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MORPHOLOGY AND PATHOMORFOLOGY

Neuromorphological Basis of the Therapeutic Effect of Neurohumoral Agents of the Cerebrospinal Fluid

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The possibility of plastic rearrangements of the descending tracts from the intact hemisphere is of great interest in the problem of restoring lost functions after unilateral damage to the brain [6]. Endogenous oligo- and polypeptides from the cerebrospinal fluid (CSF) have been found to take part in the pathogenesis of central motor disturbances [1] and in later compensatory processes [3]. Treating animals with brain damage with different combinations of these agents results in an accelerated restoration of impaired motor function [8]. Previous physiological and biochemical inves-

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TABLE 1. Number of Labeled Neurons in Different Experimental Groups ($M \pm m$)

Recipients	Mode of HRP administration	CSF	HRP-positive neurons in sensorimotor cortex	
			right	left
Control:				
intact	Bilateral	none	107±8	102±8
	21 day	140±13*	145±11*	
	N	122±9**	103±13**	
Control:				
intact	Unilateral	none	28±3	93±7
	21 days	93±12*	117±13*	
Control:				
operated	Unilateral	none	32±5	–
	21day	166±25*	–	
	N	66±6*	–	

Note. In the control (operated) animals the neurons of the left hemisphere were removed; one asterisk means $p<0.05$ in comparison with control, two asterisks mean $p>0.05$ in comparison with control ; N: CSF of intact animals; 21 days: CSF from donors – convalescent (21 days after trauma).

tigations have yielded littleinformation about the morphological changes induced by donor CSF.

The aim of the present study was to examine the morphological rearrangements induced by donor CSF using special features of horseradish peroxidase (HRP) transport in the corticospinal projections in intact animals and in animals with damage to the left sensorimotor cortex.

MATERIAL AND METHODS

Experiments were carried out on 50 male albino rats weighing 180-230 g. One microliter of a 30% aqueous HRP solution (Sigma VI) was injected ipsi-(on the right side) or bilaterally in the lumbar region of the spinal cord at the L_1 level. Two days after enzyme injection all the animals were killed by perfusion of saline followed by a fixative solution (0.4 % paraformaldehyde + 1.25% glutaraldehyde on 0.1 M phosphate buffer (pH=7.4)). The removed brain was postfixed with the same solution for 5 h and then immersed in 30% sucrose on the same buffer for 48 h. Serial sections of the brain were prepared on a freezing microtome. Every other section (40m) was stained [10] and the total number of HRP-labeled neurons in layer V of the sensorimotor cortex of the right and left hemispheres in the zone of cortical representation of the hind leg was counted visually under the microscope.

CSF was obtained by puncture of the cisterna magna in intact (10 rats) and operated (21 days after damage of the left sensorimotor cortex, 12 rats) donors. The recipients (28 rats) were intact and operated animals with ipsi- and bilateral HRP injection. All animals were administered 2 mg freeze-dried CSF, dissolved in 20 ml water, injected intracisternally under ether anesthesia 4 h before HRP administration. Cortical damage was performed in donors and recipients with a vacuum-extractor under hexenal anesthesia (40 mg/kg). The operated recipients were treated with CSF immediately after the operation. The data were statistically processed using the Student t test.

RESULTS

After HRP injection, in the lumbar swelling of the spinal cord labeled neurons appear in layer V of the sensorimotor cortex [9,11]. The results of calculation of the neuron number in the different groups of animals are listed in Table 1.

There are differences in the number of labeled neurons in intact animals of the control group and intact recipients treated with CSF from convalescent donors (21 days after damage). Bilateral HRP injection in intact animals significantly enhances HRP transport in the cortical neurons of both hemispheres. The average number of HRP-positive neurons in the animals treated with donor CSF from the central nervous system of intact animals did not differ from the normal one.

After ipsilateral HRP injection in intact recipients, labeled neurons are found mostly in the contralateral (left) cortex. Against the background of the convalescent CSF injection the number of labeled neurons reliably increases both in the contra- and in the ipsilateral hemispheres.

Previously it was shown that for unilateral damage of the corticospinal tracts HRP is transported from the denervated hemisegments of the lumbar region of the spinal cord to the ipsilateral (intact) hemisphere [7]. It is seen from Table 1 that this process is activated by CSF of convales-

cents. The same effect in the CSF of intact donors is less pronounced but also reliable.

Thus, CSF of convalescents activates HRP transport in the cortical neurons both in intact recipients and in animals with brain damage. The CSF of intact donors exhibits the same activity in recipients with a damaged CNS. It is known that an intracisternal injection of convalescent CSF speeds up the restoration of the functions of the contralateral extremities after unilateral removal of the sensorimotor cortex in rats. It may be assumed that the effect of the donor fluid is associated with the mobilization of neuronal connections that are not normally active. The biochemical inductors of this process may be oligopeptide and polypeptide agents which were previously determined to be the factors of postural asymmetry and compensation [2]. The preexistence of oligopeptide factors of postural asymmetry in the CSF of a donor with an intact CNS [4,5] probably accounts for the above-described effect of "intact" fluid.

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Quantitative Analysis of Rat Myocardial Tissue for General Overheating of the Organism

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A general, even short-lived, overheating of a homoiothermic animal induces pronounced and often irreversible morphofunctional changes in many internal organs and systems [13]. This is the rationale for the use of hyperthermia in oncotherapy, since tumor cells have been shown to exhibit a lower resistance to such treatment [3,14]. However, the induced alterations not only do not disappear after the termination of overheating, but actually may be intensified during the period of postheating restitution [7]. The long-lasting circulation of endogenous toxic metabolites [1] probably causes the destructive changes and the disturbances of circulation in different organs [8,10]. From the first minutes of thermal action the cardiovascular system undergoes functional reorganizations, such as a redi-

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