

Within the third month the quantity of sites with emphysema increases. Many bronchial tubes are deformed, have a star-shaped form on a section and contain congestions of mucin, infiltrated by cells. Mucosa of bronchial tubes forms protrusions, similar to polyps. Process of an atrophy of a muscular cover of bronchial tubes progresses that is expressed in reduction of its volume and increase in intermuscular connective tissue.

The tendency to permeability strengthening remains, on the course of vessels numerous narrowings, expansions are formed. In the external cover of vessels and extravascular space collagen deposits are well seen. When coloring strong green walls of vessels visually lose the integrity, in them the "emptiness" corresponding to intermuscular stratifications and cellular infiltrates are formed.

The fourth month of experiment is characterized by rough destructive changes. The number of the deformed bronchial tubes with protrusions of walls similar to sacks, characteristic for bronchiectasia increases. Atrophic processes in a bronchial wall are caused by growth and hypostasis of intermuscular connecting tissue. Lightness of lungs increases with increase of volume of the parenchyma, which has changes in a type of emphysema.

In a vascular wall the muscular cover is exposed to processes of an active collagen deposits. Intensive diffusion coloring of extravascular space by fuchsin shows an increase in collagen deposits with distribution of process to a surrounding parenchyma. Thickness of a vascular wall is increased, the clearness of its contours is lost due to plasmatic treatment and a cellular infiltration. The described changes in a vascular wall in luminescent microscopy are followed by significant increase in its permeability.

In a weak Schiff-positive stromal connective tissue of bronchial tubes presence of intensively painted fibers which appearance is connected with simultaneous of a proteolysis and formation of collagen is recorded. Intermittence and a disorientation of these fibrous structures, especially in the field of massive cellular infiltration is noted.

## CONCLUSIONS

Long influence of the damaging factor accounts for development of chronic pathological process. Inhalation of low concentration of the hydrogen sulfide containing gas within the first month of experiment is followed by changes of functional character. Long inflammatory process causes a perversion of compensatory reactions that leads to destruction of lung parenchyma and activation of proliferative reactions. The developing deficiency in stromal connective tissues affects the structural organization of lungs and promotes further progressing of pathological process.

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# THE PATHOLOGY AND MORFOLOGY OF LESION IN MICE BRAIN CORTEX UNDER INFLUENZA VIRUS A (H3N1 1/62) INFECTION

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The pathology caused by so-called "non neurotropic" virus infection, is not well understood. The urgency of the problem is not just the nature of the epy – and pandemic influenza with enormous human and economic troubles but the frequency lesion, if CNS damages is the second after respiratory.

There are a number of features in the CNS contributing to the persistence of the virus. In addition to

the above mentioned these include: 1) mostly humoral factors of immune protection; 2) existence of special cells of mononuclear phagocytic system-microglial cells; 3) low capacity to develop interferon. So far, the question remains – what are the qualitative differences in morphological and pathogenetic entity lesion in viral influenza infection.

Our own examinations, performed in the period from 1996, have shown a direct cytopathic effect of influenza A virus (H3N1, H3N2, Yong Kong 1/68) on nuclei of medulla oblongata, parasympathetic nervous system with its primary damage. The neural transport of viruses from the periphery to the CNS is not well investigated but we and other authors have demonstrated on the CBA mice models that n. vagus, nuclei of m. oblongata and brain stem are most affected.

The aim of study is to reveal pathologic changes of brain cortex nervous elements in influenza A virus infection in experiment and to clarify neural disturbance by analyzing their ultra structural findings in

mice under experimental influenza virus infection in first 16 days of experiences.

All experiments using live A Hong Kong H3N1 viruses were performed in a biosafety level-3 laboratory approved for use by Georgian Disease Control Center.

Ultra structural and histological appearance also volume fraction of neurons from parieto-temporal cortex in 6-week-old mice were carried out after 24, 48-72 hours and 5-16 days of intranasal inoculation of influenza virus A (H3N1 1/62) Hong Kong.

Nerve cells in layer V of the cerebral cortex of mice respond to infection with influenza virus by complex changes: acute swelling, chromatolysis, vacuolization, shrinkage and neuronophagia.

Quantitative changes in the “acute” phase of infection up to 72 hours to testify of cortex cytoarchitectonic in the form of lower differentiation and higher monotony size of neurons with their polarization from very small cells to large, hypertrophied, which is unusual for the “intact” model of brain.

## THE STUDY OF MYOCARDIUM REMODELING AFTER INTERVENTIONAL TREATMENT OF ATRIAL FIBRILLATION IN PATIENTS WITH ISCHEMIC HEART DISEASE AND ARTERIAL HYPERTENSION

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### OBJECTIVE

To study of electrical myocardium remodeling after interventional treatment of atrial fibrillation (AF) with patients with ischemic heart disease (IHD) and arterial hypertension (AH).