Correspondence

Anesthesiology
51:95, 1979

The Air Test for Regional Blocks

To the Editor:—Dr. Dudley suggests that the presence of a taut axillary sheath following an axillary block is a useful preliminary indication of a successful block.[1] In my experience, palpable distention also occurs after other perivascular nerve blocks: interscalene, femoral–obturator and, infrequently, after an infraclavicular block (producing axillary distention). Unfortunately, it is often exceedingly difficult to palpate the distended neurovascular bundle in the well-developed or obese individual. As an aid to teaching regional anesthesia, I often inject air, 2 ml, following the local anesthetic. The resulting crepitus markedly accentuates the ability to palpate the distended neurovascular sheath. An unsuccessful nerve block produces a diffuse patch of subcutaneous emphysema—an additionally useful sign. The injection of air, normally avoided, is in this instance quite safe since definitive test for an intravascular injection (*i.e.*, injection of local anesthetic) has already been made.

RICHARD M. ROSENBLATT, M.D.
Assistant Professor
Department of Anesthesiology
School of Medicine
University of California
Davis, California 95616

REFERENCE

1. Dudley WR: Axillary-sheath distention, a useful sign in performing axillary block (correspondence). ANESTHESIOLOGY 49:302, 1978

(Accepted for publication January 11, 1979.)

Anesthesiology
51:95–96, 1979

A Possible Interaction of PCB and Halothane in Man

To the Editor:—In 1976, Sipes and Brown[1] developed an animal model of halothane hepatic injury. Necrosis was induced in the livers of rats by exposure to halothane, 1 per cent, for two hours following pretreatment with a single dose of a polychlorinated biphenyl (PCB). It was postulated that PCB quantitatively or qualitatively altered the biotransformation of halothane, resulting in increased covalent bonding of reactive intermediates to microsomal lipids and proteins. The possibility of the additive toxic effects of an additional xenobiotic was also considered. Although these investigators were careful to disclaim any causal relationship between exposure to PCB and halothane-induced hepatic damage in man, they did speculate that "their persistence in the body could result in a continuous exposure of the liver to their enzyme-inducing effects." I was recently called upon to discuss a case that at least circumstantially confirms in man the animal model of Sipes and Brown.

A 33-year-old Caucasian woman previously in good health was admitted to our hospital with a presumptive diagnosis of cystitis. Results of physical and laboratory examinations were consistent with the patient's age and general good health. She had had a total abdominal hysterectomy for fibroids three years prior to the current admission. A review of the chart indicated that this operation had been performed with nitrous oxide–oxygen–halothane anesthesia following induction with thiopental. Muscle relaxation had been obtained with *d*-tubocurarine. Recovery had been uneventful, and the patient had been discharged on the fifth postoperative day.

On the first morning of her current admission the patient underwent cystoscopy and retrograde pyelography with nitrous oxide–oxygen–halothane anesthesia. Once again, induction was with thiopental. Recovery was uneventful, and the patient was discharged five hours after the examination of her urinary tract failed to reveal any abnormality. On the third postoperative day the patient returned to the emergency room complaining of nausea, vomiting, malaise, and fever. Physical examination revealed

that the patient was anxious, with tachycardia, tachypnea and a temperature of 40 C. There was tenderness in the right upper quadrant, and the liver was thought by some observers to extend below the right costal margin. Results of laboratory studies at this time were consistent with hepatocellular damage. On the tenth post-hospital day a needle biopsy of the liver was performed. Microscopic examination of the tissue showed architectural changes in the central lobular areas consistent with "halothane hepatitis." Following treatment with steroids and dietary restrictions, the patient made an uneventful recovery and was discharged on the fifteenth day.

Careful review of the medical record failed to shed any light on the cause of the hepatic damage. Inspection of the face sheet containing the patient's financial and insurance data, however, was most revealing. In the years between her two surgical procedures the patient was employed as a machinist in a plastics plant that fabricated components for electric generators. She was exposed daily to contamination with polychlorinated biphenyls. Unfortunately, the patient was lost to follow up before tissue PCB levels could be determined. This case appears to confirm the animal model of hepatic damage secondary to halothane anesthesia. It also emphasizes the need for the anesthesiologist to evaluate the occupational history.

HOWARD L. ZAUDER, M.D.
Professor of Anesthesiology and Pharmacology
Chairman, Department of Anesthesiology
State University of New York College of Medicine
Upstate Medical Center
Syracuse, New York 13210

REFERENCE

1. Sipes IG, Brown BR Jr: An animal model of hepatotoxicity associated with halothane anesthesia. ANESTHESIOLOGY 45: 622–628, 1976

(Accepted for publication January 12, 1979.)

Anesthesiology
51:96, 1979

Another Indication for an Epidural Blood Patch

To the Editor:—Recently, Lee and Roberts[1] reported a case of paresis of the fifth cranial nerve following spinal anesthesia. This was attributed to a low cerebrospinal fluid (CSF) pressure following dural puncture. Their patient was successfully treated with intravenous fluid therapy and an abdominal binder, which presumably increased the pressure in the peridural venous plexus. If the paresis were due to a low CSF pressure resulting from a dural leak, then an epidural blood patch would have been useful both for confirmation of the cause and for relief of the symptoms.

JEFFREY J. STEVENS, M.D.
Staff Anesthesiologist
Presbyterian Hospital Center
1100 Central, S.E.
Albuquerque, New Mexico 87102

REFERENCE

1. Lee JJ, Roberts RB: Paresis of the fifth cranial nerve following spinal anesthesia. ANESTHESIOLOGY 49:217–218, 1978

(Accepted for publication January 22, 1979.)

Anesthesiology
51:96, 1979

In reply:—Although an epidural blood patch is a useful means for treating symptoms from low CSF pressure, it is our opinion that conservative treatment in the form of intravenous fluids and abdominal binder should be tried first. This is especially true when the differential diagnosis includes viral infection or brainstem thrombosis, as discussed in our report. Fortunately the patient's symptoms subsided within 36 hours in response to conservative methods and there was no need to pursue an epidural blood patch.

JOON J. LEE, M.D.
R. BRYAN ROBERTS, M.D.
Department of Anesthesiology
Wright State University School of Medicine
Dayton, Ohio 45409

(Accepted for publication January 22, 1979.)