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Unsuccessful Suicide by Carbon Monoxide: A Secondary Benefit of Emissions Control

DENNIS LANDERS, MD, PhD, *Boise, Idaho*

Emission systems and devices are required on automobile engines to reduce air pollution problems. Catalytic converters have been used on most 1975 and newer automobiles to reduce hydrocarbon and carbon monoxide (CO) emissions to a value that meets the Environmental Protection Agency requirements established for 1975 and 1976. The 1980-1981 Boise, Idaho, study shows that with a functioning catalytic converter either unmeasurable or sublethal quantities of CO appear in automobile exhaust. Thus, emissions control has produced a secondary benefit in reducing the number of suicides by CO poisoning from automobile exhaust fumes.

CARBON MONOXIDE (CO) POISONING has been responsible for many accidental deaths as well as serving as an extremely effective, relatively nondisfiguring, and easily accessible method of suicide. It is responsible for half of the fatal poisonings in the United States.[1] Because of the toxicity of the domestic gas supply in Great Britain, suicidal persons there traditionally have "put their heads in the gas oven" to commit suicide.[2] In the United States the traditional means of intentional carbon monoxide poisoning has been inhalation of car-exhaust fumes.[1,3] It has previously been shown that automobile exhaust will saturate the car's interior or the interior of a small garage in 15 minutes to half an hour with a lethal amount of carbon monoxide.[1] The addition of emission systems on automobile engines, however, has resulted in substantially reducing the amount of CO in auto exhaust and, in turn, has reduced the number of deaths from inhalation of these fumes. We present one such case.

From the Emergency Department, St. Alphonsus Hospital, Boise, Idaho.

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Reprint requests to: Dennis Landers, MD, PhD, St. Alphonsus Hospital, 1055 North Curtis Road, Boise, ID 83706.

Report of a Case

A 35-year-old man was brought to the Emergency Department by paramedics after having been found lying unconscious in a closed garage behind a running 1980 four-cylinder Chevrolet Chevette. The patient had been lying 12 inches from the exhaust pipe, with the car running, for at least three hours before being pulled from the garage. The patient initially had minimal respiratory effort; however, after being pulled from the garage into the cool morning air, his respirations slowly increased. When the paramedics arrived, they found him awake, with vital signs as follows: blood pressure 132/72 mm of mercury, pulse 100 beats per minute and respiratory rate 24 breaths per minute. The skin of his face and his oral mucous membranes were red. He was retching, but no vomitus was noted. The air inside the

ABBREVIATIONS USED IN TEXT

CO=carbon monoxide
HC=hydrocarbons

garage was thick with exhaust fumes, which had caused considerable irritation to the man's eyes and respiratory tract.

When the patient was examined in the Emergency Department, blood pressure was 142/92 mm of mercury, pulse 100 beats per minute and respirations 28 per minute. There was some residual redness of the face, and he was awake but voluntarily not speaking. He was given 100 percent oxygen by face mask, and his lungs were clear to auscultation and percussion.

Laboratory evaluation showed hemoglobin to be 16.8 grams per dl and the leukocyte count 8,100 per cu mm. An SMAC screen gave all normal values. Arterial blood gas determinations gave the following values: pH 7.34, bicarbonate 23 mEq per liter, partial pressure of carbon dioxide 42 and partial pressure of oxygen 116 mm of mercury, and oxygen saturation 72 percent. The carboxyhemoglobin was 36 percent and the level of alcohol in the blood was recorded at 0.09 grams per dl.

After initial evaluation and treatment in the Emergency Department, the patient was transferred to the mental health unit where he remained in hospital for ten days before being dismissed without neurological sequelae.

Discussion

Emission control systems and devices have been installed on automobile engines to reduce products of combustion—specifically, carbon monoxide (CO), hydrocarbons (HC) and oxides of nitrogen. Since 1975 automobile manufacturers have used catalytic converters (Figure 1) to reduce HC and CO emissions to a value that meets the Environmental Protection Agency standards.[4]

The catalytic converter works by providing an additional area to oxidize or burn the HC and CO after it leaves the engine. It is a component that looks much like a muffler and is located between the engine block and the muffler. This system uses platinum and palladium as a catalyst to speed up the change of HC and CO to water and carbon dioxide (Figure 2).

While automobile manufacturers' recommended standards since 1975 have allowed up to 1 percent CO exhaust emission for catalytic-equipped automobiles (Table 1), most of the vehicles studied in the 1980-1981 Idaho Auto Emissions test gave values of less than 0.5 percent CO.[5] As shown in Table 2, many 1980 Chevrolets that were studied, using a SUN EPA Model 75 electronic gas analyzer, did not put out enough carbon monoxide to register on the machine. The analyzer used was a solid-state, nondispersive infrared exhaust analyzer that measures levels of HC and CO in the exhaust of an internal combustion engine. CO levels can be measured between 0 and 10 percent with a cross-sensitivity level of less than 0.1 percent of full scale reading on the CO meter measured against a 15 percent carbon dioxide sample. The complete system is accurate overall to ±2.5 percent of full scale reading and is the standard detector used to measure HC and CO concentrations in exhaust fumes.

The findings from all 33 of the 1980 Chevrolets tested in the 1980-1981 Idaho Auto Emissions

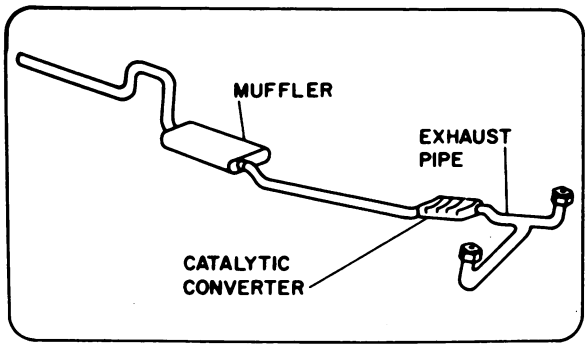

Figure 1.—Single exhaust system with catalytic converter. (From EPA-450, Motor Vehicle Emissions Control Book 7.)

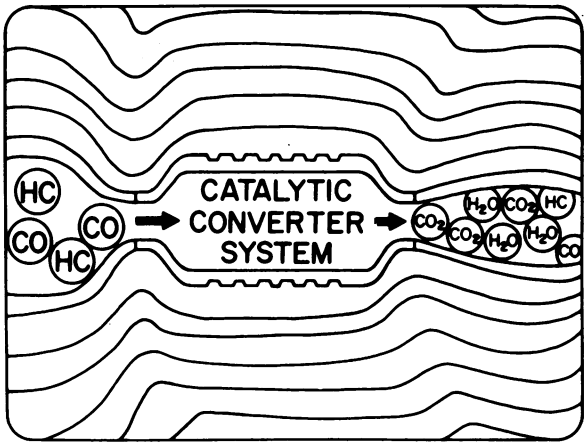

Figure 2.—Catalytic converter system, showing conversion of carbon monoxide (CO) and hydrocarbons (HC) to carbon dioxide (CO_2) and water (H_2O). (From EPA-450, Motor Vehicle Emissions Control Book 7.)

Study are as follows: unmeasurable amounts of CO in 12 of the vehicles; 0-0.1 percent CO in 14 of the vehicles, and greater than 0.1 percent CO in 7 of the vehicles.

The toxic effect of carbon monoxide on animals has probably been known by man since the discovery of fire. However, it remained for Haldane to show that the main toxic property of carbon monoxide is its ability to bind to hemoglobin to a much higher degree than oxygen. Although oxygen combines with hemoglobin ten times more readily than CO, oxygen also dissociates from hemoglobin 2,400 times more rapidly than carbon monoxide. Thus, the affinity of carbon monoxide for hemoglobin is 240 times greater than it is for oxygen.[6-8]

Because carbon monoxide competes with oxygen for binding sites on the hemoglobin, the oxyhemoglobin saturation is decreased. It is important to realize the oxygen tension (Po_2) will remain near normal, but oxygen content (or saturation) will fall sharply as oxygen is replaced by carbon monoxide in the hemoglobin molecule.[6,7] In addition to the reduced oxyhemoglobin saturation, the oxyhemoglobin dissociation curve is shifted to the left so that oxygen cannot be released from the hemoglobin unless a dangerously low oxygen tension exists in the tissues (Figure 3).[8,9]

Carbon monoxide has been shown to have little

TABLE 1.—*Automobile Manufacturers' Exhaust Emissions Standards*

Voluntary inspection standards

Model Year of Vehicle	*CO (percent)*
Pre-1968	8.5
1968-1969	7.0
1970-1974	5.0
1975-1979	3.0

Manufacturers' recommended standards (vehicle tuned to manufacturers' specifications)

Model Year of Vehicle	*CO (percent)*	*HC (ppm)*
1975-1979 Noncatalytic	1.5	150
1975-1979 Catalytic equipped	1.0	75

CO = carbon monoxide; HC = hydrocarbons

TABLE 2.—*Findings of the 1980-1981 Idaho Emissions Study for 1980 Chevrolets**

1980 Chevrolet	*CO (percent)*	*HC (ppm)*
1	0.00	50
2	0.10	10
3	0.00	0
4	0.10	25
5	0.00	50
6	0.00	0
7	0.10	200
8	0.10	10
9	0.00	50
10	0.00	25

CO = carbon monoxide; HC = hydrocarbons

*Partial list of 1980 Chevrolets tested in Boise, Idaho, study.

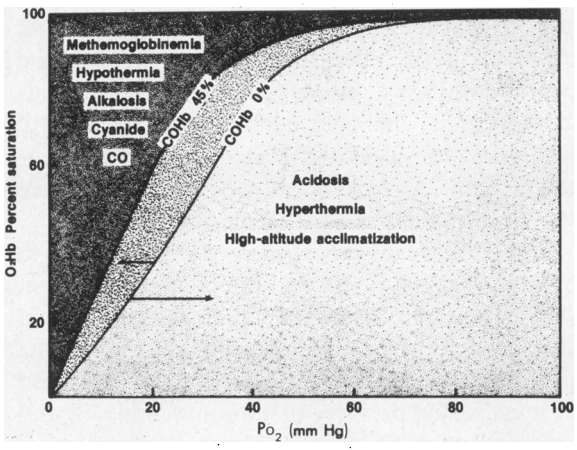

Figure 3.—Oxygen-hemoglobin dissociation curve (CO = carbon monoxide; COHb = carboxyhemoglobin; O_2Hb = oxyhemoglobin; Po_2 = partial pressure of oxygen). (Reproduced with permission from *Hospital Practice* 2: 48-72, Feb 1981, by Peter Frishauf, Editor.)

TABLE 3.—*Correlation Between CO Concentration and the Signs and Symptoms of CO Poisoning**

Percent CO in Atmosphere	*COHb Concentration (percent)*	*Signs and Symptoms*
	0-10	None (angina may be noted in patients with coronary artery disease).
0.007	10-20	Slight headache, exercise-induced angina, dyspnea on vigorous exertion.
0.012	20-30	Throbbing headache, dyspnea on moderate exertion.
0.022	30-40	Severe headache, nausea, vomiting, weakness, visual disturbance, impaired judgment.
0.035-0.052	40-50	Syncope, tachycardia, tachypnea.
	50-60	Coma, convulsions, Cheyne-Stokes respiration.
0.080-0.122	60-70	Compromised cardiorespiratory function.
0.195	70-80	Death.

NOTE: If the rise in COHb level has been unusually rapid, coma may ensue before other symptoms become apparent. Symptoms correlate best with the maximal pretreatment levels.

CO = carbon monoxide; COHb = carboxyhemoglobin.

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in the way of a true toxic effect. Rather, the tissue damage from CO is a result of hypoxia. Factors that may contribute to toxicity include total carboxyhemoglobin concentrations, length of exposure, metabolic activity during exposure, underlying disease states and presence of alcohol.[9,10]

The correlation between CO concentration and symptoms of CO poisoning is shown in Table 3. The symptoms of carbon monoxide poisoning are subtle and may range from slight headache and exercise-induced angina to severe headache with nausea and vomiting. On physical examination, cherry pink or red discoloration of the skin and mucous membranes, tachycardia and tachypnea may be noted. These symptoms progress to unconsciousness at a level of 40 percent to 50 percent carboxyhemoglobin concentration, which may be reached with as little as 0.035 percent to 0.052 percent CO in the atmosphere.[9]

Treatment for carbon monoxide poisoning consists of the administration of high-flow oxygen to hasten carboxyhemoglobin dissociation.[12] It has been shown that the excretion of carbon monoxide, like its absorption, is entirely through the respiratory system and that the elimination of CO follows an exponential time curve.[9-11] When ambient air is breathed, the carboxyhemoglobin concentration falls by half in approximately 250 minutes. When high-flow oxygen is administered, the concentration of carboxyhemoglobin is reduced by half during each 40-minute treatment period.[9,11] Using this rule of thumb, it can be assumed that our patient had a maximum carboxyhemoglobin level of between 55 percent and 65 percent, with about 0.080 percent CO in the atmosphere. These figures are compatible with those obtained from the emission tests for 1980 Chevrolets in the Idaho study.

Conclusion

Reduction in suicides can be shown to be the result of primary prevention—that is, improved knowledge and treatment of psychiatric illness—and secondary prevention—reduction in the lethality of traditional methods of suicide.[2] In this case, secondary prevention of suicide is aptly demonstrated, and represents a significant and heretofore unrecognized benefit of emissions control.

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