VENOUS AIR EMBOLISM
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BASICS
DESCRIPTION
Venous air embolism (VAE) describes the entrapment of air or gas in the operative field or other communication with the environment into the vasculature, resulting in mechanical obstruction and secondary chemical and inflammatory mediators that impair gas exchange.

EPIDEMIOLOGY
Incidence
- Difficult to assess accurately due to the variable
- Posterior fossa surgery: 80%
- Central line placement and removal should be
- Perioperative care
- Avoidance of nitrous oxide is suggested particularly
- Laparoscopies: 69%
- Two requisites: Direct communication between the
- Central vascular access: The equipment allows direct
- PEEP: Theoretically, it increases intrathoracic
- Hydration: Increases CVP, and reduces the negative
- Scuba diving, aviators, and positive
- Patient positioning: The sitting position is utilized to
- Operative site above the heart (5 cm):

MORBIDITY/MORTALITY
- Proportional to the volume and rate of air entry, patient position, and entry site, as well as underlying cardiac disease

ETOLOGY/RISK FACTORS
- Operative site above the heart (CJV):
- Vascular access
- Mechanical insufflations
- Mechanical insufflations: CO2 enters inadvertent

PHYSIOLOGY/PATHOPHYSIOLOGY
- Non-invasive: Direct communication between the air source and vasculature, plus a pressure gradient favoring the passage of air into the circulation.
- Operative site >5 cm from the heart: Venous pressure exceeds atmospheric pressure, except when above the level of the heart. An injury to the wall allows air entry that travels to the right heart and then the lungs. This increases with large, uncompromised venous channels, such as dural sinuses in neurosurgery.
- Mechanical insufflations: CO2 enters inadvertent open vascular channels from surgical manipulation. It is 20 times more dissolved in blood than oxygen and significantly more than nitrogen, which may explain why, despite its high occurrence, it is often subclinical.
- Pressure infusions/liquid irrigation: When air is accidentally introduced into the system, it can subsequently be "driven" by the fluid pressure into veins or cancellous bony surface.
- Central vascular access: The equipment allows direct access to the vein during insertion or removal and when the patient is spontaneously ventilating, a negative pressure breath can provide the driving force.
- Inadvertent introduction of air into arterial lines, peripheral/IV, and other access is also a cause.
- Clinical manifestations result from mechanical and chemical pathophysiologic mechanisms, with the key factors being volume and rate of entrainment. The alveolar-capillary interface is capable of absorbing and exhausting gas from the circulation; when it occurs over a slow period, it is capable of withstanding large quantities. When this mechanism is overwhelmed (large amount and/or fast rate), symptoms result.
- Mechanical obstruction results from a gas-airlock in the right ventricular outflow tract (RVOT) or pulmonary circulation (V/Q mismatch: dead space being ventilated, but not perfused). Partial RVOT obstruction causes right heart strain (increased CVP, JVP, peaked P waves), decreased CO (increased wedge pressure; reduced MVO2, BP), with resultant cardiac and cerebral ischemia (myalgic cardiomyopathy, ST changes). Complete RVOT obstruction results in heart failure, CV collapse, and possible death.
- Chemical pathways: Air in the right heart and pulmonary circulation trigger secondary injury from the activation of complement and inflammatory pathways (endothelin 1, platelet activator inhibitor fibrin, leukotriene, lipid droplets, etc.). This results in pulmonary vasoconstriction/hypertension, microvascular permeability, platelet aggregation, noncardiogenic pulmonary edema, bronchoconstriction, and subsequent V/Q mismatch (shunting; ventilation hindered, perfusion continues and returns non-oxygenated blood to the left heart circulation).
- Paradoxical air embolism: The presence of a patent foramen ovale (25–30% of the population) allows for a gas bubble to travel from the right to left side of the heart (pulmonary angiography, direct delivery to cerebral and coronary arteries, resulting in ischemia or infarct).

PREVENTATIVE MEASURES
- Patient positioning: The sitting position is utilized to provide adequate surgical conditions (visualization, reduced bleeding). Maintain a high level of suspicion, reduce the driving pressure gradient, and implement appropriate monitoring. Alternatives include the prone and park bench position (strongly consider with documented PFO).
- Hydration: Increases CVP, and reduces the negative pressure gradient at the level of insertion. Studies have suggested maintaining the CVP at 10–15 cm H2O. Consider zeroing the transducer at the level of the right atrium, and then elevating it to the surgical site to determine if a negative pressure gradient exists.
- Central line placement and removal should be performed in the Trendelenburg position (increases venous pressure at access site by gravity). During insertion and removal, consider occlusion of the needle hub, avoidance of deep inspiration when the patient is UV (causal negative pressure suction effect), and PEEP when the patient is being PEEP. When Trendelenburg is contraindicated (increased ICP), consider temporary positioning during insertion of the guidewire or catheter, or raising the legs to increase venous return and pressure in the right atrium.
- PEEP: Theoretically, it increases intrathoracic pressure, thus reducing air entrapment into the surgical site (note: in patients with increased ICP, the goal is to reduce intrathoracic pressure to facilitate venous drainage of blood into the right atrium). Conversely, because it may increase the risk of paradoxical arterial embolism (increased intrathoracic, pulmonary vasculature pressures, facilitates flow through the PFO and may exacerbate hypotension when there is a sudden release. Consider PEEP when oxygenation needs to be improved, rather than to prevent a VAE.
- Avoidance of nitrous oxide is suggested particularly for sitting cisternotomies. However, studies have not shown an increased incidence of VAE.

DIAGNOSIS
- Spectrum of effects is dependent on rate and volume of air entrainment.
- Signs and symptoms: Dizziness, diaphoresis, continuous cough, gasp reflex, lightheadedness, vertigo.
- Pulmonary: Wheezing, bronchoconstriction, pulmonary hypertension, cyanosis, tachypnea.
- Cardiac: Systolic murmurs, peaked P waves, ST segment changes, JVP, right heart failure, low BP, CV collapse.
- Cerebral: AMS, seizures, ischemia due to hypoperfusion or air embol in the cerebral circulation from a patent foramen ovale.
- Transesophageal echocardiogram: Permits detection of microemboli and macroemboli, as well as paradoxical cerebral complications. Most sensitive is detected at a little as 0.02 mL/kg, limited by cost, invasiveness, and low sensitivity (not clinically relevant levels).
• Precordial Doppler ultrasound: Audible detection of RVOT blood flow (normal flow produces a ‘whistling machine’/turbulent sound; VAE results in an erratic, high-pitched swishing noise, or ‘mill wheel’ sound). Placed along the right heart border (second to fourth intercostals space), confirm position with “bubble test” (1 mL air = 9 mL saline delivered fast push into the central or peripheral access). Most sensitive non-invasive monitor 0.05 mL/kg or 0.25 mL of air. Limited by obesity, prone or lateral position, and concurrent use of cautery.

• Pulmonary artery catheter: VAE causes mechanical obstruction and decreased CO (obstruction in pulmonary artery results in increased PAOP, and CV pressure; reduced CO seen as reduced MVO2, and CV ischemia can increase HVR). Additionally, small amounts of air may be aspirated from the lungs. Limited by being relatively insensitive (requires at least 0.25 mL/kg air), invasive (not justified as monitor unless patient has qualifying comorbidities), and minimal ability to aspirate air.

• Transcranial Doppler: Useful in detecting cerebral air embolism during surgical procedures (increased with Valvulare maneuvers), non-invasive.

• ETO2: Air is ~ 75% nitrogen. If utilizing 100% O2 in the circuit, the detection of N2 in expired gas suggests VAE. It is the most sensitive gas-analysis method (can detect as little as 0.04% N2, and 30–90 seconds earlier than ETCO2). However, it is not routinely available on all monitors.

• ETCO2: Reduced CO2 in exhaled air results from dead space (CO2 continues to increase in the blood, but cannot be delivered or excreted via the alveoli). Moderate sensitivity (change of 2 mm Hg may indicate VAE). Standard ASA monitor, but lacks specificity and reliability. Hypoproteinemia also produces “dead space” effects. Consider adjusting the “low” level alarm to optimize detection.

• O2 saturation: Late finding.

**DIFFERENTIAL DIAGNOSIS**

- Acute coronary syndrome
- Cardiogenic shock
- Cerebrovascular accidents

**TREATMENT**

- Prevention
  - Stop further air entry. Inform surgeon, cover surgical field with saline-soaked dressings, assess and if immediate entry site (causity, bone wax), increase CVP with fluid hydration (reduces pressure gradient).
  - Til OR table Trendelenburg, oversea Trendelenburg, lateral decubitus) to place the likely source of air entry below the level of the heart (eliminates the negative pressure gradient).

- Neurosurgical procedures, consider transventricular venous compression (increases venous pressure, helps identify open dural sinuses from retrograde flow; however, this increases ICP, and may complicate the cardiac anatomy with resultant decreases in the CPP).
- Reduce volume of entrained air: Aspirate air from the right atrium via the central line port, if present. Controversial whether to place after VAE occurrence (distorts from other treatment, capable of removing only 15–20 mL). Surgical procedure with a high risk for VAE, consider a multisite catheter.

- Reduce-embolic obstruction: Strong, effective chest compressions. Consider positioning the patient in the left lateral decubitus position (Durant’s maneuver) to relieve airlock in the right heart (no data to support this and will interfere with chest compressions).

- Hemodynamic support/supportive measures: High-flow oxygen (FiO2 1.0; may reduce embolism prevention). Experimental therapies: Fluorocarbon derivatives (decreases diffusion gradient 1.0, at a partial pressure of 2000 mm Hg). Typically a 5-hour session of therapy, if not already in place.

- High-flow oxygen (FiO2 1.0): May reduce embolism volume by eliminating N2, increases oxygen delivery to ischemic areas. CPR (defibrillation and compression) provides blood flow and oxygenation, particularly in pulseless electrical activity. Inotropic support to the right ventricle (failure from increased pulmonary circulation afterload) can be achieved with dobutamine, epinephrine, dopamine, and norepinephrine.

- Hyperbaric oxygen therapy: Utilized for decompression therapy. Reduces the size of air bubbles by creating a high O2 diffusion gradient that accelerates N2, therefore, FiO2 1.0, at a partial pressure of 2000 mm Hg. Typically a 5-hour session for the initial treatment, followed by sessions 1–2 times a day for symptom relief.

- Repair: Propylactic administration may reduce the severity of symptom. Steroids: May reduce secondary inflammatory pathway. However, it does not have any effect on cytotoxic brain edema seen in VAE.

- Lubricate: Propylactic administration may reduce the effect of VAE on the brain by decreasing brain edema.

- Experimental therapies: Fluorocarbon derivatives enhance the reabsorption of bubbles and enhance the solubility of gases in the blood. Fluorocarbon FP-43 enhances O2, CO2, and N2 solubility to 100,000 times that of plasma. Human data lacking at this time.

**FOLLOW-UP**

- If hemodynamic stability is recovered, may consider continuing with surgical procedure
- Consider continued intubation with positive pressure ventilation, vasopressors
- Consider central line placement for monitoring and therapy, if not already in place.

**REFERENCES**


7. See Also Topic, Algorithm, Electronic Media Element

- Trendelenburg
- Ventilation and perfusion mismatching
- Central line placement
- Laspacor

**CODES**

<table>
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<tr>
<th>ICD9</th>
<th>Definition</th>
<th>Payment range</th>
<th>Claims resulting in payment</th>
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<td>937</td>
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<td>$20,800–$3,302,700</td>
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**CLINICAL PEARLS**

- Two requirements: Direct communication between air source and vasculature PLUS a driving pressure gradient.
- Pathophysicsy is composed of mechanical obstruction and secondary insult due to chemical and inflammatory mediators.
- Treatment should consist of prevention, prevention, prevention. Followed by stopping further embolization, removal of entrained air, and hemodynamic support.
- Iatrogenic and preventable: New technologies have expanded the realm of procedures where the patient is at risk; thus, it requires a high index of suspicion.