Complications of Regional Anesthesia and Acute Pain Management

Terese T. Horlocker, MD

KEYWORDS

• Perioperative nerve injuries • Regional anesthesia
• Neurologic deficits • Surgical complications

Perioperative nerve injuries have long been recognized as a complication of regional anesthesia. Fortunately, severe or disabling neurologic complications rarely occur. Risk factors contributing to neurologic deficit after regional anesthesia include neural ischemia (hypothetically be related to the use of vasoconstrictors or prolonged hypotension), traumatic injury to the nerves during needle or catheter placement, infection, and choice of local anesthetic solution.[1–4] In addition, postoperative neurologic injury due to pressure from improper patient positioning or from tightly applied casts or surgical dressings, as well as surgical trauma, are often attributed to the regional anesthetic.[5] Lynch and colleagues[6] reported a 4.3% incidence of neurologic complications following total shoulder arthroplasty. The neurologic deficit localized to the brachial plexus in 75% of affected patients. Importantly, the level of injury occurred most commonly at the upper and middle nerve trunks—the level at which an interscalene block is performed, making it impossible to determine the cause of the nerve injury (surgical vs anesthetic). Patient factors such as body habitus or a preexisting neurologic condition may also contribute.[7–9] For example, the incidence of peroneal nerve palsy following total knee replacement is increased in patients with significant valgus or a preoperative neuropathy and the severity is increased in patients receiving epidural analgesia (**Table 1**).[10,11] The safe conduct of neuraxial anesthesia involves knowledge of the large patient surveys as well as individual case reports of neurologic deficits following neural blockade. Prevention of complications, along with early diagnosis and treatment are important factors in management of regional anesthetic risks.

INCIDENCE OF NEUROLOGIC COMPLICATIONS

Although severe or disabling neurologic complications are rare, recent epidemiologic series suggest the frequency of some serious complications is increasing. A

Department of Anesthesiology, Mayo Clinic, 200 First Street SW, Rochester, MN, 55905, USA
E-mail address: horlocker.terese@mayo.edu

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Table 1
Risk profile for peroneal nerve palsy after total knee arthroplasty

Risk Factor	Peroneal Palsy (n = 8)	No Peroneal Nerve Palsy (n = 353)
Age (y)	64 ± 10	69 ± 10
Valgus (degrees)	13 ± 5[a]	9 ± 7
Tourniquet time (min)	141 ± 52[a]	103 ± 28
Neurologic condition	4[a]	30
Anesthetic technique		
General	3	112
Spinal	1	67
Epidural	4	174
Epidural analgesia	4[b]	104
Postoperative bleeding	3[a]	4

[a] P<0.05.
[b] Although postoperative epidural analgesia was not a risk factor for peroneal nerve palsy, all cases of peroneal nerve palsy with motor deficits occurred in patients with postoperative epidural analgesia.
Adapted from Horlocker TT, Cabanela ME, Wedel DJ. Does postoperative epidural analgesia increase the risk of peroneal nerve palsy after total knee arthroplasty? Anesth Analg 1994;79: 495–500; with permission.

prospective survey in France recently evaluated the incidence and characteristics of serious complications related to regional anesthesia.[2] Participating anesthesiologists kept a log of all cases and detailed information of serious complications occurring during or after regional anesthetics. All patients with a neurologic deficit lasting more than 2 days were examined by a neurologist; patients with cauda equina syndrome were evaluated with a CT scan to rule out compressive causes. A total of 103,730 regional anesthetics, were performed over 5 months. The incidence of cardiac arrest and neurologic complications was significantly higher after spinal anesthesia than other types of regional procedures (**Table 2**). Neurologic recovery was complete within 3 months in 29 of 34 patients with deficits. In 12 of 19 cases of radiculopathy after spinal anesthesia, and in all cases of radiculopathy after epidural or peripheral block, needle placement was associated with either paresthesia during needle insertion, or pain with injection. In all cases, the radiculopathy had the same topography as the associated paresthesia. The investigators concluded that needle

Table 2
Complications related to regional anesthesia

Technique	Cardiac Arrest	Death	Seizure	Neurologic Injury
Spinal (N = 40,640)	26 (3.9–8.9)	6 (0.3–2.7)	0 (0–0.9)	24 (3.5–8.3)
Epidural (N = 30,413)	3[a] (0.2–2.9)	0 (0–1.2)	4 (0.4–3.4)	6[a] (0.4–3.6)
Peripheral Blocks (N = 21,278)	3[b] (0.3–4.1)	1 (0–2.6)	16[c] (3.9–11.2)	4[c] (0.5–4.8)
IV Regional (N = 11,229)	0 (0–3.3)	0 (0–3.3)	3 (0.5–7.8)	0 (0–3.3)

Data presented are number and (95% confidence interval).
[a] Epidural versus spinal (*P*<.05).
[b] Peripheral nerve blocks versus spinal (*P*<.05).
[c] Peripheral nerve blocks versus epidural (*P*<.05).
Data from Auroy Y, Narchi P, Messiah A, et al. Serious complications related to regional anesthesia: results of a prospective survey in France. Anesthesiology 1997;7:479–86.

trauma and local anesthetic neurotoxicity were the causes of most neurologic complications. In a follow-up investigation performed with similar methodology 5 years later, the investigators reported a slight decrease of neurologic complications related to regional anesthetic technique.[12]

An epidemiologic study evaluating severe neurologic complications after neuraxial block conducted in Sweden between 1990 and 1999 reported some disturbing trends.[13] During the 10 year study period, approximately 1,260,000 spinal and 450,000 epidural (including 200,000 epidural blocks for labor analgesia) were performed. A total of 127 serious complications were noted, including spinal hematoma (33), cauda equina (32), meningitis (29), and epidural abscess (13). The nerve damage was permanent in 85 patients. Complications occurred more often after epidural than spinal blockade, and were different in character; cauda equina syndrome, spinal hematoma, and epidural abscess were more likely to occur after epidural block, whereas meningitis was more often associated with a spinal technique. Undiagnosed spinal stenosis (detected during evaluation of the new neurologic deficits) was a risk factor for cauda equina syndrome and paraparesis with both techniques. In the 18 cases of cauda equina syndrome following spinal anesthesia, 5% hyperbaric lidocaine was administered in eight cases, while bupivacaine (hyperbaric or isobaric) was the local anesthetic in 11 cases. This large series suggests that the incidence of severe anesthesia-related complications is not as low as previously reported. Moreover, since serious complications were noted to occur even in the presence of experienced anesthesiologists, continued vigilance in patients undergoing neuraxial anesthesia is warranted.

For example, Cheney and colleagues[14] examined the American Society of Anesthesiologists (ASA) Closed Claims database to determine the role of nerve damage following regional-pain block or general anesthesia in malpractice claims filed against anesthesia care providers. Of the 4,183 claims reviewed, 670 (16%) were for anesthesia-related nerve injury, including 189 claims involving the lumbosacral roots (105 claims) or spinal cord (84 claims); spinal cord injuries were the leading cause of claims for nerve injury that occurred in the 1990s, whereas injuries to the ulnar nerve or brachial plexus were more common previously. In addition, lumbosacral nerve root injuries having identifiable causes were associated predominantly with a regional (compared with general) anesthetic technique (92%), and were related to paresthesias during needle or catheter placement or pain during injection of local anesthetic. Major factors associated with spinal cord injury were blocks for chronic pain management and systemic anticoagulation in the presence of neuraxial block. A more recent ASA Closed Claims analysis of the 1005 cases of regional anesthesia claims from 1980 to 1999, reported that the majority of neuraxial complications associated with regional anesthesia claims resulted in permanent neurologic deficits.[15] Hematoma was the most common cause of neuraxial injuries and the majority of these cases were associated with either an intrinsic or an iatrogenic coagulopathy; 89% of patients had a permanent deficit. Conversely, complications caused by meningitis or abscess were more likely to be temporary. In a subset comparison of obstetric versus nonobstetric neuraxial anesthesia claims, obstetrics had a higher proportion of claims with low-severity and temporary injuries.

SPINAL CORD AND ROOT INJURY FROM NEURAXIAL NEEDLE AND CATHETER PLACEMENT

Direct needle or catheter-induced trauma rarely results in permanent or severe neurologic injury. A retrospective study of 4,767 spinal anesthetics noted the presence of a paresthesia during needle placement in 298 (6.3%) of patients. Importantly, four

of the six patients with a persistent paresthesia postoperatively complained of a paresthesia during needle placement, identifying elicitation of a paresthesia as a risk factor for a persistent paresthesia.[16] As previously noted, in the series by Auroy and colleagues,[2] two-thirds of the patients with neurologic complications experienced pain during needle placement or injection of local anesthetic. In all cases, the neurologic deficit had the same distribution as the elicited paresthesia. It is unknown whether clinicians should abandon the procedure if a paresthesia is elicited (rather than repositioning the needle), in an effort to decrease the risk of nerve injury. This decision is complicated by the series of conus medullaris injuries following spinal (three cases) or combined spinal-epidural (four cases) anesthesia with a pencil point needle reported by Reynolds.[1] All seven patients complained of pain on needle insertion (only one noted pain on injection) and suffered damage to more than a single nerve root. In all patients, the anesthesiologist believed needle placement to have occurred at or below L2-3. A syrinx was noted on MRI in six cases suggesting intracord injection was the cause of the deficits. Cases of cord damage from needle insertion were also reported in the series by Auroy and colleagues[12] and Moen and colleagues.[13] Importantly, in all cases, the proceduralist had presumed the level of insertion to be below L1. These cases support the recommendation to insert needles below L3 to reduce the risk of direct needle trauma.[1,17]

The passage and presence of an indwelling catheter into the subarachnoid or epidural space presents an additional source of direct trauma. However, there is a lower frequency of persistent paresthesia or radiculopathy following epidural techniques, which are typically associated with (epidural) catheter placement, compared with single injection spinal anesthesia.[2,12,13] Although the incidence of neurologic complications associated with thoracic epidural techniques has historically been judged to be higher than that of lumbar placement, Giebler and colleagues[18] noted only a 0.2% incidence of postoperative radicular pain in 4185 patients undergoing thoracic epidural catheterization; all cases were responsive to catheter removal.

NERVE INJURY FROM PLEXUS-PERIPHERAL NEEDLE AND CATHETER PLACEMENT

Many anesthesiologists intentionally elicit a paresthesia during the performance of peripheral regional techniques. Although the elicitation of a paresthesia may represent direct needle trauma and increase the risk of persistent paresthesia associated with regional anesthesia, there are no clinical studies that definitively prove or refute the theory.[19–23] Selander and colleagues[21] reported a “higher” incidence of postoperative nerve injury in patients where a paresthesia was sought during axillary block (2.8%) compared with those undergoing a perivascular technique (0.8%). However, the difference was not statistically significant. Importantly, 40% of patients in the perivascular group reported unintentional paresthesias during the procedure, demonstrating the difficulty with standardization of technique and analysis of neural injury. Postoperative neurologic deficits ranged from slight hypersensitivity to severe paresis, and persisted from 2 weeks to greater than 1 year. In a prospective study using a variety of regional anesthetic approaches including paresthesia, transarterial, and nerve stimulator techniques, Urban and Urquhart[23] noted that mild paresthesias were common the day after surgery, occurring after 9% of interscalene blocks and after 19% of axillary blocks. At 2 weeks the incidence had decreased significantly, with near complete resolution noted at 4 weeks. Stan and colleagues[22] reported a 0.2% incidence of neurologic complications after axillary blocks performed with the transarterial approach. However, vascular complications such as transient arterial spasm, unintentional vascular injection, and hematoma formation occurred in 1.4% of patients.

Theoretically, localization of neural structures with a nerve stimulator would allow a high success rate without increasing the risk of neurologic complications, but this has not been formally evaluated. Fanelli and colleagues[19] prospectively evaluated 3996 patients undergoing sciatic-femoral, axillary, and interscalene blocks using a multiple injection, nerve stimulator technique. During the first month after surgery, 69 patients (1.7%) developed neurologic dysfunction; recovery was complete in all but one in 4 to 12 weeks. (This frequency is similar to that reported using a paresthesia technique). The only variable associated with neurologic injury was tourniquet inflation pressure greater than 400 mm Hg. Use of a nerve stimulator does not prevent intraneural injection. Indeed, serious neurologic injury has been reported following uneventful brachial plexus block using a nerve stimulator technique.[24,25] Equally interesting are the cases in which apparent intraneural injection did not result in neurologic injury.[26,27]

The use of ultrasound as a technique for neural localization continues to gain popularity and application. However, a superior efficacy and safety compared with other techniques has not been consistently demonstrated. For example, a recent systematic review (including both randomized control trials and case series) reported that use of ultrasound does not consistently improve the success of regional anesthesia versus most other techniques. However, ultrasound was not inferior for efficacy, did not increase risk, and offers other potential patient-oriented benefits.[20,28]

Currently, no compelling evidence exists to endorse a single technique as superior with respect to success rate or incidence of complications.[20] Needle gauge, type (short vs long bevel), and bevel configuration may also influence the degree of nerve injury, although the findings are conflicting and there are no confirmatory human studies (**Box 1**).[20,29,30]

Box 1
Recommendations for limiting peripheral nerve injury

- There are no animal or human data to support the superiority of one nerve localization technique—paresthesia, nerve stimulation, ultrasound—over another with regards to reducing the likelihood of nerve injury.
- Animal data have linked high injection pressures to subsequent fascicular injury, but there are no human data that confirm or refute the effectiveness of injection pressure monitoring for limiting nerve injury.
- There are no human data to support the superiority of one local anesthetic or additive over another with regard to reducing the likelihood of neurotoxicity.
- Patients with diseased or previously injured nerves (eg, diabetes mellitus, severe peripheral vascular disease, or chemotherapy) may theoretically be at increased risk for block-related nerve injury. Although isolated case reports have described new or progressive neurologic deficits after regional anesthetic techniques in patients with multiple sclerosis or previous exposure to chemotherapy, clinical experience can neither refute nor confirm these concerns. Based on limited animal data, consideration may be given to avoiding local anesthetics that are more potent, reducing local anesthetic doses and/or concentration, and avoiding or limiting vasoconstrictive additives in these patients.
- If damage to protective tissue barriers such as the perineurium is suspected from an abnormally painful paresthesia or pain on injection of local anesthetic, further injection should be halted immediately, and the needle repositioned. Consideration may be given to aborting the block procedure to avoid further deposition of local anesthetic and additive.

From Neal JM, Bernards CM, Hadzic A, et al. ASRA practice advisory on neurologic complications in regional anesthesia and pain medicine. Reg Anesth Pain Med 2008;33:404–15; with permission.

The potential added risk of neurologic complications resulting from placement of a plexus or peripheral nerve catheter remains undefined.[31] Although difficulty during catheter insertion may lead to vessel puncture, tissue trauma and bleeding, significant complications are uncommon and permanent sequelae are rare. In a recent prospective study involving 1,416 patients with continuous catheters, there were 12 patients (0.84%) experiencing serious adverse events and 3 (0.21%) patients had neurologic lesions attributed to the continuous peripheral nerve catheter.[32]

LOCAL ANESTHETIC TOXICITY

Neurologic complications after neuraxial anesthesia may be a direct result of local anesthetic toxicity. There is both laboratory and clinical evidence that local anesthetic solutions are potentially neurotoxic and that the neurotoxicity varies among local anesthetic solutions.[4,33–35] Neurotoxicity is dependent on Pka, lipid solubility, protein binding and potency. In histopathologic, electrophysiologic, and neuronal cell models, lidocaine and tetracaine appear to have a greater potential for neurotoxicity than bupivacaine at clinically relevant concentrations.[36] Additives such as epinephrine and bicarbonate may also affect neurotoxicity. The presence of a preexisting neurologic condition may predispose the nerve to the neurotoxic effects of local anesthetics.[9,33]

Although most local anesthetics administered in clinical concentrations and doses do not cause nerve damage, prolonged exposure, high dose, and/or high concentrations of local anesthetic solutions at the spinal roots may result in permanent neurologic deficits.[37] For example, cauda equina syndrome has been reported after single dose and continuous spinal anesthesia, intrathecal injection during intended epidural anesthesia, and repeated intrathecal injection after failed spinal block with lidocaine.[2,4,38] Presumably, injection (and/or reinjection) results in high concentrations of local anesthetic within a restricted area of the intrathecal space and causes neurotoxic injury. In the study by Auroy and colleagues,[2] 75% of the neurologic complications after uneventful (atraumatic) spinal anesthesia occurred in patients who received hyperbaric lidocaine, including one patient who received 350 mg over 5 hours with a 5% lidocaine infusion. Drasner[39] has recommended a maximum dose of 60 mg of lidocaine and the avoidance of epinephrine to prolong lidocaine spinal anesthesia. In addition, many clinicians recommend the use of isobaric solutions during continuous spinal techniques to reduce the risk of nonuniform distribution within the intrathecal space. Attention to patient positioning, total local anesthetic dose, and careful neurologic examination (evaluating for preferential sacral block) will assist in the decision to inject additional local anesthetic in the face of a patchy or failed block (**Box 2**).[40]

2-Chloroprocaine was introduced nearly 50 years ago as a local anesthetic for epidural administration. However, concern for neurotoxicity emerged 2 decades ago with a series of eight cases of neurologic injury associated with the use of Nesacaine-CE, a chloroprocaine solution containing the antioxidant sodium bisulfite. In all cases, the injury occurred after a large volume of anesthetic solution intended for the epidural space was accidentally administered intrathecally. Subsequent laboratory investigations evaluating the toxic contributions of 2-chloroprocaine, bisulfite, epinephrine, and pH reported that the commercial solution of 3% chloroprocaine (containing 0.2% sodium bisulfite, pH 3) produced irreversible block, but exposure to the same solution buffered to pH 7.3 resulted in complete recovery.[41] It was assumed that bisulfite was the source of neurotoxicity and that solutions that were bisulfite-free were safe for intrathecal use. More recently, these experiments were repeated with a more appropriate animal model and yielded different results: nerve

Box 2
Recommendations for anesthetic administration after a "failed spinal"

- Aspiration of cerebrospinal fluid (CSF) should be attempted before and after injection of anesthetic.
- Sacral dermatomes should always be included in an evaluation of the presence of a spinal block.
- If CSF is aspirated after anesthetic injection, it should be assumed that the local anesthetic has been delivered into the subarachnoid space; total anesthetic dosage should be limited to the maximum dose a clinician would consider reasonable to administer in a single injection.
- If an injection is repeated, the technique should be modified to avoid reinforcing the same restricted distribution (eg, alter patient position or switch to a local anesthetic of different baricity).
- If CSF cannot be aspirated after injection, repeat injection of a full dose of local anesthetic should not be considered unless careful sensory examination (conducted after sufficient time for development of sensory anesthesia) reveals no evidence of block.

From Drasner K. Local anesthetic neurotoxicity: clinical injury and strategies that may minimize risk. Reg Anesth Pain Med 2002;27:576–80; with permission.

injury scores were greater after administration of plain chloroprocaine compared with those of chloroprocaine containing bisulfite. These findings suggest clinical deficits associated with unintentional intrathecal injection of chloroprocaine likely resulted from a direct effect of the anesthetic, not the preservative. In addition, the data suggest that bisulfite can actually reduce neurotoxic damage induced by intrathecal local anesthetic.[42] Although recent clinical and volunteer studies[43] have not reported neurologic symptoms following spinal anesthesia with low-dose 2-chloroprocaine (30–40 mg), the laboratory evidence for toxicity warrants a cautious approach until additional toxicity data are available.

Transient Neurologic Symptoms

Transient neurologic symptoms (TNS) were first formally described in 1993. Schneider and colleagues[44] reported four cases of severe radicular back pain occurring after resolution of hyperbaric lidocaine spinal anesthesia. All four patients had undergone surgery in the lithotomy position. No sensory or motor deficits were detected on examination, and the symptoms resolved spontaneously within several days. Multiple laboratory and clinical studies have been performed in an attempt to define the causes, clinical significance, and risk factors associated with TNS. However, our understanding remains incomplete.

The incidence of TNS has ranged between 0% and 37%,[45–47] and is dependent on anesthetic, surgical, and probably undefined patient factors. A large, multicenter, epidemiologic study involving 1863 patients was recently performed to identify potential risk factors for TNS.[48] The incidence of TNS with lidocaine (11.9%) was significantly higher than that with tetracaine (1.6%) or bupivacaine (1.3%). The pain was described as severe in 30% of patients and resolved within a week in over 90% of cases. Outpatient status, obesity, and lithotomy position also increase the risk of TNS for patients who receive lidocaine. This suggests that the risk of TNS is high among outpatients in the lithotomy position (24.3%) and low for inpatients having surgery in positions other than lithotomy (3.1%). However, these variables were not risk factors with tetracaine or bupivacaine. The investigators also reported that neither gender, age, history of back pain or neurologic disorder, lidocaine dose or

concentration, spinal needle or size, aperture direction, nor addition of epinephrine increased the risk of TNS (**Box 3**). These findings were confirmed in a systematic review of TNS.[49]

The high frequency of TNS with lidocaine spinal anesthesia has resulted in a search for a safe and effective alternative. The intrathecal administration of 2-chloroprocaine is under reconsideration due to the concern regarding toxicity, as previously mentioned. Mepivacaine may be a suitable substitute. In a series of 1273 patients undergoing spinal or combined spinal-epidural anesthesia, TNS occurred in only 78 (6.4%; 95% CI 5.1%–8%).[50]

The causes and clinical significance of TNS are unknown. Recent studies suggest local anesthetic toxicity, although the mechanism may not be identical to that of cauda equina syndrome.[51] Although many anesthesiologists believe that the reversible radicular pain is on one side of a continuum leading to irreversible cauda equina syndrome, there are no data to support this concept. It is important to distinguish between factors associated with serious neurologic complications, such as cauda equina syndrome, and transient symptoms when making recommendations for the clinical management of patients. For example, increasing the concentration or dose of lidocaine and adding epinephrine increases the risk of irreversible neurotoxicity, but has little effect on the risk of TNS. Therefore, the clinician must determine the appropriate intrathecal solution, including adjuvants, given the surgical duration and intraoperative position for each individual patient.

NEURAL ISCHEMIA

Local anesthetic solutions have varied effects on spinal cord blood flow. For example, lidocaine and tetracaine either maintain or increase blood flow, whereas bupivacaine and levobupivacaine result in a decrease.[52–55] The addition of epinephrine or phenylephrine results in a further decrease. However, in laboratory investigations, the alterations in blood flow are not accompanied by changes in histology or behavior.

Box 3
Factors that did not increase the risk of developing TNS after lidocaine spinal anesthesia

- Gender
- Age (<60 yr vs 60+ yr)
- Preexisting neurologic disorder or back pain
- Needle type (Quincke vs pencil point)
- Needle size (22 gauge vs 24–25 gauge vs 26–27 gauge)
- Bevel direction during injection (caudad vs cephalad vs LATERAL)
- Lidocaine dose (<50 mg vs 51–74 mg vs >75 mg)
- Intrathecal epinephrine
- Intrathecal opioid
- Intrathecal dextrose
- Paresthesia during needle placement

Data from Freedman JM, Li DK, Drasner K, et al. Transient neurologic symptoms after spinal anesthesia: an epidemiologic study of 1,863 patients. Anesthesiology 1998;89:633–41.

Likewise, large clinical studies have failed to identify the use of vasoconstrictors as a risk factor for temporary or permanent deficits. Most presumed cases of vasoconstrictor-induced neurologic deficits have been reported as single case reports, often with several other risk factors present.[2,56]

Peripheral nerves have a dual blood supply consisting of intrinsic endoneural vessels and extrinsic epineural vessels. A reduction or disruption of peripheral nerve blood flow may result in neural ischemia. Intraneural injection of volumes as small as 50 to 100 μL may generate intraneural pressures that exceed capillary perfusion pressure for as long as 10 minutes and thus cause neural ischemia.[57] Endoneural hematomas have also been reported after intraneural injection.[30] Epineural blood flow is also responsive to adrenergic stimuli.[58,59] The use of local anesthetic solutions containing epinephrine theoretically may produce peripheral nerve ischemia, especially in patients with microvascular disease.[3,33]

Finally, the addition of vasoconstrictors may potentiate the neurotoxic effects of local anesthetics. In a laboratory model, it was determined that the neurotoxicity of intrathecally administered lidocaine was increased by the addition of epinephrine.[60] A recent investigation by Sakura and colleagues[61] noted the addition of phenylephrine increased the risk of TNS in patients undergoing tetracaine spinal anesthesia (although no patient had sensory or motor deficits). However, the actual risk of significant neurologic ischemia causing neurologic compromise in patients administered local anesthetic solutions containing vasoconstrictors appears to be very low.

HEMORRHAGIC COMPLICATIONS

The actual incidence of neurologic dysfunction resulting from hemorrhagic complications associated with neuraxial blockade is unknown; however, recent epidemiologic studies suggest the incidence is increasing. In a review of the literature between 1906 and 1994, Vandermeulen and colleagues[62] reported 61 cases of spinal hematoma associated with epidural or spinal anesthesia. In 87% of patients, a hemostatic abnormality or traumatic or difficult needle placement was present. More than one risk factor was present in 20 of 61 cases. Importantly, although only 38% of patients had partial or good neurologic recovery, spinal cord ischemia tended to be reversible in patients who underwent laminectomy within 8 hours of onset of neurologic dysfunction.

The need for prompt diagnosis and intervention in the event of a spinal hematoma was also demonstrated in two reviews of the ASA Closed Claims database involving claims related to nerve injury.[14,15] Cheney and colleagues[14] examined the claims of nerve injury associated with general or regional block between 1990 and 1999 and noted that spinal cord injuries were the leading cause of claims in the 1990s. Furthermore, spinal hematomas accounted for nearly half of the spinal cord injuries. Patient care was rarely judged to have met standards due to delay in the diagnosis, and resultant poor outcome. Consequently, the median payment was very high. A more recent in-depth analysis of the claims related to nerve injury following regional anesthesia between 1980 and 1999 reported 36 spinal hematomas, associated mainly with vascular or orthopedic surgical procedures. Three-fourths of patients had evidence of a preexisting or iatrogenic coagulation abnormality.[15] Over half the patients received intravenous heparin during a vascular surgical or diagnostic procedure, often in combination with other medications that impair coagulation. Consistent with Vandermeulen and colleagues,[62] the presenting symptom was increased motor block (83% of cases), rather than back pain (25% of cases). Importantly, the presence of postoperative numbness or weakness was typically attributed to local anesthetic effect rather than spinal cord ischemia, which delayed the diagnosis. Although the

symptoms were noted typically on the first postoperative day, often 24 hours or more elapsed before diagnosis. There were permanent deficits in 90% of patients.

It is impossible to conclusively determine risk factors for the development of spinal hematoma in patients undergoing neuraxial blockade solely through review of the case series, which represent only patients with the complication and do not define those who underwent uneventful neuraxial analgesia. However, large inclusive surveys that evaluate the frequencies of complications (including spinal hematoma), as well as identify subgroups of patients with higher or lower risk, enhance risk stratification. Moen and colleagues[13] investigated serious neurologic complications among 1,260,000 spinal and 450,000 epidural blocks performed in Sweden over 10 years. Twenty-four of the 33 spinal hematomas occurred in the last 5 years of the decade surveyed. Of the 33 spinal hematomas, 24 occurred in females and 25 were associated with an epidural technique. A coagulopathy (existing or acquired) was present in 11 patients; two of these patients were parturients with hemolysis, elevated liver enzymes, and low platelets (HELLP) syndrome. Pathology of the spine was present in six patients. The presenting complaint was typically lower extremity weakness. Only 5 of 33 patients recovered neurologically (due to delay in the diagnosis or intervention). These demographics, risk factors, and outcomes confirm those of previous series. However, the methodology allowed for calculation of frequency of spinal hematoma among patient populations. For example, the risk associated with epidural analgesia in women undergoing childbirth was significantly less (1 in 200,000) than that in elderly women undergoing knee arthroplasty (1 in 3600, $P<.0001$). Likewise, women undergoing hip fracture surgery under spinal anesthesia had an increased risk of spinal hematoma (1 in 22,000) compared with all patients undergoing spinal anesthesia (1 in 480,000).

Overall, these series suggest that the risk of clinically significant bleeding varies with age (and associated abnormalities of the spinal cord or vertebral column), the presence of an underlying coagulopathy, difficulty during needle placement, and an indwelling neuraxial catheter during sustained anticoagulation (particularly with unfractionated, standard, or low-molecular- weight heparin [LMWH]); perhaps in a multifactorial manner. They also consistently demonstrate the need for prompt diagnosis and intervention.

Plexus and Peripheral Blockade in the Anticoagulated Patient

Although spinal hematoma is the most significant hemorrhagic complication of regional anesthesia due to the catastrophic nature of bleeding into a fixed and noncompressible space, the associated risk following plexus and peripheral techniques remains undefined. The most significant study involving the risk of hemorrhagic complications associated with peripheral blocks included 670 patients undergoing continuous lumbar plexus blocks who were anticoagulated with warfarin.[63] Nearly all catheters were removed on the second postoperative day. At the time of catheter removal, 36% of patients had an international normalized ratio (INR) greater than 1.4. One case of local bleeding was noted in a patient with a corresponding INR of 3.0, which was treated with local pressure.

The Third American Society of Regional Anesthesia and Pain Medicine Practice Advisory on Regional Anesthesia and Antithrombotic Therapy reviewed all published cases of clinically significant bleeding or bruising after plexus or peripheral techniques.[64] In all patients with neurodeficits, neurologic recovery was complete within 6 to 12 months. Thus, although bleeding into a neurovascular sheath may result in significant decreases in hematocrit, the expandable nature of peripheral site may decrease the chance of irreversible neural ischemia. Of the 13 patients with bleeding

complications following peripheral or plexus block in patients without anticoagulation, 5 were serious and required hospitalization, transfusion and/or surgical intervention (including one emergency tracheostomy after traumatic stellate block). Two of the 13 complications occurred after lumbar sympathetic or paravertebral techniques. There were also 13 cases of hemorrhagic complications associated with peripheral or plexus block in patients receiving antithrombotic therapy preblock and/or postblock. Twelve of these complications were serious, including one death due to massive hemorrhage following lumbar sympathetic block in a patient receiving clopidogrel. In all but one patient, hospitalization was complicated and prolonged. Nearly half of the patients received enoxaparin within 24 hours of the technique. Although this may implicate LWMH, it is also representative of the orthopedic patients who undergo lower extremity block and subsequently undergo thromboprophylaxis. Three of the patients were receiving nonsteroidal antiinflammatory drugs only.

This series of 26 patients is insufficient to make definitive recommendations. However, trends are evolving which may assist with patient management. For example, these cases suggest that significant blood loss, rather than neural deficits may be the most serious complication of non-neuraxial regional techniques in the anticoagulated patient. In addition, hemorrhagic complications following the deep plexus or peripheral techniques (eg, lumbar sympathetic, lumbar plexus, and paravertebral), particularly in the presence of antithrombotic therapy, are often serious and a source of major patient morbidity. Consequently, for patients undergoing deep plexus or peripheral block, it is recommended that guidelines regarding neuraxial techniques be similarly applied.

The decision to perform spinal or epidural anesthesia or analgesia and the timing of catheter removal in a patient receiving thromboprophylaxis should be made on an individual basis—weighing the small, though definite, risk of spinal hematoma with the benefits of regional anesthesia for a specific patient. Alternative anesthetic and analgesic techniques exist for patients considered to be at an unacceptable risk. The patient's coagulation status should be optimized at the time of spinal or epidural needle or catheter placement, and the level of anticoagulation must be carefully monitored during the period of epidural catheterization (**Table 3**). It is important to note that patients respond with variable sensitivities to anticoagulant medications. Indwelling catheters should not be removed in the presence of a significant coagulopathy, as this appears to significantly increase the risk of spinal hematoma.[13,62,65] In addition, communication between clinicians involved in the perioperative management of patients receiving anticoagulants for thromboprophylaxis is essential to decrease the risk of serious hemorrhagic complications. The patient should be closely monitored in the perioperative period for signs of cord ischemia. If spinal hematoma is suspected, the treatment of choice is immediate decompressive laminectomy.

INFECTIOUS COMPLICATIONS

Bacterial infection of the central neuraxis may present as meningitis or cord compression secondary to abscess formation. Possible risk factors include underlying sepsis, diabetes, depressed immune status, steroid therapy, localized bacterial colonization or infection, and chronic catheter maintenance. The infectious source for meningitis and epidural abscess may result from distant colonization or localized infection with subsequent hematogenous spread and CNS invasion. The anesthetist may also transmit microorganisms directly into the CNS by needle or catheter contamination through a break in aseptic technique or passage through a contiguous infection. An indwelling neuraxial catheter, though aseptically sited, may be colonized with skin flora and consequently serve as a source for ascending infection to the epidural or intrathecal space.

Table 3
Neuraxial anesthesia and anticoagulation

Warfarin	Discontinue chronic warfarin therapy 4–5 days before spinal procedure and evaluate INR. INR should be within the normal range at time of procedure to ensure adequate levels of all vitamin K-dependent factors. Postoperatively, daily INR assessment with catheter removal occurring with INR <1.5
Antiplatelet medications	No contraindications with aspirin or other NSAIDs. Thienopyridine derivatives (clopidogrel and ticlopidine) should be discontinued 7 d and 14 d, respectively, before procedure. GP IIb/IIIa inhibitors should be discontinued to allow recovery of platelet function before procedure (8 h for tirofiban and eptifibatide, 24–48 h for abciximab).
Thrombolytics/fibrinolytics	There are no available data to suggest a safe interval between procedure and initiation or discontinuation of these medications. Follow fibrinogen level and observe for signs of neural compression.
LMWH	Delay procedure at least 12 h from the last dose of thromboprophylaxis LMWH dose. For "treatment" dosing of LMWH, at least 24 h should elapse before procedure. LMWH should not be administered within 24 h after the procedure. Indwelling epidural catheters should be maintained with caution and only with once daily dosing of LMWH and strict avoidance of additional hemostasis altering medications, including ketorolac.
Unfractionated SQ heparin	There are no contraindications to neuraxial procedure if total daily dose is less than 10,000 units. For higher dosing regimens, manage according to intravenous heparin guidelines.
Unfractionated IV heparin	Delay needle/catheter placement 2–4 hours after last dose, document normal aPTT. Heparin may be restarted 1 h following procedure. Sustained heparinization with an indwelling neuraxial catheter associated with increased risk; monitor neurologic status aggressively.

Abbreviations: aPTT, activated partial thromboplastin time; GP IIb/IIIa, platelet glycoprotein receptor IIb/IIIa inhibitors; INR, international normalized ratio; LMWH, low-molecular-weight heparin; NSAIDs, nonsteroidal antiinflammatory drugs.

Data from Horlocker TT, Wedel DJ, Benzon H, et al. Regional anesthesia in the anticoagulated patient: defining the risks (the second ASRA Consensus Conference on Neuraxial Anesthesia and Anticoagulation). Reg Anesth Pain Med 2003;28:172–97; and Horlocker TT, Wedel DJ. Anticoagulation and neuraxial blockade: historical perspective, anesthetic implications, and risk management. Reg Anesth Pain Med 1998;23:129–34.

Historically, the frequency of serious CNS infections such as arachnoiditis, meningitis, and abscess following spinal or epidural anesthesia was considered to be extremely low—cases were reported as individual cases or small series.[66,67] However, recent epidemiologic series from Europe suggest that the frequency of infectious complications associated with neuraxial techniques is increasing.[13,68,69] In a national study conducted from 1997 to 1998 in Denmark, Wang and colleagues[69] reported the incidence of epidural abscess after epidural analgesia was 1 in 1930 catheters. Patients with epidural abscess had an extended duration of epidural catheterization (median 6 days, range 3–31 days). In addition, the majority of the patients with epidural abscess were immunocompromised. Often the diagnosis was delayed; the time to first symptom to confirmation of the diagnosis was a median of 5 days. *Staphylococcus*

aureus was isolated in 67% of patients. Patients without neurologic deficits were successfully treated with antibiotics, while those with deficits underwent surgical decompression, typically with only moderate neurologic recovery.

In the series by Moen and colleagues[13] there were 42 serious infectious complications. Epidural abscess occurred in 13 patients; 9 (70%) were considered immunocompromised as a result of diabetes, steroid therapy, cancer, or alcoholism. Six patients underwent epidural block for analgesia following trauma. The time from placement of the epidural catheter to first symptoms ranged from 2 days to 5 weeks (median 5 days). Although prevailing symptoms were fever and sever backache, 5 developed neurologic deficits. All seven positive cultures isolated *S aureus*. Overall neurologic recovery was complete in 7 of 12 patients. However, 4 of the 5 patients with neurologic symptoms did not recover. Meningitis was reported in 29 patients for an overall incidence of 1:53,000. A documented perforation of the dura (intentional or accidental) occurred in 25 of 29 cases. In the 12 patients in whom positive cultures were obtained, alpha-hemolytic streptococci (pathogens common to the oropharynx) were isolated in 11 patients and *S aureus* in 1.

These large epidemiologic studies represent new and unexpected findings regarding the demographics, frequency, causes, and prognosis of infectious complications following neuraxial anesthesia. Epidural abscess is most likely to occur in immunocompromised patients with prolonged durations of epidural catheterization. The most common causative organism is *S aureus*, which suggests the colonization and subsequent infection from normal skin flora as the pathogenesis. In addition, delays in diagnosis and treatment result in poor neurologic recovery, despite surgical decompression. Conversely, patients who develop meningitis following neuraxial blockade typically are healthy and have undergone uneventful spinal anesthesia. Furthermore, the series by Moen and colleagues[13] validates the findings of individual case reports of meningitis after spinal anesthesia—the source of the pathogen is mostly likely to be in the upper airway.[70]

Infectious complications may also occur after plexus and peripheral techniques. Indwelling catheters theoretically increase the risk of infectious complications. However, although colonization may occur, infection is rare.[32] Risk factors appear to be similar to those associated with neuraxial blockade and include duration of catheterization, compromised immune status, and absence of antibiotic prophylaxis.[32,71]

Aseptic Technique

Although previous publications have repeatedly recommended meticulous aseptic technique, only recently have standards for asepsis during the performance of regional anesthetic procedures been defined.[72,73] Hand washing remains the most crucial component of asepsis; gloves should be regarded as a supplement to—not a replacement of—hand washing.[74] The use of an antimicrobial soap reduces bacterial growth and reduces the risk of bacteria being released into the operative field should gloves become torn or punctured during the procedure. An alcohol-based antiseptic provides the maximum degree of antimicrobial activity and duration. Prior to washing, all jewelry (eg, rings, watches) should be removed; higher microbial counts have been noted in health care workers who do not routinely remove these items before hand washing. Sterile gloves protect not only patients from contamination, but also health care workers from blood-borne pathogens and are required by the Occupational Safety and Health Administration. Glove leaks are more likely to occur with vinyl compared with latex gloves (24% vs 2%), with contamination of the health care workers' hands noted following the leaks in 23% of cases.[75] Conversely, the use of gowns does not further reduce the likelihood of cross contamination in an intensive

care unit setting compared with gloves alone. At this time, there are insufficient data to make recommendations regarding routine use for single injection or temporary neuraxial or peripheral catheter placement. However, placement of an indwelling permanent device, such as a spinal cord stimulator, warrants the same asepsis as a surgical procedure, including gowns, hats, and antibiotic pretreatment.[72,76] Surgical masks, initially considered a barrier to protect the proceduralist from patient secretions and blood, are now required by the Center for Disease Control[77] due to the increasing number of cases of post-spinal meningitis, many of which result from contamination of the epidural or intrathecal space with pathogens from the operator's buccal mucosa.[13,70,78]

Antiseptic solutions

Controversy still exists regarding the most appropriate and safe antiseptic solution for patients undergoing neuraxial and peripheral techniques. Povidone iodine and chlorhexidine gluconate (with or without the addition of isopropyl alcohol) have been most extensively studied.[79,80] In nearly all clinical investigations, the bactericidal effect of chlorhexidine was more rapid and more effective (extending its effect for hours following its application) than povidone iodine. The addition of isopropyl alcohol accelerates these effects. Chlorhexidine is effective against nearly all nosocomial yeasts and bacteria (gram-positive and gram-negative); resistance is extremely rare. It also remains effective in the presence of organic compounds, such as blood. It must be noted that chlorhexidine-alcohol labeling contains a warning against use as a skin preparation before lumbar puncture. The Food and Drug Administration has not formally approved chlorhexidine for skin preparation before lumbar puncture because of the lack of animal and clinical studies examining the neurotoxic potential of chlorhexidine—not because of the number of reported cases of nerve injury. Indeed, it is important to note that there are no cases of neurotoxicity with either chlorhexidine or alcohol.[72] Therefore, because of its superior effect, alcohol-based chlorhexidine solutions are considered the antiseptic of choice for skin preparation before any regional anesthetic procedure.[72,73]

REGIONAL BLOCK IN PATIENTS WITH PREEXISTING NEUROLOGIC DISORDERS

Patients with preexisting neurologic disease present a unique challenge to the anesthesiologist. The cause of postoperative deficits is difficult to evaluate, because neural injury may occur because of surgical trauma, tourniquet pressure, prolonged labor, improper patient positioning, or anesthetic technique. Progressive neurologic diseases such as multiple sclerosis may coincidentally worsen perioperatively, independent of the anesthetic method. The most conservative legal approach is to avoid regional anesthesia in these patients. However, high-risk patients, including those with significant cardiopulmonary disease, may benefit medically from regional anesthesia and analgesia. The decision to proceed with a regional anesthesia in these patients should be made on a case-by-case basis.

The presence of preexisting deficits, signifying chronic neural compromise, theoretically places these patients at increased risk for further neurologic injury. The presumed mechanism is a "double crush" of the nerve at two locations resulting in a nerve injury of clinical significance.[81] The double crush concept suggests that nerve damage caused by traumatic needle placement or local anesthetic toxicity during the performance of a regional anesthetic may worsen neurologic outcome in the presence of an additional patient factor or surgical injury.[8,9,11,82,83] Progressive neurologic diseases may also coincidentally worsen perioperatively, independent of the anesthetic method. If a regional anesthetic is indicated or requested, the patient's

preoperative neurologic examination should be formally documented and the patient must be made aware of the possible progression of the underlying disease process (**Box 4**).[20]

Multiple Sclerosis

It is difficult to define the relative risk of neurologic complications in patients with preexisting neurologic disorders who receive regional anesthesia; no controlled studies have been performed, and accounts of complications have appeared in the literature as individual case reports. Although laboratory studies have identified multiple risk

Box 4
Recommendations for performing regional anesthesia in patients with preexisting neurologic conditions

- Overall approach to patients with preexisting neurologic deficits
  - Patients with preexisting neurologic disease may be at increased risk of new or worsening injury regardless of anesthetic technique. When regional anesthesia is thought to be appropriate for these patients, modifying the anesthetic technique may minimize potential risk. Based on a moderate amount of animal data, such modifications may include using a less potent local anesthetic, minimizing local anesthetic dose, volume, and/or concentration, and avoiding or using a lower concentration of vasoconstrictive additives. Limited human data neither confirm nor refute these modifications.
- Preexisting peripheral neuropathy
  - Patients with chronic diabetes mellitus, severe peripheral vascular disease, multiple sclerosis, or previous exposure to chemotherapy (eg, cisplatin or vincristine) may have clinical or subclinical evidence of a preexisting peripheral neuropathy. Peripheral nerve block may theoretically increase the risk of new or progressive postoperative neurologic complications in these patients. However, existing data can neither confirm nor refute this theory in clinical practice. Under these clinical conditions, a careful risk-to-benefit assessment of regional anesthesia to alternative perioperative anesthesia and analgesia techniques should be considered.
- Preexisting CNS disorders
  - Definitive evidence indicating that neuraxial anesthesia or analgesia may increase the risk of new or progressive postoperative neurologic complications in patients with preexisting CNS disorders (eg, multiple sclerosis, postpolio syndrome) is lacking. However, under these clinical conditions, a careful risk-to-benefit assessment of regional anesthesia to alternative perioperative anesthesia and analgesia techniques should be considered.
- Spinal stenosis or mass lesions within the spinal canal
  - In patients with known severe spinal stenosis or mass lesions within the spinal canal, a careful risk-to-benefit assessment of regional anesthesia to alternative perioperative anesthesia and analgesia techniques should be considered. In these patients, high local anesthetic volume neuraxial techniques (eg, epidural anesthesia) may be associated with a higher risk of progressive mass effect when compared with low volume techniques (eg, spinal anesthesia).
  - For patients receiving neuraxial injection for treatment of pain (eg, cervical epidural injection of steroids via an interlaminar route), radiologic imaging studies such as computed tomography or magnetic resonance imaging should be used to assess the dimensions of the spinal canal, and this information should be considered in the overall risk-to-benefit analysis, as well as guiding the selection of the safest level for entry.

Adapted from Neal JM, Bernards CM, Hadzic A, et al. ASRA practice advisory on neurologic complications in regional anesthesia and pain medicine. Reg Anesth Pain Med 2008;3:404–15; with permission.

factors for the development of neurologic injury after regional anesthesia, clinical studies are lacking. Even less information is available for the variables affecting neurologic damage in patients with preexisting neurologic disease. The largest series of neuraxial anesthesia in the patient with a preexisting CNS condition involved 139 patients.[8] Postpolio syndrome and multiple sclerosis were the most common CNS disorders. The majority of patients had sensorimotor deficits at the time of block placement. There were no patients with new or worsening postoperative neurologic deficits when compared with preoperative findings (0.0%; 95% CI 0.0%–0.3%). The investigators concluded that the risks commonly associated with neuraxial block in patients with preexisting CNS disorders may not be as high as thought and that these conditions should not be an absolute contraindication to spinal or epidural techniques. Because multiple sclerosis is a disorder of the CNS, peripheral nerve blocks do not affect neurologic function and are considered appropriate anesthetic techniques. However, the clinician should be aware of the potential for the presence of an associated peripheral neuropathy (which exists in over 10% of patients).[84]

Diabetes Mellitus

A substantial proportion of diabetic patients report clinical symptoms of a neuropathy. However, a subclinical neuropathy may be present before the onset of pain, paresthesia, or sensory loss and may remain undetected without electrophysiologic testing showing typical slowing of nerve conduction velocity. The presence of underlying nerve dysfunction suggests that patients with diabetes may have a decreased requirement for local anesthetic. The diabetes-associated microangiopathy of nerve blood vessels decreases the rate of absorption, resulting in prolonged exposure to local anesthetic solutions. The combination of these two mechanisms may cause nerve injury with an otherwise safe dose of local anesthetic in diabetic patients. In a study examining the effect of local anesthetics on nerve conduction block and injury in diabetic rats, Kalichman and Calcutt[33] reported that the local anesthetic requirement is decreased and the risk of local anesthetic-induced nerve injury is increased in diabetes.

A recent retrospective review of 567 patients with a sensorimotor neuropathy or diabetic polyneuropathy who underwent neuraxial block evaluated the risk of neurologic complications. All patients had a single neurologic diagnosis; there were no coexisting spinal canal or CNS disorders.[9] The majority of patients had sensorimotor deficits at the time of surgery. Two (0.4%; 95% CI 0.1%–1.3%) patients experienced new or worsening postoperative neurologic deficits in the setting of uneventful neuraxial block and without surgical or positioning risk factors. In these patients, who had severe sensorimotor neuropathy preoperatively, it is likely the neuraxial technique contributed to the injury.

Spinal Stenosis and Lumbar Root Disease

Moen and colleagues[13] identified spinal stenosis as a risk factor for postoperative cauda equina syndrome and paraparesis. Importantly, deficits would often occur after uneventful neuraxial technique. These findings agree with those of a recent investigation that examined the overall success and neurologic complication rates among 937 patients with spinal stenosis or lumbar disc disease undergoing neuraxial block.[83] Two hundred seven patients had a history of prior spinal surgery before undergoing neuraxial block, although the majority were simple laminectomies or discectomies. Ten (1.1%; 95% CI 0.5%–2.0%) patients experienced new or progressive neurologic deficits when compared with preoperative findings. A surgical cause was presumed to be the primary cause in four of ten patients. The primary cause of the remaining six

complications was judged nonsurgical (including anesthetic-related factors). The investigators concluded that patients with a history of preexisting spinal stenosis or lumbar radiculopathy are at increased risk of neurologic complications following neuraxial blockade. Because the cause of the complications is likely multifactorial, until the relative contribution of existing patient and potential surgical contributing factors is known, the decision to perform neuraxial blockade in these patients should be made cautiously.

In general, patients with preoperative neurologic deficits may undergo further nerve damage more readily from needle or catheter placement, local anesthetic systemic toxicity, and vasopressor-induced neural ischemia. Consequently, when feasible, dilute or less potent local anesthetic solutions should be used in order to decrease the risk of local anesthetic toxicity. Because epinephrine and phenylephrine also prolong the block and, therefore, neural exposure to local anesthetics, the appropriate concentration and dose of local anesthetic solutions must be thoughtfully considered.[20]

REGIONAL ANESTHESIA IN ANESTHETIZED ADULTS

The actual risk of neurologic complications in patients undergoing regional techniques while anesthetized or heavily sedated has not been formally evaluated. However, epidemiologic series report direct trauma and toxicity as the causes of most neurologic complications and have identified pain during needle placement or injection of local anesthetic as major risk factors.[2,12,14] Thus, performance of regional blocks while the patient is under general anesthesia theoretically increases the risk of perioperative neurologic complications, since these patients are unable to respond to the pain associated with needle- or catheter-induced paresthesias or intraneural injections. Despite these findings, there are few data to support these concerns. Cases are typically reported individually; no randomized study or large review has been performed to date.[24,25] Importantly, the apparent safety of performing regional techniques under general anesthesia that is demonstrated in the pediatric literature must be carefully interpreted. There are also medicolegal issues.

Peripheral and plexus blocks (compared with neuraxial techniques) may represent additional risk when performed on an anesthetized patient. The larger dose of local anesthetic given as a single bolus over a relatively short interval increases the risk of systemic toxicity, whereas heavy sedation or general anesthesia diminishes the patient's ability to report early signs of rising local anesthetic blood levels. In addition, although some peripheral techniques are performed as a field block, most require that the nerve or sheath be directly identified by eliciting a paresthesia or nerve stimulator response or by locating an adjacent vascular structure. However, the use of a nerve stimulator or ultrasound does not replace the patient's ability to respond to the pain of needle trauma or intraneural injection. Urmey and Stanton[85] performed interscalene blocks on patients who were not already medicated using paresthesia techniques with insulated (10 patients) and noninsulated (20 patients) needles. Paresthesias were elicited with the nerve stimulator power off. Upon elicitation of the paresthesia, the nerve stimulator was turned on and the amperage slowly increased to a maximum of 1.0 mA. Only 30% of patients exhibited any motor response. Benumof[24] reported four cases of permanent cervical spinal cord injury following interscalene block performed with the patient under general anesthesia or heavy sedation. In three cases, a nerve stimulator was used to localize the brachial plexus. These results suggest that since it is possible to have sensory nerve contact and not elicit a motor response, use of a nerve stimulator (and unpublished data associated with ultrasound guided blocks) does not

protect the anesthetized patient from nerve injury.[20] Thus, the decision to perform a regional anesthetic on a heavily sedated or anesthetized patient should not be made indiscriminately.

SUMMARY

In conclusion, major complications after regional anesthetic techniques are rare, but can be devastating to the patient and the anesthesiologist. Prevention and management begin during the preoperative visit with a careful evaluation of the patient's medical history and appropriate preoperative discussion of the risks and benefits of the available anesthetic techniques. The decision to perform a regional anesthetic technique on an anesthetized patient must be made with care since these patients are unable to report pain on needle placement or injection of local anesthetic. Efforts should also be made to decrease neural injury in the operating room through careful patient positioning. Postoperatively, patients must be followed closely to detect potentially treatable sources of neurologic injury, including constrictive dressings, improperly applied casts, and increased pressure on neurologically vulnerable sites. New neurologic deficits should be evaluated promptly by a neurologist, or neurosurgeon, to formally document the patient's evolving neurologic status, arrange further testing or intervention, and provide long-term follow-up.[20]

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