STATE OF THE ERYTHROCYTES IN RATS WITH EMOTIONAL STRESS

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Neuropsychic stress is a leading risk factor in the development of cardiovascular diseases [1, 7, 8]. Overexcitation of the emotiogenic zones disturbs the mechanisms of self-regulation of several systems of the body (cardiovascular, respiratory, endocrine) [1, 3, 9]. In this connection the study of the effect of emotional stress (ES) on erythrocyte function is particularly interesting, because a disturbance of their functions, particularly respiratory, most undoubtedly be reflected in the intensity of metabolism at the level both of individual organs and of the body as a whole.

The aim of this investigation was to study the effect of neurogenic stress on the antioxidative properties of erythrocytes, and also on quantitative and qualitative changes in the red blood cells.

EXPERIMENTAL METHOD

Experiments were carried out on 73 male Wistar albino rats weighing 200-230 g. ES was reproduced by the use of a model of neurogenic stress of the "conflict of afferent excitation" type [1]. Blood samples were taken from the heart of animals of the control group and also of rats subjected to 1 (for 6 min), 5 (30 min), 10 (60 min), and 20 (120 min) cycles of exposure to stress, under hexobarbital anesthesia. The hematocrit index, hemoglobin concentration, and ESR were determined [5]. The antioxidative properties of the erythrocytes were judged from the accumulation of malonic dialdehyde during incubation for 1.5 and 3 h [4] and from superoxide dismutase (SOD) activity [10]. The efficiency of the respiratory function of the erythrocytes was estimated from the partial pressure of oxygen (pO_2) and carbon dioxide (pCO_2) in the blood, which was measured by means of a type OP-925/2 biological microanalyzer.

EXPERIMENTAL RESULTS

Neurogenic stress increases the intensity of free-radical oxidation of cell membrane lipids [6]. The level of peroxidation is limited by structural components, and also by the natural antioxidant system. Determination of MDA accumulation during incubation for 1.5 h showed that the level of lipid peroxidation of erythrocyte membranes was highest in animals subjected to one cycle of stress, namely 143.3% (119.9% in the control, $P < 0.05$). With an increase in the duration of stress the level of MDA accumulation fell.

The MDA concentration rose significantly in the erythrocyte membrane as a result of incubation for 3 h after exposure to stress for 6 min (up to 181.3% compared with 141.5% in the control); exposure to stress for 30 and 60 min led to maintenance of a high MDA level in the cell membranes (168.5 and 172.1% respectively, $p < 0.05$). Only after an exposure of 120 min did the MDA level in erythrocyte membranes fall (Table 1).

A study of the activity of the antioxidant enzyme (SOD) showed an increase by 31% ($P < 0.05$) after the very first cycle (6 min) of exposure to stress, and remained at that level regardless of the duration of the experiment. The rise in the SOD level in the early period of stress was evidently already not sufficient to inhibit the burst of free-radical oxidation observed under these circumstances. Later, however, other protective mechanisms may perhaps be mobilized, with consequent depression of lipid peroxidation.

Evidence of adaptation of the rat to stress was given also by the sharp fall in pO_2 in the initial periods of stress, down to 69.6 and 67.9% ($P < 0.01$) respectively after 1 and 5 cycles of exposure (81.3% before stress).

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TABLE 1. Effect of Stress on Erythrocyte Function in Rats

Parameter	Control	Number of cycles of exposure to stress			
		1	5	10	20
Hemoglobin, g/liter	130,0±3,70	141,0±2,30	148,0±2,80	138,0±2,80	140,0±3,0
P		0,05	0,02	0,1	0,1
SOD	2,3±0,58	3,4±0,55	3,3±0,61	3,8±0,36	4,2±1,03
P		0,1	0,05	0,05	0,1
MDA accumulation, %					
Incubation for 1.5 h	119,9±5,20	143,3±10,79	133,6±10,27	138,0±9,87	126,2±5,92
		0,05	0,2	0,2	0,2
Incubation for 3 h, %	141,5±6,72	183,1±27,99	168,6±14,58	172,1±12,03	158,2±12,77
P		0,2	0,2	0,05	0,5
SOD, conventional units	1,94±0,144	2,55±0,213	2,17±0,133	2,25±0,157	2,52±0,177
P		0,05	0,1	0,1	0,05
pO_2	81,3±3,09	69,6±1,8	67,9±2,2	72,0±1,97	78,1±2,3
P		0,01	0,01	0,1	0,5
pCO_2	27,6±2,6	37,1±2,5	34,1±2,4	31,1±2,5	27,1±1,5
P		0,02	0,1	0,5	0,5

The value of pCO_2 at these same stages of ES was considerably increased. With an increase in the duration of stress these parameters were restored to their original levels (Table 1).

The investigations showed that neurogenic stress of varied duration has a significant effect on parameters of the red blood cells. Although there was no significant change in the erythrocyte count (as judged from the hematocrit), the hemoglobin concentration was increased even after the first cycle of exposure to stress to 141.0 g/liter (130.0 g/liter) in the control, $P < 0.05$). Five cycles of exposure caused an even greater increase in the hemoglobin concentration (to 148.0 g/liter, $P < 0.03$). Further exposure to the stimuli lowered this parameter a little.

ES of varied duration led to an increase in ESR. This increase was particularly marked after 10 cycles of exposure to stress, and this could lead to microcirculatory disturbances in different regions of the circulation.

ES thus has a significant effect on erythrocyte function, for by activating free-radical oxidation of lipids of erythrocyte membranes, it causes changes in their permeability and disturbance of the fixation and release of oxygen.

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