

Case Scenario: Nerve Injury after Knee Arthroplasty and Sciatic Nerve Block

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TOTAL knee arthroplasty is one of the most common lower extremity surgeries performed in the United States for progressive pain caused by severe arthritis. Anesthetic management for postoperative pain includes intermittent intravenous opioids and patient-controlled analgesia as well as peripheral nerve blockade. Although peripheral nerve blocks have allowed for a reduction of opioid systemic side effects, perioperative nerve injury can be a distressing complication. Anesthetic- or surgical-related etiologies should be investigated, and patient-specific risk factors such as preexisting neuropathy, diabetes mellitus, extremes of body habitus, male gender, and advanced age¹ taken into consideration.

Case Scenario

Preoperative Management

A 60-yr-old, 104-kg, 71-inch-tall man presented for a left total knee arthroplasty for degenerative joint disease. His medical history included hypertension, chronic obstructive pulmonary disease, gout, and a 40-pack-year smoking history. His home medications included naproxen, atenolol, and allopurinol. Naproxen had been discontinued 1 week before surgery. Results of preoperative neurologic exam were

normal. Examination by the orthopedic surgeon noted the absence of a valgus deformity or flexion contracture.

In the preoperative holding area, sciatic and femoral nerve catheters were placed for postoperative pain management. Before placement of the blocks, 100 µg fentanyl and 2 mg midazolam were administered intravenously. With the patient lying on his right side, the operative leg in the uppermost position and rolled forward with the knee flexed at a 90-degree angle (modified Sims' position), the sciatic nerve block was performed first using the classic Labat approach. (A line was drawn from the posterior superior iliac spine to the greater trochanter, and a second line was drawn perpendicularly from the midpoint and extended caudally 5 cm. This point was the site of needle insertion.) Using a peripheral nerve stimulator, the sciatic nerve was located by eliciting plantar flexion with a Contiplex® 10-inch, 18 gauge short bevel needle (B. Braun Melsungen AG, Melsungen, Germany); muscle twitch was lost at 0.48 mA. Twenty milliliters ropivacaine (0.5%) with epinephrine (0.005 mg/ml) was injected incrementally, without report of pain or paresthesia. A perineural 20-gauge epidural catheter was easily threaded using catheter-through-needle technique and secured 5 cm deep to the skin. The patient then turned supine, and a left femoral nerve block was performed. One centimeter lateral to the femoral artery pulse and 2 cm inferior to the inguinal ligament, the needle was advanced in the sagittal plane. The femoral nerve was identified by quadriceps contraction with the same peripheral nerve stimulator. The twitch was lost at 0.46 mA, at which point 35 ml ropivacaine (0.5%) with epinephrine (0.005 mg/ml) was injected in a similar manner. A 20-gauge epidural catheter was threaded and secured 5 cm deep to the skin.

Intraoperative Management

The patient was transferred to the operating room, where a subarachnoid block was performed for surgical anesthesia using a 25-gauge Whitacre pencil-point spinal needle (Becton, Dickinson, Franklin Lakes, NJ) through which 1.2 ml hyperbaric bupivacaine (0.75%) was injected. A T10 level of anesthesia was attained. The patient was positioned supine for surgery, and a tourniquet was applied to the left thigh.

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Before surgical incision, the tourniquet was inflated to a pressure of 250 mmHg. The surgery was completed uneventfully. Total tourniquet time was 46 min.

Postoperative Management

In the recovery room, infusions in each catheter were begun immediately after surgery; the catheters were test dosed with no intravenous symptoms, and bupivacaine (0.25%) infusion was begun in each catheter at a rate of 7 ml/h. On the morning of postoperative day 1, the local anesthetic infusions were discontinued, and both catheters were removed to optimize physical therapy. On the afternoon of postoperative day 1, after presumed recovery from any residual effects of local anesthetic, physical exam by the hospital's acute pain service was notable for a left lower extremity motor strength score of 3 of 5 (Medical Research Council scale), with the exception of ankle dorsiflexion, which was absent. The patient reported no pain. There was no tenderness or ecchymosis at the sites of nerve block injection. On postoperative day 2, the patient reported pain on the plantar surface of the left foot, pain over the posterior left thigh, and a left foot drop was noted again. The patient continued physical therapy for the remainder of his inpatient stay and was discharged home on postoperative day 4, without improvement in motor examination. An outpatient appointment was made with neurology during the week after discharge, but the patient failed to appear. Despite several telephone call reminders, he did not follow up.

Outpatient Follow-up

Three and one half weeks later, the patient presented to the emergency department reporting pain and weakness in the left leg and foot. Physical exam revealed a warm left lower extremity with mild swelling, absent dorsiflexion, and decreased sensation over both the medial and lateral aspect of the foot. A complex regional pain syndrome type one was considered in the emergency department; gabapentin was prescribed by the emergency department physician, and plans were made for the patient to follow up with a neurologist the next week. At the neurologist's appointment, the patient still had unresolved lower extremity symptoms, although the plantar surface foot pain had been replaced by numbness over the dorsum of the foot and occasional shooting pains up the posterior aspect of the leg and thigh. On physical exam, there was decreased sensation to pinprick on the dorsum of the foot, weakness of both foot dorsiflexion (score 0 of 5) and plantar flexion (score 3 of 5), mild weakness (score 4— of 5) of the hamstrings, and absence of the left ankle deep tendon reflex. Electrodiagnostic testing demonstrated absent left sural and superficial peroneal sensory responses, and absent left peroneal (extensor digitorum brevis and tibialis anterior recording) and tibial motor responses. The right sural sensory response was normal. Needle examination showed increased insertional activity in the tibialis anterior without recruitable motor units. The left medial gastrocnemius and the long head of the biceps femoris

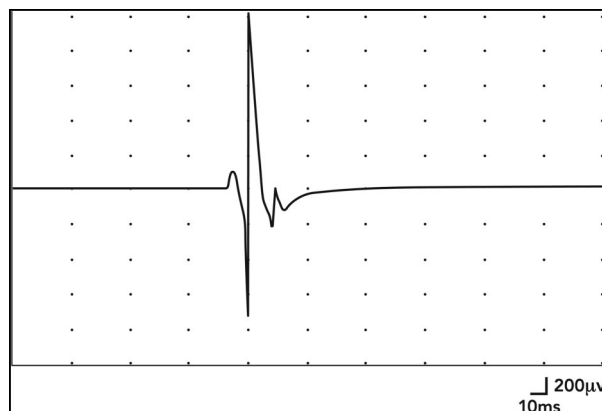


Fig. 1. Electroneuromyographic concentric needle examination recording demonstrating polyphasic, prolonged duration, and increased amplitude motor unit potential. ms = milliseconds; μ V = microvolts.

showed decreased recruitment of normal morphology motor units (fig. 1). The vastus lateralis, gluteus medius and maximus, and low lumbosacral paraspinals were normal. Both clinical examination and electrodiagnostic testing pointed to a sciatic mononeuropathy in the mid-thigh.

Postoperative plain radiographs of the femur showed vascular calcifications in the vessels of the thigh (fig. 2). Contrast-enhanced magnetic resonance imaging of the thigh was performed. There was no sciatic nerve abnormality, but there was an abnormal signal in the mid- to distal femur that was consistent with a bone infarct (fig. 3).

The patient continued physical therapy. By 1 yr after surgery, he had a score of 4 of 5 dorsiflexion strength, was able to walk independently, and had only mild sensory loss over the dorsum of the foot.



Fig. 2. Lateral radiograph of the knee and femur showing intact prosthesis and no significant lower femur lucencies. Arrows indicate multiple vascular calcifications.

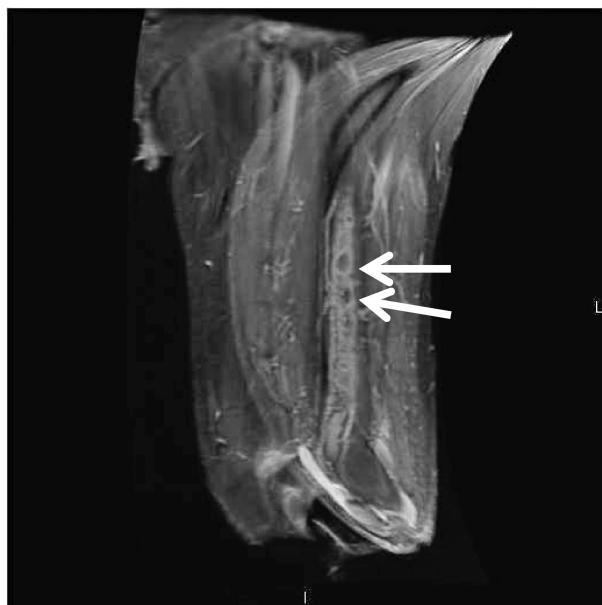


Fig. 3. Coronal fast spin-echo inversion-recovery magnetic resonance imaging sequence of the left thigh, showing abnormal mid- to distal femur medullary serpiginous signal consistent with a bone infarct. Arrows indicate region of bone infarct.

Discussion

“Foot drop” is a colloquial term that aptly describes the most striking physical finding of peroneal nerve injury. Compensation for weakness of foot dorsiflexion results in the characteristic high-stepping gait used to prevent tripping over the toes. Weakness of foot eversion and toe extension is also present. This clinical picture can be caused by a lesion anywhere along the course of the L5 nerve fascicles, from the anterior horn cells to the peroneal nerve itself. For example, damage to the sciatic nerve can result in foot drop if there is injury to the lateral trunk because this trunk becomes the peroneal nerve.²

Peroneal nerve injury causing foot drop is a well-known complication of total knee arthroplasty. Injury in this setting can be caused by compression, ischemia, traction, crush, or laceration. The incidence of this injury based on more recent observations is estimated to be 0.79%.^{3,4} One of the most commonly suggested risk factors is a valgus deformity of the joint greater than or equal to 10 degrees; intraoperative correction of the deformity results in traction on the nerve. A preoperative flexion contracture of the knee also increases stretch on the nerve after surgery and is a predisposing factor.^{5–8} A baseline neuropathy increases risk,^{9,10} and preoperative neurologic evaluation has been recommended so that surgical technique can be adjusted if a known underlying neuropathy is present.⁹ Other predisposing factors may include tourniquet time longer than 120 min, rheumatoid arthritis, constrictive dressings, postoperative bleeding,^{3,11} and previous lumbar laminectomy.⁷ One study identified tourniquet pressures of greater than 400 mmHg as a predisposing factor.¹² The numerous factors that influence the risk

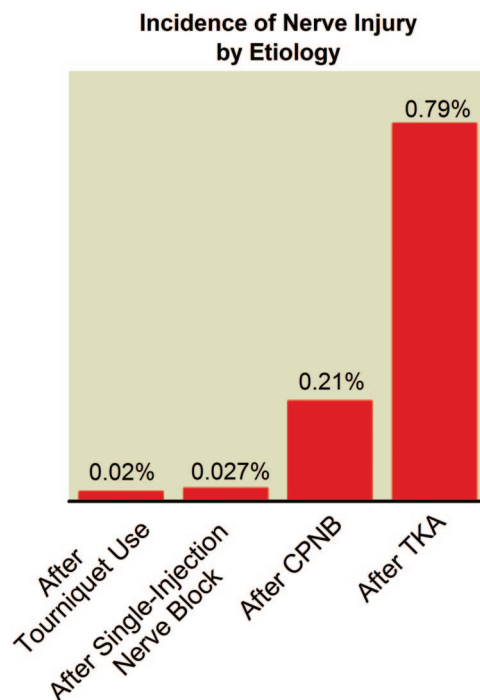


Fig. 4. Maximum estimated incidences of nerve damage after tourniquet use,²² single-injection peripheral nerve block,¹⁴ continuous peripheral nerve block (CPNB),¹⁵ and total knee arthroplasty (TKA).⁴ The higher incidence of foot drop after total knee arthroplasty may be explained by the multiple etiologies of injury possible during this surgery.

of nerve injury after knee arthroplasty can make the determination of a single cause of injury challenging. It is likely that a combination of these factors explains the much higher incidence of nerve injury after total knee arthroplasty than is associated with regional nerve block or tourniquet use alone (fig. 4).

The estimated incidence of neuropathy after peripheral nerve blockade varies depending on the site of block and other factors. One recent meta-analysis reported an incidence of approximately 3% for interscalene, axillary, and femoral nerve blocks combined; permanent neurologic injury was rare.¹³ Serious neurologic injury in one large prospective study occurred in 0.027% of peripheral nerve blocks.¹⁴ Continuous nerve block may be associated with a somewhat higher risk of neurologic complications, with one group reporting an incidence of 0.21% after continuous femoral nerve block.¹⁵ The risk of injury from sciatic nerve block in particular is not well established. The paucity of injury reports may be the result of lower relative numbers of these blocks; the lack of easily searchable, consistent records of peripheral nerve block placement in many institutions; and the low frequency of regional block-related neuropathy, especially in the lower extremity. One group reported the incidence of neuropathy after sciatic block as 0.41% based on three studies; this incidence is likely overestimated by the inclusion of one study that reported a 2.07% incidence in both sciatic and femoral nerve blocks.¹³ A large prospective study reported an incidence of 0.024%.¹⁴

Sciatic nerve blockade, like other peripheral nerve blocks, can cause neurologic injury from needle trauma, intraneural injection of local anesthetic, neuronal ischemia, and neurotoxicity of local anesthetic. True symptoms of neuropathy, which include allodynia, hyperpathia, and hyperesthesia, must be distinguished from perceived nerve injury resulting in paresthesia. Lack of pain on injection is not a reliable indicator of safety; resistance on injection has been suggested to be more indicative of intraneural placement of the needle.¹⁶

An epidural in place has been implicated as a risk factor for peroneal nerve injury after total knee arthroplasty.^{3,7,8,17} It has been proposed that the sensory (and especially proprioceptive) deficits of this technique may result in patient tolerance of either unsafe leg position or overly constrictive dressings. Epidural anesthesia is also suspected of delaying the diagnosis of nerve injury of other etiologies.⁷ If epidural anesthesia is a predisposing factor for perioperative peroneal nerve injury, it may be of value to consider the risks of the sensory deficits of catheter-based sciatic nerve block. Our patient received a spinal block for the operation, which should not have masked any postoperative neurologic injury because of its short duration of action compared with the peripheral nerve blocks. However, intravenous sedation with benzodiazepines and opioids could mask the patient's perception of pain or paresthesia in the presence of intraneural injection.

The etiology of the nerve injury has implications for prognosis and treatment and is of medicolegal interest because nerve injuries represent approximately one third of all anesthesia-associated claims in the United States.¹⁸ Axonal preservation portends improved outlook for recovery. Compressive or stretch injuries are often associated with damage to the myelin sheath only and may be more likely to resolve. If the axon is disrupted, as might be the case with direct needle trauma or from ischemia, recovery is slower and perhaps less complete.¹⁹ Injection of local anesthetic can cause time- and concentration-dependent injury, which is additive to any other neural injury.²⁰ The type of local anesthetic used may also influence risk of nerve injury. For example, the literature suggests that ropivacaine may cause a reduction in peripheral nerve blood flow without significant histopathologic changes,²¹ but it may cause additional dysfunction in a predisposed nerve. Although our patient's nerve injury was distant from the site of needle placement, in cases of nerve injury near the site of needle placement, the type of local anesthetic injected may be of relevance when considering factors influencing injury.

In this case, the importance of imaging is highlighted because it suggests the most likely location, and thus the etiology, of injury. Electrodiagnostic studies supported the clinical impression of sciatic nerve injury, rather than isolated peroneal nerve injury. Given the plain radiograph findings, the possibility of an ischemic lesion affecting the watershed area of the sciatic nerve became apparent. Magnetic resonance imaging of the thigh did not show any sciatic nerve

abnormalities, although the resolution may not have been adequate for this purpose; however, the signal abnormalities did point to an ischemic bone lesion in the mid-thigh, which supported the idea of an ischemic sciatic mononeuropathy.

The imaging findings suggested intraoperative tourniquet use as a contributor to injury. This was surprising given the short tourniquet time. Clinically manifested tourniquet injury is rare, with an estimated overall incidence of 0.01–0.02%.^{22,23} Subsequent investigations have determined that subclinical postoperative electromyographic abnormalities are much more common than previously thought; one group of post-knee-arthroscopy patients demonstrated a 71% incidence of asymptomatic denervation.²⁴

Tourniquet injury after total knee arthroplasty has been studied after prolonged tourniquet times of greater than 2 h. The incidence of injury is significantly increased in this circumstance, with one estimate being 7.7% for combined peroneal and tibial nerve palsies.¹¹ The mechanism is thought to be both ischemia and mechanical trauma.

In larger series of sciatic mononeuropathies, sciatic neuropathy caused specifically by tourniquet was not found to be a common etiology. There are two series of sciatic mononeuropathies published.^{25,26} In one retrospective series of 73 patients that focused on clinical and prognostic features, 16 (21.9%) patients were status post total hip replacement without mention of type of anesthesia. Only one (1.4%) case was secondary to pneumatic thigh tourniquet compression. Seven (9.6%) had nerve infarction: five in the setting of vasculitis, one with diabetic mononeuropathy, and one in the setting of common iliac artery thrombosis. In the other series of 100 patients, which focused more on electrodiagnostic features, the authors noted that 93% of cases had axonal loss on electromyographic testing. Only seven patients had demyelinating lesions: six compressive and one idiopathic. The peroneal division of the sciatic nerve was affected more than the tibial division in 64% of cases. Overall, the clinical recovery was 30% by 1 yr, 50% by 2 yr, and 75% by 3 yr. A long and incomplete recovery was predicted by the absence or low amplitude of a motor-evoked response recorded from the extensor digitorum brevis and the initial absence of foot plantar and dorsiflexion on initial examination.

Underlying vascular compromise may have made our patient more vulnerable to tourniquet injury, despite a tourniquet time of only 46 min. He had numerous risk factors for peripheral arterial disease, including age, obesity, history of smoking, and hypertension. The vascular calcifications on thigh radiography also strengthened the argument for underlying vascular disease contributing to injury.

Knowledge Gap and Recommendations

Because postoperative neuropathy of any cause is of concern to the anesthesiologist, it is prudent to evaluate patients' risk factors for subclinical neuropathy before regional blocks. Although a single institution cohort study suggested that age, type of surgical procedure, and total tourniquet time were

the main factors associated with perioperative nerve injury,⁴ other factors, such as uremia, human immunodeficiency virus, alcohol and smoking habits, and previous chemotherapeutic agent use, may contribute to such a neuropathy. Age is a risk factor for axonal degeneration, which in turn decreases conduction velocity in both motor and sensory nerve fibers.²⁷ It has also been shown that the duration of local anesthetic action in the elderly patient population (age older than 70 yr) is prolonged.²⁸ Diabetes could place a patient at higher risk for nerve injury. A small study revealed 100% intraneural injection of the sciatic nerve in hyperglycemic dogs when using low electrical stimulation threshold.²⁹ Certain surgical specialties, such as neurosurgery, cardiac surgery, general surgery, and orthopedic surgery, have also been shown to be associated with perioperative peripheral nerve injury.³⁰

Reducing the dose, concentration, or potency of local anesthetic and eliminating or reducing the concentration of vasoconstrictive additives are also potential considerations.^{2,31} Careful and well-documented preoperative neurologic exam and clear communication with the surgeon as to the anticipated difficulty of surgery will aid in making an anesthetic plan. Obvious surgical risk factors for nerve injury, such as valgus deformity and flexion contracture, should be identified.

Although awareness of risk factors may alert the practitioner to an increased likelihood of subclinical neuropathy, evaluation for peripheral vascular disease (ankle-brachial indices), blood testing for thyroid dysfunction and vitamin deficiency, and procedures such as electromyography and nerve biopsies are all impractical and cost prohibitive for preoperative evaluation in every at-risk patient. A practical recommendation for the regionalist would be to obtain a careful history for any symptoms (burning, numbness, paresthesias, pain, paresis, or claudication) that may be associated with neuropathy and perform a more thorough neurologic exam before block placement if these are present. In addition, confirmation of a repeat normal neurologic exam before starting postoperative peripheral nerve catheter infusions may be helpful in early identification of postoperative nerve injury. If an acute peripheral neuropathy is identified, early imaging by computed tomography or ultrasound should be performed to rule out local hematoma. Electromyographic assessment for the integrity of the nerve should be performed, with timing of this study guided by input obtained from the neurologic consult. Recovery of function can take as long as 2 yr, depending on the level of the lesion and the severity of damage.³²

The use of ultrasound may prove to be another method for reducing the incidence of nerve injury. The increasing adoption of ultrasound guidance for peripheral nerve block placement is an important advance in regional anesthesia, and this technique has been shown to have several advantages when used for nerve localization, including speed of block onset and block success rate.³³ Although ultrasound guidance likely does increase safety,³⁴ this has not yet been

shown; future directions for research may include the demonstration of superior safety with its use.

This case also highlights the importance of a careful, detailed preoperative evaluation; future research exploring the importance of risk factors for subclinical neuropathy and the influence of tourniquet times and pressures using preoperative and postoperative electroneuromyographic recordings may provide anesthesiologists and surgeons with methods to reduce injury in higher-risk patients.

Neurologist's Comments

1. *Why did the neurologic exam show a score of 3 of 5 plantar flexion weakness with no recruitable tibial motor response?*

The inability to record motor potentials despite clinical evidence for muscle contraction was technical in nature, influenced by the patient's leg swelling that was noted clinically. Leg edema may make recordings of the sensory and motor responses more difficult and may overestimate the degree of nerve function loss. This was confirmed by the ability to elicit muscle contraction during needle examination. This ultimately conferred a better chance for recovery of function in our patient.

2. *Tell us about the pathophysiology of nerve injury and specifically of tourniquet-induced nerve injury.*

The pathophysiology of compressive nerve injury is complex. The anatomopathologic classification of focal nerve injuries by Seddon³⁵ and Sunderland³⁶ takes into consideration the epineurium, endoneurium, and perineurium. It describes a continuum of nerve injury, from neurapraxia to axonotmesis to neurotmesis, with progressive involvement of the three nerve compartments. This classification has no good correlate in clinical or electrodiagnostic diagnosis because lesions to these supporting tissues do not directly affect myelin or axon function. For prognostic purposes, we separate focal neuropathies into demyelinating and axonal lesions, and try to determine the degree of axonal loss. The ischemic theory of nerve injury has dominated the literature after the classic experiments of pressure cuff inflation by Lewis in the 1930s.³⁷ However, more recent experiments in the 1970s³⁸ suggested that the main pathophysiology is one of compression, with nodes of Ranvier and paranodal myelin displacement as main causes of electrical conduction failure. Microvascularization experiments³⁹ also suggest that the endoneurial vascular flow remains well preserved in focal crush injuries, making the ischemic theory even less likely.

3. *What is the role of early electromyography, ultrasound, and other imaging in the workup of possible peripheral nerve injury?*

Early electromyography is rarely performed because the results may not be able to differentiate between demyelination and early axonal loss and may miss early axonal loss

if done too early. For a lower extremity injury, approximately 3 weeks after the injury is about right to pursue electromyographic studies. Early imaging is definitely recommended. Ultrasound may not be adequate to rule out local hematoma; computed tomography is probably superior for this purpose.

References

1. Neal JM, Hebl JR, Gerancher JC, Hogan QH: Brachial plexus anesthesia: Essentials of our current understanding. *Reg Anesth Pain Med* 2002; 27:402-28
2. Stewart JD: Foot drop: Where, why and what to do? *Pract Neurol* 2008; 8:158-69
3. Nercessian OA, Ugwonalie OF, Park S: Peroneal nerve palsy after total knee arthroplasty. *J Arthroplasty* 2005; 20:1068-73
4. Jacob AK, Mantilla CB, Sviggum HP, Schroeder DR, Pagnano MW, Hebl JR: Perioperative nerve injury after total knee arthroplasty: Regional anesthesia risk during a 20-year cohort study. *ANESTHESIOLOGY* 2011; 114:311-7
5. Rose HA, Hood RW, Otis JC, Ranawat CS, Insall JN: Peroneal-nerve palsy following total knee arthroplasty: A review of The Hospital for Special Surgery experience. *J Bone Joint Surg Am* 1982; 64:347-51
6. Asp JP, Rand JA: Peroneal nerve palsy after total knee arthroplasty. *Clin Orthop* 1990; 261:233-7
7. Idusuyi OB, Morrey BF: Peroneal nerve palsy after total knee arthroplasty. Assessment of predisposing and prognostic factors. *J Bone Joint Surg Am* 1996; 78:177-84
8. 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
9. Dellon AL: Postarthroplasty 'palsy' and systemic neuropathy: A peripheral-nerve management algorithm. *Ann Plast Surg* 2005; 55:638-42
10. Blumenthal S, Borgeat A, Maurer K, Beck-Schimmer B, Kliesch U, Marquardt M, Urech J: Preexisting subclinical neuropathy as a risk factor for nerve injury after continuous ropivacaine administration through a femoral nerve catheter. *ANESTHESIOLOGY* 2006; 105:1053-6
11. Horlocker TT, Hebl JR, Gali B, Jankowski CJ, Burkle CM, Berry DJ, Zepeda FA, Stevens SR, Schroeder DR: Anesthetic, patient, and surgical risk factors for neurologic complications after prolonged total tourniquet time during total knee arthroplasty. *Anesth Analg* 2006; 102:950-5
12. Fanelli G, Casati A, Garancini P, Torri G: Nerve stimulator and multiple injection technique for upper and lower limb blockade: Failure rate, patient acceptance, and neurologic complications. Study Group on Regional Anesthesia. *Anesth Analg* 1999; 88:847-52
13. Brull R, McCartney CJ, Chan VW, El-Beheiry H: Neurological complications after regional anesthesia: Contemporary estimates of risk. *Anesth Analg* 2007; 104:965-74
14. Auroy Y, Benhamou D, Bargues L, Ecoffey C, Falissard B, Mercier FJ, Bouaziz H, Samii K, Mercier F: Major complications of regional anesthesia in France: The SOS Regional Anesthesia Hotline Service. *ANESTHESIOLOGY* 2002; 97:1274-80
15. Capdevila X, Pirat P, Bringuier S, Gaertner E, Singelyn F, Bernard N, Choquet O, Bouaziz H, Bonnet F, French Study Group on Continuous Peripheral Nerve Blocks: Continuous peripheral nerve blocks in hospital wards after orthopedic surgery: A multicenter prospective analysis of the quality of postoperative analgesia and complications in 1,416 patients. *ANESTHESIOLOGY* 2005; 103:1035-45
16. Hadzic A, Vloka JD: *Peripheral Nerve Blocks*. New York, McGraw-Hill, 2004, pp 67-77
17. Cohen DE, Van Duker B, Siegel S, Keon TP: Common peroneal nerve palsy associated with epidural analgesia. *Anesth Analg* 1993; 76:429-31
18. Kroll DA, Caplan RA, Posner K, Ward RJ, Cheney FW: Nerve injury associated with anesthesia. *ANESTHESIOLOGY* 1990; 73:202-7
19. Sorenson EJ: Neurological injuries associated with regional anesthesia. *Reg Anesth Pain Med* 2008; 33:442-8
20. Neal JM, Bernards CM, Hadzic A, Hebl JR, Hogan QH, Horlocker TT, Lee LA, Rathmell JP, Sorenson EJ, Suresh S, Wedel DJ: ASRA Practice Advisory on Neurologic Complications in Regional Anesthesia and Pain Medicine. *Reg Anesth Pain Med* 2008; 33:404-15
21. Bouaziz H, Iohom G, Estèbe JP, Campana WM, Myers RR: Effects of levobupivacaine and ropivacaine on rat sciatic nerve blood flow. *Br J Anaesth* 2005; 95:696-700
22. Odinson A, Finsen V: Tourniquet use and its complications in Norway. *J Bone Joint Surg Br* 2006; 88:1090-2
23. Middleton RW, Varian JP: Tourniquet paralysis. *Aust N Z J Surg* 1974; 44:124-8
24. Dobner JJ, Nitz AJ: Postmeniscectomy tourniquet palsy and functional sequelae. *Am J Sports Med* 1982; 10:211-4
25. Yuen EC, So YT, Olney RK: The electrophysiologic features of sciatic neuropathy in 100 patients. *Muscle Nerve* 1995; 18:414-20
26. Yuen EC, Olney RK, So YT: Sciatic neuropathy: Clinical and prognostic features in 73 patients. *Neurology* 1994; 44:1669-74
27. Dorfman LJ, Bosley TM: Age-related changes in peripheral and central nerve conduction in man. *Neurology* 1979; 29:38-44
28. Hanks RK, Pietrobon R, Nielsen KC, Steele SM, Tucker M, Warner DS, King KP, Klein SM: The effect of age on sciatic nerve block duration. *Anesth Analg* 2006; 102:588-92
29. Rigaud M, Filip P, Lirk P, Fuchs A, Gemes G, Hogan Q: Guidance of block needle insertion by electrical nerve stimulation: A pilot study of the resulting distribution of injected solution in dogs. *ANESTHESIOLOGY* 2008; 109:473-8
30. Welch MB, Brummett CM, Welch TD, Tremper KK, Shanks AM, Gugliani P, Mashour GA: Perioperative peripheral nerve injuries: A retrospective study of 380,680 cases during a 10-year period at a single institution. *ANESTHESIOLOGY* 2009; 111:490-7
31. Neal JM: Effects of epinephrine in local anesthetics on the central and peripheral nervous systems: Neurotoxicity and neural blood flow. *Reg Anesth Pain Med* 2003; 28:124-34
32. Borgeat A, Aguirre J, Curt A: Case scenario: Neurologic complication after continuous interscalene block. *ANESTHESIOLOGY* 2010; 112:742-5
33. Walker KJ, McGrattan K, Aas-Eng K, Smith AF: Ultrasound guidance for peripheral nerve blockade. *Cochrane Database Syst Rev* 2009; 4:CD006459
34. Lupu CM, Kiehl TR, Chan VW, El-Beheiry H, Madden M, Brull R: Nerve expansion seen on ultrasound predicts histologic but not functional nerve injury after intraneural injection in pigs. *Reg Anesth Pain Med* 2010; 35:132-9
35. Seddon HJ: A classification of nerve injuries. *Br Med J* 1942; 2(4260):237-9
36. Sunderland S: A classification of peripheral nerve injuries producing loss of function. *Brain* 1951; 74:491-516
37. Lewis T, Pickering GW, Rothschild P: Centripetal paralysis arising out of arrested bloodflow to the limb, including notes on a form of tingling. *Heart* 1931; 16:1-32
38. Ochoa J, Danta G, Fowler TJ, Gilliat RW: Nature of the nerve lesion caused by a pneumatic tourniquet. *Nature* 1971; 233:265-6
39. Xu Q, Midha R, Zochodne DW: The microvascular impact of focal nerve trunk injury. *J Neurotrauma* 2010; 27:639-46