epileptogenesis. In assessing the safety and effectiveness of new methods of treating epilepsy, a combination of models of various types is used, which allows us to study their effect on various aspects of icto- and / or epileptogenesis and to predict the type of seizures on which this therapy may be most effective.

The idea of the etiology of epilepsy is based on the knowledge of a number of exogenous and endogenous factors that play a certain role in the origin of the disease. There are three types of inclination: acquired, congenital and hereditary. Today, there are many experimental models designed to study the mechanisms of epileptogenesis and increased convulsive readiness - pharmacological, based on chemical or electrical kindling, as well as studies on animals genetically predisposed to certain forms of epilepsy. Most often pilocarpine, kainate, pentylenetetrazole models are referred to, as well as maximum electroshock and kindling. Each model allows you to study the mechanisms of generation and development of both pathological and normal synchronization in the brain from various angles, to understand the structural and functional interaction of various regions of the brain. In addition, tests on the effectiveness of existing antiepileptic drugs are made, and new treatments for various forms of epilepsy are suggested. Each model presents some advantages and disadvantages, but the ideal model is still not found. At present it is chosen depending on the design and purpose-target of the study. It is on experimental models that it is possible to study the main "targets" and the spectrum of effects of basic and new antiepileptic drugs, dose selection and study of possible drug combinations.

Despite advances in epileptology and numerous clinical, neurophysiological, biochemical, morphological studies, epilepsy still remains an incompletely studied disease with complex diagnostics, drug selection, and a low possibility of predicting the effectiveness and safety of therapy for a particular patient. This circumstance determines the relevance of studying pharmacogenetic aspects of epilepsy.

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THE CONDITION OF THE LIPID PEROXIDE OXIDATION SYSTEM AND ANTIOXIDANT RAT LIVER PROTECTION UNDER LASER RADIATION

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Laser irradiation is widely used in the treatment of hepatobiliary system diseases, as well as other organs and systems. Its influence on the state of the microcirculatory tract, metabolic and redox processes in tissues is indicated. The possibility of peroxide homeostasis correction in pathology of diverse etiology with a high level of lipid peroxide oxidation (LPO) is noted.

One of the strongest and most universal protective enzymatic regulatory systems of LPO is the glutathione system, which participates in numerous metabolic reactions and has a pronounced antioxidant effect. About 90% of all circulating glutathione under physiological conditions is provided by the liver, which is the main organ of its synthesis in mammals.

The objective of this study was to investigate the effect of laser radiation on the state of POL-AOP system in the liver of rats.

The study was performed on 36 white nonlinear male rats weighing 120-150 g, which were kept on a regular vivarium diet. Laser irradiation was conducted through pre-shaved skin on the liver for 60 s on LGN-207-A device (λ = 632.8 nm, beam diameter 0.3 cm, power 1.3 mWt) for 10, (group 1), 20 (group 2) and 30 (group 3) days. Decapitation of rats was performed under ether anaesthesia at the end of the irradiation course, 10, 20 and 30 days after the end of the laser irradiation course. The state of LPO was assessed by the content of its primary - diene conjugates (DC) and secondary - malonic dialdehyde (MDA) products. The content of reduced glutathione (RG), glutathione peroxidase (GPO), superoxide dismutase (SOD) activity, and catalase (CT) were determined. Protein was determined by Lowry system. The research results were processed according to Student's criterion.

According to the results of studies, the content of DC after laser irradiation differed a little from the control in the 1st and 2nd groups and increased in the 3rd group by 27%. The dynamics of

changes were fluctuating in nature and did not return to normal position until the 30th day: 67% and 87% in the 2nd and 3rd groups respectively, 130% - in the 1st group. There was a decrease in the content of MDA in the 1st group, 24% increase in the 3rd experimental group. In group 2, MDA content differed a little from the control. In dynamics MDA content in the 1st group increased, in the 2nd and 3rdgroups changes were fluctuating. In 30 days, MDA content in all experimental groups remained below the control values.

Laser irradiation in all the experimental groups led to decrease in the activity of SOD, which did not return to normal level 30 days later. Ten days of laser irradiation caused decrease in catalase and GPO activity and increase in GSH content. Twenty and thirty days of irradiation led to increase in catalase activity and a decrease in the content of reduced glutathione. The dynamics of change were fluctuating. By day 30, catalase and GPO activity were higher than those of the controls in group 1 and decreased in groups 2 and 3. The content of reduced glutathione decreased in all the experimental groups.

Thus, laser radiation affects the redox state of the liver of rats, the changes are fluctuating in dynamics and depend on the duration of laser radiation.

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THE MECHANISM OF BIOCHEMICAL CHANGES IN THE BRAIN CELLS AFTER ISCHEMIA-REPERFUSION ON THE DEVELOPMENT OF GLIOMA

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Glioma is a heterogeneous group of brain tumors of neuroectodermal origin.

The relevance of our research is in theoretical studying of the interrelation between brain ischemia-reperfusion syndrome (BIRS) and the development of glioma in patients. It may help to predict the course of the disease and to determine ways to prevent the growth of brain tumors as well as their treatment. In addition, our research may help to establish the methods of the correct management of the post-ischemic period.

According to statistics, about five thousand people find out that they have glioma in Ukraine annually. Every year, this disease takes the lives of near 2 thousand Ukrainians. In the world, the incidence of different types of this tumor is about 15 cases per 100 000 of the population.

Yen-Tsung Huang and al. in their research "Genotype-based gene signature of glioma risk" established that glioma develops from the glia cells of the white brain matter. The main cause of these changes is genetic abnormalities. In most cases, there are mutations of the genes TP53 and BCL2 that encode p53 and Bcl-2 respectively. The protein p53 is the antioncogene, and the protein Bcl-2 is the protooncogene. Bcl-2 is involved in the regulation of cell death by inhibiting apoptosis. The tumor suppressor p53 is involved in the induction of apoptosis. The mistakes of regulation of Bcl-2 play the main role in the malignant transformation of tissues. The damage of the TP53 gene “turns it off”, so uncontrolled cell division begins causing the development of tumors.

It was experimentally determined (Kmet T. I., Tkachuk S. S. “Dynamics of changes in the morphofunctional state of p53-positive cells of the cortex of the temporal lobe of rat cerebral hemispheres under the influence of carotid ischemia-reperfusion”), that bilateral BIRS reduces the percentage of p53-positive gliocytes in the late post-ischemic period (more than 30%), due to that, the concentration of p53 in glia cells extremely decreases.

In the work of Ibragimov U. K. “Morphological changes in brain tissues during the experimental ischemia-reperfusion” has been found that after six hours of reperfusion the reaction of rosette formation of monoclonal Bcl-2 gliocytes begins. With time there are manifested the chaotic arrangement of a large number of Bcl-2 cells, which increases the likelihood of the formation of glioma with the reduced density of p53-positive gliocytes.

Therefore, the BIRS can provoke the development of glioma in the post-ischemic period by reducing the concentration of p53 and increasing Bcl-2 in glia cells of the white brain matter.