AIR OR GAS EMBOLISM

Air or gas embolism occurs when gas bubbles enter arteries, veins and/or capillaries. This results in reduced blood flow and poor oxygen delivery to the areas supplied by the affected circulation. If not fatal, gas embolism can result in severe, long-standing and irreversible physical and emotional disabilities. There can be weakness or paralysis in the limbs; vision can be impaired or absent; brain, heart, lung and other organ damage may occur. Limited use of remaining functions can be sufficiently severe that total disability results. Those who do not die may be limited to walking with canes, crutches or walkers. Those more severely disabled may be wheelchair confined or bedridden. These outcomes may be permanent and may severely impact quality of life. Maximal medical treatment of the condition is necessary to ensure the best possible degree of recovery from this potentially disastrous problem.

Hyperbaric oxygen has been shown to reduce the size of bubbles obstructing circulation. The increased pressure in the hyperbaric chamber reduces bubble size and drives the remaining gas into physical solution, while the high oxygen pressure washes out inert gas from the bubble. When bubbles are smaller or resolved, blood flow resumes. Poorly oxygenated tissues then receive higher levels of oxygen delivery. Another problem in gas embolism is that vessels obstructed by bubbles may leak fluid into surrounding tissues, resulting in swelling. Such swelling can further reduce tissue blood flow. When flow is restored, the local swelling will subside with resultant improvement in circulation and oxygen supply. Finally, the high levels of oxygen provided in the hyperbaric chamber have the potential to immediately restore cellular oxygen levels while blood flow impairment and tissue swelling are being corrected.

Hyperbaric oxygen treatment is the primary treatment for gas embolism and a major review of reported cases clearly indicates superior outcomes with its use compared to non-recompression treatment.

References


Selected references on Gas Embolism:

- Waite CL, Mazzzone WF, Greenwood, ME, et al: Cerebral air embolism, I. Basic studies. Submarine Medical Research Laboratory, U.S. Naval Submarine Medical Center Report No. 493:1-14. Historically significant publication from the U.S. Navy Bureau of Medicine and Surgery. It compares hyperbaric treatment to no hyperbaric treatment in an open-brain model of gas embolism. Dogs not treated hyperbarically "all died or were left with severe residual neurological defects". All of the
hyperbarically-treated animals made a complete recovery, with one exception. This research paved the way for the modern hyperbaric treatment protocols for gas embolism.


- Helps SC, Gorman DF: *Air embolism of the brain in rabbits pretreated with mechlorethamine*. Stroke 1991;22:351-354. The third in a series of articles published in STROKE by these authors. They have demonstrated a more complex pathophysiology than that previously appreciated. Cerebral arterial embolization results in flow deficit, ischemia, followed by a reperfusion-like injury component in many cases. Such ischemia-reperfusion complications require the presence of leukocytes. Hyperbaric oxygen is necessary to support areas of critical flow impairment. Hyperbaric oxygen will also serve to antagonize leukocyte-mediate ischemic-reperfusion injury (see #5, Acute Traumatic Peripheral Ischemia, Article #9, Zamboni, et al).


- Leitch DR, Greenbaum, LJ, Hallenbeck: *Cerebral arterial air embolism: IV. Failure to recover with treatment, and secondary deterioration*. Undersea Biomed Res 1984;11(3):265-274. Comparison of treatment protocols in a "severe" model of gas embolism. Hyperbaric doses of air and oxygen were compared to normobaric (non-hyperbaric) air. In extrapolating this model to the clinical arena "…the majority of patients with severe arterial gas embolism will achieve maximum benefit from compression to 2.8 bar while breathing oxygen", i.e., hyperbaric oxygen therapy.
