NERVE INJURY AFTER PLEXUS AND PERIPHERAL NERVE BLOCKS FOR REGIONAL ANESTHESIA AND MEDICOLEGAL IMPLICATIONS

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STEM CASE AND KEY QUESTIONS

A 56-year-old male is scheduled for a revision rotator cuff repair surgery. He injured his right shoulder in a motor vehicle accident 4 years earlier and previously underwent a right rotator cuff repair. His medical history is significant for sleep apnea, osteoarthritis, hypertension, tobacco use, and his body mass index is 40. He also reports chronic neck pain and stiffness since his motor vehicle accident. A recent magnetic resonance imaging (MRI) scan demonstrates a supraspinatus tear. His surgeon has booked the case, requesting an interscalene block (ISB) and general anesthesia.

- How common are nerve injuries after plexus and peripheral nerve blocks (PNB)?
- What is the risk of nerve injury after arthroscopic shoulder surgery?
- Is this patient at increased risk for a perioperative nerve injury (PNI)?
- What are the risk factors for developing a PNI?

The patient tells you he received an ISB as part of his previous anesthetic and although it provided good pain relief, he felt short of breath for several hours after he woke up and prefers not to have a nerve block again; however, his surgeon is insistent that he have a nerve block as part of his procedure.

- How does this information affect your anesthetic plan?
- Should he still receive a block? Why or why not?
- Are there any regional anesthesia alternatives other than an ISB for arthroscopic shoulder surgery?
- What constitutes informed consent?
- How should this be documented?
- Is a separate anesthesia consent necessary?

The patient is moved into a block room, a peripheral intravenous line is placed, monitors are attached, and a pre-procedure timeout is called.

- What constitutes the standard of care for a regional block?
- Why is this important?
- Which elements of the block procedure should be documented in the anesthesia record?

- Is there a preferred type of needle localization guidance that will decrease the risk of intraneural injection and/or PNI?
- How common is intraneural injection during a nerve block?
- Are there any local anesthetics or adjuvants that decrease the risk of PNI?
- Does placing a catheter with a continuous infusion of local anesthetic increase the risk of developing a PNI versus a single injection?

After minimal sedation, an ultrasound-guided superior trunk block proceeds uneventfully, with 15 mL of bupivacaine 0.5% and 4 mg of perineural dexamethasone. After 90 minutes of surgery under general anesthesia, the patient is extubated and transported to the post-anesthesia care unit. He reports minimal pain at discharge. A member of the anesthesia team calls him the next day and the patient reports that he is doing well and not experiencing any breathing difficulties.

- How would you document your postoperative call?
- Why is this important?

Ten days later, during his first postoperative office visit, the patient states that his right hand is numb and that he is having difficulty using eating utensils. He also claims he first noticed this after returning home. On examination, the surgeon notes decreased sensation over the palmar thenar aspect of the hand, including digits I through III, along with decreased grip strength. The patient is also having difficulty flexing all of his fingers.

- Which peripheral nerves appear to be injured?
- Does ultrasound have a role in the initial evaluation?
- Is a PNB an independent risk factor for developing a PNI?
- What is the classification system most commonly used to describe nerve injury and prognosis, and how would you approach a differential diagnosis?

On his subsequent clinic visit (24 days postoperatively), the patient is angry and upset that his hand is still numb and weak, and he is now experiencing severe pain in his right arm and right hand. On examination, the hand is discolored and exquisitely sensitive to touch. In contrast, the shoulder appears to be normal. The surgeon refers the patient to a neurologist for further work-up.

- How likely is it that this is a block-related injury?
- What are electrodiagnostic studies (EDX) studies and what can they tell you?
- When should they be ordered?
- Is an MRI necessary?

EDX study results obtained 4 weeks after surgery are summarized in Tables 1 through 3.

Table 1 SENSORY NERVE STUDY RESULTS*

Nerve/Stim. Site:	Rec. Site	Onset ms	Peak ms	NP Amp µV	PP Amp µV	Dist cm	Vel m/s
Sensory							
Right Median: Wrist	Dig III	NR					75.7
Left median: Wrist	Dig III	1.85	2.70	67.3	113.2	14	75.7
Right Ulnar: Wrist	Dig V	2.25	2.95	55.4	73.5	14	62.2
Left Ulnar: Wrist	Dig V	2.15	2.95	66.2	71.2	14	65.1
Right Radial: Forearm	Thumb	1.5	2.00	38.5	19.2	10	66.7
Right LABCN: Elbow	Forearm	2.6	4.9	66.3	67.0	12	46.1
Right MABCN: Elbow	Forearm	2.4	4.75	65.9	71.2	12	50.0

*SNAP, sensory nerve action potentials; NP, negative peak amplitude; PP, positive peak amplitude; LABCN, lateral antebrachial cutaneous nerve; MABCN, medial antebrachial cutaneous nerve.

Table 2 COMPOUND MUSCLE ACTION POTENTIALS (CMAP) RESULTS

Nerve/Stim. Site:	Rec. Site	Latency ms	Amp mV	Normal Amp mV	Dist cm	Vel m/s	Norm Vel m/s
Motor							
Right Median:							
Wrist	APB	3.7	0.2	>5.0	7.7	–	–
Elbow	APB	7.2	0.3	>5.0	18.6	47.2	>50
Left Median:							
Wrist	APB	3.6	7.4	>5.0	7.5	–	–
Elbow	APB	7.1	7.3	>5.0	18.5	52.8	>50
Right Radial:							
Proximal	EIP	1.8	5.1	>5.0	4	–	–
Elbow	EIP	5.1	5.3	>5.0	18	54.5	>50
Right Ulnar:							
Wrist	ADM	3.1	8.6	>5.0	7.5	–	>50
Below Elbow	ADM	5.8	9.1	>5.0	17.5	64.8	–
Above Elbow	ADM	7.3	8.9	>5.0	8.0	53.3	>50

APB, abductor pollicus brevis; EIP, extensor indicis proprius; ADM, abductor digiti minimi.

Table 3 ELECTROMYOGRAPHY RESULTS

Muscle	Nerve	Root	Spontaneous Activity			MUAP*				
			Ins Act	Fib	PSW	Amp	Dur	PPP	Recruit	Int Pat
R. deltoid	Axillary	C5-6	0	0	0	N	N	None	N	Complete
R. biceps	MSCT	C5-6	0	0	0	N	N	None	N	Complete
R. triceps	Radial	C6-8	0	0	0	N	N	None	N	Complete
R. abd. pol. brev.	Median	C8-T1	2+	4+	3+	N	Inc	2+	Rapid	25%
R. pronator teres	Median	C6-7	0	0	0	N	Inc	None	N	Complete
R. 1st D inteross	Ulnar	C7-8	0	0	0	N	N	None	N	Complete
R. abd. dig. min.	Ulnar	C8-T1	0	0	0	N	N	None	N	Complete
R. flex. carpi ulnaris	Ulnar	C8-T1	+2	3+	2+	N	N	None	Rapid	50%
R. flex. dig. prof. III-IV	Ulnar	C7-8, T1	1+	4+	3+	N	N	None	Rapid	25%
Paraspinal C5-T1		C5-T1	0	0	0					

*MUAP, Motor unit action potential; Ins Act, insertional activity; Fib, fibrillation potentials; PSW, positive sharp waves; Amp, amplitude; Dur, duration; PPP, polyphasia; Int Pat, interference pattern; MSCT, musculocutaneous nerve; 1st D inteross, 1st dorsal interosseous.

- Based on the EDX results, does this appear to be a serious injury?
- Is this a neurapraxia or axonotmesis?
- How likely is it that a full recovery will occur?
- Is there any evidence of a pre-existing subclinical nerve injury?
- Can you localize the injury site in the peripheral nervous system based on these findings?
- How do the EDX study results refine the differential diagnosis?

- What kind of surgical treatment options for nerve injury are available, and when should they be considered?

You receive notification that the patient has filed a civil suit alleging malpractice on your part in performing the PNB.

- How common are malpractice lawsuits in anesthesiology?
- Does administering PNB increase your risk?
- Which blocks are more likely to cause neurologic complications?

DISCUSSION

Permanent nerve injury is rare after PNB, although some form of transient injury is common. A review of the literature examining the upper limit of postoperative neurologic symptoms (PONS) reported in all studies since 1997 indicates that up to 19% of patients may report PONS on the first postoperative day. This rapidly diminishes to 2.2% at 3 months, 0.8% at 6 months, and 0.2% at 1 year.[1-3] Permanent injury (PONS > 1 year) is rare, with estimates ranging from a high of 9:10,000 to a low of 2:10,000 after PNB.[4-9]

PNI is a multifactorial process, with a differential diagnosis that includes surgery-, patient-, and anesthesia-related factors. PNB is not deemed to be an independent risk factor for developing PONS, even in joint replacement surgeries involving the shoulder, hip, and knee.[3,7,10] Orthopedic surgical procedures are associated with an elevated risk of developing PONS, even in the absence of PNB.[3,7,11] A recent systematic review of factors associated with neurologic complications after PNB found the strongest associations with certain types of blocks (ISB, axillary, femoral, sciatic, popliteal) and the use of long, sharp bevel needles.[12]

PONS in arthroscopic shoulder surgery are reported to range between 0.1% and 10%, with most of these injuries being transient.[11] Mechanisms of injury include stretching the brachial plexus for surgical positioning and direct injury to nerves from surgical portal and anchor placements. The anterior shoulder portal may damage the lateral cord of the brachial plexus and musculocutaneous nerve, while the lateral portal can damage the axillary nerve. The suprascapular nerve can be injured secondary to mechanical compression forces exerted by anchors placed in the superior aspect of the glenoid during superior labrum repairs.[11]

Patient-specific risk factors for PNI and PONS include the co-morbidities such as diabetes, hypertension, and tobacco use, revision surgical joint procedures, and pre-existing nervous system disorders, including subclinical entrapment neuropathies (carpal tunnel), cervical spondylosis, being post-chemotherapy (cisplatin, paclitaxel, vincristine), and alcohol-related polyneuropathy.[3,13]

These conditions may induce a subclinical ischemic neuropathy that can predispose a patient to developing clinical manifestation of PONS after surgery and anesthesia if additional damaging forces are exerted on the nerve. However, a recent systematic review did not find an association between pre-existing neuropathy and the risk of developing PONS after PNB, although recovery from PONS was worse in this subset of patients.[12]

A patient with a history of osteoarthritis and a previous neck injury with pain may have an underlying subclinical injury, with possible compression of nerve roots and the spinal cord from degenerative changes in the intervertebral foramen and central spinal stenosis. This has the potential to cause further neural compromise related to surgical positioning and the administration of a PNB. Cervical spine disease may also be a risk factor for developing chronic phrenic nerve paralysis after ISB.[14]

ALTERNATIVES TO ISB FOR SHOULDER SURGERY

Alternatives to the ISB that provide non-inferior analgesia while minimizing the risk of hemidiaphragmatic paralysis include the selective anterior suprascapular nerve block and the superior trunk block. Two recent studies have compared the adverse effects of ISB versus suprascapular nerve block with 15 mL of ropivacaine 0.5% in conjunction with general anesthesia, or selective superior trunk block versus ISB as the primary anesthetic utilizing 15 mL of bupivacaine 0.5%. These two alternatives decrease the risk of hemidiaphragmatic paralysis to 10% and 4.8%, respectively.[15,16]

There are many benefits associated with PNB in patients with a history of sleep apnea and elevated body mass. In addition to superior postoperative pain control, the potential for respiratory depression can be mitigated by avoiding or minimizing opiate analgesics.

INFORMED CONSENT

Informed consent must contain the following patient and physician components:

- Voluntary
- Competency
- Capacity
- Disclosure
- Authorization
- Documentation

There is no requirement by the Centers for Medicare/Medicaid Services or The Joint Commission for a stand-alone anesthesia consent form, although local hospital policies may differ from federal requirements. The state of Texas requires a specific written anesthesia consent prior to epidural and spinal blocks, with mandated language describing risks in these forms. The American Society of Anesthesiologists recommends that its members use a separate written anesthesia consent form to mitigate malpractice risk.[17] This form should include an explanation of the type of anesthesia and monitoring being proposed and the material risks associated with these techniques, along with alternatives. The legal standard for disclosure of risk is a “reasonable person standard,” i.e., what a reasonable person would want to know to make an informed decision. This is important in the setting of regional anesthesia, where offering a PNB is always a patient choice and is never the only option.[18] The consent form should be dated and timed, with the patient’s name and signature authorizing the anesthesia plan; the name and signature of the anesthesia provider explaining the procedure and risks; and a witness signature from someone outside of the surgery–anesthesia team. For medicolegal purposes, this should be completed prior to any documentation of pre-procedural sedation.

STANDARD OF CARE, DOCUMENTATION, AND ANESTHETIC TECHNIQUE FOR PNB

The "standard of care" is a legal term broadly defined as requiring compliance with the generally recognized and accepted practices and procedures that would be followed by average competent practitioners in the physician's field of medicine under the same or similar circumstances. This definition recognizes flexibility in tailoring clinical decision-making to a variety of patient care scenarios.[19] Standard of care is not defined by clinical practice guidelines or advisories. For example, standard of care in regional anesthesia pertaining to administering a PNB would warrant informed consent prior to commencing the procedure, a medical record documenting the details of the procedure, placement of an intravenous fluid line, routine monitoring (electrocardiography, blood pressure, pulse oximetry), availability of resuscitation equipment, and not exceeding the maximal recommended dose of local anesthetics. Standard of care allows for a variety of PNB approaches and needle localization choices, block needle types, local anesthetic agents, volumes and concentrations, perineural adjuvants, and the decision to employ single-injection or continuous catheter techniques.

A block note should be sufficiently granular to ensure that the documentation would allow a third party to review and understand the details of the block. The record could be essential in establishing standard of care practice and successfully defending against a future block-related medicolegal claim. In the real world, these notes vary based on individual practice settings and preferences; nonetheless, a comprehensive block note might include some or all the following data:

- Focused neurologic examination prior to the block
- Time-out (patient and block site identified, marked, and informed consent verified)
- Patient level of awareness during block (was meaningful verbal contact maintained during the procedure?)
- Aseptic skin preparation and drape
- Type of needle used (e.g., 22-gauge short bevel, 150 mm, manufacturer name)
- Skin depth to target prior to injection (if catheter, distance inserted after placement)
- Needle localization method (ultrasound, neurostimulation, paresthesia seeking, or combination)
- Initial and minimal current threshold prior to injection, and motor response observed with neurostimulation (e.g., deltoid muscle contraction)
- Presence or absence of paresthesia or pain with needle advancement prior to injection. If paresthesia occurred, did it immediately resolve prior to injection? Describe anatomic location of paresthesia.
- Presence or absence of paresthesia with onset of injection. If present, was the injection aborted, and did the paresthesia resolve with needle withdrawal prior to second injection?
- Presence or absence of resistance to onset of injection. If resistance was present, was the needle repositioned and the injection recommenced without further resistance?

- Was opening injection pressure (OIP) monitored or was a pressure injection-limiting device used? Was the pressure <15 psi? How was the pressure measured (e.g., commercial device and name, or syringe with compressed air injection technique)?
- Test initial injection of 0.5 to 1.0 mL and absence of nerve diameter expansion (using ultrasound needle guidance)
- Negative or positive aspiration for blood prior to test injection
- Single-injection or multi-injection technique
- Local anesthetic type, concentration, and volume
- Perineural additives and dose (epinephrine, bicarbonate, dexamethasone, clonidine, buprenorphine, dexmedetomidine, preservative-free)
- Ultrasound pre- and post-injection image capture, or video loop of injection
- Block readiness for surgery (complete, partial, unable to assess, failure)
- Block supplementation [yes, no, local anesthetic concentration and volume, block supplementation site (e.g. axillary)]

The electronic medical record can create a customized PNB template with these data fields pre-populated and selected via check boxes, with the opportunity to input numerical values or free text entries where appropriate. A comprehensive block note can be quickly created with the help of an assistant entering information in real time.

A record of an inpatient follow-up visit, or phone call for an outpatient, should also be recorded in the medical record. Data collection can include the length of analgesia, the time of return to normal sensation and motor function, the presence and location of any sensory or motor deficits, the presence of paresthesia or dysesthesia, and patient satisfaction. When a continuous catheter is used, data collection can include the time of catheter removal and condition of the skin at the entry site to assess for inflammation or infection. If there is a persistent sensory or motor deficit, the patient should be called daily until the deficit resolves or the patient is assessed in clinic.

Almost all upper extremity ultrasound-guided PNB can be performed with single-injection techniques; there is no advantage to multi-injection techniques in terms of block success rates, and the risk of procedural paresthesia increases with multiple injections.[20] In contrast, some lower extremity ultrasound-guided nerve blocks have demonstrated higher success rates with multi-needle pass injection techniques.[21]

NEEDLE–NERVE LOCALIZATION AND AVOIDING INTRANEURAL INJECTION

Although there are many advantages to ultrasound needle guidance, there is no difference in the incidence of PONS compared to other needle localization techniques.[2,4,22] Ultrasound probes do not have the lateral resolution to distinguish intraneural–intrafascicular needle placement from intraneural–extrafascicular needle placement[3,4,13,23] (Figure 1). Additionally, regional anesthesiologists do not always visualize their needle tip. Given these limitations, there may be a role for the selective use of additional

needle localization monitoring technology, including neurostimulation and injection pressure monitoring.

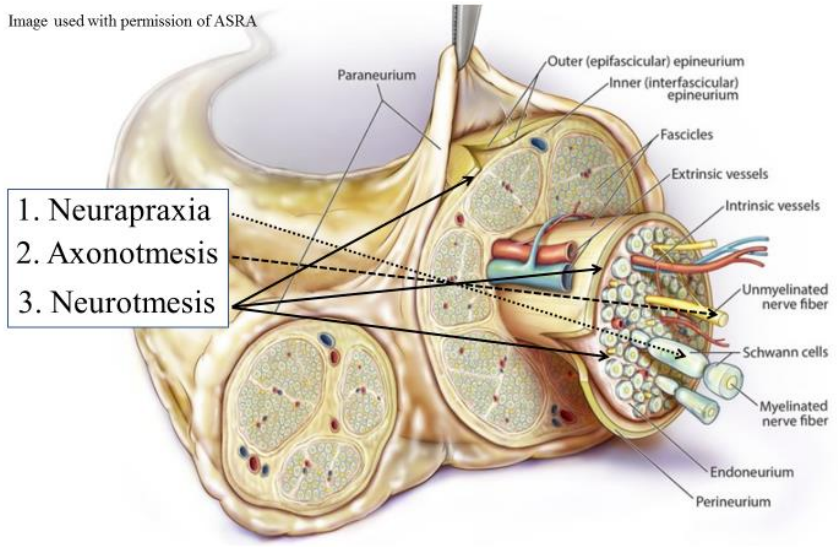

Figure 1. Peripheral nerve microanatomy and Seddon nerve injury classification.

Inadvertent intraneural needle entry and injection occur more commonly than previously appreciated – in up to 17% of PNB that use ultrasound.[24,25] In Hara et al. and Sala Blanch et al., none of the patients that sustained intraneural injections developed PONS. Using neurostimulation needle localization techniques, the incidence is even higher, with an intraneural injection rate of 75% when an evoked motor response (EMR) is elicited at 0.5 mA.[26] This is not surprising when other investigators have determined the sensitivity of needle–nerve contact associated with neurostimulation at 0.5 mA in the axillary brachial plexus to be only 74.5%.[27] In the same study, the sensitivity of paresthesia with needle–nerve contact was only 38.2%. This helps us interpret other evidence demonstrating that the elicitation of a paresthesia is not an independent risk factor to develop PONS and that PONS can occur without the presence of a paresthesia.

Forced needle entry into neural tissue triggers an inflammatory response, even without injection, although these inflammatory changes resolve without functional injury.[28] Intraneural injection of as little as 0.5 mL (85% sensitivity) can be detected by knowledgeable regional anesthesiologists with ultrasound experience.[29] Routinely using a test dose of 0.5 to 1.0 mL of local anesthetic can limit the volume of an inadvertent intraneural injection and allow time for needle repositioning prior to injecting larger volumes.

Several investigators have demonstrated that successful ISB can be performed in human patients without needle entry into the interscalene groove by depositing local anesthetic adjacent to the plexus and not between the nerve roots.[30,31] The maximum effective distance away from the plexus to deposit local anesthetic and provide block success in 95% of patients has been calculated to be 1.6 mm.[31]

Animal and human models demonstrate that neurostimulation is a specific but non-sensitive measure of needle–nerve contact. With needle–nerve contact or intraneural needle placement in animal models, EMR can occur with a stimulating current as low as 0.2 mA; however, it may not occur unless the

stimulating current is increased up to 1.8 mA.[32] The presence of an EMR at low stimulating currents <0.5 mA can only occur when the needle tip is very close to the nerve, in contact with the nerve, or inside the nerve.[3]

When neurostimulation is used as the primary needle localization method, the initial current may be set at 1.1 mA, and no attempt should be made to advance the needle, seeking an EMR below 0.5 mA. Neurostimulation can be an adjunct to ultrasound-guided needle localization in the setting of poor image quality and difficulty visualizing the needle tip. If an EMR is still present at <0.5 mA, the needle should be withdrawn until the EMR disappears prior to injecting a test dose of local anesthetic.

In contrast to neurostimulation, OIP monitoring is sensitive for needle–nerve contact and intraneural placement, but it is not specific. Contact with fascia, ligaments, cartilage, and bone can also elevate OIP. Several investigations in animal models link elevated OIP (>15 psi) to histologic and functional injury; other studies demonstrate histologic injury but not functional injury with OIP < 15 psi despite evidence of intraneural injection as assessed by ultrasound.[33,34] Human models demonstrate elevated OIP with needle–nerve contact against the roots of the brachial plexus and the femoral nerve, as well as contact with the fascia iliaca.[35,36] A cadaver study has demonstrated elevated OIP with needle–nerve contact or intraneural placement in all lower extremity nerves. In contrast, extraneural needle placement was consistently associated with an OIP < 15 psi.[37]

OIP monitoring is not used as a primary needle localization technology in clinical practice; however, it can supplement ultrasound and neurostimulation guidance. Animal data suggest the major benefit of OIP monitoring lies in confirming extraneural (or intraneural but extrafascicular) placement, as no functional injuries haver been observed in animal models with low OIP. However, there are limited human data that confirm or refute the effectiveness of OIP in limiting PONS.[3,12,38]

Commercial pressure-monitoring devices can be inserted in the injection line to measure or limit pressure to <15 psi. The subjective feel of pressing on the syringe plunger and assessing OIP is unreliable.[39] A compressed air injection technique can be used to accurately assess OIP without having to purchase a commercial device.[40]

Other modalities to reduce the risk of needle–nerve contact and intraneural injection that are under investigation and development include electrical impedance monitoring and electromagnetic needle tracking guides that display current and projected future needle positions.[41]

In summary, although intraneural injection (as defined by the visible enlargement of nerve diameter under ultrasound imaging) occurs more commonly than previously appreciated and does not invariably lead to functional nerve injury, the practice of intentionally seeking intraneural injection is not recommended.[3,12,42] Targeted intracluster injections in the supraclavicular brachial plexus using India ink in cadavers remained subperineural in 24% of observed injections.[43] Recent studies by Cappelleri et al. involving intentional intraneural injection (defined as increase in nerve diameter during injection as visualized by ultrasound) of the popliteal nerve in humans supports the animal evidence.[38] Injection pressures were limited to <15 psi, implying intraneural–extrafascicular injection. Although no functional deficits were observed at 5 weeks and 6 months postinjection, the results of more sensitive EDX testing demonstrated a 40% to 50% reduction in compound motor action potentials (CMAP) at 6 months, implying significant axonal damage. Accompanying editorials to these publications reiterated the advice

that intentional intraneural injection should be avoided and not encouraged, as even small volumes of intraneural local anesthetics can cause long-term axonal damage.[44-47]

CONTINUOUS CATHETER TECHNIQUES, PERINEURAL ADJUVANTS, AND NERVE INJURY

There is limited evidence comparing the risk of developing PONS after single-injection PNB to continuous catheter PNB, and a systematic review did not demonstrate any difference.[12] The most comprehensive data using ultrasound-guided procedures exist in the Dartmouth clinical registry, in a subset containing 2,0003 single-injection ISB and 230 continuous catheter ISB blocks that did not show a statistical difference in the risk of developing long-term PONS between the two techniques, although there was a trend for a higher injury rate in patients with continuous catheters.[8]

All commonly used perineural adjuvants (dexamethasone, dexmedetomidine, clonidine, buprenorphine) demonstrate minimal neurotoxicity signals when tested in vitro under simulated clinical concentrations.[48] These adjuvants exhibit substantially lower neurotoxicity compared to ropivacaine. In other animal models, intrafascicular injection of dexamethasone did not injure axons, and intraneural injection of dexmedetomidine with ropivacaine demonstrates anti-inflammatory and neuroprotective effects.[44,49] Clinical data from the Dartmouth registry and a more recent case series did not show any statistically significant association between using adjuvants and developing PONS.[8,50]

All local anesthetics reduce neural blood flow, demonstrating a dose-dependent and concentration effect. The reduction in neural blood flow is greater with epinephrine than with local anesthetics, although the clinical consequences associated with perineural epinephrine are controversial.[51]

NERVE INJURY AND CLASSIFICATION

The fundamental mechanism of nerve injury is an ischemic injury to a metabolically active tissue, resulting in a loss of signaling function of the axons within the peripheral nerve. Mechanisms of injury can be attributed to mechanical, vascular, chemical, thermal, and/or inflammatory factors. Due to the multifactorial nature of these injuries (patient-, surgery-, and/or anesthesia-related), it can be difficult to determine causation, although imaging and EDX studies can be helpful in refining the differential diagnoses, as well as the prognosis for recovery.

All local anesthetics exhibit concentration-dependent cytotoxic, neurotoxic, and myotoxic effects in vitro. However, in vivo, connective tissue barriers between the axon and extraneural tissue where local anesthetics are deposited progressively dilute the concentration of local anesthetic to non-toxic levels before the local anesthetic reaches the axon. Most of the nerve diameter expansion visualized on ultrasound, when block needles inadvertently enter nerves, can be attributed to intraneural–extrafascicular injection. However, needle entry into the epineurium and subepineurium can damage the vasa nervorum (providing approximately 50% of neural blood flow) and lacerate blood vessels in the space between nerve fascicles, causing intraneural hematoma and compressive ischemic injury[11] (Figure 1).

The Seddon classification system of nerve injury is a structural, functional, and prognostic description of peripheral nerve injury (Figure 1). Three categories of injury are described: neurapraxia, axonotmesis, and neurotmesis.[52] Neurapraxia is the most commonly observed PNI, characterized by damage to the myelin layer around axons and resulting in the slowing of nerve conduction velocity. Although

neurapraxia can result in severe motor and sensory impairment, the natural course is for complete recovery within weeks to months as the myelin layer spontaneously regenerates inside the intact endoneurial tube.

Axonotmesis is a more serious injury, with damage to the axon that results in Wallerian degeneration. Although the axon has the potential to regenerate within the endoneurial tube, complete recovery does not always occur, and prognosis is guarded. Time to recovery is measured in months or years. Once regeneration begins, axons will extend at the rate of 1 mm per day, or 1 inch per month.

Neurotmesis is the most serious injury, with damage to the axon and surrounding connective tissue. The least serious subcategory is damage limited to the axon and endoneurium, with potential to reestablish axonal continuity over months to years. More serious subcategories include disruption of the perineurium and epineurium that requires surgical intervention to restore continuity to these connective tissue layers.

EVALUATION AND TREATMENT OF PONS

A neurology consult should be obtained by any patient with significant motor loss that persists beyond the expected duration of the block. Early recognition and documentation of PONS is imperative to help establish causation and to diagnose and treat reversible lesions such as compressive hematoma.[53] A detailed examination of the plexus and peripheral nerves using ultrasound to scan along the length of the structure can assess for hematoma, discontinuity, and intraneural edema.[54] MRI can also reveal the presence of hematoma, discontinuity lesions, and evidence of inflammation and edema with hyperintense signals (Figure 2). Although MRI can detect inflammatory changes, these changes are not specific enough to determine causation. Interpretation requires clinical correlation with the patient history and physical, details of the surgical procedure, and other diagnostic modalities such as EDX studies. EDX studies can localize the site of injury in the peripheral nervous system and distinguish between demyelinating and axonal injury.

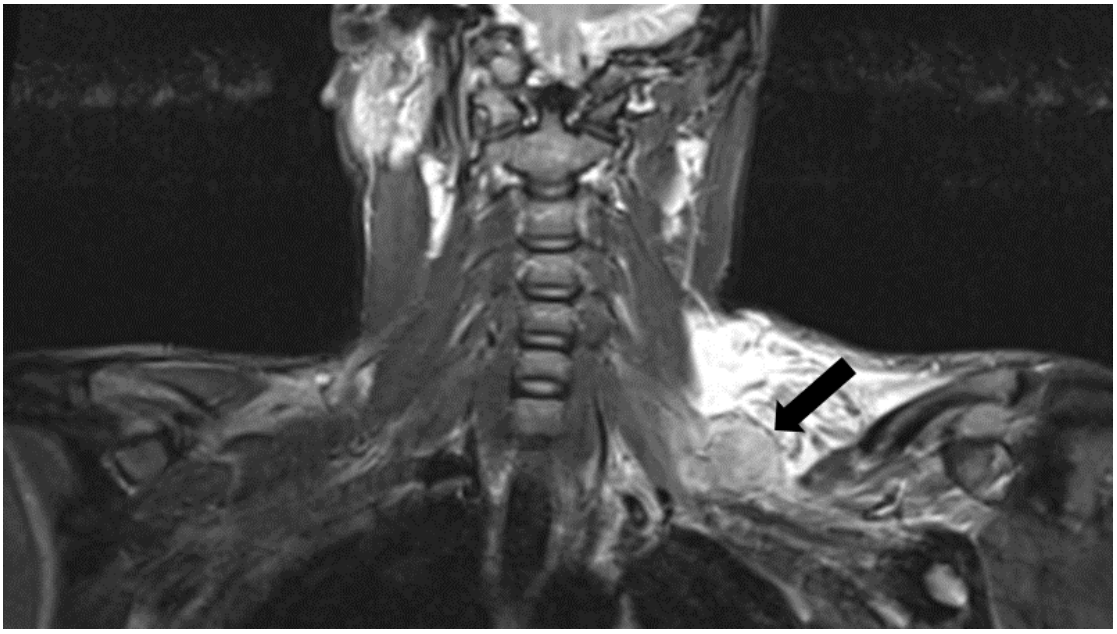

Figure 2. MRI image of brachial plexus hematoma (arrow) with inflammatory changes (bright signal).

Nerve conduction studies (NCS) are subdivided into motor and sensory nerve studies (Figure 3). CMAP are measurements taken in motor nerves, with a proximal supramaximal stimulus of the nerve and recording electrodes placed over a distal target muscle that records electrical signals generated as myofibrils depolarize. In contrast, sensory nerve action potentials (SNAP) are measurements taken in sensory nerves, with a proximal supramaximal stimulus of the nerve and recording electrodes placed over a distal part of the nerve that record electrical signals as the nerve depolarizes.

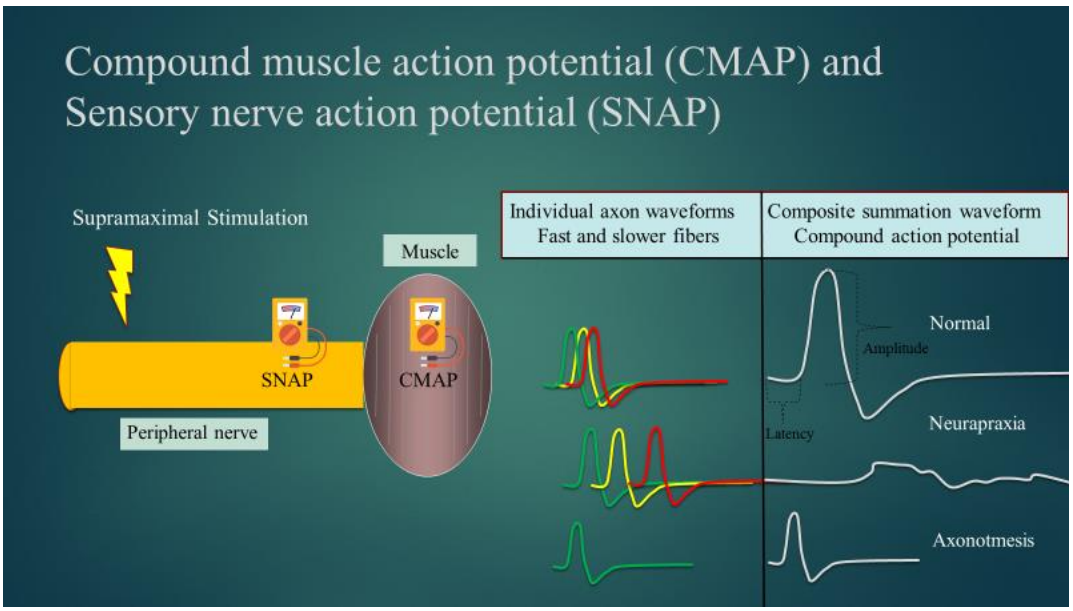

Figure 3. Example of nerve conduction study demonstrating features of a normal examination, neurapraxia (demyelination), and axonotmesis (axonal injury). Composite waveform is the algebraic sum of positive and negative wave amplitudes along the time axis.

The hallmark of a focal demyelinating injury (neurapraxia) is a reduction in conduction velocity, increase in latency, and decrease in amplitude. Conduction velocities are <75% of normal values and latencies are >130% of normal.[54] In contrast, axonal injuries (axonotmesis) appear as a reduction in amplitude without the associated decrease in conduction velocities and increases in latency. No signals are recorded with neurotmetic injuries.

NCS are not always helpful in distinguishing block-related from non-block-related causes. Most PNB are located at proximal sites in the upper and lower extremities, whereas NCS are located at distal sites in the extremities. Focal demyelinating injuries at proximal locations related to PNB will not be detected by stimulating distal to the site of injury because measured conduction velocities will be normal. It is technically difficult to stimulate proximal to the axilla, as nerves move away from the surface into deeper tissue and supramaximal stimulation is no longer possible. The opposite situation holds true with proximal axonal injuries: any stimulation of the nerve distal to the injury site will demonstrate abnormal signal amplitude. The site of injury cannot be determined by progressively “marching up” distal to proximal unless CMAP is measured in a new muscle that is proximal to the site of injury. In practice, this

is done by using needle electromyography (EMG) instead of CMAP or SNAP, and sampling proximal muscles, looking for pathologic denervation potentials and abnormal motor unit action potential (MUAP) activity.[55,56]

The presence of denervation potentials imply injury to nerve axons at or proximal to the branch point of the most proximal muscles with denervation potentials. EMG studies also assess the number and type of MUAP and measure recruitment and activation of motor units.[55,56]

Morphologic changes in MUAP over time are used to follow the presence of reinnervation after axonal injury. With moderate injury, normal motor units adjacent to injured motor units will send out collateral neural branches to innervate adjacent muscle fibers denervated by damaged axons. This will increase the size of the motor unit (number of fibers innervated by a given axon) and increase the duration and number of phases of the MUAP.[55,56]

EDX studies are typically obtained 3 to 4 weeks after injury when the most information is obtainable from a single study.[52,53,55,56] Pathologic denervation potentials associated with axonal injury are first detected in muscle 3 to 4 weeks after injury, allowing demyelinating injuries to be distinguished from axonal injuries. With evidence of moderate to severe axonal injury, EDX studies can be repeated at 3 and 6 months to assess the presence and degree of reinnervation, with referral to a reconstructive peripheral nerve surgeon for injuries not continuing to improve.[52,57]

INTERPRETATION OF EDX STUDY RESULTS

EDX study results obtained 4 weeks postinjury can be seen in Tables 1 through 3. They should be interpreted in context of the clinical examination that demonstrated injury to the median and ulnar nerves in the hand.

As illustrated in Table 1, no sensory nerve signal was obtained in the right median nerve supplying the abductor pollicus brevis (APB). This part of the median nerve originates in the medial cord, which contains the C8-T1 nerve roots. All other sensory nerve distributions displayed normal amplitudes and conduction velocities. The other medial cord-derived sensory nerve, the medial antebrachial cutaneous nerve (MABCN), was normal. This implies that the injury is distal to the takeoff of the MABCN from the medial cord. The type, location, and acuity of this injury cannot be determined from SNAP information alone.

Motor nerve studies illustrated in Table 2 provide further information about the type and severity of the injury. Conduction velocities for all nerves studied were within normal limits; however, the amplitude recorded in the right APB muscle was only 4% of the normal value. This signifies axonal injury, or axonotmesis. The percentage reduction in normal amplitude is a proxy for the number of axons injured within the nerve, and this represents a severe injury, with most of the axons being damaged. No additional information is available from this study to pinpoint the anatomic location of the median nerve injury.

EMG studies presented in Table 3 confirm axonal injury in the distribution of the median nerve branch supplying the right APB, with the presence of denervation potentials (0–4+ scale) in this muscle. These fibrillation potentials and positive sharp waves are not normally seen until 3 to 4 weeks after injury. There is also evidence of pre-existing chronic injury, as demonstrated by the increased duration of

MUAP and polyphasia in the APB. These signs of reinnervation do not occur until several months after injury, implying that there may have been some type of pre-existing subclinical injury prior to the surgical procedure and nerve block. The EMG also demonstrates evidence of axonal injury in the branches of the ulnar nerve not tested in the motor examination. Selective injury in branches of the ulnar nerve derived from the C8-T1 roots of the medial cord, along with injury to medial cord-derived branches of the median nerve, are most likely localized to the forearm. Normal paraspinal EMG recordings indicate no pathology at the C5-T1 nerve root level, and muscles innervated with nerves derived from superior and middle trunk nerve roots (axillary, musculocutaneous, and radial) are all normal, excluding superior or middle trunk pathology. Denervation potentials in the flexor digitorum profundus muscle (which includes the C7 nerve root passing through the middle trunk) are most likely attributed to a distal injury, given that other muscles supplied from the lateral and posterior cord containing the C7 nerve root (pronator teres, triceps) are normal.

EDX study results confirm that the superior trunk nerve block was unlikely to have contributed to this patient's injury. Block-related axonal damage at the level of the superior trunk would manifest with denervation potentials in the deltoid, supraspinatus, and biceps muscle – with possible extension into middle trunk-innervated structures such as the pronator teres and triceps muscles. Arthroscopic shoulder surgery would most likely cause injuries to the axillary, musculocutaneous, and suprascapular nerves or diffuse brachial plexus injuries involving all trunks and cords secondary to a stretch injury associated with positioning.

The most likely explanation for this injury is a post-surgical inflammatory neuropathy (PSIN). These injuries can be multifocal and are often characterized by intense pain after a block wears off, followed by unexplained muscle weakness in multiple nerve locations. PSIN is underdiagnosed and frequently not considered in the differential diagnosis of PONS, with causation often erroneously attributed to PNB.[3,53,58]

SURGICAL TREATMENT OPTIONS

Peripheral nerve reconstruction surgery should be considered for any persistent serious motor nerve injury on clinical examination that affects shoulder, elbow, or hand function and minimal evidence of reinnervation on EDX studies at 6 months.[52,57] Irreversible functional changes in the neuromuscular junction and muscle of affected tissues will occur beyond 1 year after denervation, limiting the effectiveness of delayed surgical reconstructive surgery restoring axonal continuity to target tissue. The emphasis has shifted from interposition autologous grafts to nerve transfer options.[57]

NEGLIGENCE AND MEDICAL MALPRACTICE RELATED TO PNB

Compared to other physician specialties, anesthesiologists face slightly below average risk of being named in a malpractice lawsuit.[59] With respect to anesthesiology-related claims, nerve injury is the second most common category of claim filed; however, most of these claims are associated with general anesthesia and neuraxial anesthesia, with only 2% of all anesthesia claims related to PNB.[60] ISB was the most frequently represented PNB in claims databases, although only 3.5% of these claims resulted in a payment.[61] Femoral and popliteal nerve blocks were the 2nd and 3rd most common PNB associated with claims in a recent analysis, with 50% of popliteal sciatic nerve block claims resulting in a payment.[61]

CONCLUSIONS

- PONS or PNI are frequently observed after PNB and orthopedic surgery.
- The incidence of long-term PONS following ultrasound-guided PNB is estimated to range between 2:10,000 and 9:10,000, which is not appreciably different from nerve stimulation needle localization techniques.
- Many instances of PNI are falsely attributed to PNB, and clinicians fail to consider other surgery- and patient-related causes, including inherent risks associated with orthopedic surgery and patient-related conditions, that predispose to subclinical peripheral neuropathy.
- PSIN may be responsible for many cases of PONS that have been incorrectly attributed to PNB.
- A separate anesthesia consent form and a comprehensive block note can establish standard of care practice for PNB and are encouraged for diagnostic and medicolegal reasons.
- Any serious motor nerve injury warrants a neurology consultation followed by either MRI or EDX studies. Together, these studies usually localize the site and severity of injury, although clinical correlation is required to assess causation.
- PNB blocks at the highest risk for a claim include ISB, femoral, sciatic, and popliteal sciatic.

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