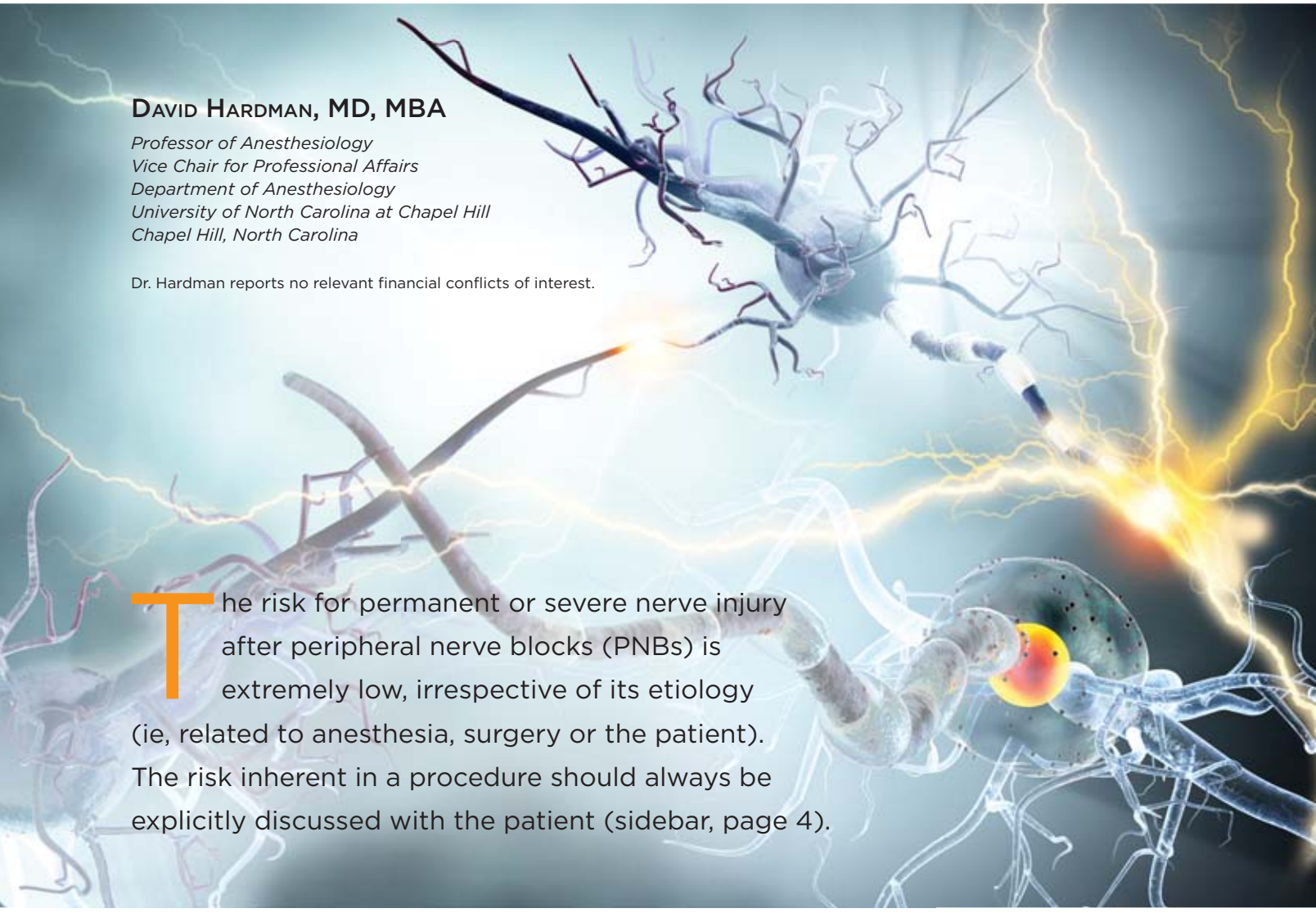
Nerve Injury After Peripheral Nerve Block:

Best Practices and Medical-Legal Protection Strategies



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Dr. Hardman reports no relevant financial conflicts of interest.

The risk for permanent or severe nerve injury after peripheral nerve blocks (PNBs) is extremely low, irrespective of its etiology (ie, related to anesthesia, surgery or the patient). The risk inherent in a procedure should always be explicitly discussed with the patient (sidebar, page 4).

In fact, it may be better to define this phenomenon as postoperative neurologic symptoms (PONS) or perioperative nerve injuries (PNI) in order to help standardize terminology. Permanent injury rates, as defined by a neurologic abnormality present at or beyond 12 months after the procedure, have consistently ranged from 0.029% to 0.2%, although the results of a recent multicenter Web-based survey in France, in which ultrasound-guided axillary blocks were used, demonstrated a very low nerve injury rate of 0.0037% at hospital discharge.[1-7]

A 2009 prospective case series involving more than 7,000 PNBs, conducted in Australia and New Zealand, demonstrated that when a postoperative neurologic symptom was diagnosed, it was 9 times more likely to be due to a non–anesthesia-related cause than a nerve

block-related cause.[6] On the other hand, it is well documented in the orthopedic and anesthesia literature that there is an alarmingly high incidence of temporary postoperative neurologic symptoms after arthroscopic shoulder surgery, both with and without regional blocks. Most of these involve minor sensory paresthesias and dysesthesias, but they can range as high as 16% to 30% in the first week postoperatively.[1,8,9]

The PNI rate associated with total shoulder arthroplasty has been previously reported to be 4% under general anesthesia alone, and represents the underlying independent surgical risk.[10] Despite advances in surgical techniques, this number has not changed appreciably over time.

The most recent data from a clinical registry at Mayo Clinic, for 1993 to 2007, demonstrated a PNI rate of 3.7% during general anesthesia.[11] This contrasts with a PNI rate of 1.7% in patients who received a single-injection interscalene block (ISB). Patients who received an ISB had significantly reduced odds for PNI (odds ratio, 0.47).[11] Factors not associated with an increased risk for PNI in this study included patient sex and longer operative time.

Over 97% of patients who developed PNI eventually recovered completely or partially at 2.5 years after the procedure, and 71% experienced full recovery. Notably, there was no difference in overall recovery from PNI between patients who received ISB and those who received general anesthesia alone.[11]

Not all surgical procedures have the same incidence of PNI, and this variation may be due to procedure-specific risk for nerve injury, apart from the use of peripheral nerve blockade and regional anesthesia. Data from three clinical registries at a single institution demonstrated a PNI incidence of 2.2% after total shoulder arthroplasty, 0.79% after total knee arthroplasty and 0.72% after total hip arthroplasty (Figure).[11-13]

The use of regional anesthesia was not an independent risk factor for PNI in any of these procedures; in fact, it reduced the risk for PNI in total shoulder arthroplasties.

Arthroscopic shoulder surgery ± regional block, 7 days postoperatively:

16%-30%

Total shoulder arthroplasty:

2.2%

Single-injection interscalene block:

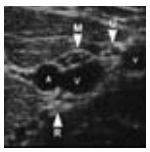
1.7%

Total knee arthroplasty:

0.79%

Total hip arthroplasty:

0.72%



Ultrasound-guided axillary blocks, at discharge:

0.0037%

Figure. Rates of perioperative nerve injuries following each type of procedure.

Strategies To Reduce Medical-Legal Risk

Before initiating a block, and particularly in a patient with previous injuries, I recommend that you take a focused history for the presence of current or previous paresthesias, dysesthesias, or pain in the limb that will receive the block. It would also be helpful to do a quick, focused sensory and motor neurologic exam. Many of these patients have preexisting lesions; unfortunately, they are not noticed until the postoperative period, when we become much more observant of abnormalities.

Be careful with the administration of sedatives during the block procedure in order to not obscure any symptoms of paresthesia, dysesthesia, or pain during injection.[14] Refer to the American Society of Regional Anesthesia and Pain Medicine (ASRA) Practice Advisory on Complications in Regional Anesthesia.[15] Be advised that a favorite tactic of medical malpractice attorneys is to argue that patients given any amount of sedation would be unlikely to be able to report pain or paresthesia on injection.

I would recommend that you document in the chart that meaningful verbal communication with the patient was maintained throughout the block procedure.

Documentation of blocks is essential for clinical care, regulatory, billing, and medical-legal reasons. ASRA has published a recommended PNB note template.[16] My experience reviewing cases for potential medical-legal problems has shown me that many of the block notes are poorly documented.

This is an area that can be rectified with the introduction of an electronic anesthesia medical record, which can allow you to create custom templates for every type of block you perform, and document detailed

information pertaining to the block. Table 1 shows an example of a block form.

Patients discharged home after a PNB procedure should receive written instructions with precautions about how to take care of an insensate extremity, and how to prevent injury. Patients with a single-injection block should be called the next day and questioned about complete block resolution or persistent symptoms, and this contact should be documented until the symptoms resolve. Any patient with persistent motor weakness beyond the normal expected recovery time should be seen in clinic immediately, for examination and potential neurologic consultation.

You should be particularly vigilant when dealing with a patient returning for a second surgical procedure and block within an intervening short interval, for example, 3 months or less. Nerve injury can exist with subclinical symptoms, and a second insult, either distal or proximal, without necessarily having anything to do with your nerve block, can elicit clinical findings postoperatively. This phenomenon is known as the double-crush theory of nerve injury.[17]

Is There Anything We Can Do To Prevent Nerve Injury?

Ultrasound-guided techniques have been shown to have many advantages, including shorter procedure time, faster block onset, lower drug volume, fewer vascular punctures and, most recently, a reduction in the incidence of local anesthetic systemic toxicity (relative risk reduction, 65%).[4,18-20] Although many benefits are associated with ultrasound-guided blocks, there is insufficient evidence to demonstrate a lower neurologic complication rate with this technique.[21,22] For that matter, there is no evidence to show fewer neurologic complications associated with neurostimulation techniques versus paresthesia-seeking techniques.[23]

Many publications call into question the sensitivity and specificity of nerve stimulation techniques, and studies demonstrate that intraneural injections (defined as cross-sectional expansion in diameter of a nerve, but not necessarily intrafascicular) as observed using ultrasound occur frequently and do not invariably lead to nerve injury, during both supraclavicular and axillary blocks.[24]

Accidental intraneural injections (defined as cross-sectional expansion in the diameter of a nerve) have also been shown to occur during ultrasound-guided blocks (without paresthesias) in about 17% of upper- and lower-extremity blocks, in 2 case series without neurologic complications, even in the hands of experienced regional anesthesiologists.[25,26]

There has been an ongoing debate about whether or not these intraneural injections are preventable, whether they are subepineural or below a connective tissue outer wrapper outside the epineurium[27] (ie, subparaneural), and whether or not they invariably lead to harm. Because of the limited resolution of current ultrasound probe technology, combined with the fact that it is challenging to keep the tip of the needle visualized in the plane of the ultrasound beam at all times, it is difficult to distinguish between a subfascial, subepineural, or intrafascicular injection.[28]

Even exceptionally well-trained experts in regional anesthesia have subsequently realized that they may have contributed to a PNI after reviewing video clips of an interscalene block demonstrating intraneural injection, despite an uneventful block procedure without pain or paresthesia.[29]

Current thinking is geared to depositing local anesthesia farther away from the nerves, rather than around the nerves in the interscalene brachial plexus region.[30] We should consider thinking about the maximum effective distance from the plexus that will still result in an effective block,[31] with a paraplexus approach rather than an intraplexus approach. A conservative technique would involve using a hydrodissection approach with needle advancement, along with a nerve stimulator (no data support this) and a lower anesthetic mass and volume.[32]

Table 1. A Form Template for Describing a Block

An example of a block form might include the following items:
Focused neurologic exam prior to block
Time-out (patient and block site identified and marked, informed consent verified)
Patient level of awareness during block
Aseptic skin prep, drape
Type of needle used, depth to target prior to injection, and if catheter, depth at skin
Ultrasound and/or nerve stimulator, with minimum threshold current
Presence or absence of paresthesia or pain. If paresthesia, did it immediately resolve?
Presence or absence of resistance to injection. Was pressure monitored? If resistance, was the needle repositioned?
Negative or positive aspiration for blood
Local anesthetic, with concentration and volume
Additives (perineural, IV, intramuscular), including total dose and preservative-free documentation
Success of block (complete, partial, not yet assessable, failed)
Block supplementation (yes or no)
Ultrasound pre- and post-injection image capture and storage

Informed Consent and Medical Negligence (Malpractice)

Although anesthesiologists may be eager to tout the benefits of peripheral nerve blocks (PNBs), many of us are not doing a very good job of disclosing the potentially catastrophic risks of these procedures to our patients.

A 2007 survey of academic regional anesthesiologists indicated that most of the respondents disclosed the minor risks for bruising, pain, and mild temporary neurologic symptoms such as paresthesias and dysesthesias, but almost 40% did not disclose the risks for local anesthetic systemic toxicity (ie, seizure and cardiac arrest) or long-term and disabling neurologic injury.[38] At the same time, a recent international survey measuring patient satisfaction after peripheral nerve blockade affirmed that 90% of the respondents were satisfied or completely satisfied with the information provided about the nerve block, as well as the patient–anesthesiologist interaction.[39]

A shared decision-making approach when discussing a PNB procedure with a patient is a good idea, given the fact that the benefits of the block are short-term (for example, reduced pain and nausea as well as earlier readiness to discharge), without the accompanying long-term benefits such as improved functional outcomes.

Informed consent for a procedure involves 4 aspects:

1. A state of voluntariness
2. Competency and capacity for decision making
3. Disclosure of information about the procedure and risks associated with that procedure
4. Authorization by the patient to undergo the procedure

Disclosure of information about risk should include procedure-specific risk, as well as patient-related relative risk. Patients should always be informed of alternative treatment options, and the entire discussion should be documented in the medical record. There is a trend to have an anesthesia consent that is separate from the surgical consent (although this is not required by regulatory agencies), and recent publications question whether or not a patient who is competent to sign a surgical consent has the same competency and capability to understand an anesthesia consent.[40]

My practice is to circle the words "nerve injury" on a paper consent form and initial it, to document that I specifically discussed this with the patient, as well as to sign, date and specify the time. Informed consent is a conversation with the patient, and much more than merely obtaining his or her signature on a form.

Lack of informed consent is a frequent allegation made by patients who have been injured, but it is usually successfully defended. Unfortunately, poor expectation management can set the litigation process in motion, and root cause analysis frequently demonstrates that patients and their families did not know a bad outcome could occur, which led to negative emotions, triggering a desire to sue. Fortunately, only a small minority of the claims in the American Society for Anesthesiologists' Closed Claims project are based on informed consent issues.[41]

Medical negligence (malpractice) is ultimately determined in civil court and covered under tort law. It must be established that:

1. You had an obligation to take care of a patient (ie, duty),
2. You practiced below the local medical community standard of care (ie, breach of duty),
3. This breach of duty resulted in the injury (ie, proximate cause) and
4. The injury was significant enough that the patient is entitled to recover damages commensurate with the injury.[42]

What this boils down to with respect to regional anesthesia cases is proving that you did not provide prudent care to prevent an avoidable intraneural injection, or proper positioning and padding to prevent a positioning-related peripheral nerve injury, and that failure to provide this prudent care was the direct cause of the injury. This is an extremely high hurdle to overcome and, consequently, most of these cases will never go to trial, although they are a nuisance and time-consuming to defend.

On the other hand, if it is established that informed consent did not occur, this may be sufficient to prove negligence without having to demonstrate breach of duty or proximate cause; hence, the paramount importance of documenting informed consent in the medical record.

Using a test injection of as little as 0.5 mL of local anesthetic solution has been shown to be a sensitive indicator of potential intraneural needle placement, as evidenced by an increase in intraneural diameter under ultrasound.[33] This may provide you with an opportunity to withdraw the needle to an extraneural position prior to injecting the remaining dose of local anesthetic solution.

Injection-pressure monitoring is a new modality, and has been recently demonstrated to have a sensitivity of 97% for detecting needle-nerve contact at the roots of the brachial plexus, with opening pressures greater than 15 psi.[34] Presently, the major value of injection pressure monitoring may be in its negative predictive value, with low opening pressures as a marker to exclude either needle-nerve contact at the epineurium or subepineural needle placement at a location that could lead to nerve injury prior to injection.[35]

Although the presence of a catheter might seem to be inherently more likely to cause nerve injury than a single injection, multiple large series, case studies, and a meta-analysis have not shown this to be the case.[1,36,37]

The rationale for using adjuvants is to improve the quality, duration, or safety of the block. With continuous infusions for PNB catheters, there is no indication for using adjuvants other than perhaps when rebolusing a catheter after a secondary block failure, and adding epinephrine as a marker for intravascular injection.

Epinephrine, in concentrations of 1:200,000 to 1:400,000, has been used as a marker for intravascular injection in non–β-blocked patients in order to prevent delivering a full dose of local anesthetic and potentially prevent local anesthetic systemic toxicity (LAST). Solutions containing epinephrine have also been used to decrease systemic levels of local anesthetics via vasoconstriction and minimizing local absorption, and hence also increase duration of action, particularly with intermediate-duration local anesthetics such as mepivacaine and lidocaine.

Interestingly, the studies demonstrating a reduction in LAST with the use of ultrasound were performed in patient populations where the majority did not receive local anesthetic injections containing epinephrine.[3,4,18] There is concern that when local anesthetic solutions with epinephrine are used in diabetic animal models, there is an increase in neurotoxicity.[43] Case series in diabetic humans receiving epinephrine in local anesthetic solutions also show excessively prolonged block duration; hence, a conservative approach in diabetic patients may be to avoid epinephrine altogether, especially in large-diameter nerves such as the sciatic nerve.

Other commonly used adjuvants to enhance block quality and extend duration, without necessitating the use of continuous catheters, include buprenorphine, clonidine, dexmedetomidine, and dexamethasone.[44] These are all off-label indications. When evaluating adjuvants, it is important to distinguish between systemic and perineural effects, while also appreciating the potential for perineural toxicity.[45]

Buprenorphine, clonidine, and dexmedetomidine[46] appear to have direct perineural effects without perineural toxicity[45] when used in normal clinical doses in preservative-free solutions, and have been shown to increase the duration of PNBs. Dexmedetomidine may even have neuroprotective effects in animal models of nerve injury.[46]

Dexamethasone has become an increasingly popular adjuvant, as studies have shown that it enhances the duration of ropivacaine blocks in the upper and lower extremity by a factor of 1.9, when given in doses of 8 to 10 mg perineurally.[47,48] However, this effect is also present when the drug is administered systemically (IV or intramuscular) instead of perineurally.[47,48]

Liposomal bupivacaine (Exparel, Pacira) is an extended-release form of bupivacaine, and is approved for use to provide analgesia at the surgical incision site via direct local infiltration. Although not approved for perineural infiltration, there are reports of practitioners administering liposomal bupivacaine off-label for perineural and transversus abdominus plane (TAP) blocks.

Mechanisms of Nerve Injury

When analyzing the cause of neurologic injury after regional anesthesia,[49] it may be conceptually helpful to organize the causes of injury as being related to the patient's underlying condition, the surgical procedure, or the block procedure. Most of the cases of PNI that we see have multifactorial etiology, and it is difficult to differentiate the magnitude of the contribution to the overall injury by the many component factors.

In one of the largest observational database studies of postoperative nerve injuries, which looked at 380,680 patients undergoing anesthetic procedures over a 10-year period at a major academic medical center, the authors concluded that peripheral nerve blockade was not an independent predictor of nerve injury after surgery.[10] In contrast, patients with diabetes or hypertension and those using tobacco products were at higher risk, along with patients undergoing orthopedic surgery, neurosurgery, cardiac surgery, and general surgery.

The forces that cause nerve injury can be classified as those related to stretch, compression, ischemia, metabolic or toxic chemical injury, inflammation[50] (Parsonage-Turner syndrome), and trauma (blunt or lacerating). Needle-related injury to the brachial plexus associated with performance of the block would cause either blunt or lacerating trauma as a mechanism of injury, or compression and ischemia from an intra- or extraneural hematoma.

Arthroscopic shoulder surgery has its own inherent risks for nerve injury,[14] independent of anesthetic techniques, and these risks are associated with traction on the brachial plexus, due to positioning during surgery with abduction of the shoulder joint. In addition, irrigating fluid extravasation can cause tissue edema and compress the brachial plexus and peripheral nerves. Arthroscopic portals can damage nerves, especially given the anatomic variability of nerve distribution.

The Seddon classification of nerve injury (Table 2) is a useful clinical model to describe nerve injury, severity, and prognosis, dividing peripheral nerve injuries into 3 grades.[49,51,52]

Diagnosis and Treatment

It is important to examine the patient and document the injury immediately, and then rule out a treatable cause, such as a hematoma or other mass effect causing compression and ischemia. This can be done with palpation on physical examination, or via imaging studies such as ultrasound or magnetic resonance imaging/magnetic resonance neurography (MRI/MRN).

While purely sensory deficits can be managed conservatively and observed, any motor weakness is a serious injury and warrants an immediate neurologic consultation. This workup should include electrodiagnostic (EDX) studies with nerve conduction studies (NCS; motor and sensory) and needle electromyography (EMG).

EDX studies, EMG, and NCS are helpful in that they can provide clues to the location, timing, and severity of the injury, and early signs of recovery.[52-54] However, they cannot distinguish the cause of the injury, although they may be helpful when interpreted in light of the clinical picture.

Although it is usually recommended to obtain NCS 3 to 4 weeks after the diagnosis of a nerve injury, as most of them will have resolved spontaneously, in the event of a severe or profound deficit, a baseline study is appropriate. If there is a previously underlying and undetected injury, the EMG will show signs of chronic denervation, including increased insertional activity, fibrillation potentials, and sharp waves. EDX studies should be repeated at 1 month after injury, and then every 3 months to monitor recovery if the deficit does not show significant improvement.

There is no pharmacologic therapy that has been demonstrated to enhance neuroregeneration, so treatment is limited to physical therapy to maintain muscle mass and prevent flexion contractures, along with analgesic therapy using neuropathic agents and non-narcotic analgesics.

If there is no significant improvement in motor function by 6 to 9 months after injury, reconstructive nerve transfers or grafts should be considered, as the muscle fibers and neuromuscular junctions will irreversibly degenerate with fibrosis and function is unlikely to be restored. In the event that nerve transfers or grafting do not re-innervate the affected muscles, the only remaining surgical option to restore function is via tendon transfers from another viable muscle.

Although beyond the scope of this article, generally a demyelinating injury is diagnosed via NCS with a defining characteristic of a prolonged latency in motor and sensory stimulation. The needle EMG exam will confirm this with the absence of increased insertional activity and spontaneous activity, along with a lack of fibrillation potentials. All of these needle EMG findings are hallmarks of axonal injury. Axonal injury is further characterized on NCS with normal latencies but dramatically reduced amplitudes.

NCS can localize the site of the conduction block, and confirm or refute that the PNI lesion is at the site of the PNB; however, it may not always be possible to distinguish between anesthesia-related and surgical causes, when the surgical incision site and anesthetic block site, or tourniquet, are in close proximity.

Generally, block-related nerve injury for blocks performed at the brachial or lumbar plexus level is more likely to involve injury to multiple nerve distributions due to overlapping nerve root innervations. However, a non–anesthetic-related inflammatory neuropathy such as neuralgic amyotrophy (Parsonage-Turner syndrome) could also mimic this presentation, along with stretch injuries to the brachial or lumbar plexus. In contrast,

Table 2. Seddon Classification of Nerve Injury

Neurapraxia
The most common and the least severe, this injury has the best prognosis.
This injury is limited to damage of the myelin sheath around the individual axon. Depending on the extent of damage to the sheath, nerve conduction may be slowed or completely blocked.
This is the injury seen usually as the result of nerve compression and stretch caused by patient positioning or due to tourniquet-related compression, stretch, and ischemia.
Since the axon is undamaged and remains in continuity, the nerve usually returns to normal function over a period of days to weeks with myelin regeneration and complete recovery.
Axonotmesis
Constitutes more severe damage, with injury to the axon and the myelin sheath inside the protective endoneurium tube.
Due to preservation of the endoneurium, perineurium, and epineurium connective tissue highway, the nerve has the potential to regenerate on its own, although in some cases only incomplete recovery occurs.
Neurotmesis
The most severe type of injury, this involves complete transection of the nerve, along with the connective tissue layers.
Surgical repair involving nerve transfers or nerve interposition grafts may completely or partially restore function, but the results are highly variable.

a surgically caused injury or positioning injury would manifest as a mononeuropathy, or a mononeuropathy multiplex related to trauma to multiple nerves at or near the surgical site.

Conclusion

Serious and permanent PNI after nerve block is a rare event, and most likely a result of multifactorial causes not necessarily related to the administration of a PNB. However, temporary minor injuries may be more common than appreciated. It is important to set expectations with patients about the risk for potential nerve injury during the informed consent process, and meticulously document the block process in the medical record.

Post-block and postsurgical nerve injuries are neither entirely predictable nor preventable, even with expertly trained physicians utilizing best practices. EDX studies may be helpful in assessing the site of the nerve injury, its severity, whether or not a previously undiagnosed injury was present, and the time course and potential for recovery of function. It is important to understand the limitations of EDX and MRI/MRN with respect to determining the etiology of the nerve injury.

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