

# Carboxyhaemoglobin levels in workers in Leicestershire garages

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**ABSTRACT** Blood carboxyhaemoglobin (Hb<sub>CO</sub>%) levels were measured in 61 workers in 35 garages in Leicester and Leicestershire. Of 26 workers in large garages in winter, 44% of the non-smokers and 20% of the smokers had increased CO absorption, the highest Hb<sub>CO</sub>% being 26.0 in a non-smoker. Of 35 workers in 24 smaller, randomly chosen garages visited throughout the year (two per month) the corresponding figures were 43% and 14%, the highest Hb<sub>CO</sub>% being 21.0. The possible relevance of these findings to coronary heart disease in garage workers is discussed.

Petrol engine exhaust gases may contain up to 13% carbon monoxide (CO), depending on the engine's adjustment (Lawther, 1975). Garage workers are therefore at risk of absorbing significant amounts of CO during their daily work; the present investigation was planned to see if this theoretical risk is borne out in practice.

Our interest in this problem started when a man became semiconscious while painting the car showroom of a local garage, and was admitted to hospital with a presumptive diagnosis of CO poisoning. Unfortunately this diagnosis could not be substantiated because the patient was given oxygen for several hours before a diagnostic blood sample was taken; however, blood samples taken later from two other workers in the same showroom showed carboxyhaemoglobin saturations (Hb<sub>CO</sub>%) of 22 (non-smoker) and 28 (smoker, 25 cigarettes a day), respectively. The source of the CO was the garage's central heating system. This was of the hot air type with an enclosed combustion chamber, with its air intake, not in the open air as common sense would suggest, but in the main garage workroom where car engines were being run without local exhaust ventilation!

Enquiries revealed that it is unusual for local garages to have adequate means of preventing air contamination by exhaust fumes, hence this investigation.

## Methods

### SUBJECTS

Garages in Leicester and Leicestershire were visited

without warning, and venous blood samples were taken from mechanics in each. Individual mechanics were free to refuse to give a blood sample, the acceptance rate being about 80% of those asked. All visits were made after 4 pm because we wanted to sample the results of the day's CO build-up.

Two groups of garages were studied. Nine of the large main dealers, each employing more than ten mechanics, were visited in winter when the outside air temperature was below 5°C. In our experience these are the circumstances which encourage CO build-up, and we wanted to see what happened under the worst conditions. Twenty-four randomly chosen garages, comprising the second group, were visited throughout the year, two per month. The number of mechanics employed in each ranged from 0 to 16, but was mostly 0, 1 or 2, a reflection of the fact that, main dealers apart, most Leicestershire garages are small or very small. Twenty-six blood samples were obtained from mechanics in the first group of garages and 35 from those in the second group of garages.

### COLLECTION AND ANALYSIS OF SAMPLES

Blood samples were collected into 5 ml glass bottles containing EDTA as an anticoagulant (Labco Ltd). These were filled with blood and tightly secured. All analyses were completed within 24 h of collecting the blood.

The Hb<sub>CO</sub>% was determined by the method described by Conway (1957). Briefly, carbon monoxide was liberated from the blood by addition of sulphuric acid in a Conway microdiffusion unit, and absorbed in palladium chloride solution. During incubation of this solution free palladium and hydrochloric acid are formed, and the latter is titrated with potassium hydroxide, thus permitting calculation of

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the amount of CO liberated.

The measured results were corrected in accordance with the calibration curve shown in Figure 1. To produce this curve, Hb<sub>CO</sub> standards were made by anaerobically mixing variable quantities of blood which had been equilibrated with 100% CO and blood which had been equilibrated with oxygen. Due allowance was made for dissolved carbon monoxide.

## Results

The blood carboxyhaemoglobin saturations (Hb<sub>CO</sub>%) found in the 26 mechanics in the large garages in winter are shown in Figure 2. Taking the upper limit of blood Hb<sub>CO</sub>% in non-occupationally exposed workers to be 5 in non-smokers and 10 in smokers (see Discussion), then 7 out of 16 non-smokers (43.8%), and 2 out of 10 smokers (20.0%) show evidence of excessive CO absorption. Three (11.5%) had blood Hb<sub>CO</sub>% above 15, the highest being 26.0 in a non-smoker.

The Hb<sub>CO</sub>% values in the randomly chosen garages are shown in Figure 3, and the individual results in Figure 4. Six out of 14 non-smokers (42.9%), and 3 out of 21 smokers (14.3%) show evidence of excessive CO absorption, and two (5.7%) had blood Hb<sub>CO</sub>% levels above 15, the highest being 21.0 in a non-smoker.

Rather surprisingly there is little evidence in Figure 4 that CO absorption is more of a problem in winter than in summer, when natural ventilation of garage workrooms tends to be increased.

## Discussion

Heliövaara *et al.* (1978) measured blood carboxyhaemoglobin levels in a randomly chosen group of non-occupationally exposed men, and found that roughly 95% lay below 3% in non-smokers and 8% in smokers. On this basis we suggest that an Hb<sub>CO</sub>% above 5 in a non-smoker and 10 in a smoker is evidence of occupational exposure to CO. In the present investigation these levels were exceeded by 43.3% of the non-smokers and 16.1% of the smokers; blood Hb<sub>CO</sub>% values above 15 were found in 5 individuals (8.2% of total).

In the last decade there have been three studies of blood Hb<sub>CO</sub> levels in garage workers in other countries, but none to our knowledge in Britain. Buchwald (1969) looked at this problem in the winter months in Alberta, Canada, and found evidence of CO over-exposure (on the above-defined criteria) in 32% of workers, with 5% exceeding a Hb<sub>CO</sub>% level of 15. Köhl and Lob (1975) studied garage workers in 7 garages in Lausanne, Switzerland, and found Hb<sub>CO</sub>% average values ranging from 3.7 to 11.3 with

a highest individual result of 19 in a moderate smoker. Clausen and Rastogi (1977) found that 19% of autoworkers in Swedish garages had Hb<sub>CO</sub>% levels in excess of 12, but do not give further details.

Our results, therefore, do not differ greatly from those of other workers, although they suggest that CO exposure is rather greater in garages in Leicester and Leicestershire than in Alberta or Lausanne. This is not entirely surprising because all the garages in the Swiss study, and the majority of those in the Canadian one were provided with ducting for extraction of exhaust gases, whereas such local extraction was available in only two of the Leicester garages. Further, Köhl and Lob measured atmospheric CO levels in their garages, and the highest value they

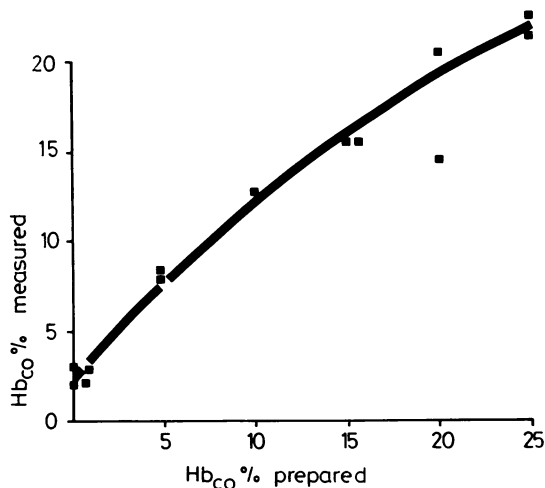


Fig. 1 Calibration curve for Hb<sub>CO</sub> measurements (see text).

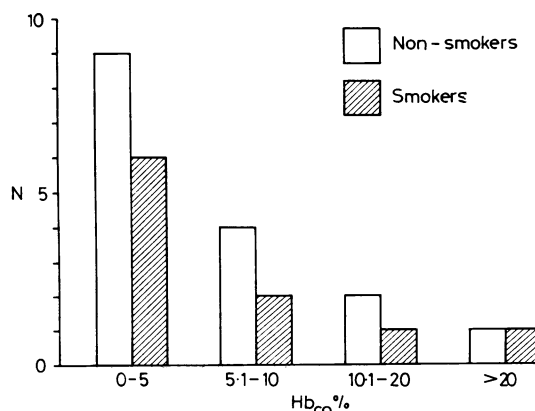


Fig. 2 Hb<sub>CO</sub>% levels in 26 workers in large garages in winter.

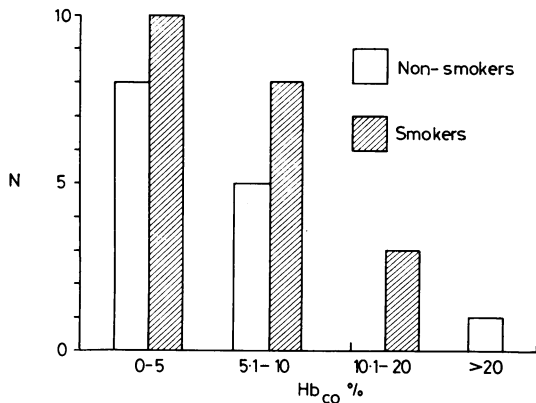


Fig. 3 Hb<sub>co</sub>% levels in 35 workers in random sample of garages (see text).

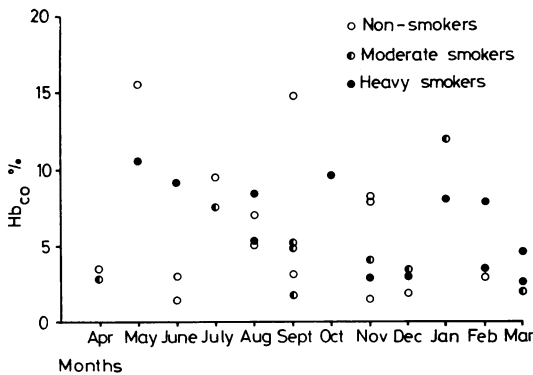


Fig. 4 Hb<sub>co</sub>% levels in 35 workers in random sample of garages, variation with time of year; heavy smoker > 15 cigarettes/day; moderate smoker ≤ 15 cigarettes/day or pipe smoker.

found was 77 ppm, with all the others below 50 ppm: we measured atmospheric CO levels in two of the large garages in our study, and found levels of 110 and 130 ppm, respectively. The present TLV for CO is 50 ppm.

Thus there is evidence of significant CO absorption in a substantial proportion of the garage workers included in the present investigation, and it is necessary to consider whether such absorption is likely to be of medical importance.

It has been shown experimentally (Stewart *et al.*, 1970) that exposure of normal subjects to CO sufficient to produce Hb<sub>co</sub>% levels of 15 caused mild headaches, and that higher levels induced severe frontal headaches which were made worse by exer-

cise and persisted even after exposure had ceased. We have no accurate figures but it is noteworthy that many of the workers we studied had symptoms, such as headache, muzziness and impaired concentration, which they attributed to poor ventilation at work. Köhl and Lob (who found rather lower Hb<sub>co</sub>% levels than those in the present study) found similar symptoms in some 34% of the workers they investigated.

There is also quite a body of circumstantial evidence which links chronic exposure to low concentrations of CO with the development of coronary heart disease; although in the absence of more direct epidemiological evidence this must be treated with circumspection, we think it is sufficiently impressive to warrant a brief review here.

Astrup *et al.* (1967) showed that chronic exposure of cholesterol-fed rabbits to levels of CO sufficient to give blood Hb<sub>co</sub>% levels of about 15 caused the appearance of atheroma-like arterial lesions. This finding has since been repeated in primates (Birnstingl *et al.*, 1970; Webster *et al.*, 1970). In addition, Thomsen and Kjeldsen (1974) showed that holding the Hb<sub>co</sub> level of rabbits at 18% for two days resulted in myocardial ultrastructural changes.

Ayres *et al.* (1970) showed, in both dogs and humans, that blood Hb<sub>co</sub> levels of 5–10% caused an increase of coronary blood flow with a decrease of coronary arteriovenous oxygen difference. If, therefore, the coronary blood flow was unable to increase because of atheroma, this degree of carboxyhaemoglobinemia would be expected to cause myocardial hypoxia. It would seem that this is, indeed, the case, because Anderson *et al.* (1973), in a double-blind and properly controlled trial, showed that even very low Hb<sub>co</sub>% levels caused a significant decrease of the amount of exercise needed to induce angina, and also an increase in the duration of the pain. Aronow and Isbell (1973) found a similar result, and De Bias *et al.* (1976) found that blood Hb<sub>co</sub>% levels of around 10 increased the susceptibility of primate hearts to electrically induced ventricular fibrillation.

Our findings relate to a small number of garages in one part of the country only. However, there seems no reason to doubt that similar results would be obtained more widely. On the basis of these findings, taken in conjunction with the above experimental evidence from the literature, we suggest that CO may play a part in inducing, or exacerbating, coronary heart disease in garage workers: we suggest that garage proprietors should take more care than they do at present to prevent build-up of CO in their premises. This can be done fairly readily by installing local exhaust ventilation to remove the CO at source, and/or by improving the general ventilation of areas where petrol engines must be run indoors.

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