

ADRENALIN, NORADRENALIN, DOPAMINE, AND DOPA
IN THE BLOOD AND OTHER TISSUES OF ALBINO
RATS FOLLOWING HEAD INJURY

É. Sh. Matlina
and T. B. Rakhmanova

UDC 617.51-001.3-07:616-008.944.52-074

Closed head injuries are characterized by the development of a state of stress [1]. In this condition the diencephalon, one of the more important centers for regulation of the autonomic and metabolic functions of the organism, is subjected to powerful influences. An increase in the concentration of adrenalin-like substances in the blood and an increase in the excretion of adrenalin and noradrenalin in the urine have been described in patients with closed head injuries [2].

However, the character of the changes in the catecholamines and their precursors in the tissue has not been adequately studied.

The object of the present investigation was to study the changes in the concentrations of adrenalin, noradrenalin, dopamine, and DOPA in the blood and other tissues of rats before and after subjection to powerful stress caused by a closed head injury.

EXPERIMENTAL METHOD

Adrenalin, noradrenalin, and their precursors were determined in the blood, adrenals, heart, brain, and kidneys of male albino rats weighing 150-200 g, before and 15 min after infliction of a measured head injury.

The animals were decapitated. The concentration of adrenalin and noradrenalin in the blood plasma was determined by a fluorometric method [3]. To determine the adrenalin, noradrenalin, DOPA, and dopamine in the tissues, these substances were extracted by perchloric acid, the excess of potassium perchlorate was removed, and subsequent treatment was carried out as described previously [4].

EXPERIMENTAL RESULTS

The results obtained are given in the table. This shows that 15 min after injury, the concentration of adrenalin was increased in the blood, brain, heart, and kidneys. The increase in its concentration in the adrenals was not statistically significant.

The concentration of noradrenalin in the heart and brain showed a significant decrease. In the kidneys and adrenals this decrease was not statistically significant. The noradrenalin concentration in the blood was increased. The concentration of substances determined as dopamine showed a significant decrease in the brain and adrenals, while in the heart the concentration of this substance showed a tendency to fall. No dopamine was found in the kidneys of either the control or the experimental animals.

The decrease in the DOPA concentration was well marked only in the heart tissue, and the decrease in the brain, kidneys, and adrenals was not statistically significant.

These results showing the concentration of adrenalin and noradrenalin in the blood, adrenals, brain, heart, and kidneys are in general agreement with data given in the literature for the level of these substances in the organs [5, 6, 7, 10, 11]. The slight increase in the relative concentration of adrenalin in the blood and kidneys was evidently associated with the stress caused by decapitation.

Laboratory of Neuro-Humoral Regulation, Academy of Sciences of the USSR and Central Research Institute of Balneology and Physiotherapy (Presented by Active Member of the Academy of Medical Sciences of the USSR N. I. Grashchenkov). Translated from *Byulleten' Éksperimental'noi Biologii i Meditsiny*, Vol. 63, No. 3, pp. 55-57, March, 1967. Original article submitted March 24, 1965.

Concentration of Adrenalin, Noradrenalin, Dopamine, and DOPA in the Tissues (in $\mu\text{g/g}$ Tissue) and in the Blood Plasma (in $\mu\text{g/liter}$ Plasma) of Rats 15 Min after Head Injury

Organ	Groups of animals	No. of experiments	Adrenalin		Noradrenalin		Dopamine		DOPA	
			$M \pm m$	P	$M \pm m$	P	$M \pm m$	P	$M \pm m$	P
adrenal	control	23	1134 \pm 105		291,0 \pm 80,5		4800 \pm 630		37,0 \pm 4,7	
	experimental	13	1352 \pm 87	>0,1	88,3 \pm 60,6	<0,1>0,05	3206 \pm 438	0,05	35,6 \pm 8,3	>0,1
brain	control	23	0,04 \pm 0,017		0,51 \pm 0,05		3,9 \pm 0,43		0,04 \pm 0,009	
	experimental	13	0,10 \pm 0,019	<0,05	0,20 \pm 0,03	<0,001	1,6 \pm 0,58	<0,01	0,03 \pm 0,007	>0,1
heart	control	23	0,18 \pm 0,03		1,02 \pm 0,09		7,2 \pm 1,3		0,16 \pm 0,025	
	experimental	13	0,35 \pm 0,06	<0,05	0,21 \pm 0,03	<0,001	4,2 \pm 0,8	>0,05	0,07 \pm 0,02	<0,01
kidney	control	23	0,05 \pm 0,01		0,50 \pm 0,06		0 0		0,06 \pm 0,013	
	experimental	13	0,2 \pm 0,035	<0,01	0,30 \pm 0,08	>0,1	0 0		0,06 \pm 0,02	
blood	control	50	5,4 \pm 0,16		1,3 \pm 0,36		— —		— —	
	experimental	28	9,1 \pm 0,26	<0,001	3,4 \pm 0,32	<0,001	— —		— —	

In the adrenals, a high concentration of substances determined as dopamine was found. It has been shown, however, that the level of dopamine in this organ does not exceed 2% of the total dopamine content [8]. The content of dopamine found in these experiments in the heart and brain was from 4 to 6 times greater than the values obtained by other authors [6, 10, 11]. Evidently in the adrenals, and to a lesser degree in other organs, substances are present capable of giving fluorescence in the conditions of dopamine determination.

It may be concluded from reports in the literature that after head injury adrenalin is liberated from the adrenals, as a result of which its concentration in the blood is increased and it is subsequently distributed among the tissues. Because of these processes the level of adrenalin in the brain, heart, and kidneys regularly rises. However, it has also been reported in the literature that adrenalin, in high concentrations, may depress the function of the sympathetic-adrenal system by the feedback principle, evidently through its action on certain hypothalamic centers [9].

The authors consider that the decrease in the concentration of noradrenalin, dopamine, and DOPA in the tissues was associated with depression of the biosynthesis of catecholamines in these tissues in response to the increase in the adrenalin concentration. The increase in the amount of adrenalin reaching the hypothalamic centers in these conditions is possible because of the increased permeability of the blood-brain barrier observed after head injury [2].

LITERATURE CITED

1. N. I. Grashchenkov, E. M. Boeva, B. I. Kamenetskaya, et al., *Vopr. Neurokhir.*, No. 3, 1 (1964).
2. G. N. Kassil', N. I. Grashchenkov, É. Sh. Matlina, et al., *Doklady Akad. Nauk SSSR*, 158, No. 6, 1455 (1964).
3. É. Sh. Matlina, In the book: *Clinical and Experimental Methods of Investigation of Hormones and Mediators* [in Russian], p. 84. Moscow (1965).
4. É. Sh. Matlina, Z. M. Kiseleva, and I. E. Sofieva, In the book: *Clinical and Experimental Methods of Investigation of Hormones and Mediators* [in Russian], p. 25. Moscow (1965).
5. A. Bertler, *Acta physiol. scand.* 51, 75 (1961).
6. A. Bertler and E. Rosengren, *Experientia Basel*, 15, 382 (1959).
7. L. M. Gunne, *Acta physiol. scand.* 56, 324 (1962).
8. P. Holtz, *Pharm. Rev.*, 11, 2, 2, 317 (1959).
9. J. Malmejac, *Pharm. Rev.*, 44, 186 (1964).
10. J. Sano, K. Taniguchi, T. Jamo, et al., *Klin. Wschr.*, 38, S. 57 (1960).
11. P. A. Shore and J. S. Olin, *J. Pharmacol*, 122, 295 (1958).