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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