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Transient Neurologic Symptoms after Spinal Anesthesia

An Epidemiologic Study of 1,863 Patients

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Background: Recent evidence suggests that transient neurologic symptoms commonly follow lidocaine spinal anesthesia. However, information concerning factors that affect their occurrence is limited. Accordingly, to evaluate many potential risk factors, the authors undertook a prospective, multicenter, epidemiologic study.

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Methods: On a voluntary basis, anesthetists at 15 participating centers forwarded a data sheet on patients who had spinal anesthesia to a research nurse blinded to the details of anesthesia and surgery. A subset was randomly selected for follow-up. The pressure of transient neurologic symptoms, defined as leg or buttock pain, was the principal outcome variable. Logistic regression was used to control for potential confounders, and adjusted odds ratios and confidence intervals were used to estimate relative risk.

Results: During a 14-month period, 1,863 patients were studied, of whom 47% received lidocaine, 40% bupivacaine, and 13% tetracaine. Patients given lidocaine were at higher risk for symptoms compared with those receiving bupivacaine (relative risk, 5.1; 95% CI, 2.5 to 10.2) or tetracaine (relative risk, 3.2; 95% CI, 1.04 to 9.84). For patients who received lidocaine, the relative risk of transient neurologic symptoms was 2.6 (95% CI, 1.5 to 4.5) with the lithotomy position compared with other positions, 3.6 (95% CI, 1.9 to 6.8), for outpatients compared with inpatients, and 1.6 (95% CI, 1 to 2.5) for obese (body mass index >30) compared with nonobese patients.

Conclusions: These results indicate that transient neurologic symptoms commonly follow lidocaine spinal anesthesia but are relatively uncommon with bupivacaine or tetracaine. The data identify lithotomy position and outpatient status as important risk factors in patients who receive lidocaine. Among other factors postulated to increase risk, obesity had an effect of borderline statistical significance, whereas age, sex, history of back pain, needle type, and lidocaine dose and concentration failed to affect risk. (Key words: Anesthetic technique; bupivacaine; lidocaine; local anesthetic; neurologic complications; tetracaine.)

MAJOR neurologic injury after spinal anesthesia is exceedingly rare.¹⁻³ However, recent reports of permanent deficits after central neuraxial blockade have renewed concern about the potential for toxic effects with local anesthetics injected into the intrathecal space.⁴⁻⁸ Schneider *et al.*⁹ subsequently proposed that pain, dysesthesia, or both in the buttocks or lower extremities after uncomplicated lidocaine spinal anesthesia might also result from a direct toxic effect of the local anesthetic. A follow-up prospective study performed by

Hampl *et al.*¹⁰ at the same institution documented that such symptoms commonly follow spinal anesthesia with lidocaine but occur infrequently with bupivacaine. More recent studies have confirmed a higher incidence of symptoms in patients who receive lidocaine compared with bupivacaine.¹¹⁻¹³ Data from these studies also indicate that the concentration of glucose,^{11,13} the osmolarity of the anesthetic solution,¹¹ and the injected concentration of lidocaine^{13,14} do not affect the incidence of symptoms, whereas the role of patient positioning and the effect of vasoconstrictors remain poorly defined.^{13,15}

Although controlled studies are of great value, analysis is generally limited to a single variable. Large-scale epidemiologic studies are necessary to identify factors that warrant study in controlled trials and to investigate factors that are not readily manipulated. Observational studies also permit investigators to evaluate elements of current practice that they may be hesitant to incorporate into a controlled study. Accordingly, we undertook a prospective, multicenter, epidemiologic study of spinal anesthesia in a large health maintenance organization to capture the wide diversity of current clinical practice and to obtain a sample size with sufficient statistical power to analyze many potential risk factors for leg or buttock pain after spinal anesthesia. In addition, we wanted to define further the clinical significance of these symptoms by examining their duration and severity, and their association with other neurologic symptoms after spinal anesthesia. Our analysis focused on pain and dysesthesia because of their reported high prevalence,¹⁰⁻¹⁴ their potential link to more significant neurologic complications, and our own clinical experience suggesting that this controversial and poorly understood postoperative phenomenon may significantly affect patient satisfaction with spinal anesthesia.

Methods

After receiving institutional review board approval and doing a power analysis and a pilot project to develop an efficient postoperative follow-up protocol, we conducted a 14-month prospective study wherein all patients receiving spinal anesthesia at any of 15 medical centers in the Kaiser-Permanente Northern California regional hospital system were eligible for entry into the study.

All anesthesiologists administering spinal anesthesia were requested, on a voluntary basis, to enter patients into the study by completing a detailed data sheet for each pa-

tient undergoing spinal anesthesia. The data sheet solicited information about patient characteristics, technical aspects of the anesthetic administration, type of surgery, position during surgery, and clinical course of the anesthetic. No attempt was made to modify individual practice. Data sheets were deposited in a receptacle after completion of surgery and sent by facsimile within 24 h to the study center.

From the patients entered into the study, a subset was randomly selected and contacted by telephone for postoperative follow-up by a research nurse (M.C.J.) who was blinded to all information except patient name, medical record number, surgical procedure, and date of surgery. The appendix shows the items in the structured questionnaire used in the telephone interview to obtain detailed information about postoperative recovery, including neurologic symptoms, if present. The initial follow-up call was made within the first 6 postoperative days. All patients who reported unresolved neurologic symptoms at the time of the initial call were contacted again within 7-10 days. Further follow-up evaluation proceeded on a case-by-case basis until the problem resolved, the patient was unable or unwilling to be contacted again, or the primary physician requested no further follow-up calls by the research nurse. In the latter two cases, further clinical follow-up was obtained by chart review, direct communication with the patient's primary or consultant physician(s), or both. Patients manifesting neurologic symptoms other than pain or dysesthesia received additional care directed by the study coordinator at the patient's primary facility; this person was responsible for ensuring appropriate clinical follow-up and communicating pertinent information to the research nurse.

Transient neurologic symptoms (TNSs) were defined as pain or dysesthesia or both occurring in the legs or buttocks after recovery from spinal anesthesia, as previously defined by Hampl *et al.*¹⁴ In addition, pain was rated on a scale from 0-10 where 0 represented "no pain" and 10 was "the worst pain imaginable." The relative risk associated with lidocaine, bupivacaine, and tetracaine solutions was examined. Logistic regression was used to control for potential confounders and to identify risk factors for TNSs. Adjusted odds ratios and confidence intervals obtained from logistic regression were used to estimate relative risk. In addition, risk factors for TNSs after lidocaine spinal anesthesia were compared with a bupivacaine reference.

Four other postoperative neurologic outcomes were examined to assess their relative incidence after spinal

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anesthesia and any association with TNSs: lower extremity weakness, lower extremity numbness, lower extremity paresthesias, and urinary retention requiring catheterization. Any new, exacerbated, or newly recrudescing symptoms that persisted more than 24 h after surgery were included in the analysis.

Finally the incidence of postoperative orthostatic headache and postoperative nonorthostatic headache in the lidocaine, bupivacaine, and tetracaine groups were examined to provide a standard to compare the groups with respect to their tendency to report pain.

Results

During the study period from August 1, 1995 through September 30, 1996, data from 6,092 of approximately 8,400 patients undergoing spinal anesthesia at participating hospitals were submitted to the study center (73% capture rate). Of those submitted, 2,555 patients (42%) were randomly selected for follow-up, of whom 1,883 (74%) were successfully interviewed. Of the 672 patients who were not interviewed, 61 patients declined to participate and the rest could not be reached by telephone, were unable to participate because of language problems, or were too ill to participate. Fourteen of 1,883 patients were excluded from study because of leg or buttock pain preexisting in the immediate preoperative period, and an additional six patients were excluded because of incomplete data for one or more key variables on the submitted spinal anesthetic data sheet.

Among the 1,863 study participants, 47% received lidocaine, 40% bupivacaine, and 13% tetracaine. Compared with patients receiving bupivacaine and tetracaine, those given lidocaine had certain significantly different characteristics. They were more likely to be male, to have had surgery in the lithotomy position, to have undergone inguinal, rectal, or urologic surgery, and to have had outpatient surgery; and they were less likely to have had hip surgery (table 1).

Patients in the lidocaine group demonstrated a significantly higher risk of developing TNSs compared with patients receiving bupivacaine or tetracaine (table 2). After adjustment for age, sex, race, preexisting neurologic condition(s), body mass index, type of surgery, inpatient/outpatient status, and surgical position, the relative risk associated with lidocaine was 5.1 (95% CI, 2.5 to 10.2) compared with those receiving bupivacaine, and 3.2 (95% CI, 1.04 to 9.84) compared with those receiving tetracaine.

Table 1. Characteristics of the Study Population

	Lidocaine (N = 873)	Bupivacaine (N = 744)	Tetracaine (N = 246)
Age			
<60 yr	436 (49.9)	387 (52.0)	89 (36.2)
≥60 yr	437 (50.1)	357 (48.0)	157 (63.8)
Sex			
Male	510 (58.4)	307 (41.3)	125 (50.8)
Female	363 (41.6)	437 (58.7)	121 (49.2)
Race			
American Indian	0	3 (0.4)	0
Asian	55 (6.3)	45 (6.0)	7 (2.8)
Black	48 (5.5)	56 (7.5)	13 (5.3)
Hispanic	47 (5.4)	58 (7.8)	10 (4.1)
White	708 (81.1)	564 (75.8)	211 (85.8)
Other	5 (0.6)	4 (0.5)	1 (0.4)
Unknown	10 (1.1)	14 (1.9)	4 (1.6)
ASA Class			
1	144 (16.4)	93 (12.5)	27 (11.0)
2	496 (56.9)	466 (62.8)	142 (58.0)
3	222 (25.5)	177 (23.9)	7 (2.9)
4	11 (1.3)	6 (0.8)	5 (2.0)
Body mass index (BMI)			
≤30 kg/m ²	638 (73.1)	514 (69.1)	182 (74.0)
>30 kg/m ²	235 (26.9)	230 (30.9)	64 (26.0)
Inpatient/outpatient			
Outpatient	592 (67.8)	136 (18.3)	10 (4.1)
Inpatient	281 (32.2)	608 (81.7)	235 (95.5)
Surgical position			
Nonlithotomy	547 (62.7)	573 (77.0)	183 (74.4)
Lithotomy	326 (37.3)	171 (23.0)	63 (25.6)
Needle type			
Pencil point	402 (46.1)	313 (42.1)	102 (41.5)
Quincke	471 (54.0)	431 (57.9)	144 (58.5)
Technical difficulty (attempts at >1 interspace)			
No	782 (89.6)	635 (85.4)	216 (87.8)
Yes	91 (10.4)	109 (14.7)	30 (12.2)
Type of surgery			
Foot/ankle	77 (8.8)	67 (9.0)	23 (9.4)
Thigh/leg/knee	191 (21.9)	173 (23.3)	73 (29.7)
Inguinal	91 (10.4)	28 (3.8)	10 (4.1)
Rectal	86 (9.9)	12 (1.6)	1 (0.4)
Hip	23 (2.6)	91 (12.2)	68 (27.6)
Gynecological	62 (7.1)	51 (6.9)	17 (6.9)
Abdominal	66 (7.6)	188 (25.3)	6 (2.4)
Urologic	277 (31.7)	134 (18.0)	47 (19.1)
Other	0	0	1 (0.4)

Values are number (%).

Follow-up data on patients with leg or buttock pain revealed complete resolution of symptoms within 72 h for most patients in all three local anesthetic groups (table 3). Ninety-three percent of patients in the lidocaine group were pain free at 1 week.

Data for pain intensity were obtained from 93 of the

Table 2. Local Anesthetics and Transient Neurologic Symptoms

	Leg or Buttock Pain		OR* (95% CI)
	No [number (%)]	Yes [number (%)]	
Lidocaine (N = 873)	769 (88.1)	104 (11.9)	
Bupivacaine (N = 744)	734 (98.7)	10 (1.3)	5.1 (2.5–10.2), lidocaine vs. bupivacaine
Tetracaine (N = 246)	242 (98.4)	4 (1.6)	3.2 (1.04–9.84), lidocaine vs. tetracaine

OR = odds ratio; CI = confidence interval.
* Adjusted for age, sex, race, preexisting neurologic condition(s), body mass index, type of surgery, inpatient/outpatient status, and patient position.

118 patients experiencing buttock or leg pain; comparable data were not available in the remaining 25 cases, because this question was added to the interview protocol after the study began. Of the patients in the lidocaine group who experienced pain, 30.1% rated their pain as 8 or greater on a scale from 0 to 10, whereas 48.2% rated it in the intermediate range of 4 to 7 (table 3). Only 1 of 8 patients in the bupivacaine group rated their pain in the 8 to 10 range. However, this apparent difference in pain intensity between lidocaine and bupivacaine was not statistically significant. The number of patients queried in the tetracaine group (n = 2) was too low to evaluate. Of note, none of the patients in this study reported dysesthesia as an isolated finding, which effectively reduced the outcome variable to pain in the buttocks, lower extremities, or both.

Analysis of potential factors that might increase the risk of TNSs with lidocaine failed to demonstrate an

Table 3. Duration and Intensity of Leg or Buttock Pain

	Lidocaine	Bupivacaine	Tetracaine	Total
Time to resolution	(N = 104)	(N = 10)	(N = 4)	(N = 118)
≤72 h	70 (67.3)	4 (40.0)	2 (50.0)	76 (64.4)
>72 h to 1 wk	27 (26.0)	5 (50.0)	1 (25.0)	33 (28.0)
>1 wk to 1 mo	6 (5.8)	1 (10.0)	0	7 (5.9)
>1 mo to 6 mo	1 (1.0)	0	1 (25.0)	2 (1.7)
>6 mo	0	0	0	0
Pain intensity*	N = 83	N = 8	N = 2	N = 93†
Mild (1–3)	18 (21.7)	2 (25.0)	0	20 (21.5)
Moderate (4–7)	40 (48.2)	5 (62.5)	1 (50.0)	46 (49.5)
Severe (8–10)	25 (30.1)	1 (12.5)	1 (50.0)	27 (29.0)

Values are number (%). * Pain rated on scale of 0 to 10 where 0 = “no pain” and 10 = “the worst pain you can imagine.” † Data for 25 of the 118 patients with leg or buttock pain were not obtained because pain intensity was added to the interview protocol after the study had commenced.

Table 4. Lidocaine Dose and Transient Neurologic Symptoms

Lidocaine Dose (mg)	Leg or Buttock Pain	
	Yes [number (%)]	No [number (%)]
<50	11 (12.8)	75 (87.2)
51–74	75 (12.0)	552 (88.0)
75+	18 (11.3)	142 (88.8)

effect of lidocaine dose (table 4). In contrast, surgery in the lithotomy position showed a significant effect (table 5); that is, the lithotomy position (compared with other positions) conferred a relative risk of developing TNSs of 2.6 (95% CI, 1.5 to 4.5) if the patient had received lidocaine. This effect of the lithotomy position was not seen with bupivacaine, and the difference between these groups was statistically significant. Outpatient status was a significant risk factor for TNSs after lidocaine (relative risk, 3.6; 95% CI, 1.9 to 6.8), whereas obesity (body mass index, >30) had borderline significance (relative risk, 1.6; 95% CI, 1 to 2.5). In contrast to lithotomy, outpatient status and obesity showed similar effects with bupivacaine, and the effect observed with lidocaine did not differ significantly when compared with the bupivacaine reference. However, outpatient status and obesity were not statistically significant within the relatively small group of patients in whom TNSs developed after they received bupivacaine.

The type of spinal needle used did not affect the relative risk of developing TNSs after lidocaine (table 6). Other factors examined for their potential effect in modifying TNSs in patients who received lidocaine but that were not found to exert an effect are listed in table 6. Although adding epinephrine to the anesthetic solution did not affect the incidence of TNSs with lidocaine, all four patients in whom TNSs developed after tetracaine had received epinephrine.

Of the four other neurologic symptom outcomes de-

Table 5. Effect of Surgical Position and Outpatient Status on Incidence of Transient Neurologic Symptoms

	Lithotomy		Nonlithotomy	
	Outpatient	Inpatient	Outpatient	Inpatient
Lidocaine	57/235 (24.3)	7/91 (7.7)	34/357 (9.5)	6/190 (3.1)
Bupivacaine	0/46 (0)	3/125 (2.4)	4/90 (4.4)	3/483 (0.6)
Tetracaine	0/3 (0)	1/59 (1.7)	0/7 (0)	3/177 (1.7)

Values are number (%).

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Table 6. Factors That Did Not Increase the Risk of Developing Leg or Buttock Pain in Patients Receiving Lidocaine

Factor	Category
Gender	Male vs. female
Age	<60 yr vs. 60+ yr
Race	White vs. other
ASA Class	I, II, III, or IV
Height	Bottom 20% in study sample vs. top 20%
History of back pain	Yes vs. no
Preexisting neurologic condition	Yes vs. no
Needle type	Quincke vs. pencil point
Needle size	22 vs. 24–25 vs. 26–27
Position during injection	Sitting vs. lateral
Approach for injection	Midline vs. paramedian
Interspace for injection	L2–3 or L3–4 vs. L4–5 or L5–S1
Bevel/aperture direction on injection	Caudad vs. cephalad vs. lateral
Lidocaine dose	≤50 mg vs. 51–74 mg vs. ≥75 mg
Intrathecal epinephrine	Yes vs. no
Intrathecal fentanyl	Yes vs. no
Intrathecal morphine	Yes vs. no
Intrathecal dextrose	Yes vs. no
Technical difficulty	Yes vs. no
Paresthesia	Yes vs. no
Blood-tinged CSF	Yes vs. no
Final block height (pinprick)	<T10 vs. T10 or higher
Satisfactory block for procedure	Yes vs. no
Hypotension	Yes vs. no
Intravenous vasopressor	Yes vs. no

CSF = cerebrospinal fluid.

tected (table 7), all were low in incidence, ranging from 0.16% for lower extremity weakness to 0.66% for persistent urinary retention (>24 h) requiring catheterization. The numbers were too small for meaningful statistical treatment. Five of the 16 patients who reported one or more of the symptom outcomes listed in table 7 had preexisting spinal disease and a history of radiculopathy, and another was morbidly obese. Four of the 16 patients also reported leg or buttock pain, and all four had received lidocaine. All patients with lower extremity neurologic symptoms had complete resolution of their symptoms within 1 month, except for one patient in the bupivacaine group who reported paresthesias that gradually resolved over 2 months, one patient in the lidocaine group with spinal stenosis who reported leg weakness that resolved over 6 months with palliative therapy, and one patient in the lidocaine group with a history of L5–S1 radiculopathy and a diagnosis of “probable idiopathic peripheral neuropathy” who reported numbness of the feet after operation that resolved almost com-

pletely over 18 months of clinical follow-up (the patient continued to report left fifth toe hypesthesia). One morbidly obese patient in whom numbness and paresthesias developed in an L3 radicular distribution had received intrathecal bupivacaine after unsatisfactory lidocaine epidural anesthesia for cesarean section delivery. The paresthesias had resolved, but numbness persisted at the 1-month follow-up call, and thereafter the patient could not be contacted, did not seek evaluation of her symptoms, and was lost to follow-up.

Of the nine patients who experienced urinary retention that required catheterization, two had preexisting urinary retention and two others had urinary tract infections. All patients with urinary retention symptoms were free of symptoms and no longer required a catheter by the end of 1 week.

Of the database of 1,863 patients, 12 received a repeated spinal injection after an unsatisfactory block, and these cases were distributed proportionally among the three local anesthetic groups (six lidocaine, four bupivacaine, two tetracaine). None of the 12 experienced TNSs. One of these patients developed numbness and tingling of the legs that lasted 1 week after two injections of lidocaine, 60 and 75 mg, respectively.

The incidence of postoperative orthostatic and non-orthostatic headache was similar among the three local anesthetic groups.

Discussion

Our results correspond with previous data indicating that TNSs commonly follow lidocaine spinal anesthesia but are relatively uncommon with bupivacaine or tetracaine.^{10–15} Our data add to the existing literature by providing evidence that lithotomy position and outpatient status are significant risk factors predisposing to TNSs in patients who receive lidocaine. Although the mechanism of the interaction between lidocaine and lithotomy position is unknown, it has been postulated that lithotomy positioning predisposes to lidocaine toxicity because of stretching of the cauda equina, reducing tissue perfusion and increasing vulnerability of the nerve fibers.⁹

In a study of patients receiving intrathecal lidocaine for hernia repair or knee arthroscopy, a lower incidence of symptoms was observed among the supine hernia patients (5%) compared with the arthroscopy patients (13%) in whom the nonoperative leg was flexed at the knee while the operative leg was manipulated to facili-

Table 7. Local Anesthetics and Other Neurologic Symptom Outcomes*

	Lidocaine (N = 873)	Bupivacaine (N = 744)	Tetracaine (N = 246)	Total (N = 1,863)
Lower extremity weakness				
No	870	744	246	1,860
Yes	3	0	0	3
Lower extremity numbness				
No	867	741	245	1,853
Yes	6	3	1	10
Lower extremity paresthesias				
No	867	740	245	1,852
Yes	6	4	1	11
Urinary retention				
No	589	576	177	1,342
Yes	2	5	2	9
Excluded†	282	163	67	512

* Seven patients reported more than one of these symptom outcomes.

† Patients who had urologic surgery or indwelling Foley catheters as part of their routine postoperative care were excluded from evaluation of urinary symptoms.

tate surgery.¹³ The authors suggested that, as with lithotomy, positioning for arthroscopy produced stretch of the lumbosacral nerves, contributing to the development of symptoms. In contrast, a more recent study found that tetracaine with phenylephrine resulted in a high incidence of symptoms, but this incidence was not further increased by lithotomy position.¹⁵ The results of the present study suggest that such discrepant findings result from administration of different local anesthetics, because risk with lithotomy was not increased in those patients who received bupivacaine or tetracaine. However, the small number of patients experiencing TNSs with bupivacaine or tetracaine in the present study necessitate caution in interpreting this finding.

Outpatients demonstrated an increased risk for developing TNSs after lidocaine, suggesting that early ambulation, return to activity, or less complicated convalescence might be important factors, as has been postulated.^{16,17} In addition, obesity had a borderline effect. This could result perhaps from higher concentrations of local anesthetic in cerebrospinal fluid because obese patients have a lower cerebrospinal fluid volume.¹⁸

Many factors postulated to increase risk for TNSs after lidocaine spinal anesthesia did not appear to exert an effect. Most surprising, lidocaine dose did not modify risk, even when adjusted for lithotomy position, outpatient status, body mass index, and patient height (table 4). Also surprising are the data concerning the effect of the spinal needle. Some authors have suggested that pencil-point, or non-Quincke needles, may predispose patients to anesthetic neurotoxicity because of flow

characteristics that may promote local anesthetic pooling within the subarachnoid space.^{19,20} Others have suggested that pencil-point needles may dislodge the arachnoid membrane from the dural sheath more than Quincke needles, increasing the likelihood of symptoms resulting from subdural dissection of blood and cerebrospinal fluid.¹⁶ Needle size also has been postulated to have an effect; specifically, the lower flow rates associated with smaller needles could predispose patients to higher anesthetic concentrations in cerebrospinal fluid. Supporting this concept, *in vitro* spinal model dye studies show high peak concentrations when slow injections are made through small-bore needles.²⁰ Nonetheless, we did not observe an effect of needle type or size on the incidence of TNSs. In addition, aperture direction of pencil-point needles did not affect risk.

Other technical aspects of the spinal anesthetic also failed to affect the risk for developing TNSs. A previous study suggested that a higher level of dural puncture increases the likelihood that pain in the legs, back, or both will develop after lidocaine spinal anesthesia,²¹ but our data failed to confirm an effect of interspace. It could be postulated that injections made with the patient seated rather than in the lateral decubitus position could increase the risk for TNSs by promoting pooling within a restricted area of the subarachnoid space. However, we did not detect any increased risk associated with patient position during local anesthetic injection.

We could not document any effect of the components of the anesthetic solution on lidocaine-associated TNSs. Data from previous studies suggest that decreasing the lidocaine concentration from 5% to 2% does not signifi-

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cantly reduce the incidence of TNSs,^{13,14} and TNS symptoms have been reported in patients receiving concentrations of lidocaine as low as 0.5%.²² The results of the present study also suggest that injectate concentrations ranging from 1.5% to 5% confer a similar risk. Similarly, previous studies have shown that neither hyperosmolarity nor the presence of dextrose affect the risk for TNSs,^{11,13} which is consistent with our current data. Neither intrathecal fentanyl nor intrathecal morphine affected the risk for TNSs. Neither did we observe an effect of epinephrine on the incidence of TNSs associated with lidocaine. These results are consistent with a previous clinical study¹³ but are inconsistent with animal data suggesting that adding epinephrine increases neurotoxicity of intrathecal lidocaine.²³ However, it has not been established that anesthetic-induced neurologic injury and transient pain or dysesthesia share a common mechanism.

Previous clinical data indicate that phenylephrine increases the risk of tetracaine-induced TNSs.¹⁵ Although our sample did not include any phenylephrine-containing anesthetics, it is interesting to note that all four of the patients with TNSs after tetracaine had received a solution containing epinephrine. This observation reinforces the need for a directed study to determine whether any vasoconstrictor added to tetracaine increases risk, rather than being an effect specific to phenylephrine.

Table 6 lists other factors of interest that did not increase the risk for TNSs to develop with lidocaine. A previous study of 54 patients suggested that younger patients were more likely to develop pain in the legs, back, or both after lidocaine spinal anesthesia,²¹ but we did not find an effect of patient age. Similarly, sex had no effect. Our data, therefore, suggest that the high (37%) incidence of TNSs in women receiving lidocaine spinal anesthesia for elective gynecologic and obstetric procedures reported by Hampl *et al.*¹⁰ is more likely attributable to surgery in the lithotomy position and outpatient status than to an effect of sex or age.

We were interested in the effect of repeating a spinal anesthetic for the same procedure, because this factor has been implicated in the development of postoperative neurologic symptoms or injury.^{3,19} However, there were too few cases in this category to allow meaningful statistical analysis. Although we were also interested in the effect of the type of surgery on the incidence of TNSs, we could not evaluate this factor independently because of its high correlation with patient position.

No major neurologic sequelae were found in our 1,863 patients. However, there were 16 cases of transient

lower extremity weakness, numbness, paresthesia, or all three. One patient reported mild residual hypesthesia of the left lateral fifth toe that was still improving after 18 months. We are unaware of reports of permanent deficits in association with TNSs, and no permanent deficits developed in any of the 118 patients with TNSs in our study.

It has been postulated that there is a risk of neurotoxicity with repeat injection after a failed spinal block. Specifically, if maldistribution is the cause of failure, reinjection may result in high concentrations of anesthetic within a restricted area of the subarachnoid space, with resulting neurotoxic injury.⁷ This mechanism may account for one of the transient deficits observed in the present study: In this case, the patient developed numbness and tingling of the legs persisting for 1 week after receiving two injections of lidocaine, 60 and 75 mg, respectively. That only 1 of the 12 repeated spinal injections resulted in a deficit is not inconsistent with this mechanism. First, not all failed spinal blocks result from maldistribution; that is, inadequate block may also result from failure to deliver the drug into the subarachnoid space. Second, in three of these cases the total dose from the two injections did not exceed current manufacturers' recommendations for single-injection spinal anesthesia, and it was within 10% of the suggested maximum dose in three other cases. Finally, of the remaining 6 cases, only three involved repeated administration of lidocaine, which likely has a lower therapeutic index than bupivacaine or tetracaine.²⁴⁻²⁸

Any consideration of the clinical significance of TNSs must account for the severity of the pain. In some previous reports, pain has been so incapacitating as to require repeated hospitalization for pain management.²⁹ In the current study, 25 of 83 patients with TNSs in the lidocaine group rated their pain as severe (≥ 8 of 10) compared with only one of eight symptomatic bupivacaine patients.

Our study has several limitations. First, because the anesthesiologists administering the spinal blocks were not blinded, bias cannot be excluded. However, the research nurse who contacted patients and collected the postoperative data was blinded to all aspects of anesthetic care. Second, it is possible that voluntary participation could have resulted in a case submission bias; that is, there could have been a bias against submitting cases where complications or difficulties were encountered intraoperatively, or, alternatively, complicated cases could have been viewed as more interesting and therefore more likely to be submitted.

However, it is doubtful that such an effect would differentially affect the local anesthetic subsets. Furthermore, the reported incidence of technical difficulty (*i.e.*, attempts at more than one interspace) was comparable for all three groups, and their relatively high incidence (10.4% to 14.7%) would argue against a selection bias toward less difficult cases. Third, we cannot rule out the presence of an unidentified factor that could cause the lidocaine group to be more likely to report leg or buttock pain symptoms. However, all three groups exhibited a similar incidence of headaches, both orthostatic and nonorthostatic, suggesting that the lidocaine group did not have a greater tendency to report discomfort. Fourth, our study was limited by its focus on symptoms, not objective findings. However, these symptoms are the hallmark of this complication. In fact, the term TNS was proposed by Hampl *et al.*¹⁰ as a replacement for their earlier term, *transient radicular irritation*,⁹ because it does not imply objective signs or presume a particular mechanism. Furthermore, as is the case for postdural puncture headache, a lack of objective findings does not negate the importance of this complication. Finally, our study did not include a general anesthesia cohort. However, the very low incidence of symptoms in the bupivacaine group effectively permits its use as a control; that is, the dramatic difference between the lidocaine and bupivacaine groups strongly suggests that TNSs are etiologically related to the lidocaine anesthetic and not to nonspecific factors related to surgery or to the spinal technique.

In conclusion, our data demonstrate that spinal anesthesia with lidocaine markedly increases the risk for TNSs to develop compared with bupivacaine or tetracaine. These symptoms are relatively common after lidocaine spinal anesthesia (11.9%) but uncommon after bupivacaine (1.3%) or tetracaine (1.6%). The pain associated with TNSs is often severe (30.1%), and >90% of cases resolve completely within 1 week. Our data identify lithotomy position, outpatient status, and perhaps obesity as significant factors that increase the risk for TNSs after spinal anesthesia with lidocaine. Our data indicate a spectrum of risk for TNSs after lidocaine, ranging from 3.1% for inpatients having surgery in positions other than lithotomy to 24.3% for outpatients in the lithotomy position. We failed to identify any other statistically significant risk factor for TNSs with lidocaine. Of most relevance to clinical practice are our observations that neither age, sex, history of back pain, lidocaine dose or concentration, spinal needle type or size, aper-

ture direction, sitting position during injection, nor addition of epinephrine appear to affect the risk for developing TNSs after lidocaine spinal anesthesia. Our follow-up data on other neurologic symptoms suggest that such postoperative problems are uncommon and that most resolve spontaneously within 1 month from the time of the anesthetic.

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Follow-up Interview Questions

1. Do your eyes feel sensitive to light?
2. Have you had any nausea or vomiting after your operation? (IF YES) When did it start?
3. Do you still have nausea or vomiting?
4. Have you had any problem(s) with bowel movements? (If constipated) Did you get any medication for it?
5. Have you had any problem(s) urinating? (IF YES) What kind of problem(s)? When did the problem(s) start? Do you still have these problems?
6. Have you had any unusual sensation(s) anywhere in your body? (IF YES) Can you describe them for me? When did they start? Do you still have these unusual sensations?
7. Have you had numbness anywhere in your body? (IF YES) Can you describe this for me? When did it start? Do you still have numbness?
8. Have you had any weakness of your arms or legs? (IF YES) Where is/was the weakness? When did it start? Do you still have weakness?
9. Did you have to take any pain medication after your operation? IF YES) What is/was the pain medication for?
10. Did you have a headache after your operation? (IF YES) Does/did the headache go away when you lay flat on your back and then return when you sat up or stood up? (IF YES) Was it continuous or did it come and go? Was it mild, moderate, or severe? Which part of your head hurt? When did it start? Do you still have the headache?
11. Besides your surgical pain (and your headache—if present), have you had any other pain anywhere else in your body? (IF YES) Where is/was the pain? (IF BACK, BUTTOCK, HIP, or LEG) Have you had this pain before your surgery? What part of your _____ hurt(s)? Which side? Can you describe your pain to me? Is/was it continuous or does it come and go? Does/did the pain get worse in a certain position? Does/did a certain position take the pain away? When did the pain start? When would you say was the time your _____ was most painful? Does/did it get worse at a specific time of the day? Do you still have the pain? (IF NO) When did it disappear? (FOR BUTTOCK and LEG ONLY) On a scale of 0-10, with 0 being no pain and 10 being the worst pain you can imagine, how would you rate your _____ pain?
12. Have you had any pain that travels from one part of your body to another? (IF YES) Can you describe it to me? Has this pain limited your activity after the operation?