Occult Carbon Monoxide Poisoning

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A syndrome of headache, fatigue, dizziness, paresthesias, chest pain, palpitations and visual disturbances was associated with chronic occult carbon monoxide exposure in 26 patients in a primary care setting. A causal association was supported by finding a source of carbon monoxide in a patient's home, workplace or vehicle; results of screening tests that ruled out other illnesses; an abnormally high carboxyhemoglobin level in 11 of 14 patients tested, and abatement or resolution of symptoms when the source of carbon monoxide was removed. Exposed household pets provided an important clue to the diagnosis in some cases. Recurrent occult carbon monoxide poisoning may be a frequently overlooked cause of persistent or recurrent headache, fatigue, dizziness, paresthesias, abdominal pain, diarrhea and unusual spells.

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Although acute carbon monoxide (CO) poisoning is a well-described clinical condition, chronic CO poisoning is a seldom-recognized and poorly understood clinical disorder. I report a surprising incidence of 26 cases in two years in a general internal medical practice and present clinical and laboratory observations.

Patients and Methods

Patients included in this report sought care between 1982 and 1984 for fatigue, headache or at least two of the symptoms listed in Table 1. Criteria for their inclusion in the series were having had symptoms for at least one month and improvement in their condition after a source of CO in their environment was removed. Urine and blood specimens from the principally exposed person in each family were used for routine screening tests—complete blood count, erythrocyte sedimentation rate, a battery of chemistry tests and urinalysis. A patient's carboxyhemoglobin level was measured as soon as possible after the suspected CO exposure. Of 32 patients and family members given questionnaires regarding defective equipment, duration of symptoms before diagnosis and recovery time, 25 returned the questionnaires. Also included in the report are four patients with symptoms who had had furnace problems, had heard about the study and volunteered to fill out questionnaires. In these instances, no tests were done and carboxyhemoglobin levels were unknown because the faulty source of CO had already been repaired. Three patients were eliminated from the study because they had symptoms that persisted more than six months after the suspected source of CO had been repaired. The profile of the 26 patients is given in Table 2.

Household pet morbidity and mortality were also surveyed because patients frequently told us of bizarre pet behavior and death (Table 3). Six pets had died and six others had had symptoms conceivably related to CO exposure.

A causal association between CO exposure and clinical illness is suggested by case histories, identifying a CO source, carboxyhemoglobin levels exceeding normal values (less than 3% for nonsmokers) and symptom resolution on repair of the CO source. The exact quantity of CO was measured in a patient's environment in one case, and in most other cases a mechanic or repairer found a cracked heat exchanger, a faulty muffler or a plugged furnace exhaust chimney. The defective equipment was repaired after atmospheric screening devices (for example, Drager tubes) registered unsafe concentrations of environmental CO.

Results

Questionnaire results are shown in Tables 1 through 3. In this series of 26 cases, 15 patients were exposed to 9 defective gas furnaces, 3 to faulty oil furnaces, 7 to malfunctioning automobile exhaust systems and 1 to a defective bus exhaust system. The exhaust system defects were found in mufflers, catalytic converters and a gas manifold (Tables 1 through 3).

Carboxyhemoglobin Levels

Carboxyhemoglobin levels were measured in blood specimens of 14 patients within five hours of suspected CO exposure. The concentrations ranged from 8.8% to 36.8% (mean 15.1%). The mean concentration for patients exposed to an automobile with exhaust problems was 14.0% and that for patients exposed to a faulty furnace, 15.8%. Three patients with chronic symptoms presented in coma; all three had nat-

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ural gas furnaces. (They are included in this report as having long-term exposure because their symptoms were present for several days to weeks before the episodes of unconsciousness began.)

Symptoms

Most patients reported fatigue and headache (Table 1). A majority also reported having trouble thinking, dizziness, a pounding heart and shortness of breath. Nine patients had impaired visual acuity; it was the initial complaint in three patients. Diarrhea and abdominal pain were also frequently described, as were chest pain, paresthesias and "vague spells."

TABLE 1.—*Frequency of Symptoms in Patients Exposed to Carbon Monoxide (26 Patients)*

Symptom	Patients	
	Number	Percent
1. Fatigue	24	92
2. Headaches	22	85
3. Trouble thinking	20	77
4. Dizziness	19	73
5. Nausea	15	65
6. Trouble sleeping	15	65
7. Heart pounding	14	54
8. Shortness of breath	14	54
9. Numbness or tingling	12	46
10. Chest pain	9	35
11. Decreased vision	9	35
12. Diarrhea	9	35
13. Unusual spells	9	35
14. Abdominal pain	7	27

Illustrative Cases

Cases 1 through 6. Patient 1, a 34-year-old mother of three, presented with persistent fatigue, headache, hearing loss, sinus congestion and chest pain. Symptoms had begun the previous winter but had abated slightly during the summer. In October and November her fatigue, headache and lightheadedness precluded all volunteer work, housework and cooking. Family members were initially unaffected.

On physical examination she had mild hypertension (blood pressure 140/100 mm of mercury). Values for urinary excretion of catecholamines were 118 μg per 24 hours (normal 30 to 100) and vanillylmandelic acid, 10.76 mg per 24 hours (normal 1 to 8). Results of a repeat study of a 24-hour urine collection during the hospital stay were normal, as were concentrations of plasma catecholamines. A computed tomographic scan of the abdomen was also normal, and she was discharged with no diagnosis.

Meanwhile her mother, who was staying with the family to help with household chores, began to feel fatigued, hypersomnolent and complained of acroparesthesias. When the gas furnace in the home was checked, a CO leak was detected in

TABLE 2.—*Patient Profile**

Patient	Age, Yr	Sex	Smoker	Symptoms†	Duration of Symptoms, Months	Occupation	Carboxyhemoglobin Level, Percent	Source of CO	Time Until Symptoms Resolved, Weeks
1	34	♀	Yes‡	1-14	3	Homemaker	8.8	Gas furnace	4-6
2	39	♂	No	1-3,6-9,11,12	3	Accountant	15.8	Gas furnace	2-4
3	8	♀	No	1-6,10,14	3	Student	5.0	Gas furnace	2-4
4	8	♀	No	1-4,6,10	3-6	Student	3.5	Gas furnace	2-4
5	5	♂	No	1-6,7,9,12,14	3-6	Student	7.8	Gas furnace	2-4
6	53	♀	No	1-4,6,8-11,13,14	3-6	Homemaker	NC	Gas furnace	4-8
7	57	♂	Yes	1,4-6,8,9,12	6	Stevedore	19.0	Gas furnace	4-6
8	29	♂	No	1-14(coma)	1	Carpenter	25.9	Gas furnace	12
9	28	♀	No	1-8,11-13(coma)	1	Veterinarian's assistant	19.8	Gas furnace	2-8
10	61	♀	No	1-3,6,7	<1	Homemaker	36.8	Gas furnace	12
11	62	♂	No	1,2,4,5,7,8,12	<1	Physician	NC	Gas furnace	1
12	47	♂	No	2,6,8,10	3	Army officer	NC	Gas furnace	1
13	41	♀	No	1-3,8,10	6	Office worker	NC	Gas furnace	2-4
14	30	♂	No	1-7,9	3	Accountant	1.5§	Gas furnace	4
15	33	♀	No	1,2,5-7,9	1	Research technician	NC	Gas furnace	1-2
16	25	♀	No	1-3,5,6,12,13	3	Nurse	1.0§	Oil furnace	2-4
17	61	♀	No	1,2,4,7	6	Homemaker	3.3§	Oil furnace	2
18	34	♀	No	1-11,13,14	3-6	Homemaker	3.0§	Oil furnace	12
19	43	♀	No	1-4,8,12	>6 (4 yrs)	Psychologist	8.8	Auto muffler/hatchback door	>8
20	26	♀	Yes	1-5,8	12	Receptionist	2.8§	Auto exhaust manifold	8
21	36	♀	No	3,4,11	1	Teacher	22.0	Auto exhaust recycler	<1
22	36	♀	No	1-11,13,14	6	Beautician	16.3	Auto catalytic converter	12
23	27	♀	No	1,3-5,7,12-14	6	Attorney	21.9	Auto catalytic converter	2
24	30	♀	No	1-5,8-10,13	1	Medical assistant	11.0	Auto exhaust manifold	4-8
25	27	♂	No	1	3	Medical student	NC	Auto muffler	1
26	42	♀	No	1-4,7-9,11,13	1	School bus driver	NC	Bus tailpipe	4

CO=carbon monoxide, NC=not checked

*Patients 1 to 5 are in the same family, patients 8 and 9 are married and patients 10 and 11 are married.
†Symptoms numbered as in Table 1.
‡5 cigarettes per day.
§Blood sample drawn >24 hours after exposure to CO.

TABLE 3.—*Sick Pets in Households of People Exposed to CO*

1	Dead canary
4	Dead puppies
1	Dead cat
2	Cats behaving strangely ("potty trained until period of exposure")
3	Dogs behaving strangely ("lethargic, would not lie on floor, spacey")
1	Dog with diabetes insipidus (polyuria, polydypsia)

the heat exchanger. Carboxyhemoglobin levels in the patient (who smoked four to five cigarettes a day), her husband (a nonsmoker) and their twin daughters and son were 8.8%, 15.5%, 7.5%, 5.0% and 3.5%, respectively.

Her husband (patient 2), an accountant, noted an inability to commit figures to memory and difficulty with dictation, which he attributed initially to "the stress of my wife's illness." Their 8-year-old twins (patients 3 and 4) had not felt well, and their 5-year-old son (patient 5) had been having school problems, headaches, dizziness and fatigue.

COMMENT. The severity of the illness seemed to correlate with the relative exposure to the contaminated environment. In this family, the mother (patient 1) and youngest child, who spent more time at home, had more severe symptoms than the father and older children.

Cases 8 and 9. A 29-year-old male carpenter and his 28-year-old wife (a veterinarian's assistant) came to an emergency room with headache, fatigue, nausea, weakness, dizziness, shortness of breath, chest pain and diarrhea of a week's duration. Initial evaluation led to a presumptive diagnosis of a viral illness. Two days later they were seen in an outpatient clinic with progressive symptoms and were given the same diagnosis. The next day the symptoms became excruciating (headache, weakness, vomiting and diarrhea). They returned to the emergency room and were again sent home with a diagnosis of "the flu." During this time, three of their one-month-old puppies had died, and an autopsy showed "pulmonary consolidation and emphysema." In retrospect the couple noted that their symptoms decreased when they were away from home.

Two days later, when the couple did not keep an engagement, the host became concerned and found them comatose at home, their last puppy dead, one adult dog unconscious and another dog aimlessly wandering around the house. The fire department was called and found their furnace chimney clogged with soot. The couple was given oxygen and taken to a local emergency room. Carboxyhemoglobin levels determined from blood specimens drawn after an undetermined interval of oxygen administration were 19.8% for patient 9 and 25.9% for patient 8. They were transferred to the Virginia Mason Hospital (Seattle) Hyperbaric Unit for treatment of their acute and chronic CO poisoning. They were lethargic, confused and ataxic and complained of nausea and severe headache on arrival. Symptoms abated with administration of hyperbaric oxygen, but persistent ataxia and headache, plus bilateral subhyaloid hemorrhages in the woman, necessitated a second hyperbaric treatment the following day.

Patient 8's course was complicated by a deep venous thrombosis and pulmonary infarct four days later. He also continued to have minor difficulties with balance, requiring a change in job from high-rise scaffold carpentry. Patient 9 recovered completely after several weeks of mild headaches and visual disturbances.

COMMENT. The health of household pets can provide an important diagnostic clue in an otherwise obscure illness. When several family members are ill simultaneously and household pets are more severely affected, the type and condition of heating appliances should be determined.

Case 23. The patient, a 27-year-old female attorney, saw her physician because of a syncopal spell. She said she experienced episodes of palpitations, weakness, severe fatigue, nausea and lightheadedness almost daily at work, usually following afternoon coffee breaks. She was a nonsmoker, but others in the staff lounge smoked. When tests were done on serum specimens taken during such an attack, her blood glucose level was 85 mg per dl (normal 75 to 115) and her carboxyhemoglobin concentration was 21.9% (normal less than 3%). The source of CO was a faulty catalytic converter in her car. Repair of the defect resulted in complete resolution of her symptoms within two weeks.

COMMENT. Patients who receive repeated sublethal doses of CO probably accumulate CO in body tissues. In this patient, small additional subsequent exposures from bus fumes and cigarette smoke may have been enough to provoke symptoms. Laboratory results from blood specimens obtained during an attack eliminated the possibility of hypoglycemia and established the diagnosis.

Discussion

More than 3,800 people die annually of carbon monoxide poisoning and at least 10,000 miss at least one day of work because of a sublethal exposure.[1] Considering how obvious the existence of a toxic environment seems once a CO source is found, one must consider why repeated occult CO exposure is seldom recognized. The index cases in our series were a family of five in a home with a faulty gas furnace. The wife, a homemaker with ongoing exposure to CO, became almost completely disabled. The husband's work performance declined, and the children's school work deteriorated. The family members' vague symptoms and impaired cognition initially misled their physicians. After her condition improved in hospital, the mother had a relapse. Only when other family members home for Christmas vacation began to note similar symptoms and when symptoms developed in the patient's mother after she came to stay with the family was the correct diagnosis of a toxic home environment made.

Diagnosis

Suspected chronic CO poisoning is difficult to document in the office. Ascertaining the carboxyhemoglobin level is the only way to quantitate exposure. No test of tissue levels of CO is available, though this information may be more closely related to the severity of symptoms than the carboxyhemoglobin level.[2] Carboxyhemoglobin determination is limited in usefulness because it correlates poorly with symptoms and it decreases in sensitivity as an indicator of CO poisoning as the time from last exposure approaches four hours because of carboxyhemoglobin's rapid clearance. Its half-elimination time in room air is about five hours, in 100% oxygen two hours and with hyperbaric oxygen only 23 minutes.[3]

Promptly collecting a serum specimen after exposure is, therefore, a critical factor in interpreting the carboxyhemo-

globin level. But if 100% oxygen has been administered to a comatose patient for one hour in the ambulance during transport and the car in which the patient tried to commit suicide has run out of gas, a carboxyhemoglobin level of 5% or less should not rule out the diagnosis of CO poisoning.

To further confound interpretation, carboxyhemoglobin levels are increased in smokers; a smoking history must be taken into account when interpreting the test results. According to the National Bureau of Health Statistics,[4] 98% of nonsmokers have a blood level of less than 2%. A smoker, whose carboxyhemoglobin level may be 10% to 15% after smoking, has no symptoms most of the time, perhaps because he or she gets a reprieve between cigarettes and during the night. The carboxyhemoglobin measurements of four smokers in this series ranged from 19.0% and 8.8% when tested within the four-hour period, down to 3.0% and 2.8% for two tested eight hours and seven days after exposure, respectively. These latter two measurements reflect the background level of CO exposure or endogenous production for these patients, rather than the exposure related to their increased symptoms. Obviously, smoking is a confounding variable and must be considered in evaluating the carboxyhemoglobin level.

Symptoms have failed to correlate well with the level of carboxyhemoglobin. For example, there are reports of patients with severe exposure (coma) who have carboxyhemoglobin levels of zero.[5] Others have levels in the 20% to 30% range with minimal symptoms. Patients in this series with long-term daily exposure may have many symptoms and yet have levels—5% to 15%—that are considerably lower than the arbitrary value set by the standard symptom tables.[6] These tables have been derived from cases of sudden and intense exposure and, even in that context, individual symptoms are highly variable at moderate to high levels of carboxyhemoglobin. The correlation of blood CO levels with the symptoms of patients with daily sublethal exposures is not known.

Confirming a diagnosis requires identifying an exposure source, compatible symptoms and prompt carboxyhemoglobin determination. Under these conditions, lower levels of carboxyhemoglobin, such as 5% to 15%, should be accepted as evidence of CO toxicity, but just what these values should be remains uncertain.

Controlled studies comparing air quality in homes and vehicles with carboxyhemoglobin levels of the occupants (with specimens drawn at the site) are not yet available, nor did we address this important issue. This testing requires suitable equipment and staff to go to the site, resources not available for this series. Patients made their own arrangements with utilities personnel to help investigate the condition of home heating systems.

Sources of CO

When an elevated level of carboxyhemoglobin is found, it can reflect a wide range of sources of CO, including combustion gases, cigarettes and absorption of a solvent that metabolizes to CO. Virtually any indoor appliance in which a fuel is burned without adequate ventilation can be a source of lethal or sublethal CO poisoning. Grace and Platt[7] reported the cases of two patients with myocardial infarction and pulmonary emboli who were exposed to carbon monoxide from a defective natural gas water heater and furnace. Fisher[8] reported the cases of 12 patients exposed to four kerosene space heaters. Stewart and Hake[9] implicated methylene chloride (the active solvent in paint strippers and carburetor cleaners) as the precipitating factor in two cases of fatal myocardial infarction. Sterno (canned fuel) used in a high school chemistry laboratory was responsible for multiple symptoms in several students.[10] Charcoal briquettes and pressed woodchip logs producing relatively smokeless fires generate large amounts of CO.[11] Aronow and Isbell pointed out that vehicle exhaust may have favored an onset of angina in patients exposed to Los Angeles freeway traffic.[12] Ice skaters and hockey players have also had symptoms from exposure to the exhaust of faulty ice-resurfacing machines.[13]

Wood, oil and kerosene are all possible sources of CO when there is inadequate oxygen available for combustion. The combustion of these fuels usually also produces irritating smoke that alerts the potential victim, but not invariably. Several of the patients in this series were unaware of such exposure when it occurred.

A special problem exists with gas appliances. Although natural gas burns most efficiently and most cleanly compared with other forms of fuel, it is also the most potentially lethal.[14] Of 340 CO deaths reported in 1982 by the US Consumer Product Safety Commission, 290, or 85%, were due to gas appliances. A major medical textbook is misleading when discussing CO poisoning. The statement, "much higher concentrations [of CO] are present in most illuminating and heating gases, but not in natural gas,"[15] although true, does not go the necessary step further to say "unless combustion is incomplete." In circumstances of inadequate ventilation or a defective exhaust pathway, natural gas appliances may emit potentially lethal CO *without any irritating fumes*. Therefore, natural gas appliances must be treated with utmost caution, and all suggested maintenance schedules must be followed. When unusual symptoms occur in patients who use gas appliances and these symptoms worsen during the heating season, appliances should be inspected.

Vehicle exhaust is the most common source of CO-induced morbidity and mortality. In particular, children who lie down in the rear of station wagons may be affected, even when the occupants of the front seats have no symptoms.[16] Faulty exhaust systems in cars, buses and trucks may also account for chronic symptoms of headache, dizziness, drowsiness, nausea, visual disturbances and fatigue. The incidence is unknown but probably substantial.

The use of catalytic converters (which convert carbon monoxide to carbon dioxide and water)[17] and emissions control testing, mandatory for vehicle license renewal in certain cities with excessive air pollution, have been helpful in reducing atmospheric CO content and identifying problem vehicles. Older cars that are not required to be tested, however, represent a potential CO source that remains undetected by current emission testing.

Finally, cigarette smoke is another source of CO and represents a hazard to the active smoker, to the passive bystander and, during a smoker's pregnancy, to the fetus.[18]

Pets

The effect of carbon monoxide on animals (see Table 3) deserves special comment. Miners used to take canaries or mice down mineshafts before CO detectors were available. If

the bird or animal died, they knew that levels of CO were high.

Physicians often inquire about recent travel or whether there are sick animals in the houses of patients with vague complaints. Their thoughts, however, usually revolve around transmission of infection through contaminated food and water sources or infected animals. Household pets are apt to be more severely affected than their owners by domestic carbon monoxide as they have smaller muscle mass, higher metabolic rates and may be indoors 24 hours a day. An autopsy diagnosis such as "pulmonary consolidation and emphysema" in the mysterious deaths of several four-week-old puppies in cases 8 and 9 should bring to mind the possibility of an environmental toxin such as carbon monoxide, particularly in a gas-heated home where the humans are also feeling ill.

Carbon monoxide poisoning might easily be misdiagnosed as "a viral syndrome," particularly during the home heating season. Transmission of infection from household pets to humans and vice versa does occur,[19] but the common "flu-season" viruses are not transmitted between pets and owners. Therefore, any unusual behavior or illness in domestic animals may provide a clue to the correct diagnosis.

Differential Diagnosis

The most common symptoms of any CO exposure are headache and fatigue, which, along with dizziness, nausea, trouble thinking and concentrating, palpitations and sleep disturbance, can be symptoms of depression. Clues that help differentiate CO poisoning from depression and other conditions are the health of pets and other family members, a negative social history and the presence of other unusual symptoms. Impaired visual acuity, which was the presenting complaint in three patients, should alert a clinician to the possibility of CO poisoning if a potential source is present.

In our series, a third of the patients had diarrhea and abdominal pain, symptoms not commonly associated with a diagnosis of CO poisoning. A plausible mechanism is hypoxia of the intestinal mucosa.

The workup for a patient with "spells" or syncope does not traditionally include questions about environmental CO sources or a test of the carboxyhemoglobin level. The frequency of this complaint in our patients (35%) suggests occult CO poisoning as a possible cause in the undiagnosed case.

Finally, CO poisoning is most frequently misdiagnosed as a viral syndrome. Distinguishing features of CO poisoning are the absence of myalgias, fever, sore throat and adenopathy; simultaneous illness in homebound family members and pets, and improvement with exposure to fresh air.

Nonspecific vague symptoms such as in this series often represent functional illness rather than CO poisoning. A causal association is supported, however, by finding a source of carbon monoxide in the home, workplace or vehicle; negative screening tests for other illnesses; an abnormal carboxyhemoglobin level in 11 of 14 patients, and abatement or resolution of symptoms when the source of carbon monoxide was removed. Furthermore, none of these patients have since reported recurrent symptoms.

Conclusion

The historical findings that should trigger an investigation of possible CO sources are persons whose occupations expose them to vehicle exhaust; households where people and pets are all feeling less well than usual; persons who use solvents containing methylene chloride, including paint strippers and carburetor cleaners, and anyone who has several of the symptoms associated with ongoing CO exposure.

The problem of CO accumulation in homes may become more frequent as more people insulate their homes. In the Pacific Northwest, where electric heat has long been much less expensive than other heat sources, electric power is now rising in cost. Many homes may be converted to using combustion fuels, with greater potential for CO leaks. Clinicians in other areas where higher priced electricity is seldom used for heating may already be seeing a significant number of patients with CO exposure not caused by smoking.

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