# IV. ENVIRONMENTAL DATA

Information concerning environmental exposure to CO has been principally documented in the open literature concerned with air pollution.[2-5] Although CO has several valuable uses in industry (e.g., as a reducing agent), its presence in the industrial setting represents a source of pollution. In the first part of Chapter III it is pointed out that with the exception of carbon dioxide ($CO_2$), CO represents the largest single source of air pollution, and in Table II a summary of environmental pollution sources within industry is presented.

Because the emission of CO is on so large a scale it becomes difficult to single out, identify and rank individual sources in order of importance as concerns occupational exposure. Indeed, as emphasized earlier in this report, the smoking habits of the worker have a tremendous bearing on his actual daily exposure to CO. The CO pollution from traffic to and from the place of employment likewise will affect his total daily exposure. Several studies concerning occupational exposure to CO are presented below.

In an early study (1928) Broomfield[108] surveyed automobile repair shops in fourteen cities in the U.S. for CO concentrations. The average CO concentration of the twenty-seven facilities visited in the study was 210 ppm. Approximately 60 percent of the 102 samples at such facilities had concentrations of CO in excess of 100 ppm and 18 percent contained over 400 ppm. A nonsmoking individual engaged in light work (alveolar ventilation rate of 18 liters/min) and continuously exposed to these concentrations for four hours would be expected to have COHb levels approximately 23 percent (210 ppm), 13 percent (100 ppm) and 38 percent, respectively.

A survey by Buchwald[100] of COHb levels in Canadian garage and service station operators demonstrated, after six hours of exposure, that 60 percent of the smokers had COHb levels in excess of 5 percent as compared to less than 30 percent of the nonsmokers. In a nonexposed control group 50 percent of the smokers had COHb over 5 percent while in nonexposed controls there was a COHb range of 0-5 percent with a mean of 1.4 percent. Buchwald extrapolated the COHb levels in the smokers in both groups (exposed and unexposed) to relate to six-hour CO exposures of 80 ppm in the occupationally exposed group and 55 ppm in the nonexposed group. He suggested that the data in the study indicated cigarette smoking to be of greater significance to elevated COHb levels than automobile emissions.

A survey of employees in a parking garage prompted Ramsey[98] to conclude that occupational exposure to CO was more instrumental than was smoking in the study in producing COHb levels. Ramsey found that three nonsmoking employees exposed to an average of 84 ppm of CO had an average end-of-day COHb level of greater than 11 percent. In the main portion of the study, nonsmoking exposed workers had an end of day mean COHb concentration of 7.3 percent compared to 9.3 percent for smokers. In a non-occupationally exposed control group, nonsmokers had end-of-day average COHb levels of 0.81 percent and smokers in this group registered 3.9 percent.

Breysse and Bovee[99] conducted a survey of stevedores, gasoline-powered fork lift truck drivers and winch operators to determine their occupational exposure to CO. Using expired air breath samples the investigators found 5.7 percent of the approximately 700 COHb determinations were in excess of 10 percent and, after the workday, 7 percent of the stevedores and 18 percent of the lift truck operators had over 10 percent COHb. The obvious contribution of smoking upon the results of the study was demonstrated on "before work" COHb levels. Thirty percent of the smokers, but only 2 percent of the nonsmokers, exhibited COHb levels in excess of 5 percent.

Several reports[109] have concerned the exposure of inspectors to CO at U.S.-Mexican border-crossing stations. Although the hourly average CO concentrations fluctuated greatly in the studies (from 5 to 170 ppm in one study),[97] in the study by Cohen[97] an hourly average CO concentration of 114 ppm was found during the evening shift. During this shift nonsmokers had an average 1.4 percent COHb before duty and 4.0 after duty while smokers had an average 3.8 percent COHb before duty and 7.6 afterward.

Sievers, Edwards and Murray[110] in 1942 published the results of an in-depth medical study of men exposed to measured amounts of CO as workers in the Holland Tunnel for thirteen years. Although many parameters were measured, including examination for cardiac disease and neurologic disorders, it was concluded that the employees were in exceptionally good health and that there were no indications that CO or any other occupational exposure had influenced their state of health. The average CO concentration, which rarely exceeded 200 ppm, was 70 ppm. However, these employees were only exposed in the tunnel for two hours at a time, alternating two hours on an outside plaza where the CO concentration was "negligible." A nonsmoking individual engaged in light activity (alveolar ventilation rate of 18 liters/minute) could be expected to have a COHb level of approximately 6 percent at the termination of a continuous two-hour exposure period.

In a 1929 study of the COHb content of steel mill employees, Farmer and Crittenden[111] found an average 6 to 7 percent COHb saturation following an eight-hour workday. The COHb level at the beginning of the work shift, without regard to smoking habits, was approximately 2 percent and four out of fourteen employees had COHb levels greater than 10 percent.

In a study on the COHb levels in British steelworkers, Jones and Walters[112] found a 4.9 percent end-of-shift COHb saturation in nonsmoking blast furnace workers compared to 1.5 percent saturation in nonsmoking, unexposed controls. In heavy cigarette smokers the levels were 7.4 percent for blast furnace workers and 4.0 percent for controls. The range in values for the controls was 0.8 to 6.6 percent and in the blast furnace workers was 1.3-14.9 percent.

These studies indicate that while smokers definitely have higher COHb levels at the end of a workday, a significant portion of this is directly associated with their smoking habits. The studies also demonstrate that a significant portion of the work force is occupationally exposed to concentrations of CO sufficient to produce COHb levels of 5 percent and above in nonsmokers.

## V. DEVELOPMENT OF STANDARD

### Basis for Previous Standards

The former maximum allowable concentration (MAC) of 100 ppm for CO recommended by the American Standards Association (now American National Standards Institute) in 1945, was based principally on the work of Henderson and co-workers[113] published in 1921 and the often quoted work by Henderson and Haggard[114] published in 1943. Henderson and co-workers stated that when the concentration (ppm) x time (hrs) = 300, there was no perceptible effect; at 600, there was just a perceptible effect; at 900, there was headache and nausea; and at 1500 or more, the condition was dangerous to life. Stated differently, at 100 ppm a three-hour exposure produced no effect, but a six-hour exposure produced a perceptible effect and a nine-hour exposure caused headache and nausea.

In 1929 Sayers and co-workers[115] reported that in an experiment with six men who were exposed four to seven hours daily over a period of 68 days to 200 ppm CO, some of the more susceptible developed slight, but not discomforting symptoms after only two hours of exposure. After exposures for five to six hours COHb levels of 25 percent were reached.

Sievers and co-workers[116] reported in 1942 that a group of 156 tunnel traffic officers exposed over a thirteen-year period to an average concentration of 70 ppm CO did not reveal any evidence of injury to health attributable to the exposure. It should be pointed out that these men worked two hours inside the tunnel and two hours outside for eight-hour shifts. The average CO concentration in the tunnel was 70 ppm and the average COHb was 5 percent. None of the usual symptoms (e.g., headache, nausea, anorexia, etc.) were observed.

It should be emphasized that these early and many other later investigators have stressed the fact that the effect of CO on man is enhanced by many environmental factors. These have included rate of exercise, high environmental temperatures, altitudes above 2000 feet, and simultaneous exposure to narcotic solvents.

The American Conference of Governmental Industrial Hygienists' (ACGIH) Committee on Threshold Limit Values recommended 100 ppm for CO in 1945, and in 1965 the conference recommended that the limit be reduced to 50 ppm, a value that was officially adopted by ACGIH in 1967. In recommending the lower value[116] the Committee stated: "...for conditions of heavy labor, high temperatures of work 5000-8000 feet above sea level, the threshold limit value should be appropriately reduced to 25 ppm. No further benefit under any circumstances could be expected by reducing the level below 5 to 10 ppm since at this concentration one is practically in equilibrium with the normal blood level of around 1 percent COHb. The recommended TLV for CO of 50 ppm is thus based on an air concentration that should not result in blood CO levels above 10 percent, a level that is just below the development of signs of borderline effects." Since the "borderline effects" are not elaborated it is presumed that they are the clinical symptoms mentioned in the documentation (i.e., headache, fatigue and dizziness).

Mention should be made that the current MAC for CO in the U.S.S.R. and in Czechoslovakia is 18 ppm.[117] Furthermore, the U.S. Navy established a limit for the average concentration of a continuous exposure to CO for prolonged submarine voyages at 25 ppm,[118] and the National Aeronautics and Space Administration considers 15 ppm CO to be the maximum average concentration for space flights.[119] This latter low level was selected because of possible impairment of certain behaviors required of astronauts, a condition which could affect their performance during extended space flights.

The American Industrial Hygiene Association has recommended a community air quality guide for CO exposure of 20 ppm as an eight-hour average, which is stated to be equivalent to 3 percent COHb.[120] In addition a limit of 70 ppm has been recommended for a one-hour period, which is also stated to be equivalent to 3 percent COHb. The recommended limits are based on levels of CO concentration which will not exceed one-half of 5 to 6 percent COHb prior to tobacco consumption. It was considered that the recommended CO concentration would permit susceptibles with heart disease to obtain a time-based margin of safety prior to reaching the 5 to 10 percent COHb range.

From the viewpoint of health, limiting occupational exposure to CO to a TWA concentration which will produce no greater than 5 percent COHb may not provide a margin of safety for the employee with clinical symptoms of CHD, since Knelson has demonstrated that deleterious myocardial effects can occur at 3 to 5 percent COHb in patients with angina pectoris. However, limiting CO exposure to this level should protect the individual with asymptomatic CHD from developing clinical symptoms.

From the viewpoint of safety, limiting CO exposure to a concentration producing 5 percent COHb or less would appear to provide adequate protection for the worker against impairments in vigilance, coordination, timing behavior, visual perception, and certain cognitive functions.

Although the conditions by which a level of 5 percent COHb is reached in different individuals vary, dependent upon such parameters as activity, altitude, length of exposure, and CO concentration, as well as individual differences in the CO uptake, the nonsmoker who is engaged in sedentary activity will approach this level in eight hours if continuously exposed to 35 ppm of CO.

Basis for Recommended Standard

The recommended standard is based upon the cardiovascular and behavioral evidence presented in Chapter III of this report which generally documents the initiation or enhancement of deleterious myocardial alterations in individuals with CHD who are exposed to CO concentrations sufficient to produce a COHb level greater than 5 percent. The work of Ayres and co-workers[49,63] demonstrating restricted coronary blood flow and myocardial lactate production under such circumstances and the recent studies of Knelson[78] concerning CO exposure and exercise of patients with angina pectoris are germane to the recommended standard. Both investigations clearly demonstrate the potential hazards faced by workers with CHD who are exposed to CO of sufficient concentration to produce a COHb level in excess of 5 percent. The synergistic relationship imposed by chronic cigarette smoking and concomitant exposure to CO upon the enhancement of such detrimental myocardial alterations has been documented in a 1971 report to the Surgeon General on The Health Consequences of Smoking.[66] Based on the available evidence, the imposition of a COHb level of 5 percent on an active worker with clinical or asymptomatic CHD is unwarranted.

The extrapolation of this level of COHb (5 percent) in an exposed worker to a meaningful ambient CO concentration in the workplace imposes certain difficulties. Primarily, air sampling methodology must rely on statistical techniques to achieve an eight-hour, time-weighted, integrated analysis. Secondly, the rate of activity of the worker will increase the exposure to CO by decreasing the length of time to COHb equilibrium and by maintaining a higher COHb level in the active worker than in the sedentary worker who has not reached equilibrium. The significance of the first difficulty can be practically resolved by an air sampling protocol which will insure that sufficient quantities of such samples are taken to provide a reliable, statistical estimation of the proposed eight-hour standard. The activity factor can be practically minimized by the use of the Coburn[121] equation which takes into consideration, among other parameters, the activity of the worker in terms of alveolar ventilation rate and pulmonary diffusion rate.

The recommended TWA standard of 35 ppm CO is based on a COHb level of 5 percent, which is the amount of COHb that an employee engaged in sedentary activity would be expected to approach in eight hours during continuous exposure. The ceiling concentration of 200 ppm is based upon the restriction of employee exposure to CO to transient excursions above 35 ppm which would not be expected to significantly alter his level of COHb.

The recommended standard does not take into consideration the smoking habits of the worker since the level of COHb in chronic cigarette smokers has generally been found to be in the 4 to 5 percent range prior to CO exposure.

The recommended standard is based on the utilization of the Coburn equation[121] to predict the mean COHb level of nonsmoking employees exposed to a known TWA concentration of CO for an eight-hour workday. The applicability of the equation for this purpose has been validated by a study of Peterson and Stewart[122] in which the COHb levels of sedentary young males exposed to known TWA concentrations of CO for known periods of time were accurately predicted by the equation.

The investigators commented on the results of the study by saying:

"The series of experiments just described is unique in that the nature of the exposures is similar to those most commonly encountered in home, industrial, and urban environments."

While the values assigned to several of the variables in the equation by the investiagtors were for sedentary individuals (e.g., alveolar ventilation rate of 6 liters/minute; CO pulmonary diffusion rate of 30 milliliters/minute/ mm Hg), it is recognized that the range of activities of employees in the workplace may vary considerably. Under such circumstances, however, when the recommended occupational exposure standard is based on the prediction of a COHb level by the use of a theoretical equation, it is necessary that the prior applicability of the equation be demonstrated.

For this reason, the values assigned by the investigators to the variables for alveolar ventilation rate (6 liters/minute) and CO pulmonary diffusion rate (30 milliliters/minute/mm Hg) were retained in determining the recommended standard.

The Coburn equation has recently been programmed by Roslinski* and the compiled data is presented in Appendix IV. It will be noted that the two variables relating activity to rate of CO uptake [i.e., alveolar ventilation rate ($V_A$) and CO pulmonary diffusion rate ($D_L$)] are instrumental in determining the COHb level at the termination of a particular period of exposure. For example, an eight-hour TWA exposure to CO at the recommended exposure standard of 35 ppm will produce 4.89 percent COHb when $V_A$ = 6 liters/minute and $D_L$ = 30 milliliters/minute/mm Hg. However, when $V_A$ = 30 liters/minute and $D_L$ = 60 milliliters/minute/mm Hg, this same level of COHb will be reached by the second hour of exposure to the TWA. It is incumbent upon the employer

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to recognize the effect that the level of activity has upon the uptake of CO and to judiciously evaluate the exposure of his employees and limit their activity accordingly. The data in Appendix IV have been included specifically for this purpose. In addition, the employer must give special consideration to limiting the activity of employees exposed to CO at high altitudes in order to compensate for the dual loss in oxygen-carrying capacity of the blood.

## VI. COMPATIBILITY WITH AIR QUALITY STANDARDS

The Environmental Protection Agency (EPA), under provisions of the Clean Air Act (PL 91-604), promulgated national primary and secondary air quality standards on April 30, 1971.[123] The primary and secondary standards for CO are:

"(a) 10 milligrams per cubic meter (9 ppm)--maximum eight-hour concentration not to be exceeded more than once per year.

(b) 40 milligrams per cubic meter (35 ppm)--maximum one-hour concentration not to be exceeded more than once per year."

The EPA standard was based on criteria presented in "Air Quality for Carbon Monoxide" (35 F.R. 4768). The specific data upon which the EPA based the CO standard was primarily the work of Beard and Wertheim[39] who presented evidence that low levels of carboxyhemoglobin in human blood may be associated with impairment of ability to discriminate time intervals.

In promulgating this standard the Administrator of EPA made the following statement[123] concerning comments raised about the evidence used to support the proposed standard:

> "In the comments, serious questions were raised about the soundness of this evidence. Extensive consideration was given to this matter. The conclusions reached were that the evidence regarding impaired time-interval discrimination had not been refuted and that a less restrictive national standard for CO would therefore not provide the margin of safety which may be needed to protect the health of persons especially sensitive to the effects of elevated carboxyhemoglobin levels. The only change made in the national standards for CO was a modification of the 1-hour value. The revised standard affords protection from the same low levels of blood carboxyhemoglobin as a result of short-term exposure. The national standards for carbon monoxide, as set forth below, are intended to protect against the occurrence of carboxyhemoglobin levels above 2 percent. It is the Administrator's judgment that attainment of the national standards for carbon monoxide will provide an adequate safety margin for protection of public health and will protect against known and anticipated adverse effects on public welfare."

The air quality standard is designed to protect the population-at-large and takes into consideration 24-hour per day exposure of the very young, the very old, and the seriously ill. The evidence presented in this (NIOSH) criteria documentation supports the concept of the necessity of providing protection for that portion of the general worker population with coronary heart disease (CHD), who are especially sensitive to elevated levels of COHb.

Although the Administrator of EPA has stated above that the evidence concerning the impairment of time-interval discrimination at low levels of COHb, presumably as low as 2 percent, has not been refuted, neither has such evidence been confirmed.