References

1. Liguori GA, Zayas VM, Chisholm MF: Transient neurologic symptoms after spinal anesthesia with mepivacaine and lidocaine. ANESTHESIOLOGY 1998; 88:619–23

2. Martinez-Bourio R, Arzuaga M, Quintana JM, Aguilera L, Aguirre J, Saez-Eguilaz JL, Arizaga A: Incidence of transient neurologic symptoms after hyperbaric subarachnoid anesthesia with 5% lidocaine and 5% prilocaine. ANESTHESIOLOGY 1998; 88:624–8

3. Hampl KF, Heinzmann-Wiedmer S, Luginbuehl I, Harms C, Seeberger M, Schneider MC, Drasner K: Transient neurologic symptoms after spinal anesthesia. ANESTHESIOLOGY 1998; 88:629–33

4. Rigler ML, Drasner K: Distribution of catheter-injected local anesthetic in a model of the subarachnoid space. ANESTHESIOLOGY 1991; 75:684–92

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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 mepivacaine 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.

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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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.*,[1] 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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Reference

1. Holman SJ, Robinson RA, Beardsley D, Stewart SFC, Klein L, Stevens R: Hyperbaric dye solution distribution characteristics after pencil-points needle injection in a spinal cord model. ANESTHESIOLOGY 1997; 86:966–73

(Accepted for publication August 14, 1998.)