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a normal redox state and the potential futility of overly exuberant attempts at hyperresuscitation.

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Postanesthetic Apnea in Full-term Infants after Pyloromyotomy

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POSTANESTHETIC apnea in the former premature infant less than 60 weeks postconceptional age is a well described phenomenon, with specific strategies recommended for its prevention, monitoring, and treat-

ment.¹⁻⁶ Apnea has been described in four full-term infants after anesthesia for a variety of surgical procedures.⁷⁻¹⁰ We report four cases of apnea after anesthesia for pyloromyotomy for full-term infants without perinatal problems.

Patient characteristics, preoperative laboratory values, and details of the anesthetic techniques are presented in table 1.

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Case 1

This infant appeared mildly dehydrated, and after 2 h of rehydration consisting of normal saline 20 ml/kg, he was taken to the operating

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room. Upon arrival in the postanesthesia care unit (PACU), rectal temperature was 37.2° C. In the first 15 min the patient had two episodes of apnea, each lasting 15–20 s. They were detected by direct observation by the PACU nurse.

Hemoglobin oxygen saturation, measured *via* pulse oximeter (Sp_{O_2}), decreased from 99% to 93% while the infant breathed 3 l/min nasal cannula oxygen, but his heart rate remained at 140–150 beats/min. Both apneic episodes were terminated using tactile stimulation. After 2.5 h observation in the PACU with no further apnea, the patient was monitored on the ward with an apnea–bradycardia monitor. He was discharged home 36 h after surgery without further apnea.

Case 2

In the operating room, shortly after extubation, this infant had two brief episodes of apnea (each 5–10 s), without change in heart rate, that resolved spontaneously. The infant's rectal temperature was 35.8° C at the time of admission to the PACU. Fifteen minutes later the patient had a single apneic episode lasting 15 s without a decrease in Sp_{O_2} while breathing 8 l/min oxygen by face mask. This episode was detected by direct observation by the PACU nurse. Heart rate remained unchanged at 125 beats/min, and the episode was terminated by tactile stimulation. After 2 h of observation in PACU, the patient was transferred to the pediatric intensive care unit for apnea–bradycardia monitoring. He was discharged home 48 h after surgery without further episodes of apnea or bradycardia.

Case 3

This patient presented to the emergency department appearing slightly dehydrated. After administration of lactated Ringer's solution 20 ml/kg and rehydration for 14 h, the patient was taken to the operating room. Forty-five minutes after uncomplicated induction of anesthesia, the Sp_{O_2} decreased to 77% briefly (less than 30 s). Because of lack of adequate breath sounds and poor chest excursion with manual ventilation, the trachea was reintubated. Because of the possibility of bronchospasm, the patient was given six breaths of metaproterenol with a metered-dose inhaler *via* the tracheal tube. Breath sounds were clear after the reintubation. He had no subsequent intraoperative problems, and the trachea was extubated in the operating room. A chest radiograph in the PACU was normal, and after 1 h the infant was discharged to the ward with apnea–bradycardia and pulse oximeter monitoring.

Seven hours after surgery the apnea–bradycardia alarm sounded. The patient had received no opioids and had been observed by the nurse to be awake and crying 8 min previously. The infant was noted to take two gasping respirations 10 s apart and then became apneic and pulseless. The patient required tracheal intubation, 8 min of external cardiac compressions, three doses intravenous (iv) epinephrine, and two doses iv sodium bicarbonate. Arterial blood gas values 15 min after resuscitation were pH 7.23, carbon dioxide tension 28 mmHg, oxygen tension 563 mmHg, and base deficit 15 mEq/l, and serum glucose was 129 mg/dl. The patient had a generalized seizure shortly after the arrest and received a loading dose of iv phenobarbital. The lungs were mechanically ventilated for 3 days because of depression in consciousness level with hypoventilation, presumably resulting from the hypoxic central nervous system insult and phenobarbital. The infant recovered full neurologic function by the time of discharge.

The bedside nurse had assessed the infant 1 h before the cardiorespiratory arrest and had noted clear breath sounds on bilateral auscultation and a respiratory rate of 44 breaths/min with no distress. At no time during or after cardiorespiratory arrest was bronchospasm noted. Seven days after surgery the patient had a 20-s apneic spell, with bradycardia and cyanosis, that was terminated by tactile stimulation. Phenobarbital had been the only medication for 96 h. Other diagnostic procedures during the hospital stay revealed two normal electroencephalograms, normal 12-lead electrocardiogram, and no cardiac arrhythmias noted over 7 days of monitoring. Cerebral computed tomographic scan 1 day after the resuscitation revealed only mild cerebral edema, and cerebral magnetic resonance imaging scan 10 days later was normal. Polysomnography 9 days after surgery revealed no apnea or bradycardia but an abnormal response to 17% inspired oxygen challenge, with Sp_{O_2} decreasing to 85% and onset of periodic breathing with respiratory pauses up to 6 s. The patient was discharged home 11 days after the operation with a home apnea–bradycardia monitor.

Case 4

Upon transfer to our hospital, the patient was moderately dehydrated. She had experienced three brief episodes of apnea, each lasting less than 15 s, with bradycardia to 100 beats/min. Cerebral computed tomographic scan, obtained because of a history of lethargy and a full anterior fontanel, was normal. After the patient received iv fluid therapy for 10 h, the infant's lethargy and dehydration had resolved, and she was taken to the operating room. The patient was given caffeine citrate 10 mg/kg iv because of the preoperative apnea.

One hour after surgery, two episodes of apnea with heart rate decreasing to 100 beats/min occurred. They were detected by direct observation by the PACU nurse. The episodes were 15 s in duration with Sp_{O_2} decreasing from 100% to 90%, and both episodes were terminated by tactile stimulation of the patient. The infant was transferred to the pediatric intensive care unit 2 h after surgery and was given an additional 10 mg/kg caffeine citrate iv at that time. She had no further episodes of apnea. The Pediatric Pulmonary Service was consulted and ascribed the preoperative apnea spells to the dehydration and metabolic alkalosis (see below). The postoperative apnea was believed clearly to have been associated with the anesthetic. Because of the self-limited nature of the apnea and the patient's young gestational age, a polysomnogram was not done. The patient was discharged home 4 days after the operation.

Discussion

There have been only four reported cases of postanesthetic apnea in a full-term infant.^{7–10} The ages of the patients ranged from 1 to 40 days, and surgical procedures consisted of cataract extraction, inguinal herniorrhaphy, bladder exstrophy repair, and cystoscopy with drainage of imperforate hymen. None of these infants had a history of apnea or intraoperative complications that might have predisposed them to apnea. The apneic episodes occurred 45 min to 6 h after anesthesia. On examination by polysomnogram one infant demonstrated multiple brief episodes of apnea with

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Table 1. Clinical Data and Anesthetic Techniques

	Patient			
	1	2	3	4
Age (days)	34	19	11	19
Postconceptional age (weeks)	42	43	42	43
Weight (kg)	3.9	3.6	4.3	3.8
Preoperative	138	137	153	149
Na ⁺ (mEq/l)				
K ⁺ (mEq/l)	5.8	5.9	4.7	5.3
Cl ⁻ (mEq/l)	105	101	110	104
CO ₂ (mEq/l)	23	19	21	30
Hemoglobin (g/dl)	11.6	16.9	16.4	14.1
Glucose (mg/dl)	NA	76	101	122
Anesthetic agents	A, T, sux, N ₂ O, hal, P	A, T, sux, N ₂ O, hal, bupiv, V	A, T, sux, hal, V	A, T, sux, iso, V
Fluids (ml)	D51/3 NS 135, 15 25% albumin	D51/3 NS 90	D2.51/2 NS 120	D2.51/2 NS 190
Estimated blood loss (ml)	5	2	10	5
Neuromuscular blockade reversal drugs (μg/kg)	Neostigmine 80, atropine 25	Neostigmine 80, glycopyrrolate 16	Neostigmine 70, glycopyrrolate 14	Neostigmine 70, glycopyrrolate 14
Train-of-four monitoring	Yes	Yes	Yes	Yes
Anesthetic time	1 h, 20 min	1 h, 10 min	1 h, 15 min	1 h, 50 min
Surgical time	55 min	50 min	45 min	1 h, 5 min
Time of apnea, postoperative	15 min (×2)	15 min	7 h	1 h

Na⁺ = serum sodium concentration; K⁺ = serum potassium concentration; Cl⁻ = serum chloride concentration; CO₂ = serum carbon dioxide concentration; A = atropine; T = thiopental; sux = succinylcholine; N₂O = nitrous oxide; hal = halothane; P = pancuronium; bupiv = 0.25% bupivacaine infiltration to wound; V = vecuronium; iso = isoflurane; D51/3 NS = 5% dextrose in one third normal saline; D2.51/2 NS = 2.5% dextrose in one half normal saline.

desaturation that resolved after treatment with theophylline. Three of the four infants were scheduled for outpatient surgery.

The possibility of apnea immediately after pyloromyotomy has been described^{11,12}; however, no cases have been reported in the anesthesia or surgical literature. Our patients all were full-term and experienced apnea 15 min to 7 h after anesthesia. We ruled out other factors commonly contributing to apneic spells in young infants: anemia (by hemoglobin measurement and minimal surgical blood loss); hypothermia (one infant did have a rectal temperature of 35.8° C, but this is within normal limits in full-term infants)¹³; hypoglycemia (by measurement of serum glucose or because of the rather large amounts of glucose administered); sepsis (by clinical course); or central nervous system abnormalities (by cerebral imaging or neurologic examination).¹⁴ All patients had documented reversal of neuromuscular blockade by train-of-four monitoring, and none had received opioids. All patients' tracheas were extubated when they were fully awake. Several of these infants did receive generous amounts of iv fluid and glucose, but no infant had signs

of respiratory distress from possible fluid overload in the postoperative period. Patient 3 did have an episode of inadequate ventilation during surgery, either due to bronchospasm or bronchial intubation, but this was not believed to be a contributing factor in his life-threatening apneic episode. Patient 4 did have several preoperative episodes of apnea and bradycardia, but she was the only patient with a hypochloremic metabolic alkalosis. (See below.)

There are several possible mechanisms for the apnea seen in these patients. First, the repeated vomiting associated with pyloric stenosis often leads to a hypochloremic metabolic alkalosis.¹⁵ Cerebrospinal fluid pH, which is one of the determinants of respiratory drive,¹⁶ may still be elevated after correction of serum electrolytes. However, only one of the four patients exhibited this electrolyte pattern, and she did have preoperative apneic spells. Second, subanesthetic concentrations of volatile agents depress hypoxic ventilatory drive in adult volunteers.¹⁷ This may have played a role in the early postoperative apnea seen in these patients, although it is unlikely to have contributed in patients 3 and 4. Third, it is possible that diagnosis of

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the duration of the apneic spells in patients 1, 2, and 4 was inaccurate.¹⁸ No hard copies of the respiratory channel on the apnea-bradycardia monitor were obtained. The duration of the apnea may have been overestimated, and thus the episodes may actually have been within what would be considered normal limits for this gestational age. A small percentage of normal full-term infants of 42–43 weeks' postconceptional age may have very infrequent respiratory pauses of 12–18 s.^{19–23} In addition, patients 1 and 2 did not suffer cyanosis or bradycardia. (Both were receiving supplemental oxygen.) However, the apneic spells in patients 1, 2, and 4 were terminated by tactile stimulation to prevent severe cyanosis and bradycardia. It is impossible to speculate about exactly how long the apnea in these cases would have lasted without intervention. Much more disturbing is the case of the infant who developed severe apnea, bradycardia, and cardiac arrest 7 h after the operation. A thorough search revealed no precipitating causes other than general anesthesia.

Further study is required to define better the effect of general anesthesia on the incidence and severity of apnea in this patient population. Until such information is available, no firm recommendations can be made about postoperative apnea monitoring in young full-term infants. However, we believe that permanent neurologic injury and possibly death were prevented in patient 3 by the apnea-bradycardia monitor. Despite the well known limitations of the use of apnea monitors with small infants,²⁴ we believe that consideration should be given to short-term apnea monitoring in young full-term infants who undergo general anesthesia.

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