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Pulmonary Edema Following Air Embolism

JAMES A. STILL, JR., M.D.,* DAVID S. LEDERMAN, M.D.,* WADE H. RENN, M.D.†

The following case report suggests that acute pulmonary edema can occur as a consequence of air embolization.

REPORT OF A CASE

A 19-year-old youth, in good health except for numbness over a C2-to-T4 distribution and decreased deep tendon reflexes in the right arm, was admitted for exploration of the posterior fossa. Results of all diagnostic studies, which included an electrocardiogram, complete blood count, urinalysis, serum electrolytes and chest roentgenogram, were normal. A myelogram two months preoperatively had showed an Arnold-Chiari malformation and widening of the spinal cord from C2 to T4, consistent with hydromyelia.

Preoperatively, the patient was given pentobarbital, 100 mg, hydroxyzine, 100 mg, and atropine, 0.6 mg, im. An hour later anesthesia was induced with intravenous administration of thiopental, followed by succinylcholine and endotracheal intubation. Anesthesia was maintained with oxygen (2 l/min), nitrous oxide (3 l/min), halothane (0.5 to 1.5 per cent) and *d*-tubocurarine. A 16-gauge catheter was passed with difficulty via the basilic vein, and a subsequent roentgenogram showed that the tip of the catheter was in the superior vena cava. A second catheter was placed in the radial artery, and heart sounds were monitored continuously through an esophageal stethoscope. Ventilation was controlled with a volume-limited ventilator attached to the circle absorption system of an anesthesia machine.

The operation proceeded without incident, with the patient in the sitting position, for four and a half hours, while the surgeon exposed and decompressed the malformation of the cerebellum and cervical spinal cord. Suddenly, during closure, a harsh, high-pitched systolic murmur and a decrease in systolic blood pressure from 130 torr to 95 torr were noted. Approximately 30 ml of air were aspirated through the venous catheter, and the surgeon sought unsuccessfully for an open vein. Nitrous oxide and halothane were discontinued and the fluid infusion rate was increased. Despite these maneuvers, the systolic blood pressure remained 95 torr, and the murmur persisted for 30 minutes. Then, over a 20-minute period, the blood pressure increased to 130 torr and the murmur disappeared. Central venous pressure at the end of the operation was less than 3 cm H_2O. The two remaining hours of the operation were without incident.

Blood loss was estimated to be 500 ml. Intravenous fluid administration consisted of 2,500 ml of Ringer's lactate solution and 500 ml of 5 per cent dextrose in water. Urinary output was 1,250 ml. Neuromuscular blockade was reversed with neostigmine.

Arterial blood gases showed two changes intraoperatively. The alveolar–arterial oxygen difference increased from an initial value of 60 torr (F_{IO_2} = 0.4) to 403 torr after embolization (F_{IO_2} = 1.0), and Pa_{CO_2} increased 20 torr after embolization, despite constant minute ventilation.

The endotracheal tube was left in place and the patient, who was breathing spontaneously, was taken to the Intensive Care Unit. He awakened promptly, and the findings on neurologic examination were the same as prior to operation. He was given humidified oxygen via a T-tube assembly, and Pa_{O_2} was 165 torr at F_{IO_2} = 0.93. Auscultation of the chest revealed diffuse rales bilaterally. A portable chest roentgenogram taken one hour postoperatively showed a butterfly pattern consistent with an interstitial infiltrate of pulmonary

* Resident in Anesthesiology.
† Resident in Neurological Surgery.
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edema (fig. 1). The deadspace-to-tidal volume ratio was 0.55 and the alveolar–arterial oxygen tension difference was 468 torr. Arterial–venous oxygen content differences were 5.3 and 6 vol per cent on two occasions.

Over the next six hours the patient was treated with positive-pressure ventilation and 10 cm H_2O PEEP; Pa_{O_2} improved. However, a second chest roentgenogram six hours postoperatively showed increased pulmonary edema. The patient was then treated with intravenous administration of plasma and furosemide.

On the first postoperative day the patient's condition was improved, and positive-pressure ventilation with PEEP was changed to intermittent mandatory ventilation[1] with PEEP. The patient's condition continued to improve. PEEP was discontinued, then IMV, and on the second postoperative day the endotracheal tube was removed. A subsequent roentgenogram showed clearing of both lungs; the alveolar–arterial oxygen difference had decreased to 117 torr at $F_{I_{O_2}} = 0.38$. The patient was discharged on the tenth postoperative day, without sequelae.

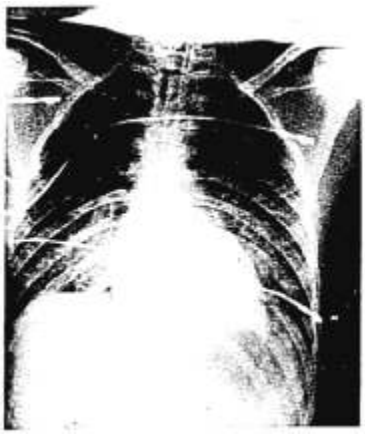

FIG. 1. Chest roentgenogram one hour postoperatively, showing an interstitial infiltrate consistent with pulmonary edema.

DISCUSSION

Animal studies have shown that right ventricular pressure is acutely elevated[2] and pulmonary arterial hypertension occurs with air embolization.[3-5] Increases in alveolar–arterial oxygen gradients and pulmonary edema also have been found in animals receiving lethal or near-lethal air embolization.[6] These changes are usually accompanied by systemic hypotension. However, we are unaware of any previous report of respiratory complications secondary to air embolism in humans.

Pulmonary edema can result from pulmonary vascular leaking from increased pressure, heart failure, hypoxia and loss of capillary integrity, and fluid overload.

The evidence does not point to fluid overload as a cause of the pulmonary edema in this patient. The fluid intake of 3,000 ml minus the 1,750-ml output leaves 1,250 ml intake of fluid by the 71-kg patient in 16 and a half hours. The calculated normal fluid requirements, exclusive of replacing blood loss, would be 1,650 ml, making fluid overload highly unlikely. The low central venous pressure and high specific gravity of urine immediately postoperatively (1.020 to 1.025) further support this conclusion.

Likewise, heart failure is not a reasonable cause for pulmonary edema in this patient. This is evidenced by a lack of fluid excess in a young healthy patient, a normal heart size on postoperative chest roentgenogram, interpreted as pulmonary edema not related to failure, and absence of any peripheral sign of heart failure. There was never any hypoxia or severe hypotension which would lead to cardiac decompensation, and the arterial–venous oxygen content difference was normal on each of two occasions, suggesting normal myocardial function.

The association of pulmonary arterial hypertension, intrapulmonary shunting, and pulmonary edema has been found in experimental animals given air emboli.[3-6] This patient presents a similar picture. Pulmonary hypertension may have been present, but was not documented. Air bubbles in the vascular space would account for the sudden increases in Pa_{CO_2} and $A\text{-}aD_{O_2}$ seen in this patient. Local hypoxia in the face of pulmonary hypertension could predispose to a fluid leak and pulmonary edema. One must conclude, therefore, that the air embolism probably caused acute pulmonary edema in this patient. This case report also emphasizes the importance of proper placement of the venous

catheter and meticulous postoperative follow up and care of such patients.

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Minimal-flow Nitrous Oxide Anesthesia

ROBERT W. VIRTUE, M.D.*

The common practice of using high flows of nitrous oxide and oxygen for anesthesia is wasteful[1-3] and is responsible for pollution of the atmosphere of operating rooms which may have harmful effects on health of personnel.[4-7] Availability of an oxygen electrode[8] has made it practical to measure within seconds the concentration of oxygen in an anesthetic system, thereby permitting the use of nitrous oxide in a closed system. The following report presents data accumulated using an oxygen electrode to minimize the waste of nitrous oxide and other agents during clinical anesthesia.

METHODS

Three groups of adult patients were studied. Current anesthetic methods were employed, namely, induction with thiopental, relaxation where necessary with gallamine, succinylcholine, and *d*-tubocurarine, and flows of 3½ l of nitrous oxide and 1½ l of oxygen for 15 to 20 minutes to eliminate nitrogen from the system and provide for absorption of generous amounts of nitrous oxide. At 15 minutes or shortly after, the flows of both N_2O and O_2 were decreased for the first group of 118 patients to quantities which would maintain the inflow concentration of O_2 at 35 per cent (arbitrarily chosen) and would keep the reservoir bag at a constant volume. This necessitated minute-to-minute adjustments of gas flows. Oxygen concentrations were monitored throughout the procedures using Hudson, Harlake, and Foregger oxygen meters, which were calibrated daily with pure oxygen and room air. Minute flows of gas necessary to achieve these conditions were recorded each 5 minutes. Ventilation of the patients was measured by means of either a graduated bellows or a Wright ventilation meter. Respiration was manually controlled at 1.5 times that predicted from the Radford nomogram.[9]

Group II consisted of 108 patients who were anesthetized as were those of Group I, but were mechanically ventilated at 1½ times the level predicted from the Radford nomogram 15 to 25 minutes after induction. Gas flows were decreased to 300 ml O_2 and 200 ml N_2O using either halothane or methoxyflurane as needed. The oxygen concentration of the inhaled gas was monitored and data were recorded each 5 minutes.

A third group of 117 patients was anesthetized and mechanically ventilated as in Group II, with the exception that gas inflows at 20 minutes were decreased to 500 ml each of N_2O and O_2. Data were recorded at one, two and three hours.

* Department of Anesthesia, General Rose Hospital, Denver, Colorado; Professor Emeritus, University of Colorado.

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