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VIEWPOINT

Hyperbaric oxygen therapy for acute smoke inhalation injuries

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Patients with acute thermal burns often have pulmonary injuries secondary to the inhalation of superheated gases and toxins. For victims trapped in closed spaces, the likelihood of such injuries is increased. Inhalation of steam causes edema and closure of the upper airway, while inhalation of smoke and hot, dry gas injures the pulmonary parenchyma. Combustion of hydrocarbons generates toxic by-products, such as carbon monoxide and cyanide, causing additional pulmonary injuries and tissue asphyxia. Some of these injuries can benefit from hyperbaric oxygen therapy.

Hyperbaric oxygen therapy is defined as ventilation with 100% oxygen at pressures greater than those found at the earth's surface. By virtue of its vasoconstrictive properties, hyperbaric oxygen is beneficial to smoke-injured pulmonary tissue. It has been shown to reduce extravascular lung water by 20% in experimentally induced thermal inhalation injuries.[1] Other reports show a reduction in respiratory morbidity in burn patients treated with hyperbaric oxygen.[2-4] It is considered the primary therapy in cases of carbon monoxide intoxication and is proving a useful adjunct in treating cyanide poisoning.

ACUTE SMOKE INHALATION—Because soft-tissue swelling from mucosa injured by smoke and extreme heat can rapidly occlude an airway, aggressive management is needed and early tracheal intubation may be required. Once the airway has been secured, fluid resuscitation started, and internal injury ruled out, hyperbaric oxygen therapy should be initiated for patients with significant pulmonary injury. To be maximally effective, this therapy should be started within 4 hours of injury.[3] We recommend an initial treatment at 3.0 atmospheres absolute (ATA) for 30 minutes, followed immediately by exposure to 2.5 ATA for 60 minutes. If symptoms persist, additional treatments at 1.5 to 2.0 ATA are given at 2- to 6-hour intervals in the first 48 hours, alternated with breathing at 1.0 ATA.

continued

Table 1. Effect of hyperbaric oxygen on the half-life of carboxyhemoglobin

Breathing medium	Pressure (ATA)	Half-life (min)	Time reduction factor (%)
Air	1	320	100
Oxygen	1	90	28
Oxygen	3	23	7

ATA, atmospheres absolute.

CARBON MONOXIDE INHALATION—Carbon monoxide is produced from the incomplete combustion of fossil fuels. In addition to combining with hemoglobin in a tight but reversible fashion, this pulmonary asphyxiant binds to cytochrome a3 in the mitochondrial respiratory chain and inhibits oxidative phosphorylation.[5] The oxyhemoglobin dissociation curve is shifted to the left, further exacerbating tissue hypoxia.

Current information suggests that hypoxic encephalopathy secondary to carbon monoxide poisoning results from a reperfusion injury. Products of lipid peroxidation from the reperfusion injury contribute significantly to the morbidity and mortality of this condition.[6,7] This explains why carboxyhemoglobin levels are poor indicators of the severity of the intoxication[8] and why patients with significant carbon monoxide poisoning may have low levels.

The half-life of carbon monoxide in room air is 5½ hours. Hyperbaric oxygen therapy decreases the half-life to 90 minutes at 1.0 ATA and to 23 minutes at 3.0 ATA[9] (table 1). Animal studies have demonstrated reduced morbidity and mortality rates using hyperbaric oxygen therapy compared with using oxygen at 1.0 ATA.[10-12] Hyperbaric oxygen therapy, but not at 1.0 ATA, has been shown in animal studies to significantly reduce lipid peroxidation.[7] Clinical reports attest to the efficacy of hyperbaric oxygen therapy in reversing the severe neurologic and cardiovascular deficits seen in carbon monoxide poisoning.[13-15]

"Delayed neurologic sequelae" is a term used to describe the neurologic signs and symptoms that develop after the patient recovers from acute carbon monoxide poisoning. The incidence in patients not treated with hyperbaric oxygen ranges from 10% to 20%, compared with 0% to 4% in treated patients.[16,17]

CYANIDE INHALATION—Cyanide poisoning also may result from the inhalation of gases produced by burning synthetic materials. Cyanide is an asphyxiant that blocks oxidative phosphorylation and rapidly leads to death.[18] Many smoke inhalation victims have significantly elevated blood levels of cyanide.[19] Because most clinical laboratories require 6 hours or more to determine cyanide blood level, the diagnosis must be made presumptively if the patient is to survive. The most consistent feature of cyanide poisoning is profound metabolic acidosis, which occurs secondary to the accumulation of lactate.

Standard treatment for cyanide poisoning is the administration of amyl nitrite, sodium nitrite, and sodium thiosulfate,* which are available in a cyanide antidote kit.[20] Oxygen administration is important in the treatment of cyanide inhalation. Reports show that hyperbaric oxygen therapy increases the survival rate in patients who fail to respond to the antidotes.[2,21-23]

*Cyanide Antidote Package, Eli Lilly and Company, Indianapolis, IN 46285.

SUMMARY—Hyperbaric oxygen therapy is an important adjunct in the management of respiratory injuries secondary to smoke inhalation, especially when injury is complicated by inhalation of a toxic chemical such as carbon monoxide or cyanide. For carbon monoxide poisoning, such therapy has become a standard of practice. As more information becomes available concerning the ability of hyperbaric oxygen to reduce reperfusion injuries, we anticipate that this therapy will become a standard of practice for managing smoke inhalation injuries and cyanide poisoning as well. **PGM**

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