Management of Venous Air Embolism

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Introduction and Pathophysiology

Venous air embolism (VAE) is a potentially lethal complication that can occur occur during any surgical procedure in which the operative site is 5 cm or higher above the right atrium or gas is forced under pressure into a body cavity. Although VAE is typically described as a complication of neurosurgery, it can occur during procedures involving the head and neck, laparoscopic procedures, vaginal delivery and caesarean section, and spinal instrumentation procedures. VAE has been reported to occur during liver transplantation. Although many occurrences of VAE are asymptomatic, entrainment of large quantities of air can lead to cardiovascular collapse, severe neurologic injury, and death.

The incidence of VAE is highest for neurosurgical procedures in the sitting position; the incidence ranges from 10% for cervical laminectomy to 80% of patients undergoing craniotomy in the sitting position. The incidence of VAE in patients undergoing hip replacement surgery is approximately 30%. Laparoscopic surgery also carries the risk of gas embolism. Ten to 15% of patients undergoing laparoscopic cholecystectomy were found to have evidence of gas embolism on transesophageal echocardiography.

Intraoperative venous air embolism produces can range in severity from being asymptomatic and causing severe injury or death. The factors that determine the morbidity of an episode of VAE include the rate of air entrainment, the volume of air entrained, and the position of the patient at the time of the embolism. The lethal dose of intravascular air in humans is unknown, but accidental injections of between 100 and 300 ml have been fatal. The mechanism of death from massive air embolus is circulatory obstruction and cardiovascular collapse resulting from air trapped in the right ventricular outflow tract.

VAE that does not cause immediate death can cause paradoxical embolization by acutely increasing right atrial pressure resulting in right to left shunt through a patent foramen ovale. Pulmonary microvascular occlusion can also occur; the air can produce increasing obstruction to blood flow, undergo resorption, or result in increased dead space. Bronchoconstriction may result from release of endothelial mediators, complement production, and cytokine release. During spontaneous respiration, slow entrainment of air that causes obstruction of 10% of the pulmonary circulation causes a "gasp" reflex that results in chest pain and tachypnea. The resulting decrease in intrathoracic pressure and right atrial pressure can increase the rate of air entrainment.

Morbidity and mortality from air embolism are directly related to the size of the embolus and the rate of entry. Doses of air greater than 50 ml (1 ml/kg) cause hypotension and dysrhythmias. 300 ml of air entrained rapidly can be lethal. Bronchoconstriction results in increased airway pressure, and wheezing. Other manifestations of air embolism include hypoxemia, hypercapnia and decreased ETCO2 (due to increased functional dead space). Hypotension, cardiac dysrhythmias, and cardiovascular collapse occur as air entrainment

continues.

Monitoring

The methods of monitoring for VAE are listed in order of their sensitivity from most to least sensitive. The technique used should be chosen based on its invasiveness and the likelihood that VAE will occur during the planned surgical procedure. The patient's underlying medical condition and the invasiveness of the surgery also should be considered when making this decision.

A "four-chamber" view. The right atrium is in the upper left corner of the picture. The central venous catheter can be seen as a small dot in the center of the atrium.

Transesophageal echocardiography is the most sensitive monitoring technique for VAE, and is able to detect 0.02 ml/kg of air administered by a bolus intravenous injection. It is also expensive, more invasive, and more difficult to place and to interpret. It does, however, allow determination of the volume of air aspirated. Transesophageal echocardiography will also show air passing through a patent foramen ovale into the left atrium and into the systemic circulation.

Doppler ultrasound is also a fairly sensitive monitoring technique, and is commonly used during neurosurgical procedures that carry a risk of VAE. A properly positioned Doppler ultrasound probe can detect 0.25 ml of air. It has been recommended that the Doppler transducer be located over the right heart, along the right sternal border, although placement over the left sternal border may occasionally provide satisfactory detection of turbulence and heart sounds associated with VAE.

Another four-chamber view recorded during dissection of a vascular tumor. Air bubbles are visible as a "snow-storm" appearance in the right atrium. Note that no air is crossing into the left atrium. (The left atrium is above and to the right of the right atrium.)

The Doppler ultrasound monitor uses ultrahigh frequency sound waves (usually between 2 and 3 megahertz) to measure blood flow velocity and changes in blood density. This information is converted to a characteristic sound. it enters the pulmonary circulation. The sound produced by intravascular embolism is readily identifiable, even when the anesthesiologist is attending to other tasks in the operating room.

The Doppler ultrasound is does have some disadvantages. It is not quantitative; there is no way to determine the amount of intracardiac air by listening to the sounds produced. There is, therefore, no way to differentiate between a massive air embolism and a physiologically insignificant episode. Crystallized mannitol solutions or rapid injections of aqueous solutions may mimic intracardiac air. Placement of the transducer may be difficult on some patients, especially those with a chest wall deformity or who are obese. The Doppler does not function during electrocautery because of radio frequency interference, and is unable to detect air embolism during that time.

The pulmonary artery catheter is the next most sensitive monitor. Air entering the pulmonary circulation causes mechanical obstruction and reflex vasoconstriction due to pulmonary hypoxemia, resulting in increased pulmonary artery pressure. This allows detection of the embolus, and a determination of whether the procedure should continue. Because of the small lumen in the PAC and the fixed location of the ports, air aspiration may be difficult or impossible. Moreover, increased PA pressure is not specific for air. Because there are more sensitive and less invasive techniques for monitoring and treatment of VAE, use of the pulmonary artery catheter should not be considered routine.

Mass spectrometry for end-tidal nitrogen is as sensitive as the pulmonary artery catheter. It is highly specific for air, but is not available in many operating rooms. The concentration of

exhaled nitrogen is usually less then 2%, and is below the threshhold of some commercial mass spectrometers.

End-tidal carbon dioxide is a standard intraoperative monitor that is used in nearly every surgical procedure. It is not specific for air embolism, however. Hyperventilation, low cardiac output, other types of emboli, and COPD can also decrease end-tidal CO2. If carbon dioxide is being used for the detection of venous air embolism, the lower alarm limit should be set to two to three mmHg below the patient's baseline.

The least sensitive monitor is the precordial or esophageal stethoscope. A "millwheel murmur" indicates a massive air embolism. When a millwheel murmur is heard, cardiovascular collapse is imminent.

Prevention

Strategies for the prevention of VAE concentrate on decreasing the pressure gradient between the surgical site and the right atrium. Several studies suggest that the use of nitrous oxide does not increase the risk of venous air embolism. The head should be elevated only as much as necessary to obtain adequate exposure. The patient should be kept hydrated to increase CVP, which decreases the risk of embolism, and to increase LAP, which minimizes the risk of paradoxical embolism to the left side of the circulation. There is no "standard" right atrial pressure, although a pressure of 10 to 15 mmHg is reasonable. The surgeons should be meticulous about cauterizing and tying blood vessels and applying wax to the edges of the bone flap.

The use of mechanical ventilation with positive end-expiration pressure during surgery is controversial. The use of moderate amounts of PEEP has not been shown to increase central venous pressure in humans in the seated position. High levels of PEEP will increase CVP, but may also decrease cardiac output. Sudden loss of PEEP with air in the right side of the heart (as may happen if the endotracheal tube is disconnected from the anesthesia circuit) may result in a paradoxical air embolus in patients with a patent foramen ovale.

Treatment

The primary goals in the treatment of VAE are to prevent further air entry, reduce the volume of the entrained air, and support the cardiovascular system while air is resorbed.

The surgeon should be informed as soon VAE is suspected. The surgeon should flood the surgical field with irrigating solution, cauterize open blood vessels, and apply bone wax to exposed edges. FiO2 should be increased to 1.0. N2O diffuses into air bubbles faster than nitrogen can diffuse out, and increases the size of the bubble. If N2O is used, it therefore should be discontinued when an air embolism occurs. If a multiorifice central venous catheter has been placed in the right atrium, are can be aspirated through the catheter. The optimal site for the tip of the catheter is at the SVC-RA junction.

If significant amounts of air have entered the circulation, the jugular veins can be manually occluded. This will prevent additional air from being entrained while the surgeons obtain hemostasis. The blood pressure should be supported by administering fluids. Pharmacologic support should include inotropic drugs such as epinephrine.

If possible, the operative site should be positioned below the level of the heart. This can be done by tilting the table into the Trendelenberg position. This will increase venous pressure at the operative site and reduce air entrainment. If a large volume of air has been entrained, and surgical conditions permit (i.e., the head is not in pins), positioning the patient in the left lateral decubitus position will help to keep air in the right atrium from entering the ventricle.

The right atrial catheter should be aspirated until no more air can be obtained.

References

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