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Inability to Consistently Elicit a Motor Response following Sensory Paresthesia during Interscalene Block Administration

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Background: **Two methods of nerve block based on eliciting neural feedback with the block needle currently exist. The paresthesia technique uses sensory feedback to ascertain that the needle tip is close to the nerve. By contrast, a peripheral nerve stimulator makes use of motor responses to electrical stimulation. The relation of motor responses to an electrical peripheral nerve stimulator and sensory nerve contact (paresthesia) had not been studied.**

Methods: **Thirty consecutive unpremedicated patients who presented for shoulder surgery with interscalene block anesthesia were prospectively studied. Interscalene block was performed by the single paresthesia method of Winnie, using an insulated or noninsulated needle connected to a peripheral nerve stimulator with the power off. At the precise point of paresthesia, the peripheral nerve stimulator was turned on, and the current was slowly increased to 1.0 mA with a pulse width of 0.2 ms. Presence and location of any motor responses were observed and recorded.**

Results: **All patients had easily elicited paresthesias. The site of first paresthesia was to the shoulder in 73% of patients. Only 30% of patients exhibited any motor response to electrical stimulation up to 1.0 mA. There was no relation between site of paresthesia and associated motor nerve response.**

Conclusion: **Elicitation of paresthesia does not translate to an ability to elicit a motor response to a peripheral nerve stimulator in the majority of patients.**

WINNIE'S original description[1] of the interscalene block technique used the elicitation of a single paresthesia to the arm or hand in an awake patient as evidence that the tip of the needle was contacting the brachial plexus nerve roots. This study was designed to prospectively evaluate whether elicitation of paresthesia, or sensory response, during interscalene block was necessarily associated with the ability to elicit a motor response by peripheral nerve stimulation. The hypothesis was that the sensory response to nerve contact may be dissociated from any motor response to electrical nerve stimulation in some cases. This was the first clinical study to examine the relations between paresthesia and response to electrical nerve stimulation with the needle in a single position during peripheral nerve block.

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Materials and Methods

After approval by the Institutional Review Board of the Hospital for Special Surgery and informed consent, 30 consecutive patients with American Society of Anesthesiologists physical status class I–III were studied. Exclusion criteria included any patient with a history of neurologic disease, diabetes, or neuropathy. All patients were scheduled to have shoulder surgery with anesthesia by interscalene brachial plexus block. Patients were unpremedicated and unsedated. In all 30 patients, interscalene block was performed by a single paresthesia method as originally described by Winnie.[1] A 22-gauge, 3.8-cm needle was advanced slowly at the C6 (cricoid) level until the first reported paresthesia. The needle was connected to a peripheral nerve stimulator (Digi Stim III; Neuro Technology, Houston, TX) with the power turned off. The nerve stimulator had fresh batteries, which were checked by a digital voltmeter before each study patient. At the precise moment that a paresthesia was reported, the needle was stopped and immobilized. The location and character of the sensory response was recorded. The peripheral nerve stimulator was turned on by an assistant, and the electrical current was slowly increased in 0.1-mA increments with a 0.2-ms pulse width until a maximum current of 1.0 mA was reached. In patients 1–10 and 21–30, a noninsulated Quincke point needle (Becton-Dickinson, Franklin Lakes, NJ) was used. In patients 11–20, an insulated needle (Braun Stimuplex, Bethlehem, PA) was used. The patient's shoulder, arm, and hand were observed by the anesthesiologist performing the block and by a second observer. If any motor response (movement or twitching) occurred in response to electrical nerve stimulation, the presence and location were recorded. After electrical stimulation at 1.0 mA or the occurrence of a motor response (whichever occurred first), 1.5% mepivacaine with 1 mEq/ml bicarbonate and 5 μg/ml epinephrine was injected in divided doses for a total volume of 50 ml. A single observer measured sensory block onset by pin prick at 5 and 15 min after local anesthetic injection. Similarly, motor onset was measured at 5 and 15 min by hand-grip dynamometry and by ability to lift the extremity against gravity at the deltoid or biceps muscle. Motor strength was assessed and graded for the triceps, flexor dig comm, and interossei muscles, as well. The operating surgeon, who was blinded to the block technique, was asked to clinically grade the interscalene block at the procedure's conclusion as "poor," "good," or "excellent."

Table 1. Site of Paresthesia and Associated Motor Responses

	Site of Motor Response				
Site of Paresthesia	Deltoid	Triceps	Biceps/ Finger	Hand	Pectoral
Shoulder	2	1	1*	0	0
Arm	2	0	0	1	0
Elbow	1	0	0	0	0
Hand	0	0	0	1	0

* Patient who exhibited a motor response using an insulated needle.

Statistical Analysis

Unless otherwise indicated, data are expressed as mean ± SD. Data were entered into Statview for Windows (SAS Institute Inc., Cary, NC) statistical package. Motor strength at baseline and 15 min after injection was by two-tailed paired *t* test. Block characteristics, motor strength, and sensory patterns between patients who did and did not exhibit a motor response to electrical stimulation were compared by chi-square and two-tailed unpaired *t* test as appropriate. $P < 0.05$ was considered significant.

Results

Each of the 30 patients had an easily elicited paresthesia. The site of the first elicited paresthesia was to the shoulder in 22 patients, to the arm in 6 patients, and to the hand in 2 patients. Therefore, 73% of the paresthesias elicited were to the shoulder. Nine of the 30 patients studied (30%) had evidence of a motor response to electrical nerve stimulation after paresthesia. A motor response to electrical stimulation was observed in eight patients (40%) in the noninsulated needle group and in one patient (10%) in the insulated needle group. The site of paresthesia and associated motor responses are presented in table 1. There was no relation between the site of paresthesia and associated motor response. When a motor response was elicited, the mean electrical current amperage necessary to elicit the motor response was 0.2 ± 0.1 mA (range, 0.1–0.4 mA).

All patients had clinically successful blocks. There was no significant difference in block characteristics between patients who exhibited a motor response with electrical stimulation and those who did not. No patient required general anesthesia. No patient was able to move the upper extremity immediately after surgery. All patients had good evidence of dense sensory and motor blockade at 15 min after local anesthetic injection. Mean diminution in hand-grip strength was 87 ± 15% (range, 44–100%) from baseline (preblock; $P < 0.001$ [paired *t* test]). Surgeon grading of the block quality was recorded for 27 patients. Twenty-six patients (96%) had "excellent" anesthesia, and one patient (4%) had "good" surgical anesthesia.

Discussion

This was the first clinical study to examine the relations between a sensory response (paresthesia) and a motor response to electrical stimulation in peripheral nerve or plexus block. The ability to elicit paresthesia in every patient in this study constituted evidence that nerve contact was made by the tip of the needle in every case. In addition, the fact that all patients had successful surgical anesthesia and sensory evidence of brachial plexus block constituted evidence that the tip of the needle was located within the interscalene space. Nevertheless, evidence of sensory nerve contact, *i.e.*, paresthesia, and presence of the needle in the plexus did not translate to an ability to elicit motor nerve stimulation by peripheral nerve stimulator in 70% of the patients in the 0–1.0 mA range. The only conceivable explanation for these observations is that, although the needle contacted a nerve root, in the majority of cases, this contact was only with a sensory component or fascicle of the nerve root. Despite sensory nerve contact, the tip of the needle was sufficiently remote from any motor fascicles or motor nerve components that visible evidence of motor nerve stimulation did not occur.

In clinical practice, motor stimulation with a peripheral nerve stimulator at amperages below 0.5 mA signifies a close proximity of the tip of the needle to nerve. In this study, the tip of the needle was not close enough to the motor nerve to elicit muscle contraction at amperages up to 1.0 mA.

We sought to determine whether separation of sensory and motor responses would occur with either noninsulated or insulated needles because both are used clinically. The 30% increase in ability to cause motor nerve stimulation with the noninsulated needle compared with the insulated needle can be easily explained by the less-precise location of the stimulating current in relation to the motor nerves in the plexus. However, this was not formally compared by random assignment to needle type. In all likelihood, the shaft of the needle was close enough to motor nerve fascicles to result in the increased incidence of motor nerve stimulation. An interesting outcome was that in the 30% of patients in whom a motor response did occur, the stimulating current necessary to produce the response was very low, 0.2 ± 0.1 mA (range, 0.1–0.4 mA). This indicated that the needle was either in very close contact or was distant from the motor nerve components of the nerve roots, *i.e.*, almost an all-or-nothing response was observed.

With slow, deliberate advancement of the needle, a sensory paresthesia to shoulder was encountered first in an overwhelming majority of the patients (73%). This surpasses the 45% incidence of shoulder paresthesia first encountered in the study published by Roch *et al.*[2] The reason for the large percentage of shoulder paresthesias can be understood by careful examination of the anat-

omy of the brachial plexus at the cricoid level, where interscalene block is performed. The nerve roots at this level are compact and narrowly stacked in a cephalocaudad axis. The properly advanced needle trajectory is along this axis. The C5 and C6 nerve roots, which supply sensory innervation to the shoulder, would therefore be expected to be contacted first, resulting in the shoulder paresthesias as reported by 73% of patients.

The concept of the needle's first contact usually occurring with the more cephalad, more superficial C5–C6 nerve roots is also supported by the recently published findings of Silverstein *et al.*[3] These investigators used a nerve stimulator to locate the brachial plexus. They encountered deltoid twitches, biceps twitches, or both in 100% of their study patients using a peripheral nerve stimulator to stimulate the C5 or C6 nerve roots.

Use of a nerve stimulator has proven to be a useful technical support in peripheral nerve or plexus blockade. It is most useful when blocking a peripheral nerve or plexus of nerves with a large complement of motor neurons. The femoral nerve is an excellent example of a predominantly large motor nerve to the quadriceps muscles. Use of a peripheral nerve stimulator to elicit quadriceps contractions is easily accomplished and interpreted and results in an excellent success rate.[4] By contrast, paresthesias are more difficult to elicit when probing the femoral nerve and are often subtle in character when they do occur. A nerve stimulator is also very useful and yields excellent success when used to locate and block a compact plexus of nerves with an adequate motor nerve representation. The brachial plexus at the cricoid level for interscalene block represents such a situation. By contrast, if a nerve is purely or mostly sensory (*e.g.*, the lateral femoral cutaneous nerve), a nerve stimulator can be used for sensory feedback,[5] but muscle contraction will not result. When the plexus of nerves is not compact in nature (*e.g.*, the brachial plexus at the axillary level where the nerves are terminal and more anatomically separated), a nerve stimulator with a single twitch yields lower success rates.[6] This may reflect the existence of anatomic septae, which may act to interfere with the spread of local anesthetic within the axillary sheath. The use of multiple paresthesias has been proposed on this basis. Therefore, more than a single motor response is often sought after when blocking the brachial plexus at the axillary level.[4]

Winnie[1] discussed the structure of the peripheral nerve, pointing out that the motor fibers exist on the outer mantle, by contrast to the sensory fibers, which are more central in location. Although the explanation of the findings of this study is dependent on the anatomic separation of sensory and motor nerve fibers, Winnie's concept cannot completely explain the results of this study.

Incidence of severe or permanent nerve damage associated with use of the nerve stimulator is rare. In the overwhelming majority of cases, the nerve stimulator identifies the sought-after nerve or nerves without being intraneural. However, as reinforced by the cases reported by Benumof,[7] this does not mean that we can become complacent with regard to technique. For this reason, the findings of this study are clinically relevant in that they show that nerve contact can be made in the absence of a motor response.

This study showed that a sensory response (paresthesia), presumably due to nerve contact, was not associated with ability to elicit a motor response in 70% of patients. This lack of motor responses occurred in the majority of patients despite increasing amperage to 1.0 mA, which exceeds the minimal value accepted by most anesthesiologists. The converse of these findings may be more clinically relevant. Results of this study provided evidence that a lack of motor response does not rule out the possibility of sensory nerve contact by the injection needle. Conversely, when probing to elicit a motor response to nerve stimulation, attention must be paid to the patient's separate sensory responses, if any. Subsequent to our initial report, our findings were reproduced and our data interpretations were reiterated by the results of a recently published study by Choyce *et al.*[8] of 72 patients who received axillary brachial plexus block. These investigators used a protocol with almost identical methodology to that which we had previously published, with the exception that a noninsulated needle was advanced into the axilla. Paresthesia was associated with a motor response to electrical stimulation up to 0.5 mA in 77% of these patients. Therefore, these investigators found, using our experimental model, that 23% of patients lacked a motor response in the axillary brachial plexus. Further studies of this nature should be performed for other plexus or peripheral nerve blocks as well.

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