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A Prolonged Chloroprocaine Epidural Block in a Postpartum Patient with Abnormal Pseudocholinesterase

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The rapid hydrolysis of 2-chloroprocaine by pseudocholinesterase usually limits its potential for toxicity in patients with normal enzyme. We describe a case involving a postpartum patient with abnormal pseudocholinesterase activity who had a prolonged chloroprocaine epidural block.

REPORT OF A CASE

A 31-year-old woman, G4, P3, AB1, was scheduled for a tubal ligation the day following an uneventful spontaneous vaginal delivery without anesthesia. No premedication was given prior to placement of an epidural needle in the L3–L4 interspace. Following negative aspiration for blood or CSF, a test dose of 5 ml 3 per cent chloroprocaine was administered. At this time, the patient reported that her "head felt numb." The needle was repositioned, 20 ml 3 per cent chloroprocaine was administered through the needle, and an additional 5 ml was administered through a cannula. Adequate sensory analgesia was obtained to the T5 level. Because the patient was unusually somnolent throughout the procedure, an intravenous injection of the test dose was suspected, and a blood sample was drawn to check chloroprocaine levels 15 min after the second dose (20 min after the test dose). However, the sample was inadvertently collected in a tube that did not contain a cholinesterase inhibitor and was left at room temperature. In addition, an abnormally long duration (approximately 3 hours) of the epidural block was noted in the recovery room.

Three and one half hours later, intra-abdominal bleeding was suspected and an exploratory laparotomy under general endotracheal anesthesia was scheduled. Ketamine was used for induction of anesthesia which was maintained with 70 per cent nitrous oxide. Succinylcholine was used to provide muscle relaxation (200 mg). The laparotomy revealed a hemoperitoneum and an active bleeding site in the right mesosalpinx. The estimated blood loss was 1500 ml and one unit of whole blood was administered intraoperatively.

Following completion of the operative procedure, the patient was unable to breathe spontaneously or respond to stimuli for a period of three hours. In addition, no twitch response could be elicited from a peripheral nerve stimulator. Low pseudocholinesterase levels were suspected and midway through the apneic period a blood sample was drawn correctly for drug and cholinesterase levels. The patient subsequently received two more units of whole blood during the first postoperative night. Serum for enzyme levels was drawn on postpartum days 1, 2, 6, 7, and at six weeks postpartum. Her postpartum course was also complicated by a paralytic ileus of 5 days duration. Her prepregnancy history included an appendectomy under general anesthesia with 60 mg succinylcholine. There was no documentation of prolonged apnea in the record from this surgery.

A plasma concentration of 5.5 ng/ml chloroprocaine was found in the tube without inhibitor taken 15 min after the second dose of chloroprocaine and 8.5 ng/ml in the sample taken correctly five hours later. Drug levels were measured by gas chromatography/mass spectrometry.[1] The initial sample was not collected correctly, but the level was still considered too low, in retrospect, to confirm a suspected intravenous injection.[2] The dibucaine numbers and pseudocholinesterase activity which are shown in table 1 were found to be exceptionally low. Cholinesterase activity and dibucaine numbers were determined according to Kalow and Genest.[3] The dibucaine number and cholinesterase activity at six weeks postpartum confirmed the presence of atypical pseudocholinesterase.

DISCUSSION

This case illustrates the effect of abnormal pseudocholinesterase on the metabolism and actions of chloroprocaine in a postpartum patient. The patient apparently could not adequately metabolize chloroprocaine. This may have led to excessive somnolence intraoperatively; normally there would be no excessive somnolence. Fur-

TABLE 1. Dibucaine Numbers and Pseudocholinesterase Activity

	Dibucaine Number	Enzyme Activity μm Acetylcholine/ml/h/37°C
Intraoperatively*	32‡	40‡
Postoperatively†	34 ± 5	62 ± 13
Six weeks postpartum	27	84

* Sample obtained after 1 unit of whole blood had been administered.

† Means ± SD of six samples obtained during postoperative days 1–7 and after two more units of whole blood were given during the first postoperative night.

‡ Normal values in our laboratory are: dibucaine number 70–80, and enzyme activity 125 ± 56 and 216 ± 69 μm Ach/ml/h/37°C for peripartum and nonpregnant patients, respectively.[4]

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thermore, it led to a prolonged chloroprocaine block; normally the block would last only 45–60 minutes. Finally, it led to detectable serum chloroprocaine five hours after administration; normally no chloroprocaine would be detectable 25–30 min following epidural anesthesia in peripartum patients.[4]

Abnormal pseudocholinesterase was verified in two ways. First, it was inadvertently verified by the prolonged apnea following subsequent succinylcholine administration. Second, her dibucaine number at six weeks postpartum was indicative of a homozygote for the atypical cholinesterase variant (E^aE^a) or a heterozygote with one gene for the atypical variant and one gene for the silent variant (E^aE^a).[5,6] Her dibucaine number in the immediate postoperative period although still abnormal, was probably higher due to the presence of active cholinesterase received from three units of transfused blood.[7]

We are not aware of other reports of unusual reactions to chloroprocaine in patients with abnormal or low cholinesterase. In fact, a lack of any sequelae in a pregnant patient who received an apparent inadvertent intravenous injection of chloroprocaine was reported recently.[2] However, the latter patient had enzyme levels appropriate for peripartum patients. This case illustrates that an abnormal reaction to chloroprocaine can occur in a patient with atypical cholinesterase.

This case also suggests than an abnormal reaction to chloroprocaine has clinical implications beyond a prolonged epidural block. An abnormal reaction to chloroprocaine should alert the anesthesiologist to the possibility of a pseudocholinesterase deficiency. This in turn, should suggest that the subsequent use of succinylcholine may result in a prolonged neuromuscular blockade.

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Right Atrial Catheter Placement: Use of a Wire Guide as the Intravascular ECG Lead

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Pre-operative insertion of a right atrial catheter is a widely accepted precautionary measure in patients at risk for intraoperative air embolus. Michenfelder *et al.* reported on the use of the intravascular ECG for positioning of the catheter precisely in the right atrium.[1] The author describes the use in 42 patients, of a J-tipped wire guide as the intravascular lead of the ECG.

Materials and Methods

An electrically isolated ECG monitor which had been tested previously for absence of leakage current was connected to the patient, and all other electrical devices were disconnected. Under sterile conditions, the right internal jugular vein was cannulated with a 19-gauge thin wall needle. A J-tipped wire guide was threaded into the vessel and the needle removed. A 12-inch 16-gauge Teflon®-coated catheter was passed over the wire guide a sufficient distance to ensure entry into the vein. The wire guide then was withdrawn slowly from the catheter until a sensation of resistance was felt as the J-tip impinged on the proximal end of the catheter. Alternatively, before insertion one could measure the length of wire guide extending from the catheter when the J-tip is in proper position.

The V lead of the ECG was connected with an alligator clip to the distal end of the wire guide at the edge of the sterile field (fig. 1). Then, while observing the ECG, the catheter and wire guide were advanced as a unit. The characteristic ECG changes have been described by Martin.[2] When the right atrium was entered,

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