

- JB, Turner ME: Survival and probability of cure without and with operation in complete atrioventricular canal. *Ann Thorac Surg* 27:104-111, 1979
8. Park SC, Mathews RA, Zuberbuhler JR, Rowe RD, Neches WH, Lenox CC: Down syndrome with congenital heart malformation. *Am J Dis Child* 131:29-33, 1977
 9. Katlic MR, Clark EB, Neill C, Haller JA: Surgical management of congenital heart disease in Down's syndrome. *J Thorac Cardiovasc Surg* 74:204-209, 1977
 10. Soundheimer HM, Byrum CJ, Blackman MS: Unequal cardiac care for children with Down's syndrome. *Am J Dis Child* 139: 68-70, 1985
 11. Smith, DW: Recognizable patterns of malformation: Down syndrome, *Recognizable Patterns of Human Malformation*, 3rd edition. Edited by Smith, DW. Philadelphia, WB Saunders, 1982, pp 10-13
 12. Rowland TW, Nordstrom LG, Bean MS, Berkhardt H: Chronic upper airway obstruction and pulmonary hypertension in Down's syndrome. *Am J Dis Child* 135:1050-1052, 1981
 13. Levine OR, Simpser M: Alveolar hypoventilation and cor pulmonale associated with chronic airway obstruction in infants with Down syndrome. *Clin Pediatr* 21:25-29, 1981
 14. Cooney TP, Thurlbeck WM: Pulmonary hypoplasia in Down's syndrome. *N Engl J Med* 307:1170-1173, 1982
 15. Yamaki S, Horiuchi T, Sekino Y: Quantitative analysis of pulmonary vascular disease in simple cardiac anomalies with the Down syndrome. *Am J Cardiol* 51:1502-1506, 1983

Anesthesiology
65:224-226, 1986

Use of Local Anesthetics with Epinephrine for Epidural Anesthesia in Preeclampsia

PAUL J. HELLER, M.D.,* CAREN GOODMAN, M.D.†

Fear of excessive hypertension in preeclamptic parturients has led to the notion that epidurally administered local anesthetics should not be mixed with epinephrine.^{1-3,‡} However, this seems to be a theoretical concern and actual occurrence of excessive hypertension in this situation has not been reported. We describe four cases in which epinephrine-containing solutions were used in such patients without any deterioration in the condition of the parturient or the fetus.

REPORT OF FOUR CASES

Case 1. A 25-yr-old woman G₅P₂, 40-weeks gestation with preeclampsia, requested lumbar epidural anesthesia (LEA) for labor analgesia. She was asthmatic and had a past history of iv drug abuse (heroin) and hepatitis B. Medications included: anhydrous theophylline (sustained release), 300 mg q 8h and metaproterenol inhaler, 2 puffs qid prn. She had received LEA for vaginal delivery in the past without incident. Height was 170 cm, weight was 75 kg. Arterial blood pressure was 180/92 mmHg and heart rate 90 beats/min. Physical examination revealed mild expiratory wheezing, without acute respiratory distress, 3+/4 deep tendon reflexes and 1+/4 clonus. Clotting profile was normal. She was receiving a magnesium sulfate infusion at 2 g/h. After receiving 1000 ml of lactated Ringer's solution, LEA was performed uneventfully at L4-5, using 2% lidocaine with 1:200,000 epinephrine.

A total dose of 15 ml (75 µg epinephrine) was given incrementally to achieve a T-10 level. Arterial blood pressure was 180/90 mmHg and heart rate 80 beats/min before LEA and remained unchanged 30 min post-LEA. Shortly afterward a vigorous female infant, with apgar scores of 9/9 (at 1 and 5 min, respectively), was born. The preeclampsia resolved shortly thereafter.

Case 2. A 20-yr-old woman, 41-weeks gestation, with preeclampsia presented for a cesarean section for failure to progress during labor. She had no significant past medical history. On physical examination, she was 163 cm, 79.5 kg, with moderate generalized edema. Arterial blood pressure varied between 140/80 to 170/100 mmHg, and she had 2+/4 clonus. Clotting profile was normal. Magnesium sulfate was administered iv at a rate of 2 g/h. LEA was performed uneventfully at the L3-4 interspace, using 2% lidocaine with 1:200,000 epinephrine following infusion of 1500 ml lactated Ringer's solution. A total of 26 ml (130 µg epinephrine) was given incrementally to achieve a T4 level of anesthesia. At the start of the procedure, arterial blood pressure was 130/90 mmHg and heart rate 85 beats/min. Thirty minutes later, arterial blood pressure decreased to 100/60 mmHg with a heart rate of 130 beats/min. Treatment with ephedrine 5 mg iv resulted in a return to baseline values. During delivery, the arterial blood pressure remained at 110/70 mmHg and a heart rate of 100-130 beats/min was sustained for the remainder of the procedure. A vigorous male infant, apgar scores of 9 at both 1 min and 5 min following delivery, was born. The preeclampsia resolved after 2 days, at which time the magnesium sulfate was discontinued.

Case 3. A 13-yr-old girl with preeclampsia requested LEA for labor analgesia at 5 cm dilation. She had no significant past medical history. On examination, she was 157 cm tall and weighed 85 kg. Arterial blood pressure was 140/104 mmHg, and she had mild generalized edema. Clotting studies were normal. No magnesium sulfate was administered because reflexes were normal. LEA was performed uneventfully at the L3-4 interspace, after receiving 1000 ml lactated Ringer's solution, using a combination of bupivacaine 0.5% plus lidocaine 2% with 1:200,000 epinephrine mixed in equal volumes (to yield bupivacaine 0.25%, lidocaine 1%, and epinephrine 1:400,000). A total of 8 ml (20 µg epinephrine) was given incrementally to achieve a T9 level. The pre-LEA arterial blood pressure was 140/100 mmHg with a heart rate of 100 beats/min initially, and they did not change

* Assistant Professor of Anesthesiology.

† Resident in Anesthesiology.

Received from the Department of Anesthesiology, Yale University School of Medicine, 333 Cedar Street, New Haven, Connecticut 06510. Accepted for publication March 24, 1986.

Address reprint requests to Dr. Heller.

Key words: Anesthesia: obstetrics, eclampsia. Anesthetic techniques: epidural. Sympathetic nervous system: epinephrine.

‡ Bonica JJ: Obstetric Analgesia and Anesthesia. Amsterdam, World Federation of Societies of Anesthesiologists, 1980, pp 144.

following the completion of LEA. A reinforcement of 7 ml of bupivacaine 0.25%, lidocaine 1%, epinephrine 1:400,000 (17.5 μ g epinephrine) was given 2 h later, again without change in arterial blood pressure. Three and one-half hours after performing LEA a male infant, with apgar scores of 9 at both 1 min and 5 min following delivery, was born. The preeclampsia resolved 5 hours after delivery.

Case 4. A 26-yr-old woman, 35 weeks gestation, 160 cm, 75.5 kg, requested LEA for labor. She was admitted to the labor suite 24 h earlier with severe preeclampsia. Arterial blood pressure was 202/112 mmHg, and she had marked generalized edema with 4+/4 reflexes and 4+/4 proteinuria. Magnesium sulfate was started at 2 g/h iv. In addition, intermittent bolus administration of hydralazine 5 mg iv was given for hypertension. Labor was augmented with oxytocin. Several hours after admission, she began to complain of shortness of breath. Auscultation showed that lung fields were clear, but a chest roentgenogram showed mild congestive heart failure. Clotting studies were normal. A right internal jugular central venous pressure (CVP) catheter was inserted; CVP was 20 cmH₂O. As a result, fluids were restricted, and the CVP catheter was exchanged for a pulmonary artery (PA) catheter. An arterial catheter was inserted as well. The PA diastolic pressure was 7 mmHg with a wedge pressure of 5 mmHg. At the time LEA was administered, the arterial blood pressure was 160/90 mmHg with a heart rate of 100 beats/min, pulmonary artery diastolic pressure (PAD) was 7–10 mmHg, and urine output was 20–40 ml per h. LEA was done at L4–5 with great difficulty due to severe presacral edema, using bupivacaine 0.25%, lidocaine 1%, and epinephrine 1:400,000. Seven milliliters (17.5 μ g epinephrine) were given incrementally to achieve a T-10 level of anesthesia. No fluids were given iv. Arterial blood pressure decreased to 120/80 mmHg with LEA. The fetal heart rate was continuously monitored and was stable. Reinforcement injections of 8–10 ml bupivacaine 0.25%, lidocaine 1%, and epinephrine 1:400,000 (20–25 μ g epinephrine) were given about every 1½ h. Arterial blood pressure remained in the 140/80–150/90 mmHg range, and never rose with any reinforcement. PAD pressures remained in the 5–10 mmHg range. After about 10 h, tachyphylaxis began to develop and reinforcement doses were required every 45–60 min. Twelve hours after instituting LEA, the decision was made to perform a cesarean section for failure-to-progress during labor. By that time, 90 ml of the lidocaine–bupivacaine (225 μ g epinephrine) combination had been given. The patient was transferred to the operating room and placed on the operating table with left uterine displacement and given O₂ by face mask at 4 l/min flow. Fifteen ml 2% lidocaine with 1:200,000 epinephrine (75 μ g epinephrine) was given incrementally to achieve a T-6 level of anesthesia, which was spotty, and was supplemented with 10 ml 3% 2-chloroprocaine. There was no change in arterial blood pressure after the injection of lidocaine with epinephrine, and the PAD remained in the 10–15 mmHg range. A female infant, with apgar scores of 6 1 min after delivery and 9 5 min after delivery, was delivered 17 min after skin incision. Diazepam and fentanyl were given for sedation after delivery, and the procedure was completed uneventfully. The patient's condition stabilized postoperatively with the maintenance of magnesium sulfate, and the PA and arterial catheters were removed 24 h later. Magnesium sulfate was maintained for 3 days until the edema decreased and reflexes became normal.

DISCUSSION

It has been suggested that local anesthetic solutions containing epinephrine be avoided when performing LEA for preeclamptic patients.^{1–3,†} The rationale for this has been the supposition that these hypertensive patients may be more sensitive to epinephrine administration than normal parturients resulting in a worsening of the hy-

pertension. However, there are no data to support this hypothesis. On the other hand, studies of the cardiovascular effects of LEA in normal nonpregnant subjects have shown a greater degree of reduction of systemic vascular resistance and mean arterial pressure when local anesthetic solutions containing epinephrine are used than when epinephrine is omitted.^{4,5} This presumably is due to the slow absorption of small amounts of epinephrine from the peridural space, which would have primarily beta-adrenergic agonistic effects of lowering systemic vascular resistance and mean arterial blood pressure while increasing heart rate, stroke volume, and cardiac output.^{4,5} No data exist to indicate that preeclamptic patients behave in any different fashion in this regard. Our four cases suggest that these patients are not likely to experience a worsening of hypertension when small amounts of epinephrine are injected epidurally. Also significant is the absence of hypotension in these patients, who tend to be dehydrated. This points to the efficacy of adequate iv fluid preloading in preventing hypotension from regional blockade under these circumstances. On the other hand, accidental iv injection of epinephrine may lead to clinically significant increases in blood pressure. In addition, iv-administered epinephrine can cause a dose-related reduction in uterine flow without adversely affecting the normal fetus.⁶ Thus, meticulous technique is mandatory, and only small incremental doses of epinephrine containing local anesthetic solutions should be administered.

Patient number two had an increase in heart rate from 85 to 130 beats/min and a decrease in blood pressure from 130/90 mmHg to 100/60 mmHg with the institution of LEA. Although the other patients did not manifest these changes, significant increases in heart rate can occur when epinephrine is given epidurally.^{4,5} Patient number two also received a much larger dose of epinephrine at one time (130 μ g) than the other patients. Theoretically, a certain threshold dose of epinephrine must be administered epidurally before a significant beta-adrenergic effect will be seen, especially under conditions of slow absorption of small amounts of epinephrine.

LEA (without epinephrine) in preeclamptic patients will result in unchanged or improved placental blood flow, provided that hypotension is avoided.^{7,8} This appears to be due to a reduction in the high vascular resistance found in preeclampsia⁹ resulting in improved flow. Previous work has demonstrated that local anesthetics containing epinephrine administered epidurally have no significant effect on placental flow in normal parturients with presumably normal systemic vascular resistance.^{10,11} Hypothetically, under conditions of elevated systemic resistance, such as in preeclampsia, the greater reduction of systemic resistance produced with epinephrine-containing solutions may improve uterine flow to an even greater degree. As neonatal morbidity and mortality are signifi-

cantly greater in preeclampsia,¹² this is an important factor to consider, although it is pure conjecture at this point.

In conclusion, we have presented four cases in which preeclamptic patients were given LEA with local anesthetic solutions containing epinephrine. In no case was the hypertension enhanced. Previously, preeclamptic patients were felt to be at risk of a worsening of an already elevated blood pressure when given epinephrine in the epidural space.^{1-3,†} However, the amount of epinephrine absorbed from the peridural space seems to have primarily a beta-adrenergic agonistic effect and does not worsen preexisting hypertension in preeclamptic patients.

REFERENCES

1. Gutsche B: Anesthetic considerations for preeclampsia-eclampsia, *Anesthesia for Obstetrics*. Edited by Shnider S, Levinson G. Baltimore, Williams and Wilkins, 1979, pp 224-234
2. Marx G, Hodgkinson R: Special considerations in complications of pregnancy, *Obstetric Analgesia and Anaesthesia*. Edited by Marx G, Bassel G. New York, Elsevier-North Holland-Biomedical Press, 1980, pp 296-334
3. Wright J: Anesthetic considerations in preeclampsia-eclampsia. *Anesth Analg* 62:590-601, 1983
4. Kennedy W, Sawyer TK, Gerbershagen HU, Cutler RE, Allen GD, Bonica JJ: Systemic cardiovascular and renal hemodynamic alterations during peridural anesthesia in normal man. *ANESTHESIOLOGY* 31:414-421, 1969
5. Bonica JJ, Akamatsu TJ, Berges PU, Morikawa K-I, Kennedy WF: Circulatory effects of peridural block. II. Effects of epinephrine. *ANESTHESIOLOGY* 34:514-522, 1971
6. Hood DD, Dewan DM, Rose JC, James FM: Maternal and fetal effects of intravenous epinephrine containing solutions in gravid ewes (abstract). *ANESTHESIOLOGY* 59:A393, 1983
7. Jouppila R, Jouppila P, Hollmen A, Koivula A: Epidural analgesia and placental blood flow during labor in pregnancies complicated by hypertension. *Br J Obstet Gynecol* 86:969-972, 1979
8. Jouppila P, Jouppila R, Hollmen A, Koivula A: Lumbar epidural analgesia to improve intervillous blood flow during labor in severe preeclampsia. *Obstet Gynecol* 59:158-161, 1982
9. Groenendijk R, Trimbos JBMJ, Wallenburg HCS: Hemodynamic measurements in preeclampsia: Preliminary observations. *Am J Obstet Gynecol* 150:232-236, 1984
10. Albright G, Jouppila R, Hollmen AI, Jouppila P, Vierola H, Koivula A: Epinephrine does not alter human intervillous blood flow during epidural anesthesia. *ANESTHESIOLOGY* 54:131-135, 1981
11. Jouppila R, Jouppila P, Hollmen A, Kuikka J: Effect of segmental extradural analgesia on placental blood flow during normal labor. *Br J Anaesth* 50:563-567, 1978
12. Pritchard J, MacDonald P, Gant N: Hypertensive disorders in pregnancy, *Williams Obstetrics*, 17th edition. Edited by MacDonald P, Gant N. East Norwalk, CT, Appleton-Century-Crofts, 1985, pp 525-560

Anesthesiology
65:226-228, 1986

Maternal Heart Rate Changes with a Plain Epidural Test Dose

PHILIP D. CARTWRIGHT, M.B., CH.B., F.F.A.R.C.S.,* S. MÁIRE MCCARROLL, M.B., B.CH., F.F.A.R.C.S.,*
CHRISTINA ANTZAKA, M.D.*

The argument as to what constitutes an effective test dose for epidural anesthesia has continued for many years. Following initial recommendations,^{1,2} the use of test doses fell out of favor because the test doses used did not fulfill their function or were too time consuming.³ Some reports highlighted the value and limitations of the technique, and that the overall complication rate was not different

in centers where test doses were or were not employed.^{4,5,‡}

The purpose of an epidural test dose is to reveal accidental intrathecal and iv injection using a small dose of drug. Renewed enthusiasm for the use of test doses followed a statement by Moore and Batra⁶ that "for a single test dose of a local anesthetic solution to be of value . . . it must contain 0.015 mg epinephrine and a milligram dose of the local anesthetic drug which rapidly results in evidence of spinal anesthesia." The use of epinephrine as part of the test dose has subsequently been advocated extensively.⁷⁻¹⁰

A test dose containing 15 µg epinephrine will detect intravascular injection by causing a mean rise in heart

* Clinical Fellow.

Received from the Departments of Anaesthesia, Royal Victoria Hospital and McGill University, Montréal, Québec, Canada H3A 1A1. Accepted for publication March 24, 1986.

Address reprint requests to Dr. Cartwright: Department of Anaesthesia, Royal Victoria Hospital, 687 Pine Avenue West, Montreal, Quebec, Canada H3A 1A1.

Key words: Anesthetic techniques: epidural, obstetric. Sympathetic nervous system: epinephrine.

‡ Soni V, Peeters C, Covino B: Value and limitations of a test dose prior to epidural anesthesia. *Regional Anesthesia* 6:23-25, 1981.