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CASE REPORT

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Diesel Fumes *Do* Kill: A Case of Fatal Carbon Monoxide Poisoning Directly Attributed to Diesel Fuel Exhaust with a 10-year Retrospective Case and Literature Review*

ABSTRACT: While it is known that diesel fuel combustion engines produce much lower concentrations of carbon monoxide (CO) than gasoline engines, these emissions could certainly generate lethal ambient concentrations given a sufficient amount of time in an enclosed space and under suitable environmental conditions. The authors report a case of CO poisoning which was initially referred for autopsy as a presumed natural death of a truck driver found in the secure cab of a running diesel tractor trailer truck. Completion of the preliminary investigation ascribed death to complications of ischemic heart disease (IHD), pending toxicological analysis that included quantification of CO. When the toxicology results showed lethal blood COHbg, the cause of death was re-certified as CO intoxication secondary to inhalation of (diesel) vehicular exhaust fumes. Because of the unique source of fatal CO intoxication in this case, the contributory IHD and the possible contaminants in the putrefied blood, a 10-year retrospective review was conducted on all nonfire related CO deaths autopsied (n = 94) at the Office of the Chief Medical Examiner in Louisville, KY from 1994 to 2003. For validation of the COHbg detection method used by the Kentucky Office of Forensic Toxicology (KYOFT), blood samples from these cases along with controls were submitted to three laboratories using various analytical methods yielding no statistically significant differences. Lastly, an extensive literature review produced no scientifically reported cases of fatal CO poisoning attributed to diesel fuel exhaust.

KEYWORDS: forensic science, toxicology, postmortem decomposition, carbon monoxide intoxication, diesel fuel exhaust

Often called the "silent killer," carbon monoxide (CO) is the most common fatal poisoning in the U.S.A., claiming 1000–3500 lives every year. Although suicides constitute the majority of fatalities in nonfire CO poisoning, accidents account for *c.* 25–30% of annual deaths (1,2). CO is produced by the incomplete combustion of organic material and high environmental concentrations can rapidly accumulate under many different scenarios. The most common sources of fatal CO intoxication are from inhaled fumes in fires or motor vehicle emissions (2). Typical accidental poisonings usually involve unsuspected increases in CO levels in an enclosed environment, which can include secured motor vehicles, vehicles in open spaces, residential or parking garages, car washes, homes, and camping tents (2–8). CO poisoning has been notoriously attributed to the inhalation of fumes emitted from gasoline powered motor vehicular exhaust when personal-use automobiles were involved, even when the engine possessed a catalytic converter (1,2). Although currently in the U.S.A. a small fraction of personal automobiles have a diesel engine, tractor trailer trucks utilize diesel fuel because of its efficiency. Accounts of CO exposure from engine exhausts of diesel powered boat motors and generators on open water conditions or within their secured cabins are detailed in recent scientific literature (7–12). Agreeably, death from CO intoxication is not unusual in the medical examiner case-load. However, in the medical examiner arena, lethal CO poisoning from inhalation of diesel fumes from any make or model of on-road vehicle is virtually unheard of and contemporary medical literature does not report it. The purpose of presenting this unique case is to raise the index of suspicion among medicolegal death investigators that diesel fume-related CO fatalities *do* occur and are preventable.

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Case Presentation

A moderately decomposed 52-year-old male truck driver was found in a secure tractor trailer truck. Coroner information detailed that the decedent's diesel truck was parked at an interstate highway truck stop. The engine was running and the heat was on in the cab. The decedent lay prone and partially covered with a blanket on the floor between the sleeper and driver compartments of the cab. He had worked as a truck driver for several years. According to the widow of the decedent, he had been feeling ill with flu-like symptoms during the trip in the days prior and up to the time of his death and had purchased over-the-counter cold medication. Generic diphenhydramine packaging was found in the truck cab upon discovery of the body. Neither alcohol or illicit drugs, nor gas-powered cooking implements or combustibles were found in the truck cab. The widow reported that her husband recently had a normal annual physical examination.

At autopsy, external examination of the 6-foot, 165-pound atraumatic body revealed moderate-to-severe decompositional changes evidenced by dark purple-brown discoloration of the face, red discoloration of the chest, and purple discoloration of the right lower

abdomen. Cherry red lividity (classically associated with CO poisoning) was difficult to ascertain because of decomposition. On internal examination, early putrefaction and partial liquefaction of the thoracic and abdominal cavities and all major internal organs were present. Ischemic heart disease (IHD) was illustrated by moderate-to-severe coronary atherosclerosis and hypertensive enlargement. The mid portion of the left anterior descending coronary artery exhibited calcific atherosclerotic plaque formation with 80% stenosis to complete occlusion. The proximal right coronary artery showed 60% stenosis and the proximal portion of the left circumflex coronary artery showed 60–80% stenosis. The enlarged heart (420 *g*) exhibited concentric left ventricular hypertrophy with a left ventricular free-wall thickness of 1.8 cm. There was no evidence of myocardial infarction or scar. Nephrosclerosis was also present.

As a result of the circumstances of the death, blood was submitted for COHbg saturation in addition to the standard alcohol and drug screen. Pending the results of the toxicology studies, the cause of death was attributed to IHD arising from coronary atherosclerosis and hypertension. Blood COHbg resulted in 67% saturation by differential spectrophotometry analyzed by the Kentucky Office of Forensic Toxicology (KYOFT). After verifying the CO value with the supervisor of the KYOFT, the cause and manner of death were amended. The cause of death was certified as CO intoxication from inhalation of motor vehicle exhaust. Because of the significant anatomic findings, IHD was listed as a contributing death factor. The manner of death was deemed accidental. The coroner was immediately notified of these changes with a request for a follow-up investigation of the vehicle.

Discussion and Study-Design

This unique case involves a death from CO poisoning arising from inhaled fumes generated by a diesel engine truck. While diesel engines produce exhaust containing CO, the concentration in parts per million (ppm) is lower than what is produced in gasoline engines. An extensive search of both medical and engineering literature failed to produce any specific case report or incidence of CO poisoning directly attributable to inhalation of diesel fuel exhaust. While there are innumerable case reports of death by CO poisoning from exposure to motor vehicle exhaust, the type of vehicle at fault is far less frequently reported. Medical death investigators and researchers generally have not distinguished between gasoline and diesel vehicles in these particular cases. This report details a case of unintentional death from CO poisoning directly attributable to *diesel fuel* exhaust.

Carbon Monoxide Pathogenicity

As the most common fatal poisoning in the U.S.A., the major sources of CO intoxication are from inhaled fumes in fires or motor vehicle emissions. Fire-related CO poisoning is the most common overall cause of fatalities and the most frequent cause of immediate fire-related death. Suicides constitute the majority of fatalities outside of fire-related deaths—of the *c.* 2700 CO fatalities per year, about 2000 are suicides (1,2). Almost all suicides involve the inhalation of motor vehicle exhaust (1,2).

Carbon monoxide is produced by the incomplete combustion of organic material, the most commonly implicated source being petrol-based fuels (2). High environmental concentrations of CO rapidly accumulate during incomplete combustion in a contained area. Common sources of CO poisoning include the operation of motor vehicles in enclosed or semi-enclosed areas, malfunctioning residential heating systems, and improperly vented combustion appliances (2,13). The circumstances of CO intoxication often involve an unsuspected increase of ambient CO in an enclosed environment. Victims often are unaware that their activity or environment placed them at a risk for CO poisoning. Improperly vented gas water heaters or kerosene heaters are responsible for the majority of accidental CO exposures.

The adverse health effects and toxicity of CO are related primarily to the development of tissue hypoxia. CO accumulates rapidly in the air, lungs, and blood. It binds reversibly to hemoglobin (Hbg) with 200–250 times the affinity of oxygen (O_2), reducing the O_2-carrying capacity and impairing the release of O_2 to the brain, heart, and other body tissues (14–17). The mechanism of death is cardiac dysrhythmia induced by hypoxia. Acute effects correlate roughly with COHbg concentrations in whole blood. Toxicity increases with length of CO exposure, decreased metabolic activity, and the presence of underlying cardiac or cerebrovascular disease. The organs with the highest O_2 requirement, the heart and brain, are most susceptible. Mild-to-moderate CO exposure causes flu-like symptoms, such as headache, nausea, fatigue, and general malaise. CO may also be associated with delayed fatalities, occurring days after toxic exposure when blood COHbg has decreased to negligible levels (18,19). Table 1 summarizes the toxic effects of CO as related to blood %COHbg in a healthy 70 kg adult.

Specific CO toxicity predominantly results from the environmental CO concentration; however, the duration of exposure is extremely important. Environmental CO distribution in a confined space also depends upon additional factors, such as the size of the space (volume), CO rate change, and ventilation rate—all of which independently influence the speed of blood distribution. The effects of CO are more prominent as heat and humidity are increased. In sum, measured %COHbg in the blood at equilibrium depends on the atmospheric CO concentration, the duration of exposure, and the respiratory minute ventilation (17,20). The usual atmospheric CO has a concentration <0.001% or <10 ppm (17). Even very low atmospheric CO concentrations will rapidly combine with Hbg until equilibrium is reached. In the absence of other extrinsic factors, an increase in minute ventilation, such as that associated with exercise, shortens the length of time necessary for CO to reach the equilibrium concentration. Atmospheric concentrations of 50, 100, 200, 500 ppm produce approximate equilibrium COHbg concentrations of 8%, 16%, 30%, and 45%. When higher concentrations of CO, such as 0.05% (500 ppm), are inspired, the equilibrium concentration of COHbg increases to the highly toxic level of 45% within a matter of a couple of hours (20). Exposure to CO concentrations >50,000 ppm can cause cardiac arrhythmia and death prior to significant elevation of COHbg (21). CO is produced in abundance in house fires, where environmental CO levels may reach as high as 100,000 ppm, or 10% ambient air concentration. Generally, in healthy persons 30–40% COHbg is considered lethal, but COHbg saturation levels of >50% have been reported in the blood taken at autopsy. In individuals at the extremes of age and those with debilitating diseases,

TABLE 1—*% Carboxyhemoglobin and systemic effects.*

%COHbg	Effects
<10	Mild judgment impairment
10–20	Exertional dyspnea, lightheadedness
20–30	Severe headache
30–40	Nausea, dizziness, muscle weakness
40–50	Syncope, death
≥60	Death

such as IHD or chronic respiratory illnesses, lethal COHbg saturation levels may drop to 20–30% (2,20).

Recommended exposure limits to CO are set by the Occupational Safety and Health Administration (OSHA) and the National Institute for Occupational Safety and Health (NIOSH). The current OSHA standard for CO allows exposure to a time-weighted average of 50 ppm over an 8-h workday, with an upper limit of 200 ppm over a 15 min period (22). The NIOSH recommended exposure limit for CO is 35 ppm (0.035 mL of CO/L of air) as an 8-h time weighted average (23). NIOSH ceiling limits for CO exposure does not exceed 200 ppm at any time. A CO concentration of 1200 ppm (0.12%) is designated as the exposure amount causing immediate danger to life and health (24). High concentrations of inspired CO lead to coma and subsequent death within minutes. A CO content of 2000 ppm (0.2%) in inspiratory air will cause unconsciousness in <1 h. Even with a normal O_2 content of 21%, an increased CO content of 1000 ppm (0.1%) in inspired air causes 50–60% of the Hbg molecule to be occupied within <2 h of exposure. In idling motor vehicles emitting exhaust laden with 7–12% CO concentrations, the atmospheric CO level in a garage could reach 1% (10,000 ppm), enough to cause human death in 10 min. Nonlethal CO poisonings are treated with O_2 therapy with fairly rapid reversal of signs and symptoms and a correspondent measured decreased COHbg % saturation in blood specimens. In more severe cases of CO intoxication, hyperbaric O_2 is utilized but even rapid treatment and early intervention cannot be expected to fully prevent cognitive deficits resulting from toxic neuronal injury (16,25).

Tripartite Case Study

Four questions arose as investigators searched for the determination of the specific source of CO in this decedent's intoxication.

1 Could the COHbg have reached the high concentration of 67% in light of the severity of the victim's heart disease?
2 Can CO intoxication present without the classically described cutaneous cherry red lividity?
3 Was the KYOFT COHbg detection methodology scientifically accurate and reliable given the victim's state of decomposition?
4 Does diesel fuel exhaust contain lethal amounts of CO and could diesel exhaust emitted through the carriage of the tractor trailer truck be implicated as the CO source in this case?

To answer these questions, we conducted a three-part study: (i) a 10-year retrospective chart review from the Office of the Chief Medical Examiner in Louisville KY (OCME) of all nonfire related CO deaths was performed; (ii) for confirmation of the validity of the COHbg detection method used by the KYOFT, blood samples from cases representing varying degrees of decomposition with controls were submitted to two independent commercial laboratories and one federal laboratory; and (iii) finally, an exhaustive literature search and personal communication yielded no reported cases of fatal CO poisoning, accidental or suicidal, directly attributed to diesel fuel exhaust from a motor vehicle or boat. Considerable information, however, was available regarding the CO content of diesel exhaust and its potential lethality in reference to animal models.

Retrospective Case Study

Using a computerized database system, we conducted a 10-year retrospective case review of all nonfire-related CO deaths autopsied at the OCME from 1994 to 2003. The review compared the present case to autopsy and toxicological findings with scene investigation of 94 postmortem cases. Specific autopsy findings gleaned from the study were severity of IHD, postmortem decomposition, and the presence of cutaneous cherry red skin discoloration. Table 2 provides a summary of the pertinent trends examined for comparison to our case.

TABLE 2—*Autopsy findings in nonfire CO intoxication autopsy cases.*

Total cases reviewed: 94
Average (COHb): 54.8%
Total cases with any heart disease: 50
Average (COHb): 52.6%
Total cases with moderate-to-severe CAD: 16
Average (COHb): 56.0%
Total cases with no decomposition: 71
Average (COHb): 56.6%
Total cases with decomposition: 23
Average (COHb): 49.4%
Total cases with no cherry red lividity: 28
30% of all reviewed cases did not show classic cherry red discoloration

Carbon monoxide has well-documented ill-effects on the cardiovascular system, particularly in people with known coronary artery disease (CAD). Exposure to lower concentrations of ambient CO hasten angina and myocardial ischemia, as noted by electrocardiographic ST wave depression in these susceptible patients (20,26). At autopsy, the victim in our study case presented with critical CAD, moderate decomposition and discolored skin, and viscera. Postmortem toxicology yielded COHbg saturation of 67%. In comparison, the overall average of %COHbg in the 94 cases was 54.8%. With regard to cases that showed any degree of heart disease (n = 50), the average %COHbg was 52.6%, lower than that of the victim in this case, but still well above a lethal level typically reported in the literature. In the cases exhibiting moderate-to-severe CAD (n = 16), the average COHbg was 56.0%. This saturation was reported for individuals with significant underlying heart disease, but was still lower than the %COHbg of the victim. Based on these numbers we were able to conclude that %COHbg can reach very high levels, even in victims with significant IHD.

Fatal CO intoxication has been described in persons who did not exhibit the classical cherry red cutaneous lividity (27–29). Although the presence of cherry red lividity in these victims aids in postulating a potential cause of death, it is not always a reliable characteristic feature. Twenty-eight cases in our study pool, representing *c.* 30% of the total cases (n = 94) reviewed, failed to show classic cherry red lividity at autopsy. In the victims, who exhibited neither decompositional changes nor cherry red lividity (n = 13), COHbg ranged from 29% to 71.5%. Classic cherry red lividity was absent in decomposed cases secondary to the literal rainbow of cutaneous putrefactive discoloration. From the data from our study pool, we conclude that CO intoxication often occurs without cherry red lividity, in part from decompositional color alterations manifested at autopsy. After scrutinizing OCME records and comparing similar cases to the present case, we conclude that the latter is not an atypical CO poisoning. But the possibility that there may be false elevation of the CO concentration in decomposed blood needed to be resolved.

Decomposed Blood Specimens Warrant Comparative Toxicology Analyses

Very small amounts of endogenous CO are produced as a byproduct of Hbg, myoglobin, and other heme protein metabolism, which may cause erroneously, however negligible, elevated COHbg

results in postmortem blood specimens (30–33). Dominguez et al. (31) found that COHbg is not significantly altered by decomposition in dogs. Methemoglobin (MetHbg), a Hbg breakdown product, may be elevated in the blood of fire death and exhaust fume exposure victims. Heat denaturation of the blood is considered to be the cause of increased MetHbg formation in fire victims, and nitrogen oxides increase MetHbg in the blood of victims succumbing to exhaust fumes (32,33). Heating of postmortem blood will also cause small elevations of MetHbg (33). Hbg degradation products, in particular MetHbg, may increase with decomposition and interfere with COHbg saturation determination by spectrophotometric techniques caused by increased background signals or "noise" (33). Hence, the validity of COHbg measurements in decompositional postmortem blood has been questioned. The prospect that the blood COHbg could be falsely elevated in the presented case raised concerns as it demonstrated elevated % saturation and because the specimen was taken from a decomposed man found in a heated environment who died from vehicular exhaust.

The concern of spurious laboratory results in any potential medicolegal case prompted a three-tiered validation study of the KYOFT laboratory method used to quantitate COHbg saturation in blood. Analytically, in a decomposed specimen, increased levels of blood MetHbg must be eliminated to confirm the elevated COHbg measurements for proper cause of death certification. The KYOFT laboratory utilizes a reducing agent for the neutralization of Hbg breakdown products in postmortem blood CO testing to yield valid COHbg results. To validate the KYOFT primary method, blood samples (n = 14) were taken from casework CO fatalities and negative controls. Samples tested included nonputrefied and putrefied specimens. All samples were first tested by the KYOFT primary method. This method involves differential spectrophotometric determination of COHbg in the blood using NH_4OH as a reducing agent (Cleveland Method) to neutralize Hbg breakdown products that may falsely elevate the measured COHbg. The reducing agent helps with interpretation of compounds that are manufactured and found within the blood as part of decomposition, i.e., MetHbg, sulfhemoglobin, and sulfmethemoglobin (34–36).

The results of the primary KYOFT CO analysis were then compared to the three different laboratory methods in all of the specimens as follows: a similar, scientifically acceptable COHbg detection method as the KYOFT analysis using a different reducing agent, sodium dithionite; then employing a combined method with both NH_4OH and sodium dithionite as reducing agents; and finally utilizing the CO-oximeter method in which whole blood is analyzed spectrophotometrically. In brief summary of our validation study, the KYOFT laboratory procedure for COHbg detection is both comparable to and perhaps superior to the widely used and commercially available methods that were tested in our study for the analysis of both nonputrefied and putrefied blood samples.

Comparison of Gasoline and Diesel Fuel Combustion

Vital to the present case is the diesel source contributing to the CO intoxication, which is not traditionally recognized as containing lethal amounts of the gas. At the time this study was completed, there were no known reported cases of fatal CO poisoning directly attributed to diesel fuel exhaust from land- or water-based motor vehicles in the U.S. forensic literature. Numerous reports of CO poisoning from boats, especially house boats, have been documented and some boat engines and generators use diesel fuel (7–12). However, diesel fumes have not been directly implicated in any of these. To detail effects of diesel fumes on animal and human morbidity, three scientific investigations have been published (37–39). Perhaps most infamously, diesel fumes were reported as a method of extermination in Nazi concentration camps during WWII (40,41).

Carbon monoxide concentrations produced by the combustion of gasoline and diesel fuels varies. In general, gasoline engines without a catalytic converter produce between 1% and 10% CO in exhaust gases. As part of modern gasoline engines, catalytic fuel converters produce lower CO concentrations at <0.1% compared to their nonequipped counterparts possessing CO outputs of 1–10% (29,42). Furnishing automobiles with these devices has led to a decline in CO-related mortality in the U.S. over the past three decades (42). The combination of the low CO output of the engine resulting in a lower blood CO and O_2 deprivation may be responsible for the mechanism of death in cases involving modern gasoline-fueled vehicles (43). Standard engines not equipped with catalytic converters include boat motors and gas-powered generators. Diesel engines generally produce 0.1% CO in exhaust gases, but this level is higher under certain well-defined engine operating conditions (29). According to some experts, the overall fleet of heavy-duty diesel-fueled trucks produces well over 100 ppm CO, or 0.1%, in their exhaust gases under normal operating conditions. Certain factors or malfunctions, which can occur in the everyday operation of heavy-duty diesel engines, may increase engine workload or decrease engine combustion efficiency and increase CO output from diesel engine exhaust. These factors include the following: abrupt changes in power setting; abrupt load changes; suboptimal injection timing, fuel aerosolization, fuel distribution, or combustion chamber shape; low combustion temperature; low operating revolutions per minute; blocked air intake; large, heavy-duty engine size versus light-duty size; and improper engine maintenance. Concentrations of 1000–5000 ppm CO in undiluted diesel exhaust gases are common, and levels up to 60,000 ppm or higher, are possible (44). Of course, at these high levels, death would be expected within a short period of time, if CO were allowed to accumulate within an enclosed space, such as a tractor trailer cab.

During the investigation of this case, CO output tests were conducted on an identical make and model of the tractor trailer implicated in the victim's death. The testing was performed on the truck's exhaust system under two different running conditions. Three separate tests were performed with resultant CO output measurements as follows:

1. CO output 367 ppm when engine idling.
2. CO output 360 ppm when engine idling.
3. CO output 485 ppm with engine at 1500 rpm.

When an individual is exposed to the ambient CO levels between 360 and 485 ppm, CO may accumulate very rapidly in the blood to reach highly lethal concentrations, especially if it occurs within in a small enclosed space. From these data, it appears that diesel fuel combustion *will* produce lethal amounts of CO under the right circumstances. Further examination of available data on diesel-powered engines, when tested on animals and humans, support these toxic effects (37–39).

Diesel Fuel CO Investigations: Three Scientific Reports

Three published studies have examined the effects of diesel fume exposure. The first study, which was conducted in 1956 by Pattle et al. (37), was published in the British Journal of Industrial Medicine. The study examined the toxicity of diesel fumes in laboratory animals under different diesel engine operating conditions. Animals, including mice, guinea pigs, and rabbits, were placed in exposure chambers measuring 10 m^3 and diesel engine exhaust was piped in

through two metal pipes. Four different study groups corresponding to four different engine operating conditions were exposed for periods of 5 h. During the exposure periods, the animals were monitored for symptoms of noxious gas irritation and intoxication, and mortality was calculated following the exposures. The running conditions for each group represented successive addition of engine load and increases in engine wear and air intake obstruction. Successive running conditions were designed to simulate causes of decreased engine combustion efficiency similar to that which may be encountered in on-road vehicles. As engine load increased and air intake became obstructed, lethality caused by CO intoxication approached 100%. Lacrimation was observed in all study groups, and signs of intense pain were observed in animals from the last study group. In this group, all animals died from CO intoxication, with %COHb averaging 60% in mice and 50% in rabbits. The engine in the last study group was run with simulated load and partially obstructed air intake and produced an exposure chamber CO concentration of 1700 ppm. Although this study was conducted in rodents, the lethality of diesel fumes is clearly demonstrated and potential effects on humans may be easily extrapolated from the data.

Rudell et al. (38) conducted a study in 1993 examining the effects of diesel fume exposure in human beings. The study was designed to develop an experimental setup in which the acute effects of diesel exhaust exposure on humans could be studied. Healthy nonsmoking volunteers were seated in an exposure chamber measuring *c.* 21 m^3, and diluted diesel exhaust was piped in from an idling vehicle parked outside the chamber. The subjects' acute symptoms were reported and lung function measured for any changes caused by the exposure. All subjects reported an unpleasant smell, eye irritation, and nasal irritation. Some subjects reported headaches, nausea, dizziness, fatigue, throat irritation, and coughing. There were no measurable effects on lung function. The chief symptoms were attributed to the presence of nitrogen dioxide, a noxious gas produced by diesel fuel combustion. The median CO steady state concentration in the exposure chamber was 27 ppm, and COHbg % saturations were *not* measured in the study subjects (38). Obviously, lethality was not part of the research design.

Nightingale et al. (39) measured the cardiopulmonary response to diesel exhaust inhalation. Ten healthy nonsmoking volunteers were placed in exposure chambers and subjected to a controlled concentration of diesel exhaust particulates for 2 h. No changes in cardiovascular parameters or pulmonary function were observed. Exhaled CO concentrations were increased by 50% following exposure to diesel as compared with air exposure (air = 2.9 ± 0.2 ppm, diesel = 4.4 + 0.3 ppm; $p < 0.0005$). The diesel particulate matter was shown to cause an inflammatory airway response as evidenced by increased sputum neutrophil activity after exposure (39). Blood COHbg % saturation was not measured and the subjects were not evaluated at lethal exposure concentration. This study shows that diesel exhaust is capable of significantly elevating exhaled CO concentrations.

Conclusion

This study comprises a comprehensive investigation into the mechanism of death by CO poisoning in a motor vehicle with a diesel fuel-powered motor: (i) a 10-year retrospective evaluation of deaths at the KY OCME attributable to CO poisoning; (ii) internal and external toxicology validation studies of current analytical methodologies to quantitate CO; and (iii) an intensive literature review of CO-related morbidity or mortality of animals and humans from diesel fuel-powered sources. On the basis of this analysis, we have reached an irrefutable conclusion about the cause and manner of death in the presented case.

The victim in this case represents a typical case of accidental fatal CO poisoning. With a high degree of suspicion in this decomposed individual with significant IHD, unmasking the CO poisoning is interesting, but not unheard of. The cases reviewed were chosen for point-by-point comparisons of IHD, postmortem decomposition with description of lividity patterns, and circumstances of the deaths. The toxicology study scientifically validates the KYOFT COHbg detection method use in this case. The available literature concerning the CO content of diesel fuel recognizes its potential lethality under certain circumstances. History demonstrates the lethality of diesel exhaust when used as a method of extermination.

Reconstruction of the possible course of events leading up to the death of this truck driver are as follows: as he felt ill with "flu-like" symptoms a few days leading up to his death, the victim may have been exposed to a constant low-level exposure of CO through an exhaust leak. The exposure was interrupted by opening the window and door at various times of the day. After prolonged CO exposure in an enclosed truck cab, feeling ill or fatigued, he stopped at a rest area. He likely experienced chills, evidenced by a blanket covering him and the cabin heater running at the time of discovery, during the month of June. Once the cab was sealed with the engine idling, the CO rapidly accumulated and poisoned him. Even though the CO concentration was extremely high in his blood and the proximate cause of death was CO intoxication, IHD was included as a significant anatomic factor contributing to his death because it may have disabled him from getting out of the noxious environment in which he was found.

This accidental death represents an under-recognized and under-reported incidence of diesel-derived CO intoxication. We encourage law enforcement, crime scene and forensic investigators, medical and legal professionals to consistently distinguish between gasoline and diesel fuel types when a suspected CO poisoning occurs in order to provide more accurate information about this mechanism. Additionally, in instances of suspected CO poisoning such as this, it is imperative that a stat CO be performed via collaborative efforts through the Medical Examiner's office and the supporting toxicology laboratory. In instances of a faulty device or motor vehicle, the public health benefits of rapidly making this diagnosis before another person is exposed to the danger, will save lives.

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