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. 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 com-

pared 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-