54-year-old man undergoing irrigation of a herniorrhaphy wound with 3% hydrogen oxygen. Apparently irrigation under pressure of hydrogen peroxide solution into a semiclosed wound composed of bleeding friable tissue with small exit would lead to absorption of a significant amount of hydrogen peroxide and the nascent oxygen bubbles so produced into the venous system, which resulted in the near-fatal outcome. Fuson *et al.* reported that 1 ml of 3% hydrogen peroxide would release 10 ml of oxygen upon decomposition following contact with catalase in blood.[8]

The near-fatal outcome of our patient probably stems from the combination of late discovery of the gas embolism in a patient under general anesthesia and an initially unsuccessful cardiopulmonary resuscitation with a period of poor perfusion as reflected by the metabolic acidosis shown on arterial blood gas study. Nevertheless, our cases reaffirms the danger of using undiluted 3% peroxide solution for irrigation in a closed wound where systemic absorption of hydrogen peroxide and/or oxygen bubble may lead to serious sequalae of gas embolism and it serves a warning to discourage the continuation of such application.

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Naloxone Reversal of Postoperative Apnea in a Premature Infant

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In the perioperative period, preterm infants have a high incidence of complications, one of which is apnea.[1,2] Apnea may be caused by ventilatory muscle fatigue,[3] postoperative hypoxemia with abnormal ventilatory response to hypoxia,[4] depressant effects of inhaled anesthetics on the chemoreceptor response to hypoxemia,[5] and abnormal breathing control mechanisms in infants susceptible to sudden infant death syndrome (SIDS).

Infants with the apnea syndrome who have not had previous surgery have high endorphin levels in the cerebrospinal fluid.[7] Anesthetics also increase the release of endorphin.[8] Thus, elevation of cerebral endorphins may play a role in perioperative apnea by preterm infants. On the basis of this hypothesis, we administered the opiate antagonist naloxone to an infant with postoperative apnea resulting in the restoration of normal respiration.

Report of a Case

A 4-week-old, 2.4-kg male infant, delivered by cesarean section at 35 weeks of gestational age, presented with vomiting and failure to thrive, caused by pyloric stenosis. The infant had been discharged from the nursery a week after delivery, and there was no preanesthetic history of apneic episodes or other respiratory problems. Blood electrolyte and glucose levels were normal, and Hb was 12.8 g/dL. No preanesthetic medication was given. Following breathing of 100% oxygen via mask, an awake tracheal intubation was performed. Anesthesia consisted of nitrous oxide, oxygen (40%), and halothane 1–1.5%. Ventilation was assisted with the use of the Jackson-Rees modification of the Mapleson D system. During the operation, which lasted 40 min, 60 ml of iv 4.3% glucose in 0.18% normal saline was administered. Heart rate ranged from 120 to 130 bpm. Arterial blood pressure (Doppler method) remained between 80/50 and 70/40 mmHg. Rectal temperature at the end of surgery was 36.1° C. Halothane was stopped 5 min before the termination of surgery,

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and nitrous oxide was continued until the operation was complete. Extubation of the trachea was performed during inhalation of 100% oxygen when the infant was fully awake, with stable vital signs, and reacting to the endotracheal tube. Oxygen was continued by a mask and his respiratory patterns appeared normal.

Apnea and bradycardia (80–90 bpm) occurred 2–3 min after extubation. Breathing resumed after manual stimulation and oxygen administration by mask and bag. However, episodes of apnea recurred every 2–3 min during the following 30 min. Naloxone 0.02 mg (8 μg/kg) was given iv. This dose is only slightly more than the usual recommended dose for infants, *i.e.*, (5 μg/kg). As soon as the injection was complete, there was a dramatic resumption of spontaneous respiration, an increase in heart rate of 120–130 bpm, and full awakening, accompanied by crying and movement. The child was retained in the operating room for a further 20 min and was then transferred to the intensive care unit for further observation. There was no subsequent recurrence of apnea.

Discussion

Endorphins and opiate analgesics diminish the ventilatory response to hypercarbia and hypoxia[9] and depress heart rate when they are given into the cisterna magna in dogs.[10] These, and other actions of endorphins and opiates, can be antagonized by naloxone.[11] Infants with apneic episodes or near-miss SIDS reportedly have high levels of endorphins in the cerebrospinal fluid.[7] Some effects of inhaled anesthetics such as halothane, enflurane, and nitrous oxide also may be antagonized by naloxone and may therefore result from the release of endogenous opioid peptides.[8] Naloxone reduces the duration of experimentally induced primary apnea in newborn rabbits,[12] implying the involvement of opioid peptides in this condition.

These data suggest the possibility that endogenous opioids may have been implicated in the occurrence of recurrent apnea in this infant who did not receive any opiate analgesic either before or during surgery. We do not think it is likely that the apneic episodes were caused either by hyperventilation during anesthesia or by residual halothane because the anesthesia was completed and the trachea extubated at least 40 min before the naloxone was given.

The combined effects of anesthetics and prematurity, each of which results in raised endorphin activity, may explain the high incidence of apneic episodes in preterm infants in the perioperative period. This experience suggests that naloxone may be useful in the treatment of postanesthetic apnea in infants, even when exogenous narcotics have not been administered. Further studies are necessary to confirm its efficacy and safety in this clinical situation.

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