

Anesthesiology
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Is Midazolam Desirable for Sedation in Parturients?—Reply

To the Editor:—In their letter to the Editor, Camann *et al.*¹ observed that sedation of parturients with midazolam (Versed®) after the umbilical cord is clamped during regional anesthesia for cesarean section may cause amnesia for the birth of the baby.

We wish to confirm these observations, and have subsequently changed our procedure to postpone midazolam administration until after pediatric ministrations to the neonate in the operating room are completed, and the baby is brought to the mother and shown to her at the head of the table. Since we effected this procedural change, we have had no further complaints of amnesia for the birth experience.

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Allergic Reactions to Muscle Relaxants

To the Editor:—We are in the process of developing a laboratory test to detect antibodies to muscle relaxants in patients' blood. This test can be used to document that a patient has had a reaction or to predict which muscle relaxants the patient may safely receive, as to a muscle relaxant, cross reactivity may occur.

In order to validate this test, we need serum from patients who have had such reactions. If you have had, or know of, a patient in which a severe anaphylactoid reaction has occurred, and in which there is a reasonable chance that the offending agent was a muscle relaxant, we ask you to contact us. It is in your patient's interest and in

your interest that these reactions be documented and the agent identified.

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Epinephrine Should Not Be Used with Local Anesthetics for Epidural Anesthesia in Pre-eclampsia

To the Editor:—I am disturbed by the recent case report on the use of epinephrine in local anesthetic solutions used in pre-eclampsia.¹

The authors postulate a vasodilatory effect from absorbed epinephrine that "may improve uterine flow." This assumes that uterine vessels will also dilate in common with systemic vessels, as has been previously postulated in normal pregnant patients.² However, in pre-

eclampsia, the uterine vasculature has excessive vasoconstrictive reactivity to catecholamines.³ Accidental intravascular injection poses a relatively greater threat to placental circulation in pre-eclampsia; 15 µg of intravenous epinephrine is not without fetal ill effects in the normal laboring patient.⁴ If systemic vasodilation does occur in pre-eclampsia, this may "steal" blood from the placenta. Improvements in placental blood flow in pre-eclampsia

following epidural blockade are thought to result from reductions in circulating catecholamines⁵ caused by extensive sympathetic block.^{6,7} Absence of maternal hypertensive effects is no guarantee of absence of reduction in placental blood flow. In the cases presented, routine Apgar scores are the sole source of fetal evaluation.

The use of epinephrine in obstetric anesthetics is controversial enough^{8,9} without this ill-considered addition. Let us not allow these four cases to interfere with our appropriate use of epidural blockade in pre-eclampsia^{7,10}—without epinephrine.

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In Reply:—I thank Dr. Robinson for his letter. He alerted me to an omission from the cases we reported.¹ We failed to report that patients 1 and 3 had continuous fetal heart rate monitoring during performance of the peridural block. For patient 2, fetal heart rate was ascertained by auscultation after performance of the block. Careful examination of the details of case 4, as they were described, reveal that we noted that the fetal heart rate was continuously monitored. In no case was there any indication of fetal distress with the institution of peridural block using local anesthetic solutions with epinephrine.

Dr. Robinson claims that “in preeclampsia the uterine vasculature has excessive vasoconstrictive reactivity to catecholamines.” His reference for this statement is a study by Talledo *et al.*² However, this reference shows that 1) epinephrine was not studied; only the responses to iv infusions of angiotensin II and norepinephrine, and 2) the reactivity of the uterine vasculature was not evaluated. The measurements made were of systemic blood pressure *via* a femoral artery catheter. These data cannot be extrapolated to predict how the uterine vasculature will respond when exposed to low doses of epinephrine injected peridurally. Epinephrine, unlike norepinephrine, has very strong activity at beta₂ receptors located in the peripheral vasculature.³ Because of this, at low doses, it

has primarily beta agonist effects,⁴ and can lower blood pressure even when injected intravenously.³ Vasodilation from peridural block with epinephrine-containing local anesthetic solutions is more extensive than that seen with the administration of plain solutions in resting, nonlaboring, nonpregnant volunteers.⁵ Injection of epinephrine alone (without local anesthetic) into the peridural space has been shown to result in mild decreases in systemic vascular resistance.⁶ It has been postulated that human placental vessels dilate when exposed to peridurally administered epinephrine.⁶ Albright *et al.*⁶ found an average increase in intervillous blood flow of 50% when using 2-chloroprocaine with 1:200,000 epinephrine peridurally for labor analgesia in normal parturients. There is clearly no constriction of the uterine vasculature.

Dr. Robinson also notes that “15 µg of intravenous epinephrine is not without fetal ill-effects in the normal laboring patient.” The source in this instance is an abstract presented by Leighton *et al.*^{*8} at the 1986 meeting of the Society of Obstetrical Anesthesia and Perinatology, as well

* Leighton BL, Norris MC, Sosis M, Epstein R, Chayen B, Larijan GE. Epinephrine test dose may not be safe in labor. Abstract from Eighteenth Annual Meeting of Society for Obstetrical Anesthesia and Perinatology, 1986.