Failure of Preoperative Echo Testing to Prevent Paradoxical Air Embolism: Report of Two Cases

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Much of the information about paradoxical air embolism (PAE) has been acquired because of the clinical significance of this problem in neuroanesthesia. In an attempt to reduce the risk of this complication, we and others have suggested the possible value of detection of patent foramen ovale (PFO) prior to the occurrence of venous air embolism. The role of preoperative echo is not fully defined but has been advocated because it seems to be reasonable in helping to define the population at risk for PAE.

These case reports demonstrate that preoperative and intraoperative testing that fails to detect a PFO does not assure safety from PAE.

CASE REPORTS

Case 1. The patient was a 47-yr-old white male scheduled for suboccipital craniotomy for a pineal tumor. The patient had received radiation therapy for the lesion. It had continued to increase in size with an increase in symptoms. There was no history of cardiac or pulmonary disease.

Preoperative precordial echo was performed by an experienced echocardiographer (R.A.N.). A standard two-dimensional echocardiogram was initially done, viewing the heart from multiple tomographic positions, as previously described. Special attention was made to visualize the atrial septum, which appeared intact from the parasternal and subcostal windows. Agitated indocyanine green dye was then injected into the left brachial vein, followed by a flush injection of normal saline while imaging the heart from the apical four-chamber position during spontaneous respiration. This resulted in prompt opacification of the right atrium and right ventricle from the microbubbles in the indocyanine green dye solution with absence of microbubbles in the left sided chambers. Two more injections were performed during the release of a Valsalva maneuver. There was an increase in jugular venous pressure and a reflex tachycardia during the strain phase of the Valsalva maneuver indicating that the maneuver was done properly. The injections were made during the end of the straining phase of the maneuver to assure maximal appearance of microbubbles during the release phase. There was no evidence of right to left shunting either during spontaneous respiration or following the Valsalva maneuver.

Intraoperative transesophageal echo (TEE) testing was also performed by a physician experienced with this technique (R.F.C.). A clear four-chamber view of the heart was obtained with a 3.5-MHz Diasonics esophageal echo probe mounted on a 5-mm bronchoscope body and connected to a 3400 Diasonics® echocardiographic machine. A multiorifice right atrial-superior vena caval catheter was inserted using electrocardiographic control. With the patient anesthetized with isoflurane 0.5% inspired and 50% nitrous oxide in oxygen partially paralyzed with vecuronium used for intubation, and in the upright position for surgery, 10 ml of agitated saline was injected at endexpiration without passage of the microbubbles into the left atrium. The injection was repeated twice with 20 cm H2O positive end expiratory pressure (PEEP) and once with 50 cm H2O PEEP with no evidence of left atrial microbubbles. The injection was performed just before the release of PEEP. The central venous pressure (CVP) remained at 8 mmHg just after the test until venous air embolism (VAE) occurred later.

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Thirty minutes after incision the patient suffered VAE, which continued intermittently throughout the 105 min of surgical exposure. The nitrous oxide was discontinued and $\text{F}_{1}\text{O}_2 = 1.0$. Air was aspirated from the central catheter and $\text{ETCO}_2$ decreased from 25 mmHg to 17 mmHg over 90 min. Fluid bolus of 500 ml of 5% albumin and 1,000 ml Ringer’s lactate was administered early in the problem period. After the exposure was nearly complete, the TEE was noted to show air bubbles in the left side of the heart and the aorta (Fig. 1). The CVP was 3 mmHg at that time. After brief discussion it was decided to close the wound and to complete removal of the tumor at a later day. Postoperatively, the patient had no change in his neurologic status from preoperative examination. There was no clinical evidence of paradoxical embolism. Chest x-ray immediately after surgery was unchanged and the trachea was extubated. Repeat chest x-ray on the first postoperative evening showed bilateral interstitial pulmonary infiltrates, which resolved by the second postoperative day. This was thought clinically to represent pulmonary effects of VAE. Arterial blood gases at an $\text{F}_{1}\text{O}_2$ of 1.0 showed a $\text{PaCO}_2$ of 428 mmHg intraoperatively, decreasing to 65 mmHg at an $\text{F}_{1}\text{O}_2$ of 1.0 via close fitting mask after extubation the first evening and returning to 155 mmHg the next morning at which time supplemental oxygen was decreased. He was returned to the operating room 48 h postoperatively for reoperation while in the sitting position. No air embolism was detected by Doppler or TEE and the procedure was uneventful.

Case 2. The patient was a 27-yr-old female undergoing suboccipital craniotomy for arterial-venous malformation of the posterior fossa. History and physical examination did not reveal cardiac or pulmonary problems. Preoperative echo, anesthetic agents and management, and intraoperative echo were the same as with case 1. No PFO was identified preoperatively or intraoperatively. The patient had four episodes of air embolism, the third of which occurred 1 h after incision and was associated with Doppler change, right atrial catheter air aspiration, decreased $\text{ETCO}_2$, increased $\text{ETH}_2$, and a 20 mmHg decrease in arterial pressure. Paradoxical air was noted on echo in that episode only. The source of the air was found and surgery was completed without difficulty. No sequelae occurred postoperatively.

**DISCUSSION**

Although VAE is a common occurrence during posterior craniectomy (40%) in patients in the sitting position, morbidity and mortality from VAE are rare. The problem of paradoxical air embolism producing cerebral ischemia, although rare, can be a devastating one. While the focus of paradoxical air embolism in anesthesia is on upright neurosurgical patients, symptomatic paradoxical CO$_2$ embolism is reported in patients undergoing laparoscopy, although the incidence is not known. With the advent of TEE, anesthesiologists have an instrument that enables one to make the diagnosis of PAE at a subclinical level, i.e., before air is seen in the arteries of the brain and before poor neurologic outcome is inevitable on emergence. Although the incidence of PFO
seen at autopsy is 27%, no direct comparative study of a group of echocardiographically studied patients at autopsy has been performed. The use of preoperative precordial echo has shown a detection rate of 10% to 50%. Even in the hands of an experienced operator, failure of echocardiography to demonstrate a PFO may still occur due to poor image resolution and/or the inability to create pathophysiologic conditions producing flow through the defect by temporal changes in the relative atrial pressures during the study and surgical procedure.

Since our cases and the two reported studies, color flow doppler echo has become available on TEE units. Perhaps this will enhance the rate of detection of PFO. The combined use of preoperative and intraoperative echo testing is currently being studied at our institution and others. These cases suggest that failure to detect PFO does not eliminate the possibility of PVE. The reasons for this are not clear. Probably the Valsalva maneuver (and its release) and the application of PEEP to the airway of the anesthetized patient do not adequately mimic the changes in pulmonary artery and atrial pressures that VAE produces. VAE uniquely may increase right atrial pressure as a reflection of pulmonary hypertension and decrease left atrial pressure by reducing filling pressure, thus accentuating a right to left gradient in a way that these maneuvers cannot mimic.

There are conflicting views on the effect of PEEP on the incidence of PAE in patients experiencing or at risk for VAE. The use of PEEP to increase intrathoracic pressure, CVP, and cerebral venous pressure and, therefore, decrease the risk for VAE has been advocated by some. However, other studies suggest that clinically useful levels of PEEP (10–20 mmHg) are ineffective in increasing cerebral venous pressure and decreasing the risk of VAE. The adverse hemodynamic effects of PEEP in patients in the sitting position are also a deterrent to its use. Perhaps the most controversial area concerning PEEP and air embolism is the influence of PEEP on the risk for PAE. Studies have demonstrated that PEEP may increase the incidence of right atrial pressure being greater than pulmonary capillary wedge pressure, thereby potentially increasing the risk for PAE, and right to left shunting at the atrial level has been demonstrated in some patients only with PEEP. Other studies have failed to show an effect of low levels of PEEP on the atrial pressure gradient. In a study in animals with a surgically created atrial septal defect, neither PEEP nor the gradient between the mean arterial pressures affected the incidence of PAE during venous air infusion. The important factor was the instantaneous atrial pressure gradient. If the right atrial pressure exceeded the left atrial pressure during a portion of the cardiac cycle, PAE could occur. The use of PEEP had little effect on either the gradient between the mean atrial pressures or the instantaneous atrial pressure gradient. With the discontinuation of PEEP, however, there was a transient but marked shift in the atrial pressure gradients such that the right atrial pressure exceeded the left atrial pressure for a much greater portion of the cardiac cycle than during PEEP. At the same time a surge of right to left passage of air was noted. These changes lasted several cardiac cycles. This is consistent with the observed augmentation of right to left shunting with the release phase of the Valsalva maneuver. A possible mechanism is that increased intrathoracic pressure impedes venous return and that with the release of this pressure venous return to the heart increases, acutely increasing right heart pressures at a time when left heart filling remains lower. This resolves within a few cardiac cycles. During the release phase of the Valsalva maneuver increases in right atrial pressure preceding increases in pulmonary capillary wedge pressure by several seconds have been demonstrated. It seems that the use of PEEP is not associated with increased risk of PAE, but the discontinuation of PEEP during VAE may be. Because of this, as well as the ineffectiveness of PEEP in decreasing risk for VAE and the adverse hemodynamic effects of PEEP, the use of PEEP in patients in the sitting position may be inadvisable unless necessary for adequate ventilation or oxygenation.

Whether significant volumes of venous air can cross through the pulmonary vasculature to cause PAE remains controversial. However, a case of known cerebral air embolism in a patient with no intracardiac defects at autopsy has been reported. This suggests that significant PAE via the pulmonary vasculature is possible but rare.

In patients in the sitting position undergoing posterior fossa craniotomy procedures the risk of venous air embolism is about 40%. Perhaps those patients with a known PFO should have their operation performed while in a horizontal surgical position where the incidence of VAE is known to be lower (12%). The cases reported here suggest that negative data from preoperative echo does not eliminate the risk of PAE. The comparative value of preoperative echo to intraoperative TEE monitoring in reducing clinical outcomes from PAE remains to be defined.

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Anesthesia for Open Lung Biopsy in a Patient with Intracardiac Tumor

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Although there are reports of anesthetic management of patients with intracardiac tumors, 1,2 to our knowledge there has been no previous description of anesthetic management of a patient with an intracardiac tumor undergoing noncardiac surgery. We report a case of a patient with an intracardiac tumor who underwent open lung biopsy.

REPORT OF A CASE

A 23-year-old male farmer presented with a 6-month history of fever, night sweats, progressive dyspnea on exertion, pleuritic chest pain, anorexia, and weight loss. One week prior to admission, hemoptysis developed. A chest x-ray showed multiple nodular lesions.

On admission he was not dyspneic at rest or lying flat, but his exercise tolerance was limited to climbing six steps.

On physical examination the patient was afebrile and in no acute distress. The heart rate was 120 beats/min and regular with a blood pressure of 120/90 mm Hg. The respiratory rate was 32 breaths/min. The weight was 70 kg. There was decreased air entry at the left base and fine rales were heard over the right middle lobe. The jugular venous pulse appeared normal with no hepatomegaly or reflex. The apex was hyperdynamic with a palpable systolic thrill. The heart sounds were normal, but there was a 4 of 6 systolic ejection murmur at the left sternal border, which increased on inspiration. The rest of the physical examination was unremarkable.

The ECG revealed sinus tachycardia with right bundle branch block. The chest x-ray showed multiple nodular lesions throughout both lung fields (fig. 1) with suggestion of right ventricular enlargement. A two-dimensional echocardiogram (fig. 2) showed a large mass almost completely filling the right atrium and extending through the tricuspid valve into the right ventricle, occupying a large portion of the ventricle and narrowing the right ventricular outflow tract. Tricuspid valve leaflets were not seen. The pulmonary valve was normal. There was a second small mass posterior to the left ventricle, but the left atrium and left ventricle appeared normal, both anatomically and functionally. There was no pericardial fluid. CT scan (fig. 3) confirmed a large...