Anesthesiology
53:348–349, 1980

Hypertension in a Paraplegic Parturient

To the Editor:—I read with interest the recent case report by Stirt *et al.*[1] Their patient was well managed, and she survived. I would like to report the case of another paraplegic patient in whom the outcome was not as favorable. This was the only such case we had at Magee-Womens Hospital among 36,257 deliveries in the last five years.

The patient, a 25-year-old black woman, had had a previous normal delivery in 1973. Paraplegia (T3) had developed in 1974 due to a gunshot wound in the neck. There was no definite history of autonomic hyperreflexia following the accident. She had had a chronic urinary tract infection with acute exacerbations. During her antepartum course arterial blood pressure had varied between 90/60 and 142/98 torr.

On October 26, 1978, at 10:20 A.M., the patient was admitted to the labor suite at 38 weeks' gestation and in active labor. The blood pressure on admission was 150/80 torr, and the patient complained of severe headache. Despite the fact that the lesion was above T10, the patient felt uncomfortable with the contractions. She received meperidine, 75 mg, and hydroxyzine, 25 mg, intramuscularly. Measurement of blood pressure every 10 minutes showed a gradual rise, which was associated with deterioration of the state of consciousness. At 11:55 A.M., blood pressure was 220/120 torr, and the patient became incoherent. There were neither convulsions nor signs of localization. The cause of the hypertension was assumed to be preeclampsia, as evidenced by hyperreflexia in the upper limbs. Accordingly, $MgSO_4$, 4 g, was administered by intravenous injection as a bolus, followed by 2 g every hour. Blood pressure decreased to 110/70 torr, and was then maintained at about 120/90 torr. Oxygen was given by face mask. At 1:50 P.M., owing to the comatose state of the patient and excessive secretions, nasotracheal intubation was performed, without difficulty and without anesthesia. At 2:05 P.M., mid-forceps were used to deliver a 2,420-g female infant with Apgar scores of 4 and 7. At the time of delivery, the maternal and fetal blood gases were as shown in table 1.

Post partum, lumbar puncture showed gross blood in the cerebrospinal fluid. The patient remained unconscious, and the CT scan showed bilateral cerebral hemorrhage. The neurosurgeon felt that she was not a candidate for surgery at the time, and she was kept under observation in the intensive care unit. Her condition continued to deteriorate, and she died two days later. Postmortem examination was declined by the family.

Robertson stated that, in contrast to the situation in paraplegia due to spinal-cord disease, labor is painless with traumatic paraplegia above the T10 level.[2] Our experience indicates that this is not always the case, but rather, that pain is related to the degree of severance of the spinal cord. It also affirms that spontaneous labor and adequate uterine contractions can still occur in a parturient with a cord transection above the motor nerve supply of the uterus (T5–T10). The presence of blood in the cerebrospinal fluid was probably due to the cerebral hemorrhage, proved by CT scanning, with blood gaining access to one of the ventricles.[3]

TABLE 1. Maternal and Fetal Blood Gases and Acid–Base Status at Delivery

	*p*H	P_{O_2} (torr)	P_{CO_2} (torr)	Base Excess (mEq/l)
Maternal arterial (100 per cent O_2)	7.27	333	24.2	−13
Umbilical venous	7.21	28	39.6	−12
Umbilical arterial	7.14	9	52.8	−12

The association of hypertension with paraplegia or quadriplegia is a special condition, although the most common cause in the parturient is preeclampsia. Eleven per cent of paraplegics were reported to experience hypertension during parturition despite the fact that the incidence of preeclampsia in paraplegic patients is not higher than that in the rest of the population.[2] The hyperreflexia of the upper limbs in our patient led the obstetrician to lean towards the diagnosis of toxemia of pregnancy. However, hyperreflexia is a sign that is difficult to quantitate and, in a paraplegic patient, only the upper extremities can be so evaluated. The absence of convulsions despite the progressive deterioration of the state of consciousness was a sign against eclampsia and in favor of an intracerebral lesion. Therefore, autonomic hyperreflexia should have been considered as the primary cause of hypertension. Ideally, a continuous epidural technique should have been instituted upon the patient's admission, as described by Stirt and associates,[1] to control the arterial blood pressure. Epidural analgesia may have other advantages over magnesium sulfate administration in such patients. For example, magnesium sulfate can lead to generalized muscle weakness, including weakness of muscles of ventilation, which is precarious in the quadriplegic patient. Also, in our case, the follow-up of the patient's state of consciousness was important; therefore, epidural analgesia would have offered better therapy than magnesium sulfate, which is a known central nervous system depressant.

EZZAT ABOULEISH, M.D.
Magee-Womens Hospital
Pittsburgh, Pennsylvania 15213

REFERENCES

1. Stirt JA, Margo A, Conklin KA: Obstetric anesthesia for a quadriplegic patient with autonomic hyperreflexia. ANESTHESIOLOGY 51:560–562, 1979
2. Robertson DNS: Pregnancy and labor in the paraplegic. Paraplegia 10:209–212, 1972
3. Isselbacher KJ, Adams RD, Brainwald E, et al: Harrison's Principles of Internal Medicine. New York, McGraw-Hill, 1980, p 1932

(Accepted for publication April 25, 1980.)

Anesthesiology
53:349, 1980

High Altitude and Intravenous Lidocaine

To the Editor:—In the May 1979 issue, Poulton and James concluded that the intravenous injection of lidocaine decreased cough reflexes in a group of subjects.[1] This information can be useful when an inhalational anesthetic is given with spontaneous ventilation by mask at high altitude.

In Albuquerque, the mean elevation is 5300 feet and the corresponding barometric pressure averages 635 torr. Therefore, a mask induction with nitrous oxide and a volatile agent may be relatively difficult. When 70 per cent nitrous oxide is used, the inspired partial pressure is reduced from approximately 530 torr (at sea level) to 445 torr.

Even after 2–4 mg/kg thiopental, the average mask induction with either nitrous oxide and oxygen or nitrous oxide, oxygen, and enflurane is accompanied by a higher incidence of coughing and bucking in patients than I have observed at sea level. The incidence of coughing on induction is even higher in patients with ventilation/perfusion abnormalities. Fortunately, these problems have been eliminated by the use of intravenously administered lidocaine during a mask induction of anesthesia with spontaneous ventilation.

In their study, Poulton and James used 1.5 mg/kg lidocaine and had satisfactory results.[1] In 1958, Steinhaus and Howland used approximately 4 mg/kg lidocaine over a 4–5 minute period to facilitate anesthetic induction.[2] They saw no electroencephalographic evidence of seizure activity when the lidocaine was given in the presence of a barbiturate.

I have success (lack of coughing during mask induction) in using 0.5–0.75 mg/kg lidocaine after a 2 mg/kg dose of thiopental. These drugs are used to supplement 70 per cent nitrous oxide and enflurane. The low dose of thiopental is used so as not to interfere with the patient's spontaneous respiration.

Steinhaus recommended intravenous administration of lidocaine for ear, nose, throat, and endoscopic procedures.[2] I would also use it for patients with ventilation–perfusion abnormalities, and for patients who are having ocular surgical procedures. The advantage of intravenously administered lidocaine as a supplement to general anesthesia is that it can be used not only to ease induction, but also in all procedures where it is imperative that bucking and coughing be avoided.

JEFFREY J. STEVENS, M.D.
1120 Bernalillo Place, S.E.
Albuquerque, New Mexico 87123

REFERENCES

1. Poulton TJ, James FM: Cough suppression by lidocaine. ANESTHESIOLOGY 50:470–472, 1979
2. Steinhaus JE, Howland DE: Intravenously administered lidocaine as a supplement to nitrous oxide thiobarbiturate anesthesia. Anesth Analg (Cleve) 37:40–46, 1958

(Accepted for publication April 25, 1980.)

Anesthesiology
53:349–351, 1980

Clinical Implications of Effects of Thoracic Epidural Anesthesia on Endocardial/Epicardial Flow Distribution Ratio Are Not Justified

To the Editor:—Klassen *et al.*[1] have provided the readers of the Journal with a beautiful pharmacologic and physiologic study of the effect of sympathectomy by means of continuous epidural anesthesia on *one aspect of the control of coronary circulation.* However, their conclusions, especially as regards the clinical care of patients with ischemic heart disease, are not justified from the results obtained in this highly artificial model. In their introduction they state:

> The purpose of our investigation was to examine the effect of reversible sympathectomy induced by epidural cervical anesthesia on myocardial blood flow distribution during myocardial