Literature Review

Nerve Injury After Peripheral Nerve Blockade

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Nerve injury following peripheral nerve blockade (PNB) is, thankfully, uncommon but nonetheless still occurs. Multiple investigations have assessed the frequency of this complication, although many are limited by study design and inconsistent neurodiagnostic follow-up. Moreover, it is difficult to determine the precise etiology of postoperative neurologic deficits (PNB-related versus surgical). Despite these limitations, adequate data exist to reach reasonable conclusions regarding this infrequent complication.

The mechanism of nerve injury after surgery accompanied by PNB may be related to several factors, including block-related events such as needle trauma, intraneural injection (INI) and local anesthetic (LA) neurotoxicity; surgical factors such as surgical trauma, stretch injuries and the impact of tourniquets, hematomas, compressive dressings and positioning; and the potential impact of pre-existing conditions such as bony deformities and peripheral neuropathies. Needle trauma is probably uncommon, while INI seems to be the likely mechanism of block-related nerve injury in most patients. Data suggest that high injection pressures and severe pain on injection may be indicators of INI and subsequent fascicle disruption.[1,2]

The peripheral nerve is a complex structure bounded by the epineurium that encases multiple nerve fascicles surrounded by a perineural layer. Each fascicle contains myelinated neurons that can be damaged by the intrafascicular injection of LA, which appears to produce neurologic injury by swelling and edema of the fascicle, subsequent neurovascular compromise and potential LA toxicity. Interestingly, Bigeleisen[3] demonstrated that 81 percent of patients undergoing ultrasound (US)-guided axillary block had evidence of INI in at least one nerve with no subsequent evidence of neurologic injury, suggesting that not only does small volume INI not produce clinical nerve injury but that INI appears to occur in a high percentage of patients, a finding confirmed by clinicians experienced with US-guided PNB (personal communication). Although it might be assumed that US would permit proper LA deposition *around* rather then *within* the nerve, there are no clinical data to validate that assumption. These findings of Bigeleisen also imply that injection beneath the perineurium is the probable site of injury from INI.[4]

In addition to block-related injury, surgical factors appear to be especially important in producing neurologic deficits. Experimental data, as well as electrophysiologic studies in patients with nerve injury, demonstrate the compressive and neuronal ischemic effects of excessive tourniquet duration and inflation pressure on peripheral nerves. Horlocker et al.[5] and Fanelli et al.[6] demonstrated that duration of tourniquet inflation and pressures > 400 mm Hg, respectively, were associated with an increased incidence of postoperative neurologic deficits after limb surgery. Retractor injuries to the femoral nerve during hip arthroplasty, stretch injuries of the brachial plexus during shoulder arthroplasty and peroneal nerve injury related to preoperative valgus deformities and flexion contractures after knee arthroplasty are additional mechanisms of injury that produce postoperative neurologic deficits, unrelated to PNB. In fact, Horlocker et al.[5] noted that 89 percent of neurologic deficits after 1,614 axillary blocks were related to the surgical procedure itself, a finding consistent with other clinical reports. In addition, 4 percent of patients undergoing shoulder arthroplasty sustain brachial plexus injuries[7] in the absence of PNB, again suggesting surgical nerve injury. Candidio et al.[8] observed that of the 4.4 percent of 684 patients experiencing a postoperative paresthesia after interscalene block (ISB) for shoulder surgery, 45 percent were located *at the site of the block*, and 23 percent were in the distribution of the greater auricular nerve; more serious distal sensorimotor neuropathies were thus infrequent. Finally, although preoperative neuropathies and nerve localization techniques *may* be associated with postoperative nerve injury, well-designed prospective studies have failed to show any consistent relationship between diabetes, pre-existing neuropathies or the use of nerve localization techniques and the incidence of neurologic deficit after PNB.[8,9,6]

Most postoperative neurologic complaints manifest within the first 48 hours after surgery, are typically sensory deficits and usually resolve within two to four weeks, although some deficits may rarely require up to nine months for complete recovery. Nerve conduction studies (NCS) and electromyography (EMG) typically reveal conduction delays consistent with neuropraxia, a temporary injury pattern associated with functional recovery. Assessment of neurologic deficits should include a careful neurologic examination, and, in most cases, NCS and EMG. Repeat studies are commonly performed at four to six weeks, after which clinical assessment appears to suffice in the absence of severe motor deficits.

Determining the incidence of block-related nerve injury is limited by a variety of factors, including inconsistent follow-up, the use of self-reporting and survey analyses of complications, the lack of standardized documentation of preoperative and postoperative neurologic function, the absence of routine postoperative neurodiagnostic testing and the limited ability of these tests to determine the precise etiology of nerve injury. Despite

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these limitations, however, prospective analyses of more than 70,000 patients using carefully conducted survey studies suggest incidence rates of 0.02 percent, a likely underestimation due to self-reporting but nonetheless an impressively infrequent rate of nerve injury.[5,6] Other prospective and retrospective analyses have shown higher complication rates of 0 to 8 percent after single injection upper-extremity blockade and rates < 0.5 percent for

Table 1: Characteristics of Perioperative Nerve Injury After Peripheral Nerve Block

Author	Pt#	Study Design	SS vs. CC	Block Type UE	Block Type LE	Incidence (F/U time)	Recovery (@mos)	Deficit
Auroy 2002	50,223	Pro (Survey)	SS	All	All	0.02% (NST)	42 (6 mos)	neuropathy
Auroy 1997	21,278	Pro (Survey)	SS	All	All	0.02% (48 hrs)	100%	neuropathy
Fanelli 1999	3,996	Pro (Survey)	SS	ISB, AX	FB, SB	1.7% (1 mos)	99% (3 mos)	neuropathy
Klein 2002	2,382	Pro	SS	All	FB, SB	0.25% (7 days)	100% (3 mos)	paresthesias, numbness
Stan 1995	1,995	Pro	SS	AX	—	0.2% (NST)	100% (2 mos)	sensory paresthesias
Horlocker 1999	1,614	Retro	SS	AX	—	8.4% (2 wks)	100% (5 mos)	pain, numbness
Candido 2005	693	Pro	SS	ISB	—	8.5% (2d -1 mos)	97% (4 mos)	pain, pare-hypesthesias, IS pain
Bishop 2005	568	Retro	SS	ISB	—	2.3% (2 wks)	91% (6 mos)	sensory neuropathy
Davis 1991	543	Retro	SS	BRPLX	—	0% (NST)	—	NST
Borgeat 2001	520	Pro	Both	ISB	—	14% (10 days)	99% (6 mos)	paresthesias, dysesthesias
Capdevila 2005	1,416	Pro	CC	All	All	0.2% (24 hrs)	100% (3 mos)	femoral nerve lesions (n=3)
Singelyn 1999	1,142	Pro	CC	—	FNC	0.1% (1 wk)	64% (NST)	dysesthesias (2), motor weakness (1)
Borgeat 2003	700	Pro	CC	ISC	—	8% (10 days)	100% (7 mos)	paresthesias, pain, dysesthesias
Swenson 2006	620	Pro	CC	ISC	FNC,SC, PC	0.3% (1 wk)	100% (2 mos)	weakness, sensory loss
Bergman 2003	405	Pro	CC	AXC	—	1% (postop)	100 (NST)	pain, numbness

Pt: patient; SS: single shot; CC: continuous catheter; F/U: follow-up; mos: months; NST: not clearly stated; Pro: prospective; Retro: retrospective; AX: axillary; ISB: interscalene block; FB: femoral block; SC: sciatic block; BRPLX: brachial plexus; FNC: femoral nerve catheter; PC: popliteal catheter; ISC: interscalene catheter.

Table includes largest studies with neurologic outcome data to assess; some incidence values reflect all neurologic deficits, regardless of etiology (surgical versus block-related), while others reflect only block-related deficits.

lower-extremity blocks. Studies of continuous peripheral nerve block (CPNB) techniques have revealed similarly low neurologic rates. Capdevilla[12] et al. demonstrated a 0.21-percent incidence of nerve injuries after 1,416 upper- and lower-extremity CPNBs, as did Swenson[13] et al. in a similar analysis of 620 CPNBs.

In conclusion, nerve injury can occur after PNB but is infrequent, is typically a transient sensory neuropraxia, may primarily be related to surgical rather than block-related mechanisms, *may* represent INI that can be minimized by discontinuing injection when either high injection pressures or pain are encountered, and appears unrelated to the type of nerve localization technique employed. Whether US will reduce the already low incidence of these complications has yet to be studied, but it certainly holds promise for visual, real-time assessment of needle placement and LA deposition. Ultimately, however, as long as needles, nerves and local anesthetics are in close proximity, the potential for nerve injury will always exist, and clinicians must continue to use proper techniques to reduce the risk of INI and postoperative neurologic deficits.

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Incoming President Seeks 'Added Value' for ASRA Membership

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chronic pain also are envisioned. Such "e-chapters" will be peer-reviewed by a panel derived from the editorial board of *Regional Anesthesia & Pain Medicine.*

Swelling the Ranks

As a Society's lifeblood is its influx of new members, ASRA is making efforts to attract and maintain a lifelong relationship with anesthesiology residents and pain fellows. ASRA is expanding its efforts to provide educational programs directed specifically to those still in training. These efforts consist of Cracker Barrel sessions, lectures of specific interest to residents/fellows, such as employment contracting, and cadaver-based workshop for fellows. In addition, ASRA provides five $1,000 travel awards for residents submitting abstracts for presentation at each of the annual meetings.

With the very successful completion of the Annual Regional Anesthesia Meeting and Workshops in Vancouver, we now look forward to the Annual Pain Medicine Meeting and Workshops in Boca Raton, Florida, on November 15-18, 2007. Program Chair Nirmala Hidalgo, M.D., promises us a unique meeting with "something old" (review of chemical neurolysis) and "something new" (review and workshop on the uses of botulinum toxin for pain). Continuing in the tradition of the very popular "mock" trial at last year's Annual Pain Medicine Meeting, there will be more didactic sessions on the interaction of medicine and the legal system.

In summary, the ASRA Board of Directors hopes to continue to provide "added value" to its constituency. We are avidly seeking ideas, projects and initiatives that will provide "value" to membership.