The Efficacy of 1.5% Lidocaine with 7.5% Dextrose and Epinephrine as an Epidural Test Dose for Obstetrics

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Recent reports concerning local anesthetic toxicity, especially neurotoxicity with chloroprocaine solutions and cardiotoxicity with bupivacaine, have rekindled interest in the routine use of a test dose during epidural anesthesia. Because the test dose must allow accurate and rapid determination of subarachnoid or iv injection of the needle or catheter, we sought to determine whether the subarachnoid block obtained with 1.5% lidocaine in 7.5% dextrose can be differentiated easily from epidural block and whether the addition of 15 µg epinephrine to the test dose aids in detection of unintentional iv injection.

We studied two groups of obstetric patients. In Group 1, 1.5% lidocaine in 7.5% dextrose with 15 µg epinephrine was used as an epidural test dose. In Group 2, 1.5% lidocaine in 7.5% dextrose was administered as a spinal anesthetic.

METHODS

This study was approved by the Human Investigation Committee at our institution.

Group 1: Epidural. An epidural catheter was inserted through a Tuohy needle at the L2–3 or L3–4 interspace in 250 obstetric patients requesting epidural anesthesia. No drugs were administered through the epidural needle. Aspiration tests were performed after the epidural catheter was taped in place. No cerebrospinal fluid (CSF) was aspirated in any patient. If blood was aspirated, the catheter was either withdrawn until no blood appeared or reinserted at another interspace. The test dose solution then was injected through the catheter. The first 150 patients received 3 ml of 1.5% lidocaine with 7.5% dextrose and 15 µg epinephrine. The next 100 patients received 2 ml of 1.5% lidocaine with 7.5% dextrose and 15 µg epinephrine. The test dose solution was made by adding 15 µg epinephrine (in a 0.1 ml volume) to commercially available 1.5% lidocaine for spinal anesthesia, which already includes 7.5% dextrose (Astra Pharmaceutical Products, Worcester, Massachusetts). The epinephrine solution from which 0.1 ml was taken was made by adding 2 ml 1:1,000 epinephrine to 11 ml perservative-free normal saline. At the time of the test-dose injection, the head of the bed was elevated 10 degrees, and all patients were in left lateral decubitus position or supine with 15 degrees left uterine tilt using a wedge. If the patient was in labor, the anesthesiologist waited until just after a contraction to inject the test dose. This was done so that the likelihood of a painful stimulation causing an increase in heart rate coincident with injection of the test dose would be minimized. Maternal heart rate was monitored continuously by palpation of the radial pulse or by ECG monitoring for 2 min following injection, and fetal heart rate was monitored by continuous transabdominal doppler ultrasound or fetal scalp electrode.

Objective sensory loss was tested by pinprick at 1-min intervals over all lower thoracic, lumbar, and sacral dermatomes bilaterally. The time to onset of objective sensory loss to pinprick was recorded for each patient, as well as the dermatome where that loss first occurred. The maximum change in maternal heart rate during the first minute following injection, as well as changes in fetal heart rate or pattern following injection, also were noted. The number of dermatomes blocked at 20 min following injection was recorded. If a dermatome was blocked unilaterally, then it was counted as 0.5 dermatomes for the purpose of calculating the total number blocked. If no evidence of block had developed by 20 min, and there had been no increase in maternal heart rate immediately following the test dose, then the test dose was repeated. If there was neither evidence of block nor an increase in maternal heart rate after the second test dose, the epidural catheter was replaced at another interspace or an alternate anesthesia technique was used.

The mean time to onset of objective sensory loss to pinprick and the mean number of dermatomes blocked at 20 min following epidural injection were determined. The mean number of dermatomes blocked with a 2-ml volume was compared with a 3-ml volume.

Group 2: Subarachnoid. Spinal anesthesia was administered to 15 patients requesting spinal anesthesia for ob-

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stetric procedures. A 25-gauge spinal needle was inserted at L2–3 or L3–4 in the left lateral decubitus position with the head of the bed elevated 10 degrees; 1.5% lidocaine, 2 ml in a 7.5% dextrose solution (Astra Pharmaceutical Products, Worcester, Massachusetts) was used as the spinal anesthetic solution. All patients immediately were turned supine (with a 15-degree left tilt using a wedge) and the head of the bed was elevated 10 degrees. Following the injection, the patients were tested at 15-s intervals for sensory loss to pinprick at the S-2 dermatome. The time to onset of sensory loss at S-2 and the level of anesthesia obtained 20 min following the block were recorded.

All results were expressed as mean ± SEM. The Student's t test and Mann-Whitney test were used for statistical analyses. A P value ≤ 0.05 was considered significant.

RESULTS

There were no differences in gestational age, height, and weight between Groups 1 and 2 (table 1). In Group 1, of the 250 patients who received the epidural test dose, 232 developed an objective sensory block by 20 min. In these 232 patients, mean onset time of objective block was 8.82 ± 0.22 min, and only one patient demonstrated objective sensory loss before 4 min (fig. 1). This patient had evidence of objective sensory loss at only the L-2 dermatome at 3 min following injection. The volume of solution did not influence the number of dermatomes blocked (3.47 ± 0.17 vs. 3.69 ± 0.16, 2 vs. 3 ml, P > 0.05). In patients who demonstrated objective sensory block, the mean maternal heart rate before injection was 84. The average change in heart rate in the first minute following injection was +1 beats/min (range −20 to +17).

Eighteen patients had no sensory block by 20 min. These included eight patients who had both a transient increase in maternal heart rate during the first minute and no evidence of block by 20 min. In these eight patients, mean maternal heart rate rose from 76 ± 2 to 109 ± 6, and the range of increase was +24 to +55 beats/min. These were presumed to be iv injections. In these eight patients, fetal heart rate traces showed no evidence of fetal distress (i.e., persistent tachycardia, bradycardia, or loss of beat-to-beat variability) immediately following injection of the test dose. No other patients subsequently demonstrated signs of local anesthetic toxicity suggestive of undetected iv placement of the epidural catheter. Of the remaining 10 patients, three required a second epidural test dose to establish objective sensory block to pinprick. Seven patients had neither objective evidence of block nor an increase in heart rate following the second test dose, and the catheters were presumed not to be in the epidural space, although iv placement could not be ruled out absolutely.

Group 2: Subarachnoid. Mean time to onset of objective sensory block at S-2 was 1.45 ± 0.12 min. All patients developed objective sensory block by 2 min following injection (fig. 1). The average cephalad spread of block was T9 ± 1.

Mean time to onset of block at the S-2 dermatome following subarachnoid injection of the test dose was significantly shorter than mean time to onset at any lumbar, thoracic, or sacral dermatome following epidural injection (Group 2 vs. Group 1, 1.45 ± 0.12 vs. 8.82 ± 0.22 min, P < .001).

DISCUSSION

Although epidural anesthesia has become increasingly popular, there is increasing concern about local anesthetic toxicity during its administration. Several ways to avoid serious toxic reactions and complications have been described. These include 1) fractionation of the epidural dose into aliquots (usually 5 ml)² with careful observation for prodromal signs of local anesthetic toxicity after each aliquot; 2) careful aspiration of the epidural catheter or needle before injection of an epidural dose; and 3) the routine use of a test dose before injection of any local anesthetic through an epidural needle or catheter.⁷,⁸ Al-

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**Table 1. Gestational Age, Height, and Weight (mean ± SEM)**

<table>
<thead>
<tr>
<th>Group 1 (epidural)</th>
<th>Group 2 (spinal)</th>
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</thead>
<tbody>
<tr>
<td>(n = 250)</td>
<td>(n = 15)</td>
</tr>
<tr>
<td>Gestational age (wk)*</td>
<td>39.3 ± 0.2 (38-43)</td>
</tr>
<tr>
<td>Height (m)*</td>
<td>64.0 ± 0.2 (56-72)</td>
</tr>
<tr>
<td>Weight (kg)*</td>
<td>75.1 ± 0.9 (54-162)</td>
</tr>
</tbody>
</table>

* P > 0.05, Group 1 versus Group 2.

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**Fig. 1.** Time to onset of objective sensory loss (to pinprick) following epidural (Group 1) and spinal (Group 2) administration of a hyperbaric 1.5% lidocaine solution.
though all of these suggestions are theoretically important, when used alone none are completely safe and reliable.

The careful observation of patients who receive local anesthetics via any route is mandatory. However, during attempted epidural anesthesia, significant volumes or mass of drug may be accidentally injected iv before a toxic reaction or complication is detected by the anesthesiologist. This may especially be true in a sedated patient. As little as 30 mg of bupivacaine, for example, can result in a serious toxic reaction when injected iv. Moore and Batra point out that pharmacokinetic properties of the amide local anesthetics, namely the cumulative effect on plasma level of incremental doses, may make fractionation hazardous if it is the only technique used. In addition, the mass of local anesthetic required to detect iv injection may be excessive if administered accidentally subarachnoid or if more than the optimal amount required for the intended epidural block (i.e., obstetric analgesia with 0.125% bupivacaine). Reliance on aspiration of the catheter is also not an entirely dependable way of avoiding subarachnoid or iv injection. In our series of 250 catheter insertions, eight iv catheters were undetected by aspiration alone. Carr and Hehre have reported the unintentional placement of intrathecal catheters through which CSF could not be aspirated, but which were detected by the use of a test dose.

The routine use of a test dose is now being recommended. The components of such a test dose, however, continue to be controversial. Most authors agree that a test dose should contain a local anesthetic to provide readily detected evidence of sensory blockade if injected into the subarachnoid or epidural space. We feel the anesthesiologist should be familiar with the local anesthetic used, both as a spinal and as an epidural drug, so that epidural injection can be differentiated from subarachnoid injection. Lidocaine in a 1.5% concentration has been available for many years as both a spinal agent (with 7.5% dextrose) and epidural anesthetic agent and therefore would be a logical choice as the local anesthetic to be used in the test dose. Chloroprocaine is not used as a spinal local anesthetic, and procaine and pontocaine are not widely used as epidural local anesthetics.

Bupivacaine is marketed commercially both as a spinal local anesthetic and as an epidural test dose. The spinal solution is 0.75% bupivacaine in 8.25% dextrose and has become available only recently for use on a wide scale. The epidural test dose solution is 0.5% bupivacaine (without dextrose) with 1:200,000 epinephrine. Therefore, one must inject 15 mg bupivacaine to include 15 μg epinephrine in a test dose. Fifteen milligrams of bupivacaine may be excessive if administered subarachnoid, especially in the obstetric patient.

The use of a hyperbaric local anesthetic solution will result in a more controlled level of anesthesia when administered in the subarachnoid space. Therefore, if a test dose solution is hyperbaric, cephalad spread could be limited following unintentional subarachnoid administration by keeping the head of the patient elevated. Our study demonstrates that subarachnoid injection of 1.5% lidocaine in 7.5% dextrose results in both a rapid onset of perineal sensory loss and a mean cephalad spread limited to T9 ± 1 in obstetric patients.

The test dose also should indicate iv injection. Depending on the mass of local anesthetic injected and the rapidity of injection, the local anesthetic itself may or may not provide prodromal evidence of central nervous system toxicity. For this reason, another drug that can provide evidence of iv injection was included in our test dose. Fifteen milligrams of epinephrine, when administered iv, has been shown to consistently result in an increase in heart rate in patients who are not receiving beta-adrenergic blocking drugs and has been recommended for inclusion in epidural test doses in both nonobstetric and obstetric patients. In our study, eight iv catheters (that were not detected by aspiration) were detected by a transient increase in maternal heart rate within the first minute following test dose administration.

Finally, the test dose volumes should be the minimum necessary, so that if subarachnoid injection does occur, the spread will be limited. In our study, a 2-ml volume provided good evidence of both spinal and epidural anesthesia. Thus, the use of larger volumes appears unnecessary, and even smaller volumes may be useful, although this was not evaluated in our study.

In obstetric patients, the subarachnoid injection of 2 ml of 1.5% lidocaine in 7.5% dextrose resulted in perineal (S-2) sensory block within 2 min. This block was easily differentiated from the slower onset of sensory loss observed following epidural injection. In addition, unintentional iv injections of the test dose were detected by transient increases in maternal heart rate following administration of the test dose containing 15 μg epinephrine. We conclude that 2 ml of 1.5% lidocaine in 7.5% dextrose with 15 μg epinephrine will aid the differentiation between subarachnoid, epidural, and iv injection. Therefore, this combination can be used in a routine protocol that renders obstetric epidural anesthesia probably safer. That protocol should contain the “tried” of fractionation, observation, and, of equal importance, the use of an appropriate test dose before all epidural blocks.

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REFERENCES
Neurolytic Lumbar Sympathetic Block in the Treatment of Raynaud’s Phenomenon

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Raynaud’s phenomenon is described as episodic, cold-induced pallor and cyanosis with subsequent suffusion and erythema. The paroxysmal vasospasm of the distal extremities characterizes Raynaud’s disease if it occurs alone, or Raynaud’s phenomenon if associated with other connective-tissue or autoimmune disorders. Progressive systemic sclerosis (PSS), or scleroderma, is a connective tissue disease that presents with variable severity as a multisystem disease. The nonpitting tautness involving hands, forearms, and face is accompanied by involvement of the gastrointestinal, pulmonary, cardiovascular, renal, and musculoskeletal systems. Raynaud’s occurs in 90% of patients with the skin changes of scleroderma.1

The many therapeutic measures used to treat the paroxysmal vasospasm have met with variable success. These modalities include beta-adrenergic blockers, arterial vasodilators, intraarterial reserpine, iv guanethidine and reserpine, and renin-angiotensin inhibitors.2

Although neurolytic sympathetic blockade is effective in the treatment of rest pain in patients with obliterator arterial disease of the lower limbs,3,4 no published data are available for similar success in patients suffering from pain and ischemic changes in the lower limbs from Raynaud’s phenomenon. One reason may be that Raynaud’s symptoms are almost always limited to the hands.5

We describe a case of a patient with known PSS presenting with severe Raynaud’s phenomenon and compromise of toe perfusion with cyanosis and ulceration, who was treated with bilateral lumbar sympathetic phenol blocks after diagnostic lumbar epidural anesthesia.

REPORT OF A CASE

A 69-year-old man with PSS diagnosed 2 yr previously, with skin changes over the arms and hands, esophageal obstruction requiring numerous dilatation procedures, interstitial pulmonary fibrosis, and polyarthralgias, had symptoms consistent with Raynaud’s phenomenon of the feet. Complaints included pain brought on by cold, with color changes from white to blue involving the toes but not the fingers. Symptoms had progressed over the previous 4 weeks. Physical examination revealed superficial ulcerations of the lateral two toes of the right foot. The involved toes demonstrated poor capillary refill of greater than 20 s, were cool to the touch, and had a dark, cyanotic appearance. He was taking penicillinamide 250 mg po tid, cimetidine 300 mg po qid for reflux symptoms, and nifedipine 20 mg po qid to promote vasodilatation.

A diagnostic lumbar epidural block was performed to assess the role of reversible vasospasm. The block was performed via a catheter at L2-3, using 10 ml of a 0.5% solution of lidocaine, which resulted in a T-11 level by sensation of temperature. Skin temperature was measured with thermistor probes on each great toe. Although an adequate sympathetic block was expected, temperature did not change more than 0.5° C for 60 min, and he was discharged from the clinic when the block was regressing. However, at the 2-h postblock evaluation, the patient had good relief of pain, and both feet were noticeably warmer to the touch, with improved capillary refill of 12 s. Skin temperatures were not taken, but skin plethysmography demonstrated improved flow volume in the pedal cutaneous circulation bilaterally.

Because of the favorable short-term response, the patient returned 1 week later for a diagnostic and possible neurolytic sympathetic block. On return visit, he had minimal pain relief, and time for capillary refill had increased to greater than 20 s. Needles were inserted for bilateral sympathetic block at L-3, their position confirmed by cross-table ra-