
Special Feature

Handling carbon monoxide poisoning cases

by Brien A. Roche and Eric Kessel

More people die or are injured worldwide by carbon monoxide than by any other poison.¹ Carbon monoxide is most dangerous because it is colorless, odorless, tasteless, and nonirritating. From a victim's point of view, carbon monoxide can be deadly. From an attorney's point of view, carbon monoxide cases can be especially dangerous.

Most carbon monoxide liability claims arise out of defective gas burners. Those gas burners may be hot water heaters or simply gas heating appliances. These cases primarily fall into three categories: improper design and construction; improper maintenance; and defective product theories. Typical defendants would be the owner of the premises, property management and maintenance companies, and, in the case of a defective product, the product manufacturer. In this sense, a carbon monoxide case is at heart just like any other premises liability or product liability case when it comes to theories of liability. The complexity, and real danger to the practitioner, comes from the science and theories of causation and damages. To understand the complexity of these cases, it is necessary to understand how these gas appliances work, the characteristics of carbon monoxide, the effect that carbon

monoxide has upon human beings and how those injuries may manifest themselves and how they are to be proven.

Let us begin at the beginning and talk about gas appliances in general. From a simplistic analysis any gas appliance involves an inlet valve, a burning chamber, and then a flue for the combustion products to exit from the burning chamber. The entry of gas into the burning chamber is frequently controlled at two different points. It may be controlled at the street level, which is where the gas company supplies the gas to the individual property owner by means of an underground pipe called the street level valve, or inlet valve. The gas pressure at that level as a general rule is irrelevant. Even if the gas pressure coming in at that point is too high, there is a secondary check point before the gas actually enters the burning chamber. That secondary check point is called the manifold gas valve. This valve controls the actual entry of gas into the burning chamber. If too much gas is being poured into the burning chamber, then there may be incomplete combustion of the gas. This incomplete combustion results in the production of an excessive amount of carbon monoxide, which in itself can be dangerous.

The second thing to look at in terms

of the gas appliance is whether the flue tubes are properly cleaned. If they are not cleaned and properly maintained, then there may be a blockage at that level which impedes the combustion of the gas and retards the exit of the combustion products out of the burning chamber. Any impediment to the complete combustion of the gas is a violation of the property maintenance code.²

Another factor to look at in the gas appliance is whether the chimney is of proper height and properly maintained. Regulations pertaining to chimney dimensions as well as vent locations are contained in Chapter 5 of the IFGC. The chimney is designed to maintain an updraft so that the products of combustion (the effluents) can properly exit from the burning chamber into the open air. The chimney has to be of sufficient height to allow that updraft. In addition, there generally has to be some sort of cap on the chimney to prevent the entry of rain and also to prevent outside wind from causing a downdraft. If that "mushroom" cap is rusted through or is not properly maintained, then with adverse wind conditions, you can have a situation where you are getting a downdraft in the chimney. This downdraft prevents the effluents from exiting up through the chimney, causing them to spill out of the

burning chamber and into the room where the gas appliance is maintained. The IPMC requires that chimneys be kept in good repair.³

Large hot water boilers generally will have a door to allow entry into the burning chamber for cleaning the flue tubes. That door or opening may be sealed with a gasket. If that gasket is defective, then that may allow the effluents to escape into the room where the boiler is maintained.

The boiler room itself has to be subject to some inspection. Typically a boiler room in a commercial building is sealed so that if the effluents escape from the burning chamber, they will be trapped in the boiler room and not get to the rest of the building and its occupants. In addition, the boiler room should be ventilated so as to draw those effluents out of the boiler room and into the open air, preventing injury to any occupants.

In most carbon monoxide cases, there is no one factor that causes the carbon monoxide poisoning. Typically it is a failure of the components mentioned above that together cause the escape of effluents into the occupied part of the building, resulting in injury or death.

Virginia mandates a state inspection of commercial boilers every two years.⁴ That inspection, however, is simply external, and does not involve any internal examination of the combustion components of a boiler. Most maintenance and HVAC personnel will agree that regular maintenance of any commercial boiler is necessary. That regular maintenance involves at least annual "tuneups" and of course, frequent inspection and maintenance as the need dictates. Another safety measure that is frequently employed and is very inexpensive is the installation of carbon monoxide detectors. This is especially important in sleeping quarters.⁵

Problems can also arise from the use of unvented room heaters. These unvented heaters are now required to have oxygen-depletion-sensitive safety shutoffs. IFGC, §620.6.⁶

One problem that frequently arises in carbon monoxide cases is trying to determine exactly how the carbon monoxide got from the boiler room to the occupied portions of the building. That can generally be determined by a smoke test using a smoke compound with the same density as carbon monoxide. Carbon monoxide is much like air in that it has essentially the same density as air and therefore travels in the same fashion as does air. A smoke test to determine the path of travel of the carbon monoxide in a particular case can be especially effective if it is videotaped so that a jury can see how the smoke was set loose in the boiler room and then can see how it emanates from vents in other parts of the building that were occupied by the injured parties.

Perhaps the most difficult hurdle in a carbon monoxide case is causation. To establish causation, you are going to need not only a medical doctor

to testify as to the plaintiff's condition, but also an expert who has a more detailed knowledge of carbon monoxide and its characteristics than what is possessed by your typical family doctor. Dr. David George Penny of Wayne State University Medical School is such an expert. He is a world-renowned expert on carbon monoxide exposure and the effects of carbon monoxide poisoning, and has written more about the subject than any other person alive.⁷ Another well-respected authority on carbon monoxide poisoning is Dr. Roy Meyer of the University of Maryland School of Medicine in Baltimore, Maryland. His 1986 article on carbon monoxide poisoning provides a good overview of the subject.⁸

Carbon monoxide is a gas produced by the incomplete combustion of carbon-containing materials.⁹ The main sources of carbon monoxide are fires, car-exhaust fumes, paint removers containing methylene chloride, wood stoves, Sterno fuel, and malfunctioning heating systems and appliances.¹⁰ During normal combustion, each atom of carbon in the burning fuel joins with two atoms of oxygen to form the relatively harmless gas carbon dioxide. When there is a lack of oxygen to ensure complete combustion, each atom of carbon links up with only one atom of oxygen, forming carbon monoxide gas. Carbon monoxide can escape from any fuel-burning appliance, furnace, water heater, fireplace, wood stove, or space heater. Carbon monoxide can spill from vent connections and poorly maintained or blocked chimneys. If the flue liner is cracked or deteriorated, carbon monoxide can seep through the liner and slowly creep up to dangerous levels. If a nest or other material restricts or blocks the exit of carbon monoxide from the burning chamber, then carbon monoxide can spill back into the building structure.

Carbon monoxide is breathed into the body through the nose and mouth and is filtered through the lungs where it is absorbed and dispersed throughout the body. Carbon monoxide accumulates in the body by binding to the hemoglobin in the red blood cells, thereby displacing the oxygen that is necessary to nourish the body's cells. In normal respiration, oxygen molecules attach to hemoglobin, which is contained in each red blood cell. The red blood cells transport the oxygen throughout the body. When carbon monoxide is present, the hemoglobin picks up the carbon monoxide molecules instead of the oxygen molecules, forming a toxic compound known as carboxyhemoglobin (COHb). Hemoglobin's affinity for carbon monoxide is approximately 200 to 250 times that for oxygen.¹¹ Because of this great affinity, substantial amounts of carbon monoxide can bind to hemoglobin even at very low exposure levels.¹² The severity of the poisoning is dependent primarily on the duration of the exposure as well as the concentration of carbon monoxide in the air.¹³

The concentration of carbon monoxide in the

air is measured in parts per million. An acceptable level of concentration is 10 parts per million. While OSHA regulations permit workers to be exposed to 35 parts per million for eight hours,¹⁴ even this level may present dangers.¹⁵

The formation of carboxyhemoglobin impairs the oxygen carrying capacity of the red blood cells as well as the release of available oxygen to body tissues. The experts agree that this oxygen deficiency is the factor responsible for initiating cellular injury. Because of the systemic nature of the oxygen deprivation, virtually all body cells are affected by the carbon monoxide, though the primary targets are the heart and brain.¹⁶

The displacement of the oxygen in the hemoglobin results in a lack of necessary oxygen being transported to the various systems within the body. As a result, various neurological symptoms may be manifested by exposure victims. In addition, it is believed that the attachment of those carbon monoxide molecules to the hemoglobin, aside from simply displacing the oxygen, has a destructive effect upon the myoglobin and cytochromes within cells. Further, carbon monoxide molecules increase oxygen's adhesion to hemoglobin, thus making it more difficult for oxygen molecules to move from the hemoglobin into the body's cells.

To summarize, the effect of carbon monoxide on the body is as follows:

1. Displacement of oxygen from hemoglobin resulting in incomplete oxygenation of the body;¹⁷
2. Destruction of the myoglobin and cytochromes within cells, thus decreasing cellular respiration and potentially causing myocardial, skeletal muscle, and central nervous system dysfunction;¹⁸ and
3. Increased adhesion of oxygen molecules to hemoglobin, inhibiting effective transfer of oxygen from the hemoglobin to the cells.¹⁹

All of these conditions cause a lack of oxygen to the brain and/or heart, resulting in potential heart and brain damage.

The diffuse neurological symptoms that are exhibited typically do not produce mass lesions that can be portrayed on available radiological studies or other types of diagnostic tests. As such, a gross neurological exam will not display any readily discernable symptoms.²⁰ Studies are ongoing regarding whether carbon monoxide poisoning leaves behind specific biochemical markers that would indicate brain injury.²¹ Neuropsychological testing may present a better means of assessing damage resulting from mild carbon monoxide poisoning.²²

The array of symptoms that are typically exhibited as a result of carbon monoxide poisoning include:

- Headache²³
- Lightheadedness, weakness, sleepiness
- Decreased exercise tolerance

- Visual disturbances
- Palpitations
- Chest pain
- Nausea and vomiting
- Rapid breathing and rapid heart rate
- Fever
- Confusion, disorientation
- Sinus problems
- Earaches
- Shortness of breath
- Dizziness
- Hypotension
- Arrhythmia
- Fainting
- Coma
- Convulsions
- Respiratory failure

Moderate levels of poisoning are usually manifested by headache, dizziness, weakness, nausea, confusion, shortness of breath, visual disturbances, chest pain, loss of consciousness, abdominal pain, and muscle cramping.²⁴ Because of the wide array of symptoms associated with carbon monoxide poisoning, it is estimated that one-third of all victims of such poisoning are misdiagnosed.²⁵

As mentioned above, a measurement that is employed to determine the level of carbon monoxide in the blood is known as the carboxyhemoglobin (COHb) level. This level is stated as a percentage ranging from 0% to 100%. Anything over 70% is generally considered to be fatal.²⁶ Anything in the range of 10% to 50% can generally produce the diffuse neurological symptoms that are mentioned above.²⁷ A COHb level of 10% is equal to an air concentration of 70 parts per million.²⁸

The defense may frequently focus on carboxyhemoglobin (COHb) levels as evidence of the fact that the plaintiff could not have suffered significant injury. Frequently, however, COHb levels taken at a hospital are not illustrative of true levels of exposure because they are generally taken after the victim has been given pure oxygen by the rescue squad at the scene, additional pure oxygen en route to the hospital, and then, in many cases, given additional pure oxygen upon arrival at the hospital. The victim may also have been out in the open air for an extended period of time before the rescue squad arrives at the scene.²⁹ Finally, the COHb level does not reflect the length of exposure, or "soak time." A patient with a low COHb level and a high "soak time" may have suffered significantly more damage to his cellular structure than one with a high COHb level and a low "soak time."³⁰

Carbon monoxide removal can be sped up by raising the oxygen concentration in the blood either by giving the patient pure oxygen at normal atmospheric conditions (normobaric oxygen), or by placing the patient in a pressurized chamber, called a hyperbaric oxygen chamber, where the pure oxygen is administered under a higher pres-

sure, generally at two to three times the normal atmospheric pressure.³¹ This increase in pressure increases the amount of oxygen dissolved in the blood plasma (from 0.32 to 6.0 mL oxygen / 100 mL blood at three atmospheres)³² and bypasses the bound hemoglobin.³³ In addition, hyperbaric treatment speeds the elimination of carbon monoxide.³⁴ This hyperbaric treatment is generally considered to be most effective when provided as soon as possible.³⁵ There is some controversy as to whether this hyperbaric treatment, provided days or weeks after the exposure, has any real effect.³⁶ The proper administration of 100 percent oxygen washes the system of carbon monoxide. The sooner that oxygen therapy is provided after the exposure, the greater chance there is that the patient will avoid permanent injury. Carbon monoxide, unlike many other foreign elements that invade the body, does its damage and then leaves. If a victim is properly and promptly treated, the damage to the organs and musculature may be minimal or nonexistent; however, if the damage has been done to the organs and/or musculature then the victim may well be left with significant symptoms despite treatment.

Another factor that needs to be considered in that regard is that if the people exposed are smokers, then they may typically have a COHb level of up to 7%.³⁷ As such, they may normally have a higher COHb level than would a nonsmoker. Nonsmokers typically have COHb levels between 1 percent and 3 percent.³⁸

More troubling is the question of the delayed onset of symptoms. Studies have shown that anywhere from 2.8 percent³⁹ to 40 percent⁴⁰ of the victims of acute carbon monoxide intoxication present delayed neurologic sequelae. These victims have a lucid interval during which they appear to have recovered, only to suffer subsequent deterioration.⁴¹ This lucid interval ranges from three to 240 days.⁴² Between 50 and 75 percent of those afflicted with the delayed onset of symptoms recover within one year.⁴³ The delayed onset of symptoms, coupled with the difficulty in making the original diagnosis of carbon monoxide poisoning in the absence of a clear history of exposure,⁴⁴ can make it difficult first for patients to receive appropriate care and treatment, and second, for a nexus between the exposure and the injury to be established to a judge or jury's satisfaction.

In terms of discovery, some of the basic items that need to be obtained in a carbon monoxide case are the following:

1. All fire department reports and documents on the exposure;
2. All Hazmat readings of carbon monoxide;
3. Building, architectural, and mechanical plans for the site of the exposure;
4. All records of service and inspection of the appliance that is believed to be the source of the carbon monoxide;

5. The identity of all persons in the building at the time of exposure;
6. The identity of all persons reporting carbon monoxide exposure or that were treated for such; and
7. All notices of violation as to the appliance that is believed to be the source of the carbon monoxide.

In terms of case selection, prospective clients with radiologically identifiable findings fit the ideal case pattern. Cases with diffuse neurological symptoms and negative neurological findings are more difficult and will require more medical and lay testimony as to causation and therefore, more circumspection as part of the case selection process.

Endnotes

1. Stephen R. Thom, et al., "Neuronal Nitric Oxide Synthase and N-methyl-D-aspartate Neurons in Experimental Carbon Monoxide Poisoning," 19:4 *Toxicology and Applied Pharmacology*, 280-295, 280 (2004).
2. In Virginia, the Board of Housing and Community Development has the power to adopt and promulgate a Uniform Statewide Building Code. Va. Code §36-98. The Building Code is found at VAC §5-62-10 *et seq.* The International Property Maintenance Code 2000 (IPMC), published by the International Code Council, Inc. (ICC), is incorporated by reference. Uniform State Building Code §127.1. Other ICC model codes, including the 2000 International Fuel Gas Code (IFGC), International Building Code 2000 (IBC), 2000 International Residential Code for One- and Two-Family Dwellings (IRC), and the 2000 ICC International Mechanical Code (IMC) are also incorporated by reference. Uniform State Building Code §108.1. *See* IPMC §603.1 (all mechanical appliances shall be maintained in safe working condition); §603.5 (requiring an air supply sufficient for complete combustion).
3. IPMC §303.11.
4. Va. Code §40.1-51.10(c)(2); 16 VAC 25-50-30. Note that boilers located in private residences or in apartment buildings of less than four units are exempt from this inspection. Va. Code §40.1-51.8(2).
5. While Virginia has adopted regulations regarding the use and placement of smoke detectors, no regulations have been adopted requiring the use of carbon monoxide detectors. *See* IPMC §704.1 (requiring smoke, but not carbon monoxide, detectors).
6. The input rating of such heaters is also limited by the size of the room. IFGC §620.5.
7. Dr. Penny's website is <http://www.coheadquarters.com>.
8. "Carbon Monoxide Poisoning," 34:5 *American Family Physicians*, 186-194 (1986).
9. Joseph Varon, et al., "Carbon Monoxide Poisoning: A Review for Clinicians," 17:1 *J. Emerg. Med.* 87-93, 87 (1999).
10. *Id.* at 88.

11. Alan Abelsohn, et al., "Identifying and Managing Adverse Environmental Health Effects: 6. Carbon Monoxide Poisoning," 166:13 *Canadian Medical Association Journal* 1685-1690, 1686 (2002).
12. *Id.*
13. Larry G. Martindale, "Carbon Monoxide Poisoning: The Rest of the Story," 15:2 *J. Emergency Nursing* 101-104, 102 (1989).
14. The Canadian Federal-Provincial Advisory Committee on Environmental and Occupational Health recommends limits of 11 ppm over 8 hours in residences, with a maximum concentration of 25 ppm over one hour. Abelsohn, *supra*, at 1687.
15. Lindell K. Weaver, "Carbon Monoxide Poisoning," 15:2 *Critical Care Clinics: Environmental Emergencies* 297-317, 310 (1999).
16. *See, Id.* at 303-306 for a more thorough discussion of the pathophysiology of carbon monoxide intoxication.
17. Louis Marzella & Roy A. M. Myers, "Carbon Monoxide Poisoning," 34:5 *Am. Fam. Physician* 186-194 (1986).
18. Jose-Ramon Alonso, et al., "Carbon Monoxide Specifically Inhibits Cytochrome C Oxide of Human Mitochondrial Respiratory Chain," 93:3 *Pharmacology & Toxicology* 142-146 (2003). *See Varon* at 88.
19. Marzella & Myers.
20. *See* Kazumasa Sakamoto, et al., "Clinical Studies on Three Cases of the Interval Form of Carbon Monoxide Poisoning: Serial Proton Magnetic Resonance Spectroscopy as a Prognostic Predictor," 83 *Psychiatry Research: Neuroimaging* 179-192 (1998) (reporting that neuroimaging findings normalize over time and that IMP SPECT findings did not always reflect the clinical symptomology). *See also* Jeffrey S. Jones, et al., "Computed Tomographic Findings After Acute Carbon Monoxide Poisoning," 12:4 *Am. J. Emerg. Med.* 448-451 (1994) (finding it possible that some patients with a normal CT scan will experience delayed neurological symptoms). *But see* Kon Chu, et al., "Diffusion-Weighted MRI and 99mTc-HMPAO SPECT in Delayed Relapsing Type of Carbon Monoxide Poisoning: Evidence of Delayed Cytotoxic Edema," 51:2 *European Neurology* 98-103 (2004) (finding diffusion-weighted magnetic resonance images can show brainstem abnormalities associated with carbon monoxide poisoning that do not show up on a conventional MRI); Kee Hyun Chang, et al., "Delayed Encephalopathy after Acute Carbon Monoxide Intoxication: MR Imaging Features and Distribution of Cerebral White Matter Lesions," 184 *Radiology* 117-122 (1992) (finding a correlation between white matter cerebral lesions and symptomology in cases of acute carbon monoxide poisoning).
21. *Compare* Miran Brvar, et al., "The Potential Value of the Protein S-100B Level as a Criterion for Hyperbaric Oxygen Treatment and Prognostic Marker in Carbon Monoxide Poisoned Patients," 56:1 *Resuscitation* 105-109 (2003) and Miran Brvar, et al., "S100B Protein in Carbon Monoxide Poisoning: A Pilot Study," 61:3 *Resuscitation* 357-360 (2004) (reporting link between elevated S100B levels and carbon monoxide poisoning) with L. S. Rasmussen, et al., "Biochemical Markers for Brain Damage After Carbon Monoxide Poisoning," 48:4 *Acta Anaesthesiol Scand* 469-473 (2004) (finding no significant difference in NSE or S-100B levels between control subjects and carbon monoxide poisoned subjects).
22. Sherral A. Devine, et al., "MRI and Neuropsychological Correlates of Carbon Monoxide Exposure: A Case Report," 110:10 *Environmental Health Perspectives* 1051-1055, 1055 (2002); Donna Seger & Larry Welch, "Carbon Monoxide Controversies: Neuropsychologic Testing, Mechanism of Toxicity, and Hyperbaric Oxygen," 24:2 *Annals Emerg. Med.* 242-248, 243 (1994).
23. Headache is the most common symptom, presenting in 90 percent of the subjects in one study. However, the location and intensity of a carbon monoxide induced headache is highly variable. Neil B. Hampson & Lindsay A. Hampson, "Characteristics of Headache Associated with Acute Carbon Monoxide Poisoning," 42:3 *Headache* 220-223, 221-222 (2002).
24. Armin Ernst & Joseph D. Zibrak, "Carbon Monoxide Poisoning," 339:22 *New Eng. J. Med.* 1603-1608, 1605 (1998).
25. Abelsohn, *supra*, at 1687.
26. Martindale, *supra*, at 101.
27. *Id.*
28. Abelsohn, *supra*, at 1688.
29. COHb has a half-life of four to five hours when a poisoning victim is breathing normal, carbon-monoxide free air. This half-life is reduced to between 45 to 80 minutes when the victim is breathing NBO 100 percent oxygen. Devine, *supra*, at 1053.
30. Martindale, *supra*, at 102. *See* Carlos D. Scheinkestel, et al., "Hyperbaric or Normobaric Oxygen for Acute Carbon Monoxide Poisoning: A Randomised Controlled Clinical Trial," 170 *Med. J. Australia* 203-210 (1999) (finding no correlation between COHb level and efficacy of hyperbaric treatment).
31. Neil D. Gillespie, et al., "Severe Parkinsonism Secondary to Carbon Monoxide Poisoning," 92 *J. Royal Soc. Med.* 529-530, 530 (1999).
32. Dan Waisman, et al., "Hyperbaric Oxygen Therapy in the Pediatric Patient: The Experience of the Israel Naval Medical Institute," 102:5 *Pediatrics* e53 (1998).
33. Martindale, *supra*, at 103.
34. HBO treatment is approximately four times faster than pure oxygen treatment, and 16 times faster than fresh air treatment. *Id.*
35. P. O'Donnell, et al., "The Magnetic Resonance Imaging Appearances of the Brain in Acute Carbon Monoxide Poisoning," 55:4 *Clinical Radiology* 273-280, 278 (2000). *See* Stephen R. Thom, et al., "Delayed Neuropsychologic Sequelae After Carbon Monoxide Poisoning: Prevention by Treatment with Hyperbaric Oxygen," 25 *Am. Emerg. Med.* 474-480 (1995).
36. *See* Kent R. Olson & Donna Seger, "Hyperbaric Oxygen for Carbon Monoxide Poisoning: Does it Really Work?" 25 *Ann. Emerg. Med.* 535-537 (1995). *See also* Thom, "Delayed Neuropsychologic Sequelae." *See also* Benjamin Gilmer, et al., "Hyperbaric Oxygen Does Not Prevent Neurologic Sequelae after Carbon Monoxide Poisoning," 9:1 *Academic Emergency Medicine* 1-8, 6 (2002) (theorizing that HBO treat-

ment may reduce the body's natural production of adenosine and prevent the adenosine from acting to reduce excitotoxic brain damage); Scheinkestel (finding hyperbaric treatment to be detrimental). *But see* Lindell K. Weaver, et al., "Hyperbaric Oxygen for Acute Carbon Monoxide Poisoning," 347:14 *New Eng. J. Med.* 1057-1067, 1065 (2002) (reporting that HBO treatment appears to reduce the frequency of cognitive symptoms arising six weeks after acute carbon monoxide poisoning by 46 percent, as compared with patients receiving NBO treatment); Robert L. Sheridan & Eric S. Shank, "Hyperbaric Oxygen Treatment: A Brief Overview of a Controversial Topic," 47:2 *J. Trauma: Injury, Infection and Critical Care* 426-435 (1999) (noting studies demonstrating the efficacy of delayed hyperbaric treatments).

37. Martindale, *supra*, at 102.
38. Abelson, *supra*, at 1686.
39. Chang, *supra*, at 119.
40. Martindale, *supra*, at 101.
41. Weaver, *supra*, at 309.
42. Ernst & Zibak, *supra*, at 1605.
43. *Id.*
44. See Varon, *supra*, at 90 (discussing misdiagnoses of flu, depression, and food poisoning); Jones at 449 (discussing reports of misdiagnoses of epilepsy, cerebral tumors, and strokes).

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