HYPERBARIC OXYGEN TREATMENT FOR CARBON MONOXIDE INTOXICATION ACQUIRED IN THE SEALED ROOM DURING THE PERSIAN GULF WAR

YOHAI ADIR, HAIM BITTERMAN, SHAHAR KOL and YEHUDA MELAMED

Israel Naval Hyperbaric Institute, and Israel Defense Forces Medical Corps, Haifa, Israel

ABSTRACT. During the recent Persian Gulf war, the civilian population in Israel was frequently instructed to stay in sealed rooms in preparation for a possible chemical missile attack. The war broke out in mid-winter, and in many instances it was necessary to heat the rooms. The use of open fires or malfunctioning heating appliances inside sealed rooms could create ideal conditions for carbon monoxide (CO) poisoning. Six patients with CO intoxication resulting from confinement inside sealed rooms were referred for hyperbaric oxygen (HBO) treatment. Indications for HBO therapy were loss of consciousness and metabolic acidosis. The treatment protocol consisted of 90-min exposure to 100% oxygen at 2.5 atmospheres absolute (ATA), with repeated exposures when required. All patients made a full recovery. The risk of CO poisoning should be taken into consideration and should determine the selection of heating devices for future use in similar circumstances. *Isr J Med Sci* 1991;27:669–673

Keywords: Persian Gulf war; carbon monoxide; hyperbaric oxygenation; confined atmosphere; oxygen

During the period preceding the Persian Gulf war, the Israel Defense Forces prepared the civilian population for the possibility of a chemical missile attack. Masks providing protection against poisonous gases were distributed, and the population was instructed to seal a room in each house and to lay in supplies of food and water, a first aid kit, a radio, and anything else that might be required if a prolonged period had to be spent inside the room. The Gulf war broke out in mid-winter, and since most missile attacks took place during the night it became necessary to heat the unventilated sealed room. In these circumstances, the use of an open coal fire and malfunctioning heating devices created ideal conditions for carbon monoxide (CO) poisoning (1–3). The public was not sufficiently aware of the problem of possible CO poisoning, and only after the first case appeared were preventive measures taken. Six patients with CO poisoning were nevertheless referred for hyperbaric oxygen (HBO) treatment. All patients had been exposed to CO inside a sealed room heated by coal, oil, kerosene or gas-fired heating appliances.

Address for correspondence: Dr. Y. Adir, Israel Naval Hyperbaric Institute, POB 8040, 31080 Haifa, Israel.

PATIENTS AND METHODS

During the Gulf crisis (18 January to 28 February 1991), six patients were diagnosed as having acute CO poisoning after staying in a sealed room and were referred to our Institute for HBO treatment. The commonly accepted criteria for the transfer of CO intoxication casualties to the hyperbaric facility are disturbances in the level of consciousness, neurologic findings on examination, changes in the ECG, metabolic acidosis, or a carboxyhemoglobin (COHb) level above 40% (4).

HBO therapy is carried out in a hyperbaric chamber, which is used for the administration of pure oxygen at high ambient pressures. The chamber is filled with compressed air at the treatment pressure,

and the patient breathes 100% oxygen at the same ambient pressure via a mask.

Before the HBO treatment, patients underwent a comprehensive neurological, physical and otolaryngological examination; chest X-ray and ECG were taken, and blood was drawn for blood gases, electrolytes and COHb. HBO was administered in a large chamber provided with medical equipment and facilities required for the management of critically ill patients. The HBO treatment protocol consisted of courses of 90 min breathing 100% oxygen via a demand mask at an ambient pressure of 2.5 atmospheres absolute (ATA) every 8–12 h, until full recovery was achieved.

Three of our patients were males and three were females, and their average age was 15 years (range 2.5–32 years) Table 1. The indications for HBO treatment in our group of patients were loss of consciousness during the acute episode in four patients, and metabolic acidosis in five. Oxygen via a mask was administered as first aid at the site of the accident, and all patients regained full consciousness after 10 to 15 min. All patients were suffering from moderate CO poisoning and required only one HBO session, except for one patient who continued to complain of dizziness and weakness and required an additional treatment session. All patients made a full recovery.

Representative Case Histories

Case 1. A 32-year-old male was found unconscious inside a sealed room. The room was heated by an open gas burner. From the patient's history, an exposure time of approximately 3 h was estimated. Oxygen was administered via a mask at the site of the accident, and the patient was transferred to the local hospital. On reaching the emergency room he regained full mental alertness, but complained of headache, dizziness and weakness. Blood pressure of 110/70 mm Hg, a regular pulse of 82 and a respiratory rate of 18/min were recorded. Physical and neurological examinations were normal. Chest X-ray and ECG trace were within normal limits. Arterial blood gases on admission were: pO_2 78 mm Hg, pH 7.34, pCO_2 36 mm Hg, bicarbonate 22 mEq/*l*, base excess –3 mEq/*l*. The patient was transferred to the Israel Naval Hyperbaric Institute for HBO therapy, where COHb on arrival, 6 h after the exposure, was 15%. He was given two sessions of HBO, each comprising 90 min of 100% oxygen at 2.5 ATA. Recovery was uneventful, and the patient was discharged 1 day later.

Case 2. A 3-year-old boy was brought to the emergency room due to severe headache and vomiting that began after he spent the night in a sealed room heated by a kerosene heater. Physical examination was normal. No pathological neurological findings were noted. Chest X-ray and the ECG were normal. However, arterial blood gas analysis disclosed metabolic acidosis, with pH 7.27, pO_2 80 mm Hg, pCO_2 30 mm Hg, bicarbonate17 mEq/*l*, and BE –6 mEq/*l*. Oxygen was given via a mask and the patient was transferred to the Israel Naval Hyperbaric Institute for HBO therapy. COHb level upon arrival, 9 h after the exposure, was 2.8%. Bilateral ear drum paracentesis was performed in order to prevent barotrauma of the ear during the hyperbaric therapy. The patient was given one session of HBO. He

Table 1. Relevant clinical data of patients with CO poisoning

Case no.	Age (yr)	Sex	Duration of exposure to CO	Level of consciousness at site of accident	Patient's complaints	CoHb level[a] (%)	Metabolic acidosis	ECG	No. of HBO sessions
1	32	M	3 h	Coma	Headache, dizziness, weakness	15.0	+	Normal	2
2	16	F	Overnight	Coma	Headache, nausea, vomiting, confusion	16.0	+	Normal	1
3	14	F	Overnight	Coma	Headache, chest pain	NA	+	Normal	1
4	24	F	Overnight	Coma	Headache, vomiting	4.6	–	Normal	1
5	3.5	M	Overnight	Normal	Headache, vomiting	2.8	+	Normal	1
6	2.5	M	Overnight	Normal	Headache, vomiting	2.0	+	Normal	1

[a]At the Israel Naval Hyperbaric Institute.
CO = carbon monoxide; NA = not available.

recovered fully and was discharged from hospital the same day.

DISCUSSION

Carbon monoxide is one of the five most important neurotoxins for humans, and CO intoxication is the leading cause of death from unintentional poisoning in many countries (1–3). CO is a colorless, odorless and nonirritating gas produced by the incomplete combustion of carbon-containing materials. The effects of CO poisoning are apparent in most body systems, but are most pronounced in areas of high blood flow and oxygen demand (i.e., the brain and the heart) (1). CO has an affinity for hemoglobin 200–250 times greater than that of oxygen, and functions as a competitive inhibitor of oxygen binding to hemoglobin (5). Exposure to CO results in the formation of COHb. Some binding of CO to heme containing protein in nucleated cells also occurs, and about 10–15% of the total body CO is located in extravascular tissue (1,2). COHb causes a left shift of the oxyhemoglobin dissociation curve (6), so that the oxygen which remains bound to hemoglobin is not as readily available to the cells. At the cellular level, CO blocks the intracellular oxygenation system by binding with cytochrome oxidase A_3 (7). Other mechanisms, such as increased brain lipid peroxidation at a concentration sufficient to cause unconsciousness, have recently been suggested as an explanation of a number of clinical observations regarding CO poisoning that are at present poorly understood (8). All of these processes may lead to severe tissue hypoxia.

The presenting symptoms of CO poisoning are often vague and variable. The most common manifestation of mild intoxication is frontal headache. A moderate level of poisoning is usually manifested by weakness, lightheadedness, sleepiness, visual disturbances, palpitations, nausea, vomiting and syncope. The signs of severe poisoning are tachypnea, confusion, disorientation, unconsciousness and convulsions. Depressed cardiac and respiratory function and death may ensue (1–4).

The rationale for HBO treatment is threefold. At high ambient pressure, the amount of oxygen dissolved in the plasma is greatly increased and is sufficient to meet most of the tissue oxygen requirements at rest (9). HBO also considerably shortens the half-time for the elimination of COHb. This has been shown to be 320 min when breathing air at atmospheric pressure, 80 min when breathing 100% oxygen at atmospheric pressure, and dropping to as little as 23 min during 100% oxygen breathing at 3 ATA (10). In addition, it has been demonstrated that oxygen exerts a beneficial effect on cerebral edema and intracranial pressure (11). HBO is also effective in preventing the late neurologic and psychiatric effects of the poisoning (12).

The clinical improvement observed in our six patients was most probably due to the liberation of CO from the tissues and the reversal of residual hypoxic damage. Currently accepted indications for HBO therapy in CO poisoning include alterations in mental status or neurological findings, circulatory collapse, pulmonary edema, ECG signs of myocardial ischemia, metabolic acidosis, or arterial carboxyhemoglobin levels above 40% (4). HBO treatment is not indicated in mild cases of CO poisoning, in which the highest available oxygen percentage should be given via a mask until resolution of all symptoms. The criteria for the selection of patients for HBO treatment should also take into account the risk of delayed neuropsychiatric deterioration, which tends to increase in relation to the duration of the state of unconsciousness during the anoxic episode. Myers et al. (12) demonstrated the beneficial effect of HBO therapy on neurologic status in a follow-up of 71 adults with CO poisoning. In this series, HBO therapy prevented the development of delayed neuropsychiatric sequelae. This contrasts with the work of Smith and Brandon (13) who found a 33% incidence of personality deterioration and a 43% incidence of memory impairment in 63 patients not treated by HBO. The current policy is therefore to use HBO therapy in every case of metabolic acidosis or disturbance in the level of consciousness at the site of the accident, even if the patient regains consciousness and irrespective of COHb levels.

The first case we encountered of CO intoxication as the result of being in a sealed room heated by an open coal fireplace occurred 6 days after the outbreak of the Persian Gulf war. After this case came to the notice of the authorities, action was taken to avoid repetition of the incident. Radio and television broadcasts explained the risks and dangers of CO poisoning, as well as means of prevention. (It should be borne in mind that none of the commercially available gas masks provides protection against CO.) One important factor that helped reduce the incidence of CO intoxication was the relatively mild winter, which to a certain extent eliminated the need to use heating appliances. Nevertheless, after the first case, another five patients with CO poisoning were referred for HBO therapy.

In conclusion, the risk of CO poisoning in the sealed room was not properly explained to the public before the outbreak of the war. The publicity given to the first case and the resultant increase in public awareness of the problem, together with the clement

weather, lowered the expected number of cases of CO poisoning. The risk of CO poisoning should be borne in mind in the event of similar attacks necessitating the use of sealed rooms in the future, and the public should be prepared accordingly.

REFERENCES

1. Dolan MC. Carbon monoxide poisoning. *Can Med Assoc J* 1985;**132**:392–396.
2. Marzella L, Myers RAM. Carbon monoxide poisoning. *Am Fam Physician* 1984;**34**:186–194.
3. Olson KR. Carbon monoxide poisoning: mechanism, presentation and controversies in management. *J Emerg Med* 1984;**1**:233–243.
4. Gozal D, Ziser A, Shupak A, Melamed Y. Accidental carbon monoxide poisoning. Emphasis on hyperbaric oxygen treatment. *Clin Pediatr* 1985;**24**:132–135.
5. Douglas CG, Haldane JS, Haldane JBS. The laws of combination of haemoglobin with carbon monoxide and oxygen. *J Physiol (Lond)* 1912;**44**:275–304.
6. Roughton FJW. Transportation of oxygen and carbon dioxide. In: Fenn WO, Rann H, eds. Handbook of physiology, Section 3: Respiration, vol. 1, Washington, DC: American Physiological Society, 1964:767–791.
7. Goldbaum LR, Orellano T, Dergal E. Mechanism of the toxic action of carbon monoxide. *Ann Clin Lab Sci* 1976;**6**:372–376.
8. Thom SR. Carbon monoxide-mediated brain lipid peroxidation in the rat. *J Appl Physiol* 1990;**68**:997–1003.
9. Kindwall EP. Carbon monoxide and cyanide poisoning. In: Davis JC, Hunt TK, eds. Hyperbaric oxygen therapy. Bethesda, MD: Undersea Medical Society, 1977:177–190.
10. Pace N, Strajman E, Walker EL. Acceleration of carbon monoxide elimination in man by high pressure oxygen. *Science* 1950;**111**:652–654.
11. Sukoff MH, Hollin SA, Jacobson JH II. The protective effect of hyperbaric oxygenation in experimentally produced cerebral edema and compression. *Surgery* 1967;**62**:40–46.
12. Myers RAM, Snyder SK, Linberg S, Cowley RA. Value of hyperbaric oxygen in suspected carbon monoxide poisoning. *JAMA* 1981;**246**:2478–2480.
13. Smith JS, Brandon S. Acute carbon monoxide poisoning. Three years experience in the defined population. *Postgrad Med J* 1970;**46**:65–70.