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Plasma Levels of 2-Chloroprocaine and Lack of Sequelae Following an Apparent Inadvertent Intravenous Injection

To the Editor:—We recently reported measurable levels of 2-chloroprocaine in maternal plasma following epidural anesthesia during cesarean section and labor.¹ In that study the highest plasma levels found were less than 0.1 $\mu\text{g/ml}$ following a single dose. We now report a plasma level and clinical outcome following an apparent inadvertent intravenous injection of 2-chloroprocaine through an epidural catheter.

REPORT OF A CASE

A 23-year-old black woman, G5, P2, weighing 60 kg, was scheduled for repeat cesarean section with epidural anesthesia. As part of a research protocol, a heparin lock was inserted in a superficial vein and a premedication blood sample was drawn; plasma esterase activity was found to be normal for a pregnant patient. Using a 17-gauge Touhy needle, the epidural space was entered at the L₃-L₄ interspace. After a negative aspiration for blood and cerebrospinal fluid, 600 mg of 3 per cent 2-chloroprocaine were administered. Following this, no area of analgesia was detected. An epidural catheter was inserted, and over the next 30 min an additional 600 mg of 3 per cent 2-chloroprocaine were administered. Despite this, sensory analgesia was inadequate, and the operation was completed using general anesthesia. A normal 2,800-g infant, Apgar scores 7 at 1 min and 9 at 5 min, was delivered 58 min after the initial dose of 2-chloroprocaine. Ten minutes following the first dose of 2-chloroprocaine and prior to any subsequent dose, a blood sample was drawn, which was later analyzed for 2-chloroprocaine; the plasma level was found to be 17 $\mu\text{g/ml}$. In addition, a maternal blood sample drawn at delivery was found to have 0.33 $\mu\text{g/ml}$ 2-chloroprocaine, and the cord vein sample contained 0.016 $\mu\text{g/ml}$. Despite this apparent intravenous injection and subsequent high level of 2-chloroprocaine, no adverse sequelae, including central nervous system effects or changes in vital signs, occurred.

We believe that this is the first report of an apparent inadvertent intravenous injection of 2-chloroprocaine

with a documented high plasma drug level. The intravenous injection seems very likely from the lack of sensory analgesia and the high maternal plasma level of 2-chloroprocaine. The plasma level of 17 $\mu\text{g/ml}$ in this patient was several hundred times higher than those we have previously found following epidural anesthesia for cesarean section.¹ One hour later the maternal blood and cord vein blood levels at delivery were still high, but were within the range of those reported previously.

The rapid hydrolysis of 2-chloroprocaine by plasma cholinesterases limits its potential for toxicity in patients with normal enzyme levels. However, cholinesterase activity decreases during pregnancy,^{1,2} and the potential for toxicity of 2-chloroprocaine in pregnant patients has not been determined. The lack of sequelae in this patient with measured high plasma drug levels is evidence for a wide range of safety of this drug during pregnancy.

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Naloxone as an Antagonist in Alcohol Intoxication

To the Editor:—Naloxone is a well-known specific antagonist of endogenous and exogenous opiates.^{1,2} Alcohol intoxication and opiate poisoning have similar effects, and may be related. Alcohol may produce intoxication by liberating endogenous opiates,³ so naloxone therapy may antagonize the effects of alcohol intoxication. Below is a case report of possible antagonism of alcohol intoxication by naloxone.

A 28-year-old man was brought to the Emergency Room because he had fallen, hit his head, and become unconscious, with heavy epistaxis. During the preceding six days he had been drinking heavily; at the same time, he had been taking tranquilizers (oxazepam and chlordiazepoxide). On admission the patient was afebrile. Blood pressure was 130/50 torr, and pulse rate 120 beats/min. Although the lungs had a few rales in both bases, examination of the heart and abdomen disclosed no abnormality. Neurologic examination revealed that the patient was unconscious, with no sign of focalization. The following diagnoses were made: alcohol intoxication, acute brain injury? and nasal contusion. An intravenous infusion was started and nasal tamponade was initiated. Radiographic examination of the head and nasal cavity disclosed no evidence of fracture. Possible aspiration pneumonitis was evident on the chest radiograph. Results of serologic tests were within normal values except for a blood alcohol level of 3.84 g/l.

At 2:20 P.M., the patient was excited and, again, neurologic examination failed to reveal signs of focalization or cerebral damage. About 3:20 P.M., we

believed the case was essentially a case of acute alcohol intoxication. Based in the reports made by Jeffcoate³ and Sørensen,⁴ we decided to try naloxone therapy. At 3:40 P.M., naloxone, 0.4 mg, was given iv, without response. Ten minutes later a second dose of 0.4 mg was given. The patient began to open and close his eyes. At 4:00 P.M. he awakened, called his wife, and asked several relevant questions. An hour later he was still fully awake, conscious and without excitation.

To our knowledge, naloxone is a pure narcotic antagonist. Assuming alcohol produces intoxication by liberating endogenous opiates, we think naloxone therapy should be kept in mind in cases of alcohol intoxication.

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