REVIEW ARTICLE

Air Embolism in Sitting Position during Neurosurgical Operations and It's Prevention: A Narrative Review

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Venous air embolism (VAE) is an entrapment of air or exogenously delivered gas from the operative field or the communicated environment into the venous system due to gradient pressure. VAE and paradoxical air embolism (PAE) are potentially serious neurosurgical complications and carry high neurologic, respiratory, and cardiovascular morbidity or even may be fatal. Some procedures are at risk of developing these conditions, but the sitting and semi-sitting positions represent a higher risk. It's occurrence during anesthesia is challenging to the anesthesiologists in terms of early discovery and management. Anesthesiologist can play an important role in detection and urgent treatment of VAE in case of occurrence. If the sitting position is needed for a neurosurgical necessity it can be used with vigilant follow up throughout the procedure to detect any occurrence of VAE.

Keywords: **Vascular air embolism; Venous air embolism; Paradoxical air embolism; Sitting position; Neuroanesthesia**

Vascular air embolism is the entrapment of air or exogenously delivered gas from the operative field or other environment into the venous or arterial vasculature. An intravascular air embolism is a rare and preventable event. Although, this complication is serious which may lead in morbidity and mortality. In the past 30 years alone more than 4,000 articles have been published to illustrate the frequency and severity of this vascular event. Its incidence ranges between 10% and 80% [1], depending on the studied literature model. In pediatric neurosurgery the reported incidence ranges between0.42% and 9.8% [2-3]. This wide range of VAE incidence may be due to the ample variety of surgical and anesthetic techniques reported and different diagnostic methods used. VAE-related mortality in neurosurgery is unclear, but there are some case reports of fatal outcomes following massive air embolism [4]. Many cases of VAE have no observable symptoms, resulting in no untoward outcome, and thus go unreported. So, the true incidence of VAE may never be known, much depending on the sensitivity of detection methods used during the procedure. VAE occurs because of a pressure gradient that allows air to enter the blood stream, and subsequently occlude blood flow. Both volume and rate of air accumulation are dependent on the size of the vascular lumen as well as the pressure gradient.

These factors determine the morbidity and mortality of any episode of VAE.

On the other hand, pathophysiologic pathways are highly dependent on the volume of gas accumulated within the right ventricle. Intracardiac shunting through a patent ovale window is the most common cause of paradoxical air emboli (PAE).

In neurosurgical practice, VAE has been most frequently observed during operations in the sitting position or elevated head and PAE may occur as a secondary complication of VAE [5-6]. VAE is most often associated with posterior fossa surgery, cervical spine surgery in the sitting position as well as craniosynostosis repair [7-8]. There is still an ongoing debate about the use of the sitting and semi sitting positions during neurosurgical operations on the dorsal cervical spine and the posterior fossa [9]. This position provides several surgical advantages [10]. Gravity helps to keep the operating field free from blood and cerebrospinal fluid. It has been reported that the sitting position (SP) can reduce the risk of injuries to the cranial nerves and allows the anesthetist better access to the airway [11]. The incidence of VAE in the sitting position is variable but has been described from 25% to 76% of the cases [6]. Looking to the seriousness of this complication, it is imperative for anesthesiologists to be aware of the causes, diagnosis, treatments, and adoption of practice patterns of VAE that best lead to the prevention or treatment of this potentially fatal condition.

Pathophysiology:

Once the air enters into the circulation it lodges inside the superior vena cava and the right atrium. Some of that amount of air may pass through tricuspid valve and reach the pulmonary artery [12]. The rate of air entrainment is also important because of the interface of the pulmonary circulation and alveoli allow for dissipation of intravascular gas. If entrapment of air is slow, the heart may be able to

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withstand large amount of air despite entrainment over a prolonged time. In general, when the amount of air is small the pulmonary capillaries have the ability to filtrate it without producing symptoms, but when the volume of air is larger, the air passes in to the pulmonary capillaries causing vasoconstriction and impairment of ventricular and pulmonary ratio [13]. Any reduction in the pulmonary venous return will lead to reduced left ventricular preload with resultant decreased cardiac output and eventual systemic cardiovascular collapse [8,14].

The entrapped air can affect the pulmonary vasculature and lead to serious inflammatory changes in the pulmonary vessels; these include direct endothelial damage and accumulation of platelets, fibrin, neutrophils, and lipid droplets. Secondary injury as a result of the activation of complement and the release of mediators and free radicals can lead to capillary leakage and eventual noncardiogenic pulmonary edema [8,15]. Air embolism has been described as a potential cause of the systemic inflammatory response syndrome triggered by the release of endothelium-derived cytokines [16].

Alteration in the resistance of the lung vessels and ventilation-perfusion mismatching can lead to intra-pulmonary right-to-left shunting and increased alveolar dead space with subsequent arterial hypoxia and hypercapnia [8,17].

Paradoxical air embolism (PAE) occurs when the air passes from right to the left atrium and then to the systemic circulation through a patent foramen ovale. PAE potentially can enter any arterial vessels, such as coronary artery causing myocardial ischemia, but they are often found in the brain vessels [5,18]. Air entering the cardiac or cerebral vasculature causes end arteriolar obstruction, resulting in distal hypo perfusion and hypoxia. Cellular damage leads to tissue edema, further reducing oxygenation.

Etiology

Air embolism exists only when there is a connection between air and the vascular system in the presence of pressure gradient. The procedures with the highest risk of VAE are when the patient is in the sitting or semi-sitting position especially in a case of posterior fossa surgery and cervical spine surgery. However, it is important to mention that the alternative positions such as prone or "park bench" position do not rule out the probability of VAE [12]. Surgery to correct craniosynostosis is one of the procedures with high incidence of VAE and represents an important cause of morbidity or mortality in the pediatric population [19]. Recently, surgery for deep brain stimulation with the patient awake for the treatment of Parkinson' disease or other motion disorders has been reported to be with a high risk of VAE. Anytime the surgical site is more than 5 cm above the right atrium can carry a risk of VAE [20]. The presence of abundant, large, non-compressed, venous channels in the surgical field also increase the risk of VAE.

VAE may also result from many surgical procedures, including vascular procedures, liver transplantation, orthopedic procedures such as (hip replacement, spine surgery, arthroscopy) etc. Not only negative pressure gradients but also positive pressure insufflations of gas may present a serious VAE. Mechanical insufflations or infusion during arthroscopy or laparoscopy is another cause of VAE. Positive-pressure ventilation during mechanical ventilation puts patients at risk for barotrauma and, arterial and/or venous air embolism [8,15]. VAE has also been described in neck and craniofacial injuries. In contrast, arterial air embolism occurs paradoxically following direct instillation of air into the arterial system, through a septal defect or patent foramen ovale (PFO). Though the higher intravascular pressure in the arterial system is somewhat protective, arterial air embolism has the potential to produce ischemia or infarction in any organ with limited collateral blood supply, even when the volume of air is small. Additionally, air embolism most commonly occurs with ambient air but it has also been reported to occur with a variety of gases including helium, nitrogen, and carbon dioxide [21].

Clinical manifestation

Volume of entrained air and rate of accumulation are the two fundamental factors that determine morbidity and mortality of VAE. These variables are mainly affected by the position of the patient and height from the right atrium. In humans, the lethal dose of air embolism is unknown but from case reports of accidental intravascular delivery of air [8,23], the adult lethal volume has been described as between 200 and 300 ml, or 3–5 ml/kg [8,22]. The volume of air in the venous system that may trigger clinical manifestations is about 100 ml for the adult [13]. The rate of air entrainment is also important, because the pulmonary circulation and alveolar interface provide for a reservoir for dissipation of the intravascular gas. If entrainment is slow, the heart may be able to withstand large quantities of air despite entrainment over a prolonged period.

The main affected systems in VAE are cardiovascular, respiratory, and central nervous system. The clinical effect of arterial embolization is highly dependent on the site. However, especially the heart, lungs and brain are more sensitive and small volumes of air will result in significant complications [23].

Cardiovascular: Chest pain and palpitations associated with brady and tachyarrhythmias are possible in awake patient. Ischemic ECG changes may be found. ECG may show ST segment changes or right ventricular strain pattern [24].

Air accumulating in the right heart and pulmonary vasculatures even a small volume will lead to a gradual elevation in pulmonary artery pressure, which in turn will put the right heart under increasing strain. Blood pressure decreases as cardiac output falters. Continuous air entrainment is more likely to be problematic as a large volume of air in the right ventricle (RV) will result in outflow tract obstruction and acute right sided heart failure [24]. The central venous pressure (CVP) also increases as a secondary effect of right heart failure, and jugular venous distension may be noted. The reduction in the RV outflow will compromise left ventricular preload leading to cardiovascular collapse. A volume of 5ml/kg is considered large enough to cause an “air-lock” effect in the right ventricular outflow tract, with resultant cardiovascular collapse due to catastrophic reduction in cardiac output [8].

Small volumes of air entrained into the coronary circulation, especially the left anterior descending artery, rapidly leads to ischemia and cardiac arrest. PAE commonly results in symptoms of angina and/or an embolic stroke [24].

Respiratory: In an anaesthetized patient, a sudden drop in end tidal carbon dioxide (ETCO2) is observed due to the dead space ventilation caused by air in the pulmonary

circulation. Decrease of arterial oxygen saturation (SaO2) and tension (PO2), along with hypercapnia, may be detected. The degree of ventilation-perfusion mismatch will be revealed as hypoxemia and hypercarbia on arterial blood gas analysis. Vascular air embolism can also trigger an inflammatory cascade, resulting in an acute lung injury and non-cardiogenic pulmonary edema. Sudden shortness of breath and pleuritic sub-sternal chest pain can occur with a dry cough when the patient is awake. Other pulmonary symptoms include acute dyspnea, continuous coughing [8,25], urgent complaints of breathlessness [8], lightheadedness, and a sense of "impending doom." The common response of gasping for air as a consequence of dyspnea forces a further reduction in intrathoracic pressure, frequently resulting in more air entrainment. Pulmonary signs of VAE include rales, wheezing, tachypnea, and Hemoptysis is a relatively late sign [24].

Central nervous system (CNS) stroke can occur due to arterial air embolism, which can manifest clinically as abnormal pupillary response to light or failure to wake up following general anesthesia. The patient may deteriorate into coma as cerebral edema develops during the postoperative period. If blood supply to the brain stem is interrupted in an awake patient, there may be cardiac dysrhythmias and apnea, sudden onset of confusion, dysarthria, hemiparesis and seizure. Gas bubbles may rarely be observed in retinal vessels on fundoscopy [24].

Method

Close patient monitoring plays a crucial role in early detection of air embolism, allowing early active management and measures to prevent further air entrainment. It is the responsibility of the anesthetist to stay vigilant and pay careful attention throughout the operation. Each hospital has an anesthesia monitoring protocol to detect VAE episodes. The monitoring devices that are used to detect VAE should be sensitive, easy to use, and noninvasive. Selection depends on the position of the patient, the expertise of the anesthesiologist in using the device, and the medical condition of the patient [8]. Some of which are standard, and several specifically used for detecting VAE. High risk cases necessitate suitable detection devices and a high index of suspicion. Clinical indicators are late signs of VAE.

There are two detection methods; direct and indirect clinical detection methods.

Direct clinical detection methods:

Mainly two devices are used to detect VAE in a direct way: The Transesophageal Echocardiography (TEE) and the Precordial Doppler.

1-Transesophageal Echocardiography (TEE) is the most sensitive monitoring device for VAE and may detect as little as 0.02 ml/kg of air administered by bolus injection. It allows both the ability to detect micro emboli and also has the advantage of identifying paradoxical air embolism that may result in ischemic cerebral complications, some of structures can be imaged and evaluated better with the TEE, including the aorta, pulmonary artery, valves of the heart, both atria, atrial septum, left atrial appendage, and coronary arteries. However, it is invasive, expensive, requires expertise and constant vigilance, and the risk of esophageal injury during prolonged use has limited its clinical use [13]. The use of TEE as standard of practice represents only 38% of patients undergoing intracranial procedures, compared with the use of precordial Doppler [8]. In addition, because it does not have alarm capability, TEE requires constant surveillance by someone trained in echocardiography, thus limiting its use for the solo anesthesiologist.

2-Precordial Doppler: has a high sensitivity and may detect as little as 0,25ml of air (0,05ml/kg) [8,13]. It is highly sensitive as compared to capnography and conventional hemodynamic monitoring for the diagnosis of VAE. Regarding its positioning, the Doppler probe is normally placed on either the right or the left sternal border (second to fourth intercostal spaces) or, alternatively, between the right scapula and the spine [8]. In order to confirm if the Doppler probe is in the right place (which means that the anesthesiologist may listen to the heartbeat clearly), the probe is placed along the right heart border, to pick up signals from the right ventricular outflow tract. The anesthesiologist should be familiar with the sound of the pericardial Doppler when the air flows in to the heart cavities. However, this device presents some weaknesses, namely the inclusion of sound artifacts when the patient is in the prone or lateral positions [8,26]. Another limitation is the absence of air volumes' quantification. This information is mandatory for the anesthesiologist in order to prescribe suitable clinical treatments [26]. Detection of the VAE relies on the continuous attention of the anesthesiologist but this may be difficult in long duration neurosurgeries, where the anesthesiologist has to focus his attention in other tasks. Besides this, the surgery room is not a noise-free environment and the presence of noise could mask the existence changes in the Doppler Heart Sound.

Indirect clinical detection methods:

These methods are frequently included in the monitoring protocols, providing useful information about the patient's state and that may be useful do discern about the occurrence of VAE episodes [27].

1- End Tidal CO2(ETCO2) monitor is the most convenient and practical American Society of Anesthesiologists monitor used in the operating room, and critical importance must be paid to this monitor for a high-risk case [8]. ETCO2 monitor is the common device used to detect relative pressure changes of carbon dioxide in the expired air. A change of 2 mmHg ETCO2 can be an indicator of VAE. Therefore, the low-level alarm should be adjusted to detect even this small decrement, especially in high-risk procedures [28]. ETCO2 monitoring is not highly specific, and its reliability in the event of systemic hypotension is difficult to assess. In addition, in spontaneously breathing patients, this monitor may become unreliable during periods of upper airway obstruction, mouth breathing, and variations in respiratory rate or obstruction of the gas analyzer port by mucus or condensation [8].

2-End-tidal Nitrogen (ETN2) is the most sensitive gas-sensing VAE detection method, can measure increases in ETN2 as low as 0.04% [8]. The presence of nitrogen in the expired gas monitor when the patient is breathing 100% oxygen is highly suggestive of VAE. It has been shown that changes in ETN2 occur 30–90 s earlier than changes in ETCO2 [8,29]. The sensitivity compares to or exceeds that of ETCO2 during large-bolus VAE but may be less sensitive during slower entrained volumes. Unfortunately, not all anesthetic monitors have the capability to measure ETN2, and this method is not useful if nitrous oxide is used as a

carrier gas. The presence of ETN2 may also indicate air clearance from the pulmonary circulation prematurely, and the method is limited by hypotension. This method is not widely used as a routine for neuroanesthesia monitoring [8].

3- Pulmonary artery catheter (PAC): A pulmonary artery catheter is a relatively insensitive monitor of air entrainment (0.25 ml/kg). A small amount of entrained air can increase PAP. The degree and extent of this increase correlates with the amount and rate of air entrainment. Thus, while the PAC offers early detection of VAE and has the possible advantage of offering prognostic information as to whether the surgical procedure should be continued [13]. It is not generally helpful in a therapeutic context, i.e., withdrawing air because of the small caliber and fixed distance between the proximal and distal ports and variability in patient size. The PAC is slightly more sensitive than capnography. The use of the pulmonary artery catheter is thus restricted to those patients who have significant comorbidities that may benefit from its use as a monitoring tool for cardiac output or mixed venous saturation. One other factor to consider is that for procedures in which the patient is positioned 90 degrees upright (e.g., seated posterior fossa craniectomy), the PAC may also assist in the management of hemodynamic changes inherent in assuming this position. A 25 cm, 9 Fr multi-orifice sheath has been developed that permits aspiration of air and simultaneous placement of a PAC [13].

4-Transcranial Doppler (TCD) and transcranial color Doppler (TCCD) are kinds of Doppler ultrasonography that measure the velocity of blood flow through the brain's blood vessels by measuring the echoes of ultrasound waves moving transcranially. Contrast-enhanced transcranial Doppler has been shown to be highly sensitive in the detection of a patent foramen ovale and has been used as a screening tool for patients undergoing high-risk procedures. The role of transcranial Doppler in the diagnosis of VAE is limited to the identification of arterial air embolism in the presence of patent foramen ovale [30].

5- The observation of the surgical field: it is very important and should be frequent during seated procedures. Observing the absence of oozing venous blood from bone during removal of a craniectomy flap prior to bone wax applications is indicative that the venous pressure is less than the atmospheric pressure and poses a potential VAE risk. One hope is to find slow continuous venous bleeding as an indication that local venous pressure is greater than atmospheric pressure. If bleeding is absent, the index for suspicion for VAE should be extremely high [31].

6- Others: A change in oxygen saturation SpO2 is a late finding of VAE and typically requires a severe physiologic disturbance because patients often are exposed to a high fraction of inspired oxygen during surgery. Esophageal stethoscope can be used to detect VAE but its sensitivity has been shown to be very low in detecting a mill wheel murmur, mill wheel is audible only in large embolus (1.7 ml/ kg /min). Also, the electrocardiograph ECG may be useful in VAE detection. Changes are seen early only with rapid entrainment of air, and generally reflect an already compromised cardiac status, ST–T changes are noted first, followed by supraventricular and ventricular tachyarrhythmias [32].

VAE prevention

The optimal management of VAE is prevention. Those patients undergoing surgeries associated with a significant risk of VAE should be identified preoperatively to allow preventive measures and appropriate monitoring to be used. Good communication between all the members of the involved medical staff is imperative. Preoperative screening for PFO in patients scheduled for procedures carrying a significant risk of VAE has been recommended [31]. Minimizing the pressure gradient between the site of potential entry and the right atrium is essential in prevention of VAE.

The most important measures to prevent VAE is to avoid injury to the venous system in the first place. Special care should be taken during the craniotomy, where an opening in the venous system may be difficult to close because of the limited access at that stage of surgery. Careful preoperative study of the cranial CT scan and another image techniques are mandatory for the neurosurgeon to get aware of and prepare for quick closure of emissary veins. Burr holes should not be close to the transverse or sigmoid sinus. Any necessary exposure to the transverse or sigmoid sinus is always performed in the last step to ensure an optimal access in a case of an injury to the venous system [31].

During craniotomies, bilateral compression of the jugular veins should be performed frequently, even without suspicion of a VAE. Bilateral rather than unilateral jugular compressions are necessary to increase the intracranial venous pressure quickly. Increasing venous pressure may identify open Dural sinuses and result in retrograde flow thus decrease air entry into the heart [8]. This is a controversial maneuver since it may raise the intracranial pressure, reduce brain perfusion and concomitantly compress the carotid arteries resulting in a decrease in cerebral blood flow and possible dislodgement of atheromatous plaque, venous engorgement leading to cerebral edema, and carotid sinus stimulation causing severe bradycardia. Inflatable neck tourniquet can be used for compression of the jugular veins during neurosurgery in the sitting position. This simple device allows increasing intrasinusal pressure above the atmospheric due to external compression of jugular veins [5,33]. This method is 100% effective but it consumes time, as for safety reasons it requires catheterization of the jugular vein (venous pressure control) above the tourniquet position [34]. Small venous entry points in the skull or musculature revealed with this method should be closed immediately with bone wax to prevent more blood leak and avoid the air to be enter the venous system [31].

Hydration: an increased incidence of VAE has been reported in patients with a low central venous pressure, which enhances the negative pressure gradient at the wound site compared to the right atrium [8]. Intravenous infusion of fluid directly expands plasma volume. Prophylactic intravenous fluid loading increases vascular volume, subsequently and usually improves blood pressures if the heart is preload-responsive, and often improves oxygen delivery and tissue oxygenation. These alterations are profoundly influenced by the cardiac and peripheral vascular status [35]. Prophylactic intravenous fluid loading also reduces the incidence of a right-left atria1 pressure gradient and the associated risk of PAE [13]. It has been proposed to maintain the right atrial pressure between 10 and 15 cm H2O, depending on the elevation of the patient [8-36]. Regarding hydration, optimizing volume status should be adjusted to prevent wide gradients between the right atrium and the entraining vein, which may be guided by

measurement of central venous pressure, along with other parameters of volume assessment such as respiratory variations in systolic blood pressure and urine output.

Medical anti shock trousers (MAST) this method has been used to increase venous return to the heart. The amount of blood auto transfused is estimated to be 750 to 1000 mls [37]. The use of military anti shock trousers during surgery have been shown to elevate right atrial pressure (RAP) in the sitting position. By maintaining a MAST pressure of 50 cmH2O, RAP is sustained above atmospheric pressure during the period of inflation. There are some complications during applying MAST including hypo perfusion to intraabdominal organs with excessive pressures. If the time to apply MAST is prolonged, patients may suffer respiratory acidosis and decrease pulmonary vital capacity, which could be harmful inpatients with preexisting respiratory disease [37]. MAST application is contraindicated in patients with diaphragmatic injury, thoracic injury, abdominal evisceration and gravid uterus among others. Patients presenting with pulmonary edema, acute myocardial infarction, cardiac tamponade, cardiogenic shock should not have MAST applied because the usage may worsen the cardiac conditions of these patients [38].

Positive end-expiratory pressure (PEEP) has been used to treat and prevent venous air embolism in patients in the seated position undergoing neurosurgical operations. However, the safety of PEEP has been questioned, because of concern that PEEP might increase right atrial pressure (RAP) more than left atrial pressure, thereby predisposing patients with a patent foramen ovale to paradoxical air embolism. PEEP can interfere with intracranial pressure (ICP) by increasing intrathoracic pressure. In humans, moderate PEEP fails to elevate cerebral venous pressure in the seated position [13]. The use of PEEP during procedures in the sitting position is controversial and few controlled studies have been done. Earlier studies advocated the use of PEEP to decrease the risk of VAE by increasing right atrial pressure and, in turn, increasing cerebral venous pressure above atmospheric pressure [39]. Some of these studies suggest that the use of about 10 cm H2O of PEEP during neurosurgical procedures performed in the sitting position should be abandoned as it does not decrease the incidence of venous air embolism but is associated with significant adverse cardiovascular effects [40].

Also, the effect of ventilation (normo-, hypo- and hyperventilation) on transverse sinus pressure (TSP), central venous pressure (CVP), mean arterial blood pressure (MABP), and heart rate was studied by el Zentner J al et. [41] in 15 patients undergoing neurosurgical treatment in the sitting position for tumors of the posterior fossa and the findings were compared with the influence of PEEP on these parameters. They Presumed that the risk of venous air embolism is closely related to the level of TSP.

They concluded that, PEEP does not seem to be effective in preventing venous air embolism, hyperventilation is dangerous in the sitting position, as TSP is reduced to the atmospheric and even sub atmospheric range. Hypoventilation is recommended during the most critical period of exposing the posterior fossa to prevent air embolism moderate followed by normoventilation when surgery of the actual lesion has begun [41].

Surgical position:

The anesthesiologist must be aware that surgery in the head-up position places the patient at risk for VAE. This may occur during craniotomy, spine procedures, shoulder surgeries and other procedures near the head and neck. In such conditions, the tendency of incurring a negative gradient between the open site veins and the right atrium can be decreased by increasing right atrial pressure via leg elevation and using the “flex” option on the operating table control [8].

Avoidance of Nitrous Oxide:

Studies have illustrated that in the presence of VAE, anesthesia with inhaled nitrous oxide in oxygen–air permits lower volumes of delivered venous gas to more rapidly exacerbate the hemodynamic effects of the embolism. Nevertheless, nitrous oxide can dramatically increase the size of the entrained volume of air. Although without uniform consensus, there is ample data to discourage the use of nitrous oxide in any high-risk case. In moderate- or low-risk procedures, the benefits of this agent should be weighed against the possible risks, and appropriate monitoring should be used [8,42].

Central venous catheter:

Central venous catheters are often positioned in the right atrium during surgery to remove air that may be introduced elsewhere in the venous system. It is possible for an air embolism to develop during this central venous application. Therefore, the anesthesiologist should be aware during catheter insertion to prevent more air that might be introduced in to the venous system [26]. Trendelenburg position is common during the insertion of central venous catheters in the jugular or subclavian veins. Nonetheless, even using optimal positioning and techniques, air embolism has been reported [26].

Intra jugular balloon catheter:

An intra jugular balloon catheter was inserted to demonstrate that this device prevents air embolism. In an in vivo study, this device was bilaterally placed into jugular vessels in pigs. They found out that the intrajugular balloon catheter reliably prevented further air entry. Additionally, accumulated air could be aspirated from an orifice of the catheter. Bilateral inflation of the cuff significantly increased the proximal jugular venous pressure from9.8 mmHg to 14.5 mmHg. Under conditions mimicking an air embolism, air passage across the inflated cuffs was prevented and 78 (20%) of the air dose could be aspirated by the proximal orifice of the catheter [43].

Carbon dioxide field flooding

In order to reduce the hemodynamic disturbances of VAE, Pierluigi et al. changed the composition of the surgical field air partially replacing nitrogen with carbon dioxide (CO2) that dissolves better in human tissues. The idea of changing the atmosphere air composition using CO2 is because this gas is 25 times more soluble in tissues and blood than air and can rapidly be discharged through the exhaled air. They concluded that CO2 enriched sitting position surgical microenvironment significantly reduces the hemodynamic effects of VAE. Arterial CO2 emboli are more soluble and consequently much better tolerated than air emboli due to the higher solubility of CO2 in blood and tissues [21].

VAE treatment and management

The first goal in treatment of VAE is the prevention of further air entry and, if possible, a reduction in the volume

of air entrained. When there is clinical suspicion of air embolism, a number of initial steps should be taken quickly to manage the situation.

The treatment of VAE is aimed at stopping the air inflow in to the circulation and managing any complication that may be arise. When VAE is suspected the surgeon should be immediately notified to flood the field with saline and cover the surgical field with saline-soaked dressings. The surgeon should then locate and eliminate the entry site while the anesthesiologist initiates resuscitative effort to control the situation. In case of sitting position, the head of the patient should be repositioned at the level of the right atrium whenever possible to eliminate the negative air pressure gradient, and if possible, place the patient in the left lateral decubitus position (Durant maneuver) so that the air bubbles move toward the right atrium, thus relieving the "air-lock" effect responsible for potentially catastrophic cardiopulmonary collapse. Transient bilateral jugular venous compression may be applied to increase cerebral venous pressure and to reduce the air inflow through the exposed venous sinuses. Nitrous oxide or any air/oxygen mixture should be discontinued. Inspired gas should be changed to oxygen100%. This increases the partial pressure of oxygen in the blood and tissues. High flow oxygen may also aid the reabsorption of nitrogen gas from the bubble into the blood and then in to the alveoli (nitrogen washout) thus reducing the size of the air embolus [44]. Clinical experience suggests that air may not clear rapidly after VAE and may remain susceptible to augmentation by nitrous oxide if reinstituted [8].

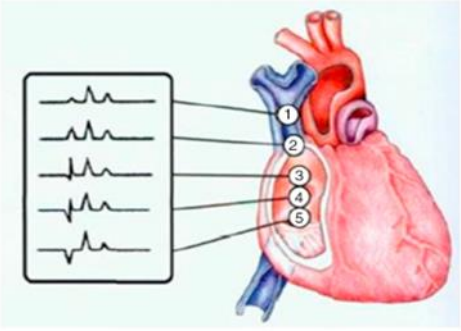
Hemodynamic support

Early consideration should be given to inotropic support of the right ventricle when VAE is suspected. Clinical VAE increases right ventricular afterload, resulting in acute right ventricular failure and a subsequent decrease in left ventricular output. Circulatory support should be commenced rapidly to increase venous pressure. These include administering fluids via large bore intravenous cannula as well as vasopressor or inotropic support as required such as dobutamine or epinephrine to improve cardiac output. Jardin et al. [45] used dobutamine in patients with VAE induced hemodynamic dysfunction and reported an increased cardiac index and stroke volume while decreased pulmonary vascular resistance. Archer et al. [46] described ephedrine for management of VAE. Norepinephrine has also been studied in the management of hypotension secondary to VAE in a canine model [8].

Aspiration of the air from the right atrium

For cases with a high incidence of VAE such as seated craniotomy and possibly seated cervical laminectomy or any neurosurgical procedure in the sitting position the right atrial catheter should be inserted prior to surgery. The best available device probably is the Bunegin-Albin multi-orifice catheter with a success rate of 30–60% [8]. The optimum site of CVP tip is 2cm below the junction with superior vena cava. This position should be checked by chest x-ray or ECG Technique [47]. When P wave amplitude peaks, the catheter tip is in the distal segment of the vena cava and when the P waveform become negative, the catheter has passed the vena cava and entered the right atrium (Figure1). The catheter should be positioned at the point where a large negative P is first obtained [13]. Surveys show the catheter will not effectively aspirate the air unless the catheter orifice is situated in the air lock within the right atrium. If a pulmonary artery catheter is in situ, it is improbable to be effective in aspirating air because of the catheter orifices are small and the catheters would not be in the optimal position for aspiration by the nature of their intended use. To aspirate an air embolism, the Trendelenburg and left lateral decubitus positions are advocated to be most effective positions in aspirating air. There are case reports, describing withdrawing 15 ml of air from the right heart percutaneously in a case of VAE resulting in prompt hemodynamic improvement. 15–20 ml of air has been the average amount that has been reported aspirated with a variety of devices during the past several decades.

Figure1- P wave morphology changes according to the position of the CVC tip [47].



Cardiopulmonary Resuscitation and Chest Compressions

In the case of an unresponsive patient, the first priority is a cardiopulmonary resuscitation (CPR) when necessary. Initiation of cardiopulmonary resuscitation with defibrillation and chest compression has presumptively demonstrated efficacy for massive VAE that results in cardiac standstill [48]. In the sitting position, there is usually access to the thorax to allow defibrillation but chest compressions will not be possible. Therefore, the patient should be placed in the supine position. Performing CPR on a patient with an open wound, who requires CPR, with or without the need to be turned into the supine position, should have any instruments removed to prevent accidental tissue injury. Protect the wound with a saline-soaked swab and then cover it with an adhesive dressing. There are no reports of cerebral injury caused by CPR in Resuscitation 11 August 2014 Management of cardiac arrest during neurosurgery in adult patients with an open cranium. However, following successful resuscitation, control of bleeding from the surgical site, particularly a posterior one, may be problematic. Even without need for cardiopulmonary resuscitation, the rationale behind closed-chest massage is to force air out of the pulmonary outflow tract into the smaller pulmonary vessels, thus improving forward blood flow. In canine studies, cardiac massage has been shown to be equally beneficial as left lateral positioning and intracardiac aspiration of air [8].

Hyperbaric oxygen therapy (HBOT)

Hyperbaric oxygen therapy plays a key role in the treatment of air embolism especially in the presence of cerebral arterial gas embolism. This intervention is widely regarded as the gold standard of VAE treatment [44,49]. HBOT is breathing of oxygen in a pressurized chamber in which the atmospheric pressure is increased up to three

times higher than normal. In these conditions, our lungs can collect up to three times of possible breathing oxygen at normal air pressure. The size of the air bubble is inversely proportional to the atmospheric pressure. It can reduce gas volume, cerebral edema and enhances partial pressure of dissolved oxygen in the blood [geebmusder]. Currently, HBOT is not administered routinely to all patients with air embolism, as it may not always be indicated and its appropriateness must be assessed, keeping in mind the potential detriments involved in administering it [50]. Evidence suggests that when indicated, it must be stressed that HBOT ideally is started within the first four to six hours after onset of neurologic symptoms, and also at any manifestation of end-organ damage, cardiopulmonary, or hemodynamic compromise [50]. Some studies indicate that HBOT treatment can still have a beneficial role up to 30 h after the initial event [51]. The decision to pursue delayed HBO has been dependent on the amount of air entrained and the persistence of clinical signs. HBO method has some contraindications especially in a patient with pneumothorax due to increased risk of gas embolism. It is also contraindicated in hyperthermia, acidosis, and epileptics due to increased risk of seizures. Chronic obstructive pulmonary disease, malignant tumours, pregnancy, claustrophobia, hereditary spherocytosis and optic neuritis are the other relative contraindications for the use of HBO therapy [52].

Results

Air embolism may occur in the venous system, arterial vasculature or paradoxically when air passes from right side of the heart to the left side and then enters in to the arterial circulation. VAE and paradoxical air embolism (PAE) are potentially serious neurosurgical complications and carry high neurologic, respiratory, and cardiovascular morbidity or even may be fatal. An intravascular air embolism is a rare and preventable event. Prevention is the optimal management of VAE. The most important measures to prevent VAE is to avoid injury to the venous system in the first place [31]. Good hydration, bilateral compression of jugular veins or Inflatable neck tourniquet, intrajugular balloon catheter, and medical antishock trousers, as well as avoidance of N2O are the best measures to prevent vascular air embolism. Furthermore, the use of moderate PEEP is to improve the alveolar oxygenation not to prevent VAE [41]. It is imperative for anesthesiologists to be aware of the causes, diagnosis, treatments, and adoption of practice patterns of VAE that best lead to the prevention or treatment of this potentially fatal condition. Anesthesiologist can play an important role in detection and urgent treatment of VAE in case of occurrence. If sitting position (SP) is needed for a neurosurgical necessity it can be used with vigilant follow up throughout the procedure to detect any occurrence of VAE. The SP in an experienced team of surgeons and anesthesiologists seems to be safe and that despite the detection of VAEs devastating events seldom occur [53-54].

References

1. Tobias JD, Johnson JO, Jimenez DF, Barone CM, McBride DS. Venous air embolism during endoscopic strip craniectomy for repair of craniosynostosis in infants. Anesthesiology. 2001; 95(2):340-2.
2. Harrison E, Mackersie A, McEwan A, Facer E. The sitting position for neurosurgery in children: a review of 16 years' experience. Br J Anaesth. 2002; 88(1):12-7.
3. Aleksic V, Radulovic D, Milakovic B, Nagulic M, Vucovic D, Antunovic V, et al. A retrospective analysis of anesthesiologic complications in pediatric neurosurgery. Pediatr Anesth. 2009; 19(9):879-86.
4. Wei S-T, Chen DC. Catastrophic venous air embolism during craniotomy in the supine position: The bleeding pattern as a warning sign? J Craniofac Surg. 2013; 24(3):e228-e9.
5. Anan'ev E, Polupan A, Savin I, Goryachev A, Troitskiy A, Kolokol'nikov A, et al. Paradoxical air embolism resulted in acute myocardial infarction and massive ischemic brain injury in a patient operated on in a sitting position. Zh Vopr Neirokhir Im NN Burdenko. 2016; 80(2):84-92.
6. Porter J, Pidgeon C, Cunningham A. The sitting position in neurosurgery: A critical appraisal. Br J Anaesth. 1999; 82(1):117-28.
7. Felema GG, Bryskin RB, Heger IM, Saswata R. Venous air embolism from Tisseel use during endoscopic cranial vault remodeling for craniosynostosis repair: A case report. Pediatr Anesth. 2013; 23(8):754-6.
8. Mirski MA, Lele AV, Fitzsimmons L, Toung TJ. Diagnosis and treatment of vascular air embolism. Anesthesiology. 2007; 106(1):164-77.
9. Kaye AH, Leslie K. The sitting position for neurosurgery: Yet another case series confirming safety. World Neurosurg. 2012; 77(1):42-3.
10. Günther F, Frank P, Nakamura M, Hermann EJ, Palmaers T. Venous air embolism in the sitting position in cranial neurosurgery: Incidence and severity according to the used monitoring. Acta Neurochirurgica. 2017; 159(2):339-46.
11. Ramina R, Tatagiba M, Soares M. Tumors of the Jugular Foramen: Springer; 2017.
12. Giraldo M, Lopera LM, Arango M. Venous air embolism in neurosurgery. Colombian Journal of Anesthesiology. 2015; 43:40-4.
13. Palmon SC, Moore LE, Lundberg J, Toung T. Venousair embolism: A review. J Clin Anesth. 1997; 9(3):251-7.
14. Pronovost PJ, Wu AW, Sexton JB. Acute decompensation after removing a central line: Practical approaches to increasing safety in the intensive care unit. Ann Intern Med. 2004; 140(12):1025-33.
15. Hulst RA, Klein J, Lachmann B. Gas embolism: Pathophysiology and treatment. Clin Physiol Funct Imaging. 2003; 23(5):237-46.
16. Kapoor T, Gutierrez G. Air embolism as a cause of the systemic inflammatory response syndrome: A case report. Critical Care. 2003; 7(5):R98.
17. Sheasgreen J, Terry T, Mackey JR. Large-volume air embolism as a complication of augmented computed tomography: Case report. Can Assoc Radiol J. 2002; 53(4):199-201.
18. Albin MS. Textbook of neuroanesthesia with neurosurgical and neuroscience perspectives: McGraw-Hill; 1997.
19. Faberowski LW, Black S, Mickle JP. Incidence of venous air embolism during craniectomy for craniosynostosis repair. Anesthesiology. 2000; 92(1):20-.
20. Van Heerden P, Woods W, Sviri S. Air embolism-a case series and review. Crit Care Resusc. 2004; 6(4):271.
21. Longatti P, Marton E, Feletti A, Falzarano M, Canova G, Sorbara C. Carbon dioxide field flooding reduces the hemodynamic effects of venous air embolism occurring in the sitting position. Childs Nerv Syst. 2015; 31(8):1321-6.
22. Toung TJ, Rossberg MI, Hutchins GM. Volume of air in a lethal venous air embolism. Anesthesiology. 2001; 94(2):360-1.
23. Natal BL, Doty C. Venous air embolism. Medscape; 2012.
24. Adam Lea. Air embolism and anesthesia. Anesthesia Tortorial of the Week AOTW. 2016.
25. Balki M, Manninen PH, McGuire GP, El-Beheiry H, Bernstein M. Venous air embolism during awake craniotomy in a supine patient. Can J Anesth. 2003; 50(8):835.
26. Vesely TM. Air embolism during insertion of central venous catheters. J Vasc Interv Radiol. 2001; 12(11):1291-5.
27. Tedim R, Amorim P, Castro A. Clinical Monitoring and Automatic Detection of Venous Air Embolism. Encyclopedia of Information Science and Technology, Third Edition: IGI Global; 2015. p. 5515-22.
28. Brechner TM, Brechner VL. An audible alarm for monitoring air embolism during neurosurgery. J Neurosurg. 1977; 47(2):201-4.
29. Matjasko J, Petrozza P, Mackenzie C. Sensitivity of end-tidal nitrogen in venous air embolism detection in dogs. Anesthesiology. 1985; 63(4):418-23.

30. Stendel R, Gramm H-J, Schröder K, Lober C, Brock M. Transcranial Doppler ultrasonography as a screening technique for detection of a patent foramen ovale before surgery in the sitting position. Anesthesiology. 2000; 93(4):971-5.
31. Feigl GC, Decker K, Wurms M, Krischek B, Ritz R, Unertl K, et al. Neurosurgical procedures in the semisitting position: evaluation of the risk of paradoxical venous air embolism in patients with a patent foramen ovale. World Neurosurg. 2014; 81(1):159-64.
32. Gildenberg PL, O'Brien RP, Britt WJ, Frost EA. The efficacy of Doppler monitoring for the detection of venous air embolism. J Neurosurgery. 1981; 54(1):75-8.
33. Sale J. Prevention of air embolism during sitting neurosurgery. Anaesthesia. 1984; 39(8):795-9.
34. Lubnin A, Oskanova M. Prevention of air embolism in neurosurgical patients operated on in a sitting posture: A comparative study of 3 methods. Anesteziol Reanimatol. 1994: 22-6.
35. Navarro LHC, Bloomstone JA, Auler JOC, Cannesson M, Della Rocca G, Gan TJ, et al. Perioperative fluid therapy: A statement from the international Fluid Optimization Group. Perioper Med (Lond). 2015; 4(1):3.
36. Domaingue C. Anaesthesia for neurosurgery in the sitting position: A practical approach. Anaesth Intensive Care. 2005; 33(3):323-31.
37. Lateef F, Kelvin T. Military anti-shock garment: Historical relic or a device with unrealized potential? J Emerg Trauma Shock. 2008; 1(2):63-9.
38. Loubieres Y, Vieillard-Baron A, Beauchet A, Fourme T, Page B, Jardin F. Echocardiographic evaluation of left ventricular function in critically ill patients: Dynamic loading challenge using medical antishock trousers. CHEST. 2000; 118(6):1718-23.
39. Schmitt HJ, Hemmerling TM. Venous air emboli occur during release of positive end-expiratory pressure and repositioning after sitting position surgery. Anesth Analg. 2002;94(2):400-3.
40. Giebler R, Kollenberg B, Pohlen G, Peters J. Effect of positive end-expiratory pressure on the incidence of venous air embolism and on the cardiovascular response to the sitting position during neurosurgery. Br J Anaesth. 1998;80(1):30-5.
41. Zentner J, Albrecht T, Hassler W. Prevention of an air embolism by moderate hypoventilation during surgery in the sitting position. Neurosurgery. 1991; 28(5):705-8.
42. Junghans T, Böhm B, Meyer E. Influence of nitrous oxide anesthesia on venous gas embolism with carbon dioxide andhelium during pneumoperitoneum. Surg Endosc. 2000; 14(12):1167-70.
43. Eckle V, Neumann B, Greiner T, Wendel H, Grasshoff C. Intrajugular balloon catheter reduces air embolism in vitro and in vivo. Br J Anaesth. 2015; 114(6):973-8.
44. McCarthy CJ, Behravesh S, Naidu SG, Oklu R. Air embolism: practical tips for prevention and treatment. J Clin Med. 2016; 5(11):93.
45. Jardin F, Genevray B, Brun-ney D, Margairaz A. Dobutamine: a hemodynamic evaluation in pulmonary embolism shock. Critical Care Medicine. 1985; 13(12):1009-12.
46. Archer DP, Pash MP, Macrae ME. Successful management of venous air embolism with inotropic support. Can J Anaesth. 2001; 48(2):204-8.
47. Argoti-Velasco Y, Carrillo-Torres O, Sandoval-Mendoza R, Paez-Amaya W, CAhuantzi-Caballero X. Proper electrocardiography-guided placement of a central venous catheter. Revista Médica del Hospital General de México. 2016.
48. Ericsson JA, Gottlieb JD, Sweet RB. Closed-chest cardiac massage in the treatment of venous air embolism. N Engl J Med. 1964; 270(25):1353-4.
49. Eoh EJ, Derrick B, Moon R. Cerebral arterial gas embolism during upper endoscopy. A A Case Rep. 2015; 5(6):93-4.
50. McCarthy CJ, Behravesh S, Naidu SG, Oklu R. Air embolism: Diagnosis, clinical management and outcomes. Diagnostics. 2017; 7(1):5.
51. Blanc P, Boussuges A, Henriette K, Sainty J, Deleflie M. Iatrogenic cerebral air embolism: importance of an early hyperbaric oxygenation. Intensive Care Med. 2002; 28(5):559-63.
52. Mehta V, De A, Balachandran C. Hyperbaric oxygen therapy. Journal of Pakistan Association of Dermatology. 2016;19(3):164-7.
53. Dilmen OK, Akcil EF, Tureci E, Tunali Y, Bahar M, Tanriverdi T, et al. Neurosurgery in the sitting position: retrospective analysis of 692 adult and pediatric cases. Turk Neurosurg. 2011; 21(4):634-40.
54. Ganslandt O, Merkel A, Schmitt H, Tzabazis A, Buchfelder M, Eyupoglu I, et al. The sitting position in neurosurgery: indications, complications and results. a single institution experience of 600 cases. Acta Neurochir (Wien). 2013; 155(10):1887-93.