

Fetal Bradycardia during Spinal Anesthesia in a Parturient with Oligohydramnios

To the Editor:—For several years it has been our practice to monitor the fetal heart rate (FHR) during induction of spinal or epidural anesthesia for cesarean section. The following case illustrates the utility of such monitoring in a parturient with oligohydramnios (*i.e.*, decreased amniotic fluid volume).

A 26-yr-old woman, gravida 3, para 2, was referred to the University of Iowa Hospitals and Clinics at 28 weeks gestation with oligohydramnios. The patient had a history of previous cesarean section complicated by postoperative deep venous thrombosis. Physical examination included a weight of 83 kg and a height of 173 cm. Ultrasound examination confirmed the presence of oligohydramnios and fetal breech presentation, and suggested that the fetal kidneys were small and perhaps dysplastic. The patient was admitted for bed rest, prophylactic subcutaneous heparin administration (*i.e.*, 7500 U every 12 h), and daily FHR monitoring.

Umbilical cord blood was obtained percutaneously, and fetal blood chromosome analysis was normal. Daily FHR monitoring revealed occasional spontaneous FHR decelerations. At 32 weeks gestation, FHR decelerations occurred more frequently. Although there was uncertainty regarding fetal renal function, both the obstetricians and patient concurred with a decision to perform elective cesarean section for fetal indications. Heparin was discontinued, and subsequent platelet count, PT, and PTT were 196,000, 11 s, and 31 s, respectively.

In the operating room the patient was given 30 ml of 0.3 M sodium citrate orally, and 2000 ml of Ringer's lactate intravenously. Continuous external FHR monitoring revealed a rate of approximately 150 beats per min with normal variability. The patient was placed in the right lateral decubitus position, a 25-G needle was used to identify the subarachnoid space, and the patient received 12 mg of hyperbaric bupivacaine with 0.2 mg of preservative-free morphine. The patient was then positioned supine. Left uterine displacement was accomplished by placing a folded blanket beneath the right buttock and tilting the operating table leftward. Immediately thereafter, abrupt and profound fetal bradycardia occurred (fig. 1). Maternal blood pressure and heart rate remained stable at 110/60 mmHg and 80 beats per min, respectively. Anesthesia was adequate for surgery, and the obstetricians proceeded with immediate low transverse cesarean section. Approximately 8 min after the onset of fetal bradycardia, a 1,460-g male infant was delivered. The infant's trachea was intubated immediately after delivery. Apgar scores were 2 and 4 at 1 and 5 min, respectively. Umbilical cord venous blood analysis included a pH of 7.324, a P_{O_2} of 31 mmHg, a P_{CO_2} of 42 mmHg, and a base excess of -3.1 mEq/l. Umbilical cord arterial blood analysis included a pH of 7.066, a P_{O_2} of 3 mmHg, a P_{CO_2} of 89 mmHg, and a base excess of -6.1 mEq/l. These results were compatible with acute cord compression and respiratory acidosis.

The mother did well and was discharged on the fourth postoperative day. Unfortunately, it was not possible to adequately ventilate the lungs and oxygenate the infant, and chest x-ray suggested the presence of hypoplastic lungs bilaterally. Ultrasound examination of the infant suggested renal agenesis, and the infant died 3 h after delivery. Autopsy confirmed the presence of bilateral renal cystic dysplasia and pulmonary hypoplasia.

Given the reassuring FHR tracing obtained before induction of anesthesia, it would have been tempting to forego FHR monitoring during induction of spinal anesthesia. But the decreased amniotic fluid volume placed the umbilical cord at increased risk for compression. The abrupt fetal bradycardia undoubtedly resulted from umbilical cord

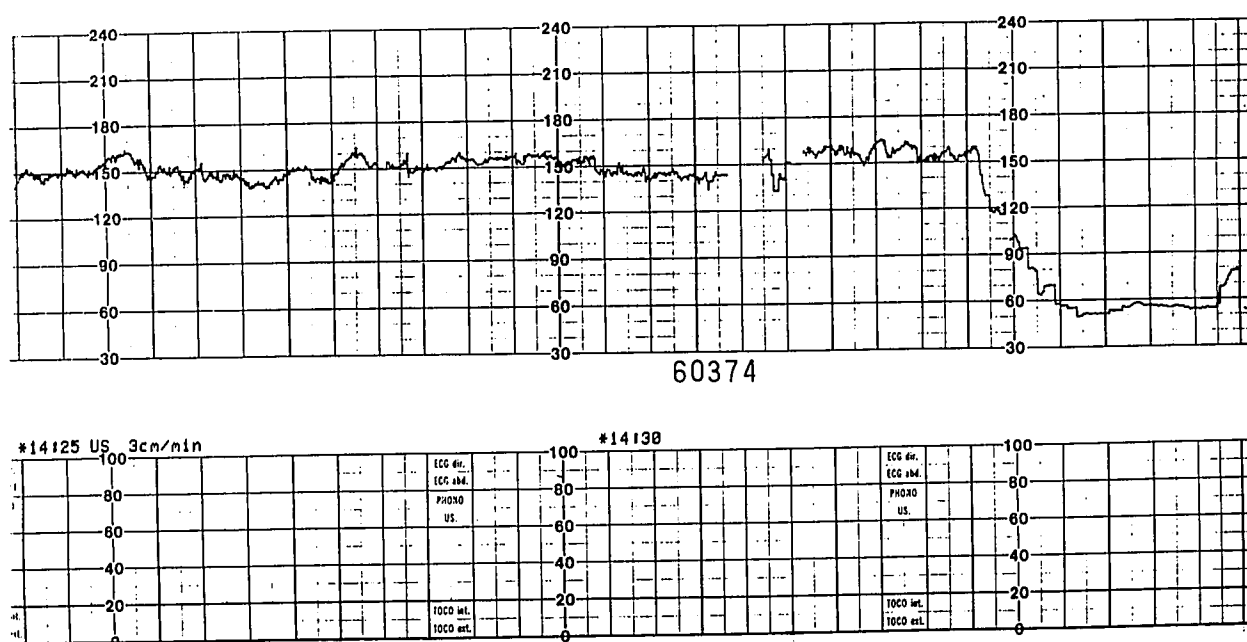


FIG. 1. Fetal heart rate tracing before, during, and after intrathecal injection of bupivacaine and morphine. The fetal bradycardia occurred immediately after the patient turned from the right lateral decubitus position to the supine (leftward tilt) position. Monitoring was discontinued at the right edge of the figure to allow preparation of the abdomen and performance of surgery.

compression that occurred at the time of the change in maternal position. Electronic FHR monitoring allowed immediate detection of the fetal bradycardia, which prompted expeditious delivery before the development of fetal hypoxia and metabolic acidosis. This case illustrates the utility of FHR monitoring during induction of regional anesthesia for cesarean section, especially when oligohydramnios is present.

Anesthesiology
71:804, 1989

DAVID H. CHESTNUT, M.D.

*Departments of Anesthesia and Obstetrics and Gynecology
University of Iowa College of Medicine
Iowa City, Iowa 52242*

(Accepted for publication July 14, 1989.)

A Dangerous Defect in a Heat and Moisture Exchanger

To the Editor:—A heat and moisture exchanger (HME) can be a valuable addition to the anesthetic circuit by preventing heat loss and protecting the large airway mucosa from drying. The following is a report of a case in which a defective HME nearly caused serious complications.

A previously healthy 31-yr-old female came to the emergency room at 2:00 A.M. with pelvic pain and an adnexal mass. Vital signs were stable with an hematocrit of 34 after 2 liters of iv fluid. Blood pressure was 115/65, pulse 90. Following preoperative medication with midazolam 1.25 mg iv and metaclopramide 10 mg iv, she was brought to the operating room where a rapid sequence induction using thiopental and succinylcholine was used. Anesthesia was maintained with isoflurane and O₂, and atracurium was given for muscle relaxation. Pulse and blood pressure remained stable. An HME (ICOR, AB, Sweden) was inserted between the elbow of the anesthesia circuit and the endotracheal tube.

Within a few minutes blood pressure decreased to 67/45. By this time the pelvic cavity had been entered and a ruptured ectopic pregnancy had been discovered, but there was no active bleeding. It was assumed that the patient was hypovolemic. Intravenous fluids were increased and the isoflurane (1.5%) was discontinued. The ventilator was turned off and the lungs were ventilated by hand. It was immediately apparent that increased effort was required. The pressure gauge showed a rapid rise to 40 cm H₂O followed by a rapid decline in pressure. Breath sounds were decreased but present bilaterally with a markedly prolonged expiratory phase. No wheezing was heard.

The inspiratory and expiratory valves were functioning. The pulse oximeter showed 100% saturation. The CO₂ waveform on the mass spectrometer was narrowed and decreased in amplitude. Air trapping due to a plugged endotracheal tube was assumed, and it was decided to reintubate the trachea. When the circuit was disconnected between the HME and the endotracheal tube, a large amount of air rushed out of the endotracheal tube as if under pressure. The HME was removed and the circuit reconnected to the endotracheal tube. The lungs were easily ventilated and the blood pressure immediately returned to 120/60. The case proceeded uneventfully without any evidence of pulmonary trauma.

The HME was found to have a circular plug of plastic inside the chamber between the heat exchanging material and the expiratory limb (fig. 1). This had been acting as a one-way ball valve causing partial air trapping in the lungs. The resulting high intrathoracic pressure caused hypotension due to decreased cardiac filling.

This case points out the value of discontinuing mechanical ventilation immediately upon the onset of sudden hypotension. This maneuver may improve cardiac filling in cases of hypovolemia, and in this case, it allowed the anesthesiologist to more rapidly diagnose a problem in the breathing circuit.

HMEs should be inspected before insertion for the same reason we inspect the anesthesia machine and circuit.

(The author wishes to thank David Haskin, Chief of Medical Media, and Dr. Bert Myers at the V.A. Medical Center in New Orleans for performing the x-ray photography.)

WILFRED PRADOS, M.D.

*Staff Anesthesiologist
St. Jude Medical Center
Kenner, Louisiana
Staff Anesthesiologist
Veterans Administration Medical Center
New Orleans, Louisiana
Assistant Professor of Anesthesiology
Louisiana State University
New Orleans, Louisiana
200 West Esplanade Avenue
Suite 304
Kenner, Louisiana 70065*

(Accepted for publication July 18, 1989.)

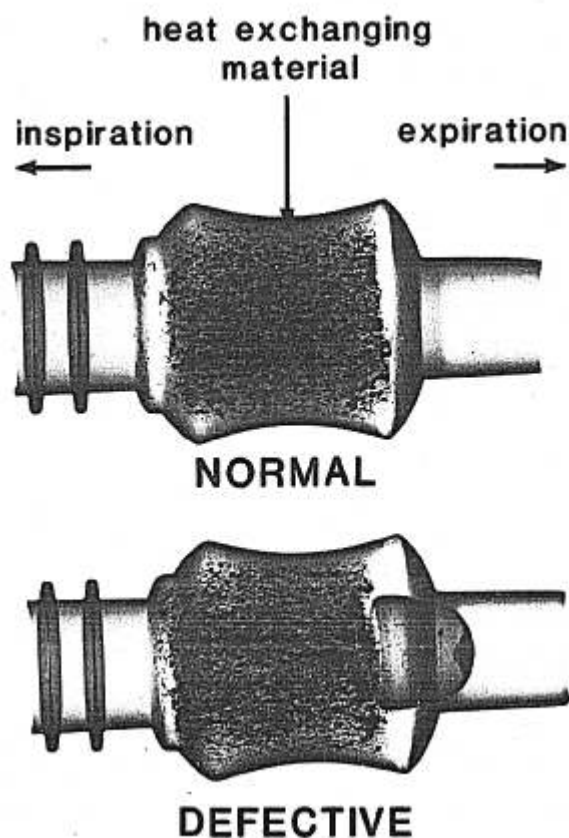


FIG. 1. X-ray photograph showing the plastic plug obstructing the expiratory limb of the HME.