

that cuff design and lubricants both have effects on the production of sore throat. It is hoped that the relative contributions of individual factors will be further elucidated by studies now under way. In addition, cuff-wall thickness has an effect in larger cuffs that is probably related to the degree and size of wrinkles on the cuff surface, with thinner cuffs giving better results (Bernhard W: Personal communication).

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Inadvertent Intravascular Injections during Lumbar Epidural Anesthesia

To the Editor:—In his letter regarding inadvertent intravascular placement of caudal epidural blocks, Schweitzer¹ suggests use of a wick (after injecting 2 ml of local anesthetic solution) for detecting blood in the hub of the needle when it cannot be aspirated. Blood staining of the wick indicates an intravascular position. Since one cannot differentiate between venous blood in the hub and traumatic blood staining of a properly placed solution, this test may cause a repetition of the block in some patients while not guaranteeing absence of an intravascular position in the remainder. Additionally, Schweitzer states that the 2-ml dose of local anesthetic is sufficient to produce symptoms of mild systemic toxicity and is a further test of position. In an audit of 4,003 instances of obstetric epidural anesthesia at the Hospital of the University of Pennsylvania from January 1978 to December 1979, 194 patients were found to have had inadvertent intravascular placement, and 13 had had the problem recur with a second placement of the catheter. The intravascular position was recognized by aspiration of blood initially in 130 cases. Apparent intravascular migration of the epidural catheter occurred in 12 other patients when blood was aspirated prior to reinjection of anesthetic during a functioning block. In the 65 remaining cases (including those of ten further patients with intravascular migrations of the catheter) the patients received 2 ml of local anesthetic without epinephrine to test for an inadvertent subarachnoid position. Symptoms of mild toxicity occurred in three patients, two of whom had recurrent symptoms and blood return in the catheter when further volumes of 3 and 5 ml were administered. In the third patient, the catheter was placed in another interspace with recurrence of the symptoms on each injection despite an excellent block.

In 11 patients, blood was aspirated either immediately after the test dose or prior to further drug administration. Thirty-nine of 51 patients who received additional local anesthetic intravenously had symptoms of systemic toxicity (five had tremor or seizure). The remaining 12 had no symptoms in spite of aspiration of blood immediately after injection. Representative doses were 8–10 ml of .25–.75 per cent bupivacaine (nine patients) and 4–20 ml of 2–3 per cent 2-chloroprocaine (three patients). These findings illustrate that 2 ml of local anesthetic solution without epinephrine do not produce symptoms in most patients, and that rather large quantities apparently administered intravenously are well tolerated in 23 per cent of patients. In our experience, when the test solution contains epinephrine, 1:200,000, 2 ml will increase the heart rate when given intravenously. Unfortunately, Schweitzer does not specify whether or not epinephrine was used in the local anesthetic solution. If indeed epinephrine was used by Schweitzer, this might well explain why a 2-ml test dose produced symptoms in his patients but not in ours. Elimination of epinephrine from obstetric anesthetic practice has made detection of intravascular catheters more difficult. Prevention of inadvertent intravascular injection of lumbar epidural catheters continues to be a problem, particularly in pregnant patients, where epidural veins are markedly dilated and easily entered.² Careful aspiration prior to and after every drug administration, employment of test doses prior to each supplemental injection, and use of small, closely spaced incremental doses rather than large bolus administration of local anesthetics may avoid serious toxic symptoms from intravascular injection, as well as inadvertent spinal blockade.

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REFERENCES

1. Schweitzer SA: Avoiding intravascular injections during epidural anesthesia. *ANESTHESIOLOGY* 53:81, 1980
2. Bromage PR: Physiology and pharmacology of epidural analgesia. *ANESTHESIOLOGY* 28:592-662, 1967

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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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