Symptoms of carbon monoxide poisoning do not correlate with the initial carboxyhemoglobin level

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ABSTRACT

Background – Symptoms in carbon monoxide (CO) poisoned patients have traditionally been described as being related to corresponding carboxyhemoglobin (COHb) levels without substantive support for the relationship. This study sought to determine whether prospectively collected symptoms correlate with specific COHB level ranges in a large population of CO-poisoned patients.

Methods – Data from patients reported in the initial two years of operation of the joint UHMS/CDC CO Poisoning Surveillance System were used to compare presenting COHb levels with symptoms collected with a standardized questionnaire.

Results – Data from 1,323 CO-poisoned patients referred for hyperbaric oxygen therapy from August 2008 to July 2010 were analyzed with regard to initial COHb level and symptoms. Of approximately 50 categories of symptoms reported, none was associated with a specific range of COHb levels.

Conclusions – While symptoms are common in acute CO poisoning, none can be directly correlated to COHb levels, even in a population of more than 1,000 patients. The concept of a table relating specific symptoms to specific COHb levels is invalid. One such table that has often been published comes from a 1923 U.S. government publication and appears to be based at least in part upon the symptoms experienced by three men in a total of 10 low-level laboratory CO exposures.

BACKGROUND

Carbon monoxide (CO) poisoning is common in the United States, accounting for an estimated 50,000 emergency department visits for diagnosed cases annually [1]. It is believed that many more cases go undiagnosed, either because they are unsuspected or attributed to other etiologies. Symptoms are not uncommon in CO poisoning, but instead the typical symptoms of the condition – headache, nausea, vomiting, dizziness – are so non-specific that it is difficult to use them to differentiate CO poisoning from other common conditions such as viral illness or food poisoning [2].

When clinical suspicion for CO poisoning does exist, an elevated blood carboxyhemoglobin (COHb) measurement is used to confirm a clinical diagnosis of exposure to carbon monoxide. An elevated COHb level (greater than 2% for non-smokers and greater than 10% for smokers) [3] strongly suggests exposure to exogenous CO and supportsa clinical diagnosis of CO poisoning in the appropriate setting.

Most clinicians actively involved in the field of CO poisoning feel that the degree of elevation of the COHb level does not correlate well with the patient's clinical presentation and do not use it to direct management, only to support diagnosis. We demonstrated previously in a retrospective review of 1,407 patients referred for hyperbaric oxygen treatment of CO poisoning that while average COHb levels had statistically significant differences between cohorts for some clinical variables, the absolute differences were so small that they were clinically unhelpful [4]. In their respective reviews of clinical CO poisoning, Piantadosi wrote, "…the correlation between clinical deficits and measured COHb level is quite weak" [5], while Weaver stated, "The level does not correlate with the presence or absence of initial symptoms" [2].

For decades, however, papers and book chapters on CO poisoning containing tables or charts relating the degree of COHb elevation to specific symptoms or signs have been published and taught in medical schools. The current *Merck Manual Professional Edition* contains such an example *(Table 1, Page 640)*, accompanied by the statement, "Symptoms tend to correlate well with the patient's peak blood carboxyhemoglobin levels" [6]. Interestingly, the tables in the literature relating COHb levels to specific symptoms have common characteristics.

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TABLE 1. ***Example of published table correlating specific symptoms with specific COHb levels***

COHb LEVELS	**Corresponding clinical manifestations** ***(according to Reference 6)***
10-20%	Headache and nausea
> 20%	Vague dizziness, generalized weakness, difficulty concentrating, impaired judgment
> 30%	Dyspnea during exertion, chest pain *(in patients with coronary artery disease)*, confusion
HIGHER LEVELS	**Syncope, seizures, obtundation**
> 60%	Hypotension, coma, respiratory failure, death

First, most tables are remarkably similar, if not identical, in content, suggesting a common source. Second, a reference for the data is often not given. When a citation is provided, it typically directs the reader to a paper containing the same table, this time without a reference.

The predictability of symptoms and signs with specific COHb measurements is clearly a matter of dispute. The present study was conducted to:

1. compare initial COHb levels with symptoms collected in a prospective fashion from a large population of CO-poisoned patients, attempting to resolve this issue; and
2. seek out the origin of the table that is commonly published and evaluate the validity of the data supporting it.

METHODS

Since August 2008, the Undersea and Hyperbaric Medical Society (UHMS) has maintained an online system for surveillance of cases of carbon monoxide poisoning treated with hyperbaric oxygen. When a patient is treated in a facility enrolled in this voluntary program, facility staff log on to a secure website and enter non-identifiable demographic and epidemiological data about the exposure. The system is funded by and operated in conjunction with the Centers for Disease Control and Prevention, in an effort to expand surveillance of CO poisoning and also to test the hypothesis that teaming with a medical specialty society in this fashion is an effective and efficient way to perform disease surveillance. Complete details of development and operation of the program are available in another publication [7].

Among the 40 data fields collected for each patient are "Initial Carboxyhemoglobin Level," to be completed if the test was performed and the result is available, and "Symptoms." The Symptoms screen lists 10 radio-button options and instructs users to mark as many as apply. These include chest pain, confusion, dizziness, fatigue, headache, loss of consciousness (LOC), memory complaints, nausea / vomiting, shortness of breath, and "other," with a box provided for freeform entry of the latter. Facilities are also asked to estimate the time from removal from the CO exposure to obtaining the sample for COHb measurement, whether supplemental normobaric oxygen was administered prior to hyperbaric treatment, and the route by which it was administered.

Hard copies of the survey are available to participating facilities for use as a worksheet prior to online entry of the surveillance data. Respondents are encouraged to collect the survey information during the two to three hours that the patient is in the hyperbaric chamber rather than from retrospective chart review, both for reporter convenience, as well as optimizing completeness of the data gathered. With the patient available on-site, answers to questions can be obtained that might not otherwise be recorded in the medical record (*e.g.*, educational level).

In the first two years since the system went live in August 2008, information on 1,358 CO-poisoned patients treated with hyperbaric oxygen were prospectively reported from 58 hyperbaric facilities in 39 states. No carboxyhemoglobin level was entered for 35 patients, and they were excluded from analysis. Data from the remaining 1,323 with regard to initial carboxyhemoglobin levels and symptoms form the basis of this report.

RESULTS

The 1,323 patients were 59% male, average age 39 ± 17 years, 61% non-Hispanic White and 84% primary language English *(Table 2, facing page)*. The most common CO sources were furnaces 23%, motor vehicles 21% and generators 16%. COHb averaged 23.3 ± 10.6% (mean ± SD; range 1.0-77.0%), measured 1.8 ± 2.2 hours (range 0-60 hours) following CO exposure. A total of 15% were intubated with average COHb 31.6 ± 19.6% (range 1.0-77.0%). Fifteen per cent of the patients showed signs of cardiac injury.

TABLE 2. ***Clinical characteristics of patients included in this analysis***

COHb%	*N*	Male	Age *(years)*	#1 Source	#2 Source	#3 Source	Intubated
0-10.0	130 (9.8%)	61 (47%)	36±22	Furnace 36 (28%)	Motor vehicle 24 (8%)	Generator 14 (11%)	13 (10%)
10.1-20.0	388 (30%)	217 (54%)	36±22	Furnace 91 (23%)	Generator 79 (20%) (	Motor Vehicle 64 16%)	37 (9%)
20.1-30.0	492 (37%)	294 (60%)	41±20	Furnace 128 (26%)	Motor vehicle 96 (20%)	Generator 87 (18%)	55 (11%)
30.1-40.0	226 (17%)	150 (66%)	43±19	Motor vehicle 57 (25%)	Furnace 38 (17%)	Generator 32 (14%)	55 (24%)
40.1-50.0	66 (5%)	45 (68%)	45±20	Motor vehicle 27 (41%)	Fire 11 (17%)	Generator 9 (14%)	29 (43%)
>50.0	10 (1%)	8 (80%)	42±15	Motor vehicle 6 (60%)	Fire 4 (40%)		7 (70%)
All	1,323	775 (59%)	39±17	Furnace 299 (23%)	Motor vehicle 275 (21%) (16%)	Generator 212 (15%)	196 (15%)

Of the total 1,323 patients reviewed, 1,294 (98%) were known to have received supplemental normobaric oxygen prior to hyperbaric treatment. It was delivered by non-rebreather reservoir face mask in 1,092 cases, by endotracheal tube in 146, by simple face mask in 23, by nasal cannulae in 14 and by an unknown route in 19.

An estimated time from extraction from CO exposure to obtaining a sample for COHb measurement was provided in 992 cases (75%) and averaged 2.0 ± 3.4 hours (range 0-60 hours). Of those 992 cases, 867 received normobaric oxygen prior to hyperbaric treatment by non-rebreather reservoir face mask and had a delay to COHb sampling of 2.0 ± 3.1 hours (range 0-45), 91 via endotracheal tube with delay of 1.2 ± 0.8 hours (range 0.25-4.5), 15 via simple face mask with delay of 1.6 ± 1.9 hours and 11 via nasal cannulae with delay of 6.8 ± 16.8 hours (range 0.25-60).

Literally hundreds of symptoms were reported in total, many of which were permutations of other symptoms. When consolidated in Table 3 *(Page 642)*, approximately 50 different symptoms in nine different organ systems were reported by CO-poisoned patients.

Table 4 *(Page 642)* shows the eight most common symptoms, as related to COHb level. At least one of these eight symptoms was present in 1,025 (77%) of the study population. The most common symptoms were headache (54%) and loss of consciousness (49%). When looking at any one of these common symptoms, it is present in a significant proportion of patients within any presenting COHb range. Dizziness, for example, was present in 35% of those with COHb 0.0-10.0% and 30% of those with COHb 40.1-50.0%. None of the common symptoms can be related or isolated to any particular COHb level range.

The number of symptoms per patient is illustrated in Figure 1 *(Page 642)*. When looking at all patients, symptoms / patient remain relatively constant as blood COHb rises, ranging from 2.7 to 3.1 with COHb concentrations up to 50% and then decreasing to 1.9 with COHb ≥ 50.1%. However, if one removes the intubated patients and only looks at symptoms per non-intubated patient, a progressive increase is seen with COHb≥40.1%.

DISCUSSION

This study clearly demonstrates that symptoms are common in CO poisoning. One would expect that each poisoned patient would have at least one sign or symptom, since most definitions of CO poisoning include a history of exposure, consistent signs or symptoms, and an elevated blood COHb level to make a clinical diagnosis. It is interesting to learn that most patients have multiple symptoms when assessed in a prospective manner. Previous studies using retrospective review of emergency department records have likely underestimated the number of symptoms due to the tendency to document only the most critical information in the urgent care setting.

It was not surprising to see the numbers of symptoms per patient actually decline at the highest COHb levels *(Figure 1)*. The rate of endotracheal intubation rose with increasing COHb levels *(Table 2)*, and intubated patients generally describe few or no symptoms. When they were

TABLE 3. ***All symptoms reported by patients with acute CO poisoning, grouped by organ system***

Cardiac	Chest pain, heaviness, fullness, tightness Left arm pain Palpitations
Gastrointestinal	Abdominal pain Diarrhea Fecal incontinence Hematemasis Nausea Vomiting Xerostomia
Neurological	Aphasia Confusion Coordination problems Dysarthria Facial droop Gait disturbance, ataxia, balance problems Headache Hemiparesis "Jerky" movements Loss of consciousness Memory complaints Numbness (focal, diffuse) Pain (numerous sites) Paraparesis Paresthesias Seizure Tremor Twitching
Ophthalmologic	Ocular burning or pain Vision disturbance (blindness, blurring, diplopia, scotomata)
Otologic	Dizziness Hearing loss Tinnitus Vertigo
Psychiatric	Anxiety
Respiratory	Cough Dyspnea
Urologic	Flank pain Urinary incontinence
Miscellaneous	Chilling Diaphoresis Drowsiness Fatigue Fussiness Giddy Hot flashes Irritability Lethargy Lightheadedness Muscle cramps Myalgias Rash

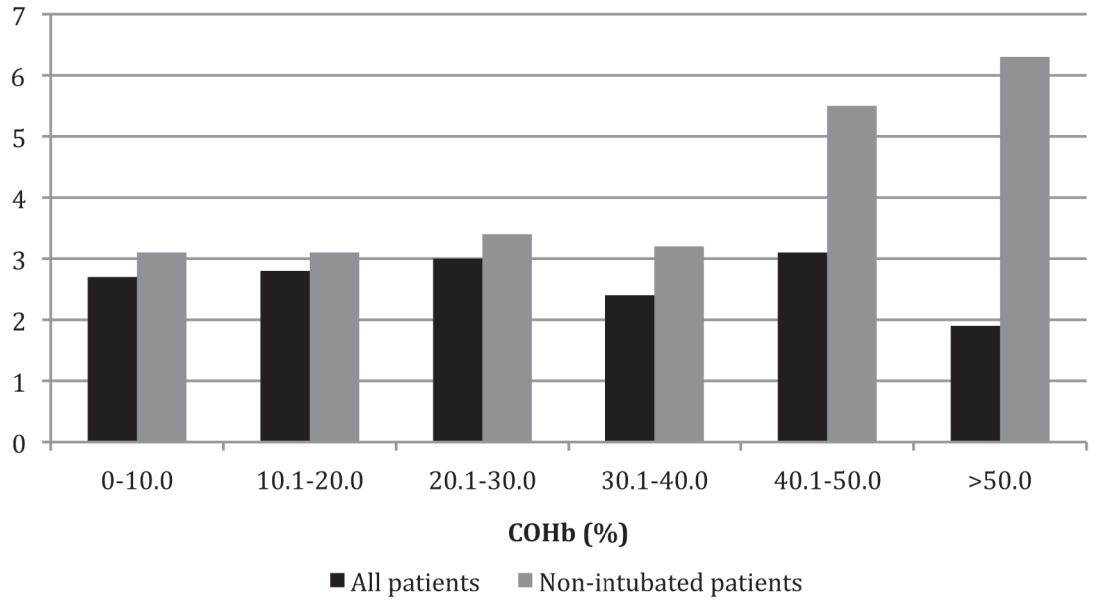
TABLE 4. ***Common symptoms reported by patients with acute CO poisoning and prevalence of symptoms at various COHb levels***

COHb	*N*	Headache	Dizziness	*N*/V	Confusion	Fatigue	Chest pain	SOB	LOC
0.0-10.0%	130	73 (56%)	38 (29%)	52 (40%)	39 (30%)	44 (34%)	12 (9%)	9 (7%)	50 (36%)
10.1-20.0%	399	220 (55%)	163 (41%)	156 (39%)	109 (27%)	100 (25%)	27 (7%)	25 (6%)	181 (45%)
20.1-30.0%	492	304 (62%)	239 (49%)	198 (40%)	139 (28%)	137 (28%)	44 (9%)	45 (9%)	214 (43%)
30.1-40.0%	226	88 (39%)	85 (38%)	66 (29%)	66 (29%)	31 (14%)	22 (10%)	17 (8%)	137 (61%)
40.1-50.0%	66	28 (42%)	19 (29%)	24 (36%)	22 (33%)	14 (21%)	5 (8%)	7 (11%)	60 (79%)
>50.0%	10	1 (10%)	1 (10%)	1 (10%)	2 (20%)	1 (10%)	0 (0%)	0 (0%)	9 (90%)
	1,323	714	545	497	377	327	110	103	651

removed from the analysis, the number of symptoms / patient actually rose with increasing COHb levels. At COHb levels over 40%, non-intubated patients averaged more than five symptoms each. When looking at the total population, the greatest number of symptoms/ patient was seen in the COHb range of 20-30%. It is certainly possible that one could screen groups of CO-exposed patients with a standard symptom list and use the gross number of symptoms to identify those with extreme elevatons. However, since most practitioners in the field of CO poisoning do not feel that the degree of COHb elevation should play a significant role in management, the usefulness of identifying those with greater than 40% or 50% COHb is difficult to know.

However, despite the fact that symptoms were methodically recorded and reported, none of them were

FIGURE 1. ***Number of symptoms reported per patient with acute CO poisoning***



specific for any given range of blood carboxyhemoglobin levels, as is so often suggested in the medical literature. While the incidence of loss of consciousness did rise with increasing COHb levels, it is technically a sign and not a symptom. Even so, it was still present in one-third of patients with the lowest range of COHb levels, likely because it is a common criterion for referral for hyperbaric treatment. While such a high incidence of LOC with low COHb levels may at first seem surprising, it must be remembered that these are the initial COHb measurements performed, and they may be quite delayed from the exposure, confounded by interval normobaric oxygen administration. Indeed, in another study of 972 accidentally poisoned patients referred to one hyperbaric center for treatment, 47% of those with initial COHb levels of 0-9% had experienced LOC [4].

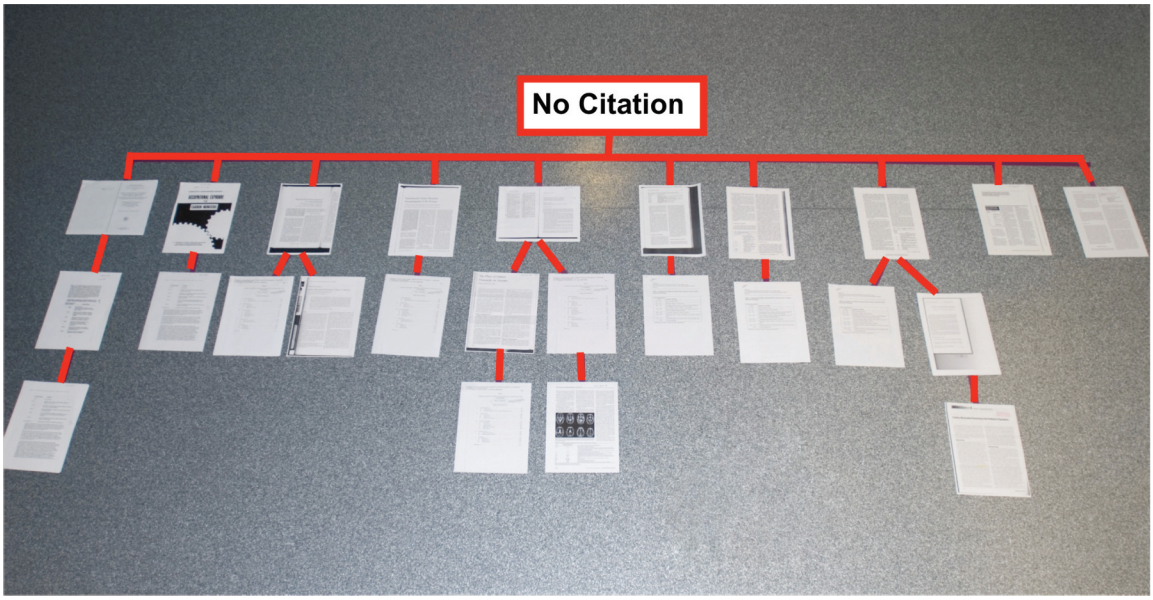
The mean COHb level for the 1,323 patients in this report was 23.3 ± 10.6%, not statistically different from the 25.0 ± 9.6% measured in the 76 hyperbaric-treated patients in Weaver's 2002 randomized trial ($p = 0.195$; two-tailed unpaired T-test) [8]. If the five symptoms reported in that study are compared with the current data, Weaver's patients had a higher incidence of each symptom, but the rank order of symptom frequency was approximately the same. This again supports the premise that particular symptoms are not related to specific COHb levels.

Also interesting is the wide range of symptoms that were reported *(Table 3)*. To our knowledge, this is the first list of this type to be assembled. It should be noted that these are symptoms which CO-poisoned patients described but may not have been causally related to the poisoning itself. However, the findings do underscore the fact that CO intoxication is a systemic poisoning, affecting tissues throughout the body.

Even with these extensive prospective data from more than 1,000 patients, one would have a difficult time constructing a table ascribing symptoms to particular levels of COHb. When it was apparent that the concept of such a table was invalid, we attempted to locate the original source for the widely published table of symptoms *vs.* COHb levels. Our department's collection of several hundred articles on clinical CO poisoning was manually searched. When a paper including a symptom / COHb table was identified, the citation for the table was used in the construction of a reference tree. Figure 2 *(Page 644)* displays 25 papers on CO poisoning containing a table relating CO poisoning symptoms to specific COHb levels. The branching lines show the earlier publication cited in each as the source of the information. In total, they can be traced back to 10 papers, each with a symptom / COHb table, but none with a citation for its source.

However, our literature review included a 2003 letter to the editor of Environmental Health Perspectives written by Donnay [9], critiquing an article the journal

FIGURE 2. ***Reference tree outlining the citations used in publications for a table linking specific symptoms to specific COHb levels.***



had published which included a symptom / COHb table [10]. In his letter, Donnay noted the original source for the table to be a "Report of Investigations" published by the U.S. Bureau of Mines in 1923 [11].

Indeed, the table which has been most commonly reproduced in identical or modified form in the medical literature is found in that government publication *(Figure 3, below)*. As Donnay accurately pointed out in his letter, the table was qualified in the original publication with the phrase "in general" and gave no source for the data included. However, when discussing absorption of CO by the blood, the publication did mention a prior 1922 publication by the same authors entitled, "Physiological Effects of Exposure to Low Concentrations of Carbon Monoxide" [12].

FIGURE 3. ***Table of symptoms present at specific ranges of COHb% from 1923 Bureau of Mines publication [11]***

0 - 10	No symptoms.
0 - 20	Tightness across forehead; possibly slight headache, dilatation of cutaneous blood vessels.
20 - 30	Headache; throbbing in temples.
30 - 40	Severe headache, weakness, dizziness, dimness of vision, nausea and vomiting, collapse.
40 - 50	Same as previous item with more possibility of collapse and syncope, increased respiration and pulse.
50 - 60	Syncope, increased respiration and pulse; coma with intermittent convulsions; Cheyne-Stokes' respiration.
60 - 70	Coma with intermittent convulsions, depressed heart action and respiration, possibly death.
70 - 80	Weak pulse and slowed respiration; respiratory failure and death.

In their 1922 paper, two surgeons and a chemist employed by the U.S. Bureau of Mines built a gas exposure chamber for humans. They then exposed themselves to CO at concentrations of 200-400 ppm for several hours, drawing blood for COHb measurement and recording symptoms. Ten total exposures were made between the three men, achieving peak COHb levels of 16%-28%. Three of the 10 exposures resulted in COHb levels greater than 25%. Symptoms recorded included "tightness across forehead," "slight headache," "dizziness" and "throbbing headache," among others, many of which were carried over to the table in their 1923 publication at respective COHb levels. It is unknown how they obtained clinical information about physiological responses to COHb levels of 30-80% because none of their exposures resulted in blood levels of that magnitude.

The major limitation to our study is lack of knowledge about the population's peak COHb levels. Measurements were obtained after variable lengths of time following removal from the CO exposure and after variable

amounts of oxygen administration. It is possible that symptoms might more accurately correlate with peak rather than presenting COHb levels. However, the practicing physician does not have that information either, so our conclusions are similar to the situation in the real-world clinical environment.

It is impossible for us to confidently back-calculate to estimate peak COHb levels. While participants reported estimated delay to COHb measurement and reported whether normobaric oxygen was administered prior to hyperbaric treatment, they were not specifically asked whether oxygen was administered during the period from extraction from CO exposure to COHb measurement.

If one were to make the unsupported assumptions that normobaric oxygen was continuously delivered starting at the moment of extraction and then only by the one route reported for each patient, one can try to back-calculate to peak COHb levels for a subgroup of the population. Since even the oxygen concentration delivered by a non-rebreather reservoir mask has been demonstrated to be far below 100% [13], only those who were intubated could be assured of having received 100% oxygen. That subgroup had an average initial COHb level of approximately 32% and an average delay to measurement of 1.2 hours. If the 83-minute half-life of COHb breathing 100% normobaric oxygen used by Weaver in his 2002 paper is applied [13], an average peak level of 55% for the group is estimated. The potential for inaccuracy of this type of calculation is apparent.

In summary, this paper has shown that multiple symptoms are common in patients with acute CO poisoning and that none can be directly correlated to COHb levels even in a population of more than 1,000 patients. The concept of a table relating particular symptoms to specific COHb levels is invalid. The commonly published table comes from a 1923 U.S. Bureau of Mines publication and appears to be based at least in part upon the symptoms experienced by three men in a total of 10 low-level CO exposures. ■

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APPENDIX

Hyperbaric facilities enrolled in the joint UHMS/CDC CO poisoning surveillance system and reporting patients during the first two years of system operation

Hyperbaric Facility	State	# Cases Reported
Scottsdale Healthcare	AZ	3
Fresno Community Regional Medical Center	CA	24
HyOx Medical Treatment Center	CA	8
Long Beach Memorial Medical Center	CA	3
UCSD Hyperbaric Medicine Center	CA	13
Poudre Valley Health System	CO	1
Hartford Hospital	CT	8
Norwalk Hospital	CT	21
Florida Hospital	FL	13
University of Miami Hospital Hyperbaric Medicine Dept	FL	1
Iowa Methodist Medical Center	IA	16
The Center for Wound Healing and Hyperbaric Medicine	ID	20
Eastern Idaho Regional Medical Center	ID	3
Advocate Lutheran General Hospital	IL	96
St. Joseph Hospital	IN	22
St. Margaret Mercy Hospital	IN	2
University of Kentucky Hospital	KY	29
West Jefferson Medical Center	LA	10
University of Maryland/Cowley Shock Trauma Center	MD	117
Detroit Medicine Center	MI	26
Spectrum Health	MI	77
Cox Hyperbaric Medicine and Wound Care Center	MO	15
Liberty Hospital	MO	2
Duke University Medical Center	NC	38
Carolinas Medical Center	NC	10
The Nebraska Medical Center	NE	31
Dartmouth Hitchcock Medical Center	NH	1
Wound Healing Center at Concord Hospital	NH	3
Presbyterian Hospital	NM	30
Brookdale University Hospital and Medical Center	NY	3

APPENDIX

Hyperbaric facilities enrolled in the joint UHMS/CDC CO poisoning surveillance system and reporting patients during the first two years of system operation

Hyperbaric Facility	State	# Cases Reported
Long Island Hyperbarics	NY	5
OGH Center for Wound Healing & Hyperbaric Medicine	NY	1
SUNY Upstate Medical University Hospital	NY	9
St Elizabeth Health Center	OH	20
St. Luke's Hyperbaric Medicine	OH	25
The Toledo Hospital/Toledo Children's Hospital	OH	14
Providence Portland Medical Center	OR	29
Altoona Regional Health System	PA	1
Lombard Hyperbaric Oxygenation Medical Center	PA	2
UPMC Presbyterian University Hospital	PA	35
University of Pennsylvania	PA	120
Kent Hospital	RI	22
Richland Memorial Hospital	SC	5
Spartanburg Regional Medical Center	SC	4
Avera McKennan Hyperbaric Medicine	SD	4
Erlanger Hospital	TN	20
Memorial Hermann Center for Hyperbaric Medicine	TX	28
Northwest Wound Care Center & Hyperbaric Oxygen Therapy	TX	1
San Antonio Military Medical Community Hyperbaric Center	TX	1
United States Air Force School of Aerospace Medicine	TX	1
Dixie Regional Medical Center	UT	1
Intermountain Medical Center	UT	102
Utah Valley Wound Care and Hyperbaric Medicine Center	UT	38
Inova Mt Vernon Hospital	VA	5
Deaconess Regional Hyperbaric Center	WA	14
Tri-State Memorial Hospital Wound Care and Hyperbaric Ctr	WA	1
Virginia Mason Medical Center	WA	124
Aurora Saint Luke's Medical Center	WI	48
Presbyterian/St. Lukes Medical Center	WI	31