Ultrasound Guidance and Peripheral Nerve Injury

Is Our Vision as Sharp as We Think It Is?

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First, please understand that the authors of this editorial were selected primarily on a self-deprecating criterion—the presence of more than just a few gray hairs—that, and an interest in neurologic injury associated with regional anesthesia, for which there are assuredly experts younger and brighter than ourselves, but who lack the perspective that we wish to convey. We began our anesthesiology training in the 1980s, shortly after the introduction of peripheral nerve stimulation (PNS) into clinical practice. This new nerve localization tool was exciting because it promised to end the "Neanderthal practice of no paresthesia, no anesthesia" and in so doing put an end to regional anesthesia–related nerve injury. Elegant diagrams depicted how emitted currents of electricity would identify neural targets but supposedly prevent the needle from actually touching the nerve. Alas, more than a quarter-century later, there are no convincing data that the frequency of nerve injury has been significantly reduced by PNS. Will the same hold true for ultrasound-guided regional anesthesia (UGRA), the newest nerve localization tool and one of the most exciting advances in regional anesthesia during our lifetimes?

In less than 2 decades, ultrasound guidance has evolved from enabling the crude deposition of local anesthetic near a vascular structure known to be anatomically adjacent to a poorly imag(in)ed nerve, to a robust technical innovation that has become a major focus of anesthesia departmental budgetary planning and continuing medical education. Studies show that ultrasound guidance hastens block onset, reduces unintended vascular puncture, and lessens the volume of local anesthetic necessary to anesthetize a nerve—all worthy attainments that have not yet resulted in demonstrably higher success rates for surgical anesthesia or consistent reduction in serious local anesthetic systemic toxicity (or other complications).[1,2] Ultrasound has clearly brought about renewed interest in regional anesthesia, perhaps increased its use, and has resulted in calmer attending anesthesiologists, who can now actually see just how far off-target a resident's block needle has strayed. Real-time observation of nerves and other anatomy thus give us a sense of control that heretofore was missing in our practice and one that we suspect has led believers and nonbelievers alike to hope that UGRA might meaningfully reduce our patients' risk of peripheral nerve injury. But actually proving that UGRA is safer than PNS or even blind paresthesia techniques is unlikely because permanent nerve injury is so rare that it precludes meaningful statistical analysis. For example, one might begin with the knowledge that a month or two after peripheral nerve block, about 3% of our patients manifest a new, persistent neurologic symptom that may or may not be block related.[3] To prove that UGRA could reduce this incidence by 50% (as compared with PNS) would require approximately 3000 patients per group.[4] Although this study may be possible in a busy center, the results might not be as helpful as we would wish for because most of early postoperative nerve symptoms are transient. Indeed, a reasonable estimate is that only 4 of 10,000 patients undergoing peripheral nerve block will have a needle placement–related deficit 12 months afterward.[5,6] Using this number, we calculate that more than 70,000 patients per group would be required to prove a 50% reduction ($\alpha = 0.05$, $\beta = 0.8$). Such a study is unlikely to be undertaken in the resource-limited world of anesthesiology research. In addition, because the frequency of nerve injury varies by block,[3] a well-designed study would be exponentially more difficult if limited to a single block with idealized standardization of local anesthetic concentration and volume, adjuvant, needle type, and so on.

Acknowledging the absent "Holy Grail" of a definitive and sufficiently powered randomized controlled trial, what do we currently know about peripheral nerve injury and UGRA? Recent reviews of randomized controlled trials that compared UGRA to PNS found no difference in the incidence of unintended intraoperative paresthesia or postoperative neurologic symptoms.[1,2] Two large case series[5,7] have added further insight, but no definitive answers. Barrington et al[5] collected data on

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more than 8000 peripheral blocks and found no statistical difference in the surrogate marker of block-related paresthesia between ultrasound-guided, PNS, or combined techniques. Meticulous neurologic evaluation of the 30 patients with persistent (<12 months) deficits determined only 3 to be block related, and there was no difference based on localization technique. Interestingly, this is the same frequency of approximately 4 long-term injuries per 10,000 patients as reported by Auroy et al[8,9] a decade earlier, wherein PNS was the exclusive nerve localization tool. In the other large case series, a quality-assurance database of more than 5000 peripheral blocks, Orebaugh et al[7] used neurologic consultation to identify 3 long-term nerve injuries (2 were improving) and found no statistical difference between techniques.

Although systematic reviews and large case series such as the above provide valuable population data, they lack the individual patient detail contained in a case report. A single published case report has described a patient who sustained permanent injury associated with an ultrasound-guided interscalene block, but the injury was more likely explained by surgical causes and preexisting multiple sclerosis.[10] This issue of *Regional Anesthesia and Pain Medicine* contains 2 new case reports that document serious anesthesia-related nerve injury in the setting of UGRA. These reports are all the more remarkable because they come from 2 groups with unassailable reputations for excellence in regional anesthesia. Together they provide cautionary notes not only about the fallibility of UGRA, but about our overall practice of peripheral nerve blockade.

Cohen and Gray[11] describe an otherwise healthy 36-year-old man who underwent ultrasound-guided interscalene block for shoulder surgery. The unsedated patient reported no unusual sensations during an unremarkable block procedure. The next day the patient noted flaccid paralysis and sensory deficit within the fifth and sixth cervical nerve root distribution. A previously unrecognized intraneural injection into a component of the brachial plexus was seen on subsequent review of stored video images. Fortunately, the patient fully recovered within 6 weeks. The case reported by Reiss et al[12] involves an ultrasound-guided, PNS-assisted supraclavicular block for thumb arthroplasty and ligament transfer. Despite having received midazolam 8 mg and fentanyl 50 μg, the 68-year-old female patient was described as "verbal and responsive to commands" during the uneventful block placement. Over the next 6 days, she developed wrist drop and severe allodynia. Electrophysiological studies at 3 weeks confirmed acute denervation of the radial nerve and brachial plexopathy at the level of the supraclavicular fossa. Complete wrist drop still remained at 8 months. The courageous and laudable reports of these complications by Cohen and Gray[11] and Reiss et al[12] provide us with detail of nerve injuries that were almost certainly block-related and that occurred in the setting of ultrasound guidance. Despite our collective hope that ultrasound would at least reduce anesthesia-related nerve injury, few of us really expected that it would completely eliminate the complication. We now have 2 large case series and 3 case reports that confirm these suspicions.

Importantly, these reports point to larger issues related to peripheral nerve blockade, of which the choice between UGRA and PNS localization is only a part. First, intraneural injections during the course of UGRA have been documented by recent studies.[13,14] None of the 46 patients in these 2 studies had nerve damage, which challenges the conventional wisdom that intraneural injection is often associated with injury. There are a handful of case reports, including one from Gray's group,[15] in which unintentional ultrasound-guided intraneural injection resulted in no injury. We believe this to be provocative information, but are concerned when book chapters extrapolate this information to suggest intentional ultrasound-guided intraneural injection might be a powerful tool for achieving successful blockade.[16] One can rationalize that on cross section, peripheral nerves primarily consist of connective tissue (more so distally than proximally)[17] and that dull block needles are perhaps more likely to penetrate connective tissue than the fascicles that contain and protect individual neurons.[18] Conversely, sharp needles penetrate cadaveric human sciatic nerve fascicles 3.2% of the time,[18] and even if dull needles seem less likely to enter fascicles, they are known to cause more damage when they do so.[19] Moreover, the typically acquired 1- to 2-mm resolution of ultrasound beams is incapable of consistently identifying fascicles, many of which are less than 1 mm in diameter. Indeed, even with a 15-MHz transducer, only one third of sciatic nerve fascicles can be seen ultrasonically as compared with light microscopy.[20] Cohen and Gray[11] have thus provided strong clinical evidence that *recognized* unintentional intraneural injection can indeed result in injury; the patient of Reiss et al[12] may have sustained an unrecognized intraneural injection as a component of her apparently permanent injury. Considering the above evidence, we strongly agree with Cohen and Gray's admonishment that intentional intraneural injection seems to be an unnecessary roll of the dice, particularly for undocumented benefits that may be as trivial as marginally faster block onset or longer duration.

Two other aspects of peripheral nerve block practice that extend beyond the choice of nerve localization tool deserve comment. If the case report of Reiss et al[12] seems short on details, it is because the authors responsibly reported only the information that was available to them and refrained from speculation. The absence of detailed records of block procedures is all the more frustrating when we try to analyze very rare complications. Electronic medical records may improve this, depending on the amount of detail incorporated in the database. However, there is little reason for any of us not to adopt easy-to-use records specifically designed for documenting peripheral nerve blocks.[21] This is an easy solution.

Unfortunately, our final comment is not as easily addressed. How should we monitor peripheral nerve block procedures? Ultrasound allows us to observe the nerve, its surrounding tissues, and local anesthetic spread. Yet nerve expansion is not a completely reliable indicator of functional injury,[22] and keeping the needle tip in view is not always possible.[23] Peripheral nerve stimulation, despite its insensitivity for accurately identifying needle-to-nerve proximity,[24] may provide valuable information, such as knowing that increased electrical impedance[25] or a motor response at 0.2 mA or less is suggestive of intraneural needle placement.[26] Monitoring injection pressures is a promising and evolving science that may eventually be validated as providing useful information in our patients.[27,28] And although both case reports contained in this issue demonstrate that serious nerve injury can occur in patients who are unsedated or sedated but cooperative, many anesthesiologists believe that wakefulness is yet another reasonable monitor.[29] Could the use of some combination, if not all, of these monitoring modalities reduce the frequency of nerve injury? We agree with the suggestion of Reiss et al that multiple monitors might increase the odds of detecting an atypical patient—or monitor—response during block placement. Recent publications support this notion by observing ultrasound-proven intraneural injection coincident with pain, motor response of less than 0.2 mA, and elevated injection pressure.[14,26] Although these observations are intriguing, there are admittedly no randomized data to guide us, much less offer any degree of assuredness that we can know with confidence the valid end points for each individual monitor.

Perhaps in another decade or so some other editorialists with gray hair will tell us the answer. From the perspectives symbolized by our own gray hair, we have seen that although the full promise of PNS was never realized, it was nevertheless a revolutionary tool for regional anesthesia and still plays an important role. We suspect that the same will be true for ultrasound. The importance of the thought-provoking case reports discussed here is not that they "proved" injury can happen despite the use of ultrasound guidance, but that they remind us that safe and effective practice of regional anesthesia does not reside (and never has resided) in the use of a single piece of equipment. Safety is a mix of proper training, reliable monitors, good judgment, and plain old common sense. We should not expect ultrasound to solve all of our problems, nor should we extrapolate its benefits as justification for pushing the limits of patient safety.

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Nerve Injury Complicating Ultrasound/Electrostimulation–Guided Supraclavicular Brachial Plexus Block

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Background and Objectives: Neurologic complications after peripheral nerve blocks (PNBs) are relatively uncommon. It has been postulated that real-time, needle-nerve visualization during ultrasound-guided PNBs might further reduce the risk of neurologic or vascular complications.

Case Report: In this report, we describe the occurrence of a severe brachial plexus injury after combined ultrasound and nerve stimulator–guided supraclavicular brachial plexus block.

Conclusions: Ultrasound guidance should not preclude development of additional monitoring and protocols to decrease the risk of complications with PNBs.

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Nerve injury associated with peripheral nerve blocks (PNBs) is a relatively uncommon complication[1,2] and permanent injury is rare. It has been postulated that the introduction of ultrasound (US) guidance during nerve localization and injection of local anesthetic may even further reduce neurologic injury by allowing monitoring of the needle-to-nerve relationship. In this report, we describe an occurrence of severe brachial plexus injury after US-guided supraclavicular brachial plexus block.

CASE REPORT

A 68-year-old woman presented for arthroplasty and volar capsulodesis of her left thumb basal joint with ligament transfer and carpal tunnel release. The patient gave her permission for the publication of this report. Other than osteoarthritis, her medical history was unremarkable. Preoperative neurologic examination by the orthopedic surgeon was reported as normal except for a positive median nerve compression test. However, there was no thenar atrophy or weakness, and flexor and extensor movement of the left arm and forearm was normal. Two-point discrimination was 5 mm throughout, and grip strength was 50 lb, consistent with a normal examination.

Upon application of standard American Society of Anesthesiologists (ASA) monitors, the patient was premedicated with incremental intravenous doses of midazolam and fentanyl (totaling 8 mg and 50 μg, respectively). The patient was sedated and calm but remained verbal and responsive to commands throughout the block procedure. A left supraclavicular block was performed using a 5-cm, 22-gauge Stimuplex needle (BBraun Medical, Inc, Bethlehem, Pa) under US guidance (SonoSite S-Nerve Ultrasound System fitted with a L38X 10- to 5-MHz transducer, SonoSite, Inc, Bothell, Wash). A nerve stimulator (Digistim; BBraun Medical, Inc) was simultaneously used to confirm needle position. The brachial plexus was visualized lateral to the subclavian artery and approached by the block needle from lateral-to-medial using an in-plane technique. The block was performed by a CA-2 anesthesiology resident under the supervision of an anesthesiologist with 20 years of experience of performing PNBs and 4 years of experience with US-guided PNBs. During the needle positioning, mixed motor responses of the arm and forearm were noted first. The minimal current at which the motor response of the forearm persisted was reported as 0.4 mA (0.1 millisecond). Unfortunately, the information pertaining to specific motor response at the time of injection, resistance to injection, and the US images were not documented. A total of 30 mL ropivacaine 0.6% without additives was injected using two 20-mL syringes; there was no pain on injection. Fifteen minutes after the injection, adequate sensory-motor block was documented.

The surgical procedure commenced and was completed uneventfully using the supraclavicular block as the sole anesthetic. An arm tourniquet was applied (200 mm Hg) for 1 hr 45 mins. Postoperatively, a short arm cast was placed from just below the elbow to the proximal digits. The patient was discharged home and subsequently reported that the block lasted approximately 24 hours after the surgery.

On postoperative day (POD) 2, the patient contacted the surgeon complaining of severe pain in the posterior arm, over the triceps muscle, and the lateral forearm. The pain radiated to the parascapular area, and its intensity was reported as 10/10. On POD 3, with the cast in place, the pain persisted and was associated with inability to extend fingers, prompting evaluation in the surgeon's office. At the time of the evaluation, she was unable to extend any of her left fingers and had a wrist-drop. Compression from the cast was excluded based on location of the pain above the cast, lack of finger swelling or discoloration, and the extent of the neurologic dysfunction, all of which clinically suggested that the injury to the radial nerve occurred above the elbow. On POD 6, the patient developed severe allodynia over the posterior arm above the level of the cast as well as the forearm. She was referred to our pain management division for evaluation. After removal of the cast, physical examination confirmed an inability to extend the left wrist (wrist-drop), a marked decrease in triceps strength, and allodynia over

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the radial nerve distribution. A magnetic resonance imaging of the brachial plexus was ordered to rule out hematoma or an interruption of the continuity of the brachial plexus and was reported to be normal. Gabapentin 600 mg 3 times daily, oxycodone extended-release 20 mg twice daily, and oxycodone immediate-release 5 to 10 mg every 6 hrs were prescribed for the presumptive diagnosis of nerve block–related neuropraxia. The patient was referred to physical therapy.

Three weeks postoperatively, the patient still had pain over the posterior arm and forearm and the inability to extend her fingers. Her pain control was improved with gabapentin, decreasing from a visual analog scale score of 10/10 to 5/10. Six weeks postoperatively, electromyography and nerve conduction studies indicated acute denervation of the radial nerve and left brachial plexopathy at the level of the supraclavicular fossa, as well as absent sensory and motor latency of the left radial nerve consistent with acute injury to the posterior cord/division of the brachial plexus. The median and ulnar nerves were normal. Eight months after the injury, the patient remains disabled with complete wrist-drop and inability to use her hand, although the neuropathic pain has resolved.

DISCUSSION

The incidence of complications related to PNB is reportedly low—from approximately 3% within 4 to 6 weeks of surgery to approximately 2 to 4/10,000 at 1 year.[1] Moreover, recent reports suggest that most neurologic complications detected postoperatively may be related to surgery, rather than to regional anesthesia and that many neurologic injuries related to regional anesthesia tend to be transient (reversible) in nature.[1–3] However, our patient had a permanent neurologic injury associated with a supraclavicular brachial plexus block, suggesting that the ongoing developments in PNB such as US guidance and improvements in peripheral nerve stimulation have not fully eliminated the possibility of serious complications.[4,5]

The ability to visualize the needle-to-nerve relationship and the deposition of the local anesthetic has led some clinicians to postulate that sole reliance on US guidance is adequate for the safety and effectiveness of PNBs. The development of severe brachial plexus injury related to the US-guided supraclavicular brachial plexus block in our patient makes a compelling argument against such a stance. Several recent publications also suggest that new neurologic symptoms can, and do, occur with US-guided PNBs, with an incidence that may be similar to that reported using non–US-guided techniques.[4–8]

Determining the exact etiology of PNB-related neurologic complications retrospectively in our or any other patient is notoriously difficult. Multiple factors may contribute to nerve injury after a PNB including preexisting patient condition, traumatic needle injury, hydrostatic force during injection, and local anesthetic neurotoxicity, as well as surgical trauma. Excessive sedation was unlikely an issue in our patient because she reportedly remained conversant during the block performance. A motor response at a very-low-current intensity (eg, <0.2 mA, 0.1 millisecond) may warn of an intraneural needle placement. The minimum current used in our patient was 0.4 mA; this however, does not specifically rule out intraneural needle placement.[9,10] Moreover, reliance on nerve stimulation to rule out an intraneural injection may be further diminished in the setting of multiple injections of local anesthetic.[11]

In summary, our report describes a severe and apparently permanent neurologic injury that occurred despite the patient being awake and having undergone dual US nerve stimulation–guided nerve block. Because no single monitoring system to prevent neurologic complications is perfect, it may be advantageous to combine several modalities to reduce the risk of severe complications of PNBs.

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