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Anesthesiology
1997; 87:1244-7

Air Embolization in Seated, Sedated, Spontaneously Breathing, Neurosurgical Patients

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INNOVATIVE neurosurgical operations are performed with increasing frequency in semi-sitting or head-up positions and without general anesthesia or extensive monitoring.[1-5] To demonstrate the necessity for proper monitoring, we present two cases of venous air embolism that developed in seated, sedated, spontaneously breathing patients soon after a cranial burr hole was placed and the dura was opened.

Case 1

A 47-yr-old man with progressive, medication-resistant Parkinson's disease underwent a pallidotomy with local anesthesia and conscious sedation. He was otherwise in good health. Intravenous midazolam, 1 mg, and fentanyl, 50 μg were administered to apply a preoperative stereotactic headframe for computed tomography. In the operating room, unconsciousness was induced with a 30 $\mu g \cdot kg^{-1} \cdot min^{-1}$ infusion of propofol. The patient became unresponsive to verbal commands, although breathing remained spontaneous and regular. O_2 was delivered *via* nasal prongs at 4 l/min. Electrocardiograph (ECG), blood pressure, O_2 saturation, and end-expired PCO_2 (infrared) were monitored with the respiratory gas sampling port in the posterior nasopharynx, 8 cm from the nares.[6] Data were obtained every 10 s, with median values recorded every 50 s (fig. 1). The scalp was incised with the patient in a 45° semi-sitting position, and a 0.5-inch cranial burr hole was drilled. Bone wax was applied, and the dura cauterized before a microelectrode was inserted into the brain. Soon thereafter, the patient began to cough and hyperventilate. Chest auscultation revealed normal heart and breath sounds. End-expired PCO_2 and O_2 saturation rapidly decreased from 40 to 11 mmHg and from 98 to 88%, respectively. Concurrently, respiratory rate increased from 14 to 38 breaths/min, and heart rate increased from 65 to 91 beats/min. The surgeons were notified, and the operative field was irrigated. The propofol infusion was discontinued. Blood pressure increased slightly at first and then decreased to 80/45 mmHg over 10 min. As consciousness was regained, coughing subsided, and vague chest discomfort was reported. The ST segments were unchanged. Without additional therapy, vital signs gradually returned toward baseline, and surgery proceeded without further complication.

Case 2

A 65-yr-old woman with an expressive aphasia and progressive hemiparesis had a brain biopsy with local anesthesia and conscious sedation. She received midazolam, 1.5 mg, fentanyl, 125 μg, and nasal O_2. She was placed in a 45° semi-sitting position.

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Received from the Departments of Anesthesiology and Neurosurgery, Columbia University, College of Physicians and Surgeons, New York, New York. Submitted for publication February 18, 1997. Accepted for publication June 12, 1997.

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Key words: Anesthesia: conscious or semi-conscious sedation. Monitoring: end-expired PCO_2; capnometry; O_2 saturation; pulse oximetry. Neurosurgery: craniotomy. Position: sitting. Respiration: spontaneous. Venous air embolism.

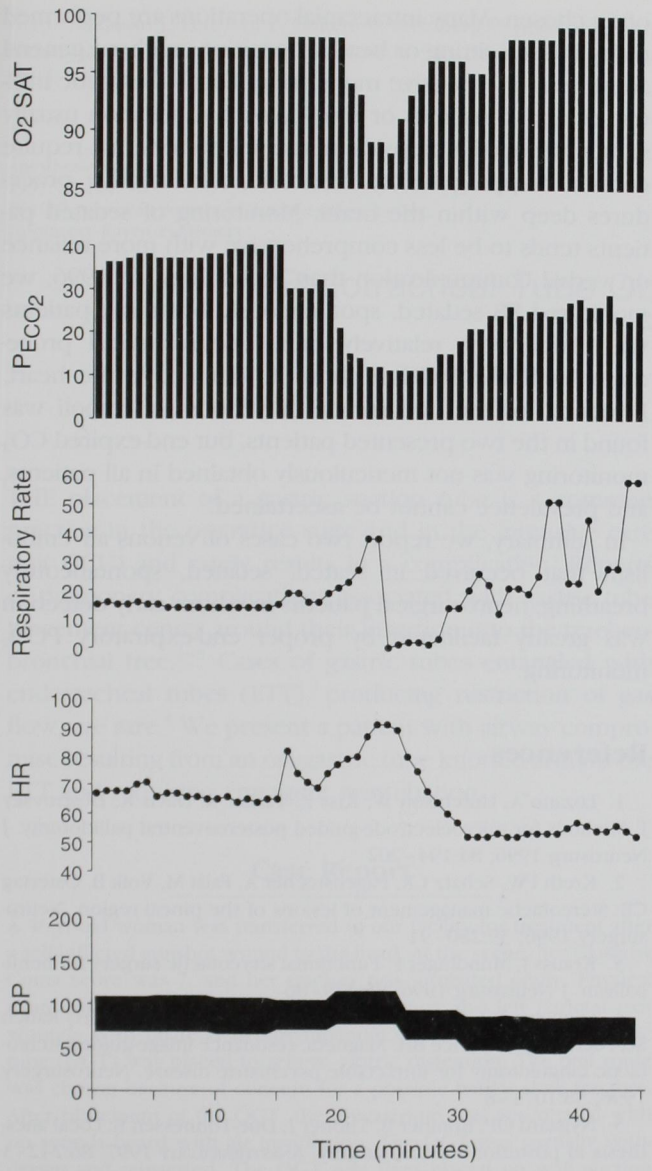

Fig. 1. Respiratory and hemodynamic data obtained during case 1.

Her breathing was quiet and regular. ECG, blood pressure, O_2 saturation, and end-expired PCO_2 were monitored as in Case 1 (fig. 2). A tiny cranial burr hole was made, and a trocar was advanced along a stereotactic trajectory. Soon after dural puncture, the patient opened her eyes and appeared startled. She claimed to be comfortable, but her end-expired PCO_2 rapidly decreased from 36 to 12 mmHg, and O_2 saturation decreased from 100% to 92%. Concurrent increases occurred in respiratory rate (from 12 to 30 breaths/min), heart rate (from 70 to 110 beats/min), and blood pressure (from 115/70 to 160/80 mmHg), and the ECG showed 1–3 mm ST segment depression in leads I, II, and V5. Chest auscultation was unremarkable, but hypotension developed. The surgical team was informed, and the trocar and wound were filled with saline. Without further therapy, the vital signs gradually improved, and the biopsy proceeded uneventfully.

Discussion

Characteristic, pathognomonic features of venous air embolization, such as altered precordial Doppler sonography, expired N_2 without recent inhalation, acute pul-

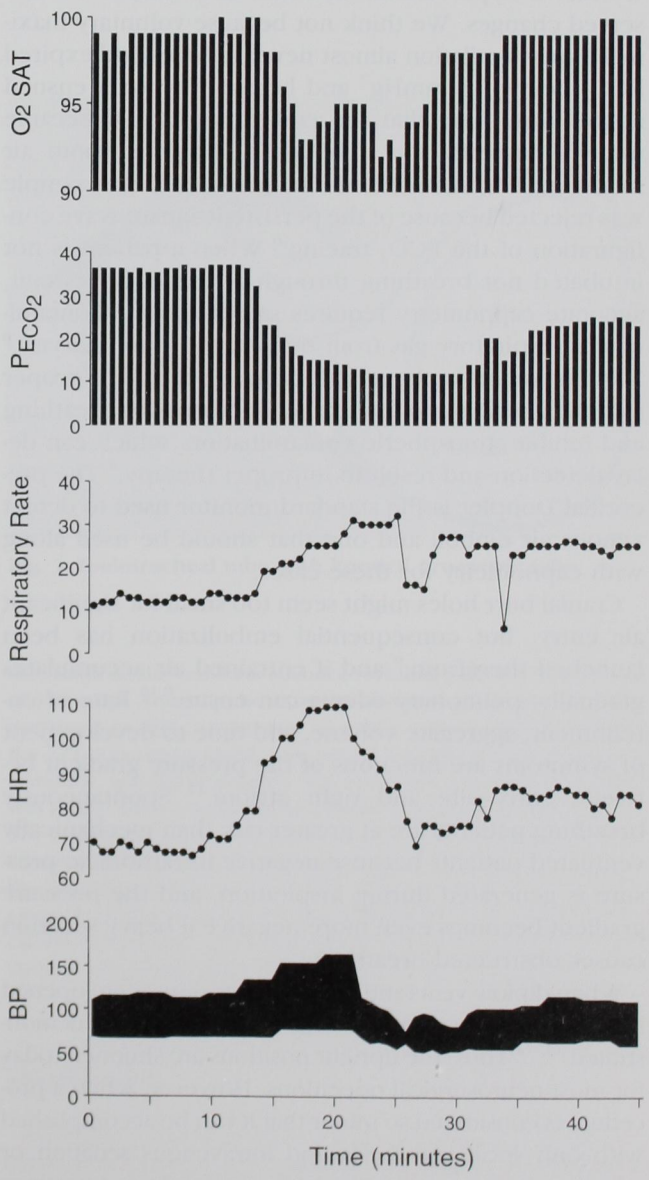

Fig. 2. Respiratory and hemodynamic data obtained during case 2.

monary artery pressure increase, echocardiographic imaging of intracardiac air, and central venous air withdrawal all require detection with special equipment that was not in use in our two cases. Diagnosis was based solely on other events that happened simultaneously soon after cerebral venous channels, located above the heart, were opened and exposed to the atmosphere: end-expired PCO_2 and O_2 saturation decreased precipitously, and abrupt changes occurred in vital signs and respiratory pattern.

Tachypnea developed in both cases, and one might wonder if hyperventilation could have caused the observed changes. We think not because voluntary maximal hyperventilation almost never reduces end-expired PCO_2 below 15 mmHg[7] and because hypoxia ensued at the same time that the end-expired PCO_2 became unphysiologically low. The possibility that room air might have contaminated the respiratory gas sample was rejected because of the persistent square wave configuration of the PCO_2 tracing.[6] When a patient is not intubated nor breathing through a respiratory circuit, accurate capnometry requires sampling of uncontaminated, respiratory gas from deep within the pharynx.[6] Nasal trumpets or large soft cannulae facilitate proper sampling of expired gas during spontaneous breathing and inhibit atmospheric contamination, which can delay detection and result in improper therapy.[8] The precordial Doppler is the standard monitor used to detect venous air emboli and one that should be used along with capnometry for these cases.

Cranial burr holes might seem too small for significant air entry, but consequential embolization has been launched therefrom,[9] and if entrained air accumulates gradually, pulmonary edema can ensue.[8,10] Rate of entrainment, aggregate volume, and time to development of symptoms are functions of the pressure gradient between entry site and right atrium.[11] Spontaneously breathing patients are at greater risk than mechanically ventilated patients because negative intrathoracic pressure is generated during inspiration, and the pressure gradient becomes even more negative if heavy sedation causes obstructed breathing.

When diploic veins and dural venous sinuses are opened in the sitting position, air emboli can often be demonstrated.[12-14] Thus, the upright positions are shunned today for most neurosurgical operations. However, when a procedure is considered so minor that it can be accomplished with only local anesthesia and intravenous sedation or when patients must respond to commands or questions during surgery, the more comfortable seated positions are often chosen. Many intracranial operations are performed today in semi-sitting or head-up positions without general anesthesia or extensive monitoring. Local anesthetic infiltration and conscious or semi-conscious sedation usually suffice for needle biopsies, minicraniotomies that require cortical mapping before resection, and ablative procedures deep within the brain. Monitoring of sedated patients tends to be less comprehensive with more reliance on verbal communication than technology. In 1996, we monitored 88 sedated, spontaneously breathing patients who underwent relatively minor neurosurgical procedures with the incision about 1 foot above the heart. Evidence for the occurrence of venous air emboli was found in the two presented patients, but end-expired CO_2 monitoring was not meticulously obtained in all patients, and prevalence cannot be ascertained.

In summary, we report two cases of venous air embolism that occurred in seated, sedated, spontaneously breathing, neurosurgical patients in whom early detection was greatly facilitated by proper end-expiratory PCO_2 monitoring.

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Anesthesiology
1997; 87:1247–8

Endotracheal Tube Obstruction after Orogastric Tube Placement

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THE placement of a gastric suction tube is a common practice in the operative suite and in the intensive care unit (ICU) and rarely results in a complication. Reports of pulmonary complications associated with gastric tube placement center around their insertion into the tracheobronchial tree.[1–3] Cases of gastric tubes entangled with endotracheal tubes (ETT), producing restriction of gas flow, are rare.[4] We present a patient with airway compromise resulting from an orogastric tube knotted around the ETT and requiring emergent reintubation.

Case Report

A 46-yr-old woman was transferred to our facility for treatment after a self-inflicted gunshot wound to the head. At the scene, her Glasgow Coma Score was 7, and her trachea was intubated orally. Physical examination revealed right parietal entrance and left parietal exit wounds with bilateral hemotympanum. In the ICU, an orogastric tube (OGT) was placed to relieve gastric distention. The oral route was chosen because of concern for a possible basilar skull fracture. After placement of the OGT, the epigastrium was auscultated with no sounds heard with air insufflation. The OGT was partially withdrawn and reinserted. The OGT was then placed on wall suction with no output noted and again, repositioned without removal. Resistance was noted whenever the OGT was withdrawn or advanced with concomitant movement of the ETT. At this time, the high pressure alarm on the ventilator sounded revealing peak airway pressures of 50 cmH_20. Arterial pulse oximeter alarmed and oxygen saturation decreased to 88%. An attempt to pass a suction catheter through the ETT was unsuccessful. The patient was disconnected from the ventilator, and bag ventilation was attempted, but no air movement was noted. While extubating the patient, the OGT also was removed. The patient was easily reintubated using direct laryngoscopy, which revealed no abnormalities of the vocal cords or laryngeal structures. She was then placed back on mechanical ventilation with normal airway pressures, and bilateral chest expansion was noted. After clinical stabilization, the ETT was inspected and found to have the OGT knotted around the distal end, thus constricting the lumen (fig. 1).

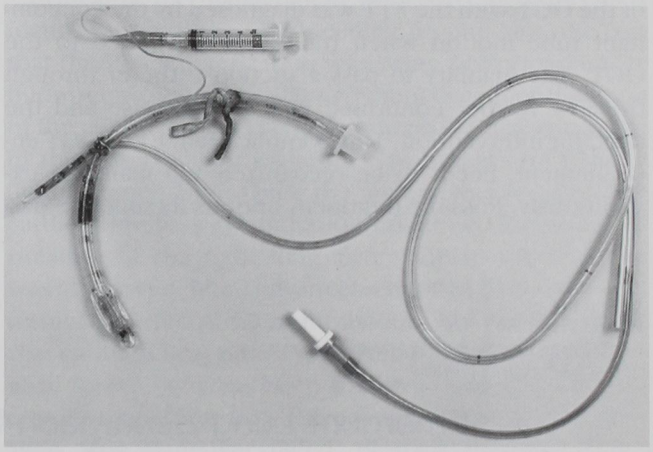

Fig. 1. Endotracheal tube with knotted orogastric tube.

Discussion

Insertion of gastric tubes into the tracheobronchial tree has been reported and may cause pulmonary complications, especially if left unrecognized. Complications include infusion of enteral feeds, with subsequent infection, direct

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Received from the Department of Anesthesiology, The University of Texas Medical School, Houston, Texas. Submitted for publication April 18, 1997. Accepted for publication June 12, 1997.

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Key words: Complications. Endotracheal tube obstruction. Orogastric tube.