

Hazard from Carbon Monoxide Poisoning for Bus Drivers in Tehran, Iran

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Air pollution is a worldwide problem with no geographical boundaries. Each year, 200 million tons of man-made waste products are released into the air, with internal combustion engines being responsible for almost one half (Dreisbach and Robertson 1987). One of the main pollutants in air is carbon monoxide (CO), which is of a significant public health concern and is toxic at low doses. Exposure to CO for the general population comes mainly from automobile exhaust, heating equipment, tobacco smoke, fire, and paint remover methylene chloride (Ellenhorn et al. 1997). Epidemiologic studies have consistently provided evidence of adverse health effects of this air pollutant (Krenzelok et al. 1996; La-Harpe 1997; Lester 1995). Chronic toxicity affects various body organs especially the central nervous system (CNS) and cardiovascular systems (Ellenhorn et al. 1997). Previous epidemiological study in Los Angeles county has suggested that the possibility of increased mortality from myocardial infarction, associated with high (9-16 mg/m³ or 8-14 ppm) atmospheric level of CO (Hexter and Goldsmith 1971). Daily fluctuation in CO levels have also been shown to be related to acute respiratory hospital admissions in children, to school and kindergarten absences, to decrements in growth rates of normal children, and to increased medication use in children and adults with asthma (Dreisbach and Robertson 1987).

One of the best ways to evaluate the extent of CO exposure is to determine the blood carboxyhemoglobin level (COHb). It is well-known that the air in cities with high concentrations of motor vehicles in many countries may be at risk of CO pollution, and Tehran, Iran has heavy automobile traffic and the highest air pollution of the nation (Abdollahi et al. 1997, 1995; Cook et al. 1991; Czerczak and Jaraczewska 1995; La-Harpe 1995; Moolenaar et al. 1995; Revert et al. 1995). Bus driving is an occupation in which, there is a potential for a continuous daily exposure to CO. In this regard, the present study was conducted on a random sample of bus drivers in Tehran, Iran to determine their blood CO level and any clinical subjective symptoms related to chronic toxic effects of CO.

MATERIALS AND METHODS

Forty-nine male bus drivers who worked 8 hours a day and 6 days a week in center locations of Tehran containing high concentrations of CO in air, were chosen for this study. The age of the subjects was between 27-47 years with job record of 5 to 25 years. By far the most common cause of high carboxyhemoglobin concentrations in man is the smoking of tobacco and the inhalation of the products by the smoker. Thus controls were selected from healthy non-smoker males who stayed and worked in low air pollution sites in Tehran where there were low levels of air CO pollution. The selected persons were invited to complete a questionnaire about their lifestyle, job record, assignment, work absence history, age, presence of any acute or chronic illness in the past or present, and subjective symptoms. All possible symptoms of CO chronic poisoning such as severe headache, nausea, abdominal pain, weakness and fatigue, dizziness, loss of sensation in fingers, amnesia (Ellenhorn et al. 1997) were included in the questionnaire. Concentrations of CO in air from various parts of Tehran were determined by automatic carboxymeters which were installed by the municipal government. These devices have been installed in different parts of Tehran to determine CO level in all parts of Tehran from 0100 to 2400 hours. Method of second-derivative spectroscopy was chosen for determination of blood COHb level (Parks and Worth 1985). All chemicals were purchased from Sigma chemical Co. (UK). About 1 ml of whole blood samples were obtained by venipuncture and stored in heparinized test tubes in a refrigerator at 4°C until transported to the laboratory for the determination of COHb level. COHb was measured by second-derivative spectroscopy between 390 and 450 nm using Shimadzu 160A scanning spectrophotometer. All samples were prepared between 1100 to 1200. A calibration curve was prepared from heparinized blood obtained from healthy nonsmokers. Two preparations of 0 and 100% COHb standards were provided by treatment of samples with 100% oxygen and the other with 100% CO; the remainder standards were prepared by mixing different portions of 100% and 0% CO preparations. Difference between values was analyzed by student t-test. Z-test was used to evaluate the significance of relationships between COHb values and symptom scores. In all analysis, $p < 0.05$ was assumed significant.

RESULTS AND DISCUSSION

There was a significant ($p < 0.01$) increase (70%) in blood COHb level in bus drivers ($4.19 \pm 0.658\%$) compared to controls ($1.09 \pm 0.275\%$) (Figure 1). About 6.12% of bus drivers and 98% of control subjects had acceptable blood COHb levels (1-2%). Fourteen percent of drivers were asymptomatic, whereas fatigue, weakness, nervousness, headache,

and amnesia were the most frequently complained symptoms in the remainder (Table 1). Bus drivers had higher symptom scores based on the total number of symptoms in Table 1, maximum of 7, than controls (3.42 ± 0.25 versus 1.14 ± 0.04 ; $p < 0.01$). Also there was a significant correlation between COHb (%) and overall reported symptoms ($r = 0.9$, $p < 0.01$, Figure 2).

Table 1. Frequency of chronic symptoms of carbon monoxide poisoning reported by 49 bus drivers

Symptoms	Frequency (%)
Weakness & Fatigue	53
Nervousness	38
Headache	33
Amnesia	13
Loss of sensation in fingers	11
Abdominal pain	8
Nausea	4

There was no relationship between COHb (%) and job record. Data obtained by automatic air carboxymeters indicate different values for atmospheric CO level in various parts of the city and time of the day. The higher level of CO was reported for central parts of the city between 1900-2200 hours (12.30 ppm) and then 0700-1000 hours (8.7 ppm). There was approximately a constant level for air CO (6.7 ppm) during 1100-1200 hours (data not shown). Therefore, sample collection was done during this time in this study. Natural background levels of carbon monoxide are low (0.01-0.9 mg/m³ or 0.01-0.8 ppm). Carbon monoxide concentrations in urban areas are closely related to motor traffic density and to weather and vary greatly with time and distance from the sources. The configuration of buildings is important and concentrations fall sharply with increasing distance from the street. Thus bus drivers are the best subjects to study adverse effects of air CO. An adverse level of CO for community air has been set at 9 ppm for a continuous period of 8 hours. The exhaust from gasoline internal combustion engines contains 3-7% CO. Present standards for new cars require limitation of CO emission to 0.5% (Krenzelok et al. 1996). There are usually well-marked diurnal patterns with peaks corresponding to

the morning and evening “rush hours”. Data from Japan and the USA show that 8-h mean concentrations of carbon monoxide are generally less than 20 mg/m³ (17 ppm). However, maximum 8-h mean concentrations of up to 60 mg/m³ (53 ppm) have occasionally been recorded (Hexter and Goldsmith 1971). Much higher relatively transient peaks may be observed in still weather where there is traffic congestion, and high concentrations can be found in confined spaces such as tunnels, garages, and loading bays in which vehicles operate and in vehicles with faulty exhaust systems.

A concentration in air of 6 ppm CO will increase the amount of hemoglobin in combination with CO by 1%. Our data indicate that approximately 94% of drivers study population have greater level of COHb than controls. It is important to take into account that chronic poisoning in the sense of accumulation of CO in the body might not occur. After the blood COHb level has returned to normal, susceptibility to CO is not increased unless cerebral damage was increased.

However, repeated anoxia from CO absorption will cause gradually increasing CNS damage, with loss of sensation in the fingers, poor memory, and mental deterioration (Ellenhorn et al. 1997).

Our study drivers, who have been exposed to automobile exhaust complained of weakness, fatigue, nervousness, headache, or amnesia. A recent review of occupational CO exposure indicates that mental changes such as amnesia and concentration deficits are most commonly observed with chronic CO toxicity (Wesle-Ely et al. 1995). This view is supported by our findings (Table 1) as amnesia and sensation disturbances in fingers were observed in the bus drivers. Other effects of CO exposure such as headache, nervousness, fatigue, or weakness, which were observed in our study population, have also been reported previously, but these are not specific for CO exposure and might be observed as a consequence of chronic lead exposure (Abdollahi et al. 1995). As shown in Figure 2, there was a relationship between COHb level and overall of complained symptoms, as previously noted (Krenzelok et al. 1996; Wesle-Ely et al. 1995). It was also found previously that the absorption of CO and the resulting symptoms are closely dependent on the concentration of CO in expired air, time of exposure, and state of activity of the person exposed (Cook and Simon 1995). Vehicle motor exhaustion was the main source of CO exposure in our population study. Our findings are strongly in agreement with the notion that CO exposure can lead to toxicity. In particular, the increased sensitivity of sick and aged people, as well as children, should be taken into account. It is suggested that the air concentration of CO must be kept below the exposure limit at all times by proper ventilation and all combustion devices must be vented to the outside air.

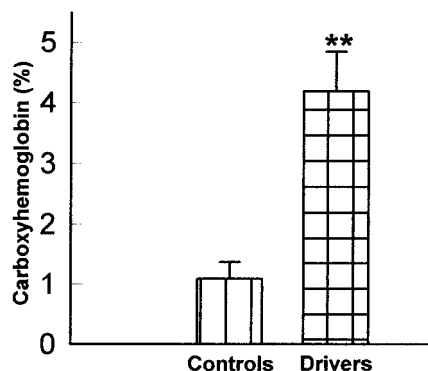


Figure 1. Blood carboxyhemoglobin level (%) in controls and bus drivers. Data are mean \pm SE. Number of controls=49. Number of bus drivers=49.

** difference between control and test groups is significant at $p < 0.01$

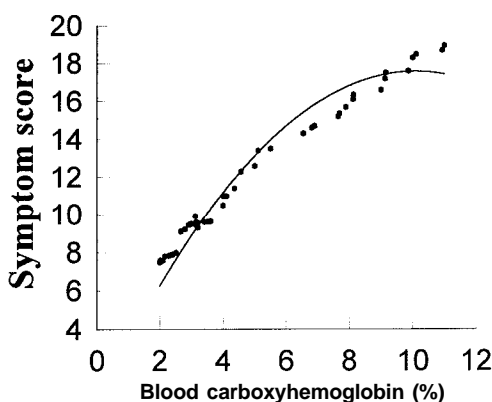


Figure 2. Relationship between blood carboxyhemoglobin level (%) and chronic carbon monoxide poisoning symptoms score in bus drivers. A symptom score based on a total of 7 common chronic symptoms (Table 1) with maximum of 7 was selected. Number of controls=49. Number of bus drivers=49. $r=0.9$, significant at $p < 0.01$.

REFERENCES

- Abdollahi M, Jalali N, Sabzevari O, Hoseini R, Ghanea T (1997) A retrospective study of poisoning in Tehran. *J Toxicol Clin Toxicol* 35: 387-391.
- Abdollahi M, Shohrati M, Nikfar, S, Jalali N (1995) Monitoring of lead poisoning in bus drivers of Tehran. *Iran J Med Sci* 20: 29-33.
- Cook M, Simon P A, Hoffman R E (1991) Unintentional carbon monoxide poisoning in Colorado, 1986 through 1991. *Am J Public Health* 85: 988-990.
- Czerczak S, Jaraczewska W (1995) Acute poisoning in Poland. *J*

- Toxicol Clin Toxicol 33: 669-675.
- Dreisbach R H, Robertson WO (1987) Handbook of Poisoning: Prevention, Diagnosis & Treatment. 12th eds., Appleton & Lange, NewYork, pp 259-263.
- Ellenhorn M J, Schonwald S, Ordog G, Wasserberger J (1997) Ellenhorn's Medical Toxicology (Diagnosis and Treatment of Human Poisoning), Williams and Wilkins, Maryland, pp 1465-1476.
- Hexter AC, Goldsmith JR (1971) Carbon monoxide: association of community air pollution with mortality. Science 172: 265-267.
- Krenzelok E P, Roth R, Full R (1996) Carbon monoxide the silent killer with an audible solution. Am J Emerg Med 14: 484-486.
- La-Harpe R (1995) Suicide in the Geneva canton (1971-1990). An analysis of the forensic medicine autopsy sample. Arch Kriminol 195: 65-74.
- Lester D (1995) The toxicity of car exhaust and its use as a method for suicide. Psychol Rep 77: 1090-1091.
- Moolenaar R L, Etzel R A, Parrish R G (1995) Unintentional deaths from carbon monoxide poisoning in New Mexico, 1980 to 1988. A comparison of medical examiner and national mortality data. West J Med 163: 431-434.
- Parks J, Worth G J (1985) Carboxyhemoglobin determination by second-derivative spectroscopy. Clin Chem 31: 279-281.
- Revert M, Brotons C, Navarro J, Gutierrez C, Doz J F, Cervantes M (1995) Winter epidemic of carbon monoxide poisoning in Badial. Aten Primaria 16: 261-264.
- Wesle-Ely E, Moorehead B, Haponik E F (1995) Warehouse workers' headache: Emergency evaluation and management of 30 patients with carbon monoxide poisoning. Am J Med 98: 145-155.