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The Nature of Destructive and Compensator-Restorative Processes in the Rat Heart Myocardium after a Traumatic Brain Injury Performing Various Physical Exercises

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Abstract

Traumatic brain injury (TBI) is one of the most common causes of disability and is an economic burden for society. The pathophysiological mechanisms of brain damage in TBI are based on the action of primary and secondary damaging factors. Secondary injuries are cascading biochemical, inflammatory, stress reactions, one of which is nonspecific systemic activation of hemostasis caused by hypoxia, an increase in free reactive oxygen species, a slowdown in blood flow, an increase in blood viscosity. The primary aim of this article is to examine the nature of destructive and compensator-restorative processes in the rat heart myocardium after a traumatic brain injury performing various physical exercises. To meet the aim of the study, some experiments have been carried out on outbred laboratory albino rats aged from 21 to 210 days. Meanwhile, the basis for the age periodization of rats is the anatomical and physiological characteristics of animals. The results of the experiments demonstrate that the group of animals with unlimited motor activity (UMA) after 30 days has no recovery of the parameters of cellular structures to the values of the control group. Furthermore, over the first day after modeling the traumatic brain injury, all age groups of experimental animals manifest a decrease in the number of intact neurons and an increase in the number of degenerative changed neurons. Consequently, it can be concluded most desirable mode of physical activity for rats of all age groups is the performance of dynamic exercises in the form of systematic swimming.

Keywords: Compensator-restorative processes; Heart myocardium; Physical exercises; Traumatic brain injury

Introduction

The acute period of severe traumatic brain injury (TBI) is characterized by the activation of vascular-platelet hemostasis [1,2], mainly in the microcirculation system [2-4]. All components of the vascular wall (endothelium, subendothelium, middle and outer membranes) are involved in maintaining hemostasiological homeostasis and interrelationships between the blood aggregate state regulation system and other functional systems of the body; however, the vascular endothelium deserves special attention [1,2,5,6]. Other research papers on the process of blood coagulation in TBI mainly study the characteristics of the damaged endothelium in the focus of brain damage. Given the fact that endothelial damage in critical conditions is nonspecific [1,4-8], it is logical to assume that similar endothelial cell disorders can be formed during brain injury and in the microvasculature of other organs. The literature has a lot of data that the plasmolemma of endothelial cells is not constant and can change its luminal relief under the influence of various factors. The factors of changes in the microrelief of the luminal surface of the endotheliocyte can be intravascular disorders of microcirculation, hypoxia, excessive accumulation of reactive oxygen species [6,9,10]. At the same time, a large number of irregularities appear on the endothelium in the form of micro-elevations and bay-like depressions, vacuatization and blistering, as well as cytoplasmic outgrowths and folds. This rearrangement of the cytoskeleton of endothelial cells is a significant factor in the activation of the vascular-platelet link of the hemostasis system [1,2,4,9,11]. To confirm the formation of disorders of blood microvessels outside the area of primary injury, we carried out a light and electron microscopic examination of the wall components and the contents of the lumen of capillaries, arterioles and venules of the left ventricular myocardium in rats after TBI. The objective of our research was to assess the structural changes in capillaries, arterioles, venules, intravascular and extravascular disorders in the myocardium of rats after TBI.

There is evidence that already in the first hours of the post-traumatic period a severe tissue (bioenergetic) hypoxia develops [1]. In this case, under reduced oxygen delivery to the cell, a complex multistep process develops, the final stage of which is a violation of the electron transport function of the terminal portion of the respiratory chain [2]. One of the manifestations of these disorders is the activation of free radical lipid peroxidation (LPO) [1]. This occurs against the background of a rapid depletion of the activity of the enzymatic and, especially, low-molecular antioxidant systems, which leads to the development of severe oxidative stress in the early stages after injury [3]. The formed excess amount of free radicals can cause oxidative modification of various structures of body cells: membrane phospholipids, DNA, cellular proteins, followed by a loss of their biological activity [4].

One of the promising ways of rehabilitation for TBI can be the systematic performance of various exercises.

Materials 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, proposed by V.I. Makhinko, V.N. Nikitin (1975). 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.

Within each age group, animals were divided into four subgroups. Each subgroup was subjected to its own established regimen of physical activity. Animals of the first subgroup (control) were kept in the usual conditions of the vivarium for 9-12 animals, under unlimited motor activity (UPA). The second subgroup was subjected to an enhanced motor mode. The animals systematically and forcibly performed stepwise muscle swimming training (MST) increasing in time. The third subgroup of animals was limited in motor activity, i.e. hypokinesia (LMA). Animals of this group were subjected to daily long-hour restriction of physical activity by stretching and fixing the limbs on a special table. The fourth subgroup of animals was systematically subjected to the isometric exercise (IE) regimen. On the turntable, animals with fixed limbs hung upside down daily. The execution time gradually increased, from 5 minutes on the first day and up to about 2 hours at the end of the experiment.

Traumatic brain injury was simulated under ether anesthesia in the animal in a prone position with its limbs fixed. The surgical stage of anesthesia 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 the 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, we took sections of the right and left ventricles of the heart.

Sections prepared on MS-2 sliding microtome (Tochmedpribor, Ukraine) were hematoxylin- and eosin-stained. The section thickness was 7 μm. Viewing and photographing of the finished sections was performed using Vizo 101 microvisor (Lomo, Russia). For electron microscope studies, the left ventricular myocardium tissue was placed in a 2.5% glutaraldehyde solution, followed by additional fixation with a 1% solution of osmic acid, dehydration in alcohols with increasing concentrations, and embedded in a mixture of epoxy resins (araldite and epon 812).

Ultrathin sections were prepared on an ultramicrotome (Leica Microsystems, Austria), viewed with Morgagni 268D electron microscope (FEI, USA), photographed with Mega View III video camera, and the structural changes of cardiomyocytes were studied.

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

Morphometric changes in the heart in immature rats exposed to different modes of physical activity after a traumatic brain injury

As Table 1 shows, during the histological examination of the heart tissue of animals in the control group, subject to unlimited motor activity, on the 30th day after TBI, the thickness of the posterior wall of the right ventricle averaged 0.9 ± 0.18 mm, the thickness of the cardiomyocyte in the outer longitudinal layer of the myocardium was 5.74 ± 0.186 μm, the minimum diameter of its nucleus was 1.65 ± 0.08 μm; in the left ventricle these parameters were as follows: 2.6 ± 1.04 mm, 6.84 ± 0.042 μm, and 2.64 ± 0.092 μm, respectively.

In immature rat pups subjected to enhanced motor mode at 51 days of age after traumatic brain injury and performing dynamic swimming exercises for 30 days, the thickness of the posterior wall of the right ventricle was 0.6 ± 0.12 mm, which is 0.3 mm less than in the control group of animals, the thickness of the cardiomyocyte in the outer longitudinal layer of the myocardium was 4.38 ± 0.127 μm, which is 1.36 μm less than in the group of unlimited motor activity; the diameter of the cardiomyocyte nucleus was 1.42 ± 0.06 μm, which is 0.23 μm less than the indicators of animals in the control group. The thickness of the posterior wall of the left ventricle of animals subject to increased motor activity (MST) was 2.2 ± 0.84 mm by the age of 51 days, which is 0.4 mm lower than in the control group of animals; there is also a smaller thickness of the cardiomyocyte 5.98 ± 0.027 μm versus 6.84 ± 0.042 μm in rats with unlimited motor activity; this tendency is the same for the diameter of the cardiomyocyte nucleus -2.43 ± 0.074 μm, which is 0.21 μm less than in the control group. 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 the myocardium, a decrease in degenerative-dystrophic changes in cardiomyocytes and their nuclei.

At 51 days of age, in immature rat pups with limited motor activity (LMA), the thickness of the posterior wall of the right ventricle was 1.3 ± 0.26 mm, which is 0.4 mm more than in the control group ($P<0.05$). Histological examination of the tissue of the right ventricle of animals subjected to limited motor activity on the 30th day after TBI showed that the thickness of the cardiomyocyte of the right ventricle was 6.89 ± 0.219 μm, which is 1.15 μm more than in animals with unlimited motor activity ($P<0.05$). The diameter of the cardiomyocyte nucleus of animals subjected to limited motor activity was 0.82 μm larger than in the control group. Microscopic examination of the left ventricle found the same difference. Thus, the wall thickness of the left ventricle in animals subjected to limited

motor activity was 2.8 ± 1.12 mm, which is 0.2 mm more than the thickness of the posterior wall of the left ventricle in animals of the control group. The thickness of the cardiomyocyte was 7.46 ± 0.074 μm, which is 0.62 μm more than the thickness of the left ventricular cardiomyocyte in animals with unlimited motor activity. The diameter of the cardiomyocyte nucleus of animals subjected to limited motor activity was 0.2 μm larger than in the control group.

Histological examination of the right ventricle in immature rat pups performing isometric exercises (IE) at 51 days of age, revealed the following parameters: the thickness of the posterior wall of the right ventricle averaged 1.7 ± 0.32 mm, the thickness of the cardiomyocyte in the outer longitudinal layer myocardium was 8.65 ± 0.312 μm, the minimum diameter of its nucleus was 2.94 ± 0.15 μm, which was 0.8 mm, 2.91 μm, and 1.29 μm more than in the control group, respectively ($P<0.05$). Microscopic examination revealed that the thickness of the posterior wall of the left ventricle of animals subjected to isometric exercises was 3.3 ± 1.25 mm, which is 0.7 mm more than in the control group of animals. Histological examination revealed that the thickness of the left ventricular cardiomyocyte was 2.1 μm more than the corresponding parameters of rats subjected to unlimited motor activity. The diameter of the cardiomyocyte nucleus of animals performing isometric exercises was 0.42 μm more than in the control group of animals. Consequently, the performance of isometric exercises after a traumatic brain injury leads to a significant thickening of the myocardium of the right and left ventricles.

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 cardiac tissues, while the performance of isometric exercises after a traumatic brain injury leads to a significant deterioration in compensatory and recovery processes [12].

Heart region	Investigated parameters	Motor modes			
		UMA (C)	MST	LMA	IE
Right ventricle	Posterior wall thickness (mm)	0.9 ± 0.18	0.6 ± 0.12	1.3 ± 0.26	1.7 ± 0.32
	Cardiomyocyte thickness (μm)	5.74 ± 0.186	4.38 ± 0.127	6.89 ± 0.218	8.65 ± 0.312
	Cardiomyocyte nucleus (μm)	1.65 ± 0.08	1.42 ± 0.06	2.47 ± 0.10	2.94 ± 0.15
Left ventricle	Posterior wall thickness (mm)	2.6 ± 1.04	2.2 ± 0.84	2.8 ± 1.12	3.3 ± 1.25
	Cardiomyocyte thickness (μm)	6.84 ± 0.042	5.98 ± 0.027	7.46 ± 0.074	7.94 ± 0.086
	Cardiomyocyte nucleus (μm)	2.64 ± 0.092	2.43 ± 0.074	2.84 ± 0.108	3.06 ± 0.114

Table 1: Morphometric changes in the heart of immature rats after a traumatic brain injury subjected to various modes of motor activity

Morphometric changes in the heart of mature rats exposed to different modes of physical activity after a traumatic brain injury

As Table 2 shows, during the histological examination of the heart tissue of mature animals in the control group, subject to unlimited motor activity, on the 30th day after TBI, the thickness of the posterior wall of the right ventricle averaged 1.4 ± 0.34 mm, the thickness of the cardiomyocyte in the outer longitudinal layer of the myocardium was 9.37 ± 0.384 μm, the minimum diameter of its nucleus was 2.72 ± 0.13 μm; in the left ventricle these parameters were as follows: 3.4 ± 1.21 mm, 7.88 ± 0.083 μm, and 2.93 ± 0.112 μm, respectively.

In mature rats subjected to enhanced motor mode at 70 days of age after traumatic brain injury and performing dynamic swimming exercises for 30 days, the thickness of the posterior wall of the right ventricle was 1.1 ± 0.27 mm, which is 0.3 mm less than in the control group of animals, the thickness of the cardiomyocyte in the outer longitudinal layer of the myocardium was 7.43 ± 0.237 μm, which is 1.94 μm less than in the group of unlimited motor activity; the diameter of the cardiomyocyte nucleus was 2.15 ± 0.11 μm, which is 0.57 μm less than the indicators of animals in the control group. The thickness of the posterior wall of the left ventricle of animals subject to increased motor activity (MST) was 3.2 ± 1.18 mm by the age of 51 days, which is 0.2 mm lower than in the control group of animals; there is also a smaller thickness of the cardiomyocyte 7.26 ± 0.058 μm versus 7.88 ± 0.083 μm in rats with unlimited motor activity; this tendency is the same for the diameter of the cardiomyocyte nucleus -2.86 ± 0.107 μm, which is 0.07 μm less than in the control group. 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 faster recovery of the myocardium, a decrease in degenerative-dystrophic changes in cardiomyocytes and their nuclei [13].

At 100 days of age, in immature rat pups with limited motor activity (LMA), the thickness of the posterior wall of the right ventricle was 2.1 ± 0.45 mm, which is 0.7 mm more than in the control group ($P<0.05$). Histological examination of the tissue of the right ventricle of animals subjected to limited motor activity on the 30th day after TBI showed that the thickness of the cardiomyocyte of the right ventricle was 13.96 ± 1.115 μm, which is 4.59 μm more than in animals with unlimited motor activity ($P<0.05$). The diameter of the cardiomyocyte nucleus of animals subjected to limited motor activity was 0.7 μm larger than in the control group. Microscopic examination of the left ventricle found the same difference. Thus, the wall thickness of the left ventricle in animals subjected to limited motor activity was 3.7 ± 1.46 mm, which is 0.3 mm more than the thickness of the posterior wall of the left ventricle in animals of the control group. The thickness of the cardiomyocyte was 8.33 ± 0.109 μm, which is 0.45 μm more than the thickness of the left ventricular cardiomyocyte in animals with unlimited motor activity. The diameter of the cardiomyocyte nucleus of animals subjected to limited motor activity was 0.06 μm larger than in the control group.

Histological examination of the right ventricle in mature rats performing isometric exercises (IE) at 100 days of age revealed the following parameters: the thickness of the posterior wall of the right ventricle averaged 2.8 ± 0.56 mm, the thickness of the cardiomyocyte in the outer longitudinal layer myocardium was 16.74 ± 1.218 μm, the minimum diameter of its nucleus was 3.96 ± 0.105 μm, which was 1.4 mm, 7.37 μm, and 1.24 μm more than in the control group, respectively (P<0.05). Microscopic examination revealed that the thickness of the posterior wall of the left ventricle of animals subjected to isometric exercises was 4.2 ± 1.84 mm, which is 0.8 mm more than in the control group of animals. Histological examination revealed that the thickness of the left ventricular cardiomyocyte was 1.24 μm more than the corresponding parameters of rats subjected to unlimited motor activity. The diameter of the cardiomyocyte nucleus of animals performing isometric exercises was 0.25 μm more than in the control group of animals. Consequently, the performance of isometric exercises after a traumatic brain injury leads to a significant thickening of the myocardium of the right and left ventricles.

Thus, the results obtained indicate that modeling a traumatic brain injury at 70 days of age leads to significant quantitative and qualitative changes 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 heart myocardium [14].

Heart region	Investigated parameters	Motor modes			
		UMA (C)	MST	LMA	IE
Right ventricle	Posterior wall thickness (mm)	1.4 ± 0.34	1.1 ± 0.27	2.1 ± 0.45	2.8 ± 0.56
	Cardiomyocyte thickness (μm)	9.37 ± 0.384	7.43 ± 0.237	13.96 ± 1.115	16.74 ± 1.218
	Cardiomyocyte nucleus (μm)	2.72 ± 0.13	2.15 ± 0.11	3.42 ± 0.077	3.96 ± 0.105
Left ventricle	Posterior wall thickness (mm)	3.4 ± 1.21	3.2 ± 1.18	3.7 ± 1.46	4.2 ± 1.84
	Cardiomyocyte thickness (μm)	7.88 ± 0.083	7.26 ± 0.058	8.33 ± 0.109	9.12 ± 0.127
	Cardiomyocyte nucleus (μm)	2.93 ± 0.112	2.86 ± 0.107	2.99 ± 0.100	3.18 ± 0.122

Table 2: Morphometric changes in the heart of mature rats after a traumatic brain injury subjected to various modes of motor activity.

Morphometric changes in the heart of presenile rats exposed to different modes of physical activity after a traumatic brain injury

As Table 3 shows, during the histological examination of the heart tissue of pre-senile animals in the control group, subject to unlimited motor activity, on the 30th day after TBI, the thickness of the posterior wall of the right ventricle averaged 2.6 ± 0.48 mm, the thickness of the cardiomyocyte in the outer longitudinal layer of the myocardium was 15.47 ± 1.108 μm, the minimum diameter of its nucleus was 3.84 ± 0.11 μm; in the left ventricle these parameters were as follows: 3.9 ± 1.53 mm, 8.76 ± 0.114 μm, and 3.06 ± 0.118 μm, respectively.

In pre-senile rats subjected to enhanced motor mode at 210 days of age after traumatic brain injury and performing dynamic swimming exercises for 30 days, the thickness of the posterior wall of the right ventricle was 2.3 ± 0.51 mm, which is 0.3 mm less than in the control group of animals, the thickness of the cardiomyocyte in the outer longitudinal layer of the myocardium was 13.75 ± 1.112 μm, which is 1.72 μm less than in the group of unlimited motor activity; the diameter of the cardiomyocyte nucleus was 3.43 ± 0.106 μm, which is 0.41 μm less than the indicators of animals in the control group. The thickness of the posterior wall of the left ventricle of animals subject to increased motor activity (MST) was 3.6 ± 1.42 mm by the age of 210 days, which is 0.3 mm lower than in the control group of animals. There is also a smaller thickness of the cardiomyocyte 7.94 ± 0.106 μm versus 8.76 ± 0.114 μm in rats with unlimited motor activity; this tendency is the same for the diameter of the cardiomyocyte nucleus -2.84 ± 0.106 μm, which is 0.22 μm less than in the control group. 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 a faster recovery of the myocardium, a decrease in degenerative-dystrophic changes in cardiomyocytes and their nuclei.

At 210 days of age, in immature rat pups with limited motor activity (LMA), the thickness of the posterior wall of the right ventricle was 3.4 ± 0.62 mm, which is 0.8 mm more than in the control group (P<0.05). Histological examination of the tissue of the right ventricle of animals subjected to limited motor activity on the 30th day after TBI showed that the thickness of the cardiomyocyte of the right ventricle was 17.86 ± 1.127 μm, which is 2.39 μm more than in animals with unlimited motor activity (P<0.05). The diameter of the cardiomyocyte nucleus of animals subjected to limited motor activity was 0.42 μm larger than in the control group. Microscopic examination of the left ventricle found the same difference. Thus, the wall thickness of the left ventricle in animals subjected to limited motor activity was 4.4 ± 1.76 mm, which is 0.5 mm more than the thickness of the posterior wall of the left ventricle in animals of the control group. The thickness of the cardiomyocyte was 9.34 ± 0.132 μm, which is 0.58 μm more than the thickness of the left ventricular cardiomyocyte in animals with unlimited motor activity. The diameter of the cardiomyocyte nucleus of animals subjected to limited motor activity was 0.15 μm larger than in the control group [13].

Histological examination of the right ventricle in pre-senile rats performing isometric exercises (IE) at 210 days of age revealed the following parameters: the thickness of the posterior wall of the right ventricle averaged 3.8 ± 0.74 mm, the thickness of the cardiomyocyte in the outer longitudinal layer myocardium was 19.46 ± 1.132 μm, the minimum diameter of its nucleus was 5.12 ± 0.136 μm, which was 1.2 mm, 3.99 μm, and 1.28 μm more than in the control group,

respectively (P<0.05). Microscopic examination revealed that the thickness of the posterior wall of the left ventricle of animals subjected to isometric exercises was 5.1 ± 1.84 mm, which is 1.2 mm more than in the control group of animals. Histological examination revealed that the thickness of the left ventricular cardiomyocyte was 1.11 μm more than the corresponding parameters of rats subjected to unlimited motor activity. The diameter of the cardiomyocyte nucleus of animals performing isometric exercises was 0.68 μm more than in the control group of animals. Consequently, the performance of isometric exercises after a traumatic brain injury leads to a significant thickening of the myocardium of the right and left ventricles.

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 the performance of isometric exercises after a traumatic brain injury leads to a significantly worse recovery of the brain tissues [15].

Heart region	Investigated parameters	Motor modes			
		UMA (C)	MST	LMA	IE
Right ventricle	Posterior wall thickness (mm)	2.6 ± 0.48	2.3 ± 0.51	3.4 ± 0.62	3.8 ± 0.74
	Cardiomyocyte thickness (μm)	15.47 ± 1.108	13.75 ± 1.112	17.86 ± 1.127	19.46 ± 1.132
	Cardiomyocyte nucleus (μm)	3.84 ± 0.1 10	3.43 ± 0.106	4.26 ± 0.121	5.12 ± 0.136
Left ventricle	Posterior wall thickness (mm)	3.9 ± 1.53	3.6 ± 1.42	4.4 ± 1.76	5.1 ± 1.84
	Cardiomyocyte thickness (μm)	8.76 ± 0.114	7.94 ± 0.106	9.34 ± 0.132	9.87 ± 0.124
	Cardiomyocyte nucleus (μm)	3.06 ± 0.118	2.84 ± 0.102	3.21 ± 0.127	3.74 ± 0.132

Table 3: Morphometric changes in the heart of pre-senile rats after a traumatic brain injury subjected to various modes of motor activity.

Discussion

During the first day after modeling the 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. This is because the main primary consequences of a brain injury are hemorrhages, acute death of neurons, and 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, gliogenesis, 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.

Summary

During the first day after modeling the 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 favorable 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.

Conclusion

The most favorable 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.

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