Postgraduate Medical Journal (October 1973) **49**, 684–687.

Blood carboxyhaemoglobin changes during tobacco smoking

M. A. H. RUSSELL
B.M., M.R.C.P., D.P.M.

Institute of Psychiatry, London, S.E.5

Summary

Using multiple samples collected via an indwelling venous cannula changes in blood carboxyhaemoglobin (COHb) were studied in five subjects before, during and up to 2½ hr after smoking. On average the smoking, with inhalation, of one cigarette in 6½ min raised the % COHb by 1·6, compared with 2·2 for 24 min of inhaled cigar smoking and only 0·2 for 20 min of non-inhaled pipe smoking. It is suggested that it may be paradoxically safer to smoke high rather than low nicotine cigarettes as those with a high nicotine content will tend to be inhaled less.

Introduction

Tobacco smoke is the main source of chronic exposure to carbon monoxide (CO). The blood carboxyhaemoglobin (COHb) level of non-smokers seldom exceeds 3%, whereas the average for smokers is about 5% and levels are found as high as 15% (Surgeon General's Report, 1972; Goldsmith, 1970; Jones *et al.* 1972; Lawther & Commins, 1970). By comparison with smoking the amount of CO absorbed from traffic pollution is very small. Average COHb levels of non-smoking taxi-drivers and policemen exposed to traffic in London were only 2·3% (range 1·4–3·0) (Jones *et al.*, 1972) and 1·9% (range 0·6–3·8) (Lawther & Commins, 1970) respectively. Increased interest is now being taken in the potential danger to smokers of chronic inhalation of CO, especially as a factor in the pathogenesis of arteriosclerosis (Astrup, 1972). It has been shown that the COHb level of smokers rises during the day roughly in accordance with the amount smoked and the degree of inhalation (Goldsmith, 1970), but no study has yet been published showing serial changes before, during and after smoking.

Materials and methods

Three cigarette smokers (all inhalers), a pipe-smoker (non-inhaler) and a cigar smoker (inhaler) were studied. Subjects were required to refrain from smoking for at least 12 hr (overnight) before the experiment. A butterfly cannula (No. 19 gauge) was inserted into a forearm vein and connected via a 3-way tap to a slow saline drip. Blood samples were collected into heparinised syringes which were sealed by bending back the needle. An initial sample was taken before smoking. Subjects then smoked their usual tobacco product in their usual way. The three cigarette smokers each smoked one cigarette, the pipe-smoker had one pipeful and the cigar smoker one cigar. Unknown to the subjects, an observer counted and timed every puff until smoking was discontinued spontaneously. Repeated blood samples were taken during smoking and at intervals up to 2½ hr after completion of smoking. Throughout the experiment subjects were seated in an armchair.

Analysis for COHb was done using an IL 182 Co-oximeter. This is an accurate instrument with reproducibility over the range employed having 95% confidence limits within 0·1% COHb (Russell *et al.*, 1973).

Results

Except for the non-inhaled pipe smoking, there was a rapid rise in COHb during smoking (Fig. 1). In two cases the COHb continued to rise during the first 1–2 min after spontaneous discard of the

TABLE 1. Tobacco consumption and blood COHb changes in five subjects

Subject	Sex	Age	Usual daily tobacco consumption	Smoking in experiment: Amount	No. of puffs	Time in min	Blood COHb: Before smoking %	1–2 min after smoking %	2½ hr after smoking %
1	F	31	30 cigarettes	1 cigarette	9	6·7	7·0	8·6	6·9
2	M	45	60 cigarettes	1 cigarette	17	7·0	7·6	8·2	6·8
3	F	41	17 cigarettes	1 cigarette	7	5·5	4·2	6·8	5·0
4	M	51	¾ oz pipe tobacco*	1 pipeful	92	20	6·2	6·4	6·1
5	M	40	1 cigar per week	1 cigar	30	24	2·9	5·1	4·5

* Subsequently confessed to smoking over 20 cigarettes a day in addition to the pipe.

cigar or cigarette. The increases in COHb varied between subjects but did not seem to be closely related to puff-rate. The greatest % COHb increase (2·6) occurred in subject 3 who took only seven puffs (see Table 1). On average the smoking of one cigarette in 6½ min raised the % COHb by 1·6 compared with 2·2 for 24 min of cigar smoking and only 0·2 for 20 min of non-inhaled pipe smoking. The fall in COHb after completion of smoking was very much slower (Fig. 2). On average it took 2½ hr for the COHb level of the three cigarette smokers to drop the 1·6% gained in only 6½ min of smoking.

Discussion

Despite the small number of subjects, the results clearly demonstrate the rapid rise of blood COHb which is evident within the first few puffs of tobacco smoking with inhalation. Provided the subject inhales the COHb rises; it matters not whether a cigar or cigarette is smoked. Yet little CO was absorbed after considerable puffing at a pipe without inhalation. This accords with the findings of Dalhamn *et al.* (1968) that 54% of CO taken into the lungs is absorbed compared with only 3% absorption through the buccal mucosa. It is likely that inhaled pipe smoking would increase COHb appreciably while non-inhaled cigarette and cigar smoking would not. Further studies are required to confirm this and to investigate whether different tobaccos vary in CO yield. It has been suggested that a high content of organic acids may increase CO yield (Baumberger, 1923).

Subject 4 was a former cigarette smoker who had received treatment from the author. He claimed to have stopped all cigarette smoking. The fact that he absorbed so little CO from his pipe during the experiment was difficult to reconcile with the high initial level of 6·2% COHb. He was confronted with this inconsistency and confessed to smoking over

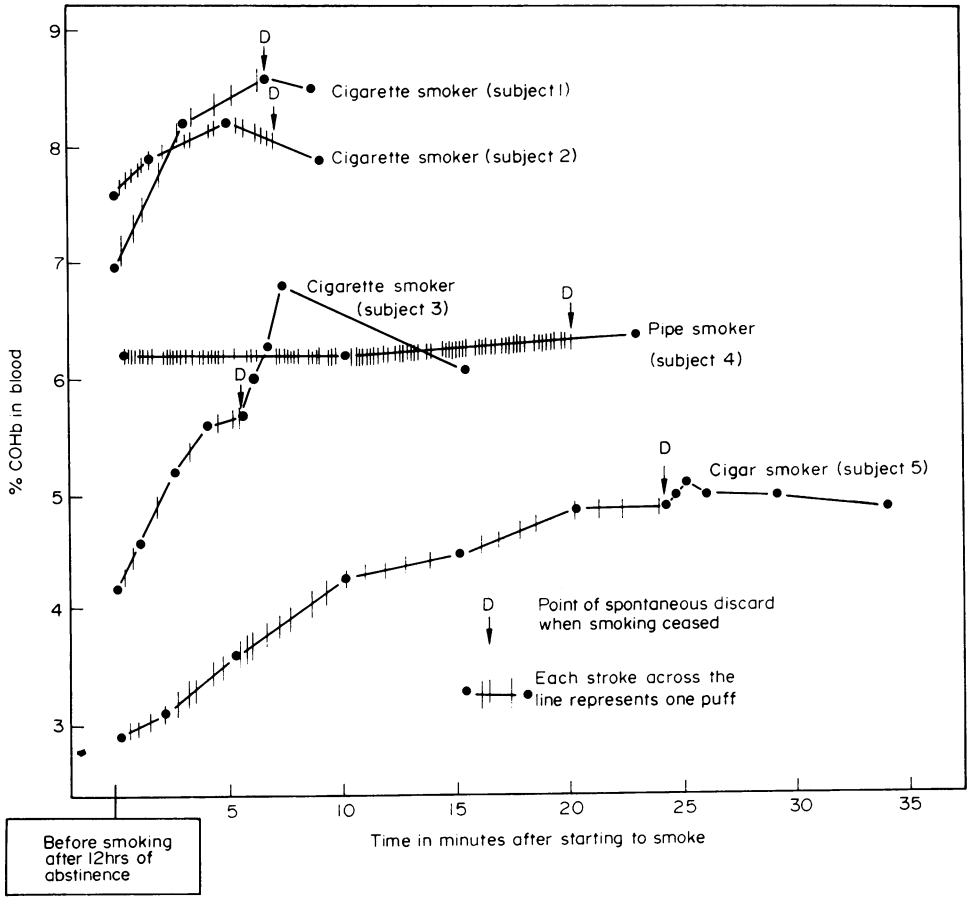

FIG. 1. Changes in blood COHb during smoking and up to 10 min after smoking.

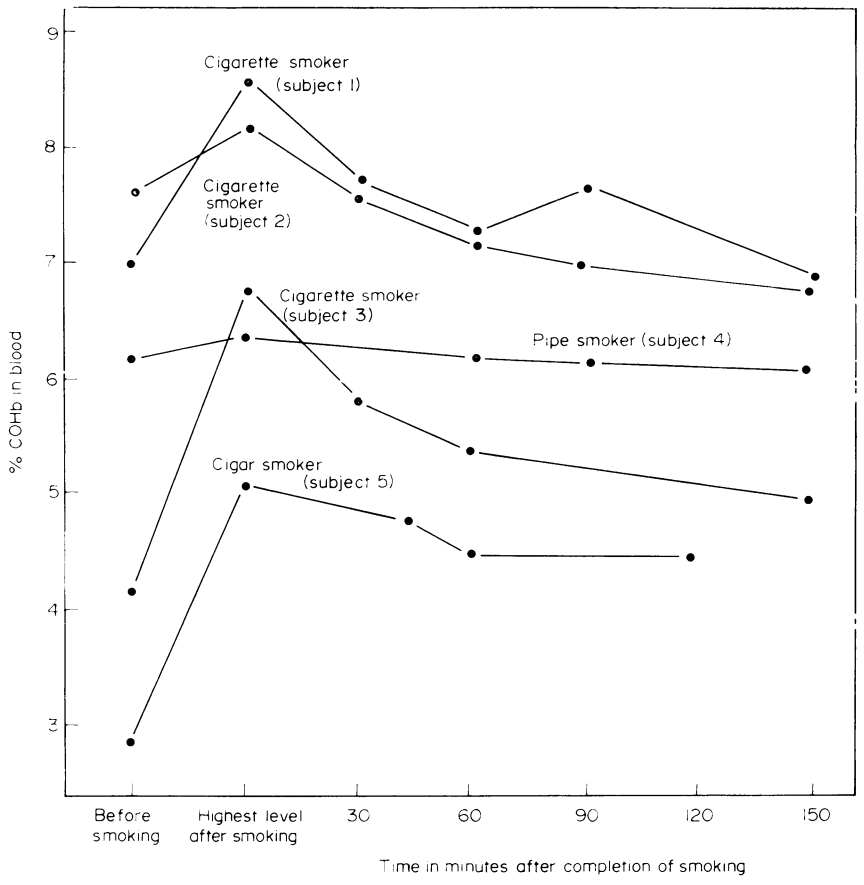

FIG. 2. Changes in blood COHb up to 2½ hr after smoking.

twenty cigarettes per day in addition to his pipe. This illustrates the potential use of COHb estimations to validate claims of non-smoker status.

In another study (Russell *et al.*, 1973) the average increase in % COHb of six smokers who were smoking in a smoke-filled room was 0·7 per cigarette smoked. This is very much less than the average rise of 1·6 shown by the three subjects of this study who smoked one cigarette. This difference may be due to the fact that the subjects of the former experiment may not have inhaled as deeply because they were recuctant to smoke having been instructed to smoke more than they were naturally inclined, whereas in the latter case subjects were eager to smoke having undergone 12 hr prior abstinence.

All this indicates therefore that the degree of inhalation is probably the most important determinant of CO absorption from tobacco smoke. Reduced CO absorption is likely to emerge as an important component of safer smoking. A tendency to regard low nicotine cigarettes as safer needs to be reconsidered. Most people probably smoke to obtain nicotine (Russell, 1971; Jarvik, 1970). There is evidence that with low nicotine cigarettes there may be a compensatory increase in inhalation to maintain the required nicotine intake. It may, therefore, be paradoxically safer to smoke high nicotine cigarettes requiring less inhalation, thereby reducing the absorption of CO and other potentially pathogenic products.

Acknowledgments

I am grateful to Dr Crammer and the Nursing Staff of the Metablic Ward at the Maudsley Hospital for their help. Mr R. Sillett, Chief Technician, Cardiothoracic Department, Central Middlesex Hospital, measured the COHb levels, and Dr Keith Ball gave some useful comments.

References

ASHTON, H., WATSON, D.W., MARSH, R. & SADLER, J. (1970) Puffing frequency and nicotine intake in cigarette smokers. *British Medical Journal*, **3**, 679.

ASTRUP, P. (1972) Some physiological and pathological effects of moderate carbon monoxide exposure. *British Medical Journal,* **4,** 447.

BAUMBERGER, J.P. (1923) The carbon monoxide content of tobacco smoke and its absorption on inhalation. *Journal of Pharmacology and Experimental Therapeutics,* **21,** 23.

DALHAMN, T., EDFORS, M.L. & RYLANDER, R. (1968) Retention of cigarette smoke components in human lungs. *Archives of Environmental Health,* **16,** 831 and **17,** 746.

GOLDSMITH, J.R. (1970) Contribution of motor vehicle exhaust industry, and cigarette smoking to community carbon monoxide exposures. *Annals of New York Academy of Science,* **174,** 122.

JARVIK, M.E. (1970) The role of nicotine in the smoking habit. In: *Learning Mechanisms in Smoking.* (Ed. by W. A. Hunt). Aldine Publishing Co., Chicago, 155.

JONES, R.D., COMMINS, B.T. & CERNIK, A.A. (1972) Blood lead and carboxyhaemoglobin levels in London taxi drivers. *Lancet,* **ii,** 302.

LAWTHER, P.J. & COMMINS, B.T. (1970) Cigarette smoking and exposure to carbon monoxide. *Annals of the New York Academy of Sciences,* **174,** 135.

RUSSELL, M.A.H. (1971) Cigarette dependence: nature and classification. *British Medical Journal,* **2,** 330.

RUSSELL, M.A.H., COLE, P.V. & BROWN, E. (1973) Absorption by non-smokers of carbon monoxide from room air polluted by tobacco smoke. *Lancet,* **i,** 576.

SURGEON GENERAL'S REPORT (1972) The health consequences of smoking. *U.S. Dept. of Health, Education and Welfare.*