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Histomorphological Features of Brain Recovery in Rats with Traumatic Brain Injury During Various Exercises

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ABSTRACT

Microscopic examinations of the tissue of the medulla from the cortical-subcortical region on the injured side after a traumatic brain injury and various exercises were performed. During the first day after modelling of traumatic brain injury all age groups of experimental animals showed a decrease in the number of intact neurons, an increase in the number of degenerative changed neurons, as well as glial elements. The most favourable mode of physical activity for all age groups of animals is dynamic exercises in the form of systematic swimming. While limited motor activity and performance of isometric exercises after a traumatic brain injury restrain the processes of restoration of the cellular structures of the brain tissue.

Key words: Traumatic brain injury, Microscopic examination, Brain, Rats, Physical activity

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INTRODUCTION

Traumatic brain injury (TBI) is an urgent medical problem, as it is one of the most common and dangerous brain conditions that cause chronic neurological damage in surviving patients. In Russia, about 700 thousand people receive TBI every year, 50 thousand of them become disabled. Typically, the primary trauma is followed by secondary pathophysiological processes that continue for several months after the trauma, which provides a wide therapeutic window for effective treatment and recovery. Unfortunately, to date, there is no effective method of therapy and rehabilitation of TBI, confirmed by clinical studies.

In recent years, the mechanisms of neuronal death after traumatic brain injury (TBI) have been the subject of close attention due to the high vulnerability of neurons and subsequent chronic neurodegeneration. Neuronal death due to trauma is a complex phenomenon possibly resulting from the simultaneous activation of various molecular cascades in response to trauma, and this activation remains effective over a long period of time and causes progressive brain atrophy with subsequent neurological dysfunction.

Astrocytes, which release trophic factors and cytokines, both inhibiting and promoting regeneration, fibroblast growth factor, interleukin-1, interleukin-3, interleukin-6, tumor necrotic factor, nerve growth factor, etc., play a significant role in the survival of neurons after trauma and the formation of synapses. After injury, the number of astrocyte organelles increases, which is probably associated with the need to produce many growth factors.

The most important challenges of intensive therapy for patients with TBI (including in the postoperative period) are the prevention and treatment of secondary brain damage, including secondary cerebral ischemia, and physical rehabilitation, which reduces the effect of secondary damaging factors and allows nerve cells to avoid death. One of the promising ways of rehabilitation for TBI can be the systematic performance of various exercises [1-10].

MATERIAL AND METHODS

The experiments were conducted on outbred laboratory albino rats aged from 21 to 210 days. The basis for the age periodization of rats was anatomical and physiological characteristics of animals. Work with laboratory animals complied with the basic regulatory and ethical requirements for laboratory and other experiments with the participation of experimental animals of different species.

Animals were divided into control and experimental groups. The experimental group was divided into age groups. The first subgroup is immature animals, i.e., from 21 to 51 days of age. The second subgroup is mature animals, from 70 to 100 days of age. The third subgroup is pre-senile animals, from 180 to 210 days of age. Control group included intact animals.

Traumatic brain injury was simulated under ether anaesthesia in the animal in prone position with its limbs

fixed. The surgical stage of anaesthesia was determined by the absence of a corneal reflex in the animal. A median longitudinal incision (2 cm) was made in the right parietal region of the scalp shaved and treated with an aseptic solution, the adjacent soft tissues were separated from the parietal bone and the resection craniotomy was performed in the right parietal region. For this, a 0.5×0.5 cm milling hole was made using a high-speed cutter. The dura mater remained unopened. For this purpose, the weight, which is a steel cylinder weighing 114.6 g, was dropped from a height of 20 cm along a polyethylene guide tube. The brain was exposed to 0.224 N.

The injury was caused once. The injury did not lead to rupture of the dura mater; there was a visible severe focal brain damage with progressive edema and slight bleeding thereunder. After injury, the skin of the animals was tightly sutured with surgical thread (0.2 mm), the suture was treated with an antiseptic solution. Bacterial therapy was carried out intramuscularly with gentamicin solution.

Immediately after injury, all experimental animals had a neurological deficit appearing in the form of gross hemiparesis on the opposite side of the injury in the form of plegia of the upper and lower extremities.

For microscopic examination, parts of the medulla were taken from the cortical-subcortical region in the injured area.

After decapitation, the skull was quickly opened, the brain was removed, washed in saline and placed in 5-10% neutral buffered formalin solution at pH 7.2-7.4. Frontal sections of the brain with an area of 0.5-1 cm2 prepared using a microtome, taken at the level of bregma, were first exposed to alcohols with an increasing concentration (70°, 80°, 90°, 96°, 100°), after which they were embedded into paraffin wax in accordance with the standard procedure for light microscopy. The prepared blocks were further cut into sections with a thickness of 5-7 μm, which were hematoxylin and eosin stained. Qualitative morphological changes in the brain tissues were assessed using Zeiss light microscope (Germany). For targeted ultratomy and in-depth assessment of the studied processes in the brain tissue, semi-thin sections up to 1 μm thick were cut out from epoxy blocks, stained with hematoxylin-eosin and toluidine blue, and examined with Opton light-optical microscope (Germany). The processes observed in the brain tissue were identified by morphometric processing of semi-thin sections (histological examination).

Morphometric analysis was performed. The absolute number or percentage in 100 cells observed in 10 microscopic fields of view of both intact and damaged neurons and glial cells was calculated. The neuron-glia ratio was calculated as the ratio of the total number of neurons to the number of glial cells.

All materials obtained were processed by the methods of variation statistics in Microsoft Excel. The reliability of the data obtained between groups was determined by the student's t-test.

RESULTS AND DISCUSSION

Changes in neurons and glial cells in immature rats exposed to different modes of physical activity after a traumatic brain injury

As Table 1 shows, on the first day after TBI modelling, the number of intact neurons in the damaged hemisphere in the experimental group was slightly lower than in the control group of animals by an average of 13%. During the subsequent maintenance of control rat pups under unlimited physical activity for 30 days, i.e., by 51 days of age, the number of intact neurons was 91 ± 4.2, which is 8 (10%) more than in the initial data. Consequently, immature rat pups during natural growth and development from 21 to 51 days of age had a natural age-related increase in the number of normal neurons. The neuron-glia ration in the control group of 21-day-old animals was 4.9. In the experimental group, after modelling traumatic brain injury, this ratio averaged 3.7.

Table 1: Morphometric changes in neurons and glial cells in immature rats after a traumatic brain injury subjected to various modes of motor activity.

Parameters	Motor modes	Value M ± m	
		Day 21 (After trepanation)	Day 51 (Experiment)
Number of normal neurons	C (without trepanation)	83 ± 3.4	91 ± 4.2
	UMA	72 ± 3.6	79 ± 2.3
	MST	73 ± 2.1	85 ± 3.6
	LMA	71 ± 4.3	69 ± 2.8
	IE	70 ± 3.4	57 ± 1.5
Number of damaged neurons	C (without trepanation)	5 ± 1.3	3 ± 1.1
	UMA	14 ± 2.1	11 ± 2.3
	MST	12 ± 1.7	7 ± 1.5
	LMA	15 ± 3.1	16 ± 2.5

	IE	11 ± 2.3	21 ± 3.1
Number of glial elements	C (without trepanation)	17 ± 1.2	36 ± 2.5
	UMA	20 ± 1.1	25 ± 1.6
	MST	19 ± 1.5	31 ± 2.3
	LMA	20 ± 1.2	23 ± 3.1
	IE	18 ± 1.3	22 ± 1.7
Neuron-glia ratio	C (without trepanation)	4.9	2.5
	UMA	3.6	3.2
	MST	3.8	2.7
	LMA	3.6	3
	IE	3.9	2.6

The histological study of the brain tissue of the animals of the experimental group showed an increase in the number of dystrophic-altered neurons in the affected hemisphere of the brain by 160% already on the 1st day after the TBI and averaged 13. Significant difference in the number of glial cells of the animals of the control and experimental (after TBI) groups at 21 days of age were identified.

In rat pups of the group of unlimited motor activity at 21 days of age, after modelling a traumatic brain injury, the number of intact neurons was 72 ± 3.6. This value was 11 (13%) less than in the control group (P<0.05). During the subsequent maintenance of these rat pups for 30 days the number of intact neurons increased to 79 ± 2.3, which is 7 (10%) more than the initial data and 12 (13%) less than in the control group (P<0.05). Consequently, immature control animals with TBI had no age-related decrease in heart rate during the next 30 days under unlimited motor activity (UMA) show a significantly smaller increase in the number of intact neurons. We should note an increase in the number of glial cells by 5 (25%), as well as a decrease in the number of degenerative and dystrophic neurons in comparison with the initial data by 3 (21%). In our opinion, this is explained by edema and subsequent formation of foci of coagulation necrosis because of the traumatic brain injury.

In immature rat pups under enhanced motor mode at 21 days of age after inflicting traumatic brain injury, the number of unchanged neurons was 73 ± 2.1, which is 10 (12%) less than in the control group (P<0.05). Microscopic examinations revealed an increase in the number of damaged neurons by 7 (140%) and glial cells by 2 (12%) compared to the control group (P<0.05). The neuron-glia ratio was 3.8. Dynamic swimming exercises during 30 days in these rat pups caused a significant increase in intact neurons and amounted to 85 ± 3.6, which was 12 (16%) more than the initial data (P<0.05). Enhanced motor mode (MST) by 51 days of age caused a decrease in the number of damaged neurons by 5 (42%) and an increase in glial elements by 12 (63%) compared with the initial data (P<0.05). The neuron-glia ratio was 2.7. Consequently, it can be argued that in immature animals that underwent a craniocerebral injury at 21 days of age, the performance of gradually increasing dynamic exercises in the form of swimming contributes to a faster recovery of neurons, a decrease in degenerative-dystrophic neurons and an increase in the number of glial cells.

At 21 days of age, in immature rat pups with limited motor activity (LMA), the number of intact neurons was 71 ± 4.3, which is 12 (14%) less than in the control group (P<0.05). Histological examination of the brain tissue of animals with limited motor activity on the 1st day after TBI revealed an increase in the number of damaged neurons by 10 (200%), as well as an increase in glial cells by 3 (18%) (P<0.05). The neuron-glia ratio was 3.6. In the process of daily long-hour restriction of physical activity, by stretching and fixing the limbs on a special table for 30 days in these rat pups, the number of intact and damaged neurons was 69 ± 2.8 and 16 ± 2.5, respectively, which is identical to the initial data and 22 (24%) less than the number of intact cells, and 13 (433%) more than the number of damaged neurons in animals of the control group at 51 days of age (P<0.05). We should note that the number of glial cells was 23 ± 3.1, which is 3 (15%) more than the initial data, but 13 (36%) less than in the control group. Thus, the subsequent limitation of motor activity (LMA) from 21 to 51 days of age in immature rat pups after traumatic brain injury did not cause a significant change in the number of investigated cellular structures. Therefore, it can be argued that the subsequent 30-day restriction of motor activity in immature animals with traumatic brain injury at 21 days of age inhibits the recovery of brain tissue.

In immature rat pups who perform isometric exercises (IE) at 21 days of age, the number of intact neurons was 70 ± 3.4, which is 13 (16%) less than in the control group (P<0.05). The number of damaged neurons and glial cells roughly corresponded to the number of these cells in groups of animals exposed to other modes of motor activity. Starting from the age of 21 days after a traumatic brain injury, over the next 30 days, the animals were tightly fixed on the turntable and gradually accustomed to hanging upside down (antiorthostasis). The performance of systematically increasing isometric

exercises for 30 days led to a significant decrease in the number of intact neurons, there were 57 ± 1.5 of them, which is 13 (19%) less than the initial data and 24 (26%) less than the control group at 51 days of age (P<0.05). Microscopic examinations revealed that the number of damaged neurons was 21 ± 3.1, which is 10 (90%) more than the initial data and 17 (566%) more than in the control group of animals (P<0.05). Consequently, the performance of isometric exercises after a traumatic brain injury leads to a significant deterioration in the compensatory and recovery processes.

Thus, the results obtained indicate that, after a traumatic brain injury, the most favorable mode of physical activity for immature rat pups is the performance of dynamic exercises in the form of systematic swimming. Limited motor activity inhibits the recovery of brain tissue, while performance of isometric exercises after a traumatic brain injury leads to a significant deterioration in compensatory and recovery processes.

Changes in neurons and glial cells in mature rats exposed to different modes of physical activity after a traumatic brain injury

In mature animals of 70 days of age, on the 1st day after TBI, the number of intact neurons in the damaged hemisphere was significantly lower in the experimental group than in the control group of animals by an average of 30% (P<0.05). During the subsequent maintenance of control rat pups under unlimited physical activity for 30 days, i.e., by 100 days of age, the number of intact neurons was 97 ± 3.4, which is 2 (2%) more than in the initial data. Consequently, mature rats had no significant changes in the number of normal neurons during natural growth and development from 70 to 100 days of age. The neuron-glia ration in the control group of 70-day-old animals was 2.3. In the experimental group, after modeling traumatic brain injury, this ratio averaged 1.35.

Microscopic examination of the brain tissue of the animals of the experimental group showed an increase in the number of dystrophic-altered neurons in the affected hemisphere of the brain by 14 times already on the 1st day after the TBI and averaged 28,5. We should note an increase in the number of glial cells in the group of experimental animals of 70 days of age after TBI by an average of 8 (20%) compared with the control group (P<0.05).

In rat pups of the group of unlimited motor activity at 70 days of age, after modeling a traumatic brain injury, the number of intact neurons was 68 ± 2.7. This value was 27 (28%) less than in the control group (P<0.05). During the subsequent maintenance of these rat pups for 30 days the number of intact neurons increased to 82 ± 3.1, which is 14 (20%) more than the initial data and 15 (15%) less than in the control group (P<0.05). Consequently, mature control animals with TBI had no age-related decrease in heart rate during the next 30 days under unlimited motor activity (UMA) show a significant increase in the number of intact neurons. We should note a decrease in the number of degenerative and dystrophic neurons in comparison with the initial data by 8 (28%), as well as the preservation of the number of glial elements at the initial level.

In mature rat pups under enhanced motor mode at 70 days of age after the traumatic brain injury, the number of intact neurons was 66 ± 1.5, which is 19 (20%) less than in the control group (P<0.05). Histological examinations of the brain tissue revealed an increase in the number of damaged neurons by 13 times and glial cells by 8 (20%) compared to the control group (P<0.05). The neuron-glia ratio was 1.3. Dynamic swimming exercises during 30 days in these rat pups caused a significant increase in intact neurons and amounted to 91 ± 2.8, which was 25 (38%) more than the initial data, but 8 (8%) less than in the control group of animals (P<0.05). Enhanced motor mode (MST) by 100 days of age caused a decrease in the number of damaged neurons by 11 (41%) and an increase in glial elements by 5 (10%) compared with the initial data (P<0.05). The neuron-glia ratio was 1.7. Consequently, it can be argued that in mature animals that underwent a craniocerebral injury at 70 days of age, the performance of gradually increasing dynamic exercises in the form of swimming contributes to a significantly faster recovery of neurons, a decrease in degenerative-dystrophic neurons and an increase in the number of glial cells.

At 70 days of age, in mature rat pups with limited motor activity (LMA), the number of intact neurons was 67 ± 1.2, which is 28 (29%) less than in the control group (P<0.05). Microscopic examination of the brain tissue of animals with limited motor activity on the 1st day after TBI revealed an increase in the number of damaged neurons by 14 times, as well as an increase in glial cells by 8 (20%) (P<0.05). The neuron-glia ratio was 1.4. In the process of daily long-hour restriction of physical activity, by stretching and fixing the limbs on a special table for 30 days in these rat pups, the number of intact neurons in these rat pups was 78 ± 3.3, which is 11 (16%) more than the initial indicators, but 19 (20%) less than the indicators of the control group of animals of the same age. We should note that the number of damaged neurons by the end of the training process was 24 ± 3.2, which is 4 (14%) less than the initial indicators and 6 times more than the number of damaged neurons in animals of the control group of 100 days of age (P<0.05). One more thing to note is that the number of glial cells after 30-day training was approximately identical to the initial values and amounted to 48 ± 2.6, which was 9 (16%) less than in the control group. Thus, the subsequent limitation of motor activity (LMA) from 70 to 100 days of age in mature rat pups after traumatic brain injury caused an insignificant change in the number of investigated cellular structures. Therefore, it can be argued that the subsequent 30-day restriction of motor activity in mature animals with traumatic brain injury at 70 days of age inhibits the recovery of brain tissue.

In mature rat pups who perform isometric exercises (IE) at 70 days of age, the number of intact neurons was 69 ± 2.3, which is 26 (27%) less than in the control group (P<0.05). The number of damaged neurons and glial cells

roughly corresponded to the number of these cells in groups of animals exposed to other modes of motor activity. The performance of systematically increasing isometric exercises for 30 days led to an insignificant increase in the number of intact neurons, there were 73 ± 3.1 of them, which is 4 (6%) less than the initial data and 24 (25%) less than the control group at 100 days of age (P<0.05). Histological examinations revealed that the number of damaged neurons was 36 ± 4.1, which is 6 (20%) more than the initial data and 9 times more than in the control group of animals (P<0.05). We should also note a decrease in the number of glial cells at the end of the training process, their number was 43 ± 3.1, which is 5 (10%) less than the initial data and 14 (25%) less than the indicators of animals in the control group at 100 days of age (P<0.05). Consequently, the performance of isometric exercises after a traumatic brain injury leads to a significant deterioration in the compensatory and recovery processes. Thus, the results obtained indicate that modeling a traumatic brain injury at 70 days of age leads to a significant change in the number and ratio of the studied cellular structures. However, subsequent modes of motor activity for 30 days do not equally affect the recovery of brain tissue. The most favorable mode of physical activity for mature rats with traumatic brain injury is the performance of dynamic exercises in the form of systematic swimming. Limited motor activity and performance of isometric exercises after a traumatic brain injury significantly inhibit the physiological recovery of the cellular structures of the brain (Table 2).

Table 2: Morphometric changes in neurons and glial cells in mature rats after a traumatic brain injury subjected to various modes of motor activity.

Parameters	Motor modes	Value M ± m	
		Day 70 (after trepanation)	Day 100 (experiment)
Number of normal neurons	C (without trepanation)	95 ± 4.2	97 ± 3.4
	UMA	68 ± 2.7	82 ± 3.1
	MST	66 ± 1.5	91 ± 2.8
	LMA	67 ± 1.2	78 ± 3.3
	IE	69 ± 2.3	73 ± 3.1
Number of damaged neurons	C (without trepanation)	2 ± 0.4	4 ± 0.3
	UMA	29 ± 3.5	21 ± 2.7
	MST	27 ± 3.1	16 ± 1.3
	LMA	28 ± 2.3	24 ± 3.2
	IE	30 ± 3.4	36 ± 4.1
Number of glial elements	C (without trepanation)	41 ± 3.5	57 ± 3.9
	UMA	51 ± 4.3	50 ± 3.7
	MST	49 ± 2.7	54 ± 4.2
	LMA	47 ± 3.4	48 ± 2.6
	IE	48 ± 4.1	43 ± 3.1
Neuron-glia ratio	C (without trepanation)	2.3	1.7
	UMA	1.3	1.6
	MST	1.3	1.7
	LMA	1.4	1.6
	IE	1.4	1.7

Changes in neurons and glial cells in pre-senile rats exposed to different modes of physical activity after a traumatic brain injury

The number of intact neurons in control animals aged 180 days was 58 ± 2.6 (Table 3). During the natural vital activity of animals, this indicator gradually decreased and by the age of 210 days amounted to 51 ± 1.4, which is 7 (12%) less than the initial data (P<0.05). We should note that the number of damaged neurons, on the contrary, increased from 32 ± 3.7 to 39 ± 3.1, the difference was 7 (22%) (P<0.05). Another thing to note is the number of glial elements, which decreased from 14 ± 1.8 to 9 ± 1.1 (P<0.05). Consequently, during natural vital activity of rats from 180 to 210 days of age, there is a natural decrease in the number of intact neurons and glial cells, as well as an increase in the number of degenerative neurons.

Table 3: Morphometric changes in neurons and glial cells in pre-senile rats after a traumatic brain injury subjected to various modes of motor activity.

Parameters	Motor modes	Value M ± m	
		Day 180 (After trepanation)	Day 210 (Experiment)
Number of normal neurons	C (without trepanation)	58 ± 2.6	51 ± 1.4
	UMA	33 ± 2.1	22 ± 1.0
	MST	31 ± 1.8	39 ± 2.7
	LMA	30 ± 1.9	19 ± 1.2
	IE	29 ± 1.3	14 ± 1.1
Number of damaged neurons	C (without trepanation)	32 ± 3.7	39 ± 3.1
	UMA	53 ± 2.1	61 ± 4.2
	MST	51 ± 2.5	38 ± 2.7
	LMA	54 ± 3.2	69 ± 4.5
	IE	52 ± 2.9	76 ± 4.8
Number of glial elements	C (without trepanation)	14 ± 1.8	9 ± 1.1
	UMA	18 ± 2.1	13 ± 1.5
	MST	17 ± 1.5	10 ± 1.4
	LMA	19 ± 1.3	21 ± 2.7
	IE	17 ± 1.9	28 ± 3.2
Neuron-glia ratio	C (without trepanation)	4.1	5.6
	UMA	1.8	1.7
	MST	1.8	3.9
	LMA	1.6	0.9
	IE	1.7	0.5

In animals with unlimited motor activity at 180 days of age, after modeling a traumatic brain injury, the number of intact neurons was 33 ± 2.1. This value was 25 (43%) less than in the control animals of the same age (P<0.05). During the subsequent maintenance of these rat pups for 30 days the number of intact neurons decreased to 22 ± 1.0, which is 11 (33%) less than the initial data and 29 (57%) less than in the control group (P<0.05). Consequently, pre-senile experimental animals with TBI had no age-related decrease in heart rate during the next 30 days under unlimited motor activity (UMA) have a decrease in the number of intact neurons. We should note a decrease in the number of glial cells by 5 (28%), as well as an increase in the number of degenerative and dystrophic neurons in comparison with the initial data by 8 (15%). The neuron-glia ratio was 5.6, which is 1.5 more compared to the initial data. In our opinion, this is explained by more severe edema and subsequent foci of coagulation necrosis as a consequence of the traumatic brain injury against the background of age-related degenerative-dystrophic changes in the brain tissue.

In pre-senile animals under enhanced motor mode at 180 days of age after the traumatic brain injury, the number of intact neurons was 31 ± 1.8, which is 37 (47%) less than in the control group (P<0.05). Microscopic examinations revealed an increase in the number of damaged neurons by 19 (59%) and glial cells by 3 (21%) compared to the control group (P<0.05). The neuron-glia ratio was 1.8. Dynamic swimming exercises during 30 days in these rats caused a significant increase in intact neurons and amounted to 39 ± 2.7, which was 8 (26%) more than the initial data (P<0.05). Enhanced motor mode (MST) by 210 days of age caused a decrease in the number of degenerative neurons by 13 (25%) and a decrease in glial elements by 7 (41%) compared with the initial data (P<0.05). The neuron-glia ratio was 3.9. Consequently, it can be argued that in pre-senile animals that underwent a craniocerebral injury at 180 days of age, the performance of gradually increasing dynamic exercises in the form of swimming contributes to an increase in the number of normal neurons, and a decrease in the number of degenerative-dystrophic neurons and glial cells.

At 180 days of age, in rats with limited motor activity (LMA), the number of intact neurons was 30 ± 1.9, which is 28 (48%) less than in the control group (P<0.05). Histological examination of the brain tissue of animals with limited motor activity on the 1st day after TBI revealed an increase in the number of damaged neurons by 22 (69%), as well as an increase in glial cells by 5

(36%) (P<0.05). The neuron-glia ratio was 1.6. In the process of daily long-hour restriction of physical activity, by stretching and fixing the limbs on a special table for 30 days in these rat pups, the number of intact neurons in these rat pups was 19 ± 1.2, which is 11 (37%) less than the initial values, and by more than 2.5 times less than the indicators of the control group of animals (P<0.05). The number of damaged cells by the end of the training process was 69 ± 4.5, which is 15 (28%) more than the initial values and 30 (77%) more than in control animals (P <0.05). We should note that the number of glial cells was 21 ± 2.7, which is 2 (11%) more than the initial data, but 12 (133%) more than the values of the control group. The neuron-glia ratio was 0.9 by the end of the training process. Thus, the subsequent limitation of motor activity (LMA) from 180 to 210 days of age in pre-senile rats after traumatic brain injury caused a significant change in the number of investigated cellular structures. Therefore, it can be argued that the subsequent 30-day restriction of motor activity in these animals with traumatic brain injury at 180 days of age aggravates the traumatic changes in the brain tissue.

In pre-senile rats who perform isometric exercises (IE) at 180 days of age, the number of intact neurons was 29 ± 1.3, which is 2 times less than in the control group (P<0.05). The number of damaged neurons and glial cells roughly corresponded to the number of these cells in groups of animals exposed to other modes of motor activity. Starting from the age of 180 days after a traumatic brain injury, over the next 30 days, the animals were tightly fixed on the turntable and gradually accustomed to hanging upside down (antiorthostasis). The performance of systematically increasing isometric exercises for 30 days led to a significant decrease in the number of intact neurons, there were 14 ± 1.1 of them, which is 15 (51%) less than the initial data and by more than 3.5 times less than the control group at 210 days of age (P<0.05). Microscopic examinations revealed that the number of damaged neurons was 76 ± 4.8, which is 24 (46%) more than the initial data and 37 (95%) more than in the control group of animals (P<0.05). The number of glial cells by the end of the training process was 28 ± 3.2, which is 11 (65%) more than the initial data and more than 3 times more than the number of glial elements in animals of the control group at 210 days of age (P <0.05). Consequently, the performance of isometric exercises after a traumatic brain injury leads to a significant deterioration in the compensatory and recovery processes, inhibition of the restoration of cellular structures and their relationships in the brain tissue, and therefore inhibits recovery after a traumatic brain injury.

Thus, the results obtained indicate that, after a traumatic brain injury, the most favorable mode of physical activity for pre-senile rats is the performance of dynamic exercises in the form of systematic swimming. Limited motor activity inhibits the recovery of brain tissue, while performance of isometric exercises after a traumatic brain injury leads to a significantly worse recovery of the brain tissues [11-15].

CONCLUSION

During the first day after modelling of traumatic brain injury all age groups of experimental animals showed a decrease in the number of intact neurons, an increase in the number of degenerative neurons, as well as glial elements. This is since the main primary consequences of a brain injury are haemorrhages, acute death of neurons, damage to the blood-brain barrier. We also found that the group of animals with unlimited motor activity (UMA) after 30 days had no recovery of the parameters of cellular structures to the values of the control group. In our opinion, this is due to repeated (delayed) changes, which are considered a set of biomechanical, structural, and molecular changes resulting from primary damage. These include inflammation, excitotoxicity, and neurodegeneration. Reparative processes include neurogenesis, glycogenesis, and angiogenesis. We also found that injuries in old age are characterized by a long recovery period and a worse prognosis compared to injuries at an earlier age. This may be due to increased neuroinflammation and vascular permeability.

The most favourable mode of physical activity for rats of all age groups after a traumatic brain injury is the performance of dynamic exercises in the form of systematic swimming. We associate this feature with a decrease in the effect of secondary damaging factors, which in turn allows nerve cells to avoid death.

Limited motor activity and performance of isometric exercises after a traumatic brain injury significantly inhibit the recovery of the cellular structures of the brain tissue, which is associated with an increase in arterial and intracranial pressure, and therefore an increase in secondary changes.

SUMMARY

- During the first day after modelling of traumatic brain injury all age groups of experimental animals showed a decrease in the number of intact neurons, an increase in the number of degenerative neurons, as well as glial elements.
- The most favourable mode of physical activity for rats of all age groups is the performance of dynamic exercises in the form of systematic swimming.
- Limited motor activity and performance of isometric exercises after a traumatic brain injury significantly inhibit the recovery of the cellular structures of the brain tissue.

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