Comparison of subgluteal sciatic nerve block duration in type 2 diabetic and non-diabetic patients

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Editor’s key points

- Animal models of diabetes show reduced onset and increased duration of peripheral nerve blocks.
- This study aimed to evaluate the difference in block properties between diabetic and non-diabetic patients.
- Using careful evaluation of sensory dysfunction, diabetic patients were found to have a prolonged duration of a sciatic nerve block.
- Further study of the mechanisms of this longer action in diabetic patients is needed.

Background. Although animal studies demonstrated delayed recovery after nerve block in laboratory models of diabetes, the duration of the action of sciatic nerve blocks clinically in patients with diabetes remains to be determined. We studied the duration of a sciatic nerve block in type 2 diabetic patients compared with non-diabetic patients.

Methods. We prospectively included consecutive patients aged 50–80 yr, with type 2 diabetes with minor nerve injury (confirmed with 5.07 at 10 g monofilament test, n=23) and non-diabetic patients (n=49) scheduled for distal lower limb surgery. Before surgery, a subgluteal sciatic nerve block (20 ml of ropivacaine 4.75 mg ml\(^{-1}\)) was performed with an ultrasound approach coupled with nerve stimulation. The primary endpoint was the sensory block duration.

Results. There was no significant difference between groups for age, but haemoglobin A1c and creatinine values were significantly higher in the diabetic group. There was no difference in 5.07 (10 g) monofilament testing, but the diabetic group had lower scores for the 0.4 and 0.07 g tests (\(P<0.01\)). There was no significant difference in the median onset time for the sensory block (25 vs 25 min, NS), but the median duration of the sensory block (21 vs 17 h, \(P<0.01\)) and the motor block (16 vs 12 h, \(P<0.01\)) were higher in the diabetic group. No complication occurred in either group.

Conclusions. These findings demonstrate that diabetic patients with pre-existing incipient neuropathy exhibit delayed recovery from the block with ropivacaine, confirming animal studies.

Clinical trial registration. ClinicalTrials.gov, NCT01704612.

Keywords: anaesthetic techniques, regional; local anaesthetics, ropivacaine; diabetes mellitus, type 2

Accepted for publication: 21 October 2012

Over the past three decades, type 2 diabetic patients have more than doubled, making it one of the major healthcare challenges to all nations.\(^1\) The anaesthetic management of these patients is more challenging because of more frequent difficulties in airway control, association with myocardial dysfunction, renal disease, and the occurrence of perioperative dysglycaemia.\(^1\)–\(^6\) For upper or lower limb surgery in diabetic patients, peripheral regional anaesthesia is an interesting alternative to general anaesthesia because it provides effective analgesia, may decrease haemodynamic complications, and reduce glycaemia dysregulation.\(^5\)–\(^8\) The fear of nerve injury after regional anaesthesia in diabetic patients is a concern that has neither been confirmed nor refuted by the current literature.\(^9\)–\(^11\) Diabetic patients with neuropathy may be considered at an increased risk because of the possibility for double crush syndrome when a chronic axon lesion related to diabetes is associated with an unexpected distal nerve injury related to regional anaesthesia.\(^9\)

In streptozotocin-induced diabetic rats, nerve degeneration is associated with a loss of myelinated and unmyelinated fibres, paranodal demyelination, segmental degeneration, microvascular dysfunction (endothelial alteration), axonal Ca\(^{2+}\) dyshomeostasis (i.e. Ca\(^{2+}\) currents), and mitochondrial dysfunction.\(^12\)–\(^14\) These diabetic rats demonstrated nerve conduction velocity alterations. Several studies observed a decrease in conduction velocity in the sciatic nerve after...
perineural local anaesthetic (LA) agent administration.\textsuperscript{15, 16} In addition, diabetic rats exhibited axonal degeneration and demyelination of the sciatic nerve after administration of LA, which did not occur with saline. This is thought to be related to LA toxicity. However, the precise effects of regional anaesthesia in diabetic patients remain poorly described.\textsuperscript{17, 18} In diabetic patients, no clinical study clearly demonstrated any increased risk of nerve dysfunction or injury due to perineural LA agent injection or needle approach, while several case reports illustrated delay or absence of recovery from the block.\textsuperscript{10, 11, 19} Why diabetes would delay recovery from the block is a controversial subject. One theory is that diabetes induces axonal degeneration.\textsuperscript{20} Axonal degeneration is associated with alteration of nerve sensitivity to LA agents.\textsuperscript{20} Another theory suggests that diabetes reduces the activity of potassium and sodium channels in the nerve fibres, influencing the threshold and the conduction velocity in these neurons.\textsuperscript{12–14} Consequently, the onset time and the block duration after a peripheral nerve block may be modified in diabetic patients.

We undertook a prospective observational single-blinded study comparing a sciatic nerve block in type 2 diabetic and non-diabetic patients, hypothesizing that recovery from the block is delayed in diabetic patients.

**Methods**

**Patients and group**

This trial was a prospective observational study approved by the institutional human investigation committee [Comité de Protection des Personnes, Iles de France, Paris VI (CPP-73-11, Eudra CT 2011: A00737: 34)] and registered on ClinicalTrials.gov (NCT01704612). After checking eligibility criteria and obtaining written informed consent on the day before surgery, consecutive patients aged between 50 and 80 yr and undergoing elective surgery of the lower limb (knee, ankle, foot) were included between July 2011 and March 2012 in two French centres (APHP, La Pitié-Salpêtrière Paris, France; Hôpital Carêmeau, Nîmes, France). Criteria for non-eligibility were as follows: refusal of a sciatic nerve block, age <50 or >80 yr, ASA status >IV, presence of contraindications to local anaesthesia (known allergy to LAs, sepsis), emergency surgery, patients unlikely to be fully co-operative during the study, psychiatric disorders, or abusing alcohol or drugs, and participation in another study within operative during the study, psychiatric disorders, or abusing alcohol or drugs, and participation in another study within psychiatric disorders or abusing alcohol or drugs. Patients with previous history of diabetes (A1c) level >9\% or with type 1 diabetes mellitus (insulin therapy) were not included. Other causes of neuropathy (such as familial, alcoholic, nutritional, and uremic polyneuropathy) were excluded by a history and clinical evaluation.\textsuperscript{19} Moreover, patients with diffuse neuropathy affecting the peripheral nerves, defined as diabetic sensorimotor polyneuropathy with 5.07 (10 g) monofilament test <4 (score: 0–8), were excluded (see the ‘Monofilament test score’ section).\textsuperscript{21–25}

Diabetic patients who were ‘diet-controlled’ only and those requiring insulin were excluded as study participants.

Patients were divided into two groups according to their diabetic status: type 2 diabetic (patients with elevated fasting plasma glucose levels currently receiving an oral hypoglycaemic agent) and non-diabetic patients (control).

**Monofilament test score**

Before operation, monofilament examination was performed bilaterally using a Semmes-Weinstein 10 g (size 5.07) monofilament (Biomedex ITM Laboratoires, Lyon, France) according to previous studies.\textsuperscript{21–25} Briefly, the patient’s eyes were closed and the monofilament was applied to a non-calloused site on the foot (dorsal and plantar) using a smooth motion; the skin was touched, the monofilament was advanced perpendicularly to the skin surface and was bent for a full second and then lifted from the skin. This manoeuvre was repeated 4 times per foot in a random arrhythmic manner. The responses were tallied to produce a score ranging from 0 to 8 (normal (1 point assigned), decreased (0.5 point assigned) or absent (0 points assigned)). A score of 0 represented a complete lack of perception, whereas a score of 8 represented full perception of all stimuli.\textsuperscript{26} Moreover, monofilament 4 g (size 4.56), 2 g (size 4.3), 0.4 g (size 3.61), and 0.07 g (size 2.83) were tested in both groups to detect incipient neuropathy.\textsuperscript{26}

**Ultrasound subgluteal sciatic nerve block**

All patients received oral hydroxyzine (100 mg) 1 h before surgery and were monitored (Sp\textsubscript{O\textsubscript{2}, ECG, non-invasive arterial pressure); then venous access was secured.

In both groups, a sciatic nerve block was performed using ultrasound guidance plus peripheral nerve stimulation. Briefly, patients were placed in the ventral position. The ultrasound transducer (8–12 Hz, GE logiq E, GE Health Care Canada, Mississauga, Ont., Canada) was initially positioned on the skin, transversely in the popliteal region (tibial and fibular sciatic nerve identification). Then the probe was translated to subgluteal region with a continuous sciatic nerve short-axis view. In the subgluteal region, a 100 mm insulated needle (Stimuplex, B Braun, Melsungen, Germany) connected to a nerve stimulator (HNS 12, B Braun) was introduced. The needle was inserted parallel and in line with the ultrasound transducer and then advanced slowly under real-time ultrasound guidance until it was in close proximity to the nerve. The stimulating current was set initially at 2.0 mA (frequency, 1 Hz; time, 0.1 ms) to obtain a sciatic motor response (foot plantar flexion or extension). Then, the current ampage was slowly decreased and minimal effective ampage for motor response (tibial or fibular) was recorded. Then, an extraneural injection of ropivacaine (20 ml of 4.75 mg ml\textsuperscript{-1}, by mixing an equal amount of 2 and 7.5 mg ml\textsuperscript{-1} solutions) was performed around the sciatic nerve in both groups. If necessary, the needle tip was repositioned to produce a circumferential spread of the LAs around the nerve. All
blocks were performed or supervised by the authors and assessed by a blinded investigator.

Surgery was performed under general anaesthesia (using propofol, sufentanil, cisatracurium, and sevoflurane as appropriate) for all patients. A haemostatic tourniquet was placed at the middle of the thigh, inflated to a pressure of 130 kPa.

Efficacy measurements and variables
The sensory and motor block were assessed every 5 min for a 50 min period by an anaesthesiologist who was unaware of the patient’s status. The sensory block was assessed using a pin-prick test in the three peripheral sensory distributions of the sciatic nerve (tibial: plantar side of the foot, peroneal common: lateral cutaneous side of the calf, peroneal superficial: dorsal aspect of the foot) and two motor distributions (tibial, peroneal). The sensory block was determined using a rating scale, whereby 0, normal sensation; 1, blunted sensation; and 2, absence of sensation (anaesthesia). The motor block was tested: plantar flexion of the foot (tibial nerve), dorsiflexion of the foot (peroneal nerve). The rating scale for the motor block was as follows: 0, normal contraction; 1, reduced contraction (paresis); and 2, no contraction (paralysis). A complete motor or sensory block was considered when the responses in all nerve distributions had a score of 2. If the sensory block was not 2 in at least one of the sciatic areas, at the end of the 50 min assessment period, the sciatic block was considered incomplete and not analysed for resolution time. The onset time of the sensory block and the motor block was defined as the interval between time 0 (end of LA injection) and a complete block. Block resolution was defined as complete regression when both sensory and motor blocks in all distributions returned to baseline. The nurse or resident in charge of the patient tested the resolution of the sensory (pin-prick test) and the motor block (movement) every hour after surgery, until complete regression.

Patient’s characteristics (age, sex, BMI), surgery (type and duration), diabetes duration (yr), associated diseases (nephropathy, retinopathy, cardiac ischaemic disease, arterial hypertension, foot ulcer), and oral therapy were recorded (nephropathy, retinopathy, cardiac ischaemic disease, arteriel hypertension, foot ulcer), and oral therapy were recorded before surgery. Blood samples were obtained to measure the following serum concentrations: sodium (mM), potassium (mM), haemoglobin A1c (%), creatinine (μmol litre−1), and glycaemia (g litre−1).

The incidence of acute nerve injury was evaluated at 48 h (paraesthesia, sensory or motor alteration when compared with preoperative assessment) by authors. A nerve evaluation was recorded during the surgical follow-up visits at 45 days by the surgeon for motor and sensory alteration. Sensory evaluation at 45 days was only performed with size 5.07 Semmes-Weinstein 10 g.

Endpoints
The main endpoint was the time (h) to complete the resolution of sensory block (pin-prick test). Secondary endpoints were sensory and motor onset times (min) for the sciatic block and complete block resolution (h). Sensory onset time was defined as the time elapsed from the end of injection for the sciatic nerve block to the complete sensory block (pin-prick).

Statistical analysis
According to animal studies, we aimed to detect a 25% difference (h) for the sensory recovery time between groups. On the basis of a previous study, we calculated that we would need to include at least 20 patients per group to detect a significant difference between the two groups (α risk=0.05, β risk=0.20) and thus decided to include 23 patients in the diabetic group (10–20% of block failure expected). Because the frequency of surgery in patients with type 2 diabetes is less than in non-diabetic patients, non-diabetic patients were continuously included in the control group until the appropriate number of diabetic patients was reached, without any interim analysis.

Data are expressed as mean (so) or median and 25–75th inter-quartiles or percentage and 95% confidence interval (CI). Comparison between the two groups was performed using Student’s t-test, the Mann–Whitney U-test, and the Fisher exact method, as appropriate. Onset and recovery times were compared using the log-rank test (Kaplan–Meier diagram). Correlation was performed between the duration of the block and age (Pearson’s correlation coefficient). All P-values were two-tailed and a P-value of <0.05 was required to exclude the null hypothesis. Statistical analysis was conducted using SAS (release 8.0; SAS Institute, Cary, CA, USA).

Results
Over the study period, 223 patients had a sciatic nerve block (Fig. 1), and 23 were analysed in the diabetic group and 49 in the non-diabetic group. The main exclusion criteria in the diabetic group were insulin therapy (n=25), creatinine clearance <50 ml min−1 (n=17), and age >80 (n=24). Characteristics of patients are reported in Table 1. The mean age was not significantly different between the groups and there was no significant correlation between duration of block and age (P=0.11, r2=0.04). ASA status was greater in the diabetic group compared with the control group. Creatinine concentration and haemoglobin A1c were significantly increased in the diabetic group (Table 1). The maximum value of haemoglobin A1c was 7.8% in the diabetic group. Preoperative evaluations were comparable with 5.07 (10 g) monofilaments, but diabetic patients presented lower scores with 2, 0.4, and 0.07 g monofilaments (Table 1).

The minimal intensity of stimulation was not significantly different in the diabetic group vs control group (Table 2). However, there was a significant difference in the number of patients in whom a motor response was unable to be elicited in the diabetic group (Table 2). The sciatic nerve block was complete for all patients 45 min after ropivacaine administration. The onset time of complete sensory and motor block was comparable between the two groups (Table 1). The duration of the sciatic nerve sensory block
was significantly prolonged in the diabetic group (Table 2 and Fig. 2). The sensory block duration (median, 25–75th) was 21 (16–24) h for the diabetic group vs 17 (14–20) h for the control group ($P<0.01$), and the motor block duration was 16 (10–20) h for the diabetic group vs 12 (9–16) h for the control group ($P<0.01$). No complication was noted after 4 weeks (including paraesthesia).

**Discussion**

This prospective study demonstrated that type 2 diabetic patients compared with non-diabetic patients presented an increased recovery duration after a sciatic nerve block performed with a long-acting LA agent.

Although there is a lack of human studies, diabetic animal studies tend to show peripheral nerve block onset time reduction and delayed recovery. In streptozotocin-induced diabetic rats, Kalichman and Califet performed a sciatic nerve block with 1% procaine and observed a faster and more complete motor block in the diabetic group. In contrast, 1% lidocaine exhibited a similar potency and time course for the motor block in streptozotocin-diabetic rats compared with saline-treated animals. Recently, Kroin and colleagues administered 1% lidocaine alone, 1% lidocaine with 5 µg ml$^{-1}$ epinephrine, 1% lidocaine with 7.5 µg ml$^{-1}$ clonidine, or 0.5% ropivacaine alone, and a longer sciatic nerve sensory block was observed for all solutions in diabetic rats. With 0.5% ropivacaine (0.25 ml into the soft tissue next to the sciatic nerve), the duration of the sciatic sensory block was significantly prolonged in diabetic rats [146 (8) vs 114 (4) min]. Thus, for Kalichman and Califet and Kroin and colleagues, animal diabetic models demonstrated a prolonged recovery from the block, but it still remained to be demonstrated whether these empirical findings have a clinical parallel and impact. This study provides data showing a prolonged effect of LAs in diabetic compared with non-diabetic patients, using ropivacaine 4.75 mg ml$^{-1}$ for the sciatic nerve block (20 ml). We demonstrate that not only the median sensory block duration (3–4 h) but also the extreme range (40 h) is increased in diabetic patients. On

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**Fig 1** Flow diagram of the study.
the other hand, none of the non-diabetic patients presented a sensory block after 25 h (Fig. 2). Similarly, in a healthy young population [53 (10) yr], Casati and colleagues showed that the sciatic block duration was 13 (11–14) h with 5 mg ml$^{-1}$ ropivacaine, with no delay in recovery from the block after 1 day. In our study, the duration of sensory blocks in the control group was longer than those previously reported by Casati and colleagues. This discrepancy is probably explained by the older age of our patients because conduction velocities, number of large diameter fibres, and peripheral nerve (Na$^+$, K$^+$) ATPase decrease during ageing. We arbitrarily chose a range: 50–80 yr to reduce the influence of age. A possible bias related to age can be ruled out since the mean age was not significantly different between the two groups (Table 1) and no significant correlation between age and duration of block was noted within this age range. Several mechanisms may account for the increased sensitivity of diabetic patients to LA agents. This clinical study was not designed to answer this fundamental question. In diabetic rats, Kroin and colleagues demonstrated that the total percentage of nerve fibre degeneration was correlated to the duration of the sensory ($R=0.64$, $P=0.004$) and the motor block ($R=0.60$, $P=0.008$) of the sciatic nerve. These authors suggested that diabetes-induced nerve injury was the main mechanism involved in the prolonged duration of block. However, other mechanisms have to be investigated because neuropathy in diabetic patients is also associated with microvascular dysfunction (endothelial alteration), which can reduce LA absorption and finally increase the block duration.

| Table 1 Characteristics of the patients. Data are mean (sd), median (25th–75th), or number (%). Age was expressed with mean and range. NA, not applicable. *P<0.05 vs the control group |
|-----------------|-----------------|
| **Diabetic group (n=23)** | **Control group (n=49)** |
| Age (yr) | 69 (66–78) | 70 (64–76) |
| Height (cm) | 165 (9) | 168 (9) |
| Body mass index (kg m$^{-2}$) | 31 (5) | 31 (6) |
| Men/women | 10 (44%)/13 (56%) | 22 (45%)/27 (55%) |
| ASA status (I/II/III/IV) | 1/4/18/1* | 9/32/8/0 |
| Diabetic duration (yr) | 7 (3–10) | NA |
| Chronic treatment | | |
| Insulin use | 0 | NA |
| Oral hypoglycaemic agent | 23 (100%) | 0 |
| ACE inhibitor | 12 (52%) | 28 (57%) |
| History | | |
| Cardiac disease | 4 (17%) | 7 (14%) |
| Retinopathy | 0 | 0 |
| Foot ulcer | 0 | 0 |
| Nephropathy | 1 (4%) | 0 |
| Monofilament score | | |
| 10 g (size 5.07) | 6 (1) | 6.5 (1) |
| 4 g (size 4.56) | 5 (2) | 6 (2) |
| 2 g (size 4.31) | 4 (3)* | 5.5 (2) |
| 0.4 g (size 3.61) | 3 (2)* | 5.5 (3) |
| 0.07 g (size 2.83) | 2 (2)* | 4 (2) |
| Surgical procedure | | |
| Duration (min) | 120 (100–132) | 115 (100–130) |
| Side (right/left) | 11 (48%)/12 (52%) | 22 (45%)/27 (55%) |
| Tourniquet use | 23 (100%) | 48 (96%) |
| Tourniquet duration (min) | 90 (60–125) | 87 (60–122) |
| Type of surgery | | |
| Foot | 12 (55%) | 23 (46%) |
| Ankle | 4 (15%) | 10 (22%) |
| Knee | 7 (30) | 16 (32%) |
| Biological variables | | |
| Serum creatinine (μM) | 95 (27)* | 71 (15) |
| Creatinine clearance (ml min$^{-1}$) | 66 (18)* | 92 (24) |
| Haemoglobin A1c (%) | 6.7 (1)* | 5.1 (0.5) |
Further studies analysing the effects of various concentration of LA on diabetic nerve fibres are needed to find the best concentration (ratio benefit–risk, i.e. nerve injury toxicity), since the causative link between nerve toxicity and LA concentration is still not proved.

In a retrospective investigation of patients who received ropivacaine (3 mg kg\(^{-1}\)) for a supraclavicular block, Gebhard and colleagues reported that diabetes mellitus was associated with a higher success rate. To explain this difference, these authors speculate ‘that nerve fibres of diabetic humans may have a higher sensitivity to local anaesthetic solutions, as has been demonstrated in nerve fibres of diabetic rats’. This finding is not supported by our results since no significant difference in block success and its onset time was observed. It could be argued that we performed all blocks under ultrasound control. Ultrasound control reduced block failure due to the nerve stimulation technique alone in diabetic patients. Table 2 shows that no motor response was elicited in several diabetic patients, while the needle tip under ultrasound approach was in close proximity to the nerve. Moreover, in our study, patients were selected for incipient nerve injury with haemoglobin A1c <8%, while Gebhard and colleagues included patients with type 1 or 2 diabetes ‘with neither the presence nor the degree of a preexisting diabetic neuropathy documented’.

Various clinical methods are used to define and examine diabetic nerve neuropathy. The main difficulty of this prospective study was to select a homogeneous population of diabetic type 2 patients because nerve injury is not correlated with duration of diabetes and because ageing influences block duration. We decided to include diabetic patients using a 10 g monofilament test to evaluate the neuropathy. First, this method allowed us to exclude patients who were not able to perform a pin-prick test (no perception to 10 g monofilament). Secondly, except electrodiagnostic studies, 10 g monofilament testing followed by 2, 4, and 8 g tests has been described as the most sensitive and specific test for the clinical evaluation of diabetic neuropathy. In our study, both groups of patients presented similar tests with 10 and 4 g monofilament testing, indicating that we successfully excluded patients with major nerve injury. Although all patients in the diabetic group had normal clinical sensory assessment with a positive 10 g monofilament test, the sciatric nerve block sensory duration was greater in the diabetic group when compared with the control group. This confirms that clinical tests are unable to detect incipient nerve injury that induced the prolonged nerve block duration in this study. The main mechanism of clinical nerve injury is hyperglycaemia toxicity. Because our sample size of diabetic patients was small, we were not able to assess the possible effect of glycaemic control. Further studies with a larger diabetic population are needed in order to answer this question.

Some limitations in our study should be considered. First, we did not perform an electrodiagnostic evaluation in our patients, which is considered as the reference method for the assessment of neuropathy. However, in the present...
study, patients had comparable age and were selected according to the absence of clinical signs and a positive 10 g monofilament test. In this situation, the use of the electrodagnostic method for nerve injury evaluation has been recently recommended.10 Secondly, we used ropivacaine and our findings have to be confirmed using other LA agents, particularly lidocaine, because animal studies demonstrated conflicting results with 1% lidocaine when compared with 2% and 4%. Lastly, we studied only diabetic patients with incipient nerve injury. These patients had no renal dysfunction (creatinine clearance >50 ml min\(^{-1}\)). Thus, the application of our findings to patients suffering from clinical nerve injury, distal clinical ulceration, or renal dysfunction that are correlated with major nerve damage is limited.1

In conclusion, in this prospective study that investigated the nerve block duration with ropivacaine, we demonstrated that sensory and motor block durations were significantly prolonged in type 2 diabetic patients. The prolonged block identified in the current study did not translate into an increased risk of nerve injury due to a limited study population.

Declaration of interest
None declared.

Funding
This work was supported by institutional sources (Association Recherche En Anesthésie, Nimes, France, 01/2011).

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Handling editor: L. Colvin