No Clinical or Electrophysiologic Evidence of Nerve Injury after Intraneural Injection during Sciatic Popliteal Block

Xavier Sala-Blanch, M.D.,* Ana M. López, M.D., Ph.D.,* Jaume Pomés, M.D.,† Josep Valls-Sole, M.D., Ph.D.,‡ Ana I. García, M.D.,† Admir Hadzic, M.D., Ph.D.§

ABSTRACT

Background: Intraneural injection during nerve-stimulator–guided sciatic block at the popliteal fossa may be a common occurrence. Although intraneural injections have not resulted in clinically detectable neurologic injury in small studies in human subjects, intraneural injections result in postinjection inflammation in animal models. This study used clinical, imaging, and electrophysiologic measures to evaluate the occurrence of any subclinical neurologic injury in patients with intraneural injection during sciatic popliteal block.

Methods: Twenty patients undergoing popliteal block were enrolled; 17 patients completed the study protocol. After tibial nerve response was achieved by nerve stimulation (0.3–0.5 mA; 2 Hz; 0.1 ms), 20 ml mixture of mepivacaine (1.25%) and radiopaque contrast (2 ml) were injected. Location and spread of the injectant were assessed by ultrasound measurements of the sciatic nerve area before and after injection, and by computed tomography. In addition to clinical neurologic evaluations, serial electrophysiologic studies (nerve conduction and late response studies using predefined criteria) were performed at baseline and at 1 week and 3 weeks after the block for signs of subclinical neurologic dysfunction.

Results: Sixteen injections (94%, 95% CI: 71–100%) met criteria for an intraneural injection. Postinjection nerve area on ultrasound increased by 45% (95% CI: 29–58%), $P < 0.001$. Computed tomography demonstrated fascicular separation in 70% (95% CI: 44–90%), air within the nerve in 29% (95% CI: 10–56%), contrast along bifurcations in 65% (95% CI: 38–86%), and concentric contrast layers in 100% (95% CI: 84–100%). Neither clinical nor electrophysiologic studies detected neurologic dysfunction indicating injury to the nerve.

Conclusions: Nerve-stimulator–guided sciatic block at the popliteal fossa often results in intraneural injection that may not lead to clinical or electrophysiologic nerve injury.

What We Already Know about This Topic

- Ultrasonography indicates peripheral nerve blocks often result in intraneural needle placement, and intraneural injection results in acute inflammation in animals

What This Article Tells Us That Is New

- In 16 patients, an injection into the epineurium of the sciatic nerve at the popliteal fossa did not lead to postoperative neurologic dysfunction as assessed by serial physical examinations and nerve conduction studies

THE exact location and disposition of local anesthetics during peripheral nerve blockade were poorly understood before the introduction of ultrasound in the practice of regional anesthesia. Ultrasound monitoring during nerve blockade has shown that intraneural injection of local anesthetic is a common occurrence.[1–5] Data from animal studies indicate that evoked motor response to low-current intensity (less than 0.5 mA) nerve stimulation during sciatic popliteal block (SPB) occurs primarily when the needle enters the subepineural space.[6–8] Thus, intraneural needle placement is likely to occur during nerve-stimulator–guided SPB.[1,2,8] Although intraneural injections are thought to carry a risk for nerve injury, reports based on small series of patients suggest that clinically overt neurologic complications are not common.[1–5] However, intraneural injections in animals are associated with postinjection inflammation, although clinical correlation and long-term outcome remain unknown.[8,9] The primary purpose of this study was to determine whether low-

◇ This article is featured in "This Month in Anesthesiology." Please see this issue of ANESTHESIOLOGY, page 9A.

* Staff Anesthesiologist, † Staff Radiologist, Department of Anesthesiology; ‡ Consultant, Neurophysiology Unit, Department of Neurology, Hospital Clinic, University of Barcelona, Barcelona, Spain. § Director of Regional Anesthesia, St. Luke's-Roosevelt Hospital Center, Professor of Clinical Anesthesiology, College of Physicians and Surgeons, New York, New York.

Received from the Department of Anesthesiology, Hospital Clinic of Barcelona, Barcelona, Spain. Submitted for publication September 15, 2010. Accepted for publication May 20, 2011. Support was provided solely from institutional and/or departmental sources. Abstract presented at the American Society of Anesthesiologists Annual Meeting, October 13–17, 2007, San Francisco, California.

Address correspondence to Dr. Sala-Blanch: Department of Anesthesiology, Hospital Clinic C. Villarroel 170, 08036 Barcelona, Spain. xsala@clinic.ub.es. Anesthesiology 2011; 115:589–95

current intensity SPB is associated with subclinical electrophysiologic evidence of neurologic injury. We hypothesized that electrophysiologic studies will reveal some evidence of electrophysiologic subclinical neurologic injury in patients with intraneural injection during SPB.

Materials and Methods

After approval was given by the Institutional Review Board (Hospital Clinic, Barcelona, Spain) and written informed consent was obtained, 20 patients (American Society of Anesthesiologists physical status I–II) undergoing SPB for hallux valgus repair were studied. Exclusion criteria included known history of peripheral neuropathy in the lower extremities, coagulation disorders, morbid obesity (body mass index more than 40 kg/m^2), allergy to local anesthetics or iodine radiopaque contrast, contraindications to computed tomography (CT) imaging, diabetes mellitus, or medical conditions that can lead to imaging artifacts in the popliteal space (*e.g.*, knee prosthesis). Patients who were unable to complete the study or who were noncompliant with the study protocol also were excluded from the study and statistical analysis.

On the day of surgery, all patients had a baseline clinical examination and electrodiagnostic study of both lower extremities to detect any preexisting neurologic disorder.[10]

Patients were premedicated with 1–2 mg intravenous midazolam and placed in the prone position, with the foot to be blocked elevated 10 cm on a cushion. An anesthesiologist with experience in ultrasound imaging who was blinded to the details of the anesthetic procedure and the purpose of the study performed the ultrasonographic examination just before and after the nerve-stimulator–guided block procedure. The ultrasonographic examination consisted of localizing the sciatic nerve with a 5–10 MHz linear transducer (Titan; Sonosite, Bothel, WA), 5–7 cm proximal to the popliteal crease. At this level, the sciatic nerve appears as a hyperechoic oval structure with hypoechoic areas within the nerve. After the best view of the sciatic nerve at its smallest diameter proximal to the point of bifurcation was obtained, the nerve dimensions were determined. The exact position of the transducer was then marked on the skin. Another anesthesiologist performed the SPB block through the posterior approach, using only nerve-stimulator guidance. After the skin was prepared with a povidone-iodine solution and infiltrated (1 ml lidocaine, 2%), a 50-mm long, 22-gauge, 15-degree bevel stimulating needle (Stimuplex D 50; B. Braun Melsungen AG, Melsungen, Germany) was advanced at a 90-degree angle to the skin plane. The nerve stimulator was set to deliver 1.5 mA current (2 Hz; 0.1 ms; Stimuplex NHS; B. Braun). When a tibial nerve response (plantar flexion of the foot) was elicited with a current of 0.3–0.5 mA, a mixture of 18 ml mepivacaine (1.25%) and 2 ml radiopaque contrast (Ultravist 300; Schering AG, Berlin, Germany) was injected. A syringe-injection pump (Asena GH MK III; Alaris Medical UK Ltd., Basingstoke, United Kingdom) was used to ensure a constant rate of injection (20 ml/min) and variability in the spread of local anesthetic. Injection pressure was monitored by an in-line pressure monitor (BSmart; Concert Medical, LLC, Norwell, MA). When the injection pressures exceeded 15 psi or the patient reported pain on injection, the injection was stopped, and the needle was slightly withdrawn before the injection resumed. To reassess the nerve dimensions (area) and location of the injectant, the second ultrasonographic examination was performed immediately (1 min) after completion of the block injection at the identical transducer position as that used for the preblock evaluation. Serial clinical assessments of the block were performed by a blinded anesthesiologist in 10-min intervals to 30 min.

Patients were then transferred to the radiology suite, where the popliteal fossae were imaged by CT (Somaton Sensation 64; Siemens Medical Systems, Erlangen, Germany). A scout view of 512 mm was obtained at the center of the needle insertion site identified by a radiopaque skin marker. A CT image was acquired of the popliteal fossa region 8 cm cephalad and caudad to the level of injection. The images were obtained with a rotation time of 0.5 s, slice collimation of 0.6 mm, 120 kV, and 90 effective mA current. Axial, coronal, and sagittal reconstructions were performed with 3-mm sections. The images were evaluated by a radiologist expert in musculoskeletal CT imaging to help confirm the anatomic location and dispersion of the injectant.

All patients were transferred to the operating room for their scheduled surgery upon completion of CT imaging. Intraoperatively, additional sedation with midazolam was administered if needed. Remifentanil ($0.05–0.2\ mcg \cdot kg^{-1} \cdot min^{-1}$) was infused if the calf tourniquet (inflated 100 mg Hg above systolic blood pressure) became uncomfortable to the patient. Sciatic nerve motor and sensory functions were assessed by an anesthesiologist 24 h after surgery to evaluate recovery of the block. Data on any remaining sensory or motor deficits were recorded during the evaluation.

At 1 and 4 weeks after surgery, identical neurologic evaluations and electrodiagnostic studies were performed. For each procedure, data were recorded, and results were kept blinded until completion of the study.

Data Collection

Clinical Nerve Block. Minimal intensity of the stimulating current (mA) that elicited the tibial motor response, any pain or paresthesias, or injection pressure ≥15 psi during injection were recorded. Sensory block of the tibial nerve (sensation of sole of the foot), superficial peroneal nerve (skin over the dorsal foot), and deep peroneal nerve (interdigital skin between the first and second toe) to pinprick was graded on a 4-point scale: (3) normal sensation, (2) discomfort, (1) analgesia (no pain), and (0) anesthesia (no sensation). Motor block of the tibial nerve (toe flexion) and the peroneal nerve (toe extension) was graded on a 4-point scale: (3) full strength, (2) weak response against resistance, (1) paresis, and (0) paralysis. Fast-onset block was considered when complete motor and sensory block were present at 20 min.

Postblock Imaging. Presence or absence of an intraneural injection was assessed by the anesthesiologist (ultrasound images) and the radiologist (CT images) using the following criteria:

Ultrasound. Intraneural injection was defined as an increase in nerve area greater than 15% from baseline with the presence of proximal and/or distal local anesthetic diffusion of at least 2 cm proximal and distal to the injection level.[2] Nerve area was determined as follows: The largest anteroposterior and mediolateral diameters of the sciatic nerve were measured. The area was calculated by drawing an ellipse that contained both diameters, and values obtained before and after the block were compared. Deposition of the local anesthetic around the nerve *per se* was not counted as the actual nerve diameter increase. Diffusion of local anesthetic was classified as one of the following three patterns or combination thereof: (1) hypoechoic halo around the sciatic nerve, (2) the presence of hypoechoic aliquots of fluid between fascicles, or (3) the presence of hypoechoic aliquots between tibial or peroneal nerves. For the diffusion to qualify as a supporting criterion for an intraneural injection, patterns 2 or 3 had to be present at 2 cm proximal and distal to the injection site.

CT Imaging. Axial and longitudinal slices of the right and left sciatic nerve were analyzed and compared. Distribution of the contrast was assessed along both planes. Unequivocal signs of intraneural injection were defined as: (1) air or contrast within the sciatic nerve and/or (2) fascicular separation at the injection level. Signs suggestive of intraneural injection were defined as: (1) presence of concentric contrast fluid layers around the nerve and (2) contrast or air within and along the sciatic nerve and/or its bifurcations. For the purposes of this study, intraneural injection by CT criteria was defined as the presence of at least one unequivocal sign or both signs suggestive of intraneural injection.

For an injection to be qualified as intraneural or partially intraneural in our study, at least one unequivocal ultrasound or CT sign and two additional ultrasound or suggestive CT signs had to be present simultaneously.

Nerve Function

Clinical Evaluation. The following data were obtained at baseline and at 24 h and 1 and 4 weeks after the block. Motor function was evaluated for dorsal and plantar flexion of the toes using a 6-point scale adapted from the Medical Research Council scale,[11] where 0 indicates absence of movement and 5 indicates normal strength. Sensory function to tactile stimuli using cotton balls, to pinprick, and to vibration sense using a 256-Hz fork applied to the toes was evaluated by a 4-grade scale (0 = absence of sensation and 3 = normal sensation). Achilles' tendon reflexes also were assessed (0 = absence of response, 1 = weak reflex, and 2 = normal reflex). Patient reports of pain or disesthesia were recorded. Numerical data were obtained for each of the parameters measured in the baseline examination and in the postblock examinations.

Electrophysiologic Studies. Conventional electrophysiologic tests[12] were performed at baseline and at 1 and 4 weeks after the intervention. The following data were obtained bilaterally: (1) motor conduction velocity of the deep peroneal and posterior tibial nerves in the knee to ankle segment, and that of the sural and peroneal superficial nerves in the distal third of the leg; (2) amplitude of the action potentials in the common peroneal, posterior tibial, and sural nerve; (3) minimum latency of the posterior tibial nerve F wave from 20 consecutive stimuli; and (4) minimum latency of the soleus H wave. Nerve injury was defined as a change in latency (more than 120%) or in amplitude and conduction velocity (less than 80%) compared with baseline data obtained in the same individual. These values were based on the well-established criteria for the presence of demyelinization and nerve damage[11] and extracted from the normative reference values for the same tests and procedures performed on healthy subjects in our own laboratory.[12]

Statistical Analysis

We chose latency in soleus H wave as our primary outcome variable because the H reflex is conveyed by large Ia afferent fibers and is one of the most susceptible electrophysiologic tests to reflect demyelination.[13] Because epidemiologic studies have reported a 3% incidence of clinical nerve dysfunction after peripheral nerve block, we assumed that subclinical nerve damage caused by intraneural injection may be present in at least 20% of patients.[14] For the paired design (each patient as his own control), sample size was estimated at 16 patients for the two-tailed test to detect a 20% change in latency or amplitude of soleus H wave from baseline to postblock at $\alpha = 0.05$ (type I error), $1\text{-}\beta = 0.80$ (power), and SD of the difference of 0.27. Summary statistics are presented as mean ± SD, 95% CI, or n (%). Each of the seven electrophysiologic signs was tested over the three time points (baseline, 1 and 4 weeks) by repeated measures ANOVA. If overall F ratios reached statistical significance, analyses of pairwise differences were adjusted for multiple comparisons (Bonferroni). Because each of the seven electrophysiologic signs were meaningful to the goals of this study, each overall F ratio was tested for statistical significance at $P < 0.05$.

Data were analyzed using the Software Package for Social Sciences (Version 15.0 [2006]; SPSS Inc., Chicago, IL).

Results

Twenty patients were enrolled, and 17 patients successfully completed the study. One patient was excluded from the study because of signs of preexisting subclinical polyneuropathy during the baseline electrodiagnostic evaluation. Two additional patients were excluded from the analysis because a timely CT scan could not be performed

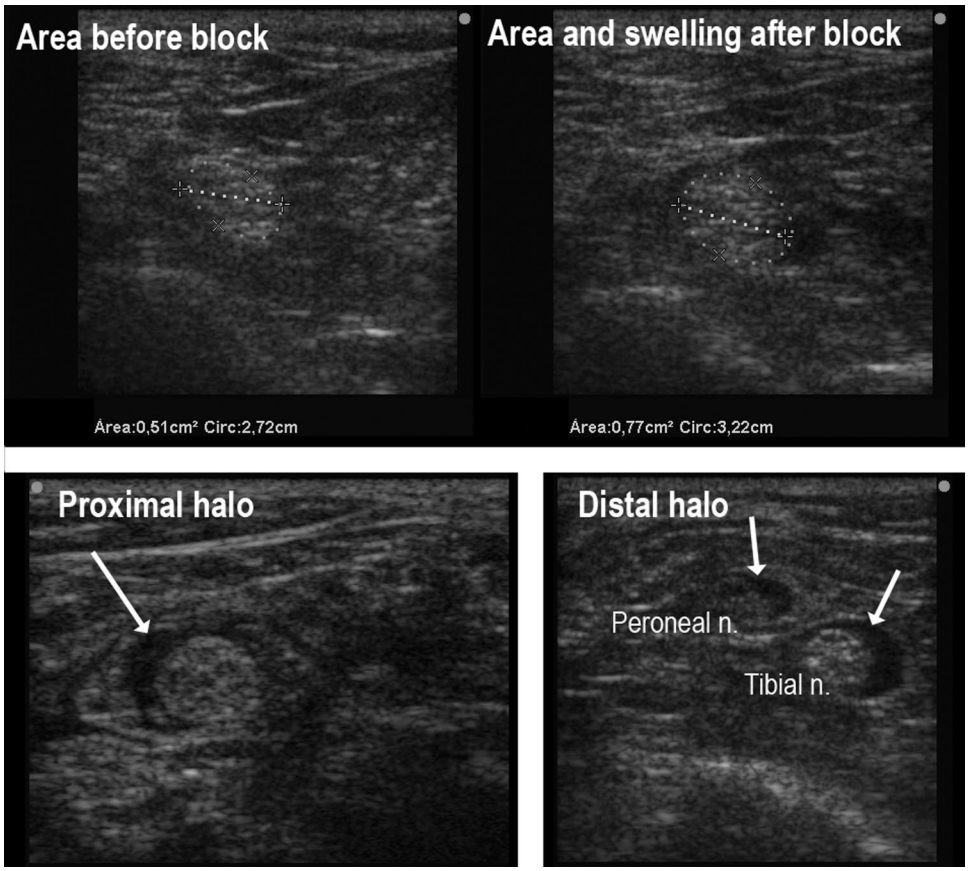

Fig. 1. Ultrasound signs used to define intraneural injection criteria. Ultrasound images demonstrate increased sciatic popliteal nerve dimensions after the block and proximal and distal diffusion of local anesthetic following the path of the nerve and its two divisions (*arrows*).

after the block for technical and organizational reasons. The remaining 17 patients (2 men, 15 women; 63 ± 8 years of age, weight 71 ± 12 kg, height 160 ± 6 cm) successfully completed the study.

Clinical Nerve Block Data

No patient reported paresthesia, pain during nerve localization, or injection pressure more than 15 psi. The minimal current intensity required to maintain evoked motor response to electrical stimulation was 0.37 ± 0.05 mA (range, 0.30–0.44 mA); plantar flexion was obtained in all patients. All blocks were successful, and no patient required supplemental infiltration of local anesthetics by the surgeon or additional sedation during surgery. Seven patients had complete sensory and motor blocks within 20 min, eight patients at 30 min, one at 40 min, and one at 60 min. Blocks regressed completely at 360 ± 110 min (210–480 min) after the injection. Clinical neurologic evaluation 24 h after block placement did not reveal any signs or symptoms of sensory or motor impairment in any patient.

Postblock Imaging Data

Using the aforementioned combined criteria, 16 (94%, 95% CI: 71–100%) injections were judged to be intraneural by the ultrasonographer and/or the radiologist. Postblock ultrasound scan (fig. 1) showed a significant increase in sciatic nerve dimensions compared with preblock values; in 15 patients (88%, 95% CI: 64–99%) the increase was ≥15% (table 1). The average increase in postinjection nerve area was 45% (95% CI: 29–58%; $P < 0.001$). Data on proximal and distal diffusion is shown in table 2. CT examinations were

Table 1. Block-induced Changes in Sciatic Popliteal Nerve Dimensions

—	Preblock	Postblock	Increase	% Increase	*P* Value
Mediolateral diameter (cm)	0.98 (0.88–1.08)	1.19 (1.08–1.30)	0.23 (0.14–0.32)	24 (15–33)	<0.001
Anteroposterior diameter (cm)	0.62 (0.58–0.66)	0.74 (0.67–0.81)	0.12 (0.08–0.16)	19 (13–26)	<0.001
Area (cm^2)	0.51 (0.47–0.55)	0.74 (0.63–0.84)	0.23 (0.15–0.30)	45 (29–58)	<0.001

Data are mean (95% CI).

Table 2. Signs Suggesting Intraneural Injection and Quick Block Onset by Patient

	Ultrasound Signs			CT Signs					
Patient	Area >15%	Proximal Diffusion	Distal Diffusion	Fascicular Separation	Air/ Contrast	Contrast in Bifurcation	Concentric Layers	Quick Block Onset*	Minimum Current Intensity (mA)
1	+	+	+	−	−	+	+	−	0.33
2	+	+	+	−	+	+	+	−	0.4
3	+	+	+	+	+	+	+	+	0.4
4	+	+	+	+	−	−	+	−	0.35
5	+	−	−	+	−	−	+	−	0.44
6	+	+	+	+	−	+	+	+	0.35
7	+	+	+	−	−	−	+	−	0.4
8	+	+	+	+	−	+	+	−	0.4
9	+	+	+	+	+	+	+	+	0.4
10	+	+	+	+	+	+	+	+	0.4
11	−	+	+	−	−	−	+	−	0.4
12	+	+	+	+	−	+	+	−	0.35
13	+	+	−	−	−	−	+	−	0.4
14	+	+	+	+	+	+	+	+	0.3
15	+	+	+	+	−	−	+	−	0.4
16	+	+	+	+	−	+	+	+	0.3
17	−	+	+	+	−	+	+	+	0.3
Total+	15	16	15	12	5	11	17	7	Median 0.4
%	88	94	88	71	29	65	100	41	Range (0.3–0.44)

Only patient 11 did not met criteria for intraneural injection.

* Complete sensory block <20 min.

+ Presence of event; − Absence of event; CT = computed tomography; mA = milliampere.

performed 56 ± 27 min after the block. CT axial scans at the level of needle insertion demonstrated significant changes in sciatic nerve anatomy in all patients compared with the control images of the sciatic nerve on the nonblocked side (fig. 2). CT images documented fascicular separation in 70% (95% CI: 44–90%), air within the nerve in 29% (95% CI: 10–56%), contrast along bifurcations in 65% (95% CI: 38–86%), and concentric contrast layers in 100% (95% CI: 84–100%) (table 2).

Postblock Nerve Function Data

Clinical Follow-up. No patient developed pain, dysesthesia, or weakness at 24 h or at 1 or 4 weeks after surgery. There were no significant changes in sensory-motor function between the baseline and postblock examinations.

Electrophysiologic Data. One patient did not meet the criteria for intraneural injection (patient 11 in table 2) and was excluded from statistical analyses. There were no differences in any of the electrophysiologic signs over the three time points (baseline or 1 or 4 weeks). Because only one patient had missing values for common peroneal nerve (conduction velocity and amplitude) and H wave (ipsilateral and contralateral) of this nerve, mean values for these measures were imputed, and the repeated measures ANOVAs were reanalyzed. The overall F ratios were unchanged with the inclusion of imputed values (table 3).

Discussion

Our data indicate that intraneural needle placement is common during SPB with low-current–intensity electrical nerve localization and can occur without clinically overt neurologic injury.[1–5] Because intraneural injections result in postinjection inflammatory changes in peripheral nerves in animals, we postulated that intraneural injections could result in a subclinical, electrophysiologic neurologic impairment that might have gone undetected by the clinical neurologic examinations used in previous studies.[8]

However, our study found neither clinical nor electrophysiologic evidence of subclinical neurologic injury associated with 16 intraneural injections that occurred during nerve-stimulator–guided SPB.

The exact location of needle placement and disposition of local anesthetic in clinical practice of regional anesthesia were not well understood until the recent introduction of ultrasound guidance. Our current findings concur with those of several other reports and suggest that low-current–intensity, nerve-stimulator guidance during SPB often results in subepineural placement of the needle.[1,2,6] In most studies, motor response to nerve stimulation at very–low-current intensities (0.3–0.5 mA) is accomplished only after the needle tip enters the epineurium of the sciatic nerve. This suggests that the low-current nerve localization that has been a norm with nerve-stimulator–guided nerve blocks often unknowingly resulted in intraneural injections. Indeed, injections in our study invariably led to a needle insertion within the epineural sheath, and an injection within the sciatic nerve as evidenced by ultrasound imaging and CT confirmation.

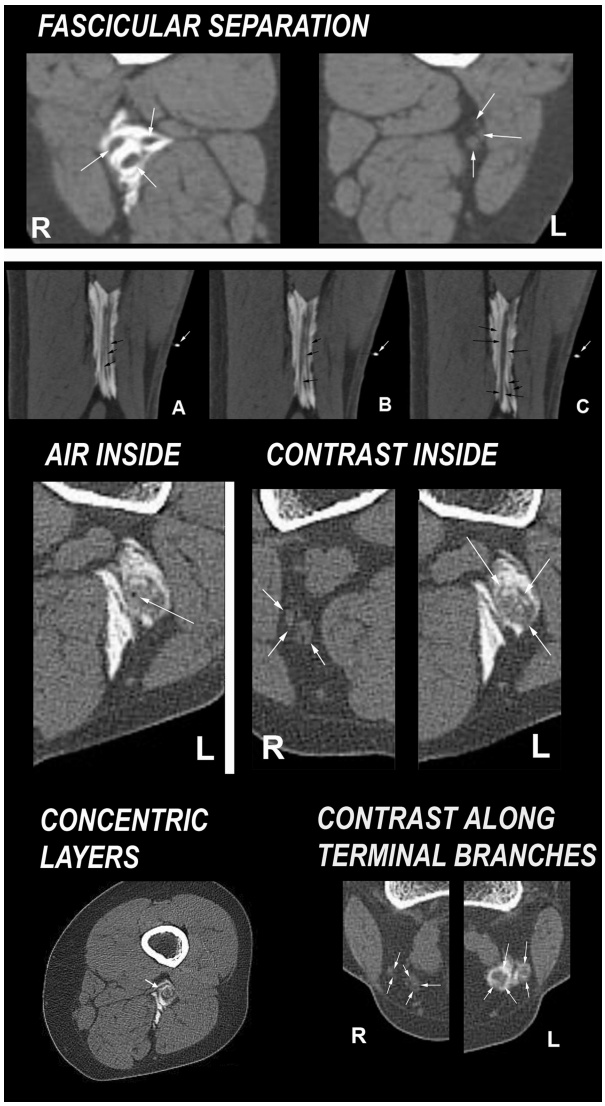

Fig. 2. Computed tomography (CT) scan signs used to define intraneural injection criteria. CT images of the sciatic popliteal nerve after the injection demonstrates fascicular separation at the injection level, the presence of air or contrast inside the nerve, concentric layers of contrast around the nerve, and contrast following both bifurcations distally to the injection level.

The absence of neurologic injury with intraneural injections during SPB in various nerve block models is at odds with conventional thinking that intraneural injection invariably leads to nerve injury.[1–5] However, an intraneural but extrafascicular injection may carry less risk for injury than an intraneural, intrafascicular injection. Mackinnon *et al.* suggested that the physical location of the injectant (intrafascicular *vs.* perifascicular) is a key factor that determines whether neurologic injury will result from an intraneural injection.[16,17] Low incidence of complications after intraneural injections with SPB also may be attributable to the anatomic characteristics of the sciatic nerve in the popliteal fossa. The connective tissue of the sciatic nerve comprises as much as 80% of the cross-sectional area of the nerve, thus redirecting needles primarily through the path of lesser resistance (adipose tissue) rather than through more compact fascicles.[18,19] For instance, needles deliberately inserted into sciatic nerves are more likely to pass between, rather than transverse, the fascicles.[20] Two recent studies in animal models suggest that low-pressure injection is more likely to be associated with interfascicular, rather than intrafascicular, injection.[8,9] In studies that reported intraneural injections without neurologic consequences, resistance to injection was judged to be normal.[1–3,8]

Of note, morphologic changes of the nerves after intraneural injection were described a century ago, when an intraneural injection under direct visual guidance was an accepted practice for peripheral nerve blockade.[21] In their book *L'Anesthésie Regionale*, Victor Pauchet and Paul Sourdat described their observations during intraneural injections: "A fusiform enlargement of the nerve ensues that disappears quickly. The injectant diffuses along both sides, that is why an intraneural injection can exit through the branches that leave the nerve close to the injection site." An entire century later, contemporary experimental studies confirmed these observations by showing that an intraneural injection results in temporary enlargement of the nerve area as imaged by ultrasound.[8,9] In our study, for ethical reasons, we did not perform intraneural injections intentionally; instead, we simply studied the postblock morphologic changes of the sciatic nerve with two imaging techniques. The increase of the nerve area evidenced by ultrasound measurement as well as the fascicular separation observed in CT scans, proximal and distal diffusion of local anesthetic seen by ultrasound, and the presence of contrast along the nerve division in CT scans were all equivalent signs of an intraneural injection, as described by Pauchet.

In conclusion, low-current–intensity, nerve-stimulator–guided SPB commonly results in intraneural, subepineural injection. In our series, none of the patients exhibited clinical or electrophysiologic evidence of neurologic injury. Of note, our findings are relevant to the blocks performed at the smallest diameter of the sciatic nerve proximal to its divergence. However, rates of intraneural injection may vary greatly with more proximal or distal injections. In addition, although intraneural injections may not always cause injury, no information is available with respect to the volume that might be tolerated, whether underlying conditions (*e.g.*, diabetes mellitus) might affect safety, and whether the use of a different needle, a higher concentration of mepivacaine or other anesthetic, or inclusion of other adjuvants, particularly vasoconstrictors, also would be tolerated. Thus far, research on intraneural injections has included only a small number of subjects, and more data are needed to understand the significance of intraneural injections with respect to injury. Consequently, our data should not be interpreted as support for

Table 3. Electrodiagnostic Examinations for all the Parameters Measured

Parameters	Mean ± SD			*P* Value
	Baseline	1 Week	1 Month	
CPN conduction velocity	51.5 ± 4.8	51.5 ± 3.4	51.8 ± 2.6	0.654
CPN amplitude	7.6 ± 2.4	7.5 ± 2.2	7.6 ± 2.1	0.996
Sural nerve conduction velocity	52.6 ± 4.8	51.5 ± 4.8	51.9 ± 4.9	0.346
Sural nerve amplitude	12.4 ± 5.9	11.3 ± 5.3	11.8 ± 5.5	0.206
Latency of F wave	48.7 ± 2.8	49 ± 2.6	49.6 ± 2.3	0.077
Latency of ipsilateral H wave	31.1 ± 1.5	31 ± 1.6	30.8 ± 1.7	0.325
Latency of contralateral H wave	31.2 ± 1.5	30.7 ± 1.5	30.9 ± 1.6	0.329

Analysis performed without the patient who did not met intraneural injection criteria (patient 11 in table 2). One patient had missing values for CPN (conduction velocity and amplitude) and H wave (ipsilateral and contralateral) of this nerve; mean values for these measures were imputed.

CPN = common peroneal nerve.

sciatic intraneural injections during SPB in routine clinical practice or liberally extrapolated to other peripheral nerve block models.

References

1. Robards C, Hadzic A, Somasundaram L, Iwata T, Gadsden J, Xu D, Sala-Blanch X: Intraneural injection with low-current stimulation during popliteal sciatic nerve block. Anesth Analg 2009; 109:673–7
2. Sala Blanch X, López AM, Carazo J, Hadzic A, Carrera A, Pomés J, Valls-Solé J: Intraneural injection during nerve stimulator-guided sciatic nerve block at the popliteal fossa. Br J Anaesth 2009; 102:855–61
3. Bigeleisen PE: Nerve puncture and apparent intraneural injection during ultrasound-guided axillary block does not invariably result in neurologic injury. ANESTHESIOLOGY 2006; 105:779–83
4. Sala-Blanch X, Pomés J, Matute P, Valls-Solé J, Carrera A, Tomás X, García-Diez AI: Intraneural injection during anterior approach for sciatic nerve block. ANESTHESIOLOGY 2004; 101:1027–30
5. Bigeleisen PE, Moayeri N, Groen GJ: Extraneural *versus* intraneural stimulation thresholds during ultrasound-guided supraclavicular block. ANESTHESIOLOGY 2009; 110:1235–43
6. Tsai TP, Vuckovic I, Dilberovic F, Obhodzas M, Kapur E, Divanovic KA, Hadzic A: Intensity of the stimulating current may not be a reliable indicator of intraneural needle placement. Reg Anesth Pain Med 2008; 33:207–10
7. Chan VW, Brull R, McCartney CJ, Xu D, Abbas S, Shannon P: An ultrasonographic and histological study of intraneural injection and electrical stimulation in pigs. Anesth Analg 2007; 104:1281–4
8. Lupu CM, Kiehl TR, Chan VW, El-Beheiry H, Madden M, Brull R: Nerve expansion seen on ultrasound predicts histologic but not functional nerve injury after intraneural injection in pigs. Reg Anesth Pain Med 2010; 35:132–9
9. Altermatt FR, Cummings TJ, Auten KM, Baldwin MF, Belknap SW, Reynolds JD: Ultrasonographic appearance of intraneural injections in the porcine model. Reg Anesth Pain Med 2010; 35:203–6
10. Johnsen B, Fuglsang-Frederiksen A: Electrodiagnosis of polyneuropathy. Neurophysiol Clin 2000; 30:339–51
11. Kendall F, McCreary E: Muscle Testing and Function, 3rd edition. Baltimore, Maryland, Williams & Wilkins, 1983
12. Kimura J: Long and short of nerve conduction measures: Reproducibility for sequential assessments. J Neurol Neurosurg Psychiatry 2001; 71:427–30
13. Buschbacher RM: Normal range for H-reflex recording from the calf muscles. Am J Phys Med Rehabil 1999; 78:S75–9
14. Brull R, McCartney CJ, Chan VW, El-Beheiry H: Neurological complications after regional anesthesia: Contemporary estimates of risk. Anesth Analg 2007; 104:965–74
15. Mackinnon SE, Hudson AR, Gentili F, Kline DG, Hunter D: Peripheral nerve injection injury with steroid agents. Plast Reconstr Surg 1982; 69:482–90
16. Mackinnon SE, Hudson AR, Llamas F, Dellon AL, Kline DG, Hunter DA: Peripheral nerve injury by chymopapain injection. J Neurosurg 1984; 61:1–8
17. Reina MA, López A, De Andrés JA: [Adipose tissue within peripheral nerves. Study of the human sciatic nerve]. Rev Esp Anestesiol Reanim 2002; 49:397–402
18. Sunderland S: The sciatic nerve and its tibial and common peroneal divisions, Anatomical Features, Nerve and Nerve Injuries. Edited by Sunderland S. Edinburgh, Churchill Lvingstone, 1972, pp 1012–68
19. Sala-Blanch X, Ribalta T, Rivas E, Carrera A, Gaspa A, Reina MA, Hadzic A: Structural injury to the human sciatic nerve after intraneural needle insertion. Reg Anesth Pain Med 2009; 34:201–5
20. Pauchet V, Sourdat P: L'Anesthésie Régionale. Paris, Octave Doin, 1914