1. AIR OR GAS EMBOLISM

Richard E. Moon

Rationale

Gas embolism occurs when gas bubbles enter arteries or veins. Arterial gas embolism (AGE) was classically described during submarine escape training, in which pulmonary barotrauma occurred during free ascent after breathing compressed gas at depth. Pulmonary barotrauma and gas embolism due to breath holding can occur after an ascent of as little as one meter (1). AGE has been attributed to normal ascent in divers with lung pathology such as bullous disease and asthma (2, 3). Pulmonary barotrauma can also occur as a result of blast injury in or out of water (4, 5), mechanical ventilation (6), penetrating chest trauma (7), chest tube placement (8) and bronchoscopy (9).

Venous gas embolism (VGE) occurs commonly after compressed gas diving (10, 11). Normally, VGE bubbles are trapped by the pulmonary capillaries and do not cause clinical symptoms. However, in large volumes, VGE can cause cough, dyspnea and pulmonary edema (12, 13), and may overwhelm the capacity of the pulmonary capillary network, allowing bubbles to enter the arterial circulation (14, 15). VGE can also enter the left heart directly via an atrial septal defect or patient foramen ovale (16-19).

Causes of gas embolism other than diving include accidental intravenous air injection (20, 21), cardiopulmonary bypass accidents (22), hemodialysis (23), central venous catheter placement or disconnection (24, 25), gastrointestinal endoscopy (26), hydrogen peroxide irrigation (27, 28) or ingestion (29-31), arthroscopy (32, 33), percutaneous hepatic puncture (34), blowing air into the vagina during orogenital sex (35-37), sexual intercourse after childbirth (38), laparoscopy (39-43), transurethral prostatectomy (44). Massive VGE can occur due to passive entry of air into surgical wounds that are elevated above the level of the heart (such that the pressure in adjacent veins is subatmospheric) (45). This has classically been described in sitting craniotomy (46), but has also occurred during cesarean section (47), prostatectomy using the radical perineal (48) and retropubic (49, 50) approaches, spine surgery (51), hip replacement (52), liver resection (53), liver transplantation (54) and insertion of dental implants (55, 56).

Clinical deficits can occur after intra-arterial injection of only small volumes of air. Intravenous injection is often asymptomatic. Injection of up to 0.5-1 mL/kg has been tolerated in experimental animals (57). In humans, continuous IV infusion of oxygen at 10 mL/min has been reported as well tolerated, while 20 mL/min caused symptoms (58). Compared with constant infusions, bolus injections are more likely to cause clinical abnormalities (59).

There are several possible mechanisms of injury, including intracardiac 'vapor lock', with resulting hypotension or acute circulatory arrest, and direct arterial occlusion. Animal studies using a cranial window have demonstrated that bubbles can cause a progressive decline in cerebral blood flow (60, 61) even if they don't occlude vessels. This effect appears to require neutrophils (62), and may be initiated by bubble-induced endothelial damage (63-65). In some cases of cerebral AGE there is clinical improvement followed by delayed deterioration a few hours later (66). Mechanisms that have been hypothesized include edema, bubble re-growth and secondary thrombotic occlusion.

Manifestations of arterial gas embolism include loss of consciousness, confusion, focal neurological deficits, cardiac arrhythmias or ischemia. Venous gas embolism manifests as hypotension, tachypnea, hypocapnia, pulmonary edema or cardiac arrest (67-69). AGE in divers

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with a pre-existing inert gas load (due to a dive) can precipitate neurological manifestations that are more commonly seen with DCS, such as paraplegia due to spinal cord damage (70). While imaging studies sometimes reveal intravascular air, brain imaging is often normal even in the presence of severe neurological abnormalities (71-74).

First aid treatment for gas embolism includes support of breathing and circulation. Supplemental oxygen is recommended not only to maintain arterial oxygenation, but also to facilitate bubble resorption by establishing a gas diffusion gradient. Nitrous oxide causes bubbles to grow. Therefore, if gas embolism is suspected in an anesthetized patient, nitrous oxide should be discontinued in favor of oxygen.

Head-down position is sometimes recommended for the initial treatment of patients with AGE, in order to minimize the risk of additional cerebral embolization because of buoyancy, and shrinkage of bubbles due to increased hydrostatic pressure, and some anecdotal cases support its use (75). Lateral decubitus position has been recommended for VGE. However, buoyancy has little if any effect upon arterial (76) or venous (77) distribution of intravascular air, and head-down position can worsen cerebral edema (78). Therefore, except for a brief period (less than 10 min) of head-down position, which might conceivably result in enhanced clearance of bubbles from the cerebral circulation after arterial blood embolism, the supine position is preferable.

HBO_2 to treat gas embolism remains the definitive treatment for gas embolism (79, 80). Indications for treatment include neurological manifestations or cardiovascular instability. A review of 597 published cases of arterial gas embolism reveals superior outcomes with the use of HBO_2 compared to non-recompression treatment (36, 67, 81-90). HBO_2 treatment is not required for asymptomatic VGE, however it can effect clinical improvement in patients with secondary pulmonary edema (91). Gas bubbles have been known to persist for several days and there are many reports noting success when HBO_2 treatments were begun after delays of hours to days (36, 89, 92, 93). A trial of HBO_2 therapy may be indicated even for those patients coming to a hyperbaric unit after a significant delay following the inciting event. Because of the tendency for patients with AGE to deteriorate after apparent recovery, early HBO_2 is recommended even for patients who appear to have spontaneously recovered. One author has suggested that the presence or absence of air detectable by brain computed tomography should be used as a criterion for HBO_2 therapy (94). However, timely administration of HBO_2 usually causes some clinical improvement, even in the absence of demonstrable air. Performing brain imaging usually delays the initiation of appropriate HBO_2 treatment and rarely serves a useful clinical purpose.

In patients with AGE caused by pulmonary barotrauma there may be a coexisting pneumothorax, which could develop into tension pneumothorax during chamber decompression. Therefore, if the patient will be treated in a monoplace chamber, placement of a chest tube prior to HBO_2 is recommended. For multiplace chamber treatment careful monitoring is a feasible option. Coexisting pneumomediastinum does not generally require any specific therapy, and will usually resolve during HBO_2.

Administration of repetitive treatments is recommended until there is no further stepwise improvement, typically after no more than 1-2 hyperbaric treatments, but occasionally up to 5-10 (95).

Immediate recompression to 6 atm abs pressure was recommended in the past. However there is no conclusive evidence that pressures higher than 2.82 atm abs (18 msw, 60 fsw) offer any advantage. If possible, an initial compression to 2.82 atm abs (60 fsw or 18 msw equivalent depth) breathing 100% oxygen is recommended, using USN Treatment Table 6 or equivalent. If the clinical response to treatment is judged to be suboptimal, options including deeper recompression or extension of the treatment table can be instituted according to the expertise and resources available.

The standards against which other treatment schedules ("tables") should be compared are those of the U.S. Navy (USN Diving Manual) and similar procedures used by other navies and commercial diving operations (79, 95).

Adjunctive therapies such as intravenous fluids, corticosteroids and lidocaine are discussed in a separate volume (96). A summary of current recommendations for adjunctive therapy is available on the Undersea and Hyperbaric Society website (**http://www.uhms.org**).

Guidelines for Use of HBO_2 in Gas Embolism

The use of HBO_2 for arterial gas embolism and symptomatic venous gas embolism should be considered an AHA level I recommendation in spite of the absence of type 1 evidence (randomized controlled trials). There are no other definitive treatments for these disorders. Hyperbaric oxygen has been safely and effectively administered for many years. All other treatments should be considered adjunctive to hyperbaric oxygen.

Utilization Review

Utilization review is recommended after 10 treatments.

Cost Impact

The primary treatment of choice for air embolism from any cause is HBO_2 therapy. Decreased high mortality rates and prevention or moderation of permanent neurologic damage make this modality cost effective.

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