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CARBON MONOXIDE IN-FLIGHT INCAPACITATION:
AN OCCASIONAL TOXIC PROBLEM IN AVIATION

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16. Abstract Results from the toxicological study of samples from 4,072 pilots killed in general aviation accidents have revealed that carbon monoxide has been the cause of incapacitation in 21 (0.5 percent) of the cases. Two cases are presented that are typical of accidents caused by incapacitation from carbon monoxide, in that no cause was determined until after toxicological examination of the victims was made. Since no suitable system is available to warn pilots of elevated carbon monoxide levels in the cabin, education of pilots should be undertaken to make them aware of the hazards of exposure to and symptoms produced by this highly toxic gas.					
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INTRODUCTION.

Carbon monoxide is the most abundant air pollutant in the lower atmosphere. The compound is produced from the incomplete combustion of organic materials. Common sources of the gas are exhaust from internal combustion engines, cigarette smoke, and a myriad of industrial processes. In 1974, Stewart (1) reported that urban nonsmokers averaged 1-2 percent carboxyhemoglobin and smokers 5-6 percent. Seppänen (2) has reported that levels of carboxyhemoglobin below 12 percent saturation had no effect on psychomotor performance but did have a detrimental effect on visual perception. Symptoms of carbon monoxide poisoning may vary depending on whether the exposure is to a sudden high concentration or to a prolonged lower amount. At a blood saturation of less than 10 percent the individual usually shows no recognizable symptoms; below 20 percent saturation, headaches, and shortness of breath on exertion are common; nausea, sleepiness, severe headache, and blurred vision usually occur between 20-40 percent blood saturation. Crane (3) has reported that incapacitation occurs at a blood saturation of 46.5 percent, and a lethal level is reached at 67.5 percent saturation. Symptoms of carbon monoxide poisoning, which are a result of decreased oxygen-carrying capacity, may occur at lower levels of blood saturation if the subject is in an atmosphere of decreased oxygen tension, as will occur in flying if supplemental oxygen is not used.

For over 14 years the Federal Aviation Administration (FAA) Civil Aeromedical Institute (CAMI) has operated a forensic toxicology laboratory analyzing blood and other specimens from air carrier and general aviation accident victims. During that period, specimens have been received from over 4,300 fatal accidents and 7,000 victims. Findings from a number of cases have been previously reported (4). In a number of accidents, no significant traumatic injuries were demonstrated at autopsy but the cause of death was inhalation of toxic gases resulting from postcrash fires. Such victims usually have had significantly elevated carboxyhemoglobin levels and many also have had an elevated blood hydrogen cyanide concentration.

Here we report on cases involving general aviation aircraft in which no evidence of fire was found and carbon monoxide was the only toxic substance present in the specimens from the victims. These data are compared to the data from all cases in which a mixture of toxic gases has been present in accident victims and to information found at autopsy.

METHODS.

Carbon monoxide saturation is determined on all blood samples. When blood is not available, fluid containing sufficient hemoglobin can usually be obtained from tissue by freezing and thawing or by the gentle application

of pressure. Hemoglobin is determined by the standard cyanmethemoglobin method or by an alkaline hematin method. Samples containing more than 5 g percent of hemoglobin are first analyzed by liberating the CO, diffusing it into a palladium chloride solution and measuring it colorimetrically (5). For confirmation, or when greater sensitivity and specificity are needed, carboxyhemoglobin content is determined by a gas chromatographic method utilizing a flame ionization detector after the catalytic conversion of carbon monoxide to methane (6). The instrument and its operating conditions are as follows: Varian Aerograph 1400; columns (1/8 in x 6 ft) Carbosphere, 80-100 mesh; flame ionization detector; carrier gas, hydrogen, 30 mL/min; injection port 200°C, column 80°C, followed by a nickel-on-firebrick column (1/8 in x 1 ft) maintained at 370°C; and a Hewlett Packard 3390A reporting integrator. Blood and bloody fluids are diluted 1 to 1 with distilled water. This system may be used to determine as little as 3 percent saturation in a sample containing 2 g percent of hemoglobin. Samples containing more than 10 percent carboxyhemoglobin are routinely analyzed for cyanide. Hydrogen cyanide is diffused in a Conway diffusion cell and trapped in sodium hydroxide. The trapped cyanide is then measured colorimetrically (5).

FINDINGS.

Carboxyhemoglobin levels above 10 percent have been found in 109 pilots from a series of 4,072 fatal general aviation accidents. In 21 of these cases it was established that postcrash fire could not have contributed to the elevated carboxyhemoglobin levels and that the CO must have been acquired prior to, or during, flight. The following two cases illustrate this occasional cause of fatal aircraft accidents. In both cases the source of the cockpit air contamination was found.

Case 1. A Piper Cherokee departed Savannah, Georgia, at 2150 on a rainy September night with three persons aboard a VFR flight. The flight continued for 1 hour and 20 minutes at an altitude of 2,500 feet. During the last 15 minutes of the flight the pilot was in contact with the Macon, Georgia, radar approach control and was vectored around thunderstorms that were in the area. The last communication with the flight occurred 4 minutes before radar contact was lost. The wreckage was found shortly after the accident and there were no survivors. The aircraft impacted in a near vertical attitude as evidenced by damage to only one tall pine tree from near the top to the ground. The engine and propeller were almost completely buried at the base of the tree. Both front seat occupants were partially decapitated. Carboxyhemoglobin levels in two victims were 43 percent and 47 percent. Samples were not obtained on the third occupant. After the laboratory analysis revealed high CO levels with no hydrogen cyanide, and the autopsy evidence indicated that the occupants could not have survived the impact to acquire CO from the postcrash fire, investigators carefully examined the cabin heating system. They found a crack in the exhaust muffler. Analysis of scrapings from the inner wall of the heater duct confirmed the presence of

fuel ignition by-products, supporting the contention that the crack existed prior to the crash. The crack had allowed exhaust gas to enter the aircraft cabin through the cabin air heater system. The accident occurred when the CO in the occupants reached incapacitating levels.

Case 2. The pilot, the only occupant of a Piper TriPacer, departed a local airport at 1700 on a cold December afternoon for the purpose of practicing touch-and-go procedures. The flight before the fatal accident was of 30-minutes' duration. The crash occurred in a wooded area adjacent to a large open field. The plane descended into the trees at a shallow angle paralleling the border of the open field. No autopsy was performed on the pilot but blood was submitted for toxicological analysis. A carboxyhemoglobin level of 23 percent was found. The pilot had been thrown free of the burned aircraft and gross injuries suggested that he died immediately after the crash. The finding of an elevated CO saturation prompted close inspection of the exhaust and heater system. Investigators found a cracked exhaust muffler and staining of the heat exchange shroud by the exhaust gases (Figure 1). The



Figure 1. Muffler from a single engine aircraft. Note the split in the muffler and the discoloration caused by exhaust gases as they contacted the wall of the cabin heat exchanger shroud. The pilot was incapacitated in flight by carbon monoxide and killed in the crash that followed.

rent in the muffler was along the seam where the muffler had been welded in manufacture. A backfire in the exhaust system probably caused the muffler to split. The elevated carboxyhemoglobin level, proved by the laboratory, pointed accident investigators in the right direction, resulting in a satisfactory explanation of the cause of the accident.

DISCUSSION.

The Occupational Safety and Health Administration (OSHA) has succinctly summarized the toxicology of carbon monoxide as follows: (8)

"Carbon monoxide (CO) gas causes tissue hypoxia by preventing the blood from carrying sufficient oxygen. CO combines reversibly with the oxygen-carrying sites on the hemoglobin molecule with an affinity ranging from 210 to 240 times greater than that of oxygen; the carboxyhemoglobin thus formed is unavailable to carry oxygen. In addition, carboxyhemoglobin interferes with the release of oxygen carried by unaltered hemoglobin. With exposure to high concentrations such as 4000 ppm and above, transient weakness and dizziness may be the only premonitory warnings before coma supervenes; the most common early aftermath of severe intoxication is cerebral edema. Exposure to concentrations of 500 to 1000 ppm causes the development of headache, tachypnea (rapid breathing), nausea, weakness, dizziness, mental confusion and in some instances, hallucinations, and may result in brain damage. The affected person is commonly cyanotic. Concentrations as low as 50 ppm result in blood COHb levels up to 10% in an 8-hour day. This greatly increases the risk of angina pectoris and coronary infarctions by decreasing the oxygen supply in the blood and also in the myoglobin of the heart muscle. These effects are aggravated by heavy work, high ambient temperatures, and high altitudes. Pregnant women are especially susceptible to the effects of increased CO levels. Smoking also increases the risk: cigarette smoke contains 4% CO, which results in 5.9% COHb if a pack a day is smoked. The blood of persons not exposed to CO contains about 1% CO, probably as a result of normal heme metabolism. The diagnosis of CO intoxication depends primarily on the demonstration of significantly increased carboxyhemoglobin in the blood. Levels over 60% are usually fatal; 40% is associated with collapse and syncope; above 25% there may be electrocardiographic evidence of a depression of the S-T segment; between 15% and 25% there may be headache and nausea. The reaction to a given blood level of carboxyhemoglobin is extremely variable: some persons may be in a coma with a carboxyhemoglobin level of 38% while others may maintain an apparently clear sensorium with levels as high as 55%. The blood of cigarette smokers contains 3 to 10% carboxyhemoglobin, and nonexposed persons have an average level of 1%, probably as a result of normal heme metabolism. Several investigators have suggested that the results of behavioral tests such as time discrimination, visual vigilance, choice response tests, visual evoked responses, and visual discrimination thresholds may be altered at levels of carboxyhemoglobin below 5%."

That exposure to carbon monoxide in the cabins of aircraft can lead to incapacitation has been recognized for many years. In 1964, Slusher (7) reported that although engine exhaust system failures in general aviation aircraft were not frequent, in 70 percent of the failures a carbon monoxide hazard was created in the cabin atmosphere. Exhaust gases from internal combustion engines may contain as much as 5 to 7 percent carbon monoxide. After mixing with other air, a much lower concentration would be found in the cabin air.

The FAA limits levels of carbon monoxide in cabin air to 0.005 percent or 50 parts per million (ppm). The OSHA permissible exposure limit is 50 ppm of air averaged over an 8-hour work shift. The National Institute for Occupational Safety and Health (NIOSH) has recommended that the permissible exposure limit be reduced to 35 ppm, averaged over a work shift of up to 10 hours per day, 40 hours per week, with a ceiling of 200 ppm (8). Work done at the FAA's Aviation Toxicology Laboratory by Crane (3) has led to a mathematical expression of the relationship between respiration rate, body weight, carbon monoxide content of inspired air, and time-to-incapacitation (T_i) and time-to-death (T_d). Table 1 was derived from this relationship and shows the time-to-incapacitation for man as a function of carbon monoxide concentration (at near sea level).

Table 1. Carbon Monoxide in Air Versus Time to Incapacitation

Carbon Monoxide Concentration		Time to Incapacitation
%	ppm	minutes
0.05	500	220
0.10	1000	110
0.20	2000	55
0.30	3000	37
0.40	4000	27
0.50	5000	22

As noted in the OSHA summary above, serious decrements in vision, judgment, and motor skills are reached long before total incapacitation (unconsciousness). Such degradations in function occur more quickly at increased altitudes because of the diminishing oxygen transport to peripheral tissues.

A carbon monoxide detector has been developed and is used by the United States Air Force. The apparatus draws air from a preselected location in the aircraft and illuminates a warning light when concentrations of CO exceed 0.005 to 0.007 percent. This instrument has not been used in

general aviation aircraft because of cost. There are available, in general aviation, paper discs, impregnated with a chemical that reacts and changes color in relation to the concentration of carbon monoxide. The color may be compared to a printed color scale. Under strict laboratory conditions this system is capable of detecting hazardous levels of carbon monoxide but when used to monitor cabin air it has proven unreliable because of interference from sunlight, varying humidity, other gases, and cigarette smoke.

The two cases presented are typical of accidents caused by incapacitation from carbon monoxide in that no cause was determined until after toxicological examination of the victims was made. Both defective exhaust systems were found after the field investigator had been advised of the laboratory findings. Although the exhaust system has been found to be the culprit in most of the 21 cases, other routes of entry of carbon monoxide into the cabin have been found in this and other series (7,9).

Not all in-flight exposures to carbon monoxide result in accidents. Four members of a family, on a cross-country flight, developed headaches and the pilot-father became nauseated. The plane was safely landed at Wichita, Kansas, and the occupants were admitted to a hospital. Laboratory analysis revealed elevated carboxyhemoglobin levels. They were observed and released in a few hours. Inspection of the aircraft turned up a leaky manifold and a firewall that was not airtight. In this instance a problem was recognized in time to prevent a tragedy.

Carbon monoxide, though odorless, occurs with other products of combustion in exhaust gases. Most people are able to detect an odor when exposed to exhaust from an internal combustion engine. When any of the symptoms of carbon monoxide poisoning such as headache, drowsiness, nausea, or blurred vision occur in a pilot or passengers, use of the cabin heater, if in operation, should be discontinued. Fresh outside air vents should be opened and, if possible, descent should be made to a lower altitude. The aircraft should be landed as soon as safely possible and inspected for exhaust leaks.

Although less than 0.5 percent of fatal general aviation accidents can be attributed to pilot incapacitation from carbon monoxide, pilots should know of the hazards and symptoms of exposure to carbon monoxide. Only by early recognition can a pilot take the necessary actions to prevent incapacitation and a possible serious crash.

Of course, prevention of exposure to carbon monoxide is the best safety measure; prevention mandates an intact engine exhaust system. At periodic checks exhaust systems should be inspected to assure that exhaust fumes cannot enter the cabin.

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