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CASE REPORTS/CASE SERIES

A case of intrapulmonary transmission of air while transitioning a patient from a sitting to a supine position after venous air embolism during a craniotomy

Un cas de transmission d'air intrapulmonaire pendant le changement de la position assise au décubitus dorsal après embolie gazeuse veineuse au cours d'une craniotomie

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Abstract

Purpose *Since certain surgical procedures still require a sitting or reverse Trendelenburg position, it remains important to evaluate the risk for paradoxical embolization. Intracardiac shunting, the most common cause being a patent foramen ovale, can be excluded by contrast-enhanced transesophageal echocardiography. There are, however, less described cases which result from patency of intrapulmonary functional arteriovenous anastomoses and lead to extra-cardiac paradoxical air embolism during anesthesia. We report a unique case to increase awareness of this real and potentially dangerous complication.*

Clinical features *A 52-yr-old male was scheduled for resection of a tumour at the cerebellopontine angle. Preoperative evaluation excluded intracardiac shunts. During a craniotomy in the sitting position, recurrent venous air emboli entered the patient's right heart, leading to a sudden decline in end-tidal CO_2, an increase in $PaCO_2$, and a reduction of PaO_2. The exact source of surgical entrance could not be identified; therefore, the surgical wound was closed provisionally and the patient was repositioned supine to prevent further venous air emboli. During transition to the supine position, we observed clinically significant crossover of air into the left heart originating from the left pulmonary vein, as detected by transesophageal echocardiography. In all likelihood, the etiology was an opening of intrapulmonary right-to-left anastomoses. The patient recovered without neurological or pulmonary sequelae.*

Conclusion *In the presence of massive venous air emboli, intrapulmonary right-to-left paradoxical air emboli can occur while intraoperatively transitioning a patient from the sitting to the supine position.*

Résumé

Objectif *Dans la mesure où certaines interventions chirurgicales requièrent la position assise ou en Trendelenburg inversé, il est important d'évaluer le risque d'embolie paradoxale. Une communication intracardiaque (la cause la plus fréquente étant un foramen ovale perméable) peut être exclue par échocardiographie transœsophagienne avec produit de contraste. Il y a, toutefois, des cas moins souvent décrits qui peuvent être dus à la perméabilité d'anastomoses artério-veineuses fonctionnelles et aboutir à une embolie gazeuse paradoxale extracardiaque au cours de l'anesthésie. Nous décrivons un cas unique pour sensibiliser à l'existence de cette complication réelle et potentiellement dangereuse.*

Caractéristiques cliniques *Un homme âgé de 52 ans devait subir la résection d'une tumeur de l'angle*

Author contributions *Jennifer Schlundt, Irene Tzanova,* and *Christian Werner* were involved in the concept and design of the article, and *Jennifer Schlundt* drafted the article. *Jennifer Schlundt and Irene Tzanova* were involved in the acquisition and interpretation of data, and *Irene Tzanova* and *Christian Werner* were involved in the critical revision of the intellectual content of the article.

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pontocérébelleux. Le bilan préopératoire avait exclu l'existence de communications intracardiaques. Au cours de la craniotomie en position assise, des embolies d'air veineux récidivants ont pénétré dans le cœur droit du patient, aboutissant à une chute soudaine du CO_2 télé-expiratoire, à une augmentation de la $PaCO_2$ et à une baisse de la PaO_2. La source exacte de la brèche chirurgicale n'a pu être identifiée; en conséquence, la plaie chirurgicale a été fermée provisoirement et le patient a été repositionné en décubitus dorsal pour prévenir d'autres embolies gazeuses veineuses. Au cours du changement de position vers le décubitus dorsal, nous avons observé un passage d'air cliniquement significatif dans le cœur gauche en provenance de la veine pulmonaire gauche, détecté par l'échocardiographie transœsophagienne. En toute probabilité, la cause en était l'ouverture d'anastomoses droites-gauches intrapulmonaires. Le patient s'est rétabli sans séquelles neurologiques ou pulmonaires.

Conclusion *En présence d'embolies gazeuses veineuses massives, une embolie gazeuse paradoxale droite-gauche intrapulmonaire peut survenir en cours d'intervention lors du changement de position du patient, de la position assise au décubitus dorsal.*

The existence of intrapulmonary arteriovenous pathways dominating in the lung apices and measuring up to 0.5 mm in diameter has been documented in human cadavers of previously healthy individuals.[1,2] Transpulmonary arteriovenous anastomoses have been shown in infants[3] and several animal models under different conditions.[4-7] While these physiological transpulmonary arteriovenous pathways are subclinical in most instances, they have potential to open in up to 90% of individuals during hyperdynamic situations such as exercise.[8-10] A recent review of pulmonary pathways and mechanisms pertaining to their autoregulation highlights the interest and importance of this subject.[11] Neuroanesthesiologists are well aware of the multiple pathways for paradoxical air embolism (PAE) to occur. The following case description highlights a role for continuous transesophageal echocardiography (TEE) to detect or exclude the possibility of PAE during surgeries with a high potential for venous air emboli (VAE). The patient provided written consent for the publication of this report.

Case report

A 52-yr-old Caucasian male was scheduled for tumour resection at the cerebellopontine angle to be performed with the patient in the sitting position. Preoperative evaluation revealed an uneventful medical history and laboratory results were within normal limits. The electrocardiogram (ECG) was normal except for an incomplete right bundle branch block. Transcranial Doppler examination and colour duplex sonography showed no pathology of the carotid arteries. In the operating room, standard monitors were applied in addition to invasive arterial blood pressure measurement, five-channel ECG, continuous central venous pressure, TEE, pre-cardiac Doppler, and near-infrared spectroscopy (regional oximetry technology Model 7600® by Nonin, Hudiksvall, Sweden) applied over the left and right frontal cerebral cortical areas. Following an unremarkable anesthetic induction, special care was given to cervical and head positioning using a Mayfield® clamp. The central venous line was positioned at 23 cm at the superior vena cava-right atrial junction, confirmed by TEE. The preoperative TEE Vivid S6® (GE Healthcare, Milwaukee, WI, USA) probe was inserted after induction. No TEE evidence of patent foramen ovale, atrial septal defect, or ventricular septal defect was observed in both the supine and sitting positions using contrast-enhanced ultrasound with Gelafundin 4% microbubbles injected during a simulated Valsalva maneuver (ventilation maneuver at 25-30 cm H_2O; TEE monitoring during strain with focus on release phase). The initial blood gas analysis after induction in the supine position showed a $PaO_2 = 351$, fraction of inspired oxygen (F_iO_2) = 0.78 mmHg, and $PaCO_2 = 32$ mmHg. Shortly after transferring the patient to the sitting position, the PaO_2 was 194 mmHg and F_iO_2 was 0.45. The anesthesia machine, Zeus® Infinity® (Dräger; Lübeck, Germany), was used to control ventilation of the patient's lungs in the autoflow and pressure mode.

During tumour resection, VAE entered the right heart during the 25th, 75th, 95th, and 140th (Fig. 1, Video 1) minutes of surgery. During all episodes, manual

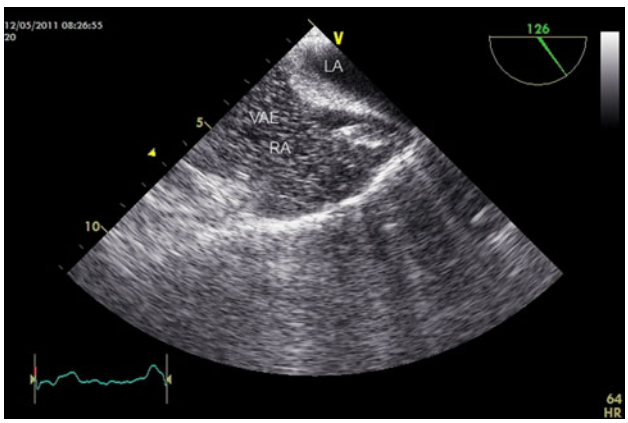

Fig. 1 Transesophageal echocardiography showing a large quantity of air emboli in the right atrium. Mid-esophageal bicaval view in a multiplane angle at 126°. RA = right atrium; LA = left atrium; VAE = venous air emboli

compression of jugular veins was applied repeatedly and aspiration of air was attempted through the central venous line. Potential extra-surgical sources of air (central and peripheral venous lines) were excluded. During the first and second VAE, the end-tidal (Et) CO_2 and hemodynamic variables were stable, and VAE were minimal and terminated quickly. During the third VAE episode, the $EtCO_2$ values decreased from 33 to 26 mmHg but recovered rapidly to 32 mmHg. At this point, surgical demand was the reason to postpone the positional change, and neurosurgical intervention seemed to end VAE as in the first two episodes; the hemodynamic situation remained stable. Surgical priority was focused on cerebral venous drainage, avoiding the pooling of blood in the surgical field, and minimizing blood loss at that critical stage of surgery. The fourth VAE (140^{th} minute) led to a drop in $EtCO_2$ to 22 mmHg. At this point, the changes in $PaCO_2$ corresponded, increasing from 33 to 49 mmHg. The PaO_2 decreased from 233 to 105 mmHg, while the F_iO_2 was constant at 0.5. Surgical exploration to determine a portal of air entry was unsuccessful in terminating the fourth VAE. The patient's blood pressure decreased to 80/40 mmHg while heart rate increased from 71 to 90 beats·min^{-1}. At this point, norepinephrine at an infusion rate of 0.02-0.05 mg·kg^{-1}·min^{-1} was required to achieve hemodynamic stability. Given this new hemodynamic instability, the surgical wound was closed temporarily and the patient was promptly transferred to the supine position in order to prevent further VAE.

The TEE images were examined before and during the transition to the supine position, and we detected a significant crossover of air bubbles into the left heart during the change in position (Fig. 2, Video 2). The TEE studies were repeated by two different attending cardiac anesthesiologists, both of whom verified the crossover of air deriving from the left pulmonary vein. This finding led to the presumptive diagnosis of a patent right-to-left intrapulmonary arteriovenous pathway allowing transmission of air emboli during the transition to the supine position. After entrance of air into the left heart, the regional cerebral oximetry values reflected a concordant reduction of around 12% bilaterally relative to baseline values of 85/89 (left/right). However, the portal of entry of air into the right heart resolved after repositioning the patient to the supine position. The right regional cerebral oximetry values increased rapidly to 98% after cessation of VAE, but the left-side oximetry values remained at 88%. Surgery ensued with the patient lying in the supine position, his head in a Mayfield clamp and rotated to the right side to facilitate surgical exposure to the left cranium.

Postoperatively, the patient was transferred to the intensive care unit (ICU) with his trachea intubated while he was sedated with propofol 6 mg·kg^{-1}·hr^{-1} and with his lungs ventilated at an $F_iO_2 = 0.6$, $P_{insp} = 16$ mmHg, positive end-expiratory pressure = 5 mmHg, and respiratory rate = 14 breaths·min^{-1}. The patient remained hemodynamically stable while receiving norepinephrine 0.05 mg·kg^{-1}·hr^{-1}. On arrival in the ICU, the hemoglobin was 12.6 g·dL^{-1} and the patient's pupils were equal and sluggishly reactive to light.

The first ICU chest *x-ray* (Fig. 3) in the supine position showed bilateral prominent hili and bilateral perivascular interstitial edema. The initial postoperative cerebral computed tomography scan showed air in the frontal cerebral cortex and the ventricles; there were no signs of cerebral ischemia. On the fourth postoperative day, the chest-*x-ray*

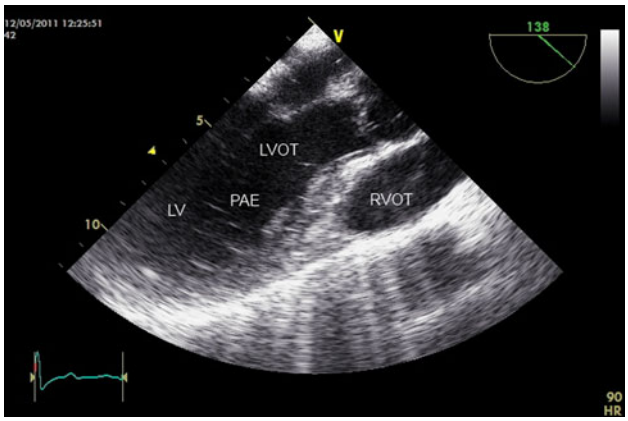

Fig. 2 Transesophageal echocardiography showing air emboli in the left heart and the ventricular outflow tract after transitioning to the supine position. Mid-esophageal long axis view in a multiplane angle at 138°. LV = left ventricle; LVOT = left ventricular outflow tract; RVOT = right ventricular outflow tract; PAE = paradoxical air emboli

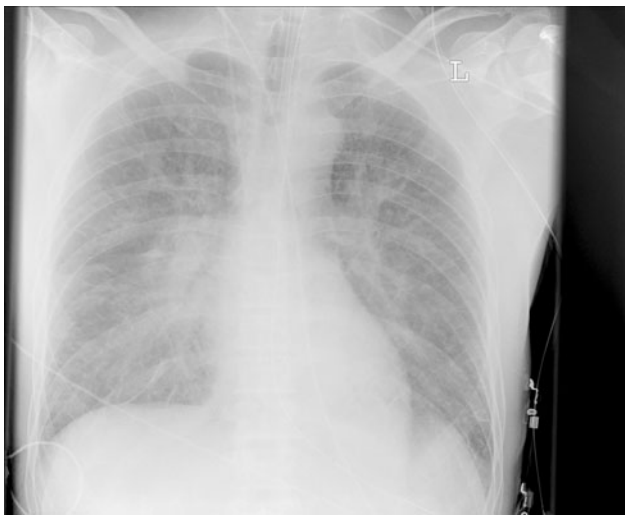

Fig. 3 Chest *x-ray* anteroposterior (supine) on the first postoperative day in the intensive care unit showing bilateral prominent hili and bilateral perivascular interstitial edema

was normal. The patient's trachea was extubated and he was transferred to the neurosurgical ward. Here, the patient was fully oriented and cooperative, and his neurological exam was normal except for minimal left ear tinnitus. He had a slow and somewhat unsteady gait, but his condition had improved compared with his preoperative status. There was no dyspnea at rest or upon exertion and breath sounds were clear bilaterally. A postoperative chest computed tomography scan excluded evidence of arteriovenous malformations but showed a persistent left superior caval vein (SCV). The postoperative chest magnetic resonance imaging scan confirmed absence of anatomical arteriovenous malformations and showed evidence of persistent SCV blood flow into the coronary sinus and into the right heart.

Discussion

This case highlights how, in the presence of massive venous air emboli, intrapulmonary right-to-left paradoxical air emboli can occur when transitioning a patient intraoperatively from the sitting to the supine position. Careful examination of this patient preoperatively excluded evidence of intracardiac functional shunts, deliberately considered due to the known risks of neurosurgery in the sitting position.[12] Cases of paradoxical emboli without presence of an intracardiac shunt are rare with but a few isolated case reports.[13,14] The terms intrapulmonary *shunting* or *shunts* have been deliberately avoided throughout this article because this case does not take into account whether these arteriovenous anastomoses contribute to gas exchange, a fact still being debated.[15,16] The focus of this report is the potential for intrapulmonary arteriovenous blood to flow through vessels allowing unfiltered air emboli to bypass the pulmonary capillaries.

There are two explanations for transit of paradoxical air emboli, either by means of the capillary bed in the lung or through pulmonary arteriovenous anastomoses that bypass the capillary system. These can present either in the form of a classic arteriovenous malformation, as with hereditary hemorrhagic teleangiectasia,[17-19] or during hyperdynamic circulation and/or when the alveolar-arterial partial pressure gradient of oxygen is high, as in the case of this patient whose lungs were ventilated with $F_iO_2 = 0.5$.

If sufficiently massive and rapid, venous air emboli can induce a functional obstruction of the pulmonary capillary bed, the pulmonary arteries, and the right ventricular outflow tract. The pulmonary circuit in healthy individuals can filter small volumes without consequences to pulmonary vascular pressure.[20] A significant amount of venous air emboli entering the pulmonary capillaries may cause functional obstruction of the microvasculature in the form of a precapillary block and may lead to increased pulmonary vascular resistance, sustained pulmonary hypertension, and increased peripheral resistance.[21,22] While the property of turbulent flow should not be neglected, when taking Hagen–Poiseuille's equation $\left(\mathbf{dV} = \frac{\pi \mathbf{r}^4}{8\mathbf{l}\mu\mathbf{P}}\right)$ into account, an increase in vessel diameter would decrease driving pressure needed to maintain flow. It has been suggested that intrapulmonary arteriovenous anastomoses may function as "pop-off valves" in dogs in response to increases in flow and pulmonary vascular resistance mediated by norepinephrine.[23] This might imply that recruitment of intrapulmonary anastomoses could be an adaptive mechanism to reduce the potential damaging effects of high perfusion pressures, and they may be in place when the pulmonary vascular resistance is increased due to an obstruction, as with venous air emboli.

Another mechanism which may allow opening of intrapulmonary vessels to facilitate passage of microbubbles may be hypoxia-mediated. With deteriorating gas exchange, as reflected by an increased difference between $EtCO_2$ and $PaCO_2$ and impaired oxygenation, pulmonary vasoconstriction may have been present in some pulmonary regions in this patient. This concept is supported by evidence from a study by Lovering *et al.*[9] which showed that, during exercise, 90% (8/9) of subjects recruited intrapulmonary shunt pathways during normoxia, whereas all subjects shunted during hypoxia. If patency of intrapulmonary anastomoses can possibly be modified through adjustment of the F_iO_2, then this should be taken into account. When administering anesthesia for operations involving risk of intrapulmonary right-to-left transmission, anesthesiologists should consider maintaining higher levels of F_iO_2. Different authors have shown that hyperoxia may prevent or reduce blood flow through arteriovenous pathways bypassing the capillary system when they are exercise-induced. Lovering *et al.* showed that "breathing oxygen for one minute reduced shunting and breathing oxygen for two minutes eliminated shunting in all subjects".[24] It remains unknown whether F_iO_2 or oxygen tension specifically regulates these recruited anastomoses or opens them indirectly, and more research is required.

There are two different reasons why patient positioning and changes in a patient's position may play a role. The focus of the first mechanism is on the ventilation/perfusion mismatch concept and diffusion abnormalities. It is known that positioning a patient in the sitting position increases perfusion of the lung base and facilitates relatively improved ventilation for the lung apices. Therefore, when the patient is maintained in the sitting position, apical arteriovenous pathways that open would be less relevant than those that open following patient transitioning to the supine position. In the latter case, the apical pathways become better perfused and may become clinically relevant, as in transmission of

paradoxical air emboli. The second theoretical mechanism related to postural change is anatomical in nature. If VAE occur in the sitting position, they should collect predominantly in the right atrium, anatomically the most cranial structure in the heart, which should pool the air emboli. When a patient is transferred to the supine position, the most ventral structure is the right ventricular outflow tract with the pulmonary artery. This postural transitioning may drive more air into the pulmonary microvasculature, and as a result, pulmonary hypertension may increase to some degree. Body positioning can be a factor in recruitment of these transpulmonary pathways, especially in the supine position.[8] Therefore, changing the patient's position from sitting to supine during an interventional procedure under increased pulmonary microvasculature pressure, i.e., when significant venous air emboli are present, may increase the risk for transpulmonary transmission, especially via pathways in the apical lung.

The imaging studies performed while this patient was at rest and in normoxia were able to exclude large anatomical arteriovenous malformations. However, they could not exclude a transient opening of these intrapulmonary pathways from reoccurring during extreme physical activity or hemodynamic stress combined with hypoxia (as was present during surgery). Although it remains unclear if the persisting left SCV contributed to the occurrence or quantity of VAE, this anatomical variation may have provided a more direct pathway for VAE into the right heart when left-sided brain surgery was performed. This case report emphasizes the importance of understanding the mechanisms and mediators for patency of transpulmonary arteriovenous pathways. Surgeries in the sitting position are high-risk procedures and must be focused on patient safety. Optimal monitoring (with pre-cardiac Doppler and TEE) and correct positioning of the central venous line (ultrasound or biphasic P wave) are key safety elements. Before a clinically significant amount of VAE occurs, early interdisciplinary discussion should prompt transferring the patient to a different patient position (right side up), if appropriate, and rapid means to occlude the portal of air entry.

Conflicts of interest None declared.

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