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Venous Air Embolism: A Case Report and Review

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VENOUS AIR EMBOLISM: A CASE REPORT AND REVIEW

by

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PERMISSION

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ABSTRACT

Title: Venous Air Embolism: A Case Report and Review

Background: A venous air embolism is a complication where air enters into the vasculature causing obstruction of blood flow. Ultimately, hemodynamics become compromised and death may result. The incidence and mortality rates appear to be very high; therefore, every practitioner should be made aware of the management.

Purpose: The purpose of this literature search is to provide a case report and provide an extensive review of the evidence for its management.

Process: A review of the literature was performed using PubMed and CINAHL databases from the University of North Dakota's Harley E. French Library of the Health Sciences. Each reference used was carefully selected and applied.

Results: Management of a vascular air embolism begins with prevention. Specific interventions such as avoiding the sitting position during surgery, providing adequate hydration, and avoiding positive end-expiratory pressure and the administration of nitrous oxide can all decrease the risk of a venous air embolism. A transesophageal echocardiogram is the most sensitive method of detection for a venous air embolism; however it also has disadvantages, invasiveness being one. Furthermore, a precordial doppler ultrasound is the most sensitive noninvasive form of monitoring, but its reliability is questionable. End-tidal carbon dioxide monitoring has been proven to be a simple standard intraoperative monitor that is the first response to a venous air embolism. Treatment goals are aimed at preventing further entry of air, reducing the amount of air entrained, and providing hemodynamic support. Interventions include administering vasopressors, positive inotropic agents, and volume expanding agents. Also, providing 100%

oxygen, eliminating nitrous oxide, and providing cardiac massage will help improve outcomes. Utilizing the Durant maneuver and a central venous catheter for air removal are controversial. Lastly, hyperbaric oxygen therapy has been proven to decrease neurological morbidities.

Implications: By utilizing specific interventions to prevent, obtain early detection, and manage a venous air embolism, morbidity and mortality may be reduced.

Keywords: Venous air embolism, vascular air embolism, gas embolism, carbon dioxide embolism, and management

Venous Air Embolism: A Case Report and Review

A venous air embolism (VAE) is a potentially fatal condition that occurs when air enters into the vasculature. Air may be entrained from any situation that involves the vasculature to be freely open with the environment and (Shaikh & Ummunisa, 2009) a pressure gradient exists. It is not a new phenomenon as it has been reported since the 19th century (Mirski, Lele, Fitzsimmons, & Toung, 2007). However, many cases of VAE are not reported because several incidences of VAE are subclinical and the different methods of detection vary in sensitivity. Therefore, the true incidence of VAE is unknown and may never accurately be verified (Mirski et al., 2007; Shaikh & Ummunisa, 2009). Studies have shown patients undergoing neurosurgery, especially posterior fossa surgeries, in the sitting position have the highest incidence of VAE. VAE is estimated to occur in 10 to 80% of those cases. In laparoscopic surgeries, it is estimated VAE may occur in up to 69% of patients, while the incidence of orthopedic and obstetric-gynecological surgeries are 57% and 11-97% respectively (Shaikh & Ummunisa, 2009). Lastly, with cervical laminectomies, approximately 10% of cases are complicated by a VAE (Rodgers, Dangel-Palmer, & Berner, 2000).

Mortality rates of a VAE have been shown to be quite high. In 2010, a case series described the outcomes of 119 patients with a VAE. Death was seen in 21% of those patients (Bessereau et al., 2010). Furthermore, recent data suggests the mortality rate of a VAE is 28% (Park, Kwon, & Kim, 2012).

Clearly, a VAE can be detrimental to one's health and is a complication of which every practitioner should be made aware. In the following pages, a case report of a healthy young female undergoing a laparoscopic hysterectomy that developed a VAE is presented. A review of

literature that covers the pathophysiology, clinical signs and symptoms, detection, prevention, and management of a VAE follow.

Purpose

The purpose of this independent project is to review a case report and provide anesthesia professionals with evidence-based research regarding the management of a VAE. By utilizing specific interventions to prevent, obtain early detection of, and manage a VAE, morbidity and mortality may be reduced. Thus, every practitioner should be made aware of and review the management for a VAE.

Case Report

A 45-year-old, 70 kg, 168 cm, Hispanic female presented for a total laparoscopic hysterectomy for treatment of uterine fibroids and abnormal uterine bleeding. She denied having any allergies. Her medical history included migraine headaches, dyslipidemia, uterine fibroids, intrauterine fetal death related to placental abruption, and disseminated intravascular coagulation related to placental abruption. Surgical history included three cesarean sections and two umbilical hernia repairs. There was no history of anesthetic complications. Current medications included norgestimate/ethinylestradiol, iron, acetaminophen, and a multivitamin.

The patient was considered an American Society of Anesthesiologists physical status level two with a Mallampati class II. Bilateral breath sounds were clear to auscultation and her cardiovascular system was found to be normal. Preoperative vital signs were: heart rate 64, blood pressure 122/81, respirations 12, pulse oximetry 97%, and temperature 36.8° Celsius.

The patient was given midazolam 2 milligrams (mg) intravenously (IV) in the holding room and then transported to the operating room where she was assisted onto the operating table.

Non-invasive monitors were applied which included: a finger pulse oximeter, a 5-lead electrocardiogram (EKG), and a blood pressure cuff.

The patient was pre-oxygenated via facemask at 6 liters per minute. Vital signs were: heart rate 89 with normal sinus rhythm, blood pressure 168/71, and pulse oximetry 100%. Fifty micrograms (mcg) of fentanyl, 50 mg of lidocaine, 180 mg of propofol, 5 mg of rocuronium, and 140 mg of succinylcholine were all given IV for induction. A 7.0 mm cuffed endotracheal tube (ETT) was placed by direct laryngoscopy utilizing a Miller two. After intubation, bilateral breath sounds were auscultated. End-tidal carbon dioxide ($ETCO_2$) monitoring was present and the ETT was secured in place. The patient was placed on the ventilator in synchronized intermittent mechanical ventilation (SIMV) mode with a respiratory rate of 10, a tidal volume of 500 milliliters (mL), and 4 centimeters of water (cm H_2O) of positive end-expiratory pressure (PEEP). Sevoflurane was initiated with end-tidal concentrations of 1.7 to 2.6 throughout the case. Inspired oxygen was titrated down to 70%. A nasopharynx temperature probe was inserted for temperature monitoring and bispectral index (BIS) monitor was applied to the forehead. Eight milligrams of dexamethasone and 4 mg of ondansetron were given IV for nausea prophylaxis. Furthermore, 2 grams of cefazolin was administered IV for a preoperative antibiotic.

The patient was repositioned in the lithotomy position, prepped, and draped. An upper Bair hugger was placed, 50 mcg of fentanyl and 30 mg of rocuronium were given IV just prior to incision. From anesthesia start time to incision, 30 minutes had elapsed. Two minutes after incision, insufflation of the abdomen began and there was an immediate drop in $ETCO_2$ from 32 mmHg to 15 mmHg and then to 0 mmHg. Additionally, there was a related drop in blood pressure, heart rate, and oxygen saturation, which were 44/30, 32, and 76% respectively. The

surgeon was notified and the abdomen was evacuated. Furthermore, the patient was taken off of the ventilator to be manually ventilated with 100% oxygen and 25 mg of ephedrine was administered IV. A code blue was called overhead and the patient was placed back on the ventilator with the previous settings and 100% oxygen.

The patient was managed medically with 0.5 mg IV of epinephrine and 0.5 mg IV of atropine. It was noted that the patient's heart rhythm had changed from sinus bradycardia to a wide complex ventricular tachycardia, which was treated with 1 gram of calcium chloride IV and 50 milliequivalents (mEq) of sodium bicarbonate IV. Seven minutes later the patient was found to be in asystole so cardiac chest compressions were initiated. Ten minutes had elapsed from the onset of symptoms of a VAE to the start of compressions. Chest compressions ceased after 2 minutes for a rhythm check in which the patient had a pulse and her heart rhythm was atrial fibrillation. The blood pressure and heart rate were noted to be 158/102 and 135 respectively. A total of 2 mg IV of epinephrine and 2 mg IV of atropine were given in 0.5 mg IV doses to support the patient's heart rate and blood pressure in the time frame of code blue being called to the end of chest compressions.

At this time, it was decided to cancel the surgery, so the drapes were taken down. However, upon doing so, approximately 300 mL of blood was found to be pooling from the patient's perineal area. The decision to emergently open the abdomen was clear. The patient was prepped and draped again and given an additional 2 mg IV of midazolam and 20 mg IV of rocuronium. Two additional intravenous catheters, an arterial line, and central line were all placed. Labs were drawn and showed a pH of 7.05, potassium of 3.5 mmol/L and hemoglobin of 9.0 g/dL. The patient received 3 units of packed red blood cells IV and 1 unit of fresh frozen plasma IV to achieve a hemoglobin level of 11.5 g/dL. Furthermore, an additional 150 mEq of

sodium bicarbonate was given IV in doses of 50 mEq to obtain a pH of 7.3. Twenty mEq of potassium was given IV to correct the potassium deficit. Blood pressure was supported throughout the case with a phenylephrine drip.

During the abdominal exploration, the endotracheal tube was witnessed to have pink, frothy sputum in it from pulmonary edema. Forty mg of furosemide was given IV. Throughout the remainder of the case, the patient remained unstable and required aggressive hemodynamic support. She remained in atrial fibrillation, BIS read 0, $ETCO_2$ remained in the mid 20's, and oxygen saturation in the low 90's. Ultimately, the patient's uterus was removed. During the three-hour case, she had received a total of 5,600 mL of intravenous fluids, 500 mL of 5% albumin, and had lost 900 mL of blood. She was taken to the surgical critical care unit with fixed pupils, ventilator dependent, and not following commands. Critical care service subsequently assumed responsibility. Within the next 36 hours, the patient was weaned from the ventilator and extubated. She showed left-sided weakness and was diagnosed as having a cerebral vascular accident in addition to a VAE. Six days later the patient was discharged from the hospital.

Literature Search

A review of the evidence was sought out with an online literature search using CINAHL and PubMed. Both search engines were accessed through the University of North Dakota's Harley E. French Library of the Health Sciences. Each search resulted in journal articles that were carefully examined and selected in order to provide evidence for the management of a VAE.

Databases were chosen based on recommendation of the two health databases that should be used as a starting point for researchers (Mateo & Foreman, 2014). "The two literature

databases considered essential are MEDLINE (PubMed) and CINAHL (Cumulative Index to Nursing and Allied Health Literature)" (Mateo & Foreman, 2014, p. 22). These databases were accessed to begin the research for a VAE.

The first search was conducted using CINAHL. The search words "embolism treatment" were used with the all text option. This yielded 7,116 results. The all text option was then switched to the title option to try to narrow the results. This produced 141 articles. The exploration was further refined by searching "embolism treatment" as a title AND "air" as an all text option. Five results came back, in which one article was chosen to be used. Upon researching that article's reference list, five more journal articles were added for utilization. These articles were found by typing in the title and authors of the journal article in the search field of PubMed.

PubMed was the next database chosen for the literature search. A total of four searches were led. First, all fields were searched for "venous air embolism." This returned 2,360 results. "Venous air embolism" was then searched as a title and this yielded 402 articles, in which 11 articles were saved. One journal article's reference list was searched and provided three additional journal articles by searching the title and authors in PubMed. The search was refined to "venous air embolism" as a title AND "gas" as a title. Six journal articles were shown in which none were found to be of use. Therefore, the final step in this search consisted of searching "venous air embolism" as a title AND "gas" as a title/abstract option. Thirty-three articles were found. Three articles were duplicates and the rest were not of any use.

The second search using PubMed was performed by using the terms "carbon dioxide embolism" in all fields. Nine hundred and ninety-one articles were found. "Carbon dioxide embolism" was then searched as a title/abstract. A total of 88 articles were found in which five

of those articles were saved for use. Furthermore, in the reference list of one of the journal articles, three additional journal articles were found by typing in the title and authors of the journal article in the search field of PubMed.

A third search was performed with PubMed using the terms "gas embolism" in all fields. This search resulted in 7,918. Therefore, the same terms were searched as a title/abstract to narrow the results to 880. Again, the same terms were searched but as a title. Four hundred and sixty-eight results were yielded and eight of the articles were used for this independent project.

The last search performed using PubMed used the terms "vascular air embolism" in all fields. One thousand eight hundred and sixty-eight results were found. Thus, the same terms were searched as a title and yielded 15 results. One journal article was saved for use; the reference list provided an additional article to use by searching PubMed.

After all searches were refined using the databases CINAHL and PubMed as well as browsing the reference list and locating those resources with PubMed, a total of 38 journal articles were saved. With this research, a review of these articles is presented in the following sections.

Review of Literature

Pathophysiology and Clinical Presentation

The main elements that make up the pathophysiology of a VAE and determine the severity include the volume of air entering the vasculature and the air entrainment rate (Mirski et al., 2007). In adults, the lethal volume of air remains unknown, but it is estimated to be from 200 to 300 mL or 3 to 5 ml/kg. However, patients with decreased cardiac function are more likely to have cardiac arrest with less air (Rodgers et al., 2000; Toung, Rossberg, & Hutchins, 2001). The rate the air enters the vasculature is also an important factor because the lungs can serve as a

reservoir to rid the gas. If the rate of air entrapment overcomes the amount of air the pulmonary system can eliminate in a given time, the air may cause blood flow obstruction and create fibrin clots creating more outflow blockage. It has been shown that 100 mL of air per second can be entrained through a 14-gauge needle with a pressure gradient of 5 cm H_2O (Mirski et al., 2007). Clearly, the lethal dose of air can be achieved very quickly.

The presenting signs and symptoms of a VAE are determined by the same elements that make up the pathophysiology, the volume and rate of the air entrainment. In a spontaneously breathing patient, tachypnea and gasping may be seen when 10% of pulmonary blood flow is obstructed with slow air entrainment. Unfortunately, the gasping reflex decreases right atrial pressure, which can enlarge the emboli (Palmon, Moore, Lundberg, & Toung, 1997). Animal studies indicate larger volumes of air entrapment are better tolerated with a paralytic because it will prevent this gasping (Adornator, Gildenberg, Ferrario, Smart, & Frost, 1978). When air is entrained rapidly, shallow and rapid respirations may be recognized followed by apnea or irregular respirations. Peak airway pressures will increase in patients who have controlled ventilation. Because pulmonary dead space is increased and blood flow is obstructed with large volumes of air, a decrease in $ETCO_2$ and oxygen saturation are seen along with cyanosis (Palmon et al., 1997; Park et al., 2012).

Additional signs and symptoms of a VAE include a slow increase in pulmonary artery pressures with slow air entrainment. As the air is absorbed in the pulmonary system, the pulmonary artery pressure will normalize. With a rapid bolus of air, pulmonary artery pressures increase rapidly (Palmon et al., 1997). A decreasing pulmonary artery pressure and increased central venous pressure from right-sided heart failure may indicate circulatory collapse (Mirski et al., 2007). Furthermore, chest pain and electrocardiogram changes are often accompanied. The

electrocardiogram changes can include tachycardia, bradycardia, asystole, atrioventricular blocks, and S-T segment changes from myocardial ischemia or right heart strain (Mirski et al., 2007; Palmon et al., 1997; Park et al., 2012). Lastly, a "mill-wheel" murmur may be auscultated as a late sign (Shaikh & Ummunisa, 2009). This murmur is a splashing sound due to the air in the cardiac chambers and great vessels (Muth & Shank, 2000).

Neurological symptoms present themselves by two different mechanisms. Cardiovascular collapse from obstruction, right-sided heart failure, or ischemia results in hypoperfusion to the brain. Altered mental status is an acute symptom, which may be followed by focal deficits leading to a coma. The second mechanism in which neurological symptoms can occur is through a cerebral air embolism. If a patent foramen ovale is present, air has a direct route to the cerebral vasculature. Any patients with postoperative neurological changes should have a VAE included in their working diagnosis (Mirski et al., 2007; Shaikh & Ummunisa, 2009).

Overall, death can occur from a VAE due to circulatory collapse from gas lock in the pulmonary circulation. Right ventricular outflow is impeded resulting in a decreased left atrial preload. Consequently, decreased cardiac output and hypotension occur (Muth & Shank, 2000; Palmon et al., 1997).

Detection

There are various methods for detecting a VAE. Some methods are standard while others are specific. Detection sensitivity of a VAE varies between methods. Currently, transesophageal echocardiogram (TEE) is the most sensitive method of detection as it has been shown to detect as little as 0.02 ml/kg of air (Mirski et al., 2007). "It allows both the ability to detect microemboli and also has the advantage of identifying paradoxical air embolism" (Palmon et al., 1997, p.

253). However, in order to use this method of detection, the transgastric inferior vena cava view should be utilized because administering fluids and medications causes gas bubbles to arise in the right atrium (Park et al., 2012). The disadvantages of using a TEE include invasiveness, it is expensive, it requires constant monitoring by the practitioner, it places the patient at risk for glottic injury, it has no alarm capabilities, and it is complex (Mirski et al., 2007; Palmon et al., 1997; Park et al., 2012).

Of the noninvasive methods of detecting a VAE, the use of a precordial doppler ultrasound is the most sensitive. It has the ability to detect as little as 0.05 ml/kg of air and has versatility in that it can be placed on either the left or right sternal border and may be used on either adults or children. However, the prone or lateral position, sound artifacts during cautery, and morbid obesity are the weaknesses of its use (Mirski et al., 2007). Furthermore, the effectiveness is questionable when comparing studies. Kim et al. (2009) observed a degree of a VAE in 100% of patients undergoing laparoscopic hysterectomies when using a TEE for detection. Conversely, in one study there was no detection of a VAE in 100 patients undergoing laparoscopic procedures when using a precordial doppler ultrasound (Wadhwa, Mckenzie, Wadhwa, Katz, & Byers, 1978). Additionally, a study of 61 patients undergoing laparoscopic cholecystectomy did not have any detection of a VAE using a precordial doppler ultrasound (Landercasper, Miller, Strutt, Olson, & Boyd, 1993). Clearly, there is some discrepancy with the use of a precordial doppler ultrasound when compared to a study that proved 100% of patients have a degree of VAE during laparoscopic procedures.

The use of a pulmonary artery catheter can help aid in the diagnosis of a VAE; however, it is considered insensitive for detecting air entrainment. A pulmonary artery catheter can only detect 0.25 ml/kg or greater. Obviously, this is inferior to the other methods previously

described and does not go without risk when being utilized. Therefore, its use solely for the purpose of VAE detection should not occur. However, if a patient would benefit from the use of a pulmonary artery catheter as a monitoring tool due to the physical status of the patient, its use for VAE monitoring is warranted (Mirski et al., 2007).

Another method for detecting a VAE is through the use of $ETCO_2$ monitoring, which is also a standard intraoperative monitor. "A change of 2 mmHg ET_{co2} can be an indicator of VAE" (Mirski et al., 2007, p.169). One would assume a decrease in $ETCO_2$ would be seen with a VAE related to blood flow obstruction and the increase in the pulmonary dead space. This occurrence proves true for the majority of cases and has been demonstrated as to be the initial response to a VAE (Park et al., 2012). Smith (2011) reports a case of a healthy 34-year-old female who underwent a dilation and curettage and developed a VAE. The patient's $ETCO_2$ was noted to be 0 mmHg. Additionally, a case was reported of a 69-year-old female who underwent a laparoscopic cholecystectomy and developed a VAE. The patient's $ETCO_2$ decreased from 40 mmHg to 7 mmHg and then to 0 mmHg (Cadis, Velasquez, Brauer, & Hoak, 2014). Conversely, there have been instances where an increase in $ETCO_2$ was seen perhaps due to the carbon dioxide (CO_2) dissolved in the blood. Hynes and Marshall (1992) found one patient had a rise in their $ETCO_2$ during their study of patients who developed a VAE as a complication from laparoscopic surgery. Additionally, Shulman and Aronson (1984) saw a rise in the $ETCO_2$ before it declined in a case study of healthy female undergoing a diagnostic laparoscopic procedure whom developed a VAE. Therefore, with any change in the $ETCO_2$, VAE should not be ruled out as a possible cause. This is especially true with high-risk procedures.

Lastly, using a precordial stethoscope to hear a "mill-wheel" murmur has been demonstrated. Hynes and Marshall (1992) report a "mill-wheel" murmur heard in 100% of

patients they observed to have possible VAEs. Conversely, detection of a VAE has been shown to be unreliable with this method. "A recent review of seven episodes of carbon dioxide embolism during laparoscopy revealed that a "mill-wheel" murmur was reported by fewer than half of the patients" (Park et al., 2012, p. 463). Although the utilization of a precordial stethoscope is cost effective and a simple noninvasive monitor, the effectiveness of its use to detect a VAE is controversial.

Prevention

The gold standard of management of a VAE is prevention. Therefore, recognizing high-risk patients and surgeries is vital to prevent a VAE. As mentioned previously, the incidence of a VAE is highest with neurosurgery patients, especially posterior fossa surgery, who assume the sitting position (Rodgers et al., 2000). Any surgery in which the surgical site is above the level of the heart creates a pressure gradient and places the patient at an increase risk for a VAE. These surgeries may include craniotomies, spinal surgeries, or shoulder surgeries (Mirski et al., 2007). Clearly, avoiding this position would reduce the risk. "Alternative positioning such as prone or "park bench" provides adequate surgical conditions" (Mirski et al., 2007, p. 170). However, if the sitting position needs to be performed, decreasing the pressure gradient by increasing the right atrial pressure will decrease the risk for a VAE. This can be achieved by increasing venous return to the right side of the heart by elevating the patient's legs (Mirski et al., 2007).

Another position that appears to have an increased incidence of VAE is the left uterine displacement position for women undergoing a cesarean section (Mirski et al., 2007). A study was conducted of 207 women undergoing cesarean delivery. The results showed a 5° reverse Trendelenburg's position significantly decreased (44% to 1%) the occurrence of a VAE

compared to the horizontal position (Fong, Gadalla, & Druzin, 1991). Therefore, it is recommended the patient "should be placed in a 5° reverse Trendelenburg's position with left uterine displacement unless there is some medical contraindication" (Fong et al., 1991, p. 194).

Hydration is a vital factor that plays an important role in the development of a VAE. Patients with decreased central venous pressure (CVP) have been shown to have an increased incidence of VAE. This is related to the negative pressure gradient that exists (Domaingue, 2005). Therefore, increasing the right atrial pressure (CVP) will decrease the risk of a VAE. One study was performed on pigs undergoing laparoscopic surgery. It was concluded that by increasing the CVP above the intraperitoneal pressure, the risk for a VAE was reduced (Schmandra, Mierdl, Bauer, Gutt, & Hanisch, 2002). Additionally, there was a study of 16 patients undergoing laparoscopic nephrectomy where gas embolism was detected by TEE in only 1 patient. The authors attribute an increased CVP through fluid replacement as a possible risk reducer (Fahy, Hasnain, Flowers, Plotkin, Odonkor, & Ferguson, 1999). Likewise, fluid loading can decrease the risk of paradoxical embolism since fluid loading increases both right and left atrial pressures. Data suggests right atrial pressure exceeds left atrial pressure during the time of a paradoxical air embolism; increasing left atrial pressure with volume loading will decrease this risk (Colohan, Perkins, Bedford, & Jane, 1985). One study researched 20 patients undergoing surgery in the sitting position. The authors concluded that, "perioperative intravenous volume loading can decrease the number of cases in which right atrial pressure exceeds pulmonary capillary wedge pressure, and thus reduce the risk of paradoxical air embolism during seated neurosurgical operations" (Colohan et al., 1985, p. 842). Unfortunately, there is no set standard for how high the CVP should be increased. However, Palmon et al. (1997) suggests a CVP of 10 to 15 mmHg as goal.

A debatable subject about the prevention of a VAE is the use of positive end-expiratory pressure (PEEP). Concerning a paradoxical air embolism, where right atrial pressure exceeds left atrial pressure during the time of the embolism, two studies were conducted but yielded different results (Colohan et al., 1985). The first study concluded that when PEEP is utilized, up to 10 cm H_2O, right atrial pressure exceeded left atrial pressure, consequently, placing the patient at risk (Perkins & Bedford, 1984). However, a study published 4 years later concluded PEEP up to 10 cm H_2O increased both right and left atrial pressures, and therefore, did not increase the risk of paradoxical air embolism (Zasslow, Pearl, Larson, Silverberg, & Shuer, 1988). Furthermore, there are recent studies to support the use of PEEP. Schmitt and Hemmerling (2002) conducted a study of 18 patients in which 10 patients had developed a VAE when PEEP was released. Lastly, there was a study of 16 patients undergoing laparoscopic nephrectomy where gas embolism was detected by TEE in only 1 patient. The authors attribute the use of PEEP as a possible risk reducer (Fahy et al., 1999). PEEP is recommended to be used to improve oxygenation; however, it should not be used to reduce the risk of embolism (Mirski et al., 2007).

It is well known nitrous oxide is 34 times more soluble than nitrogen, which occupies air-containing spaces. That space will expand, if able, when nitrous oxide is administered. Consequently, if a VAE occurs and nitrous oxide is being utilized, the VAE will expand because it contains nitrogen (Nagelhout & Plaus, 2014). A study was conducted on 20 pigs to prove this phenomenon. When carbon dioxide was given intravenously, all pigs were able to tolerate the rate. However, when nitrous oxide was added, over half of the pigs were unable to tolerate it (Junghans, Bohm, & Meyer, 2000). Additionally, a case study was presented where a 50 year-old patient underwent surgery in the sitting position and developed a VAE. One hundred and

forty minutes after the VAE, nitrous oxide was administered which resulted in a decreased $ETCO_2$, tachycardia, and hypotension. These finding are indicative of volume expansion from the nitrous oxide administration (Sibai, Baraka, & Moudawar, 1996). Mirski et al. (2007) concludes that nitrous oxide should be avoided with all high-risk surgeries, and with low or moderate risk surgeries the benefits need to outweigh the risks.

Management

As previously discussed, when a VAE occurs, detrimental hemodynamic effects may result. Specific interventions should be immediately implemented to help improve patient outcomes. "Principal goals of management where VAE is strongly suspected include prevention of further air entry, a reduction in the volume of air entrained, if possible; and hemodynamic support" (Mirski et al., 2007, p. 172). After the surgeon has been notified, insufflation should cease, if used, and all suspected routes of entry should be considered. The surgeon should cover any route suspected of entry with saline soaked gauze to prevent more air emboli (Palmon et al., 1997; Park et al., 2012). Rapid fluid volume expansion is recommended to elevate venous pressure in order to prevent more gas entrainment (Muth & Shank, 2000). If the patient is in the sitting position, evidence has shown jugular venous compression can help identify potential areas of entry and limit the amount of air entrainment. When using this maneuver, careful attention should be used to not compress the carotid arteries. Also, increased intracranial pressure may be a contraindication (Losasso, Muzzi, & Cucchiara, 1992; Sale, 1984).

Ceasing of any nitrous oxide administration should occur as it increases the size of the VAE (Palmon et al., 1997). This will allow 100% of oxygen delivery, causing washout of nitrogen and carbon dioxide, thus, decreasing the size of the embolism. Also, 100% oxygen will

help improve hypoxemia from the ventilation perfusion mismatch (Park et al., 2012; Shaikh & Ummunisa, 2009).

Positioning is an intervention that can be used to reduce the obstruction if air lock occurs. Some evidence suggests placing the patient in the left lateral decubitus position (Durant maneuver). A study by Durant, Oppenheimer, Lynch, Ascanio, and Webber (1954) found that:

> In the left-side-down position the amount of air in the pulmonary artery either fluctuated or progressively decreased in animals which recovered, and was generally reciprocally related to the amount of air in the right atrium and ventricle. In the latter chambers, the air was trapped away from the outflow tract of the right ventricle, so that continuity of blood column from the cavae to the pulmonary artery was maintained. This evidently permitted circulation to continue until the air could be eliminated from the right heart by contraction or by inspiratory aspiration or both. (p. 519)

Conversely, recent animal studies indicate the Durant maneuver to be ineffective. A similar study was performed in canines with results that demonstrated no improvement in hemodynamic performance (Geissler, Allen, Mehlhorn, Davis, Morris, & Butler, 1997). Undoubtedly, there is conflicting data in which a conclusion cannot be drawn.

Another intervention that can be performed at the time of a VAE is air retrieval through a central venous catheter in order to reduce the obstruction. Case studies have found it to be successful. One case report stated they retrieved several mL of air and then an additional 12 mL of air, in which the patient survived (Herron, Vernon, Gryska, & Reines, 1999). Another case report stated they retrieved 8 mL of air and again this patient survived with this intervention (Burcharth, Burgdorg, Lolle, & Rosenberg, 2012). Lastly, a study indicated 2 to 20 mL of air could be aspirated from a central venous catheter. In this study, the researchers looked at 100

patients who developed a VAE. Hemodynamics improved with aspirating the air embolism; however, other interventions were being performed at the same time as the aspiration so full credit cannot be given to the air retrieval intervention (Bedford, Marshall, Butler, & Welsh, 1981).

Many animal and lab studies indicate unreliable amounts of air can be aspirated which may not be significant enough when compared to the amount of air that can be entrained in just a few seconds. Swan-Ganz catheters have success rates between 6 and 16%. The best device was the Bunegin-Albin multiorfice catheter with success rates as high as 30 to 60% (Colley & Artru, 1989; Hanna, Gravenstein, & Pashayan, 1991; Mirski et al., 2006; Palmon et al., 1997; Park et al., 2012). In dogs, 9 to 53% of air was shown to be able to be retrieved (Sink, Comer, James, & Loveland, 1976). However, during a hysteroscopy, a woman was noted to have developed a VAE. At that time, a central venous catheter was placed under echocardiography, which showed air bubbles in the right ventricle; after several attempts of air retrieval, it was unsuccessful (Nishiyama & Hanaoka, 1999). Because evidence is variable, recommendations are to not use a central venous catheter for aspirating air during a VAE as a first line treatment. If a central venous catheter is already present, then its use is warranted.

Cardiopulmonary resuscitation, sometimes-requiring defibrillation, and hemodynamic support are required to maintain oxygenation and perfusion to vital organs during the event of a VAE. With cardiac obstruction, chest compressions have been proven to be effective in relieving the gas lock. The logic behind this intervention is that closed cardiac massage will force the air into smaller vessels, thus, relieving the gas lock and allowing the continuity of blood flow (Mirski et al., 2007). Additionally, with the use of positive inotropic agents, an increase in cardiac output may be achieved. This is achieved by increasing cardiac contractility and

decreasing pulmonary vascular resistance (Palmon et al., 1997). The following case report demonstrates these successful interventions. A 68-year-old female undergoing a hysteroscopy developed a VAE. Cardiac massage and positive inotropic agents were initiated and continued until the return of spontaneous circulation (Nishiyama & Hanaoka, 1999). A similar case study demonstrates the same results. A 28-year-old female underwent a laparoscopic tubal ligation. After insufflation, the patient's $ETCO_2$ decreased to 16 mmHg and then to 0 mmHg. Also, a drop in blood pressure and ventricular tachycardia were noted. The patient was suspected of having a VAE. Chest compressions were initiated and continued for 5 minutes and dopamine was administered IV until return of spontaneous circulation was seen (Duncan, 1992). Other pharmacological agents used for the management of a VAE include prostaglandin analogues or phosphodiesterase inhibitors to treat pulmonary hypertension, and vasopressors to support blood pressure (Park et al., 2012).

Another intervention that has been shown to be effective for the treatment of a VAE is hyperbaric-oxygen therapy (HBO). This seems to be most helpful when neurological symptoms are present. Park et al. (2012) states:

> Hyperbaric compression reduces bubble size (one-third of the original volume at three atmospheres), restores blood flow, and limits detrimental effects of the gas-blood interface. Other potential beneficial effects include a reduction in intracranial pressure and increased tissue oxygenation via diffusion. (p. 464)

With this therapy, patients inspire 100% oxygen at pressures above atmospheric pressure. Patients are able to achieve an arterial partial pressure of oxygen greater than 2000 mmHg. However, the timing for initiation of HBO therapy is not well understood (Muth & Shank, 2000).

Discussion

The patient in this independent project was undergoing a gynecologic laparoscopic procedure, which places her at risk for developing a VAE from the carbon dioxide insufflation. Also, the patient was mechanically ventilated with PEEP. This, too, places the patient at an increased risk of VAE. As discussed, utilizing a paralytic will prevent the gasping reflex, therefore, preventing more air entrainment. This was a positive intervention for the patient. Additionally, no nitrous oxide was used which increased the patient's chances for survival. However, the patient's fluid status was probably not optimized as she had been fasting throughout the night and fluid replacement did not begin until the beginning of the surgery.

$ETCO_2$ monitoring was utilized during this case because it is a standard intraoperative monitor. No other avenues of VAE detection were utilized because of the healthy status of the patient. When the patient developed the VAE, the $ETCO_2$ decreased immediately, including the blood pressure, heart rate, and oxygen saturation. These symptoms coincided with the evidence presented in this independent project. Furthermore, the heart rhythm changed to sinus bradycardia, then to wide complex ventricular tachycardia, then asystole, followed by atrial fibrillation. This, too, was found in the evidence presented. Neurological changes were seen as the patient was diagnosed with a cerebral vascular accident. Evidence states this can occur.

Treatment for this patient included administering 100% oxygen, providing hemodynamic support, giving external cardiac massage, and administering fluid for volume expansion. All of these interventions were important for the patient's positive outcome and are supported by the evidence. Auscultation was not attempted for a "mill-wheel" murmur, as this is a late sign and shown to be unreliable. The Durant maneuver also was not performed. This too is a controversial intervention. Air retrieval was not attempted. Evidence shows this is an unreliable

intervention. However, a central line was placed emergently for rapid fluid administration. Overall, the management of this patient's VAE is supported by evidence. Perhaps, these interventions can be attributed to her survival.

Conclusion

A VAE is a major complication where air enters into the vasculature due to an existing pressure gradient. Ultimately, hemodynamics become compromised due to right ventricular outflow obstruction, or gas lock. The severity of the VAE is dependent on the rate and amount of air entrainment. Early detection can help aid in the management of a VAE. A transesophageal echocardiogram is the most sensitive method of detection for a venous air embolism but it is invasive in nature. A precordial doppler ultrasound is the most sensitive noninvasive form of monitoring, however, its reliability is questionable. Initial response of a VAE occurs with end-tidal carbon dioxide monitoring, which is a simple standard intraoperative monitor.

Management of a vascular air embolism begins with prevention. Specific interventions such as avoiding the sitting position during surgery, providing adequate hydration in order to increase the right atrial pressure, avoiding positive end-expiratory pressure and the administration of nitrous oxide can all decrease the risk of a venous air embolism. Treatment goals are aimed at preventing further entry of air, reducing the amount of air entrained, and providing hemodynamic support. Treatment interventions include administering vasopressors, 100% oxygen, and fluid volume expanding agents. Also, administering positive inotropic agents and closed cardiac massage will help improve outcomes by supporting the forward flow of blood. Research indicates, utilizing the Durant maneuver and a central venous catheter for air removal is controversial. Lastly, hyperbaric oxygen therapy has been shown to decrease

neurological morbidities, but the timing for initiation is unclear. Overall, many recommended interventions for the management of VAE are supported by evidence. If these are implemented, good patient outcomes will result.

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Venous Air Embolism: A Case Report and Review

Jared R. Onder, SRNA

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Introduction

- A Venous Air Embolism (VAE) is a potentially fatal complication where air enters into the vasculature causing obstruction of blood flow (gas lock).
- Severity is related to:
  - Volume of air entrained
  - Air entrainment rate

Mirski, Lele, Fitzsimmons, & Toung, 2007

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Introduction Cont'd

- Incidence:
  - Neurosurgery (Sitting Position): 10-80%
  - Laparoscopic Surgeries: Up to 69%
  - Orthopedic Surgeries: Up to 57%
  - Cervical Laminectomies: Approximately 10%
- Mortality: Up to 28%

Rodgers, Dangel-Palmer, & Berner, 2000; Shaikh & Ummunisa, 2009; Park, Kwon, & Kim, 2012

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Case Information

- Total Laparoscopic Hysterectomy
- 45 year-old
- 70 Kg
- 168 cm
- Female
- ASA 2
- No Known Allergies

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Pre-operative Evaluation

- Past Medical History:
  - Migraine headaches, dyslipidemia, uterine fibroids, intrauterine fetal death related to placental abruption, and disseminated intravascular coagulation (DIC) related to placental abruption
- Surgical History:
  - Cesarean section X 3 and umbilical hernia repair X 2

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Pre-operative Evaluation Cont'd

- Pre-op VS:
  - Heart rate 64, blood pressure 122/81, respirations 12, pulse oximetry 97%, and temperature 36.8° Celsius
- Mallampati class II

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Anesthetic Course

- Holding Room Drugs:
  - 2 mg Midazolam IV
- Induction Drugs:
  - 50 mcg Fentanyl IV, 50 mg Lidocaine IV, 180 mg Propofol IV, 5 mg Rocuronium IV, 140 mg Succinylcholine IV
- Other Drugs:
  - 4 mg Ondansetron IV, 8 mg of Dexamethasone IV, 2 g Cefazolin IV
- Airway:
  - 7.0 mm cuffed endotracheal tube
- Ventilation:
  - Synchronized intermittent mechanical ventilation, respiratory rate of 10, 500 mL tidal volume, and 4 centimeters of water of positive end-expiratory pressure (PEEP)
- Sevoflurane end-tidal concentrations of 1.7 to 2.6 throughout the case
- Lithotomy Position
- Prior to Incision:
  - 50 mcg Fentanyl IV and 30 mg of Rocuronium IV

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Intraoperative Issues

- Pneumoperitoneum Established:
  - VS: $ETCO_2$ from 32 mmHg to 15 mmHg and then to 0 mmHg, heart rate 32, blood pressure 44/40, oxygen saturation 76%
- Abdomen evacuated, manual ventilation, 25 mg Ephedrine IV given
- Code Blue:
  - EKG: bradycardia - wide complex ventricular tachycardia - asystole
  - Drugs: 2 mg Epinephrine IV in 0.5 mg doses, 2 mg Atropine IV in 0.5 mg doses, 1 g Calcium Chloride IV, and 50 mEq Sodium Bicarbonate IV
- Return of Spontaneous Circulation:
  - VS: Blood pressure 158/102, heart rate 135

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Intraoperative Issues Cont'd

- Perineal blood found (300 mL) - decision to open:
  - 2 mg Midazolam IV, 20 mg Rocuronium IV
  - 2 IV catheters, arterial line, and central line placed
- Labs: pH 7.05, potassium 3.5 mmol/L, Hgb 9.0 g/dL
- Treatment: 3 units PRBCs, 1 unit FFP, 150 mEq Sodium Bicarbonate IV in 50 mEq doses, 20 mEq Potassium IV
- Repeat Labs: pH 7.3, Hgb 11.5
- Pulmonary edema treated with 40 mg furosemide IV
- Blood pressure supported with Phenylephrine drip

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Intraoperative Issues Cont'd

- 3 Hour Surgery:
  - Patient remained in atrial fibrillation
  - BIS: 0
  - $ETCO_2$ remained in the mid 20's
  - Oxygen saturation in the low 90's
  - 5,600 mL of intravenous fluids
  - 500 mL of 5% albumin
  - EBL 900 mL

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Intensive Care Unit

- Transfer to ICU:
  - Fixed Pupils
  - Ventilatory dependent
  - Not following commands
- 36 Hours Later:
  - Weaned from ventilator and extubated
  - Left-sided weakness due to a CVA
- Discharged home 6 Days Later

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Detection

- Transesophageal Echocardiogram (TEE):
  - **Most sensitive method** of detection (0.02 ml/kg of air)
- Precordial Doppler Ultrasound:
  - Most sensitive noninvasive method of detection (0.05 ml/kg of air)
  - Versatile with both adults and children
  - Questionable effectiveness
- Pulmonary Artery Catheter:
  - Considered insensitive for detecting air entrainment (0.25 ml/kg or greater)
  - Best used for a patient who would benefit from its use as a monitoring tool due to their physical status

Kim et al., 2009; Landercasper, Miller, Strutt, Olson, & Boyd, 1993; Mirski et al., 2007; Palmon et al., 1997; Park et al., 2012; Wadhwa, Mckenzie, Wadhwa, Katz, & Byers, 1978

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Detection Cont'd

- $ETCO_2$ Monitoring:
  - A change of 2 mmHg can be an indicator of VAE
  - A decrease in $ETCO_2$ is the **initial** response to a VAE
  - Most cases will show a decreased value with VAE; however, an increased value may also indicate VAE
- Precordial Stethoscope:
  - Used to auscultate a "mill-wheel" murmur
  - Effectiveness is controversial

Cadis, Velasquez, Brauer, & Hoak, 2014; Hynes & Marshall, 1992; Mirski et al., 2007; Park et al., 2012; Shulman & Aronson, 1984; Smith, 2011;

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Prevention

- Prevention is Key!
- Identify high risk patients:
  - Sitting Position:
    - **Surgical site above the level of the heart**
    - If used, increase right atrial pressure by elevating the patient's legs
  - Left Uterine Displacement Position:
    - If used, place patient in a 5° reverse Trendelenburg's position with left uterine displacement

Fong, Gadalla, & Druzin, 1991; Mirski et al., 2007; Rodgers et al., 2000

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Prevention Cont'd

- Adequate Hydration:
  - Patients with decreased central venous pressure (CVP) have increased risk of VAE
  - CVP Goal: 10-15 mmHg
- Use of PEEP:
  - Controversial, recommended to use PEEP to improve oxygenation, not for VAE risk reduction
- Avoid Nitrous Oxide:
  - 34 times more soluble
  - Expands the volume of a VAE

Colohan, Perkins, Bedford, & Jane, 1985; Domaingue, 2005; Fahy, Hasnain, Flowers, Plotkin, Odonkor, & Ferguson, 1999; Junghans, Bohm, & Meyer, 2000; Mirski et al., 2007; Nagelhout & Plaus, 2014; Palmon et al., 1997; Perkins & Bedford, 1984; Schmandra, Mierdl, Bauer, Gutt, & Hanisch, 2002; Schmitt and Hemmerling, 2002; Sibai, Baraka, & Moudawar, 1996; Zasslow, Pearl, Larson, Silverberg, & Shuer, 1988

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Management

- Goals: Prevent Further Air Entry, Reduce the Volume of Air Entrained, and Provide Hemodynamic Support-
  - Stop Insufflation
  - Cover any suspected site with saline soaked gauze
  - Rapid fluid volume expansion
  - Cease the use of any nitrous oxide
  - Left lateral decubitus position (Durant Maneuver)
    - Controversial

Durant, Oppenheimer, Lynch, Ascanio, and Webber, 1954; Geissler, Allen, Mehlhorn, Davis, Morris, & Butler, 1997; Losasso, Muzzi, & Cucchiara, 1992; Mirski et al., 2007; Muth & Shank, 2000; Palmon et al., 1997; Park et al., 2012; Sale, 1984; Shaikh & Ummunisa, 2009

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Management Cont'd

- Goals: Prevent Further Air Entry, Reduce the Volume of Air Entrained, and Provide Hemodynamic Support-
  - Air retrieval through a central venous catheter
    - Swan-Ganz: 6-16% Success
    - Bunegin-Albin multiorfice catheter: 30-60% success
    - Not a first line treatment!
  - Cardiopulmonary resuscitation and positive inotropic agents
    - Proven to relieve gas lock

Bedford, Marshall, Butler, & Welsh, 1981; Burcharth, Burgdorg, Lolle, & Rosenberg, 2012; Colley & Artru, 1989; Duncan, 1992; Hanna, Gravenstein, & Pashayan, 1991; Herron, Vernon, Gryska, & Reines, 1999; Mirski et al., 2006; Nishiyama & Hanaoka, 1999; Palmon et al., 1997; Park et al., 2012; Sink, Comer, James, & Loveland, 1976

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Recommendations

- TEE is the most sensitive method of detection
- Precordial doppler ultrasound is the most sensitive noninvasive form of VAE detection
  - Reliability is questionable
- Initial response of a VAE is a decrease in $ETCO_2$

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Recommendations Cont'd

- Management begins with prevention:
  - Avoid the sitting position if possible
  - Provide adequate hydration to increase right atrial pressure
  - Avoid PEEP
  - Avoid using nitrous oxide
- Treatment includes:
  - Administering vasopressors, 100% oxygen, and fluid volume expanding agents
  - Positive inotropic agents and closed cardiac massage will support forward flow of blood
  - Durant maneuver and air removal through a central venous catheter remains controversial

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Conclusion

- Many recommended interventions for the detection, prevention, and management of a VAE are supported by evidence. When used, good patient outcomes may result.

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Thank You
Are There Any Questions?

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