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Severe Brachial Plexopathy after an Ultrasound-guided Single-injection Nerve Block for Total Shoulder Arthroplasty in a Patient with Multiple Sclerosis

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DESPITE the known benefits of regional anesthesia for patients undergoing joint arthroplasty, the performance of peripheral nerve blocks in patients with multiple sclerosis (MS) remains controversial. MS has traditionally been described as an isolated disease of the central nervous system, without involvement of the peripheral nerves, and peripheral nerve blockade has been suggested to be safe.[1,2] However, careful review of the literature suggests that MS may also be associated with involvement of the peripheral nervous system, challenging traditional teachings. There is a paucity of evidence with regard to safety in using peripheral nerve regional anesthesia in these patients. This makes it difficult to provide adequate "informed consent" to these patients. This case report describes a patient with MS who sustained a severe brachial plexopathy after a total shoulder arthroplasty during combined general anesthesia and interscalene nerve block.

Case Report

A 65-yr-old right-hand-dominant man, American Society of Anesthesiologists physical status III, presented for a right total shoulder arthroplasty secondary to osteoarthritis. The patient's medical history was significant for hypothyroidism, benign prostatic hypertrophy, mitral valve prolapse, and MS. His medications included 40 mg pravastatin by mouth daily, 75 μg levothyroxine by mouth daily, and 15 mg oxybutynin (extended release) by mouth daily. The patient was allergic only to oysters, which had caused anaphylaxis in the past. Although without clinical changes for 2 yr, his MS was remarkable for bilateral lower extremity weakness (walker needed for ambulation) and the requirement for self– urethral catheterization.

After informed consent, the patient underwent an interscalene nerve block and general anesthetic. In the preanesthetic block room, sedation was provided with 50 μg intravenous fentanyl and 2 mg intravenous midazolam. An ultrasound-guided "single-shot" injection using an in-plane needle approach and nerve stimulation was performed. The injection was made at the mid-neck level at the nerve roots of the brachial plexus. The needle direction was in reference to the middle scalene muscle from the lateral toward medial direction. Three injections were made starting laterally on C5, then anteriorly on C5, and then medially to C5. The injections were made to create circumferential spread around the roots of the brachial plexus. The injection was performed as previously described.[3,4] A 50-mm, 22-gauge b-bevel (B. Braun Medical, Bethlehem, PA) was inserted in plane with the ultrasound beam during visualization of the roots of the brachial plexus on short axis. The needle (stimulating at 0.45 mA, 0.1-ms pulse duration, 2 Hz) was directed until it approached the outer edge of the C5 nerve root. The needle was not seen to penetrate the epineurium by our ultrasound image (fig. 1). After the demonstration of biceps contraction, an injection of 30 ml bupivacaine (0.5%), 1:400,000 epinephrine, and 50 μg clonidine was injected using a 10-ml Luer-Lok controlled stroke syringe. The local anesthetic was noted to surround the C5–C6 nerve roots. The needle was repositioned three times to generate complete coverage of the C5–C6–C7 roots. During the procedure, the patient experienced no discomfort, and there was not resistance to injection. The block was checked for success by the senior regional resident. This patient was noted to have partial sensory (to ice) blockade over the anterior shoulder (axillary nerve distribution C5) and partial motor (by strength testing) and sensory (to ice) blockade of the musculocutaneous nerve distribution 10 min after regional blockade.

After the induction and maintenance of general endotracheal anesthesia, the patient was placed in the beach chair position. Consistent with the sitting position, an episode of hypotension (77/46 mmHg, mean arterial pressure 56 mmHg) was noted after induction and patient repositioning to the sitting position. The patient initially required a total administration of 2 l incrementally of lactated Ringer's solution and a total of 15 mg ephedrine (in 5-mg dosing increments) to return to a mean arterial pressure greater than 70 mmHg. Intraoperatively, the patient's temperature ranged from approximately 35 to 36.4 centigrade. The arm was held in place by the Spider Arm Retractor (Tenet Medical Engineering, Calgary, Alberta, Canada). A Zimmer anatomic total shoulder system was used (Zimmer Inc., Warsaw, IN). During placement of the glenoid component, the arm was positioned in 35° of external rotation and 45° of abduction. The estimated blood loss was 400 ml, and the patient received 2,800 ml lactated Ringer's solution. Surgical time was 3 h 45 min. After emergence in the postanesthesia recovery unit, the patient was noted by nursing staff to have a dense motor and sensory block and was also noted to be comfortable for the first hour. The patient then began to report right arm pain that was described as burning in quality. It was rated as 5 out of 10 on a visual analog scale. A neurologic examination was performed by the operating orthopedic resident within 4 h postoperatively. At that time, the

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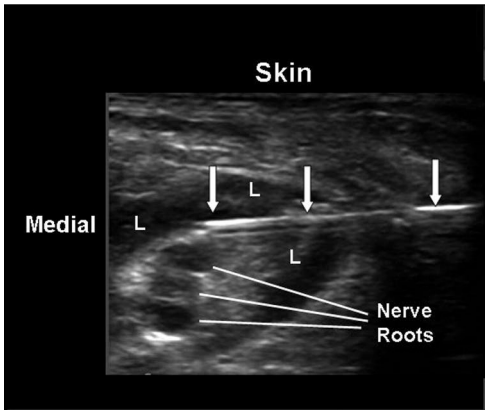

Fig. 1. Interscalene nerve block in our patient with multiple sclerosis. Image shows the C5–C7 ventral roots of the right brachial plexus. *Arrows* indicate the needle in plane with the ultrasound beam. The needle tip was completely visualized throughout the procedure. *L* indicates local anesthetic completely surrounding nerve roots, *i.e.*, "donut sign."

patient was again noted to have a dense motor and sensory block of the operative extremity, as would be expected 10 h after a successful regional blockade.

On postoperative day 1, the patient continued to have shoulder pain with a persistent flaccid motor block of his entire right upper extremity. This pain was exacerbated by shoulder and arm movement and not by neck movement or a Valsalva maneuver, as can be seen in cervical radiculopathy. A consultation by the neurology service on postoperative day 2 found sensation to temperature throughout dermatomes C4–T1, with absent light touch sensation in C6–T1. Vibration and joint position perception were absent throughout. A magnetic resonance image of the chest was performed on postoperative day 3, which demonstrated postsurgical changes without any evidence for compressive or avulsive pathology. However, it was diagnostic for brachial neuritis (fig. 2). High-dose methylprednisolone was initiated to treat a presumed autoimmune brachial neuritis. An electromyelogram performed on postoperative day 4 showed loss of the median and ulnar F waves. In addition, there was no voluntary recruitment of the following muscles: deltoid, triceps, biceps, brachioradialis, wrist extensors, and first dorsal interosseous. At this time, there was no evidence of active denervation in any of the muscles examined. On postoperative day 11, a complete paresis of the patient's entire arm persisted; an electromyelogram demonstrated active denervation of all muscles and no voluntary motor recruitment. This study demonstrated low-amplitude compound muscle action potentials of the median and ulnar motor nerves. Median ulnar and radial sensory nerve action potentials were absent. Electromyographic examination revealed active denervation in all of the muscles previously examined, with no voluntary motor recruitment (table 1).

A follow-up electromyelogram 3 months from the date of surgery showed improvement. There was reduced voluntary motor recruitment with evidence of reinnervation in all of the muscles that were previously examined. The patient's unaffected limbs were tested, and studies of the radial and sural sensory nerves and ulnar and peroneal motor nerves with F waves yielded normal results. Nerve fiber loss can still be significant despite normal nerve conduction study results. Therefore, a normal electromyelogram does not completely rule out subclinical peripheral neuropathy.

At 8 months postoperatively, the patient continued to have significant range of motion and strength deficits. His distal hand function remained limited secondary to stiffness from the prolonged neurologic recovery. Range of motion at the wrist, metacarpophalangeal joints, proximal interphalangeal joints, and distal interphalangeal joints were significantly limited, with approximately 50% loss of motion at each level. The patient also continued to have visible isolated muscle atrophy of proximal musculature, including the pectoralis major and pos-

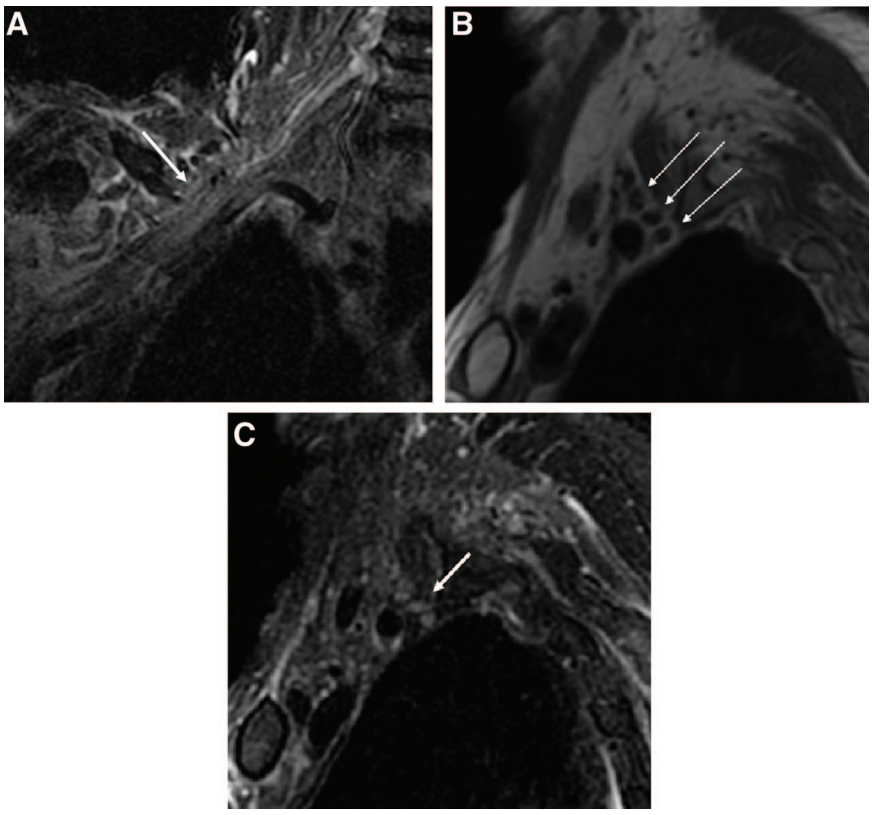

Fig. 2. (*A*) T2 coronal image demonstrating increased signal intensity of the right brachial plexus (*arrow*). (*B*) Sagittal T1 image demonstrating swelling of the brachial plexus (*arrows*) posterior and superior to the subclavian artery flow void. (*C*) Sagittal T2 image demonstrating increased girth and increased signal of the brachial plexus (*arrow*) posterior and superior to the subclavian artery flow void.

Table 1. Needle Electromyography of Patient on Postoperative Day 11

	Spontaneous Activity			Volitional Activity		
Muscle	Fibrillations	+Waves	Polyphasic	Amplitude	Duration	Recruitment
Deltoid, right	2+	2+	Increased	Increased	Increased	Single
Biceps, right	1+	1+	No	Voluntary	Motor	Units
Triceps, right	1+	1+	No	Voluntary	Motor	Units
Brachioradialis, right	1+	1+	No	Voluntary	Motor	Units
FDI, right	0	0	No	Voluntary	Motor	Units
FPL, right	1+	1+	No	Voluntary	Motor	Units
EDC, right	0	0	No	Voluntary	Motor	Units
Trapezius, right	0	0	0	Normal	Normal	Full

EDC = extensor digitorum complex; FDI = first dorsal interossei; FPL = flexor pollicis longus.

terior deltoid. His final diagnosis was an inflammatory brachial neuritis (IBN).

Discussion

Multiple sclerosis is described as a chronic disease of the central nervous system that usually begins in young adults. Pathologically, MS is characterized by multiple areas of central nervous system white matter inflammation, demyelination, and glial scarring or sclerosis.[5] The clinical course of MS varies from a benign, largely symptom-free disease to a rapidly progressive and disabling disorder. The etiology of MS is likely due to autoimmune mechanisms, possibly triggered by infectious and other environmental factors in genetically susceptible individuals.[6]

Controversy exists in providing regional anesthesia to patients with neurologic diseases. The "double-crush" phenomenon suggests that patients with preexisting neural compromise may be more susceptible to injury at another site when exposed to a secondary injury.[7] The performance of a neuraxial technique in patients with preexisting central nervous system disorders may increase the risk of a double-crush phenomenon.[8] In contrast to a spinal or epidural block, a peripheral nerve block in MS patients is theoretically attractive because the neural pathology is presumed to be located in the central nervous system. However, this association seems to be incomplete and is based on the fact that the clinical involvement of the peripheral nervous system in MS patients has traditionally been ignored by modern textbooks. This is despite the fact that the description of this link dates back a half century.[9] Importantly, this conventional teaching is also present in the anesthesia literature.[1,2,10–13] Careful assessment of the literature reveals that multiple recent studies have shown the existence of subclinical peripheral neuropathy in some patients with MS.[14–18] Pogorzelski *et al.*[14] noted both sensory and peripheral motor nerve lesions of a demyelinating-axonal character. They also noted that sensory abnormalities were more pronounced than motor ones. Another study found electrophysiologic abnormalities in the 14.7% of all peripheral nerves examined (n = 244) in patients with MS.[17] This is well above the reported prevalence of 2.4% in the general population. In the elderly, the prevalence is reported to be as high as 8%, mostly due to diabetes mellitus.[19] Hughes *et al.*[20] described an association of a demyelinating peripheral neuropathy in MS patients. Other inflammatory demyelinating diseases exist that have both central and peripheral components, such as chronic inflammatory demyelinating polyneuropathy.[21]

Patients with underlying peripheral neurologic disorders may be more susceptible to nerve injury with the use of regional techniques.[22] Despite testing modalities such as electromyography and magnetic resonance imaging, it may be difficult to differentiate between multiple etiologies, including direct trauma during the regional procedure, neurotoxicity from local anesthetics (and additives), and patient positioning, such as extreme abduction and external rotation, which has been implicated in surgical stretch injury of the brachial plexus. All of these could occur in a patient undergoing total shoulder replacement. The other confounding variable in diagnosing the etiology of a postoperative neurologic deterioration is that the clinical course of MS may be exacerbated from many nontraumatic-related reasons, such as hyperthermia, electrolyte abnormalities, stress, and pain.

Brachial plexus injury after total shoulder arthroplasty has been estimated at 2.8%.[23] To our knowledge, this is the first report of an IBN after total shoulder replacement in a patient with MS. This is also the first report of IBN in a patient using an ultrasound-guided regional anesthesia technique. Brachial plexus injury after interscalene nerve blockade has been previously described.[24] IBN has also been reported to occur in patients during treatment for MS.[18] IBN is a well-recognized clinical syndrome characterized by brachial pain followed by patchy atrophy of muscles in the shoulder girdle and arm innervated by individual branches of the brachial plexus.[25–27] Postsurgical IBN has not been widely recognized since Parsonage and Turner's original description.[27]

In summary, we report a case of a severe brachial plexus injury that occurred in a patient with MS after a

total shoulder replacement during combined general anesthesia and interscalene nerve block. Although the mechanisms of this injury are unclear, the potential preexisting pathology of the peripheral nervous system may have contributed. It is possible that this patient preoperatively had an occult peripheral neuropathy, and his underlying MS predisposed him to development of a peripheral autoimmune injury leading to a brachial neuritis. The individual decision to perform peripheral regional anesthesia in a patient with MS must rest on the perceived benefits of avoiding non–opioid-based analgesia and/or avoiding general anesthesia. Anesthesiologists should recognize that the peripheral nervous system may also be abnormal in patients with MS.

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Ethanol-induced Coma after Therapeutic Ethanol Injection of a Hepatic Cyst

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HEPATIC cyst is a common congenital malformation, the incidence of which varies from 0.1% to 4.5%.[1,2] Hepatic cysts are most often asymptomatic. Clinical symptoms comprise abdominal compression revealed by abdominal pain, gastric satiety, vomiting, biliary compression with jaundice, dilatation of biliary ducts or cholestasis, and vascular compression of the inferior vena cava or hepatic vessels.[3] Cyst sclerotherapy may be required in such cases, as well as in intracystic hemorrhage. Sclerotherapy is usually performed by percutaneous ethanol injection *in situ* into the cyst. Such therapy is currently recommended for the treatment of symptomatic hepatic cyst, because of its efficiency and the absence of reported severe complications.[4] Here, we report the original case of a patient who demonstrated ethanol-induced coma requiring mechanical ventilation after ethanol injection of a symptomatic hepatic cyst.

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Case Report

A 69-yr-old woman (168 cm, 65 kg) was admitted to the recovery room after ethanol injection of a hepatic cyst performed during general anesthesia. Her medical history included arterial hypertension. Medication was bisoprolol. She reported no alcohol consumption. Intracystic hemorrhage of a 22 × 20 × 15-cm hepatic cyst located to the right lobe occurred 3 weeks before admission and led to a decision to treat the cyst by *in situ* ethanol injection. General anesthesia was provided by continuous infusion of intravenous propofol while the patient was spontaneously breathing an oxygen–air mixture (6 l/min; fraction of inspired oxygen [F_{IO_2}], 0.5) delivered *via* a facemask tightly connected to the face. The patient was monitored with an electrocardioscope, a noninvasive blood pressure device, a pulse oximeter, and an end-tidal carbon dioxide measurement device. The procedure was performed by an experienced radiologist, under sonographic guidance. Cystic puncture was performed with a pigtail catheter, and 3,500 ml fluid was evacuated. Postevacuation opacification ruled out communication between the cyst and the biliary tree, and 240 ml ethanol, 95%, was injected into the cyst cavity. The patient was then positioned alternately on left and right lateral decubitus to allow ethanol to reach the maximum area of the cyst cavity. Fifty minutes later, the same quantity of liquid was removed from the cyst, and the procedure ended uneventfully. The total dose of propofol delivered to the patient was 210 mg. No additional anesthetic or opioid was administered during the procedure. The patient was able to properly respond to verbal command and was discharged to the postanesthesia care unit. Shortly after arrival in postanesthesia care unit, the patient developed lethargy and became unresponsive. Her breath smelled of alcohol. Consciousness rapidly deteriorated and was followed by a coma scored as 3 on the Glasgow Coma Scale. The trachea was intubated, and mechanical ventilation was initiated (F_{IO_2}, 0.4; tidal volume, 650 ml; respiratory rate, 12 breaths/min). An ethanol-induced coma was suspected and confirmed by measurement of the patient's blood ethanol level, which was 3.10 g/l. The patient progressively recovered satisfactory consciousness and was extubated 11 h after the procedure. Her ethanol blood levels were 1.88 g/l at hour 7 and 0.27 g/l at hour 15 after the procedure. The patient was discharged uneventfully from the institution 2 days later.

Discussion

We report here a massive ethanol intoxication leading to coma after ethanol sclerosis of a hepatic cyst. To our knowledge, this is the first description of severe ethanol-induced coma after ethanol injection of a hepatic cyst.

Mild alcoholemia-related clinical signs after hepatic cyst alcoholization have been scarcely published, and no alcoholemia-related morbidity has been described. Maximal ethanol blood levels up to 1.02 g/l have been reported 1 h after the procedure.[5,6] Hepatic cysts are avascular tumors. Systemic absorption of ethanol may therefore have occurred *via* two pathways. At first, ethanol could have entered biliary ducts and then the gut *via* transmural absorption by mesenteric blood vessels. However, the demonstration of absence of communication between the hepatic cyst and biliary ducts after opacification likely rules out such a scenario in our case. Similarly, the delayed onset of symptoms, with respect to the time of ethanol administration, is hardly consistent with an accidental vascular injection. More likely, ethanol was directly absorbed through the cyst wall formed by an epithelium which resembles biliary epithelium and a stroma, made of a thin layer of connective tissue.[7] The giant size of the cyst, the large volume of ethanol used,[8] and the long time in contact surely contributed to this unusually high absorption rate. The alcoholic smell of the patient's breath was rapidly detected postoperatively, supporting ethanol as the cause of the coma. The diagnosis was further confirmed by measurement of the ethanol blood level. The rapid decrease in ethanol blood level after the procedure was consistent with the fact that excessive ethanol absorption had occurred both intraoperatively and in the early postoperative period.

Conclusion

Ethanol-induced coma must be considered in the absence of recovery, or deterioration of consciousness after apparently normal awakening, after ethanol injection of a hepatic cyst performed during general anesthesia. Anesthesiologists as well as radiologists should be aware of this rare but potentially life-threatening complication. A limited volume of injected ethanol is warranted. Ethanol levels should be assessed in the early postoperative stage.

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To Be or Not to Be

To the Editor:—We read with interest the case report published by Koff *et al.*[1] and the editorial by Hebl.[2] How can Dr. Hebl discuss the role that the use of an ultrasound may have played in this case? Ultrasound allows us to visualize the nerves and the spread of local anesthetic. From the authors' description, it is clear that except for the use of 0.5% bupivacaine, the technique used to perform the interscalene block could not have led to such a catastrophic outcome. The injection of local anesthetic was not intraneural, because the authors reported that "the local anesthetic was noted to surround C5–C6" and that intraneural injections have been demonstrated to produce swelling of the nerve.[3] In addition, how would a 22-gauge blunt needle, even in the hands of a resident under the supervision of an attending, be able to damage the three trunks? What was really surprising about the case report and the editorial is that none of the authors questioned the use of 30 ml bupivacaine, 0.5%. Bupivacaine neurotoxicity is well established.[4] Because general anesthesia was the main anesthetic technique, why did the author choose to perform an anesthetic (0.5% bupivacaine) and not an analgesic block (0.25% bupivacaine)? More importantly, why was bupivacaine chosen rather than a less toxic drug such as ropivacaine?[5] In the presence of a theoretical increase in the possibility of nerve injury, would it be logical to choose the local anesthetic and the concentration with the least potential for neurotoxicity? There is no doubt that considerations should be given to the role played by multiple sclerosis (MS) in the postsurgical complication. Before arguments can be presented to contraindicate the use of peripheral nerve block in the patient with MS, could we at least also consider the possibility that MS might increase the surgical risk of a nerve injury, especially when considering that shoulder surgery is associated with a risk of permanent nerve injury much more frequently than peripheral nerve block?[6,7] In conclusion, from the data presented, it is impossible to determine whether the complication presented was directly related to the surgery or was the result of an MS-related increase in the surgical risk or an MS-related increase in the local anesthetic toxicity. What is certain is that the use of ultrasound had nothing to do with the outcome.

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Severe Brachial Plexopathy after an Ultrasound-guided Single-injection Nerve Block for Total Shoulder Arthroplasty in a Patient with Multiple Sclerosis: What Is the Likely Cause of This Complication?

To the Editor:—The occurrence of severe brachial plexopathy after an ultrasound-guided single-injection nerve block for total shoulder arthroplasty in a patient with multiple sclerosis (MS) presented by Koff *et al.*[1] raised several issues regarding the cause of this complication. Intraneural injection, the most feared complication when performing regional block, can in this case be definitely excluded. The possibility of having transfixed the upper or median cord during the procedure seems, although possible, unlikely. Moreover, it has been shown that even injection of local anesthetics beyond the epineurium does not invariably result in nerve damage.[2] The existence of a preexisting subclinical polyneuropathy has been shown to increase the toxic potential of local anesthetics in certain circumstances.[3] In the current case, MS has been highlighted as a risk factor. MS is a chronic disease characterized by multiple areas of central nervous system white matter inflammation, demyelination, and glial scaring or sclerosis.[4] Despite reports of peripheral nerve alterations, peripheral nervous system involvement remains rare and, if present, subclinical in most cases, due to subtle nerve lesions without any frank demyelination. This is supported by the work by Boerio *et al.*[5]: In MS patients with no nerve conduction abnormalities, assessment of the absolute and relative refractory periods showed significant increase in refractoriness compared with a control group. However, these minor changes could not be considered as significant alteration of the nerve myelin sheath. A recent study described the occurrence of a new inflammatory demyelinating disease unlike MS or chronic inflammatory demyelinating polyradiculopathy occurring in MS patients with a relapsing–remitting course in which the central nervous system involvement preceded peripheral nerve system involvement.[6] The current case does not fulfill the criteria for this diagnosis. The authors have suspected an acute "inflammatory" neuritis, but unfortunately this was not further investigated by either sural nerve biopsy or cerebrospinal fluid analysis for elevation of protein content reflecting nerve root inflammation.[7] The presence of a preexisting polyneuropathy could have been disclosed if conduction studies had been performed on postoperative day 3. The recordings would have shown signs of demyelination because pathologic features found on peripheral nerves in patients with MS are either segmental demyelination or reduction in myelin thickness.[8] This was not the case in this patient, and unfortunately electroneuromyography studies of the contralateral arm have not been performed. The latter recording would have given an objective state of the peripheral nerve system. These elements make the likelihood of a previous polyneuropathy very unlikely. This assumption is also supported by normal elec-

tromyography performed on the patient's unaffected limbs 3 months later. How, then, can this event be explained? First, the occurrence of burning pain—neuropathic character—despite a dense motor block 5–6 h after a successful block performed with 30 ml ropivacaine, 0.5%, is unusual because the duration of the sensory block is approximately 12–15 h. This suggests an "acute trauma" of the brachial plexus. Second, the long duration of surgery (3 h 45 min) let us think that the procedure was complicated, meaning that the placement of the prosthesis had probably required a large amount of traction—physically induced stress—on the brachial plexus. Studies have shown that abduction challenges the brachial plexus.[9] Arm extension, wrist extension, and head rotation to the contralateral side add further stress on the nerves.[9,10] Ikeda *et al.*[11] have demonstrated in experimental studies that an elongated nerve is much more vulnerable to compression injury (surgical retractors). This constellation favors an acute "physically induced trauma" of the brachial plexus to explain the development of this complication. This is supported by the electromyography recordings on day 11, consistent with axonal loss. On the other hand, the toxic effect of local anesthetic placed outside the epineurium, as shown by ultrasound in the current case, would have more likely shown signs of demyelination. Last, testing the anterior part of the shoulder with cold ice gives information regarding blockade of the medial branch of the supraclavicular nerve, not the axillary nerve. Positioning and surgically induced stress are certainly greatly underestimated by anesthesiologists as causes of brachial plexus damage after shoulder surgery.

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Nerve Blocks, Ultrasounds, and Multiple Sclerosis

To the Editor:—I read with great interest the case report by Koff *et al.*[1] The authors rightly highlighted two important points of general interest. First, patients with multiple sclerosis may have a compromise of the peripheral nerves. Second, anesthesiologists must be aware that patients with a preexisting neurologic deficit (even if subclinical) may be more susceptible to perioperative injuries (double-crush phenomenon).

However, I would like to express some consideration about this case. The authors stated that "despite testing modalities, it may be difficult to differentiate between multiple etiologies of brachial plexus injuries." I perfectly agree with this statement but, sometimes, useful clues about the etiologies of brachial plexus damage may be achieved by the research of the site of the initial injury. I would like to examine two possible local causes of "second crush": the peripheral nerve block and the surgical procedure.

An injury caused by the needle or by a toxic effect of the local anesthetic mixture injected at the interscalene level should probably affect, at least at the beginning, the highest part of the plexus, with a sparing of the lowest roots (C8–T1), usually not reached by the needle or by the local anesthetic. *Vice versa*, a local surgical factor (*e.g.*, a compression by a retractor protracted for several hours)[2] may cause an injury at the cord level (deltopectoral approach), with a possible block of the arm from the shoulder to the fingers (including the median and the ulnar nerves) and a sparing of the nerves emerging from the roots or the trunks, like the long thoracic, the dorsal scapular, and the suprascapular nerves.

Unfortunately, the authors did not provide us with data on the function of the long thoracic, the dorsal scapular, and the suprascapular nerves. Therefore, we can only analyze the clinical and instrumental data available.

On postoperative day 1, these are the data recorded: loss of light touch sensation in C6–T1, shoulder pain exacerbated by arm movements (a normal postoperative pain?), and flaccid motor block of the entire extremity (obviously including the hand). The magnetic resonance imaging performed on postoperative day 3 demonstrated swelling and increased signal of the brachial plexus at the thoracic level (no data on the cervical part of the plexus). The electromyelogram performed on postoperative day 4 showed loss of the median and ulnar F waves. On postoperative day 11, the same procedure demonstrated active denervation of all the muscles examined and absence of median, ulnar, and radial sensory nerve action potentials. All of these clinical and instrumental data seem to indicate, in my opinion, a distal (cord) site of secondary injury.

The only fact that could indicate a proximal site of injury is the recording of visible atrophy of the proximal musculature at 8 months postoperatively. However, I do not know whether this finding might be attributable to a specific nerve lesion or to the prolonged inactivity of the whole arm.

On the basis of these data (albeit incomplete), I think that, in this patient, the most probable responsible of the "second crush" should be searched at the surgical field and that the anesthesiologic factors did not play a main role in the development of the postoperative neurologic deficit. Therefore, in my opinion, other evidences are necessary before establishing a correlation between peripheral nerve blocks and nerve damage in multiple sclerosis patients.

Moreover, this case report does not give us any further information about the usefulness of ultrasound-guided techniques in the prevention of neurologic injuries.[3]

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