The elusive polluter

Carbon monoxide makes up a large share of air pollution, but its physiological effects in small quantities are hard to pin down

An invisible component of smog, carbon monoxide blood levels are measured by Drs. Dinman (left) and Paul M. Giever.

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by Jeanne Bockel

Carbon monoxide is a colorless, odorless, tasteless gas. It seductively, subtly and often fatally asphyxiates its victims within a matter of minutes. A product of combustion, it is also the most abundant of urban pollutants.

Although acute carbon monoxide poisoning is well understood, chronic poisoning, by low doses over prolonged periods of time, is not. Signs and symptoms are not striking and are hard to find. Chronic monoxide poisoning was first mentioned in 1869 when it was termed "insanity of cooks," a consequence of bending over badly ventilated stoves. But even though certain illnesses are attributed to chronic poisoning, few concrete facts are known.

In view of this, a nine-member committee was established by the National Academy of Sciences' National Research Council in July of 1968 to review the effects of chronic exposure to low levels of atmospheric carbon monoxide on human health (SN: 11/16, p. 503). The report, about to be published, reviews the existing literature and correlates the incidence of disease with levels of air pollution.

So far, it appears that atmospheric concentrations of carbon monoxide have no deleterious effect upon the tissues of the body, but they do have a definite effect on the central nervous system and may aggravate heart disease.

Carbon monoxide acts on the body by way of the hemoglobin, the substance which carries oxygen in the blood. By having 210 times the affinity for hemoglobin that oxygen does, it displaces the oxygen in the hemoglobin and combines with it itself, forming carboxyhemoglobin (COHb). This lack of oxygen in the blood and circulatory apparatus eventually affects not only the tissues but all organs of the body.

More than 94 million tons of carbon monoxide are emitted annually in the United States alone, with 50 parts per million in the air of most cities. It is a product of incomplete combustion and 78 percent of the load is from motor vehicle exhaust. Over an eight-hour period the metropolitan concentrations produce a level of approximately 5 percent COHb in the blood, as opposed to the normal level of 1.0 percent. In the atmosphere, every 100 parts per million of CO produces a level of about 15 percent COHb in the blood. The loading usually occurs within a couple of hours.

And, according to Dr. John T. Middleton, Commissioner of the National Air Pollution Control Administration, Arlington, Va. this is enough to make the situation grave.

At the level of concentration in the cities, carbon monoxide affects function and behavior primarily. Dr. Robert E. Forster of the University of Pennsylvania Medical School says this syndrome is characterized by fatigue, headache, confusion, irritability, dizziness and disturbed sleep. This could even occur at fairly low levels.

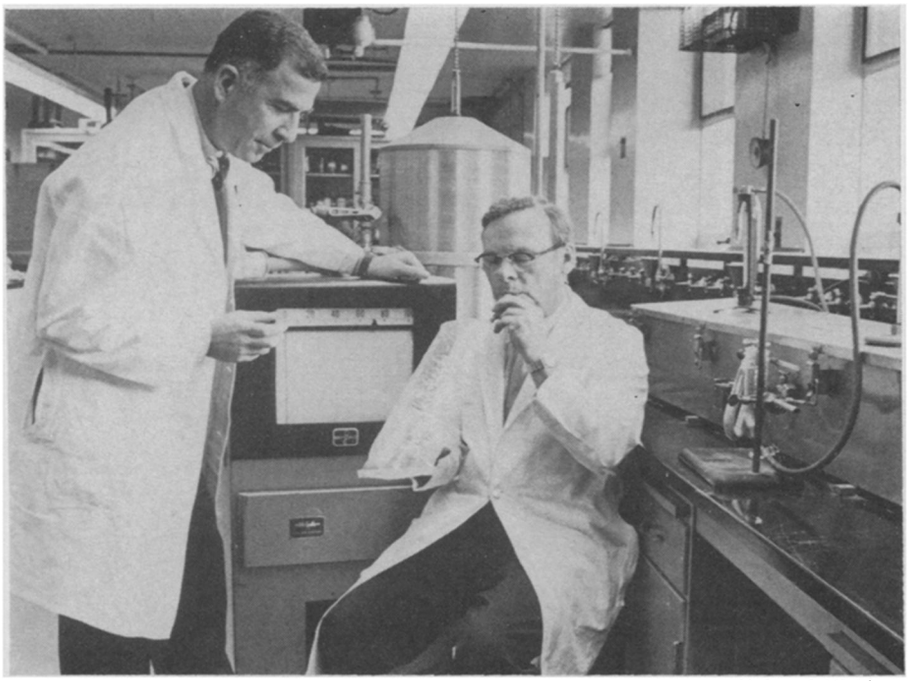
Dr. John H. Schulte of Ohio State University noted variations in performance at COHb levels well below 5 percent—possibly even at levels as low as 2 percent. He says significant changes in response after carbon monoxide exposure occur in pulse rate, respiratory rate, blood pressure, neurologic reflexes and psychomotor functions. When healthy subjects were exposed for 90 minutes to carbon monoxide in concentrations as low as 50 parts per million (COHb levels less than 2 percent), discrimination was impaired. Some could not perceive time intervals between noises, and others could not discriminate between time intervals of 10 and 30 seconds. For drivers, this is a hazard.

Dr. Forster says evidence is accumulating that driver performance shows a decrement at 20 to 25 percent COHb. And one study showed accident-involved drivers to have elevated COHb levels. Committee member Dr. Bertram D. Dinman, however, believes more sophisticated tests regarding decision-making during driving are needed because this impairment can be overcome with increased attention and proper motivation.

Although cigarettes do not significantly affect amounts of atmospheric carbon monoxide, they are a source of personal pollution. The median COHb concentration for pack-a-day smokers who inhale is 5.9 percent, a serious threat in persons with conditions like vascular insufficiency which require oxygen. Dr. Dinman, of the University of Michigan Medical Center, calls smoking a greater threat than atmospheric carbon monoxide. "COHb levels rise to 10 percent in some smokers and (pollution-caused) carbon monoxide loading, even under the worst conditions of ventilation, does not come anywhere near this."

There are fewer questions about the effects of higher concentration.

Exposures to carbon monoxide concentration of 200 to 350 parts per million in cholesterol-fed rabbits increased the arteriosclerotic processes, and in



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humans such levels have caused tissue damage to the central nervous system and myocardium (middle layer of the heart wall). Although this has not been evidenced with low concentrations of carbon monoxide, it suggests that carbon monoxide, at any level, may aggravate heart disease, even if subtly. Dr. John R. Goldsmith of the California State Department of Public Health says that in Los Angeles carbon monoxide pollution has definitely been associated with fatality in patients with myocardial infarction. And the facts linking cigarette smoking with coronary heart disease strengthen this theory.

It is not believed that carbon monoxide in any way relates to the cause of heart disease, but there is clinical foundation for the speculation about its aggravation of heart conditions. Patients with coronary artery disease compensate for their impairment by dilating the blood vessels and increasing flow to the heart. Myocardial oxygen consumption must be good in congestive heart failure, but oxygen is deficient in the myocardium at both 5 and 10 percent COHb levels, explains Dr. Dinman.

Although carbon monoxide air pollution has been a considered factor in respiratory diseases, the facts appear to be against it. Carbon monoxide uptake, explains Dr. Dinman, varies directly with depth and frequency of breathing; patients with chronic obstructive lung disease breathe less efficiently and would pick up less carbon monoxide.

This should apply as well to any chronic pulmonary disease. Furthermore, patients with chronic obstructive lung disease have increased blood oxygen content. Pollutants which irritate the lungs, thereby increasing airway resistance, are probably a more important factor among these patients. Basically, other than its interference with oxygen, carbon monoxide is chemically unreactive and doesn't affect the lungs, says Dr. Schulte. Other pollutants, especially sulfur dioxide and nitrogen dioxide when combined with aldehydes, are currently more under suspicion.

Research has shown that other conditions, such as cerebral vascular incompetence and increased thyroid activity, both of which require oxygen, are vulnerable to carbon monoxide poisoning. In anemia, hemoglobin is deficient and high COHb levels can be dangerous. Some diseases involving circulation to the extremities might also be aggravated by high monoxide levels.

In urban areas, says Dr. Dinman, community air quality standards should be adjusted to meet the needs of those individuals with conditions such as severe anemia or impaired circulation who are most vulnerable. He recommends the 8-hour community standard for carbon monoxide be dropped from 30 parts per million to 20 parts per million during the work day. Dr. Goldsmith says that exposures which produce 5 percent COHb concentration (30 parts per million over 8 hours along with occupational and smoking exposures) could increase the rates of illness and death. "Reducing levels to 20 parts per million is a totally justifiable concern for physically disadvantaged individuals," says Dr. Dinman.

On the other hand, says Dr. Schulte, air pollution control costs money and as yet there is no hard evidence that atmospheric carbon monoxide is a threat to health.

In general scientists do feel that a burden of 5 or 10 percent does not impose health risk, but that enough chronic studies as yet have not been conducted to warrant a decision. The results so far are contradictory.

One study conducted as far back as 1936 on men exposed to low doses (50 parts per million) of carbon monoxide working in the Posey Vehicular Tube in California, revealed the classic syndrome of carbon monoxide poisoning. But more recent studies of men with 13 years' duty in the Holland Tunnel (70 parts per million carbon monoxide) revealed no injury.

Urban air concentrations of carbon monoxide even at 30 parts per million cannot compare with that produced by 400-475 parts per million carbon monoxide found in cigarette air streams, says Dr. Dinman. However, to decide upon limits for community atmospheric carbon monoxide concentrations, "The high performance task of operating a motor vehicle on a high-speed freeway might be a point of departure," he suggests.

The length of time carbon monoxide remains in the atmosphere varies from 0.1 year to 5 years (SN: 11/1, p. 405), and it is estimated that atmospheric carbon monoxide would increase 0.03 parts per million per year. But though monoxide is rising in metropolitan areas due to motor vehicles, it is not increasing in unpopulated areas. Though not entirely understood, it is believed that a true carbon monoxide cycle exists in nature where the gas is either abosrbed, combined with other elements, or used metabolically by plants and animals.

It has been proposed that the committee next study the health effects of other contaminants such as oxides of nitrogen, sulfur dioxide, hydrogen sulfides, hydrocarbons and particulates such as lead, rubber and asbestos. □

SUMMARY OF NATIONWIDE CARBON MONOXIDE EMISSIONS, 1966

Source	CO Emissions	
	10^6 tons per yr.	Percent of Total
Transportation	**71.2**	**75.7**
Motor vehicles	67.3	71.6
gasoline	67.2	71.5
diesel	0.1	0.1
Aircraft	2.0	2.1
Railroads	0.1	0.1
Vessels	0.3	0.3
Nonhighway motor fuels	1.5	1.6
Fuel combustion: stationary	**1.9**	**2.0**
Coal	0.9	0.9
Fuel oil	neg.	neg.
Natural gas	neg.	neg.
Wood	1.0	1.1
Industrial processes	**7.8**	**8.3**
Solid waste disposal	**4.5**	**4.8**
Miscellaneous	**8.6**	**9.2**
Forest fires	7.2	7.7
Structural fires	0.2	0.2
Coal refuse	1.2	1.3
Total	**94.0**	**100.0**

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