Investigations of electrical activity of MU in patients with disturbances of the vascular system (endarteritis and atherosclerosis) also revealed a decrease in the duration of the MU action potentials [7]. This suggests that in patients with spinal cord trauma shortening of the duration of the MU action potentials is due to changes in the peripheral circulation and subsequent atrophy of the muscle fibers. The cause of these changes is evidently prolonged adynamia. This hypothesis is supported by the results of investigations of hypokinesia, leading to impairment of metabolism [2, 4, 6], and of anatomical changes in the peripheral vascular system [1, 8, 11].

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MONOAMINE CONTENT IN THE MOTOR CORTEX AFTER INJURY TO THE OPPOSITE MOTOR CORTEX

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UDC 616.831-001-031.4-07:616.831.31-008.932.33-031.74-07

In chronic experiments on eight cats a spectrofluorometric study was made of the serotonin, dopamine, and noradrenalin content in the sigmoid cortex of the left cerebral hemisphere 2, 3, 4, and 5-8 days after removal of the symmetrical cortex of the right hemisphere. A decrease in the dopamine content and a tendency for a decrease in the noradrenalin and serotonin content were observed on the 2nd day, at the time of maximal disturbances of locomotor function. On the 3rd-4th and 5th-8th days, during the period of recovery of motor activity, the serotonin level increased, the dopamine content remained low, whereas the noradrenalin level rose considerably. The role of biochemical changes in the motor cortex in the mechanisms of recovery of locomotor function after injury to the symmetrical cortical region is discussed.

KEY WORDS: injury to the motor cortex; biogenic amines.

Compensation and restoration of functions of the injured brain have for a long time engaged the attention of clinicians and experimental scientists [1-3]. Published work has shown that after injury to the brain changes are observed in the monoamine content in its structures [8, 9]. It is accordingly particularly interesting to study the neurohumoral mechanisms of recovery, for this offers the prospect of being able to influence the course of compensation of disturbed brain functions through the action of physiologically active substances [4, 6].

The object of the present investigation was to study changes in the content of serotonin, dopamine, and noradrenalin in the motor cortex of the same animal after removal of the symmetrical cortex from the opposite hemisphere and in the course of recovery of the disturbed locomotor function.

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(Presented by Academician of the Academy of Medical Sciences of the USSR A. M. Chernukh.) Translated from Byulleten' Éksperimental'noi Biologii i Meditsiny, Vol. 86, No. 9, pp. 270-271, September, 1978. Original article submitted March 27, 1978.

0007-4888/78/8609-1132$07.50

TABLE 1. Changes in Serotonin, Dopamine, and Noradrenalin Content in Anterior Sigmoid Cortex of Cat at Different Times after Injury to Symmetrically Opposite Cortex, in ng/g Frozen Tissue; M ± m

Group of animals	Biopsy on					
	2nd Day			5th-8th Day		
	serotonin	dopamine	noradrenalin	serotonin	dopamine	noradrenalin
Control	623±4,9	461±3,6	373±6	905±13,2	708±10,5	245±13,2
Experimental	595±35	204±4,5	279±78,7	1465±62	396±10,4	395±8,1
Difference	−4,49%	−55,7%	−25,2%	+61,8%	−44,0%	+61,2%
P	>0,05	<0,001	>0,05	<0,001	<0,001	<0,001

EXPERIMENTAL METHOD

Chronic experiments were carried out on 8 cats, 4 of which acted as the control. Before the experiments the animals were anesthetized and transparent plastic sockets 1.8 cm in diameter were inserted into the cranial bones at symmetrical regions of the opposite hemispheres above the anterior sigmoid gyri. The dura covering the cortex was removed through the hole in the sockets. Ten days after the preparatory operation the sigmoid cortex of one hemisphere was removed. The content of monoamines (serotonin, dopamine, and noradrenalin) was determined in the sigmoid cortex of the intact hemisphere spectrofluorometrically [7, 10]. To do this, in chronic experiments on the same animal, cortical biopsy was performed in three stages: on the 2nd, 3rd-4th, and 5th-8th days after injury to the cortex. Biopsy of the signoid cortex of the control cats was carried out, in the absence of any injury in the other hemisphere, also in three stages at the same intervals as on the experimental animals. The results of biochemical analysis of the biopsy specimens were compared with each other at each stage. The reason for this type of comparison was that biopsy itself caused changes in the content of biogenic amines in the adjacent parts of the brain.

Amines were extracted from the extirpated areas of brain tissue by means of butanol and heptane. Noradrenalin and dopamine were adsorbed on alumina, from which they were subsequently eluted with phosphate buffer. Serotonin was adsorbed on a column of the ion-exchange resin Dowex 50 × 4 in the sodium form, from which it was eluted with caustic soda. Catecholamines were oxidized with iodine. The intensity of fluorescence of noradrenalin was measured at 375/485 nm, of dopamine at 315/375 nm, and of serotonin at 285/330 nm.

The animals were killed 15 days after the beginning of the experiments and the brain removed for morphological verification of the site of injury. The results of the biochemical tests were subjected to statistical analysis by means of Student's t-test.

EXPERIMENTAL RESULTS

During the first 30 min after unilateral extirpation of the motor cortex a marked disturbance of motor activity of the contralateral limbs was observed (the forelimb rested on the dorsum of the foot, rigidity of the hind limb was present, the gait was ataxic, and so on). These disturbances continued on the 2nd day; on the 3rd and 4th days they gradually disappeared, and on the 5th-8th days recovery from the disturbances of motor function was almost complete.

During disturbance and recovery of the animal's motor activity, changes took place also in the content of biogenic amines in the symmetrical sigmoid cortex of the opposite hemisphere (Table 1). On the 2nd day, i.e., at a time of maximal disturbances of locomotor function, a significant change was observed in the dopamine concentration, with a tendency for the noradrenalin and serotonin content in the experimental animals to fall compared with the controls. On the 3rd-4th and 5th-8th days, during the period of recovery of motor activity, the serotonin content increased. The dopamine content fell compared with the control, whereas the noradrenalin content rose substantially. The changes described, which were particularly marked on the 5th-8th day, are given in Table 1. In view of the fall in the level of dopamine, which is a precursor of noradrenalin [5], the increase observed in the noradrenalin content may be presumed to have been due to some extent to its increased synthesis of dopamine.

The results suggest that a probable mechanism of the recovery of locomotor function after unilateral injury to the motor cortex may be the participation of the symmetrical area of the opposite hemisphere in compensatory processes through the increased synthesis of noradrenalin from dopamine.

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MECHANISMS OF INHIBITION OF SYNAPTIC TRANSMISSION IN THE SYMPATHETIC GANGLIA OF RATS WITH ALLOXAN DIABETES

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UDC 612:891:616.379-008.64

In experiments on isolated cranial sympathetic ganglia of rats with alloxan diabetes the preganglionic nerve was stimulated and combined presynaptic action potentials (APs) and EPSPs of neurons of the ganglion were recorded. In rats with moderately severe alloxan diabetes progressive depression of rhythmic APs of the ganglion correlated completely with inhibition of the excitatory power of the presynaptic endings, i.e., with a decrease in the liberation of mediator and exhaustion of its operative fraction. In rats with the severe form of diabetes postsynaptic inhibition of neurons of the ganglion also was observed. The dynamic characteristics of conversions of the mediator, assessed on the basis of examination of posttetanic potentiation patterns, showed a very small change in the output of mediator but a substantial (by 38%) depression of replenishment of the mediator reserves per second compared with the control.

KEY WORDS: synaptic ganglion; synaptic transmission; alloxan diabetes.

Diabetes mellitus causes severe disturbances of the activity of the central and peripheral nervous system [2-4, 7, 8, 12, 13], including the autonomic division. In this disease degenerative changes are found in the lateral horns of the spinal cord and in the autonomic ganglia [2, 8, 12], and these must inevitably cause changes in the functional properties of these formations. However, no detailed investigation of the function of such important centers as the sympathetic ganglia has yet been carried out in diabetes.

This paper gives the results of an investigation of the function of the synaptic structures of the cranial cervical sympathetic ganglion (CCSG) of rats with alloxan diabetes.

EXPERIMENTAL METHOD

Experiments were carried out on 31 Wistar rats weighing 150-250 g. To obtain diabetes the rats were given a subcutaneous injection of alloxan in a dose of 17-50 mg/100 g body weight [9]. The blood sugar con-

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(Presented by Academician of the Academy of Medical Sciences of the USSR V. N. Chernigovskii.) Translated from Byulleten' Éksperimental'noi Biologii i Meditsiny, Vol. 86, No. 9, pp. 272-274, September, 1978. Original article submitted March 20, 1978.

0007-4888/78/8609-1134$07.50