contractures. Stimulation of the orbicularis oculi muscle can provide a reliable trend for monitoring neuromuscular paralysis, but may underestimate the degree of neuromuscular blockade of other muscles.[10]

This patient presented with both an airway that appeared difficult to manage and a history of gastroesophageal reflux. General anesthesia or sedation prior to endotracheal intubation might have resulted in inability to ventilate the lungs or in aspiration of gastric contents. An awake intubation with local anesthesia was planned. Physical restraint was not necessary because of marked immobility due to the patients disease. Several reports have described unique methods to establish and maintain a patent airway in pediatric patients.[8–10] The need to inspect her larynx for tissue hyperplasia made tracheal intubation assisted by fiberoptic visualization ideal. A 3.2-mm pediatric fiberoptic bronchoscope can be inserted through tracheal tubes with an internal diameter of 4.5 mm and larger.[9,10] A 4.5-mm tracheal tube was too large for our patient and likely would have caused laryngeal trauma. We inserted a soft-tipped guide wire through the suction port of the bronchoscope to use as a guide for the tracheal tube. The technique allowed laryngeal visualization to confirm correct tube placement and could be used to pass any size endotracheal tube.[10]

In summary, juvenile hyaline fibromatosis is an unusual disease with profound anesthetic implications. Assessment of multiple organ involvement, uneventful tracheal intubation assisted by placing a guide wire through a pediatric bronchoscope, care in positioning, and appropriate neuromuscular monitoring all led to an uneventful outcome.

References

1. Fayad MN, Yacoub A, Salman S, Khudr A, DerKaloustian VM: Juvenile hyaline fibromatosis: Two new cases and a review of the literature. Am J Med Genet 26:123–131, 1987
2. Remberger K, Krieg T, Kunze D, Weinmann H, Hubner G: Fibromatosis hyalinica multiplex (juvenile hyaline fibromatosis). Cancer 56:614–624, 1985
3. Aldred M, Crawford P: Juvenile hyaline fibromatosis. Oral Surg Oral Med Oral Pathol 63:71–77, 1987
4. Marty-Double C, Balmes P, Mary H, Allieu Y, Pignoddel C, Targhetta R, Lesbros D, Metge L: Juvenile fibromatosis resembling aponeurotic fibroma and congenital multiple fibromatosis. Cancer 61:146–152, 1988
5. Teng P, Warden M, Cohn WL: Congenital generalized fibromatosis (renal and skeletal) with complete spontaneous regression. J Pediatr 62:748–753, 1963
6. Plaschkles J: Congenital fibromatosis: Localized and generalized forms. J Pediatr Surg 9:95–101, 1974
7. Caffrey RD, Warren ML, Becker KE: Neuromuscular blockade monitoring comparing the orbicularis oculi and adductor pollicis muscles. ANESTHESIOLOGY 65:95–97, 1986
8. Borland L, Swan D, Leff S: Difficult pediatric endotracheal intubation: A new approach to the retrograde technique. ANESTHESIOLOGY 55:577–578, 1981
9. Rucker R, Silva W, Worcester CC: Fiberoptic Bronchoscopic Nasotracheal Intubation in Children. Chest 76:56–58, 1979
10. Patil V, Stehling L, Zauder HL: Fiberoptic Endoscopy in Anesthesia. Chicago, Year Book Medical Publishers Inc., 1983, p 113

Anesthesiology
72:203–205, 1990

Venous Air Embolism in the Recovery Room Producing Unexplained Cardiac Dysrhythmias: A Case Report

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Venous air embolism is a well-known complication of neurosurgical procedures performed upon patients who are in the sitting position but has also been reported during head and neck surgery, total hip replacement, cesarean section, and other surgical procedures.[1–3] The authors are unaware of any previous report of this complication occurring in a patient in the recovery room. We report a case of venous air embolism in the recovery room initially manifest as unexplained cardiac dysrhythmias in a patient recovering from general anesthesia for posterior cervical fusion.

Report of a Case

A 14-yr-old female sustained an odontoid fracture without neurologic damage in a motor vehicle accident. Despite immobilization in a halo vest since the time of injury, the fracture did not heal. Four months after sustaining the injury, the patient was admitted to the hospital for elective C1-C2 fusion. The patient's past medical history was unremarkable with the following exception: while being observed in the intensive care unit at the time of the initial injury, unifocal PVCs were noted. Antidysrhythmics were not required, and the ectopy resolved

Received from the Department of Anesthesiology, Mayo Clinic and Mayo Foundation, Rochester, Minnesota 55905. Accepted for publication August 22, 1989.

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Key words: Embolism: air. Heart: dysrhythmia.

spontaneously within 24 h. The etiology of the dysrhythmia was unclear. Physical examination, laboratory studies, chest x-ray, electrocardiogram, echocardiogram, and cardiac enzymes were unremarkable.

On the current admission, the patient denied any history of palpitations, chest pain, dyspnea, orthopnea, or syncope. She was taking no medication. Auscultation of the heart and lungs was normal. The patient was ambulating without difficulty and was neurologically intact by physical examination. Preoperative chest x-ray, ECG, hemoglobin, electrolytes, and somatosensory evoked potentials were normal.

The patient was brought to the operating room with the halo vest in place and was monitored initially with ECG, pulse oximeter, and blood pressure cuff. ECG showed normal sinus rhythm. Awake fiberoptic nasotracheal intubation was performed without difficulty. No vasoconstrictors such as epinephrine or cocaine were used to facilitate intubation. Anesthesia was induced with thiopental 3 mg/kg, fentanyl 8 μg/kg, and pancuronium 0.1 mg/kg iv. The patient was turned prone and the head stabilized on a "horseshoe" headrest. All parts of the halo vest system were removed from the patient with the exception of the halo which remained fixed to the patient's skull by four screws. The patient's lungs were mechanically ventilated. Anesthesia was maintained with 70% nitrous oxide in oxygen, fentanyl infusion at 2 $\mu g \cdot kg^{-1} \cdot h^{-1}$, and isoflurane. Pancuronium was used to maintain muscle relaxation. Intraoperative monitoring consisted of ECG, blood pressure cuff, pulse oximeter, esophageal stethoscope and temperature probe, capnometer, peripheral nerve stimulator, radial artery catheter, Foley catheter, and cortical somatosensory evoked potentials (SSEP).

The operation was performed with the patient in the prone position. The duration of the surgical procedure was 4.5 h and the anesthetic course was unremarkable. Vital signs remained stable throughout the surgical procedure. No episodes of hypotension, dysrhythmias, sudden decreases in end-tidal CO_2, or arterial desaturation occurred. Arterial blood gases and serum electrolytes remained within normal limits and SSEPs remained unchanged. Estimated blood loss was less than 200 ml. No blood or blood products were transfused.

The four screws holding the halo in place on the patient's head were the original screws placed 4 months earlier at the time of the initial injury. To reduce the risk of infection, at the end of the surgical procedure and with the patient still anesthetized, screws were inserted at four new sites and the old screws removed. No dressing was applied at the old screw sites. The vest was reattached to the halo and the patient turned supine. Isoflurane and N_2O administration were discontinued and neuromuscular blockade was reversed with atropine 20 μg/kg and neostigmine 60 μg/kg iv. The patient emerged from general anesthesia and moved all extremities to command. The ECG showed normal sinus rhythm with BP 132/80, HR 78, and temperature 97.6° F. The patient was transported to the recovery room supine, breathing spontaneously, with the endotracheal tube in place and supplemented with oxygen by T-piece. ECG and arterial line monitoring were not used during transport to the recovery room.

The patient arrived in the recovery room in the supine position. As soon as ECG monitoring was re-established, intermittent multifocal PVCs, PACs, and bigeminal rhythm were observed. The patient was hemodynamically stable with BP 120/60–140/75, HR 78–105, and respirations 8–12 per min. Oxygen saturation was 100% by pulse oximeter. The patient denied chest pain and shortness of breath. Arterial blood gases obtained immediately revealed Pa_{O_2} 374, Pa_{CO_2} 37, *p*H 7.36, BE −3.2, Hb 9.5, K^+ 3.7. Dysrhythmias persisted despite lidocaine 1.5 mg/kg iv.

With the likely etiologies of the dysrhythmias ruled out, we considered the possibility of air entrainment from one of the original halo screw sites. A precordial Doppler was positioned over the patient's right parasternal region. With each inspiration, Doppler sounds of venous air embolism were clearly audible. During inspiration, Doppler sounds of venous air embolism were most intense and between breaths, the Doppler gradually cleared. Also, the frequency of ectopic beats seemed greatest during inspiration. After placing betadine-coated gauze pads over the original screw sites, Doppler sounds of venous air embolism were no longer heard. The frequency of ectopic beats decreased dramatically, but a rare PVC, less than 1 beat per min was still observed. Three of the four original screw sites were covered with betadine ointment and a bandage. The muscle and soft tissue overlying the old left anterior screw site was closed with suture; during removal from the site intraoperatively, this screw was noted to be inserted deeper than normal and had likely perforated the inner table of the skull.

Despite venous air embolism and dysrhythmias, the patient at no time became hypotensive. Chest x-ray was unremarkable and 12-lead ECG showed no evidence of myocardial ischemia. The trachea was extubated and the remainder of the recovery room stay was uneventful. The patient was observed in the intensive care unit overnight. Rare unifocal PVCs were observed while in the ICU but resolved by the next morning. ECG, serial cardiac enzymes, and echocardiogram obtained on the first postoperative day were normal.

Discussion

Venous air embolism can occur whenever a noncollapsible venous channel is open to the atmosphere and a negative pressure gradient exists between the vessel opening and the right atrium. Neurosurgical patients operated upon while in the sitting position are at risk for venous air embolism intraoperatively because the surgical site is located above the level of the heart.[1–3] Most commonly, air entrainment occurs from a site within the operative field. However, sites remote from the operative field such as burr holes and pinion sites of neurosurgical head holders have been identified as the source of air entrainment during sitting neurosurgical procedures.[4–7] Air entrainment from screw sites of a halo frame might be expected if this patient had experienced VAE in the sitting position. What is most significant in this case is the occurrence of VAE with the patient in the supine position and manifest initially in the recovery room as cardiac dysrhythmias.

With the patient supine, the hydrostatic pressure gradient between the original anterior screw sites and the right atrium must have been close to zero. We postulate that a negative pressure gradient was created between the screw site(s) and right atrium as a result of negative intrathoracic pressure generated during inspiration. In spontaneously breathing subjects, during inspiration a negative pressure gradient is created between intrathoracic and extrathoracic structures. It is likely that this same pressure gradient that is necessary for airflow into the lung during inspiration was responsible for air entrainment into open venous channels at the original screw sites. This phenomenon has been demonstrated in experimental animals. In supine, spontaneously breathing dogs with a catheter in the external jugular vein and left open to the atmosphere, air entrainment was observed during inspiration.[8]

This mechanism of air entrainment helps to explain why VAE and the associated dysrhythmias were not manifest until arrival in the recovery room. Neuromuscular

blockade was not reversed until just prior to transfer to the recovery room. With conversion from controlled ventilation, during which intrathoracic pressure is never less than atmospheric, to spontaneous ventilation, in which intrathoracic pressure is less than atmospheric during inspiration, the problem of venous air entrainment developed. Because the patient was transported to the recovery room without ECG monitoring, cardiac dysrhythmias were unrecognized until ECG monitoring was resumed in the recovery room.

Common causes of cardiac dysrhythmias such as hypoxia, hypercarbia, anemia, hypo- or hyperkalemia, and myocardial ischemia were immediately ruled out after arrival in the recovery room. Michenfelder has reported that common dysrhythmias associated with VAE are PVCs and bigeminy,[9] both of which occurred in our patient. However, only after excluding the common causes of dysrhythmias did we consider the possibility of VAE. The diagnosis of VAE was confirmed using a precordial Doppler. The precordial Doppler has been shown to be one of the most sensitive monitors for VAE.[10] Doppler sounds were most intense during inspiration and gradually cleared during expiration and between breaths. Although air was most likely being entrained by the patient during inspiration, changes in the intensity of Doppler sounds during the respiratory cycle may also have been due, at least in part, to positional changes of the Doppler relative to cardiac structures. Because air was no longer heard on the Doppler and the cardiac dysrhythmias resolved after the left anterior screw site was occluded, suggests that air entrainment occurred from scalp veins or diploic veins in this area.

This case demonstrates several important points. Episodes of venous air embolism are not restricted to the intraoperative period and can occur during spontaneous respiration in patients in the supine position. Also, one should consider VAE in the differential diagnosis of cardiac dysrhythmias in patients with exposed venous channels when other more likely etiologies have been ruled out. Finally, the importance of patient monitoring during transport is reemphasized.

References

1. Black S, Ockert DB, Oliver WC, Cucchiara RF: Outcome following posterior fossa craniectomy in patients in the sitting or horizontal positions. ANESTHESIOLOGY 69:49–56, 1988
2. Spiess BD, Sloan MS, McCarthy RJ, Lubenow TR, Tuman KJ, Matz SD, Ivankovich AD: The incidence of venous air embolism during total hip arthroplasty. J Clin Anesth 1:25–30, 1988
3. Younker D, Rodriguez V, Kavanaugh J: Massive air embolism during cesarean section. ANESTHESIOLOGY 65:77–79, 1986
4. Wilkins RH, Albin MS: An unusual entrance site of venous air embolism during operations in the sitting position. Surg Neurol 7:71–72, 1977
5. Edelman JD, Wingard DW: Air embolism arising from burr holes. ANESTHESIOLOGY 53:167–168, 1980
6. Cabezudo JM, Gilsanz F, Vaquero J, Areito E, Martinez R: Air embolism from wounds from a pin-type head-holder as a complication of posterior fossa surgery in the sitting position. J Neurosurg 55:147–148, 1981
7. Pang D: Air embolism associated with wounds from a pin-type holder. J Neurosurg 57:710–713, 1982
8. Coles BC, Richardson HF, Hall GE: Experimental gas embolism: II. Factors other than air as a cause of death in some cases diagnosed "air embolism." Can Med Assoc J 37:24–25, 1937
9. Michenfelder JD, Martin JT, Altenburg BM, Rehder K: Air embolism during neurosurgery. JAMA 208:1353–1358, 1969
10. Glenski JA, Cucchiara RF, Michenfelder JD: Transesophageal echocardiography and transcutaneous O_2 and CO_2 monitoring for detection of venous air embolism. ANESTHESIOLOGY 64: 541–545, 1986

Anesthesiology
72:205–207, 1990

Fiberoptic Intubation Complicated by Pulmonary Edema in a 12-Year-Old Child with Hurler Syndrome

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Hurler syndrome (mucopolysaccharidosis storage disease, Type I-H) is an inherited progressive metabolic disorder.[1] The tongue, tonsils, adenoids, and nasopharyngeal tissues are hypertrophic in these children and may cause upper airway obstruction. Micrognathia, a very short neck, and restricted motion at the temporomandibular

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Received from the University of Minnesota Hospital and Clinic Minneapolis, Minnesota. Accepted for publication August 25, 1989.
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Key words: Anesthesia, pediatric: Hurler Syndrome. Anesthesia, techniques: fiberoptic tracheal intubation. Complications: pulmonary edema.