

played a role in the reversal of the shunt, because results of arterial blood-gas analysis 10 min prior to the onset of cyanosis were within normal limits.

In conclusion, if cyanosis appears suddenly with deterioration of P_{aO_2} immediately following the institution of positive-pressure breathing or PEEP in patients with left-to-right shunts, the possibility of reversal of intracardiac shunt should be suspected. In such situations, administration of 100 per cent oxygen may not increase arterial blood P_{O_2} , and addition of PEEP may worsen the arterial oxygenation. Therapy should be aimed at reducing RAP in comparison with LAP. This could be achieved by careful administration of vasodilators, which both reduce the preload to the right atrium by systemic venous dilatation and reduce the afterload of the right ventricle by producing pulmonary vasodilatation, thereby decreasing the shunting of blood from right to left.

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Obstetric Anesthesia for a Quadriplegic Patient with Autonomic Hyperreflexia

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The quadriplegic patient with autonomic hyperreflexia represents an unusual and complex array of medical and surgical problems, necessitating highly individualized anesthetic care for successful management.^{1,2} One case of labor and delivery in a quadriplegic patient complicated by autonomic hyperreflexia but without anesthetic intervention has been re-

ported.³ A single report of epidural anesthesia in a quadriplegic patient for urologic surgery exists.⁴ We report here what is to our knowledge the first documented case of labor and delivery in a chronic quadriplegic patient managed with epidural anesthesia.

REPORT OF A CASE

A 23-year-old quadriplegic woman, gravida 2, spontaneous ab 1, para 0, with an intrauterine pregnancy was admitted to the obstetrical unit for perinatal management and delivery. She had been quadriplegic since the age of 12 years following traumatic damage to the cervical spinal cord at the C5-6 level, leaving sensation to T4 intact. She had had an indwelling Foley catheter since that

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time, and the medical history included numerous episodes of urinary tract infection and pyelonephritis, the most recent a culture-proven *Pseudomonas aeruginosa* infection at 21 weeks of gestation, which had been treated successfully with tobramycin. The patient had manifested autonomic hyperreflexia on numerous occasions in the past: the most recent episode had occurred after inadvertent sustained clamping of the bladder catheter at 21 weeks' gestation, following which the blood pressure rose precipitously from a resting level of 70/35 to 280/220 torr, accompanied by anxiety, diaphoresis, tremors, and spasticity in all extremities.

On admission, the duration of pregnancy was estimated to be 34 weeks by dates and 36 weeks by sonography. She was in early labor, with uterine contractions every 8 min. Amniocentesis at this time revealed a lecithin/sphingomyelin (L/S) ratio of 1.9, an 80 per cent effaced, closed cervix, and the fetal head at +1/+2 station. Isoxsuprine administration successfully terminated the premature labor. After a week, repeated amniocentesis yielded an L/S ratio of 2.1.

The patient weighed 45 kg on admission, and suffered persistent nausea and vomiting over the ensuing week, losing 2.5 kg in weight. This necessitated peripheral hyperalimentation. Elective induction of labor and vaginal delivery were planned.

On the day of induction, a peripheral intravenous site was secured. A percutaneous radial arterial catheter and central venous pressure catheter were placed. A continuous epidural catheter was placed in the L3-4 interspace without difficulty, but was not activated. Preparations for infusion of 0.1 per cent trimethaphan were made. Amniotomy yielded slightly yellow-tinged fluid. A fetal scalp electrode and internal uterine catheter were placed. Two hours after rupture of the membranes, no effective labor pattern was noted, and intravenous administration of oxytocin was begun to augment uterine contractions.

The blood pressure varied from 70/35 torr between contractions to 130/80 torr with contractions. Three hours after amniotomy, blood pressure between contractions rose to 100/50 torr. Oxygen from a nonbreathing system was administered by mask, the patient placed supine on a right hip wedge, and the epidural analgesia was administered for the first time with a 2-ml test dose, followed by 6 ml of 0.25 per cent bupivacaine. There was no change in sensory level, and the blood pressure remained 100/50 torr in the face of increasingly stronger and more frequent uterine contractions.

Blood pressure between contractions gradually rose to 120/70 torr in the next 8 h, reaching values as high as 180/90 torr during contractions. Eight hours after the onset of labor, between contractions, which occurred every 5 min and lasted approximately 30 sec, the patient became apprehensive, and facial flushing, profuse diaphoresis, and coarse shaking tremors of the entire body developed, accompanied by a rise in blood pressure with her next contraction to 220/100 torr, along with slowing of the heart rate from 80 to 50/min. These symptoms and signs indicative of autonomic hyperreflexia persisted for 4 min after the contraction ended, when 15 ml of 0.5 per cent bupivacaine were administered. Within 2 min of administration of this dose of bupivacaine, the symptoms began to abate, even though another uterine contraction occurred, and blood pressure fell to 110/60 torr following this contraction, 5 min after bupivacaine administration. The frequency of bupivacaine supplementation was correspondingly increased in an attempt to mitigate these problems, and no further manifestation of autonomic hyperreflexia occurred. After 12 hours of labor, doses of 16 ml of 0.5 per cent bupivacaine were being administered hourly, with subsequent decreases in peak blood pressures from 220/100 to 170/90 torr. No change from the original sensory level of T4 was seen, nor was there any evidence of fetal distress. A total of 515 mg bupivacaine was administered over the 11-hour course.

The patient's temperature suddenly increased from 37 to 38 C nine hours after amniotomy, and remained elevated. Specimens of urine, blood, and amniotic fluid were obtained and sent to the laboratory for culture. Microscopic examination of the urine and amniotic fluid revealed no evident site of infection. Fetal scalp capillary blood samples had a pH of 7.36. Twelve hours after amniotomy, cervical dilatation was 5 cm, and it was felt that in the face of poor progression of labor and continued elevated maternal temperature, cesarean section was indicated. A final dose of 16 ml of 0.5 per cent bupivacaine was administered via the epidural catheter.

The patient was taken to the operating room, where a final vaginal examination found the cervix completely dilated and the fetal head at +3 station. The fetus was deemed deliverable vaginally, and outlet forceps were applied, resulting in the delivery of a 2,520-g male infant with 1- and 5-minute Apgar scores of 7 and 9. The patient's blood pressure returned to pre-labor levels of between 100/50 and 70/35 torr within 12 hours of delivery. Administration of antibiotics was followed by rapid defervescence, and the patient was discharged on the fourth day after delivery.

DISCUSSION

The clinical course of this patient illustrates many of the known features peculiar to the physiology of quadriplegic and paraplegic patients, the most important of these being the constellation of signs and symptoms known as autonomic hyperreflexia, or "mass reflex." First described in 1917, the syndrome is manifested by pilomotor erection, sweating, facial flushing, severe headache, and bradycardia, with blood pressure rapidly reaching precipitous heights, leading to loss of consciousness and convulsions.⁵ The syndrome has not been observed to occur when the lesion is below T7. Although more than 85 per cent of patients who have injuries above this level experience it, others do not, for reasons unknown.⁶ Retinal and fatal cerebral or subarachnoid hemorrhages have been reported to occur subsequent to the sudden rise in systolic blood pressure.⁷

Autonomic hyperreflexia is explained on the basis that afferent impulses, somatic and visceral sensory, enter the isolated cord and initiate segmental reflexes that are neither modulated nor inhibited by higher centers.² The reflexes have to a great extent lost their local responses, and the reaction is massive rather than specific, hence the term "mass reflex."¹ Stimulation of the skin below the level of the lesion or distention and contraction of a hollow viscus such as the bladder, gut, or uterus precipitates a massive stimulation of the isolated adrenal medulla and sympathetic nervous system.⁸ In addition, vascular supersensitivity in quadriplegic patients may exacerbate the problem.⁹ Because the cord transection does not interfere with the afferent connections of the baroreceptors and slowing of the heart rate via the vagus nerve, the only defense against the uncontrolled hypertension is bradycardia.¹⁰

The case presented exemplifies many of these points. Knowing this patient had had autonomic hyperreflexia in the past due to bladder distention, we sought to prevent its recurrence insofar as was possible by blocking the afferent and efferent limbs of the reflex. Failing this, we were prepared to control the blood pressure with the ganglionic-blocking agent trimethaphan, which has been used successfully in quadriplegic patients for control of blood pressure during anesthesia.¹¹

We sought a balance between control of the acute blood pressure rises during contractions and the much lower levels between contractions in order not to jeopardize uterine perfusion and fetal well-being. Thus, we were reluctant to increase our anesthetic dosage further or to institute trimethaphan to lower the blood pressure any further.

We were obviously unable directly to monitor sensation in this patient below T4 although observing changes in specific segmental reflex activity and spasticity below this level might have yielded an indirect indication of the height of anesthetic block. We simply sought to maintain our block below T4 and determined its effectiveness by the patient's blood pressure and overall clinical appearance. We originally used small doses of local anesthetic, gradually increasing the dose as necessary so long as no sign of toxicity or increased sensory level occurred.

Both spinal and epidural anesthesia were considered as part of the management plan. Spinal anesthesia has been reported to prevent autonomic hyperreflexia in quadriplegic patients undergoing various types of surgical procedures.¹² On the other hand, spinal anesthesia in paraplegic and quadriplegic patients is considered dangerous by some authorities because control of the level of block is often unpredictable due to inherent distortion of the vertebral column, and severe hypotension can occur.^{1,11} Poor vascular tone and hypovolemia exaggerate the problem.¹³

Labor and vaginal delivery had been deemed the safest course for this patient, and a one-shot spinal anesthetic administration was impractical for a prolonged period of reflex control. Thus, we elected to manage the labor and delivery with continuous epidural anesthesia.

Bupivacaine was chosen as the anesthetic agent because of its prolonged duration of action. Because of its high degree of ionization and binding to plasma proteins, placental transfer is minimized.¹⁴ Although

the patient weighed 43 kg at the time of labor and delivery, and the maximal safe dose for the average 70-kg individual is approximately 500 mg,¹⁵ no toxic manifestation was seen with the use of a total of 515 mg over 11 hours.

In summary, labor and delivery of a quadriplegic patient with a spinal cord fracture-dislocation at C5-6 were successfully managed using epidural anesthesia. An understanding of the physiologic alterations inherent in such patients is essential for a rational and safe anesthetic course.

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