sideration for tracheal tube lubrication, the addition of a lubricant seems unnecessary. However, lubrication of the tracheal tube may be used as a vehicle for drug administration, e.g., lidocaine. Or, it may be used to facilitate passage of the tube through the oropharynx, in which case, the least expensive lubricant may be chosen.

The authors thank Ms. Joan Alster for statistical evaluation of the data, and Ms. Lynn Carroll for editorial assistance.

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Air Embolism During Radical Hysterectomy

J. Stephen Naulty, M.D., Lee B. Meisel, M.D., Sanjay Datta, M.D., Gerard W. Ostheimer, M.D.

Air embolism has been reported during surgery involving the head and neck in intracranial procedures in the sitting position, during laparoscopy and hysteroscopy, and as a hazard of intravenous therapy. Its pathophysiologic factors and treatment have been discussed extensively, and precordial doppler monitoring, right atrial or pulmonary artery catheterization, and end-tidal carbon dioxide measurement have become accepted procedures when air embolism is anticipated. Although several reviews mention the possibility of air embolism during pelvic procedures, no case reports of such an occurrence have been reported since Larson's postmortem description of intracardiac air following a cardiac arrest in 1951. The following report describes a case of massive air embolism during hysterectomy in a situation in which air embolism was not anticipated, and in which no monitoring devices were in place to allow rapid diagnosis and treatment.

REPORT OF A CASE

A 26-year-old, 60-kg woman underwent abdominal hysterectomy with bilateral pelvic node dissection for invasive cervical carcinoma. Her past medical history was unremarkable and preoperative laboratory studies, physical examination, chest roentgenogram, and electrocardiogram were normal. The patient was classified as ASA physical status I.

The patient received 6 mg morphine sulfate and 0.4 mg atropine, im. Ninety minutes later, anesthetia was induced with 300 mg thiopental, iv. The trachea was intubated after neuromuscular blockade was induced by 100 mg succinylcholine, iv. Anesthesia was maintained with 70% nitrous oxide and 1–2% enflurane. Tubocurarine was administered intravenously to provide muscle relaxation, but the patient was not paralyzed completely. Ventilation at a minute volume of 5.4 l was controlled with a volume-limited ventilator. An ECG recorded cardiac rate and rhythm, and temperature was monitored via an esophageal thermistor probe. A Foley catheter was inserted in the bladder from which urinary output was monitored. The patient was positioned in Trendelenburg’s position (approximately 10° head-down tilt) and surgery was started.

The operation proceeded uneventfully until approximately three hours after induction of anesthesia. The vaginal cuff was being closed after the uterus and adnexa had been removed, when it was noticed that the patient had suddenly become deeply cyanotic, and was making gasping respirations. Frequent premature ventricular contractions were present on ECG, and the external jugular veins had become distended. Arterial blood pressure at this time was 90/60 mmHg, a slight decrease from previous values. Mechanical ventilation and administration of all anesthetic drugs were discontinued, and the correct placement of the endotracheal tube confirmed. Severe bradycardia then ensued, and heart sounds became absent from the esophageal stethoscope. No peripheral pulses were palpable and no blood pressure was obtainable via the sphygmomanometer. Atropine, 0.6 mg, and 20 mg ephedrine iv were administered. An aortic pulse was then palpable in the abdomen. Upon removal of the abdominal packs, the iliac veins were noticed to look “funny.” Upon inspection, they were found to contain large air bubbles. Simultaneously, heart sounds were noticed and a mill wheel murmur was found to be present. The inferior vena cava was occluded and the patient was placed in the steep Trendelenburg position. A mixture of air and foam, 60 ml, was aspirated from

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the exposed iliac veins by the surgeon, and a 24-inch Deseret intracavitary catheter was inserted into the inferior vena cava and 20 ml of air removed. The catheter was aspirated until no further air was obtained. The heart rate and blood pressure quickly returned to pre-embolus levels, but her pupils became fixed and dilated. Decadron, 10 mg, iv, was administered and anesthesia re instituted with enfurane and oxygen when the blood pressure rose to 150/90 mmHg. The abdominal incision was closed rapidly and no air could be aspirated from the left internal jugular vein. Continuous precordial doppler monitoring revealed no further air embolism. Arterial blood gases obtained 5 minutes post embolization (FiO2 1.0) revealed a PaO2 of 66 mmHg, PaCO2 46 mmHg, and a pH of 7.30. Transient ST depression was noticed on the ECG monitor, which resolved in approximately 10 min.

The anesthesia was discontinued 3.5 hours after induction of anesthesia and the neuromuscular blockade reversed with 1.0 mg atropine and 2.5 mg neostigmine, iv. The patient quickly regained consciousness and adequate ventilation, and the trachea was then extubated. Her recovery room stay was uneventful, and her postoperative chest roentgenogram revealed increased vascular markings, which cleared 3 h after administration of 10 mg furosemide, iv. A ventilation-perfusion scan (technetium aggregated albumin) was performed 6 h post-embolus and revealed "multiple filling defects consistent with air embolism."

**DISCUSSION**

When the possibility of air embolism is anticipated, (e.g., craniotomy in a sitting position), procedures for diagnosis of embolization should be employed constantly, and immediate treatment should be possible. In cases where air embolus is not anticipated, the etiologic factors of the sudden cardiovascular collapse may be obscure, as in our patient, and proper treatment may be delayed. In this case, the esophageal stethoscope apparently gave little or no warning of the embolus, as has been reported previously. The rapid development of bradycardia and cardiovascular collapse undoubtedly contributed to this lack of development of the classic "millwheel" murmur. Atropine and ephedrine rapidly restored cardiac activity, and later, when heart sounds were heard, the murmur was present.

The diagnostic possibilities we initially considered included compression of the inferior vena cava by abdominal packs; an anesthesia ventilator-machine malfunction leading to hypoxia; myocardial ischemia; and pulmonary thromboembolism. The possibility of air embolism initially was not considered, although the diagnosis was obvious in retrospect, since several features of the syndrome of air aspiration were evident. To enable others encountering a similar episode to reach the correct diagnosis more quickly, we shall briefly discuss some of the pathophysiologic characteristics of air embolism.

A necessary condition for the development of air embolism is the presence of an open vein, and the pressure in the vein must be less than the ambient pressure. Such conditions may exist when dissection is performed around the pelvic veins. These veins exposed during radical hysterectomy are large, thin walled, and difficult to delineate. In the Trendelenburg position, the pressure in this venous plexus may be subatmospheric, particularly if the central venous pressure is low. In this situation, large amounts of air may be entrained in the venous system. The cardiovascular effects or air embolism depend upon the rate of air infusion. Adornato et al. found that if a sufficient bolus of air (1 ml/kg) rapidly enters the right atrium and ventricle of a dog, a froth is produced which impairs the function of the cardiac valve and reduces cardiac output suddenly, the so-called "air-lock." This produces a sudden hypotension and the appearance of cardiovascular collapse. With slower rates of infusion (0.4–1.8 ml·kg⁻¹·min⁻¹), the air passes through the right side of the heart, and occludes the pulmonary circulation. This produces an acute increase in right ventricular afterload, a rise in pulmonary artery and central venous pressures, and myocardial ischemia with right ventricular strain. With slow rates of infusion, a characteristic "gasp" may occur, which by acutely decreasing the central venous pressure, may convert a sublethal slow infusion of air into a lethal bolus.

Our patient clearly demonstrates both types of air embolism. At first, a slow infusion of air probably took place, leading to the acute rise in jugular venous pressure, arrhythmias, and the gasping respiration typical of air embolism. We postulate that these gasps exacerbated the air entrainment, causing the patient's momentary cardiac arrest. By placing her in steep Trendelenburg position and aspirating the air, her cardiac function was restored. This procedure was not without risk, since theoretically, we could have increased the amount of air aspirated with this maneuver. The exposed iliac veins allowed the most rapid access for removal of the venous and intracardiac air, and this was apparently successful.

The infusion of air into the pulmonary artery also produces hypoxia, presumably on a reflex basis, and occurs with a volume of approximately 1 ml·kg⁻¹·min⁻¹ of air embolization. Pulmonary edema has been reported following air embolism. This contributes to the hypoxia which may follow air embolism. Our patient demonstrated severe, transient hypoxia following the episode, which slowly resolved. She also experienced a transient hypercarbia, reflecting the increased V/Q ratio which may develop following air embolism. Interestingly, even hours later, perfusion defects were evident on V/Q scanning, although by that time her arterial blood gases had returned to normal. A V/Q scan two days later revealed resolution of these perfusion defects. The persistence of the perfusion deficit in this case seems unusual, but pulmonary edema following air embolism may persist for 16–24 hours.

The treatment of air embolism is accomplished by
stopping the entrainment of air, minimizing the size of the air embolus, and removing air from the heart and great vessels. In the usual setting, e.g., in craniotomies in the sitting position, this can be accomplished by flooding the wound with saline, placing the patient in the head-down position, occluding the jugular veins, and removal of air via a previously placed central venous catheter. Obviously some of these maneuvers were impossible in our patient, and the possible one, placing the patient in the head-down position, carried the risk of worsening the air embolism by increasing the negative pressure in the pelvic plexus. When this maneuver was performed in our patient, we were unaware of the presence of the air embolus. When the diagnosis was made, we kept the patient in this position until the air was removed, since it appeared to improve her cardiovascular function. The surgeons manually occluded the iliac vein to reduce the likelihood of further aspiration of air, and passed an intravenous catheter quite rapidly via the same vein, to remove the intravenous and intracardiac air. It probably would have been helpful to perform Durant’s maneuver (left lateral decubitus position with head-down tilt) and flood the pelvis with saline solution, but by the time the diagnosis of air embolism had been made, the patient had improved sufficiently to make these maneuvers unnecessary. Prompt discontinuation of nitrous oxide should reduce bubble size.10

The incidence of air embolism during pelvic procedures is unknown. It probably is an uncommon occurrence, since we found only one previous report of its occurrence during open pelvic surgery, which was not recognized as an air embolism until postmortem examination.11 It has been noticed during gas infusion for laparoscopy, hysteroscopy, and during parturition and sexual activity.1 Radical hysterectomy with extensive dissection around the pelvic veins may be a relatively higher risk procedure than other pelvic procedures. We are now monitoring patients with a precordial doppler and inserting right atrial catheters during radical hysterectomies in an attempt to determine the incidence of air embolism in these procedures, and aspirate air from the heart should it occur.

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