

by sinuvertebral nerves,¹⁷ which may cause local and referred pain if stimulated.^{18, 19} However, in some cases, the disc may be penetrated without pain. In addition, local anesthetic was infiltrated during passage of the needle, which should have reduced the sensitivity of the annulus. In the setting of celiac plexus block, however, disc injection is a potentially serious situation because of the continuity that may exist between a degenerated disc and the vertebral canal. A degenerated disc can offer minimal resistance to injection, and injection might also not cause pain. On the contrary, a small test dose of local anesthetic could produce pain relief if some spread *via* the disc to the epidural space, falsely indicating correct needle placement. If, in the belief that the needle is in the correct position, the full neurolytic dose of alcohol should then be injected, paraplegia could result from the neurolytic agent spreading into the epidural space.

These cases provide further support for the practice of radiologic control in real time during celiac blockade.

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Subdural Block during Attempted Caudal Epidural Analgesia for Labor

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Although subdural blocks following attempted lumbar epidural injections are well documented,¹⁻⁹ there are no reports of suspected subdural injections following attempted caudal epidural anesthesia. We describe a case

of unexpected, delayed high motor block with prolonged regression following attempted caudal epidural analgesia in a patient in labor.

CASE REPORT

A 27-yr-old, gravida 2 para 1 patient at term gestation was admitted in labor. She weighed 73 kg and was 178 cm tall. Her review of systems

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as well as her prenatal course were unremarkable except for a history of mitral valve prolapse. She had no history of chronic back pain, back injury, or prior surgery. Her physical examination was normal. When her cervix was completely effaced and 6 cm dilated, she requested pain relief and was evaluated for epidural analgesia. Because she had had a prior normal vaginal delivery without a prolonged second stage, and this infant was judged not to be significantly larger than her first, it was elected to proceed with caudal epidural analgesia.

Monitors used during initiation of the caudal anesthesia included noninvasive blood pressure, ECG, continuous fetal heart monitoring, and tocodynamometry. The patient received 30 ml 0.3 M sodium citrate orally and a 500-ml intravenous bolus of lactated Ringer's solution prior to positioning for the block. The patient was placed in a modified left lateral decubitus (Sims') position; the sacral hiatus was easily identified; and the presacral area cleaned with iodophor and draped. The skin overlying the sacral hiatus was anesthetized with 1% lidocaine, and an 18-G Tuohy needle was placed through the sacrococcygeal ligament, redirected cranially, and advanced 1.0 cm into the caudal canal with no return of blood or cerebrospinal fluid. Five milliliters of saline injected through the needle gave no evidence of subcutaneous injection. A 20-G open-tip Teflon® catheter (Abbott, Chicago, IL) with the stylet withdrawn 2.5 cm was passed easily through the needle for a distance of 4 cm, when slight transient resistance was felt. An additional 2 cm was advanced so that the tip would lie high in the sacral canal to provide first-stage pain relief. No blood or cerebrospinal fluid could be aspirated through the catheter. The patient's blood pressure was 115/55 mmHg. The patient received a test dose of 3 ml 1.5% lidocaine with 1:200,000 epinephrine. There was no change in blood pressure or increase in heart rate within 2 min, nor was there a sign of sensory block in the L2-L3 dermatomes at 3 min after injection of the test dose. Lacking any evidence of subarachnoid or intravenous injection of the test dose, 20 ml 0.25% bupivacaine was given in 5-ml increments over 2 min.

Within 5 min the patient noted paresthesia in both legs. Thirty-five minutes later, the patient was comfortable, with a T1 sensory level. One hour after injection, relative hypotension occurred (95/36 mmHg), which responded to one dose of 10 mg intravenous ephedrine. A total of 2 l crystalloid was infused. The patient's sensory anesthesia level continuously progressed cephalad (see table), and by 1 h and 11 min following injection, a C2 sensory level was evident, with a decrease in hand strength and loss of ability to vocalize except in a whisper. The patient was not dyspneic. Her cranial nerve sensation was normal. However, by 1 h and 25 min all hand strength was lost, and it was elected to protect the airway and assist with ventilation. The trachea was intubated following intravenous administration of 200 mg sodium thiopental and 40 mg succinylcholine. She awakened within 10 min, began spontaneously breathing, and delivered vaginally minutes later. The 4.0-kg infant was vigorous, with Apgar scores of 7 at 1 min and

9 at 5 min. Umbilical arterial blood gas values were: pH 7.11, P_{CO_2} 77 mmHg, and P_{O_2} 21 mmHg. Umbilical venous gases were 7.22, 68 mmHg, and 29 mmHg, respectively. Simultaneous maternal P_{ETCO_2} was 33 mmHg.

The patient's motor strength gradually improved, and her trachea was extubated 55 min after intubation, at which point her hand grasp returned and she had a negative inspiratory force of 25 cmH₂O. Six hours and 35 min after the initial dose, her sensory level was L3, and at 7 h and 45 min, S1. Eight hours and 50 min after the bupivacaine dose, the patient was able to lift her hips off the gurney, and she was discharged from labor and delivery to the postpartum ward. The patient was ambulating the following day with no headache and was discharged on the second postoperative day without sequelae.

DISCUSSION

Lubenow and colleagues describe a method to diagnose a subdural block,² including two major criteria (negative aspiration test and unexpected widespread sensory blocks after epidural injection) and three minor criteria (delayed onset of 10 min or more of a sensory or motor nerve block; varying degrees of motor block occurring despite the use of low doses of bupivacaine; or sympatholysis out of proportion to the administered dose of local anesthetic). A subdural block is judged to exist if both major and one minor criteria are present and there is no evidence of a wet tap.²

Despite numerous case reports of subdural blocks meeting these criteria following attempted lumbar epidural anesthesia,^{1,4-9} there are no reports of such blocks following attempted caudal epidural anesthesia. This may be due to the relatively longer distance from the tip of a needle in the sacral hiatus to the end of the dural sac, as well as the relatively acute angle at which the catheter would contact the dural sac when advanced from the sacral hiatus. There was one report of delayed but sudden onset of a high sympatholytic block reported following the caudal application of local anesthetic and steroid¹⁰; however, that patient received a large (40-ml) volume of local anesthetic and had a history of prior chymopapain discolysis at L5-S1 which failed to resolve symptoms, and the authors believed that the case was due probably to an exaggerated epidural block and not to subdural spread.

Our patient met the criteria proposed by Lubenow *et al.*,² suggesting that our patient had a subdural spread of the local anesthetic. She had no history of back problems. There was a negative aspiration test, an unexpectedly wide spread for a local anesthetic volume of 20 ml, a delayed onset (and therefore not subarachnoid spread), and a profound motor block for 0.25% bupivacaine. To our knowledge, this is the first reported case of an attempted caudal epidural anesthetic resulting in a subdural block in a patient without prior back pathology.

When passing the catheter in our patient, we felt a transient resistance at a depth of insertion that corresponds to the location of the caudal end of the dural sac.

TABLE 1. Progression of Sensory and Motor Block Following Caudal Injection of 20 ml 0.25% Bupivacaine

| Time from Initial Dose (min) | Sensory Level | Motor Strength | |
|------------------------------|---------------|--------------------------|-----------|
| | | Right Hand | Left Hand |
| 35 | T1 | 5/5 | 5/5 |
| 71 | C2 | 3/5 | 3/5 |
| 85 | C2 | 0/5 | 0/5 |
| 155 | C2 | 1/5 | 3/5 |
| 165 | C2 | 2/5 | 4/5 |
| 395 | L3 | | |
| 465 | S1 | | |
| 520 | | Wiggles toes, lifts hips | |

Indeed, subdural catheter passage was probably the result of dural puncture and further advancement of the catheter past this point. Our case provides evidence that an epidural catheter itself and not just the needle can cause dural puncture, since the needle was advanced only 1.0 cm past the sacrococcygeal ligament, well below the usual termination of the dural sac.

The importance of vigilant observation in the period of time following local anesthetic administration during epidural anesthesia cannot be overemphasized. This patient's block continued to spread cephalad well beyond the 20–30-min period during which the final sensory level is reached following normal epidural administration of bupivacaine. The accompanying decrease in blood pressure required prompt diagnosis and management to avoid adverse fetal effects. Likewise, potential respiratory and/or airway compromise demanded prompt treatment. The possibility of occurrence of a high block, even if judged to be remote, requires forethought and the availability of anesthesia care providers as well as the required instruments and drugs. Nursing personnel caring for such patients should also be aware that delayed high blocks can occur following attempted epidural analgesia, even *via* the caudal route.

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