TOBACCO INFLUENCE ON CARBOXYHEMOGLOBIN, OXIHEMOGLOBIN DISSOCIATION AND ERYTHROCYTE FILTRATION

J. FREITAS
Dept. of Biochemistry. Faculty of Medicine of Lisbon. Lisbon. Portugal.

SUMMARY

Ten patients were studied in 2 stages. Stage A: after 8 hours without smoking. Stage B: four hours after stage A, having each patient meanwhile smoked 5 cigarettes.

A significant increase of carboxyhemoglobin percentage ($p < 0.001$) and blood filtration time ($p < 0.01$) was reported while the P50std decreased ($p < 0.01$).

INTRODUCTION

The substances inhaled from tobacco smoke not only act on the vessel wall but also influence erythrocyte behaviour. The effect of chronic tobacco consumption on red cell filtration, leading to a rise of the filtration time, is widely known.[1-3]

This work was meant to study the influence of acute tobacco intoxication on blood filtration and on oxygen transport by hemoglobin as assessed by the percentage of carboxyhemoglobin present.

MATERIAL AND METHODS

Ten patients (5 men and 5 women) were evaluated in 2 stages. Stage A: After 8 hours without smoking; stage B: four hours after stage A, having each patient meantime smoked 5 cigarettes.

To each patient, 15 ml of blood was collected from antecubital vein during stages A and B; each sample was used to evaluate the following parameters: percentage of carboxyhemoglobin (CO-Hb), filtration time of 1 ml of blood and P50 std.

The percentage of CO-Hb was assessed after reduction of oxyhemoglobin with sodium ditionite using an oxymeter OSM2, according to the method of Siggaard-Andersen.[4].

The filtration time of 1 ml blood was estimated making the blood collected on EDTA flow through Nucleopore filters with 5μ orifices, under the pressure of -20 cm H_2O. The modified (Fig. 1) technique of Reid et al[5] was used and each sample was assessed four times being the final result the average of all the four measurements.

The estimation of P50std, that represents the partial pressure of oxygen for which 50% of the hemoglobin is saturated with O_2, was evaluated by tonometric method using a Radiometer system BMS 2MK2 connected to the pH unit (PHM 72 MK2) and to the O_2 electrode (E 5046), according to modified Astrup method.[6] The values of pH and PO_2 corresponding to two levels of SO_2 (measured with oxymeter OSM2 Radiometer) were obtained by tonometry and the value of P50std was afterwards deduced accordingly.[7]

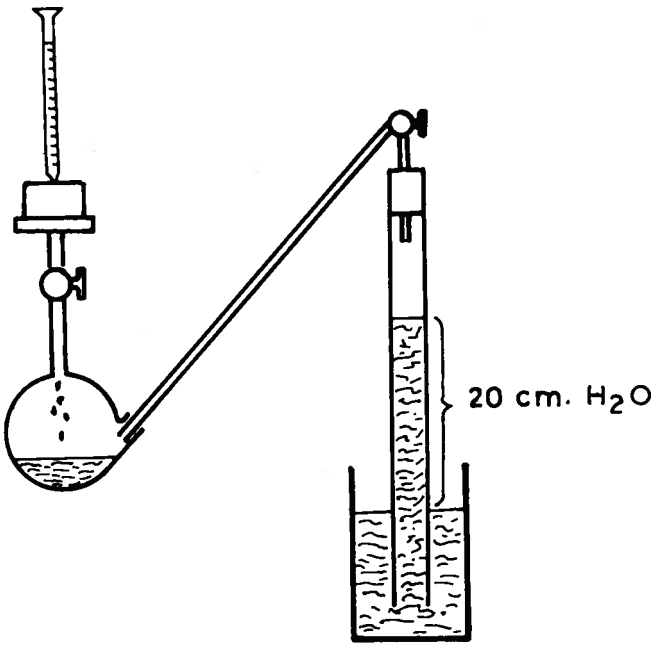

Fig. 1 — Draft of the system devised for the passage of blood across polycarbonate filters (Nucleopore Corp.) with sieves of 5μ in diameter, under the pressure of -20 cm H_2O.

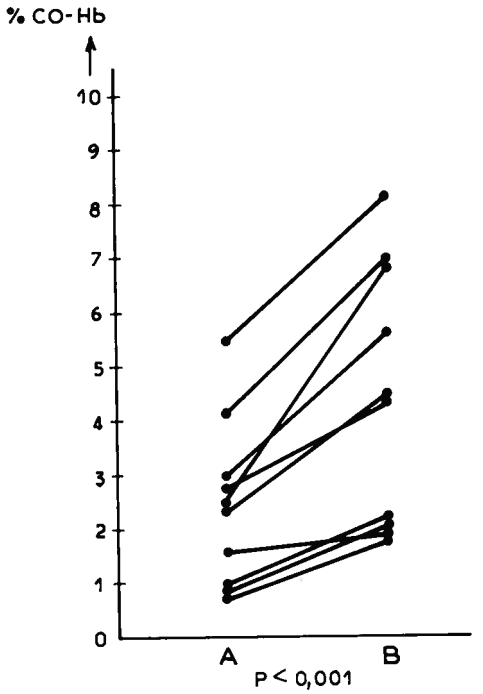

Fig. 2 — Percentage of carboxyhemoglobin: before tobacco intoxication (A) and after tobacco intoxication (B); the difference was statistically significant ($p < 0.001$).

RESULTS

Significant variations between the two stages were observed for the following parameters: increased ($p < 0.001$) percentage of CO-Hb (Fig. 2); increased ($p < 0.001$) filtration time of 1 ml of blood (Fig. 3) and decreased ($p < 0.01$) P50std (Fig. 4).

DISCUSSION

Several works point out evidence of acute and chronic abnormalities of red cell filtration induced by tobacco smoke, denoting that inhaled tobacco components, besides acting on the vessel wall, also exercice influence upon its contents, red cells included. Erythrocyte filtration rate decrease after 40 cigarettes/day,[1] after the inhalation of the smoke of 2 or 3 cigarettes,[1,2] but does not seem to be affected at all by inhaling the smoke of only one cigarette.[1,3]

The way by which tobacco interferes on red cell deformability is still unknown; several mechanisms have been put forward such as the rise of carboxyhemoglobin,[1,3,8,9] intrinsic red cell enzyme abnormalities,[3,10,11] decreased red cell concentration of ATP[1] and increased secretion of catecholamines.[1,10] The rise of neutrophil polymorphonuclear leukocytes could also be an important factor acting on chronic tobacco intoxication.[1]

The results suggest that the rise of carboxyhemoglobin percentage and the leftwards shift of the oxyhemoglobin dissociation curve, that course with acute tobacco intoxication, might cause chemical changes of hemoglobin structure and attendant abnormalities of internal red cell viscosity responsible for the reduction of erythrocyte deformability and the rise of blood filtration time.

ACKNOWLEDGMENTS

Supported in part by a grant from INIC (MbL2). We thank the collaboration from Hoechst Portuguesa.

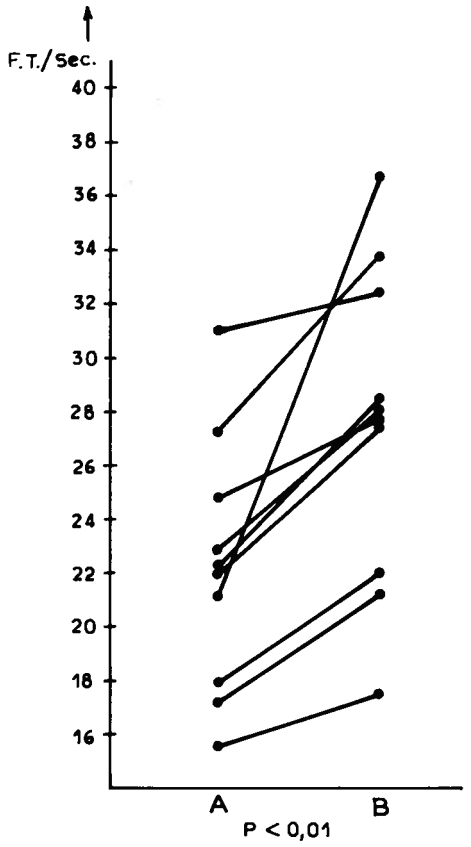

Fig. 3 — Filtration time of 1 ml of blood in seconds: before tobacco intoxication (A) and after tobacco intoxication (B); the difference was statistically significant ($p < 0.01$) .

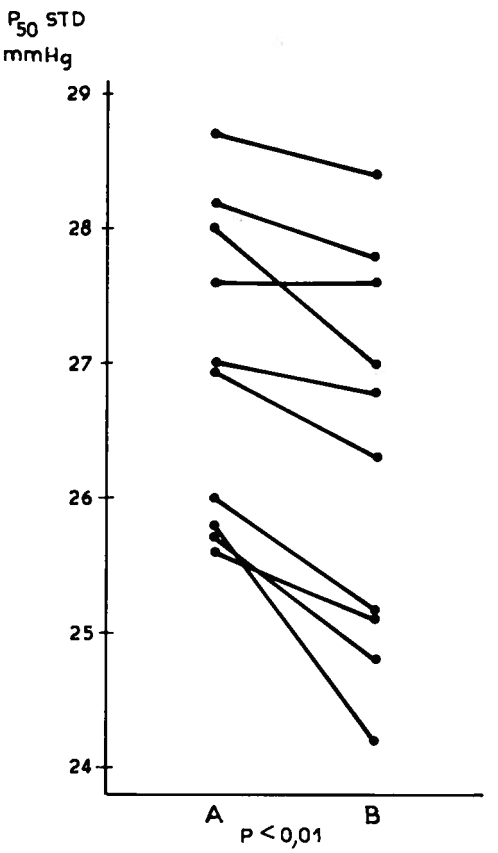

Fig. 4 — P50std in mmHg: before tobacco intoxication (A) and after tobacco intoxication (B); the difference was statistically significant ($p < 0.01$).

REFERENCES

1. LAGRUE, E.; MARCEL, G. A.:; FAUCHER, G.; GALLAIS, M. and BRANELLEC, A.: Nouv. Presse Med., 8: 4079 (1979).
2. MARCEL, G. A. and LAGRUE, G.: Nouv. Presse Med., 8: 4077 (1979).
3. LANDGRAF, H. and EHRLY, A. M.: Clin. Hemorheology, 33: 241 (1981).
4. SIGGAARD-ANDERSEN, O.: Scand J. Clin. Lab. Invest., 37 (suppl. 146): 45, 1977.
5. REID, H. L.; LOCK, P. J. and DORMANDY, J. A.: J. Clin. Path., 29: 855 (1976).
6. ASTRUP, P.; ENGEL, K.; SEVERINGHAUS, J. W. and MUNSON, E.: Scand. J. Clin. Lab. Invest., 17: 515, 1965.
7. ABERMAN, A.; CANAVILLES, J. M.; WEIL, M. H. and SHUBIN, H.: J. Appl. Physiol., 38: 171, 1975.
8. LAU, P. and ASAKURA, T.: J. Biol. Chem., 255: 1617 (1980).
9. VANIXEM, D.; GUILLOT, G. and GRINAUD, CH.: Nouv. Presse Med. 10:911 (1981).
10. STOCTZ, J. F.: Clin. Hemorheology, 3: 257 (1981).
11. NORTON, J. M. and RAND, P. W.: Blood., 57:671 (1981).

Address for reprints: J. Freitas
Depart. of Biochemistry
Faculty of Medicine
Lisbon. Portugal