Anesthetic and Hemodynamic Effects of Single Bolus Versus Incremental Titration of Hyperbaric Spinal Lidocaine Through Microcatheter

Vincent W. S. Chan, MD*, Frances Chung, MD†, Manuel Gomez, MD†, Chanth Seyone, MD†, and Godofredo Baylon, MD

Departments of Anesthesia, *University of California, San Francisco, California, and †University of Toronto, Toronto, Ontario, Canada

This study examined anesthetic development and hemodynamic changes during two techniques of hyperbaric lidocaine administration through 27-gauge intrathecal catheters for continuous spinal anesthesia in 55 elderly patients undergoing transurethral prostatic resection. Twenty-five patients were randomly assigned to receive hyperbaric lidocaine 5% solution in a single bolus of 75 mg to achieve blockade to approximately T6, and 30 patients to receive hyperbaric lidocaine 2.5% solution in increments of 25 mg to achieve T6 or to a maximum of three doses. Hemodynamic measurements of arterial pressure, heart rate, cardiac output, stroke volume, and ejection fraction were made serially after the induction of spinal anesthesia. Anesthesia failed to spread beyond the sacral region in 9/25 (36%) patients in the single-dose group (SD), but was successfully induced in all patients given titrated doses (TD) at total doses of 50 mg (n = 15) (TD50) and 75 mg (n = 15) (TD75). The mean maximal level of sensory block in all three groups was comparable: T5, T4, and T6 in groups SD, TD50, and TD75, respectively. The onset and progression of sensory block were rapid and similar in the SD and TD50 groups, in contrast to a gradual, stepwise development of block in group TD75. Grade 3 motor block occurred in response to the first dose of 25 mg lidocaine in 7/30 (23%) patients receiving titrated doses, but the overall incidence of leg paralysis did not differ among the three groups by the end of lidocaine dosing. Mean arterial pressure (MAP) decreased significantly relative to baseline in all three groups. The maximum decline in MAP was comparable in all groups although at different time points: SD = −19.7% ± 3.9%; TD50 = −23.1% ± 5.7%; and TD75 = −16.3% ± 3.2%. The decline in cardiac output, stroke volume, and heart rate was similar in all three groups. Our results indicate that a single-bolus administration of 75 mg of hyperbaric 5% lidocaine does not provide blockade as consistently as does a sequential-bolus administration of either 50 mg or 75 mg of hyperbaric 2.5% lidocaine in 25-mg increments. Furthermore, sequential dosing does not consistently result in stepwise development of motor and sensory block. When lidocaine was injected as described in this study, the hemodynamic effects of the two administration techniques do not differ significantly. Interindividual variability in response to lidocaine may account for the varying degrees of blockade and cardiovascular effect produced by the same (low) dose.

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The technique of continuous spinal anesthesia (CSA) is thought to have the advantage of providing greater control over anesthetic development and hemodynamic derangement than the conventional single-bolus needle injection technique. However, objective evidence is needed to support such a claim. In theory, administration of local anesthetic through an indwelling intrathecal catheter offers the flexibility of sequential dosing in small increments guided by the patient's anesthetic response. It is generally believed that the use of titration dosing for CSA permits greater control of the gradual development of anesthetic blockade to a level appropriate for surgery, thereby avoiding an undesirably high blockade with precipitous hypotensive consequence (1,2). Repetitive dosing via an intrathecal catheter also obviates the need for a long-acting anesthetic, matches anesthetic duration to surgical time, and potentially permits the administration of analgesics in the postoperative period (3).

Although the concept of CSA is attractive, many of its perceived advantages over conventional single-bolus

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Address correspondence to Vincent W. S. Chan, MD, Department of Anesthesia, University of California, San Francisco, Box 0648, San Francisco, CA 94143-0648.

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injection have not been proven. Results of studies examining the benefits of sequential dosing are not consistent because dosing intervals were not standardized or because titration technique varied from bolus dosing (4–6) to continuous infusion (7). Additionally, prior studies of the anesthetic and hemodynamic effects of CSA focus primarily on bupivacaine solutions of varying baricities (4–7); little is known about lidocaine. In the present study, we compare two clinically common methods of lidocaine administration for continuous spinal anesthesia—single-bolus dosing of hyperbaric 5% lidocaine versus sequential-bolus dosing of hyperbaric 2.5% lidocaine—to evaluate the anesthetic efficacy and hemodynamic consequences of each technique. We hypothesize that sequential-bolus dosing will produce gradual development of anesthetic blockade in a controllable manner and a lesser degree of hypotension.

Methods

With approval from our institutional review committee and informed consent, we studied 55 ASA physical status II or III patients scheduled for transurethral resection of prostate during spinal anesthesia. Patients ranged in age from 54 to 87 yr, in weight from 53 to 100 kg, and in height from 161 to 191 cm. All were free of significant neurologic disease, myocardial dysfunction, or uncontrolled hypertension. Patients taking chronic antihypertensive or antianginal medications continued their normal dosing regimens until the morning of surgery.

All patients were premedicated with diazepam, 5–10 mg *per os* 90 min prior to the procedure. After hydration with approximately 500 mL intravenous (IV) crystalloid solution (plasmalyte), dural puncture was performed using a 22-gauge spinal needle inserted in the midline at the L2–3 or L3–4 interspace while the patient lay in lateral decubitus position. An indwelling 27-gauge spinal catheter (Medevice, Mississauga, Ontario, Canada) was placed to a depth of 2–3 cm in the subarachnoid space. All patients were then turned supine and maintained in a horizontal position for the initial 45 min of study.

Patients were randomly divided into two study groups. To provide sensory block to approximately T6, the first group (n = 25) received a single 75-mg dose (SD) of 1.5 mL hyperbaric 5% lidocaine with 7.5% dextrose (specific gravity = 1.033), and the second group (n = 30), incremental 25-mg doses of 1 mL hyperbaric 2.5% lidocaine with 7.5% dextrose (specific gravity = 1.030) every 15 min, to a maximum of three doses (75 mg). During each dosing period, catheter injection was accomplished within 30–60 s. The investigator performing clinical assessments was not aware of the solution or the dose of lidocaine administered.

During the first 45 min after the induction of spinal anesthesia, the levels of sensory and motor block and hemodynamic variables were measured in the absence of concurrent surgery. The dermatomal level of sensory anesthesia was determined by the loss of pinprick sensation to a 23-gauge needle and the degree of motor block by applying the modified Bromage scale (0 = no block; 1 = hip flexion with extended leg blocked; 2 = knee flexion blocked; 3 = complete motor block) at 3-min intervals. After baseline recording, arterial blood pressure and heart rate were measured automatically (Dinamap) at 1-min intervals while cardiac output, stroke volume, and ejection fraction were measured at 3-min intervals noninvasively using the thoracic electrical bioimpedance method (BoMed NCCOM3, Irving, CA). After the initial 45-min study period, hemodynamics (blood pressure and heart rate) and level of blockade were measured every 15 min during the intraoperative and postoperative periods until complete anesthetic recovery. Routine intraoperative monitoring included electrocardiography and pulse oximetry.

If hypotension occurred, as defined by a decline of systolic blood pressure of 30% or more from preoperative baseline level, IV ephedrine was administered in 5-mg increments until it resolved. If surgical anesthesia was inadequate, supplemental boluses of 12.5–25 mg lidocaine were administered intrathecally as required. However, if anesthetic insufficiency was related to restricted caudal block, further lidocaine doses were withheld and general anesthesia was substituted.

Total lidocaine dose, total administered fluids, and duration of surgery were recorded for both study groups. Statistical analyses were performed using analysis of variance for repeated measures and Student's t-test for paired and nonpaired data. χ^2 analysis was used to compare the incidence of hypotension and incomplete anesthesia in both groups. A value of $P < 0.05$ was considered statistically significant. All results are expressed as mean ± SEM.

Results

Twenty-five patients were enrolled in the single-dose (SD) group and 30 patients in the group given titrated doses (TD). Age, weight, height, duration of surgery, and amount of administered fluids were comparable in the two groups (Table 1).

In the SD group, 9/25 patients (36%) developed insufficient surgical anesthesia restricted primarily to the sacral and perianal regions (S2–4). Surgery in these patients was completed under general anesthesia and their sacral anesthesia resolved completely within 6 h. Data from these patients were therefore excluded, resulting in a total of 16 patients in the SD group with analyzable data (Table 2).

Table 1. Patient Characteristics, Administered Fluids, and Duration of Surgery

	Group SD	Group TD50	Group TD75
Age (yr)	74.3 ± 2.0	71.2 ± 1.7	67.4 ± 2.3
Weight (kg)	79.4 ± 3.4	78.6 ± 2.6	84.2 ± 6.7
Height (cm)	168 ± 3.6	171.3 ± 2.0	172.1 ± 1.1
Crystalloid (mL)			
Pre[a]	496.9 ± 46	486.7 ± 27	384.4 ± 45
Total[b]	1213.3 ± 97	1050 ± 75	931.3 ± 53
Surgical time (min)[c]	41.3 ± 4.2	46.1 ± 5.8	36.1 ± 3.7

SD = single dose; TD50 = titrated doses of 50 mg; TD75 = titrated doses of 75 mg.
[a] Pre = total fluid administration preblockade.
[b] Total = total fluid administration at the end of surgery.
[c] Surgical time reflects the time after the initial 45-min study period.

Table 2. Results for the Single-Bolus and Titrated Regimens

	Group SD	Group TD50	Group TD75
No. of patients	25	15	15
Successful block	16	15	15
Lidocaine solution	5%	2.5%	2.5%
No. of injections	1	2	3
Injectate volume	1.5 mL	1 mL	1 mL
Total injected volume/dose	1.5 mL/ 75 mg	2 mL/ 50 mg	3 mL/ 75 mg
Injection site L2–3/L3–4	2/14	0/15	0/15

SD = single dose; TD50 = titrated doses of 50 mg; TD75 = titrated doses of 75 mg.

In the TD group, none of the patients developed sacrally restricted anesthesia. Anesthetic block to approximately T6 was achieved successfully in all 30 patients. However, 15/30 patients required only two 25-mg doses (50 mg total) to achieve satisfactory blockade while 15 required three 25-mg doses (75 mg total) (Table 2). Therefore, we subdivided the TD group into groups TD50 and TD75 to permit comparison of hemodynamic dose response, resulting in a total of three study groups being compared.

Sensory Block

The speed of onset and spread of anesthesia for all three study groups are presented in Figure 1. The maximum level of sensory anesthesia achieved at the end of the initial 45-min study period was comparable in all groups: T5 (range, T2–T9) in Group SD; T4 (range, T2-T7) in Group TD50; and T6 (range, T2-T12) in Group TD75.

Progression of sensory block, however, was distinctly different. In the SD group, anesthesia in response to pinprick was rapidly detectable in low thoracic dermatomal segments (mean, T10–11) within 3 min after the single 75-mg lidocaine bolus and ascended to T7 and T5 by 15 and 30 min, respectively. Maximum cephalad spread was reached within 13.5 ± 1.5 min (range, 9–33 min) in the SD group. In the TD group, anesthetic response differed by subgroup. Patients in the TD50 group achieved rapid anesthesia, comparable in speed of onset and progression of block to those in the SD group (Figure 1). That is, onset of sensory block to T7 was apparent in the TD50 group within 15 min after the first 25-mg bolus. With the second 25-mg bolus, the level of sensory block ascended further cephalad to approximately T4 by 30 min. Maximum cephalad spread was achieved within 24.2 ± 1.8 min (range, 18–27 min).

Anesthetic response was much slower and gradual in the TD75 group: sensory block to T6 was achieved gradually in a stepwise manner with each successive 25-mg lidocaine dose (Figure 1). Fifteen minutes after the first dose, sensory block was detectable only in the low lumbar region (L1–2). With the two subsequent doses, sensory block was achieved to T10 and T6 by 30 and 45 min, respectively. Maximum cephalad spread was achieved within 42.3 ± 1.7 min (range, 33–55 min).

Excluding the nine patients with block failure in the SD group, all other patients obtained adequate sensory block as assessed by pinprick testing during the first 45 min of study. However, a single supplemental bolus of intrathecal lidocaine (12.5–25 mg) was required to maintain surgical anesthesia in four patients in the SD group at 45–80 min, two patients in the TD50 group at 82–85 min, and in one patient in TD75 group at 64 min from the time of spinal anesthesia induction. Regression of sensory block to T12 level was detected by 92.5 ± 7.3 min, 108.8 ± 8.4 min, and 114.7 ± 7.4 min in the SD, TD50, and TD75 groups, respectively, while the time to reach complete block resolution was 130.7 ± 7.4 min, 125.3 ± 7.1 min, and 143.8 ± 5.1 min, respectively.

Motor Block

Development of motor block was most rapid in the SD group (Table 3). Complete lower-limb paralysis was detectable by 3–15 min in most SD patients, compared with 18–27 min in the TD50 group and 33–45 min in the TD75 group. After the single 75-mg lidocaine bolus dose, Grade 3 blockade was apparent in 81% of patients in the SD group at 15 min, 88% at 30 min, and 94% at 45 min.

In the TD group, the pattern of motor block response differed by subgroup (Table 3), as had the progression of sensory block. Fifteen minutes after the first 25-mg dose, 6/15 patients (40%) in the TD50 group had developed Grade 3 motor block but only 1/15 in the TD75 group had reached this level. After the second 25-mg dose, all 15 patients in the TD50 group had obtained complete leg paralysis, compared with 7/15 patients (47%) in the TD75 group. Even after the third 25-mg

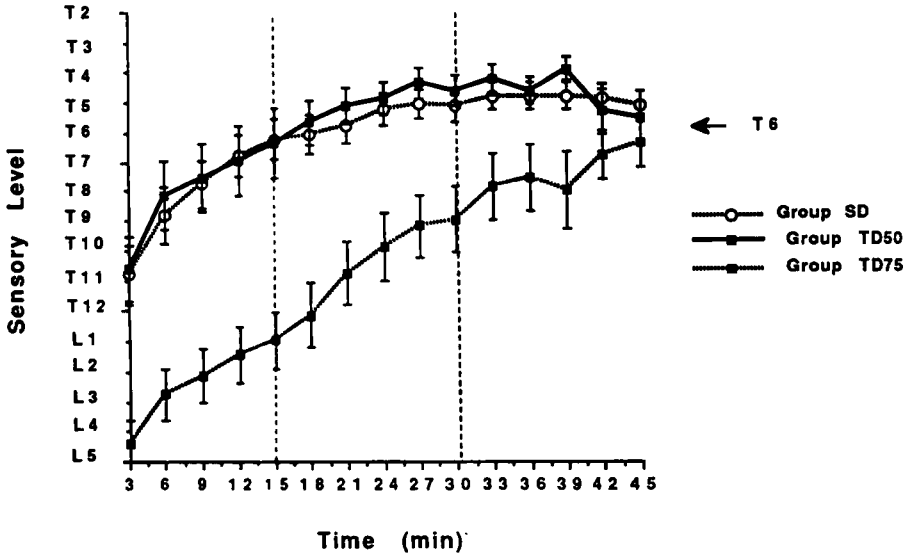

Figure 1. The speed of onset and progression of anesthesia were similar for the single-dose (SD) and the 50-mg titrated dose (TD50) groups, and significantly slower in the 75-mg titrated dose (TD75) group. However, the maximum level of sensory anesthesia achieved at the end of the initial 45-min study period was comparable in all groups: T5 (range, T2–9) in Group SD; T4 (range, T2–7) in Group TD50; and T6 (range, T2–12) in Group TD75, all values calculated as the mean ± SEM. The progression of sensory block in the SD and TD50 groups was indistinguishable clinically, despite differences in the injection technique and the total administered dose.

Table 3. Development of Complete (Grade 3) Motor Block

Time (min)	Group SD (n = 16)	Group TD50 (n = 15)	Group TD75 (n = 15)
9	69%	33%	7%
15	81%	40%	7%
30	88%	100%	47%
45	94%	100%	80%

SD = single dose; TD50 = titrated doses of 50 mg; TD75 = titrated doses of 75 mg.

dose, only 12/15 patients (80%) in the TD75 group had obtained complete leg paralysis.

Hemodynamic Changes

As shown in Table 4, baseline values for mean arterial pressure (MAP), heart rate, cardiac output (CO), stroke volume (SV), and ejection fraction did not differ significantly among the three study groups. The changes in hemodynamic values during the initial 45-min period of study after induction of spinal anesthesia also did not differ significantly (Table 5). Although the maximum decrease in MAP occurred earlier in the SD group (Figure 2), the magnitude of this decline (mean) did not differ significantly from that for the TD50 and TD75 groups: SD = −19.7% ± 3.9%; TD50 = −23.1% ± 5.7%; and TD75 = −16.3% ± 3.2%. As measured by the thoracic electrical bioimpedance method, all three groups displayed similar patterns of maximal decline in CO and SV, but the extent of the decrease in CO cannot fully account for the degree of hypotension observed (Table 5). Although mild slowing of the heart rate was observed in all three groups, bradycardia (<50 bpm) did not occur. Ejection fraction declined in both TD groups, but increased in the SD group.

Ephedrine was required in two and four patients in the SD and TD50 groups, respectively. In the SD group,

Table 4. Baseline (Preblockade) Hemodynamic Data for the Three Study Groups

Measurements	Group SD (n = 16)	Group TD50 (n = 15)	Group TD75 (n = 15)
MAP (mm Hg)	113.2 ± 5.1	105.1 ± 3.3	112.5 ± 4.6
CO (L/min)	5.0 ± 0.4	5.6 ± 0.4	5.6 ± 0.7
SV (mL)	74.1 ± 6.1	82.9 ± 4.4	81.5 ± 10.9
HR (bpm)	68.6 ± 2.1	66.1 ± 2.8	70.2 ± 2.8
EF (%)	57.9 ± 1.7	59 ± 2.2	57.8 ± 3.4

Values are expressed as the mean ± SEM. There were no significant differences among groups in baseline hemodynamic values.

SD = single dose; TD50 = titrated doses of 50 mg; TD75 = titrated doses of 75 mg; MAP = mean arterial blood pressure; CO = cardiac output; SV = stroke volume; HR = heart rate; EF = ejection fraction.

the two hypotensive episodes (a decline of 36% and 49% from baseline systolic pressure) occurred at 8 and 11 min after the single 75-mg dose while in the TD50 group, the decline of 37%–70% was seen at 20–41 min. No patient in the TD75 group required a vasopressor. The amount of perioperative crystalloid infusion did not differ among the groups (Table 1).

Discussion

Our comparison of the single-bolus and sequential-dosing techniques of hyperbaric lidocaine administration for continuous spinal anesthesia suggests that anesthetic efficacy is influenced by the choice of technique. Sequential 25-mg dosing with hyperbaric lidocaine 2.5% solution in 1-mL aliquots was successful in establishing sensory block at total doses of 50 or 75 mg in all patients given titrated doses, whereas 36% of patients failed to achieve lower limb anesthesia with single-bolus administration of hyperbaric 5% solution at 75 mg.

Although the occurrence of sacrally restricted block during CSA has been recognized, we were surprised by

Table 5. Maximum Change of Hemodynamic Variables from Control Values (0%) and Time of Maximum Change

	Group SD (n = 16)		Group TD50 (n = 15)		Group TD75 (n = 15)	
	% Change	Time (min)	% Change	Time (min)	% Change	Time (min)
MAP (mm Hg)	−20%	15–30	−23%	30–45	−16%	30–45
CO (L/min)	−10%	30–45	−15%	15–30	−7%	30–45
SV (mL)	−9%	15–30	−10%	15–30	−8%	30–45
HR (bpm)	−5%	30–45	−10%	30–45	−2%	30–45
EF (%)	+8%	30–45	−11%	15–30	−7%	30–45

Values are expressed as the mean ± SEM and are reported only for the first 45-min period of study. There were no significant differences among groups.
SD = single dose; TD50 = titrated doses of 50 mg; TD75 = titrated doses of 75 mg; MAP = mean arterial blood pressure; CO = cardiac output; SV = stroke volume; HR = heart rate; EF = ejection fraction.

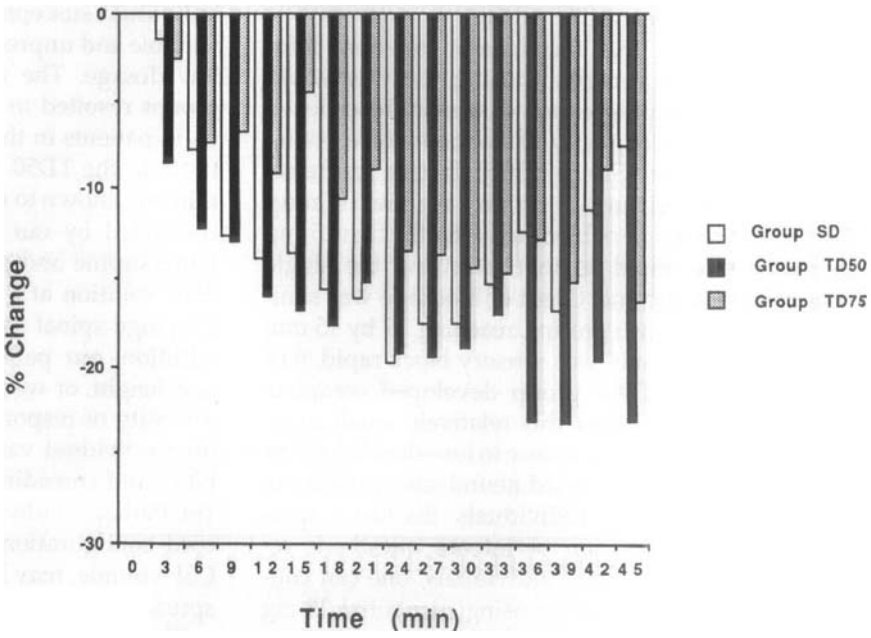

Figure 2. The maximum decrease (% change) in mean arterial pressure (MAP) occurred earlier in the single-dose (SD) group (within ≈24 min after local anesthetic injection) than in the 50-mg titrated dose (TD50) group (within ≈36 min) or the 75-mg titrated dose (TD75) group (within ≈45 min). However, the mean magnitude of the decrease in MAP did not differ significantly among groups: SD = −19.7% ± 3.9%; TD50 = −23.1% ± 5.7%, and TD75 = −16.3% ± 3.2%.

the high incidence (36%) observed in the single 75-mg dose group, in contrast to previously reported anesthetic failure ranging from 4% to 7% (4,5). Difficulty with catheter insertion was not encountered in the present study and the presence of restricted sacral anesthesia strongly suggested that catheters were lying in the subarachnoid space, but may have been directed sacrally rather than cephalad. All sacral blocks were resolved completely within 6 h and none of these patients developed complications of cauda equina syndrome, probably because the dose of lidocaine was not high (8,9).

A sacrally restricted anesthetic block may result from either drug maldistribution or catheter malpositioning (10–12). We used 27-gauge catheters and injected 1.5 mL lidocaine within 1 min. This may have retarded anesthetic cerebrospinal fluid (CSF) mixing resulting in drug maldistribution. Within the subarachnoid space, it is also conceivable that the catheters were unintentionally placed with tips pointing caudad or at the peak of lumbar lordosis curvature, allowing an injected dose to gravitate preferentially to the sacral-dependent end of the spinal canal. Finally, as in other cases of failed anesthetic, the intrathecal catheter may have advanced to sit at the sacrum (13). Without radiographic studies, it is impossible to accurately determine the cause of sacrally restricted blockade in our patients in the single-dose group.

Interestingly, none of the patients receiving sequential doses of hyperbaric lidocaine 2.5% solution developed a sacral block. Differences between groups beyond technique include the use of a more dilute solution (2.5% vs 5%) and a larger total volume of injection (3 mL vs 1.5 mL). Among these factors, the volume of local anesthetic injection may be most crucial. If anesthetic maldistribution and poor spreading are related to limited CSF mixing, perhaps a larger volume bolus would facilitate drug dispersement. The slow progression of anesthetic response observed in the TD75 group of patients may indicate limited anesthetic spreading during the initial period, but administration of additional lidocaine successfully moved the level of blockade cephalad, possibly by improved CSF-local anesthetic mixing due to the use

of gradually higher volumes of lidocaine. Van Gessel et al.'s (4) finding that satisfactory block was achieved in most cases when hyperbaric bupivacaine 0.25% was injected in a single 3-mL bolus provides some support for this volume-dependent hypothesis. Possibly, when administered using the conventional needle technique, spinal anesthetic spreading is more dependent on the injected dose than on volume. Uniform CSF mixing can be achieved easily by rapid anesthetic injection through the needle. However, when the rate of anesthetic distribution is slowed by small-bore spinal catheters, the injected volume may then be particularly important to facilitate adequate drug-CSF mixing.

We hypothesized that incremental low-dose lidocaine (25 mg) given at regular dosing intervals would produce a stepwise progression of sensory block level, but this was observed in only 50% of patients receiving the titrated regimen (Group TD75). In fact, in the remaining patients given the titrated regimen (Group TD50), the progression of blockade after the first 25-mg injection was as rapid as that following the single 75-mg bolus of lidocaine. Onset of blockade was similarly rapid in these two groups, reaching T7 by 15 min after injection. Not only was sensory block rapid, 6 of 15 patients in the TD50 group developed complete lower limb paralysis after this relatively small dose. The dramatic anesthetic response to low-dose lidocaine may be explained by increased neural susceptibility to local anesthetic in these individuals. Because unpredictable rapid progression of intense anesthetic response may occur in some individuals, one can conclude that anesthetic titration using sequential 25-mg dosing is not reliably effective. If the degree of titratability is dose-dependent, perhaps a smaller starting lidocaine dose, less than 25 mg, may facilitate a more gradual anesthetic development.

Similar to what we have observed, both Labaille et al. (6) and Petros et al. (5) reported that surprisingly small amounts of hyperbaric bupivacaine (2.5–5 mg) were required to achieve surgical anesthesia in patients undergoing lower limb surgery. Elderly patients receiving 4.8 mg or 16.1 mg plain bupivacaine developed sensory analgesia to T4–10 levels, with no significant difference based on dosage. Despite the three-fold difference in dose, the duration of bupivacaine block was only 30% shorter in the low-dose group. Similarly, in our TD50 group, the initial sensory response within 15 min of injection of 25 mg of lidocaine was clinically identical to that of patients given a single 75-mg dose, and the ultimate level of blockade (T4 and T5, respectively) was similar within a similar time frame.

Although patients in the TD50 and TD75 groups received lidocaine in an identical manner, only those receiving sequential dosing in the TD75 group demonstrated the expected stepwise development of blockade. Patients in the TD75 group developed both sensory and motor block in an exceptionally controlled manner, showing a slow progression, but appropriately intensified response, to each additional 25-mg bolus, to a total of 75 mg. In this group of patients, the level and degree of block may be adjusted by the frequency and amount of dosing. Although we arbitrarily established the dosing interval in this study at 15 min, our data suggest that this interval can be shortened to approximately 10 min, by which time the anesthetic response to each incremental dose has reached a plateau.

One explanation for the difference in progression of blockade in the TD50 and TD75 groups may be that individual susceptibility to local anesthetic is highly variable and unpredictable even when administered at low dosage. The first 25-mg dose in each of these groups resulted in initial mean block levels as low as L1 in patients in the TD75 group and as high as T7 in those in the TD50 group. Most of the extrinsic determinants known to influence anesthetic spreading were controlled by our study protocol. All patients were lying supine and given the same dose of hyperbaric 2.5% solution at a similar speed of injection through 27-gauge spinal catheters inserted at similar sites. In addition, our patients did not differ significantly in age, height, or weight. We postulate that the observed diversity of response likely results from considerable interindividual variation in local anesthetic susceptibility and spreading as noted in previous studies (14–16). Perhaps, individual intrinsic factors such as spinal cord configuration, the extent of lumbar lordosis, or CSF volume, may have greater influence on anesthetic spreading.

The degree of arterial hypotension after intrathecal administration of local anesthetic correlates with the extent of sympathetic block, which is generally induced at two dermatomal segments higher than the sensory level. We hypothesized that greater hemodynamic disturbance would occur in response to the single-bolus technique than to titration because blood pressure is likely to decrease more precipitously with rapid than with slower onset of sympathetic block.

Our findings indicated that the two techniques of lidocaine administration produced the maximum reduction in MAP at different time intervals; earlier in the SD group and later in the TD75 group of patients (Table 5). The degree of maximum decline in MAP was, however, comparable in all three groups. We failed to confirm Palas' (1) observation that the degree of hypotension was less during CSA most likely because we have aimed to achieve similar anesthesia target level of approximately T6 in all of our patients. As a result, similar hemodynamic responses were seen independent of total dose administered or the technique of administration.

Our findings are consistent with Rooke et al.'s (17) data demonstrating that administration of hyperbaric

lidocaine in 30–50 mg decreased MAP by 29% compared to 16–23% after 50–75 mg in the present study. However, the cause of hypotension was somewhat different. Spinal anesthesia decreased both cardiac output and systemic vascular resistance, in almost equal proportion, in the present study but principally produced vasodilatation in the study by Rooke et al. (17).

In this study, we have chosen to make hemodynamic measurements at 3-min intervals during the induction of anesthesia using the thoracic electrical bioimpedance method. Although this method only estimates left ventricular SV and CO from the change in electrical bioimpedance accompanying cardiac ejection, it provides reliable trends of hemodynamic changes in a noninvasive and continuous manner (18,19). Correlation with the standard thermodilution method was strong (correlation coefficient 0.88) in the absence of aortic insufficiency, ventricular septal defect, and sepsis (20). For the purpose of this study, we thought that information about the trends of hemodynamic changes was sufficient and invasive technique of measurement was not warranted.

In summary, our comparison of the single-bolus and sequential-dosing techniques of local anesthetic (lidocaine) administration for continuous spinal anesthesia suggests that anesthetic efficacy is influenced by the choice of technique. Hemodynamic response appears to be influenced primarily by the level of sympathetic block. The use of a titrated regimen (incremental 25-mg doses) and a hyperbaric local anesthetic solution may permit the gradual and more controllable development of sympathetic, sensory, and motor block in some patients. In others, the onset and progression of blockade can be unpredictably rapid, similar to that with a single-bolus regimen, even at a small starting dose of 25 mg. The difference in onset and progression of effect in patients given the titrated regimen may be due to interindividual variability in susceptibility to local anesthetic. The use of a single-bolus regimen using a 27-gauge catheter resulted in a high degree of failure. Our data suggest that the failure of the single-bolus technique may have been due to anesthetic maldistribution resulting in a restricted sacral blockade.

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