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In Reply:—In our study,[1] we connected the negative (cathodal) electrode of the nerve stimulator to the insulated needle. We reported that we considered our "stimulating needle to be close to the nerve when the stimulating current that elicited a motor response was <1 mA." This was the same current reported by Pither *et al.*,[2] Singelyn *et al.*,[3] and Mansour.[4] We actually used currents less than 1 mA. I reexamined our data and found that the currents used ranged from 0.5 to 0.8 mA (mean ± SD, 0.71 ± 0.1 mA). The stimulation intensities used for each elicited motor response were as follows: (1) eversion: 0.63 ± 0.12 (range, 0.5-0.8 mA); (2) inversion: 0.75 ± 0.12 (range, 0.5-0.8 mA); (3) plantar flexion: 0.7 ± 0; and (4) dorsiflexion: 0.77 ± 0.10 (range, 0.7-0.8 mA).

The ideal stimulus intensity required to stimulate a nerve has not been clearly defined. Pither *et al.* stated that movement is elicited in the appropriate muscle when the needle tip is likely to be 1-2 cm from the nerve and that 0.5-1 mA is required when the needle is touching or very close to the nerve.[2] Magora *et al.* showed that 0.5 mA was needed for direct stimulation of the obturator nerve and that their blocks were unsuccessful when 1-3 mA were needed to elicit a motor response.[5] Riegler found that currents ranging from 0.2 to 1.5 mA were sufficient for localization of the brachial plexus whether the interscalene, supraclavicular, or axillary approach was used.[6] A review of the minimum currents used by Riegler showed 0.66 ± 0.4 mA for the interscalene, 0.71 ± 0.03 mA for the supraclavicular, and 0.72 ± 0.03 mA for the axillary approach. These stimulating intensities are the same as the ones we used in our study. Shannon *et al.* accepted 0.6 mA as their endpoint with their lateral femoral cutaneous block.[7] In the new edition of Cousins and Bridenbaugh, a stimulus intensity of 0.5 mA was recommended.[8]

Our hesitancy to use currents less than 0.3 mA was precipitated by the occurrence of an intraneural injection with 0.2 mA during one of our trial blocks, before we formally started our study. The subject had severe shooting pain to his foot during the initial injection of 2 ml, and although the needle was withdrawn 1 mm, he had paresthesias for 1 week. Singelyn *et al.* noted the occurrence of paresthesias with stimulating currents "less than 1 mA."[3] In our clinical practice, we use stimulating intensities of 0.3-0.8 mA.

Bridenbaugh and Crews stated that the "injection of 1 to 2 ml of local anesthetic will immediately abolish nerve stimulation and muscle contraction if the tip of the needle is at the site of the nerve."[8] This has been our experience, as well as that of other investigators.[2,9,10] As stated by Dr. Vloka, this rapid response is the result of the nerve being displaced away from the needle tip. This phenomenon has been confirmed in studies wherein air produced the same response as the local anesthetic.[11] If the needle tip is beyond the nerve and if the shaft of the needle is causing the stimulation, then the injection will not change the motor response. In this case, the needle should be withdrawn slightly and the test repeated.

In our initial study sessions, we used initial currents of 2-3 mA because these were the currents recommended by Riegler.[6] We then decreased the current output as we approached the target nerve. After four study sessions, we used initial currents of 1.5-2 mA because 3 mA was painful. Dr. Vloka will probably agree that the initial current used is less important than the actual current when the injection was made.

Dr. Vloka made calculations based on "1.0-mA stimulus intensity used by Benzon" when we stated clearly that we used currents "less than 1 mA." Perhaps it was our fault and may be we should have been more specific.

The 98% success rate of Vloka and Hadzic is to be expected because they "stimulated the division of the popliteal nerve that predominantly innervated the surgical area."[12] It was also not surprising that the common peroneal nerve was the nerve that was usually stimulated first in their lateral approach group because the common peroneal nerve is located laterally, in relation to the tibial nerve. These two points emphasize the importance of knowledge of the anatomy involved; simply demonstrating nerve stimulation at low current is not enough to ensure adequate block.[2,13]

Dr. Vloka stated that they performed sensory evaluations in their study.[12] However, they did not assess, in detail, the sensory blockade of the areas in the foot innervated by the different branches of the sciatic nerve, *i.e.*, the posterior tibial, deep peroneal, superficial peroneal, and sural nerves. Incomplete blockade of some of the areas innervated by the branches of the sciatic nerve may have been masked by adequate sensory anesthesia in the operative area.

Although there may be a common epineural sheath as Dr. Vloka mentioned, there may also be a sheath within the nerves. In two study sessions in our study,[1] we found that partial blockade of the posterior tibial nerve involved the area innervated by the medial calcaneal branch of the tibial nerve, with no blockade of the medial and lateral plantar branches.

Based on Dr. Vloka's publications, it appears that the lateral approach to sciatic nerve blockade in the popliteal fossa is reliable and should be used more frequently. We use the posterior approach simply because of familiarity with the technique. We have used this approach even in patients in the lateral position and found it simple and effective. If we find that inversion or combined inversion/plantar flexion (the two elicited foot movements associated with complete sensory blockade of the foot[1]) is difficult to elicit, then we use the double injection technique.[14] In this technique, we inject two 15-ml injections after identification of the tibial (elicited plantar flexion) and peroneal components (elicited dorsiflexion or eversion) of the sciatic nerve.

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Spinal Cord Injury in Patients with Undiagnosed Cervical Spine Fractures

To the Editor:—Muckart *et al.* blamed tracheal intubation for the spinal cord injuries sustained by two patients with undiagnosed cervical fractures, who were given anesthesia and having surgery for other injuries.[1] This conclusion is not tenable. Their frank report made it clear that no particular attention was given to the disposition of the head and body, so that it is possible that the period of intubation was the *only* time that reasonable alignment occurred. The fact is that Muckart *et al.* presented no evidence other than that the patients were paralyzed after surgery. The cause is unlikely to be simple—what caused the quadriparesis reported after awake intubation and positioning in a patient with a diseased but stable neck?[2] The possible case of spinal cord damage after direct laryngoscopy that Muckart *et al.* cite is suspect because the intubation (which failed) may have been necessitated by neurologic deterioration rather than being the cause of it and was complicated by severe hypotension, acidosis, and hypoxemia.[3] Direct laryngoscopy produces little movement below C3,[4] not "maximal movement and extension of the entire cervical spine."[1]

When treating patients with actual or suspected cervical abnormality, we should concentrate on maintaining spinal cord blood flow (SCBF). SCBF autoregulation is believed to be unreliable in disease, so that hypotension can cause cord ischemia; severe hypotension has caused quadriplegia in normal patients.[5] This means avoiding hypotension at all times and taking care with positioning. In some patients, the SCBF may be so unstable that even maintaining normotension, awake intubation, and positioning is insufficient to prevent quadriparesis.[2]

Muckart *et al.* did not tell us whether their patients became hypotensive. I should be surprised if they did not because both had serious injuries (multiple gun shot wounds and broken legs), and cord damage itself causes hypotension. If hypotension was closely related to intubation, this could constitute evidence in support of their belief that it caused the cord damage. However, it would also be possible that any hypotension at induction resulted from hypovolemia. My "most likely" explanation of the cord injury sustained by Muckart *et al.*'s patients is that their SCBF was at risk because of cervical trauma, and cord ischemia resulted from hypotension during surgery.

All patients with a history of neck trauma are at risk of the subsequent development of acute spinal cord injury, whether a fracture is present or not. Care should certainly be taken with intubation and positioning, but avoiding hypotension is probably even more important.

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