Notes and miscellanea

Carboxyhaemoglobin levels in workers in Leicestershire garages

Kelman and Davies (36:238-41) showed that some non-smokers working in garages attain higher Hb_{co} levels than their smoking colleagues. This point was mentioned but no discussion or explanation was given for this finding.

This phenomenon had been noted previously by Curphey¹ and by Sievers *et al.*² The latter group, in their study covering New York's Holland Tunnel traffic officers for 13 years, found that the CO blood levels of non-smokers in the tunnel exceeded those of the smokers. All these men, smokers and non-smokers, remained healthy after 13 years' exposure, with a rate of heart disease no worse or better than the general population. Kelman and Davies should have considered the several papers available on tunnel traffic officers when making their conjectures on heart disease and garages.

Hugod and Astrup's recent paper³ on the possible aetiology of atherosclerosis could not show that carbon monoxide is responsible for the triggering of preatherosclerotic changes in the intima of the coronary arteries.

Kelman and Davies state that their interest was initially piqued "when a man became semiconscious while painting the car showroom of a local garage." Would it be reasonable to consider that paint solvent fumes might have been responsible? This possibility is not discussed.

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References

- ¹ Curphey TJ. Carboxyhemoglobin in relation to smoking. *Natl Cancer Inst Monogr* 1968;28:231-5.
- ² Sievers RF, Edwards TL, Murray AL. Effect of exposure to known concentrations of carbon monoxide. *JAMA* 1945; 118:585-8.
- ³ Hugod C, Hawkins LH, Kjeldsen K, Thomsen HK, Astrup P. Effect of carbon monoxide exposure on aortic and coronary intimal morphology in the rabbit. *Atherosclerosis* 1978;30:333-42.

Kelman replies:

Although on average the smokers had higher Hb_{co} levels than the non-smokers, some non-smokers had higher levels than some smokers. This is just what one would expect and therefore does not need detailed discussion, although we agree that the two groups are not as well separated as one might expect. The study by Sievers *et al* on tunnel traffic officers was epidemiologically weak in that it included only "survivors"; but nevertheless several officers had ECG abnormalities and impaired exercise performance, and it is therefore not true to say that they "remained healthy."

We agree that the evidence that CO causes atherosclerosis is shaky, particularly after Astrup's recent paper. But the evidence that CO absorption exacerbates existing myocardial hypoxia is reasonably strong, and is well reviewed by Becker and Haak.¹ Nevertheless, we stick to our original caveat, that "... in the absence of more direct epidemiological evidence this (the circumstantial evidence linking coronary heart disease and CO exposure) must be treated with circumspection. . . ."

Obviously, solvent fume narcosis was considered in the differential diagnosis of the painter's semi-conscious state, but this was of no relevance to our investigation.

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- ¹ Becker LC, Haak ED. Augmentation of myocardial ischemia by low level carbon monoxide exposure in dogs. *Arch Environ Health* 1979;34:274-9.