

Abstract

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Title of diploma thesis: Trauma and pathophysiological of brain damage

Form: Master Thesis

Name of University: Charles University in Prague

Faculty of Pharmacy in Hradec Králové

Department of Biological and Medical Sciences

Field: Pharmacy

Background:

Work in the first part describes the construction of the nervous and vascular system at key points which relate to the topic. Focuses on basic functional principles of both systems, which together are both anatomically and functionally linked.

In its special section then discusses his own brain injury. Includes incidence, causes and mechanisms of its formation, classify its severity. Describes the pathophysiological processes in the brain injury that are the focus of today's scientific research.

Findings and conclusion:

Based on the work of our search, we concluded that the pathophysiological picture of cerebral tissue damage involves many, mutually interlocking mechanisms. We found that the toxic effect of excitatory amino acids leads to a massive increase in intracellular calcium concentration. The resulting stepped-pathological activation of many enzymes and inhibition of mitochondrial function. Damage to nerve cells is also caused by free radicals that cause damage to cell membranes, proteins and molecular genetic information. The latter intervention radicals stimulates apoptotic process, multi-level mechanism for accelerating cell lysis. We also found out that in the pathophysiology of brain damage plays an important role as an inflammatory process involving the action of immunocompetent cells and their released mediators and brain edema deteriorating terrain damaged tissue.

Swelling participates in the creation of the ischemic state in the brain, which is necessary due to increased anaerobic metabolism and lactic acidosis. The course of brain damage also complicates hyponatremia. While watching the spectrum of molecules circulating in the blood and cerebrospinal fluid, we found also a number of markers, which determination may be beneficial in terms of prognosis of brain damage.

The above facts pathobiochemical extended view of the mechanisms of secondary damage. Knowing this versatility also extends the therapeutic possibilities of intervention. The effect of these complex mechanisms can mitigate the consequences, which induces primary brain damage.

Key words: pathophysiology of brain trauma, severe traumatic brain.