

With respect to CSA, size does matter, particularly with regard to regulatory restrictions, because practitioners currently remain at liberty to use large-bore catheters for this purpose.⁶ Indeed, many resort to this technique after inadvertent dural puncture during attempted epidural placement. More fundamentally, there are important differences in subarachnoid distribution between injections made through large- and small-bore catheters,¹⁰ although these result from differences in flow rate, which will be blurred with drugs administered by slow infusion.

Because of the substantial challenges and obstacles in conducting a study of this nature, the current data are likely the best that will be collected anytime soon, which is unfortunate given the numerous questions that remain. Among the most critical, identification of the optimal combination of analgesic/anesthetic agents and the optimal method of delivery has yet to be determined. It is well established that slow infusion potentiates restricted distribution,¹⁰ and a reduction in required dosage, improved analgesia, and reduced risk of anesthetic neurotoxicity might be achievable if an anesthetic is administered by repetitive bolus injection. However, the extent to which this can be realized with these high-resistance catheters also remains a question.

In their 1944 report of CSA for labor and delivery, Hinebaugh and Lang³ concluded: "While no serious complications occurred in this series, further trial is neces-

sary to evaluate its future place in obstetrical anesthesia." These words are perhaps as relevant now as they were 60 years ago.

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Ultrasound-guided Regional Anesthesia and the Prevention of Neurologic Injury

Fact or Fiction?

PERIOPERATIVE nerve injuries have long been recognized as potentially devastating complications of regional anesthesia. A recent review of the published literature estimates that neurologic complications may occur in up to 3% of patients undergoing peripheral

nerve blockade and in 0.4% of patients after neuraxial techniques.¹ Fortunately, the number of these complications progressing to severe or disabling injury is extremely low. In fact, it has been estimated that 1 in 14,000 patients will develop a severe neurologic injury after spinal or epidural anesthesia.² Despite these encouraging results, the potential for devastating sequelae will always be a concern for both patients and providers. In this issue of *ANESTHESIOLOGY*, Koff *et al.*³ further accentuate these concerns by presenting a case of severe brachial plexopathy after an ultrasound-guided interscalene block in a patient with multiple sclerosis (MS). The case is unique in that it highlights several important factors that should be considered by clinicians when evaluating patients and assessing the risk of regional anesthetic techniques. Important considerations include identifying potential contributors to perioperative nerve injury, understanding the importance of

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This Editorial View accompanies the following article: Koff MD, Cohen JA, McIntyre JJ, Carr, CF, Sites BD: Severe brachial plexopathy after an ultrasound-guided single injection nerve block for total shoulder arthroplasty in a patient with multiple sclerosis. *ANESTHESIOLOGY* 2008; 108:325-8.

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preexisting neurologic deficits, and recognizing the limitations of ultrasound-guided technology in preventing neurologic injury.

Perioperative nerve injury is a complex phenomenon that can be caused by a multitude of clinical factors. Patient, surgical, and anesthetic risk factors have all been identified as potential contributors to postoperative neurologic dysfunction. The case presented by Koff *et al.* likely represents a clinical scenario in which several patient, surgical, and anesthetic variables contributed to an adverse neurologic event. It is unlikely that a single identifiable agent was the definitive cause of injury. In fact, a review of the American Society of Anesthesiologists Closed Claims database suggests that despite intensive medicolegal investigation, the cause of postoperative neurologic injuries is rarely identified.⁴

Patient Risk Factors

Patient risk factors most commonly associated with perioperative nerve injury include male sex, increasing age, extremes of body habitus, and preexisting diabetes mellitus.⁵ However, it has been suggested that patients with preexisting neurologic deficits may be at increased risk as well. The patient presented in the case report by Koff *et al.* was an elderly man with preexisting MS. The presence of chronic, underlying neural compromise secondary to mechanical, ischemic, toxic, metabolic, or in this case demyelinating conditions may theoretically place these patients at increased risk of further neurologic injury.^{6,7} As described by Koff *et al.*, the “double-crush” phenomenon suggests that patients with preexisting neural compromise may be more susceptible to injury at another site when exposed to a secondary injury.⁷ Secondary injuries may include a variety of concomitant patient, surgical, or anesthetic risk factors.

Many clinicians are unaware that subclinical neural compromise may be present within the peripheral nervous system of patients with MS.^{8,9} In fact, subclinical sensorimotor deficits have been identified in 45%⁹ to 74%⁸ of MS patients, with up to 43% having abnormalities in more than one peripheral nerve distribution. This often ignored or poorly recognized phenomenon has been appropriately highlighted by Koff *et al.* The authors emphasize the need for clinicians to consider these and other factors when evaluating MS patients for peripheral nerve blockade. Unfortunately, neural compromise may be present within the peripheral nervous system in the absence of clinical signs or symptoms and does not seem to be correlated with patient age, disease onset, or progression of the disease course. This lack of clinical correlation presents a unique challenge to anesthesia providers when evaluating MS patients for peripheral regional techniques.

Surgical Risk Factors

Surgical risk factors associated with perioperative nerve injury include direct intraoperative trauma or stretch, vascular compromise, perioperative infection or inflammation, hematoma formation, tourniquet ischemia, or improperly applied immobilizers or casts. Surgical variables may be the primary etiology of postoperative neurologic deficits in up to 88% of cases.¹⁰ One of the most important surgical risk factors may be the surgical procedure itself. Koff *et al.* briefly alluded to the fact that the surgical procedure may have been a contributing factor in the development of the patient's severe brachial plexopathy. Total shoulder arthroplasty may be associated with postoperative neurologic deficits in up to 4.3% of cases—regardless of anesthetic technique—with the majority of injuries being localized to the upper trunks of the brachial plexus.¹¹

Anesthetic Risk Factors

Regional anesthetic factors that may contribute directly or indirectly to perioperative nerve injury include needle- or catheter-induced mechanical trauma, ischemic nerve injury secondary to vasoconstrictors or neural edema, and chemical injury from direct local anesthetic neurotoxicity.¹² Several authors have investigated the role of mechanical trauma, including the role of needle gauge, type, and bevel configuration on peripheral nerve injury. The disruption of perineural tissue around nerve fascicles compromises the blood-nerve barrier and results in the herniation of endoneurial contents (*i.e.*, myelinated nerve fibers) into the perineural space. However, needle-to-nerve contact by itself—in the absence of local anesthetic injection—rarely produces clinical or functional abnormalities. Rather, it is the combined effect of needle penetration and injection of local anesthetic into the neural fascicle that causes axonal degeneration and subsequent neurologic injury.¹²

Limitations of Ultrasound-guided Regional Anesthesia

Finally, the ability of ultrasound-guided regional anesthesia to become the “holy grail” of regional anesthesia—providing neural blockade with rapid onset, long duration, and improved success, without complications—has recently been discussed.¹³ Although many advocates of ultrasound theorize that direct visualization of neural targets and needle advancement may decrease the frequency (and severity) of neurologic injury, preliminary results do not support the hypothesis that ultrasound guidance decreases the risk of neurologic complications.¹³ This should not be surprising if we consider

the risk factors associated with neurologic injury and the ability (or lack thereof) of ultrasound in preventing these risk factors from making a clinical impact. For example, clearly the use of ultrasound guidance will have no impact on patient risk factors associated with nerve injury. The patient described by Koff *et al.* will have the associated risk factors of male sex, increasing age, and a preexisting neurologic deficit regardless of anesthetic technique. Similarly, the use of ultrasound guidance will have no effect on surgical factors. Patients undergoing total shoulder arthroplasty will still be at risk of intraoperative trauma or stretch to the brachial plexus, hematoma formation, and perioperative inflammation. However, it is not unreasonable to presume that ultrasound may have a positive impact on anesthetic risk factors—albeit small. Of the anesthetic risk factors involved in perioperative nerve injury (mechanical trauma, neural ischemia, and local anesthetic toxicity), ultrasound guidance may be able to modify one, or at most two contributing factors, namely, mechanical trauma and local anesthetic neurotoxicity.

The ability of ultrasound guidance to avoid needle-to-nerve contact and mechanical trauma is an appealing assumption. However, is this assumption a true reflection of clinical practice? For example, the ability to visualize both the needle tip and relevant neural targets at all times is extremely difficult. In fact, data from Koff *et al.*'s own institution suggests that failure to maintain needle visualization during advancement may occur in up to 43% of novices (<10 ultrasound-guided blocks) and 10% of experienced providers (>60 ultrasound-guided blocks) performing ultrasound-guided techniques.¹⁴ This is not a criticism, but rather a reflection of the difficulty associated with maintaining needle alignment within the narrow plane (1 mm) of the ultrasound beam. Finally, preliminary evidence is beginning to suggest that ultrasound-guided technology may allow regional techniques to be performed with lower volumes of local anesthetic while maintaining similar degrees of block efficacy. This benefit may theoretically influence risk factors of neural injury associated with direct local anesthetic neurotoxicity. However, definitive data are currently lacking on these assumptions as well.

In summary, the case report by Koff *et al.* highlights several important points. First, clinicians must identify all potential risk factors associated with perioperative

nerve injury prior to performing regional techniques. This includes recognizing that patients with preexisting neurologic deficits may be particularly susceptible to secondary injuries. Second, consider whether the perceived benefits of regional anesthesia justify the potential for added risk (mechanical trauma, neural ischemia, local anesthetic toxicity). If so, consider modifying your anesthetic technique to minimize the impact of additional risk factors. Modifications may include reducing local anesthetic concentrations, eliminating epinephrine additives, or proceeding with general anesthesia. Finally, recognize the limitations of ultrasound-guided technology in reducing the risks associated with neurologic complications. Failure to appreciate the limitations of ultrasound may breed complacency and create an illusion of safety—factors that may *increase* the risk of nerve injury and adverse patient outcomes.

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