

the shoulder: the supraclavicular and the axillary nerves. The first of these is not derived from the brachial plexus, but arises from the superficial cervical plexus. In theory, to achieve optimal postoperative analgesia, all sensory nerves should be blocked.

Interscalene nerve block has been the gold standard for shoulder surgery, as it covers all relevant nerves except the supraclavicular nerves. We believe that suprascapular nerve block should, particularly for patients receiving total shoulder arthroplasty, be supplemented with additional blocks. Leaving one or more of the pertinent nerves without any local anesthetic may cause unnecessary postoperative pain after shoulder surgery.

Competing Interests

The authors declare no competing interests.

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Intraoperative Considerations of the Suprascapular Nerve Block

To the Editor:

Hussein *et al.*¹ make a strong case for the utility of the suprascapular nerve block for analgesia after shoulder surgery, and the substitution of this block for those who cannot undergo an interscalene block. However, it should be noted that, in all of the studies the authors reviewed, these blocks were placed for analgesia, to supplement general anesthesia, rather than for surgical blockade. This is an important distinction, because many practitioners utilize the interscalene block as a primary anesthetic, in combination with propofol for sedation. Given its limited area of innervation, a suprascapular block alone cannot be used in this fashion, and even the addition of a peripheral axillary nerve block does not provide complete anesthesia for the shoulder

capsule and skin overlying the area of the incisions for open or arthroscopic shoulder surgery. In considering the differences between a suprascapular and interscalene nerve block, there are a number of advantages to the use of deep sedation, typically with propofol, with the more comprehensive interscalene block, *versus* that of general anesthesia with a more limited block. These advantages include reduced incidence of postoperative nausea and vomiting^{2–5}; earlier return of eating, drinking, and ambulation²; significantly shorter discharge times^{2,3,5}; higher likelihood of bypassing the postanesthesia care unit^{3,4}; and a lower incidence of unexpected admissions for ambulatory procedures.^{3,4}

Competing Interests

The authors declare no competing interests.

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In Reply:

We thank Dr. Musso *et al.* for the comments on our recently published paper.¹ They correctly point out that the shoulder joint innervation is not limited to the suprascapular nerve. While we agree with this statement, it seems that the contribution of other nerves is modest as compared to the suprascapular nerve. Indeed, the suprascapular block does not provide a full sensory block; however, when it comes to postoperative analgesia, the differences we observed between suprascapular and interscalene blocks were not clinically important. This suggests that the use of multimodal analgesia is sufficient to mitigate any difference in analgesic efficacy between the two blocks.

We also thank Dr. Brotman *et al.* for their comments. Our work¹ demonstrated the utility of suprascapular block as an analgesic alternative to interscalene block for shoulder surgery. It is clear that we did not propose a swift transition to suprascapular block as a new care standard for shoulder surgery. Interscalene block, by virtue of its blockade of the brachial plexus at the level of the roots, continues to be the care standard. With some adjustments to the dose, injectate distribution, and intraoperative sedation level, the interscalene block can provide adequate surgical anesthesia. However, this does not dismiss the ability of interscalene block alternatives to provide surgical anesthesia. In cases where it is desirable to avoid both general anesthesia and phrenic nerve block, and when the number of injections is not a barrier, determined regional anesthesiologists may wish to consider either a combination of (1) suprascapular and infraclavicular blocks,² or (2) suprascapular, axillary, supraclavicular, and the lateral pectoral nerve blocks,³ in order to achieve surgical anesthesia. In our clinical experience, these combinations can provide reliable surgical anesthesia, if needed.

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Catching a Unicorn: Neostigmine and Muscle Weakness—Not Neostigmine for All, but Quantitative Monitoring for Everyone!

To the Editor:

We read the paper by Murphy *et al.*¹ with great interest. Their conclusions that administration of 40 µg/kg of

neostigmine after recovery of the train-of-four ratio to greater than 0.9 (measured with acceleromyography) causes no subsequent decrease in the train-of-four ratio, nor clinical signs of muscle weakness at around 15 min after administration, are supported by their well-constructed study. They restate the 2016 editorial “that neostigmine should be routinely administered unless full neuromuscular recovery has been documented with quantitative neuromuscular monitoring.”^{1,2}

However, we would like to respectfully question five areas of the study.

Neostigmine May Cause Weakness Secondary to Depolarizing Neuromuscular Blockade

Neostigmine-induced *depolarizing* neuromuscular blockade was not excluded in this study because twitch height was not recorded. In 1959, Churchill-Davidson recognized that therapeutic doses of neostigmine could cause neuromuscular block and suggested this was a depolarizing block, evidenced by its similarity to that caused by decamethonium with reduction of twitch height, with no fade.³

Chapter 35 of Miller’s *Textbook of Anesthesia*, edited by Professor Murphy, states: “The adverse physiologic effects of neostigmine in the setting of complete neuromuscular recovery can potentially have negative respiratory consequences in postoperative surgical patients. The mechanisms proposed for this effect include sensitivity of the upper airway muscles to an overabundance of acetylcholine with desensitization of the ACh receptor, depolarizing blockade, or an open channel blockade.”⁴

Clinical Tests for Weakness Are Unreliable

Clinical tests of muscle strength, as used by Murphy *et al.*,¹ to detect residual neuromuscular block are unreliable, insensitive, and may be performed with train-of-four ratios less than 0.5.^{5,6} The authors acknowledge this limitation of their trial, stating, “Although the incidence of observable upper airway events was not increased in patients administered an anticholinesterase, neostigmine might produce more subtle effects on the respiratory system that were not detectable on clinical examination (*e.g.*, upper airway critical closing pressure).”¹ Interestingly, while all patients were able to perform 5-s head lift before extubation, nine patients could not pass this test 15 min after arrival in recovery.

Timing Is of the Essence

Neostigmine-induced clinical weakness is expected to peak at 5 to 10 min after IV administration. This is the likely timeframe not only for tracheal extubation but also impaired upper airway patency and respiratory function due to paradoxical muscle weakness. Murphy *et al.*’s