ERYTHROCYTE FILTRABILITY AND OXYHEMOGLOBIN DISSOCIATION IN HYPERTENSIVE PATIENTS

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SUMMARY

Twenty one patients with essential hypertension were studied.

A significant decrease ($p < 0.05$) of hemoglobin oxygen affinity and a marked reduction ($p < 0.001$) of erythrocyte filtrability was detected. The results suggest an abnormality of the erythrocyte deformability in the microcirculation of the hypertensive patients, which implies a shift to the right of the oxyhemoglobin dissociation curve as a means of adaptation to the relative tissue hypoxia.

INTRODUCTION

Among cardiovascular risk factors hypertension plays a major role. The abnormalities of the walls of small vessels are well documented; however, oxygen transport and the adequacy of peripheral tissue perfusion are regulated not only by the integrity of the vascular system, but also by rheologic properties of the blood[1,2] and by hemoglobin oxygen affinity[3,4] that may favour cell delivery of this gas.

MATERIAL AND METHODS

Twenty one outpatients (10 men and 11 women), with essential hypertension were evaluation; they were 50.2 ± 10.5 years old (mean ± SD), within the age limits of 34 and 81 years.

The blood pressure levels in the day of the consultation were 167.5 ± 27.5 mmHg (mean ± SD) systolic and 106.2 ± 16.0 diastolic. Twenty ml of venous blood was collected from each patient in order to assay hemoglobin concentration (Hb), hematocrit (Ht), P50 standard (P50 std), carboxyhemoglobin percentage (CO-Hb) and rate of erythrocyte filtration (FR).

Hb was evaluated by photometric assay in oxymeter OSM2 (Radiometer, Copenhagen). The Ht was estimated by microcentrifugation at 10.000g for 5 minutes. P50 std, that represents the PO_2 at which 50% of the Hb is saturated by oxygen, was assessed by tonometric method resorting to a Radiometer system (BMS 2HK2) and oxygen electrode (E 5046). Using modified Astrup method,[5] the values of pH and PO_2 at 37° C corresponding to two levels of SO_2 (estimated with oxymeter OSM2 Radiometer) were evaluated and therefrom the value of P50 std was calculated.[6]

The percentage of carboxyhemoglobin was assessed after reduction of the oxyhemoglobin with sodium ditionate, using an oxymeter OSM2 according to Siggaard-Andersen method.[7]

The erythrocyte filtration rate, adjusted for the Ht, was calculated according to the method of Reid et al,[8] making blood collected into EDTA tubes flow through polycarbonate filters (Nucleopore Corp) with pores of 5 μ of diameter, under the pressure of —20 cm H_2O. Each sample was assayed 4 times and the average of the results was taken as the final value.

The group of hypertensive patients was compared with a group of normotensive individuals whose clinical examination and routine laboratory tests disclosed no abnormalities. For the evaluation of P50 std, % of CO-Hb and FR, only nonsmokers and women not taking contraceptive pills were used as controls.

TABLE 1 — Values (mean ± standard error of the mean) of hemoglobin concentration (Hb), hematocrit (Ht), carboxyhemoglobin (CO-Hb) levels, hemoglobin-oxygen affinity (P50 st) and red cell filtration rate (FR) in normal controls and patients with arterial hypertension.

Parameters	Controls	*p* values*	Hypertensives
Hb (g/100ml)	15.18 ± 1.05 (21)	NS	15.00 ± 0.98 (21)
Ht (%)	44.71 ± 2.50 (21)	NS	46.66 ± 2.62 (20)
CO-Hb (%)	0.76 ± 0.50 (17)	NS	0.61 ± 0.45 (18)
P50 st (mmHg)	27.74 ± 0.86 (33)	< 0.05	29.07 ± 1.82 (21)
FR (μl/sec)	15.80 ± 0.83 (34)	< 0.001	11.41 ± 1.31 (17)

NS — nonsignificant; *Student's *t* test.

RESULTS AND COMMENTS

An increased P50 std (Table 1) among the hypertensive patients ($p < 0.05$) denotes a rise in the capacity of hemoglobin to deliver oxygen to the peripheral blood. A rather significant decrease ($p < 0.001$) in erythrocyte filtrability, manifested by a longer time of filtration through the 5 μ pores, was also detected in the patients.

These results indicate hemorheologic alterations in hypertensive patients (decreased filtrability) that may depend on globular and plasmatic factors, and so may result in an impaired oxygenation of peripheral and system tissues.[1,2] The rightward shift of the oxyhemoglobin dissociation curve could be considered as a mechanism of adaptation or compensation[3] of a relative tissue hipoxia.

ACKNOWLEDGMENTS

This study was supported by a grant from INIC (MbL2).

We thank the collaboration from Hoechst Portuguesa.

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