

of artificial gravity on the animals was perhaps not completely equivalent to the action of the earth's force of gravity, for a decrease in the content of water-soluble proteins was observed in the white matter of the spinal cord of animals exposed to an artificial force of gravity during the flight. It can tentatively be suggested that under the conditions of flight the character of transport of metabolites in the system of nerve fibers of the spinal cord was modified (retarded).

The results of these investigations thus show that the decrease in the content of water-soluble proteins in the gray and white matter of the spinal cord and spinal ganglia of rats kept during flight in a state of weightlessness, observed a few hours after the end of the flight, is evidently attributable to removal of the static load from the locomotor apparatus.

LITERATURE CITED

1. A. V. Gorbunova and V. V. Portugalov, *Byull. Éksp. Biol. Med.*, No. 8, 168 (1977).
2. B. L. Van Der Waerden, *Mathematical Statistics* Springer-Verlag, New York (1969).
3. O. H. Lowry, N. J. Rosebrough, A. L. Farr, et al., *J. Biol. Chem.*, 193, 265 (1951).
4. D. P. Lloyd, *J. Neurophysiol.*, 6, 317 (1943).

BIOCHEMICAL CHARACTERISTICS OF SYNAPTOSOMES AND MITOCHONDRIA OF THE MOTOR CORTEX AFTER SENSORY DEPRIVATION

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Changes in cytochrome oxidase, monoamine oxidase, acetylcholinesterase, and Na,K-ATPase activity following early light deprivation were found in fractions of heavy and light synaptosomes and mitochondria isolated from the bodies of neurons of the rat motor cortex by gradient centrifugation. These changes differed in direction for different metabolic cycles and were specific in individual ultrastructures of the cell. The effect of sensory impulsation on functional activity of neurons in the various cortical projection areas is discussed.

KEY WORDS: enzyme activity of synaptosomes and mitochondria; motor cortex; light deprivation.

The writer showed previously that absence of visual afferentation in the early stages of ontogeny leads to depression of cytochrome oxidase (CO), Na,K-ATPase, and acetylcholinesterase (AChE) activity in the subcellular fractions of the visual cortex in rabbits [6]. These changes were regarded as the result of morphological and functional underdevelopment of specific neurons and their synaptic structures. Activation of monoamine oxidase (MAO) in the structures studied, which evidently reflected compensatory changes in particular metabolic cycles maintaining growth and development of the animal under changed external environmental conditions, was observed under these same conditions.

Considering the data in the literature showing an increase in the functions of other analyzer systems of the brain in visually deprived animals [7], the investigation described below was carried out in order to study the same enzymes in the synaptosomes and free mitochondria of the motor cortex under conditions of light deprivation.

EXPERIMENTAL METHOD

The brains of 20 control and 20 experimental rabbits were used. From birth the experimental animals were kept in a dark room for ten weeks, and then used in the experiment. The fractions of synaptosomes and mitochondria were isolated from the motor cortex by the method

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TABLE 1. Protein Content and Specific Enzyme Activity (in units/mg protein/60 min) in Subcellular Fractions of Rabbit Motor Cortex under Normal Conditions and after Light Deprivation ($M \pm m$)

Fraction	Protein, mg/ g tissue	CO, E ₅₅₀	Mg-ATPase, μmoles P _i	AChE, μmoles ACh	Na,K-ATPase, MAO, E ₄₅₀	Na,K-ATPase, μmoles P _i
Control						
Light synap- tosome	6,2±0,81	733,3±20,0	27,9±0,69	4,0±0,45	0,19±0,02	11,1±0,32
Heavy synap- tosomes	6,0±0,60	19,4±46,6	52,9±2,54	2,25±0,28	0,33±0,02	12,6±1,43
Free mitochondria	2,7±0,46	107,0±37,1	81,6±6,54	3,07±0,28	0,37±0,03	5,7±0,64
Deprivation						
Light synap- tosomes	6,6±0,63 106%	579,0±46,4 78,8%*	56,7±3,9 203,2%*	3,40±0,34 85%	0,50±0,04 263,2%*	8,1±0,48 73%*
Heavy synap- tosomes	6,1±0,40 102%	780±44,5 94,9%	70,0±3,8 132,3%*	2,90±0,20 129%*	0,52±0,05 157,6%*	24,9±1,77 198%*
Free mitochondria	4,3±3,28 159%*	756,5±53,0 70,8%*	74,9±8,18 91,8%	4,00±0,31 130,3%*	0,44±0,04 118%	3,1±0,45 55%*

Legend: 1) Asterisk indicates $P < 0.05$ compared with corresponding control. 2) E — optical density; ACh — acetylcholine; P_i — inorganic phosphate.

of De Robertis et al. [11] with some modifications. Activity of CO was determined spectrophotometrically in a micromodification of the method in [12], MAO as in [5], AChE as in [13], and Na,K-ATPase activity from the liberation of inorganic phosphate from ATP by the method in [15]. Protein was determined by Lowry's method [14].

EXPERIMENTAL RESULTS

The mean data of the protein content and specific activity of the enzymes tested in the subcellular fractions of the motor cortex of control and visually deprived animals are given in Table 1.

After light deprivation an increase (by 59%) in the protein content in the fraction of free mitochondria was found compared with the control. The protein content in the fractions of synaptosomes was indistinguishable from normal. Under these conditions changes were observed in the relative activity of the enzymes tested; the changes were manifested unequally and specifically in different subcellular structures. In the heavy synaptosome fraction significant activation of all enzymes was found except for a normal CO level, in the fraction of light synaptosomes there was significant activation of Mg-ATPase and MAO together with a statistically significant decrease in CO and Na,K-ATPase activity. In the fraction of free mitochondria a significant fall in CO activity was observed. The results thus indicate that visual deprivation in the early stages of ontogeny has a definite effect on metabolism of the neurons and their synaptic structures not only in the visual, but also in the motor cortex. Changes in enzyme activity in the subcellular fractions after visual deprivation are considered to reflect changes in mediator metabolism and ion transport in synaptic membranes and also in oxidative metabolism in the mitochondria of nerve endings and cell bodies in the brain. Differences in the functional state of heavy synaptosomes compared with other fractions can be judged in respect of Na,K-ATPase and AChE activity. Depression of their activity in the light synaptosome fraction against the background of activation in the heavy synaptosome fraction evidently also reflects the active state of the chemical mechanisms of these structures of the motor cortex. Meanwhile after light deprivation signs of a marked metabolic deficit were observed in the fraction of heavy synaptosomes in the visual cortex, and this must reflect the specific functioning of these nerve endings at the cortical level of the visual system [8].

Activation of MAO, as these experiments showed, was characteristic of both types of synaptosomes in the brain of the experimental animals. These data evidently reflect compensatory changes in MAO activity connected with the organization of the metabolism of biogenic amines after deprivation. In particular, a decrease in serotonin binding with synaptic receptors of the motor cortex has been demonstrated in similar experiments [9].

Neurons of the rabbit sensomotor cortex are mainly polymodal [3, 10] and the system as a whole is integratively triggered and can be regarded as the highest apparatus of interanalyzer integration [1, 2]. This may perhaps explain the reorganization of metabolism observed by the present writer and other workers [4] in the subcellular fractions of this region when the balance between external and internal environmental factors is disturbed as a result of sensory deprivation.

LITERATURE CITED

1. O. S. Adrianov, Principles of Organization of Integrative Activity [in Russian], Moscow (1976).
2. A. S. Batuev, The Function of the Motor Analyzer [in Russian], Leningrad (1970).
3. L. L. Voronin, in: Mechanisms of Unification of Neurons in a Nervous Center [in Russian], Leningrad (1974), p. 172.
4. L. M. Gershtein, Tsitologiya, No. 1, 53 (1976).
5. V. Z. Gorkin, I. V. Verevkina, and L. I. Gridneva, in: Modern Methods in Biochemistry [in Russian], Vol. 2, Moscow (1968).
6. E. L. Dovedova, Ukr. Biokhim. Zh., No. 5, 17 (1977).
7. A. Ya. Nachkebiya, S. O. Lordkipanidze, and N. N. Postnikova, Zh. Vyssh. Nerv. Deyat., 27, No. 3, 648 (1977).
8. Z. D. Pigareva, M. M. Busnyuk, L. M. Gershtein, et al., in: The Physiology and Biochemistry of Ontogeny [in Russian], Leningrad (1977), pp. 119-124.
9. M. G. Uzbekov, Zh. Vyssh. Nerv. Deyat., 26, No. 6, 1291 (1976).
10. P. Buser and K. E. Bignall, Internat. Rev. Neurobiol., 10, 111 (1967).
11. E. De Robertis et al., J. Neurochem., 10, 225 (1963).
12. H. Hess and A. Pope, J. Biol. Chem., 204, 269 (1953).
13. S. Hestrin, J. Biol. Chem., 180, 249 (1949).
14. O. H. Lowry, N. J. Rosebrough, A. L. Farr, et al., J. Biol. Chem., 193, 265 (1951).
15. A. Samson and M. Quinn, J. Neurochem., 14, 421 (1967).