8 Carbon Monoxide Poisoning and Its Management in the United States

Neil B. Hampson

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8.1 SIGNIFICANCE OF CARBON MONOXIDE POISONING IN THE UNITED STATES

Carbon monoxide (CO) poisoning is a significant health problem in the United States, killing an estimated 3700 people annually.¹ It is the single most common cause of poisoning death in the country. During the decade of the 1980s, there were approximately 1100 deaths each year due to accidental CO exposure and an additional 2600 suicidal deaths.¹ The death rate from unintentional CO poisoning declined in the 1980s, but this was offset by an equal rise in the suicidal death rate.

Nonfatal CO poisoning is even more common. For many years, it has been widely quoted that CO intoxication causes approximately 10,000 affected individuals to seek medical attention or miss at least 1 day of normal activity annually. However, this estimate is decades old, first published the medical literature in the early 1970s,² and derived by the U.S. Public Health Service from limited data in the late 1960s.³ A recent study of hospitals in the Pacific Northwest found an emergency department visit rate for CO poisoning of 18.1/100,000 population per year in a three state region and estimated over 40,000 visits for CO poisoning annually in the entire country.⁴

The actual number of nonfatal cases in the United States annually is likely to be significantly larger for several reasons. Even this higher estimate of disease incidence includes only emergency department visits for recognized CO poisoning. The signs and symptoms of CO poisoning are nonspecific⁵ and underdiagnosis of CO poisoning is well described.^{6,7} Additionally, not all patients are treated in emergency departments. Those treated in medical offices or clinics would not be represented. Finally, patients may attribute the nonspecific symptoms of CO poisoning (e.g., headache, nausea) to alternative causes such as viral illness, staying home from work or school but not seeking medical evaluation.

8.2 TEMPORAL AND GEOGRAPHIC EPIDEMIOLOGY OF CARBON MONOXIDE POISONING

CO poisoning has a seasonal distribution, being much more common in winter than in summer. 1.8-17 In the case of accidental exposures, this is often attributed to the fact that home furnace accidents are more common during the indoor heating season. Severe winter storms can play a role, as well, resulting in CO poisoning when individuals without power use alternative energy sources for heating and/or cooking or when they are trapped in motor vehicles by snow. 18-22 Suicidal CO deaths have been reported to be more common in the United States during the months of March through May. A recent report found an increased number of patients treated in Seattle for intentional CO poisoning in the months of March, April, and October, correlating with the amount of rain on the days prior to a suicide attempt. 23

CO poisoning also has a geographic distribution in the United States, again related at least in part to climate and home heating. The cold and high-altitude states have the highest accidental death rate due to CO poisoning, while warmer states have the lowest rates. The CO-related death rate in Alaska (2.72/100,000) is approximately 50-fold that reported in Hawaii (0.05/100,000). California has the second lowest accidental death rate (0.25/100,000), felt to be due to stringent automobile emission standards, in addition to the relatively warm climate. Few data have been published on the geographic distribution of nonfatal CO poisoning. One survey of U.S. hyperbaric oxygen (HBO) treatment facilities found that the largest number of treatments for CO poisoning was performed in Minnesota during the year studied. 16

8.3 DEMOGRAPHICS OF INDIVIDUALS WITH CARBON MONOXIDE POISONING

Although individuals of all ages are victims of CO poisoning, it is most common among those of middle age. U.S. Consumer Product Safety Commission (CPSC) data indicate that accidental non-fire-, non-automobile-related CO poisonings are most common in the 25 to 44 year age group. Is In a survey of U.S. hyperbaric facilities, the age group most commonly referred for treatment of severe CO poisoning was also 25 to 44 years. Case series of consecutive patients reported from individual treatment facilities confirm this, with average patient ages of 34 years in Illinois, 4 34 years in North Carolina, 5 35 years in Utah, 4 and 34 years in Washington

State.²⁵ The source of CO may have a relationship to victim age. Patients treated at a hyperbaric facility in Seattle for CO poisoning due to indoor burning of charcoal briquets were significantly younger than those who sustained poisoning during recreational boating activities (mean ages 28 vs. 37 years, respectively; p < 0.02).^{10,13}

As middle-aged individuals are most frequently poisoned, it is not surprising that they account for the greatest number of deaths from CO poisoning. 1,9,12,15 However, the age-specific death rate is highest among the elderly, suggesting an increased susceptibility to the toxic effects of CO. A Centers for Disease Control study of U.S. deaths due to accidental CO poisoning in the 1980s found the highest death rate to be among those over 75 years of age. The death rate was reported to be significantly higher among persons 65 years of age and older in studies of unintentional CO poisoning in both Colorado and Michigan. 9,11

CO poisoning has a preponderance for the male sex. Males accounted for 56% of unintentional nonfatal CO poisonings in Colorado from 1986 to 1991. Among various series of patients treated for accidental and/or intentional CO intoxication at U.S. hyperbaric facilities, males have been reported to account for 63 to 73% of patients. Among Tolorado Tolorado

Mortality has been more common among males than females in all series reported. In a national study of deaths from accidental CO poisoning of all causes in the 1980s, the death rate was almost three times greater for males than for females (0.78 vs. 0.26/100,000). Of accidental U.S. non-fire-, non-automobile-related CO deaths from 1990 to 1994, 70% were male. Similar findings have been described in a variety of studies on individual states. Males have accounted for 72% of unintentional CO deaths in California, 181% in Michigan, 74% in New Mexico, 12 and 72% in Colorado. In the latter study, the death rate was 2.6 times greater for males than for females (1.3 vs. 0.5/100,000).

In addition to age and sex, race is also a risk factor for CO poisoning. Members of minority races are more likely both to be treated for and to die from unintentional CO intoxication. While a number of reports provide data on the racial and/or ethnic composition of the population studied, only a few compare this with the general population to allow calculation of poisoning rates or an estimation of risk. A study of all patients accidentally poisoned with CO and treated with HBO in Washington State from 1987 to 1997 found that the relative risk of accidental poisoning compared with whites was 3.96 for Hispanics and 2.91 for blacks.²⁷ In New Mexico, the annual death rate from unintentional CO poisoning per 100,000 persons has been reported as 2.41 for blacks, 0.83 for Native Americans, 0.47 for Hispanics, and 0.46 for whites.¹² A national study of accidental CO deaths found race-specific death rates to be more than 20% higher for blacks than for whites.¹

Possible explanations for higher death rates among minorities could include excess exposure to CO within these groups, enhanced susceptibility to CO intoxication, and/or poorer access to medical care. The relative contributions of these factors is unknown. Limited data exist to support the concept of excess exposure to

CO in certain racial or ethnic groups. For example, in a study of CO poisoning due to the indoor burning of charcoal briquets, it was found that the incidence among Asians and Hispanic whites was far in excess of their representation among the general population. Common reasons for indoor charcoal use were found to be cooking and home heating, apparently a continuation of ethnic customs following immigration to the United States. Enhanced susceptibility to the effects of CO poisoning among specific racial groups has never been reported and, therefore, seems unlikely to be the explanation for higher death rates. It is also difficult to implicate lack of access to health care as the reason when individual minority groups receive a disproportionate number of HBO treatments for poisoning. 27

8.4 SOURCES OF CARBON MONOXIDE INVOLVED IN POISONINGS

CO poisoning has been reported to occur from exposure to virtually every form of combustion that exists in society. While specific sources of CO have been reported to predominate at certain times of the year and/or among select subpopulations (e.g., specific racial groups), some studies do provide information on CO sources for large unselected populations of poisoned individuals. Among 807 nonfatal unintentional poisonings in Colorado, the CO source was a furnace in almost half the cases (Table 8.1).11 The list of sources of CO was much more heterogeneous in a series of 631 accidental, severe CO poisonings referred for HBO treatment (Table 8.1).25 Results from a national survey of U.S. hyperbaric treatment centers, obtaining information from 51% of facilities, found that an indoor gas appliance was the most common source of CO (33%) among patients treated from 1994 to 1995. Among 295 CO-poisoned patients with CO poisoning treated at a Utah hyperbaric facility, the most common sources of CO were internal combustion engines (50%) and furnaces (37%).14 The U.S. CPSC tracks hospital emergency department visits for non-fire-, non-automobile-related cases of CO poisoning resulting from consumer products. 15 From their sample of 1110 cases reported from 1992 to 1996, consumer products implicated in CO poisonings included heating systems (71%), gas ranges/ovens (6%), grills (4%), portable generators and pumps (4%), fuel-powered tools (3%), gas water heaters (2%), and gas clothes dryers (1%). Differences in CO sources among the various series are likely related to differences in the population studied.

The most common source of CO resulting in unintentional CO-related death in the United States is motor vehicle exhaust, comprising 57% of such fatalities in a national study. The majority are associated with stationary automobiles. CPSC show that the consumer products most likely to be sources of CO for non-fire-, non-automobile-related deaths are heating systems (73%), charcoal grills (10%), gas water heaters (5%), camp stoves/lanterns (5%), and gas ranges/ovens (5%). The control of the consumer of the consumer of the control of the consumer of the consumer of the control of the consumer of the con

Series of cases from individual states show some variability in these figures, again likely related to geographic and population influences. Among 444 accidental CO deaths in California, common sources of CO were heating and cooking appliances (40%), motor vehicles (31%), and charcoal grills (13%).¹⁷ In a series of 74 unintentional CO deaths from New Mexico, the most frequent sources of CO were home heating equipment (50%) and motor vehicles (46%).¹² Finally, the most

42 (4.7%)

201 (31.9%)

Sources of Carbon Monoxide in Cases of Unintentional Poisoning			
Ref. 11 $(n = 807)$	Ref. 25 $(n = 631)$		
345 (42.7%)	65 (10.3%)		
178 (22.1%)	108 (17.1%)		
53 (6.6%)	77 (12.2%)		
Not reported	79 (12.5%)		
Not reported	59 (9.4%)		
73 (9.0%)	Not reported		
63 (7.8%)	Not reported		
	Ref. 11 (n = 807) 345 (42.7%) 178 (22.1%) 53 (6.6%) Not reported Not reported 73 (9.0%)		

TABLE 8.1
Sources of Carbon Monoxide in Cases of Unintentional Poisoning

common sources of CO in 174 unintentional CO fatalities in Colorado were fire (36%), motor vehicles (34%), and furnaces (10%).¹¹

Not reported

95 (11.8%)

8.5 CIRCUMSTANCES OF CARBON MONOXIDE POISONING

Boat

Other/unknown

CO poisoning may be accidental or intentional. Among series of patients referred to HBO facilities for treatment of severe poisoning, the proportion of accidental cases has been 76% in North Carolina,²⁵ 82% in Utah,¹⁴ and 72% in Washington State.²⁵ Among fatalities from CO poisoning, only 31% are unintentional.¹ Males account for 71 to 76% of suicidal CO deaths and motor vehicles are the source of CO in 97% of cases.^{1,25}

Simultaneous exposure of multiple individuals is relatively common in incidents of CO poisoning. Among cases of CO poisoning due to indoor burning of charcoal briquets treated at a Seattle HBO facility, 69% of incidents involved poisoning sufficiently severe to require HBO treatment of more than one individual.¹⁰ This likely relates to the indoor use of charcoal in a family setting for heating or cooking. In a series of accidental California CO fatalities, most vehicular exposures resulted in a single death (94%), while multiple deaths were more common when heating and cooking appliances were the source of CO (33%).¹⁷ Among cases of CO poisoning collected by the CPSC (non-fire, non-automobile consumer product related), 22% of fatal incidents involved more than one death and 45% of nonfatal incidents resulted in more than one person poisoned.¹⁵

The location of the individual at the time of poisoning is obviously related to the source of CO. In the case of nonvehicular poisonings, the majority occur in a residential setting, in either a home or garage. 12,15,17,28 Other relatively common locations include campers, tents, boats. 13,15 In one series of 80 accidental poisonings occurring in residential settings, 39 (49%) occurred while the individual was asleep. 28 This has obvious implications for prevention with CO detectors/alarms.

Prior consumption of ethanol is common among those with CO poisoning, presumably affecting judgment, altering consciousness, and predisposing to exposure

which would not otherwise have occurred. Alcohol has been reported to be involved in 31 to 42% of unintentional CO deaths in series of poisoning from all causes. ^{12,17,28} The rate of alcohol use appears to vary depending upon the source of CO. ^{17,28} In one study, alcohol was detected in 80% of adult fire victims, all of whom had elevated carboxyhemoglobin (COHb) levels. ²⁹ Blood alcohol levels were higher among victims discovered in bed as compared with those found near an exit. Among deaths from unintentional CO poisoning from motor vehicle exhaust, 47 to 68% have been reported to have blood alcohol concentrations of at least 0.10 g/dl. ^{9,12,30} In a series of 59 patients dying from accidental CO poisoning from charcoal briquets, 37% involved alcohol consumption. ³¹ In a report of 16 deaths from CO poisoning due to faulty home heating systems, only 5 individuals (31%) had a positive blood or liver ethanol test, and none was over 0.10 g/dL. ³²

While alcohol undoubtedly increases risk for CO exposure, there are limited data that suggest it might actually be protective in CO poisoning. One clinical study of fatal CO poisonings noted that blood COHb levels were higher among those who also had the highest blood alcohol levels, raising the possibility that longer exposure to CO is tolerated before death in individuals with higher blood ethanol levels.³³ One laboratory study demonstrated that pretreatment of rats with ethanol increased both tolerance and survival at various levels of CO exposure.³⁴

8.6 MANAGEMENT OF CARBON MONOXIDE POISONING IN THE UNITED STATES

As mentioned previously, the death rate from accidental CO poisoning has been declining in the United States. Proposed explanations for this observation have included (1) disease prevention, related to factors such as automobile emission control regulations, (2) more stringent occupational exposure standards, and (3) public education. Additional possibilities include improvement in diagnosis and medical management of the disease. While underdiagnosis of CO poisoning has been described, it is possible that physician recognition of CO poisoning is improving. Approximately 400 articles on CO poisoning were published in the Englishlanguage medical literature from 1985 to 1998. It is not unreasonable to expect that this degree of availability of information about the disease would enhance knowledge and awareness among medical practitioners.

Another possible explanation for the declining death rate from accidental CO poisoning is improved treatment of the disease. All agree that appropriate management of acute CO poisoning includes removal of the individual from the source of exposure and administration of supplemental oxygen to enhance clearance of CO from the body. Some degree of disagreement exists in the U.S. medical community as to the exact roles for normobaric vs. HBO therapy in management of the CO-poisoned patient.^{36,37} However, this is an area that is the subject of intense research, the majority of which supports HBO treatment in at least some subgroups of CO-poisoned patients.

As of January 1999, six prospective clinical trials have been reported comparing normobaric oxygen and HBO in the treatment of patients with acute CO poisoning. Three of these have been published in peer-reviewed form^{38–40} and three only in

abstract form to date. 41-43 Among the five trials in which the treatment groups have been unblinded, three showed statistically superior clinical outcomes among patients treated with HBO, 39,40,43 while two showed equivalent outcomes with normobaric oxygen and HBO. 38,42

Two of the trials have been performed in the United States. In one, which is still ongoing, 41 patients with acute CO poisoning are being randomized to three HBO treatments in 24 h (50 min oxygen at 3.0 atmospheres absolute, or atm abs, followed by 60 min oxygen at 2.0 atm abs) or three sham HBO treatments with 100% oxygen breathing of the same duration at 1.0 atm abs. Interim analysis of results after enrollment of 49 patients demonstrated a 32% (8/25) rate of neurologic sequelae in one treatment arm and 17% (4/24) in the other. As the sequelae rates were not statistically different (p = 0.0538), the study was not unblinded and continues to accrue patients.

The second U.S. trial randomized 60 patients with mild CO poisoning, excluding those with history of unconsciousness or cardiac compromise, to treatment with HBO vs. normobaric oxygen at 1.0 atm abs until asymptomatic. ⁴⁰ The HBO protocol utilized included 30 min of oxygen breathing at 2.8 atm abs, followed by 90 min at 2.0 atm abs. Patients were followed with serial neuropsychological testing in an attempt to detect development of delayed neurologic sequelae (DNS). DNS developed in 7 of 30 patients (23%) treated with normobaric oxygen and in no patients following treatment with HBO (p < 0.05). Among those developing DNS, impairment persisted for an average of 6 weeks and often interfered with activities of daily life.

Data collected by the Maryland Institute of Emergency Medical Services System demonstrate that the number of HBO treatments performed annually in the United States for CO intoxication has increased steadily over the past two decades.⁴⁴ In a 1992 study of North American HBO facilities, it was found that 51 multiplace and 90 monoplace facilities in the United States utilized their hyperbaric chamber for treatment of acute CO poisoning.⁴⁵ In that year, multiplace hyperbaric chambers were used to treat 1117 CO-intoxicated patients, with individual facilities treating 0 to 161 patients (mean 22 patients per facility). Monoplace chambers treated a total of 1240 patients in 1992, with individual facilities treating 0 to 112 patients (mean 14 patients per facility). Combining data from monoplace and multiplace facilities found that 2357 total patients were treated at 141 centers, averaging 17 patients per facility. In the case of multiplace facilities, this represented a 34% increase in patients treated annually as compared with figures from 2 years earlier.⁴⁶

With regard to patient selection for treatment with HBO, recommendations are provided by the Hyperbaric Oxygen Therapy Committee of the Undersea and Hyperbaric Medical Society.⁴⁷ In their 1999 report, the committee recommends that CO-intoxicated patients with transient or prolonged unconsciousness, neurological signs, cardiovascular dysfunction, or severe acidosis be referred for HBO therapy irrespective of their COHb levels. The same report notes that the role of neuropsychological testing in patient selection for HBO is unclear. Finally, the committee suggests that treatments be performed at a pressure of 2.4 to 3.0 atm abs. In patients with persistent neurologic dysfunction after the initial treatment, retreatment may be performed once or twice daily until there is no further improvement in cognitive functioning, to a maximum of five HBO treatments.

TABLE 8.2
Proportion of North American Hyperbaric Unit Medical Directors Utilizing Various Criteria to Determine Need for HBO Therapy in Patients with CO Poisoning Presenting Less Than 3 h after Exposure⁴⁵

Criteria	Percent		
Patient arrives at emergency department unconscious with COHb = 9.5%	98		
Initially unconscious upon CO exposure, arriving at the emergency department awake and asymptomatic, with normal neurologic examination and COHb = 9.5%	77		
History of CO exposure, no loss of consciousness, COHb = 9.5% and			
No signs or symptoms	7		
Headache and dizziness only	48		
Electrocardiogram suggesting acute myocardial ischemia	91		
Focal neurologic abnormality on physical examination	94		
Abnormal psychometric testing	91		
No loss of consciousness, presenting with headache, nausea, COHb = 40%, and normal neurological examination, electrocardiogram, and neuropsychiatric testing	92		

While these recommendations may appear clear, there exists tremendous variation in clinical HBO practice in North America with regard both to patient selection criteria and treatment protocols utilized for this disease. When each area is examined in detail, it is seen that consensus exists in only selected aspects of the management of the patient with CO poisoning.

Medical directors of hyperbaric chamber facilities in the United States and Canada were recently surveyed to determine patient selection criteria utilized for application of HBO in CO poisoning. Approaches were found to be most similar when dealing with the more severely poisoned patient (Table 8.2). A significant majority of medical directors administer HBO to CO-poisoned patients with coma, focal neurologic deficits, ischemic changes on electrocardiogram, abnormal neuro-psychiatric testing, or transient loss of consciousness, despite a relatively low COHb level. The approach to patients with milder degrees of poisoning is less clear. Only a minority of hyperbaric facility medical directors surveyed in that study would utilize HBO for the patient with a slightly elevated COHb level and either no symptoms or headache and dizziness only (Table 8.2).

The appropriate role of the COHb level in determining which cases of CO poisoning warrant HBO treatment remains undefined. Nearly all North American medical directors (92%) would administer HBO to a poisoned patient with COHb of 40%, headache, and nausea (Table 8.2). Only two thirds, however, identify COHb level as an independent criterion for the HBO treatment of an asymptomatic patient.⁴⁵ Therefore, while the majority use HBO to treat the patient with a specified minimum COHb level irrespective of clinical signs, some also require symptomatic

manifestation of the poisoning before recommending HBO treatment. Based upon the results of that study, it would appear that manifestations of headache or nausea are considered sufficient symptoms by these physicians to administer HBO therapy.

When the COHb level is applied as an independent indication for HBO therapy, the range of COHb values utilized is quite wide. ⁴⁵ A COHb level of 25% is identified most often, but this value is used by only half of those applying COHb level as a sole criterion for HBO treatment. This variability may result from the fact that it is not possible to draw firm conclusions from the published clinical literature with regard to the role of COHb in determining need for HBO therapy.

The importance placed upon the delay from CO exposure to medical evaluation as a factor in determining appropriateness for HBO treatment is also quite variable among hyperbaric facility medical directors.⁴⁵ Previous studies have demonstrated that effectiveness of HBO therapy decreases with the duration of delay to treatment.^{48,49} Delayed treatment is associated with an increased incidence of residual neurologic deficits after treatment, as well as increased mortality. Neither these nor other studies, however, have precisely defined time limits beyond which HBO therapy for CO poisoning will be ineffective and should therefore be withheld. This lack of information is apparent from the time limits utilized by HBO physicians. One half of North American medical directors use a time limit to deny HBO treatment to a patient with only transient loss of consciousness.⁴⁵ When time limits are applied in such instances, intervals ranging from 6 to 48 h are most commonly used, but delays of 1 to 2 months are allowed by some physicians. In the CO-poisoned patient presenting with focal neurologic findings, time limits are applied by only one quarter of directors to determine eligibility for HBO treatment.

Related to the issue of temporal delay is the patient presenting with delayed development of neurologic or neuropsychiatric sequelae after CO poisoning. A majority of medical directors in the United States and Canada utilize HBO to treat such patients.⁴⁵ Published data regarding the efficacy of such treatment are contradictory.^{50–52}

Interestingly, management of acute CO poisoning in pregnancy remains a topic of controversy in North America. Only 74% of American and Canadian hyperbaric facilities have treated or would treat pregnant patients with CO intoxication. One quarter do not use HBO for pregnant CO-poisoned patients despite a lack of data demonstrating increased risk from such treatment and recommendations from authors in both the United States and Europe that such patients be treated. 53,54

As noted above, there is greatest agreement among hyperbaric physicians regarding use of HBO for the most severely poisoned patients. This seems to be reflected in hyperbaric medicine practice. A recent study found that 6.9% of those evaluated for CO poisoning in emergency departments in Washington, Idaho, and Alaska were referred for HBO therapy.⁴ The report also estimated the number of emergency department visits nationally for CO poisoning (42,890). Using a 1992 figure for the number of CO-poisoned individuals treated with HBO in the United States (2355), a nationwide hyperbaric treatment rate of 5.7% was calculated. Thus, while HBO is recommended for treatment of CO poisoning, it is generally reserved for a select population of patients.

Once the decision has been made to utilize HBO for CO poisoning, physician opinions regarding hyperbaric treatment protocols are quite varied. In 1990, 1023

cases of acute CO poisoning were treated in 42 multiplace hyperbaric chamber facilities in North America, with 38 U.S. facilities treating 832 patients and four Canadian facilities treating 191 patients. A total of 18 different hyperbaric protocols were used at those facilities for primary treatment of acute CO poisoning. These include 3 protocols with a maximum pressure of 3.0 atm abs, 13 protocols with a maximum pressure of 2.8 atm abs, and 2 protocols with a maximum pressure of 2.4 to 2.5 atm abs. The oxygen dose delivered by these protocols (calculated by multiplying the minutes of 100% oxygen breathing by atm abs pressure) differs by a factor of over threefold. In the year studied, 28% of patients were treated at facilities utilizing 3.0 atm abs, 55% at facilities utilizing 2.8 atm abs, and 17% at facilities utilizing 2.4 to 2.5 atm abs. While this might suggest that a consensus exists for treatment at 2.8 atm abs, it should be recognized that the slight majority of patients treated at that pressure were divided among 13 protocols.

The most frequently identified multiplace treatment protocol in North America utilizes a maximum pressure of 3.0 atm abs and is commonly known as the "U.S. Air Force" protocol, developed and applied by the U.S. Air Force for treatment of CO poisoning. So While this protocol was identified as the primary treatment protocol by more multiplace facilities than any other, it is utilized by only 33% of facilities. Furthermore, just 15% of patients treated in multiplace hyperbaric chambers in North America in 1990 were managed by this protocol, attesting to the lack of consensus in this area.

There are no published prospective studies comparing outcome of patients with acute CO poisoning treated with different HBO protocols. While the relative benefit of different protocols has not been directly compared, side effects of some treatment protocols have been evaluated. A large study reviewed 300 patients treated at each of three hyperbaric pressures to define the incidence of central nervous system (CNS) oxygen toxicity associated with treatment at various partial pressures of oxygen.²⁵ It found that CNS toxicity, as manifest by grand mal seizure activity, was significantly more common among patients treated at 2.80 or 3.00 atm abs, as compared with 2.5 atm abs.

8.7 CONCLUSIONS

CO intoxication is a common health problem in the United States. The mortality rate from accidental CO poisoning appears to be decreasing, probably due to improvements in both disease prevention and treatment. While a consensus does exist among North American HBO medical directors with regard to many issues in CO poisoning, discrepancy still persists about several aspects of patient selection and HBO treatment protocols. Despite these discrepancies, there are suggestions that the benefits seen with HBO therapy in clinical trials are indeed impacting the outcome of the disease nationally. When one compares the increasing number of HBO treatments performed in the United States for CO poisoning with the declining number of accidental CO-related deaths, a statistically significant correlation is seen. It is hoped that future refinements in patient selection criteria and hyperbaric treatment protocols for CO intoxication will result in improved management of this common form of poisoning.

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