JOURNAL OF NEUROSURGERY:
Case Lessons

**J Neurosurg Case Lessons** 5(12): CASE2342, 2023
DOI: 10.3171/CASE2342

# Intraoperative air embolism diagnosis and treatment using hyperbaric oxygen therapy after craniotomy: illustrative case

Armaan K. Malhotra, MD,[1] Ashton P. Chang, MBBS,[2] Joseph P. Lawton, BSc,[3] Aderaldo Costa Alves Jr., MD, PhD,[4] Angela Jerath, MD,[2,5] Bourke W. Tillmann, MD,[6,7] Harry Foster, MD,[2,5] Azad Mashari, MD,[8] Leodante da Costa, MD,[4] and Ashish Kumar, MD, MCh[4]

[1]Division of Neurosurgery, Department of Surgery, University of Toronto, Toronto, Ontario, Canada; [2]Department of Anesthesiology, Sunnybrook Health Sciences, Toronto, Ontario, Canada; [3]Department of Health Sciences, McMaster University, Hamilton, Ontario, Canada; [4]Division of Neurosurgery, Department of Surgery, Sunnybrook Health SciencesCentre, Toronto, Ontario, Canada; [5]Department of Anesthesiology, Schulich Heart Program, Sunnybrook Research Institute, Sunnybrook Health Sciences Centre, Toronto, Ontario, Canada; [6]Division of Critical Care Medicine, Sunnybrook Health Sciences Centre, Toronto, Ontario, Canada; [7]Interdeparmtental Division of Critical Care, University of Toronto, Toronto, Ontario, Canada; and [8]Division of Anesthesiology, University Health Network, Toronto, Ontario, Canada

**BACKGROUND** This report describes the use of hyperbaric oxygen therapy for the acute management of an intraoperative air embolism encountered during a neurosurgical procedure. Furthermore, the authors highlight the concomitant diagnosis of tension pneumocephalus requiring evacuation prior to hyperbaric therapy.

**OBSERVATIONS** A 68-year-old male developed acute ST-segment elevation and hypotension during elective disconnection of a posterior fossa dural arteriovenous fistula. The semi-sitting position had been used to minimize cerebellar retraction, raising the concern for acute air embolism. Intraoperative transesophageal echocardiography was utilized to establish the diagnosis of air embolism. The patient was stabilized on vasopressor therapy, and immediate postoperative computed tomography revealed air bubbles in the left atrium along with tension pneumocephalus. He underwent urgent evacuation for the tension pneumocephalus followed by hyperbaric oxygen therapy to manage the hemodynamically significant air embolism. The patient was eventually extubated and went on to fully recover; a delayed angiogram revealed complete cure of the dural arteriovenous fistula.

**LESSONS** Hyperbaric oxygen therapy should be considered for an intracardiac air embolism resulting in hemodynamic instability. In the postoperative neurosurgical setting, care should be taken to exclude pneumocephalus requiring operative intervention prior to hyperbaric therapy. A multidisciplinary management approach facilitated expeditious diagnosis and management for the patient.

https://thejns.org/doi/abs/10.3171/CASE2342

**KEYWORDS** air embolism; neurosurgery; hyperbaric oxygen therapy; complication; pneumocephalus

Vascular air embolism (VAE) is a rare and potentially life-threatening intraoperative complication that requires prompt recognition and management.[1–3] There is a wide range of incidences reported for intraoperative neurosurgical VAE, with cumulative estimates up to 76% described in different series; however, reported rates of symptomatic VAE range between 0% and 14%.[1–15] Both surgical and patient factors have been described to contribute VAE, including abdominal or pelvic air insufflation, ventilation of patients with cavitary lung lesions, and most classically, neurosurgical procedures in the sitting position.[4–8] Although the evidence is controversial, potential benefits of the semi-sitting and sitting positions include reduced intraoperative cerebellar retraction and improved bimanual dissection capability, particularly for the infratentorial supracerebellar corridor.[9,10] Due to negative intravascular pressure associated with the upright position, there exists the potential for air to entrain into the vasculature through violated diploic venous channels, emissaries, or venous sinuses, making neurosurgical procedures particularly prone to this complication.[11,12]

**ABBREVIATIONS** ATA = atmospheres absolute; CT = computed tomography; dAVF = dural arteriovenous fistula; FSW = feet of sea water; ICU = intensive care unit; RVOT = right ventricular outflow tract; SSEP = somatosensory evoked potential; TEE = transesophageal echocardiogram; VAE = vascular air embolism.

**INCLUDE WHEN CITING** Published March 20, 2023; DOI: 10.3171/CASE2342.
**SUBMITTED** January 21, 2023. **ACCEPTED** February 24, 2023.

The morbidity of VAE depends on both the volume and rate of air entrained as well as the presence of intracardiac shunting (i.e., patent foramen ovale).[2] Previous reports estimate that severe and hemodynamically unstable VAEs require air entry volumes of approximately 200–300 mL (3–5 mL/kg).[13] The treatment of VAE requires interdisciplinary collaborative management among surgical, anesthesia, and critical care teams. Furthermore, given the relative rarity of severe VAE, the reporting of cases with successful management remains a priority. Herein, we describe a case of severe intraoperative VAE with hemodynamic compromise during a semi-sitting supracerebellar infratentorial craniotomy in addition to a concomitant tension pneumocephalus diagnosis managed successfully using prompt interdisciplinary collaboration.

## Illustrative Case

A 68-year-old male presented to the emergency department with 2 discrete episodes of transient right upper-extremity weakness with hand paresthesias and numbness. These presentations occurred within 48 hours of each other, and in both instances, the symptoms resolved within a few minutes. Other than mild bifrontal headache, there was no associated prodrome, thunderclap event, aura, or other symptoms. He was on daily aspirin for primary coronary artery disease prevention, and his medical history was significant for alcohol use, remote migraines, dyslipidemia, hypertension, and smoking. He was neurologically intact without focal deficits. Neuroimaging revealed a left posterior frontal convexity subarachnoid hemorrhage with suspicion for infratentorial dural arteriovenous fistula (dAVF) (Fig. 1A). He was broadly investigated for nonstructural subarachnoid hemorrhage etiologies, and his rheumatological, infectious, and inflammatory workups were negative. In the hospital his transient right-sided symptoms continued, and antiepileptic therapy was initiated. Magnetic resonance imaging demonstrated a nonocclusive sinus thrombosis at the torcula, and right vertebral artery injection digital subtraction angiography demonstrated a tentorial vascular lesion with supply from the posterior meningeal and superior cerebellar arteries and prominent cerebellar cortical venous reflux consistent with a Borden type III dAVF (Fig. 1B). Given the remote location, it was difficult to ascertain with complete confidence the rupture status of the lesion. The patient was discharged home after a period of observation in stable neurological condition.

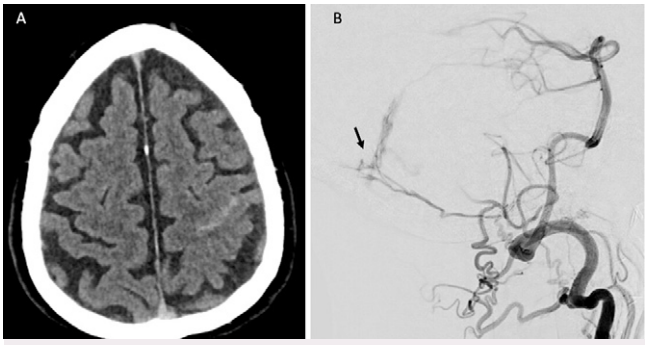


**FIG. 1. A:** Axial CT image demonstrating left posterior frontal convexity acute subarachnoid hemorrhage. **B:** Lateral right vertebral artery injection digital subtraction angiogram depicting a Borden type III dAVF (*black arrow*).

The patient's case was discussed in a vascular conference. Given the superficial location and Borden III dAVF morphology, the decision was made to proceed with elective surgical treatment 4 weeks after his initial presentation. The patient was brought to the operating room in stable condition with no ongoing symptoms. After inducing general anesthesia with placement of an intra-arterial line, a midline suboccipital craniotomy was performed with the patient in the semi-sitting Fowler position (knees slightly flexed, head approximately 45–60° elevated) to minimize cerebellar retraction. Adjunctive navigation and somatosensory evoked potentials (SSEPs) were utilized. Care was taken during positioning and exposure to fully coagulate any dural vessels and avoid sinus injury. Visualization, disconnection, and confirmation of fistula occlusion with intraoperative indocyanine green angiography occurred in a routine fashion.

Subsequently, there were slight ST-segment electrocardiogram elevations without accompanying physiological changes in oxygen requirement or arterial carbon dioxide tension ($PaCO_2$) readings. During closure, the anesthesia team noted acidosis, hypoxia, and elevated end-tidal $CO_2$, and concurrently the SSEPs in the left leg were reduced by 50%. Given the acute deterioration, an air embolism was suspected. The operative field was flooded with irrigation, closure was completed expeditiously, and the intensive care unit (ICU) team was called for an intraoperative consultation. Simultaneously, the anesthesia team obtained a transesophageal echocardiogram (TEE), which demonstrated a large volume of left ventricular air without air in the right heart (Fig. 2, Video 1). Air was visualized entering the left atrium from the pulmonary venous circulation for several minutes, and no intracardiac shunts or clots were observed. The operating table and head of the bed were placed in the Trendelenburg position to direct air toward the feet. The patient was also positioned in the right lateral decubitus position in an attempt to trap air within the apex of the left ventricle (air was already present on echo in the left heart). A discussion was held regarding the role of hyperbaric therapy. Prior to initiation of the hyperbaric therapy, it was deemed necessary to rule out any immediate postoperative neurosurgical complications explaining the reduction in SSEPs and to ensure there was no ongoing source of air leak requiring immediate intervention.

**VIDEO 1.** Clip showing the intraoperative view of the left ventricle revealing high burden of intracardiac air bubbles. Click here to view.

The patient was supported with vasoactive medications and taken for immediate computed tomography (CT) imaging of the

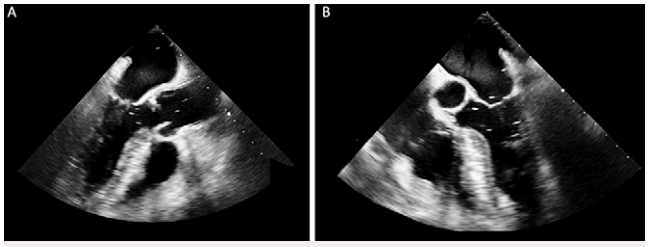


**FIG. 2.** Two-dimensional intraoperative transesophageal echocardiography, long-axis (**A**) and 4-chamber (**B**), demonstrating intracardiac air bubbles in the left atrium and the left ventricle exiting into the aorta with no air seen in the right atrium and ventricle.

head and chest. Imaging was negative for hemorrhage or stroke but concerning for tension pneumocephalus (Fig. 3A). Importantly, the tension pneumocephalus was considered a distinct complication of surgery, not the etiology of the VAE; the pneumocephalus was a concern for anticipated hyperbaric therapy given the potential for re-expansion following treatment. The patient was taken for emergent burr hole evacuation to treat the pneumocephalus. In discussion with the regional hyperbaric team, it was determined that, given the large volume of intracardiac air and vasopressor requirements, the patient would likely benefit from hyperbaric therapy, with a goal of initiating therapy within 6 hours of diagnosis. Upon completion of the surgical evacuation, repeat CT showed improvement in the pneumocephalus, at which point the patient was transferred to the regional hyperbaric center. After the placement of bilateral myringotomy tubes, the patient was treated in a multi-place hyperbaric chamber, according to US Navy Table 6 treatment (Fig. 4) with a maximum pressure of 2.8 atmospheres absolute (ATA) for a total dive time of 4 hours, 55 minutes. Table 6 treatment course proceeded as follows: 10-minute compression on air to 2.8 ATA (60 feet of sea water [FSW]) for 75 minutes, consisting of three 20-minute oxygen intervals separated by three 5-minute air breaks. This was followed by a 30-minute decompression to 1.9 ATA (30 FSW, on oxygen) for 150 minutes consisting of six 5-minute air breaks separating six 20-minute oxygen intervals and a final decompression over 30 minutes on oxygen. Total oxygen exposure time was 3 hours, 30 minutes.

Delayed postoperative CT demonstrated improvement in the pneumocephalus (Fig. 3B), and follow-up echocardiography normalized. The patient was briskly obeying commands on postoperative day 1 with no lateralizing motor weakness and was extubated. At follow-up, the patient was neurologically intact without residual deficits and independent, and his delayed angiogram demonstrated complete fistula disconnection (Fig. 3C).

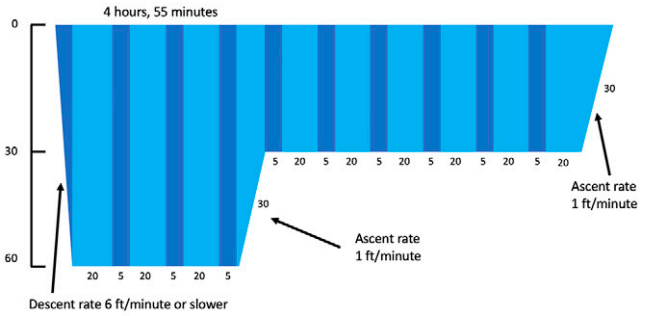


**FIG. 4.** US Navy treatment Table 6 hyperbaric oxygen therapy protocol utilized for treatment of the patient following evacuation of tension pneumocephalus.

## Discussion

### Observations

When a pressure gradient is present between the surgical field and the venous system air can be entrained from the atmosphere into the venous circulation. This gradient most often occurs when the surgical field is above the level of the right atrium. In addition to standard intraoperative monitoring recommended by the Canadian Anesthesiologists' Society, we advocate for VAE monitoring consideration during high-risk procedures. Such monitoring could include TEE, precordial Doppler, and esophageal stethoscope. TEE remains the most sensitive modality, able to detect VAE volumes as little as 0.02 mL/kg.[16] It also allows for visualization of paradoxical air emboli in the left-sided circulation.[2] The main limiting factor for the routine use of TEE is that it requires expertise in its operation and interpretation, thus precluding its utility for most noncardiac anesthetists. TEE is also invasive and requires constant observation, which may not be feasible for surgical procedures of the cranium, head, and neck. In the case described, TEE demonstrated a large amount of air in the left-side circulation with no evidence of an intracardiac shunt, suggesting that the paradoxical air embolism resulted from an intrapulmonary shunt.

Once VAE is suspected, management must be promptly initiated. As the volume of air entrained increases and makes its way to the right heart, an airlock can develop within the right ventricular outflow tract (RVOT), causing obstructive shock physiology. It is imperative that the surgical team rapidly identify and address any possible source of air entrainment in the surgical field. While initiating definitive management, the surgical field should be flooded with saline or saline-soaked gauze to prevent further entrainment.[2,16,17] Immediate anesthetic management includes initiating 100% oxygen

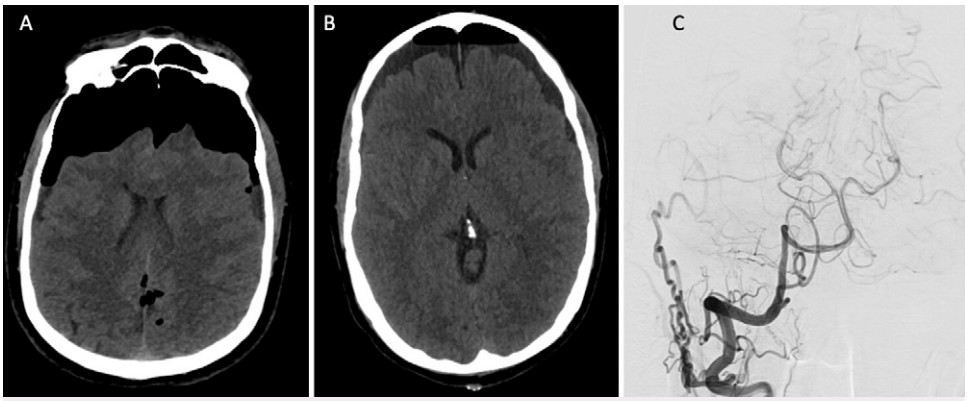


**FIG. 3. A:** Immediate postoperative appearance concerning for tension pneumocephalus (Mount Fuji sign). **B:** Image obtained following hyperbaric therapy and bifrontal burr hole pneumocephalus evacuation. **C:** Angiography at 3 months revealed complete treatment of the lesion.

therapy and providing hemodynamic support with vasopressors and inotropic medications as needed.[2] The patient should be positioned wherever possible to place the surgical field below the right heart to reduce the pressure gradient. Placing the patient in the Trendelenburg and left lateral tilt position also promotes air to move from the RVOT into the apex of the right ventricle, which may reduce the obstructive effects of the air embolism.[2,16,17] In our case, we favored the right lateral decubitus position since a large volume of air had already been visualized in the left atrium and ventricle. Attempts to directly aspirate air from the right ventricle can be done via a centrally inserted venous catheter. Unfortunately, success rates are poor, with the success rate of aspiration via a pulmonary artery catheter quoted at 16%.[2,18] Ultimately, once VAE is identified, management is largely supportive. Depending on the severity of hemodynamic instability, patients may require postoperative ICU care for hemodynamic and ventilatory support.

An additional complicating feature was a simultaneous diagnosis of tension pneumocephalus; although unrelated to the VAE, recognition and management had important implications in hyperbaric therapy safety. Prior studies have described a wide dose range required for VAE, with patients requiring between 1 and 9 hyperbaric sessions.[2,19] Mechanistically, the application of hyperbaric oxygen therapy accelerates nitrogen resorption, facilitating intravascular gas volume contraction. It has also been postulated that increases in the partial pressure of oxygen dissolved in the bloodstream may aid in end-organ perfusion.[18,20] Previous reports have shown favorable outcomes with hyperbaric therapy in the context of intracerebral arterial air embolism and iatrogenic VAE.[21,22] In a retrospective cohort study of 86 patients by Blanc et al.,[23] individuals with venous cerebral VAE treated within 6 hours had better outcomes than those treated later than 6 hours. This underscores the importance of an early decision for hyperbaric therapy when symptomatic severe VAE is encountered. There is, however, case report evidence of a benefit associated with hyperbaric treatment even following delayed diagnosis ($>$30 hours).[24] Importantly, the existing literature suggests the pursuit of hyperbaric therapy in the presence of clinical signs related to VAE regardless of the time of onset. We also emphasize the importance of diagnosing and managing tension pneumocephalus prior to consideration of hyperbaric oxygen therapy, as any trapped gas pockets that equilibrate with the hyperbaric environment during treatment can expand rapidly during decompression and cause further injury and recurrent gas embolism. Although this theoretical risk exists, the exact relationship between hyperbaric therapy for pneumocephalus requires further clarification and likely requires a nuanced understanding of underlying anatomical case-specific details and clear communication between treatment teams. Previous groups have reported the utility of hyperbaric therapy for faster resolution of pneumocephalus, whereas others have documented catastrophic gas expansion complications following hyperbaric therapy, resulting in morbidity[25,26] and even mortality. In our case, we believed it prudent to evacuate the high-volume pneumocephalus and ensure a stable post-evacuation CT scan prior to any consideration of hyperbaric oxygen therapy.

### Lessons

VAE is an intraoperative emergency that, when left unrecognized, can rapidly cause hemodynamic collapse. A coordinated approach by all members of the operative team is crucial when managing a patient with a suspected VAE. Diagnosis can be difficult, and although several modalities are available to detect VAE, it is important to have a high index of suspicion as well as to identify patients who are most at risk.

We described the case of a patient undergoing a sitting-position craniotomy complicated by intraoperative VAE with resultant hemodynamic instability. The immediate postoperative diagnosis of tension pneumocephalus was suspected and managed with bifrontal burr hole evacuation and the hemodynamically significant VAE with prompt hyperbaric therapy. We summarize the existing literature for hyperbaric therapy in the management of VAE and suggest initiating hyperbaric care pathways early following the diagnosis of symptomatic VAE. To our knowledge, this case highlights for the first time the application of hyperbaric therapy for post-neurosurgical intracardiac air embolism and underscores the importance of a multidisciplinary management plan.

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## Disclosures

The authors report no conflict of interest concerning the materials or methods used in this study or the findings specified in this paper.

## Author Contributions

Conception and design: Malhotra, Alves, Jerath, Tillmann, Kumar. Acquisition of data: Malhotra, Foster, Mashari, Kumar. Analysis and interpretation of data: Malhotra, Jerath, Kumar. Drafting of the article: Malhotra, Chang, Jerath, Tillmann. Critically revising the article: Malhotra, Chang, Jerath, Tillmann, Mashari, da Costa, Kumar. Reviewed submitted version of the manuscript: Malhotra, Chang, Lawton, Alves, Jerath, Tillmann, Mashari, da Costa, Kumar. Approved the final version of the manuscript on behalf of all authors: Malhotra. Administrative/technical/material support: Jerath, Mashari.

## Supplemental Information

Video

*Video 1*. https://vimeo.com/802712160.

## Correspondence

Armaan K. Malhotra: University of Toronto, Sunnybrook Health Sciences Centre, Toronto, ON, Canada. ak.malhotra@mail.utoronto.ca.