

# Lumbar Plexus Block Using High-pressure Injection Leads to Contralateral and Epidural Spread

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**Background:** The main advantage of lumbar plexus block over neuraxial anesthesia is unilateral blockade; however, the relatively common occurrence of bilateral spread (up to 27%) makes this advantage unpredictable. The authors hypothesized that high injection pressures during lumbar plexus block carry a higher risk of bilateral or neuraxial anesthesia.

**Methods:** Eighty patients undergoing knee arthroscopy (age 18–65 yr; American Society of Anesthesiologists physical status I or II) during a standard, nerve stimulator–guided lumbar plexus block using 35 ml mepivacaine, 1.5%, were scheduled to be studied. Patients were randomly assigned to receive either a low-pressure (< 15 psi) or a high-pressure (> 20 psi) injection, as assessed by an inline injection pressure monitor (BSmart®; Concert Medical LLC, Norwell, MA). The block success rate and the presence of bilateral sensory and/or motor blockade were assessed.

**Results:** An interim analysis was performed at n = 20 after an unexpectedly high number of patients had neuraxial spread, necessitating early termination of the study. Five of 10 patients (50%) in the high-pressure group had a neuraxial block with a dermatomal sensory level T10 or higher. In contrast, no patient in the low-pressure group (n = 10) had evidence of neuraxial spread. Moreover, 6 patients (60%) in the high-pressure group demonstrated bilateral sensory blockade in the femoral distribution, whereas no patient in the low-pressure group had evidence of a bilateral femoral block.

**Conclusions:** Injection of local anesthetic with high injection pressure (> 20 psi) during lumbar plexus block commonly results in unwanted bilateral blockade and is associated with high risk of neuraxial blockade.

LUMBAR plexus block (LPB) is a useful anesthetic technique.<sup>1</sup> The main advantages of LPB over neuraxial anesthesia are unilateral blockade, resulting in greater hemodynamic stability, an improved ability to ambulate, and absence of urinary retention.<sup>2</sup> The relatively common occurrence of epidural spread (up to 27% in adults and up to 92% in children depending on the approach) often limits the usefulness of LPB, particularly when long-acting local anesthetics are used.<sup>3–7</sup> Moreover, in a

prospective survey of major complications of regional anesthesia, Auroy *et al.*<sup>8</sup> reported cardiac and respiratory arrests associated with LPB. These patients were found to have a high dermatomal block before the arrest, strongly suggesting either epidural or spinal spread of local anesthetic as the underlying cause of these complications. In this study, we hypothesized that forceful injection with high injection pressures increases the risk of contralateral spread of local anesthetic or neuraxial anesthesia during LPB.

## Materials and Methods

### Study Subjects

The study protocol was approved by the St. Luke's and Roosevelt Hospitals Institutional Review Board, New York, New York. Any American Society of Anesthesiologists physical status I or II patient aged 18–65 yr and scheduled to undergo elective, outpatient knee arthroscopy was included. Patients were excluded if they had a contraindication to lumbar plexus block (*e.g.*, coagulopathy, history of spinal surgery, deformity), allergy to local anesthetic, or a body mass index greater than 35 kg/m<sup>2</sup>. After obtaining informed consent, patients were randomly assigned using a method of sealed envelopes to receive LPB using either a low-injection-pressure (LIP group; injection pressure < 15 psi) or a high-injection-pressure (HIP group; injection pressure > 20 psi) injection of local anesthetic. These pressure limits were selected based on injection pressures attained by anesthesiologists during nerve block simulation in a previous study.<sup>9</sup> A commercially available in-line injection pressure manometer that is routinely used in our practice for nerve blocks (BSmart®; Concert Medical LLC, Norwell, MA) allowed for convenient monitoring due to its colored markings indicating the selected pressure ranges (*i.e.*, < 15 psi and > 20 psi).

### Block Procedure

All blocks were performed in the operating room during monitoring with electrocardiography, noninvasive blood pressure, pulse oximetry, and capnography (qualitative). Patients were placed in the lateral decubitus position with the operative side up, and intravenous sedation (0.05–0.08 µg/kg midazolam) and analgesia (0.5–2 µg/kg fentanyl or 5–15 µg/kg alfentanil) were administered until patients were comfortable during the block procedure while maintaining a meaningful patient

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Received from the Department of Anesthesiology, St. Luke's-Roosevelt Hospital Center, New York, New York. Submitted for publication March 3, 2008. Accepted for publication June 13, 2008. Support was provided solely from institutional and/or departmental sources.

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contact (score of 4 on the Sedation–Agitation Scale).<sup>10</sup> After sterile skin preparation and local infiltration, a 100-mm-long, 21-gauge stimulating needle (Stimuplex®; B. Braun Medical Inc., Bethlehem, PA) attached to an in-line injection pressure manometer (Bsmart®) was inserted perpendicular to the coronal plane at a point 4 cm lateral to the midline on the intercrystal line.<sup>1</sup> A nerve stimulator (Tracer II®; LifeTech Inc., Stafford, TX) set to deliver 1.5 mA (0.1 ms, 2 Hz) was used to elicit a quadriceps muscle motor response as a sign of successful localization of the lumbar plexus. The current was then decreased until twitches were observed between 0.5 and 1.0 mA. After a negative aspiration for blood test, an initial bolus of 1 ml local anesthetic was administered during monitoring of the opening injection pressure to decrease the risk of intraneural injection (< 20 psi) before the study was continued.<sup>11</sup> The remainder of the local anesthetic was then injected in 5-ml aliquots, with intermittent aspiration, using the appropriate amount of injection force to maintain either a low injection pressure (< 15 psi) or a high injection pressure (> 20 psi) throughout the injection. In the latter case, the pressure was applied to exceed the 20 psi threshold. A total of 35 ml mepivacaine, 1.5%, with epinephrine (5 µg/ml) was used in all patients.

Patients were then placed supine, and surgery was allowed to commence. Sedation was maintained throughout the procedure using a propofol infusion (25–50 µg · kg<sup>-1</sup> · min<sup>-1</sup>). Additional alfentanil or fentanyl was permitted to be administered at the discretion of the anesthesiologist to manage intraoperative pain not covered by the block.

### Postoperative Evaluation

In the postanesthesia care unit, patients were evaluated by the research team blinded to the injection pressure information. Sensory testing was performed bilaterally in the femoral (anterior thigh), obturator (medial thigh), and sciatic (posterolateral calf, plantar surface of foot) nerve distributions, as well as for dermatomal levels on the abdomen and thorax to determine the cephalad extent of the block, if any. When a pinprick stimulus was applied to the skin, patients were asked to rate the sensation as “sharp” (score of 2), “dull” (score of 1), or “unable to feel the stimulus” (score of 0). Application of the pinprick stimulus to both the ipsilateral lower and upper limb (in case of bilateral spread) was used as a control. Motor blockade was evaluated by assessing the strength of knee extension (femoral nerve), thigh adduction (obturator nerve), and plantar flexion and dorsiflexion of the ankle (sciatic nerve). Patients were asked to perform the movement against resistance, and the power was scored as 0 = complete absence of power, 1 = moderate weakness, 2 = mild weakness, or 3 = full power. These assessments were repeated every 30 min until the sensory and motor block fully resolved. Neuraxial block was defined as the presence of a sensory

block in any dermatome at or cephalad to T11, as measured by pinprick stimulus.

Electrocardiography, noninvasive blood pressure monitoring, pulse oximetry, and respiratory rate monitoring continued in the postanesthesia care unit every 30 min until discharge to the phase 2 recovery area. If patients were discharged home with the sensory block still in effect, a follow-up phone call was made the next day to determine the time of block resolution, defined as time of first pain or discomfort at the surgical site.

### Statistical Analysis

Based on the reported incidence of epidural block after LPB, we predicted that an injection with high pressure would have a 30% chance of contralateral spread and that a low-pressure injection would result in an incidence of less than 10%. A power analysis indicated that a sample size of 80 patients per group would be required to detect a reduction of 20% in incidence of contralateral spread with a significance level of 0.05, a power of 0.8, and an estimated rejection rate of approximately 10%. Data were analyzed using SPSS for Windows, version 11.0.1 (SPSS, Chicago, IL), and are presented as mean, range, SD, and 95% confidence interval where applicable. Continuous variables were compared using the Student *t* test, whereas dichotomous variables were treated with a Fisher exact test.

### Results

Patient demographic and intraoperative data are shown in table 1. There were no significant differences between patient groups with respect to age, sex, height, weight, or body mass index. The duration of surgery, doses of intraoperative sedation, and requirement for opioids for each group were similar. No patient reported paresthesiae during the injection. With the exception of one patient (subject 1) who required two doses of 10 mg intravenous ephedrine to treat arterial hypotension, all patients were hemodynamically stable, with no significant decrease in heart rate or systolic blood pressure (defined as a 30% change from baseline).

After enrollment of 14 patients, it became apparent to the blinded investigators that the incidence of bilateral blockade and epidural blockade was higher than expected. Five of 14 enrolled patients (36%) developed a bilateral femoral block, as well as a neuraxial block at level T11 or higher. One of these patients had a block extending to the T4 level. After accounting for possible bias due to the small sample size at the time, we suspected that we had largely underestimated the risk of contralateral and neuraxial spread after a high-injection pressure LPB. Because of the inherent risk of a serious cardiovascular or respiratory adverse event associated with a high epidural or neuraxial block associated with LPB,<sup>8</sup> a decision was made to perform an interim analysis

**Table 1. Patient Demographics and Intraoperative Data**

	High-pressure Injection, n = 10, Mean $\pm$ SD (Median, Range)	Low-pressure Injection, n = 10, Mean $\pm$ SD (Median, Range)	P Value
Age, yr	45.1 $\pm$ 10.1 (50, 29–56)	53.6 $\pm$ 14.3 (56, 22–71)	0.14
Sex, F/M	4/6	6/4	0.65
Body mass index, kg/m <sup>2</sup>	29.0 $\pm$ 4.3 (28.7, 21.6–34.9)	27.2 $\pm$ 4.0 (25.3, 22.7–34.3)	0.66
Height, cm	166.1 $\pm$ 16.7 (165.1, 142.2–193.0)	170.7 $\pm$ 10.8 (167.6, 154.9–193.0)	0.48
Weight, kg	79.0 $\pm$ 17.4 (81.8, 51.8–95.1)	81.3 $\pm$ 15.4 (82.2, 62.7–99.0)	0.76
Duration of surgery, min	54.6 $\pm$ 18.2 (50, 36–90)	55.0 $\pm$ 16.3 (53, 30–80)	0.96
Midazolam dose, mg	5.6 $\pm$ 1.3 (6.0, 4.0–8.0)	4.3 $\pm$ 1.9 (4.0, 2.0–8.0)	0.25
Propofol dose, mg	64.7 $\pm$ 74.6 (62.3, 0–238.1)	87.4 $\pm$ 71.9 (65.1, 0–195.5)	0.92
Fentanyl dose, $\mu$ g	6 patients received propofol 35.0 $\pm$ 47.4 (0, 0–100)	8 patients received propofol 60 $\pm$ 93.7 (0, 0–250)	0.06
Alfentanil dose, $\mu$ g	4 patients received fentanyl 700.0 $\pm$ 483.1 (1,000, 0–1,000)	5 patients received fentanyl 275 $\pm$ 415.8 (0, 0–1,000)	0.66
Fentanyl equivalent, $\mu$ g	7 patients received alfentanil 128.3 $\pm$ 45.8 (133.3, 50.0–233.3)	4 patients received alfentanil 120.8 $\pm$ 74.9 (83.3, 0–250.0)	0.80

Fentanyl equivalent = [(total alfentanil dose/7.5) + total fentanyl dose].

at n = 20 patients. Each group had 10 patients enrolled at the time of analysis; postoperative assessment data are presented in tables 2–4.

### Sensory Blockade

Five patients in the HIP group demonstrated a neuraxial block with a sensory level at or above T11. In contrast, no patient in the LIP group had evidence of neuraxial spread. Moreover, 6 patients (60%) in the HIP group had evidence of bilateral sensory blockade in the femoral distribution, whereas no patient in the LIP group had evidence of a bilateral femoral block. Ipsilateral (block side) sciatic sensory blockade was present in 5 patients (50%) in the HIP group and in none (0%) of the LIP group. Of these 5 patients, 3 (30%) in the HIP group also had contralateral sciatic sensory block; all 3 of these had bilateral sensory block extending caudally to the S2 dermatome.

### Motor Blockade

In the HIP group, 10 (100%) patients had motor blockade of the femoral and obturator nerves on the ipsilateral side, and 5 (50%) on the contralateral side. In addition, some degree of sciatic motor block was present bilaterally in 5 of the HIP patients (50%). In contrast, only 1 patient of 10 in the LIP group exhibited contralateral motor blockade of

any nerve branch. This patient (subject 15) had a failed block, as defined by the absence of any sensory block of the ipsilateral femoral, obturator, or lateral femoral cutaneous nerves. On postoperative examination, she was found to have intact sensation bilaterally, but displayed some decreased motor strength in both lower limbs. However, her weakness (which was also present in her upper limbs) was likely related to the excess sedation and analgesia required as a result of the failed block. This apparent “motor weakness” resolved within 30 min of arrival to the recovery room.

All patients were successfully discharged home without sequelae. Based on the results of the interim data analysis, a decision was made to terminate the study early, because the research team thought that the continuation of the study could be associated with an unanticipated risk of high neuraxial blockade.

### Discussion

Under the conditions of our study, high-pressure injection (> 20 psi) of 35 ml mepivacaine, 1.5%, during LPB was associated with a 60% incidence of bilateral femoral nerve block, whereas no patient in the low-pressure injection group (< 15 psi) developed a bilateral femoral

**Table 2. Postoperative Assessment**

	High-pressure Injection, n = 10, Mean $\pm$ SD (Median, Range)	Low-pressure Injection, n = 10, Mean $\pm$ SD (Median, Range)	P Value
Duration of analgesia, min	149.6 $\pm$ 42.4 (140, 100–230)	146.4 $\pm$ 36.4 (140, 90–200)	0.86
Home discharge time, min	110.5 $\pm$ 47.9 (105, 60–210)	82.1 $\pm$ 26.1 (75, 50–126)	0.12
Incidence of bilateral femoral blockade (95% exact CI)	60% (6/10) (26.2–87.8%)	0% (0/10) (0.00–30.9%)	0.01
Incidence of neuraxial blockade (95% exact CI)	50% (5/10) (18.7–81.3%)	0% (0/10) (0.00–30.9%)	0.03
Incidence of ipsilateral sciatic nerve block (95% exact CI)	50% (5/10) (18.7–81.3%)	0% (0/10) (0.00–30.9%)	0.03

Neuraxial blockade = sensory block in any dermatome at or cephalad to T11, by pinprick stimulus.

**Table 3. Sensory and Motor Block Data for High-pressure Injection Group**

Patient	Ipsilateral Side								Contralateral Side							
	Sensory					Motor			Sensory					Motor		
	Fem	Obt	LFC	Sci	Range*	Fem	Obt	Sci	Fem	Obt	LFC	Sci	Range*	Fem	Obt	Sci
1	0	0	0	0	T6-S2	0	0	0	0	1	0	2	T4-L2	0	1	2
2	0	0	0	0	L4-S2	0	0	3	2	2	2	2	N	3	3	3
3	0	0	0	2	L2-L4	0	0	3	2	2	2	2	N	3	3	3
5	0	0	0	2	L2-L4	0	0	3	2	2	2	2	N	3	3	3
7	0	0	0	2	L2-L4	0	0	3	2	2	2	2	N	3	3	3
9	0	0	0	0	T10-S2	0	0	0	0	0	0	0	T8-S2	0	0	0
11	0	0	0	2	T10-L4	0	0	2	0	0	0	2	T12-L4	0	0	2
12	0	0	0	0	T9-S2	0	0	0	0	0	0	0	T9-S2	0	0	0
13	0	0	0	0	T11-S2	0	0	0	0	0	0	0	T12-S2	0	0	0
16	0	0	0	2	T12-L4	0	0	3	0	1	0	2	T12-L4	1	2	3

Sensory block: 0 = unable to feel stimulus; 1 = dull; 2 = sharp. Motor block: 0 = no power; 1 = moderate weakness; 2 = mild weakness; 3 = full power.

\* Sensory dermatomes tested individually with pinprick test.

Fem = femoral nerve; LFC = lateral femoral cutaneous nerve; N = no sensory block; Obt = obturator nerve; Sci = sciatic nerve.

block. Moreover, HIP resulted in a 50% incidence of neuraxial block extending to T11 or above. These data suggest that the force of injection plays an important role in the spread of the local anesthetic and the risk of high dermatomal block during application of LPB.

Epidural spread after the posterior approach to the LPB has been a known side effect since the description of the technique by Winnie *et al.*<sup>12</sup> in 1974. The mechanism of unilateral or bilateral epidural blockade after LPB remains inadequately clarified, although several theories have been proposed. The paravertebral space in which the proximal lumbar plexus lies is contiguous with the epidural space *via* the intervertebral foramina, and contrast dye studies performed during LPB have shown that while injectate remains primarily within the body of the psoas muscle, it may also travel medially toward the neuraxis.<sup>13-15</sup> A forceful injection during LPB may drive the local anesthetic through the foramina toward the epidural space. Contralateral spread anterior

to the vertebral body has been described in thoracic paravertebral techniques and is another possibility.<sup>16</sup> In addition, a needle inserted or directed too medially may result in an injection into the epidural space itself.<sup>17</sup> The dural cuff surrounding the nerve extends beyond the intervertebral foramen, leading to a risk of spinal anesthesia should the injection be made within the epineurial cuff.<sup>18</sup> Spinal deformity may also have an effect on the central spread of local anesthetic in LPB, similar to the risk seen with neuraxial techniques.<sup>19</sup> Finally, although it would be logical that injection of larger volumes of local anesthetic could lead to greater risk of epidural spread, this has not been substantiated.<sup>7,13,20,21</sup>

In our study, three of the five sciatic blocks observed were bilateral, suggesting neuraxial spread as the mechanism. The remaining two sciatic blocks were only detected on the ipsilateral side; this suggests either limited epidural spread that affected only one

**Table 4. Sensory and Motor Block Data for Low-pressure Injection Group**

Patient	Ipsilateral Side								Contralateral Side							
	Sensory					Motor			Sensory					Motor		
	Fem	Obt	LFC	Sci	Range*	Fem	Obt	Sci	Fem	Obt	LFC	Sci	Range*	Fem	Obt	Sci
4	0	0	0	2	T12-L4	0	0	3	2	2	2	2	N	3	3	3
6	0	0	0	2	L2-L4	0	0	3	2	2	2	2	N	3	3	3
8	0	0	0	2	L2-L4	0	0	3	2	2	2	2	N	3	3	3
10	0	1	1	2	L3-L4	0	1	3	2	2	2	2	N	3	3	3
14	0	0	0	2	L2-L4	0	0	3	2	2	2	2	N	3	3	3
15	2	2	2	2	N	1	2	2	2	2	2	2	N	2	2	2
17	0	0	0	2	L1-L4	0	0	3	2	2	2	2	N	3	3	3
18	0	0	0	2	L1-L4	0	0	3	2	2	2	2	N	3	3	3
19	0	0	0	2	T12-L4	0	0	3	2	2	2	2	N	3	3	3
20	0	2	2	2	L3-L4	0	2	3	2	2	2	2	N	3	3	3

Sensory block: 0 = unable to feel stimulus; 1 = dull; 2 = sharp. Motor block: 0 = no power; 1 = moderate weakness; 2 = mild weakness; 3 = full power.

\* Sensory dermatomes tested individually with pinprick test.

Fem = femoral nerve; LFC = lateral femoral cutaneous nerve; N = no sensory block; Obt = obturator nerve; Sci = sciatic nerve.



side, or paravertebral spread of the local anesthetic caudally.

In previous efforts to study the risk of neuraxial spread of local anesthetic after LPB, much effort has been expended on the anatomical variables and position of the needle tip. For example, studies using the Chayen approach<sup>20</sup> in adults found bilateral spread in 1.5–5% of subjects.<sup>21,22</sup> These results are similar to those in studies using the technique described by Capdevila *et al.* (0–1.8%).<sup>3,23</sup> In contrast, the Dekrey approach at L3 has an incidence of epidural spread of 16–27%,<sup>6,13</sup> whereas the Winnie technique seems to result in an intermediate frequency of bilateral spread, with reports of 8.8–10.7%.<sup>5,24</sup> Capdevila *et al.* postulated that their more medial insertion point led to less need for medial angulation of the needle to contact the plexus compared with Winnie's approach, and therefore avoided coming close to the central neuraxis. Chayen's approach is significantly more lateral and distal than any other approach and is associated with a low incidence of bilateral spread, which may fit with this hypothesis. On the other hand, the L3 Dekrey technique is more cephalad and closer to the midline (3 cm lateral), which may predispose extension of injectate into the intervertebral foramina. However, without verifying the ultimate needle position with radiographic imaging, the long needle path during LPB (typically 7–9 cm) makes it difficult to determine the exact needle tip position. Consequently, the clinical relevance of these relatively minor technique modifications affecting the needle orientation and/or entry site is questionable due to the inherent lack of reproducibility.

In contrast to the previous research that focused on anatomical landmarks, our study suggests that the force of injection and, more specifically, high injection pressure during LPB are associated with a significant risk of block extension to ipsilateral sciatic nerve, contralateral femoral nerve, or thoracic epidural space. In the absence of injection pressure monitoring, it is possible that injection pressures varied among previous research groups. This, in turn, may have contributed to the disparity in the published data regardless of the approach used, because subjective estimation of the force of injection is a poor predictor of actual injection force during injection of local anesthetic.<sup>9</sup> A simple and logical explanation for our observation could be that a forcefully injected local anesthetic has a greater likelihood of dissecting through the tissue compartments within the psoas compartment alongside the paths of least resistance, and hence potentially toward the intervertebral foramina. Contrast studies would be required to determine the exact mechanisms of the local anesthetic spread with a greater degree of certainty.

One theoretical limitation of our study's findings is the use of a large volume (35 ml) of local anesthetic. It is possible that the use of a lower volume could have

resulted in a lower risk of bilateral and/or cephalad spread. However, for surgical anesthesia, complete blockade of the lumbar plexus is volume dependent.<sup>1</sup> Moreover, the higher risk of neuraxial or contralateral spread of local anesthetic with larger volumes of local anesthetic in LPB has not been supported by the literature.<sup>17</sup> Finally, none of the *low pressure* injections resulted in bilateral blockade.

The sensory block to a T4 spinal level in one of the patients is especially concerning, because such blocks may be associated with unexpected but significant sympathetic block after LPB, potentially leading to hemodynamic or respiratory arrest.<sup>8</sup> The patients with neuraxial blocks in our study did not have significant hemodynamic or respiratory consequences; however, our study was not designed nor powered to detect the true incidence and severity of cardiovascular instability. In contrast, larger-scale studies suggest that a high level of neuraxial sensory block after LPB may reflect the development of total spinal anesthesia. As an example, Auroy *et al.*<sup>8</sup> reported a case of a T2 block and bilateral mydriasis preceding irreversible cardiac arrest after performance of LPB. Although this patient most likely had an intrathecal injection of a large dose of local anesthetic leading to a total spinal anesthesia, hemodynamic collapse and cardiac arrest have also been reported after accidental high thoracic levels after lumbar epidural anesthesia.<sup>25</sup>

Commercially available pressure monitors may not be readily available in all centers. However, a recent *in vitro* study suggested that a compressed air injection technique based on Boyle's law could also be used to limit the injection pressure during peripheral nerve blockade.<sup>26</sup>

In summary, under the conditions of our study, high injection pressures (> 20 psi) during LPB led to a high risk of neuraxial spread and bilateral lumbar and sacral plexus blockade. Future studies are indicated to determine whether maintaining low pressures with injection pressure monitoring may decrease the complication rate of LPB related to the spread of local anesthetic injectate.

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