injected epidurally due to the physical barrier presented by the dura, and by the simultaneous uptake of morphine into the general circulation by Batson's epidural venous plexus. Morphine, 8 mg, injected extradurally reaches a peak cerebrospinal fluid concentration at 120 min with approximately 3,500 ng/ml, whereas at 10 min, 1 mg of intrathecal morphine has a concentration of 40,000 ng/ml. Thus, if one can extrapolate, then even 0.4 mg of intrathecal morphine still will have a very high cerebrospinal fluid concentration and readily will produce respiratory depression if encouraged to reach the fourth ventricle by coughing, straining, or, as occurred in this patient, by being put in the Trendelenberg position when using a hyperbaric solution.

The original reports of intrathecal morphine were using 0.5–1 mg, but doses as high as 20 mg have been given intrathecally. The effectiveness of doses of 0.5–1.0 mg has been demonstrated clearly. No dose response curve for intrathecal morphine, however, has been made yet, thus establishing the minimal effective dose for intrathecal use. The presently recommended dose is 0.5–1 mg. However, with very high concentrations of morphine entering the cerebrospinal fluid from intrathecal administration, and extrapolating this against cerebrospinal fluid concentrations that are effective after epidural injection, then doses of possibly 0.05–0.1 mg may well be as effective and much safer.

P. S. A. Glass, FFA (S.A.)
Senior Registrar
Johannesburg Hospital
Johannesburg 20000
Republic of South Africa

REFERENCES


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Bupivacaine Cardiotoxicity May Be More Related to Technique than to the Drug

To the Editor:—Drs. Conklin and Ziadou-Rad described a case that they felt illustrated several points that are pertinent to the prevention of bupivacaine cardiotoxicity. I would maintain that, while certain of the steps they advocate might lessen the incidence of large intravascular drug boluses associated with epidurals, there are still assumptions in both their original and amended manner of performing the injection sequence that should be examined.

At the outset of their procedure, the authors placed a catheter in what they felt was the epidural space and then aspirated. Finding no evidence of blood or other fluid, they injected small amounts of chloroprocaine as test doses based on the claim that “... we have found that in parturients, 5 ml of 3% chloroprocaine uniformly produces signs of CNS toxicity without producing seizures.”

This claim was not substantiated in any verifiable manner. That would seem to require deliberate intravascular injections of at least mildly toxic doses of local anesthetics; and, because of this, I am not sure that their statement can be supported in any but an anecdotal manner. Also, their statement seems to imply that all parturients respond in like fashion to a fixed dose of drug. This would be contrary to clinical experience. Thus, when authors state that some amount of anesthetic uniformly produces symptoms and that those symptoms are uniformly limited, they assume a burden of proof I do not believe they can support.

If one cannot prove the test dose to be completely reliable in detecting an intravascular catheter, then to inject the entire blocking dose at one time, as Conklin and Ziadou-Rad did, seems inappropriate. The authors partially address this by acknowledging that “fractionating” the full dose may lessen the risk inherent in a single large injection. However, they do not ask the next logical question: How much is a safe increment? I assume that their 5 ml per minute of 0.75% bupivacaine represents a guess rather than an empiric observation, since they did not make a claim for it similar to the one made for 5 ml 3% chloroprocaine. Had they applied the same standard of observed safety to their bupivacaine dose as to their test dose, I think they might have injected the bupivacaine in a different manner.

A test dose of a local anesthetic represents the largest safe increment that one knows to give. Even though it cannot be proven infallible as an indicator of an intravenous injection, the next increment is given with a much
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, these 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 extraneous 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.

ROBERT M. KNAPP, D.O.
Assistant Professor of Anesthesia
Director, Obstetric Anesthesia
University of Cincinnati
College of Medicine
Cincinnati, Ohio 45267

REFERENCE


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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. 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 asterognosis 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. Neurologic impression was transient ischemia attack-stroke profile with the lesion at the left posterior tempo-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