

## DISCUSSION

Failure of a single-dose spinal anesthetic following a satisfactory spinal tap is not unusual. It is often attributed to movement of the needle after aspiration of cerebrospinal fluid, but before injection of the anesthetic, or to the bevel of the needle's being only partially within the subarachnoid space. These errors are not possible explanations when a catheter is positioned in the subarachnoid space such that cerebrospinal fluid can be aspirated from it at will.

Dripps, in a series of 506 continuous spinal anesthetics utilizing a catheter, reported 43 failures.<sup>1</sup> Eighteen had no anesthesia at all, and in a few instances (exact number not indicated) fluid either could be withdrawn via the catheter or would drip from the catheter. Dripps speculated that this resulted from leakage of cerebrospinal fluid into the epidural space and placement of the catheter in that space. He also reported seven cases in which the first drug injected (tetracaine four times, procaine three times) produced no anesthesia, whereas injection of a second local anesthetic agent did produce anesthesia. It was speculated that the initial failures in the latter cases

were due to drug resistance. Neither of these explanations is possible in the case reported here.

That the catheter was indeed within the subarachnoid space was amply demonstrated by the ease of aspiration and brisk flow of cerebrospinal fluid, the elicitation of a parathesia, and the radiographic study. The small deviation from normal of the pH of the cerebrospinal fluid is insufficient to explain this failure. Resistance of this individual to tetracaine was excluded by skin testing. Other uremic patients have had adequate spinal anesthesia at this institution. The reason for failure of tetracaine introduced into the subarachnoid space to provide anesthesia for this man on two separate occasions remains unexplained. Perhaps some of the failures of single-dose spinal anesthetic which we now attribute to the above-mentioned reasons result from the same unelucidated cause.

## REFERENCE

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## Anesthetic Management for Surgical Separation of Thoracopagus Twins

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Through 1967, 24 attempts to separate conjoined twins had been recorded.<sup>1-11</sup> An excellent review of anesthetic management of conjoined twins has been published by Keats *et al.*<sup>12</sup> We hope that our report will be of value to others who have to manage such cases, just as the experience of others was of tremendous help to us.

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## REPORT OF A CASE

Female conjoined twins were born on January 12, 1969 to a para III woman (without antenatal observation). The labor was conducted by a village midwife (who was unaware of the twin pregnancy) without trauma to the mother or twins. Delivery was at full term, with cephalic presentation. The twins were admitted to All-India Institute of Medical Sciences Hospital on January 13, 1969, where they were studied.

*Medical History.* The twins were joined from xiphisternum to umbilicus, the circumference of their union being 21 cm (fig. 1). No other defects or abnormalities were seen. Their combined weight on admission was 4,750 g. Blood values were within normal limits. Roentgenograms showed separate hearts and gastrointestinal tracts. Twin

I, the first born, was slightly weaker than Twin II and had dextrocardia. Liver scan with radioactive rose Bengal showed a common hepatic mass, with considerable crossover circulation. While separation was being planned, the twins developed renal vein thrombosis, malnutrition and weight loss. Blood urea was elevated (159–436 mg/100 ml). Surgical operation was postponed and energetic treatment, which included exchange transfusions, was instituted. The twins gradually improved and put on weight. They were separated July 13, 1969, at the age of six months.

*Pre-operative Assessment and Management.* Certain mechanical problems were present because of the face-to-face position of the twins. We anticipated difficulties during tracheal intubation, marked cervical hyperextension due to consistent pulling of the twins away from each other (fig. 2), some degree of jaw recession, and the hazard of lifting one twin over the other to facilitate intubation, which could lead to hypovolemia and hypotension in the upper twin.

The twins had had renal vein thrombosis with consequent renal malfunction, and in the choice of anesthetic technique we had to take account of this. At the time of surgery, blood urea was elevated (66 mg/100 ml). The joint weight of the twins at the time of separation was 8,400 g. Twin I was still slightly weaker in build than Twin II, and had dextrocardia and a pulmonic systolic murmur due to rotation of the heart, although pulmonary stenosis was a possibility. Because hepatic union was extensive, considerable hemorrhage was anticipated during division of the liver. Despite a crossover circulation between the twins through the liver bridge, it was anticipated that administration of anesthetic to one twin would not anesthetize the other. We felt that general anesthesia would be the best available method. Tracheal intubation was essential because of the possibility of opening pleurae during separation of the common chest wall. Even if the pleurae were not opened, the possibility of paradoxical respiration due to disruption of the integrity of the chest wall and abdomen was kept in mind. Breathing difficulties both during the surgical operation and in the postoperative period were anticipated. Since spontaneous breathing would have led to tremendous energy expenditure with inefficient ventilation, possibly paradoxical, it was considered desirable to apply intermittent positive-pressure breathing during the procedure. As reported by Aird,<sup>4</sup> one of the twins may be dominant in providing corticosteroids to both, and after separation the other twin may have adrenocortical insufficiency. Therefore, each twin was given 15 mg hydrocortisone intramuscularly the day prior to separation and on the morning of operation. They also received 0.2 mg atropine and 5 mg promethazine each, intramuscularly, an hour before induction of anesthesia. Twin II, who was the healthier of the two, had a cutdown performed under local anesthesia and a PVC venous



FIG. 1. The twins shortly after birth.

catheter was inserted in the femoral vein. This was connected to a 2.5 per cent glucose drip. Before the cutdown of the smaller Twin I, it was decided to anesthetize the twins. We planned to intubate them while conscious, so that adequate time would be available for an atraumatic, unhurried intubation, without the hazards of hypoxia due to anesthesia or muscle relaxants.

*Conduct of Anesthesia.* After application of xylocaine viscous to the epiglottis and inlet of the larynx with a surgically clean finger, under gentle restraint, Twin II was laid supine, slightly lifting the other, but without an actual face to face position, to avoid hypotension in the elevated twin. The larynx was visualized and a non-cuffed Rusch plastic endotracheal tube was atraumatically inserted in it, followed by placement of a thin Foley catheter in the esophagus to act as an esophageal stethoscope. A small sterile gauze pack rinsed in Reverin (rolitetracycline, Hoecht) in saline solution was lightly placed in the pharynx around the tube, which was securely fixed to the face with adhesive tape. The intubated twin was then anesthetized with nitrous oxide, oxygen and halothane. Conscious intubation followed by general anesthesia was then carried out in a similar manner in the other twin. The intubation and induction of anesthesia was easily accomplished without hypoxia or trauma. Although nitrous oxide and halothane had been administered to Twin II before intubation of Twin

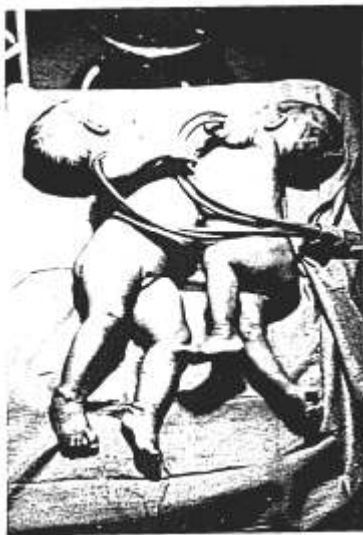


FIG. 2. The twins immediately before separation.

I, the latter had remained fully conscious. Cut-down similar to that in Twin II was performed in the other twin and 5 mg thiopentone was administered to each. The twins were then paralyzed with *d*-tubocurarine, Twin I receiving 1 mg and Twin II, 1.5 mg. Controlled ventilation with an Ayres T piece and a double-ended bag without soda lime absorber, using a flow of 6 l/min (3 l oxygen, 3 l nitrous oxide) was administered to each twin, using two separate Boyle's anesthesia machines. Two anesthetic teams took charge of the twins. The lower portion of the thoracic cage at the xiphoid level had to be cut through and the lungs within their intact pleural cavities were visualized after the conjoint portion of the diaphragm was incised. Pleurac were not opened in either twin. After separation of soft structures along the anterior abdominal wall and the fused thoracic cage, the common liver bridge was divided between liver tourniquets. There was brisk hemorrhage, which was controlled by pressure, sutures and gelfoam. Blood loss was replaced as it occurred. The separation was then completed by division of posterior junctional areas and the twins were separated. Until separation of the liver, blood pressures and heart rates of both twins remained satisfactory, varying between 80 and 130

mm Hg systolic and 110 and 150 beats/min, respectively. The condition of the smaller twin after section of the liver caused some anxiety, due to a fall of systolic blood pressure to 60 mm Hg. This responded to blood replacement. Both twins were maintained on positive-pressure breathing throughout the procedure, apnea being produced by *d*-tubocurarine and hyperventilation. Small increments of *d*-tubocurarine (0.5-1 mg) and thiopentone (2-3 mg) were given as required. Anesthesia was maintained with nitrous oxide, oxygen and traces of halothane.

Decurarization was achieved by intravenous administration of 0.1 mg atropine and 0.2 mg prostigmine to Twin I and 0.1 mg atropine and 0.25 mg prostigmine to Twin II. The babies opened their eyes and made feeble attempts to cry 30 minutes after completion of the separation, when the endotracheal tubes were removed after oxygenation and gentle suction. Intermittent oxygen was given from a plastic hood. Throughout the surgical operation vital signs were satisfactory, and blood pressure was well maintained except for the brief period after section of the liver bridge when Twin I showed moderate hypotension, which responded satisfactorily to blood replacement and intravenous administration of a small dose of a vasopressor and a corticosteroid.

**Monitoring.** The following monitoring was done:

**Temperature:** A rectal thermistor was inserted in each twin and connected to a multiple-channel compensated thermocouple. The twins were kept on a mattress connected to a hypothermia-hyperthermia unit, through which water at 40 C was circulated. The temperatures of the twins remained between 37 and 38 C.

**Arterial pressure:** Arterial pressure was monitored by pediatric blood pressure cuffs attached to separate manometers. Systolic blood pressures ranged between 80 and 130 mm Hg.

**Cardiac action:** Cardiac activity (QRS) was monitored in the twins by two Burdick Telecor monitors, with needle electrodes. These gave both visual and auditory signals. Heart rates ranged from 110 to 150 beats/min in both twins, and there were no arrhythmias. Cardiac sounds were also auscultated through the esophageal stethoscope.

**Blood loss:** Blood loss was measured by Beckman's blood loss monitor, model BLM-2. A running account of blood loss was maintained. This was a great help in replacing blood loss as it occurred, maintaining the replacement slightly ahead of the loss. Total blood loss was approximately 760 ml before separation. No blood loss occurred after the liver margins had been approximated. Twin I received 350 ml of blood, whereas Twin II received 290 ml. In addition, Twin I received 20 ml of soda bicarbonate (7.5 per cent) solution and 80 ml of Ringer's lactate solution. Twin II received 25 ml of 7.5 per cent soda bicarbonate, and 60 ml of Ringer's lactate solution.

**Postoperative Management and Course.** The separated twins were kept on two thermostatically controlled incubator cots, fitted with Kreiselman's infant resuscitators in an isolated air-conditioned nursery. Hydrocortisone (10 mg) was administered to each twin at four-hour intervals during the first 24 hours. A total of 150 mg of hydrocortisone was injected into the twins during operation and in the following 24 hours. The rationale of administering corticosteroids to infants was based on the assumption that one of the twins might have been dominant in providing cortisone for both; the other twin might then have had adrenocortical deficiency after separation. Furthermore, the anti-inflammatory action of steroids would help prevent laryngeal reaction following tracheal intubation and would help during stress. During the postoperative period the respiration, blood pressure, color and general condition of the twins were carefully watched. Oxygen (3 l/min) was administered through a plastic oxygen hood for an hour at two-hourly intervals. The oxygen in the hood was kept at around 40 per cent. Urinary output, electrolyte and fluid balance were attended to.

During the first 24 hours of the postoperative period, Twin II had a slight elevation in blood pressure to about 140-170 mm Hg, systolic. Both twins also had elevated blood urea, considered to be due to the pre-existing renal damage consequent to the renal vein thrombosis from which the twins had recovered.

The separated infants were kept for two days in a slightly head-up position to prevent collapse of the upper lobes of the lungs. Postoperative roentgenographic studies of the chest showed no areas of un-aerated lung. The postoperative period gave no cause for anxiety. Both children are hale and hearty and are growing satisfactorily.

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## Obstetrics and Pediatrics

**FETAL ACIDOSIS AND HEART RATE** Fetal base deficit values were correlated with fetal heart rate patterns in 139 fetuses of mothers in active labor. Normal fetal heart rates and early deceleration "type 1 dip" (thought to be due to head compression) were not associated with any appreciable abnormality in fetal pH. Mild and moderate variable deceleration (thought to be due to intermittent cord compression) and mild late deceleration "type 2 dip" (due to uteroplacental insufficiency) were associated with increases in fetal base deficit. Severe variable, moderate, and severe late deceleration patterns were associated with more pronounced increases in fetal base deficits. These fetal acid-base findings support the etiologic and prognostic significance assigned by the author to different fetal heart rate patterns. High correlations were seen in babies with low Apgar scores where ominous fetal heart rate patterns and fetal acidosis were both present. (Hon, E. H., and Khazin, A. F.: *Observations on Fetal Heart Rate and Fetal Biochemistry. I. Base Deficit, Amer. J. Obstet. Gynec.* 105: 721 (Nov.) 1969.)