REFERENCES

1. Brajtbord D, Paulsen AW, Ramsay MAE, Swygert TH, Valek TR: Controversies associated with autotranfusion during hepatic transplantation (abstract). ANESTHESIOLOGY 69:A171, 1988
2. Black HA, Dearing JP: Exchange transfusion prior to cardiopulmonary bypass in sickle cell anemia. J Extra-Corp Technol 12: 82–85, 1980
3. Romanoff ME, Woodward DG, Bullard WG: Autologous blood transfusion in patients with sickle cell trait. ANESTHESIOLOGY 68:820–821, 1988

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A Humidification Device for Nasal Oxygen

To the Editor:—Inhalation of dry oxygen causes uncomfortable symptoms such as nasal dryness, stuffiness, and itching, despite flows as low as 3 l/min. We have devised a simple, inexpensive means of humidification of oxygen for use with nasal cannulae. As shown in figure 1, the Y-piece of the breathing circuit is attached to an Airlife brand (American Pharmaseal Company) or similar humidifier bottle *via* a double male 22-mm corrugated tubing adaptor. Humidification is achieved by bubbling oxygen through water, the humidity being low enough to avoid condensation in the long narrow tubing that is connected to the nasal cannulae.

We have tested the device on 40 unselected women undergoing cesarean section under regional analgesia. Unannounced change from dry to humidified oxygen was always followed by statements that breathing was suddenly easier, while change from humidified to dry oxygen led to complaints of discomfort. Flow rates of up to 5 l/min were well tolerated. Pulse oximetry revealed no differences in oxygen saturation between inhalation of dry *versus* humidified oxygen.

We recommend use of this simple device for any situation requiring inhalation of nasal oxygen by conscious patients.

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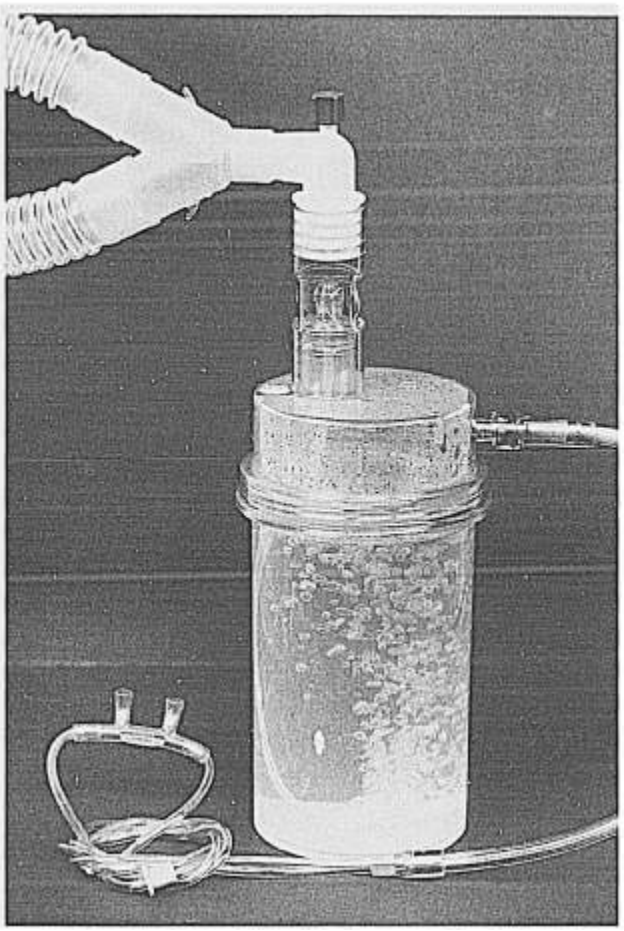

FIG. 1. Humidification device for nasal oxygen (from top to bottom): Y-piece of anesthesia breathing circuit; double male 22-mm adaptor; humidification bottle; and nasal cannula.

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Postoperative Apnea in a Full-term Infant

To the Editor:—We read with interest the report of postanesthetic apnea in a healthy full-term infant.[1] We recently cared for a term infant who experienced a similar single episode of apnea, accompanied by bradycardia, 6 h after a 2-h general anesthetic. We write to support

the concept that some full-term infants may well be at risk for such occurrences and to reinforce the suggestion that these observations should have some impact on planning day-surgery care for such babies.

Our patient was a 2950 gm female, delivered vaginally at term with Apgar scores of 8 at 1 min and 9 at 5 min. She was noted to have bladder exstrophy and was subsequently transferred to our Children's Hospital where she underwent primary closure at 30 h of age. Her initial cardiopulmonary examination was normal. No murmur was heard. She had voided spontaneously several times before surgery. Initial vital signs were as follows: T = 36.8° C (r); P = 120; BP = 80/60; and R = 28 without nasal flaring or retractions. Preoperative laboratory values: HGB = 17.9 GMS%; HCT = 54%; and WBC = 16,500.

Following administration of atropine 0.15 mgm im, she was taken to a warmed operating room where anesthesia was induced *via* mask with nitrous oxide-oxygen-halothane (1.5–2.0%). Pancuronium 0.3 mgm allowed easy intubation with an uncuffed 3.0-mm tracheal tube. The operative procedure lasted 105 min, requiring 2 h of anesthetic time. Anesthesia was maintained with nitrous oxide (3 l), oxygen (2–4 l), and halothane. Intravenous fluids consisted of 50 ml of 5% dextrose in half-normal saline. Monitoring included pulse oximetry (99–100%) and capnometry (30–34 mmHg). Vital signs remained stable near preoperative values throughout the case. Perioperative antibiotics were administered, consisting of ampicillin 75 mgm (25 mgm/kgm) and gentamycin 7.5 mgm (2.5 mgm/kgm). Blood loss was recorded as 15 ml.

At completion of the operation, neostigmine 0.25 mgm and atropine 0.1 mgm were administered intravenously. Train-of-four response to stimulation increased from 75% before these agents to 100% after their administration. The baby was then transported to the Intensive Care Unit with the tracheal tube in place, breathing spontaneously. She was noted to have bilateral breath sounds without retractions, and heart rate and respiratory rate were monitored. Her vital signs on arrival in the ICU were normal, except for a temperature of 34.7° C (r). She was placed under a radiant warmer with prompt recovery of normothermia.

One hour following the end of the procedure, with an end-tidal carbon dioxide of 34 mmHg, BP = 77/50, HR = 130, and T = 36.6° C, the trachea was extubated without difficulty. For the subsequent 4½ h she was repeatedly noted to be awake with a good suck and strong cry. She was breathing easily without retractions during this entire interval. No opiate analgesics were required postoperatively and none had been administered preoperatively or intraoperatively.

Four hours and 23 min after extubation, she became suddenly apneic. Vital signs just minutes prior to the episode were as follows: HR = 140; RR = 36; BP = 68/42; and T = 36.6° C. Physical stimulation restored spontaneous breathing but apnea recurred, accompanied by cyanosis and lethargy. Her lungs were ventilated *via* mask and bag with 100% oxygen and, because of a heart rate in the mid thirties, external cardiac massage was briefly instituted. Seven minutes later, she was breathing spontaneously and her heart rate was 60. Two minutes later, HR = 170 and RR = 28. Marked peripheral cyanosis persisted for 20 min and then gradually resolved. Pulse oximetry on the left hand was 97–99%, despite the cyanosis. Laboratory data obtained during this episode were as follows: Arterial blood gases; *p*h = 7.15, p_{CO_2} = 34, p_{O_2} = 251, and HCO_3 = 11.9. Serum chemistries: Na = 141, K = 5.2, Cl = 115, CO2 = 12.9, ionized Ca^{++} = 4.52 (normal), osmolality = 296, creatinine = 0.7. Her hematocrit was 45%. A portable chest radiograph was normal, as were head ultrasound and lumbar puncture.

The infant's rapid recovery from the apneic episode forestalled direct intervention to treat the metabolic acidosis that had been demonstrated by the arterial blood gas data. She became fully active without deficit, and after extensive discussion between all consultative services (neonatalogy, anesthesia, critical care, and surgery), it was elected to forego treatment with intravenous buffering agents. Her normal clinical state and negative workup led to the decision to avoid subjecting the infant to further invasive diagnostic tests, and, thus, no further arterial blood gas determinatives were made.

The baby remained in the ICU environment for the next 24 h and was then transferred to the newborn floor in an isolette with apnea monitor. She remained free of any further problems, was started on formula by mouth and environmentally weaned to bassinette and crib without any physiologic instability. She was discharged with apnea monitoring, her parents having been instructed in cardiopulmonary resuscitation prior to taking her home. She is now 5 months of age and thriving. No episodes of apnea or bradycardia have been noted.

Although genito-urinary tract anomalies are associated with cardiac defects, none was in evidence in this case. Furthermore, we are not aware of any association of bladder exstrophy with specific cardiopulmonary disease, which would predispose to episodes of apnea/bradycardia. It is apparent, however, that sudden infant death syndrome occurs with a higher frequency in babies with congenital defects.[2] There were no elements in our patient's anesthetic management that placed her at specific risk for this occurrence. It is clear that such major genito-urinary anomalies are not repaired in infancy on a "day-surgery" basis, but other congenital G-U anomalies (*e.g.*, hypospadias) are frequently corrected in this setting. It is our practice to limit outpatient general anesthesia for term infants to those of 12 months postconceptual age and older. We believe that the observations of Tetzlaff *et al.* and ourselves reinforce this recommendation. We would urge that such limitations, even for minor procedures, be widely adopted for term infants in this age group until further studies of term newborns with and without congenital anomalies more fully define these risk factors.

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REFERENCES

1. Tetzlaff JE, Annand DW, Pudimat MA, Nicodemus HF: Postoperative apnea in a full-term infant. ANESTHESIOLOGY 69: 426–428, 1988
2. Kelly DH, Shannon DC: Sudden infant death syndrome and near sudden infant death syndrome: A review of the literature, 1964 to 1982. Pediatr Clin North Am 19: 1241–1261, 1982

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