blocks: A biochemical and electron microscopic study. Anesth Analg 62:796–801, 1983

41. Newman RJ, Radda GK: The myotoxicity of bupivacaine, a ^{31}P n.m.r. investigation. Br J Pharmacol 79:395–399, 1983

42. Loghmanee F, Tobak M: Fatal malignant hyperthermia associated with recreational cocaine and ethanol abuse. Am J Forensic Med Pathol 7:246–248, 1986

43. Merigan KS, Roberts JR: Cocaine intoxication: Hyperpyrexia, rhabdomyolysis and acute renal failure. Clin Toxicol 25:135–148, 1987

44. Merlin SI, Brissie RM, Kapila A, Hauser T: Hyperpyrexia, seizures and hypotension in a 31-year old man. Alabama J Med Sci 25: 274–279, 1988

45. Warrian WG, Halikas JA, Crosby RD, Carlson GA, Crea F: Observations on increased CPK levels in "asymptomatic" cocaine abusers. Cocaine: Physiological and Pathophysiological Effects. Binghamton, Haworth, 1992, pp 83–95. NIDA monographs

46. Danko I, Fritz JD, Jiao S, Hogan K, Latendresse JS, Wolff JA: Pharmacological enhancement of *in vivo* foreign gene expression in muscle. Gene Ther (in press)

47. Parris WCV, Dettbarn WD: Muscle atrophy following bupivacaine trigger point injection. Anesth Rev 16:50–53, 1989

48. Rainin EA, Carlson BM: Postoperative diplopia and ptosis: A clinical hypothesis based on the myotoxicity of local anesthetics. Arch Ophthalmol 103:1337–1339, 1985

Anesthesiology
80:947–950, 1994

Paradoxic Air Embolism during Venous Air Embolism: Transesophageal Echocardiographic Evidence of Transpulmonary Air Passage

Eric A. Bedell, M.D., Keith H. Berge, M.D.,† Thomas J. Losasso, M.D.†*

PARADOXIC air embolism (PAE) can occur during venous air embolism (VAE) and may have devastating consequences.[1,2] Although intracardiac right-to-left shunting has been the proposed mechanism of PAE in the majority of cases, a number of case reports have documented PAE in which no cardiac defects could be detected either by intraoperative transesophageal echocardiography (TEE)[3] or at autopsy.[4–6] In the cases reported by Cucchiara *et al.*,[3] it is suggested that PAE could have occurred either through an undetected intracardiac defect, specifically a patient foramen ovale, or by transpulmonary air passage. Although PAE *via* pulmonary shunts have been identified in dogs,[7–10] this mechanism of PAE has not been clearly demonstrated in humans. We present a case of TEE-detected PAE in a patient without a demonstrable intracardiac right-to-left shunt in whom there was direct evidence of transpulmonary PAE using intraoperative TEE.

* Resident in Neuroanesthesia, Mayo Graduate School of Medicine.

† Assistant Professor of Anesthesiology, Mayo Medical School.

Received from the Department of Anesthesiology, Mayo Clinic, Rochester, Minnesota. Accepted for publication November 30, 1993.

Address reprint requests to Dr. Berge: Department of Anesthesiology, Mayo Clinic, 200 First Street, Southwest, Rochester, Minnesota 55905.

Key words: Anesthesia: neurosurgical. Complications: embolism. Embolism: air. Equipment: echocardiography.

Case Report

A 65-yr-old man was scheduled for an occipital artery to posterior inferior cerebellar artery bypass *via* suboccipital craniectomy for unstable brain stem ischemia and impending infarction. The patient had a 1-yr history of progressive imbalance, ataxia and speech disturbances secondary to brainstem ischemia. He had suffered acute focal deficits 6 weeks preoperatively with evidence, by magnetic resonance imaging, of a cerebellar infarction, and a second infarction 1 week preoperatively with worsening dysarthria and new cranial nerve (VII, IX, and XII) deficits. Other pertinent medical problems included a history of mild chronic obstructive lung disease and an inferior wall myocardial infarction suffered 6 yr earlier. The patient had no known drug allergies and was medicated with Carafate (sucralfate) and heparin. Cerebral arteriogram demonstrated patent carotid arteries, a completely occluded left vertebral artery, 99% occlusion of the right vertebral artery and brainstem blood flow *via* meningeal collaterals. Electrocardiogram demonstrated normal sinus rhythm and findings consistent with an old inferior wall infarction. Pulmonary function studies demonstrated mild obstructive lung disease with moderate loss of diffusing capacity (forced vital capacity

110%, forced expiratory volume in 1 s 87%, the ratio of forced expiratory volume in 1 s to forced vital capacity 60%, and diffusing capacity of the lung for carbon monoxide 48% of predicted). Preoperative blood pressure ranged between 130/70 and 175/95 mmHg.

The patient was brought to the operating room and general anesthesia was induced with sodium thiopental and fentanyl. Anesthesia was maintained with isoflurane and nitrous oxide in oxygen. Muscle relaxation was achieved and maintained with vecuronium. Intraoperative monitors included electrocardiogram, pulse oximetry, mass spectrometry, intraarterial blood pressure monitoring by an indwelling radial artery catheter, pulmonary artery catheter *via* basilic vein cannulation, precordial Doppler, two-dimensional TEE, electroencephalography, somatosensory evoked potentials, and brainstem auditory evoked response. Initial hemodynamic assessment after induction of anesthesia showed a cardiac index of 1.9 $l \cdot min^{-1} \cdot m^{-2}$, pulmonary artery pressure of 28/9 mmHg and pulmonary capillary wedge pressure of 6 mmHg. After both prone and lateral positioning, the surgeon decided that having the patient in the sitting position would permit optimal surgical exposure.

As described elsewhere, two-dimensional contrast TEE with positive end-expiratory pressure (PEEP)-release testing was performed before placing the patient in the sitting position to evaluate for the presence or absence of a potential right-to-left interatrial shunt.[11-14] Three separate injections of agitated saline were made into the right atrium *via* the central venous port of the pulmonary artery catheter during PEEP-release testing. During each injection of agitated saline, transient complete atrial opacification was achieved. Additionally, right-to-left interatrial septal bulging was observed on the TEE during PEEP-release, signifying a transient period during which right atrial pressure exceeded left atrial pressure. These injections showed no evidence of interatrial right-to-left shunting. The patient was placed in the sitting position and three injections of agitated saline were made during PEEP-release testing as well as during intermittent positive-pressure ventilation without PEEP. Again there was no evidence of an interatrial right-to-left shunt.

Throughout the case, blood pressure was maintained at normal values with phenylephrine as needed and dopamine infusion utilized to increase the cardiac index, achieving flows between 2.5 and 3.5 $l \cdot min^{-1} \cdot m^{-2}$. During occlusion of the posterior inferior cerebellar artery, a single episode of hemodynamically insignificant VAE was detected by precordial Doppler and TEE. Administration of nitrous oxide was discontinued for the remainder of the surgical procedure. The air entrainment spontaneously resolved in less than 30 s. During dural closure, a second episode of VAE was detected by TEE and precordial Doppler. Associated acute changes included a decrease in end-tidal carbon dioxide tension from 32 to 16 mmHg, decrease in hemoglobin oxygen saturation from 99% to 80% by pulse oximetry, a decrease in blood pressure from 170/85 to 80/35 mmHg, a decrease in cardiac index from 3.4 to 2.1 $l \cdot min^{-1} \cdot m^{-2}$, and an increase in pulmonary artery pressure from 28/7 to 73/53 mmHg. Aspiration of the proximal port of the pulmonary artery catheter yielded no air. Aspiration from the distal port of the pulmonary artery catheter yielded 50 ml of air followed by withdrawal of frothy blood. After the removal of an estimated 100 ml of air and administration of 100 μg of epinephrine intravenously, the hemodynamic parameters returned to pre-VAE values. PAE became evident by TEE during removal of air from the pulmonary artery, with air emboli evident in the left atrium and left ventricle. After the initial episode of massive VAE, a slow but steady entrainment of venous air continued. This was not associated with hemodynamic compromise and VAE ceased once expeditious closure had been completed and the patient was returned to the supine position. With the patient in the supine position, TEE demonstrated continued air traversing the left atrium, left ventricle and exiting *via* the aortic valve for approximately 15 min after venous air entrainment ceased. During this time there was no evidence of air emboli within the right atrium or right ventricle, suggesting that the origin of the left heart air entrainment was from the pulmonary vasculature.

Before removal of the TEE probe at the completion of the case, agitated saline injection during PEEP-release testing (a total of five injections) again showed no evidence of intracardiac right-to-left shunting. When the patient did not awaken promptly at the end of the case, a computed tomographic scan of the head was obtained. This showed no evidence of gross intravascular air, but did demonstrate a large bifrontal pneumocephalus. The diagnosis of tension pneumocephalus was confirmed after intracranial air was released using a twist-drill burr hole and an 18-G needle to penetrate the dura over the frontal region. The patient's level of consciousness rapidly improved. Given the patient's rapid improvement in level of consciousness without evidence of focal neurologic deficit, hyperbaric oxygen therapy was felt to be unnecessary. A transthoracic two-dimensional color flow echocardiographic examination in the intensive care unit on the 1st postoperative day revealed no evidence of intracardiac right-to-left shunting, although no provocative maneuvers were utilized. On the 4th postoperative day, the patient returned to the operating room for wound revision and repair of a CSF leak under general anesthesia. Two-dimensional contrast TEE with agitated saline and PEEP-release testing (a total of four injections) again showed no right-to-left interatrial shunting. The patient tolerated the anesthesia well and the trachea was extubated at the completion of the procedure.

The patient's condition improved steadily after the wound revision and he demonstrated no new neurologic deficits. The patient had resolution of his brain stem ischemic symptoms and was discharged from the hospital on the 56th hospital day.

Discussion

VAE is a recognized risk associated with the use of the sitting position.[15] PAE during VAE is a rare occurrence but can result in serious morbidity and mortality. At the current time, PAE can be diagnosed specifically using TEE. However, during most clinical episodes of VAE, this monitoring modality does not allow one to discriminate between right-to-left interatrial shunting and transpulmonary passage of air emboli. Until the early 1980s, it was presumed that PAE resulted from intracardiac right-to-left shunting. In patients with an intact interatrial and interventricular septum, the most common mechanism for right-to-left shunting should be through a patent foramen ovale. Marquez *et al.*[4] reported a perioperative fatality associated with intraoperative VAE and PAE in a patient with no evidence of an intracardiac defect at autopsy. Subsequent cases

were described in which PAE occurred in patients without TEE[3] or autopsy[4–6] evidence of an intracardiac defect. TEE with PEEP-release testing may not detect a potential right-to-left interatrial shunt when one in fact exists (*i.e.*, a false-negative PEEP test). However, when an intracardiac defect is ruled out by autopsy, as described by Marquez, PAE can only result from transpulmonary passage of air. Existence of arterial-to-venous anastomoses (*Sperrarteries*) in the lung were theorized approximately 50 yr ago.[16,17] Subsequent canine studies have repeatedly demonstrated the possibility of PAE during VAE by transpulmonary air passage.[7–10] To date, real-time observation of transpulmonary air transport from the venous to arterial circulation during VAE has been lacking in humans.

In the case described here, the patient experienced an episode of VAE with associated PAE detected by TEE. We believe that PAE resulted from transpulmonary passage of air. No potential right-to-left interatrial shunting was demonstrable by TEE despite multiple injections of agitated saline with PEEP-release testing before the surgical procedure, after resolution of the episode of massive VAE, and during a subsequent surgical procedure. Although false-negative testing (as defined above) is a possibility, we feel this explanation to be unlikely. We performed more than 15 trials of agitated saline injection with PEEP-release and consistently observed evidence of right atrial pressure exceeding the left atrial pressure (as indicated by right-to-left bulging of the interatrial septum) during PEEP-release. Yet, there was no right-to-left interatrial shunting demonstrable during any test injection. More convincingly, we observed the continuation of air emboli traversing the left-sided cardiac chambers for 15 min after the cessation of venous air entrainment and clearing of the right atrium and ventricle of any air. This strongly suggests that residual air remaining in and traversing the pulmonary vasculature was the source of the left-sided air.

The lungs' ability to inhibit transpulmonary air passage has been quantified in the canine model and demonstrates a finite limit to filtering ability. At infusion rates of 0.3 $ml \cdot kg^{-1} \cdot min^{-1}$, VAE in the canine model is tolerated without PAE, whereas at 0.4 $ml \cdot kg^{-1} \cdot min^{-1}$, 71% of animals demonstrated PAE.[9] The protective filtering properties of the human lung against PAE during episodes of VAE appear to be substantial; however, our case suggests that overloading these protective mechanisms can result in transpulmonary PAE. Perhaps pathways involved in transpulmonary air transport (*i.e.*, anatomic shunts) become functionally open only during episodes of VAE in which significant elevations of pulmonary artery pressure occur. Such an increase in pulmonary artery pressure was observed in this case. The potential contribution of the patient's chronic obstructive pulmonary disease to the transpulmonary PAE is indeterminate. It is clearly documented that morbidity and mortality can result from PAE in patients with no identifiable right-to-left intracardiac shunt.[4–6] The clinically used forms of TEE reveal the presence, but not the volume, of air emboli nor does this monitoring modality indicate the ultimate destination of arterial air emboli. Although TEE-detected PAE can be limited to a subclinical event when the source of air entrainment is promptly identified,[3] it is not clear what volume of PAE can safely be considered insignificant. In order to prevent serious morbidity and mortality from PAE, the occurrence of PAE, regardless of the actual pathway involved, must be considered a serious intraoperative event. Therefore, at the current time, the clinician is obligated to assume that any amount of PAE places the patient at risk for serious morbidity or mortality and promptly initiate aggressive measures to cease further air entrainment.

To our knowledge, this is the first case report of PAE for which there was strong intraoperative TEE evidence of transpulmonary air passage during an episode of VAE. TEE remains the only intraoperative monitor capable of detecting PAE. We use intraoperative TEE monitoring for PAE when the risk of VAE is significant.

References

1. Gronert GA, Messick JM, Cucchiara RF, Michenfelder JD: Paradoxic air embolism from a patent foramen ovale. ANESTHESIOLOGY 50:548–549, 1979

2. Lechat PH, Mas JL, Lascault G, Loron PH, Theard M, Klimczac M, Drobinski G, Thomas D, Grosgogeat Y: Prevalence of patent foramen ovale in patients with stroke. N Engl J Med 318:1148–1152, 1988

3. Cucchiara RF, Nishimura RA, Black S: Failure of preoperative echo testing to prevent paradoxic air embolism: Report of two cases. ANESTHESIOLOGY 71:604–607, 1989

4. Marquez J, Sladen A, Gendell H, Boehnke M, Mendelow H: Paradoxical cerebral air embolism without an intracardiac septal defect. J Neurosurg 55:997–1000, 1981

5. Ahmat KP, Riley RH, Sims C, House AK, Cooke OT: Fatal air embolism following anesthesia for insertion of a peritoneovenous shunt. ANESTHESIOLOGY 70:702–704, 1989

6. Albin MS, Ritter RR, Pruett CE, Kalff K: Venous air embolism during lumbar laminectomy in the prone position: Report of three cases. Anesth Analg 73:346–349, 1991

7. Yahagi N, Furuya H: The effects of halothane and pentobarbital on the threshold of transpulmonary passage of venous air emboli in dogs. ANESTHESIOLOGY 47:905–909, 1987

8. Prinzmetal M, Ornitz EM, Simkin B, Berman HC: Arterio-venous anastomoses in liver, spleen and lungs. Am J Physiol 152:48–52, 1948

9. Butler BD, Hills BA: Transpulmonary passage of venous air emboli. J Appl Physiol 59:543–547, 1985

10. Yahagi N, Furuya H, Sai Y, Amakata Y: Effect of halothane, fentanyl and ketamine on the threshold for transpulmonary passage of venous air emboli in dogs. Anesth Analg 75:720–723, 1992

11. Cucchiara RF, Nugent M, Seward JB, Messick JM: Air embolism in upright neurosurgical patients: Detection and localization by two-dimensional transesophageal echocardiography. ANESTHESIOLOGY 60: 353–355, 1984

12. Cucchiara RF, Seward JB, Nishimura RA, Nugent M, Faust RJ: Identification of patent foramen ovale during sitting position craniotomy by transesophageal echocardiography with positive airway pressure. ANESTHESIOLOGY 63:107–109, 1985

13. Black S, Muzzi DA, Nishimura RA, Cucchiara RF: Preoperative and intraoperative echocardiography to detect right-to-left shunt in patients undergoing neurosurgical procedures in the sitting position. ANESTHESIOLOGY 72:436–438, 1990

14. Konstadt SN, Louie EK, Black S, Rao TLK, Scanlon P: Intraoperative detection of patent foramen ovale by transesophageal echocardiography. ANESTHESIOLOGY 74:212–216, 1991

15. Losasso TJ, Muzzi DA, Dietz NM, Cucchiara RF: Fifty percent nitrous oxide does not increase the risk of venous air embolism in neurosurgical patients operated upon in the sitting position. ANESTHESIOLOGY 77:21–70, 1992

16. von Hayek H: Kurz- und nebenschlüsse des menschlichen lungenkreislaufes in der pleura. Z Anat Entwicklungsgeschichte 112: 221–228, 1942

17. Weibel E: Die blutgefäßanastomosen in der menschlichen lunge. Z Zellforsch Mikroskip Anat Abt Histochem 50:653–692, 1959

Anesthesiology
80:950–952, 1994

Repeated Stellate Ganglion Blockade Using a Catheter for Pediatric Herpes Zoster Ophthalmicus

Mazin Elias, M.B., Ch.B., F.C.Anae., Maia U. Chakerian, M.D.†*

TREATMENT for the pain associated with acute herpes zoster traditionally has included sympathetic blockade.[1,2] In many cases, a series of blocks may need to be performed over a short period of time. The performance of nerve blocks in children is more complex than that in adults, because it may be impossible to attain the required level of cooperation from the patient without concurrent administration of general anesthesia. We report on our experience with a pediatric patient with acquired immune deficiency syndrome (AIDS) who had acute herpes zoster ophthalmicus.

* Pain Fellow.

† Assistant Clinical Professor.

Received from the Department of Anesthesiology, University of California at San Diego Medical Center, University of California at San Diego, San Diego, California. Accepted for publication December 3, 1993.

Address reprint requests to Dr. Chakerian: Department of Anesthesiology, University of California at San Diego Medical Center, University of California at San Diego, San Diego, California 92103-8812.

Key words: Catheter. Herpes zoster ophthalmicus. Pediatric. Stellate ganglion blockade.

Case Report

A 24 kg, 8-yr-old boy with a history of human immunodeficiency virus (HIV) infection acquired perinatally, was admitted to the Pediatric Service. He had a 2-week history of acute herpes zoster of the ophthalmic division of the right trigeminal nerve and complained of severe, constant, burning facial pain and itching. Treatment was started with acyclovir (200 mg) intravenously three times a day, codeine phosphate (30 mg) and acetaminophen (300 mg) (Tylenol #3, McNeil Pharmaceutical, Raritan, NJ) orally every 4 h or as needed for pain), and morphine sulfate (1 mg) intravenously every hour or as needed for severe pain. Failure of these measures to control his pain resulted in consultation of the Anesthesia Pain Service.

On examination, the patient appeared anxious, in pain and tearful, with a pulse of 120 beats per min and a respiratory rate of 24 breaths per min. A maculopapular rash with crusted lesions covered the right side of his face. He had conjunctivitis and cervical lymphadenopathy. There was allodynia and hyperesthesia of the affected area. The rest of his physical examination yielded no abnormal results.