greater degree of confidence. That increased confidence, however, is not certainty and never can be. Why then should the second increment be any larger than the first or the third any larger than the second? I believe that the same idea that leads to formulating a test dose should govern all injections of local anesthetics and that a full blocking dose should be the sum of several reasonably safe increments given within a reasonable period of time. This, if accepted, leads to the need for an acceptable drug increment and a reasonable time interval.

My own observations of unintentional intravenous injections have led me to regard 15 mg of bupivacaine, as well as 100 mg of lidocaine, as fulfilling most of the criteria that Conklin and Ziadlou-Rad ascribe to 5 ml of 3% chloroprocaine. Hence, those are the limits I use for all epidural dosing increments. The same observations that led to them also caused me to believe that the interval from injection to appearance of CNS symptoms is about 20 s in most parturients. Therefore, I have taken 30 s as my minimum observation interval between increments. This method of dosing epidurals in an incremental sequence has proven useful to me in detecting unsuspected intravascular catheters before encountering severe CNS or cardiovascular symptoms. I feel that this method, although not entirely failsafe, is at least on sounder conceptual footing than the practice of administering a large bolus of local anesthetic. In addition, it may be used in the clinical situation without undue loss of time.

According to the authors, their case illustrated bupivacaine cardiotoxicity. Since a 20-ml intravenous bolus of 0.75% bupivacaine is indeed capable of producing seizure activity and its concomitant effects, and since their assumption of an extravenous epidural catheter rests entirely on an unprovable claim about their test dose, I feel that the questions this case raises concern assumptions made about epidural technique more than bupivacaine's cardiotoxicity.

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Intracerebral Hemorrhage after Dural Puncture and Epidural Blood Patch: Nonpostural and Noncontinuous Headache

To the Editor:—Persistent nonpostural headache after dural puncture may indicate the presence of a serious intracranial lesion.[1] The following is a case report of intracerebral hemorrhage after dural puncture and epidural blood patch. The patient exhibited two features not usually associated with postdural puncture headache. First, a nonpostural headache persisted in addition to postural headache and secondly, relief of the headache occurred for almost 24 h between the third and the fourth day.

REPORT OF A CASE

A 58-year-old woman complained of postural headache on the second day after a myelogram that was done for back pain. Unresponsive to analgesics and bed rest, her headache became severe even in the supine position on the second day. Between the third and the fourth day, for almost 24 h, her headache apparently was relieved. With the reappearance of her headache, an epidural blood patch was done on the fifth day; 10 ml of the patient's blood was used. Arterial blood pressure and heart rate were unchanged before, immediately, and 1 h after the blood patch. The patient stated that her headache was less painful when asked to sit up an hour after the blood patch. Five hours later, she complained of weakness of her right upper arm. Six hours after the patch, she had expressive aphasia, right facial palsy, and asteregnosis of her right hand. Blood pressure was 110/70 mmHg, heart rate 60 beats/min, and respiratory rate 20/min. ECG and electrolytes were normal. Neurology impression was transient ischemia attack–stroke profile with the lesion at the left posterior temporo-parietal area. Eleven hours after the blood patch, she developed grand mal seizures for 2–3 min, and 500 mg phenytoin (dilantin) was given iv. Fourteen hours after the blood patch, the patient became unresponsive to verbal or tactile stimuli but had positive Doll's eyes movements. A CAT scan done showed areas of blood density in the left frontal and parietal regions with shift of the midline structures to the right. The patient lost her Doll's eye movements, became comatose, and went into decerebrate posture the following day. She was unresponsive to deep pain and without brain stem reflexes. The trachea was intubated, and a flow-directed catheter was inserted. Arterial blood pressure was maintained with an iv dopamine drip. Electroencephalogram showed minimal cerebral activity; this became flat on the third day. The patient died on the fifth day. Her family refused autopsy.

Postdural puncture headache is classically postural.* The nonpostural nature of the headache or the return of the headache after 24 h of no headache might have indicated the occurrence of a developing intravascular

* Bridenbaugh PO: Postdural puncture headache. Regional Anesthesia 3:5–8, 1978.

pathology. This case report emphasizes closer observation, including neurologic consultation, of patients with unusual symptoms.

The role of the blood patch is not clear. Although radicular back pain has been reported,[2] two series of 108 and 116 epidural blood patches did not reveal any neurologic complication.[3,4]

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Anaphylaxis to Intraperitoneal Dextran

To the Editor:—The use and administration of various substances by our surgical colleagues may on occasion lead to serious complications that the anesthesiologist must both diagnose and treat. The following brief case presentation illustrates this situation and calls attention to a previously unreported problem.

REPORT OF A CASE

A 66-yr-old woman with ascites due to ovarian carcinomatosis was brought to the operating room for the placement of an intraperitoneal catheter with attached subcutaneous reservoir device (Port-a-Cath). This system is used to administer chemotherapeutic agents. She had mild hypertension, which was well controlled with thiazide diuretics. A halothane-N_2O-O_2 endotracheal anesthetic was administered, and monitoring consisted of a cardioscope, stethoscope, blood pressure cuff, temperature probe, and spectrographic analysis of inspired and exhaled gases. Blood pressure remained stable at 120–130/80 mmHg and pulse rate was 100 beats/min. At the conclusion of the operative procedure, 20 ml 32% dextran-70 in 10% dextrose (Hyskon) were injected by the surgeon into the reservoir and catheter system to prevent the formation of adhesions. Shortly thereafter, the patient's blood pressure decreased to 50/30 mmHg and the pulse rate was 30 beats/min, with an idioventricular rhythm and marked depression of the ST segments. The patient was treated initially with ephedrine and then a dopamine infusion for what was thought to be cardiogenic shock secondary to acute myocardial injury. Her blood pressure responded only marginally and she began wheezing and developed a flushed appearance. The diagnosis of anaphylactoid reaction to the dextran was entertained, and intravenous epinephrine was administered in two 200-μg doses. This resulted in a substantial improvement in perfusion pressure and a diminution in the wheezing. An arterial line was placed as well as a pulmonary artery catheter, and the patient was admitted to the coronary care unit maintained on an epinephrine infusion. Although the differential included myocardial infarction and pulmonary thromboembolism, all studies for these possibilities were negative over the next 36 h. The patient fully recovered and was discharged to home.

The occurrence of anaphylactoid reactions to dextrans has been well documented, and the incidence appears to be approximately 3/10,000 administrations.[1–3] The management of acute anaphylaxis has similarly been well described in the current literature.[4,5] The purpose of this report is to alert the anesthesiologist to the possibility of this complication with intraperitoneal placement of dextran. The possibility of intravascular injection in our case does remain even though no blood could be aspirated from the system. The package insert accompanying the solution (Hyskon®) does caution that the absorption characteristics from the peritoneal and uterine cavities are unknown and that anaphylaxis is a possibility. This substance is utilized in gynecologic procedures to distend the uterine cavity for hysteroscopy, to prevent tubal adhesions after reconstructive tubal surgery for infertility, and for the purpose indicated in this case. Anesthesiologists must remain aware that such an adverse reaction may occur even with intended intraperitoneal placement and they must be prepared to treat accordingly.

ADDENDUM

Since the preparation of this letter, I have become aware of a very recent case report in the gynecologic literature describing a similar anaphylactoid response to intraperitoneal dextran.[6] The authors' contribution should be acknowledged as the first such report.

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