To the Editor.—We read with great interest the review by Dr. Joshi et al. on intracarotid delivery of drugs. In spite of recent advances in functional magnetic resonance imaging, the Wada test is still an important test for presurgical evaluation of patients with epilepsy. The intracarotid sodium amyloid test (Wada test) has been used to lateralize cerebral dominance for speech and to evaluate memory in each hemisphere. Anesthesiologists are generally not involved in this procedure. In some centers, an anesthesiologist is on stand-by during the procedure to manage potential complications, namely stroke. In the article by Dr. Joshi et al., there is mention that baseline sedation provided by the anesthesiologist in attendance would further complicate the interpretation of the Wada test and also suggests use of a judicious amount of sedation during the procedure. We disagree with the suggestion by the authors for the use of sedation during the procedure. These tests are done for evaluation of memory and language with unilateral intracarotid injection of drugs. Any sedation during the procedure will interfere with the memory testing.

As a result of the recurrent shortages in the availability of sodium amyloid, other agents are being used for Wada testing. As mentioned in the article, propofol or methohexitone have been used with limited success. Jones-Gotman et al. have published their work in the use of etomidate for the Wada test. They have shown that etomidate is a viable alternative to sodium amyloid, and its administration by bolus followed by infusion offers an improvement over the traditional Wada test. It is given as a 2 mg initial bolus (0.05-0.04 mg/kg) over 30 to 60 s, then an infusion of 0.005 to 0.004 mg/kg/min (approximately 6 ml/h neat etomidate 2 mg/ml) until the speech assessment and memory objects have been introduced. Many centers in the world are now switching to this etomidate speech and memory test. Manufacturer’s recommendations mandate an anesthesiologist to administer etomidate, so many anesthesiologists are now involved.

In Reply.—We thank Dr. Venkatraghavan for raising two specific comments on our review: The first is the use of sedation during Wada testing. We are thankful to Dr. Venkatraghavan for elaborating on the use of etomidate for Wada testing that is also supported by our experimental data. Hence, even if data from healthy volunteers might suggest a lack of amnesic effect with low doses of midazolam, caution needs to be exercised in prescribing sedatives or hypnotics to those with suspected neurologic deficits. What helped in framing the policies at our institution was a quality improvement review of patient data to assess the use of midazolam during Wada testing that found no impairment of memory.

We are thankful to Dr. Venkatraghavan for elaborating on the use of etomidate for Wada testing that is also supported by our experimental data. These comments illustrate the diversity in the selection and dose of drugs used intraarterially or systemically during endovascular procedures. We emphasize that the surgeons and anesthesiologists should formulate a coherent management strategy based on the specific needs of the patient and endovascular procedure.

References


(Accepted for publication January 7, 2009.)
To the Editor.—We read with interest the article published by Rigaud et al.1 The authors addressed the very important and insufficiently explored relationship between the parameters of electrical nerve stimulation and the precise position of the stimulating needle tip. Unfortunately, little data exist defining this relationship in special clinical situations like neuromuscular and metabolic diseases, and we applaud the authors for their effort.

While acknowledging the factual results of this study, we disagree with the authors’ interpretation of the findings regarding hyperglycemic dogs. Specifically, ink streaks under the epineurium were interpreted as needle penetration and intraneural injection. In our opinion, review of the photomicrographs (fig. 3 in the publication) does not necessarily support this conclusion. In these figures the amount of ink lodged under the epineurium appears negligible in comparison with that located outside the nerve, and the internal neural architecture remains intact.

It is possible that such marginal staining could have a biochemical rather than a mechanical explanation. In diabetes, an impairment in energy balance and tissue edema could result in a sufficient increase in epineurial permeability to allow some ink already in close contact to lodge under the epineurium. An alternative explanation could be migration of ink via dilated vasa nervorum. Eventually, performing the same experiment with ultrasound-guided injection would be very interesting.

In the absence of clinical data suggesting frequent nerve damage from performing electrical guided nerve blocks on diabetic patients, one of two possible conclusions of this study should be considered: penetration of local anesthetic inside the epineurium (with or without needle penetration) does not result in nerve damage, or that the results of this study are pertinent only to this specific experimental condition and do not warrant clinical extrapolation.

The answer to this question has particular importance in the context of the ongoing debate about the relative risk of electrical stimulation-guided blocks in comparison with ultrasound guidance.

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Reference


In Reply.—We thank Drs. Yanovski et al. for their interest in our paper.1 They raise the point that there are alternative explanations for our observations of traces of ink within the sciatic nerves of hyperglycemic dogs other than our conclusion that the needle tip was positioned there during the ink injection. They argue that the accumulation of ink within the nerve is less than what was found outside the nerve, and ink penetration through the intact epineurium is a more likely explanation.

Publication necessarily degrades the images and limits their number, so not all of the relevant sections can be shown. The original high-resolution images (see figure 1, Supplemental Digital Content 1, http://links.lww.com/A1219; figure 2, Supplemental Digital Content 2, http://links.lww.com/A1220; figure 3, Supplemental Digital Content 3, http://links.lww.com/A1212) show clear dissection of ink among the fascicles of the nerves and travel of this ink as rivulets within the nerve to areas distant from the external accumulation in patterns not expected for direct diffusion. In other images, there is substantial destruction of the normal nerve anatomy at the injection site.

Finding the majority of the ink outside the nerve is compatible with our interpretation that injection was originally into the nerve, since the nerve is not capacious and the short path for retrograde flow along the outside of the needle shaft is not likely to be occluded by adjacent tissue pressure. Passage of the ink through membranous barriers is unlikely. The tissue was harvested immediately and frozen within 10 min, so limited time was available for such a process. Also, particulate ink such as was used for this study does not transit through membranes or vascular walls, and for this reason is routinely employed for vascular labeling.2

Bleeding seen within the nerves is clearly visible in the original photographs only in specimens showing intraneural ink (please see the supplemental digital content), which independent of ink distribution patterns conclusively indicates an intraneural needle placement. Overall, we believe the most likely explanation for these various observations is that needle insertion guided by electrical stimulation resulted in intraneural placement in the hyperglycemic dogs.

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References
