

Intraoperative Hypoxia from an Erroneously Filled Liquid Oxygen Reservoir

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Hypoxia from failure of the anesthesia machine or the oxygen supply system is a recognized hazard of anesthesia. Reported causes of oxygen failure during anesthesia include: 1) depletion of the oxygen cylinder; 2) substitution of a non-oxygen cylinder at the oxygen yoke resulting from absence or failure of the pin index; 3) an erroneously filled oxygen cylinder; 4) insufficient opening of the oxygen cylinder to permit a free flow of gas as pressure decreases; 5) failure of gas pressure in a piped oxygen system; 6) failure to open the valve of a piped oxygen system; 7) faulty locking of the piped oxygen system to the anesthesia machine; 8) inadvertent switching of the Schrader adapters on piped lines; 9) crossing of piped lines during construction; 10) failure of a reducing valve or gas manifold; 11) inadvertent disturbance of the setting of the oxygen flowmeter; 12) employment of the fine oxygen flowmeter instead of the coarse flowmeter; 13) fractured or sticking rotameters; 14) transposition of rotameter tubes.¹⁻³ We report here two cases of hypoxia from an unusual cause of oxygen failure: the erroneous filling of a liquid oxygen reservoir with nitrogen.

REPORT OF TWO CASES

Case 1. An 18-year-old youth was scheduled for open reduction and internal fixation of a fractured left mandible. The medical history, physical examination, and laboratory findings were otherwise normal. Following preanesthetic medication with pentobarbital and atropine, anesthesia was induced with Innovar, meperidine, and thiopental while the patient breathed oxygen (5 l/min) via a mask. Succinylcholine facilitated the passage of a nasotracheal tube under direct vision of the larynx. Anesthesia was maintained with 70 per cent nitrous oxide and oxygen, using a total gas flow of 5 l/min with controlled respiration. The blood pressure, pulse, electrocardiogram, esophageal heart

and pulmonary sounds, and esophageal temperature were monitored. Shortly after the nasotracheal tube was secured, it was noted that the superficial vessels of the neck were engorged and that the upper chest and extremities were cyanotic. The blood pressure ranged from 180/60 to 160/40 torr and the pulse ranged from 130 to 110 beats/min (preoperative values were 140/70 torr and 95 beats/min). Examination of the patient, the anesthesia machine and circuit, and the position of the nasotracheal tube provided no explanation for the changes in the patient's condition. Although breath sounds were present bilaterally, the nasotracheal tube was withdrawn 2 inches to eliminate a chance endobronchial intubation; then arterial blood was drawn for gas analysis. Because this blood sample appeared dark (subsequent analysis revealed P_{aO_2} , 21 torr, P_{aCO_2} , 43 torr, pH 7.2), the nitrous oxide was discontinued and anesthesia was maintained with 0.5 per cent halothane, pancuronium, and oxygen. After discontinuation of the nitrous oxide, the venous engorgement and cyanosis decreased. Since a repeat arterial blood sample again appeared dark (subsequent analysis revealed P_{aO_2} , 31 torr, P_{aCO_2} , 24 torr, pH 7.4), the oxygen supply from the cylinders on the anesthesia machine was substituted for the wall oxygen supply. Following this substitution, the venous engorgement and cyanosis promptly disappeared. Subsequent arterial blood values were P_{aO_2} , 324 torr, P_{aCO_2} , 21 torr, pH 7.47. At the conclusion of operation, the patient regained consciousness quickly, and postoperative recovery was uneventful.

Case 2. A 44-year-old woman with uterine leiomyomas was scheduled for an abdominal hysterectomy. The medical history, physical examination, and laboratory findings were normal except for the presence of an incomplete right bundle-branch block on the electrocardiogram. Preanesthetic medication and induction of anesthesia were similar to those described in the first case except that an orotracheal tube was used. Anesthesia was maintained with meperidine, pancuronium, nitrous oxide, 66 per cent, and oxygen, using a total gas flow of 6 l/min with controlled respiration. After induction of anesthesia, the blood pressure ranged from 165/105 to 110/70 torr and the pulse ranged from 160 to 130 beats/min (preinduction values were 140/90 torr and 124 beats/min). When the skin incision was made, the patient's blood was noted to be dark, although cutaneous cyanosis was not obvious. Examination of the patient, the anesthesia machine and circuit, and the endotracheal tube position revealed no cause for the dark blood. After the oxygen concentration was increased to 50 per cent, arterial blood gas values were P_{aO_2} , 26 torr, P_{aCO_2} , 27 torr, pH 7.5.

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At this time, it was learned that a similar problem with oxygenation was occurring in the only other operating room in use (Case 1); therefore, the cylinder oxygen supply on the anesthesia machine was substituted for the wall oxygen supply. This change in the oxygen source improved the color of the patient's blood, and repeat arterial blood-gas values were P_{aO_2} 285 torr, P_{aCO_2} 16 torr, pH 7.5. The subsequent course of anesthesia and post-operative recovery were uneventful.

DISCUSSION

Failure to deliver adequate concentrations of oxygen to the anesthetized patient has resulted in significant morbidity and mortality.^{1,2,3} In the cases reported here, the cause of the hypoxia was identified early enough to prevent disastrous outcomes. Analysis of the gas delivered at the wall oxygen outlets in the operating rooms revealed an oxygen concentration of 24 per cent. After dilution with 70 per cent nitrous oxide (as in Case 1), this concentration of oxygen would have resulted in the administration of 7.2 per cent oxygen to the patient. Coincident analysis of the gas delivered at the wall oxygen outlets in the recovery room, emergency room, intensive care unit, and nursery revealed similar low concentrations of oxygen. Fortunately, few patients were receiving oxygen in these other areas of the hospital, and these patients did not experience clinically significant hypoxic episodes.

The source of the wall oxygen supply failure was found to be the bulk oxygen supply for the entire hospital. In our hospital, the bulk oxygen supply consists of two liquid oxygen reservoirs that alternately supply the main oxygen pipeline to the hospital. This supply is backed up by a reserve bank of oxygen cylinders that is used only in case of an emergency. Analysis of the contents of the liquid oxygen reservoirs revealed that one contained pure nitrogen and the other pure oxygen. Theoretically, delivery of pure nitrogen could have occurred; however, because of the intermittent opening and closing of pressure-regulated valves, a differential flow of gas from both reservoirs occurred, resulting in the low oxygen mixture delivered at the wall outlets.

The erroneously filled reservoir was labeled as liquid oxygen and was fitted with

valve outlet connections that met the Diameter-Index Safety System (DISS) requirements for oxygen. Filling of this type of tank is performed by using DISS cylinder valve outlet and inlet connections in order to prevent errors in filling the tank. In attempting to fill the tank in question, an employee of the liquid oxygen supply company was thwarted initially by the DISS valve connections when he attempted to fill the tank with nitrogen; however, after attaching an adapter to bypass the DISS connections, he was successful in filling the reservoir with liquid nitrogen.

The prevention of disasters from failure of oxygen delivery systems depends largely upon the alertness of the anesthesiologist. Thorough checking of anesthetic equipment immediately prior to its use, constant observation of the physiologic responses of the patient, and adequate knowledge of the mechanical construction and inherent shortcomings of anesthetic equipment can help prevent misadventures. Use of mechanical aids, oxygen alarms, and oxygen monitors are also important. Standards for color coding,⁴ pin-indexing,⁵ and labeling of gas cylinders⁶; for valve outlet and inlet connections⁷; and for bulk oxygen⁷ and central supply systems have been introduced.⁸ Low-oxygen warning aids and fail-safe devices that give audible and visual signals when the pressure in the oxygen line decreases have also been introduced.^{1,2,3} Since these pressure-activated alarm systems detect only decreases in oxygen pressure, they are of no use when decreases in oxygen concentration occur. However, it is technically and economically feasible to monitor the oxygen concentration in an anesthetic circuit continuously,² and use of such monitors would have indicated the failure of the oxygen delivery system that occurred in these cases.

As a result of the oxygen-failure incident reported here, the industrial gas manufacturer that supplied the erroneously filled liquid oxygen reservoir is now checking the concentration of oxygen in each reservoir before delivery and has taken steps to standardize liquid container fittings, labels, and color codes in use at its plant. There is no nationwide standardization of liquid reser-

voir fittings or labels to which industrial gas producers must adhere. Such standards, combined with continuous monitoring of the oxygen concentration in the anesthetic circuit and in the central oxygen supply line, would contribute to the safety of oxygen administration.

REFERENCES

1. Ward CS: The prevention of accidents associated with anesthetic apparatus. *Br J Anaesth* 40:692-701, 1968
2. Mazze RI: Therapeutic misadventures with oxygen delivery systems: The need for continuous in-line oxygen monitors. *Anesth Analg* (Cleve) 51:787-792, 1972
3. Epstein RM, Rackow H, Lee ASJ, et al: Prevention of accidental breathing of anoxic

- gas mixtures during anesthesia. *ANESTHESIOLOGY* 23:1-4, 1962
4. Standard Color-marking of Compressed Gas Cylinders Intended for Medical Use in the United States. Pamphlet C-9. Compressed Gas Association, Inc., New York, New York
 5. ANSI Standard B57.1-1965, Compressed Gas Cylinder Valve Outlet and Inlet Connections. Compressed Gas Association, Inc., New York, New York
 6. ANSI Standard Z48.1-1954, Method of Marking Portable Compressed Gas Containers. Compressed Gas Association, Inc., New York, New York
 7. NFPA 50, Bulk Oxygen Systems 1973. National Fire Protection Association, Boston, Massachusetts
 8. NFPA 56F, Nonflammable Medical Gas Systems 1973. National Fire Protection Association, Boston, Massachusetts

Inability to Reverse Pancuronium Blockade in a Patient with Renal Failure and Hepatic Disease

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Gallamine is used with caution or not at all in the management of patients with renal failure, since renal excretion is the major pathway for its elimination.¹ In usual clinical doses, *d*-tubocurarine has not been associated with similar prolonged elimination, presumably because of hepatic mechanisms for its elimination. For similar reasons, the new nondepolarizing neuromuscular blocking agent, pancuronium bromide, has been suggested to be particularly appropriate for use in the anephric patient. This report describes prolonged muscular weakness following pancuronium in an anephric patient who, in addition, had hepatic dysfunction secondary to acute biliary obstruction.

REPORT OF A CASE

A 45-year-old Negro woman who had renal failure and peptic ulcer disease was to undergo

drainage of a large pancreatic pseudocyst under general anesthesia. Three weeks earlier a rejected renal transplant had been removed, with fluorene-nitrous oxide anesthesia. Liver function tests had been normal at that time. Muscle relaxation had been achieved with *d*-tubocurarine, 21 mg, which was easily reversed with neostigmine, 2.5 mg, 45 minutes after the *d*-tubocurarine had been given. In the postoperative period, the patient had had numerous episodes of hypotension which were promptly treated with the intravenous administration of fluids and albumin. Chronic hemodialysis was also re-established.

At the end of the first postoperative week, the patient developed an abdominal mass and became jaundiced. Liver enzyme values were slightly increased, and the diagnosis of pancreatic pseudocyst with acute biliary obstruction was made. Her medications included prednisolone, nystatin, folic acid, and Aquamephyton. Physical examination revealed that the patient was debilitated, lethargic, and cushingoid, with scleral icterus. The blood pressure was 75/50 mm Hg, heart rate 100/min, temperature 38.4 C (rectal), and central venous pressure 2 cm H₂O. Weight was 60 kg. There was marked pitting pretibial edema. The chest was clear. Examination of her heart disclosed no abnormality except sinus tachycardia. A Scribner hemodialysis shunt was present in the left arm. A roentgenogram of the chest was clear and an electrocardiogram was normal. Abnormal laboratory values were: Na 134 mEq/l, K 2.9 mEq/l, Cl 86 mEq/l, total protein 5.6 g/100 ml, albumin 2.6 g/100 ml, bilirubin 8.9 mg/100 ml, BUN 79 mg/

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