What is Clinical Smoke Poisoning?

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In this 13-year study, 51 patients were admitted with the primary diagnosis of "smoke poisoning," "carbon monoxide (CO) poisoning" or "respiratory burns." Forty patients (78%) had diagnosis of smoke poisoning with minor or no skin burns. The study indicated that clinical diagnosis of CO poisoning cannot be made reliably without carboxyhemoglobin (COHg) determination and that smoke poisoning patients often had CO poisoning. Seventeen of 19 smoke poisoning patients (89%) had CO poisoning above COHb levels of 15% saturation. Carbon monoxide was successfully removed from the blood by improving alveolar ventilation and oxygen concentration. However, there were 2 smoke poisoning deaths as the result of gaseous chemical injury. There was a correlation coefficient of 0.87 between initial COHg levels and patients' hospital days primarily determined by patients' pulmonary complications. Since CO is non-irritating, COHb levels may be used as an additional indicator of suspected pulmonary injury by noxious combustion gases.

THE SCIENTIFIC DEFINITION of smoke and the medical meaning of "smoke poisoning" or "smoke inhalation" are not well understood. The purpose of this paper is to attempt to define more specifically "clinical smoke poisoning" as we now understand it. "Respiratory burn" pathophysiology has been described earlier.[22]

In 1962 Phillips and Cope[10] labeled respiratory tract damage as a "principal killer" in burn victims. In 1967, Stone reported "respiratory burns" in 15% of 197 patients some of whom may have had concomitant smoke poisoning.[18] Because of recent recognition and difficulty in differentiation between respiratory burn and smoke poisoning, there have been no available clinical statistics concerning incidences of the two conditions or the incidence of their coexistence.

Smoke poisoning and/or asphyxia was the primary cause of death in 30% of all fire fatalities in New York City during the years 1966 and 1967.[23] Respiratory tract involvement was found in 70% of deaths under 12 hours and in 46% of victims who survived more than 12 hours. 53.5% of 185 victims who survived less than 12 hours had smoke poisoning and/or asphyxia as the only cause of death. Significantly, 79% of those having primary diagnosis of smoke poisoning and/or asphyxia had actual carbon monoxide poisoning. Twenty-four per cent of these patients had lethal levels of CO (above 50% carboxyhemoglobin saturation).[23]

Since almost one-third of fire fatalities are due to smoke poisoning, and for many years to come combustion will remain the main source of energy, we must regard smoke and fumes of combustion as major threats to life. Therefore, it behooves us to understand better the acute clinical "smoke poisoning" which in turn may help our understanding of the subtle chronic effects of air pollution and cigarette smoking. Through a better understanding of "smoke poisoning," we may find effective and expeditious methods of treatment and prevention.

Materials and Methods

In the 13-year period 1960–1972, 51 patients were admitted with the primary diagnoses of "smoke inhalation," "CO poisoning" or "respiratory burns." Patients whose main problem was their body surface burns were not included in this study. Thirty of these patients were admitted during 1960–1970 and 21 patients in the two years thereafter. Their records were evaluated critically with respect to admission diagnoses, laboratory analyses, hospital course and treatment.

Results

These 51 patients were admitted through the emergency services of Columbia-Presbyterian Medical Center. Forty (78%) fell in the category of "smoke poisoning" on the basis

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PRIMARY CLINICAL DIAGNOSIS	CASES	BSA BURNS		CLINICAL EVIDENCE OF LATE SEQUELAE		LABORATORY DIAGNOSIS OF CO POISONING	DEATHS
		Subjects % burn	Average % burn	Neurologic	Pulmonary		
SMOKE POISONING	40	3% 1% 10% 10% 15%	0.97	1	6	16	2 [2 days and 12 days]
CARBON MONOXIDE POISONING	6	0	0	3	0	1	1 [7 hours]
RESPIRATORY BURN	5	30% 20% 5% 4% 3%	12·4%	0	2 [no follow up in 1]	0	2 [4 days and 8 days]
TOTAL	51		2%	4	8	17	5

FIG. 1. Clinical results 1960–1972 of primary inhalation injuries.

of having been exposed to the smoke of a fire with minor or no body burns. There were six primary clinical diagnosis of carbon monoxide poisoning and five patients with the primary diagnosis of respiratory burns by examination. The ages of these patients ranged 6–76 years and there were 37 males and 14 females (Fig. 1).

During the decade of 1960–1970 there was no blood carbon monoxide determination because emergency room equipment* for the test was not available. With the availability of a quick laboratory determination in the 2-year period that followed (1971–1972) all 21 patients admitted for possible inhalation injuries from a fire were tested for carbon monoxide poisoning.

Clinical diagnosis of CO poisoning without the help of blood CO determination was made in 5 patients during 1960–1970 primarily on the basis of history and neurologic sequelae. It is interesting to note that clinical diagnosis of acute CO poisoning was made on the basis of cherry-red skin in only one patient before the laboratory report of a blood CO saturation of 51%. Whereas 17 of 21 (80%) had acute CO poisoning at above 15% carboxyhemoglobin saturation during 1971 and 1972.

This study also shows that where clinical diagnosis of "smoke inhalation" or "smoke poisoning" is made, if blood CO level is determined, 16/19 (84%) will reveal some degree of carbon monoxide poisoning (CO saturation above 15%). Nine patients had carboxyhemoglobin saturation of 29% or more.

Ten patients (20%) had sustained burns of face, upper trunk and extremities. Their burns were mostly 1st and 2nd degree burns. Patients with the primary diagnosis of smoke poisoning had an average of 0.97% body burns whereas respiratory burn patients had an average of 12.4% body surface burns. Only one of these patients had a third degree burn necessitating a small skin graft (Fig. 1).

Ten patients (25%) with the primary diagnosis of smoke poisoning and all 5 patients with the diagnosis of respiratory burns had some visible evidence of respiratory tract injury ranging from erythema and blisters of the orophyarynx to congestion and edema of the tracheobronchial tree as observed by direct or indirect laryngoscopy or broncoscopy. In only three patients did the initial chest X-ray reveal pulmonary edema. Although only three admission chest X-rays showed initial pulmonary changes, a total of 11 patients (28%) developed either pulmonary edema or X-ray evidence of pneumonitis during their hospitalization. The rest of the patients were treated for atelectasis, probable pneumonitis or bronchitis on the basis of fever and their chest physical findings.

Since 1970, the mode of initial respiratory resuscitation has been influenced greatly by the admission arterial blood gases and CO determinations. Six patients required MA-1 respirator; three resuscitation via tracheostomy, and in three patients, nasotracheal or orotracheal tubes were used. Seven patients were treated with IPPB, five of whom required tracheostomy. The remaining cases received humidified oxygen by nasal catheter or oxygen tent for varying periods of time.

Fourteen patients were placed on prophylactic antibiotics, usually penicillin, though ampicillin, keflin and tetracycline were also utilized. An additional six patients developed significant pulmonary infection requiring specific antibiotic therapy.

Ten patients were treated with bronchodilators, and three patients required digitalis and or diuretics for delayed pulmonary edema and congestive heart failure in elderly patients. Eight patients of 40 smoke poisoning and one patient out of five respiratory burn and none of the CO

* CO-Oximeter, Instrumentation Laboratory, Inc., Watertown, Mass 02172.

poisoning patients received corticosteroids. The steroids were given in 2–4 divided doses within the first 24–48 hours only.

The overall mortality was 9.8%, five patients: 3 cardiorespiratory arrests and 2 with intractable pulmonary edema (4 days post injury) and overwhelming pulmonary infection (12 days post injury) respectively (Fig. 1). There were two deaths in 40 smoke poisoning patients, one death in six carbon monoxide poisoning and two deaths in five respiratory burns.

The carbon monoxide clearance from the blood was followed with serial determinations of carboxyhemoglobin levels. The two patients who had the highest CO levels, 82% and 51%, both on MA-1 respirators with 100% O_2, had rapid clearance of CO from the blood within four hours. Those patients who had CO levels below 30% were treated fairly effectively with nasal oxygen and ventimask, bringing their blood CO levels to 10–15% within four hours (Fig. 2).

It is also interesting to note that most patients who were hospitalized for longer than 2 days had initial CO levels above 15%. There seems a definite relationship between the initial blood CO level and the patient's length of hospitalization (Fig. 3). The patient with 82% carboxyhemoglobin who died 7 hours after admission with fatal CO poisoning and another patient whose hospital stay was prolonged because of skin grafting were excluded in the calculation of correlation coefficient. The 0.87 correlation coefficient between initial blood CO levels and hospital days indicated a fairly good correlation between initial blood CO levels and pulmonary damage as well, since patients length of hospital stay was primarily determined by the degree of pulmonary complications as a result of exposure to smoke. Since CO is not noxious, and as we have shown in dog experiments,[20] does not cause pulmonary edema, the reason for this relationship may be nothing more than CO acting as an indicator of other gaseous exposure which is injurious to the lungs, but not detected in blood or other tissues. Therefore, we have clinically used carboxyhemoglobin levels as a possible index of pulmonary injury by other injurious gases which coexist with carbon monoxide in smokes of structural fires. Smoke poisoning patients who have clinical evidence of tracheobronchial tree or pulmonary injury and those patients without clinical evidence of such injury but who have carboxyhemoglobin levels of 20% or more, are usually hospitalized for observation for at least 24 hours if the blood CO determination was made within one hour of acute exposure to fire products. It must be pointed out that in many smoke poisoning patients evidence of pulmonary injury may not become clinically obvious until 12–48 hours. A chart has been constructed on the basis of clinical blood CO clearance curves and the experimental data. From such a chart one can interpolate the approximate blood CO levels at the time of exposure if the delay time and emergency blood CO determination are known (Fig. 4).

Discussion

Clinical

We believe that the significance and diagnosis of inhalation injuries in fire victims, by and large, have been underestimated and overlooked. The inhalation injury may be predominantly "smoke poisoning" without significant body

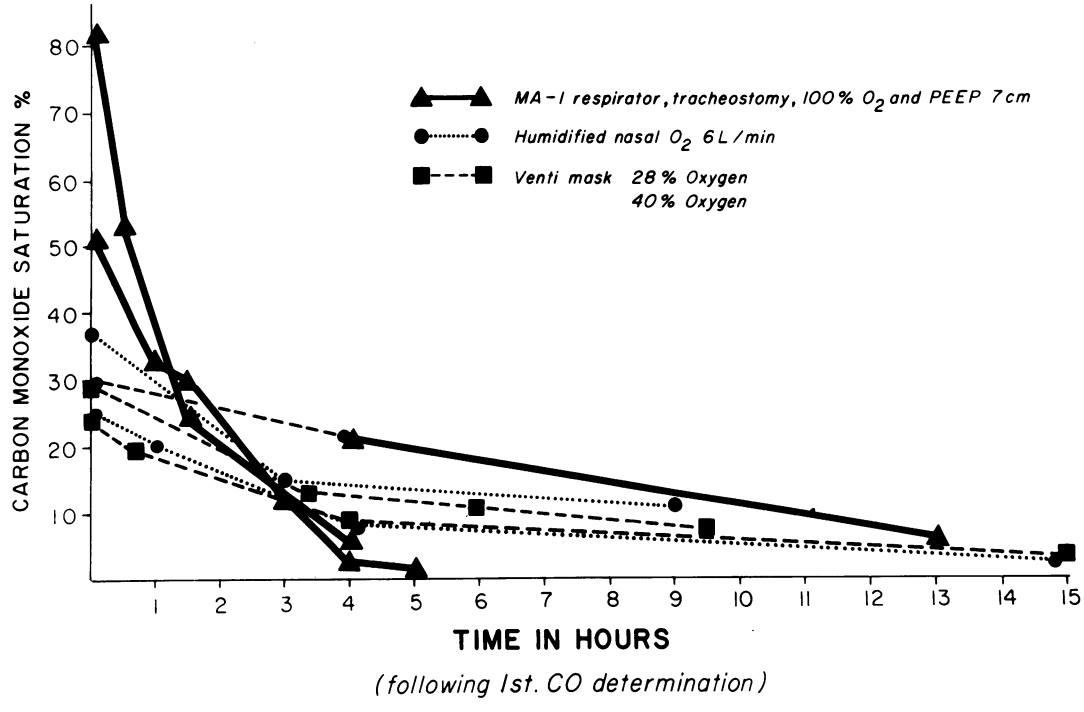

FIG. 2. Carbon monoxide disappearance in blood of smoke poisoning patients.

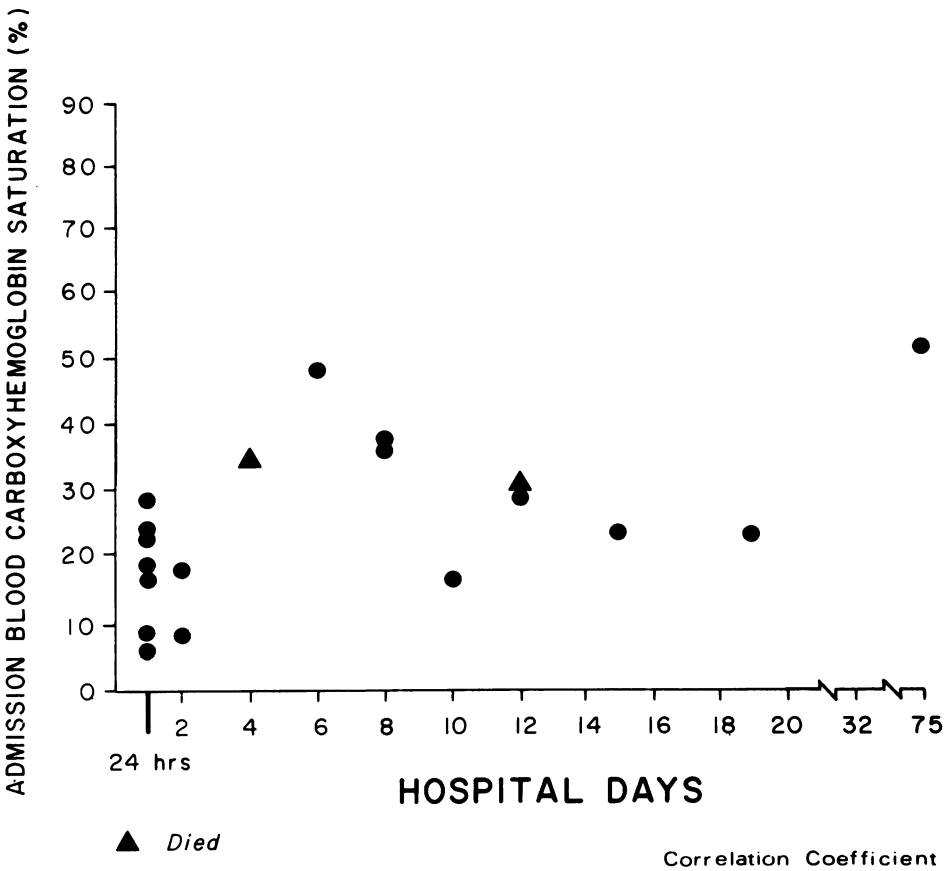

FIG. 3. Correlation of initial carboxyhemoglobin saturation and patients hospital stay based primarily on pulmonary complications.

surface burns and appears to be primarily a chemical injury to tracheobronchial tree and lung parenchyma. Such injury can extend to the peripheral respiratory tissues and rapidly emerge as dyspnea with wheezes and rales. Whereas in the "respiratory burn" occurring in patients with significant burns of anterior chest, neck, face, mouth and/or nose with singed vibrissae, the thermal energy seems to inflict the primary injury. This injury often is central, *e.g.*, involving upper respiratory tract, pharynx larynx, trachea and probably proximal major bronchi. Blood carboxyhemoglobin level may have no relevance in such primary injury. Pathophysiology of this type of injury has been described in another publication.[22] No doubt there are some patients who have a combination of chemical gaseous and thermal injuries to the respiratory tissues. The intention of this paper is to discuss the "smoke poisoning" as the primary injury.

From our studies it appears that the clinical smoke poisoning or "smoke inhalation" is often a chemical respiratory injury with concomitant carbon monoxide poisoning. This study shows that visible respiratory tract damage is seen in about 25% of smoke poisoning cases and in five out of five (100%) respiratory burn patients. Because of low specific heat of gases the thermal injury does not seem to proceed beyond the major bronchi,[9] whereas chemical gas injury easily reaches the alveolar level. It is thus interesting that most tracheostomies were done in respiratory burn patients for upper airway obstruction and in those with chemical injury of smoke poisoning, tracheostomy was done after prolonged intubation (longer than 5 days) and ventilatory support for patchy atelectasis, interstitial edema, and/or excessive secretions. There is no doubt that one should avoid doing tracheostomies when it is not absolutely necessary since it acts as an insult to injury.[21]

The study clearly points out as did the autopsy studies,[23] that clinical diagnosis of carbon monoxide poisoning on the basis of cherry coloring of skin or mucus membranes is unreliable. Therefore, only by blood CO determinations or blood oxygen content determinations that we will be able to know that carboxyhemoglobin is the cause of hypoxia in these patients and therefore treat them accordingly. The treatment is directed to improve the alveolar ventilation and increase the alveolar oxygen tension so that by mass action the oxygen will replace the CO on the hemoglobin molecule.[1,2,8,11,12,15] No doubt the vigor of treatment will vary with the extent of carbon monoxide poisoning. There have been good results reported with rapid clearance of CO from the blood in hyperbaric chambers.[16,17]

In smoke poisoning patients who had very high carboxyhemoglobin saturation 82% and 51% with associated clinical interstitial edema, intubation (naso or orotracheal) and use of respirators with PEEP were indicated for both

clearance of carbon monoxide and the treatment of hypoxia and interstitial edema. Most of our smoke poisoning patients seemed to rid themselves of CO fairly adequately which may be due to their short exposure time in high concentrations of CO. Their problem, with the exception of one patient, seemed to be injury to respiratory tissue by probably other noxious and irritating gases than the effect of CO per se. The one patient who had carboxyhemoglobin saturation of 82% died 7 hours after admission despite vigorous resuscitation as a result of CNS and myocardial damage. Possibly hyperbaric (2-2½ atmosphere) oxygen chamber may have been of benefit in this case.

The evaluation of late sequelae following inhalation injury was difficult to determine since many of the patients admitted were not seen in the hospital prior to their emergency admission. In the followup clinic visits, six of 40 smoke poisoning patients were felt to have some pulmonary disease such as bronchitis, asthma, and amphysema. Two of five respiratory burn patients similarly demonstrated some mild degree of pulmonary ailment and/or disease. There was one of 40 smoke poisoning patients and three of six carbon monoxide poisoning patients with neurologic sequelae such as residual encephalopathy (poor mentation, choreoathetosis, hemiparesis) and/or peripheral neuropathy. Four of these patients were electively admitted primarily for neurologic evaluation of previous furnace and stove carbon monoxide poisoning.[4]

In spite of controversy in regard to the use of steroids in inhalation injuries, it is our impression that patients with smoke poisoning who sustain peripheral respiratory injury may benefit from 2–4 doses of steroids within the first 24–48 hours, possibly helping to reduce interstitial edema and more significantly to release bronchial and bronchiolar spasm. In respiratory burns, on the other hand, the injury is proximal and central with possible break of respiratory mucosal barrier against invasive infection for which little would be gained by use of steroids and may be harmful since infection plays a major role in such injury. Consequently, in respiratory burns, we have recommended use of penicillin against B-hemolytic streptoccus for the first 3 or 4 days and then the use of proper antibiotics according to culture and sensitivity. Whereas in the case of smoke poisoning since mucosal break is not common initially, the problem is peripheral with bronchospasm, edema, atelectasis and increased secretions. In such cases, therefore, proper antibiotics are only used on the basis of culture and sensitivity in established infections.

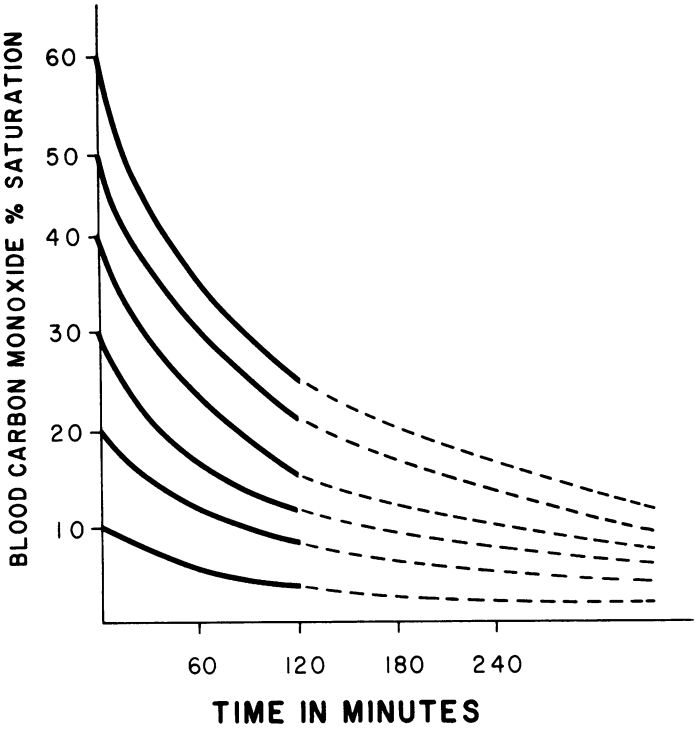

FIG. 4. Carbon monoxide disappearance from blood with spontaneous breathing.

Experimental

In order to learn more as to what smoke poisoning was etiologically as well as pathophysiologically, we had conducted several sets of experiments. In one set we exposed anesthetized mongrel dogs to standardized smokes of wood and kerosene. To our surprise, kerosene smoke neither killed nor produced pulmonary edema whereas wood smoke killed 50–60% of the animals and caused heavy lungs with pulmonary edema and congestion.[20] It was also found that neither carbon monoxide nor soot *per se* caused pulmonary edema or death in animals following resuscitation. Therefore, it was concluded that an invisible noxious gas or gases were responsible for pulmonary damage and death in animals. When wood and kerosene smokes were analyzed by mass spectrometry, gas chromatography and chemical analyses, there were significant quantitative differences in only two gases; carbon monoxide and aldehydes. There was 10 times more carbon monoxide and 15 times more aldehydes in wood smoke than in kerosene smoke. Since carbon monoxide was known and was shown in the above mentioned experiments as non-irritating, aldehydes were considered the likely etiologic gases in the production of exudative pulmonary edema. Short chain aldehydes are well known irritants of mucus membranes in industrial toxicology. In experimental animals these aldehydes have caused pulmonary edema and death. For example, acrolein, a three carbon aldehyde compound in a concentration of 5.5 ppm (parts per million) has been shown to cause irritation of the upper respiratory tract and death with pulmonary edema occurring in a few minutes at a concentration of 10 ppm.[3] In human volunteers, inhalation of acrolein at concentration of 0.805 ppm causes lacrimation and irritation of all exposed mucous membranes.[14] The industrial maximal allowable (MAC) concentration of acrolein is 0.1 ppm.[13] Acrolein was present in wood smoke as 50 ppm and in

kerosene smoke as 1 ppm. It is likely that the agents causing tracheo-bronchial and pulmonary parenchymal damage of smoke poisoning in man are also the aldehydes which are found in large quantities in smoke and combustion of wood, cotton, furniture and non-synthetic structural materials.[19]

A mechanism accepted as the basis for the partially irreversible reaction between aldehydes (e.g., formaldehyde) and an amino acid functional group is regarded as being responsible for processes as diverse as tanning leather and producing toxoids for use in vaccines. The inactivation of viruses with formaldehydes, however, is said to be by reaction of the fixative with nucleic acid compound (i.e. RNA in the tobacco mosaic virus) rather than the protein contained in the virus particles.[7] More specifically, the reaction is said to be one of denaturation followed by reaction with freed nucleotide amino groups, especially in the case of adenine and cystosine residues with the reaction most likely occurring with the nitrogen in position 6.[5,6] The denaturation of amino acids or RNA by aldehydes appears to be partial or complete according to the extent of involvement of hydrogen binding sites. It is reasonable to believe that such denaturation by aldehydes is cumulative during long term or repeated exposures. As a result, the possible role of aldehydes in lung damage by cigarette smoking is presently under consideration.

Although our experiments identified carbon monoxide and aldehydes as the poisonous gases from wood, kerosene, cotton and furniture smokes, there is no doubt that other noxious gases such as sulfur or nitrogen oxides, hydrochlorides, hydrocyanic acids, chlorine, fluorine, lead oxides, ammonia and possibly some other gases can damage respiratory tissues when produced in significant concentrations from combustion of other materials.

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