its derivate: RGPU−149 (47,6 mg/kg), RGPU−150 (49,2 mg/kg) and RGPU−151 (48,1 mg/kg). For phenibut derivates the doze is 1/10 from LD_{50}.

The Results of the study are presented in table.

Table 1. The influence of phenibut and its derivate at RHDT forming in the conditions of the experimental immunesuppression

Animal groups	Control 1: (phys.solution): n = 10	Control 2: CPH n = 10	Experimental I: PHEN1BUT+ CPh n = 10	Experimental 2: RGPU−149+ CPh n = 10	Experimental 3: RGPU−150+ CPh n = 10	Experimental 4: RGPU−151+ CPh n = 10
Index RHDT, M ± m, %	*11,4 ± 0,5*	*8,2 ± 0,6***	*19,7 ± 2,7**	*21,5 ± 1,5****	*31,7 ± 2,4****	*26,5 ± 4,4****

In the course of the carried out tests it was fixed that the single inside intraperitoneal leading cyclophosphamid is conducire to the suppression of the cell reaction of the delayed type (reaction index below control № 1 in 1,5 times). The leading of phenibut and its derivates to animals with the immunesuppression model is accomponing with the stimulating action with the regard to the cell section of the immune reactivity, it reveals itself with the increase of the reaction index RHDT more than 50% not only by comparison with the animals from the control group № 1 ($p<0,05$), but more than 40% with respect to the exponents in the mice groups, receiving «placebo» ($p<0,05$).

So, phenibut and its derivates with the laboratory codes RGPU−149. RGPU−150 and RGPU−151 are removing pharmacoinduced immune deficiency, it is evidence of the immunecorrecting qualities presence. The present conclusion allows to regard the learning substances as the perspective ones as the means of the correction of the neuroimmune pathology.

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INCREASING OF PHAGOCYTES FREE RADICALS ACTIVITY UNDER THE INFLUENCE OF MAGNETOTHERAPY AMONG PATIENTS WITH ISCHEMIC HEART DISEASE

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Nowadays the Ischemic Heart Disease (IHD) gets younger thus this disease used to appear among 50-65-years old people but now it appears among 35-years old people. It could be caused by negative ecological factors, which may lead to both increasing and decreasing free radicals aggression of human blood phagocytes. One of the main parts of heart destruction is an intensification of cardiac hystiocytes' lipids free radicals oxidation, which correlates with its level of destruction and with the level of lipids peroxidation products and antioxidants concentration in the blood plasma (Abramova J.I., Vladimirov U.A.). That is why Red-Ox balance is a very important characteristic for diagnostic and correction. One of the most popular types of correction of different diseases is common magnetic therapy (CMT).

Therefore effectiveness of magnetic treatment was assessed by chemiluminescent analyze. The investigation includes examination of blood samples which were taken from people with IHD both gender (male = 56 and female = 26) age was from 43 to 66 years old. The magnetic field was made by special apparatus "Magnetoturbotron-2" (frequency is equal 10 Hz, intensiveness equals 1 milli Tesla). Course of treatment includes ten everyday and 20-minutes treatments. There were assessed different medical indexes such as quantity of leucocytes and phagocytes. The functional phagocyte activity was estimated by biochemiluminometer.

The investigations show that magnetic field did not increase leukocytosis and did not suppressed phagocytic function. But functional activity of leucocytes have increased and exceeded normal level among 61% of patient moreover it increased during the magnetic treatment. As the result, quantity of patient with normal reactivity of phagocyte decreased. Such kind type of chemiluminescet corresponds to ineffective phagocytosis when reactive types of oxygen are generated out of cells it can be risk of peroxide destruction of the nearest tissues.

Thus magnetic field with the level of magnetic induction 1 milliTesla suitable only for the forming local stress-reaction, but it is not universal strategy of treatment such dangerous diseases as ischemic heart disease. That is why it is necessary to investigate more suitable dose of magnetic induction with the help of

chemiluminescent analyze which is very sensitive to the blood free radicals level.

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MOLECULAR-GENETIC PRE-CONDITIONS FOR THROMBOSIS DEVELOPMENT

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One of the most important tasks of medical practice in modern society is an early detection of thrombophilic states, as various forms of thrombophilia are the origin causes of such severe diseases as infarctions, insults, and also the cause of operative intervention, pregnancy and inflammation complications resulting in disability and lethal outcomes.

Arterial and venous thromboses development risk can both be connected with the effect of acquired risk factors (operative therapy, oncologic and cardiovascular diseases, atherosclerosis, inflammation, pregnancy, stresses, etc.) and be of hereditary nature. According to modern ideas the thrombotic complications at cardiovascular diseases in 50-65% of the cases (on various authors' evidence) are connected with the defects of the genes controlling the hemostasis system components.

The **research purpose** is to find out the hemostasis system genetic mutations occurrence frequency, endothelial factors and define the significance of analysis for the diagnosis, purposeful pathogenetic therapy choice in the patients with thromboses of various localization vessels.

Materials and methods: The examination on the mutations in the hemostasis system, folate cycle, the genes controlling the vascular wall state and drug metabolism in the liver, warfarin (CYP2C9) in particular, was carried out for the purpose to estimate the predilection to the thrombophilia development and to define individual sensibility to warfarin.

The material for the molecular-genetic analysis was venous blood taken into plastic tubes with EDTA. For the hemostasis system investigation the tubes with citrate were used. The definition of allelic variants of the genes investigated was carried out by the polymerase chain reaction method with the following analysis of restriction fragment length polymorphism (PCR/RFLP).

48 patients aged from 24 to 59 years old with the diagnoses myocardial infarction, ischemic insult, and lower limbs deep venous thrombosis were examined. The first group (29 patients) was formed by the persons, who the treatment was prescribed after the investigation of the hemostasis system functional state and simultaneous molecular-genetic analysis; the 2nd group (19 persons) was made up of the patients appealed for the consultation from other medical and prophylactic institutions with non-effective therapy of the present disease and difficulties in drug dosage of the indirect anticoagulant – warfarin. For the warfarin sensibility estimation two quantity indexes were used: induction phase duration – terms of the INR therapeutic level achievement (number of days) and weekly warfarin dosage (mg), which was required for maintaining of the achieved effect.

Results: The heterozygous mutation of the folate cycle enzyme methylenetetrahydrofolate reductase (MTHFR), the polymophous substitution 677C->T (A223V) was registered by us in 56% of the cases; the homozygous mutation was registered in 5 patients. In 21% of the examinees the methionine synthase (MTR) gene mutation, the polymorphous substitution 2756A->G(D919G) was detected. These mutations lead to the substitution of amino acid residues in the polypeptide chain of enzymes, that decreases their specific activity. One of the manifestations of the MTHFR and MTR deficit is an abundant accumulation of homocysteine in blood that results in the negative influence on the endothelium, disturbing the cell permeability and decreasing the nitrogen oxide production. The hyperhomocysteinemia was detected in 21,6% of the cases, in three patients among them the homocysteine level exceeding the normal one 1,8 times.

From the number of the examinees with repeated infarctions and/or ischemic insult, deep venous thrombosis there were polymorphous variants of the genes controlling the thrombocytic hemostasis registered. In 24% of the cases the polymorphous locus containing variable number of tandem repeats (VNTR) of the thrombocytic glycoprotein gene 1b(GP1ba). In 32% of the cases the polymorphous substitution 1565T→C (Leu33Pro) of the IIIa thrombocytic glycoprotein gene (integrin beta 3), and in 18% - the polymorphous substitution 807C→T of the Ia thrombocytic glycoprotein gene (Gp1a-integrin - alpha -2).

The FII prothrombin gene mutation connected with the substitution of G with A (20210 G→A) and leading to the protein and prothrombin level increase in blood, was registered in our patients in 26% of the cases.

The most severe thrombophilia cases were characterized by the **combined** polymorphous variants of the genes of the thrombocyitc hemostasis, folate cycle, coagulative part of hemostasis and polymorphism of the genes of the endothelial part – the plas-