Maternal Heart Rate Changes with a Plain Epidural Test Dose—Validity of Results Open to Question

To the Editor—Cartwright et al. report an increased heart rate 3 ml of plain 0.5% bupivacaine injected through an epidural catheter at the rate of 1.0 ml/s.¹ They concluded: “Thus, the interpretation of the epinephrine test dose for obstetric anesthesia is difficult because of the poor specificity of the test.” We question the validity of this study based on the stated rate of injection through the epidural catheter.

When administering a single-injection epidural block via a 19-gauge needle, a 3-ml test dose of a local anesthetic can be injected in 3 s.² On the other hand, doing so through standard length epidural catheters is impossible.* When holding the catheter’s connector in one hand, exerting maximum pressure on the syringe’s plunger with the other, and timing the injections with a stop watch, a minimum of 4.5 s elapsed with a 3 ml syringe, 5 s with a 5 ml syringe, and 5.5 s with a 10 ml syringe. Furthermore, the “fire hose”-type stream exiting from the end of the catheter shot at least 3 m (10 ft) across the room. Evidently, Cartwright et al. exerted maximum pressure on the plungers of the syringes. Therefore, one wonders whether the effects of the jet stream in the epidural space was responsible for heart rate increases.

To avoid the “fire hose” effect when injecting through an epidural catheter, the rate of 0.2 ml/s should not be exceeded.* Nonetheless, we have found that injecting 3 ml of a local anesthetic containing 1:200,000 epinephrine (0.015 mg) at this rate will determine an intravascular injection into an epidural blood vessel. However, during its injection and for 45 s after it, the patient must not be stimulated by a uterine contraction, nor should anyone talk to, move, or examine the patient. Otherwise, a false positive may result; that is, a heart rate increase which, on cessation of stimulation, immediately returns to the control level. Conversely, the heart rate increase from 0.015 mg of epinephrine as monitored by an electrocardioscope is sustained for 20 s or more and 30–60 s elapse from when it starts to decline until it returns to its control level.²

To conclude, in our hands, the test dose has been “foolproof.” Since 1980, † no systemic toxic reactions have resulted in more than 8,500 surgical and 6000 obstetrical single- or intermittent-injection epidural blocks. Prior to then, one of us (DCM) treated three local anesthetic induced convulsions per year.

† Moore DC: Necessary ingredients of a test dose prior to epidural or caudal block. (Abstract) ANESTHESIOLOGY 53:S214, 1980

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Pregnancy and Oxygen Dissociation

To the Editor—I read with interest the article “Effect of Normal and Preeclamptic Pregnancies on the Oxygenhemoglobin Dissociation Curve.”¹¹ The authors confirmed a stepwise decrease of oxygen hemoglobin affinity during normal pregnancy, but did not provide an explanation for this finding.

Drs. North and Brake found that the 2,3 DPG content of erythrocytes increased in the ninth month of pregnancy

Anesthesiology

* Tubing tested was that which: 1) will pass through a 18-gauge thin-walled needle; 2) had a single opening at the end; and 3) was 90 cm (Deseret Medical, Inc., Abbott Laboratories, Inc., Tact Medical, Inc.) or 97.5 cm (Burron Medical, Inc.) in length.

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to about 130% of the level present in erythrocytes of non-pregnant women. In this study, the actual and the standardized P 50 were increased to about 2.5–3.0 mmHg. Moreover, hormonal changes during pregnancy could affect the red cell metabolism, e.g., the 2,3 DPG level has been shown to be sensitive to changes in the concentration of thyroxin and prostaglandin.\(^2\),\(^3\)

Furthermore, the authors did not discuss the leftward shift of the oxyhemoglobin dissociation curve in preeclamptic parturients. One explanation for the leftward shift could be hypophosphatemia which increases oxygen affinity of hemoglobin.\(^4\) This electrolyte imbalance could be induced by excessive loss of phosphate, which occurs when hypokalemia is provoked by diuretic therapy in these patients. In any case, this remarkable observation should be commented upon.

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In Reply.—The effect of normal pregnancy on oxyhemoglobin dissociation (OHD) has been reported by several investigators. A leftward shift of OHD seen in preeclamptic pregnant women is new and surprising. Both normal and preeclamptic pregnancies are associated with complex changes in a variety of hormones, including prostaglandins. Several of these hormones are also known to cause shifts in OHD. We have discussed the possible causes for a leftward shift of OHD in a recent abstract describing the effect of pregnancy induced hypertension on OHD.\(^1\) Evidence also suggests that there is increased red cell destruction in patients with toxemia of pregnancy.\(^2\) In addition, we have recently reported that preeclamptic patients have significantly higher levels of carboxyhemoglobin compared with normal pregnant women.\(^5\) We believe that the increased level of carboxyhemoglobin is principally responsible for the observed leftward shift in OHD. Our preeclamptic patients did not receive any diuretic therapy and, thus, hypophosphatemia from diuretic therapy is unlikely. Also, none of our patients at the time of the study was receiving any other drug that could possibly cause a shift in OHD.

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\[\text{pH-adjusted Lidocaine Does Not "Sting"}\

To the Editor.—The widespread practice at our institution of adding sodium bicarbonate to lidocaine solutions (ratio 1:10) to improve the success rate of regional anesthesia\(^1\) has led to the observation that these pH-adjusted solutions do not “sting” when injected intradermally or subcutaneously. The improvement in comfort when providing cutaneous anesthesia is striking. Starting an iv in a child, for example, is greatly facilitated when pH-ad-