Comparison of Lidocaine and Saline for Epidural Top-Up During Combined Spinal-Epidural Anesthesia in Volunteers

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This study was designed to determine the efficacy of saline as an epidural top-up to prolong spinal anesthesia during combined spinal-epidural anesthesia (CSEA). Eight volunteers received three separate CSEAs with intrathecal lidocaine (50 mg). After two-segment regression, each subject received either a saline (10 mL), lidocaine 1.5% (10 mL), or control sham (0.5 mL saline) epidural injection in a randomized, double-blind, triple cross-over fashion. Sensory block was assessed by pinprick and tolerance to transcutaneous electrical stimulation (TES) equivalent to surgical stimulation at the knee and ankle. Motor strength was assessed with isometric force dynamometry. Data were analyzed with a repeated measures analysis of variance and a paired *t*-test. Sensory block to pinprick was prolonged in the thoracolumbar dermatomes only by lidocaine ($P < 0.05$). Neither lidocaine nor saline prolonged the duration of tolerance to TES at the tested sites. Instead, saline decreased the duration of tolerance to TES by 20 and 24 min at the knee and ankle ($P < 0.05$). Recovery from motor block at the quadriceps was prolonged by an epidural injection of lidocaine ($P < 0.05$). We conclude that when 10 mL of epidural saline is administered after two-segment regression, it is an ineffective top-up and may decrease the duration of spinal anesthesia during CSEA.

(Anesth Analg 1997;84:574–7)

Combined spinal epidural anesthesia (CSEA) is gaining popularity as a regional anesthetic technique for obstetrical and ambulatory surgery (1,2). CSEA allows the use of low doses of spinal anesthetic, which provides quick onset and decreased patient recovery time (1,2). On the other hand, use of low doses of intrathecal local anesthetic may also result in required epidural supplementation, which may prolong recovery time. Previous studies have observed that an epidural injection of saline soon after the administration of an intrathecal local anesthetic can increase cephalad extent of the sensory block to pinprick (3,4). Such a practice may provide an increased duration of anesthesia without prolonging anesthetic recovery time. However, no study has examined effects of epidural saline during regression of spinal anesthesia, when an epidural top-up would be desirable. This study was designed to investigate whether epidural saline could prolong sensory and motor block during regression of CSEA.

Supported in part by a grant from B. Braun Medical, Inc.

Presented in part at the annual meeting of the International Anesthesia Research Society, March 8–12, 1996, Washington, DC.

Accepted for publication October 21, 1996.

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Methods

After institutional review board approval, and in complete accordance with the World Medical Association Declaration of Helsinki and Tokyo, informed consent was obtained from eight healthy volunteers (four male and four female). Subjects ranged in age from 28 to 42 years, in height from 140 to 162 cm, and in weight from 55 to 80 kg. Each subject received three CSEAs in a randomized, double-blind, triple cross-over fashion. Anesthetics were separated by at least 5 days in each subject.

Subjects were given nothing by mouth for 6 hours prior to each session and voided immediately before administration of CSEA. Lactated Ringer's solution was administered 6 mL · kg^{-1} · min^{-1} during the 15 min prior to CSEA, followed by 8 mL · kg^{-1} · h^{-1} for the first h, then 2 mL · kg^{-1} · h^{-1} thereafter. With subjects in the left lateral decubitus position, the epidural space was identified at the L2-3 interspace by loss of resistance using a commercially available combined spinal-epidural kit (Espocan®, B. Braun Medical, Inc., Bethlehem, PA). No more than 0.5 mL of saline was injected into the epidural space at loss of resistance. A 27-G Sprotte needle was then placed through the epidural Espocan® needle with the orifice of the Sprotte facing cephalad. After aspiration of

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0.2 mL cerebrospinal fluid, 50 mg lidocaine (3.33 mL 1.5% plain lidocaine) was injected at a rate of approximately 0.25 mL/s. After intrathecal injection, an epidural catheter was placed 3 cm into the epidural space and secured. No injection was made through the epidural catheter at this time. Subjects were then placed supine and remained level for the duration of the study. All subjects were monitored with a noninvasive blood pressure cuff every 5 min and with pulse oximetry for the duration of the anesthetic. At two-segment dermatomal regression to pinprick, subjects received either 10 mL saline, 10 mL lidocaine (1.5%), or control sham (0.25 mL saline) injections via the epidural catheter.

Transcutaneous electrical stimulation (TES) was used to assess sensory block as previously described (5). TES leads were placed bilaterally at L2-3 (medial aspect above knee) and L5-S1 (lateral aspect above ankle). Baseline tolerance to TES was assessed prior to any anesthetic and then every 10 min after induction of spinal anesthetic until return to baseline. Testing was for 5 s of 50 Hz tetanus initially at 10 mA and then in 10-mA increments up to 60 mA with a commercially available nerve stimulator (Fisher & Paykel, Auckland, New Zealand). Tetanic stimulation with 60 mA for 5 s at 50 Hz has been previously demonstrated to approximate surgical incision (6,7). Each TES location was tested in a systematic order, moving from distal to proximal sites. In addition, dermatomal levels to pinprick (18-G needle) was assessed every 5 min after injection of spinal solution until recovery of pinprick at S2.

A commercially available isometric force dynamometer (Micro FET, Hoggan Health Industries, Draper, UT) was used to assess 5-s isometric maximal force contraction of the right quadriceps and gastrocnemius, as previously described (5). Measurements were performed at baseline and then every 10 min after injection of spinal anesthesia until return to 90% of baseline. Measurements were performed in triplicate and then averaged at each measurement period.

All subjects received a standard fluid infusion as outlined above. Subjects attempted to void when level of pinprick reached dermatomal level S2. If subjects were unable to void immediately, repeat attempts were made every 15 min, and time from injection of spinal solution was recorded.

Differences in dermatomal level of sensory block to pinprick and in motor block were assessed with repeated measures analysis of variance followed by *post hoc* testing with Fisher's protected least significant difference test. Durations of tolerance to TES and time to void were analyzed with a paired *t*-test with Bonferroni correction for multiple comparisons. A value of $P < 0.05$ was considered significant.

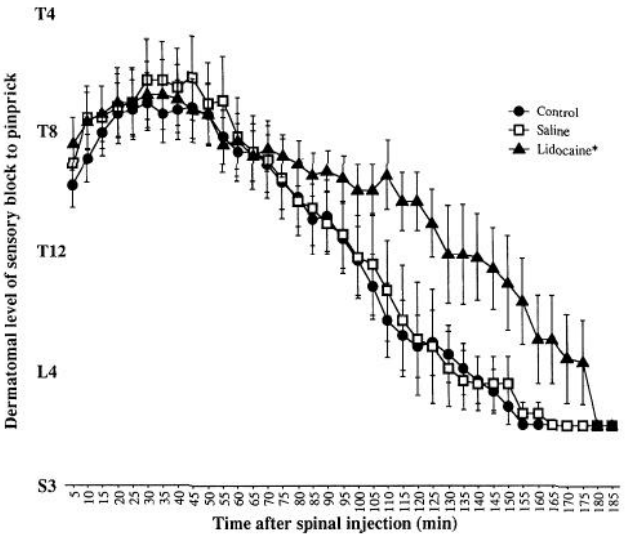

Figure 1. Time course of cephalad dermatomal level of sensory block to pinprick. * = different from control and saline epidural injections ($P < 0.05$).

Results

Epidural lidocaine prolonged sensory block to pinprick in the thoracolumbar dermatomes by an average of 28 min ($P < 0.05$) compared with saline and control sham injection (Figure 1, Table 1). Epidural injection of saline did not prolong sensory block to pinprick compared with sham injection (Figure 1, Table 1). Neither epidural lidocaine nor saline prolonged the duration of tolerance to TES at the knee or ankle. Instead, epidural saline decreased the duration of tolerance to TES at the knee by 20 min and at the ankle by 24 min compared with sham injection ($P < 0.05$, Table 1). Epidural lidocaine prolonged recovery from motor block in the quadriceps compared with saline and control but did not prolong motor block in the gastrocnemius (Figure 2). Epidural saline did not affect motor block. All subjects were able to void immediately after regression of sensory block to pinprick to dermatome S2.

Discussion

A means of prolonging initial spinal anesthesia during CSEA without prolonging recovery time would be desirable. As cephalad extension in sensory block to pinprick occurs from epidural injection of saline soon after induction of spinal anesthesia (3,4), epidural top-up with saline could perhaps prolong anesthesia without prolonging recovery. However, our data indicate that epidural saline is an ineffective top-up when administered during the regression of initial spinal anesthesia. This finding would seem to contradict previous studies by Blumgart et al. (3) and Stienstra et al. (4). These investigators observed cephalad

Table 1. Sensory Block to Pinprick and Transcutaneous Electrical Stimulation

	Control	Saline	Lidocaine
Sensory block to pinprick			
Time to peak block height (min)	21.6 ± 15.0	23.81 ± 9.2	21.9 ± 14.1
Two-segment regression (min)	64.4 ± 18.0	61.3 ± 15.8	67.5 ± 23.3
Regression to S2 (min)	128.1 ± 22.7	125.0 ± 23.2	156.9 ± 20.9*
Duration of tolerance to TES			
Knee (min)	77.5 ± 31.5	57.5 ± 38.2*	83.7 ± 27.7
Ankle (min)	85.0 ± 31.6	61.2 ± 43.9*	98.7 ± 24.7

Values are mean ± SD.
Dermatomal heights are median (interquartile range).
TES = transcutaneous electrical stimulation.
* = different from control ($P < 0.05$).

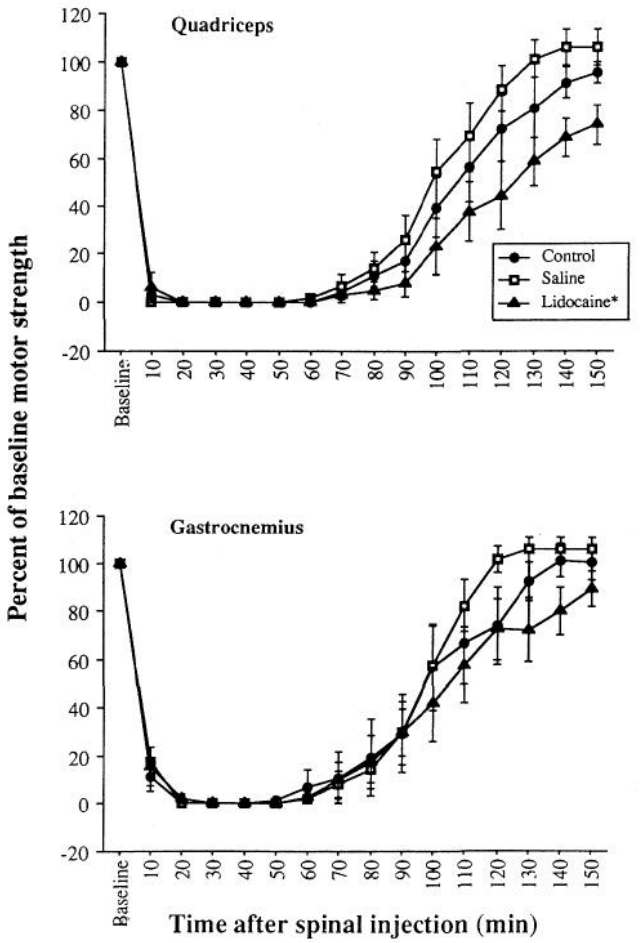

Figure 2. Time course of motor block at quadriceps and gastrocnemius muscles. * = different from control and saline epidural injections ($P < 0.05$).

extension of plain bupivacaine spinal anesthesia from epidural injection of saline five minutes after spinal injection in parturients (3) and during plateau of spinal anesthesia in orthopedic patients (4). The underlying mechanism of extension of spinal anesthesia by epidural saline was theorized to be increased distribution of intrathecal local anesthetic due to dural compression from epidural saline (8). However, our time of epidural injection was both temporally and functionally later than both of these previous studies. It is possible that by the time of regression of spinal anesthesia, intrathecal local anesthetic had "fixed" to neural tissues and was thus unaffected by changes in epidural volume and pressure. Although the most effective time to activate the epidural portion of CSEA has not been fully determined, previous studies have investigated the timing of epidural top-up doses during continuous epidural anesthesia and suggest that two-segment regression to pinprick is a reasonable time (9). Therefore, our selection of timing for injection of an epidural top-up at two-segment regression of spinal anesthesia more closely reflects clinical practice than top-ups at five minutes after spinal injection or during plateau of spinal anesthesia.

Another difference in our study was the use of intrathecal lidocaine. Both previous studies (3,4) used plain bupivacaine for spinal anesthesia, which has been previously observed to undergo cephalad extension after positional changes even 115 minutes after spinal injection (10). This finding suggests that plain bupivacaine does not fix in the neural tissues for a prolonged period after spinal injection. Unfortunately, no data are available regarding late cephalad extension of spinal anesthesia after plain lidocaine, and it remains unclear what period of time passes before fixing of lidocaine. Thus, intrinsic differences in blocking characteristics between spinal lidocaine and bupivacaine may also explain our differing findings. Our use of lidocaine rather than bupivacaine may be more clinically relevant for ambulatory surgery due to lidocaine's popularity as a short-acting spinal anesthetic (2).

Although analgesia to pinprick is a common clinical measure of sensory block after regional anesthesia, its relevance to surgical anesthesia is unclear. Previous studies examining minimum alveolar anesthetic concentration of volatile anesthetics have determined that transcutaneous electrical stimulation with 60 mA of five seconds of tetanus at 50 Hz approximates skin incision (6,7). Thus, duration of tolerance to TES provides a noninvasive model of the duration of surgical anesthesia. As epidural saline did not affect sensory

block to pinprick, it is not surprising that the duration of tolerance to TES was not prolonged. In fact, the injection of epidural saline decreased the duration of tolerance to TES at both the knee and the ankle. A previous study (11) noted decreased duration of motor block from epidural anesthesia after epidural injection of saline and postulated a "washing out" or dilution of local anesthetic from the epidural space. As there is minimal flux of epidural solution across small-gauge dural punctures (12), it is unlikely that dilution is the mechanism behind the decreased efficacy of intrathecal local anesthetic. Therefore, different mechanisms of action probably apply to the effects of epidural saline on intrathecal local anesthetic compared with epidural local anesthetic.

We propose two possible mechanisms for antagonistic effects of epidural saline on intrathecal local anesthetic. First, compression of the intrathecal space by increased epidural pressure may have diluted any remaining local anesthetic in the cerebrospinal fluid to subanesthetic levels. Second, the increased epidural pressure may have increased uptake of local anesthetic into the spinal cord vasculature and thus hastened clearance. Further studies are needed to determine exact mechanisms.

Somewhat surprisingly, 150 mg epidural lidocaine also did not prolong duration of tolerance to TES at either the knee or the ankle. If our electrical model of surgical stimulation is applicable, this finding suggests that this is an inadequate dose of local anesthetic for prolongation of surgical anesthesia. Although a Type II error is possible, it seems likely that relatively large doses of epidural local anesthetic are needed for top-up during regression of spinal anesthesia due to the segmental nature of epidural anesthesia and the antagonistic effects of epidural injection on the initial spinal anesthetic.

We used isometric force dynamometry to quantify motor block during the course of CSEA. Isometric force dynamometry has been previously determined to be a reproducible and sensitive measure of motor strength after spinal and epidural anesthesia (5,13). Motor block in both the quadriceps and the gastrocnemius were unaffected by the epidural injection of saline, although the intensity of the sensory block was decreased (decreased duration of tolerance to TES). This discrepancy in the effects of epidural saline on sensory and motor block is difficult to explain. We speculate that the lipid-rich myelin sheath of motor fibers may have augmented intrathecal local anesthetic binding and counterbalanced the increased clearance by injection of epidural saline. Alternatively, intrinsic differences in local anesthetic action on A and C fibers may also explain our observation (14). Again, further studies are needed to determine the mechanisms behind these differences in sensory and motor block. Not surprisingly, lumbar (L2-3) epidural injection of lidocaine prolonged the motor block of the quadriceps and not of the gastrocnemius. As the quadriceps are innervated by the L2-3 nerve roots and the gastrocnemius by the S1-2 nerve roots, our observation is consistent with the segmental nature of epidural anesthesia. Previous studies have also noted difficulty in blocking large sacral nerve roots with lumbar epidural anesthesia (15).

In conclusion, 10 mL of epidural saline, when administered after two-segment regression, is an ineffective top-up and may decrease duration of initial spinal anesthesia during CSEA. Epidural top-up with 150 mg of lidocaine prolonged sensory block to pinprick and motor block at the quadriceps but may be insufficient to prolong surgical anesthesia.

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