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Brachial Plexus Injury as a Complication after Nerve Block or Vessel Puncture

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Brachial plexus injury is a potential complication of a brachial plexus block or vessel puncture. It results from direct needle trauma, neurotoxicity of injection agents and hematoma formation. The neurological presentation may range from minor transient pain to severe sensory disturbance or motor loss with poor recovery. The management includes conservative treatment and surgical exploration. Especially if a hematoma forms, it should be removed promptly. Comprehensive knowledge of anatomy and adept skills are crucial to avoid nerve injuries. Whenever possible, the patient should not be heavily sedated and should be encouraged to immediately inform the doctor of any experience of numbness/paresthesia during the nerve block or vessel puncture. (Korean J Pain 2014; 27: 210-218)

Key Words:

brachial plexus, brachial plexus neuropathies, nerve block, subclavian vein.

INTRODUCTION

The brachial plexus block (BPB) is a popular technique for providing operative anesthesia and pain control of the upper extremities [1–3]. Also subclavian or jugular vein catheterization is widely performed by anesthesiologists [4,5]. However, these procedures are not always safe and may cause various complications including brachial plexus injury (BPI) [6–9]. Additionally, the axillary arteriography, which has been used if the femoral route is not available, may also cause BPI [10].

Nerve injury is a serious complication. The patient with BPI may suffer only minor transient pain. However, the injury may result in permanent sensory disturbance or motor loss with poor recovery [4,11]. This paper presents literature reviews of BPI as a complication after BPB or vessel puncture including mechanism, clinical course, management and methods for prevention.

METHODS

A PubMed search was performed from 1950 to 2014 using the search terms brachial plexus, brachial plexus injury, brachial plexus neuropathies, brachial plexus block, nerve block, and different structures relevant to this review including subclavian vein, jugular vein and axillary artery.

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ANATOMY OF THE BRACHIAL PLEXUS

The brachial plexus is formed by the union of the anterior primary divisions (ventral rami) of the C5–C8 and T1 spinal nerves with variable contributions from the C4 and T2 nerves. As the nerve roots leave the intervertebral foramina, they form trunks, divisions, cords, branches and terminal nerves, in that order [12]. It is important to understand how the brachial plexus provides sensory and motor innervations to the upper limbs (Table 1) [13,14].

If the BPI is to happen during BPB or vessel puncture, it could be more common in distal nerves to the intraforaminal dorsal root ganglion. A supraclavicular injury usually occurs at the root and trunk levels, while an infraclavicular injury typically occurs at distal to the cord level.

INCIDENCE AND DATA

1. Brachial plexus block

Several studies have evaluated the incidence of postoperative neurological symptoms after BPB for surgery (Table 2). The incidences are quite variable and may be influenced by the used methods to identify neurologic symptoms.

The locus of BPB seems to influence the incidence of nerve injury. Fanelli et al. [7] prospectively studied interscalene blocks (n = 171) and axillary blocks (n = 1,650) using multiple injection technique with a nerve stimulator. The relative incidence of neurologic dysfunction was higher in patients receiving interscalene blocks (4%) than in patients receiving axillary blocks (1%).

Seeking paresthesia during a nerve block may increase the risk of post-anesthetic neurological sequelae in itself. Selander et al. [15] studied the frequency of postanesthetic nerve lesions after axillary BPB with/without searching for paresthesia. They found that all patients with nerve injury had reported painful paresthesia during the blocking procedure. Ultrasonographic guidance may improve the success rate and reduce BPB-related seizures [16,17]. However, it is unclear if it can actually reduce the incidence of neurological sequelae.

Patients on heavy sedation or general anesthesia may be at increased risk of nerve injury. Ben-David et al. [18] investigated adult patients (aged > 14 years) undergoing

Table 1. Nerves of the Brachial Plexus

Level	Nerve	Origin	Motor	Sense
Root	Long thoracic nerve	C5, C6, C7	Serratus anterior muscle	
	Dorsal scapular nerve	C4, C5	Levator scapulae and rhomboid muscles	
	Branch to phrenic nerve	C5	Diaphragm	
Trunk	Suprascapular nerve	C5, C6	Supraspinatus and infraspinatus muscles	
	Nerve to the subclavius	C5, C6	Subclavius muscle	
Cord	Lateral pectoral nerve	C5, C6, C7	Pectoralis major and pectoralis minor muscles	
	Medial pectoral nerve	C8, T1	Pectoralis major and pectoralis minor muscles	
	Thoracodorsal nerve	C6, C7, C8	Latissmus dorsi muscle	
	Subscapular nerves	C5, C6	Subscapularis and teres major muscles	
Branch	Axillary nerve	C5, C6	Deltoid and teres minor muscles	Deltoid region and superior posterior arm
	Musculocutaneous nerve	C5, C6, C7	Anterior compartment of the arm (flexion of the elbow)	Lateral forearm
	Radial nerve	C5, C6, C7, C8, T1	Posterior compartment of the arm and forearm (extension of the elbow, wrist and digits)	Posterior arm and forearm Lateral two-thirds of dorsum of hand and fingers
	Median nerve	C5, C6, C7, C8, T1	Anterior compartment of the forearm (flexion of the of the wrist and digits) with two exceptions (flexor carpi ulnaris and ulnar half of flexor digitorum profundus)	Lateral two-thirds of palm of hand and fingers
	Ulnar nerve	C8, T1	Most of the intrinsic muscles of the hand Flexor carpi ulnaris and ulnar half of flexor digitorum profundus	Medial surface of dorsum and palm of hand

Table 2. Incidences of the Brachial Plexus Injuries after Brachial Plexus Blocks

Author	Method	Number of patients to be investigated	Incidence of neurologic symptoms	Time to resolution of neurologic symptoms
Interscalene brachial plexus block				
Urban and Urquhart [6]	Paresthesia	266	9% on the first day 3% at 2 weeks	Within 6 weeks (except 1 patient)
Fanelli et al. [7]	Nerve stimulation	171	4% within the first month	Within 3 months
Orebaugh et al. [17]	Nerve stimulation	892	0% (permanent injury)	
	Ultrasound with a nerve stimulator	1,093	0% (permanent injury)	
Supraclavicular brachial plexus block				
Moberg and Dhuner [54]	Paresthesia	300	5.7%	Within 3 weeks (except 7 patients)
Liu et al. [55]	Ultrasound with a nerve stimulator	135	0% within the first week	
Axillary brachial plexus block				
Stan et al. [56]	Transarterial	1,000	0.2%	Within the first month
Urban and Urquhart [6]	Transarterial	242	19% on the first day 7% at 2 weeks	Within the first month (except 1patient)
Selander et al. [15]	Paresthesia	290	2.8% within 3 weeks	Within 3 months (except 3 patients)
	Arterial pulsation	243	0.8% within the first week	Within 2 months
Fanelli et al. [7]	Nerve stimulation	1,650	1% withinthe first month	Within 3 months
Orebaugh et al. [17]	Nerve stimulation	96	0% (permanent injury)	
	Ultrasound with a nerve stimulator	220	0% (permanent injury)	

an axillary block and found that patients with awake or light sedation were less predisposed to a neurological injury than fully anesthetized patients (2.6% vs. 4.1%). Pediatric patients who had a block under general anesthesia had the highest rate of postoperative neurological complications (10.3%).

2. Central venous catheterization

The location of the subclavian vein between the clavicle and the first rib provides a convenient place for central venous catheterizations. Brachial plexus divisions lie superior to the subclavian artery and vein at the level of the supraclavicular triangle. The complications of subclavian vein catheterization include arterial puncture, pneumothorax, hemothorax, catheter malposition and so on. Of them, the reported incidences of nerve injury have been relatively rare (0–0.6%) [19,20]. But several case reports presented the possibility of BPI as a procedure complication [4,5,8].

Percutaneous catheterizations of the internal jugular vein have been shown to be relatively safe with a lower incidence of serious complications than the subclavian route [9]. However, another report showed similar overall rates of failure and complication between the subclavian and jugular approaches [21]. There are also several case reports on BPI after internal jugular vein catheterization [22,23].

3. Axillary angiography

The axillary artery route can be used for an arteriography if the femoral route is contraindicated or not available, such as in those patients with aortoiliac disease, aneurysm in the abdominal aorta or coarcted aorta. Axillary arteriography may also cause BPI. Chitwood et al. [10] reviewed 842 transaxillary arteriography cases and found 14 (1.7%) patients with nerve injuries.

MECHANISM OF INJURY

1. Direct needle trauma

A suspected mechanism during nerve block is a direct needle trauma. Patients typically have acute symptoms at the time of needle placement with painful electric shocks radiating distally down the limb in the distribution of the nerve [3,11]. It is believed that paresthesia is a marker for a potentially traumatic needle contact with a nerve. Therefore, the technique using the elicitation of a paresthesia may be associated with postanesthetic neurological sequelae [15].

A direct needle trauma can also occur as a result after repeated attempts of subclavian vein catheterization [4,5,8,24-29]. The subclavian vein is separated from the subclavian artery and brachial plexus by the anterior scalene muscle. However, a misdirection of the needle such as not parallel to the coronal plane, too far laterally or too deeply can cause damage to the nerve [5]. Anatomical variations in the brachial plexus may contribute to the nerve injury too [8].

2. Chemical neurotoxicity

The nature of the injection agent is another important factor. In an animal study, both extrafascicular injection and extraneural placement of a neurotoxic agent such as ropivacaine caused nerve damage with focal demyelination. Intrafascicular injection of ropivacaine resulted in more severe damage with histological abnormality including edema, axonal destruction and wallerian degeneration. In contrast, normal saline resulted only in an intraneural edema without demyelination or wallerian degeneration even if it was injected into the nerve fascicle [30]. Obviously, intrafascicular injections of neurotoxic agents cause more damage to the nerve than extrafascicular or extraneural injections [30,31].

Experimental studies have shown that all local anesthetics (efocaine, lidocaine, tetracaine, bupivacaine, mepivacaine, ropivacaine, etc) are potentially neurotoxic [32-35]. In particular, lidocaine and tetracaine seems to have a greater potential for neurotoxicity than other local anesthetics [33,34]. The neurotoxic effects of bupivacaine and ropivacaine may be more reversible compared with lidocaine and mepivacaine [34]. The degree of damage is associated with the concentration of local anesthetics and time of exposure of the nerve to the local anesthetics [34,35].

Supplemental epinephrine causes vasoconstriction and reduces peripheral nerve blood flow. It can result in nerve ischemia and potentiate local anesthetic-induced toxicity. Therefore, it appears reasonable to use lower doses of adjuvant epinephrine in peripheral nerve block [36]. The use of an unsterile or contaminated agent, and oil-based preparations may lead to a nerve injury, too [3].

3. Hematoma, pseudoaneurysm or medial brachial fascial compartment syndrome

Hematoma or pseudoaneurysm may be formed after BPB, axillary arteriography or subclavian/jugular catheterization. They could pressurize the nerve and induce ischemic changes [4,11,22,37]. The myelin sheath may become damaged even with trivial injuries, thereby leading to a conduction block. Wallerian degeneration may be occurred if the nerve remains compressed for 24 hours. However, axonal death may occur after a much shorter period of compression [37].

Tsao and Wilbourn [11] proposed the medial brachial fascial compartment syndrome as an injury mechanism following axillary regional block or angiography. The medial brachial fascial compartment is formed by tough brachial fascia extending from the axilla to the elbow. A needle injury of the axillary artery results in slow leakage of blood within this compartment. Increased pressure in the compartment can be sufficient to compromise microcirculation without increasing intra-arterial pressure. Thus, distal circulation and pulses may be normal in the presence of considerable but imperceptible nerve damage in most patients. This leads to a delayed presentation onset in patients after the axillary block or angiography.

PRESENTATION

A direct injection into the nerve may result in immediate severe pain radiating distally down the limb and neuromotor/neurosensory dysfunction in the distribution of the nerve [5,11]. The patient may continue to complain of pain, paresthesia or weakness on the affected side even after the removal of needle or catheter [8]. However, if the hematoma is formed by an injury, symptoms may be delayed and the patient may present only after the hematoma has expanded to a size large enough to compress the nerves. It can occur after hours or days [4,11].

After the nerve injury, the patient can complain of only mild numbness and tingling sensation. But pain is present in the majority of cases and may be described as a shooting or burning sensation. It can be exacerbated by physical factors such as pressure or touch. Severe allodynia or paresthesia can be developed with difficulty of motor activity [4,8,38].

If small fibers are involved, neurosensory loss to pin prick, hyperalgesia, hypesthesia, paresthesia and disturbed sympathetic innervation may be found. On the other hand, if large fibers are involved, motor power disturbance and loss of touch/vibration sense can be more common. In general, chemical toxicity leads to injury in small fibers, while excessive pressure leads to injury in large fibers [3].

Neurological symptoms have been reported according to the procedures in a varied distribution spectrum of the brachial plexus. Tsao and Wilbourn [11] noted that an nerve injury following an axillary regional block primarily involved the median nerve alone or in combination with the ulnar nerve and that no other nerve was injured in isolation. Another study reported that motor weakness after axillary angiography was most commonly involved in the median nerve distribution, followed by the ulnar nerve [37]. This could be explained by the medial brachial fascial compartment. Nerves at the level of the upper arm exit the medial brachial fascial compartment in the sequence (from proximal to distal) of musculocutaneous, axillary and radial. However, the median and ulnar nerves travel within the compartment to the elbow level. Therefore, the median and ulnar nerve are preferentially involved if there is a hematoma within this compartment [11]. On the other hand, the lower trunk, especially the ulnar nerve which arises from the lower trunk fibers, is the most vulnerable to be injured during infraclavicular subclavian vein catheterization [4,5]. But the upper trunk of brachial plexus could be injured during subclavian vein catheterization due to anatomical variations [8].

Connective tissue proliferation and scar formation may occur with time. The early sign of axonal regeneration with the reduplication of Schwann cells and axonal sprouting could be seen 1 to 2 weeks after nerve injury and the further regeneration with an improvement of initial symptoms is usually well advanced by 2 months after the injury. However, the regeneration is often inadequate and the initial loss could persist with severe sensory and/or motor disturbances. In such case, it may not be improved without a surgical intervention [39,40].

DIAGNOSIS

The past medical history, social habits, a detailed history associated to the nerve injury, and a review of systems should be evaluated in detail. Also it is important to conduct a physical examination about the sensory and motor disturbances including the level and severity of injury and changes of symptoms [39,41].

Electromyography can be used to diagnose BPI [8,11]. Electromyography may show complete or decreased functional loss if the nerve is stimulated proximal to the lesion, while normal response may be shown if the nerve is stimulated distal to the lesion. Classical signs of a denervation may not be detectable for up to 1-2 weeks after acute nerve transection. Therefore, electromyography should be performed at least 3-4 weeks after nerve damage [37]. Nerve conduction study can be helpful to diagnose nerve injuries, too. It is considered to be abnormal if the responses are less than 50% compared with the contralateral side or age-adjusted normal values [11].

Magnetic resonance imaging and ultrasonography are further methods to diagnose BPI. Magnetic resonance imaging can visualize an edematous nerve or a hematoma formation compressing the nerve [4,11,37]. On ultrasonography, normal brachial plexus appears as a hypoechoic mass containing a tubular structure on a transverse scan and a longitudinal hypoechoic structure containing small hyperechoic linings on a longitudinal scan. The injured nerve can be visualized as an enlarged and edematous nerve with loss of the hyperechoic lining or the discontinuity of the nerve [42].

The BPI must be differentiated from those of the original injury, operation or misuse of a tourniquet. Tourniquet injury usually affects the radial nerve with/without the median and ulnar nerves and is correlated with a good outcome in most cases [11].

TREATMENT

In general, the management for BPI is similar to that of other nerve lesions. The treatment of BPI should be individualized for each patient depending on the injury site, degree of damage, lag between injury and repair, age, occupation, and so on.

Conservative management is indicated if the lesions in continuity are non-degenerative or if the fascicles are intact. The treatment with medications consists of both opioid and non-opioid analgesics. Other drugs to control acute neuropathic pain include antidepressants (tricyclic antidepressants, serotonin and norepinephrine reuptake inhibitors), antiepileptic drugs (gabapentin, phenytoin, pregabalin), membrane stabilizers (intravenous lidocaine), ketamine and systemic glucocorticoid [43]. In addition, physiotherapy should be immediately initiated to prevent atonia [4,44,45]. Galvanic stimulation to the affected muscles and nerve ganglion blocks has been reported to provide symptom relief [3,46].

If a hematoma forms, its prompt evacuation may significantly reduce symptoms. Chitwood et al. [10] reported a better prognosis (eight-fold) in patients with medial brachial fascial compartment syndrome when a surgical evacuation was conducted within 4 hours of injury compared with after 4 hours from injury. Sensory changes after axillary artery puncture should be considered as an early indicator of developing BPI due to hematoma formation [37].

Lesions should be treated with surgical intervention if sensory or motor disturbances persist with a severe degeneration. An intraoperative examination of the nerve action potential is a useful method for the assessment of the neural function. The presence of nerve action potential beyond an injury indicates a preserved axonal function with good recovery. On the other hand, the absence of nerve action potential means an inadequate regeneration with poor recovery. Preganglionic injuries are usually treated with nerve transfers, while postganglionic lesions are treated with resection and repair, or nerve graft (an excision of the damaged segment and nerve autograft between two nerve ends) (Fig. 1) [39,41,47,48].

PREVENTION

Sufficient knowledge of anatomy, understanding of procedure, and adept skills in needle placement are essential for prevention of BPI. Only medical agents that have been proven reliability and safety should be used for nerve block. New agents should be used as cautiously as possible [3].

Nerve blocks should be performed without searching for paresthesia. The paresthesia method for the nerve block may increase the risk of postanesthetic neurological sequelae compared with no paresthesia methods [15], though paresthesia at the time of needle insertion does not always lead to postanesthesia nerve injury [49]. Currently, the paresthesia technique is not widely used for BPB. Instead of it, nerve stimulation has been the standard method for decades. In recent years, ultrasound guided

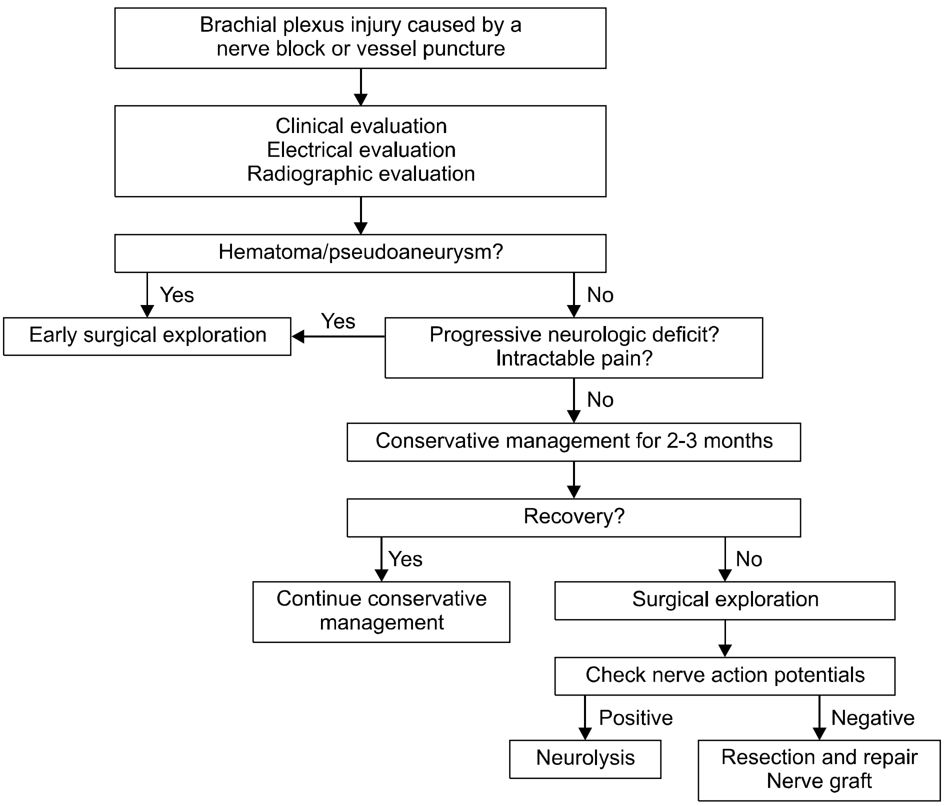

Fig. 1. Flowchart for the treatment of brachial plexus injury caused by a nerve block or vessel puncture.

nerve block has become popular with the improvements of ultrasound technology. Ultrasound allows a direct visualization of various peripheral nerves and localization of the local anesthetics. This may increase the success rate, decrease performance time and reduce the volume of local anesthetics [50,51]. However, further studies are needed to clarify the issue if ultrasound guidance could actually reduce the risk of nerve injury.

The type of needle seems to influence nerve penetration. Hirasawa et al. [52] studied the effect of three different types of needles (a short-bevelled, a long-bevelled and a tapered needle) on the degree of nerve injury in rabbits. A tapered needle did not cause any damage or tearing of the nerve fibers and resulted in the lowest level of damage to the perineurium. With both short- and long-bevelled needles, neural damage was reduced when the face of the bevel was inserted parallel to the nerve fibers.

Discovery of complications of BPB may be missed, since BPB is frequently performed as an out-patient procedure. Patients may confuse symptoms of BPI following BPB with symptoms caused by the original injury or operation. A careful observation and follow-up are imperative after the operation as well as during procedures [3,53].

While performing a subclavian catheterization, the interventionist should move the needle parallel to the coronal plane and avoid entering extreme depths [8]. A subclavian venipuncture should not be too laterally or deeply attempted [4]. If a fist attempt is failed, repeated attempts in the same region should be avoided. Multiple attempts could increase the chance of complications, because an anatomical anomaly may be present [8]. Puncturing the subclavian artery may lead to hematoma formation, which may compress the nerves and cause neurologic damage. If the patient has a prolonged bleeding time, coagulopathy should be corrected first before the performance of subclavian venipuncture or extrathoracic veins like the jugular vein should be used for catheterization [4]. The chances of a BPI are really greater when an inexperienced physician is performing a subclavian vein catheterization [4].

Whenever possible, the patient should not be heavily sedated and should be encouraged to immediately inform any experience of numbness/paresthesia during BPB or vessel puncture. If there is any notice, the needle should be withdrawn immediately [4,5,18].

CONCLUSIONS

A brachial plexus injury is a potential complication of a nerve block or vessel puncture. With an early recognition of nerve injuries, an appropriate management should be performed in order to reduce neurologic deficits and to maximize recovery. Comprehensive knowledge of anatomy and adept skills are crucial to avoid nerve injuries. Whenever possible, the patient should not be heavily sedated and should be encouraged to immediately inform a practitioner about any experiences of numbness/paresthesia during the nerve block or vessel puncture.

REFERENCES

1. Iwata T, Mitoro M, Kuzumoto N. Feasibility of early and repeated low-dose interscalene brachial plexus block for residual pain in acute cervical radiculopathy treated with NSAIDS. Korean J Pain 2014; 27: 125-32.
2. Johnson PS, Greifenstein FE. Brachial plexus block anesthesia. J Mich State Med Soc 1955; 54: 1329-31.
3. Woolley EJ, Vandam LD. Neurological sequelae of brachial plexus nerve block. Ann Surg 1959; 149: 53-60.
4. Karakaya D, Baris S, Güldogus F, Incesu L, Sarihasan B, Tür A. Brachial plexus injury during subclavian vein catheterization for hemodialysis. J Clin Anesth 2000; 12: 220-3.
5. Trentman TL, Rome JD, Messick JM Jr. Brachial plexus neuropathy following attempt at subclavian vein catheterization. Case report. Reg Anesth 1996; 21: 163-5.
6. Urban MK, Urquhart B. Evaluation of brachial plexus anesthesia for upper extremity surgery. Reg Anesth 1994; 19: 175-82.
7. 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.
8. Porzionato A, Montisci M, Manani G. Brachial plexus injury following subclavian vein catheterization: a case report. J Clin Anesth 2003; 15: 582-6.
9. Jernigan WR, Gardner WC, Mahr MM, Milburn JL. Use of the internal jugular vein for placement of central venous catheter. Surg Gynecol Obstet 1970; 130: 520-4.
10. Chitwood RW, Shepard AD, Shetty PC, Burke MW, Reddy DJ, Nypaver TJ, et al. Surgical complications of transaxillary arteriography: a case-control study. J Vasc Surg 1996; 23: 844-9.
11. Tsao BE, Wilbourn AJ. Infraclavicular brachial plexus injury following axillary regional block. Muscle Nerve 2004; 30: 44-8.

12. Madison SJ, Ilfeld BM. Peripheral nerve blocks. In: Morgan & Mikhail's clinical anesthesiology. 5th ed. Edited by Butterworth JF, Mackey DC, Wasnick JD. New York (NY), McGraw-Hill. 2013, pp 981-3.
13. Jenkins GW, Kemnitz CP, Tortora GJ. Anatomy and physiology from science to life. Hoboken (NJ), John Wiley & Sons, Inc. 2007.
14. Mian A, Chaudhry I, Huang R, Rizk E, Tubbs RS, Loukas M. Brachial plexus anesthesia: a review of the relevant anatomy, complications, and anatomical variations. Clin Anat 2014; 27: 210-21.
15. Selander D, Edshage S, Wolff T. Paresthesiae or no paresthesiae? Nerve lesions after axillary blocks. Acta Anaesthesiol Scand 1979; 23: 27-33.
16. Kapral S, Greher M, Huber G, Willschke H, Kettner S, Kdolsky R, et al. Ultrasonographic guidance improves the success rate of interscalene brachial plexus blockade. Reg Anesth Pain Med 2008; 33: 253-8.
17. Orebaugh SL, Williams BA, Vallejo M, Kentor ML. Adverse outcomes associated with stimulator-based peripheral nerve blocks with versus without ultrasound visualization. Reg Anesth Pain Med 2009; 34: 251-5.
18. Ben-David B, Barak M, Katz Y, Stahl S. A retrospective study of the incidence of neurological injury after axillary brachial plexus block. Pain Pract 2006; 6: 119-23.
19. Bernard RW, Stahl WM. Subclavian vein catheterizations: a prospective study. I. Non-infectious complications. Ann Surg 1971; 173: 184-90.
20. Ryan JA Jr, Abel RM, Abbott WM, Hopkins CC, Chesney TM, Colley R, et al. Catheter complications in total parenteral nutrition. A prospective study of 200 consecutive patients. N Engl J Med 1974; 290: 757-61.
21. Sznajder JI, Zveibil FR, Bitterman H, Weiner P, Bursztein S. Central vein catheterization. Failure and complication rates by three percutaneous approaches. Arch Intern Med 1986; 146: 259-61.
22. Fuller GN, Dick JP, Colquhoun IR. Brachial plexus compression by hematoma following jugular puncture. Neurology 1994; 44: 775-6.
23. Paschall RM, Mandel S. Brachial plexus injury from percutaneous cannulation of the internal jugular vein. Ann Emerg Med 1983; 12: 58-60.
24. Jackson L, Keats AS. Mechanism of brachial plexus palsy following anesthesia. Anesthesiology 1965; 26: 190-4.
25. García-Fages LC, Castillo J, Gomar C, Villalonga A, Nalda MA. Transient block of the brachial plexus after catheterization of the subclavian vein. Ann Fr Anesth Reanim 1990; 9: 93-4.
26. Goven'ko FS, Rogulov VA. Injury of the brachial plexus after catheterization of the subclavian vein in a 6-month-old child. Vestn Khir Im I I Grek 1983; 130: 121-2.
27. Defalque RJ. Percutaneous catheterization of the internal jugular vein. Anesth Analg 1974; 53: 116-21.
28. Daily PO, Griepp RB, Shumway NE. Percutaneous internal jugular vein cannulation. Arch Surg 1970; 101: 534-6.
29. Matz R. Complications of determining the central venous pressure. N Engl J Med 1965; 273: 703.
30. Whitlock EL, Brenner MJ, Fox IK, Moradzadeh A, Hunter DA, Mackinnon SE. Ropivacaine-induced peripheral nerve injection injury in the rodent model. Anesth Analg 2010; 111: 214-20.
31. Gentili F, Hudson AR, Hunter D. Clinical and experimental aspects of injection injuries of peripheral nerves. Can J Neurol Sci 1980; 7: 143-51.
32. Nowill WK, Hall H, Stephen CR. Neurological complications following the use of efocaine. AMA Arch Surg 1953; 67: 738-40.
33. Yamashita A, Matsumoto M, Matsumoto S, Itoh M, Kawai K, Sakabe T. A comparison of the neurotoxic effects on the spinal cord of tetracaine, lidocaine, bupivacaine, and ropivacaine administered intrathecally in rabbits. Anesth Analg 2003; 97: 512-9.
34. Radwan IA, Saito S, Goto F. The neurotoxicity of local anesthetics on growing neurons: a comparative study of lidocaine, bupivacaine, mepivacaine, and ropivacaine. Anesth Analg 2002; 94: 319-24.
35. Selander D. Neurotoxicity of local anesthetics: animal data. Reg Anesth 1993; 18: 461-8.
36. 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.
37. O'Keefe DM. Brachial plexus injury following axillary arteriography. Case report and review of the literature. J Neurosurg 1980; 53: 853-7.
38. Reiss W, Kurapati S, Shariat A, Hadzic A. Nerve injury complicating ultrasound/electrostimulation-guided supraclavicular brachial plexus block. Reg Anesth Pain Med 2010; 35: 400-1.
39. Spinner RJ, Kline DG. Surgery for peripheral nerve and brachial plexus injuries or other nerve lesions. Muscle Nerve 2000; 23: 680-95.
40. Gentili F, Hudson AR, Kline D, Hunter D. Early changes following injection injury of peripheral nerves. Can J Surg 1980; 23: 177-82.
41. Yoshikawa T, Hayashi N, Yamamoto S, Tajiri Y, Yoshioka N, Masumoto T, et al. Brachial plexus injury: clinical manifestations, conventional imaging findings, and the latest imaging techniques. Radiographics 2006; 26 Suppl 1: S133-43.
42. Shafighi M, Gurunluoglu R, Ninkovic M, Mallouhi A, Bodner G. Ultrasonography for depiction of brachial plexus injury. J Ultrasound Med 2003; 22: 631-4.
43. Gray P. Acute neuropathic pain: diagnosis and treatment. Curr Opin Anaesthesiol 2008; 21: 590-5.
44. Dhir S, Tureanu L, Stewart SA. Axillary brachial plexus block

complicated by cervical disc protrusion and radial nerve injury. Acta Anaesthesiol Scand 2009; 53: 411.
45. Kim DH, Murovic JA, Kline DG. Brachial plexus injury: mechanisms, surgical treatment and outcomes. J Korean Neurosurg Soc 2004; 36: 177-85.
46. Po BT, Hansen HR. Iatrogenic brachial plexus injury: a survey of the literature and of pertinent cases. Anesth Analg 1969; 48: 915-22.
47. Hems T. Nerve transfers for traumatic brachial plexus injury: advantages and problems. J Hand Microsurg 2011; 3: 6-10.
48. Wilkinson MC, Birch R, Bonney G. Brachial plexus injury: when to amputate? Injury 1993; 24: 603-5.
49. Pearce H, Lindsay D, Leslie K. Axillary brachial plexus block in two hundred consecutive patients. Anaesth Intensive Care 1996; 24: 453-8.
50. Gelfand HJ, Ouanes JP, Lesley MR, Ko PS, Murphy JD, Sumida SM, et al. Analgesic efficacy of ultrasound-guided regional anesthesia: a meta-analysis. J Clin Anesth 2011; 23: 90-6.
51. Klaastad O, Sauter AR, Dodgson MS. Brachial plexus block with or without ultrasound guidance. Curr Opin Anaesthesiol 2009; 22: 655-60.
52. Hirasawa Y, Katsumi Y, Küsswetter W, Sprotte G. Experimental studies on peripheral nerve injuries caused by injection needles. Reg Anaesth 1990; 13: 11-5.
53. Dooley J, Fingerman M, Melton S, Klein SM. Contralateral local anesthetic spread from an outpatient interscalene catheter. Can J Anaesth 2010; 57: 936-9.
54. Moberg E, Dhuner KG. Brachial plexus block analgesia with xylocaine. J Bone Joint Surg Am 1951; 33-A: 884-8.
55. Liu SS, YaDeau JT, Shaw PM, Wilfred S, Shetty T, Gordon M. Incidence of unintentional intraneural injection and postoperative neurological complications with ultrasound-guided interscalene and supraclavicular nerve blocks. Anaesthesia 2011; 66: 168-74.
56. Stan TC, Krantz MA, Solomon DL, Poulos JG, Chaouki K. The incidence of neurovascular complications following axillary brachial plexus block using a transarterial approach. A prospective study of 1,000 consecutive patients. Reg Anesth 1995; 20: 486-92.