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Selected Topics Toxicology

TREATMENT OF SMOKE INHALATION BY HYPERBARIC OXYGEN

George B. Hart, MD, FACS,* Michael B. Strauss, MD, FACS, AAOS,† Paul A. Lennon, MD, FRACP,‡ and Daniel D. Whitcraft III, MD, FACEP§

*Director, †Associate Director, ‡Associate, Baromedical Department; §Director, Emergency Department, Memorial Medical Center of Long Beach, California
Reprint address: George B. Hart, MD, FACS, Memorial Medical Center of Long Beach, PO Box 1428, Long Beach, CA 90801-1428

☐ **Abstract—Five patients with smoke inhalation from house fires presented to the hospital in a comatose state. Carboxyhemoglobin levels were elevated in all five patients, mean = 32% ± 6. Arterial blood gases revealed the following means: pH 7.16 ± 0.06; P_{CO_2} 35 mm HG ± 10.5; HCO_3 12.6 mEq/L ± 0.07; base excess −16 mEq/L ± 1.58; P_{O_2} 353 mm Hg ± 149; O_2 saturation 66% ± 5.5. The patients were presumed to have both cyanide and carbon monoxide intoxication and were treated with the cyanide antidote kit and hyperbaric oxygen (HBO). Four of five patients awoke within 15 minutes of reaching maximum pressure and remained neurologically intact thereafter. The fifth patient died one week later. Cyanide blood levels drawn prior to treatment revealed a mean of 1.62 μg/mL ± 1.44. The highest cyanide level was 3.9 μg/mL (the death) and the lowest 0.35 μg/mL. We conclude that smoke inhalation can result in acute cyanide poisoning and that hyperbaric oxygen is a useful adjunct in the treatment of smoke inhalation.**

☐ **Keywords—smoke inhalation; hyperbaric oxygen; cyanide; carbon monoxide**

Introduction

The inhalation of toxic products from fires causes approximately 6,400 (80%) of the fire fatalities occurring annually in the United States.[1] It has been estimated that 77% of deaths having combined burns and smoke inhalation should have survived the burn injury.[2] Of these deaths, 76% had respiratory involvement, 48% had smoke poisoning or asphyxia, 50% had carbon monoxide intoxication, and 27% had pathologic pulmonary findings.[2]

We reported previously[3] that there was an apparent reduction in respiratory morbidity in burn patients treated with hyperbaric oxygen (HBO). This observation was amplified in ventilatory studies by Grossman.[4] Animal studies demonstrate that HBO significantly improves survival in chemical pneumonitis.[5,6] HBO has been shown to rapidly eliminate carbon monoxide in humans[7] and has been advocated as the best possible modality for treating severe carbon monoxide intoxication, as there is improvement in survival when applied early (within the first six hours)[8] and the late sequelae (neuropathies and encephalopathies) avoided.[9] Recent reports reveal several intoxicants in smoke such as hydrogen cyanide,[10] phosgene, chlorine, acrylonitrile, and others.[11] Animal studies[12-14] indicate

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HBO is effective in treating cyanide intoxication. There is only one brief report where HBO was used in treating a human cyanide victim.[15]

Therefore, recognizing that smoke contains pulmonary irritants (phosgene, chlorine) and cytotoxic agents (carbon monoxide, cyanide, and acrylonitrile), we undertook the following prospective, although uncontrolled, study of patients arriving unconscious from a closed-space fire.

Method

All patients arriving unconscious from a closed-space fire were intubated and placed on 100% oxygen. Arterial blood gases were drawn and the following tests performed: Po_2, CO_2, pH, base excess, carboxyhemoglobin levels (IL 282 Cooximeter, Instrumentation Laboratories, Inc., Lexington MA 02173), and cyanide levels (Conway diffusing procedure). Patients received the cyanide antidote if the base deficit exceeded -10 and the pH <7.3 without waiting for the cyanide level, which requires at least six hours to perform. The cyanide antidote (Eli Lilly and Company, Indianapolis IN 46285) used is: (1) amyl nitrite perls broken over airway $\times$ 15 seconds each, (2) sodium nitrite 300 μg (3% solution) intravenously over a two- to four-minute period, (3) sodium thiosulfate 12.5 g (25% solution) intravenously over a 10-minute period for adults. The nitrite (3% solution), 0.39 mL/kg, and sodium thiosulfate (25% solution), 1.95 mL/kg, are both given carefully while monitoring the blood pressure and heart rate. Chest radiographs were taken at admission. The following baseline studies were performed on venous blood samples: complete blood count, blood alcohol, cardiac enzymes, and electrolytes. Each patient was placed in a monoplace hyperbaric chamber and treated at two atmospheres absolute (ATA) for 90 minutes. The patients were hyperventilated using a ventilator designed for use with the monoplace chamber. Electrocardiograms and blood pressures were monitored during the treatment. Each patient was placed in soft restraints prior to HBO. A bolus of sodium bicarbonate solution was given to correct the acidosis preceding HBO based on the following formula: 1 mEq $NaHCO_3$/kg body weight per one tenth in the pH below 7.3.

Results

Five patients (three males and two females, average age 27 years) arrived comatose from May 1981 to May 1983, with physical evidence of smoke inhalation (carbonaceous deposits in nasal passages and oropharynx). All exposures occurred secondary to closed-space fires in their homes. The youngest was 9 months old and the eldest 68 years. After receiving the cyanide antidote, three patients were decerebrate, one decorticate, and one reactive to pain but unable to follow commands. The admitting laboratory studies (Table 1) reveal the following means: pH 7.16 ± 0.06, Pco_2 35 mm Hg $\pm$ 10, HCO_3 12 ± 2 mEq/L, base deficit -16 mEq/L $\pm$ 1.51, Po_2 353 mm Hg $\pm$ 149, O_2 saturation 66 ± 6, percent carboxyhemoglobin (COHb) 32 ± 6, and cyanide level 1.62 μg/mL $\pm$ 1.4. The blood alcohol levels were elevated in the three adults, average 220 mg/dL $\pm$ 80 (Table 2). Only one patient had significant burns that required grafting (3½-year-old boy with 30% total body surface burn, 10% full thickness).

Table 2 represents laboratory data drawn within an hour of the first HBO treatment and reveals the rapid clearing of the cytotoxic gases as well as normal arterial blood gases. Methemoglobin levels drawn one to four hours after HBO were $<1.5\%$.

Electrocardiograms in all patients demonstrated a tachycardia (average 130/minute, range 110 to 170 beats per minute), and revealed depressed T waves in all leads (Figure 1). The heart rate was reduced during HBO to an average of 90 beats per minute (high 110, low 70). There was one death, a 43-year-old woman who received cardiopulmonary resuscitation at the scene by par-

Table 1. Admitting Laboratory Values

Patient	Age	Sex	Arterial Blood Gases						ETOH (mg/dL)	COHb (%)	Cyanide (μg/mL)	Time From Smoke Exposure to HBO	Outcome
			pH	PCO_2 (mm Hg)	HCO_3 (mEq/L)	Base Excess (mEq/L)	PO_2 (mm Hg)	O_2 (Sat)					
1	43	F	7.18	32	11	−16	359	68	312	32	3.9	5 h	Expired 4 days after admission
2	68	M	7.12	35	11	−18	394	60	189	38	1.8	45 min	Survived neurologically intact
3	3½	M	7.16	37	13	−15	123	69	0	29	0.35	45 min	Survived neurologically intact
4	9 mo	M	7.10	54	16	−14	351	73	0	24	0.42	45 min	Survived neurologically intact
5	21	F	7.26	26	12	−17	539	61	160	38	1.65	3 h	Survived neurologically intact

Table 2. Laboratory Values After First HBO Treatment

Patient	Age	Sex	Arterial Blood Gases						COHb (%)	Cyanide (μg/mL)
			pH	PCO_2 (mm Hg)	HCO_3 (mEq/L)	Base Excess (mEq/L)	PO_2 (mm Hg)	O_2 (Sat)		
1	43	F	7.33	32	16	−8	195	100	0	<.15
2	68	M	7.33	36	18	−6	144	99	0	<.15
3	3½	M	7.41	33	20	−3	329	100	0	<.15
4	9 mo	M	7.37	43	24	−1	76	73	0	<.15
5	21	F	7.43	33	21	−2	163	98	0	<.15

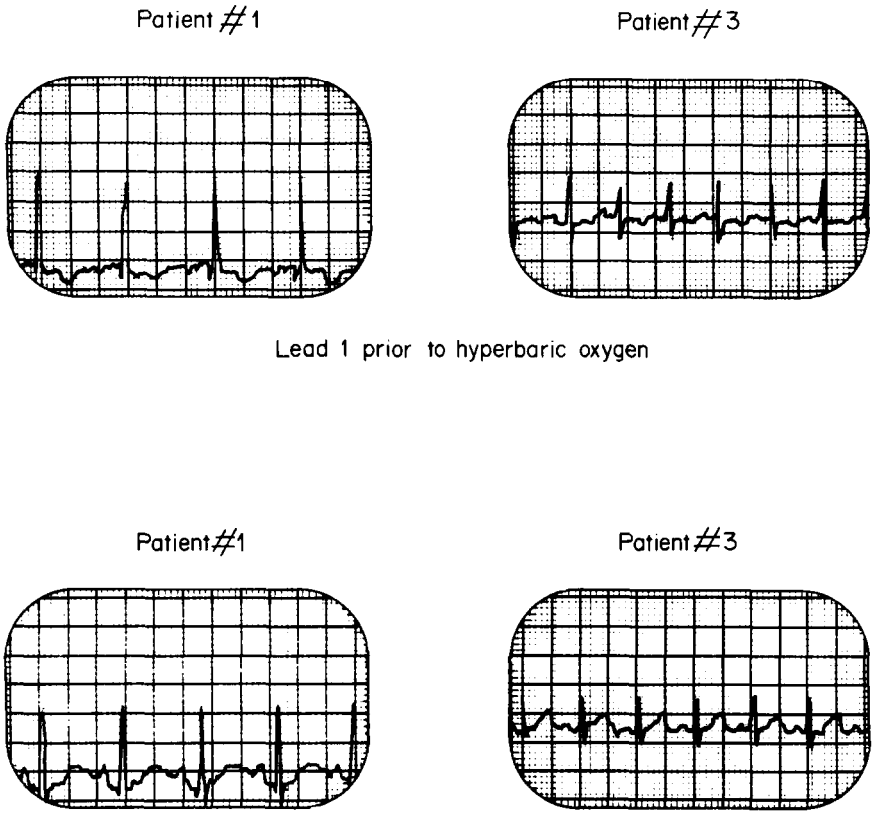

Figure 1. Electrocardiograms of patients.

amedics. The patient died four days following admission, secondary to brain death. The four other patients awoke in the first 15 minutes at 2 ATA oxygen. Three of the five were extubated after the first HBO treatment, except the 3½-year-old boy, and the patient who subsequently died. Chest x-ray studies revealed marked pulmonary congestion in all five prior to HBO, clearing in three within 12 hours, and resolving more in two others. The four were discharged neurologically intact with an average hospital stay of 14 days, the shortest, 4 days (9-month-old male infant); and the longest 28 days (3½-year-old boy who had significant third degree burns requiring grafting). Only one patient received steroids (43-year-old woman). All five patients received antibiotics prophylactically (cephalosporins in the adults and ampicillin in the children) to avoid opportunistic bacterial pneumonia (none occurred).

Of interest is the 21-year-old woman (patient 5) exposed to a closed-space fire in a bedroom, and her bedmate, a 23-year-old man (active duty military). He was transferred to a military hospital and was shown to have similar clinical and laboratory findings. He died within the next 24 hours without regaining consciousness. He had received the antidote for cyanide, but did not receive HBO.

Discussion

It has been argued that giving the cyanide antidote kit is potentially dangerous because of methemoglobin formation. This is particularly true in those patients with signifi-

cant carboxyhemoglobin levels. However, at 2 ATA oxygen there are 4 volumes percent oxygen dissolved in the circulating plasma, and as shown previously,[16] animals who are experimentally exsanguinated may survive with HBO if the circulating volume is replaced by an acellular perfusate. Additionally, HBO is shown in an animal model to significantly reduce the methemoglobin levels and mortality from methemoglobinemia.[17]

Conversely, there is an in vivo report[18] noting no advantage of HBO to atmospheric oxygen in the treatment of cyanide intoxication. However, the pressures of oxygen used in this report (4 ATA $O^2 \times 2$ hours) were excessive to the prior reports[12,14] and may have contributed to the morbidity and mortality.

Smoke inhalation is toxic to respiratory function in three ways: (1) as an irritant (ie, chlorine, phosgene, and ammonia), (2) by decreasing effective oxygen transport: by carbon monoxide combining with hemoglobin, and (3) at a cellular level where cyanide and carbon monoxide combine with cytochrome oxidase, affecting intracellular metabolism. The patients herein reflected the above findings, as they demonstrated severe metabolic acidosis, had elevated cyanide and carbon monoxide levels, and showed pulmonary congestion on radiographs of the chest.

We recommend the administration of the cyanide antidote kit when patients present from a closed-space fire with the findings of metabolic acidosis, elevated carboxyhemoglobin levels, and a decrease in arterial oxygen saturation, as present laboratory methods require four to six hours for quantitative measurement of cyanide. When the carboxyhemoglobin levels are extremely elevated (ie $>40\%$), it may be prudent to administer the cyanide antidote kit only after the patient is already at pressure in the hyperbaric chamber.

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