Pulmonary Edema Secondary to Laryngospasm in Children

K. W. Terry Lee, M.D.,* and John J. Downes, M.D.;†

Although reported in patients with other forms of upper airway obstruction,1,4 pulmonary edema associated with intense laryngospasm during or after anesthesia appears to be a rare complication. No adult and only one adolescent case has been reported in which a 14-year-old, 74-kg boy with breast hyperplasia was described who developed laryngospasm and pulmonary edema following tracheal extubation after uncomplicated general anesthesia for a simple mastectomy.5 We observed two children ages 3 months and 10 years with postanesthetic laryngospasm who developed pulmonary edema without evidence of cardiac enlargement or cardiovascular disease.

REPORT OF TWO CASES

Patient 1. A 3-month-old infant, 5.0 kg in weight, was admitted for repair of bilateral inguinal hernias. The infant was born at 33 weeks gestation, with a weight of 1,600 g. Respiratory distress syndrome developed, which required tracheal intubation with continuous positive airway pressure (CPAP) for 3 days. The infant's recovery was uneventful, and the remainder of the history and physical examination were normal. Preinduction heart rate was 148 beats/min, respiratory rate 38/min, and rectal temperature 37.0° C. We induced anesthesia with 70% nitrous oxide and halothane and started an iv infusion. Following pancuronium (0.5 mg) iv, the trachea was intubated easily with a 3.0-mm I.D. polyvinylchloride tube, and anesthesia was maintained with 70% nitrous oxide without halothane. Heart rate remained at 160 to 180 beats/min and systemic arterial pressure at 110–130/60–70 mm Hg throughout surgery.

The infant received a total of 120 ml 5% dextrose in 0.25% saline iv during the 90-min period of anesthesia and operation. His normal maintenance iv fluid requirement per hour is 20 ml; his fluid deficit from an 8-h overnight fast was estimated to be 160 ml. Because of the unusually long fast and resultant fluid deficit, we replaced most of this deficit during anesthesia so that if the iv cannula malfunctioned postoperatively, the infant could tolerate the expected reduced oral intake in the first 4–8 h after anesthesia.

At the end of surgery, anesthesia was terminated and ventilation was controlled (FiO2 = 1.0). We injected atropine and neostigmine iv for reversal of the neuromuscular blockade. Within two min, the infant began coughing, flexed both upper and lower extremities, and appeared to have normal spontaneous breathing. Although he was not fully awake, we elected to extubate the trachea. Immediately after extubation, the infant developed severe laryngospasm, which could not be relieved by administration of positive airway pressure via bag and mask. Cyanosis and a slowing heart rate to 90 beats/min ensued. Administration of succinylcholine iv (10 mg) allowed resumption of controlled ventilation via bag and mask with 100% oxygen. Heart rate rose to 110 beats/min with the return of a pink color. To facilitate removal of upper airway and tracheal secretions, and to prevent gastric distension, we reintubated the trachea. Abundant pink secretions immediately flowed from the tube and subsequently were removed by tracheal aspiration.

With a FiO2 of 1.0, we controlled ventilation and performed periodic endotracheal toilet for removal of the tracheal secretions and edema fluid. The heart rate remained between 170 and 180 beats/min and systemic systolic arterial pressure at 120 mm Hg. With a FiO2 of 1.0 PaO2 was 201 mm Hg, PaCO2 34 mm Hg, pH 7.20, and BE −8.0 m Eq/L. To reduce the PaCO2, we increased minute ventilation. Because we had rapidly replaced his fluid deficit and to reduce the production of edema fluid, we gave furosemide (5 mg) iv. Because of persistence of pulmonary edema fluid and the wide alveolar–arterial oxygen tension difference (AaDO2), we judged that an artificial tracheal airway would be needed for an extended period. Thus, to facilitate mouth care and provide optimal stability of the tracheal airway, we replaced the orotracheal with a nasotracheal tube. Ten minutes after the iv injection of succinylcholine, apparently adequate spontaneous ventilation resumed. A chest roentgenogram taken at this time showed haziness in the right middle lobe, but no cardiomegaly or other abnormality, and the tracheal tube in an appropriate position. Following spontaneous ventilation with 100% oxygen but no continuous positive airway pressure (CPAP) for 30 min, PaO2 was 115 mm Hg, PaCO2 34 mm Hg, pH 7.36, and BE −6 m Eq/L. The volume of pulmonary edema fluid by this time was minimal, despite increasing AaDO2. Manually assisted ventilation with CPAP of 5 cmH2O was resumed; the subsequent AaDO2 improved. The infant appeared fully awake and responsive and remained so except for periods of normal sleep throughout his hospitalization.

One hour after the episode of laryngospasm, we transferred the infant to the Pediatric Intensive Care Unit, where he breathed spontaneously with a CPAP of 4 cmH2O at an FiO2 of 0.40. With this therapy, 3 h after the episode of laryngospasm, PaO2 was 126 mm Hg (FiO2 0.40), PaCO2 was 33 mm Hg, pH was 7.44, BE was −1.0 m Eq/L, and hematocrit was 34%. Five hours after anesthesia, his breath sounds were clear and tracheal secretion minimal; we extubated the trachea and placed the infant in a hood with humidified oxygen (FiO2 = 0.30) overnight. A repeat chest roentgenogram 7 h after anesthesia showed clearing of the right middle lobe haziness, and the PaO2 was 104 mm Hg (FiO2 = 0.30). On the following morning, while the infant breathed room air, PaO2 was 79 mm Hg. We discharged him from the PICU.
on the first postoperative day, and he left the hospital on the second postoperative day without apparent sequelae.

Patient 2. A 10-year-old boy weighing 29 kg was admitted for elective repair of a right undescended testis. The remainder of his medical history, physical examination, as well as urinalysis and complete blood count were normal. Heart rate was 75 beats/min, systemic arterial pressure was 100/80 mmHg, respiratory rate was 20/min, and rectal temperature was 37.2°C. Pentobarbital (100 mg), morphine (3.0 mg), and atropine (0.5 mg) were given im 1 h prior to anesthetic induction. The child had been fasting for 18 h because of an unpredicted delay in starting surgery, resulting in a 1,260-ml fluid deficit.

Prior to induction of anesthesia, we applied a precordial stethoscope, Doppler blood pressure apparatus, and ECG leads; we inserted a rectal temperature probe after induction of anesthesia. We induced and maintained anesthesia with 70% nitrous oxide and halothane, using manually assisted ventilation via a mask. Following induction, 5% dextrose in 0.25% saline was administered iv; a total of 500 ml was infused during surgery. No problems occurred during the 40-min surgery.

Upon awakening from general anesthesia, the patient coughed and suddenly developed intense laryngospasm. This persisted, despite positive airway pressure with oxygen (FiO₂ = 1.0), resulting in severe cyanosis and tachycardia (heart rate 155 beats/min). Following administration of succinylcholine (60 mg) iv we regained control of ventilation (FiO₂ 1.0) with subsequent return of normal heart rate and color. The trachea then was intubated, and immediately abundant pink foamy secretions flowed from the tube. These secretions had a pH of 8.00. We controlled ventilation at an FiO₂ of 1.00 with intermittent tracheal aspiration. Shortly after intubation of the trachea, PaO₂ was 306 mmHg, PacO₂ was 81 mmHg, pH was 7.02, BE was −9 meq/L, hematocrit was 30%, and serum osmolality was 293 osm/L. We immediately increased the minute ventilation to correct the hypercapnia. A chest roentgenogram was read as normal with no signs of cardiomegaly. Within 15 min, the child’s appearance improved dramatically with a decrease in heart rate to normal levels, vigorous spontaneous breathing, and improved responsiveness.FUROSEMIDE was not given because the volume of edema fluid was diminishing, and the child still had a 700-ml fluid deficit with a normal serum osmolality.

While breathing spontaneously (FiO₂ 1.0) PacO₂ was 357 mmHg, PaCO₂ was 56 mmHg, pH was 7.18, and BE was −8 meq/L. Sodium bicarbonate 25 mEq was given iv to correct one-half of the base deficit in the extracellular fluid space. We then transferred the patient to the recovery room.

Because of continuous clinical improvement, including full awakening and responsiveness, FiO₂ was reduced over a 3-h period to 0.21, at which time PacO₂ was 54 mmHg, PaCO₂ was 44 mmHg, pH was 7.33, and BE was −2 meq/L. We extubated the trachea and placed him in a tent with humidified oxygen (FiO₂ = 0.30). The following morning, while he was breathing room air, PaO₂ was 98 mmHg, PaCO₂ was 34 mmHg, pH was 7.41, and BE was −2 meq/L. He was discharged from the hospital on the second postoperative day and made an uneventful recovery.

**DISCUSSION**

Mechanisms favoring the transfer of fluid from the pulmonary capillary into the alveolus during spontaneous breathing against a closed glottis (Mueller maneuver) include the following:

1. Transpulmonary intrathoracic pressure gradients created by inspiratory efforts result in subatmospheric pressures up to and exceeding 60 cmH₂O. This provides a large pressure difference across the alveolar-capillary membrane with a low interstitial and alveolar pressure, compared with the intravascular capillary pressure. According to the Starling equation, such a condition favors a flow of fluid out of the capillary into the interstitium and eventually into the alveolus.⁶⁷

2. Hypoxia can cause pulmonary postcapillary and venous constriction (as well as arteriolar and precapillary constriction),⁸⁹ resulting in increased postcapillary vascular resistance in some areas of the lung. This causes, in turn, a rise in intracapillary pressure with consequent predisposition to transudation of fluid out of the capillary and into the alveolus.⁸⁻¹⁰

3. A large subatmospheric pressure gradient results in significant changes in cardiovascular function¹¹⁻¹²: left ventricular afterload increases; left ventricular ejection fraction decreases; left ventricular end systolic and end-diastolic volumes decrease; velocity of contraction decreases; and right heart increases. These changes favor a rise in left atrial and pulmonary blood volume, resulting in eventual increases in pulmonary capillary pressure that dispose to the transudation of fluid from the capillary into the alveolus.

We speculate that both of our patients developed pulmonary edema, which was induced by laryngospasm, hypoxia, and sustained ventilatory efforts against a closed glottis, resulting in large subatmospheric transpulmonary pressure gradients. The absence of alveolar ventilation during laryngospasm and the subsequent serious ventilation–perfusion disturbance associated with pulmonary edema resulted in persistent and severe increases in AaDO₂, hypercarbia, and acidosis.

Our experience suggests that to diminish the likelihood of postextubation laryngospasm, the infant or child, before tracheal extubation, should open his eyes and mouth spontaneously, move all extremities vigorously, and resume a normal breathing pattern after a cough. With patients inhaling anesthesia via a mask, we recommend removal of upper airway secretions while the patient remains fully anesthetized to avoid initiating laryngospasm as the airway reflexes return during awakening.

Laryngospasm that does not respond within 30 s to consistent positive airway pressure and relief of soft tissue obstruction probably should be treated immediately by neuromuscular blockade with iv succinylcholine (2 mg/kg), controlled ventilation with high inspired concentrations of oxygen and reintubation of the trachea if necessary to maintain an adequate airway. Pulmonary edema, if it occurs following laryngospasm, can be assumed to be associated with hypoxia, hypercarbia, and acidosis that will respond readily to reintubation of
the trachea, controlled ventilation with high inspired concentrations of oxygen, CPAP, and the iv administration of furosemide.

REFERENCES

Evaluation of Pulse Oximetry

MARK YELDERMAN, M.D.,* AND WILLIAM NEW, JR., M.D., PH.D.†

Continuous assessment of arterial oxygenation is important in clinical management of critically ill or anesthetized patients. Analysis of arterial blood gases is reliable but is invasive and only provides intermittent information. Transcutaneous oxygen tension measurement provides continuous information but requires special site preparation, airtight probe mantling, and a potentially harmful local heat source to induce “arterialization.” Even then transcutaneous oxygen monitoring fails to perfectly reflect true arterial oxygenation.1 Analysis of arterial blood gases and transcutaneous oxygen measurements both provide oxygen tension (PO2) data from which the oxygen content and percentage of hemoglobin saturated with oxygen can be estimated.

Arterial oxygen saturation of hemoglobin can be determined directly and continuously in vivo by using spectrophotometric techniques.2–5 The wavelength dependence of reduced versus oxyhemoglobin is evident from the prominent color differences in spectral light absorbance of “red” oxyhemoglobin and “blue” reduced hemoglobin. The light absorbances differences between reduced and oxyhemoglobin are described quantitatively by the molecular extinction coefficients in Beer’s Law. Other methods measure saturation using relative light absorption of two or more wavelengths directed through the ear vasculature. “Arterialization” is achieved by combinations of heat and chemical treatment to dilate the vascular bed in the measurement area. Such devices offer limited clinical utility because they are inconvenient and because it is technically difficult to consistently differentiate light absorbance of the desired arterial blood from the absorbance of tissue and venous blood.6 This deficiency can be circumvented by measuring light absorbance changes time coherent with arterial pulsation.7

Pulse oximetry functions by positioning any pulsating arterial vascular bed between a two-wavelength light source and a detector. The pulsating vascular bed, by expanding and relaxing, creates a change in the light path length that modifies the amount of light detected. The familiar plethysmograph waveform results. The amplitude of the varying detected light depends upon the size of the arterial pulse change, the wavelength of