

Gas embolism in anaesthesia

S Webber FRCA

J Andrzejowski FRCA

G Francis FRCA

Gas embolism is essentially an iatrogenic clinical problem caused by the ingress of gas into the vascular system. In this article, the term 'air embolism' will be used since air is by far the commonest culprit in most clinical situations. However, embolism following the use of other gases such as carbon dioxide (laparoscopy) and nitrous oxide may also occur.

The majority of clinical problems are caused by gas entering the **venous** system resulting in venous air embolism (VAE). **Arterial** air embolism (AAE) can also occur, usually due to air crossing into the systemic circulation via heart defects or transpulmonary shunts. This is known as paradoxical air embolism (PAE). Other causes of AAE involve direct arterial cannulation such as in cardiac surgery or angiography. Venous and arterial embolisms have different presentations and effects, many of which can be fatal if not recognised or left untreated. Vigilance and rapid action are the key to preventing morbidity and mortality in at risk clinical situations.

Pathophysiology

Any amount of air entering the venous system is carried to the right atrium and on to the right ventricle. It has its pathological effects in three main ways.

Cardiovascular function is affected by the compressible air causing obstruction to right ventricular ejection at the level of the pulmonary outflow tract. This type of 'air lock' is thought to be more common following a bolus air embolism. Slower infusions of air become trapped at the level of the pulmonary arterioles causing pulmonary arterial hypertension and subsequent right ventricular failure.

A second effect is seen following more gradual air entrainment that results in micro-emboli

entering the circulation. The effect of microbubbles entering the pulmonary arterioles is not only to obstruct flow but, in addition, neutrophils are attracted to the network of fibrin, red blood cells, fat globules and platelets that build up around the bubble. The ultrastructural damage that results from this leads to increased basement membrane permeability and, ultimately, to pulmonary oedema.

The third effect of VAE is that of the paradoxical embolism. This is usually only found following a large embolus. Until recently, it was thought that air could only pass into the systemic circulation via a patent foramen ovale (PFO), which is estimated to be present in approximately 27% of adults. However, it has now been shown that transpulmonary gas shunting is also a factor in paradoxical air embolism (PAE). This means that sufficient pressure builds up on the right side of the heart to push gas through the pulmonary circulation to the left atrium. Any increase in pulmonary artery pressures will favour the transfer of air into the arterial circulation.

Gas may also enter the arterial system directly. Small volumes of gas are well tolerated by most organs. However, the high metabolic requirements of the heart and brain render these organs particularly susceptible to damage following arterial air embolism. Air entering the cardiac or cerebral vasculature causes end-arteriolar obstruction, resulting in distal hypoperfusion and hypoxia. Cellular damage leads to tissue oedema, further reducing oxygenation.

Aetiology

For air to be admitted into the venous system there must be a source of gas, a communication between this source and the venous system and a pressure gradient enabling the ingress of the gas. It is the size of the above

Key points

The risk of air embolism is not confined to neurosurgery

Clinical signs are late indicators of venous air embolism

No single monitor offers absolute sensitivity and specificity for venous air embolism

The initial treatment of air embolism is 'ABC'

Paradoxical embolism can occur in the absence of a patent foramen ovale

S Webber FRCA

Specialist Anaesthetic Registrar,
Royal Hallamshire Hospital, Glossop Road,
Sheffield S10 2JF

J Andrzejowski FRCA

Consultant Anaesthetist, Royal
Hallamshire Hospital, Glossop Road,
Sheffield S10 2JF

G Francis FRCA

Consultant Anaesthetist, Royal
Hallamshire Hospital, Glossop Road,
Sheffield S10 2JF

E-mail: Gary.Francis@sth.nhs.uk
(for correspondence)

Table 1 Procedures associated with gas embolism

VENOUS	
Neurosurgery	Craniotomy, particularly sitting position Spinal surgery
Orthopaedics	Arthroplasty Arthrography
Obstetrics	Caesarean section Termination of pregnancy Removal of placenta
Head and neck surgery	Including laser procedures
Intravascular catheters	Central venous catheter insertion or disconnection Air within i.v. administration sets
Others	Laparoscopy Endoscopy Positive pressure ventilation Hydrogen peroxide
ARTERIAL	
Gas entry directly into arterial system	Cardiac surgery Cardiopulmonary bypass Angiography Carotid endarterectomy Laparoscopy Decompression sickness
Paradoxical air embolism	Intracardiac shunt Transpulmonary shunt

communication and the pressure gradient that are of critical importance in the outcome of VAE. In 1948, Durant demonstrated that the rate of air entrapment, the volume entrained and the position of the patient at the time of VAE all affect eventual outcome.

It would appear that both species and body size are also important as studies in dogs have found that the lethal volume of air, when injected rapidly, is 7.5 ml kg⁻¹. However, in smaller animals such as rabbits, the lethal volume is only 0.55 ml kg⁻¹. The effect of VAE in children is greater than in adults as a given volume of gas will be a larger proportion of the ventricular volume. Slower infusions of air are better tolerated since air has time to be re-absorbed. For example, the dog can tolerate up to 1400 ml of air over several hours. The total volume of gas tolerated in humans is harder to estimate. Small volumes can lead to symptoms, particularly if a PAE ensues, potentially causing cardiovascular and/or neurological problems. Volumes between 100–300 ml can be fatal (approximately 1 ml kg⁻¹). This volume is relevant, since it has been shown that 100 ml of air per second can be entrained through a 14-gauge cannula with only a 5 cm pressure gradient.

VAE has been reported in most areas of clinical medicine (Table 1). The seated craniotomy is the classical ‘at risk’ situation for VAE, with an incidence of up to 100% being reported. Reported rates of VAE during other surgical procedures

include incidences of 40% during Caesarean section, 30% during hip arthroplasty and 10% during anterior cervical discectomy.

Presentation

Venous air embolism

The presentation of VAE is dependent upon the rate and volume of air entrained. VAE can range from being clinically undetectable to potentially fatal. Rapid entrainment of large volumes of air will result in apnoea, hypoxia and cardiovascular collapse. Slower rates of entrainment may result in the patient complaining of light-headedness, breathing difficulties, shortness of breath, chest pain and a sense of impending death. A 10% obstruction to the pulmonary circulation can cause a gasp reflex which, in itself, reduces right atrial pressure (RAP) further increasing air entrainment.

Significant embolism leads to tachypnoea, tachycardia, and hypotension. This may be accompanied by altered mental status, decreased conscious level or focal neurological deficits. Auscultation of the heart might reveal the classical ‘mill wheel’ murmur. Pulmonary oedema may develop later.

End-tidal carbon dioxide falls as a consequence of an increase in physiological dead-space and intrapulmonary shunting. Arterial oxygen saturation falls. ECG abnormalities described include tachyarrhythmias, atrioventricular block, signs of right ventricular strain, ST segment elevation or depression and non-specific T wave changes.

In 25% of patients, the CVP is elevated and the pulmonary artery pressure rises in 50%. Arterial blood gases may reveal hypoxaemia and, less commonly, hypercarbia. Chest X-ray is initially normal but signs of non-cardiogenic pulmonary oedema may develop later.

When respiratory symptoms and signs predominate, differential diagnosis includes pulmonary embolism, pneumothorax, bronchospasm and pulmonary oedema. When the symptoms and signs are predominantly cardiovascular in nature, differential diagnosis includes cardiogenic shock, hypovolaemia, myocardial failure and septic shock.

Arterial air embolism

Most organs tolerate some air within their arterial system relatively well. However, end-arterial obstruction in the heart or brain can lead to significant morbidity and mortality.

The presence of air within the coronary arteries may produce anginal chest pain, hypotension, dysrhythmias, myocardial

depression and ventricular failure, potentially progressing to cardiac arrest. ECG changes are typical of ischaemia or infarction.

The presentation of cerebral air embolism is dependent on the volume of gas and the area of brain affected. Its onset is rapid, with symptoms and signs ranging from headache, confusion and minor motor weakness through to complete disorientation, hemiparesis, convulsions, loss of consciousness and coma. Pupillary responses may be asymmetrical and there may be impairment in vital brain stem centres resulting in abnormal respiratory patterns, cardiac dysrhythmias and circulatory failure. Anaesthetised patients may exhibit delayed awakening. Following initial presentation, the patient's condition can improve transiently before relapsing. Gas bubbles may be visible in the retina. CT or MR imaging may reveal intracerebral air and, subsequently, the development of hypodense lesions, which can be difficult to differentiate from cerebral infarction.

Paradoxical air embolism

PAE presents with symptoms and signs of both VAE and AAE.

Prevention

Those patients undergoing procedures associated with a significant risk of VAE should be identified pre-operatively to allow preventative measures and appropriate monitoring to be utilised. Good communication between all members of the medical staff involved is imperative. Patients with a PFO should, ideally, not be subjected to known high risk procedures such as sitting neurosurgical operations. Pre-operative screening for PFO in patients scheduled for procedures carrying a significant risk of VAE has been recommended. One bed-side method entails using transcranial Doppler to identify tiny bubbles in the middle cerebral artery following intravenous injection of an agitated saline solution.

The negative pressure gradient should be kept to a minimum by reducing the height between the operative site and the right atrium and by elevating the right atrial and central venous pressures (patient's cardiac status permitting). This can be achieved with intravenous fluid loading, jugular venous compression during periods of high risk, positive end-expiratory pressure (PEEP) and other devices that elevate venous pressure, *e.g.* military antishock trousers.

The use of PEEP in sitting neurosurgical patients is controversial. Very high levels are required to elevate RAP sufficiently to prevent VAE. However, 10 mmHg of PEEP may result in the pooling of embolised air in the superior vena cava

or internal jugular veins, thus lessening the likelihood of the embolism entering the heart. Application or release of PEEP can elevate RAP to levels greater than left atrial pressure, risking PAE in the presence of a PFO. Because of this and inconclusive data regarding its efficacy, it is recommended that PEEP should not be used for VAE prevention in sitting neurosurgical patients.

Intra-operatively, haemostasis should be meticulous and the venous circulation should be left open to atmospheric pressure for the shortest time period possible. When inserting or removing central venous catheters, the venous pressure at the catheter site should be kept above RAP by positioning the site below the level of the heart using the Trendelenberg or reverse Trendelenberg positions. Care should be taken to ensure that intravascular catheters and fluid administration sets are free from air and suitable occlusive dressings applied.

Detecting a venous air embolus

Early detection of VAE requires careful clinical observation. High risk cases necessitate suitable detection devices and a high index of suspicion. Clinical indicators are late signs of VAE. Moreover, they are non-specific. Monitors are more sensitive but are costly, require training and may give false positives. End-tidal capnography and precordial Doppler offer the optimum combination of sensitivity and affordability. However, neither is specific for VAE. Both are non-invasive, readily available and should be used in any surgical procedure where VAE is a significant risk. Transoesophageal echocardiography is by far the most sensitive and specific monitor to detect VAE. It is positioned to obtain a two-dimensional 4-chamber view of the heart to detect both VAE and PAE and can detect microbubbles within the chambers. Its disadvantages include cost, training requirement, need for continuous observation and risk of glottic injury. Invasive monitoring with a pulmonary artery or central venous catheter can detect VAE as an abrupt pressure increase and, in addition, a central venous catheter can be used for aspirating entrained air. A summary of the detection devices available is shown in Table 2.

Treatment of a venous air embolus

Immediate resuscitative measures should be initiated following the principles of 'ABC'. The airway should be secured, 100% oxygen administered and cardiopulmonary resuscitation commenced if necessary. Subsequently, more specific treatments can be instituted with the aim of preventing further air entry, reducing the size of the embolism and overcoming the mechanical obstruction caused by the embolism.

Table 2 Monitoring available for detection of VAE

Monitor	Sensitivity	Specificity	Advantages	Disadvantages
Transoesophageal echocardiography	0.02 ml kg ⁻¹ 10 times more sensitive than precordial Doppler	Not absolute Detects fat emboli and blood micro-emboli	Most sensitive Semiquantitative	Requires continuous observation Invasive with potential glottic injury Expensive, cumbersome May interfere with precordial Doppler Requires intensive training
Precordial Doppler	0.2 ml kg ⁻¹	Moderate	Non-invasive Earliest detector of VAE	Not quantitative Difficult placement False negatives Affected by electrocautery
End-tidal CO ₂	0.4 ml kg ⁻¹	Poor	Non-invasive Semi-quantitative Widely available	Accuracy affected by cardiac output/COPD
End-tidal N ₂	0.1 ml kg ⁻¹	Absolute	Detects sooner than end-tidal CO ₂	May miss subclinical air embolus Accuracy affected by hypotension Expensive
PA catheter	Fair	Poor	Quantitative Widely available Slightly more sensitive than end-tidal CO ₂	Placement for monitoring not conducive to aspiration Non-specific for air Expensive, invasive
Precordial/oesophageal stethoscope	Poor: mill-wheel murmur only with large VAE	Poor	Widely available Cheap Non-invasive	Requires constant supervision Gives little pre-warning of cardiovascular collapse
Transcranial Doppler	Good	Good	Non-invasive Pre-operative detection of patent foramen ovale	Training requirement for accurate placement Cumbersome Not widely available
CVP	Poor	Poor	Aids aspiration of air Cheap Widely available	Risk of trauma May itself be a source of VAE
Clinical signs	Poor	Poor	Routinely available	Late signs of VAE only
Observation of respiration	Poor	Poor	Routinely available	High anaesthetic concentration required Late signs of VAE in spontaneous respiration only

Preventing further air entry

The surgeon should flood the operative site with saline and compress the wound edges. Venous pressure at the procedural site should be elevated by: (i) positioning it below the level of the right atrium (if possible); (ii) intravenous volume loading; and (iii) increasing intrathoracic pressure with a Valsalva manoeuvre, thus reducing venous return.

Jugular venous compression will reduce venous return from the head and elevate cerebral venous pressure. Elevating the venous pressure may also help the surgeon to identify the site of air entry. In other circumstances, the source of gas should be eliminated immediately, *e.g.* abdominal insufflation discontinued in laparoscopic surgery.

Reducing the size of the embolus

Nitrous oxide is 34 times more soluble than nitrogen and thus will diffuse rapidly into the bubble, increasing its size. Therefore, nitrous oxide should be discontinued immediately and 100% oxygen administered. This increases the partial pressure of oxygen in the blood and tissues, favouring nitrogen diffusion out of the bubbles and into the alveoli (nitrogen washout).

It may be possible to aspirate air from the right atrium and in high risk cases a central venous catheter should be inserted prior to surgery. The optimum site for the tip of the catheter is in the right atrium 2 cm below the junction with the superior vena cava. Ideally, this position should be checked radiologically, or

using the changes seen on an ECG trace taken from the tip of the catheter as it enters the heart. However, following an unexpected air embolus, resuscitation should not be delayed in order to allow a central venous catheter to be sited. Multi-orifice catheters may be more effective than single orifice. Recent work suggests that, unless the catheter orifice is situated in the air lock within the right atrium, the catheter will not effectively aspirate air. A multi-orifice catheter may increase the chance of an orifice being situated within the air lock. The lumen of a pulmonary artery catheter is too narrow to permit effective air aspiration. If available and the patient's condition stable enough for transfer, the use of hyperbaric oxygen should be considered.

Overcoming the mechanical obstruction

The left lateral decubitus position described by Durant may help overcome the airlock within the right ventricle by positioning it superior to the right ventricular outflow. The Trendelenberg position has a similar effect. Inotropic agents (a bolus dose may be necessary) will increase cardiac contractility and may help improve cardiac output and systemic blood pressure in the face of increased pulmonary vascular resistance. In massive embolism, placing the patient on cardiopulmonary by-pass may be life-saving. Air can also be removed from the pulmonary artery at thoracotomy.

Treatment of an arterial air embolism

As in VAE, 'ABC' is the first line of treatment. The source should be identified and further air ingress prevented. The treatment of choice for arterial air emboli is hyperbaric oxygen. The administration of hyperbaric, 100% oxygen produces tissue hyperoxia and large pressure gradients for oxygen to diffuse into and nitrogen diffuse out of the emboli.

Delivery of oxygen to potentially ischaemic tissues distal to the obstruction is also improved. The use of anticoagulation with heparin and corticosteroids is not currently recommended. Intravenous lidocaine may be beneficial; however, its efficacy is, as yet, unproven.

Summary

Venous, arterial and paradoxical air emboli are potential complications in many of the clinical scenarios encountered by anaesthetists. Capnography is easy to use, routinely available and should detect most clinically significant emboli. In high risk situations, prevention is better than cure and it is essential that the appropriate preventative measures, monitoring tools and treatment modalities are in place. Vigilance and good communication between all clinicians involved is essential. In the event of a large embolism, cardiopulmonary resuscitation may be required. Additional treatment is aimed at preventing further air entry, overcoming the air lock and reducing the size of the embolism. Paradoxical air embolism may occur in the absence of a PFO. The treatment of choice for significant arterial air emboli is hyperbaric oxygen therapy.

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See multiple choice questions 39–42