

CORRESPONDENCE

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Acute Hypoglycemia following Combined Spinal-Epidural Anesthesia (CSE) in a Parturient with Diabetes Mellitus

To the Editor:—Intrathecal opioids, with or without local anesthetics, are commonly administered to parturients for analgesia in early labor. Women in labor have increased cortisol and epinephrine concentrations, which are known to stimulate hyperglycemia.¹ With the onset of pain relief from neuraxial analgesia, a significant decrease in the concentration of catecholamines has been shown to occur,² possibly preventing an increase in blood sugar.¹ We report a case of acute hypoglycemia after onset analgesia in a diabetic parturient after combined spinal-epidural anesthesia (CSE).

The patient was a 26-yr-old woman, gravida 6 para 4, at 38 weeks' gestation with class 2 gestational diabetes mellitus controlled by diet only. She had no other medical problems. She was admitted in active labor and requested regional anesthesia for labor and delivery. Her blood sugar concentration ranged from 94 to 121 mg/dl, and her blood pressure (BP) ranged from 118/70 to 126/76 mmHg throughout pregnancy. One hour before combined spinal-epidural anesthesia, her blood sugar concentration was 121 mg/dl. Her BP at this admission ranged from 138/68 to 146/72 mmHg, her heart rate ranged from 88 to 110 beats/min, and an electrocardiogram showed a normal sinus rhythm.

After hydration with 1,000 ml lactated Ringer's solution, the block was performed with the patient in the sitting position. A 17-gauge epidural needle was advanced into the epidural space at the L3-L4 interspace, and a 25-gauge pencil-point needle was passed through the epidural needle into the subarachnoid space. A mixture of 25 µg fentanyl and 1.25 mg plain bupivacaine was injected intrathecally, and the spinal needle was withdrawn. An epidural catheter was threaded into the epidural space to a depth of 3 cm. At this time, the patient told us that her contractions were no longer painful. Her BP and heart rate were unchanged. A test dose of 3 ml lidocaine, 1.5%, with 1:200,000 epinephrine was injected into the epidural catheter. Approximately 2 min after the test dose, there was a sudden increase in her heart rate from 110 to 138 beats/min; her BP remained unchanged. Although the delayed tachycardia did not fit the classic definition of a positive response to the test dose, we decided to replace the epidural catheter, which was accomplished easily with the patient still in the sitting position. While the second epidural catheter was being secured to her

back, the patient suddenly felt dizzy and became pale and diaphoretic and her BP decreased to 62/48 mmHg. No drug had been administered through the catheter at this time. The heart rate remained at 140-144 beats/min with sinus tachycardia. The patient was placed in the supine position and turned to her left side, and oxygen was administered via facemask. Fluids were infused rapidly, and a total of 50 mg ephedrine was administered intravenously in divided doses in 2 to 3 min. BP increased to 112/53 mmHg, but the patient continued to experience dizziness and "feeling faint." There was no evidence of motor block and sensory level to pin prick was at the T10 dermatome. At this time her blood sugar was 57 mg/dl. With rapid administration of 5% dextrose, blood sugar increased to 128 mg/dl, and all symptoms were alleviated. The epidural catheter was subsequently used to provide pain relief for labor for several hours, and she delivered a healthy infant with an Apgar score of 9 and 9 at 1 and 5 min.

Rapid onset of intense analgesia after CSE can decrease the BP to levels before the onset of painful contractions in parturients, but it seldom causes profound hypotension as in this patient. Decreased venous return resulting from aortocaval compression and sympathetic tone can also cause hypotension. In our patient, aortocaval compression was unlikely because the procedure was performed with the patient in the sitting position. With a sensory level of T10, the sympathetic block can extend a few segments higher, causing a decrease in the BP. However, even after the restoration of BP with fluids and ephedrine, the patient remained dizzy and diaphoretic. The symptoms disappeared with the rapid administration of intravenous dextrose. In this patient, blood sugar levels had been stable before CSE placement necessitating no insulin therapy. The cause of acute hypoglycemia and its relation, if any, to profound hypotension after CSE is unclear. We speculate that the abrupt decrease in the levels of catecholamines and cortisol associated with the rapid onset of analgesia from intrathecal opioids may have triggered these rare events in this patient.

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Positive Breath Alcohol Readings following "Generic" Propofol Administration

To the Editor:—In a previously described method,¹ the use of a breath alcohol analyzer combined with the addition of an ethanol marker to irrigation fluid allowed the detection of rapid fluid absorption during operative hysteroscopy. Our usual anesthetic technique for such procedures involves propofol induction and maintenance with periodic end-tidal breath alcohol determinations to detect the presence of ethanol from absorbed irrigation fluid. The device used to detect the ethanol marker is an Alco-Sensor III (Intoximeters, Inc., St. Louis, MO). This device analyzes discrete samples taken as needed from the patient's expired gas flow and uses fuel-cell technology together with an algorithm to provide a readout. This readout is calibrated to correspond with g/100 ml of blood ethanol and is approved by the U.S. Department of Transportation for evidentiary use. The device is not, however, specific for ethanol, and it will respond to other alcohols as well. Because the algorithm for producing the readout depends on the assumption that the alcohol measured is ethanol, it is not known how other substances relate to the device readout.

With the introduction of an alternative formulation of propofol, pharmacoeconomic considerations led to the use of the preparation manufactured by Gensia-Sicor (Irvine, CA). Shortly thereafter, it was noted that a patient undergoing hysteroscopic surgery showed positive breath alcohol levels before the start of surgery. This was initially attributed to alcohol use by the patient; however, more than 12 subsequent patients administered the Gensia-Sicor formulation were observed, and all were noted to have positive breath alcohol readings only after induction of anesthesia, but before surgery. It was also noted that discontinuing the propofol infusion and switching to sevoflurane eliminated the presence of positive readings and that the reinstatement of the infusion caused a reappearance of the positive readings. Further, positive breath alcohol readings were never present if the brand-name

propofol, Diprivan (Astra-Zeneca, Wilmington, DE), was used. The readout on the device after a standard induction dose of the Gensia-Sicor propofol was from 0.010 to 0.020 gm/100 ml, a reading that, if result of ethanol from fluid absorption, would cause discontinuation of the procedure. The response characteristics of the instrument during these circumstances are also curious. Upon taking a sample of known ethanol, the reading increases and stabilizes within a few seconds whereas when measuring the sample of a patient administered the Gensia-Sicor preparation, the reading increases over the course of as much as 1 min before stabilizing.

These findings have been discussed with the manufacturers of both the generic and the brand-name drugs, but, to date, neither has supplied an explanation. There are differences in the formulation of the two preparations, but no alcohols are added to the Gensia-Sicor formulation. Whatever the cause, further investigation of the source of the phenomenon is warranted because the use of breath alcohol sampling by fuel-cell-based instruments is unreliable in patients administered generic propofol.

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