

# Diving Medicine Online

Comprehensive updated online information about diving and undersea medicine for the non-medical diver, the non-diving medical professional and an excellent reference source for the diving medical specialist.

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## Arterial Gas Embolism

### Pathophysiology

Arterial gas embolism is a major cause of death in diving and the initiating cause (pulmonary barotrauma) usually goes undetected. Caused most often by the expansion of respiratory gases during ascent, it also occurs when the breath is held during ascent from a dive, when there is local pulmonary pathology, when there is dynamic airway collapse in the non-cartilaginous airways and if there is low pulmonary compliance, particularly if this is not distributed evenly throughout the lungs. Boyle's law is the physical law controlling the event. Experimental evidence indicates that intratracheal pressures of about 10 kPa (4 fsw or 1.22 m. or ascending from 170 feet or 51.82 m. to 120 feet or 36.58 m.) are all that's needed for it to happen. Distention of the alveoli leads to rupture, alveolar leakage of gas, and extravasation of the gas into the arterial circuit.

### Origin of Bubbles

Bubbles in the arterial circulation can arise from basically three sources: venous gas embolism with breach of the pulmonary vascular filter (paradoxical gas embolism), patent foramen ovale (paradoxical gas embolism) and tear of the pulmonary parenchyma with entry of gas into pulmonary venous outflow. Studies show that systemic venous bubbles are trapped in the pulmonary arterial tree and are usually completely eliminated from that site. The lung traps the air and excretes it into alveoli from the arterioles. (RG Presson, J Appl Physiol; 1989;67(5),1898-1902)

The syndrome of **paradoxical air embolism** (from septal defects) was first described by J. Cohnheim in 1877. (J Cohnheim, ZV Berline, Hirschwald, 1877;1:134). Hagan at the Mayo Clinic reported on 965 normal hearts and showed that more than 25% of patients with a history of cardiac disease have a 'probe patent' foramen ovale at autopsy. (PT Hagan, Mayo Clinic Proc, 1984; 59:17-20.).

The other main mechanism for arterial gas embolism is by way of the **pulmonary**

**overpressure syndrome or 'burst lung'**. This occurs from baropressure increases as the diver on compressed air ascends with a closed glottis or a free diver takes a breath of compressed air at depth and ascends. Because of Boyle's law, maximal changes in volume occur in the 4 feet (1.22 m.) closest to the surface and the diver sustains a tear in the pulmonary parenchyma with the escape of air into the pulmonary venous outflow. This can result in several outcomes: pneumothorax (collapsed lung), pneumomediastinum (air in the space around the heart), subcutaneous emphysema (bubbles of air in the fatty tissues under the skin) and air into the pulmonary capillaries.

As the diver takes his first breath after surfacing, the extra-alveolar gas enters the torn blood vessels, migrates to the left side of the heart and is distributed systemically as emboli sent to areas determined by buoyancy.

Arterial gas emboli arise from gas bubbles in the pulmonary capillaries => pulmonary veins to the left side of the heart => possible coronary artery emboli (rare) or internal carotid and vertebro-basilar arteries to the brain => cerebral artery embolism (blockage) with the clinical picture of a stroke.

The foam or bubbles block arteries of the 30-60 micron caliber and cause distal ischemia, with astrocyte and neuronal swelling. As the bubble passes over the endothelium, there are direct cellular effects (within 1-2 minutes) causing PMN stimulation. The bubble itself has surface effects causing local swelling, downstream coagulopathy with focal hemorrhages. There is immediate increased permeability of the blood-brain barrier, loss of cerebral auto-regulation, rise in CSF and a rise in the systemic blood pressure. A phenomenon called 'no-reflow' occurs with a post-ischemic impairment of microvascular perfusion. This is thought to be the result of Factor VIII interacting with the prostaglandin system and possibly other blood/tissue factors.

## Clinical Manifestations

The clinical manifestations of cerebral gas embolism include a sudden onset of unconsciousness associated with a generalized or focal seizure. There is often confusion, vertigo (extreme dizziness) and cardiopulmonary arrest. In a series of 24 USN cases in which the time was known, 9 occurred during ascent in the water, 11 within one minute at the surface and 4 occurred within 3-10 minutes at the surface.

Other clinical manifestations include the sudden onset of hemiplegia (paralysis on one side), focal weakness, focal hypesthesia (loss of feeling), visual field defect (blank areas in vision), blindness, headache and cranial nerve defects (vision, hearing, eye movements, facial muscles and feeling). The operative word here is "sudden"--nearly all of these symptoms can also be caused by neurological decompression sickness. Less common manifestations are chest pain and bloody, frothy sputum.

# Management Outline

Recognition \*This usually occurs during or immediately after surfacing\*

- Symptoms
  - Bloody froth from mouth or nose
  - Disorientation
  - Chest pain
  - Paralysis or weakness
  - Dizziness
  - Blurred vision
  - Personality change
  - Focal or generalized convulsions
  - Other neurological abnormalities
  - Hemoptysis (bloody sputum)
  - Signs
  - Bloody froth from nose or mouth
  - Paralysis or weakness
  - Unconsciousness
  - Convulsions
  - Stopped breathing
  - Marbling of the skin
  - Air bubbles in the retinal vessels of the eye
  - Liebermeister's sign (a sharply defined area of pallor in the tongue).
  - Death
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## Early management

- CPR, if required
- Open airway, prevent aspiration, intubate if trained person available
- Give O<sub>2</sub>, remove only to open airway or if convulsions ensue.
- If conscious, give nonalcoholic liquids
- Place in horizontal, neutral position
- Restrain convulsing person loosely and resume O<sub>2</sub> as soon as airway is open.
- Protect from excessive cold, heat, water or fumes.
- Transport to nearest ER for evaluation and stabilization in preparation for removal to the

nearest recompression chamber.

- Call DAN (919-684-8111) or your own preferred emergency number
  - Air evacuation should be at sea level pressure or as low as possible in unpressurized aircraft
  - Contact hyperbaric chamber, send diver's profile with the diver, and send all diving equipment for examination or have it examined locally.
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## Treatment

Recompression as soon as possible

Oxygen

Cautious hydration

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## Links

- [Reducing the Risks for Pulmonary Barotrauma](#)
- [Pulmonary barotrauma References](#)