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MORPHOFUNCTIONAL CONSEQUENCES OF EXPERIMENTAL CHRONIC ADRENALINE-INDUCED AND PSYCHO-EMOTIONAL STRESS IN RATS

МОРФОФУНКЦІОНАЛЬНІ НАСЛІДКИ ЕКСПЕРИМЕНТАЛЬНОГО ХРОНІЧНОГО АДРЕНАЛІН-ІНДУКОВАНОГО ТА ПСИХОЕМОЦІЙНОГО СТРЕСУ У ЩУРІВ

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Activation of the sympathoadrenal system is a leading mechanism in the development of systemic disturbances under chronic stress conditions: excessive catecholamine exposure is accompanied by dysregulation of the hypothalamic – pituitary – adrenal (HPA) axis, development of oxidative stress, and endothelial dysfunction [1]. Prolonged stimulation of α- and β-adrenergic receptors disrupts vascular tone, regional blood flow, and

microcirculation, creating prerequisites for ischemic tissue injury, while excessive generation of reactive oxygen species, depletion of the antioxidant system, and impairment of redox homeostasis potentiate structural alterations of cellular membranes and intercellular contacts [2]. The vascular endothelium is one of the primary targets of such damage: permeability increases, NO-dependent regulation is impaired, and proinflammatory mechanisms are activated [1]; vasodilatory capacity decreases, vascular resistance increases, and microthrombosis develops. Simultaneously, activation of the complement system, changes in adhesive properties of the vascular wall, and remodeling of the glycocalyx further aggravate microcirculatory disturbances [3].

Involution of immunocompetent organs and structural remodeling of the adrenal glands are considered characteristic morphological manifestations of chronic neuroendocrine activation [4]. Elevated glucocorticoid levels induce lymphocyte apoptosis, reduction of thymus and spleen mass, and suppression of immune cell proliferative activity. At the same time, adrenal gland hypertrophy reflects a compensatory response to prolonged functional overload, which may eventually progress to a stage of exhaustion [5].

Materials and methods. The study was conducted on adult rats divided into four experimental groups: control, PTSD-like state, adrenaline-induced stress (AIS), and combined modeling of PTSD-like state and AIS. The PTSD-like condition was reproduced through multifactorial exposure to stress-related sensory stimuli [6]. AIS was induced by chronic systemic administration of adrenaline hydrotartrate solution. After 6 weeks, animals were euthanized, organs were isolated, and adrenal glands and thymus were weighed with subsequent calculation of organ indices (organ weight / body weight × 1000). The condition of small intestinal vessels was examined ex situ using light microscopy to assess signs of vascular dysfunction and microcirculatory disturbances.

Results. After 6 weeks of modeling the PTSD-like state, a significant decrease in body weight by 19.8% ($p<0.01$) was observed compared with the control group. Simultaneously, adrenal gland mass increased by 58.4% ($p<0.01$), whereas thymus mass decreased by 40.2% ($p<0.01$). The adrenal gland organ index increased by 98.6% ($p<0.001$), while the thymus organ index decreased by 27% ($p<0.01$) relative to control values. These changes reflect a neuroendocrine response to prolonged stress exposure and are consistent with literature data regarding HPA axis activation and involution of immunocompetent organs under chronic stress conditions [4].

In the group with adrenaline-induced stress combined with emotional PTSD exposure, after 6 weeks body weight decreased by 16.5% ($p<0.05$), adrenal gland mass by 21.5% ($p<0.01$), and thymus mass by 17.4% ($p<0.01$). Comparison of organ indices demonstrated that the body weight-

to-adrenal gland mass index decreased by 56.5% relative to the AIS group, while the body weight-to-thymus mass index decreased by 15.2% and 21.5% relative to control values. These findings indicate enhanced maladaptive processes under combined pathological exposure, consistent with the concept of adaptive exhaustion during prolonged catecholamine stimulation [1, 2].

Morphological examination of small intestinal vessels revealed pronounced structural alterations of the vascular wall, including endothelial damage and disorganization, irregular lumen with alternating dilation and spasm, stasis, and congestive blood filling. Microcirculatory heterogeneity and changes in vascular tone of different calibers were observed, corresponding to manifestations of endothelial dysfunction under prolonged stress and excessive adrenergic stimulation [3]. In animals with the PTSD-like state, these changes were systemic, while in the combined AIS model they were more intense and widespread, indicating potentiation of vascular dysfunction under combined pathological influence [1, 2].

Thus, the combination of AIS with chronic psychoemotional stress leads to the formation of a systemic morphofunctional syndrome characterized by neuroendocrine activation, immune remodeling, and vascular dysfunction. Morphological data confirm the development of systemic vascular maladaptation, more pronounced under combined exposure. Organ indices may serve as quantitative markers of stress-induced systemic alterations.

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HOLOGRAPHIC MODELING AND LANDSCAPE IMAGINATIVE KINESIOTHERAPY IN CORONARY ARTERY DISEASE, ARTERIAL HYPERTENSION, AXIETY-DEPRESSIVE DISORDERS REHABILITATION

ГОЛОГРАФІЧНЕ МОДЕЛЮВАННЯ ТА ЛАНДШАФТНА ОБРАЗНА КІНЕЗІОТЕРАПІЯ В РЕАБІЛІТАЦІЇ ПРИ ІШЕМІЧНІЙ ХВОРОБІ СЕРЦЯ, АРТЕРІАЛЬНІЙ ГІПЕРТЕНЗІЇ, ТРИВОЖНО-ДЕПРЕСИВНИХ РОЗЛАДАХ

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Patients with coronary artery disease, arterial hypertension and anxiety-depressive disorders are characterized by high psychoemotional exertion, wich significantly complicates their rehabilitation and requires additional correction, in particular pharmacological [1, 2, 3, 4]. For basic stabilization and effective rehabilitation of this patients and minimization of therapeutic