following epidural blockade are thought to result from reductions in circulating catecholamines[5] 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.

DAVID A. ROBINSON, F.F.A.R.C.S.
Visiting Assistant Professor of Obstetric Anesthesiology
Department of Anesthesiology
University of Texas Health Science Center
San Antonio, Texas 78284-7838

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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 $beta_2$ 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.

as the 1986 meeting of the American Society of Anesthesiologists. Maternal and fetal heart rate changes with the intravenous injection of either 15 μg of epinephrine or normal saline were examined. Fetal distress was noted in 2/10 patients given epinephrine, and in none of ten patients given normal saline ($P = 0.47$). In other words, these events could have occurred by chance. No negative conclusions can be drawn about the safety of 15 μg of intravenous epinephrine in laboring parturients with this information.

Dr. Robinson postulates that "if systemic vasodilation does occur in pre-eclampsia, this may steal blood from the placenta," and implies that any improvements in intervillous flow occurring in preeclamptic patients after peridural blockade result from reduction of high levels of circulating catecholamines. However, the vasodilating properties of peridural anesthesia are well known. Since this occurs even in subjects who are not acutely stressed,[5] it probably has little to do with reduction of high levels of circulating catecholamines. There is no reason to believe that preeclamptic parturients will not respond with vasodilation as well. It has been documented that intervillous blood flow improves significantly in preeclamptic patients given peridural analgesia without epinephrine.[8] Clearly, no placental steal occurs with the vasodilation of peridural analgesia.

As noted in our response[9] to the letter of Drs. Costin and Millikin,[10] we have administered local anesthetics with epinephrine into the peridural space of several preeclamptic patients without ill effect in any instance, and we do not believe it is harmful when used correctly.

PAUL J. HELLER, M.D.
Assistant Professor of Anesthesiology
Yale University School of Medicine
New Haven, Connecticut 06510

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Atracurium for Open Eye Injuries

To the Editor:—Badrinath *et al.*[1] reported that anesthesia induced with large doses of iv anesthetics in conjunction with atracurium 0.6–0.8 mg/kg provided excellent intubating conditions with minimal changes in intraocular pressure (IOP), and recommend this technique for the management of patients with open eye injuries. However, the median intubation score in five of their seven patient groups indicated that coughing may have been present during intubation. In two groups, at least one patient experienced bucking, coughing, and straining. Such responses to intubation may cause dramatic transient increases in IOP not measured in this study that are not desirable in patients with open eye injuries. Atracurium in these doses apparently does not consistently provide acceptable intubating conditions for these patients during rapid-sequence induction.

DAVID O. WARNER, M.D.
Resident in Anesthesiology
Mayo Clinic
Rochester, Minnesota 55905

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