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SEMEY MEDICAL UNIVERSITY

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ALZHEIMER'S DISEASE. ASL. MULTIPLE SCLEROSIS

DEPARTMENT OF PATHOLOGICAL ANATOMY AND JUDICIAL MEDICINE NAME OF PRUGLO Y.V.

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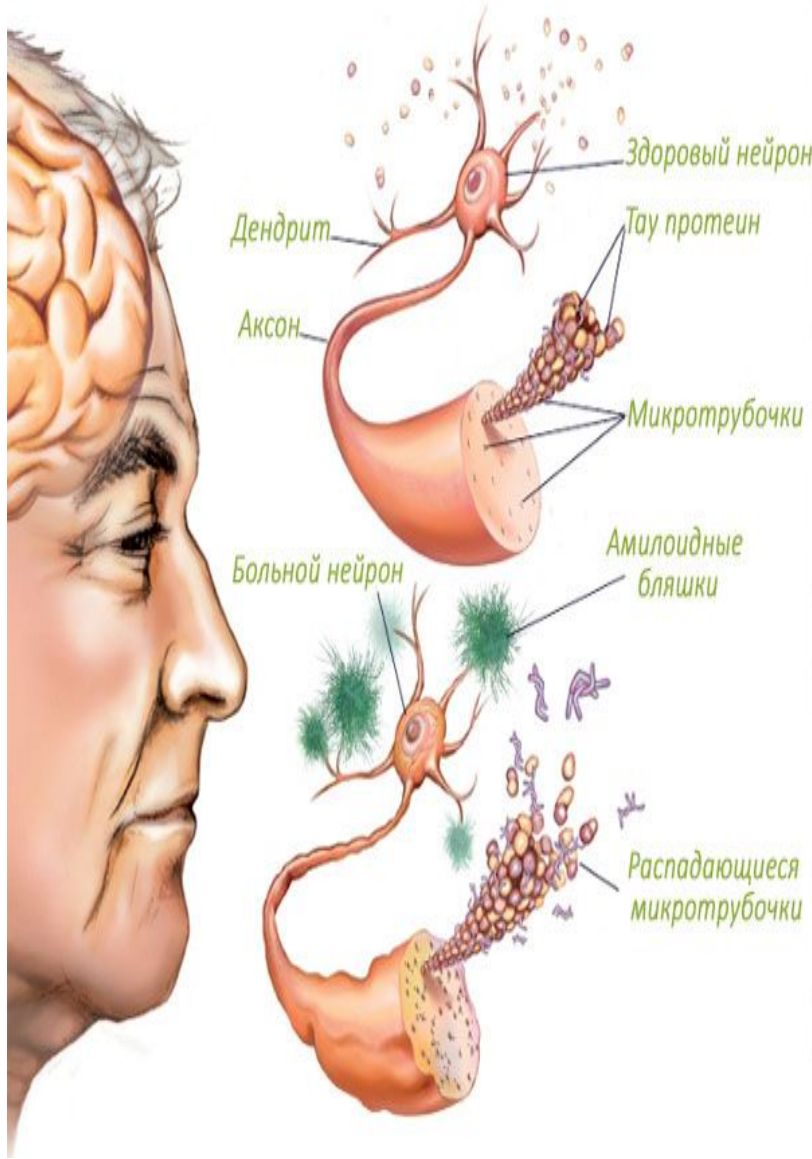
ALZHEIMER'S DISEASE

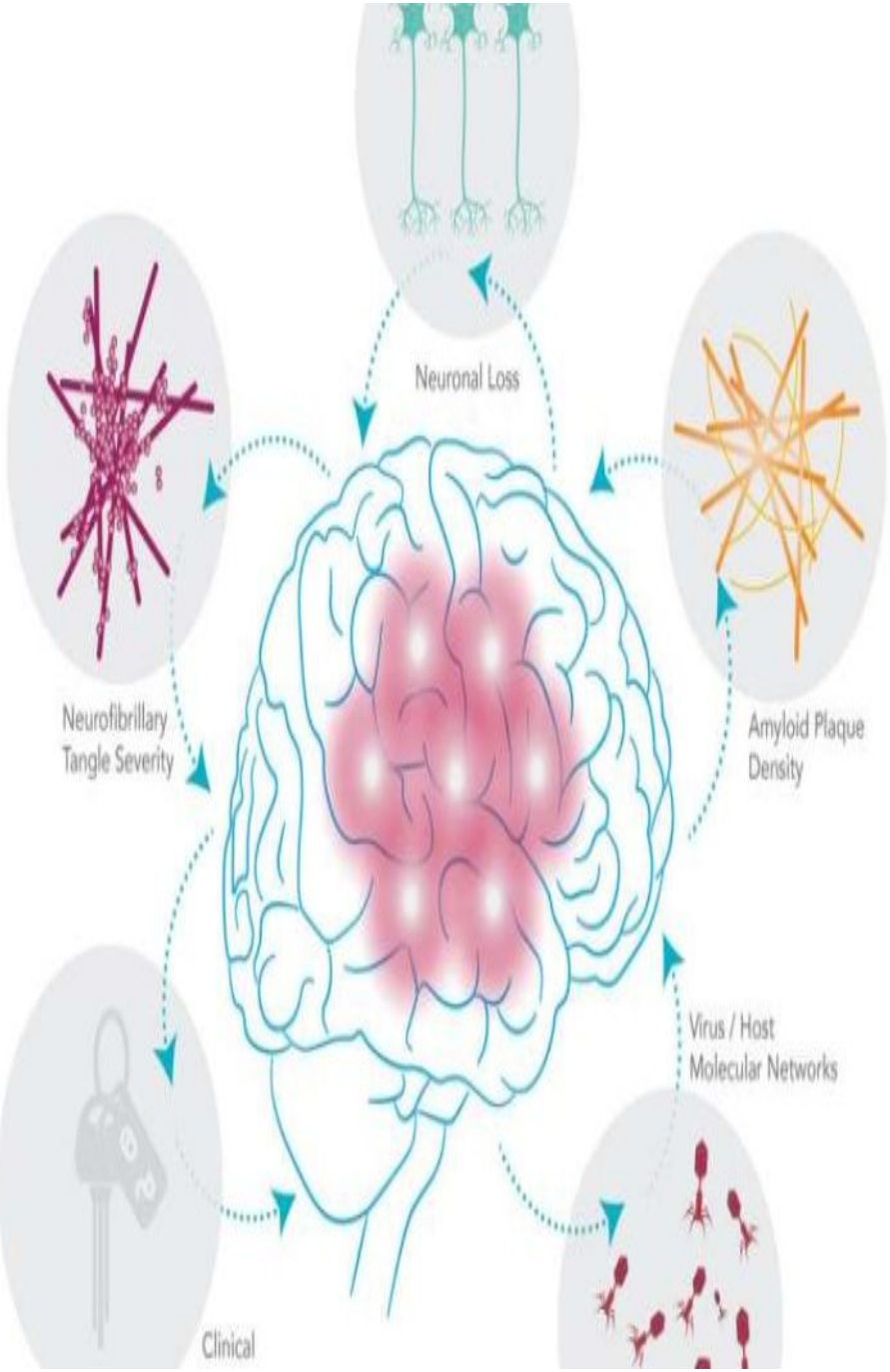
- Alzgeimer's disease (also senile dementia of the Alzheimer's type) is the most common form of dementia, a neurodegenerative disease, first described in 1907 by the German psychiatrist Alois Alzheimer. As a rule, it is found in people over 65 years of age but there is also early Alzheimer's disease - a rare form of the disease. The global morbidity rate for 2006 was estimated at 26.6 million people, and by 2050 the number of patients could increase by four
- Alzheimer's disease occurs with severe intellectual disorders and emotional lability, while focal neurological symptoms are absent. Clinical manifestations of the disease are associated with progressive general atrophy of the brain, but especially of the frontal, temporal, and occipital regions.

ETIOLOGY AND PATHOGENESIS

- The cause and development of the disease is idiopathic. It was assumed that the cause of the disease is an acquired deficiency of acetylcholine and its enzymes in the structures of the cerebral cortex. Recently it has been shown that the clinical manifestations of Alzheimer's disease are associated with senile cerebral amyloidosis, which is found in 100% of cases. In this regard, there has been a tendency to view Alzheimer's disease as a form of cerebral senile amyloidosis. Amyloid deposits are detected in senile plaques, brain vessels and membranes, as well as in vascular plexuses. It is established that cerebral amyloid protein is represented by 4 CD-a protein, whose gene is localized on chromosome 21.



- Along with the synthesis of extracellularly located amyloid fibrils, which are the basis of senile plaque, pathology is expressed in Alzheimer's disease and intracellular fibrillar structures - cytoskeleton proteins. It is represented by the accumulation of pairwise twisted filaments and straight tubules in the cytoplasm of neurons, which can fill the entire cell body, forming peculiar neurofibrillary plexuses. The filaments of the neurofibrillary tangles have a diameter of 7–9 nm, give a positive reaction to a number of specific proteins (tau protein), microtubule proteins an neurofilaments. Cytoskeleton pathology is expressed in Alzheimer's disease and in proximal dendrites, in which actin microfilaments accumulate (Hirano calf).



STAGES OF ALZHEIMER'S DISEASE

Effects of ageing on memory but not AD
Forgetting things occasionally
Misplacing items sometimes
Minor short-term memory loss
Not remembering exact details

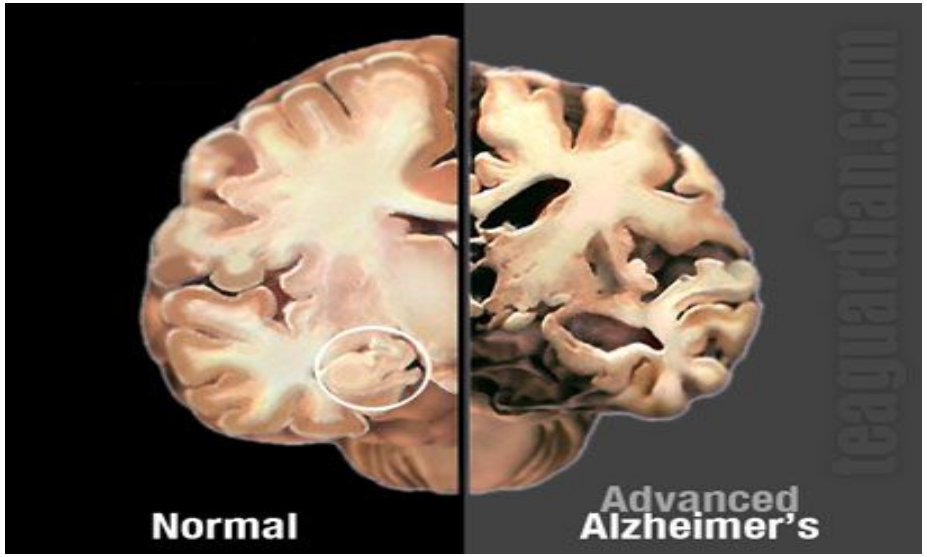
Early stage Alzheimer's
Not remembering episodes of forgetfulness
Forgets names of family or friends
Changes may only be noticed by close friends or relatives
Some confusion in situations outside the familiar

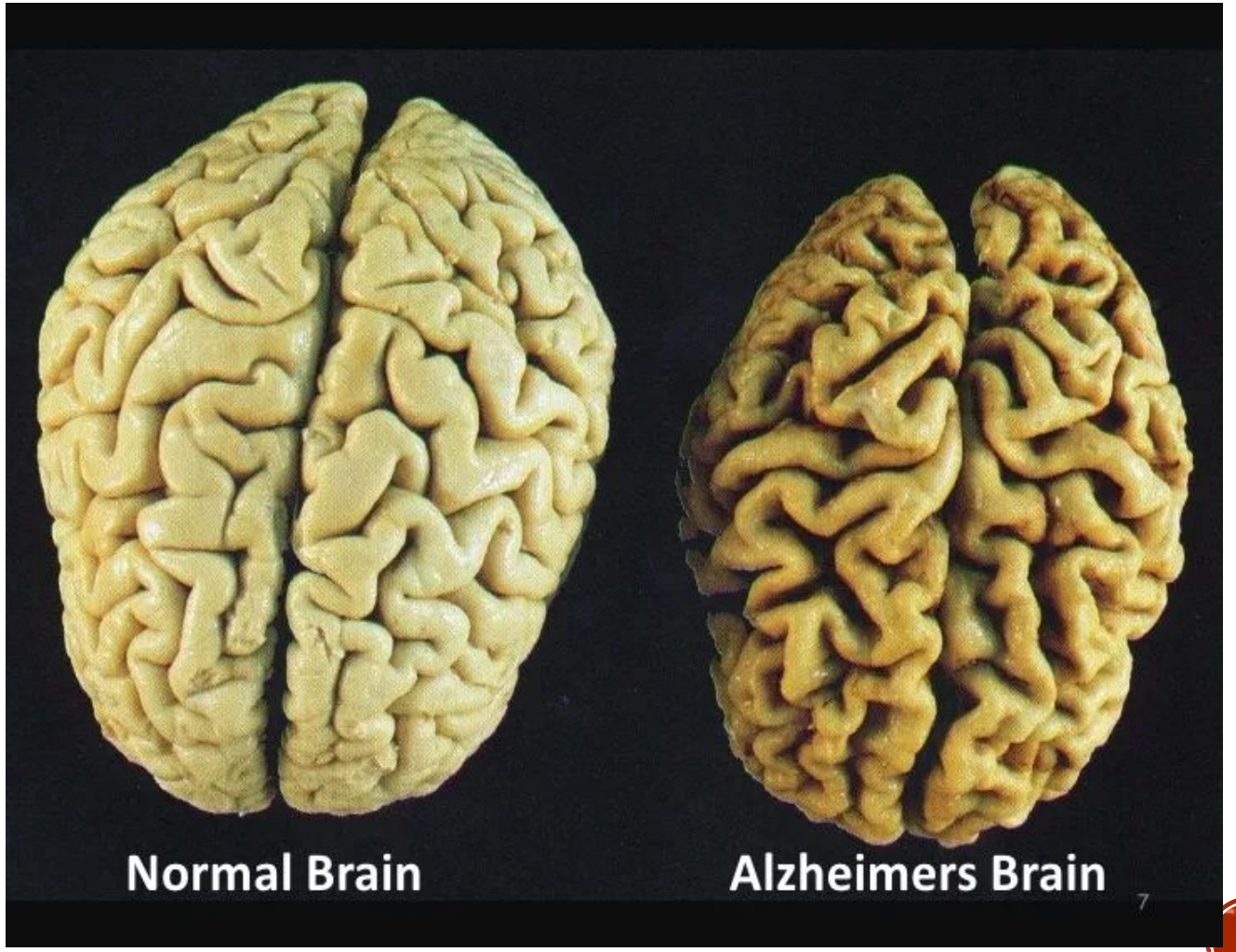
Middle stage Alzheimer's
Greater difficulty remembering recently learned information
Deepening confusion in many circumstances
Problems with sleep
Trouble determining their location

Late stage Alzheimer's
Poor ability to think
Problems speaking
Repeats same conversations
More abusive, anxious, or paranoid

PATHOLOGICAL ANATOMY.

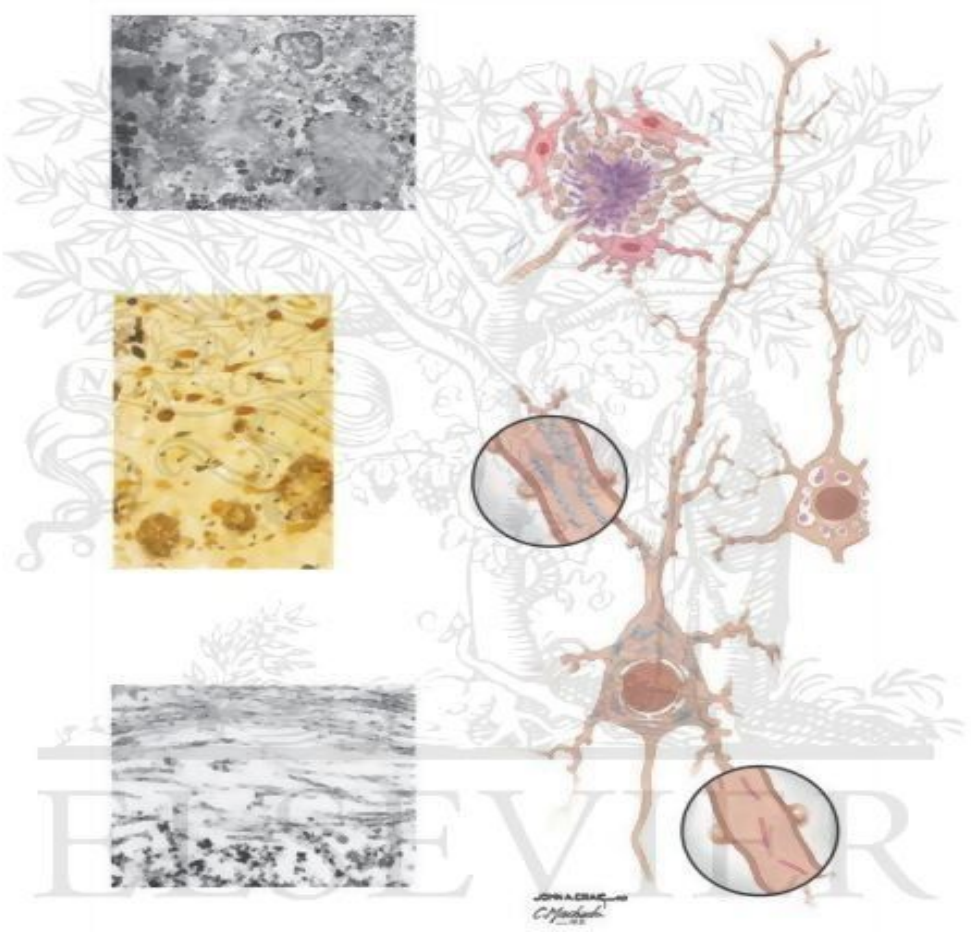
- At autopsy, atrophy of the cerebral cortex is found (the thinning of the cortex predominates in the frontal, temporal and occipital lobes). In connection with brain atrophy, hydrocephalus often develops.





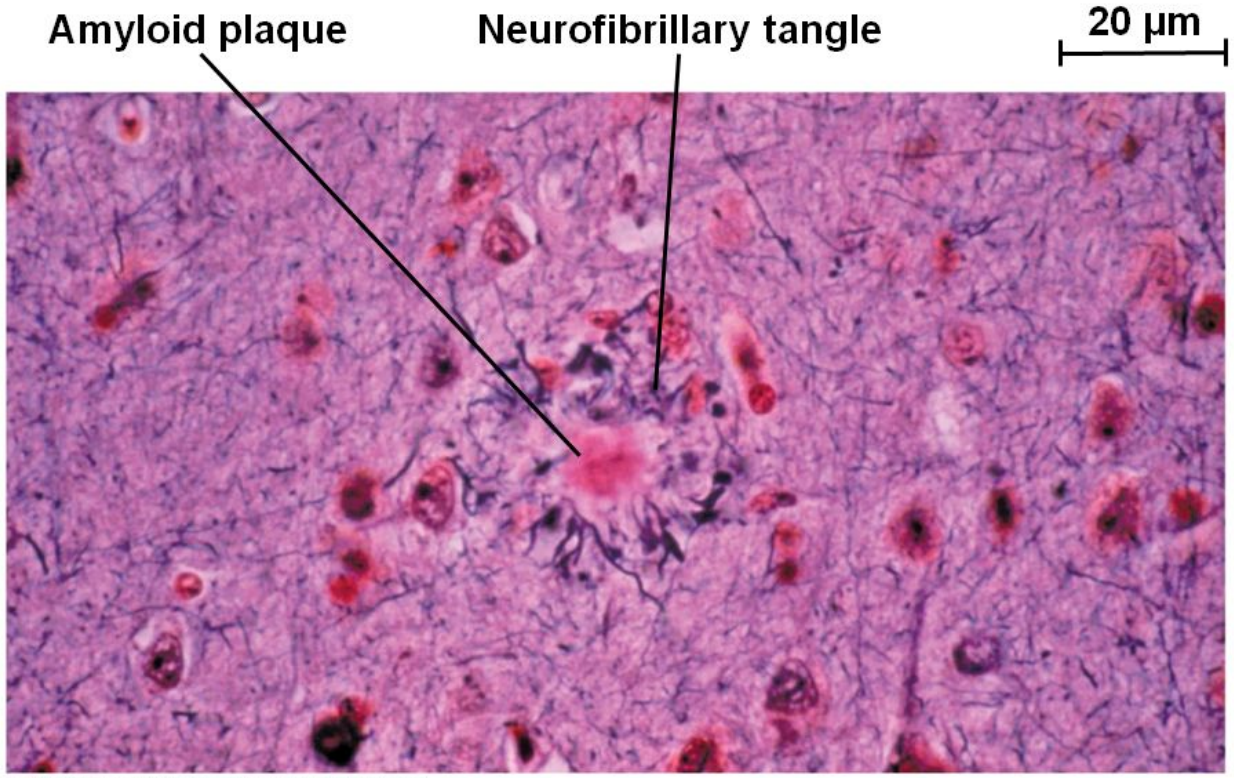
MICROSCOPIC EXAMINATION

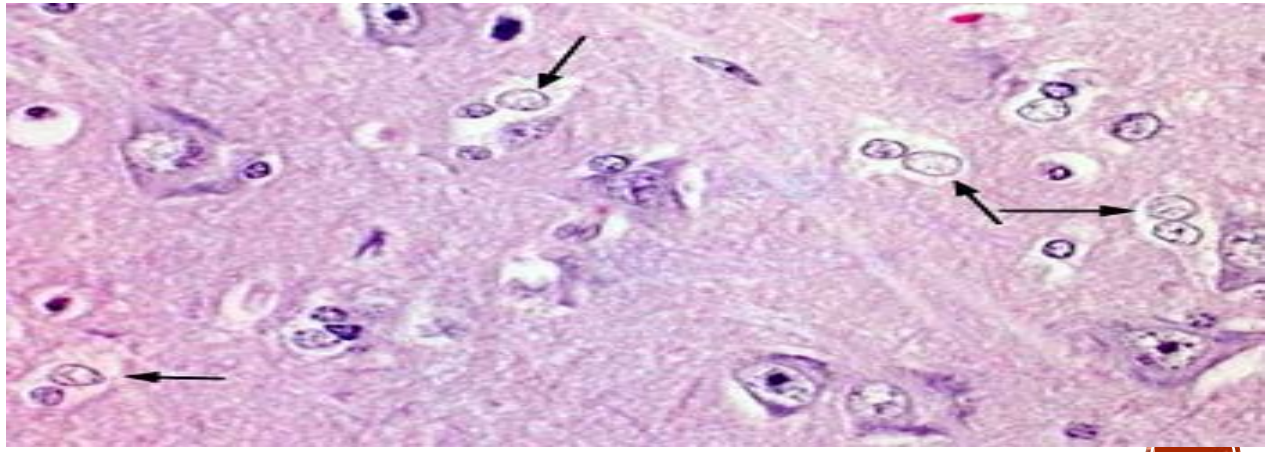
- of the cortex of the atrophic lobes of the brain, the hippocampus and the amygdala reveals senile plaques, neurofibrillary tangles (tangles), neuronal damage, Hirano body. Senile plaques and neurofibrillary plexuses are detected in all parts of the cerebral cortex, excluding motor and sensitive areas, neurofibrillary plexuses are also more often found in the basal nucleus of Meinert, Hirano bodies are detected in neurons in the hippocampus.



- Senile plaques consist of amyloid deposits, surrounded by twisted filaments; on the periphery of plaques microglia often find cells, sometimes astrocytes. Neurofibrillary tangles are represented by spiral-shaped, pairwise twisted filaments detected by silver impregnation methods. They look like tangles or nodules of fibrillar material and straight tubes in the cytoplasm of neurons; filamentous masses are ultrastructurally identical to neurofilaments. Neurons in the affected areas are reduced in size, their cytoplasm vacuolized, contains argyrophil granules. Hirano bodies found in proximal dendrites have the form of eosinophilic inclusions and are represented by a cluster of oriented actin filaments.

Microscopic signs of Alzheimer's disease



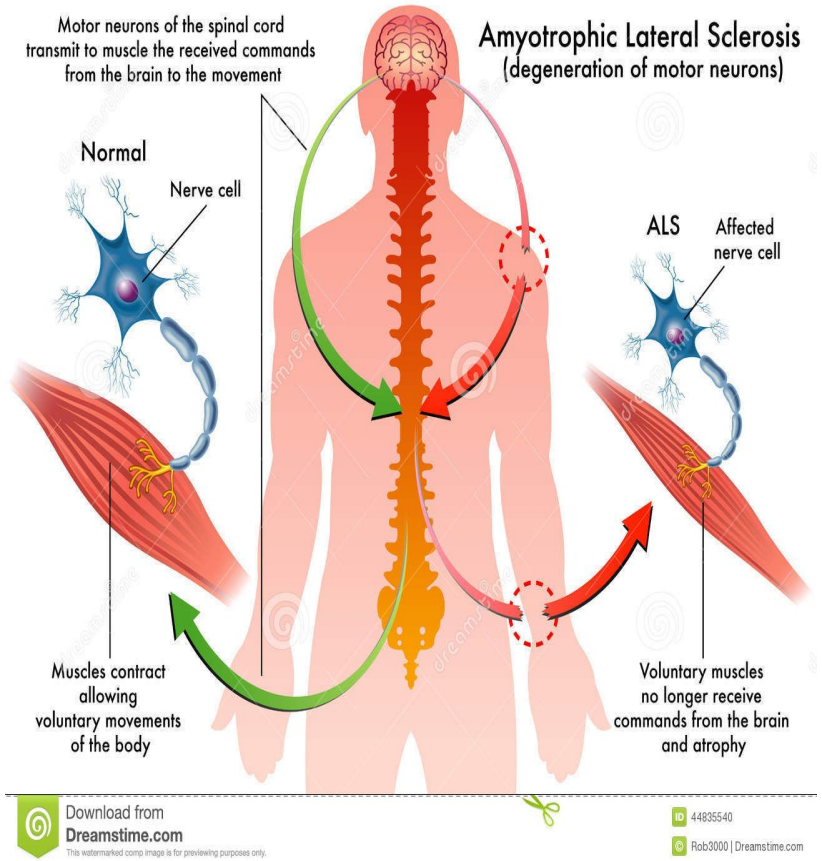


Amyotrophic lateral sclerosis (Charcot's disease)

is a progressive disease of the nervous system associated with simultaneous damage to the motor neurons of the anterior and lateral columns of the spinal cord and peripheral nerves. Characteristic is the slow development of spastic paresis, mainly of the arm muscles, joined by muscular atrophy, increased tendon and periosteal reflexes. Men get sick twice as often as women. Clinical manifestations of the disease usually begin in middle age, the steady progression of movement disorders ends in death after a few (2-6) years. Sometimes the disease has an acute course.

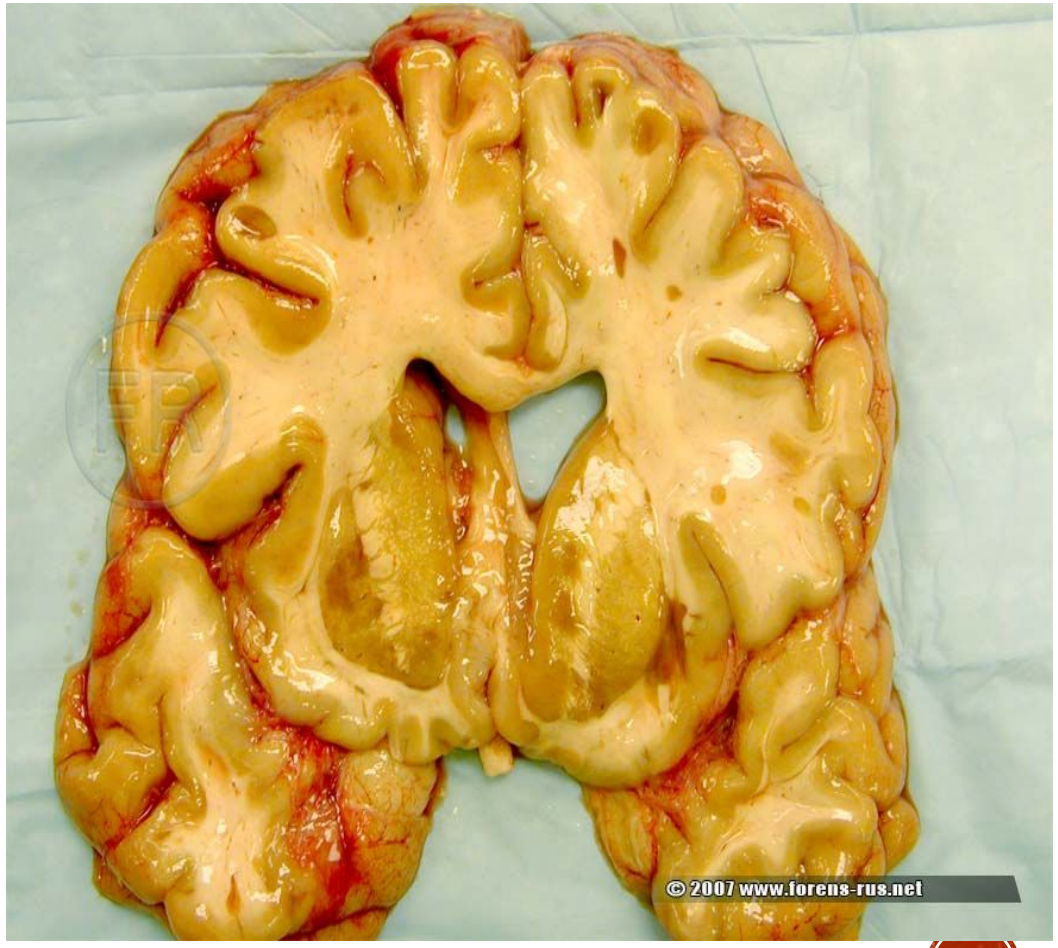
Etiology and patogenesis

The cause and mechanism of the disease is unknown. The role of viruses, immunological and metabolic disorders is discussed. A number of patients with a history of polio. In such cases, poliomyelitis virus antigen is found in jejunal biopsy specimens, and immune complexes are found in the blood and renal glomeruli. Based on these data, amyotrophic lateral sclerosis is believed to be associated with chronic viral infection.



Pathological anatomy.

At autopsies are selective atrophy of the anterior motor roots of the spinal cord, they are thinned, gray; while the rear sensitive roots remain normal. On the transverse sections of the spinal cord, the lateral cortico-spinal tracts are condensed, whitish in color, delimited from other tracts by a clear line. In some patients, atrophy of the pre-cerebral gyrus of the brain is noted, sometimes atrophy captures VIII, X and XII pairs of cranial nerves. In all observations expressed atrophy of skeletal muscles.



Microscopic examination

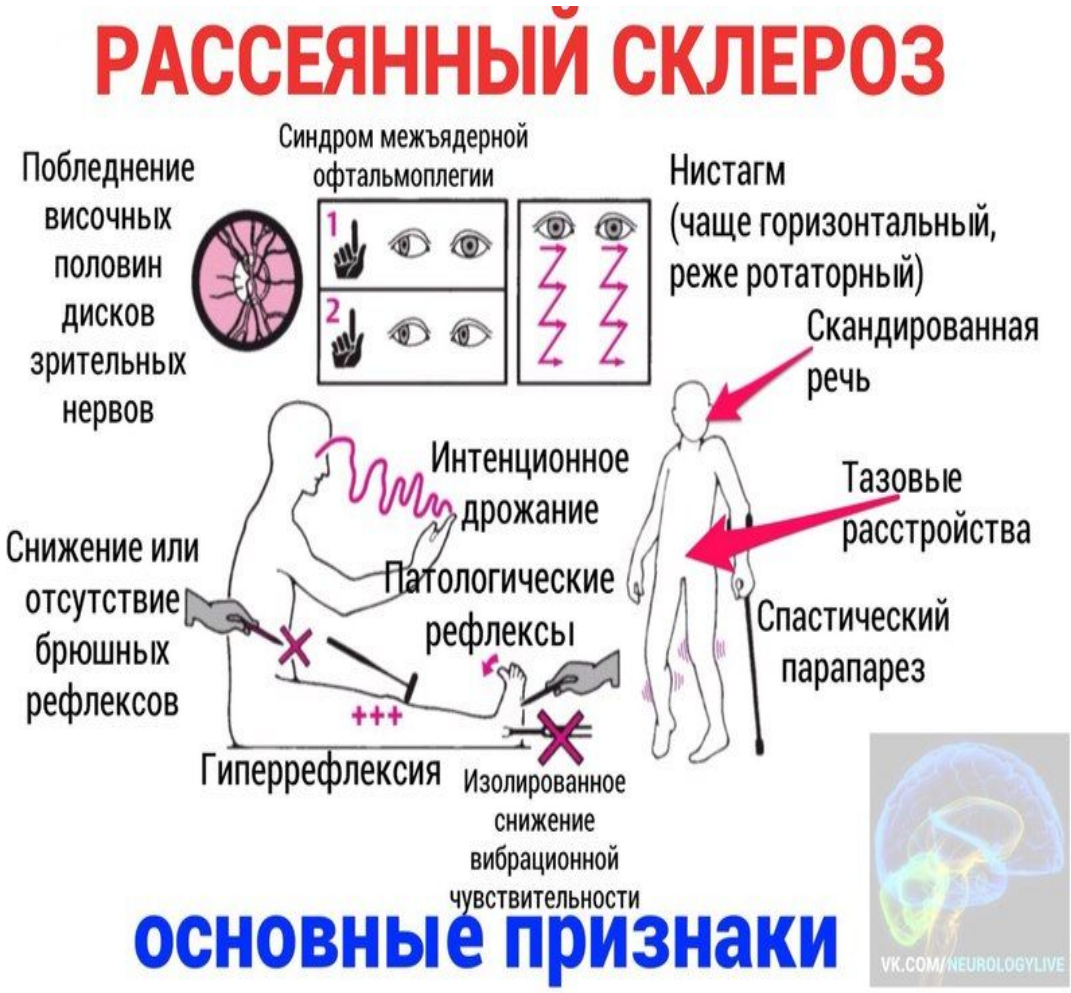
in the anterior horns of the spinal cord find pronounced changes in nerve cells; they are wrinkled or in the form of shadows; extensive fields of neuron loss are found. Sometimes foci of neuron prolapse are found in the brain stem and precentral gyrus. In the nerve fibers of the affected areas of the spinal cord are determined demi-linisation, uneven swelling with subsequent disintegration and death of axial cylinders. The demyelination of nerve fibers usually extends to peripheral nerves. Often, the pyramidal paths are involved in the process throughout their length - the spinal cord and the medulla, up to the cortex of the cerebral hemispheres. As a rule, reactive proliferation of glial cells is noted. Some observations describe minor lymphoid infiltrates in the spinal cord, its lining and peripheral nerves along the vessels.

Multiple sclerosis

is a chronic progressive disease characterized by the formation in the brain and spinal cord (mainly in the white matter) of scattered demyelination foci in which the growth of glia occurs with the formation of foci of sclerosis - plaques. It usually begins at the age of 20-40 years, more often in men; proceeds in waves, periods of improvement are replaced by exacerbations of the disease. Differences and multiple localization of lesions of the brain and spinal cord determine the diversity of the clinical manifestations of the disease: intentional tremor, nystagmus, scanned speech, a sharp increase in tendon reflexes, spastic paralysis, visual disturbances. The course of the disease is different. There may be an acute and severe course (acute forms of the disease) with the rapid development of blindness and cerebellar disorders, and perhaps a mild course with minor dysfunction of the central nervous system and its rapid recovery.

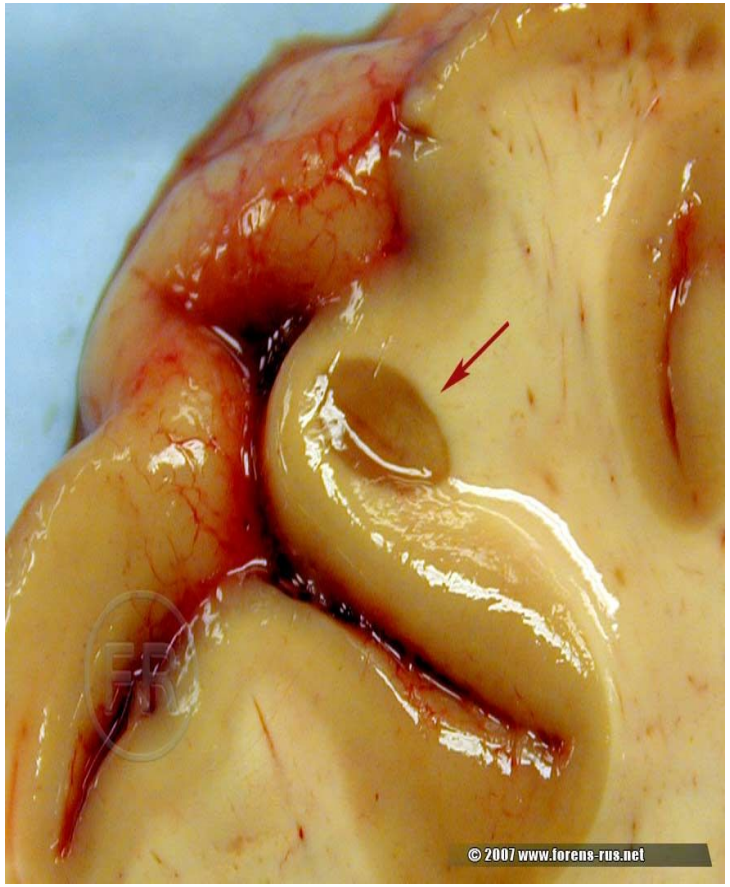
Etiology and pathogenesis.

The causes of the disease remain unclear. The viral nature of the disease is most likely, in 80% of patients antiviral antibodies are found in the blood, but the spectrum of these antibodies is quite wide. The virus is believed to be tropic to oligodendrogly cells related to myelination processes. The role of autoimmunization does not exclude the development and progression of the disease. Evidence of immune aggression against myelin and oligodendroglia cells has been obtained.

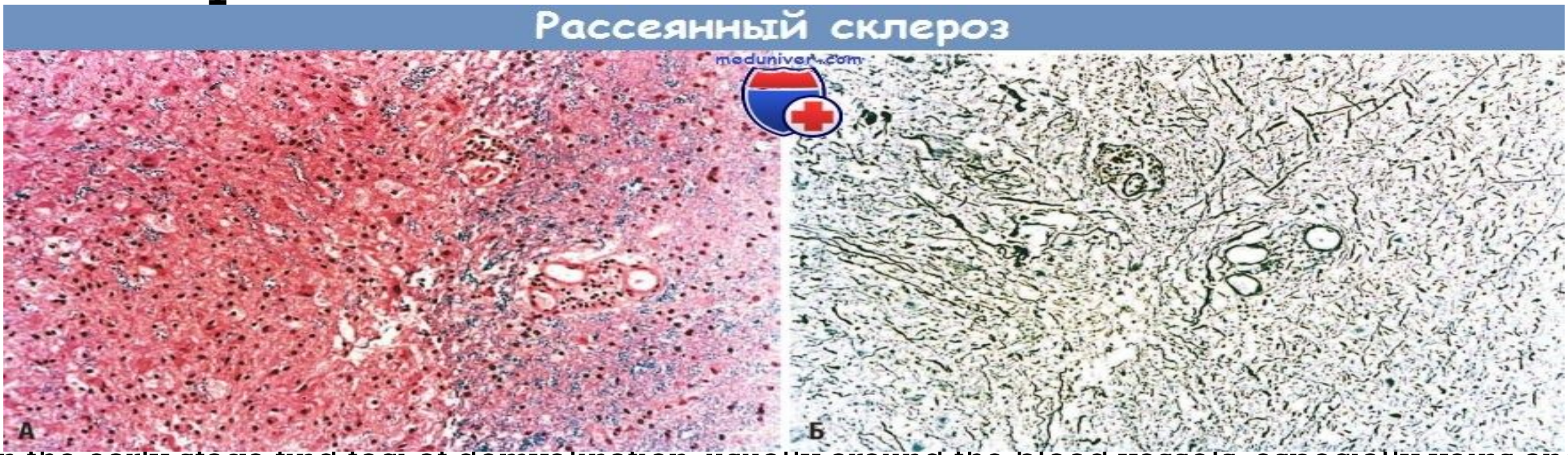


Marphogenesis

Externally, the superficial parts of the brain and spinal cord are little changed; occasionally edema and thickening of the pia mater. On sections of the brain and spinal cord, a large number of gray-colored plaques scattered in white matter are found (sometimes they have a pinkish or yellowish tint), with They are found around the ventricles of the brain, in the spinal and medulla, brain stem and optic hillocks, in the white matter of the cerebellum; less plaques in the hemispheres of the large brain. In the spinal cord, lesions may be located symmetrically. Often, the optic nerves, chiasm, and visual pathways are affected.



Microscopic examination



in the early stage find foci of demyelination, usually around the blood vessels, especially veins and venules (perivenial demyelination). Vessels are usually surrounded by lymphocytes and mononuclear cells, axons are relatively preserved. With the help of special stains on myelin, it is possible to establish that at first myelin sheaths swell, tinctorial properties change, irregularity of their contours, spherical thickenings along the fibers appear. Then the fragmentation and disintegration of the myelin sheaths occurs. The breakdown products of myelin are absorbed by microglial cells, which turn into granular balls. Changes in axons can be found in fresh foci - enhanced silver impregnation, uneven thickness, swelling; severe axon destruction is rarely observed.

Outcomes

The outcome of the death of Alzheimer's diseas, ALS, Multiply sclerosis are mainly pneumonia, bronchopneumonia, cachexia, infectious diseases and other

Conclusion

Dementia is a loss of cognitive functioning — thinking, remembering, and reasoning — and behavioral abilities to the extent that it interferes with everyday life and human activities. These functions include memory, language skills, visual perception, problem solving, self-management, and the ability to focus and pay attention. Some people with dementia cannot control their emotions, and their personalities may change. The severity of dementia varies from the easiest stage, when it only begins to affect the functioning of a person, to the most severe stage, when a person must be completely dependent on others for the main types of life activity. And we need to better understand why this is happening to fight it.

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