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NEUROPLASTICITY ASPECTS OF THE TRAUMATIC BRAIN INJURY CONSECUENCES

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Introduction. Pathogenetic cascade damage in traumatic braim imjury (TBI) has been well studied , but the possibility of adaptation and repair processes is not well understood. It is known that endogenous protective activity provide: neyrotrofichnost , neuroprotection , neuroplasticity and angiogenesis.

Aim. To study the neuroplasticity aspects of the traumatic brain injury consequences.

Results. It is known, that neuroplasticity is a collection of various remodeling processes of synaptic connections, designed to optimize the functioning of neuronal networks. Neuroplasticity is the process of biological adaptation script associated with the structure and the functional reorganization of the central nervous system. It begins at the cellular level, and neuronal processes , including synaptic transmission, and reconstitution of neuronal functioning. Through the processes of neuroplasticity nervous tissue capable of resuming its function by qualitative and quantitative changes perekroek - neuronal and glial cells. Activation is accompanied by stimulation of neuroplasticity expression of certain genes, biosynthesis of receptor molecules and ion channels , synaptic proteins filamentous cytoskeletal neurotransmitter components of synaptic membranes, intercellular adhesion molecules, immature form of contacts, their aging, activation, hypertrophy and reorganization of active synapses . The main principle of neuroplasticity is the phenomenon of synaptic sprouting: the brain in the process of unloading and creating of connections between neurons. Distinguish between fast and slow neuroplasticity. Fast neuroplasticity occurs in acute stress situations. It is based on the activation of the cerebral cortex is not involved previously horizontal connections, as well as modulation of synaptic transmission. After a traumatic head injury observed neuroplasticity occurring during recovery of lost functions after damage nerve tissue structures. Local damage to brain tissue lead to the activation of reactive and reparative mechanisms of neuroplasticity. Also TBI may occur the pathological neuroplasticity, in which there are new false connections, which are not in the normal conditions. Under the influence of pathological neuroplasticity activity increases activity of pathological functional systems that are becoming resistant to various, including medication. Pathological neuroplasticity contributes to pathological excitation generators. From this position can be attributed to the pathogenesis of many forms of epilepsy , including post-traumatic epilepsy.

Conclusion. TBI occurs the activation of neuroplasticity, rigid plastic response surviving neurons in the affected area, the formation of new interneuronal communication comes restructuring similar to neuronal function, not previously involved and located at a distance from the site of injury.