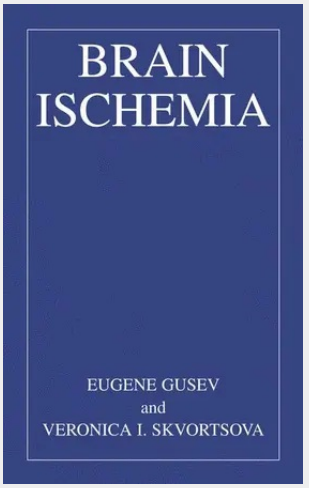
Gusev / Skvortsova

Brain Ischemia



Ischemia is localized tissue anemia due to obstruction of the inflow of arterial blood, thus brain ischemia is the condition where insufficient blood is delivered to the brain. Many physiological processes occurring in the brain critically depend on the state of its energy metabolism. The state of brain energy metabolism in turn depends on the delivery of oxygen and glucose to the brain via the bloodstream. Although it comprises only 2% of the total body weight, the human brain consumes 20-25% of the oxygen and up to 70% of the free glucose taken in by the body. The brain respires more intensively than any other organ of the body. The intensity of oxygen consumption by cortical brain tissue much exceeds the demands of other tissues (5.43 mmol 02/g per h versus 3.06 and 4.02 mmol for heart at rest and intensively working, respectively, 2.4 mmol for kidneys, and 1.8 mmol for liver). Oxidative phosphorylation in mitochondria generates 95% of the adenosine triphosphate (ATP) that is formed in the brain. Thus, it is clear why insufficiency of oxygen delivery to brain cells adversely affects brain function. Glucose is the main energy-providing substrate in the brain. The basic pathway of its metabolism in neural tissue is aerobic glycolysis.

This monograph highlights modern concepts of brain ischemia and strategies of neuroprotective therapy. The first part of the book is devoted to mechanisms of ischemic brain damage. The authors present the results of their own clinical and experimental studies conducted in the last two decades, as well as the achievements of native and foreign neurological researches that open a new stage in understanding how reversible changes of blood flow and metabolism are transformed into a permanent morphological lesion, i.e. brain infarction. The most important advances in areas of ischemic energy failure, main mechanisms of the glutamate-calcium cascade, influence of metabolic acidosis on ischemic damage, delayed neuronal death connected with microglial activation, local inflammation, autoimmune reactions, trophic dysfunction, and apoptosis, as well as of reaction of the stress-mediating endocrine system to focal brain ischemia are shown in animals and humans, and the relevant literature is cited and critiqued. The book also explores topics that recently have experienced substantial growth, such as gene expression and subsequent molecular events in response to acute brain ischemia, connected with both tissue damage and reparation. The authors demonstrate not only universal features of the process of brain ischemia, but also individual peculiarities of its course in patients and analyse their reasons. The modern strategies of neuroprotective therapy are disclosed in the second part of the book. The review of main neuroprotectors' studies performed under both experimental (in vitro and in vivo) and clinical conditions, as well as results of the authors' own investigations of original Russian neuroprotectors are presented. The book's balanced demonstration of progress in the field of fundamental researches in patients with ischemic stroke and in experimental models of acute brain ischemia is valuable for clinicians (neurologists, cardiologists, neurosurgeons), as well as for basic scientists and research trainees.

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