms remains unclear.

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