

## Defending Carbon Monoxide Claims

by Steven E. Leder and Kevin J. Willging

Tennis star Vitas Gerulaitis, the winner of the Australian Tennis Open, was found dead of carbon monoxide (“CO”) poisoning in 1995 after he went into a beach guest cottage adjacent to a pool heater for a nap in Southampton, Long Island. In the same year, forty-four people were hospitalized in Virginia Beach after gas leaked from a water-heating furnace at a beach hotel. In 1998, eight people were found dead of CO poisoning in Lake Placid, Florida. Data published by the National Center for Health Statistics reported 56,133 CO-related fatalities in a 10-year period.

One reason that carbon monoxide claims are so common is that CO is everywhere. CO is produced by automobiles, furnaces, gas water heaters, fireplaces, wood stoves, gas stoves, gas dryers, charcoal grills, lawnmowers, snow blowers and other yard equipment and cigarettes—anything that burns. One of the reasons it is so deadly is that there are few warning signs that signal a problem; it is colorless, odorless, tasteless, and non-irritating.

CO can cause headache, weakness and dizziness, confusion, dimness of vision, nausea, vomiting, collapse and death. Most of the deaths occur at home while the victims are asleep. Yoon, et al, “Deaths from unintentional carbon monoxide poisoning and potential for prevention with carbon monoxide detectors,” 279 J.Am.Med.Assn. 685 (1998). CO poisoning may be intentional or accidental. It is the leading cause of death due to toxic exposure in the United States. Cobb, “Unintentional carbon monoxide-related deaths in the United States,” 266 J.Am.Med.Assn. 659 (1991).

Plaintiffs frequently allege permanent injuries as a result of the most casual exposure. The natural reaction of most people (and jurors) when they hear that a plaintiff was exposed to CO is to think that they must have suffered brain damage or death. Plaintiffs and their attorneys prey on the uninformed by focusing on this general fear: if they were exposed to CO, they must be harmed, and all of the plaintiff’s symptoms must be the result of those fumes. This has been called the “one molecule theory;” i.e., that exposure to one molecule of a toxin can be responsible for the most severe permanent injury that the toxin can produce.

Toxicologists have a saying: “the poison is in the dose.” CO, like all toxins, is only dangerous when you are exposed to too much of it. CO poisoning may be acute, subacute, or chronic. You can rebut the plaintiff’s allegations with specific knowledge of CO and facts concerning the exposure.

### Liability

There are four elements which a plaintiff must prove in order to find liability in a CO case: (1) exposure to CO; (2) in an amount sufficient to cause injury; (3) due to the fault of the defendant; and (4) resulting in the plaintiff having symptoms consistent with the CO exposure. See, e.g. *Thompson v. PetroUnited Terminals, Inc.*, 536 So.2d 504 (La. Ct. App. 1989) (death by fire rather than CO poisoning); *Hanlon v. Lane*, 98 N.E.2d 26 (Ohio Ct. App. 1994) (no duty by utility company to warn customers of hazard of CO); *Raschke v. Carrier Corp.*, 703 P.2d 556 (Ariz. Ct. App. 1985) (lack of sensing mechanism which would turn off furnace not a defect) *Sears, Roebuck and Co. v. Harris*, 630 So.2d 1018 (Ala. 1994) (causation); *Ashley v. R.D. Columbia Assoc.* 54 F.3d 498 (8<sup>th</sup> Cir. 1995) (causation); *Harris*

*v. Atlanta Stove Works, Inc.*, 428 So.2d 1040 (La. 1983) (myocardial infarction consistent with exposure to CO); *Durden v. Hydro Flame Corp.*, 955 P.2d 160 (Mont. 1998) (whether plaintiffs exposed in trailer and whether symptoms from CO are issues for jury).

The theories of liability will vary depending upon the status of the defendant. If the defendant is a furnace manufacturer, for example, the plaintiff may bring the usual products liability causes of action; i.e., strict liability, breach of warranty, and negligence. *See, e.g. Sears, Roebuck and Co., supra; Reddick v. White Consolidated Indus.* 295 F.Supp. 243 (S.D. Ga. 1969). If the defendant is a landlord, the only viable action may be negligence. Regardless of the theory, there is usually a requirement that the defendant did something wrong. However, in some states the doctrine of *res ipsa loquitur* is available. *See, e.g., Weeks v. Rupp*, 966 S.W.2d 387 (Mo. Ct. App. 1998).

Let's look at an example. Suppose your client is the owner of an apartment building. The plaintiff testifies that he and his family awoke in their apartment with blistering headaches and nausea. They thought it was the flu or food poisoning, so they went back to bed. When their baby lost consciousness, they went to the hospital where an alert physician ordered carboxyhemoglobin tests, which revealed the baby's blood contained 12 to 20 percent of carbon monoxide. Since that time, the entire family claims it has suffered migraine headaches every day, twitching, slurred speech, disrupted vision, and cognitive problems.

How do you defend against such debilitating symptoms which allegedly occurred on the premises of your client?

#### Find the Source of the Carbon Monoxide

The first step for the lawyer defending the owner of the apartment building is to determine the origin of the CO. The boiler and the furnace are obvious candidates, but do not forget to look for other combustion equipment that may be within the separate control of the occupant of each apartment; e.g., stoves, unvented gas or kerosene space heaters, clothes dryers, fireplaces, improperly vented appliances. Is a gas oven being used inappropriately as a home-heating source? Is a hibachi or charcoal heater being used indoors? "Mourners Gassed at Mother's Wake," My Yahoo!—News Alerts—Reuters, March 27, 2000. The local fire department, HazMat response crews, OSHA inspectors, or EPA agents may have been called to the scene. If so, they probably prepared a report, which may help you or hurt you. The gas or fuel oil company or the contractor that installed or maintained the furnace may have been called to the scene. These are, of course, biased sources.

You should also obtain the building records from your client. These include: the purchase, repair and/or maintenance records of the HVAC system; the records of complaints and responses from any tenants; and records of cleaning services, including use of equipment which may generate CO.

In most cases, you should immediately hire a consultant to inspect the premises before they are substantially changed. The consultant may be a heating contractor, an engineer, or an industrial hygienist, depending upon the circumstance. If the boiler or the furnace is the culprit, find out whether the landlord performed proper maintenance or had notice of a problem. If there is no notice or reason to know of a problem, the landlord may be able to avoid liability. In many states, the plaintiff must prove that the owner or management company of the premises was on notice of the defect that caused the harm in order to hold them liable. *See, e.g., Burnworth v. Harper et al.*, 109 Ohio App.3d 401, 672

N.E. 2d 241 (Ohio Ct. App. 1996); *Livingston v. Begay*, 652 P.2d 734 (N.M. 1982); *Rodriguez v. Houston Corp.*, 167 So.2d 746 (Fla. Dist. Ct. App. 1964); *Hatch v. Sokolow*, 151 So.2d 341 (Fla. Dist. Ct. App. 1963).

Witnesses tend to disappear quickly; so it is best to obtain their statements (and important personal information to track them down later) as quickly as possible. If the local television station or newspaper covers the incident, get copies of the video or articles. The media, while not always accurate, may identify witnesses and other sources of information.

### Exposure to Carbon Monoxide

Plaintiffs must prove that their complaints are due to exposure to carbon monoxide. This can be a significant hurdle since CO is odorless and colorless. A governmental agency will often take air samples that can pinpoint the specific gas or fume that was released. After all, if the exposure is to another gas and not CO, your client may be exonerated. However, air samples are usually not taken immediately after an incident. If no air samples were taken at the time of the exposure, the *plaintiff* must re-create the circumstances of the incident in order to pinpoint what, if anything, was released. If the plaintiff does follow this route, compare the information you found in your investigation with the true properties of CO. Frequently the claims and the properties will not match.

If the plaintiff’s industrial hygienist “re-creates” the exposure, closely examine the circumstances and the manner in which the air samples are taken and analyzed. Many, if not most, re-creations are set up in such a manner that their real purpose is to validate the plaintiff’s claim. In these circumstances, it is important to retain your own industrial hygienist as an expert or consultant. He or she can be your environmental detective—poking holes in the plaintiff’s case, helping you discover what really happened, and explaining it to the jury.

### Amounts Sufficient to Cause Injury

A number of governmental agencies and associations set standards for permissible amounts of exposure to CO—the Occupational Safety and Health Administration (OSHA), the National Institute for Occupational Safety and Health (NIOSH), and the American Conference of Governmental Industrial Hygienists (ACGIH). The standards differ. The current OSHA permissible exposure limit (PEL) is 50 ppm (parts per million) of air as a time weighted average (TWA) over an eight-hour period. The NIOSH recommends an exposure limit (REL) of only 40 ppm TWA, and the ACGIH has a 25 ppm Threshold Limit Value (TLV) as a TWA over a 40-hour work week. See *Occupational Safety and Health Guideline for Carbon Monoxide* (OSHA April 13, 2000). The following chart demonstrates the effects of CO at various levels of exposure.

Level of Exposure	Effects
12,800 ppm	Death within 1 to 3 minutes
1,600 ppm	Nausea within 20 minutes; death within 1 hour
800 ppm	Nausea and convulsions, death within 2 hours
400 ppm	Frontal headaches 1 to 2 hours; life

	threatening after 3 hours
25 to 50 ppm	Maximum concentration for continuous exposure in any 8 hour period
9 ppm	Maximum acceptable level of CO in a living space
0 ppm	Desirable level

Thus, it is important to determine the period the plaintiff was allegedly exposed to the CO, as well as the level of exposure. For instance, an acute exposure requires a much higher level of CO content to cause permanent injury than does chronic exposure. Duration and concentration measure toxic exposure. Eaton & Klaassen, *Casarett and Doull's Toxicology: The Basic Science of Poisons*, at 15 (5th ed. 1996).

#### The COHb Level

After the alleged exposure to CO, the exposed person must be tested to determine just how much CO he has in his circulatory system. This amount is called the carboxyhemoglobin, or COHb, level. When CO reacts with the hemoglobin in the blood, it forms COHb. COHb prevents the hemoglobin from transferring oxygen.

CO exposure is frequently determined by drawing a blood sample and then sending it to a laboratory for blood/gas analysis. Results may be obtained as quickly as two hours if the analysis is undertaken immediately after the exposure. These tests will reveal the COHb level in the patient's blood, and are a good measure of whether the plaintiff was exposed to a high level of CO that may cause injury.

COHb tests are critical for determining the extent of the likely injury. The chart below, from Gossel, et al., *Principles of Clinical Toxicology*, at 113 (3d ed. 1994), demonstrates the relationship between levels of COHb in the patient's blood and resulting signs and symptoms.

<b>% COHb</b>	<b>Signs and Symptoms</b>
0-10	No symptoms; asymptomatic
10-20	Tightness across the forehead, possible slight headache, dilation of the cutaneous blood vessels, exertional dyspnea
20-30	Headache and throbbing in the temples, easily fatigued, possible dizziness
30-40	Severe headache, weakness, dizziness, confusion, dimness of vision, nausea, vomiting, and collapse
40-50	Same as above, a greater possibility of collapse, syncope, and increased pulse and respiratory rate
50-60	Loss of consciousness, increased respiratory and pulse rate, coma, intermittent convulsions and Cheyne-Stokes respiration
60-70	Coma, intermittent convulsions, depressed heart action and respiratory rate, and possibly death
70-80	Weak pulse, slow respiration, respiratory failure, and death within a few hours
80-90	Death in less than an hour

% COHb	Signs and Symptoms
90+	Death within a few minutes

The period that the plaintiff was breathing normal air after the exposure will influence the COHb level. The fact that an individual has a detectable level of CO in his or her system does not necessarily indicate that an injury has occurred. In fact, normal nonsmoking adults may have about one percent COHb in their blood. Heavy smokers may have 5-10 COHb. Certain groups are at higher risk for CO poisoning—infants/children, pregnant women, elderly people, heart patients, and anyone with anemic conditions.

#### Are the Symptoms Consistent with Exposure to Carbon Monoxide?

Exposure to CO can cause a number of difficulties, most of which are fleeting in nature. Early symptoms, such as shortness of breath, confusion, headache, general weakness, dizziness, dimness of vision, clumsiness, nausea, and vomiting, are found when the COHb reaches 20 to 40 percent. Adams, Victor & Ropper, *Principles of Neurology*, at 1113 (6th ed. 1997). Blindness, visual field defects, and inflammation of the optic disc may occur at slightly higher levels. COHb levels of 50-60 percent and higher are associated with coma, seizures, and death. Many of these symptoms are subjective and cannot be objectively documented.

Plaintiffs often “throw in the kitchen sink” of ailments; i.e., they allege all sorts of symptoms, many of which are incompatible with the nature of CO poisoning. For example, CO does not cause wheezing and coughing. Therefore, if a plaintiff says he was exposed to CO, and has ongoing coughing and wheezing, his problems (assuming they exist) are probably not related to the exposure.

CO exposure rarely causes symptoms with delayed onset. In a small percentage of cases, neurological deterioration may occur one to three weeks after exposure. *Id.* One study found delayed neurological deterioration in three percent of those exposed and twelve percent of those who sought treatment. See Choi, “Delayed neurologic sequelae in carbon monoxide intoxication,” 40 *Arch.Neurol.* 433 (1983). Yet, in litigation, a large portion of persons allegedly exposed to CO will claim delayed onset of neurological deterioration.

Cherry-red coloration of the skin sometimes is seen, but it is rare and not considered a reliable diagnostic sign. Berkow, *The Merck Manual*, at 2566-67 (16th ed. 1990).

Symptoms from carbon monoxide poisoning, as with alcohol, usually subside shortly after the COHb returns to normal. Still, the plaintiff often claims permanent injury. To succeed in his or her lawsuit, the plaintiff must establish that the exposure was sufficient to cause the alleged permanent condition.

#### Treatment

The key to recovery from CO poisoning is to get the CO out of the patient’s system. Removing the individual from the source of exposure is usually enough where the level of COHb in the blood is less than 40 percent.

When the COHb is over 40 percent, a more proactive approach is needed: hyperbaric oxygen must be administered. This procedure involves people breathing oxygen at greater than normal pressure (up to two atmospheres) and in greater concentration in the air they breathe (e.g., 80 percent oxygen). (The air we breathe normally contains about 21

percent oxygen). The theory is that the increase in surrounding pressure displaces the carbon monoxide from the tissues and replaces it with fresh oxygen.

### Doing your Homework

In preparing for the defense of your client, a CO poisoning claim is treated the same as other toxic exposure cases. You need to obtain a complete medical history of the claimant and a complete history of his or her exposure to other toxic substances. Check all of the plaintiff's medical records that you can access. Pay particular attention to the complaints/symptoms immediately after the alleged exposure. Try to obtain: emergency room records; tests (pulmonary function tests, carboxyhemoglobin saturation tests, blood gases, etc.); x-rays; records from doctor visits; work/school evaluations/examinations; and prior/subsequent lawsuits or workers' compensation claims records. Work history (e.g., furnace repair), residential history (older houses with antiquated heating systems), and hobbies (tinkers with antique automobiles in his garage) may turn up alternative possible sources of carbon monoxide or other toxic exposure. Frequently, the cause of the plaintiff's symptoms is other toxic exposures, other medical problems, or other psychological problems.

### Experts

Early retention of consultants and experts who are knowledgeable about CO poisoning is essential in this area of litigation.

An industrial hygienist will assist counsel in determining the source of the exposure and its amounts. How do you find such an expert? The most highly qualified may be members of the American Industrial Hygienists Association. Obtain a list of "full" members of the AIHA. Beyond full membership, the AIHA offers the title of "Certified Industrial Hygienist" for those who have achieved certain experience and passed a written test.

A toxicologist may provide evidence as to the effects of CO and causation. There are a number of organizations from which you may find a qualified expert. They include the American Board of Applied Toxicology, the American Academy of Clinical Toxicology, and the American College of Toxicology.

A neurologist is an expert who can testify as to medical causation. The neurologist can be particularly helpful if the plaintiff complains of a delayed onset of symptoms.

A neuropsychologist/neuropsychiatrist may assess cognitive injury and can testify as to cognitive dysfunction. The neuropsychologist should not, however, be permitted to testify as to causation.

Each of these experts retained by defense counsel can provide helpful advice as counsel prepares to depose similar experts for the plaintiff.

### Conclusion

The defendant is automatically at a disadvantage in a carbon monoxide poisoning case. The jury already knows that CO can cause brain damage, and it can kill. Therefore, defense counsel must start to educate the jury in the opening statement. Every substance has a safe dose, including CO. Everything is toxic if you take too much of it. Too much aspirin, too many vitamins, too much water—all are potentially toxic. Every day each one of us is exposed to many sources of CO, including car exhaust and cigarette smoke. The human body is equipped to deal with CO in the levels we are exposed to every day. It is rare indeed

for the plaintiff to have been exposed to CO levels that will correlate with the symptoms he or she is claiming.

Often the true facts differ sharply from the headline that appears in the morning paper after the incident. The source, the duration of exposure, the level of injury—all are factors that you must investigate, along with your experts. You can win carbon monoxide cases by conducting a thorough investigation of the plaintiff and a developing a thorough knowledge of the science.

## **AUTHORS**

Steven E. Leder is a partner, and Kevin J. Willging is a former associate with the Baltimore law firm of Niles Barton & Wilmer. Both authors are members of the Defense Research Institute. Thomas W. Hale, a law student at the University of Baltimore Law School, assisted in the preparation of this article.

## **[Sidebar]**

Defense of a CO case involves a thorough understanding of the science of CO exposure. The following is a list of useful references for CO and other toxic exposure cases.

### **Books**

Eaton & Klaassen, *Casarett & Doull's Toxicology: The Basic Science of Poisons* (5th ed. 1996).

Goldfrank, et al., *Goldfrank's Toxicologic Emergencies* (3d ed. 1986)

Gossel & Bricker, *Principles of Clinical Toxicology* (3d ed. 1994)

Kaye, Sidney, M.Sc., Ph.D., D.A.B.C.C. Handbook of Emergency Toxicology, A guide for Identification, Diagnosis and Treatment of Poisoning. 5<sup>th</sup> ed. Illinois: Charles C. Thomas, 1988.

### **Medical Journals**

National Library of Medicine (free access to MEDLINE) (<http://nlm.nih.gov>)

Kales, Stephen. "Carbon Monoxide Intoxication." Am Fam Physician 1993;48(6): 1100-4.

Hee, Jean, et al. "Smokers' Behaviour and Exposure According to Cigarette Yield and Smoking Experience." Pharmacol Biochem Behav. 1995;52:195-203.

Ernst, Armin and Joseph D. Zibrak. "Carbon Monoxide Poisoning." N Engl J Med 1998;339(22):1603-8.

Hardy, Kevin R. and Stephen R. Thom. "Pathophysiology and Treatment of Carbon Monoxide Poisoning." J Toxicol Clin Toxicol 1994;32:613-29.

Zagami, Alessandro S., A. Keith Lethlean, and Ross Mellick. "Delayed neurological deterioration following carbon monoxide poisoning: MRI findings." J Neurol 1993;240:113-6.

Hausberg, Martin and Virend K. Somers. "Neural Circulatory Responses to Carbon Monoxide in Healthy Humans." Hypertension 1997;29:1114-8.

Ilano, Aaron L. and Thomas A. Raffin. "Management of Carbon Monoxide Poisoning." Chest 1990;97:165-9.

Seger, Donna and Larry Welch. "Carbon Monoxide Controversies: Neuropsychologic Testing, Mechanism of Toxicity, and Hyperbaric Oxygen." Ann Emerg Med 1994;24:242-8.

### **Toxicology Associations**

American Conference of Governmental and Industrial Hygienists  
American Board of Applied Toxicology  
American Academy of Clinical Toxicology (<http://www.clintox.org>)  
American College of Medical Toxicology (<http://www.acmt.net/DEFAULT.HTM>)  
American College of Toxicology (<http://actox.org>)  
Society of Toxicology ([www.toxicology.org](http://www.toxicology.org))

### **Medical: Societies & Associations**

American Medical Association (<http://www.ama-assn.org>)  
American Psychological Association (<http://www.apa.org>)  
National Academy of Neuropsychology (<http://nanonline.org>)  
American Board of Professional Psychology (<http://www.abpp.org/home.htm>)  
American Academy of Neurology (<http://www.aan.com>)

### **Industrial Hygiene**

American Industrial Hygiene Association (<http://www.aiha.org>)

### **Federal Government Agencies**

Agency for Toxic Substances and Disease Registry (<http://www.atsdr.cdc.gov>)

Centers for Disease Control & Prevention (CDC) (<http://www.cdc.gov>)

Environmental Protection Agency (<http://www.epa.gov>)

Occupational Health & Safety Administration (<http://www.osha.gov>)

Food & Drug Administration (<http://www.fda.gov>)

National Institute of Occupational Safety & Health (<http://www.noish.gov>)

**Private companies** within relevant industries, such as HVAC manufacturers. They may have information about chemicals and applicable standards that they deal with regularly.