Ultrasound-Guided Popliteal Block Demonstrates an Atypical Motor Response to Nerve Stimulation in 2 Patients With Diabetes Mellitus

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Background and Objectives: Nerve stimulation is a useful technique to identify peripheral nerves before blockade. We report 2 cases of the failure of nerve stimulation to accurately localize the sciatic nerve in patients with diabetes mellitus undergoing outpatient foot procedures. We also introduce a novel approach to performing a popliteal fossa block using ultrasound guidance.

Case Report: Ultrasound-guided popliteal fossa blocks were performed in 2 patients with diabetes mellitus. Both patients failed to develop an appropriate motor response or paresthesia to nerve stimulation. The needle positions were confirmed by ultrasound guidance and injections of local anesthesia were made uneventfully. Appropriate surgical anesthesia was established and the procedures were performed uneventfully.

Conclusion: Ultrasound facilitated the accurate localization of the sciatic nerve in 2 patients with diabetes mellitus. Neither patient had a paresthesia or muscle twitch below 2.4 mA. There is theoretical concern that patients with underlying neuropathy, such as patients with diabetes mellitus, may have an altered response to either motor or sensory stimulation. *Reg Anesth Pain Med 2003;28:479-482.*

Key Words: Diabetes, Nerve block, Sciatic nerve, Ultrasound.

For a successful nerve block, local anesthesia must be deposited in the vicinity of the nerve. Exactly how close to the nerve the injection must be made is unclear. The 2 most popular techniques are inducing a paresthesia or generating a motor response via electrical stimulation. It is generally accepted that motor stimulation should be present at 0.2 to 0.5 mA.[1,2] However, the maximal intensity of current still consistent with a successful block is unknown. It is possible that patients with underlying neurologic dysfunction may have altered responses to nerve stimulation either by electricity or contact (paresthesia). We report 2 cases of patients with diabetes mellitus who demonstrated a decreased sensitivity of their sciatic nerves to electrical stimulation and introduce the use of ultrasound to facilitate sciatic nerve localization at the popliteal fossa.

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Accepted for publication June 9, 2003.

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1098-7339/03/2805-0021$30.00/0
doi:10.1016/S1098-7339(03)00227-X

Case Reports

Case 1

A 58-year-old, 96-kg male was scheduled for removal of a plate and screws from his distal fibula under a popliteal fossa block. His comorbidities included hypertension, obesity, hyperlipidemia, and non–insulin-dependent diabetes mellitus. The patient had diabetes mellitus for almost 10 years and carried the diagnosis of neuropathy. On preoperative lower extremity examination, the patient had normal motor function and decreased sensory function below his knees to cold temperature. After informed consent and the application of standard American Society of Anesthesiologists monitors, the patient was placed in the prone position. Two mg of intravenous midazolam were given for sedation. Traditional landmarks were identified for the posterior popliteal approach to the sciatic nerve.[3] A high-resolution ultrasound (180 plus, Sonosite, C11 probe, 11 mm broadband curved array transducer with a frequency of 4-7 MHz, Bothell, WA) was used to confirm the location of the sciatic nerve. The setup for the use of the ultrasound is depicted in Fig 1. The primary operator held the ultrasound probe, covered in a sterile sheath, in the left hand and the stimulating needle in the right hand. The ultrasound probe was placed 6 cm above the popliteal crease and 1 cm lateral to midline. The

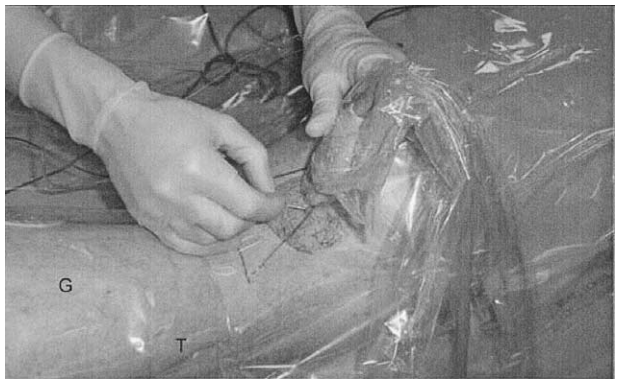

Fig 1. Ultrasound-guided approach to the sciatic nerve. Patient in prone position with the popliteal space exposed. The patient's head is to the right in this picture. The ultrasound probe is held in left hand while the needle is held in the right hand. For this patient, the sciatic nerve was identified 6 cm cephalad from the popliteal crease and 1 cm lateral to the midline. A second operator is needed to control the nerve stimulator and the injection sequence. Abbreviations: G, gastrocnemius; T, tuberosity of tibia.

second operator controlled the nerve stimulator (Stimuplex, B. Braun, Bethlehem, PA) and the injection syringes. A cross-sectional image of the sciatic nerve was identified proximal to its division into the common peroneal nerve and the tibial nerve. A 22-gauge, 50 mm B-bevel stimulating needle (Stimuplex, B. Braun, Bethlehem, PA) was inserted while stimulating at 1.5 mA. Although real-time ultrasound images confirmed perineural location of the needle, no motor responses were identified. The stimulating current was increased to 2.6 mA and weak plantar flexion and foot inversion were observed. We repositioned the needle several times but failed to improve the motor response to less current. The patient denied experiencing a paresthesia or dysesthesia at any time. Given that the ultrasound image demonstrated correct needle position, we injected 30 mL of 1.5% lidocaine with epinephrine 5 μg/mL. The local anesthetic distributed circumferentially around the sciatic nerve (Fig 2B) and the patient developed a rapid block, as indicated by loss of motor and sensory function in his foot. The surgery took 45 minutes and was completed without any additional sedation. The patient was discharged home and a follow-up phone call revealed that the block resolved approximately 8 hours after the injection.

Case 2

A 63-year-old, 76-kg female with insulin-dependent diabetes mellitus, hypertension, and coronary artery disease presented for a little (5th) toe bunionectomy under a popliteal fossa nerve block. Her diabetes had been present for more than 5 years and had only required insulin for the past month. The patient had no appreciable motor or sensory deficits on preoperative examination. After informed consent, the patient was positioned and monitored as described for Patient 1. Using ultrasound guidance in a similar manner as with Patient 1, a 22-gauge, 50-mm B-bevel stimulating needle was inserted 5.5 cm above the popliteal crease and 1 cm lateral to the midline. The nerve stimulator was set at 1.5 mA. Again, the ultrasound identified the perineural location of the needle. Because no motor response was identified, the current was increased slowly to 2.4 mA before weak plantar flexion and foot inversion were noted. Several attempts were made to elicit a motor response at lower currents, but these failed. The patient denied experiencing a paresthesia or dysesthesia at any time. As with Patient 1, 30 mL of 1.5% lidocaine with epinephrine 5 μg/mL was injected under ultrasound visualization. The local anesthesia distributed around the sciatic nerve forming a "doughnut sign" similar to that in Patient 1. A rapid block developed indicated by loss of motor and sensory function in Patient 2's foot. The surgery proceeded without incident and no further sedation was given. The patient was discharged home and a follow-up phone call revealed that the block resolved approximately 7 hours after local anesthetic injection.

Discussion

These 2 cases are examples of atypical responses to nerve stimulation. Traditionally, practitioners assume adequate location of the needle when the appropriate muscle twitch is identified at 0.2 to 0.5 mA. Although the maximal amount of current still consistent with a successful block is unknown, it

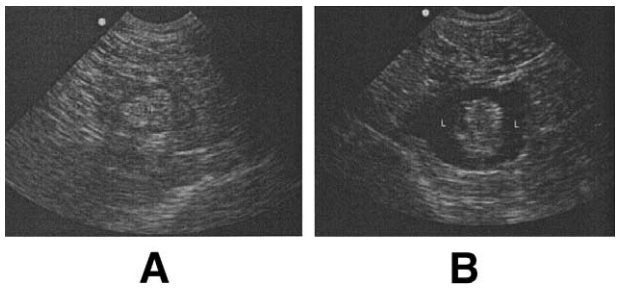

Fig 2. (A) Cross-sectional view of the sciatic nerve before injection. The dot represents anterior. N indicates the sciatic nerve before division into the common peroneal nerve and the tibial nerve. (B) Cross-sectional view of the sciatic nerve after injection demonstrating the "doughnut" sign. Abbreviations: N, sciatic nerve; L, lidocaine.

was our convention, before the use of ultrasound, to abort nerve blocks if muscle twitches could not be identified at <0.7 mA. This stems from our clinical experience that a suboptimal block often results if greater current is used, presumably because the tip of the needle is too distant from the target structure.[4]

Discussion of the mechanics of nerve stimulation is warranted. The ability of a nerve stimulator to generate a motor response is based on the distance of the stimulus from the target as well as the duration and intensity of the current used.[4] Hadzic et al recently demonstrated a wide range of accuracies in various commercially available nerve stimulators.[5] They found a median error of 2.4% when attempting to deliver currents of 0.5 mA. The authors noted that this error in current administration could obscure nerve localization and possibly result in iatrogenic injury. Our nerve stimulator was tested by our biomedical engineering department and found to be without significant error. In addition, the amount of current we used (>1.5 mA) was not found to be associated with clinical error by Hadzic et al.[5] Therefore, we believe our inability to generate an adequate motor response was not the result of mechanical problems with the nerve stimulator.

Patients with diabetes mellitus are known to have underlying neurologic dysfunction. The etiology of this is unclear but involves a progressive impairment of sensory and motor function.[6] In addition, studies indicate that patients with diabetes mellitus experience progressive decreases in nerve conduction velocity and amplitude in sensory and motor nerves.[7] To our knowledge, no data exist to support or refute the concept that patients with underlying neurologic dysfunction present more difficulty with block placement or are at a higher risk of iatrogenic injury. In our 2 cases, the stimulating needle was satisfactorily positioned based on the ultrasound images and the success of the blocks. The fact that supranormal current levels were necessary to generate a motor response may reflect underlying neurologic dysfunction. Choyce et al demonstrated an inconsistent motor response to electrical stimulation in neurologically normal patients who experienced a paresthesia,[1] suggesting that patients may not always develop an appropriate motor response. However, 13% of their patients required supplementation and 9% required general anesthesia. Therefore, one could argue that with this high failure rate, an absence of a correlating motor response simply suggests that the needle was not in the correct position. In addition, in our cases, there was a lack of both paresthesia and motor response at <2.4 mA.

Ultrasound has been shown to facilitate the performance of brachial plexus blockade at the axillary, supraclavicular, and infraclavicular locations.[8-10] We confirm the findings of Ootaki et al, who first described the ultrasound appearance of a "doughnut sign" appearing around the axillary artery and brachial plexus while performing ultrasound guided infraclavicular blocks.[10] As indicated in Fig 2, the injected solution of local anesthesia can be seen surrounding the sciatic nerve. This is thought to represent contact of the perineural tissue with local anesthesia. It is our clinical experience that higher frequencies (7 MHz) are needed to distinguish neurologic structures from surrounding tissue. This is in contrast to vascular structures, which are easy to distinguish even at lower frequencies because of the sharp ultrasound interface created by blood and surrounding tissue. One limitation of ultrasound is that, when a needle passes perpendicular to the ultrasound beam, it is not always apparent where its tip is located. To help minimize inaccuracies, many ultrasound companies have devised needle guides to direct the needle to the target of interest.

In summary, we described the successful use of ultrasound to guide the placement of a popliteal fossa block in 2 patients with diabetes mellitus in whom adequate nerve stimulation or paresthesia was not obtainable. A randomized trial examining the efficacy of ultrasound-guided versus conventional nerve block in patients with diabetes mellitus could be helpful answering the concerns raised by these case reports.

References

1. Choyce A, Chan V, Middleton W, Knight P, Peng P, McCartney C. What is the relationship between paraesthesia and nerve stimulation for axillary brachial plexus block? *Reg Anesth Pain Med* 2001;26:100-104.
2. Cousins MJ, Bridenbaugh PO. Neural Blockade in Clinical Anesthesia and Management of Pain. 3rd ed. Philadelphia, PA: Lippincott-Raven, 1998:188.
3. Brown DL. Atlas of Regional Anesthesia. 2nd ed. Philadelphia: WB Saunders, 1999:123.
4. Pither CE, Raj PP, Ford DJ. The use of peripheral nerve stimulators for regional anesthesia: A review of experimental characteristics, technique, and clinical applications. *Reg Anesth* 1995;10:49-58.
5. Hadzic A, Vloka J, Hadzic N, Thys D, Santos A. Nerve stimulators used for peripheral nerve blocks vary in their electrical characteristics. *Anesthesiology* 2003;98: 969-974.
6. Amanda A, Boyko E, Ahroni J, Stensel V, Forsberg R, Smith D. Risk factors for diabetic peripheral sensory neuropathy: results of the Seattle prospective diabetic foot study. *Diabetes Care* 1997;20:1162-1167.
7. Partanen J, Niskanen L, Lehtinen J, Mervaala E, Siitonen O, Uusitupa M. Natural history of periph-

eral neuropathy in patients with non-insulin dependent diabetes mellitus. *New Engl J Med* 1995; 333:89-94.
8. Ting PL, Sivagnanaratnam V. Ultrasonographic study of the spread of local anaesthetic during axillary brachial plexus block. *Br J Anaesth* 1989;63: 326-329.
9. Kapral S, Krafft P, Eibenberger K, Fizgerald R, Gosch M, Weinstable C. Ultrasound-guided supraclavicular approach for regional anesthesia of the brachial plexus. *Anesth Analg* 1994;78:507-513.
10. Ootaki C, Hayashi H, Amano M. Ultrasound-guided infraclavicular brachial plexus block: an alternative technique to anatomical landmark-guided approaches. *Reg Anesth Pain Med* 2000;25:600-604.
11. Harris M, Eastman R, Cowie C. Symptoms of sensory neuropathy in adults with NIDDM in the U.S population. *Diabetes Care* 1993;16:1446-1452.