air embolism

general

- Air embolism, the entry of gas into vascular structures, is a largely iatrogenic clinical entity responsible for serious morbidity, and even mortality, in many varied medical specialties
- Furthermore, it is one of the most serious problems in diving medicine.
- The medical use of varied gases has created numerous other gas embolisms, including carbon dioxide, nitrous oxide, and nitrogen emboli.
- There are two broad categories of gas embolism, venous and arterial, depending on the mechanism of gas entry and where the emboli ultimately lodge.

aetiology

venous gas embolism

- A venous gas embolism occurs by the entry of gas into the systemic venous system.
- This gas is then transported to the lungs via the pulmonary arteries, causing interference in gas exchange, arrhythmias, pulmonary hypertension, right ventricular strain, and finally cardiac failure.
- Predispositions that allow the entry of gas into the venous system include incision of noncollapsed veins and the presence of subatmospheric pressure in these vessels. These conditions occur when the surgical field is above the level of the heart
- Other potential pathways include entry of air into central venous and hemodialysis catheters and entry into the veins of the myometrium in the peripartum period.

arterial gas embolism

(i) paradoxical embolism
- A paradoxical embolism arises when air/gas entrained in the venous circulation manages to enter the systemic arterial circulation causing symptoms of end-artery obstruction.
- There are a number of mechanisms by which this can occur. One of these is the passage of gas across a patent foramen ovale to the systemic circulation. A patent foramen ovale is detectable in about 30% of the population and makes right-to-left shunting of gas bubbles possible.
- Elevated pulmonary arterial pressure due to a venous gas embolism may be reflected in elevated right atrial pressures predisposing to bubble transport across a patent foramen. In addition, the decrease in left atrial pressure caused by controlled ventilation and use of positive end-expiratory pressure may create a pressure gradient across the patent foramen ovale favoring passage of gas into the systemic circulation.

(ii) overwhelming venous embolism
- In other situations, venous gas may enter the arterial circulation by overwhelming the filter capacity of the lungs normally in place to prevent arterial gas emboli.

(iii) overexpansion of the lung through decompression barotraumas in diving, pulmonary barotraumas in the ventilatory therapy for critical care patients, and paradoxical embolism.

(iv) all cardiac surgical operations with extracorporal bypass are a potential mechanism for these events.

pathophysiology

venous gas embolism

- With rapid entry, or larger volumes of gas, increasing strain on the right ventricle follows because of the migration of the emboli to the pulmonary circulation. The pulmonary arterial pressure increases, while the increased resistance to right ventricle outflow causes diminished pulmonary venous return.
- These alterations of lung vessel resistance and ventilation-perfusion mismatch in the lung cause intrapulmonary right-to-left shunt with increased alveolar deadspace, leading to arterial hypoxia and hypercapnia.

arterial gas embolism

- Entry of gas into the aorta causes distribution of gas bubbles into nearly all organs.
- Small emboli in the vessels of the skeletal muscles or viscera are well tolerated, although organ dysfunctions such as rhabdomyolysis and/or renal insufficiency may occur as well.
- Embolization to the cerebral or coronary circulation may result in severe morbidity or death.

manifestations

venous air embolism
(i) visible evidence of air being sucked into veins
(ii) significant embolism leads to tachycardia, hypotension & tachypnoea
(iii) there is gasping respiration (if breathing spontaneously) - a 10% obstruction to the pulmonary circulation can cause a gasp reflex
(iv) ETCO2 falls due to an increase in physiological dead space & intrapulmonary shunting
(v) ECG abnormalities described include signs of right ventricular strain, atrioventricular block, tachyarrhythmia, ST depression or elevation & other non-specific ST changes
(vi) blood pressure falls:
- with bolus air embolism air causes obstruction to the right ventricular outflow tract
- with slower infusions, air becomes trapped at the pulmonary arterioles causing pulmonary hypertension and RV failure (these not only obstruct flow but lead to and inflammatory response that causes pulmonary oedema)
(vii) a change in heart sounds (mill wheel murmur) is very late
(viii) CVP is elevated in 25% and PAP rises in 50%

arterial gas embolism
- The signs and symptoms associated with cerebral arterial gas embolism can develop suddenly. The clinical presentation is determined by the absolute quantity of gas and the brain areas affected.
- After surgeries with risks for the development of gas embolism, a delayed recovery from general anesthesia or a transitional stage of impaired consciousness can be a clue to a cerebral arterial gas embolism.

differential diagnosis

- if respiratory features predominate, consider pulmonary embolism, pneumothorax, bronchospasm & pulmonary oedema
- if cardiovascular features predominate, consider cardiogenic shock, hypovolaemia, myocardial infarction & septic shock

treatment

	Venous Gas Embolism	Arterial Gas Embolism
Prevent Further Gas Entry	Increase venous pressure (e.g., Valsalva, IV fluids) Identify and disable entryway for gas	Identify and disable entryway for gas
Definitive Therapy	Supportive	Hyperbaric oxygen therapy as soon as patient stable for transfer to hyperbaric oxygen facility
Supportive Therapy	Oxygen, intravascular volume expansion, catecholamines	Oxygen, intravascular volume expansion, catecholamines
Positioning	Supine	Supine
Evacuation of Embolized Gas	Aspiration of multi-lumen central venous catheter; patient in left lateral decubitus position	Hyperbaric oxygen
Adjunctive Therapy	Hyperbaric oxygen	Lidocaine, antiepileptics

- Protection and maintenance of vital functions is the primary goal.
- If warranted, cardiopulmonary resuscitation must be performed, because not only venous but also primary arterial gas embolism may lead to serious impairment of the cardiovascular system.

- For somnolent or comatose patients, endotracheal intubation should be performed to maintain adequate oxygenation and ventilation. Additionally, oxygen should be administered in as high a concentration as possible.
- This is important not only to treat hypoxia and hypoxemia but also for the elimination of the gas in the bubbles through a diffusion gradient favoring egress of gas from the bubble.

- The current therapeutic recommendation is to maintain a flat supine position for these patients, because neither head-down positions nor an elevated head position provides any detectable cardiovascular benefits and may aggravate the cerebral insult.

- Cerebral gas embolism may be associated with the development of generalized seizures that may resist management by benzodiazepines. In these cases it is advised to suppress the seizure activity with barbiturates. It must be stressed, however, that with sufficient doses of barbiturates, respiratory drive is depressed and the patient's ventilation must be supported.

- The definite treatment of arterial gas embolism is hyperbaric oxygen therapy with best results reported when performed as early as possible.
- Hyperoxia produces a diffusion gradient for oxygen into the gas bubble, as well as for egress of nitrogen (or other gas) from the bubble.
- Hyperoxia also enables significantly larger quantities of oxygen to be dissolved in the plasma and also increases the diffusion distance of oxygen in tissues.