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The EXIT Procedure Facilitates Delivery of an Infant with a Pretracheal Teratoma

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THE *ex utero* intrapartum technique (EXIT) procedure allows for the continuance of fetoplacental circulation during cesarean section. Initially, only the infant's head and shoulders (but not the placenta) are delivered, thus maintaining uteroplacental blood flow. After the infant's airway is secured, the umbilical cord is clamped and delivery of the infant is completed. It has proven useful in cases of anticipated difficult airway instrumentation of the neonate (*e.g.*, large fetal neck masses causing airway obstruction).[1] The authors report a case of an antenatally diagnosed, large pretracheal teratoma, wherein the EXIT procedure was used to secure the infant's airway during cesarean section performed during general anesthesia.

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Case Report

During a routine ultrasonographic examination, a 29-yr-old woman, gravida 3, para 2, weighing 84 kg, with no significant medical history was noted at 24 weeks' gestation to have a fetus with a cystic structure on the anterior neck. The mass was consistent with teratoma. No other fetal anomalies were noted. Increasing hydramnios was noted between 26 and 32 weeks' gestation. Consequently, preterm contractions developed, which were treated successfully with terbutaline subcutaneously. Ultrasonography at 31 weeks' gestation showed that the mass increased in size to 8.6 × 7.8 cm. In anticipation of difficulty with securing the infant's airway, the decision was made to perform the EXIT procedure when delivery became imminent.

The patient was admitted at $32^{6/7}$ weeks' gestation with increased preterm contractions. Expectant management with terbutaline subcutaneously and nonstress testing, in addition to two doses of betamethasone intramuscularly, was continued until $33^{3/7}$ weeks' gestation at which time cervical dilation was 5 cm. The decision was made to proceed with delivery.

Preoperatively, 20 mg famotidine and 10 mg metaclopramide were administered intravenously and 30 ml sodium citrate was administered orally. At arrival in the operating room, the mother was positioned supine with left-sided uterine displacement. Standard noninvasive monitors were applied.

After preoxygenation and abdominal preparation, rapid-sequence induction of anesthesia was performed with 300 mg thiopental and 120 mg succinylcholine intravenously. A 6.5-mm endotracheal tube was placed without difficulty *via* direct laryngoscopy. Isoflurane in 100% oxygen was administered (~2.8% end-expired concentration). A second 18-g intravenous catheter was placed in the right upper extremity after induction of anesthesia. An intravenous infusion of angiotensin II (2.5 μg/ml in saline) was started immediately after induction

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and adjusted to maintain the maternal systolic blood pressure (BP) greater than 100 mmHg. Infusion rate ranged from 10 to 55 ng · kg^{-1} · min^{-1}. The patient's skin color remained pink with no generalized apparent blanching, despite the high infusion rates of angiotensin II, and a good waveform was noted on the pulse oximeter. Controlled ventilation of the mother was maintained during the delivery. Maternal arterial oxygen saturation remained at 100% during the entire procedure. Maternal end-tidal carbon dioxide (ET_{CO_2}) ranged from 20–34 mmHg during the surgery. Paralysis was maintained with intravenous cisatracurium and monitored using a peripheral nerve stimulator.

Delivery of the infant's head and shoulders was accomplished 5 min after skin incision. Fetal pulse oximetry was attempted but was unsuccessful because of nonadherence of the probe. Therefore, the infant's circulation was monitored continuously by palpation of the umbilical cord and precordium by one of the obstetricians. The infant's heart rate ranged between 130–140 beats/min during the period when the airway was being secured. Several attempts at endotracheal intubation by a neonatologist were unsuccessful. Only the tip of the epiglottis was visualized and blind passage of the endotracheal tube could not be accomplished. Attempts at rigid bronchoscopy by the pediatric surgeons also were unsuccessful.

The decision was then made to proceed with tracheostomy. This proved to be difficult because of the distorted anatomy, but was nevertheless accomplished 51 min after the delivery of the head and shoulders. A noncuffed 3.0 endotracheal tube was placed into the tracheostomy site and ventilation was confirmed. The cord was then clamped and delivery of the female infant was completed. Apgar scores were 4/5/10 at 1, 5, and 10 minutes, respectively. The umbilical blood gas tensions were obtained after ventilation of the infant. The cord was not clamped until after confirmation of the endotracheal tube position was accomplished. Umbilical blood gas and acid-base measurements revealed arterial *p*H: 7.23; partial pressure of carbon dioxide (P_{CO_2}): 56 mmHg; partial pressure of oxygen (P_{O_2}): 41 mmHg; and bicarbonate (HCO_3): 22 mM; and venous pH: 7.37; P_{CO_2} 37 mmHg; P_{O_2}: 68 mmHg; HCO_3: 21 mM.

Immediately after umbilical cord clamping, the angiotensin II infusion was discontinued, the inspired concentration of isoflurane was decreased to less than 0.5%, nitrous oxide was added (fractional inspired oxygen tension [Fi_{O_2}] of 34%) and 250 μg fentanyl was administered intravenously. Pitocin (80 units in 1,500 ml of lactated Ringer's solution) intravenously, 0.2 mg methyl ergonovine maleate intramuscularly, and 250 μg carboprost methanine intramuscularly were administered to achieve uterine contraction. A second dose of 0.2 mg methyl ergonovine maleate intramuscularly was administered given 21 min after the first dose when the uterus remained distended. After the surgical repair was completed, 3.5 mg neostigmine and 0.6 mg glycopyrrolate were administered intravenously. The patient was awakened and extubated uneventfully.

The infant was transferred to Children's Hospital where resection of the tumor was performed uneventfully 2 days later. At the completion of the procedure, oral tracheal intubation was performed and the tracheostomy site was closed. Pathology showed that the tumor measured 12.8 × 9.5 × 7.5 cm and weighed 368 g (fig. 1). The microscopic examination revealed a teratoma composed of all three germ layers (*i.e.*, ectoderm, mesoderm, and endoderm). Subsequently, a tracheostomy was necessary because of failed extubation secondary to right vocal cord paralysis and tracheomalacia. The infant is at home doing well.

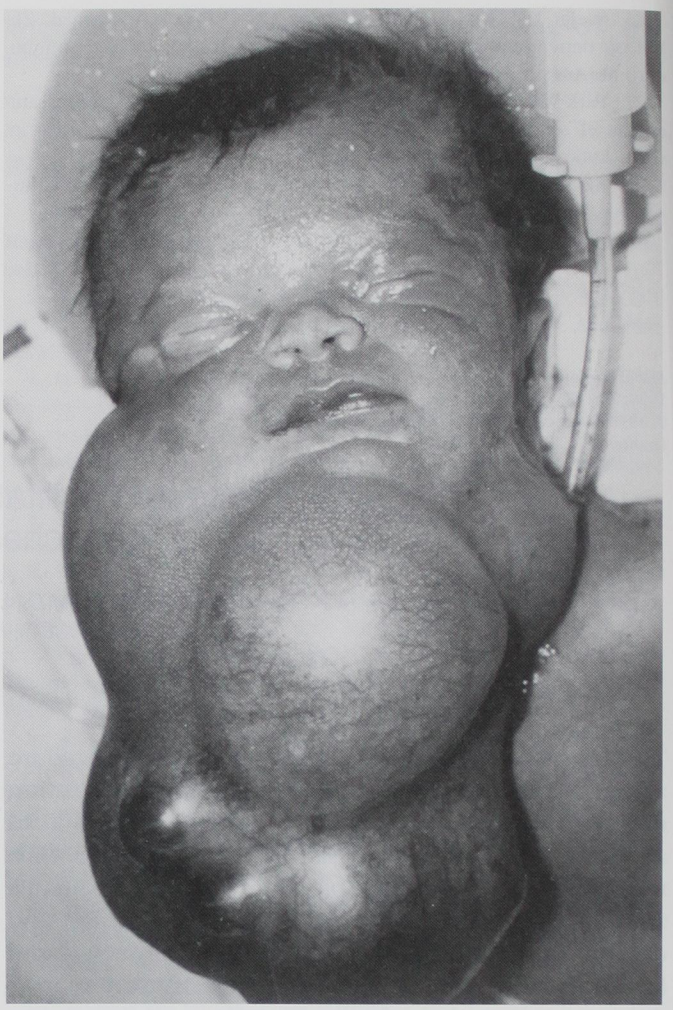

Fig. 1. Newborn infant with an anterior neck teratoma delivered *via* the *ex utero* intrapartum technique procedure

Discussion

Pretracheal masses diagnosed *in utero* can cause airway obstruction and death in newborn infants. First described in 1990 by Zerella and Finberg[2] for tracheal obstruction in a neonate and Levine *et al.*[3] for epignathus, the EXIT procedure has been refined by Mychaliska *et al.*[1] after their experience with eight newborns and their review of several case reports. A variety of masses, in addition to laryngeal stenosis, was diagnosed antenatally by ultrasonography. These masses included cystic hygroma, cervical or oropharyngeal teratomas, or both, and hemangiomas.[1–11] The EXIT procedure has proven to be safe and efficacious, allowing establishment of an airway in a controlled manner as the placenta allows continued gas exchange during airway manipulation.

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The profound uterine relaxation achieved by deep inhalational anesthesia may allow up to 60 min of fetoplacental circulation for the operative procedure on the infant.[1] In contrast, fetoplacental circulation may persist for as few as 5 min after delivery of a neonate in unanesthetized women.[5,12] Mychaliska *et al.*[1] achieved adequate uterine relaxation when using 2 to 3% isoflurane in oxygen. These concentrations of volatile anesthetics frequently will result in maternal hypotension, which may result in fetal compromise. Therefore, vasopressors are administered to increase BP and to help restore uteroplacental perfusion. The uterine vascular bed is substantially more responsive to α-adrenergic agonists than is the systemic circulation.[1] Pure α-adrenergic agonists such as phenylephrine may elevate BP without increasing uterine blood flow. Ephedrine, because of its combined α and β, direct and indirect effects, has been the obstetric vasopressor of choice, even though it is associated with occasional fetal acidemia, despite normalization of maternal BP.[14] In pregnant animals, angiotensin II causes less vasoconstriction in uterine vessels than in other systemic blood vessels, and recent studies indicate that the incidence of fetal acidemia is not increased when angiotensin II is used to prevent maternal hypotension during spinal anesthesia for cesarean delivery.[15] Angiotensin II does not cross the placenta.[14]

Based on these considerations, angiotensin II was chosen as the vasopressor for this case. Maternal BP was supported during deep isoflurane anesthesia without fetal compromise, as evidenced by normal umbilical arterial and venous blood gases after 1 h of the EXIT procedure.

With the ability to diagnose fetal anomalies *in utero* by ultrasonography, a multidisciplinary approach can be instituted in formulating a plan for delivery and airway management antenatally. By providing up to 1 h of uteroplacental support, establishment of an airway can be performed in a controlled manner at birth. What historically has been considered a lethal anomaly may now be approached with the EXIT procedure, resulting in a favorable outcome for the infant.

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