is the first reported case of an endotracheal tube fire ignited by "spray"-type coagulation cautery.

Three factors are necessary to create our complication: 1) a carbon source or fuel (PVC endotracheal tube); 2) an oxidant (N_2O/O_2 environment caused by a retrograde gas leak); and 3) an ignition source (electrocautery). In attempting to change the carbon source, most authors recommend using red rubber or silicone tubes for laser surgery,[3-5] avoiding PVC tubes. PVC tubes are more easily ignited by laser, and, once ignited, produce a more intense and uncontrollable flame.[4] This may be secondary to the release of vinyl chloride gas, which is itself highly flammable.

In attempting to change the oxidant, studies have shown that the N_2O/O_2 combination may support combustion better than 100% O_2.[6] Only when decreasing the O_2 below 30% in N_2 was there a difference in the flammability of endotracheal tubes.[7,8] Although studies have been done with respect to lasers,[3,5] no controlled study has been performed to determine the effect of varying these factors with electrocautery as the ignition source.

Perhaps when using intraoral or pharyngeal electrocautery, especially when set on "spray," we should consider using cuffed endotracheal tubes, even in children. This would leave an atmosphere of air in the mouth and pharynx. Perhaps we should avoid the use of electrocautery in close proximity to PVC endotracheal tubes.

In summary, we report a case of a pharyngeal electrocautery-ignited endotracheal tube fire. For laser surgery, using red rubber or silicone tubes and less than 30% O_2 in N_2 in the inspired gas is beneficial. It is not clear whether these data are applicable to electrocautery-ignited endotracheal tube fires.

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Massive Air Embolism during Cesarean Section

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Air embolism may occur during any surgical procedure in which the operative field is above the level of the heart.[1-3] That such a catastrophe has complicated pregnancy or the puerperium has been confirmed almost exclusively at post mortem examinations.[4-9] In particular, venous air embolism during cesarean section has a high incidence of mortality,[5,6,10,11] with a devastating morbidity should the patient survive.[12] Rapid, accurate diagnosis is essential for the successful therapy of this life-threatening complication. We describe a case of significant air embolism during cesarean section that was diagnosed intraoperatively by an acute decrease in the end-tidal CO_2 tension and by aspiration of air from the central venous circulation.

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Report of a Case

A 28-yr-old, 92-kg, G_4 P_3 Ab_0 patient at term gestation arrived at the labor suite with massive vaginal hemorrhage. Her pregnancy had been noteworthy for numerous hospital admissions due to vaginal bleeding from a complete placenta previa. Her past medical history revealed a 8- to 10-yr history of intermittent wheezing self-treated

with nonprescription inhalers, but the wheezing had not been present for several months. Her blood pressure was 115/70 mmHg, heart rate 120 beats/min, respiratory rate 16 breaths/min, and oral temperature 36.7° C. The patient appeared slightly anxious, but was very cooperative and in no obvious distress. A hematocrit obtained 8 h previously was 33%. Approximately 500–700 ml of bright red blood and clots soaked her bed sheets.

Two 16-gauge iv catheters were inserted under local anesthesia prior to the patient's receiving 1,800 ml of Plasmalyte A® solution. She was given 30 ml of antacid orally and 0.2 mg of glycopyrrolate iv. The patient was then placed supine on the operating room table with 20° left uterine displacement. A Dinamapp®, ECG electrodes, precordial stethoscope, skin temperature probe, and a pulse oximeter were applied. An in-line O_2 analyzer and an end-tidal CO_2/O_2 monitor were calibrated and inserted into the anesthesia circuit.

Following breathing 100% oxygen, general endotracheal anesthesia was induced using a rapid-sequence technique consisting of cricoid pressure, *d*-tubocurarine 3 mg iv, lidocaine 120 mg iv, ketamine 75 mg iv, and succinylcholine 100 mg iv. Anesthesia was maintained with $N_2O:O_2$ 4:4 l/min while muscle relaxation was provided by a 0.2% succinylcholine infusion. The pulse oximeter registered a hemoglobin saturation of 99% and the end-tidal CO_2 monitor displayed a partial pressure of 26–28 mmHg.

The operating table was completely level at the start of the cesarean section, which was performed through a classic incision. Immediately after the delivery of a viable female infant (1-min Apgar of 4, 5-min Apgar of 8), massive hemorrhage occurred at the operative site. The attendant surgeons quickly proceeded to perform an abdominal hysterectomy. The uterus at all times remained within the abdominal cavity. During the initial moments of this procedure the end-tidal CO_2 monitor failed to register any expired carbon dioxide; an idioventricular rhythm appeared on the ECG monitor; the arterial blood pressure became unobtainable. Bilateral breath sounds were heard, and the lungs were ventilated with an $F_{I_{O_2}}$ of 1.0. Although the blood in the operative field remained bright red, the patient herself appeared mottled and cyanotic. Resuscitative measures following the ACLS protocol§ were instituted. The patient's rhythm degenerated into asystole and simultaneously the pulse oximeter registered a hemoglobin saturation of 38–42%.

Moments later, the attendant surgeons noted that air was bubbling through the flooded surgical field following dislodgement of a clamp from the uterine artery. The diagnosis of air embolism was made, and the patient was placed in the extreme left-lateral tilt and head-down position. Surgery and resuscitative efforts continued while a central venous catheter was inserted through the right internal jugular vein. Frothy air (1.5 ml) was then aspirated from the central venous circulation.

Forty-five minutes after the initial surgical incision, the patient was successfully resuscitated. At the time of wound closure, vital signs were: arterial blood pressure 160/110 mmHg, heart rate 110 beats/min, respiratory rate 6 breaths/min and labored, esophageal temperature 33° C. The estimated surgical blood loss was 1,800 ml. Although transfusion with five units of packed red blood cells produced a central venous pressure (CVP) of 14 mmHg, urine output was less than 10 ml after a normal perfusion pressure had been assured for over 1 h. She was given furosemide, 40 mg iv, mannitol, 12.5 g iv, and a dopamine infusion at 3 $\mu g \cdot kg^{-1} \cdot min^{-1}$. These measures resulted in a brisk diuresis. During mechanical ventilation, analysis of her arterial blood gases with an $F_{I_{O_2}}$ 1.0, tidal volume 900 ml, respiratory rate 10 breaths/min, and 5 cm PEEP revealed Pa_{O_2} 270 mmHg, Pa_{CO_2} 37 mmHg, pH_a 7.32, and a hemoglobin saturation of 99%. A brief neurologic examination performed before sedation was given revealed 3–4 mm, midpositioned pupils equally reactive to light, bilateral upgoing plantar responses, and some purposeful spontaneous movements. She did not respond to her name.

The patient was transferred to the intensive care unit (ICU). Her trachea remained intubated, and ventilation was controlled to achieve normocarbia and maximal hemoglobin saturation. She was allowed to remain mildly hypothermic. Blood glucose concentrations were closely monitored to maintain normoglycemia. Phenytoin, 250 mg, was given slowly iv as tolerated hemodynamically. A dopamine infusion of 1.0–2.0 $\mu g \cdot kg^{-1} \cdot min^{-1}$ was titrated as needed to maintain a urine outflow of greater than 100–150 ml/h. The CVP was maintained between 8–12 mmHg with infusion of crystalloid solutions and blood as needed. Minimal sedation was provided with small doses of diazepam iv.

With the use of this conservative regimen, the patient was weaned from ventilatory support, and her trachea extubated 22 h after surgery. She was closely monitored in the ICU for 72 h in order to detect any sudden deterioration in mental status. Repeated neurologic examinations revealed no neurologic deficits except for a slight and rapidly resolved impairment of short-term memory. She was subsequently discharged to her home, fully ambulatory and able to care for her child.

Discussion

In 1947 Durant *et al.*[1] were among the first to point out that the uterine sinuses are theoretically vulnerable to the entrance of air, especially in the presence of placenta previa or during manual extraction of the placenta. Indeed, precordial ultrasonic Doppler changes consistent with venous air entrainment were first documented in 1985 in 40% of women during cesarean section.[13] Thus, the pregnant patient undergoing abdominal delivery may be at far greater risk for development of a hemodynamically significant air embolism than is generally realized. Such a risk is obviously magnified if a hypovolemic patient is placed in the Trendelenburg position.

Precordial Doppler monitoring is a simple and efficacious method for detecting the presence of intracardiac air prior to the development of significant circulatory changes.[14,15] This allows time for the surgical team to halt the entrapment of air at its source. However, precordial Doppler monitoring alone will not permit distinction between cardiovascular collapse resulting from venous air embolism and that occurring from pulmonary thromboembolism. Once hypovolemia has been excluded, an appropriately functioning end-tidal CO_2 monitor would provide rapid detection of an acute decrease in CO_2 excretion from either cause. Because hemodynamically significant air embolism during caesarean section is admittedly a rare event, the use of additional precordial Doppler monitoring may be reasonably restricted to cases considered also at risk for air embolism, such as those involving profound hypovolemia, abruptio placentae, or placenta previa.

If massive air embolism occurs without premonitory signs, the surgical field should be flooded and the patient should be positioned with extreme left-lateral tilt and her

§ McIntyre KM, Lewis AJ: Textbook of Advanced Cardiac Life Support. American Heart Association, 1983, pp 297, 300.

chest dependent.[16-18] Nitrous oxide should be discontinued immediately and the lungs ventilated with an FI_{O_2} of 1.0.[19] A catheter should be inserted into the central venous circulation for aspiration of air and infusion of resuscitative drugs. It is important that circulatory and ventilatory support be maintained without interruption, because once in the pulmonary circulation the entrapped air can be excreted only by diffusion through the lungs.[20]

Vigilant post-resuscitative care is essential for a favorable neurologic outcome. Hypotension, hypoxemia, and hypoglycemia should be avoided. Seizure activity is a metabolically expensive event for the postanoxic brain; therefore, it should be suppressed with anticonvulsants. Focal neurologic deficits suggest the presence of a patent foramen ovale, and care should be taken that no further air be entrained through peripheral or central venous access. Finally, repeated neurologic examinations are mandatory for the detection and prompt therapy of potentially fatal cerebral edema.

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Predicting the Need for Postoperative Mechanical Ventilation in Myasthenia Gravis

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Myasthenia gravis causes weakness of voluntary muscles, sometimes including those of respiration, so that patients with this diagnosis may be at increased risk of developing postoperative respiratory failure. Several authors have proposed criteria for predicting which myasthenic patients will require prolonged postoperative mechanical ventilation.[1-3] When Leventhal *et al.* recently described such a preoperative scoring system for myasthenic patients

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