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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[1] to Dr. Saidman's previous editorial,[2] 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 for and editors of this and similar journals.

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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.[1,2,3] 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 Drasner[4] 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.*[1] reported injecting the 3-ml test solution over approximately 30 s, which is equal to the rate reported by Rigler and Drasner[4]; 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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