the brain tissue. It is considered that activation of the microglia results in the changes of its morphology, migration and proliferation.

The results of our studies show that in the remote period after the incident of total ischemia of brain the number of perivascular phagocytes and microglial cells increases. Degenerative changes in neurons as well as interrupted blood-brain barrier result in mobilization of defense system represented by active microglia and perivascular phagocytes.

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**A quantitative study on morphological types of microglia and astroglia in the human fetal mesencephalon**

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Quantitative changes associated with the differentiation of microglial and astroglial cells in fetal mesencephalon between 8 and 22 week of gestation (GW) were studied. Using lectin (RCA-1) labelling, two morphological types of RCA-1 positive cells, ameboid microglia (AM) and ramified microglia (RM) were identified on the basis of the cell body shape and the configuration of cytoplasmic processes. Astrocytes (AS) were identified by immunocytochemical labelling of GFAP. Measurements of microglial and astroglial cells were carried out in mesencephalon tectum and tegmentum under the microscope in sequential segments corresponding to equal sized non-overlapping areas. The quantitative data about number and percentage distribution of AM, RM and AS from each of the grid-quartiles were analyzed. The study revealed a time-sequence of appearance, characteristic pattern of distribution along perpendicular and longitudinal axes originating from aqueduct and changes in percentage distribution of both types of microglial cells and astrocytes.

Ameboid type of microglia was already present in 8 GW fetus in tectum (22.5 cells/mm$^2$) and in tegmentum (10.2 cells/mm$^2$). During the fetal development the number of AM peaked in tectum around 8-9 GW accounting close to aqueduct 29 cells/mm$^2$, and around 11-12 week in tegmentum reaching 10.2-11 cells/mm$^2$. As the fetus development advanced the number of AM cells both in tectum and tegmentum was slowly falling down reaching in 20/22 GW about 1.5 cells/mm$^2$.

The ramified microglial cells as well as AS emerged in the fetal mesencephalon after 11 GW. A quantitative study revealed also a rapid increase in the density of RM in 13-16 GW (6.3 cells/mm$^2$ - close to aqueduct) and AS cells in 13-16 GW (25-47 cells/mm$^2$ - close to aqueduct) and later the decrease in cell number was observed. Similarly to AM equilibrium in number of RM and AS cells was reached around 20/22 GW. The percentage distribution of each type of microglia and astrocytes cells both in tectum and tegmentum differed markedly during the fetus development dependently on the localization along the axes from aqueduct.

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**Mechanisms of calpain activation under ischemia**

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Calpains, are $Ca^{2+}$-dependent non-lysosomal neutral proteinases, proposed to participate in many important intracellular processes, such as turnover of cytoskeletal proteins and regulation of kinase and transcription factors. The ubiquitous distribution of calpains and the complex regulation of their activity indicate that these proteases play important roles under both physiological and pathological conditions. Recent studies have supported a neurotoxic role of calpain(s) in the pathology of cerebral ischemia. In the present study calpains (μM and mM $Ca^{2+}$ concentrations-sensitive) and their endogenous inhibitor-calpastatin were examined in rat brains subjected to acute postdecapitative global ischemia or to ischemic/hypoxic conditions prolonged to 24 hours. Acute ischemia up to 15 min duration resulted in a gradual, time-dependent decrease of total μ-calpain activity (to 60% of control values) and in the elevation of calpastatin activity (by 28%). The decrease or total μ-calpain activity coincided with its remarkable increase/translocation (to more than 300% of control values) in plasma membranes. Ischemic-hypoxic conditions produced similar, however, less pronounced, changes of μM $Ca^{2+}$-sensitive calpain form. In the case of m-calpain the only observed effect was its translocation and, in consequence the elevation of its activity in plasma membranes. The accumulation of breakdown products, resulting from calpain-catalyzed proteolysis of fodrin, that was observed *in situ* and in electrophoresis Western blotting experiments, indicated an activation of calpain in an ischemic episode. The findings suggest that activation of calpain during ischemia involves its partial translocation toward membranes and is followed (at least in the acute phase) by enzyme down-regulation and increased calpastatin activity.

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**Brain aspergillosis in the AIDS cases**

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Brain aspergillosis is a rather rare type of fungal pathology in the course of AIDS. In reports originating from both European and American large collections, only in Swiss material one case of the brain aspergillosis was described. In our own material comprising 100 brains of patients observed and treated either in Institute of Infectious Diseases, School of Medicine, Warsaw or in AIDS Centre of Wolski Hospital for Infectious Diseases in Warsaw in the course of 1987-1995 years in 3 cases brain pathological examination revealed changes characteristic for aspergillosis. In the all three cases brain pathology was mixed in nature. In one case aspergillosis coexisted with micronodular cytomegalic encephalitis, in the second one - with HIV - specific changes combined with cytomegaly. The third case comprised aspergillosis with progressive multifocal leukoencephalopathy. Pathomorphology of aspergillosis infections varied greatly in all the cases. In the first case an isolated large brain abscess was found at the periphery of the frontal lobe. Extensive granulomatous surrounding of the abscess was characterized by abundance of CMV cells with typical intranuclear inclusions. In the second case the inflammatory-necrotic process involved cerebral cortex of gyrus rectus and orbital gyri in both cerebral hemispheres and their leptomeninges, in particular within the interhemispheric fissure as well as optic nerves and optic chiasma. Localization of the brain pathology was suggestive of direct penetration of the process from the nasal cave. In the third case the process was multifocal and widely spread within cerebral and cerebellar hemispheres. The particular foci were composed of micronecrotic tissue with evident inflammatory reaction, varying greatly in their size and morphology, the latter depending on the localization and advancement of the process. The most-typical feature for all the cases was the appearance of severe inflammatory thrombotic changes within the blood vessels involved by pathological process. Abundant fungi were present both within intravascular thrombotic material, vascular walls and the surrounding tissue. No giants cells of Langerhans type were found within granulomatous necroses. They are considered to be a typical component of aspergillous changes in cases without HIV infection.