In Reply:

We thank all the authors who commented on our study regarding the intraneural local anesthetic injection. Although the general concern regarding this approach is understandable, the conclusions in the received comments apparently do not stem from scientific evidence. Allocating resources to avoid intraneural local anesthetic injection should not be based on common sense but on clear evidence that it could be worse than the extraneural injection.

Jiang et al. suggested that the axonal damage could be attributable to a higher concentration/volume of the local anesthetic deposited around the nerve fascicles after injection within the epineurium. Furthermore, the injection pressure below 15 psi used in our study could be too high for sciatic nerve causing ischemia. Although the hypothesis of nerve fascicle ischemia as the cause of axonal damage is plausible, the possible mechanism described by Jiang et al. is not supported by evidence. A previous animal study demonstrated that nerve fascicle damage ensued only when the intraneural injection pressure was greater than 25 psi. Moreover, in our previous study, we found that the amplitude reduction seen after injection out of the epineurium was comparable with that observed after intraneural injection. When the sciatic nerve, with an intact epineurium, was exposed to a clinically relevant concentration of lidocaine, the epineurium contained 30% of the “total neural local anesthetic” when the equilibrium was reached. Unfortunately, this drop in concentration caused by the epineurium seems to reduce the block effectiveness but not to prevent axonal damage.

In our 2016 work, we directly compared the intraneural injection with the extraneural one (named as subparaneural), hoping to demonstrate that intraneural injection was as safe as the subparaneural at the electrophysiographic test. Surprisingly, we found that both techniques resulted in 100% of axonal nerve damage 5 weeks after surgery. As highlighted by both the Swenson et al. and Lai and Rosenblatt letters, the study was underpowered for this secondary outcome; nevertheless, our result is worth considering. Swenson et al. underlined that in our trial a blind radiologist found that 22% of extraneural injection (named subparaneural) were actually inadvertent intraneural. We had also found that 16% of the intraneural injections were actually subparaneural. Data were reanalyzed after recording group allocation, and the results were comparable with those of the intention-to-treat analysis.

Inadvertent intraneural injection was frequent before ultrasound technique introduction. Reported neurologic complications were rare but, worryingly, they did not diminish with the use of ultrasounds. In our previous studies, no patient reported clinically evident neurologic symptoms. In their letter, Swenson et al. correctly pointed out that a phone call is not equivalent to a clinical visit. Nevertheless, a routine clinical visit after peripheral nerve block, as well as an electrophysiologic test, is hardly performed regardless of the approach used.

New insights on the different layers enveloping the sciatic nerve have allowed new approaches to the sciatic block, but peripheral nerve block safety did not develop at a similar rate. Our knowledge regarding possible nerve damage after peripheral nerve block does not consider the actual structure of the sciatic nerve. Animal studies are dated or have investigated only the intraneural injection effects. No study addressed the effects of the purportedly safer extraneural injection (in or out of the paraneural sheath).

Common experience would suggest avoiding intraneural injection, taking for granted that local anesthetic injection outside of the epineurium is safer and effective at the same time. Instead, our previous findings raise doubt about whether the axonal damage after peripheral nerve block was related to the site of local anesthetic injection or to local anesthetic itself.

In conclusion, we do not propose that the intraneural local anesthetic injection could be better than extraneural, because we agree with Lai and Rosenblatt that systemic local anesthetic toxicity after peripheral nerve blocks is rare, and the increased effectiveness of intraneural injection may have a clinical impact only in case of sciatic nerve block as a sole anesthetic technique. Instead of spreading unjustified alarm, we would rather stimulate further investigation to verify to what extent the struggle to avoid intraneural injection is justifiable. It is evident how this is an ethically, economically, and scientifically grounded issue.

Competing Interests

The authors declare no competing interests.

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References

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Supraclav Suprascap
Interscalene Shoulder
Surgery: Comment

To the Editor:

W e read with great interest the noninferiority trial by Auyong et al.1 We greatly appreciate the authors for their novel technique, the anterior suprascapular nerve block. They have shown that it provides noninferior analgesia compared to that of interscalene block, and at the same time preserves vital capacity and has lower incidence of Horner syndrome.

Our question is: when the anterior suprascapular block, which did not target the superior trunk, offers a noninferior analgesia to interscalene (targeting the roots and trunks), how did the suprascapular block targeting the superior and middle trunk not offer a noninferior analgesia? As per the authors, for suprascapularized block, a large volume of local anesthetic is required as the cross-sectional area of the brachial plexus increases at the supraclavicular level. This could have been a good explanation if the brachial plexus divisions were targeted, but the authors had targeted the superior and middle trunk in the suprascapular group.

When 15 ml volume of local anesthetic was deposited at the suprascapular nerve, laterally away from superior trunk had spread and blocked the axillary and subscapular nerves, arising from the posterior division of superior trunk (in the anterior suprascapular group), how did the same volume of local anesthetic that was deposited directly on the superior trunk (in the supraclavicular group) not block them?

In table 2 of Auyong et al., (PACU Pain and Opioid Consumption—Interscalene, Suprascapular, and Anterior Suprascapular), all values in all the three groups have SD more than the mean. For example, the average postoperative numerical rating scale score at 60 min postsurgery (scored from 0 to 10) in the interscalene group, has mean ± SD of 2.1 ± 2.6, which implies the values ranges from −0.5 to 4.7 (2.1 to 2.6 is equal to −0.5 to 2.1 + 2.6 = 4.7). Logically pain score and opioid consumption cannot be represented negatively when the minimum score is zero. When the SD is more than the mean while analyzing data which is nonnegative (pain score, opioid consumption), it implies nonnormal or skewed distribution. The primary outcome of this trial is pain in the postanesthesia care unit and one-way ANOVA has been applied. For the ANOVA to be applied, the data has to be of normal distribution. If data collected is of nonnormal distribution, the recommendation is to use the median as a measure of central tendency and the interquartile range as a measure of dispersion.2

We would like to get clarification from the authors as to whether the data collected for pain scores at 60 min postsurgery was of nonnormal distribution and skewed, and whether application of mean as measure of central tendency in such nonnormal distribution has hindered the supraclavicular group to meet the noninferiority criteria in comparison to the interscalene group.

Competing Interests

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