Intraneural injection during nerve stimulator-guided sciatic nerve block at the popliteal fossa

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Background. Exact location of the needle tip during nerve stimulation-guided peripheral nerve blocks is unknown. Using high-frequency ultrasound imaging, we tested the hypothesis that intraneural injection is common with nerve stimulator-guided sciatic nerve (SN) block in popliteal fossa.

Methods. Forty-two patients scheduled for hallux valgus repair were studied. Sciatic block at the popliteal fossa was accomplished using nerve stimulation. When a motor response was elicited at <0.5 mA (2 Hz, 0.1 ms), 40 ml of local anaesthetic (LA) was injected. Using ultrasound (Titan, Sonosite, 5–10 MHz), the diameters and area of the SN were measured before and after the injection. The presence of nerve swelling and proximal or distal diffusion of LA were also assessed. Intraneural injection was defined as nerve area (NA) increase of ≥15% and one or more additional ultrasonographic markers (nerve swelling, proximal–distal diffusion within epineural tissue). Clinical neurological evaluation was performed 1 week after the block.

Results. Post-injection NA increase ≥15% was seen in 32 (76%) patients [0.54 (SD 0.19) cm² vs 0.76 (0.24) cm²; P<0.05]. Nerve swelling with fascicular separation was observed in 37 (88%) patients; proximal and distal diffusion of LA were present in six (14%) and 14 (38%) patients, respectively. Intraneural injection criteria were met in 28 (66%) patients. Greater NA increase was present in patients with fast block onset [61 (45) vs 25 (33)%; (Diff 35% 95% CI 61–9%); P<0.05]. No patient developed neurological complications.

Conclusions. Intraneural (subepineurial) injection is a common occurrence after nerve stimulator-guided SN block at the popliteal fossa, yet it may not inevitably lead to neurological complications.


Keywords: anaesthetic techniques, regional, sciatic; monitoring, ultrasound; nerve, damage

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Nerve stimulation is an established method of nerve localization during application of peripheral nerve blocks. It is generally accepted that the electrical current at the tip of the insulated needle evokes a motor response before the needle makes a contact with or enters the nerve. This concept is thought to reduce the risk of mechanical needle trauma to the nerve and decrease the risk of an intraneural injection.1 2 However, recent data suggest that evoked motor response may not be always present even when needle appears to be in physical contact with the nerve.3–5 Moreover, data from animal models suggest that motor response to electrical stimulation occur only after the needle pierces the epineurium.6 Assuming adequate images, ultrasound guidance during nerve blocks permits detection of the site of the local anaesthetic (LA) injection, and recognition of the signs of intraneural injection, such as an increase in nerve area (NA) and swelling of the nerve.7–9 Recent reports suggest that intraneural injection may not be invariably associated with neurological injury.7 10 We hypothesized that intraneural (subepineurial) injection...
during nerve stimulator-guided sciatic nerve (SN) blocks at the popliteal fossa may be frequent. Our secondary hypotheses were that intraneural (subepineural) injections result in faster onset of the nerve block and may not inevitably lead to neurological complications.

Methods

After approval by the Institutional Review Board (Hospital Clínic, Universitat de Barcelona, Spain) and written informed consent, forty-two ASA I–III patients scheduled for correction of hallux valgus surgery were studied. Exclusion criteria were known neuropathy in the lower extremities, morbid obesity (body mass index ≥40), and limited range of motion of the ipsilateral foot.

Patients were premedicated (midazolam 1 mg) and positioned in the prone position with the foot slightly elevated on a cushion. An anaesthesiologist with experience in ultrasound peripheral nerve imaging and not involved in the patient care performed the examinations and measurements before and after the block. A linear ultrasound transducer 5–10 MHz (L38, Titan Sonosite, Bothel, EEUU) was used to image the SN at approximately 7 cm above the popliteal crease. The position of the transducer at the site of optimal image acquisition of the short axis of SN was carefully marked on the skin at both sides of the transducer. Images of the short-axis view of the SN, distance from skin to the nerve, major and minor diameters were acquired and stored for analysis. Nerve cross-sectional area was obtained post-imaging by applying an ellipse containing both diameters.

The nerve block was then performed by an anaesthesiologist who did not participate in data analysis. After infiltration of the skin at the marked insertion site with 1 ml of 2% lidocaine, a 22 G, 50 mm, 15° bevel needle (Stimuplex D™, Braun, Melsungen, Germany) was connected to a nerve stimulator (Stimuplex HNS 11™, Braun, Melsungen, Germany) and vertically advanced. The end goal was to obtain either a plantar or dorsal flexion of the foot with current intensity of <0.5 mA (2 Hz; 0.1 ms). Minimum current intensity required to maintain evoked motor response and the distance to the skin at which the nerve was located were also recorded. After nerve localization, 40 ml of 1.2% mepivacaine were injected using 20 ml syringes at a rate of approximately 20 ml min⁻¹ and with utmost care to avoid resistance (high pressure) on injection. Any symptom of pain or discomfort on injection were actively solicited and recorded if reported by the patients. Immediately after the injection, ultrasound scanning was used to acquire the specified views by the blinded anaesthesiologist. Nerve measurements were performed and additional signs suggestive of intraneural (subepineural) injection were sought: (i) Nerve swelling (defined as the change in US echogenicity owing to an increase of hypoechoic areas within the nerve); (ii) Proximal subepineural diffusion of LA (defined as the presence of a concentric hypoechoic area around the nerve 2–3 cm proximal to the injection site); (iii) Distal subepineural diffusion (defined as the presence of a hypoechoic area following both branches of the SN, tibial, and peroneal nerves distal to the injection site). A blinded radiologist with expertise in ultrasound nerve imaging analysed the specified changes in nerve dimension and appearance (swelling) and proximal–distal extension of the injectate. Intraneural (subepineural) injection was defined as ≥15% increase of the NA and at least one additional ultrasound criterion documented ultrasonographically after the block by an anaesthesiologist and concurred by the radiologist.

The onset of sensory and motor nerve block in the distribution of the terminal branches of the SN (tibial nerve, superficial peroneal, and deep peroneal nerves) was assessed in 10 min intervals by an anaesthesiologist blinded to the ultrasound images. The sensory block was tested by pinprick and graded according to a 4-level qualitative scale (3=normal sensation, 2=analgnesia, 1=anaesthesia). The onset of motor block was graded on 4-level qualitative scale: (3) full strength; (2) weak response against resistance; (1) paresis; (0) no motor strength. Presence of full sensory-motor block after 20 min in both the tibial and peroneal nerves was considered as a fast onset of block.

When the quality of the block was considered adequate, the surgery was allowed to start. At the end of the procedure, patients were transferred to the post-anaesthesia care unit and block regression was evaluated. Twenty-four hours later, sensory-motor assessment was performed to determine any remaining block, neurological deficit, or both. Patients were specifically asked to report any symptoms of paraesthesia, disaesthesia, or residual motor block; any symptoms suggestive of neurological dysfunction were noted. One week after the block placement, a follow-up neurological examination in the surgical office evaluated the presence of the aforementioned symptoms or neurological injury.

Statistical analysis

We conducted an observational study to detect the incidence of our primary outcome variable, increased NA after SN block under standard nerve stimulation technique. Based on previous reports, increased NA after injection may occur in up to 30% of patients. However, our clinical impression is that increase of NA suggestive of an intraneural injection may occur in more than 60%. Therefore, sample size was calculated to detect an incidence of increased NA of 60% with an α error of 0.05 and a power of β=0.2. Accordingly, the needed number of study subjects was calculated as 40. Data are presented as mean (sn) or percentage. To compare nerve dimensions before and after block χ² test and paired Student’s t-test were performed where indicated. Differences in collected variables between intraneural or non-intraneural injections and...
normal vs fast-block onset were assessed using Student’s t-test for continuous data, \( \chi^2 \) test, or both for categorical data. The Pearson test was used to analyse the relation between two continuous parameters. A \( P \)-value of 0.05 was considered significant.

## Results

All enrolled patients successfully completed the protocol and are included in the statistical analysis. The studied cohort consisted of four men and 38 women of mean age 63 (11) yr, weight 66 (11) kg, and height of 158 (8) cm.

No patient complained of pain during needle insertion or injection of LA, although six patients reported paraesthesia during nerve localization. Using a hand-feel technique during injection of LA, injection pressure was gauged to be normal (<20 psi) in all injections. The sought motor responses were successfully elicited in all patients at intensities \( /C20 \) 0.5 mA [0.3 (0.09) mA]. Six patients had complete block within 10 min and 25 patients within 20 min after the injection. Therefore, onset of block was a met criteria for the fast onset in 25 patients (60%). All patients had full sensory-motor block at 30 min after injection (Fig. 1); no patient required additional analgesia or sedation for completion of the surgical procedure. Mean time for complete block regression was 285 (56) min.

Clinical examination performed at 24 h and 1 week post-block did not reveal impairment of sensory or motor function or symptoms of nerve injury in any of the patients. SN measures in the transverse view at 7 cm above the popliteal crease before and after the block are shown in Table 1. The increase in diameters and area pre- and post-injection was statistically significant (Table 1). Twenty-eight patients (66%) met the specified criteria for intraneural injection (Table 2 and Fig. 2). Swelling, proximal, and distal diffusion occurred more frequently in patients with increase in NA of \( \geq 15\% \) (Table 2).

The depth of the SN (skin-to-nerve, as assessed by US) was similar before and after the injection; [2.4 (0.4) vs 2.5 (0.6) cm; \( r^2=0.79 \)]. However, the location of the SN

![Fig 1](https://academic.oup.com/bja/article-abstract/102/6/855/262536)
measured with the needle during NRS technique was deeper [3.5 (0.6) cm] than the depth assessed by US ($P<0.05$) (Fig. 3).

There was no statistical difference between minimal current intensity at injection between the group of 28 patients with intraneural injection criteria [0.33 (0.1) mA] and the group of 14 patients whose injections did not meet the criteria of intraneural injection [0.35 (0.05) mA]. The increase in NA (%) did not correlate with the minimal stimulation intensity (mA; Fig. 4).

There was no statistical difference in the number of patients with fast-block onset between the group of 28 patients with intraneural injection criteria (17/28, 60%) and the group of 14 patients without (6/14, 43%); odds ratio [95% CI] 2.1 (0.6–7.6)]. However, patients with faster block onset (within 20 min) had a significantly greater increase in the NA [61 (45)% vs 25 (33)%; $P<0.05$; Fig. 5].

**Discussion**

Our data suggest that intraneural injection after low current intensity (<0.5 mA) nerve localization of the SN at the popliteal fossa may be a common occurrence. However, no patient developed clinical evidence of neurological dysfunction after the block regression, a finding in agreement with clinical experience that nerve blocks carry relatively low risk of neurological complications.

Our findings are in agreement with several previous anecdotal reports suggesting that intraneural injections may be relatively common with nerve stimulator-guided nerve blocks. The most pertinent study to our findings...
is that of Robards and colleagues, who have also found high incidence of intraneural injections after nerve stimulator-guided sciatic block at the popliteal fossa. With the advent of high resolution ultrasound imaging, intraneural injections can be recognized as an increase in nerve dimension after injection. In addition, a change in nerve appearance is often seen, typically described as a swelling of the nerve with increase in echo-lucent areas indicative of fluid (LA) accumulation within the nerve. In the SN block at popliteal space, a thin layer of LA surrounding the nerve and its two main divisions (tibial and common peroneal nerves) proximally and distally, has also been reported during intraneural injection, indicating spread of the LA within the epineural sheath. In this study, we used a minimum increase of ≥15% in NA immediately after the injection as a sign of intraneural injection. The ≥15% criterion was selected to decrease the chance of error owing to repeated assessments. We chose

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**Fig 3** (A) Correlation between the deepness of the nerve measured by ultrasound before and after the block. (B) Correlation between the deepness of the nerve measured by ultrasound before the injection and the real deepness at which the needle found the nerve (motor response at intensity <0.5 mA).

**Fig 4** Correlation between nerve area increase (%) and minimal nerve stimulation intensity.
this criterion because in a preceding pilot control study, the inter-rater and within-rater variability during repeated sciatic NA measurements was <10%.

Similar to the findings of Robards and colleagues,12 up to 79% of the patients in our study may have had some degree of intraneural injection, based solely on NA increase. However, our study design did not include ultrasound imaging during injection, therefore real-time observation of the increase in NA during the block was not possible. For that reason, our criteria for intraneural injection required the presence of at least one more suggestive sign on ultrasound examination, possibly resulting in a falsely lower incidence of intraneural injection.12

Only six patients (14%) in our study reported paraesthesia during nerve localization. Other investigators have similarly reported the presence of paraesthesia at current intensity <0.5 mA in 23% of blocks during axillary block and as high as 73% during interscalene block when higher intensity current is used (1 mA).3–5

Our findings are intriguing as they suggest that the nerve stimulation-guided SN block may not reliably prevent needle-nerve contact (paraesthesia) and intraneural injections as was the common assumption among clinicians. Indeed, in canine models, intensity of the stimulation current at which motor response occurs does not correlate well with the needle-nerve distance.6 15 Likewise, the increase in NA in our study did not correlate with the minimum current intensity at which motor response was elicited.

Another interesting finding in our study is that ultrasound underestimated the depth of the nerve when compared with the assessment by the stimulating needle by approximately 1 cm. This discrepancy between the ultrasound and stimulating needle estimation of the depth of the nerve has been previously reported also in the infracavicular approach of brachial plexus block.16 This discrepancy is conceptually trivial as it is likely caused by the pressure exerted by the transducer during imaging. However practically, the discrepancy may have implications in clinical practice particularly for clinicians who use ultrasound to pre-locate SN before solely using nerve stimulation to localize SN.

The main limitation of our study is that the ultrasound transducer was removed and re-applied between measurements. This could have caused inconsistencies in the calculation of sciatic NA. However, we took an extra effort to minimize the potential errors by marking the exact position of the transducer and keeping the same spatial orientation (left/right).

Secondly, our findings of high incidence of intraneural injections with nerve stimulator-guided SN block should not be extrapolated to blocks of other peripheral nerves. SN is surrounded by a thick epineurium that also extends and protects fascicles and groups of fascicles with its extension, known as interfascicular epineurium. As such, the connective tissue comprises 72–75% of the cross-sectional area of the SN. This has two important ramifications to our model. The first one is that the thick epineurium and adipose tissue within the nerve may prevent elicitation of a motor response at current intensity <0.5 mA. The second one is that high ratio of connective vs nerve tissue carries a relatively low risk of fascicular injury after nerve puncture, as the needle is more likely to separate rather than pierce the fascicle.17

Therefore, an intraneural injection with SN at the popliteal fossa may carry less risk of nerve injury than say, intraneural injection into the ulnar nerve where >80% of the nerve comprises nerve tissue. Injections within the epineurium of the SN (but outside of fascicles) may result in a faster and more complete block compared with extraepineurial injection and slow diffusion of L.A.14 18 However, intentional intraneurial injection to block the SN cannot be recommended until monitoring methods become available to reliably rule out an interfascicular injection.

In conclusion, under conditions of our study, intraneural (subepineurial) LA injection during nerve stimulation-guided SN block at the popliteal fossa may be a common occurrence without imminent neurological injury (up to 66%). Based on our findings, intra-epineural injections may result in faster onset of nerve block. Further research is necessary to confirm these findings and their clinical implications.

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